Diagnosing Mental Disorders

Big issue: deciding at which point a behaviour is so abnormal it needs diagnosis + treatment.

Deviance

- extent behaviour is rare within society
- depends on context + culture.
- can also look at statistical deviation (normal distribution)

Dysfunction

- extent behaviour interferes with pts life.
- all aspects of everyday life looked at.
- eg. stop being productive / don't leave house

Distress

- extent behaviour upsets the individual
- look at in isolation as could still function.
- subjective experience

Danger

- danger to themselves + others should be assessed
- could indicate intervention needed.

Duration (?)

- lots of behaviours could fill 4Ds in short term
- if behaviours persist psychiatric attention could be required.

Limitations:

Deviation
- sometimes good to be statistically rare → doesn't account for desirability of behaviour
- social norms differ in each culture + context.

Distress
- subjective
  ↳ could cause distress for one but normal to another

Dysfunction
- 'fully functioning' is subjective as diff tests give diff results
- can fully function w/ some mental disorders.
- ✓ very observable/easily noticed.

Danger
- can be too late before noticed (danger already occured).
- normal people do dangerous stuff
- subjective.

General
- no clear scale between normal + abnormal
- can be other explanations
- based on clinicians judgement
- 5th D? duration.

Classification Systems

Reliable diagnoses are essential to ensure correct treatment is given

No obvious measurable physiological signs so depends on interpretation of symptoms

Classification systems describe clusters of symptoms that define disorders → should lead to better quality diagnoses.

International Classification of Diseases (ICD)

- currently ICD-10
- all diseases! section F is specific to MHDs.
- groups each disorder as part of a 'family'
  eg. family: mood (affective) disorders
  includes: depression + all its forms
  code: F32 → disorder (depression unipolar)
  MHD section ↙ ↓ family
- decimal points make categorisation even more specific
- use to guide diagnosis through interview
- free on WHO website

Diagnostic and Statistical of Mental Disorders (DSM)

DSM-V

- current!
- linked disorders grouped together
- use w/ info from interview + records (like ICD)
- 3 sections
  ↳ introduction
  ↳ classification of main MHD
  ↳ future + other assessment measures

DSM-IV-TR

- multiaxial tool
- 5 axes

1) major clinical syndromes
2) described symptoms related to personality axis.
3) described medical conditions used to explain onset of clinical issues
4) described psychosocial/enviro probs that could be involved w/ MtHDs eg. being made redundant
5) scale to assess global functioning of an individ.

↓

- score from scale used to help w/ diagnosis + assess need + type of treatment.

Reliability and Validity of Diagnosis.

Reliability → the extent to which clinicians agree on the same diagnosis for the same patient.

Validity → the extent to which a diagnosis genuinely reflects the underlying disorder.

Reliability

Inter-rater Reliability

- used to test reliability levels
- show 2+ clinicians details of a person's case history
- assess level of agreement between them
- early diagnostic symptoms had low irr

Patient Factors

- give inaccurate info
  ↳ memory/denial/shame etc
- specific issues eg. disorganised thoughts can also cause issues

Clinician Factors

- focus on certain symptom presentation
  ↳ unstructured nature of interview
  ↳ different info gathered
- subjective judgement
  ↳ depends on background/training/exp. of clinician

RELIABLE ≠ VALID (see Rosenhan!)

Validity

Concurrent validity

- look at another diagnostic tool + see if it agrees

Aetiological Validity

- examine what you know about the causes + match to person's history

Predictive Validity

- predict future course + see if it applies
- look at effectiveness of treatment

Implicit Bias

- preconceived ideas
- eg. women can't have schiz

Comorbidity

- 2 disorders @ same time
- May only diagnose one → invalid
- Diff clinicians may pick up on diff aspects → unreliable

Schizophrenia

Spectrum of psychological disorders that are characterised by abnormalities

Symptoms

Positive (Type 1) → ADD to experience of patient
Negative (Type 2) → SUBTRACT from normal behaviour
→ persist longer + burden of care.

Delusions

beliefs held by the individual that, despite not true, cannot be changed by others
- grandoise delusions → remarkable qualities
- persecutory delusions → someone out to get them

Hallucinations

perception of external stimulus but without any actual stimulus present
- auditory hallucinations → hearing things that are not there

Disorganised Thinking/Speech

ideas are loosely connected
- thinking best diagnosed from speech
- skip from topic to topic in convo

Catonia

significant decrease in the individual's responsiveness to the environment.
- sit completely still in odd postures
- continued, repetitive movements.

Avolition

general lack of motivation to complete usual, self-motivated tasks eg. work
- behavioural state
- NEG as reduction in normal functioning.

Features

Prevalence

- 0.3 → 0.7% of developing schiz
- MALES → higher proportion of neg symptoms
  → longer duration of the disorder
- late adolescence to mid 30s.
- episodes develop gradually over time.

Prognosis

- 20% of diagnosed respond well to treatment
- Large % remain chronicly ill w/ regular treatment

Other

- many pts show cognitive functioning defecits.
  ↳ working memory, language, speed of info process
- mood abnormalities are common

Diagnosis

- 2 or more of key symptoms ~~present~~
- present for high proportion of last month
- one symptom must be in blue
- clinician must consider pts life
- brain damage/substance misuse could account for altered behaviour → should be considered.

Neurotransmitter Theory

Dopamine is thought to be the key neurotransmitter associated with psychosis

Pts that had abused large amounts of amphetamines showed +ive symptoms of psychosis

Randrup and Munkvad (1966) raised dopamine levels in rat's brains by injecting with amphetamines. They became stereotyped, agressive + isolated. Dopamine level change = psychotic behaviour

This evidence led to the dopamine hypothesis

↓

a significant link between overstimulation of dopamine receptors and schizophrenia

Recent version refers to the hypersensitivity of D2 receptors

Lieberman et al (1987) states about 75% of pts with schiz show new symptoms or increase in psychosis after taking amphetamine

Owen et al (1978) in post-mortems find a higher density of D2 receptors in certain areas of the brain in schiz pts.

Seeman et al (2013) - amount of receptors account for only 6% increase from normal. Schiz pts may have a higher no. of D2 receptors

Evaluation

✓ Typical anti-psychotic drugs work by blocking dopamine receptors

X Not all schiz pts respond to this ^ medication

X New atypical drugs (eg. clozapine) also block serotonin receptors = more than 1 nt!

X Clozapine been found to increase dopamine in parts of the brain

✓ When Parkinson's pts dose of L-Dopa (dopamine antagonist) is too high they experience schiz symptoms
↳ only Type 1 (+)!

X Correlational research! Schiz may cause chemical changes in brain rather than vice versa

X Dopamine antagonists can cause up-regulation (no. of receptors increase)

✓ Can explain neg symptoms: ↓ dopamine in mesocortical pathways, linked to mesolimbic synap system, can lead to flattened effect.

Genetic Explanation

There is evidence of a strong heritable factor in the development of the disorder

General population chance of schiz: less than 1%

Gottesman (1991) found a 48% chance of diagnosis if you have a MZ twin with schiz.

The greater the degree of genetic relatedness = higher the risk of developing the disorder

Evaluation

X failed to isolate a single gene that causes schiz

X Harrison and Owen (2003) report up to 6 genes involved in susceptibility to disorder

X Family studies don't recognise that findings could be due to similar environment.

X Concordance rate for MZ not 100% → other factors.

✓ Adoption studies account for enviro factors eg. Tienari (2002) found 7% of adoptees w/ schiz had bio mothers with schiz

X children usually placed w/ families of similar background (enviro similarities)

X Can explain using diathesis-stress model

Cognitive Theory

✡ Attributes five symptoms to biological causes

↓

eg. Hallucinates bc of bio cause (eg. ↑ dopamine) BUT when they try to understand experience they begin to show other symptoms

Hallucinates (bio cause) → Asks others if they can see/hear (they say no) → Become wary of others + don't trust

↓

Creates paranoia/ delusions of persecution (Think others are deliberately denying)

Frith's Work (1979)

- Schiz pts have increased self awareness

↓

Inability to filter out unnecessary cognitive noise from internal info processing

- May experience their thoughts as voices telling them what to do
  ↳ try to make sense of + can't = more, worse symp.

Evaluation

✓ Gold and Harvey (1993) ppl w/ schiz score lower on attention, memory, problem solving tests → shows cognitive deficits.

✓ McGuigan (1966) just before auditory hallucinations, vocal centre activated → misinterpreted inner voice

✓ McGuire et al (1966) during hallucinations there's reduced activity in temporal lobe (monitors inner speech) → internal convo

✓ Corcoran et al (1995) schiz pts had deficits in theory of mind (read/interpret others behaviour)

X Underlying cause attributed to bio factors

X Beck et al (2009) reduced dopamine causes brain to struggle more w/ info processing

X Sitskoorn et al (2004) cog deficits in schiz pts also found in family w/o schiz → genetic component to cog deficits

X Correlational research! cause/effect, schiz could cause the deficits

Drug Therapy

Antipsychotic medication is the frontline treatment offered to schiz pts as it helps to alleviate symptoms associated w/ a psychotic episode

Psychotic symptoms can affect a pt's quality of life + make other forms of treatment difficult

The first antipsychotic drugs from the 1950s are known as 'typical' antipsychotics eg. chlorpromazine but they had lots of unpleasant side effects

This led to the development of 'atypical' antipsychotics eg in the 90s eg. clozapine. These have fewer reported side effects. They also reduce some negative symptoms (as well as positive) - which typicals do not

Drugs work by helping to reduce the level of dopamine in areas of the brain associated with symptoms. They block D2 receptors which prevents dopamine binding, depolarising the neuron

However, atypical drugs don't bind so tightly and also block serotonin receptors (5-HT2A)

Seeman (2002) suggested it was this difference that reduced side effects of atypicals

Can be given as tablets or injected.

Evaluation

Can have serious side effects eg. rapid heart rate and blurred vision
- Typical drugs can result in tardive dyskinesia
- Atypical have weight gain + metabolic change as side effects which can increase risk of diabetes and high cholesterol
- Risk of fatal blood disorder → see below ~~look up~~.

Because of the side effects, people stop taking the medication which has consequences for the effectiveness of the drug (Lieberman 2005)

Clozapine → most effective → fewer side effects BUT increases risk of agranulocytosis which reduces white blood count → tests every 2 weeks

McEvoy (2006) found that out of 4 atypical drugs, clozapine was the most effective as it was taken for considerably longer. Most improvement was made by those taking clozapine.

Social control can be an issue as they have been used to make abnormal behaviour more manageable. Regarded as pharmacological straitjackets.

Can be seen that pharmaceutical companies are more interested in money than helping MHDs

Is it ethical to inject someone w/o permission? You can under Sec 3 of the Mental Health Act. Do they know whats best for them?

Family Therapy

Not a treatment per se → does not cure disorder BUT helps families to cope, offer support, encourage medication compliance and create a manageable family environment.

Develop a support network + collaborative relationship between the family + professionals

Should be offered during course of treatment and has been shown to reduce relapse rates and increase treatment compliance

Important feature: talking openly and educating. Allows 'blame' to be broken down and understanding that symptoms can't be controlled to develop

Family offered info about how medication works and what side effects to expect

Its a chance to discuss day to day concerns by looking at different viewpoints + how to work together

Family may be ashamed or embarassed to talk about effects of schiz to others. It can be frustrating and emotionally draining to live with someone with a MHD

↓

Therapy is opportunity to air concerns, find solutions and develop understanding to reduce neg emotions

↓

Pt feels more supported which can impact effectiveness of treatment.

Evaluation

Family interventions combined with medication were more effective in reducing relapse rates than medication alone - Goldstein and Mirowitz (1995) - BUT they said that type of intervention affected level of effectiveness

Pilling et al (2002) compared family therapy and CBT for schiz and found family therapy was effective for reducing relapse rates for episodes of psychosis and improved compliance with medication prescribed

Relies heavily on whole family being open and honest as well as willing to work with the therapist.
↳ lack of commitment can account for drop-out rates

Not as effective by itself

Not widely available on NHS BUT reduced relapse rates would save NHS money

Not designed to relieve symptoms but to reduce expressed emotion and prevent relapse ~~and~~ through medication + support.

Anorexia Nervosa

↓

eating disorder characterised by persistent low body weight in the sufferer

Symptoms:

There are 3 criteria that must be met to be diagnosed

- restriction of energy intake resulting in body weight below expected for age and height
- intense fear of gaining weight or participating in persistent behaviour that will interrupt weight gain eg. excessive exercise
- distortion in body image (overestimation)

Features:

There are 2 subtypes:

✡ restricting → weight loss or weight gain prevention within the past 3 months

✡ binge eating/purging → recurrent bouts of binge-eating alternated w/ purging during past 3 months

- Usually diagnosed during adolesence
- Significantly more females are diagnosed
- Onset often coincides with life stressor eg. start uni
- Amenorrhoea is an associated physical effect
- Higher prevalence in high-income countries

Genetic Explanation

May be an inherited factor to development

Grice et al (2002) looked at families where one person was diagnosed with anorexia, at least one other family member had been diagnosed w/ an eating disorder. When focused on group where 2 relatives diagnosed w/ restrictive type they found evidence for a susceptibility gene on chromosome 1

Scott van Zeeland et al (2013) compared 152 genes in sample of women w/ an and those without and found sig. differences in and around the EPHX2 gene. Produces enzyme → metabolises cholesterol. Suggests disorder may be caused by disruption in how body processes cholesterol which would affect both mood + eating behaviours.

To support:

- Anorexia pts often observed to have higher cholesterol than expected
- In pts with depression, weight loss can lead to an increase in cholesterol levels

✡ NEXT: How do these genetic factors cause + maintain behaviours associated w/ anorexia nervosa?

There may be a number of genes associated

Evaluation

May be multiple factors that cause illness. eg. genes may increase risk but societal/cultural factors may trigger illness (diathesis stress)

Family studies do support genetic explanation BUT they share an environment which could be the real cause

correlational so we cannot establish
Research is^ cause and effect so genetic changes could be a result of the disorder rather than a cause.

Well documented that poor diet and malnutrition can cause biological changes in the body

Difficult to separate maternal health + foetus effects and genetics passed on. Therefore hard to tell if illness is due to genes or prenatal factors.

Sociocultural Theory

Felt the A.N is more likely to occur in social/cultural groups that emphasise 'slim is beautiful'

Anorexia more likely to develop in dance or modelling students than others because a slim body is valued as part of their image - Garner and Garfinkel (1980)

Jobs w/ emphasis on body weight have greater risk of developing eating disorders

Rackoff and Haing (2006) created the female athlete triad of anorexia, athletics and amenorrhoea showing link between these 3, young women and intense physical activity.

Garner et al (1980) found that between 1970-80 there was a huge increase of diet articles in women's magazines → suggests a societal preoccupation to lose weight

BUT we are all exposed, why do only some develop?

Hoek et al (2005) found that influence of cultural ideals ~~even~~ is strong even when immersed in culture w/ diff values as in Curcao they ~~have~~ see overweight as socially acceptable so almost no anorexia but zoomed in on minority white population, anorexia levels almost the same as USA

Evaluation

There are many people who are not influenced by these ideals to develop this order (individual differences) which suggests sociocultural factors are one of many

↓

They may only influence those with a predisposition to the disorder (diathesis stress model)

- To support the theory: diagnoses have increased since 1950s, when there was a change to slimmer models

↓

- More males diagnoses relates to increase in more diet + fitness in men's magazines

↓

Relationship between changes in cultural views and increased diagnoses of anorexia

Drug Therapy

NICE states drugs should not be used as primary or only treatment for anorexia

Very little evidence to show they are useful

Drug therapy may be very effective for those with comorbid conditions eg. depression

Selective serotonin reuptake inhibitors (SSRIs) which are a form of antidepressant. They block the reuptake of serotonin meaning there is more in the synapse. Therefore more passed to post synaptic neuron → greater levels of serotonin.

Olanzapine is another common drug for treatment and is an atypical antipsychotic also used to treat anxiety. It blocks absorption of dopamine and serotonin in certain pathways.

Use of medication to treat comorbid symptoms may enable the pt to benefit more from psychological therapies.

Evaluation

Some anorexia sufferers already have serious heart problems so drugs can be risky as some have cardiac side effects

Pts taking SSRIs and olanzapine find that they gain weight which can be difficult to cope with (although desired). Can make it hard to continue medication.

Ferguson et al (1999) found no significant difference in terms of clinical symptoms and reports of anxiety between those taking SSRIs and those not.

Kaye et al (2001) found that people fluoxetine were more likely to stay on medication up to a year after outpatient treatment, and had lower relapse rates.

Jensen and Mejlnede (2000) found positive effects for 3 patients treated with 5mg of olanzapine a day. (Effects on body image). Suggest antipsychotic makes body image more realistic BUT first 2 months difficult due to hunger + weight gain side effects.

↓

BUT small sample! (case study of 3)

Lots of success can be attributed to psychological therapies given alongside

Cognitive Behavioural Therapy

Enhanced cognitive behaviour therapy (CBT-E) has been specifically developed for eating disorders.

Aimed at tackling the thoughts and behaviours associated with an eating disorder

Conducted on a one-to-one basis and course of about 20 sessions initially advised (40 if significantly underweight)

Detailed interview (over 2 sessions) to assess pt's suitability and allow them to ask qs.

Stage 1:
- 4 sessions / 2 per week
- to encourage rapid change in behaviour
- weekly weighing and regular eating
- educated about disorder + treatment programme

Stage 2:
- 2 appts / 1 week apart → brief
- discuss progress and 'take stock' of how pt is

Stage 3:
- 8 appts / 1 per week
- tackle factors involved in maintenance of disorder eg. body image, dietary rules + event-related changes
- look at behaviours eg. body checking that make them feel fat + impact of rules on QOL

Stage 4:

- look to the future
- consider factors that need to be managed to prevent relapse
- 3 appts / 2 weeks apart
- personalised agreed plan drawn up
- consider mindset: relapse ≠ failure
- post-treatment review appt 5 months later

Evaluation

Client has to be motivated or not effective

It is flexible so can be adapted to pt's needs

Most common form only effective when disorder not maintained by clinical perfectionism, self-esteem or interpersonal probs. BUT there are other forms for these groups.

Pike et al (2003) found relapse rates for CBT were significantly lower than nutritional counselling.

Byrne et al (2011) found 2/3 showed significant improvement in symptoms of all EDs after CBT-E

More suited to those with independent access to treatment + living away from family (its about taking control)

Individual Differences

Race and culture are issues in diagnosis
- common behaviour in one culture could be seen as symptomatic of a disorder in another
- if clinician is different race/culture, a pt may not share their symptoms due to a sense of cultural shame
- 'normal or not' boundary varies across cultures.

Culture bound syndromes are examples of cultural differences. eg. ghost sickness. DSM and ICD fail to include these syndromes despite being 'universal and scientific'

In sociocultural theory we realise that there are factors that cause anorexia which we are all exposed to. However, we don't all develop it which suggests individual differences are a factor.
- pts with anorexia score highly on measures of perfectionism (a personality trait)
- Hoek et al (2005) found that it is socially acceptable to be overweight in Curaçao + they had lower incidence of anorexia.

Developmental Psychology

The latest DSM shows an understanding that some psychiatric illnesses occur during certain periods in our development by starting with disorders that occur early in life

Schizophrenia can be explained through the

process of development. The neurochemical imbalance could be affected by many thing inc. genes from conception, prenatal factors eg. maternal illness and chemical exposure eg. drugs used throughout life.

Schizophrenia develops in late adolescence/early adulthood so developmental psychologists need to establish what social/emotional changes happen during this period in life that could account for its onset