

Cambridge AS & A Level · Psychology 9990 · Notes

Mood (affective) disorders A2

Read online at pastopic.com/cambridge/psychology-9990/notes/mood-disorders

Last checked 6 October 2026

Read first: [Research methods](#)

© 2026 Pastopic. Free for personal study. Please share the link, not the file.

What you need to know

Mood (affective) disorders are the second disorder in the Clinical psychology option (Papers 3 and 4). You should be able to:

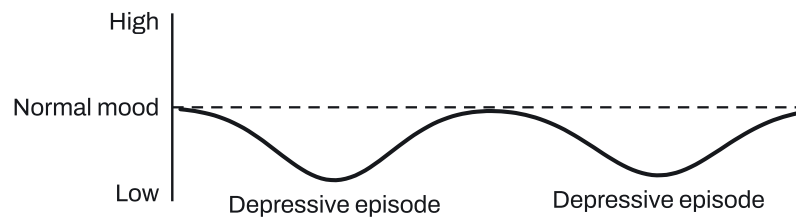
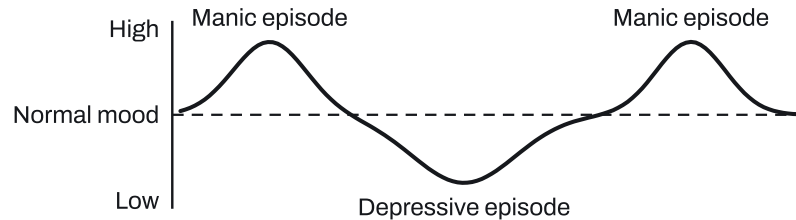
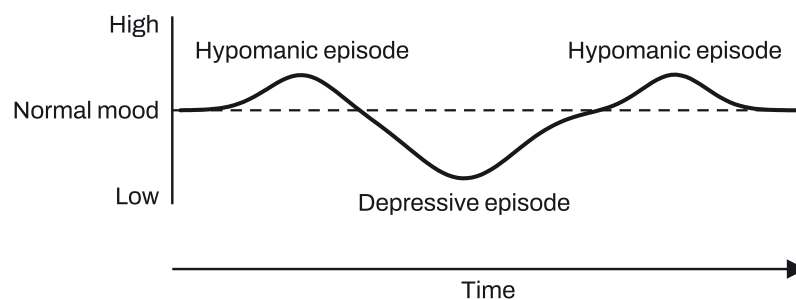
1. **Diagnose them with ICD-11:** the criteria for **depressive disorder (unipolar)** and for **bipolar disorders**, including what **manic**, **hypomanic**, **mixed** and **depressive episodes** are.
2. **Describe the Beck depression inventory (BDI):** its items, how it is scored and how the total is interpreted.
3. **Explain depression biologically:** the **biochemical** explanation and the **genetic** explanation, using the key study by **Oruč et al. (1997)**.
4. **Explain depression psychologically:** **Beck's cognitive theory** and **learned helplessness / attributional style**, with a study such as **Seligman et al. (1988)**.
5. **Describe biological treatments:** antidepressants (**tricyclics**, **MAOIs** and **SSRIs**).
6. **Describe psychological therapies:** **Beck's cognitive restructuring** and **Ellis's rational emotive behaviour therapy (REBT)**.
7. **Evaluate all of it**, using the issues, debates and methods the syllabus names: individual and situational explanations, cultural differences, quantitative and qualitative data, psychometrics, validity, nature versus nurture, reductionism versus holism, determinism versus free will, experiments, reliability, application to everyday life and generalisations from findings.

Understand it

What a mood disorder is

A **mood (affective) disorder** is more than ordinary ups and downs: the mood is extreme, lasts for weeks, and stops the person coping with daily life.

- **Depressive disorder (unipolar):** mood only ever goes **down**, in one or more depressive episodes. "Unipolar" means one pole.
- **Bipolar disorder:** mood swings to **both poles**. The person has highs (mania or hypomania) and usually lows (depression), with episodes lasting days to months, not minutes or hours.

Depressive disorder (unipolar)**Bipolar type I disorder****Bipolar type II disorder****ICD-11: depressive episodes and depressive disorder**

ICD-11, the World Health Organization's classification, groups the symptoms of a **depressive episode** into three clusters:

Cluster	Symptoms
Affective (mood)	depressed mood (sad, empty, irritable); markedly reduced interest or pleasure in activities
Cognitive-behavioural	poor concentration or indecisiveness; low self-worth or excessive guilt ; hopelessness about the future; recurrent thoughts of death or suicide
Neurovegetative (body)	disturbed sleep (too little or too much); changed appetite or weight; slowed or restless movement; low energy and fatigue

A depressive episode needs **at least 5 of these 10** symptoms, **at least one** of them from the affective cluster, present most of the day, nearly every day, for **at least 2 weeks**, and causing real difficulty in daily life. Episodes are graded mild, moderate or severe.

Depressive disorder (unipolar) is one episode (single episode) or more (recurrent). It is **never** diagnosed if the person has **ever** had a manic, mixed or hypomanic episode: that would make it bipolar.

ICD-11: mania and bipolar disorders

Episode	What it is	How long
Manic	an extreme state of euphoria , irritability or expansiveness with much more activity and energy, plus symptoms such as pressured speech , racing thoughts (flight of ideas), grandiosity , little need for sleep , distractibility and reckless behaviour (spending, gambling, risky sex)	at least 1 week (less if treatment cuts it short)
Hypomanic	a milder version of mania: raised mood or irritability and energy, noticeable to others, but without serious harm to functioning	several days
Mixed	several manic and several depressive symptoms at once, or switching very fast (within a day)	at least 2 weeks
Depressive	as above	at least 2 weeks

- **Bipolar type I: one or more manic or mixed episodes.** Most people also have depressive episodes, but one manic episode is enough for the diagnosis.
- **Bipolar type II: one or more hypomanic episodes and at least one depressive episode**, and **no** manic or mixed episode ever. The highs are less severe; the depressions are what usually bring the person to a doctor.
- **Cyclothymic disorder** is the mild, long-lasting form: about two years of unstable mood with many hypomanic and depressive symptoms that never reach a full episode.

"Feeling happy" or "being moody" is **not** mania. Mania needs named, extreme symptoms such as euphoria, racing thoughts or hardly sleeping.

Measuring depression: the Beck depression inventory (BDI)

The BDI (Beck, 1961) is a **psychometric self-report questionnaire** that measures **how severe** a person's depression is.

- **21 items**, each a symptom such as sadness, pessimism, guilt, self-dislike, crying, loss of interest, tiredness, sleep and appetite.
- Each item has four statements scored **0 to 3**; the person picks the one that fits how they have felt (over the **past two weeks** in the BDI-II; the original asked about the past week). For the sadness item, the statements go from not feeling sad at all (0), through feeling sad some of the time (1) and all the time (2), to being so sad it is unbearable (3).
- Add the 21 scores: the total runs from **0 to 63** (21×3). The higher it is, the more severe the depression.

Total	Interpretation (the bands mark schemes use)
1–10	normal ups and downs
11–16	mild mood disturbance
17–20	borderline clinical depression

Total	Interpretation (the bands mark schemes use)
21–30	moderate depression
31–40	severe depression
over 40	extreme depression

The BDI has been revised. The **BDI-II (1996)** dropped items on body image, weight loss, health worries and work difficulty, added items such as agitation, worthlessness, concentration and loss of energy, and lets sleep and appetite change in **either** direction (more or less). The exact band cut-offs differ a little between versions.

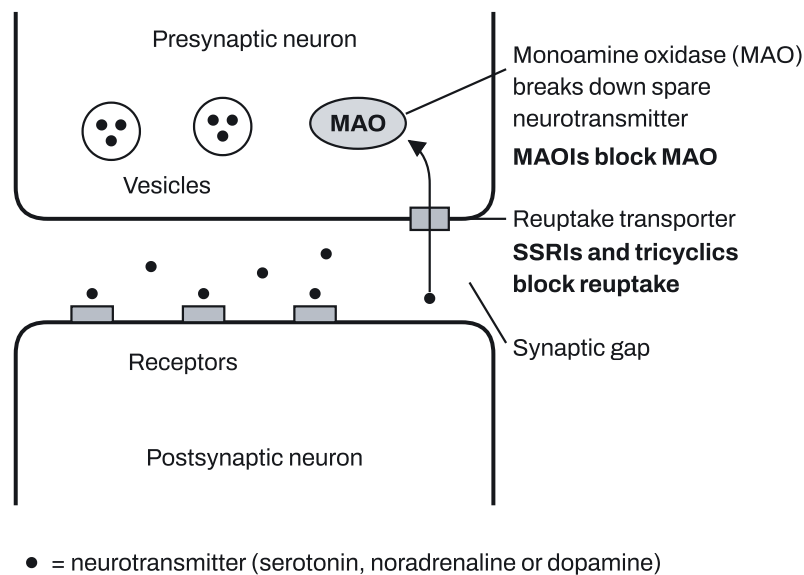
Evaluating diagnosis and the BDI

Issue	For	Against
Validity	ICD-11 is written by experts, updated, and lists clear symptoms and durations. The BDI covers 21 symptoms, both cognitive (guilt) and physical (sleep), uses 4 options rather than yes/no, matches the ICD-11 symptoms, and its scores correlate with other depression scales such as the Hamilton scale	Depression often comes with other disorders (comorbidity), so it is hard to say whether it is one disorder. Clinicians may be biased , e.g. diagnosing women more readily. Patients may hide or exaggerate symptoms (social desirability , embarrassment)
Reliability	Fixed criteria and the same 21 items for everyone mean two clinicians should reach the same result	Clinicians must judge "most of the day" or "marked"; a person's BDI answers can change from day to day
Quantitative and qualitative data	The BDI gives a number : easy to compare across people or before and after treatment, and to analyse with statistics	A number shows how bad, not why. Diagnosis by clinical interview gives qualitative detail about the person's life, but needs interpretation
Psychometrics	A standardised test lets a score be compared with the normal population	People read the 0–3 statements differently; the score does not say how to treat the problem
Idiographic versus nomothetic	ICD-11 and the BDI are nomothetic : the same rules for everyone	Each person's depression is unique (idiographic); a fixed list may miss what matters most to them
Individual and situational	Both treat depression as a state of the individual	Low mood can be a reaction to a situation (bereavement, job loss); the BDI does not ask why
Cultural differences	ICD-11 is used in many countries, so diagnoses can be compared	In some cultures depression is described mainly through physical complaints, and other countries use DSM-5, whose criteria are similar but not identical

Explanation 1: biochemical

Neurons pass messages across a **synapse** using **neurotransmitters**. Serotonin, noradrenaline and dopamine are called **monoamines**. After a message is sent, spare neurotransmitter is taken back into the first neuron (**reuptake**) and broken down by the enzyme **monoamine oxidase (MAO)**.

The biochemical explanation says depression comes from **too little monoamine activity**, especially **serotonin** and **noradrenaline**; mania has been linked to too much activity. **Evidence for:** antidepressants raise monoamine levels and lift mood in many patients. **Evidence against:** the drugs change levels within hours but mood takes **weeks** to improve, not everyone responds, and the research is **correlational**, so low serotonin could be a **result** of depression rather than its cause.



Explanation 2: genetic

Mood disorders, bipolar disorder above all, run in families: a person with an affected **first-degree relative** (parent, sibling or child) has a higher risk. Because serotonin is linked to mood, researchers looked for variations in **serotonin genes**.

Key study: Oruč et al. (1997)

Aim: to find out whether variations (**polymorphisms**) in two serotonin genes, the **serotonin receptor 2c gene (5-HT_{2c})** and the **serotonin transporter gene (5-HTT)**, are associated with **bipolar type I disorder**.

Sample (learn at least four features):

Group	Detail
Patients	42 unrelated patients with bipolar type I disorder : 25 female and 17 male , aged 31–70
Where from	in-patients and out-patients of the REBRO and Vrapče psychiatric hospitals in Zagreb, Croatia

Group	Detail
Family history	16 of the 42 had at least one first-degree relative with a major affective disorder
Controls	40 healthy people (25 female, 15 male), age- and sex-matched , from the hospital staff and their friends , with no history of mental illness

Procedure: patients were given a **clinical interview** by a psychiatrist (a face-to-face meeting to diagnose and assess), which used the **SADS-L** (Schedule for Affective Disorders and Schizophrenia, Lifetime version) and asked about their relatives' mental health. Diagnoses were checked against **medical records** and reviewed by a **second psychiatrist**. Each participant's **DNA** was analysed for the polymorphisms in both genes, and patients were compared with controls.

Results: **no significant association** was found between either gene and bipolar disorder in the patient sample as a whole. When men and women were analysed **separately** (serotonin works a little differently in the two sexes), there were **trends** towards an association for **both** polymorphisms in **female** patients.

Conclusion: variations in these serotonin genes may give a **small increase in susceptibility** to bipolar disorder in **women**; genes are, at most, one part of the cause.

Evaluation: DNA analysis is **objective** and **quantitative**, the procedure is **standardised** and could be repeated (**reliable**), the **control group** gives a baseline, and diagnoses were checked in more than one way. But the sample was **small** and all **Croatian**, so it is hard to **generalise**; relatives' diagnoses came from patients' reports and records made at different times; and the results were only **trends**, so the genetic link is weak. The study is about bipolar disorder, not unipolar depression.

Explanation 3: Beck's cognitive theory

Beck argued that depression comes from **faulty information processing**: the depressed person thinks in biased, negative ways.

- **Negative schemas:** ways of seeing things, formed by early bad experiences such as criticism or loss, that are triggered later by stress ("I am unlovable").
- **The negative cognitive triad:** negative views of **the self** ("I'm worthless"), **the world** ("everyone is against me") and **the future** ("it will never get better"). Each feeds the others, so mood spirals down.
- **Cognitive biases:** errors such as **overgeneralising** (one failure means "I fail at everything"), **selective attention** to the bad, **magnifying** problems and **personalising** (blaming yourself for things you didn't cause).
- These produce **negative automatic thoughts**: quick, unquestioned thoughts such as "they didn't text back because they don't like me".

Explanation 4: learned helplessness and attributional style

Learned helplessness: in early experiments by Seligman, dogs that had been given electric shocks they could not escape later **did not try** to escape shocks they could have avoided. They had learned that nothing they did made a difference. Applied to people: after repeated failures or bad events that seem **uncontrollable**, a person learns they are helpless, **stops trying** to improve things, and becomes depressed.

Attributional style (Abramson, Seligman and Teasdale, 1978) adds **how a person explains bad events**. A **pessimistic** style explains them as:

Dimension	Pessimistic (depression-prone)	Optimistic
Internal or external	"It's my fault"	"It was the situation"
Stable or unstable	"It will always be like this"	"It was a one-off"
Global or specific	"It ruins everything"	"It's just this one thing"

Study: Seligman et al. (1988)

- **Aim:** to see whether attributional (explanatory) style is linked to depression, and whether it changes as depression lifts in cognitive therapy.
- **Participants:** 39 outpatients with **unipolar depression** and 12 with **bipolar disorder** in a depressed phase (mean age about 36), from the same clinic, plus a small **non-clinical** comparison group.
- **Measures:** a **short form of the BDI** and the **Attributional Style Questionnaire (ASQ)**. The ASQ gives 12 **imagined events** (6 good, 6 bad); the person writes the main cause of each and rates that cause on 7-point scales for how **internal**, **stable** and **global** it is.
- **Procedure:** the unipolar patients were tested **before** cognitive therapy, **at the end** of it and **one year later**. It is a **quasi-experiment**: patients could not be randomly allocated to being depressed.
- **Results:** a **pessimistic** style for **bad** events (internal, stable, global) **correlated** with how **severe** the depression was, at all three times. As therapy went on, **explanatory style became less pessimistic and depression fell**, and both stayed improved a year later. The bipolar patients showed the same pattern, but less strongly.
- **Conclusion:** the way people explain bad events is an important part of depression, and changing it helps recovery.

Biological treatments: antidepressants

Drug type	Example	How it works	Problems
Tricyclics (from the 1950s)	amitriptyline	block reuptake of noradrenaline and serotonin , so more stays in the synapse	dry mouth, constipation, blurred vision, drowsiness, weight gain; dangerous in overdose

Drug type	Example	How it works	Problems
MAOIs (monoamine oxidase inhibitors, from the 1950s)	phenelzine	inhibit the enzyme MAO , which normally breaks down noradrenaline , serotonin and dopamine , so their levels stay high	interact with other medicines and with foods rich in tyramine (aged cheese, cured meat, some wines, fermented soya), which can cause a hypertensive crisis (dangerously high blood pressure); headache, drowsiness, insomnia. Now mostly used when other antidepressants have failed
SSRIs (selective serotonin reuptake inhibitors, from the late 1980s)	fluoxetine (Prozac), sertraline	block reuptake of serotonin only	fewer side effects, but nausea, headaches, sleep problems, sexual problems; take several weeks to work

All antidepressants have to be taken every day for months; stopping suddenly can bring withdrawal effects or relapse. They treat the **symptoms** (the chemistry), not life events or thinking. Effectiveness is tested in **randomised controlled trials (RCTs)**: patients are **randomly allocated** to the drug or a placebo, often **double blind**, and depression is measured (e.g. with the BDI) before and after.

Psychological therapy 1: Beck's cognitive restructuring

A talking therapy, usually about **6–12 sessions**, that changes the faulty thinking in Beck's theory.

1. The therapist **explains** the therapy and the cognitive triad.
2. Together they **identify** unpleasant emotions, the **situations** where they happen, and the **negative automatic thoughts** that go with them. The client keeps a **diary** as homework.
3. The therapist **challenges** each thought with questions: "What is the evidence? Is there another explanation?" (**reality testing**). The client may test a thought, e.g. by phoning a friend they believe dislikes them.
4. The negative thoughts are **replaced** with more balanced ones ("I failed this test, but I passed the last three").
5. Deeper **dysfunctional beliefs** (schemas) are challenged, then therapy ends, and the client can use the skills alone.

It also helps **bipolar** disorder: the therapist challenges the **overly positive** thoughts of mania ("I can't fail") as well as the negative ones of depression. **Dobson (1989)**, in a meta-analysis, found cognitive therapy did better on average than drug treatment.

Psychological therapy 2: Ellis's rational emotive behaviour therapy (REBT)

Ellis (1962) built on the ancient **Stoic** idea that events do not upset us directly; what upsets us is **how we see them**. REBT uses the **ABCDE** model:

Step	Meaning	Example
A	Activating event	Not being picked for the team
B	Belief about A (the key step)	"I must be picked or I'm worthless"
C	Consequences: feelings and behaviour	Low mood, quitting sport
D	Disputing the irrational belief	"Where is it written that you must? Does one decision make you worthless?"
E	Effect: a new, rational belief and healthier feelings	"I'm disappointed, but I can train and try again"

Irrational beliefs Ellis named include **awfulising** ("it would be a total disaster"), **I-can't-stand-it-itis** (low frustration tolerance), **self-downing** ("I am a failure") and **musturbation** ("I must always succeed"). The therapist disputes them **actively and directly**, with **logical** ("does that follow?"), **empirical** ("where's the evidence?") and **pragmatic** ("does believing it help you?") questions. The client practises disputing between sessions and learns to **accept** themselves, others and life as imperfect. REBT focuses on **present** beliefs, not the past.

Evidence: Lyons and Woods (1991) combined 70 REBT studies in a meta-analysis: clients who had REBT improved significantly more than control groups. Iftene et al. (2015) found REBT as effective as antidepressant medication for young people with depression.

Issues and debates

Debate	How it applies to mood disorders
Nature versus nurture	Genes (Oruč) and neurotransmitters are nature ; schemas learned from early experience, learned helplessness and stressful events are nurture . Oruč's weak results and the role of life events suggest both matter.
Reductionism versus holism	The biochemical and genetic explanations, and drugs, are reductionist : one chemical or gene. Beck and attributional style look at complex thought processes, so they are more holistic , but still ignore biology. REBT accepts that events (A) matter as well as beliefs (B).
Determinism versus free will	Genes and neurotransmitters are biologically determined . Learned helplessness is environmental determinism. Cognitive restructuring and REBT assume free will : the client can choose to change their thinking. Taking a drug or not is also a choice.
Individual versus situational	Biochemistry, genes, negative schemas and attributional style are individual : two people in the same situation react differently. Life events such as bereavement or redundancy are situational . Drugs and therapy treat the individual.
Application to everyday life	Drugs are cheap and easy to take but have side effects and food rules. Therapies have no side effects and teach lasting skills, but cost more, need a trained therapist, and need motivation , which depressed people often lack; REBT's direct disputing can feel confrontational.

Debate	How it applies to mood disorders
Generalisations from findings	Meta-analyses show treatments work on average, but individuals differ: not everyone responds to a drug or engages with therapy. Oruč's small Croatian sample is hard to generalise.
Experiments and reliability	Seligman is a quasi-experiment, so its findings are correlational; using the same BDI and ASQ at three points is standardised and replicable. RCTs of drugs use random allocation and blinding.

Key facts and formulas

There is no formula list in the 9990 exam. "Learn it" means you must remember it.

Fact	Status
ICD-11 depressive episode: at least 5 of 10 symptoms (at least one of depressed mood or loss of interest or pleasure), most of the day, nearly every day, for at least 2 weeks; never any manic, mixed or hypomanic episode for depressive disorder	Learn it
ICD-11 bipolar type I: one or more manic or mixed episodes (manic episode at least 1 week). Bipolar type II: one or more hypomanic episodes (several days) plus at least one depressive episode, never a manic episode	Learn it
Mania symptoms: euphoria or irritability, more energy and activity, pressured speech, racing thoughts, grandiosity, little need for sleep, distractibility, reckless behaviour	Learn it
Beck depression inventory (BDI): 21 items, each scored 0–3, total 0–63; 1–10 normal, 11–16 mild, 17–20 borderline, 21–30 moderate, 31–40 severe, over 40 extreme	Learn it
Oruč et al. (1997): 42 bipolar I patients (25 female, 17 male, aged 31–70, Zagreb, Croatia; 16 with an affected first-degree relative) and 40 age- and sex-matched controls; DNA for 5-HTT; no overall association, trends for both in women	Learn it
Beck's negative cognitive triad: negative views of the self, the world and the future	Learn it
Pessimistic attributional style for bad events: internal, stable, global	Learn it
Seligman et al. (1988): 39 unipolar and 12 bipolar outpatients; short BDI and ASQ (12 events, 6 good and 6 bad, 7-point scales) before, at the end of and a year after cognitive therapy; pessimistic style correlated with severity, and both improved	Learn it
Antidepressants: tricyclics block reuptake of noradrenaline and serotonin; MAOIs inhibit monoamine oxidase (tyramine foods risk a hypertensive crisis); SSRIs block reuptake of serotonin only	Learn it
REBT (Ellis): Activating event, Belief, Consequences, Disputing, Effect; Lyons and Woods (1991) meta-analysis of 70 studies	Learn it

Fact	Status
Percentage = $\frac{\text{part}}{\text{total}} \times 100$	Learn it

How to do it

We have tagged 41 questions on this topic, from 2021 to 2026 (Papers 3 and 4; 29 different questions, as variants repeat them). 29 of them have parts of more than one type, so the counts add up to 84. Questions before 2024 sometimes use the old syllabus (ECT for depression, "bipolar and related disorders"): learn the current content, and use those questions for the skill they test.

Question type	Questions
Describe content for an essay (AO1, 6–8 marks)	9
Evaluate in an essay, including a named issue (AO3, 10 marks)	9
Outline or describe a treatment (drugs, ECT, REBT, cognitive restructuring)	9
Explain a strength or weakness of a treatment or the BDI	8
Evaluate the method of a key study (sample, controls, clinical interview, RCT)	7
Describe a key study (Oruč et al. or Seligman et al.)	6
Define bipolar disorder or mania	6
Plan a study and justify its choices	6
Discuss strengths and weaknesses with a conclusion (5 marks)	6
Outline a psychological explanation of depression	5
Apply knowledge to a named patient	4
Explain an issue or debate applied to mood disorders	4
Measure depression: BDI scoring or another measure	3
Compare two treatments (similarity and difference)	2

1. Describe content for an essay (AO1, 6–8 marks) (9 questions)

Paper 3 part (a) names two things (ICD-11 criteria and the BDI; MAOIs and REBT; Beck and learned helplessness) or one study. Each is marked separately and the two levels are added, so describing **only one** caps the part at 3 or 4 of the 6 marks.

1. **Diagnosis:** unipolar and bipolar criteria with durations, a clear definition of **mania**, and the difference between **type I and type II**. **BDI:** 21 items, 0–3, an example item with its statements, the total out of 63 and the bands.

2. **Treatments**: how each **works** and how it **reduces symptoms** (MAO enzyme and the three neurotransmitters; ABC plus D and E), with one or two named findings (Lyons and Woods).
3. **Explanations**: the triad, schemas and automatic thoughts; helplessness plus internal, stable, global attributions.
4. **Oruč**: sample (numbers, groups), procedure (interview, DNA, two genes) **and** a result. Full marks need all three.
5. Keep evaluation out of part (a), and don't drift into explanations when diagnosis is asked (March 2026 report).

2. Evaluate in an essay, including a named issue (AO3, 10 marks) (9 questions)

Each evaluation point is levelled, then the best fit is given. Leaving out the named issue limits the mark, and so does writing about the named issue alone (June 2023 report).

1. **Go issue by issue**, named issue first, and compare **both** items under each issue (June 2023 and March 2026 reports).
2. Use **context from part (a)** in every point: the tyramine foods, the 21 items, the 7-point ASQ, Lyons and Woods.
3. Add **analysis**: a contrast between the two items, a "however", or research evidence.
4. Choose issues that fit: the tables "Evaluating diagnosis and the BDI" and "Issues and debates" give both sides of each. Nature versus nurture does not fit an essay on diagnosis and the BDI (March 2026 report), and evaluating the BDI earns nothing when the question is about explanations.

3. Outline or describe a treatment (drugs, ECT, REBT, cognitive restructuring) (9 questions)

2–4 marks: name it, then explain **how it reduces depression**.

- **REBT**: A, B and C alone earn 1 mark; you need **D (disputing)** and **E (effect)** for 2 (March 2023 and March 2025 reports). "Why must a client's thinking change?": because it is the **belief** about an event, not the event, that causes the low mood (November 2021 report). "An irrational belief": name one of Ellis's and give an example ("self-downing: I am a failure").
- **Cognitive restructuring**: the stages, with an example thought challenged and replaced. Describing the triad alone is an explanation, not a treatment (November 2021 report).
- **Drugs**: MAOIs block the MAO enzyme so noradrenaline, serotonin and dopamine stay high; SSRIs stop serotonin being taken back up.
- **ECT** (old syllabus questions, depression or schizophrenia): anaesthetic and **muscle relaxant**; electrodes **unilateral or bilateral**; a brief current causes a **seizure**, seen only as twitching; the patient wakes and recovers; usually a course over several weeks.
- "Another treatment" or "another psychological treatment": any named one in detail. Plain "CBT" with no detail earns nothing, and ECT or drugs are not psychological.

4. Explain a strength or weakness of a treatment or the BDI (8 questions)

2 marks a point: the point (1) plus an example, evidence or link to the treatment (1).

- **REBT strengths:** clients can dispute beliefs **anywhere, any time** once taught; **Lyons and Woods (1991)** showed it beats control groups; **Iftene et al. (2015)** found it as good as drugs; no side effects.
- **Cognitive restructuring:** no side effects (unlike antidepressants); lasting skills for future relapses; short (6–12 sessions); but needs motivation, which severe depression takes away, and costs therapist time (June 2021 report).
- **Drug weakness:** name the problem **and** what it is ("MAOIs can cause a hypertensive crisis if the patient eats aged cheese"). "Addictive" or "side effects" alone earns 1 (March 2023 report). It must be for the **same drug** you described.
- **BDI weaknesses:** assumes everyone has the same 21 symptoms (**nomothetic**); quantitative, so the person's unique experience is lost. A bare "social desirability" earns nothing.
- **BDI validity:** wide range of cognitive and physical symptoms; 4 choices, not yes/no; matches ICD-11; agrees with other scales. Not reliability.

5. Evaluate the method of a key study (sample, controls, clinical interview, RCT) (7 questions)

- **Why Oruč had a control group:** a **baseline** of people with **no bipolar disorder** and **no affected first-degree relatives**, so gene variations in patients could be compared.
- **Strengths of Oruč's sample** (not "large", and not the sampling technique, March 2025 report): in- and out-patients were **known** to the hospitals, so they reliably met the criteria; the hospital was a **familiar** place, so they felt more at ease; all were **clinically diagnosed**; controls were **age- and sex-matched**; males and females with a **wide age range** (31–70).
- **Clinical interview:** a face-to-face meeting between a doctor and patient to diagnose or assess. Oruč used it to **confirm the diagnosis**, ask about **relatives**, and give the **SADS-L**. Strength: the patient knows their own history and family and can explain. Weakness: they may be vague, misremember, or not understand terms like "first-degree relative".
- **Without a second psychiatrist**, a wrong diagnosis might go unnoticed, so unsuitable patients could be included and the data would be invalid.
- **Seligman's controls** (levels, 6 marks): the same BDI and ASQ at all three times (standardised, reliable, change measurable); unipolar and bipolar groups compared; but the non-clinical group was only a baseline with no therapy, and as a **quasi-experiment** the results are correlational.
- **RCT for MAOIs:** randomly allocate patients to MAOI or placebo, so each has an equal chance of either; strength: the researcher cannot bias who gets the drug, so participant variables are spread evenly.

6. Describe a key study (Oruč et al. or Seligman et al.) (6 questions)

One mark per accurate detail; full marks need the **sample**, **how data were collected** and **a result**. Learn the two study sections in "Understand it". Oruč studied **bipolar** disorder, not depression, and is not Gottesman and Shields (November 2022 report). Seligman et al. (1988) is the attributional style study with patients, not the dog experiments (June 2023 report).

7. Define bipolar disorder or mania (6 questions)

2 marks.

- **Bipolar disorder**: two contrasting poles of mood (1): episodes of **mania** (high) and of **depression** (low), each lasting weeks or months (1).
- **Mania**: two named symptoms, 1 mark each: euphoria, irritability, racing thoughts, pressured speech, little need for sleep, grandiosity, risky behaviour. "Happy", "excited" or "sleeps badly" earn nothing.
- **Two characteristics**: one from the high phase and one from the low phase.

8. Plan a study and justify its choices (6 questions)

Since 2024: (a) a plan [10], (b)(i) the psychology it is based on [4], (b)(ii) how two features shaped the plan [4], (c) three 2-mark reasons or weaknesses, each applied. See the research-methods note for the method features.

1. **Use the named method**: a questionnaire question needs a questionnaire, not an experiment (March 2025 report).
2. **Specific features**: technique (postal, online), format (open and closed), **example questions** that use the topic, **scoring**, number of questions; or IV, DV, design, controls, blinding for an experiment. Then sample, sampling, ethics, data.
3. **Make the content specific**: for REBT, ask about disputing ("When you have an upsetting thought, how often do you challenge it? always / sometimes / never"), not "did you like the therapy?"; for drugs, say which drug each group gets and for how long (weeks, not one week), with the BDI as the DV (June 2022 report); for attributional style, model the ASQ's good and bad events and 7-point scales.
4. **(b)(i)**: the theory or treatment (REBT's D and E, how MAOIs and SSRIs work, the ASQ), not methods. The BDI does not measure attributional style. Give the full name before an acronym (March 2025 report).
5. **(b)(ii)**: link each feature to a line in your plan. **(c)**: quote your own plan in each answer.

9. Discuss strengths and weaknesses with a conclusion (5 marks) (6 questions)

Old Paper 4: 1 mark per strength or weakness about the named thing (up to 4, at least two of each), plus 1 for a **conclusion** that makes a judgement, not a summary. Relate each point: "side effects such as memory loss after ECT", not just "side effects" (March 2023 report).

- **Drugs**: easy (a pill), work for many; but side effects, non-adherence, treat symptoms not causes.

- **Psychological treatments:** no medication or addiction, lasting skills; but take time and effort, need a paid therapist. Use Beck or Ellis as examples (November 2021 report).
- **ECT** (for depression or schizophrenia): used when drugs fail and helps many; but nobody knows how it works, memory loss, can be given without consent.
- **Clinical interviews:** patients know their family history and can explain; but may exaggerate or misremember, and the interview is subjective.

10. Outline a psychological explanation of depression (5 questions)

- **Learned helplessness** (2): after uncontrollable bad events a person believes nothing they do can change things (1), so they stop trying and become depressed (1). The dog study gets credit only if it explains this.
- **Beck** (2): faulty, negative thinking (1) plus the triad or an example of a biased thought (1). Don't just list symptoms of depression (November 2022 report).
- **"Other than biological"** (4): Beck or attributional style in detail, e.g. internal, stable and global attributions for bad events leading to helplessness.

11. Apply knowledge to a named patient (4 questions)

Use the patient's details in **every** point.

- **Unipolar or bipolar?** Bipolar shows **manic or mixed episodes** (give the symptoms and that it lasts at least a week); unipolar mood stays **low**, with no highs.
- **Cognitive restructuring for bipolar:** challenge **both** the over-positive manic thoughts and the negative depressive ones, quoting the patient's own words; one pole only caps the mark.
- **Using the BDI:** 21 items scored 0–3; which items this patient would score highly on (from their symptoms); the band their likely total falls in.

12. Explain an issue or debate applied to mood disorders (4 questions)

(a) define the debate (1–2); (b) link it to the explanation. Repeating the question's words earns nothing.

- **Learned helplessness is individual:** it depends on the person's own perception of control; two people in the same situation, one feels able to act and the other gives up.
- **Determinism versus free will:** how far behaviour is caused by forces beyond our control or chosen by us. A **strength of determinism** for the biochemical explanation: if low serotonin causes depression, it can be treated (with SSRIs), and the patient is not **blamed**.
- **"Nature versus nurture is irrelevant"** (old 12-mark essay): argue it is relevant (it guides research and treatment: drugs for nature, therapy for nurture; Oruč versus Seligman), then that it isn't (symptoms are treated either way; the cause may differ between people), then judge. Stay on nature and nurture, and on explanations (June 2022 report).

13. Measure depression: BDI scoring or another measure (3 questions)

- **Interpreting the BDI** (2): add the 0–3 scores of all 21 items (1); the total out of 63 falls in a band, e.g. 21–30 moderate (1). Learn the numbers exactly (March 2026 report).
- **Another measure** (2): a clinical **interview**, an **observation** with behavioural categories (how often the person cries, hours spent in bed), or a rating-scale questionnaire, with an example.
- **Measuring REBT by questionnaire**: closed rating-scale questions on how often the client disputes irrational beliefs, or open questions about how they now cope with activating events; the BDI before and after counts if you describe it.

14. Compare two treatments (similarity and difference) (2 questions)

Explain each with examples from both treatments (June 2022 report). **Similarities**: both reduce symptoms in studies with control groups; both need a professional; both take weeks to work. **Differences**: drugs have side effects, REBT has none; drugs change chemistry, REBT changes beliefs and gives skills that last after therapy stops. Don't claim REBT removes the cause or that SSRIs act at once, and compare with REBT itself, not CBT.

Worked examples

Example 1

Daniel is 19. For the past three weeks he has felt low nearly all day, has lost interest in playing football, sleeps 12 hours a night yet feels exhausted, cannot concentrate on his course, and tells his mother he is worthless. He has never had a period of unusually high mood. It started after he failed his driving test, when he said: "I must pass first time. Anyone who fails is a complete loser."

(a) Explain whether Daniel's symptoms could meet the ICD-11 criteria for a depressive episode. [3]

(b) Suggest how rational emotive behaviour therapy (REBT) could help Daniel. [4]

(a) ICD-11 needs at least 5 of 10 symptoms, including depressed mood or loss of interest, most days for at least 2 weeks **(1)**. Daniel has six: low mood and loss of interest in football (both affective), oversleeping, fatigue, poor concentration and worthlessness **(1)**. They have lasted three weeks, and he has never had a high, so this fits a depressive episode in depressive disorder (unipolar), not bipolar disorder **(1)**.

(b) The therapist would use the ABC model: the activating event (A) is failing the test, and the consequence (C) is his low mood and loss of interest **(1)**. The cause is his belief (B), "I must pass first time", a "must" belief that leads to self-downing ("I'm a complete loser") **(1)**. The therapist would dispute it (D): "Where is the evidence that everyone who fails is a loser? Many good drivers fail once." **(1)** The effect (E) is a rational belief, "I'm disappointed, but I can practise and retake it", which should lift his mood and get him back to football **(1)**.

Example 2

From the key study by Oruč et al. (1997) on mood (affective) disorders:

- (a) Calculate the percentage of the patient group who were female, to 1 decimal place. [1]
- (b) Suggest one reason why the researchers analysed male and female participants separately. [2]
- (c) Explain one weakness of the sample used in this study. [2]

(a) $\frac{25}{42} \times 100 = 59.5\%$ (1).

(b) Serotonin works a little differently in men and women (1), so an effect of the serotonin genes in one sex could be hidden when everyone is pooled; analysed separately, trends appeared for both genes in women (1).

(c) The sample was small and from one country (1): 42 patients with bipolar type I from two hospitals in Zagreb, so the results may not generalise to other countries or to unipolar depression (1).

Example 3

(a) Describe what psychologists have discovered about SSRIs and Beck's cognitive restructuring as treatments for mood (affective) disorders. [6]

(b) Evaluate what psychologists have discovered about SSRIs and Beck's cognitive restructuring as treatments for mood (affective) disorders, including a discussion of application to everyday life. [10]

(a) SSRIs. Selective serotonin reuptake inhibitors, such as fluoxetine (Prozac), are antidepressants used since the late 1980s. Normally, after serotonin crosses the synapse, spare serotonin is taken back into the presynaptic neuron (reuptake). SSRIs block this for serotonin only, so more stays in the synapse, which fits the biochemical explanation that depression comes from too little serotonin. They are taken daily and take several weeks to lift mood. Side effects include nausea, headaches and sleep problems, though fewer than with tricyclics or MAOIs.

Cognitive restructuring. Beck's talking therapy targets the negative cognitive triad. Over about 6–12 sessions the therapist and client identify negative automatic thoughts and the situations that trigger them, using a diary as homework. The therapist then challenges each thought with evidence (reality testing), for example "I'm useless at everything" after one poor mark, and helps the client replace it with a balanced thought, before tackling deeper negative schemas. Dobson's (1989) meta-analysis found it did better on average than drug treatment.

Why it reaches the top level: both treatments are described accurately with named examples, and each is linked to how it reduces symptoms.

(b) Application to everyday life (the named issue). SSRIs are cheap and need only a prescription and a daily pill, so they reach far more people than therapy, and a severely depressed person who cannot face talking can still take them. But the side effects and weeks of waiting make some people stop. Cognitive restructuring has

no physical side effects and teaches skills a person can use for life, but it needs a trained therapist, costs more, and depends on motivation and homework, which severe depression undermines. In practice the two are often combined: the drug lifts mood enough for the client to engage in therapy.

Reductionism versus holism. SSRIs reduce depression to one neurotransmitter and ignore the person's thoughts and circumstances. Cognitive restructuring is more holistic, working with how the person sees themselves, the world and the future, though it still ignores biology. Its lasting effect after sessions end suggests that changing thinking reaches more of the problem than changing chemistry alone.

Individual and situational. Both treat depression as a problem inside the individual, yet a depression triggered by a situation such as redundancy may return if that situation is unchanged.

Why it reaches the top level: issue by issue, the named issue first and in detail, both treatments compared within each issue, with evidence and context from (a).

Common mistakes

- **Saying bipolar episodes last minutes or hours**, or that mood flips several times a day. Each episode lasts days to months (June 2021 and November 2021 reports).
- **Giving symptoms of other disorders**, such as delusions and hallucinations, for bipolar disorder, or calling it "moodiness" (June 2021 and June 2022 reports).
- **Describing REBT as just A, B and C:** the treatment is the disputing (D) and its effect (E) (March 2023 and March 2025 reports).
- **Describing the cognitive triad when a treatment is asked for:** it is Beck's explanation; the treatment is challenging and replacing the thoughts (November 2021 and March 2023 reports).
- **Calling ECT or drugs psychological treatments** (November 2021 report).
- **Comparing REBT with CBT**, or saying REBT removes the cause or SSRIs work immediately (June 2022 report).
- **Confusing the key studies:** Seligman et al. (1988) with the dog experiments (June 2023 report); Oruč with Gottesman and Shields, or saying Oruč studied depression rather than bipolar disorder (November 2022 report).
- **Getting the BDI's scoring wrong**, or mixing it up with other measures (March 2026 report).
- **Unexplained evaluation:** "it is costly", "it takes time" or "it is reductionist" without saying why, or "side effects" without saying of what (June 2021 and March 2023 reports).
- **In a plan, using the wrong method or vague questions**, not saying which drug or for how long, or not linking (b)(i) to the plan (June 2022 and March 2025 reports).

How to get the marks

- **"Identify", "give", "state":** short exact facts (numbers in a sample, a named symptom).
- **"Outline"** (2 marks): the point plus a detail or example.
- **"Explain" or "suggest"** (2 marks a point): the point (1) applied to depression, the study or the patient (1).

- **Patient questions:** use the person's name, words and symptoms in every sentence.
- **"Describe" in an essay** (6 marks): both named items, accurate terms (monoamine oxidase, reuptake, ABCDE, triad, internal-stable-global), each linked to symptoms.
- **"Evaluate"** (10 marks): issue by issue, the named issue first, both items compared, context from (a), evidence, a short judgement.
- **"Discuss ... with a conclusion":** at least two strengths and two weaknesses, then a reasoned judgement.
- **Plans:** the named method, specific features with example questions, real patients, ethics; then the knowledge, linked feature by feature.

Quick check

1. A patient's BDI answers are: 9 items scored 1, 5 items scored 2, 2 items scored 3 and the rest 0. What is the total, which band is it in, and what is the highest total possible?
2. Which diagnosis fits each person? (a) Ten days of euphoria, little sleep and reckless spending, and no depressive episode yet. (b) Several depressive episodes and two spells of four days with raised mood and energy that did not harm their work. (c) A month of low mood and five other symptoms, with no history of high mood.
3. On the ASQ, a man rates the cause of a bad event 6 for internal, 6 for stable and 7 for global, on 7-point scales. What is his mean rating, to 1 decimal place, and what does it suggest?
4. A client says: "If I don't get this job, it will be the worst thing that could ever happen." Name the irrational belief and write one disputing question.
5. A student writes: "One weakness of MAOIs is side effects." Why does this earn only 1 mark, and how could it earn 2?

Answers

1. $9 \times 1 + 5 \times 2 + 2 \times 3 = 25$, which is in the 21–30 band: **moderate depression**. The highest possible is $21 \times 3 = 63$.
2. (a) **Bipolar type I**: one manic episode (at least a week) is enough. (b) **Bipolar type II**: hypomanic episodes (several days, no serious harm) plus depressive episodes. (c) **Depressive disorder (unipolar)**: at least 5 symptoms for more than 2 weeks, never a high.
3. $(6 + 6 + 7) \div 3 \approx 6.3$. That is near the top of the scale: a **pessimistic** style (internal, stable, global) for bad events, which Seligman et al. linked to more severe depression.
4. **Awfulising**. Disputing: "Would it really be the worst thing that could ever happen? What else could you do if you don't get it?"
5. It identifies a weakness but doesn't explain it. Better: "MAOIs react with tyramine in foods such as aged cheese (1), which can cause a dangerous rise in blood pressure, so patients must follow a strict diet (1)."

Past questions to try

- [9990/32/O/N/25 Q1](#): telling unipolar from bipolar disorder in a patient's behaviour.
- [9990/33/M/J/24 Q3](#): cognitive restructuring for both poles of bipolar disorder.
- [9990/42/O/N/24 Q2](#): interpreting the BDI, another measure, and its weaknesses.
- [9990/32/O/N/23 Q1](#): mania, Seligman et al. (1988) and its controls.