SCHIZOPHRENIA

CLASSIFICATION AND DIAGNOSIS

Issues of reliability:
- Inter-rater reliability: whether two independent assessors give similar diagnoses – Carson, publication of the DSM-III specifically designed to tackle this, and succeeded(?)
- Whaley, reliability as low as +.11, still little evidence for high reliability – Rosenhan study highlights further problems
- Mojtabi and Nicholson, 50 US senior psychiatrists differentiated between ‘bizarre’ and ‘non-bizarre’ delusions with only +40 in-rater
- Test-retest reliability: whether tests to deliver the diagnoses are consistent over time – cognitive screening tests such as RBANS important as they measure neuropsychological impairment
- Wilks et al., 2 forms of test to schizophrenics over interval of 1-134 days, test-retest reliability high
- Precott et al., re-tests of several measures of attention and information processing in chronic schizophrenics was stable over 6 months

Issues of validity:
- the extent that a diagnosis represents something real and distinct from other disorders, and how far a classification system (ICD or DSM) measures what it claims to measure – a diagnosis cannot be valid if it is not reliable!
- Comorbidity: Buckley et al., comorbid depression in 50% of patients, lifetime diagnosis of substance abuse in 47% of patients – creates difficulties in diagnosis and decisions on treatment
- Weber et al. 6mil discharge records, many comorbid medical problems, diagnosed with SZ often lower standard of care
- comorbid depression major cause of suicide – Kessler et al. attempt rate 1% for SZ alone, 40% comorbid mood disorder
- Klosterkötter et al., assessed 489 admissions in Germany, positive symptoms more useful for diagnosis
- many symptoms found in other disorders – Ellason and Ross, those with ‘dissociative identity disorder’ have more SZ symptoms than actual schizophrenics!
- Prognosis: Bentall et al., 20% recover previous functioning level, 10% lasting improvement, 30% some improvement with relapses

Cultural differences/factors:
- Copeland, patient description to diagnose; 69% US but only 2% British
- Harrison et al., African-Caribbean 8x more likely to be diagnosed, aside from poor housing and social isolation may be due to differences in language/mannerisms between patient & clinician

BIOLOGICAL EXPLANATIONS

Genetic factors:
- Family studies: Kendler et al., first-degree relatives 18x more at risk – Gottesman, children with 2 SZ parents concordance rate of 46%, 13% if 1 SZ parent, 9% if SZ sibling
- concordance more to do with common rearing patterns – negative emotional climate in some families may trigger SZ
- Twin studies: Joseph meta-analysis, concordance rate 40.4% for MZ twins, 7.4% for DZ twins – research shows that MZ concordance compared to DZ is twice as high of greater ‘genetic similarity’ not ‘environmental similarity’
- ultimate is less than 50% MZ risk – genes can’t offer whole explanation
- Adoption studies: Tienari et al., 6.7% of 164 adoptees with SZ biological mother developed SZ, 2% of 197 with non-SZ mother developed SZ – genetic liability been ‘decisively confirmed’
- methodology: most adoption studies wouldn’t have found significant diffs between adoptees born to SZ or non-SZ parents without broadening definition to include non-psychotic ‘SZ spectrum disorders’ – e.g. Kety et al., no full SZ in first-degree relatives of adopted ‘SZ spec. dis.’ children

The dopamine hypothesis:
- SZs thought to have abnormally high numbers of D2 receptors, resulting in more dopamine binding and more neurons firing
- Amphetamines: dop. agonist stimulating nerve cells containing dop. and causing synapse to be flooded with neurotransmitter – large does can cause characteristic hallucinations/delusions
- Antipsychotic drugs: dop. antagonist, blocks activity in brain, can eliminate SZ symptoms
- Haracz, post-mortem SZs with elevated dop. levels had received antipsychotics before death – neurons struggle to compensate for deficiency and thus dop. levels actually rise!
- Davis et al. meta-analysis, 29 studies of antipsychotics vs placebo: 19% relapsed on antipsychotics, 55% on placebo
- Parkinson’s disease: low levels of dop. activity – Grilly, some taking L-dopa (to raise dop. levels) developed SZ symptoms

Evolutionary perspective: Steven and Price, SZ personalities used by ancestors to divide tribal communities when too large, charismatic leader separates himself from main group dogma and forms new community

PSYCHOLOGICAL EXPLANATIONS

Psychological theories:
- Psychodynamic: Freud (1924), regression to a pre-ego stage and attempts to re-establish ego control – SZ an infantile state with symptoms such as delusions (‘of grandeur’) reflecting this
- Fromm-Reichmann, ‘schizophrenogenic mothers’ (overbearing, rejecting) are important influence – little other support beyond reiterations by psychoanalysts
- Oltmanns et al., parents of SZ children do behave differently, but are children’s behaviour the cause?
- Cognitive; biological factors in initial sensory experiences, further features are individual’s attempts to rationalise experiences – e.g. asks others to validate symptoms but they are unable to, believes others are lying, becomes delusional
- Meyer-Lindenberg et al., basis for cognitive deficits via excess dop. in prefrontal cortex (linked to working memory)
- Yellowlees et al., hallucination machine to show patients their hallucinations are not real – no evidence for success yet

Socio-cultural factors:
- Life events: Brown and Birley, patients reported 2x stressful life events compared to control group – Falloon et al., high levels of physiological arousal due to neurotransmitter changes thought to be the stress trigger mechanism involved
- Van Os et al., no link found – bi-directional ambiguity present in studies, e.g. start of disorder may be cause of life event
- Double-bind theory: Bateson et al., contradictory messages from parents prevent internally coherent construction of reality
- Berger, SZs more double-bind statements – reliable recall?
- Hall and Levin meta-analysis, no difference in degree of agreement between verbal and non-verbal communication of parents with SZ child and those with normal child
- Expressed-emotion: family communication style involving criticism, hostility and emotional over-involvement – Linszen, patient returning to high EE family 4x likely to relapse
- Tienari et al., adopted kids with bio SZ parents more likely to become ill, but only if adoptive family was ‘disturbed’
- Kalafi and Torabi, high EE in Iranian culture (overprotective mother, rejecting father), main cause of relapse

Retrospective/prospective: Brown and Birley, 50% life event 3 weeks prior, 12% nine weeks – Hirsch et al., 71 SZs 48 weeks, ‘cumulative’ life event contribution over 12 months pre-relapse

Culture: Jenkins and Karno, EE less common outside West, due to less individualist thus less likely to blame SZ for actions?