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An independent academic psychologist, based in England, who has written extensively on different areas of psychology with an emphasis on the critical stance towards traditional ideas.

A complete listing of his writings at <http://kmbpsychology.jottit.com>.

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1. RESEARCHING SCHIZOPHRENIA: SOME EXAMPLES VERSUS CAMPAIGN AGAINST SCHIZOPHRENIA AND THE SOCIAL CONSTRUCTION ARGUMENT

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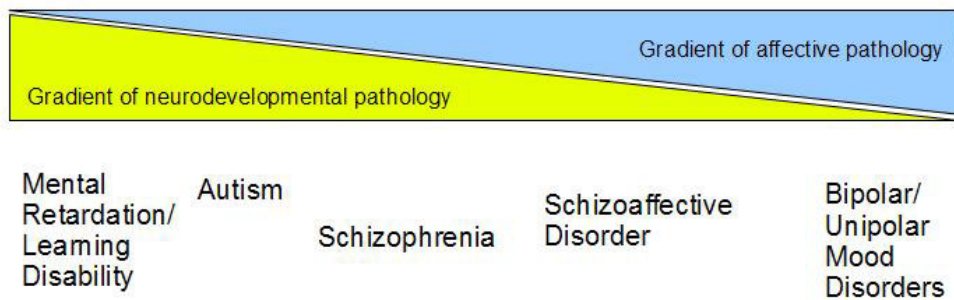
1.1. INTRODUCTION

Emil Kraepelin divided psychosis into schizophrenia and manic-depressive illness (now called bipolar disorder) at the end of the 19th century. In many ways, this distinction still dominates thinking despite the increasing knowledge about psychosis (Craddock and Owen 2010).

Most strikingly, large-scale family studies are showing that there is an overlap in genetic susceptibility between schizophrenia and bipolar disorder. For example, in a Swedish population study (Lichenstein et al 2009), sufferers of schizophrenia were more likely to have a close relative who suffered from schizophrenia or bipolar disorder, and the same for bipolar disorder sufferers as compared to the general population.

Genome-wide association studies have isolated genes common to both schizophrenia and bipolar disorder (eg: Moskvina et al 2009). However, studies like these also show enough differences between schizophrenia and bipolar disorder to rule out a single underlying cause for them. For example, schizophrenia has a stronger developmental component than bipolar disorder (Craddock and Owen 2010).

Craddock and Owen (2005) preferred a model of three overlapping domains for understanding psychosis - "prototype schizophrenia", "schizoaffective", and "prototype bipolar". Craddock and Owen (2010) offered a spectrum varying from mental retardation/learning disability to non-psychotic mood disorder (figure 1.1).



(Based on Craddock and Owen 2010 figure 1 p93)

Figure 1.1 - A spectrum of disorders.

1.2. EXAMPLES OF RESEARCH

There is a lot of research looking for indicators or predictors of developing schizophrenia or psychosis as well as in establishing the characteristics of the disorders ¹.

1. Early onset leads to more hospitalisations

The Danish Psychiatric Central Register (DPCR) has information on all admissions to psychiatric hospitals and psychiatric wards in general hospitals in Denmark since 1969, and all contacts at emergency rooms and outpatient clinics since 1995 (Munk-Jorgensen and Mortensen 1997). The date of admission, date of discharge, and the disorder(s) diagnosed are recorded.

Luo and Huang (2011) analysed a cohort from the DPCR of 286 individuals who were first admitted to hospital between 1st April 1970 and the end of that year with a diagnosis of schizophrenia. During a three-year follow-up period, 106 individuals were re-hospitalised after discharge, and it was found that earlier onset of schizophrenia (ie: before age 20) was associated with shorter gaps between hospitalisations (a 60% increase in risk compared to later onset schizophrenia sufferers).

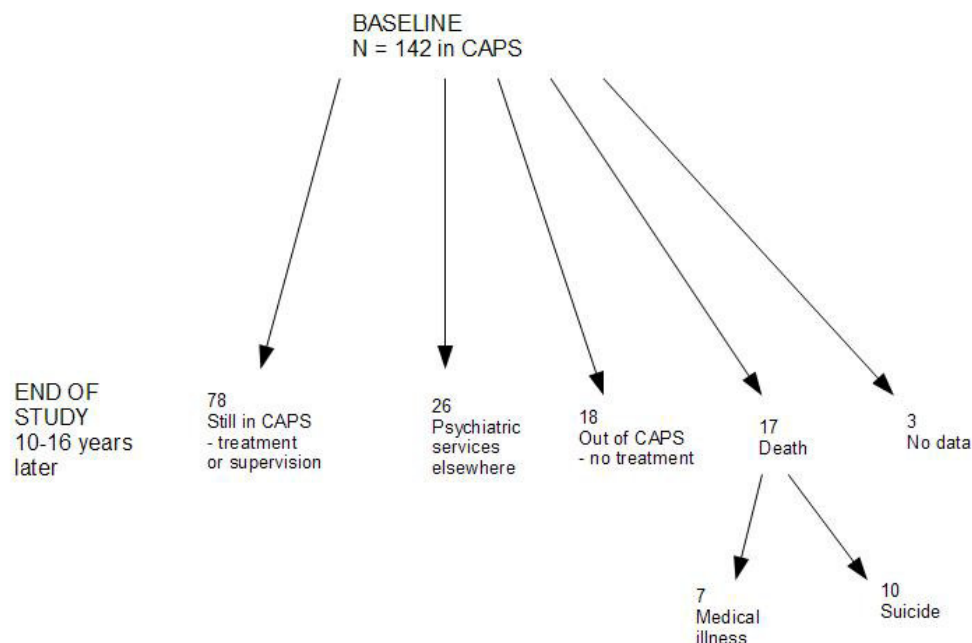
There is often pessimism by professionals about the outcome for individuals with schizophrenia despite developments in treatment. This is partly due to the "clinician's illusion" (Harding and Zahniser 1994). The

¹ A search using Google Scholar (on 20/6/11) for articles with "schizophrenia" in the title found 3860 hits for 2009 and 4410 hits for 2010.

situation where clinicians see only severe cases with poor outcomes, and these become "typical" in their minds.

Long-term outcomes can only be assessed by longitudinal studies ², like Abdel-Baki et al (2011). This involved all individuals with a first lifetime psychiatric admission for DSM-III-R schizophrenia in Montreal, Canada, between 1983 and 1987 (n = 142) (data from Catchment Area Specialised Psychiatric Services; CAPS).

At the end of the study (1999), 15% of the sample (still alive) lived without medical help, and 25% without anti-psychotic medication (figure 1.2). Better outcomes were associated with older age at admission to psychiatric hospital, being married, and being female. But 8% of the sample had ten or more hospital admissions over the study period.



(Based on Abdel-Baki et al 2011 figure 1 p95)

Figure 1.2 - Long-term outcome of 142 individuals with schizophrenia in Canadian study.

Previously, Bland and Orn (1978) reported a fourteen-year follow-up (1963-1977) of 43 first admission

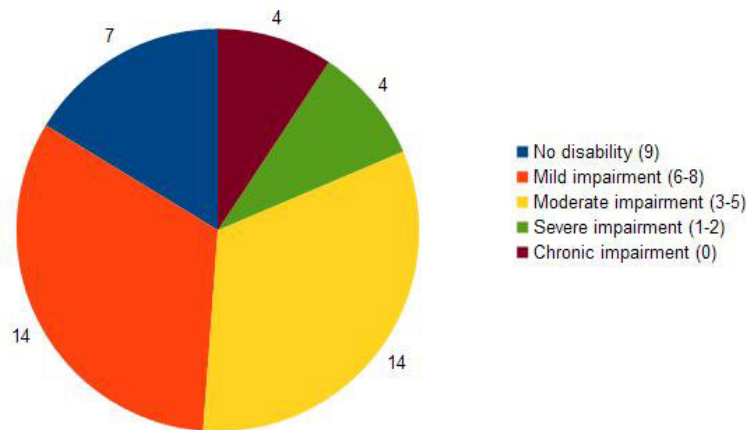
² The methodological issues for this type of study and for comparison between the studies include the location of the study, diagnosis criteria used, representativeness of the sample, length of follow-up period, and outcome measure(s) used (Bland and Orn 1978).

schizophrenics at the Alberta Hospital, Edmonton, Canada. At follow-up, nine were classed as recovered on psychiatric outcome, 22 had periodic bouts of the illness, and 12 had unremitting schizophrenia. On the criteria of economic productivity, 30% were "normal", and 37% had good social adjustment (table 1.1; figure 1.3).

Outcome criteria	Number
Psychiatric condition:	
3 - recovered	9
2 - periodic mild	13
1 - periodic severe/mild unremitting	14
0 - severe & chronic unremitting	7
Social adjustment:	
3 - good (normal relationships)	16
2 - fair (few friends)	12
1 - poor (no close relationships)	9
0 - disruptive (quarrels with any relationships)/reclusive	6
Economic productivity:	
3 - normal (hold job)	13
2 - fair (holds job for short periods)	14
1 - minimal (unemployment > employment)	8
0 - non-productive	8

(Based on Bland and Orn 1978 table 3 p334)

Table 1.1 - Number of patients and scores on three outcome measures.



(Based on Bland and Orn 1978 table 4 p336)

Figure 1.3 - Number of patients and combined outcome measures.

2. Indicators of foetal developmental problems

Neurodevelopmental explanations of schizophrenia see the cause of the disorder in the womb. Any clues to foetal developmental problems could predict adult psychosis. For example, minor physical abnormalities, like polydactyl (duplication of fingers and/or toes) which show developmental abnormalities in the womb. Cardno et al (1998) reported five cases of polydactyl among individuals with psychosis out of 234 individuals with familial psychosis being studied in the UK. This is a significantly higher rate than in the general population.

The neurodevelopmental approach focuses on the early development, but Uhlhass (2011) pointed out the "profound modifications occurring in anatomy, physiology, and functional properties of cortical networks during adolescence that may be important for the emergence of psychosis..." (p480). These changes include to neurotransmitters (Hoftman and Lewis 2011), and excessive pruning of synapses leading to reductions in grey matter of the brain (Gogtay et al 2011).

3. Indicators in childhood (pre-morbid deficits)

There are studies that do not find any difference between children who later develop psychosis and the general population. Traditionally, these are less likely to be published by journals. Done et al (1998) found no evidence of "syntactic deviance" (an inability to apply the rules of syntax correctly) in the childhood essays of individuals who developed schizophrenia in adulthood using a national cohort in Britain (National Child Development Study; NCDS). The NCDS focused on births in the week 3-9 March 1958. Twenty of these individuals who developed schizophrenia and 62 controls from the cohort had essays they wrote at 11 years old analysed by the researchers.

Among high-risk individuals (ie: at least two close relatives with schizophrenia) in the Edinburgh High Risk Study (Hodges et al 1999), those who developed schizophrenia as young adults scored higher on certain measures pre-illness than those who did not develop schizophrenia: "situational anxiety", "nervous tension", "depression", "changed perception", and "hallucinations" (Cunningham Owens et al 2005).

4. Measuring aspects of schizophrenia

The "Cloze" procedure (Taylor 1953) was designed to

test comprehension and predictability, and it involves a passage of text with words missing (usually every fifth one) that the reader has to fill in (table 1.2). The number of errors is scored.

Newby (1998) adapted this procedure (modified Cloze) to use with individuals with schizophrenia by looking at the type of errors made. The incorrect inserted word was categorised as:

- Synonymity - eg: missing word was "flower" and individual guessed "blossom".
- Form class congruence - eg: guessing "mushroom" when "flower" correct.
- Form class incongruence - eg: guessing "coming" when "flower" correct.

Newby (1998) compared three groups of in-patients (schizophrenics, manic-depressives, and non-mental disordered) at St.James's University Hospital in Leeds, England in 1984-5. The individuals with schizophrenia made more errors generally on the Cloze task, and the errors were more likely to be form class congruence and incongruence, and less likely to be synonymity. This showed a loss of predictability in speech comprehension among schizophrenics.

The house was surrounded (i) a large garden which (ii) man from the village (iii) to attend to every (iv) children were often to (v) seen playing in the (vi)...

Correct answers: (i) by; (ii) a; (iii) came; (iv) week; (v) be; (vi) garden

Synonymity - eg: (vi) park

Form class congruence - eg: (vi) street

Form class incongruence - eg: (vi) walking

(Source: Newby 1998 appendix)

Table 1.2 - Extract of Cloze passage.

5. Brain abnormalities

Modern neuroimaging has allowed researchers to see the "live" brain of individuals with schizophrenia, and to record the difference in functioning and structure as compared to non-sufferers.

Magnetic resonance imaging (MRI), for example, shows the structural differences, which include a reduction in whole brain volume as well as in the volume of specific areas like the temporal lobe in sufferers (Lawrie and

Abukmeil 1998). For example, in a meta-analysis of eighteen MRI studies, Nelson et al (1998) reported an average 4% reduction in hippocampus volume among 522 sufferers of schizophrenia compared to 426 controls.

Among the many subtle brain differences attributed to schizophrenics, deficits in lateralisation is one idea (Crow 1990). Usually the two hemispheres of the brain specialise in different abilities, like language. It was believed that the left hemisphere was solely the home of language in most people, but the recent view is that different aspects of language reside in the different hemispheres, though there will still be hemispheric specialisation and dominance (Weisbrod et al 1998). However, Crow (1998) argued that the genetic basis of schizophrenia leads to the brain developing no dominance for speech in one hemisphere, and this "blurs the normal division of labour between hemispheres" (Weisbrod et al 1998).

More recently, Georgopoulos et al (2007) found that the pattern of synchronous neural activity of the resting brain measured by magnetoencephalography (MEG) could distinguish between healthy controls and individuals with disorders like Alzheimer's disease, schizophrenia, and chronic alcoholism. The researchers concluded that "fine-grain synchronicity may be a fundamental aspect of cortical function that can be differentially disrupted by different disease processes, yielding a disease-specific signature" (p354).

Brain abnormalities may be manifest in other ways, like sleep problems. For example, individuals with schizophrenia show differences in sleep length (ie: less total sleep time) and sleep make-up (eg: differences in delta sleep ³) (Kashavan et al 1998).

6. Causes of symptoms

There are many symptoms of schizophrenia based on reality distortion (eg: hallucinations), disorganisation (eg: disordered thoughts and language disturbances), and psychomotor poverty (eg: lack of spontaneous movement) (Stirling et al 1999). One example of reality distortion is "symptoms of alien control", where the individual feels that their body or thoughts are controlled from outside.

Frith (1987) argued that this set of symptoms was a

³ "Delta sleep" describes an electroencephalographic (EEG) reading of 1-2 Hz generated by neurons in the thalamocortical area of the brain.

failure to self-monitor thoughts or actions. For example, a particular thought is perceived as coming from outside rather than from the individual themselves. The failure to self-monitor has been tested in different experimental situations.

For example, Mlaker et al (1994) asked four groups of participants to draw a simple pattern on a computer screen. After a staged "computer crash", participants were faced with four similar drawings and asked to pick out their one. Individuals with schizophrenia and alien control symptoms were poorest compared to individuals with schizophrenia without alien control symptoms, individuals with mental illnesses other than schizophrenia, and healthy controls.

Poor self-monitoring was also shown in a similar experiment by Stirling et al (1998). This time the drawing had to be done out of sight (ie: arm underneath a screen), and the four drawings to choose from were presented at different rotations.

7. Risk factors

A number of pre-natal or early life risk factors for schizophrenia are being reported. For example:

i) Pre-natal exposure to analgesics.

Foetuses exposed to painkillers taken by their mothers in the second trimester was 4.75 times more likely to develop schizophrenia than individuals not exposed (Holger et al 2004). This was based on 7999 individuals born between October 1959 and December 1961 in Denmark (Copenhagen Perinatal Cohort).

ii) Paternal age.

Children born to older fathers have increasing risk of schizophrenia (1.47 times per ten years of paternal age) due to "de novo mutations in paternal sperm" (Sipos et al 2004). The data were 754 330 Swedes born between 1973-80. When the father was under 30, 0.08% of all children developed schizophrenia compared to 0.14% where the father was older 40 years. But this relationship was only where there was no family history of schizophrenia.

The association between father's age and the child developing schizophrenia may not be due to mutations in sperm, but to late fatherhood by individuals with schizophrenia. In the former case, paternal age and conception of any child is important, while in the latter situation, it is paternal age at first child (Petersen et

al 2011).

Petersen et al (2011) attempted to distinguish between the two possible explanations using data from the Danish Civil Registration System of all individuals born between 1955 and 1992. Of 2.2 million persons, 14 211 had developed schizophrenia. If the father was in his 40s at the time of birth of the child, the incidence of schizophrenia amongst those children was 3.74 new cases per 1000 years, and 4.77 if the father in his 50s compared to less than three for fathers under 30 years old.

But if the father was in his 40s when the first child was born, the incidence of schizophrenia was 4.59 and 8.38 for older than 50 compared to less than three again for younger fathers (under 30 years old).

These figures for father's age at birth of first child and subsequent development of schizophrenia amongst the children suggested the late fatherhood explanation was supported.

iii) Virus.

Fox (2011) referred to the popular idea of an "insanity virus". Though laughed at by scientists, recent work on retroviruses has changed this view. Viruses like influenza kill the cells that they infect, whereas retroviruses (like HIV) splice their genes into the DNA of the cell that continues to live.

Perron et al (2008) reported the presence of active elements of a retrovirus, HERV-W, in the blood of half of a sample of 49 individuals with schizophrenia compared to only 4% of the general population sample. Normally, HERV-W is kept under control by the immune system, but an infection shortly before or after birth "re-awakens" the HERV-W, which causes inflammation of the brain, for example, and subsequently schizophrenia (Fox 2011).

8. Genetics

Family studies have shown that schizophrenia has a genetic component. Offspring from both biological parents with a diagnosed mental disorder have higher rates of mental disorders themselves than from one parent with a mental disorder.

Gottesman et al (2010) used the detailed population records kept in Denmark, and concentrated on 2.7 million individuals born there who were aged ten years old before 1st January 2007. Diagnosis of schizophrenia or bipolar disorder in the biological parents were based on use of psychiatric services as in- or out-patients. There were three groups of offspring:

a) Both parents - 196 couples where both parents had a diagnosis of schizophrenia, and 83 couples where both were diagnosed with bipolar disorder.

b) One parent - 8006 couples where one parent had schizophrenia and 11 995 couples with one bipolar disorder sufferer.

c) No parent - 1 080 030 couples had no diagnosis of schizophrenia or bipolar disorder.

Of the offspring of the two schizophrenic parents, 27.3% developed schizophrenia compared to 7.0% with one parent and 0.86% with no parents suffering. The rates of bipolar disorder were 24.9%, 4.4%, and 0.4% respectively. Where one parent was diagnosed with schizophrenia and the other with bipolar disorder, 15.6% of their offspring had schizophrenia and 11.7% bipolar disorder. This study was extensive, but only included cases using psychiatric services, while the diagnostic criteria changed over time (eg: ICD-8 and ICD-10).

Gottesman et al (2010) were aware of the ethical implications of their research, and pointed out that no researcher should be "allowed to be ignorant of the Nazis' barbaric uses during the Third Reich of so-called genetic information to justify their eugenic policies of sterilisation and murder. It is important to keep in mind that the yields from genetic epidemiology and the strategies implemented are applicable to groups of people, not to the individuals themselves" (p256).

In reference to the complexity of the genetics of schizophrenia, Gejman et al (2011) suggested that "a mix of many common and rare mutations" was involved. This links to the idea of "system disorder" ("the dysfunction of entire molecular networks"; Gejman et al 2011).

In the history of studying the inheritance of schizophrenia, it was quickly clear that a single gene was not involved. For example, Ernst Rudin in 1916 proposed two recessive genes together, and over time a more complex polygenic model has been implicated with, today, over 1000 genes having been tested using genome-wide association studies (Gejman et al 2011) ⁴.

1.3. SOCIAL CONSTRUCTION OF MENTAL ILLNESS

Maddox et al (2008), proposing a social constructionist viewpoint, argued that the abstract ideas of psychopathology and mental illness are "not

⁴ Details of the latest genetic research on schizophrenia at <http://www.szgene.org/>.

scientifically constructed but socially constructed". A social constructionist viewpoint is interested in "examining ways in which people understand the world, the social and political processes that influence how people define words and explain events, and the implications of these definitions and explanations - who benefits and who loses because of how we describe and understand the world" (Muehlenhard and Kimes 1999 p234; quoted in Maddox et al 2008 p12). So, social reality cannot be separated from its cultural and historical place.

This means that "if people cannot reach the objective truth about what disorder really is, then viable constructions of disorder must compete with one another on the basis of their use and meaningfulness in particular situations" (Raskin and Lewandowski 2000 p26; quoted in Maddox et al 2008 p12). The understanding of an experience that comes to dominate is "constructed through the implicit and explicit collaborations of theorists, researchers, professionals, their clients, and the culture in which all are embedded and that represent a shared view of the world and human nature" (Maddox et al 2008 p13). In the West, currently, the dominant view is scientific/medical, and in which experiences are phrased as disorders, illnesses, or psychopathology. For example, the experience of cigarette smoking is defined as "Nicotine Dependency", or drinking large amounts of coffee can lead to "Caffeine Intoxication" (Maddox et al 2008). Foucault (1971) highlighted the development of "anxiety disorders" (with associated guilt and paranoia) in the West as the outworkings of societies concerned with surveillance (Putwain et al 2000).

"The social constructionist perspective does not deny that human beings experience behavioural and emotional difficulties - sometimes very serious ones. It insists, however, that such experiences are not evidence for the existence of entities called 'mental disorders' that can then be invoked as causes of those behavioural and emotional difficulties. The belief in the existence of these entities is the product of the all too human tendency to socially construct categories in an attempt to make sense of a confusing world" (Maddox et al 2008 p15).

1.4. CAMPAIGN AGAINST SCHIZOPHRENIA

"'Schizophrenia' is a well-worn word but it is questionable that it denotes any 'thing' - that is, any specific, identifiable disease" (Jenner 2011). The singular term is commonly used, whereas the Swiss psychiatrist, Eugen Bleuler, who coined the term in 1908, used the plural "die Schizoprenien" (German) (ie:

"schizophrenias"). He later reverted to the singular (Jenner 2011).

"The purported discovery of schizophrenia was not at all the result of medical research but only a motivated tautology: over-zealous psychiatrists wished so much that there was a real disease which they could treat medically. The so-said discovery was never more than the proposition of a syndrome: because they wished it were so, influential psychiatrists gathered together the various signs and simply asserted that they constitute the symptoms of one particular disease" (Viriden 2011 p8).

Throughout the 20th century, the distinction has been made between organic mental illnesses, like senile dementia or brain degeneration due to untreated syphilis, where a clear physiological cause is evident, and functional mental illnesses (with psychological causes). Where to place schizophrenia? Psychiatry, with its medical emphasis, would argue the former ⁵. So, this means that treatment should be physiological (eg: medication), and there is limited hope of recovery from the inevitable decline.

Yet studies show about one-third of individuals diagnosed with schizophrenia fully recover and another one-third can function independently with some residual symptoms (Viriden 2011).

The Campaign for the Abolition of the Schizophrenia Label (CASL) (Hammersley and McLaughlin 2008) ⁶ argued that schizophrenia should no longer be used because it is both unscientific and harmful.

Thomas (2011) summarised the main criticisms of schizophrenia:

i) It has no scientific basis - A number of symptoms are used as "clear signs" of schizophrenia, including auditory hallucinations and delusions ("first rank symptoms"; Schneider 1974). But many of these symptoms are often found in other types of psychosis (Viriden 2011).

⁵ The lack of clear biological markers for schizophrenia, despite the search and proposal of many, Papanastasiou et al (2011) argued was because the focus was upon the brain. They proposed that schizophrenia is a "whole body syndrome", and it is related to "natural" ageing processes of the body, but earlier than the norm ("segmental progeria"). Papanastasiou et al (2011) drew the parallel with progeroid syndromes, which are inherited conditions producing premature ageing (or some of its features - "segmental ageing"). For example, Hutchinson-Guilford Progeria Syndrome causes lack of hair, stiffness of joints, and senile appearance.

Kirkpatrick et al (2008) were the first recently to hypothesise that schizophrenia is a "syndrome of accelerated ageing". A number of physiological processes and changes are similar in schizophrenia and normal ageing (eg: reduced brain weight/volume; bone mass changes) (Papanastasiou et al 2011).

⁶ <http://www.asylumonline.net/legacy/casl.htm>.

ii) It is harmful because:

a) It obstructs recovery - The label "schizophrenia" imposes a "narrative of despair". Thomas Szasz (1960), taking an anti-psychiatry position, argued that the negative label of schizophrenia becomes a self-fulfilling prophecy. The expectations of the professionals and others that schizophrenia will never get better becomes the reality.

b) It denies the importance of meaning - The "'illness-model' psychiatry which seeks to yield the secrets of schizophrenia to science; as it does so it endorses the belief that patients are not individuals with meaningful life stories but may legitimately be defined solely on the basis of aberrant biology or brain chemicals" (Longden 2011 p12).

c) It makes people feel powerless - For example, by judging that sufferers lack insight as one of the symptoms, and "having insight" is to agree that you suffer from schizophrenia.

d) It obscures the importance of context - R.D Laing (eg: 1959) preferred to view "schizophrenia" as "a special strategy that a person invents in order to live in an unliveable situation" (quoted in Virden 2011). In this sense, "schizophrenia" is viewed as a form of post-traumatic stress disorder (or complex post-traumatic stress disorder; Herman 1992) with causes based in emotional and psychological trauma (with exhaustion as key; Virden 2011) rather than genetics or biochemistry ⁷.

iii) It leads to stigma - For example, Read et al (2006) found that "biological-caused beliefs" about psychosis were associated with negative public attitudes towards the condition, but "psychosocial attributions" were associated with positive attitudes.

Chadwick (2002), who has argued for the positive aspects of psychotic experience, emphasised how the rational-scientific approach can squash any positive elements of delusions:

The problem of the one-sided selling of the clinician's worldview is also compounded by

⁷ Individuals who reported childhood trauma were more likely to be distressed by psychotic experiences as an adult, and feel that they had less control over the experiences than individuals without childhood trauma (Bak et al 2005).

the fact that in treating delusions the professional often is trying to prove the negative to the patient or service user - who could thus be said to have a logical problem as well as a psychological one. Professionals sat in their consulting rooms can also blithely decide that something "sounds like an hallucination" or "sounds like a delusion to me" simply because what the client has said is merely defined via being subjectively unusual - as if unusual things never happen in life and as if people never lead unusual lives.

If we accept nonetheless that a delusion by definition is fiction we may still see the positive side of this in that delusional thinking may be the release of a fiction-making capacity in the individual and a cryptic revolt not only against fact, reality and functional utility but a revolt against "the world of the middle ground" - which the theorising and value systems in the psychopathology field seem to so cosily inhabit. Patients may be of a genre, unlike psychopathology professionals, but very like artists and spiritual people, who relish and seek altered states of consciousness, unusual experiences, poetic resonance within and dramatic flights of the imagination. This may not be the cognitive or personality style of professional biological and social scientists (Chadwick 2005 p87).

Chadwick (2007) wrote as a "peer-professional" or "user-academic" ("a professional psychologist who... has also been psychotic") about his experiences. He hopes that he is able to bridge the gap between patients and clinicians as "this alienation between healer and sufferer results in the bulk of patients not regarding psychiatrists... as helpful in their recovery".

Reflecting on his psychotic experience, Chadwick noted how interesting connections were made: "I had noticed when writing in my journal that as my own narrative became more dramatic, powerful, and expressive, the rain on the windowpane beat harder and then regularly calmed as my writing calmed. In the very mystical frame of mind I was then in, this seemed to signal some magical connection between within and without". Subsequently, as a psychologist, he has tried to make sense of "psychotic consciousness" (eg: Chadwick 1992, 2001).

In 2002 the Japanese Society of Psychiatry and Neurology changed schizophrenia to "Integration Disorder" to cover "a syndrome based on a stress-vulnerability model" (Hammersley and McLaughlin 2008). Other suggestions for new names include "Post-Traumatic Psychosis" and "Dissociative Psychosis" (Hammersley and McLaughlin 2008).

Harding and Zahniser (1994) presented evidence to

counter seven myths held about schizophrenia by clinicians:

- "Once a schizophrenic always a schizophrenic". Reality - a variety of outcomes.
- "A schizophrenic is a schizophrenic is a schizophrenic". Reality - individuals vary.
- "Rehabilitation can be provided only after stabilisation". Reality - rehabilitation can begin before stabilisation.
- "Why bother with psychotherapy for schizophrenia?". Reality - some therapies have success for some sufferers.
- "Patients must be on medication all their lives". Reality - up to half of sufferers live without medication in the long-term.
- "People with schizophrenia cannot do anything except low-level jobs". Reality - individuals can do different levels of work depending on their abilities.
- "Families are the aetiological agents". Reality - family interactions do not cause schizophrenia by themselves, though they can play a role in relapses.

1.4.1. Pharmaceutical Companies

The social construction of schizophrenia (or more precisely, the social construction of a biological view of it) has been taken further by the pharmaceutical industry, whose "financial muscle... has helped to tip the scales in favour of a predominantly biological view of psychiatric disorder. This has submerged alternative therapeutic approaches" (Moncrieff and Thomas 2002; quoted in Read 2008 p99). Moynihan et al (2002) referred to the "corporate construction of disease" through these companies being "actively involved in sponsoring the definition of diseases and promoting them to both prescribers and consumers" (quoted in Read 2008 p99).

The term "pharmaceuticalisation" has been used to describe this increased role of pharmaceutical companies in society. It is defined as "the transformation of human conditions, capacities or capabilities into pharmaceutical matters of treatment or enhancement (Williams 2009 quoted in Abrahams 2010 p604). Pharmaceuticalisation is manifest as the use of drug treatments instead of other treatments (eg: "weight-loss"

pills over diet for obesity), and the general increase in use of pharmaceuticals ⁸ as "lifestyle drugs" (eg: "Viagra") or off-label use (ie: prescribing by clinicians for conditions outside the drug's licence) (Abrahams 2010).

Abrahams (2010) outlined five factors that go with pharmaceuticalisation:

i) Biomedicalism - The focus upon technological advances to understand medical conditions (eg: use of brain-imaging studies).

ii) Medicalisation - The description of experience in terms of disease and disorders.

iii) Pharmaceutical industry marketing - For example, between 1995 and 2005, marketing staff increased by nearly two-thirds in the UK pharmaceutical industry while research staff numbers declined (Abraham 2010).

iv) Consumerism - Health and illness are viewed in relation to purchasing products. For example, in the USA, direct-to-consumer advertising of pharmaceuticals is allowed, and this encourages consumers to "self-diagnose" and demand the "product" from their physician.

v) Behaviour of regulatory state - For example, reduction in time to review and approve by regulatory bodies after pressure from pharmaceutical industry.

Part of this influence will be through the funding and sponsorship of websites. Read (2008) found that of the top fifty websites in May 2006 on the Google and Yahoo search engines when using the word "schizophrenia", 58% received pharmaceutical company funding (based on displaying company advertisements or acknowledging financial support). The content of drug company funded (DCF) websites was compared to non-drug company funded (NONDCF) ones for causes of schizophrenia given, treatments recommended, how schizophrenia described, and links between violence and schizophrenia made (figure 1.4).

1. Causes of schizophrenia - DCF websites were significantly more likely to refer to bio-genetic explanations (eg: inherited; "brain disease") with no mention of social factors than NONDCF websites ⁹. Read

⁸ For example, NHS prescriptions in England for selective serotonin reuptake inhibitor (SSRI) anti-depressants grew from just under two million in 1993 to over 15 million in 2002 (Abrahams 2010).

⁹ Read (2008) devised scales for measuring each criteria including a five-point scale for measuring

(2008) observed that "the actual causes of, and risk factors for, 'schizophrenia' are many and complex... To minimise, or – as some DCF sites did – ignore altogether, the psycho-social factors in this complex interaction of variables might be interpreted as doing a disservice to the public" (p105).

2. Treatments recommended - Not surprisingly, DCF websites recommended medication-focused treatment over psychological treatments more often than NONDCF websites. Read (2008) commented: "While there is little doubt that anti-psychotic medications help some people there is evidence that the benefits have been exaggerated over recent years, that many do not benefit and that the newer atypical drugs can have serious adverse effects... There is also evidence that a range of psychological treatments are effective for the 'symptoms' of 'schizophrenia'... including CBT..." (p106) ¹⁰.

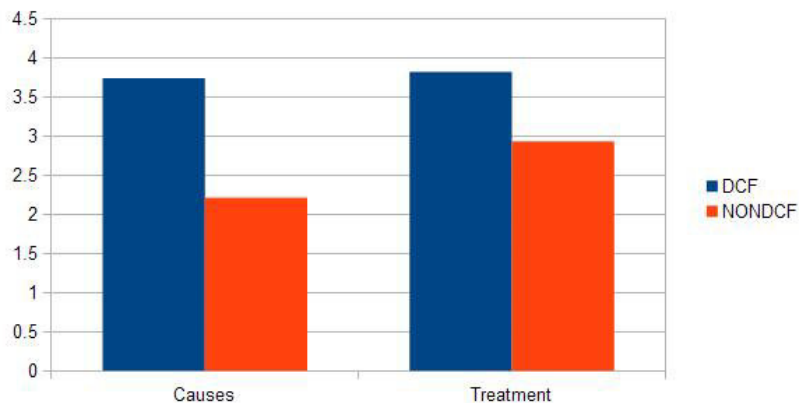
3. How schizophrenia described - Just over half of all the websites described schizophrenia as a debilitating or devastating chronic illness, but this sub-divided into about three-quarters of DCF websites and one-quarter of NONDCF ones. Research shows that on average half of schizophrenia sufferers recover, and there is great variability in the experiences and outcomes of sufferers (Read 2008).

4. Link between violence and schizophrenia - About half of the websites mentioned this, and there was no difference between DCF and NONDCF websites. However, DCF websites were more likely to link violence to coming off

causes of schizophrenia given: "1. Equal emphasis on bio-genetic and psycho-social causal factors. 2. Named psycho-social factors cited as causal, but secondary to bio-genetic causes. 3. Social stressors cited only as events which can trigger a genetic predisposition or exacerbate the existing 'illness' within a 'stress- vulnerability' model. 4. 'Stress-vulnerability' model with the only stressors cited being biological events (eg: birth complications, flu). 5. Purely bio-genetic ('brain disease', 'chemical imbalance' 'genetically inherited disease' etc.); no mention of social factors or stress-vulnerability model" (pp100, 102).

¹⁰ For example, Startup et al (2005) reported long-term benefits in terms of fewer negative symptoms and better social functioning among schizophrenia spectrum disorders sufferers in north Wales given CBT during hospital admission on top of anti-psychotic medication. While Kuipers et al (1998) found long-term benefits (18 months) for CBT with medication-resistant psychosis in London and Eastern England (29% reduction in symptoms compared to 2% in the control group).

medication, or medication preventing violence. Read (2008) concluded: "There is no evidence from the current study that the industry is reinforcing the stigmatising violence stereotype that is central to the prejudice experienced by people with this diagnosis... There is also no evidence that they are challenging it, via their own websites or those they fund" (p106).



(Causes - higher score = more bio-genetic/less social explanation.
Treatment - higher score = more medication focused/less psychological treatments)

(Source: Read 2008 table 2 p104)

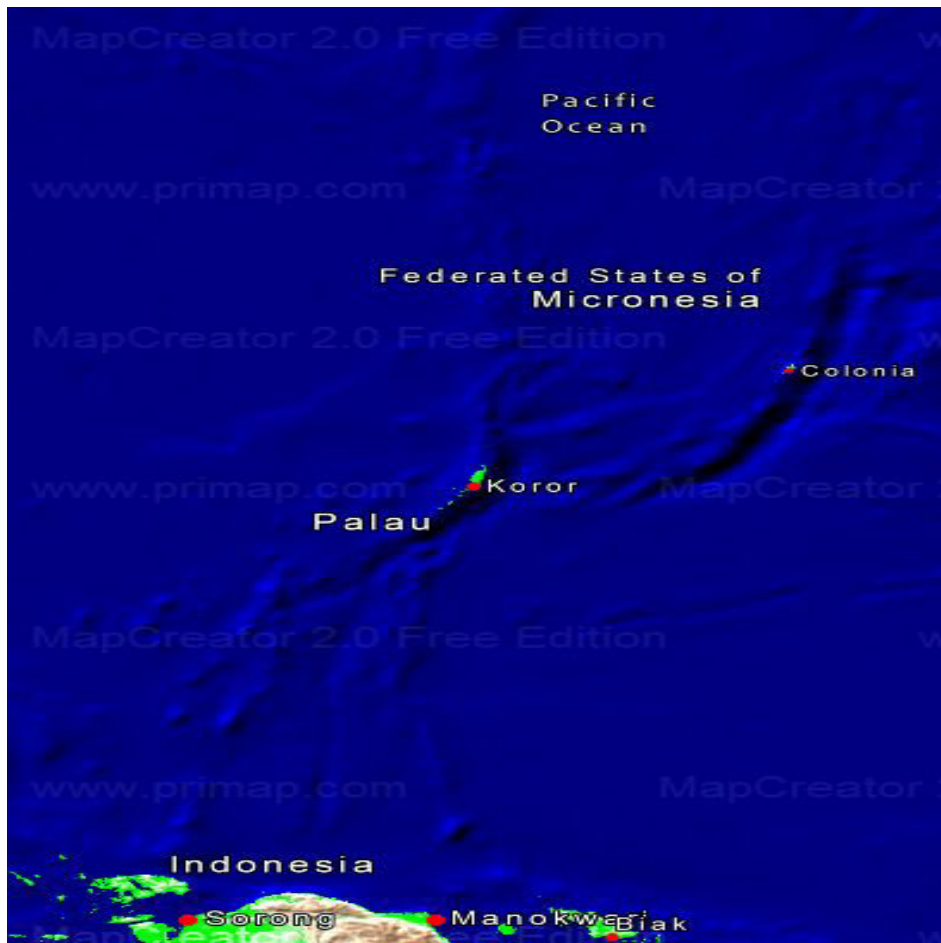
Figure 1.4 - Significant differences on mean scores (out of 5) between DCF and NONDCF websites.

1.5. SCHIZOPHRENIA IN PALAU

Sullivan et al (2007) noted: "The difficulty in identifying 'schizophrenia' at the level of the genotype or the bio-behavioural phenotype indicates that the 'science' of schizophrenia is still at a descriptive stage. Much work remains to be done in terms of gathering basic data about different manifestations of the expression of illness in individuals, in societies, and globally" (p189). The isolated islands of the Republic of Palau ¹¹ (figure 1.5) have become in recent years a "useful laboratory" (Hezel 2007) for investigating this topic.

It is often said that schizophrenia occurs in 1 in 100 people. This figure comes from studies "heavily biased" towards the USA and Europe, and it is assumed that the rest of the world is the same (Sullivan et al 2007). But the 1% is an average, and as such covers a range of rates (appendix 1A).

¹¹ Palau comprises 340 islands covering 188 square miles (Sullivan et al 2007).



(Drawn using MapCreator2)

Figure 1.5 - Position of Palau (and capital Koror).

For example, among the island nations of Micronesia in the Pacific Ocean, the average prevalence of schizophrenia is 0.54% (1 in 200). But this varies between 0.4% in the Marshall Islands and 1.7% in Palau (Sullivan et al 2007).

The variability in rates of schizophrenia around the world is important in relation to the nature-nurture debate about the cause of the illness. A genetic basis to schizophrenia (nature) would predict a similar universal prevalence rate, whereas environmental causes (nurture) expect variations between places.

Palau has been of interest because of the higher prevalence of schizophrenia. Myles-Worsley et al (1999) estimated a 2% lifetime risk (ie: probability of an a surviving adult developing the condition) (1 in 50), with a gender imbalance of over twice as common among men than

women (2.8% vs 1.2%)¹². Genetic studies have been carried out.

From the nurture side, the higher rate of the illness was explained by widespread use of exotic drugs in Palau (eg: Hezel and Wylie 1992).

Sullivan et al (2007) collected data in Palau between 1995 and 1998 with seventy inhabitants (49 men and 21 women) DSM-IV diagnosed with schizophrenia or schizoaffective disorder at the Belau National Hospital¹³.

The symptoms of schizophrenia were assessed using the Positive and Negative Syndrome Scale (PANSS) (Kay and Sevy 1990). After a detailed interview, the interviewer rates the interviewee on thirty symptoms (from 1 to 7 in severity) divided into negative and positive symptoms, and general psychopathology (table 1.3). The PANSS has comparison ("normative") scores based on a sample of 240 medicated inpatients with schizophrenia in New York in the 1980-90s.

Positive Symptoms

- P1. Delusions
- P2. Conceptual disorganization
- P3. Hallucinatory behaviour
- P4. Excitement
- P5. Grandiosity
- P6. Suspiciousness/persecution
- P7. Hostility

Negative Symptoms

- N1. Blunted affect
- N2. Emotional withdrawal
- N3. Poor rapport
- N4. Passive/apathetic social withdrawal
- N5. Difficulty in abstract thinking
- N6. Lack of spontaneity and flow of conversation
- N7. Stereotyped thinking

General Psychopathology

- G1. Somatic concerns
- G2. Anxiety
- G3. Guilt feelings
- G4. Tension
- G5. Mannerism and posturing
- G6. Depression
- G7. Motor retardation
- G8. Uncooperativeness
- G9. Unusual thought content

¹² Hammond et al (1983) reported four times greater for men.

¹³ This was 50% of all sufferers in the country at the time (Myles-Worsley 2007).

- G10. Disorientation
- G11. Poor attention
- G12. Lack of judgment and insight
- G13. Disturbance of volition
- G14. Poor impulse control
- G15. Preoccupation
- G16. Active social avoidance

(Source: Sullivan et al 2007 table 3 p194)

Table 1.3 - The PANSS.

The scores of the Palau sample on the PANSS were similar to the "normative" sample, which confirmed that "the symptomatic expression of schizophrenia in Palau is broadly comparable to its expression in other settings" (Sullivan et al 2007 p196). This finding countered any argument that the differences in prevalence between countries was due to differences in diagnosis and categorisation of schizophrenia by using the standard instrument of measurement in the PANSS.

Finding a simple genetic basis to schizophrenia is proving to be difficult ¹⁴, but there has been interest in endophenotype markers. These are measurable behaviours that are evidence of underlying problems which are heritable. One such marker that has been found among many sufferers of schizophrenia is smooth-pursuit dysfunction (SPD) (Calkins and Iacono 2000). When visually tracking a moving object, the usual smooth eye movements are replaced by short, jerky movements.

Similar to other populations of schizophrenics, Sullivan et al (2007) found that about 50% of their Palau sample showed SPD. The researchers concluded from this finding that "schizophrenia in Palau has a similar biophysiological basis to schizophrenia in other populations" (p197).

Sullivan et al (2007) questioned their sample about substance use, and the data did not support this behaviour as causing the schizophrenia. In other words, substance use was no different among individuals with schizophrenia than the general population in Palau. For example, about three-quarters of both groups chewed betel nuts, and among the individuals with schizophrenia regular chewers had lower PANSS scores than non-chewers. Along with other substance use, Sullivan et al felt that individuals with schizophrenia were self-medicating (ie:

¹⁴ Surrallés (2007) was critical of "the pseudo-scientific argument that a gene (which is always on the verge of being discovered) can explain complex phenomena such as schizophrenia, criminality, or patriotism" (p207).

illness appeared before substance use).

If schizophrenia in Palau appears to be similar to the illness elsewhere in the world, Sullivan et al (2007) highlighted cultural differences to explain the gender imbalance in prevalence. Traditional gender role expectations (concerning social relations and behavioural obligations) and the challenge in recent years of modernisation has been especially stressful for young men. This time of stress overlaps with the vulnerability period for the development of schizophrenia. Allen (1997) called such stressful in small-scale societies, "schizophrenogenic". "Having made this case, our intention has not been to claim that schizophrenia in Palau is 'caused' by the differential treatment of young Palauan men and women. Instead, it has been to engage with the manifold factors that could potentially affect the expression of schizophrenia in this setting..." (Sullivan et al 2007 p202).

1.5.1. Comments on Sullivan et al (2007)

1. Barrett (2007) was critical of the use of Kay and Sevy's (1990) New Yorkers as the "normative" sample. "We find here a tendency to render the West the normative frame of reference for the rest of the world, as if it were the 'white man's burden' to serve as a sort of psychometric benchmark for all the 'new-caught sullen peoples' of the earth..." (p203). Sullivan et al defended themselves by pointing out the use of normative in quotation marks to avoid such assumptions.

2. Harland (2007) argued that Sullivan et al ignored the stress of migration. Of the sample, half had been migrants, and some individuals had their onset of schizophrenia while abroad.

3. Hezel (2007) asked where was the information about the severity of symptoms among men and women because if women had milder symptoms, they may go undetected. In such a case, there may not be a gender imbalance in schizophrenia, only in detection.

4. Myles-Worsley (2007) felt that the sample may not have been representative of schizophrenia sufferers in Palau. The sample regularly received anti-psychotic medication, whereas Myles-Worsley et al's (1999) study of 160 Palauan individuals with schizophrenia found that many had never had medication, or were given it for a limited period

only. Also Sullivan et al's sample did not include individuals in remission.

5. Sass (2007) accused Sullivan et al of being "more struck by similarities than differences in the expression of schizophrenia in Palau and other settings" (p207). For example, the mean PANSS scores of the Palau sample were lower than the "normative" New York sample, though not statistically significant. "It is clear, in any case, that (as Sullivan et al acknowledge but perhaps insufficiently emphasise) it would be imprudent to conclude from their PANSS findings that there is an absolute uniformity of symptomatic expression between Palau and New York" (p207).

1.6. APPENDIX 1A - MEASURING THE AMOUNT OF SCHIZOPHRENIA

There are a number of problems with measuring the prevalence (how many) and incidence (new cases) of schizophrenia which limit the accuracy of estimates and make comparison between studies difficult.

1. Schizophrenia is diagnosed based on self-reports of subjective experience (Jablensky 1995).

2. Schizophrenia is relatively rare and large-scale population-based studies are costly (Jablensky 1995).

3. The means of diagnosing schizophrenia used. For example, the US Epidemiologic Catchment Area (ECA) study (Keith et al 1991) involved 18 572 individuals and diagnosis by the Diagnostic Interview Schedule (DIS). This study calculated the following rates of schizophrenia:

- Point prevalence (at this moment): 7 per 1000 (≥ 18 years old).
- One-year prevalence (in last 12 months): 10.
- Lifetime prevalence (ever): 10-19.

Using DIS, Chen et al (1993) found a lifetime prevalence of 1.2-1.3 per 1000 18-64 year-olds in Hong Kong.

Also in the USA, but using the Present State Examination for diagnosis, VonKorff et al (1985) reported 4.6 (point prevalence) and 6.4 (lifetime prevalence) for ≥ 18 years old.

The National Co-morbidity Survey in the USA

interviewed 8098 individuals with the Composite International Diagnostic Interview (CIDI) (Kessler et al 1994). This study reported 5 per 10000 for 1-year "non-affective psychosis" among 15-54 year-olds. This shows two more problems - defining which group to study, and which age group.

4. Incidence rates vary from 0.9 to 2.0 per 1000 (first admission to psychiatric hospital) depending on different factors (Jablensky 1995).

- Time period used (eg: two years).
- Variations in definition of "first admission" or "first contact" (with psychiatric services).
- Sometimes there is a "hesitation" to diagnose schizophrenia on "first contact".
- Changes in treatment such that first admission to hospital is later (ie: after treatment failure as outpatient).

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2. HISTORICAL CASE STUDIES

- 2.1. Introduction
- 2.2. King George III
- 2.3. John Nash
- 2.4. References

2.1. INTRODUCTION

This method is the bedrock of much of clinical psychology, and is concerned with collecting a large amount of information about the subject of the study, who may be an individual or a specific group of individuals (most often currently alive, but also historical case studies can be illuminating) (table 2.1).

ADVANTAGES

- Builds up detailed picture.
- Not artificial.
- Source of future hypotheses.
- Useful for treating individual problems.
- Helps to discover how past influences present.
- Studying unusual may help with understanding usual.
- Outstanding cases can be studied.
- Insight can be gained from one example.
- Tentative support for theory or challenge orthodox view.
- Documents rare cases.

DISADVANTAGES

- Not possible to generalise findings.
- Depends on accuracy of memory of participants when interviewed.
- Past events sometimes explained based on current state of mind.
- Cause and effect not possible to establish.
- Criteria for inclusion of information is subjective.
- Situation and time-bound.
- Poor replicability.
- Biased observer risk.
- Unusual and rare cases not necessarily helpful in understanding "normal" cases.
- Outstanding cases probably very different to average.

Table 2.1 - Advantages and disadvantages of the case study method.

2.2. KING GEORGE III

King George III reigned in England from 1760 to 1820. During that period, he had five major episodes of

"mental incapacity" ¹⁵. For example, he was described by a royal physician in 1788 as suffering from "an intire alienation of mind", while the Queen told a friend that the King's eyes: "she could compare to nothing but black-current jelly, the veins in his face were swelled, the sound of his voice dreadful; he often spoke till he was exhausted, and the moment he could recover his breath began again, while the foam ran out of his mouth" (quoted in Peters and Wilkinson 2010). There has been interest in understanding what this was and what caused it.

One suggestion is variegate porphyria ¹⁶ (MacAlpine et al 1968) ¹⁷. This is a malfunction in the removal of "toxins" from cells in the body. Symptoms include abdominal pain, port-wine-coloured urine, and paralysis in the arms and legs, all reported in George III's medical records. There would also be mood swings and impaired judgment (Dayan and Dayan 2011).

It is an inherited condition ¹⁸, and attacks of "mental incapacity" are triggered by exposure to toxic metals. Cox et al (2005) reported high levels of mercury, lead, and arsenic in an elemental analysis of George III's hair.

Retrospective diagnosis is not an exact science, and other researchers have diagnosed George III's illness differently - eg: manic-depressive psychosis (Peters and Beveridge 2010). Sufferers of this condition can show physical symptoms and cognitive impairment.

However, Peters and Wilkinson (2010) questioned the paralysis in arms and legs as contemporaries reported him "able to manipulate riding-reins, feed himself, play games and music, and perform feats of draughtsmanship". Furthermore, Peters and Wilkinson argued: "The symptoms manifested by George III, as detailed in the surviving historical evidence, do not provide a sound basis for a diagnosis of porphyria. None of the observations of discoloured urine survives critical scrutiny, and they are themselves numerically outweighed by observations of pale urine during attacks" (p14).

¹⁵ Four or five episodes - it is debated (Peters and Beveridge 2010).

¹⁶ Or "toxic confusional state".

¹⁷ Peters and Wilkinson (2010) were highly critical of porphyria as the illness: "It has therefore become commonplace for historians to portray the porphyria theory as more or less established fact... Yet it can be demonstrated that the porphyria diagnosis was based on weak foundations, bolstered by indiscriminate aggregation of symptoms and suppression of contrary indicators. It appears very unlikely that the King was suffering from porphyria and therefore the causes of his 'madness' remain an unsolved mystery" (p3).

¹⁸ MacAlpine et al (1968) found porphyria among many members of thirteen generations of George III's descendants.

2.3. JOHN NASH

Capps (2003) described the case of US mathematician, John Nash (born 1928), who was diagnosed with paranoid schizophrenia after a "mental breakdown" in early 1959.

He was a solitary child whose "best friends were books", and who was interested in science. As a teenager, in 1944, he and two friends built a home-made bomb which exploded killing one of them. Capps wondered how much this traumatic event at age 15 contributed to his later psychological problems.

Subsequently, Nash went to Princeton University in 1948, and later ended up as a professor at MIT. There was a second "trauma" in 1954 when he was arrested for indecent exposure.

Nasar (1998), who wrote his biography, described the mental breakdown as occurring between 31st December 1958 and the end of February 1959. During this time, Nash's behaviour became more bizarre including telling people he was the Emperor of Antarctica. He also argued that a picture of the Pope on the cover of "Life" magazine was, in fact, of him: "Nash explained that his photograph had been disguised to make it look as if it were Pope John the Twenty-third. Because John was not the Pope's given name (while Nash's was) and because 23 was Nash's favourite prime number, Nash was convinced that the editors at 'Life' magazine had played a trick on their readers" (Capps 2003 p366). He wrote letters to many ambassadors about forming a world government, and accused departmental staff of looking through his waste paper basket in order to steal his theories. He started noticing men with red ties, which he thought was a signal. Because of these behaviours and others, his wife had him involuntarily committed to a psychiatric hospital in April 1959.

There are different theories about the reasons for his breakdown. His wife believed that it was her becoming pregnant for the first time. Colleagues in the mathematics department at MIT suggested that it was his relationship with another male teacher. Nash's homosexuality is a contested issue, which his wife "categorically rejected". Capps (2003), taking a psychodynamic view, felt that the struggles with this (in a society that was strongly anti-homosexual) and his heterosexual relationships were involved in producing "acute identity confusion". "In five short years, between the ages of twenty-four and twenty-nine, Nash became emotionally involved with at least three other men. He acquired and then abandoned a secret mistress who bore his child. And he courted – or rather was courted by – a woman who became his wife" (Nasar 1998 p167 quoted in Capps 2003 p373).

The concept of identity confusion comes from the work of Erik Erikson (1959). Common life crises lead to the ego being unable to establish an identity: "a state of acute identity confusion usually becomes manifest at a time when the young individual finds himself exposed to a combination of experiences which demand his simultaneous commitment to physical intimacy (not by any means always overtly sexual), to decisive occupational choice, to energetic competition, and to psychosocial self-definition" (Erikson 1959 p133 quoted in Capps 2003 p377). Erikson described a series of conflicts or crises that have to be resolved at different stages of life. One of those is "sexual identity vs bisexual diffusion" (or "bisexual confusion; Erikson 1968). This confusion then produces a "regressive pull" (in psychodynamic terms) towards earlier life stages (eg: Pope photograph delusion is a manifestation of issues related to his father and his fatherhood as "Pope" means "papa") (Capps 2003).

Capps (2004) explored Nash's delusions when he was in his 30s. After the initial hospitalisation in 1959, and subsequent release, Nash had a period of further hospitalisations and releases over the 1960s. His delusions included saying: "I am the left foot of God on earth", and referring to himself in the third person as Johann von Nassau. As well as other delusions, he was hearing voices.

Capps (2004) interpreted the content of the delusions using Freud's (1911/1976) idea that delusion-formation is "a process of reconstruction". In other words, the delusions helped Nash to "work through" issues, primarily his "bisexual confusion". It must be remembered that within psychodynamic theory, conflicts and issues are working out in the unconscious mind, and only hints and snippets of the "truth" get through to the conscious mind because the "truth" is so disturbing. Protection of the conscious mind with ego defence mechanisms is key, and one extreme way is through delusions.

For example, Nash complained of persecution by another mathematician, Jack Bricker, who it is believed Nash was in love with. Put simply, if Nash had homosexual desires which he could not (consciously) cope with, then "techniques" were needed to hide these from the conscious mind. So, "I (a man) love him (a man)" becomes "transformed" (for the conscious mind) into "I do not love him - I hate him". Furthermore, this feeling is projected into another person: "He hates me". Thus, the delusions of persecution. "Nash's illness, then, was not 'caused' by his homosexual desires, but was precipitated when his efforts to repress them miscarried" (Capps 2004 p217).

Capps (2004) described a number of defence

mechanisms and processes like this. However, he also accepts other factors in Nash's schizophrenia, like genetics because his son also suffered from paranoid schizophrenia.

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3. SEASONAL AFFECTIVE DISORDER AND LIGHT THERAPY

Sufferers of seasonal affective disorder (SAD) experience bouts of depression and associated symptoms, like carbohydrate craving, usually during the winter when daylight is reduced. Artificial bright-light therapy was found to reduce the symptoms of SAD (Rosenthal et al 1984). Controlled trials are needed to establish the exact effect of light therapy (eg: when is exposure more beneficial - morning or evening?).

Terman et al (1998) compared exposure to light therapy in the morning and evening using a crossover design and a parallel groups design to give six conditions. Each group undertook two 10-14-day periods. In the crossover design, participants used the light box (10 000 lux for 30 minutes per day) in the morning in the first period, and then in the evening in the second period (M-E), and another group did the opposite (E-M). The parallel groups experienced light at the same time of the day in both periods (M-M and E-E). Two control groups did not use light, but two different levels of negative air ionisation (table 3.1).

	Crossover 1	Crossover 2	Parallel 1	Parallel 2	Control 1	Control 2
1st period	M	E	M	E	Low density negative air ionisation	High density negative air ionisation
2nd period	E	M	M	E	Low density negative air ionisation	High density negative air ionisation

(M = morning exposure; E = evening exposure to light)

Table 3.1 - Six conditions in the controlled trial by Terman et al (1998).

Mean depression ratings declined between baseline and the end of the study in all six groups, but morning light was more effective than evening light in all conditions (overall 54.3% vs 33.3% remission rate).

Negative air ionisation is used as a placebo because a placebo light therapy is difficult (ie: no light). But negative air ionisation has been reported to benefit mood, and so it is not a "true" placebo.

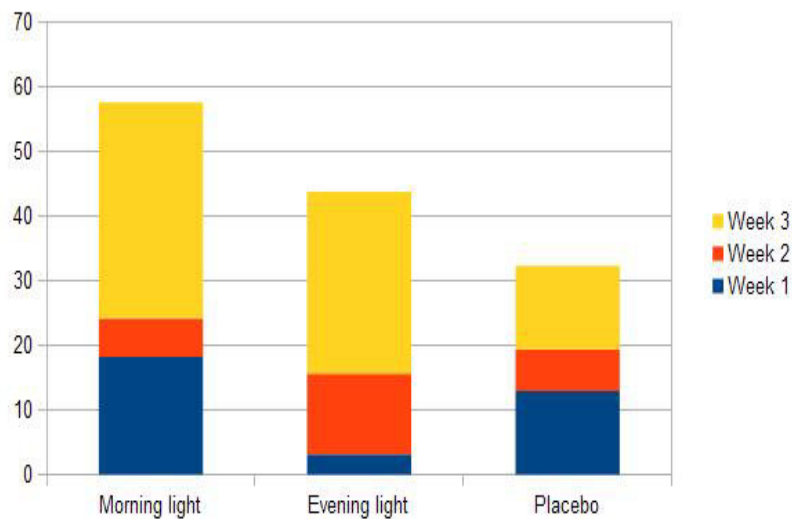
Some studies have tried using a light box that does not emit light and hums when turned "on" while telling

the participants that infra-red light is being emitted (Eastman et al 1998).

Eastman et al (1998) used sham negative air ionisation in their controlled trial. However, the expectation of improvement was created by telling the participants that they were being exposed to such air as happens naturally in the summer.

In this study, exposure to 6000-lux light lasted for 1.5 hours per day over four weeks in the evening or the morning. This was a parallel groups design with three conditions with the placebo taking place in the morning.

Participants benefited from light exposure (particularly morning) more than the placebo, but this was only significant after 3 weeks of exposure (figure 3.1).

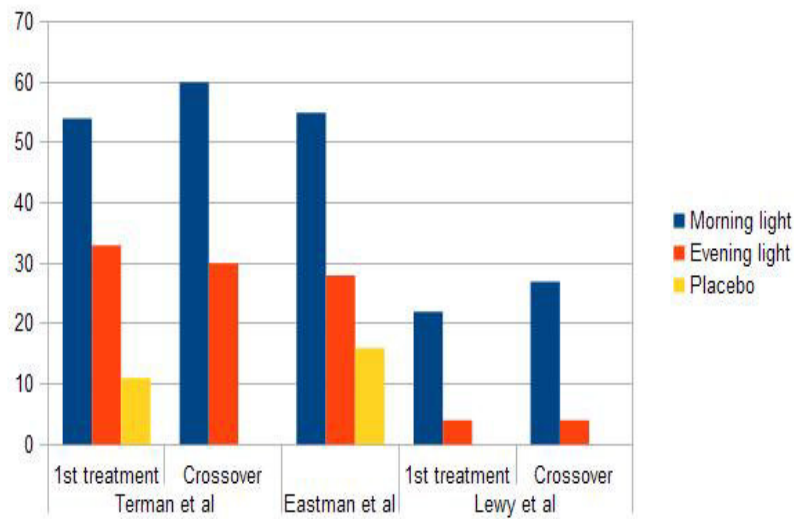


(Data from Eastman et al 1998 table 3 p887)

Figure 3.1 - Cumulative percentage of those showing remission in Eastman et al (1998) study.

Lewy et al (1998) used 2500 lux lights for two hours per day over two months of morning or evening followed by two weeks of the opposite (crossover design). Morning light was more effective.

Wirz-Justice (1998) recalculated the remission rate of the three studies to make them comparable (figure 3.2). Table 3.2. compares the three studies in terms of methodology.



(Remission = improvement of 50% or more in the score on the Structured Interview Guide for the Hamilton Depression Rating Scale - Seasonal Affective Disorder Version (SIGH-SAD) from baseline and post-treatment score of 8 or less)

(Data from Wirz-Justice 1998 table p862)

Figure 3.2 - Remission rates (%) of SAD in three studies.

	TERMAN ET AL	EASTMEN ET AL	LEWY ET AL
Design	Crossover & parallel groups	Parallel groups	Crossover & parallel groups
Light exposure	10 000 lux for 30 mins per day for 10-14 days	6000 lux for 1.5 hrs per day for 4 weeks	2500 lux for 2 hrs per day for 2 weeks
Control group	2 groups receiving negative air ionisation	sham negative air ionisation	individuals not with SAD given light therapy
Sample details	volunteers (18-65 years old); New York State area; 145 completed (158 started)	96 volunteers; Illinois area	volunteers; Oregon area; 100 completed (108 started)
Length of study	6 years	6 years	4 years

Table 3.2 - Comparison of methodology between three studies of light therapy and SAD.

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4. ABORTION AND MENTAL HEALTH: METHODOLOGICAL WEAKNESSES AND PERSONAL ATTACKS ON PRISCILLA COLEMAN

The association between an unwanted pregnancy and subsequent mental health problems can occur when giving birth (ie: keeping) to such a baby or after an abortion (also depending whether it is legal or illegal).

In a meta-analysis, Coleman (2011) found that abortion led to greater mental health problems than giving birth. This finding contradicted a systematic narrative review by Charles et al (2008), for example, of many of the same studies which found no such relationship (ie: the mental health risks of a single first-trimester abortion of an unplanned pregnancy were the same as giving birth). But the quality of the study was important, and poorer quality studies were more likely to find abortion as leading to mental health risks (Kendall et al 2012).

Coleman (2011) was heavily criticised in correspondence in the "British Journal of Psychiatry" (table 4.1).

1. Howard et al (2012) - "This quantitative synthesis and meta-analysis did not follow the robust methodologies now generally accepted for systematic reviews. There is no detail of the search strategy including search terms; the strategy is not comprehensive (only two databases included); other strategies to search the literature, including citation tracking, hand searching and contacting authors and experts in the field to try to minimise publication bias, were not carried out; and there was no assessment or rating of the quality of included studies, so that only those of at least reasonable quality are included in the meta-analysis" (p74).

2. Abel et al (2012) - Coleman (2011) appeared to contradict herself; eg: "nearly 10% of the incidence of mental health problems was shown to be directly attributable to abortion" vs "Although an answer to the causal question is not readily discerned based on the data available, as more prospective studies with numerous controls are being published, indirect evidence for a causal connection is beginning to emerge".

3. Littell and Coyne (2012) - Many methodological weaknesses including no list of excluded studies provided; the quality of included studies was not detailed; and the likelihood of publication bias was not assessed.

4. Polis et al (2012) - Failure to assess underlying validity of included studies, to examine their statistical heterogeneity, and "she illogically combines estimates for distinct outcomes".

5. Goldacre and Lee (2012) - Accused Priscilla Coleman of being an anti-abortion campaigner, and thereby the "Declaration of Interest: None" statement was untrue.

6. Robinson et al (2012) - Coleman failed to distinguish between an association and a causal relationship, and to control for pre-

existing mental health problems before pregnancy.

7. Lagro-Janssen et al (2012) - In a Dutch study it was found that women who had an induced abortion had more social problems than controls.

8. Tyrer and Waheed (2012) - In an editors' response, it was pointed out that Coleman submitted her manuscript (in October 2010) before Munk-Olsen et al (2011) had been published.

9. Coleman (2012) - Replying to the criticisms, Coleman defended herself: "My experience attempting to locate unpublished data/studies on abortion and mental health has been very disheartening over the past 15 years, with virtually all requests ignored. I suspect that reluctance to share unpublished data is an attempt to keep results that challenge contemporary views on abortion and indicate significant increased risks for adverse psychological effects out of the public domain. In contrast, I believe energy is likely invested in seeing to it that non-significant findings, suggesting abortion carries no increased psychological risks, find their way into the journals. If there is any topic wherein many editors, researchers and professional organisations are highly motivated to publish non-significant effects, it is this one, rendering publication bias less common than in other areas. Support for this notion can be found in the American Psychological Association's (APA's) 42-year history of abortion advocacy" (p79).

She said this about being an anti-abortion campaigner: "I do not hold membership in any political organisations and my work has never been funded by any pro-life group. My expertise tends to be called upon by the pro-life community and unfortunately I am never asked to present my research or perspective on the literature to groups committed to a pro-choice political position" (p80).

Finally, Coleman is critical of the Munk-Olsen et al (2011), which many of her critics supported: "There are no controls for pregnancy intendedness, pressure to abort, marital status, income, education, exposure to violence and other traumas, etc" (p80).

Table 4.1 - Debate around Coleman (2011) meta-analysis.

Many of the studies in this area have methodological weaknesses like (Kendall et al 2012):

i) Varied and differing (heterogeneity of) mental health outcomes used (eg: depression, self-harm, anxiety, substance misuse).

ii) Varied measures of mental health outcomes (eg: self-report; psychiatric services contact).

iii) Limited control of confounding variables, particularly mental health problems pre-pregnancy/abortion.

iv) Groups that were not well matched (eg: unwanted pregnancy abortion versus birth including wanted/unwanted).

v) Different countries with different formal and

informal views on abortion (eg: in the UK the legal grounds for abortion are to mitigate physical or mental harm of the pregnancy continuing, while in the USA abortion is available "on-demand" but anti-abortion pressure groups are vocal).

In a well-designed longitudinal study in Denmark, Munk-Olsen et al (2011) found that abortion and mental health risks were a correlation (ie: a two-way relationship) rather than causation in one direction. A group of women who had an abortion and a matched group who gave birth were assessed for psychiatric contact at nine months prior to the abortion or birth, and in the twelve months afterwards.

Women who had an abortion were more likely to have mental health problems at nine months prior than women who gave birth, and the rates of psychiatric contact were no greater in the following year for the former group. This showed that mental health problems could cause an abortion and/or an abortion could cause mental health problems (ie: correlation). Women who gave birth were more likely to have mental health problems after birth than nine months before.

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5. EATING DISORDERS AND NORMAL EATING BEHAVIOUR

- 5.1. Measuring eating disorders
 - 5.1.1. Operationalisation problems
 - 5.1.2. Stroop Test
- 5.2. Symptom trajectory
- 5.3. Study of genetics
- 5.4. Binge eating disorder
- 5.5. Normal dieting
- 5.6. A treatment option
- 5.7. Helping carers
- 5.8. References

5.1. MEASURING EATING DISORDERS

Eating disorders can be measured in different ways:

- i) Structured clinician-based interview - Time-consuming and requires training for use.
- ii) Self-rating scales.
- iii) Objective measures - eg: Stroop Test.
- iv) Behavioural observation.
- v) Self-rated structured diaries (eg: hour by hour, day by day) - Depends on reliability and willingness of participants.

Fichter and Quadflieg (2000) compared 377 individuals with eating disorders in Bavaria, Germany, using the Structured Interview for Anorexic and Bulimic Syndromes (SIAB-EX) (completed by a clinician) and SIAB-S (self-rating version). This questionnaire has 87 items and six sub-scales.

The two versions of the questionnaire correlated well: $r = 0.80$ (total score) (Pearson's; $p < 0.01$; range 0.56 - 0.83 for sub-scales) (figure 5.1). There were some differences, however, including self-rating lower on items about bingeing and inappropriate compensatory behaviour, but higher on general psychopathology than expert version.

Sub-scale	Expert-rated	Self-rated
I. Body image and slimness ideal	1.2	1.4
II. General psychopathology	1.1	1.5
III. Sexuality and social integration	2.1	1.9
IV. Bulimic symptoms	1.9	2.0
V. Inappropriate compensatory behaviours to counteract weight gain	0.4	0.4
VI. Atypical binges	0.8	1.4
Total score	1.2	1.4

(Source: Fichter and Quadflieg 2000 table 1 pp178-180)

Figure 5.1 - Mean scores (out of 4) for current behaviour on sub-scales on SIAB.

5.1.1. Operationalisation Problems

Diagnostic systems like DSM-IV list the symptoms of each disorder, but the operationalisation of these symptoms (ie: measurement) can lead to variability in diagnosis. Loeb et al (2011) used the example of diagnosis of eating disorders in adolescents.

One hundred and forty-four 12-18 year-olds already diagnosed with anorexia and 80 with bulimia in the USA were assessed using different operationalised criteria for the symptoms. For example, criterion A in DSM-IV for diagnosing anorexia is a "refusal to maintain body weight at or above a minimally normal weight for age and height" (eg: less than 85% of expected weight). Four different measures of this criterion were applied:

i) Less than 85% of "ideal weight" based on median for age, height, and gender in 1973 (older) National Center for Health Statistics (NCHS) norms ¹⁹.

ii) Less than 85% of "ideal weight" based on median body mass index (BMI)-for-age in 2000 (latest) NCHS norms.

iii) BMI =17.5 (ICD-10 criterion) ²⁰.

¹⁹ Madden et al (2009) reported that among a group of 5-13 year-olds in Australia with early onset eating disorders, 61% had life-threatening complications of malnutrition, but only 51% weighed less than "85% of ideal body weight according to height".

²⁰ A cut-off point based on BMI can vary with individual age and the time period studied. For example, in mid-2000s in the USA, about 5% of 18-24 year-old women would be classed as underweight using a BMI <18.5, 3% of 25-44 year-olds and 1.7% of 45-65 year-olds. Using the same criteria, in 1960-62 4% of US adults were underweight, but only 1.8% in 2003-6 (Hebebrand and Bulik 2011).

iv) BMI-for-age of lowest 5% of population.

In order of these criteria, of 144 anorexia sufferers, the following number of them would be diagnosed - 53% (criterion i), 43%, 66%, and 72% respectively.

Criterion B for diagnosing anorexia in DSM-IV is "intense fear of gaining weight or becoming fat, even though underweight". Loeb et al used three ways of measuring this:

a) A score of four or more on the "fear of weight gain" item of the Eating Disorder Examination (EDE) (12th ed) (Fairburn and Cooper 1993) ²¹. This is a questionnaire used as part of a semi-structured interview which the clinician completes about the severity of eating disorder symptoms in the last 1-6 months. Each item is scored 0-6.

b) A score of one or more on "fear of weight gain" item.

c) Fear of weight as determined by clinical interview which included self-reports, parent-reports, and direct observation by clinician.

The second way of measuring the criterion was most relaxed and 77% of patients were diagnosed with anorexia compared to 38% with an EDE score of four or above. All 100% were diagnosed using the clinical interview method.

The decision over the cut-off point on items of the EDE shows how definitions of normality/abnormality can be fluid. Is "normality" a score of 0, say, or a score of less than three (figure 5.2)? How normal is it to worry about weight gain when already underweight?

²¹ Over the past four weeks have you been afraid that you might gain weight?:

0 - No definite fear of weight gain.

1 - Definite fear of weight gain on 1-5 days.

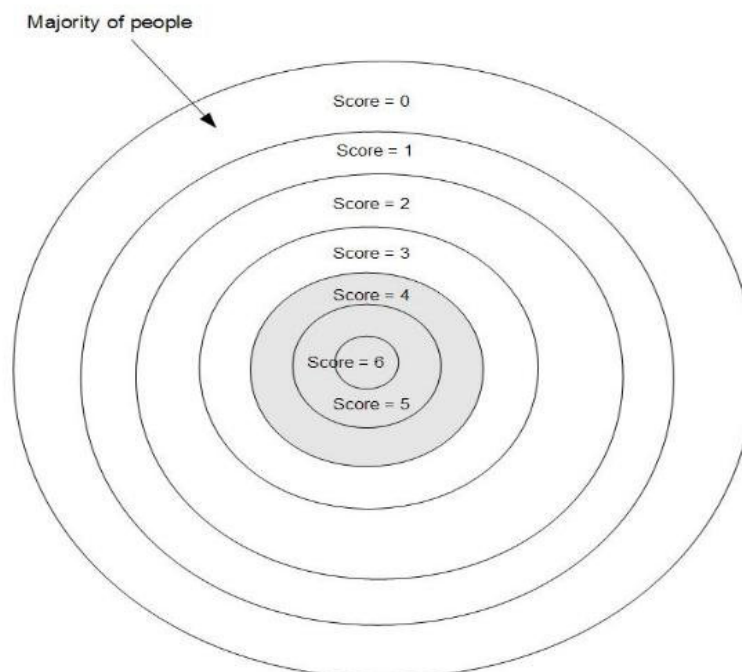
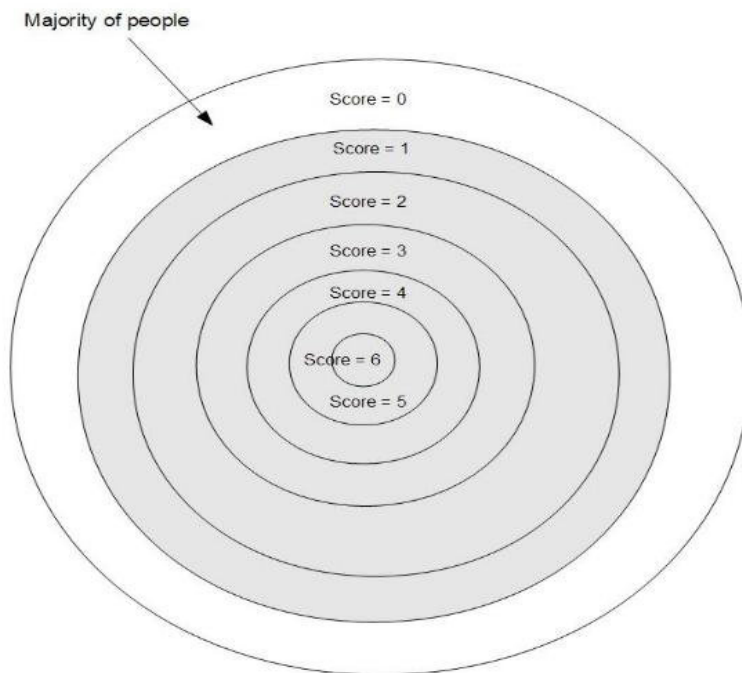
2 - Definite fear of weight gain on 6-12 days.

3 - Definite fear of weight gain on 13-15 days.

4 - Definite fear of weight gain on 16-22 days.

5 - Definite fear of weight gain on 23-27 days.

6 - Definite fear of weight gain on every day.



(Grey = "abnormal")

Figure 5.2 - Number of days in the last month worrying about weight gain and defining normality.

The cut-off point on a scale is important because of the issue of false positives (rated as ill when not) and

false negatives (rated as not ill when ill). Take this general example, based on Paulos (2012):

An illness has a prevalence rate of 0.4% (1 in 250). Thus among 1 million people, 4000 will be sufferers.

Test A has true positive rate of 99.5% and false positive rate of 1%
True positive = 3980 (99.5% of 4000 sufferers)
False positive = 9960 (1% of 996 000 non-sufferers)

Total positive tests = 13 940 (9960 + 3980)
Thus true positives only 28.6% (3980 ÷ 13940)

Test B has true positive rate of 99% and false positive rate of 2%
True positive = 3960 (99% of 4000 sufferers)
False positive = 19920 (2% of 996 000 non-sufferers)

Total positive tests = 23880 (19920 + 3960)
Thus true positives only 19.9% (3960 ÷ 19920)

In the case of bulimia, episodes of binge eating is a key diagnostic criterion. Loeb et al used a number of ways of measuring this behaviour:

- i) "Objectively large binge eating episodes" at least twice a week.
- ii) Such episodes at least once a week.
- iii) Objectively and subjectively defined episodes at least twice a week.

Of the eighty adolescents with bulimia in the study, 54% were diagnosed based on criterion (i) above, 66% and 71% based on the other two respectively. "Objectively large" is a term that can be operationalised differently also. DSM-IV uses the phrase, "an amount of food that is definitely larger than most people would eat".

How a symptom (an abstract concept) is operationalised is important for the cut-off point in diagnosis.

Hebebrand and Bulik (2011) outlined a number of problems with diagnosing eating disorders.

1. Many individuals do not reach the threshold for diagnosis of anorexia and bulimia, and are thus diagnosed with eating disorders not otherwise specified (EDNOS).

2. The temporal instability of diagnosis. This is where individuals are given one diagnosis initially, and different one for the same symptoms at a later date. In the case of anorexia to bulimia, 8-54% of individuals, and 4-27% bulimia to anorexia over five years of the

illness (depending on the study) (Hebebrand and Bulik 2011).

3. Dealing with symptom remission. "In the current classification scheme, rather than receiving a descriptor such as 'anorexia nervosa, in partial remission', these individuals would receive a new diagnosis of EDNOS" (Hebebrand and Bulik 2011 p665).

4. Specific problems with sub-type diagnosis (eg: anorexia restricting sub-type vs binge/purge sub-type). There is temporal instability with these sub-types also.

5. The relationship between eating disorders and body weight. Although most people think of anorexia and bulimia when the term "eating disorders" is used, eating disorders also include obesity and overweight-related ones. Thus individuals who are underweight, overweight, or normal weight can be diagnosed with an eating disorder. Body weight is not a clear indicator of an eating disorder in the sense that auditory hallucinations, for example, are usually present in diagnosis of psychosis. If eating disorders were related to low body weight only that might be clearer.

On the other hand, underweight is not automatically a symptom of an eating disorder if it is due to lack of food (eg: famine), or leanness as a family trait. Hebebrand and Bulik (2011) observed:

Efforts to achieve health or to attain a cultural standard of beauty lead many individuals in Westernised societies to strive for thinness – some of whom are successful in achieving or maintaining underweight without having a "harmful mental dysfunction". The boundary between healthy and harmful underweight is not sharp and cannot be defined in terms of a specific weight cut-off. Yet, a definition is required in order to highlight the transition from healthy to harmful. It would seem most appropriate to describe harmful underweight as a low body weight which has led to clinically apparent symptoms of the physiological adaptation to starvation, which negatively affect critical functions such as fertility, bone density, and psychological functioning (p666).

It is proposed that the phrase "minimally normal

weight for age and height" will be used in DSM-V ²². This confirms the idea of an "expected weight" which Hebebrand and Bulik (2011) said is "in itself misleading, because there is no such thing as an expected weight (other than a statistically expected weight based on population reference data, which more appropriately should be termed average or mean weight). Instead, body weight for height is quite variable; healthy people of the same age and height have a wide range of weights. The mere implication of a single expected weight for height and age provides clinicians, patients, and their relatives with the wrong message" (p669). Furthermore, in the USA, where obesity and overweight are an issue, the Nurses' Health Study (Manson et al 1995) found that the lowest mortality was among women 15% below the US average for women of a similar age.

Hebebrand and Bulik (2011) challenged the DSM-V proposal for anorexia nervosa, and gave their own alternative criteria which included specific behaviours as part of the diagnosis process (table 5.1).

DSM-V PROPOSAL

- A. Restriction of energy intake relative to requirements leading to a markedly low body weight. Markedly low weight is defined as a weight that is less than minimally normal, or, for children and adolescents, less than that minimally expected for age and height.
- B. Intense fear of gaining weight or becoming fat, or persistent behaviour to avoid weight gain, even though at a markedly low weight.
- C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

HEBEBRAND AND BULIK (2011)

A: Underweight associated with symptoms of starvation as defined by both:

- 1. Body mass index (BMI) < 18.5 kg/m² in adults or BMI < 10th age percentile of gender matched reference population in youth < 18 years
- 2. Presence of two or more physical symptoms indicative of physiological adaptation to starvation [eg: substantial loss of fat mass, loss of muscle mass, weak muscles, fatigue, dizziness, dry skin, nails and hair, fine (lanugo) hair over body, thinning, and/or breaking of hair, hair loss, amenorrhoea, bradycardia, arrhythmias, hypotension, hypothermia, cold intolerance,

²² The terms "underweight" and "low body weight" are also confusingly proposed within the definitions (Hebebrand and Bulik 2011).

constipation, osteoporosis, bone fractures, dehydration, low blood cell counts, hypoleptinemia]

B: Presence of one or more behaviours, which likely result in, maintain, or exacerbate underweight and/or which indicate disordered eating behaviour:

- 1. Behaviour(s) indicative of insufficient energy intake [eg: avoidance of certain foods or types of foods (eg: energy dense foods), only eating small amounts of food at a time, intermittent fasting, ritualized or prolonged eating of small amounts of food, skipping of meals, early termination of meals, or induction of reduced appetite or palatability (eg: water loading, use of appetite suppressant, excessive use of condiments, or sweeteners)].
- 2. Behaviour(s) indicative of prevention or reduction of absorption of nutrients, ie: purging [eg: vomiting, ruminating, use of laxatives, enemas, or other methods
- 3. Behaviour(s) indicative of high energy expenditure (eg: motor restlessness, hyperactivity, aerobic, or isometric exercise, under-dressing in cold climate, cold showers, use of medications to elevate resting energy expenditure)
- 4. Behaviours that reduce total body water (use of diuretics, low fluid intake)
- 5. Recurrent episodes of binge eating (at least one episode of binge eating per week for a period of three months)

C: Presence of one or more of the following cognitions or subjective experiences:

- 1. Fear of gaining weight and/or becoming fat
- 2. Continued desire to lose weight or maintain underweight
- 3. Undue influence of body weight or shape on self-evaluation, disturbance in the way in which one's body weight or shape is experienced or evaluated, and/or preoccupation with shape and/or weight
- 4. Undue influence of food intake on self-evaluation or mood (eg: the patient derives reinforcement from the ability to control food intake; minor fluctuations in weight strongly influence mood)
- 5. Persistent preoccupation with food and/or nutrition (eg: reports frequently or constantly thinking about food or weight)
- 6. Lack of concern about the physiological and psychological consequences of underweight (eg: patient labels her or his state as normal or safe)
- 7. Irritability, social withdrawal, depressed mood
- 8. Reduced or absence of libido

D: The underweight and the associated behaviours cannot better be explained by other general medical conditions (eg: infectious, neoplastic or endocrine disorders, malabsorption), psychiatric disorders (eg: schizophrenia, affective disorder, obsessive compulsive disorder), or strong personal motives/aims independent of the wish to lose weight, which are comprehensible and plausible to outside observers (eg: hunger strike).

(Source: Hebebrand and Bulik 2011)

Table 5.1 - DSM-V and alternative criteria for diagnosing anorexia nervosa.

5.1.2. Stroop Test

The Stroop Test measures the reaction time to name the ink colour of a word. In the original version, the word "RED" written in blue ink (mismatch) takes longer to name the ink colour than "RED" in red ink (match) because of the distraction of the word meaning. Adapting this idea to use with eating disordered individuals, words about food and weight will take longer to name the ink colour than non-food words²³. This is because these individuals are extremely preoccupied with thoughts of food, eating, weight, and shape, and such words distract them and increase reaction time to name the ink colour (Channon et al 1988).

The typical Stroop Test design for food words presents words in these categories (figure 5.3) (Channon et al 1988):

- Control colour Stroop - eg: letter "O" written in different colours.
- Conflicting colour Stroop - eg: "RED" in blue ink.
- Food Stroop - eg: food, dinner, baker.
- Control food Stroop - eg: good, winner, maker.
- Body size Stroop - eg: heavy, fat, hips.
- Control body size Stroop - eg: lips, that, sky.

Control colour Stroop

RED

BLUE

Conflicting colour Stroop

RED

BLUE

Food Stroop

DINNER

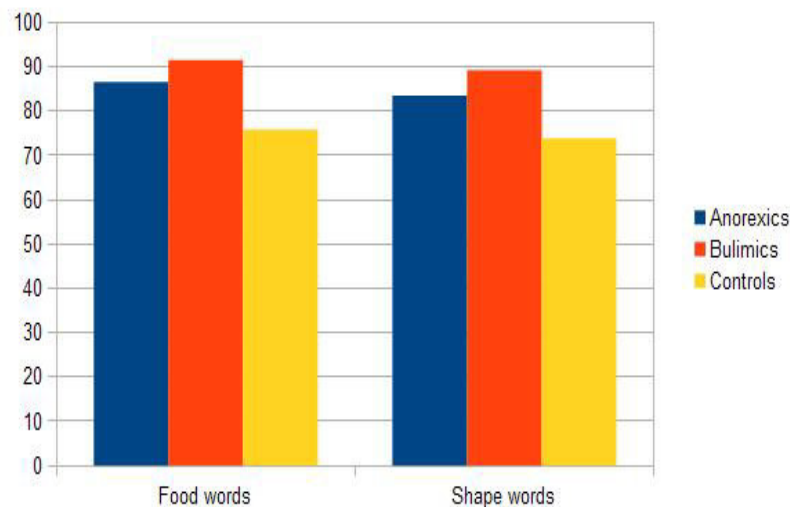
BAKER

Figure 5.3 - Example of Stroop Test.

²³ Klein (1964) extended the Stroop Test beyond colour naming of words only. It has been used with other groups of patients - eg: Watts et al (1986) found that colour naming of spider-relevant words was slower among spider phobics than non-phobics.

Ben-Tovim et al (1989) found a longer reaction time among seventeen anorexics and 19 bulimics at a Weight Disorder Unit in South Australia as compared to 38 female staff and students acting as controls (figure 5.4).

Ben-Tovim and Walker (1991) compared the reaction times of adolescent females with anorexia nervosa and with bulimia nervosa with controls in local secondary schools in South Australia. The mean reaction time to name the ink colour of food words and shape words was significantly longer for anorexics and bulimics than the controls.



(Data from Ben-Tovim et al 1989 table 2 p684)

Figure 5.4 - Mean reaction time (in seconds) to name ink colour for food and shape words.

5.2. SYMPTOM TRAJECTORY

Over time the symptoms of eating disorders can fluctuate, and this is interpreted in terms of remission, relapse, or crossover (eg: up to half of individuals with an initial diagnosis of anorexia are later diagnosed as bulimia) (Lavender et al 2011).

Longitudinal studies of eating disorders are relatively rare, and this would be the means to understand the progress of symptoms over time. One example is the Massachusetts General Hospital (MGH) Longitudinal Study of Anorexia and Bulimia Nervosa, which follows women who seek treatment in the Boston area of the USA between 1987 and 1991.

Lavender et al (2011) analysed the data for 246 such women, diagnosed with DSM-III-R anorexia or bulimia, and

the five-year trajectory of three symptoms - low body weight (=85% ideal weight), binge eating, and purging behaviour (eg: self-induced vomiting; use of laxatives). The women were interviewed every six months, and each symptom was coded as present or absent on a week-by-week basis.

Analysis of the data produced four trajectories for each symptom:

- Low body weight - persistent low weight (24.4% of sample), "gaining from low weight" (11.8%), fluctuating weight (7.7%), and the majority (56.1%) non-low weight.
- Binge eating - persistent (34.9%), "early decreasing binge" (ceased by year 3 of the study) (17.8%), and "late decreasing binge" (no decline until after year 2) (19.9%) with 27.4% showing non-binge eating.
- Purging - persistent (28.9%), "steady decreasing purging" (20.7%), increasing purging (14.2%), and 36.2% non-purging.

The findings showed the variety of symptom experience among eating disorders sufferers with some individuals showing stability of symptoms and others the tendency to fluctuate.

5.3. STUDY OF GENETICS

Family studies aid in the search for genetic causes to mental disorders by looking at the genetic family tree of sufferers (probands).

For example, Lilenfeld et al (1998) recruited 26 women with anorexia, 47 women with bulimia, and 44 control women with no history of eating disorders in the Pittsburgh area of the USA as their probands. Four hundred and sixty first-degree relatives (parents and siblings) of the probands were interviewed about eating disorders and other mental disorders. If eating disorders are inherited, sufferers should have more relatives who have suffered from them than the controls.

Relatives of the eating disorders sufferers had suffered from any eating disorder significantly more than relatives of the controls (table 5.2), and also from major depression and anxiety disorders.

	ANOREXIA	BULIMIA	CONTROL
Total number of relatives interviewed	93	177	190
Mother	26	47	44
Sister	20	42	56
Father	25	47	44
Brother	22	41	46
Prevalence (%) - any eating disorder	12	20	4

Table 5.2 - Number of relatives of probands and prevalence of any eating disorder.

5.4. BINGE EATING DISORDER

Binge Eating Disorder (BED) is included in the appendix of DSM-IV, and is characterised by bouts of binge eating without the accompanying compensatory behaviour to reduce weight, like induced vomiting, found in bulimia nervosa.

Fairburn et al (1998) were interested in isolating the risk factors for BED, particularly those distinct from bulimia. Fifty women with BED in Oxfordshire, England, were compared to 102 with bulimia nervosa, 102 with non-eating disorder psychiatric disorders, and 102 health controls. All women were volunteers, and aged 16-35 years old. They initially completed the Eating Disorder Examination-Questionnaire (EDE-Q) (Beglin and Fairburn 1992) and the General Health Questionnaire (GHQ) (Goldberg and Williams 1988). Subsequently, the women were interviewed about potential risk factors relating to personal vulnerabilities (eg: childhood problems), environmental triggers (eg: parental separation), and dieting (eg: teased about shape and weight).

Analysis of the risk factors showed the following patterns for the BED group:

(i) Compared to healthy controls.

a) Significantly more negative self-evaluation, parental depression, own major depression, childhood conduct problems, and deliberate self-harm (personal vulnerabilities).

b) Significantly greater parental criticism, high parental expectations, overprotectiveness, abuse and bullying, and lower affection (environmental factors).

c) Significantly more comments and teasing about shape and weight (dieting vulnerabilities).

(ii) Compared to non-eating disorder psychiatric disorders group - Significantly less parent contact, and more critical comments about shape and weight, and clinical obesity.

(iii) Compared to bulimia group - No significant differences.

The findings showed that there were common risk factors for BED, bulimia, and general psychiatric disorders, but these were distinct from non-eating disorder non-psychiatric disorder individuals (ie: general population).

Table 5.3 summarises the main strengths and weaknesses of this study.

STRENGTHS	WEAKNESSES
<p>1. All participants recruited from the community, which reduced the sampling bias of using individuals seeking treatment.</p> <p>2. Many different risk factors assessed (over fifty).</p> <p>3. Three different comparison groups to the BED sufferers.</p> <p>4. Matching of the group members based on current age, parental social class, and age of illness onset.</p> <p>5. Two stages to study - self-reported questionnaires and face-to-face interviews.</p>	<p>1. All information based on self-reports with no independent verification.</p> <p>2. Interviewers not blind to participant diagnosis.</p> <p>3. Information related to the past with risk of recall bias.</p> <p>4. Only women.</p> <p>5. Only volunteers. Eating disorder sufferers are secretive about their behaviour, so how representative were those who agreed to participate in the study?</p>

Table 5.3 - Main strengths and weaknesses of Fairburn et al (1998) study.

5.5. NORMAL DIETING

Polivy and Herman (1987) observed that the "current societal preference for a thin physique has spawned a corresponding societal preoccupation with dieting and weight loss" ²⁴, such that "dieting and its attendant diet

²⁴ "Nowadays, women are induced to strive toward a condition of ruddy-cheeked emaciation" (Polivy and Herman 1987 p635).

mentality" should be seen as "normal". This situation means that a term like "normal eating" is "no longer obvious" (p635).

It seems self-evident that dieters eat less than non-dieters, but in experiments and situations where the dieter's diet boundary becomes "inoperative" (eg: social pressures to indulge) dieters eat "considerably more" than non-dieters. Polivy and Herman (1987) felt that individuals who became "dieters" (ie: in permanent and semi-permanent diet state) show symptoms that are associated with eating disorders (eg: preoccupation with weight and dissatisfaction with body).

Nylander (1971) accounted for this observation with the "continuum hypothesis" that placed dieting and eating disorders as different points along a continuum. The starvation experienced during dieting could push the individual along the continuum towards "true" eating disorders. On the other hand, Crisp (1965), for example, viewed eating disorders and dieting as fundamentally different because of the motivations of the two sets of individuals being entirely different. The dieters are motivated by the positive goals of losing weight while eating disorders sufferers are using food to cope with emotional issues. For example, Crisp explained anorexia as an attempt to escape puberty and maturation among teenage women.

A study by Garner et al (1984) suggested that dieters and eating disordered-individuals share some elements, but not others. This research used the Eating Disorder Inventory (EDI) with anorexics, student dieters, non-dieters, and ballet students. Anorexics and dieters scored similar on weight and body shape preoccupation, but dieters did not share the perceptual disturbances of anorexics.

Herman and Polivy (1984) explained eating behaviour with the "boundary model of eating behaviour". In an "undisturbed organism" eating behaviour is controlled by physiological processes to start and stop (ie: hunger and satiety). Inbetween these two states is a "zone of indifference" where eating is controlled by social, environmental or cognitive factors. Dieters have another boundary ("the diet boundary") which is self-imposed between hunger and satiety. Normally, this boundary leads to eating less as in a diet, but if the boundary is violated, a form of bingeing occurs because the dieter's satiety boundary has moved further away. In other words, the imposition of the diet boundary means that the dieter loses the ability to detect the "normal" signals of satiety and thus keeps eating.

Under the boundary model, anorexics have a very strict diet boundary, but very strong cognitive factors limit any transgressions, while bulimics "choose" to

violate any self-imposed boundaries during bingeing. Obese/overeating individuals inhabit the intermediate zone, and are extra sensitive to social cues (eg: eating associated with certain situations, irrelevant of hunger or satiety, like popcorn and watching movies).

So, for Polivy and Herman (1987), dieters and eating disorders sufferers share "obsessive concerns with weight and appearance", but it is "only when these elements are combined further with the personality defects of low self-esteem, fears about interpersonal relationships and the responsibilities of maturity, and a loss of touch with internal signals of all sorts, that a societal diagnosis of abnormality is rendered" (p640).

5.6. A TREATMENT OPTION

Mitchell et al (2008) found that cognitive-behavioural therapy (CBT) delivered via telemedicine (eg: video conferencing) was as effective as face-to-face in reducing binge eating and purging among 128 bulimia sufferers. This was the "objective" side of using a new technology like telemedicine.

Ertelt et al (2011) were interested in the "subjective" side (ie: the experience of telemedicine CBT), in particular the affect on the "therapeutic alliance". Both patients and therapists completed the Working Alliance Inventory (Horvath and Greenberg 1989), which measures aspects of the experiences of therapy/counselling with 36 items.

The WAI scores of patients did not vary between face-to-face or telemedicine CBT, whereas therapists gave more positive ratings to the face-to-face version.

5.7. HELPING CARERS

Carers of people with eating disorders includes partners, relatives and friends. They usually want to help, but sometimes can unwittingly make things worse through the "rewarding" of pathological behaviour (eg: giving sufferer attention only when ill), for example. The carers may also be stressed, and have unmet needs, and problems of their own (Grover et al 2011).

Grover et al (2011) reported the development of an interactive web-based multi-media programme based on cognitive-behavioural principles to help carers of individuals with anorexia nervosa. It covers information about anorexia nervosa, self-monitoring and managing the symptoms, and the carers' own needs in the equivalent of nine "workbooks".

A small sample of UK carers showed significant improvements over the 2-9 months of using the programme

(as individuals worked at their own speed). They were less anxious and depressed, and reported more positive experiences related to caregiving (eg: improvements in relationship with person being cared for).

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6. ETHICS OF MODERN PSYCHIATRY AND TREATMENTS

- 6.1. Diagnosis
- 6.2. Treatment
- 6.3. Beware
- 6.4. Appendix 6A - Stopping drug treatment
- 6.5. Appendix 6B - Repetitive transcranial magnetic stimulation and auditory hallucinations
- 6.6. References

6.1. DIAGNOSIS

Psychiatry claims the "neutrality" of science/medicine, but "the vexed question of the value-laden nature of interpreting symptoms, which are unable to be 'measured' as in the same manner as high cholesterol or low insulin levels, continues to haunt psychiatric practice and the subsequent provision of effective evidence-based healthcare" (Bendelow 2010 p546). A value-laden diagnostic process goes hand in hand with the criticism that sees the medicalisation of social problems and the social construction of diagnostic labels in psychiatry.

The diagnostic process is faced with a number of "subjective" challenges including conceptualising the issue or problem, the diagnostic criteria to use, different interpretations of behaviour, and labelling (Bendelow 2010).

Personality disorders are a prime example of conditions that are difficult to define and to diagnose. In defining personality disorders DSM-IV uses terms which can be open to interpretation, like "deviates markedly from the expectation of the individual's culture", and "leads to distress or impairment". At one level, these phrases seem obvious, but in a world wanting "objective" diagnosis, they are problematic. One way around this problem is to have measuring instruments, like psychometric questionnaires, with a cut-off score.

Probably more than other mental disorders, personality disorders are also linked to the issue of "dangerousness" and risk assessment (especially in terms of public concern). Here is the intersection of diagnosis and the law. For example, in the UK, the category of "dangerous and severe personality disorder" (DSPD) was introduced in 2002 to cover individuals with Anti-Social Personality Disorder and Borderline Personality Disorder capable of extreme violence, but primarily it is an administrative category used to detain individuals (Bendelow 2010).

An alternative to the striving for objectivity is called "value-based medicine" (VBM) (eg: Woodbridge and Fulford 2004). This accepts that humans are subjective and value-laden, and works with it using "relationship-centred" or "patient-centred" care where the physician/psychiatrist works with the patient/sufferer.

6.2. TREATMENT

There has always been debates about the ethical issues related to treatments and therapies including whether the benefits outweigh the costs (eg: unpleasant side effects) (appendix 6A). With the development of medical technology new ethical issues will arise as in "brain-hardware interfaces".

These include brain-computer interfaces (BCI), brain-machine interfaces (BMI), and brain stimulators, all of which "enable a more or less direct contact to the human brain for exchanging electrical signals" (Clausen 2011 p495).

One particular example is deep brain stimulation (DBS) ²⁵. Electrodes are surgically placed in the brain which allow high-frequency stimulation of certain areas, like the sub-thalamic nucleus. It has been most used to treat motor impairments in end-stage Parkinson's disease where medication is ineffective or produces severe side effects (eg: Deuschl et al 2006). The major risk relates to the surgery to implant the electrodes (Clausen 2011).

In terms of ethics, DBS can also affect the individual beyond the intended target (eg: compromising decision-making; change of personality). For example, Leentjens et al (2004; quoted in Clausen 2011) reported the case of a Dutch individual with Parkinson's disease whose DBS produced severe mania. This was stopped when the stimulation was turned off, but then the motor impairments returned. While Schermer (2011; quoted in Clausen 2011) had a case of a woman with obsessive-compulsive disorder (OCD) who gained no therapeutic benefit from DBS, but wanted continued stimulation because it made her feel very happy. Should the DBS be turned off?

Clausen (2011) was not that concerned about personality change: "Even in Parkinson's disease, where the main intended outcome is ameliorating motor function, change of personality or identity cannot be an ethical criterion, as Parkinson's disease itself alters a patient's personality. A change of personality in patients severely affected by Parkinson's disease might

²⁵ A non-invasive alternative is repetitive transcranial magnetic stimulation (appendix 6B).

well be welcomed. Surely, not all personality changes are unproblematic. In a normative perspective, the crucial point is to separate unproblematic changes from problematic ones and to weigh the latter against the beneficial effects" (p496).

As with any treatment, there is the issue of informed consent for DBS including whether proxy consent is acceptable in severe Parkinson's disease cases (Glannon 2010). Clausen (2011) felt that unrealistic expectations by patients were more of an issue as it would encourage consent without an accurate understanding of risks and benefits.

Another issue is what treatments DBS should be used with. Beyond Parkinson's disease, there is major depression and OCD, and interest in its use with conditions like obesity, Alzheimer's disease, and addiction. The evidence for these latter uses is often (single-person) case studies. But this method is prone to "publication bias" - how many failed cases are not reported for every successful case published (Schlaepfer et al 2010)? Furthermore:

Investigators can be prone to conflicting interests through industrial funding, own patent rights and positions of dual agency. With respect to industrial funding, researchers should not only disclose their relationships to industry but also justify why they are necessary... In addition, investigators should avoid positions of dual agency where they should conduct and oversee the research at the same time (Clausen 2011 p498).

BCI are being developed for patients with conditions like locked-in syndrome (LIS) where the sufferer is completely paralysed but with conscious awareness and residual eye movements. These eye movements can be used to control spelling devices on the computer, and thus allow communication. There is some concern that the doctors interpret the answers of the LIS patients correctly. "As physicians are prone to compensate limited communication skills by projecting their own expectations onto the patients' signs, several specific safeguards in communicating with LIS patients have been recommended, including asking unambiguous questions one by one, consistent criteria for all answers, and video-taping of conversation to enable objective verification of answers" (Clausen 2011 p498).

Going a step further than BCI, animal experiments with BMI have taught primates to control an artificial limb, for example, through brain-wave changes (Velliste et al 2008).

Philosophical issues come into any understanding of psychiatry. These include agency, the self, and how to conceptualise "human being" (Potter 2010).

Explanations for abnormal behaviour often end up as "normative judgments" (Potter 2010). For example, anxiety disorders develop because an individual has "too much" fear, but by what standard is it "too much"?

Implicit in psychiatry is the question of the relationship between the mind/mental events/subjective experience and the brain/physical state. Though there are different positions, the biomedical dominance in psychiatry tends to view a mental event with a physical event. In other words, a mental event like hearing voices can be explained by physical processes in the brain (eg: inappropriate dopamine neurons firing). However, some have argued that there is still the need for another level of explanation as the biological one does not account for why the "voices" were saying particular things (Thornton 2011).

An alternative view is enactivism (or externalism) (Thornton 2011). The mind is seen as extended beyond the physical boundaries of the skull to include the body, or the linguistic community of the physical environment ²⁶.

6.3. BEWARE

Some of these ethical issues may seem overconcerned, but it is only necessary to look at the extremes to see where unfettered science can end up in the wrong hands; for example, the Nazis in World War II.

For example, Gejman (1997) described the involvement of Ernst Rudin (who had been involved in early twin studies and the "genetics" of schizophrenia at the beginning of the twentieth century) in the enactment of the Sterilisation Law, and in eugenic euthanasia during World War II. In both cases, the focus was upon individuals with mental "weakness" (learning disability as it is called today, and severe mental disorders) and physical disabilities. Gejman quoted a letter from historian, Benno Muller-Hill: "A German who was diagnosed 'schizophrenic' had to be sterilised without exception. Equally, a person who was diagnosed 'schizophrenic' and who was hospitalised for at least five years had a strong chance to be murdered in the euthanasia murders".

On a different issue, Seltzer and Anderson (2001) described how the systematic collection of data about a population has been misused in the extreme cases like Nazi Germany. The authors concluded that "numbers and the

²⁶ Also called the "extended mind hypothesis" (Potter 2010).

systems that produce them are morally neutral. It is what we - or others - do with them that counts" (p507).

6.4. APPENDIX 6A - STOPPING DRUG TREATMENT

Drug treatment may be reduced or stopped for individuals with serious mental disorders for reasons like (Moncrieff 2006):

- i) Patients request it (often because of the side effects being unpleasant).
- ii) The size of doses may be too high.
- iii) Long-term drug treatment may not be beneficial.

But reducing medication can lead to problems. This is assumed to be the re-emergence of the underlying condition, but Moncrieff (2006) argued that the problems are, in fact in many cases, withdrawal from long-term psychotropic drug treatment. These problems include somatic discontinuation syndromes (withdrawal or rebound reactions; eg: anxiety, sleep problems), and rebound psychosis (eg: with use of clozapine).

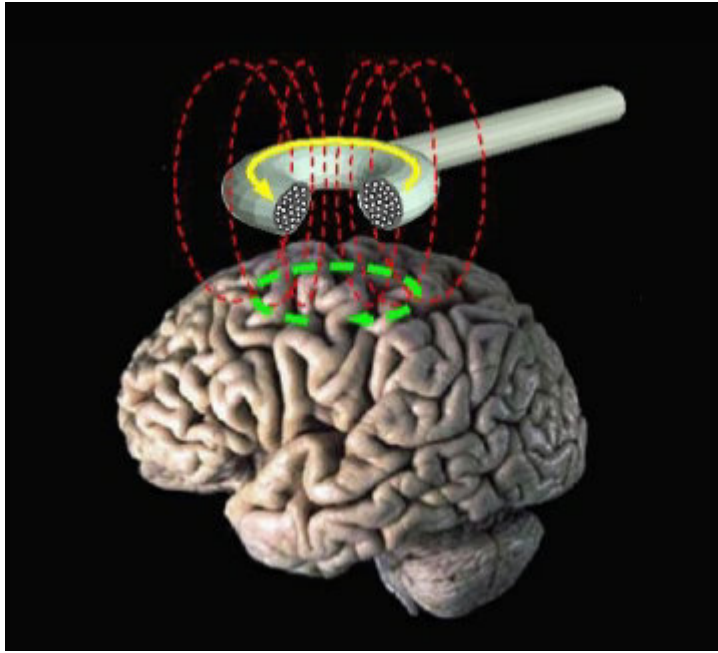
Moncrieff (2006) concluded: "I do not want to rule out that some problems following drug withdrawal may be genuine relapses that are due to the resurfacing of the underlying illness in the absence of treatment. However, the neglect of the adverse effects of drug withdrawal mean it is likely there are fewer cases of this than is generally assumed in clinical trials and practice. This adds support to those who question the benefits of long-term maintenance treatment in psychiatric conditions and suggests that the recurrent nature of psychiatric illness may sometimes be iatrogenic" (p522).

6.5. APPENDIX 6B - REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION AND AUDITORY HALLUCINATIONS

Repetitive transcranial magnetic stimulation (rTMS) involves the stimulation of brain areas using a strong magnetic pulse placed close to the skull (figure 6.1) ²⁷.

Hoffman et al (1999) were the first to report the reduction of auditory hallucinations in three individuals with schizophrenia after receiving rTMS (applied to the left temporo-parietal region at a frequency of 1Hz). Subsequently, in a larger randomised controlled trial,

²⁷ Reported side effects of TMS include scalp irritation and pain, and, rarely, seizure (Allan et al 2012).



(Source: National Institute of Neurological Disorders and Stroke;
http://intra.ninds.nih.gov/Research.asp?People_ID=196; in public domain)

Figure 6.1 - Repetitive Transcranial Magnetic Stimulation.

Hoffman et al (2005) found that just over half of fifty patients had a reduction in auditory hallucinations of at least one-fifth for a period of about three months after rTMS.

Other studies have reported similar success leading to the first meta-analysis by Aleman et al (2007), which found a large effect for 1Hz rTMS over a sham version. But this meta-analysis pooled different types of studies - double-blind crossover studies (where patients have rTMS for a certain period of time, then sham rTMS for a period, or vice versa), and double-blind parallel studies (where individuals have either rTMS or the sham version) (Montagne-Larmurier et al 2011)²⁸.

There have been other studies and meta-analyses of the efficacy of rTMS with auditory hallucinations, but there are common problems with them including (Montagne-Larmurier et al 2011):

i) Double-blind or open studies - In the former case, the patient nor the administrator of the treatment know if it is real or sham.

²⁸ Subsequently, meta-analyses have been performed including the same studies and others by Tranulis et al (2008), Freitas et al (2009), and Slotema et al (2010) (Montagne-Larmurier et al 2011).

ii) Individuals with schizophrenia medicated or not; type of medication; how much.

iii) Small sample sizes.

iv) Stimulation parameters including where rTMS applied; for how long and how often (eg: 4 days 3600 stimuli vs 20 days 24 000 stimuli); intensity of stimulation.

v) Length of wash-out period in crossover designs (ie: gap between changing from treatment to placebo or vice versa) (eg: 4 days vs 4 weeks).

vi) Nature of the placebo or sham version (eg: rTMS coil turned off vs turned on but away from head).

vii) Publication bias - Only positive results published.

With respect to these weaknesses of studies, Montagne-Larmurier et al (2011) attempted to draw conclusions about the use of rTMS.

1. What is the optimal stimulation frequency? 1Hz rTMS is most common, but 20Hz has been used successfully as has continuous theta burst stimulation (cTBS) (eg: 50Hz).

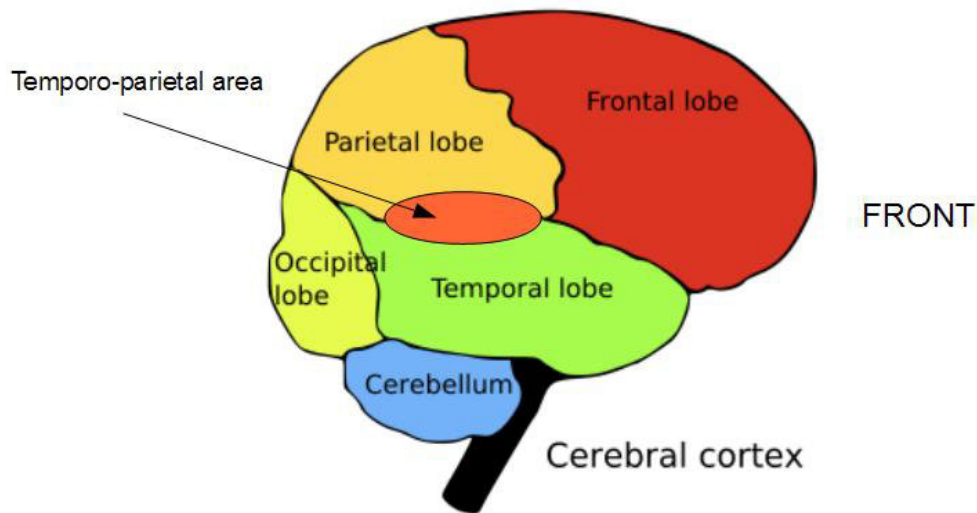
2. What is the optimal site on the brain to apply stimulation? The left posterior temporo-parietal cortex (figure 6.2) seems to have been most successful (compared to the right hemisphere or both hemisphere).

3. What are the long-term effects? Few studies have investigated whether improvements continue beyond 2-3 weeks of treatment.

4. Which patients benefit from rTMS? It is not clear from studies, but treatment-resistant schizophrenics probably benefit least.

5. Is there a placebo effect for sham rTMS? For example, Saba et al (2006) found that both the rTMS and the sham group improved over a ten-day period.

6. What are the neurobiological changes produced by rTMS? A limited number of studies have used neuro-imaging to investigate the changes to the brain after rTMS. One



(My drawing on image in public domain)

Figure 6.2 - Temporo-parietal area of cortex.

suggestion is an increase in blood oxygen levels in the stimulated area, but it is not clear.

Montagne-Larmurier et al (2011) finished their review of the studies on a downbeat note: "Using rTMS to treat auditory hallucinations currently seems less promising than it did ten years ago because of the variable clinical effects and the important drawbacks of this treatment. Said drawbacks include that rTMS is time-consuming for the medical staff, optimal treatment duration is unclear, the necessary equipment is costly, and the system is not necessarily readily available" (p538).

TMS techniques have been used with other mental disorders, like depression. For example, in New South Wales, Australia, Loo et al (2012) recently compared transcranial direct current stimulation (tDCS)²⁹ to the left prefrontal cortex with a sham version among 64 participants. All participants were diagnosed with DSM-IV major depression currently, and were randomised to fifteen treatments (active or sham) over three weeks.

The active treatment showed a significantly greater improvement than the sham group at the end of the sessions. But many of the participants continued with

²⁹ tDCS involves direct contact with the skull whereas TMS does not.

their anti-depressant medication during the study. The authors argued that this had a limited effect because the participants were medication treatment-resistant.

Medication treatment-resistant depression is the greatest challenge. "The holy grail of treatment would be one that is as effective as ECT, but is better tolerated, minimally invasive (and ideally does not require general anaesthesia) and has no serious side-effects including negative effects on cognition. Some neurostimulatory therapies, such as vagal nerve stimulation and deep brain stimulation, are a long way from routine use given that they are both more invasive than ECT, with potentially serious side-effects. They are currently not used within routine clinical settings and are at a relatively early experimental stage. In contrast, transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) promise to be well-tolerated minimally invasive treatments for depression" (Allan et al 2012 p10).

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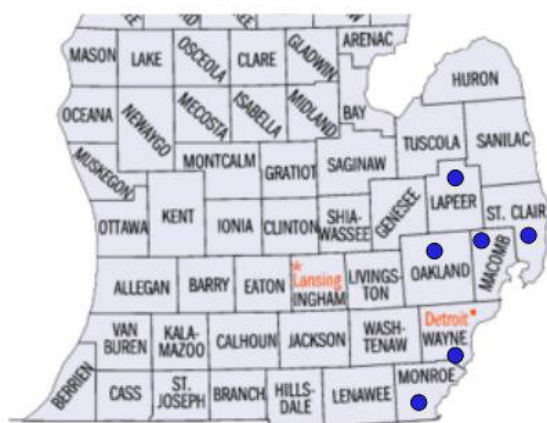
7. POST-TRAUMATIC STRESS DISORDER IN EVERYDAY LIFE AS WELL AS POST-WAR

- 7.1. Everyday life
- 7.2. Multiple trauma
- 7.3. Victims of conflict
- 7.4. References

7.1. EVERYDAY LIFE

Post-Traumatic Stress Disorder (PTSD) was originally (devised and) diagnosed in US Vietnam War veterans, but it has subsequently been applied to other trauma than war. Epidemiological studies have attempted to calculate the rate among the general population.

For example, the 1996 Detroit Area Survey of Trauma (Breslau et al 1998) recruited a representative sample of 2181 18-45 year-olds in the six-county Detroit Primary Metropolitan Statistical Area of the USA (figure 7.1)³⁰. Census data stated that the "population" (ie: those within the age range of the study) was 1 922 173 persons.



(Blue dot = part of study)

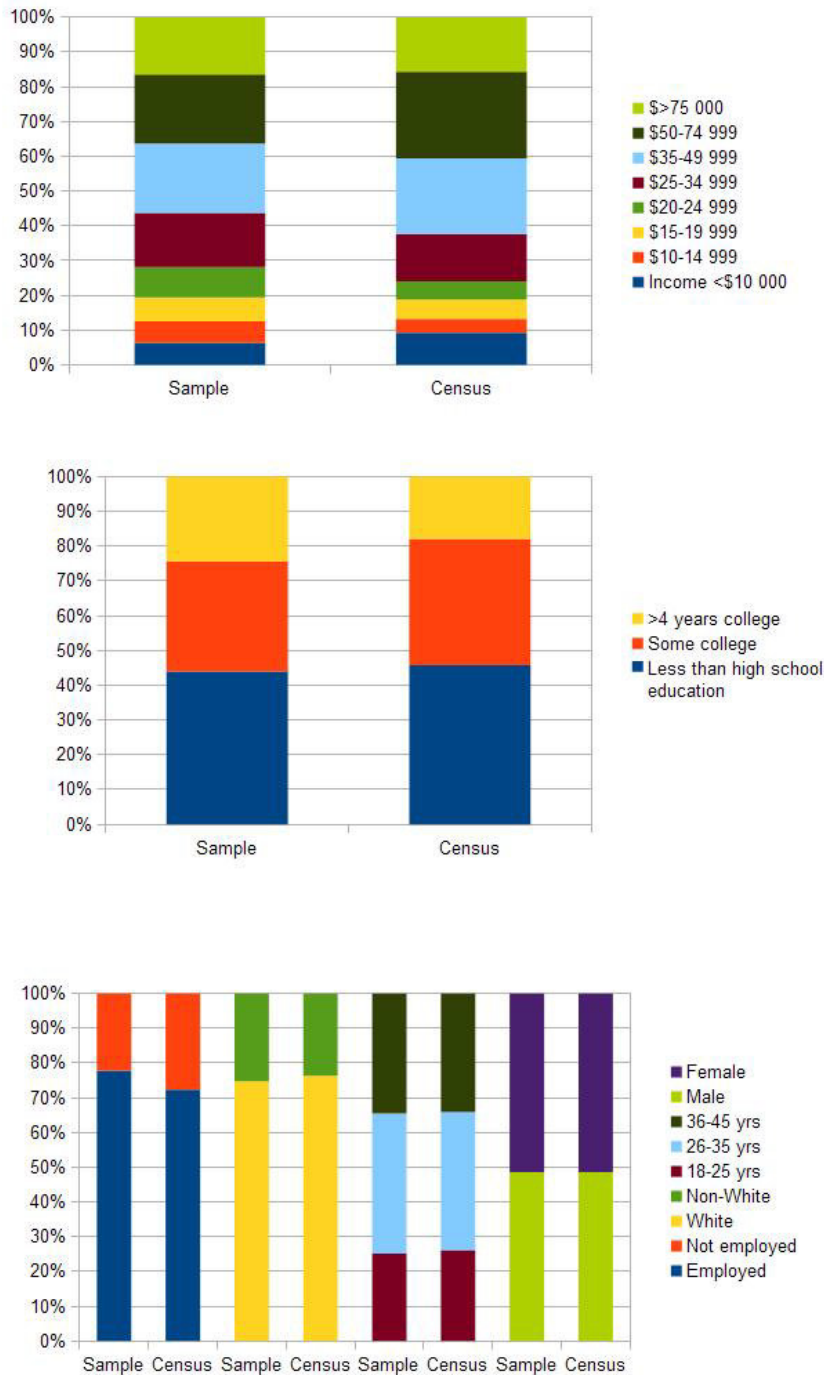
(Source: US Federal Government; in public domain)

Figure 7.1 - Map of counties in southern Michigan showing six in Detroit Area Survey of Trauma.

The sample was recruited by random-digit telephone dialling a total of 6110 households. About three-quarters of those accepted the call, and about two-thirds of them had age-eligible adults. if the household included more

³⁰ The six counties were Lapeer, Macomb, Monroe, Oakland, St.Clair and Wayne.

than one age-eligible respondent, a random selection was made. The make-up of the sample was compared to US Census data for the area to check for representativeness (eg: 48.6% male and 51.4% female) (figure 7.2).



(Data from Breslau et al 1998 table 1 p629)

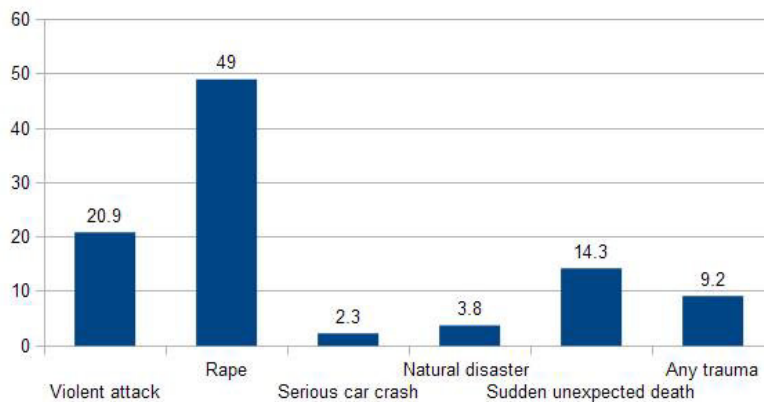
Figure 7.2 - Comparison of sample with US Census data on certain characteristics.

The telephone interview lasted about thirty minutes and asked about the lifetime experience of 19 types of traumatic events (eg: rape; sudden unexpected death of close friend or relative; serious car or motor vehicle crash). Diagnosis of PTSD for any event experienced was made using a series of diagnostic questions.

The exposure to any of the traumatic events at any time in their lives occurred in 89.6% of the sample. Sudden and unexpected death of a loved one was the most commonly experienced (60%), while being mugged or threatened with a weapon was less common (25%), and being held captive/kidnapped was rarest (1.8%). Individuals who had experienced trauma reported an average of 4.8 distinct events.

Men were more likely to have experienced violence in some form, and women more sexual-related violence. Likewise non-Whites, lower educated, and lower income groups had experienced more violence.

The prevalence of PTSD after a trauma was 9.2% (13% for women and 6.2% for men), but the rate was 39.5% after violent attack. Sudden unexpected death of a loved one was also an important cause of PTSD (figure 7.3). Overall, in three-quarters of cases the PTSD lasted longer than six months.



(Data from Breslau et al 1998 table 4 p631)

Figure 7.3 - Prevalence (%) of PTSD for selected traumas.

7.2. MULTIPLE TRAUMA

PTSD is studied more often in combatants than civilians, and among refugee civilians than people who stay in the area of conflict. Studies tend to be short-term also (eg: one year after conflict).

Priebe et al (2010) addressed these problems by studying civilians who had experienced the wars after the

collapse of Yugoslavia in the 1990s. In five countries (Bosnia-Herzegovina, Croatia, Kosovo, the Republic of Macedonia, and Serbia) (figure 7.4), adults were randomly selected for face-to-face interviews in 2005-6. Fifteen regions were chosen that had been subjected to at least seven days of shelling (eg: Srajevo in Bosnia-Herzegovina). These experiences were between 5-15 years ago. In total 3313 interviews were completed.



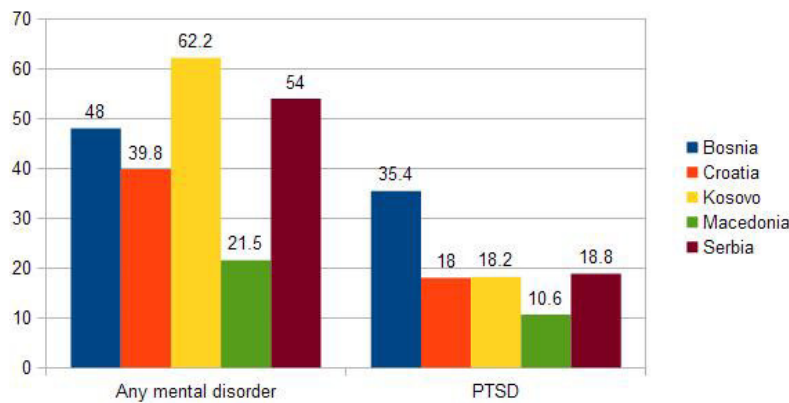
(Drawn with MapCreator2)

Figure 7.4 - Position of five countries in study.

Nearly half of the respondents (44.8%) reported a mental disorder with PTSD being the most common (mean 20.2%; range 10.6 - 35.4%) (figure 7.5). These compared to 16-37% in Algeria, Cambodia, Ethiopia, and Gaza (De Jong et al 2003), and 24.8% in Rwanda (Pham et al 2004).

Suffering from PTSD in the Balkans was associated with being older, female, and unemployed, and more traumatic experiences during and after the wars.

Table 7.1 summarises the key strengths and weaknesses of the Priebe et al (2010) study.



(Data from Priebe et al 2010 table 4 p522)

Figure 7.5 - Percentage of respondents in five countries.

STRENGTHS	WEAKNESSES
<ol style="list-style-type: none"> 1. Large-scale community-based study. 2. Random sampling. 3. Long-term effects of war. 4. Trained local interviewers using DSM-IV criteria for diagnosis. 5. Civilians and combatants in sample. 	<ol style="list-style-type: none"> 1. Point prevalence (ie: at time of interview) and no history taken. 2. No pre-war data meant that it was not possible to say with absolute certainty that the wars caused PTSD. 3. Risk of recall bias by respondents (and no independent verification of information). 4. Cultural differences in admitting to mental disorders to a stranger. 5. Different interviewers used in each country, who, despite training, may not have recorded information in the same way.

Table 7.1 - Key strengths and weaknesses of Priebe et al (2010) study.

War-related trauma is linked to subsequent mental problems including PTSD. through the experience of war, and/or through the loss of loved ones in war.

Morina and Emmelkamp (2012) explored the combined traumas in a study of women in Drenica ³¹ in Kosovo ³² ten years after the Kosovo war in 1998-9. One hundred lone mothers widowed (LMW) by the war ³³ were compared with 71 non-bereaved married mothers (NBMM) and 35 mothers bereaved post-war (but not the loss of husband) (MMPB). All participants had experienced the war (figure 7.6).

Lone mothers widowed (LMW) - war trauma + war-related loss of husband + lone parenting

Non-bereaved married mothers (NBMM) - war trauma

Married mothers post-war bereavement (MMPB) - war trauma + bereavement

Figure 7.6 - Three groups of women in study.

Significantly more women from the LMW group reported current major depressive disorder, an anxiety disorder, or substance use disorder (96%) than the NBMM (54.9%) and MMPB (60%) women (figure 7.7).

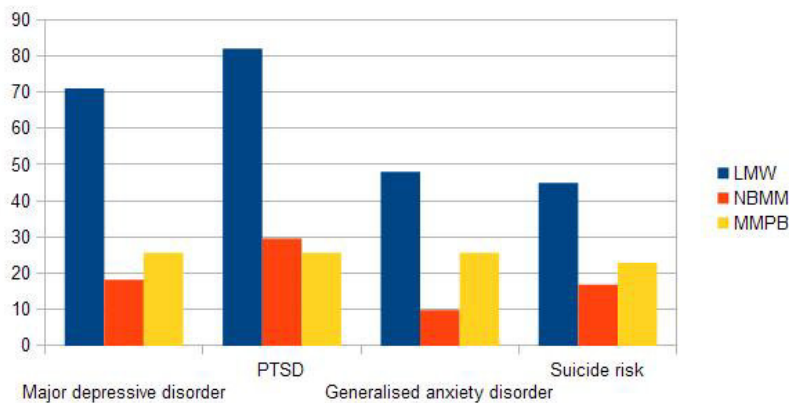


Figure 7.7 - Percentage of women diagnosed with current disorder.

The LMW group were significantly older than the

³¹ Central Kosovo region, west of capital Prishtina, including town of Glogovac.

³² Kosovo is recognised as an independent country by some nations, but contested as only a region of Serbia.

³³ Custom in Kosovo society for widowed mothers not to remarry.

other groups, had lower income and less education, and experienced more war-related trauma. Controlling for these differences, LMW women still had significantly higher rates of major depressive disorder and PTSD than the other two groups, and of generalised anxiety disorder and suicide risk than the NBMM group.

This study showed the long-term consequences of war trauma, and the effect of combined trauma for LMW. Each of the three factors - war trauma, sudden loss of husband, and enforced lone parenting - has negative consequences for psychological health as shown in previous research, as well as the consequences cumulatively.

7.3. VICTIMS OF CONFLICT

Steel et al (2009) found 161 articles³⁴ reporting 181 surveys in forty countries with 81 866 participants about PTSD among refugees and conflict-affected populations in a systematic review and meta-analysis. The mean rate was 30.6%, but the prevalence in individual studies varied greatly due to methodological differences (between 0-99%).

The studies varied in four key methodological ways³⁵:

i) Sample size - eg: smaller sample (50-100 people) and higher rate of PTSD found.

ii) Sampling method - eg: non-probability sampling of a target population and higher rate of PTSD found.

iii) Measurement and diagnosis of PTSD - 21 different measures used; eg: self-reported measures produced prevalence rates on average 10% higher than diagnostic interviews by clinicians/trained interviewers.

iv) Diagnostic time frame - eg: current PTSD (mean 32.9%) or period of time (eg: last 12 months) (mean 17.0%).

For example, as a generalisation, small sample studies of specific target populations (eg: refugees) found higher rates of PTSD than large studies using random sampling of the general population.

³⁴ In English between 1980 and May 2009.

³⁵ The studies also varied in other ways like:

i) Country - 122 surveys in low and middle-income countries, and 59 in high-income countries. Rates of PTSD varied from 10% in Vietnam to 30.3% in Cambodia.

ii) Date undertaken - 25 surveys in the 1980s, 73 in the 1990s, and 83 in the 2000s.

It was found that cumulative exposure to potentially traumatic events (PTEs) was important in the development of PTSD (and depression). For example, individuals permanently resettled in another country (away from the conflict) had lower rates of PTSD than those displaced internally or externally or living in a refugee camp.

After all the adjustments for variations between the studies, experiencing torture was the strongest predictor of PTSD.

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8. SUFFERING FROM HYPOCHONDRIASIS

Hypochondriasis (Hy) is "a fear of having, or the idea that one has, serious disease based on misinterpretation of bodily symptoms" (Persing et al 2000 p329), and this fear continues despite contrary medical evidence and reassurance.

From the doctor's point of view, the medical information and reassurance should be enough to reduce the anxiety about an illness, but the Hy sufferer continues to worry and seek more reassurance.

Persing et al (2000) questioned 20 US Hy sufferers with semi-structured interviews ³⁶ (table 8.1) to see if understanding their perspective could help explain the continued anxiety after doctors' reassurances. Their responses were compared to 26 controls.

The number of positive and negative comments made during the interview were counted and categorised in relation to the doctor, the health service, and the patient themselves. Hy sufferers made more negative comments generally than controls (mean 15.6 vs 7.7) (table 8.2), and specifically of the doctor (eg: hurried or careless) (mean 7.6 vs 3.2), and themselves (eg: blame self for health problems) (mean 3.4 vs 1.3). There was no difference in the number of positive comments between the two groups (mean 9,5 vs 9.6).

Feeling that there was a positive relationship with the doctor was key to the Hy sufferer having a positive perception. Persing et al (2000) concluded: "management of hypochondriacal patients rests on a collaborative therapeutic relationship. To establish such a relationship the physician must communicate to the patient that his or her distress and reason for seeking medical care are legitimate. The physician must recognise that the goal in treatment is not removal of symptoms but assisting the patient to cope with them" (p340).

1. What health problems have you sought help for recently?
2. What care have you received? That is, where have you gone and who have you seen? Also, is care ongoing or complete with respect to recent problems?
3. What has been the technical quality of the care you have received? That is, have the doctors been knowledgeable, thorough, and have they done appropriate tests and examinations?

³⁶ The semi-structured interview combines standardised questions (which allow comparison between interviews) with the opportunity for individual interviewees to develop their answers at length, and even take the interview in a different direction (but this idiosyncratic element limits comparability).

4. What about the personal side of the care you have received? Have the doctors shown concern, seemed interested in you and in your problems? How have you reacted to the doctors you have seen and how have they reacted to you?

5. What have you reacted negatively to and what positively?

6. Often patients are worried when they consult a doctor and hope to be reassured. How reassured have you been?

7. What has been the outcome of the care you have received? Has testing been complete? Has treatment been initiated? Has the problem you sought help for been resolved?

(Source: Persing et al 2000 p340)

Table 8.1 - Questions asked in semi-structured interview.

- Unconcerned - "You know, it was more like they were there for a paycheck".
- Disrespectful - "they get testy when you try to tell them anything".
- Hurried - "It seemed like there was a waiting room full of people and they were just trying to get rid of me".
- Rejecting - "They made me feel like it was all in my head".

(Source: Persing et al 2000)

Table 8.2 - Sample of negative comments about doctors by Hy sufferers.

REFERENCE

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International Journal of Psychiatry in Medicine 30, 4, 329-342

9. VIOLENCE, PREMATURE DEATH AND MENTAL DISORDERS

- 9.1. Violence
- 9.2. Premature death
 - 9.2.1. Therapeutic lifestyle changes
- 9.3. References

9.1. VIOLENCE

Many studies have attempted to estimate the risk of violence by individuals discharged from psychiatric hospitals (or with mental disorders) relative to the general population. But many of these studies have methodological weaknesses (Steadman et al 1998):

i) Measurement of violence via official reports like arrest records or rehospitalisation records.

ii) Measurement of violence via self-reports.

iii) Lack of information about the context of the violence (eg: domestic).

iv) Limited information about the timing of the violence (eg: how soon after release from hospital).

v) The use of samples with high rates of violence (eg: men with a history of violence).

vi) Little research on women.

Steadman et al (1998), in their study, used three sources - self-reports, "collateral informants" (eg: victims), and official records. The final sample of 1136 individuals aged 18-40 years were released from three psychiatric hospitals in the USA, and had a diagnosis of psychosis, substance abuse, or a personality disorder. Detailed information was collected about the violent act including the target, location, and seriousness over a one-year period. A comparison sample of 519 individuals living in the local communities was used.

The use of three sources of information produced higher rates of violence than previous studies. The prevalence of violence between discharged patients without substance abuse was similar to the community sample without substance abuse, but was significantly higher for patients with substance abuse or co-morbidity with substance abuse, and the community sample with substance abuse. The highest risk of victimhood by ex-patients' violence was among family members and friends.

The risk to strangers was higher among the community sample (Link and Stueve 1998).

9.2. PREMATURE DEATH

Suffering from a mental disorder is associated with premature death (from natural and unnatural causes) relative to the general population. Life expectancy is at least 20% less for individuals with mental illness compared to the general population in high-income countries, which translates to a 20-year "mortality gap" for men and 15 years for women (Thornicroft 2011)^{37 38}. This may happen for a number of reasons (figure 9.1):

i) The cause of the mental disorder is the cause of the death - the mental disorder leads to physical deterioration and consequently early death (eg: Down's syndrome).

ii) Having the mental disorder produces behaviour which can be fatal - eg: starvation by anorexia nervosa sufferers.

iii) The mental disorder itself is detrimental to health - eg: substance abuse.

iv) Having the mental disorder leads to unhealthy behaviour that consequently produces death (indirect effect) - eg: poor diet of substance abusers. There are also hidden/undiagnosed unhealthy behaviours - eg: a number of self-induced deaths among schizophrenics may be due to water intoxication (ie: excessive consumption of water) which goes unnoticed (quoted in Whitchurch and Alexander 2011).

v) There are health risks implicit with the behaviour - eg: overdose and substance abuse.

vi) Risky side effects of the disorder - eg: road traffic accidents while intoxicated (alcohol abuse).

vii) Consequences or side effects of treatment - eg: health risks of long-term use of anti-psychotics.

viii) Having the mental disorder leads to suicidal

³⁷ Between 3-30 years lost with 2.6 times greater risk of early death for alcohol/substance use disorders and 1.5-1.8 times for depression (Markkula et al 2012).

³⁸ Thornicroft (2011) felt that this difference "denotes a cynical disregard for these lost lives, and shows, in stark terms, by just how much people with mental illness are categorically valued less than others in our society" (p441).

behaviour - eg: depression.

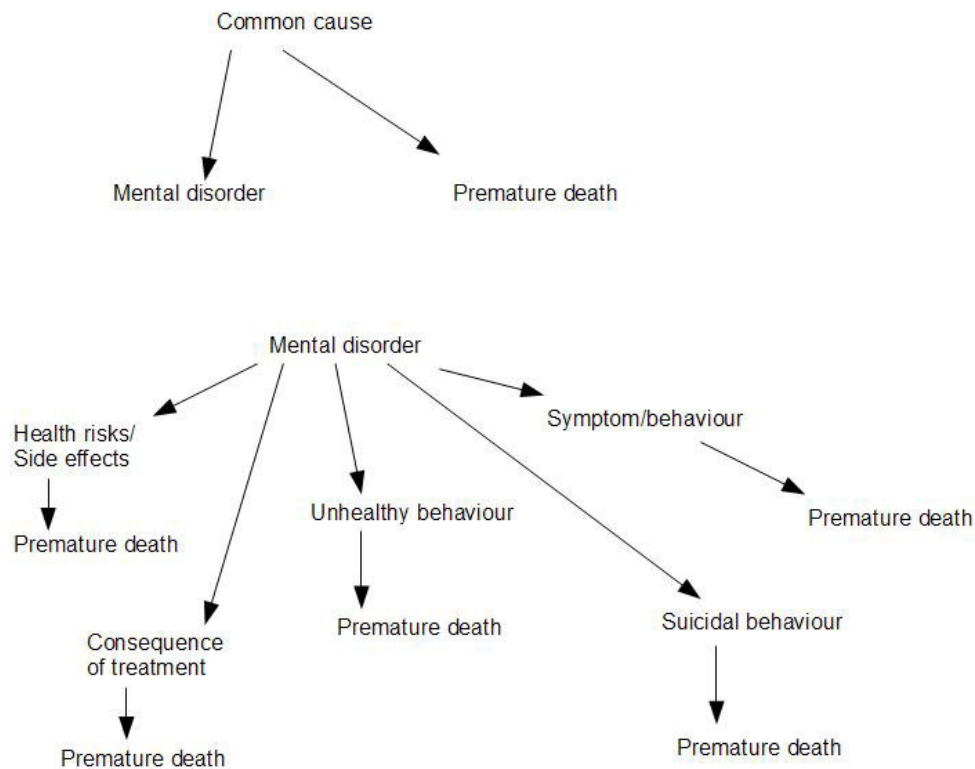


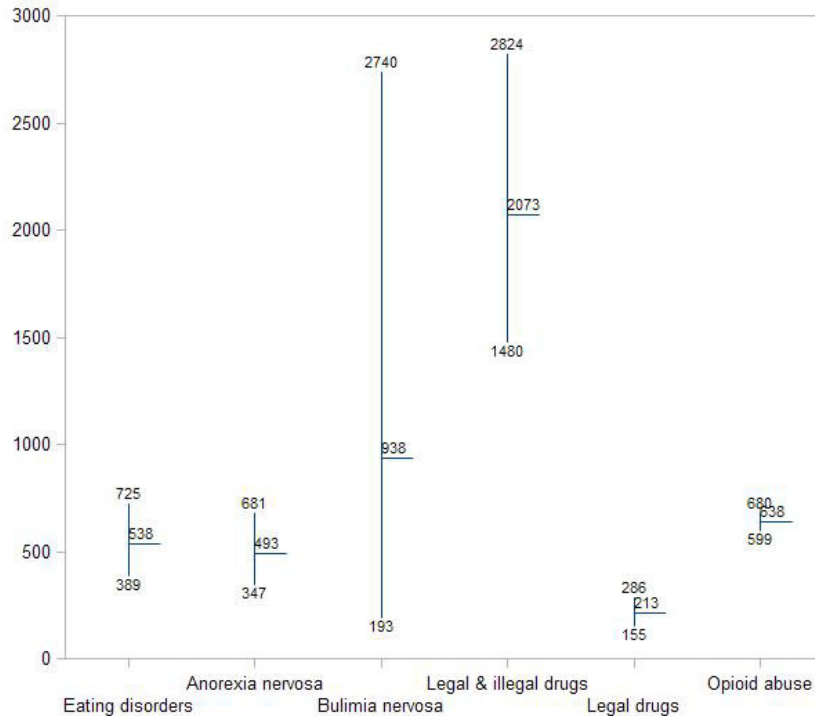
Figure 9.1 - Different ways that having a mental disorder can lead to premature death.

Thornicroft (2011) also noted poorer access to physical healthcare services than the general population, including less likely to receive screening for cancer. While "diagnostic overshadowing" leads to the misattribution by physical healthcare staff of physical symptoms to the mental disorder. For example, an individual with anxiety disorders who complains of chest pains may have them attributed as somatic symptoms of the disorder rather than symptoms of a physical illness.

Harris and Barraclough (1998) calculated the standard mortality ratio (SMR) for 27 categories of mental disorders. The SMR is the observed number of deaths divided by the expected number of deaths, multiplied by 100. The figure for the general population is 100 (ie: comparison baseline).

The categories of mental disorders with the highest SMRs were eating disorders (538) and substance abuse (eg: legal and illegal drugs 2073) (figure 9.2). The SMRs ranged from 96 (anxiety neurosis in DSM-III-R) to 2073

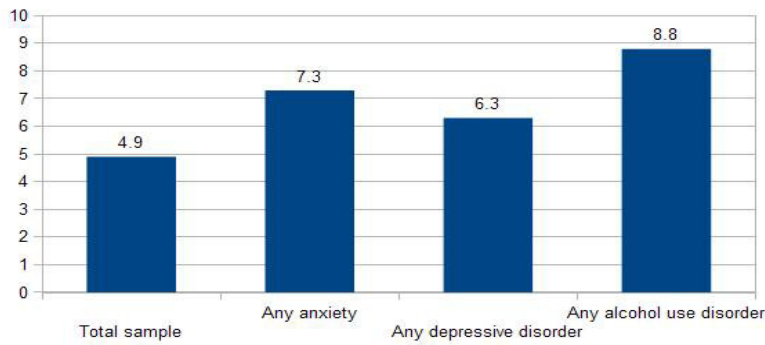
for all causes of death. For unnatural causes, like suicide, the SMRs ranged between 103 (mental retardation) and 3294 (substance abuse).



(Data from Harris and Barraclough 1998)

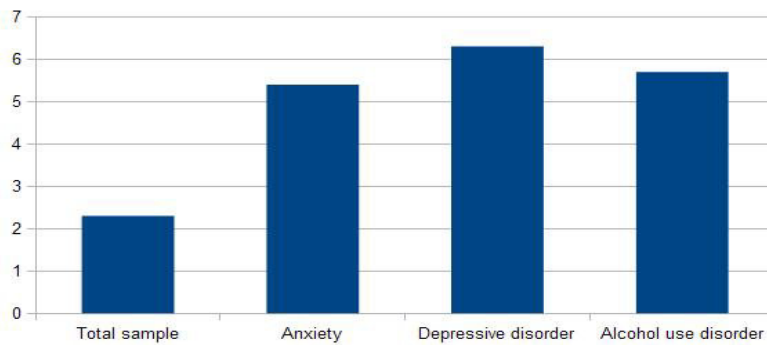
Figure 9.2 - SMRs of all deaths for eating disorders and substance abuse (with 95% confidence intervals).

Markkula et al (2012) used data from 8028 nationally representative Finns from the Health 2000 Study. Comprehensive health information was collected in 2000-1, and mortality follow-up at the end of 2008. Of the study population, 323 individuals had died (most from natural causes). An individual with depression, anxiety, or alcohol use disorders at baseline were more likely to be in this number than individuals without any of these disorders, particularly among the 30-50 year-old age group (figures 9.3, 9.4 and 9.5). Controlling for socio-demographic variables like age, and health status, depression and alcohol use disorders were significantly associated with mortality.



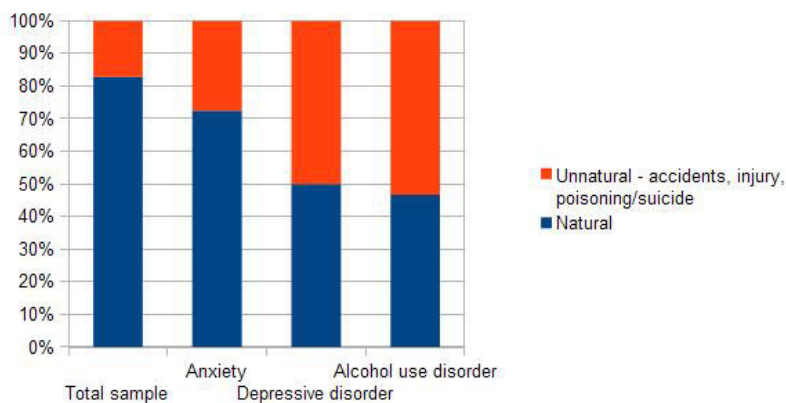
(Data from Markkula et al 2012 table 1 p145)

Figure 9.3 - Percentage of deaths in different groups.



(Data from Markkula et al 2012 table DS3)

Figure 9.4 - Percentage of deaths in different groups in 30-50 year-old age group.



(Data from Markkula et al 2012 table 1 p145)

Figure 9.5 - Percentage of different types of deaths in each group.

The baseline characteristics of the three groups of mental disorders sufferers were different to the general population in the following statistically significant ways (table 9.1).

	DEPRESSION	ANXIETY	ALCOHOL USE
Gender	female	-	male
Age	-	-	30-44 years old
Income	lowest	lower	-
Marital status	single	single	single
Smoking	-	yes	yes

(Source: Markkula et al 2012)

Table 9.1 - Statistically significant differences between three groups of mental disorders sufferers and general population.

Markkula et al (2012) pointed out that many studies do not include undiagnosed cases of mental disorders, and depend on self-reports of physical illness. Their study had a number of strengths and weaknesses (table 9.2).

STRENGTHS	WEAKNESSES
1. Large-scale study. 2. Detailed baseline measures taken 3. High participation rate (ie: low drop-out) and thus reduced of selection bias.	1. Each diagnostic group did not include large numbers of individuals or deaths. 2. Alcohol consumption and physical activity were not included in analysis. 3. Mental disorders diagnosed at baseline and no follow-up to see if status changed.

Table 9.2 - Key strengths and weaknesses of Markkula et al (2012) study.

9.2.1. Therapeutic Lifestyle Changes

Lifestyle can contribute to poor mental health (and risk of early death), and can be used to improve it in the form of therapeutic lifestyle changes (TLCs) (Walsh 2011).

Walsh (2011) summarised the benefits of eight TLCs.

- Exercise - eg: reduces risk of depression.

- Nutrition and diet - eg: a balanced diet which reduces excess calories reduces the effects of schizophrenia.
- Time spent in Nature - eg: patients recovering in hospital rooms with views of natural settings leave hospital sooner and are less stressed than in rooms with no view or an urban one.
- Having good relationships - eg: a good therapeutic relationship is a cornerstone to effective therapy.
- Recreation and enjoyable activities - eg: reduce stress.
- Relaxation and stress management - eg: varied techniques, like meditation, reduce stress.
- Religious and spiritual involvement - eg: a positive relationship between "religious" involvement and mental health.
- Contribution and service - eg: altruism produces the "helpers' high" (ie: positive emotions).

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10. STUDYING MENTAL HEALTH OF MILITARY

- 10.1. Mental health of military personnel
- 10.2. Gulf War
- 10.3. References

10.1. MENTAL HEALTH OF MILITARY PERSONNEL

The mental health of military personnel has been much studied in relation to before and after deployment, and a limited amount during operations.

An example of the latter is a survey of UK armed forces³⁹ in Iraq by Mulligan et al (2010). Between January-February 2009, a purposive sample⁴⁰ of 611 personnel⁴¹, part of Operation TELIC 13⁴², in eight locations including Basra and Baghdad completed self-reported Operational Mental Health Needs Evaluation (OMHNE) questionnaires⁴³. These were specially designed to include questions related to psychological distress, post-traumatic stress disorder (PTSD), and health.

About one-fifth of respondents were above a cut-off point for psychological distress, and, overall, 3.4% were classed as showing PTSD symptoms. Table 10.1 shows the factors linked to these cases.

In terms of studies of the whole military experience, the Millennium Cohort Study was launched in 2000 in the USA to run for 21 years. Panel 1 involved the recruitment of 77 047 US military personnel between 2001-3, and baseline measures were collected about health and mental health with over 450 questions.

Two summary scores were calculated - the mental component summary (MCS) and the physical component summary (PCS) - where a higher score is a more favourable health or mental health status. The mean scores were similar to 1998 US general population norms.

³⁹ Royal Navy, army, and Royal Air Force.

⁴⁰ Random sampling was not possible because some personnel were not available (eg: special forces), and it was a "theatre of war". The purposive sample did match the population of TELIC 13 in terms of rank and service. "Conducting an epidemiological study in a war zone inevitably entails difficulties in accessing all eligible personnel" (Mulligan et al 2010 p409).

⁴¹ It was emphasised that completion of the questionnaire was voluntary, and only one person refused to complete it. This is an exceptionally high response rate (99.8%) compared to surveys of the general population. "It may be, however, that those who did not want to complete questionnaires made themselves unavailable during visits by the research team" (Mulligan et al 2010).

⁴² Operation TELIC is the codename for UK military operations in Iraq since 2003, and TELIC 13 is the period December 2008 to June 2009.

⁴³ Diagnosis was not verified independently. The researchers admitted that the "questionnaires should therefore be considered more as screening than diagnostic instruments".

	MORE LIKELY	LESS LIKELY
PSYCHOLOGICAL DISTRESS	<ul style="list-style-type: none"> • Younger • Female • Junior rank • Experienced significant stress, emotional or family problem in lives 	<ul style="list-style-type: none"> • Stronger unit cohesion • Better perceived leadership • Received pre-deployment stress briefing
PTSD	<ul style="list-style-type: none"> • Junior rank • Having felt in danger of being killed • More combat exposure • Experienced significant stress, emotional or family problem in lives 	<ul style="list-style-type: none"> • Better perceived leadership • Received pre-deployment stress briefing

Table 10.1 - Factors related to psychological distress and PTSD.

10.2. GULF WAR

Nearly 700 000 US military personnel were involved in Operation Desert Storm in 1991 in the Persian Gulf (Kuwait and Iraq, in particular) ⁴⁴ (Kang et al 2000). Within a short time of returning home, a number of these Gulf War veterans (GWVs) reported various symptoms and illnesses (DeFraités et al 1992). This led to the creation of the Gulf War Health Registry in 1992 by the US Government (Department of Veterans Affairs).

This Registry only recorded details of GWVs who reported them. Thus it was a self-selected group of individuals concerned about their health who attended physical examinations.

A random selection of GWVs would give a more representative picture of illness among the group, particularly when compared to a random selection of non-GWVs ⁴⁵. The National Health Survey of Gulf War Era Veterans and Their Families selected 15 000 troops in each group via stratified random sampling ⁴⁶ (Kang et al 2000).

⁴⁴ The UK deployed over 53 000 military personnel (Unwin et al 1999).

⁴⁵ It is not clear if the amount of active service between the two groups was comparable.

⁴⁶ The population was divided into sub-groups (strata) based on gender, unit, and service, and then a random sample was taken from each strata.

Individuals were asked to complete a sixteen-page structured health questionnaire, and later a telephone interview ⁴⁷. Overall, 11 441 GWVs (75% response rate) and 9 476 non-GWVs (64% response rate) completed both of these.

The GWVs reported poorer general health as measured in different ways - eg: positive response to the question, "Thinking back over the past two weeks, did you stay in bed or at home all or part of any day because you did not feel well or as a result of illnesses or injury?" (27.8% vs 14.2%).

In terms of psychological symptoms, GWVs reported more anxiety (45% vs 28%) and depression (36% vs 22%).

In the UK, Unwin et al (1999) compared GWVs (n = 2961) with those deployed in the Bosnian conflict (n = 2620), and those not on active service during the Gulf War period (n = 2614) ^{48 49 50}.

The GWVs reported more symptoms, both physical and mental, than the other two groups ^{51 52}. They were over twice as likely to have symptoms of post-traumatic stress disorder, and one and a half times more likely to report psychological distress than the Bosnian conflict group.

10.3. REFERENCES

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⁴⁷ Self-reported visits to clinics and hospitals were verified, but other symptoms were not, and thus depended on the accuracy of recall and honesty of respondents.

⁴⁸ Only male service personnel were analysed, excluding special forces for security reasons.

⁴⁹ Stratified sampling based on service, rank, age, and sex was used.

⁵⁰ The overall response rate to the postal questionnaire was 65.1%.

⁵¹ These were self-reported with no physical examination for verification.

⁵² Haley (1998) referred to the "healthy warrior effect", where deployed individuals are healthier than non-deployed ones, and this would obscure the effects of deployment.

11. OBSESSIVE COMPULSIVE DISORDER AND UNDERLYING PHYSIOLOGY

According to the biomedical model the behavioural (outward) signs of illness can be traced to underlying physiological processes. This is less clear-cut for mental illnesses, and it is part of the controversy that physiological processes and differences are not evident among sufferers. There is and has been much research to establish the underlying physiology of specific mental disorders.

One case is Obsessive-Compulsive Disorder (OCD). It is proposed that frontal lobe dysfunction is involved leading to cognitive problems (eg: Malloy 1987), but is this distinct from sufferers of other mental disorders like depression? The answer is to compare OCD sufferers with other mental disorders and controls to see if the former show distinct cognitive impairments.

Purcell et al (1998) did so with patients at the Depression and Anxiety Research and Treatment Clinic at the University of Melbourne, Australia. There were thirty individuals with OCD (a type of Anxiety Disorders), 30 with Panic Disorder (another type of Anxiety Disorders), and twenty with Unipolar Depression (a different category of disorders) as well as 30 matched volunteers from the local community (healthy controls).

Each participant completed the Cambridge Neuropsychological Test Automated Battery (CANTAB) which measures cognitive abilities via the computer in a session lasting about ninety minutes. CANTAB involves seven tasks.

i) Spatial span - A number of coloured blocks appear on the screen, and then change colour in order. The task is to remember the order the blocks changed colour. It measures short-term memory capacity varying between 2 to 9 blocks on the screen. There was no difference between the four groups on this task with the average span being 5-6 blocks.

ii) Spatial working memory - Participants were required to search boxes on the screen for a hidden token without returning to boxes that previously held a token. Two scores were calculated - the number of memory errors (ie: returning to the boxes), and a systematic searching strategy score. The OCD group made significantly more errors and had a significantly different strategy score than the other three groups.

iii) Tower of London planning task - Participants are required to move coloured balls on one side of the

computer screen to form a shown pattern on the other side while following certain rules (eg: larger balls before smaller balls). This is a measure of of planning ability based on the number of moves made. There was no difference in the number of excessive moves between the groups, but OCD sufferers took longer to make the first move.

iv) Delayed matching to sample - This tests visual memory by briefly presenting a simple pattern, and after a short gap, four choices. No difference between the groups in accuracy - around 80-90% depending on the length of the gap (eg: 4 or 12 seconds).

v) Pattern recognition - A complex abstract pattern is presented briefly followed by the pattern and a new one to choose from. This measures perception and memory. All groups were around 90% accurate.

vi) Spatial recognition - A white box is briefly presented at a certain position on the computer screen followed by a choice of two boxes to choose from (of which one is in the correct position). The OCD got significantly less correct.

vii) Intradimensional-extradimensional set shift - A lot of different shapes are presented at once and participants must pay attention to only the part instructed to do so (eg: how many patterns in white boxes are red) and then shift attention when instructed (eg: how many patterns in black boxes not red). A score is calculated for successful completion of each stage of the task. Most controls completed all stages (83%), followed by panic disorder (70%), OCD (60%), and depression groups (50%).

Overall, the OCD group showed impairments in spatial memory and spatial recognition, and were slower to begin in a planning task. These problems link to frontal cortex dysfunction. But it is not possible to say if these problems caused the OCD or were a product of it.

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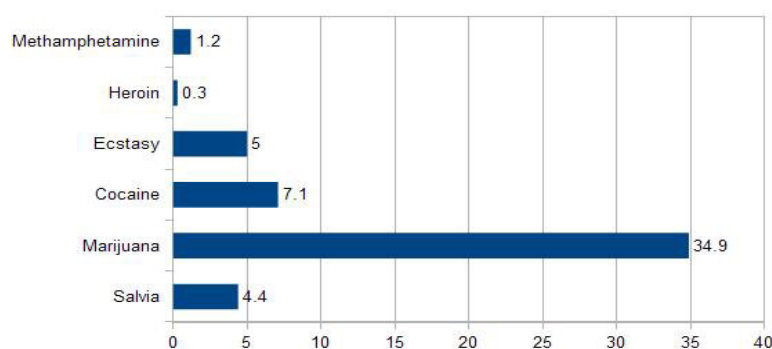
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12. STUDYING SALVIA USE

Concern about drug use usually focuses upon illicit/illegal substances like cocaine. But there are natural, legal substances that enjoy popularity in the West. Lange et al (2008) referred to *Salvia divinorum*, a species of the mint family of plants that produces hallucinogenic effects⁵³. It has been used historically by shamans among Mazatec Indians in Mexico⁵⁴.

Lange et al (2008) found that 4.4% of 1571 college students at a university in the southwestern USA had used *Salvia* at least once in the past year. In this Internet survey of drug use in the academic year 2006-7, other illicit drugs were used more often - eg: marijuana (34.9%) and cocaine (7.1%) (figure 12.1).

Salvia users were significantly more likely to be male, White, fraternity/sorority members, heavy episodic alcohol drinkers, and to use other drugs.



(Data from Lange et al 2008 table 1)

Figure 12.1 - Percentage of students using a drug at least once in the last year.

Studying the effects of drugs is usually based on later self-reports of use like this study above, or controlled laboratory observation⁵⁵. The former can be unreliable with no independent verification, and the

⁵³ Salvinorin A is the active hallucinogen (Lange et al 2010).

⁵⁴ Mazatec shamans chewed the leaves whereas in US, individuals smoke an extract-enhanced leaf product (Lange et al 2010).

⁵⁵ Laboratory studies allow precise testing of the cognitive effects of a substance. For example, a visual scanning task which presents many shapes on the computer screen at the same time, and participants are asked whether one particular shape is present or absent among them. The reaction time to answer and the number of errors are measured. This test can be used during intoxication or to assess the long-term and enduring effects of use as with Ehrenreich et al (1999). They found that early age of onset cannabis users (before 16 years of age) were significantly slower to answer than late onset users and non-users (control).

latter is artificial (table 12.1).

Lange et al (2010) described an opportunist way to study the use of Salvia. Observing individuals who posted videos of themselves taking the substance on YouTube. One hundred videos were randomly selected from those found using the search term "salvia" on YouTube. Only unedited recordings of the whole drug-taking event were included. This left 24 videos, but because the majority were males, ten videos showing females were added to give a total of 34 videos. Three trained observers coded 30-second intervals of the videos using items from the Hallucinogen Rating Scale (HRS) (Riba et al 2001).

Onset of the effect of the drug occurred within one minute of the first "hit" (eg: inhalation of smoke), and this lasted up to eight minutes. The effects were divided into five categories - hypo-movement (eg: limp hands), hyper-movement (eg: uncontrollable laughter), emotional (eg: visibly excited), speech effects (eg: problems with fluency), and heating effects (eg: flushed).

This method of research gives the opportunity to study real-life Salvia use, but information about the dose size was limited, and it was a self-selecting sample. Researching subtle and long-term effects would need a controlled laboratory study.

	CONTROLLED LABORATORY OBSERVATION	POST-EVENT SELF-REPORT QUESTIONNAIRE	INDIRECT OBSERVATION OF YOUTUBE
ADVANTAGES	<ol style="list-style-type: none"> 1. Controlled environment. 2. Physiological measures possible. 	<ol style="list-style-type: none"> 1. Information about illicit/illegal drug use. 2. Anonymity possible. 	<ol style="list-style-type: none"> 1. Real-life use. 2. Study individuals who might not participate in other research.
DISADVANTAGES	<ol style="list-style-type: none"> 1. Artificial situation. 2. Typicality of volunteers for such studies. 	<ol style="list-style-type: none"> 1. No independent verification of information. 2. Recall problems. 	<ol style="list-style-type: none"> 1. Only observable effect studied. 2. Self-selecting sample of videos.

Table 12.1 - Main advantages and disadvantages of three methods of studying drug-taking.

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13. CO-MORBIDITY OF DRUG USE AND SOCIAL PHOBIA

Social anxiety disorder (or social phobia) is a fear related to social situations. Myrick and Brady (2003) defined it as "a marked and persistent fear of situations in which an individual is exposed to unfamiliar people or to the scrutiny of others" with fear of being embarrassed and/or evaluated negatively as the hallmarks.

The lifetime prevalence varies from 2-20% depending upon the study (Furmark et al 1999) with a 12-month prevalence of 8% (Myrick and Brady 2003).

There is a generalised sub-type which involves fear across different social situations, and a non-generalised sub-type where the fear is specific to one particular situation.

Social phobia often accompanies other disorders (co-morbidity). For example, the co-morbidity of alcohol abuse and dependence and social phobia varied from 8-56% (Myrick and Brady 2003). While Myrick and Brady (1997) diagnosed 13.9% of individuals with cocaine dependence entering a drugs treatment programme as showing social anxiety disorder ⁵⁶.

Zimmerman et al (2004) found higher rates in a Swiss study of 150 drug dependent patients - 20% for the generalised sub-type and 42.6% for the non-generalised sub-type. Women were more likely to show the former sub-type.

Table 13.1 compares the two studies of social phobia co-morbidity.

ELEMENT	MYRICK AND BRADY (1997)	ZIMMERMAN ET AL (2004)
Drug dependence	Cocaine dependent	Heroin, methadone or cocaine dependent
Sample	158 individuals entering drugs treatment trial	75 outpatients and 75 inpatients at detoxification unit
Diagnosis of social anxiety disorder	Structured interview using DSM-III-R criteria	Liebowitz Social Anxiety Scale (LSAS) (Liebowitz 1987) administered by clinician
Diagnosis of drug dependence	DSM-III-R criteria	DSM-IV

Table 13.1 - Key differences in two studies of drug dependence and social anxiety disorder.

⁵⁶ For most of the sample social anxiety disorder preceded the cocaine dependence.

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14. CYCLOID PSYCHOSES

Among the categories of mental disorders are the well-known ones, like bipolar disorder and major depression, and also the lesser-known ones; for example, cycloid psychoses.

These are discrete episodes of mania or depression with complete remission inbetween. They are sub-divided into anxiety-happiness psychosis (varying from depression and paranoid anxiety to extreme elation), confusion psychosis ((incoherence of thought either excited or depressed), and motility psychosis (emotions expressed through extreme movement or lack of movement). Cycloid psychoses show common symptoms with bipolar disorder, and schizophrenia-related psychoses (eg: schizophreniform disorder). In ICD-10 they are categorised as "acute transient psychotic disorders" and as "brief psychotic disorders" in DSM-IV (Pfuhmann et al 2004).

The question is whether cycloid psychoses are distinct diagnostic categories. One way to answer this question used by researchers today is the family study. Based on the assumption that disorders are inherited, the aim is to establish whether relatives of sufferers also suffer. So, sufferers of cycloid psychoses should have first-degree relatives (eg: parents) who also suffer from it, and not from bipolar disorder or schizophrenia-related psychoses, for cycloid psychoses to be a distinct category of mental disorder. Furthermore, sufferers of bipolar disorder, say, will have relatives with bipolar disorder and not with cycloid psychoses.

Pfuhmann et al (2004) performed a family study with a sample of in-patients at the Clinic for Psychiatry and Psychotherapy of the University of Wuerzburg, Germany ("sample-of-convenience" method; Ritsner et al 1991). Living adult first-degree relatives of 45 cycloid psychoses patients and 32 bipolar disorder patients (along with 27 controls) were traced and diagnostically interviewed (total: 431 relatives).

Relatives of cycloid psychoses patients were less likely to have bipolar disorder or schizophrenia-related psychoses compared to relatives of bipolar disorder patients, but the risk of relatives having cycloid psychoses was similarly low in both groups (table 14.1). The authors interpreted the results as evidence of cycloid psychoses as a separate disorder to bipolar disorder and to schizophrenia-related psychoses.

DISORDER	CYCLOID PSYCHOSES RELATIVES (n = 172)	BIPOLAR DISORDER RELATIVES (n = 153)	CONTROL RELATIVES (n = 106)
Schizophrenia- related psychoses	10.8	35.2	5.7
Bipolar disorder	4.5	31.4	1.9
Cycloid psychoses *	4.4	1.3	0

(* not significantly different)

Table 14.1 - Percentage of relatives.

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