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Supplement - Three Essays on Clinical Psychology

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CONTENTS

A. WHAT IS OBSESSIVE-COMPULSIVE DISORDER? SOME RECENT IDEAS

A1. CLASSIFYING OBSESSIVE-COMPULSIVE DISORDER

Obsessive-compulsive disorder (OCD) [1] is classed as a type of anxiety disorder in the classification of mental disorders (DSM-IV) (APA 1994) (appendix A1) [2], but it is different to the other disorders in that category, like panic disorder or phobias (table A1). There are those who recommend that OCD should be moved to a different category based on control of urges, like Gilles de la Tourette syndrome (TS) [3] (Wenner Moyer 2011) [4].

This view is supported by the co-morbidity of TS, for example, with OCD, and the apparent common link in serotonin malfunction, and brain abnormalities in the orbitofrontal cortex (Wenner Moyer 2011) [5].

	OCD	Anxiety Disorders
Onset	Childhood	Adulthood
Gender differences	No	More female
Substance misuse	No	Increased
Benzodiazepines	Not effective	Effective

Table A1 - Examples of key differences between OCD and other anxiety disorders (Black et al 2010).

[1] Approximately 2% of individuals have their lives impaired by OCD (Murphy et al 2010).
[2] One of the six types of anxiety disorder in DSM-IV (Murphy et al 2010).
[3] TS is characterised by multiple motor tics (eg: arm movements) including complex motor tics (eg: whole-body movements), and vocal tics (eg: coughing; snorting) (Grados 2009).
[4] There also advocates of OCD to be seen as an affective disorder, particularly as depression is a frequent co-morbidity disorder (eg: up to three-quarters of OCD sufferers) (Murphy et al 2010).
[5]Analysis of the genetic "types" of TS has distinguished (Grados et al 2008): TS with minor OCD symptoms, TS with frank OCD, and TS with OCD co-morbid with ADHD. While a similar analysis of OCD found (Nestadt et al 2008): OCD only ("OCD simplex"), OCD with tic disorders and anxiety (OCD and tics), and OCD co-morbid with affective disorders (co-morbid OCD). These findings are taken to suggest an overlap between OCD and TS (ie: common genetic basis) (Grados 2009).

Key to OCD is the idea that resisting a compulsion leads to increased anxiety and distress which can only be relieved by rituals. The obsessive thoughts are "not simply excessive worries about real-life problems, but typically have senseless content" while the "repetitive behaviour is clearly excessive or is not connected in any realistic way with the anxiety it is meant to relieve, a fact that the individual typically recognises" (Yaniv 2008 p406) [6]. The specifics of this process varies widely. Studies have categorised the many symptom divisions as (Murphy et al 2010):

a) Contaminated obsessions and cleaning compulsions [7].

b) Aggression, sexual, religious, and somatic obsessions [8] with checking-related compulsions.

c) Obsessions and compulsions related to ordering and counting.

d) Hoarding obsessions and compulsions (appendix A2).

Differences in OCD also arise in terms of the sufferer's insight about their behaviour (particularly with hoarders having less insight), and whether the condition develops in childhood or adulthood. Statistical analysis (eg: Schooler et al 2008) has suggested that these could be characteristics of two OCD sub-populations. One sub-population has more genetic relatives who also suffer, earlier age of onset, more severe symptoms, and greater co-morbidity than the other (Murphy et al 2010).

Some studies have distinguished OCD with hoarding from OCD without hoarding, partly because hoarding is more common in first-degree relatives of the former, suggesting a genetic or biological basis (Murphy et al 2010).

Obsessive-compulsive behaviours can occur with other disorders like TS, and anorexia nervosa with diverse symptoms. "These heterogeneous facets of the disorder have led to the search for OCD subtypes that might be associated with different aetiologies or treatment

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[6] Yaniv (2008) proposed "that compulsory repetitions substitute for mental health in the attainment of peace of mind: the lower the individual's mental health capital, the greater the number of repetitions he or she feels compelled to perform in order to relieve his or her anxiety" (p406).

[7] Sherman and Clore (2009) found a strong association between the colour white and good moral words, and between black and bad moral words using a version of the Stroop test. Participants' reaction times to recognise words like "honesty" were faster when printed in white than black ink, and the opposite for words like "greed". The effect was stronger for individuals preoccupied with purity and pollution (as in OCD) (Herbert 2009).

[8] Body dysmorphic disorder shares many common characteristics here (appendix A3).

responses" (Murphy et al 2010 p131) [9].

One alternative for future classification is a category called "obsessive-compulsive spectrum disorders" (OCSD) (Hollander 1993), which highlights a continuum of compulsive-impulsive disorders (Murphy et al 2010) [10] [11]. The spectrum can also include dimensions of uncertainty-certainty, and cognitive-motoric (features) (Black et al 2010). Another possibility is "obsessive-compulsive-related disorders" (Hollander et al 2009) [12].

If OCSD was included in future DSM classifications as a separate category [13], what disorders to include in it? A survey of 187 OCD experts by Mataix-Cols et al (2007) found more agreement on body dysmorphic disorder (BDD) [14] (72% agreed) and tic disorders (61%), for example, but less for impulse control disorders (33%), and addictions (5%), for instance (Phillips et al 2010). Phillips et al (2010), presenting the views of the DSM-V Anxiety, Obsessive-Compulsive Spectrum, Post-Traumatic, and Dissociative Disorders Work Group, recommended the category of OCSD for DSM-IV to include OCD, BDD, and tic disorders, and possibly hypochondriasis and obsessive-compulsive personality disorder, though they preferred the name "OCD-centric" to OCSD.

A2. IMMUNE SYSTEM

The different "versions" of OCD is also seen in cases of environmentally caused sudden-onset symptoms related to streptococcal infection, following brain injury, or among schizophrenics taking atypical anti-psychotic medication [15] (Murphy et al 2010).

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[9] Murphy et al (2010) concluded: "Thus it is apparent that OCD does co-occur with a wide variety of disorders, and certainly some share enough in common to be considered OCD-related" (p143).

[10] Obsessive-compulsive spectrum "refers to a group of disorders that are presumed to be distinct from, but related to, obsessive–compulsive disorder (OCD), and which are characterised by repetitive thoughts and/or behaviours" (Phillips et al 2010 p529).

[11] OCSD can include pathological gambling and kleptomania, and, from outside DSM-IV, compulsive buying, and compulsive sexual behaviour (Black et al 2010). However, Black et al (2010) argued that pathological gambling and compulsive buying would be better grouped with substance use disorders as both cravings and withdrawal symptoms are reported by their sufferers.

[12] Murphy et al (2010) preferred to call OCD as presented in DSM-IV, "uncomplicated OCD".

[13] DSM-IV has sixteen "supraordinate categories", up from 3 in DSM-I and 10 in DSM-II (Phillips et al 2010).

[14] This is a preoccupation with imagined or slight defect in physical appearance. Similar to OCD, there are "recurrent, time-consuming, intrusive, persistent , and unwanted thoughts" (Phillips et al 2010). But insight is less and delusional beliefs more common than in OCD (eg: over half of BDD sufferers vs 2% of OCD sufferers; Phillips et al 2010).

[15] Though whether OCD is a side effect of the drugs or that the drugs allow previously suppressed symptoms to emerge is debated (Murphy et al 2010).

In relation to the first of these, recent work has linked the development of OCD to the immune system. For example, mice missing a gene involved in immune functioning showed OCD-like behaviour in the form of excessive grooming. They spent almost twice the time grooming than controls including to the point of hair removal and self-inflicted wounds, and excessive grooming of cagemates (Greer and Capecchi 2002).

The immune system is also involved in the case of children who develop OCD or tic disorders after streptococcus infection ("strep throat") [16] [17] [18]. In fighting this infection, the immune system can attack cells in the cortical-basal ganglia circuit in the brain (known to be involved in OCD) [19]. Giedd et al (2000) compared 34 children with sudden onset obsessive-compulsive symptoms and/or tic disorders after streptococcal infection and 82 healthy controls using MRI. The obsessive-compulsive group showed an enlarged basal ganglia (figure A1) [20] [21].

This is an example of paediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS) (Swedo et al 1998) [22] [23] [24].

The first case suggests an underactive immune system in the rats while the second is an overactive one (Wenner Moyer 2011).

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[16] Technically, "group A beta-hemolytic streptococcal infections" (Garvey et al 1998).

[17] Other types of infection have been linked to sudden-onset OCD including toxoplasmosis, Borna disease virus, and Mycoplasma pneumoniae (Murphy et al 2010).

[18] Early case studies of bacterial infection causing tics were reported in 1929, but it was not until the 1990s that interest in streptococcal infection peaked in the USA (Garvey et al 1998).

[19] For example, an antibody for rheumatic fever has been found in 85% of PANDAS sufferers and all those with childhood-onset OCD (Garvey et al 1998).

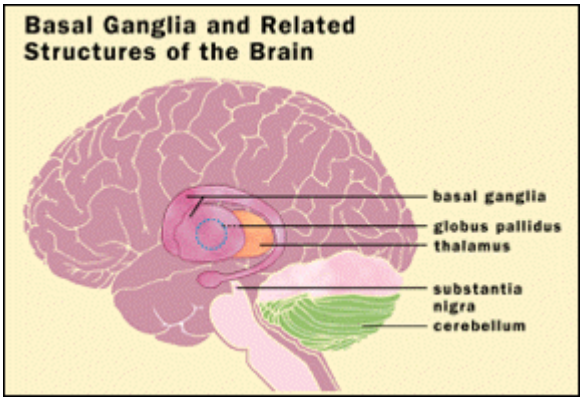
[20] Evidence for basal ganglia abnormality in OCD comes from the stop-signal reaction time (SSRT) test. An individual has to press a computer key as quickly as possible when a "go" signal is given on the screen. But, in some trials, there is an immediate "stop" signal (eg: beep) after the "go" signal. Some individuals are unable to stop themselves pressing the computer key after the stop signal, and they are more likely to suffer from OCD. This is known as process inhibitory control and is linked to the basal ganglia (Chamberlain et al 2005).

[21] On the other hand, Rosenberg et al (1997) reported smaller basal ganglia size in child OCD sufferers before treatment.

[22] OCD symptoms as part of PANDAS occur about three years younger than childhood-onset OCD, and appear "overnight" (Garvey et al 1998).

[23] The basal ganglia area of the brain is about 10% larger in children with PANDAS than healthy controls while there is no difference in total brain volume (Garvey et al 1998).

[24] Garvey et al (1998) emphasised that PANDAS is a post-streptococcal disorder, and obsessive-compulsive symptoms appear long after the symptoms of the infection have gone. This is important because PANDAS was initially diagnosed with great enthusiasm by some doctors including as a cause of autism and anorexia nervosa.



(Source: Henkel 1998; in public domain)

Figure A1 - Diagram of the brain showing position of basal ganglia.

A3. GENETIC BASIS

Interest in the hereditary of obsessions and compulsive behaviours began in 1930, though different diagnostic criteria were used back then (Grabe et al 2006).

In the search for a genetic basis to OCD, it has been found that identical twins have a higher concordance rate (ie: both twins suffer) than non-identical twins. van Grootheest et al (2005) found 29 published twin studies between 1929 and 2005, of which nineteen had less than 20 twin pairs (eleven studies being case reports of one pair) [25], and five had 100 twin pairs or more (Pauls 2010). For example, Bolton et al (2007), with 854 twin pairs, found a concordance rate for identical twins of 57% compared to 22% for non-identical twins using DSM-IV criteria.

Family studies report that sufferers have more genetic relatives with OCD than non-sufferers (Samuels 2009). For example, 10.3% of first-degree relatives [26] of OCD sufferers (probands) had "definite OCD" compared to 1.2% of relatives of non-OCD sufferers in a European study (Grabe et al 2006) (appendix A4).

Pauls et al (1995) also found significantly higher OCD in first-degree relatives compared to controls (10.3% vs 1.9%), and of sub-clinical OCD (not all symptoms for diagnosis of OCD) (7.9% vs 2.0%).

But Bellodi et al (1992) found only 3.4% of first-degree relatives showed symptoms, yet among relatives of

[25] Early studies tended to case reports, have small samples, use different diagnostic criteria, and not be blind to the co-twin (ie: whether they had OCD or not) (Pauls 2010).

[26] First-degree relatives show 50% of the same genes as the individual (proband) - siblings, parents, children (figure A2).

early-onset OCD sufferers (ie: before 14 years old) the figure was 8.8%.

Table A2 summarises two other family studies which focused on childhood-onset sufferers. Many family studies use adult sufferers which may exclude families with childhood-onset OCD that does not persist into adulthood (Grados 2009). Also males have earlier onset than females, which means that child samples are biased in that direction while adult samples tend to have more women (Hanna et al 2005).

Interestingly, OCD has been found to be higher among relatives of individuals with high levels of symmetry and ordering symptoms (Hanna et al 2005).

	Hanna et al (2005)	Rosario-Campos et al (2005)
Sample - OCD sufferers	102 relatives of 10-17 year-old sufferers	325 relatives of 106 under-18 OCD sufferers
Sample - controls	39 relatives	140 relatives of 44 controls
% relatives of OCD sufferers	22.5	22.7
% relatives of controls	2.6	0.9
Comment	No difference in tic disorders among relatives - 7.1% (OCD sufferers) vs 5.3%	Chronic tics greater among OCD sufferers' relatives (11.7% vs 1.7%). OCD higher in relatives of sufferers of OCD + tics vs OCD only (23.8% vs 14.9%)

Table A2 - Percentage of relatives with OCD.

Early family studies (prior to 1987; Pauls 2010) obtained data about family members from one or two informants. Technically, these are family history studies, and they tend to underestimate the rates of the disorder within families (Pauls 2010). Not surprisingly, the problem is their dependence upon the complete family knowledge of the informants. Often such studies defined OCD as present if the individual had been hospitalised for the disorder, and collected no information on relatives not hospitalised (Pauls 2010). Furthermore, individuals can be quite secretive about their symptoms. It is better to directly interview family members (known as direct interview family studies) (Pauls 2010).

Family and twin studies allow researchers to establish that there is a genetic component to the behaviour or disorder, while genetic linkage studies and

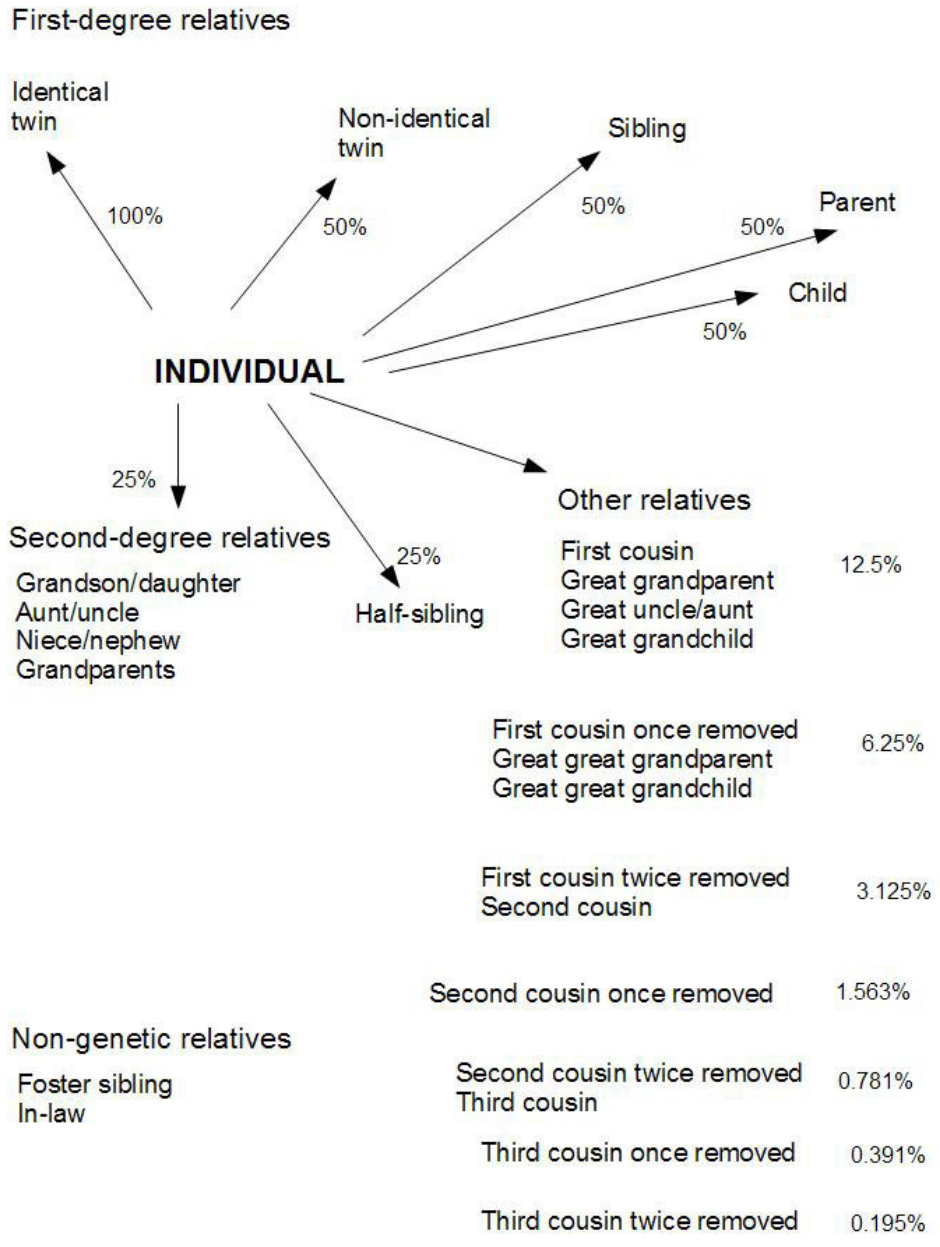


Figure A2 - Relatives and genes shared.

molecular genetic studies are the hunt for which gene(s) are involved.

Genetic linkage studies have suggested genes on certain chromosomes (eg: chromosome 3), and molecular genetic studies produced candidate genes (eg: glutamate transporter (SLC1A1) gene) (Samuels 2009) [27] [28]. When genes

[27] In terms of genetic discovery, "susceptibility" genes with minor or modest effect of a greater risk for the disorder is more likely than "major" genes - "gene networks rather than individual genes" (Gradus 2009).

are identified as relevant, they can be tested experimentally in knockout mice, for example, which are genetically engineered to lack the gene in question. But finding a genetic link for OCD is hampered by the variability and diversity in symptom manifestation between sufferers (Samuels 2009).

Modern genetic research also searches for endophenotypes. These are inherited physical intermediaries for a behaviour or disorder. Rather than OCD or the symptoms being inherited, something intervening is inherited (eg: reduced activation of several cortical areas of the brain; Samuels 2009), and this leads on to the OCD symptoms.

Genes may also be involved in neurochemical abnormalities. For example, Rosenberg and Keshavan (1998) were the first to suggest that abnormalities in the excitatory neurotransmitter glutamate was involved in OCD, and subsequent evidence has appeared (eg: magnetic resonance spectroscopy, genetic studies) (MacMaster 2010). Despite this, MacMaster (2010) recommended caution: "Clearly, a solitary neurochemical hypothesis of a psychiatric disorder is limited, as neurotransmitters do not operate in a vacuum" (p169).

OCD, like most mental disorders, is not 100% inherited, so environmental factors must be involved, like pre-natal (eg: excessive maternal weight gain during pregnancy), peri-natal (eg: prolonged labour), and post-natal (eg: childhood abuse) factors (Samuels 2009).

A4. APPENDIX A1 - CRITERIA FOR DIAGNOSING OCD

DSM-IV (APA 1994)

A. Either obsessions or compulsions:

Obsessions as defined by (1), (2), (3), and (4):

(1) recurrent and persistent thoughts, impulses, or images that are experienced, at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress;

(2) the thoughts, impulses, or images are not simply excessive worries about real-life problems;

(3) the person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action;

[28] Over eighty candidate gene studies have been published in the 21st century (Pauls 2010).

(4) the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion).

Compulsions as defined by (1) and (2):

(1) repetitive behaviours (eg: hand washing, ordering, checking) or mental acts (eg: praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly;

(2) the behaviours or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviours or mental acts either are not connected in a realistic way with what they are designed to neutralise or prevent or are clearly excessive.

B. At some point during the course of the disorder, the person has recognized that the obsessions or compulsions are excessive or unreasonable. **Note:** This does not apply to children.

C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.

A5. APPENDIX A2 - HOARDING

Compulsive hoarding is "excessive collecting and saving behaviour that results in a cluttered living space and significant distress and impairment" (Grisham and Norberg 2010 p233). It is a diagnostic criteria for obsessive-compulsive personality disorder in DSM-IV-TR (APA 2000) (though the appropriateness of this is disputed; eg: Saxena 2007). Some researchers feel that is better seen as a symptom or sub-type of OCD (eg: one-third of individuals with OCD show some hoarding behaviour; Grisham and Norberg 2010). Individuals with OCD and hoarding are more likely to have symmetry obsessions, and counting and/or ordering compulsions, and greater illness severity (Samuels et al 2007).

Other researchers want to see hoarding as a separate clinical syndrome. For example, individuals with monosymptomatic hoarding (ie: without OCD) are different from those with OCD and hoarding in terms of what is hoarded. The latter group being more likely to hoard bizarre items. Also hoarding is not so much about reducing anxiety (as in rituals and compulsions in OCD), but similar to impulse control disorders, like pathological gambling (Grisham and Norberg 2010).

The prevalence of hoarding is between 2-5% of the population, and more often men are hoarders (Grisham and Norberg 2010).

Table A3 summarises some of the characteristics of hoarders (Grisham and Norberg 2010).

- More likely to have a first-degree relative who hoards than non-hoarders (genetic ?).
- Significant stressful life event linked to onset.
- More likely to have health problems including obesity.
- Links to frontal lobe dysfunction.
- Decision-making problems generally.

Table A3 - Key characteristics of hoarders.

A6 - APPENDIX A3 - BODY DYSMORPHIC DISORDER

Body dysmorphic disorder (BDD) is a somatoform disorder in DSM-IV [29], where an individual is preoccupied with a slight or imagined defect(s) of physical appearance, usually focused on the skin, hair, or nose (though the concern can relate to any part of the body [30]). It was noted in the late 19th century as a "fear of deformity" by Enrico Morselli, and called "dysmorphophobia" [31] [32] (Bjornsson et al 2010).

Surveys of the prevalence of BDD find rates that vary from 1-2% to 13% in the general population, but over half among those seeking cosmetic surgery (Bjornsson et al 2010). There is a slightly higher rate among women than men, and age ranges from as young as five years old to 80 years old (Bjornsson et al 2010). As with anorexia, many BDD sufferers report having been teased about their appearance as a child or adolescent (Rytina 2008).

One issue is whether the perceived ugliness is delusional (ie: whether the sufferers have insight about their thoughts). For example, Phillips et al (2006) found that about one-third of sufferers could be classed as delusional (ie: certain that their beliefs were accurate). This is less insight than OCD sufferers (Bjornsson et al 2010).

BDD can show similarities to OCD in terms of repetitive, time-consuming, compulsive behaviours. In response to a mirror, sufferers can excessively check themselves, foe example. Other repetitive behaviours include excessive grooming (eg: combing hair for long periods), tanning, seeking reassurance about appearance from others, or excessive shopping for beauty products (Bjornsson et al 2010) [33].

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[29] In ICD-10, it is grouped with hypochondriasis.

[30] A specific type of BDD with muscle dysmorphia, often found among body builders, is the belief that the body is not muscular enough.

[31] Also known as "Thersites complex" after the "ugliest man in the Greek army" in Homer's "Iliad" (Rytina 2008).

[32] Dysmorphophobia was used in DSM-III (1980), the first appearance of the disorder here, but this was changed to BDD in DSM-IIIR (1987).

[33] Interestingly, these behaviours are encouraged in consumer societies that focus upon physical appearance and the selling of products in relation to that.

BDD is debilitating for the sufferers with nearly one-third of individuals completely housebound for short periods, at the extreme. Suicidal thoughts and attempts are high (eg: 80% and 25% respectively in one year) (Bjornsson et al 2010).

Studies have found cognitive processing bias among BDD sufferers including:

- Visual facial processing based on detail rather than holistic (Feusner et al 2007).
- Overestimation of the attractiveness of others' faces (Buhlmann et al 2006).
- Interpretation of neutral emotional expressions as threatening (eg: contempt) (Buhlmann et al 2006).

A7. APPENDIX A4 - DIFFERENCES RATES OF OCD IN FAMILY STUDIES

Different studies report different rates of OCD among relatives of sufferers. There are a number of reasons for this related to the methodology of the study, as shown by three studies.

1. Grabe et al (2006)

Where - Germany as part of the German Epidemiologic Network for OCD Studies (GENOS).

OCD sufferers - Grabe et al had two samples of OCD sufferers:

i) Community sample (Co) - 15 untreated OCD sufferers (mean age 44.8 years old; mean age of OCD onset 17.1 years old; 13% male) from a random sample of the population in Western Pomerania, north-east Germany. They were recruited by five self-reported obsessive-compulsive behaviours in a health survey (checking, washing, need for symmetry, obsessions, and aggressive impulses). Fifty-eight first-degree relatives were recruited (table A4).

ii) Clinical sample (Cl) - 90 outpatients from four psychiatric university hospitals (mean age 38.3 years; onset 17.7%; 41% male), and 285 of their first-degree relatives.

Comparison sample (control) - 70 non-OCD sufferers from Western Pomerania and the areas around the psychiatric university hospitals (mean age 42.1 years old; 49% male), and 247 of their first-degree relatives.

	Co (N = 58)	Cl (N = 285)	Control (N = 247)
Parents	25 (43%)	166 (58%)	116 (47%)
Siblings	9 (16%)	88 (31%)	78 (32%)
Children	24 (41%)	31 (11%)	53 (21%)

Table A4 - Nature of relatives in study.

Diagnosis - The relatives were diagnosed via face-to-face or telephone interviews using DSM-IV criteria. "Definite OCD" met all the criteria, while sub-clinical OCD was based on all the symptoms except more than one hour day on rituals. Interviewers blind to relatives' group, except in clinical group where patients known to interviewers (who were psychiatrists, clinical psychologists, or doctoral-level clinical researchers).

Findings:

- At least one relative with "definite OCD" - 33% (Co), 16% (Cl), 4% (control) (X^2 test; $p = 0.004$).
- At least one relative with sub-clinical OCD - 60% (Co), 15% (Cl), 10% (control) ($p<0.001$).
- Lifetime diagnosis of OCD - 10.3% (Co), 5.6% (Cl), 1.2% (control) ($p = 0.008$).
- Lifetime diagnosis of sub-clinical OCD - 15.4% (Co), 4.1% (Cl), 3.0% (control) (not significant).
- Overall, OCD was over five times more common in the Cl sample than the controls (odds ratio 5.6), and over twelve times more so in the Co sample than the controls (odds ratio 12.6).

2. Hanna et al (2005)

Where - Michigan, USA.

OCD sufferers - 35 cases (10-17 year-olds) from clinics in the state, and 102 first-degree relatives and 309 second-degree relatives (table A5). Thus only individuals seeking help.

	OCD (N = 102)	Control (N = 39)
Parents	69 (68%)	34 (87%)
Siblings	33 (32%)	5 (13%)

Table A5 - First-degree relatives in the study [34].

[34] Younger siblings were interviewed, possibly prior to their development of OCD, and this could

Comparison sample - 17 individuals recruited by advertisements in local newspapers and at the University of Michigan with 39 first-degree and 150 second-degree relatives.

Diagnosis - Mainly direct interview using DSM-III-R criteria (APA 1987) [35]. Of OCD group, 98 of 102 first-degree and 14 of 309 second-degree relatives interviewed as opposed to 38 of 39 and 1 of 150 respectively in the control group. Otherwise family informants were asked.

Findings:

- "Definite OCD" - 22.5% of first-degree relatives of OCD sufferers and 2.6% of controls' relatives (p = 0.021) (odds ratio 11.06).
- Sub-threshold OCD - 27.4% vs 2.6% (p = 0.01) (odds ratio 14.38).
- Second-degree relatives and lifetime OCD - 1.6% of OCD sufferers and 0.7% of controls. But because most of these relatives were not interviewed, the researchers felt that the figures many be underestimates.

3. Rosario-Campos et al (2005)

Where - USA.

OCD sufferers - 106 (under 18s) patients from the OCD clinic at Yale Child Study Centre, New Haven, Connecticut, and 325 first-degree relatives (table A6).

Comparison sample - 44 age-matched non-OCD sufferers recruited by the random-digit dialling procedure, and 140 of their relatives.

	OCD (N = 325)	Control (N = 140)
Parents	202 (62%)	78 (56%)
Siblings	123 (38%)	62 (44%)

Table A6 - First-degree relatives in the study.

Diagnosis - Interviews by trained interviewers using DSM-

underestimate the rate as could older relatives forgetting early life symptoms.

[35] DSM-III-R does not require the obsessions to cause marked anxiety and distress (as DSM-IV does).

IV criteria and blind to group. Most were direct interviews (316 of 325 in OCD group and 106 of 140 in control group) [36].

Findings:

- OCD - 22.7% of sufferers' relatives and 0.9% of controls' (odds ratio 32.5).
- Sub-clinical OCD - 29.2% and 2.4% respectively.
- Correlation between age of onset between sufferers and relatives (Pearson's; r = 0.36; p = 0.004).

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B. QUESTIONING SEASONAL AFFECTIVE DISORDER

B1. MEASURING SAD

Seasonal affective disorder (SAD) describes recurrent depression that occurs at the same time each year, usually during the winter months (Rosenthal et al 1984a). It is usually diagnosed based on a diagnosis of depression first, and then the seasonal pattern is determined.

The Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al 1984b) is a self-reported questionnaire designed to measure SAD [37]. For example, in one of the first postal surveys in the USA to use it, 1234 individuals in New York city, Montgomery County (Maryland), Nashua (New Hampshire), and Sarasota (Florida) were contacted by Rosen et al (1990), who calculated a prevalence of 7.4% for SAD with another 10.7% classed as sub-syndromal (ie: close to the threshold for diagnosis).

Other studies have found less prevalence (eg: Blazer et al 1998). Blazer et al (1998) analysed data from the National Co-Morbidity Survey (NCS), which sampled over 8000 15-54 year-olds from 48 states in the USA in 1990-2. Diagnosis was based on an interviewer-administered questionnaire (modified version of the Composite International Diagnostic Interview; CIDI).

Certain criteria were used for the seasonal pattern diagnosis:

- Onset of depression at a similar time each year.
- Remission at a similar time each year.
- At least three years of this pattern with two of them being consecutive years.
- Seasonal episodes of depression outnumber non-seasonal episodes.
- Narrow definition - two-thirds of depressive episodes begin and end at a similar time of year.
- Broad definition - half of depressive episodes begin and end at a similar time of year.
- Minor and major depression diagnosed based on DSM-III-R criteria.

[37] It contains questions like: "At what time of the year do you" - eg: feel best; feel worst, and "To what degree do the following change with seasons" - eg: mood; energy levels.

In the total sample, 1382 individuals had a lifetime history of major depression and 2330 minor depression. In the former group, 248 individuals reported that most episodes started at a similar time each year and 382 individuals with minor depression. When the broad definition and all criteria were applied, 34 individuals with major depression were diagnosed as SAD (0.4% of total sample) and 49 individuals with minor depression (giving a total prevalence of 1.0%). With the narrow definition, the figures were 0.3% (22 individuals) and 0.7% (56 individuals) respectively. Seasonal minor depression was significantly more common among women than men, whereas seasonal major depression was significantly less common for them.

The prevalence rate of SAD is lower than previous studies because of stricter criteria, and interviewer-based diagnosis rather than the self-reported SPAQ. This meant that individuals could be "pinned down" about when the symptoms actually began and ended. Also DSM-III-R criteria for depression were used which is not the case with SPAQ (Blazer et al 1998).

SPAQ can over-estimate the amount of SAD (Michalak et al 2001). For example, in Scotland, Eagles et al (1998) reported a prevalence of 9.3% using SPAQ, but only 3.5% based on diagnostic interviews (Eagles et al 1999).

Michalak et al (2001) used both SPAQ and diagnostic interviews with their north Wales sample. Initially, a sample of 1250 adults (18-64 years old) in the Glyndwr council district [38] completed a sub-scale of the SPAQ ("global seasonality score") in 1997-8. The score range available was 0 to 24, and eleven or more was taken as the cut-off point. Sixty-five respondents (5.3%) were above the cut-off score, and they were offered a diagnostic interview. Of the 55 who agreed to it, twenty-five were diagnosed with SAD [39] [40]. This gave a prevalence rate of 2.4% of the total sample.

B2. CRITICAL OF SAD

Hansen et al (2008) observed that "the diagnosis 'Seasonal Affective Disorder' does not exist in any of the two international classification systems that are used in psychiatry. Nevertheless, the concept of SAD, or 'winter depression' as it is more commonly called, leads its own life, and has crept into the mind of the general

[38] This district does not exist today.

[39] Diagnosis was based on the Structured Interview Guide for the Hamilton Depression Rating Scale - Seasonal Affective Disorder version (SIGH-SAD) (Williams et al 1992).

[40] As a comparison group, 23 individuals with SPAQ scores below eleven were interviewed, and none of them were diagnosed with SAD.

public and the medical community as something real, and presumably an important public health problem" (p121).

It was generally believed that SAD was more common in higher latitudes (latitude hypothesis). This is challenged by the fact that SAD is not higher in extreme latitudes (eg: northern Norway) with up to two months of complete darkness (Hansen et al 2008) [41]. Rosenthal (1993) responded that researchers in northern Norway could have low energy levels during the winter which limited their ability to study SAD. This comment spurred on, northern Norwegian researchers, Hansen et al (2008) who re-analysed data from Tromso. Data collected in 1979-80 showed a self-reported depression rate between November-March of 10% for men and 12% for women (the same as figures from the USA at lower latitudes). Five studies from Norway "consistently indicate that living in almost complete darkness for two months does not increase the prevalence of depression above the expected population level of depression in general" (Hansen et al 2008 p122).

In fact, Lund and Hansen (2001) found that the prevalence of SAD was higher in June (continuous daylight) than January in northern Norway, and peaked in March when day and night are of equal length.

Also Magnusson and Stefansson (1993) reported lower rates of SAD in Iceland (higher latitude) than those in the USA in Rosen et al (1990). Furthermore, the rate of SAD among individuals of Icelandic origin living in Manitoba, Canada was only marginally higher than the Florida sample in Rosen et al (1990) (Magnusson and Axelsson 1993). This has led to the focus on genetics as the main cause of SAD.

However, Axelsson et al (2006) showed that genetics alone was not the cause by comparing two groups of genetically similar individuals from Iceland living at the same latitude in Manitoba, Canada. One group (n = 210) lived in Winnipeg, and they showed a rate of 4.8% for SAD using the SPAQ, while 252 individuals in the Interlake district had a rate of 1.2% (significantly lower). Axelsson et al (2006) concluded: "No one seriously doubts the importance of light-deprivation as a causal factor in SAD. But it is clear that factors other than light must not be overlooked in our efforts to understand, and to treat, seasonal affective disorder" (p20).

Hansen et al (2008) were critical of SAD as a concept. For example, the initial description of it was

[41] A complaint called "mid-winter insomnia" has been reported by a number of Norwegians during the dark period. This is where individuals are not sleepy in the evening and cannot fall asleep (Hansen et al 2008).

based upon self-selecting volunteers who responded to an article in a US newspaper by Rosenthal ("mass-media recruitment"). Furthermore, sufferers of SAD are heavy users of health care services which can distort the prevalence if that type of sample is used.

Hansen et al (2008) found that 45 of 66 English-language prevalence studies of SAD had used SPAQ, and only six of the others employed DSM-criteria.

SPAQ has been criticised for poor discriminability. For example, Mersch et al (2004) found that SPAQ could only identify 44% of SAD sufferers from a group including SAD sufferers, non-seasonal depression outpatients, non-depressed outpatients, and controls in the Netherlands. While Thompson et al (2005) felt that SPAQ produced false positives. Hansen et al (2008) coined the term "SPAQ-iasis" ("a constructed disease") for what a high SPAQ score showed: "SPAQ does not measure peoples' general response to the season, as it purports to do. Instead, SPAQ score is probably heavily influenced by the weather condition at the time of completion" (p125). Talking about an Icelandic study (Magnusson 1996), Hansen et al (2008) concluded: "you could put up an equally strong argument for SPAQ as an instrument for identifying seasonal anxiety disorder as for seasonal affective disorder. What Magnusson's study really demonstrates is that SPAQ may be used to identify subjects with a high level of mental distress who feel worst during the winter months, regardless of type of distress" (p125).

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C. MENTAL ILLNESS AND IRISH IN BRITAIN

C1. PREVALENCE RATES

That there are ethnic, cultural and racial differences in mental disorders is well-established [42], but, in Britain, it has been "limited by an almost exclusive identification of ethnicity with skin colour" (Bracken et al 1998), thereby assuming that the White group, for example, is homogenous, and ignoring the experience of Irish people in Britain.

In Britain, Irish-born or those with Irish-born parents together make up about 5% of the population (Bracken et al 1998), but they are over-represented in the mental health system. For example, rates of mental hospital admission for disorders like schizophrenia are more than twice as likely for individuals born in the Irish Republic living in England than English-born White individuals (Cochrane and Bal 1989) (figure C1).

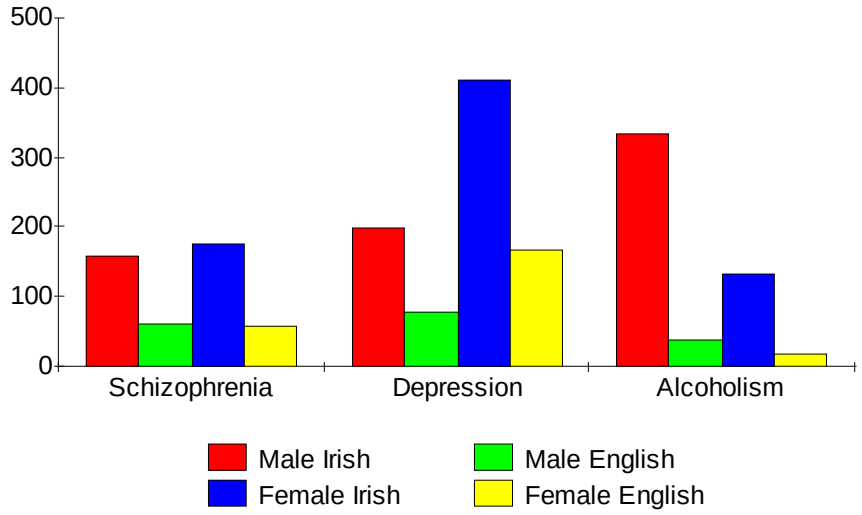
There are two possible explanations for these figures (Bracken et al 1998):

1. The figures show true differences among the Irish (appendix C2) - High mental hospital admission rates have been reported in Ireland and among Irish immigrants to North America. But, this is not found, where comparable cross-cultural cases and definitions are used (eg: World Health Organisation study of twelve cities worldwide including Dublin; Sartorius et al 1986).

2. The figures are the result of the social situation of Irish people in Britain - Many Irish individuals in Britain are migrant workers (without their families), in unskilled work (which is erratic and unhealthy), and experiencing discrimination (Bracken et al 1998).

More recently, the EMPIRIC (Ethnic Minorities Psychiatric Illness Rates in Community) study sampled 64

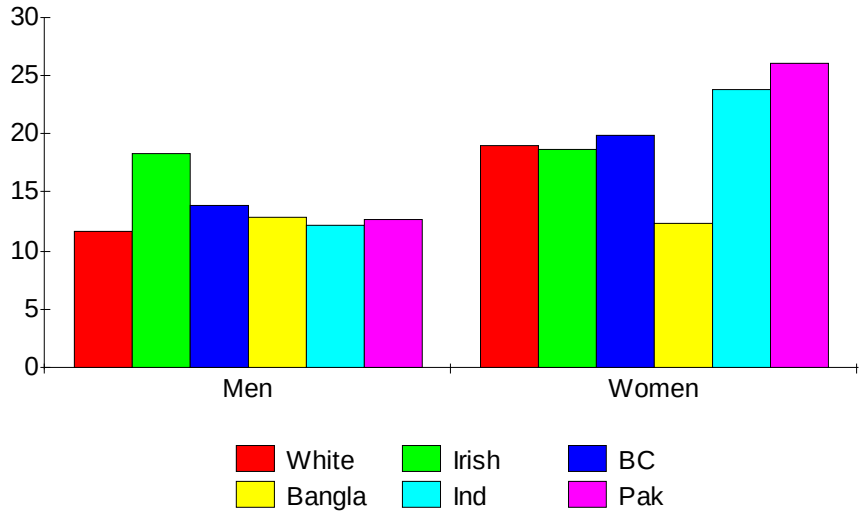
[42] For example, Takei et al (1998) followed 81 patients (49 White and 32 Afro-Caribbean) first admitted to hospital with functional psychosis in 1973-4 in the Camberwell area of south London. Over the follow-up period, the Afro-Caribbean individuals diagnosed with schizophrenia had significantly more hospital re-admissions than the White individuals with the same diagnosis (mean: 5.3 vs 3.4). They were also over twenty times more likely to have experienced an involuntary admission. Chorlton et al (2011) highlight the methodological issues with such studies (appendix C1).



(Source: Bracken et al 1998 table 2 p103)

Figure C1 - Rates of mental hospital admissions per 100 000 population in England in 1981 for three disorders based on place of birth.

000 addresses in England about ethnicity and common mental disorders (anxiety and depression) (Weich et al 2004). Irish men (especially 35-54 year-olds) had the highest prevalence (18.4%) among the ethnic groups, while Irish women had a similar rate (18.6%) but it was less than some other groups (figure C2). Overall, compared to the White group, the Irish group were twice as likely to suffer from common mental disorders.



BC = Black Caribbean; Bangla = Bangladeshi; Ind = Indian; Pak = Pakistani

(Source: Weich et al 2004 table 2 p1546)

Figure C2 - Prevalence of common mental disorders based on ethnicity and gender.

Using data from the EMPIRIC study as well, Crawford et al (2005) reported that older members (55-74 years old) of ethnic minorities were less likely to think about taking their own life compared to White English of the same age, though Irish men of that age were slightly higher. For example, Irish individuals of this age group had an odds ratio of 0.44, Black Caribbean 0.16, and Indian 0.32 compared to White (= 1.00). But the odds ratio for Irish men was 1.09 and 0.61 for women aged 55-74 years old.

C2. APPENDIX C1 - CHORLTON ET AL (2011)

When observations are made about behaviour (eg: the differences in incidence and prognosis of psychosis of Black Caribbean populations in the UK compared to the White populations), it is necessary to test them rigorously in studies. But not all studies are equally good in terms of methodology, and/or contradictions in the findings can occur which require a systematic review.

In relation to the example, Chorlton et al (2011) searched a number of databases (eg: PsycInfo) up to July 2010 using a number of keywords, like "Caribbean" and "racial and ethnic differences". This produced 2164 results, which were reduced to fourteen relevant studies based on certain criteria:

- 2048 excluded as not relevant to the topic.
- 69 excluded as relevant to the topic, but not the research focus (eg: non-UK based).
- 33 excluded as relevant to the topic, but too specific (eg: sample in secure hospitals).

Then the 14 studies were scored on criteria for methodological quality (out of 12):

i) Sample size - 0 (<15 ethnic minority individuals) to 3 (>500).

ii) Source of information about patients - 0 (case notes only [43]) or 1 (patient interview and case notes).

iii) Adjustment for confounding variables - 0 (none) to 2 (diagnosis, disease severity, age, gender) [44].

iv) Diagnostic criteria for psychosis - 0 (one set) or 1 (used ICD and DSM latest versions).

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[43] These are "often unreliable and inaccurate" (Chorlton et al 2011).

[44] "Failure to control for confounding variables may have meant that studies that already had small samples were more likely to lose [statistical] power" (Chorlton et al 2011).

v) Definition of ethnicity - 0 (indirect evidence; eg: name) to 2 (self-reported) [45].

vi) Analysis in relation to ethnicity - 0-2 (eg: ethnic groups appropriately or inappropriately combined) [46].

vii) Outcome measures - 0 (1 factor) or 1 (multiple factors).

From this scoring system, five studies were classed as high quality methodological studies (score 8-11), six medium quality (score 3-7), and three low quality (score 0-2). The studies varied on other methodological aspects including:

- Definition of psychosis sample - eg: "first-episode" vs "first admission to hospital" [47] ("dissimilar samples"; Chorlton et al 2011).
- Prospective or retrospective collection of data.
- Follow-up period - varying from 1-18 years.
- Number of participants dropping out by follow-up (ie: follow-up rate) - eg: 168 participants at baseline down to 113 at follow-up in one study vs no loss in another one.
- Origin of sample - eg: London vs Nottingham.

After this analysis of the study quality, Chorlton et al (2011) concluded that there was "currently insufficient evidence of a high enough quality" to say whether there is a difference in the course and outcome of psychosis among Black Caribbean individuals as compared to other ethnic groups in the UK.

This may mean that differences do or do not exist. For example, four studies reported that Black Caribbean individuals with psychosis were more likely to experience compulsory hospitalisation than Whites, while two studies did not find any difference. The latter two studies used broad categories of ethnicity (eg: Black and White), which may have obscured differences (Chorlton et al 2011).

C3. APPENDIX C2 - CULTURAL DIFFERENCES AND MENTAL DISORDERS

Kelleher (1972) found that a sample of Irish

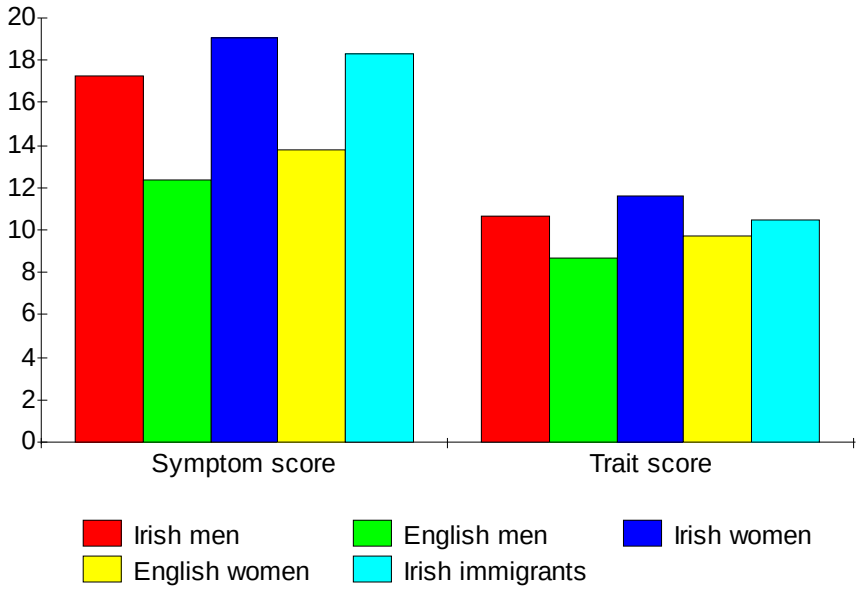
[45] Some studies used "official" UK census categories, while others chose their own - eg: "White British"/"Afro-Caribbean" vs "White"/"Black"/"Asian"/"Other".

[46] Also good studies should involve data analysis blind to ethnicity.

[47] These individuals may have experienced the illness for many years and had treatment.

accident cases on orthopaedic wards [48] (in four hospitals in Cork) had a significantly higher score on the Leyton Obsessional Inventory (LOI) [49] (Cooper 1970) than an English sample (in two London hospitals) [50] (figure C3).

Among the Irish group, individuals from rural Cork had a higher score than those from Cork city (Tyrer et al 1998). Kelleher (1970) explained this difference as due to certain factors like the "house-proud" nature of the "Irish country housewife", and the personal insecurity and isolation of rural life. In the latter case, obsessional symptoms were a defence against these concerns. Generally, Kelleher felt that anxiety and guilt over sexual relations in relation to Catholicism might partly explain the higher LOI scores in the Republic of Ireland.



(Source: Kelleher 1970 tables 1 and 2 p36)

Figure C3 - Mean LOI scores.

Scott et al (1982) confirmed the national differences, and that a Scottish sample was lowest of the

[48] Advantages of using these groups included being non-psychiatric populations, and having time on their hands to complete questionnaires, but they may not be comparable between countries nor representative of the general population in each country (Kelleher 1972).

[49] This has 69 questions divided into 46 symptom-related and 23 trait-related. The questions include: "are you very strict about keeping the house always very clean and tidy?", "do you like furniture or ornaments to be in exactly the same place always?", and "do you have difficulty making up your mind?" (Kelleher 1972).

[50] Seventy-three individuals in Cork (51 male/22 female), 77 (48 male/29 female) in London, and 12 Irish immigrants (nine male/three female) in London hospitals. Individuals too ill or suffering from disturbances of consciousness from an accident were excluded by the nursing staff from the study.

three countries.

Comparisons among "cultural or ethnic geographically defined groups in rates of psychopathology (either generally or confined to specific disorders) requires attention to a range of methodological issues" (Rutter and Nikapota 2002 p280). These include comparability in sampling and assessment of the disorder, as well as the "need to ensure sensitivity to concepts and features that may be much more applicable in one group than others" (Rutter and Nikapota 2002 p280). For example, migrants have "chosen" to move to a new country, which may limit their comparability to a random sample of the indigenous population (ie: the migrants are a self-selected group).

Many studies use self-reports of mental disorders which are not independently verified. Leaving aside lying, individuals may respond differently to the questions and terms used (eg: "have you been anxious recently?").

In relation to the concept of culture, Rutter and Nikapota (2002) highlight two important points:

> First, although culture tends to be conceptualised as all-encompassing, there are marked individual variations within cultures. Many people feel part of several different cultures. Secondly, cultures are far from static. They represent dynamic social processes that reflect a complex changing mix of ethnic, class, gender, religious and societal influences (p283).

There is a wider debate about the universality of mental disorders. In DSM-IV, an appendix includes disorders called "culture-bound syndromes". This refers to disorders, like "dhat" [51] or "hwa-byung" [52], which appear to exist only in some cultures [53].

But are these local names for universal conditions or specific disorders to a particular culture? For example, in the former case, indigenous seal hunters in Greenland can experience "kayak angst" (sudden panic out in the ocean, and the need to return to land). This seems quite similar to the symptoms of panic disorder with agoraphobia in DSM-IV (Lilienfeld and Arkowitz 2009). On the other hand, "2-D love", reported in Japan recently,

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[51] Anxiety, fatigue, and fear about loss of semen, mainly reported in India and Pakistan (Lilienfeld and Arkowitz 2009).

[52] Insomnia, fatigue, and physical symptoms attributed to the suppression of anger in Korea (Lilienfeld and Arkowitz 2009).

[53] Culture-bound syndrome is often used in the West with reference to "exotic" disorders, but there are disorders limited to Western societies, like chronic fatigue syndrome or anorexia nervosa (Rutter and Nikapota 2002).

seems unique. This is where men become attracted to animated female characters, and behave as if in a relationship with them. While "windigo" (extreme anxiety, and fear of cannibalising others) is reported among Native Americans, particularly in Canada (Lilienfeld and Arkowitz 2009).

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