weave

Centre for Integrative Psychiatry

WEAVE 100 / PAPER I

Brain, Mind and Method

One hundred ten-mark questions, solved as answer maps

MD Psychiatry theory. DNB revision fits too.



ILLUSTRATED REVIEW EDITION

weave.clinic

Dr. Wilfred Dsouza · Dr. Niharika Reddy

WEAVE

A NOTE ON WHO MADE THIS

Weave, Centre for Integrative Psychiatry

Weave is a psychiatry practice and a publishing house for the things clinicians and families actually need to read. Two consultant psychiatrists run it, online across India and in person in Mumbai.

WHAT WEAVE DOES	Integrative psychiatry, online and in Mumbai, and a free library for residents, clinicians and families.
WHO RUNS IT	Dr. Niharika Reddy and Dr. Wilfred D'souza, Consultant Psychiatrists.
WHAT THAT MEANS HERE	This book is free because the practice pays for it. There is nothing to buy on any page of it.

WEAVE 100 - PAPER I

Brain, Mind and Method

One hundred ten-mark questions, solved as answer maps

THE WEAVE 100 SERIES

TITLE

Weave 100, Paper I: Brain, Mind and Method

Book one of Weave 100, one hundred solved ten-mark questions for each paper of the MD psychiatry written examination.

WRITTEN BY

Dr. Wilfred Dsouza and Dr. Niharika Reddy

PUBLISHED BY

Weave, Centre for Integrative Psychiatry · weave.clinic

EDITION

First compiled 2026. Free to read, share and print for study.

First edition (R1), August 29, 2026.
Illustrated edition, September 2026. Original answer maps with 179 selected plates. Page references updated for this edition.

SOURCES

Question records were read from the published papers and archives of the boards named on the back cover. Board names appear throughout because the count is only meaningful with them. No board has endorsed this book and none was asked to.

The clinical content is written for doctors and doctors in training. It is a study and revision aid, not a substitute for current guidelines or clinical judgement. Verify doses, thresholds and statutory detail against the current references and your local protocols before any clinical use.

Contents

FRONT MATTER

How to use this book 6

Where the marks have gone 7

How the hundred were picked 8

What this book cannot promise 9

THE HUNDRED, IN WEIGHT ORDER

01 **Neurochemistry and neurophysiology** I-01 to I-13 · 13 slots · 14.5% 10

02 **Functional neuroanatomy and neuroimaging** I-14 to I-25 · 12 slots · 13.5% 44

03 **Personality theory and psychodynamics** I-26 to I-35 · 10 slots · 10.5% 89

04 **Learning, memory and general psychology** I-36 to I-44 · 9 slots · 9.9% 110

05 **Psychometry and assessment** I-45 to I-53 · 9 slots · 9.1% 139

06 **Genetics in psychiatry** I-54 to I-62 · 9 slots · 9.0% 169

07 **Social, cross-cultural and evolutionary psychology** I-63 to I-70 · 8 slots · 7.8% 192

08 **Biostatistics and epidemiology** I-71 to I-78 · 8 slots · 7.6% 217

09 **Research methodology** I-79 to I-84 · 6 slots · 5.3% 246

10 **Developmental psychology and attachment** I-85 to I-90 · 6 slots · 5.1% 265

11 **Nosology, classification and descriptive psychopathology** I-91 to I-96 · 6 slots · 4.9% 279

12 **Neuroendocrinology and basic psychopharmacology** I-97 to I-100 · 4 slots · 2.8% 296

BACK MATTER

Ten sets, ten sittings 313

Index 315

Illustrations 319

Credits and colophon 324

HOW TO USE THIS BOOK

One question, one page

Every page after the weightage chapter is one question and its answer map. Nothing continues onto a second page, so a map can be read in the time you have rather than the time it deserves. Five blocks, always in the same order.

1	**Question band**	The slot number, the question as a board set it, and three chips: the mark split, the area, and where the question has been seen.
2	**Skeleton**	Three to five rows. The mark column sums to ten, so you can see the allocation before you read a word of content.
3	**Body**	Two to four components, chosen for the question: a comparison table, a flowchart, a plate, typed lines, at most one pitfall box.
4	**What earns the marks**	Three or four things an examiner ticks. Each one names something that is on the page above it.
5	**Links**	The related slots in this book, and the Weave Sprint day that teaches the underlying topic in full.

The colour code

Structure	The shape of the answer: headers, rules, table heads, the question band. When a line is indigo it is telling you how the answer is organised.
Key number	A figure to carry exactly: a mark split, a threshold, a count, a cut-off. Amber means memorise the number, not just the shape of it.
Trap	The mistake the examiner is waiting for. When a line is red it is warning you off an error that looks right until it costs you the mark.

Two ways to work. Read an area straight through, banner first, to see how the boards have carved it up. Or use the ten sets at the back: each is ten questions and one hundred marks, drawn one from each rank decile, so a set is a paper-shaped sitting rather than ten easy questions or ten hard ones.

The chips are traceable. A recurrence chip names the boards and gives a count against a stated denominator. If it says three of seventy-eight sittings, that is three sittings out of the seventy-eight this book counted, and the boards are listed by name so you can check.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper I.

Twelve areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Neurochemistry and neurophysiology	0.455	0.532	0.479	14.52%	13
02 Functional neuroanatomy and neuroimaging	0.456	0.422	0.446	13.53%	12
03 Personality theory and psychodynamics	0.321	0.407	0.347	10.52%	10
04 Learning, memory and general psychology	0.335	0.304	0.326	9.89%	9
05 Psychometry and assessment	0.318	0.254	0.299	9.07%	9
06 Genetics in psychiatry	0.252	0.396	0.295	8.96%	9
07 Social, cross-cultural and evolutionary psychology	0.326	0.098	0.257	7.81%	8
08 Biostatistics and epidemiology	0.232	0.295	0.251	7.61%	8
09 Research methodology	0.115	0.311	0.174	5.27%	6
10 Developmental psychology and attachment	0.192	0.110	0.168	5.09%	6
11 Nosology, classification and descriptive psychopathology	0.051	0.422	0.162	4.92%	6
12 Neuroendocrinology and basic psychopharmacology	0.077	0.127	0.092	2.79%	4

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper I. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper I reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	12	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The twelve weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 11 anchor, 18 high, 3 recurrent, 68 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 403 selectable Paper I questions in the record, 342 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper I has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 12 · P1-A02

Neurochemistry and neurophysiology

13	14.5%	I-01	I-13
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 2 · SINGLE 9

REVISION ORDER

The memory block first and together: four slots share one answer and two of them carry the anchor weight in this area. Sleep next, as a set of three, and the classification slot last of the three because it is the only one that turns clinical. The dopamine pathways after that, short and set often. The three transmitter slots, the electrophysiology slot and the markers slot are independent and each stands alone.

WHAT IS IN THIS AREA

Memory in four slots (the neurobiology with its clinical relevance, the cortical contribution with memory impairment across illnesses, the types of memory with rehabilitation in chronic illness, and synaptic plasticity). Sleep in three (the neurobiology of sleep and wakefulness, polysomnography with the stages and their EEG findings, and the classification with restless legs and a benzodiazepine taper). Single transmitters in three: nitric oxide, glutamate and the endocannabinoids. The dopamine pathways, the electrophysiological abnormalities of four disorders, and the biological-markers slot.

I - 01

Describe the Neuro-biology of memory and its relevance in clinical practice. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

KUHS, MUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The four stages	Encoding, consolidation, storage, retrieval, each a separate failure
3	Systems and their anatomy	One structure per system, not one memory centre
3	Cellular basis	Long term potentiation, and what makes a trace permanent
2	Clinical relevance	The bedside uses that follow from the anatomy

THE LINE WORTH WRITING FIRST

Memory is not one faculty in one place. It is several parallel systems, each with its own anatomy, its own timescale and its own way of failing. An answer that names the systems separately has already earned more than one that describes memory in general.

SYSTEM	ANATOMY	WHAT IT HOLDS	WHERE IT FAILS
Working	Dorsolateral prefrontal cortex	Seconds of actively held material	Schizophrenia, ADHD, delirium
Episodic	Hippocampus, medial temporal lobe	Events with a time and a place	Alzheimer disease, Korsakoff, ECT
Semantic	Anterolateral temporal cortex	Facts stripped of their context	Semantic dementia
Procedural	Striatum and cerebellum	Skills and habits	Parkinson disease; spared in amnesia

The last row is the one that separates the bands: procedural learning survives dense amnesia, which is what proves the systems are separate.

- **Cellular basis.** Long term potentiation at glutamatergic synapses. Strong activity relieves the magnesium block on the NMDA receptor, calcium enters, kinases move more AMPA receptors into the membrane, and the synapse is stronger.
- **Making it last.** Early potentiation needs no new protein. Late potentiation does: calcium signalling reaches CREB, new proteins are made, spines change shape. That gene step is why consolidation takes hours and why it can be blocked.
- **Clinical relevance.** Sparing of procedural learning guides rehabilitation; ECT produces retrograde and anterograde deficits that mostly recover; benzodiazepines and alcohol block encoding rather than erase storage, which is what blackout means.

WHAT EARNS THE MARKS

- **Four stages named before any anatomy.** It lets you say which stage a drug or a lesion damages.
- **Each system with its own structure,** and procedural memory identified as the spared one.
- **Relevance made concrete.** Blackout, ECT and rehabilitation are the three the examiner expects.

SEE ALSO Slot I - 10 types of memory and rehabilitation Slot I - 13 synaptic plasticity SPRINT Day 1, Functional Neuroanatomy and Neurophysiology

I - 02

Describe the role of cortex in memory formation. Discuss about memory impairment in various psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **Which cortex, for what**	Prefrontal for control, sensory cortex for the trace
3 **Systems consolidation**	How a trace moves from hippocampus to neocortex
3 **Impairment by illness**	Which stage fails, and whether it persists
2 **What it changes**	Assessment and rehabilitation that follow

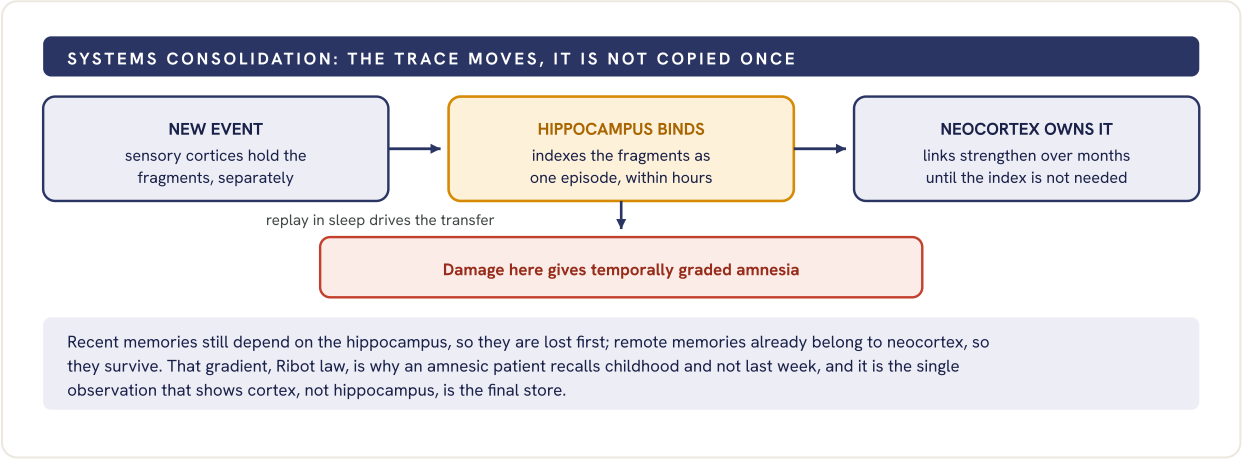


Fig 002.1 Consolidation drawn as a handover: cortex holds the fragments throughout, the hippocampus indexes them, and the index is gradually made unnecessary.

- **Which cortex does what.** Dorsolateral prefrontal cortex runs encoding strategy and retrieval search; ventrolateral prefrontal cortex selects among competitors; the sensory cortices are where the content itself is finally stored.
- **Impairment by illness.** Schizophrenia: verbal declarative and working memory deficits present from onset and largely independent of symptoms. Depression: effortful encoding fails with attention and motivation, plus overgeneral autobiographical recall. Bipolar disorder: deficits persist into euthymia.
- **The reversible causes, kept separate.** Delirium, benzodiazepines and alcohol, ECT, thiamine deficiency, hypothyroidism and dissociative amnesia. Naming which of these are treatable before describing the untreatable is the marked move.

WHAT EARNS THE MARKS

- **Cortex named as the final store,** with the hippocampus as a temporary index.
- **The temporal gradient, and what it proves.** It is the evidence, not just a clinical sign.
- **Deficits described by stage,** encoding against retrieval, rather than as poor memory.
- **Reversible causes listed separately** from the disorders themselves.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 23 hippocampal formation **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 03

Describe in brief neurobiology of sleep and wakefulness. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, KUHS, RGUHS, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3	**Ascending arousal system**	Two branches out of the brainstem, and their transmitters
2	**Sleep promoting system**	The preoptic switch, GABA and galanin
2	**Flip-flop switch**	Mutual inhibition, and what orexin does to it
2	**REM sleep**	The cholinergic and aminergic reciprocal model
1	**Drug correlates**	Each sedating drug named to the system it acts on

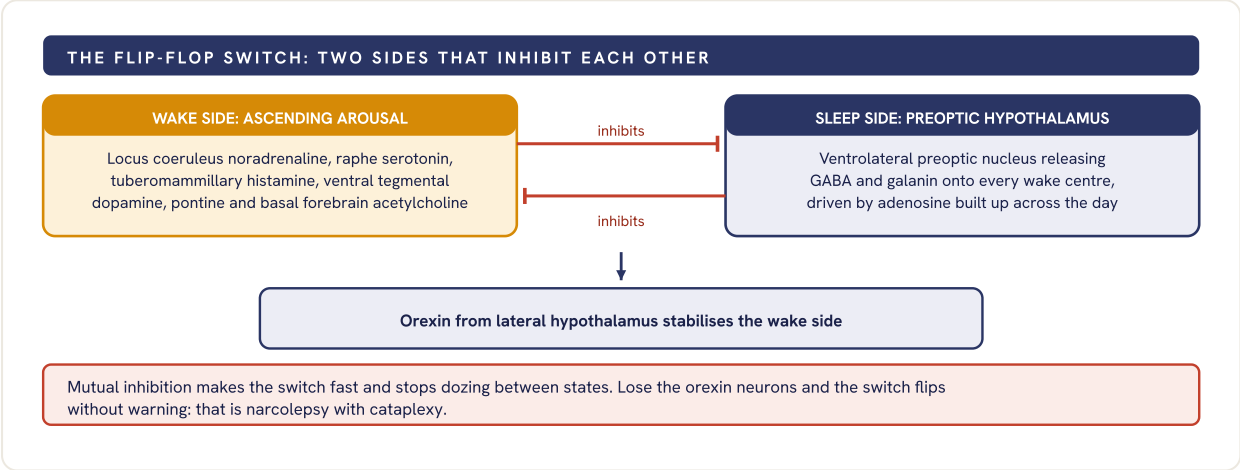


Fig 003.1 Wake and sleep drawn as two mutually inhibiting populations with orexin as the stabiliser, which is why the state changes abruptly rather than by degrees.

- **REM sleep.** The reciprocal interaction model: cholinergic pontine neurons fire to generate REM while noradrenergic and serotonergic neurons fall silent. Loss of that aminergic tone with active cholinergic drive gives the vivid dreams and the muscle atonia.
- **Two clocks, not one.** A homeostatic drive from accumulating adenosine, and a circadian drive from the suprachiasmatic nucleus signalling through melatonin. Sleep happens where the two align.

DRUG	WHAT IT ACTS ON	THE EFFECT THIS PREDICTS
Benzodiazepines, Z drugs	GABA-A receptor, the sleep side	Faster sleep onset, less slow wave sleep
Sedating antihistamines	Histamine, a wake transmitter	Sedation, and daytime hangover
Caffeine	Adenosine receptor blockade	Removes the homeostatic sleep signal
Melatonin agonists	MT1 and MT2, the circadian arm	Shifts timing rather than sedating

WHAT EARNS THE MARKS

- **Both sides of the switch, with transmitters attached.** A list of nuclei without transmitters is half the answer.
- **Mutual inhibition explained as a mechanism,** and orexin given its stabilising role.
- **Drugs mapped back onto the diagram,** which is what makes this a pharmacology answer too.

SEE ALSO Slot I - 09 polysomnography and sleep stages Slot I - 11 classification of sleep disorders **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 04

Describe the synthesis, mechanism of action and neuropathological role of nitric oxide. [3+3+4]

3 + 3 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Synthesis**	One substrate, three enzymes, and what triggers them
3	**Mechanism of action**	Why it breaks every rule of a classical transmitter
4	**Neuropathological role**	The same molecule as signal and as poison

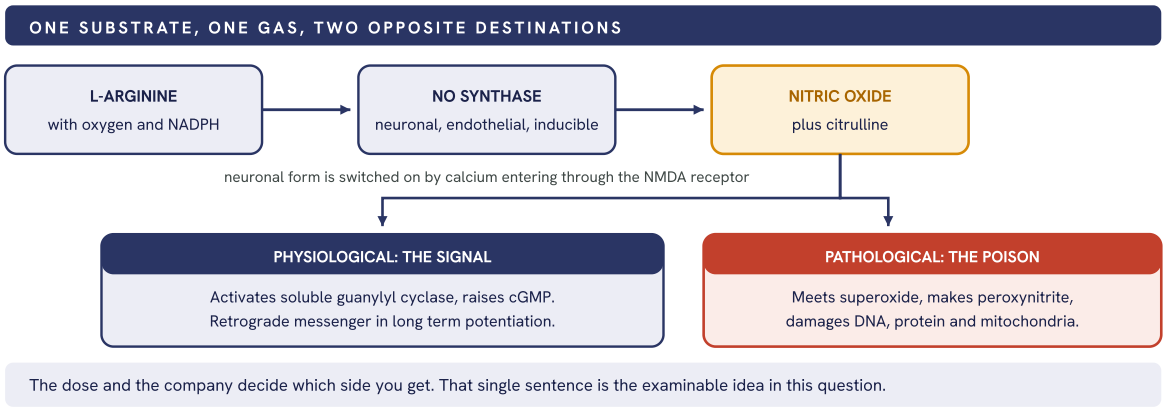


Fig 004.1 Nitric oxide drawn once and used twice: the same synthetic step feeds a signalling arm and an excitotoxic arm, and only the local conditions choose between them.

- **Why it is unusual.** A gas: not stored, not exocytosed, no membrane receptor. Made on demand, it crosses membranes freely and acts within microns for seconds, signalling backwards from the postsynaptic cell to the terminal that fed it.
- **Where it turns pathological.** Excess glutamate opens NMDA receptors, calcium floods in, neuronal NO synthase runs hard, and peroxynitrite forms. This is the final common path of excitotoxicity in stroke, neurodegeneration and, as an inducible form in microglia, in neuroinflammation.

THE PSYCHIATRIC HOOK

Nitric oxide signalling is downstream of the NMDA receptor, which is the same receptor implicated in the glutamate model of schizophrenia and targeted by ketamine. It is also the mediator of penile erection, which is why phosphodiesterase inhibitors treat one of the commonest antidepressant side effects.

WHAT EARNS THE MARKS

- **The synthetic step written out,** arginine with oxygen and NADPH to nitric oxide and citrulline, and all three synthase isoforms named.
- **The four ways it breaks transmitter rules.** Not stored, not exocytosed, no receptor, membrane permeable.
- **Signal and poison as one mechanism,** separated by amount and by the presence of superoxide.

SEE ALSO Slot I - 06 glutamate receptors and pathways Slot I - 13 synaptic plasticity

I - 05

Describe the electrophysiological abnormalities in the following conditions. a) Schizophrenia. b) Catatonia. c) Obsessive compulsive disorder. d) Delirium. [3+2+2+3]

3 + 2 + 2 + 3 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Schizophrenia	Evoked potentials and gating, not the resting record
2	Catatonia	Mostly to exclude something, and what that something is
2	Obsessive compulsive disorder	The error signal, and frontal rhythm
3	Delirium	The one diagnosis where EEG genuinely decides

CONDITION	RESTING EEG	EVOKED AND EVENT RELATED	WHAT IT IS USED FOR
Schizophrenia	Non-specific; no diagnostic pattern	Reduced P300 amplitude with delayed latency, P50 gating failure, reduced mismatch negativity, weak gamma response	Endophenotypes for research, not diagnosis
Catatonia	Normal, or diffuse non-specific slowing	Not diagnostically useful	To exclude non-convulsive status epilepticus
Obsessive compulsive disorder	Increased frontal beta in some series	Enhanced error related negativity, present before onset and in unaffected relatives	A candidate trait marker
Delirium	Diffuse slowing, loss of alpha, more theta and delta; triphasic waves in hepatic causes	Slowing tracks severity and settles as the patient clears	Separates delirium from a functional psychosis

Amber cells are the discriminators. Only one row on this page describes a test that changes what you do next, and saying which one is itself a mark.

- **What each measure means.** P50 gating asks whether the brain suppresses a repeated click; mismatch negativity, whether it notices an odd tone unattended; P300, whether it registered a rare target. All three are weak in schizophrenia and attention independent, which is why they survive as endophenotypes.
- **Delirium, in practice.** Slowing appears early and is graded, so a normal record in a confused patient argues against delirium and toward a functional cause.
- **The catatonia trap.** Catatonia and non-convulsive status epilepticus look alike at the bedside, and a benzodiazepine improves both. The EEG is what separates them, so it is ordered for exclusion rather than for confirmation.

WHAT EARNS THE MARKS

- **Resting EEG kept apart from evoked potentials.** Merging them is the commonest error on this question.
- **Each finding named with its purpose,** research marker or clinical decision.
- **Non-convulsive status epilepticus named under catatonia,** which is the safety point the question is testing.
- **Delirium identified as the one diagnostic use,** with the direction of the change stated.

SEE ALSO Slot I - 08 biological markers Slot I - 09 sleep stages and EEG **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 06

Describe the synthesis, receptors and major pathways of glutamate in brain. [3+3+4]

3 + 3 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Synthesis and disposal**	The glutamine cycle, and why the astrocyte is essential
3 **Receptors**	Three ionotropic, three metabotropic groups, with their jobs
4 **Pathways and relevance**	Four named tracts, then the psychiatric use

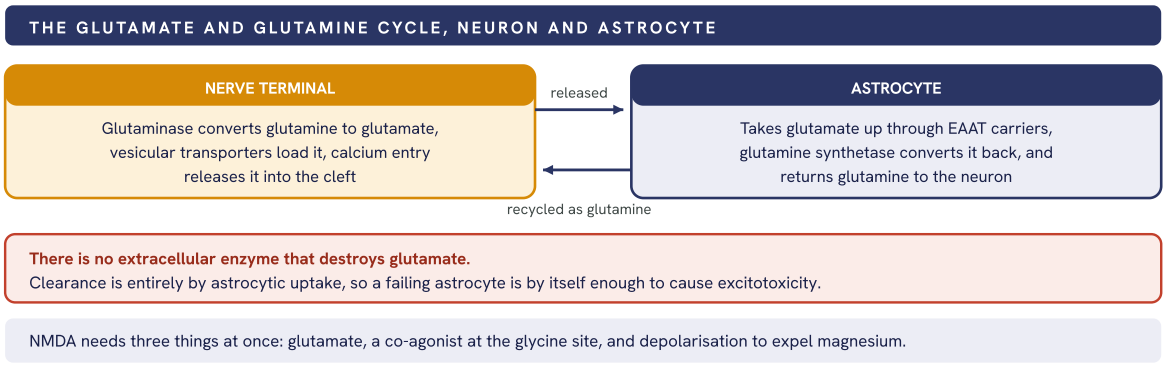


Fig 006.1 Synthesis and clearance as one loop between neuron and astrocyte, with the two facts that follow: no enzymatic breakdown, and a receptor that fires only on coincidence.

RECEPTOR	TYPE	WHAT IT DOES	DRUG THAT ACTS HERE
AMPA	Ionotropic, sodium	Fast excitation, moment to moment	Perampanel in epilepsy
NMDA	Ionotropic, calcium	Coincidence detection, plasticity	**Ketamine, memantine, alcohol**
Kainate	Ionotropic	Modulates release, some fast excitation	Topiramate in part
mGluR groups I to III	Metabotropic, G protein	Tunes gain rather than carrying the signal	Investigational only

- **Major pathways.** Corticocortical association fibres, the corticostriatal projection, reciprocal corticothalamic and thalamocortical fibres, and inside the temporal lobe the perforant path and Schaffer collaterals.
- **Why psychiatry cares.** Ketamine and phencyclidine block the NMDA receptor and reproduce positive, negative and cognitive symptoms in healthy volunteers, the strongest argument for the glutamate model of schizophrenia. The same receptor carries ketamine's rapid antidepressant effect.

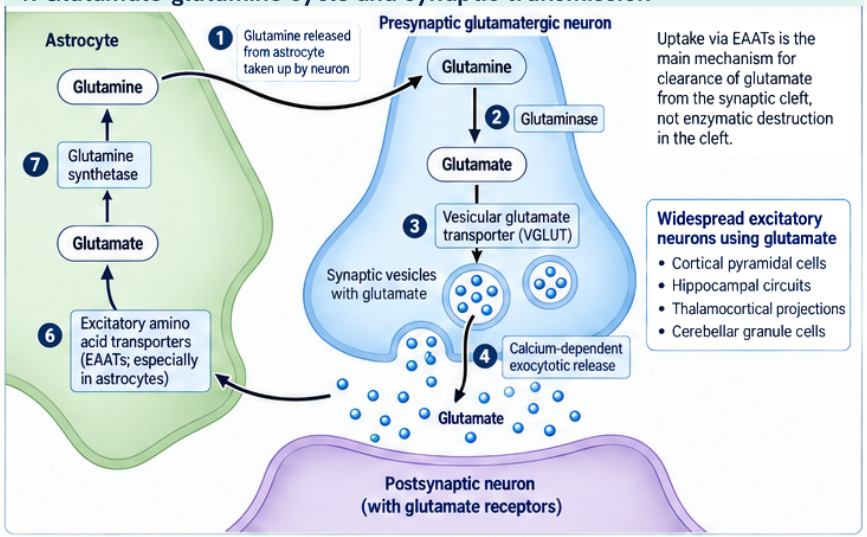
WHAT EARNS THE MARKS

- **The astrocyte named as the only clearance route.** It is the fact the whole excitotoxicity story rests on.
- **Ionotropic and metabotropic separated,** with all three ionotropic receptors named.
- **The three NMDA requirements listed together.** Glutamate, co-agonist, depolarisation.
- **Named pathways, not just named receptors,** since the question asks for both.

SEE ALSO Slot I - 04 nitric oxide Slot I - 13 synaptic plasticity

Glutamate

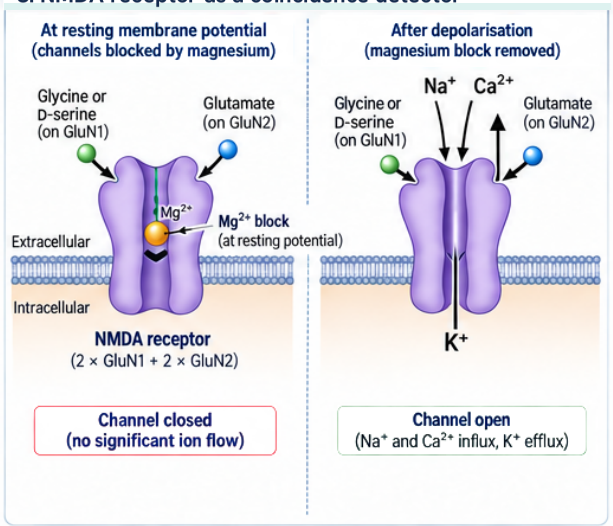
1. Glutamate-glutamine cycle and synaptic transmission



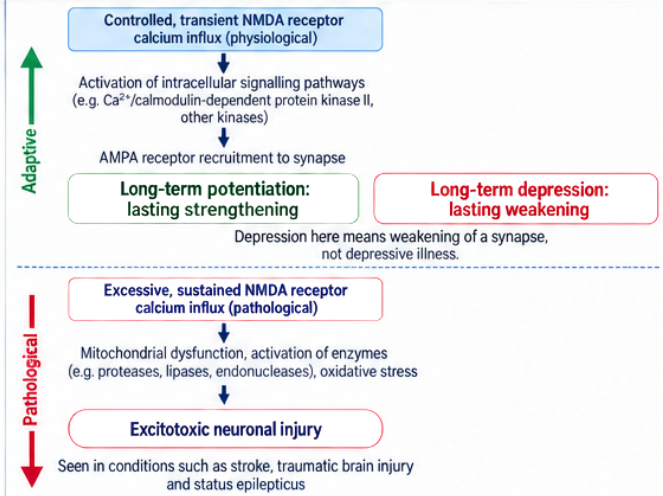
2. Glutamate receptors

Receptor (family)	Key properties	Signalling	Main locations / notes
Ionotropic receptors (tetrameric cation channels)			
Alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor	Fast excitatory synaptic transmission	Na^+ influx K^+ efflux	Widely distributed Major mediator of fast excitatory postsynaptic potential
Kainate receptor	Fast excitation; modulates neurotransmitter release	Na^+ influx K^+ efflux	Pre- and postsynaptic Modulates glutamate and GABA release
N-methyl-D-aspartate (NMDA) receptor	Slower kinetics; requires glutamate, glycine or D-serine, and depolarisation (removal of Mg^{2+} block)	Na^+ and Ca^{2+} influx K^+ efflux	Common structure: 2 × GluN1 + 2 × GluN2 Coagonist (glycine or D-serine) binds GluN1; glutamate binds GluN2
Metabotropic glutamate receptors (G protein-coupled receptors)			
Group I (mGlu1, mGlu5)	Typically postsynaptic	Gq → phospholipase C → inositol trisphosphate (IP_3) / diacylglycerol → ↑ Ca^{2+} signalling	Modulate synaptic plasticity
Group II (mGlu2, mGlu3)	Often presynaptic	Gi/o → ↓ cyclic adenosine monophosphate (CAMP)	Often reduce glutamate release (location dependent)
Group III (mGlu4, mGlu6, mGlu7, mGlu8)	Often presynaptic	Gi/o → ↓ cAMP	Often reduce glutamate release (location dependent)

3. NMDA receptor as a coincidence detector



4. From signalling to plasticity or injury



5. Glutamate and psychiatry

Schizophrenia (proposed mechanism)
NMDA receptor hypofunction, particularly on inhibitory interneurons, may lead to disinhibition of cortical pyramidal cells. This is a proposed model; there is not simply 'too little glutamate' globally.

Ketamine and esketamine (antidepressant)
NMDA receptor antagonists. Downstream AMPA signalling and brain-derived neurotrophic factor (BDNF) / mechanistic target of rapamycin (mTOR) mediated synaptic plasticity are proposed contributors to rapid antidepressant effects.

Memantine (Alzheimer disease)
Low-to-moderate affinity NMDA receptor antagonist.
Symptomatic treatment in moderate-to-severe Alzheimer disease.

Anti-NMDA receptor encephalitis
Autoantibodies (usually IgG) against the GluN1 subunit.
New psychiatric symptoms (e.g. psychosis), plus seizures, movement disorder, autonomic instability or reduced consciousness → consider autoimmune encephalitis. Cerebrospinal fluid antibody assessment is important.

Important clinical point
The effects of NMDA receptor antagonism can differ by dose, neural circuit and disease state (e.g. psychotomimetic at lower doses in healthy individuals, but potential antidepressant effects).

Glutamate. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-06.

Credits and references: retained within the illustration.

I - 07

Describe the biosynthesis, transmission and implications in psychiatric diagnosis of endocannabinoids. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Biosynthesis	Two ligands, made on demand from membrane lipid
3 Transmission	Backwards across the synapse, then broken down locally
5 Psychiatric implications	Cannabis and psychosis first, then the honest limits

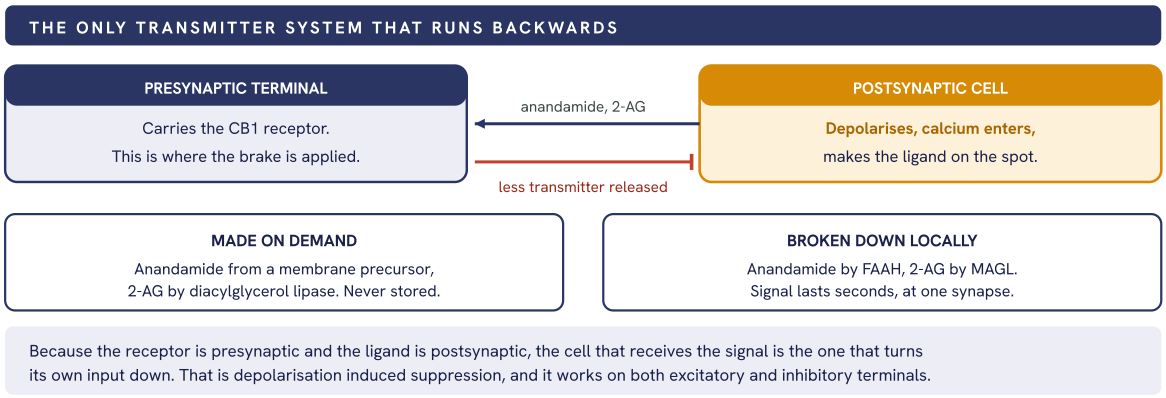


Fig 007.1 The endocannabinoid synapse: ligand made postsynaptically on demand, receptor sitting presynaptically, and a brake applied by the receiving cell onto its own input.

- **The two ligands and the two receptors.** Anandamide and 2-arachidonoylglycerol are the ligands, both derived from arachidonic acid in membrane phospholipid. CB1 is the brain receptor, dense in hippocampus, basal ganglia, cerebellum and cortex and sparse in the brainstem, which is why cannabis is not respiratory depressant. CB2 sits mainly on immune cells and microglia.
- **Cannabis and psychosis.** Tetrahydrocannabinol is a partial CB1 agonist and reproduces positive and cognitive symptoms in volunteers. Prospective cohorts show a dose dependent rise in later psychosis, larger for early and for high potency use. Cannabidiol is not psychotomimetic and has been studied as an antipsychotic.
- **Where the evidence stops.** No endocannabinoid measure is a diagnostic test. Raised anandamide in cerebrospinal fluid and altered CB1 binding are reported in schizophrenia but are not specific. Causation in the cannabis association is supported by dose response and by genetic studies, but reverse causation and confounding are not fully excluded.

WHAT EARNS THE MARKS

- **Retrograde signalling stated as the defining property,** with the receptor placed presynaptically.
- **Made on demand, not stored,** and each ligand paired with the enzyme that ends it.
- **The sparse brainstem CB1 density explained,** since it is why overdose does not kill.
- **Diagnosis answered honestly.** No test exists, and saying so with the reason earns the top of this band.

SEE ALSO Slot I - 06 glutamate receptors Slot I - 08 biological markers

I - 08

Discuss the biological markers of any two major psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Define and classify	State against trait, and the four properties a marker needs
3	Schizophrenia	Electrophysiological, imaging, ocular, biochemical
3	Depression	Endocrine, sleep, inflammatory
2	The verdict	Why none is yet a diagnostic test

DEFINE IT BEFORE YOU LIST ANYTHING

A biological marker is an objectively measured biological characteristic that indicates a normal process, a disease process, or a response to treatment. A state marker changes with the episode; a trait marker persists in remission and in unaffected relatives, which is what makes it an endophenotype.

DOMAIN	SCHIZOPHRENIA	DEPRESSION	STATE OR TRAIT
Electrophysiology	P50 gating failure, reduced P300 and mismatch negativity	Shortened REM latency, increased REM density	**Mostly trait**
Endocrine	Blunted growth hormone response to dopamine agonists	Dexamethasone non-suppression, raised cortisol	State
Imaging	Enlarged ventricles, reduced temporal grey matter	Reduced hippocampal volume with recurrence	**Trait for the first, cumulative for the second**
Ocular	Smooth pursuit eye movement abnormality	Not established	**Trait, present in relatives**
Inflammatory	Raised inflammatory markers in a subgroup	Raised interleukin 6 and C reactive protein	State
Neurotrophic	Reduced BDNF reported inconsistently	Low serum BDNF that rises with treatment	State

Amber cells are the trait markers. They are the ones that survive recovery and appear in well relatives, which is what makes them useful for genetics rather than for diagnosis.

- **The four properties a marker must have.** Sensitivity, specificity, reliability across labs, and independence from treatment. Almost every marker above fails at least one, most often specificity.
- **The verdict.** No biological marker is currently diagnostic in psychiatry. They separate groups, not individuals, and the distributions of ill and well people overlap. The working uses today are research stratification, endophenotypes for genetics, and monitoring rather than diagnosis.

WHAT EARNS THE MARKS

- **Two illnesses chosen and named at the top,** since the question allows a choice and expects you to make it.
- **State separated from trait,** applied to each marker rather than stated once.
- **Markers grouped by domain,** which keeps ten items readable as two answers.
- **The honest closing verdict.** Claiming a diagnostic test loses the last marks.

SEE ALSO Slot I - 05 electrophysiological abnormalities Slot I - 99 personalised medicine

I - 09

What is polysomnography? Briefly describe various stages of sleep along with their EEG findings. Discuss the neurobiology of sleep and wakefulness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Polysomnography	The three channels that stage sleep, then the rest
3 The stages	Each with its signature waveform and its share of the night
5 Neurobiology	The switch, the two drives, and the REM mechanism

STAGE	EEG SIGNATURE	EYES AND MUSCLE TONE	SHARE OF THE NIGHT
N1	Posterior alpha of wake drops out, low voltage mixed frequency, vertex sharp waves	Slow rolling eye movements	Roughly a twentieth
N2	**Sleep spindles and K complexes**	Eyes still, tone reduced	**About half**
N3	**Delta, under 4 hertz, high amplitude**	Eyes still, tone low	About a fifth, front loaded
REM	Low voltage mixed frequency, saw-tooth waves	**Rapid eye movements with atonia**	About a fifth, back loaded

- **What polysomnography is.** A multi-channel overnight recording. Three channels stage it: EEG, electro-oculogram, chin electromyogram; the rest measure breathing, oxygen, heart, legs and position.
- **Neurobiology.** Wake is held by the ascending arousal system: noradrenaline, serotonin, histamine, dopamine, acetylcholine. Sleep is imposed by the ventrolateral preoptic nucleus using GABA and galanin. The two inhibit each other; orexin stabilises wake, and adenosine with the suprachiasmatic clock sets timing.
- **REM, and what shortens its latency.** Pontine cholinergic neurons generate REM while aminergic firing falls silent, giving dreams with atonia. Short REM latency, increased REM density and reduced slow wave sleep is the classic depression profile. Sleep onset REM points to narcolepsy.



Fig 009.1 The architecture of one night, with the parasomnias placed in the half of the night whose sleep stage produces them.

WHAT EARNS THE MARKS

- **Three staging channels named,** EEG, eye movement and chin tone, before the rest of the montage.
- **Spindles and K complexes tied to N2,** delta to N3, sawtooth to REM.
- **Both drives named,** homeostatic adenosine and the circadian clock, not sleep pressure alone.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 11 classification of sleep disorders **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 10

What are the various type of the memory? Describe neurobiology of the memory. Describe rehabilitation of memory problems in chronic mental illness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The classification	Name the axis, then divide: awareness first, duration second
3 Neurobiology	Anatomy per branch, then the shared cellular mechanism
5 Rehabilitation	Restore, compensate, adapt, and what the evidence supports

BRANCH	SUBTYPE	WHAT IT HOLDS	ANATOMY
Declarative	Episodic	Events, with their time and place	**Hippocampus, medial temporal**
Declarative	Semantic	Facts, stripped of context	Anterolateral temporal cortex
Non-declarative	Procedural	Skills and habits	**Striatum, cerebellum**
Non-declarative	Priming	Easier processing after exposure	Neocortex
Non-declarative	Conditioning	Learned associations, including fear	Amygdala, cerebellum
By duration	Sensory, short term, working, long term	The second axis, cutting across the first	Prefrontal for working memory

The axis is awareness. Duration is a second, independent axis, so working memory is not a stage on the way to long term memory.

- **The shared mechanism.** Whatever the branch, the cellular event is the same: long term potentiation, made permanent by new protein synthesis and structural change at the spine.
- **Rehabilitation, three strategies.** Restorative training of the impaired function; compensatory training that routes around it; and environmental adaptation that removes the demand. In chronic mental illness the compensatory and adaptive routes carry most of the benefit.

WHAT THE EVIDENCE SUPPORTS IN CHRONIC ILLNESS

Errorless learning and spaced retrieval, leaning on intact procedural memory. External aids: alarms, pill boxes, written routines. Cognitive remediation improves cognition in schizophrenia. Treat the reversible contributors first: sedative load, anticholinergic burden, depression, poor sleep, thyroid and vitamin B12.

WHAT EARNS THE MARKS

- **The axis of division stated before the tree.** A list with no axis reads as recall.
- **Duration named as a second, independent axis,** not a deeper level of the first.
- **Errorless learning justified by the anatomy:** intact procedural carrying damaged declarative.
- **Reversible contributors named first.** Anticholinergic burden is most often missed.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 02 memory impairment in psychiatric illness **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 11

Classify sleep disorders. How would you manage a case of restless leg syndrome? How do you manage benzodiazepine dependence in a chronically mentally ill person? [2+4+4]

2 + 4 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Classification	Seven categories, named, with one example each
4	Restless legs	Confirm, correct the cause, then treat in order
4	Benzodiazepine taper	Assess, switch, taper slowly, support throughout

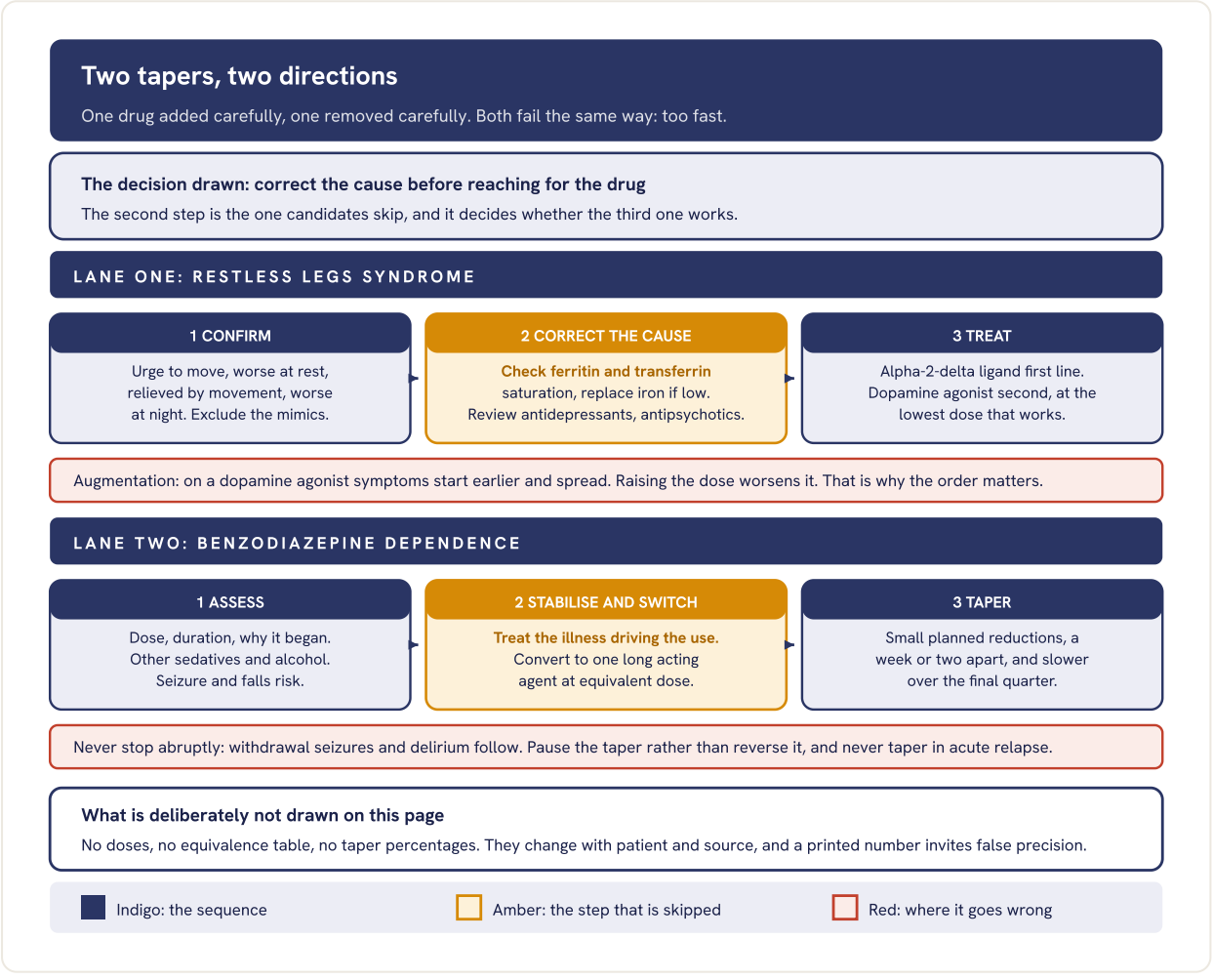


Fig 011.1 Both managements as three steps of the same shape, each with its failure mode below.

CATEGORY	ONE EXAMPLE EACH
Insomnia disorders	Chronic insomnia disorder
Sleep related breathing disorders	Obstructive sleep apnoea
Central disorders of hypersomnolence	Narcolepsy
Circadian rhythm sleep-wake disorders	Delayed sleep-wake phase disorder
Parasomnias	Sleepwalking, REM sleep behaviour disorder
Sleep related movement disorders	Restless legs syndrome

Six of the seven categories; the seventh is a residual other. Filing restless legs under movement disorders, not insomnia, is the classification mark.

- **The one that catches people out.** Antipsychotics and antidepressants can cause or worsen restless legs, and akathisia is the differential. Akathisia is generalised inner restlessness at any hour; restless legs is in the legs, at rest, in the evening, and eased by walking.

WHAT EARNS THE MARKS

- **Named categories,** with restless legs filed under movement disorders.
- **Iron studies before any drug,** and augmentation as the reason for the order.
- **The illness treated and a long acting agent substituted before tapering.**
- **Akathisia offered as the differential.** It is this question's discriminator.

SEE ALSO Slot I - 03 neurobiology of sleep Slot I - 09 polysomnography **SPRINT** Day 17, Eating and sleep-wake disorders

I - 12

Name various dopamine pathways of brain. Describe their significance in Psychiatry in detail. [3+7]

3 + 7 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, NTRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The four pathways	Origin and destination for each, named precisely
3	The blockade problem	One drug, four pathways, four different consequences
2	Receptors and synthesis	D1 and D2 families, and where the transmitter comes from
2	Beyond blockade	Salience, addiction, and partial agonism

PATHWAY	FROM, TO	WHAT IT NORMALLY DOES	WHAT BLOCKADE PRODUCES
Mesolimbic	Ventral tegmental area to nucleus accumbens	Assigns salience and reward value	**Positive symptoms settle; this is the therapeutic pathway**
Mesocortical	Ventral tegmental area to prefrontal cortex	Working memory, motivation, drive	**Negative and cognitive symptoms worsen**
Nigrostriatal	Substantia nigra pars compacta to dorsal striatum	Selects and releases movement	**Parkinsonism, dystonia, akathisia, tardive dyskinesia**
Tuberoinfundibular	Arcuate nucleus to anterior pituitary	Tonically inhibits prolactin release	**Hyperprolactinaemia, galactorrhoea, amenorrhoea, sexual dysfunction**

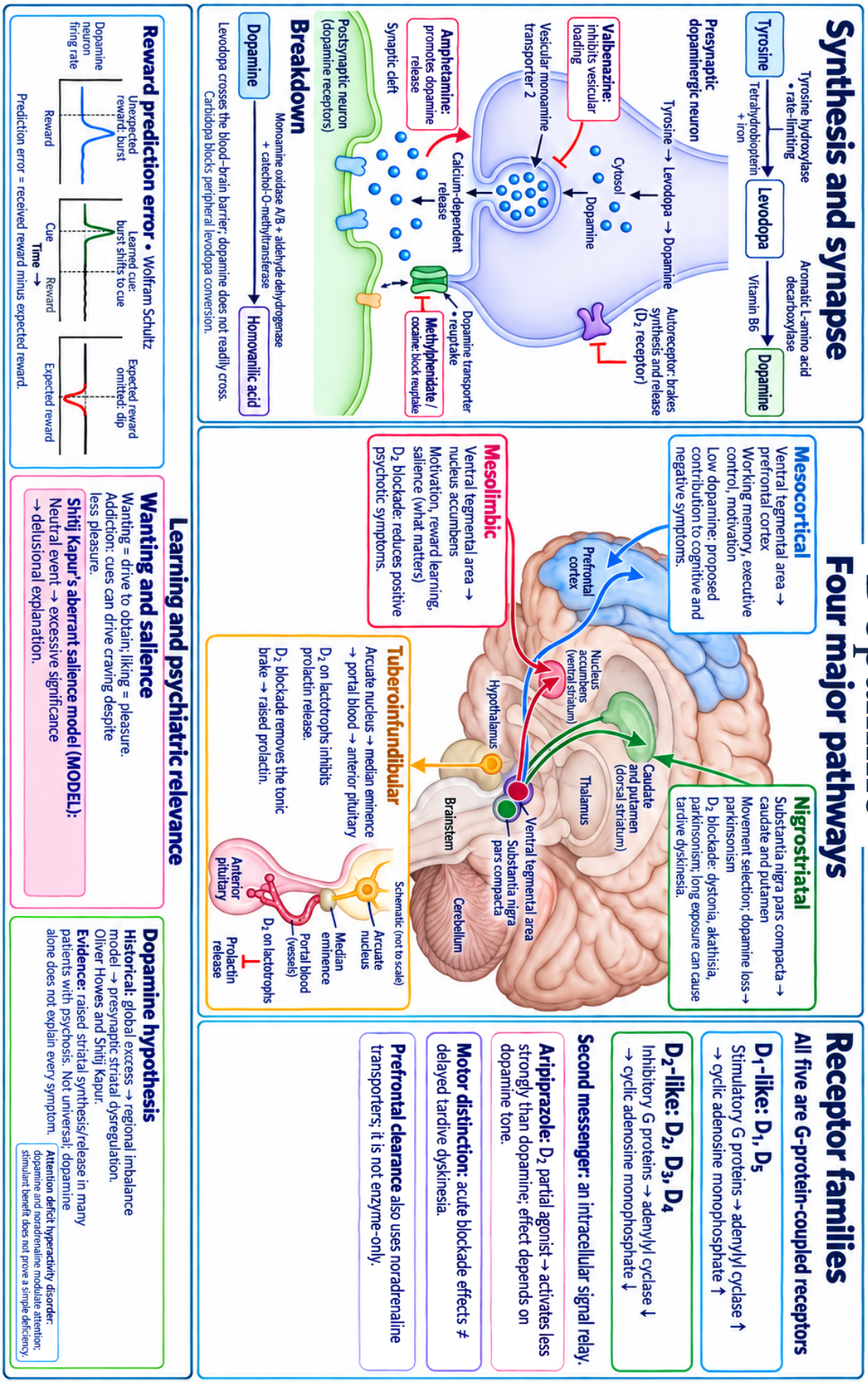
Every amber cell is produced by the same molecule at the same receptor. An antipsychotic cannot choose its pathway, which is why the side effect profile is predictable from this table alone.

- **Synthesis and receptors.** Tyrosine to DOPA by tyrosine hydroxylase, the rate limiting step, then to dopamine by DOPA decarboxylase. Receptors fall into a D1 family that stimulates adenylyl cyclase and a D2 family that inhibits it. Antipsychotic potency tracks D2 affinity.
- **Salience, not pleasure.** Mesolimbic dopamine marks what matters rather than what feels good. Aberrant salience gives ordinary events undue significance, which is a mechanism for how delusions form, and explains why blockade dampens conviction before it removes the belief.
- **Where the simple model fails.** Blockade is immediate but the antipsychotic response takes weeks, so receptor occupancy cannot be the whole story. Clozapine works best with relatively low D2 occupancy, and partial agonists such as aripiprazole stabilise rather than block, which is why they raise prolactin less.

WHAT EARNS THE MARKS

- **Four pathways with both ends named.** Mesolimbic and mesocortical share an origin, and saying so shows the pathways are understood.
- **One drug, four consequences,** laid out as a single table rather than as a side effect list.
- **Salience rather than pleasure** as the account of mesolimbic function.
- **The delay problem raised honestly.** It is the strongest evidence that blockade alone is insufficient.

SEE ALSO Slot I - 21 basal ganglia pathways Slot I - 97 pineal gland and prolactin rhythm



Dopamine. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-12.

Credits and references: retained within the illustration.

I - 13

Define synaptic plasticity. How is it important in psychiatry? [2+8]

2 + 8 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition and forms**	One sentence, then short term against long term, functional against structural
3 **The mechanism**	Induction, expression, and what makes it last
3 **Disorders**	Where plasticity is excessive, deficient, or misdirected
2 **Treatments**	Every effective treatment as a plasticity intervention

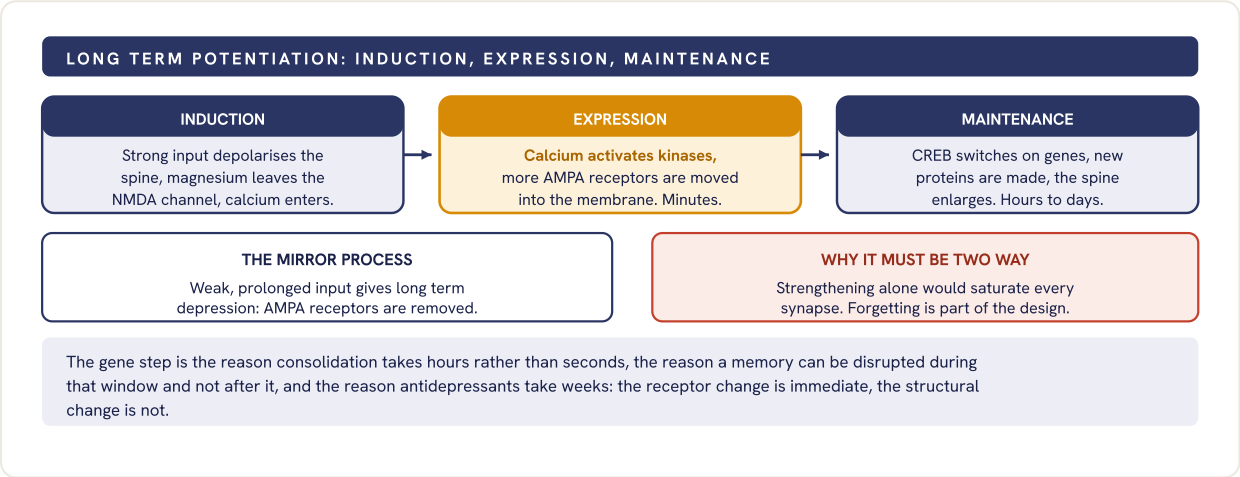


Fig 013.1 Potentiation in three stages with its mirror process alongside, and the gene step marked as the source of every clinically important delay.

- **The definition.** The capacity of a synapse to change the strength of its transmission in response to activity. It is functional when receptor number or release probability changes, and structural when spines and connections are added or lost.
- **Where it goes wrong.** Excessive synaptic pruning in adolescence, with complement C4 implicated, in schizophrenia. Stress and glucocorticoids retract dendrites in hippocampus and prefrontal cortex while the amygdala grows, in depression. Misdirected strengthening of reward circuits in addiction, and of fear circuits in post-traumatic stress.
- **Every treatment is a plasticity treatment.** Antidepressants raise BDNF signalling and restore spine density over weeks. Ketamine produces rapid synaptogenesis through mTOR. Electroconvulsive therapy and repetitive transcranial stimulation induce plasticity directly. Psychotherapy and exposure work by extinction learning, which is plasticity in prefrontal to amygdala pathways.

WHAT EARNS THE MARKS

- **A one sentence definition first,** since only two marks are on offer for it.
- **Potentiation and depression given as a pair.** Naming only strengthening misses why the system works.
- **The gene step used to explain a clinical delay.** That single link is the top band on this question.
- **Psychotherapy included among plasticity treatments,** not only the drugs.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 06 glutamate receptors

Classification of Neurotransmitters

1. Major chemical classes

Neurotransmitters

Small molecules (classical transmitters)

Amino acids
- Glutamate
- Gamma-aminobutyric acid (GABA)
- Glycine

Monoamines
- Catecholamines: dopamine, noradrenaline, adrenaline
- Indolamine: serotonin
- Histamine

Acetylcholine
(separate chemical class)

Purines
- Adenosine triphosphate (ATP)
- Adenosine

Peptides (neuropeptides)

- Substance P
- Neuropeptide Y
- Corticotropin-releasing hormone (CRH)
- Oxytocin
- Vasopressin
- Endogenous opioid peptides (e.g. endorphins, enkephalins, dynorphins)

Non-classical messengers

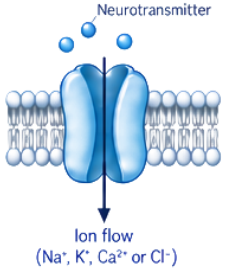
Gaseous signals
- Nitric oxide (NO)
- Other gas signals: carbon monoxide (CO), hydrogen sulfide (H_2S) (context-dependent evidence)

Endocannabinoids (lipid mediators)
- Anandamide (AEA)
- 2-arachidonoylglycerol (2-AG)

2. Classification by receptor type

Ionotropic receptors (ligand-gated ion channels)

Directly gated by neurotransmitter binding. Fast synaptic responses.

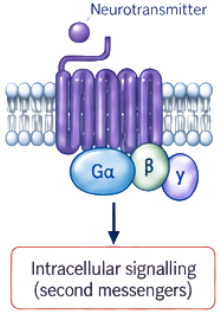


Examples
- AMPA and NMDA (glutamate)
- GABA-A
- Glycine
- Nicotinic acetylcholine
- Serotonin 5-HT_3
- P2X (ATP)

Exception: the serotonin 5-HT_3 receptor is ionotropic (despite serotonin usually acting at metabotropic receptors).

Metabotropic receptors (G-protein coupled receptors, GPCRs)

Activate intracellular signalling via G proteins. Slower, modulatory responses.



Examples
- Muscarinic acetylcholine
- GABA-B
- Dopamine (D1–D5)
- Serotonin receptors except 5-HT_3
- Metabotropic glutamate (mGluR)
- P2Y (ATP/ADP), P1 (adenosine)

3. Typical functional effects in the CNS

Neurotransmitter class	Main functional effect (in mature CNS)	Notes
Glutamate	Mainly excitatory	Principal excitatory transmitter in the CNS.
GABA	Mainly inhibitory	Principal inhibitory transmitter in the CNS.
Glycine	Mainly inhibitory	Important in spinal cord and brainstem.
Monoamines (dopamine, noradrenaline, adrenaline, serotonin, histamine)	Often modulatory	Can be excitatory or inhibitory depending on receptor subtype and circuit.
Acetylcholine	Often modulatory	Can be excitatory or inhibitory depending on receptor subtype and circuit.
Purines (ATP, adenosine)	Often modulatory	Effects depend on receptor subtype (P2X/P2Y/P1) and circuit.
Neuropeptides	Mostly modulatory	Typically longer-lasting effects.
Gaseous signals (NO, CO, H_2S)	Mostly modulatory	Diffuse, volume transmission; context-dependent evidence for some.
Endocannabinoids (anandamide, 2-AG)	Mostly modulatory	Usually act retrogradely to modulate transmitter release.

The actual effect of any neurotransmitter depends on the receptor subtype, ion gradients and the neural circuit in which it acts.

4. Synthesis, storage and release: comparison

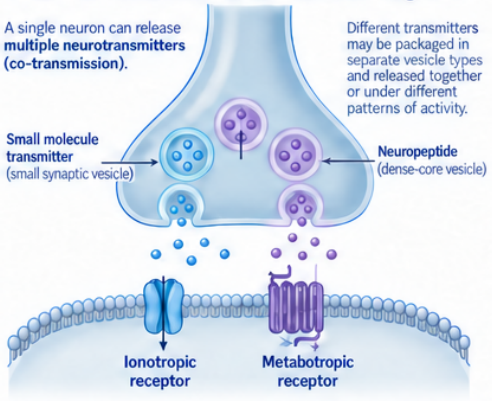
Feature	Small molecules (e.g. glutamate, GABA, monoamines, ACh, purines)	Neuropeptides (e.g. substance P, NPY, CRH, oxytocin, vasopressin, endogenous opioids)	Gases and endocannabinoids (e.g. NO, CO, H_2S, anandamide, 2-AG)
Site of synthesis	Usually in nerve terminal from precursors	Synthesized in cell soma as larger precursors (prepropeptides)	Often on-demand in neurons (e.g. NO) or from membrane lipids (endocannabinoids)
Storage	Packaged into small synaptic vesicles	Stored in large dense-core vesicles	Not stored in vesicles
Release mechanism	Calcium-dependent exocytosis	Calcium-dependent exocytosis (often promoted by higher-frequency activity)	Usually synthesized and released on-demand; non-vesicular (diffuse across membrane)
Termination/ inactivation	Reuptake transporters and enzymatic breakdown	Extracellular peptidases degrade peptides	Diffusion and chemical/ metabolic removal; endocannabinoids degraded by enzymes (FAAH for anandamide, MAGL for 2-AG)
Duration of action	Typically fast (milliseconds)	Often longer-lasting (seconds to minutes)	Variable; often longer and can diffuse beyond synapse (volume transmission)

These are typical features; there is heterogeneity within each class.

5. Co-transmission and related signals

A single neuron can release **multiple neurotransmitters (co-transmission).**

Different transmitters may be packaged in separate vesicle types and released together or under different patterns of activity.



Neurotransmitter identity is not fixed: there is no one-neuron-one-transmitter rule.

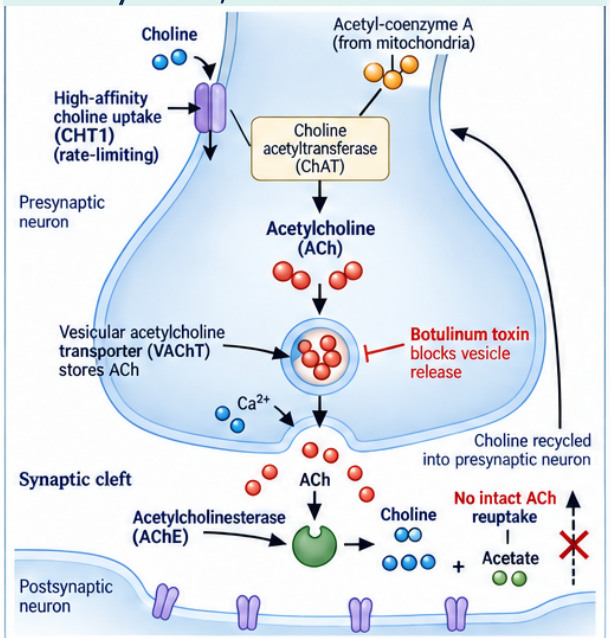
Neurotrophins (e.g. brain-derived neurotrophic factor) are growth-factor signals important for development, plasticity and survival, and are not classified as classical fast neurotransmitters.

Classification of Neurotransmitters. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

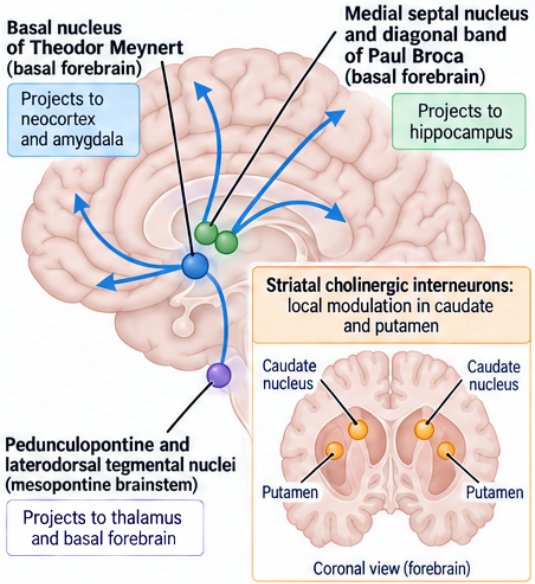
Credits and references: retained within the illustration.

Acetylcholine

Synthesis, release and breakdown



Major Cholinergic Pathways in the Brain



Acetylcholine Receptors

Nicotinic acetylcholine receptors (nAChRs)

Pentameric ligand-gated cation channels (Na^+, K^+, Ca^{2+}). Fast excitation.

Subtype	Key locations	Features
Neuronal α4β2	Ventral tegmental area, nucleus accumbens (reward pathway)	Involved in nicotine reward. **Varenicline** is a partial agonist at α4β2.
Neuronal α7	Widely in brain (e.g. cortex, hippocampus)	Highly permeable to Ca^{2+}. Implicated in sensory gating (research area).
Muscle type (α1β1δε)	Neuromuscular junction (skeletal muscle)	Mediates end-plate potentials and muscle contraction.

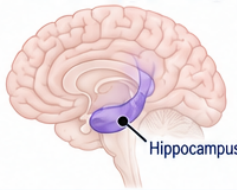
Muscarinic acetylcholine receptors (mAChRs)

G protein-coupled receptors (GPCRs).

Subtype	G protein → signalling	Main locations	Key effects
M1	G_q → phospholipase C → ↑ intracellular Ca^{2+}	Cortex, hippocampus	Cognition, excitability
M2	$G_{i/o}$ → ↓ cyclic adenosine monophosphate (cAMP)	Heart, presynaptic (autoreceptor)	↓ Heart rate, ↓ ACh release
M3	G_q → phospholipase C → ↑ intracellular Ca^{2+}	Exocrine glands, smooth muscle	Secretion, smooth muscle contraction
M4	$G_{i/o}$ → ↓ cAMP	Striatum	Modulates dopamine signalling
M5	G_q → phospholipase C → ↑ intracellular Ca^{2+}	Midbrain dopamine neurons, vasculature	Excitability, vasodilation

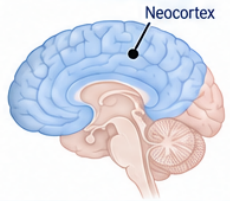
Physiological Functions

Attention and new-memory encoding



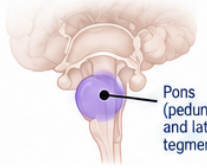
Cholinergic signalling in the cortex and hippocampus supports attentional processes and encoding of new memories (e.g. declarative memory).

Cortical arousal



Basal forebrain cholinergic input promotes cortical arousal and wakefulness.

Rapid eye movement (REM) sleep



Cholinergic neurons in the mesopontine brainstem contribute to REM sleep. The John Allan Hobson–Robert William McCarley reciprocal-interaction model is a classical model; modern sleep circuits are more complex:

Clinical Relevance

Condition / Context	Key points	Clinical effects
Alzheimer disease	Degeneration of basal forebrain cholinergic neurons (nucleus of Theodor Meynert) contributes to cognitive deficits.	Donepezil and galantamine are acetylcholinesterase inhibitors. Rivastigmine also inhibits butyrylcholinesterase. Provide symptomatic benefit, not disease modification.
Antimuscarinics (e.g. benztropine, procyclidine)	May treat drug-induced parkinsonism and acute dystonia (by reducing relative cholinergic excess).	Can impair cognition, cause delirium, dry mouth, constipation and urinary retention. Not a routine treatment for tardive dyskinesia.
Xanomeline–trospium	Xanomeline: central muscarinic agonism (M1/M4 implicated). Trospium: largely peripheral muscarinic antagonist to reduce peripheral adverse effects.	Approved for schizophrenia. Detailed antipsychotic mechanism is not fully established.

Exam distinction: Smoking and cytochrome P450 1A2

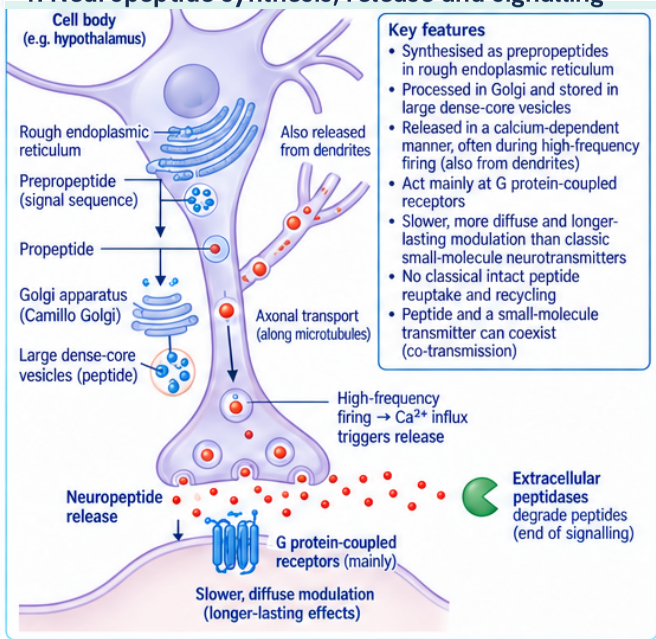
Polycyclic aromatic hydrocarbons **(not nicotine)** in tobacco smoke induce cytochrome P450 1A2. Stopping smoking can increase plasma levels of clozapine and olanzapine.

Acetylcholine. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Neuropeptides

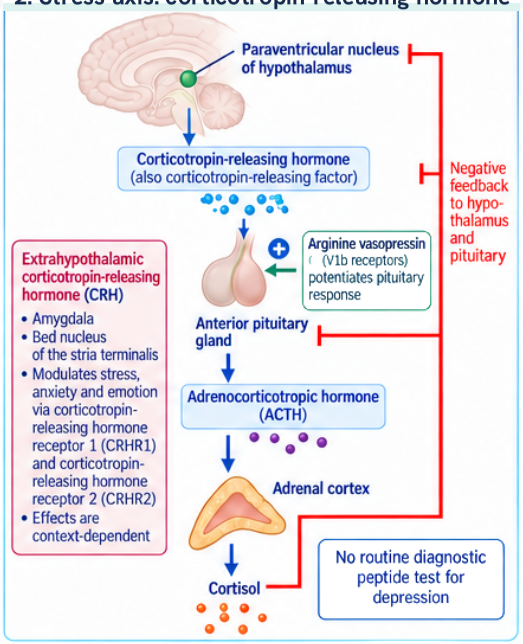
1. Neuropeptide synthesis, release and signalling



Key features

- Synthesised as prepropeptides in rough endoplasmic reticulum
- Processed in Golgi and stored in large dense-core vesicles
- Released in a calcium-dependent manner, often during high-frequency firing (also from dendrites)
- Act mainly at G protein-coupled receptors
- Slower, more diffuse and longer-lasting modulation than classic small-molecule neurotransmitters
- No classical intact peptide reuptake and recycling
- Peptide and a small-molecule transmitter can coexist (co-transmission)

2. Stress axis: corticotropin-releasing hormone

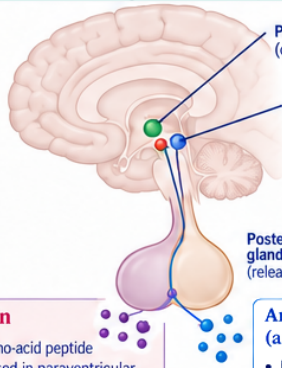


Extrahypothalamic corticotropin-releasing hormone (CRH)

- Amygdala
- Bed nucleus of the stria terminalis
- Modulates stress, anxiety and emotion via corticotropin-releasing hormone receptor 1 (CRHR1) and corticotropin-releasing hormone receptor 2 (CRHR2)
- Effects are context-dependent

No routine diagnostic peptide test for depression

3. Oxytocin and arginine vasopressin



Oxytocin

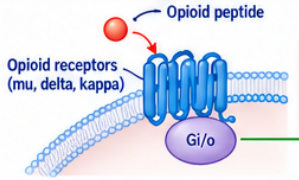
- Nine-amino-acid peptide
- Synthesised in paraventricular and supraoptic nuclei
- Released from posterior pituitary (also acts in brain centrally)
- **Oxytocin receptor (OXTR)**
  - Uterine contractions (labour)
  - Milk ejection (let-down)
  - Context-dependent social processing (e.g. bonding, social salience)
- Not a universal 'trust hormone'
- Not an established treatment for autism spectrum disorder

Arginine vasopressin (antidiuretic hormone)

- Nine-amino-acid peptide
- Synthesised in paraventricular and supraoptic nuclei
- Released from posterior pituitary (also acts in brain centrally)
- Receptors and key effects:
  - V1a receptors: social behaviour and vascular effects
  - V1b receptors: potentiates stress axis (ACTH release)
  - V2 receptors: renal water conservation (antidiuretic)

4. Opioid peptide precursors and receptors

Precursor	Peptide(s)	Preferred receptor affinity (not exclusive)	Key effects
Proopiomelanocortin (POMC)	β-endorphin	Mu (μ) and delta (δ)	Analgesia, reward modulation, stress response
Proenkephalin	Enkephalins (Met-enkephalin, Leu-enkephalin)	Delta (δ) and mu (μ)	Analgesia, modulation of mood and stress
Prodynorphin	Dynorphins (e.g. dynorphin A)	Kappa (κ)	Analgesia; often associated with aversion, dysphoria and stress responses



↓ Voltage-gated calcium channel activity (reduced Ca^{2+} entry)

Potassium conductance increases; potassium exits

Clinical points

- Analgesia and reward modulation (e.g. mesolimbic pathways)
- Kappa receptor activation is often associated with aversion/dysphoria
- **Naloxone** for opioid overdose reversal (non-selective antagonist)
- **Naltrexone** for alcohol use disorder and opioid use disorder (longer-acting antagonist)
- Receptor pairings are preferences, not absolute

5. Other high-yield neuropeptides

Neuropeptide	Main receptor(s)	Key functions (selected)	Clinical notes
Substance P	Neurokinin 1 (NK1) receptor	Pain transmission, emesis (vomiting), neurogenic inflammation	Aprepitant (NK1 antagonist) used as an antiemetic. Not an established antidepressant.
Neuropeptide Y (NPY)	Y receptors (e.g. Y1, Y2, Y5)	Feeding behaviour, stress resilience, anxiety (context-dependent effects)	Research interest in stress-related disorders. Complex, context–deependent effects.
Cholecystokinin (CCK)	CCK2 receptor (CCK-B)	Satiety, anxiety and panic signalling	CCK4 (tetrapeptide) can induce panic-like symptoms experimentally (research tool).
Galanin	Galanin 1, 2 and 3 receptors (Gal1–3)	Modulates monoamine neurotransmission, stress responses, cognition	Research interest in mood and anxiety disorders.
Orexin A and B (hypocretins)	Orexin 1 and 2 receptors (OX1R, OX2R)	Produced in lateral hypothalamus. Stabilises wakefulness, arousal and energy balance.	Loss of orexin neurons associated with narcolepsy type 1. Suvorexant, lemborexant and daridorexant are dual orexin receptor antagonists for insomnia.

Important final point: Plausible mechanisms from animal studies do not necessarily translate into proven human treatments. Poor blood–brain barrier penetration and peptide degradation complicate the development of neuropeptide-based drugs.

Neuropeptides. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Opiates and the Endogenous Opioid System

1. Terminology and sources

Opiates

Naturally occurring opium alkaloids from the opium poppy (*Papaver somniferum*) such as morphine and codeine.



Papaver somniferum

Opioids

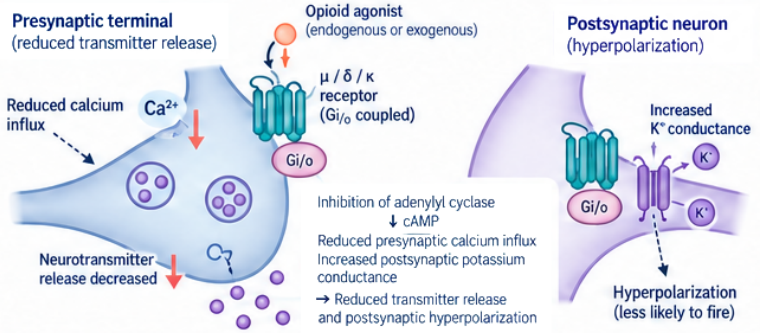
Broader group including endogenous peptides, semisynthetic and synthetic agents, as well as naturally occurring opiates.

Examples of opioid drugs

- Morphine and methadone (mu agonists)
- Buprenorphine (high-affinity partial mu agonist)
- Naloxone (rapid antagonist for overdose reversal)
- Naltrexone (longer-acting antagonist for relapse-prevention contexts)

2. Endogenous opioid peptides and receptors

Precursor (gene product)	Endogenous peptide(s)	Preferred receptor(s)	Key points
Proopiomelanocortin (POMC)	Beta-endorphin	Mu (μ) and delta (δ)	Higher preference, not exclusive
Proenkephalin	Met-enkephalin Leu-enkephalin	Delta (δ) and mu (μ)	Higher preference, not exclusive
Prodynorphin	Dynorphins	Kappa (κ)	Higher preference, not exclusive
Pronociceptin	Nociceptin (orphanin FQ)	NOP (nociceptin) receptor	Related to opioid system but distinct receptor

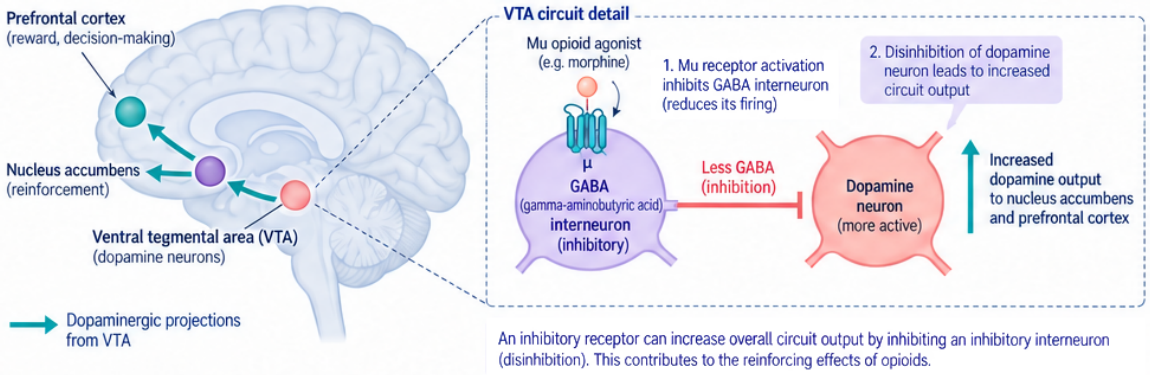


3. Receptor subtypes and functions

Receptor	Key functions (established)	Notes
Mu (μ)	• Analgesia • Reward and reinforcement • Respiratory depression	Major target for therapeutic opioids (e.g. morphine, methadone)
Kappa (κ)	• Analgesia • Stress and aversive effects (e.g. dysphoria)	May counteract rewarding effects at mu receptors
Delta (δ)	• Analgesia • Mood-related modulation	Involved in affective regulation
NOP (nociceptin)	• Modulates pain, stress and reward (research model)	Related to, but distinct from, classical opioid receptors

All classical opioid receptors (mu, delta and kappa) are $G_{i/o}$ protein-coupled receptors that inhibit adenylyl cyclase, reduce presynaptic calcium influx and increase postsynaptic potassium conductance, leading to reduced transmitter release and neuronal hyperpolarization.

4. Reward circuit: how an inhibitory receptor can increase output



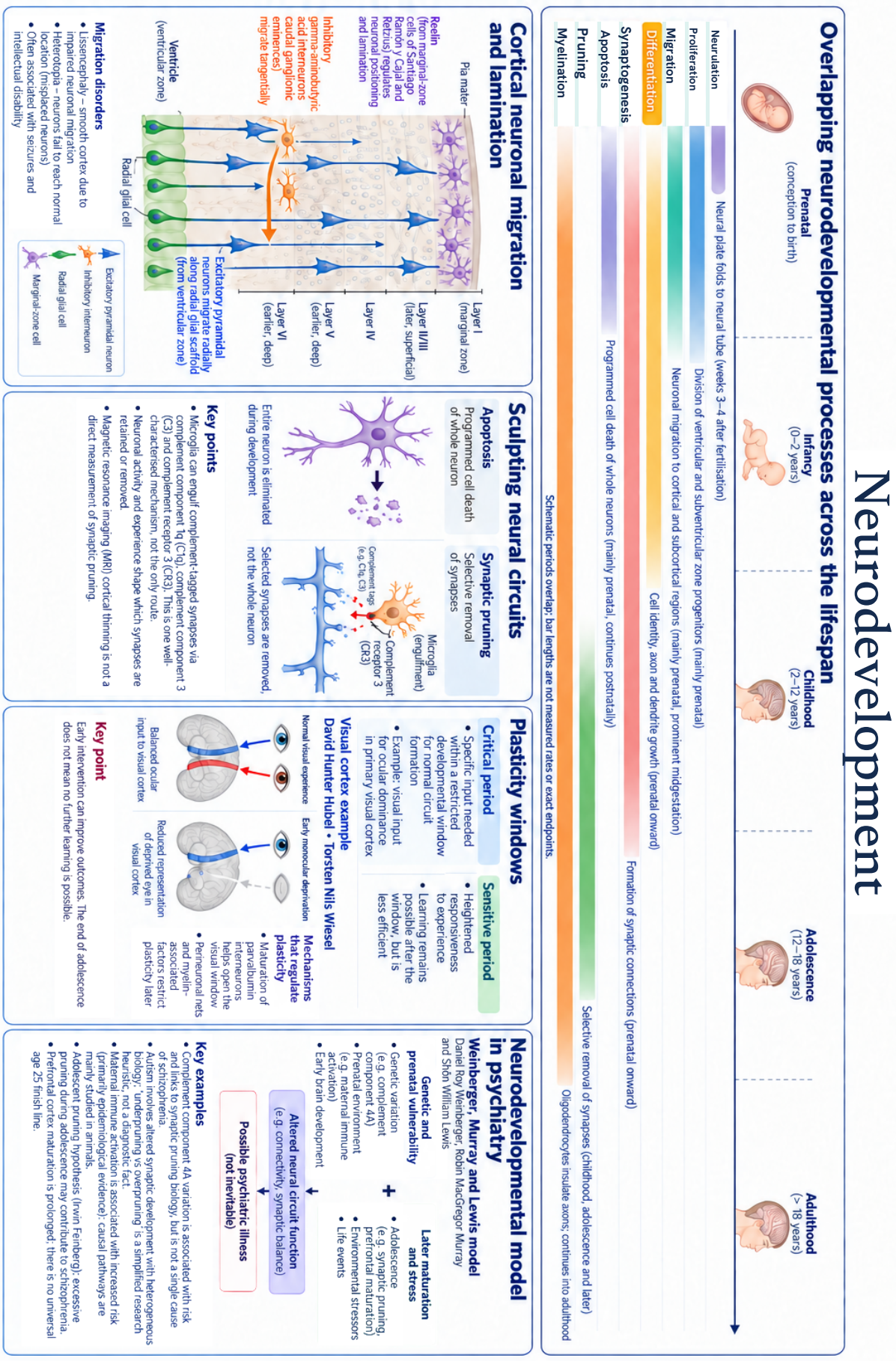
An inhibitory receptor can increase overall circuit output by inhibiting an inhibitory interneuron (disinhibition). This contributes to the reinforcing effects of opioids.

5. Clinical considerations

Topic	Key points
Drug actions	• Morphine and methadone: mu agonists • Buprenorphine: high-affinity partial mu agonist (with a ceiling to some effects, but risk of respiratory depression not eliminated) • Naloxone: rapid antagonist for overdose reversal • Naltrexone: longer-acting antagonist for relapse-prevention contexts
Tolerance, dependence and opioid use disorder	• Tolerance and physical dependence are not identical to opioid use disorder. • Withdrawal (a physiological state) and compulsive use (a behavioural pattern) are different concepts.
Respiratory depression	• Risk increases with concomitant central nervous system (CNS) depressants (e.g. alcohol, benzodiazepines). • Partial agonists (e.g. buprenorphine) have a ceiling to some effects, but this does not eliminate the risk.

Opiates and the Endogenous Opioid System. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.



Neurodevelopment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Neurotrophins

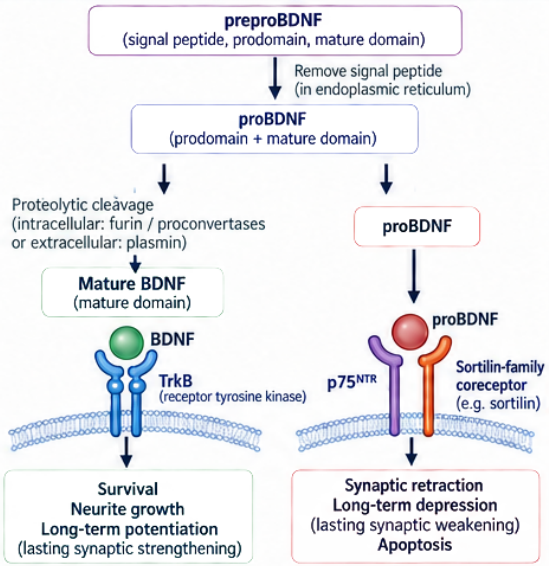
1. Neurotrophin family and receptors

Neurotrophin	Preferred receptor (full name)	Key functions and sites
Nerve growth factor (NGF)	Tropomyosin receptor kinase A **(TrkA)**	Supports sympathetic and sensory neurons. Maintains basal forebrain cholinergic neurons (relevant to Alzheimer disease).
Brain-derived neurotrophic factor (BDNF)	Tropomyosin receptor kinase B **(TrkB)**	Synaptic plasticity, learning and memory.
Neurotrophin 4/5 (NT-4/5)	Tropomyosin receptor kinase B **(TrkB)**	Synaptic plasticity, particularly in corticostriatal circuits.
Neurotrophin 3 (NT-3)	Tropomyosin receptor kinase C **(TrkC)**	Neuronal differentiation and survival.

All classical neurotrophins (NGF, BDNF, NT-3, NT-4/5) can also interact with the p75 neurotrophin receptor (p75NTR), a member of the tumour necrosis factor receptor family.

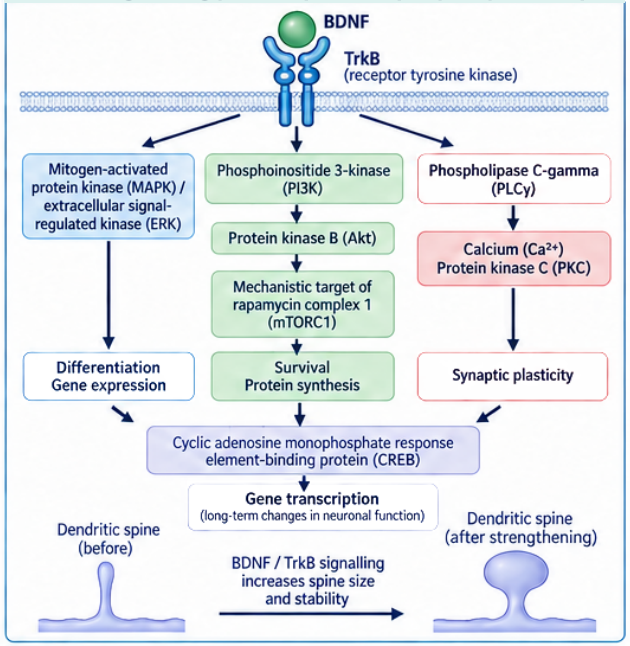
Discovery: Nerve growth factor was discovered by Rita Levi-Montalcini and Stanley Cohen in the 1950s, establishing the neurotrophin field.

2. BDNF precursor processing and divergent effects

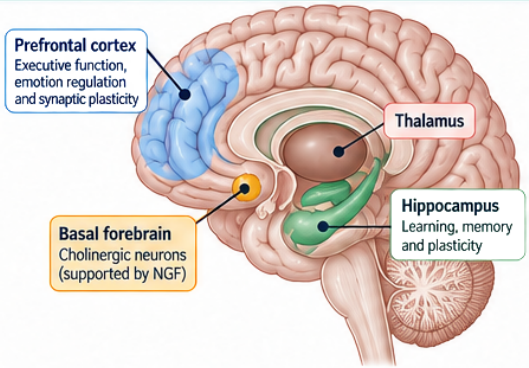


The cellular context, receptor combination and downstream signalling determine the outcome. p75NTR is not always a death switch.

3. TrkB signalling pathways and synaptic plasticity



4. Brain regions, functions and relevance to depression



Activity-dependent release

Neurotrophins such as BDNF are released from active neurons and strengthen or maintain selected synapses, supporting learning and memory and helping to refine neural circuits.

Neurotrophic hypothesis of depression

Chronic stress can reduce trophic support and dendritic complexity in some brain circuits, particularly the hippocampus and prefrontal cortex. Treatments (e.g. antidepressants, exercise) can increase BDNF signalling and plasticity.

Important caveats

- Strong evidence in animal models.
- Effects are region- and context-dependent in humans.
- There is no universal finding that stress always lowers BDNF across all brain regions or in all individuals.

5. Clinical and exam-relevant points



BDNF Val66Met polymorphism

- Val66Met = valine → methionine substitution at codon 66 in the BDNF PRODOMAIN (affects intracellular trafficking and activity-dependent secretion).
- Mature BDNF protein sequence is unchanged.
- Not a routine test to select psychotherapy or drugs.



Serum or plasma BDNF

- Does not reflect brain BDNF levels.
- Affected by platelets, physical activity, circadian factors and sample handling.
- Not a diagnostic test for depression.



Ketamine and neurotrophins

- Ketamine can increase downstream BDNF/TrkB signalling and protein synthesis (e.g. via mTORC1).
- This is a proposed mechanism, not a proven or exclusive mechanism of its antidepressant effect.



NGF and Alzheimer disease

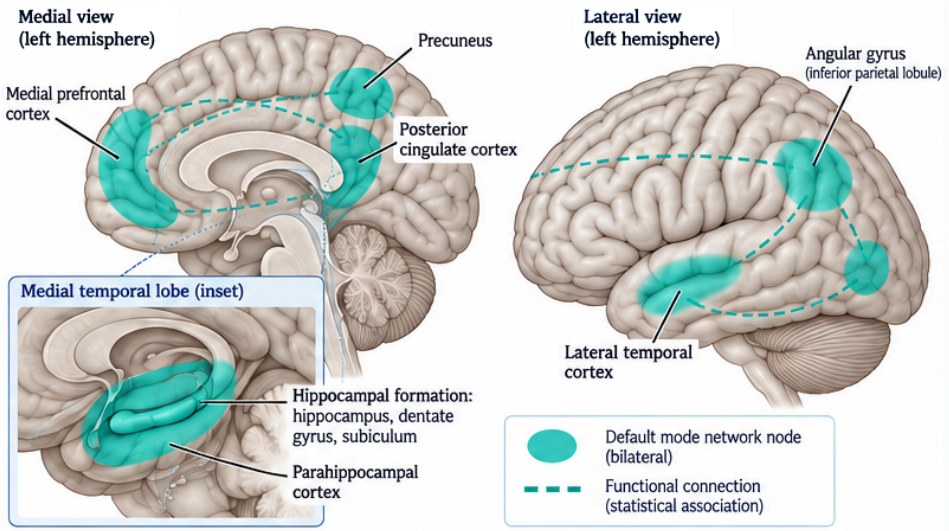
- NGF supports basal forebrain cholinergic neurons, which are vulnerable in Alzheimer disease.
- Trophic support and neurotrophin replacement strategies are an active area of research but are not an established treatment.

Neurotrophins. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Default mode network

1. Anatomy and network

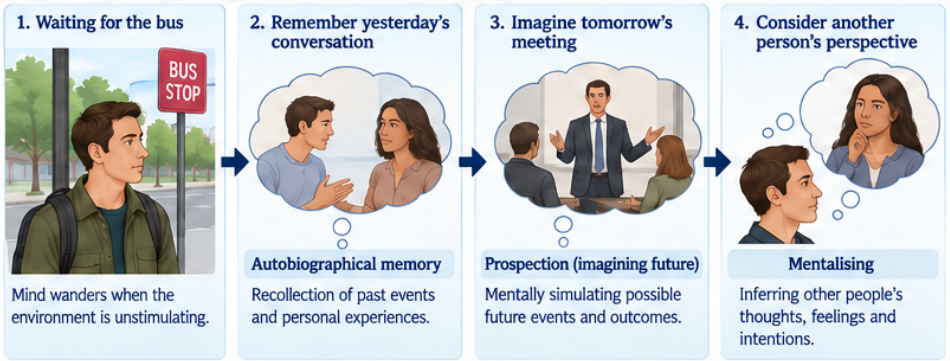


2. Nodes and functions

Brain region (anatomical name)	Key functions
Medial prefrontal cortex (mPFC)	Self-relevance, valuation, and integration of personal information.
Posterior cingulate cortex (PCC) and precuneus	Integrate internal information, support self-referential thought and scene construction.
Angular gyrus (inferior parietal lobule)	Semantic and episodic integration, linking memories, concepts and perspectives.
Hippocampal formation (hippocampus, dentate gyrus, subiculum) and parahippocampal cortex	Memory-based scene construction and future imagination (prospection).
Lateral temporal cortex	Conceptual knowledge, social knowledge and representation of other people.

Subsystems overlap and interact; there is no single node that "stores the self".

3. Everyday sequence: internally directed thought



Self-reflection, memory, imagination and consideration of others are everyday functions of the default mode network. Internally directed thought can be useful and goal-directed, not just mind-wandering.

4. Discovery and task relation

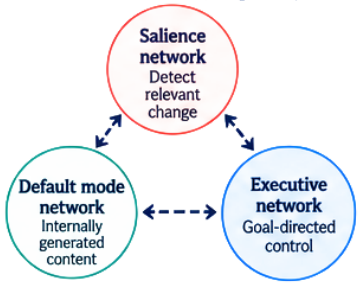
Marcus Raichle and colleagues (2001): positron emission tomography studies characterised a default mode of brain function; later functional magnetic resonance imaging mapped connectivity.

- Historically termed a "task-negative" network.
- The network remains active; it is not an idle or off switch.
- Can cooperate with frontoparietal control networks during autobiographical planning.
- There is no requirement for rest to show maximal activity everywhere.

External task
Activity often decreases relative to baseline.

Internal planning
Can cooperate with executive control.

Network interactions (simplified)



Networks can cooperate; they are not permanently opposed.

5. Relevance to psychiatry

Condition	Findings (group-level patterns)
Depression	Altered default-mode/subgenual anterior cingulate cortex coupling associated with rumination in some studies.
Attention deficit hyperactivity disorder	Default-mode interference proposed as a contributor to task lapses (difficulty disengaging).
Schizophrenia	Altered within- and between-network organisation and task disengagement.
Alzheimer disease	Posterior default-network connectivity and metabolism affected.

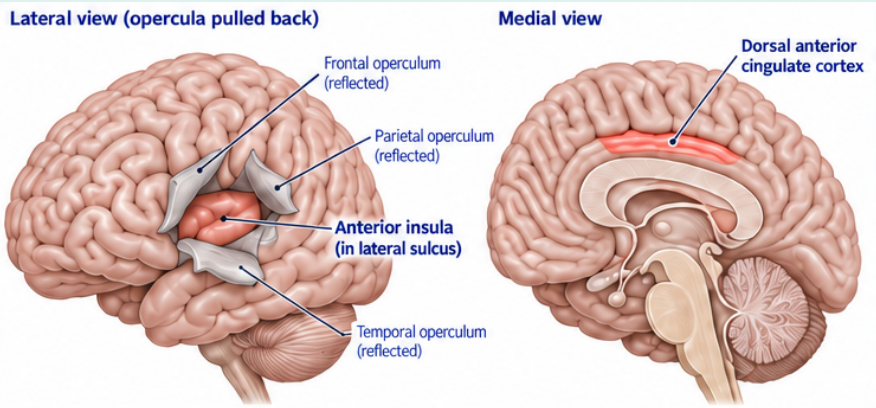
Group-level patterns are heterogeneous and not diagnostic for individuals. Rumination means repetitive, negatively biased thought, and is not a synonym for all default mode network activity.

Default mode network. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

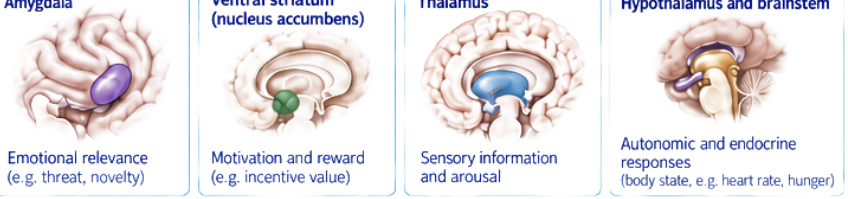
Credits and references: retained within the illustration.

Salience network

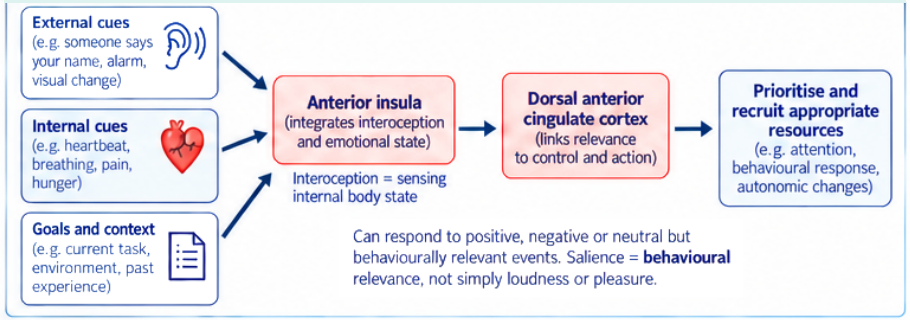
1. Key anatomy and connected regions



Functionally connected partner regions (not exhaustive)



2. How the salience network works

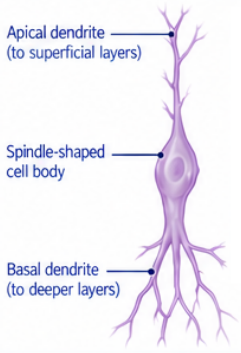


3. Everyday example: hearing your stop on the bus



An alarm may be highly salient, whereas background traffic is usually not. Salience depends on context and current goals.

4. Cells: Constantin von Economo neurons



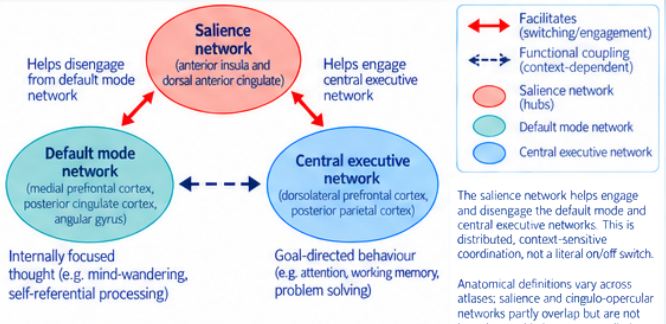
- Constantin von Economo neurons are large, spindle-shaped projection neurons.
- Enriched in the anterior insula and anterior cingulate cortex (salience network regions).
- Vulnerable in behavioural-variant frontotemporal dementia.
- Not unique to humans (also found in other mammals).
- Proposed to support rapid signalling across distributed networks, but they are not proven to be master-switch cells.

5. Relevance to psychiatry

Condition	Key points
Anxiety and panic disorder	Heightened threat and body-signal processing in some contexts (e.g. increased interoceptive sensitivity).
Psychosis	Aberrant significance assignment to neutral events has been studied (Shitij Kapur's dopamine-based aberrant salience hypothesis).
Behavioural-variant frontotemporal dementia	Degeneration of von Economo neurons and related regions affects social-emotional behaviour and salience processing.
Addiction	Drug-related cues gain motivational priority and drive attention and behaviour.

Shitij Kapur's dopamine-based aberrant salience hypothesis and the imaging-defined salience network are related concepts, but not identical explanations. There is no diagnostic scan pattern for salience network function.

6. Network interactions (Vinod Menon's model)

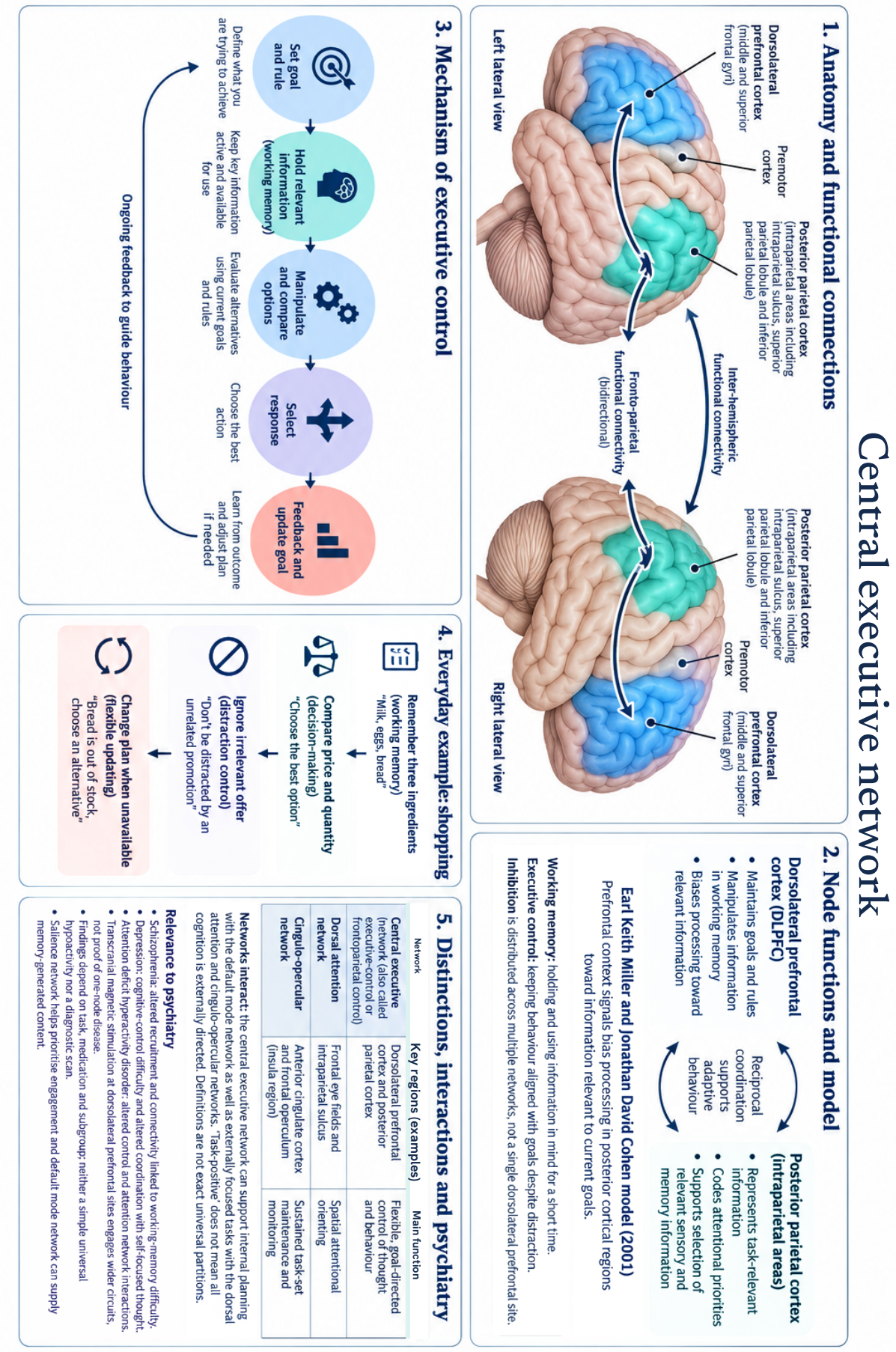


The salience network helps engage and disengage the default mode and central executive networks. This is distributed, context-sensitive coordination, not a literal on/off switch.

Anatomical definitions vary across atlases; salience and cingulo-opercular networks partly overlap but are not interchangeable in every parcellation.

Salience network. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.



Central executive network. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Triple-network interaction

Default mode network



Supplies internally generated content: autobiographical memories, imagined futures and perspectives.

Salience network



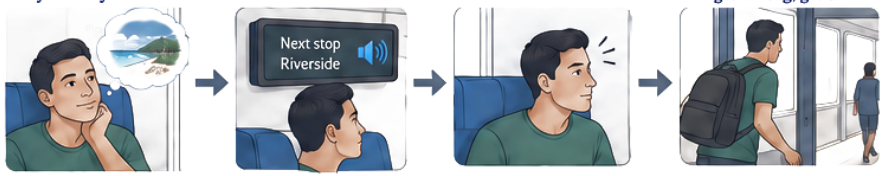
Prioritises relevant internal and external events; helps redirect resources.

Central executive network



Maintains goals, manipulates information and organises goal-directed action.

SWITCHING – from internal to external



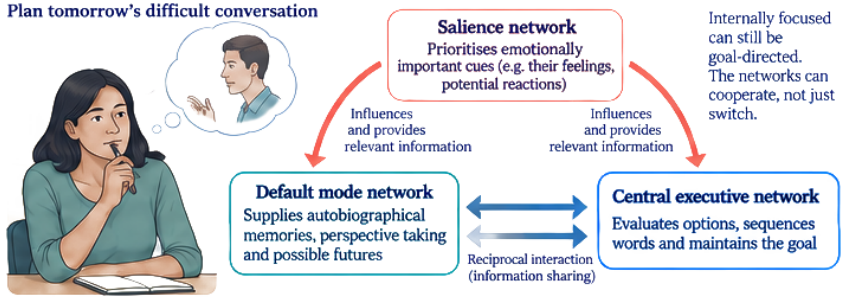
Default mode network relatively more engaged (internal focus).

Salient external change detected (e.g. sound, context).

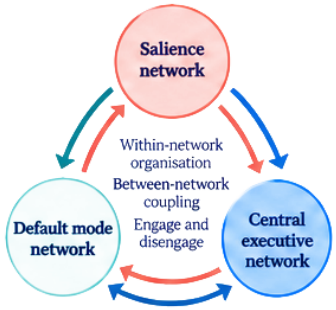
Salience network identifies importance and helps recruit executive control.

Executive network relatively more engaged (task demand). Default mode engagement decreases.

COOPERATION – planning a difficult conversation



Vinod Menon (2011) – triple-network model



Vinod Menon (2011) proposed that psychopathology may involve altered within-network organisation, between-network coupling and the ability to engage and disengage these networks.

A 2008 functional imaging study implicated right frontoinsular cortex in switching. This is model-dependent evidence, not proof of a single controlling centre.

Failure modes and hypotheses (group-level associations)

Pattern	Possible consequence	Examples (hypotheses)
Persistent negative internal focus	Difficulty disengaging from self-referential thought	Rumination, depression
Poorly timed disengagement or re-engagement	Inefficient switching between networks	Attention lapses, attention deficit hyperactivity disorder
Inappropriate significance assigned to cues	Irrelevant stimuli treated as important	Psychosis models
Over-prioritised threat or body cues	Excessive allocation of resources to perceived danger	Anxiety

Many-to-many associations: these are hypotheses and group-level findings, not a one-to-one mapping between a network and a disorder.

Key exam distinctions

- Anticorrelation means signals tend to vary oppositely in a given analysis, but it does not mean the networks are compulsorily opposed in every task.
- Default and executive systems can **cooperate**.
- Connectivity does not equal **activation**. Correlation does not equal **causation**.
- Functional magnetic resonance **imaging** blood-oxygen-level-dependent signal is an indirect measure of neural activity. Motion, global-signal processing and parcellation affect results.
- The triple-network model is a useful organising framework, but it is not a complete explanation and cannot be used as an individual diagnostic test.

Triple-network interaction. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Neurochemistry and neurophysiology.

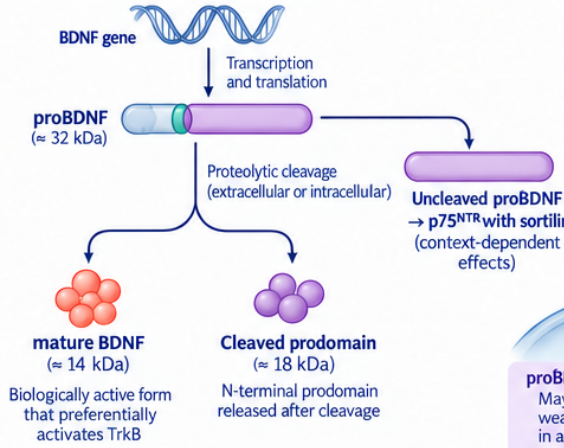
Credits and references: retained within the illustration.

Brain-Derived Neurotrophic Factor

BDNF

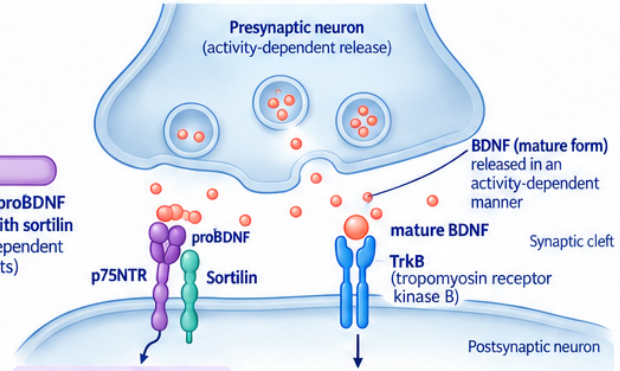
1. From gene to mature BDNF

BDNF is synthesised as a precursor protein that is proteolytically processed to produce mature BDNF.



2. Activity-dependent signaling at the synapse

Neuronal activity stimulates BDNF release, which acts on distinct receptors with different, context-dependent effects.

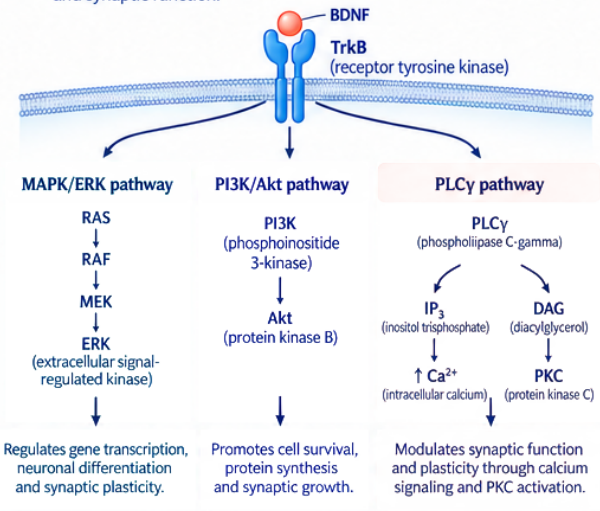


proBDNF–p75^{NTR}–sortilin
May favour synaptic weakening or pruning in a context-dependent manner.
p75NTR is not always a death switch.

mature BDNF–TrkB
Supports synaptic plasticity, neuronal survival and synaptic strengthening.

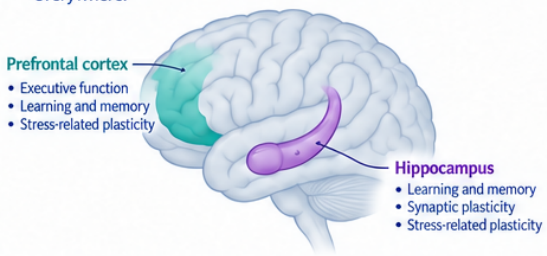
3. TrkB intracellular signaling cascades

TrkB activates multiple pathways that support plasticity, survival and synaptic function.



4. Brain regions and functions

BDNF contributes to learning, memory and stress-related plasticity, particularly in the hippocampus and prefrontal cortex. Its effects vary by brain region and context and are not universally beneficial everywhere.



The effects of BDNF are region-specific and context-dependent, and are not uniformly beneficial across all brain circuits.

5. Clinical and research considerations

	**Neurotrophic hypothesis of depression** Strong experimental support for links among stress, plasticity and antidepressant effects on BDNF. However, this is not a complete or proven causal account of human depression.	Established links, but not a complete causal model.
	**Ketamine and rapid antidepressant effects (research model)** Ketamine may increase AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid) receptor throughput, leading to BDNF release, TrkB activation and downstream mTOR (mechanistic target of rapamycin) signaling.	A proposed mechanistic pathway, not the sole established mechanism.
	**Val66Met polymorphism** A common BDNF prodomain variant that affects intracellular trafficking and activity-dependent secretion of BDNF. The mature BDNF protein sequence is unchanged.	Established molecular effect; functional consequences vary by context.
	**Psychotherapy and individual response** There is no routine BDNF test to select a type of psychotherapy or to predict individual response.	Not clinically indicated.
	**Peripheral BDNF levels** Peripheral BDNF is influenced by platelets, physical activity and sample handling. Blood BDNF level is not a direct measure of brain BDNF and is not diagnostic.	Not a brain measure and not diagnostic.

6. BDNF and synaptic plasticity

BDNF is involved in both strengthening and weakening of synapses, depending on the form of BDNF, receptor engaged and neural context.

Long-term potentiation (LTP)
Persistent strengthening of synaptic connections.



- Associated with learning and memory
- Supported by BDNF–TrkB signaling
- Increased synaptic strength

Long-term depression (LTD)
Persistent weakening of synaptic connections.



- Can be influenced by proBDNF–p75^{NTR} signaling (context-dependent)
- Involved in synaptic pruning and plasticity
- Decreased synaptic strength

Long-term potentiation and long-term depression refer to synaptic plasticity and are not the same as mood depression.

Brain-Derived Neurotrophic Factor. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

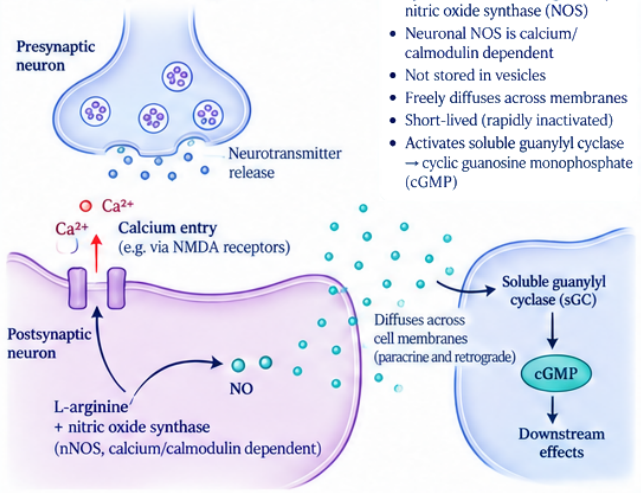
Credits and references: retained within the illustration.

Novel Neurotransmitters

Non-classical neural signalling

Nitric oxide (NO)

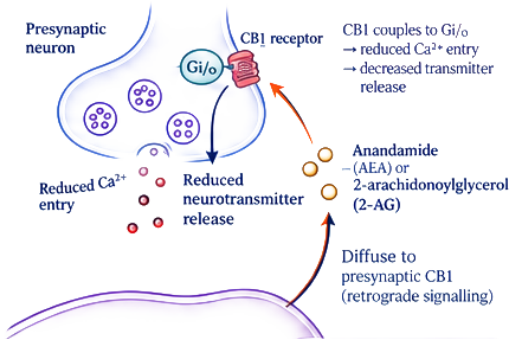
Activity-dependent gaseous signal



- Synthesised from L-arginine by nitric oxide synthase (NOS)
- Neuronal NOS is calcium/calmodulin dependent
- Not stored in vesicles
- Freely diffuses across membranes
- Short-lived (rapidly inactivated)
- Activates soluble guanylyl cyclase → cyclic guanosine monophosphate (cGMP)

Endocannabinoids

Lipid mediators of synaptic signalling



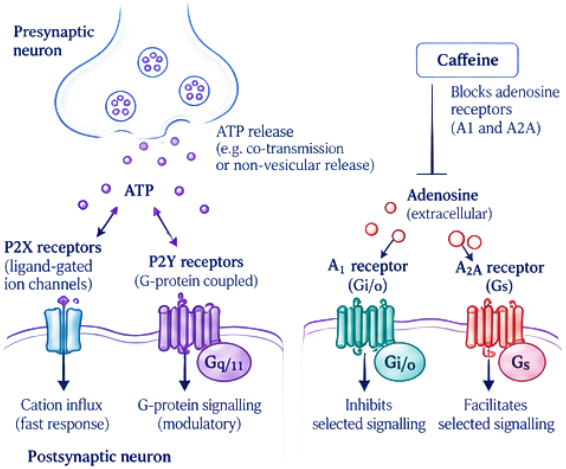
Synthesised on demand from membrane lipid precursors (e.g. arachidonic acid derivatives) in response to activity

Termination

- Fatty acid amide hydrolase (FAAH) mainly breaks down anandamide
- Monoacylglycerol lipase (MAGL) mainly breaks down 2-arachidonoylglycerol

Purines

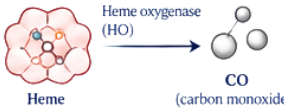
ATP and adenosine as neural signals



Other gaseous candidates

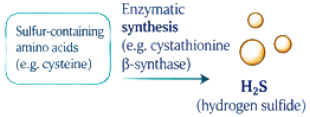
Carbon monoxide and hydrogen sulfide

Carbon monoxide (CO)



- Produced from heme by heme oxygenase
- Diffuses across membranes
- Modulates neuronal signalling (e.g. via guanylyl cyclase, ion channels – context dependent)
- Physiological roles are context-dependent and not equivalent to all classical neurotransmitter criteria

Hydrogen sulfide (H_2S)



- Produced from sulfur metabolism
- Diffuses across membranes
- Modulates neuronal signalling (e.g. ion channels, intracellular pathways – context dependent)
- Physiological roles are context-dependent and not equivalent to all classical neurotransmitter criteria

Key points

- "Novel" is a historical teaching label, not proof of recent discovery.
- Lipid and gas signals can bypass vesicular release.
- These transmitters do not meet all the criteria for classical neurotransmitters (e.g. vesicular storage, quantal release).
- They can act as retrograde, paracrine or local messengers.
- There is no universal excitatory or inhibitory effect – actions are context dependent (cell type, receptor and circuit).
- Many proposed mechanisms and functions remain research models rather than established facts.

Clinical connections

- Involved in synaptic plasticity and learning.
- Modulate pain processing.
- Influenes sleep and arousal (e.g. adenosine).
- Implicated in reward and addiction pathways (e.g. endocannabinoids, dopamine interactions).
- Contribute to vascular regulation (e.g. nitric oxide, carbon monoxide, hydrogen sulfide).
- Experimental targets in these systems are not established psychiatric treatments.

Comparison table

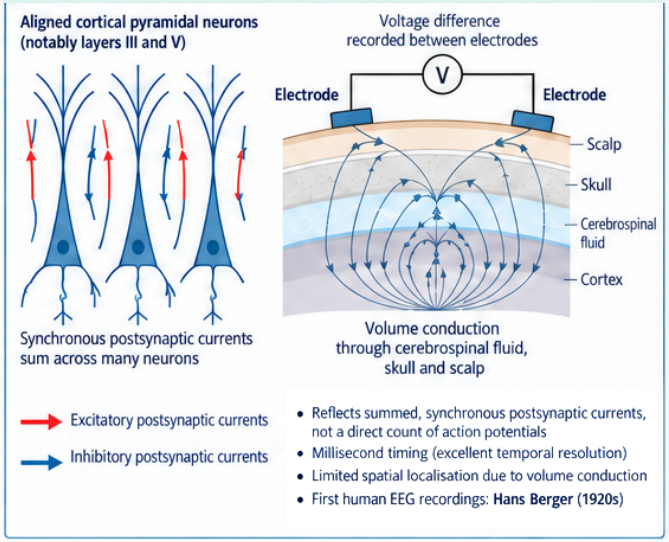
Feature	Nitric oxide (NO)	Endocamabinoids (AEA, 2-AG)	Purines (ATP, adenosine)	Other gases (CO, H_2S)
Chemical nature	Gas	Lipids (endogenous cannabinoids)	Nucleotide and nucleoside	Gases
Main synthesis	L-arginine + NOS (nNOS calcium/ (calmodulin dependent)	On demand from membrane lipid precursors	ATP release; adenosine from ectonucleotidases	CO: heme oxygenase H_2S: sulfur metabolism
Storage in vesicles	No	No	ATP: can be stored; adenosine: no	No
Direction of signalling	Diffuses locally (paracrine and retrograde)	Postsynaptic to presynaptic (retrograde)	Local, paracrine and synaptic	Diffuses locally (paracrine)
Main receptors/ targets	Soluble guanylyl cyclase → cGMP	CB1 receptor (Gi/o)	P2X (ion channels) P2Y (G-protein) P1 adenosine receptors (A1, A2A)	Multiple targets (context dependent)
Key actions	Increases cGMP; modulates signalling	Reduces Ca^{2+} entry and transmitter release	ATP excites (P2X/P2Y); A1 inhibits; A2A facilitates selected signalling	Modulatory roles (context dependent)
Termination	Rapid inactivation (short-lived)	FAAH (anandamide); MAGL (2-AG)	Ectonucleotidases; reuptake; metabolism	Diffusion and chemical/metabolic removal
Notes	Not a classical neurotransmitter	Not a classical neurotransmitter	Both co-transmitter and neuromodulator roles	Not classical neurotransmitters

Novel Neurotransmitters. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

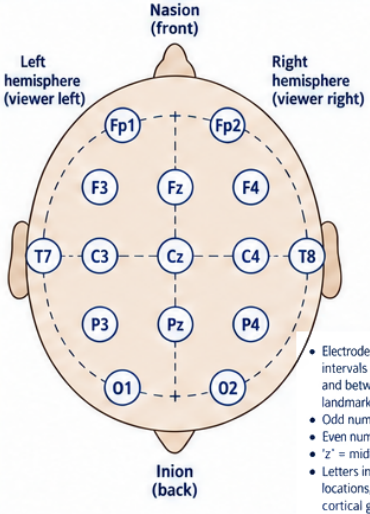
Electroencephalography

1. Electrical basis of the EEG



- Reflects summed, synchronous postsynaptic currents, not a direct count of action potentials
- Millisecond timing (excellent temporal resolution)
- Limited spatial localisation due to volume conduction
- First human EEG recordings: **Hans Berger (1920s)**

2. 10-20 electrode system (view from above)



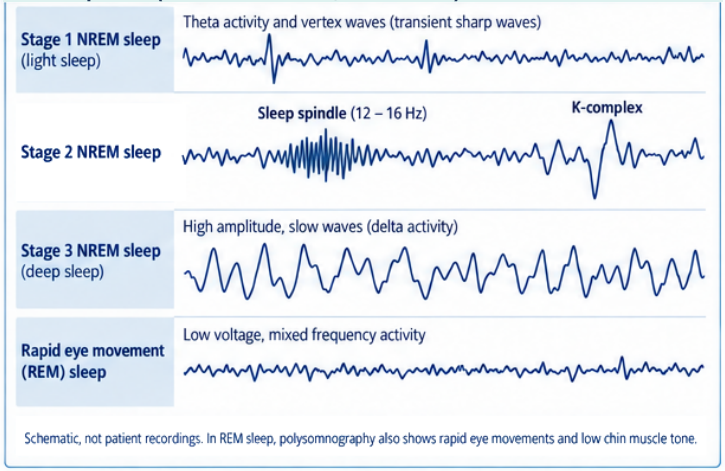
- Electrodes placed at 10% or 20% intervals between nasion-inion and between preauricular landmarks
- Odd numbers = left hemisphere
- Even numbers = right hemisphere
- 'z' = midline (Fz, Cz, Pz)
- Letters indicate scalp electrode locations, not exact, isolated cortical generators

3. EEG frequency bands

Band	Frequency (Hz)	Typical features and normal associations	Schematic, not patient recordings
Delta	0.5 – < 4	High amplitude, slow activity. Prominent in deep non-rapid eye movement (NREM) sleep (stage 3).	
Theta	4 – < 8	Seen in drowsiness; also normal task-related frontal theta (e.g. working memory).	
Alpha	8 – 13	Posterior dominant rhythm, seen with eyes closed and relaxed. Attenuates with eyes open.	
Beta	> 13 – 30	Low amplitude, fast activity. Increased with benzodiazepines and barbiturates.	
Gamma	> 30	Higher frequency activity, involved in cognition and sensory integration (research). Muscle artefact can contaminate this range.	

Frequency boundaries vary slightly by convention.

4. Sleep EEG (normal features, schematic)



Schematic, not patient recordings. In REM sleep, polysomnography also shows rapid eye movements and low chin muscle tone.

5. Important EEG patterns and clinical points

Pattern	Main associations and notes
Diffuse slowing	Generalised slowing of background activity. Seen in cerebral dysfunction (e.g. delirium, encephalopathy). Nonspecific.
Generalised ~3 Hz spike–wave	Typical of childhood absence epilepsy.
Generalised polyspike–wave	Typical of juvenile myoclonic epilepsy.
Slow spike–wave (< 2.5 Hz)	Characteristic of Lennox–Gastaut syndrome.
Chaotic, high amplitude, multifocal spikes	Hypsarrhythmia pattern, seen in infantile epileptic spasms (West syndrome).
~1 Hz periodic sharp wave complexes	Supports a diagnosis of sproadic Creutzfeldt–Jakob disease (in the appropriate clinical context).
Generalised periodic discharges with triphasic morphology	Seen in toxic / metabolic encephalopathy. Not specific to hepatic encephalopathy.
Spike vs sharp wave	**Spike:** 20 – 70 ms. **Sharp wave:** 70 – 200 ms. A sharp appearance alone does not imply epilepsy.

Key clinical points

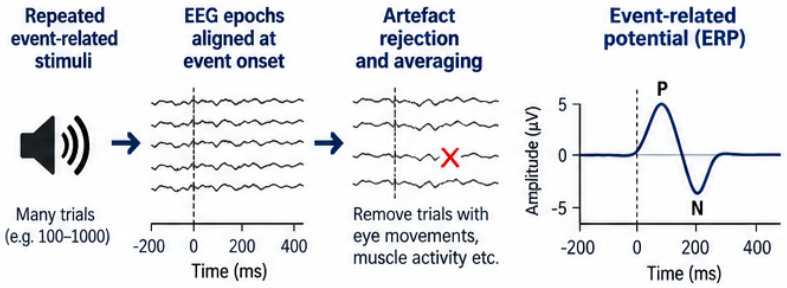
- A normal routine EEG does not exclude epilepsy.
- An abnormal EEG does not itself establish epilepsy or exclude a psychiatric illness.
- Hyperventilation can activate absence seizures.
- Intermittent photic stimulation can precipitate photosensitive responses.
- Sleep (drowsiness or sleep deprivation) can improve yield.
- Eye movement, muscle and electrode artefact can affect the tracing.
- Medications can influence the EEG (e.g. sedatives increase beta activity).
- During electroconvulsive therapy, EEG is used to assess ictal evolution, seizure quality and postictal suppression, not duration alone.
- There is no routine diagnostic EEG for schizophrenia, depression or attention deficit hyperactivity disorder (ADHD).

Electroencephalography. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

Event-related potentials

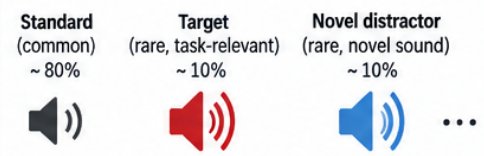
1. Recording principle



- Averaging reduces uncorrelated background EEG noise by the square root of the number of trials ($\propto 1/\sqrt{N}$).
- **Amplitude** = voltage size (μV); **latency** = time (ms) to a feature.
- **Positive (P)** deflections are plotted upwards; negative (N) deflections downwards.
- Early sensory components and later, more context-dependent components form a useful continuum, not a rigid cutoff.
- All are event-related responses to a stimulus, response or other significant event.

2. Oddball paradigm and key components

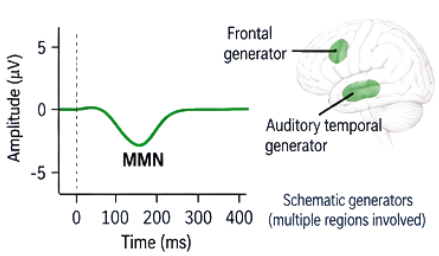
Typical oddball sound stream



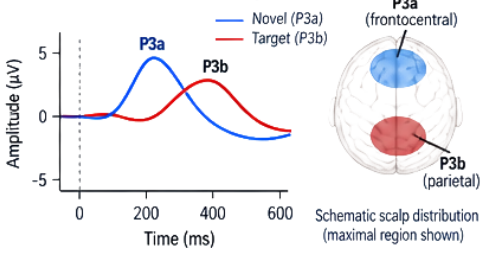
Key components

- **Mismatch negativity (MMN, 100–250 ms)**: deviant minus standard response, frontocentral negativity. Automatic auditory change detection even without active attention. Generators in auditory temporal and frontal regions. Involvement of N-methyl-D-aspartate (NMDA) receptors.
- **P3a**: earlier frontocentral positivity to novelty/orienting.
- **P3b**: usually ~300–600 ms parietal positivity to task-relevant target, reflecting attention and context updating.
- Emanuel Donchin and Michael Coles proposed a context-updating model for the P3. P3 latency is not fixed exactly at 300 ms.

Mismatch negativity (deviant minus standard, schematic)

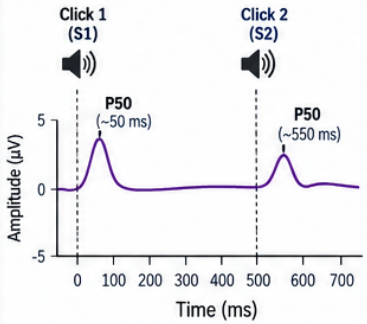


P3 (schematic waveforms)



3. Paired-click P50 auditory gating

Two identical clicks separated by 500 ms, each evokes a P50 approximately 50 ms after the click.



- The second P50 is usually smaller than the first.
- **S2/S1** amplitude ratio indexes sensory gating (suppression). A higher ratio indicates less gating, but methodology varies between studies.
- **Alpha7** nicotinic acetylcholine receptors are implicated, but this is not a single-receptor assay.
- **P50** gating is a different process from oddball mismatch and P3 responses.

4. Other commonly studied components

Component	Typical latency (ms)	Main function and features	Typical distribution / notes
N400	~400	Semantic expectancy and integration. e.g. "She drank tea with sugar" vs unexpected "... with shoes".	Centroparietal negativity.
N170	~170	Face processing (structural encoding of faces).	Occipitotemporal negativity.
Contingent negative variation (CNV)	Slow negativity between cues	Slow negative shift between a warning cue and an imperative cue. Anticipation and preparation.	Frontocentral. Reflects expectancy and motor preparation.
Error-related negativity (ERN)	~0–100 after error	Performance monitoring after an error response.	Frontocentral. Medial frontal/anterior cingulate contribution. Typically response-locked (not stimulus-locked).

5. Psychiatry and limitations

Findings in psychiatric disorders

- **Schizophrenia**: reduced **MMN** and **P3** amplitude, and impaired P50 suppression. N400 changes also probe language processing.
- **Obsessive-compulsive disorder and anxiety**: often enhanced error-related negativity.
- **P3** reductions have been studied in familial externalising and alcohol-use risk. P3 latency is influenced by age and cognitive impairment.

Candidate endophenotype

- A measurable intermediate trait with evidence for heritability/familial aggregation and state-independence, not just an abnormality.
- No ERP alone diagnoses schizophrenia, autism, dementia or predicts an individual's psychosis with certainty.

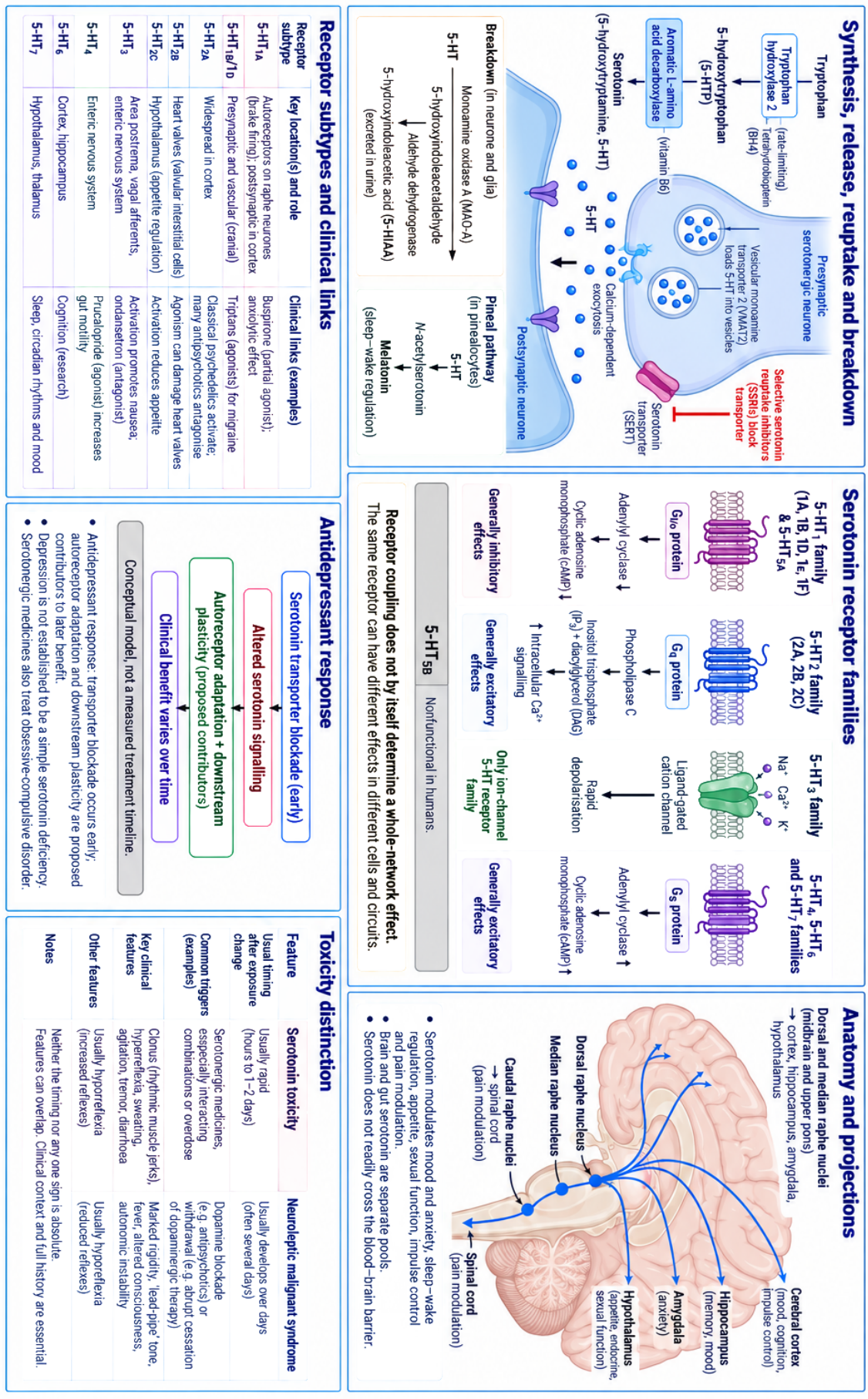
Other sensory event-related potentials (examples)

Modality	Component(s)	Main clinical relevance
Visual	P100 (~100 ms)	Optic pathway function (e.g. demyelination).
Auditory	Brainstem auditory early waves (waves I, III, V; ~1–10 ms)	Hearing and brainstem function.
Somatosensory	N20 / P25 (~20–25 ms)	Somatosensory pathways.

Avoid oversimplified one-wave–one-brainstem-nucleus mapping. Multiple generators contribute to each component.

Event-related potentials. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

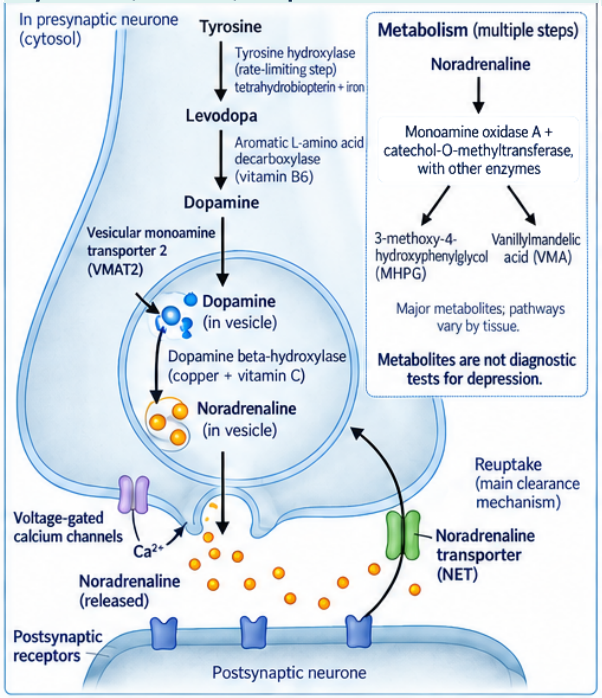


Serotonin. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

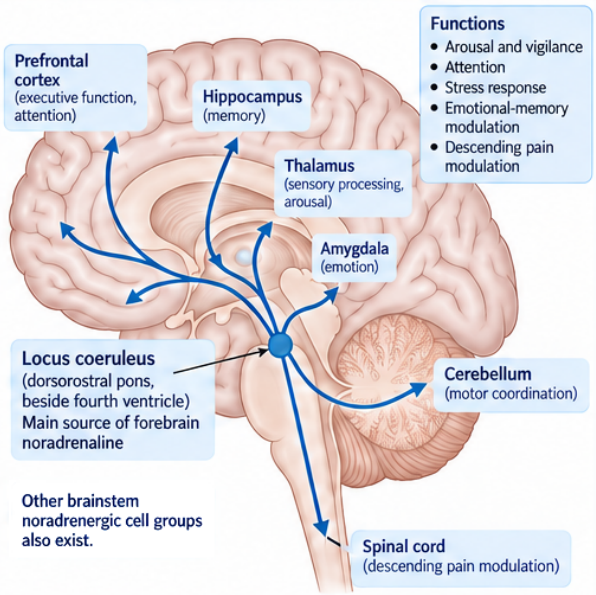
Credits and references: retained within the illustration.

Noradrenaline

Synthesis, release, reuptake and metabolism



Locus coeruleus and noradrenergic projections



Central adrenaline (epinephrine) cell groups are in the medulla, not the locus coeruleus.

Noradrenaline receptors

Receptor subtype	G protein	Main intracellular signalling effect	Examples of location (in brain)
Alpha-1 (α_{1A}, α_{1B}, α_{1D})	Gq	Phospholipase C → intracellular calcium signalling ↑	Widespread (including cortex, hippocampus)
Alpha-2 (α_{2A}, α_{2B}, α_{2C})	Gi/o	Adenylyl cyclase ↓ → cyclic adenosine monophosphate (cAMP) ↓	Presynaptic terminals and postsynaptic regions (e.g. prefrontal cortex)
Beta-1 (β_1)	Gs	Adenylyl cyclase ↑ → cyclic adenosine monophosphate (cAMP) ↑	Widespread (including cortex, hippocampus)
Beta-2 (β_2)	Gs	Adenylyl cyclase ↑ → cyclic adenosine monophosphate (cAMP) ↑	Widespread
Beta-3 (β_3)	Gs	Adenylyl cyclase ↑ → cyclic adenosine monophosphate (cAMP) ↑	Various regions (including brain and periphery)

Two distinct locations for alpha-2A (α_{2A}) receptors

1. Presynaptic autoreceptor
(on noradrenergic terminal)

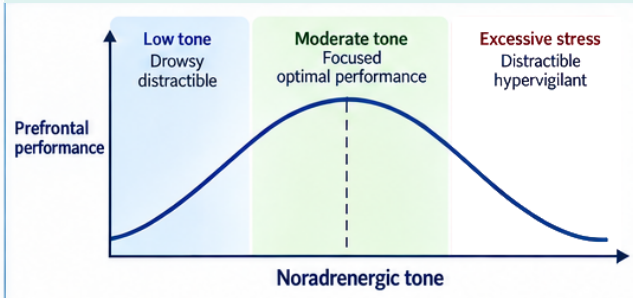


2. Postsynaptic receptor
(e.g. prefrontal cortex pyramidal neurone)



Guanfacine: postsynaptic action matters; not merely less transmitter.

Noradrenergic tone and prefrontal function



Tonic firing = baseline level of activity.

Phasic firing = brief, stimulus-linked burst of activity.

Conceptual relationship; depends on task and state.

Drug logic (selected examples)

Drug	Mechanism (at noradrenaline system)	Main clinical use(s)
Atomoxetine	Noradrenaline transporter (NET) inhibitor → increases synaptic noradrenaline	Attention deficit hyperactivity disorder
Guanfacine / Clonidine	Alpha-2 (α_2) agonists → decreased noradrenergic release (presynaptic) AND postsynaptic effects (e.g. prefrontal cortex)	Attention deficit hyperactivity disorder; clonidine also used for hyperarousal and sleep
Mirtazapine	Alpha-2 (α_2) antagonist → increases noradrenergic release (and serotonin release)	Depression
Prazosin	Alpha-1 (α_1) antagonist	Used for trauma-related nightmares
Propranolol	Beta (β) antagonist → reduces peripheral autonomic symptoms	Performance anxiety and other anxiety states (e.g. tremor, palpitations)
Duloxetine	Serotonin and noradrenaline reuptake inhibitor (SNRI)	Depression and neuropathic pain

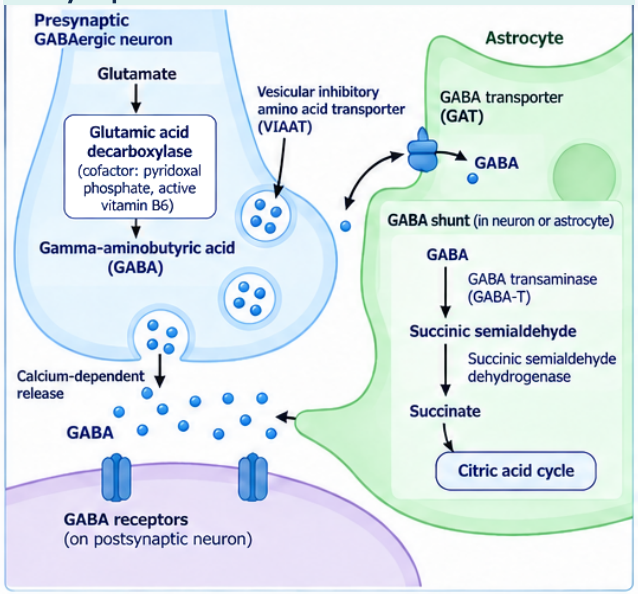
Key points: Autoreceptor agonism can lower release. Noradrenaline and adrenaline are not interchangeable. Drug benefit does not prove a simple transmitter deficiency.

Noradrenaline. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

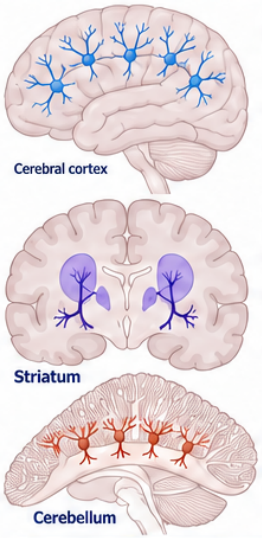
Credits and references: retained within the illustration.

Gamma-aminobutyric acid

1. Synapse and GABA shunt



2. Anatomy of GABAergic systems



Cerebral cortex inhibitory interneurons

- Diverse local interneurons (e.g. parvalbumin, somatostatin, vasoactive intestinal peptide)
- Regulate timing and synchrony of pyramidal neuron activity
- Provide local, feedforward and feedback inhibition

Striatum GABAergic projection neurons

- Medium spiny neurons (γ-aminobutyric acid [GABA]-ergic)
- Inhibit distant targets in basal ganglia circuits (direct and indirect pathways)
- Regulate movement, habit and reward processing

Cerebellum: Jan Evangelista Purkinje cells

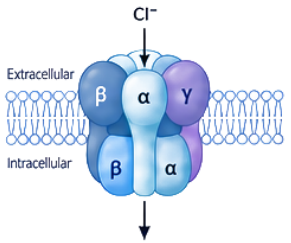
- GABAergic projection neurons
- Inhibit deep cerebellar nuclei
- Essential for motor coordination and motor learning

A distributed inhibitory system, not one brainstem source.

3. GABA receptors

GABA-A receptor

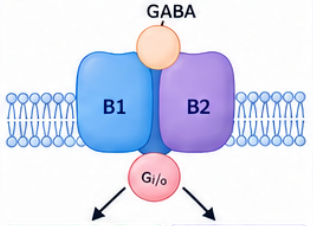
Fast, ligand-gated chloride channel



- Pentamer, commonly 2 alpha + 2 beta + 1 gamma subunits
- GABA binding opens chloride channel (Cl^- influx)
- In mature neurons, usually hyperpolarising or shunting inhibition (reduces effect of excitatory input)
- Developmental chloride gradients can make GABA-A depolarising
- Depolarising does not always mean net excitation

GABA-B receptor

Slow, G protein-coupled receptor



- Heterodimer of B1 and B2 subunits
- Activates Gi/o proteins
- Presynaptic: reduces voltage-gated calcium entry → less neurotransmitter release
- Postsynaptic: increases potassium conductance → potassium leaves cell (hyperpolarisation)
- Old GABA-C terminology refers to rho-containing GABA-A receptors

4. Drugs and mechanisms

Drug or class	Primary action	Key points
Benzodiazepines (e.g. diazepam)	Positive allosteric modulator at GABA-A (alpha/gamma interface)	Increase channel opening frequency. Require GABA.
Barbiturates (e.g. phenobarbital)	Positive allosteric modulator at GABA-A	Classically increase channel opening duration. High concentrations can directly gate the channel.
Zolpidem	Alpha1-preferring GABA-A modulator	Enhances GABA-A activity. More selective for sedative effects.
Baclofen	GABA-B receptor agonist	Activates GABA-B receptors (B1 + B2).
Tiagabine	Inhibits GABA reuptake	Blocks GABA transporter (GAT) → increases synaptic GABA.
Vigabatrin	Irreversibly inhibits GABA transaminase (GABA-T)	Increases brain GABA by preventing its breakdown.
Allopregnanolone	Neurosteroid, positive allosteric modulator at GABA-A	Enhances GABA-A activity.
Gabapentin / Pregabalin	Bind alpha2delta subunit of voltage-gated calcium channels	Reduce presynaptic calcium entry and neurotransmitter release. Not GABA receptor agonists.

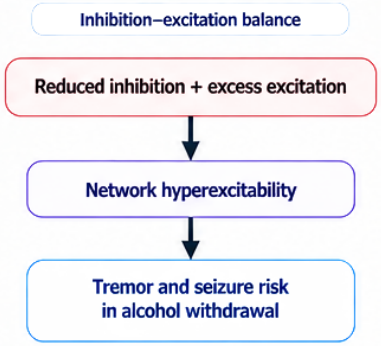
5. Functions and clinical relevance

Key functions

- Inhibition balances excitation in neural circuits
- Regulates anxiety and stress responses
- Important for sleep
- Raises seizure threshold
- Essential for motor coordination

Clinical relevance

- GABA-A receptor enhancement explains anxiolysis, sedation, anticonvulsant effects and impaired coordination
- Alcohol withdrawal: loss of adapted inhibitory drive plus excess excitation → tremor and seizure risk
- Schizophrenia: cortical interneuron dysfunction is a proposed circuit mechanism, not a diagnostic GABA test



GABAergic inhibition is essential for normal brain function.

Gamma-aminobutyric acid. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neurochemistry and neurophysiology.

Credits and references: retained within the illustration.

AREA 02 OF 12 · P1-A01

Functional neuroanatomy and neuroimaging

12	13.5%	I-14	I-25
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 5 · RECURRENT 1 · SINGLE 6

REVISION ORDER

The limbic group first and as one block: the Papez circuit, the limbic system, the amygdala and the two hippocampal slots overlap almost completely. The cortical group next, prefrontal, frontal, cerebral cortex and occipital, and the frontal slot is the long one. The caudate nucleus is asked twice and is one answer at two widths. Thalamus, corpus callosum and basal ganglia last, and all three are short.

WHAT IS IN THIS AREA

Twelve structures, close to one each: the corpus callosum, the Papez circuit with the caudate nucleus, the prefrontal cortex, the amygdala, the cerebral cortex with its cellular organisation and circuitry, the thalamus, the limbic system, the basal ganglia with the hippocampus, the frontal lobe with three injury syndromes and parietal atrophy, the hippocampal formation with its subfield volumes, the caudate nucleus on its own, and the occipital cortex. The registered name of this area also carries neuroimaging. Imaging is a limb of one slot here rather than the subject of any, and the area is described by what its slots actually ask.

I - 14

Discuss the role of Corpus callosum. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The largest commissure, and its five named segments
3 **Fibre topography**	Which cortex each segment joins, front to back
3 **What it does**	Transfer, lateralisation, bimanual and interhemispheric control
2 **When it fails**	Agenesis, callosotomy, and the disconnection signs

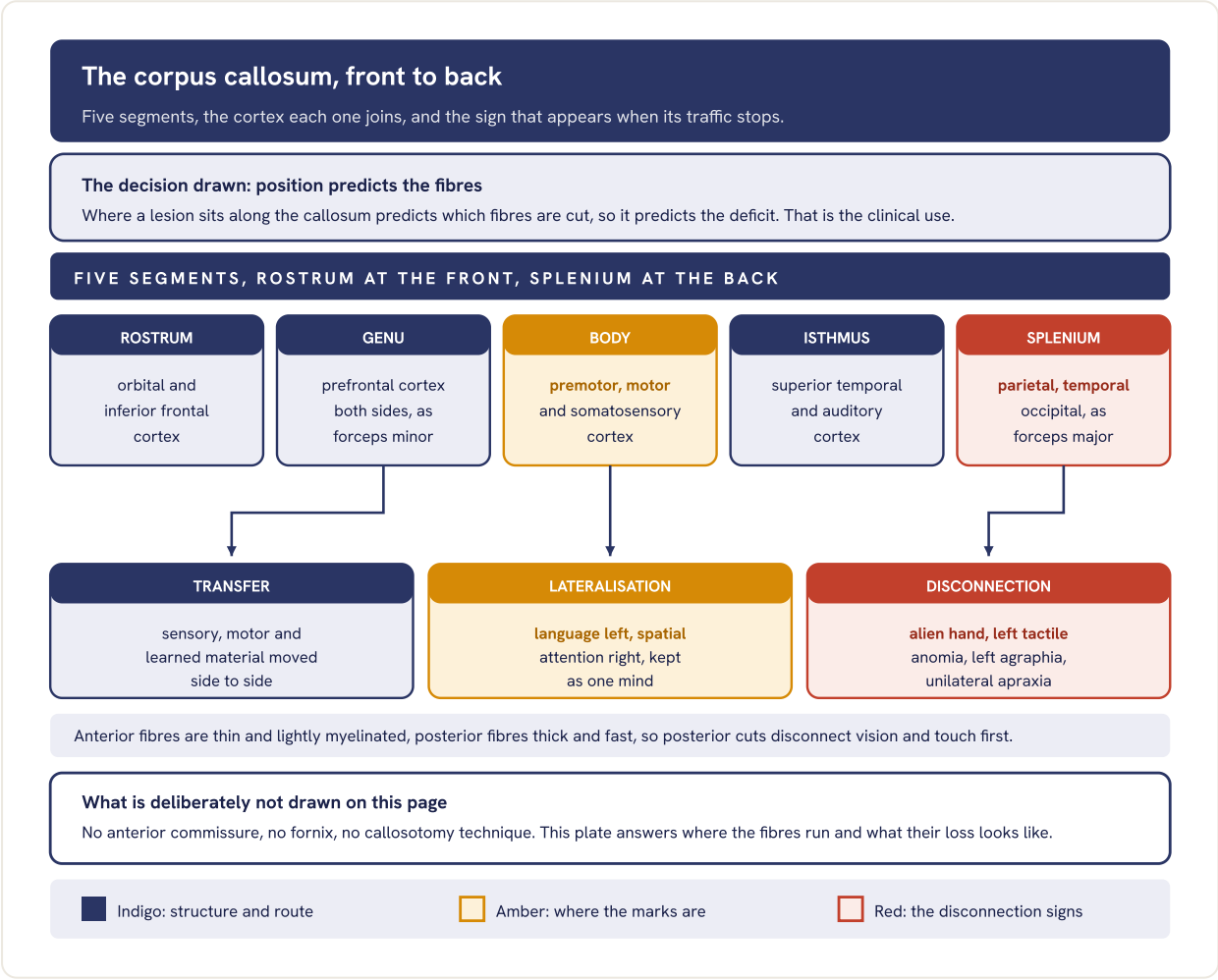


Fig 014.1 The callosum drawn as five segments with the cortex each joins, and the three consequences that follow from the traffic they carry.

THE DEFINITION

The largest white matter commissure of the brain, a sheet of myelinated axons joining homologous and heterologous cortical areas of the two hemispheres. It is topographically ordered: rostrum and genu in front carry frontal fibres, the splenium behind carries parieto-occipital fibres.

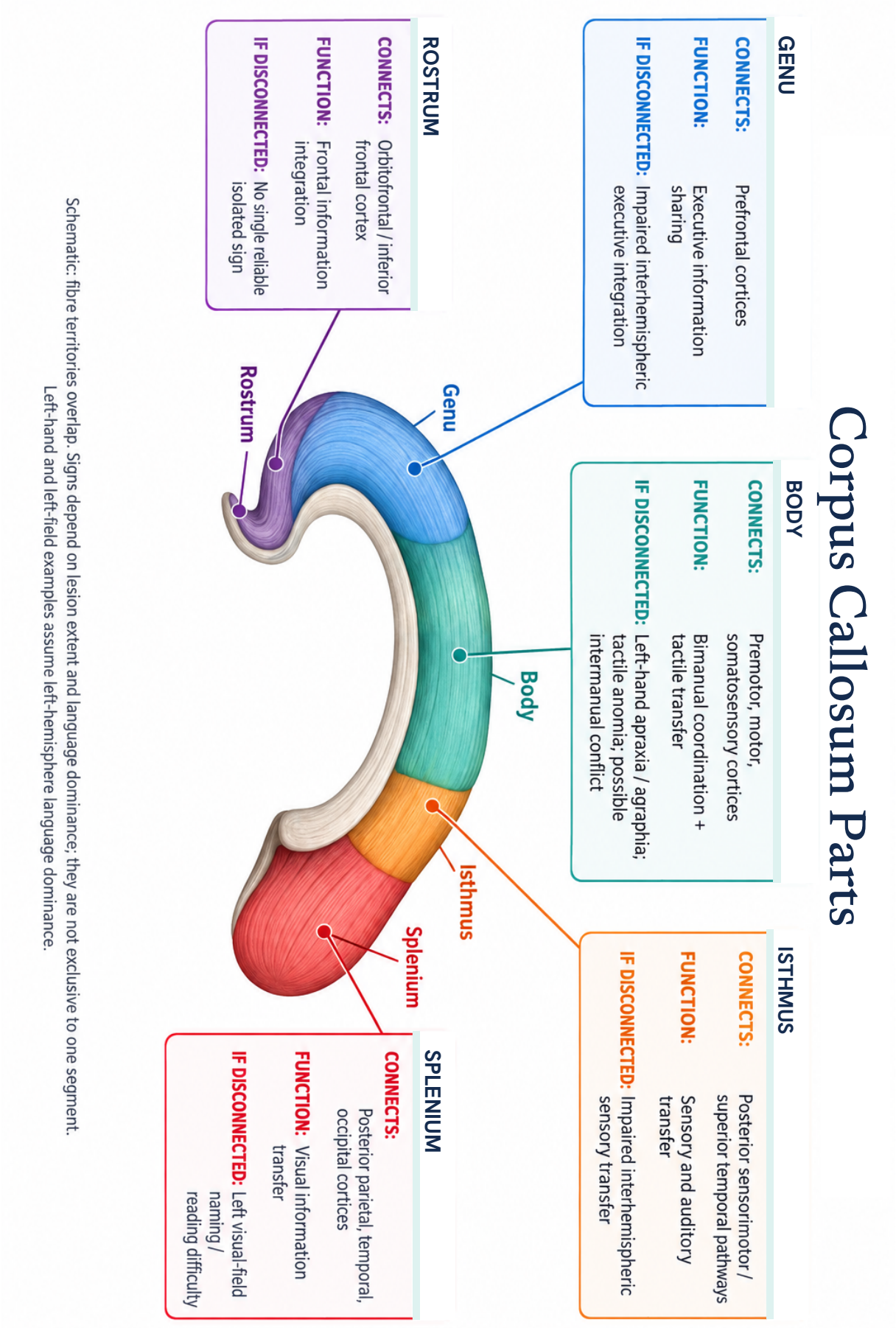
- **Transfer.** Sensory, motor and learned information crosses here, which is why a split-brain patient cannot name an object felt only by the left hand.

- **Integration with lateralisation.** Language sits left and spatial attention right; the callosum lets specialised hemispheres act as one, and coordinates bimanual movement.
- **Callosal syndromes.** Agenesis (often with other midline defects), surgical callosotomy for intractable epilepsy, and Marchiafava-Bignami disease from chronic alcohol use.

WHAT EARNS THE MARKS

- **The five segments named in order.** Rostrum, genu, body, isthmus, splenium, each with the cortex it joins.
- **Topography stated as a rule** before any sign is listed: front carries frontal, back carries posterior sensory.
- **Disconnection signs tied to a segment.** Left tactile anomia and left agraphia belong to anterior fibres, hemialexia to the splenium.
- **The three failure states,** agenesis, callosotomy and Marchiafava-Bignami, named as such.

SEE ALSO Slot I - 16 prefrontal cortex Slot I - 22 frontal and parietal lobe syndromes **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology



Corpus Callosum Parts. Use the labelled structures to connect anatomical location with function. Read with I-14.

Credits and references: retained within the illustration.

I - 15

Neuroanatomy of Papez circuit and a brief mention about the caudate nucleus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The proposal	Papez 1937, and what the loop was meant to explain
4	The loop in order	Six stations, hippocampus back to hippocampus
2	What it explains	Diencephalic amnesia sits on this ring
2	Caudate, briefly	Position, loop membership, two clinical uses

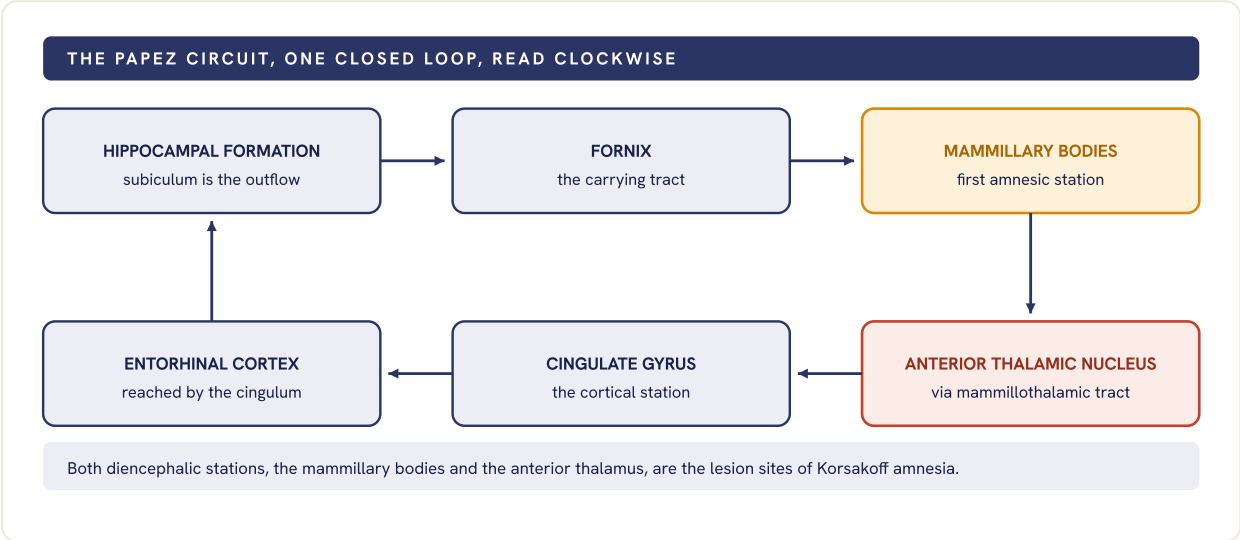


Fig 015.1 The circuit as a closed ring, with the two diencephalic stations that carry the amnesia marked out from the four that do not.

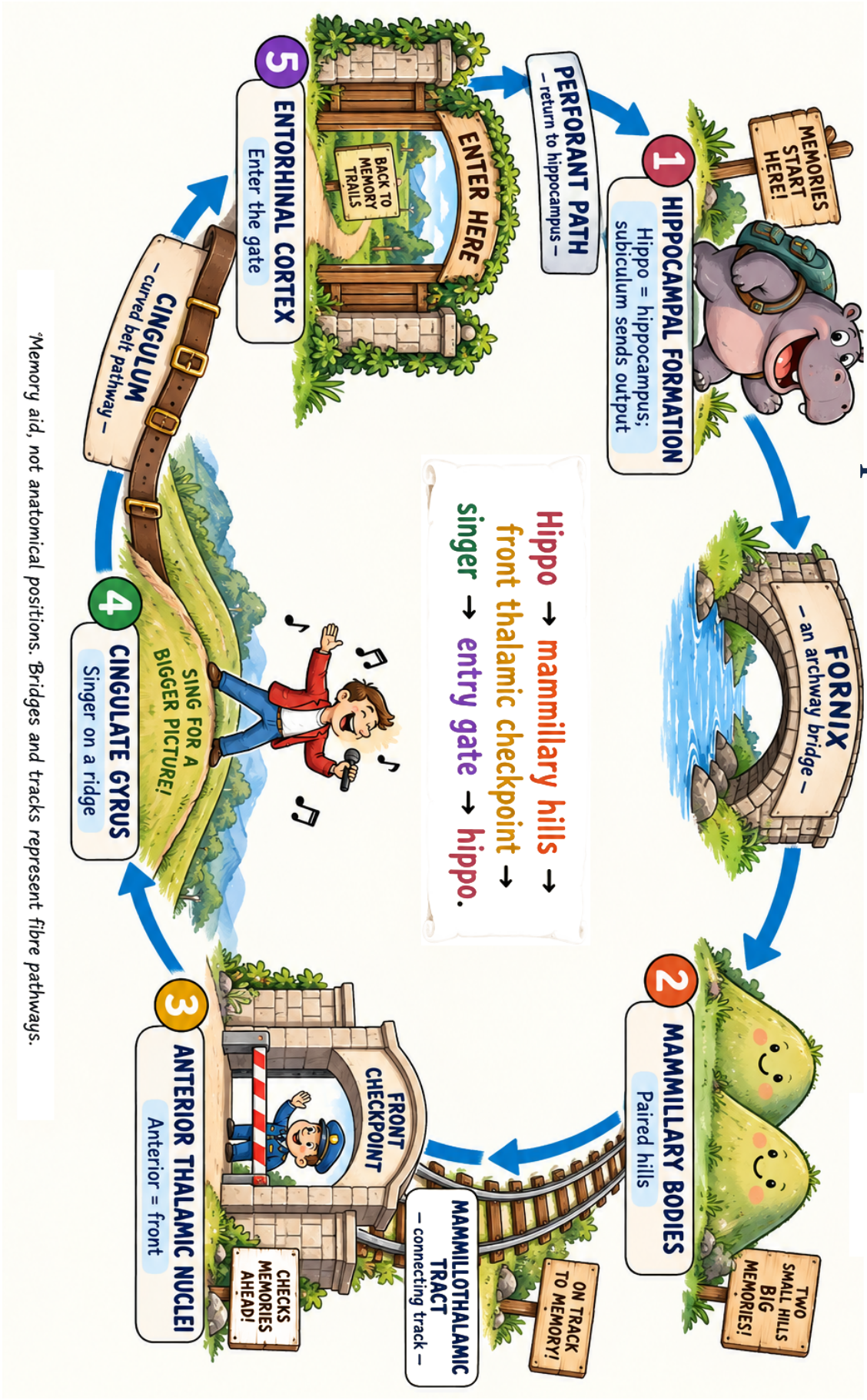
- **What Papez proposed.** In 1937 a cortical to hypothalamic loop offered as the substrate of emotional experience. MacLean later widened it into the limbic system.
- **Why the anatomy outlived the claim.** It is now taught as an episodic memory circuit: thiamine deficient Korsakoff amnesia, fornix section and anterior thalamic infarcts all sit on this ring.
- **Caudate nucleus.** A C-shaped mass with head, body and tail curving in the lateral ventricle wall, continuous with the putamen and forming the associative striatum.
- **Caudate, clinically.** Head atrophy with boxcar ventricles in Huntington disease, hypermetabolism in obsessive compulsive disorder that settles as the illness responds.

WHAT EARNS THE MARKS

- **All six stations, in order, with the two tracts named.** Fornix and mammillothalamic tract are the links most answers skip.
- **The circuit tied to amnesia, not only to emotion.** Saying where Korsakoff sits is the line that separates the bands.
- **One honest limit.** Emotion is not made by one ring; the amygdala and prefrontal circuits lie outside Papez.

SEE ALSO Slot I - 20 limbic system Slot I - 24 caudate nucleus SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology



Papez Circuit Mnemonic. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with I-15.

Credits and references: retained within the illustration.

I - 16

Prefrontal cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What defines it	Granular cortex in front of premotor, defined by its thalamic input
4	The three subdivisions	Each with its job and the syndrome its loss makes
2	Connections	Loops with striatum, thalamus, amygdala and hippocampus
2	Psychiatric relevance	Late maturation, hypofrontality, and what is testable

THE DEFINITION

The granular association cortex of the frontal lobe lying anterior to the premotor areas. It is defined anatomically by its reciprocal connection with the mediodorsal nucleus of the thalamus, and functionally as the seat of executive control: holding a goal, selecting an action, and suppressing the rest.

SUBDIVISION	WHERE IT SITS	WHAT IT DOES	WHAT ITS LOSS LOOKS LIKE
Dorsolateral	Middle frontal gyrus, lateral surface	Working memory, planning, set shifting, abstraction	Dysexecutive: perseveration, concrete thinking, poor planning
Orbitofrontal	Above the orbit, with gyrus rectus	Reward valuation, impulse control, social judgement	Disinhibited: tactless, impulsive, socially coarse
Ventromedial and anterior cingulate	Medial wall	Motivation, drive, error monitoring, autonomic tone	Apathetic: abulia, and akinetic mutism when severe

Three syndromes, not one frontal syndrome. Naming which subdivision produced the picture is what the examiner is checking.

- **Connections.** Five parallel cortico-striato-thalamo-cortical loops run through the caudate and thalamus, with heavy dopaminergic input from the ventral tegmental area and direct links to amygdala and hippocampus.
- **Development.** It myelinates last, into the third decade, which is the anatomical reason adolescent judgement and impulse control mature after appetite and emotion do.
- **Psychiatric relevance.** Reduced dorsolateral activation on working memory tasks in schizophrenia, subgenual cingulate overactivity in depression, and orbitofrontal loop overactivity in obsessive compulsive disorder.

WHAT EARNS THE MARKS

- **The mediodorsal thalamic connection as the definition.** It is what makes prefrontal cortex a region and not a direction.
- **Three syndromes, each tied to its subdivision.** A single undifferentiated frontal syndrome is a mid-band answer.
- **One bedside test per subdivision.** Verbal fluency and Wisconsin sorting for dorsolateral, go and no-go for orbitofrontal.

SEE ALSO Slot I - 22 frontal lobe syndromes Slot I - 19 thalamic nuclei SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 17

Write briefly about the functional neuroanatomy of amygdala. Discuss the psychiatric implications related with amygdala. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Position and nuclei**	Three nuclear groups, and which one is the gate
3	**Input and output**	Two roads in, four autonomic routes out
3	**Disorders**	Anxiety, trauma, mood, psychosis, and the direction of change
2	**What treatment does**	Prefrontal control as the mechanism of extinction

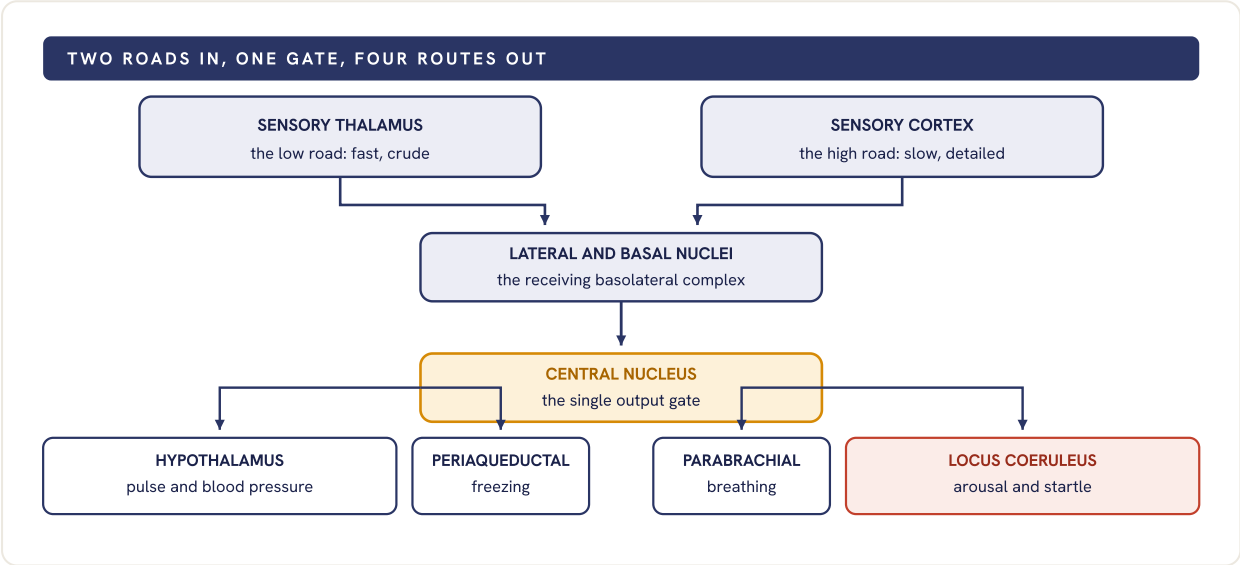


Fig 017.1 The fear pathway drawn as two afferent roads converging on the basolateral complex, one central output gate, and the four brainstem and hypothalamic routes that produce the bodily signs.

- **Position and nuclei.** An almond of grey matter in the medial temporal lobe, in front of the hippocampal head. Three groups: basolateral (input), centromedial (output), and cortical with the olfactory link.
- **What it computes.** Assigns emotional value to a stimulus, then drives the body to match. Fear conditioning is learned here, and the prefrontal cortex learns extinction by inhibiting it.
- **Across disorders.** Overactive to threat and to fearful faces in anxiety and post-traumatic stress with weak prefrontal restraint. Overactive to negative faces in depression. Reduced volume and abnormal face processing in schizophrenia and in autism.
- **The lesion picture.** Bilateral loss gives Kluver-Bucy syndrome, with placidity, hyperorality and loss of fear, and abolishes the startle potentiation that fear normally produces.

WHAT EARNS THE MARKS

- **The three nuclear groups, with their traffic direction.**
- **Two roads in and named routes out.** Each output tied to the sign it produces earns more than a list of nuclei.
- **The direction of change per disorder,** up or down, not simply involvement.

SEE ALSO Slot I - 20 limbic system in psychiatric disorders **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 18

Describe the neuroanatomy and cellular organization of cerebral cortex. Describe its functional circuitry with a suitable diagram. [3+3+4]

3 + 3 + 4 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gross organisation	Lobes, allocortex against neocortex, Brodmann mapping
3 Cellular organisation	Six layers, two cell classes, the column as the unit
4 Functional circuitry	The diagram: what enters at four, what leaves at five and six

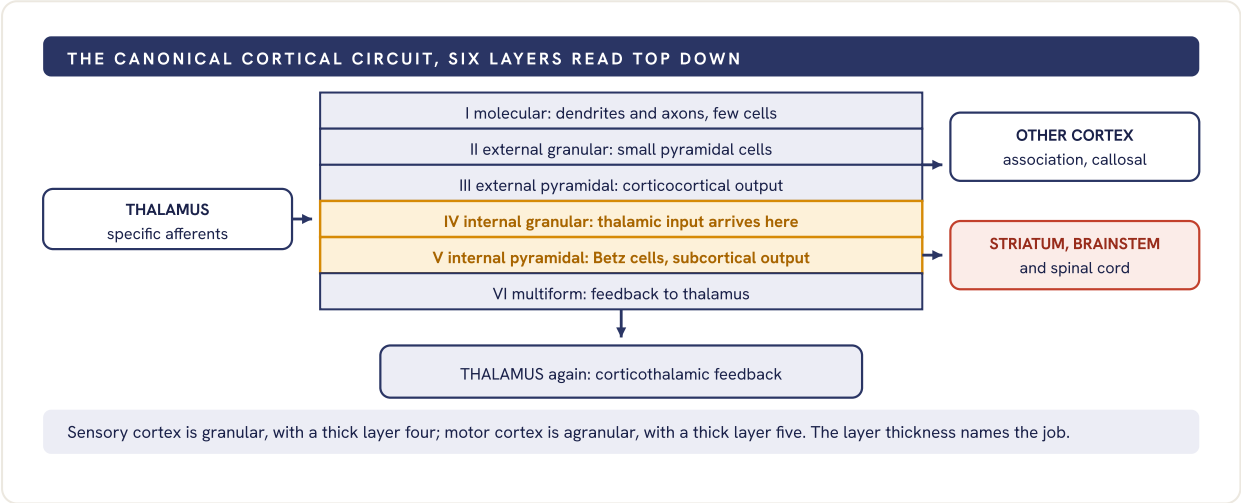


Fig 018.1 The six laminae with the traffic each carries: input at layer four, corticocortical output from layers two and three, subcortical output from layer five, thalamic feedback from layer six.

- **Gross organisation.** Neocortex is six layered and covers most of the surface; allocortex, meaning the hippocampal and olfactory cortex, has three layers. Brodmann divided the surface by cytoarchitecture, and those numbers are still the working map.
- **Two cell classes.** Pyramidal cells are excitatory, glutamatergic and projecting; interneurons are inhibitory, GABAergic and local. Parvalbumin interneurons pace gamma rhythm, and their loss is a leading account of cognitive symptoms in schizophrenia.

UNIT	WHAT IT IS	WHY IT MATTERS
Minicolumn	A vertical chain of about a hundred cells sharing one receptive field	The smallest repeating processing unit
Cortical column	Minicolumns grouped across all six layers	Same circuit everywhere, tuned by its input; a Brodmann number names a set of them

WHAT EARNS THE MARKS

- **Six layers named in order,** with the traffic of each, not simply the list.
- **The diagram, since the question asks for one.** Input at four, output at five, feedback at six is the whole circuit.
- **Granular against agranular cortex** explained by layer thickness rather than asserted.

SEE ALSO Slot I - 19 thalamic nuclei Slot I - 16 prefrontal cortex **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 19

Describe functions of Thalamus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The gateway to cortex, and the one sense that bypasses it
4 **Nuclei and their traffic**	Grouped by the lamina, each with its cortical target
2 **Beyond relay**	Gating, arousal, and the generation of sleep rhythms
2 **Clinical correlates**	Amnesia, thalamic pain, and the schizophrenia findings

NUCLEUS	MAIN INPUT	PROJECTS TO	WHAT ITS LOSS SHOWS
Anterior	Mammillary bodies, by the mammillothalamic tract	Cingulate gyrus	Amnesia, on the Papez ring
Mediodorsal	Amygdala, olfactory and limbic cortex	Prefrontal cortex	Amnesia, executive loss
Ventral posterior	Medial lemniscus, spinothalamic, trigeminal	Postcentral gyrus	Hemisensory loss, thalamic pain
Geniculate bodies	Optic tract, inferior colliculus	Visual and auditory cortex	Field defect, deafness
Reticular and intralaminar	Cortex and brainstem collaterals	Thalamus itself and diffuse cortex	Impaired arousal and attention

The reticular nucleus is the exception in the table: it is GABAergic, it projects to the thalamus rather than to cortex, and it is the gate the others pass through.

- **More than a relay.** Every sense but olfaction is routed here, and the relay is not passive: the reticular nucleus gates what reaches cortex, which is the anatomy behind selective attention.
- **Rhythm generation.** Thalamocortical loops with the reticular nucleus produce sleep spindles and the slow oscillations of deep sleep, so thalamic damage disturbs sleep architecture as well as sensation.
- **Psychiatric correlates.** Reduced mediodorsal volume and disrupted thalamocortical connectivity are among the more replicated structural findings in schizophrenia, and the pulvinar is implicated in the attention deficits.

THE LINE WORTH WRITING FIRST

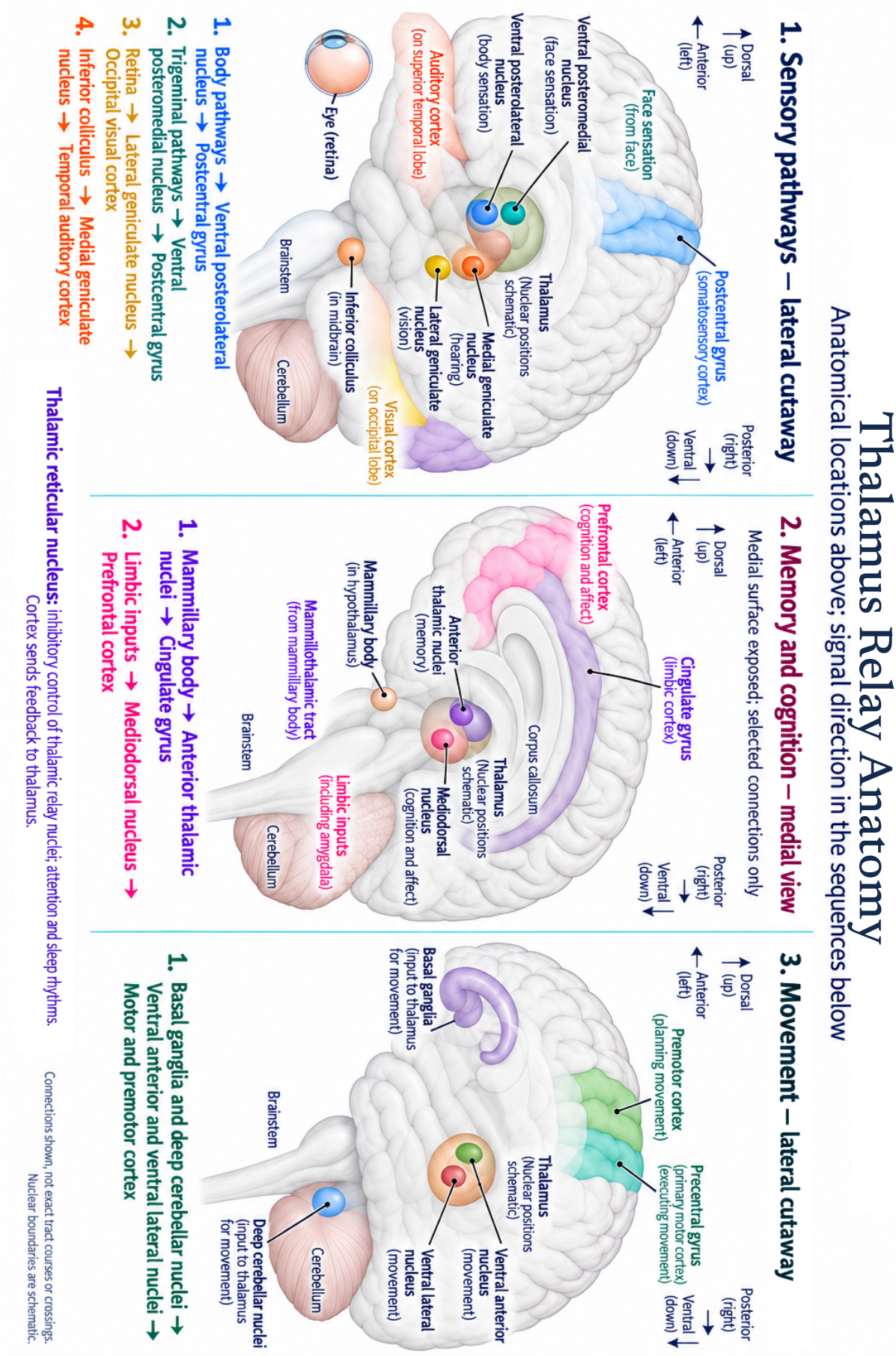
The thalamus is the obligatory gateway to the cerebral cortex for all sensory modalities except olfaction, and every cortical area sends a return projection to the thalamic nucleus that supplies it. Relay is reciprocal, never one way.

WHAT EARNS THE MARKS

- **The olfaction exception, stated as an exception.** It is the cheapest mark on the page.
- **Nuclei grouped, each with its cortical target.** A bare list of names without destinations is recall.
- **Gating and rhythm named alongside relay,** which is what turns a list into a function answer.
- **Two clinical anchors,** diencephalic amnesia and thalamic pain, each tied to its nucleus.

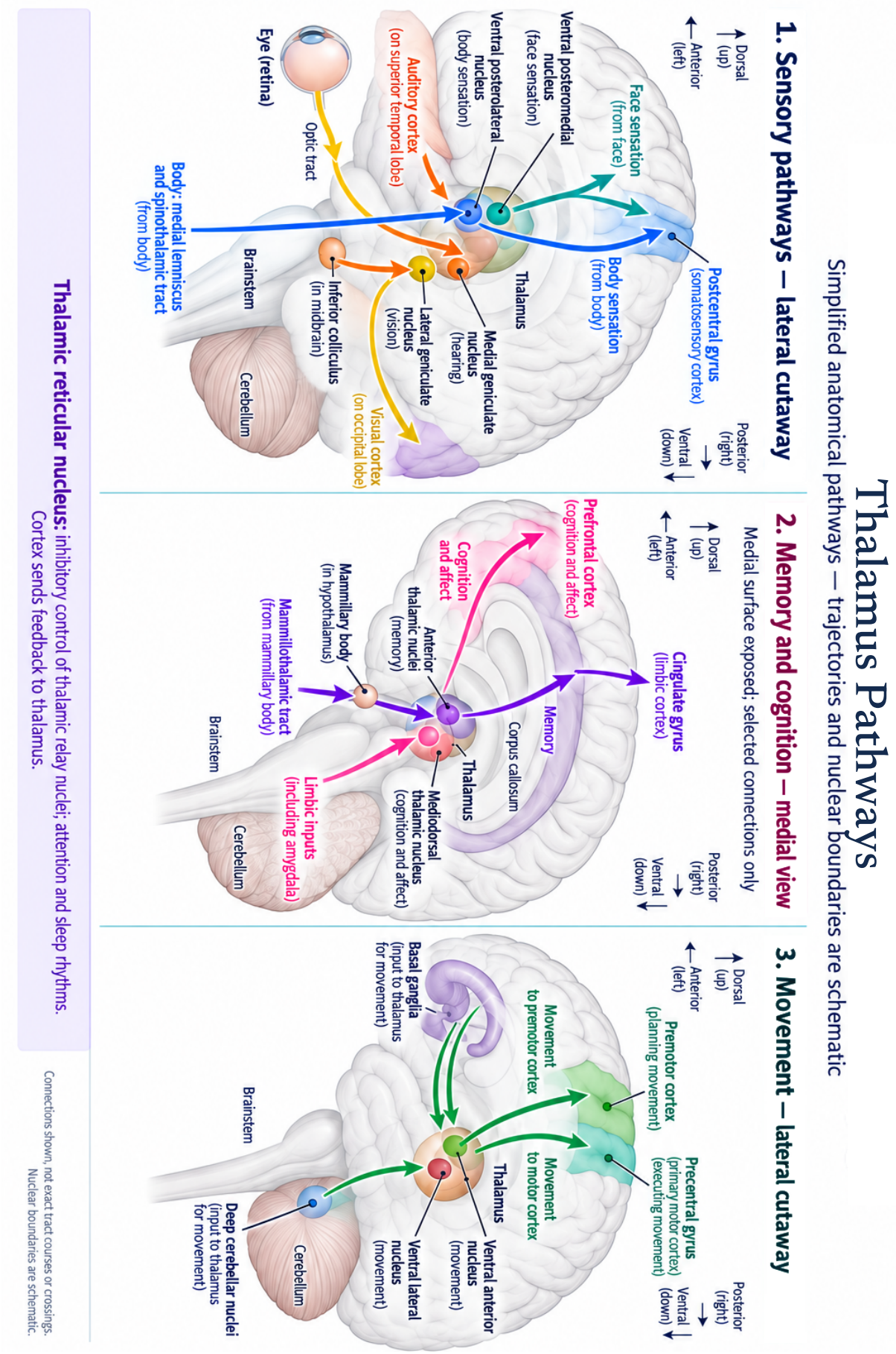
SEE ALSO Slot I - 15 Papez circuit Slot I - 18 cortical circuitry **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology



Thalamus Relay Anatomy. Use the labelled structures to connect anatomical location with function. Read with I-19.

Credits and references: retained within the illustration.



Thalamus Pathways. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with I-19.

Credits and references: retained within the illustration.

I - 20

a) Write about functional neuroanatomy of limbic system. b) Discuss role of limbic system in psychiatric disorders. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What the term covers	Broca to MacLean, and why the boundary is disputed
3	The components	Three tiers, cortical, subcortical, diencephalic
3	Disorder by disorder	Which node, which direction of change
2	What follows for treatment	Targets that are actually used in practice

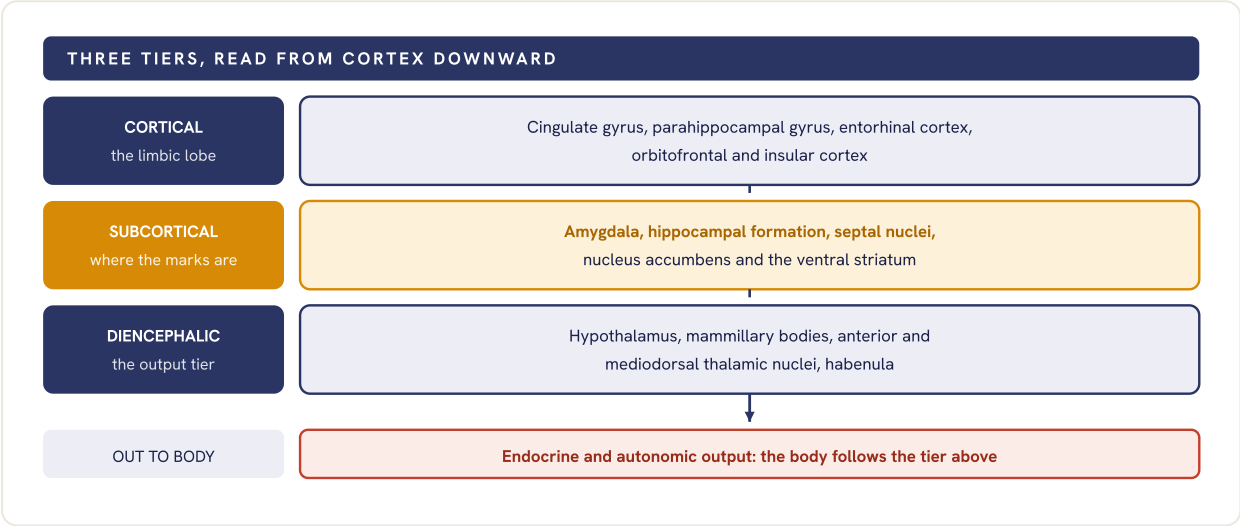


Fig 020.1 The limbic system laid out as three tiers rather than a ring, so that the cortical, subcortical and diencephalic members can be named separately and the output route stays visible.

- **What the term covers.** Broca named the limbic lobe as the ring of cortex around the corpus callosum; Papez gave it a circuit and MacLean gave it a name. The boundary has never been settled, so name your list before you use it.
- **Across disorders, and where they are targeted.** Amygdala overactivity with weak prefrontal restraint in anxiety and post-traumatic stress; smaller hippocampal volume in recurrent depression; subgenual cingulate overactivity in depression, where deep brain stimulation remains investigational; ventral striatal underactivity to reward in anhedonia and negative symptoms. Cingulotomy and capsulotomy are last resort options in severe refractory obsessive compulsive disorder.
- **Do not stop at the emotional brain.** It also carries episodic memory, olfaction, reward and endocrine control, and the triune brain framing MacLean built around it is no longer supported.

WHAT EARNS THE MARKS

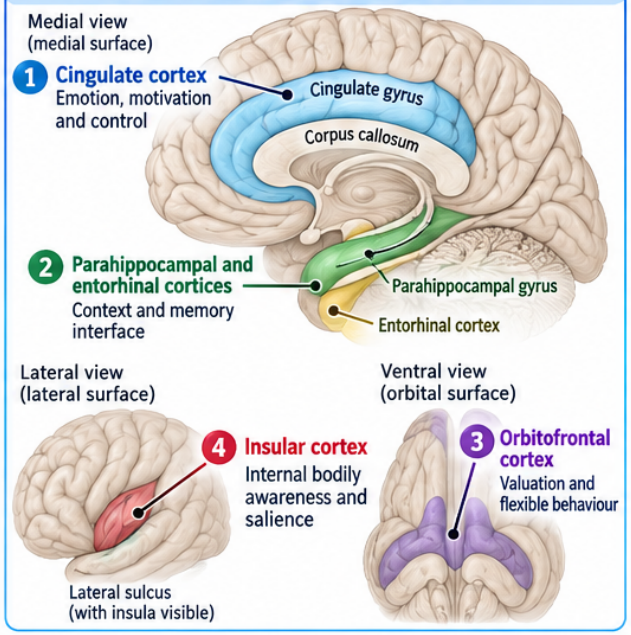
- **A defined membership list before any function.** Saying the boundary is disputed reads as command, not hedging.
- **Three tiers rather than one heap,** with the output route to hypothalamus named.
- **Direction of change per disorder.** Amygdala up, hippocampus down, ventral striatum down to reward.

SEE ALSO Slot I - 15 Papez circuit Slot I - 17 amygdala **SPRINT**

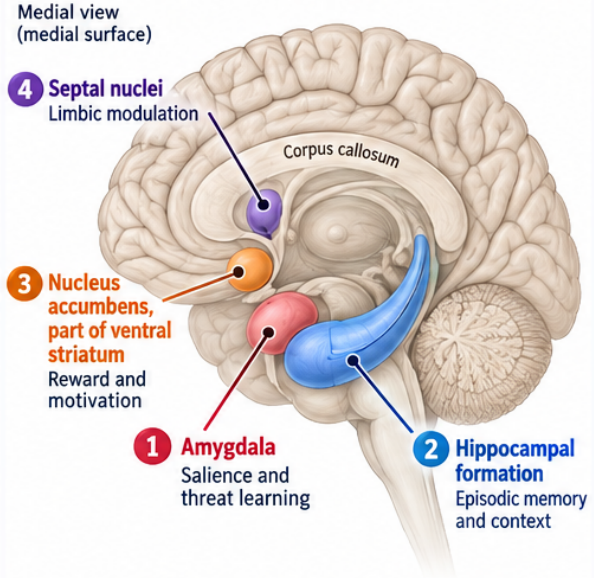
Day 1, Functional Neuroanatomy and Neurophysiology

Parts of Limbic System

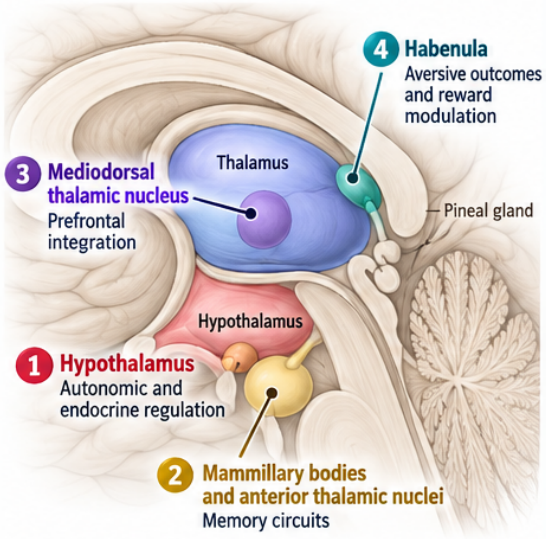
1. Cortical tier
Medial and lateral cortical structures



2. Subcortical tier
Medial temporal and basal forebrain structures



3. Diencephalic tier
Diencephalic structures (sagittal, magnified view)



Diencephalon is NOT brainstem.

What becomes dysregulated? Selected group-level findings, not diagnostic lesions.

1 Anxiety and post-traumatic stress disorder
Threat-reactivity and prefrontal–amygdala regulation altered; context and safety learning disrupted.

2 Depression
Mood-regulation networks altered; reduced reward responses; smaller hippocampal volume in some recurrent-depression groups.

3 Obsessive-compulsive disorder
Orbitofrontal–cingulate–striatal–thalamic loops implicated in persistent errors, urges and repetitive behaviour.

4 Addiction
Reward and habit learning, stress responses and prefrontal control become dysregulated.

5 Schizophrenia
Hippocampal–prefrontal–thalamic network dysfunction; blunted reward anticipation can accompany negative symptoms.

6 Diencephalic amnesia
Mammillary–anterior thalamic memory-circuit injury can impair new learning; example: Korsakoff syndrome.

Psychiatric disorders involve distributed networks. Findings vary by task, symptom, treatment and individual.
No one-to-one brain-area-to-disorder mapping. Three tiers are a teaching grouping, not three evolutionary brains.

Parts of Limbic System. Use the labelled structures to connect anatomical location with function. Read with I-20.

Credits and references: retained within the illustration.

I - 21

Describe the functional neuroanatomy of basal ganglia. Describe different parts of hippocampus. Discuss the psychiatric implications of basal ganglia and hippocampus. [3+2+5]

3 + 2 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Basal ganglia	The nuclei, then the direct and indirect pathways
2 Hippocampal parts	Dentate, the four CA fields, subiculum
5 Implications	Structure by structure, with the direction of the finding

THE ORGANISING IDEA

Both structures work as loops rather than as centres. Cortex projects to the striatum, the striatum acts on the pallidum and thalamus, and the thalamus returns to the same cortical area. The hippocampus runs a comparable loop through entorhinal cortex. Naming the loop is worth more than naming the parts.

- **Basal ganglia, the parts.** Caudate and putamen make the striatum, globus pallidus has an external and an internal segment, with subthalamic nucleus and substantia nigra completing the set.
- **The two pathways.** The direct pathway carries D1 receptors and releases movement; the indirect pathway carries D2 receptors and suppresses it. Antipsychotic blockade of striatal D2 receptors tips the balance toward suppression, which is parkinsonism.
- **Hippocampal parts.** Dentate gyrus, the cornu ammonis fields CA1 to CA4, and the subiculum, together with entorhinal cortex forming the hippocampal formation. CA1 is the field most vulnerable to hypoxia.

STRUCTURE	THE FINDING	WHERE IT IS SEEN	WHAT IT MEANS CLINICALLY
Caudate	Overactivity in the orbitofrontal loop	Obsessive compulsive disorder	Settles as the illness responds
Caudate	Head atrophy, boxcar ventricles	Huntington disease	Chorea with psychiatric onset
Striatum	D2 blockade above the therapeutic range	Antipsychotic treatment	Parkinsonism, akathisia, dystonia
Hippocampus	Reduced volume, most in CA1 and CA3	Recurrent depression, post-traumatic stress	Partly reversible with recovery

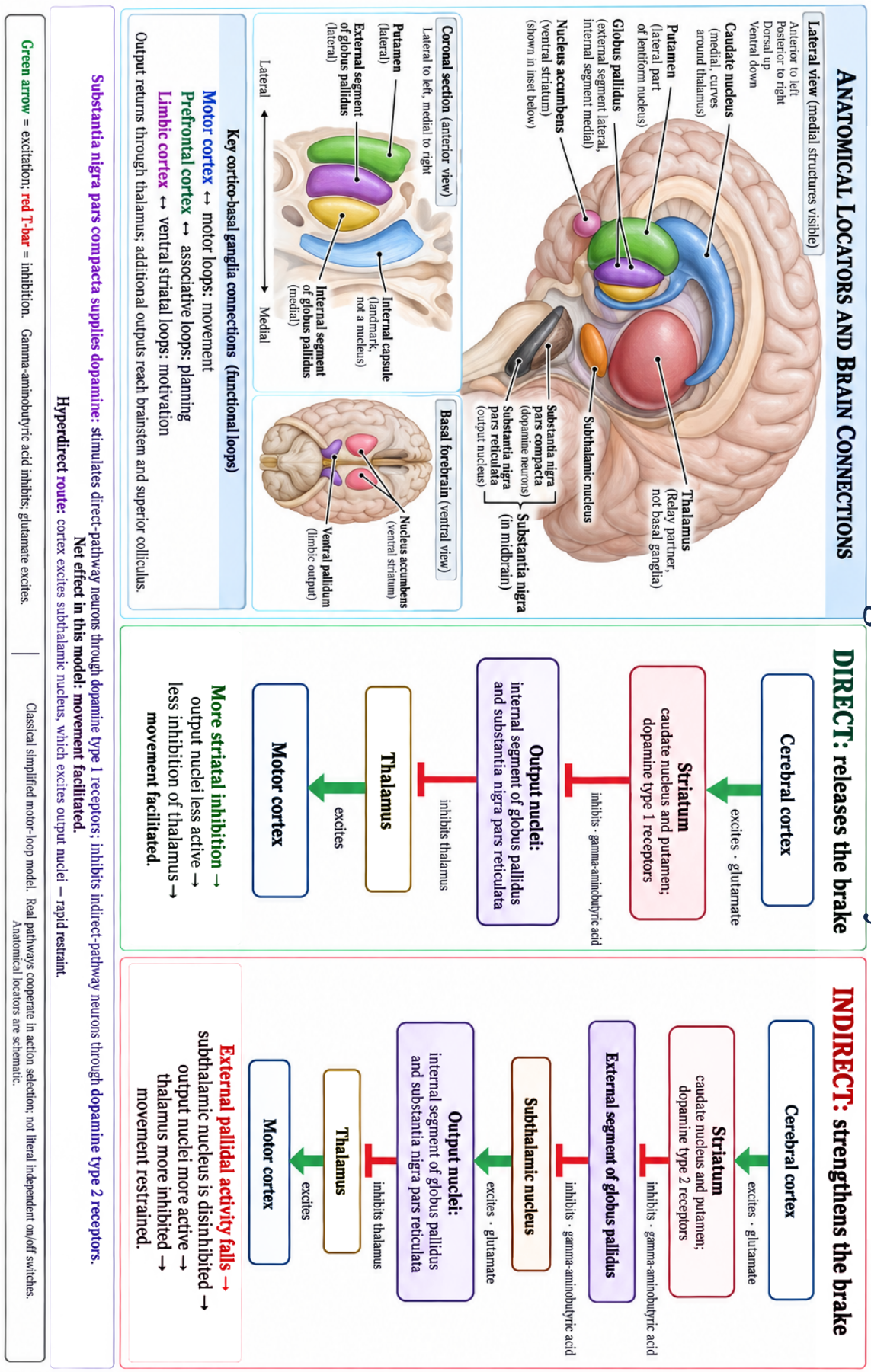
WHAT EARNS THE MARKS

- **Direct against indirect pathway, with D1 and D2 attached.** It is the line that explains extrapyramidal side effects.
- **All hippocampal parts named,** dentate, four CA fields and subiculum, with CA1 flagged as the vulnerable one.
- **Findings given a direction,** up or down, and a disorder. Involvement alone earns little.

SEE ALSO Slot I - 23 hippocampal subfields Slot I - 24 caudate nucleus **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

Basal Ganglia and Pathways



Basal Ganglia and Pathways. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with I-21.

Credits and references: retained within the illustration.

I - 22

Discuss the functions of frontal lobe with a suitable diagram. Write 3 syndromes associated with injury to frontal lobe. Describe signs and symptoms of parietal lobe atrophy. [3+4+3]

3 + 4 + 3 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Frontal functions	The diagram: motor at the back, executive in front
4 Three syndromes	Each named, sited, and given its picture
3 Parietal atrophy	Dominant against non-dominant, then the bilateral case

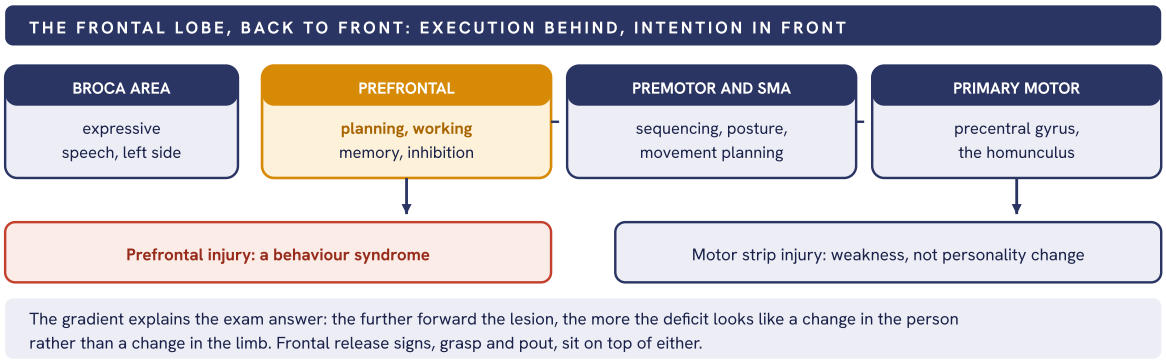


Fig 022.1 The frontal lobe as a back to front gradient, execution at the central sulcus and intention at the pole, with the two kinds of injury picture that follow from it.

SYNDROME	WHERE THE LESION SITS	THE PICTURE
Dysexecutive	Dorsolateral prefrontal	Poor planning and sequencing, perseveration, concrete thinking, reduced verbal fluency
Disinhibited	Orbitofrontal	Tactless, impulsive, jocular, socially coarse, utilisation behaviour, preserved formal testing
Apathetic	Medial frontal and anterior cingulate	Abulia, loss of initiative, sparse speech, akinetic mutism when the lesion is large and bilateral

- **Parietal atrophy, dominant side.** Gerstmann syndrome, meaning agraphia, acalculia, finger agnosia and right to left disorientation, with ideomotor apraxia and receptive language difficulty.
- **Parietal atrophy, non-dominant side.** Contralateral neglect and anosognosia, dressing and constructional apraxia, and loss of topographical sense. Bilateral posterior atrophy gives Balint syndrome: optic ataxia, oculomotor apraxia and simultanagnosia.

WHAT EARNS THE MARKS

- **The diagram, since the question asks for one,** drawn as a back to front gradient rather than a list of areas.
- **Exactly three syndromes, each with its site.** The question counts them, so the answer should too.
- **Parietal signs split by side** before the bilateral case. An unsided list loses the localising mark.

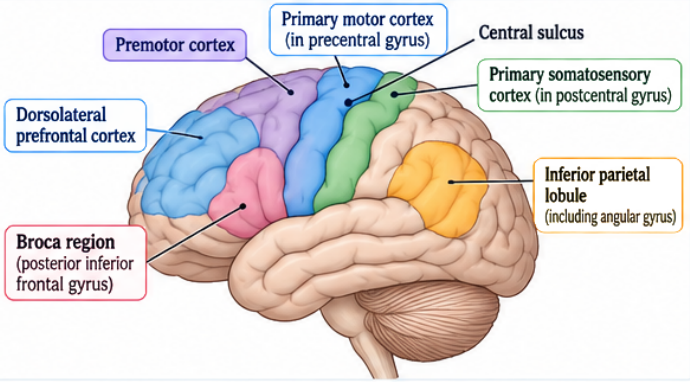
SEE ALSO Slot I - 16 prefrontal cortex Slot I - 25 occipital cortex **SPRINT**

Day 34, Stroke, TBI, delirium and focal brain syndromes

Frontal Lobe Syndromes

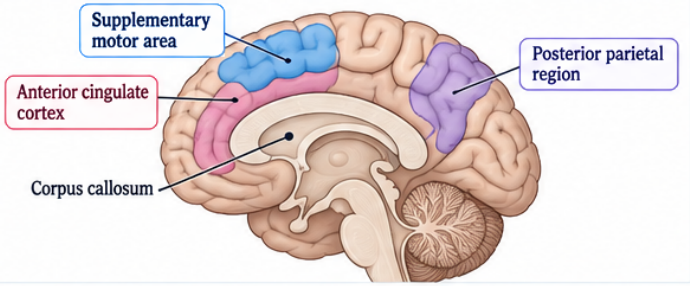
Left hemisphere, lateral view

Front (anterior) ← → Back (posterior)



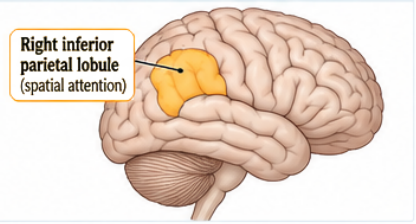
Left hemisphere, medial view

Front (anterior) ← → Back (posterior)



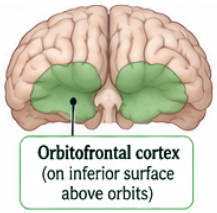
Right hemisphere, lateral view

Back (posterior) ← → Front (anterior)



Inferior (frontal) view

Front (anterior) view



THREE FRONTAL BEHAVIOURAL SYNDROMES

1. Dysexecutive — dorsolateral prefrontal

Poor planning and sequencing; perseveration (repeating a response); concrete thinking; reduced verbal fluency.

2. Disinhibited — orbitofrontal

Impulsivity, poor social judgement, inappropriate joking; utilisation behaviour (automatically using presented objects). Routine cognitive tests may miss deficits.

3. Apathetic — medial frontal and anterior cingulate

Abulia (reduced drive), little initiative or speech. Extensive bilateral injury can cause akinetic **mutism:** awake but scarcely moving or speaking.

DOMINANT PARIETAL — usually left

4. Gerstmann syndrome — inferior parietal network

- **Agraphia:** impaired writing
- **Acalculia:** impaired calculation
- **Finger agnosia:** difficulty identifying fingers
- **Left–right disorientation**

5. Associated dominant-side signs

- **Ideomotor apraxia:** cannot perform learned gestures despite adequate basic strength and comprehension.
- Language difficulty may accompany temporoparietal involvement.

NON-DOMINANT PARIETAL — usually right

6. Neglect and spatial dysfunction

- **Contralateral neglect:** misses opposite space, usually left
- **Anosognosia:** unawareness of deficit
- **Dressing apraxia:** difficulty orienting clothing
- **Constructional impairment:** difficulty copying or assembling
- **Topographical disorientation:** difficulty navigating.

BILATERAL PARIETO-OCCIPITAL

7. Bálint syndrome — THREE features

- **Optic ataxia:** inaccurate visually guided reaching —
- **Oculomotor apraxia:** difficulty directing gaze voluntarily
- **Simultanagnosia:** cannot perceive the whole scene at once.

OTHER LOCALISING SIGNS

- **Primary motor injury:** opposite-side weakness.
- **Dominant Broca-network injury:** nonfluent aphasia.
- **Parietal sensory cortex injury:** impaired cortical sensory discrimination.
- **Frontal release signs:** grasp and pout reflexes; limited specificity.

Patterns localise networks, not single isolated spots. Lesion extent, connections and language dominance alter presentation.
Atrophy does not guarantee every listed sign. Selected syndromes relevant to Question 22.

Frontal Lobe Syndromes. Use the labelled structures to connect anatomical location with function. Read with I-22.

Credits and references: retained within the illustration.

Lobe Function Tests

Frontal Lobe

Executive function, behaviour and motor programming

Bedside tests

- Verbal fluency (language and education dependent)
- Alternating hand sequences (fist–edge–palm; Luria)
- Go/no-go inhibition
- Similarities and abstraction
- Set shifting (Trail Making Test B)
- Working memory (digit span backward)

Functional subdivisions and typical deficits

- **Dorsolateral prefrontal cortex** – executive dysfunction (poor planning, cognitive flexibility)
- **Orbitofrontal cortex** – disinhibition (inappropriate behaviour, poor social judgment)
- **Medial frontal cortex** – apathy / abulia (reduced initiation and drive)

Alternating hand sequence (fist–edge–palm)



Fist Edge Palm

Demonstrate the sequence, then ask the patient to repeat it with one hand.

Go / no-go inhibition



No-go (withhold response)

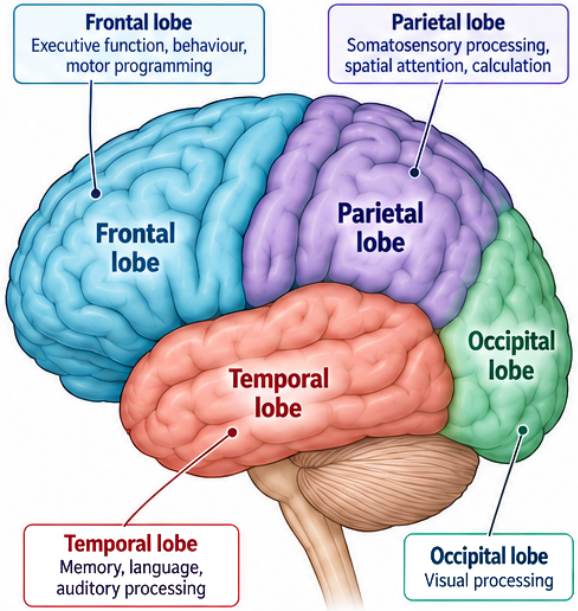


Go (make response)

Verbal fluency

"Tell me as many animals as you can in one minute."

Language and education dependent.



Left lateral view

Parietal Lobe

Somatosensation, spatial processing and calculation

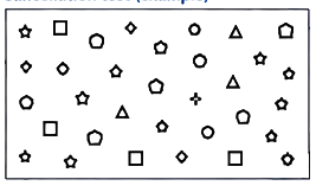
Dominant (usually left) parietal lobe

- Calculation
- Writing (agraphia)
- Finger recognition
- Right–left orientation
- **Gerstmann tetrad:**
  - Agraphia (writing)
  - Acalculia (calculation)
  - Finger agnosia (finger recognition)
  - Right–left disorientation

Non-dominant (usually right) parietal lobe

- Line bisection and cancellation
- Double simultaneous stimulation (for neglect and extinction)
- Spatial attention and awareness

Cancellation test (example)



Ask the patient to cross out all the target stars (☆).

Double simultaneous stimulation (example)



Touch one side Touch both sides together

Failure to report one of the touches suggests neglect or extinction.

Higher somatosensory tests

- Test stereognosis (identify object by touch) and graphesthesia (recognise drawn number/letter on skin) only after primary sensation is intact.

Temporal Lobe

Memory, language and auditory processing

Bedside tests

- Delayed verbal learning and recall (dominant medial temporal lobe)
- Nonverbal memory (often non-dominant temporal lobe)
- Naming (e.g. objects, pictures)
- Word comprehension (dominant temporal language network)
- Auditory recognition (e.g. environmental sounds, voices)

Examples

Delayed verbal learning

"I am going to tell you three words: apple, table, coin. Please repeat them and try to remember them for later."

Naming



"What is this?"

Auditory recognition



"What sound is this?" (e.g. dog barking, telephone)

Worked bedside sequence

A structured approach to cognitive testing

1. **Attention**
   e.g. alertness, sustained attention (spell a word backwards, months in reverse)
2. **Language**
   e.g. fluency, naming, comprehension, repetition
3. **Primary sensation and vision**
   e.g. hearing, visual acuity, confrontation fields, somatic sensation, motor function
4. **Higher cortical tasks**
   e.g. memory, praxis, visuospatial function, executive tests

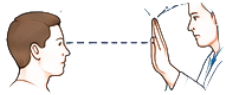
Occipital Lobe

Visual processing

Bedside tests

- Confrontation visual fields (test first)
- Color recognition
- Object recognition
- Interpret complex visual scenes (visual agnosia)

Confrontation visual fields (example)



Ask the patient to look at your nose and report when they see your moving finger.

Key points

- Use color and object recognition tasks to distinguish higher visual dysfunction from acuity or field loss.
- Homonymous field loss may arise anywhere retrochiasmally (optic tract, lateral geniculate nucleus, optic radiations or occipital cortex).
- Prosopagnosia (impaired face recognition) involves occipitotemporal networks, not a pure occipital lobe sign.

Critical points

- These are distributed network tests, not one-test-one-lobe diagnoses.
- Delirium, sensory loss, motor impairment, language and education can all confound results.
- Always consider alternative explanations for poor performance.
- Use a combination of tests and clinical context to localise dysfunction.
- Findings suggest which networks are likely involved, rather than providing definitive localisation.

Lobe Function Tests. Use the labelled structures to connect anatomical location with function. Read with I-22.

Credits and references: retained within the illustration.

I - 23

What is hippocampal formation? Discuss its role in learning. Describe hippocampal subfield volumes and its use in psychiatry. [2+3+5]

2 + 3 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Dentate, CA1 to CA4, subiculum, entorhinal: a formation, not a nucleus
3 Role in learning	The trisynaptic circuit, long term potentiation, consolidation
5 Subfield volumetry	How it is measured, what it shows, and what it cannot do

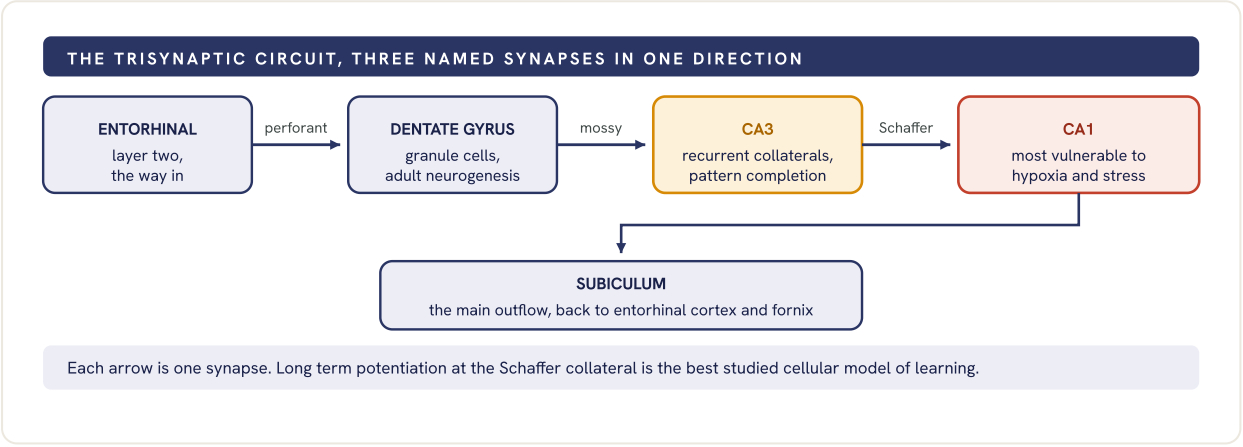


Fig 023.1 The trisynaptic circuit as a one way chain, with the field that completes patterns in amber and the field that dies first in red.

- **Role in learning.** It binds a new episode into a single trace, then hands it to neocortex over months, which is systems consolidation. Damage gives dense anterograde amnesia with graded retrograde loss and intact procedural learning, the pattern first described in the patient H M.

SUBFIELD VOLUMETRY: WHAT IT SHOWS, AND WHAT IT CANNOT

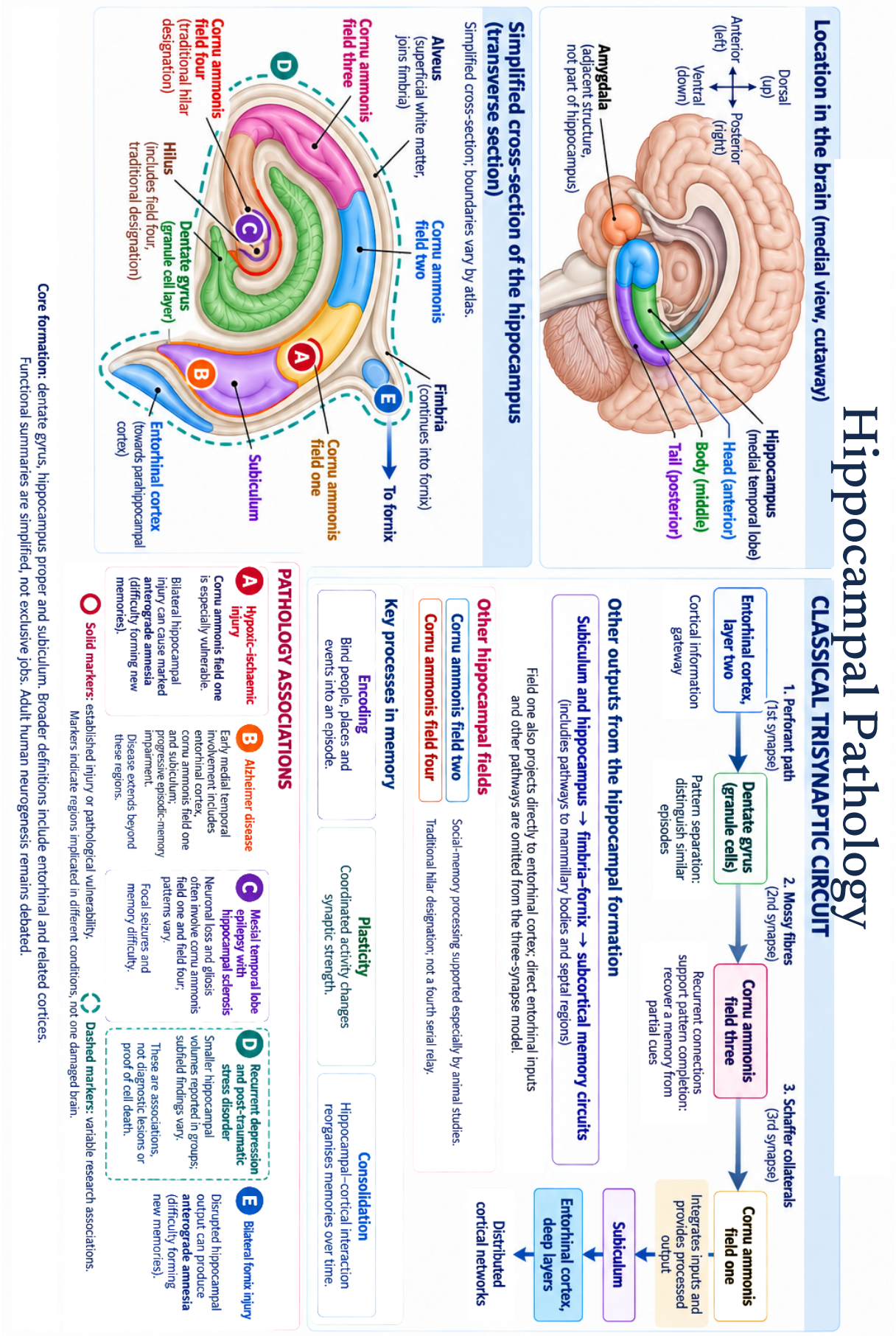
High resolution imaging with automated segmentation gives per field volumes rather than one hippocampal number. CA1 and subiculum loss tracks early Alzheimer disease; CA3 and dentate loss tracks chronic stress and recurrent depression. But values overlap heavily between the ill and the well, and depend on the software and field strength. It does not diagnose an individual, and saying so is a mark.

WHAT EARNS THE MARKS

- **The formation listed as a set,** with entorhinal cortex included.
- **Three synapses named,** perforant path, mossy fibres, Schaffer collaterals, with long term potentiation attached.
- **Subfields tied to specific conditions,** with the limits of the measure stated. An answer that claims diagnostic use has overreached.

SEE ALSO Slot I - 21 basal ganglia and hippocampus Slot I - 13 synaptic plasticity **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology



Hippocampal Pathology. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-23.

Credits and references: retained within the illustration.

I - 24

Describe Caudate nucleus and its functions. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Anatomy	Shape, parts, relations, and what it is made of
2 Connections	Where its input comes from and where its output goes
4 Functions by loop	Motor, executive, limbic and oculomotor, one line each
2 Clinical correlates	The four conditions that turn on this nucleus

LOOP	CORTEX IT SERVES	WHAT THE LOOP DOES	WHEN IT FAILS
Motor	Motor and premotor cortex	Selects and releases a wanted movement	Chorea, bradykinesia
Executive	Dorsolateral prefrontal cortex	Set shifting, habit learning, sequencing	Perseveration, poor planning
Limbic	Orbitofrontal and anterior cingulate	Weighs reward, stops an unwanted act	Obsessions, compulsions, apathy
Oculomotor	Frontal eye fields	Directs voluntary saccades	Impaired saccade control

One nucleus, four parallel loops. Naming the loop that failed is what turns a movement sign or a psychiatric sign into the same explanation.

- **Anatomy.** A C-shaped grey mass with head, body and tail, curving along the lateral ventricle. The head bulges into the frontal horn; the tail ends near the amygdala. It joins the putamen across the internal capsule through grey bridges, and together they are the striatum.
- **Cells and transmitters.** Mostly GABAergic medium spiny neurons, receiving glutamate from cortex and dopamine from the substantia nigra pars compacta, with local cholinergic interneurons. D1 cells begin the direct pathway, D2 cells the indirect.
- **The four clinical anchors.** Head atrophy with boxcar ventricles in Huntington disease, caudate and orbitofrontal overactivity in obsessive compulsive disorder, caudate involvement in Sydenham chorea, and reduced volume in Tourette syndrome.

PITFALL

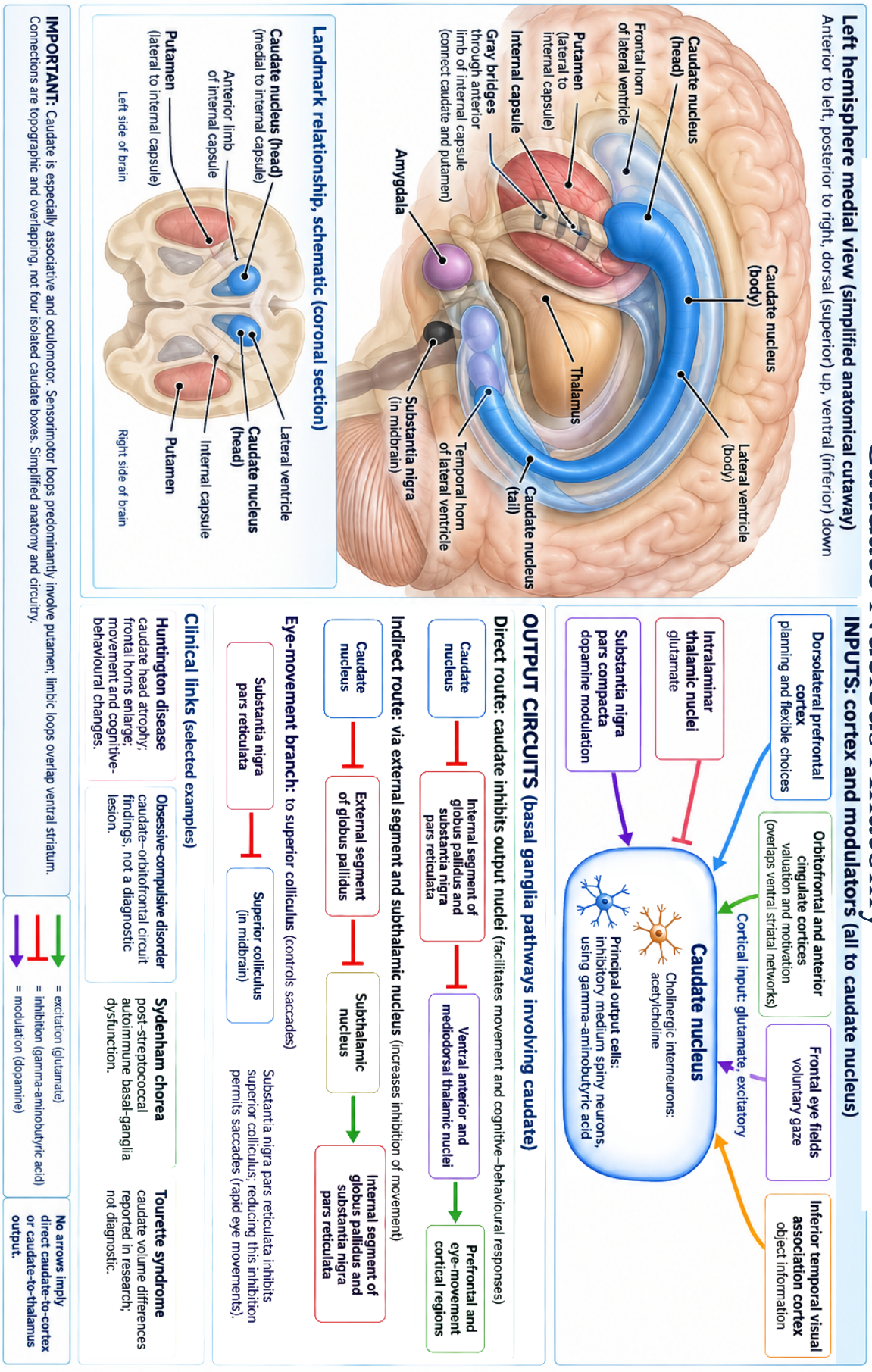
Do not treat the caudate as a purely motor structure. Its largest loops serve prefrontal cortex, which is why a caudate answer in a psychiatry paper must reach obsessive compulsive disorder, apathy and habit learning, and not stop at chorea.

WHAT EARNS THE MARKS

- **The C shape with head, body and tail,** and its relation to the lateral ventricle.
- **Medium spiny neurons with their three inputs.** Cortical glutamate, nigral dopamine, local acetylcholine.
- **Four loops, each with its cortex,** which is the structure of the four-mark half.
- **Both a movement disorder and a psychiatric disorder** among the clinical correlates.

SEE ALSO Slot I - 21 basal ganglia Slot I - 16 prefrontal cortex SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology



Caudate Nucleus Anatomy. Use the labelled structures to connect anatomical location with function. Read with I-24.

I - 25

Functions of the occipital cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Anatomy**	Striate and extrastriate cortex, and the retinotopic map
3	**The two streams**	Dorsal where, ventral what, and what each computes
3	**Lesion syndromes**	Field defects, then the agnosias, sited
2	**Psychiatric relevance**	Visual hallucination, and what its shape tells you

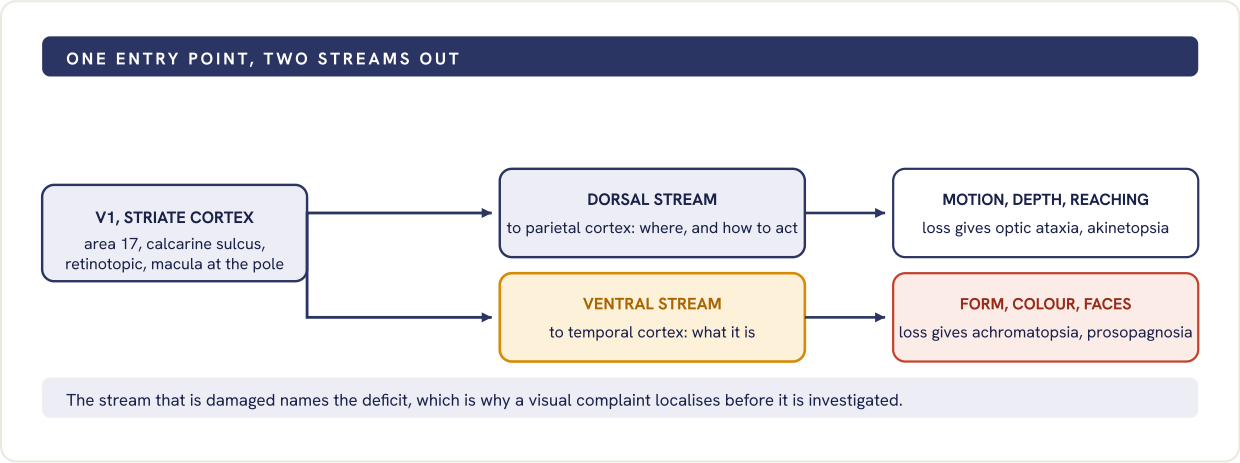


Fig 025.1 Visual cortex as one retinotopic entry point feeding two streams, each with the recognisable deficit that follows when it is cut.

- **Anatomy.** Striate cortex, area 17, lines the calcarine sulcus and is named for the stria of Gennari, an unusually thick layer four. Extrastriate areas 18 and 19 surround it and carry the higher processing.
- **Psychiatric relevance.** Occipital and temporal lesions give formed visual hallucinations with insight preserved. Charles Bonnet syndrome, formed hallucinations in an eye-diseased older person with intact cognition, is the presentation not to mistake for psychosis.

LESION	THE SIGN	WHAT IT TELLS YOU
Unilateral occipital	Homonymous hemianopia with macular sparing	Posterior cerebral territory, dual blood supply at the pole
Bilateral occipital	Cortical blindness, sometimes denied	Anton syndrome, blindness with anosognosia
Left occipital with splenium	Alexia without agraphia	A disconnection, not a language cortex lesion
Ventral occipitotemporal	Prosopagnosia, achromatopsia	The what stream, not the eye

WHAT EARNS THE MARKS

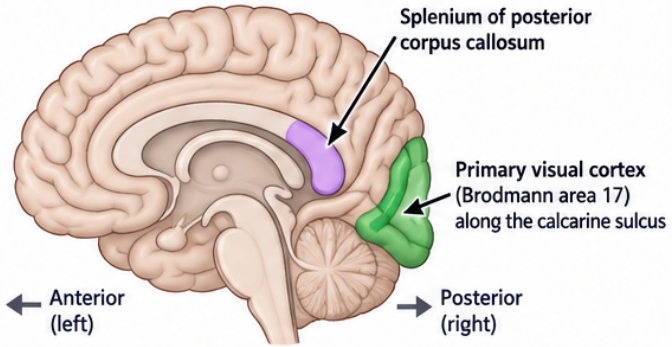
- **Retinotopy stated, with macular sparing explained.** It is the classic clinical consequence of the anatomy.
- **Two streams named and separated,** each with the deficit its loss makes.
- **Charles Bonnet syndrome named,** since this is a psychiatry paper and the differential is the point.

SEE ALSO Slot I - 14 corpus callosum Slot I - 22 parietal lobe signs SPRINT

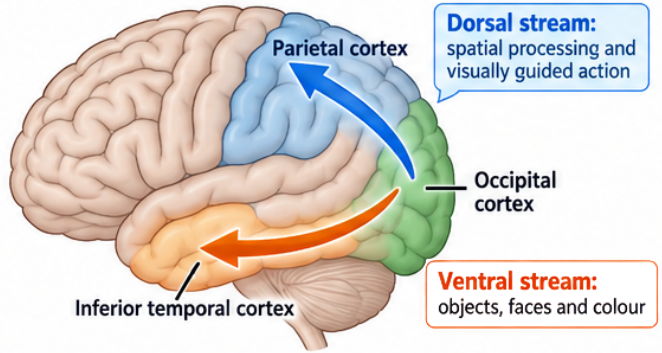
Day 1, Functional Neuroanatomy and Neurophysiology

Visual Cortex

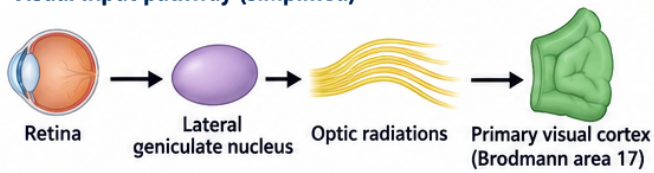
Medial view (inside surface of brain)
Anterior (left) → Posterior (right) Dorsal (up)



Lateral view (side of brain)
Anterior (left) → Posterior (right) Dorsal (up)



Visual input pathway (simplified)



1 Unilateral occipital injury

- Opposite homonymous hemianopia (same half-field missing in both eyes).
- Macular sparing may occur.

2 Bilateral occipital injury

- Cortical blindness (loss of conscious visual perception).
- Pupillary light reflexes often preserved.

3 Anton syndrome

- Cortical blindness with denial or unawareness of blindness.
- Confabulation may occur (patients may invent visual experiences).

4 Charles Bonnet syndrome

- Visual hallucinations with visual impairment and usually preserved insight (recognises hallucinations are not real).
- Visual release phenomenon, not a specific occipital lesion or necessarily psychosis.

5 Alexia without agraphia

- Cannot read but can write.
- Classic left occipital lesion with splenium of posterior corpus callosum disconnection (disconnects visual input from language areas).

6 Prosopagnosia

- Difficulty recognising familiar faces.
- Right or bilateral ventral occipitotemporal network.

7 Cerebral achromatopsia

- Loss of colour perception.
- Ventral occipital damage (ventral visual pathway).

8 Visual object agnosia

- Sees but cannot identify objects visually.
- Ventral visual network (occipitotemporal cortex).

9 Akinetopsia

- Impaired perception of motion (moving objects appear static or jumpy).
- Motion-sensitive extrastriate cortex (e.g. middle temporal visual area).

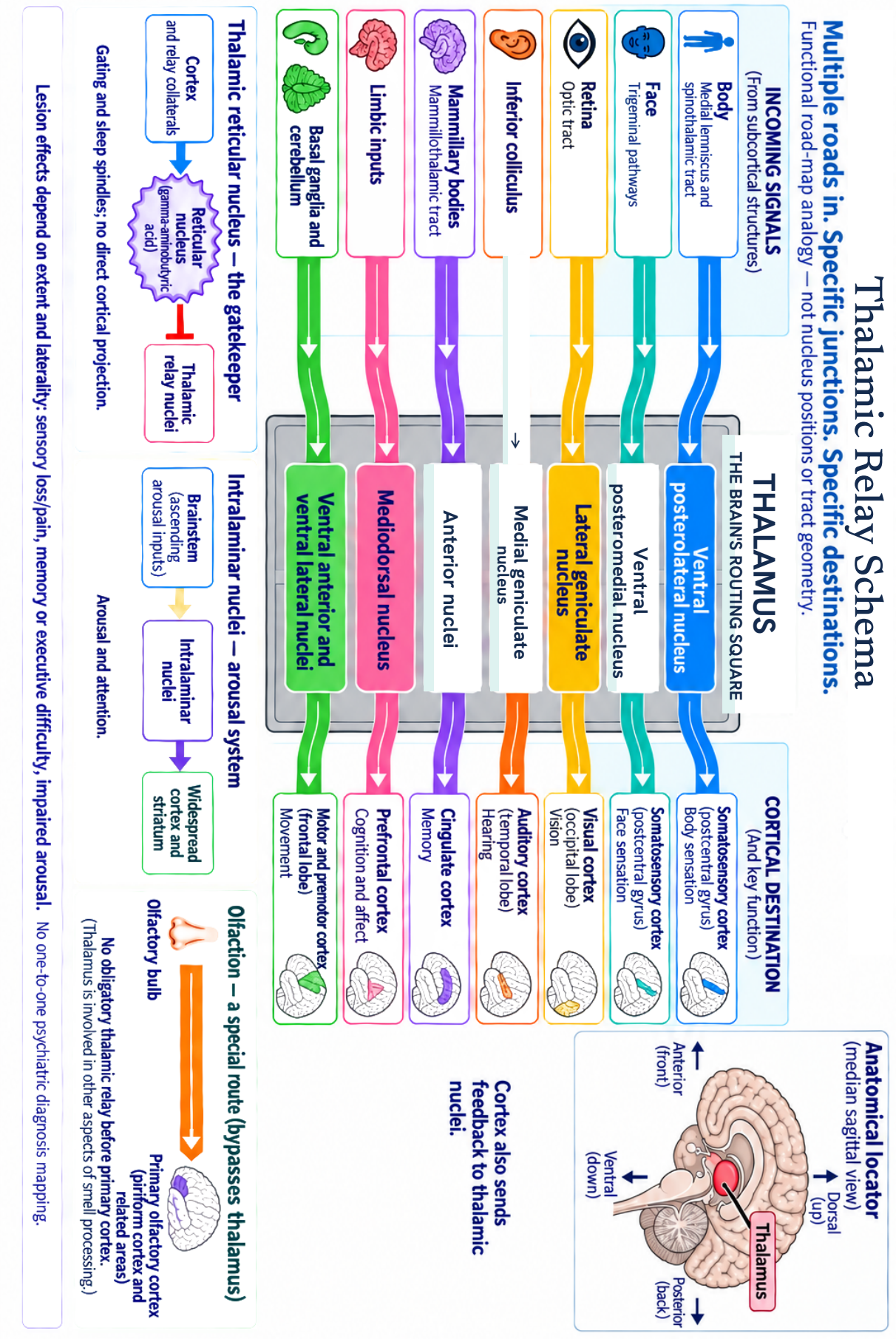
10 Bálint syndrome

- Bilateral parieto-occipital injury causing:
  - Optic ataxia (misreaching under visual guidance).
  - Oculomotor apraxia (difficulty directing gaze).
  - Simultanagnosia (cannot perceive the whole scene; only parts).

Simplified network locations, not exact atlas boundaries. Findings depend on extent and cause of injury.

Visual Cortex. Use the labelled structures to connect anatomical location with function. Read with I-25.

Credits and references: retained within the illustration.

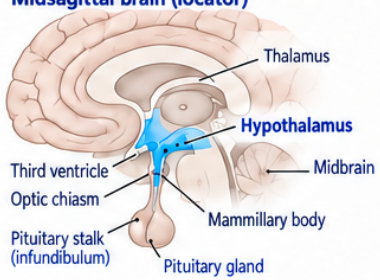


Thalamic Relay Schema. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Hypothalamus

Midsagittal brain (locator)



Nuclear groups — regional schematic (not an anatomical section)

Anterior / preoptic

Preoptic area: heat loss; reproductive control

Ventrolateral preoptic nucleus: sleep

Anterior nucleus: heat dissipation

Suprachiasmatic nucleus: circadian clock

Supraoptic and paraventricular nuclei: vasopressin and oxytocin

Tuberal / middle

Arcuate nucleus: energy sensing; dopamine inhibits prolactin

Ventromedial nucleus: satiety regulation

Dorsomedial nucleus: feeding, circadian and autonomic integration

Posterior

Posterior nucleus: heat conservation

Tuberomammillary nucleus: histamine; wakefulness

Mammillary nuclei: memory relay

Lateral hypothalamic area spans regions: feeding; orexin supports wakefulness

Medial–lateral zones, beside third ventricle

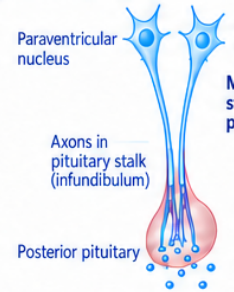
Periventricular: neuroendocrine	Medial: autonomic and metabolic	Lateral: feeding and arousal

Regional groupings simplify overlapping circuits.

Two pituitary routes

1. Neuronal route — posterior pituitary (vasopressin and oxytocin)

Large-cell neurons in supraoptic and paraventricular nuclei

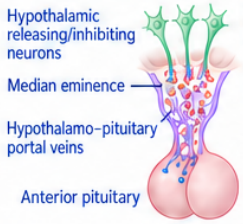


Made in hypothalamus; stored and released posteriorly.

Vasopressin: water retention
Oxytocin: uterine contraction and milk ejection

2. Humoral route — anterior pituitary (releasing and inhibiting hormones)

Small-cell hypothalamic neurons release hormones into the median eminence.



Releasing hormones stimulate pituitary axes.

Arcuate dopamine inhibits prolactin

Dopamine (from arcuate nucleus) ⊣ Prolactin

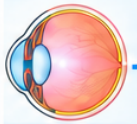
Example: stress axis

Corticotropin-releasing hormone (CRH) → Adrenocorticotropic hormone (ACTH) → Adrenal cortisol

Cortisol **inhibits** hypothalamic and pituitary drive.

Connections — simplified routes

Visual input to circadian clock



Retinohypothalamic tract → Suprachiasmatic nucleus

Retina

Light entrains the clock.

Memory circuit (Papez circuit component)



Fornix → Mammillary bodies → Mammillo-thalamic tract → Anterior thalamic nuclei

Hippocampus

Memory circuit.

Emotion to autonomic response



Stria terminalis / ventral amygdalofugal pathway → Hypothalamus → Brainstem and spinal autonomic centres

Amygdala

Emotion → bodily response (neuroendocrine and autonomic).

Lesion patterns and exam distinctions

Anterior injury
Impaired cooling (heat loss mechanisms) → hyperthermia.

Posterior injury
Impaired heat conservation → hypothermia.

Vasopressin pathway injury
Central diabetes insipidus (large volumes of dilute urine).

Lithium causes nephrogenic diabetes insipidus.

Orexin loss
Narcolepsy type 1 (cataplexy, sleep fragmentation).

Mammillary circuit injury
Amnesia (anterograde > retrograde, classically). Common causes include thiamine deficiency and Korsakoff syndrome.

Stalk damage and prolactin
Prolactin may rise (loss of dopamine inhibition) and other pituitary axes may fail.
Dopamine-blocking drugs raise prolactin without generally suppressing every other pituitary hormone.

Feeding lesions (classical)
Ventromedial nucleus → overeating. Lateral hypothalamic area → reduced eating.

Appetite uses distributed circuits, not two isolated switches.

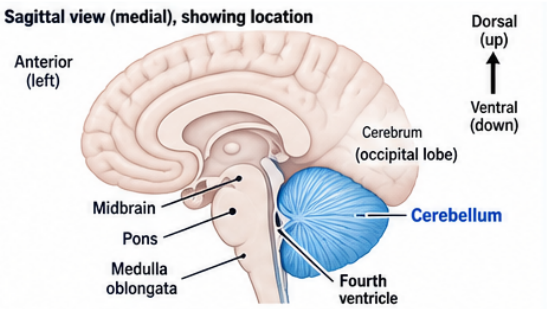
Hypothalamus. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

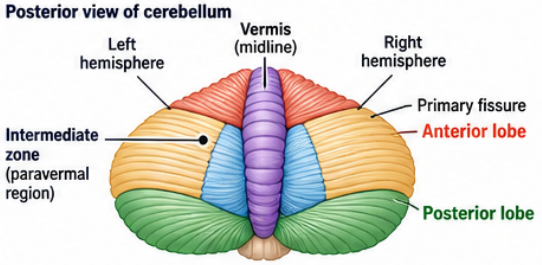
Cerebellum Anatomy and Pathology

1. ANATOMY

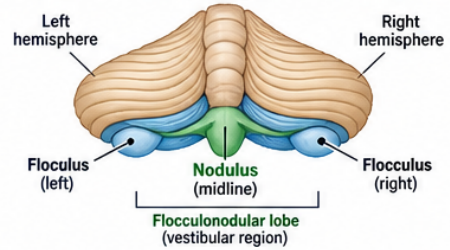
Sagittal view (medial), showing location



Posterior view of cerebellum



Inferior view of cerebellum (showing flocculonodular lobe)

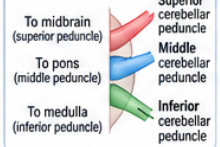


Deep cerebellar nuclei (medial to lateral)



Globose and emboliform = interposed nuclei

Cerebellar peduncles (lateral view, right side)



2. CONNECTIONS

Major functional routes (source → cerebellar region → output → role)

1. Vestibular / balance circuit

Vestibular apparatus and vestibular nuclei → Flocculonodular lobe (vestibular region) → Vestibular nuclei → Balance and gaze stabilization

Vestibular nuclei receive direct Purkinje-cell output.

2. Vermis / posture circuit

Spinal and brainstem sensory inputs → Vermis (midline) → Fastigial nucleus → Vestibular and reticular nuclei → Axial posture and gait

3. Intermediate zone / limb correction circuit

Spinal sensory information and cortical input → Intermediate zone (paravermal region) → Interposed nuclei (globose and emboliform) → Red nucleus and thalamus → Online limb correction

4. Cerebrocerebellar / cognitive circuit

Cerebral cortex (motor, premotor and association areas) → Pontine nuclei (opposite side) → Opposite lateral cerebellar hemisphere → Dentate nucleus → Thalamus (then to motor, premotor and association cortices) → Planning, timing and cognition

Inputs from the cerebral cortex cross in the pons.
The major output from the dentate nucleus crosses in the superior cerebellar peduncle to the opposite thalamus.

Cerebellar peduncles — key roles

Superior cerebellar peduncle	Mainly deep-nuclear output; also anterior spinocerebellar input.
Middle cerebellar peduncle	Cortical information via opposite pontine nuclei; input.
Inferior cerebellar peduncle	Mainly spinal, vestibular and inferior olivary inputs; also some outputs.

Key cellular and fibre principles

- Inferior olive → climbing fibres: teaching / error-related signals.
- Mossy fibres carry other major inputs.
- Purkinje cells inhibit deep or vestibular nuclei using gamma-aminobutyric acid (GABA); deep nuclei integrate this with excitatory inputs.

3. CLINICAL PATTERNS AND EXAMPLES

A. Vermis / midline



- Truncal and gait ataxia — wide-based unsteady gait.
- Alcohol-related degeneration often affects the anterior superior vermis.

B. Hemispheric motor regions



- Ipsilateral limb ataxia.
- Dysmetria (over/undershoot).
- Intention tremor.
- Dysdiadochokinesia (impaired rapid alternating movements).
- Dysarthria (imprecise speech).

C. Flocculonodular / vestibular circuits



- Imbalance, nystagmus and impaired gaze stabilization.

D. Posterior cognitive–affective networks



- Cerebellar cognitive affective syndrome (Schmahmann syndrome).
- Executive, visuospatial, language and affect-regulation deficits.
- Not explained by motor ataxia alone.

E. Postoperative cerebellar mutism syndrome



- Delayed reduced or absent speech and emotional lability, especially after childhood posterior fossa surgery.
- Cerebellar output-network disruption.

F. Causes across regions

- Cerebellar stroke, tumour, multiple sclerosis, hereditary or degenerative ataxias, toxins and immune-mediated disease.
- Each can affect several circuits.

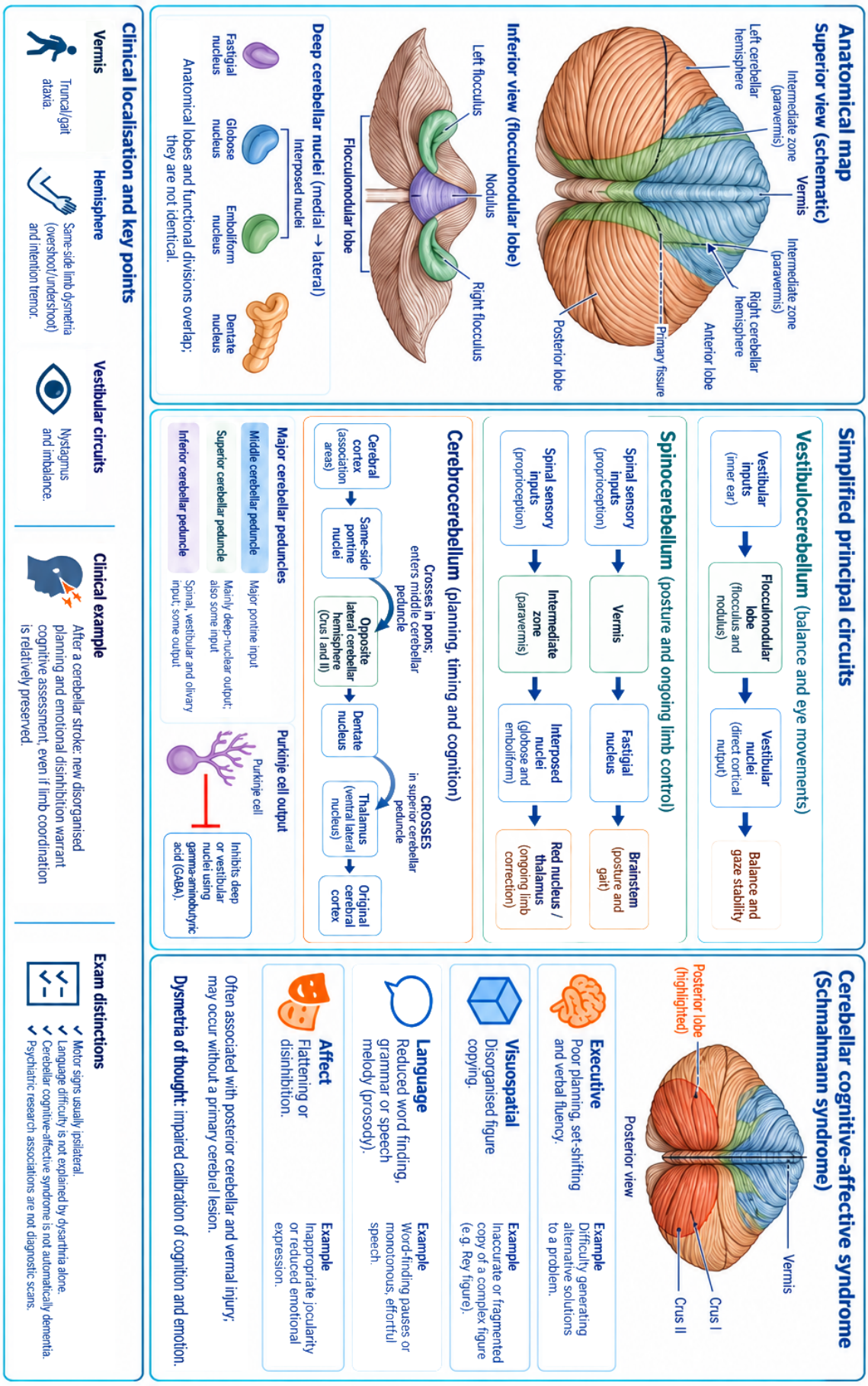
The cerebellum calibrates movement and thought; it does not directly command muscle contraction.

- Limb signs usually occur on the same side as the cerebellar lesion.
- Functional regions overlap; anatomical lobes are not identical to functional divisions.
- Simplified connectivity, not an exhaustive atlas.

Cerebellum Anatomy and Pathology. Use the labelled structures to connect anatomical location with function. Section companion: Functional neuroanatomy and neuroimaging.

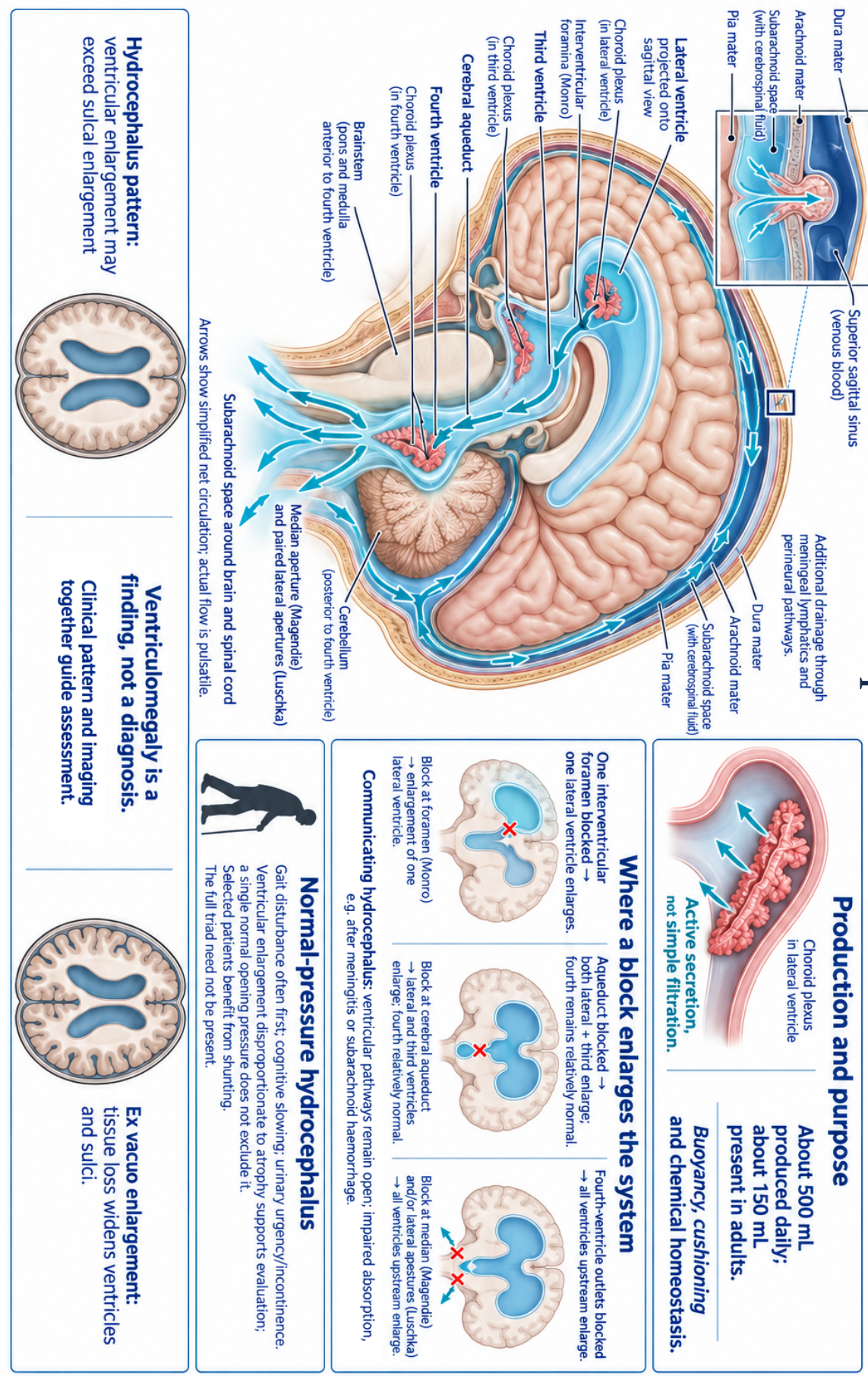
Credits and references: retained within the illustration.

Cerebellum



Cerebellum. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

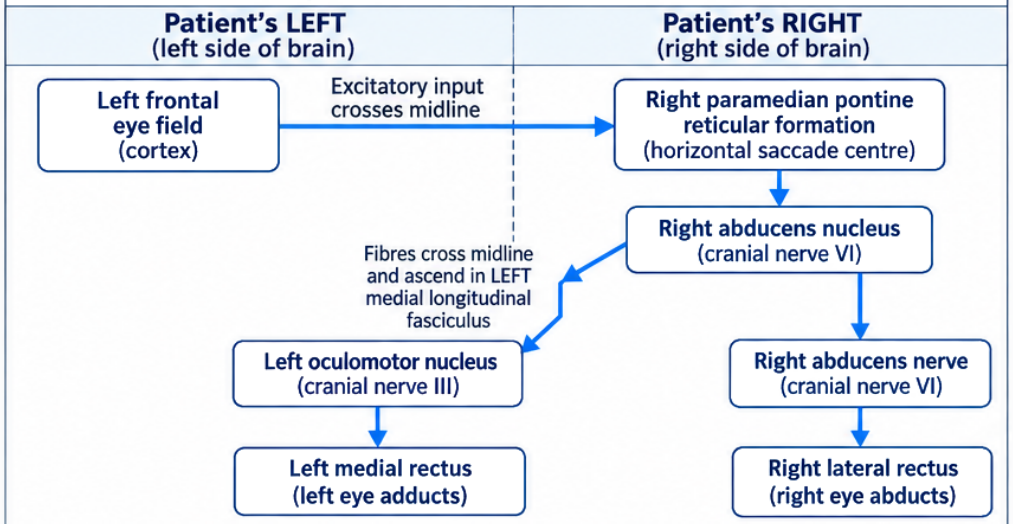


Cerebrospinal fluid. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

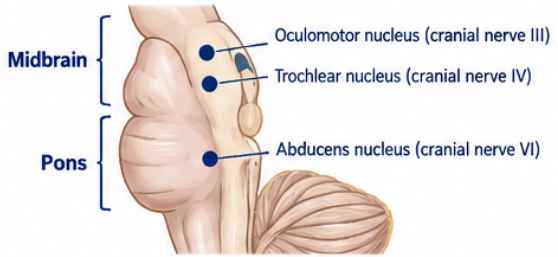
Eye-movement control

Example: looking to the patient's RIGHT



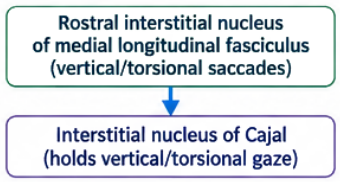
Adduction = *toward nose*; ***abduction*** = *away from nose.*

Brainstem anatomy (side view, patient's left side)



Trochlear fibres cross before exiting dorsally; each nerve supplies superior oblique muscle.

Vertical gaze control (schematic)



Saccade = rapid gaze shift.
Pursuit = tracking a moving target.
Vestibulo-ocular reflex stabilises gaze during head movement.

Clinical correlations (left-sided lesions)

1	**Left medial longitudinal fasciculus lesion → LEFT internuclear ophthalmoplegia**	Look right: left eye fails/slows adduction; right eye abducts with nystagmus. Convergence often preserved. *Named for the eye that fails to adduct.*
2	**Left abducens NERVE lesion**	Only left-eye abduction is weak.
3	**Left abducens NUCLEUS lesion**	Both eyes cannot look left. An isolated pontine saccade-centre lesion mainly impairs saccades; reflex gaze may be spared.
4	**Left one-and-a-half syndrome**	Left horizontal gaze centre + left medial longitudinal fasciculus. Neither eye looks left; on right gaze only right-eye abduction remains.
5	**Left frontal eye field**	Destructive lesion: gaze tends LEFT. Irritative seizure: gaze tends RIGHT. Typical cortical pattern (not seen with every lesion).
6	**Dorsal midbrain syndrome**	Upgaze palsy; pupils respond better to near than light; convergence-retraction movements. Consider pineal-region compression (e.g. tumour), but not all causes of vertical gaze palsy are due to a pineal tumour.

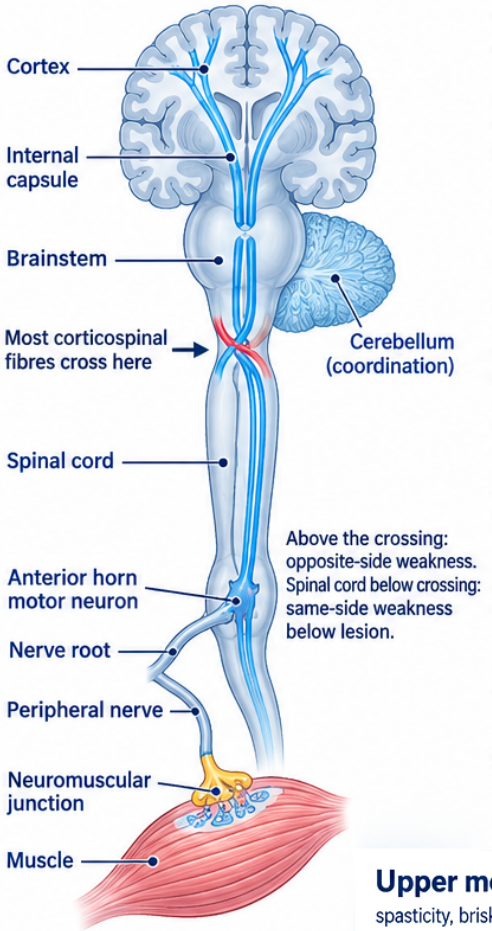
Exam distinctions: nucleus ≠ nerve; internuclear ≠ ocular muscle weakness. Multiple sclerosis and stroke are common causes of internuclear ophthalmoplegia; pattern alone does not establish cause. Abnormal pursuit in schizophrenia is a research association, not a diagnostic test.

Eye-movement control. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Bedside neurological localisation

Simplified motor pathway

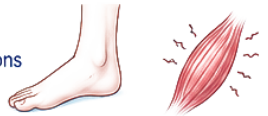


Finding	Likely site	Simple example
Weakness plus aphasia or neglect.	Cortex	Right arm weakness + impaired language → left dominant hemisphere.
Dense opposite face-arm-leg weakness without cortical signs.	Internal capsule	Left hemiparesis without aphasia/ neglect → consider right capsule; other motor-pathway sites remain possible.
Crossed cranial-nerve and limb signs.	Brainstem	Left whole-face weakness + right limb weakness → left pons.
Sensory level, sphincter symptoms, upper motor neuron signs below.	Spinal cord	Stiff weak legs + trunk sensory boundary → thoracic cord.
Myotomal weakness, dermatomal pain/sensory change, reduced corresponding reflex.	Root	Weak plantar flexion + reduced ankle jerk → consider first sacral root.
Weakness and sensory loss in one nerve distribution.	Peripheral nerve	Foot drop + weak eversion with preserved inversion → common fibular nerve.
Fluctuating fatigable weakness, usually intact sensation.	Neuromuscular junction	Variable ptosis and diplopia → consider myasthenia gravis.
Usually symmetrical proximal weakness without sensory loss.	Muscle	Trouble rising from chair and lifting arms → myopathy pattern.
Same-side limb incoordination.	Cerebellum	Right finger–nose overshoot + intention tremor → right cerebellar circuits.

Upper motor neuron pattern:
spasticity, brisk reflexes, extensor plantar response; marked early wasting unusual.



Lower motor neuron pattern:
reduced tone/reflexes, wasting, possible fasciculations (visible motor-unit twitches).



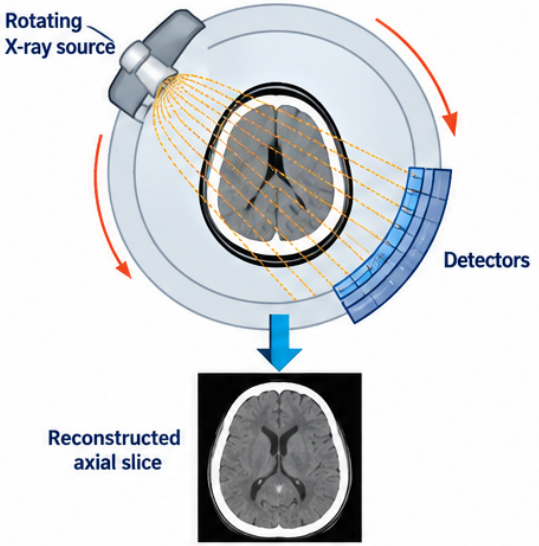
Examine strength, tone, reflexes, sensation, coordination and cramial nerves together. Acute central lesions may initially be flaccid.
A single sign suggests a site; it does not establish a cause. Sixth-nerve palsy can be a false localising sign of raised intracranial pressure.

Bedside neurological localisation. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Computed tomography

How CT works



X-ray attenuation → computer reconstruction

Ionising radiation. Fast acquisition.

Tissue appearance on CT

Air (very dark)	Cerebrospinal fluid (dark)	Brain (grey)	Skull (bone) (white)

Hounsfield scale (HU)

Air ≈ −1000

Water = 0

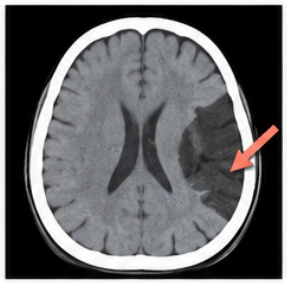
Bone strongly positive

Hyperdense = brighter; hypodense = darker

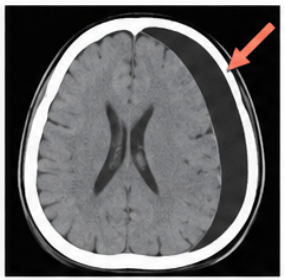
Illustrative scan patterns — not patient images



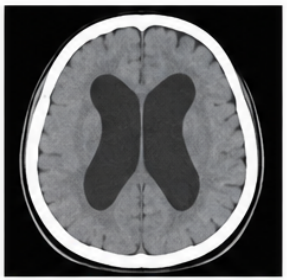
Acute haemorrhage: usually hyperdense (bright).



Ischaemia: early scan may be normal; later hypodensity and loss of grey–white distinction.



Chronic subdural collection: crescent outside brain; density varies with age and rebleeding.



Hydrocephalus / mass effect: enlarged ventricles, compressed sulci or shifted midline, depending on cause.

Ventriculomegaly needs clinical interpretation.

Why psychiatry uses it

- New confusion after a fall → subdural collection?
- Acute focal deficit or sudden severe headache → haemorrhage / stroke pathway?
- Rapid cognitive or personality change → structural lesion?
- Possible hydrocephalus → ventricular pattern?
- Used when urgency, access or magnetic resonance imaging constraints favour computed tomography.
- **Contrast is question-dependent:** Non-contrast study: acute blood assessment. Iodinated contrast may help assess masses, vessels or enhancement; consider kidney function and prior contrast reactions.

Strengths

- Speed.
- Accessibility.
- Acute blood.
- Bone and calcification.

Limitations

- Radiation.
- Less soft-tissue detail than magnetic resonance imaging.
- Posterior-fossa beam-hardening artefact.
- Subtle lesions and early ischaemia may be missed.

Exam mistakes

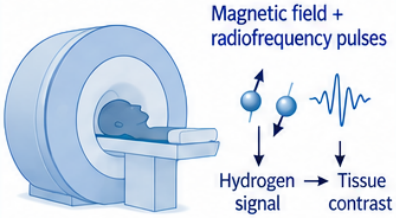
- Bright does not always mean blood (bone, calcium and contrast can be bright).
- A normal computed tomogram does not exclude encephalitis or early stroke.
- Enlarged ventricles do not diagnose schizophrenia.
- Imaging investigates structural causes; it does not independently diagnose a primary psychiatric disorder.

Computed tomography. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

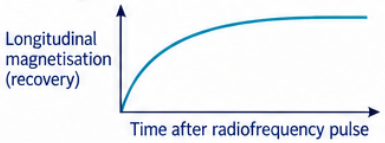
Structural magnetic resonance imaging

How MRI produces images

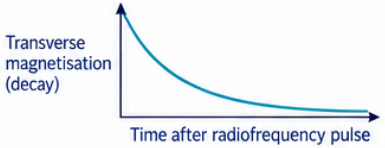


No ionising radiation.
Each sequence emphasises a different tissue property.

T1: longitudinal relaxation

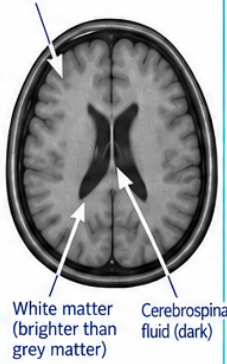


T2: transverse relaxation



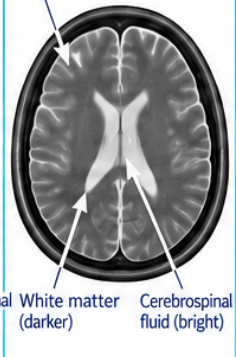
Illustrative sequence patterns — not patient scans

T1-weighted



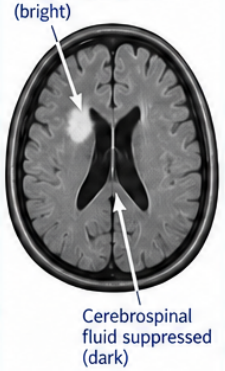
Anatomy, volume and tissue boundaries. Cerebrospinal fluid dark; white matter brighter than grey matter.

T2-weighted



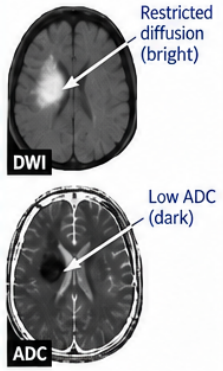
Fluid and oedema bright; many lesions conspicuous.

Fluid-attenuated inversion recovery (FLAIR)



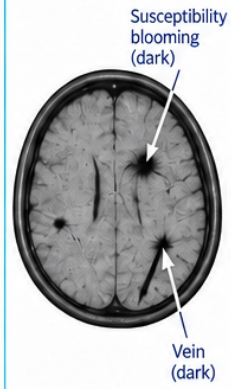
Suppresses free cerebrospinal fluid signal; tissue oedema remains bright. Useful near ventricles and cortex.

Diffusion-weighted imaging (DWI) + apparent diffusion coefficient (ADC) map



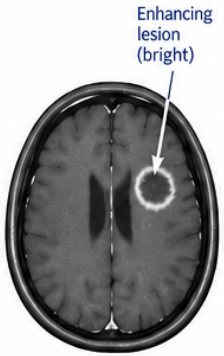
Restricted diffusion: bright diffusion image + low coefficient.
Acute infarction is one cause; abscess and other lesions can also restrict.

Susceptibility-weighted imaging (SWI)



Sensitive to susceptibility from blood products, veins and some mineral deposits; not specific for haemorrhage.

T1 after gadolinium



Enhancement reflects contrast distribution / barrier disruption; not a tumour diagnosis by itself.

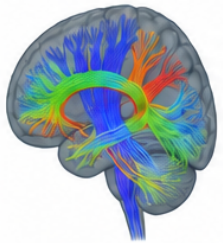
Clinical use in psychiatry



Investigate structural causes of cognitive, behavioural or psychiatric change: tumour, vascular injury, hydrocephalus, demyelination and selected encephalitis patterns.

Dementia assessment: regional atrophy supports the clinical picture.

Research extensions



Volumetry measures structure size.

Diffusion tensor imaging estimates directional water diffusion and reconstructs probable fibre trajectories.

Tractography is not a direct photograph of axons.

Limits and common errors

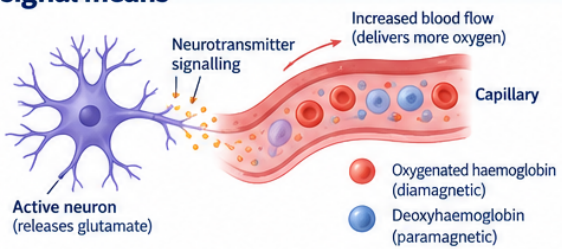
- Slower; motion and claustrophobia can impair study.
- Screen implants and devices for magnetic resonance compatibility.
- Gadolinium has separate indication and safety checks.
- Bright diffusion signal alone may be T2 shine-through; check coefficient map.
- White-matter hyperintensities and atrophy are nonspecific.
- A normal scan does not exclude all neurological disease.
- No structural pattern independently diagnoses schizophrenia or depression.

Structural magnetic resonance imaging. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Functional magnetic resonance imaging

What the blood-oxygen-level-dependent signal means

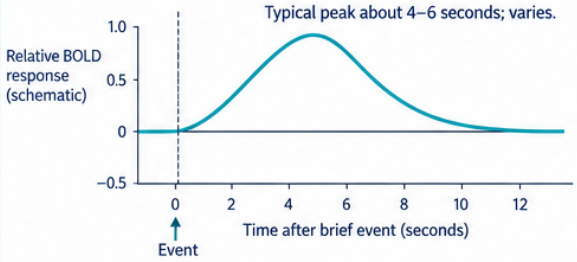


Neural activity → vascular response → blood flow rises more than oxygen use → less deoxyhaemoglobin → usually higher T2*-weighted signal.

Deoxyhaemoglobin = haemoglobin without bound oxygen; its magnetic susceptibility alters the signal.

Blood-oxygen-level-dependent (BOLD) contrast is an indirect haemodynamic measure, not a recording of spikes.

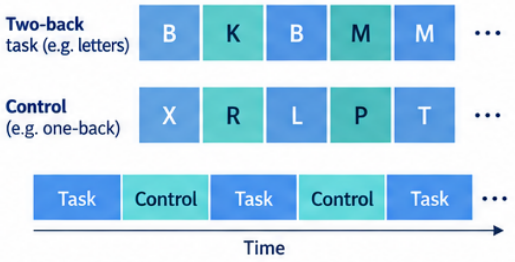
Typical BOLD response to a brief event



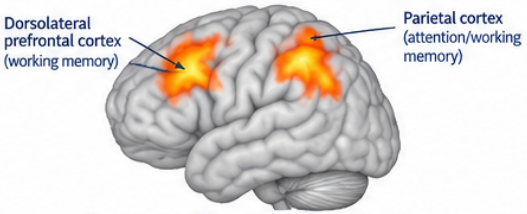
Task-based study

Compare two-back working memory with a control condition.

Two-back: identify a stimulus matching the one two steps earlier.



Task – control statistical contrast



Illustrative statistical maps — not patient scans

The map depends on the chosen baseline, performance and analysis threshold.

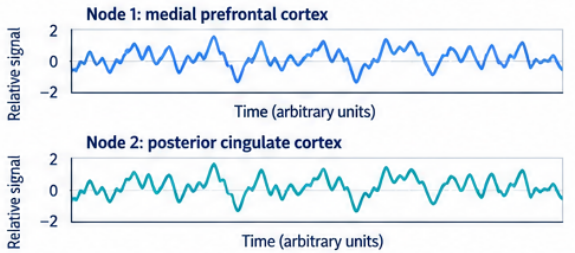
Clinical use: selected presurgical functional mapping.
Psychiatry: research on cognitive and emotional circuits.

Resting-state study

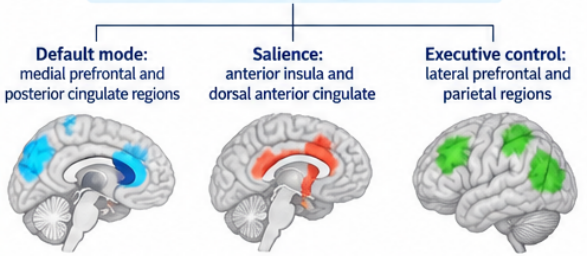
Measure spontaneous, low-frequency fluctuations in a relaxed awake state.



No explicit task; still requires staying sufficiently still and monitoring wakefulness.



Correlated spontaneous fluctuations → functional connectivity estimate



Illustrative statistical maps — not patient scans

What you may conclude / what you may not

Can:
compare task contrasts or statistical coupling under specified conditions.

Cannot:
read thoughts, infer a unique mental state from one active region, or establish causal communication from correlation.

Strengths:
useful spatial localisation; repeatable without ionising radiation.

Limits:
sluggish vascular response; motion, breathing, vascular disease, medication and task performance can confound results; multiple-comparison correction matters.

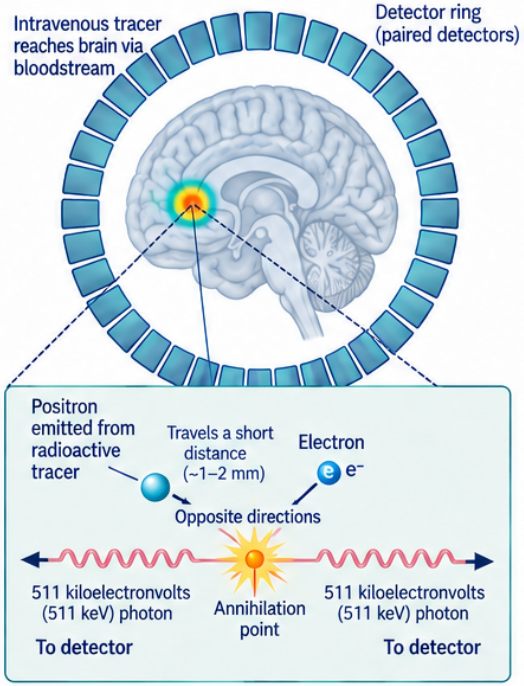
Exam traps:
activation is relative to a comparison; no coloured activation does not mean no neural function. Group differences do not independently diagnose schizophrenia, depression or attention-deficit/hyperactivity disorder.

Functional magnetic resonance imaging. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Positron emission tomography

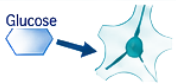
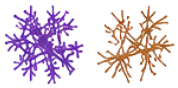
Principle and physics



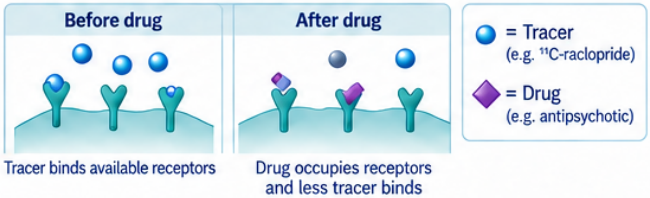
Coincidence detection reconstructs tracer distribution

Ionising radiation; tracer choice determines the biological question.

Common tracers and what they measure

Tracer (full name)	Illustration	Main measure
Fluorine-18 fluorodeoxyglucose (^{18}F-FDG)		Glucose uptake/metabolism.
Fluorine-18 fluorodopa (^{18}F-DOPA)		Presynaptic dopamine synthesis capacity.
Carbon-11 raclopride (^{11}C-raclopride)		Dopamine D2/D3 receptor availability; drug occupancy and dopamine competition.
Oxygen-15 water (^{15}O-water)		Cerebral blood flow.
Fluorine-18 florbetapir / flortaucipir (^{18}F-florbetapir / ^{18}F-flortaucipir)		Amyloid / tau respectively.

D2 receptor occupancy: effect of a drug



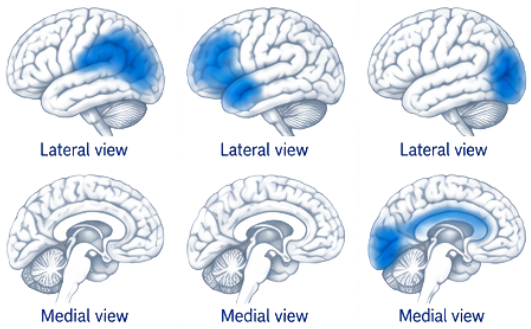
Occupancy estimate = 1 − (binding after drug ÷ binding before drug) under model assumptions.

Reduced binding may reflect drug occupancy or endogenous dopamine competition; not simply fewer receptors.

Uses and exam distinctions

Clinical: selected dementia evaluations, epilepsy localisation and other specialist indications.

Typical fluorodeoxyglucose hypometabolism patterns



Patterns support the clinical assessment; overlap occurs.

Psychiatry research: presynaptic dopamine function, receptor occupancy, drug mechanisms and metabolic networks.

Classical D2-antagonist studies: roughly 65–80% striatal occupancy often balances response and motor adverse effects. Not a universal target; clozapine and partial agonists differ.

Limitations and common errors

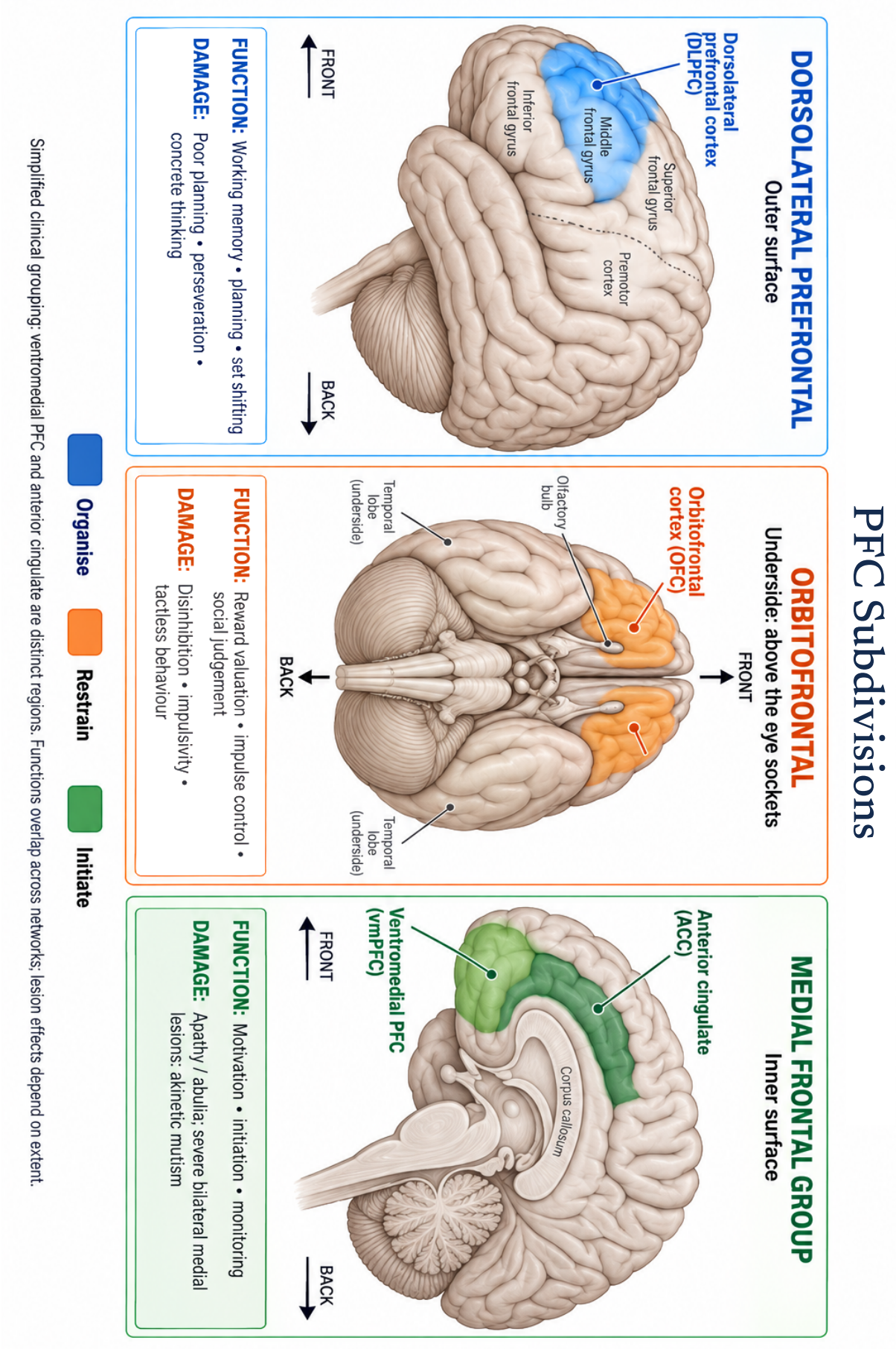
Radiation, expense, tracer availability, motion, blood glucose for fluorodeoxyglucose, medications and analytic modelling affect results. Limited anatomical detail: often combined with computed tomography or magnetic resonance imaging.

Metabolic reduction ≠ neuronal death. Amyloid positivity ≠ proof that amyloid explains every symptom. A dopamine synthesis study is not a receptor-density study. No PET pattern independently diagnoses schizophrenia or depression.

Illustrative maps — not patient scans

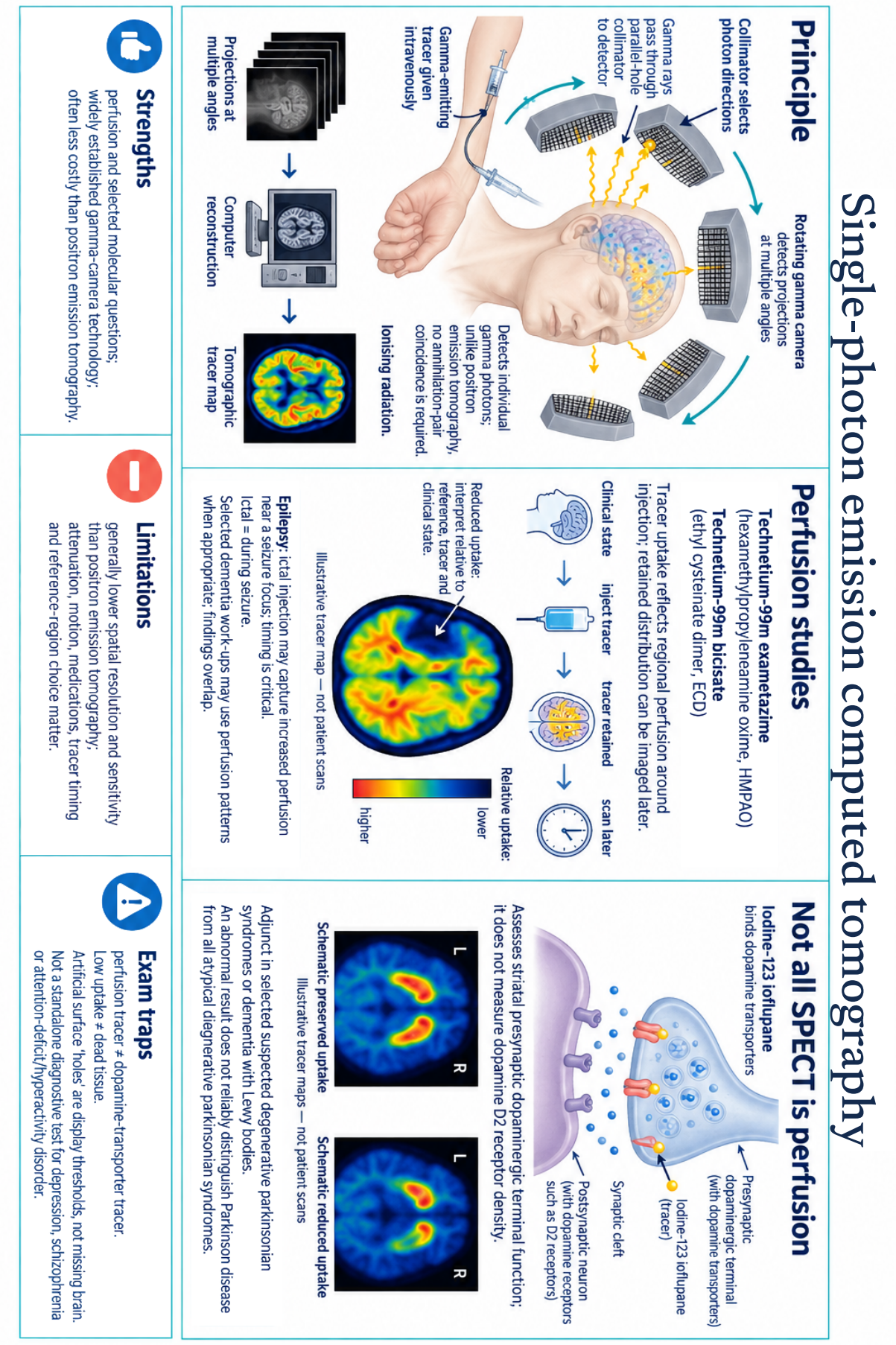
Positron emission tomography. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.



PFC Subdivisions. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

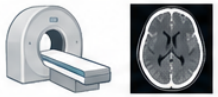
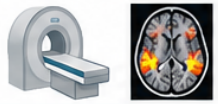
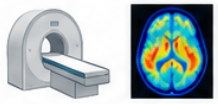
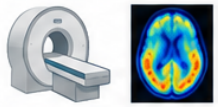
Credits and references: retained within the illustration.



Single-photon emission computed tomography. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Neuroimaging comparison

Modality	What it measures	Useful question	Main limits / common error
 **Computed tomography**	X-ray attenuation; tissue density contrast. Ionising radiation.	Acute blood, trauma, bone, hydrocephalus or urgent structural assessment.	Less soft-tissue detail; early ischaemia may be missed. Bright density is not always blood.
 **Structural magnetic resonance imaging**	Hydrogen-based tissue contrast: relaxation, diffusion and susceptibility. No ionising radiation.	Detailed anatomy: mass, demyelination, vascular injury, regional atrophy.	Motion, scan time, device compatibility. Atrophy or white-matter lesions alone do not establish a psychiatric diagnosis.
 **Functional magnetic resonance imaging**	Blood-oxygen-level-dependent haemodynamic signal. No ionising radiation.	Task contrast, statistical connectivity; selected presurgical mapping.	Indirect and delayed signal. Correlation is not causation; activation depends on the comparison.
 **Positron emission tomography**	Positron-emitting tracer distribution, detected using paired annihilation photons. Ionising radiation.	Tracer-dependent metabolism, perfusion, receptors, amyloid or tau.	Tracer access, cost, motion and modelling. Metabolism is not the same as receptor density.
 **Single-photon emission computed tomography**	Gamma-emitting tracer distribution using a collimated camera. Ionising radiation.	Perfusion; selected dopamine-transporter studies use a different tracer.	Generally lower resolution than positron emission tomography. Low uptake is not a hole in the brain.

Common clinical questions and example imaging strategies

Sudden confusion after a fall



Urgent structural assessment: often computed tomography

Progressive cognitive decline



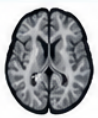
Clinical assessment + structural imaging; selected molecular/perfusion studies if they will resolve a question

Research on working memory



Task functional magnetic resonance imaging; interpret performance, baseline and confounds

Selected tracer and sequence readouts



Diffusion-weighted bright + low apparent diffusion coefficient → restricted diffusion



Fluorodeoxyglucose → glucose metabolism



Fluorodopa → dopamine synthesis capacity



Ioflupane → dopamine transporter availability



Raclopride → dopamine D2/D3 receptor availability

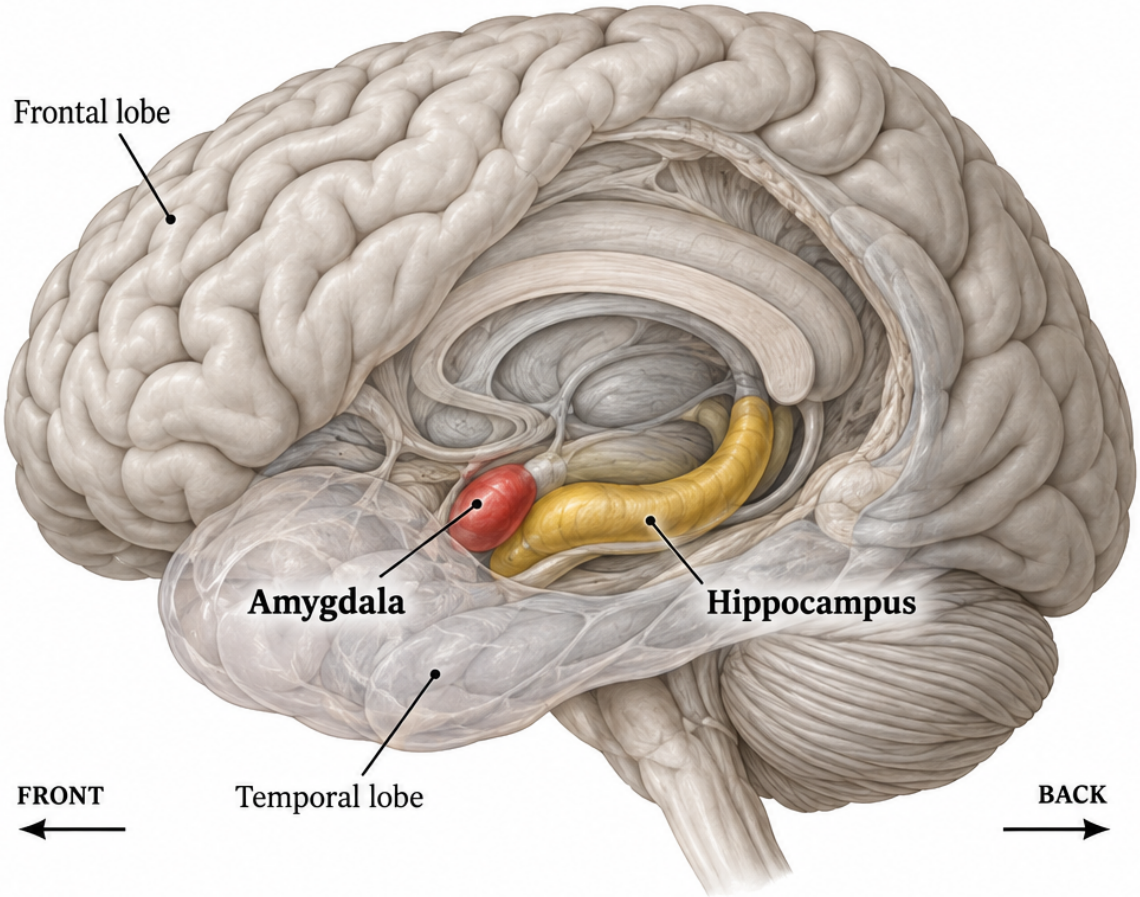
Choose the test for a specific question. Clinical findings and imaging must agree. Group-average differences do not independently diagnose schizophrenia, depression, bipolar disorder or attention-deficit/hyperactivity disorder.

Neuroimaging comparison. Read across the labelled contrasts to distinguish the related concepts. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

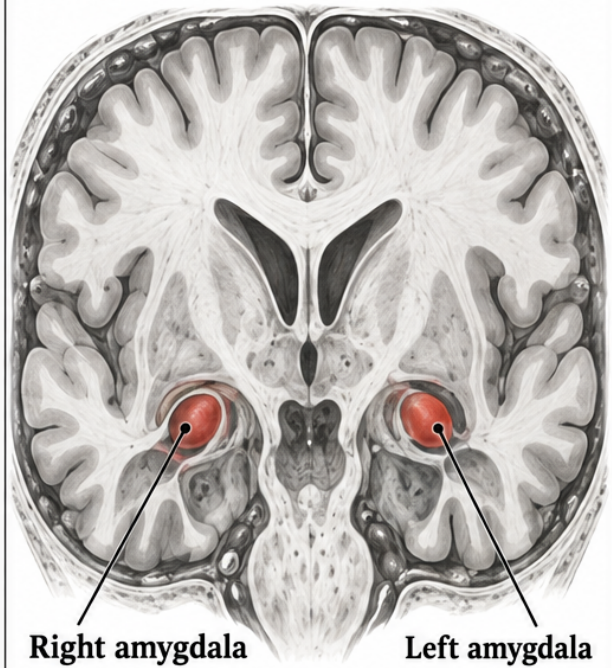
Amygdala Location

Left cerebral hemisphere (lateral view, medial temporal structures visible)
Outer temporal cortex made translucent to show amygdala and hippocampus



One on each side

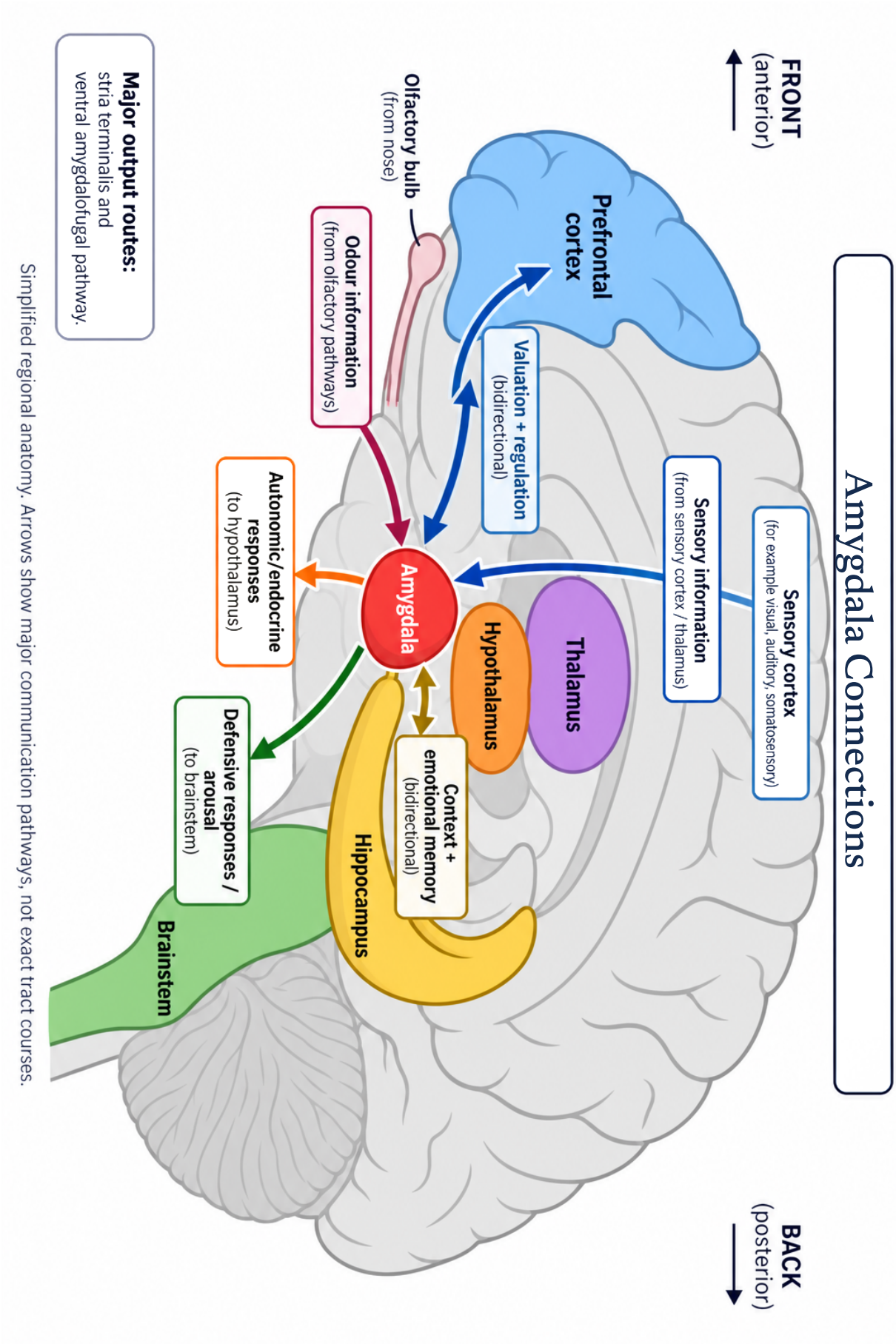
Coronal view: patient's right is image left



Amygdala = almond-shaped cluster of nuclei, deep in the medial temporal lobe, anterior to the hippocampal head.
Colours added for clarity; simplified anatomical illustration.

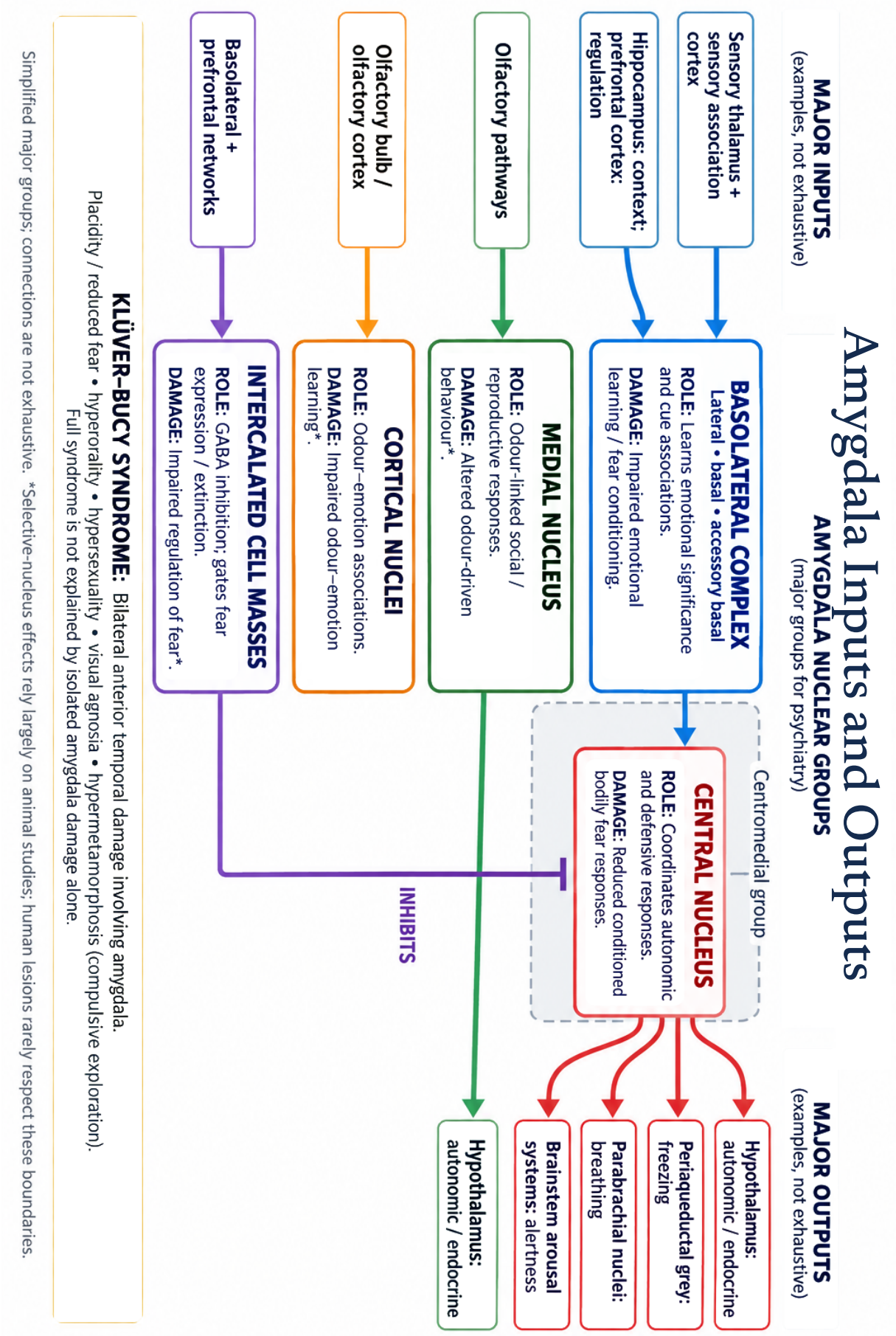
Amygdala Location. Use the labelled structures to connect anatomical location with function. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.



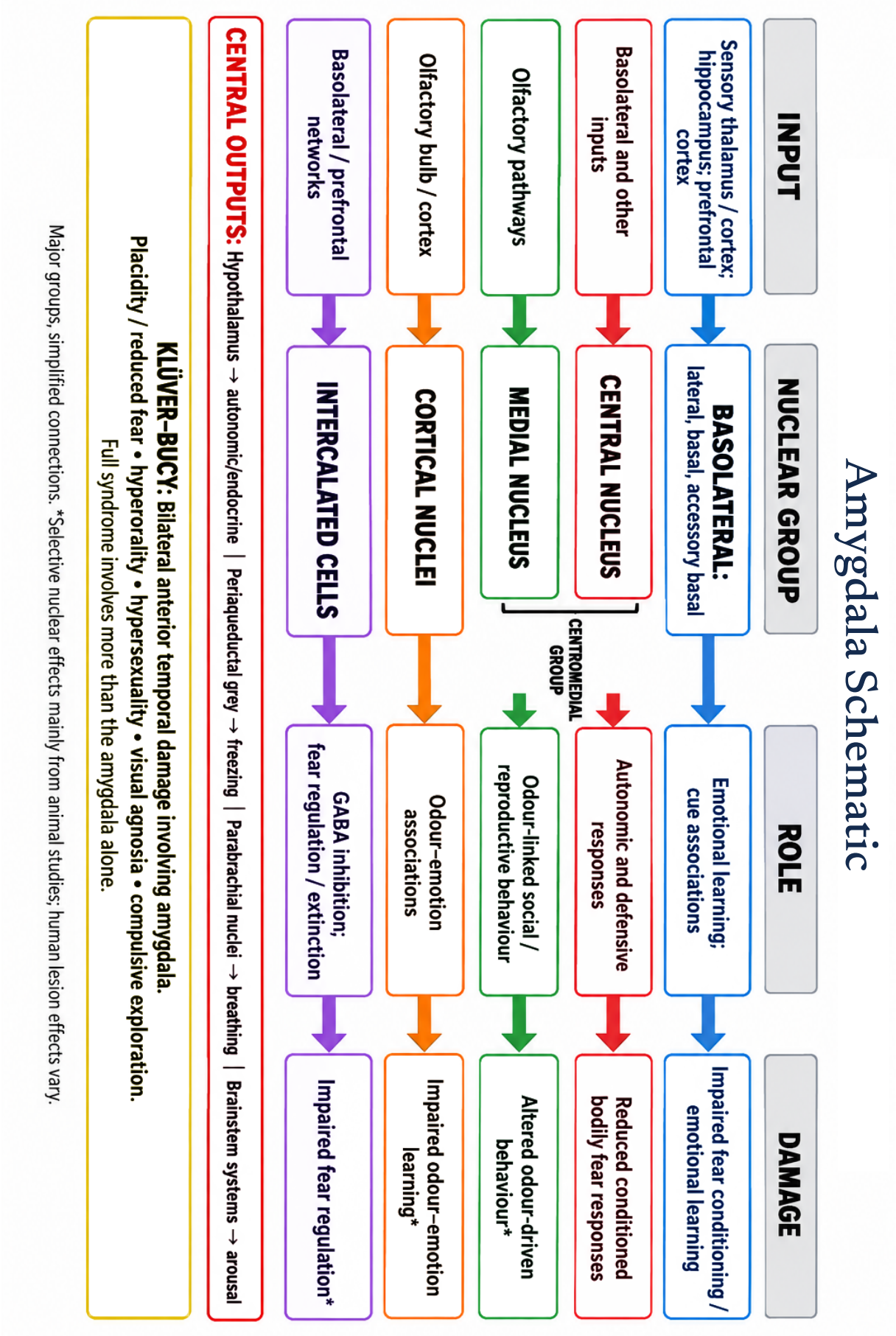
Amygdala Connections. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.



Amygdala Inputs and Outputs. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

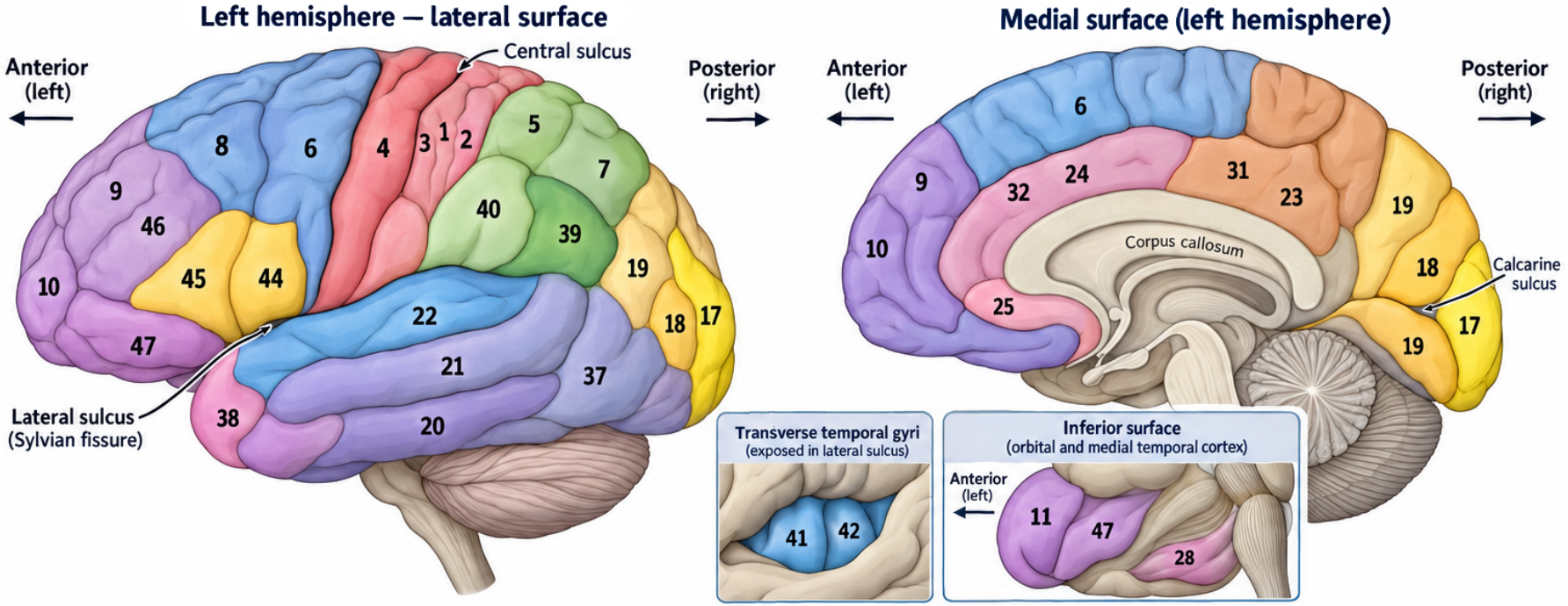
Credits and references: retained within the illustration.



Amygdala Schematic. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

Brodmann Areas



Movement and sensation

- **4** — Primary motor cortex: voluntary movement
- **6** — Premotor and supplementary motor cortices: movement planning
- **8** — Frontal eye fields: voluntary gaze
- **3, 1, 2** — Primary somatosensory cortex: body sensation
- **5, 7** — Superior parietal cortex: sensory integration and spatial processing

Language and perception

- **44, 45** — Broca region: speech and language production
- **22, posterior portion** — Language comprehension network
- **39** — Angular gyrus: semantic, reading and number processing
- **40** — Supramarginal gyrus: phonological and praxis networks
- **41** — Primary auditory cortex: sound processing
- **42** — Auditory association cortex: higher auditory processing
- **17** — Primary visual cortex: initial cortical visual processing
- **18, 19** — Visual association cortex: visual interpretation

Executive and limbic

- **9, 46** — Dorsolateral prefrontal cortex: working memory and cognitive control
- **10** — Frontopolar cortex: prospective planning
- **11, 47** — Orbitofrontal and inferior frontal regions: valuation and behavioural regulation
- **24, 32** — Anterior cingulate cortex: motivation, monitoring and control
- **25** — Subgenual cingulate cortex: mood-related networks
- **23, 31** — Posterior cingulate cortex: internally directed thought and memory

Temporal memory and recognition

- **20, 21** — Temporal association cortex: recognition and semantic processing
- **37** — Occipitotemporal cortex: visual recognition and reading networks
- **38** — Temporal pole: semantic and social-emotional integration
- **28** — Entorhinal cortex: interface with hippocampal memory networks

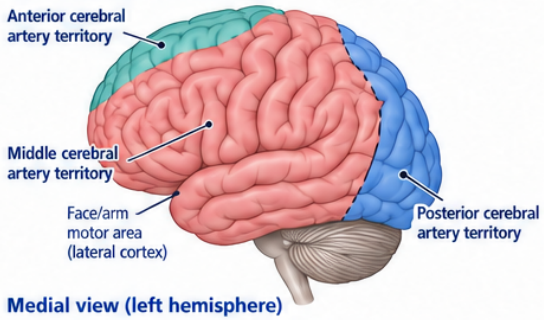
Cytoarchitectonic areas, not isolated function boxes. Boundaries and functional specialisation vary. Language dominance is usually left-sided. Selected high-yield areas, not a complete atlas.

Brodmann Areas. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

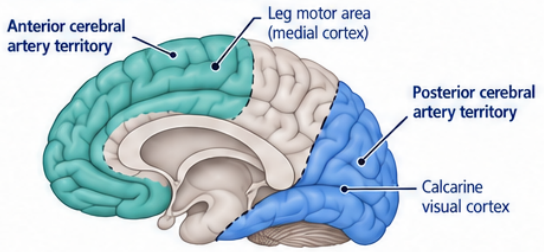
Credits and references: retained within the illustration.

Cerebral blood supply

Lateral view (left hemisphere)

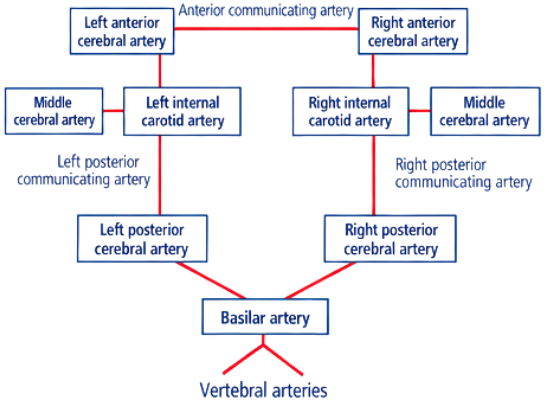


Medial view (left hemisphere)

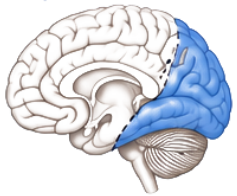


Schematic territories; individual anatomy varies

Circle of Willis — connection schematic

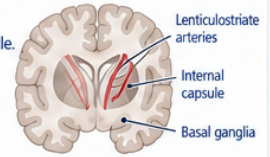


Middle cerebral arteries branch outside the ring.

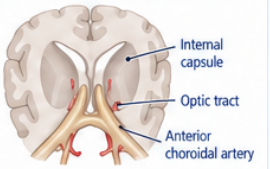
Territory	Main structure supplied (cortical)	Typical clinical deficit
Anterior cerebral artery (ACA)	**Medial frontal/ parietal cortex** 	• Opposite leg-predominant weakness • Abulia (loss of initiative) • Bilateral medial frontal injury may cause akinetic mutism.
Middle cerebral artery (MCA)	**Lateral cortex** 	• Opposite face/arm-predominant weakness. • Dominant hemisphere: aphasia (language impairment). • Non-dominant hemisphere: neglect (failure to attend to opposite space).
Posterior cerebral artery (PCA)	**Occipital and inferomedial temporal cortex** 	• Opposite homonymous hemianopia (same half of both visual fields); macular sparing may occur. • Hippocampal injury → amnesia. • Dominant occipital + splenial injury → alexia without agraphia (can write, cannot read).

Deep and posterior circulation

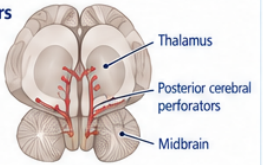
Lenticulostriate arteries
Basal ganglia and internal capsule.
Lacunar motor syndromes.



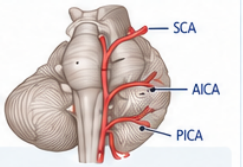
Anterior choroidal artery
Posterior internal capsule and optic pathway.
Weakness, sensory loss and visual-field loss may combine.



Posterior cerebral perforators
Thalamus and rostral midbrain.
Artery of Percheron variant supplies both paramedian thalami → reduced arousal, amnesia, vertical gaze disturbance.



Vertebrobasilar branches
Brainstem and cerebellum.
Superior cerebellar artery; anterior inferior cerebellar artery; posterior inferior cerebellar artery.



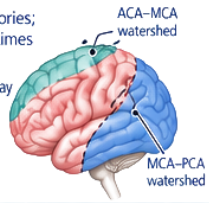
Superior: superior cerebellum
Anterior inferior: lateral pons / anterior-inferior cerebellum
Posterior inferior: inferior cerebellum; lateral medulla often vertebral or posterior inferior territory

Localise with the pattern

1. Right face/arm weakness + aphasia → left middle cerebral territory.
2. Left leg weakness + abulia → right medial frontal territory.
3. Pure opposite face-arm-leg weakness without cortical signs → internal capsule or another motor-pathway site.

Watershed and variability

- Watershed = border between territories; vulnerable to hypoperfusion, sometimes emboli.
- Bilateral anterior border-zone injury may cause proximal arm weakness.



Exam traps

- Arterial maps are variable.
- Cortical signs distinguish many large cortical strokes from lacunes.
- Sudden behavioural change can reflect stroke; localisation needs examination and imaging.

Not every case follows a textbook pattern.

Cerebral blood supply. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Functional neuroanatomy and neuroimaging.

Credits and references: retained within the illustration.

AREA 03 OF 12 · P1-A07

Personality theory and psychodynamics

10	10.5%	I-26	I-35
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 8

REVISION ORDER

The two defence-mechanism slots together, they are one answer at two widths, and the Anna Freud slot sits directly on top of them. The three neo-Freudian slots next, where Jung is most of all three. The topographical model and the concept of self after that. Transactional analysis is short and independent. The family-theories slot is the one here that needs reading of its own: it is a critical evaluation, not a description.

WHAT IS IN THIS AREA

Defence mechanisms in two slots, one asking for them and one asking for their classification and their classical use in treatment. The neo-Freudians in three, twice with Carl Jung named inside the question and once with Jung alone. Anna Freud, the topographical model with primary and secondary process thinking, the concept of self, transactional analysis, and the slot that asks whether the family theories of schizophrenia are still relevant.

I - 26

Ego defence mechanisms. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KUHS, NTRUHS, RGUHS - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	**Definition**	What the ego defends against, and with whose authority
2	**The organising frame**	Vaillant's four levels, named as levels
4	**The mechanisms**	Each at its own level, one line, one example
2	**The confusable pairs**	The four separations that mark a good answer

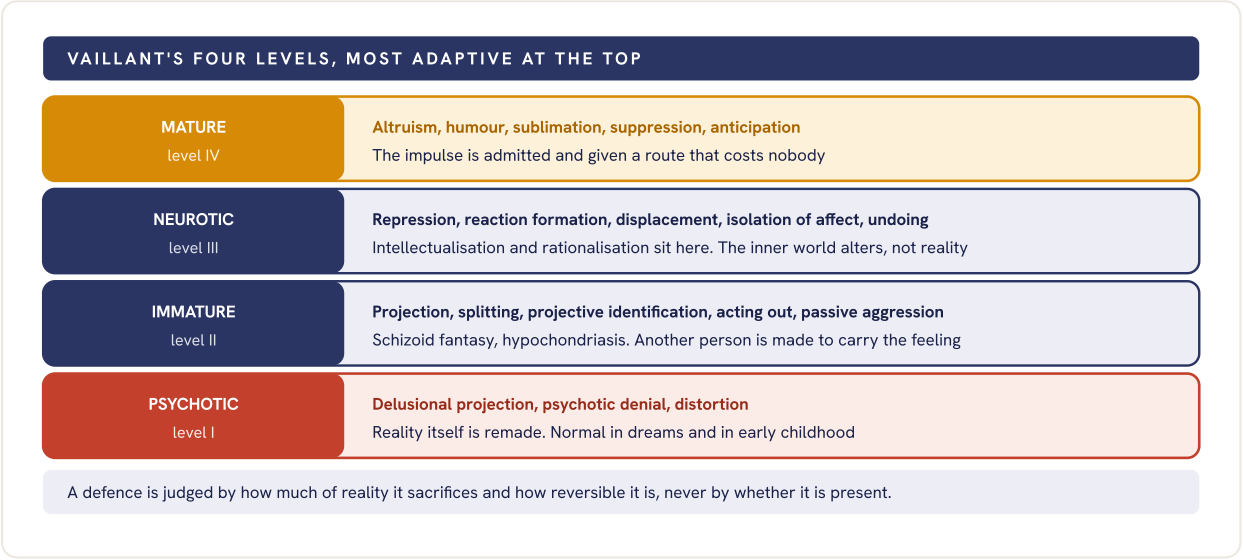


Fig 026.1 The four levels as rungs, the mature rung in amber because it is the one most often left out, the psychotic rung in red because only there does reality testing itself fail.

- **The definition, with its authority.** An unconscious operation of the ego that keeps an unacceptable impulse, affect or idea out of awareness and so holds anxiety down. Anna Freud gave the first systematic account in The Ego and the Mechanisms of Defence, 1936; her father had named repression in 1894 as the prototype from which the rest follow.
- **How they are recognised.** A defence is inferred from what is absent: the affect that fails to arrive with the content, or the theory offered in place of a feeling.
- **The four separations.** Projection puts one's own impulse into another; projective identification also makes the other feel and act it. Denial refuses a perception; repression removes an idea that was once conscious. Isolation of affect keeps the idea and strips the feeling; intellectualisation puts a theory where the feeling was. Suppression is conscious, repression is not.

WHAT EARNS THE MARKS

- **The definition attributed.** Anna Freud 1936, repression as Freud's prototype.
- **Four levels, named as levels,** with the mechanisms inside them rather than in one flat list. A list with no level is the standard mid-band answer.
- **The confusable pairs separated.** Projection against projective identification is the one examiners probe.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 34 Anna Freud SPRINT

Day 5, social, learning and personality psychology

I - 27

Relevance of family theories of Schizophrenia today. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

1	The claim they made	The family as cause, in its own era
5	The theories	Each with author, year and verdict
2	What survived	Expressed emotion, and why it lived
2	Relevance today	From blame to burden

THEORY	WHAT IT CLAIMED	WHOSE, AND WHEN	WHERE IT STANDS TODAY
Schizophrenogenic mother	A cold, rejecting mother produces the illness	Fromm-Reichmann, 1948	Abandoned; it harmed a generation of families
Double bind	Contradictory injunctions, no escape, comment forbidden	Bateson, Jackson, Haley, Weakland, 1956	Not causal; survives as a description of confusing talk
Marital schism and skew	Open chronic conflict, or one partner's dominance accepted	Lidz, 1957	Not causal; discord is a stressor like any other
Pseudomutuality and rubber fence	A surface of harmony that forbids real difference	Wynne, Ryckoff, Day and Hirsch, 1958	Not causal; the boundary idea survives clinically
Communication deviance	Fragmented, unclear attention in family speech	Singer and Wynne, from 1963	Predicts thought disorder in high-risk samples
Expressed emotion	Criticism, hostility and over-involvement predict relapse	Brown, Birley and Wing, 1972; Vaughn and Leff, 1976	**Stands; replicated, and the basis of family intervention**

The fourth column answers the word today. Five rows are history; the amber row is the one still in the guidelines.

- **Why expressed emotion survived when the rest did not.** It was measured rather than asserted, on the Camberwell Family Interview, and it made a prospective claim about relapse rather than a retrospective claim about cause. It predicts relapse; it does not predict onset. Vaughn and Leff set high contact at more than thirty-five hours a week face to face.
- **The error the stem is testing.** Treating expressed emotion as a modern version of the schizophrenogenic mother. One is a blaming aetiological claim with no evidence; the other is a measured predictor of course that says nothing about who caused the illness.
- **The Indian evidence, and its limit.** Expressed emotion runs lower in Indian families than in the British samples it was derived from, and the World Health Organization comparative studies reported a better course in centres in developing countries. Both are contested on method, and neither licenses the claim that the joint family is protective in itself.

WHAT EARNS THE MARKS

- **Each theory with its author and decade.** The stem is historical, so unattributed theories earn little.
- **A verdict against every row,** not a description of every row.
- **Expressed emotion separated from blame.** Predicting relapse is not claiming cause, and the shift from blame to burden is what the answer ends on.

SEE ALSO Slot I - 30 the Neo-Freudians and their use in psychotherapy **SPRINT**

Day 5, social, learning and personality psychology

I - 28

What is the concept of self? Discuss its perspective in psychiatric disorders. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the self is	The knower and the known
2	Formal features	Jaspers' four, named as four
4	Failure by disorder	Each against the condition that breaks it
2	The psychodynamic self	What Kohut and Winnicott add

THE DEFINITION

The self is the person as known to himself: at once the subject that experiences and the object experienced. William James, 1890, separated the I, the knower, from the Me, the known, and divided the Me into material, social and spiritual selves.

ASPECT OF THE SELF	HOW IT FAILS	WHERE THE FAILURE IS SEEN	WHOSE ACCOUNT
Awareness of activity	Thought and act are experienced as authored from outside	**Thought insertion and passivity in schizophrenia**	Jaspers
Awareness of unity	More than one self is experienced at one time	Dissociative identity presentations	Jaspers
Awareness of continuity	The thread back to the earlier self is cut	Dissociative amnesia and fugue	Jaspers
Awareness of boundaries	Self and world stop being demarcated	Ego boundary loss in schizophrenia; drug and ecstatic states	Jaspers
The bodily and narrative self	The self feels unreal, or will not hold one shape across roles	Depersonalisation; borderline identity diffusion	Schilder, Winnicott

- **The four accounts worth naming.** James, 1890, the I and the Me. Cooley, 1902, the looking-glass self, assembled from what we believe others see. Mead, 1934, the self arising in social interaction through the generalised other. Jaspers, 1913, four formal characteristics of self-awareness.
- **The psychodynamic self.** Kohut made the self, not the drive, the unit: built by selfobject responses of mirroring, idealisation and twinship, and fragmenting when they fail. Winnicott separated a true self, rooted in the body, from a false self built to comply.
- **Three words that are not synonyms.** Self-esteem is the value placed on the self, self-concept is its content, self-awareness is the formal fact of having one. Only the third breaks in schizophrenia.

WHAT EARNS THE MARKS

- **The I and the Me, attributed to James.** Two of the four concept marks live in that one sentence.
- **Jaspers' four characteristics named as four,** because the disorder half is hung on them.
- **A named disorder against each characteristic,** not a general claim that the self is disturbed in mental illness.

SEE ALSO Slot I - 31 Carl Jung and the Self as archetype Slot I - 33 the topographical theory of mind SPRINT Day 5, social, learning and personality psychology

I - 29

How do you classify the defense mechanisms? Describe their classical use in treatment of psychiatric disorders. [3+7]

3 + 7 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The classifications**	Three schemes, and what each one is for
4	**The treatment decision**	Support, interpret or convert, decided by level
2	**The sequence**	Resistance before content, defence before impulse
1	**The limit**	Where interpreting a defence does harm

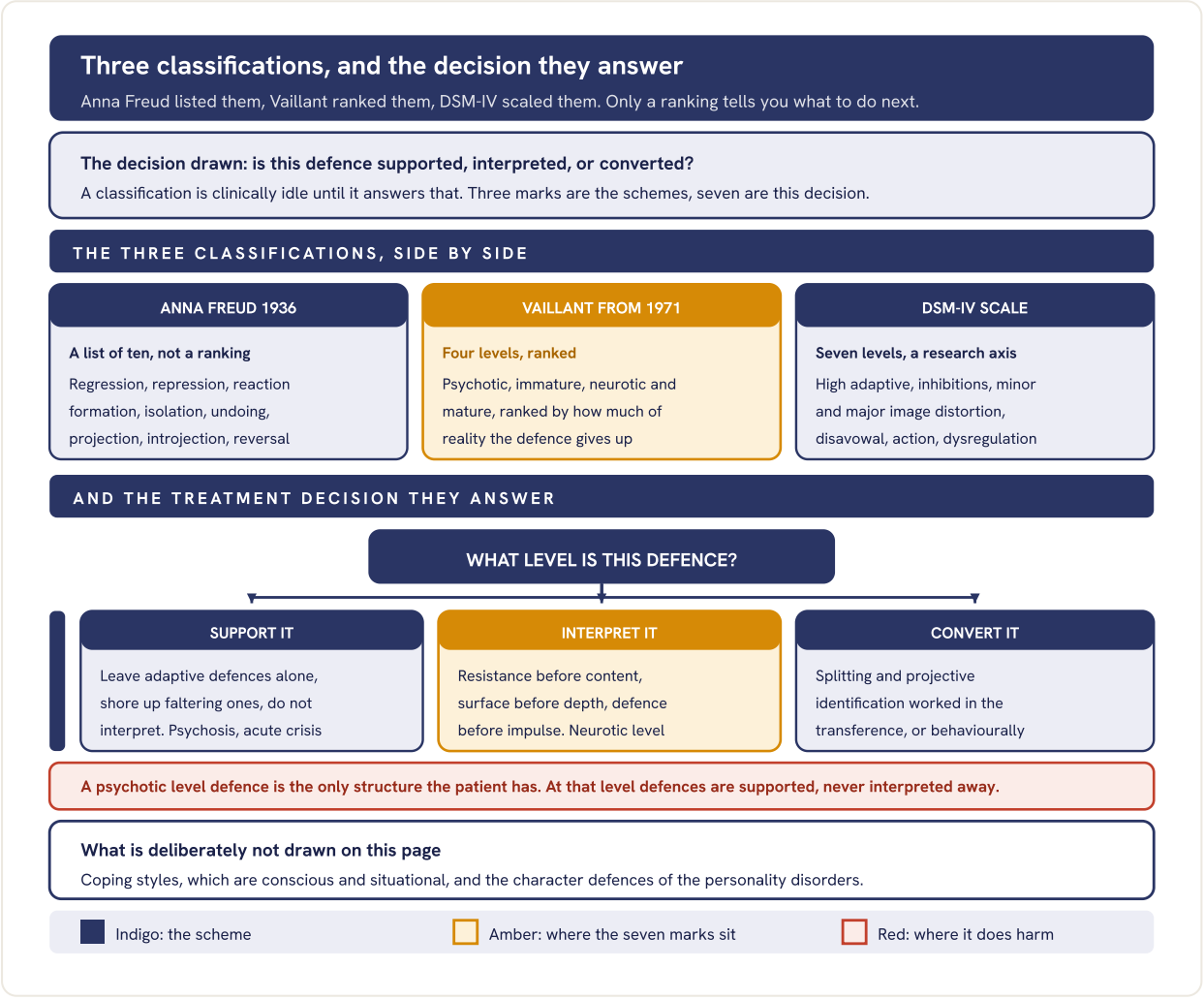


Fig 029.1 The three schemes across the top, the level question as the single node, and the three treatment lanes below it, with the interpretive lane in amber.

- **The sequence, which is the classical use.** Interpret resistance before content, surface before depth, and the defence before the impulse it hides. The aim is never to remove a defence but to replace it with a more adaptive one doing the same job.
- **What each scheme is for.** Anna Freud's list is what a clinical description draws on. Vaillant's levels are what a formulation and a prognosis draw on, because only a ranking supports a decision.

WHAT EARNS THE MARKS

- **Three schemes with their authors,** not one undifferentiated list of mechanisms.
- **The level used to make a decision.** The stem says classical use, so stopping at the classification throws away seven marks.
- **The interpretive sequence, in order.** Resistance, then content; defence, then impulse.
- **The red strip.** Supporting rather than interpreting a psychotic level defence is what shows clinical use.

SEE ALSO Slot I - 26 the ego defence mechanisms described Slot I - 34 Anna Freud **SPRINT**

Day 5, social, learning and personality psychology

I - 30

Describe various theories of Neo-Freudians. Discuss their use in psychotherapy. [5+5]

5 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	The axis	What makes a theorist Neo-Freudian rather than Freudian
4	The theories	Each with its break from Freud and its signature concept
4	Use in psychotherapy	The mechanism each one gave the consulting room
1	The honest limit	Where the label itself is loose

THEORIST	THE BREAK WITH FREUD	SIGNATURE CONCEPTS	WHAT IT GAVE PSYCHOTHERAPY
Alfred Adler	Social striving replaces the sexual drive	Inferiority and compensation, style of life, social interest	**Encouragement and goal-focused brief work; an ancestor of cognitive therapy**
Carl Jung	Libido is general psychic energy	Collective unconscious, archetypes, individuation, introversion and extraversion	Dream and symbol work; the tasks of the second half of life
Karen Horney	Basic anxiety, not libido, is the root of neurosis	Moving toward, against and away from people; the tyranny of the should	Work on the idealised self; the founding critique of Freud on women
Harry Stack Sullivan	Personality exists only between people	Self-system; good me, bad me, not me; parataxic distortion	**The therapist as participant observer; the parent of interpersonal psychotherapy**
Erich Fromm	Society and the burden of freedom form character	Escape from freedom, productive orientation	Existential and humanistic therapies
Erik Erikson	Development is psychosocial and runs the whole life span	Eight stages, epigenetic principle, identity, moratorium	Identity work in adolescence; life review in later life

The fourth column is the second half of the stem. Amber marks the two whose method survives under its own name in current practice.

- **The axis, stated before the list.** Every one of them kept the unconscious and the developmental frame and gave up the primacy of the sexual drive, putting culture, relationship or social striving in its place. Otto Rank and Wilhelm Reich are sometimes counted with them.
- **The five psychotherapy marks, which most answers lose.** Name a mechanism per theorist rather than a school: encouragement and goal setting from Adler, symbol work from Jung, the idealised self from Horney, the participant observer from Sullivan, identity and moratorium from Erikson.

WHAT EARNS THE MARKS

- **The axis in one line before any name.** A list of theorists with no stated criterion reads as recall.
- **Each theorist tied to what he or she abandoned,** not merely to a concept.
- **A therapeutic mechanism per row.** The stem splits five and five, so the last column carries half the paper.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 31 Carl Jung in full **SPRINT**

Day 5, social, learning and personality psychology

I - 31

Carl Jung. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The man and the break	Dates, and the one revision that made him Jungian
4	Structure of the psyche	Collective unconscious, archetypes, complexes
2	Typology and method	Attitudes, functions, word association, amplification
2	Individuation	The goal, and why it belongs to the second half of life

THE MAN, AND THE ONE REVISION

Carl Gustav Jung, 1875 to 1961, Swiss psychiatrist, Freud's designated successor until the break of 1913. He founded analytical psychology on a single revision: libido is general psychic energy rather than sexual energy, and the unconscious is collective as well as personal.

DOMAIN	THE CONCEPT	WHAT JUNG MEANT BY IT	WHERE IT SHOWS UP
Structure	Collective unconscious	An inherited layer common to all people, beneath the personal unconscious	The recurrence of the same myths and symbols
Structure	Archetypes	Inherited forms that organise experience: persona, shadow, anima and animus, the Self	**Persona and shadow are the two most often asked for**
Structure	Complexes	Feeling-toned clusters of ideas in the personal unconscious	Shown on his own Word Association Test
Typology	Attitudes and functions	Introversion and extraversion, crossed with thinking, feeling, sensation, intuition	The ancestor of later personality typologies
Development	Individuation	Lifelong integration of the unconscious into a whole centred on the Self	The task of the second half of life
Method	Amplification	A symbol elaborated through myth rather than reduced to biography	Dream work, and active imagination

- **The two confusions that cost marks.** The Self is not the ego: the ego is the centre of consciousness, the Self is the centre and the totality of the whole psyche and is the goal of individuation. The shadow is not the evil in a person but everything unlived and unacknowledged, undeveloped good included.
- **Where he still touches practice.** Symbol and dream work in analytical psychotherapy, midlife presentations read as a developmental task rather than a decline, and the word association method, which was an early objective demonstration that unconscious material can be measured.

WHAT EARNS THE MARKS

- **The revision named, not just the split.** Libido as general psychic energy is what makes him Jungian.
- **Four archetypes named,** and the Self distinguished from the ego.
- **Typology given as two attitudes crossed with four functions,** which is the form Jung used.
- **Individuation defined as a goal,** not as a synonym for growing up.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 28 the concept of self SPRINT

Day 5, social, learning and personality psychology

I - 32

Who are Neo-Freudians? How they are different from Post-Freudians? Write about contributions of Carl Jung. [2+3+5]

2 + 3 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Who they are**	The names, and the criterion that groups them
3 **The difference**	Broke away against built upon, domain by domain
4 **Jung's contributions**	Five, each named in his own terms
1 **The caveat**	Where the two labels overlap

DOMAIN	NEO-FREUDIANS	POST-FREUDIANS	WHAT SETTLES IT
Who	Adler, Jung, Horney, Sullivan, Fromm	Anna Freud, Hartmann, Klein, Winnicott, Fairbairn, Bion, Kohut	**Whether drive theory was abandoned or extended**
Relation to Freud	Broke away, usually in person as well as in theory	Stayed inside psychoanalysis and built on it	**Broke away against built upon**
What drives behaviour	Social striving, culture, inter-personal security	The drive kept, with the ego or the internal object added	The place still given to libido
Central unit	The person in a social field	The ego and its functions, or the internal object	Field against internal structure
Period	Roughly 1910 to 1940	Roughly 1930 onward	Suggestive, not decisive
Technique	Shorter, more active, more directive	The analytic frame kept, inter-pretation deepened	How far the method moved

The amber cells are the discriminators. The rest of the grid supports them, and saying which is which is itself a mark.

- **Jung's contributions, which carry five of the ten marks.** The collective unconscious and its archetypes, persona, shadow, anima and animus, and the Self. Complexes, demonstrated on the Word Association Test. A typology of two attitudes crossed with four functions. Individuation as the task of the second half of life. And amplification: the symbol elaborated rather than reduced.
- **The caveat that earns the last mark.** The label is loose. Jung and Adler are counted as Neo-Freudian because they broke away; Erikson is often counted with them although he never left. State the criterion you are sorting on before you sort the names.

WHAT EARNS THE MARKS

- **Both lists of names given,** because the stem asks who before it asks how.
- **The discriminator identified as the discriminator.** Abandoned against extended, not six differences of equal weight.
- **Five Jung contributions, separately named.** The last third of the stem is half the paper.

SEE ALSO Slot I - 31 Carl Jung in full Slot I - 30 the Neo-Freudian theories in psychotherapy SPRINT

Day 5, social, learning and personality psychology

I - 33

What is primary and secondary process of thinking as proposed by Freud? Write a brief note on topographical theory of mind. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Primary process**	The mode of the unconscious
2	**Secondary process**	The mode of the ego
4	**The topography**	Three systems, and what bars each door
2	**Its successor**	What the structural model added in 1923

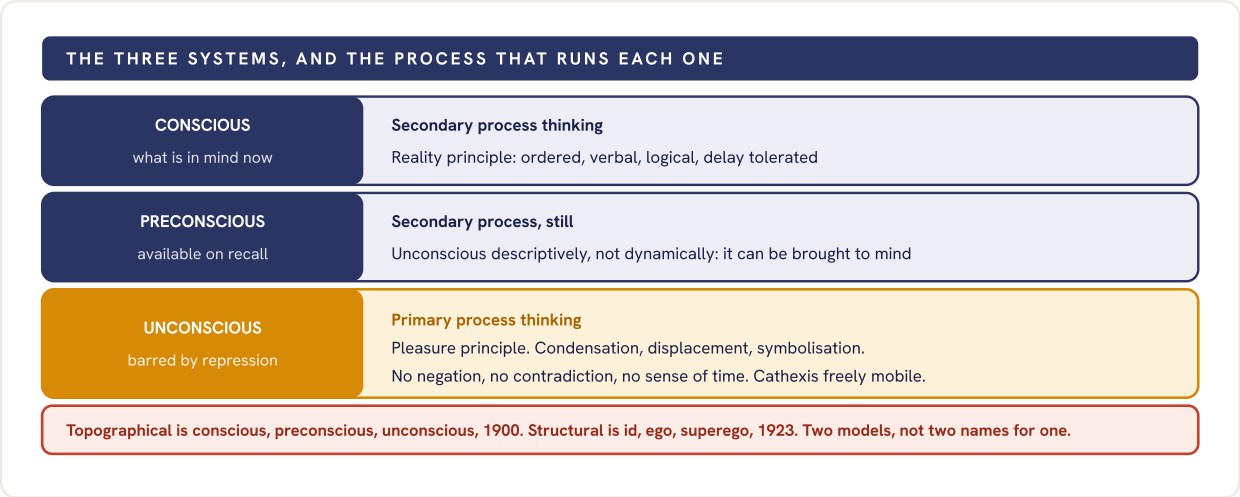


Fig 033.1 The three systems as strata with the correct mode of thinking hung on each. Amber marks the unconscious stratum, where the primary process marks are won; red marks the model most often confused with this one.

- **The two processes, as Freud defined them.** Primary process is the mode of the unconscious and of the infant: immediate discharge under the pleasure principle, cathexis freely mobile, working by condensation, displacement and symbolisation. Secondary process is the mode of the ego: cathexis bound, the reality principle, logic, language, and delay tolerated.
- **Where each is seen.** Primary process shows in dreams, in free association, in slips and jokes, and in the formal thought disorder of psychosis. Secondary process is ordinary waking thought, and its loss is what makes psychotic speech read as dreamlike.
- **Why it needed a successor.** The topographical model was supplemented in 1923, not abandoned. Freud found that much of the ego and all of the superego are themselves unconscious, which a model built on the single axis of awareness cannot hold.

WHAT EARNS THE MARKS

- **Both processes defined by their principle,** pleasure against reality, not by their qualities alone.
- **Condensation, displacement and symbolisation named.** They are what make primary process specific.
- **Preconscious separated from unconscious properly.** Descriptive against dynamic is the distinction examiners check.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 28 the concept of self **SPRINT** Day 5, social, learning and personality psychology

Sigmund Freud - Models of the Mind

Topographical model ▪ 1900

Classifies by accessibility to awareness

Conscious
in awareness now

"I feel embarrassed."



Supervisor: "Your presentation was disorganised."

Recall

Preconscious
not in awareness, readily recalled

"Yesterday's feedback comes to mind when asked."

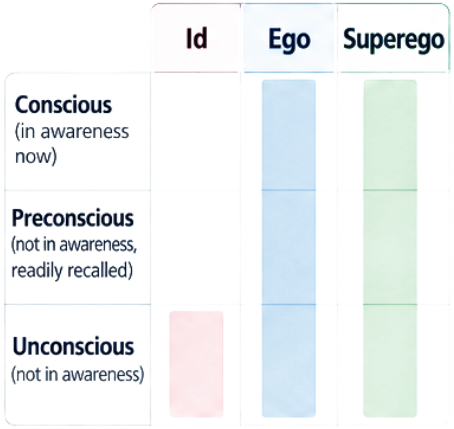
Repression: unconscious exclusion

Dynamic unconscious
wishes kept outside awareness by repression



Hypothetical Freudian interpretation:
"A retaliatory wish may be excluded from awareness."

Two models, one mind



Schematic—not anatomy or measured proportions.

Unconscious ≠ id · Conscious ≠ ego

Ego defences and aspects of the superego are unconscious.

Structural model ▪ 1923

Classifies by psychological function

Id
Instinctual drives; pleasure principle; immediate gratification.

Example: "Retaliatory impulse."

Superego
Internalised standards.

Conscience: prohibitions and guilt.

Ego ideal: standards to aspire to.

Example: "I should remain respectful."

Id demands

External reality
Demands and consequences in the real world.

Superego standards

External reality

Ego
Mediates id, superego and external reality; reality testing, planning, impulse control and defences.

Example: "Pause, consider consequences, discuss feedback calmly."

Primary process

Pleasure principle; immediate wish fulfilment; time and contradiction disregarded.

Condensation: several ideas combined.
Displacement: emphasis shifts to another idea.

Secondary process

Reality principle; logical thought, planning and delayed gratification.

Suppression is consciously setting something aside; **repression** is unconscious.

Clinical use and limits

A framework for exploring conflict, guilt and defences.

Historical constructs, not brain compartments. Nonconscious processing does not itself prove Freud's dynamic unconscious.

Sigmund Freud - Models of the Mind. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-33.

Credits and references: retained within the illustration.

Sigmund Freud - Psychosexual Development

Freud proposed that libido—psychic energy associated with pleasure and sexuality—shifts its main focus across development.

Historical theory · Approximate textbook ages · Fixation claims are **not established causes**

Oral
Birth - 1 year



Focus: mouth, sucking and biting.

Proposed conflict: feeding, weaning and dependence.

Proposed fixation: dependency; oral habits such as smoking or overeating.

Anal
1 - 3 years



Focus: bowel control.

Proposed conflict: toilet training; autonomy and control.

Proposed fixation: retentive—orderliness, parsimony (stinginess), obstinacy; expulsive—messiness and defiance.

Phallic
3 - 6 years



Focus: genitals.

Proposed conflict: Oedipal wishes and rivalry; identification and superego development.

Freud described castration anxiety and penis envy; these are contested historical claims.

Latency
6 years - puberty



Sexual impulses relatively less prominent or redirected.

School, friendships and skills.

Consolidation rather than a new dominant erogenous zone.

Genital
Puberty onward



Sexuality integrated with adult intimacy, reciprocity and work.

Freud's ideal of mature relating.

Do not use his historical heterosexual ideal to judge maturity or sexual orientation.

Oedipal conflict: Freud's proposed desire for one parent and rivalry with the other.
'Electra complex' was Carl Gustav Jung's term, not **Freud's**.

Fixation ≠ regression

Fixation
Persistent investment in an earlier developmental pattern.

Freud proposed that fixation points shape regression. →

Stress → Regression
Return to an earlier mode of coping.

Example: an adult seeks dependent reassurance under stress.
This illustrates regression; it does not prove oral fixation.

Exam interpretation and limits

- **Learn** stage → age → focus → proposed conflict.
- **Freud** linked early conflict to adult character; these specific links lack robust empirical support.
- Early experiences matter, but personality continues to change. Adult habits cannot identify a childhood fixation.
- **Clinical relevance:** historical language for developmental formulations, not a diagnostic test.

Sigmund Freud - Psychosexual Development. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-33.

Credits and references: retained within the illustration.

I - 34

Anna Freud. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The person	Dates, position, and the book
4	The contributions	Each stated in its own terms
2	The dispute with Klein	What each held, and what it split
2	What survives	Defence analysis, developmental profile

THE PERSON, AND THE BOOK

Anna Freud, 1895 to 1982, youngest child of Sigmund Freud, a lay analyst who founded child psychoanalysis in the British tradition and, with Hartmann, ego psychology. The book every answer must name is The Ego and the Mechanisms of Defence, 1936.

CONTRIBUTION	WHAT SHE ACTUALLY DID	THE CONCEPT TO NAME	WHERE IT STILL SHOWS
Defence, made systematic	Gathered scattered ideas into one account of how the ego wards off anxiety	**Ten mechanisms, and defence analysis as a technique**	Every later classification descends from it
Ego psychology	Moved analytic attention from the id to the ego and its adaptation	The ego as an agent with its own development	Formulation in terms of ego strength
Child analysis	Analysed children through play, education and a real working alliance	The child's ego needs support before it can bear interpretation	Child psychotherapy technique
Developmental lines	Traced development as continuous strands rather than stages alone	From dependency to emotional self-reliance, and others	Developmental profile in child assessment
Direct observation	Ran wartime residential nurseries for separated children in London	Observation admitted as psychoanalytic evidence	The empirical strand Bowlby then took up

- **Her definition of defence, which is the quotable line.** An unconscious operation by which the ego wards off an unacceptable instinctual demand, affect or idea, holding anxiety within bounds. She listed ten: regression, repression, reaction formation, isolation, undoing, projection, introjection, turning against the self, reversal and sublimation.
- **Anna Freud against Melanie Klein, one line each.** Anna Freud held the child's ego too immature for classical interpretation, so the work opens with an educational and supportive alliance. Klein held play to be the child's free association, and interpreted deeply and early. The disagreement split British psychoanalysis into three groups.

WHAT EARNS THE MARKS

- **The 1936 book named with its year.** On a named-person stem the title is a mark on its own.
- **Contributions kept separate,** not merged into one paragraph about child analysis, and ego psychology credited to her and Hartmann together.
- **The Klein dispute given as two positions,** with what it split, rather than as a personal quarrel.

SEE ALSO Slot I - 26 the ego defence mechanisms described

Slot I - 29 classifying the defences and using them in treatment SPRINT Day 5, social, learning and personality psychology

I - 35

Transactional analysis. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The frame	Berne, and the four levels of analysis
3	Ego states	Three, with divisions and voice
3	Transactions	Complementary, crossed, ulterior
2	Positions and games	Life positions, strokes, the script

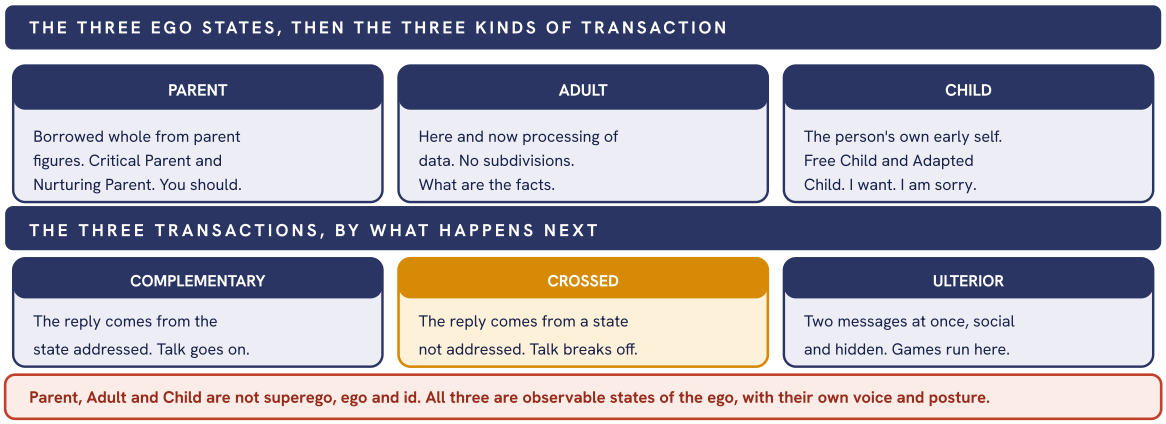


Fig 035.1 Ego states above, transactions below, sorted by what happens to the conversation. Amber marks the crossed transaction, the one this question asks to be illustrated.

LIFE POSITION	STANCE	WHERE IT IS SEEN
I am OK, you are OK	Get on with	Healthy and mutual; the therapy goal
I am OK, you are not OK	Get rid of	Blaming; paranoid and antisocial
I am not OK, you are OK	Get away from	Commonest; depressive, dependent
I am not OK, you are not OK	Get nowhere	Futility; chronic hopelessness

- **The frame, and its four levels.** Eric Berne, from 1958, built a contractual therapy in plain language. Structural analysis names the ego states, transactional analysis reads the exchanges, game analysis names repeated ulterior sequences with a predictable payoff, set out in Games People Play, 1964, and script analysis reads the life plan laid down in childhood.
- **Strokes and time.** A stroke is a unit of recognition, and people take negative strokes over none, which is what keeps a game running. Berne ranked six ways of structuring time: withdrawal, ritual, pastime, activity, game, intimacy.

WHAT EARNS THE MARKS

- **The four levels of analysis named as four.** Structural, transactional, game, script.
- **The three transactions with an example each,** crossed illustrated rather than merely defined.
- **Ego states kept apart from the structural model.** It is the discrimination this question is built on.

SEE ALSO Slot I - 30 the Neo-Freudian theories in psychotherapy Slot I - 33 the topographical theory of mind SPRINT Day 5, social, learning and personality psychology

Humanistic Theories of Personality

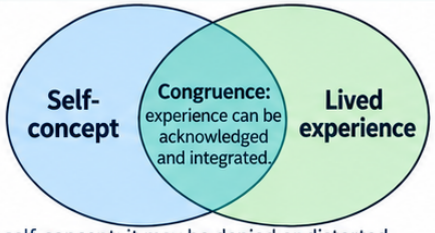
Humanistic psychology emphasises subjective experience, agency, relationships and growth.

Carl Ransom Rogers

How experience and the self fit together

Actualising tendency: the proposed organism-wide movement toward maintenance, growth and potential.

Self-concept: beliefs about who I am.
Ideal self: who I would like to be.



Incongruence: experience conflicts with the self-concept; it may be denied or distorted.

Conditional approval: 'You matter when you succeed.' → **Condition of worth:** 'I am worthwhile only if I excel.' → **Experience:** 'I am tired and need help.' → **Conflict with** 'I must always cope'.

Conditions of worth: learned requirements for feeling acceptable.
Therapeutic climate: empathy, unconditional positive regard and therapist congruence (genuineness).
Unconditional positive regard values the person; it does not approve every behaviour.
Fully functioning person: open to experience, self-trusting, flexible and creative.
Assessment link: Q-sort compares self-descriptions, including current and ideal self.

Abraham Harold Maslow

What needs organise motivation?



Growth: fulfilment can invite further development

Priorities overlap. Lower needs need not be completely satisfied before higher pursuits. The sequence is not a universal rigid rule.

Deficiency needs: motivated by lack.

Self-actualisation: an ongoing expression of potential, not perfection or status.
Maslow's proposed characteristics: autonomy, creativity, realistic perception and meaningful relationships.
Peak experience: a brief episode of intense fulfilment or unity, not a permanent stage.

Same student, two questions

Rogers: Can I acknowledge needing help without losing self-worth?



Maslow: Are sleep, safety, belonging and esteem needs being supported alongside learning?

Exam distinction and limits

Rogers centres congruence and the relational climate; Maslow centres needs and growth motivation.
Clinical relevance: collaborative goals and respect for experience. Broad growth constructs and a universal needs order are difficult to establish; empathy alone does not validate the whole theory.

Humanistic Theories of Personality. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Object Relations Theories

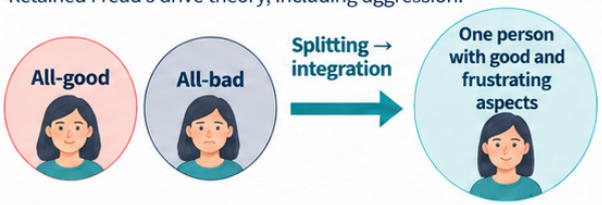
Object = a person, or an aspect of a person, toward whom feelings are directed. | **Internal object** = a mental representation of that person.

These theories describe how early relationships may organise inner experience.

Melanie Klein

Inner fantasy, anxiety and integration

Unconscious phantasy: imagined relationships outside awareness.
Retained Freud's drive theory, including aggression.



Part-object: one aspect; **whole-object:** the same person as a whole.

Paranoid-schizoid position: splitting and fear of persecution.

Depressive position: concern for the loved person, guilt, ambivalence and reparation—trying to make amends.

Positions recur; they are not psychiatric diagnoses.

Projective identification: in unconscious fantasy, parts of self are located in another person. Later accounts also examine feelings evoked in the other.

Example: after one disappointment, "My therapist is entirely uncaring"; integration allows "I am angry, and they have also helped me.'

William Ronald Dodds Fairbairn

Relationships are the primary motive

Libido is object-seeking: connection is the goal, not only pleasure or tension release.

Painful relationships may be internalised and preserved rather than relinquished.

Internal self-object pairs

Central ego	⟷	**ideal/accepted object**
Libidinal ego	⟷	**exciting object**
Antilibidinal ego	⟷	**rejecting object**

Libidinal = longing; **exciting** = promising but frustrating; **antilibidinal** = attacking or rejecting the longing self.

The antilibidinal ego is the 'internal saboteur'.

Example: someone keeps hoping for an unavailable partner's approval, then attacks themselves for needing it.

Donald Woods Winnicott

A facilitating environment supports a real self

Good-enough caregiving: responsive care that gradually allows manageable frustration; perfection is not required.

Holding environment: reliable physical and emotional support.

Primary maternal preoccupation: heightened early attunement to the infant.

Transitional object: a blanket or teddy that bridges felt closeness and separateness; a first 'not-me' possession.



Potential space: the shared area of play, creativity and experience.

True self: spontaneous, felt aliveness.

False self: protective compliance; ordinary social adaptation is not automatically pathological.

Example: a child uses a familiar teddy during a caregiver's absence; an adult may hide preferences to keep approval.

Klein: integrate split experiences • **Fairbairn:** understand internalised relational ties • **Winnicott:** support spontaneity within reliable care.

Clinical use: exploring recurring relationships and responses to the therapist.

These are interpretive models, not proven infant mechanisms; do not infer parenting blame or equate them with attachment classifications.

Object Relations Theories. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Otto Rank

Separation, will and becoming an individual

Otto Rank · 1884–1939

Early collaborator of Sigmund Freud; later developed a distinct relationship-centred approach.

Birth-trauma theory ▪ 1924

In *The Trauma of Birth*, Rank proposed birth as the first major separation and a prototype of anxiety.

He interpreted later separations as reactivating this primal tension.

Shift of emphasis: from Freud's Oedipal conflict to earlier mother–infant separation.

Historical hypothesis—not an established cause of anxiety.

Not equivalent to obstetric injury, neonatal trauma or a recalled birth memory.

The tension of individuality

CLOSENESS / UNION

Connection
Belonging
Being with others



SEPARATENESS / AUTONOMY

Independence
Self-direction
Standing on one's own

Death fear: losing the distinct self through absorption, conformity or stagnation.

Life fear: isolation and responsibility associated with living as a separate individual.

These are Rank's theoretical terms, not diagnostic categories.

Will: the capacity to choose, organise action and affirm one's own direction.

Creative will: shaping a personally meaningful life while remaining able to relate.

Individuality need not require isolation; connection need not erase the self.

Will therapy

- Present therapeutic relationship, active participation and self-direction.
- Work with conflicting wishes for closeness and independence.
- Ending therapy becomes an opportunity to experience separation while retaining agency.
- Will is not mere stubbornness, compliance with the therapist or 'trying harder'.
- English publication of *Will Therapy*: 1936.

Fictional example: leaving home for training



Rankian focus: acknowledge both fears and make an owned choice; do not infer a hidden birth memory.

Legacy and limitations

- Influenced **Carl Ransom Rogers**, humanistic and existential thinking, and functional social work.
- Separation and agency remain useful themes.
- This does not validate birth trauma as a universal explanation; historical claims rely heavily on interpretation.

Otto Rank. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Trait and Type Approaches to Personality

Type: a discrete category. **Trait:** a continuum. Traits describe tendencies, not destinies.

ALLPORT · the individual

Cardinal: rare, life-dominating disposition.
Central: broad characteristics.
Secondary: specific preferences.

Functional autonomy: a motive can outgrow its origin — studying for praise becomes studying from curiosity.



CATTELL · factor analysis

Surface traits: visible clusters.
Source traits: underlying factors.
16PF: 16 primary personality factors.
Data: L = life records; Q = self-report; T = objective tests.

Factor solutions depend on the measures used.

EYSENCK · PEN

Psychoticism · **E**xtraversion · **N**euroticism.
Responses → habits → traits → broad dimensions.
EPQ: questionnaire assessment.
Biological explanations have mixed support.
Psychoticism is not a diagnosis of psychosis.



BIG FIVE · OCEAN

Openness · curious, open to experience.
Conscientiousness · organised, dependable.
Extraversion · outgoing, energetic.
Agreeableness · cooperative, trusting.
Neuroticism · prone to negative emotion.
NEO-PI-R: five domains, six facets each.

Exam guardrail: Allport's levels, PEN, 16PF and OCEAN are different models. Interpret traits in context; they are not diagnoses.

Trait and Type Approaches to Personality. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Adolf Meyer's Psychobiology

Core idea: understand the whole person through the course of their life.

1 · THE PERSON OVER TIME



Biology, habits and the social world develop together.
Adaptation: responding to demands with available capacities and resources.
Meyer described many disorders as maladaptive reaction patterns.

2 · HISTORY → CHART → FORMULATION



Life history: the detailed narrative.
Life chart: the chronological visual summary.
Formulation: vulnerabilities, triggers, maintaining factors and strengths.

Chart four tracks: life events · health and symptoms · habits and coping · help and resources.
Temporal association is a clue, not proof of causation.

3 · PRACTICAL LEGACY

Habit training; occupation, relationships, community care and prevention. "Reaction types" influenced DSM-I terminology; "ergasiology" is now obsolete.

Exam guardrail: Meyer anticipated later biopsychosocial formulation; Engel articulated the later model. Life history complements diagnosis and biological assessment.

Adolf Meyer's Psychobiology. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Existential School of Psychology

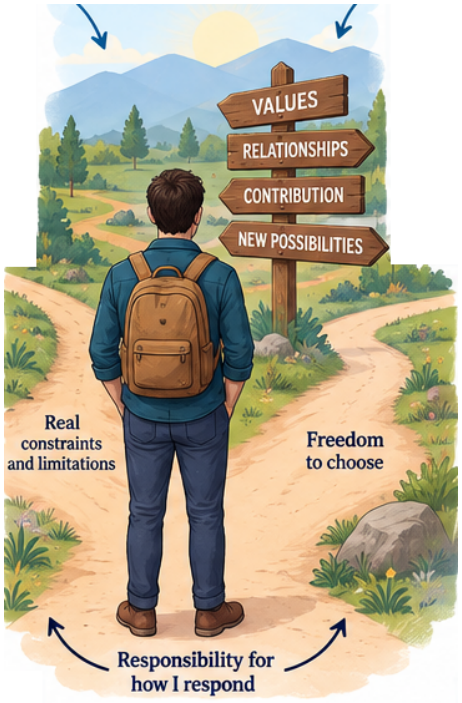
PHILOSOPHICAL ROOTS

Kierkegaard: choice and anxiety.
Nietzsche: values and meaning.
Heidegger: being-in-the-world.
Sartre: freedom and responsibility.

CLINICAL CONTRIBUTORS

Binswanger / Boss: lived experience and being-in-the-world.
May: anxiety and courage.
Frankl: logotherapy; will to meaning.
Yalom: four ultimate concerns.

Humanistic: growth and actualisation.
Existential: finitude, choice and meaning.
The approaches overlap.



Freedom has real constraints. Responsibility does not mean blaming people for adversity.

YALOM · FOUR CONCERNS

Death.
Freedom and responsibility.
Existential isolation.
Meaninglessness.

THERAPEUTIC FOCUS

Explore the person's experience.
Clarify values and meaningful choices.
Examine avoidance.
Use an authentic therapeutic relationship.

Frankl: dereflection and paradoxical intention require careful, individual use.

Meaning: contribution, relationships and the attitude taken toward unavoidable suffering.

Exam guardrail: existential anxiety can be human experience; persistent or disabling distress needs assessment. This framework is not a universal causal explanation.

Existential School of Psychology. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

Positive Psychology

Empirical study of wellbeing, strengths and flourishing

1. Definition and lineage

Positive psychology is the empirical study of wellbeing, strengths and flourishing.

- Martin Seligman championed the modern positive psychology programme from 1998.
- Humanistic antecedents include Abraham Maslow (self-actualisation) and Carl Rogers (unconditional positive regard).
- Positive psychology is an empirical programme, informed by but not identical to humanistic psychology.

2. PERMA model (Martin Seligman)

PERMA describes components of wellbeing, not diagnostic criteria.

	Element	Description
P	Positive emotion	Enjoyment, gratitude, hope and other pleasant emotions.
E	Engagement	Deep absorption in activities and use of personal strengths.
R	Relationships	Supportive, positive connection with others.
M	Meaning	Serving something larger than oneself.
A	Accomplishment	Progress, mastery and achievement in valued areas.

3. Character strengths (VIA framework)

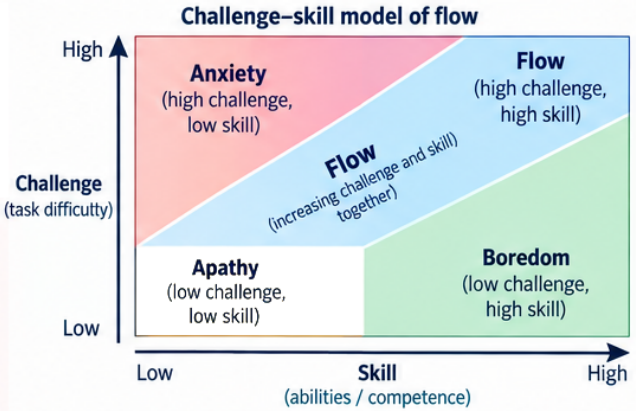
Peterson and Seligman's VIA framework identifies 24 character strengths grouped under 6 virtues. Examples are illustrative, not complete lists.

Virtue (6 total)	Example strengths
Wisdom and knowledge	Curiosity, creativity
Courage	Bravery, perseverance
Humanity	Love, kindness
Justice	Fairness, teamwork
Temperance	Prudence, self-regulation
Transcendence	Gratitude, hope

4. Flow (Mihaly Csikszentmihalyi)

Flow is an optimal state of intense absorption in an activity, with: action-awareness merging, reduced self-consciousness, altered time experience, and intrinsic reward.

- More likely when there are clear goals and immediate feedback.
- Flow typically occurs when challenge and skill are both high and increase together. It is not simply a result of low challenge and low skill.



5. Applications and limits

Potential applications

- Strengths-based goals (e.g. using personal strengths in daily life).
- Gratitude practices.
- Meaningful activity and purpose.
- Relationships and engagement may support wellbeing.
- Benefits vary by person and context.

Important limits

- Complements, but does not replace, standard assessment and indicated treatment.
- Does not require constant happiness, deny the reality of suffering, or replace professional care for mental illness.
- Wellbeing and psychiatric symptoms are related but distinct constructs.

Key recall strip | **PERMA** = five wellbeing elements | **VIA** = 24 strengths / 6 virtues | **Flow** = absorbed engagement

Positive Psychology. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Personality theory and psychodynamics.

Credits and references: retained within the illustration.

AREA 04 OF 12 · P1-A05

Learning, memory and general psychology

9	9.9%	I-36	I-44
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 4 · HIGH 2 · RECURRENT 1 · SINGLE 2

REVISION ORDER

The learning block first: four slots and largely one answer, with the two conditioning slots almost interchangeable. The two emotion slots next, likewise, and the James-Lange slot carries the evaluation the other does not ask for. Intelligence after that, theories first and then the Indian test. Emotional intelligence and social learning theory are short and stand on their own.

WHAT IS IN THIS AREA

Learning in four slots: the definition with the models and their clinical importance, classical against operant conditioning with an example of each, Pavlovian conditioning inside behaviour therapy, and social learning theory. Emotion in two, the basic emotions with the theories, and the theories again with James-Lange critically evaluated. Intelligence in three, the theories with their application, emotional intelligence against IQ, and the Indian standardised test. This is the anchor-heaviest area in the book: four of its nine slots have come back across boards and years.

I - 36

Theories of intelligence and their application in Psychiatry. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KNRUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	What the construct is, and the general factor debate
4	The theories	Each theorist, his claim, the structure proposed
2	How it is measured	Ratio and deviation quotient, and what a test yields
2	Use in psychiatry	Where the figure changes a clinical decision

THE DEFINITION

Wechsler defined intelligence as the global capacity to act purposefully, to think rationally and to deal effectively with the environment. Binet called it judgement applied to circumstances. Both are functional definitions: intelligence is inferred from performance, never observed directly.

THEORIST	THE CLAIM	STRUCTURE PROPOSED	WHAT IT IS USED FOR
Spearman, 1904	One general ability runs through every task	Two factors, general and specific	The rationale for a single quotient
Thurstone, 1938	There is no single ability, only several	Seven primary mental abilities	Profile testing, subtest scatter
Cattell and Horn	Ability splits by how it ages	Fluid and crystallised intelligence	Separating decline from a low baseline
Guilford, 1967	Ability is a three way classification	Operations by contents by products	Divergent thinking and creativity
Gardner, 1983	Intelligences are plural and independent	Seven, later eight, intelligences	Strengths based educational planning
Sternberg, 1985	Intelligence is what succeeds in context	Analytic, creative and practical	Vocational and rehabilitation planning

- **Measurement.** Stern's ratio quotient, mental age over chronological age times a hundred, fails in adults because mental age plateaus. Wechsler replaced it with the deviation quotient, mean a hundred, standard deviation fifteen. Vernon's hierarchical model is the shape modern batteries take.
- **Application, and its limit.** Intellectual disability needs a low quotient **and** impaired adaptive functioning; the older bands read mild fifty to sixty nine, moderate thirty five to forty nine, severe twenty to thirty four, profound below twenty. Fluid ability falls in dementia while crystallised ability holds, which is what makes that pair useful.

WHAT EARNS THE MARKS

- **A definition with a name attached,** written out before the theories start.
- **Every row of the grid carrying a theorist and a date.** Theories listed without authors read as recall.
- **The amber row, fluid against crystallised,** because it is the one that does clinical work at the bedside.
- **Adaptive functioning named alongside the quotient.** A number alone does not make the diagnosis.

SEE ALSO Slot I - 43 the Indian standardised intelligence test Slot I - 38 emotional intelligence and its relation to quotient

SPRINT Day 4, general and developmental psychology

I - 37

Social learning theory. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The claim	Learning by watching, without doing and without reward
3 The four processes	Drawn in order, with the point where learning is complete
3 The constructs	Reciprocal determinism, self efficacy, vicarious reinforcement
2 Clinical use	The treatments it produced, and the harms it explains

THE CLAIM

Behaviour is acquired by observing a model, held in symbolic form, and performed later when the incentive is right. Direct reinforcement of the learner is not required, and that is what separates this from operant theory.

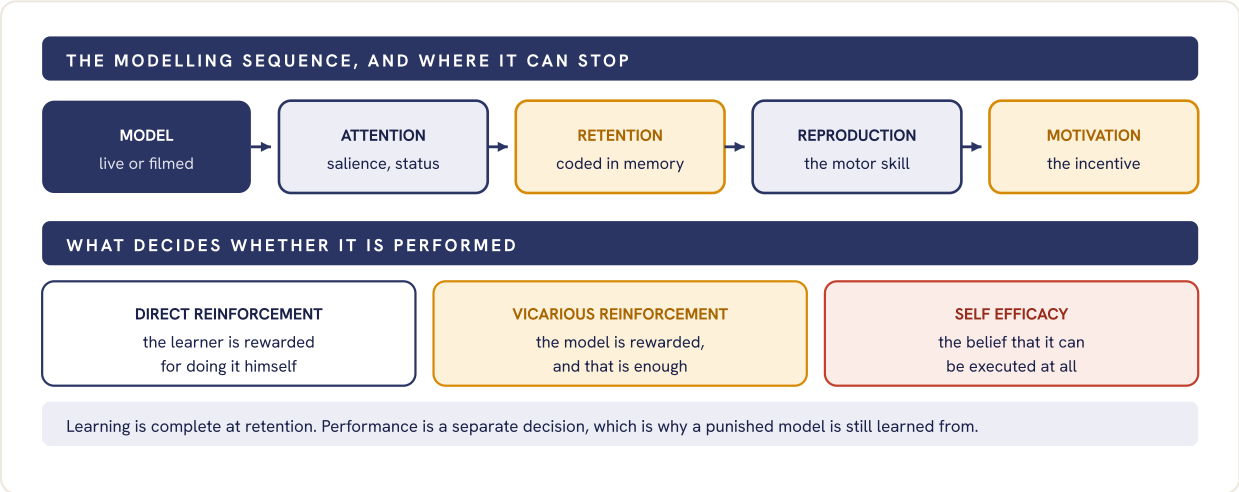


Fig 037.1 The four mediational processes in order, with the two amber stages that carry the theory's whole argument, and the three influences that decide whether what was learned is ever shown.

- **The evidence and the constructs.** Children who watched an adult attack an inflatable doll reproduced the aggression; those who saw the model punished performed less, yet showed on incentive that they had learned as much. Reciprocal determinism holds that person, behaviour and environment each shape the other two.
- **Clinical use, both directions.** Participant modelling in phobia work, behavioural rehearsal in social skills training, video modelling with children. The same mechanism explains learned illness behaviour in a family and imitative self harm after media coverage.

WHAT EARNS THE MARKS

- **Learning separated from performance,** which is the sentence the whole theory turns on.
- **All four mediational processes named in order,** exactly as the figure draws them.
- **Self efficacy named and defined,** built from mastery, vicarious experience, persuasion and bodily state.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 41 classical and operant conditioning **SPRINT** Day 5, social, learning and personality psychology

I - 38

What is emotional intelligence? Describe components of emotional intelligence. Mention the relationship between emotional intelligence and I.Q. [2+4+4]

2 + 4 + 4 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition	What it is, and who named it in which year
4	The components	Three rival models, each with its own component list
3	Against the quotient	What each predicts, and what the overlap really is
1	Clinical bearing	The disorders that sit at the low end

THE DEFINITION

Salovey and Mayer, who named the construct in 1990, defined it as the ability to monitor one's own and other people's feelings, to discriminate among them, and to use that information to guide thinking and action. Goleman's 1995 book widened it well beyond that.

MODEL	COMPONENTS	HOW IT IS MEASURED	STANDING
Ability, Salovey and Mayer	Perceiving emotion, using emotion to aid thought, understanding emotion, managing emotion	Performance test with scored right answers	**The only model that behaves like an intelligence**
Mixed, Goleman	Self awareness, self regulation, motivation, empathy, social skill	Self report and rating by others	Popular, evidence weaker than the claims
Social and emotional, Bar-On	Intrapersonal, interpersonal, adaptability, stress management, general mood	Self report inventory	Broad, close to wellbeing
Trait, Petrides	Emotional self perceptions sited within personality	Self report questionnaire	Overlaps heavily with personality

- **The relationship with the quotient.** Positively but only modestly related, and not substitutes. The quotient remains much the stronger predictor of academic attainment and of cognitively demanding work. Ability emotional intelligence adds a small increment for interpersonal outcomes once quotient and personality are accounted for; trait and mixed measures add least, being largely personality under another name.
- **Clinical bearing, and the trap.** The low end is recognisable in clinic: alexithymia, the empathy profile of autism, the intact cognitive with impaired affective empathy of antisocial personality, the emotion dysregulation of borderline personality. The trap is not naming which model you mean, because a performance test and a self report questionnaire do not measure the same thing.

WHAT EARNS THE MARKS

- **The 1990 definition with both authors,** kept separate from Goleman's later version.
- **Components given model by model,** because the models disagree about what they are.
- **The amber row.** Only the ability model is measured the way an intelligence is measured.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 39 basic emotions and theories of emotion **SPRINT** Day 4, general and developmental psychology

I - 39

What are the basic emotions? Discuss briefly the theories of emotion. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Basic emotions	The criteria first, then the named sets
5	The theories	Each one as a sequence, with its support and its flaw
2	The appraisal turn	What cognition added, and what it displaced
1	Why it matters	One line linking the theory to a treatment

THEORY	THE SEQUENCE PROPOSED	ITS STRONGEST SUPPORT	WHERE IT FAILS
James and Lange, 1884	Bodily change first, emotion is its reading	Facial feedback; blunting after cord injury	Visceral change too slow, too alike
Cannon and Bard, 1927	Thalamus fires to cortex and body at once	Emotion survives loss of feedback	The thalamus is too simple a relay
Schachter and Singer, 1962	Arousal, then a label taken from context	Adrenaline felt as anger or elation by context	Replication has been uneven
Lazarus, 1966	Appraisal first, and it can be automatic	One event, different appraisals, different emotions	Appraisal is hard to measure apart
Papez and MacLean	A closed circuit carries the experience	The connections named are real	Superseded as an account of emotion

- **What makes an emotion basic.** Universal recognition across cultures, a distinct facial expression, quick onset and brief course, a distinct physiological signature, automatic appraisal, a counterpart in other primates.
- **The three sets worth naming.** Ekman gives six, from universally recognised facial expressions: happiness, sadness, fear, anger, disgust, surprise. Plutchik gives eight as four opposed pairs on a wheel, joy against sadness, trust against disgust, fear against anger, surprise against anticipation. Panksepp names seven systems from the circuits themselves: seeking, rage, fear, lust, care, panic and grief, play.
- **The clinical pay-off.** Schachter and Singer is the panic model in embryo. Benign arousal receives a catastrophic label, and it is the label, not the arousal, that interoceptive exposure changes.

PITFALL

These theories are not a league table with a winner. Each was framed against the one before it, and the position now held is that bodily change, appraisal and context all contribute. Writing one as simply correct throws away the discussion marks.

WHAT EARNS THE MARKS

- **The criteria for basic, before any list.** A list with no criteria is recall.
- **Each theory written as a sequence,** exactly as the second column sets it out.
- **Named sets with their authors,** six, eight and seven, not a single unattributed list.

SEE ALSO Slot I - 42 the theories of emotion evaluated at depth Slot I - 38 emotional intelligence SPRINT Day 4, general and developmental psychology

I - 40

Define learning. Write in brief about the basic tenets of different models of learning and their clinical importance in psychiatry. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Definition**	What counts as learning, and what is excluded
3	**The models drawn**	One axis, three branches, the theorists attached
3	**The basic tenets**	The mechanism of each model, one line each
2	**Clinical importance**	A named therapy standing under every branch

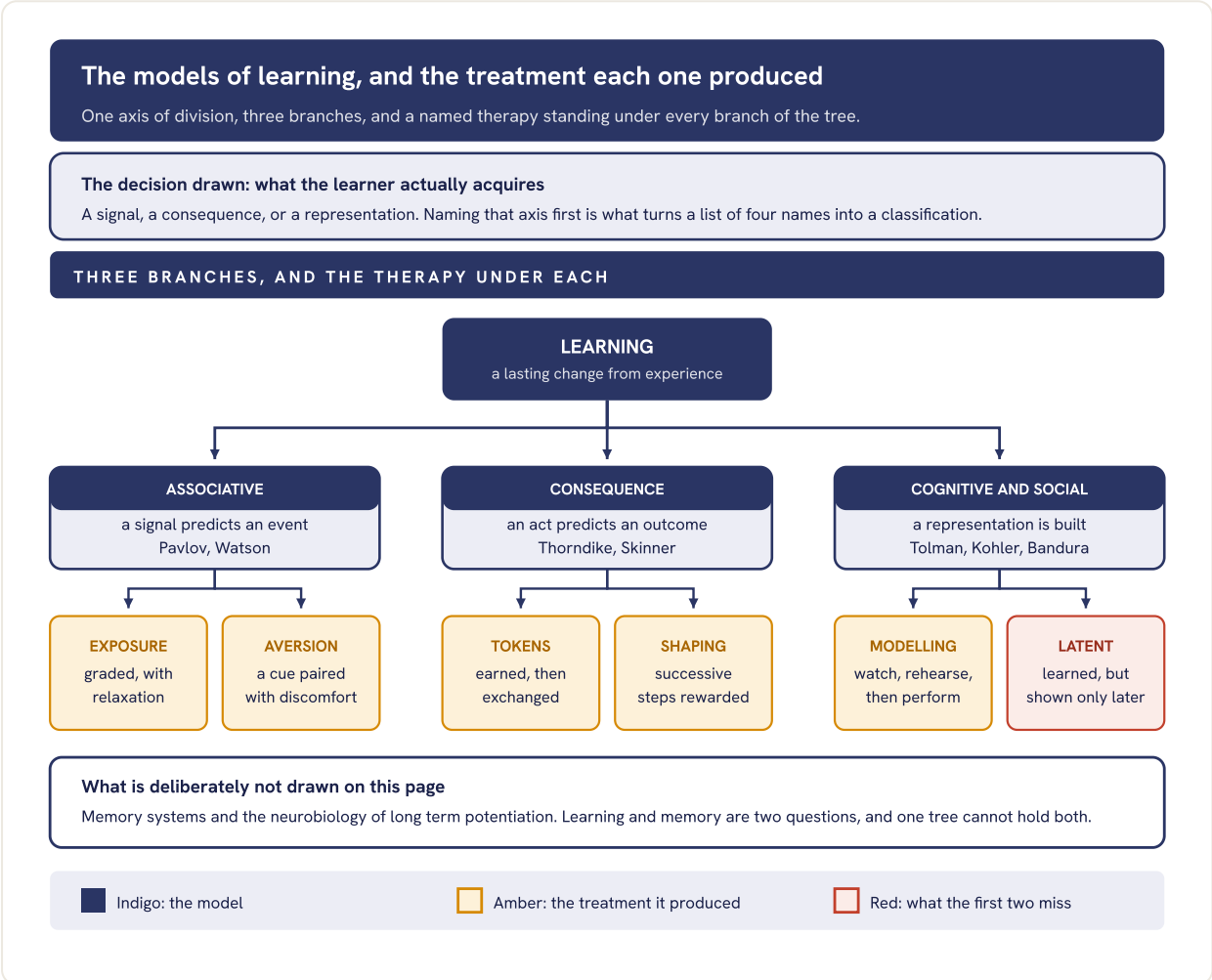


Fig 040.1 The models of learning divided on what the learner acquires, with the therapy each branch produced set in amber beneath it, and latent learning marked in red because it is the finding the associative and consequence models cannot account for.

- **The tenets, one line each.** Classical conditioning pairs a neutral stimulus with one that already elicits a reflex until it elicits that reflex alone. Operant conditioning makes a behaviour more or less likely by what follows it. Observational learning is acquired by watching a model and stored before it is performed. Cognitive learning builds an internal representation, shown by latent learning and by insight.

- **The definition, and what it excludes.** Learning is a relatively permanent change in behaviour, or in the capacity for it, following experience. Change from fatigue, maturation, injury or a drug is not learning.

PITFALL

Naming the models without naming the treatment each produced answers half the question. Clinical importance carries the second eight marks, and every branch has a named therapy under it.

WHAT EARNS THE MARKS

- **The definition with its exclusions,** before the classification begins.
- **The axis stated in one line.** A tree that does not name its axis is a list.
- **Every branch with its theorists,** Pavlov, Skinner, Tolman, Bandura.
- **A named therapy under each branch.** The amber leaves are the clinical marks.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 44 Pavlovian conditioning in behaviour therapy

SPRINT Day 5, social, learning and personality psychology

I - 41

Classical conditioning and Operant conditioning. Discuss their application in clinical setting with one example for each. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 Classical	The paradigm, and the four terms in their order
2 Operant	The four consequences, named without error
4 Applications	A named treatment and one example for each
2 Shared phenomena	Extinction, generalisation, discrimination, schedules

FEATURE	CLASSICAL	OPERANT	WHAT SETTLES IT
What is learned	A signal predicts an event	An act produces an outcome	**Is the behaviour elicited or emitted**
The response	Reflexive, autonomic, involuntary	Skeletal, voluntary, acting on the world	Could the person withhold it
Where the event sits	The signal comes before the response	The consequence comes after it	**Which side of the behaviour the event falls**
The law invoked	Contiguity of two stimuli, Pavlov and Watson	Effect of the consequence, Thorndike and Skinner	Pairing against payoff
Clinical example	Graded desensitisation for a phobia of heights	A token economy on a rehabilitation ward	Is the cue changed, or the consequence

- **The operant terms, used correctly.** Reinforcement raises the rate of a behaviour and punishment lowers it; positive means a stimulus is added and negative means one is removed. That gives four cells, not two. Negative reinforcement is therefore not a punishment, and it is precisely what maintains avoidance in every anxiety disorder.
- **What both paradigms share.** Acquisition, extinction, spontaneous recovery, stimulus generalisation and discrimination all appear on either side. Among the operant schedules a variable ratio produces the behaviour hardest to extinguish, which is why the machine rather than the salary is the model for gambling.

PITFALL

The commonest error is calling negative reinforcement a punishment. The second is offering exposure as the operant example: graded exposure works by extinguishing a conditioned response and belongs on the classical side of this table. Aversion therapy sits there too.

WHAT EARNS THE MARKS

- **Both paradigms defined before the comparison begins,** each with its theorists named.
- **The two amber cells,** elicited against emitted, and which side of the behaviour the event falls.
- **One named treatment and one concrete example for each,** which is what the stem asks for twice.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 44 Pavlovian conditioning in behaviour therapy **SPRINT** Day 5, social, learning and personality psychology

I - 42

Discuss various theories of emotions and critically evaluate James-Lange theory and its implications. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The field in brief	Four positions, one line each, in date order
2	The claim itself	James and Lange stated so it can be tested
4	The evaluation	Test by test, with a verdict on each
2	Implications	What survives, and what it is used for now

THE CLAIM, STATED SO IT CAN BE TESTED

James held that bodily change is not the expression of emotion but its cause: we are sad because we weep. Lange said the same of vasomotor change. Stated strongly, peripheral feedback is necessary and sufficient for feeling; stated weakly, it only modulates intensity.

THE TEST APPLIED	WHAT WAS FOUND	HOW THE THEORY STANDS	VERDICT
Cutting visceral feedback	Emotional behaviour persists in animals	Feedback is not necessary for it	**Strong form refuted**
Comparing autonomic patterns	Fear and anger look largely alike	Some patterning since demonstrated	Partly answered
Timing the visceral response	Viscera are slow, sparsely innervated	Facial and muscular feedback is faster	Partly answered
Injecting adrenaline	Arousal without any felt emotion	A cognitive label must be added	**Reframed by Schachter**
Studying spinal cord injury	Higher lesions, less intense feeling	Feedback modulates intensity	Weak form supported

- **The field, in one line each.** Cannon and Bard put the thalamus at the centre, sending to cortex and body at once. Schachter and Singer made emotion arousal plus a label. Lazarus put appraisal first. Papez and MacLean supplied the circuit, not the psychology. LeDoux separated a fast subcortical route from a slower cortical one.
- **Implications, the second half of the stem.** Facial feedback and its therapeutic offshoots; relaxation working on the body to change the feeling; beta blockade for performance anxiety, where removing the peripheral signal lowers the fear; interoceptive exposure in panic; and Damasio's somatic marker account of how bodily states bias decision.

WHAT EARNS THE MARKS

- **The strong and weak forms separated in the definition box.** Critical evaluation is impossible until the claim is stated precisely.
- **A verdict in every row,** not a list of criticisms with no conclusion attached.
- **The implications answered separately.** They carry their own marks and most answers omit them entirely.

SEE ALSO Slot I - 39 basic emotions and the theories in brief Slot I - 38 emotional intelligence SPRINT

Day 4, general and developmental psychology

I - 43

What do you understand by the term intelligence? Describe the one test commonly used for measuring intelligence which has been standardized for use in India. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	What intelligence is	A definition, and the quotient it is expressed as
2	The Indian tests	Named, with the one you will describe marked
3	The chosen test	Structure, administration, scoring
2	Interpretation	What the figure settles, and what it cannot

TEST	AUTHOR AND YEAR	AGE RANGE	WHERE IT IS USED
Malin's Intelligence Scale for Indian Children	Malin, 1969	Six to fifteen years	**The standard child scale, described below**
Binet-Kamat Test of Intelligence	Kamat, 1934	Childhood into early adulthood	Indian adaptation of the Binet tradition
Bhatia Battery of Performance Tests	Bhatia, 1955	Eleven years and above	Wholly performance based, so language and literacy do not limit it
Vineland Social Maturity Scale, Indian adaptation	Malin	Infancy to adulthood	Social maturity, not intelligence; used alongside, never instead

- **Structure of the chosen test.** An Indian adaptation of the Wechsler children's scale, with Indian norms and content. Eleven subtests in two halves. Six verbal: information, general comprehension, arithmetic, analogies and similarities, vocabulary, digit span. Five performance: picture completion, block design, object assembly, coding, mazes. It yields verbal, performance and full scale quotients.
- **Administration and scoring.** Individually administered by a trained examiner in about an hour, alternating the two halves to hold attention. Raw scores convert to scaled scores, and those to a deviation quotient of mean a hundred, standard deviation fifteen. Subtest scatter is read alongside the total.

INTERPRETATION, AND THE HONEST LIMITS

Used for intellectual disability certification, for specific learning disorder where a verbal and performance discrepancy matters, and for documenting decline against a baseline. The limits belong in the answer: norms drift, schooling inflates the verbal half, sensory and motor impairment depress the performance half, and a quotient never diagnoses disability alone.

WHAT EARNS THE MARKS

- **A definition before the test.** The first part of the stem carries its own marks.
- **One test chosen and named,** with author and year, rather than a survey of five.
- **The subtests in their two halves,** which is the body of the description.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 49 rating scales and their psychometric properties SPRINT Day 4, general and developmental psychology

I - 44

Discuss Pavlovian conditioning. Discuss its role in behaviour therapy. [5+5]

5 + 5 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The paradigm	Four terms, in the order in which they occur
3	The phenomena	Extinction onward, each with its named effect
3	In behaviour therapy	Each treatment traced back to a phenomenon
2	Why relapse happens	Extinction is inhibition, never erasure

THE PARADIGM

An unconditioned stimulus already produces an unconditioned response. A neutral stimulus presented repeatedly just before it comes to produce a similar response alone; it is then a conditioned stimulus, its effect a conditioned response. Rescorla's correction is worth a line: what is learned is prediction, not mere pairing.

- **The phenomena to name.** Acquisition, extinction when the signal comes without the event, spontaneous recovery after a rest. Generalisation and discrimination. Higher order conditioning. Experimental neurosis, from demanding a discrimination that cannot be made. Blocking, where a predictive cue prevents a new one being learned. Preparedness, which is why snakes condition readily and sockets do not. Taste aversion, learned in one trial across hours.
- **The therapy lineage.** Watson and Rayner conditioned fear in an eleven month old infant; Jones then removed a child's fear by pairing the animal with food and with unafraid children, the first behaviour therapy on record. Wolpe formalised it as systematic desensitisation: a graded hierarchy, a response incompatible with anxiety, exposure to each step until it settles. Response prevention in obsessive compulsive disorder, flooding, aversion therapy, cue exposure for craving, the bell and pad for enuresis and interoceptive exposure in panic all descend from it.
- **Conditioning as pathogenesis, not only treatment.** Conditioned craving and conditioned withdrawal explain relapse on returning to the place of use. Anticipatory nausea before chemotherapy is a conditioned response to the clinic. Agoraphobic avoidance begins as generalisation from a single panic.

PITFALL

Extinction does not erase the original learning. It builds a new inhibitory association tied to its context, which is why fear returns with time, in a new setting, or after one unexpected exposure.

WHAT EARNS THE MARKS

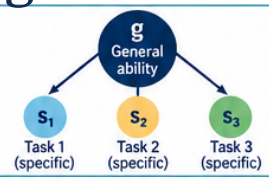
- **The four terms in the right order,** with prediction rather than pairing named as what is learned.
- **Named phenomena, not a paraphrase.** Blocking, preparedness and taste aversion are the ones most answers miss.
- **The relapse paragraph.** Context dependent extinction is the discriminating point on this stem.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 40 the models of learning classified SPRINT Day 5, social, learning and personality psychology

Theories of Intelligence and Tests

1 Charles Spearman
Two-factor theory (1904)

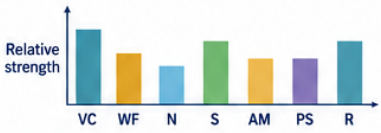
General ability shared across tasks + ability specific to each task.



General ability shared across tasks + ability specific to each task.

2 Louis Thurstone
Seven primary mental abilities (1938)

Verbal comprehension • Word fluency • Number • Space • Associative memory • Perceptual speed • Reasoning



Different strengths; abilities can correlate.

3 Raymond Cattell & John Horn
Fluid and crystallised intelligence

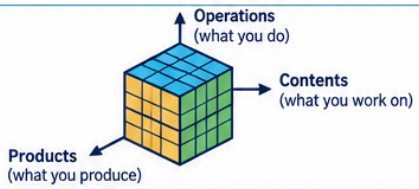
Fluid: solve unfamiliar problems.
Crystallised: use accumulated knowledge.



New reasoning versus learned knowledge.

4 Joy Paul Guilford
Structure of intellect

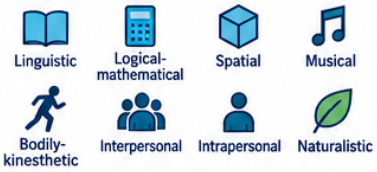
Operations × Contents × Products



A classification of abilities; includes divergent thinking.

5 Howard Gardner
Multiple intelligences (1983)

Linguistic • Logical-mathematical • Spatial • Musical • Bodily-kinesthetic • Interpersonal • Intrapersonal • Naturalistic



Originally seven; naturalistic added later.
Not learning styles.

6 Robert Sternberg
Triarchic theory (1985)

Analytical • Creative • Practical



Three complementary forms of intelligence for real-world functioning.

FOUR SELECTED INTELLIGENCE TESTS

Two international-origin tests + two Indian adaptations

INTERNATIONAL

Wechsler Adult Intelligence Scale
Fifth Edition: ages 16–90
Use: detailed adult cognitive profile and overall intelligence estimate.
Example: adult referred for comprehensive cognitive assessment.

India: select an appropriately normed edition; Fourth Edition India exists.

INTERNATIONAL

Raven's Progressive Matrices
Second Edition: ages 4–90
Use: nonverbal abstract reasoning with reduced language demands.
Example: person with limited proficiency in the test language.

Not culture-free; not a full cognitive battery.

INDIAN ADAPTATION

Malin's Intelligence Scale for Indian Children
Ages 6–15
Use: verbal and performance profile in school-age children.
Example: child with school difficulties needing intellectual assessment.

Legacy norms; does not diagnose a learning disorder alone.

INDIAN ADAPTATION

Binet–Kamat Test of Intelligence
Age-graded assessment
Use: developmental estimate of general intellectual functioning.
Example: child with suspected developmental delay.

Legacy norms; check manual, language and age suitability.

Theories differ in empirical support. No one-to-one theory–test mapping.
Select tests using current appropriate norms. Intellectual disability assessment also requires adaptive functioning and developmental history.

Theories of Intelligence and Tests. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Perception

Sensation: detection and transduction of sensory input. **Perception:** organisation and interpretation of that input.

Two processing directions

Bottom-up (data-driven)

Stimulus → sensory features → object recognition



Top-down (concept-driven)

Knowledge + context + expectations → interpretation of input

Knowledge (e.g. objects) + Context (e.g. setting) + Expectations (e.g. what is likely) → Interpretation of input

↕ Bottom-up and top-down processes interact continuously.

James Jerome Gibson

Direct perception: information in the environment guides perception and action. Affordances are action possibilities relative to the observer: a handle affords grasping.

Richard Langton Gregory

Constructivist theory: perception tests hypotheses informed by prior experience; helps explain ambiguity and illusions.

Gestalt organisation

Max Wertheimer ▪ Wolfgang Köhler ▪ Kurt Koffka

Gestalt = organised form. The whole differs from the sum of its parts.

Figure–ground



Edgar Rubin vase: reversible figure and ground (vase or two faces).

Proximity



Dots that are close together are seen as a group.

Similarity



Elements with similar features are seen as a group.

Closure



A complete circle is perceived despite missing parts.

Good continuation



Smooth, continuous lines are preferred over abrupt changes in direction.

Common fate



Elements moving in the same direction are seen as a group.

Prägnanz: favour a simple, stable organisation allowed by the stimulus.

Expectations and perceptual set

Context changes interpretation

A 13 C 12 13 14

Same input, different context.

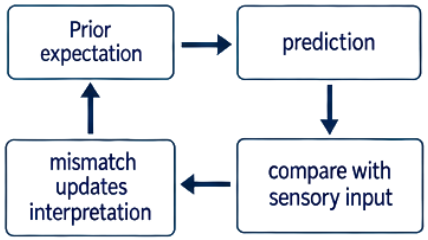
Perceptual set

Readiness to interpret input in a particular way; shaped by experience, context, motivation and emotion.

Example (auditory)

A creak may be heard as footsteps when frightened.

Predictive processing (proposed framework)



Influential model; neural details remain under investigation.

Illusion: a real stimulus is misperceived.
Hallucination: percept-like experience without a corresponding stimulus. Illusions can occur in health.

Gestalt grouping need not be conscious reasoning.

A changed report may reflect response bias; it does not by itself prove changed perception.

Perception. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Attention

Selection and prioritisation of information for processing; capacity is limited.

Focused	Sustained	Selective	Alternating	Divided	Executive control
respond to a specific stimulus.	maintain focus over time.	prioritise a target amid distraction.	switch task sets.	share resources.	inhibit distraction and maintain goals.
turn toward a bell.	monitor for a rare signal.	follow one conversation.	read, then calculate.	take notes while listening.	resist checking your phone.

These are overlapping functional categories.

Selective-attention theories

Donald Eric Broadbent (1958) — early filter



Selects by ear, pitch or location before semantic analysis. Challenge: some meaningful unattended information breaks through.

Anne Marie Treisman (1964) — attenuation



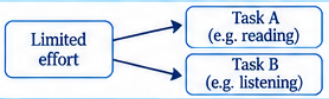
Unattended input is weakened, not erased. Personally meaningful words may cross a lower recognition threshold.

John Anthony Deutsch and Diana Deutsch (1963); Donald Arthur Norman (1968) — late selection



Selection follows meaning analysis in these historical models. Challenge: not all unattended input receives full semantic processing.

Daniel Kahneman (1973) — capacity model



Resources are allocated according to demands, goals and arousal. Shared demands create interference.

Load matters — Nilli Lavie

High perceptual load	High working-memory/ control load
fewer resources left for distractors.	poorer distractor control.
Complex target task (high load)	Demanding working-memory task (high load)
↓	↓
Uses more perceptual resources	Uses more central executive resources
↓	↓
Fewer resources available for distractors	Reduced capacity to inhibit distraction
↓	↓
Distractors less likely to be processed	Distractor interference more likely

Load theory: a testable account, with task-dependent findings.

Example

Cocktail party: track one voice; your own name may capture attention. Breakthrough is possible, not inevitable.

Clinical and exam notes

- Inattention is central to delirium and occurs in many conditions; one attention test is not a diagnosis.
- Divided attention is limited; apparent multitasking often includes rapid switching.
- Early versus late refers to selection relative to meaning, not reaction speed.
- Hearing a stimulus does not ensure conscious awareness.

Attention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Forgetting

Failure to retrieve previously learned information. Several mechanisms can operate together; poor *initial encoding* is a separate possibility.

Trace decay

Explanation

A memory representation weakens over time.



Proposed mechanism

Example

A briefly heard unfamiliar sound becomes harder to reproduce after a delay.

Evidence and limits

Time-related loss is observed, but time usually brings interference and contextual change. Decline alone cannot establish decay, especially in long-term memory.

Interference

Explanation

Previously learned information can compete with new or existing memories.

Proactive: old disrupts new



Example: The old password intrudes when recalling the new one.

Retroactive: new disrupts old



Example: After learning a new password, the old one is harder to recall.

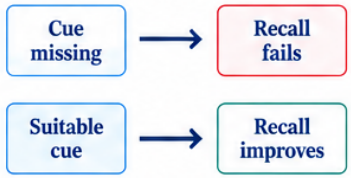
Evidence and limits

Controlled competing-learning experiments support interference; similarity and retrieval conditions matter. Do not infer literal erasure.

Retrieval failure

Explanation

Information may remain available but be inaccessible with the current cues.



Example

A name comes back when a colleague mentions where you met.

Endel Tulving and Donald Thomson: encoding specificity — cues work when they match how information was encoded.

Evidence and limits

Cue benefits support retrieval accounts; failure with one cue does not prove storage loss or an intact trace.

Motivated forgetting

Explanation

Forgetting can sometimes be influenced by goals, emotions or unconscious processes.

Suppression
Deliberate attempts to stop retrieval.
Example: choosing not to dwell on an embarrassing event.

Repression
Sigmund Freud's proposed unconscious exclusion of threatening material.

Evidence

Think/no-think studies can show reduced later recall after deliberate retrieval suppression; effects depend on task and strategy.

Limits

This does not by itself establish unconscious repression, trauma-specific amnesia, or the accuracy of a later recovered memory.

Hermann Ebbinghaus: forgetting curve



Describes a pattern, not its cause.

Relearning savings can reveal retained learning.

Exam distinctions

- **Proactive** = **OLD** interferes with NEW. **Retroactive** = **NEW** interferes with **OLD**.
- Forgetting is not always permanent loss.
- Neither confidence nor vividness alone establishes memory accuracy.

Forgetting. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Memory errors and distortions

Reconstructive memory — Frederic Charles Bartlett

Event details + prior knowledge + present context → reconstructed recall

Remembering rebuilds an account; reconstruction can be accurate or distorted. A schema is an organised knowledge framework.

Example: An unfamiliar story is retold using more familiar details.

Confabulation

False or misplaced recollections produced without intent to deceive, often with impaired memory monitoring. Not necessarily conscious gap-filling.

Provoked: elicited by a memory question. Asked about breakfast, a person reports yesterday's meal as today's.

"What did you have for breakfast today?" — "I had scrambled eggs and toast."

Spontaneous: arises without a memory-testing prompt; may guide behaviour. A retired person in hospital announces they must leave for work.

"I'd better get going now — I'm late for work."

Michael Kopelman (1987): distinguish by how the account arises. Spontaneous does not necessarily mean fantastic.

Confabulation	Experienced as recollection; no deliberate deception.
Deliberate lying	Knowingly false account intended to deceive.
Delusion	A belief held despite counterevidence; may involve the past but is not defined by a memory gap.

Clinical confabulation can accompany Korsakoff syndrome or frontal dysfunction; not diagnostic by itself.

Familiarity and recognition

False recognition: wrongly judging a new item or person as previously encountered.
Example: calling an unseen test word 'studied'.

Déjà vu: a present situation feels familiar despite knowing the familiarity is inappropriate.
Example: a first visit feels strangely familiar.

Jamais vu: something known feels unfamiliar.
Example: a familiar word suddenly looks strange after repetition.

These experiences can occur in health. Stereotyped episodes with other seizure features need clinical assessment; déjà vu alone is not epilepsy.

Source attribution

Source-monitoring error: remembering content but misattributing its origin.
Example: recalling a rumour as something personally witnessed.

Cryptomnesia: remembered material is experienced as a new, original idea.
Example: unknowingly reproducing a previously heard melody.

Earlier exposure → source forgotten → 'my new idea'

Unintentional; not deliberate plagiarism.

Distorted content

Retrospective falsification: past experience is unintentionally reshaped by present emotion, experience or beliefs.
Example: during depression, recalling a mixed past as entirely unsuccessful.

Not synonymous with delusional memory.

False memory: recollection of an event or detail that did not occur.
Example: remembering broken glass absent from a crash film.

Misinformation effect: misleading post-event information alters later memory reports.

Crash film → leading wording → later erroneous report

Elizabeth Loftus and John Palmer (1974): 'smashed' increased speed estimates and later reports of nonexistent glass.

Exam safeguards

Familiarity ≠ recollection of an episode. | A false belief need not be a false memory. | Confidence and vividness alone do not establish accuracy. | Reconstruction is a general process; confabulation is a distinct clinical phenomenon.

Memory errors and distortions. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Problem-solving and decision-making

Problem-solving finds a route to a goal; decision-making selects among alternatives.

Ways to solve

Algorithm: a defined sequence of steps. A correct, applicable algorithm yields the specified result; it need not be exhaustive or slow.

Long division: 84 ÷ 7 = 12

```
   1 2
7 ) 8 4
  - 7
    1 4
  - 1 4
      0
```

84 ÷ 7 = 12

Heuristic: a practical shortcut that reduces search; useful but not guaranteed.

Start → Subgoal → Goal

Means–ends analysis: reduce the gap by setting subgoals.

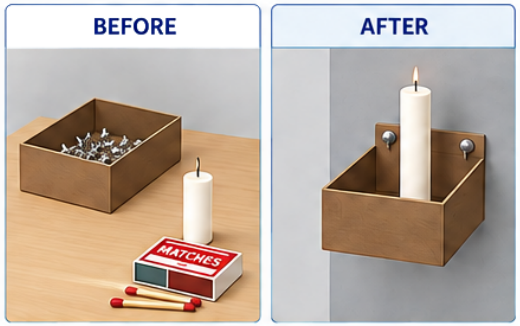
Insight: a solution seems to emerge suddenly after restructuring the problem. Wolfgang Köhler's work helped establish this Gestalt account.

An **'aha'** feeling does not guarantee correctness.

What blocks a solution?

Mental set — Abraham Samuel Luchins: persist with an old successful strategy despite a simpler option. Water-jar tasks demonstrate this.

Functional fixedness — Karl Duncker: see an object only in its usual role.



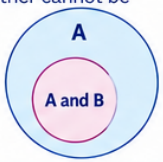
Drawing pins inside box; candle and matches on table.

Box attached to wall with drawing pins, candle in box (upright).

Reframe the box: container → candle support.

Countermove: restate the goal, list constraints, consider alternative uses, then test the solution.

Heuristics and biases — Amos Tversky and Daniel Kahneman

Pattern	Example / error
Availability	A recent vivid case makes that diagnosis seem too common.
Representativeness	A prototype match overrides the base rate (how common the condition is).
	Conjunction error: A and B together cannot be more probable than A alone. $P(A \text{ and } B) \leq P(A)$ P = probability 
Anchoring	The first estimate holds later judgement too close, despite new evidence.
Confirmation bias	Seek supporting findings and neglect disconfirming ones.
Framing	'90% survive' and '10% die' produce different preferences.
Overconfidence	Certainty exceeds demonstrated accuracy.
Sunk-cost bias	Continue a failing plan because of resources already spent.

Clinical and exam distinctions

Fast, intuitive versus slow, deliberative processing are useful descriptive modes, not two literal brain compartments.

Deliberation can also be biased; heuristics can be accurate.

Use base rates, alternatives and disconfirming evidence; reassess when new information arrives.

Biases occur in health and are not diagnostic of a psychiatric disorder.

Problem-solving and decision-making. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Motivation

Processes that initiate, direct and sustain behaviour.

One situation: a resident preparing for an examination.

Drive-reduction

Clark Leonard Hull (1943)

Core idea

A physiological deficit creates a drive; reducing it restores homeostasis (internal balance).



Same resident

Hunger interrupts revision; eating reduces the hunger drive.

Limit

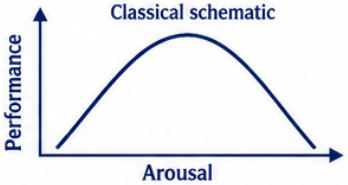
Explains the meal, not the whole ambition to qualify; curiosity can persist without a deficit.

Arousal approaches

Robert Mearns Yerkes and John Dillingham Dodson (1908)

Core idea

Seek a workable level of activation, rather than always reducing it.



Same resident

Seeks a challenge when bored; reduces distraction when overwhelmed.

Limit

The original study examined discrimination learning in mice. Human effects vary by task and person; no universal anxiety optimum.

Incentive approaches

Core idea

Anticipated outcomes attract or deter behaviour.



Same resident

Qualification and recognition make revision worthwhile; failure is an outcome to avoid.

Limit

The same reward has different value across people and situations. Incentives interact with internal needs and learning.

Cognitive approaches

Victor Harold Vroom

Core idea

Beliefs about effort, performance and valued outcomes influence motivation.



Same resident

Will practice improve performance?
Will performance lead to qualification?
How much do I value it?

Limit

A useful model, not a universal numerical law; goals, habits and emotion also matter.

Self-determination — Edward Deci and Richard Ryan

Intrinsic: revise because understanding is enjoyable.
Extrinsic: revise for a separable outcome, such as qualification.
Extrinsic motivation can be personally endorsed, not only externally controlled.



Supportive feedback and achievable progress can help sustain engagement.

Exam distinctions

- **Drive-reduction:** reduce a deficit.
- **Arousal:** regulate activation.
- **Incentive:** anticipated outcome.
- **Cognitive:** beliefs and goals.

- **Wanting** (motivational pull) differs from **liking** (pleasure).
- Several motives can operate at once; reduced motivation is not simply laziness.
- Abraham Maslow's needs hierarchy is a historical framework, not a rigid universal staircase.

Motivation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Creativity

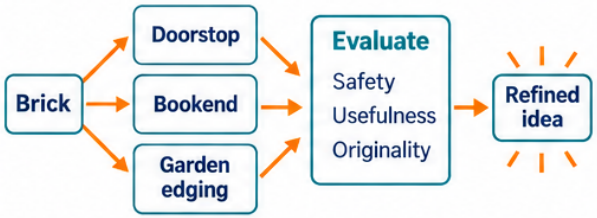
Producing ideas or work that are both novel and useful or appropriate.

Generate ↔ Evaluate

Joy Paul Guilford

Divergent thinking
Many varied possibilities.

Convergent thinking
Select or derive the best answer under constraints.



Both contribute to creativity; neither equals creativity alone.

Four dimensions (with brick examples)

Fluency	Number of relevant ideas.	e.g. doorstop, bookend, garden edging, paperweight ...
Flexibility	Different categories.	e.g. household item, office use, garden use.
Originality	Rarity relative to others.	e.g. use as a musical instrument.
Elaboration	Developing an idea with detail.	e.g. doorstop with non-slip base and decorative design.

Examples illustrate the dimensions; there is no universal scoring.

Creative process — Graham Wallas (1926)

1. Preparation
- Define the problem.
- Gather knowledge.

Example: study material for a revision diagram.

2. Incubation
- Set it aside.

Example: take a break and do something else.

3. Illumination
- An idea emerges, the 'aha'.

Example: see a visual analogy that links the ideas.

4. Verification
- Test, refine and implement.

Example: check accuracy and readability, then finalise the diagram.

Historical model, not a compulsory linear sequence. Incubation may help, but its mechanisms remain debated. Insight still needs checking.

Assessment

Alternative Uses Task
- List unusual uses for an ordinary object.
- Samples divergent thinking.
- **e.g. brick:** doorstop, bookend, garden edging, paperweight, musical instrument ...

Torrance Tests of Creative Thinking — Ellis Paul Torrance
- Verbal and figural tasks.
- Verbal commonly scores fluency, flexibility and originality.
- Figural scores fluency, originality, elaboration, abstractness of titles and resistance to premature closure.
- Scores depend on form and manual.

Remote Associates Test — Sarnoff Mednick
- Find one linking word.

COTTAGE / SWISS / CAKE
↓
CHEESE

- Convergent in response format; probes associative connections.

Use portfolios and real-world products as well; no single score captures all creativity. Language, culture, expertise and scoring affect results.

Context matters:
knowledge + motivation + opportunity + evaluation.

Avoid myths:
no universal intelligence-quotient threshold of 120; creativity is not confined to the right hemisphere.

Creativity is not a diagnosis.
Associations with bipolar traits do not prove that mania causes creativity; idea quantity is not useful, completed work.

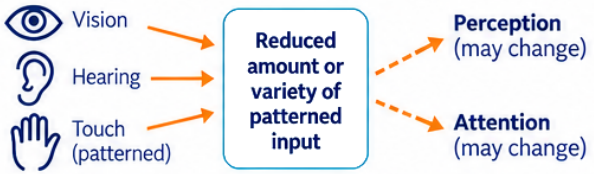
Creativity. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Sensory deprivation

Marked reduction in the amount or variety of external sensory input—not complete absence of sensation.

What is reduced?



Classic McGill work (1954), in Donald Olding Hebb's laboratory: translucent goggles, sound-damped cubicle and restricted patterned touch.



Flotation research — John Cunningham Lilly: buoyant water, reduced light and sound. Gravity, proprioception and internal bodily signals remain.



Gravity, proprioception and internal bodily signals remain.

Sensory deprivation — less input.
Perceptual deprivation = less meaningful variation, even if stimulation persists.
Social isolation = reduced contact with others.
Sleep deprivation = loss of sleep.
These may coexist but are different.

Possible effects—not inevitable

Perception

- Flashes or geometric patterns
- Complex visual experiences (e.g. scenes, faces or shapes)
- Altered body perception
- Illusions and hallucinations may occur.

Cognition

- Impaired sustained attention
- Difficulty organising thought
- Altered time estimation (time may feel slower or faster).

Emotion

- Boredom
- Restlessness
- Irritability
- Anxiety or distress
- Some experience relaxation.

Effects vary with duration, control, expectations, sleep and individual susceptibility.

There is no obligatory sequence from illusion to hallucination.

Proposed mechanisms

Reduced incoming sensory input → Disinhibition or perceptual **release:** internally generated activity becomes more prominent.

Weak sensory evidence → Greater influence of expectations: predictive-processing account.

Monotony, altered arousal and source-monitoring difficulty → Impaired interpretation or attribution.

These are complementary hypotheses, not interchangeable proven mechanisms.

Evidence limits:

- Classic small samples.
- Expectancy and demand characteristics.
- Sleep disruption and isolation can confound results.
- Hallucinations here do not establish a psychotic disorder.

Clinical comparison

Voluntary flotation

Reduced Environmental Stimulation Therapy (REST). Short, controlled sessions may reduce state anxiety.

Preliminary trials do not establish broad or lasting efficacy.

Prolonged involuntary isolation

Distress and perceptual/cognitive disturbance; social deprivation, threat and loss of control also matter.

Not equivalent to voluntary flotation.

Clinical settings

Visual loss may accompany Charles Bonnet visual hallucinations; hearing loss may accompany musical hallucinations.
Delirium is multifactorial.
Support orientation, daylight, sleep, spectacles and hearing aids; both overstimulation and understimulation can be problematic.

Sensory deprivation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

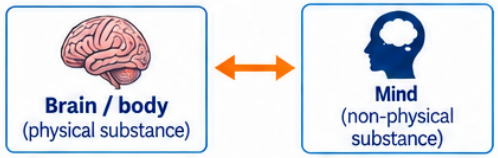
Credits and references: retained within the illustration.

Mind-body problem

How do brain processes relate to thought, feeling and subjective experience?

Substance dualism — René Descartes

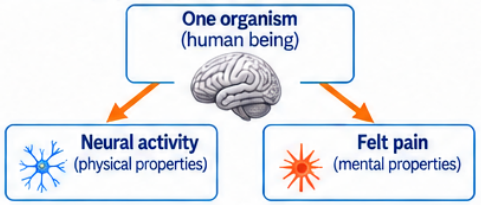
Mind and matter are distinct substances.
Interactionism: brain/body ↔ mind.



Example: injury produces pain; intention produces movement.
Challenge: how can different substances causally interact?

Property dualism

One physical organism has irreducible mental as well as physical properties.



Not simply two useful descriptions; claims irreducible properties.
Challenge: mental causation.

Physicalism / identity theory

Physicalism: reality is fundamentally physical.
Type identity theory identifies a kind of mental state with a kind of brain state.



Challenge: multiple realisability — could different nervous systems support the same mental state? Pain is not literally just C-fibre firing.

Functionalism — Hilary Putnam

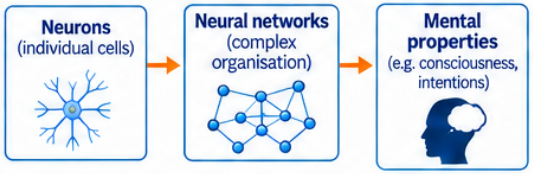
Mental states defined by causal role, not material alone.



Potentially different substrates, same role.
Challenge: does function explain how pain feels? Often physicalist, but not logically restricted to physicalism.

Emergentism

Mental properties arise with complex organisation.



Weak emergence: higher-level patterns remain physically explainable.
Strong emergence: irreducible novelty or causal powers.
These are different claims, not a settled midpoint.

Other positions

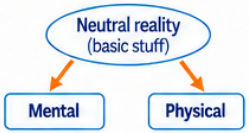
Epiphenomenalism
Brain processes give rise to conscious experience, but experience has no causal effect on physical events.



Idealism — George Berkeley
Reality is fundamentally mental.



Neutral monism — Bertrand Russell
Mental and physical derive from a more basic neutral reality.



Monism does not always mean materialism.

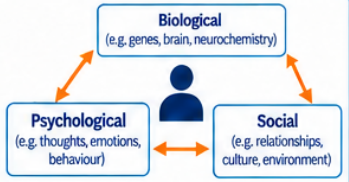
Experience and explanation

Qualia = felt qualities, such as the hurt of pain.
Joseph Levine: explanatory **gap** between physical description and experience.
David John Chalmers: hard problem — why is physical processing accompanied by experience?
Frank Cameron Jackson's Mary: a scientist knows all physical colour facts before first seeing colour; what does she learn?
Thought experiments are arguments, not empirical disproofs of physicalism.



Psychiatry

George Libman Engel's biopsychosocial model integrates biological, psychological and social influences.
A clinical framework, not a proof of property dualism or a solution to consciousnees.
Treatment-aetiology fallacy: benefit from a drug does not prove a single chemical cause.
Studying component mechanisms is not the same as eliminating psychological explanation.
Physicalism does not by itself settle free will or responsibility.



Exam rule: distinguish a philosophical position, a causal model and an empirical finding. No universally accepted solution.

Mind-body problem. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Social Learning

Learning from observing models and the consequences of their behaviour; direct reinforcement is not required for acquisition.

1 KEY ATTRIBUTIONS



Albert Bandura
Social learning and later social cognitive theory.



Richard Walters
With Bandura, social learning and personality development.



Albert Bandura, Dorothea Ross & Sheila Ross
Bobo doll studies showed imitation of modelled aggression; consequence studies distinguished acquisition from performance.

Laboratory findings do not establish that every observer will imitate in everyday life.

2 FOUR-PROCESS MODEL

Learning can occur without immediate performance.

ATTENTION
Notice relevant actions. Salience, similarity and credibility matter.

↓

RETENTION
Symbolic or verbal coding and mental rehearsal.

↓

REPRODUCTION
Translate representation into action. Ability, practice and feedback matter.

↓

MOTIVATION
Anticipated outcomes and incentives influence enactment.

3 MODEL TYPES



Live model
Person demonstrates behaviour.



Symbolic model
Film, story or other media model.



Verbal instruction
Describes how to act.

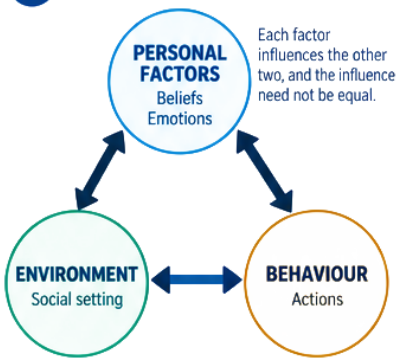


Coping model
Shows gradual improvement (e.g. overcoming fear).



Mastery model
Demonstrates fluent, competent performance.

4 RECIPROCAL DETERMINISM



Each factor influences the other two, and the influence need not be equal.

5 SELF-EFFICACY

Belief that one can execute a specific action.

Distinct from outcome expectation: **"Can I do it?"** (self-efficacy) | **"Will it help?"** (outcome expectation)

Four sources:

- **Mastery experiences** — Successful practice.
- **Vicarious experiences** — Seeing similar others succeed.
- **Verbal persuasion** — Credible encouragement.
- **Physiological and affective states** — Interpretation of arousal and mood.

Not global self-esteem or proof of actual skill.

6 CONSEQUENCES AND SELF-REGULATION

Direct reinforcement
Consequence to learner (e.g. reward, praise).

Vicarious reinforcement/ punishment
Observed consequences influence enactment.

Self-regulation
Self-observation → compare with standards → self-reaction (e.g. self-reward or self-criticism).

Reinforcement affects motivation; it is not necessary for all observational acquisition.

7 CLINICAL UTILITY

Social skills training	Participant modelling for phobia	Parent training	Child interventions	Rehabilitation	Health behaviour change
Demonstration, rehearsal, feedback.	Observe coping then practise with support.	Model consistent adaptive responses.	Live or video models of functional skills.	Model and practise daily living skills.	Build self-efficacy and realistic goals.

Models can also transmit maladaptive habits; observation is not destiny.

8 WORKED CASE

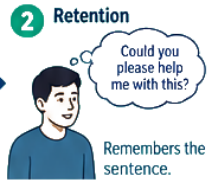
Fictional example: Alex struggles to make requests.

1 Attention



Watches therapist calmly request help.

2 Retention



Remembers the sentence.

3 Reproduction



Rehearses with feedback.

4 Motivation



Tries it when ready and sees the benefit.

Repeated successful practice builds self-efficacy.

9 COMPARE: CLASSICAL, OPERANT AND SOCIAL LEARNING

Classical conditioning
A cue predicts an event (learned association).

Operant conditioning
Own action changes consequences (learned through reinforcement or punishment).

Social learning
Learn from others through representation (observation and modelling).

Limits:

- Attention
- Ability
- Prior learning
- Context constrain performance.

Based on: Paper I question 37; Bandura's social cognitive theory.

All illustrations © 2024. Created for psychiatry exam revision. Not for commercial use.

Social Learning. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Emotional Intelligence and Theories

Question 38 • Definition, components and relationship with intelligence quotient • 2 + 4 + 4 marks

DEFINITION

Peter Salovey & John D. Mayer, 1990

The capacity to recognise and distinguish emotions in oneself and others, and use emotional information to guide thought and action.



Different models define emotional intelligence differently.

1 ABILITY MODEL – Salovey & Mayer

Perceive emotions → Use emotions to support thinking → Understand emotions → Manage emotions



Measurement: performance tasks — what a person can do.

Example: Mayer–Salovey–Caruso Emotional Intelligence Test.

Check edition: newer test domains and scoring have been revised.

2 MIXED MODEL – Daniel Goleman

Classic five components:

Self-awareness • Self-regulation • Motivation • Empathy • Social skills



Measurement: self-report and observer ratings of competencies.

Broader than cognitive ability; later competency frameworks differ.

3 EMOTIONAL–SOCIAL MODEL – Reuven Bar-On

Classic five areas:

Intrapersonal • Interpersonal • Adaptability • Stress management • General mood



Measurement: Emotional Quotient Inventory — self-report.

Current inventory versions reorganise these scales.

4 TRAIT MODEL – Konstantinos V. Petrides

Emotional self-perceptions within personality.

Wellbeing • Self-control • Emotionality • Sociability



Measurement: Trait Emotional Intelligence Questionnaire — self-report.

How capable I believe I am, rather than demonstrated task performance.

ONE EXAMPLE THROUGH THE FOUR ABILITY BRANCHES

Fictional case: a colleague speaks sharply.

1. **Perceive:** notice an angry tone.
2. **Use:** let the emotional cue focus attention.
3. **Understand:** consider frustration or fear; check rather than assume.
4. **Manage:** pause, ask calmly, choose a useful response.



EMOTIONAL INTELLIGENCE AND INTELLIGENCE QUOTIENT

Intelligence quotient: broad reasoning and cognitive performance.



Related, but not interchangeable; the association depends on the measure.

Ability emotional intelligence shares some cognitive variance; trait measures overlap with personality.

Neither guarantees success. Added predictive value depends on outcome and study design.

Emotional intelligence: emotional abilities or self-perceptions, depending on the model.



Related, but not interchangeable; the association depends on the measure.

Ability emotional intelligence shares some cognitive variance; trait measures overlap with personality.

Neither guarantees success. Added predictive value depends on outcome and study design.



Clinical use: explore emotion identification and regulation. A score does not diagnose a psychiatric disorder.
Alexithymia means difficulty identifying and describing feelings; it is not synonymous with low emotional intelligence.

Always name the model and the measure.

Emotional Intelligence and Theories. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Basic Emotions and Theories

Question 39 • The requested six theories, with Plutchik's model

1 William James & Carl Lange — Peripheral feedback

Stimulus → See a threat
Bodily response → heart races
Perception of bodily response → feel bodily changes
Felt emotion → feel afraid

Example: See a threat → heart races → feel afraid.
Caveat: Bodily feedback contributes, but is not the whole explanation.

2 Walter Cannon & Philip Bard — Parallel response

Stimulus → Central processing → Felt emotion; Bodily arousal

Emotion and bodily response arise in parallel in the classical model.

3 Stanley Schachter & Jerome Singer — Two-factor theory

Bodily arousal + Interpretation of context → Experienced emotion

Example: A racing heart can be interpreted as fear or excitement.
Arousal + cognitive label.

4 Richard Lazarus — Cognitive appraisal

Event → Appraisal of personal meaning → Emotional response

Example: Threat appraisal → fear; challenge appraisal → determination.
Appraisal can be rapid and outside awareness.

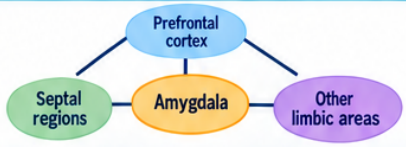
5 James Papez — Circuit model (1937)

Hippocampal formation → Fornix → Mammillary bodies → Mammillothalamic tract → Anterior thalamic nuclei → Cingulate cortex → Cingulum → Entorhinal cortex → Hippocampal formation

Historical emotion circuit; these pathways are especially important for memory.

6 Paul MacLean — Limbic-system expansion

Expanded the emotion network to include the amygdala, septal regions and connections with prefrontal cortex.
Triune-brain idea: reptilian, paleomammalian, neomammalian.
Historical model — not literal evolutionary layers or three independent brains.



ROBERT PLUTCHIK'S EMOTION WHEEL

Eight basic emotions • Opposites • Intensity • Blends



Intensity increases toward the centre.

Opposite emotion pairs:
- Joy ↔ Sadness
- Trust ↔ Disgust
- Fear ↔ Anger
- Surprise ↔ Anticipation

Blends between adjacent emotions (clockwise from top):
Optimism (Joy–Anticipation)
Love (Joy–Trust)
Submission (Trust–Fear)
Awe (Fear–Surprise)
Disapproval (Surprise–Sadness)
Remorse (Sadness–Disgust)
Contempt (Disgust–Anger)
Aggressiveness (Anger–Anticipation)

A theoretical map of emotion, not a diagnostic test.

DISTURBANCES OF MOOD & AFFECT

Symptoms and signs—not diagnoses by themselves.

- **Depressed mood** — sustained sadness or low mood.
- **Elevated mood** — unusually increased cheerfulness or elation.
- **Anhedonia** — reduced ability to experience pleasure.
- **Anxiety** — apprehension about anticipated threat.
- **Irritability** — increased readiness to become annoyed or angry.
- **Labile affect** — rapid, marked shifts in emotional expression.
- **Blunted / flat affect** — reduced / nearly absent outward expression.
- **Incongruous affect** — expression inconsistent with the situation or stated experience.
- **Alexithymia** — difficulty identifying and describing feelings.

Mood: sustained internal emotional state.
Affect: observed emotional expression.

Modern view: bodily feedback, appraisal, context and distributed brain networks interact. These are complementary historical perspectives, not a list with one final winner.

Basic Emotions and Theories. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Learning Theories

Major models and three core mechanisms compared

A relatively enduring change in behaviour or capacity, arising from experience.
Exclude transient fatigue, drug effects and maturation alone.

What is acquired?

ASSOCIATIVE / SIGNAL BASED
A cue predicts an event.

Ivan Pavlov — classical conditioning
John Watson — conditioned emotional responses

CONSEQUENCE BASED
An action changes its outcomes.

Edward Thorndike — trial and error; law of effect
Burrhus Frederic Skinner — operant conditioning

COGNITIVE & SOCIAL
Internal representations and observed models.

Edward Tolman — cognitive maps; latent learning
Wolfgang Köhler — insight learning
Albert Bandura — observational / social cognitive learning

This is an exam grouping: operant conditioning is also associative in the broader sense.

KEY COGNITIVE IDEAS

- **Latent learning:** learning may remain unexpressed until an incentive appears.
- **Insight:** reorganising a problem can produce an apparent sudden solution.
- **Social cognition:** person, behaviour and environment influence one another.

CLASSICAL CONDITIONING

Core link: stimulus → stimulus
Response is elicited by a learned cue.

Pavlov's example

Before: Food (unconditioned stimulus) → Salivation (unconditioned response)

Pairing: Tone + food → Salivation

After: Tone (conditioned stimulus) → Salivation (conditioned response)

Main processes

Acquisition • extinction • spontaneous recovery • generalisation • discrimination

Clinical example

Phobia: a formerly neutral cue evokes fear. Exposure builds new learning that the cue need not predict harm.

Systematic desensitisation pairs graded exposure with relaxation; relaxation is not required for every exposure method.

OPERANT CONDITIONING

Core link: behaviour → consequence
Consequences alter future behaviour.

Four consequences

	ADD stimulus (positive)	REMOVE stimulus (negative)
BEHAVIOUR INCREASES	Positive reinforcement	Negative reinforcement
BEHAVIOUR DECREASES	Positive punishment	Negative punishment

Positive = add; negative = remove.
Reinforcement increases behaviour; punishment decreases it.

Main processes

Shaping: reward successive approximations.
Schedules: fixed / variable ratio; fixed / variable interval.

Clinical example

Token economy: completing agreed self-care earns tokens exchangeable for rewards.

Maintenance example: escaping a feared situation relieves anxiety, strengthening avoidance — negative reinforcement.

SOCIAL LEARNING

Core link: observe → represent → perform
Learning can occur without direct reward.

Bandura's four processes

1. **Attention** — notice the model.
2. **Retention** — remember the action.
3. **Reproduction** — ability to enact it.
4. **Motivation** — reasons to perform it.

Main constructs

Vicarious reinforcement: observing a model's reward influences performance.
Self-efficacy: belief in one's ability to perform a task.
Reciprocal determinism: person ↔ behaviour ↔ environment.

Clinical example

Social skills training: watch a therapist model an assertive request, rehearse it, then use it.

Learning and performance are distinct.

Also include NON-ASSOCIATIVE learning: habituation = reduced response to repeated stimulation; sensitisation = increased responsiveness.

Exam anchors: prediction • consequences • representation. | Negative reinforcement is not punishment. Extinction does not necessarily erase original learning.

Learning Theories. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

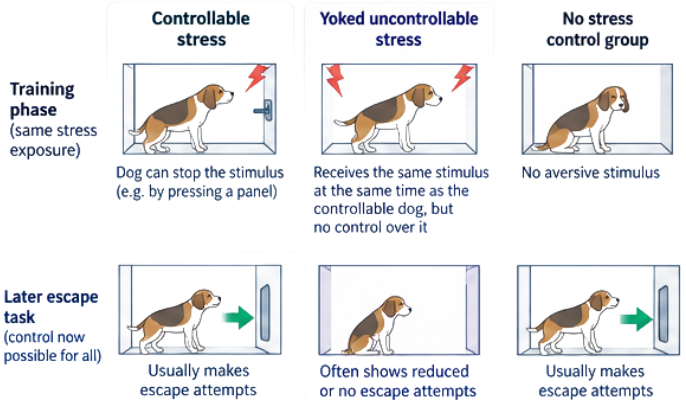
Credits and references: retained within the illustration.

Learned Helplessness

The expertimenal parradigm

Martin Seligman and Steven Maier

Animals are exposed to the same aversive stimulus, but differ in whether they can control it. They are later tested in a situation where escape is possible.



The critical difference is control during the initial stress exposure, not the amount of stress received.

Original theoretical model

Martin Seligman and Steven Maier

Experience of events that are uncontrollable

↓

Learned response–outcome independence
Actions do not affect outcomes

↓

Reduced initiation of behaviour
Difficulty learning when control is possible

↓

Emotional and motivational effects
Distress, passivity

Attributional reformulation in humans

Lyn Abramson, Martin Seligman and John Teasdale (1978)

In humans, the effects of negative events depend on how they are explained (attributed).

Attributional dimension	Negative style (risk for helplessness)	More adaptive style
Internality (cause)	**Internal** "I am to blame"	**External** "It was due to external factors"
Stability (duration)	**Stable** "It will last"	**Changeable** "It can improve"
Globality (scope)	**Global** "It affects everything"	**Specific** "It is limited to this situation"

Example: exam setback

Helplessness-prone explanation

"I failed because I am incapable forever at everything."

- Internal, stable and global attribution
- Associated with low self-esteem, persistence of negative expectations and generalisation to other areas

More helpful explanation

"I didn't pass this time, but I can improve with different strategies and support."

- Specific and changeable attribution
- Encourages problem solving and further effort
- Combine with practical support (e.g. study skills, mentoring, addressing environmental barriers)

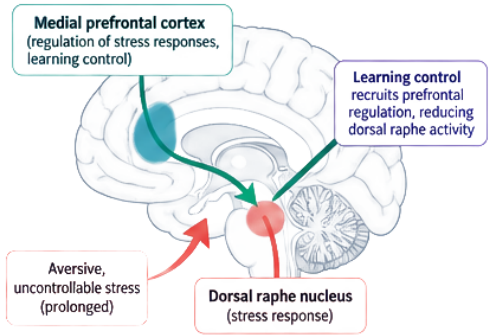
Modern revision and neural model

Steven Maier and Martin Seligman (2016)

Prolonged exposure to aversive, uncontrollable stress can produce a default state of passivity. Learning control again recruits medial prefrontal regulation of dorsal raphe stress responses.

Key points

- Uncontrollable stress can engage neural systems that promote behavioural inhibition (passivity).
- Learning control activates medial prefrontal cortical regulation of dorsal raphe activity, reducing stress responses.
- Based primarily on animal-model evidence.
- This is a research model, not a diagnostic brain map in humans.



Animal-model evidence

Clinical and practical implications

Approaches to reduce learned helplessness

Graded, achievable actions
Start with small, realistic steps to rebuild a sense of control.

Feedback linking action to outcome
Provide clear evidence that actions can make a difference.

Attributional work
Identify and challenge unhelpful internal, stable and global explanations.

Behavioural activation
Increase engagement in rewarding and meaningful activities.

Address real environmental barriers
Remove or reduce practical obstacles (e.g. academic, financial, social) that limit control.

Key distinctions and caveats

Learned helplessness (control)	Hopelessness (future expectations)
Belief that one's actions do not affect outcomes in the present.	Negative expectations about the future, regardless of current control.

Scope of the model

- Helps to explain aspects of depression, such as passivity, reduced motivation and negative attributional style.
- Does not explain every cause of depression or every patient.
- Psychological difficulties are not simply due to insufficient. effort, and may arise in the context of significant biological, psychological and social factors.

Learned Helplessness. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Classical Conditioning

A previously neutral cue acquires predictive significance through experience with another event.

KEY FIGURES

- **Ivan Pavlov** — Conditioned reflexes in dogs (1900s)
- **John Watson & Rosalie Rayner** — Little Albert fear conditioning (1920; historical, ethically problematic)
- **Mary Cover Jones** — Early fear reduction in Little Peter (1920s)
- **Joseph Wolpe** — Systematic desensitisation (1950s)
- **Robert Rescorla & Allan Wagner** — Prediction error theory (1970s)
- **Leon Kamin** — Blocking (1960s)
- **John Garcia** — Taste-aversion learning (1960s)

THE CLASSICAL CONDITIONING PARADIGM (PAVLOV'S DOGS)

Note: the conditioned response need not exactly copy the unconditioned response.

1. BEFORE CONDITIONING

- **Food** (unconditioned stimulus, US) → **Salivation** (unconditioned response, UR)
- **Tone** (neutral stimulus, NS) → **No food-related salivation** (no conditioned response)

2. DURING CONDITIONING

Tone followed by food (NS + US) → **Salivation** (unconditioned response, UR)

3. AFTER CONDITIONING

Tone (conditioned stimulus, CS) → **Salivation** (conditioned response, CR)

TIMING OF THE CUE AND EVENT

Different temporal relationships produce different learning. Delay is often most effective. The success of conditioning depends on biological preparation and timing. Prediction matters more than simple coincidence.

1. DELAY

Cue (CS) / Event (US)

Cue starts before event and overlaps.

Time →

2. TRACE

Cue (CS) ---- Event (US)

Cue ends, gap, then event.

Time →

3. SIMULTANEOUS

Cue (CS) / Event (US)

Cue and event together.

Time →

4. BACKWARD

Event (US) Cue (CS)

Event occurs before cue.

Time →

KEY PHENOMENA IN CLASSICAL CONDITIONING

Acquisition	Extinction	Generalisation	Discrimination	Higher-order conditioning	Blocking	Preparedness	Taste aversion	Experimental neurosis
Cue gains predictive value through pairing with event.	Cue without expected event reduces conditioned response.	Similar cues evoke a similar response.	Differentiate between predictive and nonpredictive cues.	A new cue paired with an established conditioned cue can also become conditioned.	A previously learned predictor limits learning about an additional cue.	Some associations (e.g. fear, taste aversion) are learned more readily due to evolutionary predispositions.	Can develop after just one trial and across long delays (e.g. hours).	Historical finding of behavioural disruption under difficult, conflicting discrimination.

WHY LEARNING CHANGES

Prediction error = outcome received – outcome expected.

- Positive prediction error increases associative strength.
- Negative prediction error decreases associative strength.
- Extinction commonly creates new, competing learning, not erasure of the original association.

RELAPSE AFTER EXTINCTION

- **Spontaneous recovery** — Return of responding after time has passed (even without further exposure).
- **Renewal** — Return of responding outside the context where extinction occurred.
- **Reinstatement** — Return of responding after re-exposure to the unconditioned event.

Use a variety of contexts and practise retrieval of new learning to reduce relapse.

CLINICAL UTILITY

Phobias	Graded exposure / new safety learning.
Systematic desensitisation	Hierarchy + relaxation + graded exposure.
Panic	Interoceptive exposure to feared sensations.
Obsessive-compulsive disorder	Exposure and response prevention also interrupts operant ritual reinforcement.
Cue-triggered craving	Conditioned cues inform formulation; cue exposure benefits vary.
Enuresis	Alarm training has conditioning components.
Anticipatory nausea	Clinic cues can acquire nausea responses.
Aversion therapy	Historical/selective application, not routine first-line care.

WORKED EXAMPLE: FEAR CONDITIONING AND RECOVERY

1. TRAUMATIC EXPERIENCE

Dog bite (unconditioned stimulus) → fear (unconditioned response).

2. CONDITIONED FEAR

Later, dog sight (conditioned stimulus) → fear (conditioned response).

3. NEW LEARNING THROUGH SAFE EXPOSURE

Repeated, planned encounters without harm builds new, competing expectations (safety learning).

Avoidance can maintain fear through negative reinforcement (the relief from anxiety strengthens avoidance behaviour).

EXAM PITFALLS

- Exposure is not always paired with relaxation.
- Extinction is not guaranteed erasure of learning.
- Biological constraints and cognitive expectations modify conditioning.
- Be clear about timing, prediction and specific phenomena (e.g. blocking, renewal, reinstatement).

Classical Conditioning. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Operant Conditioning

Behaviour changes as a function of its consequences.

1. KEY THEORISTS AND CONTRIBUTIONS

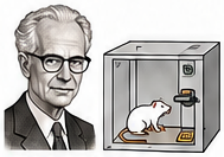
Edward Thorndike



- Puzzle box
- Trial and error learning
- Law of effect

"Behaviours followed by satisfying consequences are more likely to recur.

Burrhus Frederic Skinner



- Operant chamber
- Shaping and schedules of reinforcement

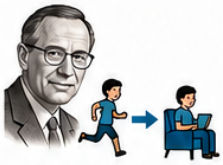
"Consequences shape behaviour."

Charles Ferster & Burrhus Frederic Skinner



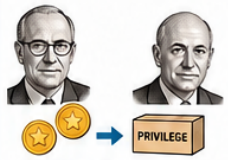
- Reinforcement schedules
- Systematic analysis of operant behaviour

David Premack



- High-probability behaviour can reinforce lower-probability behaviour (Premack principle).

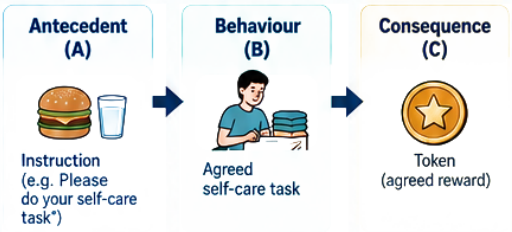
Teodoro Ayllon & Nathan Azrin



- Token economy
- Tokens exchanged for real rewards
- Used in clinical and institutional settings.

2. ABC FUNCTIONAL ANALYSIS

Discriminative stimulus signals when a consequence is available; it does not force a reflex.



3. THE FOUR QUADRANTS OF OPERANT CONDITIONING

Define by observed effect, not intention.

Positive/negative mean add/remove a stimulus; reinforcement/punishment mean increase/decrease behaviour.

	ADD STIMULUS (+ Positive)	REMOVE STIMULUS (– Negative)
BEHAVIOUR INCREASES (↑ more likely)	**Positive reinforcement** Add a pleasant stimulus (e.g. praise after practice) → more practice.	**Negative reinforcement** Remove an aversive stimulus (e.g. fastening seatbelt stops buzzer) → more fastening.
BEHAVIOUR DECREASES (↓ less likely)	**Positive punishment** Add an aversive stimulus (e.g. reprimand for interruption) → less interruption If it actually decreases.	**Negative punishment** Remove a pleasant stimulus (e.g. loss of game privilege) → less rule-breaking If it actually decreases.

4. REINFORCERS

Primary reinforcers



- Unlearned biological value (e.g. food, drink, rest)
- Meet basic needs.

Conditioned reinforcers



- Learned value (e.g. tokens, praise)
- Gain value through association.

Generalised conditioned reinforcers



- Linked to several rewards (e.g. money)
- Highly flexible across situations.

The effectiveness of a reinforcer depends on the **individual, context, timing** and **current motivation.**

5. REINFORCEMENT SCHEDULES

Schedule	Definition	Typical pattern of responding	Key point
Continuous	Every target response	Every response reinforced; rate varies	Useful for acquisition of new behaviour.
Fixed ratio (FR)	After a set number of responses	High rate, often a pause after reinforcement	e.g. FR5.
Variable ratio (VR)	After varying number of responses	High, persistent rate	Very resistant to extinction. e.g. gambling.
Fixed interval (FI)	First response after a set time	Low at first, then accelerates before reinforcement available	e.g. FI 10 min.
Variable interval (VI)	First response after varying time	Steady, moderate rate	Relatively resistant to extinction.

Ratio = number of responses. Interval = elapsed time. Partial reinforcement often resists extinction.

6. OPERANT TECHNIQUES

Shaping
Reinforce closer approximations to the target behaviour.

Chaining
Link established steps to build a complex behaviour.

Prompting and fading
Add prompts then gradually withdraw help.

Differential reinforcement
Reinforce alternative or incompatible responses, or absence of target behaviour.

Extinction
Stop maintaining reinforcement. Temporary burst may occur (extinction burst).

Response cost
Remove earned reinforcer (e.g. lose tokens).

Time-out
Temporary removal from positive reinforcement, not simply any pause.

Generalisation
Transfer behaviour across people, settings and situations.

7. CLINICAL UTILITY

- **Token economies** — Rehabilitation and self-care (e.g. inpatient units).
- **Contingency management** — Reinforce agreed, measurable targets in substance-use care.
- **Parent training** — Consistent reinforcement of adaptive behaviour.
- **Behavioural activation** — Re-engagement with rewarding activities.
- **Skills training** — Shape and rehearse functional tasks.

Use with consent and dignity. Basic needs (e.g. food, water, safety, warmth) are never contingent rewards.

8. MAINTENANCE LOOP: ANXIETY AND AVOIDANCE



Relief negatively reinforces avoidance. Escape ends an ongoing aversive stimulation; avoidance prevents or postpones it.

KEY POINTS AND LIMITATIONS

- Punishment does not teach a replacement skill and can produce fear or avoidance.
- Reinforce useful alternatives.
- Behaviour need not be consciously chosen.
- A reward is a reinforcer only if future behaviour increases.

Source basis: Weave Paper I questions 40–41; standard learning theory.
Not a substitute for formal teaching.

Operant Conditioning. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

Applied Behaviour Analysis

Functional Behaviour Chain (ABC)

Behaviour occurs in a context and has a consequence.
When the consequence meets a need, the behaviour may be maintained.



If this escape **contingency is repeated, the behaviour may be maintained.**
Observation of the ABC pattern suggests a possible **function, but does not prove it.**

Functions of Behaviour

Common functions that maintain behaviour.

Function	Example
Attention	Gaining social attention (e.g. adult response, peer interaction)
Access to item or activity	Obtaining a preferred item or activity (e.g. toy, tablet, free time)
Escape or avoidance	Avoiding or ending a demand or aversive situation (e.g. difficult task, noisy environment)
Automatic reinforcement	Behaviour is intrinsically reinforcing (e.g. sensory stimulation, pleasure)

From Problem Behaviour to Functional Communication

Replace challenging behaviour with a more appropriate and effective way to meet the same need.

1. **Teach a functional communication response**
   e.g. "Can I have a break please?"
2. **Reinforce the communication**
   Give the requested break when appropriate.
3. **Adjust task difficulty**
   Match demands to current skills and provide supports.
4. **Gradually build tolerance**
   Slowly increase task duration or complexity with ongoing reinforcement and support.



Descriptive Functional Assessment vs Experimental Functional Analysis

Different methods are used to understand the function of behaviour.

	Descriptive functional assessment	Experimental functional analysis
Focus	Observes behaviour in natural environments	Systematically manipulates antecedents and consequences
How it works	Interviews, records, direct observation (e.g. ABC charts)	Tests different conditions (e.g. attention, demand, tangible, play/alone) to identify maintaining consequences
Outcome	Generates a hypothesis about function	Provides stronger evidence for function
Use	Often first step, guides intervention planning	Useful when function is unclear or behaviour is severe

The Seven Dimensions of ABA

Applied Behaviour Analysis is defined by seven key dimensions (credited to Donald Baer, Montrose Wolf and Todd Risley).

1. **Applied (social significance)**
   Focuses on behaviours that are socially important and meaningful to the individual.
2. **Behavioural (observable and measurable)**
   Targets are clearly defined, observable and measurable.
3. **Analytic (demonstrated relation)**
   Demonstrates a functional relation between the intervention and behaviour change.
4. **Technological (replicable procedures)**
   Interventions are described in sufficient detail to be replicated.
5. **Conceptually systematic (learning principles)**
   Based on established principles of learning.
6. **Effective (meaningful change)**
   Produces behaviour change that is meaningful for the individual.
7. **Generality (across time and settings)**
   Skills and behaviour change are maintained over time and generalise to other settings, people and situations.

Key Methods and Principles

Common strategies used in ABA.

- **Operational definition**
  Clearly define the target behaviour (e.g. "pushes worksheet away" as an observable action).
- **Baseline**
  Measure current frequency, duration or other relevant metric before intervention.
- **Antecedent supports**
  Modify the environment (e.g. adjust task difficulty, provide choices, visual supports).
- **Prompting and fading**
  Provide prompts to teach a skill, then gradually reduce support.
- **Shaping and chaining**
  Reinforce successive approximations or link steps to build complex skills.
- **Differential reinforcement**
  Reinforce desired behaviour (e.g. functional communication) and withhold reinforcement for the problem behaviour.
- **Generalisation and maintenance**
  Plan for practice across people, settings and over time to maintain gains.

Reinforcement vs Punishment

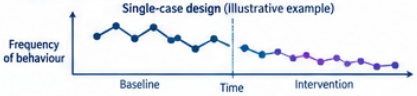
	Reinforcement	Punishment
	Increases the likelihood of a behaviour occurring in the future.	Decreases the likelihood of a behaviour occurring in the future.
	Positive reinforcement Adds a desirable event (e.g. praise, access to an activity).	**Positive punishment** Adds an aversive event (e.g. reprimand).
	Negative reinforcement Removes an aversive event (e.g. task is removed), which increases the behaviour. This is not punishment.	**Negative punishment** Removes a desirable event (e.g. loss of a privilege).

Example ABC and Intervention Plan

Situation	Antecedent	Behaviour	Consequence	Function (hypothesised)	Intervention strategies
Math lesson	Difficult worksheet given	Pushes worksheet away and says "No!"	Teacher removes task	Escape from demand	• Teach request for break • Adjust task difficulty • Reinforce appropriate request • Gradually build tolerance

Measuring Outcomes

Evaluate change in behaviour and quality of life using appropriate designs, particularly **single-case designs** where suitable.



Consider multiple outcomes:
- Frequency
- Duration
- Context and function
- Skill acquisition
- Quality of life (e.g. participation, independence)

Ethical Considerations and Scope

- Collaborative and meaningful goals developed with the individual and family or carers.
- Obtain assent and consent, and respect communication and autonomy.
- Use the least restrictive supports necessary.
- Do not suppress harmless self-regulation behaviours solely for conformity (e.g. stimming) when they are not harmful.
- Do not use painful or aversive procedures.
- ABA is a scientific approach with applications beyond autism (e.g. education, mental health, acquired brain injury).
- Outcomes vary and evidence is intervention-specific.

Applied Behaviour Analysis. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Learning, memory and general psychology.

Credits and references: retained within the illustration.

AREA 05 OF 12 · P1-A09

Psychometry and assessment

9	9.1%	I-45	I-53
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 4 SINGLE 5

REVISION ORDER

The three projective slots together: they are one body of material asked at three widths, and the catalogue slot half writes the other two. The two rating-scale slots next, they share their psychometrics. The mini mental state examination and the structured assessment slot after that, both short. The personality-tools slot and the socioeconomic slot are independent of everything else in this area.

WHAT IS IN THIS AREA

Projective testing in three slots: the Thematic Apperception Test in full, the classification with any one instrument described in detail, and the catalogue. The mini mental state examination. The classification of rating scales with the psychometric properties and the standardisation of a newly built intelligence test. The scales of depression, with one described as used in practice. Structured clinical assessment, and a slot that wants five theories of personality evaluated with four assessment tools. One slot here asks for the determination of socioeconomic strata in India. That is a social classification question the boards set inside an assessment area, and it is answered on its page as it was set.

I - 45

What is apperception? Describe administration and interpretation of Thematic Apperception Test. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT

DNB, KNRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Apperception	Perception plus the meaning the perceiver adds
4	Administration	Cards, instruction, sessions, the inquiry
3	Interpretation	Hero, need, press, thema, across stories
1	Standing	A battery supplement, never a diagnosis

THE DEFINITION

Apperception is perception interpreted: a new percept absorbed into, and given meaning by, the perceiver's past experience, needs and expectations. Make the stimulus ambiguous enough and the meaning can come only from the perceiver, which is the idea Murray built the test on.

STAGE	WHAT THE TESTER DOES	WHAT IS RECORDED
Selection	Picks a subset from the card series using the age and sex codes each card carries, one card being blank	Which cards, and in what order
Instruction	Asks for a story with what led up to the scene, what is happening, what the figures think and feel, and how it ends	**The story, verbatim, in the subject's own words**
Sessions	Around twenty cards over two sittings classically; about ten in ordinary clinical use	Latency to first word, time per card
Inquiry	Returns afterwards to ask about vague endings, omitted figures and refusals	Blocks, evasions and corrections

The cards are copyrighted. Describe the procedure and the codes, never the pictures and never a story.

- **Murray's frame.** Identify the hero, the hero's needs, the press acting on the hero from outside, and the outcome. Needs and press together give the thema.
- **Read across, not down.** A single story means little. Recurrent theme, repeated conflict, constant emotional tone and stereotyped resolution across the set are the data.
- **The limit.** Formal systems such as Bellak's exist, but routine use stays qualitative, so interpreter dependence is the stated weakness. It yields no diagnosis and supplements a battery when self-report is guarded.

WHAT EARNS THE MARKS

- **Apperception defined as meaning added to a percept,** not as a synonym for perception.
- **The instruction written out in its four parts.** Past, present, thoughts and feelings, ending.
- **Hero, need, press and thema named as the interpretive frame,** with Murray attached.
- **The honest limit.** Supplement, not a diagnostic test, and interpreter dependent.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

I - 46

Describe the methods of determination of socioeconomic strata in India. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why it is measured	A covariate in every Indian clinical study
3	The household-head route	Kuppuswamy: education, occupation, income
3	The per capita route	Prasad, and why it needs re-indexing
2	Rural and asset routes	Pareek, standard of living, wealth index

SCALE	UNIT IT SCORES	DOMAINS SCORED	WHERE IT FITS
Modified Kuppuswamy	Head of the family	Education, occupation, monthly family income	**Urban and semi-urban, the default in Indian psychiatry papers**
B G Prasad	Per capita monthly income	Income alone, in five graded classes	**Rural and urban, single-variable, quick**
Udai Pareek	The rural household	Caste, occupation, education, land, housing, possessions, social participation	Village and agrarian samples
Standard of living index	Household assets	Housing, sanitation, fuel, durables, land	Large surveys where income recall fails
Wealth index	Household assets	Same asset set, weighted by principal component analysis	National survey work and quintile comparison

Amber marks the two an examiner expects you to be able to apply, not merely name.

- **Kuppuswamy, the structure.** Three components scored separately: education one to seven, occupation one to ten, family income one to twelve. The total runs from three to twenty-nine and falls into five classes, upper, upper middle, lower middle, upper lower and lower.
- **The number you must not memorise.** The income slabs of both Kuppuswamy and Prasad are re-indexed every year to the consumer price index for industrial workers. Quote the year of the revision you used, never a remembered rupee figure.
- **Choosing one.** Head of family for urban clinic samples, per capita for community samples, asset based where income is under-reported or seasonal.
- **Where they fail.** Joint families dilute per capita income, agricultural income is seasonal and under-declared, and none of these scales measures caste disadvantage or debt.

WHAT EARNS THE MARKS

- **Named scales, not the phrase socioeconomic status.** Four names on the page is four ticks.
- **Kuppuswamy's three components with their score ranges,** and the five classes they produce.
- **The re-indexing rule stated explicitly.** It is the single point that separates a current answer from a copied one.
- **One line on choosing between them** for a stated study setting.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 83 the National Mental Health Survey

I - 47

Mini mental state examination. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Author, year, and the one job it does
4 Structure	Seven domains, thirty points, the allocation
2 Interpretation	The traditional cut, and what shifts it
2 Limits in India	Literacy load, and the adaptation to use instead

THE DEFINITION

The Mini Mental State Examination (Folstein, Folstein and McHugh, 1975) is a brief clinician-administered screen of global cognitive function, scored out of thirty and completed in about five to ten minutes. It screens. It does not diagnose, it does not localise, and it is not a neuropsychological battery.

DOMAIN	POINTS	WHAT IT SAMPLES
Orientation to time	5	Temporal orientation, the earliest to go
Orientation to place	5	Spatial orientation across nested levels
Registration	3	Immediate encoding of new material
Attention and calculation	5	Sustained attention and working memory
Recall	3	Short-term retention after interference
Language	8	Naming, repetition, comprehension, reading, writing
Construction	1	Visuoconstructional copying

Thirty points across seven domains. The amber rows are where delirium and early dementia declare themselves first.

- **The cut.** Below twenty-four traditionally suggests cognitive impairment, with twenty-four to thirty read as unimpaired, eighteen to twenty-three as mild and seventeen or below as severe. Age and education move the threshold, so the cut is a prompt to test further, never a diagnosis.
- **What it misses.** It is insensitive to mild impairment and largely blind to executive and visuospatial failure: a patient can score full marks and still be grossly dysexecutive. The Montreal Cognitive Assessment, also out of thirty, is preferred when early change is the question.
- **The Indian caveat.** It is language and literacy loaded, so Western cut-offs misclassify non-literate patients. Use a validated vernacular adaptation such as the Hindi Mental State Examination, and screen in a language the patient reads.

WHAT EARNS THE MARKS

- **The point allocation reproduced,** domain by domain, summing to thirty.
- **The cut given with its qualifier.** A bare "less than twenty-four" without age and education loses the second mark.
- **Screen, not diagnosis,** written as a sentence rather than assumed.
- **The vernacular adaptation named.** It is the Indian mark on this question.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 95 disorders of consciousness SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

I - 48

What do you understand by projective tests? Name different projective tests and describe any one in detail. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **The projective hypothesis**	Ambiguous stimulus, hidden key, broad band
2 **The classification**	Five response modes, named tests under each
4 **One in detail**	Rorschach: material, phases, scoring codes
2 **Standing**	What it is good for, and the honest weakness

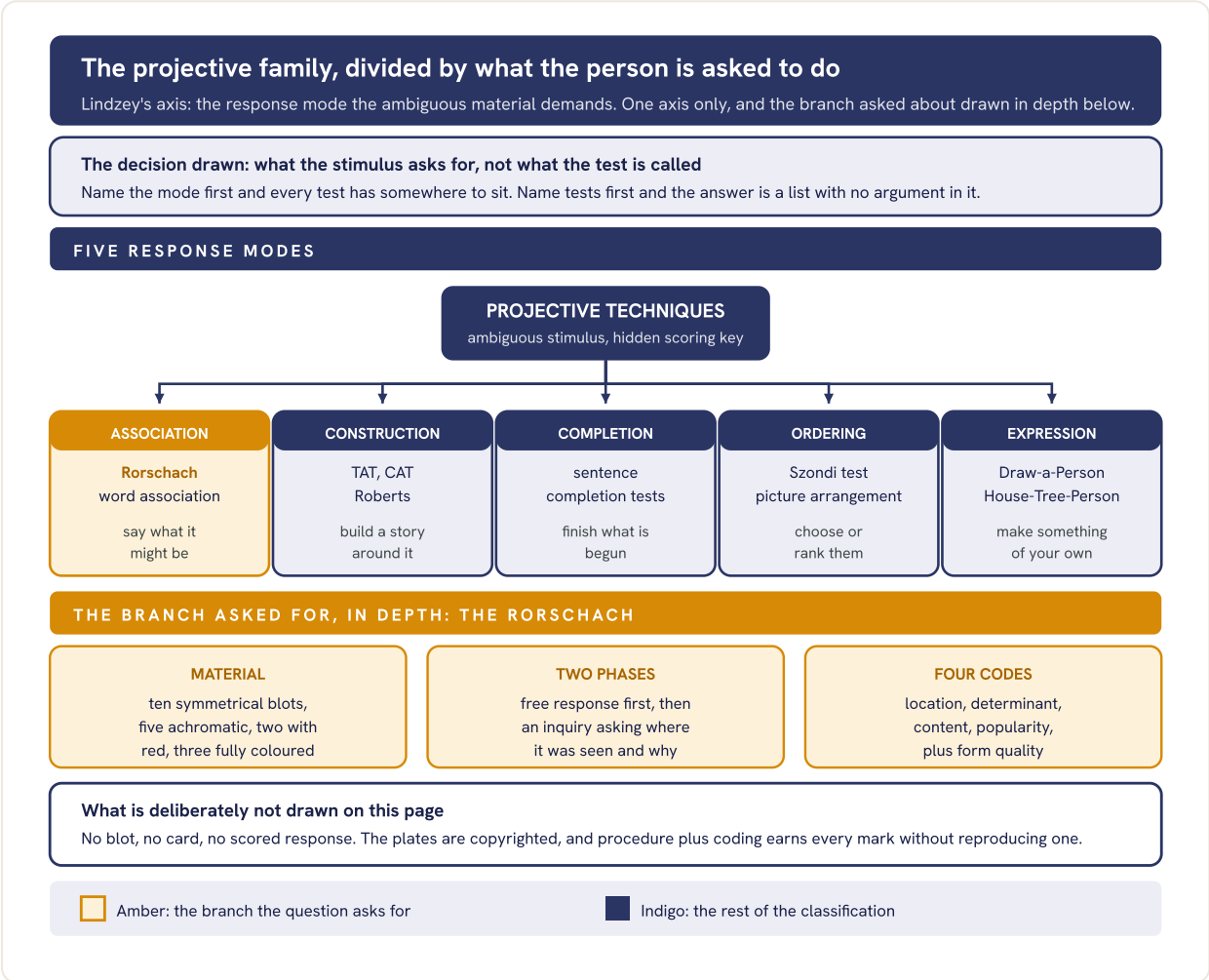


Fig 048.1 The projective techniques divided on one axis, the response mode, with the association branch opened out into the material, the phases and the coding of the Rorschach.

- **The projective hypothesis.** When the stimulus is ambiguous and the scoring key hidden, the structure the person imposes must come from within, so needs, conflicts and defences are read off it. Sampling is broad band and it is harder to fake than self-report.
- **Rorschach in one line.** Hermann Rorschach, Psychodiagnostik, 1921; scored today by the Exner Comprehensive System or its successor, the Rorschach Performance Assessment System.

- **The honest weakness.** Inter-rater and criterion validity are contested, norms are culture bound, administration is long. It supplements a battery when thought process is the referral question, never standing alone.

WHAT EARNS THE MARKS

- **The axis named before the list.** Response mode, then the five branches, then the tests.
- **Every branch of the plate reproduced.** Five modes with a named test under each is the two-mark listing part complete.
- **Depth only in the branch chosen,** material, phases and coding, and nowhere else.
- **The contested psychometrics named,** not defended and not hidden.

SEE ALSO Slot I - 45 the Thematic Apperception Test Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

Projective Tests

Definition • classification • administration • interpretation • clinical utility

DEFINITION

Relatively ambiguous or open-ended tasks used to explore patterns in perception, narratives and personality functioning.

PROJECTIVE HYPOTHESIS

- Responses may reflect personal concerns, needs and conflicts.
- They also depend on stimulus, context, culture and ability.
- Not direct access to the unconscious.

CLASSIFICATION OF PROJECTIVE TESTS (Gardner Lindzey - response mode)

1. ASSOCIATION

- Rorschach
- Word association (Carl Jung)

2. CONSTRUCTION

- Thematic Apperception Test
- Children's Apperception Test
- Roberts storytelling tests

3. COMPLETION

- Sentence completion, e.g. Rotter Incomplete Sentences Blank
- Sacks Sentence Completion Test

4. CHOICE / ORDERING

- Szondi
- Picture-selection techniques (historical)

5. EXPRESSION

- Draw-a-Person
- House-Tree-Person
- Kinetic family drawing

RORSCHACH

Hermann Rorschach, Psychodiagnostik, 1921.

MATERIAL 10 cards: 5 black/grey, 2 black/grey with red, 3 multicoloured.



Rorschach card — Science Museum Group

TASK Report what each blot might be.

ADMINISTRATION

- Rapport and standard instructions.
- Response phase: record responses verbatim.
- Inquiry / clarification: where and what features made it look that way.
- Follow chosen system's administration rules.

CODING (five clearly defined rows)

Location	Whole blot or detail used.
Determinants	Form, colour, shading or perceived movement.
Content	Category of what is perceived.
Form quality	Fit between response and blot contours.
Popularity	Frequency in normative responses.

SYSTEMS John Exner's Comprehensive System. Rorschach Performance Assessment System. **Do not mix coding systems or norms.**

INTERPRETATION

- Patterns across the protocol can inform perceptual organisation, thinking and interpersonal functioning.
- Interpret supported variables in context.
- No single response proves psychosis or a personality trait.

UTILITY Supplementary personality and thought-process assessment when appropriate to the referral question.

THEMATIC APPERCEPTION TEST

Christiana Morgan & Henry Murray, 1935.

APPERCEPTION Perception given meaning through past experience, needs and expectations.

MATERIAL 31 cards: 30 black-and-white/grayscale pictures + 1 blank; manual. Selected subset used; classical protocol: 20 cards over two sessions.



TAT kit — Hogrefe

FOUR STORYTELLING PROMPTS

1. What led up to the scene?
2. What is happening now?
3. What do characters think and feel?
4. How does it end?

RECORD

- Verbatim story, response latency, behaviour and relevant clarification.
- Ask neutral follow-up questions.

MURRAY INTERPRETATION FRAMEWORK

Hero	Central character.
Need	Motivation or wish.
Press	Environmental demands or influences.
Thema	Interaction of need and press.
Outcome	Resolution.

Look across stories for recurring conflicts, relationships, emotional tone and endings.

SCORING Qualitative thematic approaches and structured systems, including Bellak's framework and Social Cognition and Object Relations Scale — Global Rating Method. Evidence depends on scoring method.

UTILITY Explore interpersonal representations, motivations and conflicts to inform formulation; corroborate with interview and other measures.

OTHER IMPORTANT EXAM EXAMPLES

CHILDREN'S APPERCEPTION TEST

Leopold & Sonia Bellak.

- Typically ages 3–10.
- 10 animal-scene cards.
- Human-figure version also exists.
- Storytelling adapted to developmental level.

SENTENCE COMPLETION

- Finish incomplete statements.
- Rotter / Sacks examples.
- Explores attitudes and concerns.
- More structured, language and literacy dependent.

WORD ASSOCIATION

Carl Jung tradition.

- Responses and response patterns historically explored for emotionally salient associations.
- Latency alone is nonspecific.

DRAWING TECHNIQUES

Karen Machover: projective Draw-a-Person.

- John Buck: House-Tree-Person.
- Draw then inquire.
- Distinguish projective interpretation from developmental drawing scores.
- Isolated drawing signs do not establish abuse, conflict or diagnosis.

CLINICAL STANDING

- Supplement a multimethod assessment.
- Standardised administration, trained scoring and relevant norms matter.
- Reliability and validity differ across tests, systems and individual scores.
- Cultural/language effects and examiner bias require attention.
- Not a lie detector; not immune to deliberate response distortion.

EXAM ANSWER ROUTE

1. Define.
2. Classify with examples.
3. Describe chosen test's material, administration, scoring and interpretation.
4. Utility and limitations.

For question **48:** 2 + 2 + **6** marks. Related questions **45** and **53.**

Projective Tests. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-48.

Credits and references: retained within the illustration.

I - 49

Classify psychiatry rating scales. What are the psychometric properties of a psychiatric scale? Describe different procedures of standardization of a newly developed intelligence test. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Classify the scales	By rater, by purpose, by breadth, by scaling
2	The properties	Reliability, validity, and sensitivity to change
5	Standardisation	Items, pilot, fixed procedure, norms, IQ scale
1	Release	What has to be true before it is used

STEP	WHAT IS DONE	EVIDENCE IT YIELDS	PROPERTY SECURED
Blueprint and items	Define the construct, write a surplus of items, pilot them	Difficulty and discrimination	Content validity
Fix the procedure	Freeze instructions, order, timing, stop rules, scoring key	One administration everywhere	Standardisation
Norming	A large sample stratified by age, sex, education, region	Age-graded norm tables	Comparability
Transformation	Raw score to a deviation quotient, fixed mean and spread	The deviation IQ	Interpretability
Reliability, validity	Re-test, split-half, alternate forms, inter-rater; then correlate against an established test	A coefficient per method	Both, in that order

STATUS: WHAT HAS TO BE TRUE BEFORE IT IS USED

A test becomes usable when the procedure is frozen, the norms come from the population it will be used on, and a manual exists that a second examiner can follow to the same score. An imported test used without local norms is unstandardised, whatever its reputation abroad.

- **Classifying the scales.** By rater, self, clinician or informant. By purpose, screening, diagnostic, severity or outcome. By breadth, global against symptom specific. By scaling, categorical, ordinal, Likert or visual analogue.
- **The properties.** Reliability is consistency: re-test, internal consistency, alternate forms, inter-rater agreement. Validity is accuracy: face, content, criterion and construct evidence. A severity scale needs one more, sensitivity to change.
- **The order that matters.** Reliability is necessary and not sufficient, since a test can be consistently wrong. Validity evidence comes after reliability, never instead of it.

WHAT EARNS THE MARKS

- **A classification with a stated axis,** not an unsorted list of scale names.
- **Reliability and validity each broken into their named subtypes.**
- **Norming written as a step separate from procedure fixing.** They are the two halves of standardisation and most answers merge them.

SEE ALSO Slot I - 52 scales in research and depression instruments Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

Positive and Negative Syndrome Scale

PANSS • Standard 30-item version (Kay, Fiszbein & Opler, 1987)

Purpose and what the PANSS assesses

Purpose
Clinician-rated symptom severity and treatment change in schizophrenia and related psychotic disorders, primarily adults.

Not a diagnostic, cognitive or disability test
Not a stand-alone measure.

How it is rated
A trained clinician uses a semi-structured interview, behavioural observation and collateral information.

Usual reference period
Past 7 days.

What the subscales measure

Positive symptoms
Added experiences or behaviours (e.g. delusions, hallucinations).

Negative symptoms
Reduced expression or engagement (e.g. blunted affect, withdrawal).

General psychopathology
Includes anxiety, depression, attention and other symptoms.

Scoring and subscales

30 items, each rated 1–7
Where 1 = absent and 7 = extreme
No zero rating

Subscale	Number of items	Possible range	Typical items include
Positive (P)	7	7 – 49	Delusions, hallucinations, conceptual disorganization, excitement, grandiosity, suspiciousness, hostility
Negative (N)	7	7 – 49	Blunted affect, emotional withdrawal, poor rapport, passive social withdrawal, lack of spontaneity
General psychopathology (G)	16	16 – 112	Anxiety, depression, guilt, poor attention, unusual thought content
Total (P + N + G)	30	30 – 210	Sum of all three subscales

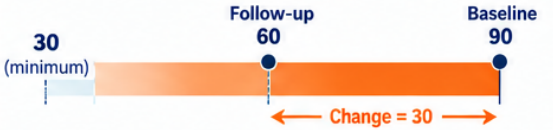
Worked example (invented data)
P21 + N14 + G40 = 75

Positive (7–49)	Negative (7–49)	General (16–112)	Total
21	14	40	75

Change over time and interpretation

Floor-adjusted percentage reduction
Use the correct method and state your study convention.

Baseline total score	90
Follow-up total score	60
Minimum possible score	30
Absolute change	90 – 60 = 30
Baseline above floor	90 – 30 = 60
Percentage reduction (floor-adjusted)	30 / 60 × 100 = **50%**



Without subtracting the floor (i.e. 30/90 × 100), the result is **33.3%**, which differs.

Interpretation

- Higher scores indicate more symptoms.
- No universal diagnostic or mild/moderate/severe cutoff.
- A single total score can hide different positive, negative and general psychopathology profiles.

Key distinctions and important notes

Minimum score is 30, not 0
The total score is the sum of all three subscales.

Negative score is not a pure measure of primary negative symptoms
Depression, sedation and extrapyramidal symptoms may contribute.

Symptomatic remission (research convention)
Uses eight specified items, each rated ≤ 3 for at least 6 months. It is not a total-score cutoff and not functional recovery.

No disability percentage or entitlement from PANSS alone
PANSS does not provide a disability rating or entitlement to services.

Positive and Negative Syndrome Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Brief Psychiatric Rating Scale

BPRS • 18-item version with 1–7 scoring

Overview and Key Points

Purpose	Broad psychiatric symptom severity and monitoring, chiefly adults with psychotic or severe affective illness.
Administration	Trained clinician integrates patient report and observed behaviour in a clinical interview.
Timeframe (for this sheet)	**Past week.** Keep this timeframe consistent for ratings and follow-up.
What it is NOT	Not a diagnostic test, not a cognition battery, not a disability percentage.
Domains (for teaching)	• Psychotic experiences and suspiciousness • Disorganized thinking • Withdrawal and reduced affect • Anxiety, depressed mood and guilt • Hostility, excitement and motor/behavioural abnormalities Grouped for teaching, NOT fixed official subscales.

Scoring (1–7 for each of 18 items)

Score	Symptom severity rating
1	Not present
2	Very mild
3	Mild
4	Moderate
5	Moderately severe
6	Severe
7	Extremely severe
0	**NOT ASSESSED** (in the 18-item form, 0 means not assessed, not absent)

Total possible range (when all 18 items assessed)

18 – 126

Hypothetical worked example

Items rated	Score per item	Subtotal
6 items	1	6
8 items	3	24
4 items	5	20
18 items total	**6 + 24 + 20 = 50**	

Interpretation and Use

What does the total mean?	Higher total = greater symptom burden.
No universal cutoff	There is no single universal diagnostic cutoff or mild/moderate/severe band; published thresholds depend on the population and scoring version.
Look beyond the total	Equal totals may conceal different symptom patterns. Compare item profiles and repeated ratings under the same version and timeframe.
Limitations	• Rater training, interview quality and cultural interpretation matter. • Sedation and motor adverse effects can influence observed signs. • Total alone misses everyday functioning and risk.



Key Exam Distinctions

Original 1962 development had 16 items.
18-item and expanded 24-item versions coexist. Always name the item count and rating convention.

This sheet uses the 18-item form with a total range of 18 – 126.
Not the PANSS 30 – 210.

BPRS does not provide separate PANSS positive/negative/general totals.

No diagnosis, disability certification or entitlement from a total alone.

Source: Overall & Gorham 1962; San Mateo County 18-item form.

Brief Psychiatric Rating Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Indian Disability Evaluation and Assessment Scale - IDEAS

IDEAS • Government-modified 2002 scoring version; 2024 guidance caveat

1. PURPOSE & DOMAINS

Purpose

To assess functional disability associated with mental illness, chiefly for adult psychiatric use.

Not a measure of symptom severity or intelligence.

Who assesses?

A trained mental-health professional assesses, based on caregiver interview, patient interview and records.

Legal certification requires the notified medical authority.

Four domains (each scored 0–4)

Self-care	Daily personal care (e.g. bathing, dressing, eating)
Interpersonal activities	Social relationships and interactions
Communication and understanding	Communication and understanding
Work	Employment, household work OR education

Scoring for each domain

0 = none **1** = mild **2** = moderate **3** = severe **4** = profound

Four-domain subtotal: 0 – 16

What it measures

Assesses real-world functioning and course of illness.

This government-modified version has no single past-week symptom window.

2. MODIFIED-VERSION SCORING

A. Add illness-duration score

< 2 years	= **1**
2 – 5 years	= **2**
6 – 10 years	= **3**
> 10 years	= **4**

B. Global score

Global score = four-domain subtotal + duration weight

Maximum score = **20 (16 + 4).**

C. Worked example (invented)

Self-care	2
Interpersonal activities	1
Communication and understanding	2
Work	3
Four-domain subtotal	**= 8**
Eight-year illness (6 – 10 years) adds	**= 3**

Global score = 8 + 3 = 11

D. Modified-version published bands

Global score	Disability band	Indicative disability(%)
0	None	0%
1 – 6	Mild	< 40%
7 – 13	**Moderate**	40 – 70%
14 – 19	Severe	71 – 99%
20	Profound	100%

Example: global score 11 → Moderate band. NOT 55%. Never calculate total/20 × 100.

3. IMPORTANT VERSION CONFLICT

The 14 March 2024 Gazette Appendix XIII reproduces older IPS material:

- Uses months ill in the previous 2 years.
- Has the moderate band starting at 8 (instead of 7, as in the government-modified scoring above).

This differs from the government-modified scoring version described above.

- **Do not mix versions.**
- **Do not present a score of 7 as an automatic current legal entitlement.**
- **For certification, verify the authority's applicable protocol and clarification.**

4. CURRENT CERTIFICATION CONTEXT

Key points from 14 March 2024 guidance (notification 12 March 2024)

- Permanent certification generally requires ≥ 2 years of illness and residual disability despite adequate documented treatment.
- Time-valid certificates are possible earlier, with severe/profound exceptions considered by the board.

Scale administration ≠ diagnosis ≠ certification ≠ entitlement.

Exam-ready reminders

- IDEAS is a disability scale, not PANSS/BPRS symptom severity.
- Unemployment alone is not disability.
- Illness duration is not the same as months symptomatic.
- Official appendix contains legacy text: use current main guidance for legal context.

Source: Grover et al. 2014 (government-modified validation); Gazette 2024 (section 24 and Appendix XIII), checked 14 Sep 2026.

Do not invent certainty where official versions conflict.

Indian Disability Evaluation and Assessment Scale - IDEAS. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Indian Scale for Assessment of Autism - ISAA

ISAA · Standard 40-item version

1. PURPOSE AND USE

- Clinician-rated assessment of autism-related characteristics and severity. Used in Indian disability assessment.
- Developed chiefly with children; interpret scores by age and developmental level.
- Not an intelligence or adaptive-behaviour scale; not sufficient alone to diagnose autism.
- Trained professional uses direct observation, caregiver interview across settings and testing. Typically takes about 20–30 minutes.
- **Timeframe:** current observed/reported **pattern.** The manual does not specify a fixed past-week or past-month recall window. Do not confuse the three-month test–retest interval with the rating timeframe.

SIX DOMAINS AND ITEM COUNTS

Domain	Number of items
Social relationship and reciprocity	9
Emotional responsiveness	5
Speech–language and communication	9
Behaviour patterns	7
Sensory aspects	6
Cognitive component	4
Total	**40**

2. SCORING

ITEM SCORING (1-5)

Score	Descriptor	Frequency (proportion of time behaviour is seen)
1	Rarely	Up to 20%
2	Sometimes	21–40%
3	Frequently	41–60%
4	Mostly	61–80%
5	Always	81–100%

These scores describe frequency categories, **NOT** disability percentages.

TOTAL SCORE RANGE: 40 – 200

WORKED EXAMPLE: DOMAIN SUBTOTALS

Domain	Domain score
Social relationship and reciprocity	27
Emotional responsiveness	10
Speech–language and communication	27
Behaviour patterns	21
Sensory aspects	12
Cognitive component	8
Total score	**105**

RUNNING SUM

27 + 10 = 37 → 37 + 27 = 64 →
64 + 21 = 85 → 85 + 12 = 97 → 97 + 8 = **105**

3. INTERPRETATION AND LEGAL DISTINCTION

ISAA SEVERITY BANDS (TOTAL SCORE 40-200)

Total score range	Scale band	Example
40 – 69	Below original cutoff	–
70 – 106	Mild	
107 – 153	Moderate	
154 – 200	Severe	

Example: A score of 105 falls in the mild scale band.
A score < 70 does not clinically rule out autism.

STATUTORY DISABILITY MAPPING (GAZETTE 2024) FOR DIAGNOSED AUTISM, AGE ≥ 6 YEARS

Total score range (ISAA)	Disability percentage (category)
70 – 106	40 – 59%
107 – 153	60 – 79%
> 153	≥ 80%

Example: A score of 105 maps to the **40 – 59%** band under this framework, not 105/200 × 100.

CURRENT GUIDANCE (14 MARCH 2024; NOTIFICATION 12 MARCH)

Children under 6 years with diagnosed autism receive **60 – 79%** disability, with later **reassessment.**
Do not apply the age ≥ 6 table to under 6 years.

IMPORTANT POINTS

- Diagnosis uses the specified diagnostic pathway, not ISAA alone.
- A notified medical board certifies the disability.
- Older manual disability conversion tables differ: use current guidance.

KEY LIMITATIONS

- Language, developmental level, coexisting intellectual disability and informant quality affect ratings.
- Severity score is not support need in every setting or automatic entitlement.

Never reproduce secure test items or form from the ISAA manual.

Source: National Trust ISAA manual; Gazette 2024 section 23, checked 14 September 2026.

Indian Scale for Assessment of Autism - ISAA. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Rating Scales

Question 49 • classification, quality and test standardisation

Rating scale = a structured way to turn observations or answers into scores.
Example: Rate sleep difficulty from 0 (none) to 3 (severe).
A score summarises information; it is not a diagnosis by itself.

CLASSIFICATION OF RATING SCALES

These overlap: one scale fits several branches.

RATING SCALES

WHO RATES?

- **Self-report** — Person rates themselves, e.g. Beck Depression Inventory.
- **Clinician-rated** — Trained examiner rates, e.g. Hamilton Depression Rating Scale.
- **Informant-rated** — Relative/carer reports, e.g. informant version of Strengths and Difficulties Questionnaire.

WHY USE IT?

- **Screening** — Identify who needs assessment.
- **Severity** — How much difficulty now?
- **Outcome** — Has it changed with treatment?
- **Diagnostic assessment** — Structured criteria help clinical diagnosis.

HOW BROAD?

- **Global** — Overall functioning/ severity, e.g. Clinical Global Impression.
- **Specific** — One domain, e.g. Young Mania Rating Scale.

HOW ANSWERED?

- **Yes/no** — Category.
- **Ordered choices** — None/mild/ moderate/severe.
- **Visual analogue** — Mark a position on a line.

RELIABILITY

How consistent are the scores when real ability or symptoms have not changed?

Test–retest	Same person, different occasions: similar scores if stable.
Inter-rater	Two assessors: do they agree?
Internal consistency	Items intended to measure same construct work together.
Alternate forms	Equivalent versions give comparable results.

Example: Two clinicians assessing the same stable patient give similar severity scores.

Caution: Consistency alone does not make the interpretation correct.

A USEFUL ANALOGY



A weighing scale always reads 5 kg too high: **consistent, but inaccurate.**

Useful analogy for reliability versus validity.

VALIDITY

Does evidence support what we claim the score means and how we use it?

Content	Items adequately cover the intended domain.
Construct	Scores behave as theory predicts; related measures correlate (convergent), distinct concepts remain distinguishable (discriminant).
Criterion	Agreement with an appropriate external reference, now (concurrent) or later (**predictive**).
Face	Looks relevant; appearance alone is weak evidence.

Example: A depression scale should reflect depression, not merely physical tiredness.

Caution: Reliability supports validity but cannot guarantee it.

OTHER QUALITY CRITERIA

- **Responsiveness** — Detects meaningful real change over time; not the same as screening sensitivity.
- **Sensitivity** — Among people who have the condition, how many screen positive?
- **Specificity** — Among people without it, how many screen negative?
- **Measurement error** — How much score variation is noise? Report uncertainty.
- **Feasibility** — Time, training, cost and acceptability.
- **Fairness** — Language, culture, literacy and disability should not distort interpretation.

Sensitivity/specificity apply to classification at a chosen cut-off; they are not required outputs of every scale.

STANDARDISING A NEW INTELLIGENCE TEST — six-mark sequence

1. **Define construct and blueprint** — What abilities should it sample?
2. **Write items and pilot** — Try them; check difficulty, discrimination and bias.
3. **Fix procedure** — Same instructions, timing, starting/ stopping rules and scoring.
4. **Select representative norm sample** — Relevant age, language, region, education and population.
5. **Build norms** — Compare raw scores with peers; derive age-based standard scores, often IQ mean 100, SD 15.
6. **Evaluate quality** — Reliability, validity, fairness and measurement error; revise and recheck.
7. **Publish manual** — Administration, scoring, norms, limitations; train users and update when needed.

Norms = reference scores from an appropriate comparison population.

Standardisation = consistent procedures; **norming** = constructing the comparison reference.

Development is iterative: revise and test again.

Exam structure: classification 2 marks • properties 2 marks • standardisation 6 marks.
Understand terms first; apply them to one concrete example.

Rating Scales. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Young Mania Rating Scale

YMRS • 11-item clinician-rated version (Young et al., 1978)

What is the YMRS?

- Measures manic symptom severity and treatment change.
- Mainly used in adults with mania.
- Not a diagnostic tool.
- Does not assess adaptive functioning or disability.

How is it administered?

- Trained clinician interviews the patient and observes behaviour.

Timeframe for ratings

- Patient's preceding 48 hours plus behaviour during the interview.



Domains assessed (11 items)

7 standard-weight items (score 0–4 each)

- Elevated mood
- Increased motor activity / energy
- Sexual interest
- Sleep (decreased need for sleep in context)
- Language – thought disorder
- Appearance
- Insight

4 double-weight items (score 0–8 each)

- Irritability
- Speech (rate and amount)
- Thought content
- Disruptive – aggressive behaviour

Scoring and score arithmetic

7 standard-weight items (0 – 4 each)

Maximum = 28

+

4 double-weight items (0 – 8 each)

Maximum = 32

Total YMRS range = 0 – 60

Weighting is already built into the item anchors: do not double a 0 – 8 score again.

Hypothetical example

Item group	Possible range per item	Subtotal (score)	Maximum for group
7 standard-weight items	0 – 4	**14**	28
4 double-weight items	0 – 8	**16**	32
Total YMRS score	**0 – 60**	**14 + 16 = 30**	**60**

Follow-up and treatment response

Time point	Total YMRS score
Baseline	**30**
Follow-up	**12**
Reduction	**(30 – 12) / 30 × 100 = 60%**

Meets a ≥ 50% response definition.

Interpreting the total score

- Higher total YMRS score means greater manic symptom severity.
- No single universal mild / moderate / severe classification.
- Trial entry and remission thresholds vary and must be specified.
- A total score of 30 cannot by itself diagnose mania or establish a severity category universally.

Key limitations and cautions

- Limited insight may affect what the patient reports; combine clinician observation and collateral history.
- Sedation may reduce activity without full mood recovery.
- Irritability, psychosis or agitation may have other causes (e.g. substance use, medical conditions, other psychiatric disorders).

Use YMRS as part of a comprehensive clinical assessment.

What YMRS does NOT provide

- It is not a diagnostic test.
- It does not assess adaptive functioning or disability.
- It does not provide a legal disability percentage.

KEY EXAM DISTINCTIONS

- 11 items does not mean a maximum score of 44.
- The sleep item concerns decreased need for sleep in context, not all insomnia.
- A score change does not equal syndromal remission.
- Suicide risk, violence and vulnerability need direct assessment (not captured by the total score).
- There is **no** legal disability percentage provided by the YMRS.

Young Mania Rating Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

Yale-Brown Obsessive Compulsive Scale

Y-BOCS • Original adult 10-item severity scale (1989)

Purpose and key points

- Measures the severity of obsessive–compulsive symptoms and change over time.
- Not a stand-alone diagnosis or disability assessment.
- A trained clinician conducts a semi-structured interview. Rate the average severity over the past week, including the interview day.

Symptom checklist

- A checklist helps identify the content of obsessions and compulsions (e.g. contamination, harm, symmetry).
- The checklist is **not** added to the severity total.

What the scale measures

Rates the severity of obsessive–compulsive symptoms across 10 items (5 obsessions, 5 compulsions), each assessed for the same domains over the past week.

Obsessions (5 items)

Intrusive, unwanted thought, image or urge that repeatedly enters the mind.

Five obsession severity dimensions:

- **Time occupied** — How much time it takes up
- **Interference** — Impact on functioning
- **Distress** — How upsetting it is
- **Resistance** — Efforts to resist the thought
- **Control** — Perceived control over the thought



Scoring and worked example

- Each item is rated from 0–4.
- **Obsessions subtotal: 0–20.**
- **Compulsions subtotal: 0–20.**
- **Total score: 0–40.**

Domain	Obsessions (item scores)	Compulsions (item scores)
Time occupied	3	3
Interference	2	2
Distress	3	2
Resistance	2	2
Control	2	1
Subtotal (0–20)	12	10
Total score (0-40)	22	

Compulsions (5 items)

Repetitive act or mental ritual performed to reduce distress or prevent a feared outcome.

Five compulsion severity dimensions:

- **Time occupied** — How much time it takes up
- **Interference** — Impact on functioning
- **Distress** — How upsetting it is
- **Resistance** — Efforts to resist the behaviour
- **Control** — Perceived control over the behaviour



Interpretation (Conventional teaching bands)

Total score	Severity band
0 – 7	Subclinical
8 – 15	Mild
16 – 23	Moderate
24 – 31	Severe
32 – 40	Extreme

Example: total score 22 falls in the **moderate** band.

Important considerations

- These are conventional teaching bands, not universal: adult empirical bands differ (Storch et al., 2015).
- A low total score neither establishes nor excludes a diagnosis.
- Avoidance, insight and family accommodation require additional enquiry.
- Resistance is not the same as insight or therapeutic willingness.

Limitations

- The Y-BOCS focuses on severity, not the full clinical picture.
- Avoidance, insight and family accommodation are not fully captured.
- Assess functional impairment and risk (e.g. self-harm or harm to others) clinically.
- Use alongside a comprehensive clinical assessment.

Key exam distinctions

- Count severity, not the number of obsessions or the unusualness of their themes.
- Original total is 40 (1989); the second edition total is 50 (Y-BOCS-II).
- The Children's Yale–Brown Obsessive Compulsive Scale (CY-BOCS) is a separate age-adapted instrument.
- Supplemental ratings are not added to the original 10-item total.
- Assess impairment and risk clinically.

Sources: Goodman et al. (1989). Yale–Brown Obsessive Compulsive Scale. Original scale reprint.
Storch et al. (2015). Empirically based conversion of the Y-BOCS in adults.

Yale-Brown Obsessive Compulsive Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-49.

Credits and references: retained within the illustration.

I - 50

What is structured clinical assessment? Discuss its role and limitations in psychiatry. [2+5+3]

2 + 5 + 3 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Fixed questions, fixed probes, operational scoring
3 What it fixes	Information variance and criterion variance
2 Where it is used	The named instruments, by setting
3 Limitations	Time, rigidity, culture, and what it cannot do

THE DEFINITION

Structured clinical assessment replaces the free interview with fixed questions in a fixed order, with defined probes and operational rules for scoring each response against diagnostic criteria. A fully structured instrument fixes the wording and can be given by a trained lay interviewer; a semi-structured one fixes coverage and criteria but leaves judgement in the rating, so it needs a clinician.

- **The two variances it removes.** Information variance, two clinicians eliciting different facts because they asked different questions. Criterion variance, two clinicians applying different thresholds to the same facts. Structuring the interview removes the first; operational criteria remove the second. Together they are the main source of unreliable diagnosis.
- **The instruments, by setting.** Semi-structured and clinician-rated for research diagnosis and for schedules built on a glossary. Fully structured and lay-administrable for large epidemiological surveys, which is how national and cross-national prevalence figures are generated at all. Brief structured instruments for screening in busy clinical services, and child versions for developmental work.
- **What else it buys.** A reproducible case definition for a trial, a teachable framework for a trainee, and a defensible record where the diagnosis may be examined later.
- **The limits.** Long, needing training and re-calibration. It rates presence rather than severity or function, is rigid with comorbid and subthreshold presentations, and inherits every error in the classification it encodes. Translated instruments need re-validation, not only translation.

PITFALL

Treating the schedule as the assessment. It produces a diagnosis, not a formulation. Nothing in it asks why this person became ill now, and the rapport lost to a questionnaire is the same rapport the treatment will need.

WHAT EARNS THE MARKS

- **Structured and semi-structured told apart,** by who may administer and what is fixed.
- **Information variance and criterion variance named as terms.** They are the reason the whole method exists.
- **Instruments placed by setting,** research, survey, clinic, rather than listed loose.
- **Diagnosis separated from formulation** in the limitations, which is where the last marks sit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 49 rating scales and their properties SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

Structured Interviews

Question 50 • Definitions, examples, utility and limitations

Structured clinical assessment = systematically collecting and rating information using predefined domains, questions and decision rules.
Diagnostic interviews are one component of a complete assessment.

UNSTRUCTURED

Clinician guides a flexible conversation.

- No fixed script; order and probes adapt.

Strength: Narrative, rapport and individual context.

Limitation: Important areas may be missed; interviewers may elicit different information.

SEMI-STRUCTURED

Standard core topics and criteria, flexible clarification and probing.

- Trained interviewer applies clinical judgement.

Strength: Systematic coverage with room for nuance.

Limitation: Training, time and rater judgement still matter.

Example: **SCID**.

FULLY STRUCTURED

Prescribed questions, wording, routing and scoring rules; follow manual's permitted clarification.

- Trained nonclinicians can administer some instruments.

Strength: Reproducibility and scalability.

Limitation: Less flexibility; ambiguous responses need careful handling.

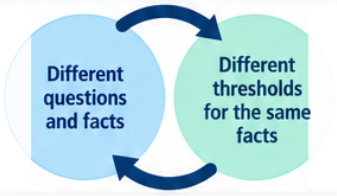
Examples: **MINI** and **CIDI**.

Structure is a continuum. A routine clinical interview can also be systematic; unstructured does not mean careless.

INFORMATION VARIANCE

Interviewers ask different questions → collect different facts.

Standard coverage reduces this.



CRITERION VARIANCE

Interviewers apply different thresholds to the same facts.

Operational criteria reduce this.

Structure reduces variation; it does not eliminate all error.

SCID
Structured Clinical Interview for DSM Disorders

DSM = Diagnostic and Statistical Manual of Mental Disorders.
Format: SEMI-STRUCTURED despite its name.
SCID-5 examples for DSM-5 diagnoses; versions cover different disorder groups and settings.
Administrator: trained interviewer with diagnostic expertise.
Best-known use: detailed clinical/research diagnostic assessment and study case definitions.
Strength: clinical probing and criterion-based decisions.
Trade-off: longer, training-intensive; duration varies by modules and complexity.
Attribution: Robert Spitzer and colleagues; later versions led by Michael First and colleagues.

MINI
Mini International Neuropsychiatric Interview

Format: brief **STRUCTURED** diagnostic interview with modules and **skip** rules.
Attribution: David Sheehan, Yves Lecrubier and collaborators.
Use: efficient assessment of major disorders in clinics and research.
Strength: relatively brief and standardised.
Trade-off: less depth; positive modules and complex presentations require clinical follow-up.
Cllassification coverage depends on version.
It is a diagnostic interview, not merely a symptom screen.

CIDI
Composite International Diagnostic Interview

Format: FULLY STRUCTURED; trained lay interviewers can administer.
Attribution: World Health Organization development; World Mental Health survey programme.
Use: large population and cross-national epidemiological studies.
Strength: scalable, comparable diagnostic data.
Trade-off: potentially lengthy; depends on recall, language adaptation and programmed diagnostic rules.
Cllassification and time windows depend on version.

One example question or approach across formats

Illustrative descriptions, not actual instrument items.

Unstructured
"Tell me what has been difficult recently."

Semi-structured
Cover required mood symptoms, then clarify the person's meaning and examples.

Fully structured
Ask prescribed symptom questions and follow the instrument's routing.

Clinical role of structured interviews (5 key points)

1. Systematic coverage of key symptoms and diagnostic criteria.
2. Identify comorbidity.
3. Create reproducible research groups and study case definitions.
4. Require training to improve reliability and validity.
5. Support clear documentation.

Limitations (3 key points)

1. Time and training.
2. Cultural/language validity.
3. Recall and reporting bias.

- Dependence on classification system.
- Limited flexibility.
- Rapport must be actively maintained.

Interview result ≠ complete formulation.

Add history, mental state examination, risk assessment, collateral information, physical/substance causes, functioning and personal context.

Memory hook: SCID: flexible clinician probing • MINI: brief structured assessment • CIDI: structured population surveys

Exam focus:
2 definition + 5 role + 3 limitations

Structured Interviews. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-50.

Credits and references: retained within the illustration.

I - 51

a) What are the 5 major theories of personality? b) Critically evaluate those major theories. c) Briefly write about any four such personality assessment tools related to those theories. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Name the five	One line each, with the theorist attached
4 Critical evaluation	One strength, one failure, per theory
3 Four tools	Each tied to the theory that generated it
1 The verdict	Which framework the evidence now favours

THEORY	CORE CLAIM	THE CRITIQUE THAT STICKS	TOOL IT GENERATED
Psychoanalytic	Personality is structure and unconscious conflict, laid down early	Retrospective, hard to falsify, weak predictive evidence	**Rorschach, Thematic Apperception Test**
Trait and dispositional	Personality is a small set of stable, measurable dimensions	Describes without explaining; underrates the situation	**16PF, NEO-PI-R, Eysenck questionnaire**
Behavioural and social learning	Personality is learned behaviour, cued and reinforced by situations	Under-explains cross-situational consistency and inner life	**Behavioural analysis, locus of control scale**
Humanistic	Personality is the self, organised around growth and self-actualisation	Optimistic, poorly operationalised, thin on psychopathology	Q-sort, self-concept measures
Cognitive and constructivist	Personality is the system of personal constructs used to anticipate events	Narrow range, limited clinical purchase	**Repertory grid**

Amber marks the four tools that answer part c. Naming a tool without the theory behind it answers half the question.

- **The evaluation, in one argument.** The five differ on where personality is located: inside, in traits, in situations, in the self, or in constructs. The trait framework has won on measurement because it alone produced replicable dimensions with normative data; the psychoanalytic framework has kept its clinical usefulness in formulation while losing the measurement argument.
- **The trap in part c.** A projective and an inventory are not interchangeable tools. Projective tools follow from the psychoanalytic claim that the important material is not available to self-report; inventories follow from the trait claim that it is.
- **Indian use.** Trait inventories need local norms before a raw profile means anything, and translated inventories need re-validation rather than translation alone.

WHAT EARNS THE MARKS

- **Five named theories with a theorist each,** before any evaluation begins.
- **One critique per theory,** written as a limitation rather than as dislike.
- **Four tools, each bolted to its parent theory.** That link is what part c is testing.
- **A stated verdict** on which framework the measurement evidence supports.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note SPRINT
Day 5, social, learning and personality psychology

Personality Theories

Major traditions, theorists, contributions and contrasts

Personality: characteristic patterns of thinking, feeling and behaving.

Read approximate dates within each panel. Parallel traditions, not a single succession.

1 HISTORICAL TYPES
Antiquity; 1920s–1940s revival.

Hippocrates & Galen: humoral temperaments; Ernst Kretschmer & William Sheldon: physique types.

- **Core idea** • Early classification of differences in temperament types.
- **Added** • Provided early attempts to classify personality difference types.
- **Changed** • Later developed into dimensional approaches.
- **Limitation** • Humours and body-type determinism are obsolete.

2 PSYCHOANALYTIC
1890s–1930s.

Sigmund Freud.

- **Core idea** • Unconscious conflict, id–ego–superego, defences, early development.
- **Added** • Dynamic motives and developmental explanation beyond surface description.
- **Changed** • Influenced later developmental, relational and humanistic ideas.
- **Limitation** • Many specific claims difficult to test.

3 NEO-FREUDIAN / PSYCHOSOCIAL
1910s–1950s.

Alfred Adler; Carl Jung; Karen Horney; Harry Stack Sullivan; Erich Fromm; Erik Erikson.

- **Adler** – inferiority, striving, social interest.
- **Jung** – archetypes, individuation.
- **Horney** – basic anxiety.
- **Sullivan** – interpersonal patterns.
- **Fromm** – freedom and society.
- **Erikson** – lifespan psychosocial stages.

Key difference Broader psychosocial and cultural perspectives.

Limitation Diverse claims with uneven evidence.

4 EGO / OBJECT RELATIONS / ATTACHMENT
1930s–1970s.

Anna Freud; Melanie Klein; Ronald Fairbairn; Donald Winnicott; John Bowlby & Mary Ainsworth.

- **Anna Freud** – ego defences.
- **Klein and Fairbairn** – internal object relations.
- **Winnicott** – caregiving and true/false self.
- **Bowlby and Ainsworth** – attachment.

Key difference Emphasis on relationships and internal working models.

Limitation Distinct traditions, not one theory; evidence differs.

5 BEHAVIOURAL
1910s–1950s.

John Watson; Burrhus Frederic Skinner.

- **Core idea** • Learned behaviour shaped by environmental cues and consequences.
- **Added** • Observable, testable environmental mechanisms.
- **Changed** • Contrasted with inferred unconscious explanations.
- **Limitation** • Strict behaviourism underrepresents internal meaning.

6 TRAIT / DISPOSITIONAL
1930s onward.

Gordon Allport; Raymond Cattell; Hans Eysenck; Lewis Goldberg; Paul Costa & Robert McCrae.

- **Allport** – individual traits.
- **Cattell** – 16 factors.
- **Eysenck** – extraversion, neuroticism, psychoticism.
- **Goldberg, Costa and McCrae** – Big Five tradition.

Key difference Five factors widely supported (Extraversion, Agreeableness, Conscientiousness, Neuroticism, Openness); psychoticism less central.

Limitation Descriptions alone do not explain all mechanisms; Eysenck's psychoticism is not a psychosis diagnosis.

7 HUMANISTIC / EXISTENTIAL
1940s–1960s.

Carl Rogers; Abraham Maslow; Rollo May.

- **Rogers** – self-concept and congruence.
- **Maslow** – needs and self-actualisation.
- **May** – meaning, choice, responsibility.

Key difference Emphasis on growth, freedom and personal experience against determinism.

Limitation • Some constructs hard to operationalise.

8 PERSONAL CONSTRUCT / COGNITIVE
1950s onward.

George Kelly; Aaron Beck.

- **Kelly** – personal constructs.
- **Beck** – schemas and beliefs.

Key difference How people make sense of the world; meaning mediates responses.

Limitation • Beliefs are only one level of explanation.

9 SOCIAL COGNITIVE / INTERACTIONIST
1950s–1990s.

Julian Rotter; Albert Bandura; Walter Mischel & Yuichi Shoda.

- **Rotter** – expectancy, reinforcement value, locus of control.
- **Bandura** – modelling, self-efficacy, reciprocal determinism.
- **Mischel and Shoda** – if-then person–situation patterns.

Key difference Integrated person, behaviour and environment.

Limitation Complex reciprocal effects hard to isolate.

10 BIOLOGICAL / TEMPERAMENT
1940s onward.

Hans Eysenck; Jeffrey Gray; Alexander Thomas & Stella Chess; Robert Cloninger.

- **Eysenck** – biological trait hypotheses.
- **Gray** – reinforcement sensitivity.
- **Thomas and Chess** – temperament and goodness of fit.
- **Cloninger** – temperament and character.

Key difference Heritable dispositions and biological systems interacting with environment.

Limitation • Heritability is not destiny; biological mappings are incomplete.

11 EVOLUTIONARY / CULTURAL
late 20th century onward.

David Buss; Hazel Markus & Shinobu Kitayama.

- **Buss** – evolutionary perspective.
- **Markus and Kitayama** – cultural self-construal.
- **Separate perspectives:**
  - **evolution:** adaptive problems and individual differences.
  - **culture:** independent/interdependent self-construal.

Key difference Adaptive and cultural contexts shape personality.

Limitation • Evolutionary claims need testing; cultures are not uniform.

12 CONTEMPORARY INTEGRATION
late 20th century onward.

Dan McAdams.

- **Core idea** • Multiple levels: dispositions + goals/beliefs + life story + context.
- **Added** • Integrates traits, cognitive, social, biological, cultural and narrative approaches.
- **Changed** • Greater synthesis across levels and methods.
- **Limitation** • No single universal replacement theory; integration needs specific testable mechanisms.

Assessment links (not one-to-one ancestry): **Trait** → Sixteen Personality Factor Questionnaire, NEO inventories, Eysenck questionnaires • **Personal constructs** → repertory grid • **Humanistic self-concept** → Q-sort • **Social learning** → locus-of-control / self-efficacy measures • **Psychodynamic formulation** → narrative/projective methods, interpreted cautiously.

Question 51 groups five: **psychoanalytic • trait • behavioural/social learning • humanistic • cognitive/constructivist.** This wider map shows their branches and later developments. **Compare each on:** what drives personaliity, how it develops, evidence, and limitations.

Personality Theories. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-51.

Credits and references: retained within the illustration.



Personality Theories Compared

A fictional trainee prepares meticulously, fears criticism, avoids speaking in meetings and feels relieved afterwards. They say: 'If I make a mistake, I am worthless.'

These observations permit questions and hypotheses—not a diagnosis.

Psychodynamic

Sigmund Freud and relational traditions

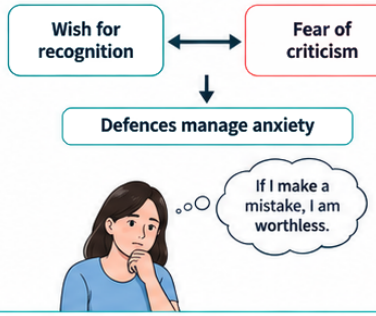
Focus: unconscious conflict, defences and recurring relationships.

Possible reading: a wish to be recognised conflicts with fear of criticism and a harsh internal standard; overpreparation may help manage anxiety.

Explore: what criticism means, patterns with authority, and feelings in the therapeutic relationship.

Clinical direction: recognise conflict and develop more flexible responses.

Limit: a coherent interpretation is not proof of an unconscious cause; do not invent childhood events.

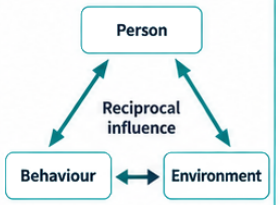


Behavioural / social-cognitive Burrhus Frederic Skinner ▪ Albert Bandura

Behavioural focus: learning history and consequences.

Meeting → silence → immediate relief → silence more likely next time

Negative reinforcement: relief strengthens avoidance; not punishment.



Social-cognitive focus: expectations, self-efficacy and observation. 'I cannot cope with a question' = low task-specific self-efficacy.

Assess: triggers, behaviour and consequences; beliefs and modelling.

Clinical direction: graded practice and new learning.

Limit: a maintenance account may not explain every origin or enduring trait.

Trait

Gordon Willard Allport ▪ Raymond Bernard Cattell ▪ Hans Jürgen Eysenck ▪ Five-factor model

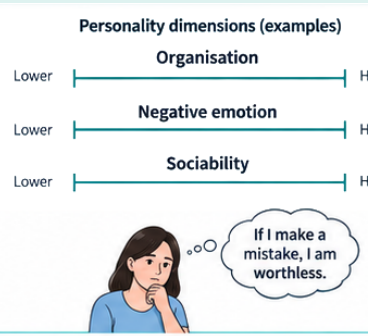
Focus: relatively enduring patterns across time and situations.

Possible profile to assess: conscientiousness and negative emotionality. No scores can be inferred from this vignette. Reserved behaviour may reflect fear rather than low extraversion.

Assess: validated inventories plus longitudinal history and information across settings.

Clinical direction: adapt formulation and communication to enduring tendencies.

Limit: naming a trait describes a pattern; it does not by itself explain why it developed.



Humanistic

Carl Ransom Rogers ▪ Abraham Harold Maslow

Focus: lived experience, self-worth, needs and growth.

Rogers: 'I must be flawless' may be a condition of worth; needing help conflicts with the self-concept.

Maslow: explore unmet safety, belonging and esteem needs alongside meaningful growth.

Assess: the person's own account, self-concept and goals; Q-sort may compare current and ideal self.

Clinical direction: empathy, acceptance and collaborative self-directed goals.

Limit: growth concepts are broad, culturally shaped and not a complete account of psychopathology.



Compare the question each asks: conflict and meaning · learning and expectations · enduring tendencies · experience and growth.

Integrate cautiously: assess biology and social context, test hypotheses, and distinguish descriptive fit from causal evidence.

Personality Theories Compared. Read across the labelled contrasts to distinguish the related concepts. Read with I-51.

Credits and references: retained within the illustration.

I - 52

a) What are the types of scales used in psychiatry from research point of view? b) Name the rating scales and questionnaires for depression and describe one rating scale in detail that you are using in your clinical practice. [4+6]

4 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Types of scales	Measurement level first, then rater, then purpose
2 Name them	Clinician-rated and self-rated depression instruments
3 One in detail	Structure, range, bands, response and remission
1 Its bias	Where the chosen scale reads high, and why

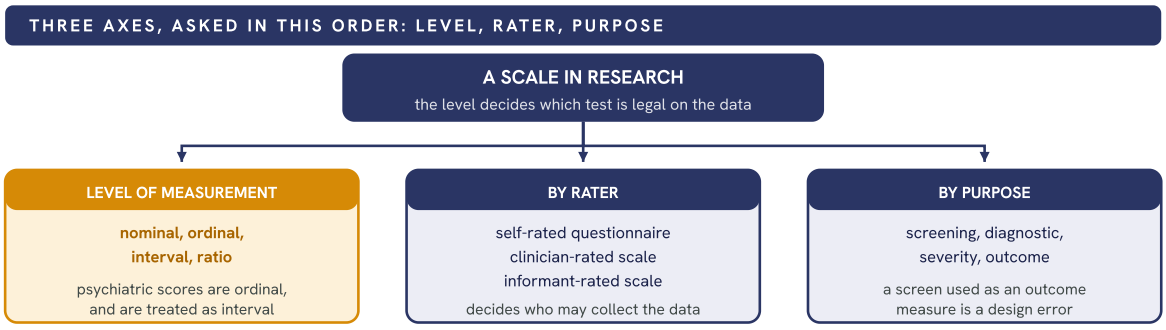


Fig 052.1 The three axes a research scale is classified on, the measurement level in amber because it decides which statistical test the data will allow.

ONE SCALE IN DETAIL: THE HAMILTON DEPRESSION RATING SCALE

Hamilton, 1960. Clinician-rated at interview over the past week, usually in its seventeen-item form: nine items score zero to four and eight zero to two, so the total runs zero to fifty-two. Bands: zero to seven none, eight to sixteen mild, seventeen to twenty-three moderate, twenty-four and above severe. In trials, response is a fall of half and remission a total of seven or below.

- **Name them.** Clinician-rated: Hamilton, Montgomery Asberg. Self-rated: Beck Depression Inventory, the nine-item Patient Health Questionnaire, Zung. Population specific: Geriatric Depression Scale, Edinburgh postnatal scale.
- **Where it reads high.** Hamilton is weighted to sleep, appetite and somatic items, so it inflates in physical illness and in the elderly. Montgomery Asberg was built less somatic and is preferred there.
- **The one rule.** A rating scale measures severity, never diagnosis. A high score is a reason to interview.

WHAT EARNS THE MARKS

- **Measurement level answered first,** with the consequence for test choice stated.
- **Both rater classes named for depression,** with instruments under each.
- **Its known bias stated.** The somatic weighting reads as practice rather than recall.

SEE ALSO Slot I - 49 classifying rating scales and standardisation Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

Hamilton Depression Rating Scale

17-item version • HAM-D17 / HDRS17 • Max Hamilton, 1960

Purpose and administration

Clinician-rated depressive symptom severity and change, mainly in adults with depression. Originally developed in depressed inpatients.

Trained clinician: interview plus observation; assess the past week. Usually 20–30 minutes.

Measures symptom severity, not adaptive functioning or legal disability; does not establish a diagnosis.

Scoring

9 items rated 0–4 + **8 items rated 0–2**

Use item-specific anchors; not all items share one range.

Maximum = (9 × 4) + (8 × 2) = 36 + 16 = 52

Total range: 0–52; higher = greater symptom severity.

Interpretation - stated convention

Zimmerman et al., 2013 bands; also used in Weave 100

Total score	Severity range
0–7	none/minimal
8–16	mild
17–23	moderate
24–52	severe

Other published band systems differ; state the convention rather than mixing thresholds.

What it covers

Mood and cognition: depressed mood, guilt and suicidal thinking
Sleep: early, middle and late insomnia
Activity: work/interests, psychomotor slowing and agitation
Anxiety: psychic and bodily symptoms
Physical/other: gastrointestinal and general somatic symptoms, sexual symptoms, health preoccupation, weight loss and insight

Psychomotor slowing = slowed speech, thought and movement.

Hypothetical worked example

Subtotal of nine 0–4 items = 18
Subtotal of eight 0–2 items = 6

Total = 18 + 6 = 24

With the stated bands: severe symptom range.

Response ≠ remission

Response: commonly ≥50% reduction from baseline.
Remission: commonly total ≤7.

Example: Baseline 24 → follow-up 6
(24 − 6) ÷ 24 × 100 = 75% reduction

Meets both stated score criteria; also assess symptoms, functioning and clinical course.

Exam distinctions and limitations

- Do not add the extra four items of a 21-item form to the HAM-D17 total.
- Somatic and insomnia weighting can confound physical illness; hypersomnia and increased appetite are poorly captured.
- Training and consistent interviews matter for inter-rater reliability.
- A low total never replaces an individual suicide-risk assessment.
- Symptom score ≠ diagnosis or disability entitlement.

Hamilton Depression Rating Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-52.

Credits and references: retained within the illustration.

Montgomery-Åsberg Depression Rating Scale

MADRS • Original 10-item clinician-rated version

Purpose and key features

Purpose
- Measures depressive symptom severity and change during treatment in adults.
- Not a diagnostic or disability scale.

Administration
- Trained clinician uses a structured interview and observation.

Time window
- Past week for this sheet.
- Original instructions allow other documented intervals; keep interval consistent.

Domains (10 items, grouped)
- Apparent and reported sadness (2 items)
- Inner tension (1 item)
- Sleep and appetite reduction (2 items)
- Concentration difficulty and lassitude (difficulty initiating activity) (2 items)
- Inability to feel (1 item)
- Pessimistic and suicidal thoughts (2 items)

Scoring

10 items × 0–6 = total 0–60

Higher scores = greater severity

Rating anchors (per item)

0	2	4	6
Absent	Mild	Marked	Extreme

Intermediate ratings 1, 3 and 5 are used when the severity is between anchors.

Worked example (hypothetical)

Item group	Number of items	Score per item (in this example)	Subtotal
Five items	5	3	15
Four items	4	2	8
One item	1	1	1
Total	**10**	–	**24**

Total = 15 + 8 + 1 = 24 (out of 60)

Interpretation and change over time

Common published severity bands (Snaith et al., 1986)

Total score	Severity (convention)
0 – 6	Absent / minimal
7 – 19	Mild
20 – 34	Moderate
35 – 60	Severe

These bands are conventions, not a diagnosis.

Follow-up example (hypothetical)

Baseline total score	24
Follow-up total score	10
Reduction	(24 – 10) / 24 × 100 = 58.3%

Meets a ≥ 50% response definition.

Response describes change in symptom severity. Remission requires a separately specified protocol threshold.

Key exam points and distinctions

- More focused on change in core depressive symptoms than the Hamilton scale, but still contains sleep/appetite items.
- Interview skill and baseline symptom severity matter for reliable ratings.
- Medical illness and medication can affect ratings.
- A low total does not exclude significant suicide risk: assess risk directly.
- Do not confuse response, remission and diagnosis.

Montgomery & Åsberg 1979; Snaith et al.1986.

Montgomery-Åsberg Depression Rating Scale. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-52.

Credits and references: retained within the illustration.

I - 53

Projective tests. [10]

10 PSYCHOMETRY AND ASSESSMENT RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The hypothesis	Ambiguity forces the answer to come from within
5	The catalogue	Test, material, task, what is read from it
2	Clinical use	The referral questions they actually answer
1	Standing	One honest sentence on the evidence

TEST	MATERIAL	THE TASK	WHAT IS READ FROM IT
Rorschach	Ten symmetrical inkblots	Say what it might be, then show where	Perceptual organisation, thought process, reality testing
Thematic Apperception Test	Ambiguous interpersonal scenes	Tell a story with a past and an ending	Needs, external press, object relations
Children's Apperception Test	Animal or human scenes, ages three to ten	Tell a story	Childhood conflict around feeding, rivalry, control
Sentence completion	Incomplete written stems	Finish each stem with the first response	Attitudes to defined areas of life
Draw-a-Person	Blank paper and a pencil	Draw a person, then one of the other sex	Body image and self concept

Amber marks the two an examiner will follow up on. The materials are copyrighted and are described in words only.

- **The projective hypothesis.** The stimulus is unstructured and the scoring key is hidden, so the person has nothing external to lean on and the structure they impose is taken to reveal needs, conflicts and defences. Sampling is broad band, the whole personality rather than one trait.
- **What they are actually used for.** Where self-report is guarded or defended, where object-relational and motivational material is the referral question, and as one element of a battery in personality assessment. Never as a stand-alone diagnostic instrument.
- **Graded transparency.** Sentence completion is the most structured and most transparent, so the easiest to fake; the inkblot is the least. Word association, the oldest of the group, reads complexes from blocking and long latency.

PITFALL

Calling a projective test objective because it has a scoring system. The two families measure personality by opposite logic: ambiguous stimulus with inferential scoring against fixed-choice items with an empirical key. The reliability weakness follows from that logic and better scoring has not removed it.

WHAT EARNS THE MARKS

- **The hypothesis stated before any test is named.** A bare list is a mid-band answer.
- **Material and task given separately for each test.** That pairing is what the grid is for.
- **The referral question each one answers,** rather than a claim that they diagnose.
- **One honest line on the contested psychometrics.**

SEE ALSO Slot I - 48 projective tests classified and the Rorschach in detail Slot I - 45 the Thematic Apperception Test

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

Neuropsychological assessment overview

A structured evaluation of brain–behaviour relationships: history, standardised cognitive tasks, observation and everyday functioning.

Referral question → History and daily function → Select tests → Check validity and norms → Interpret profile → Recommendations

Domains and illustrative tasks

- **Attention and working memory** — hold and manipulate information; digit span
- **Processing speed** — speed of simple mental operations; timed symbol coding
- **Learning and memory** — acquisition, delayed recall and recognition; word-list or figure learning
- **Language** — naming, comprehension and verbal expression
- **Visuospatial and constructional ability** — judge spatial relationships and copy designs
- **Executive functions** — inhibit, shift, plan and monitor; rule-switching and planning tasks

Motor/sensory function and social cognition: add when relevant.

Choose tests for the person

Fixed battery
- standard set
- broad, comparable profile
- lengthy and not exhaustive.

Flexible battery
- tailored to the referral
- efficient
- guard against missing unexpected deficits.

Check age, education and literacy; preferred language and culture; hearing, vision and motor abilities; test reliability, validity and suitable norms.

Use trained examiners, standard instructions and documented adaptations.

Screening identifies concern; comprehensive assessment characterises strengths and weaknesses.

Interpret a coherent pattern

Hypothetical profile



Adult with treated depression and cognitive complaints. Describe slowed performance with preserved learning/retention; review sleep, mood, medication and other causes. The profile alone does not identify the cause.

Compare appropriate norms, prior ability, error types and everyday function.

Check performance validity: are scores interpretable?

Translate findings into rehabilitation and practical support.

Exam distinctions and limitations

- Normal screen ≠ no selective deficit
- One low score ≠ diagnosis or precise lesion
- Tasks involve several cognitive processes
- Mood, pain, fatigue and language can alter performance
- Repeat testing: consider practice effects and measurement error
- Scores inform, but do not alone determine, capacity or disability certification

Neuropsychological assessment overview. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychometry and assessment.

Credits and references: retained within the illustration.

ADPMR Disability Assessment

ADPMR • Original five-domain version, Nizamie et al., 2005

Historical publication wording; current clinical term is intellectual disability.

1. PURPOSE AND ADMINISTRATION

Purpose

- Trained-observer rating of disability and age-appropriate everyday performance in people already diagnosed with intellectual disability, including associated multiple disabilities.
- Not an intelligence test or a stand-alone diagnosis.
- Original field sample: 51 children; mean age 14 years; not universal adult norms.

Administration

- By trained rehabilitation/mental-health personnel, special educators, social workers or nurses.
- Based on direct observation and caregiver interview, with teacher reports and records.
- No standard test kit required.

Timeframe

- Rates current, age-appropriate functioning.
- No fixed retrospective window specified.
- A four-week retest in the validation was a study interval, not a rating timeframe.

2. THE FIVE DOMAINS (ONE ANCHORED RATING EACH)

1. **Perceptual–motor**
   Gross and fine coordination (e.g. movement, manipulation).
2. **Self-care**
   Daily personal care and safety (e.g. feeding, dressing, hygiene, safety).
3. **Communication and socialization**
   Expressive and receptive communication, interpersonal interaction and social behaviour.
4. **Academic**
   Formal and non-formal learning (e.g. reading, writing, numeracy, functional academics).
5. **Occupational**
   Domestic, pre-vocational and vocational functioning (e.g. simple work skills, productive activities).

3. SCORING AND WORKED EXAMPLE

Domain scoring (per domain)

0 – 4, where:

- 0 = none
- 1 = mild
- 2 = moderate
- 3 = severe
- 4 = profound

Total score: 0 – 20.
The manual permits selected intermediate scores such as 1.5, 2,5, 3,5; apply its instructions rather than invent anchors.

Worked hypothetical example (illustrative only)

Domain	Score
Perceptual–motor	1
Self-care	2
Communication and socialization	2
Academic	3
Occupational	3
Total (1 + 2 + 2 + 3 + 3)	**11**

Original integer-score bands (Nizamie et al., 2005)

Total score	Level of disability (original scale scheme)
0	None
1 – 7	Mild
8 – 13	**Moderate**
14 – 19	Severe
20	Profound

- **Example: 11 = Moderate (in the original scale scheme).**
- Original publication places ≥ 8 in its ≥ 40% category.
- This is not a current statutory entitlement rule.

4. LIMITATIONS (KEEP IN MIND)

- Small initial validation sample.
- Age, culture and opportunity affect performance.
- Coexisting sensory or motor problems can significantly influence ratings.
- A total score hides support needs in individual domains.

5. CLINICAL INSTRUMENT VERSUS LEGAL CERTIFICATION

- Indian guidance dated 14 March 2024 (notification 12 March) uses a separate intellectual-disability assessment pathway with Vineland Social Maturity Scale-based disability calculation.
- ADPMR is not a substitute for that notified pathway.
- Diagnosis, disability assessment and certification are distinct; only the notified authority certifies.
- ADPMR has 5 domains; IDEAS has 4 functional domains plus duration weighting.
- Never calculate disability as 100 – intelligence quotient.

This is a clinical instrument for assessment. It does not by itself determine legal entitlement.

Source: Nizamie et al. (2005), Part I: The ADPMR Scale. Indian Journal of Social Psychiatry, 21(3–4), 41–49. | Gazette of India, 2024, Section 21 (notification dated 12 March 2024; guidance dated 14 March 2024).

ADPMR Disability Assessment. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Psychometry and assessment.

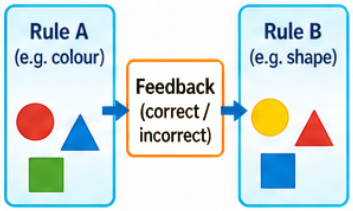
Credits and references: retained within the illustration.

Executive-function tests

Executive functions organise goal-directed behaviour: initiation, inhibition, flexibility, planning and monitoring.

Wisconsin Card Sorting Test

Sort cards using feedback; infer the rule and adapt when it changes.



MAIN PROCESS

Assesses concept formation and cognitive flexibility (set-shifting).

WHAT TO RECORD

Record categories achieved and perseverative errors.

INTERPRETATION CAVEAT

Perseveration = persisting with an old response despite changed feedback.
Poor performance is not specific to a frontal lesion.

Stroop Test

Name ink colour while suppressing automatic word reading.



MAIN PROCESS

Assesses response inhibition and interference control.

WHAT TO RECORD

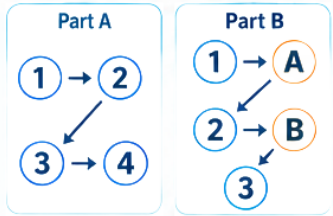
Record time/correct responses and errors using that version's manual.

INTERPRETATION CAVEAT

Reading ability, language, colour vision and baseline speed matter.

Trail Making Test

Part A: connect numbers in order.
Part B: alternate numbers and letters.



MAIN PROCESS

A: visual search, attention and processing speed.
B: adds switching and working-memory demands.

WHAT TO RECORD

Record completion time and errors; longer time generally indicates poorer performance.

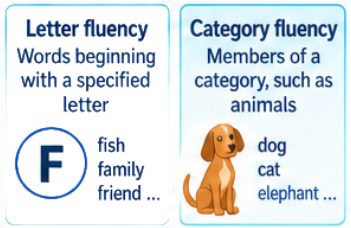
INTERPRETATION CAVEAT

Example: A = 35 s, B = 90 s; B – A = 55 s.

This reduces some speed influence but is not a pure executive score.

Verbal Fluency

Generate words under a rule within a time limit, commonly 60 seconds.



MAIN PROCESS

Assesses initiation, strategic search and monitoring; category fluency also draws strongly on semantic knowledge.

WHAT TO RECORD

Record valid unique words, repetitions and rule violations. Consider language, education and culture.

INTERPRETATION CAVEAT

Language, education and cultural background affect performance.

Planning Tasks

Tower of London / Tower of Hanoi: move objects from a starting arrangement to a goal while following rules.



MAIN PROCESS

Assesses planning, sequencing, working memory and inhibition.

WHAT TO RECORD

Record excess moves, rule violations, planning time and solution time; scores vary by task/version.

INTERPRETATION CAVEAT

Motor ability and understanding of rules affect results.

Exam distinctions

- Switching ≠ inhibition ≠ planning
- All tasks involve multiple processes
- A normal Mini-Mental State Examination can miss executive dysfunction
- Use age-, education- and language-appropriate norms; never diagnose from one score.

Executive-function tests. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychometry and assessment.

Credits and references: retained within the illustration.

Memory assessment

1. Stages of memory and the role of attention

Attend — Select and focus on information → **Encode and learn** — Process and form a memory trace → **Retain over a delay** — Maintain information over time → **Retrieve** — Access stored information → **Recall:** produce information / **Recognition:** identify previously learned information

Poor attention at input can look like poor memory.

2. Two independent comparisons

	Immediate: after presentation	**Delayed:** after a defined interval
Verbal: words, stories, paired associates	Immediate verbal memory	Delayed verbal memory
Visual: figures, designs, spatial locations	Immediate visual memory	Delayed visual memory

Immediate and delayed tasks are **not interchangeable; specify the test and delay.**
Visual tasks can still be **verbally mediated.**

3. What to examine

- **Learning curve across trials:** improvement with repetition.
- **Immediate recall:** amount recalled soon after presentation.
- **Delayed free recall:** amount recalled after a defined interval.
- **Benefit from cues:** improvement with semantic or category cues.
- **Recognition hits AND false positives:** correct identifications and incorrect endorsements.
- **Intrusions and repetitions:** recall of non-presented material and repeated responses.

- **Intrusion:** recall of material not presented.
- **False positive:** endorsing a new item as previously seen.
- **Memory** scores also depend on attention, language, executive strategy, vision and construction.

4. Common instruments

Test	What it assesses (key features)
Wechsler Memory Scale, Fourth Edition	Auditory/visual memory, visual working memory, immediate/delayed memory indices.
Rey Auditory Verbal Learning Test	Repeated word-list learning, interference, delayed recall and recognition.
California Verbal Learning Test	Verbal learning with error and strategy analysis.
Hopkins Verbal Learning Test–Revised	Brief verbal learning and delayed memory.
Rey–Osterrieth Complex Figure Test	Copy organisation plus visual recall.
Postgraduate Institute Memory Scale	Indian battery sampling several memory-related abilities.

Versions, administration and norms differ; use the correct manual.

5. Interpret patterns cautiously

Weak acquisition: inspect attention, comprehension and encoding.

Poor free recall with cue/recognition benefit may suggest retrieval difficulty.

Poor delayed recall AND recognition may suggest impaired encoding/storage, but is not disease-specific.

For a complex figure, poor copying can limit later recall.

No single pattern proves depression, dementia or **a lesion.**

6. Hypothetical 8-word task - not a standard test

Learning trials: 3 → 5 → 7 words

Delayed recall: 6 words

Recognition: 7 of 8 targets identified, but 3 of 8 distractors wrongly endorsed

Calculation:
Hit rate = 7/8 = 87.5%
False-positive rate = 3/8 = 37.5%

Conclusion:
Learning improves; recognition is not simply '7/8 normal' because false positives matter. Compare with appropriate norms; these numbers have no diagnostic cut-off.

Exam essentials: **memory ≠ intelligence; recall ≠ recognition; working memory ≠ long-term episodic memory.**
Account for sleep, mood, medications, language, effort and practice effects.

Memory assessment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychometry and assessment.

Credits and references: retained within the illustration.

Neuropsychological batteries and Indian adaptations

A battery combines tests to sample a cognitive profile; the correct version and normative reference group determine interpretation.

Halstead-Reitan Neuropsychological Test Battery



- Classic fixed battery.
- Samples abstraction, attention, sensory–motor and perceptual functions.
- Named components: Category Test and Tactual Performance Test.
- **Impairment Index:** proportion of designated tests in the impaired range; 0 to 1, higher = greater impairment.
- Historical brain-dysfunction approach; does not prove a precise lesion.

Luria-Nebraska Neuropsychological Battery



- Standardised battery based on Alexander Luria's functional-systems approach.
- Samples motor, sensory, language, academic and memory abilities.
- Produces norm-referenced clinical-scale profiles.
- The standardised battery is distinct from Luria's original qualitative method.

NIMHANS Neuropsychology Battery



- National Institute of Mental Health and Neurosciences.
- Rao, Subbakrishna and Gopukumar, 2004 adult manual.
- Indian normative data across attention, speed, working memory, executive abilities and learning/memory.
- Select age- and education-appropriate normative tables.
- Child and elderly batteries are separate versions.

AIIMS Comprehensive Neuropsychological Battery in Hindi



- All India Institute of Medical Sciences.
- Hindi battery applying Luria–Nebraska principles; profiles motor, sensory, language, reading/writing, arithmetic, memory and intellectual processes.
- Hindi administration does not make norms suitable for every Indian language group.

PGI Battery of Brain Dysfunction



- Postgraduate Institute, Chandigarh.
- Combines five instruments: PGI Memory Scale; Bhatia Short Battery; Verbal Adult Intelligence Scale; Nahor–Benson Test; Bender Visual-Motor Gestalt Test.
- Broad dysfunction profile.
- The PGI Memory Scale can also be used separately; it is not the full battery.

Adaptation is more than translation

Language and content → **Standard administration** → **Local validation** → **Appropriate norms**

- Check age, education, literacy, language, region and normative sample date.
- Nonverbal does not mean culture-free.
- Document sensory/motor limitations and deviations from the manual.

Example: A Kannada-speaking adult with limited schooling: choose validated language-appropriate tasks and suitable norms; do not apply Hindi or English norms merely because the test was developed in India.

Exam errors to avoid

- Battery ≠ single screen
- Memory scale ≠ intelligence test
- Indian norms ≠ universal Indian norms
- A score supports clinical formulation; it does not alone establish diagnosis or legal disability certification.

Neuropsychological batteries and Indian adaptations. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychometry and assessment.

Credits and references: retained within the illustration.

Adaptive functioning and developmental assessment

Adaptive functioning: everyday performance in context

Conceptual
language, literacy, numbers, time and money



Social
relationships, social judgement, understanding rules and vulnerability



Practical
self-care, health, travel, safety and work routines



Judge skills relative to age, sociocultural expectations, opportunities and support.

Three different questions

Intelligence assessment:
how does the person reason and solve problems under standard conditions?



Adaptive assessment:
what does the person usually do in daily life?



Developmental assessment:
which abilities and milestones has the young child acquired across domains?



History + Direct assessment + Caregiver/teacher information → **Integrated formulation**

Main instruments

Instrument	Role and method	Output or key distinction
Vineland Adaptive Behavior Scales, Third Edition	caregiver interview or parent/caregiver and teacher forms; adaptive functioning	Adaptive Behavior Composite uses Communication, Daily Living Skills and Socialization. Motor skills are separate, not part of this composite.
Vineland Social Maturity Scale, Indian adaptation	informant-based social maturity	social age and Social Quotient; distinct from Vineland-3.
Developmental Screening Test, Bharat Raj	Indian developmental screen	developmental age and Developmental Quotient; identifies need for further assessment.
Denver II	developmental screen, birth to 6 years	personal-social, fine motor-adaptive, language and gross motor; screen, not intelligence quotient.
Bayley Scales of Infant and Toddler Development, Fourth Edition	comprehensive assessment, 16 days to 42 months	cognitive, language and motor direct assessment; social-emotional/adaptive caregiver information; norm-referenced scores.
Gesell Developmental Schedules	milestone-based developmental evaluation	interpret the specific edition and its norms.
Seguin Form Board	timed visuomotor performance task	limited estimate of nonverbal performance; not a complete adaptive or intelligence assessment.

Illustrative age-ratio calculations

Social Quotient = social age ÷ chronological age × 100.
Example: 3 years ÷ 5 years × 100 = 60.

Developmental Quotient = developmental age ÷ chronological age × 100.
Example: 18 months ÷ 24 months × 100 = 75.

These are age ratios, not disability percentages. Bayley-4 and Vineland-3 standard scores are not calculated by these formulas.

Key points for examination and clinical practice



Intellectual disability requires impaired intellectual AND adaptive functioning with developmental onset.



Clinical severity emphasises adaptive functioning.



A screening result alone does not diagnose intellectual disability.



Consider hearing, vision, language, motor ability, educational opportunity and informant bias.



Clinical assessment and statutory certification follow different rules.

Adaptive functioning and developmental assessment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychometry and assessment.

Credits and references: retained within the illustration.

AREA 06 OF 12 · P1-A04

Genetics in psychiatry

9	9.0%	I-54	I-62
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 7

REVISION ORDER

The two mapping slots together, they are the same answer asked twice, and the association-study slot sits directly on top of them. Twin and adoption designs next, as a pair. The schizophrenia slot after that: it is the anchor here and it reuses the family-risk ladder. Epigenetics, the gene-environment slot and the genetic-correlation slot are independent and each is short.

WHAT IS IN THIS AREA

The methods in five slots: twin studies with the bipolar findings, adoption designs with schizophrenia, the gene-mapping strategies with their strengths and limits, gene mapping set against linkage and association, and the genome-wide association studies. Then the genetics of schizophrenia, epigenetics, gene and environment in neuropsychiatric disease, and the slot that runs genetic correlation and pleiotropy across substance use and psychiatric disorders.

I - 54

Genetics of Schizophrenia. [10]

10 GENETICS IN PSYCHIATRY KNRUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 The architecture	Highly heritable and highly polygenic, said in one line
3 Family, twin, adoption	The risk gradient, and what each design controls for
3 Molecular findings	Common variants, then the rare ones with big effects
2 The models	Two-hit and neurodevelopmental, and where they meet

THE LINE WORTH WRITING FIRST

Schizophrenia is among the most heritable of psychiatric disorders and among the most polygenic. Twin studies put heritability at about eighty per cent. Thousands of common variants of very small effect carry most of that, and a small number of rare copy number variants carry large effects in a few families.

RELATIONSHIP TO AN AFFECTED PERSON	GENES SHARED	APPROXIMATE LIFETIME RISK
No relation, general population	Baseline	About 1 in 100
Sibling	One half	About 9 in 100
Child of one affected parent	One half	About 13 in 100
Dizygotic co-twin	One half	About 17 in 100
Monozygotic co-twin	Essentially all	About 48 in 100

The pooled family study figures. The last two rows are the argument: both twin types share a womb and a household, only the genes differ, and the risk trebles. Note also that a monozygotic co-twin is far from certain to be affected, which is where environment enters.

- **What molecular work added.** The 2014 consortium study named 108 genome-wide significant loci, and later analyses several hundred. The strongest signal sits in the major histocompatibility complex on chromosome 6 and is driven by complement C4, which tags synaptic pruning. Rare copy number variants, the 22q11.2 deletion above all, carry far larger individual risk.
- **The two models to close with.** Two-hit: genetic vulnerability is the first hit, an environmental insult the second. Neurodevelopmental: early insults stay latent until adolescent synaptic pruning unmasks them. The C4 finding is where the two models meet.

WHAT EARNS THE MARKS

- **The monozygotic against dizygotic comparison stated as an argument,** not offered as two loose numbers.
- **Polygenic architecture named before any single gene.** A candidate-gene answer reads as dated.
- **Complement C4 tied to pruning.** It is the one molecular finding with a mechanism attached.

SEE ALSO Slot I - 58 twin studies in bipolar affective disorder Slot I - 60 adoption study designs SPRINT Day 3, Psychiatric Genetics

Genetics in Bipolar and Schizophrenia

Family risk • twins • adoption • shared and distinct genetic findings

Both are highly heritable, polygenic and multifactorial. Heritability describes variation in a population—not an individual's probability of illness.

Evidence	Schizophrenia	Bipolar disorder
One affected parent	7.0%	4.4%
Two affected parents (same disorder)	27.3%	24.9%
Parent figures: same-disorder cumulative incidence by age 52 in a Danish register cohort (Irving Gottesman and colleagues, 2010). Broader diagnoses give higher estimates; these are not universal lifetime risks.		
Probandwise twin concordance: monozygotic	33% — Danish register study, 2018	43% — Finnish bipolar I study, 2004
Probandwise twin concordance: dizygotic	7% — same Danish study	6% — same Finnish study
Twin estimates are study-specific, from different cohorts—not a head-to-head comparison. The bipolar sample was small, with wide uncertainty.		

Monozygotic = identical twins, nearly all inherited variants shared.
Dizygotic = fraternal twins, about half shared on average.
Probandwise = given an affected twin, how often is the co-twin affected?

Complete ascertainment: probandwise = 2C/(2C + D); pairwise = C/(C + D).
C = pairs with both affected; D = pairs with only one affected.
Example: C=10, D=20 → probandwise 50%; pairwise 33%.
Interpretation: identical → fraternal supports genetic influence; concordance below 100% shows genes are not destiny.
Equal-environments assumption concerns exposures relevant to illness.

ADOPTION - SCHIZOPHRENIA

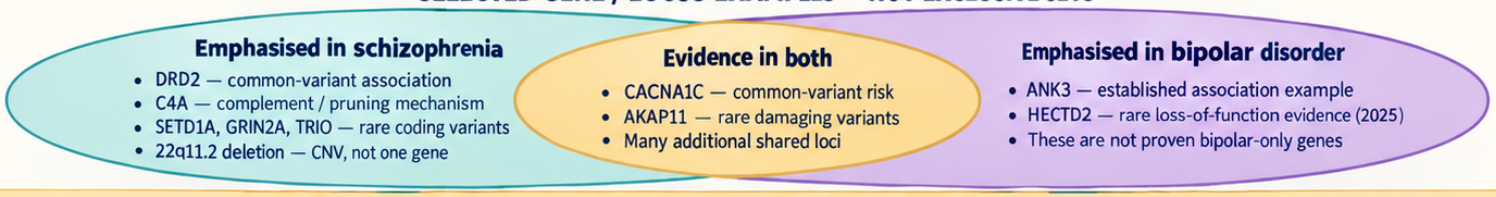
- **Seymour Kety:** schizophrenia-spectrum illness concentrated in biological relatives, supporting inherited liability.
- **Pekka Tienari:** adverse adoptive-family functioning predicted spectrum illness particularly in genetically high-risk adoptees —evidence of gene–environment interplay.

ADOPTION - BIPOLAR DISORDER

- **Swedish extended adoption study (2020):** transmission largely reflected genetic effects; modest rearing effects remained possible.
- **Shared inherited liability** also linked bipolar disorder with schizophrenia and depression.

Adoption separates biological relatedness from rearing, imperfectly: prenatal exposures, selective placement and early shared environment remain limitations.

SELECTED GENE / LOCUS EXAMPLES — NOT EXCLUSIVE SETS



No defensible complete list of exclusive genes: findings depend on variant type, sample size and phenotype. **Association is not causation; absence of evidence is not exclusivity.**

Inherited vulnerability overlaps; diagnoses are not inherited as fixed packages.

Sources: Gottesman 2010; Hilker 2018; Kieseppä 2004; Kety adoption studies; Tienari 2004; Swedish adoption study 2020; *CACNA1C* 2010; *AKAP11* 2022; *HECTD2* 2025.

Medical education, not individual genetic counselling.

Genetics in Bipolar and Schizophrenia. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-54.

Credits and references: retained within the illustration.

I - 55

Explain the genetic and environmental factors in neuropsychiatric diseases. [5+5]

5 + 5 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The genetic side**	Complex and polygenic is the rule; name the exceptions
2	**Heritability, read correctly**	What the number is and is not about
3	**The environmental side**	Ordered by timing, prenatal to adult
2	**How the two meet**	Interaction, correlation, epigenetic mediation

CONDITION	GENETIC SIDE	ENVIRONMENTAL SIDE	WHAT THE PAIR SHOWS
Huntington disease	One dominant gene, a CAG repeat	Almost none	**Genes can be sufficient**
Schizophrenia	High heritability, polygenic	Obstetric events, cannabis, urban birth, migration	Neither side alone is sufficient
Depression	Moderate heritability	Childhood adversity, loss events, chronic illness	Environment carries more weight
Alcohol use disorder	Moderate heritability	Availability, price, peer group	**Policy can move the phenotype**

The amber cells are the two ends of the spectrum. Everything examined in psychiatry sits between a gene that is sufficient and an exposure that can be legislated.

- **Environment, ordered by timing.** Prenatal: maternal infection, famine, hypoxia, valproate. Perinatal: obstetric complications. Childhood: abuse, neglect, separation, urban upbringing, migration. Later: cannabis, stimulants, alcohol, head injury, social defeat.
- **Three ways genes and environment meet.** Interaction, where an exposure harms only the vulnerable. Correlation, where genes shape which exposures a person meets, passively, evocatively or by active choice. Mediation, where the exposure leaves an epigenetic mark on expression.

PITFALL

High heritability is not fixity. Heritability describes how much of the variance in one population at one time is genetic; it says nothing about how modifiable an individual is. Phenylketonuria is fully genetic and fully preventable by diet, and that one example settles the point.

WHAT EARNS THE MARKS

- **Complex inheritance named as the rule,** with Mendelian conditions given as the exception rather than the model.
- **Exposures ordered by timing,** which shows a developmental frame rather than a memorised list.
- **All three modes of interplay,** not interaction alone.
- **The heritability caveat.** It is the sentence that separates a top answer here.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 59 epigenetics in psychiatry **SPRINT** Day 3, Psychiatric Genetics

I - 56

What are Genome Wide Association Studies? Describe their contribution to understanding etiology of psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Hypothesis-free, common variants, cases against controls
3 How it is done	The four steps, and the threshold that guards them
3 What it has contributed	Architecture, shared risk, and one drug-target lead
2 What it cannot do	The honest limits, stated as limits

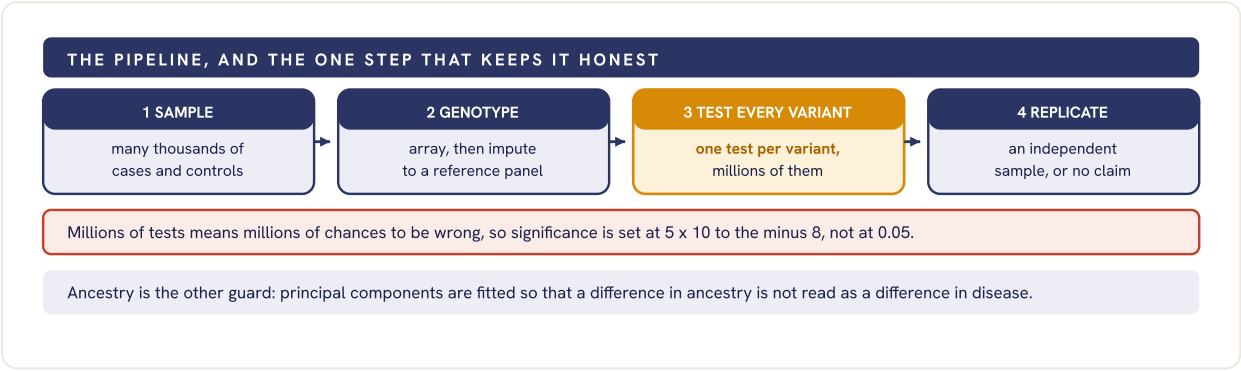


Fig 056.1 The four steps, with the two safeguards that make the output interpretable printed underneath rather than buried inside the method.

- **What it has contributed.** It settled the architecture: psychiatric disorders are highly polygenic, built from many common variants of tiny effect, not from a few broken genes. It showed substantial shared genetic risk across schizophrenia, bipolar disorder and depression. It produced polygenic risk scores, useful for research. In schizophrenia it delivered the complement C4 lead and pointed at calcium channel genes.
- **What it cannot do.** Each hit explains almost nothing on its own, and together they still leave heritability unaccounted for. The signal is a region, not a gene, because of linkage disequilibrium. Rare variants and copy number variants are invisible to it. Most samples are of European ancestry, so risk scores transfer poorly. Nothing here is a clinical test.

WHAT EARNS THE MARKS

- **Hypothesis-free stated explicitly,** and contrasted with the candidate-gene era it replaced.
- **The genome-wide threshold given with its reason.** Multiple testing, not convention.
- **Contribution framed as architecture, not as a gene list.** Polygenicity and cross-disorder overlap are the findings.
- **Limits named as limits,** including the ancestry bias, which is the one most candidates miss.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 62 genetic correlation and pleiotropy

SPRINT Day 3, Psychiatric Genetics

Genome-wide association studies

Genome-wide association study (GWAS): tests genetic variants across the genome for association with a trait.
It is a study design, not a single laboratory platform.

1 RECRUIT

Large, well-defined schizophrenia case group and appropriately selected controls; informed consent; comparable phenotyping.



Example is hypothetical, not study data.

2 MEASURE AND CHECK

Genotype or sequence DNA (deoxyribonucleic acid); check sample and variant quality, relatedness and batch effects. Imputation estimates untyped variants using reference data.



Single-nucleotide polymorphism (SNP) = common variation at one DNA base.

3 COMPARE EACH VARIANT

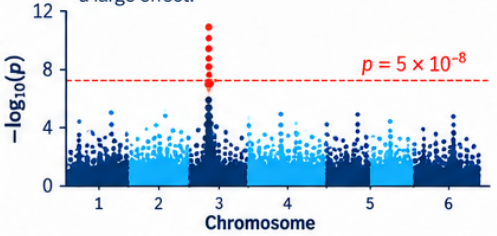
Fit association models adjusting for ancestry and relevant covariates; report effect estimate, confidence interval and p-value.

Illustrative allele A frequency	
Cases (schizophrenia)	Controls (no schizophrenia)
30%	27%

Frequency difference alone is not an adjusted finding.

4 CONTROL MULTIPLE TESTING

Conventional common-variant threshold: $p < 5 \times 10^{-8}$. Many tests demand stringent correction; a tiny p-value does not mean a large effect.



Each dot represents one tested variant; height is evidence, not risk.
Schematic, not allele A study results.

REPLICATE AND INTERPRET

Replicate in an independent sample; assess consistency and heterogeneity.

Risk locus = associated genomic region, not automatically the causal variant or nearest gene.



Linkage disequilibrium: alleles occur together more often than expected.

Fine-mapping and functional studies test explanations; association alone is not causation.

LIMITATIONS AND CONFOUNDING

Population stratification: ancestry differences associated with both allele frequencies and case status can create spurious association. Ancestry principal components (summaries of genetic variation) and appropriate models help address this.

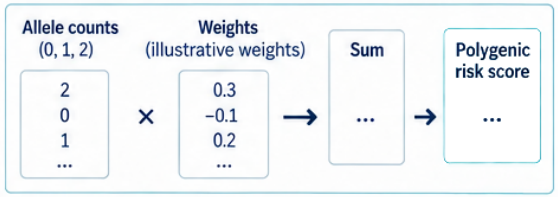


Schematic ancestry imbalance

Small effects require large samples. Phenotype heterogeneity, selection bias and incomplete variant coverage matter. Common-variant studies do not capture all rare or structural variation.

POLYGENIC RISK SCORE (PRS)

Weighted sum of many allele dosages using GWAS effect estimates; evaluate in an independent sample. It may include variants below genome-wide significance.



Score is not an absolute probability, diagnosis or destiny. Predictive accuracy and calibration depend on ancestry, population and context. Not a routine diagnostic test for common psychiatric disorders.

Exam distinctions: **Risk** = probability p. **Odds** = $p/(1-p)$. **Odds ratio** compares odds, not risks. **Significance ≠ effect size ≠ clinical utility.** **PRS prediction ≠ heritability.**

Weave Sprint; National Human Genome Research Institute; International Society of Psychiatric Genetics. Source details in notes.

Genome-wide association studies. Use the visual organisation to recall the key distinctions and structure your answer.
Read with I-56.

Credits and references: retained within the illustration.

I - 57

What are the different gene mapping strategies? Write down the basic idea, strength and limitations of the strategies. [3+2+2+3]

3 + 2 + 2 + 3 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The strategies	Name the axis, then the four families of method
2 Basic idea of each	One sentence per strategy, no more
2 Strengths	What each one is uniquely good at finding
3 Limitations	The failure mode of each, and the one they share

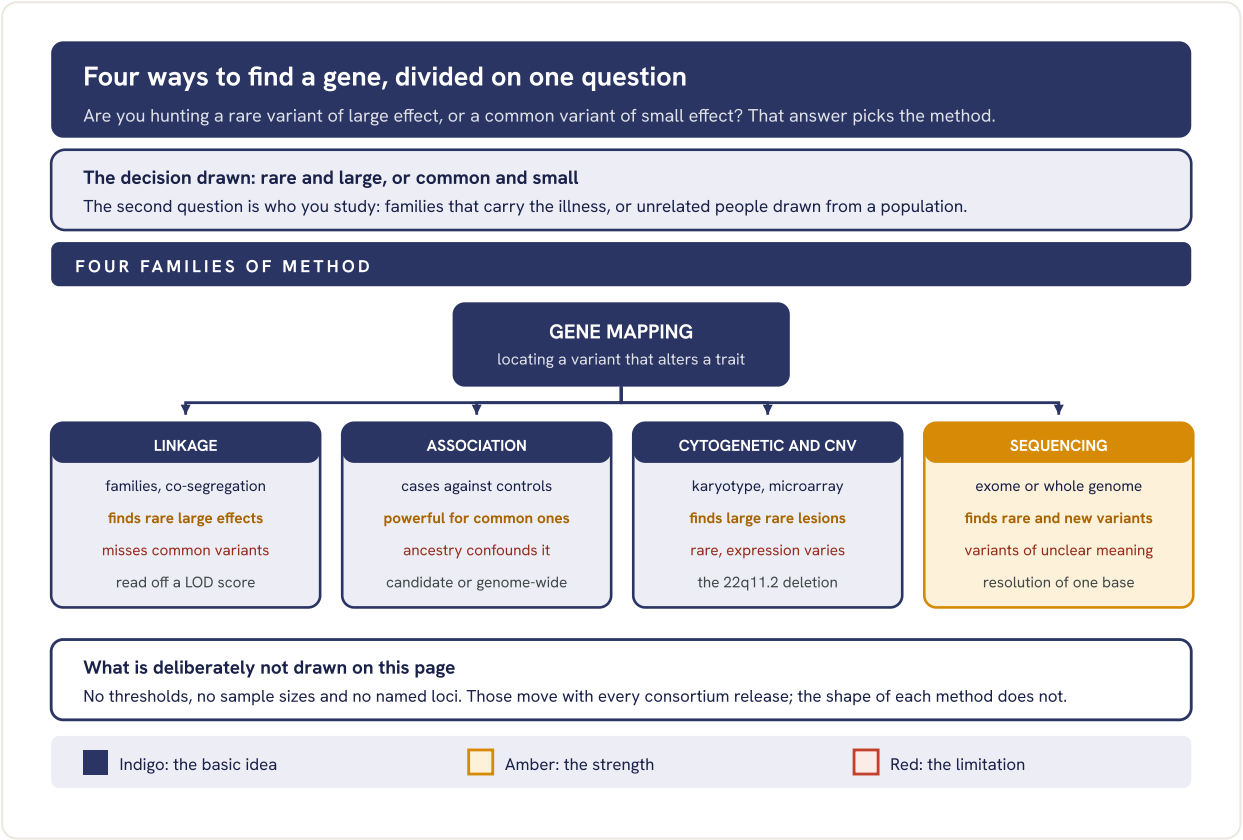


Fig 057.1 One axis, four branches, and inside each branch the same four lines in the same order, so the strategies can be read down the page as well as across it.

- **The basic ideas in one line each.** Linkage asks whether a marker travels with the illness through a family. Association asks whether an allele is commoner in cases than controls. Cytogenetics and microarray look for missing or duplicated stretches of chromosome. Sequencing reads the bases and compares them.
- **How they hand over to each other.** Linkage narrows a large region; association and sequencing resolve it to a variant. Practice today runs association genome-wide first, and keeps linkage for pedigrees with a striking inheritance pattern.

PITFALL

A genome-wide hit is not the gene. Linkage disequilibrium means the associated marker usually stands in for a nearby causal variant, and fine mapping and functional work are separate jobs that often fail. Saying so is the limitation mark that most answers leave on the table.

WHAT EARNS THE MARKS

- **The axis stated before the list.** Rare and large against common and small is what organises the four.
- **Idea, strength and limitation for every strategy,** in that order, because the stem asks for all three.
- **Linkage and association placed under gene mapping,** not offered as its alternatives.
- **The shared limitation.** A located region is not a mechanism.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 56 genome wide association studies

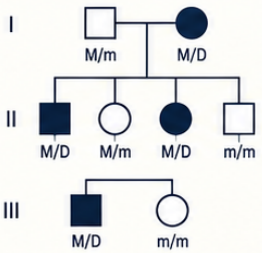
SPRINT Day 3, Psychiatric Genetics

Gene Mapping Strategies

Gene mapping is the goal; these are methods. Discovery is **not the same as a diagnostic test.**
Question 57 • Basic idea + example + strength + limitation

1. LINKAGE

Follow the clue through a family.



Marker allele: M
Disease allele: D

The DNA marker (M) travels with the illness (D) through generations. The marker is a nearby signpost, not necessarily the cause.

Basic idea / meaning
Does a DNA marker repeatedly travel with illness across generations?

Worked example
Huntington disease: family linkage mapped a marker to chromosome 4 (1983); *HTT* was identified later.

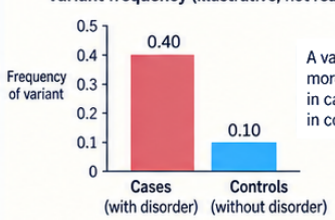
Strength
Useful for large-effect inherited variants in informative pedigrees.

Limitation
Broad region; weak for many tiny genetic effects.

Key exam words
Co-segregation • recombination • LOD = logarithm of odds.

2. ASSOCIATION

Compare many people.



A variant is more frequent in cases than in controls.

Basic idea / meaning
Is a variant more frequent in people with the disorder?

Worked example
Schizophrenia GWAS: associations near *DRD2*; 108 loci in the 2014 study.

More exam examples
Bipolar disorder: *CACNA1C* and *ANK3*.
Alzheimer disease: *APOE* ε4 is a risk allele, not a diagnosis.

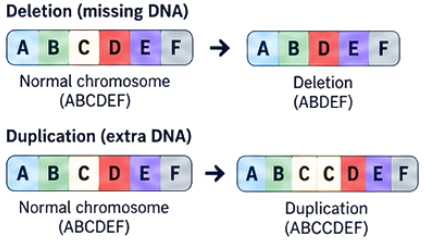
Strength
Finds common variants with small effects in huge samples.

Limitation
Ancestry can confound; association does not prove causation.

Key exam words
GWAS = genome-wide association study • population stratification • linkage disequilibrium.

3. CYTOGENETICS & CNV

Look for missing or extra DNA.



Basic idea / meaning
Karyotype views large chromosome changes; microarray finds smaller deletions and duplications.

Worked example
22q11.2 deletion: missing chromosome segment; increased schizophrenia risk.

More exam examples
Trisomy 21: extra chromosome, seen on karyotype.
16p11.2 deletion/duplication: neurodevelopmental risk.

Strength
Detects chromosome number changes and copy-number variants.

Limitation
Microarray misses balanced rearrangements and most single-letter changes.

Key exam words
CNV = copy-number variant • deletion • duplication • variable expression.

4. SEQUENCING

Read the DNA letters.

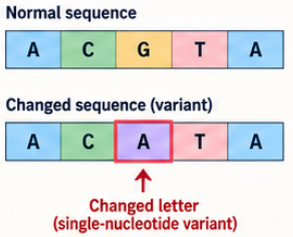


Illustration: A single letter change in the DNA sequence.

Basic idea / meaning
Read the base sequence. Exome = coding regions; whole genome = coding and non-coding regions.

Worked example
Schizophrenia exome studies: rare damaging variants in *SETD1A*.

More exam examples
Schizophrenia: *GRIN2A* and *TRIO*.
Familial Alzheimer disease: *APP, PSEN1, PSEN2*.

Strength
Finds rare coding variants; parent–child trios can identify new variants.

Limitation
A detected change may have uncertain meaning; routine exome can miss repeat expansions.

Key exam words
De *novo* = new in the child • variant of uncertain significance.

FAMILY → LINKAGE | FREQUENCY → ASSOCIATION | MISSING / EXTRA → CYTOGENETICS & CNV | LETTERS → SEQUENCING

Methods overlap and can complement one another. A mapped region is not automatically a causal gene. Repeat expansions such as *FMR1* often need dedicated testing.

Sources: NHGRI Huntington mapping history; Psychiatric Genomics Consortium 2014; SCHEMA 2022; MedlinePlus Genetics. Selected exam examples, not an exhaustive list.

Gene Mapping Strategies. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-57.

Credits and references: retained within the illustration.

I - 58

Twin studies in Bipolar affective disorder. [10]

10 GENETICS IN PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The twin method	Why two kinds of twin answer a genetic question
3	Design and assumptions	Zygosity, concordance, and the modelling that follows
3	What it shows in bipolar	The pattern, the heritability, and the spectrum finding
2	Limitations	The assumption that carries the whole method

THE METHOD IN TWO SENTENCES

Monozygotic twins share essentially all their segregating genes, dizygotic twins about half, and both kinds share a womb and a household. So any excess of monozygotic over dizygotic concordance is attributed to genes, provided the two kinds of twin really do share their environment equally.

ELEMENT	WHAT IT MEANS	WHAT IT DELIVERS	WHERE IT IS CHALLENGED
Zygosity	Established by genotyping, not by looks	The two comparison groups	Misclassification dilutes the estimate
Probandwise concordance	Counts each affected twin as a proband	The figure comparable with population risk	**Pairwise counting gives a lower number for the same data**
Equal environments	Both twin types share family environment equally	Makes the excess interpretable as genetic	**Identical twins are treated and treat each other more alike**
Variance modelling	Splits variance into genes, shared and unique environment	The heritability estimate	Assumes no gene-environment interaction

Single concordance percentages are not printed here on purpose: they swing with how wide the diagnostic boundary is drawn and with how the pairs were counted. Quote the pattern and the assumption, and you cannot be caught out on the number.

- **What twin studies show in bipolar disorder.** Monozygotic concordance is several times dizygotic concordance, and heritability estimates sit around eighty per cent, among the highest in psychiatry. Concordance is nowhere near complete, so environment and chance still matter.
- **The finding examiners want beyond the numbers.** Co-twins who are not bipolar are still at raised risk of unipolar depression and of other affective illness, which is evidence that what is inherited is a broad affective liability rather than a diagnosis.

WHAT EARNS THE MARKS

- **The logic of the method before any figure.** Two twin types, one difference, one inference.
- **The equal environments assumption named,** and named as the method's weak point.
- **Incomplete concordance used as an argument,** not treated as a failure of the genetic case.
- **The broadened phenotype in co-twins,** which is what makes twin work informative about nosology.

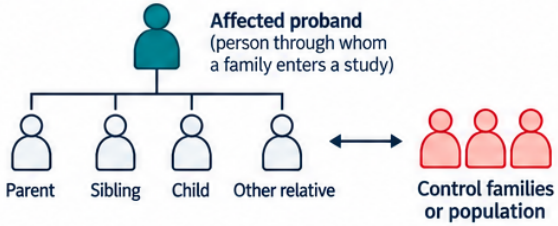
SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 60 adoption study designs SPRINT

Day 3, Psychiatric Genetics

Family, twin and adoption studies

FAMILY STUDIES

Assess biological relatives of an affected proband and compare with control families or population.



- Establish familial aggregation and estimate recurrence risk.
- Genes and shared environment remain confounded.
- Limitations: selection, diagnostic or reporting bias, age at risk and shared exposures.
- Familial does not automatically mean genetic.

TWIN STUDIES

Monozygotic (MZ) twins
(one fertilised egg; essentially the same inherited variants)



MZ twins
(100% of segregating variants shared)

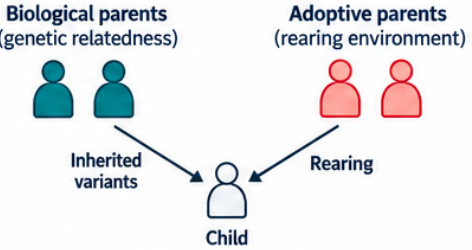
Dizygotic (DZ) twins
(two eggs; about half of segregating variants shared on average)



DZ twins
(~50% of segregating variants shared)

- Compare correlations for quantitative traits or concordance for diagnoses.
- Greater MZ similarity supports genetic contribution under assumptions.
- Equal-environments assumption: trait-relevant environmental similarity is comparable across zygosity; not that twins must dress differently or have identical lives. Violations may bias estimates.
- Ascertainment, diagnosis, non-additive effects and assortative mating (partners resembling each other) also matter.
- Reared-apart twins still share prenatal history; placement/contact can confound.

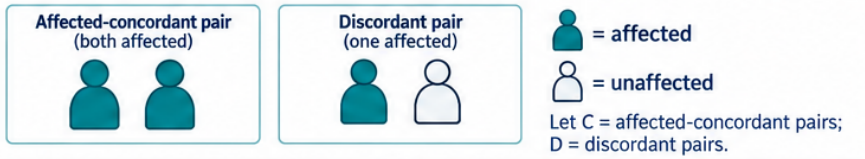
ADOPTION STUDIES



Prenatal influences can accompany biological relatedness.

- Compare adopted-away offspring of affected versus unaffected biological parents, or biological versus adoptive relatives of affected adoptees.
- Cross-fostering compares combinations of biological and rearing risk.
- Supports separation of genetic relatedness from rearing; not perfect experimental control.
- Limits: selective placement, prenatal/early-life exposures, later contact, restricted adoptive-home range.

CONCORDANCE: COUNT PAIRS OR PEOPLE?



Let C = affected-concordant pairs; D = discordant pairs.

$$\text{Pairwise} = \frac{C}{C + D}$$

$$\text{Probandwise} = \frac{2C}{2C + D}$$

Hypothetical: **C = 10, D = 20.**

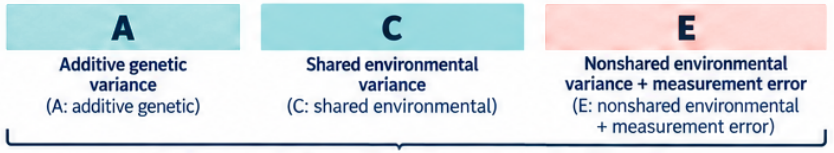
Pairwise = 10/30 = 33.3%.

Probandwise = 20/40 = 50%.

- Pairwise counts pairs with at least one affected twin; probandwise estimates affected co-twin probability given an affected twin.
- These simple formulas assume complete ascertainment; sampling may require correction.

HERITABILITY: POPULATION VARIANCE

Proportion of trait variance attributable to genetic differences in a specified population and environment. For diagnoses, often modelled on an underlying liability scale.



ACE variance model; C here is not the concordant-pair count.

- Narrow-sense h^2 concerns additive variance; broad-sense H^2 includes all genetic variance.
- Falconer approximation: $h^2 \approx 2(r_{MZ} - r_{DZ})$, using correlations under a simple additive model, not raw concordance percentages.

Heritability is NOT an individual's percentage of genetic causation, transmission probability or immutability. Concordance is NOT heritability.

Weave Sprint; MedlinePlus Genetics; McGue (1992).

Family, twin and adoption studies. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-58.

Credits and references: retained within the illustration.

I - 59

Define epigenetics. Discuss its contribution and implications in Psychiatry. [2+4+4]

2 + 4 + 4 | GENETICS IN PSYCHIATRY | DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	Change in expression without change in sequence
4 Mechanisms	Three marks, each with its direction of effect
2 Contribution	What it explains that genetics alone could not
2 Implications and limits	Targets and biomarkers, and why to be careful

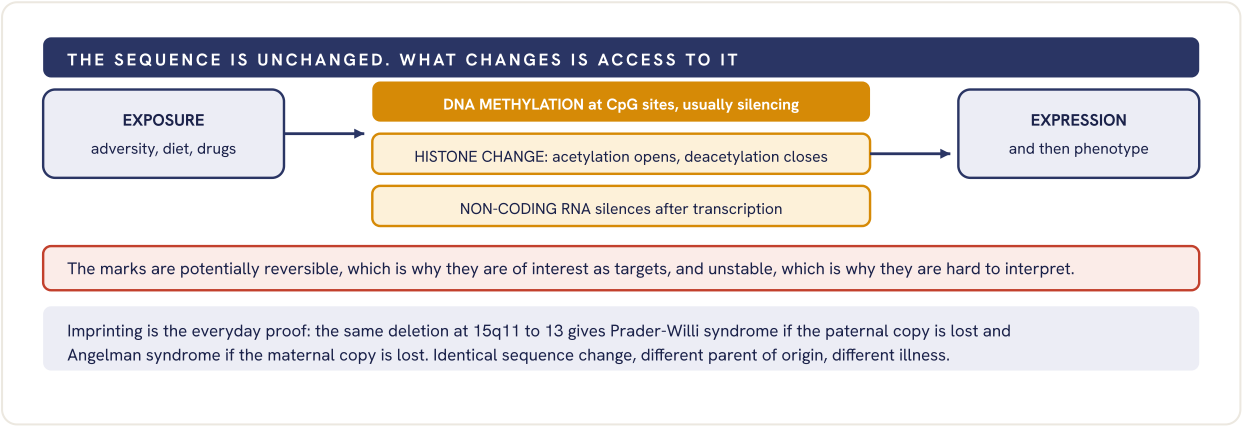


Fig 059.1 The three marks drawn as one layer between exposure and expression, with imprinting placed beneath as the demonstration that parental origin alone can change a phenotype.

- **Contribution to psychiatry.** It supplies a mechanism for gene-environment interplay, so early adversity can leave a lasting mark on stress-axis gene expression. It helps explain why monozygotic twins are discordant. Valproate is a histone deacetylase inhibitor, and folate and vitamin B12 feed the methyl supply, so the mechanism already touches prescribing.
- **The limits to state plainly.** Most human work measures blood, and blood is not brain. Direction of causation is rarely established: illness and its treatment change methylation too. Findings are strongly tissue and cell specific, and few replicate. No epigenetic test is in clinical use in psychiatry.

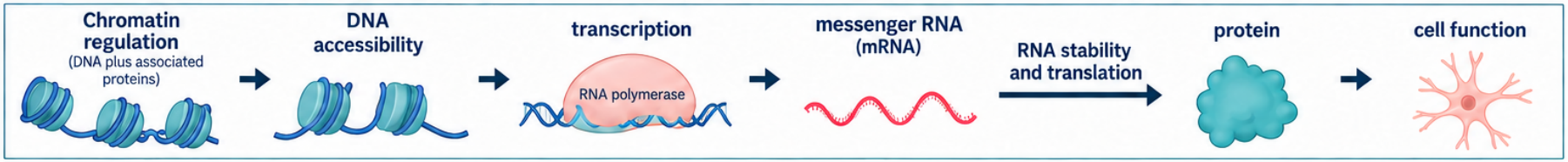
WHAT EARNS THE MARKS

- **The definition tied to sequence.** Expression changes, sequence does not, and heritable through cell division.
- **All three mechanisms, with a direction each.** Methylation alone is the commonest half answer.
- **One worked example.** Imprinting at 15q11 to 13 is the cleanest one available.
- **Reversibility named as the reason for interest,** and instability named as the reason for caution.

SEE ALSO Slot I - 55 genetic and environmental factors Slot I - 54 genetics of schizophrenia **SPRINT** Day 3, Psychiatric Genetics

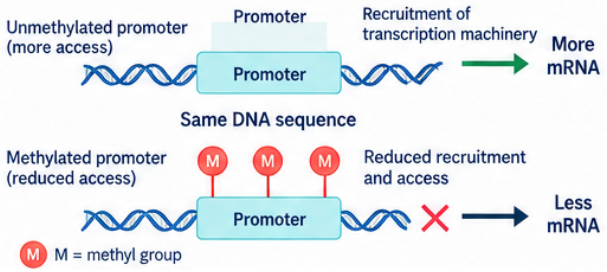
Epigenetics

Regulation of gene activity without changing the DNA sequence. Some states persist through cell division; not every regulatory change is heritable.



DNA METHYLATION

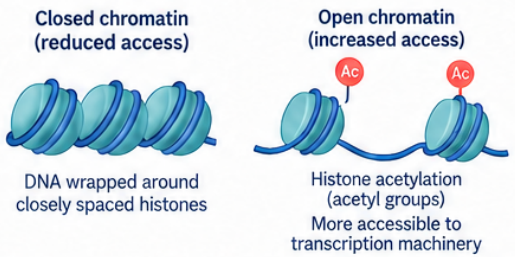
Methylation is a chemical mark, not a DNA mutation.
Methyl groups (CH_3) are added to cytosine, often at CpG sites (cytosine followed by guanine).
Promoter = regulatory region controlling transcription.



Promoter methylation often reduces transcription; effects elsewhere are context-dependent.

HISTONE MODIFICATION

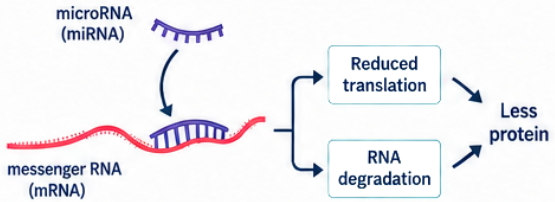
Histones are proteins around which DNA wraps.
Histone acetylation often increases accessibility and transcription; deacetylation often reduces them.
Histone methylation may activate or repress depending on the residue and context.



Chromatin remodelling repositions nucleosomes (DNA wrapped around histones) to alter access.

NON-CODING RNA

RNA that is not translated into protein can regulate expression.



Long non-coding RNA (lncRNA) may recruit chromatin-regulating complexes.

These mechanisms can interact; RNA regulation is not automatically a stable inherited epigenetic state.

RODENT EXAMPLE

Weaver et al., 2004: maternal care in rats was linked to hippocampal glucocorticoid-receptor promoter methylation, receptor expression and stress responses; cross-fostering supported environmental programming.

Higher licking/grooming → **Lower methylation at the studied promoter** → **Higher receptor expression** → **Stronger stress-hormone feedback**

Animal mechanism example, not a proven human parenting rule.

EXAM DISTINCTIONS

- **Maintenance through cell division ≠ transmission across generations.**
  Many marks are reset during germline/early development. Human transgenerational claims require much stronger evidence than a parent–child association.
- **Blood marks need not represent brain tissue.**
  Illness, medication and cell composition can alter measured marks; association alone does not establish cause.
- **No routine epigenetic test diagnoses common psychiatric disorders.**

DNA = deoxyribonucleic acid **RNA** = ribonucleic acid

Sources: Weave Sprint; National Human Genome Research Institute; Weaver et al., *Nature Neuroscience* (2004).

Epigenetics. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-59.

Credits and references: retained within the illustration.

I - 60

What do you understand by adoption studies? Describe different designs of adoption studies with special reference to Schizophrenia. [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What they are	The one thing adoption separates that twins cannot
4 The four designs	Who is sampled first decides which design it is
3 Findings and limits	What schizophrenia work established, and what to concede

DESIGN	WHO IS SAMPLED FIRST	WHAT IT SEPARATES	WHAT SCHIZOPHRENIA WORK SHOWED
Adoptee study	Adopted-away children of affected biological parents	Genes from rearing, prospectively	**Raised rates despite rearing away from the affected parent**
Adoptee's family method	Adopted people who are already ill	Biological relatives from adoptive relatives	**Excess illness in biological relatives only**
Cross-fostering	Children of well parents reared by affected adoptive parents	The effect of rearing on its own	Being reared by an affected parent did not raise risk
Adoptive family study	High-risk and low-risk adoptees, with the adoptive family assessed	Interaction between the two	High genetic risk mattered mainly in disturbed adoptive families

The amber rows are the two designs that carry the genetic claim, from opposite ends. The fourth put interaction back into a literature that had been read as proof of genes alone.

- **What adoption adds that twins cannot.** A twin pair shares its rearing, so a twin study cannot fully separate genes from family environment. Adoption breaks the two apart: the child carries the genes of one family and grows up in another.
- **The spectrum finding.** Biological relatives of adopted people with schizophrenia showed an excess not only of schizophrenia but of schizotypal and paranoid presentations. That is the evidence base for the idea of a schizophrenia spectrum, and it came from adoption work.
- **Limitations to concede.** Placement is not random and adoptive homes are screened, which narrows the environmental range. Late placement leaves early rearing shared. The biological mother still supplies the prenatal environment. Numbers are small, and adoption itself selects an unusual population.

WHAT EARNS THE MARKS

- **The comparison with twin studies made explicit,** since it is the reason the design exists.
- **All four designs named and told apart by who is sampled first.**
- **The spectrum finding credited to adoption work,** which is the "special reference to schizophrenia" the stem is asking for.
- **The prenatal environment named among the limits.** It is the one candidates most often miss.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 58 twin studies in bipolar affective disorder SPRINT Day 3, Psychiatric Genetics

I - 61

What is gene mapping? How is it different from linkage analysis and association studies? [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gene mapping defined	The umbrella, with the other two placed under it
4 The grid	Domain by domain, with the discriminators marked
3 Overlap and what follows	Where the two methods meet, and how they are sequenced

DOMAIN	LINKAGE ANALYSIS	ASSOCIATION STUDY	WHAT SETTLES IT
Who is studied	Families with several affected members	Unrelated cases and controls, or trios	**Related people or strangers**
What is tracked	Co-segregation of a marker with illness within a family	Allele frequency difference between groups	**Within a pedigree or across a population**
Distance it works over	Long, tens of millions of bases	Very short, the span of linkage disequilibrium	**Resolution: a region against a variant**
Statistic	The LOD score	A p value against a genome-wide threshold	Different traditions, same logic
Best suited to	Rare variants of large effect	Common variants of small effect	**Effect size and allele frequency**
Main failure mode	Underpowered for common small effects	Confounded by population stratification	Each fails where the other works

Amber cells are the discriminators. Each reduces to the same pair of questions: whose DNA is compared, and how big is the effect being hunted.

- **The overlap.** Both are gene mapping, and family-based association tests sit in between: the transmission disequilibrium test compares the alleles a parent passes on with the alleles the same parent does not, so it is an association test that cannot be confounded by ancestry.
- **What follows from the difference.** Linkage narrows the search to a region; association resolves the region to a variant; sequencing reads it. They are stages, not rivals, and saying so answers the stem's hidden question.

PITFALL

The stem invites you to treat all three as parallel alternatives. They are not. Gene mapping is the goal; linkage and association are two ways of reaching it. Opening with that sentence earns the first three marks before any comparison begins.

WHAT EARNS THE MARKS

- **Gene mapping defined as the umbrella,** in the first line, not implied by the layout.
- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The discriminators identified as discriminators,** rather than a flat list of six differences.
- **The family-based association test offered as the overlap,** which is the depth mark on this question.

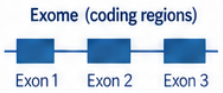
SEE ALSO Slot I - 57 gene mapping strategies Slot I - 56 genome wide association studies SPRINT Day 3, Psychiatric Genetics

Modern genetic approaches and gene therapy

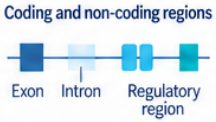
Find a variant
Generate and interpret genetic data

FIND AND INTERPRET VARIANTS

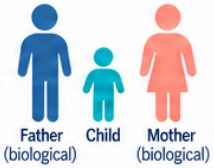
Whole-exome sequencing (WES)
- Reads protein-coding regions (exons).
- Useful for rare coding variants.



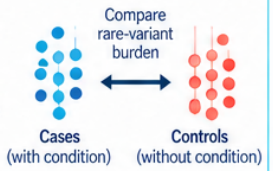
Whole-genome sequencing (WGS)
- Reads coding and non-coding regions.
- Broader structural-variant detection.
- Both WES and WGS have coverage and interpretation limits; no platform detects every variant.
- Dedicated testing may be needed for repeat expansions.



Trio sequencing
- Child + both biological parents → identify candidate *de novo* (new) variants.
- Negative parental blood testing does not exclude germline mosaicism.



Rare-variant burden test
- Aggregate rare variants in a gene → compare cases with controls.
- Statistical enrichment does not make every variant pathogenic.

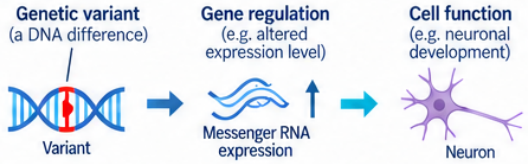


Clinical use
- Selected developmental or syndromic presentations.
- Interpret with phenotype, family history and variant classification.

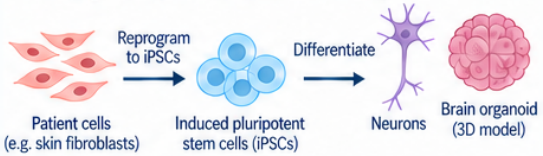
Test its function
Understand biological impact

TEST BIOLOGICAL FUNCTION
(research)

Variant → gene regulation → cell function
- Expression quantitative trait locus (eQTL) = variant associated with gene-expression level.
- Single-cell RNA sequencing = measures ribonucleic acid (RNA) to identify cell-type expression.
- Association prioritises targets, not proof of mechanism.



Patient cells → induced pluripotent stem cells → neurons or brain organoids
- Patient cells can be reprogrammed to induced pluripotent stem cells (iPSCs) and then differentiated into neurons or brain organoids (simplified 3D tissue models).



Genome editing
- Guide RNA directs a DNA-modifying enzyme to a target.
- Clustered regularly interspaced short palindromic repeats (CRISPR) with Cas9 (CRISPR-associated protein 9) can disrupt or alter DNA; base and prime editing offer other changes.
- Compare edited cells with matched controls and test rescue.
- Causality in a cell model is not proven efficacy in people.



DNA = deoxyribonucleic acid; RNA = ribonucleic acid.

Assess treatment safely
Develop and evaluate therapies

INTERVENE
(clinical status)

ESTABLISHED EXAMPLES — UNITED STATES APPROVALS

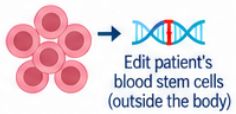
Onasemnogene abeparvovec
- Gene addition using an adeno-associated virus (AAV) vector for selected spinal muscular atrophy (SMA) patients.
- Supplies survival motor neuron 1 (SMN1).



Nusinersen
- Antisense oligonucleotide changes survival motor neuron 2 (SMN2) RNA splicing.
- RNA treatment, NOT DNA editing.



Exagamglogene autotemcel
- Edits a regulatory region in a patient's blood stem cells outside the body.
- Approved for selected sickle cell disease and beta-thalassemia indications.



These are not psychiatric treatments.

EXPERIMENTAL
- Huntingtin-lowering and other gene-directed interventions for Huntington disease remain investigational.
- A trial does not establish benefit.

NOT ESTABLISHED
- No approved gene therapy for common schizophrenia, bipolar disorder or major depression.

KEY EXAM DISTINCTIONS
- Sequencing reads; gene addition supplies; RNA therapy changes RNA; genome editing changes DNA.
- Somatic treatment targets the treated person; heritable germline editing raises distinct safety and ethical concerns.
- Delivery, off-target effects, durability, immune reactions and clinical outcomes require testing.
- Approval depends on jurisdiction and indication.
- Sources: FDA product information; GeneReviews; Weave Sprint.
- Status checked September 2026.

Modern genetic approaches and gene therapy. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-61.

Credits and references: retained within the illustration.

I - 62

Genetic correlation, pleiotropy, and associations between substance use and psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The two terms	Defined apart, because they are not the same claim
2	How they are measured	Twin designs first, then summary-statistic methods
3	Substance use findings	What overlaps with what, and the measurement caveat
2	What it does not prove	Correlation, direction, and how direction is tested

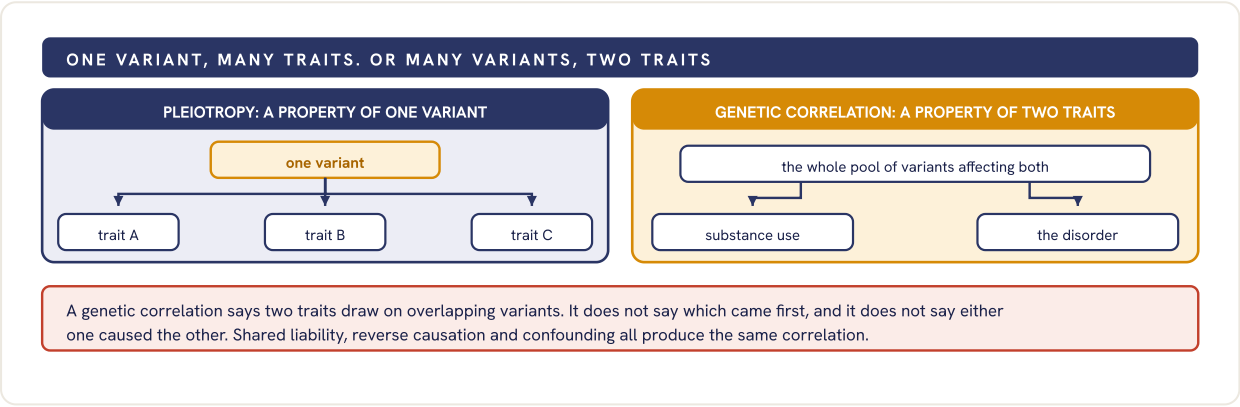


Fig 062.1 The two constructs drawn side by side because they are routinely merged: pleiotropy is a statement about one variant, genetic correlation a statement about two traits.

- **How they are measured, and what is found.** Twin designs give cross-twin cross-trait correlations; modern estimates come from genome-wide summary statistics by linkage disequilibrium score regression. Substantial genetic correlation runs across schizophrenia, bipolar disorder, depression and ADHD, and substance use traits correlate with all of them, most strongly with ADHD and with depression.
- **The measurement caveat, and the causal test.** How much a person drinks and whether the drinking has become a disorder are genetically different traits that correlate differently with psychiatric illness, so say which you mean. For direction the tool is Mendelian randomisation, run both ways between cannabis and schizophrenia.

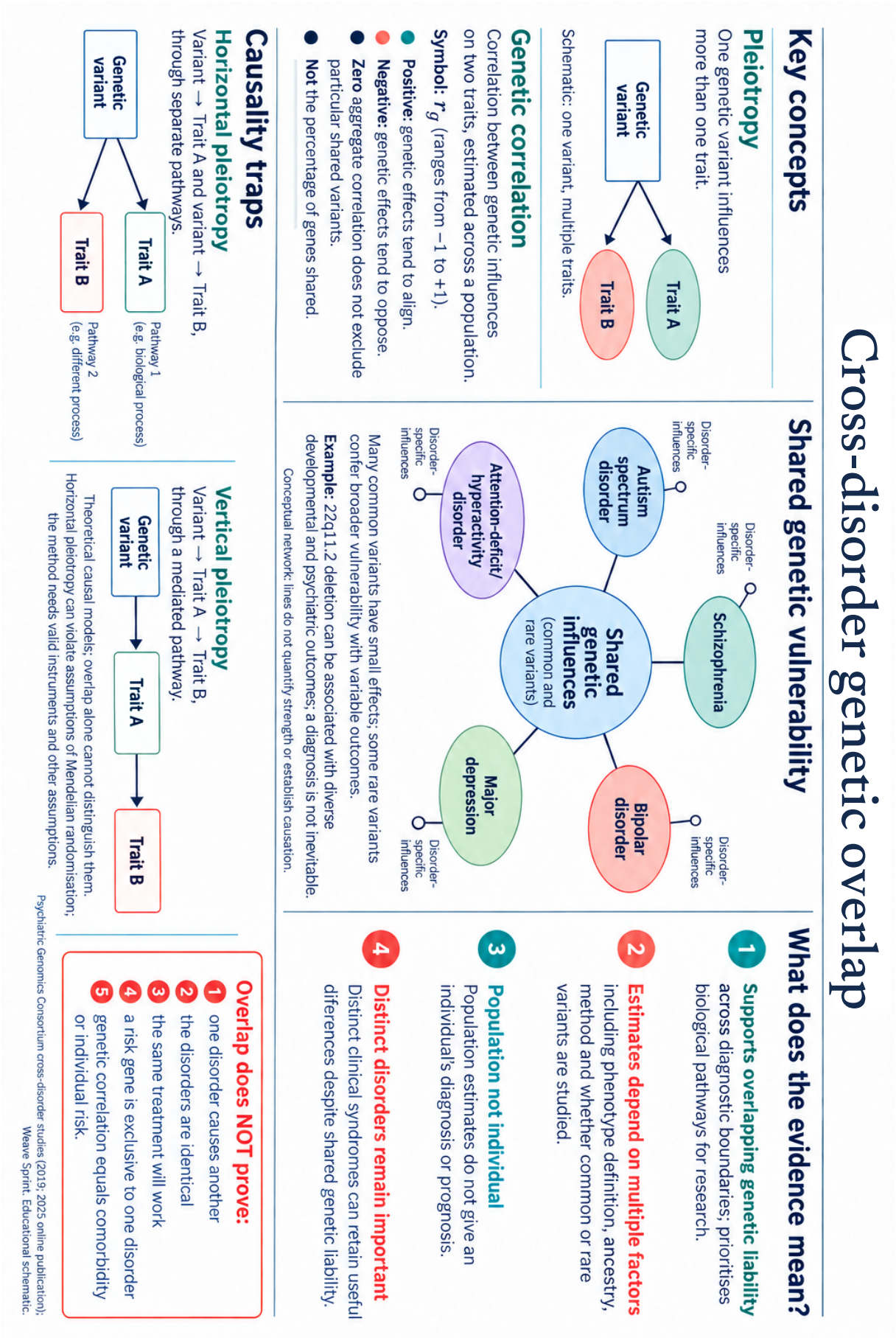
PITFALL

Do not use the two terms as synonyms. Separate horizontal pleiotropy, one variant affecting two traits independently, from vertical, acting on the second only through the first. Mendelian randomisation is valid only under the horizontal assumption.

WHAT EARNS THE MARKS

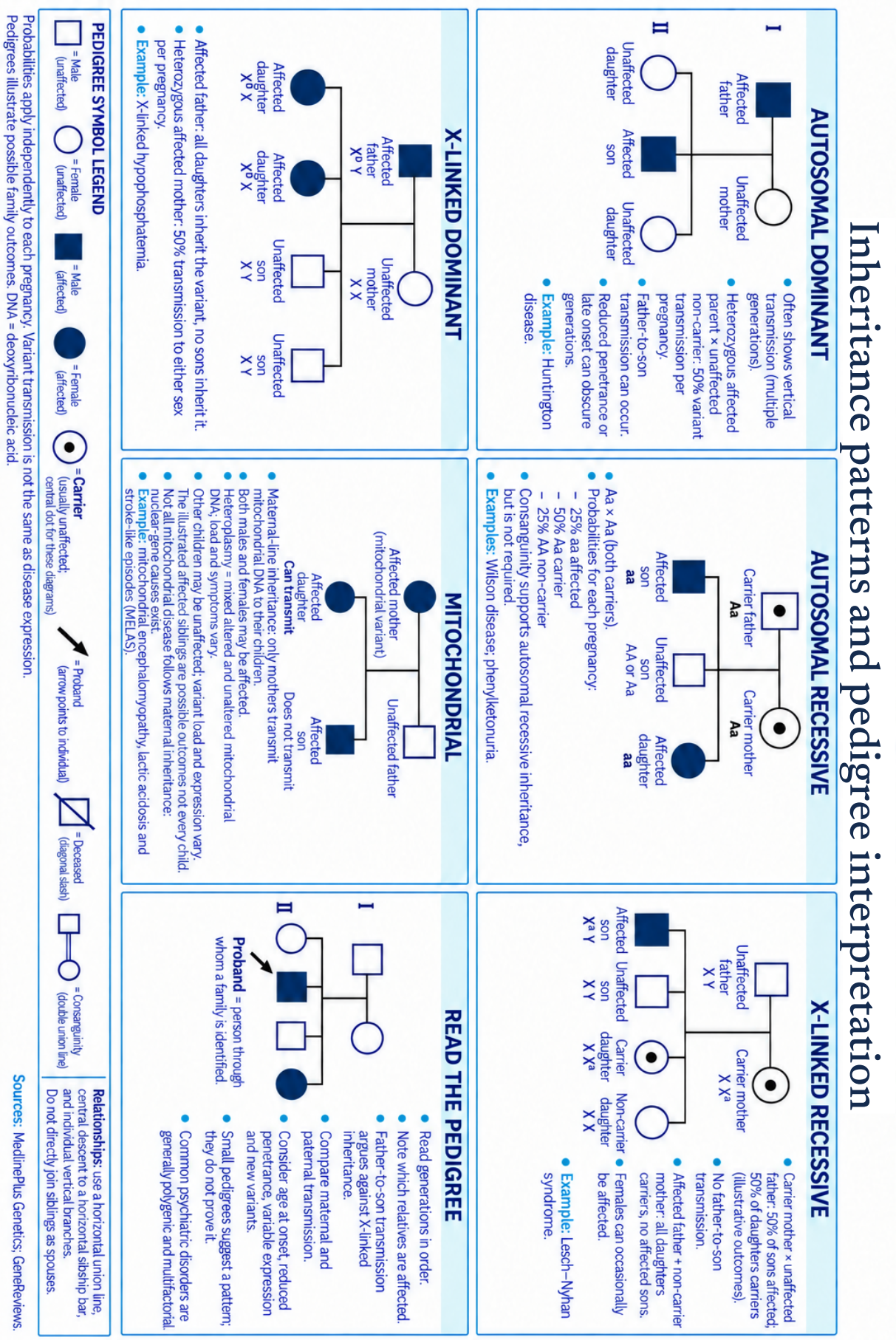
- **Two separate definitions,** one about a variant and one about a pair of traits.
- **Horizontal separated from vertical pleiotropy,** which is the discriminator this question is built around.
- **Consumption distinguished from disorder** when reporting any substance use finding.
- **Causation addressed explicitly,** with Mendelian randomisation named as the method that tests it.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 54 genetics of schizophrenia **SPRINT** Day 3, Psychiatric Genetics



Cross-disorder genetic overlap. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-62.

Credits and references: retained within the illustration.



Inheritance patterns and pedigree interpretation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Genetics in psychiatry.

Credits and references: retained within the illustration.

Genetic terminology and mechanisms

Foundation: key terms

- **Allele** = a version of a gene.
- **Genotype** = genetic constitution.
- **Phenotype** = observable features.
- **Heterozygous** = two different alleles.
- **Homozygous** = two matching alleles.

PENETRANCE

"Does the phenotype appear?"

Proportion of people with a specified genotype who express the phenotype, defined for an age and context.

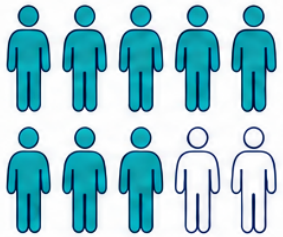


Illustration only: 8/10 = 80% penetrance

Example

Huntington disease alleles with 36–39 cytosine–adenine–guanine (CAG) repeats have reduced penetrance.

Not a transmission probability.

VARIABLE EXPRESSIVITY

"How does it appear?"

People with the same disease-associated genotype can have different features or severity.



(All have the same disease-associated genotype)

Example

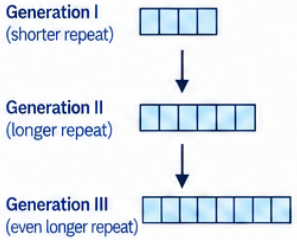
Neurofibromatosis type 1 can vary markedly within a family.

Penetrance = whether the phenotype appears. Expressivity = how it appears (severity or features).

ANTICIPATION

"Earlier onset in successive generations."

Repeat expansion can produce earlier onset, sometimes greater severity.



Schematic: increasing repeat length across generations

Example

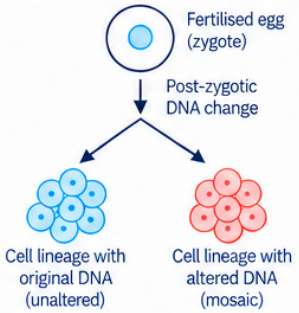
Huntington disease; expansion more often during paternal transmission.

Apparent earlier onset alone does not prove anticipation.

MOSAICISM

"Different genetic cell populations in one person."

One fertilised egg → post-zygotic DNA change → two cell lineages, one altered and one unaltered.



Somatic mosaicism: tissue-limited phenotype.

Germline mosaicism: eggs or sperm affected, so recurrence is possible despite negative parental blood testing.

Example

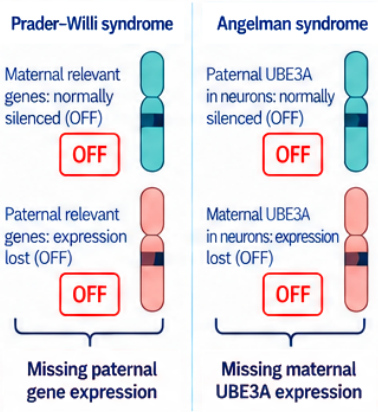
Mosaic neurofibromatosis type 1.

Mosaicism differs from chimerism, which involves more than one zygote.

GENOMIC IMPRINTING

"Parent of origin changes expression."

Some alleles are epigenetically silenced according to maternal or paternal origin.



UBE3A = ubiquitin protein ligase E3A gene.

Deletion, uniparental disomy or imprinting defects can cause loss of expression; not every case is a deletion.

Uniparental disomy = both copies from one parent.

Key contrasts

- Same genotype, different phenotype does not automatically mean a new mutation.
- Imprinting is parent-of-origin expression, not dominant versus recessive inheritance.

Sources: MedlinePlus Genetics; GeneReviews.

DNA = deoxyribonucleic acid.

Genetic terminology and mechanisms. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Genetics in psychiatry.

Credits and references: retained within the illustration.

Genetic counselling in psychiatry

A supportive communication process that helps people understand familial vulnerability, uncertainty and options, and make informed choices.

1. Explore concerns and goals
2. Clarify diagnosis and medical history
3. Build a three-generation family history
4. Explain multifactorial inheritance and empirical risk
5. Discuss testing or referral where useful
6. Check understanding, support and follow-up

COMMUNICATING RISK

- Family history changes probability, not destiny.
- Common psychiatric disorders usually reflect many genetic variants plus environmental and developmental factors.
- Use absolute risk, a named outcome, time horizon, source population and uncertainty.
- Avoid replacing recurrence risk with heritability or twin concordance.
- Teaching example only, NOT a psychiatric risk estimate: if risk were 10 in 100, 90 in 100 would not develop that specified outcome over the same period.

10 + 90 = 100

- Ask whether numerical estimates are wanted; address guilt, blame and stigma.
- No universal Mendelian 25% or 50% for schizophrenia or bipolar disorder.

WHEN REFERRAL OR TESTING MAY HELP

- **Clinical genetics referral:**
  - intellectual disability or developmental delay
  - congenital anomalies or unusual physical features
  - epilepsy or developmental regression
  - suspected chromosomal or single-gene syndrome
  - known familial pathogenic variant
  - informative family history or consanguinity with a suggestive phenotype.

Example
Psychosis with developmental and congenital features → consider a syndrome such as 22q11.2 deletion → specialist-directed testing.

- **Test chosen by phenotype:**
  - chromosomal microarray
  - sequencing (for example, single-gene or gene panel)
  - dedicated repeat-expansion testing
- Counselling is useful even without testing.
- Distinguish diagnostic testing, predictive testing for a known familial condition (such as Huntington disease), and drug-specific pharmacogenetic testing.
- Polygenic risk scores are research tools, not routine psychiatric diagnostic or predictive tests.

ETHICS AND RESULT INTERPRETATION

- **Voluntary informed consent;** assess decision-specific capacity and support it.
- **Right to know** or not know.
- **Confidentiality;** agree family involvement and disclosure.
- **Discuss secondary findings** before broad testing.
- **Pathogenic result** may explain a syndrome; susceptibility does not guarantee illness.
- **Variant of uncertain significance (VUS)** = insufficient evidence; not a diagnosis.
- **Negative result** does not exclude genetic contribution.
- **Protect reproductive autonomy;** no pressure about marriage, pregnancy or childbearing.
- **Address ancestry-related validity,** cost and equitable access.
- **Predictive testing of minors** for adult-onset conditions is generally deferred without a childhood medical benefit.

Counselling supports understanding and autonomous decisions; it does not promise certainty or select a 'correct' reproductive choice.

Sources: International Society of Psychiatric Genetics testing statement; Weave Sprint Day 3.
Clinical status checked September 2026.

Genetic counselling in psychiatry. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Genetics in psychiatry.

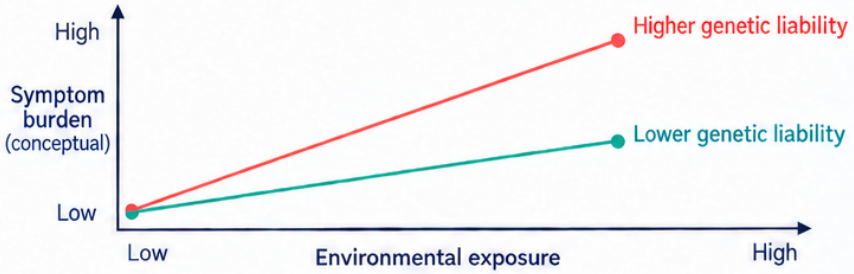
Credits and references: retained within the illustration.

Gene–environment interaction versus gene–environment correlation

Interaction: does exposure have a different effect?

Gene–environment interaction (G × E)

The effect of an environmental exposure on an outcome differs according to genotype or genetic liability.



Illustrative non-parallel slopes on this scale; not patient data. Lines need not cross.

Phenylketonuria: enzyme deficiency

High phenylalanine exposure → Accumulation → Neurodevelopmental harm

Early controlled phenylalanine intake → Much harm can be prevented

Given phenylalanine hydroxylase deficiency, changing exposure modifies harm; the genetic condition remains.
Clinical dietary care requires specialist supervision.

Correlation: who encounters the exposure?

Gene–environment correlation (rGE)

Genetic influences are associated with differences in environmental exposure, often through behaviour.

1 Passive — parents provide both

Parent's genetically influenced verbal traits → Child's inherited tendencies; Language-rich home

The child does not have to choose or evoke the environment.

2 Evocative — responses from others

Child's partly heritable talkativeness → More conversation from adults

The child's behaviour elicits a response.

3 Active — selecting environments

Partly heritable interest in conversation → chooses discussion clubs and talkative peers

The person seeks a compatible setting.

Illustrative tendencies involving many genes and environments; no single "talkativeness gene".

Key distinctions and exam traps

- Interaction concerns outcome differences across exposure; correlation concerns non-random exposure. Both can coexist.
- Statistical interaction depends on the measurement scale; replication and adequate sample size matter.
- An environmental measure showing genetic influence can still have causal environmental effects.
- Correlation does not imply inevitability, conscious genetic choice or parental blame.
- Classic candidate-gene × stress findings are contested; do not present one variant as a settled psychiatric prediction test.

Weave Sprint; primary twin and gene–environment studies; source details in accompanying notes.

Gene-environment interaction versus gene-environment correlation. Read across the labelled contrasts to distinguish the related concepts. Section companion: Genetics in psychiatry.

LOD Score

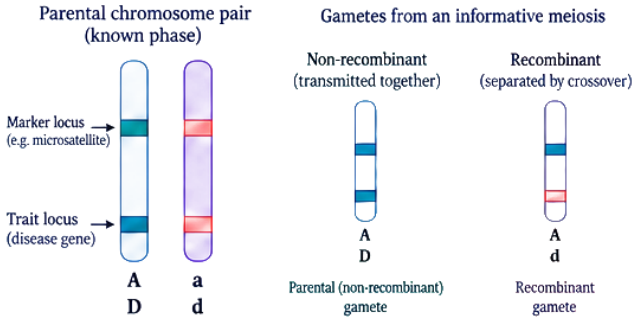
Logarithm of odds in genetic linkage

Concept and formula

$$Z(\theta) = \log_{10}\left[\frac{L(\text{data} \mid \text{linkage at } \theta)}{L(\text{data} \mid \text{no linkage}, \theta = 0.5)}\right]$$

- LOD (logarithm of odds) score compares how likely the observed pedigree data are under linkage at a given recombination fraction (θ) versus no linkage ($\theta = 0.5$).
- θ is the recombination fraction, ranging from 0 to 0.5.
- Small θ suggests close linkage; $\theta = 0.5$ indicates no detectable linkage (independent assortment).
- A LOD score is not a p-value and not a posterior probability.
- Multiple testing and the genetic model used can affect interpretation.

Informative meiosis: co-segregation and recombination



Non-recombinant gametes inherit the marker and trait allele together; recombinant gametes arise after a crossover between the loci.

Interpreting LOD scores

LOD score (Z)	Interpretation (at given θ)	Likelihood ratio (in favour of linkage)
$Z \geq 3$	Supports linkage	$\geq 1000 : 1$
$-2 < Z < 3$	Inconclusive	Between $1 : 100$ and $1000 : 1$
$Z \leq -2$	Evidence against linkage	$\leq 1 : 100$

- These thresholds are conventional and apply at a specific θ.
- Multiple-testing and model context (e.g. number of loci tested, inheritance model) matter for interpretation.
- A LOD score is not a p-value and not a posterior probability.

Worked example (idealized model)

Fully informative markers, known phase, independent meioses. Observe 10 non-recombinant and 0 recombinant meioses at $\theta = 0$.

Model	Likelihood	Value
Linkage at $\theta = 0$ (no recombination)	$L_{\text{linked}} = (1-\theta)^{10}\theta^{0}$	$1^{10} = 1$
No linkage ($\theta = 0.5$)	$L_{\text{unlinked}} = (0.5)^{10}$	$\frac{1}{1024}$

Likelihood ratio = $1 / (1/1024) = 1024$

$$Z = \log_{10}(1024) = 3.0103$$

This provides strong support for linkage at $\theta = 0$ in this idealized example.

Real pedigrees require specification of penetrance, allele frequencies and a genetic model. This example assumes fully informative meioses and no genotyping error.

Same worked example across theta

$Z(\theta) = 10 \log_{10}[2(1-\theta)]$ for 10 non-recombinants and 0 recombinants.

Recombination fraction (θ)	LOD score (Z)
0	3.0103
0.1	2.5527
0.2	2.0412
0.3	1.4613
0.4	0.7918
0.5	0.0000

Maximum at $\theta = 0$ (most likely recombination fraction).

- LOD scores across independent families add at the same θ under a compatible genetic model.

Linkage versus association

Linkage (pedigrees)	Association (populations)
• Tracks co-segregation of marker and trait within families.	• Tests for an allele–trait relationship in populations (e.g. case–control or cohort studies).
• Tests whether loci are inherited together more often than expected by chance ($\theta < 0.5$).	• Based on linkage disequilibrium between marker and causal variant.
• Most powerful for rare, large-effect variants in families.	• More powerful for common variants with small effects.

Important caveats

- Psychiatric disorders are often polygenic and genetically heterogeneous, so single large-effect linkage signals may be absent.
- The recombination fraction (θ) is not an exact physical distance; 1% recombination ($\theta = 0.01$) approximates 1 centimorgan (cM) only over short distances.
- Real analyses require model assumptions (e.g. penetrance, allele frequencies, genetic heterogeneity) and allow for genotyping error.
- Consider multiple testing when scanning many loci or markers.

LOD Score. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Genetics in psychiatry.

Credits and references: retained within the illustration.

AREA 07 OF 12 · P1-A08

Social, cross-cultural and evolutionary psychology

8	7.8%	I-63	I-70
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

Build one culture answer properly and four of the eight slots open. Take the definitional slot first and the culture-bound syndromes last of the four, because the syndromes are a list and the other three are an argument. The social theories of suicide next, it is the one that needs its own reading. Religion, marriage and imprinting are independent and all three are short.

WHAT IS IN THIS AREA

Culture in four slots: its role in psychiatry, the definition with illness behaviour and symptomatology, the culture-bound syndromes with five Indian ones and the cultural theories of mental illness, and the slot that runs culture through expression, assessment and treatment and adds immigration. Then the social theories of suicide, religion and psychiatry, marriage as an institution, and imprinting, which is the one ethology slot in this book.

I - 63

What is culture bound syndrome? Describe any five Indian culture bound syndrome. Discuss different theories of mental illness from cultural perspective. [1+5+4]

1 + 5 + 4 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	Definition	The term, and what DSM-5 put in its place
5	Five Indian	Each with its region and its presentation
3	The theories	Etic against emic, and the category fallacy
1	The caution	Where the concept is criticised

THE DEFINITION

A recurrent, locality-specific pattern of aberrant behaviour and distressing experience that is recognised as an illness within its own culture and often has no direct counterpart in the international classifications.

SYNDROME	WHERE	THE PRESENTATION	BEST UNDERSTOOD AS
Dhat	North and west India, South Asia widely	Fatigue, weakness and anxiety attributed to semen passed in urine	**An idiom of distress; usually depression or anxiety**
Koro	Epidemics in Assam and West Bengal	Acute terror that the genitals are retracting, with fear of death	Acute anxiety, spread by contagion
Possession states	Across India, more often in women	Trance, altered identity, a spirit speaking through the person	Dissociative trance and possession disorder
Ascetic syndrome	Described in India by Neki	Adolescent, usually male: withdrawal, severe religiosity, self-imposed austerity	Often the prodrome of schizophrenia
Gilhari syndrome	Rajasthan	A swelling believed to move under the skin and to kill on reaching the neck	A culturally shaped conversion presentation

- **The theories, which are the last four marks.** The universalist or etic position holds disorders to be the same everywhere, with culture only colouring them. The relativist or emic position holds categories to be meaningful only inside the culture that made them. Kleinman named the category fallacy, the error of carrying a category built in one culture into another where it has no coherence, and set the patient's explanatory model against the professional one.
- **What DSM-5 did to the term.** It retired culture-bound syndrome and put three things in its place: cultural syndromes, cultural idioms of distress, and cultural explanations of distress. Saying so is worth a mark.

WHAT EARNS THE MARKS

- **Exactly five, each with a region and a presentation.** The stem says five, so a sixth earns nothing and a fourth loses a mark.
- **Etic and emic named as positions,** not described as attitudes.
- **The category fallacy, attributed.** It is the single highest-value term in the last section.

SEE ALSO Slot I - 70 culture, illness behaviour and symptomatology

Slot I - 67 the role of culture across the clinical pathway **SPRINT** Day 5, social, learning and personality psychology

I - 64

Concept of imprinting in ethology. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Rapid, timed, unrewarded, lasting
3 The classic work	Lorenz, the geese, and the window
3 The two kinds	Filial and sexual, and their separate windows
2 Use in psychiatry	The line from ethology to attachment

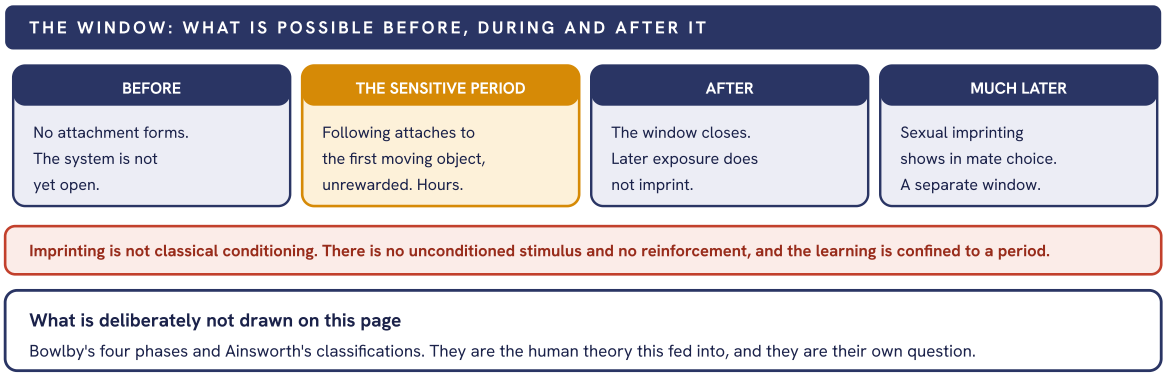


Fig 064.1 Imprinting drawn as a window rather than a process. Amber marks the sensitive period itself, which is the claim the concept stands or falls on; the fourth panel is the separate, much later window for sexual imprinting.

- **The definition, with its authority.** A rapid form of learning, confined to a sensitive period early in life, by which a young animal forms an attachment to a moving object, usually the parent, without reward and with lasting effect. Konrad Lorenz established it in greylag geese from 1935, the observation having been made by Spalding in the previous century. Lorenz shared the 1973 Nobel Prize with Tinbergen and von Frisch.
- **Why psychiatry keeps it.** It supplied the biological argument Bowlby used: attachment as a species-typical behavioural system with its own sensitive period, not a by-product of feeding. Harlow's monkeys made the same point from the other side, preferring the cloth mother to the one that fed them.

WHAT EARNS THE MARKS

- **All four defining features.** Rapid, period-bound, without reinforcement, and persistent.
- **Lorenz and the geese, with the year.** The classic experiment is the answer's evidence.
- **Filial and sexual imprinting separated,** with their windows named as separate.
- **The bridge to Bowlby.** On a psychiatry paper an ethology answer that never reaches attachment is incomplete.

SEE ALSO Slot I - 89 attachment theory SPRINT Day 5, social, learning and personality psychology

I - 65

Critically evaluate marriage as an institution. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The institution	What marriage is for, stated as functions
4	The case both ways	Domain by domain, for and against
2	The evidence	What the mental health data support
2	The verdict	A position, defended, not a fence

DOMAIN	THE CASE FOR	THE CASE AGAINST	WHAT THE EVIDENCE SHOWS
Mental health	Less psychiatric morbidity and lower suicide rates among the married	A bad marriage is worse than none	**Protection is larger for men; selection and causation both operate**
Social function	Legitimises children, transmits property, organises care of the old and ill	Concentrates unpaid care on women	The function is real, its distribution is contested
Support in illness	A confiding relationship buffers against depression	The spouse can be the source of the stress	A confidant protects; marriage does not guarantee one
Indian context	Near universal; family support is built in	Dowry, early marriage and domestic violence carry direct harm	Both hold at once and belong in one answer
Stability	Divorce is uncommon	Low divorce is not high satisfaction	Stability and quality are separate measures

The fourth column is what makes this a critical evaluation rather than a list. Amber marks the finding the verdict rests on.

- **The verdict, which is what the verb asks for.** Marriage is associated with better mental health; the association is stronger for men than for women; it is partly selection rather than protection; and the quality of the marriage predicts more than the fact of it. Commit to that and defend it, rather than balancing two lists and stopping.
- **The psychiatric handles.** Marital status is a standard variable in suicide and depression epidemiology, marital discord is a recognised focus of clinical attention, and couple therapy is an evidence-based treatment for depression where discord maintains it.

WHAT EARNS THE MARKS

- **A stated verdict.** Critically evaluate is marked on whether a position was taken and defended.
- **Both sides of at least four domains,** which is what stops the answer becoming an opinion.
- **Selection distinguished from protection.** It is the methodological point that lifts the answer.
- **The Indian material named,** not implied by the setting.

SEE ALSO Slot I - 66 social theories of suicide SPRINT Day 5, social, learning and personality psychology

I - 66

Social theories of suicide. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The claim	Suicide rates as social facts
4	Durkheim's four	Two axes, each failing in two directions
2	After Durkheim	Status integration, the critique, contagion
2	The limit	Rates against individuals

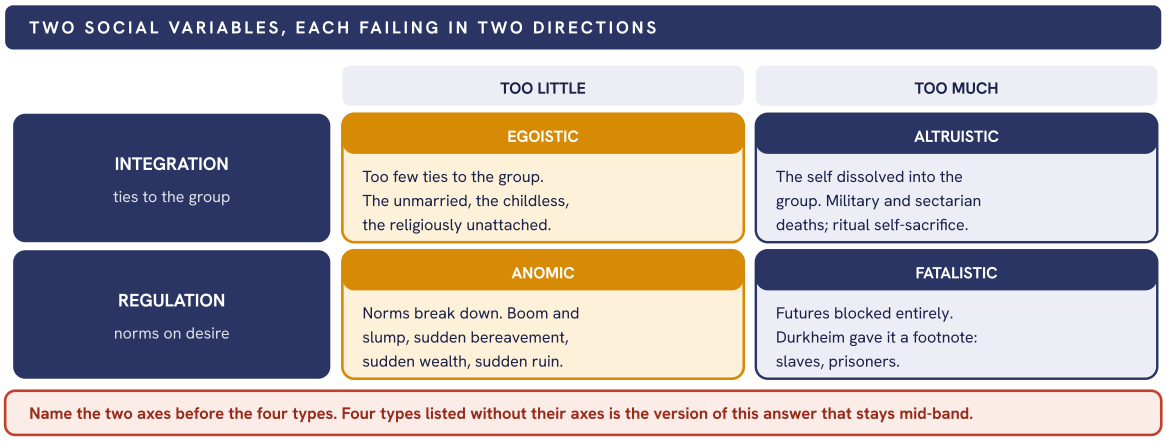


Fig 066.1 Durkheim's typology drawn as one grid: two social variables down the side, too little and too much across the top. Amber marks the two types that carry most of the modern evidence.

AFTER DURKHEIM	THE CLAIM	WHOSE
Status integration	Rates fall where a person's statuses are compatible and role conflict is low	Gibbs and Martin
The statistics critique	Official suicide rates are produced by coroners, so they are social constructions rather than raw facts	Douglas, 1967
Ecological studies	Suicide clusters in socially disorganised districts, whoever lives in them	Sainsbury, 1955
Media contagion	Reported suicides are followed by a rise, the Werther effect; responsible reporting reverses it, the Papageno effect	Phillips, 1974; Niederkrotenthaler, 2010

- **The limit, which is the last two marks.** Social theories explain rates, not individuals. They cannot say which patient will die, and an answer that offers Durkheim as a clinical risk assessment has mistaken the level of explanation.

WHAT EARNS THE MARKS

- **The two axes named before the four types,** and fatalistic included, since it is the type most often dropped.
- **At least two theories after 1897,** with their authors, since the stem says theories rather than Durkheim.
- **The level-of-explanation limit,** stated rather than implied.

SEE ALSO Slot I - 65 marriage as an institution SPRINT Day 5, social, learning and personality psychology

SOCIETY
PEOPLE
IDEAS
CONTEXT

Theories of Suicide

Historical foundations → **Émile Durkheim** → **later sociology** → contemporary psychological models

DIFFERENT
PERSPECTIVES
A DEEPER
UNDERSTANDING

Early history — 19th century — **1897** — 20th century — 21st century → *Ideas evolve. New questions emerge.*

BEFORE DURKHEIM - historical context

Earlier eras: moral and religious interpretations; historical beliefs, not clinical explanations.

1838 · Jean-Étienne Dominique Esquirol
medical and psychological explanations, shifting attention toward mental illness and emotional distress.

1879 · Enrico Morselli
comparative statistics of suicide across populations; early demographic and social analysis.

These were precursors, not modern validated prediction models.

1897 · ÉMILE DURKHEIM

Suicide rates as social facts: patterns linked to integration and regulation.

	TOO LITTLE (low levels)	TOO MUCH (high levels)
INTEGRATION → social belonging	**EGOISTIC** weak social ties and belonging	**ALTRUISTIC** individual interests subordinated to the group
REGULATION → social rules	**ANOMIC** disruption of norms during major social change	**FATALISTIC** oppressive control and a blocked future

Memory: belonging → egoistic / altruistic; rules → anomic / fatalistic.
Explains population patterns; does not identify which individual will act.

AFTER DURKHEIM - SOCIOLOGICAL DEVELOPMENTS

1955 · Peter Sainsbury
social ecology: neighbourhood isolation and social disorganisation.

1964 · Jack Gibbs & Walter Martin
status integration: incompatible social roles and weak integration.

1967 · Jack Douglas
social meanings: official statistics reflect how deaths are interpreted and classified.

1974 · David Phillips
Werther effect: certain media portrayals can contribute to imitation.

2010 · Thomas Niederkrotenthaler
Papageno effect: coping and recovery narratives can have protective effects.

CONTEMPORARY MODELS - SUICIDAL THOUGHTS AND ACTIONS HAVE PARTLY DIFFERENT EXPLANATIONS

INTERPERSONAL THEORY

Thomas Joiner, 2005; Kimberly Van Orden and colleagues, 2010

Thwarted belongingness + Perceived burdensomeness + Hopelessness about change → Suicidal desire

Capability is a separate proposed contributor to acting on desire.

Plain language: Disconnected; believing oneself a burden.

INTEGRATED MOTIVATIONAL–VOLITIONAL MODEL (IMV)

Rory O'Connor, 2011; Rory O'Connor & Olivia Kirtley, 2018

Pre-motivational background vulnerability and life events. → **Motivational** defeat / humiliation → entrapment → suicidal ideation. → **Volitional** separate moderators influence transition to behaviour.

Plain language: Feeling defeated and unable to escape.

THREE-STEP THEORY (3ST)

E. David Klonsky & Alexis May, 2015

1. **Pain** + hopelessness → ideation.
2. Ideation strengthens when pain outweighs connectedness.
3. **Capability** helps explain progression toward action.

Plain language: Pain, connection, and the thought–action distinction.

Exam focus: social theories = Durkheim + later sociology. Modern psychological models add individual mechanisms. Evidence varies; no theory alone predicts an individual outcome.

Arrows = proposed relationships, not inevitable stages.

Sources: Durkheim 1897; Gibbs & Martin 1964; Van Orden et al. 2010; O'Connor & Kirtley 2018; Klonsky & May 2015.

Theories of Suicide. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-66.

Credits and references: retained within the illustration.

I - 67

Role of culture in Psychiatry: [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The claim	Culture at every step, not only at diagnosis
4 The pathway	Each step, with its clinical consequence
2 The instrument	The cultural formulation, and what it asks
2 The two errors	Category fallacy and stereotyping

THE CLAIM TO OPEN WITH

Culture enters at every step of the clinical pathway, not only at the point of diagnosis. It shapes what counts as illness, how distress is spoken, who is consulted first, what treatment is accepted, and what outcome follows.

STEP	WHAT CULTURE DOES THERE	THE CLINICAL CONSEQUENCE
Aetiology	Alters exposure to risk and protection: family structure, migration, discrimination, drinking norms	Risk profiles differ between populations
Expression	Supplies the idioms of distress, and the content of delusion and hallucination	Somatic presentation is the rule in much of Indian practice
Help-seeking	Sets the pathway: faith healer, family elder, general practitioner, then psychiatrist	Long untreated intervals; the first contact is rarely psychiatric
Diagnosis	Decides whether a behaviour reads as normal, deviant or ill	**Risk of category fallacy; the Cultural Formulation Interview exists to check it**
Treatment	Governs adherence, and who makes the decision	Consent and disclosure are family matters more often than individual ones
Outcome	Shapes stigma, family tolerance, and re-entry to work and marriage	The WHO comparative studies reported a better course in developing-country centres

- **The instrument to name.** DSM-5 carries a Cultural Formulation Interview: the cultural definition of the problem, its perceived causes, supports and stressors, cultural factors in coping and past help-seeking, and factors in current help-seeking including the clinician relationship. Its predecessor is the DSM-IV Outline for Cultural Formulation.
- **Two errors, named.** The category fallacy is applying a category built in one culture where it has no coherence. Cultural stereotyping is reading a person as a specimen of a culture instead of asking what this person believes. The second is the commoner clinical error.

WHAT EARNS THE MARKS

- **A pathway, not a list of topics.** The structure is what turns six observations into an argument.
- **The formulation instrument named,** with at least three of the domains it asks about.
- **Both errors, kept apart.** One is a nosological error, the other is an interviewing error.

SEE ALSO Slot I - 69 cultural factors in expression, assessment and treatment Slot I - 63 culture-bound syndromes

SPRINT Day 5, social, learning and personality psychology

I - 68

Religion and Psychiatry. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The two frames	Religion as a variable, and as a context
4	The evidence	Association, coping, and the boundary tests
2	The boundary	When belief becomes a clinical concern
2	The Indian setting	Faith healing, and how to work with it

DOMAIN	WHAT IS ESTABLISHED	WHAT TO DO WITH IT
Religiosity and outcome	Religious involvement is associated with less depression, less substance use and lower suicide rates; the effects are modest and confounded	Ask about it as a resource, not only as a symptom
Religious coping	Pargament separated positive coping, collaborative and benevolent, from negative coping, punishing God and spiritual struggle	**Negative coping predicts worse outcome; the kind matters, not the amount**
Belief against delusion	A belief is not delusional because it is religious; the test is whether the person's own community shares it and holds it in that manner	The community is the reference, not the clinician
Obsession against devotion	Religious obsessions are intrusive, resisted and unwanted; devout practice is none of these	Ask about resistance and distress, not content
Possession and trance	Classified as dissociative states when involuntary, distressing and outside accepted ritual	Involuntary and unwanted is the threshold

- **The two frames to name at the start.** Religion as a variable, studied for its association with outcome, and religion as a context, which sets what a community counts as normal. An answer with only the first is epidemiology; an answer with only the second is anthropology.
- **The error runs in both directions.** Reading every religious experience as pathology, and refusing to read any of it as pathology. Both substitute a prior position for an assessment.

THE INDIAN SETTING

Temple and dargah healing is a common first contact and often runs in parallel with treatment. Collaboration is more useful than confrontation: attacking the belief system costs the alliance and the adherence, and the family usually holds both frames at once without difficulty.

WHAT EARNS THE MARKS

- **The two frames separated in the opening lines.** It is what organises everything after.
- **The community test for delusion,** stated as a test rather than as a caution.
- **Positive and negative religious coping distinguished.** It is the finding examiners look for.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway SPRINT

Day 5, social, learning and personality psychology

I - 69

Discuss in detail cultural factors involved in expression, assessment and treatment of mental illness in an individual. Add a note on immigration and mental health. [10]

10 | ASKED AT 15 MARKS | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Expression	Idioms of distress, and what culture supplies
3	Assessment	Whose normality, and the formulation instrument
3	Treatment	Explanatory models, family, and adherence
2	Immigration	The excess, and the mechanism proposed

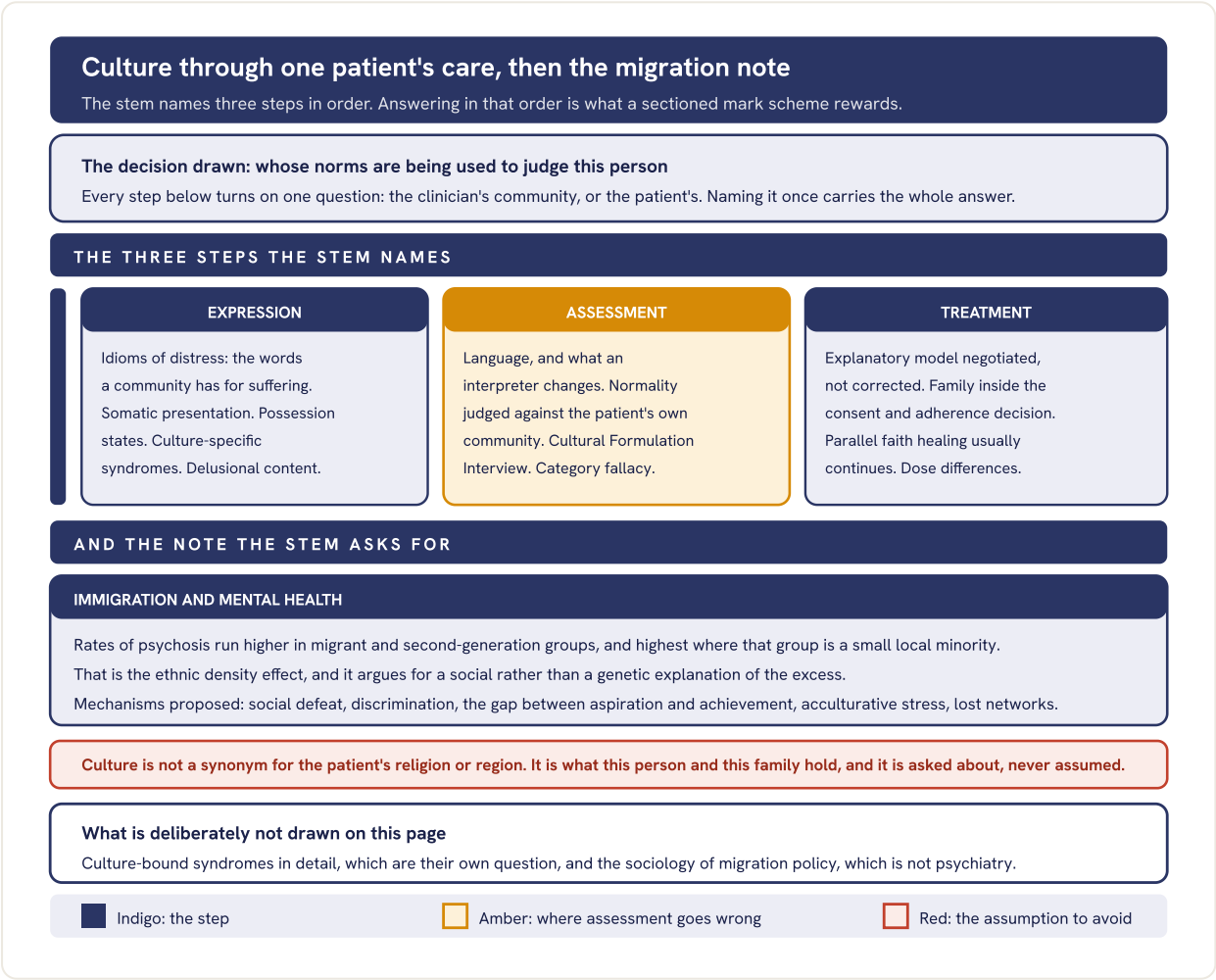


Fig 069.1 The three steps in the stem's own order along one spine, with the migration note as its own band below. Amber marks assessment, where the category fallacy is made.

- **Answer in the stem's order.** Expression, assessment, treatment, then the note. A fifteen-mark version of this question is marked by section, so a well-informed answer in the wrong order still loses marks it has earned.
- **The ethnic density effect, stated carefully.** Rates rise as the proportion of a migrant's own group in the local population falls. It is among the more robust findings in social psychiatry, and it is the sentence that turns a list of migration stressors into an argument.

WHAT EARNS THE MARKS

- **Three sections in the stem's order,** each headed, because the mark scheme is sectioned.
- **The formulation instrument named at the assessment step,** which is where its marks sit.
- **Migration answered as a mechanism,** not as a list of hardships.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway

Slot I - 70 culture, illness behaviour and symptomatology **SPRINT** Day 5, social, learning and personality psychology

I - 70

Define culture. Describe the role of culture in shaping illness behaviour and symptomatology in mental disorders. [2+8]

2 + 8 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Define culture**	Learned, shared, transmitted, changing
4	**How culture acts**	The six effects, named as mechanisms
2	**Illness behaviour**	Mechanic and Parsons, and what each adds
2	**Symptomatology**	What is shaped, and what is not

THE DEFINITION

Culture is the shared, learned and transmitted system of meanings, values and practices by which a group interprets experience. It changes, and it is not inherited.

EFFECT	WHAT CULTURE DOES TO THE DISORDER	EXAMPLE
Pathogenic	Culture causes the disorder by supplying the belief	**Koro and dhat: the belief is the illness**
Pathoselective	Culture selects which reaction is chosen under stress	Amok; family suicide in some societies
Pathoplastic	Culture shapes the content of a universal disorder	Delusional content: technology or spirits
Pathoelaborating	Culture intensifies a universal behaviour into an extreme form	Trance elaborated through ritual
Pathofacilitative	Culture raises the frequency without changing the form	Alcohol use disorders where drinking is normative
Pathoreactive	Culture governs the reaction to the illness and what it is called	Stigma; a psychosis named as possession

The six are Tseng's. Amber marks the only row where culture is a cause, not a shaper.

- **Illness behaviour, defined and attributed.** Mechanic's term for the way symptoms are perceived, evaluated and acted upon, which varies between cultures independently of the disease. Parsons' sick role sets what a society exempts the sick person from, and what it asks in return: to seek help, and to want to get well.
- **The eight marks are the mechanisms, not the examples.** Name the effect, then attach the example. An answer built from colourful syndromes with no typology behind them stays mid-band however many it lists.

WHAT EARNS THE MARKS

- **A definition with all four properties.** Learned, shared, transmitted, changing. Two marks in one sentence.
- **The effects named as effects,** in the technical vocabulary, with one example each.
- **Illness behaviour attributed to Mechanic,** separated from the sick role, and one line on what culture does not change.

SEE ALSO Slot I - 63 culture-bound syndromes Slot I - 69 cultural factors in expression, assessment and treatment

SPRINT Day 5, social, learning and personality psychology

Conformity, compliance and obedience

Conformity

Adjust behaviour or judgement to a group norm; no explicit request required.



Echo the team's view

Compliance

Agree to a request; private agreement is not necessary.



Agree when a colleague asks

Obedience

Follow a direct order from a perceived authority.



Follow a senior's instruction

Here compliance means response to a request; public compliance can also describe outward conformity without private acceptance.

Solomon Asch: line judgements, 1950s





Correct match: C



Confederates = people working with the experimenter

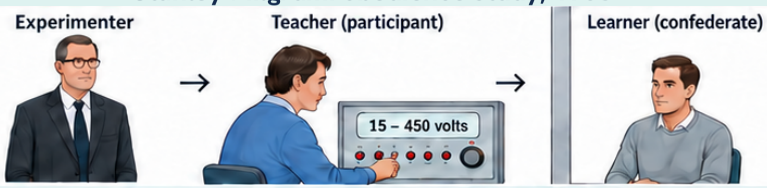
An obvious perceptual task; a unanimous majority deliberately gave wrong answers.

Findings: About one third of critical-trial responses conformed; roughly three quarters conformed at least once.

Responses ≠ participants

Dissenting ally and private responding reduced conformity; unanimity, task ambiguity and group relevance can increase it.

Stanley Milgram: obedience study, 1963



The participant believed shocks were delivered; the learner received none.

Finding: 26 of 40 men (65%) reached 450 volts in the published condition.

A result from one condition, not a universal obedience rate.
Other conditions: learner proximity, authority presence/legitimacy and disobedient peers changed obedience.

Agentic-state account: feeling an instrument of authority,
An interpretation, not a directly proven mechanism.

Why people conform

Normative influence: seek acceptance or avoid rejection.

Informational influence: use others as evidence about reality.

Muzafer Sherif: ambiguous apparent movement of a stationary light → shared estimates.

Both motives may coexist; public answers alone do not prove private belief changed.

Compliance techniques

Foot-in-the-door: small request → larger request.

Door-in-the-face: large request refused → smaller request.

Low-ball: initial agreement → later added cost.

Effects depend on context; these are influence techniques, not ethical recommendations.

Ethics and limitations

Asch: deception, social pressure and restricted student samples.

Milgram: deception, substantial distress and pressure conflicting with free withdrawal.

Artificial tasks, historical context and participant beliefs limit generalisation.

Obedience is not evidence of agreement, lack of distress, or an unchangeable personality.

Clinical takeaway: Invite dissent; separate authority from evidence; protect the right to refuse.

Conformity, compliance and obedience. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Group processes

A group involves interaction, interdependence and shared identity. **Norms** = shared **expectations**; **roles** = expected conduct; cohesion = bonds between members.

Social facilitation - Robert Zajonc

Presence of others → arousal → stronger dominant response

Dominant response = the most likely response.



Well-learned task: dominant response often correct → **better performance.**

Novel or difficult task: dominant response often wrong → **worse performance.**

A classic explanation; evaluation concerns, attention and task demands also matter.

Social loafing

Reduced individual effort when working collectively, especially when contributions are hard to identify.



Pooled output + low identifiability
↓
lower accountability
↓
reduced effort

Max Ringelmann: rope-pulling; later work separated motivational and coordination losses.

Reduce it: identifiable contributions, meaningful tasks, clear roles, feedback and shared commitment.

Loafing = motivational loss. Poor coordination is a different process.
It is not simply the opposite of social facilitation.

Group polarisation

Discussion shifts members towards a more extreme version of their initial tendency.

Initially cautious → **More cautious after discussion**

Initially risk-tolerant → **More risk-tolerant after discussion**

Mechanisms

- **Persuasive arguments:** hear more reasons for the favoured side.
- **Social comparison and identity:** align with the valued group position.



A cautious committee discusses risk and becomes still more cautious.

Risky shift is one possible direction; polarisation is not always increased risk.

Groupthink - Irving Janis

The drive for agreement undermines critical appraisal and consideration of alternatives.



Risk conditions: insulation, directive leadership, stress and weak decision procedures; cohesion alone is insufficient.

Common symptoms

Overestimation: invulnerability; belief in group morality.

Closed-mindedness: rationalisation; stereotyping opponents.

Uniformity pressure: self-censorship; illusion of unanimity; pressure on dissenters; mindguards.

Mindguards = members who screen out conflicting information.

Prevent: leader speaks last, independent opinions, outside critique, devil's advocate and a second review.

Facilitation = audience and performance • **Loafing** = effort in pooled work • **Polarisation** = direction of opinion shift • **Groupthink** = quality of decision process.

Groupthink grew from retrospective case analyses; its causes and measures are debated. Agreement, cohesion or a bad outcome alone does not establish groupthink.

Group processes. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Aggression

Behaviour intended to harm someone who is motivated **to avoid that harm.**

Aggression = behaviour | **Anger** = emotion | **Hostility** = attitude | **Assertiveness** = expressing needs without intending harm.

Reactive vs instrumental aggression

Reactive aggression: response to perceived threat or provocation; often impulsive and emotionally aroused.



Example: Shouting after an insult.

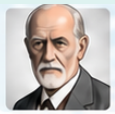
Instrumental aggression: harm used as a means to another goal; often planned.



Example: Intimidating someone to obtain money.

Motives may overlap; these are dimensions, not diagnoses.

Historical explanations



Sigmund Freud — Historical
death-drive account: aggression as destructive drive.
Historical, difficult to test.



Konrad Lorenz — Evolutionary
ethological account: evolved aggressive tendencies; hydraulic energy model.
Animal-to-human extrapolation and inevitability are limited.



John Dollard, Neal Miller and colleagues, 1939 — Psychological
frustration–aggression hypothesis: blocked goals generate aggression.
The original strong claim is not supported: **frustration** can lead to many responses.

Leonard Berkowitz: cognitive neoassociation

Aversive event → **negative affect** → **aggression-related thoughts and action tendencies**



Cues, interpretation and self-control influence what follows.

Frustration is one aversive input; it does not inevitably cause aggression.

Albert Bandura: social learning



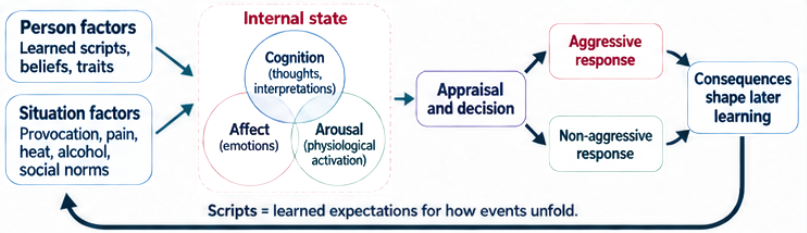
Adult demonstrates aggressive actions towards an inflatable doll.

Child observes from a distance.

Later imitates with a doll.

- **Bobo-doll studies, 1961:** children exposed to an aggressive model reproduced more modelled aggressive acts.
- Observation → learning; expected consequences influence performance.
- **Vicarious reinforcement** = seeing a model rewarded.
- Short-term imitation towards a toy does not by itself establish enduring interpersonal violence. Restricted samples and task expectations matter.

Craig Anderson and Brad Bushman: General Aggression Model



An influential integrative framework; no single factor determines an individual's behaviour.

Situational influences and limits

Interpretation of intent: stress and frustration; intoxication; available models; peer reinforcement; accountability.

Biology and experience interact; there is no single aggression gene, chemical or brain centre.

Group-level associations are not individual prediction.

Catharsis myth: aggressive venting does not reliably drain anger and can sustain it.
Calm reflection, reducing arousal and non-aggressive coping are different.

Psychiatric diagnosis alone does not establish dangerousness.

Aggression. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Crowd and mob psychology

Crowd: people gathered in a shared setting. **Mob:** a crowd engaged in collective aggression or disorder. Most crowds are peaceful; membership alone does not imply irrationality.

Classical accounts — historical theories

Gustave Le Bon (1895)
anonymity + suggestibility + contagion → proposed loss of individual judgement.

Historically influential; elitist assumptions and weak systematic evidence.

Sigmund Freud (1921)
identification with a leader and fellow members; shared emotional bonds.

Psychoanalytic interpretation, not an established universal mechanism.



Deindividuation: changing self-awareness

Leon Festinger, Albert Pepitone and Theodore Newcomb (1952)
reduced individuation in groups.

Philip Zimbardo
anonymity, arousal and reduced accountability may loosen restraints.

Edward Diener
reduced self-awareness was proposed as a mechanism.



Effects depend on norms and context; anonymity does not inevitably cause aggression.

Emergent norms

Ralph Turner and Lewis Killian
in ambiguous situations, interaction helps establish local rules.



Collective behaviour can be organised and meaningful, even without advance planning.

Social identity perspectives

Stephen Reicher
people may shift from personal identity ("I") to a shared social identity ("we"); group norms guide action.

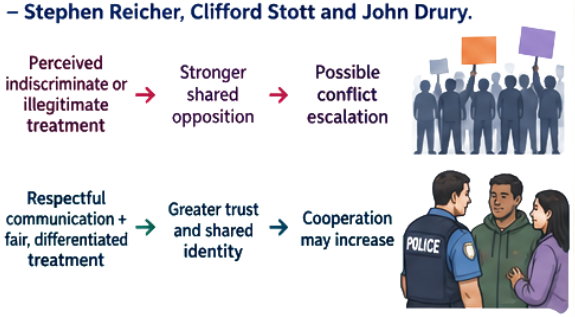
Social Identity Model of Deindividuation Effects – Stephen Reicher, Russell Spears and Tom Postmes
anonymity can strengthen the influence of a salient group identity.



Evidence challenges a simple loss-of-control account; no single theory explains every crowd.

Intergroup dynamics and escalation

Elaborated Social Identity Model – Stephen Reicher, Clifford Stott and John Drury.



This is an interactional account, not a claim that all disorder has one cause.

Evidence, limits and practical implications

- Field observations, experiments and case studies each have limits: context, selection, reconstruction and causal inference.
- Shared identity can support solidarity, mutual aid and self-organisation in emergencies.
- Crowd crush can result from dangerous density and restricted flow; do not automatically label it mass panic.
- Plan safe capacity and exits; communicate clearly, support local cooperation and avoid indiscriminate confrontation.

EXAM DISTINCTIONS | Contagion ≠ proof of irrationality • Deindividuation ≠ no identity • Crowd ≠ violent mob • Crowd crush ≠ necessarily panic

Crowd and mob psychology. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Psychological aspects of sexual violence

Sexual violence involves sexual acts or attempts obtained through coercion or without valid consent. Responsibility lies with the perpetrator.

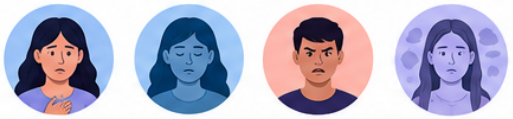
Social context and explanatory models



Motives and pathways are heterogeneous: power, hostility, sexual motives, opportunity and social norms can interact. No single offender profile explains all cases.

Risk associations are not proof of causation or accurate individual prediction. Alcohol never removes responsibility.

Trauma responses vary



Fear, shock, anger or numbness

Freezing or tonic immobility = involuntary inability to move or respond

Dissociation = altered sense of self, surroundings or awareness



Intrusions, avoidance, hyperarousal, sleep disturbance and shame may follow.

Calmness, fragmented recall or delayed disclosure can occur.
None alone establishes or disproves assault.
Absence of resistance does not equal consent.

Historical exam construct

Ann Wolbert Burgess and Lynda Lytle Holmstrom (1974): rape trauma syndrome.

Acute disorganisation



Overt distress OR a controlled, composed presentation.

Reorganisation



Ongoing adjustment; intrusive memories, avoidance and relationship changes may occur.

Historical descriptive framework, not a mandatory sequence or a standalone contemporary psychiatric diagnosis.
Recovery is individual and often non-linear.

Myth → corrective fact

Only strangers assault	→	Partners and acquaintances may perpetrate violence.
No injury or no struggle means consent	→	Injury and resistance are not required to establish lack of consent.
Clothing, drinking or past sex caused it	→	None transfers responsibility to the survivor.
A 'real' survivor reacts one way	→	Responses and disclosure patterns vary.

Victim-blaming and disbelief can cause secondary victimisation: further harm from other people's or institutions' responses.

First-line support - World Health Organization

L **Listen** with empathy, without pressure.

I **Inquire** about needs and concerns.

V **Validate** the experience; do not blame.

E **Enhance** safety collaboratively.

S **Support** access to chosen services and follow-up.



Explain confidentiality and its limits; seek consent at each step.
Avoid repeated unnecessary retelling or forced emotional debriefing.

Clinical implications

- Assess immediate safety, distress, self-harm risk and practical needs.
- Coordinate timely medical, psychological and forensic options with informed consent and applicable safeguarding duties.
- Assess for acute stress disorder, post-traumatic stress disorder, depression, substance problems and sexual difficulties when indicated.
- Offer evidence-based trauma-focused treatment when clinically appropriate; arrange continuity and social support.
- Distress does not always become a disorder. Avoid diagnosing from a single reaction or judging credibility from demeanour.

Consent ≠ absence of resistance • Trauma response ≠ credibility test • Historical syndrome ≠ inevitable course • Support restores choice and control

Psychological aspects of sexual violence. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Rumour

1. Definition and related concepts

A rumour is a circulating claim relevant to a current situation whose truth is not yet verified. It may ultimately be true or false.

Related but distinct concepts:

Term	Key features	Example
Rumour	Unverified claim circulating in a population. May ultimately be true or false.	"There might be a change to the exam timetable." (uncertain and unverified)
Gossip	Often social or personal talk about others. May or may not be true.	"I heard they're going out together." (personal and social)
Misinformation	False information, irrespective of intent.	"The exam is cancelled." (passed on in good faith, but false)
Disinformation	Deliberately deceptive information, shared with an intent to mislead.	"The exam is cancelled." (knowingly false, shared to cause disruption)

2. Allport and Postman's heuristic

Gordon Allport and Leo Postman proposed that rumour intensity is approximately related to the importance of the topic and the ambiguity of the information.

Rumour intensity ≈ Importance × Ambiguity

(conceptual heuristic, not a validated numerical prediction or a universal equation)

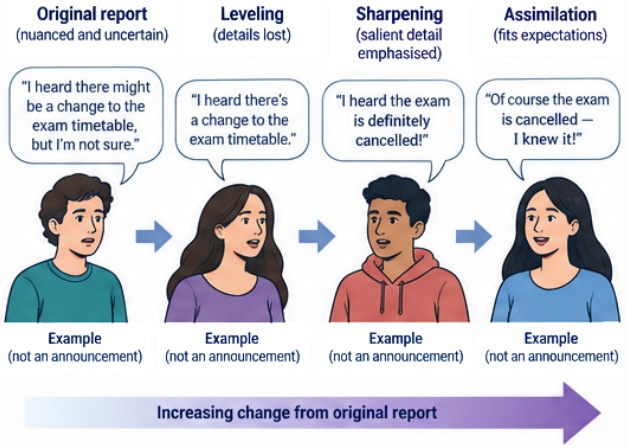
Greater importance
- more relevant to people's goals, safety or wellbeing
- affects larger numbers
- higher perceived stakes

Greater ambiguity
- unclear, incomplete or conflicting information
- rapidly changing situations
- lack of authoritative sources

Higher rumour intensity (more likely to spread)

3. Serial transmission

As a rumour is repeated, it often changes in predictable ways described by Allport and Postman.



4. Types, reasons for spread and containment

Robert Knapp's types (historical)

Robert Knapp described three broad types of rumour.

Wish or pipe-dream rumour
Reflects hopes and desires (e.g. good news people would like to be true).

Fear or bogie rumour
Reflects fears and anxieties (e.g. threatening or harmful possibilities).

Wedge-driving rumour
Aims to create division, hostility or conflict between groups (e.g. about an out-group).

Why rumours spread

Uncertainty
People seek explanations when information is lacking.

Anxiety
Rumours can reduce anxiety temporarily by providing a narrative.

Information gaps
Lack of timely, clear and trusted information.

Social trust
More likely to be shared within trusted networks and in-group members.

Identity and group belonging
Sharing can signal group membership and shared attitudes.

Repeated exposure
Familiarity increases belief. Repetition is not verification.

Containing rumours

Acknowledge uncertainty
Be honest about what is not yet known.

Provide timely, specific evidence
Share clear information from an accountable and authoritative source.

Explain what is known and unknown
Set out the current position and what is being investigated.

Update transparently
Provide regular updates as new information becomes available.

Correct without shaming
Address false claims respectfully to maintain trust.

Avoid amplifying unsupported claims
Do not repeat rumours unnecessarily, especially through wide or official channels.

5. Rumour versus delusion

Social circulation of a claim does not, on its own, establish a clinical delusion. Assess the individual's belief and context.

Feature	Rumour	Delusion
Nature	Circulating claim within a social group	Individual, fixed false belief
Scope	Shared by multiple people	Typically held by one individual (though may occur in shared delusional disorder)
Basis	Not yet verified; may be true or false	False belief, not consistent with evidence
Change with evidence	May be modified or discarded as new information emerges	Typically resistant to contradictory evidence
Context	Arises in a social and situational context	Consider within a clinical and personal context
Implication	A widely circulated rumour does not indicate a clinical delusion	Requires clinical assessment of the individual's belief, functioning and context

The presence of a rumour in the community should not be taken as evidence that individuals who believe it have a delusional disorder.

Rumour. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Prosocial behaviour and helping

Prosocial behaviour: voluntary action intended to benefit another.

Helping: assistance to someone in need.

Altruism: helping motivated by concern for the other's welfare, rather than self-gain.

Behaviour is observable; motive is harder to establish. Mixed motives are possible.

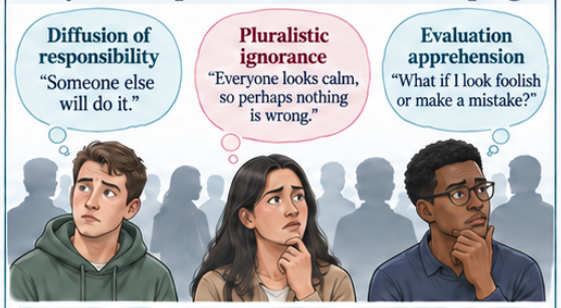
Bibb Latané and John Darley: decision model



5. **Act** — danger, costs and fear of judgement may inhibit action.
4. **Know how to help** — skills and confidence matter.
3. **Take responsibility** — someone else may seem responsible.
2. **Interpret as needing help** — ambiguity may obscure the emergency.
1. **Notice** — distraction or hurry may interfere.

A useful teaching model; real helping need not follow a rigid conscious sequence.

Why others' presence can inhibit helping



Bystander effect: the presence of others can reduce an individual's probability or speed of helping.

Not universal: danger, shared identity, skills and supportive others can change or reverse the pattern.

An individual's chance of helping is different from the chance that ANYONE in the crowd helps.

Why people help: competing accounts

- **Daniel Batson — empathy–altruism:** empathic concern can motivate other-directed help.
- **Robert Cialdini — negative-state relief:** helping can reduce one's own distress.
- **Social exchange:** perceived rewards and costs shape helping.
- **Empathy studies support altruistic motivation,** but motives are difficult to isolate.

Evolutionary explanations

- **William Hamilton: kin selection** helping relatives can favour shared genetic interests.
- **Robert Trivers: reciprocal altruism** repeated exchanges can support helping non-relatives.
- **Ultimate explanation:** why a tendency evolved. **Proximate explanation:** the person's immediate psychological motive.
- Evolutionary function does not imply conscious genetic calculation.

Classic experiments

John Darley and Bibb Latané, 1968: a simulated seizure heard over an intercom, believing other witnesses were present reduced or delayed reporting.

Bibb Latané and John Darley, 1968: smoke-filled room; passive others could inhibit reporting.

Deception, artificial emergencies and restricted samples limit generalisation.

Factors that support help

Clear need · Empathy · Relevant skills · Shared identity · Helpful models · Manageable danger · Explicit responsibility



Be specific, coordinate safely and act within competence.

Inaction need not mean indifference. Do not repeat the disputed story that 38 witnesses simply watched the entire Kitty Genovese attack.

Prosocial behaviour and helping. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

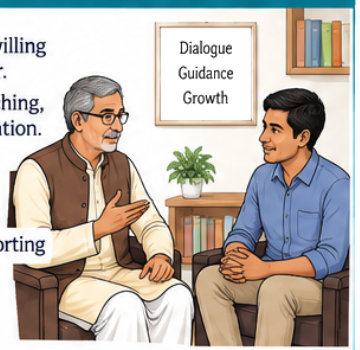
Guru-chela relationship

Guru = teacher or guide • Chela = disciple or learner

Jaswant Singh Neki (1973) proposed this traditional relationship as a possible culturally informed therapeutic paradigm.

Traditional model — Neki's account

- Trust, reverence and willing reliance on the teacher.
- Guidance through teaching, example and identification.
- A warm, personal, hierarchical bond.
- The teacher accepts responsibility for supporting the learner's growth.



A conceptual cultural model; traditions and individuals vary.

Why it matters in therapy

Expectations of guidance → **feeling understood and supported** → **possible trust and engagement** → **learning and greater agency**

- Some patients welcome an active, explanatory therapist.
- A wish for direction may reflect cultural expectations, distress or uncertainty.
- Ask each person's preferences; do not infer them from nationality or religion.



Transference, identification and dependence

Transference:	feelings and expectations from important relationships become directed towards the therapist.
Identification:	adopting valued qualities or coping strategies from a model.
Idealisation:	viewing the therapist as exceptionally wise or powerful.

Neki treated willing dependence and idealisation as potentially understandable phases. They require thoughtful assessment, not automatic pathologising or uncritical acceptance.

Use trust to strengthen the patient's capacity to choose.

Adapt the strengths; retain clinical safeguards

Traditional emphasis	Therapeutic adaptation
Revered guide	Warm, competent, accountable clinician
Personal authority	Explain options and share decisions
Teacher's example	Model reflection and coping
Willing reliance	Support agency; review dependence
Broad personal bond	Agreed professional roles and boundaries

Guidance can be collaborative. Cultural responsiveness does not remove informed consent.

Power and boundaries

- Discuss goals, roles, fees, confidentiality and limits.
- Welcome questions, disagreement and second opinions.
- Monitor idealisation, overdependence and the therapist's wish to be admired or needed.
- Prevent coercion, exploitation and sexual or financial boundary violations.
- Use supervision; plan endings and support beyond therapy.



Accountability + autonomy

SAFE RELATIONSHIPS
ETHICAL PRACTICE
BETTER OUTCOMES

Clinical example and limitations



- Historical proposal, not evidence that this approach is superior for all Indian patients.
- India is culturally diverse; authority and autonomy are not mutually exclusive, nor neatly divided into East and West.
- Do not impose spirituality, demand obedience or take on an infallible guru role.

Understand the expectation → negotiate the relationship → preserve consent and boundaries → build autonomy.

Guru-chela relationship. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Culture and Psychiatry

Questions 69–70 • Expression → Assessment → Treatment • Cultural effects

INDIVIDUALISTIC ORIENTATION: personal goals, independence, self-expression and privacy.

COLLECTIVISTIC ORIENTATION: interdependence, family obligations, group harmony and shared decisions.

Dimensions, not fixed categories. People may value both; **variation within societies is substantial. Ask about this person—never infer beliefs from nationality.**

EXPRESSION

How is distress experienced and communicated?

Idioms of distress: locally meaningful words, bodily complaints or relational difficulties.

Culture can shape symptom content; content alone does not establish a diagnosis.

Individualistic emphasis may frame distress around personal identity, achievement or autonomy.

Collectivistic emphasis may frame distress around family conflict, duty, belonging or reputation.

Emotional restraint or disclosure depends on audience, context, gender and generation.

Example: The same difficulty may be described as 'I feel hopeless' or 'My body feels exhausted' or 'I cannot fulfil my family role'. These are expressive forms—not diagnostic equivalents.

ASSESSMENT

Whose meaning and norms are being used?

Explore language, identity, explanatory beliefs, stressors, supports and help-seeking.

Use a trained interpreter when needed; check that translated questions retain their meaning.

Ask about cultural context, distress, impairment and the overall clinical picture.

Individualistic emphasis: explore privacy, personal goals and desired independence.

Collectivistic emphasis: explore family roles, support, expectations and who the person wants involved.

Arthur Kleinman's category fallacy: applying a diagnostic category without checking its meaning in the local context.

Example: Ask: 'What do you call the problem? What do you think caused it? What does your family think? What help would fit your needs?'

TREATMENT

What care fits this person's goals and circumstances?

Negotiate a shared understanding; explain treatment in familiar language and metaphors.

Individualistic emphasis: collaborative personal goals, choice, privacy and self-management.

Collectivistic emphasis: family or community support and relational goals—when the person wants them.

Autonomy can include choosing shared decisions. Family involvement does not automatically transfer decision-making authority.

Explore faith healing and other care respectfully; address harmful practices and treatment delays.

Adapt communication and delivery without abandoning effective treatment.

Example: Ask: 'Who would you like involved? What would improvement look like? What worries you about this treatment?'

OTHER DIMENSIONS TO EXPLORE: power distance and clinician authority • stigma and disclosure • religion and spirituality • gender and generation • education, poverty, caste and discrimination • migration and language.

MIGRATION NOTE: consider loss of networks, acculturative stress, discrimination and access barriers. Lower own-group neighbourhood density is associated with higher psychosis rates in some minority populations; effects vary by group and setting.

CULTURAL FORMULATION INTERVIEW (CFI): definition of the problem • perceived causes • context and support • coping and past help-seeking • current help-seeking and care preferences.

HOW CULTURE INFLUENCES PSYCHOPATHOLOGY — WEN-SHING TSENG

PATHOGENIC
Contributes to the development of distress.
Example: semen-loss beliefs contributing to Dhat-related anxiety.

PATHOSELECTIVE
Patterns the form of a stress response.
Example: Amok is a classic historical example.

PATHOPLASTIC
Shapes symptom content or presentation.
Example: delusions involving spirits or surveillance technology.

PATHOELABORATING
Amplifies or elaborates a behavioural pattern.
Example: culturally elaborated startle responses in Latah.

PATHOFACILITATIVE
Makes a condition more frequent.
Example: permissive drinking norms increasing alcohol-related problems.

PATHOREACTIVE
Shapes responses to illness.
Example: stigma, support or interpreting distress as possession.

PROTECTIVE / SALUTOGENIC EFFECTS: belonging, meaning, social support and helpful coping. Additional protective perspective—not one of the book's six terms.

Ask, don't assume. Culture shapes care; it does not replace individual assessment.

Sources: American Psychiatric Association Cultural Formulation Interview; Wen-Shing Tseng cultural effects framework; tutor synthesis.

Culture and Psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

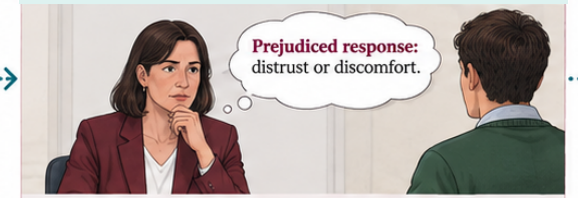
Prejudice, stereotypes and discrimination

Stereotype - belief about a group



A generalised belief about a group, often inaccurate and oversimplified.

Prejudice — evaluative attitude towards a group



A negative or positive evaluative attitude towards a group based on a stereotype, rather than individual characteristics.

Discrimination - unequal treatment based on group membership



Unequal treatment based on group membership, rather than individual merit.

Related but separable; one does not inevitably produce the next.

Stereotypes may sound positive yet erase individual differences; discrimination can be institutional as well as interpersonal.

How prejudice develops

Gordon Allport	ordinary categorisation can become rigid overgeneralisation.
Social learning	family, peers, media and institutions transmit group beliefs and norms.
Theodor Adorno and colleagues	authoritarian-personality account: submission, rigidity and displaced hostility. Historical account; measurement and causal claims are limited.
Muzafer Sherif	realistic conflict: perceived competition for scarce resources can increase hostility.
Henri Tajfel and John Turner	social identity: categorisation and group identification can foster in-group favouritism.

Scapegoating: blame a less powerful group for frustrations.
No single theory explains all prejudice.

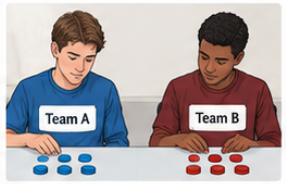
Classic findings, carefully read

Robbers Cave, 1954: competitive camp groups developed hostility; repeated cooperation towards shared goals helped reduce it.



Restricted sample and researcher-shaped setting limit generalisation.

Minimal-group studies: arbitrary group labels can produce biased reward allocations.



In-group favouritism does not necessarily imply out-group hatred.

Consequences

- **Exclusion** from housing, work and care
- **Chronic stress,** concealment and reduced help-seeking
- **Public stigma:** social devaluation
- **Self-stigma:** internalising negative beliefs
- **Structural stigma:** policies and practices restrict opportunity.



Effects vary; do not assume every person internalises stigma.

Reducing prejudice and discrimination

Gordon Allport: contact is helped by...

- Equal status within the contact setting
- Common goals
- Cooperation
- Institutional support

Thomas Pettigrew and Linda Tropp, 2006: contact generally reduces prejudice; these conditions facilitate effects, but are not strict prerequisites.
Contact is not automatically beneficial; quality, power and context matter.

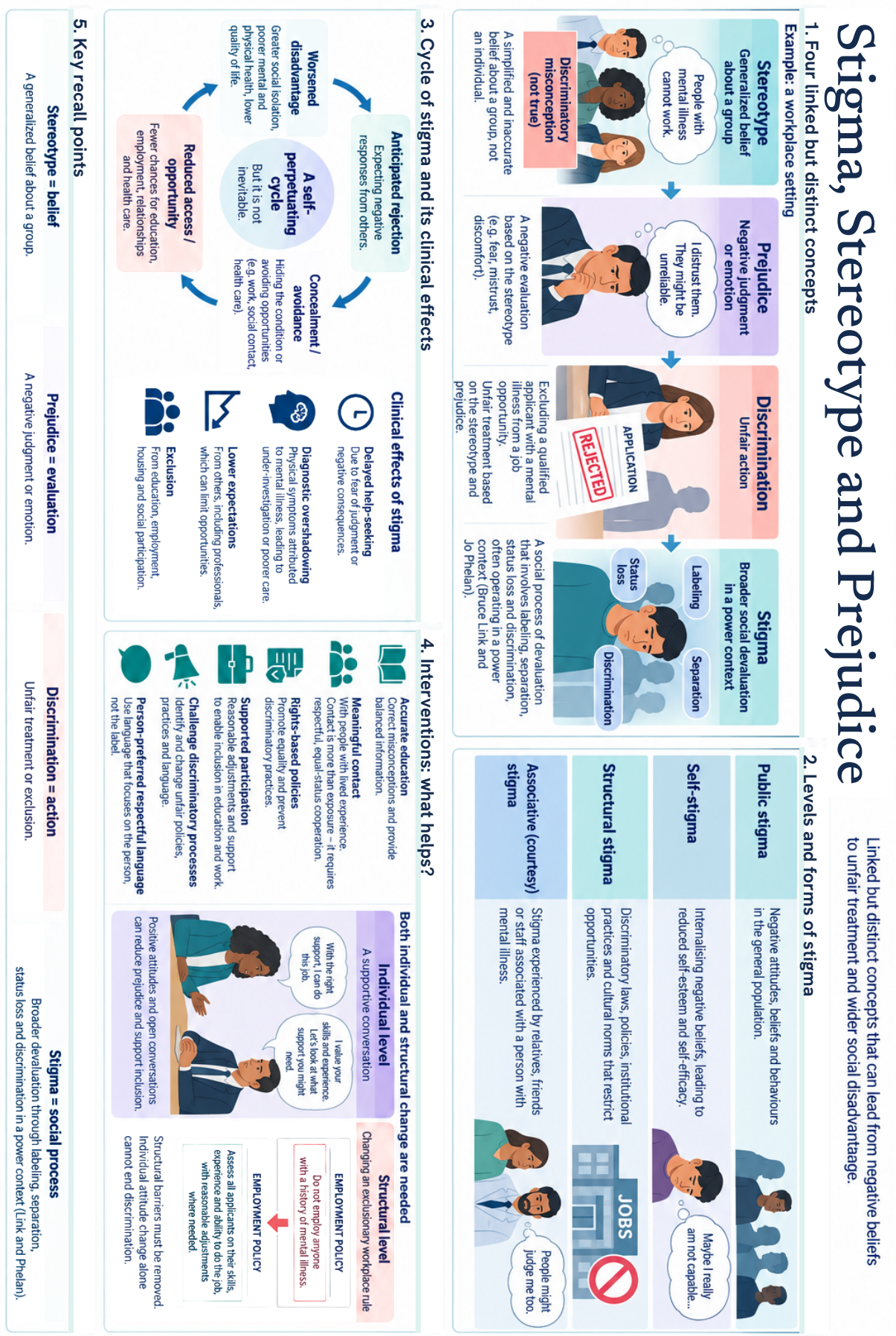
Other useful approaches

- Education and individuating information
- Perspective-taking
- Inclusive norms and accountability
- Fair policies and access
- Meaningful contact led with people with lived experience

Measure attitude change and actual treatment separately. Awareness alone may not change discriminatory behaviour.

Prejudice, stereotypes and discrimination. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.



Stigma, Stereotype and Prejudice. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

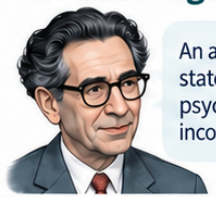


Attitudes and attitude change. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Cognitive dissonance

Leon Festinger, 1957



An aversive motivational state arising from psychologically inconsistent cognitions.

Cognitions = beliefs, knowledge, attitudes or awareness of behaviour.



Discomfort

Motivation to restore consistency

Inconsistency is psychological, not merely a logical contradiction. Important beliefs and personal commitment can amplify discomfort.

Classic experimental findings

Leon Festinger and James Carlsmith, 1959

Induced compliance



Participants did boring tasks, then told another person they were enjoyable.

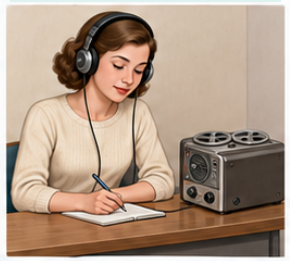
$1	$20
$1: little external justification → greater attitude change	**$20:** ample external justification → less attitude change

The $1 group later rated the task more favourably than the $20 and no-advocacy control groups.

Theory: insufficient external justification encourages internal justification.

Elliot Aronson and Judson Mills, 1959

Effort justification



An embarrassing initiation preceded listening to a dull group discussion.

"I worked hard to join, so the group must be worthwhile."

Severe initiation group later evaluated the group more positively than mild/no-initiation groups.

Jack Brehm, 1956

Post-decision dissonance

Choose between similarly rated consumer goods; rate them again.



Chosen option rated more positively

Rejected option rated less positively

Spreading of alternatives

Ways to reduce dissonance



Change behaviour → "Stop smoking."



Change a cognition → "Minimise the perceived health risk."

Examples of rationalisation, not health advice.



Add consonant cognitions → "I exercise, so I offset the harm."

Examples of rationalisation, not health advice.



Reduce importance → "Health is not my main priority."

Examples of rationalisation, not health advice.

Avoiding conflicting information or seeking reassurance may also protect an existing belief. Reduction need not improve accuracy.

Qualifications that earn marks



Classic studies used restricted student samples and artificial tasks; generalisation requires care.



Choice, commitment and available justification influence effects; a small reward does not always force attitude change.



Later work: the **traditional** free-choice design can create apparent preference change from noisy ratings; better controls are needed.



Daryl Bem: self-perception infers attitudes from behaviour without requiring discomfort, especially when prior attitudes are weak.



Dissonance ≠ ordinary disagreement, ambivalence or simple hypocrisy. Ambivalence means mixed positive and negative evaluations.



Explore a person's own discrepancy between values and behaviour collaboratively, do not assume confrontation or shame produces therapeutic change.

Cognitive dissonance. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

Attribution and attribution biases

Attribution = explaining the causes of behaviour and events



Fritz Heider, 1958: person or situation?

Internal: effort, ability, disposition

External: traffic, task, circumstances

A dispositional explanation is not automatically an error; the error is neglecting relevant evidence.

Harold Kelley: covariation model

Consensus: do other people respond similarly?

Distinctiveness: does this person respond this way only to this stimulus?

Consistency: does it recur with this stimulus over time?

Consensus	Distinctiveness	Consistency	Suggested cause
High	High	High	Stimulus
Low	Low	High	Person
—	—	Low	Temporary circumstances

Idealised cue patterns, not proof of causation.

Fundamental attribution error

Others' behaviour

Overweight disposition and underweight situational constraints.



Related: correspondence bias — infer a matching trait even when behaviour is constrained.

Actor-observer differences

Self versus other

Classic claim: my actions reflect the situation; yours reflect your disposition.



Bertram Malle, 2006: meta-analysis found no general actor–observer asymmetry of this simple form; effects depend on conditions.

Self-serving bias

Success versus failure

Take internal credit for success; explain failure externally.



Can reflect self-protection and information differences; varies across people, tasks and cultures.

Bernard Weiner: achievement attributions

Locus: internal ↔ external (Where does the cause lie?)

Stability: enduring ↔ changeable (Will it persist over time?)

Controllability: controllable ↔ uncontrollable (Can the person influence it?)

	Internal	External
Stable	**Ability** (internal/stable)	**Task difficulty** (external/stable)
Unstable	**Effort** (internal/unstable)	**Luck** (external/unstable)

Classic perceived categories; circumstances can change these classifications.

Other useful distinctions

Edward Jones and Keith Davis: freely chosen, distinctive acts inform trait inferences.

Melvin Lerner: just-world belief can encourage victim-blaming.

False consensus: overestimate how widely others share one's view.

Qualifications and clinical use

Culture shapes attention to context; avoid East–West or Indian stereotypes.

Depression may involve internal, stable, global explanations of adverse events; this is not universal or diagnostic.

Actor–observer compares **SELF/OTHER**; **self-serving** compares **SUCCESS/FAILURE**.

Before judging: check constraints, ask for missing information, compare repeated observations.

Attribution and attribution biases. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Social, cross-cultural and evolutionary psychology.

Credits and references: retained within the illustration.

AREA 08 OF 12 · P1-A11

Biostatistics and epidemiology

8	7.6%	I-71	I-78
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · RECURRENT 1 · SINGLE 6

REVISION ORDER

The two selector slots first and together: they open the other three, and the paired and unpaired t-tests belong inside them. Chi square next, it is the anchor here and it is short. Analysis of variance after that. The two odds-ratio slots are one pair and should be prepared as one. Regression stands alone and is the longest here.

WHAT IS IN THIS AREA

The tests of significance in five slots: the selector question twice, once opened out on the paired test and once on the unpaired, then chi square, the t-test and analysis of variance each alone. The odds ratio in two, once against relative risk and once with confounding, effect modification and mediation. Regression analysis takes the eighth.

I - 71

Chi Square test. [10]

10 BIOSTATISTICS AND EPIDEMIOLOGY

KNRUHS, KUHS, NTRUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 What it is	Observed against expected, for categorical data
4 The working	Expected counts, the statistic, the degrees of freedom
2 Assumptions	And the exact test that replaces it when they fail
2 Its three uses	Association, goodness of fit, homogeneity

THE DEFINITION AND THE FORMULA

A non-parametric test of whether two categorical variables are associated, built entirely on counts. The statistic is the sum over all cells of observed minus expected, squared, divided by expected. Each expected count is its row total times its column total, divided by the grand total, and the degrees of freedom are rows minus one times columns minus one.

CELL, ILLUSTRATIVE COUNTS	OBSERVED	EXPECTED	CONTRIBUTION
Adherent, relapsed	20	30	3.33
Adherent, no relapse	80	70	1.43
Not adherent, relapsed	40	30	3.33
Not adherent, no relapse	60	70	1.43
Total	200	200	Chi square 9.52, one degree of freedom

Made-up counts, arithmetic real. Row totals 100 and 100, column totals 60 and 140, so every expected count is 30 or 70. At one degree of freedom the statistic clears both the 3.84 cut at five per cent and the 6.63 cut at one per cent.

- **Assumptions.** Independent observations, raw counts and never percentages, and adequate expected counts: in a two by two no expected cell below five. When that fails, use Fisher's exact test. Paired categorical data need McNemar's test, not this one.
- **Its three uses.** Test of association between two categorical variables; goodness of fit against an expected distribution; and test of homogeneity of a proportion across several groups.
- **What it does not tell you.** A significant result says the variables are associated, not how strongly and not in which direction. Report an effect measure, an odds ratio or a relative risk, alongside it.

WHAT EARNS THE MARKS

- **The expected-count formula written out,** not just the chi square formula.
- **Degrees of freedom derived from the table shape,** with the number stated.
- **The expected-count-below-five rule with Fisher's exact test as the remedy.**
- **The three uses separated,** and McNemar named for paired data.

SEE ALSO Slot I - 74 tests of significance Slot I - 78 odds ratio and relative risk SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 72

Enumerate various statistical tests used to measure the differences. Describe the paired t-test in detail with an example. Describe any two non-parametric tests used for group difference. [4+2+4]

4 + 2 + 4 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The enumeration	Drawn as a selector, by data type and pairing
3	Paired t-test	What is tested, the statistic, an example
2	Two non-parametric	Mann-Whitney and Wilcoxon, and what each replaces
1	The choosing rule	Pairing first, then distribution

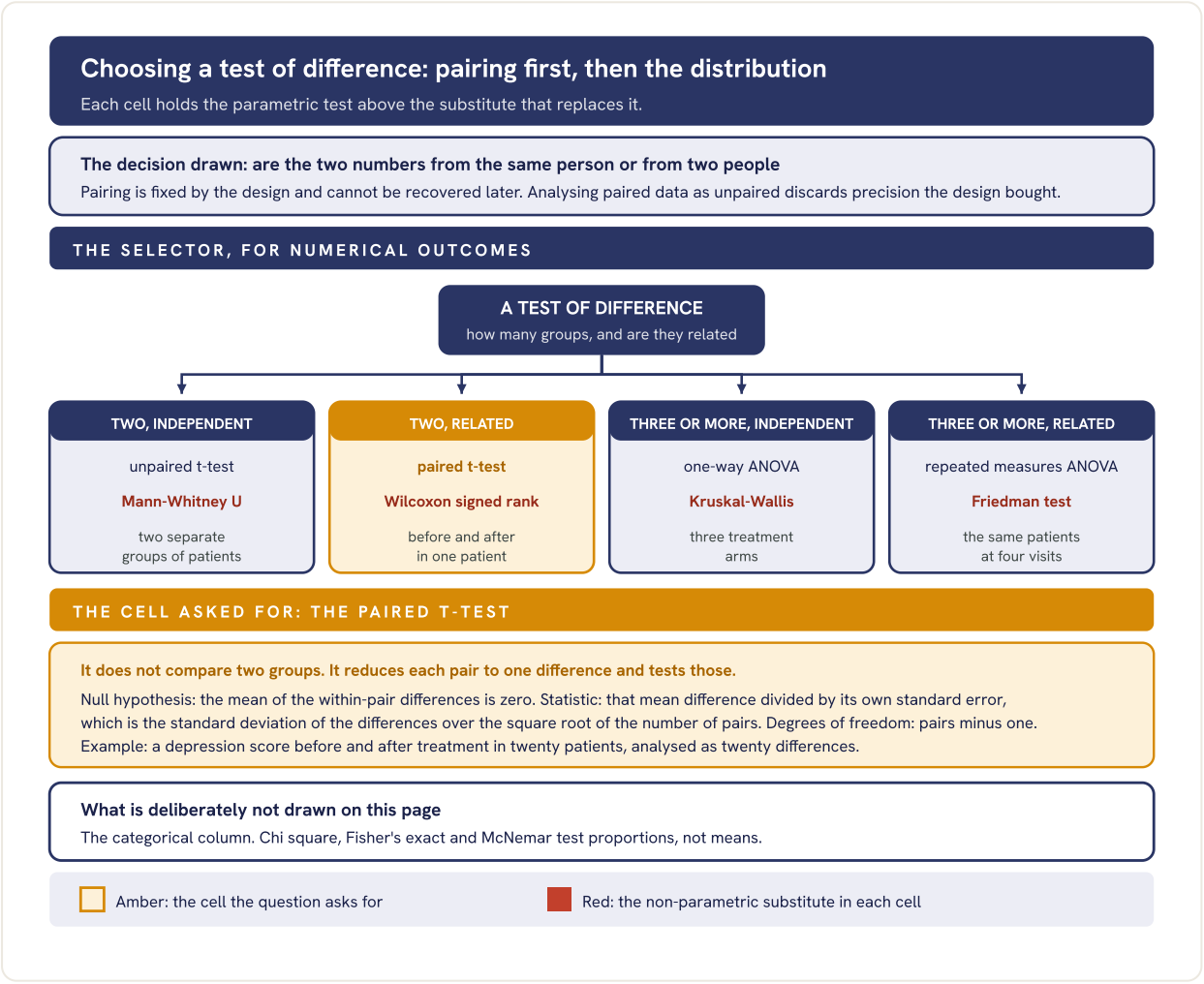


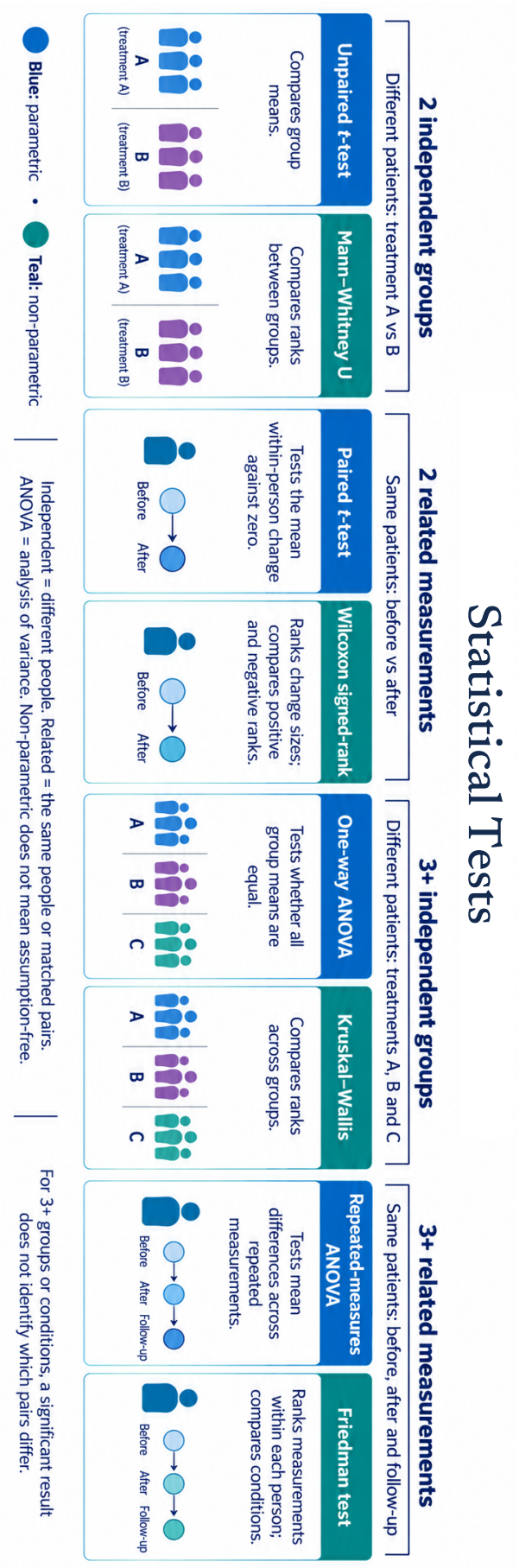
Fig 072.1 The difference tests as a two-way selector, groups against pairing, with the paired cell opened out below.

- **The two non-parametric tests.** Mann-Whitney U pools the observations, ranks them, and asks whether high ranks cluster in one group; it replaces the unpaired t-test. Wilcoxon signed rank ranks the absolute within-pair differences and compares positive against negative rank sums; it replaces the paired t-test.
- **The choosing rule.** Settle pairing from the design first, then distribution. Non-parametric tests need no normality, cost a little power when it does hold, and answer a question about ranks rather than means.

WHAT EARNS THE MARKS

- **The enumeration drawn as a selector,** so each test arrives with the situation it belongs to.
- **The paired test described as a one-sample test on differences,** which is what it actually is.
- **Degrees of freedom given as pairs minus one,** not as total observations minus two.
- **Each non-parametric test matched to the parametric one it replaces.**

SEE ALSO Slot I - 76 the unpaired t-test worked through Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology



Statistical Tests. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-72.

Credits and references: retained within the illustration.

I - 73

What is odd's ratio? Write briefly about confounding, effect modification and mediation. [4+2+2+2]

4 + 2 + 2 + 2 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Odds ratio	Definition, formula, design, interpretation
2	Confounding	A distortion, and how it is removed
2	Effect modification	A real finding, reported not removed
2	Mediation	On the causal path, so never adjusted away

THE DEFINITION

The odds ratio is the odds of the outcome among the exposed divided by the odds among the unexposed, computed from a two by two table as a times d over b times c. One means no association, above one a positive association, below one a protective one. It is the measure a case-control study can produce, because such a study fixes the number of cases and so cannot yield a risk. Read it with its confidence interval: an interval containing one is not a significant result.

QUESTION	CONFOUNDING	EFFECT MODIFICATION	MEDIATION
Where it sits	Beside the exposure, linked to both exposure and outcome	Beside the exposure, changing how strongly it acts	**On the causal path, between exposure and outcome**
What it does	Distorts the estimate, up or down	Splits one true estimate into two real ones	Carries part of the true effect
How you find it	The estimate moves when you adjust for it	**The stratum estimates differ from each other**	The effect shrinks when you adjust, and that is expected
What you do	Remove it: restrict, match, randomise, or adjust	Report it: give the estimate stratum by stratum	**Do not adjust it away if the total effect is the question**

Amber marks the discriminators. One question separates all three: is the third variable on the causal path, beside it, or changing its strength.

- **The one-line test.** Adjusting for a confounder corrects the answer. Adjusting for a mediator destroys it. Adjusting for an effect modifier hides the finding, because the finding is that the effect differs.
- **Where it goes wrong.** Randomisation controls confounding by variables you never measured, which is its whole advantage; adjustment controls only what you thought to record. Effect modification is often reported as a subgroup result without a formal interaction test, which is how spurious subgroup claims enter the literature.

WHAT EARNS THE MARKS

- **The formula written as a times d over b times c,** with the design it comes from named.
- **The confidence interval mentioned** in the same breath as the point estimate.
- **All three third variables told apart by position,** on the path or beside it.
- **The remove, report, do-not-adjust triad,** which is the whole second half of the question.

SEE ALSO Slot I - 78 odds ratio against relative risk Slot I - 80 internal and external validity SPRINT
Day 7, research methods, biostatistics and epidemiology

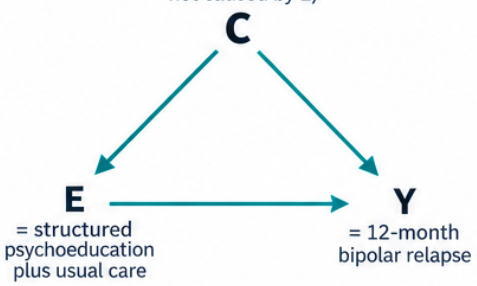
Confounding, effect modification and mediation

Same **exposure** and **outcome** in each panel. **Examples illustrate mechanisms**, not empirical findings.

CONFOUNDING

A common cause of the exposure and outcome can create a spurious or distorted association by mixing its influence with the exposure association.

C = baseline illness severity
(e.g. number of prior episodes)
(precedes intervention, not caused by E)



C is a pre-exposure common cause of E and Y.
It is not caused by E.

Analysis:

- Design for or control relevant pre-exposure **common causes** (e.g. randomisation, restriction, appropriate adjustment/stratification).
- Avoid indiscriminate adjustment.
- The exposure–outcome association may change after confounding control.

EFFECT MODIFICATION

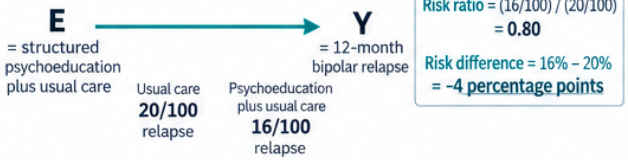
The effect of the exposure on the outcome differs across levels of a third variable (effect modifier), such as baseline family support.

Baseline family support
(effect modifier, not caused by E)

Low family support



High family support

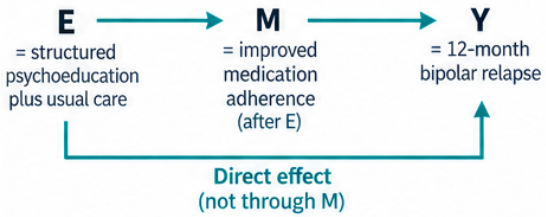


Numbers are hypothetical (per 100 people).

- The effect differs by subgroup on a specified scale (e.g. risk ratio or risk difference).
- Report stratum-specific effects and their uncertainty.
- Formally evaluate interaction (e.g. include an interaction term).
- The scale matters; modification on the risk difference scale need not imply modification on the risk ratio scale.
- One statistically significant subgroup and one non-significant subgroup do not prove interaction.
- Report stratum effects and evaluate interaction.

MEDIATION

The exposure affects the outcome partly or wholly through a mediator that lies on the causal pathway after the exposure.



- M occurs after E and is on the causal pathway from E to Y.
- The **total effect** includes all pathways (direct and mediated). Do not condition on M when estimating the total effect if the aim is to assess the overall impact of E.
- The **direct effect** excludes the specified mediated pathway. Use suitable mediation methods and appropriate confounding assumptions.
- Simply adding the mediator to a regression model does not necessarily identify a causal direct effect.
- A change in the exposure–outcome association after adjustment for the mediator alone does not prove mediation.

Confounder (C)

Precedes the exposure, is a **common** cause of E and Y, and is not caused by E.
Can create a spurious or distorted association.

Effect modifier

Asks: **for whom or when does the effect** of E on Y differ?
Defines subgroups with different effects.
Report stratum effects and evaluate interaction.

Mediator (M)

Occurs after the exposure and explains how the effect of E on Y operates (on the causal pathway).
Total effect includes all pathways; direct effect excludes the mediated pathway.

E = structured psychoeducation plus usual care **M** = improved medication adherence **Y** = 12-month bipolar relapse

Arrows denote hypothesised causal directions, except for comparisons between strata, which are not causal arrows.

Examples are hypothetical and illustrate mechanisms, not empirical findings.

Confounding, effect modification and mediation. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-73.

Credits and references: retained within the illustration.

I - 74

Discuss different types of test of significance in research. [10]

10 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The axis	Data type, number of groups, pairing, distribution
5	The selector	Parametric and non-parametric, side by side
2	Assumptions	What a parametric test demands before it is legal
1	Reading the result	What a p value is, and what it is not

WHAT IS COMPARED	PARAMETRIC TEST	NON-PARAMETRIC TEST	DATA NEEDED
Two independent groups	Unpaired t-test	Mann-Whitney U	Numerical
Two related measures	Paired t-test	Wilcoxon signed rank	Numerical
Three or more independent	One-way analysis of variance	Kruskal-Wallis	Numerical
Three or more related	Repeated measures analysis of variance	Friedman	Numerical
Two proportions, independent	Z test for proportions	Chi square, or Fisher's exact when cells are sparse	Categorical
Two proportions, paired	Not applicable	McNemar	Categorical
Association of two numerical variables	Pearson correlation	Spearman rank correlation	Numerical

Amber marks the categorical rows, because they are where a candidate who has memorised the t-test family runs out.

- **What a parametric test assumes.** Numerical data on an interval scale, approximately normally distributed, with comparable variances across groups, and independent observations. Fail any of these and the non-parametric column is not a compromise, it is the correct test.
- **Reading the result.** A p value is the probability of a result at least this extreme if the null hypothesis were true. It is not the probability that the null is true, and it says nothing about the size of the effect. Report the effect with its confidence interval alongside it.
- **The two errors.** A type one error rejects a true null, its rate set by the significance level. A type two error misses a real effect, its rate falling as power rises. Multiple testing inflates the first, which is why a correction or a pre-specified primary outcome is required.

WHAT EARNS THE MARKS

- **A selector, not a list.** Every test arrives attached to the comparison it serves.
- **Parametric and non-parametric paired row by row,** rather than in two separate lists.
- **The four parametric assumptions named.**
- **The p value defined correctly,** which is the single most commonly lost mark on this question.

SEE ALSO Slot I - 72 the selector drawn, and the paired test Slot I - 71 the chi square test SPRINT Day 7, research methods, biostatistics and epidemiology

I - 75

What is regression analysis? In which situations it is indicated? Discuss giving examples, how multiple regression analysis is conducted. [2+2+6]

2 + 2 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Modelling one outcome from one or more predictors
2 When it is indicated	Predict, adjust, quantify a dose-response
5 How it is conducted	Specify, check, fit, report, in that order
1 The caution	Association modelled is not causation shown

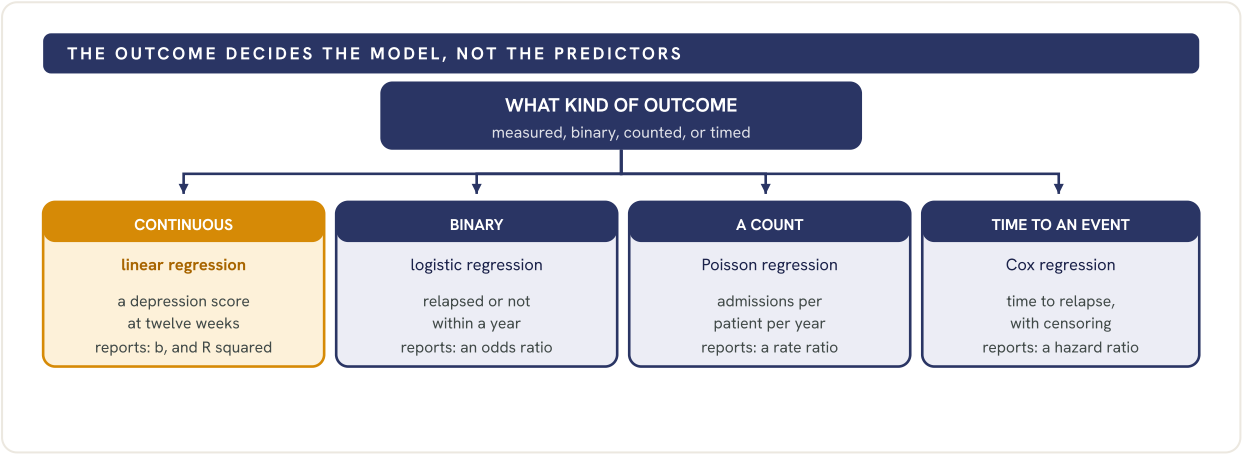


Fig 075.1 The regression family selected on the type of the outcome variable, each branch naming the effect measure it reports, with the linear case in amber as the one the question opens with.

HOW A MULTIPLE REGRESSION IS ACTUALLY CONDUCTED

Specify the outcome and choose predictors from clinical reasoning, never from a screening of p-values. Check the assumptions: a linear relation, independent residuals with constant spread and roughly normal distribution, and no severe collinearity between predictors. Keep enough data for the model, about ten observations for every predictor entered. Fit the model, entering variables together or in pre-planned blocks. Report each adjusted coefficient with its confidence interval, and the variance explained.

- **When it is indicated.** To predict an outcome from several variables, to adjust an exposure effect for measured confounders, and to quantify a dose-response relation across a range of exposure.
- **Two cautions.** Correlation quantifies the strength of a relation and makes no distinction between the two variables; regression names one as outcome and predicts it. And a stepwise search over many candidate predictors overfits, producing a model that does not replicate.

WHAT EARNS THE MARKS

- **The outcome type named as the thing that selects the model.**
- **Each model tied to the effect measure it reports,** coefficient, odds ratio, rate ratio, hazard ratio.
- **Assumptions checked before fitting,** written as a step of the method.

SEE ALSO Slot I - 73 confounding, effect modification and mediation Slot I - 83 Firth's penalized logistic regression

SPRINT Day 7, research methods, biostatistics and epidemiology

Factor analysis

Do several anxiety symptoms reflect a smaller number of shared dimensions?

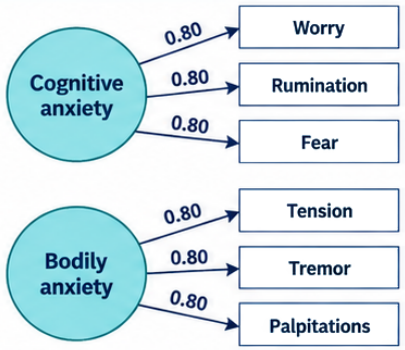
Factor = an unobserved (latent) dimension used to explain why measured items correlate. It is not a diagnosis or a directly measured symptom.

Illustrative correlation matrix—not actual patient data

Item	1 Worry	2 Rumination	3 Fear	4 Tension	5 Tremor	6 Palpitations
1. Worry	1.00	0.64	0.64	0	0	0
2. Rumination	0.64	1.00	0.64	0	0	0
3. Fear	0.64	0.64	1.00	0	0	0
4. Tension	0	0	0	1.00	0.64	0.64
5. Tremor	0	0	0	0.64	1.00	0.64
6. Palpitations	0	0	0	0.64	0.64	1.00

Each cell is the correlation of two items; the diagonal 1.00 is an item with itself. Two clusters suggest two factors.

Illustrative two-factor measurement model



One exact illustrative model: standardised items, uncorrelated factors, independent residuals. Arrows express a measurement model, not proven biological causation.

Reading a loading

A **loading** is an item's coefficient on a factor; in this simple standardised model it is also the item–factor correlation.

Loading 0.80 → common variance = 0.80^2 = 0.64 = 64%.

Remaining item-specific/error variance = 1 − 0.64 = 0.36 = 36%.

Predicted correlation of two items on one factor = 0.80 × 0.80 = 0.64.

Not 80% of patients;
Not 80% diagnostic accuracy.

Exploratory vs confirmatory factor analysis

Aspect	Exploratory factor analysis	Confirmatory factor analysis
Main question	What structure could explain the correlations?	Does a pre-specified structure fit new data?
Approach	Explore factor number and loadings; use theory, scree plot and parallel analysis.	Specify item–factor links in advance; examine fit and residuals.
Other features	Rotation aids interpretation.	Ideally test in an independent sample.

Key exam notes

1. **Communality** = proportion of an item's variance explained by common factors.
2. **Scree plot:** factor/component number on the x-axis, eigenvalue on the y-axis; look for an elbow. **Parallel analysis** compares observed eigenvalues with random-data eigenvalues. Do not rely only on the eigenvalue > 1 rule.
3. **Orthogonal rotation** assumes uncorrelated factors; **oblique rotation** allows correlation, often plausible for symptoms.
4. **Principal component analysis** summarises total variance; **common factor analysis** separates shared variance from unique/error variance.
5. Use adequate representative samples and methods suited to ordinal items.
6. Factor names and number require judgement; good fit does not prove the model true.

Factor analysis. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-75.

Credits and references: retained within the illustration.

Regression Analysis

Choose the model by the **OUTCOME.** Multiple regression = several predictors in one model.

Common predictors (in one model): Baseline severity • medication adherence • stress

CONTINUOUS

Linear regression



Outcome: depression score at 12 weeks

Effect: coefficient *b*

Example: *b* = +2 means 2 more score points per 1-unit increase in stress, holding other predictors fixed.

Check: linearity, independent errors, constant error variance; residual normality for usual small-sample inference.

R^2: proportion of outcome variance explained in the fitted data.

Illustrative associations, not causal claims.

BINARY

Logistic regression



Outcome: relapse — yes or no

Effect: odds ratio, OR = exp(*b*)

Example: OR = 2 means twice the ODDS of relapse, not twice the risk.

Check: independent observations, suitable log-odds relationship, adequate events; separation can cause problems.

Illustrative associations, not causal claims.

COUNT

Poisson regression



Outcome: number of admissions during follow-up

Effect: rate ratio, exp(*b*), with a follow-up-time offset

Example: rate ratio = 2 means twice the admission rate per person-year.

Check: conditional variance ≈ conditional mean. Overdispersion may need negative binomial regression.

Illustrative associations, not causal claims.

TIME TO EVENT

Cox proportional hazards regression



Outcome: time until first relapse

Effect: hazard ratio, HR = exp(*b*)

Example: HR = 2 means twice the instantaneous relapse rate among those still relapse-free.

Check: proportional hazards and non-informative censoring, conditional on predictors.

Censoring: follow-up ends before relapse is observed.

Illustrative associations, not causal claims.

WHAT ADJUSTMENT DOES

- **Confounder:** a common cause of exposure and outcome; **appropriate adjustment** reduces confounding.
- **Mediator:** lies on the causal path. **Adjustment** depends on whether total or direct effect is wanted.
- **Effect modification:** an association differs across groups; model an interaction and report it.

HOW TO READ THE RESULT

- **Effect size** = magnitude. **95% confidence interval** = precision and compatible values under the model.
- **Null value:** *b* = 0; OR, rate ratio and HR = 1.
- A small p value is **not a** large effect or proof of causation.

BUILD A USEFUL MODEL

- Specify outcome and predictors → check assumptions → fit → report adjusted effects with confidence intervals → validate predictions.
- Check collinearity, missing data and overfitting.
- No universal "10 observations per predictor" rule.

Regression Analysis. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-75.

Credits and references: retained within the illustration.

I - 76

Enumerate various statistical tests used to measure the differences. Describe the t-test in detail with an example. [4+6]

4 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Enumerate	By pairing and by number of groups, in one line
2	What the t-test is	A signal to noise ratio, with its three forms
4	The worked example	Pooled spread, standard error, t, degrees of freedom
1	Assumptions	And the substitute when they fail

THE ENUMERATION, AND THE THREE FORMS OF THE T-TEST

Two independent groups take the unpaired t-test or Mann-Whitney U; two related measures take the paired t-test or Wilcoxon signed rank; three or more groups take analysis of variance or Kruskal-Wallis; proportions take chi square, Fisher's exact or McNemar. The t-test itself has three forms: one sample against a known value, two independent samples, and paired. Each is a difference divided by the standard error of that difference.

STEP	WORKING, ON ILLUSTRATIVE DATA	VALUE
The data	Ten patients per arm; means 18.0 and 22.0, standard deviations 4.0 and 5.0	n equals 10 and 10
Difference in means	22.0 minus 18.0	4.00
Pooled variance	Nine times 16, plus nine times 25, all over 18	20.50
Pooled standard deviation	The square root of 20.50	4.53
Standard error of the difference	4.53 times the square root of one fifth	2.02
The statistic and the verdict	4.00 divided by 2.02, on 18 degrees of freedom, against a two-sided cut of 2.10	**t equals 1.98, not significant**

Made-up data, arithmetic real. A four-point difference that looks clinically worth having fails to reach significance at this spread and this sample size, which is the lesson the example is here to teach.

- **Read the failure correctly.** Not significant does not mean no difference. It means this study could not distinguish the observed difference from chance, which is a statement about the study's power as much as about the treatment.
- **Assumptions.** Numerical outcome, roughly normal within each group, comparable variances, independent observations. Unequal variances take the Welch correction; non-normality with small samples takes Mann-Whitney U instead.

WHAT EARNS THE MARKS

- **The enumeration organised by pairing and group number,** not as an unsorted list.
- **The three forms of the t-test named,** which most answers reduce to two.
- **Arithmetic shown line by line** to the degrees of freedom and the critical value.
- **A verdict written in words,** including what a non-significant result does not prove.

SEE ALSO Slot I - 72 the selector drawn, and the paired t-test Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 77

Analysis of Variance (ANOVA). Its role in research in behavioral Medicine. [10]

10 | ASKED AT 7 MARKS | BIOSTATISTICS AND EPIDEMIOLOGY

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What it is	Variance partitioned, then compared as a ratio
3	The working	The F ratio and its two degrees of freedom
2	Its forms	One-way, two-way, repeated measures, covariance
3	Role in the field	Where a behavioural trial needs it, and post hoc

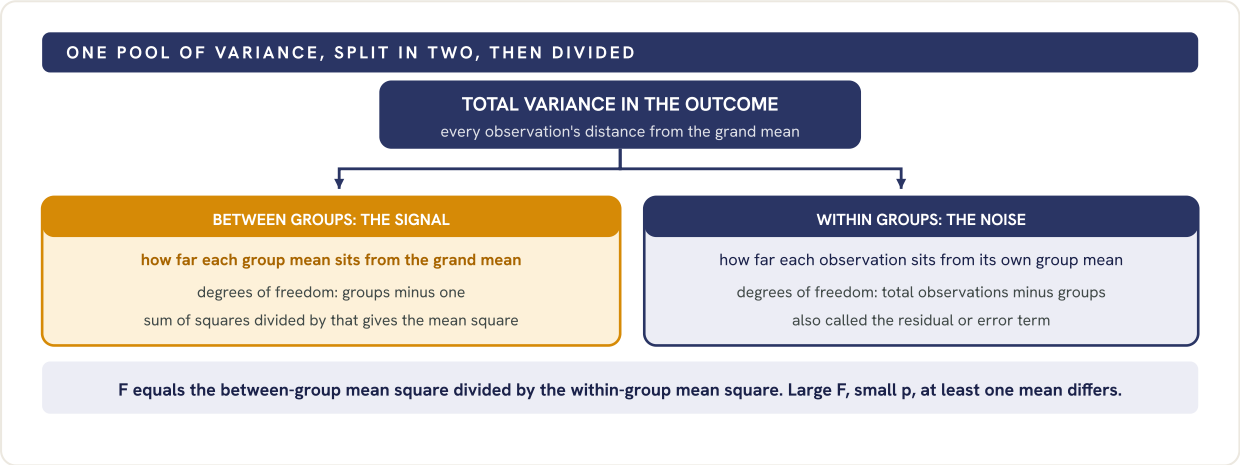


Fig 077.1 Analysis of variance drawn as what its name says: the total variation split into a between-group part and a within-group part, the ratio of the two being the test statistic.

- **Why not repeated t-tests.** Three comparisons at five per cent each carry about a fourteen per cent chance of a false positive. One F test holds it at five.
- **What a significant F does not say.** Not all means are equal, never which pair differs. Only then a post hoc test: Tukey, Bonferroni or Scheffe.
- **Its forms and its assumptions.** One-way for one factor; two-way for two, which makes an interaction testable; repeated measures over time; covariance to adjust for baseline. It assumes independence, normal residuals, comparable variances.

ITS ROLE IN BEHAVIOURAL RESEARCH

Behavioural work rarely compares two things once. It compares three or more arms, or the same participants at baseline, end of treatment and follow-up. Two-way and repeated measures forms are what let a trial test an interaction.

WHAT EARNS THE MARKS

- **The partition named,** between-group against within-group, before the F ratio.
- **Both degrees of freedom given,** groups minus one and observations minus groups.
- **The inflated error rate quantified,** not just asserted.
- **Interaction named as what two-way analysis buys.**

SEE ALSO Slot I - 74 tests of significance Slot I - 76 the t-test worked through SPRINT
Day 7, research methods, biostatistics and epidemiology

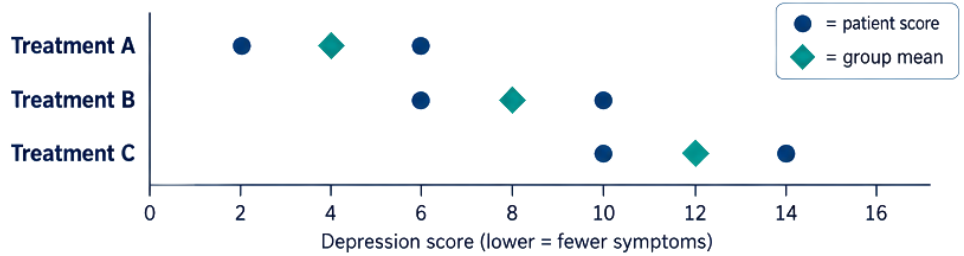
ANOVA Explained

Are treatment averages far apart compared with the variation among patients receiving the same treatment?

Illustrative depression scores after treatment • different patients in each group • lower scores = fewer symptoms

	Treatment A	Treatment B	Treatment C
Patient 1	2	6	10
Patient 2	4	8	12
Patient 3	6	10	14
Mean	**4**	**8**	**12**

Same data on a common score axis (dots = patients, diamonds = group means)



1 Find the averages

Group means:
A = 4, B = 8, C = 12.

Overall mean =
72 ÷ 9 = 8.

2 BETWEEN: how far apart are group averages?

Compare each mean with 8: −4, 0, +4.

Square: 16, 0, 16.

Each mean represents 3 patients.

Sum = 3 × 16 + 3 × 0 + 3 × 16 = **96.**

3 WITHIN: how much do patients vary inside each group?

In A: 2, 4, 6 differ from its mean 4 by −2, 0, +2.

Squared gaps:
4 + 0 + 4 = 8.

B and C each also give 8.

Total = **8 + 8 + 8 = 24.**

4 Put both on a comparable scale

Source	Sum of squares	Degrees of freedom	Mean square
Between	96	3 − 1 = 2	96 ÷ 2 = 48
Within	24	9 − 3 = 6	24 ÷ 6 = 4

Degrees of freedom account for how many independent pieces of information remain.

5 F ratio = BETWEEN mean square ÷ WITHIN mean square

***F* = 48 ÷ 4 = 12**

F near 1: similar amounts of variation.

A sufficiently large F: evidence that not all population means are equal.

6 Interpret

For degrees of freedom 2 and 6, the 5% cutoff is 5.14.
Our 12 is larger:
***p* = 0.008.**
At least one population mean differs.
ANOVA does not say which pairs: use follow-up comparisons, such as Tukey.

ONE-WAY ANOVA

One grouping factor: treatment A, B or C.

Compares their average depression scores.

TWO-WAY ANOVA

Two factors: medication type × psychotherapy yes/no.

Tests both main effects and their interaction: does the medication difference depend on psychotherapy?

REPEATED-MEASURES ANOVA

Same patients at baseline, after treatment and follow-up.

Accounts for related measurements.

ANCOVA - analysis of covariance

Compares group means while adjusting for a continuous variable, such as baseline depression score.
Covariance describes how two variables vary together; ANCOVA uses regression adjustment.

Assumptions: independent patients, approximately normal residuals, similar group variances for ordinary ANOVA.
Tiny groups here simplify arithmetic; this is not a real trial.

ANOVA Explained. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-77.

Credits and references: retained within the illustration.

I - 78

What is odds ratio? How does it differ from relative risk? Describe the differences giving examples. [2+8]

2 + 8 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Both, defined	Odds against risk, and the table both come from
4 The grid	Design, denominator, meaning, behaviour
3 Worked both ways	A common outcome, then a rare one
1 The rule	When the two may be spoken of as one

DOMAIN	RELATIVE RISK	ODDS RATIO	WHAT SETTLES IT
What it divides	Risk in the exposed by risk in the unexposed	Odds in the exposed by odds in the unexposed	**Risk uses everyone as denominator, odds uses the non-affected**
Design it comes from	Cohort study or randomised trial	Case-control, and also cohort or trial	**A case-control fixes the cases, so it can yield no risk**
How it is read aloud	Directly: twice as likely	As odds, which is not the same as likely	Only relative risk may be spoken as times the chance
Common outcome	Stays interpretable	Sits further from one than the risk ratio does	**Above about ten per cent, the two separate**
Rare outcome	Close to the odds ratio	Approximates the relative risk	The rare disease assumption

WORKED TWICE, ON ILLUSTRATIVE COUNTS

Common outcome. Of 100 exposed, 30 develop the outcome; of 100 unexposed, 15 do. Risks are 0.30 and 0.15, so the relative risk is 2.00. Odds are 30 over 70 and 15 over 85, so the odds ratio is 2.43. The odds ratio has overstated the risk ratio. Rare outcome. Of 1000 exposed, 3 develop it; of 1000 unexposed, 1 does. The relative risk is 3.00 and the odds ratio is 3.01. Now they agree.

- **The rule the two examples prove.** The odds ratio approximates the relative risk only when the outcome is uncommon. When the outcome is common, the odds ratio exaggerates the effect in whichever direction it points, and reporting it as though it were a risk ratio overstates the finding.
- **Where this bites in practice.** Logistic regression returns odds ratios whatever the frequency of the outcome, so an adjusted odds ratio from a study of a common outcome is routinely read as a risk ratio and routinely overstated. Say odds, and give the confidence interval.

WHAT EARNS THE MARKS

- **Odds distinguished from risk in the first two lines,** by what sits in the denominator.
- **The design constraint stated,** why a case-control study cannot report a risk.
- **Numbers on the page.** The stem asks for examples, so the two worked sets are the examples.
- **The rare disease assumption named as an assumption,** with the condition under which it fails.

SEE ALSO Slot I - 73 the odds ratio and confounding Slot I - 75 regression and the measures it reports SPRINT Day 7, research methods, biostatistics and epidemiology

Absolute risk reduction and number needed to treat

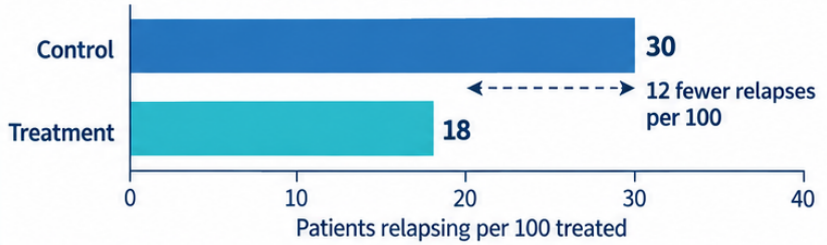
How many patients need treatment to prevent one additional relapse—and what harms occur?

Randomised trial data (12-month follow-up)

Group	Patients	Relapses by 12 months	Adverse events by 12 months
Control	100	30	4
Treatment	100	18	12

Outcomes are separate: a person can have both a relapse and an adverse event. Denominator 100 is the number in that trial arm.

Relapse risk at 12 months



BENEFIT: preventing relapse

Control event risk (CER) = 30 ÷ 100 = 0.30 = 30%.

Experimental event risk (EER) = 18 ÷ 100 = 0.18 = 18%.

Absolute risk reduction (ARR) = CER – EER
= 0.30 – 0.18 = 0.12 = 12 percentage points.

Number needed to treat (NNT) = 1 ÷ ARR
= 1 ÷ 0.12 = 8.33 → round UP to **9.**

Treat 9 comparable patients for 12 months to prevent one additional relapse on average, compared with control.

HARM: causing an additional adverse event

Control adverse-event risk = 4 ÷ 100 = 0.04 = 4%.

Treatment adverse-event risk = 12 ÷ 100 = 0.12 = 12%.

Absolute risk increase (ARI) = treatment risk – control risk
= 0.12 – 0.04 = 0.08 = 8 percentage points.

Number needed to harm (NNH) = 1 ÷ ARI
= 1 ÷ 0.08 = 12.5 → round UP to 13.

Treat 13 comparable patients for 12 months for one additional adverse event on average.

Key exam points and distinctions

1. Use proportions: 1 ÷ 0.12, NOT 1 ÷ 12.
2. NNT is an average additional benefit, not a guarantee that exactly one of every 9 benefits.
3. Always state outcome, comparator, population and follow-up duration. Do not compare a 6-week NNT with a 12-month NNT as if equivalent.
4. Lower NNT and higher NNH may be desirable, but relapse and harm differ in severity: numbers alone do not decide treatment.
5. Point estimates show effect magnitude; confidence intervals assess uncertainty. Statistical significance and clinical importance are separate.
6. NNT depends on baseline risk. No universal NNT cutoff defines a worthwhile treatment. If ARR = 0, no finite NNT; if negative, treatment increases that outcome.

Absolute risk reduction and number needed to treat. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-78.

Credits and references: retained within the illustration.

Types of data and measurement scales

Fictional psychiatric clinic data

Record	Diagnosis	Severity	First admission year	Previous admissions	Sleep last night (hours)
A	Depression	Mild	2020	0	4
B	Bipolar disorder	Moderate	2022	2	6.5
C	Schizophrenia	Severe	2024	4	8

What have I recorded—and which comparisons are meaningful?

	NOMINAL	ORDINAL	INTERVAL	RATIO
Property ladder	**Categories**	**Order**	**Equal intervals**	**True zero**
Definition and example	**Categories, no inherent rank.** Diagnosis names are labels; codes 1, 2, 3 do not create quantities.	**Ordered categories; equal gaps are not established.** Mild → Moderate → Severe; arrow means increasing severity, NOT equal steps.	**Equal units; arbitrary zero.** Calendar year: 2022 − 2020 = 2 years; 2024 − 2022 = 2 years. Differences are meaningful, ratios of calendar-year numbers are not. Intelligence quotient is conventionally treated as interval, not a ratio of intelligence.	**Equal units AND meaningful zero.** Zero admissions means none. 4 admissions ÷ 2 admissions = 2: twice as many admissions. Ratios meaningful.
How to summarise	Counts, percentages, mode (most frequent category).	Category frequencies and median category.	Mean, standard deviation (when appropriate), differences between values.	Mean, standard deviation (when appropriate), ratios and other arithmetic operations.

A separate distinction: discrete versus continuous

Previous admissions (count)



Discrete: separate possible values; not 2.5 admissions.

Sleep duration (hours)



Continuous: intermediate values are possible. 8 hours ÷ 4 hours = 2: twice as long asleep, not twice the sleep quality.

Both are RATIO; one is discrete, the other continuous.

Key exam distinctions

1. Single rating items are ordinal; approximate interval treatment of a summed scale needs justification.
2. Rounding does not change an underlying continuous quantity.
3. Measurement scale informs analysis; study design and statistical assumptions also matter. Do not assume all numerical data require parametric tests.

Types of data and measurement scales. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

OR vs RR

One illustrative example, two different denominators.

Illustrative 2x2 data (medication and relapse)

	Relapse (outcome)	No relapse (non-outcome)	Total
Irregular medication (exposed)	40	60	100
Regular medication (reference)	20	80	100
Total	60	140	200

Relative risk (RR) calculation

Risk in exposed $= \frac{40}{100} = 0.40$ (40%)

Risk in reference $= \frac{20}{100} = 0.20$ (20%)

$$\mathbf{RR} = \frac{0.40}{0.20} = \mathbf{2.00}$$

Interpretation: Twice the risk of relapse in irregular versus regular medication-takers.

Odds ratio (OR) calculation

Odds in exposed $= \frac{40}{60} = 2/3$

Odds in reference $= \frac{20}{80} = 1/4$

$$\mathbf{OR} = \frac{(2/3)}{(1/4)} = \mathbf{2.67}$$

Interpretation: 2.67 times the odds of relapse in irregular versus regular medication-takers.

Exam differences

Feature	Relative risk (RR)	Odds ratio (OR)
What it compares	Probabilities: outcome / all people	Odds: outcome / non-outcomes
Usual study design	**Cohort studies and randomised trials**	**Case-control studies; also cohorts and trials**
Why case-control differs	Cannot directly calculate risks from ordinary case-control samples	Can compare exposure odds in cases versus controls: ad/bc
Interpretation in this example	Twice the risk of relapse	2.67 times the odds of relapse
Common outcome	Directly describes relative probability	Farther from 1 than RR for the same 2×2 table when association exists
Rare outcome in both groups	RR and OR become close	Approximates RR
Regression	Log-binomial or modified Poisson can estimate adjusted RR	Logistic regression estimates adjusted OR

Both: null value = 1; report 95% confidence intervals; association does not establish causation.

Absolute difference here: 40% − 20% = 20 percentage points. Never call an OR "times the risk".

OR vs RR. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Sensitivity and specificity

Among people with and without depression, how often is the screen correct?

Teaching numbers—not performance claims for a named instrument.

Screen result	Depression present (reference interview)	Depression absent (reference interview)	Total
Screen positive	True positive (TP) 90	False positive (FP) 90	180
Screen negative	False negative (FN) 10	True negative (TN) 810	820
Total	100	900	1,000

Choose the denominator from the reference-status COLUMN.

Choose the denominator from the reference-status COLUMN.

SENSITIVITY — among those WITH depression

= true positives ÷ all with depression
= TP ÷ (TP + FN)
= 90 ÷ (90 + 10) = 90 ÷ 100 = 0.90 = 90%.

90 of 100 cases are detected; 10 are missed.

False-negative rate = FN ÷ (TP + FN) = 10 ÷ 100 = 10% = 1 − sensitivity.

SPECIFICITY — among those WITHOUT depression

= true negatives ÷ all without depression
= TN ÷ (TN + FP)
= 810 ÷ (810 + 90) = 810 ÷ 900 = 0.90 = 90%.

810 of 900 non-cases screen negative; 90 are falsely flagged.

False-positive rate = FP ÷ (TP + TN) = 90 ÷ 900 = 10% = 1 − **specificity.**

Exam distinctions

- Prevalence = all with depression ÷ all tested = 100 ÷ 1,000 = 10%.
- Sensitivity is **NOT** the chance of depression after a positive result; that is positive predictive value.
- **SnNout:** highly **Sensitive** + **Negative** helps rule **OUT. SpPin:** highly Specific + Positive helps **rule IN.** These are aids, not guarantees; pre-test probability and likelihood ratios determine the remaining uncertainty.
- At a fixed threshold and case mix, changing prevalence alone need not change **sensitivity** or **specificity.** Different settings, disease severity, reference standards and verification bias can change measured performance.
- Both positive and negative screens should receive the reference interview in a validation study; interpret independently. A positive screen needs clinical assessment.

Sensitivity and specificity. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Predictive values and likelihood ratios

Given this screen result, how likely is depression?

Screen results among 1,000 people
(Sensitivity 90%, specificity 90%, prevalence 10%)

Screen result	Depression present (diagnostic interview +)	Depression absent (diagnostic interview –)	Total
Positive	90 (true positive)	90 (false positive)	180
Negative	10 (false negative)	810 (true negative)	820
Total	**100**	**900**	**1,000**

Predictive values: read across the result row

Positive predictive value (PPV) = true positives ÷ all positive
= 90 ÷ (90 + 90) = 90 ÷ 180 = **50%.**

Half of positive screens are true cases.

Negative predictive value (NPV) = true negatives ÷ all negative
= 810 ÷ (810 + 10) = 810 ÷ 820 ≈ **98.78%.**

After a negative screen, probability of depression =
10 ÷ 820 ≈ **1.22%.**

Likelihood ratios: how much the result shifts odds

Positive likelihood ratio (LR+) = sensitivity ÷ (1 – specificity)
= 0.90 ÷ (1 – 0.90) = 0.90 ÷ 0.10 = **9.**

Positive is 9 times as likely with depression as without.

Negative likelihood ratio (LR–) = (1 – sensitivity) ÷ specificity
= (1 – 0.90) ÷ 0.90 = 0.10 ÷ 0.90 = 1/9 ≈ 0.111.

LR = 1 gives no update; larger LR+ and smaller LR– are more informative.

Probability → odds → updated probability

p = pre-test probability; odds = p ÷ (1 – p).
Pre-test odds = 0.10 ÷ 0.90 = 1/9.
Post-test odds = pre-test odds × likelihood ratio.

- **Positive:**
  (1/9) × 9 = 1.
  Post-test probability = odds ÷ (1 + odds) = 1 ÷ 2 = **50%.**
- **Negative:**
  (1/9) × (1/9) = 1/81.
  Post-test probability = (1/81) ÷ (1 + 1/81) = 1/82 ≈ **1.22%.**

Multiply ODDS, not probability.

What if prevalence falls to 1%?

Hold sensitivity and specificity at 90%. Among 1,000: 10 cases, 990 non-cases → 9 true positives, 1 false negative, 99 false positives, 891 true negatives.

Screen result	Depression present	Depression absent	Total
Positive	9 (true positive)	99 (false positive)	108
Negative	1 (false negative)	891 (true negative)	892
Total	**10**	**990**	**1,000**

PPV = 9 ÷ (9 + 99) = 9 ÷ 108 ≈ **8.33%.**
NPV = 891 ÷ (891 + 1) = 891 ÷ 892 ≈ **99.89%.**

Lower prevalence → lower PPV, higher NPV. Most positive screens are now false positives.

Important considerations and limitations

This comparison holds test performance fixed.

Threshold, case mix and reference-standard quality can change likelihood ratios in practice.

Individual pre-test probability should reflect clinical context; it need not equal population prevalence.

Teaching numbers, not claims for a named test.

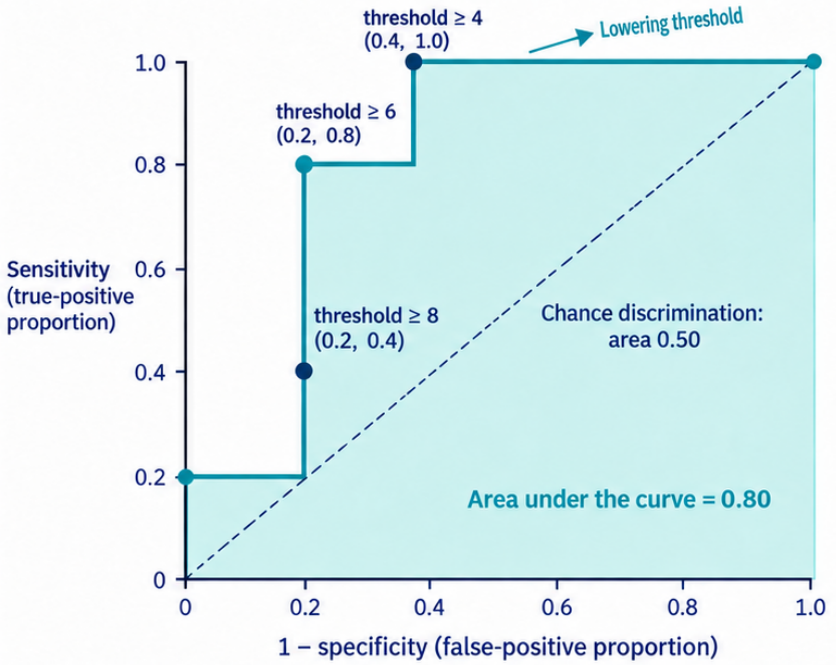
Predictive values and likelihood ratios. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Receiver operating characteristic curve

Where should a depression-screen threshold be placed?

Receiver operating characteristic (ROC) curve



Data: 10 fictional people; diagnostic interview defines status (higher screen score suggests depression)

Depression present (5 people)	4	6	7	8	10
Depression absent (5 people)	1	2	3	5	9

No ties. A positive screen means score ≥ threshold.

Test performance at selected thresholds

Positive if score	Sensitivity (true-positive proportion)	Specificity (true-negative proportion)	1 – specificity (false-positive proportion)
≥ 8	2/5 = 40%	4/5 = 80%	1/5 = 20%
≥ 6	4/5 = 80%	4/5 = 80%	1/5 = 20%
≥ 4	5/5 = 100%	3/5 = 60%	2/5 = 40%

Denominators: 5 reference-positive people for sensitivity; 5 reference-negative people for specificity.
At threshold 6, detect 4 of 5 cases and falsely flag 1 of 5 non-cases.

Area under the curve (AUC)

Area under the curve (AUC) = chance that a randomly chosen case scores higher than a randomly chosen non-case; ties count half.

5 cases × 5 non-cases = 25 pairs.
Case scores 4, 6, 7, 8, 10 beat 3, 4, 4, 4, 5 non-cases.
AUC = (3 + 4 + 4 + 4 + 5) / 25 = 20 / 25 = 0.80.

Key points and exam takeaways

1. Lower threshold generally raises sensitivity and lowers specificity; either can stay unchanged between adjacent cutoffs.
2. AUC describes ranking, NOT the chance of disease after a positive result, calibration, or 80% diagnostic accuracy.
3. AUC: 0.5 chance; 1 perfect; below 0.5 can indicate reversed score direction.
4. Choose a threshold using missed-case harms, false-positive harms, prevalence and available follow-up. Validate on independent data; 10 people only illustrate the arithmetic.

Receiver operating characteristic curve. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

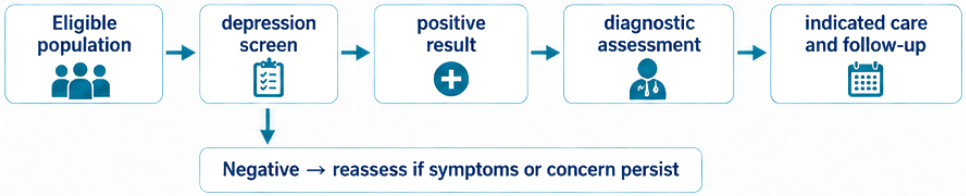
Credits and references: retained within the illustration.

Screening principles and biases

Does finding depression earlier improve outcomes—or only change the statistics?

Screening: systematically identifies possible unrecognised disorder or increased risk in a defined population. A positive result prompts assessment.

Diagnosis: clinical evaluation establishes or excludes a disorder, including symptoms, duration, impairment and alternatives. Screening can uncover symptoms not volunteered.

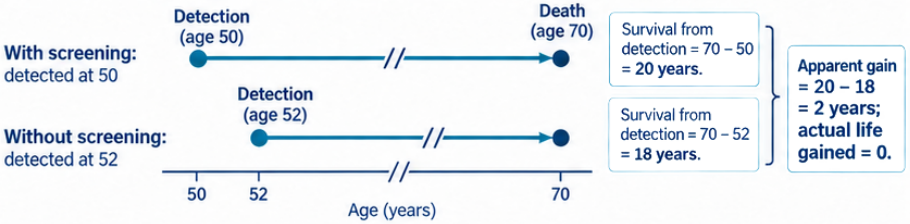


Wilson and Jungner: 10 classic criteria

Category	No.	Criterion
Condition	1	Important health problem.
	2	Recognisable latent or early stage.
	3	Natural history understood.
Test	4	Suitable test.
	5	Acceptable to the population.
Treatment	6	Accepted treatment.
	7	Facilities for diagnosis and treatment.
Programme	8	Agreed policy on whom to treat.
	9	Economically balanced cost.
	10	Continuing process.

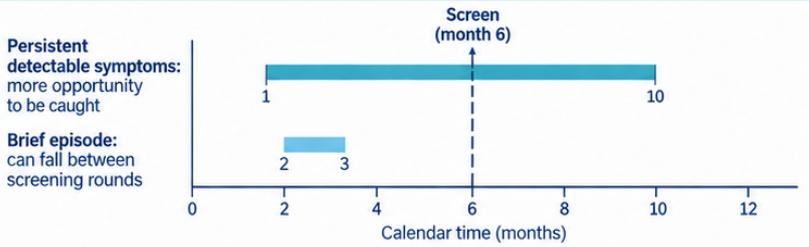
LEAD-TIME BIAS: the clock starts earlier

Hypothetical timing illustration; no benefit assumed.



Earlier detection alone does not prove longer life.

LENGTH BIAS: longer detectable courses are sampled more often



Periodic screens over-represent longer detectable episodes. In classic cancer screening, slow progression often means better prognosis; that is not automatic in psychiatry.

Overdiagnosis ≠ false positive

Overdiagnosis detects a real condition that would never have caused clinically important harm. A false positive is positive despite absence of the target condition. Harms include labelling, anxiety, unnecessary assessment and treatment.

Judge the whole programme

Compare screened and unscreened groups from a common starting point, ideally randomised. Evaluate patient-important symptoms, functioning, quality of life and relevant morbidity or mortality, alongside harms, access and costs. More diagnoses or longer survival after diagnosis alone are insufficient.

Psychiatric application

A questionnarire without accessible assessment, effective care and follow-up is not a complete screening programme.

Screening principles and biases. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Incidence and prevalence

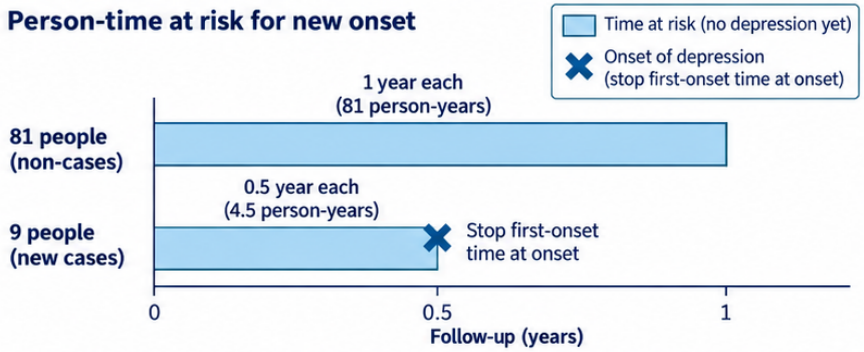
How many people have depression—and how quickly do new cases arise?

Cohort data (closed community, 1 year follow-up)

Observation	People
Population	100
Depression already present at start	10
Never depressed and at risk of first onset at start	90
First episodes starting exactly at 6 months	9
Of the 10 initial cases, recovered during the year	4
New cases recovered by year-end	0
Losses, deaths, entrants	0

All 100 people remain observed for the full year. Each new case contributes only the first 6 months to first-onset person-time.

Person-time at risk for new onset



PREVALENCE: existing cases

Point prevalence = current cases ÷ population at that point.

- At start = 10 ÷ 100 = 10%.
- At year-end = (10 – 4 + 9) ÷ 100 = 15 ÷ 100 = 15%.

One-year period prevalence = unique people depressed at any time in the year ÷ population.

- (10 + 9) ÷ 100 = 19 ÷ 100 = 19%.
- Count a person once, even if episodes recur.

Lifetime prevalence = ever affected by survey date ÷ population surveyed; depends on recall and age, not just current illness.

INCIDENCE PROPORTION: risk over a stated period

New first cases ÷ people at risk at start.

- 9 ÷ (100 – 10) = 9 ÷ 90 = 0.10 = 10% over 1 year.
- Ten in 100 initially at-risk people developed a first episode during the year.
- Denominator excludes the 10 existing cases.
- This simple risk calculation assumes complete follow-up.

INCIDENCE RATE: speed of new onset

New first cases ÷ total person-time at risk.

- 81 non-cases × 1 year = 81 person-years.
- 9 new cases × 0.5 year = 4.5 person-years.
- Total = 81 + 4.5 = 85.5 person-years.
- Rate = 9 ÷ 85.5 ≈ 0.1053 cases per person-year = 10.53 cases per 100 person-years.
- Not 10.53%: a rate has time in its denominator.

Prevalence describes burden; incidence describes new occurrence.

In a stable population with a rare condition: prevalence ≈ incidence rate × mean duration.
Longer duration can raise prevalence without more new cases.
Specify case definition, population and time window. First onset differs from recurrence; this example counts first episodes.

Incidence and prevalence. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Survival analysis

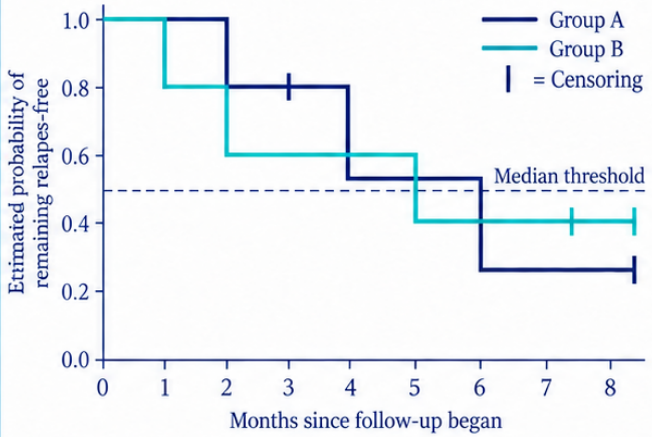
How long do patients remain relapse-free when follow-up differs?

Fictional follow-up data (all patients start relapse-free)

Patient	Group A: month, status	Group B: month, status
1	2, relapse	1, relapse
2	3, censored	2, relapse
3	4, relapse	5, relapse
4	6, relapse	7, censored
5	8, censored	8, censored

Censored = relapse not observed up to that month; later outcome unknown.
Event here is relapse, not death.

Kaplan–Meier survival curves



Median time to relapse: A 6 months; B 5 months.
First time estimated survival ≤ 0.5.

Build Group A's curve

Event month	At risk just before (n)	Relapses (d)	Survival update
2	5	1	$1 \times (4/5) = 0.80$
4	3	1	$0.80 \times (2/3) \approx 0.533$
6	2	1	$(8/15) \times (1/2) = 4/15 \approx 0.267$

At month 3 one patient is censored; the month 4 denominator is 3, not 4.

At each event: new survival = previous survival × (n − d) / n.
n = still observed and relapse-free just before the event;
d = relapses then.
(n − d) / n is the conditional fraction surviving that step.

Censoring

- Use each patient's observed time.
- A censoring mark does not mean relapse, cure, or zero risk.
- Remove that person from later risk sets; do not drop their earlier follow-up.

Log-rank test

- Null hypothesis: group survival distributions are the same.
- Compares observed with expected events across event times, using the risk sets.
- Gives evidence of a difference, not its magnitude.
- Crossing curves can reduce its power.
- No p-value is inferred from this sketch.

Assumptions and limits

- Independent patients; censoring unrelated to unobserved event risk, conditional on modelled information.
- Informative dropout can bias results.
- Few people at risk make tails uncertain.
- Cox regression estimates a hazard ratio; proportional hazards is an additional assumption.
- If a curve never reaches 0.5, median is not reached.

Survival analysis. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Central tendency and dispersion

What is typical—and how much do patients differ?

Data and working table

Fictional psychiatric admission length of stay (days)

Patient	Stay x (days)	Deviation x - 5 (days)	Squared deviation (days^2)
A	2	−3	9
B	4	−1	1
C	4	−1	1
D	6	1	1
E	9	4	16
Totals	**25**	**0**	**28**

x = one stay; n = number of patients = 5.

Measures of central tendency

Mean = sum of stays ÷ n
= (2 + 4 + 4 + 6 + 9) ÷ 5
= 25 ÷ 5 = **5** days.

Median = middle ordered stay
= **4** days.

Mode = most frequent stay
= **4** days (two patients).

Measures of spread

Range = maximum − minimum
= 9 − 2 = 7 days.

Quartiles: exclude the middle observation for this example.

Lower half 2, 4 → Q_1 = (2 + 4) / 2
= 3 days.

Upper half 6, 9 → Q_3 = (6 + 9) / 2
= 7.5 days.

Q_1 = first quartile; Q_3 = third quartile.

Interquartile range = $Q_3 - Q_1$
= 7.5 − 3 = **4.5** days.

Quartile conventions differ, especially in small samples.

Variance and standard deviation

Sample variance s^2 = sum of squared deviations ÷ $(n - 1)$
= 28 ÷ (5 − 1)
= 28 ÷ 4 = 7 days^2.

Sample standard deviation $s = \sqrt{7}$
≈ 2.65 days.

s = sample standard deviation;
s^2 = sample variance.

Why $n - 1$?
Estimating the mean uses one degree of freedom; $n - 1$ corrects bias in the variance estimate. For a complete population, divide by N (population size).

Dot plot of length of stay

Each dot represents one patient



Key takeaway

A long stay pulls the mean upward; the median resists outliers. Common summaries: mean with standard deviation for roughly symmetric data; median with interquartile range for skewed data. Standard deviation is in days; variance is in days^2.

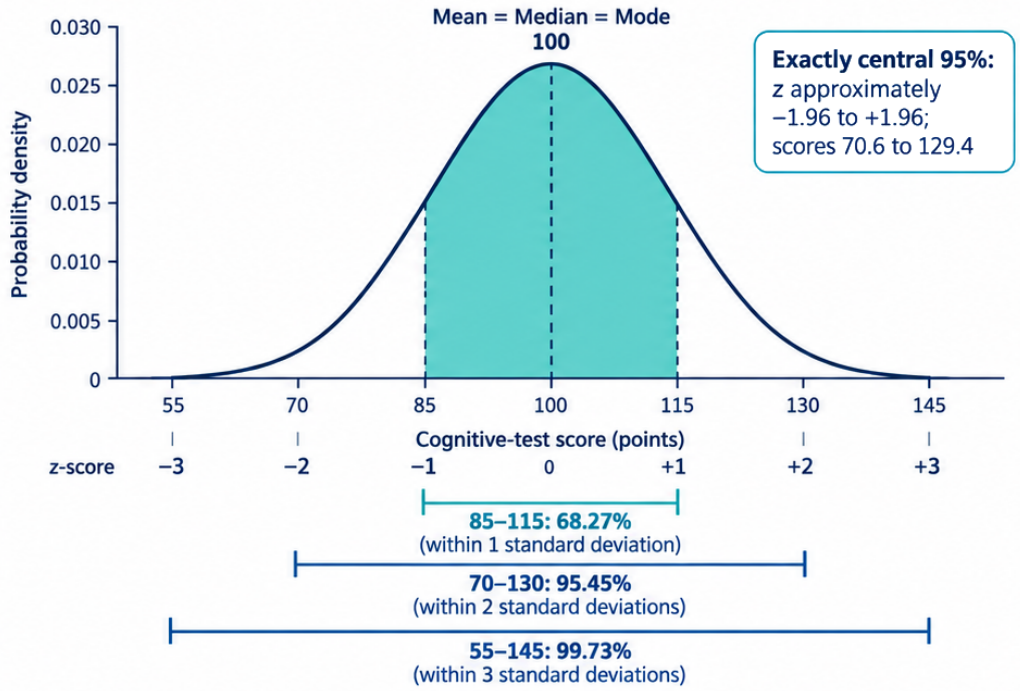
Central tendency and dispersion. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Normal distribution and standard scores

How unusual is this cognitive-test score?

Assumed normal reference distribution—not a patient dataset



Exactly central 95%: *z* approximately −1.96 to +1.96; scores 70.6 to 129.4

Inputs and standardised positions

Raw score, *x* (points)	70	85	100	115	130
z-score	−2	−1	0	+1	+2

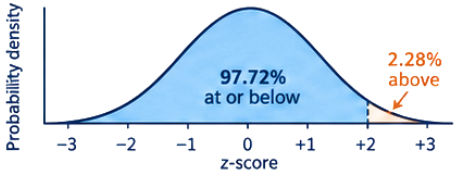
x = observed score
μ (mu) = population mean
σ (sigma) = population standard deviation
z = distance from the mean in standard deviations

Worked example: Score 130

$z = (x - \mu) \div \sigma$
$= (130 - 100) \div 15$
$= 30 \div 15 = +2$

130 is two standard deviations above the reference mean.

Position on the distribution (*z* = +2)



About the 97.7th percentile: about 97.7% of the reference population scores at or below 130.

Reading area

- Total area = 1 = 100%.
- Area between two cutoffs = probability of a score in that interval.
- Curve height alone is not a probability.
- A single exact value has zero area in a continuous model.

Key points and caveats

1. Standardising gives mean 0 and standard deviation 1; it does not make a skewed distribution normal.
2. Use normal-curve percentiles only when the normal model is appropriate.
3. A cognitive score alone does not establish a psychiatric diagnosis.

Normal distribution and standard scores. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Standard deviation, standard error and confidence intervals

How different are patients—and how precisely have we estimated their mean sleep?

Patient	A	B	C	D	E	F	G	H	I
Sleep last night (hours)	4	4	4	4	6	8	8	8	8

$\bar{x}$ = sample mean (*mean of the observed values*)
n = sample size (*number of patients*)

$n = 9$ patients

$\bar{x} = \frac{54}{9} = 6$ hours

STANDARD DEVIATION: spread of patients

Squared deviations from 6: four × $(-2)^2 + 0^2$ + four × 2^2 = 16 + 0 + 16 = 32 hours².

Sample variance = $\frac{32}{9-1} = \frac{32}{8} = 4$ hours².

Standard deviation $s = \sqrt{4} = 2$ hours.

Describes variation in individual sleep durations.

Sleep durations in the sample (stacked dot plot)



● = one patient

STANDARD ERROR: uncertainty in the mean

$SE = \frac{s}{\sqrt{n}} = \frac{2}{\sqrt{9}} = \frac{2}{3} \approx 0.67$ hours.

SE (standard error) estimates how much sample means would vary across repeated independent samples.

More patients → smaller SE, if variability and sampling design stay comparable.

Hypothetical comparison

Same $s = 2$, but $n = 36$:

$SE = \frac{2}{\sqrt{36}} = \frac{2}{6} = 0.33$ hours.

Patient variability does not automatically shrink with sample size.

95% CONFIDENCE INTERVAL: estimate + uncertainty

For a normally distributed population with unknown standard deviation, use Student's t:

95% interval = $\bar{x} \pm t^* \times SE$

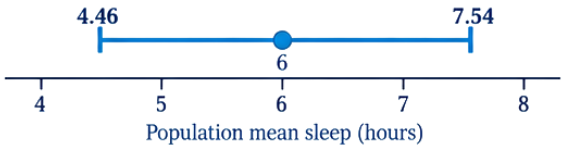
$t^* = 2.306$ for $n - 1 = 8$ degrees of freedom.

$= 6 \pm 2.306 \times (2/3)$

$= 6 \pm 1.54 = 4.46$ to 7.54 hours.

t^* is the two-sided 95% critical multiplier.

95% confidence interval for the population mean



KEY POINTS, INTERPRETATION AND ASSUMPTIONS

Interpretation

If repeated samples were analysed this way, about 95% of the intervals would contain the fixed population mean. This is NOT a range containing 95% of patients.

Assumptions

Independent, representative observations; small-sample t interval assumes an approximately normal population. This tiny sample illustrates arithmetic, not proof of normality.

Larger samples and confidence levels

For large samples the t multiplier approaches 1.96.
Greater confidence gives a wider interval.

What each measure describes

SD describes patients;
SE and the confidence interval describe precision.

Credits and references: retained within the illustration.

Hypothesis testing, errors and power

Practical question: Is the observed change inconsistent with no systematic change?

A fully worked *p*-value

H_0 (null hypothesis): positive and negative changes are equally likely; no systematic direction of change.

H_1 (alternative): the probabilities differ.

Independent patients, no zero changes. Under H_0 each sign has probability 1/2.

Six all-positive signs: $(1/2)^6 = 1/64$.

Six all-negative signs: 1/64.

Two-sided sign-test $p = (1 + 1)/64 = 2/64 =$ **0.03125.**

Numerator **2** counts the two equally extreme sign patterns; denominator 64 = 2^6 possible sign patterns.

With pre-specified $\alpha = 0.05$, **$p < \alpha$: reject H_0.**

α (alpha) is the chosen significance level; p is calculated from the observations.

Example data: six patients with depression scores

Patient	A	B	C	D	E	F
Before	10	12	14	16	18	20
After	9	11	12	14	15	17
Improvement (before – after)	+1	+1	+2	+2	+3	+3

Higher score means worse symptoms.

Decision table: hypotheses and possible outcomes

	H_0 true	H_0 false: specified real effect
Reject H_0	**Type I error:** false alarm. Probability α.	Correct detection. Probability $1 - \beta$ = **power.**
Do not reject H_0	Correct non-rejection. Probability $1 - \alpha$.	**Type II error:** missed effect. Probability β.

β (beta) = chance of missing a specified real effect.
Example design target: $\beta = 0.20 \rightarrow$ power = 1 – 0.20 = 0.80 = 80%.
This is a planning illustration, not power calculated for these six patients.

What *p* means

- Assuming H_0 and the test model, probability of results at least as extreme as observed.
- NOT the probability H_0 is true.
- Non-significant does not prove no effect.

Evidence, size, importance

- $p = 0.03125$ describes statistical evidence.
- Median improvement = 2 points describes magnitude.
- Whether 2 points matters requires clinical context.
- Pre/post improvement alone does not establish a treatment effect.

Power increases with

- Larger sample.
- Larger specified effect.
- Lower variability.
- A stricter alpha usually reduces power, all else equal.
- Plan power before collecting data; address multiple testing.

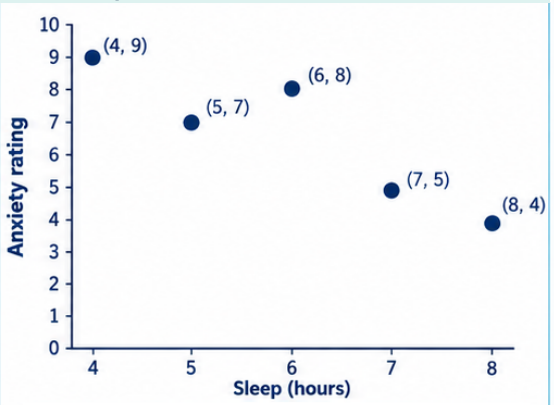
Hypothesis testing, errors and power. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

Correlation

Practical question: Do sleep duration and anxiety tend to vary together?

Scatterplot of the data



One point = one patient; overall downward association.

Clinic dataset (n=5)

Patient	Sleep (hours)	Anxiety rating	Sleep rank	Anxiety rank	d	d^2
A	4	9	1	5	−4	16
B	5	7	2	3	−1	1
C	6	8	3	4	−1	1
D	7	5	4	2	2	4
E	8	4	5	1	4	16

Anxiety is a single ordinal rating; higher means worse.
Rank 1 is the lowest value.
d = sleep rank − anxiety rank. d^2 = squared rank difference.
Sum of d^2 = 38; n = 5 paired observations.

Pearson vs Spearman correlation

Pearson r

- Uses numerical values; measures LINEAR (straight-line) association.
- Sensitive to influential outliers.
- Normality is not required to calculate r; conventional small-sample inference has additional distributional assumptions.

Spearman ρ (rho)

- Pearson correlation of RANKS; measures MONOTONIC association (overall consistently rising or falling).
- Useful for ordinal data; no normality requirement.
- Less affected by extreme magnitudes, not immune to outliers.

Worked Spearman rank correlation (rho)

No ties in this example:

$$\rho = 1 - \frac{6 \times \sum d^2}{n(n^2-1)} = 1 - \frac{6 \times 38}{5 \times (25-1)} = 1 - \frac{228}{120} = 1 - 1.9 = -0.90.$$

- Strong negative rank association in this tiny sample; not a population certainty.
- With ties, assign average ranks and correlate the ranks; the shortcut needs adjustment.

Interpretation of a correlation coefficient

−1	perfect negative (variables vary in opposite directions)
0	no association of the type measured
+1	perfect positive (variables vary in the same direction)

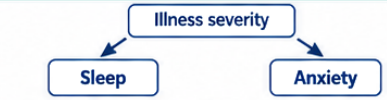
Sign = direction; magnitude = strength.

Important caution

Zero Pearson correlation can hide a strong U-shaped relationship.

Always inspect the scatterplot.

Possible explanation, not established



Reverse causation, confounding, selection and chance can produce association. Correlation alone cannot establish cause.

Final note

A coefficient is an effect size; its p-value is not clinical importance.

Independent paired observations and adequate sample size matter.

Correlation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Biostatistics and epidemiology.

Credits and references: retained within the illustration.

AREA 09 OF 12 · P1-A10

Research methodology

6	5.3%	I-79	I-84
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 5

REVISION ORDER

Reliability and validity first, three of the other five lean on it. Sampling and sample size next. The qualitative slot and the research-question slot after that, both of them arguments rather than lists. The survey slot and the registry slot are independent, and each carries a second half that has to be prepared separately from the first.

WHAT IS IN THIS AREA

Qualitative research set against quantitative. Reliability with its types, and internal against external validity. Sampling with the principles of sample-size calculation for two designs. The research question with the types of hypothesis and a critique of meta-analysis and network analysis. The National Mental Health Survey with Firth's penalised logistic regression. The clinical trials registry with the steps to be completed before a trial opens.

I - 79

What do you understand about qualitative research? How does it differ from quantitative research in terms of essential features, strength and limitations? [2+3+3+2]

2 + 3 + 3 + 2 | RESEARCH METHODOLOGY | DNB - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	**Both, defined**	Meaning and process against magnitude and frequency
4	**The grid**	Question, sampling, data, analysis, rigour
2	**Strengths and limits**	What each buys and what each cannot say
2	**Choosing**	The question decides, and mixed methods

DOMAIN	QUALITATIVE	QUANTITATIVE	WHAT SETTLES IT
The question	What is it like, how does it work, why does it happen	How many, how much, how strongly associated	**Does the question ask for meaning or for a number**
Sampling	Purposive, small, continued until saturation	Probability based, size fixed by a power calculation	**Saturation against a power calculation**
Data	Interviews, focus groups, observation, documents	Measurements, scores, counts	Words against numbers
Analysis	Coding into themes, framework analysis, grounded theory	Statistical tests and effect estimates	Interpretive against inferential
Rigour	Credibility, transferability, dependability, confirmability	Internal validity, external validity, reliability, precision	**Trustworthiness against validity**
Output	Theory and thick description, transferable not generalisable	A population estimate with a confidence interval	Whether a number can be quoted from it

Amber cells are the discriminators an examiner is checking for. The rest of the column supports them.

- **Strengths of the qualitative arm.** It reaches what a questionnaire cannot: illness explanation, treatment refusal, stigma, family decision-making. It generates the hypothesis that a later trial will test, and it explains why a trial that worked elsewhere failed here.
- **Its limits, stated honestly.** No prevalence, no effect size, no generalisation to a population. It is labour intensive, and the analyst is part of the instrument, which is why reflexivity, triangulation, member checking and an audit trail are demanded in its place.
- **Mixed methods.** Sequential where qualitative work builds the instrument a survey then uses, or explains a trial result; concurrent where both run inside one study and are integrated at interpretation.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **Saturation named against power.** It is the sampling discriminator and most answers miss it.
- **The four trustworthiness criteria named,** rather than a claim that qualitative work has no rigour standard.
- **Mixed methods offered as the resolution,** not as a hedge.

SEE ALSO Slot I - 80 reliability and validity Slot I - 81 sampling and sample size SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 80

What is reliability? Discuss the different types of reliability with examples. What is internal validity and external validity of a study? [2+4+4]

2 + 4 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Reliability defined	Consistency, and how it differs from accuracy
4	The four types	Each with its statistic and one example
2	Internal validity	Selection, information, confounding
2	External validity	Generalisability, and the trade-off between the two

THE DEFINITION

Reliability is the consistency of a measurement: the same instrument on the same unchanged subject gives the same answer again. Validity is accuracy, whether the instrument measures what it claims. A scale that always reads five kilograms heavy is perfectly reliable and wholly invalid, which is why reliability is necessary and never sufficient.

TYPE	WHAT IT ASKS	STATISTIC	EXAMPLE
Test-retest	Is the score stable over time in a stable subject	Correlation coefficient	The same scale re-given after two weeks
Inter-rater	Is the score independent of who rates it	Cohen's kappa for categories	Two clinicians rating one interview
Internal consistency	Do all items measure one construct	Cronbach's alpha, split-half	Odd items correlated with even items
Parallel forms	Are two versions interchangeable	Correlation between forms	Two equivalent versions given in turn

Split-half correlations are corrected upward by the Spearman-Brown formula; alpha is the average of all possible split halves.

- **Internal validity.** Whether the association observed inside this study is real for the people studied. It is threatened by selection bias, information and measurement bias, confounding, and chance. Randomisation, blinding, standardised measurement and adjustment are the defences.
- **External validity.** Whether the finding transfers to other patients, settings and times. It is judged from the sampling frame, the inclusion and exclusion criteria, the setting and the comparator used.
- **The trade-off.** They pull against each other. A tightly criteria-bound trial buys internal validity by excluding the comorbid, elderly and substance-using patients who fill an actual clinic, and pays for it in external validity. Internal validity is judged first: a biased study does not generalise, it only travels.

WHAT EARNS THE MARKS

- **Reliability separated from validity in the first two lines,** with the consistently-wrong example.
- **Four types, each with a statistic and an example.** The statistic is the mark most answers drop.
- **Internal validity given as its three named threats,** not as a definition alone.
- **The trade-off stated,** with internal validity judged first.

SEE ALSO Slot I - 49 psychometric properties of a scale Slot I - 79 qualitative and quantitative research SPRINT Day 7, research methods, biostatistics and epidemiology

Reliability and Validity

Reliability: are measurements consistent?

Measurement validity: does evidence support what the score is intended to mean?

A scale that always adds 5 kg is consistent but inaccurate. Reliability alone does not establish validity.

TYPES OF RELIABILITY

Type	Plain meaning	Example (depression)	Common statistic
Test–retest	Same instrument, different times	Stable patient: similar depression scores two weeks apart.	Intraclass correlation for continuous scores.
Inter-rater	Different assessors, same interview	Two psychiatrists independently give similar ratings.	Cohen's kappa for categories; intraclass correlation for scores.
Internal consistency	Related items work together	Items intended to assess depression show related responses.	Cronbach's alpha or omega; high alpha does not prove one dimension.
Parallel forms	Equivalent versions give comparable results	Two versions of a memory test produce comparable scores.	Correlation plus assessment of agreement.

MEASUREMENT VALIDITY

Type	Plain meaning	Example (depression)
Face validity	Does it look relevant?	A depression scale asks about low mood; appearance alone is weak evidence.
Content validity	Does it cover the intended domain?	Expert review checks coverage of mood, sleep, interest and other relevant symptoms.
Criterion validity — concurrent	Agreement with a credible criterion measured now	Compare a depression screen with an independent diagnostic interview.
Criterion validity — predictive	Predicts a relevant future outcome	Baseline risk score predicts later relapse.
Construct validity	Behaves as theory predicts	Convergent: relates to another depression measure. Discriminant: remains distinguishable from different constructs.

STUDY VALIDITY - a different level from validity of a scale

INTERNAL VALIDITY

Is the study's conclusion credible for the people studied?

Example: treatment groups differ because of treatment rather than baseline severity or biased assessment.

Protect with appropriate randomisation, blinding, sound measurement and control of confounding.

EXTERNAL VALIDITY

Does the finding apply to other people, settings or times?

Example: do trial results apply to routine clinic patients with comorbidities?

Judge recruitment, eligibility, setting and similarity to the target population.

A study can have strong internal validity but limited applicability. Improving one does not automatically worsen the other.

Reliability and Validity. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-80.

Credits and references: retained within the illustration.

I - 81

Describe different type of sampling in psychiatric research. What is the sample size? Describe principle and essential components of sample size calculation for any two types of research designs? [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The sampling tree	Probability against non-probability, named methods
2 Sample size	The smallest n that can answer the question
4 Two designs	A prevalence survey and a two-arm trial
1 Inflation	Attrition, and the cluster design effect

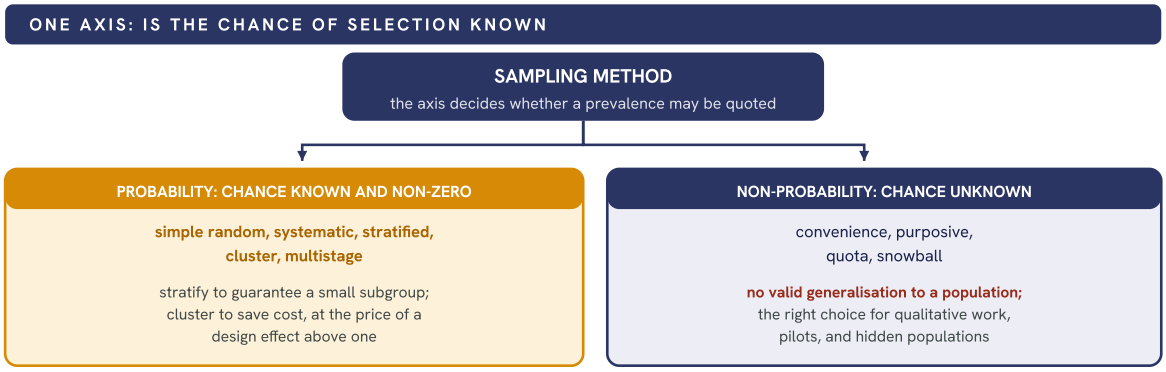


Fig 081.1 Sampling divided on the one axis that matters, whether the probability of selection is known, with the branch that permits a population estimate in amber.

TWO DESIGNS, THE COMPONENTS SPELLED OUT

A prevalence survey needs the expected proportion, the absolute precision wanted either side of it, and the confidence level, giving the familiar form n equals Z squared times p times one minus p, divided by d squared. A two-arm trial comparing means needs the smallest difference worth detecting, the standard deviation of the outcome, the significance level, usually five per cent two-sided, and the power, usually eighty or ninety per cent.

- **The principle.** Sample size is the smallest number that gives an acceptable chance of detecting an effect that is real and worth detecting. It is fixed before recruitment, from the primary outcome only, and never revised because the result was disappointing.
- **Then inflate it.** Add for expected attrition, and multiply by the design effect in any cluster or multistage design. An unadjusted cluster sample is over-precise and its confidence intervals are too narrow.

WHAT EARNS THE MARKS

- **The axis stated before the list,** known against unknown probability of selection.
- **Power and significance level both named,** with their conventional values.
- **The design effect,** which separates a competent answer from a memorised formula.

SEE ALSO Slot I - 79 qualitative and quantitative research Slot I - 82 research question and hypothesis **SPRINT** Day 7, research methods, biostatistics and epidemiology

Sampling and Sample Size

Sampling = how participants are selected. **Sample size (n) = how many participants are included.**
Example: select students from a college to study depression.

PROBABILITY SAMPLING
known, non-zero selection chances

Simple random	Draw student IDs randomly from the full list.
Systematic	Random starting point, then every 10th student; avoid periodic patterns.
Stratified	Divide by year of study, then randomly sample within every year.
Cluster	Randomly select classes, then assess students within selected classes.
Multistage	Randomly select colleges, then classes, then students.

NON-PROBABILITY SAMPLING
selection chances not known

Convenience	Recruit students who are readily available.
Consecutive	Include every eligible clinic attendee during a defined period.
Purposive	Select participants with relevant experiences for in-depth interviews.
Quota	Fill preset subgroup numbers without random selection.
Snowball	Participants refer others from a hard-to-reach group.

Probability sampling supports design-based population estimates; coverage and nonresponse still matter.
Non-probability samples need caution about representativeness.

Planning: choose primary outcome and design → specify precision OR meaningful difference → calculate n → allow for clustering and missing follow-up.
More precision, more variability or more power usually needs more participants. A larger sample does not fix selection bias.

PREVALENCE SURVEY - estimate a proportion

$$n = \frac{Z^2 \times p(1-p)}{d^2}$$

n: total completed responses
Z: confidence multiplier (1.96 for 95%)
p: expected proportion (0.50)
d: absolute margin of error (0.05 = 5 percentage points)

Example (students and depression):

$$n = \frac{1.96^2 \times 0.50 \times 0.50}{0.05^2} = 384.16 \rightarrow \textbf{385 students}$$

Simple random sample approximation for a large population; finite populations may need correction.

TWO-ARM TRIAL - compare mean depression scores

$$n \text{ per group} \approx \frac{2\sigma^2(Z_{1-\alpha/2} + Z_{1-\beta})^2}{\Delta^2}$$

σ: expected standard deviation = 10
Δ: meaningful mean difference = 5 points
α: two-sided significance level = 0.05 ($Z = 1.96$)
power $1 - \beta = 80\%$ ($Z = 0.84$)

Example (depression score trial):

$$n \text{ per group} \approx \frac{2 \times 10^2 \times (1.96 + 0.84)^2}{5^2} = 62.72 \rightarrow \textbf{63 per group}$$

Equal independent groups, common variance; normal approximation.

Allow for 20% loss: divide by 0.80, then round UP (63/0.80 → **79 per group**). For clustered designs, account for design effect before loss allowance.

Sampling and Sample Size. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-81.

Credits and references: retained within the illustration.

I - 82

How to generate research question? Describe different types of hypothesis in research. Discuss advantages and disadvantages of meta analysis and network analysis. [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Generating the question	Where it comes from, then the two frameworks
2	Types of hypothesis	Null and alternative, directional, simple, complex
4	Meta-analysis	What pooling buys, and what it cannot repair
2	Network meta-analysis	Indirect comparison, and the assumption it rests on

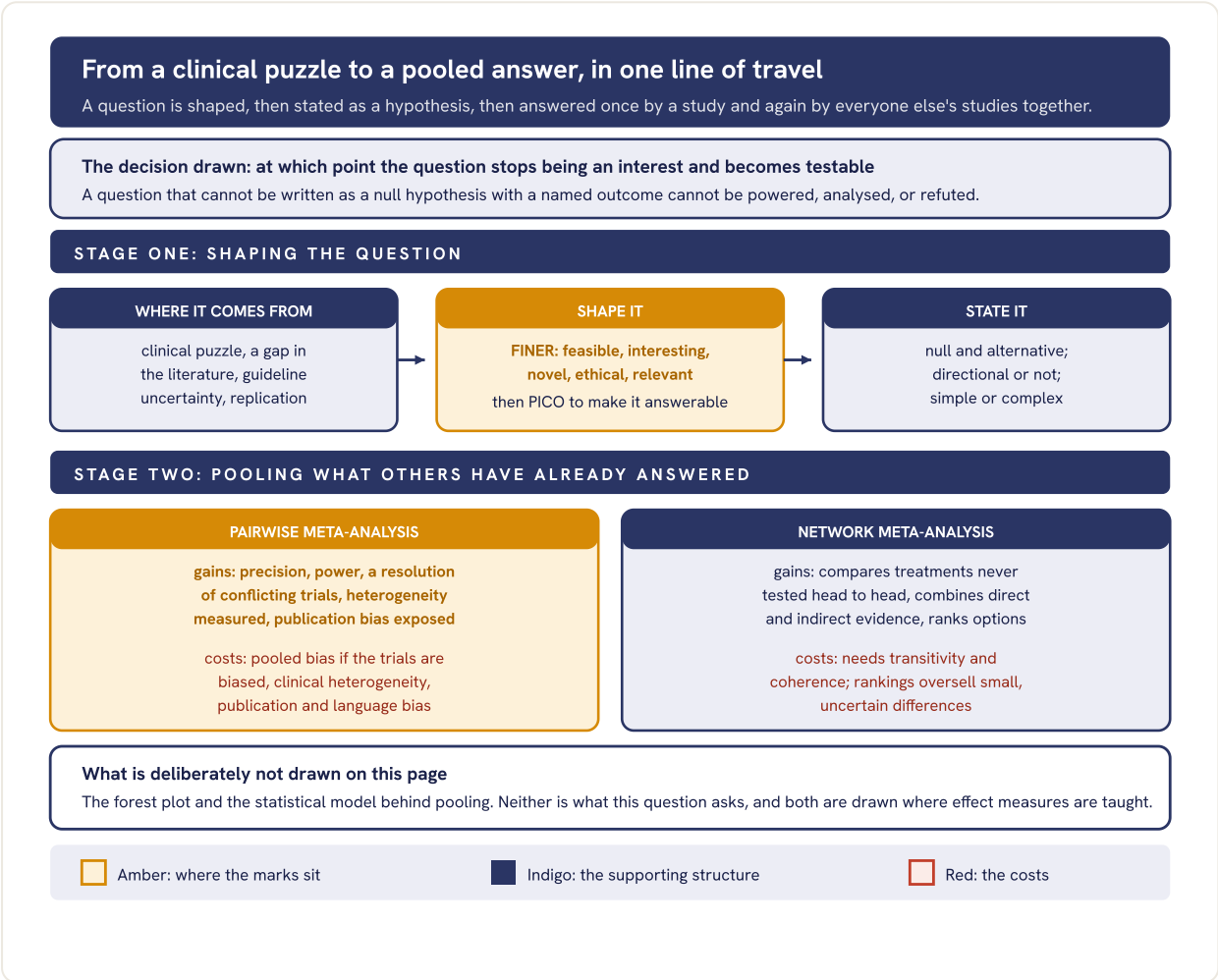


Fig 082.1 The question shaped and stated in the upper band, then answered a second time by synthesis in the lower band, with gains and costs kept apart inside each method.

- **The hypothesis types.** Null against alternative; directional, tested one-sided, against non-directional, tested two-sided; simple, one predictor and one outcome, against complex, several of either; and the research hypothesis, written in words, against the statistical hypothesis, written about parameters.
- **The other reading.** If the examiner means symptom network analysis rather than network meta-analysis, say so and answer briefly: symptoms modelled as nodes with partial correlations as edges, with central symptoms proposed as treatment targets, and stability of the estimated network the standing criticism.

WHAT EARNS THE MARKS

- **A named framework for shaping the question,** not a paragraph on curiosity.
- **Null and alternative written out,** with one-sided and two-sided attached to directionality.
- **Gains and costs kept in separate lists for each method,** as the plate keeps them.
- **Transitivity named** as the assumption a network analysis stands or falls on.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 84 the clinical trials registry and pre-trial steps **SPRINT** Day 7, research methods, biostatistics and epidemiology

Systematic review and meta-analysis

All examples are hypothetical and for educational purposes only.

A transparent, reproducible approach to summarising evidence to inform clinical decisions

1 Focused question (using PICO)

- **Population:** adults with bipolar disorder in remission
- **Intervention:** adjunct structured psychoeducation
- **Comparator:** usual care
- **Outcome:** relapse over 12 months

2 Protocol before review

- Specify eligibility criteria (inclusion and exclusion)
- Define outcomes and methods
- Register where appropriate (e.g. PROSPERO, International Prospective Register of Systematic Reviews)
- Document and justify any amendments

3 Comprehensive search

- Search multiple relevant databases (e.g. MEDLINE, Embase, PsycINFO, Cochrane Library)
- Search trial registries (e.g. ClinicalTrials.gov, Word Health Organizationl Trials Registry Platform
- Check reference lists
- Seek unpublished evidence where possible (e.g. theses, conference abstracts)
- Use reproducible search terms and record dates for all searches

4 Study selection

- Deduplicate records
- Independently screen titles and abstracts
- Retrieve and screen full texts
- Resolve disagreements (e.g. discussion or third reviewer)
- Record reasons for exclusion at full-text stage
- Present flow of studies with counts (e.g. PRISMA flow diagram)

5 Extract data and appraise

- Use duplicate or checked data extraction
- Extract study characteristics (e.g. population, intervention, comparator, outcomes, follow-up)
- Record effect estimates (e.g. risk ratio, mean difference) and missing results
- Use design-appropriate risk-of-bias tools (e.g. Cochrane Risk of Bias 2 for randomised trial results)

6 Decide synthesis

Are studies sufficiently compatible and data usable?

YES

Meta-analysis

- Statistical pooling of suitable estimates (e.g. random-effects model, if appropriate)
- Justify chosen model and methods

NO

Structured synthesis without meta-analysis

- Group and compare findings transparently
- Explain why pooling is inappropriate
- Do not merely count statistically significant studies

7 Interpret and draw conclusions

- Report effect size(s) with 95% confidence interval(s)
- Assess heterogeneity (variation beyond sampling error) and explore possible causes
- Consider risk of bias, missing evidence and certainty of evidence
- Use **GRADE (Grading of Recommendations Assessment, Development and Evaluation)** for outcome-level certainty where appropriate
- Judge clinical importance and applicability to the target population

Key concepts

Systematic review (SR) = Explicit, reproducible methods to identify, appraise and synthesise evidence.

Meta-analysis (MA) = Statistical technique combining results from multiple studies.

Important distinction = A systematic review may have no meta-analysis. **A pooled estimate alone is not a systematic review.**

Reasons not to pool data in a meta-analysis

- Important clinical or design differences between studies (e.g. different populations, interventions, comparators).
- Incompatible outcomes or time points (e.g. relapse at 6 months vs 12 months).
- Too few usable estimates.
- Unusable data (e.g. insufficient information to calculate an effect estimate).

Heterogeneity

- Important heterogeneity requires investigation (e.g. check clinical and methodological differences, consider subgroup or sensitivity analyses).
- Do not automatically pool highly heterogeneous studies.
- Do not automatically reject meta-analysis; pooling may still be appropriate with a justified approach.

Reporting

- **Follow PRISMA 2020 (Preferred Reporting Items for Systematic Reviews and Meta-Analyses)** to enhance transparency and complete reporting.
- PRISMA 2020 supports transparency but does not guarantee the validity or reliability of the review.

All examples are hypothetical and for educational purposes only.

Systematic review and meta-analysis. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-82.

Credits and references: retained within the illustration.

I - 83

Discuss various findings of National Mental Health Survey of India 2016. What is Firth's penalized logistic regression used in this survey? [6+4]

6 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the survey was	Design and scope, before any number
4	The findings	Burden, the treatment gap, what it cannot say
3	Firth's method	The problem it solves in a rare-outcome model
1	Why it matters here	Rare disorders in small subgroups

WHAT IS ASKED	WHAT THE SURVEY REPORTED	WHY IT MATTERS
Design and scope	A cross-sectional multistage stratified random household survey across twelve states, run in 2015 to 2016	**Known selection probabilities, so a national estimate is legitimate**
Current burden	Weighted current prevalence of any mental disorder about 10.6 per cent	Roughly one adult in ten is ill now
Lifetime burden	Lifetime prevalence near 13.7 per cent	The denominator for any service projection
Treatment gap	**Above 80 per cent overall, and widest for substance use**	The policy headline, and the reason the survey is quoted at all
What it cannot say	These are prevalence figures, a stock of existing cases	Prevalence is roughly incidence times duration, so it cannot show whether new cases are rising

FIRTH'S PENALIZED LOGISTIC REGRESSION

Ordinary logistic regression fits by maximum likelihood, which is biased in small samples and breaks outright under separation, where a predictor perfectly or almost perfectly divides the outcome and the estimated odds ratio runs to infinity with an unusable standard error. Firth's method adds a penalty term to the likelihood, an invariant prior on the parameters, which removes most of the small-sample bias and always returns a finite estimate with a usable interval.

- **Why a prevalence survey reaches for it.** Individual disorders are uncommon and subgroup cells are small, so a model of a rare disorder against a rare exposure can easily contain a zero cell. The stem states the survey used it; what it buys, in general, is a finite and interpretable odds ratio where the ordinary fit would give none.
- **Read the survey as a design, not a table.** The examinable point is that this design, cross-sectional, multistage and probability sampled, is what makes those percentages quotable at all. A convenience sample of clinic attenders could not have produced a national figure whatever its size.

WHAT EARNS THE MARKS

- **The design named before the numbers,** cross-sectional and multistage.
- **Current and lifetime prevalence given as two separate figures,** with the treatment gap third.
- **Separation named** as the specific failure Firth's penalty repairs.
- **The prevalence-is-not-incidence line,** which is the honest limit of the whole dataset.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 75 regression analysis SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 84

What is clinical trial registry of India? Describe its role in research. What are the steps to be followed before starting a clinical trial in your hospital? [2+3+5]

2 + 3 + 5 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Who runs it, since when, and its standing
3	Its role	Transparency, selective reporting, publication
4	The steps	Protocol, ethics, regulator, registration, consent
1	The line	Nothing is enrolled before all of it is done

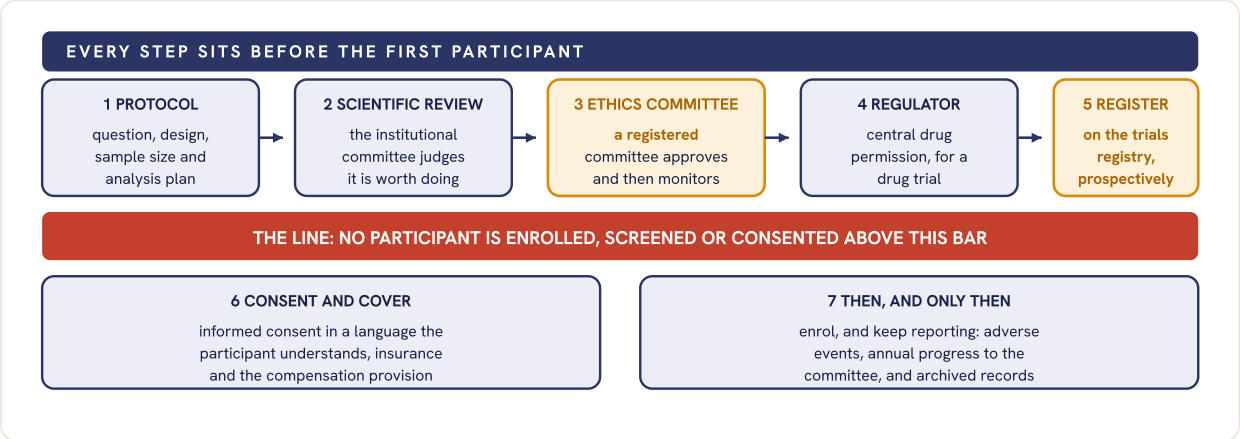


Fig 084.1 The pre-trial sequence with the enrolment bar in red, because the marks in the second half of this question are for putting each step on the correct side of it.

- **What the registry is.** The Clinical Trials Registry of India, launched in 2007 and run by the national medical statistics institute under the Indian Council of Medical Research. It is a primary registry of the World Health Organization platform, and prospective registration has been mandatory since June 2009.
- **Its role.** A public record of what was planned before any result existed, which makes selective outcome reporting and quiet abandonment visible, and which journals now require.
- **The Indian frame.** Human research is governed by the national ethical guidelines of 2017, a registered ethics committee being mandatory; a drug trial adds the new drugs and clinical trials rules of 2019.

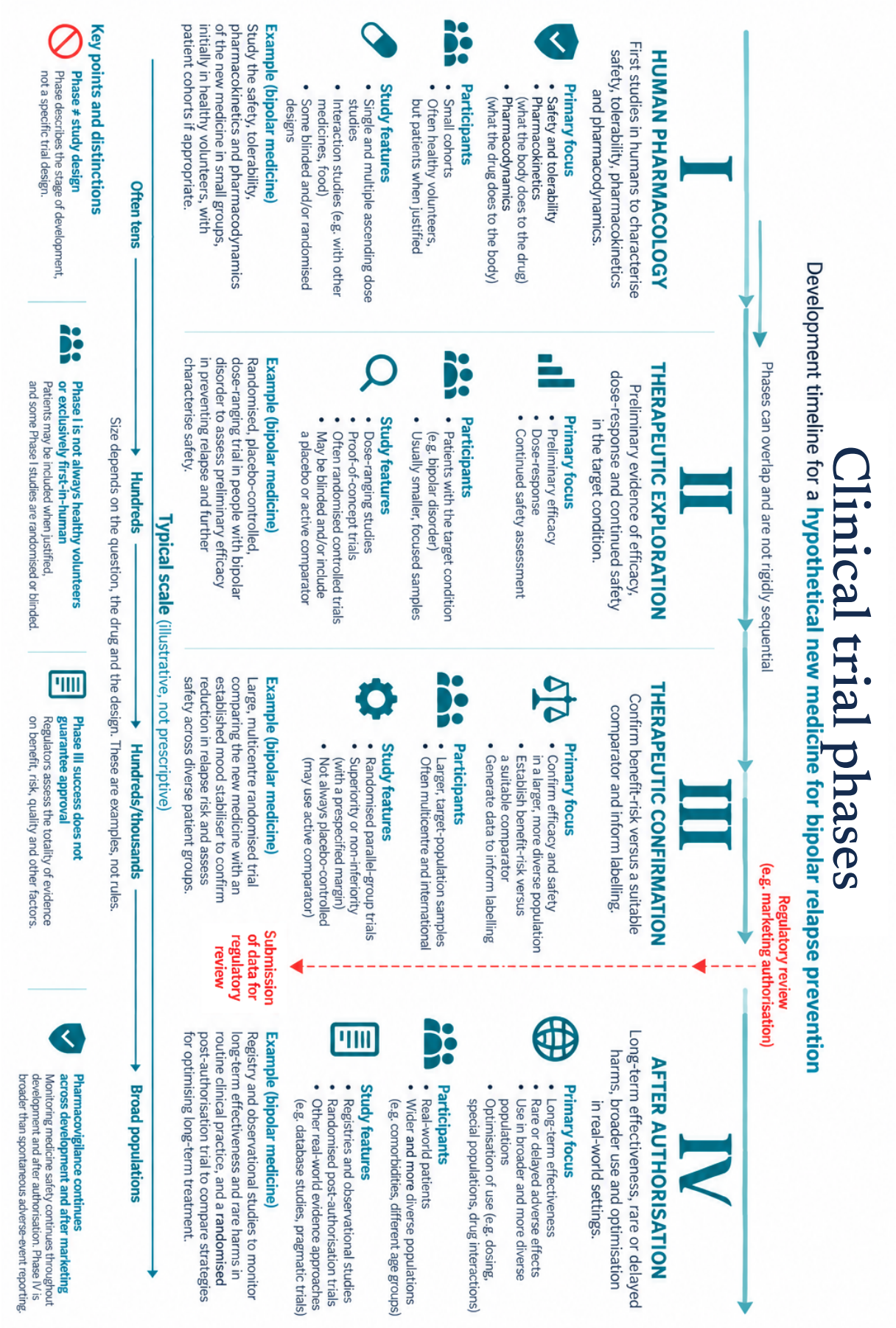
PITFALL

Registering after recruitment has begun. Retrospective registration defeats the purpose of a registry, is refused by most journals, and cannot be repaired later.

WHAT EARNS THE MARKS

- **The registry placed institutionally,** who runs it and under whose platform.
- **Prospective registration named as the rule,** with the reason it exists.
- **Steps in order with the enrolment line drawn,** and both the 2017 guidelines and the 2019 rules cited for a drug trial.

SEE ALSO Slot I - 82 research question and hypothesis Slot I - 81 sampling and sample size SPRINT
Day 7, research methods, biostatistics and epidemiology



Clinical trial phases. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-84.

Credits and references: retained within the illustration.

Research ethics and informed consent

ETHICAL FOUNDATIONS

Respect for persons / autonomy
- Free choice and self-determination
- Additional protection when decision-making capacity is impaired or when persons are vulnerable

Beneficence and non-maleficence
- Social and scientific value
- Minimise possible harm
- Proportionate benefit-risk balance

Justice
- Fair recruitment and inclusion
- Equitable distribution of burdens and benefits

Integrity
- Sound scientific methods
- Transparent registration and reporting
- Disclosure and management of conflicts of interest

CONSENT IS A PROCESS

1 Explain in understandable language
- Purpose of the research
- Procedures and randomisation
- Duration and follow-up
- Risks and uncertain benefits
- Alternatives and usual care
- Confidentiality and its limits
- Payments or reimbursement
- Care for research-related injury and compensation
- Contacts for questions or concerns

2 Check understanding and decision-specific capacity
- Use teach-back (ask the person to explain in their own words)
- Assess understanding, appreciation, reasoning and ability to communicate a choice
- Capacity may fluctuate
- A signature alone is insufficient evidence of understanding or capacity

3 Ensure voluntary choice
- Decision must be free from coercion, pressure or undue inducement
- Refusal or withdrawal must not compromise usual clinical care
- Allow time and opportunity to discuss with family or others if the person wishes
- **Avoid excessive payments or other undue inducements**

4 Document agreement and give a copy
- Written informed consent (or documented verbal consent where permitted)
- Provide a copy of the information sheet and signed consent form
- Record the consent discussion in research records

5 Revisit consent as circumstances change
- Reassess if there are changes in procedures, risks, clinical state or new information
- Seek ongoing consent at appropriate intervals
- Confirm continued willingness to participate

Example (hypothetical):
A randomised trial of group psychoeducation for bipolar disorder. Both groups continue to receive usual clinical care.

PSYCHIATRIC VULNERABILITY
- Mental illness does not automatically mean incapacity.
- Assess understanding, appreciation, reasoning and ability to communicate a choice; capacity may fluctuate.
- Support decisions with accessible explanations and time; reassess when clinical state changes.
- Family permission does not replace a capable adult's consent.
- For someone unable to consent, apply legally required representative consent and additional safeguards; respect dissent and seek the person's own consent when capacity returns.
- Indian Mental Healthcare Act 2017, section 99 adds State Mental Health Authority permission and nominated-representative consent, subject to statutory conditions, for specified interventional research in a person unable to consent who does not resist.

Proxy consent alone is not sufficient.

PRIVACY AND CONFIDENTIALITY
- Privacy concerns access to the person; confidentiality concerns the handling of information.
- Conduct private interviews in a quiet and safe setting.
- Use coded identifiers with a separate key.
- Restrict access to identifiable data to authorised personnel only, and store data securely (physical and electronic).
- Share only appropriate de-identified data for research or regulatory purposes.
- Explain limits of confidentiality, for example:
  - where there are serious safety concerns (risk of harm to self or others)
  - when required by law (e.g. court order, mandatory reporting).
- Coded or re-identifiable data are not anonymous.

ETHICS OVERSIGHT
- An independent Ethics Committee (EC) reviews protocol scientific validity, benefit–risk balance, informed consent, fair selection of participants, vulnerability and privacy **BEFORE** recruitment.
- Continuing review, amendments and safety reports are required throughout the study.
- Waiver of consent requires EC approval and applicable criteria; it is not a researcher decision.
- Therapeutic misconception; research aims to generate knowledge; personalised benefit is not guaranteed.
- Example (hypothetical): In a bipolar psychoeducation trial, participants may be randomised to psychoeducation plus usual care or usual care alone. Both groups retain usual bipolar care; declining to join the research does not remove clinical care.

INDIA UPDATE

Indian Council of Medical Research (ICMR): National Ethical Guidelines 2017 with applicable addenda. New Drugs and Clinical Trials Rules 2019, as amended, apply to regulated studies. Register trials with Clinical Trials Registry–India before first enrolment. ICMR 2026: single ethics review currently covers non-regulatory multicentre biomedical/health research, subject to law; retain site-specific safeguards and oversight.
Global reference: World Medical Association Declaration of Helsinki 2024.

Verified 14 September 2026

Research ethics and informed consent. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-84.

Credits and references: retained within the illustration.

Randomised controlled trial

Example: adults with bipolar disorder in remission – adjunct structured psychoeducation versus usual care

1. Focused question

In adults with bipolar disorder in remission, does adjunct structured psychoeducation reduce relapse over 12 months compared with usual care?

2. Protocol

- Prespecified methods
- Prespecified relapse definition (e.g. meeting diagnostic criteria for a mood episode requiring a changein treatment)
- Primary outcome at 12 months

3. Ethics approval and prospective trial registration

- Ethics committee approval
- Prospective trial registration (e.g. Clinical Trials Registry–India) before recruitment

4. Recruitment and informed consent

- Approach eligible participants
- Provide written and verbal information
- Obtain informed consent
- Baseline assessment

Key methodological safeguards

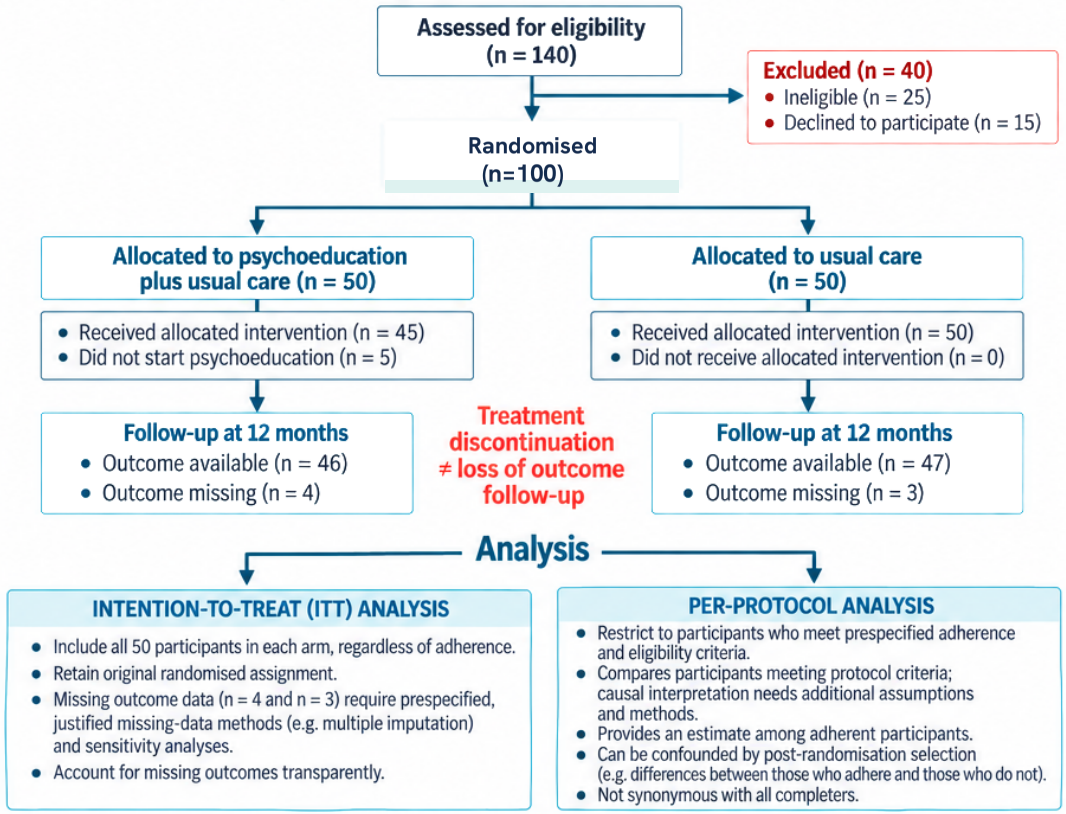
Random sequence generation
e.g. computer-generated random numbers (unpredictable sequence).

Concealed allocation
e.g. central randomisation or sequentially numbered, opaque, sealed envelopes (SNOSE).

Blinded outcome assessment
Use blinded assessors when participant blinding is impractical (e.g. psychological interventions).

Equal follow-up
Similar follow-up procedures and timing in both arms.

Prespecified analysis
Analysis plan prespecified in protocol and registry (e.g. primary analysis, handling of missing data, sensitivity analyses).



Additional considerations

Prespecified outcome
Risk of relapse over 12 months using a clear, objective definition.

Balance at baseline
Randomisation balances prognostic factors in expectation; chance imbalance can remain. Report baseline characteristics.

Harms
Systematically collect and report adverse events in both arms.

Transparency
Follow CONSORT (Consolidated Standards of Reporting Trials) guidelines.

Cautions

- Per-protocol estimates can be misleading due to post-randomisation selection.
- Missing data can bias results if not handled appropriately.
- ITT is not guaranteed to be conservative.
- Interpret findings in the context of clinical importance, not statistical significance alone.

ITT estimates the effect of assignment; it does not magically remove missing-data bias or always give a conservative result.
Report effect size, confidence interval, harms and clinical importance.

Randomised controlled trial. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-84.

Credits and references: retained within the illustration.

Study designs compared

		Selection and enquiry diagram	Measures	Strengths	Limitations
OBSERVED EXPOSURE	**Cross-sectional** Sample clinic attendees, assess psychoeducation participation and current mood episode at one visit (snapshot).	**Sample adults with bipolar disorder attending clinic (one time point)**  Snapshot of exposure and outcome at the same time At one visit assess: • Prior participation in structured psychoeducation • Current mood episode (depressed, manic or mixed vs not)	• Prevalence of current mood episode • Prevalence ratio (comparing those with vs without psychoeducation participation)	• Quick and relatively inexpensive • Can assess several exposures and several outcomes	• No clear temporality between exposure and outcome • Reverse causation possible (current mood state may influence participation in psychoeducation)
	Case-control Select relapse cases and controls from the same eligible source population over a defined period. Look back to prior psychoeducation participation.	**Select from same eligible population (e.g. clinic patients over past two years)** Cases (relapse during study period) Controls (no relapse during study period)  Look back to prior psychoeducation participation (from records or recall)	• Exposure odds ratio (odds of prior psychoeducation in cases compared with controls)	• Efficient for rare outcomes; relapse is not necessarily rare • Can assess multiple exposures	• Recall bias (particularly if relying on participant recall) • Control selection bias • Usual sample cannot directly provide incidence or risk
	Cohort Enroll adults with bipolar disorder in remission, group by observed psychoeducation exposure (yes or no), and follow for 12 months to assess relapse.	**Adults with bipolar disorder in remission (grouped by observed psychoeducation exposure)**  Participated in psychoeducation Did not participate in psychoeducation Follow for 12 months New relapse (yes or no)	• Risk (incidence of relapse over 12 months) • Risk ratio (comparing exposed with unexposed) • Risk difference (absolute difference in risks)	• Clear temporality (exposure measured before outcome) • Can assess multiple outcomes	• Confounding (non-random exposure) • Loss to follow-up (attrition) • More expensive and time-consuming than cross-sectional or case-control designs
ASSIGNED INTERVENTION	**Experimental randomised trial** Consenting eligible adults with bipolar disorder in remission. Random allocation to psychoeducation plus usual care versus usual care alone. Follow for 12 months.	**Eligible adults in remission (consenting)**  Random allocation Psychoeducation plus usual care Usual care alone Follow for 12 months New relapse (yes or no)	• Risk (incidence of relapse over 12 months) • Risk ratio (comparing intervention with control) • Risk difference (absolute difference in risks)	• Supports causal inference when well conducted • Randomisation balances known and unknown confounders in expectation	• Ethical and practical constraints • Adherence (uptake and ongoing participation) • Loss to follow-up (attrition) • Difficult to blind participants to the intervention

Key exam distinctions and reminders

- Retrospective cohort: exposure-based groups and subsequent outcomes are reconstructed from past records.
- A case-control study is selected by outcome (cases and controls) and looks back to prior exposure (this is an arrow of enquiry, not a causal arrow).
- Temporality alone does not prove causation.
- Randomisation balances confounders in expectation, not perfectly in every sample.
- Non-randomised interventions are often called quasi-experimental.
- A snapshot endpoint equals current episode prevalence; a longitudinal endpoint equals new relapse.
- This is a hypothetical teaching example, not evidence from an actual trial.

Study designs compared. Read across the labelled contrasts to distinguish the related concepts. Section companion: Research methodology.

Credits and references: retained within the illustration.

Bias in research

Bias is a systematic error in an estimate, distinct from random error (which is due to chance).

Shared question: Does adjunct psychoeducation reduce 12-month relapse in adults with bipolar disorder?

Type of bias	How it occurs	Hypothetical bipolar psychoeducation example	Prevention
Selection bias	Entry or comparison groups differ in outcome-related ways.	Enrolling only regular clinic attenders can distort the association if attendance is related to both receiving psychoeducation and to risk of relapse.	• Define the source population. • Use representative recruitment. • Use appropriate controls. • Use concealed random allocation in trials.
Information bias	Exposure or outcome measured inaccurately or differently between groups.	Assess relapse more thoroughly in the psychoeducation group (e.g. more frequent follow-up), leading to different detection of relapse.	• Use standardised definitions. • Use trained, masked assessors. • Use comparable, validated measures in all groups.
Types of information bias: **Recall bias**	Past exposure is remembered differently by outcome status (e.g. cases vs controls).	Participants who relapse may remember their prior psychoeducation session attendance differently from those who do not relapse.	• Use prospectively recorded attendance (e.g. registers). • Use the same recall period for all participants.
Types of information bias: **Interviewer bias**	Researcher expectations influence questioning or recording of information.	An unblinded interviewer probes usual-care patients more for relapse symptoms, leading to higher reported relapse in that group.	• Use standardised interview scripts. • Train interviewers. • Use masked assessment of outcomes.
Attrition bias	Missing outcomes are related to the outcome and possibly the group.	Participants who relapse disproportionately disappear from follow-up (e.g. stop responding to contact), leading to an underestimate of relapse.	• Maintain regular contact with participants. • Collect outcome data even after the intervention stops. • Prespecify methods for missing data and conduct sensitivity analyses.

Attrition bias can produce selection bias

A larger sample reduces random error but does not repair systematic bias. → **Randomisation does not prevent** measurement or attrition bias. → **Intention-to-treat analysis alone cannot recover missing outcomes.**

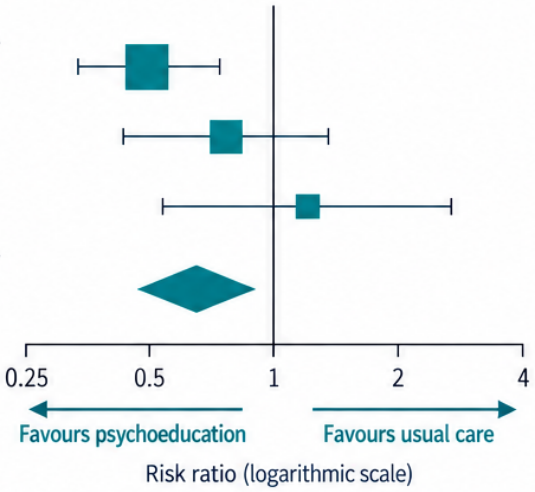
Bias in research. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Research methodology.

Credits and references: retained within the illustration.

Forest plots and heterogeneity

Example: psychoeducation vs usual care for bipolar disorder – 12-month relapse (fewer relapses is better)

Study	Risk ratio (95% confidence interval)	Weight
A	0.50 (0.34 – 0.74)	59.0%
B	0.80 (0.44 – 1.44)	26.2%
C	1.20 (0.55 – 2.63)	14.8%
Pooled (fixed effect)	**0.64 (0.48 – 0.87)**	100%



Illustrative fixed-effect calculation; not treatment evidence.

How to read a forest plot

1. **Identify the measure and which direction benefits.**
   - Here the effect measure is risk ratio (RR).
   - The null value for a ratio is 1.
   - Values < 1 favour psychoeducation (fewer relapses); values > 1 favour usual care.
   - (For a difference, the null value is 0.)
2. **Square = study estimate.**
   - The area of the square shows the study's weight (precision), not study quality.
3. **Line = 95% confidence interval (CI).**
   - The horizontal line shows the 95% CI.
   - If the CI crosses 1, the result is statistically compatible with no relative effect.
4. **Diamond = pooled effect.**
   - The centre of the diamond is the pooled estimate.
   - The width of the diamond shows the 95% CI.
5. **Pooled CI excluding 1 indicates statistical significance at the conventional two-sided 5% level.**
   - This does not imply clinical importance.
   - It does not rule out bias or other limitations.

Fixed-effect model

Assumes one common true effect across all studies.

- Each study is weighted by the inverse of its variance:

$$w = 1/SE^2$$

- For a risk ratio, SE is the standard error (SE) of the log risk ratio (lnRR).
- More precise estimates (smaller SE) carry more weight.
- Appropriate when studies are estimating the same underlying effect and are sufficiently similar.

Random-effects model

Assumes a distribution of related true effects across studies.

- Includes within-study uncertainty and between-study variance (tau-squared, τ^2).
- Gives relatively more weight to smaller studies than a fixed-effect model.
- Estimates the mean of a distribution of true effects.
- Often produces a wider confidence interval.
- Not a cure for incompatible studies or bias.
- Model choice follows the review question and assumptions, not only a heterogeneity test.

Heterogeneity

Differences in participants, interventions and methods can cause variability beyond sampling error.

- I-squared (I^2) estimates the proportion of observed variability attributable to between-study heterogeneity rather than sampling error. It is not the percentage of studies that disagree.
- For this example: Q = 4.55, df = 2.
  $I^2 = \max(0, (Q - df) / Q) \times 100 = \max(0, (4.55 - 2) / 4.55) \times 100 \approx 56\%$.
- Q is the heterogeneity statistic and df the degrees of freedom.
- With only 3 studies this estimate is highly uncertain.
- Investigate clinical and methodological reasons for heterogeneity.
- A prediction interval can describe the dispersion of true effects when estimable.
- Avoid overclaiming an absolute benefit from the risk ratio alone; baseline risk is required to estimate absolute effects.

Forest plots and heterogeneity. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Research methodology.

Credits and references: retained within the illustration.

Critical appraisal and reporting guidelines

From a clinical question to trustworthy and applicable evidence

1 Focused question and appropriate design?



- Population – who is studied?
- Intervention or exposure – what is done?
- Comparator – what is it compared with?
- Outcome – what is measured?
- Time – over what period?

→ Does the study design match the question (e.g. randomised trial, cohort study)?

2 Comparable groups?



- Randomisation and allocation concealment (for randomised trials).
- Or, plausible control of confounding (for non-randomised studies).
- Are groups similar at baseline on important prognostic factors?
- Any major imbalances?

3 Trustworthy measurement and follow-up?



- Masked (blinded) and objective outcome assessment where possible.
- Are the measurement methods valid and reliable?
- How many outcomes are missing?
- Is there differential attrition between groups?
- Could missing data bias the results?

4 Credible analysis?



- Were key outcomes prespecified?
- Is the target effect clear: assignment to treatment or adherence to treatment?
- Is the effect estimate reported with a 95% confidence interval (CI)?
- Is there adjustment for multiplicity (multiple comparisons)?
- Any evidence of selective reporting?

5 Meaningful results?



- Consider both relative and absolute benefit (e.g. absolute risk reduction, number needed to treat).
- Consider harms and adverse effects.
- Are the results precise (95% CI)?
- Are the effects clinically important, not just statistically significant?
- Statistical significance alone is insufficient.

6 Applicable here?



- Is the baseline risk similar to your patients?
- Are the setting and healthcare context comparable?
- Do the participant reflect your population (e.g. age, comorbidity, severity)?
- Is the intervention feasible in your service?
- What are the costs and resource implications?
- Do the results align with your patients' priorities and values?

Key reporting guidelines

Guideline	Full name	Scope (study design)	Key reporting elements
CONSORT 2025	Consolidated Standards of Reporting Trials	Randomised trials	Allocation methods, participant flow, outcomes, harms and analysis.
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology	Cohort, case-control and cross-sectional studies	Selection, measurement, confounders, missing data and limitations.
PRISMA 2020	Preferred Reporting Items for Systematic Reviews and Meta-Analyses	Systematic reviews with or without meta-analysis	Searches, eligibility, selection flow, appraisal and synthesis.



Reporting guidelines

"Was the study reported transparently and completely?"

- They are not design prescriptions.
- They are not quality scores.
- They are not proof of validity.
- They specify what information should be reported.



Risk-of-bias tools

"Could systematic errors distort this result?"

- Cochrane Risk of Bias 2 (RoB 2) – for randomised trial results.
- Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I) – for non-randomised intervention studies.
- Risk Of Bias In Systematic reviews (ROBIS) – for systematic reviews.

Good reporting enables appraisal but cannot repair flawed methods.

Missing reporting may leave bias uncertain.

Psychiatric example



A trial reports that psychoeducation reduces relapse in bipolar disorder. Ask: do the participants, setting, intervention and outcomes apply to your bipolar clinic?

Even if the study is well reported and at low risk of bias, consider whether it is relevant and feasible in your patients.



GRADE (Grading of Recommendations Assessment, Development and Evaluation) assesses the certainty of a body of evidence for each outcome (e.g. high, moderate, low, very low).
This is different from reporting completeness and from study risk of bias. GRADE considers risk of bias, inconsistency, indirectness, imprecision and publication bias across studies.

Critical appraisal and reporting guidelines. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Research methodology.

Credits and references: retained within the illustration.

Randomisation, allocation concealment and blinding

Different steps. Different purposes. All help to reduce bias in randomised controlled trials.

BEFORE ENROLMENT → **AT ASSIGNMENT** → **AFTER ASSIGNMENT / FOLLOW-UP**

Define eligible population, obtain consent, collect baseline information.

Irreversible enrolment and allocation to a trial arm.

Deliver intervention and follow participants. Assess and analyse outcomes.

Randomisation

Creates a chance-based sequence for assigning participants to trial arms.

Who? Independent statistician or computer program.

Mechanism Generates a random (chance-based) allocation sequence (e.g. 1:1, 1:2) before enrolment, using a predefined method.

Prevents Balances both measured and unmeasured prognostic factors between arms in expectation.
Not guaranteed to achieve exact baseline balance in every trial.

Types of randomisation

Simple randomisation Each participant has an independent chance of any arm (e.g. computer-generated random numbers).

Block randomisation Ensures similar numbers in each arm within completed blocks (e.g. blocks of 4 or 6).

Stratified randomisation Creates separate randomisation sequences within levels of a key **prognostic factor** (e.g. illness severity: mild vs severe).

Allocation concealment

Keeps the upcoming assignment hidden until the participant is irreversibly enrolled in the trial.

Who? Recruiter or enroller must not be able to predict the next assignment.

Mechanism Allocation is revealed only after consent and enrolment, using a secure method.

Prevents Selective enrolment (conscious or unconscious) of participants into a preferred arm, which would undermine the benefits of randomisation.

Examples of appropriate methods

Secure central web-based randomisation system (preferred), with proper safeguards (e.g. audit trail, access control).

Sequentially numbered, opaque, sealed envelopes (alternative if a central system is not available), prepared and safeguarded properly to prevent tampering.

Predictable methods are not adequate

- Alternation (e.g. every other patient) **and** date of birth (e.g. even vs odd) are predictable and are not genuine randomisation or allocation concealment.

Blinding

Keeps the assigned treatment unknown after allocation to specified parties.

Who can be blinded? Participants, therapists or clinicians, outcome assessors, and data analysts (as feasible). State exactly who is blinded.

Mechanism Use of identical placebo or active comparators, masking of treatment details, and separation of roles (e.g. independent blinded assessors).

Prevents Reduces deviations from intended treatment (e.g. differential co-interventions, differential care) and outcome measurement biases (e.g. assessor or participant expectations).

Important points

- Use precise terms (e.g. "participants and outcome assessors blinded"), not ambiguous labels such as "single blind", "double blind" or "triple blind".
- An open-label trial can still conceal allocation and blind outcome assessors.
- Allocation concealment and blinding cannot substitute for each other.

Random sampling vs random allocation

Random sampling ⟷ **Random allocation**

Selects which people are invited to participate (increases generalisability).

Assigns enrolled participants to trial arms (reduces confounding).

They are different steps and both are important.

Hypothetical psychiatric example: bipolar psychoeducation trial

1. A statistician creates a randomisation sequence (e.g. 1:1).
2. After the participant gives consent and is enrolled, the recruiter obtains the allocation only at that point (e.g. via secure web system).
3. The participant and therapist know the psychoeducation sessions (open-label for treatment delivery).
4. An independent assessor, masked to allocation, assesses relapse at follow-up.

Key take-home messages

- Randomisation creates a chance-based sequence and balances measured and unmeasured prognostic factors in expectation (not guaranteed exact balance).
- Allocation concealment prevents selective enrolment.
- Blinding reduces deviations and outcome measurement biases.
- Alternation and date of birth are predictable, not genuine randomisation.
- State exactly who is blinded.
- Concealment and blinding cannot substitute for each other.

Randomisation, allocation concealment and blinding. Use the labelled structures to connect anatomical location with function. Section companion: Research methodology.

Credits and references: retained within the illustration.

AREA 10 OF 12 · P1-A06

Developmental psychology and attachment

6	5.1%	I-85	I-90
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 SINGLE 4

REVISION ORDER

The three Erikson slots together and in that order: the full life cycle first, then the childhood stages, then the biographical slot, which the first two have already written most of. Piaget next, and it is the highest-scoring question in this book. Attachment theory and theory of mind last, and both are short.

WHAT IS IN THIS AREA

Erikson in three slots: the eight stages of the life cycle, the childhood half of them, and the man with his contribution. Piaget's stages of cognitive development. Attachment theory. Theory of mind, with its place in normal development and in disorder.

I - 85

Describe the Piaget's theory and the various stages of cognitive development. [2+8]

2 + 8 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB, KUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The theory	Adaptation, and the four invariants that drive it
5	The four stages	Age, defining operation, and the error still made
2	Clinical use	Explanation, consent, delay, a child's account
1	Criticism	One honest limit, named rather than hinted at

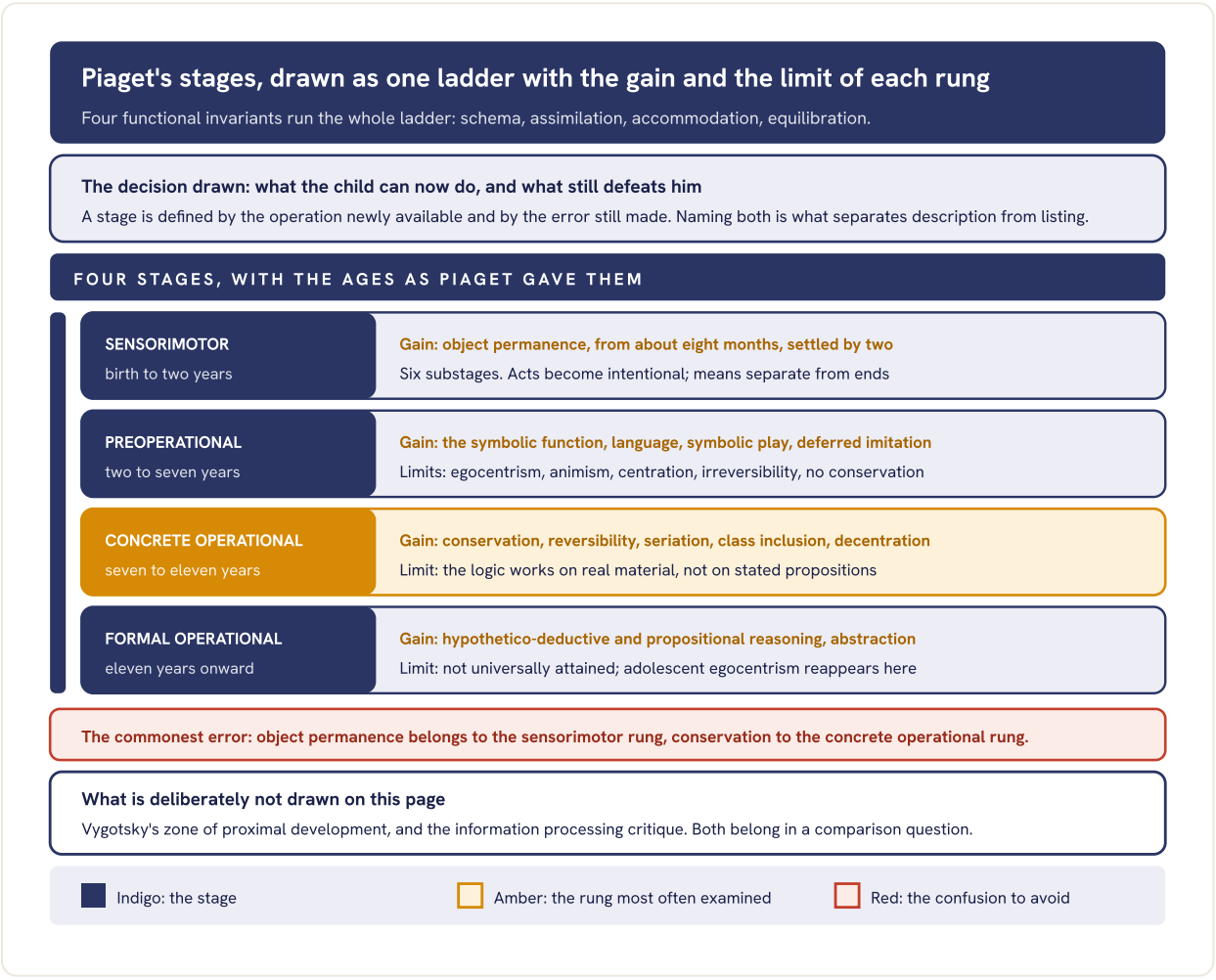


Fig 085.1 The four stages as rungs on a single spine, each carrying its age range, the operation it makes available in amber, and the error that still defeats the child at that rung.

- **The theory, not only the stages.** Intelligence is adaptation. The schema is the unit; assimilation fits experience into an existing schema, accommodation alters the schema to fit experience, equilibration drives the alternation. The sequence is invariant, the rate is not, and each stage is a different logic, not more of the last.
- **Clinical use, and the honest criticism.** The stages set how illness is explained to a child, the threshold for assent, and the weight given to a child's account. Piaget underestimated infants because his tasks demanded motor and verbal responses, underplayed teaching and culture, and formal operations are not universally reached.

WHAT EARNS THE MARKS

- **The four invariants named before the ladder.** Schema, assimilation, accommodation, equilibration.
- **Every rung with its age and its defining operation.** Ages without the operation is where the eight marks go.
- **The limit of each stage, not only its gain.**
- **The red strip.** Object permanence and conservation belong to different rungs.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable Slot I - 88 Erikson's theory of child development

SPRINT Day 4, general and developmental psychology

Lawrence Kohlberg - Moral Development



Heinz's wife is dying. A potentially lifesaving drug is unaffordable.
The pharmacist refuses a lower price or delayed payment.
Should Heinz steal it—and why?



The justification determines the stage, not the yes/no verdict.

PRECONVENTIONAL
Consequences for oneself.

1 Punishment and obedience

"Do not steal: he could go to jail."



Focus: avoiding punishment.

2 Instrumental exchange and self-interest

"Steal: he needs his wife's help and companionship."



Focus: personal benefit and transactional reciprocity.

CONVENTIONAL
Relationships and social expectations.

3 Interpersonal accord (good-boy/good-girl orientation)

"Steal: a good husband saves his wife."



Focus: approval and role expectations.

4 Law and social-order maintenance

"Do not steal: society depends on respect for laws."



Focus: duty and maintaining the system.

POSTCONVENTIONAL
Critically examined rights and principles.

5 Social contract and individual rights

"Laws should protect basic rights; laws that prevent saving life need revision."



Focus: revisable agreements protecting rights.

6 Universal ethical principles
Limited empirical support

"Protect equal human dignity, even against law or majority opinion—and even for a stranger."



Focus: impartial principles of justice and dignity.

Kohlberg's proposed sequence: personal consequences → social expectations → ethical principles ical principles

1 vs 4: punishment vs social order • **3 vs 5:** role approval vs rights • **5 vs 6:** social agreement vs universal principle

Limitations

Reasoning does not guarantee behaviour. Hypothetical dilemmas and verbal skill affect assessment. Original male sample; Western justice emphasis. Carol Gilligan: care and relationships also matter—avoid gender stereotypes. Context affects reasoning; no rigid age timetable or universal attainment.

Method

Moral Judgment Interview: probe reasons across dilemmas; one answer cannot classify a person.

Lawrence Kohlberg - Moral Development. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-85.

Credits and references: retained within the illustration.

Lev Vygotsky - Sociocultural Development

Higher mental functions develop through social interaction and cultural tools, then become individually organised.

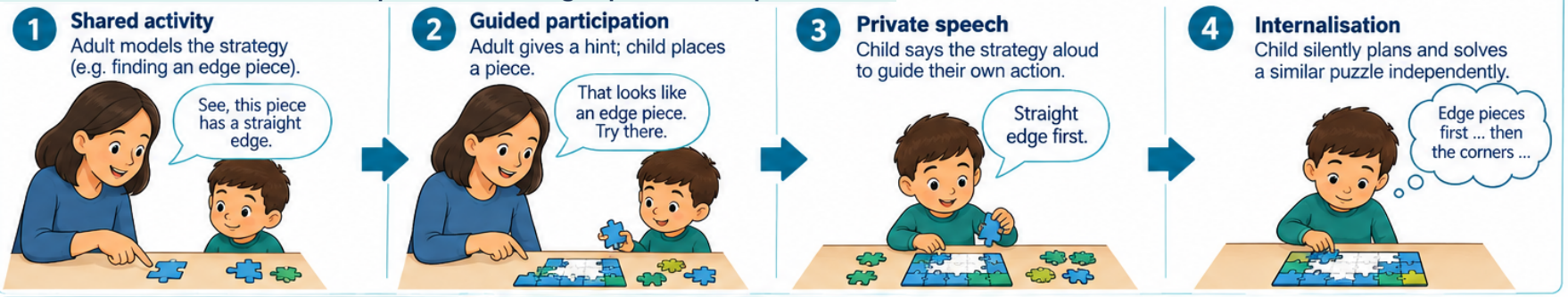
Social mediation

- A parent, teacher or more capable peer supports learning.
- Language, counting systems and cultural practices organise thinking.
- **Example:** Adult: 'Find the straight edges first.'

The zone of proximal development



From social interaction to independent thinking: a puzzle example



Private speech: audible self-talk used to guide attention and action.

Inner speech: internal verbal thought.

Internalisation: transforming socially shared activity into one's own mental processes—not simply memorising instructions.

Scaffolding

Scaffolding = temporary, responsive support that is reduced as competence grows.

Model → prompt → feedback → fade support

Key attribution

Zone of proximal development: Lev Vygotsky.
'Scaffolding': David Wood, Jerome Bruner and Gail Ross, 1976—a later term compatible with this framework.

Key exam distinctions

- Supported success ≠ independent mastery.
- Private speech ≠ immature or meaningless speech.
- The more capable partner may be a peer.
- No universal age-stage ladder.

Limitations

- The zone is task- and support-dependent and difficult to measure precisely.
- Effective support requires active learner participation.
- Private and inner speech can coexist; no rigid age cut-off.

Lev Vygotsky - Sociocultural Development. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-85.

Credits and references: retained within the illustration.

I - 86

Erik H.Erikson - Stages of Life Cycle. [10]

10 ASKED AT 15 MARKS DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

TN-MGRMU, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The frame	Epigenetic principle, and the crisis as a ratio
6 The eight stages	Age, crisis, virtue, and the pathology if unresolved
2 Clinical use	Where the life cycle enters a formulation

STAGE AND AGE	CRISIS	VIRTUE	IF UNRESOLVED
Infancy, to one year	Trust against mistrust	Hope	Withdrawal, suspicion
Early childhood, one to three	Autonomy against shame and doubt	Will	Compulsion, self doubt
Play age, three to six	Initiative against guilt	Purpose	Inhibition, guilt
School age, six to eleven	Industry against inferiority	Competence	Inertia, inferiority
Adolescence, twelve to eighteen	Identity against role confusion	Fidelity	Identity diffusion, repudiation
Young adulthood	Intimacy against isolation	Love	Exclusivity, isolation
Middle adulthood	Generativity against stagnation	Care	Rejectivity, self absorption
Late adulthood	Ego integrity against despair	Wisdom	Disdain, despair

- **The epigenetic principle.** Erik Homburger Erikson held that development unfolds on a ground plan, each stage arising at its proper time and the whole personality being the sum of all eight. A crisis is resolved as a ratio, never as a victory: enough trust to function alongside enough mistrust to stay safe.
- **Where it is used.** Identity work in adolescent presentations, generativity in midlife depression, life review and integrity in late life. Erikson extended Freud's five psychosexual stages across the whole life span and made the conflict social rather than instinctual, which is the sentence that names his contribution.

WHAT EARNS THE MARKS

- **All eight stages, with the crisis written as a pair of opposites,** which is the form Erikson used.
- **The virtue attached to each resolved crisis.** Most answers give the crisis and stop there.
- **The epigenetic principle named.** It is what makes this a theory rather than a table.

SEE ALSO Slot I - 88 Erikson's theory of child development Slot I - 90 Erikson, the man and the contribution SPRINT Day 4, general and developmental psychology

I - 87

Define "Theory of Mind" (TOM). What is the significance of TOM in normal development of children? Describe its significance in various psychiatric disorders. [1+3+6]

1 + 3 + 6 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	The capacity, and who named it in which year
3 Normal development	The timetable, drawn, with the task at each point
6 In disorder	Four conditions, each with the specific failure named

THE DEFINITION

The capacity to attribute mental states, beliefs, desires, intentions and knowledge, to oneself and to others, and to grasp that another person's may differ from one's own. Premack and Woodruff named it in 1978.

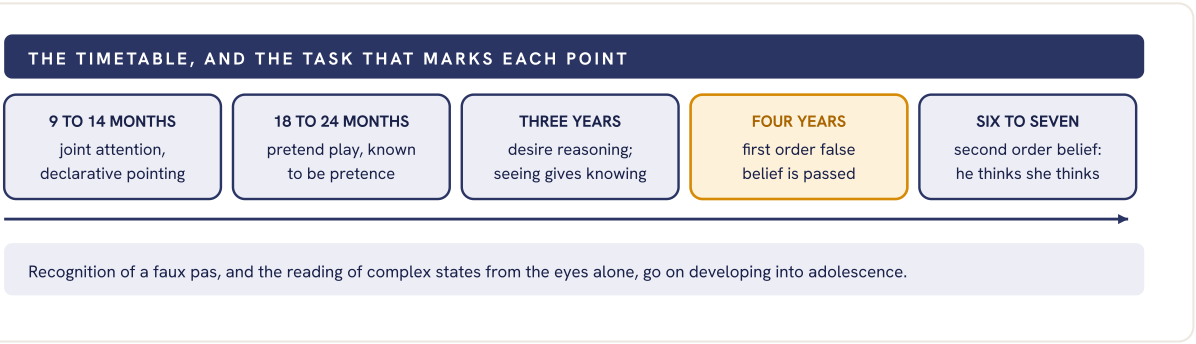


Fig 087.1 The developmental timetable, with the first order false belief task at four years marked in amber because it is the milestone every version of this question turns on.

- **Significance in normal development.** It is what makes deception, persuasion, teaching, keeping a secret, empathy and negotiated friendship possible. The false belief task of Wimmer and Perner is its standard marker, in the Sally-Anne form used by Baron-Cohen, Leslie and Frith.
- **Significance in disorder, named condition by condition.** Autism, where the false belief failure was first shown and framed as mindblindness. Schizophrenia, where mentalising failure tracks persecutory delusions and passivity phenomena. Antisocial personality, where cognitive theory of mind is intact while the affective side is not. It is reported too in attention deficit hyperactivity disorder, in bipolar disorder, in anorexia nervosa and in frontotemporal dementia.

WHAT EARNS THE MARKS

- **The definition in one sentence with its 1978 attribution.** One mark, thirty seconds.
- **The timetable with ages,** and the four year first order task named as the amber milestone.
- **A specific failure per disorder,** not a list of diagnoses said to have a deficit.

SEE ALSO Slot I - 85 Piaget and the stages of cognitive development Slot I - 89 attachment theory **SPRINT** Day 4, general and developmental psychology

I - 88

Erikson's theory of child development. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The departure	Psychosocial against psychosexual, stated first
6 The childhood crises	Five, each with its age and its Freudian parallel
2 What the child needs	What each stage asks of the people around it

CRISIS	AGE	FREUD'S PARALLEL	SEEN IN CLINIC AS
Trust against mistrust	To one year	Oral	Difficulty trusting any caregiver, later any clinician
Autonomy against shame and doubt	One to three	Anal	Control battles; shame carried into adult self doubt
Initiative against guilt	Three to six	Phallic	Inhibited play and questioning; guilt at wanting
Industry against inferiority	Six to eleven	Latency	School refusal, inferiority, giving up on effort
Identity against role confusion	Twelve to eighteen	Genital	Identity diffusion, and the borderline presentations near it

- **How Erikson departed from Freud.** Freud's stages end at puberty and turn on the site of instinctual gratification. Erikson's run the whole life span and turn on a social task set by the widening circle of people around the child. The ego is autonomous and adaptive, not merely the servant of drive and reality.
- **What each stage asks of the environment.** Consistent responsive care in the first year; graded permission to act and to fail in the next two; tolerance of questions and noisy play in the preschool years; real tasks with real feedback through school; and in adolescence a moratorium in which roles can be tried without permanent cost.

PITFALL

The stem asks for child development, so the three adult stages are context and not content. Listing all eight and describing none of them is the commonest way this answer goes wrong.

WHAT EARNS THE MARKS

- **Psychosocial set against psychosexual in the opening lines.** It frames everything after it.
- **Five childhood crises with ages,** each written as a pair of opposites.
- **What the environment must supply at each stage,** which is what turns the table into developmental psychology.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 85 Piaget and the stages of cognitive development

SPRINT Day 4, general and developmental psychology

I - 89

Attachment theory. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The claim	A behavioural system, not a by-product of feeding
3	Development	Four phases, and the separation sequence
3	The patterns	Four classifications, and how each is elicited
2	Clinical use	The disorders, and what a pattern predicts

THE CLAIM

Bowlby held attachment to be a biologically based behavioural system, selected because proximity to a caregiver kept the infant safe. Its set goal is felt security. It is activated by fear, illness and separation, and terminated by contact.

PATTERN	IN THE STRANGE SITUATION	CAREGIVER PATTERN	ADULT INTERVIEW
Secure	Protests separation, is comforted, returns to play	Consistently available and responsive	Autonomous
Insecure avoidant	Little protest, ignores the return, play looks unaffected	Rejecting of distress, pushes independence	Dismissing
Insecure resistant	Intense protest, then angry and clingy, hard to settle	Inconsistent and unpredictable	Preoccupied
Disorganised	Contradictory approach, freezing, apprehension at the caregiver	Frightened or frightening; loss and trauma	Unresolved

- **The developmental frame.** Four phases: preattachment in the first weeks, attachment in the making to about six months, clear-cut attachment with separation protest and stranger wariness from then to two years, and goal corrected partnership after it. Separation runs protest, then despair, then detachment. What is laid down is an internal working model of self and other.
- **Evidence and clinical use.** Harlow's infant monkeys clung to the cloth mother rather than the one that fed them, which separated attachment from feeding. Ainsworth's Strange Situation, eight brief episodes with two separations, is the classification instrument; disorganised was added later by Main and Solomon; the Adult Attachment Interview is the adult counterpart. Clinically: reactive attachment disorder, disinhibited social engagement disorder, separation anxiety, and disorganised attachment as an antecedent of borderline presentations.

WHAT EARNS THE MARKS

- **Attachment named as a system with a set goal,** not as a synonym for a close bond.
- **All four patterns in the grid,** disorganised included, with what elicits each.
- **The instrument named,** Strange Situation for infants, Adult Attachment Interview for adults.
- **The internal working model,** which is what carries the childhood pattern into adult relationships.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable

Slot I - 64 imprinting and the ethological background SPRINT Day 4, general and developmental psychology

I - 90

Eric Erikson. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The man	Dates, training, and the phrase he gave the language
5 The contribution	Four ideas, each stated in its own terms
2 Standing today	Where it is used, and the criticism it carries

THE MAN

Erik Homburger Erikson, 1902 to 1994. He trained in Vienna in the analysis of children, with Anna Freud as his analyst, and held neither a medical nor a doctoral degree. He gave psychoanalysis the whole life span, gave the ego its own developmental line, and gave the language the phrase identity crisis.

- **The four contributions, named separately.** The epigenetic principle, that development unfolds on a ground plan with each part rising at its proper time. The eight psychosocial stages, which carried Freud's five past puberty to the end of life. Ego psychology, in which the ego has its own energies and adaptive tasks rather than serving drive and reality alone. And the ego virtues, one strength issuing from each crisis resolved.
- **Identity, which is his central idea.** Identity is a sense of sameness and continuity within oneself, matched by the recognition of that sameness by others. Its failure in adolescence is role confusion. He described the psychosocial moratorium, a sanctioned delay in which roles are tried without permanent commitment, and identity diffusion as the outcome when it does not close.
- **Psychohistory, and the criticism.** He applied the stage frame to whole lives, in studies of Luther and of Gandhi, arguing that a personal identity crisis and a historical moment can resolve each other. This is the part of the work most criticised, since a single life cannot be a controlled observation, and the stages themselves are hard to falsify.

WHERE ERIKSON IS STILL USED

Adolescent presentations read as identity work rather than as settled trait pathology. Midlife depression framed as stagnation against generativity. Life review in late life and in palliative care, where integrity against despair is the live question rather than a theoretical one.

WHAT EARNS THE MARKS

- **The epigenetic principle and the eight stages named as two contributions,** because they are two.
- **Identity defined rather than invoked.** Sameness within, recognised from without.
- **One honest criticism.** On a named-person stem it is what separates command from recitation.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 34 Anna Freud SPRINT
Day 4, general and developmental psychology

Alexander Thomas and Stella Chess - Temperament

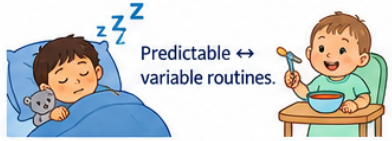
Temperament: early-emerging differences in the style of emotional, motor and attentional responses.
Relatively stable, but shaped by development and experience.

Nine dimensions of temperament (Thomas and Chess)

1 Activity level
Amount of movement.



2 Rhythmicity (regularity)
Predictability of sleep and hunger.



3 Approach–withdrawal
FIRST response to novelty.



4 Adaptability
Adjustment OVER TIME.



5 Intensity of reaction
Strength, not positivity.



6 Threshold of responsiveness
Stimulation needed for a response.



7 Quality of mood
Usual positive/negative emotional tone.



8 Distractibility
How easily outside stimuli interrupt.



9 Attention span and persistence
Duration and continuation despite difficulty.



Patterns of temperament

Historical New York Longitudinal Study patterns;
sample proportions, not population norms.

Easy ~ 40%
Regular, adaptable, generally positive.



Difficult ~ 10%
Irregular, intense, slow to adapt, often negative reactions.



Slow-to-warm-up ~ 15%
Initially withdrawing, low activity and mild reactions; warms gradually.



Mixed/unclassified ~ 35%
Does not fit one pattern.



Goodness of fit: match the environment's demands and support to the child's style.



Cautious child + forced greeting → distress; observe beside parent, then join gradually.



Active child + prolonged sitting → conflict; short tasks and movement breaks.

Key distinctions for exams

Approach = first response;
adaptability = later adjustment.
Threshold = trigger strength;
intensity = response strength.

Dimensions are continua. Patterns are not diagnoses or fixed destinies. Outcomes reflect child–environment interaction; avoid blaming child or caregiver.

Alexander Thomas and Stella Chess - Temperament. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Developmental psychology and attachment.

Credits and references: retained within the illustration.

Margaret Mahler - Separation-Individuation

Historical psychoanalytic theory • approximate ages in the Weave Sprint convention, not screening milestones

Separation: developing a sense of being distinct from the caregiver.

Individuation: developing one's own capacities and individuality.

Precursor
'Normal autistic' phase
• birth – 1 month

Historical claim: largely self-contained, focused on bodily needs.



Obsolete description; newborns are socially responsive. Not autism spectrum disorder.

Precursor
'Normal symbiotic' phase
• 1 – 5 months

Historical claim: infant and caregiver experienced as a dual unity.



Psychological fusion in the theory, not biological fusion.

1
Differentiation ('hatching')
• 5 – 9 months

Growing self–other distinction; explores caregiver's face and attends to unfamiliar people.



2
Practising
• 9 – 16 months

Early: crawling away.
Later: upright exploration.
Emotional refuelling: returning for contact, then exploring again.



3
Rapprochement
• 16 – 24 months

Wants independence and closeness. "I do it!" then "Help me!"
Ambitendency: alternating clinging and moving away.



4
Consolidation
• 24 – 36 months

More stable individuality and emotional object constancy: an enduring, integrated inner image of the caregiver, including loving and frustrating aspects.



Greater stability is typically seen in this period, but development continues beyond these ages.

Exploring the world while holding the caregiver in mind



Emotional object constancy: the relationship feels enduring during absence or frustration.

Object permanence vs emotional object constancy



Object permanence: knowing a hidden object still exists.



Emotional object constancy: maintaining an integrated emotional representation of a person.

Clinical note (exams)

Later psychodynamic links between rapprochement difficulties and borderline pathology are hypotheses, not established causes; avoid caregiver blame.

Key text

The Psychological Birth of the Human Infant (1975): Margaret Mahler, Fred Pine and Anni Bergman.

Limitations: Observations were interpreted through psychoanalytic assumptions. Early fusion claims are challenged by infant research. Ages vary across accounts. Development continues beyond these ranges.

Margaret Mahler - Separation-Individuation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Developmental psychology and attachment.

Credits and references: retained within the illustration.

Language Development

Receptive language: understanding messages.



Expressive language: conveying meaning through words, signs or symbols.



Speech is sound production; language also includes vocabulary, grammar and social use.



Selected Centers for Disease Control and Prevention milestones: skills ≥75% of children show BY each age. These are surveillance checkpoints, not average onset ages or diagnostic tests.

Age	Understanding or communication response	Expression
4 months	Turns toward a familiar voice	Coos and vocalises back
9 months	Responds to own name	Repeated syllables such as 'bababa'
12 months	Understands 'no'	Uses a special name for a parent
15 months	Looks at named objects; follows words plus gestures	Attempts 1–2 words beyond parent names
18 months	Follows a one-step instruction without gestures	Attempts ≥3 words beyond parent names
2 years	Points to named pictures and ≥2 body parts	Combines ≥2 words
3 years	Participates in conversation	≥2 back-and-forth exchanges; asks questions
4 years	Answers simple function questions	Sentences of ≥4 words
5 years	Answers questions about a story	Tells a story with ≥2 events

Typical sequence of speech and language development:
Cooing → babbling → meaningful single words → word combinations → sentences and narrative.

Key theories of language development

Burrhus Frederic Skinner

learning: reinforcement and modelling shape verbal behaviour.
Limitation: imitation alone cannot explain novel grammar.



Noam Chomsky

nativist: an innate language-learning faculty; universal grammar is a proposed set of shared grammatical principles.
The language acquisition device is hypothetical, not an anatomical organ.



Jean Piaget

cognitive: symbolic language develops alongside representational thought.
Limitation: cognition alone does not explain all language learning.



Jerome Bruner and Lev Vygotsky

social interaction: shared attention, responsive dialogue and cultural support develop communication.
Biological capacities and learning processes also matter.





Worked example
'Goed' instead of 'went': applying a learned pattern too broadly, not just copying; does not uniquely prove one theory.

Holophrastic = one word expressing a whole message.
Telegraphic = brief content-word combinations.

Assessment concerns: missing expected skills, poor understanding, limited gestures or response to sound, or caregiver concern → hearing and developmental/language assessment. No word attempts beyond parent names at 18 months or no two-word combinations at 2 years warrants assessment; do not wait until ages 2–3 to act. Any loss of skills requires prompt evaluation.

Milestone failure is not a diagnosis. Assess all languages and communication modes; multilingual exposure does not itself cause language disorder.

Language Development. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Developmental psychology and attachment.

Credits and references: retained within the illustration.

Gross motor: posture, balance and large-muscle movement.

Motor Development

Fine motor: hand–finger control and visual–motor coordination.

Selected Centers for Disease Control and Prevention checkpoints - skills ≥75% show By each age; not average onset or diagnostic cut-offs.

Age	Gross motor	Fine motor and adaptive
4 months	 Head steady; props on forearms	 Holds a toy placed in hand
6 months	 Rolls tummy to back; supports sitting with hands	 Reaches for a wanted toy
9 months	 Gets into sitting; sits unsupported	 Transfers objects; rakes with fingers
12 months	 Pulls to stand; cruises along furniture	 Thumb–index pincer grasp
15 months	 Takes a few independent steps	 Stacks ≥2 small objects
18 months	 Walks independently	 Scribbles; tries a spoon
2 years	 Runs; kicks ball; walks up a few stairs 	 Uses spoon; one hand stabilises while other acts
3 years	—	 Draws a circle after demonstration; strings large beads 
4 years	 Catches a large ball	 Unbuttons; holds pencil with fingers and thumb 
5 years	 Hops on one foot	 Buttons some buttons; writes some name letters

— No selected gross-motor item in this abbreviated row.

Typical sequence of motor development

Head → trunk → standing balance → walking



Whole-hand grasp → thumb–index precission



Cephalocaudal: head toward feet. **Proximodistal:** centre toward extremities. Broad tendencies, not rigid rules.

World Health Organization observed windows (1st-99th percentiles, months)

Sitting without support	3.8 – 9.2
Hands-and-knees crawling	5.2 – 13.5
Standing alone	6.9 – 16.9
Walking alone	8.2 – 17.6

These population windows are not referral thresholds. Crawling can be skipped.

When to be concerned (assessment prompts)

Missing checkpoints warrant developmental review; no steady head at 4 months, no unsupported sitting at 9 months, or no independent walking at 18 months are important prompts. Do not wait if concerned. Skill loss, marked asymmetry or abnormal tone need prompt assessment.

Sources of variation

Consider opportunity to practise, health, vision and cultural experience. For preterm infants, use corrected age in the first 2 years: chronological age minus weeks born before 40 weeks.

Key exam point

Milestone timing alone does not diagnose cerebral palsy or intellectual disability. Assess trajectory, movement quality and all developmental domains.

Motor Development. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Developmental psychology and attachment.

Credits and references: retained within the illustration.

AREA 11 OF 12 · P1-A12

Nosology, classification and descriptive psychopathology

6	4.9%	I-91	I-96
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 6

REVISION ORDER

The three phenomenology slots together: they share one definitional grammar and the marks are in the criteria, not in the length. The two classification slots next, the neo-Kraepelinians first because the revision slot argues from them. The spectrum slot last, and give it time: it is three questions in one.

WHAT IS IN THIS AREA

Three phenomenology slots: delusion with the primary forms and the dimensions, consciousness with its dimensions and quantitative changes, and hallucination with the true against pseudo distinction. Three nosology slots: the neo-Kraepelinians and their influence on the current systems, the purpose of revising DSM and ICD and how far that purpose has been met, and the spectrum concept with the mixed episode and the epilepsy spectrum. Every slot in this area was seen once in the record, which is the whole of what its chips claim.

I - 91

Who are the neo-Kraeplineans? How they influence the current classificatory system? [3+7]

3 + 7 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Who they were**	The St Louis school, and why Kraepelin is the ancestor
2	**What they demanded**	Illness, boundaries, criteria, validation
4	**The influence**	Feighner, the RDC, DSM-III, and what ICD took
1	**The honest limit**	Reliability bought, validity still owed

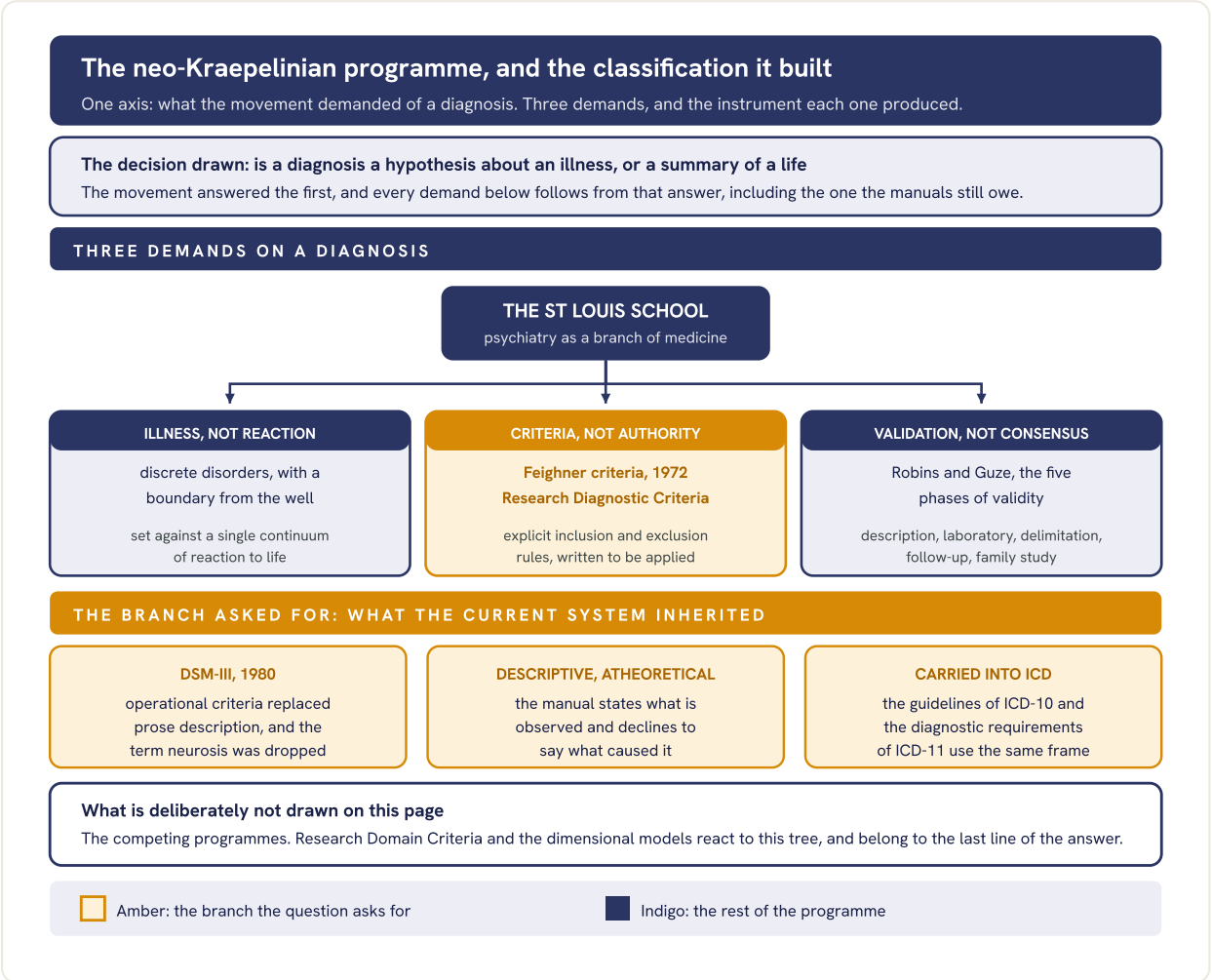


Fig 091.1 The neo-Kraepelinian programme drawn as three demands made on a diagnosis, with the criteria branch opened out into the architecture DSM-III built and both manuals still use.

- **Who they were.** A group working in St Louis from the late 1960s, Eli Robins, Samuel Guze and George Winokur, named neo-Kraepelinian by Gerald Klerman, who set out the movement's credo. Kraepelin is the ancestor because he classified by course and outcome rather than by presumed cause.
- **What it has not delivered.** Reliability improved; validity did not follow. No laboratory finding delimits these categories, comorbidity is the rule rather than the exception, and both manuals have answered by adding dimensions to a frame that stays categorical.

WHAT EARNS THE MARKS

- **The names attached to the movement.** Robins, Guze and Winokur, and Klerman who named it.
- **Operational criteria named as the inheritance,** rather than DSM-III named as an event.
- **The Robins and Guze validators listed.** They are the standard the system is still judged against.
- **The unfinished part stated.** Reliability without validity is the honest close.

SEE ALSO Slot I - 93 the purpose of revising a classification Slot I - 92 spectrum concepts in psychiatry **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

I - 92

Discuss the concept of spectrum disorder(s) in psychiatry with suitable diagrams. How has current understanding of mixed episode developed? What is epilepsy spectrum disorder? [3+5+2]

3 + 5 + 2 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What a spectrum is	Two senses, drawn: one graded condition, one family
2	Why it is claimed	Family, genetic and dimensional evidence
3	Mixed episode	Kraepelin, DSM-IV, the DSM-5 specifier, ICD-11
2	Epilepsy spectrum	What the term covers, and where it stands

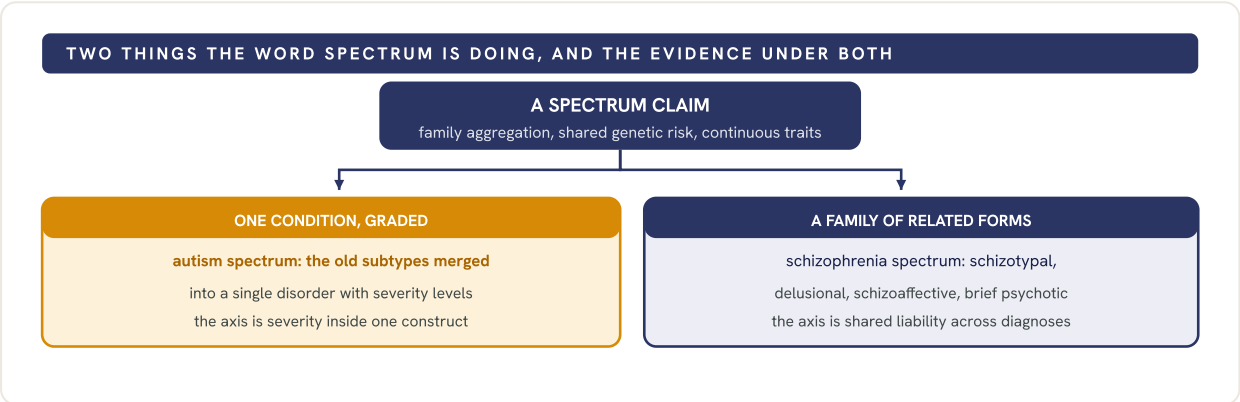


Fig 092.1 The two senses the word spectrum carries in psychiatry, severity graded within one construct on the left and shared liability across neighbouring diagnoses on the right, with the evidence both rest on named at the root.

- **How the mixed episode changed.** Kraepelin drew mixed states from the independent variation of mood, thought and volition. DSM-IV required full criteria for mania and for depression at once, a threshold so rarely met that most mixity went unrecorded.
- **Where the systems now part.** DSM-5-TR carries mixity as a with-mixed-features specifier, three non-overlapping symptoms of the opposite pole. ICD-11 keeps a mixed episode as an episode type. Mixed features predict poorer outcome and higher suicide risk.

WHAT THE THIRD PART IS ACTUALLY ASKING

Epilepsy spectrum disorder names no category in either manual. It is used for the temporolimbic psychiatric picture seen with epilepsy: interictal dysphoric disorder, postictal and interictal psychosis, and the interictal behaviour syndrome of hyperreligiosity, hypergraphia, viscosity and altered sexuality.

WHAT EARNS THE MARKS

- **Both senses of spectrum given, with an example under each.**
- **The mixed episode traced, not merely defined.** Kraepelin, DSM-IV, DSM-5, ICD-11.
- **The specifier stated exactly.** Three non-overlapping symptoms of the opposite pole.
- **The honest line on the third part.** The term names no category in either manual.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 93 the purpose of revising a classification

I - 93

There have been many revisions of diagnostic systems (e.g. DSM, ICD) in psychiatry. In your view, what is the purpose of the exercise of revision and to what extent the purpose has been achieved by DSM-5 and ICD-11? [5+5]

5 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Why a system is revised	Evidence, usability, harm, and the users served
3	What the two did	Regrouping, dimensions, and what was removed
2	Where it was met	Organisation and utility, field tested
2	Where it was not	No validators, comorbidity, threshold creep

PURPOSE OF REVISING	WHAT DSM-5 AND DSM-5-TR DID	WHAT ICD-11 DID	HOW FAR IT WAS MET
Follow the evidence	Chapters regrouped by shared mechanism	The same regrouping; subtypes dropped	**Architecture moved, validators did not**
Raise reliability	Field trials in routine clinics, reported openly	Field studies across many countries	**Measured honestly, and modest**
Serve the user	Kept criteria with counts and durations	Essential features written as prose	**ICD-11 chose the clinician**
Admit dimensions	Severity specifiers; trait model kept apart	Personality rebuilt as severity plus traits	Carried further by ICD-11
Reduce harm	Bereavement exclusion removed	Gender incongruence moved out of the chapter	Met in part, contested in both

- **What a revision is for.** To move the manual closer to nature where evidence allows, to keep it usable by the people who must apply it, and to stop it doing harm. The three pull against each other, so every revision is a compromise.
- **The charge that sticks.** Neither revision produced a diagnostic validator, and comorbidity is still the rule, which is what a valid categorical system should not produce.

PITFALL

Answering with a list of what changed. The stem asks for a purpose and then for a verdict, so every change named has to be tied to the purpose it served and judged against it. A changed manual is not by itself an achieved purpose.

WHAT EARNS THE MARKS

- **Purposes named before any change is described.** The stem asks for a view, and a view needs a criterion.
- **The structural contrast stated,** counted criteria against prose essential features.
- **A verdict per purpose,** rather than one verdict on the whole revision.
- **The missing validator named.** It is the honest answer to the second half.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 92 spectrum concepts in psychiatry

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 94

Define delusion. What are primary delusions? Discuss dimensions of delusions. [2+3+5]

2 + 3 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Three criteria, stated as criteria
3 Primary delusions	Un-understandable, and the forms it takes
4 The dimensions	Each one varying free of the others
1 Why they matter	Insight, function, and where the risk sits

THE DEFINITION

A false belief held with extraordinary conviction and incomparable subjective certainty, not open to counter-argument or to the evidence of experience, and out of keeping with the person's social, cultural and educational background. Jaspers named three external criteria: certainty, incorrigibility, impossibility of content.

- **Delusional mood.** An oppressive sense that something momentous is under way, before any belief has formed. It settles when the delusion crystallises.
- **Delusional perception.** A normal percept given a private and unmediated significance: two members, the percept and the meaning. A first-rank symptom.
- **Sudden idea, and delusional memory.** A belief arriving fully formed with no trigger, one member only; and a real memory recalled as carrying a meaning it never had.
- **Primary against secondary.** A primary delusion is un-understandable: it cannot be derived from mood, personality or circumstance. A secondary one can, and is better called a delusion-like idea.

DIMENSION	WHAT IT RANGES OVER	WHY IT IS ASKED FOR
Conviction	Passing doubt through to unshakeable certainty	**Tracks insight, and predicts response to treatment**
Extension	One corner of life through to every part of it	Predicts functional impairment
Bizarreness	Culturally possible through to physically impossible	**Rated unreliably, so it no longer stands alone as a criterion**
Disorganisation	A single fragment through to a systematised structure	Systematised or fragmentary, and how far it is elaborated
Pressure	An occasional thought through to constant, driven preoccupation	**Later work added distress and action, the item that carries risk**

WHAT EARNS THE MARKS

- **The three criteria named as criteria.** Stopping at false belief is half a mark.
- **Un-understandability given as what makes a delusion primary.**
- **The five dimensions named as a set:** conviction, extension, bizarreness, disorganisation, pressure.
- **Independence stated.** Total conviction with no distress and no action is common.

SEE ALSO Slot I - 96 true and pseudo hallucinations Slot I - 95 consciousness and its disturbances

I - 95

Define consciousness. Write the dimensions and quantitative changes in consciousness. [2+4+4]

2 + 4 + 4 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Awareness of self and surroundings, on two axes
3 The dimensions	Level, content, field, and awareness of self
4 Quantitative changes	The ladder, each rung told from the one above
1 How it is scored	Three subscales, and the total they give

THE DEFINITION

Consciousness is awareness of the self and of the environment. Clinically it runs on two axes that vary independently: the level of arousal, a brainstem and thalamic function of the ascending reticular activating system, and the content of awareness, which is cortical.

- **The dimensions.** Level of arousal, the quantitative axis. Content of awareness, the qualitative axis. The field, which narrows in twilight and dissociative states. And awareness of self: Jaspers' activity, unity, identity over time, and self against not-self.
- **How it is scored.** The Glasgow Coma Scale rates eye opening one to four, verbal response one to five and motor response one to six, giving a total of three to fifteen.

LEVEL	AROUSAL AND RESPONSE	WHAT TELLS IT FROM THE RUNG ABOVE
Full alertness	Awake, attention sustained and directable	The baseline the rest are measured against
Clouding	Threshold raised, attention and comprehension fluctuate	**Fluctuation across hours: the core of delirium**
Drowsiness	Rousable by ordinary speech, then drifts back	Rouses without a physical stimulus
Stupor	Responds only to vigorous or painful stimulation	**Reflex and postural responses are kept**
Coma	Unrousable, no purposeful response to anything	**No sleep-wake cycle at all**

THE WORD STUPOR

Psychiatry and neurology use it differently: in psychiatry akinesis and mutism with consciousness preserved, as in catatonia; in neurology a depressed level of arousal. Rate arousal and awareness separately, as the locked-in syndrome shows.

WHAT EARNS THE MARKS

- **Arousal and awareness separated in the definition itself.**
- **Every rung named with what distinguishes it** from the rung above.
- **Jaspers' four aspects of self-awareness listed,** the part most answers omit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 94 delusion, defined and dimensioned

I - 96

Define hallucinations. How do true and pseudo hallucinations differ? What is their clinical significance? [2+5+3]

2 + 5 + 3 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both, defined**	A percept without an object, and the two exclusions
4	**The grid**	Space, quality, insight, and where each is seen
2	**The disagreement**	Two traditions, and they do not overlap
2	**Clinical significance**	Modality points to cause; not all are illness

THE DEFINITION

A hallucination is a percept without an object: a sensory experience arising with no external stimulus, in external objective space, carrying the force and clarity of a true perception. It is not an illusion, which misperceives something real.

DOMAIN	TRUE HALLUCINATION	PSEUDOHALLUCINATION	WHAT SETTLES IT
Where it is located	External objective space	Inner subjective space	**The space it occupies, Jaspers' test**
Sensory quality	Concrete, as a real percept is	Figurative, without substance	**Concreteness, not vividness**
Insight	Usually absent, held as real	Usually present	**Separates them in the British usage only**
Where it is seen	Psychotic and organic states	Dissociative states, bereavement, trauma	Suggestive, never decisive

Amber marks the discriminators. Both are involuntary, so control settles nothing between them.

- **Two traditions, not one.** Jaspers separates the two by sensory quality and by the space they occupy, not by insight. The later British usage separates them by preserved insight. The two do not pick out the same experiences.
- **Modality points to the cause.** Auditory verbal in schizophrenia; visual with fluctuating consciousness in delirium; olfactory and gustatory in temporal lobe epilepsy; tactile in stimulant use and alcohol withdrawal; complex visual with insight kept in Charles Bonnet syndrome.
- **Not all are pathological.** Hypnagogic and hypnopompic hallucinations, those of bereavement and those of sensory deprivation occur in health.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The two traditions named and told apart.** One definition is not enough here.
- **Modality tied to aetiology,** which is most of the significance mark.
- **Command hallucinations flagged for risk,** and the healthy forms kept out.

SEE ALSO Slot I - 94 delusion, defined and dimensioned Slot I - 95 consciousness and its disturbances **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

Normality

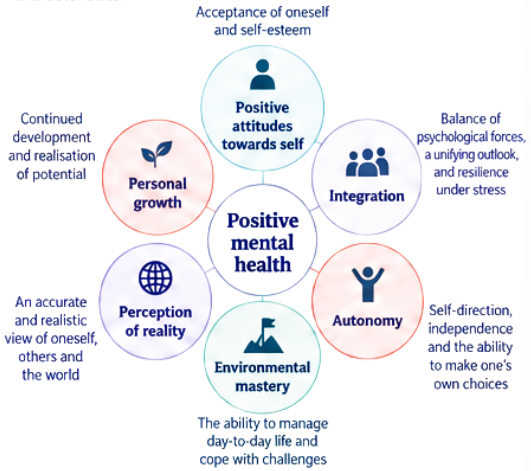
1. Historical perspectives on normality

Daniel Offer and Melvin Sabshin identified four ways of thinking about normality.

Perspective	Key idea	Practical strength	Practical limitation
Normality as health	Normality as the absence of disease or illness.	Highlights the importance of identifying and treating illness.	Absence of illness does not necessarily equate to positive wellbeing.
Normality as utopia	Normality as an ideal state of optimal functioning and human potential.	Provides an inspiring vision of human flourishing.	The ideal may be unattainable and can set unrealistic standards.
Normality as average	Normality as statistical typicality (what is common in a population).	Useful for describing population patterns and variation.	What is rare does not necessarily mean pathological, and what is common does not necessarily mean healthy.
Normality as process	Normality as a dynamic process of adaptation over time.	Recognises change, resilience and the ability to adapt to life's challenges.	Adaptation depends on context and time, and what is adaptive in one situation may not be in another.

2. Marie Jahoda's model of positive mental health

Marie Jahoda proposed that positive mental health involves more than the absence of illness. She identified six key characteristics.



This is a historical conceptual model of positive mental health, not a diagnostic checklist.

3. Modern clinical assessment

Assessment of mental health and disorder considers multiple domains. None alone defines all disorders.



- **Distress** — Subjective psychological suffering (e.g. low mood, anxiety).
- **Dysfunction** — Impact on functioning in daily life (e.g. work, relationships, self-care).
- **Persistence** — Duration and course of symptoms.
- **Risk** — Risk to self or others.
- **Developmental and cultural context** — Age, developmental stage, cultural background and social context.
- **Longitudinal change** — Patterns over time, including recovery and recurrence.

A lack of subjective distress does not exclude the presence of a mental illness.

4. Different ways of being 'abnormal'

Statistical rarity, social deviance and clinical disorder are related but distinct concepts.

Statistical rarity



- Refers to how common or rare a characteristic is in a population.
- What is rare is not necessarily pathological.
- Unusual artistic ability may be rare without any impairment.

Social deviance



- Refers to behaviour, beliefs or lifestyles that differ from social norms.
- Influenced by cultural, moral and historical factors.
- Cultural diversity, dissent, sexual orientation and gender diversity are not pathology in themselves.

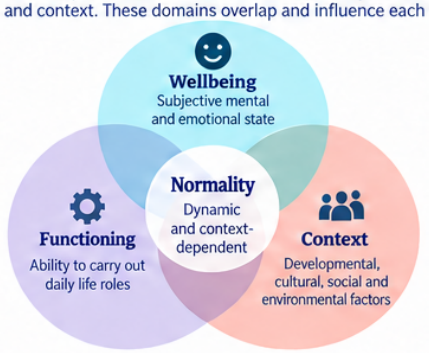
Clinical disorder



- Involves significant distress, dysfunction, persistence and/or risk.
- Assessed in developmental and cultural context.
- Ordinary grief may be painful and distressing without automatically being a mental disorder.

5. Normality is multidimensional

Normality involves an interaction between subjective wellbeing, functioning and context. These domains overlap and influence each other.



- These domains are interdependent and can overlap.
- A person may have high wellbeing despite some difficulties, or may experience distress in response to normal life events.
- Normality involves variation, adaptation and change over time.
- Diagnostic classification is a useful tool for communication and care, but it is not a measure of human worth.

Normality. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Mood and affect terminology

Mood - the person's report

Mood: prevailing emotional state over time, primarily elicited from the person's report.

"I have felt low most days this week."



Affect - what you observe

Affect: observed emotional expression during the interview—face, voice, gesture and changes with topic.

"Tearful, soft voice; briefly smiles when discussing family."



A smile does not exclude depressed mood.

Expression does not directly measure inner feeling.

Describe affect using six dimensions

1. Quality / tone

General emotional character.

Common descriptors:

- **euthymic** (neither persistently low nor elevated)
- **depressed**
- **anxious**
- **irritable**
- **elevated**



"I feel on edge"; tense expression.

2. Range

Breadth of emotional variation.

- **Full** – normal variation across topics.
- **Restricted / constricted** – little variation.



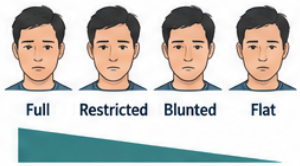
Restricted range (little change) — Full range (variation)

Example: little change across sad and pleasant topics.

3. Intensity

Strength of expression.

- **Blunted** = markedly reduced.
- **Flat** = near-absent.



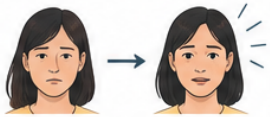
Decreasing intensity of expression

Common shorthand, not a calibrated scale.

4. Reactivity

Change in response to meaningful stimuli.

- **Reactive** – briefly brightens with good news.
- **Non-reactive** – little observable response.



Reactive: briefly brightens with good news.

Different from lability.

5. Congruence / appropriateness

Comparison of affect with reported mood, topic or situation.

Example: laughter during a painful account warrants exploration of embarrassment, culture and context.



Consider embarrassment, culture and context.

6. Stability

Stable vs labile.

- **Stable** – affect is generally consistent.
- **Labile** – rapid, marked shifts, e.g. tears → laughter within minutes.



Lability: tears → laughter within minutes.

Not evidence by itself of bipolar cycling.

Common mood words

Dysphoria	Unpleasant distress or discontent; may include irritability.
Elation / euphoria	Heightened cheerfulness or well-being; meanings overlap across authors, not a fixed severity ladder.
Irritability	Lowered threshold for annoyance or anger.
Anxiety	Apprehensive anticipation of threat; may have an identifiable focus.
Expansiveness	Unrestrained expression and interpersonal overfamiliarity.

Common confusions

Anhedonia	Reduced pleasure. *"Music no longer gives me pleasure."*
Apathy	Reduced motivation or concern. *"I do not feel moved to do anything."*
Alexithymia	Difficulty identifying and describing feelings. *"I cannot tell what I feel."*
Blunted affect	Reduced expression; pleasure or distress can still be present.
Emotional incontinence	Poorly controlled laughing/crying, disproportionate to context or subjective feeling; consider neurological causes.

Framework note

Contemporary usage often grades blunted/flat by reduced expression. Classical descriptions may use "blunting" for coarsened emotional responsiveness. Restricted and constricted are commonly synonymous; describe actual findings.

Worked examination description

"Reports low mood; affect sad, restricted in range, mildly reduced in intensity, reactive to pleasant topics, congruent with the account and stable."

Examination points

Observe throughout the interview. Consider baseline style, culture, medication, parkinsonism and sensory barriers. Mood-incongruent psychotic content is a different judgement from inappropriate affect.

Mood and affect terminology. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Formal thought disorder

Describe how thought is expressed: rate, continuity, organisation and information content.
Findings are inferred from speech, not diagnoses.

Circumstantiality

Unnecessary detail, eventually answers.

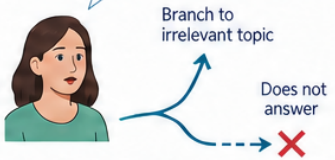
Q: How did you arrive?
"I checked the timetable, missed one bus, waited at the stop, spoke to a friend, walked to another stop, and then I came by bus."



Tangentiality

Oblique or irrelevant reply that does not answer.

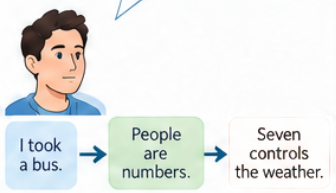
Q: How did you arrive?
"Transport costs too much. Cities need planning."



Derailment

Disrupted links between successive ideas. Not defined only by failure to reach a goal.

"I took a bus. People are numbers. Seven controls the weather."



Disrupted links between ideas

Flight of ideas

Rapid shifts with traceable superficial associations in classical descriptions.

"Bus — traffic — lights — stage lights — I should perform!"



Rapid but linked by superficial associations

Prolixity

Milder, ordered overproductive flow, eventually reaches goal.

Q: How did you arrive?
"Well, I left home this morning, checked the timetable, decided to take the bus, had a coffee while waiting, and then I came by bus."



Ordered, detailed, eventually reaches answer

Rate, quantity and continuity

Pressure of speech	Increased output, hard to interrupt. [rapid] "Wait, I have much more to tell you..."
Slowed thinking	Delayed replies and slowed flow. **[long pause]** "It takes time to think."
Poverty of speech	Little spontaneous elaboration. "Tell me about your day." "Nothing."
Poverty of content	Many words, little information. "Things happened, as things do; it was that kind of day."
Blocking	Sudden interruption with subjective blankness. "I went to the... [stops] My mind is blank."
Distractible speech	External stimulus redirects speech. "I went... is that a bird outside?"

Meaning and words

Incoherence	Speech becomes incomprehensible within clauses or sentences; grammar need not be broken. "Tomorrow eats the silent because."
Illogicality	Conclusion does not follow. "All doctors work; I work, so I am a doctor."
Clang	Sound drives word choice. "Train, rain, brain, plain."
Neologism	Invented word with private meaning. "I feel flintered." [an invented private term]
Word approximation	Unusual understandable construction for a familiar object. "Writing-stick" for pen.
Knight's-move thinking	Abrupt illogical transitions; overlaps with derailment.

Repetition and abstraction

Perseveration	Repeats a prior response despite a new question. "Where are you?" "Hospital." "Your occupation?" "Hospital."
Verbigeration	Stereotyped repetition. "Round we go, round we go..."
Echolalia	Repeats another's speech. Examiner: "How are you?" Reply: "How are you?"
Concrete thinking	Literal interpretation. "Spill the beans" → "Tip beans out of a tin."

Frameworks

Classical teaching separates stream from form.
Nancy Coover Andreasen:: Thought, Language and Communication scale.
Flight of ideas is grouped under derailment with pressured speech.
Tangentiality: answers. Derailment: spontaneous transitions.
Positive: disorganised output. Negative: impoverished output.

Historical anchors

Eugen Bleuler — loosening of associations.
Norman Cameron — asyndesis (missing links) and overinclusion (irrelevant concepts included).
Kurt Goldstein — loss of abstract attitude.

Examination approach

Describe before diagnosing.
Language, culture and neurological language disorders matter.
Blocking alone is not thought withdrawal.

Formal thought disorder. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Thought possession and passivity phenomena

Separate ownership, agency and privacy

Ownership: "This thought is mine."

Agency: "I am initiating it."

Privacy: "Others cannot directly access it."

These can dissociate.

Thought insertion

Thoughts experienced as alien and placed in the mind.



Not merely an unwanted thought.

Thought withdrawal

Thoughts experienced as removed by an outside force.



Not just forgetting or a blank mind.

Thought broadcasting

Thoughts experienced as accessible to others without speaking.



Hearing a voice is not required; ownership may remain.

Passivity / made experiences

An action, urge, feeling or bodily sensation occurs, but is experienced as externally imposed or controlled.

Made action (volition)



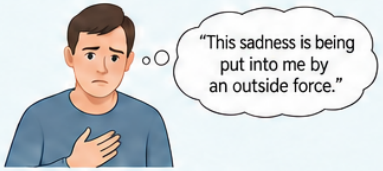
Movement occurs; personal authorship is lost.

Made impulse



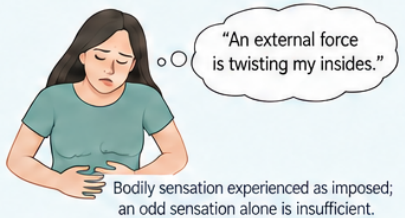
The imposed urge is the finding; it need not be acted on.

Made feeling (affect)



Different from feeling sad because someone upset you.

Somatic passivity



Bodily sensation experienced as imposed; an odd sensation alone is insufficient.

Comparison with related experiences

Related experience	Teaching example	Key distinction
Intrusive thought / obsession	"I hate this thought, but it is my own."	Unwanted does not mean inserted; insight and resistance vary.
Ordinary influence	"My friend persuaded me; I decided to go."	Influence with retained agency.
Depersonalisation	"I feel robotic, but know I am acting."	Detachment is not automatically external control.
Thought blocking	"My mind suddenly went blank."	Sudden interruption; withdrawal adds external removal.
Thought echo	"I hear my own thoughts spoken aloud."	Auditory experience; broadcasting concerns others' access.

Assess without leading

- "Tell me exactly what happens."
- "Does it feel like your own thought?"
- "How do you understand its source?"
- "Do you mean this literally or as a comparison?"

Explore:

- Ownership (is the thought/experience felt to be one's own?)
- Authorship (do they feel they are initiating it?)
- Privacy (do they feel others can access it?)
- Conviction (how strongly do they hold the belief?)
- Cultural context (is the experience explained within their cultural or spiritual framework?)
- Distress (how much does it bother them?)

Do not infer passivity from "out of control" alone.

Exam point

These belong to Kurt Schneider's first-rank framework, but are neither necessary nor sufficient for schizophrenia.

See the separate complete first-rank image.

Thought possession and passivity phenomena. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Kurt Schneider's first-rank symptoms

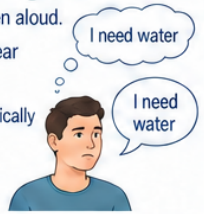
A. SPECIAL AUDITORY EXPERIENCES (3)

Audible thoughts / thought echo

Own thoughts heard as spoken aloud.

"I think 'I need water" and hear those words aloud."

Thought echo often refers specifically to repetition after the thought; audible thoughts is broader.



Voices discussing or arguing

Voices talk about the person.

Voice A: "He is leaving."

Voice B: "He should stay."



Running commentary

A voice narrates actions as they occur.

"She picks up the cup; now she sits down."

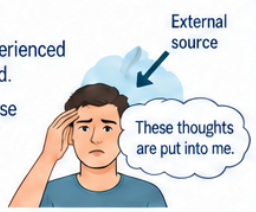


B. THOUGHT ALIENATION (3)

Insertion

Alien thoughts experienced as put into the mind.

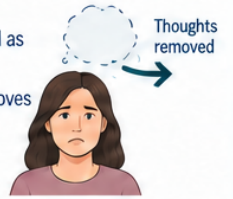
"Someone puts these thoughts into me."



Withdrawal

Thoughts experienced as taken away.

"An outside force removes my thoughts."



Broadcasting

Thoughts directly accessible to others without speech.

"Others know my thoughts even when I am silent."



C. PASSIVITY/MADE EXPERIENCES (4)

Made action

Action experienced as externally controlled.

"Someone else is moving my arm."



Made impulse

Urge experienced as imposed.

"They put the urge to stand up into me."



Made feeling

Emotion experienced as imposed.

"They are making me feel this joy."



Somatic passivity

Bodily sensation experienced as externally imposed.

"An outside force is twisting my stomach."



D. DELUSIONAL PERCEPTION (1)

A real, correctly perceived event acquires an immediate, unwarranted personal delusional meaning.

1. **Real perception**
   (correctly perceived event)

   "The traffic light turns red."



2. **Delusional significance**
   (immediate, unwarranted meaning)

   "This proves I have been appointed ruler."



The percept is **real**; its attributed meaning is abnormal.
Not an illusion or hallucination.

EXAM DISTINCTIONS

- **Command content** alone is **not a first-rank category.**
  A command voice may coexist with other first-rank forms.
- **Broadcasting** = others access thoughts; **thought echo** = hearing thoughts aloud.
- **Blocking alone** does not establish withdrawal.
- **"First-rank"** means historical diagnostic priority, not greatest severity, earliest symptom or a negative symptom.

DIAGNOSTIC LIMITS

- **Neither necessary nor sufficient for schizophrenia.**
  May occur in mood-related, substance-related and medical psychoses.
- Assess the **whole syndrome**, duration, course, mood relationship and medical/substance causes.
- Modern diagnostic systems **do not diagnose schizophrenia from** a first-rank symptom alone.
- See also: Thought possession and passivity phenomena.

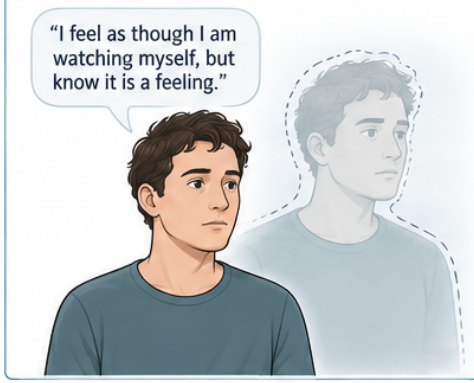
Kurt Schneider's first-rank symptoms. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Disorders of self-experience

SELF FEELS UNREAL (Depersonalisation)

Detachment or unreality concerning oneself, one's body, thoughts, actions or feelings.



Key points

- Detachment or unreality concerning oneself, one's body, thoughts, actions or feelings.
- **Reality testing** about the experience is preserved.
- Symptoms alone do not establish depersonalisation-derealisation disorder.

WORLD FEELS UNREAL (Derealisation)

Surroundings feel unreal, dreamlike, distant or artificial.



Key points

- Surroundings feel unreal, dreamlike, distant or artificial.
- **Reality testing** about the experience is preserved.
- Both depersonalisation and derealisation may co-occur.
- Symptoms alone do not establish depersonalisation-derealisation disorder.

Related disturbance of feeling

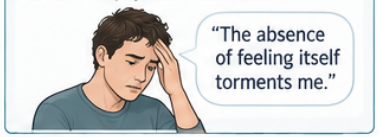
Emotional numbing

Reduced felt emotion.



Anaesthesia dolorosa psychica

Painful awareness of not being able to feel. Not physical anaesthesia.



Ownership, agency and boundaries

Ownership

"This experience belongs to me."



Disturbance:
"This thought is alien, not mine."

Agency

"I am initiating this action."



Disturbance:
"My arm moves, but an outside force is making it."

Ownership and agency can separate

"It is my hand, but the movement is being controlled."



Self-boundaries (demarcation)

Distinction between self and others/world.



Clarify the exact experience and conviction.
Thought insertion, withdrawal, broadcasting and made experiences overlap with this domain; see the two dedicated images.

Distinguish from other experiences

Depersonalisation	"I feel robotic, but know I am acting." **Not** automatically passivity.
External-control experience	"Another agent makes my actions." Explore literal conviction and attribution.
Nihilistic delusion	"I literally do not exist." The wording "as if" alone does not settle reality testing.
Autoscopy	Actually seeing a double of oneself; not simply feeling like an observer.
Déjà vu	Unfamiliar event feels familiar.
Jamais vu	Familiar event feels unfamiliar. Familiarity changes are not synonymous with derealisation.

Historical framework

Karl Jaspers

Analysed activity, unity, identity through time and demarcation.

Christian Scharfetter's five dimensions

Vitality	Activity	Consistency/ coherence	Demarcation	Identity
Sense of being alive/existing.	Sense of initiating experience.	Sense of a unified self.	Self/non-self boundary.	Sense of being the same self.
"My sense of existing is fading."	"My actions no longer feel self-directed."	"I feel fragmented."	"The boundary between me and others feels blurred."	"I no longer feel like the same person."

These are phenomenological dimensions, not one-to-one diagnostic criteria. Continuity belongs with identity; do not substitute it for vitality.

Assess



"What feels changed—yourself, the world, ownership or controll?"

Explore:

- onset, duration, triggers
- reality testing
- distress, function
- sleep, substances and medical context
- Avoid infering a diagnosis from one metaphor.

Disorders of self-experience. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Attention and orientation in the mental state examination

PREPARE

Set the conditions for a fair assessment



Check alertness first.
Explain tasks and confirm understanding.



Use preferred language and an interpreter when needed.



Check hearing, vision, literacy, education and familiarity with numbers or calendars.



Reduce distractions; consider pain, anxiety, fatigue, medication and intoxication.



Record adaptations.
"Not testable" is not "failed".

ASSESS

Test attention and orientation systematically

ATTENTION

Different aspects, with specific tests

	What it involves	How to assess (example)	Key points and limits
Selective attention	Focus on a target despite distraction.	Observe whether conversation is repeatedly diverted (e.g. by background noise or interruptions).	Note if the person can keep to the task despite distractions.
Sustained attention / concentration	Maintain focus over time.	Observe persistence through a task (e.g. months backward or vigilance).	Note ability to continue and effort over time.
Working memory	Hold and manipulate information. Related to attention, but not identical.	See digit span backward and other tasks.	Adds working-memory and executive demands.

Tests with examples and limits

Digit span forward	"Repeat 4–1–7–9."	Tests immediate attention/span.	Use a standard sequence protocol; do not infer a disorder from one trial.
Digit span backward	"Say 4–1–7 backwards." Answer 7–1–4.	Adds working-memory and executive demands.	
Months backward	December → November → October...	Tests sustained attention.	Record starting point, errors, prompts and completion.
Serial sevens	100 → 93 → 86 → 79 → 72 → 65.	Arithmetic and educational demands matter.	Not a pure attention test.
Vigilance	Respond when a target letter is heard in a standard sequence (e.g. "A").	Tests sustained attention.	Requires adequate hearing and comprehension.

ORIENTATION

Explore time, place, person and situation

	What to ask (examples)	Key points
Time	"What is the date today?" "What day is it?" "What month?" "What year?" "What is the approximate time?"	Note exact errors; a missed date alone is not a diagnosis.
Place	"Where are we?" "What is this building?" "Which ward or setting are we in?" "Which town or city?"	
Person	"What is your full name?" "Do you know who I am?" "What is my role?"	Record own identity and recognition of others' roles; record what was asked.
Situation	"Why are you here?" "What has happened?"	Often assessed separately.

Ask open questions before offering choices. Orientation depends partly on attention, memory and available contextual cues.

INTERPRET

Consider the pattern and next steps



New or fluctuating inattention, altered alertness or disorientation → investigate delirium and medical causes promptly.



Use a validated delirium assessment when indicated.
4AT = four-item assessment covering alertness, brief orientation, attention and acute change/ fluctuation.



Intact orientation does NOT exclude delirium, cognitive impairment or medical illness.



"Time → place → person" is a common teaching pattern, not an obligatory sequence.

Time → Place → Person



Poor performance is not proof of low intelligence, unwillingness, dementia or psychosis.



Compare baseline, collateral history and repeated observations; record timing and fluctuation.

DOCUMENT OBSERVATIONS

Keep the factual task performance separate from the interpretation

Example: "Alert; follows conversation. Gives correct month, year and location but not date. Months backward: two omissions, one prompt. Hearing difficulty noted; reassessed with hearing aid."

Record what was done, the person's actual performance, any adaptations, and relevant contextual factors.

Attention and orientation in the mental state examination. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Catatonic signs

Catatonia is a psychomotor syndrome across psychiatric and medical conditions. A still image cannot establish the diagnosis.

Three key posture signs. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision uses these distinctions.
The Bush-Francis Catatonia Rating Scale groups posturing/catalepsy and describes waxy flexibility as initial resistance followed by yielding.

Posturing

Spontaneously adopts and maintains a posture against gravity.



"Holds arm raised for a prolonged period without assistance."

Catalepsy

Examiner places a limb in a posture, then it remains held against gravity after release.



"Arm remains in the imposed posture after the examiner lets go."

Waxy flexibility

Slight, even resistance felt while the examiner repositions the limb.



Holding the position alone is catalepsy, not proof of waxy flexibility.

Exam / clinical limits

The twelve core signs in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision are: stupor, catalepsy, waxy flexibility, **mutism, negativism, posturing, mannerism, stereotypy, agitation, grimacing, echolalia and echopraxia.**

Three or more support the syndrome in the proper clinical assessment; rating-scale thresholds differ.

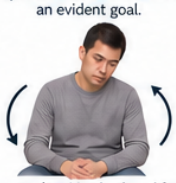
- Observe, interview, examine and obtain collateral history.
- Exclude mimics and investigate causes.
- Fever, autonomic instability, poor intake or immobility complications require urgent clinical assessment.

Historical anchor
Karl Ludwig Kahlbaum described catatonia as a syndrome.

Repetitive / odd movements

Stereotypy

Repeated movement without an evident goal.



"Repeated rocking back and forth."

Mannerism

Odd exaggeration of a purposeful action. Purposeful action, unusual execution.



"Theatrical, elaborate greeting."

Grimacing

Sustained or repeated abnormal facial expression.



"Repeated facial grimacing."

Echopraxia

Imitates another person's movement.



"Repeats the examiner's movement."

Echolalia

Repeats another person's words.



"Automatic verbal repetition."

Reduced or excessive activity

Stupor

Profound reduction in motor activity and engagement. Do not assume unconsciousness.



"Minimal movement and engagement."

Mutism

Absent or very little speech; assess language/hearing and other causes.



"No or minimal verbal response."

Negativism

Opposition or no response to instructions or external stimuli, unexplained by the situation; not simply informed refusal.



"No response or active opposition to a request."

Agitation/excitement

Excessive apparently purposeless activity not driven by external stimuli.



"Pacing and restless, purposeless movements."

Additional bedside signs

Automatic obedience

Exaggerated, automatic compliance. Assess only with safe requests.



"Immediately complies with a simple request."

Ambitendency

Hesitates between opposing movements, e.g. repeatedly advances and withdraws a hand.



"Repeatedly advances and withdraws a hand."

Gegenhalten

Resistance rises with the examiner's applied force.



"More resistance as force is increased."

Mitgehen

Limb follows very light pressure despite instruction to resist.



"Limb moves with minimal pressure."

Verbigeration

Stereotyped repetition of words or phrases.



"Repeated, stereotyped phrases."

Catatonic signs. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

Insight and judgement

INSIGHT

awareness and interpretation of one's condition

Assess separately:

Awareness of change — "Have you noticed any change in sleep, thinking or behaviour?"

Understanding symptoms — "How do you explain these experiences? Could there be another explanation?"

Consequences — "How has this affected work or relationships?"

Need for help — "What help might be useful? What concerns you about treatment?"



Insight may vary by symptom, change over time and differ from retrospective insight.

Anthony David's three overlapping dimensions: recognition of illness; relabelling unusual experiences as pathological; treatment compliance in the original model.

Modern assessment must explore barriers and preferences: adherence is not a pure measure of insight.

CLASSICAL SIX-GRADE TEACHING SCHEME (descriptive, not a capacity test)

1. Denies illness.
2. Partial awareness, with simultaneous denial.
3. Recognises difficulty but attributes it to external or physical causes.
4. Recognises something is wrong within oneself, explanation unclear.
5. Intellectual insight: recognises illness but does not translate understanding into change.
6. Emotional insight: understanding becomes integrated into behaviour.

Do not equate disagreement with the clinician, a cultural explanation, or medication refusal with absent insight.

JUDGEMENT

evaluating situations and acting on consequences

Assess personal, social and practical decisions; distinguish reasoning from behaviour.

Ask about a real recent decision:

"What options did you consider?" "What could happen? Why did you choose that?"



Explore within context:

- safety
- money
- relationships
- self-care

Hypothetical test:

"What would you do if you smelled smoke?"

A sensible answer does not establish safe real-world judgement.



Example:

"Understands overspending risks but repeatedly spends rent money impulsively."

Describe the affected domain rather than "judgement poor".

DECISION-MAKING CAPACITY

ability for THIS decision at THIS time

A **separate** functional assessment, with communication support and the applicable legal framework.

Common clinical abilities:

1. **Understand** relevant information.
2. **Appreciate** how it applies to one's own situation.
3. **Reason** about options and consequences.
4. **Communicate** a choice.

Some legal frameworks separately specify retention and using/weighing information. Do not substitute this clinical summary for local law.



Key points:

- Poor insight may affect capacity but does not automatically remove it.
- A psychiatric diagnosis, unusual choice or treatment refusal alone does not establish incapacity.

WORKED ASSESSMENT EXAMPLE



"Recognises reduced sleep and its impact, but attributes unusual experiences solely to others. Can discuss treatment benefits and adverse effects; declines one option because of prior sedation. Capacity for that specific decision requires separate assessment."

EXAMINATION FOCUS AND COMMON TRAPS



- Record the person's explanation and supporting examples.
- Assess dimensions, context, fluctuation and supports.
- Insight ≠ judgement ≠ capacity.
- Be careful not to equate disagreement, a cultural explanation or medication refusal with absent insight or incapacity.
- Avoid over-relying on hypothetical answers.
- Consider the specific decision and time frame.

Insight and judgement. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nosology, classification and descriptive psychopathology.

Credits and references: retained within the illustration.

AREA 12 OF 12 · P1-A03

Neuroendocrinology and basic psychopharmacology

4	2.8%	I-97	I-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 3

REVISION ORDER

The two stress slots together, they are one answer split at the immune limb. The pineal gland next, and it is short. The evidence-based medicine slot last: it is three terms in one question and it rewards precision rather than length.

WHAT IS IN THIS AREA

Stress in two slots, one asking for the neurobiology with a critical evaluation of coping, and one for the types with their effect on the immune system. The pineal gland. And the slot that runs evidence-based medicine into personalised medicine and pharmacogenetics. The registered name of this area carries basic psychopharmacology, and its one pharmacology slot is a pharmacogenetics question rather than a drug-class question. Four slots, and the area is described here by what they hold.

I - 97

Pineal gland. [10]

10 NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Position, and the two things that make it unusual
3 Light to melatonin	The pathway, then the synthetic chain
3 Receptors and actions	Two receptors, and what each one does
2 Clinical relevance	The drugs, the disorders, the tumour

THE DEFINITION

A small unpaired midline endocrine gland of the epithalamus, sitting behind the third ventricle. Two features carry the answer: it has no direct nerve supply from the brain, and it lies outside the blood brain barrier. It converts a light signal into a hormonal one.

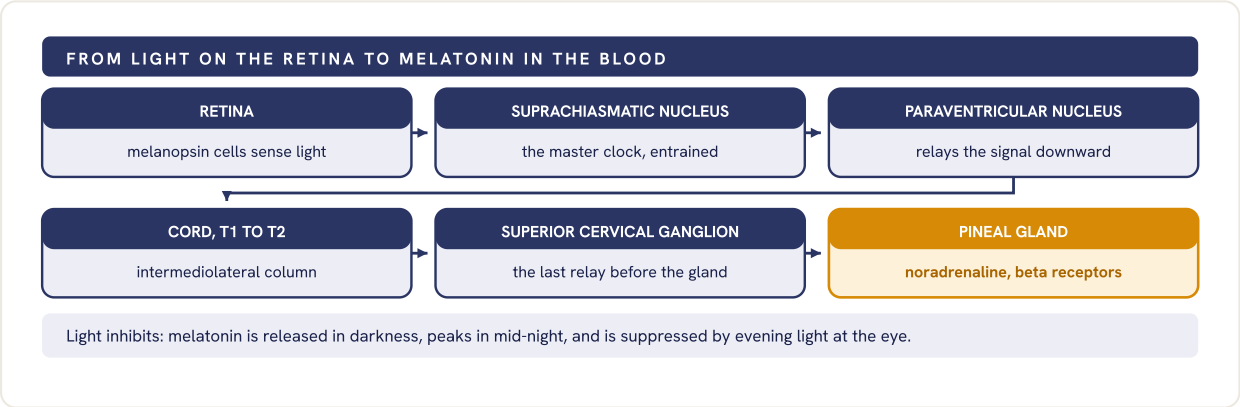


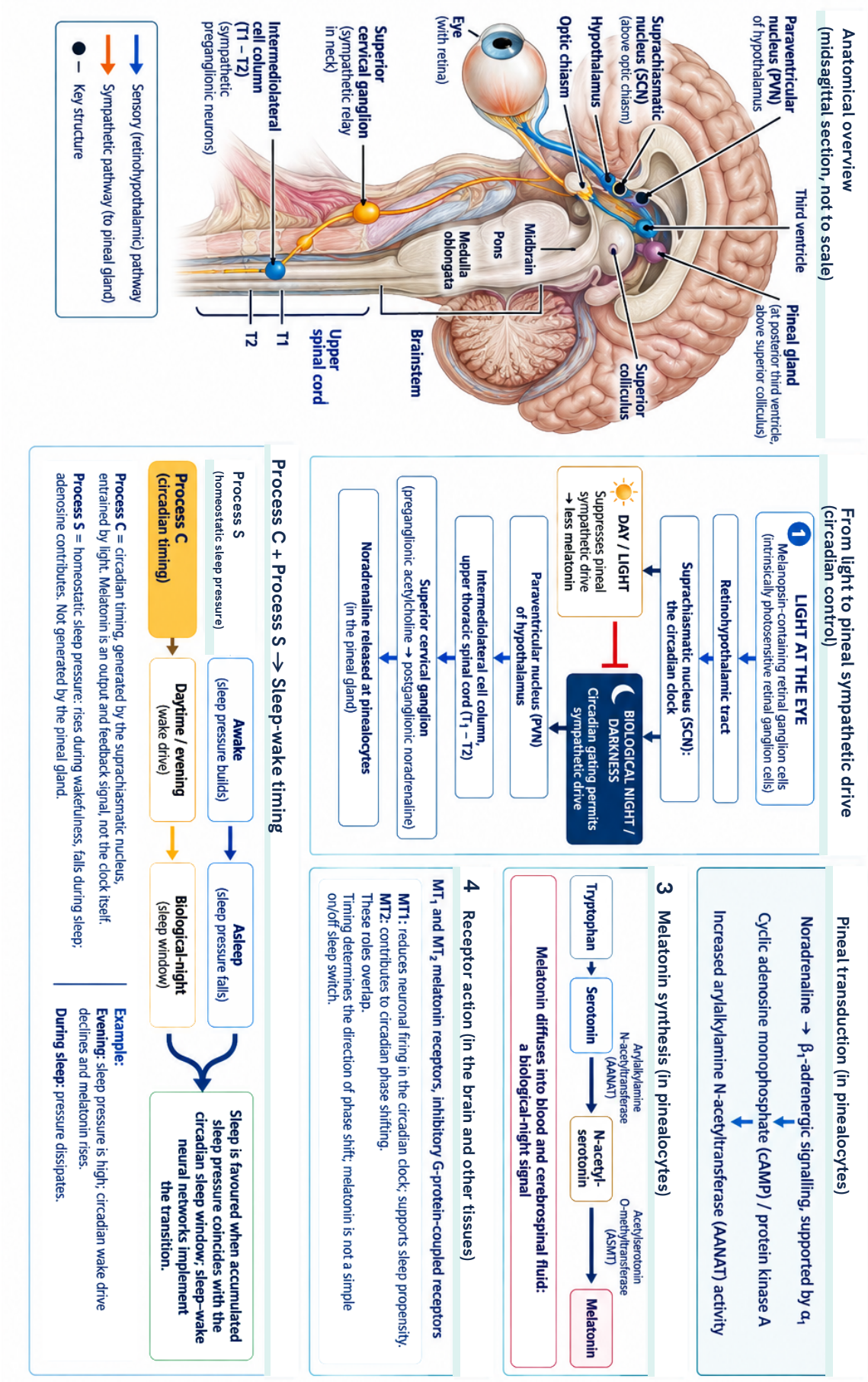
Fig 097.1 A sympathetic pathway with a hypothalamic beginning: the only route from the eye to the gland runs out of the brain, down the cord and back up the neck.

- **Synthesis.** Tryptophan to serotonin, then serotonin to N-acetylserotonin by arylalkylamine N-acetyltransferase, the rate limiting and dark-driven step, then to melatonin. Melatonin is not stored: it diffuses out as it is made.
- **Receptors and actions.** MT1 and MT2, both inhibitory and G protein coupled. MT1 damps the wake signal; MT2 shifts the phase. Melatonin re-times sleep more than it sedates.
- **Where it appears in exams.** Melatonin and ramelteon at MT1 and MT2; agomelatine, an agonist there and a 5-HT2C antagonist. Delayed sleep-wake phase disorder, free-running rhythm in blindness, jet lag. Calcification as a midline marker, and pineal tumours causing Parinaud syndrome.

WHAT EARNS THE MARKS

- **The two unusual features stated early.** No direct central innervation, and outside the blood brain barrier.
- **The whole pathway, eye to gland,** including the trip through the cord and the superior cervical ganglion.
- **The rate limiting enzyme named,** and the point that darkness drives it.
- **Phase shifting separated from sedation.** It is why melatonin is dosed by clock time, not by severity.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 09 sleep stages and polysomnography **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology



Pineal Gland Mechanism. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with I-97.

Credits and references: retained within the illustration.

I - 98

Define stress. Discuss neurobiology of stress. Critically evaluate the efficacy of different coping mechanisms in stress management. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	One physiological, one transactional
3 Neurobiology	A fast axis, a slow axis, and the brake on both
5 Coping, evaluated	Match to controllability, then judge the evidence

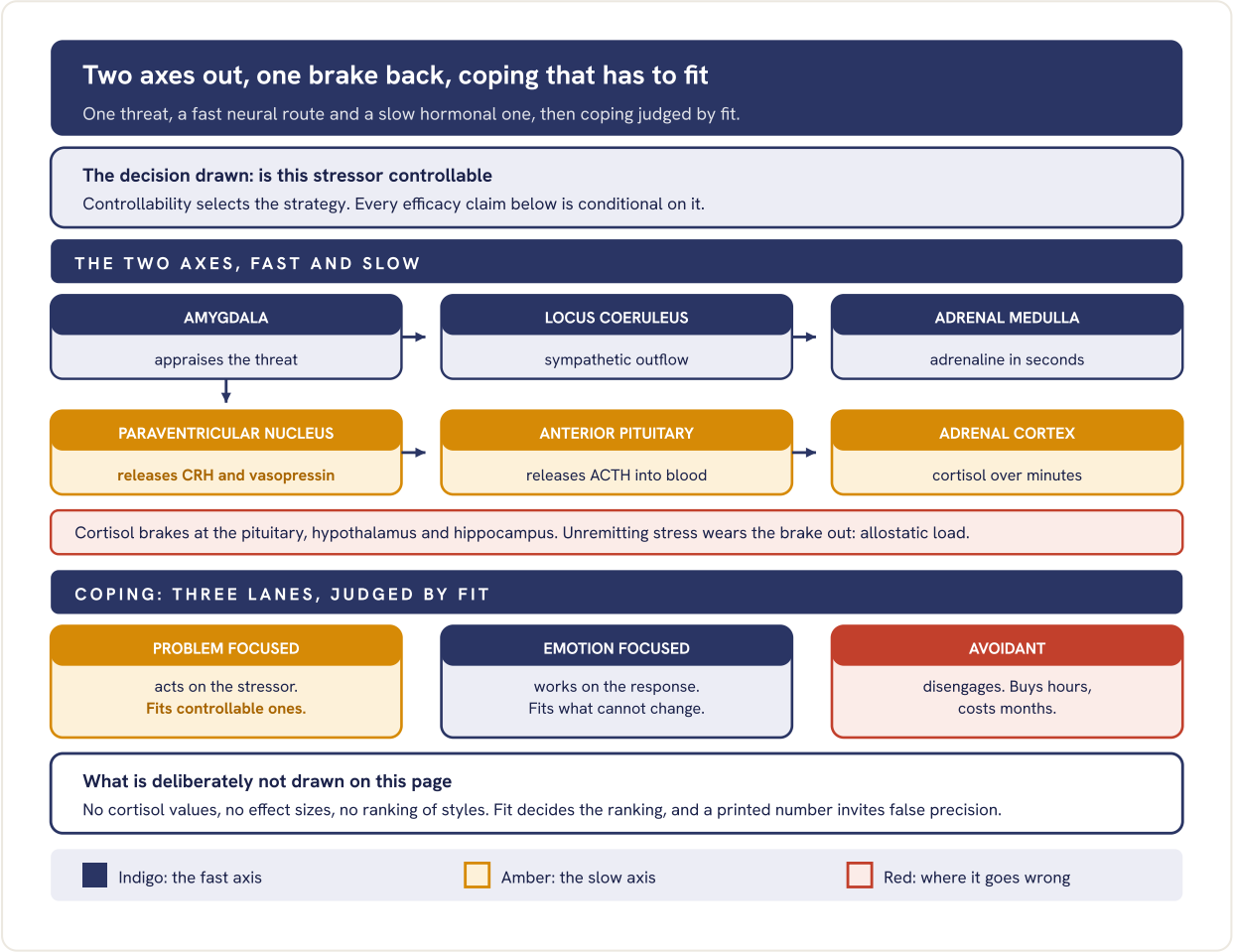


Fig 098.1 The two efferent axes drawn to one width so their timescales compare, with the brake and the coping lanes below.

COPING STYLE	WHAT THE PERSON DOES	WHERE IT WORKS	WHERE IT FAILS
Problem focused	Plans, seeks information, acts	**Controllable stressors: workload, illness self-management**	Effort on what cannot change raises distress
Emotion focused	Reappraises, accepts, relaxes, seeks support	**Uncontrollable ones: bereavement, terminal illness**	When it replaces action that was possible
Meaning focused	Reframes within values and goals	Chronic adversity, caregiving	When it minimises a real threat
Avoidant	Denies, distracts, uses substances	Briefly, in the first hours	**Over time: the most consistent predictor of poor outcome**

The amber cells are the goodness of fit rule. Avoidance reverses: what helps at hour one harms by month three.

- **The two definitions to give.** Physiological: the non-specific response of the body to any demand, through alarm, resistance and exhaustion. Transactional: a relationship appraised as taxing or exceeding the person's resources.
- **What critically evaluating means here.** Most coping research is cross-sectional and self-reported, the categories overlap, and stress management trials report modest, short-lived effects. Then say what stands: exercise, relaxation, mindfulness based stress reduction, stress inoculation.

WHAT EARNS THE MARKS

- **Both definitions.** The response without the appraisal is a half answer.
- **Both axes with their timescales,** and the brake named at all three levels.
- **The goodness of fit rule,** not styles ranked best to worst.
- **One honest limit on the evidence.** The word critically is asking for it.

SEE ALSO Slot I - 100 stress, immunity and psychiatric disorder Slot I - 29 classification of defence mechanisms

SPRINT Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

I - 99

What is evidence based medicine? What is personalized medicine in psychiatry? Discuss pharmacogenetics. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Evidence based medicine**	The definition, its three components, the five steps
3	**Personalised medicine**	What it means, and what it is in psychiatry today
3	**Pharmacogenetics that works**	Metabolism genes, and the one safety gene
2	**Where the evidence stops**	Response prediction, and why it is not in clinic

THE DEFINITION TO WRITE FIRST

Evidence based medicine is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. Three components, all required: research evidence, clinical expertise, and the patient's values.

GENE	WHAT IT CHANGES	WHAT FOLLOWS CLINICALLY	HOW STRONG THE EVIDENCE IS
CYP2D6	Clearance of risperidone, aripiprazole, venlafaxine, tricyclics	Poor metabolisers accumulate drug; ultrarapid metabolisers are underexposed	**Established, with published dosing guidance**
CYP2C19	Clearance of escitalopram, citalopram, sertraline, some tricyclics	Same direction, so dose is adjusted at the two extremes	**Established, with published dosing guidance**
HLA-B*15:02	Risk of carbamazepine Stevens-Johnson syndrome	Test before starting carbamazepine in people of South and East Asian ancestry	**Established, testing recommended**
SLC6A4, HTR2A	Proposed prediction of antidepressant response	Not usable to choose a drug	Inconsistent across studies

The amber column is the whole answer: pharmacokinetic genes and one safety gene have earned their place, response prediction has not.

- **Evidence based medicine in five steps.** Ask an answerable question, acquire the best evidence, appraise it for validity and effect size, apply it alongside expertise and patient values, and assess the outcome.
- **What personalised medicine is not.** It is not a genetic test that picks the drug. In psychiatry it is mostly clinical: staging, phenotype, previous response, family history, comorbidity and measurement based care, with pharmacogenetics contributing at the metabolic extremes and one pre-treatment safety test.

WHAT EARNS THE MARKS

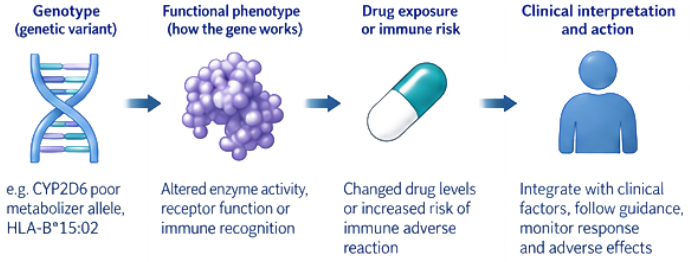
- **All three components of the definition.** Evidence alone is the commonest half answer.
- **Pharmacokinetic genes separated from pharmacodynamic ones,** and response prediction called not yet usable rather than promising.
- **HLA-B*15:02 before carbamazepine,** named as the one test with a clear ancestry-based indication.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 84 clinical trial registry of India **SPRINT** Day 3, Psychiatric Genetics

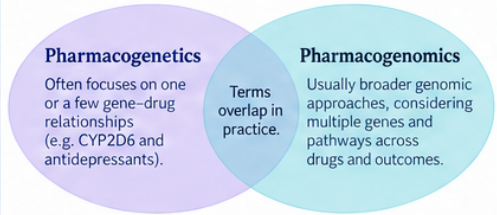
Pharmacogenomics and Pharmacogenetics

1. From genotype to clinical effect

Genetic variation can influence drug response through effects on pharmacokinetics, pharmacodynamics or immune risk.



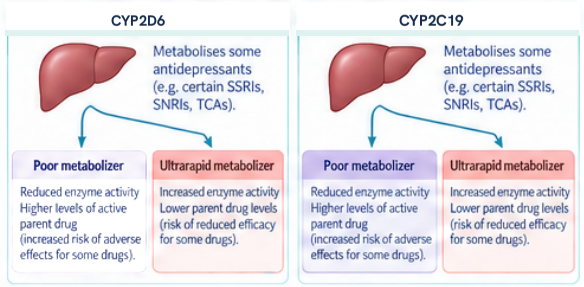
2. Pharmacogenetics, pharmacogenomics and related concepts



A commercial genomic panel cannot reliably identify a universally best antidepressant or guarantee response. A genetic result does not replace a careful diagnostic assessment, ongoing monitoring or shared decision-making.

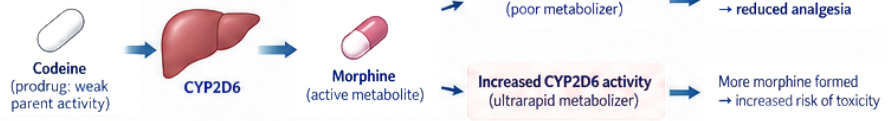
3. Key pharmacokinetic examples

CYP2D6 and CYP2C19 variants affect the metabolism of selected antidepressants. The effect depends on the specific drug and whether active metabolites are formed.



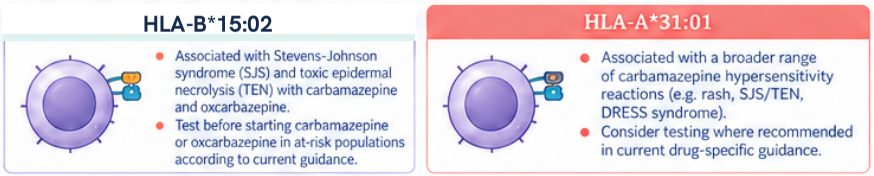
4. Special case: prodrugs (codeine example)

Codeine is a prodrug that requires CYP2D6 metabolism to form the active metabolite morphine. The clinical effect is the opposite to many other drugs.



5. Immune-mediated adverse reactions (pharmacogenetics)

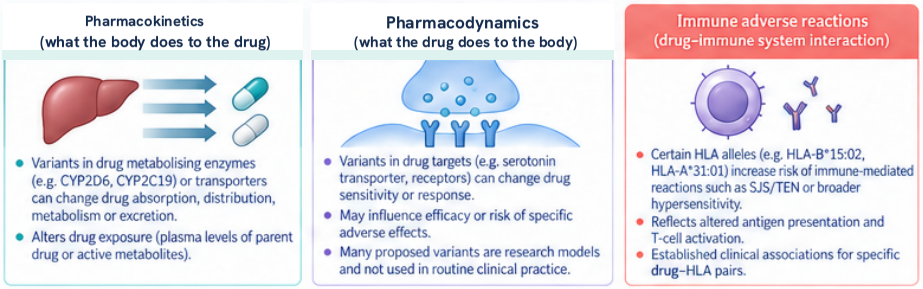
Certain HLA (human leucocyte antigen) alleles are associated with increased risk of serious cutaneous adverse reactions to antiepileptic drugs.



Allele distribution varies between populations. Ancestry is not a substitute for genotype testing.

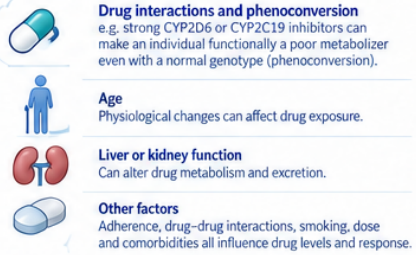
6. Three mechanistic tracks: how genes influence drug response

Genetic variation can affect pharmacokinetics, pharmacodynamics or immune risk, leading to different clinical outcomes.



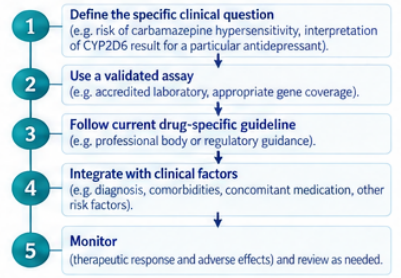
7. Other factors can alter the observed phenotype

The metabolizer phenotype observed in an individual may differ from the genotype prediction (phenoconversion) and is influenced by multiple non-genetic factors.



8. A practical decision sequence

Distinguish testing guidance from interpretation of an available result.



9. Ethical considerations

Genomic privacy
Protect genetic information and ensure secure data handling.

Informed consent
Discuss purpose of testing, potential results, limitations and implications.

Appropriate use
Use testing to support, not replace, clinical judgement and shared decision-making.

Pharmacogenomics and Pharmacogenetics. Use the visual organisation to recall the key distinctions and structure your answer. Read with I-99.

Credits and references: retained within the illustration.

I - 100

Enumerate the types of stress and its effect on the immune system and psychiatric disorders. [10]

10 | ASKED AT 15 MARKS | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The types	Name the axis first: duration, and whether there is an endpoint
3	How stress reaches immunity	Three routes, hormonal, neural and behavioural
3	What changes	Direction depends on the type, and that is the mark
2	Psychiatric consequence	Association, then the one clean causal example

TYPE OF STRESS	WHAT IT LOOKS LIKE	IMMUNE DIRECTION	EXAMPLE
Acute time limited	Minutes, with a clear end	Innate immunity mobilised, natural killer cells rise	A viva, a public talk
Brief naturalistic	Days, a real short stressor	Shift away from cellular toward humoral immunity	Examination week
Event sequences	One event with a train of consequences	Mixed, and it changes over the course	Bereavement, job loss
Chronic	No endpoint in sight, identity altered	**Broad suppression: killer cell cytotoxicity and lymphocyte proliferation fall, wounds heal slowly**	Caregiving, poverty
Distant	Long past, effects persist	**Low grade inflammation with glucocorticoid resistance**	Childhood adversity

The axis is duration and whether the stressor has an endpoint. The amber rows are where the direction reverses, and stating that reversal is what separates this answer from a list. Never write that stress suppresses immunity: it depends on the row.

- **How stress reaches the immune system.** Three routes. Hormonal, through cortisol on lymphocytes. Neural, through sympathetic fibres that directly innervate the spleen, thymus and lymph nodes. Behavioural, because stressed people sleep less, drink more, eat worse and attend less.
- **The paradox worth stating.** Cortisol is anti-inflammatory, so chronic stress ought to damp inflammation. It does the opposite, because immune cells become glucocorticoid resistant and inflammatory signalling comes off the brake.
- **Psychiatric consequence.** Raised C reactive protein and interleukin 6 are associated with depression and with poorer antidepressant response. The cleanest causal evidence is different: interferon alfa given for hepatitis produces depressive illness in previously well people.

WHAT EARNS THE MARKS

- **The types enumerated with a stated axis,** duration and whether the stressor has an end.
- **Both directions given.** Up in acute, down in chronic, is the discriminating sentence.
- **The three routes named,** hormonal, neural and behavioural, not cortisol alone.
- **Association separated from causation,** with interferon alfa offered as the causal case.

SEE ALSO Slot I - 98 neurobiology of stress and coping Slot I - 08 biological markers in psychiatric illness **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

Cytokines

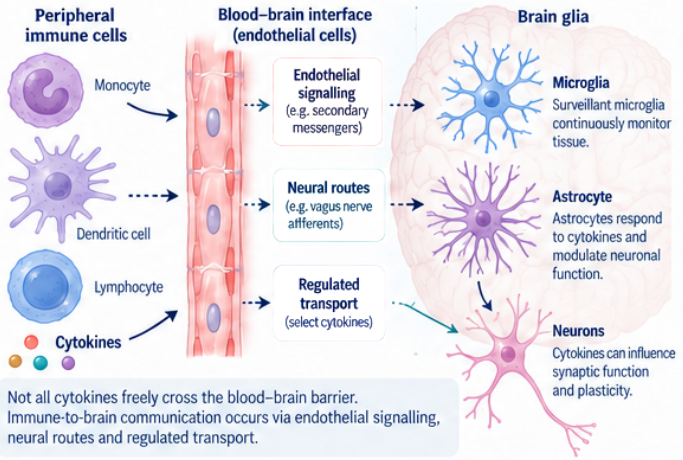
1. What are cytokines?

- Cytokines are small signalling proteins that coordinate immune responses.
- They can be pro-inflammatory, anti-inflammatory or regulatory.
- They act locally and systemically, affecting immune cells, the brain and other organs.
- Not all cytokines are inflammatory.
- C-reactive protein (CRP) is an acute-phase protein, not a cytokine.
- Cytokines have important roles in normal defence, repair and developmental processes.
- Excessive or dysregulated cytokine signalling may contribute to disease.

2. Representative cytokines and functions

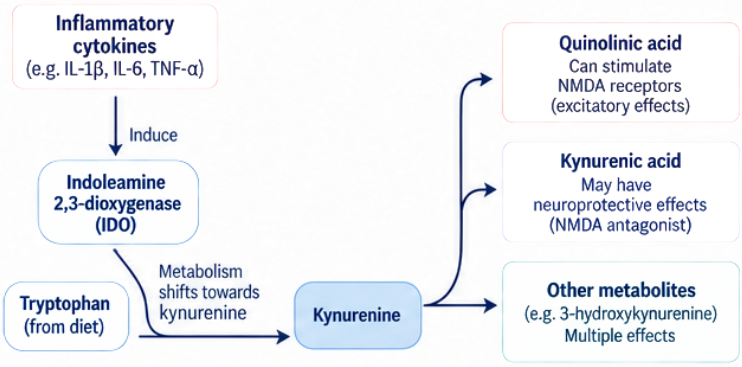
Cytokine	Full name	Typical function (context dependent)
IL-1β	Interleukin-1 beta	Pro-inflammatory signal (e.g. fever, leukocyte activation)
IL-6	Interleukin-6	Pro-inflammatory signal (e.g. acute-phase response, B cell activation)
TNF-α	Tumour necrosis factor alpha	Pro-inflammatory signal (e.g. leukocyte activation, endothelial activation)
IFN-α	Interferon alpha	Antiviral signal (enhances innate and adaptive immunity)
IL-10	Interleukin-10	Regulatory / anti-inflammatory function (limits immune responses)
TGF-β	Transforming growth factor beta	Regulatory function (promotes resolution, tissue repair)

3. From immune system to brain



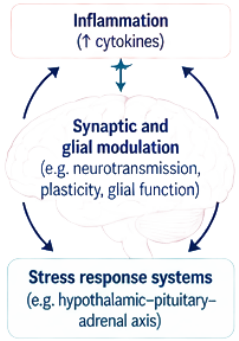
Not all cytokines freely cross the blood–brain barrier. Immune-to-brain communication occurs via endothelial signalling, neural routes and regulated transport.

4. Inflammation, tryptophan metabolism and other mechanisms



Inflammation can shift tryptophan metabolism towards the kynurenine pathway. Metabolites have different effects. This is not an inevitable serotonin depletion story.

Interacting mechanisms



Cytokines interact with stress axis activity and can modulate synaptic and glial function.

5. Clinical relevance

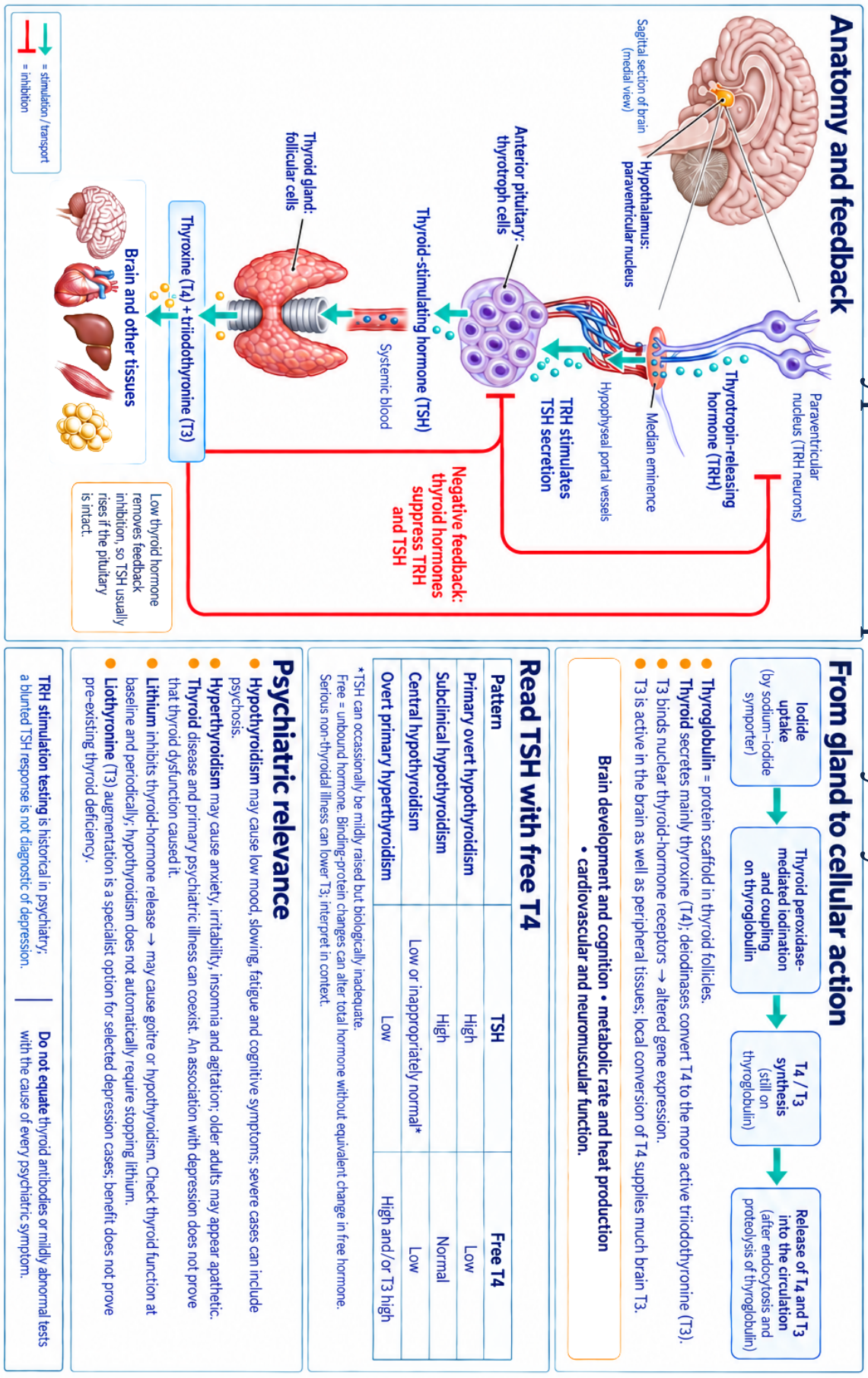
Inflammatory markers in some patient groups	• Inflammatory markers (e.g. cytokines, CRP) are elevated on average in some psychiatric patient groups. • There is substantial heterogeneity and potential confounding (e.g. physical illness, obesity, smoking, medication, stress).
Interferon treatment	• Interferon alpha (IFN-α) treatment can induce depressive symptoms in some individuals. • Supports a causal role for cytokine signalling in mood regulation.
Sickness behaviour and depression	• Sickness behaviour (e.g. low mood, anhedonia, fatigue, social withdrawal) overlaps with features of major depression but the two are not identical. • Sickness behaviour is an adaptive, time-limited response to illness, whereas major depression is a heterogeneous psychiatric disorder.
What cytokines cannot tell us	• No cytokine test diagnoses depression. • No universal CRP cutoff defines a psychiatric subtype. • Anti-cytokine medicines are not routine antidepressant treatment.
A balanced perspective	• Cytokines are essential for normal defence, repair and developmental processes. • Excessive or dysregulated signalling may be harmful.

Cytokines are one piece of a complex biological and psychosocial picture in psychiatric disorders.

Cytokines. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

Hypothalamic-pituitary-thyroid axis

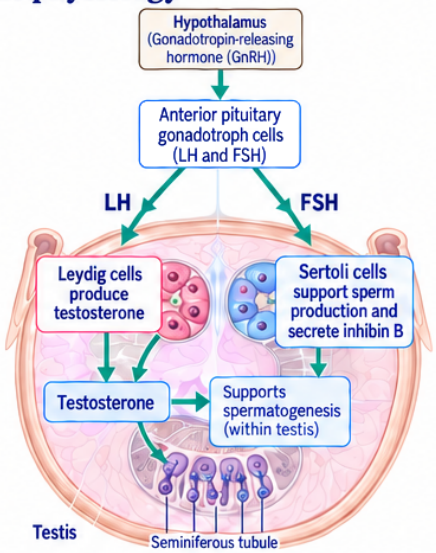


Hypothalamic-pituitary-thyroid axis. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

Hypothalamic-pituitary-gonadal axis

Male physiology



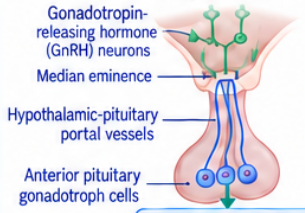
NEGATIVE FEEDBACK: Testosterone and estradiol inhibit hypothalamic GnRH and pituitary LH/FSH. Inhibin B selectively inhibits pituitary FSH.

Functions: sexual development, libido, muscle and bone. Fertility depends on Sertoli support AND high intratesticular testosterone.

External testosterone can suppress LH/FSH and sperm production.

In adult males, regulation is predominantly negative feedback. There is no monthly ovulatory surge.

Shared hypothalamic-pituitary-gonadal pathway



Pulsatile GnRH maintains LH/FSH secretion

Time →

Continuous GnRH agonist: initial stimulation, then receptor desensitisation and suppression.

Luteinising hormone (LH) + Follicle-stimulating hormone (FSH)

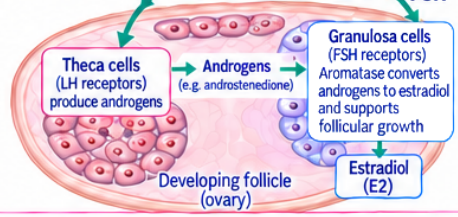
→ Gonads (testis or ovary)

Female physiology

Hypothalamus (Gonadotropin-releasing hormone (GnRH))

→ Anterior pituitary gonadotroph cells (LH and FSH)

NEGATIVE FEEDBACK: Usual estradiol levels inhibit GnRH and LH/FSH. Inhibin B (follicle) and inhibin A (corpus luteum) selectively inhibit pituitary FSH.



Sustained high estradiol → POSITIVE feedback → LH surge → Ovulation → Corpus luteum → Progesterone + estradiol + inhibin A

Functions: follicular development, ovulation, endometrial proliferation (estradiol) and secretory preparation (progesterone). Negative feedback is usual; sustained high late-follicular estradiol switches to positive feedback.

Psychiatric relevance

Dopamine and prolactin



Dopamine (from hypothalamus) ⊣ Anterior pituitary lactotrophs → Prolactin (PRL)

- Dopamine normally inhibits anterior pituitary lactotrophs.
- Dopamine D2 receptor blockade → prolactin rise → suppression of GnRH pulsatility → lower sex steroids: menstrual disturbance, sexual dysfunction, infertility and bone loss.

Neurosteroids and GABA

- Progesterone → allopregnanolone, a neurosteroid that positively modulates gamma-aminobutyric acid type A ($GABA_A$) receptors.

Premenstrual dysphoric disorder

- Abnormal sensitivity to normal ovarian-hormone fluctuations is a leading mechanism; not simply hormone deficiency.

Peripartum and perimenopausal transitions

- May increase vulnerability in susceptible people.
- Hormonal association does not establish the cause of an individual's illness.

Male hypogonadism and mental health

- Male hypogonadism may cause low libido, fatigue and mood symptoms; depression alone does not diagnose testosterone deficiency.

Menstrual cycle: hormonal changes and key events

1. Early follicular / menstruation

Corpus luteum regresses → progesterone, estradiol and inhibin fall → FSH rises and recruits follicles.



Menstruation (endometrial shedding)

2. Follicular growth

Estradiol rises; mainly **NEGATIVE** feedback. Inhibin B helps reduce FSH; dominant follicle develops.



Growing follicles (developing dominant follicle)

3. Late follicular

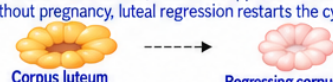
Sustained high estradiol → POSITIVE feedback → LH surge → ovulation.



Ovulation (release of oocyte)

4. Luteal

Corpus luteum progesterone + estradiol restore NEGATIVE feedback; inhibin A suppresses FSH. Without pregnancy, luteal regression restarts the cycle.



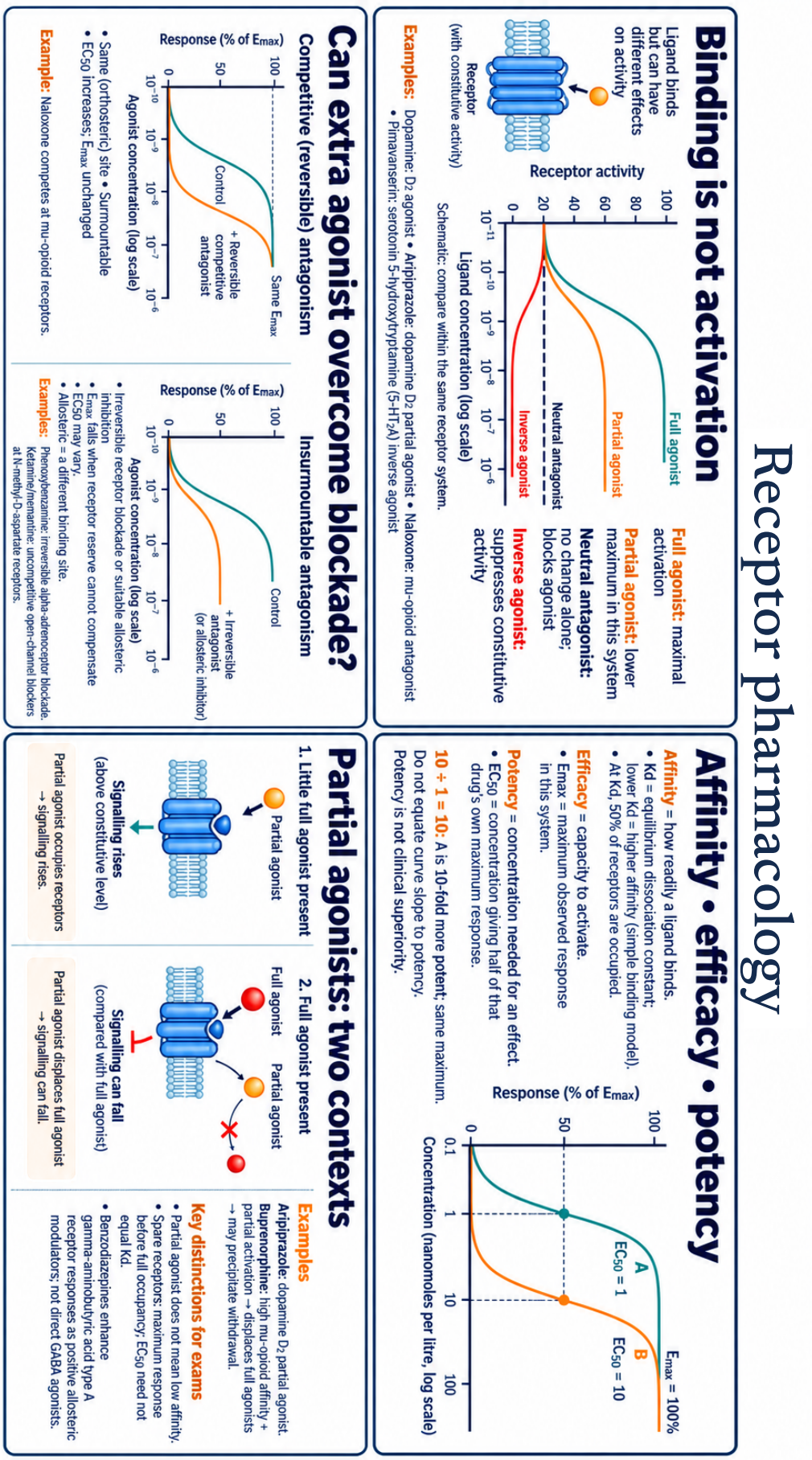
Corpus luteum (secretes progesterone, estradiol and inhibin A) ⇢ Regressing corpus luteum (leads to next cycle)

Key → = Stimulation / transport; ⊣ = Inhibition (blunt terminal); → = Positive feedback

Timing varies; ovulation is not always on day 14.
Primary gonadal failure: sex steroids low, LH/FSH high. **Central suppression:** sex steroids low, LH/FSH low or inappropriately normal.

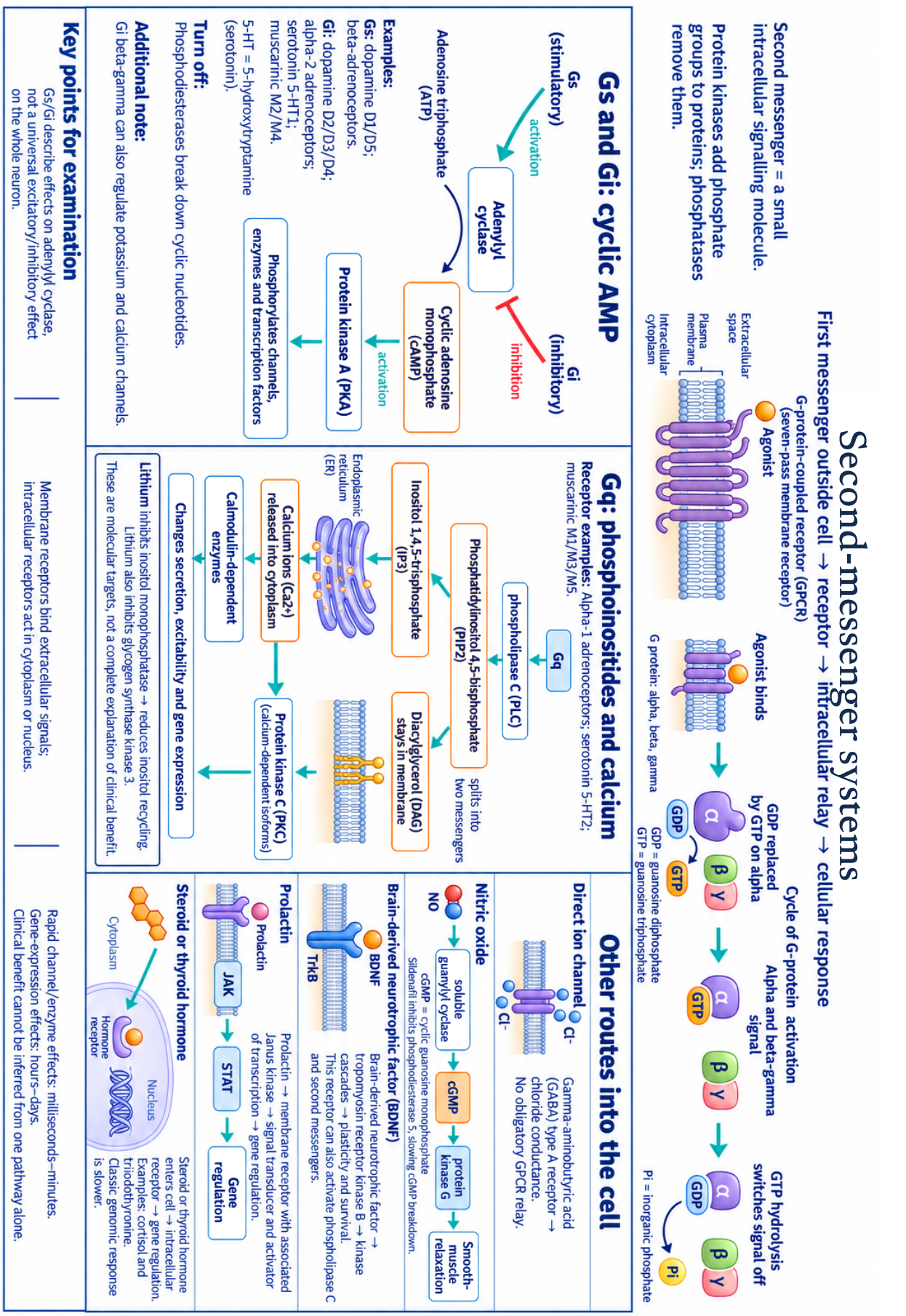
Hypothalamic-pituitary-gonadal axis. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.



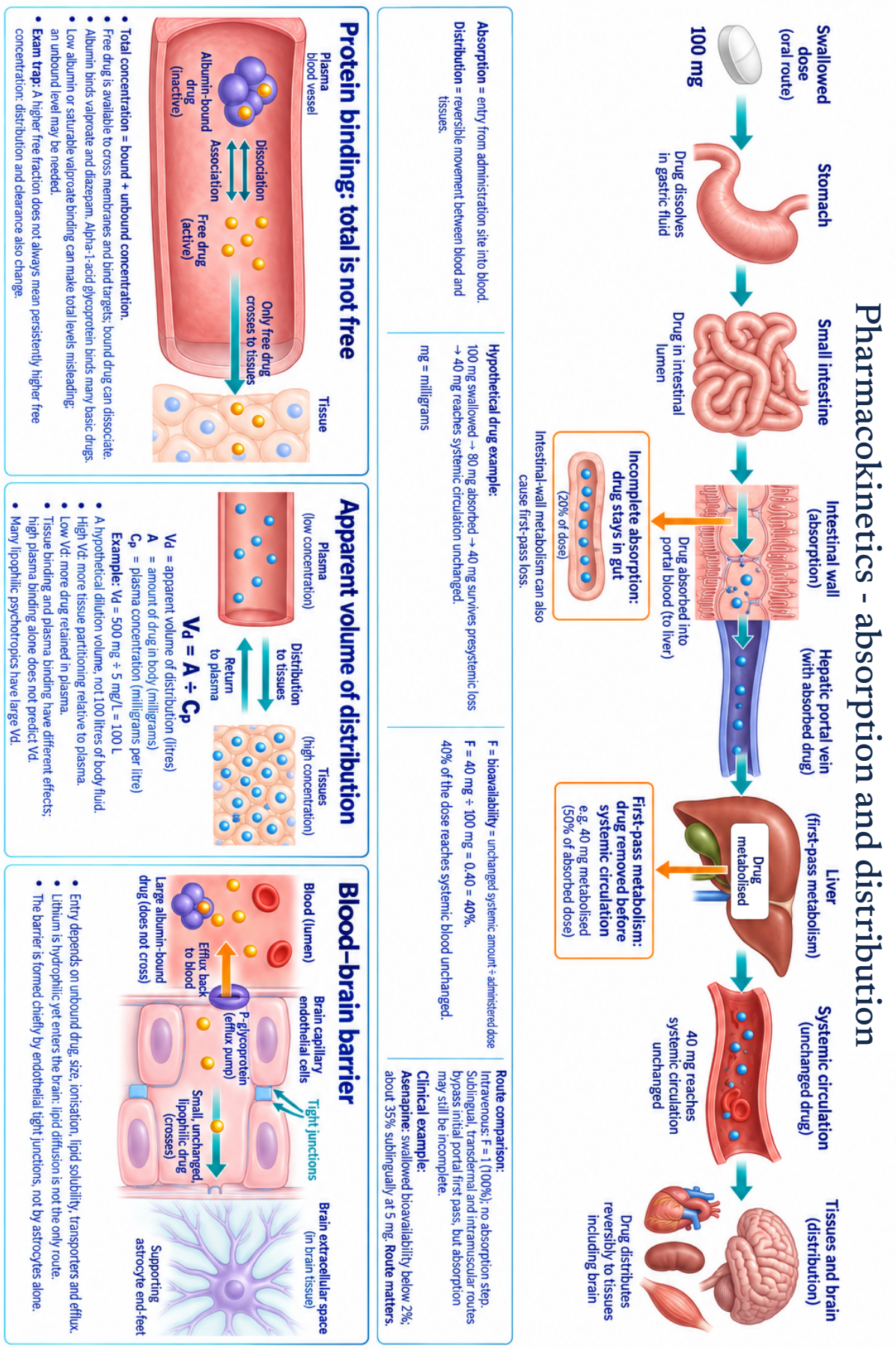
Receptor pharmacology. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.



Second-messenger systems. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

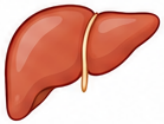


Pharmacokinetics - absorption and distribution. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

Pharmacokinetics - metabolism and elimination

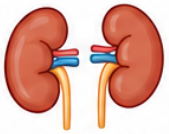
Metabolism and excretion



Phase I: oxidation, **reduction**, hydrolysis; often cytochrome P450 enzymes.

Drug → inactive, active or toxic metabolites

Phase II: conjugation, for example glucuronidation; can occur without Phase I.
Lorazepam undergoes direct glucuronidation.



Renal excretion: filtration, secretion and reabsorption.
Lithium: no hepatic metabolism; excreted unchanged by kidneys.

Elimination includes metabolism AND excretion.

Clearance (CL) = elimination rate ÷ plasma concentration
20 mg/hour ÷ 5 mg/L = 4 L/hour

Equivalent to clearing drug from 4 litres of plasma each hour; not 4 litres of urine.

Half-life and the bridge equation

$$t_{½} = 0.693 \times V_d \div CL$$

$t_{½}$ = elimination half-life (hours)
V_d = apparent volume of distribution (litres)
CL = clearance (litres/hour)
0.693 = natural logarithm of 2

Substitution:
$t_{½}$ = 0.693 × 40 L ÷ 4 L/hour = **6.93 hours**

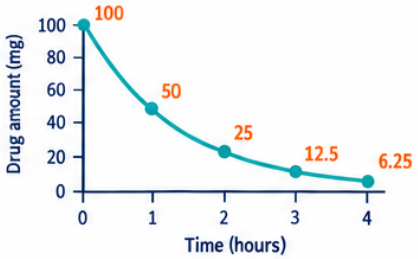
Plasma concentration halves about every 7 hours.

Assumptions: linear first-order elimination; constant clearance and volume; simple one-compartment model.

Higher V_d or lower CL → longer half-life.

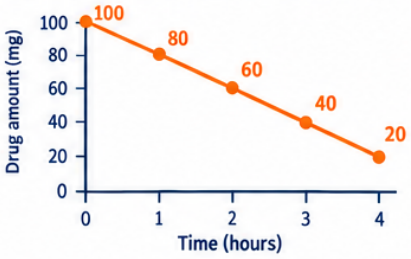
First-order versus zero-order

First-order: constant FRACTION removed



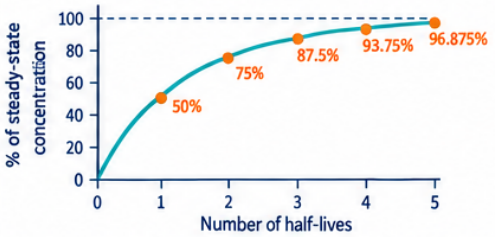
100 → 50 → 25 → 12.5 mg
at one-hour steps.
Here half-life = 1 hour; half removed each hour.
Most drugs in their linear range.

Zero-order: constant AMOUNT removed



100 → 80 → 60 → 40 mg
at one-hour steps.
Here 20 mg removed per hour; no fixed half-life.
Near saturation: ethanol; phenytoin can show capacity-limited kinetics.
Saturation is concentration-dependent, not a permanent label for a drug.

Steady state and dosing



Constant infusion from zero, linear kinetics.
At steady state: input rate = elimination rate.

About 4–5 half-lives gives 94–97% of the final concentration.

Repeated doses reach a repeating peak–trough pattern, not a flat line.

Maintenance dose rate = CL × target concentration ÷ F
4 L/hour × 5 mg/L ÷ 0.5 = 40 mg/hour

Loading dose = V_d × target concentration ÷ F
40 L × 5 mg/L ÷ 0.5 = 400 mg

F = bioavailability (fraction); 0.5 = 50%
Loading fills the distribution volume; maintenance replaces elimination.

Steady drug levels do not mean full clinical response. After 5 half-lives, 3.125% remains; washout also depends on active metabolites and pharmacodynamic effects.

mg = milligrams; L = litres; × = multiply; ÷ = divide

Pharmacokinetics - metabolism and elimination. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

Enzyme induction and inhibition

INHIBITION: reduced enzyme activity → slower clearance of an active substrate → concentration usually rises



INDUCTION: increased enzyme expression/activity → faster clearance of an active substrate → concentration usually falls



Time course

Inhibition may begin rapidly; onset and recovery depend on inhibitor half-life and mechanism. Irreversible inhibition needs enzyme recovery. Fluoxetine-related inhibition can persist for weeks after stopping.

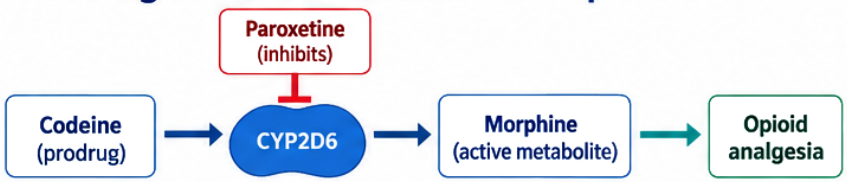
Induction builds and wanes over days to weeks; new enzyme synthesis and turnover take time.

Follow the direction: exposure → enzyme → affected drug

Perpetrator	Enzyme effect	Affected drug	Concentration and consequence
Fluvoxamine	Inhibits CYP1A2	Clozapine	Clozapine rises → toxicity risk
Tobacco smoke	Induces CYP1A2	Clozapine	Clozapine falls; STOPPING smoke reverses induction → clozapine rises
Fluoxetine	Inhibits CYP2D6	Desipramine	Desipramine rises → adverse-effect risk
Carbamazepine	Induces CYP3A4	Quetiapine	Quetiapine falls → loss of effect
Ketoconazole	Inhibits CYP3A4	Quetiapine	Quetiapine rises → adverse-effect risk
Valproate	Inhibits glucuronidation	Lamotrigine	Lamotrigine rises → greater rash risk; use its specific slow titration

CYP = cytochrome P450; 1A2, 2D6 and 3A4 identify different enzymes. Glucuronidation is a Phase II conjugation pathway mediated by uridine diphosphate-glucuronosyltransferases (UGT).

Prodrug activation reverses the simple rule



Less morphine formed → reduced analgesia, even if parent codeine rises.
A prodrug needs metabolic activation for an important part of its effect.
Do not infer active-metabolite exposure from the parent-drug level alone.
Induction effects depend on competing activation and elimination pathways.

Exam distinctions and stopping rules

- **Smoke hydrocarbons induce CYP1A2**; nicotine is not the cause. Nicotine replacement does not maintain smoke induction.
- Carbamazepine also induces its own metabolism: autoinduction.
- No established clinically important CYP2D6 inducer.
- Stopping an inducer may raise an active substrate level; stopping an inhibitor may lower it.
- Active metabolites and multiple pathways can complicate the direction.
- Check the actual combination, symptoms and appropriate drug levels; no universal dose adjustment.

Enzyme induction and inhibition. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

Tolerance, dependence, withdrawal and sensitisation

Four different concepts

Tolerance: less effect from the same dose after repeated exposure.

Physical dependence: adaptation that produces withdrawal when drug exposure falls or is blocked.

Withdrawal: drug-specific symptoms after stopping, reducing or antagonising a drug.

Sensitisation: greater response to the same dose after repeated exposure.

Addiction: impaired control and compulsive use despite harm.

Physical dependence alone is not addiction.

Tolerance: where did the response change?

Pharmacokinetic
(change before the drug reaches the target)

Same dose



Faster metabolism/ clearance (enzyme induction)

Liver

Lower target concentration

Smaller effect

Example: Enzyme induction with repeated barbiturate exposure.

Pharmacodynamic
(change at the site of action)

Same target concentration

Drug



Desensitisation = reduced signalling

Down-regulation = fewer receptors

Receptor

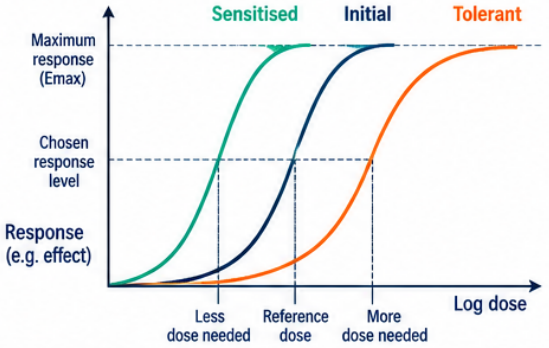
Receptor/circuit adaptation

Smaller effect

Example: Tolerance to benzodiazepine sedation.

Compensatory downstream changes also matter; not every drug simply removes receptors.

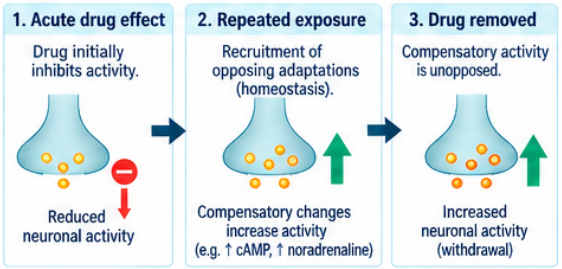
Tolerance versus sensitisation



Schematic: less dose after sensitisation; more after tolerance. Maximum response may also change.

Tolerance is effect-specific. Tolerance to one opioid effect does not guarantee protection from respiratory depression.

Dependence: adaptation unmasked



Example: Mu-opioid receptor signalling reduces activity; compensatory cyclic adenosine monophosphate signalling and noradrenergic adaptations can contribute to withdrawal.

A simplified model, not the complete mechanism.

Prescribed treatment can produce physical dependence without compulsive use.

Antagonists may precipitate withdrawal in a physically dependent person.

Recognise withdrawal; distinguish relapse

Alcohol / benzodiazepines: anxiety, tremor, insomnia; severe withdrawal may cause seizures or delirium.

Opioids: dilated pupils, sweating, runny nose, cramps and diarrhoea.

Antidepressants: dizziness, nausea, electric-shock sensations, sleep disturbance and anxiety.

Withdrawal follows a change in exposure and may include new physical symptoms. Relapse is return of the underlying illness; timing alone is not definitive.

Shorter half-life often means earlier onset. Withdrawal can be delayed or prolonged.

Individualised gradual reduction is usual; abrupt benzodiazepine cessation can be dangerous.

Sensitisation and related exam terms

Mechanism: Repeated intermittent stimulant exposure → lasting dopamine/glutamate circuit changes → enhanced response to the same exposure.

Amphetamine/cocaine sensitisation is well demonstrated experimentally; it helps explain cue-driven wanting and recurrent stimulant psychosis, but is not inevitable.

Tachyphylaxis = very rapid tolerance; ephedrine can deplete releasable noradrenaline.

Cross-tolerance = tolerance transfers to a related drug; alcohol and benzodiazepines.

Tolerance and sensitisation can coexist for different effects.

After abstinence, opioid tolerance falls: a previously tolerated dose may cause overdose.

Tolerance, dependence, withdrawal and sensitisation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuroendocrinology and basic psychopharmacology.

Credits and references: retained within the illustration.

APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings

The same hundred questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
I-S1	I-85 p 266	I-46 p 141	I-19 p 53	I-21 p 58	I-29 p 93	I-31 p 96	I-94 p 284	I-65 p 195	I-75 p 225	I-89 p 273
I-S2	I-71 p 218	I-15 p 48	I-08 p 19	I-09 p 20	I-10 p 21	I-42 p 118	I-95 p 285	I-24 p 65	I-61 p 183	I-88 p 272
I-S3	I-45 p 140	I-05 p 15	I-55 p 172	I-48 p 143	I-63 p 193	I-64 p 194	I-86 p 270	I-44 p 120	I-51 p 156	I-78 p 231
I-S4	I-36 p 111	I-04 p 14	I-20 p 56	I-39 p 114	I-11 p 22	I-13 p 26	I-43 p 119	I-74 p 224	I-52 p 159	I-70 p 202
I-S5	I-79 p 247	I-18 p 52	I-28 p 92	I-47 p 142	I-23 p 63	I-30 p 95	I-32 p 97	I-67 p 198	I-62 p 185	I-77 p 229
I-S6	I-01 p 11	I-17 p 51	I-98 p 299	I-38 p 113	I-49 p 146	I-12 p 24	I-50 p 154	I-34 p 101	I-53 p 162	I-76 p 228
I-S7	I-02 p 12	I-27 p 91	I-06 p 16	I-72 p 219	I-99 p 301	I-41 p 117	I-56 p 173	I-66 p 196	I-35 p 102	I-87 p 271
I-S8	I-03 p 13	I-16 p 50	I-07 p 18	I-92 p 282	I-81 p 250	I-84 p 256	I-57 p 175	I-59 p 180	I-68 p 199	I-69 p 200
I-S9	I-54 p 170	I-26 p 90	I-91 p 280	I-22 p 60	I-40 p 115	I-83 p 255	I-73 p 222	I-25 p 67	I-60 p 182	I-90 p 274
I-S10	I-14 p 45	I-97 p 297	I-37 p 112	I-80 p 248	I-93 p 283	I-82 p 252	I-33 p 98	I-58 p 178	I-96 p 286	I-100 p 303

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
I-S1	100	0.416	7	2	Developmental psychology 2; Functional neuroanatomy 2; Personality theory 2
I-S2	100	0.410	7	3	Neurochemistry 3; Functional neuroanatomy 2; Biostatistics 1
I-S3	100	0.402	7	3	Psychometry 3; Social 2; Neurochemistry 1
I-S4	100	0.401	6	3	Learning 3; Neurochemistry 3; Functional neuroanatomy 1
I-S5	100	0.402	7	3	Personality theory 3; Functional neuroanatomy 2; Research methodology 1
I-S6	100	0.402	7	4	Psychometry 3; Neurochemistry 2; Functional neuroanatomy 1
I-S7	100	0.402	8	3	Neurochemistry 2; Personality theory 2; Biostatistics 1
I-S8	100	0.402	6	3	Neurochemistry 2; Research methodology 2; Genetics in psychiatry 2
I-S9	100	0.392	8	2	Genetics in psychiatry 2; Functional neuroanatomy 2; Personality theory 1
I-S10	100	0.392	7	3	Neuroendocrinology 2; Research methodology 2; Nosology 2

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

Index

Every entry points at the map that answers it. A subject asked in several places carries up to three pages, and the one in bold is the map whose question is about it.

#

16PF **156**

A

acetylcholine **13**, 20, 65
ADHD **11**, **185**
adoption studies **182**
agnosia **60**, **67**
Ainsworth **194**, 273
allostatic load **299**
Alzheimer disease **11**, 63
AMPA receptor **11**, 16, **26**
amygdala **21**, 26, **51**
analysis of variance **219**, 224, **229**
Anna Freud **90**, 93, **101**
apraxia **45**, **60**
archetype **92**, 95, **96**
association study **183**
attachment theory **194**, 271, **273**
aversion therapy **117**, 120

B

B12 **21**, 180
Bandura **115**
Baron-Cohen **271**
basal ganglia **18**, 24, **58**
basic emotions **113**, **114**, 118
Beck Depression Inventory **159**
behaviour therapy **115**, 117, **120**
benzodiazepine **11**, 12, **22**
Berne **102**
Bhatia **119**
Binet-Kamat **111**, **119**
biological marker **15**, 18, **19**
Bonferroni **229**
Bowlby **101**, **194**, 273
Brodmann **52**

C

CA1 **58**, 63
CA3 58, **63**
CA4 **58**, 63
Carl Jung **92**, 95, **97**
caudate nucleus **48**, 58, 65
cerebral cortex **52**, 53
Charles Bonnet syndrome **67**, 286
Chi Square test **218**
Children's Apperception Test **162**
Circadian rhythm sleep-wake disorders **22**
classical conditioning **115**, **117**, 194
classificatory system **280**
clinical trial **252**, **256**, 301
Clinical Trials Registry of India **256**
cohort study **231**
collective unconscious **95**, **96**, 97
communication deviance **91**
comorbidity **280**, **283**, 301
complement C4 **26**, **170**, 173
concept of self **92**, 96, 98
concordance **178**
confidence interval **222**, 224, 225
confounding **18**, 185, **222**
consciousness **96**, 142, **285**
coping **93**, 198, **299**
copy number variant **170**, 173
corpus callosum **45**, 56, 67
cortisol **19**, 299, **303**
Cronbach **248**
culture **95**, 97, **202**
culture bound syndrome **193**, 200
CYP2C19 **301**
CYP2D6 **301**

D

defence mechanism **90**, 93, 101
degrees of freedom **218**, 219, **229**
delirium **11**, 12, **15**
delusional memory **284**
delusional mood **284**
delusional perception **284**
deviation quotient **111**, 119, 146
displacement **90**, **98**
dizygotic **170**, **178**
DNA methylation **180**
dopamine **13**, 19, **24**

double bind 91
dowry 195
Draw a Person 162
DSM-5 193, 198, 282
DSM-III 280, 283
Durkheim 196
Dysexecutive 50, 60

E

ECT 11, 12
EEG 15, 20
effect modification 222, 225
ego defence 90, 93, 101
Electroconvulsive therapy 26
emotional intelligence 111, 113, 114
endocannabinoid 18
endophenotype 15, 19
epigenetics 172, 180
epilepsy spectrum disorder 282
episodic memory 48, 56
equal environments assumption 178
Erich Fromm 91, 95, 97
Erikson 95, 97, 270
ethics committee 256
ethnic density 200
ethology 194
evidence based medicine 301
excitotoxicity 14, 16
Exner 143
explanatory model 193, 200
expressed emotion 91
external validity 222, 247, 248
extinction 26, 51, 120

F

family study 170, 182, 280
Feighner criteria 280
focus group 247
Folstein 142
forest plot 252
Friedman test 219, 224
frontal lobe 50, 60

G

GABA 13, 20, 52
Gardner 111
gene mapping 173, 175, 183
genetic correlation 173, 185
genetics 19, 170, 180
genome wide association 173, 175, 183
Gerstmann syndrome 60
Glasgow Coma Scale 285
glutamate 11, 14, 16
Goleman 113
grounded theory 247
Guilford 111

H

Hamilton Depression Rating Scale 159
Harlow 194, 273
heritability 170, 172, 173
heterogeneity 252
Hindi Mental State Examination 142
hippocampal formation 12, 48, 63
hippocampal subfield 58, 63
hippocampus 11, 12, 58
Huntington disease 48, 58, 172
hypnagogic 286
hypnopompic 286
hypothesis 143, 162, 252

I

ICD-11 280, 282, 283
identity crisis 274
idioms of distress 193, 198, 200
illness behaviour 112, 193, 202
illusion 286
immigration 200
imprinting 180, 194, 273
individuation 95, 96, 97
inflammation 14, 303
informed consent 256
intellectual disability 111, 119
intelligence 111, 113, 114
internal validity 247, 248

J

James-Lange 114, 118
Jaspers 92, 284, 285

K

Ketamine 16, 26
Kluver-Bucy syndrome 51
Kohler 115
Kohut 92, 97
koro 193, 202

Korsakoff 11, 48
Kraepelin 280, 282, 283
Kruskal-Wallis 219, 224, 228

L

Lazarus 114, 118
learning 11, 21, 115
limbic system 48, 51, 56
linkage analysis 183
linkage disequilibrium 173, 175, 183
LOD score 175, 183
logistic regression 225, 231, 255
long term potentiation 11, 14, 63
Lorenz 194

M

Malin 119
Mann-Whitney 219, 224, 228
Marchiafava-Bignami disease 45
marital schism 91
marriage 195, 196, 198
McNemar's test 218, 219
mediation 21, 112, 222
Melanie Klein 97, 101
melatonin 13, 297
Mendelian randomisation 185, 222, 248
meta analysis 252
migration 172, 198, 200
mini mental state examination 142, 154, 285
mixed episode 282
monozygotic 170, 178, 180
Montgomery Asberg 159
Montreal Cognitive Assessment 142
MT1 13, 297
MT2 13, 297
multiple regression 225
Murray 140

N

narcolepsy 13, 20, 22
National Mental Health Survey 141, 255
natural killer cell 303
neglect 60, 172
neo-Freudian 91, 95, 97
neo-Kraepelinian 280, 282, 283
Neocortex 21, 52
New Drugs and Clinical Trials Rules 256
nitric oxide 14, 16
NMDA receptor 11, 14, 16
Non-parametric tests 219, 224
noradrenaline 13, 20, 297
null hypothesis 219, 224, 252

O

object relations 162
Obsessive compulsive disorder 15
obstructive sleep apnoea 22
occipital cortex 60, 67
odds ratio 218, 222, 231
operant conditioning 112, 115, 117
operational criteria 154, 280

P

p value 183, 224
P300 15, 19
P50 15, 19
paired t-test 219, 224, 228
Papez circuit 48, 53, 56
parametric test 218, 219, 224
parietal lobe 45, 60, 67
Pavlov 115, 117, 120
Pearson 224
personalised medicine 19, 301
pharmacogenetics 301
Piaget 266, 271, 272
pineal gland 24, 297
pleiotropy 173, 185
polygenic risk score 173
polysomnography 13, 20, 22
possession state 193, 200
post-Freudian 95, 96, 97
prefrontal cortex 11, 12, 50
Premack 271
prevalence 154, 247, 255
primary delusion 284
procedural memory 11, 21
projection 16, 53, 90
projective hypothesis 143, 162
projective identification 90, 93
projective test 140, 143, 162
pseudohallucination 286
pseudomutuality 91
psychometric properties 119, 146, 248
psychosexual 270, 272
publication bias 252
putamen 48, 58, 65

Q

qualitative research 247
quantitative research **247**, 248, 250

R

reaction formation **90**, 93, 101
regression analysis **225**, 255
reinforcement **112**, 117, 194
relative risk 218, 222, **231**
reliability 19, 146, **248**
religion **199**, 200
repression **90**, 93, 98
Research Diagnostic Criteria 280
research question 250, **252**, 256
restless legs syndrome 22
reticular activating system 285
Rorschach **143**, 156, 162
rubber fence 91

S

Salovey 113
sample size 175, 228, **250**
sampling 143, 162, **250**
saturation 22, **247**
Schachter **114**, 118
Schaffer 16, **63**
schizophrenogenic mother 91
self efficacy 112
sensitivity 19, **146**
Sentence Completion Test 143
separation anxiety 273
serotonin 13, 20, **297**
shadow **96**, 97
Skinner **115**, 117
sleep 12, **13**, 15
Sleep related breathing disorders 22
Sleep related movement disorders 22
social learning theory 112
Spearman **111**, 224, 248
specificity 19
splitting **90**, 93
standard deviation 111, 119, **228**
standard error 219, **228**, 255
standardisation **146**, 159
Sternberg 111
stigma **198**, 202, 247
strange situation 273
stress 26, 51, **299**
striatum 11, 21, **58**
structural model **98**, 102
stupor 285
sublimation **90**, 101
substantia nigra **24**, 58, 65
suppression 18, 19, **90**
suprachiasmatic nucleus **13**, 297
synaptic plasticity 11, 14, **26**
systematic desensitisation 120

T

Test-retest 248
thalamus 13, 48, **53**
Thematic Apperception Test **140**, 143, 156
theory of mind 92, 98, **271**
Thorndike **115**, 117
Thurstone 111
Tinbergen 194
token economy 117
Tolman 115
topographical model 98
transactional analysis 102
treatment gap 255
triangulation 247
true hallucination 286
Tukey 229
twin studies 170, **178**, 182

U

Udai Pareek 141
unconscious 90, 95, **98**
undoing **90**, 93, 101

V

Vaillant 90, **93**
Vineland Social Maturity Scale 119
Vygotsky 266

W

Watson **115**, 117, 120
Wechsler **111**, 119
Werther effect 196
Wilcoxon **219**, 224, 228
Winnicott **92**, 97
word association test **96**, 97
working memory 12, **21**, 24

Z

zone of proximal development 266

Illustrations

Plate identifiers and the page on which each illustration appears. Landscape artwork is turned 270 degrees on portrait A4 sheets.

I-001 Glutamate 17
I-002 Dopamine 25
I-003 Classification of Neurotransmitters 27
I-004 Acetylcholine 28
I-005 Neuropeptides 29
I-006 Opiates and the Endogenous Opioid System 30
I-007 Neurodevelopment 31
I-008 Neurotrophins 32
I-009 Default mode network 33
I-010 Salience network 34
I-011 Central executive network 35
I-012 Triple-network interaction 36
I-013 Brain-Derived Neurotrophic Factor 37
I-014 Novel Neurotransmitters 38
I-015 Electroencephalography 39
I-016 Event-related potentials 40
I-017 Serotonin 41
I-018 Noradrenaline 42
I-019 Gamma-aminobutyric acid 43
I-020 Corpus Callosum Parts 47
I-021 Papez Circuit Mnemonic 49
I-022 Thalamus Relay Anatomy 54
I-023 Thalamus Pathways 55
I-024 Parts of Limbic System 57
I-025 Basal Ganglia and Pathways 59
I-026 Frontal Lobe Syndromes 61
I-027 Lobe Function Tests 62
I-028 Hippocampal Pathology 64
I-029 Caudate Nucleus Anatomy 66
I-030 Visual Cortex 68
I-031 Thalamic Relay Schema 69
I-032 Hypothalamus 70
I-033 Cerebellum Anatomy and Pathology 71
I-034 Cerebellum 72
I-035 Cerebrospinal fluid 73
I-036 Eye-movement control 74
I-037 Bedside neurological localisation 75
I-038 Computed tomography 76
I-039 Structural magnetic resonance imaging 77
I-040 Functional magnetic resonance imaging 78
I-041 Positron emission tomography 79
I-042 PFC Subdivisions 80
I-043 Single-photon emission computed tomography 81
I-044 Neuroimaging comparison 82
I-045 Amygdala Location 83
I-046 Amygdala Connections 84
I-047 Amygdala Inputs and Outputs 85
I-048 Amygdala Schematic 86
I-049 Brodmann Areas 87
I-050 Cerebral blood supply 88
I-051 Sigmund Freud - Models of the Mind 99
I-052 Sigmund Freud - Psychosexual Development 100
I-053 Humanistic Theories of Personality 103
I-054 Object Relations Theories 104
I-055 Otto Rank 105
I-056 Trait and Type Approaches to Personality 106
I-057 Adolf Meyer's Psychobiology 107
I-058 Existential School of Psychology 108
I-059 Positive Psychology 109
I-060 Theories of Intelligence and Tests 121
I-061 Perception 122
I-062 Attention 123
I-063 Forgetting 124
I-064 Memory errors and distortions 125
I-065 Problem-solving and decision-making 126
I-066 Motivation 127
I-067 Creativity 128
I-068 Sensory deprivation 129
I-069 Mind-body problem 130
I-070 Social Learning 131
I-071 Emotional Intelligence and Theories 132
I-072 Basic Emotions and Theories 133
I-073 Learning Theories 134
I-074 Learned Helplessness 135
I-075 Classical Conditioning 136
I-076 Operant Conditioning 137
I-077 Applied Behaviour Analysis 138
I-078 Projective Tests 145
I-079 Positive and Negative Syndrome Scale 147
I-080 Brief Psychiatric Rating Scale 148
I-081 Indian Disability Evaluation and Assessment Scale - IDEAS 149

I-082 Indian Scale for Assessment of Autism - ISAA 150
I-083 Rating Scales 151
I-084 Young Mania Rating Scale 152
I-085 Yale-Brown Obsessive Compulsive Scale 153
I-086 Structured Interviews 155
I-087 Personality Theories 157
I-088 Personality Theories Compared 158
I-089 Hamilton Depression Rating Scale 160
I-090 Montgomery-Åsberg Depression Rating Scale 161
I-091 Neuropsychological assessment overview 163
I-092 ADPMR Disability Assessment 164
I-093 Executive-function tests 165
I-094 Memory assessment 166
I-095 Neuropsychological batteries and Indian adaptations 167
I-096 Adaptive functioning and developmental assessment 168
I-097 Genetics in Bipolar and Schizophrenia 171
I-098 Genome-wide association studies 174
I-099 Gene Mapping Strategies 177
I-100 Family, twin and adoption studies 179
I-101 Epigenetics 181
I-102 Modern genetic approaches and gene therapy 184
I-103 Cross-disorder genetic overlap 186
I-104 Inheritance patterns and pedigree interpretation 187
I-105 Genetic terminology and mechanisms 188
I-106 Genetic counselling in psychiatry 189
I-107 Gene-environment interaction versus gene-environment correlation 190
I-108 LOD Score 191
I-109 Theories of Suicide 197
I-110 Conformity, compliance and obedience 203
I-111 Group processes 204
I-112 Aggression 205
I-113 Crowd and mob psychology 206
I-114 Psychological aspects of sexual violence 207
I-115 Rumour 208
I-116 Prosocial behaviour and helping 209
I-117 Guru-chela relationship 210
I-118 Culture and Psychiatry 211
I-119 Prejudice, stereotypes and discrimination 212
I-120 Stigma, Stereotype and Prejudice 213
I-121 Attitudes and attitude change 214
I-122 Cognitive dissonance 215
I-123 Attribution and attribution biases 216
I-124 Statistical Tests 221
I-125 Confounding, effect modification and mediation 223
I-126 Factor analysis 226
I-127 Regression Analysis 227
I-128 ANOVA Explained 230
I-129 Absolute risk reduction and number needed to treat 232
I-130 Types of data and measurement scales 233
I-131 OR vs RR 234
I-132 Sensitivity and specificity 235
I-133 Predictive values and likelihood ratios 236
I-134 Receiver operating characteristic curve 237
I-135 Screening principles and biases 238
I-136 Incidence and prevalence 239
I-137 Survival analysis 240
I-138 Central tendency and dispersion 241
I-139 Normal distribution and standard scores 242
I-140 Standard deviation, standard error and confidence intervals 243
I-141 Hypothesis testing, errors and power 244
I-142 Correlation 245
I-143 Reliability and Validity 249
I-144 Sampling and Sample Size 251
I-145 Systematic review and meta-analysis 254
I-146 Clinical trial phases 257
I-147 Research ethics and informed consent 258
I-148 Randomised controlled trial 259
I-149 Study designs compared 260
I-150 Bias in research 261
I-151 Forest plots and heterogeneity 262
I-152 Critical appraisal and reporting guidelines 263
I-153 Randomisation, allocation concealment and blinding 264
I-154 Lawrence Kohlberg - Moral Development 268
I-155 Lev Vygotsky - Sociocultural Development 269
I-156 Alexander Thomas and Stella Chess - Temperament 275

I-157 Margaret Mahler - Separation-Individuation 276
I-158 Language Development 277
I-159 Motor Development 278
I-160 Normality 287
I-161 Mood and affect terminology 288
I-162 Formal thought disorder 289
I-163 Thought possession and passivity phenomena 290
I-164 Kurt Schneider's first-rank symptoms 291
I-165 Disorders of self-experience 292
I-166 Attention and orientation in the mental state examination 293
I-167 Catatonic signs 294
I-168 Insight and judgement 295
I-169 Pineal Gland Mechanism 298
I-170 Pharmacogenomics and Pharmacogenetics 302
I-171 Cytokines 304
I-172 Hypothalamic-pituitary-thyroid axis 305
I-173 Hypothalamic-pituitary-gonadal axis 306
I-174 Receptor pharmacology 307
I-175 Second-messenger systems 308
I-176 Pharmacokinetics - absorption and distribution 309
I-177 Pharmacokinetics - metabolism and elimination 310
I-178 Enzyme induction and inhibition 311
I-179 Tolerance, dependence, withdrawal and sensitisation 312

WEAVE SPRINT

This book tells you what to answer. Sprint teaches you the subject.

Weave 100 is a shortlist with model answers. Weave Sprint is the reading behind it: full chapters on every topic in every paper, with a colour memory code, a clickable contents and a complete index. The Sprint day is named at the foot of every map in this book.

4

papers

One book for each paper of the written examination, cover to cover.

35

days

A day of notes per topic, written to be understood the first time.

24

volumes

Split for a coil bind, so a volume opens flat on a ward desk.

0

rupees

Free to read, free to download, free to print. It stays that way.

weave.clinic/sprint

TAP OR SCAN TO OPEN

STUDY TOGETHER

A hundred questions is a shortlist, not a syllabus.

This counting started as a room full of residents comparing papers and arguing about what actually comes. That room is still open. Bring a question we missed, a paper we have not read, and the corrections. Every one of them makes the next count better.

Join the study group

https://chat.whatsapp.com/H7TsLGK2wFl4bSe2r0Zfzn?mode=gi_t

CREDITS

Who made this, and from what

Dr. Wilfred Dsouza	Counting, area weighting, selection, and the answer maps. Lead author of the Weave 100 series.
Dr. Niharika Reddy	Clinical review of the personality, developmental and cross-cultural areas, and of the psychometry maps. Co-author.

The record. Every question in this book was read off a paper set by DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University or WBUHS. Where a paper reached us as a mirror, an archive or a transcription rather than an official file, that session carries a lower weight in the count, and the weightage chapter says by how much.

The residents. Papers, corrections and photographs of question papers came from residents across several states who had no reason to send them except that somebody else would need them. This book is mostly their work.

The tools. The counting, weighting, selection and typesetting all run as code in this repository, so any number in this book can be traced back to the row it came from.

PRINTING AND DISTRIBUTION

Notes Next Door (NND), Delhi is the printing and distribution partner for the Weave study series. The soft copy of this book is free at weave.clinic/100, and the print-ready files are published in the same place for anyone who would rather print a set themselves. Weave 100 is free and stays free.

WEAVE 100 - PAPER I

Eight boards were counted: DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University and WBUHS. Seventy-eight sittings of theory papers, weighted by how recent they are and how well the paper is evidenced.

From that count, twelve areas were weighted and one hundred questions were drawn in proportion. Each is solved as a one-page answer map: mark split, skeleton, the content that earns the marks, and what the examiner ticks. Ten ready-made sets at the back turn the hundred into ten sittings.

A frequency count is a strong prior, not a prophecy.

Free at weave.clinic/100. Print by Notes Next Door.

WEAVE

weave.clinic · wilfred@weave.clinic · niharika@weave.clinic