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A-Level Psychology

Clinical Psychology · Key Studies

Edexcel Psychology (9PS0) | Key Studies | 7 entries

The named classic and contemporary studies for Clinical Psychology - diagnosis, schizophrenia and a second disorder.

Classic study

Rosenhan (1973) - On being sane in insane places

Aim: test whether psychiatric staff can reliably tell the sane from the insane, and so question the validity of diagnosis. **Method:** a field study in which eight healthy pseudopatients sought admission to 12 US hospitals, each reporting a single symptom of hearing a voice saying 'empty', 'hollow', 'thud'; once admitted they behaved normally; a follow-up study warned one hospital to expect fakers who were never sent. **Findings:** all but one were admitted, most with a label of schizophrenia, and stayed 7 to 52 days (mean 19); normal behaviour such as note-taking was recorded as pathological, yet many real patients spotted the frauds; in the follow-up 41 genuine patients were judged to be fakes. **Conclusion:** diagnosis is unreliable and the setting, not the person, drives how behaviour is read; labels stick ('in remission') and depersonalise. **Evaluation:** + high ecological validity and a powerful real-world impact on diagnostic practice; − ethically questionable through deception of staff and wasted beds, and the symptom reported was not truly normal, weakening the design.

Contemporary studies (schizophrenia)

Carlsson et al. (2000) - Network interactions and the dopamine hypothesis

Aim: review and update the dopamine hypothesis of schizophrenia by examining how several neurotransmitter systems interact to disturb function. **Method:** a review article drawing together animal studies, drug trials and brain-imaging evidence on dopamine, glutamate, serotonin and GABA pathways. **Findings:** simple excess dopamine cannot explain all cases; low glutamate activity at NMDA receptors can release dopamine and produce symptoms, so several systems are implicated, with mesolimbic and mesocortical pathways acting differently. **Conclusion:** schizophrenia reflects a dysregulated network of interacting transmitters rather than one chemical, pointing to newer drug targets beyond dopamine antagonists. **Evaluation:** + integrates wide biological evidence and explains why some patients fail to respond to dopamine-only drugs; − largely based on animal models, so generalising the precise mechanisms to humans is uncertain, and it underplays social and psychological causes.

Guterman et al. - Symptom interpretation across cultures

Aim: test whether the meaning given to a hallucinated voice depends on culture rather than being a fixed feature of schizophrenia. **Method:** structured interviews compared how patients diagnosed with schizophrenia in different national settings described and reacted to their auditory hallucinations. **Findings:** the content and emotional tone of voices differed systematically by culture, with some groups experiencing benign or guiding voices and others hearing harsh, threatening commands. **Conclusion:** culture shapes the experience and reporting of symptoms, so diagnosis built on Western criteria may misread the same experience elsewhere. **Evaluation:** + supports a less rigid, more cross-culturally valid view of symptoms and warns against ethnocentric diagnosis; − interview self-reports are open to social-desirability and translation bias, lowering reliability.

Contemporary studies (second disorder)

Guardia et al. (2012) - Body-size estimation in anorexia (anorexia nervosa)	**Aim:** test whether women with anorexia misjudge whether their body can pass through a gap, showing a distorted body image. **Method:** 25 patients with anorexia and 25 healthy controls judged from a distance whether they could fit through door-like openings of varying width, then their actual ability was measured. **Findings:** patients overestimated their own body size, saying they could not pass through gaps they objectively could, while their judgement of a neutral object was accurate. **Conclusion:** anorexia involves a specific distortion of the perceived body rather than a general perceptual fault, an over-large internal body schema. **Evaluation:** + an objective, replicable measure that isolates body-image distortion; − a small clinical sample limits generalisability and it cannot show whether the distortion causes or results from the disorder.
Brown et al. - Heritability of anorexia from twin studies (anorexia nervosa)	**Aim:** estimate the genetic contribution to anorexia by comparing concordance in identical and non-identical twins. **Method:** twin pairs in which at least one had an eating disorder were assessed for diagnosis, and concordance rates for monozygotic and dizygotic pairs were compared. **Findings:** identical twins showed markedly higher concordance than non-identical twins, giving a high heritability estimate for anorexia. **Conclusion:** there is a substantial genetic predisposition to anorexia, though shared environment also plays a role. **Evaluation:** + a quantified biological estimate supporting a diathesis explanation; − the equal-environments assumption may be false because identical twins are often treated more alike, inflating the apparent genetic effect.
Lemonick / Bachmann - Brain dysfunction and obsessions in OCD (obsessive-compulsive disorder)	**Aim:** investigate the brain circuits that underlie the unwanted thoughts and repeated actions of OCD. **Method:** a review of imaging and case evidence comparing activity in the orbitofrontal cortex, caudate nucleus and related loop in patients with OCD against controls. **Findings:** the worry loop linking orbitofrontal cortex and basal ganglia is overactive in OCD, so a 'something is wrong' signal is not switched off, driving compulsions; activity reduces after successful drug or behavioural therapy. **Conclusion:** OCD has an identifiable neural basis in a hyperactive cortico-striatal circuit, supporting a biological model alongside learning explanations. **Evaluation:** + objective brain evidence that links symptoms to a specific circuit and predicts treatment response; − imaging shows correlation not cause, and a purely biological account neglects the role of learned associations and anxiety.
Lewinsohn et al. - Predictors of first-onset depression (major depressive disorder)	**Aim:** identify the factors that predict whether adolescents go on to develop major depression. **Method:** a longitudinal study followed a large sample of adolescents over time, measuring psychological, social and cognitive variables and recording later onset of depression. **Findings:** several factors predicted first onset, including earlier subclinical low mood, negative thinking style, poor self-esteem and stressful life events, with effects interacting rather than acting alone. **Conclusion:** depression arises from an interaction of vulnerability and stress, supporting a diathesis-stress rather than single-cause model. **Evaluation:** + a prospective design that can suggest causal direction and inform early prevention; − reliance on self-report measures risks bias, and findings from one age group may not generalise to adults.