PART I · PAPER I

I

Basic Sciences

Part I gathers the neuroscience, psychology, social science, and statistics every later answer rests on, the groundwork the discipline is built from.

ONE-DAY REVISION · PAPER 1

Neuroanatomy & neurochemistry, made retrievable

This is the heart of Paper 1. Hold the pathway (origin → target → function), the receptor (family, coupling), and the disorder map. Dopamine pathways and serotonin are asked almost every sitting; the disorder neurobiology is where the marks are won. Every section is a 10-mark answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY FACT ● MNEMONIC

The colour tells you what kind of fact it is.

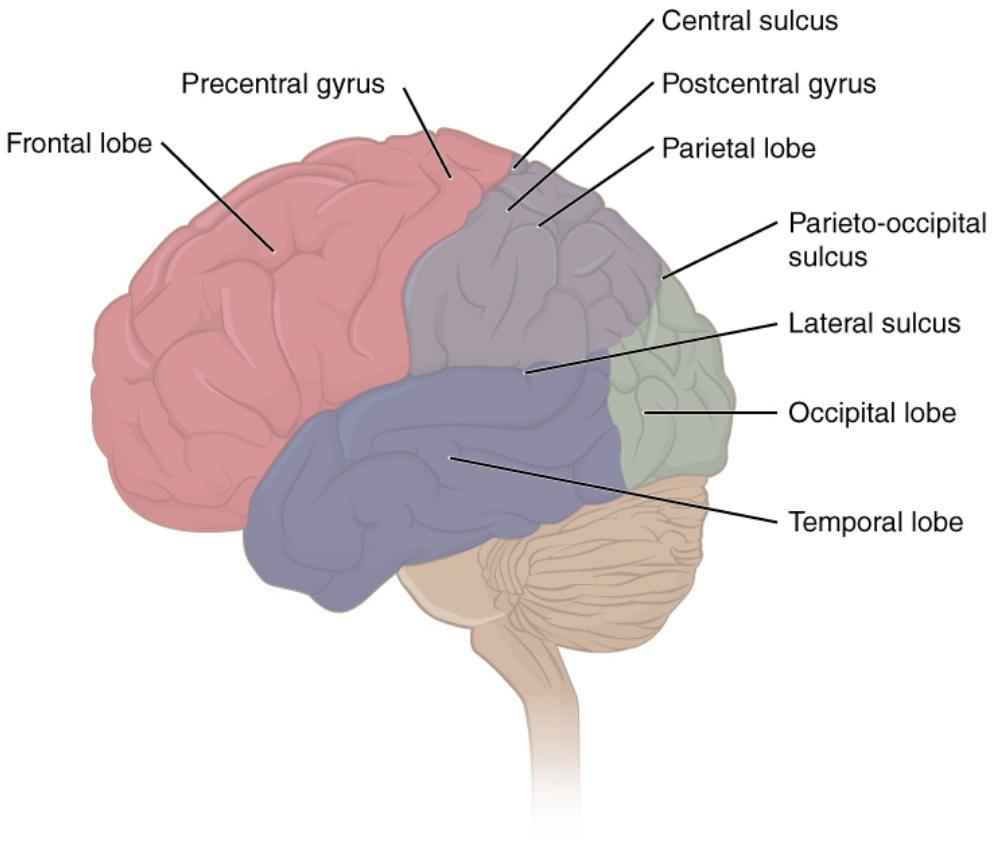
★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The four dopamine pathways (asked ~6 times, the single most reliable question): **mesolimbic** (VTA → accumbens) → positive symptoms, **mesocortical** (VTA → PFC) → negative/cognitive symptoms, **nigrostriatal** (SNc → striatum) → EPS, **tuberoinfundibular** (hypothalamus → pituitary) → prolactin (mnemonic MeMe NiTu).
2. Monoamine cell-body origins: 5-HT = raphe nuclei, NE = locus coeruleus, DA = VTA / substantia nigra, ACh = nucleus basalis of Meynert (degenerates in Alzheimer's), histamine = tuberomammillary nucleus.
3. **D2 is the target of all antipsychotics**: occupancy **65-80%** is the therapeutic window, **>80% → EPS**; D1/D5 are Gs (↑cAMP), D2/D3/D4 are Gi (↓cAMP).
4. Rate-limiting enzymes and metabolites: tyrosine hydroxylase (catecholamines, needs BH4), tryptophan hydroxylase (5-HT), GAD (GABA, needs B6); DA → HVA, 5-HT → 5-HIAA (**low CSF 5-HIAA = impulsive/violent suicide**), NE → MHPG.
5. Papez circuit (draw-it): hippocampus → fornix → mammillary bodies → anterior thalamic nucleus → cingulate gyrus → parahippocampal gyrus → hippocampus; mammillary-body damage in Korsakoff (thiamine deficiency) → amnesia plus confabulation.

1 Brain regions & lobes orientation

Map function to region first; the psychiatric relevance follows. The prefrontal cortex carries most of the marks.

LOBES & SURFACE LANDMARKS



Lateral surface: the frontal, parietal, temporal and occipital lobes, divided by the central (Rolandic) and lateral (Sylvian) sulci. OpenStax Anatomy and Physiology, CC BY 4.0.

Lobe / region	Core function	Psychiatric relevance
Frontal lobe	Voluntary movement, speech (Broca), executive function, personality	Seat of the prefrontal syndromes (below). ~1/3 of cortex
DLPFC (BA 9, 46)	Working memory, planning, set-shifting, abstract reasoning	Hypofrontality in schizophrenia; left DLPFC = high-freq TMS target for depression
OFC (BA 11, 47)	Impulse control, social behaviour, reward evaluation	Disinhibition (Phineas Gage); OFC-caudate loop hyperactive in OCD
ACC (BA 24, 32; subgenual 25)	Motivation, error / conflict monitoring, emotion regulation	Subgenual ACC (BA 25) = DBS target in treatment-resistant depression
vmPFC (BA 10, 25)	Emotional decision-making, fear extinction, theory of mind	Somatic-marker hypothesis (Damasio); poor risk appraisal
Temporal lobe	Auditory processing, language comprehension (Wernicke, BA 22), memory	Superior temporal gyrus activates during auditory hallucinations; TLE
Parietal lobe	Somatosensory integration, body schema, visuospatial attention	Right parietal lesion → left neglect; angular gyrus → Gerstmann
Limbic system	Emotion, motivation, memory (amygdala, hippocampus, cingulate)	Core of mood, anxiety, PTSD, addiction circuitry (see Section 5)

● FRONTAL SYNDROMES

DLPFC → pseudodepressed (apathy, ↓ fluency, poor planning · mimics depression). **OFC → pseudopsychopathic** (disinhibition, poor judgement · mimics mania/PD). **Bilateral ACC → akinetic mutism** (mutism, akinesia, profound apathy).

● MNEMONIC

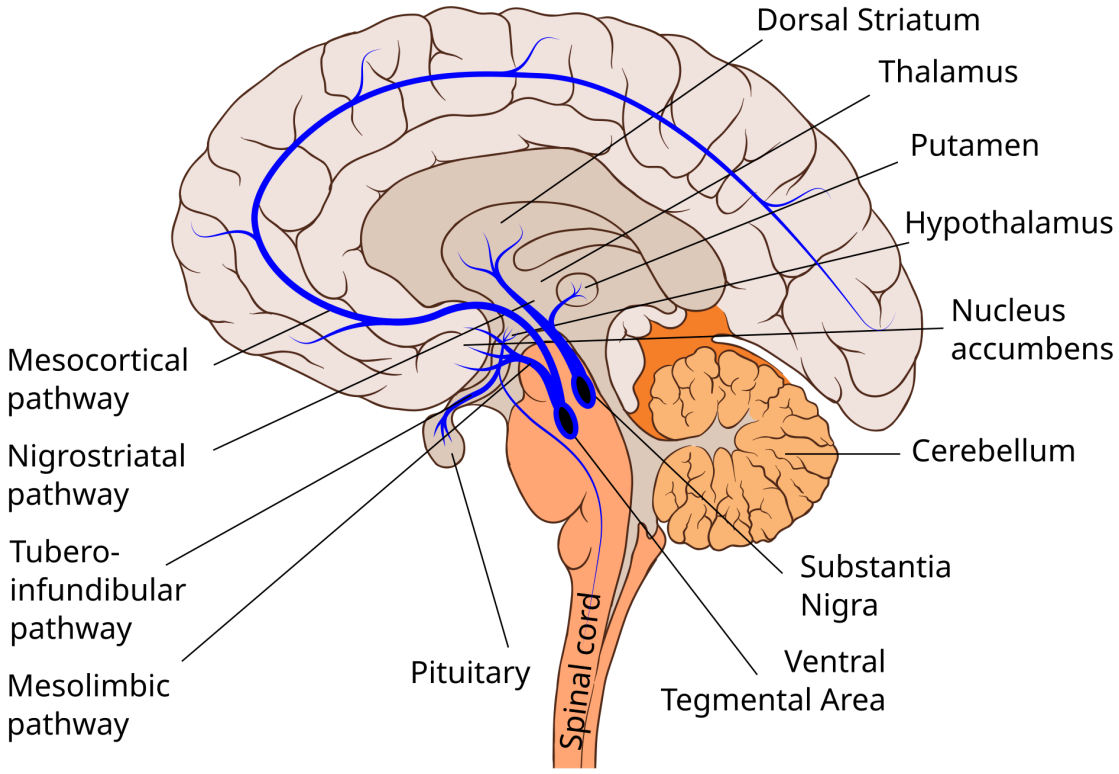
"DOVe-A"

DLPFC *plans* · **O**FC *behaves* · **V**mPFC *values* · **A**CC *motivates*. Damage maps to dysexecutive, disinhibited, impaired-decision, and akinetic-mute pictures respectively.

2 Neurotransmitter systems

The four dopamine pathways (asked ~6 times · the single most reliable question)

THE FOUR DOPAMINE PATHWAYS



Each tract labelled: mesolimbic (VTA → nucleus accumbens, positive symptoms), mesocortical (VTA → PFC, negative/cognitive), nigrostriatal (substantia nigra → striatum, EPS), tuberoinfundibular (hypothalamus → pituitary, prolactin). Wikimedia Commons, CC BY-SA.

Pathway	Origin → target	Function	Clinical relevance
Mesolimbic	VTA → nucleus accumbens, amygdala	Reward, motivation, salience	Hyperactivity → positive symptoms (hallucinations, delusions). Antipsychotic D2 blockade here = therapeutic. Addiction
Mesocortical	VTA → prefrontal cortex (DLPFC, vmPFC)	Executive function, cognition, working memory	Hypoactivity → negative & cognitive symptoms (avolition, alogia, anhedonia)
Nigrostriatal	Substantia nigra pars compacta → dorsal striatum (caudate + putamen)	Motor control, procedural learning	Degeneration (>80% loss) → Parkinson's. D2 blockade → EPS, tardive dyskinesia
Tuberoinfundibular	Hypothalamus (arcuate n.) → anterior pituitary	Tonic inhibition of prolactin	D2 blockade → hyperprolactinaemia (galactorrhoea, amenorrhoea, sexual dysfunction)

MNEMONIC

“MeMe NiTu”

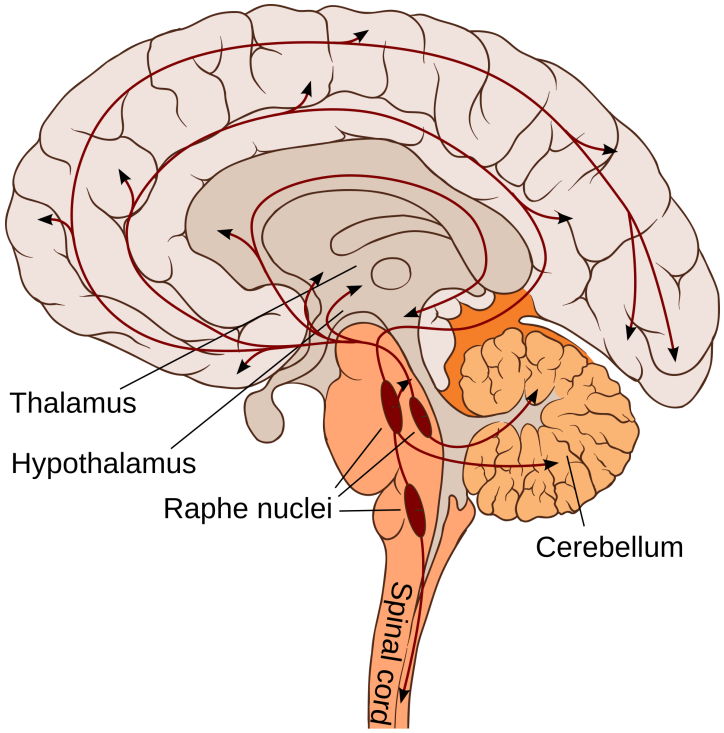
Mesolimbic, **Me**socortical, **Ni**grostriatal, **Tu**beroinfundibular. Or: *Meso rewards, Meso thinks, Nigro moves, Tubero milks* – the verb is the function.

HOLD THIS

The antipsychotic dilemma in one line: you want to block **mesolimbic** DA (helps psychosis) but the same drug blocks **nigrostriatal** (EPS) and **tuberoinfundibular** (prolactin), and worsens **mesocortical** hypofunction (negative symptoms).

Monoamine origins · the brainstem nuclei to name

SEROTONIN, NORADRENALINE & ACETYLCHOLINE ORIGINS



Serotonin (shown) arises from the raphe nuclei and projects diffusely to the whole cortex and cord. Noradrenaline arises from the locus coeruleus; acetylcholine from the nucleus basalis of Meynert. Wikimedia Commons, CC BY-SA.

System	Cell-body nucleus	Projection & function	Clinical anchor
Serotonin (5-HT)	Raphe nuclei (dorsal & median, brainstem)	Dorsal → cortex, basal ganglia, limbic (mood, anxiety); median → hippocampus (memory). Projects to virtually every region	Depression, anxiety, OCD, impulsivity. SSRI / clomipramine target
Noradrenaline (NE)	Locus coeruleus (dorsal pons)	Sole NE source for neocortex; arousal, vigilance, fight-or-flight. Fires in wake, silent in REM	The “panic button”: yohimbine provokes, clonidine calms. PTSD hyperarousal, ADHD
Acetylcholine (ACh)	Nucleus basalis of Meynert (basal forebrain); PPN/LDT (brainstem)	NBM → cortex, hippocampus (memory, attention); PPN/LDT → thalamus (REM, arousal)	NBM degenerates in Alzheimer's. Anticholinergics → delirium
Histamine	Tuberomammillary nucleus (posterior hypothalamus)	Diffuse cortical projection; wakefulness	H1 blockade → sedation + weight gain (the “hidden” NT)

System	Cell-body nucleus	Projection & function	Clinical anchor
GABA	Interneurons throughout CNS	Most abundant inhibitory NT (~30–40% of synapses)	Anxiety, epilepsy, alcohol; PV-interneuron deficit in schizophrenia (gamma oscillations)
Glutamate	Cortical pyramidal neurons (widespread)	Most abundant excitatory NT (~50–60% of synapses)	NMDA hypofunction (schizophrenia); ketamine (depression); excitotoxicity

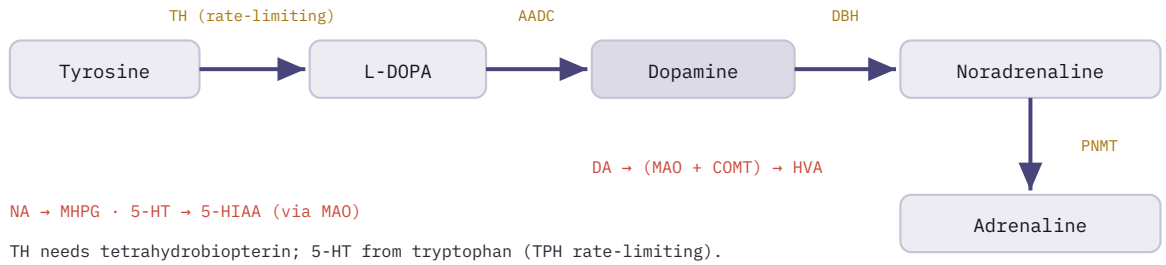
MNEMONIC

"GAG inhibits, GANG excites"

Inhibitory **GAG**: GABA (brain), Adenosine, Glycine (cord). Excitatory **GANG**: Glutamate, Aspartate, Noradrenaline, Glutamate (doubled – it is *the* excitatory NT). Shorthand: **GABA brakes, Glutamate gas.**

3 Neurochemistry · synthesis & breakdown

CATECHOLAMINE SYNTHESIS & BREAKDOWN



Tyrosine hydroxylase is the rate-limiting step for catecholamines. Metabolites: dopamine → HVA, noradrenaline → MHPG, serotonin → 5-HIAA.

◆ **Catecholamine cascade** – Tyrosine →*(tyrosine hydroxylase, rate-limiting)*→ L-DOPA →*(DOPA decarboxylase / AADC)*→ Dopamine →*(dopamine β-hydroxylase, in vesicles)*→ Noradrenaline →*(PNMT, adrenal medulla only)*→ Adrenaline.

● **Serotonin** – Tryptophan →*(tryptophan hydroxylase, rate-limiting; TPH2 in CNS)*→ 5-HTP →*(AADC)*→ 5-HT. Tryptophan is the only essential-amino-acid precursor for a monoamine.

● **GABA & glutamate** – Glutamate →*(GAD, needs vitamin B6 / pyridoxine)*→ GABA. Glutamine →*(glutaminase)*→ Glutamate. (Isoniazid overdose depletes B6 → ↓ GABA → seizures → treat with IV pyridoxine.)

NT	Broken down by	Final metabolite (measured)
Dopamine	MAO + COMT	HVA (homovanillic acid)
Serotonin	MAO-A	5-HIAA (low CSF 5-HIAA → impulsivity, violent suicide)
Noradrenaline	MAO-A (central); COMT (peripheral → VMA)	MHPG (primary CNS metabolite)

● CLASSIC TRAP

L-DOPA crosses the blood-brain barrier (hence its use in Parkinson's); **dopamine itself does not**. Likewise 5-HT does not cross the BBB – it must be synthesised centrally. COMT matters most in the PFC, where DAT density is low.

4 Receptors

System	Receptor	Class / coupling	High-yield point
Dopamine	D1, D5 (D1-like)	GPCR, Gs → ↑ cAMP	D1 most abundant; drives direct pathway
	D2, D3, D4 (D2-like)	GPCR, Gi → ↓ cAMP	D2 = target of all antipsychotics. 65–80% occupancy = therapeutic window; >80% → EPS
Serotonin	5-HT1A	GPCR, Gi (inhibitory)	Autoreceptor; buspirone partial agonist; desensitisation underlies SSRI delay
	5-HT2A	GPCR, Gq (excitatory)	Atypical antipsychotics block it; LSD/psilocybin agonise it
	5-HT3	Ionotropic (Na+/K+)	Only ionotropic 5-HT receptor; emesis (ondansetron)
GABA	GABA-A	Ionotropic, Cl− channel	Pentamer; BZD ↑ **frequency**, barbiturate ↑ **duration** of Cl− opening
	GABA-B	GPCR (Gi), metabotropic	↑K+ / ↓Ca2+; baclofen agonist
Glutamate	NMDA	Ionotropic, Ca2+-permeable	Voltage-dependent Mg2+ block; needs glutamate *and* glycine; basis of LTP. Ketamine/PCP block it
	AMPA, kainate, mGluR	AMPA/kainate ionotropic; mGluR1–8 GPCR	AMPA = fast transmission, relieves Mg2+ block; mGluR Group I = Gq, Groups II/III = Gi
Adrenergic	α1, α2, β1–3	All GPCR (α1 Gq; α2 Gi; β Gs)	α2 = presynaptic autoreceptor (clonidine); α1 block → orthostasis (prazosin for PTSD nightmares)
Cholinergic	Nicotinic; Muscarinic M1–M5	Nicotinic ionotropic; muscarinic GPCR	M1 cognition; anticholinergic side-effects; KarXT/Cobenfy = M1/M4 agonist antipsychotic (2024)

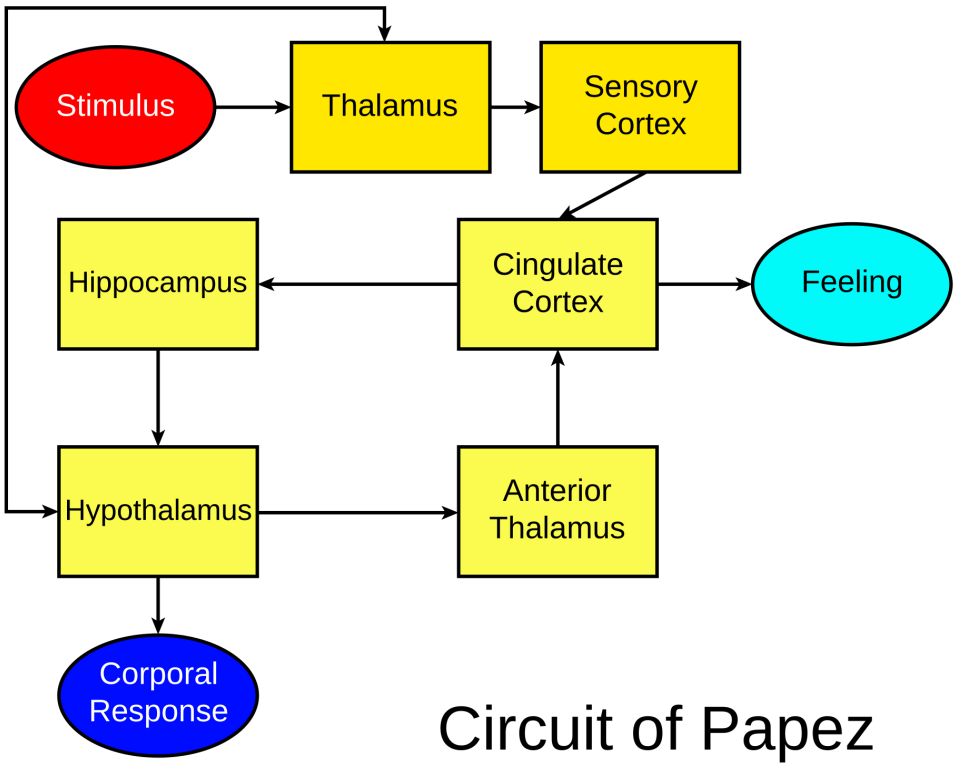
● RULE

The only **ionotropic** (ligand-gated channel) receptors in this table: GABA-A, NMDA, AMPA, kainate, nicotinic ACh, and 5-HT3. Everything else is a metabotropic GPCR. BZDs are fast because they hit ionotropic GABA-A; SSRIs are slow because monoamine receptors are metabotropic and act through downstream plasticity.

5 Key circuits

Papez circuit (draw-it question)

THE PAPEZ CIRCUIT



The emotion/memory loop: hippocampus → fornix → mammillary bodies → anterior thalamus → cingulate gyrus → back to hippocampus (with hypothalamic output). Damaged in Korsakoff. Wikimedia Commons, CC BY-SA.

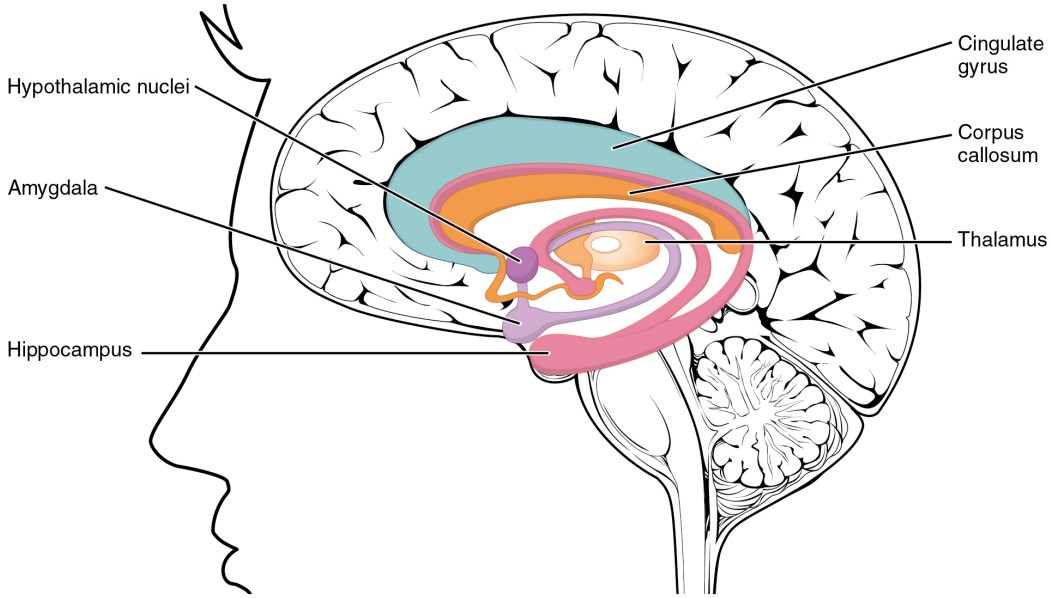
- **The loop, in order** – Hippocampus →*(fornix)*→ Mammillary bodies →*(mammillothalamic tract)*→ Anterior thalamic nucleus → Cingulate gyrus →*(cingulum)*→ Parahippocampal gyrus → back to Hippocampus.

MNEMONIC

“Hippocampus Makes All Memories Come Home”

Proposed by Papez (1937) as the emotion circuit; now seen mainly as a **memory** circuit. Damage anywhere → anterograde amnesia. Korsakoff (thiamine deficiency) damages mammillary bodies → amnesia + confabulation.

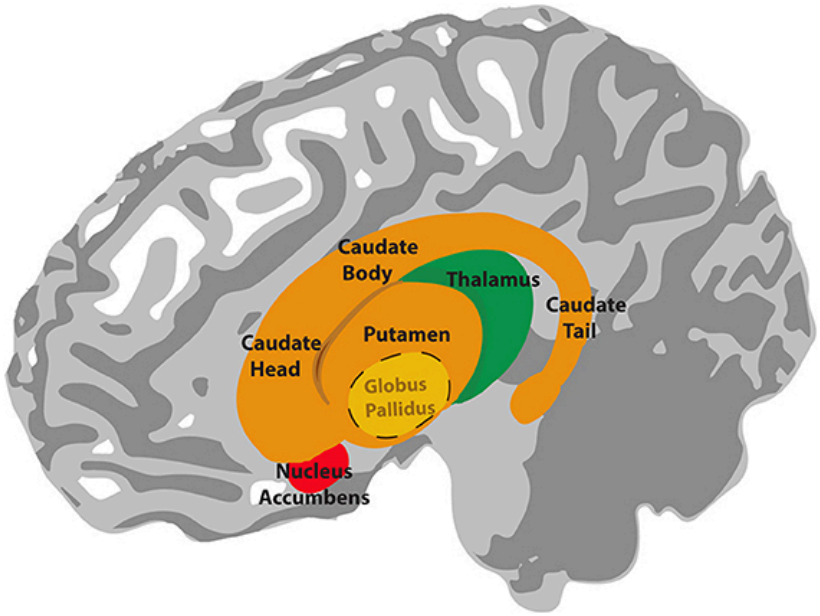
LIMBIC SYSTEM STRUCTURES



Cingulate gyrus, hippocampus, amygdala, thalamus and hypothalamus on the medial view. OpenStax Anatomy and Physiology, CC BY 4.0.

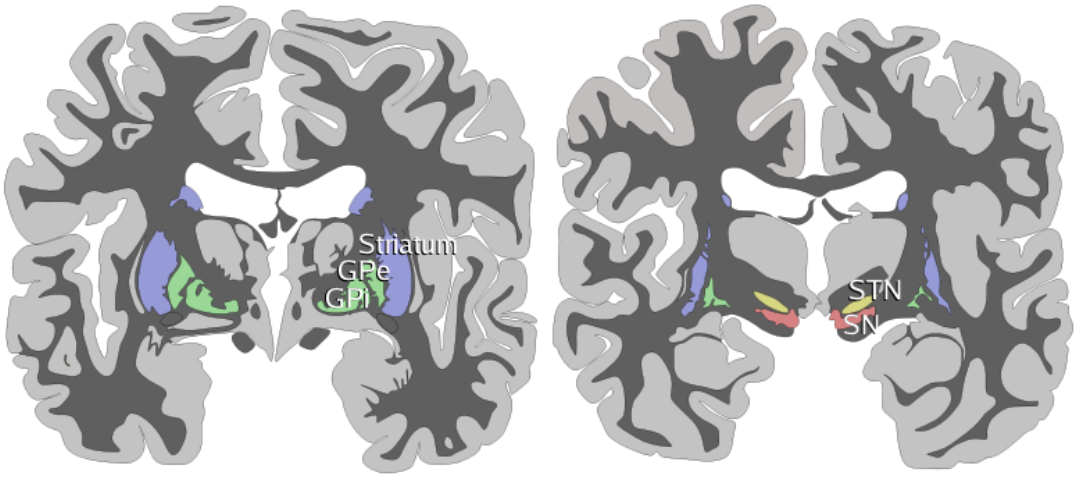
Basal ganglia · direct vs indirect

BASAL GANGLIA: THE STRUCTURES



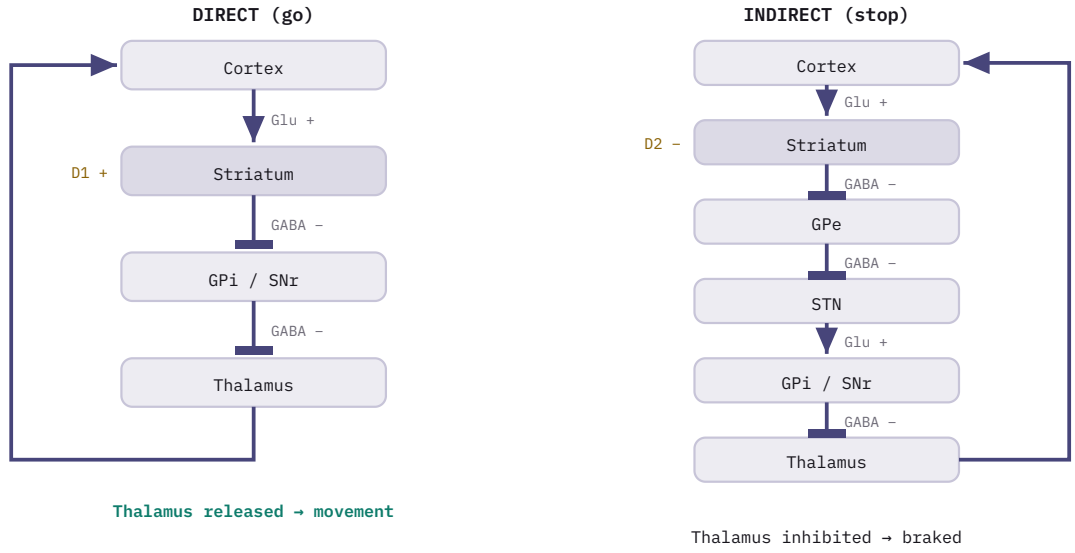
Caudate (head, body, tail) + putamen = striatum; putamen + globus pallidus = lentiform nucleus; with thalamus and nucleus accumbens. Wikimedia Commons, CC BY-SA.

CORONAL CROSS-SECTIONS



Two coronal slices: striatum and globus pallidus (GPe, GPi) anteriorly; subthalamic nucleus (STN) and substantia nigra (SN) posteriorly. Real coronal cross-section.

THE CSTC LOOP: DIRECT VS INDIRECT



Both loops start in cortex and return via thalamus. **Direct** (striatum → GPi/SNr → thalamus, two inhibitory steps) nets to **disinhibition** of thalamus, so movement is facilitated. **Indirect** adds GPe and STN, netting to extra inhibition, so movement is suppressed. Dopamine from the SNc excites D1 (direct) and inhibits D2 (indirect), so both arms favour movement; its loss explains parkinsonism. The hyperdirect cortex → STN route is a fast brake. OCD = hyperactivity of this loop.

All loops share one skeleton: **Cortex → Striatum → Pallidum/SN → Thalamus → Cortex** (cortico-striato-thalamo-cortical).

Feature	Direct pathway	Indirect pathway
Net effect	Facilitates movement	Inhibits movement
Striatal receptor	D1 (excited by DA)	D2 (inhibited by DA)
Route	Striatum → GPi/SNr → thalamus (disinhibited → cortex)	Striatum → GPe → STN → GPi/SNr → more thalamic inhibition

Feature	Direct pathway	Indirect pathway
Dopamine effect	D1 facilitation	D2 disinhibition of GPe

QUICK RULE

Both DA actions **promote movement**. Lose DA (Parkinson's) → underactive direct + overactive indirect → **hypokinesia**. Excess DA → **hyperkinesia** (chorea, dyskinesia). STN is the only excitatory (glutamatergic) node – lesion → hemiballismus.

MNEMONIC

"CPU-SS" · "Direct = Do it, Indirect = Inhibit it"

Caudate, **P**utamen, glob**u**s pallidus, **S**ubthalamic n., **S**ubstantia nigra. Caudate + putamen = striatum (input); putamen + GP = lentiform nucleus.

Reward circuit & HPA axis

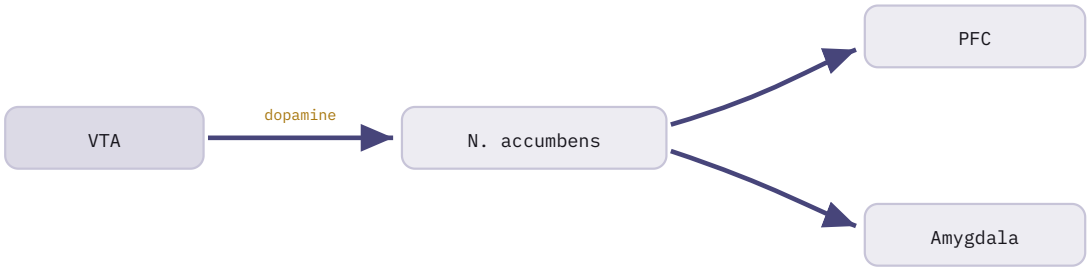
Mesolimbic reward circuit

VTA → nucleus accumbens (ventral striatum) → PFC. All drugs of abuse raise DA in the accumbens, directly or indirectly. The insula codes craving; the limbic system supplies the emotional valence. The accumbens is the DBS target for refractory addiction/OCD.

HPA axis

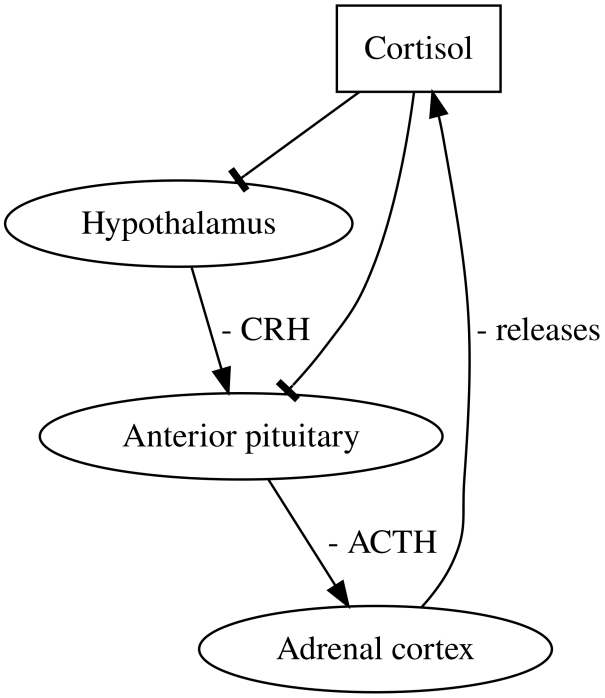
Hypothalamus (PVN) → CRH → anterior pituitary → ACTH → adrenal cortex → cortisol, with negative feedback to both. Melancholic depression: hyperactive axis, ↑ CRH/cortisol, failed dexamethasone suppression. PTSD: paradoxical ↓ cortisol with enhanced feedback.

THE MESOLIMBIC REWARD CIRCUIT



Dopamine from the VTA to the nucleus accumbens is the final common pathway of reward. All addictive drugs raise accumbens dopamine; chronic use blunts it, driving craving.

THE HPA AXIS



Hypothalamus (CRH) → anterior pituitary (ACTH) → adrenal cortex (cortisol). Cortisol feeds back negatively (bars) onto both. Hyperactive in melancholic depression: DST non-suppression. Wikimedia Commons, CC BY-SA.

- **Default mode network** – medial PFC, posterior cingulate / precuneus, angular gyrus. Active at rest and during self-referential thought; deactivates on tasks. Failure to suppress it → rumination in depression; aberrant connectivity in schizophrenia.

6 Neurobiology of psychiatric disorders

This is where examiners separate candidates. For each disorder, give a circuit, a transmitter, and a structural/imaging finding.

Schizophrenia

Hypothesis	Mechanism	Maps to
Dopamine	Mesolimbic hyperactivity	Positive symptoms (hallucinations, delusions)
	Mesocortical hypoactivity (D1 deficit in PFC)	Negative & cognitive symptoms
	Tuberoinfundibular blockade (on treatment)	Hyperprolactinaemia
Glutamate / NMDA	NMDA hypofunction on GABAergic interneurons → disinhibited pyramidal output	Why PCP/ketamine reproduce *both* positive and negative symptoms
GABA	Parvalbumin-interneuron deficit in DLPFC	Disrupted gamma oscillations → cognitive symptoms
Neurodevelopmental	Onset 15–25 (PFC maturation); >100 risk loci; ↓ cortical/hippocampal/MD-thalamus volume	Hypofrontality on imaging; cannabis raises risk 2–6×

● RULE

The glutamate hypothesis **integrates with**, not replaces, the dopamine hypothesis: NMDA hypofunction upstream produces the mesolimbic excess *and* mesocortical deficit. KarXT (xanomeline-trospium, M1/M4 agonist) is the first non-D2-blocking antipsychotic.

Depression

- **Monoamine hypothesis** – deficiency of 5-HT, NE (and DA). The original frame; explains why SSRIs/SNRIs work, but the therapeutic delay points to downstream plasticity, not raw NT levels.
- **HPA axis** – ↑ CRH, ↑ cortisol, DST non-suppression, ↑ adrenal size in melancholic depression. Chronic glucocorticoid excess is neurotoxic to the hippocampus.
- **Neuroplasticity / BDNF** – ↓ BDNF and ↓ hippocampal neurogenesis; antidepressants and ketamine raise BDNF (ketamine via AMPA → mTOR → rapid synaptogenesis).
- **Neuroimaging** – ↓ hippocampal and PFC volume; subgenual ACC (BA 25) hyperactivity (DBS target); ↑ amygdala reactivity to negative stimuli.

Bipolar disorder

- **Core picture** – episodic dysregulation of monoamines (NE/DA excess in mania, deficit in depression), with disturbed intracellular signalling. Lithium acts on the **inositol / GSK-3β** pathways; valproate and other anticonvulsants stabilise via GABA/ion channels.
- **Structure** – amygdala and prefrontal-limbic dysconnectivity; high heritability with genetic overlap with schizophrenia. NE excess underlies the reduced sleep need and hyperarousal of mania.

Anxiety disorders

- **Fear circuit** – **amygdala** is the hub; the "low road" (thalamus → amygdala, fast, crude) and "high road" (thalamus → cortex → amygdala, slow, precise). PFC normally regulates the amygdala; in anxiety/PTSD that top-down control is weak.
- **Transmitters** – GABA deficit (hyperexcitability; BZDs enhance GABA-A), 5-HT1A involvement (SSRIs first-line), and a hyperactive **locus coeruleus** (NE → hyperarousal). PTSD: small hippocampus + small PFC + overactive amygdala.

OCD

- **Circuit** – hyperactivity of the OFC–caudate–thalamic (cortico-striato-thalamo-cortical) loop. Uniquely serotonergic: responds to SSRIs (high dose) and clomipramine. Elevated glutamate in the caudate on MRS. DBS of the ventral capsule/ventral striatum for refractory cases.

Addiction

- **Common pathway** – every drug of abuse raises **dopamine in the nucleus accumbens** via the mesolimbic circuit. Cocaine blocks DAT; amphetamine reverses it; nicotine acts on α4β2 nicotinic receptors; opioids act on mu receptors; alcohol potentiates GABA-A and blocks NMDA.
- **Craving & relapse** – cues recruit the insula (craving) and prefrontal control circuits; chronic use blunts the accumbens DA response → anhedonia and the drive to use more. ~50% of addiction risk is genetic.

Dementias

Dementia	Regional / transmitter signature
Alzheimer's	Cholinergic deficit (NBM degeneration); entorhinal → hippocampal atrophy first. AChE inhibitors + memantine (NMDA antagonist)
Frontotemporal (bvFTD)	OFC + ACC atrophy → disinhibition, apathy, loss of empathy
Lewy body / Parkinson's dementia	Nigrostriatal + cortical DA loss; α-synuclein; fluctuating cognition, visual hallucinations, neuroleptic sensitivity
Vascular	Stepwise; subcortical / strategic infarcts; executive > memory early
Huntington's	Caudate atrophy (GABAergic medium spiny neurons); chorea + psychiatric change

● TRAP

Don't confuse the two hyperthermic emergencies. **Serotonin syndrome**: serotonergic excess, onset in hours, *clonus + hyperreflexia + mydriasis*. **NMS**: dopamine blockade, onset over days, *lead-pipe rigidity + hyporeflexia + ↑↑ CPK*. "SS is Clonus and Go; NMS is Rigid and Slow."

7 High-yield exam questions

◆ **Almost certain** – "Describe the dopaminergic pathways and their applied importance" (asked ~6 times). Answer with the four-pathway table + the antipsychotic dilemma line.

◆ **Very likely** – "Neurochemistry of serotonin and its role in disorders" (synthesis → raphe → receptors → depression/OCD/serotonin syndrome).

● **Reliable perennials** – structure/functions/disorders of the **basal ganglia**; **frontal lobe** syndromes; **draw and explain the Papez circuit**; "role of glutamate and GABA in depression".

● **Define-and-discuss** – "Define neurotransmitters; describe characteristics and types"; receptors relevant to affective disorders; serotonin syndrome (define + management).

QUICK-RECALL · DRILL THESE ONE-LINERS

★ DA pathways: **Mesolimbic → +ve Sx, Mesocortical → –ve/cognitive Sx, Nigrostriatal → EPS, Tuberoinfundibular → prolactin.** (MeMe NiTu)

- Origins: 5-HT = **raphe**, NE = **locus coeruleus**, DA = **VTA / substantia nigra**, ACh = **nucleus basalis of Meynert**, histamine = **tuberomammillary n.**
- Rate-limiting enzymes: **tyrosine hydroxylase** (catecholamines), **tryptophan hydroxylase** (5-HT), **GAD** (GABA, needs B6).
- Metabolites: DA → **HVA**, 5-HT → **5-HIAA**, NE → **MHPG**. Low CSF 5-HIAA = impulsive/violent suicide.
- Ionotropic only: **GABA-A, NMDA, AMPA, kainate, nicotinic, 5-HT3.** All else metabotropic GPCRs.
- D1/D5 = Gs (D1-like); D2/D3/D4 = Gi (D2-like). **D2 occupancy 65–80% = therapeutic; >80% = EPS.**
- BZD ↑ **frequency**, barbiturate ↑ **duration** of Cl– channel opening at GABA-A.
- NMDA needs glutamate + glycine + depolarisation (relieves Mg2+ block) → coincidence detector → LTP.
- Papez: **Hippocampus → fornix → mammillary bodies → ant. thalamus → cingulate → parahippocampal → hippocampus.**
- Direct = D1 = facilitate; indirect = D2 = inhibit. Lose DA → Parkinson's; STN lesion → hemiballismus.
- OCD = **OFC–caudate–thalamic hyperactivity**; addiction = **VTA → nucleus accumbens DA**; HPA = CRH → ACTH → cortisol.
- Depression: monoamine + HPA (DST non-suppression) + ↓BDNF/↓hippocampus + subgenual ACC. Alzheimer's = cholinergic (NBM).

ONE-DAY REVISION · PAPER 1

Neurophysiology

How the neuron holds a charge, fires, and talks to the next cell, and the systems physiology psychiatry leans on every day: arousal, autonomic balance, the stress axis, the barrier and the glia. Hold the ion that moves at each phase, the sequence at the synapse, and the one number attached to each rhythm.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Receptor pharmacology and named transmitters live in the Neurotransmitters pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Action potential ion sequence: rest is held by K+ leak at about −70 mV, threshold −55 mV opens voltage-gated Na+ channels (Na+ in = depolarise to ~+30 mV), then K+ channels open (K+ out = repolarise), and slow K+ closure gives the hyperpolarising undershoot.
2. Ca^{2}+ is the trigger at the chemical synapse: the AP opens voltage-gated Ca^{2}+ channels, Ca^{2}+ entry drives vesicle fusion and transmitter release; the AP itself does not cross the cleft, so no calcium means no release.
3. LTP is NMDA-receptor-dependent: the NMDA channel needs both glutamate and postsynaptic depolarisation to expel its Mg^{2}+ block, letting Ca^{2}+ in to insert more AMPA receptors. Trap: LTP is induced by NMDA but expressed through AMPA, and Mg^{2}+ (not the ligand) gates the channel at rest.
4. Ionotropic = fast ligand-gated ion channel; metabotropic = slow GPCR plus second messenger. Classic trap: 5-HT3 is the one ionotropic serotonin receptor; all other serotonin, dopamine, muscarinic and GABA-B receptors are metabotropic.
5. Autonomic transmitters: all preganglionic neurons (both divisions) release acetylcholine; postganglionic sympathetic is noradrenaline except sweat glands, which are sympathetic but cholinergic, while all postganglionic parasympathetic is acetylcholine.

1 The neuron at rest and in action

Everything starts with a charge held across the membrane. The cell spends energy to keep ions out of balance, then briefly lets them rush across to signal. Get the **ion that moves at each phase** right and the action potential tells itself.

The resting membrane potential

◆ **Resting membrane potential** – about **−70 mV** inside relative to outside. The inside is negative because the membrane at rest is far more permeable to **K+** than to Na+, so K+ leaks out down its gradient and leaves the interior negative.

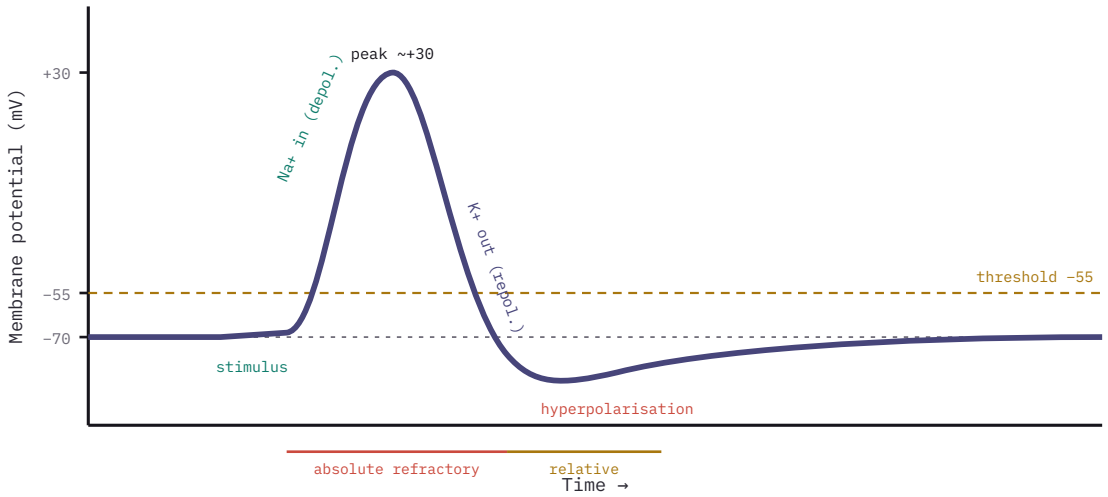
● **Who sets the gradients** – the **Na+/K+-ATPase** pumps **3 Na+ out for 2 K+ in** per ATP, maintaining high K+ inside and high Na+ outside. **Leak (K+) channels** let K+ drift out, setting the resting voltage close to the K+ equilibrium.

- **Nernst idea** – the equilibrium potential is the voltage at which the electrical pull on one ion exactly balances its concentration push. For K+ this is near −90 mV, for Na+ near +60 mV. The Nernst equation gives the equilibrium for a *single* ion.
- **Goldman idea** – the real resting potential is set by *several* ions at once, each weighted by its permeability. The Goldman (Goldman-Hodgkin-Katz) equation blends K+, Na+ and Cl−; because K+ permeability dominates at rest, resting potential sits near (but not exactly at) the K+ equilibrium.

The action potential, phase by phase

Phase	What the channels do	Ion & direction
Resting	voltage-gated channels shut; K+ leak dominates	~−70 mV held
Depolarisation	stimulus reaches **threshold** (~−55 mV); voltage-gated **Na+ channels open**	Na+ in → rapid rise to ~+30/+40 mV
Repolarisation	Na+ channels **inactivate**; voltage-gated **K+ channels open**	K+ out → voltage falls back down
Hyperpolarisation	K+ channels close *slowly*, so K+ over-leaks	undershoot below −70 mV, then the pump restores rest

THE ACTION POTENTIAL



A subthreshold stimulus does nothing; once threshold (−55 mV) is crossed, voltage-gated Na+ channels open and the spike fires all-or-none. Na+ influx drives depolarisation to ~+30 mV; K+ efflux repolarises; slow K+ closure causes the hyperpolarising undershoot before the pump restores rest.

- **All-or-none** – below threshold, nothing; at or above it, a full-size spike every time. Stimulus *strength* is coded by firing **frequency**, not by spike size.

Refractory periods

- **Absolute refractory period** – during depolarisation and most of repolarisation the Na+ channels are **inactivated**; *no* stimulus, however strong, can fire a second spike. This caps maximum firing rate and forces one-way propagation.
- **Relative refractory period** – during the hyperpolarised tail, Na+ channels have reset but the membrane is extra-negative; a **stronger-than-usual** stimulus can fire a spike.

Propagation and myelin

- **Propagation** – each spike depolarises the adjacent membrane to threshold, regenerating itself along the axon without losing amplitude. The absolute refractory zone behind it stops it going backwards.
- **Saltatory conduction** – in myelinated axons the spike *jumps* node to node (nodes of Ranvier), where voltage-gated Na+ channels cluster. Myelin (an insulator) raises speed and saves energy. Conduction velocity rises with **myelination** and **axon diameter**.
- **Who makes myelin** – **oligodendrocytes** in the CNS (one cell myelinates many axons), **Schwann cells** in the PNS (one cell, one internode).

TRAP

Demyelination (e.g. multiple sclerosis) slows or blocks conduction because the naked internode lacks the Na+ channels to regenerate the spike, not because the axon itself is cut. Local anaesthetics work differently: they **block voltage-gated Na+ channels**, so no depolarisation can reach threshold at all.

HOLD THIS

Rest = **K+ leak** (~−70 mV). Depolarise = **Na+ in**. Repolarise = **K+ out**. Undershoot = slow K+ closure. Speed rises with **myelin** and **diameter**; spikes are **all-or-none**.

2 Synaptic transmission

The spike reaches the terminal; now the signal must cross a gap to the next cell. Two designs exist, but the chemical synapse is the one psychiatry cares about. Hold the **sequence**.

Electrical vs chemical synapses

Electrical (gap junction)

Connexon channels couple cytoplasm directly. **Near-instant**, bidirectional, no fatigue. No amplification or sign change. Used where speed and synchrony matter (some interneurons, cardiac and smooth muscle).

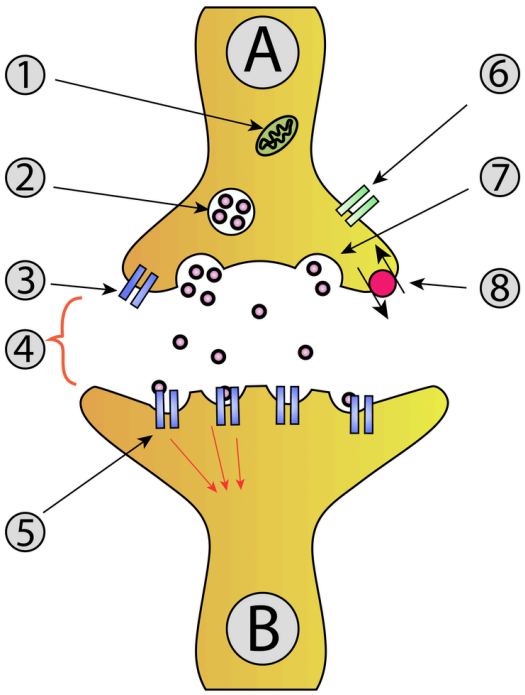
Chemical (the default)

A neurotransmitter crosses the cleft. **~0.5–1 ms synaptic delay**, one-way, fatigable, but allows **amplification, integration and excitation or inhibition**. The plastic, drug-targetable synapse.

The chemical sequence

Step	What happens
1. AP arrives	action potential depolarises the presynaptic terminal
2. Ca^{2}+ entry	voltage-gated **Ca^{2}+ channels** open; Ca^{2}+ floods in (the trigger)
3. Vesicle fusion	Ca^{2}+ drives synaptic vesicles to fuse with the membrane (SNARE proteins)
4. NT release	transmitter is released into the cleft by **exocytosis**
5. Receptor binding	NT binds postsynaptic receptors → ion channels open → a postsynaptic potential
6. Termination	signal ended by **reuptake**, **enzymatic breakdown**, or diffusion

THE CHEMICAL SYNAPSE



Presynaptic terminal A to postsynaptic terminal B. 1 mitochondrion; 2 vesicle of neurotransmitter; 3 autoreceptor; 4 synaptic cleft; 5 postsynaptic receptor; 6 voltage-gated calcium channel; 7 vesicle fusion and release; 8 reuptake transporter. Wikimedia Commons (Mouagip), CC BY-SA 3.0.

● KEY

Ca^{2}+ is the trigger. No calcium entry, no release. This is the single most testable step: the action potential does not itself cross the synapse; it converts an electrical signal into a Ca^{2}+-driven chemical one.

Postsynaptic potentials and summation

● **EPSP** – excitatory postsynaptic potential: a **depolarising** shift (typically Na+ in, e.g. glutamate) that moves the cell *toward* threshold.

▲ **IPSP** – inhibitory postsynaptic potential: a **hyperpolarising** shift (Cl– in or K+ out, e.g. GABA, glycine) that moves the cell *away* from threshold.

● **Temporal summation** – one input firing rapidly in succession; EPSPs pile up *in time* before each decays, reaching threshold.

● **Spatial summation** – many inputs firing together at *different sites*; their EPSPs add *across space*. The axon hillock sums all EPSPs and IPSPs; if the net crosses threshold, a spike fires.

The neuromuscular junction

● **NMJ** – the model chemical synapse. The motor neuron releases **acetylcholine**; ACh binds **nicotinic** receptors on the muscle end-plate, producing an end-plate potential that triggers the muscle action potential.

◆ **Termination at the NMJ** – ACh is broken down by **acetylcholinesterase** in the cleft (an example of enzymatic termination). Myasthenia gravis = antibodies against the nicotinic ACh receptor; reversed clinically by anticholinesterases.

Clearing the transmitter

- **Reuptake** – transporters pull transmitter back into the terminal (or glia) for reuse. The serotonin and noradrenaline transporters are the direct targets of SSRIs and SNRIs (cross-ref the Neurotransmitters pack).
- **Enzymatic breakdown** – enzymes degrade transmitter in or near the cleft: acetylcholinesterase for ACh; **MAO** and **COMT** for monoamines. MAOIs and COMT inhibitors act here.

3 Receptors and signalling

Two receptor families, distinguished by *speed* and *mechanism*. This is the brief version; the named receptors and their drugs sit in the Neurotransmitters pack.

	Ionotropic	Metabotropic
What it is	a **ligand-gated ion channel** (receptor and pore in one)	a **G-protein-coupled receptor** (GPCR), no pore of its own
Speed	fast (milliseconds)	slow (~100 ms to seconds, even minutes)
Action	opens a channel directly → immediate EPSP/IPSP	activates a **second messenger** cascade → modulates the cell
Examples	nicotinic ACh, GABA-A, AMPA, NMDA, glycine, 5-HT3	muscarinic ACh, GABA-B, most dopamine, noradrenaline, serotonin (bar 5-HT3) receptors

- **Second-messenger idea** – the transmitter (first messenger) binds a GPCR, which activates a G-protein, which changes an enzyme to make an intracellular **second messenger** (cyclic AMP, IP3, diacylglycerol, Ca^{2}+). The cascade **amplifies** the signal and can alter ion channels, enzymes and gene transcription. Lithium acts on this layer (inositol depletion and second-messenger signalling).

● RULE

Fast, point-to-point signalling is **ionotropic**. Slow, lasting *modulation* (mood, arousal, plasticity, most psychotropic targets) runs through **metabotropic** GPCRs and second messengers. One exception to memorise: 5-HT3 is the one ionotropic serotonin receptor.

4 Neuroplasticity & the cell biology of learning

Synapses are not fixed; their strength changes with use. This is the cellular substrate of learning and memory, and the most heavily tested basic-science link to clinical psychiatry.

- **Long-term potentiation (LTP)** – a lasting *strengthening* of a synapse after high-frequency stimulation. Classically **NMDA-receptor-dependent** in the hippocampus: the NMDA channel needs *both* glutamate *and* postsynaptic depolarisation to expel its **Mg^{2}+ block**, letting Ca^{2}+ in. That Ca^{2}+ signal drives more AMPA receptors into the membrane, making the synapse stronger.
- **NMDA as a coincidence detector** – because it opens only when pre- and postsynaptic activity coincide, the NMDA receptor reads "these two fired together," the molecular form of associative learning.
- **Long-term depression (LTD)** – the mirror image: low-frequency stimulation *weakens* a synapse (AMPA receptors removed). LTP and LTD together let circuits both add and prune strength.
- **Hebbian plasticity** – Hebb's rule: **"cells that fire together, wire together."** Coincident pre- and postsynaptic firing strengthens their connection. LTP is the cellular proof of the rule.

- **Synaptic pruning** – the brain overproduces synapses early, then eliminates the unused ones (microglia help). Peaks in childhood and again in **adolescence**, refining circuits; disordered pruning is one model of schizophrenia onset.
- **Adult neurogenesis** – new neurons are still born in adulthood, chiefly in the **hippocampal dentate gyrus** (and the subventricular zone). Stress and depression suppress it; antidepressants and exercise promote it, a leading hypothesis for the delayed onset of antidepressant action.

MNEMONIC

"Fire together, wire together"

Hebb's rule, realised by NMDA-dependent LTP. The NMDA receptor opens only on coincident activity, so it is the synapse's coincidence detector and the entry point for associative memory.

TRAP

LTP is **induced** by the NMDA receptor but **expressed** largely through AMPA receptors (more AMPA inserted, larger response). The NMDA receptor is the switch; AMPA carries the strengthened current. Magnesium, not the ligand, is what gates the NMDA channel at rest.

5 Systems physiology psychiatry leans on

Arousal: the ascending reticular activating system

- **ARAS** – the ascending reticular activating system: a brainstem network projecting up through thalamus and basal forebrain to the cortex. It sets **wakefulness, arousal and the sleep-wake cycle**; damage produces coma. It is the "volume knob" for cortical activation, using ACh, noradrenaline, serotonin, histamine and orexin.

The autonomic nervous system

	Sympathetic	Parasympathetic
Theme	fight-or-flight (catabolic, mobilise)	rest-and-digest (anabolic, conserve)
Outflow	thoracolumbar (T1–L2)	craniosacral (cranial nerves III, VII, IX, X; S2–S4)
Heart / pupils	heart rate up, pupils dilate	heart rate down, pupils constrict
Gut / bladder	digestion slows; sphincters contract	digestion and emptying resume
Postganglionic NT	**noradrenaline** (except sweat glands, which are cholinergic)	**acetylcholine** (muscarinic)

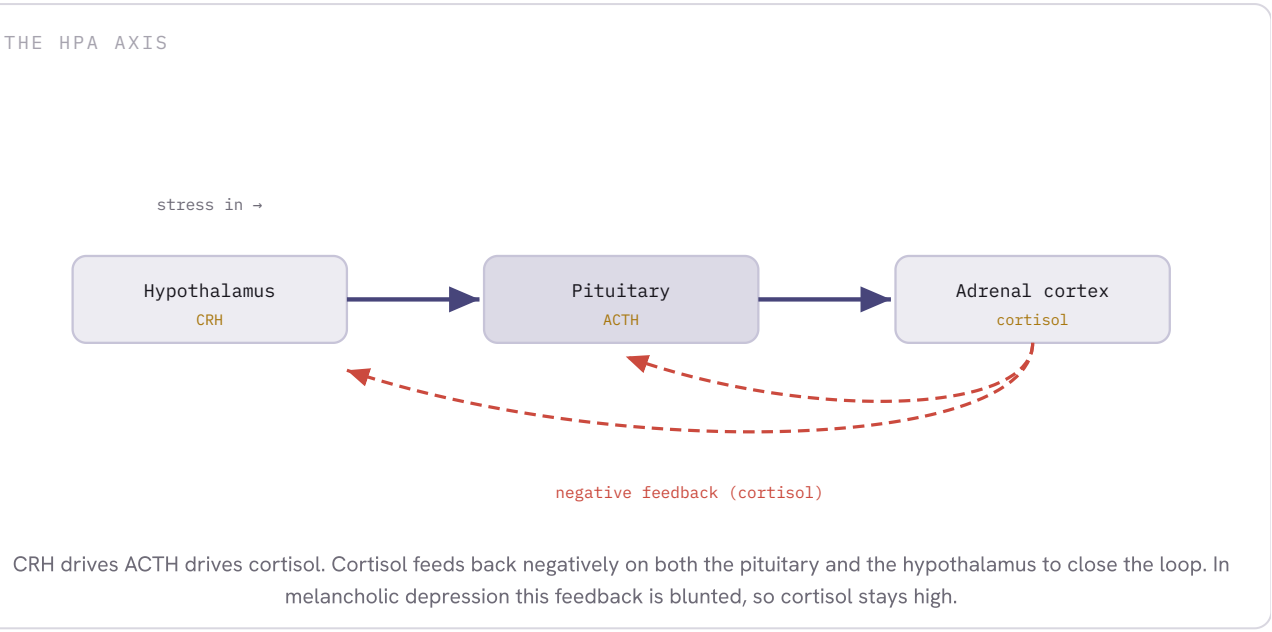
- **Fight-or-flight** – the acute sympathetic response: adrenal medulla releases **adrenaline and noradrenaline**, giving tachycardia, sweating, dilated pupils, bronchodilation and shunting of blood to muscle. This is the bodily core of the anxiety and panic presentation.

TRAP

All **preganglionic** autonomic neurons (both divisions) release **acetylcholine**. The divisions diverge only at the **postganglionic** step: sympathetic = mostly noradrenaline, parasympathetic = acetylcholine. The sweat-gland exception (sympathetic but cholinergic) is a favourite.

The HPA axis and the stress response

- **HPA cascade** – hypothalamus releases **CRH** → anterior pituitary releases **ACTH** → adrenal *cortex* releases **cortisol**. Cortisol then feeds back to *switch off* CRH and ACTH (**negative feedback**), normally keeping the axis self-limiting.



CRH drives ACTH drives cortisol. Cortisol feeds back negatively on both the pituitary and the hypothalamus to close the loop. In melancholic depression this feedback is blunted, so cortisol stays high.

▲ **Dexamethasone non-suppression** – dexamethasone (a synthetic steroid) normally suppresses morning cortisol via that negative feedback. In some severe (melancholic) depression the feedback is impaired, so cortisol fails to suppress: the **dexamethasone suppression test** is positive. Sensitive but not specific; a historical biological marker, not a routine diagnostic (cross-ref the Investigations pack).

The blood-brain barrier, CSF and glia

- **Blood-brain barrier** – tight junctions between capillary endothelial cells, supported by **astrocyte** end-feet, that keep most large or polar molecules out of the brain. Lipophilic, small molecules cross easily, the rule that decides which psychotropics reach the CNS.
- **CSF** – cerebrospinal fluid, made by the **choroid plexus**, circulating lateral → third → fourth ventricle → subarachnoid space, cushioning the brain and clearing waste.

Glial cell	Job
Astrocytes	support the BBB, buffer K+ and transmitter (glutamate uptake), supply metabolic support
Oligodendrocytes	make CNS **myelin** (Schwann cells do this in the PNS)
Microglia	resident **immune** cells; phagocytosis, synaptic pruning, neuroinflammation

6 Electrophysiology basics

The EEG records summed cortical postsynaptic potentials at the scalp. Rhythms are read by **frequency band**, and each band indexes a brain state. Hold the band order and the one state attached to each.

Rhythm	Frequency	State it indexes
Delta (δ)	< 4 Hz	deep (slow-wave) sleep, stages N3; largest, slowest
Theta (θ)	4–8 Hz	drowsiness, light sleep (N1), memory, children
Alpha (α)	8–13 Hz	relaxed wakefulness, eyes closed (occipital); blocked by opening the eyes
Beta (β)	13–30 Hz	alert, eyes-open, active thinking, anxiety; the normal awake EEG
Gamma (γ)	> 30 Hz	focused attention, feature binding, high-level processing

MNEMONIC

"Delta-Theta-Alpha-Beta-Gamma" = slow to fast, asleep to alert

As frequency rises (delta → gamma) the brain moves from deep sleep toward focused wakefulness. Amplitude falls as frequency rises. Alpha appears with eyes closed and disappears (alpha block) when the eyes open.

TRAP

Do not confuse the EEG bands with the sleep *stages*: delta waves *define* N3 (slow-wave) sleep, but REM sleep shows a low-amplitude, mixed-frequency ("awake-like") trace, which is why REM is called **paradoxical** sleep (cross-ref the Sleep pack).

- **Evoked potentials** – the averaged EEG response time-locked to a stimulus (visual, auditory, somatosensory). They test the integrity of a sensory pathway; event-related potentials such as the **P300** index cognitive processing (cross-ref the Investigations pack).

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Resting potential:** ~–70 mV, set by **K+ leak**; gradients held by the Na+/K+-ATPase (3 out, 2 in). Nernst = one ion; Goldman = several ions weighted by permeability.
- **Action potential:** threshold ~–55 mV → **Na+ in** (depolarise) → **K+ out** (repolarise) → slow K+ closure (hyperpolarising undershoot). All-or-none; strength coded by frequency.
- **Refractory:** absolute = Na+ channels inactivated, no spike possible; relative = reset but needs a stronger stimulus. Forces one-way, rate-limited firing.
- **Conduction:** saltatory, node to node; faster with **myelin** and **diameter**. Oligodendrocytes (CNS) vs Schwann cells (PNS).
- ★ **Synapse:** AP → voltage-gated **Ca^{2}+** in → vesicle fusion → NT release → receptor binding → reuptake / breakdown. **Ca^{2}+ is the trigger.**
- **PSPs:** EPSP = depolarising (glutamate); IPSP = hyperpolarising (GABA/glycine). Temporal = same input over time; spatial = many inputs at once. NMJ = ACh on nicotinic, cleared by acetylcholinesterase.
- **Receptors:** ionotropic = fast ligand-gated channel; metabotropic = slow GPCR + second messenger. 5-HT3 is the odd ionotropic serotonin receptor.
- **Plasticity:** LTP = NMDA-dependent strengthening (Mg^{2}+ block expelled, Ca^{2}+ in, more AMPA); LTD = weakening. Hebb: fire together, wire together. Pruning peaks in adolescence; adult neurogenesis in the hippocampal dentate gyrus.
- **Systems:** ARAS = arousal/wakefulness. Sympathetic = fight-or-flight (postganglionic noradrenaline); parasympathetic = rest-and-digest (ACh). All preganglionic = ACh.
- **HPA:** CRH → ACTH → cortisol, with cortisol negative feedback. Dexamethasone non-suppression in melancholic depression. BBB = tight junctions + astrocyte feet. Glia: astrocytes (support/BBB), oligodendrocytes (myelin), microglia (immune/pruning).
- **EEG:** delta <4 (deep sleep) · theta 4–8 (drowsy) · alpha 8–13 (relaxed, eyes closed) · beta 13–30 (alert) · gamma >30 (focused). Slow-to-fast = asleep-to-alert; REM is paradoxical.

ONE-DAY REVISION · PAPER 1

Neurotransmitters & receptors

The pharmacology layer: what each transmitter is built from, the enzyme that limits its making, what it breaks down into, the receptors it speaks through, and why every one of those facts is a drug target. Get the synthesis chains and the receptor families clean and most of psychopharmacology falls into place.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The dopamine, serotonin and noradrenaline tract anatomy lives in the companion Neuroanatomy pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The rate-limiting enzyme is always the hydroxylase: tyrosine hydroxylase for dopamine and noradrenaline (NA is made from dopamine, so they share it), tryptophan hydroxylase for serotonin; the decarboxylase is never the bottleneck.
2. D2 blockade is therapeutic in the mesolimbic tract (less positive symptoms) but causes the side-effects elsewhere: nigrostriatal → EPS and tardive dyskinesia, tuberoinfundibular → hyperprolactinaemia (dopamine is the brake on prolactin, so blockade raises it), mesocortical → worse negative and cognitive symptoms.
3. Antipsychotic potency tracks D2 affinity; D2-like (D2, D3, D4) couple to Gi (cAMP down) and D1-like (D1, D5) to Gs (cAMP up); clozapine is the exception (low D2, high 5-HT2A / D4).
4. At the GABA-A chloride channel, benzodiazepines increase the frequency of opening while barbiturates increase the duration and can open it without GABA at high dose, which is why barbiturate overdose is far more lethal (no ceiling).
5. 5-HT3 is the trap: it is the only serotonin receptor that is ionotropic (a ligand-gated cation channel, ondansetron antagonist); every other 5-HT receptor is a metabotropic GPCR.

1 The monoamines at a glance

Three of the most examined transmitters share one logic: an amino-acid precursor, a **rate-limiting enzyme** (the step that sets the pace), a recognisable metabolite (often the CSF/urine marker), and a receptor family. Learn this table as a unit and every later section just expands a row.

MONOAMINE ORIGINS

	SYNTHESIS				
Transmitter	**Precursor**	**Rate-limiting enzyme**	**Main metabolite**	**Main receptors**	**Psychiatric relevance**
Dopamine (DA)	tyrosine	tyrosine hydroxylase	HVA (via MAO + COMT)	D1-like (D1, D5); D2-like (D2, D3, D4)	psychosis, antipsychotic blockade, EPS, reward/addiction, Parkinsonism
Noradrenaline (NA)	tyrosine → dopamine	tyrosine hydroxylase	MHPG (central), VMA (peripheral)	α1, α2, β	arousal, anxiety, depression, ADHD; locus coeruleus

	SYNTHESIS				
Transmitter	**Precursor**	**Rate-limiting enzyme**	**Main metabolite**	**Main receptors**	**Psychiatric relevance**
Serotonin (5-HT)	tryptophan	tryptophan hydroxylase	5-HIAA	5-HT1A, 5-HT2A, 5-HT2C, 5-HT3 (+ many more)	depression, anxiety, OCD, suicide (low CSF 5-HIAA), antidepressant target

RULE

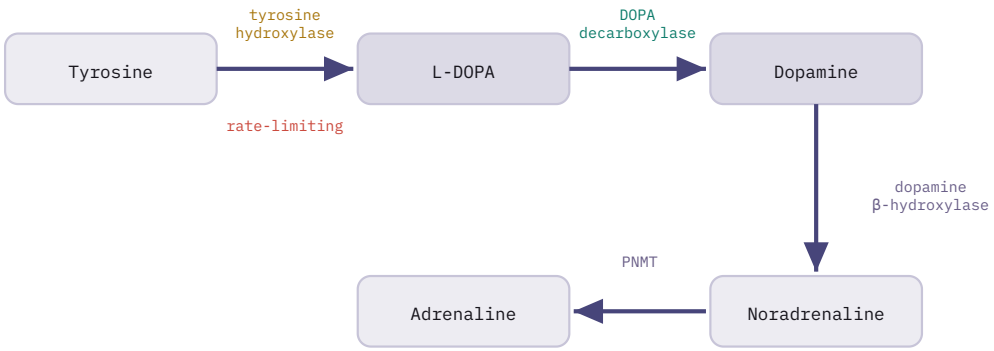
Every monoamine has its rate-limiting **hydroxylase**. For dopamine and noradrenaline the limiting enzyme is the same one, **tyrosine hydroxylase**, because NA is made *from* dopamine. For serotonin it is **tryptophan hydroxylase**. The hydroxylase, not the decarboxylase, is the bottleneck.

2 Dopamine

Synthesis

Two steps from the amino acid. The first enzyme is rate-limiting; the second is the decarboxylase shared with serotonin.

CATECHOLAMINE SYNTHESIS PATHWAY



Tyrosine → L-DOPA (tyrosine hydroxylase, the rate-limiting step) → dopamine (DOPA decarboxylase). Dopamine → noradrenaline (dopamine β-hydroxylase) → adrenaline (PNMT). The whole catecholamine line runs off one starting amino acid.

◆ **Rate-limiting step** – **tyrosine hydroxylase** converts tyrosine to L-DOPA. It is the pace-setter for the entire catecholamine pathway, which is why L-DOPA (skipping that step) treats Parkinson's where dopamine itself cannot cross the blood-brain barrier.

● **DOPA decarboxylase** – (aromatic L-amino-acid decarboxylase) converts L-DOPA to dopamine. The same enzyme converts 5-HTP to serotonin. Peripheral inhibitors (carbidopa) stop premature conversion so more L-DOPA reaches the brain.

Metabolism

● **Breakdown** – dopamine is degraded by **monoamine oxidase (MAO)** and **catechol-O-methyltransferase (COMT)**, both feeding into the final metabolite **homovanillic acid (HVA)**. MAO-B inhibitors (selegiline) and COMT inhibitors (entacapone) raise dopamine in Parkinson's.

Receptor families

All dopamine receptors are metabotropic (GPCRs). They split into two families by the G-protein they couple to.

Family	Members	G-protein / second messenger	Effect on cAMP
D1-like	D1, D5	Gs → activates adenylyl cyclase	cAMP up
D2-like	D2, D3, D4	Gi → inhibits adenylyl cyclase	cAMP down

HOLD THIS

The **D2 receptor** is the one antipsychotics block. Antipsychotic potency tracks D2 affinity. Clozapine is the exception (low D2, high 5-HT2A / D4). D2-like = Gi (cAMP down); D1-like = Gs (cAMP up).

The four pathways and what blockade does

D2 blockade is therapeutic in one tract and the source of side-effects in the others. This table is the reason antipsychotics both work and harm.

THE FOUR DOPAMINE PATHWAYS

Pathway	Normal role	Effect of D2 blockade
Mesolimbic	reward, salience; overactive in psychosis	reduces positive symptoms (the therapeutic effect)
Mesocortical	cognition, motivation (to prefrontal cortex)	worsens negative & cognitive symptoms
Nigrostriatal	motor control (basal ganglia)	extrapyramidal symptoms (EPS), parkinsonism, tardive dyskinesia
Tuberoinfundibular	dopamine tonically inhibits prolactin release	hyperprolactinaemia (galactorrhoea, amenorrhoea, sexual dysfunction)

● CLASSIC TRAP

In the **tuberoinfundibular** pathway dopamine is the brake on prolactin. Block D2 and you release the brake, so prolactin *rises*. This is why antipsychotics (especially risperidone, amisulpride and typicals) cause hyperprolactinaemia, while aripiprazole (a partial agonist) and dopamine agonists lower it.

3 Noradrenaline

Synthesis & metabolism

- **From dopamine** – inside the vesicle, **dopamine β-hydroxylase** converts dopamine to noradrenaline. In the adrenal medulla a further enzyme, **PNMT** (phenylethanolamine N-methyltransferase), converts noradrenaline to adrenaline.
- **Breakdown** – like dopamine, by MAO and COMT. Central metabolite is **MHPG** (3-methoxy-4-hydroxyphenylglycol); the main peripheral metabolite is **VMA** (vanillylmandelic acid), elevated in phaeochromocytoma.

Receptors & source nucleus

Receptor	Type	Note
α1	Gq metabotropic	post-synaptic, vasoconstriction; blockade → postural hypotension, sedation

Receptor	Type	Note
α2	Gi metabotropic	presynaptic autoreceptor: feedback brake on NA release. Clonidine agonist; mirtazapine antagonist (boosts release)
β (β1, β2)	Gs metabotropic	β1 cardiac; propranolol blockade used in performance anxiety, akathisia, tremor

- **Locus coeruleus** – the pontine nucleus that is the brain's main source of noradrenaline. Drives arousal, vigilance and the stress response; hyperactive in anxiety and panic, and the target of α2 agonists in withdrawal states.

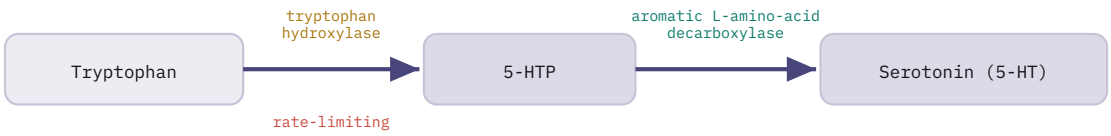
TRAP

The **α2 autoreceptor** is a negative-feedback switch: stimulating it (clonidine) *reduces* noradrenaline output, blocking it (the noradrenergic action of mirtazapine) *increases* release. Same receptor, opposite drugs, opposite direction.

4 Serotonin (5-HT)

Synthesis & metabolism

SEROTONIN SYNTHESIS PATHWAY



Tryptophan → 5-HTP (tryptophan hydroxylase, the rate-limiting step) → 5-HT (the same decarboxylase that makes dopamine). Serotonin is degraded by MAO to 5-HIAA.

- **Rate-limiting step** – **tryptophan hydroxylase** converts tryptophan to 5-HTP. Serotonin synthesis depends on dietary tryptophan, so acute tryptophan depletion lowers brain 5-HT (a research probe in depression).
- **Metabolite** – **MAO** degrades 5-HT to **5-HIAA** (5-hydroxyindoleacetic acid). Low CSF 5-HIAA is associated with impulsive aggression and suicide.

Key receptors (one functional note each)

One ionotropic receptor (5-HT3); the rest are metabotropic GPCRs. The exam favours these four.

Receptor	Type	One thing to remember
5-HT1A	Gi metabotropic	somatodendritic autoreceptor; partial agonist **buspirone** (anxiolytic); anxiolytic / antidepressant
5-HT2A	Gq metabotropic	antagonism = atypical antipsychotic action; agonism = hallucinogens (LSD)
5-HT2C	Gq metabotropic	antagonism drives appetite / weight gain (mirtazapine, olanzapine)
5-HT3	ionotropic (ligand-gated cation channel)	the one 5-HT channel; mediates nausea; ondansetron is the antagonist

- **Raphe nuclei** – the midline brainstem nuclei that are the brain's main source of serotonin, projecting widely. The serotonergic counterpart to the noradrenergic locus coeruleus.

● TRAP

5-HT3 is the odd one out: it is the only serotonin receptor that is a *ligand-gated ion channel* (ionotropic). Every other 5-HT receptor is a metabotropic GPCR. That is why 5-HT3 antagonists (setrons) act fast as anti-emetics.

5 Amino-acid & cholinergic transmitters

GABA, the main inhibitory transmitter

● **Synthesis** – GABA is made **from glutamate** by the enzyme **glutamic acid decarboxylase (GAD)**, which needs vitamin B6 (pyridoxine) as cofactor. So the brain's main inhibitory and main excitatory transmitters are one enzyme step apart.

Receptor	Type	Mechanism
GABA-A	ionotropic	ligand-gated **chloride channel**; opening hyperpolarises → inhibition
GABA-B	metabotropic (Gi)	slow inhibition via K+/Ca2+; baclofen is the agonist

◆ **GABA-A modulatory sites** – the chloride channel has distinct allosteric sites for **benzodiazepines** (increase frequency of channel opening), **barbiturates** (increase duration of opening), **alcohol**, and **neurosteroids** (such as allopregnanolone; the basis of brexanolone in postpartum depression). All enhance GABA-mediated chloride flux.

● CLASSIC TRAP

Benzodiazepines increase the **frequency** of chloride channel opening; barbiturates increase the **duration**. Barbiturates can open the channel even without GABA at high doses, which is why their overdose is far more lethal (no ceiling effect), whereas benzodiazepine overdose alone rarely kills.

Glutamate, the main excitatory transmitter

Receptor	Type	Note
NMDA	ionotropic (Ca2+)	voltage- and ligand-gated, Mg2+ block; central to LTP / learning; ketamine & PCP are antagonists
AMPA	ionotropic (Na+)	fast excitatory transmission
Kainate	ionotropic	modulatory, presynaptic roles
mGluR	metabotropic	slow modulatory glutamate signalling

▲ **Excitotoxicity** – excess glutamate over-activates NMDA receptors, flooding the cell with calcium and killing it. Implicated in stroke, head injury and neurodegeneration; memantine (a weak NMDA antagonist) is used in Alzheimer's.

◆ **NMDA-hypofunction hypothesis** – **under**-activity of NMDA receptors may underlie schizophrenia. Evidence: NMDA antagonists (ketamine, PCP) reproduce positive *and* negative symptoms in healthy people, unlike pure dopamine agonists. Complements the dopamine hypothesis rather than replacing it.

Acetylcholine

● **Synthesis & breakdown** – made from choline + acetyl-CoA by **choline acetyltransferase (ChAT)**; broken down in the synapse by **acetylcholinesterase (AChE)**. Cholinesterase inhibitors (donepezil, rivastigmine, galantamine) raise synaptic ACh in dementia.

Receptor	Type	Note
Nicotinic	ionotropic (cation channel)	fast; neuromuscular junction, autonomic ganglia, CNS
Muscarinic (M1–M5)	metabotropic (GPCR)	blockade → anticholinergic effects (dry mouth, constipation, urinary retention, blurred vision, confusion)

- **Relevance** – the **cholinergic deficit** (loss of nucleus basalis of Meynert neurons) underlies the cognitive decline of **Alzheimer's**. The flip side is iatrogenic: muscarinic blockade by tricyclics, low-potency antipsychotics and anticholinergics causes the anticholinergic burden and can precipitate **delirium** in the elderly.

Histamine & glycine (brief)

- **Histamine (H1)** – central H1 blockade explains the **sedation and weight gain** of many antipsychotics and antidepressants (mirtazapine, olanzapine, quetiapine, low-potency typicals).
- **Glycine** – an inhibitory transmitter (like GABA) acting on glycine receptors, chiefly in the brainstem and spinal cord; also a co-agonist required at the NMDA receptor.

6 Receptor pharmacology essentials

Two structural classes of receptor

	Ionotropic	Metabotropic
What it is	ligand-gated ion channel	G-protein-coupled receptor (GPCR)
Speed	fast (milliseconds)	slow (seconds, via second messengers)
Examples	GABA-A, nicotinic, NMDA, AMPA, kainate, 5-HT3	all DA, all NA (α/β), most 5-HT, muscarinic, GABA-B, mGluR

- **Second messengers (GPCRs)** – **Gs** raises cAMP (D1-like, β-adrenergic); **Gi** lowers cAMP (D2-like, α2, 5-HT1A); **Gq** activates phospholipase C → IP3 / DAG → calcium release (α1, 5-HT2, M1/M3/M5).

MNEMONIC

"Channels are fast, GPCRs are slow"

Ionotropic = ligand-gated channel = milliseconds. Metabotropic = GPCR = a second-messenger cascade = slower but amplifying. 5-HT3 is the trap: a serotonin *channel* among GPCRs.

How a drug acts at a receptor

Term	What it does at the receptor
Agonist	binds and activates fully (full intrinsic activity), mimicking the natural ligand
Antagonist	binds but does not activate; blocks the agonist (zero intrinsic activity)
Partial agonist	binds and activates *submaximally*; acts as agonist in a low-tone state, antagonist in a high-tone state
Inverse agonist	binds and produces the *opposite* effect to the agonist (reduces baseline / constitutive activity)

- **Aripiprazole** – the textbook **D2 partial agonist** (a "dopamine stabiliser"). Where dopamine is high (mesolimbic) it behaves as a functional antagonist and damps psychosis; where dopamine is low (tuberoinfundibular, mesocortical) it provides some tone, so it carries less risk of hyperprolactinaemia and EPS.

● RULE

A **partial agonist** sets a ceiling: it never produces the full maximal response even at saturating doses, and in the presence of a full agonist it actually *lowers* the net effect (competitive partial antagonism). This is the mechanism behind aripiprazole in psychosis and buprenorphine in opioid dependence.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Monoamines:** DA & NA from tyrosine (rate-limiting = tyrosine hydroxylase); 5-HT from tryptophan (rate-limiting = tryptophan hydroxylase). The hydroxylase is always the bottleneck.

- **Dopamine synthesis:** tyrosine → L-DOPA (tyrosine hydroxylase) → dopamine (DOPA decarboxylase). Metabolised by MAO + COMT → HVA.
- **DA receptors:** D1-like (D1, D5) = Gs, cAMP up; D2-like (D2, D3, D4) = Gi, cAMP down. Antipsychotics block D2.
- **DA pathways & D2 blockade:** mesolimbic → less positive symptoms (therapeutic); mesocortical → worse negative/cognitive; nigrostriatal → EPS; tuberoinfundibular → high prolactin.
- **Noradrenaline:** dopamine → NA (dopamine β-hydroxylase) → adrenaline (PNMT). Metabolites MHPG (central), VMA (peripheral). Source = locus coeruleus. α2 = autoreceptor (brake).
- **Serotonin:** tryptophan → 5-HTP (tryptophan hydroxylase) → 5-HT; metabolite 5-HIAA (low in suicide). Source = raphe nuclei.
- **5-HT receptors:** 5-HT1A (autoreceptor, buspirone); 5-HT2A (antipsychotic / hallucinogen target); 5-HT2C (weight gain); 5-HT3 = the only ionotropic one (ondansetron).
- **GABA:** made from glutamate via GAD (needs B6). GABA-A = ionotropic chloride channel with sites for benzodiazepines (frequency), barbiturates (duration), alcohol, neurosteroids. GABA-B = metabotropic (baclofen).
- **Glutamate:** NMDA, AMPA, kainate (ionotropic) + mGluR. Excitotoxicity via NMDA-calcium. NMDA-hypofunction hypothesis of schizophrenia (ketamine/PCP).
- **Acetylcholine:** ChAT makes it, AChE breaks it. Nicotinic = ionotropic; muscarinic = metabotropic (blockade = anticholinergic effects). Cholinergic deficit in Alzheimer's.
- **Histamine H1:** blockade = sedation + weight gain. Glycine = inhibitory + NMDA co-agonist.
- **Receptor classes:** ionotropic = fast channel; metabotropic = slow GPCR (Gs up cAMP, Gi down cAMP, Gq via PLC/IP3/Ca2+).
- **Drug actions:** agonist (full), antagonist (block), partial agonist (submaximal, stabiliser), inverse agonist (opposite). Aripiprazole = D2 partial agonist.

ONE-DAY REVISION · PAPER 1

Genetics in psychiatry

The major psychiatric disorders are polygenic and multifactorial: many small-effect variants stacked on a liability scale, plus environment. Hold the method that delivered each number (family, twin, adoption, GWAS), the approximate heritability for each disorder, and the one finding attached to each name. Every comparison here is a short-note answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. All heritability & concordance figures are approximate textbook ranges – they vary by study and population.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The common psychiatric disorders are **polygenic and multifactorial** (many small-effect variants plus environment), modelled as a continuous **liability with a threshold**, never a single gene; this is the single most testable opening line of the topic.
2. **Penetrance vs expressivity**: penetrance is *whether* the gene shows (proportion of carriers, so a reduced-penetrance trait can skip a generation); expressivity is *how much / what form* it shows in an affected person (variable expressivity never skips, just looks different each time).
3. Heritability is the proportion of *phenotypic variance in a population* that is genetic, **not** an individual's fixed risk; **Falconer**: $h^2 \approx 2 \times (r_{MZ} - r_{DZ})$.
4. Heritability rank order: schizophrenia **~80%**, bipolar **~80-85%**, autism **~80%**, ADHD **~75%** → alcohol dependence **~50%** → major depression **~35-40%** (the lowest of the common adult disorders); schizophrenia is ~80% heritable yet a first-degree relative's risk is only ~10%, because the two numbers answer different questions.
5. Genomic imprinting exemplar, a single 15q11-13 deletion: lose the **pat**ernal copy → **P**rader-Willi; lose the **m**aternal copy → **A**ngelman.

1 Foundations: how traits are inherited

One question organises the whole topic: is the trait carried by **one gene** or by **many**. Almost every psychiatric disorder is the second kind. Get this distinction right and the methods and numbers fall into place.

Pattern	Genetic basis	Distribution	Psychiatric examples
Mendelian (single-gene)	one major gene; autosomal dominant, recessive or X-linked	discrete: affected vs unaffected, classic pedigree ratios	Huntington's disease, fragile X, untreated PKU; *rare* in psychiatry
Polygenic	many genes, each of **small effect**, additive	continuous (height-like) liability	the genetic side of nearly all major disorders
Multifactorial	polygenic + **environment** interacting	continuous liability with a threshold	schizophrenia, bipolar, depression, autism, ADHD

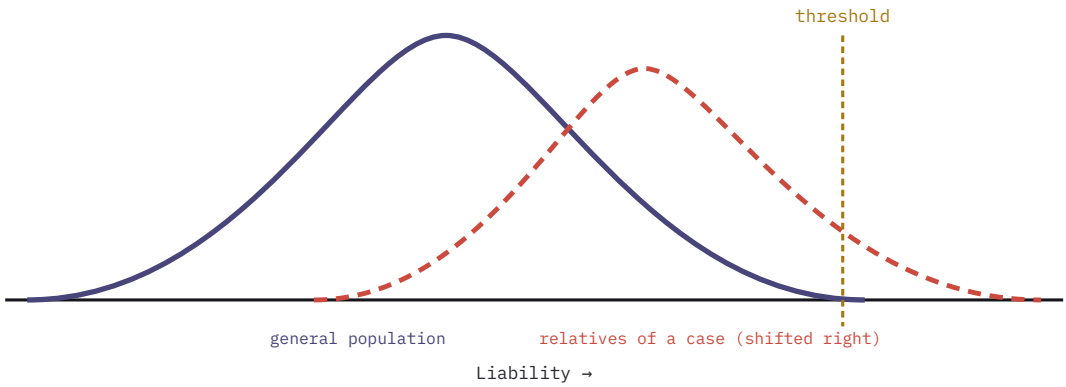
HOLD THIS

The common psychiatric disorders are **polygenic and multifactorial**: many small-effect variants plus environment, not a single gene. This is the single most testable opening line of the topic.

The threshold / liability model

- **Liability** – an unobserved, continuous, normally distributed trait pooling all genetic and environmental risk. Everyone sits somewhere on it.
- **Threshold** – the disorder appears only once liability crosses a critical point. This is how a *continuous* liability produces an *all-or-none* diagnosis. Relatives of an affected person are shifted toward the threshold, so more of them cross it.

THE THRESHOLD / LIABILITY MODEL



Liability is continuous and normally distributed. Relatives of an affected person have a higher mean liability (curve shifted right), so a larger fraction crosses the threshold and is affected.

Penetrance, expressivity, anticipation

- **Penetrance** – the *proportion* of genotype carriers who show the phenotype at all. **Reduced (incomplete) penetrance** means some carriers are unaffected, so a trait can skip a generation. A yes/no concept.
- **Expressivity** – how *severely* or in what *form* the phenotype shows among those who are affected. Variable expressivity = same genotype, different presentation. A how-much concept.
- **Anticipation** – a disorder appearing **earlier and more severely** in successive generations. Genuine mechanism: **trinucleotide repeat expansion** (e.g. Huntington's, fragile X). Reported in bipolar and schizophrenia pedigrees, but there the claim is contested and partly an ascertainment artefact.

● CLASSIC TRAP

Penetrance vs expressivity: penetrance is *whether* the gene shows (a population proportion); expressivity is *how much* it shows in an affected individual. A reduced-penetrance trait can appear to skip a generation; a variable-expressivity trait never "skips" but looks different each time it appears.

2 Methods of psychiatric genetics

The methods form a logical ladder: family studies show a disorder *runs* in families, twin studies separate genes from shared environment, adoption studies separate genes from *all* rearing environment, and molecular methods try to name the variants.

Method	Question answered	Limitation
Family studies	does risk rise with closeness of relationship?	cannot separate genes from shared environment
Twin studies	how much of the variance is genetic (heritability)?	assumes MZ and DZ share environment equally
Adoption studies	genes or rearing environment?	rare, selective placement, small samples
Linkage	which chromosomal region co-segregates?	good for rare large-effect genes, weak for polygenic
Association / GWAS	which common variants differ between cases and controls?	tiny per-variant effects; needs huge samples

Family studies

- **Degree of relationship** – first-degree relatives (parents, sibs, children) share ~50% of genes; second-degree ~25%; third-degree ~12.5%. If a disorder is genetic, **relative risk rises as shared genes rise**.
- **Relative risk** – risk in relatives of a case divided by population risk. Schizophrenia: ~1% population risk rising to ~10% in a first-degree relative is the textbook anchor.

Twin studies

- **MZ vs DZ** – monozygotic twins share ~100% of genes, dizygotic ~50%. Both share the rearing environment. So a **higher MZ than DZ concordance** points to a genetic contribution.
- **Heritability (h^2)** – the proportion of *phenotypic variance in a population* explained by genetic variance. It is a population statistic, *not* the chance an individual inherits the disorder. **Falconer's approximation**: $h^2 \approx 2 \times (r_{MZ} - r_{DZ})$, i.e. twice the difference in concordance between MZ and DZ pairs.

● CLASSIC TRAP

Heritability of ~80% for schizophrenia does **not** mean 80% of *your* risk is fixed at birth, nor that the environment is irrelevant. It means ~80% of the *variance between people* in a given population is genetic. It is population-specific and changes if the environment changes.

Adoption studies

- **The design** – the cleanest separation of genes from environment: a child shares *genes* with biological parents and *environment* with adoptive parents. Compare which predicts the disorder.
- **Heston (1966)** – children of mothers with schizophrenia, adopted away at birth, still showed raised rates of schizophrenia: genes travelled with the child, not the rearing home.
- **Danish adoption studies** – Kety, Rosenthal, Wender and colleagues used the Danish registers. Biological relatives of adoptees with schizophrenia had elevated rates; adoptive relatives did not. Landmark evidence for a genetic basis; also broadened the **schizophrenia spectrum** concept.

Molecular methods

Linkage

Within families: tracks which chromosomal region is co-inherited with the disorder across a pedigree. Powerful for **rare, large-effect** Mendelian genes. Largely **failed** for the common polygenic disorders.

Association / GWAS

Across populations: compares allele frequencies in cases vs controls. **GWAS** scans millions of common variants genome-wide. Each hit has a tiny effect, so it needs very large samples and strict significance ($p < 5 \times 10^{-8}$).

- **Candidate genes** – the older approach: pick a biologically plausible gene (a dopamine or serotonin gene) and test it. Most early candidate-gene findings **failed to replicate** in large samples; the field moved to hypothesis-free GWAS.
- **Copy-number variants (CNVs)** – deletions or duplications of stretches of DNA. **Rare but large-effect**: the 22q11.2 deletion sharply raises schizophrenia risk. CNV burden is increased in schizophrenia and autism. The complement to common-variant GWAS.
- **Endophenotypes** – measurable, heritable intermediate traits sitting *between* genes and the diagnosis (e.g. impaired smooth-pursuit eye movements, P50 sensory gating, prepulse inhibition deficits in schizophrenia). They are meant to be simpler and closer to gene action than the clinical syndrome.
- **Gene-environment interaction (G×E) vs correlation (rGE)** – **interaction**: a genotype changes *sensitivity* to an environment (the same stressor harms one genotype more). **Correlation**: genes *shape exposure* to environments (passive, evocative, active – a child's genotype influences the environments they meet).

3 Heritability & recurrence-risk table

All figures below are **approximate textbook ranges**; precise numbers vary by study, diagnostic criteria and population. Learn the *rank order* and the round anchors, not false precision.

Disorder	Heritability (approx.)	First-degree relative risk	MZ vs DZ concordance
Schizophrenia	~80%	~10% (vs ~1% population)	MZ ~40–50% > DZ ~10–15%
Bipolar disorder	~80–85%	~5–10%	MZ ~40–70% > DZ ~5–10%
Major depression	~35–40%	~2–3× population	MZ > DZ, modest gap
Autism spectrum	~80%	recurrence in sibs markedly raised	MZ >> DZ
ADHD	~75%	raised in first-degree relatives	MZ > DZ
Alcohol dependence	~50%	raised, esp. in sons of affected fathers	MZ > DZ
Alzheimer's disease	late-onset ~moderate (polygenic, **APOE-ε4** the main common risk allele); rare early-onset is **Mendelian** (APP, PSEN1, PSEN2)	raised with affected first-degree relative	MZ > DZ

QUICK RULE

Highest heritability cluster: **schizophrenia, bipolar, autism, ADHD (~75–85%)**. Lower: **alcohol dependence ~50%, major depression ~35–40%**. Bipolar sits at or slightly above schizophrenia; depression is the lowest of the common adult disorders.

TRAP

High *heritability* coexists with *modest absolute recurrence risk*. Schizophrenia is ~80% heritable yet a first-degree relative's risk is only ~10%, because the population baseline is ~1% and the threshold model needs many liability factors to coincide. The two numbers answer different questions.

4 Specific findings & mechanisms

22q11.2 deletion (velocardiofacial / DiGeorge)

◆ **22q11.2 deletion syndrome** – the strongest single common genetic risk factor for schizophrenia known: roughly **1 in 4 carriers** develops a schizophrenia-spectrum psychosis. Phenotype includes congenital heart defects, palatal anomalies, hypocalcaemia and a characteristic face. A clean example of a **CNV** with large effect.

Historically discussed candidate genes

Gene	Proposed link	Status
COMT	catechol-O-methyltransferase; prefrontal dopamine clearance (Val/Met)	much-studied, weak / inconsistent
DISC1	"Disrupted in Schizophrenia 1"; from a Scottish translocation family	landmark family, not a general risk gene
Neuregulin-1 (NRG1)	neurodevelopment, glutamatergic signalling	early hit, poor replication
BDNF	brain-derived neurotrophic factor (Val66Met); plasticity, mood	popular, small / inconsistent

● CLASSIC TRAP

These names still appear in vivas, so know them, but the honest examiner answer is that **most single candidate-gene findings did not robustly replicate**. The field shifted to large GWAS and polygenic risk scores, which confirm that risk is spread across *thousands* of common variants of tiny effect, not concentrated in a few named genes.

The shift: GWAS and polygenic risk scores

■ **Polygenic risk score (PRS)** – sums the small effects of many risk alleles across the genome into a single number for an individual. Schizophrenia GWAS now implicate **hundreds of loci** (including the **MHC / complement C4** region linked to synaptic pruning). PRS is a powerful *research* tool but **not yet a clinical diagnostic test**: it predicts at the group level, not the individual.

Epigenetics

● **Epigenetics** – heritable changes in gene *expression* without a change in DNA *sequence*. Main mechanisms: **DNA methylation** (usually silencing) and histone modification. A route through which environment marks the genome.

◆ **Imprinting** – expression depends on the **parent of origin** of the allele. The exam exemplar is a single deletion on chromosome **15q11–13** producing two different syndromes: **Prader-Willi** if the *paternal* copy is missing (hyperphagia, obesity, hypotonia, intellectual disability), **Angelman** if the *maternal* copy is missing (ataxia, seizures, inappropriate laughter, severe speech impairment).

● MNEMONIC

"Prader = Pater (father); Angelman = mAternal"

Same 15q11–13 deletion. Lose the **pa**ternal copy → **P**rader-Willi. Lose the m**a**ternal copy → **A**ngelman. The classic genomic-imprinting question.

5 Gene-environment interaction

Genes do not act in a vacuum. The clean idea is the **diathesis-stress** model: a genetic vulnerability is expressed only when an environmental stressor pushes liability over the threshold.

- **Diathesis-stress** – predisposition (diathesis) + trigger (stress) = disorder. Neither alone is sufficient in many cases. The general frame within which G×E findings sit.

The Caspi studies (the two famous G×E results)

Study	Gene	Environment	Outcome
Caspi 2003	**5-HTTLPR** short allele (serotonin transporter promoter)	stressful life events	more depression in short-allele carriers exposed to stress
Caspi 2002	**MAOA** low-activity variant	childhood maltreatment	more antisocial / conduct behaviour in maltreated low-activity carriers

REPLICATION CAVEAT (SAY THIS)

Both findings are **foundational and heavily cited**, but later meta-analyses gave **inconsistent replication**, and candidate-gene G×E as a whole is now viewed cautiously (small samples, publication bias, flexible analysis). Cite them as the landmark *illustrations* of gene-environment interaction, then add the replication caveat. That balance is the mark-scoring answer.

6 Clinical genetic counselling basics

Psychiatric genetic counselling rests on **empirical recurrence risks** (observed family-study figures), not on Mendelian pedigree calculations, because the disorders are polygenic and multifactorial.

- **Empirical risks** – quote observed rates: ~10% for a sibling or child of someone with schizophrenia, higher if more relatives are affected or onset was early. These are population averages, not individual certainties.
- **The non-deterministic message** – genes raise *probability*, they do not *determine* outcome. Most children of a parent with schizophrenia do **not** develop it. Counselling is non-directive: inform, support reproductive autonomy, reduce guilt and stigma, and address modifiable risk where possible.
- **Limits in psychiatry** – no validated predictive genetic test for the common disorders; PRS is not clinically diagnostic; diagnoses themselves shift over time; and figures are population-level. Counselling stays probabilistic and supportive, never a sequence-based verdict.

HOLD THIS

Psychiatric counselling uses **empirical recurrence risks**, delivers a **non-deterministic, non-directive** message, and has **no clinical predictive gene test**. Probability, not prophecy.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Core claim:** major psychiatric disorders are **polygenic + multifactorial** (many small-effect variants + environment), modelled by a continuous **liability with a threshold**.

- **Three confusions:** penetrance = *whether* the gene shows (proportion of carriers) · expressivity = *how much* it shows · anticipation = earlier/worse each generation (true mechanism = trinucleotide repeat expansion).
- **Methods ladder:** family (runs in families) → twin (separates genes from shared environment) → adoption (separates genes from rearing) → linkage/GWAS (names variants).
- **Twin maths:** MZ ~100% genes, DZ ~50%; MZ > DZ concordance = genetic. **Falconer:** $h^2 \approx 2(r_{MZ} - r_{DZ})$. Heritability = variance explained *in a population*, not individual fate.
- **Adoption landmarks:** Heston (1966) and the Danish studies (Kety, Rosenthal, Wender) – biological, not adoptive, relatives carry the risk.

- **Heritability anchors (approx.):** schizophrenia ~80% (first-degree ~10%, MZ ~40–50%) · bipolar ~80–85% · autism ~80% · ADHD ~75% · alcohol dependence ~50% · major depression ~35–40% (the lowest).
- **CNV star: 22q11.2 deletion** (velocardiofacial) – ~1 in 4 develop schizophrenia-spectrum psychosis. Large-effect CNV.
- **Candidates (mostly failed):** COMT, DISC1, neuregulin-1, BDNF – know the names, note the poor replication; field moved to GWAS + **polygenic risk scores** (hundreds of loci; complement C4 / pruning).
- **Imprinting exemplar:** 15q11–13 deletion – lose **pat**ernal = **P**rader-Willi, lose m**a**ternal = **A**ngelman.
- **G×E (Caspi):** 5-HTTLPR short allele × life events → depression; MAOA low-activity × maltreatment → antisocial behaviour. Always add the **replication caveat**.
- **Counselling:** empirical recurrence risks, non-directive, non-deterministic; no clinical predictive gene test; PRS is research-only.

ONE-DAY REVISION · PAPER 1

Core psychology, by who said it

You know the concepts. What slips is the name on each one. This pack pins every contribution to its author, dates the famous ones, and closes with a ledger of every figure PG psychiatry asks about. Read the eminent-figures table last, out loud.

● CONCEPT ● RULE ● TRAP ● NAME / DATE ● MNEMONIC

The colour tells you what kind of fact it is. Amber here = the attribution to memorise.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Wundt, 1879, Leipzig opened the first psychology laboratory, marking the birth of psychology as a science; this is the single most-asked date in this paper.
2. Defense mechanisms: Anna Freud, 1936 gave the first systematic classification, while Vaillant, 1977 ranked them by maturity (Grant Study); mature five = SHASA (Sublimation, Humour, Altruism, Suppression, Anticipation), with sublimation the most adaptive.
3. K-name trap: Kohler = insight learning (Gestalt), Kohut = self psychology, Kohlberg = moral development, Kernberg = borderline organisation/TFP; Cattell appears twice (16PF in personality, fluid vs crystallised in intelligence).
4. Grief models: Kubler-Ross 5 stages (DABDA, described for the dying, not the bereaved, and not linear), Worden 4 active tasks, Bowlby 4 phases, Lindemann acute grief work; Prolonged Grief Disorder entered DSM-5-TR (2022) at >12 mo and ICD-11 at 6 mo.
5. Attachment: Bowlby = theory (secure base, internal working models), Ainsworth = Strange Situation and the first three types, Main = added disorganized later (NOT Ainsworth), Harlow = contact comfort beats feeding in rhesus monkeys.

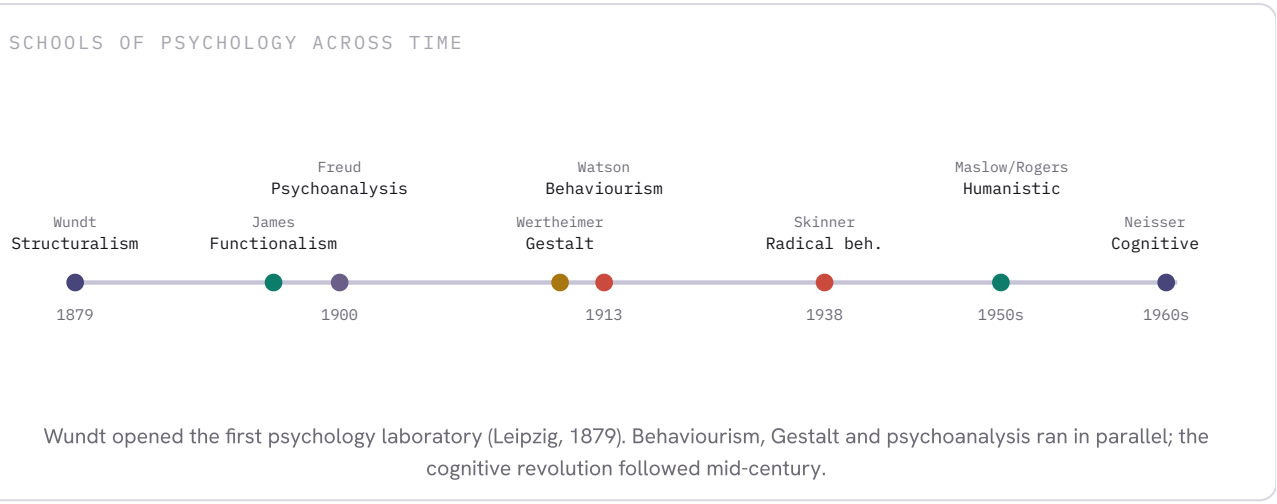
1 Schools of psychology

Asked as “name the schools and their founders”. The first laboratory date and the founder pairs are the high-value facts.

School	Founder(s)	Core idea
Structuralism	Wilhelm Wundt (1879, first lab, Leipzig); Titchener	introspection breaks consciousness into elements
Functionalism	William James	what mind is *for* (adaptation), not what it is made of
Behaviourism	J. B. Watson; B. F. Skinner	study observable behaviour only; stimulus → response
Gestalt	Wertheimer, Kohler, Koffka	the whole > sum of parts; perception is organised
Psychoanalysis	Sigmund Freud	unconscious drives, conflict, defence
Humanistic	Maslow, Carl Rogers	self-actualisation, growth, the “third force”
Cognitive	Neisser (1967, named the field)	mind as information processor; the “cognitive revolution”

HOLD THIS

Wundt, 1879, Leipzig = first psychology laboratory = birth of psychology as a science. The single most-asked date in this paper.



Wundt opened the first psychology laboratory (Leipzig, 1879). Behaviourism, Gestalt and psychoanalysis ran in parallel; the cognitive revolution followed mid-century.

2 Defense mechanisms

High yield, asked every 3 to 4 exams as "define, classify, give examples". Define first, then classify by maturity, then go disorder-specific.

● **Definition** – unconscious strategies the **ego** uses to manage conflict between id, superego and reality, reducing anxiety from unacceptable impulses.

◆ **Anna Freud, 1936** – first systematic classification (*The Ego and the Mechanisms of Defence*).
Vaillant, 1977 – the maturity hierarchy, validated in the Grant Study of Harvard men.

Level	Defenses	Seen in
Mature	sublimation, humour, altruism, suppression, anticipation	well-adjusted adults; best prognosis
Neurotic	intellectualisation, isolation of affect, reaction formation, displacement, repression, dissociation, undoing	anxiety, OCD, conversion
Immature	projection, projective identification, splitting, acting out, regression, passive aggression, somatisation, fantasy, idealisation/devaluation	personality disorders, adolescents
Psychotic	denial, distortion, psychotic (delusional) projection	psychosis, severe PD; reality lost

● MNEMONIC

"MNI" tiers · mature = "SHASA"

Mature > **N**eurotic > **I**mmature (a building, mature on top). Mature five = **S**ublimation, **H**umour, **A**ltruism, **S**uppression, **A**nticipation. Sublimation is the single most adaptive defence.

● CLASSIC TRAP

Suppression = conscious postponement (mature). **Repression** = unconscious exclusion (neurotic). **Denial** acts on **external reality**; repression acts on **internal wishes**.

Defenses in specific disorders

Disorder	Defenses	Anchor
OCD	isolation of affect, undoing, reaction formation, intellectualisation	mnemonic "I URI"
Phobia	displacement, projection, avoidance	Little Hans: castration anxiety displaced onto horses

Disorder	Defenses	Anchor
Borderline PD	splitting, projective identification, idealisation/devaluation, acting out	splitting is the cardinal BPD defence
Conversion	repression, conversion, dissociation	often with *la belle indifference*

3 Developmental psychology

Freud, Erikson and Piaget run on parallel ages. Erikson is asked **with the psychopathology of each failed stage**; that is the marks.

Erikson's eight psychosocial stages

Age	Crisis	Virtue	If unresolved (psychopathology)
0–1	**Trust vs Mistrust**	Hope	depression, paranoid traits, substance dependence
1–3	**Autonomy vs Shame/Doubt**	Will	OCD, OCPD, excessive shame, self-doubt
3–6	**Initiative vs Guilt**	Purpose	conversion, phobias, inhibition, psychosomatic illness
6–12	**Industry vs Inferiority**	Competence	inferiority complex, low self-esteem, work paralysis
12–20	**Identity vs Role Confusion**	Fidelity	identity diffusion, BPD traits, cult susceptibility
20–40	**Intimacy vs Isolation**	Love	avoidant/schizoid withdrawal, chronic loneliness
40–65	**Generativity vs Stagnation**	Care	midlife crisis, self-absorption, depression
65+	**Integrity vs Despair**	Wisdom	late-life depression, despair, fear of death

● NAMES ATTACHED

Virtues in order: **Hope, Will, Purpose, Competence, Fidelity, Love, Care, Wisdom**. Marcia extended Stage 5 into four identity statuses: achievement, moratorium, foreclosure, diffusion.

Side-by-side: Freud, Erikson, Piaget

Age	Freud (psychosexual)	Erikson (psychosocial)	Piaget (cognitive)
0–1	Oral	Trust vs Mistrust	Sensorimotor · object permanence
1–3	Anal	Autonomy vs Shame	
3–6	Phallic / Oedipal	Initiative vs Guilt	Preoperational · egocentrism, no conservation
6–7	Latency	Industry vs Inferiority	
7–11			Concrete operational · conservation, reversibility
11–18	Genital	Identity vs Role Confusion	Formal operational · abstract reasoning

● RULE

Erikson = psycho**social**, whole lifespan (8 stages). Freud = psycho**sexual**, stops at adolescence (5 stages). Piaget = cognitive, four stages. **Object permanence** → sensorimotor (~8 mo, with A-not-B error); **conservation** → concrete operational; **abstract thought** → formal operational.

Attachment

Type (Ainsworth, Strange Situation)	On reunion	Adult risk
Secure (~60%)	distressed on separation, soothed on reunion	protective

Type (Ainsworth, Strange Situation)	On reunion	Adult risk
Anxious-ambivalent / resistant (~15%)	very distressed, not comforted, clingy + angry	anxiety, dependent PD, depression
Anxious-avoidant (~20%)	little distress, ignores caregiver	schizoid, avoidant PD
Disorganized (~5%)	contradictory, freezing, "fright without solution"	BPD, dissociation, complex PTSD

● TRAP

Bowlby = attachment theory (innate, primary drive; secure base; internal working models). Ainsworth = the Strange Situation and the first three types. Main & Solomon/Hesse added **disorganized** later (not Ainsworth). Harlow = rhesus monkey wire-vs-cloth-mother studies → contact comfort beats feeding. Adult Attachment Interview by Main.

- **Kohlberg · moral development** – three levels: **pre-conventional** (punishment/reward), **conventional** (social approval, law-and-order), **post-conventional** (social contract, universal ethics). Built on Piaget's cognitive stages.
- **Mahler · separation-individuation** – normal autism → symbiosis → differentiation, practising, **rapprochement** (the crisis BPD is theorised to arise from), object constancy.

4 Grief & bereavement

The sleeper hit, asked 6+ times: define the three terms, give the models, separate normal from pathological, then manage.

Term	Meaning
Bereavement	the objective *fact* of having suffered a loss
Grief	the emotional/cognitive/behavioural *reaction* to loss
Mourning	the social/cultural *process* of adapting (rituals)

Model	Author / year	Components
5 stages	Kubler-Ross, 1969	Denial, Anger, Bargaining, Depression, Acceptance (DABDA)
4 tasks	Worden, 1991	Accept reality, Process pain, Adjust, find Enduring connection
4 phases	Bowlby, 1980	Numbness, Yearning, Disorganisation/despair, Reorganisation
Dual process	Stroebe & Schut, 1999	oscillation: loss-oriented ↔ restoration-oriented
Acute grief work	Lindemann, 1944	Cocoanut Grove fire survivors; somatic distress, guilt, hostility, "grief work"

● CLASSIC TRAP

Kubler-Ross stages were described for **dying patients**, not the bereaved, and are **not** linear or universal. Worden's are **tasks** (active, done by the mourner), not passive stages. Both distinctions are favourite questions.

Feature	Normal grief	Major depression	Prolonged Grief Disorder
Focus of distress	the loss	the self (worthlessness)	longing for the deceased
Mood	reactive, in waves	pervasive, non-reactive	persistent yearning
Duration	eases over 6–12 mo	≥ 2 weeks, persistent	> 12 mo (DSM-5-TR) / 6 mo (ICD-11)

QUICK RULE

Grief = focused on the **loss**; depression = focused on the **self**. PGD added to **DSM-5-TR (2022)** and **ICD-11**. Management of complicated grief: **Shear's Complicated Grief Treatment** (IPT + exposure), not antidepressants alone; avoid benzodiazepines.

5 Eminent figures & contributors · the reference

The heart of this pack. Every figure PG psychiatry asks about, grouped by domain, one tight line each. The amber name is what you are graded on.

Learning & behaviour

Name	Contribution	Exam one-liner
Pavlov	classical conditioning	dogs, salivation; CS + UCS; the original associative learning
Watson	behaviourism; Little Albert	conditioned fear of a white rat in an infant
Thorndike	**Law of Effect**	cats in puzzle boxes; rewarded behaviour is stamped in
Skinner	operant conditioning	Skinner box, reinforcement schedules, shaping
Tolman	latent learning, cognitive maps	rats learn the maze without reward; learning ≠ performance
Kohler	**insight learning**	chimpanzee Sultan stacks boxes; the "aha" (Gestalt)
Bandura	social learning; self-efficacy	**Bobo doll**; observation, modelling, vicarious reinforcement
Wolpe	systematic desensitisation	reciprocal inhibition; the basis of exposure therapy

Intelligence

Name	Contribution	Exam one-liner
Binet	first intelligence test	Binet-Simon scale; concept of mental age
Spearman	**two-factor**: g + s	general intelligence (g) underlies all abilities
Thurstone	primary mental abilities	7 independent factors; rejected a single g
Cattell	**fluid vs crystallised**	fluid = reasoning (declines); crystallised = knowledge (holds)
Gardner	multiple intelligences	linguistic, logical, musical, spatial, etc. (8+)
Sternberg	**triarchic** theory	analytical, creative, practical intelligence
Guilford	structure of intellect	convergent vs divergent thinking; 120+ factors
Wechsler	WAIS / WISC	deviation IQ; mean 100, SD 15; verbal + performance

Memory

Name	Contribution	Exam one-liner
Ebbinghaus	**forgetting curve**	nonsense syllables; spacing effect; exponential decay
Atkinson-Shiffrin	multi-store model	sensory → short-term → long-term (the "modal model")
Baddeley (& Hitch)	**working memory**	central executive, phonological loop, visuospatial sketchpad, episodic buffer
Tulving	episodic vs semantic	declarative memory split into events vs facts
Miller	**7 ± 2**	"the magical number"; capacity of short-term memory; chunking
Bartlett	**schema** theory	"War of the Ghosts"; memory is reconstructive
Loftus	misinformation effect	false / recovered memory; eyewitness unreliability

Emotion

Name	Contribution	Exam one-liner
James-Lange (1884)	peripheral theory	body first → emotion; "I tremble, therefore I fear"
Cannon-Bard (1927)	central / simultaneous	thalamus fires cortex + body at once

Name	Contribution	Exam one-liner
Schachter-Singer (1962)	**two-factor**	arousal + cognitive label = emotion; adrenaline study
Lazarus	cognitive appraisal	appraisal precedes and shapes emotion
Ekman	basic emotions; FACS	6 universal expressions; cross-cultural (DASH = Disgust, Anger, Sadness, Happiness, Fear, Surprise)
Papez (1937)	Papez circuit	first neural circuit of emotion (hippocampus → cingulate)
MacLean (1952)	triune brain; coined **"limbic system"**	reptilian → paleomammalian → neomammalian
Damasio (1994)	somatic marker hypothesis	Iowa Gambling Task; emotions guide rational decisions
Plutchik	wheel of emotions	8 primaries in 4 opposing pairs

Personality

Name	Contribution	Exam one-liner
Allport	trait theory	cardinal, central, secondary traits
Cattell	**16PF**	16 source traits by factor analysis
Eysenck	**PEN** model	Psychoticism, Extraversion, Neuroticism; biological basis
Costa & McCrae	**Big Five / OCEAN**	Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism
Kelly	personal construct theory	person as scientist; repertory grid technique

Social psychology

Name	Contribution	Exam one-liner
Festinger	**cognitive dissonance**	discomfort from inconsistent cognitions drives change
Asch	**conformity**	line-judgement task; group pressure overrides accuracy
Milgram	**obedience**	shock experiment; obedience to authority
Zimbardo	Stanford Prison Experiment	roles and situation shape behaviour; deindividuation
Heider	attribution theory	internal (dispositional) vs external (situational) causes
Sherif	realistic conflict; Robbers Cave	superordinate goals reduce intergroup conflict; autokinetic norms
Tajfel	**social identity** theory	in-group / out-group; minimal group paradigm

Motivation

Name	Contribution	Exam one-liner
Maslow	**hierarchy of needs**	physiological → safety → love → esteem → self-actualisation
McClelland	needs theory	need for achievement, affiliation, power
Herzberg	two-factor theory	hygiene factors vs motivators (work motivation)
Deci & Ryan	**self-determination theory**	autonomy, competence, relatedness; intrinsic motivation

Psychoanalytic & dynamic

Name	Contribution	Exam one-liner
Freud	psychoanalysis	id-ego-superego; unconscious; psychosexual stages; repression
Jung	analytical psychology	collective unconscious, archetypes, introvert/extravert
Adler	individual psychology	inferiority complex, striving for superiority, birth order

Name	Contribution	Exam one-liner
Anna Freud	ego psychology; child analysis	systematised the defence mechanisms (1936)
Klein	object relations	paranoid-schizoid & depressive positions; play technique
Winnicott	object relations	good-enough mother, transitional object, true/false self, holding
Kohut	self psychology	narcissism; selfobject; empathic mirroring
Kernberg	object relations; BPD	borderline personality organisation; TFP; splitting
Erikson	psychosocial development	8 lifespan stages; identity crisis
Mahler	separation-individuation	rapprochement; object constancy
Bowlby	attachment theory	secure base; internal working models

Cognitive therapy roots

Name	Contribution	Exam one-liner
Beck	**cognitive therapy**	cognitive triad, schemas, automatic thoughts; depression
Ellis	**REBT**	ABC model; irrational beliefs; rational emotive
Seligman	**learned helplessness**	dogs, inescapable shock; model of depression; later positive psychology

● MOST-CONFUSED NAMES

Kohler (insight learning, Gestalt) vs **Kohut** (self psychology) vs **Kohlberg** (moral development) vs **Kernberg** (BPD). **Cattell** appears twice: 16PF (personality) and fluid/crystallised (intelligence). Big Five = **Costa & McCrae**; PEN = **Eysenck**; 16 factors = **Cattell**.

QUICK-RECALL · DRILL THESE ATTRIBUTIONS OUT LOUD

★ **Wundt 1879** = first psychology lab (Leipzig). James = functionalism. Watson/Skinner = behaviourism. Neisser = cognitive (1967).

- Defenses: **Anna Freud 1936** classified; **Vaillant 1977** ranked by maturity. Mature = SHASA. OCD = isolation/undoing/reaction formation/intellectualisation. Phobia = displacement/projection.
- Erikson virtues: **Hope, Will, Purpose, Competence, Fidelity, Love, Care, Wisdom**. Object permanence = sensorimotor; conservation = concrete; abstract = formal.
- Attachment: **Bowlby** theory, **Ainsworth** Strange Situation (3 types), disorganized added by **Main**. **Harlow** = contact comfort monkeys.
- Grief: **Kubler-Ross** 5 stages (for the dying), **Worden** 4 tasks (active), **Bowlby** 4 phases, **Lindemann** acute grief. PGD = DSM-5-TR 2022.
- Emotion: **James-Lange** body-first · **Cannon-Bard** simultaneous · **Schachter-Singer** arousal + label · **Papez** circuit · **Ekman** 6 basic.
- Intelligence: **Spearman** g · **Cattell** fluid/crystallised · **Gardner** multiple · **Sternberg** triarchic · **Wechsler** WAIS.
- Memory: **Ebbinghaus** forgetting curve · **Baddeley** working memory · **Miller** 7±2 · **Tulving** episodic/semantic · **Loftus** false memory.
- Personality: **Eysenck** PEN · **Costa & McCrae** OCEAN · **Cattell** 16PF · **Kelly** constructs.
- Social: **Festinger** dissonance · **Asch** conformity · **Milgram** obedience · **Zimbardo** prison · **Tajfel** social identity.
- Motivation: **Maslow** hierarchy · **McClelland** needs · **Deci & Ryan** self-determination.
- Cognitive therapy: **Beck** cognitive triad · **Ellis** REBT/ABC · **Seligman** learned helplessness.

ONE-DAY REVISION · PAPER 1

Theories in psychology, made retrievable

The conceptual theories: emotion, perception, learning, memory, motivation, intelligence, personality. Hold the sequence, the one claim that distinguishes each, and the name attached to it. Every comparison table is a 10-mark answer in waiting.

CONCEPT · RULE · TRAP · KEY NUMBER · MNEMONIC

The colour tells you what kind of fact it is. People, defenses & developmental stages live in the companion pack.

★ HIGH YIELD — TOP 5 IF NOTHING ELSE

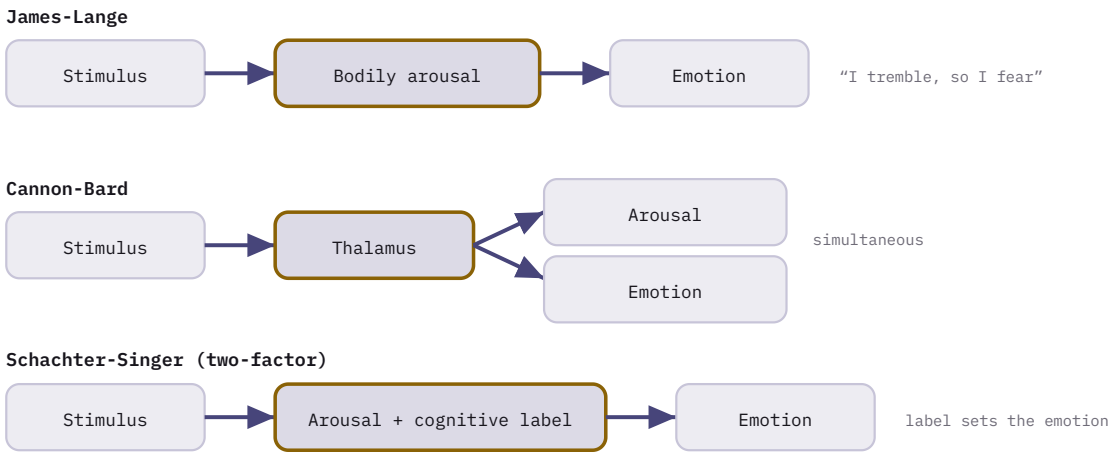
1. The four emotion theories by sequence: James-Lange = body/arousal first, Cannon-Bard = arousal and emotion simultaneous (via thalamus), Schachter-Singer = arousal plus a cognitive label (two-factor), Lazarus = cognitive appraisal first. These contrasts carry the marks.
2. Negative reinforcement is NOT punishment: it removes an aversive to increase a behaviour, and it is how avoidance in phobias and OCD is maintained (escaping anxiety negatively reinforces the avoidance).
3. Variable ratio gives the highest, steadiest response rate and is the most resistant to extinction (the slot machine); general rule, ratio > interval for rate, variable > fixed for persistence.
4. STM holds 7 ± 2 items (Miller) for ~15-30 s; Baddeley working memory = central executive directing the phonological loop, visuospatial sketchpad, and episodic buffer; declarative (episodic + semantic) vs procedural memory dissociate in patient H.M.
5. Deviation IQ is normally distributed, mean 100, SD 15 (Wechsler), so <70 is the ID cut-off and >130 the gifted range; Cattell-Horn fluid intelligence falls with age while crystallised holds or climbs.

1 Theories of emotion

The whole topic turns on one question: what comes first, the body or the feeling. Each theory answers it differently. Get the **sequence** right and the rest follows.

Theory	Sequence	Key claim	Critique
James-Lange (1884)	stimulus → **arousal** → emotion	emotion *is* the perception of bodily change. "I tremble, therefore I fear"	visceral changes too slow & too alike to explain emotion's speed and variety (Cannon)
Cannon-Bard (1927)	stimulus → thalamus → **arousal + emotion together**	body and feeling are independent, simultaneous; both fire from the thalamus	oversimplifies thalamus; bodily patterns *do* differ across emotions
Schachter-Singer (1962)	stimulus → arousal + **cognitive label** → emotion	two-factor: undifferentiated arousal + the label context supplies	replication issues; arousal not fully undifferentiated; appraisal not always needed
Lazarus appraisal (1991)	stimulus → **cognitive appraisal** → arousal + emotion	appraisal (primary: is it relevant? secondary: can I cope?) precedes and shapes emotion	Zajonc countered that some affect is pre-cognitive (mere exposure)
Damasio somatic marker (1994)	body states tag options → guide choice	"somatic markers" bias decisions; emotion serves reason, not opposes it	hard to test in full; markers are one input among several

THE CLASSIC EMOTION THEORIES COMPARED



All start from the same stimulus. James-Lange: arousal first, then emotion. Cannon-Bard: arousal and emotion at once (via thalamus). Schachter-Singer: arousal plus a cognitive label.

LeDoux: the two roads to the amygdala

- **Low road** – thalamus → amygdala. Fast, crude, subcortical. "Shoot first, ask questions later." Lets you flinch from a stick before you know it isn't a snake.
- **High road** – thalamus → cortex → amygdala. Slow, accurate, contextual. Confirms or cancels the low-road alarm.

The substrate: Papez and MacLean

- **Papez circuit** (1937) – hippocampus → fornix → mammillary bodies → anterior thalamus → cingulate → back to hippocampus. First neural model of emotion; now read as more **memory** than feeling.
- **MacLean's triune brain** – reptilian (brainstem, reflexes) → paleomammalian (**limbic system**, emotion) → neomammalian (neocortex, reason). MacLean *coined* "limbic system." Critiqued as oversimplified: brains did not evolve in neat layers.

HOLD THIS

James-Lange = **body first**. Cannon-Bard = **simultaneous**. Schachter-Singer = **arousal + label**. Lazarus = **appraisal first**. These four contrasts carry the marks.

● CLASSIC TRAP

Schachter-Singer's adrenaline study: epinephrine-injected subjects who had **no explanation** for their arousal borrowed the confederate's mood (euphoric or angry). Same arousal, different label, different emotion. This is the clinical root of **panic disorder** (catastrophic misattribution of normal arousal) and of reappraisal in CBT.

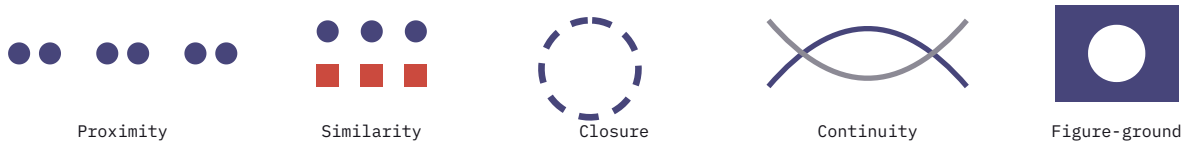
2 Perception

Perception is not passive recording. The brain organises and infers. Gestalt names *how* raw sensation is grouped into wholes.

Gestalt grouping principles

Principle	We group elements that are…
Proximity	close together
Similarity	alike in shape, colour or size
Closure	nearly complete – the mind fills the gap
Continuity (good continuation)	aligned along a smooth path
Figure-ground	separated into an object vs its background (Rubin's vase)
Common fate	moving together in the same direction

GESTALT GROUPING PRINCIPLES



The mind groups by proximity, similarity, closure (completing a gap), continuity (smooth paths) and figure-ground. Also: common fate and pragnanz (the simplest form).

● MNEMONIC

"Pragnanz" → the mind prefers the simplest, most stable whole

The Law of Pragnanz (good form) is the master Gestalt principle the six rules serve. The whole is other than the sum of its parts.

Depth cues

Binocular (two eyes)

Retinal disparity – each eye sees a slightly different image; the offset codes depth (the basis of stereopsis).
Convergence – the eyes turn inward for near objects; muscle feedback signals distance.
Effective only at near range.

Monocular (one eye, pictorial)

Relative size, interposition (overlap), **linear perspective, texture gradient, aerial perspective, light & shadow, relative height,** and **motion parallax**. These let a flat painting convey depth.

Constancy, set, and the two directions of processing

- **Perceptual constancy** – objects look stable despite changing input: size, shape, colour and brightness constancy. A door is "rectangular" even as its retinal image skews to a trapezoid.
- **Bottom-up** – data-driven, built from the raw stimulus upward (Gibson's direct perception).
- **Top-down** – concept-driven, guided by expectation, context and prior knowledge (Gregory).

Perceptual set is top-down: what you expect, you tend to see. Illusions expose where the inferences misfire.

Signal detection theory

Detecting a faint signal is never pure sensation; it is sensation plus a **decision criterion**. The same sensitivity can yield different hit rates as the criterion shifts.

	Signal present	Signal absent
Said "yes"	Hit	False alarm
Said "no"	Miss	Correct rejection

◆ **d-prime (d')** – pure **sensitivity**, independent of bias: how far apart the signal and noise distributions sit. A separate parameter, β (the criterion), captures the willingness to say "yes."

● RULE

Sensitivity (d') is intrinsic to the observer's discrimination. Criterion (bias) is strategic and shifts with payoffs and expectation. A nervous radiologist (liberal criterion) catches more tumours (more hits) but at the cost of more false alarms, while d' is unchanged.

3 Learning theories

Classical conditioning (Pavlov)

An automatic reflex is transferred to a new, neutral cue by pairing. Learning is about **stimuli** that predict each other.

Term	Meaning	Pavlov's dog
US (unconditioned stimulus)	triggers the reflex innately	food
UR (unconditioned response)	the innate reflex	salivation to food
CS (conditioned stimulus)	neutral cue, paired with the US	bell
CR (conditioned response)	learned response to the CS	salivation to the bell

- **Acquisition** – the CR builds as CS and US are paired. Strongest when the CS slightly *precedes* the US.
- **Extinction** – CS presented without the US; the CR fades.
- **Spontaneous recovery** – after a rest, an extinguished CR briefly reappears. Extinction suppresses, it does not erase.
- **Generalisation** – similar stimuli also elicit the CR (Little Albert feared all furry things).
- **Discrimination** – the opposite: responding only to the specific CS, not to similar cues.

Operant conditioning (Skinner)

Behaviour is shaped by its **consequences**. Reinforcement raises a behaviour's frequency; punishment lowers it.

	Add a stimulus (+)	Remove a stimulus (–)
Increases behaviour	Positive reinforcement (give a reward)	Negative reinforcement (remove an aversive: relief)
Decreases behaviour	Positive punishment (apply an aversive)	Negative punishment (remove a reward: a fine, time-out)

● CLASSIC TRAP

Negative reinforcement is **not** punishment. It removes something unpleasant to *increase* a behaviour. Taking an analgesic to end pain reinforces pill-taking. Avoidance in phobias and OCD is maintained this way: escaping anxiety negatively reinforces the avoidance.

Schedules of reinforcement

Schedule	Reward delivered	Response pattern
Fixed ratio (FR)	after a set number of responses	high rate, brief post-reward pause
Variable ratio (VR)	after an unpredictable number	highest, steadiest rate; most resistant to extinction
Fixed interval (FI)	first response after a set time	scalloped – speeds up near the deadline
Variable interval (VI)	first response after an unpredictable time	slow, steady

QUICK RULE

Variable ratio (the slot machine, gambling) gives the highest rate and is **most resistant to extinction**. Ratio > interval for rate; variable > fixed for persistence.

■ **Shaping** – reinforcing successive approximations toward a target behaviour. The basis of skills training and token economies.

Beyond stimulus and response

● **Observational / social learning (Bandura)** – learning by watching a model, no direct reinforcement needed. The **Bobo doll** study: children imitated filmed adult aggression. **Vicarious reinforcement**: seeing the model rewarded raises imitation.

● **Cognitive / latent learning (Tolman)** – rats formed a **cognitive map** of a maze without reward; learning stayed hidden until reward made it worth showing. Learning can occur without performance.

● **Insight learning (Kohler)** – Sultan the chimp solved the out-of-reach-banana problem suddenly, by restructuring the situation – the "aha" moment, not trial and error.

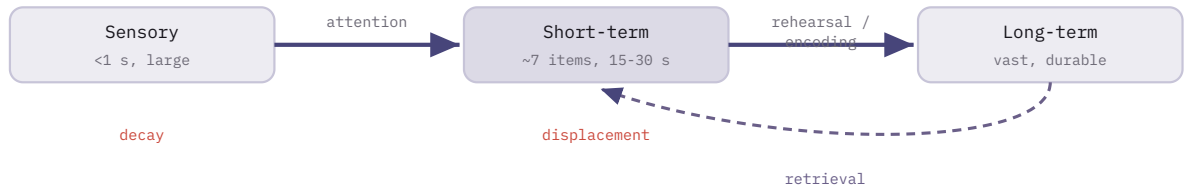
◆ **Preparedness & the Garcia effect** – we are biologically primed to learn survival-relevant links easily. **Taste aversion** forms in a *single* trial and over a delay of hours, breaking the usual contiguity rule. Explains the ease of phobias to snakes/heights over plugs/cars.

4 Memory

The models

Model	Core idea	Authors
Multi-store	three stores in series: sensory → STM → LTM; rehearsal transfers	Atkinson & Shiffrin (1968)
Working memory	STM is active, not a single store; replaces the "rehearsal buffer"	Baddeley & Hitch (1974)
Levels of processing	not stores but **depth**: semantic (deep) encoding outlasts shallow	Craik & Lockhart (1972)

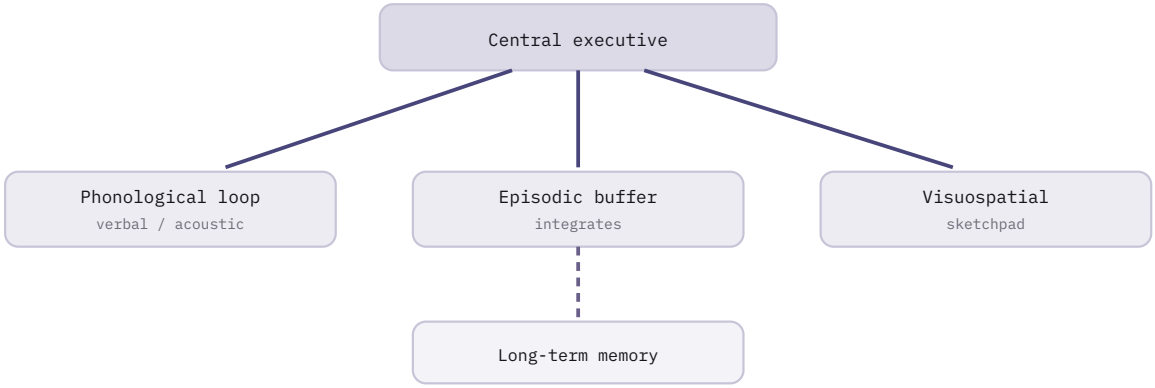
THE MULTI-STORE MODEL OF MEMORY



Atkinson-Shiffrin: attention moves sensory traces to short-term store; rehearsal/encoding consolidates them into long-term memory. Loss is by decay, displacement and interference.

◆ **Capacity & duration** – sensory store: large, <1 s (iconic, echoic). STM: **7 ± 2** items (Miller), ~15–30 s, extended by **chunking** and rehearsal. LTM: effectively unlimited, durable.

BADDELEY'S WORKING MEMORY MODEL



A central executive (attentional controller) directs three slave systems: the phonological loop (verbal), the visuospatial sketchpad, and the episodic buffer that binds them and links to long-term memory.

● **Working memory components** – the **central executive** (attentional controller) directs two slave systems: the **phonological loop** (verbal/acoustic) and the **visuospatial sketchpad** (visual/spatial). The **episodic buffer** (added 2000) binds them with LTM into coherent episodes.

Types of long-term memory

Division	Subtype	What it holds
Declarative (explicit, conscious)	Episodic	personal events, time-stamped (“my last birthday”)
	Semantic	facts and meanings (“Paris is a capital”)
Procedural (implicit, non-conscious)	Skills, habits	how to cycle; primed and conditioned responses

● TRAP

Patient **H.M.** (bilateral medial temporal lobe resection) lost the ability to form new *declarative* memories (anterograde amnesia) but could still learn *procedural* skills (mirror drawing) without any memory of having practised. Declarative and procedural memory are dissociable systems.

Forgetting

- **Decay** – the memory trace fades with disuse over time.
- **Interference** – **proactive**: old learning blocks new (calling a new partner by an ex's name). **Retroactive**: new learning overwrites old (a new phone number blots out the previous one).
- **Retrieval failure** – the trace is intact but cues are missing (tip-of-the-tongue); context- and state-dependent recall.

◆ **Ebbinghaus forgetting curve** – loss is steep at first, then levels off. **Spaced repetition** flattens the curve – the logic of this whole revision series.

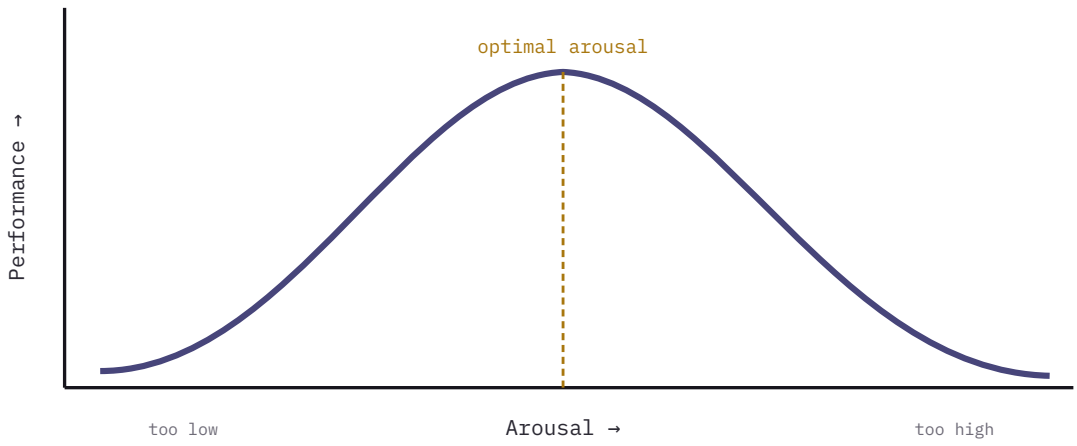
The biology

■ **LTP & consolidation** – long-term potentiation (NMDA-receptor-dependent strengthening of synapses) is the cellular basis of memory. Labile new memories are **consolidated** into stable form, a hippocampus-dependent process; sleep aids it. Reactivation can return a memory to a labile state (reconsolidation).

5 Motivation & arousal

Theory	Drives behaviour because…	Author
Drive reduction	a need creates tension (drive); we act to restore **homeostasis**	Hull
Arousal	we seek an *optimal* level of arousal, not its elimination	Yerkes-Dodson
Incentive	external rewards *pull* behaviour (vs drives that push)	–
Humanistic	needs are ranked; lower must be met before higher	Maslow
Self-determination	three innate needs: autonomy, competence, relatedness	Deci & Ryan
Need theory	learned needs for achievement, affiliation, power	McClelland

THE YERKES-DODSON LAW



Performance rises with arousal up to an optimum, then falls (the inverted U). The optimum is lower for difficult or novel tasks, higher for simple, well-learned ones.

◆ **Yerkes-Dodson law** – performance vs arousal is an **inverted U**: too little (bored, flat) or too much (anxious, frozen) both impair; a middle zone is best. The optimum is **lower for hard/complex tasks** and higher for simple/well-learned ones.

MASLOW'S HIERARCHY OF NEEDS

Lower needs are prepotent: they must be reasonably met before higher ones motivate. The first four are deficiency needs; self-actualisation is a growth need (later: self-transcendence).

- **Maslow's hierarchy** – from the base up: **physiological → safety → love/belonging → esteem → self-actualisation**. The lower four are *deficiency* needs; the top is a *growth* need. Later writing added cognitive, aesthetic and self-transcendence tiers.

TRAP

Intrinsic motivation comes from within (interest, mastery); **extrinsic** from outside rewards. The **overjustification effect**: paying someone for an already-enjoyed activity can *reduce* intrinsic motivation. Self-determination theory predicts this: controlling rewards undercut autonomy.

6 Intelligence theories

The fault line: is intelligence **one** general capacity or **many** separate abilities. Theorists line up along it.

Theory	Structure	One vs many
Spearman two-factor	**g** (general) + **s** (task-specific). g underlies all; positive manifold	strongly one (g)
Thurstone	7 **primary mental abilities** (verbal, numerical, spatial, memory, reasoning, word fluency, perceptual speed)	several, no single g
Cattell-Horn	**fluid (Gf)** = reasoning on novel problems, peaks early, declines with age; **crystallised (Gc)** = stored knowledge, holds or rises	two broad factors
Gardner multiple intelligences	~8 independent intelligences (linguistic, logical-mathematical, spatial, musical, bodily-kinaesthetic, interpersonal, intrapersonal, naturalist)	many, autonomous
Sternberg triarchic	3: **analytical, creative, practical** (“street smarts”)	three (process-based)
Guilford	Structure of Intellect: up to 120+ factors (operations × contents × products); convergent vs divergent thinking	many

- **IQ concept** – originally mental age / chronological age × 100 (Stern; Binet's scale). Modern tests use a **deviation IQ**: normally distributed, **mean 100, SD 15** (Wechsler). So ~68% fall 85–115; <70 is the ID cut-off, >130 the gifted range.
- **Flynn effect** – population IQ rose roughly 3 points per decade through the 20th century (better nutrition, schooling, test familiarity), forcing periodic re-norming. Apparent slowing or reversal in some recent cohorts.

MNEMONIC

"Fluid Falls, Crystallised Climbs"

Fluid intelligence (Gf) declines with age; crystallised (Gc) is preserved – the basis of preserved vocabulary in early dementia screening.

7 Personality theories

Four broad approaches, each asking a different question: what are the dimensions (trait), the body types (type), the unconscious forces (psychodynamic), or the drive to grow (humanistic).

Approach	Asks	Key figures & claim
Trait	what stable dimensions describe a person?	measurable, continuous dimensions
Type	what discrete categories?	body build mapped to temperament
Psychodynamic	what unconscious conflict drives it?	id/ego/superego; early experience (Freud)
Humanistic	what pulls a person toward growth?	self-actualisation, the self-concept
Behavioural / social-cognitive	how do situation and learning shape it?	reciprocal determinism (Bandura); situationism (Mischel)
Personal construct	how does the person construe their world?	each of us a "scientist" testing constructs (Kelly)

The trait theorists (most tested)

- **Allport** – cardinal (dominates a life), central (5–10 core traits), and secondary (situational) traits. Founder of the trait approach.
- **Cattell** – factor-analysed surface traits down to **16 source traits**; the **16PF** questionnaire.
- **Eysenck** – biologically based dimensions: **Extraversion, Neuroticism**, later **Psychoticism** (the **PEN** / three-factor model). Extraversion tied to cortical arousal.
- **Big Five (OCEAN)** – the dominant modern model: **O**penness, **C**onscientiousness, **E**xtraversion, **A**greeableness, **N**euroticism. Replicates across cultures (the lexical hypothesis).

MNEMONIC

"OCEAN" → the Big Five

Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism. Eysenck = **PEN**; Cattell = **16**; Allport = cardinal/central/secondary.

The other approaches

- **Type theories** – **Sheldon**: endomorph (soft, sociable), mesomorph (muscular, assertive), ectomorph (lean, restrained). **Kretschmer**: pyknic build linked to manic-depressive, asthenic/leptosomic to schizophrenia. Largely discredited but examinable.
- **Humanistic** – **Rogers**: the self-concept, conditions of worth, and the gap between real and ideal self; growth needs unconditional positive regard. **Maslow**: personality as the climb toward self-actualisation.
- **Social-cognitive** – **Bandura**: reciprocal determinism (person, behaviour and environment interact) and self-efficacy. **Mischel**: the person-situation debate – behaviour is less consistent across situations than trait theory assumes (the consistency paradox).
- **Kelly's personal construct theory** – people are intuitive scientists who anticipate events through bipolar **personal constructs**; assessed by the **repertory grid**.

● RULE

When a question says “trait theories,” line up **Allport → Cattell (16PF) → Eysenck (PEN) → Big Five (OCEAN)** in that order. Note the trend: many surface traits collapsing into a few robust, biologically grounded dimensions.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Emotion:** James-Lange = body first · Cannon-Bard = simultaneous · Schachter-Singer = arousal + label · Lazarus = appraisal first. LeDoux low road (fast, subcortical) vs high road (slow, cortical).
- **Perception:** Gestalt = proximity, similarity, closure, continuity, figure-ground, common fate (master law: Pragnanz). Binocular = disparity + convergence; rest monocular. d′ = sensitivity, β = criterion.
- **Conditioning:** classical = stimuli (Pavlov); operant = consequences (Skinner). Negative reinforcement ≠ punishment: it removes an aversive to increase behaviour.
- **Schedules: variable ratio** = highest rate, most resistant to extinction (gambling). Ratio > interval for rate; variable > fixed for persistence.
- **Learning, beyond S-R:** Bandura (Bobo, observational) · Tolman (latent, cognitive map) · Kohler (insight) · Garcia (one-trial taste aversion, preparedness).
- **Memory:** Atkinson-Shiffrin multi-store · Baddeley working memory (central executive + phonological loop + visuospatial sketchpad + episodic buffer) · Craik-Lockhart depth. STM = 7 ± 2. Declarative (episodic + semantic) vs procedural; H.M. dissociates them.
- **Forgetting:** proactive = old blocks new; retroactive = new overwrites old. Ebbinghaus curve, flattened by spacing. LTP = NMDA-dependent.
- **Motivation:** Hull (drive reduction) · Yerkes-Dodson inverted U (optimum lower for hard tasks) · Maslow ladder · Deci-Ryan (autonomy, competence, relatedness) · McClelland (achievement, affiliation, power).
- **Intelligence:** Spearman g+s · Thurstone 7 PMAs · Cattell-Horn fluid (falls) vs crystallised (climbs) · Gardner ~8 · Sternberg triarchic (analytical/creative/practical) · Guilford 120+. IQ mean 100, SD 15. Flynn effect ~3 pts/decade.
- **Personality traits:** Allport (cardinal/central/secondary) → Cattell 16PF → Eysenck PEN → Big Five OCEAN. Sheldon/Kretschmer = type. Rogers/Maslow = humanistic. Bandura/Mischel = social-cognitive. Kelly = personal constructs.

ONE-DAY REVISION · PAPER 1

Developmental psychology, made retrievable

The lifespan stage models: how thought, self, morality, attachment and language unfold with age. The marks live in the stage tables. Hold the sequence, the one idea that defines each stage, and the name attached to it. Learn the tables cold and most of this topic answers itself.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The conceptual theories (emotion, memory, learning, personality) live in the companion Psychology pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Classic swap: **object permanence** is a sensorimotor gain (object still exists when hidden, ~8 mo), whereas **conservation** (quantity unchanged despite a change in appearance) is acquired in concrete operational (~7 yr); examiners deliberately interchange them.
2. **Piaget = solo construction** (development → learning, individual equilibration); **Vygotsky = social construction** (learning → development via the ZPD, the gap between what a child does alone versus with guided help); that one contrast answers most comparison questions.
3. Erikson is the only stage theory spanning the **whole lifespan**: 8 crises → virtues (Hope, Will, Purpose, Competence, Fidelity, Love, Care, Wisdom), with identity vs role confusion (adolescence) as the signature stage; a named virtue or lifespan "vs" pair = Erikson, an erogenous zone or fixation = Freud.
4. Strange Situation is classified by **reunion** behaviour: secure (B, ~65%) settles; avoidant (A, ~20%) *ignores* the parent; ambivalent/resistant (C, ~10%) *clings and resists at once*; disorganised (D, ~5%) is tied to maltreatment and is the strongest predictor of later psychopathology.
5. Kohlberg scores the reasoning, not the verdict: in the Heinz dilemma the same "steal / don't steal" act maps to different stages ("he'll be jailed" = stage 1, "the law is the law" = stage 4, "a life outweighs property" = stage 5/6); Gilligan added an ethic of **care** alongside justice.

1 Piaget: cognitive development

The child is a little scientist who actively builds knowledge. Intelligence develops through four **invariant, universal** stages; each is qualitatively different, not just "more" of the last. The order never changes; the ages are approximate.

The engine: how knowledge is built

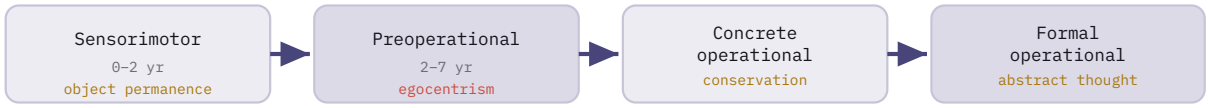
- **Schema** – a mental template for acting on or understanding the world (the "grasping" schema, the "dog" schema). The building block of thought.
- **Assimilation** – fitting new experience into an *existing* schema (a child calls a cat "doggie").
- **Accommodation** – *changing* the schema to fit new experience (learning that cats are a separate category).

■ **Equilibration** – the drive to resolve the *disequilibrium* between assimilation and accommodation. The motor that pushes the child from one stage to the next.

The four stages

Stage	Age	Defining achievement	Hallmark limitation
Sensorimotor	0–2 yr	knows the world through senses & movement; gains **object permanence** (objects exist when out of sight, ~8 mo); begins deferred imitation & symbolic thought	early on, "out of sight, out of mind"; **A-not-B error**
Preoperational	2–7 yr	symbolic thinking, language, pretend play; uses mental representations	**egocentrism, animism, centration; cannot conserve**; no reversibility
Concrete operational	7–11 yr	**conservation, reversibility, seriation, classification**; logical operations on concrete objects; decentration	logic tied to the concrete & present; struggles with abstract/hypothetical
Formal operational	11+ yr	**abstract & hypothetical-deductive** reasoning; systematic problem-solving; thinks about thinking	adolescent egocentrism (imaginary audience, personal fable); not universally attained

PIAGET'S FOUR STAGES OF COGNITIVE DEVELOPMENT



Invariant order, universal sequence; ages approximate. Each stage is qualitatively distinct.

Sensorimotor (object permanence) → preoperational (symbols, but egocentric, no conservation) → concrete operational (conservation, reversibility, seriation) → formal operational (abstract, hypothetical reasoning).

Preoperational limitations (high-yield)

▲ **Egocentrism** – cannot take another's perspective. Shown by the **three-mountains task**: the child reports the view as their own, not the doll's.

▲ **Animism** – attributes life and feelings to inanimate objects ("the sun is happy").

▲ **Centration** – focusing on one salient feature (height of liquid) while ignoring others (width), which blocks conservation.

◆ **Conservation** – understanding that quantity stays constant despite a change in appearance. Acquired in **concrete operational**. Order: number (~6) → mass/liquid (~7) → weight (~9) → volume (~11). The classic test: two equal glasses, pour one into a taller glass; the preoperational child says the tall one has "more."

● CLASSIC TRAP

Object permanence belongs to the **sensorimotor** stage; **conservation** belongs to **concrete operational**. Examiners swap them. Object permanence = the object still *exists* when hidden. Conservation = the *quantity* is unchanged when the shape changes.

Critiques of Piaget

- **Underestimated children** – simplified tasks reveal competence earlier. Object permanence shows by ~3.5 mo on violation-of-expectation tasks (Baillargeon); egocentrism eases with the "naughty teddy" / policeman-doll task (Hughes).
- **Underplayed the social world** – Vygotsky argued culture, language and tutoring drive cognition; Piaget treated the child as a lone explorer.
- **Stages are not so rigid** – development is more continuous and domain-specific than discrete; many adults never reach full formal operations.

2 Vygotsky: sociocultural cognition

Where Piaget made the child a solitary scientist, Vygotsky made cognition **social first, then internal**. Higher mental functions begin between people and are *internalised*. Culture and language are the tools of thought.

- **Zone of proximal development (ZPD)** – the gap between what a learner can do **alone** and what they can do **with guided help**. Learning happens here, just ahead of the current level. The single most tested Vygotsky term.
- **Scaffolding** – the temporary, tailored support a tutor gives within the ZPD, withdrawn as competence grows (term from Wood, Bruner & Ross, built on Vygotsky).
- **More knowledgeable other (MKO)** – anyone (parent, teacher, peer) with greater skill who guides the learner through the ZPD.
- **Private speech** – children talk aloud to guide themselves; this *self-directed* speech is internalised into silent **inner speech** that regulates thought. For Vygotsky a sign of progress, not (as Piaget held) immature egocentric speech.
- **Social constructivism** – knowledge is co-constructed through social interaction and mediated by cultural tools (above all, language).

	Piaget	Vygotsky
Engine of development	individual exploration, equilibration	social interaction, culture, language
Direction	development drives (precedes) learning	learning leads (pulls) development
Stages	universal, fixed sequence	no fixed universal stages; culturally shaped
Role of language	reflects thought (follows it)	shapes & drives thought (the key tool)
Private speech	egocentric, immature	self-guiding; becomes inner speech

HOLD THIS

Piaget = solo construction (development → learning). **Vygotsky = social construction** (learning → development, via the ZPD and language). That single contrast is the answer to most comparison questions.

3 Erikson: psychosocial development

Erikson took Freud's stages and extended them across the **whole lifespan**. Each stage poses a **psychosocial crisis**; resolving it well yields a **virtue** (basic strength). The stages are *epigenetic*: they unfold in a set order, each building on the last.

#	Crisis	Age	Virtue	Favourable outcome
1	Trust vs mistrust	0–1 yr	Hope	secure, the world is dependable
2	Autonomy vs shame & doubt	1–3 yr	Will	self-control, independence

#	Crisis	Age	Virtue	Favourable outcome
3	Initiative vs guilt	3–6 yr	Purpose	initiates tasks, plans, leads in play
4	Industry vs inferiority	6–12 yr	Competence	mastery of skills, sense of competence
5	Identity vs role confusion	12–18 yr	Fidelity	coherent sense of self & values
6	Intimacy vs isolation	young adult	Love	deep, committed relationships
7	Generativity vs stagnation	middle adult	Care	guiding the next generation, productivity
8	Integrity vs despair	late adult	Wisdom	acceptance of one's life, no regret

● MNEMONIC

Virtues: "Hope, Will, Purpose, Competence · Fidelity, Love, Care, Wisdom"

First four = childhood; last four = adolescence onward. Identity vs role confusion (adolescence) is the pivot and Erikson's signature stage.

Cross-reference: Freud's psychosexual stages

Erikson's stages 1–5 map onto Freud's five psychosexual stages, but Erikson stressed the *social* task where Freud stressed the *libidinal* zone. For Freud, frustration or over-indulgence at a stage causes **fixation**, leaving a lasting character imprint.

Stage	Age	Erogenous focus	Fixation theme
Oral	0–1 yr	mouth (sucking, biting)	dependency, gullibility; oral character (smoking, overeating)
Anal	1–3 yr	bowel/bladder control	anal-retentive (orderly, stingy) vs anal-expulsive (messy)
Phallic	3–6 yr	genitals; **Oedipus/Electra** complex	superego forms via identification; vanity, recklessness
Latency	6–12 yr	dormant; sublimation into skills/peers	(no fixation; consolidation)
Genital	puberty+	mature sexual intimacy	healthy adult sexuality if earlier stages resolved

● RULE

If a question names a **virtue** (hope, will, fidelity, wisdom) or runs "vs" pairs across the whole lifespan, it is **Erikson**. If it names an **erogenous zone** or **fixation** (oral, anal, Oedipus), it is **Freud**. Both are stage theories; only Erikson spans all of adult life.

4 Kohlberg: moral development

Kohlberg extended Piaget into the moral domain using moral dilemmas (the **Heinz dilemma**). What matters is the **reasoning** behind the judgment, not the verdict. Three levels, two stages each.

Level	Stage	What drives the moral choice
Pre-conventional (self-interest; pre-teen)	1. Obedience & punishment	avoid punishment; defer to power
	2. Instrumental / self-interest	"what's in it for me?" simple exchange
Conventional (social approval; most adults)	3. Good boy / good girl	seek approval; be "nice," meet others' expectations
	4. Law & order	obey rules, maintain social order & duty
Post-conventional (abstract principle; a minority)	5. Social contract	laws are mutable agreements for the common good
	6. Universal ethical principles	self-chosen principles (justice, dignity) override law

CLASSIC TRAP

The Heinz dilemma: a man steals an overpriced drug to save his dying wife. Kohlberg scored the **justification**, not the "steal / don't steal" answer. "He'll be jailed" = stage 1; "the law is the law" = stage 4; "a life outweighs property rights" = stage 5/6. Same action, very different stage.

The two major critiques

- **Gilligan: care vs justice** – Kohlberg's scale, built on male samples, privileges an abstract **justice/rights** orientation (typed as "male") and scores a relational **care/responsibility** orientation (more common in women) as lower. She proposed a separate **ethic of care**, not a deficit.
- **Cultural & other limits** – the post-conventional level reflects Western individualist values; reasoning does not always predict moral *action*; stage 6 is rare and was later dropped from scoring.

Piaget's earlier moral theory

- **Moral realism (heteronomous)** – ~5–9 yr: rules are fixed, handed down by authority and unchangeable. Judges by **consequences** (15 broken cups is "naughtier" than 1, regardless of intent). Believes in **immanent justice** (wrongdoing is automatically punished).
- **Moral relativism (autonomous)** – ~10 yr+: rules are social agreements that can be changed by consent. Judges by **intention**, not outcome. The mature stage.

QUICK RULE

Piaget: two moral stages, judged by **consequences → intentions**. Kohlberg: three levels / six stages, scored on **reasoning**. Gilligan: adds the **ethic of care** alongside justice.

5 Attachment & temperament

Bowlby: attachment theory

- **Attachment behavioural system** – an innate, **evolved** system that keeps the infant close to a caregiver for protection. Activated by threat, fatigue or separation; the caregiver is a **secure base** for exploration and a **safe haven** in distress.
- **Monotropy** – the infant is biased to form one primary attachment (usually the mother), which is qualitatively special and the template for later bonds.
- **Internal working model (IWM)** – a mental template of self, others and relationships built from early attachment; guides expectations in later relationships.
- **Maternal deprivation hypothesis** – prolonged disruption of the primary bond in a **critical period** (roughly the first 2–3 yr) risks lasting harm: Bowlby linked it to **affectionless psychopathy** (his 44 thieves study). Later work softened this to *privation* vs deprivation, with more reversibility than he first claimed.

Ainsworth: the Strange Situation

A standardised lab procedure (separations and reunions with caregiver and stranger). The **reunion** behaviour classifies the attachment.

Type	Separation	Reunion	Caregiver style
Secure (B, ~65%)	distressed	seeks comfort, settles, returns to play	sensitive, responsive
Insecure-avoidant (A, ~20%)	little distress	avoids/ignores caregiver	rejecting, unresponsive

Type	Separation	Reunion	Caregiver style
Insecure-ambivalent / resistant (C, ~10%)	very distressed	seeks contact *and* resists it; not soothed	inconsistent, unpredictable
Disorganised (D, ~5%)	confused, contradictory	freezing, odd/fearful approach	frightening/frightened; abuse, loss (Main & Solomon, added later)

● CLASSIC TRAP

Do not confuse **avoidant** with **ambivalent/resistant**. Avoidant = under-reacts, *ignores* the parent on reunion. Ambivalent = over-reacts, *clings and resists* at once, cannot be soothed. **Disorganised** (D) is the type tied to maltreatment and the strongest predictor of later psychopathology.

Harlow & Thomas-Chess (cross-refs)

- **Harlow's monkeys** – infant rhesus monkeys preferred a soft **cloth surrogate** over a wire one that fed them, clinging to it when frightened. **Contact comfort**, not feeding, drives attachment, refuting the cupboard-love (drive-reduction) theory.
- **Lorenz (cross-ref)** – **imprinting** in goslings during a critical period: rapid, irreversible bonding to the first moving object. Evidence that attachment has an innate, time-sensitive basis.
- **Thomas & Chess: temperament** – the New York Longitudinal Study found three constitutional styles: **easy** (~40%: regular, adaptable, positive), **difficult** (~10%: irregular, intense, withdrawing), **slow-to-warm-up** (~15%: low activity, slow to adapt, mildly negative). The rest are mixed/unclassified.
- **Goodness of fit** – outcomes depend not on temperament alone but on the **match** between the child's temperament and the demands and style of the environment. A difficult child with a patient, flexible parent can do well; a poor fit breeds conflict.

6 Language & developmental milestones

Stages of language acquisition

Stage	Typical age	What it sounds like
Cooing	~2–4 mo	vowel-like sounds ("ooo," "aah")
Babbling	~6 mo	repeated consonant-vowel strings ("bababa"); universal at first, then tuned to the native language
Holophrases (one-word)	~12 mo	single words carry a whole idea ("milk!" = "I want milk")
Telegraphic speech	~18–24 mo	two-word combos, content words only ("want cookie," "mummy go")

The nature–nurture debate on language

	Chomsky (nativist)	Skinner (behaviourist)
Mechanism	innate **language acquisition device (LAD)**; a universal grammar	operant conditioning: imitation, reinforcement, shaping
Key evidence	**poverty of the stimulus**; children produce novel sentences & regular errors ("goed," "mouses")	children learn the specific language they are exposed to
Timing	**critical / sensitive period** for acquisition (e.g. the case of Genie)	no special biological window

● RULE

Chomsky's strongest argument is the **poverty of the stimulus**: children master complex grammar far faster, and more uniformly, than imitation-plus-reinforcement could explain, and they make systematic *over-regularisation* errors they never heard. Skinner cannot account for novel, rule-governed output. The modern view is interactionist (nature provides the capacity; input shapes the specifics).

High-yield developmental milestones

DEVELOPMENTAL MILESTONES

	MOTOR			
Age	**Gross motor**	**Fine motor**	**Language**	**Social**
2 mo	lifts head when prone	follows to midline	cooing	social smile
4–6 mo	rolls over; sits with support	reaches; palmar grasp	babbling begins	laughs; recognises caregiver
9–10 mo	sits unsupported; crawls; pulls to stand	immature pincer grasp	"mama/dada" (non-specific)	stranger anxiety; plays peek-a-boo
12 mo	stands alone; first steps	mature pincer grasp	1–3 words; "mama/dada" specific	waves bye; separation anxiety
18 mo	walks well; runs stiffly	tower of 3–4 cubes; scribbles	~10–25 words; points to body parts	imitates; feeds self with spoon
2 yr	runs; walks up/down stairs	tower of 6 cubes; copies a line	**2-word phrases**; ~50+ words; half understandable	parallel play
3 yr	rides tricycle; climbs stairs alternating feet	copies a circle; tower of 9	**3-word sentences**; speech mostly understood	knows name & sex; shares
4 yr	hops on one foot	copies a cross; draws a person (3 parts)	tells stories; counts to 4	cooperative play; toilet trained
5 yr	skips; alternates feet	copies a square & triangle; draws person (6 parts)	fluent; counts to 10; asks meanings	plays games with rules; dresses self

RED-FLAG ANCHORS

No **social smile by 3 mo** · not **sitting unsupported by 9 mo** · not **walking by 18 mo** · **no single words by 16–18 mo** · **no two-word phrases by 24 mo**. Any of these warrants developmental assessment.

● NUMBERS TO LOCK

Social smile **2 mo** · sits unsupported **~6–9 mo** · stranger anxiety **~8–9 mo** · first words / first steps **~12 mo** · two-word phrases **~2 yr** · three-word sentences **~3 yr** · rides tricycle **3 yr** · copies a cross **4 yr** · copies a square **~4–5 yr**. (Ranges are approximate; vary by source.)

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Piaget stages:** Sensorimotor (0–2, object permanence) → Preoperational (2–7, egocentrism/animism, no conservation) → Concrete operational (7–11, conservation, reversibility, seriation) → Formal operational (11+, abstract/hypothetical).

★ **Piaget engine:** schema · assimilation (fit in) · accommodation (change schema) · equilibration (the motor). Trap: object permanence (sensorimotor) ≠ conservation (concrete operational).

- **Vygotsky:** ZPD = alone vs with help; scaffolding; more knowledgeable other; private speech → inner speech; social constructivism. Piaget solo (development → learning); Vygotsky social (learning → development).
- **Erikson (8 crises / virtues):** Trust/Hope · Autonomy/Will · Initiative/Purpose · Industry/Competence · Identity/Fidelity · Intimacy/Love · Generativity/Care · Integrity/Wisdom. Signature stage: identity vs role confusion (adolescence).
- **Freud psychosexual:** Oral → Anal → Phallic (Oedipus, superego) → Latency → Genital. Frustration/over-indulgence = fixation. Erikson = social task; Freud = libidinal zone.
- **Kohlberg:** Pre-conventional (punishment, self-interest) → Conventional (approval, law & order) → Post-conventional (social contract, universal principles). Heinz dilemma scores the *reasoning*. Gilligan = ethic of care vs justice.
- **Piaget moral:** moral realism (consequences, immanent justice) → moral relativism (intentions). Judge by outcome → judge by intent.
- **Bowlby:** evolved attachment system, monotropy, internal working model, maternal deprivation in a critical period (44 thieves → affectionless psychopathy). Harlow: contact comfort > feeding. Lorenz: imprinting.
- **Strange Situation:** Secure (B, ~65%) · insecure-avoidant (A, ignores) · insecure-ambivalent/resistant (C, clings + resists) · disorganised (D, maltreatment, worst outcomes). Classification is by reunion behaviour.
- **Temperament (Thomas & Chess):** easy · difficult · slow-to-warm-up. Outcome = **goodness of fit** between child and environment.
- **Language:** cooing (~2–4 mo) → babbling (~6 mo) → holophrases (~12 mo) → telegraphic (~18–24 mo). Chomsky LAD + critical period + poverty of the stimulus vs Skinner reinforcement.
- **Milestones:** social smile 2 mo · sits unsupported ~6–9 mo · first words/steps ~12 mo · two words ~2 yr · three-word sentences + tricycle 3 yr · copies cross 4 yr.

ONE-DAY REVISION · PAPER 1

Social Psychology

The science of how the actual, imagined or implied presence of others shapes thought, feeling and behaviour. The classic experiments carry the marks: hold the name, the manipulation and the one finding each. Attitudes and attribution supply the theory; conformity, obedience and the group studies supply the drama.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The landmark-experiments table in section 3 is the highest-yield single object in this pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The landmark six by name and finding: Asch = conformity (lines, ~75% conform once / ~37% of trials), Milgram = obedience (65% to 450 V), Zimbardo = roles (prison), Sherif = realistic group conflict (Robbers Cave), Latane & Darley = bystander (diffusion of responsibility), Festinger & Carlsmith = dissonance.
2. Festinger & Carlsmith (1959): the $1 group changed attitude more than the $20 group, because too little external reward forces dissonance reduction; insufficient justification is the engine, and bigger reward means no attitude change.
3. Distinguish the attribution biases: fundamental attribution error = over-attribute others' behaviour to disposition and ignore the situation; self-serving bias = success internal, failure external; FAE is weaker in collectivist cultures.
4. LaPiere (1934) is the founding demonstration of the attitude-behaviour gap (over 90% later said they would refuse Chinese guests despite serving them); attitudes predict behaviour only when strong, specific, accessible, and matched at the same level of specificity (principle of compatibility).
5. Classic discriminators to hold: Asch = normative influence vs Sherif autokinetic = informational; one dissenting ally breaks Asch conformity; social facilitation (others present, performance changes) is not social loafing (effort drops when output is unidentifiable); Allport contact hypothesis needs equal status, common goals, cooperation, and authority support.

1 Attitudes

An attitude is a learned, relatively enduring evaluation of an object, person or idea. The structure, how it forms, why it predicts behaviour poorly, and how it changes are four separate questions.

The ABC structure

Component	What it is	Example (toward smoking)
Affective	feelings, emotional response	“smoking disgusts me”
Behavioural	past actions and intentions	“I avoid smoky rooms”
Cognitive	beliefs and thoughts	“smoking causes cancer”

● MNEMONIC

"ABC" → Affect, Behaviour, Cognition

The three-component (tripartite) model of attitude structure. Affect = feeling, Behaviour = action tendency, Cognition = belief.

How attitudes form

- **Learning** – classical conditioning (pairing an object with affect), operant conditioning (reward for expressing a view), and observational learning (modelling parents, peers, media).
- **Mere exposure** – repeated exposure to a neutral stimulus increases liking for it (Zajonc). Familiarity alone breeds preference.
- **Functional approach** – attitudes serve needs: knowledge, utilitarian (reward/punishment), ego-defensive, and value-expressive functions (Katz).

The attitude-behaviour gap

▲ **LaPiere (1934)** – travelled the US with a Chinese couple; served at all but one of ~250 establishments, yet later **over 90%** said in writing they would refuse Chinese guests. Stated attitudes predicted actual behaviour poorly. The founding demonstration of the gap.

■ **When attitudes do predict behaviour** – when they are strong, specific to the behaviour, accessible, formed by direct experience, and when situational pressure is low. The **principle of compatibility**: measure attitude and behaviour at the same level of specificity.

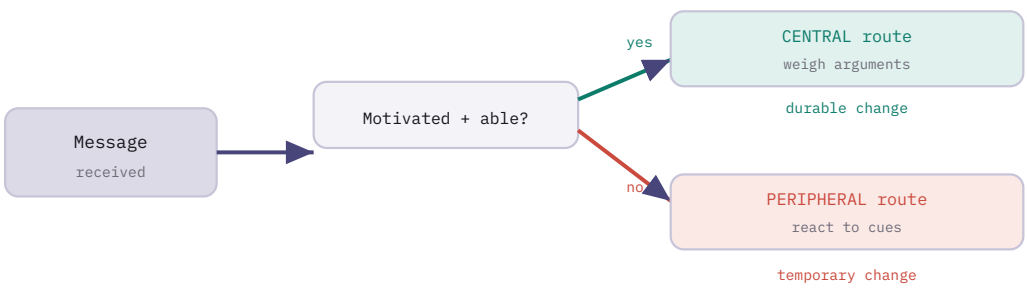
- **Theory of planned behaviour (Ajzen)** – intention (the best predictor of behaviour) is driven by attitude + subjective norms + perceived behavioural control. Extends the earlier theory of reasoned action (Fishbein & Ajzen).

Persuasion: the elaboration likelihood model (Petty & Cacioppo)

Persuasion takes one of two routes, depending on the listener's motivation and ability to think the message through (their *elaboration*).

	Central route	Peripheral route
Processing	high elaboration, effortful	low elaboration, superficial
Persuaded by	strength & quality of arguments	cues: source attractiveness, credibility, number of arguments
Needs	motivation + ability to process	used when motivation or ability is low
Resulting change	durable, resists counter-argument	temporary, easily shifted

TWO ROUTES TO PERSUASION (ELABORATION LIKELIHOOD MODEL)



When the listener is motivated and able, persuasion runs through the central route (argument quality, durable). When not, it runs through the peripheral route (cues such as source appeal, temporary).

Cognitive dissonance (Festinger, 1957)

◆ **Cognitive dissonance** – holding two inconsistent cognitions, or acting against an attitude, creates an uncomfortable tension drive. We reduce it by **changing the attitude**, adding consonant cognitions, or trivialising the conflict. We change the easier element.

● CLASSIC STUDY

Festinger & Carlsmith (1959): subjects did a boring task, then were paid **$1 or $20** to tell the next person it was fun. The **$1** group later rated the task as *more* enjoyable. With too little external justification to explain the lie, they reduced dissonance by genuinely changing their attitude. Larger reward = ample justification = no dissonance = no attitude change. **Insufficient justification** is the engine.

2 Social cognition & attribution

How we make sense of the social world: explaining causes (attribution), the shortcuts we use (heuristics, schemas) and the systematic errors that follow.

Attribution theory

● **Heider (1958)** – founder; people are "naive scientists" who attribute behaviour either to **internal/dispositional** causes (the person) or **external/situational** causes (the circumstances).

● **Kelley's covariation model** – to locate a cause we weigh three kinds of information across observations: **consensus** (do others act this way?), **distinctiveness** (only to this stimulus?) and **consistency** (every time?). High consistency + high consensus + high distinctiveness → external attribution; high consistency + low consensus + low distinctiveness → internal.

● **Weiner's model** – causal attributions vary on three dimensions: **locus** (internal/external), **stability** (stable/unstable) and **controllability**. Shapes emotion and expectancy after success or failure (relevant to learned helplessness).

Attributional biases

Bias	The error
Fundamental attribution error	over-attribute others' behaviour to disposition, under-weight the situation (Ross). The master bias.
Actor-observer bias	my behaviour = situational ("I tripped, the floor was wet"); your behaviour = dispositional ("you tripped, you're clumsy")
Self-serving bias	credit success to self (internal), blame failure on circumstance (external). Protects self-esteem.
Just-world hypothesis	belief that people get what they deserve; underlies victim-blaming (Lerner)

● CLASSIC TRAP

The **fundamental attribution error** is about explaining *others* with disposition while ignoring the situation. The **self-serving bias** is about protecting one's *own* esteem (success internal, failure external). The **actor-observer** difference partly reflects perceptual focus (the actor sees the situation; the observer sees the actor). The FAE is weaker in collectivist cultures, which attend more to context.

Heuristics and schemas

● **Availability heuristic** – judge likelihood by how easily examples come to mind (plane crashes feel common because they are memorable) (Tversky & Kahneman).

- **Representativeness heuristic** – judge category membership by similarity to a prototype, ignoring base rates (the conjunction fallacy).
- **Anchoring** – estimates stay tethered to an initial value, adjusting insufficiently away from it.
- **Schema** – an organised knowledge structure that guides perception and memory. **Scripts** are event schemas; **stereotypes** are schemas about groups. Schemas speed processing but bias toward confirming information.

Self-concept and self-perception

- **Self-concept** – the totality of beliefs about oneself, organised into self-schemas. Built partly through **social comparison** (Festinger): we evaluate ourselves against others.
- **Self-perception theory (Bem)** – we infer our own attitudes by observing our behaviour, much as we infer others' ("I eat brown bread, so I must like it"). A rival to dissonance for explaining attitude change without internal tension.

3 Social influence & the landmark experiments

The single highest-yield object in this pack. Learn each row as name · manipulation · finding.

Study	What was manipulated	The one finding
Asch (1951) conformity	confederates give an obviously wrong answer on a line-judgement task	~75% conformed at least once; ~37% of critical trials conformed. **Normative** influence.
Milgram (1963) obedience	an authority orders delivery of escalating "shocks" to a learner	65% went to the maximum 450 V. Obedience to authority, not cruelty.
Zimbardo (1971) Stanford prison	students randomly assigned to guard or prisoner roles	guards turned abusive, prisoners broke down; stopped after 6 days. Power of assigned roles.
Sherif (1954) Robbers Cave	boys' camp split into two groups, then placed in competition	competition bred hostility; only **superordinate goals** reduced it. Realistic conflict.
Latane & Darley (1968) bystander	group size during a (staged) emergency	more bystanders → *less* and slower helping. Diffusion of responsibility.
Festinger & Carlsmith (1959)	$1 vs $20 to lie that a dull task was fun	the **$1** group changed attitude more. Cognitive dissonance via insufficient justification.

HOLD THIS

Asch = **conformity** (lines). Milgram = **obedience** (shocks, 65%). Zimbardo = **roles** (prison). Sherif = **group conflict** (Robbers Cave). Latane-Darley = **bystander**. Festinger = **dissonance**. Six names, six findings.

Conformity (Asch)

- **Normative influence** – conform to be liked and accepted, to avoid standing out. Produces public compliance without private acceptance. Drives the Asch effect.
- **Informational influence** – conform because others are taken as a source of truth, especially in ambiguous situations. Produces genuine private acceptance (Sherif's autokinetic study).
- **Asch variables** – conformity falls sharply with even **one dissenting ally**, rises with group size up to ~3–4 (then plateaus), and falls when answers are private.

Compliance techniques

Technique	How it works
Foot-in-the-door	small request first; agreeing makes the later large request more likely (consistency, self-perception)
Door-in-the-face	large request first (refused); the smaller real request then seems reasonable (reciprocal concession)

Technique	How it works
Low-balling	secure agreement, then raise the cost; commitment holds
That's-not-all	add a bonus before the person decides; obligation to reciprocate

Obedience (Milgram)

- **Factors raising obedience** – close authority figure, prestigious setting, gradual escalation, remote victim, and the **agentic state** (seeing oneself as the authority's instrument, not the author of the act).
- **Factors lowering it** – authority absent or in conflict, victim close/visible, a disobedient peer model. Obedience fell from 65% toward ~10% in these conditions.

The bystander effect (Latane & Darley)

- **Diffusion of responsibility** – the more witnesses present, the less any one person feels personally responsible to act. Helping drops as group size rises.
- **Pluralistic ignorance** – each bystander reads others' inaction as a sign that nothing is wrong, so all stay passive (the smoke-filled-room study).
- **Five-step decision model** – to help, a bystander must: notice the event → interpret it as an emergency → assume responsibility → know how to help → decide to act. Failure at any step stops helping. Prompted by the **Kitty Genovese** case.

Presence of others

- **Social facilitation** – the presence of others **improves** performance on simple or well-learned tasks but **impairs** it on difficult or novel ones (Zajonc: presence raises arousal, which strengthens the dominant response).
- **Social loafing** – individuals exert *less* effort when working in a group on a collective task, because individual output is not identifiable (Ringelmann effect; the rope-pulling demonstration).
- **Deindividuation** – in a crowd, anonymity and reduced self-awareness loosen normal restraints, allowing impulsive, antinormative behaviour (mobs, uniforms, online disinhibition).

TRAP

Social facilitation (others present while you work alone, performance changes) is not **social loafing** (others share the task, your effort drops). Facilitation can *help* on easy tasks; loafing always reduces individual effort and is fixed by making contributions identifiable.

4 Group processes

- **Group polarisation** – group discussion **strengthens** the members' pre-existing average leaning; the group decision is more extreme than the mean of individual views. Includes the older "risky shift."
- **Groupthink (Janis, 1972)** – cohesive, insulated groups under directive leadership suppress dissent to preserve harmony, producing poor decisions. Symptoms: illusion of invulnerability, collective rationalisation, pressure on dissenters, self-censorship, illusion of unanimity, "mindguards." Cases: Bay of Pigs, Challenger.
- **Zimbardo Stanford prison study (1971)** – randomly assigned roles, not personality, drove the abuse; the situation overwhelmed the person. The strongest demonstration of **conformity to assigned social roles**. Now critiqued for demand characteristics and coaching of guards.

Leadership styles (Lewin)

Style	Character	Group effect
Autocratic	leader decides alone	high output, low morale; aggression when leader absent
Democratic	decisions shared	best morale & quality; output holds when leader leaves
Laissez-faire	leader hands off	least productive, least satisfied

● **Cooperation vs competition** – competition (a zero-sum frame) breeds hostility and distrust; cooperation toward shared ends builds liking. Demonstrated by Sherif's Robbers Cave and by social-dilemma games (the prisoner's dilemma, tragedy of the commons).

5 Intergroup relations

Prejudice, stereotyping, discrimination

● **The three terms** – **stereotype** = the cognitive belief about a group; **prejudice** = the affective evaluation (usually negative attitude); **discrimination** = the behaviour (unequal treatment). Map them onto the ABC of attitudes.

● MNEMONIC

"Stereotype thinks, prejudice feels, discrimination acts"

Cognitive → Affective → Behavioural. The same ABC structure as any attitude, applied to groups.

Theories of intergroup bias

Theory	Core claim	Author
Social identity theory	part of self-esteem comes from group membership, so we favour the in-group and derogate the out-group, even when groups are arbitrary (the **minimal group** paradigm)	Tajfel & Turner
Realistic conflict theory	prejudice arises from real competition over scarce resources; shared superordinate goals reduce it	Sherif
Authoritarian personality	a rigid, submissive-to-authority personality type is prone to prejudice (the F-scale)	Adorno
Scapegoat theory	frustration is displaced onto a weaker, visible out-group blamed for hardship	(frustration-aggression root)

◆ **Minimal group paradigm (Tajfel)** – assigning people to groups on a trivial basis (even a coin toss, or preferring one painter) is enough to produce **in-group favouritism** in reward allocation. Mere categorisation, with no competition or contact, generates bias.

Robbers Cave (Sherif, 1954)

● **The three phases** – (1) in-group formation (two boys' groups bond separately); (2) friction (competition for prizes breeds hostility); (3) integration (contact alone failed; only **superordinate goals** needing both groups, such as fixing the water supply, restored harmony).

Reducing prejudice

■ **Contact hypothesis (Allport, 1954)** – contact reduces prejudice only under conditions: **equal status**, common goals, intergroup cooperation, and institutional/authority support. Mere contact is insufficient and can backfire.

● **Jigsaw classroom (Aronson)** – cooperative learning in which each pupil holds one piece every other needs, forcing interdependence. An applied use of the contact and superordinate-goal principles to cut prejudice.

6 Prosocial & antisocial behaviour

Helping and altruism

- **Altruism** – helping that benefits another at a cost to the self, with no expectation of reward. Distinguished from egoistic helping.
- **Empathy-altruism hypothesis (Batson)** – genuine empathy for another produces truly altruistic motivation, not merely relief of one's own distress.
- **Other accounts** – **negative-state relief** (help to dispel one's own bad mood); **social-exchange** and **reciprocity** norms; **kin selection** (evolutionary, favouring relatives). Set against bystander apathy (section 3).

Aggression

- **Frustration-aggression hypothesis** – frustration (a blocked goal) creates a readiness to aggress (Dollard et al., 1939). Revised by **Berkowitz**: frustration produces anger/negative affect, and aggression follows especially when **aggressive cues** are present (the weapons effect).
- **Social learning of aggression (Bandura)** – aggression is learned by observing and imitating models. The **Bobo doll** study: children who watched an adult attack the doll reproduced the aggression, more so when the model was rewarded (vicarious reinforcement). *Cross-reference: learning theories pack, observational learning.*
- **Other roots** – biological (testosterone, amygdala, low serotonin), the **general aggression model**, and situational primers (heat, alcohol, deindividuation).

Interpersonal attraction

Factor	Why it draws us
Proximity	physical nearness raises contact and familiarity (Festinger's student-housing study)
Mere exposure	repeated exposure alone increases liking (Zajonc)
Similarity	shared attitudes, values and background; the matching hypothesis for looks
Reciprocity	we like those who like us (reciprocal liking)
Physical attractiveness	the "**what is beautiful is good**" halo stereotype

TRAP

Proximity and **mere exposure** are linked but distinct: proximity is the situational factor (being near someone), mere exposure is the mechanism (repeated contact breeds liking). Note the contrast with "familiarity breeds contempt": in attraction research, familiarity usually breeds *liking*.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Landmark six:** Asch = conformity (lines, 75% once / 37% trials) · Milgram = obedience (shocks, 65%) · Zimbardo = roles (prison) · Sherif = group conflict (Robbers Cave, superordinate goals) · Latane-Darley = bystander (diffusion) · Festinger-Carlsmith = dissonance ($1 > $20).
- **Attitudes:** ABC = Affective, Behavioural, Cognitive. LaPiere = attitude-behaviour gap. Ajzen = theory of planned behaviour (intention).
- **Persuasion (ELM):** central route = argument quality, durable; peripheral = cues, temporary. Used when motivation/ability low.
- **Dissonance (Festinger):** inconsistency creates tension; insufficient justification (the $1) drives attitude change.
- **Attribution:** Heider (naive scientist, internal vs external) · Kelley covariation (consensus, distinctiveness, consistency) · Weiner (locus, stability, controllability).

- **Biases:** FAE = over-blame others' disposition. Self-serving = success internal, failure external. Actor-observer = self situational, other dispositional.
- **Influence:** normative (to be liked) vs informational (to be right). FITD (small then large) · DITF (large then small). Asch: one ally breaks conformity.
- **Presence of others:** social facilitation (helps easy, hurts hard tasks) · social loafing (effort drops in groups) · deindividuation (anonymity loosens restraint).
- **Groups:** polarisation (extremer than the mean) · groupthink (Janis: cohesion suppresses dissent) · Lewin styles (democratic best morale).
- **Intergroup:** stereotype (thinks) / prejudice (feels) / discrimination (acts). Tajfel social identity + minimal group = in-group bias. Sherif realistic conflict. Allport contact hypothesis (equal status, common goal, cooperation, support).
- **Prosocial/antisocial:** Batson empathy-altruism · frustration-aggression (Dollard, revised Berkowitz) · Bandura Bobo doll (observational aggression). Attraction = proximity, mere exposure, similarity, reciprocity, attractiveness halo.

ONE-DAY REVISION · PAPER 1

Phenomenology, the words that score

Descriptive psychopathology is a vocabulary exam. The marks live in precise definitions and the fine distinctions between near-neighbours: illusion vs hallucination, pseudohallucination vs true, overvalued idea vs delusion vs obsession, depersonalisation vs derealisation. Fish, Sims and Jaspers supply the words. Use the patient's experience, never your theory of it.

● CONCEPT ● RULE ● TRAP ● KEY TERM ● MNEMONIC

The colour tells you what kind of fact it is. Phenomenology describes *form* and leaves diagnosis to the next step.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Three-way belief table is the classic 10-mark question: **delusion** = false, absolute conviction, no insight, ego-syntonic, unresisted; **overvalued idea** = understandable and championed, ego-syntonic, dominates life; **obsession** = recognised as senseless, doubt, insight present, ego-dystonic, resisted. **Insight is the discriminating axis** (absent → partial → present).
2. Delusion definition that scores: a fixed, false belief held with absolute conviction, **out of keeping with the person's social and cultural background**, not amenable to reason; the **cultural clause is essential** (a belief shared by one's community is not a delusion).
3. **Delusional perception** is the high-frequency trap and a first-rank symptom: a real, normally perceived object is given a delusional meaning in **two stages** (normal percept, then un-understandable significance); contrast with a delusional misinterpretation built logically on a real event.
4. Hallucination (Esquirol) = a **perception without an external stimulus**, with full force, located in external objective space, and not under voluntary control; **pseudohallucination** sits in inner subjective space with partial insight but, like a true hallucination, is **not voluntarily controllable** (that is the line vs imagery).
5. Schneider first-rank symptoms (**ABCD**): Auditory (thought echo, third-person voices, running commentary), Broadcast/insertion/withdrawal of thought, Controlled feelings/impulses/acts plus somatic passivity, Delusional perception; they are **characteristic of, not pathognomonic for**, schizophrenia.

1 Foundations: Jaspers and the method

Karl Jaspers founded descriptive phenomenology in *General Psychopathology* (1913). The clinician sets aside theory and, by empathic re-living, describes the patient's inner experience faithfully. The whole subject rests on a few distinctions.

◆ **Form vs content** – **form** is the kind of experience (a delusion, a hallucination, an obsession); **content** is what it is about (poison, the neighbour, God). Diagnosis rests on **form**; content reflects culture and biography. Two patients with persecutory delusions share the form; the named persecutor is content.

● **Understanding (verstehen)** – grasping *meaningful* connections from within, by empathy: how one mental state arises from another (grief follows loss). The method of phenomenology.

- **Explaining (erklaren)** – establishing *causal* connections from outside, the natural-science mode (a lesion causes a deficit). Jaspers held both are needed but kept them apart.

Distinction	One pole	Other pole
Form / content	form = the experience type (diagnostic)	content = its subject matter (biographical)
Understand / explain	verstehen: meaningful, empathic links	erklaren: causal, scientific links
Subjective / objective	the patient's reported inner experience	signs the observer sees (behaviour, affect)
Primary / secondary	arises *de novo*, not from anything else	understandable from mood, delusion or another symptom

The un-understandable and the primary delusion

◆ **Un-understandable (the praecox feeling)** – Jaspers held the **primary delusion** to be psychologically *ir*reducible: it cannot be empathically understood as arising from mood, personality or experience. This non-understandability is, for Jaspers, the hallmark of the schizophrenic process and underlies first-rank phenomena.

- **Primary delusion** – appears fully formed, un-understandable, true (autochthonous). Comes in described stages, often after a delusional mood (Wahnstimmung), a pervasive uncanny sense that something is going on.
- **Secondary delusion** – understandable: it arises from another experience, such as a hallucination, a mood state (depressive guilt delusion), or another delusion. Also called a deluded notion (delusion-like idea).

Stage of primary delusion	What the patient experiences
Delusional mood (Wahnstimmung)	a strange, tense, uncanny atmosphere; "something is about to happen", not yet articulated
Delusional perception	a real, normally perceived object is given a delusional meaning (a first-rank symptom). Two-stage: normal percept, then the abnormal significance
Sudden delusional idea (autochthonous idea)	a fully formed delusion arrives out of the blue, in one step, with no percept attached
Delusional memory (retrospective)	a real or false memory is reinterpreted with present delusional meaning

● CLASSIC TRAP

Delusional perception is the one to get right. The percept itself is normal (the traffic light turns green); the *meaning* read into it is delusional and un-understandable ("that means I am the rightful king"). Two stages, normal then abnormal. Contrast with a delusional *misinterpretation*, where a secondary delusion is built logically on a real event. Schneiderian first-rank symptom.

2 Disorders of perception

First split: a **sensory distortion** changes a *real* percept; a **sensory deception** creates a percept where the real stimulus does not warrant it.

	Real object present?	Examples
Sensory distortion	yes, but altered in intensity, quality, size or form	hyperacusis, micropsia/macropsia, dysmegalopsia, changed colour
Illusion (deception)	yes, but misperceived as something else	a dressing gown seen as an intruder
Hallucination (deception)	no real object at all	hearing a voice in an empty room

Sensory distortions

- **Changes in intensity** – hyperaesthesia (heightened, eg. in mania, migraine, hangover) or hypoaesthesia (dulled, eg. in delirium, depression).
- **Changes in quality** – altered colouring of vision: chloropsia (green), xanthopsia (yellow, eg. digoxin), erythropsia (red).
- **Changes in spatial form (dysmegalopsia)** – objects look larger (macropsia) or smaller (micropsia) than real; seen in temporal lobe and retinal disease, and as the "Alice in Wonderland" syndrome.

Illusions

◆ **Illusion** – a **misperception of a real external stimulus**. Favoured by poor stimulus (dim light), reduced attention or alertness (delirium), and strong affect. Resolves when attention is directed at it. Three named types follow.

Type of illusion	Driven by	Example
Completion illusion	inattention; the mind fills gaps	misreading a misprinted word as the intended one
Affect illusion	a strong emotional state	a frightened person sees a shadow as an assailant
Pareidolia	vividness despite full attention; effortless, even sought out	faces in clouds or fire, figures in wallpaper

● RULE

Pareidolia is the odd one out: it grows *more* vivid with attention rather than dissolving, and occurs in healthy people. Completion and affect illusions fade once the person attends or the affect settles. Pareidolia is not pathological in itself.

Hallucinations: the definition that scores

◆ **Hallucination (Esquirol)** – a **perception without an external stimulus**. It has the full force and clarity of a true perception, is experienced as **real and located in external (objective) space**, and is **not under voluntary control**. These four qualities separate it from imagery and from pseudohallucination.

Modality	Key forms and associations
Auditory (commonest in psychiatry)	Elementary (noises, tinnitus-like) vs complex (voices). **2nd person** = voice speaks to the patient ("you are evil"); seen in depression, schizophrenia. **3rd person** = voices talk about the patient as "he/she"; a first-rank symptom. **Running commentary** on actions and **thought echo** (gedankenlautwerden, hearing one's thoughts aloud) are also first-rank.
Visual	Suggest an **organic** cause until proven otherwise (delirium, occipital/temporal lesions, substances, Charles Bonnet in eye disease). Lilliputian (tiny figures) classically in delirium tremens.
Olfactory	Smells, often unpleasant. Temporal lobe epilepsy (uncinate fits), depression (smelling one's own decay), schizophrenia.
Gustatory	Tastes, frequently with olfactory ones; epilepsy, sometimes part of a poisoning delusion.
Tactile / haptic	Touch on or under the skin. **Formication** (insects crawling) in cocaine and alcohol withdrawal; the delusion built on it is delusional infestation (Ekbom syndrome).
Somatic / visceral	Deep bodily or organ sensations (organs rotting, being electrified). Overlap with passivity (somatic passivity is first-rank when felt as imposed).

Special and named hallucinations

- **Functional hallucination** – a real stimulus and the hallucination occur in the **same modality**, the stimulus triggers the hallucination, and **both are perceived** (running water triggers a voice, and the water is still heard).
- **Reflex hallucination** – a stimulus in **one** modality triggers a hallucination in **another** (a sound provokes a visual image; a kind of pathological synaesthesia).
- **Extracampine hallucination** – perceived **outside the sensory field** (a voice from 100 miles away, a figure seen behind the head). Not in itself diagnostic of psychosis.
- **Hypnagogic / hypnopompic** – vivid perceptions on **falling asleep** (hypnagogic) or on **waking** (hypnopompic). Usually *non-pathological*; prominent in narcolepsy.
- **Autoscopy** – seeing a double of oneself (the phantom mirror-image), often felt to move with the self. **Negative autoscopy**: failing to see oneself in a mirror.
- **Lilliputian hallucination** – perception of tiny people or animals, often charming rather than frightening; classic in delirium tremens.

MNEMONIC

"Functional = same sense, Reflex = crossed senses, Extracampine = out of bounds"

Functional: the trigger and the hallucination share a modality and both are heard/seen. Reflex: one modality crosses to another. Extracampine: beyond the possible sensory field. The three are routinely confused; the discriminator is whether the real stimulus is still perceived and whether the modality crosses.

Pseudohallucination and imagery

- **Pseudohallucination** – perceived in **inner (subjective) space**, lacks the substantiality of a true percept, and the person retains some insight that it is not a real external event, yet it is **not** under voluntary control (this last point separates it from imagery). A grieving person hears the dead spouse's voice "inside the head".

Feature	True hallucination	Pseudohallucination	Mental imagery
Located in	external (objective) space	inner (subjective) space	inner subjective space
Vividness / substantiality	full, like a real percept	vivid but lacks substantiality	faint, sketchy
Under voluntary control?	no	no	yes
Insight that it is not real	typically absent	partially retained	fully retained

TRAP

The single line that separates **pseudohallucination** from **imagery** is *voluntary control*: imagery can be conjured and dismissed at will; a pseudohallucination cannot. The line separating pseudohallucination from a **true** hallucination is *location* (inner vs outer space) and *retained insight*. Jaspers and Kandinsky described it; usage varies, so define it before you use it.

3 Disorders of thought

Split thought into three layers, the standard exam scaffold: **stream** (tempo and flow), **form** (the logical structure of associations), and **content** (what is believed). Possession is a fourth: who owns the thought.

Disorders of the stream of thought

Term	Definition	Typical setting
Flight of ideas	accelerated thought; ideas connect by chance links (rhyme, pun, clang, distraction) but the **connections are still understandable**; the goal keeps shifting	mania (pressure of speech)
Retardation of thinking	slowed, sparse thought; subjective "thinking through treacle"	depression
Circumstantiality	over-inclusive, full of detail and digression but **eventually reaches the goal**	epilepsy, OCPD, learning disability, normal
Perseveration	inappropriate **repetition** of a response (word, theme) after it has ceased to be relevant; the mind is stuck	organic / frontal disease, dementia
Thought block	a sudden, complete **break** in the train of thought; the patient stops, the thought is gone (felt as withdrawn if elaborated)	schizophrenia

● CLASSIC TRAP

Flight of ideas keeps understandable links between successive ideas (you can follow the rhyme or the distraction); the goal is merely never reached because it keeps changing. **Loosening of associations** loses the links themselves: there is no understandable thread. FOI lives in mania, loosening in schizophrenia. **Circumstantiality reaches the point; tangentiality never does.**

Disorders of the form of thought (formal thought disorder)

Formal thought disorder is a disturbance of the **connections** between thoughts, inferred from disordered speech. Bleuler called the core schizophrenic version *loosening of associations*; Schneider and others named subtypes.

Term	Definition
Loosening of associations / derailment	thoughts slip onto subsidiary, loosely related tracks; the thread is lost. Bleuler's core sign.
Knight's move thinking	an abrupt, illogical leap between ideas with no understandable connection (named for the chess move). A severe form of derailment.
Tangentiality	replies veer away from the question and **never return** to the point (contrast circumstantiality, which does return).
Word salad / schizophasia	speech reduced to a meaningless jumble of words and phrases; the extreme of incoherence.
Neologism	a new word coined by the patient, or an ordinary word used in a private, idiosyncratic way.
Verbigeration	meaningless, stereotyped **repetition** of words or phrases (a formal/motor-speech sign; overlaps with palilalia).

- **Cameron's terms** – **asyndesis** (lack of adequate causal links), **metonyms** (an approximate or substitute word for the exact one), **overinclusion** (failure to keep conceptual boundaries), and **interpenetration** of themes. Classic descriptors of schizophrenic thought.
- **Other named signs** – **drivelling** (disordered intermixture of constituents), **fusion**, **omission**, and **condensation** (Schneider's terms for how the threads break down).

Disorders of the content of thought: delusions

◆ **Delusion** – a **fixed, false belief**, held with absolute conviction, **out of keeping with the person's social and cultural background**, and **not amenable to reason or evidence**. The cultural clause is essential: a belief shared by one's community is not a delusion.

Delusion	Content / belief	Often seen in
Persecutory	being harmed, conspired against, watched, poisoned	schizophrenia, delusional disorder, psychotic depression
Grandiose	inflated worth, power, identity, special mission	mania, schizophrenia
Nihilistic (Cotard)	self, organs, or the world do not exist / are dead or rotting	severe psychotic depression
Reference	neutral events, objects or remarks refer specifically to the patient	schizophrenia (an idea of reference is the non-delusional form)
Control / passivity	thoughts, feelings, impulses or acts are made or controlled by an outside agency ("made" phenomena)	schizophrenia (first-rank)
Guilt / sin	having committed an unforgivable wrong	depression (mood-congruent)
Infidelity (Othello)	the partner is unfaithful, on flimsy evidence	alcohol use, delusional disorder
Hypochondriacal	having a serious illness despite reassurance	depression, delusional disorder
Amorous (de Clerambault)	another, often of higher status, is secretly in love with the patient	delusional disorder, erotomania

- **Mood-congruent vs incongruent** – **congruent**: content fits the prevailing mood (guilt and nihilism in depression; grandiosity in mania). **Incongruent**: content does not fit the mood (a passivity delusion arising in pure euphoria); raises the suspicion of schizophrenia and carries prognostic weight.
- **By form, not theme** – remember that **primary vs secondary** (Section 1) is the formal axis, and persecutory/grandiose etc. is only the content. Examiners reward classifying a delusion on both axes.

TRAP

Folie a deux (shared/induced delusional disorder): a delusion is transferred from a dominant person (primary) to a closely associated, often dependent and isolated, secondary person. Separation of the pair frequently resolves the secondary's belief. **Capgras** (a familiar person replaced by an identical impostor) and **Fregoli** (a persecutor disguises themselves as different people) are the delusional misidentification syndromes.

Disorders of the possession of thought (alienation)

Here the patient loses the sense that their thoughts are their own. All three are Schneiderian first-rank symptoms.

Phenomenon	The patient's experience
Thought insertion	thoughts that are **not the patient's own** are put into the mind by an external agency
Thought withdrawal	thoughts are **removed** from the mind by an outside force (the experiential basis of some thought block)
Thought broadcasting	thoughts **escape and are shared** with or known to others, as if transmitted

RULE

The hallmark of alienation is the loss of the **sense of ownership or agency**: insertion and withdrawal attack ownership, passivity (Section 5) attacks agency over action. Obsessions, by contrast, are recognised as the patient's *own* thoughts (ego-dystonic but self-generated), which is exactly why an obsession is not a delusion of insertion.

4 Disorders of belief, compared

This three-way table is a classic 10-mark question. Learn the columns cold: conviction, insight, resistance, and whether the idea is ego-syntonic.

Feature	Delusion	Overvalued idea	Obsession
Falsity	false (or unfounded)	understandable, may even be true, but over-dominant	recognised as senseless or excessive by the patient
Conviction	absolute, unshakeable	strong, intense, but argues a case	variable; the patient doubts and is distressed
Insight	absent	partial; the belief is held with feeling, not as madness	present (it is "my own silly thought")
Ego relation	ego-syntonic	ego-syntonic, comfortable, championed	ego-dystonic, intrusive, unwanted
Resistance	none	none (it is pursued, not resisted)	resisted, at least initially
Origin / understandability	often un-understandable (if primary)	understandable from the personality and life	arises from within; recognised as self-generated
Typical context	psychosis	anorexia, hypochondriasis, morbid jealousy, dysmorphophobia, querulant states	OCD, depression

◆ **Overvalued idea (Wernicke)** – a comprehensible, isolated, **strongly held and emotionally invested** belief that comes to dominate the person's life and conduct. It is not bizarre and not resisted; the person identifies with it. Classic vehicle of morbid jealousy, dysmorphophobia and the drive for thinness in anorexia.

Obsession vs compulsion vs rumination

- **Obsession** – a recurrent, intrusive **thought, image or impulse**, recognised as the patient's own, experienced as senseless and unwanted (ego-dystonic), and **resisted**. Provokes anxiety. The cognitive event.
- **Compulsion (ritual)** – a repetitive **act or mental act** the patient feels driven to perform, usually to neutralise an obsession or prevent a feared outcome; performing it gives temporary relief (negative reinforcement). The behavioural response.
- **Rumination** – a repetitive, circular train of unproductive **thinking around a theme** (often in depression). Unlike an obsession it is usually *not* seen as senseless or intrusive in the same way, and is less actively resisted; it is mood-congruent brooding.

Hold the chain

Obsession is the intrusive thought → anxiety → **compulsion** is the act that relieves it. The compulsion is reinforced because it ends the distress, which is why OCD self-perpetuates.

The discriminators

Obsession is resisted and ego-dystonic; an overvalued idea is championed and ego-syntonic; a delusion is held with full conviction and no insight. **Insight is the axis**: full in obsession, partial in overvalued idea, absent in delusion.

5 Disorders of the self (ego) and passivity

Jaspers described several aspects of the experienced self (ego): awareness of **activity**, of **unity**, of **continuity** over time, and of the **boundary** between self and the outside world. Disorders map onto each.

Aspect of the self disturbed	Phenomenon
Awareness of activity	depersonalisation; passivity ("made") phenomena
Awareness of unity	feeling split or double (not multiple personality)
Awareness of continuity	feeling one is not the same person as before
Awareness of boundary	loss of the self/non-self boundary: thought insertion, withdrawal, broadcast; passivity

◆ **Depersonalisation** – a change in the awareness of the **self**: one feels unreal, detached, an automaton, emotions deadened, "as if" cut off from oneself. Crucially the patient **retains insight** that it is an "as if" feeling, which keeps it out of psychosis. Non-specific: anxiety, depression, fatigue, temporal lobe epilepsy, the normal.

◆ **Derealisation** – the same unreality projected onto the **surroundings**: the world seems flat, lifeless, stage-set, "as if" behind glass. Usually accompanies depersonalisation.

● **Disorders of ego boundary** – the demarcation between self and world breaks down. This is the formal home of **thought alienation** (insertion, withdrawal, broadcast) and of passivity: the patient can no longer tell where their own mental life ends and the world begins.

◆ **Passivity (made) phenomena** – the experience that one's own **movements, sensations, impulses or emotions are made or controlled by an outside agency**. The loss is of the sense of *agency*. Forms: made acts, made impulses, made feelings, and **somatic passivity** (bodily sensations imposed from outside). Schneiderian first-rank symptoms.

● CLASSIC TRAP

In **passivity** the patient knows the act or feeling is happening to *them* but believes an external force is the author (loss of agency, not of ownership). This is the discriminator from **thought insertion**, where the very *ownership* of the thought is denied. Both are first-rank. Distinguish from a normal sense of being "driven" by emotion, which carries no delusional attribution to an outside agent.

● MNEMONIC

"Schneider's first rank: ABCD"

Auditory: thought echo, third-person voices, running commentary. **B**roadcast (and insertion, withdrawal) of thought. **C**ontrolled feelings, impulses, acts (passivity) and somatic passivity. **D**elusional perception. First-rank symptoms are characteristic of, not pathognomonic for, schizophrenia (Schneider himself stressed this).

6 Emotion, memory, motor and speech

Disorders of emotion and affect

● **Mood vs affect** – **mood** is the sustained, pervasive emotional climate (the season); **affect** is the moment-to-moment, observable emotional weather, including its range and reactivity.

Term	Definition
Blunting / flattening of affect	reduced **intensity and range** of emotional expression; the face and voice lose modulation (a negative symptom of schizophrenia). Some authors reserve "flattening" for the most severe loss.
Incongruity of affect	affect **does not fit** the thought or situation (laughing while describing a bereavement). Suggests schizophrenia.
Lability	rapid, exaggerated shifts of emotion, poorly controlled (mania, organic states).
Apathy	absence of feeling and of drive; indifference.
Alexithymia	difficulty **identifying and describing** one's own emotions, with an externally oriented thinking style. A trait, not a state; common in psychosomatic presentations.
Anhedonia	loss of the capacity to experience pleasure.

● TRAP

Blunting (a reduction in the *amount* of emotion shown) is not the same as **incongruity** (emotion shown that does not *match* the content). Both point to schizophrenia but are different signs. **Alexithymia** is a difficulty *naming* emotion, distinct from blunting, which is a reduced *display* of it.

Disorders of memory

- **Confabulation** – the filling of gaps in memory with **fabricated or misplaced material the patient believes to be true**, with no intent to deceive. Classic in Korsakoff syndrome (amnesia plus confabulation, thiamine deficiency).
- **Deja vu** – a new situation feels **uncannily familiar**, as if already lived through. **Deja vecu** (already experienced) is the related fuller form.
- **Jamais vu** – the opposite: a **familiar** situation feels strange, unfamiliar, as if never encountered. Both occur in the normal, in anxiety, and in temporal lobe epilepsy.
- **Amnesia** – **anterograde**: cannot form new memories after the insult; **retrograde**: cannot recall events before it. **Pseudodementia**: a depressive memory impairment that mimics organic dementia and is reversible.

Disorders of motor behaviour (catatonia)

Catatonia is a syndrome of psychomotor disturbance. Kahlbaum named it; it appears in mood disorders and organic states, not only schizophrenia.

Sign	Definition
Stupor	profound reduction or absence of movement and speech, with **preserved consciousness** (the patient is aware).
Waxy flexibility (cerea flexibilitas)	limbs can be moulded into postures and **held**, with a smooth, plastic resistance like bending a candle.
Echopraxia	automatic **imitation of another's movements**.
Echolalia	automatic **repetition of another's words**.
Mannerism	an odd, stylised but **goal-directed** movement (an idiosyncratic salute).
Stereotypy	a repetitive, uniform, **non-goal-directed** movement (rocking, repeated rubbing).
Negativism	motiveless **resistance** to instruction, or doing the opposite of what is asked.
Ambitendency	alternating, hesitant **start-and-stop** of a movement; conflicting impulses (the half-completed handshake).
Automatic obedience	excessive co-operation, carrying out every instruction unquestioningly (the opposite pole to negativism).

CLASSIC TRAP

Mannerism = goal-directed but odd; stereotypy = repetitive and purposeless. A salute done strangely is a mannerism; rocking back and forth is a stereotypy. Also: **negativism** (motiveless resistance) and **automatic obedience** are opposite poles; **ambitendency** is the conflicted middle. Other catatonic signs: posturing, gegenhalten (opposition proportional to force applied), mitgehen, and forced grasping.

Disorders of speech

- **Pressure vs poverty of speech** – **pressure of speech**: rapid, copious, hard to interrupt (mania, mirrors flight of ideas). **Poverty of speech (alogia)**: markedly reduced amount, brief, empty replies (a negative symptom).
- **Repetition phenomena** – **palilalia**: repetition of one's own last word or phrase, often with increasing speed. **Logoclonia**: repetition of the last syllable of a word. **Verbigeration**: stereotyped repetition of words or phrases (see Section 3).
- **Disorganised speech signs** – **clang associations** (linking by sound/rhyme), **punning**, **echolalia**, and **mutism** (absence of speech with intact apparatus). **Aphasia/dysphasia** is the organic loss of language and must be excluded before formal thought disorder is diagnosed.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Foundations:** form (diagnostic) vs content (biographical) · understand (verstehen) vs explain (erklaren) · primary delusion = un-understandable, arises de novo; secondary = understandable from mood/hallucination. Delusional perception (real percept + delusional meaning, two stages) is a first-rank symptom.
- **Perception split:** distortion (real object, altered) vs deception (illusion = real object misperceived; hallucination = no object). Hallucination = percept without stimulus, in external space, full force, involuntary (Esquirol).
- **Illusions:** completion (inattention) · affect (emotion) · pareidolia (more vivid with attention, normal). First two fade with attention; pareidolia does not.
- **Auditory:** 2nd person speaks TO patient (depression, schizophrenia); 3rd person speaks ABOUT patient, running commentary, thought echo = first-rank. Visual hallucinations → think organic.
- **Special hallucinations:** functional = same modality, stimulus still perceived · reflex = crossed modality · extracampine = outside the sensory field · autoscopy = seeing one's double · Lilliputian = tiny figures (DTs) · hypnagogic/hypnopompic = normal, sleep transitions.
- **Pseudohallucination:** inner subjective space, lacks substantiality, partial insight, but NOT under voluntary control (which is the line vs imagery).
- **Thought stream:** flight of ideas (mania, links understandable, goal shifts) vs loosening (schizophrenia, links lost). Circumstantiality reaches the point; tangentiality never does. Perseveration = stuck repetition (organic); thought block = sudden break.
- **Formal thought disorder:** loosening/derailment · knight's move · word salad · neologism · verbigeration. Cameron: asyndesis, metonyms, overinclusion.
- **Delusion types:** persecutory, grandiose, nihilistic (Cotard), reference, control/passivity, guilt, infidelity (Othello), hypochondriacal, amorous (de Clerambault). Mood-congruent vs incongruent. Capgras (impostor) vs Fregoli (disguise). Folie a deux = induced.
- **Alienation (first-rank):** insertion, withdrawal, broadcast attack ownership; passivity attacks agency. Obsessions are recognised as one's OWN thoughts, so never an insertion.
- ★ **Belief table:** delusion = false, full conviction, no insight, ego-syntonic, unresisted · overvalued idea = understandable, championed, ego-syntonic, dominates life · obsession = recognised as senseless, doubt, insight present, ego-dystonic, resisted. Insight is the axis.
- **OCD chain:** obsession (thought, resisted) → anxiety → compulsion (act, relieves it). Rumination = circular brooding, mood-congruent, not actively resisted.
- **Ego/self:** depersonalisation = self feels unreal (“as if”, insight kept); derealisation = surroundings feel unreal. Passivity = made acts/feelings/somatic, loss of agency (first-rank).
- **Emotion:** blunting = reduced display; incongruity = mismatched display; alexithymia = cannot name own emotions; anhedonia = no pleasure.
- **Memory:** confabulation = honest gap-filling (Korsakoff). Deja vu (new feels familiar) vs jamais vu (familiar feels strange). Anterograde vs retrograde amnesia.
- **Catatonia:** stupor (aware), waxy flexibility, echopraxia (acts), echolalia (words), mannerism (odd but purposeful) vs stereotypy (purposeless), negativism vs automatic obedience, ambitendency (start-stop).

ONE-DAY REVISION · PAPER 1

Assessment & psychometric testing

The instruments and the rules that make them trustworthy. Hold three things per test: what it measures, who built it, and the one number or scoring system that names it. The psychometric principles up front are the most examinable part – reliability versus validity is a guaranteed short note.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Screening statistics (sensitivity, PPV) live in the statistics pack; bedside cognition and eponymous scales cross-ref to their own packs.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. **A valid test must be reliable, but a reliable test can still be invalid** (it measures the wrong thing consistently): reliability is necessary but not sufficient for validity, and it sets the ceiling on validity. This direction is the guaranteed short note.
2. Deviation IQ is normally distributed with **mean 100, SD 15** (replacing the ratio IQ); but **IQ <70 alone is NOT intellectual disability** → DSM-5/ICD-11 require deficits in BOTH intellectual AND adaptive functioning, with onset in the developmental period.
3. **Sensitivity and specificity are intrinsic to the test and do not change with prevalence; PPV and NPV do shift with prevalence** → a good screening test in a low-prevalence population yields many false positives and a low PPV (SnNOUT, SpPIN).
4. The MMPI's examinable edge is its **validity scales L (lie), F (infrequency / faking bad) and K (defensiveness)**, which detect faking and flag whether the clinical profile can be trusted; it is empirically keyed with 10 clinical scales.
5. The ratio quotients all share the form (age-measured / chronological age) × 100: **IQ** (Stanford-Binet), **SQ** from the VSMS (social/adaptive), **DQ** from the DST (development); Indian adaptations to hold are **MISIC** (WISC) and **Bhatia's Battery** (performance, low-literacy).

1 Psychometric principles

A test is only as good as two properties: it must give the **same answer on repeat** (reliability) and it must **measure what it claims** (validity). The whole field hangs on telling these two apart.

Reliability: consistency of measurement

Type	Question it answers	How it is checked
Test-retest	stable across time?	same test, same people, two occasions; correlate the scores
Inter-rater	stable across observers?	two raters score the same material; agreement (e.g. Cohen's kappa)
Parallel / alternate form	stable across versions?	two equivalent forms given; correlate the scores
Split-half	internally consistent?	split items into two halves, correlate; Spearman-Brown corrects for length
Internal consistency	do items hang together?	Cronbach's alpha (α): average of all possible split-halves; KR-20 for binary items

◆ **Cronbach's alpha** – the standard index of internal consistency, ranging 0 to 1. A common rule of thumb: **α ≥ 0.7** acceptable, ≥ 0.8 good. Very high α (>0.95) can signal redundant items, not a better test.

Validity: are you measuring the right thing

Type	Subtype	Asks
Face	–	does it *look* like it measures the thing? (weakest, subjective)
Content	–	do items cover the whole domain? (judged by experts)
Construct	convergent / divergent	does it fit the theoretical construct? agrees with related measures, differs from unrelated ones
Criterion	Concurrent	agrees with a criterion measured **now** (gold standard, same time)
	Predictive	predicts a criterion in the **future** (e.g. test predicts later job performance)

HOLD THIS

A **reliable** test can still be **invalid** (it measures the wrong thing consistently). But a **valid** test must be **reliable** – you cannot hit the bullseye if your shots scatter. Reliability is necessary but not sufficient for validity.

● RULE

The dartboard image carries the marks: **reliable + invalid** = tight cluster, off-centre; **valid + reliable** = tight cluster on the bullseye; **unreliable** = scattered (and therefore cannot be valid). Reliability sets a ceiling on validity.

Standardisation and norms

● **Standardisation** – fixing the administration, scoring and interpretation so the test is given identically to everyone. A prerequisite for fair comparison.

● **Norms** – reference scores from a large representative **standardisation sample**, against which an individual's raw score is interpreted (percentiles, standard scores, age/grade equivalents). A score means nothing without the norm group.

Screening statistics (cross-ref: statistics pack)

Term	Definition	Property
Sensitivity	true positives among the diseased: of those with the condition, how many test positive	high = good for **ruling out** (SnNOUT)
Specificity	true negatives among the healthy: of those without it, how many test negative	high = good for **ruling in** (SpPIN)
PPV	of those testing positive, how many truly have it	depends on prevalence
NPV	of those testing negative, how many are truly free of it	depends on prevalence

● TRAP

Sensitivity and specificity are **intrinsic** to the test and do not change with prevalence. PPV and NPV **do** shift with prevalence: a good screening test applied to a low-prevalence population yields many false positives and a low PPV. Full derivations and worked numbers are in the statistics pack.

2 Intelligence tests

The lineage runs from a single age-based scale to a family of standardised batteries reporting a deviation IQ. Hold the chronology and the age band each Wechsler scale covers.

Test	Author / year	Note
Binet-Simon scale	Binet & Simon, 1905 (France)	first practical intelligence scale; built to identify schoolchildren needing help; introduced **mental age**
Stanford-Binet	Terman, 1916 (Stanford)	US revision; popularised the **ratio IQ** = (mental age / chronological age) × 100
Wechsler scales	David Wechsler, from 1939	introduced the **deviation IQ**; verbal + performance subtests → full-scale IQ
Raven's Progressive Matrices	Raven, 1938	nonverbal, **culture-fair**; pattern-completion; loads on g (Spearman) and fluid reasoning

The Wechsler family (by age)

Scale	Full name	Age range
WPPSI	Wechsler Preschool & Primary Scale of Intelligence	preschool (~2.5 to 7 yrs)
WISC	Wechsler Intelligence Scale for Children	children (~6 to 16 yrs)
WAIS	Wechsler Adult Intelligence Scale	adults (16 yrs and over)

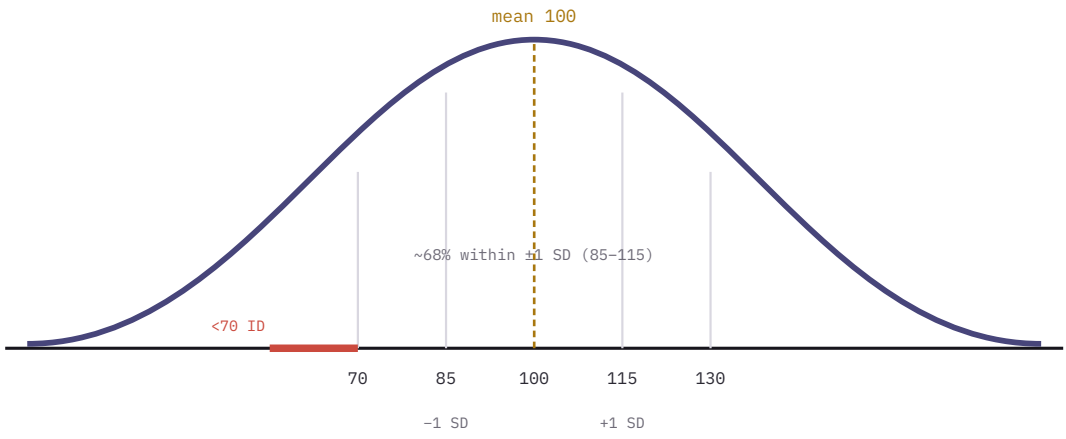
● MNEMONIC

"Preschool, Children, Adults → WPPSI, WISC, WAIS"

Youngest to oldest. WPPSI = smallest people, WAIS = adults. All three report a deviation IQ.

◆ **Deviation IQ** – the modern standard: a normal distribution with **mean 100, SD 15**. So ~68% of people fall between 85 and 115, ~95% between 70 and 130. It replaced the ratio IQ, which broke down in adulthood (mental age plateaus).

THE IQ BELL CURVE (DEVIATION IQ, MEAN 100, SD 15)



Deviation IQ is normally distributed with mean 100 and SD 15. About 68% score 85–115, about 95% score 70–130. The shaded segment below 70 (two SDs below the mean) is the intellectual disability cut-off; above 130 is the gifted range.

IQ classification and the ID cut-off

Band	IQ range	Label
Above average	>130	very superior / gifted
Average	90–109	average
Borderline	70–79	borderline intellectual functioning

Band	IQ range	Label
Intellectual disability (IQ <70)	50–69	mild ID
	35–49	moderate ID
	20–34	severe ID
	<20	profound ID

TRAP

IQ <70 alone is **not** intellectual disability. DSM-5 and ICD-11 require **both** deficits in intellectual functioning *and* in adaptive functioning, with **onset in the developmental period**. Modern severity grading leans on adaptive functioning, not the IQ number alone.

Indian adaptations

- **MISIC** – Malin's Indian Intelligence Scale for Indian Children, an Indian adaptation of the WISC (Malin, 1969), widely used for school-age children.
- **Bhatia's Battery** – Bhatia's Battery of Performance Tests of Intelligence (C. M. Bhatia, 1955); five performance subtests (e.g. Koh's block design, pass-along, pattern drawing), designed for use with low-literacy Indian populations.
- **Other Indian tools** – **Seguin Form Board** (used widely in India to estimate IQ in young children via speed of placement), **Vineland Social Maturity Scale** (Indian adaptation by Malin) and the **Developmental Screening Test** (Bharat Raj). Detailed below in section 6.

3 Objective (self-report) personality tests

Objective tests use structured items with fixed response options, scored to standard keys. They are reliable and quantifiable but vulnerable to faking and response sets.

Test	Author	What it measures
MMPI / MMPI-2	Hathaway & McKinley	psychopathology across **10 clinical scales** plus **validity scales** (L, F, K) that detect faking; **empirically keyed**. MMPI-2 = the 1989 restandardisation
16PF	Cattell	**16 source traits** of normal personality, derived by factor analysis
EPQ	Eysenck	the **PEN** dimensions: Psychoticism, Extraversion, Neuroticism (plus a lie scale)
NEO-PI-R	Costa & McCrae	the **Big Five / OCEAN**: Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism, each with 6 facets

RULE

The MMPI's edge is its **validity scales**: L (lie), F (infrequency / faking bad), K (defensiveness). They flag whether the clinical profile can be trusted. This is the feature most often asked about MMPI in vivas.

4 Projective personality tests

- **Projective hypothesis** – presented with an **ambiguous** stimulus, a person projects their own unconscious needs, conflicts and dispositions onto it. Vague material acts as a screen onto which the inner world is cast.

Test	Author	Stimulus / task
Rorschach	Hermann Rorschach (1921)	**10 symmetrical inkblots** (5 black/grey, 2 black/red, 3 multicoloured); "what might this be?"; scored by the **Exner Comprehensive System** (location, determinants, content, popularity)
TAT	Murray & Morgan (1935)	~31 ambiguous picture cards; the person tells a **story**; interpreted via Murray's **need-press** theory (the hero's needs vs environmental press)
CAT	Bellak	Children's Apperception Test: TAT for children, using **animal** (or human) figures in family scenes
Sentence Completion	Rotter (ISB) and others	complete stems ("My greatest fear..."); semi-structured, easier to score
Draw-a-Person (DAP)	Machover; Goodenough	draw a person; interpreted for personality (Machover) or, in children, as an IQ estimate (Goodenough Draw-a-Man)
House-Tree-Person (HTP)	Buck	draw a house, a tree and a person; the drawings are read for emotional and personality features

● VALIDITY CRITIQUE

Projective tests are criticised for **poor reliability and weak validity**: low inter-rater agreement, susceptibility to examiner bias, and limited standardised norms. Exner's system improved the Rorschach's standardisation, but the projective family as a whole remains contested. Objective tests outperform them psychometrically; projectives persist for their rich, hypothesis-generating clinical material.

5 Neuropsychological tests

These probe specific cognitive functions and localise dysfunction. For the exam, map each test to the **function and lobe** it tests.

Comprehensive batteries

- **Halstead-Reitan Battery** – a fixed set of tests (e.g. Category Test, Tactual Performance, Trail Making) giving a global **Impairment Index**; detects and broadly localises brain damage.
- **Luria-Nebraska Battery** – standardised from Luria's qualitative methods; assesses multiple functional domains (motor, rhythm, tactile, language, memory, intellectual processes).

Test mapped to function and lobe

Test	Function probed	Lobe / region
Wisconsin Card Sorting (WCST)	set-shifting, abstraction; **perseveration**	frontal / dorsolateral prefrontal
Stroop Test	selective attention, response inhibition	frontal (anterior cingulate / prefrontal)
Trail Making A & B	A: visual scanning, speed; B: set-shifting (executive)	frontal / executive
Verbal fluency (FAS / category)	generative word retrieval, executive search	frontal (and left temporal)
Bender-Gestalt	visuoconstructional / visuomotor copying	parietal / occipital
Wechsler Memory Scale (WMS)	verbal & visual memory, working memory	medial temporal (hippocampal)
Rey-Osterrieth Complex Figure	visuospatial construction + visual memory	parietal + medial temporal

QUICK RULE

The **WCST** is the classic **frontal/executive** test: failure to shift the sorting rule (**perseveration**) is the signature of dorsolateral prefrontal dysfunction. Pair it with **Trail Making B**, **Stroop** and **verbal fluency** as the executive cluster.

Bedside cognition (cross-ref: cognitive assessment)

- **MMSE** – Mini-Mental State Examination (Folstein); **/30**; quick global cognitive screen; weak on executive function and frontal/subcortical dementias.
- **MoCA** – Montreal Cognitive Assessment; **/30**; more sensitive than MMSE to **mild cognitive impairment**; includes executive and visuospatial items.
- **ACE / ACE-III** – Addenbrooke's Cognitive Examination; **/100**; five domains (attention, memory, fluency, language, visuospatial); good for differentiating dementia subtypes. Full scoring and cut-offs are in the cognitive assessment pack.

6 Developmental, adaptive & specific tests

Tests for development, adaptive behaviour and specific abilities – heavily used in child psychiatry and intellectual disability work, and a favourite Indian-adaptation question.

Test	Author	Measures / output
Vineland Social Maturity Scale (VSMS)	Doll; Indian adaptation Malin	adaptive / social functioning → a **Social Quotient (SQ)** = (social age / chronological age) × 100
Developmental Screening Test (DST)	Bharat Raj (Indian)	developmental level → a **Developmental Quotient (DQ)**; rapid screen, birth to ~15 yrs
Seguin Form Board (SFB)	Seguin	visuomotor speed and form perception; placement **time** estimates IQ in young children
Gesell Developmental Schedules	Arnold Gesell	developmental milestones across motor, adaptive, language and personal-social domains in infants
Bayley Scales of Infant Development	Nancy Bayley	cognitive, motor and language development in infants and toddlers

◆ **The quotients** – **IQ** = intelligence; **SQ** (social quotient) from the VSMS = adaptive/social maturity; **DQ** (developmental quotient) from the DST = overall development. All three follow the same ratio form, age-measured / chronological-age × 100.

Clinician-rated vs self-report

Clinician-rated (observer)

A trained rater scores the patient. Less subject to denial or poor insight; needs trained raters and is time-costly.
Examples: **HAM-D** (depression), **YMRS** (mania), **PANSS** (psychosis), **HAM-A** (anxiety).

Self-report (rated by patient)

The patient rates their own symptoms. Quick, captures the inner experience; vulnerable to faking, response set and insight.
Examples: **BDI** (depression), **BAI** (anxiety), **PHQ-9**, **GHQ**.

● RULE

The named symptom rating scales and their authors (the eponyms) are drilled in the eponyms pack. Here, hold only the **clinician-rated vs self-report** distinction and one or two examples on each side.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Reliability vs validity:** reliability = consistency, validity = measuring the right thing. A reliable test *can* be invalid; a valid test *must* be reliable. Reliability caps validity.

● **Reliability types:** test-retest (time) · inter-rater (observers, kappa) · parallel-form (versions) · split-half (Spearman-Brown) · internal consistency (**Cronbach's alpha**, ≥0.7).

- **Validity types:** face · content · construct (convergent/divergent) · criterion = concurrent (now) + predictive (future).
- **Screening:** sensitivity & specificity are intrinsic; PPV & NPV depend on prevalence. SnNOUT, SpPIN.
- **Intelligence lineage:** Binet-Simon (1905, mental age) → Stanford-Binet (Terman, ratio IQ) → Wechsler (deviation IQ). Raven's = culture-fair, nonverbal.
- **Wechsler by age:** WPPSI (preschool) · WISC (children) · WAIS (adults). Deviation IQ = mean **100**, SD **15**.
- **ID cut-off:** IQ <70, but requires adaptive deficits + developmental onset too. Indian adaptations: **MISIC** (WISC), **Bhatia's Battery** (performance).
- **Objective personality:** MMPI/MMPI-2 (psychopathology + L/F/K validity scales) · 16PF (Cattell) · EPQ (Eysenck, PEN) · NEO-PI-R (Big Five).
- **Projective:** projective hypothesis → ambiguous stimulus. Rorschach (10 blots, Exner) · TAT (Murray, need-press) · CAT (Bellak, children) · sentence completion · DAP / HTP. Critique: weak reliability and validity.
- **Neuropsych:** batteries = Halstead-Reitan, Luria-Nebraska. Executive cluster = **WCST** (perseveration, frontal), Stroop, Trail Making B, fluency. Bender-Gestalt = visuoconstruction (parietal).
- **Quotients:** IQ (intelligence) · SQ (VSMS, social) · DQ (DST, development) · all = age-measure / chronological age × 100. Bhatia, Seguin form board, Gesell, Bayley.
- **Rating scales:** clinician-rated (HAM-D, YMRS, PANSS) vs self-report (BDI, PHQ-9, GHQ). Eponyms in their own pack.

ONE-DAY REVISION · PAPER 1

Anthropology

The study of humankind, and the lens that keeps psychiatry honest about culture. Hold the branches, the core concepts, and above all the culture-bound syndromes table: region plus core picture is the marks-carrying pair. Know how Kleinman, Parsons and van Gennep frame illness, and how DSM-5 re-read the syndromes as cultural concepts of distress.

● CONCEPT ● RULE ● TRAP ● KEY TERM ● MNEMONIC

The colour tells you what kind of fact it is. The culture-bound syndromes table is the centrepiece – learn each region and its core picture.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Culture-bound syndromes, the marks-carrying pair is **region + core picture**: amok (SE Asia → homicidal frenzy), koro (Asia → genital retraction), latah (Malaysia → startle + echolalia/echopraxia), **dhat** (India → semen-loss anxiety), susto (Latin America → soul loss), taijin kyofusho (Japan → fear of offending others), brain fag (West Africa → student fatigue).
2. The triad: **disease = clinician's biology (etic), illness = patient's lived experience (emic), sickness = society's recognition**; the disease/illness split is Kleinman's.
3. India high-yield syndromes are **dhat, koro and possession/trance states**; expect at least one in any culture-and-psychiatry question, with the care pathway running through family, faith healers and temples/dargahs first.
4. **Emic = insider (from phon-emic), etic = outsider (from phon-etic)**; Kleinman warns against the **category fallacy**, forcing a Western diagnostic category onto a culture where it has no coherence.
5. DSM-5 retired "culture-bound syndromes" and replaced them with **cultural concepts of distress = cultural syndromes + idioms of distress + cultural explanations/perceived causes**; van Gennep rite of passage is separation → **liminality** → incorporation, with liminality (Turner: communitas) the most examined phase.

1 The branches and the field

Anthropology is the holistic study of humanity across time and place. Classically it splits into **four fields**; two applied sub-fields matter most to psychiatry.

Branch	Studies	Relevance to psychiatry
Physical / biological	human evolution, genetics, primatology, adaptation, the biological human	evolutionary roots of behaviour; bio-cultural interaction
Social / cultural	living cultures: belief, kinship, ritual, social organisation (ethnography)	the core contributor: culture shapes illness and care
Linguistic	language: structure, change, and how it shapes thought (Sapir-Whorf)	idioms of distress; how symptoms are named and voiced
Archaeological	past cultures through material remains and artefacts	least direct; historical depth of human practice

The two applied sub-fields for psychiatry

- **Medical anthropology** – how culture shapes health, illness, healing and care-seeking. Source of **explanatory models**, the illness/disease/sickness split, and the study of healers and pathways to care.
- **Psychological anthropology** – the interplay of culture and the mind/self: personality, emotion, cognition and child-rearing across cultures. Older "culture and personality" school (Mead, Benedict, Kardiner).

What anthropology contributes to psychiatry

- **Cultural relativity of normality** – what counts as deviant, distressing or mad is partly culturally defined; guards against imposing one culture's norms as universal.
- **The patient's framework** – explanatory models, idioms of distress and help-seeking pathways reveal how the patient understands and acts on the problem.
- **Critique of category-bound nosology** – warns against **category fallacy** (Kleinman): applying a Western diagnostic category in a culture where it has no coherence.

QUICK RULE

For psychiatry, the load-bearing branches are **social/cultural** anthropology and its applied arm, **medical anthropology**. The rest is context.

2 Core concepts

The vocabulary of culture. Each term is a one-line definition examiners can ask in isolation. Learn the contrasting pairs together.

Concept	Meaning
Culture	shared, learned, transmitted way of life: beliefs, values, norms, practices and meanings of a group (Tylor's classic "complex whole")
Enculturation	learning one's *own* culture, especially in childhood (socialisation into the group)
Acculturation	cultural change from sustained contact with *another* culture (migration, colonisation); linked to acculturative stress
Ethnocentrism	judging other cultures by the standards of one's own, treating one's own as superior
Cultural relativism	understanding a culture on its own terms, without external judgement (the methodological counter to ethnocentrism)

Emic vs etic: the two vantage points

Emic (insider)

The **insider's** view: meanings, categories and experience as the culture itself understands them. Derived from *phon**emic***. The patient's own model.

Etic (outsider)

The **outsider's**, comparative, analyst's view: culture-general categories applied across groups. Derived from *phon**etic***. The clinician's diagnostic frame.

MNEMONIC

"emic = insider, etic = external"

Coined by linguist Kenneth Pike from phon**emic** (insider, meaning-bearing) and phon**etic** (outsider, comparative). Good cross-cultural psychiatry holds both.

Kinship, family and the rites of passage

- **Kinship** – the culturally recognised network of relatedness (blood, marriage, adoption). Organises descent, residence and obligation; in India often the **joint / extended family**, a key social support and stressor in clinical work.
- **Family systems** – nuclear vs joint/extended; patrilineal vs matrilineal descent; patrilocal vs matrilocal residence. Shapes who decides, who cares, and who brings the patient.
- **Ritual and symbol** – ritual is patterned, repetitive, rule-bound symbolic action; a symbol stands for something beyond itself (Victor Turner). Both carry shared meaning and regulate transition and emotion.

Van Gennep: the three phases of a rite of passage

A **rite of passage** marks movement from one social status to another (birth, initiation, marriage, death). Arnold van Gennep (1909) named three universal phases; Victor Turner deepened the middle one.

Phase	What happens	Example
Separation (preliminal)	detachment from the old status / role	leaving home for initiation
Liminality (liminal)	the "in-between", ambiguous, betwixt-and-between state; old status shed, new one not yet held	the initiate, neither child nor adult
Incorporation (postliminal)	re-entry with the new status, recognised by the community	return as an adult / married person

TRAP

Liminality is the most examined of the three. It is the threshold phase (Latin *limen*, threshold): identity is suspended and ambiguous. Turner linked it to *communitas* (an unstructured bond among those passing through together). Disrupted or absent rites of passage are invoked in adolescent distress.

3 Culture and mental illness

Culture does not just colour symptoms; it shapes what counts as illness, how distress is expressed, and where help is sought. The medical-anthropology toolkit is high-yield.

Illness vs disease vs sickness

Term	Vantage point	What it captures
Disease	the **clinician's** (etic)	the biological abnormality / pathology as the practitioner defines it
Illness	the **patient's** (emic)	the lived, subjective experience of being unwell and its meaning
Sickness	the **society's**	the social role and recognition of being unwell; illness made public

HOLD THIS

Disease = clinician's biology. **Illness** = patient's experience. **Sickness** = society's recognition. The triad is a classic short-note; the disease/illness split is Kleinman's.

Kleinman: explanatory models and idioms of distress

- **Explanatory model (EM)** – Arthur Kleinman: the set of beliefs a person holds about an episode of illness – its **cause, onset, pathophysiology, course and treatment**. Patient and clinician each hold one; clinical work is negotiating between them.

- **Kleinman's 8 questions** – the practical tool to elicit a patient's EM: *What do you call it? What caused it? Why now? How does it work? How severe? What do you fear? What problems has it caused? What treatment should you receive?*

◆ **Idioms of distress** – culturally shared ways of expressing and communicating suffering (Mark Nichter). May be somatic, behavioural or metaphoric. Example: "**tension**" or "gas" / "heat in the head" in South Asia; not a diagnosis but a language of suffering.

- **Category fallacy** – Kleinman's warning: forcing a culturally alien diagnostic category onto experience where it does not fit, then mistaking the artefact for a finding.

Parsons: the sick role

Talcott Parsons (1951) framed being sick as a temporary, sanctioned **social role** with paired rights and duties.

Rights (exemptions)	Duties (obligations)
Exempt from normal social roles (proportionate to severity)	Must want to get well – the sick role is undesirable, only temporary
Not held responsible for the condition (it is beyond their control)	Must seek competent help and cooperate with treatment

● CLASSIC TRAP

The sick role assumes *acute, curable* illness and a patient who *wants* recovery. It fits chronic illness, disability and many psychiatric conditions poorly: there may be no "getting well", responsibility is contested, and stigma blocks the exemptions. This mismatch is itself an exam point.

Help-seeking and pathways to care

- **Pathways to care** – the sequence of contacts a person makes before reaching psychiatric care (Goldberg & Huxley's filter model; the WHO pathways study). In India the route often runs through **family, faith healers, religious sites and general practitioners** first.

- **Kleinman's three sectors of care** – the **popular** (family, self, lay networks – where most illness is managed), the **folk** (traditional / religious healers), and the **professional** (biomedicine, organised systems). People move between sectors.

4 Culture-bound (culture-specific) syndromes

The centrepiece. A **culture-bound syndrome** is a recurrent, locality-specific pattern of distressing experience and behaviour, recognised as an illness within a particular culture, often without a clean equivalent in standard nosology. Learn the pair: **region + core picture**.

Syndrome	Region / culture	Core picture
Amok	Malaysia, Indonesia (SE Asia)	a brooding period, then a sudden outburst of indiscriminate **violent, homicidal** aggression, often ending in exhaustion or amnesia ("running amok")
Koro	South / East Asia (China, India, Malaysia)	sudden intense fear that the **genitals are retracting** into the body and that this will be fatal; can occur in epidemics. India: "jhinjhinia" / "koro" outbreaks
Latah	Malaysia, Indonesia	exaggerated **startle** response with echolalia, echopraxia, automatic obedience and coprolalia, after a sudden fright; more in middle-aged women
Dhat	**Indian subcontinent**	anxious preoccupation with **semen loss** (in urine / nocturnally), with fatigue, weakness and somatic complaints; semen seen as a vital essence
Susto	Latin America	illness attributed to a **frightening event causing the soul to leave** the body ("soul loss"); appetite/sleep disturbance, sadness, weakness

Syndrome	Region / culture	Core picture
Taijin kyofusho	Japan	intense fear of **offending or harming others** through one's appearance, gaze, blushing or body odour (an other-focused social anxiety)
Brain fag	West Africa	"brain fatigue" in **students**: difficulty concentrating and remembering, head/neck pain or burning, visual symptoms, tied to study strain
Windigo / witiko	Algonquian peoples, North America	belief one has turned into, or is possessed by, a **cannibalistic monster**; craving for human flesh, distaste for ordinary food (existence as a syndrome is debated)
Ataque de nervios	Latino / Caribbean (Puerto Rico)	acute distress: **uncontrollable shouting, crying, trembling**, heat rising, aggression, sometimes faint or seizure-like episodes, usually after a family stressor
Possession / trance states	worldwide; common in **India**	altered identity/consciousness attributed to a spirit, deity or ancestor taking over; may be culturally sanctioned (ritual) or distressing (clinical)

INDIA HIGH-YIELD

The Indian-context syndromes are **dhat** (semen-loss anxiety), **koro** (genital-retraction epidemics) and **possession / trance states** (spirit possession, often presenting via faith healers). Expect at least one in any culture-and-psychiatry question.

● DSM-5 REFRAME

DSM-5 retired the static glossary of "culture-bound syndromes" and replaced it with **cultural concepts of distress**, under three headings: **cultural syndromes** (clusters of co-occurring symptoms, e.g. ataque de nervios), **cultural idioms of distress** (shared ways of talking about suffering, e.g. "nervios", "tension"), and **cultural explanations / perceived causes** (e.g. susto's "soul loss", "maladi moun"). The shift stresses that these are not exotic, bounded diseases but locally patterned ways of experiencing, naming and explaining distress.

5 Cultural formulation & transcultural psychiatry

Transcultural psychiatry

- **Transcultural (cross-cultural) psychiatry** – the comparative study of mental illness and its care across cultures, and of how culture shapes presentation, diagnosis, prevalence and treatment. Associated with Kraepelin's early comparative work, then Wittkower, Murphy, Leff and Kleinman.
- **Pathoplastic vs pathogenic** – culture can be **pathoplastic** (shaping the *content* and expression of an illness, e.g. the theme of delusions) or, more rarely, **pathogenic** (a genuine cause). Most accept culture as mainly pathoplastic for the major disorders.

The DSM-5 Cultural Formulation Interview (CFI)

DSM-5 carries an **Outline for Cultural Formulation** and a 16-question **Cultural Formulation Interview** to operationalise it. Learn the four classic OCF domains plus the conclusion.

Domain	What it assesses
1. Cultural identity	ethnicity, language, religion, migration, reference groups; the person's self-definition
2. Cultural conceptualisation of distress	idioms of distress, explanatory models, perceived causes; what the problem means to them
3. Psychosocial stressors & cultural features of vulnerability and resilience	stressors, supports, levels of functioning, the role of religion and kin networks
4. Cultural features of the clinician-patient relationship	differences in culture, language and status between clinician and patient, and their effect

Domain	What it assesses
5. Overall cultural assessment	synthesis for diagnosis and culturally appropriate care

MNEMONIC

"Identity · Concept · Stressors · Relationship · Assessment"

The five OCF headings, in order. The CFI's core 16 questions map onto domains 1–4; supplementary modules expand each.

The Indian context

◆ **Dhat syndrome** – the signature Indian culture-related presentation: anxious **preoccupation with semen loss** and consequent weakness, rooted in the cultural value of semen (Ayurvedic *dhatu*) as vital essence. Often somatic, presenting to GPs and vaidyas before psychiatry.

● **Possession & religious-healing pathways** – spirit possession (*bhoot*, deity or ancestor) is a common emic explanation for distress; care frequently begins at **temples, dargahs and faith healers** (e.g. healing temples), with psychiatry reached late. Engaging, not dismissing, this pathway improves care.

● **Other Indian threads** – the **joint family** as support and stressor; high **somatisation** of psychological distress; stigma channelling help-seeking through the popular and folk sectors first. India's medical pluralism (AYUSH alongside biomedicine) is the backdrop.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Branches:** physical/biological · social/cultural · linguistic · archaeological. Applied: **medical** (health, illness, healing) and **psychological** (culture & mind) anthropology. Social/cultural + medical carry psychiatry.
- **Core pairs:** enculturation (learn own culture) vs acculturation (contact change); ethnocentrism vs cultural relativism; **emic** = insider vs **etic** = outsider.
- **Van Gennep rite of passage: separation → liminality → incorporation**. Liminal = betwixt-and-between (Turner: communitas).
- **The triad:** disease = clinician's biology (etic) · illness = patient's experience (emic) · sickness = society's recognition.
- **Kleinman:** explanatory models (cause, course, treatment) & the 8 questions; idioms of distress; category fallacy; three sectors of care (popular / folk / professional).
- **Parsons sick role:** rights = exemption + not blamed; duties = want to get well + seek competent help. Fits chronic / psychiatric illness poorly.
- ★ **Syndromes:** amok (SE Asia, homicidal frenzy) · koro (Asia, genital retraction) · latah (Malaysia, startle + echolalia) · **dhat (India, semen loss)** · susto (Latin America, soul loss) · taijin kyofusho (Japan, fear of offending others) · brain fag (West Africa, student fatigue) · windigo (N. America, cannibal) · ataque de nervios (Latino/Caribbean) · possession/trance (worldwide, India).
- **India:** dhat, koro epidemics, possession states; faith-healer / temple-dargah pathways; somatisation; joint family.
- **DSM-5:** "culture-bound syndromes" → **cultural concepts of distress** = syndromes + idioms + explanations.
- **Cultural formulation (OCF):** identity → conceptualisation of distress → stressors/supports → clinician-patient relationship → overall assessment. CFI = 16 core questions. Culture mostly **pathoplastic**, rarely pathogenic.

ONE-DAY REVISION · PAPER 1

Ethology

The biology of behaviour in the natural setting: instinct, the founders and their Nobel, imprinting, and the comparative roots of attachment. For psychiatry the payoff is direct – Harlow and Bowlby turned animal observation into a theory of how human bonds form and fail. Hold the four questions, the three founders, and the Harlow/Bowlby line.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The attachment material (Harlow, Bowlby, Ainsworth) carries the psychiatry marks.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Harlow's rhesus monkeys chose the cloth mother (no food) over the wire mother that fed them: the bond is built on contact comfort, not feeding, refuting the behaviourist "cupboard love" / drive-reduction theory.
2. The 1973 Nobel Prize in Physiology or Medicine went to **Lorenz, Tinbergen and von Frisch** (imprinting, four questions, bee waggle dance), the only Nobel awarded for ethology.
3. Tinbergen's four questions split into proximate (causation + development = how) and ultimate (function + evolution = why); a complete account answers all four.
4. The psychiatry chain: Lorenz imprinting → Harlow contact comfort → Bowlby evolved attachment (secure base, monotropy, internal working model, maternal deprivation) → Ainsworth Strange Situation (secure / avoidant / ambivalent / disorganised).
5. Trap: vacuum activity = a FAP firing with **no stimulus at all** (drive overflow), whereas displacement activity = an **irrelevant** act during **motivational conflict**.

1 Foundations and the founders

Ethology is the study of animal behaviour in its **natural environment**, read through an evolutionary lens. It grew up in Europe against the American school of **comparative psychology**, and the contrast is itself examinable.

	Ethology	Comparative psychology
Roots	European zoology (Lorenz, Tinbergen)	American experimental psychology (Watson, Skinner)
Setting	natural field; observe undisturbed	controlled laboratory
Emphasis	innate, species-specific, instinctive behaviour	learned behaviour, general laws across species
Method	description, the ethogram, evolution	manipulation, conditioning, the rat and pigeon

● RULE

The two traditions later converged: behaviour is neither purely innate nor purely learned but the product of both, shaped by evolution and tuned by experience. Modern ethology and behavioural ecology absorbed the laboratory rigour of comparative psychology.

The three founders and the 1973 Nobel

◆ **1973 Nobel Prize** – the **Nobel Prize in Physiology or Medicine** was shared by **Konrad Lorenz, Niko Tinbergen and Karl von Frisch** "for their discoveries concerning organisation and elicitation of individual and social behaviour patterns." The only Nobel awarded for ethology.

Founder	Signature contribution	Remember by
Konrad Lorenz	imprinting in geese; fixed action patterns; the innate releasing mechanism; co-father of ethology	the greylag goslings following him
Niko Tinbergen	the **four questions**; sign stimuli and supernormal stimuli; the stickleback and herring-gull experiments	the four questions
Karl von Frisch	decoded the honeybee **waggle dance** as a symbolic communication of food direction and distance	the dancing bees

Tinbergen's four questions

Tinbergen (1963) argued that any behaviour can be explained at four complementary levels. A complete account answers all four; exams reward keeping the proximate pair separate from the ultimate pair.

Question	Asks	Level	Time-frame
Causation (mechanism)	what stimulus and machinery trigger it *now*?	proximate	here and now
Development (ontogeny)	how does it arise over the individual's lifetime?	proximate	the lifespan
Function (survival value)	how does it aid survival and reproduction?	ultimate	fitness payoff
Evolution (phylogeny)	how did it evolve across the species' history?	ultimate	evolutionary past

● MNEMONIC

"Causation, Development, Function, Evolution"

Two **proximate** (how it works: causation + development) and two **ultimate** (why it exists: function + evolution). Proximate = the mechanism in this animal; ultimate = the evolutionary reason across the lineage.

HOLD THIS

Founders = **Lorenz, Tinbergen, von Frisch** (1973 Nobel). Four questions = **causation, development** (proximate) and **function, evolution** (ultimate). These two lists are the section's marks.

2 Instinct and innate behaviour

Classical ethology built a vocabulary for behaviour that is **inherited, stereotyped and species-typical** – performed correctly the first time, without learning. The terms interlock: a sign stimulus trips a releasing mechanism that runs off a fixed action pattern.

- **Fixed action pattern (FAP)** – a **stereotyped, innate** sequence of movements, run to completion once started (ballistic). Classic example: the greylag goose **egg-rolling** response – remove the egg mid-roll and the goose still completes the empty retrieving motion.
- **Sign stimulus / releaser** – the specific cue that triggers a FAP. A **releaser** is a sign stimulus used in *social* communication between members of a species. The male stickleback attacks anything bearing a **red belly**; the herring-gull chick pecks at the **red spot** on the parent's bill to beg food.
- **Innate releasing mechanism (IRM)** – the hypothesised neural "filter" that recognises the sign stimulus and releases the matching FAP. Stimulus → IRM → fixed action pattern.

◆ **Supernormal stimulus** – an **exaggerated** version of the sign stimulus that triggers a *stronger* response than the natural one. Birds prefer to brood a giant dummy egg over their own; gull chicks peck hardest at a thin red rod with white bands, more than at a real bill. Tinbergen's concept; widely cited for junk food, pornography and outsized advertising as supernormal human stimuli.

● **Vacuum activity** – a FAP performed in the **complete absence** of its normal stimulus, as if the drive overflows. A hand-reared starling with no insects will still run the full catch-and-kill sequence on empty air.

● **Displacement activity** – an **irrelevant** behaviour appearing during conflict or thwarted motivation (approach vs avoidance). A bird in a territorial standoff suddenly preens or pecks the ground. Read as the root of human self-soothing tics under stress.

● **Ritualisation** – the evolutionary process by which a behaviour (often a displacement or intention movement) becomes **stylised and exaggerated** to serve as a communicative signal, e.g. courtship and threat displays.

● TRAP

Distinguish the two "wrong-place" behaviours. Vacuum activity = the FAP fires with **no stimulus at all** (drive overflow). Displacement activity = an **irrelevant** behaviour appears during **motivational conflict**. Different cause, different exam answer.

3 Imprinting and early experience

Some learning happens only inside a narrow developmental window and then locks. Lorenz's geese are the textbook case and the conceptual bridge to attachment.

● **Filial imprinting (Lorenz)** – newly hatched precocial birds (geese, ducks) form an attachment to and follow the **first moving object** they see, normally the mother. Lorenz's hand-reared greylag goslings imprinted on **him** and followed him instead.

◆ **Critical / sensitive period** – imprinting occurs only within a brief window after hatching (classically the **first 13–16 hours**, the response strongest around 13–16 h in ducklings). Miss the window and it does not happen.

■ **Irreversibility** – once formed, the imprint is **stable and hard to reverse**, distinguishing it from ordinary learning. (Later work softened "critical" to "sensitive" and showed some plasticity, but the exam answer is: rapid, time-locked, lasting.)

● **Sexual imprinting** – early experience also sets later **mate preference**: an animal reared by another species may court that species in adulthood. The window for sexual imprinting differs from the filial one.

● RULE

Imprinting is the bridge to psychiatry: a rapid, time-windowed, lasting attachment to a caregiver figure. It anticipates Bowlby's claim that human attachment too has a sensitive period and lasting consequences – though human attachment is far more flexible and not a single fixed window.

4 The comparative roots of attachment

This is the high-yield section for psychiatry. Two programmes – Harlow's monkeys and Bowlby's theory – converted ethology into a model of the human bond and its disorders.

Harlow's rhesus monkeys

Harry Harlow reared infant rhesus monkeys with two surrogate "mothers" to ask whether the infant bonds for **food** or for **comfort**. The answer overturned the prevailing "cupboard love" (drive-reduction) theory.

Surrogate	Property	Infant's behaviour
Wire mother	bare wire; **held the feeding bottle**	visited only to feed, then left
Cloth mother	soft terry-cloth; **no food**	clung to it most of the day; ran to it when frightened

◆ **Contact comfort** – infants preferred the **cloth** mother that gave no food over the **wire** mother that fed them. The bond is built on **comfort and tactile contact**, not feeding. The cloth mother also served as a **secure base** from which to explore and a haven when startled.

▲ **Social isolation** – monkeys reared in **total isolation** showed lasting damage: fear, self-clutching and rocking, social and sexual deficits, and (in the "motherless mothers") abusive or neglectful parenting. Some recovery was possible with younger "therapist" monkeys; severe, prolonged isolation was largely irreversible.

● CLASSIC TRAP

Harlow's headline finding is **contact comfort over feeding**. It refutes the behaviourist "cupboard love" idea that the infant loves the mother because she is paired with food. This is the empirical anchor for attachment theory and the harm of early deprivation.

Bowlby's ethological attachment theory

◆ **Attachment as an evolved system** – John Bowlby recast attachment as a **biologically programmed survival system**: the infant is innately driven to maintain proximity to a caregiver because proximity protected ancestral infants from predators. Crying, clinging and following are inbuilt **releasers** of caregiving – ethology applied to humans.

● **Secure base** – the attachment figure is a base from which the child **explores** and a **safe haven** to return to under threat. Drawn straight from Harlow's cloth mother.

● **Monotropy & internal working model** – a bias to form one primary bond (monotropy) within a sensitive period; from it the child builds an **internal working model** of self and relationships that shapes later bonds.

▲ **Maternal deprivation** – Bowlby held that prolonged disruption of early attachment risks lasting emotional harm (the "44 thieves" study linking it to affectionless psychopathy). Later critiqued and refined (Rutter: *privation* vs deprivation; multiple attachments matter), but the core claim shaped child psychiatry.

Into developmental psychology

● **Ainsworth: the Strange Situation** – Mary Ainsworth operationalised attachment quality in a laboratory separation-and-reunion procedure, yielding **secure, insecure-avoidant and insecure-ambivalent/resistant** types (later **disorganised**, Main & Solomon). The behavioural test of Bowlby's theory.

QUICK RULE

The chain for a psychiatry answer: **Lorenz (imprinting) → Harlow (contact comfort) → Bowlby (evolved attachment, secure base) → Ainsworth (Strange Situation, attachment styles)**. Animal ethology becomes a theory of human bonds and their disorders.

5 Learning, evolution and social behaviour

The final strand links ethology to learning theory, the evolution of social life, and modern evolutionary psychiatry.

Biological preparedness

- **Preparedness (Seligman)** – animals are evolutionarily **primed** to learn survival-relevant associations easily and resist others (the opposite extreme is "contraprepared"). Explains why phobias cluster on snakes, spiders, heights and the dark rather than plugs or cars.
- **Garcia effect (taste aversion)** – a conditioned aversion to a food forms after a **single** pairing with later nausea, even across a delay of **hours**, and links readily to *taste* but not to sights or sounds. It breaks the standard conditioning rules of contiguity and equipotentiality – a prepared, survival-tuned form of learning.

Social structure

- **Dominance hierarchy** – a ranked social order (the "**pecking order**," Schjelderup-Ebbe in hens) that reduces costly within-group fighting over food, mates and space; rank is settled by display more than by combat.
- **Territoriality** – defence of an area for feeding, nesting or mating; advertised by song, scent or display so disputes are settled **without** constant fighting.

Altruism: the central puzzle

Why would an animal help others at a cost to itself, when natural selection should favour the selfish? Two answers solve it.

- **Kin selection (Hamilton)** – genes for altruism spread if the helper aids **relatives** who share those genes. **Inclusive fitness** counts an individual's own reproduction plus its effect on kin. **Hamilton's rule**: altruism is favoured when **rB > C** (relatedness × benefit to recipient exceeds cost to actor). Haldane's quip: "I would lay down my life for two brothers or eight cousins."
- **Reciprocal altruism (Trivers)** – helping **non-relatives** pays if the favour is likely to be returned later ("you scratch my back…"), given repeated interaction, recognition and memory. The substrate for cooperation among strangers.
- **Sociobiology (E. O. Wilson)** – Wilson's 1975 synthesis applied evolutionary biology systematically to **social behaviour**, including the human; controversial for its implications about human nature, it seeded modern evolutionary psychology.

Evolutionary psychiatry

- **Attachment & psychopathology** – disrupted early attachment is a developmental risk factor for later personality, mood and relational disorder – the clinical descendant of Harlow and Bowlby.
- **Social rank theory of depression** – depression read as an evolved **involuntary subordinate / yielding** response to defeat or entrapment in a status contest (Price, Gilbert). Low mood de-escalates conflict with dominant rivals; it becomes pathological when escape is blocked.
- **Anxiety as an adaptive alarm** – fear and anxiety are an evolved **defence / smoke-detector** system. False alarms are cheap and missed threats are deadly, so the system is biased to over-fire – anxiety disorders are this alarm mistuned or chronically triggered.

● RULE

The evolutionary frame does not say a disorder is "good"; it says the underlying capacity (vigilance, social appraisal, yielding) was adaptive, and pathology is that capacity misfiring or stuck. This reframes symptoms as dysregulated defences, not random malfunctions.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Founders:** Lorenz (imprinting), Tinbergen (four questions, sign/supernormal stimuli), von Frisch (bee waggle dance) shared the **1973 Nobel** in Physiology or Medicine.
- **Four questions (Tinbergen):** causation + development = **proximate** (how); function + evolution = **ultimate** (why).
- **Instinct kit:** sign stimulus / releaser → IRM → fixed action pattern (ballistic). Supernormal = exaggerated cue, stronger response.
- **Two odd ones:** vacuum activity = FAP with **no stimulus**; displacement activity = irrelevant act during **conflict**. Ritualisation = behaviour stylised into a signal.
- **Imprinting (Lorenz):** precocial birds follow the first moving object; critical/sensitive period (~first 13–16 h); rapid, irreversible. Sexual imprinting sets mate preference.
- ★ **Harlow:** contact comfort over feeding – cloth (no food) beats wire (food). Isolation causes lasting social/emotional damage. Refutes "cupboard love."
- **Bowlby:** attachment = evolved survival system; secure base, monotropy, internal working model; maternal deprivation. → Ainsworth Strange Situation (secure / avoidant / ambivalent / disorganised).
- **Prepared learning:** Seligman preparedness (phobia content); Garcia effect = one-trial taste aversion over a delay, taste-specific.
- **Social:** dominance hierarchy (pecking order), territoriality – both cut fighting.
- **Altruism:** kin selection (Hamilton, inclusive fitness, **rB > C**); reciprocal altruism (Trivers); sociobiology (E. O. Wilson, 1975).
- **Evolutionary psychiatry:** attachment → psychopathology; social-rank theory of depression (yielding to defeat); anxiety = adaptive smoke-detector alarm, over-tuned.

ONE-DAY REVISION · PAPER 1

Biostatistics, made retrievable

Don't memorise formulas you won't compute. Hold what each thing is, when it is used, and how to read it. Every section is a 10-mark answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The p-value is the probability of the observed result (or more extreme) *if H_0 were true*, not the probability that H_0 is true; p<0.05 → reject H_0, and a 95% CI crossing 0 (a difference) or 1 (OR/RR) means not significant.
2. SnNOUT & SpPIN: a Sensitive test Negative rules OUT, a Specific test Positive rules IN; sensitivity and specificity are intrinsic, while PPV and NPV move with prevalence (as prevalence falls, PPV falls and NPV rises).
3. Match the test to the data: 2 independent groups → unpaired t / Mann-Whitney, 2 paired → paired t / Wilcoxon, 3+ groups → ANOVA / Kruskal-Wallis, categorical → chi-square (Fisher exact if expected <5).
4. Design fixes the measure: cohort / RCT → RR & incidence, case-control → OR (approximates RR only when disease is rare), cross-sectional → prevalence; RCT is the gold standard for controlling confounding.
5. Right (positive) skew → mean > median > mode, so report median + IQR; and SD is spread of individuals while SE = SD/√n is precision of the mean (always smaller, shrinks as n grows).

1 Types of data

The data type decides which test you use. Often the first thing asked.

Family	Scale	Examples	Key point
Qualitative	Nominal – no order	sex, diagnosis, blood group	count / compare proportions
(categorical)	Ordinal – ordered, unequal gaps	Likert, CGI, MMSE bands	order yes, distance no
Quantitative	Interval – no true zero	temperature °C, IQ	0 is arbitrary
(numerical)	Ratio – true zero	age, weight, no. of episodes	ratios meaningful

● MNEMONIC

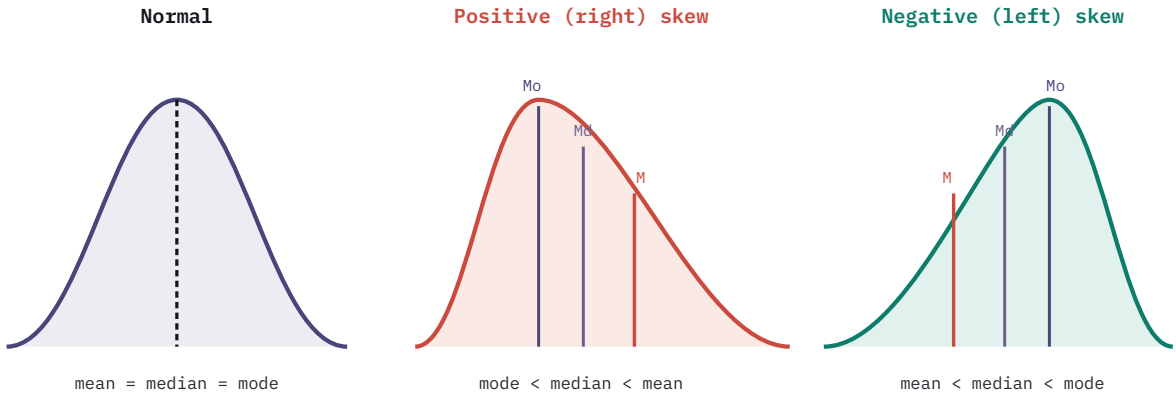
"NOIR" → Nominal, Ordinal, Interval, Ratio

Down the ladder = increasing information. Also: **discrete** (counts, e.g. admissions) vs **continuous** (any value, e.g. serum lithium).

2 Central tendency & skew

Measure	What	Best for	Watch
Mean	sum / n	symmetrical numeric data	dragged by outliers
Median	middle value	skewed data, ordinal	ignores extremes (a strength)
Mode	most frequent	nominal data	may be none or several

WHERE MEAN, MEDIAN & MODE SIT UNDER SKEW



The skew is named for the **tail**; the mean is pulled toward it. Lines mark mode, median, mean.

HOLD THIS

Skewed data → report **median + IQR**. Right (positive) skew → **mean > median > mode**.

3 Dispersion (spread)

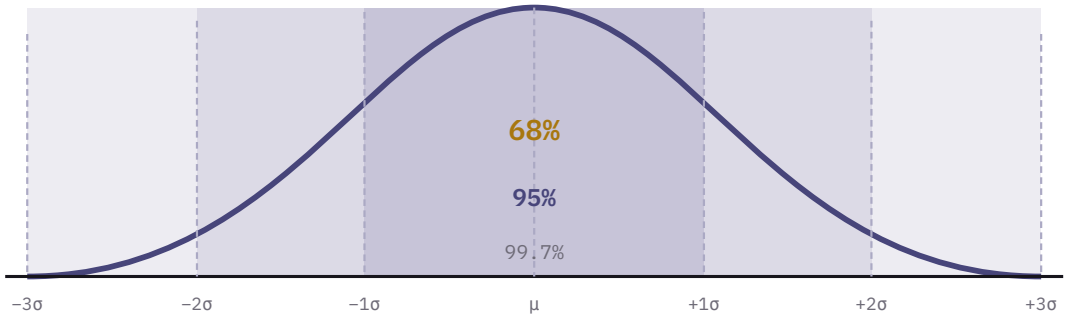
Measure	Meaning	Note
Range	max − min	crude, outlier-sensitive
IQR	Q3 − Q1 (middle 50%)	pairs with median; robust
Variance	mean of squared deviations	in squared units
SD	√variance; avg distance from mean	same units as data; pairs with mean
SE	SD / √n; spread of the sample *mean*	shrinks as n grows; used for CIs

● CLASSIC TRAP

SD = spread of **individuals**. SE = SD/√n = precision of the **mean estimate**. SE is always smaller and falls as sample size rises.

4 The normal distribution

THE 68 / 95 / 99.7 RULE



Bell, symmetrical, **mean = median = mode**. ±1σ holds 68%, ±2σ 95%, ±3σ 99.7%.

- **Shape** – defined by mean (μ) and SD (σ). Skewness = asymmetry; kurtosis = peakedness.
- **Test choice** – assumes normality → **parametric**. Not normal (skewed / ordinal / small n) → **non-parametric**.

5 Sampling methods

Probability (random)

Allows generalisation.
Simple random – equal chance.
Systematic – every kth from a random start.
Stratified – sample within strata (ensures representation).
Cluster – sample whole groups.
Multistage – clusters then sub-sample.

Non-probability

Convenient, biased, no true generalisation.
Convenience, **purposive** (judgmental), **quota**, and **snowball** (one subject recruits the next, for hidden populations such as people who inject drugs).

6 Study designs & the evidence ladder

Design	Direction	Measures	Caveat
Cross-sectional	one time point	prevalence	quick; no causality
Case-control	look back (outcome→exposure)	odds ratio	rare disease; recall bias
Cohort	follow forward (exposure→outcome)	incidence, RR	temporal; long, costly
RCT	randomised + blinded	efficacy	controls confounding; gold standard

HIERARCHY OF EVIDENCE · TOP = STRONGEST

QUICK RULE

RR or incidence → **cohort** · OR → **case-control** · prevalence → **cross-sectional**.

7 Hypothesis testing

- **Null vs alternative** – H_0 = no difference; H_1 = there is one. You *reject* or *fail to reject* H_0; you never "prove" H_1.
- **p-value** – probability of the result (or more extreme) *if H_0 were true*. p<0.05 → reject H_0. **Not** the probability that H_0 is true.
- **95% CI** – more informative than p. CI for a difference crossing **0**, or for OR/RR crossing **1** → not significant.

THE TWO ERRORS

	Truth: H_0 true	Truth: H_0 false
Reject H_0	Type I (α) false +ve	correct · power
Fail to reject	correct	Type II (β) false −ve

- **Power = 1 − β** – aim ≥ 0.80. Rises with larger sample, bigger effect, lower variability, higher α.

MNEMONIC

Type I: "I saw something that wasn't there" · Type II: "missed it"

8 Which test, when

PARAMETRIC VS NON-PARAMETRIC

Comparison	Parametric (normal)	Non-parametric
2 groups, **independent**	unpaired t-test	Mann-Whitney U
2 groups, **paired**	paired t-test	Wilcoxon signed-rank
3+ groups	ANOVA (F-test)	Kruskal-Wallis
Categorical	Chi-square (Fisher's exact if expected <5)	
Association, 2 numeric	Pearson r	Spearman ρ
Predict an outcome	linear / logistic regression	

● RULE

ANOVA compares means of 3+ groups (avoids the inflated Type I error of multiple t-tests). Significant ANOVA → **post-hoc** tests (Tukey, Bonferroni) to find which groups differ. Correlation ≠ causation.

9 Sensitivity, specificity & predictive values

THE 2×2 TABLE · DRAW THIS IN THE EXAM

	Disease +	Disease –
Test +	TP	FP
Test –	FN	TN

Term	Formula	Plain meaning
Sensitivity	TP / (TP+FN)	of those **with** disease, % caught (down Disease+ column)
Specificity	TN / (TN+FP)	of those **without**, % cleared (down Disease– column)
PPV	TP / (TP+FP)	if test +, chance truly diseased. Falls as prevalence falls
NPV	TN / (TN+FN)	if test –, chance truly clear. Rises as prevalence falls

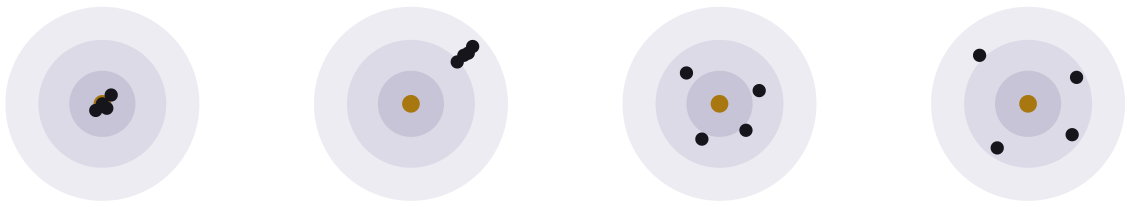
● MNEMONIC

SnNOUT · SpPIN

Snensitive test, **N**egative rules **OUT** · **Sp**ecific test, **P**ositive rules **IN**. Sensitivity & specificity are **intrinsic**; PPV & NPV **move with prevalence**.

10 Reliability vs validity

THE DARTBOARD PICTURE



Reliable + Valid
tight, on target

Reliable, not Valid
tight, off target

Valid, not Reliable
scattered, avg on target

Neither
scattered, off target

Reliability = consistency (tight grouping). **Validity** = accuracy (on the bullseye). A test can be reliable but not valid; never meaningfully the reverse.

Concept	Types	Measure
Reliability	test-retest, inter-rater, internal consistency, split-half	Cohen's κ (categorical) · ICC (continuous) · Cronbach's α (scale, >0.7)
Validity	face, content, criterion (concurrent + predictive), construct	correlation with a gold standard

11 Risk measures

Measure	From	Interpretation
Relative risk (RR)	cohort / RCT	>1 harmful, <1 protective, =1 no effect
Odds ratio (OR)	case-control	approximates RR when disease is rare
ARR	RCT	control rate − treated rate
NNT = 1 / ARR	RCT	patients treated to prevent 1 event; lower = better

● TRAP

A confidence interval for OR or RR that **crosses 1** → not statistically significant.

QUICK-RECALL · DRILL THESE ONE-LINERS

- Skewed → **median + IQR**; normal → **mean + SD**. Right skew → mean > median > mode.
- **68 / 95 / 99.7** for ±1/2/3 SD. SD = individuals; **SE = SD/ $\sqrt{n}$** = precision of the mean.
- p<0.05 = reject null; p ≠ probability null is true. CI crossing **0** (difference) or **1** (OR/RR) → not significant.
- Type I = false +ve (α); Type II = false −ve (β); **Power = 1−β**.
- 2 indep → unpaired t / Mann-Whitney · paired → paired t / Wilcoxon · 3+ → ANOVA / Kruskal-Wallis · categories → chi-square.
- **SnNOUT · SpPIN**. Sens/spec intrinsic; PPV/NPV move with prevalence.
- Cohort → RR / incidence · case-control → OR · cross-sectional → prevalence.
- κ = inter-rater (categorical) · Cronbach's α = internal consistency · ICC = continuous reliability.

ONE-DAY REVISION · PAPER 1

Eminent contributors & eponyms

The names pack. Who first described what, which era it belongs to, and the one syndrome or scale that carries the name. Indian theory papers reward exactly this: "eminent contributors to psychiatry" and "short note on [eponymous syndrome]." Drill the attribution, not the prose.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Where a year is uncertain it is given as a decade or omitted, never guessed. Psychology theorists live in the companion pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Kraepelin's term was dementia praecox; Eugen Bleuler renamed it "schizophrenia" (1908) and gave the 4 A's (Association, Affect, Ambivalence, Autism), while Schneider gave the first-rank symptoms → never credit the term to Kraepelin, never swap the A's with FRS.
2. Capgras = same face, a familiar person replaced by an impostor; Fregoli = different faces, one disguised persecutor; and Charles Bonnet is NOT a delusion (insight preserved, visual hallucinations in the visually impaired).
3. Lesion-to-eponym pairs: Gerstmann = dominant angular gyrus tetrad (agraphia, acalculia, finger agnosia, left-right disorientation); Balint = bilateral parieto-occipital triad (simultanagnosia, optic ataxia, oculomotor apraxia); Kluver-Bucy = bilateral anterior temporal/amygdala.
4. Only two psychiatry Nobel laureates: Wagner-Jauregg (malaria/fever therapy for general paresis, 1927) and Moniz (prefrontal leucotomy, 1949); and lithium = Cade 1949, chlorpromazine = Delay & Deniker 1952, ECT = Cerletti & Bini 1938.
5. Scale authorship and rating-type: Beck (BDI) = self-rated; Hamilton (HAM-D/HAM-A), Young (YMRS), and Montgomery-Asberg (MADRS) = clinician-rated; MMSE = Folstein, scored /30.

1 Founders & classifiers of psychiatry

The line runs from moral treatment, through descriptive classification, to the great divide over schizophrenia. Hold the **sequence** and the one contribution attached to each name.

Contributor	Contribution	Era
Philippe Pinel	"unchained" the insane at Bicetre / Salpetriere; **moral treatment**; *traitement moral*	late 1700s
Jean-Etienne Esquirol	Pinel's pupil; distinguished hallucination from illusion; described monomania; first dedicated psychiatry course	early 1800s
Wilhelm Griesinger	"mental diseases are brain diseases"; founder of biological psychiatry	mid 1800s
Karl Kahlbaum	described **catatonia**; named *cyclothymia*; champion of the clinical-syndrome method	late 1800s
Ewald Hecker	described **hebephrenia** (with Kahlbaum)	late 1800s
Emil Kraepelin	the great dichotomy: **dementia praecox vs manic-depressive insanity**; classification by course and outcome	1890s onward
Eugen Bleuler	coined **"schizophrenia"** (1908); the **4 A's**; coined *autism* and *ambivalence*	early 1900s

Contributor	Contribution	Era
Kurt Schneider	**first-rank symptoms** of schizophrenia; the bipolar/unipolar emphasis on form over content	1930s–50s
Karl Jaspers	*General Psychopathology*; phenomenology; **form vs content**; understanding vs explaining; the un-understandable primary delusion	1910s onward

Bleuler vs Schneider on schizophrenia

Eugen Bleuler – the 4 A's

The **fundamental** symptoms, present in all cases. Hallucinations and delusions were "accessory."
Association loosening · **A**ffect (blunted/incongruent) · **A**mbivalence · **A**utism.
(Some add a 5th: **A**ttention.)

Kurt Schneider – first-rank symptoms

Pragmatic, not theoretical: if present and no organic cause, **strongly suggest** schizophrenia. **Audible thoughts** (thought echo) · voices arguing · running commentary · thought withdrawal/insertion/**broadcast** · made acts/affect/impulses (passivity) · somatic passivity · **delusional perception**.

MNEMONIC

Bleuler's "4 A's" vs Schneider's "FRS"

Bleuler = **fundamental** (theory-driven, the A's). Schneider = **first-rank** (pragmatic, diagnostic pointers). Neither is pathognomonic; FRS also occur in mania and organic states.

The classifiers around the schizophrenia concept

- **Jacob Kasanin** – coined **"schizoaffective"** psychosis (1933), for cases mixing affective and schizophrenic features with good prognosis.
- **Gabriel Langfeldt** – split **"process" schizophrenia** (true, poor outcome) from **schizophreniform psychosis** (good prognosis); the source of the DSM "schizophreniform" term.
- **Karl Leonhard** – elaborate classification of endogenous psychoses; described **cycloid psychoses** and the systematic/unsystematic schizophrenias; first to separate **bipolar from unipolar** depression.
- **Eugen vs Manfred Bleuler** – **Eugen** (father) coined schizophrenia and the 4 A's. **Manfred** (son) ran long-term outcome studies showing a more benign, heterogeneous course than Kraepelin's pessimism implied.

TRAP

Do not credit the *term* schizophrenia to Kraepelin. Kraepelin's term was **dementia praecox**; **Eugen Bleuler** renamed it schizophrenia (a "splitting" of psychic functions, not split personality). And the 4 A's belong to **Bleuler**, the first-rank symptoms to **Schneider** – the most frequently swapped pair in vivas.

2 Psychoanalysis & the dynamic schools

One line each. The exam wants the single idea most tied to the name, and who broke from whom.

Contributor	Core contribution	School / link
Josef Breuer	the "talking cure" with Anna O.; **cathartic method**; co-authored *Studies on Hysteria* with Freud	pre-analytic
Sigmund Freud	founder of psychoanalysis; the unconscious, **id/ego/superego**, psychosexual stages, transference, defences, dream interpretation	classical
Carl Gustav Jung	**analytical psychology**; collective unconscious, archetypes, persona/shadow/anima/animus, introversion–extraversion, individuation	broke from Freud

Contributor	Core contribution	School / link
Alfred Adler	**individual psychology**; inferiority complex, striving for superiority, style of life, social interest, birth order	broke from Freud
Melanie Klein	**object relations** founder; play technique; paranoid-schizoid & depressive positions; projective identification	object relations (British)
Anna Freud	systematised the **ego defence mechanisms**; child analysis; rival of Klein in the "Controversial Discussions"	ego psychology
Donald Winnicott	**good-enough mother**, transitional object, holding environment, true vs false self	British object relations
John Bowlby	**attachment theory**; the secure base; maternal deprivation	attachment
Margaret Mahler	**separation-individuation**; the phases of normal autism, symbiosis, and the rapprochement subphase	developmental object relations
Heinz Kohut	**self psychology**; narcissism, selfobjects, empathic mirroring; the "tragic man"	self psychology
Erik Erikson	**8 psychosocial stages** across the lifespan; identity crisis; epigenetic principle	ego psychology
Harry Stack Sullivan	**interpersonal theory**; the self-system; "security operations"; chumship	interpersonal / neo-Freudian
Karen Horney	**basic anxiety**; the three neurotic trends (moving toward/against/away); critique of Freud's penis-envy ("womb envy")	neo-Freudian
Ronald Fairbairn	radical **object relations**: libido is object-seeking, not pleasure-seeking; endopsychic structure	British object relations

● RULE

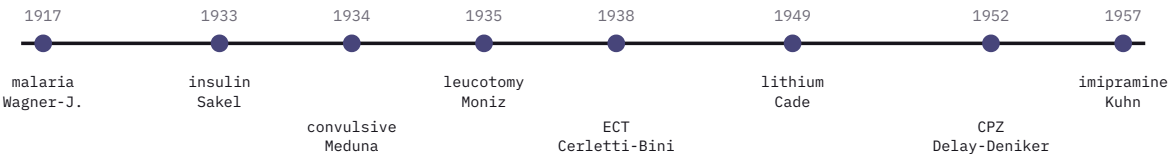
The two who **broke from Freud** are **Jung** (collective unconscious, archetypes) and **Adler** (inferiority, social interest). The British object-relations cluster is **Klein → Fairbairn → Winnicott**. Ego psychology is **Anna Freud → Erikson → Hartmann**. Self psychology is **Kohut** alone.

3 Biological & psychopharmacology pioneers

The discovery dates are high-yield. Most were serendipitous. Hold the **drug, the person, and the year**.

Discovery	Contributor(s)	Year
Malaria (fever) therapy for general paresis (neurosyphilis)	**Julius Wagner-Jauregg** – the only psychiatrist to win a Nobel Prize (1927)	1917
Insulin coma therapy	**Manfred Sakel**	1933
Convulsive therapy (pharmacological, with cardiazol/metrazol)	**Ladislas Meduna**	1934
Prefrontal leucotomy (lobotomy)	**Egas Moniz** – Nobel Prize 1949	1935–36
Electroconvulsive therapy (ECT)	**Ugo Cerletti & Lucio Bini**	1938
Lithium for mania	**John Cade** (Australia)	1949
Chlorpromazine (first antipsychotic)	**Jean Delay & Pierre Deniker**; synthesised by Charpentier, surgeon **Henri Laborit** noted its calming effect	1952
Iproniazid (first MAOI antidepressant)	**Nathan Kline** (and colleagues)	mid 1950s
Imipramine (first tricyclic antidepressant)	**Roland Kuhn** (Switzerland)	1957

THE SOMATIC-THERAPY TIMELINE, 1917–1957



The somatic era: fever, insulin and convulsive therapies (1917–1938), then the pharmacology revolution: lithium (1949), chlorpromazine (1952), imipramine (1957).

HOLD THIS

Two Nobel laureates: **Wagner-Jauregg** (malaria therapy, 1927) and **Moniz** (leucotomy, 1949). Lithium = **Cade 1949**. Chlorpromazine = **Delay & Deniker 1952**. ECT = **Cerletti & Bini 1938**.

● TRAP

Meduna introduced *chemically* induced convulsions (cardiazol); **Cerletti & Bini** introduced *electrically* induced convulsions (ECT). Both rested on the now-disproven idea that epilepsy and schizophrenia were biologically antagonistic. Don't merge the two: Meduna = chemical, Cerletti-Bini = electrical.

4 Delusional, misidentification & descriptive eponyms

Classic “short note” territory. One def atom each: the phenomenon and, where it is an eponym, the person it is named for.

- **Capgras syndrome** – the delusion that a familiar person (or pet/object) has been replaced by an identical **impostor** (the “illusion of doubles”). Named for Joseph Capgras (1923). The commonest delusional misidentification syndrome.
- **Fregoli syndrome** – the delusion that **different people are actually one persecutor in disguise**, changing appearance. Named after the quick-change actor Leopoldo Fregoli.
- **Intermetamorphosis** – the belief that people **swap identities** with each other, exchanging both face and personality. A rarer misidentification variant.
- ▲ **Cotard syndrome** – nihilistic delusion of being **dead, decaying, or non-existent** (le delire de negation), sometimes with delusions of immortality. Named for Jules Cotard. Seen in severe psychotic depression.
- **De Clerambault syndrome (erotomania)** – the delusion that a person, usually of **higher status**, is secretly in love with the patient. Named for Gaetan Gaitan de Clerambault.
- **Othello syndrome** – **delusional (morbid) jealousy**: fixed false belief in a partner's infidelity. Named after Shakespeare's character; a forensic risk for violence.
- **Ekbom syndrome** – **delusional parasitosis**: the unshakeable belief of being infested by insects/parasites under the skin. Named for Karl-Axel Ekbom. (Ekbom also described *restless legs syndrome*, sometimes called Ekbom syndrome too – context disambiguates.)

▲ **Ganser syndrome** – the "syndrome of **approximate answers**" (Vorbeireden): near-misses to simple questions (2+2=5), clouding, pseudohallucinations and conversion features. Named for Sigbert Ganser. Classed under dissociative disorders.

● **Couvade syndrome** – the **expectant father** develops pregnancy-like symptoms (nausea, weight gain, abdominal swelling) sympathetically with his partner. From French *couver*, to brood/hatch.

● **Charles Bonnet syndrome** – **complex visual hallucinations in the visually impaired**, with *preserved insight* and no psychiatric illness. Named for the Swiss naturalist Charles Bonnet.

● **Diogenes syndrome** – **severe self-neglect, domestic squalor and hoarding**, often with social withdrawal and no shame, typically in the elderly. Named (somewhat ironically) after Diogenes the Cynic.

◆ **Folie a deux** – **shared (induced) delusional disorder**: a delusion transferred from a dominant person (primary, *inducer*) to a passive, close contact (secondary). Described by Lasegue & Falret (1877). Separation often resolves it in the secondary.

● CLASSIC TRAP

Sort the misidentifications by the rule: **Capgras** = same face, different (impostor) person. **Fregoli** = different faces, same (disguised) person. **Intermetamorphosis** = identities swapped, face changes too. **Charles Bonnet** is NOT a delusion: insight is preserved and there is no psychosis – the catch most candidates miss.

5 Eponymous neuropsychiatric syndromes

Lesion or deficit, the eponym, and the one localising fact. High-yield for organic psychiatry.

● **Kluver-Bucy syndrome** – **bilateral anterior temporal lobe / amygdala** damage. Hyperorality, hypersexuality, placidity (loss of fear/rage), hypermetamorphosis (touching everything), and **visual agnosia** (psychic blindness). Described by Heinrich Kluver & Paul Bucy.

● **Wernicke encephalopathy** – acute **thiamine (B1) deficiency**: the triad of **confusion, ophthalmoplegia/nystagmus, and ataxia**. Lesions in mammillary bodies and periaqueductal grey. A medical emergency. Named for Carl Wernicke.

▲ **Korsakoff syndrome (psychosis)** – the **chronic amnestic** sequel of Wernicke: anterograde > retrograde amnesia and **confabulation**, with relatively preserved other cognition. Named for Sergei Korsakoff. The pair = **Wernicke-Korsakoff syndrome**.

● **Anton syndrome** – **denial of cortical blindness** (anosognosia for blindness): the patient is blind from bilateral occipital lesions but insists they can see and confabulates visual scenes. Named for Gabriel Anton.

● **Balint syndrome** – **bilateral parieto-occipital** lesions: the triad of **simultanagnosia** (cannot perceive a whole scene), optic ataxia (mis-reaching under vision), and oculomotor apraxia (impaired voluntary gaze shift). Named for Rezso Balint.

◆ **Gerstmann syndrome** – **dominant (left) angular gyrus** lesion: the tetrad of **agraphia, acalculia, finger agnosia, and left-right disorientation**. Named for Josef Gerstmann.

● **Geschwind syndrome** – the interictal personality cluster of **temporal lobe epilepsy**: **hyperreligiosity, hypergraphia, hyposexuality, and viscosity** ("stickiness") of thought. Described by Norman Geschwind.

● **Kleine-Levin syndrome** – recurrent **hypersomnia** with hyperphagia and hypersexuality ("Sleeping Beauty syndrome"), typically in adolescent males, in episodic bouts. Named for Willi Kleine & Max Levin.

- **Gilles de la Tourette syndrome** – childhood-onset **multiple motor tics plus at least one vocal tic** for over a year; coprolalia is classic but uncommon. Named for Georges Gilles de la Tourette (pupil of Charcot).

LOCALISATION RULE

Gerstmann = dominant angular gyrus (a 4-sign tetrad). **Balint** = bilateral parieto-occipital (a 3-sign triad). **Anton** = bilateral occipital (denial of blindness). **Kluver-Bucy** = bilateral temporal/amygdala. **Wernicke** = mammillary bodies (thiamine). Memorise the lesion next to the eponym – that is the mark.

6 Assessment & scale eponyms

The instrument, who built it, and what it rates. Note clinician-rated vs self-rated – a favourite distinction.

Scale	Author(s)	Measures / type
HAM-D (HDRS)	Max **Hamilton**	depression severity; clinician-rated
HAM-A	Max **Hamilton**	anxiety severity; clinician-rated
BDI	Aaron **Beck**	depression; self-rated
YMRS	**Young** et al.	mania severity; clinician-rated
MADRS	**Montgomery & Asberg**	depression; clinician-rated; sensitive to change
PCL-R	Robert **Hare**	psychopathy; clinician-rated (20 items)
MMSE	**Folstein, Folstein & McHugh**	global cognition; /30
BPRS	**Overall & Gorham**	psychotic symptom severity; clinician-rated
PANSS	**Kay, Fiszbein & Opler**	positive, negative & general schizophrenia symptoms
PSE / SCAN	John **Wing** et al.	structured psychiatric interview (Present State Examination; later SCAN)

MNEMONIC

"Beck the self, Hamilton the clinic"

Beck (BDI) = self-rated. **Hamilton (HAM-D/HAM-A)** and **Montgomery-Asberg (MADRS)** = clinician-rated. MMSE is out of **30**. PANSS = three subscales (positive/negative/general).

TRAP

Spelling counts in vivas: it is **Asberg** (Marie Asberg) in MADRS, and the MMSE is **Folstein** (with McHugh). The PCL-R is **Hare's** psychopathy checklist, not a depression scale – don't confuse it with a mood instrument.

7 Indian psychiatry pioneers

A compact, high-confidence row. Indian theory papers sometimes ask for "contributions of Indian psychiatrists" or the history of an institution.

- **Vidya Sagar** – pioneered **group / family involvement in treatment** at the Amritsar mental hospital, encouraging relatives to stay with and care for patients in tents on the hospital grounds; an early indigenous model of family-based and milieu therapy.
- **NIMHANS, Bangalore** – the **National Institute of Mental Health and Neuro Sciences**, formed by merging the Mysore State Mental Hospital with the All India Institute of Mental Health; today an Institute of National Importance and apex centre for mental health.

- **CIP, Ranchi** – the **Central Institute of Psychiatry**, founded in the colonial era as the Ranchi European Lunatic Asylum; one of India's oldest and foremost training and research centres in psychiatry.
- **Professional bodies** – the **Indian Psychiatric Society (IPS)** is the national professional body; the **Indian Journal of Psychiatry** is its official journal.

● VERIFY-LOCALLY FLAG

This section is kept deliberately minimal and attribution-safe: the institutions and Vidya Sagar's family-participation model at Amritsar are well established, but exact founding *years* and individual "founder" names vary across sources, so they are omitted here rather than guessed. If your local notes or department history give specific dates/founders, prefer those for the answer.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Founders:** Pinel (unchained, moral treatment) · Griesinger ("mind diseases = brain diseases") · Kahlbaum (catatonia) · Hecker (hebephrenia) · Kraepelin (dementia praecox vs manic-depressive) · **Eugen Bleuler** coined schizophrenia + 4 A's · Schneider = first-rank symptoms · Jaspers = phenomenology, form vs content.

- **Schizophrenia divide:** Bleuler 4 A's (Association, Affect, Ambivalence, Autism, fundamental) vs Schneider FRS (passivity, thought alienation/broadcast, delusional perception, third-person voices). Kasanin = schizoaffective · Langfeldt = schizophreniform · Leonhard = cycloid + bipolar/unipolar split.
- **Dynamic schools:** Breuer (catharsis, Anna O.) · Freud (id/ego/superego) · Jung (collective unconscious) · Adler (inferiority) · Klein (object relations, positions) · Anna Freud (defences) · Winnicott (good-enough mother) · Bowlby (attachment) · Mahler (separation-individuation) · Kohut (self psychology) · Erikson (8 psychosocial stages) · Sullivan (interpersonal) · Horney (basic anxiety) · Fairbairn (libido is object-seeking).
- **Somatic / pharma:** Wagner-Jauregg malaria 1917 (Nobel) · Sakel insulin 1933 · Meduna convulsive 1934 · Moniz leucotomy 1935 (Nobel) · Cerletti-Bini ECT 1938 · Cade lithium 1949 · Delay-Deniker chlorpromazine 1952 · Kline iproniazid MAOI · Kuhn imipramine 1957.
- **Misidentification:** Capgras (impostor, same face) · Fregoli (one disguised persecutor) · intermetamorphosis (identities swapped) · Cotard (nihilistic, "I am dead") · de Clerambault (erotomania) · Othello (morbid jealousy) · Ekbom (delusional parasitosis) · Ganser (approximate answers) · Couvade (sympathetic pregnancy) · Charles Bonnet (insight-intact visual hallucinations in the blind) · Diogenes (self-neglect/squalor) · folie a deux (shared delusion).
- **Neuropsychiatric:** Kluver-Bucy (bilateral temporal, hyperorality/hypersexuality/placidity) · Wernicke (thiamine, confusion-ataxia-ophthalmoplegia) · Korsakoff (amnesia + confabulation) · Anton (denies blindness) · Balint (parieto-occipital triad) · Gerstmann (angular gyrus tetrad) · Geschwind (TLE personality) · Kleine-Levin (recurrent hypersomnia) · Tourette (motor + vocal tics).
- **Scales:** Hamilton (HAM-D/A, clinician) · Beck (BDI, self) · Young (YMRS, mania) · Montgomery-Asberg (MADRS) · Hare (PCL-R, psychopathy) · Folstein (MMSE /30) · Overall-Gorham (BPRS) · Kay-Fiszbein-Opler (PANSS) · Wing (PSE/SCAN).
- **India:** Vidya Sagar (family-participation model, Amritsar) · NIMHANS Bangalore (apex centre) · CIP Ranchi (oldest training/research) · IPS + Indian Journal of Psychiatry.