PSYCHOPATHOLOGY

Belfast APT 2013

OVERVIEW

What is abnormality (definitions)
- 4 definitions

Explaining abnormality (models)
- Biological approach
- Behavioural approach
- Cognitive approach
- Psychodynamic approach

DEFINING PSYCHOLOGICAL ABNORMALITY

- Deviation from social norms
- Failure to function adequately
- Deviation from ideal mental health
- Also : *Statistical infrequency*

DEVIATION FROM SOCIAL NORMS

Abnormality involves breaking social rules
- Depends strongly on context
- Norms change over time e.g. homosexuality and vary from country to country - cultural relativity
- Used for social control e.g. Russia, Japan
- Useful for identifying unexpected or 'abnormal' behaviour

FAILURE TO FUNCTION ADEQUATELY

Abnormality as inability to cope
- Difficult to define-affects social and work roles; loss of control, irrational, observer discomfort
- Always an element of value judgment (e.g. cultural factors ,social class, minority groups)
- Not always 'abnormal'; e.g. political protests, eccentricity

DEVIATION FROM IDEAL MENTAL HEALTH

Abnormality as lack of ideal qualities (eg ability cope with stress)
- Criteria reflect western cultural ideals e.g. personal autonomy, self-actualisation
- Very few people match all six criteria!
- Subjective (Jahoda, 1958)
- Useful guide

JAHODA'S CRITERIA

- Positive attitudes to self; self-identity
- Self-actualisation; meet potential
- Resistance to stress
- Personal autonomy
- Accurate perception of reality
- Ability to adapt to the environment

STATISTICAL INFREQUENCY

Abnormality as rare behaviour
- Not always undesirable e.g. high IQ, musical ability
- Disorders such as depression are very common
- 48% in USA will experience psychological disorder at some point in their lives
- Reflects cultural differences e.g. in seeking help e.g. Asian cultures and depression

CONCLUSIONS

- No single adequate definition
- Easier for some conditions (psychoses such as schizophrenia) than others (neuroses such as depression)
- DSM and ICD use a combination of approaches e.g. deviation from social norms, failure to function adequately, personal distress, plus observable symptoms

BIOLOGICAL (MEDICAL) MODEL

- Genetic factors often important
- Changes to brain structure e.g. autism, schizophrenia
- Changes in neurotransmitters e.g. depression (decreased serotonin), schizophrenia (increased dopamine)
- Ignores psychological and environmental factors e.g. depression and life events. But often supported by modern scanning techniques and the effectiveness of drugs in e.g. depression

BEHAVIOURAL MODEL OF ABNORMALITY - CAUSES

- Abnormal behaviour is learnt through operant conditioning (rewards) classical conditioning (association) and social learning
- E.g. Phobias (Little Albert) where 'fear' becomes associated with the 'wrong' object or situation.
- E.g. Anorexia, 'slimness' may be positively rewarded (operant conditioning) . Impact of size zero role models

COGNITIVE MODEL OF ABNORMALITY - CAUSES

- Abnormality caused by irrational, faulty or maladaptive thoughts and perceptions
- Focus on confirming evidence only e.g. to maintain depression
- Beck's Depressive Triad - negative views of the world, the Self, and the future

BECK'S COGNITIVE MODEL

- Negative triad - negative views of self, the world, and the future
- Cognitive biases - arbitrary inference, selective abstraction, magnification, minimisation, internal attributions

PSYCHODYNAMIC (FREUDIAN) MODEL OF ABNORMALITY - CAUSES

- Key is the **dynamic unconscious**
- Early childhood experiences crucial
- Conflict between id, ego, superego
- Failure to resolve psychosexual stages (anal, oral, phallic etc) can lead to **fixation** at that stage, or **repression** of memories into the unconscious; this may then emerge in adulthood as abnormality e.g. early childhood abuse is repressed and emerges as depression in adulthood

PSYCHODYNAMIC MODEL

- Limited patient sample
- Non-scientific
- Theories not falsifiable
- Multiple interpretation of cases
- Overemphasis on sexual development
- Long and expensive
- Treats people as complex
- Importance of the unconscious and childhood experiences

EXAMPLE – EATING DISORDERS

- Anorexia, bulimia can both be used to good effect
- Explanations – Biological & Psychological models
- Research studies (APFCC: one for biological, one for psychological explanations)

BIOLOGICAL EXPLANATIONS

- **Genetics** – Holland et al., Kendler et al.. Concordance rates higher in MZ than DZ twins :
- **Feeding regulation** – damage to hypothalamus, serotonin (animal studies, no direct evidence, but feasible)
- **IMPORTANT:** many (all ?) physiological changes could be secondary to weight loss

PSYCHOLOGICAL EXPLANATIONS

- **Dysfunctional families**: over controlling, perfectionist; merges into psychodynamic and family dynamics (Bruch, Minuchin)
- **Individual:** low self-esteem, depression, perfectionism, body dissatisfaction
- **Socio-cultural:** media images, peer pressure and reinforcement (social learning and conditioning)

CONCLUSIONS

- Complex mix of genetic, biological, social/cultural, family and individual factors (note: many females exposed to risk factors do **NOT** develop EDs)
- Any explanation must cope with upsurge in EDs over the last 20 years, and increase in male EDs.
- Treatment long and difficult, argues against simple biological explanation

A2 PSYCHOPATHOLOGY

- Schizophrenia

- Depression

- Anxiety

WHAT DO YOU HAVE TO KNOW ?

- Clinical characteristics

- Biological explanations

- Psychological explanations

- **Don't forget the evidence**…

SCHIZOPHRENIA

DSM Definition
- Symptoms for at least one week
- Signs of disturbance for at least 6 months

Two out of:
- Hallucinations
- Delusions
- Catatonic behaviour
- Flat affect (no emotion)

Note
- Two patients may have NO symptoms in common

GENETICS

Relation to the patient and chance of developing schizophrenia
- Spouse..................1.00
- Grandchild..............2.84
- Niece/Nephew.........2.65
- Child.................. 9.35
- Sibling..................7.30
- DZ twin...............12.08
- MZ twin............   44.30
- *Gottesman et al., 1987*

These figures are effectively concordance rates

EVALUATION OF GENETIC INFLUENCES

- Concordance rate for MZ twins only 40-50% - if entirely genetic, should be 100%
- Success of 'expressed emotion' approach in improving outcome for patients with schizophrenia
- Increased frequency of schizophrenia in lower socio-economic groups ('downward drift' ?)
- Increased frequency in first generation Afro-Caribbean immigrants – a real phenomenom ?
- Popularity of diathesis-stress model

THE DOPAMINE MODEL

- The most effective antipsychotic drugs are DA antagonists, reducing DA activity in the brain. Recent atypical antipsychotics act on other systems as well e.g. serotonin

ALSO

- Not all patients respond to drug therapy (approx. 50%)
- No direct evidence for changes in the DA system in the brains of schizophrenic patients. Some findings, but not replicated
- Antipsychotic action takes some days or weeks to develop, but DA-blocking action begins immediately

SCHIZOPHRENIA AND BRAIN DAMAGE

- Johnstone et al., 1976: first report of enlarged ventricle/ brain ratio (VBR) in schizophrenia, suggesting loss of brain tissue.
- A significant minority of schizophrenics do show brain damage, but results are inconsistent; areas involved more frequently include left temporal lobe (including hippocampus) and frontal lobes
- Possibly a result of abnormal development of the nervous system: the neurodevelopmental hypothesis

CROW'S TWO SYNDROMES

- POSITIVE – Type 1
- Positive symptoms
- Good response to drugs
- Acute (short) course
- No evidence of structural brain damage
- Pathology: abnormalities in the dopamine system

- NEGATIVE – Type 2
- Negative symptoms
- Poor response to drugs
- Chronic (long term) course
- Presence of structural brain abnormalities

NON-GENETIC INFLUENCES

Evidence that rates of schizophrenia are increased by:

- Birth complications
- Living in the City as opposed to the country
- Spring birth
- Chronic cannabis abuse

BUT: effects tiny compared to the genetic influence

COGNITIVE APPROACHES (FRITH , HELMSLEY)

Symptoms of schizophrenia have often been linked to problems with perception and attention

Frith (1992) suggests that in schizophrenia, a central monitor fails to function. Because of this the person experiences their own thoughts and actions as originating outside themselves i.e. as hallucinations (inner speech) and delusions (e.g. someone is putting thoughts in their heads)

- Growing area linking cognition and neurology (cognitive neuroscience)
- BUT: no direct experimental evidence

SCHIZOPHRENIA - EVALUATION

- Evidence for genetic factors, but concordance not 100%
- Good evidence for neurotransmitters (DA and others)
- No evidence for e.g. Freudian approaches or 'refrigerator mothers'
- EE therapy suggests sensitivity to family dynamics
- Token economies effective for shaping behaviour, but no evidence for a conditioning basis to schizophrenia
- Evidence for life stress as a factor; the diathesis-stress model probably the best combined approach

DEPRESSION

- TWO TYPES

- Unipolar depression

- Bipolar depression (manic-depression)

UNIPOLAR DEPRESSION

- Five out of (for at least 2 weeks)
- Sad, depressed mood
- Loss of pleasure in usual things
- Sleeping problems
- Appetite changes
- Loss of energy
- Problems in concentration
- Change in activity level
- Thoughts of death and suicide
- Guilt and worthlessness

BIPOLAR DISORDER - MANIC DEPRESSION

- Depression, plus episodes of MANIA:
- Increased activity level
- Rapid speech
- 'Racing' thoughts
- Inflated self-esteem
- Less sleep than normal
- Distractibility
- Risky activities

BIOLOGICAL EXPLANATIONS

Monoamine hypothesis
- Depression associated with low levels of brain serotonin (5HT)
- Antidepressant drugs work by increasing serotonin

Permissive Theory
- Depression caused by low levels of 5HT and NA
- Mania caused by low levels of 5HT and high levels of NA

- Genetics: *Bipolar disorder*, MZ concordance rate 68%, DZ rate 23% (Price, 1968).
- *Unipolar disorder*, MZ rate 53%, DZ rate 28% (McGuffin, 1991) BUT: Kendler 1992, MZ rate 48%, DZ rate 42% - little difference

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BIOLOGICAL EXPLANATIONS – EVALUATION

- Drugs increase serotonin levels rapidly, but antidepressant action takes days or weeks to develop – complex effects on the brain ?
- MZ concordance rates, especially for unipolar depression, much less than 100%; evidence for non-genetic factors
- Ignores cognitive and socio-economic factors
- Drugs only work for 50-60% people; BUT can be very effective when they do work

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PLACEBO EFFECTS

- In studies comparing an active drug (antianxiety or antidepressant) with an inactive placebo in naïve patients, a good response to the drug may occur in 50-60% of the sample, but there can be a 20-40% response to the placebo.
- So, BELIEFS and EXPECTANCIES can have a powerful effect; **psychological factors**, not biological

COGNITIVE MODELS - LEARNED HELPLESSNESS

- Depression linked to stressful events over which you believe you have no control
- Dogs given unavoidable shocks fail to escape when given the chance (Seligman, 1974)
- Similar results with humans and unavoidable white noise (Hiroto & Seligman, 1975)

- So, you learn that trying to cope is 'hopeless', and this leads to depression

COGNITIVE MODELS - ATTRIBUTIONAL STYLE

- Depression caused by global, stable, internal attributional style
- e.g. failing psychology exams……..
- **Internal** - I am stupid
- **Global** – 'I am bad at everything'
- **Stable** – 'I will always be bad at everything'

BECK'S COGNITIVE MODEL

- Negative Triad - negative views of self, the world, and the future
- Negative schemata - see world in negative ways
- Cognitive biases - arbitrary inference, selective abstraction, magnification, minimisation etc

COGNITIVE MODELS - EVALUATION

- Some experimental support (learned helplessness) from animal work: can it be generalised ? Problem of EXTRAPOLATION
- Human work: Alloy 1999 - long term study of students, pessimistic cognitive style predicted first onset of major depression (17% v. 1%) and relapse (27%v6%)
- BUT: Depressive realism - nondepressed over-estimate their control over events

COGNITIVE MODELS - EVALUATION

- Emphasises cognitive factors
- Lewinsohn 1981 - NO causal link in community survey of depression and negative triad and schemata
- BUT...Brown 1995 - more likely to develop depression under stress if negative cognitive style

GENERAL EVALUATION

- Evidence for both biological and psychological explanations
- Strong genetic evidence for BD
- Strong evidence for cognitive explanations of unipolar depression, including success of cognitive-behavioural therapy
- Both approaches ignore socio-economic factors (Brown and Harris, 1978; depression associated with poverty and lack of social support. Convincing animal work that social isolation leads to depression)

EFFECTIVENESS OF THERAPIES – ELKIN ET AL., 1989

- **Multi-centre, 240 patients with depression**
- **3 Therapies....**
- Cognitive-behaviour therapy
- Psychotherapy
- Antidepressant drugs
- Placebo
- **16 weeks treatment**

FINDINGS

- Large Placebo effect - 35-40%
- All treatments add significantly to the placebo effect; overall quite similar
- Drugs best for severe depression
- Individual therapist important in psychological therapies
- **NO RADICAL DIFFERENCES IN EFFECTIVENESS**

ANXIETY SYMPTOMS - DSM

- Excessive anxiety for 6 months or more, plus six out of:
- trembling
- muscle tension
- tiredness
- palpitations
- sweating
- dry mouth
- nausea, diarrhoea
- flushes, chills
- sleeping problems
- irritability
- feeling tense
- difficulties in concentrating

ANXIETY

- Anxiety is a common state that is present in most people to some extent all the time.
- Because of this it is CO-MORBID (occurs alongside) many other conditions (e.g. depression, eating and sleep disorders, personality disorders, schizophrenia, organic disorders etc).
- This means that identifying a specific cause for anxiety disorders is extremely difficult.

PHOBIAS

- Fear of situations, objects, or animals, leading to high states of anxiety and in some cases full-scale panic attacks.
- Classified into Agoraphobia, Specific phobias (e.g. animals, natural environment such as heights or storms, or situations such as flying, tunnels etc), and Social phobia

BIOLOGICAL EXPLANATIONS

- Increased risk of social phobia in first degree relatives of phobics (Kessler, 1998); 16% compared with 5% in relatives on non-phobics
- 66% first degree relatives of blood-injection phobics also had it (Ost, 1989)
- Possibly what is inherited is highly reactive bodily arousal systems, so phobias more likely to develop
- Against is lack of drug effectiveness in phobias, and success of behavioural methods

PSYCHOLOGICAL EXPLANATIONS

- Behavioural - Watson and Little Albert. UCS (fear) comes to be associated with the white rat by classical conditioning
- **'Preparedness'** (Seligman); we are evolutionarily prepared to 'fear' certain biologically significant stimuli (e.g.snakes) which condition more easily than other stimuli (e.g. flowers- Ohman, 1976)

PSYCHOLOGICAL EXPLANATIONS

- Psychodynamic - Freud and Little Hans: anxiety created by the Oedipal complex is unconsciously displaced onto horses, which symbolise his father.
- No direct evidence from this or other cases. Psychotherapy for phobias is not usually effective.

PSYCHOLOGICAL EXPLANATIONS

- 'Preparedness' is partly a biological explanation, based on evolutionary/inherited characteristics
- In general, people often find it hard to identify a single 'conditioning' traumatic experience
- But therapies based on the behavioural approach are very effective, especially for specific phobias

DAVIDSON ET AL., 2004

- **295 patients with generalised social phobia**
- Compare:
- Fluoxetine (antidepressant)
- Cognitive behavioural therapy (CBT)
- Fluoxetine + CBT
- Placebo

FINDINGS

- All active treatments superior to placebo (19% response rate)
- After 14 weeks, no difference between treatment groups
- No superiority of combined treatment
- **Up to 50% patients do not respond**