Clinical psychology

What is Clinical psychology?

- This field is concerned with:

understanding, preventing, and relieving psychologically-based distress or dysfunction and to promote subjective well-being and personal development

- While in the past it was only concerned with mental illness, more like a psychiatry
-
- Central to its practice are psychological assessment, and psychotherapy, although clinical psychologists also engage in research, teaching, consultation, forensic testimony etc.

Mental illness – entity or spectrum?

- We experience symptoms of different mental illnesses on a daily basis:

- dissociation with daydreaming
- frantic mood when passing an important exam
- having your working desk perfectly organised

Psychosis

Manic episode - bipolar episode

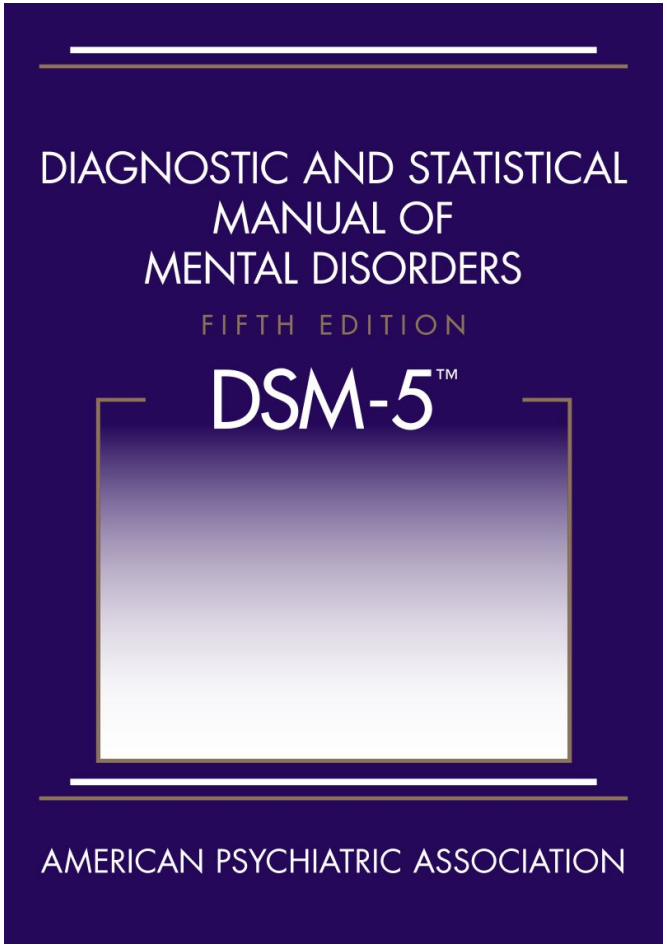
OCD

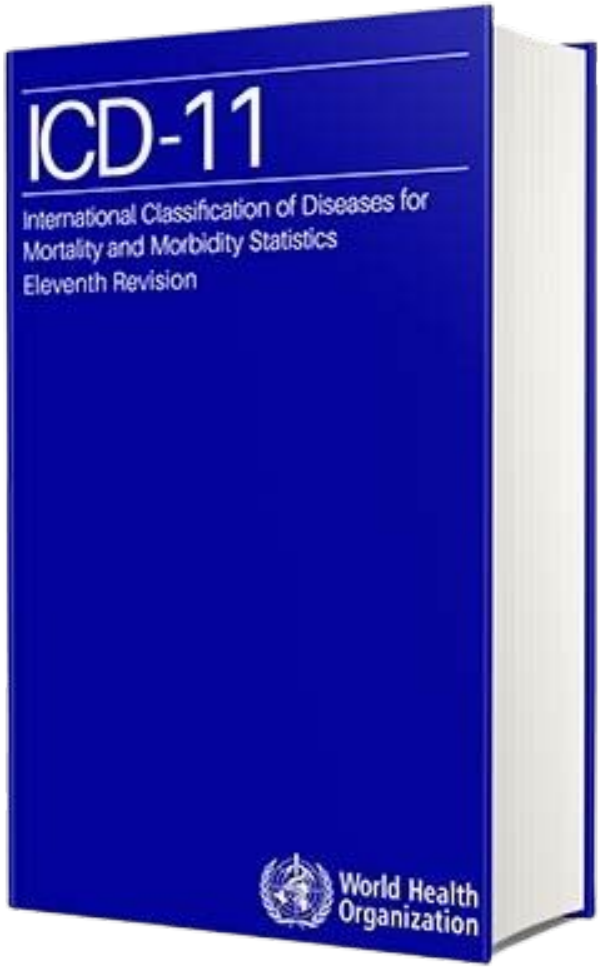
Why these aren't enough for establishing a diagnosis?

—> It's never about one symptom but

Intensity, frequency, adequacy and a specific constellation of symptoms

How mental disorders are diagnosed?





How mental disorders are diagnosed?

Differential diagnosis

Different health conditions often cause similar symptoms. For example, fatigue is a symptom that can be caused by anemia, depression, heart disease, thyroid disease, sleep disorders, and many other conditions. If you have fatigue or other symptoms that are linked to many conditions, your doctor has to figure out which condition you have so that you get the right treatment.

differential diagnosis - when there is more than one possibility for diagnosis, a doctor must differentiate between multiple diagnoses to determine the correct one and make an appropriate treatment plan

How mental disorders are diagnosed?

Comorbidity

The simultaneous presence of two or more diseases or medical conditions in a patient.

- Important implications for treatment

From Clinical psychology option you will learn:

- Diagnostic criteria
- Explanations
- Treatment for

Separately from **biological and psychologica**l approach.

And at the end, you'll learn how to evaluate them based on research evidence, methodology, and considering important issues and debates.

five different psychological disorders:

- Schizophrenia
- Mood disorders
- Impulse control disorders
- Anxiety disorders
- and Obsessive-compulsive disorder

Clinical Option:
Mood (affective) disorders

Affect = synonym for emotion
Affective disorders - affect emotions

People with mood disorders often experience episodes where either very negative, or very positive emotions, dominate their thoughts and behaviours in a way that can lead to dysfunction and distress.

Episodes can be depressive, manic, mixed and hypomanic.

We all experience changes in mood each day 🙂🙃☹️. What differentiates the disorder from daily ups and downs is **duration** of **intensity** of the experienced mood.

Types of mood disorders

- Unipolar (depression)

  https://www.youtube.com/watch?v=4YhpWZCdiZc

- Bipolar (manic-depressive disorder)

  https://www.youtube.com/watch?v=uwj5ZC3u73w

- Other mood disorders - dysthymia and cyclothymia

Diagnostic criteria: Depression (unipolar disorder)

For diagnosis to be made there must be present (core symptoms):

depressive mood or
loss of pleasure

Accompanied by other symptoms (min 5 in total) that affect individual's ability to function, such as:

- Reduced ability to concentrate, pay attention to tasks, indecisiveness.
- Beliefs of low self-worth or excessive and inappropriate guilt
- Hopelessness about the future.
- Recurrent thoughts of death
- Changes in eating or sleeping patterns
- Reduced energy, fatigue, or marked tiredness

These symptoms make up what we call a **depressive episode.** In order to be called an "episode" those symptoms must occur most of the day, nearly every day during a **period of at least 2 weeks.**

Also, a diagnosis of depressive disorder can only be made if the individual hasn't experienced a manic, mixed, or hypomanic episode, as this would indicate bipolar disorder instead.

Single episode depressive disorder is characterised by the presence of one depressive episode, without history of previous episodes.

Recurrent depressive disorder is diagnosed when there is at least two depressive episodes separated by several months (or more) without significant mood disturbances.

Bipolar disorder, type 1

At least one **manic** or **mixed episode**

Manic Episode

Both of the following features occurring concurrently and persisting for most of the day, nearly every day, during a **period of at least 1 week**.

- An **extreme mood** state characterized by euphoria, irritability, expansiveness, high self esteem that represents a significant change from the individual's typical mood.
- **Increased activity** or a subjective experience of increased energy that represents a significant change from the individual's typical level.

Mixed Episode

manic + depressive symptoms, which either occur simultaneously or alternate very rapidly (from day to day or within the same day).

Symptoms must be present most of the day, nearly every day, during a period of **at least 2 weeks.**

Bipolar disorder, type 2

At least one **hypomanic** and **depressive episode**

Hypomanic episode

→ is a less extreme version of a manic episode and involves **several days** of persistent elevated mood or increased irritability, along with increased activity or increased energy levels. Behaviours shown will be lesser versions of those in a manic episode, for example increased talkativeness, increased self- esteem and impulsivity. These behaviours will be significantly different to the individual's usual behaviour but will not cause marked impairment to functioning. There is no history of manic or mixed episodes.

Other mood disorders - dysthymia, cyclothymia

Measures: Beck Depression Inventory

Aside from using clinical interview, doctors can also use psychometric tests to gather data about symptoms. While the interview will provide qualitative data, psychometrics gather quantitative data, and are good example of nomothetic approach.

Beck's Depression Inventory (BDI):

- 21-item self-report measure that assesses symptoms of depressive disorder
- Each item in the inventory consists of at least four statements where person chooses the one that best fits how they have been feeling during a past **2 weeks**
- Total score determines the severity of the disorder (mild, moderate, severe)

→ with 10 being a cutt-of score (below that = normal ups and downs)

- Since its initial introduction, this influential tool has been updated twice

→Current version: same structure as the original; revised to include the symptoms of changed appetite and fatigue

Evaluating BDI

a way to judge validity by comparing measures of the same phenomenon in different ways at the same time to show that they produce similar results in the same circumstances

Strengths:

Concurrent validity

1.Evidence suggests this is both reliable and valid measure.
→ excellent test-retest reliability **(?)**
→ One study showed + correlation between BDI scores and another well-respected test of depression (Hamilton psychiatric rating scale for depression)

2.Quick, easy to administer, yet precise.

Weaknesses?

Evaluating BDI

Weaknesses

1. **General risks associated with self - reports**

subjectivity, social desirability

2. Individual - situational

Relaying on BDI emphasise individual approach to diagnosing, but in negative way
* not individualistic like idiographic, but failing to capture situational, contextual factors that could lead to depressive state
individual approach - factors within the person that caused her to develop disorder

Evaluating ICD - 11 diagnostic criteria

Strengths:

- ICD-11 shows high reliability in diagnosing mood disorders, with 84% agreement for bipolar disorder (type 1) and 74% for recurrent depressive disorder among clinicians.
- Superior to ICD-10

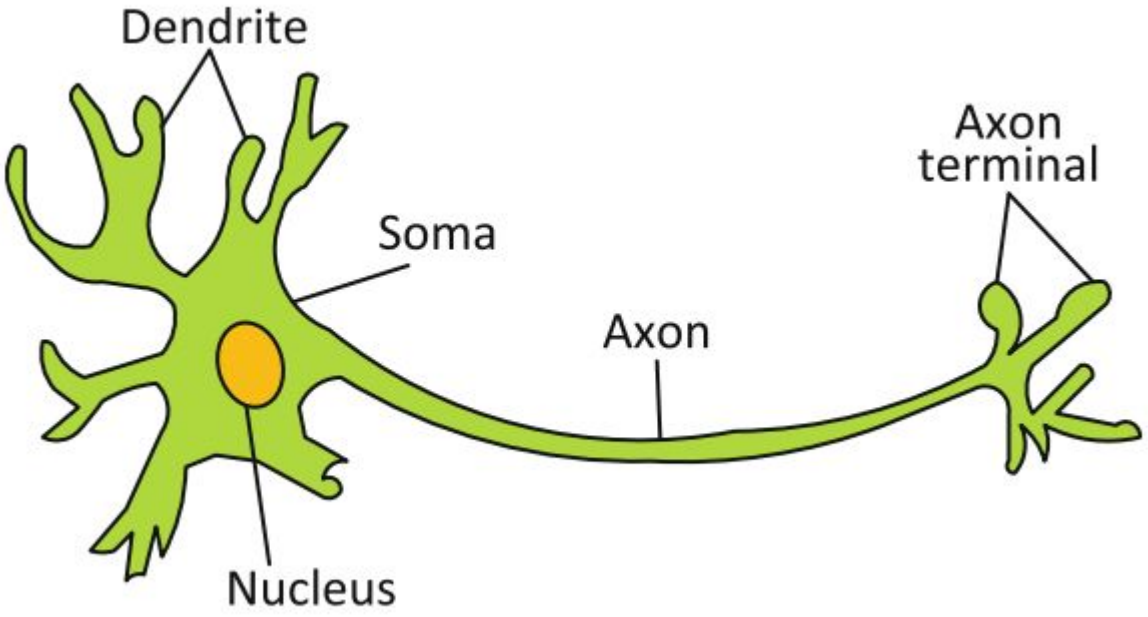
Weakness: Despite good reliability for above mentioned mood disorders, there is less favourable results for others. For e.g. the is lower agreement for bipolar (type 2) at 62% and dysthymic disorder at 45% (Reed et al., 2018).
→This suggest that some mood disorders are harder to diagnose, then others.

Cultural differences
ICD-11's recognition of cultural differences, especially somatic symptoms like aches and pains in low-to-middle-income countries (LMICs), empowers clinicians to achieve more rapid and sensitive diagnoses **(strength)**

Explanations of mood disorders

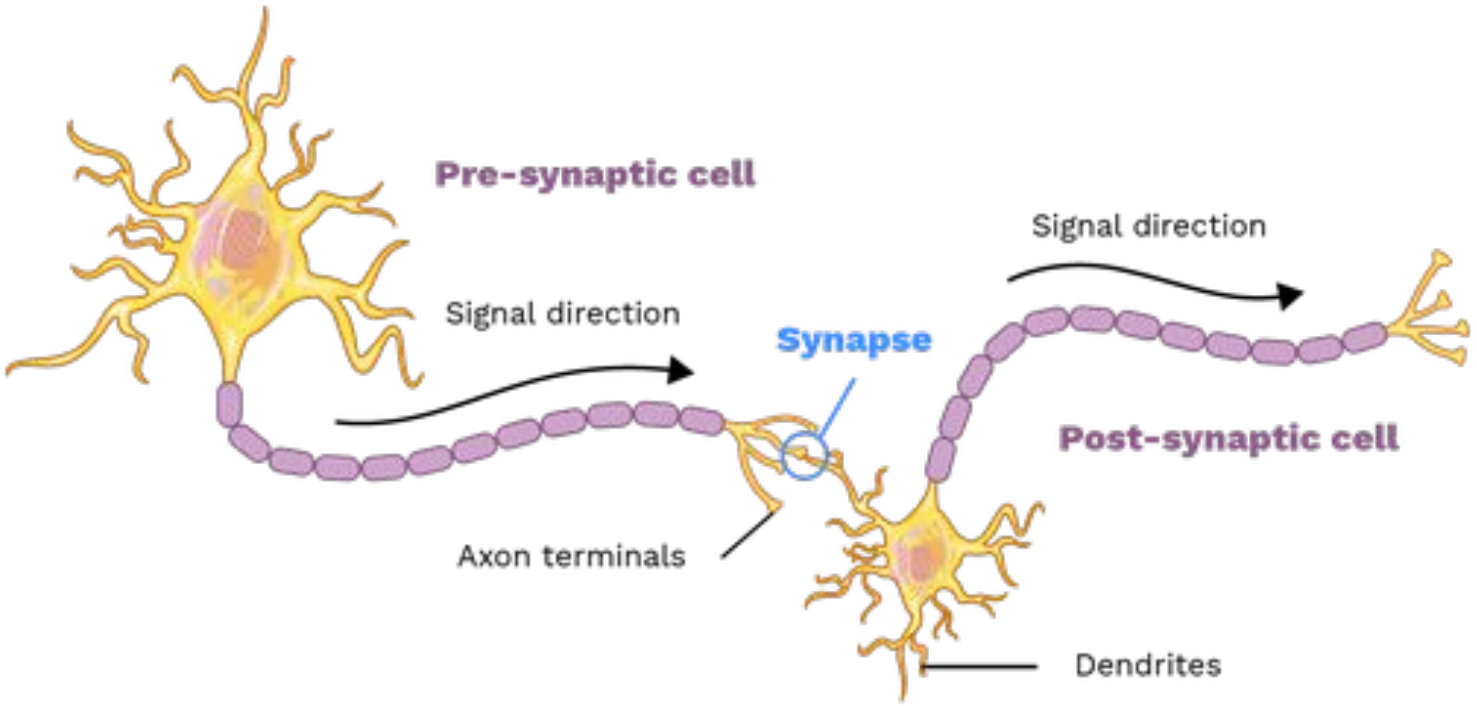
- Biological
- Psychological

Brain structure and chemistry that you need to know before we continue

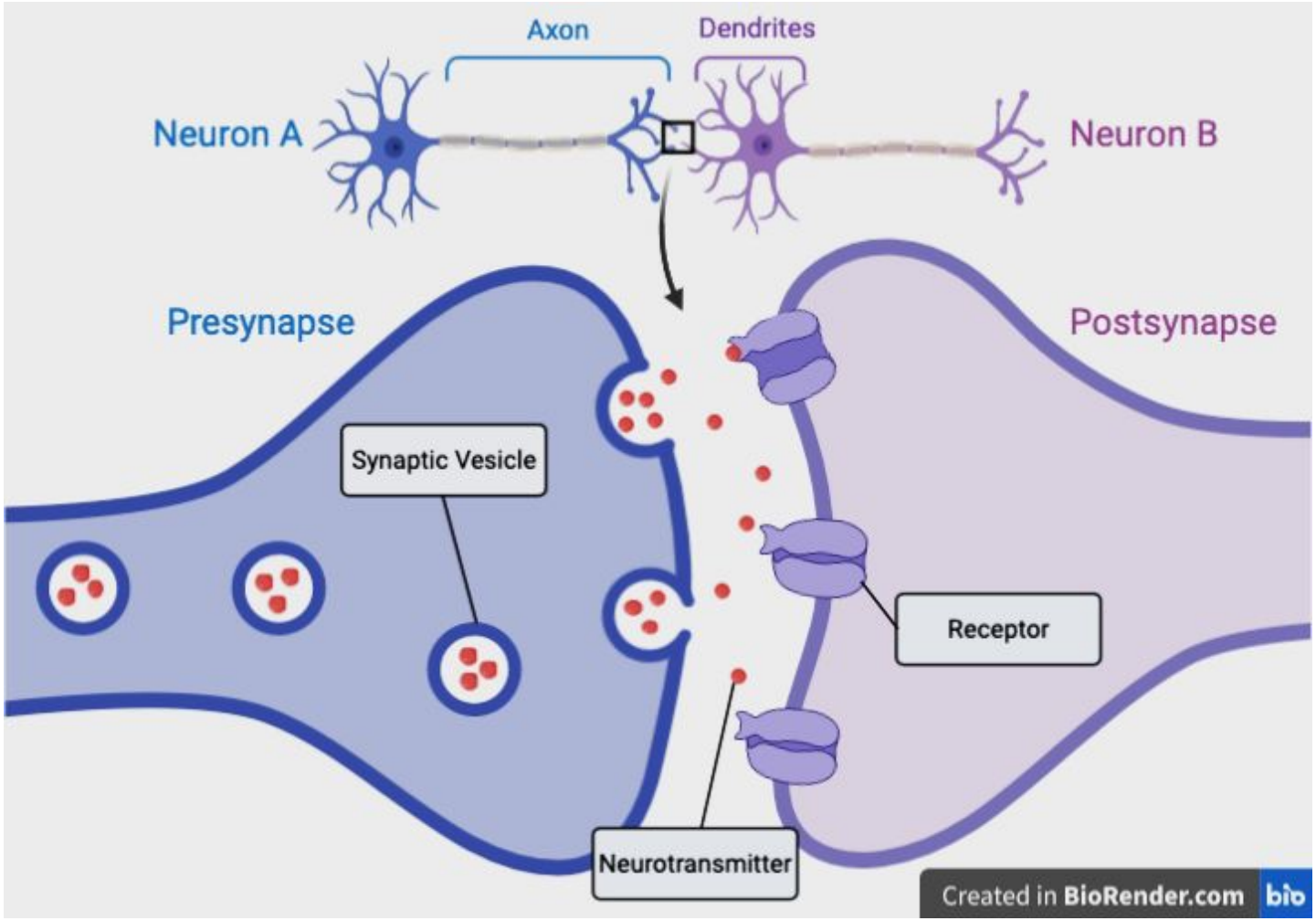


Brain cell (neuron)

Brain structure and chemistry that you need to know before we continue



Synapse - communication (signal transmission between 2 neurons)

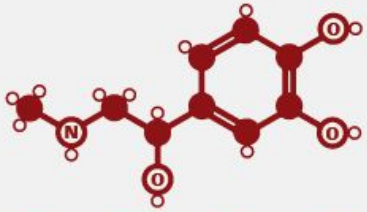


Synapse - closer look

THE STRUCTURES OF NEUROTRANSMITTERS

STRUCTURE KEY: Carbon atom · Hydrogen atom · O Oxygen atom · N Nitrogen atom · R Rest of molecule

ADRENALINE
Fight or flight neurotransmitter



Produced in stressful or exciting situations. Increases heart rate & blood flow, leading to a physical boost & heightened awareness.

NORADRENALINE
Concentration neurotransmitter

Affects attention & responding actions in the brain, & involved in fight or flight response. Contracts blood vessels, increasing blood flow.

DOPAMINE
Pleasure neurotransmitter

Feelings of pleasure, and also addiction, movement, and motivation. People repeat behaviours that lead to dopamine release.

SEROTONIN
Mood neurotransmitter

Contributes to well-being & happiness; helps sleep cycle & digestive system regulation. Affected by exercise & light exposure.

GABA
Calming neurotransmitter

Calms firing nerves in CNS. High levels improve focus; low levels cause anxiety. Also contributes to motor control & vision.

ACETYLCHOLINE
Learning neurotransmitter

Involved in thought, learning, & memory. Activates muscle action in the body. Also associated with attention and awakening.

GLUTAMATE
Memory neurotransmitter

Most common brain neurotransmitter. Involved in learning & memory, regulates development & creation of nerve contacts.

ENDORPHINS
Euphoria neurotransmitters

Released during exercise, excitement, & sex, producing well-being & euphoria, reducing pain. Biologically active section shown.

Neurotransmitters are chemicals that transmit signals between nerve cells (neurons) and play a crucial role in brain function and communication.
https://www.youtube.com/watch?v=FHNP4oPiAPE

This part is enough to learn from presentation

Biological (biochemical) explanations

There isn't just one neurotransmitter that can be attributed to depressive disorder; it is very complex, and several neurotransmitters have been identified as important

- Dopamine - impact on motivation and experiencing pleasure

→ **low levels of dopamine** observed in people suffering from depressive disorder

→ Furthermore, some **anti-depressants** reduce symptoms by **increasing dopamine levels**

- Serotonin - impact on mood, sleep, and appetite

→ **Reduced serotonin levels** are associated with low mood, anxiety, and disturbed sleeping and eating patterns

→ The most common **antidepressants (SSRIs)**, work by **increasing serotonin levels**

Biological (**biochemical**) explanations

Monoamine hypothesis

- Researchers initially focused on noradrenaline, a neurotransmitter central to brain emotion centers.
- Reserpine, a drug for high blood pressure, reduce noradrenaline levels.. but, as a common side effect, provokes depressive symptoms

→ Noradrenaline deficiency was suggested as a potential cause of depression

Shift to Serotonin:

- Attention turned to serotonin, another neurotransmitter, which is actually regulates noradrenaline →imbalance in serotonin could affect imbalance in noradrenaline levels
- Depression: Low noradrenaline levels associated with depressive episodes
- Mania: High noradrenaline levels linked to manic episodes.

Noradrenaline and serotonin are called 'monoamines', so together these ideas are referred to as the monoamine hypothesis.

Biological (**genetic**) explanations:

Biochemical explanations focus on neurotransmitters, while genetic focus on genes

Genes provide the instructions that dictate how these neural networks develop (how efficient is the neurotransmission)

Genes can have different versions (alleles)

Polymorphisms - presence of 2 or more variants of specific gene (g**enes being polymorphic - means there is different versions of them**)

Biological (**genetic**) explanations:

Some terms that we need to know:

heritable - transmissible from parent to offspring

Heritability - how much of the differences we see in traits among people are due to their genes, rather than the environment they're in

extent to which genetic factors contribute to the risk of developing certain condition IN POPULATION (not for 1 specific person)

Concordance: the probability that a pair of individuals (siblings, mother-child, MZ twins..) will both have a certain characteristic

Concordance rate - % of pairs of individuals that both have a certain trait or condition

*In an enormous twin study (n=>42,000), Kendler et al. (2006) found a concordance rate of **44%** for female monozygotic (MZ) twins compared with only 16% for female dizygotic (DZ) twins, and rates of 31% for male MZ twins and 11% for male DZ twins.*

What is the meaning of this?

This suggests 2 things:

- that depression has a genetic component, but also
- that it's more heritable in females

But bipolar disorder is even more heritable than depression (62% for MZ, 8% for DZ)

Biological (**genetic**) explanations: Core study by Oruč et al. (1997)

Learn from here instead of *Context part* in the book

Context:

Bipolar disorder etiology is complex. To be understood better both biological (genes and brain chemistry) and psychological factors must be considered. Traumatic experiences and various environmental triggers, such as physical illnesses or disruptions in sleep patterns, can contribute to the onset of this disorder.This study aims to investigate potential genetic predispositions linked to this disorder, even though identifying specific genes is challenging due to complicated inheritance patterns.

Main Theories and Explanations:

- Bipolar disorder is highly heritable, with genetic factors accounting for up to 80% of its causation.
- Specific associated genes remain poorly understood.
- Investigating neurotransmitters linked to the disorder, such as serotonin, dopamine, and noradrenaline, provides a pathway to explore related genetic factors → for instance, lower serotonin origin leads to the investigation of genes involved in serotonin regulation

Aim:

This study aimed to determine whether specific polymorphisms of two genes associated with **serotonin transmission** (**5-HT2c and the 5-HTT gene**) were more common in people with **bipolar disorder.**

Sample:

42 patients with bipolar disorder type 1, all from Croatian hospitals. There was 25F and 17M, aged 31-70.

➢ 16 of these patients reported to have a **first-degree relative** who had been diagnosed with affective disorder. This information was confirmed through medical records.

Control group: 40 participants, with no personal or family psychiatric history

→ **A matched pairs design was used:** where participants in the control group matched the patient sample in terms of age and sex.

Procedure

Blood samples were analysed to see which alleles participants were carrying for **two specific genes:**

- **the serotonin receptor** 2c **gene** (5-HTR2c), which codes for a specific type of serotonin receptor
- and the **serotonin transporter gene** (5-HTT), which codes for the serotonin transporters.

Each gene had two possible alleles (variants).

- 5-HTR2c has variants named Cys (C) and Ser (S)
- 5-HTT has variants named 1 and 2

Results

- The researchers found that there was no significant difference between control and bipolar group in those gene variants.

However, they did find some **sex differences in allele frequency:**

- S allele of the 5-HTR2c gene was more common in females with bipolar disorder than the female controls
- allele 1 of 5-HTT gene was more common in females with than without bipolar

Meaning that serotonin is sexually dimorphic → serotonin works slightly differently in males and females, they can have different natural baseline and sensitivity to it

Conclusions

- ➔ A genetic basis for bipolar disorder is not supported. However, there are limitations to the procedure used, such as the 'power' of the study and low sample size.

- ➔ The findings suggest that females might be more vulnerable to genetic alterations affecting serotonin and more susceptible to bipolar disorder than males.

Evaluation of Oruč et al. (1997) Study

Weaknesses

→ Sample

a) small number of participants(especially for genetic studies)

b) some participants in the control group were still young enough that they could be susceptible to bipolar disorder but it has not developed yet

Strengths:

1.Good equipment

DNA samples were collected and analyzed in a controlled laboratory setting,

→This enhances **validity** and minimizes **researcher bias**.

2. All participants were **carefully assessed** to check that they reached the criteria for a diagnosis of bipolar disorder (type 1). Two experienced psychiatrists used one commonly used structured diagnostic interview, which has good methodological features.

Evaluation of Oruč et al. (1997) Study

Issues and debates?

- Application to Everyday Life - may help make revolutionary drug treatments➡️ in the future we could have personalise medication matching the person's genom

For all 3 - biochemical, genetic and Oruč:

- **Determinism** vs. Free Will Debate
- **Nature** - nurture
- **Reductionism** vs Holism

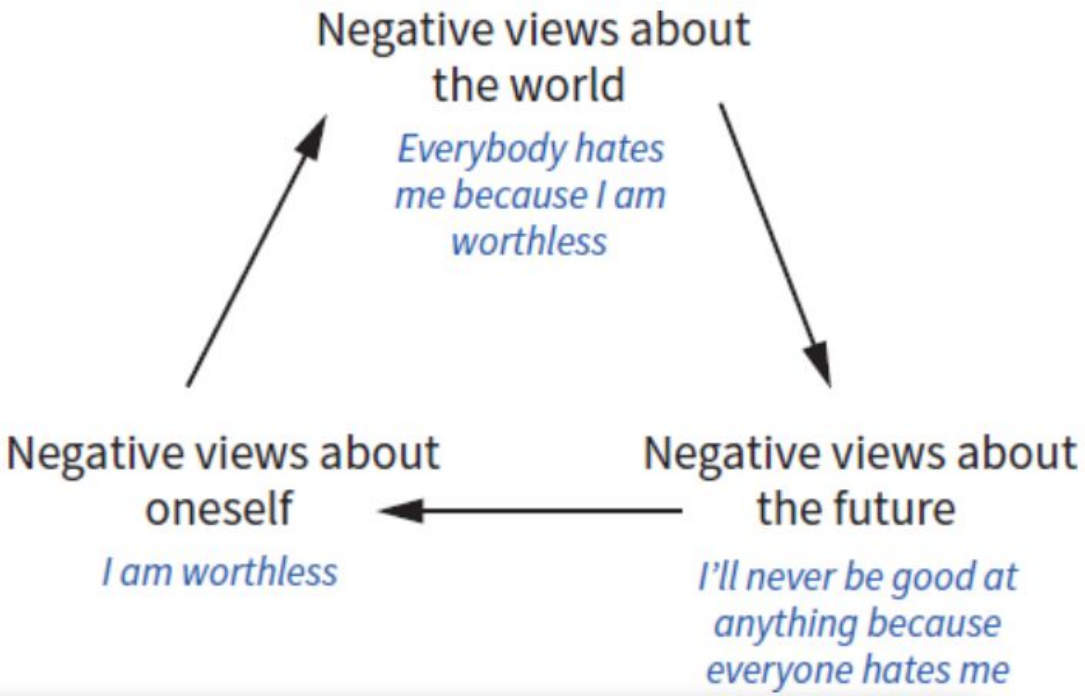
➡️ biological determinism

Psychological (cognitive) explanation

Beck (1979): depressive symptoms are caused by **negative thoughts,** linked to **dysfunctional (unhelpful) core beliefs**

depressed people hold negative beliefs about the **self, the world and the future**

Negative cognitive triad



Cognitive explanation

→ these beliefs develop in childhood as a result of negative experiences

→ process that maintain these negative beliefs is incorrect information processing (cognitive distortion)

Cognitive distortion = filtering information around us - evidence that contradicts our beliefs is ignored, they only focus and remember things that confirms their (negative) views

At least be positive

Faulty thinking strategies

Do you do any of these? Rate each one from 1 to 10 (1: 'I never do this', to 10: 'I do this every day').

- **Dichotomous thinking** ('all or nothing'): classifying events into two categories with no middle ground, such as success and failure. Sometimes called 'black or white' thinking.
- **Arbitrary inferences:** drawing negative conclusions in the absence of sufficient supporting evidence. This can lead people to make assumptions about what other people think about them ('mind reading') and how things will turn out in the future ('fortune telling'). Also linked to over-generalising – for example, not being offered a job leads the person to think they are unemployable.
- **Catastrophising:** something relatively insignificant triggers a flood of negative thoughts, resulting in overwhelming anxiety about worst-case scenarios. For example, you find a freckle you have not noticed before and end up terrified you have skin cancer, sick with worry about death and leaving behind family members with no-one to look after them.
- **Personalisation:** taking the blame for negative outcomes and failing to acknowledge situational factors, such as 'I was made redundant because I am useless', not due to the company's financial difficulties.

Learned helplessness and attributional styles

Skip the Animal research part in the book

Learned helplessness - negative state that occurs after an individual had to endure an unpleasant situation(s), where the negative outcome was inescapable; there was a real or perceived lack of control over the situation

→ result: individual generalise the helplessness from that situation to the future events; believing that they are unable to control and prevent suffering, eventually they stop trying to resist it - possible cause of depression

But why some people don't become helpless (and depressed) even though they have been exposed to many stressful situations in which they had no control?

Answer: because of different **attributional styles**

Attributional style (explanatory style) -individual's way of explaining reasons for events or outcomes. It is the way people explain the causes of their own behavior, as well as the behavior of others.

People who interpret **negative situations** as **internal, global and stable** are more vulnerable to develop depression, hereas people who are unlikely to become depressed make attributions that are **external, specific and unstable**

▼ Table 6.6 Following the breakdown of a relationship, Person A is at greater risk of developing depression due to their pessimistic

Person A: Pessimistic	Person B: Optimistic
Internal: 'I wasn't good enough for them.'	External: 'We weren't spending enough time together.'
'My friends think it's my fault, I'm rubbish at my job, I'm going to fail my exams.'	'At least I've got my friends, my job and I'm doing okay in school.'
Stable: 'I'm going to be single forever.'	Unstable: 'I'll meet someone new when I'm ready.'

This is not for exam, but for you to think about it :)

	POSITIVE SITUATION	NEGATIVE SITUATION
Optimist	**causes** → Internal, Stable, Generalized	**causes** → External, Temporary, Specific
Pesimist	**causes** → External, Temporary, Specific	**causes** → Internal, Stable, Generalized

Example study

Seligman et al. (1988) - how well attributional style predicts depressive symptoms

Sample: 39 with unipolar and 12 with bipolar - during a depressive episode

- Compared to control group (nonclinical population)

Measures used: Beck's Depression Inventory and Attributional Style Questionnaire (12 hypothetical good and bad events)

Task - to make attributions for each event (what or who they think was responsible)

- Then they completed six months of weekly cognitive therapy
- Some of them were reassessed after a month of their last therapist session, and some of them 12 months later (to compare the short versus long term effect of therapy)

Results: Both depressed and bipolar patients had more pessimistic, negative attributional style

The higher score on BDI, the more pessimistic on ASQ

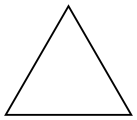
Improvement on ASQ during the treatment, correlated with lower BDI scores

Conclusion: People with both unipolar and bipolar disorder share a cognitive attributional style which favours internal, global and stable attributions of negative events in comparison with people without depression, but this tendency can be improved with therapy

Evaluating the Seligman's study

✔using more than 1 technique to assess the symptoms (BDI & clinical interview)

We call this **triangulation**? Hint:



- BDI and ASQ are good, reliable instruments. But it was only a **correlation** calculated between their scores - meaning…**?**
- Participants dropouts

Evaluating the psychological explanation

Research support - Seligman's study

Determinism - free will - both
D:according to cognitive psychologists, these negative views are thought to result from early life experiences

FW:on the other hand these negative views can be changed by the power of free will (and learning new, healthy attributional styles)

*even Seligman over time switched from studying negativity to positivity (his famous book is called "Learned optimism")

Treatments

- Biological
- Psychological

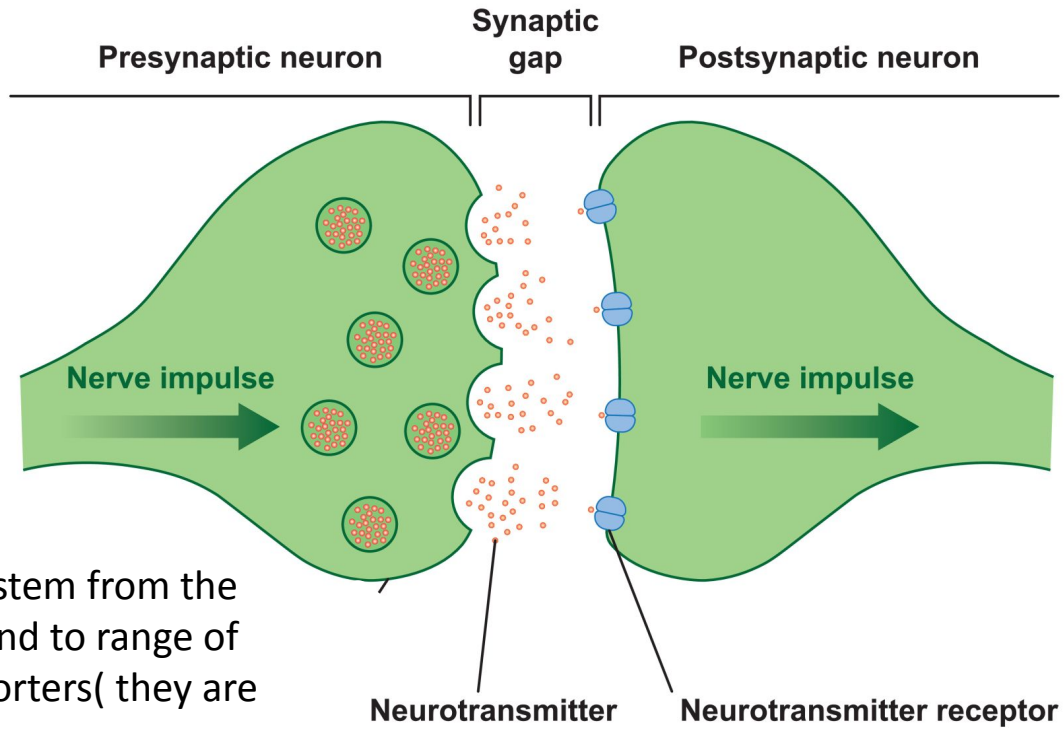
Biochemical treatments - Antidepressants

1.Tricyclics

Increase levels of serotonin and noradrenaline by stopping the process of reabsorption

How they stop reabsorption ? → by blocking serotonin and noradrenaline transporters

And, what are the transporters 🙄 ?!



Side effects of tricyclics stem from the fact that tricyclics also bind to range of different types of transporters(they are not selective)

Biochemical treatments - Antidepressants

2.MAOIs (monoamine oxidase inhibitors) inhibit enzyme known as monoamine oxidase

monoamine oxidase has the job of breaking down neurotransmitters such as serotonin, noradrenaline, and dopamine, once it's reabsorbed. One way to increase those neurotransmitters and their availability for releasing into synapse is to block what monoamine oxidase is doing

MAO-A - breaking down serotonin and noradrenaline
MAO-B - breaking down dopamine

Bigger antidepressant effect has inhibition of MAO-A

➢ But, this medication can have harmful interactions with other drugs, such as painkillers, or foods

What tricyclics and MAOIs have in common:

- Evidence for effectiveness- from the 1950s (old, first generation of AD)
- but usually used as a last option, because of side effects (tricyclics: drowsiness, nausea, vomiting, blurred vision, weight gain; MAOIs: headaches, drowsiness, insomnia, nausea, diarrhea, constipation, and even increasing a risk of stroke)

3.SSRIs - selective serotonin reuptake inhibitors

→ they are known as specific serotonin reuptake inhibitors as they only inhibit serotonin transporters…. there are some other similar drugs that can inhibit only noradrenaline transporters for e.g. (SNRIs)

→ e.g. Prozac

→ most commonly used nowadays since they have fewer and less severe side effects, although different individuals may respond better to particular drugs

What do all 3 medication have in common :
Increasing levels of serotonin available (to go to postsynaptic cell)

Evaluation of biochemical treatment

Strength: Research evidence supports the effectiveness of antidepressants for many individuals : many RCTs, and even better - many double blind RCTs showed that antidepressants are more effective than placebos

Weakness: 1. Questionable significance of improvement → Is improvement defined as a higher score on scales really significant in a real life ?

2. of course - side effects

Randomised control trials (RCTs) - 'gold standard' in clinical and health research, such as drug testing. Participants are randomly assigned to either an experimental group (who are given the drug or a treatment) or a control group (who are given nothing or placebo)

Issues and debates

Individual - situational - another weakness because applying only medication undermines the role if situational factors; should support people to remove themselves from toxic, unhealthy situations, relationships…

*for Applications to everyday life you can write about side effects

The overall conclusion is that antidepressants may be helpful in targeting some symptoms more than others →again combination of meds + psychotherapy would be the best option for more severe cases (holistic treatment approach)

Additional discussion points:

No need to learn this, but you can if you want

1.Ethical Issues in Antidepressant Use

- Sponsorship and publication bias in the $16 billion antidepressant industry
- Turner et al. (2008) found 8% publication rate for negative results vs. 97% for positive results
- False hope given to patients due to biased publication ?

2.Placebo and nocebo effects in antidepressant use

Do you know what is a nocebo effect?

- Professor Kirsch's view on antidepressant efficacy primarily as placebo effects
- Advocacy for safer alternatives like exercise and psychotherapy

Psychological treatment:

1)Cognitive restructuring (Aaron Beck)- **the process of identifying and challenging negative and irrational beliefs**

- commonly used technique in CBT an other psychotherapeutic approaches
- talking technique, 1 to 1 interaction
- Includes:

❏ Psychoeducation: patients are trained to recognize their own thoughts and understand how they influence emotions and behaviors

❏ Giving homework - usually keeping a thought/mood diary

❏ Socratic questioning - The therapist asks questions to help the client analyze and reflect on their thoughts. together they will seek alternative explanations for negative attributions

Goals:

Reality testing, reattributing, and the end goal - replace irrational beliefs, NOT with fake positivity, but with more realistic and constructive views

Evaluation:

Strengths: Cognitive therapy is now a well-established approach, lot of research is done on it

→ e.g. Wiles et al (2013) showed that it can reduce symptoms of depression in people who fail to respond to antidepressants.

individuals with depression were randomly allocated to:

a)continued usual care (antidepressants)

b) CBT (where cognitive restructuring was used)

Those who received therapy were three times more likely to respond to treatment and experience reduction of symptoms

Weaknesses: success will depend on many individual factors: cognitive flexibility (lowers with age), motivation, communication skills.. *this can be written as a Applications to everyday life as well

2. REBT - Rational emotive behavioral therapy

Albert Ellis

ABC MODEL:

A = activating event

B = beliefs

C = consequences

Three MUSTs:

1.I must do well

2. Others must treat me well

3. I must get what I want, and not what I don't want

Disputing:

Questioning and challenging 3 musts.

ABC Model


A ctivating Event
Something happens to you or in the environment around you.
B eliefs
You have a belief or interpretation regarding the activating event.
C onsequences
Your belief has consequences that include feelings and behaviors.
D isputations of beliefs
Challenge your beliefs to create new consequences.
E ffective new beliefs
Adoption and implementation of new adaptive beliefs.

Evaluating REBT

Strengths: 1.REBT can be as effective as antidepressants for young people with depression.

→ Iftene et al. (2015) study: REBT therapy resulted in increased serotonin, reduced negative thoughts and depressive symptoms (sample:teens); looks like good an alternative to drugs with potential side effects

2.**Free will**- Determinism

Weakness: 1**.** Some supporting studies may have flaws:participant dropouts are common

2. Ethic concerns - is this therapy suggesting people should accept unjust world? How will victims of crimes feel if we teach them to reject the belief “people must treat me well” ?!