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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.

CONTENTS

1. FOUR WAYS TO STUDY BIPOLAR DISORDER

Bipolar disorder covers individuals who show both extremes of mood - depression and mania - at different times, as opposed to unipolar depression or mania where the individual shows the one behaviour only [1].

1. Neuroimaging and brain physiology.

Ten individuals with a current mania episode in Canada underwent a Positron Emission Tomography (PET) scan along with ten controls. The mania sufferers were drug-naive or medication-free for many years, and it took the researchers four years to find such individuals as medication is so common. Less serotonin receptors in all areas of the cortex, especially the right hemisphere, were found in the bipolar disorder sample than the control adults.

Karine et al (2010) used diffusion tensor magnetic resonance imaging (DT-MRI), which shows the structure of the brain, with 28 individuals with bipolar disorder and 28 controls in northern England/southern Scotland. The former group were found to have white matter abnormalities, but this may have been due to past substance abuse (as half of the group had a history of illicit drug-taking).

2. Cluster randomised controlled trial and relapse prevention.

Recurrence or relapse rates are high (eg: over 50% in one year; Gitlin et al 1995). Thus the importance of relapse prevention, which teaches sufferers of bipolar disorder to recognise (and manage) the early warning signs of mania or depression. Sometimes relapse is prevented entirely, on other occasions there is an increase in the time until relapse.

Lobban et al (2010) reported that training professionals about relapse prevention, which could be passed on to sufferers, enhanced relapse prevention. The researchers used a cluster randomised controlled trial. Four National Health Service (NHS) trusts (covering 23 community mental health teams; CMHTs) in the north-west UK were randomised to treatment or control. Rather than randomising individuals, the whole CMHT (cluster) was randomised. Care co-ordinators oversaw a number of

[1] There is a recently developed debate as to whether borderline personality disorder may actually be a version of bipolar disorder (appendix 1A).

service users, and randomising individuals would be impractical as well as an ethical issue, particularly if one service users heard that another person was receiving a different level of help from the same professional. But there may be confounding variables between the CMHTs (eg: differences in "treatment as usual" or differences in service user profiles).

Care co-ordinators were either trained with twelve hours of enhanced relapse prevention (or not) to be used with ninety bipolar disorder sufferers (50 enhanced relapse prevention (ERP); forty treatment as usual (TAU).

The ERP group went an average of nine weeks longer before relapse than the control group (ie: 42 vs 33.5 weeks median survival time to recurrence).

3. Predicting bipolar disorder and longitudinal study.

Is it possible to predict adult bipolar disorder from bipolar-like (or sub-threshold) symptoms in childhood or adolescence?

Tijssen et al (2010) used data from the Early Developmental Stages of Psychopathology (EDSP) study, which followed 14-24 year-olds in the Munich area of Germany for ten years. Only a small number of the adolescents with bipolar experiences (at baseline) had bipolar disorder ten years later. Many of the experiences of the adolescents were transitory, but persistence (over three years) and quantity of symptoms predicted mental health service use as an adult. For example, individuals experiencing two or more manic symptoms in adolescence had a 10% risk of service use as an adult, and 8% with two or more depressive symptoms. Put another way, adolescents with the highest symptom loading, 15% of them sought mental health service help for bipolar symptoms as an adult. Altogether, 28% of the adolescents experienced manic symptoms and 60% depressive symptoms, but "only 1.1% met formal criteria for bipolar disorder at follow-up" (Parker 2010).

Parker (2010) raised methodological concerns, and questioned the predictive ability of these findings: "Until we know how to accurately stage and define the bipolar disorders (particularly in adolescents), we may assume that, in those who develop bipolar disorder later, some will show no early warning signs, some will meet full criteria in adolescence and earlier, some will show non-specific symptoms, some will show forme fruste [2] symptoms and states (eg: anxiety and eating disorders)..." (p88).

[2] Mild or incomplete form.

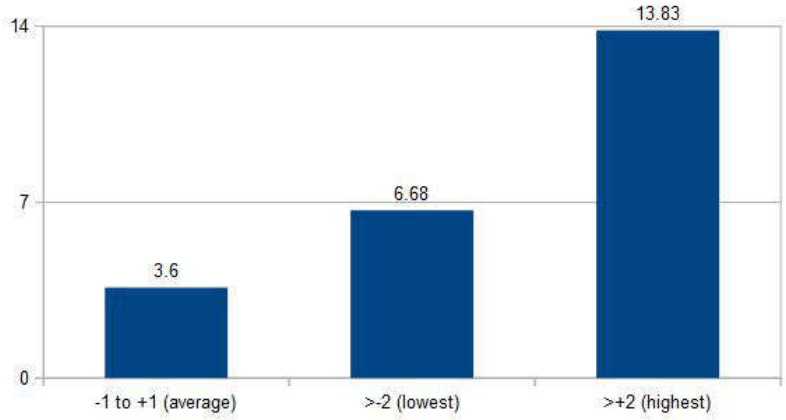
4. Associations and cohort study.

MacCabe et al (2010) found a strong association [3] between high-level school performance and later bipolar disorder, and a weak association between very poor grades and bipolar disorder among Swedish pupils.

The grades of all pupils finishing compulsory schooling between 1988 and 1997 (n = 713 876) (collected from National School Register) were linked to those who were subsequently hospitalised for psychosis (between the ages of 17 and 31 years old) (from hospital discharge register; up to end of 2003) (n = 280).

Individuals with the highest grades were four times more likely to be hospitalised for bipolar disorder, and those with the lowest grades twice as likely than the average grades (figure 1.1).

These were significant associations (not causations), so a common third factor might explain both school performance and bipolar disorder (eg: particular cognitive style; certain genes).



(Data from MacCabe et al 2010 table 2 p111)

Figure 1.1 - Incidence per 100 000 person-years of bipolar disorder based on IQ sore (z-score and standard deviations).

APPENDIX 1A - BORDERLINE PERSONALITY DISORDER AND BIPOLAR DISORDER

Tyrer (2009) argued that borderline personality disorder (BPD) is not really a personality disorder, but rather a mood disorder. Personality disorders are classed in DSMs as distinct from other mental disorders because

[3] When trying to establish if there is an association between two variables, time series regression studies is one technique used (appendix 1B).

their symptoms are trait-based. Tyrer (2009) felt that only two of the symptoms of BPD were trait-based - unstable and intense personal relationships, and persistent impulsivity (table 1.1).

At least 5 of following:

- Avoidance of real or imagined abandonment.
- Unstable and intense interpersonal relationships.
- Unstable sense of self.
- Impulsivity (eg: spending, binge eating).
- Suicidal behaviour, gestures, or threats, or deliberate self-harm.
- Mood instability.
- Extreme feelings of emptiness.
- Inappropriate, and intense anger.
- Transient, stress-related paranoid thoughts.

Table 1.1 - DSM-IV symptoms of BPD.

BPD can be hard to distinguish from bipolar disorder because of frequent mood swings. For Parker (2014), the age of onset is key: "Bipolar disorder can (rarely) present in childhood, the modal onset is late adolescence or early adulthood, and generally marked by a sharp 'trend break' (from absence of episodes to their overt presence). By contrast, those with BPD tend to show evidence of emotional dysregulation (for example anger, rage, impulsivity) from early years, and which evolves rather than showing a distinct 'onset'" (p252).

Furthermore, he said, elevated mood in bipolar disorder is characterised by euphoria, for example, whereas the "up" BPD sufferer still shows anger, hostility, and anxiety. "Another point of difference is that, following 'highs', individuals with bipolar disorder often experience guilt about indiscretions, which they 'own' with some shame. By contrast, individuals with BPD rarely feel guilt or shame following 'acting out' episodes, usually positioning them as the deserved and painful consequences of others' actions – and thus tend to blame others rather than accept any ownership" (Parker 2014 p252).

Bassett (2012) also saw the two conditions as separate for a number of reasons, including the sense of self - disrupted (in bipolar disorder) versus "emotionally noxious" (in BPD).

On the other hand, Parker (2014) asked: "Might BPD then be (at times) a spectrum depressive disorder – if not akin to 'atypical depression' – when shared features of interpersonal sensitivity, emotional dysregulation and abandonment preoccupations are respected? Both 'yes' and 'no' answers can be offered" (p253). Put together, Parker (2014) believed

that BPD could overlap with major depression, but was still distinct enough to make it separate to a mood disorder.

APPENDIX 1B - TIMES SERIES REGRESSION STUDIES

Time series regression studies require the collection of data over a period of time (ie: multiple observations of the same variables). Bhaskaran et al (2013) took the example of ozone level and deaths in London between 2002 and 2006. Data were collected each day for the two variables, to see if they were associated, along with potential confounders like temperature.

Time series regression is used to establish if day-to-day changes in the number of deaths (outcome) are explained by changes in the levels of ozone in the air. There needs to be control for seasonal variations (eg: ozone highest in summer and lowest in in winter while deaths slow the opposite pattern), for example. This can be done by dividing the data into specific time intervals (eg: calendar month), or by mathematically modelling the trend (eg: Poisson model). This should leave a straight-line which summarises the data, and daily measures can be seen in relation to that.

Bhaskaran et al (2013) calculated a 0.7% increase in deaths for each 10 microgram/m^3 rise in ozone levels ($p<0.001$) when controlling for seasonal variations. But when daily temperature is added to the model, the increase in deaths is 0.3% (not significant). "This suggests that the initially estimated positive association between current ozone level and mortality risk was largely explained by confounding by temperature" (Bhaskaran et al 2013 p1192).

More sophisticated time series regression analysis might include a time lag (eg: the ozone level seven days before the death) to establish delayed effects.

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2. IMMIGRANTS AND REFUGEES AND MENTAL DISORDERS

Worldwide, there are 44 million individuals "forcibly displaced because of conflict and persecution" (of which 31-55% of them are under eighteen years old) (Vostanis 2014). These include international refugees, internally displaced individuals, those awaiting asylum claims, living in resettlement camps [4] or in cities (ie: heterogeneous population) (Vostanis 2014).

There is also confusion between economic migrants and refugees. Though the latter have increased risk of post-traumatic stress disorder, anxiety, and depression, there should be caution about "overattributing all mental health presentations to past experiences" (ie: not to forget post-immigration stressors [5]) (Vostanis 2014).

Migration generally is seen as a risk for the development of mental disorders, particularly psychotic and mood disorders. For example, schizophrenia is over four times greater in the migrant population (first and second generation) than among the native population (eg: Bourque et al 2011), and one and half times greater for depression and bipolar disorders (eg: Swinnen & Selten 2007).

The prevalence of mental disorders also varies between migrant groups in the same country. For example, in The Netherlands, where there are immigrants from Turkey, Morocco, Surinam, and the Dutch Antilles, schizophrenia is highest in second-generation Moroccan-Dutch individuals (Selten and Sijben 1994), while mood disorders are greatest among the Turkish sub-group (Selten et al 2012).

A selection of hypotheses have been proposed for the differences between migrant and indigenous individuals in prevalence of mental disorders. An early theory was the "selective migration" hypothesis (Odegaard 1932), which suggested that individuals with schizophrenia and bipolar disorder are more restless and thus likely to migrate. It was based on Norwegian-born individuals in Minnesota, USA. This hypothesis is now discarded as higher rates of mental disorders appear in second-generation immigrants (ie: grandchildren of original migrants) than immigrants themselves (van de Beek et al 2014).

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[4] Llosa et al (2014) investigated the prevalence of mental disorders among adult refugees from Palestine living permanently at the Burj el-Barajineh camp (opened in 1948) in the suburbs of Beirut, Lebanon. A random selection of 194 individuals were interviewed by clinical psychologists in 2010. The prevalence of any current mental disorder was 19.4%, with depression as the most common condition.

[5] Eg: relative poverty, absence of healthcare, and lack of social networks (Llosa et al 2014).

This is now taken as evidence for social factors in the new country being important - eg: discrimination; lack of social support; social isolation (van de Beek et al 2014).

METHODS OF STUDY

Migrant groups are not easy to study using traditional sampling techniques because of their "hidden" nature (eg: no list of immigrants) (van de Beek et al 2014). Thus researchers have to use novel methods.

1. van de Beek et al (2014) outlined a study to be carried out on the social factors and mental disorders among Moroccan-Dutch individuals in The Netherlands who visited one particular ethnic website. The online survey placed there included questions about mood, anxiety, and psychotic symptoms (eg: "I have seen things that other people apparently cannot see"), and social factors (eg: "How many people are so close to you that you can count on them if you have serious personal problems?"). The aim is recruit one thousand respondents.

2. Pascual et al (2008) analysed 11 578 consecutive admissions to a psychiatric emergency service in Barcelona, Spain, for differences between indigenous residents and immigrants. Five sub-groups of immigrants to Spain were distinguished - North African, sub-Saharan African, South American, Asian, and Western countries. There were a number of significant differences in the nature of the referrals (table 2.1).

DISORDER	DIFFERENCES
Depression	Less North and sub-Saharan African, and Asian.
Anxiety	More South American.
Psychosis	More North and sub-Saharan African, and Asian.
Disruptive behaviour	More North African and Asian.
Drug misuse	Less South American.

Table 2.1 - Significant differences of immigrant groups to indigenous referrals.

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3. IQ AND HOMICIDE VICTIMOLOGY

Individuals with higher IQ are less likely to be victims of homicide than those with lower IQ. The risk is half less for men (Batty et al 2008) [6] [7].

Batty et al (2008) performed a cohort study using the detailed records kept in Sweden. A cohort of 968 846 men aged 18-19 years old was used. They undertook an IQ test during military conscription, and the scores were divided into three groupings (lowest, highest, average). The individuals were followed for over twenty years to see who was murdered, of which there were 191 individuals. After adjustment for variables like socio-economic status and education, if the risk of homicide was standardised as one for the lowest IQ grouping, then it was 0.48 for the middle grouping and 0.27 for the highest IQ. This is significant at $p<0.001$. Thus, there is a negative correlation between IQ and homicide victims.

Batty et al (2008) suggested a number of reasons for the findings:

i) High IQ usually means the verbal skills to negotiate a peaceful resolution to disputes (rather than violence and the risk of death).

ii) Individuals with higher IQ may have higher income etc and thus live in safer neighbourhoods.

iii) Individuals with high IQ are better at assessing risks and less likely to put themselves in a situation where homicide could happen.

iv) The majority of homicide victims in Sweden were intoxicated. Individuals with high IQ may be less likely to become intoxicated (because of an awareness of the health risks).

v) Perpetrators of homicide are more likely to have a lower IQ, and so find similar victims.

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[6] More generally, the "lower a person's measured intelligence, the greater that individual's risk of living a shorter time, developing both mental and physical ailments later in life and dying from cardiovascular disease, suicide or an accident" (Deary et al 2011 p50). But "being intelligent may not be the key ingredient for longevity. Instead, acting and deciding as intelligent people do may be the crucial factor" (Deary et al 2011 p55).

[7] Other groups also have higher mortality risk generally (eg: individuals with schizophrenia; appendix 3A), and individuals with mental disorders have a higher risk of accidental death (appendix 3B).

APPENDIX 3A - SCHIZOPHRENIA

Brown et al (2010) followed 370 adults with schizophrenia living in the community, who contacted psychiatric services in 1981-2. These individuals from Southampton in England were followed to the end of August 2006, by which time 164 had died. This was a standardised mortality ratio (SMR) of 289. The SMR is calculated by dividing the number of deaths observed by the number of deaths expected, and multiplying by 100. A SMR of 100 is the norm. Thus the number of deaths within the cohort of adults with schizophrenia was nearly three times higher than expected. The major cause of natural deaths was cardiovascular disease, which "can probably be attributed to the effects of cigarette smoking" (Brown et al 2010) [8]. Other risk factors included poor diet, little exercise, obesity, and relative poverty (Brown et al 2010).

APPENDIX 3B - ACCIDENTAL DEATH

Individuals with mental disorders have an increased risk of accidental death (which is higher than the suicidal risk). This is due to factors like medication self-effects, symptoms of the disorder (eg: fatigue, poor concentration), and risk-taking behaviour (Crump et al 2013) [9].

Crump et al (2013) used the comprehensive national data of Sweden. The study population was all adults aged twenty years and above at the beginning of 2001 (n = 6.9 million), and death from accidents between that date and the end of 2008 (ie: eight-year follow-up) [10].

During this period, there was a total of 22 419 deaths from falls, transport accidents, and accidental poisoning (0.3% of population). Among individuals with mental disorders, it was 0.9% of population (versus 0.6% for suicides).

After adjusting for sociodemographic variables like age, marital status, employment status, and education level, men with mental disorders were over six times more likely than the general population to suffer accidental death, and women over five times more (figure 3.1). The type of accidental death also varied compared to the general population (figure 3.2).

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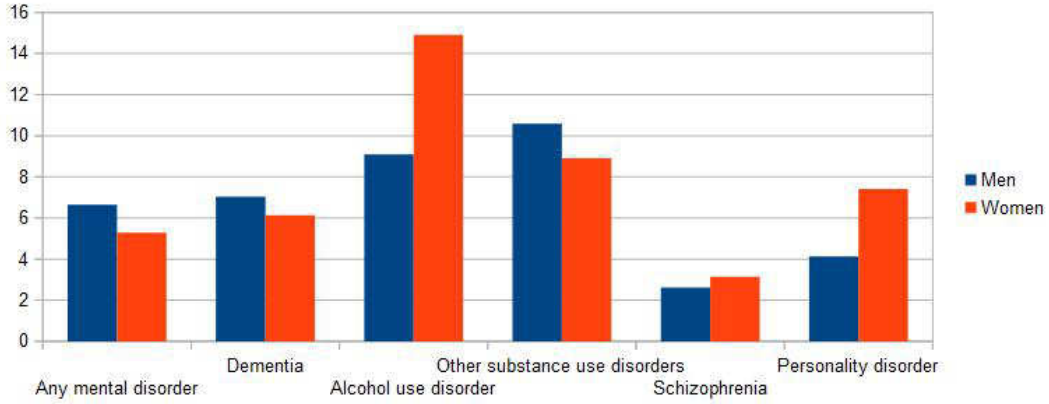
[8] At the start of the study, 73% of the cohort were smokers, which was twice the general population of 1981-2 (Brown et al 2010).

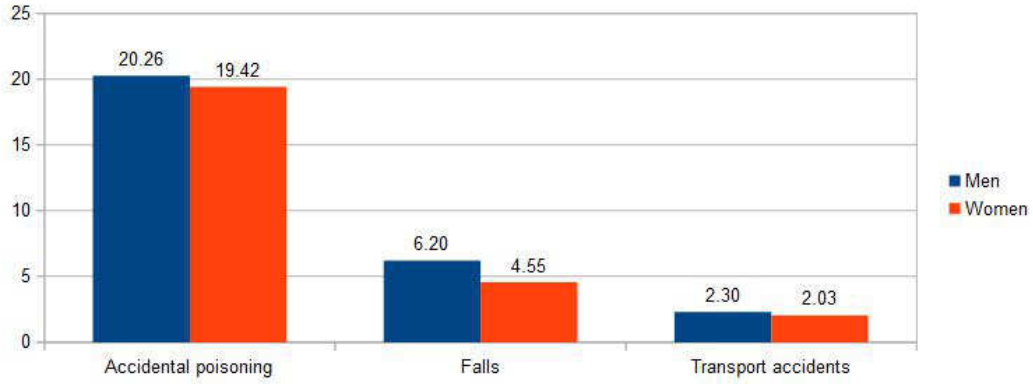
[9] "High-risk behaviour associated with some mental disorders may occur on a continuum from sub-intentional to intentional. Indifference to death, in contrast with a desire to die, may result in sub-intentional harm and risk of accidental death" (Crump et al 2013 p297).

[10] It was a prospective study.



(Data from Crump et al 2013 table 1 p299)

Figure 3.1 - Adjusted hazard ratio for accidental death based on selected mental disorder (where 1 = general population risk).



(Data from Crump et al 2013 table 3 p300)

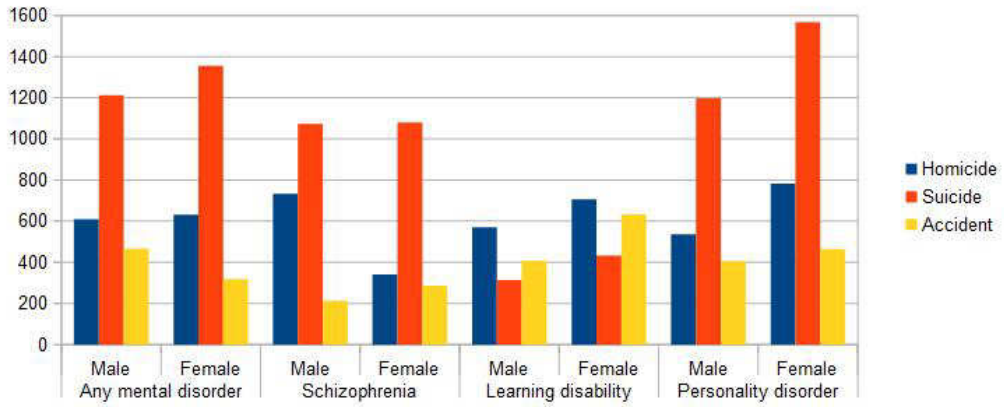
Figure 3.2 - Adjusted hazard ratio for type of accidental death for individuals with mental disorders (where 1 = general population risk).

This study only included individuals officially diagnosed with mental disorders (ie: seen by psychiatric authorities), and thus missed cases not seeking help. It was also dependent on the accurate classification of deaths by officials. Crump et al (2013) admitted: "Suicide is a stigmatising and distressing verdict for families and there can be pressure to attribute deaths to accidents rather than suicides" (p301).

The greatest strength of the study was the size of the sample, which was much larger than previous studies that used psychiatric hospital cases and after their release, small community samples (with similar design to

this study), or case-controls (ie: those who had experienced accidental death and worked backwards to see if suffering from a mental disorder - retrospective study).

Using the data from over 70 000 individuals on the Danish Psychiatric Care Register (1973-1993), Hiroeh et al (2001) found that 25% of deaths were "unnatural" (homicide, suicide, accident). The rate for individuals with any mental disorder were much higher than the general population (figure 3.3).



(Data from Hiroeh et al 2001 table p2111)

Figure 3.3 - Standard mortality rate (where 100 = general population rate) for homicide, suicide, and accidental deaths.

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4. EXPERIENCES OF HEARING VOICES

Auditory verbal hallucinations (AVHs) (hearing voices) can be a distressing experience, but it depends on a number of aspects of how the individual makes sense of the voices (Hartigan et al 2014) [11]:

a) Voice Dominance - eg: dominant, insulting voice(s).

b) Hearer Distance - whether the hearer avoids communication with the voice(s).

c) Voice Intrusiveness - the voice(s) imposing itself on the hearer.

Distress with AVHs is related to perceiving the voice(s) as domineering and bullying, trying to avoid communication with it, and feeling that it is intrusive (Sorrell et al 2010).

The experience of AVHs can change over time - for example, in frequency and severity. However, the perceived intention of the voice(s) (ie: malevolent or benevolent) did not change over six months among sixteen individuals (Csipke and Kinderman 2006).

Hartigan et al (2014) confirmed this finding in a twelve-month study with eighteen individuals in southern England. The beliefs about the voices' intention were measured by the Beliefs about Voices Questionnaire-Revised (BAVQ-R) (Chadwick et al 2000) (table 4.1), and their severity by the Psychotic Symptoms Rating Scale - Auditory Hallucinations Scale (PSYRATS-AH) (Haddock et al 1999) [12]. The Voice and You (VAY) scale (Hayward et al 2008) was also completed. This is a 28-item self-report measure of the relationship with the voice(s) (eg: "My voice tries to get the better of me"; "My voice helps me make up my mind"). All questionnaires were completed twice at twelve months apart.

Overall, the beliefs about the voices did not change over the study period (ie: BAVQ-R scores not significantly different between baseline and follow-up), though the perception about their benevolence did decline. The scores on the VAY scale and the PSYRATS-AH did not change over time either. Hartigan et al (2014) concluded: "The results of the current study suggest that styles of interrelating between voice and hearer are relatively stable and enduring, as are beliefs about the voices' malevolent intent and power, in the absence of

[11] Romme and Escher (1989) found that 15% of 173 voice-hearers reported their AVHs as positive.

[12] This has eleven items measuring characteristics of voices like frequency, duration, severity, loudness, degree of negative contact, and controllability.

- 35 items covering aspects of AVHs:

- Malevolence (eg: "My voice is punishing me for something I have done").
- Benevolence (eg: "My voice wants to protect me").
- Omnipotence (eg: "My voice is very powerful").
- Resistance (eg: "My voice frightens me").
- Engagement (eg: "My voice reassures me").

- Each item is score from 0 (disagree) to 3 (agree strongly); giving a total score range of 0-105.

- Omnipotence is a key aspect for Chadwick et al (2000), and they validated the six items specific to that characteristic with seventy-three voice-hearing individuals in southern England [13]:

- "My voice is very powerful" (86% "agree slightly" (2) or "agree strongly" (3)).
- "My voice seems to know everything about me" (79%).
- "I cannot control my voice" (75%).
- "My voice rules my life" (63%).
- "My voice makes me do things I really don't want to do" (47%).
- "My voice will harm or kill me if I do disobey or resist it" (38%).

Table 4.1 - Details of BAVQ-R [14].

specific interventions. Our findings suggest that negative relating patterns and beliefs about malevolence and omnipotence may serve as ongoing vulnerability factors for distress" (p122).

In terms of limitations of their study, Hartigan et al (2014) admitted that "the majority of participants were attending Hearing Voices Groups [15]. These participants therefore possibly represent a specific subset of voice-hearers, skilled at thinking and talking about their voices, and our findings hence lack

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[13] These are new items added to the original BAVQ (Chadwick and Birchwood 1995), which had thirty items answered "yes" or "no".

[14] The BAVQ-R does not assess the form or content of the AVHs (Chadwick et al 2000).

[15] Patient or service user involvement (and their families and carers) in mental health research is beneficial for both the participants (eg: sense of well-being), and the researchers. Ennis and Wykes (2013) found that more patient involvement was associated with success in recruiting patients. The researchers analysed 374 studies of mental health in England. Patient involvement was rated at five levels from patient consultation (lowest) to patient-controlled studies (highest). Higher patient involvement was significantly associated with meeting recruitment targets for participants. For example, patient-controlled studies were four times more likely to achieve recruitment targets than patent consultation studies.

Ennis and Wykes (2013) suggested the following reasons for the findings: "(a) the language used in materials such as information sheets is more appealing or easier to understand for patients because of vetting by other patients; (b) patients contribute insight into the realities of living with a mental health problem and therefore understand which designs will be the least burdensome; and/or (c) patients are more willing to participate in research that they know has involved other patients, as the principle of patient involvement is in itself appealing" (pp384-385).

However, Mental Health Research Network (2012) did find that there can be poor experiences for both patients and researchers, mainly due to lack of understanding of how to involve patients, as well as the benefits.

generalisability to all voice-hearers in clinical services. A focus on the hearers' predominant voice may also have been a limitation as several participants reported that they heard multiple voices. Some participants reported that their voices were quite different and therefore focusing on one voice may have discounted or distorted important information" (p122).

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5. SIX ISSUES WITH UNDERSTANDING CO-MORBIDITY OF MENTAL DISORDERS

Co-morbidity is used in a number of ways to include the overlap of symptoms, syndromes, or diagnostic groups as well as mental disorders with physical (somatic) conditions. "Consequently, there is the claim that co-morbidity might simply be an artefact of our currently imperfect diagnostic conventions and its explicit diagnostic criteria... that split previously broader diagnostic classes 'artificially' into smaller subsets" (Fava et al 2014 p93).

A working definition of co-morbidity could be "the presence of more than one specific disorder in a definite period of time" (Wittchen et al 1996 quoted in Fava et al 2014). But does that a mean primary disorder with a secondary one or two equally assessed disorders?

There are a number of issues to consider with co-morbidity of mental disorders.

1. The widening of diagnostic criteria/categories (eg: sub-threshold conditions) are more likely to find overlaps.

2. The side effects, including withdrawal, of psychotropic drugs may produce new symptoms (eg: anxiety; insomnia). Along with rebound of symptoms and supersensitivity drug-induced disorder, these can be called "iatrogenic co-morbidity" (ie: illness caused by the treatment) (Fava et al 2014). Chouinard et al (1983) referred to "iatrogenic withdrawal induced disorder type" where the rebound anxiety from withdrawal of benzodiazepines (tranquillisers) taken for anxiety is worse than the original anxiety. Kales et al (1978) reported this for insomnia, which they called "iatrogenic psychiatric disorder induced by withdrawal of psychotropic medications". While long-term use of anti-psychotics can produce dopamine supersensitivity and movement disorders (Fava et al 2014).

3. The social and cultural context of mental disorders. For example, could the stigma (appendix 5A) in a society related to a particular mental disorder produce other symptoms (eg: anxiety) that are then diagnosed as a co-morbid disorder?

4. The treatment of co-morbidity. In some cases, the treatment of one disorder also helps the other (eg: CBT for panic disorder with agoraphobia also reduces co-morbid depressive symptoms), while not in others (eg: treatment of depression does not necessarily reduce co-morbid anxiety). On the other hand, co-morbidity may

reduce the effectiveness of treatments (eg: anti-depressants less effective for depression with co-morbid pain symptoms) (Fava et al 2014).

5. Fava et al (2014) pointed out a number of methodological issues in studies of co-morbidity:

i) Conceptual level - Co-morbidity is better used for two clearly defined disorders, and the term "co-occurrence" for associations between symptoms.

ii) Units of content - For example, differences in diagnostic criteria between studies make simple comparisons of percentages of co-morbidity of limited value.

iii) Time window - The use of different time periods (eg: 3 months; 12 months) again limit the comparability of studies.

iv) Assessment method - Different studies have different methods of diagnosing disorders (eg: unstructured or structured interviews).

v) Design and analysis - Studies vary on sampling procedure, for example, or whether there is a longitudinal or cross-sectional design.

6. What is the relationship between co-morbid mental disorders? Fava et al (2014) suggested four possibilities:

a) Disorder A precedes (and increases the risk of) disorder B - eg: anxiety disorders increase the risk of co-morbid depression.

b) Disorder A or B predispose for the development of the other - "not much empirical support for this" (Fava et al 2014 p96).

c) Common risk factor X for disorder A and disorder B - eg: the personality trait of neuroticism is risk for anxiety disorders and mood disorders.

d) Risk factors X and Y cause disorder A, and risk factors X and Z cause disorder B - eg: childhood adversity and traumatic experience as adult leads to PTSD, while childhood adversity and general stress is linked to depression.

APPENDIX 5A - STIGMA AND PREVENTION

The potential benefits from preventing mental

illnesses are considerable, both in terms of illness burden for people with mental illness, their families and carers as well as in socio-economic terms. Evidence so far suggests that prevention can reduce the incidence of mental disorders and can be cost-effective" (Rusch and Thornicroft 2014 p249).

Prevention strategies can be primary (focusing on risk factors), secondary (concentrating on the early stages of the disorder), or tertiary (preventing relapse and aiding recovery). Or they can be universal (whole population - eg: alcohol misuse), selective (aimed at persons at risk - eg: children of parents with mental illness), or indicated (pre-diagnosis or sub-threshold - eg: early signs of eating disorders) (Rusch and Thornicroft 2014).

Stigma and discrimination can limit prevention in different ways (Rusch and Thornicroft 2014):

i) Prevention strategies are not implemented because of "structural discrimination" ("rules and regulations in society that intentionally or unintentionally disadvantage people with mental illness, for example by the amount of resources dedicated to mental health services"; Rusch and Thornicroft 2014) or "public stigma" (where individuals in society are prejudice or discriminate) - eg: universal programmes.

ii) Individuals do not participate in prevention programmes due to fear of public stigma or "self-stigma" (where individuals internalise negative stereotypes) - eg: selective or indicative programmes.

iii) Prevention strategies are implemented but do not succeed because of stigma and discrimination - eg: universal prevention programmes.

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6. CAPACITY TO CONSENT, COERCION AND COMPETENCE

The concept of consent is linked to the idea of the rights of the individual within society, which are attributed to philosophers like Immanuel Kant in the 18th century. In the twentieth century, this idea is developed in the Universal Declaration of Human Rights (1948), for example (Hilton 2007).

In terms of consent to treatment for mental illness in England and Wales specifically, this appeared in the Mental Health Act 1930 for non-detained individuals. Formally, for detained individuals, they had to wait until the Mental Health Act 1983 (hilton 2007). "It has been recognised for decades that, although consent to treatment criteria apply to all medical and surgical interventions, in the context of mental illness there may be specific difficulties when the patient is unable to fulfil the criteria to make such decisions because of the very mental illness which requires treatment" (hilton 2007 p219).

This is important because the Mental Health Act 1959 was a key legal framework. It was "guided by the philosophy that, whenever possible, care should be provided without the use of compulsion... However, in terms of treatment, it was assumed that detained patients admitted for treatment could be treated against their will. Although unwritten, it was considered good practice to discuss treatment in these circumstances with the patients' relatives, or to seek a second opinion from a colleague. It is unclear from the literature precisely when these good practice recommendations were put into effect, and quite how controversial or widely implemented they were" (Hilton 2007 p219).

COERCION

Coercion in the dictionary is defined as "to constrain or restrain by the application of superior force, or by authority resting upon force" (quoted in Molodynski et al 2014).

Molodynski et al (2014) pointed out that "coercion has always existed in psychiatry... [and] the 'move into the community' in many countries over recent decades and the evolution of community services have substantially altered the focus of coercion" (p2).

Furthermore:

> The power to compel (ie: to insist on containment and/or treatment) has existed in mental health services in most countries for many years through legislation. In relation to mental health treatment,

> 'compulsion' generally refers to forced treatment under legitimate legal authority, ideally subject to scrutiny and with rights of appeal. Terminology in the area is extremely complex. There are both 'subjective' and 'objective' aspects to coercion. The latter may involve 'threats' or the actual use of compulsion while the former involves the perception of threats of the use of force if one does not act according to the wishes of another. Perceived coercion [16] may occur in the absence of 'objective' coercion but the two usually occur together (Molodynski et al 2014 p3).

Coercion is viewed differently depending on the professional group. For example, among clinicians, the focus is upon the effect of coercion on symptoms and recovery, while legal professionals concentrate of civil liberty issues (Molodynski et al 2014). The right of the "patient" to refuse treatment is confronted by the failure of clinicians to help those suffering seen as "active neglect" (Molodynski et al 2014). However, most of the academic literature on the subject comes from a small number of developed countries. "In less affluent societies, treatment for some may not be available in any meaningful sense. This effectively removes the right of an individual to make a positive choice to have (or not to have) treatment for their condition and is therefore restrictive. This restriction of choice due to lack of resources may be perceived as coercive by the individual concerned" (Molodynski et al 2014 p3).

A number of issues related to coercion can be distinguished:

- Whether admission to a psychiatric hospital is voluntary or compulsory.
- The use of chemical and physical restraints in such hospitals.
- Powers to compel acceptance of support/treatment in the community.

DECISION-MAKING CAPACITY

"Decision-making capacity" (or competence) (DMC) by a patient is the ability to make their own medical decisions, and includes "the abilities to understand (ie: intellectually absorb) the relevant information and to communicate a choice" (Kim 2013 p403).

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[16] The MacArthur Admission Experience Survey (eg: Monahan et al 1995) was developed in the USA to measure perceived coercion. It includes items like, "It was my idea to come into hospital", and "They said they would make me come into the hospital" (Molodynski et al 2014). While the "coercion ladder" (Iversen et al 2002) uses a scale of 1-10 to rate the patient's feelings.

The MacArthur Competence Assessment Tool - Treatment (MacCAT-T) is a semi-structured interview technique used to measure DMC in practice (Grisso and Appelbaum 1998). It covers (Owen et al 2013):

i) Understanding relevant information - this is affected by memory, language and attention impairments, and altered emotional states, and thought disorders.

ii) Appreciating that information is relevant - "the ability to apply the information abstractly understood to his or her own situation" (Grisso and Appelbaum 1998 quoted in Owen et al 2013).

iii) Reasoning about such information - "to engage in logical processes when using the information that they have understood and appreciated in arriving at a decision" (Grisso and Appelbaum 1998 quoted in Owen et al 2013) [17]. But Owen et al (2013) noted: "Caution needs to be exercised in interpreting this as a procedural reasoning standard, as there is plenty of evidence to suggest healthy human decision makers do not conform reliably to formal reasoning" (p465).

iv) Exposing a choice based on previous steps.

There is debate about DMC for individuals with mental disorders. Owen et al (2013) compared the DMC of 164 admissions to an acute psychiatric ward [18] and 125 to a medical ward [19] in two nearby hospitals in south London. Using the MacCAT-T, understanding, appreciation, and reasoning were all scored (0-4, 0-6, or 0-8).

In both groups, individuals who scored low on understanding (ie: below the mid-point on the scale) were seen as lacking DMC. A high appreciation (ie: above the mid-point) was a better predictor of DMC in the psychiatric group, but a high reasoning score was more common in the medical group with DMC.

To summarise: "Among those with good understanding, the appreciation ability had more salience to DMC for treatment in a psychiatric setting and the reasoning ability had more salience in a medical setting" (Owen et al 2013 p461).

Lack of understanding was key to lack of DMC, but understanding by itself was not enough. For example, just over one-third of the psychiatric group had good understanding but lacked DMC (Kim 2013).

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[17] Example probe from MacCAT-T for reasoning: "You think that treatment X might be best. Tell me what it is that makes that seem better than the others" (Owen et al 2013 p466).

[18] Psychosis was the most common diagnosis.

[19] One-quarter had cognitive impairment (eg: dementia).

LACK OF COMPETENCE AND COMPLAINING

Jingree and Finlay (2013), using the critical discursive approach [20], interviewed eleven adults with mild-to-moderate learning disabilities about their residential care home and day-centre services in south-east England. The verbatim transcripts were analysed for underlying interpretative repertoires related specifically to complaining about the services.

It was clear that complaining about a service that the individual depended upon was problematic [21]. For example, "Freddie" admitted: "Got to be careful what you say", as he had previously been given an ultimatum to stop complaining by staff. Thus, individuals who complain have to walk a tightrope. Jingree and Finlay (2013) argued that this was done in the use of repertoires of the competence of the speaker, and staff members as controlling. The researchers gave three examples of how this was done.

i) Position of incompetence - "Izzy" emphasised what she could not do because of her learning disability (position of incompetence) as the basis of knowing what she could do (position of competence) and how staff pressed her to do more.

ii) Position of competence - "Freddie" described how he knew best what he wanted and the staff restricted his behaviour.

iii) Tempering dissatisfaction - For example, one interviewee "expresses dissatisfaction with one aspect of the service while simultaneously declaring that she is satisfied with other aspects of the service. Therefore, she limits her complaint and the risk of harming her relations with all of the staff who support her. This seems particularly important, given her orientation to the dangers of complaining..." (p265).

The key point for Jingree and Finlay (2013) was how do disempowered individuals make complaints. They said: "In making complaints, the speakers managed their own identities in ways that implicitly or explicitly positioned themselves as competent. They also attended to

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[20] The critical discursive approach focuses on how identity is constructed through social interactions. During interactions individuals make use of ideas in society about the topics of conversation (called discourses or interpretative repertoires), and these combine with subject positions to produce identity. The preferred method to study this process is discourse analysis.

[21] Jingree and Finlay (2013) observed: "Two aspects of the context make this particularly interesting: (1) the complaints refer to social care services in which those who are complained about have greater institutional power than those making the complaints, and (2) the speakers' institutional identities mean there is a danger their judgements might be questioned on the basis of their reliability" (p257).

the particular power relations of institutionally based social care by using staff voices to corroborate their accounts, by contrasting the practice they disapproved of with the practice of other people in positions of institutional authority (staff members and parents), and by referring to general service principles that contradicted the staff behaviour. In making complaints about people who occupy positions of greater institutional power than themselves, then, speakers drew on established authorities for corroboration" (p267).

Put another way, there is "the wider issue of how identity is negotiated in accounts of unequal power relations..., and how identities with supposedly fixed traits (here limitations in social judgment and competence) can be constructed flexibly to argue against disempowerment" (p257).

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7. NON-ADDICTIVE DRUG USE

Drug use is widespread - eg: 8% of Americans have consumed an illicit drug, and over 50% are regular consumers of alcohol, while over 80% of Europeans drink alcohol daily (Muller and Schumann 2011). There is a lot of public concern, thus, about drug addiction. "Epidemiological data show, however, that the majority of people who consume psychoactive drugs [22] with an addiction potential are not addicts and will never become addicted" (Muller and Schumann 2011 p293). For example, of the many alcohol drinkers, less than one in six are classed as addicts (Muller and Schumann 2011).

In evolutionary terms, there is a "paradox of drug reward" (Sullivan and Hagan 2002). The basis of many drugs is plant toxins (eg: nicotine) which evolved as defences against plant consumption. Why would such toxins (to deter herbivores) evolve to be rewarding to humans? One answer is dosage. "Drugs like cocaine induce euphoria only at low to medium doses. At higher doses, cocaine induces highly aversive paranoia and behavioural stereotypes... Drugs with a low euphoria component, such as nicotine or caffeine, are voluntarily consumed, usually at low, non-toxic doses... It is important to realise that the doses in which humans and animals voluntarily consume psychoactive drugs are usually below the acute toxic range..." (Muller and Schumann 2011 p294).

At low dosages, there are benefits to drug consumption - for example, in aiding achieve personal goals. Thus, psychoactive drugs are instrumentalised. "Drug instrumentalisation" has two steps: "(1) the seeking and consumption of a psychoactive drug in order to change the present mental state into a previously learned mental state, which then allows for (2) better performance of other, previously established behaviours and better goal achievement" (Muller and Schumann 2011 p295) [23]. For example, the consumption of coffee (caffeine) to improve alertness and aid in the goal of driving home late at night.

Muller and Schumann (2011) argued that there is an evolutionary basis to non-addictive drug use [24] - namely, that low dosage aids in survival (and reproduction), the ultimate goals of evolutionary behaviour [25]. Drug use

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[22] A psychoactive drug is defined as a single chemical compound that interacts with the functioning of the central nervous system, and produces changes in subjective experience and/or behaviour (Muller and Schumann 2011).

[23] Wu (2011) criticised the lack of inclusion of social factors and context in the model.

[24] Sullivan and Hagan (2011) questioned whether there is an evolutionary basis.

[25] Foxall and Sigurdsson (2011) preferred to see drug use as consumer behaviour that could be explained with the behavioural perspective model (BPM).

affects relevant behaviours for this (Muller and Schumann 2011):

- Improving social interaction - eg: alcohol reduces social inhibition and social anxiety, and increases talkativeness and social bonding. Being part of a group is highly beneficial to survival.
- Facilitate sexual behaviour - eg: alcohol.
- Improve cognitive performance - eg: caffeine.
- Aid in coping with stress - eg: cannabis.
- Self-medication for mental problems.
- Provide new sensory stimuli - eg: LSD.
- Produce euphoria and "highs".

Muller and Schumann (2011) admitted that despite the benefits of non-addictive drug use, "the instrumentalisation of psychoactive drugs comes at a price, which ultimately qualifies it as a risky behaviour... Severe damage to the brain and body peripheral organs have been documented, for example, from alcohol..., MDMA...; nicotine..., androgenic-anabolic steroids..., psychostimulants..., and cannabis... Many psychoactive drugs enhance pre-existing psychopathologies in vulnerable individuals..." (p308). As well as the risk of addiction [26].

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[26] Swendsen and Le Moal (2011) were concerned about the health policy implications of drug instrumentalisation theory.

8. SMOKING AND MENTAL HEALTH

What is the relationship between smoking and mental health? On the one hand, many smokers report mental health benefits, like relaxation and less anxiety (whether the individual is diagnosed with a mental disorder or not). On the other hand, there is a strong association between smoking and poor mental health, and smokers with a mental disorder diagnosis are heavier smokers (Taylor et al 2014).

Taylor et al (2014) observed "Although smokers with and without mental disorders think that smoking provides mental health benefits, they might be misattributing the ability of cigarettes to abolish nicotine withdrawal as a beneficial effect on mental health. Smokers experience irritability, anxiety, and depression when they have not smoked for a while, and these feelings are reliably relieved by smoking thus creating the perception that smoking has psychological benefits, while in fact it is smoking that caused these psychological disturbances in the first place".

Taylor et al (2014) saw this belief as a barrier to smoking cessation, and they performed a review of the evidence. Twenty-six longitudinal studies that assessed mental health while smoking and after cessation were found.

Quitting smoking was associated with a significant reduction in anxiety, mixed anxiety and depression, depression, and stress as well as significant improvements in psychological quality of life and positive mood. So, stopping smoking is beneficial to mental health, and there was no evidence that this finding was a product of the poor methodological of the studies. Twenty studies were rated as high quality on the Newcastle-Ottawa Scale (NOS) (Wells et al 2010) [27].

This review using observational data was not able to establish causality (ie: smoking cessation caused mental health improvements) because it was possible that mental health improvements caused smoking cessation, or another factor (eg: positive life events) caused both the smoking cessation and mental health benefits. Taylor et al (2014) admitted: "Observational data can never prove causality, but almost all we know about the harms of smoking and the benefits of cessation derive from observational studies as randomised trials to examine this have insurmountable ethical and practical difficulties".

Taylor et al (2014) suggested that there was no single factor that could explain both outcomes, and some

[27] Studies are given points up to nine for selection of participants (eg: representativeness), the comparability of cases and controls, and the nature of exposure to the independent variable or factor.

studies encouraged smoking cessation among the participants. So the view that smoking cessation caused mental health improvements seemed reasonable. Furthermore, a plausible biological mechanism is provided. Quitting smoking stops the "withdrawal cycle" which is the fluctuation in nicotine in the brain between cigarettes. This cycle produces negative mood and anxiety, for example, as time since last cigarette increases. Ending the withdrawal cycle, thus, improves mental health.

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9. THE CHANGING WORLDS OF DSM - TWO EXAMPLES

The "Diagnostic and Statistical Manual of Mental Disorders" (known as DSM) published by the American Psychiatric Association (APA) listing the symptoms and diagnostic criteria of mental disorders dominate the understanding of mental illness. DSM is intermittently updated with a new edition (the latest is DSM-5 in 2013). There are sometimes quite major changes between the editions which challenge the idea that DSM simply describes mental disorders rather than constructing them as argued by its critics (Brewer 2002).

Here are two examples of conditions that have changed during the history of DSM.

DEPENDENT PERSONALITY DISORDER

Dependent personality disorder (DPD), though not called that initially, has its origins in the oral stage of Sigmund Freud's theory of psycho-sexual development. In DSM-I, in 1952, there is no separate category, but a passive-dependent type of passive-aggressive personality, shown by "helplessness, indecisiveness, and a tendency to cling to others as a dependent child to a supportive parent" (APA 1952 quoted in Disney 2013).

DSM-III (APA 1980) introduced the separate category of DPD with three key criteria - "(1) allows others to assume responsibility for important life decisions, along with passivity in interpersonal relationships, (2) subordinates one's own needs to those of persons upon whom one depends, and (3) lacks self-confidence" (Disney 2013). DSM-III-R (APA 1987) completed revamped the diagnostic criteria to at least five of the following nine symptoms (quoted in Disney 2013):

- Unable to make everyday decisions.
- Allows others to make important decisions.
- Agrees with others even when think they wrong.
- Difficulty initiating projects.
- Does unpleasant tasks to gain the approval of others.
- Dislikes being alone.
- Devastated when a close relationship ends.
- Preoccupied with fears of abandonment.
- Easily hurt by criticism or disapproval.

DSM-IV (APA 1994) dropped the last criteria, and

diagnosis was based on five of eight symptoms [28]. This gives 93 possible combinations of symptoms (Disney 2013) [29].

Gude et al (2006) found sixty of the possible combinations among 248 individuals with DPD in Norway, and no combination appeared more than seven times. This diversity has led some to argue that there is no symptom that must be present for a diagnosis of DPD, and thus to challenge the construct of DPD. Disney (2013) defended the variety by saying that "this argument can be used for all of the PDs [personality disorders] and some of them (anti-social, for instance) are even more heterogeneous than DPD".

There are concerns about the validity of DPD. In other words, whether the criteria actually describe an underlying, separate condition. Blashfield and Breen (1989) asked sixty-one psychologists and psychiatrists to put the 142 personality disorder criteria of the time (DSM-III-R) with the correct PD. On average, two-thirds of the criteria were categorised correctly. DPD criteria were correctly identified 73% of the time. But 12% of the time, DPD criteria were assigned to borderline PD, and 13% of the time, criteria for avoidant PD were included in DPD. "The authors concluded that DPD is not one of the more problematic PDs, as it outperformed many others in this particular study (Histrionic in particular)... Accordingly, we can conclude that clinicians are able to accurately identify DPD criteria the majority of the time" (Disney 2013 p1185). There is a question as to what this says about the validity of certain other PDs.

DPD is diagnosed more often in women, and critics have argued that the symptoms are viewed as more maladaptive in women (eg: Kaplan 1983). Kaplan (1983) argued that DPD is "little more than a caricature of the traditional female role" (Disney 2013). Is it that the criteria are gender biased or that the clinicians are biased in their use of gender-neutral criteria? Disney (2013) found studies showing both sides, and admitted that "whether or not femininity and women pathologised in the realm of DPD is still unknown" (p1188).

In terms of the causes of DPD, childhood attachment problems have been implicated from overprotective or authoritarian parenting styles, or in the form of separation anxiety disorder (Disney 2013). For example, adult DPD was nearly ten times more likely in sufferers of childhood separation anxiety disorder (Loas et al

[28] "DPD was nearly deleted from DSM-5 before publication, only to be retained at the last second after much heated debate" (Disney 2013).

[29] Only four of the eight criteria had empirical support when DSM-IV was published in 1994 (Disney 2013).

2002).

"Dependent personality has received very little empirical attention, despite its presence in the DSM for the last 32 years... The research attention it has received has quite often been qualitative, anecdotal, methodologically flawed, and/or published in journals with low impact factors, where it does not receive much attention" (Disney 2013 p1193).

Studies are limited on a number of aspects of DPD (Disney 2013):

- Cultural differences (eg: individualistic vs collectivistic societies).
- Ethnic differences.
- Development of DPD across the lifespan (ie: age differences).
- Co-morbidity with other PDs, or mental disorders (eg: anxiety disorders).
- Male sufferers.

MENTAL RETARDATION

"Mental retardation" (or learning disabilities) in DSM-IV-TR (APA 2000) was categorised with "disorders usually first diagnosed in infancy, childhood, or adolescence", and was defined as "significant sub-average intellectual functioning: an IQ of approximately 70 or below on an individually administered IQ test" (p49; quoted in Hauser et al 2014).

In DSM-5 (APA 2013), the term "intellectual disability is used, and it is one of the "neuro-developmental disorders". It is defined as "deficits in intellectual functions, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience, confirmed by both clinical assessment and individualised, standardised intelligence testing" (DSM-5 p33; quoted in Hauser et al 2014).

Table 9.1 shows some key differences between DSM-IV-TR and DSM-V for diagnosing intellectual disability (Hauser et al 2014).

DSM-IV-TR	DSM-5
1. Term used: "Mental retardation". 2. Classified under "disorders usually first diagnosed in infancy, childhood or adolescence". 3. Onset before 18 years old. 4. Diagnosis based on IQ test score. 5. Severity based on IQ score - 50-55 to 70 (mild), 35-40 to 50-55 (moderate), 20-25 to 35-40 (severe), below 20-25 (profound). 6. Deficits or impairments in at least two: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health and safety.	1. Term used: "Intellectual disability (or intellectual developmental disorder). 2. Classified under "neuro-developmental disorders". 3. Onset "during developmental period". 4. Diagnosis based on broader range of tests of "intellectual functions". 5. Severity level based on expected response on three domains - conceptual, social, and practical. Mild to profound levels still used. For example, mild level for conceptual domain - "a somewhat concrete approach to problems and solutions compared with age mates" (quoted in Hauser et al 2014). 6. Includes "deficits in adaptive functioning that result in failure to meet developmental and socio-cultural standards for personal independence and social responsibility. Without ongoing support, the adaptive deficits limit functioning in one or more activities of daily life, such as communication, social participation, and independent living, across multiple environments, such as home, school, work, and community" (DSM-5 p33; quoted in Hauser et al 2014).

Table 9.1 - Intellectual disability in DSM-IV-TR and DSM-5.

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10. POST-NATAL DEPRESSION IN PAKISTAN

Maternal mood disturbances in the month after birth that are transient and mild are classed as post-natal or postpartum blues [30], but more severe symptoms that develop up to a year after birth are labelled as post-natal or post-partum depression (PPD). The rates of PPD vary around the world between 11-24% of mothers (Ali et al 2009).

Rates of PPD also vary between urban and rural areas of the same country. For example, in Pakistan, the rates vary between 24-42% in urban areas, but as high as 56% in rural ones (Ali et al 2009).

Ali et al (2009) reported a study of post-partum anxiety and depression in two areas of the Pakistan mega-city Karachi classed as peri-urban (urban fringes, but not rural). All pregnant women in the areas were identified by house to house survey, and 420 of 651 agreed to answer questions before the birth and regularly up to one year after birth (at one month, two months, six months, and twelve months). Only data for 267 women were available for the whole year.

Anxiety and depression was measured by the Aga Khan University Anxiety and Depression Scale (AKUADS) (a interviewer-administered questionnaire in Urdu developed in Pakistan). The overall prevalence of anxiety and depression was 29% (77 women [31]) (ie: a score above the cut-off point of AKUADS on at least one of the four post-natal measurements). The key risk factors were found to be domestic violence, difficulty in breast-feeding at birth, and unplanned current pregnancy [32].

APPENDIX 10A - PREGNANCY AND MOTOR VEHICLE ACCIDENTS

Redelmeier et al (2014) found a significant increase in motor vehicle accidents for pregnant women as drivers in the second trimester of pregnancy.

Data were collected for over 500 000 women who gave birth in Ontario, Canada between April 2006 and May 2011. The researchers used a self-matched longitudinal design, where each woman was her own control/comparison. Details of motor vehicle accidents as a driver, that required a visit to an emergency department, were established for four years before delivery and one year after. The period of nine months before delivery was compared to the other

[30] There is a lot of debate as well about psychological changes during pregnancy (appendix 10A).

[31] Sixty-five women on one occasion, 11 women twice, and one women classed as anxiety and depressed at three measurement points.

[32] For each variable, anxiety and depression was 2-3 times more likely having experienced it than not.

periods. Also for the same five years motor vehicle crashes as a passenger or pedestrian were recorded. In addition, comparisons were made with inadvertent falls that led to a hospital visit, and other reasons for going to an emergency department (eg: burns).

In the three years prior to pregnancy (baseline), the women were involved in 6922 crashes as drivers, which is a rate of 4.55 events per 1000 individuals annually. In the year after delivery, the rate was 2.35 (1192 crashes). During pregnancy the rate was 6.47.

The rate of crashes varied within the pregnancy - eg: 4.33 in the first month of the first trimester (169 crashes), and 2.74 for the last month of the third trimester (107 crashes). The first month of the second trimester was highest, and a significant difference compared to the baseline, with 7.66 events per 1000 individuals annually (299 crashes). This is a relative risk of 1.42. This observation was not affected by variables like age or socio-economic status of the women.

The rate of crashes where women were passengers or pedestrians did not vary between pregnancy and non-pregnancy, nor did the reasons for visiting an emergency department. There was no evidence that the women showed increased risky behaviour during pregnancy based on general questions about health behaviours (eg: stopping smoking when pregnant).

Redelmeier et al (2014) believed that the increase in crashes during pregnancy was real and not a statistical artefact, but they did not have a clear explanation. They stated: "Subjective disturbances during pregnancy are commonly reported in the obstetrical literature where absentmindedness is denoted as 'baby brain' or other negative terms. Community surveys suggest that about half of pregnant women complain of sporadic cognitive lapses; however, laboratory studies in this setting provide results with uncertain clinical relevance. The gap between popular beliefs and scientific evidence has fuelled speculations about survey respondents misattributing normal memory lapses to a current pregnancy" (Redelmeier et al 2014 p6).

However, this study used only secondary data. These are records collected by someone else (eg: hospital admission details), and the researchers had no way of knowing if these were accurate. For example, the calculation of the stage of pregnancy was done by working backwards from delivery date recorded on hospital records.

No data were collected directly from the women about their driving (eg: via driving diaries). The study was retrospective, and based on car crashes that led to emergency department visits only.

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11. EATING DISORDERS AND THE MEDIA IN FIJI

The media plays a role in disordered eating among female adolescents. For example, by the internalisation of cultural values of thinness for women as seen on television. But it is difficult to test this idea experimentally unless there is a population who is media-naive [33]. Such populations are uncommon today.

Becker et al (2002) took the rare opportunity to study the Nadroga province of Fiji (figure 11.1), where television was not introduced until 1995. A group of female ethnic Fijian adolescents (14-18 years old) were surveyed within one month of television arriving (and this was taken as the pre-television baseline), and a group of the same age three years later in 1998 (post-television). Eating attitudes and behaviours were measured by a modified version of EAT-26 (Garner et al 1982) (which asks about behaviours like bingeing and purging), and other questions about body image.

(Source: Central Intelligence Agency's World Factbook; in public domain)

Figure 11.1 - Location of Nadroga province in south-west of Viti Levu.

[33] Laboratory experiments have manipulated the exposure to media images in the short-term and then measured potential disordered eating behaviours. Some have found an effect of the images (eg: Stice and Shaw 1994), some not (eg: Cusumano and Thompson 1997), and others only for vulnerable individuals (eg: Hamilton and Waller 1993).

Two key differences were found between the two samples. Firstly, significantly more adolescents had scores above the cut-off point for disordered eating in 1998 (29.2%) than 1995 (12.7%). Secondly, self-induced vomiting to control weight had gone from zero in 1995 to 11.3% in 1998. The vast majority of respondents in 1998 felt television had influenced them to feel differently about their body shape or weight.

The introduction of television was part of general social change in Fiji, and so the changes in disordered eating cannot be solely placed on television. Eating disorders are increased by migration, urbanisation, and modernisation (eg: Nasser et al 2001).

The study did not use clinical diagnosis of eating disorders, while some indicators of disordered eating did not increase (eg: laxative use to control weight).

Becker et al (2011) developed the earlier study with 523 female ethnic Fijian 15-20 year-olds who completed questionnaires in 2007. These included the Eating Disorders Examination Questionnaire (EDE-Q) (Fairburn and Beglin 1994) [34], and the Sociocultural Attitudes Towards Appearance Questionnaire (SATAQ-3) (Thompson et al 2004) [35], which measures media influences on norms about physical appearance. Mass media exposure was measured in four ways - two direct and two indirect. The direct measures were own and parental frequency of television viewing (ie: number of evenings per week mostly spent watching television or videos: 0-7). The indirect measures were own and close friends' household electronic media access index (ownership of television and/or video, CD or MP3 player, mobile phone, and internet access: 0-4). Modernisation was measured by items like perceived conflict with parents over Westernised lifestyle.

The mean frequency of television viewing was 3.05 evenings per week for adolescents and 2.66 for parents, with 88% of respondents spending at least one evening per week viewing. The mean electronic media access index was 2.12 for own household and 2.84 for friends' household.

There were significant positive correlations between the four measures of mass media exposure and score on the EDE-Q (where a higher score is a sign of eating pathology). After controlling for modernisation, media exposure was associated with SATAQ-3 score, and subsequently with EDE-Q score.

The study showed that not only does the media have a direct influence (ie: hours of television watched) on

[34] Eg: "Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence body shape or weight?".

[35] Eg: "TV programmes are an important source of information about fashion and ‘being attractive’"; "I’ve felt pressure from TV or magazines to lose weight"; "I compare my appearance to the appearance of people in magazines".

eating pathology and body image, but there was an indirect effect (ie: electronic media access index of friends' household). In fact, the latter had the stronger association with eating pathology after adjusting for other variables. Put another way, the presence of television in a community, even if an individual household does not have one, can have an affect on adolescents (through social interaction with television-exposed peers).

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12. OCCIPITAL BENDING AND DEPRESSION

The two hemispheres of the brain can vary in tissue distribution, despite having similar volumes, weights, and densities. One example of this difference is "occipital bending" (Maller et al 2014). This is where one hemisphere wraps around the other (at the back - occipital lobe) (figure 12.1) [36].

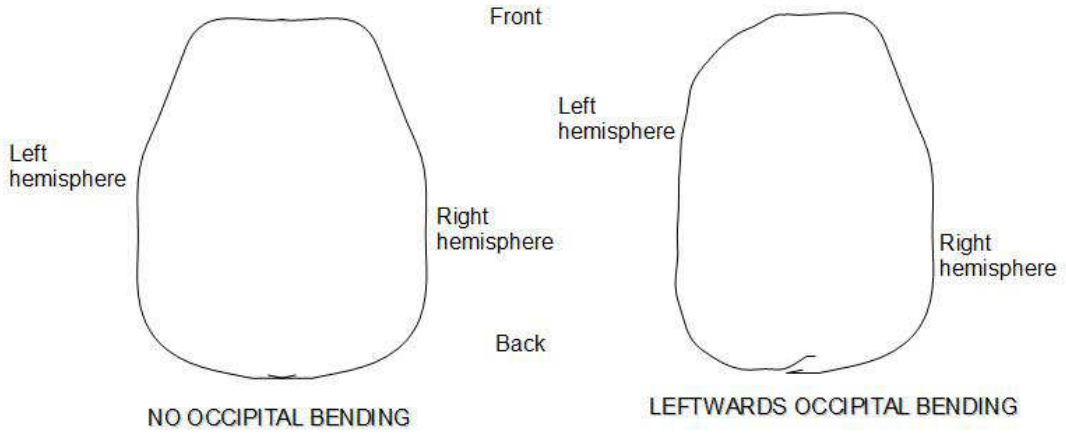

Figure 12.1 - Top view of brain showing occipital bending.

Maller et al (2014) found significantly more occipital bending in patients with major depressive disorder (MDD) than in healthy controls. This study was based on magnetic resonance imaging (MRI) scans [37] of fifty-one adults with treatment-resistant MDD and forty-eight matched controls in Victoria, Australia, and Toronto, Canada. Around one-third of the former group (35.3%) showed occipital bending compared to 12.5% of the controls. Left hemisphere wrapping around the right (rightwards direction) was more common in both groups.

The researchers did not find any difference betwee the two groups in total brain volume, though previous studies had reported greater volume among individuals with MDD (eg: Kempton et al 2011).

The differences in the brains of individuals with MDD to controls are correlational, not established as causation.

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[36] In the first few months of life neurons are pruned (ie: destroyed), but "incomplete neural pruning may lead to the cranial space available for brain growth being restricted, or ventricular enlargement may exacerbate the natural occipital curvature patterns, subsequently causing the brain to become squashed and forced to 'wrap' around the other occipital lobe" (Maller et al 2014 p1830).

[37] There are many and various ways to study the brain including techniques being developed (appendix 12A).

APPENDIX 12A - THE FUTURE OF STUDYING THE BRAIN

The study of the brain is based upon two main techniques today - electroencephalography (EEG) or electrodes [38], which measures electrical activity [39] [40], and neuroimaging, which shows the structure or activity (eg: blood flow) of the brain (appendix 12B).

Marblestone et al (2013) distinguished five main ways to measure neural activity - electrodes, light, magnetic resonance imaging and radio-frequency magnetic induction signals, molecular, and ultrasound.

Marblestone et al (2013) noted: "Simultaneously measuring the activities of all neurons in a mammalian brain at millisecond resolution is a challenge beyond the limits of existing techniques in neuroscience [41]. Entirely new approaches may be required, motivating an analysis of the fundamental physical constraints on the problem". Electrical recording of the cells not using traditionally inserted electrodes is a challenge in terms of the delivery of the recording devices, and "the ability to relate the measured electrical signals to specific cells within a circuit. As the set of neurons recorded by each electrode grows to encompass a large volume around the electrode, it will become more difficult to attribute the recorded spikes to particular neurons" (Marblestone et al 2013).

Yuste and Church (2014) argued for new, more sophisticated and varied techniques to understand the brain's physiology.

Here are a selection of new techniques that are being developed.

1. Calcium imaging - Genetically engineered neurons which fluoresce when calcium ions enter a cell after firing (eg: Smetters et al 1999). The electrical signals of neurons are produced by different ions moving between

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[38] Traditionally, electrodes are inserted into the brain, but this risks damage to the brain area during the insertion process, and in volume displacement (ie: displacement of brain tissue when an object inserted) (Marblestone et al 2013).

[39] Since the 1950s, the number of neurons that can be simultaneously recorded using wired electrodes has doubled every seven years to the current ability of hundreds at sub-millisecond timescales (Marblestone et al 2013).

[40] Electrocorticography (ECoG) records electrical activity from the surface of the cortex. It has advantages over both EEG and single-cell recording. It gives a more precise picture of electrical activity than EEG, and is not as invasive as single-cell recording, which penetrates deep into the brain. However, surgery is required to place electrodes on the surface of the brain (Leuthardt et al 2004).

[41] The human brain has an estimated 8 times 10 to the ten neurons (Marblestone et al 2013). Furthermore, not all cells in the brain fire in the same way - there is variety in length of action potential (firing) (eg: 2 ms), rate of firing (eg: once per second), and frequency (eg: 5 Hz) (Marblestone et al 2013).

the cells, simplistically [42]. This technique has been used with zebrafish larvae, which are transparent (Yuste and Church 2014). But this technique is unable to record inhibitory signals that stop electrical activity in the cell (Yuste and Church 2014).

2. Voltage imaging - This involves dyes that change colour as voltages in the cells change. The dyes can be genetically engineered into cells or deposited there in some way. The key is having dyes that do not damage the neuron (Yuste and Church 2014).

3. Nanotechnology (eg: nanodiamonds) - These minute objects are sensitive to change in electrical fields and could be deposited in the cells to record electrical activity (Yuste and Church 2014).

4. Computational optics - Computers are used to construct an image based on information about light waves. The skull is opaque (ie: difficult to see through with light), but the disordered waves of light scatter when hitting such an object. Computational methods are used to reconstruct the light waves. This may see through the skull, but not to the deepest areas of the brain (Yuste and Church 2014) [43].

5. Molecular ticker type - This technique genetically engineers molecules to change when a neuron is active. For example, a string of DNA is engineered to change its sequence with the chemical changes of cells firing (Yuste and Church 2014).

6. Optogenetics - Genetically engineered animals have light-sensitive proteins (opsins) derived from bacteria or algae in the cells of the brain, and these proteins cause the neuron to switch on or off when light is shone on it. This technique often requires the brain to be open to shine the light into, or else an endoscope could be used (Yuste and Church 2014).

7. Optochemistry - A light-sensitive substance called a "cage" holds a chemical that works on neurotransmitters in the brain. When the chemical is released, it can make the neurotransmitter active or inhibit it. Yang et al (2012) were able to stop an epileptic seizure in rats with the neurochemical GABA inside a "cage".

These new techniques raise a number of practical and

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[42] For example, calcium changes in the cell occur within one to 100 ms (Marbelstone et al 2013).

[43] How to separate light emissions from the studied neuron(s) and from other points in the brain (ie: "noise") (Marblestone et al 2013)?

ethical issues.

i) "A long path still stretches from basic research to clinical applications. Each new idea for the large-scale measurement and manipulation of neural activity will have to be tested in fruit flies, roundworms and rodents before moving on to humans" (Yuste and Church 2014 p29).

ii) The ethics of animal research, particularly in terms of genetic engineering. Though it may be less painful than old-fashioned experiments with animals, the individual animal is changed in a way that limits their life (even if they are not killed soon after the research).

iii) The vast amount of data created by measurements of thousands of cells. Computers that can handle petabytes [44] of data are needed.

iv) Much research on the brain assumes that a complete understanding of the physiology simply requires more data. In other words, when we have recorded all the cells in the brain together, then we will know the brain. What happens then? If everything is known about the brain, then there are issues about what to do with such information (eg: related to social control). On the other hand, complete data about the brain may not explain the experience of the human brain.

APPENDIX 12B - BRAIN SPECIALISATION

Traditionally, it is felt that different brain areas are responsible for different activities. "A newer school of thought, however, postulates that the brain is more like an informal social network than one having a rigid division of labour. In this view, the connections that a neuron has made with other brain cells determine its behaviour more than its position does, and the behaviour of any given region is influenced strongly by its past experiences and current situation. If this idea is correct, we can expect to see overlapping activity among the particular locations that handle the brain's responsibilities" (Lein and Hawrylycz 2014 p58).

It is a daunting task of mapping billions of connections in the brain, but a number of large-scale projects are underway including (Lein and Hawrylycz 2014):

[44] A petbyte is 10 to the 15 bytes (ie: 1 followed by 15 zeros) (http://en.wikipedia.org/wiki/Petabyte; accessed 28/04/14).

- SyNAPSE - a "digital analogue" of the brain using neurosynaptic chips [45].

- BigBrain project - a 3-D model [46].

- Non-Human Primate Atlas - mapping gene expressions in the rhesus monkey brain [47].

Work to map the activity of genes in the brain found that gene expression patterns (eg: protein production) were the same in both hemispheres, which challenged the idea of each hemisphere specialising in certain abilities (eg: left hemisphere and language).

Comparison of around one thousand genes active in the cortex of both mouse and human brains found that about one-third expressed differently. Thus, "at a genetic level, humans are not simply large mice" (Lein and Hawrylycz 2014).

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[46] See https://bigbrain.loris.ca/main.php.
[47] Details at http://blueprintnhpatlas.org/.

13. POST-TRAUMATIC STRESS DISORDER IN RWANDA

DSM-IV-TR (APA 2000) defined Post-Traumatic Stress Disorder (PTSD) as: "Exposure to an extreme traumatic stress involving direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one's physical integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm, or threat of death or injury experienced by a family member or other close associate" (p463 quoted in Connolly and Sakai 2011).

Civilians are increasingly victims of war around the world (up to 90% of the population in a region), and children as part of this (eg: two million killed in wars in the 1980s) (Schaal and Elbert 2006).

Children experience the war, injury to themselves, and loss of parents leaving them vulnerable to PTSD (eg: 52% of 9-14 year-olds interviewed after the war in former Yugoslavia; Smith et al 2002). "Child-headed households" (CHHs) is a phenomenon after conflict (eg: 300 000 children worldwide living in CHHs in 1998) (Schaal and Elbert 2006).

Concerning adults, approximately one in ten women and one in twenty men worldwide suffer from PTSD at any one time (Connolly and Sakai 2011). Much of that is due to war and armed conflicts.

For example, in Rwanda in 1994 over three-quarters of a million individuals were killed in a 100-day period ("Rwandan genocide"). Pham et al (2004) found that 24.8% of 2074 adults had PTSD when studied in 2002. Twenty-six trained interviewers in February 2002 visited four communities with a standardised questionnaire. Three communities had experienced high levels of killing and one area low. Interviewees were randomly selected.

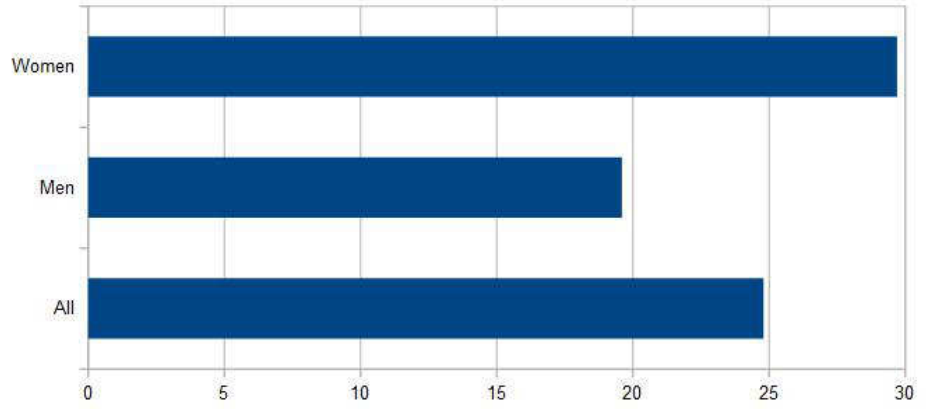
The symptoms of PTSD were measured by the PTSD Checklist-Civilian Version (PCL-C) (Blanchard et al 1996), which has seventeen items derived from the DSM-IV criteria for PTSD. Each item is scored from 1 ("not at all") to 5 ("extremely") giving a range of 17-85. A cut-off score of 44 was used [48].

The majority of respondents (75%) had had to flee their homes, and about the same number (73%) had had a close family member killed. The rate of PTSD varied between communities (12 to 33%), and was higher among women than men (figure 13.1). The most commonly reported symptom was re-experiencing the trauma. The greater the

[48] A cut-off score of 50 is used in other studies (eg: Hagengimana et al 2003).

exposure to traumatic events, the greater the likelihood of PTSD (eg: three events - three times more likely than no events).

PTSD sufferers were less open to reconciliation, though other personal and environmental factors were also relevant to this attitude (eg: educational level, perception of change in level of poverty).



(Data from Pham et al 2004 table 2 p607)

Figure 13.1 - Percentage of respondents with PTSD based on gender.

Pham et al (2004) had the following limitations:

- Not a nationally representative sample.
- No measure of level of PTSD prior to 1994, so it is assumed that current PTSD was caused by genocide.
- No independent verification of information (eg: whether respondents experienced traumatic events as said).
- The study was eight years after the genocide, and accurate recall may have been an issue.
- Honesty of replies when political situation was not completely stable in the country (eg: not democratically elected government at time).

Among 100 members of the Rwandan Widows' Association, who had lost their husband during the genocide, surveyed in 2001 using various questionnaires including the PCL, 40 reported panic attacks in the four weeks prior. Thirty-five were equivalent to a diagnosis of panic disorder. PTSD was significantly higher in widows with panic attacks (50%) than without (25%). Overall, forty-six women were above the PCL cut-off (ie:

diagnosis of PTSD) (Hagengimana et al 2003).

Between December 2003 and January 2004, Schaal and Elbert (2006) interviewed sixty-eight 13-23 year-olds living in Kigali, Rwanda. Half lived in CHHs and half lived in an orphanage. All respondents had experienced some kind of violence, and most had seen dead or mutilated bodies, and/or witnessed the death of a close family member (eg: 41% of their own parents).

Overall, 44% met the full DSM-IV criteria for PTSD. The rate of PTSD was higher for those in CHHs than in the orphanage (56 vs 32%). Schaal and Elbert (2006) pointed out: "The loss of the parents was one of the most severe events for the respondents. In the orphanage, the staff may eventually take a parenting role and give the youth a feeling of warmth and protection. On the other hand, children in a CHH face the adult responsibilities of supporting themselves and maintaining a household at an age when the care and protection of an adult are needed. This daily strain is possibly a contributing factor to the higher rate of PTSD in the CHH group. In addition many of the children are living in the very home in which their parents were killed. Quite possibly, the daily reminder of the death of their parents or the symptoms associated with these memories contribute to the maintenance of PTSD" (p102).

The average of PTSD symptoms reported was 8.8 (out of 17), but those in CHHs had 10.0 symptoms (compared to 7.66 for the orphanage). The most common symptoms were upsetting thoughts about the event, and avoidance in thinking or talking about the event.

There was a clear gender difference - 60% of girls and 27% of boys meet the criteria for PTSD. Older interviewees had more PTSD symptoms than the younger ones. Remembering that the genocide was a decade prior, the younger interviewees (13-17 years old) would have been 3-7 years old compared to 8-13 years old. There is some disagreement over whether a younger age is a greater or lesser risk factor for PTSD after trauma. Schaal and Elbert (2006) concluded: "It seems possible that younger children may be protected from post-traumatic stress by their less developed cognitive abilities. The developmental stage influences the capability to understand the meaning and consequences of traumatic events and thus whether an alarm response is triggered. The alarm response seems a necessary precondition for PTSD development..., and the younger youth may possibly not have become fully aware of the realistic threat of harm to them" (p102).

PTSD was associated with more trauma exposure, witnessing the murder of a parent, and belief that they would die.

Schaal and Elbert (2006) concluded:

> Ten years after the genocide, many survivors were significantly distressed and had received little or no psychotherapeutic support. Two-thirds of these adolescents reported that this was the first time that they had ever talked to anyone about their problems or experiences during the genocide. Nevertheless, all survivors were very willing and open about discussing their feelings and past experiences. Many, wanting to detail their traumatic experiences, asked whether we could return for another interview. This openness is in line with... experiences, in which traumatised children consider talking about their experiences to be of the most help.

Wood (2007) went as far as to suggest that everyone in Rwanda was suffering from traumatic stress in some way.

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14. PARADOXICAL TREATMENT

"Paradoxical treatment" is a technique that goes against commonsense, and prescribes the symptom as the treatment [49]. In the case of insomnia, where the individual has trouble falling asleep, traditional treatments focus upon ways to help the individual sleep. Paradoxical treatment might encourage the sufferer to stay awake.

Most controversially, paradoxical treatment involves challenging a suicidal patient to take their life. Nicholas. A. Cummings has done this as part of "focused psychotherapy" [50] (which combines psychoanalysis and strategic therapy) (Thomason 2012). Cummings (1984 quoted in Thomason 2012) took the view that "every suicide is really a homicide turned inward". Anger towards another person has become turned inwards as depression, self-directed hostility, and suicide. Challenging the patient to take their life is meant to externalise the anger towards the therapist.

The case of "Beth" (Cummings and Sayama 1995) is quoted as the success story of this technique. She was a suicidal (possibly schizophrenic) woman who Cummings believed had internalised her anger about her husband's affair and directed it towards herself. Cummings placed a cyanide capsule on the table, and told her to take it (and he accused her of being a coward) while he left the room. This was repeated four times over four weeks. Beth became angry with Cummings, and subsequently with her husband, who she divorced (Thomason 2012).

The therapist acting so unexpectedly (in "anti-therapeutic" way) surprises the patient's defence mechanisms, and "catapults a wishy-washy patient into intensity" (Cummings 2006).

This theory of suicide as anger turned inwards has a psychoanalytic origin, but it has been questioned by other psychologists. "There is no simple relationship between anger and depression, and there is no more reason to think that depression is anger turned inward than that anger is depression turned outward. Simple equations between depression and the 'direction' of anger have not been supported" (Thomason 2012 p42).

Another example of what is called in everyday language, "reverse psychology" (or calling their bluff) is where Cummings placed women with borderline personality disorder in a therapy group that he called "the losers group". He told them that he expected it to

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[49] Alfred Adler was the first to write about paradoxical prescribing in psychotherapy. The ideas were formalised in Watzlawick et al (1967) (Thomason 2012).

[50] Also described as "psychojudo" and "psychological surgery" (Cummings and Sayama 1995).

fail. The idea being that the anger of the women would motivate them to respond to the therapy. Thomason (2012) was sceptical: "It is difficult to understand why patients with a lifelong personality disorder would suddenly become motivated to overcome it simply because a therapist called them 'losers' and expressed doubt about their ability to change" (p43).

Such treatment by a therapist of individuals who are vulnerable raises ethical issues (as well as legal ones) (Thomason 2012).

There is also little research on the effectiveness of paradoxical treatment other than case studies and anecdotal reports (Thomason 2012). Fabry (2010) found nineteen outcome studies (mainly with insomnia or agoraphobia), mostly with small samples and no control groups. "Until clinical and empirical research is conducted to support the use of paradoxical interventions with suicidal patients, they are best avoided. But it is hard to imagine how it would be ethical even to conduct such research; the extremely high risk of patient's death would seem to make such research implausible. What institutional review board would approve such a study?" (Thomason 2012 p50).

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15. RECOVERY FROM SCHIZOPHRENIA AND DURATION OF UNTREATED PSYCHOSIS

Just over one in ten individuals (13.5%) with schizophrenia are classed as recovered (Jaaskelainen et al 2013) (appendix 15A). There is interest in factors that might influence this figure. One factor studied is duration of untreated psychosis (DUP) - ie: the period between the onset of the disorder and the beginning of treatment. Or put another, early treatment after onset.

For example, Boonstra et al (2012) found that long DUP was associated with increased negative symptoms of schizophrenia (eg: blunted emotions, social withdrawal) in first-episode cases (table). But Gafoor et al (2010) reported that benefits of early intervention did not necessarily persist in the long-term (ie: longer than five years). Penttila et al (2014) felt that the studies were divided over the relationship between DUP/early treatment and long-term recovery from schizophrenia.

- The researchers searched for relevant studies published between December 1992 and March 2009.
- 28 studies met the inclusion criteria (eg: standardised measures of negative symptoms like Brief Psychiatric Rating Scale (BPRS) or Positive and Negative Symptom Scale (PANSS).
- The individual data of patients was collected for 16 studies (n = 3339 participants).
- Mean DUP = 61.4 weeks; median = 12.0 weeks.
- Significant positive correlations between DUP and negative symptoms (number and severity) - ie: longer DUP and more symptoms:
  At baseline: r = 0.117 ($p<0.001$).
  In short-term (12-24 months): r = 0.180 ($p<0.001$).
  In long-term (60-96 months): r = 0.202 ($p<0.001$).
- "A DUP of less than 9 months appears to be a strong predictor of improvement of negative symptoms, while most patients with a DUP longer than 9 months show persistent negative symptoms" (p16).
- "However, it is important to emphasise that this analysis does not prove that there is a causal association between DUP and negative symptoms" (p16).

Table - Boonstra et al (2012).

Thus Penttila et al (2014) performed a meta-analysis of studies on DUP and long-term outcomes of schizophrenia. Thirty-nine articles met the inclusion criteria (eg: sample size greater than twenty; clear definition of DUP; formal diagnosis of schizophrenia). Overall, longer DUP was significantly associated with poor outcomes in terms of symptoms (eg: severity and

frequency), and failure to achieve remission or recovery.

The variation in methodology between studies, however, placed limitations on this conclusion. In particular, the definition of DUP and precision of measurement of it.

APPENDIX 15A - RECOVERY

Jaaskelainen et al (2013) began their article: "It is widely accepted that a proportion of individuals who develop schizophrenia have a favourable prognosis. Symptoms can abate over time, and a proportion of those with schizophrenia attain good outcomes on a range of clinical and functional outcomes (eg, education, employment, and relationships). The precise proportion of cases that have favourable outcomes is less clearly understood" (p1296). Part of the reason for this is the difficulty in measuring "recovery".

Hegarty et al (1994) produced a meta-analysis of 320 studies from 1895 to 1992 on outcomes of schizophrenia. Around 40% of individuals had a good outcome, but the definition of "good outcome" varied between studies (eg: improvement in symptoms or good social functioning) (Jaaskelainen et al 2013).

Menezes et al (2006) reviewed thirty-seven studies on the outcome of first-episode psychosis, and found a similar figure to Hegarty et al (1994). But the follow-up periods were quite short (Jaaskelainen et al 2013).

Warner's (2004) analysis of 114 studies published between 1994 and 2000 defined recovery as complete (ie: loss of psychotic symptoms and return to pre-illness level of everyday functioning) or social (ee: economic independence and social functioning). Complete recovery was evident for 11-33% of individuals (depending on the study) and social recovery for 22-53%.

Jaaskelainen et al (2013) found fifty studies (in English up to late 2011) for inclusion in their meta-analysis. They used a multi-dimensional definition of recovery, which included clinical remission (ie: reduction in symptoms) and social functioning, for at least two years. This was stricter than the previous meta-analyses. The median rate of recovery was 13.5% and the mean 16.4% with the inter-quartile range (ie: 25%-75% of the sample) of 8.1% to 20.0%. No difference was found in rates of recovery between men and women.

Sadly, Jaaskelainen et al (2013) confirmed the previous studies that rates of recovery had not improved over time (ie: no difference between older and newer studies). The authors stated: "Indeed, recent decades had lower numerical proportions of subjects who met our recovery criteria. This is a sobering finding — despite

major changes in the delivery of care to people with schizophrenia (eg: deinstitutionalisation, anti-psychotic medications, psychosocial interventions, and early psychosis services), the proportion of those who met recovery criteria have not improved over time. However, the studies in this meta-analysis are naturalistic, and we do not know what kind of treatment the patients received. Thus conclusions about the effect of treatments are not possible" (Jaaskelainen et al 2013 p1304).

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16. RISK OF EATING DISORDERS AMONG ELITE ATHLETES

Individuals involved in elite sport can have a higher risk of eating disorders. For women in sports emphasising leanness or low body weight, there is a higher frequency of eating problems than controls, but a lower risk for lower ability participants in aesthetic sports and running. While men competing in weight class sports have a higher risk of eating disorders than the general population, but a lower risk in sports like swimming and figure skating (Sundgot-Borgen and Torstveit 2004).

Sundgot-Borgen and Torstveit (2004) pointed out: "Athletes constitute a unique population, and the impact of factors such as training, eating pattern, extreme diets, restriction of food intake, and psychopathological profile must be evaluated differently from this impact in non-athletes" (p25). Thus, Sundgot-Borgen (1993) referred to "anorexia athletica" (AA) [51], where all diagnostic criteria for anorexia nervosa or bulimia nervosa are not met. AA sufferers have an intense fear of gaining weight or being fat despite having low body weight, and this weight is maintained through low energy intake, excessive exercise, or sometimes self-induced vomiting (Sundgot-Borgen and Torstveit 2004) [52].

Sundgot-Borgen and Torstveit (2004) studied elite Norwegian sportsmen and women. In 1997 all elite athletes representing Norway were given the Eating Disorder Inventory (EDI). The 1620 individuals represented 68 different sports, which were divided into eight sport groupings (Torstveit and Sundgot-Borgen 2005) (table 16.1). A control sample of the general population was randomly selected (n = 1696).

The mailed questionnaire was completed by 1259 athletes (572 women and 687 men) and 1203 controls (574 women and 629 men). At this stage, 182 athletes and 103 controls were above the cut-off score on the EDI. These individuals were given a clinical interview to confirm diagnosis (120 athletes and 76 controls agreed to participant).

After this two-step process, 20% of female and 8% of male athletes met the diagnostic criteria for eating disorders, and 9% of women and 0.5% of men in the control

[51] AA includes perfectionism, compulsivity, competitiveness, high self-motivation, menstrual disturbance, and at least one unhealthy method of weight control (fasting, vomiting, use of diet pills, laxatives, diuretics) (Birch 2005). Birch (2005) referred to a "female athlete triad" of osteoporosis, disordered eating, and menstrual disturbance.

[52] The eating disorder is more associated with training and sports performance than in the general population (Birch 2005).

- Technical - eg: bowling, shooting, golf.
- Endurance - eg: cycling, rowing, swimming.
- Aesthetic - eg: rhythmic gymnastics, figure skating, cheerleading.
- Weight class - eg: boxing, weightlifting, wrestling.
- Ball game - eg: football, tennis, hockeys.
- Power - eg: throwing field events like hammer, sprinting, alpine ski-ing.
- Anti-gravitation - eg: field event jumps, indoor rock climbing.
- Motor sports.

Table 16.1 - Sport types.

sample [53]. The female athlete group was significantly higher than the other groups ($p<0.001$) [54], and the female controls were significantly higher than the male controls ($p<0.001$) [55].

In terms of type of sport, two significant patterns appeared:

a) Men - greater eating disorders in anti-gravitation sports (22%) than ball game (5%) and endurance (9%) sports.

b) Women - greater eating disorders in aesthetic sports (42%) than endurance (24%), technical (17%), and ball game (16%) sports.

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[53] Five female and four male athletes, and 23 female and 16 male controls were false-positives (ie: self-reported as eating disorder but not clinical diagnosis). This is an issue for studies that depend on self-reported questionnaires only.

[54] Devine (2012) pointed out that female athletes are required to look good as well as play ("performing femininity"). She referred to the president of Fifa (football's governing body) who suggested that female players "could have tighter shorts" to increase the appeal of the game.

[55] An Australian study of elite athletes in ten sports in that country found similar differences (eg: 31% of thin-build athletes classed as eating disordered compared to 5% of controls of the same body type) (Byrne and McLean 2002).

17. CHILDREN AND EATING DISORDERS

Traditionally eating disorders have been diagnosed post-puberty (eg: onset after 13 years old), and the number of new cases in the UK have been static in recent years (eg: 1994-2000 females; Currin et al 2005). But early onset or childhood eating disorders have increased (eg: Netherlands; Van Son et al 2006). The exact numbers are not clear, which led to the British National Surveillance Study (Nicholls et al 2011).

Between March 2005 and May 2006, all new cases of eating disorders aged 5-12 years old were collected in the UK and the Republic of Ireland via monthly returns of rare conditions by consultant child psychiatrists and paediatricians [56]. There were 208 cases [57], which converts to an incidence rate of 3 per 100 000 for 5-12 year-olds in the UK and Republic of Ireland, with 37% meeting the criteria for anorexia nervosa and 43% for eating disorder not otherwise specified [58]. The majority of cases were female (82%), and most of all cases were aged 10-13 years old. Overall, the three main symptoms reported in order were food avoidance, morbid preoccupation with food, and fear of weight gain.

Other figures for childhood eating disorders tend to be based on official statistics using a single question (eg: 0% of 5-10 year-olds and 0.3% of 11-15 year-olds in Britain; Ford et al 2003), general practice data (eg: 0.3 per 100 000 0-9 year-olds in UK; Turnbull et al 1996), or retrospective data (eg: asking adults to recall their childhood).

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[56] This did not include cases seen by general practitioners and not referred on.

[57] The researchers were confident that they had identified the majority of cases, though only those who seek help would be included. Nicholls et al (2011) also accepted: "Case ascertainment in surveillance studies relies on clinician judgement and accuracy of clinician reporting. This will be more important for some data, for example binge eating and co-morbidity, than for others such as weight. Binge, or loss of control eating is likely to be particularly underrepresented, since this would require systematic screening and clear operational criteria to detect" (pp299-300).

[58] An Australian surveillance study (Madden et al 2009) over three years reported an incidence of 1.4 per 100 000 children.

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18. TWO SUICIDE TRAJECTORIES

Susceptibility to suicide is explained these days by complex models that take into account a number of different factors (or risk variables). For example, a diathesis-stress model combines genes, other biological factors, and severe childhood adversities along with the experiences of negative life events and mental health problems as an adult. "Hence, biological predisposition may cause some individuals to be more sensitive to the effects of stressful events and more likely to choose suicidal acts as a response to unbearable emotional pain" (Seguin et al 2014 p120). Alternatively, a model by Wan and Leung (2010) combines early adversity (known as distal variables) with current mental health problems (the proximal variables) (Seguin et al 2014).

Seguin et al (2014) criticised the basis of many of these models - namely, cross-sectional designs, which compare different groups at one point in time. They said: "this type of static picture fails to capture the interactions among distinct factors, the developmental period in which they emerge, and the mechanism by which psychopathology (eg: depression) becomes an outcome or a risk factor for subsequent developmental problems" (Seguin et al 2014 p120). These researchers preferred a life course approach, which aimed to understand the "developmental process of suicide trajectories" that were "person-oriented" instead of "variable-oriented".

Seguin et al (2007) described two different trajectories leading to suicide from the life events that individuals who committed suicide had experienced. The first pattern is many and severe negative life events as a child and adult (Trajectory 1), while the other trajectory is less negative life events and more of "a slow decline throughout the life course" (Trajectory 2) (Seguin et al 2014).

Seguin et al (2014) developed this research with family members of suicide victims in Quebec, Canada. The relatives helped establish a life course calendar of the victim (n = 214), which focuses on "12 life spheres" (table 18.1).

Around two-fifths of the sample were categorised as Trajectory 1, and they tended to die before age thirty, while the rest of the sample (categorised as Trajectory 2) died later in life. Many in both groups had experienced risk factors in the first ten years of life, like physical and/or sexual abuse.

Trajectory 1 individuals experience "a great number of developmental difficulties very rapidly", like parent alcohol misuse, neglect, and tension with parents, "followed by changes of residence and by the development

- Change of permanent residence.
- Relationships, events, and changes in family of origin.
- Relationships and events in current family.
- Relationships and events in starting a family.
- Interpersonal difficulties (eg: mental health).
- Social life (eg: social support).
- "Academic life path" (eg: educational successes and failures).
- Professional life (eg: unemployment, occupational stress).
- Protective factors.
- Events of loss (eg: bereavement, divorce).
- Social adversities (eg: financial).
- Experience of mental health services.

(Source: Seguin et al 2014 p121)

Table 18.1 - Twelve life spheres.

of psychopathology leading to suicide" (Seguin et al 2014). Overall, a heavy burden of adversities at a young age.

Trajectory 2 individuals had a lower burden of adversity throughout their lives. "In some cases, early sexual abuse translated into long-term marital violence, but other spheres of life - academic or professional - were unharmed... As events accumulated in the course of their lives and mental health problems created more and more suffering and adversity, these individuals' ability to resist feelings of despair crumbled" (Seguin et al 2014 p124).

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19. UNHAPPINESS OF FASHION MODELS

Western mass media presents the picture that looking good leads to happiness and fulfilment. So, professional fashion models, who obviously are very physically attractive, should have it all. Yet, at the same time, the tabloid newspapers love stories about the "dark side of modelling". Meyer et al (2007) asked: "Is it possible that a serious but neglected psychological issue lurks beyond such glaring sensationalism? Could it be that the modelling career, despite its glamour, is associated with elevated rates of psychological maladjustment? Or is modelling, in fact, truly a golden road to fame, fulfilment, and happiness, as some glossy magazines would have you believe?" (pp2-3).

Hosoda et al (2003) concluded that "physical attractiveness is always an asset for individuals" after reviewing twenty-seven articles on attractiveness and employment, promotion, and pay. Another meta-analysis by Langlois et al (2000) found that attractive individuals had higher self-esteem, and better mental and physical health.

Again this suggests that professional models should enjoy happiness and well-being. But Meyer et al (2007) argued that modelling offers limited opportunities for satisfaction of basic psychological needs (in particular, competence, relatedness, and autonomy [59]) [60], and this produces lower well-being and happiness.

In Meyer et al's (2007) first study, 56 models from agencies in the London area and fifty-six other workers [61]were given questionnaires that covered need satisfaction (eg: "I feel like I am free to decide for myself how to live my life"), life satisfaction, subjective happiness, and self-actualisation (eg: "I can express my feelings even when they may result in undesirable consequences").

The models scored significantly lower than the non-models on all measures (figure 19.1). Further analysis showed that "models appeared to experience lower well-being because their needs were less satisfied" (Meyer et al 2007) (ie: through the occupational context). But this study could only establish correlations not causation.

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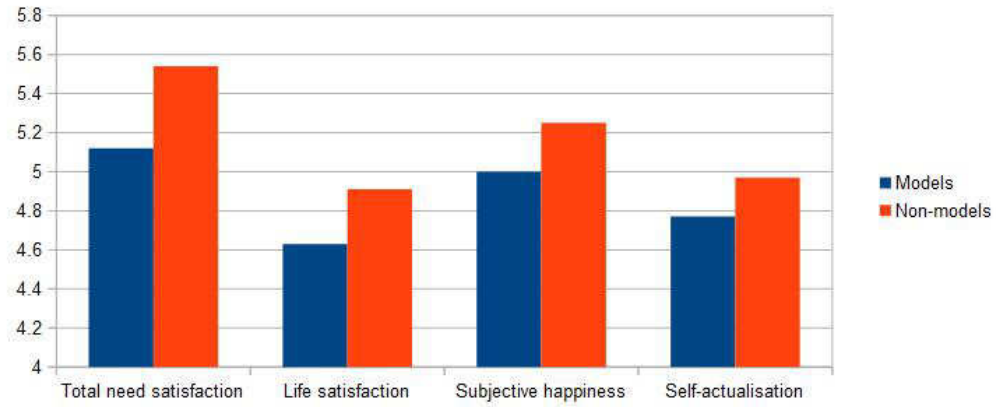
[59] Competence = mastery of skill/ability; relatedness = quality of personal relationships; autonomy = personal control of life.

[60] The self-determination theory (eg: Ryan and Deci 2002) emphasises that fulfilment of these psychological needs influences well-being, and the environment is key. Ryan and Deci (2002) stated: "the extent that an aspect of the social context allows need fulfilment, it yields engagement, mastery, and synthesis; whereas, to the extent that it thwarts need fulfilment, it diminishes the individual's motivation, growth, integrity, and well-being" (quoted in Meyer et al 2007).

[61] Approximately two-thirds of each group were women.



(Data from Meyer et al 2007 table I p6)

Figure 19.1 - Mean scores (out of 7) on questionnaires.

Meyer et al (2007) summed up:

> Why did models feel relatively less fulfilled in terms of their basic needs? We speculated in the introduction that various aspects of the modelling career might interfere with a model's ability to have her needs adequately met. Fashion models are expected to look good, but they are not valued for their ability to perform difficult, skilled tasks. Therefore, their daily routine may not permit satisfaction of the need for competence. Perhaps more than is true in many other professions, models might also have to regularly subordinate their wishes to the mandates of their clients and employers, which would interfere with the satisfaction of their need for autonomy. Finally, in their often hectic travelling from one fashion shoot to another, models might have fewer opportunities than many others to form relationships with others that go beyond superficial acquaintance. Therefore, their need for relatedness may often be met inadequately (p14).

In a second study, Meyer et al (2007) gave 35 female models and forty non-model working women in London a questionnaire that measured ten personality disorder [62] (table 19.1). The models differed significantly to the non-models on seven personality disorders - higher scores on paranoid, schizoid, schizotypal, anti-social, borderline, and narcissistic, and lower scores on obsessive-compulsive (figure 19.2). This suggested that "the models, compared to others, were slightly more suspicious, non-conforming, intensely emotional,

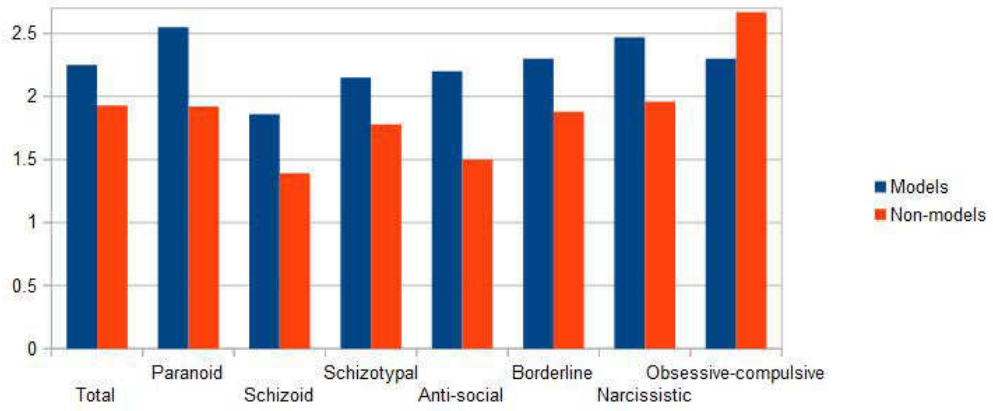
[62] There were 56 items on the Personality Disorders Features Screener (PDFS-56).

interpersonally alienated, eccentric, and self-centred. At the same time, they were less conscientious/perfectionistic but did not differ in terms of dramatic attention-seeking or shyness. The gestalt emerging from this is clearly not a flattering one. Relative to others, the models in Study 2 described their personalities as clearly less optimally adjusted" (Meyer et al 2007 p14).

Personality Disorder	Example of item used
Paranoid	I don't easily share secrets with others because they tend to use it against you at a later time
Schizoid	I actually don't have real friends, and that's okay with me
Schizotypal	I don't feel comfortable in social situations because I don't trust others
Anti-social	I've broken the law many times
Borderline	My emotions are like a roller-coaster: very intense and quickly going from very high to very low
Histrionic	I can be very sexy or seductive, and I know how to use it
Narcissistic	I'm a multi-talented person who is actually far more gifted than average people
Avoidant	I'm socially quite shy, restrained, or inhibited
Dependent	I can be a bit submissive, needy, or clingy in close relationships, but I try to not let that become a problem
Obsessive-compulsive	I'm known to be a bit of a perfectionist or "control freak"

(Source: Meyer et al 2007 table III pp10-11)

Table 19.1 - Personality disorders and example items.



(Data from Meyer et al 2007 table IV p12)

Figure 19.2 - Mean scores (out of 4) on ten personality disorders.

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20. INTEROCEPTIVE AWARENESS AND ANOREXIA NERVOSA

Media images of thin women has been proposed as a key factor in anorexia nervosa. But many individuals are exposed to such images and do not develop eating problems and with body image. So, other factors (eg: biological) would seem to be involved in the development of anorexia nervosa and body image disturbance. One suggestion is problems in interoception (awareness of the internal state of the body) [63] (Arnold 2012) [64].

Interoceptive ability can be tested by asking individuals to count their heartbeats without taking their pulse for a set period of time (heartbeat perception task) [65]. The closer the estimate to the actual number (measured objectively) is good interoceptive awareness. But individuals with a large gap in the scores have poor interoceptive awareness, and are also unable to judge other physiological changes like hunger (eg: Critchley 2004).

Peat and Muehlenkemp (2011) found that, among 214 college women, poor interoceptive awareness correlated with higher levels of body dissatisfaction and symptoms of eating disorders. Pollatos et al (2008) found that twenty-eight anorexic women from a self-help group in Munich, Germany, had an average 68% accurate in counting their heartbeat compared to 77% for gender, age, and education level-matched controls ($p<0.05$) [66]. So, a woman with poor interoceptive awareness may not sense any weight loss and perceive the body as the same even when emaciated. Also having "little internal input to anchor her sense of self, she can become overly concerned with small visual details, potentially resulting in a diminished body image" (Arnold 2012 p38).

Underlying poor interoceptive awareness is lower activation of the insula (Arnold 2012). Also, for example, Sachdev et al (2008) found little activity in the insula of anorexic women as compared to controls when shown pictures of themselves.

Interoceptive awareness can be part of a wider emotion dysregulation that has been observed in eating disorder sufferers. Muehlenkamp et al (2012) found that emotion dysregulation was a shared vulnerability for

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[63] Interoceptive awareness is the "ability to discriminate between individual sensations and to accurately respond to emotional states" (Pollatos et al 2008 p382).

[64] Accuracy of body perception is not always perfect in the general population (appendix 20A).

[65] Interoceptive awareness is also measured by self reports as in a sub-scale of the Eating Disorder Inventory (EDI-2) (Garner 1984).

[66] Heartbeat perception task for 25, 35, 45 and 100 seconds, and compared to ECG measurement.

disordered eating and non-suicidal self-injury among US college women. Emotion dysregulation is difficulty managing and expressing negative emotions, and related to this, interoceptive awareness is accurately recognising those emotional (and physiological) states.

The researchers got 118 women to complete questionnaires about eating disorders, body dissatisfaction, depression, and self-harm. Participants were divided into three groups for analysis - disordered eating only, self-harm only, or both.

APPENDIX 20A - BODY MODEL

Though propioceptive signals from muscles, for example, tell the brain where the body is positioned in space, information about the size and shape of body segments is not based on sensory information. The brain has developed a body model, which Longo and Haggard (2010) showed is objectively inaccurate.

The researchers focused on the body model of the hand. Participants placed their left hand flat on a table, this was covered over, and then they pointed to areas of the hand (eg: base of each finger, knuckles). Participants systematically underestimated the actual length of fingers (eg: over 30% for little finger), but overestimated the space between the knuckles (eg: over 100% between middle and ring finger). Thus the brain's model of the hand was shorter and wider than reality.

Interviewed about the findings, Longo said: "What we find for the hand is that the representation seems to be 'too fat'. If there's an implicit default representation of the brain to perceive the body as overly wide, then that could potentially account for the pattern you get with eating disorders" (Jha 2010).

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