Explanations of depressive disorder

Lesson 3.2.2 | Clinical psychology

Core ideas

Genetic risk can make depressive disorder more likely, but it does not mean a person will definitely develop depression.

Cognitive explanations link depression with negative beliefs, biased interpretation and patterns such as the negative triad.

A strong explanation uses evidence carefully and recognises that different people may have different pathways into depression.

Biochemical account: neurotransmitter processes, including serotonin and noradrenaline, may contribute to depressive symptoms. Evaluate reductionism: variation in mood is not explained adequately by simply labelling everyone as having a chemical imbalance.

Beck's cognitive theory includes negative views of the self, world and future, dysfunctional beliefs and biased information processing. Learned helplessness proposes that perceived lack of control discourages attempts to change outcomes; a pessimistic attributional style explains bad events as internal, stable and global.

Illustrative evidence - Seligman et al. (1988) investigated explanatory-style change during cognitive therapy for unipolar depression. Connect changing interpretations to symptoms and consider whether an association between changes establishes the direction of causation.

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Key terms

Biological explanation: An explanation based on genes, brain chemistry, hormones or body processes.

Genetic vulnerability: Inherited risk that can increase the chance of developing depression.

Neurotransmitter: A chemical messenger involved in communication between neurons.

Cognitive bias: A pattern of thinking that interprets information in a distorted or negative way.

Negative triad: Negative views of the self, the world and the future.

Attributional style: A habitual way of explaining why events occur.

Genetic association: A statistical relationship between a genetic variant and a characteristic; it does not itself establish causation.

Further detail and evidence

Key study - Oruc et al. (1997): the researchers tested associations between bipolar I and variants of the serotonin receptor 5-HT2C and serotonin transporter genes. DNA from 42 bipolar I patients and 40 healthy controls was compared; this was an association design, not random assignment to genetic conditions. Patients came from two Zagreb hospitals; diagnosis used a lifetime psychiatric interview (SADS-L) reviewed by a second psychiatrist, and the gene analysis used blood-derived DNA.

Oruc results: there was no significant association in the full patient sample. Exploratory sex-specific analyses suggested only weak possible associations in women; small subgroups and multiple comparisons limit confidence. The results do not show a single gene determining illness. Inferential probabilities are not a required recall item for this study.

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Continued

Beck: negative schemas bias attention and interpretation. An ordinary setback can activate the triad: "I am inadequate" (self), "others are hostile" (world), and "nothing will improve" (future). Selective attention and overgeneralisation can maintain mood.

Learned helplessness follows experience of uncontrollable outcomes. Attributional reformulation asks why the person thinks bad events occur: internal explanations blame the self, stable explanations predict persistence, and global explanations spread the problem across life areas. These are explanatory dimensions rather than three separate symptoms.

Applying the ideas

After failing one test, "I always fail at everything because I am useless" is internal, stable and global. "This test was difficult and my revision method needs changing" is more specific and changeable. Seligman's therapy evidence relates improvement in explanatory style to reduced depression; the association alone cannot isolate the mechanism.

Evaluation and connections

Oruc evaluation: objective genetic analysis aids replicability, but small subgroups, multiple comparisons, population differences and gene-environment interaction limit conclusions. Critically, the original study concerned bipolar I, although Cambridge places it in the mood-disorder explanations section labelled depressive disorder. Do not relabel its participants as unipolar-depression patients.

Use Oruc accurately: objective DNA comparison tests a proposed association, but its main null result does not support a simple gene explanation. Sex-specific exploratory findings require replication. Biological influences can interact with stress and cognition; do not infer a universal serotonin deficiency from antidepressant effectiveness.