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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://psychologywritings.synthasite.com/ and http://kmbpsychology.jottit.com.

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1. CHANGING VIEWS ON SCHIZOPHRENIA

1.1. SCHIZOPHRENIA OR SCHIZOPHRENIAS

The variety of symptoms [1] and experiences of schizophrenia led Bleuler (1911) to say in 1911 that "it is not a disease in the strict sense, but appears to be a group of diseases ... therefore we should speak of schizophrenia in the plural" (quoted in Jablensky 2015). One hundred years or so later "we still do not know whether schizophrenia is a single disease process with pleiotropic [2] manifestations at the level of cerebral organization and symptoms, or a collection of aetiologically divergent, only marginally overlapping, disorders" (Jablensky 2015 p105).

The current focus is upon the specific genes behind schizophrenia, for instance with genome-wide association (GWA) studies [3]. For example, Schizophrenia Working Group of the Psychiatric Genomics Consortium (2014) found 128 associations from 108 genomic loci (different places on the genome) from over 36 000 individuals with schizophrenia and 113 000 controls. A number of the associations related to genes that were expressed in the brain and in neurotransmission.

Jablensky (2015) noted the limitations of GWA studies:

i) Many of the associations are weak with so much data.

ii) The "number of detected significant associations is positively correlated with the ever-increasing sample sizes" (p105).

[1] Some of the symptoms are experienced in everyday life (appendix 1A).
[2] One gene affecting multiple unrelated traits.
[3] This method looks for genes that are common to sufferers and different to controls.

iii) Any findings are general and not predicting of individual disease risk.

Such studies end up with a "missing heritability" (ie: genes only explain some of the variation between sufferers and controls) (Jablensky 2015). Arnedo et al (2015) argued that "the heritability of schizophrenia is not 'missing' but is in fact distributed over a large number of genotypic-phenotypic sub-sets..." (ie: "schizophrenia is a composite collection of partially overlapping but distinct disorders underpinned by separate genetic networks") (Jablensky 2015 p106). In other words, schizophrenias. This was based on the analysis of cases and controls for clusters of interconnected single-nucleotide polymorphisms (SNPs), of which over seven hundred were found. Different SNP sets were associated with eight different "clinical syndromes". This study has been challenged by other researchers (Jablensky 2015).

Both studies focused on the genome assuming that the answer is there to be found. Schizophrenia is not without its critics in terms of diagnosis, and so the search for genes for it may be a search for genes for a "disease" that is actually multiple conditions and does not exist in any single disease entity sense.

1.2. PROGRESSION OF SCHIZOPHRENIA

The progression of schizophrenia has a number of stages (Puskulic et al 2015) [4]:

i) Non-specific risk factors.
ii) At-risk mental states (ARMS) or ultra-high risk (UHR) of psychosis.
iii) First episode of psychosis (FEP).
iv) Chronic course (including relapses).

In recent years, there has been a "paradigm shift" from the management of the chronic course to focus on intervening during the "early period" (Birchwood et al 1998) or "critical period" (ie: before and at the FEP) (Piskulic et al 2015) [5].

Piskulic et al (2015) found varying success for interventions and treatments at these points in the progression of schizophrenia in their literature review.

[4] There is a debate over the distinction between neurology and psychiatry, and schizophrenia is at the heart of it (appendix 1B).

[5] A meta-analysis of 2502 at-risk individuals found that 18% had developed a psychotic disorder by six months, and 36% by three years (Fusar-Poli 2012).

1. Pharmacological treatments - Anti-psychotics are "generally avoided" until the FEP, and then the response rate varies between 40-90%.

2. Psychological therapies - Cognitive-behavioural therapy (CBT) can be "moderately effective in reducing transitions to psychosis", while the effectiveness of family therapy in the ARMS is "yet to be established" (Piskulic et al 2015).

3. Cognitive remediation therapy - Focusing on cognitive deficits, this treatment has been "encouraging" with "early evidence suggesting favourable effects" (Pislukic et al 2015).

4. Psychosocial therapies - eg: "beneficial effects" for computer-based techniques to help maintain functionality (ie: "everyday life").

Treatments and interventions in "early schizophrenia are qualitatively different from preventing future cases of the disorder or from early symptom identification, but may nevertheless help reduce morbidity in those who continue through to the more chronic course" (Piskulic et al 2015 p140).They are beneficial in the first two years in particular, but not necessarily in the longer term (Pislulic et al 2015).

Individuals with schizophrenia have been reported as having lower levels of omega-3 and omega-6 polyunsaturated fatty acid (PUFA) (involved in brain cell development), which could be rectified with fish oil capsules (Amminger et al 2015) [6].

A double-blind, placebo-controlled trial showed the benefits of a twelve-week treatment with first episode psychosis at one year (Amminger et al 2010), while Amminger et al (2015) confirmed the effectiveness in a follow-up at seven years.

Originally, 81 13-25 year-olds defined as at-risk of psychosis [7] in Vienna were given a daily fish oil capsule or a placebo for twelve weeks. Amminger et al (2015) found seventy-one of them at follow-up. The outcome measure of "conversion to psychotic disorder" [8] was 10% of the treatment group and 40% of the placebo group, and the lifetime prescription of anti-psychotics was also lower in the treatment group (30 vs 55%).

Amminger et al (2015) commented "Neuronal circuits

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[6] The focus on the brain including interest in inflammation that affects brain development (appendix 1D).

[7] Eg: brief, intermittent psychotic symptoms.

[8] Based on cut-off scores on the Positive and Negative Syndrome Scale (PANSS).

in the brain are shaped during critical periods of development. It is therefore possible that an effective intervention during a circumscribed period of increased susceptibility can have longer-term effects. Omega-3 PUFAs provide a range of neurochemical activities via modulation of neurotransmitter (noradrenaline, dopamine and serotonin) reuptake, degradation, synthesis and receptor binding, as well as anti-inflammatory and anti-apoptotic effects [9], and the enhancement of cell membrane fluidity and neurogenesis. While the mechanisms of action in the present trial remains unclear, the findings imply that omega-3 PUFAs may have stopped processes associated with the manifestation of psychotic disorders" (pp5-6).

Responding to the study, one UK psychiatrist's view was that "the study needs repeating in a larger group, but that it's reasonable for people to give fish oils a try. However, he warns that over-the-counter products may not have the right dose of the right fatty acids to be of use" (Staff Writer 2015 p17) (table 1.1).

STRENGTHS	WEAKNESSES
1. Double-blind - neither participants or administrators of trial knew who was receiving the treatment and placebo. 2. Participants randomly allocated to treatment or placebo, 3. Standardised inclusion and exclusion criteria. 4. Standardised measures of at-risk and psychotic disorders. 5. Validated psychometric measure of "conversion to psychotic disorders".	1. Small sample. 2. Treatment for relatively short period of time. 3. Sample from those seeking help at specialist clinic. 4. Two-thirds of sample were female. 5. No control over other factors - eg: two individuals, who did not develop psychosis, in the treatment group took fish oil capsules for longer than twelve weeks.

Table 1.1 - Strengths and weaknesses of Amminger et al (2015).

1.3. NEW ANTI-PSYCHOTIC DRUG

Brexpiprazole is a new second-generation anti-psychotic drug that acts on serotonin and dopamine in the brain.

Correll et al (2015) reported its efficacy in a six-week international trial at sixty-five study centres (including in the USA, Latvia, and Malaysia).

[9] Apoptosis is programmed cell death.

Participants were randomly assigned to one of three groups of brexpiprazole (0.25, 2 or 4 mg per day) or a placebo condition. The main outcome measure was a reduction in baseline score of the Positive and Negative Syndrome Scale (PANSS) (Kay et al 1999) [10]. In total, there were 623 participants, of which 410 completed the study.

The 2 mg and 4 mg brexpiprazole groups showed statistically significant improvements as compared to the placebo group at week 6 [11]. The reporting of negative side effects was made by half the participants in the treatment groups, but this was lower than the adverse effects, related to schizophrenia, reported by the placebo group (ie: more of this group discontinued than the treatment groups).

But this was a very short study, there was not a comparator group (ie: another anti-psychotic), and the participants were deliberately recruited with non-co-morbid schizophrenia (ie: no other psychiatric conditions as well).

Goff (2015) summed up: "This study provides a comprehensive picture of short-term efficacy and tolerability in relation to placebo, but in the absence of an active comparator it is difficult to predict whether brexpiprozale has meaningful advantages or disadvantages compared with existing agents" (p820).

1.4. APPENDIX 1A - EVERYDAY PSYCHOTIC EXPERIENCES

A small number of individuals who are not diagnosed with a mental disorder report psychotic experiences (PEs) (ie: hallucinations and delusions). One meta-analysis (Linscott and van Os 2013) calculated a median lifetime prevalence of 7.2%.

But there are weaknesses with such a pooled prevalence figure (McGrath et al 2015):

- The use of dichotomous measures (ie: present or absent).
- Variables, like frequency of occurrence or type of PE, are not collected.
- As with any meta-analysis, studies using diverse methods and designs are combined.

In other words, the subtlety of the PE is lost. McGrath et al (2015) reported an attempt to overcome this problem with data from the World Health Organisation

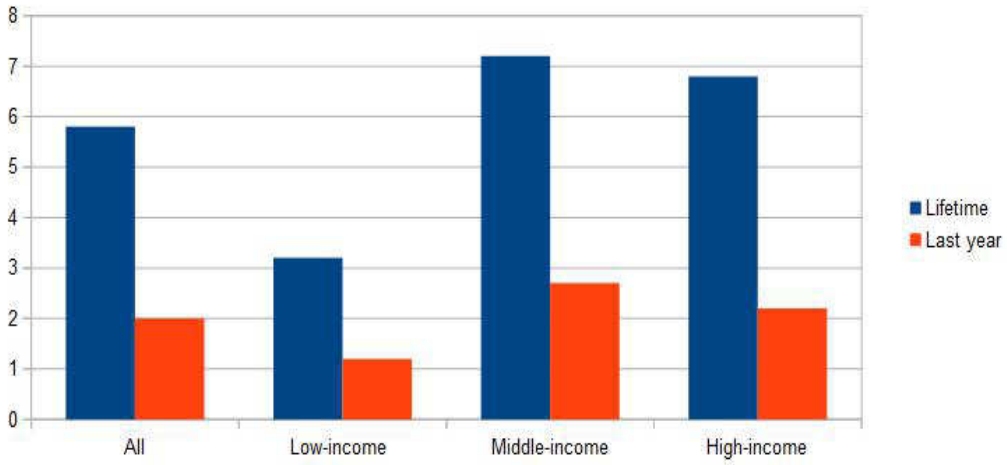
[10] Recovery is the ultimate aim of treatment, but there are issues with its measurement (appendix 1C).
[11] In a similar six-week trial (the BEACON trial) (Kane et al 2015), only the 4 mg dose group was significantly different from the placebo condition.

World Mental Health Surveys [12] in eighteen countries [13] between 2001 and 2009.

PEs reported by individuals who were or would be diagnosed with psychosis were excluded. This left data for 31 261 respondents.

Trained interviewers in face-to-face interviews asked about six types of PE - hallucinations (auditory; visual), bizarre delusions (thought insertion/withdrawal; mind control), and paranoid delusions (plot by others to harm individual; reference - eg: characters of television talking about individual). Participants were asked if they had had any such experiences, then, if so, how often in the last year and in their lifetime. The frequency was divided into five categories - once, 2-5 times, 6-10 times, 11-100 times, and more than 100 times.

Overall, lifetime prevalence of PE was 5.8%, and in the last year 2.0%. There was some variation between type of country (ie: low vs high income) (figure 1.1).



(Data from McGrath et al 2015 table 2 p701)

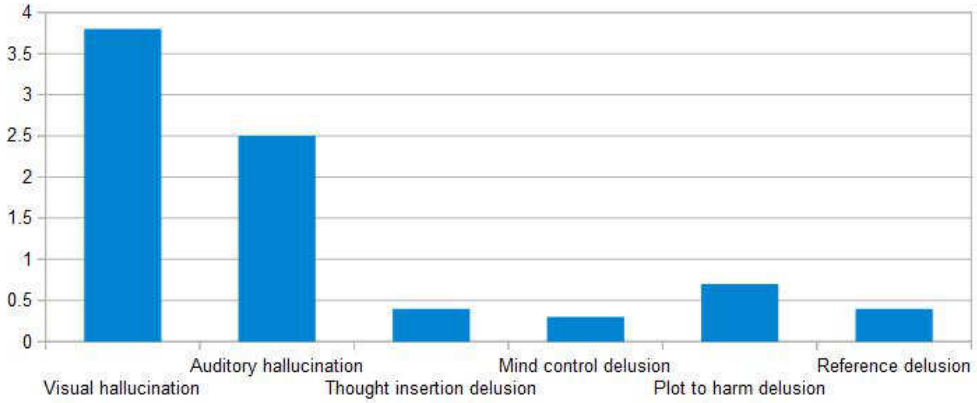
Figure 1.1 - Prevalence (%) of PEs by type of country.

The most common type of PE was visual hallucination (figure 1.2). Most participants had one PE only (figure 1.3).

Women had significantly more PEs than men, but this is mostly explained by more hallucinations (figure 1.4). PEs were significantly associated with being a homemaker (as opposed to being employed), being non-married (including widowed and divorced) (vs married), and having lower income.

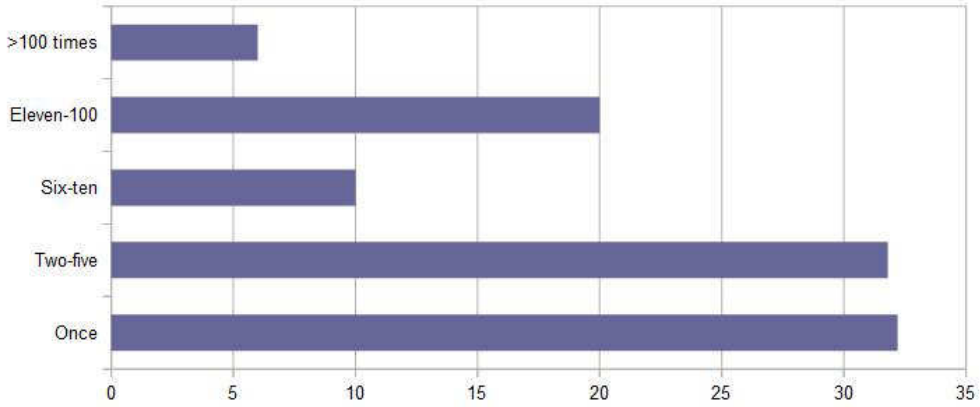
[12] Details at http://www.hcp.med.harvard.edu/wmh/.

[13] Eg: Colombia, Iraq (low income countries), Brazil, Lebanon (middle income), France and Germany (high income).



(Data from McGrath et al 2015 table 3 p702)

Figure 1.2 - Lifetime prevalence (%) of different types of PEs.



(Data from McGrath et al 2015 table 4 p703)

Figure 1.3 - Cumulative percentage of PEs by frequency.

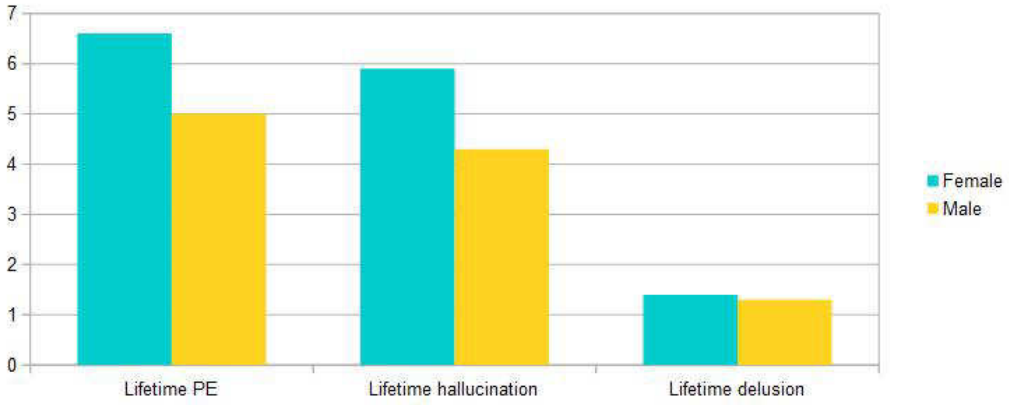


Figure 1.4 - Prevalence (%) by gender.

1.5. APPENDIX 1B - NEUROLOGY VERSUS PSYCHIATRY

A mental disorder that is associated with pathology of the central nervous system is classed as a neurological disorder (eg: Alzheimer's disease), and distinguished from a psychiatric disorder (eg: schizophrenia). But the "reality" of the psychiatry-neurology distinction is questioned (David and Nicholson 2015).

Modern neuroimaging techniques show changes in the central nervous system in schizophrenia, say. There is an issue as "both psychiatry and neurology are laying claim to the same organ, the brain" (David and Nicholson 2015).

"Going along with the idea one might then hypothesise that neurological patients show the bulk of abnormalities in the areas of the brain associated with more 'basic' functions: movement, sensation and, with respect to dementia, memory, whereas psychiatry patients show differences in 'higher' brain regions associated with self-consciousness and identity (the frontal lobes) or emotion (the 'limbic system')" (David and Nicholson 2015 p373).

A meta-analysis by Crossley et al (2015) of studies using voxel-based morphometry to assess changes in grey matter in the brain in different mental disorders reported "clear and statistically robust difference" between psychiatric and neurological disorders. The neurological side focused on the basal ganglia, and insula (expected), for example, as well as the dorsal prefrontal region in "their" disorders (unexpected), while the medial prefrontal area of the brain was involved in psychiatric disorders. "An unexpected and frankly odd finding was that regions of the visual association cortex were significantly more allied with psychiatric disorders. These included the lingual gyrus, which may after all be genuinely important given its role in face perception. The temporal cortex was, perhaps predictably, the truly contested area – containing both the hippocampus (reflecting the arbitrariness of regarding dementia as neurological or psychiatric) and components of the limbic system, as well as being a key area of epilepsy pathology, such as mesial temporal sclerosis" (David and Nicholson 2015 p374).

Goodkind et al's (2015) meta-analysis of VBM studies found that reduced grey matter in two areas of the brain (anterior cingulate and insula cortices) was common to neurological and psychiatric disorders.

David and Nicholson (2015) concluded: "The results may be taken as a slap in the face to the distinction-abolitionists, yet such individuals might take heart in them in that there is no implicit hierarchy in what emerges as the brain-based hallmark of neurological

versus psychiatric conditions; they both involve the functionally interesting parts of the brain, it is just that they are, quite subtly, different" (p374).

1.6. APPENDIX 1C - MEASURING RECOVERY

Personal recovery is the ultimate goal of any treatment. It is defined as "a deeply personal, unique process of changing one's attitudes, values, feelings, goals, skills, and/or roles. It is a way of living a satisfying, hopeful, and contributing life even with limitations caused by illness" (Anthony 1993 quoted in Williams et al 2015 p551).

How to operationalise a definition like this? One answer is with the Questionnaire about the Process of Recovery (QPR) (Nell et al 2009). There is an original 22-item version, and a fifteen-item version. The items, which are scored from 0 (disagree strongly) to 4 (agree strongly) are divided into two sub-scales (interpersonal and intrapersonal) (table 1.2).

Intrapersonal:
- Feel better about myself
- Feel part of society
- Take control of aspects of my life

Interpersonal:
- Weigh up pros and cons of treatment
- Meeting people with similar experiences

(Source: Williams et al 2015 table 3 p553)

Table 1.2 - Examples of items from QPR.

Williams et al (2015) reported the psychometric evaluation of both versions of the QPR using mental health service users in England (south London and Gloucestershire).

1. Test-retest reliability - The consistency of the questionnaire is assessed by asking the same individuals to complete the questionnaire at two different points in time, and correlate these scores (figure 1.5). A positive correlation is expected, and the closer to one the more reliable. The participants in this study completed the QPR with a two-week interval. The correlation for total score was +0.74, which is "fair to good" (Williams et al 2015).

2. Internal reliability - The score on each item is correlated with every other item to establish the internal consistency of the questionnaire. Many items are

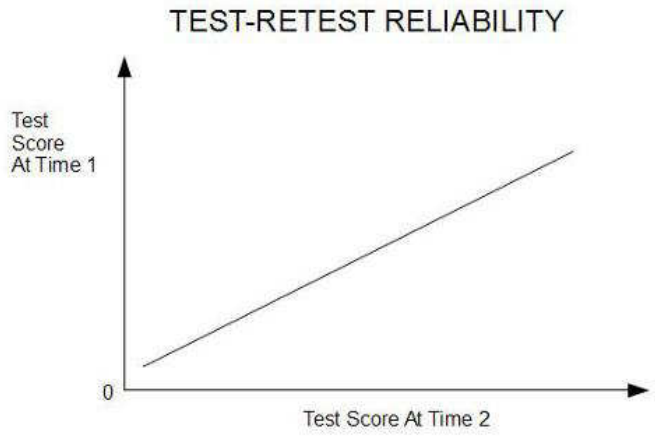


Figure 1.5 - Positive correlation and good test-retest reliability.

asking about the same behaviour, but in different ways, and so it is expected that individuals should score the same on similar items. A statistical test called Cronbach's alpha is used. The internal consistency for the interpersonal sub-scale was poor, but excellent for the intrapersonal sub-scale and the total score.

3. Concurrent validity - Validity is whether the questionnaire measures what it claims to measure. There are different ways of establishing this, but the main way is with other questionnaires of the same (or similar) behaviour (figure 1.6). The score on the QPR was correlated with the score on the Recovery Assessment Scale (RAS) (Corrigan et al 1999). The latter is a 41-item measure of five areas of recovery. It is expected that there will be a positive correlation between the two questionnaires. For total scores it was +0.73.

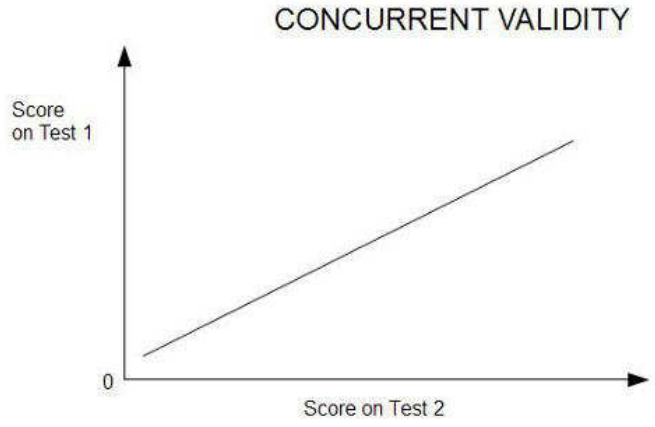


Figure 1.6 - Positive correlation and good concurrent validity.

4. Sensitivity to change - This was assessed by associating the QPR with another questionnaire (the Warwick-Edinburgh Mental Well-being Scale; WEMWBS) (Tennant et al 2007) at baseline and at three months (using regression analysis).

5. Factor structure - Confirmatory factor analysis was used to identify the two sub-scales (ie: underlying factors of recovery).

Williams et al (2015) concluded: "Both QPR intrapersonal and QPR total demonstrated adequate psychometric properties, whereas QPR interpersonal did not demonstrate psychometric adequacy" (p553).

1.6.1. Health

Any understanding of recovery requires a clear picture of what health is.

For example, the International Classification of Functioning, Disability and Health (ICF) (WHO 2001) was developed with a conceptual model that "disability is a multi-dimensional and universal phenomena placed on a continuum with health. Human functioning is understood as a continuum of health states, and every human being exhibits one or another degree of functioning in each domain, at the body, person, and society levels" (Alvarez 2012 pS70).

It is a biopsychosocial (BPS) model. This means that "'all the three levels, biologic, psychologic, and social processes, must be taken into account in every healthcare task' (Engel 1978) because they are all integral and interactively involved in physical health and illness... Therefore, the BPS model sees illness as resulting from the mechanisms interacting at the level of the organism and on the interpersonal and environmental levels. Accordingly, the study of every disease must include the individual, the body, and the surrounding environment as essential components of the entire system" (Alvarez et al 2012 pS174).

This model was developed to counter the dominance of the model, which holds that "anything that could not be objectively verified and explained was ignored or devalued" (Alvarez et al 2012 pS174).

Another alternative to the medical model comes from Venkatapuram (2011), who proposed a capability approach to health, whereby human health is seen as the capability to be healthy (CH) - ie: "to achieve, exercise or express a cluster of basic and inter-related capabilities and functionings" (Venkatapuram 2011 quoted in Buyx et al

2016) [14].

This fits with Nussbaum's (2011) ten central human capabilities:

- Life
- Bodily health
- Bodily integrity
- Senses, imagination, and thought
- Emotions
- Practical reason
- Affiliation
- Other species
- Play
- Control over one's environment (Richardson 2016).

"But having these central human capabilities depends upon lots of elements being present in the background that are not within an individual's power to put in place: educational systems, a job market, legal protections of religious freedom... and of freedom of assembly and free speech... This implies that, in many countries of the world, no one is (fully) healthy, because many countries lack legal protection of religious freedom and freedom of speech and assembly" (Richardson 2016 p4).

Broome (2002) pointed out the inseparability of factors - "asthma is less bad if you are well housed, mental handicap less bad in supportive communities, deafness less bad if you have access to the internet. Conversely, features of a person's health affect the value of other things: radios are no good to the deaf, nor running shoes to the lame. The interaction between health and other features of a person's life is so intimate that health cannot be treated as separable... "Pain is bad in itself, and it also reduces your ability to enjoy music. Is the latter an instrumental effect? It depends whether we count your ability to enjoy music as a health or non-health factor in your wellbeing. I am not sure where to draw the boundaries of health" (quoted in Richardson 2016).

Richardson (2016) criticised Venkatapuram (2011) as "not sufficiently distinguishing health from other important pre-requisites of well-being". While Tengland

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[14] Venkatapuram (2011) added a moral element - "every human being has a moral entitlement to the social bases of a sufficient and equitable CH because of its intrinsic value in constituting human dignity as well as its instrumental value for conceiving, pursuing and revising one's own life plans within contemporary global society" (quoted in Buyx et al 2016).

In any discussion of science and health, there is a "normative that lurks within the technical" (Jasanoff 2003 quoted in Caduff 2010).

(2016) criticised the definition for not including subjective well-being.

Definitions of health vary from narrow (eg: absence of disease) to broad (eg: overall well-being), while Venkatapuram (2011) takes a middle position with a holistic theory. Such theories "take the whole, acting and experiencing, individual as their starting point, and that, in general, they also define health in relation to a context, that is, a social and physical environment. The holistic theories, furthermore, differ from the broader well-being theories in that they narrow down the kinds of 'wellbeing' that belong to, or constitute, health" (Tengland 2016 p9).

Venkatapuram (2016) countered that "it is not the availability of commodities in the locality, or the person's happiness that is a good reflection of their health but her abilities to protect, maintain, and promote her beings and doings. Such an analysis also makes clear how the availability of healthcare is an important component, but cannot be the totality of the concern regarding health; we need to focus on the abilities of each individual in light of her internal endowments and skills and external environment" (p52).

1.7. APPENDIX 1D - INFLAMMATION

There is interest in the association between the immune system and the development of schizophrenia. For example, Benros et al (2011) (appendix 1E) found that a previous history of auto-immune disease [15] increased the incidence of schizophrenia by 36% compared to the general population, and prior hospitalisation for infection by 60% in a Danish study. Together the incidence was increased 125%.

It is possible that the release of pro-inflammatory cytokines by the immune system particularly during brain development, produce changes in the brain (Chaves et al 2015). This is the neuro-inflammation theory of schizophrenia.

Individuals with first episode psychosis or acute relapse have been found to have higher levels of pro-inflammatory cytokines, and individuals in remission lower levels (Miller et al 2011).

[15] Auto-immune diseases are where the immune system produces anti-bodies, normally used to fight "invaders" like infections, that "attack" the body's own tissue. These auto-anti-bodies can cross the blood-brain barrier and "attack" brain tissue.

1.7.1. Treatments

More generally, inflammation may be involved in a variety of psychiatric disorders. For example, the administration of inflammatory stimuli produces psychiatric symptoms (Miller and Raison 2015). So, does that mean that anti-inflammatory treatments will reduce psychiatric symptoms?

Miller and Raison (2015) offered a number of thoughts in answering this question:

i) "What has become increasingly clear is that no psychiatric disorder is an inflammatory disorder, and only sub-groups of patients with any given psychiatric disease exhibit an increased number of inflammatory markers. Thus, treatment trials of anti-inflammatory agents should pre-select patients with increased inflammation... [and] the indiscriminant use of anti-inflammatory treatments for psychiatric patients without inflammation... may reduce the likelihood of detecting a response and could potentially lead to the exacerbation of the disease" (Miller and Raison 2015 p527).

ii) The need for effective markers (ie: ways to measure response to anti-inflammatory treatment). For example, C-reactive protein is commonly used, but what is needed is "more nuanced profiles of inflammatory proteins and gene expression, as well as cellular immune parameters" (p527).

iii) The need to focus on inflammation in specific areas of the brain (eg: basal ganglia).

iv) "Not all anti-inflammatory drugs are created equal. A significant drawback to current studies examining anti-inflammatory strategies in psychiatric disorders is that the 'anti-inflammatory' drugs commonly used have a multiplicity of 'off-target' effects. Such drugs may inhibit inflammation and improve symptoms, but they might do so through mechanisms unrelated to their anti-inflammatory activity, thereby confounding the interpretation of results" (Miller and Raison 2015 p528).

1.7.2. Depression

A link between major depression in certain cases and inflammation has been made from two sets of evidence - (i) high levels of cytokines among depression sufferers, and (ii) pro-inflammatory medications produce depressive symptoms (Kohler et al 2014).

Anti-inflammatory treatments that have been tried with depression include non-steroidal anti-inflammatory

drugs (NSAIDs) and cytokine inhibitors.

For a meta-analysis, Kohler et al (2014) found fourteen randomised controlled trials published before 2014 on the effect of anti-inflammatory treatment on depressive symptoms. Ten trials investigated NSAIDs, six as compared to a placebo or treatment as usual (monotherapy), and four as an add-on treatment (ie: anti-inflammatory drug and anti-depressant vs placebo and anti-depressant). The other trials studied cytokine inhibitors compared to a placebo. No studies were found that met the inclusion criteria for other anti-inflammatory agents, like aspirin or statins.

Five trials had participants diagnosed with depression, and the others with depressive symptoms. Most trials lasted 6-12 weeks.

A standardised mean difference (SMD) was calculated for each study. This is the mean difference in the depression score between the treatment and control groups divided by the pooled standard deviation of the distribution of the scores in the study. This gives an effect-size, and by convention, 0.2 is a small effect, 0.4 is medium, and 0.8 a large effect. The pooled effect (ie: combined SMDs) was -0.34 [16]. This means that the treatment effect was greater than the placebo in reducing depression and depressive symptoms.

Kohler et al (2014) warned: "The potential anti-depressant treatment effects of anti-inflammatory strategies should always be balanced against the risk for adverse effects. Non-steroidal anti-inflammatory drugs increase the risk for gastro-intestinal and cardiovascular adverse effects, whereas cytokine inhibitors increase the risk for infections. We observed no increased risks of these important adverse effects; however, not all of the studies included in the present meta-analysis reported on adverse effects and treatment lasted only 6 to 12 weeks..., which potentially is too short to detect relevant adverse effects" (p1389).

1.8. APPENDIX 1E - BENROS ET AL (2011)

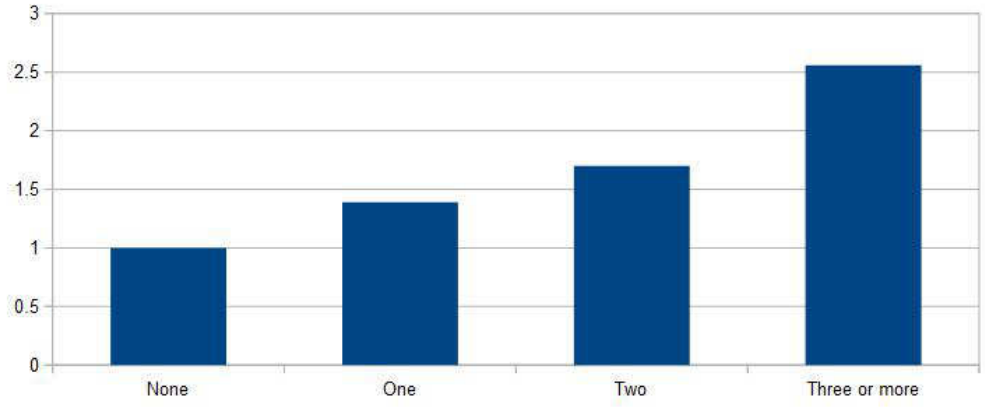
Benros et al (2011) used data from the Danish Psychiatric Central Register (DPCR), which contains all admissions to psychiatric hospitals and psychiatric out-patients since 1995, and the Danish National Hospital Registry (DNHR) (started in 1977). Individuals in Denmark are assigned a unique identifier at birth, and so it is possible to find them on these registers. Over 3.5 million people were checked for the period 1977 to 2006.

[16] The overall effect-size for depression was -0.54, but -0.27 for depressive symptoms.

For the study period, 39 076 individuals were on the DPCR for a schizophrenia spectrum disorder (ie: schizophrenia or schizophrenia-like psychoses), of which 23.6% had a prior infection and 2.4% an auto-immune disease (as stated in the DNHR). Thus, an incidence rate ratio (IRR) [17] for schizophrenia of 1.60 and 1.45 respectively, but 2.25 for both a prior infection and an auto-immune disease (where 1.00 is the normal risk in the general population).

The risk of schizophrenia was increased by:

- Number of prior hospital admissions for infection or auto-immune disease (figure 1.7).
- The type of infection or auto-immune disease - eg: an IRR of 4.89 for prior hepatitis, but 8.89 for auto-immune hepatitis.
- The recency of infection or auto-immune disease - eg: an IRR of 2.35 and 2.91 respectively for within the last year.



(Data from Benros et al 2011 table 2 p1306)

Figure 1.7 - Incidence rate ratios for schizophrenia based on number of prior infections.

The risk of schizophrenia was still significant after controlling for substance use disorders, and a family history of psychiatric disorders.

The researchers "only examined the time up to the schizophrenia diagnosis, and since many autoimmune diseases have a clinical onset later than that for schizophrenia, the autoimmune disease could have already been present, resulting in an underestimation of the

[17] An estimate of relative risk.

effect as a result of undiagnosed illness. Individuals not yet diagnosed with schizophrenia may have had unspecific psychiatric symptoms and possible initial misclassification that could have affected the results" (Benros et al 2011 p1309). Also social class and other environmental factors were not included. Ultimately, the data only covered cases where help was sought.

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2. POST-TRAUMATIC STRESS DISORDER AND POST-TRAUMA STRESSORS

2.1. CHILDREN

Children exposed to war, of which there are millions today around the world according to UNICEF, are vulnerable to mental health problems like post-traumatic stress disorder (PTSD), depression, and anxiety (internalising behaviours) [18], and behavioural problems (externalising behaviours). But the association between war exposure and mental health problems is mediated by post-conflict daily stressors (Newnham et al 2015).

Newnham et al (2015) showed this in a study of children and adolescents in Sierra Leone. The 529 individuals were recruited by the International Rescue Committee in 2002 when aged 10-17 years old, and they were interviewed subsequently twice more in 2004 and 2008 [19]. The sample included former child soldiers who did or did not receive post-conflict reintegration services, and civilian victims.

The third set of interviews in 2008 involved 363 of the original sample, and local researchers used a variety of measures translated in the appropriate languages (table 2.1).

Overall, six years after the war, exposure to war was significantly associated with PTSD (but not depression), and this relationship was mediated by post-war daily stressors (figure 2.1) [20]. So, "despite exposure to severe and chronic violence during childhood, economic and interpersonal stressors experienced in the post-conflict setting appear to play a substantial role in young people's psychological impairment" (Newnham et al 2015 p118).

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[18] These conditions are evident in situations of tyranny (appendix 2A).

[19] The longitudinal study of the sample has been previously reported in, for example, Betancourt et al (2010, 2011, 2013a & b; appendix 2B).

[20] The average was eight daily stressors with the most common being - experienced death of someone close, and lacking money for basic necessities.

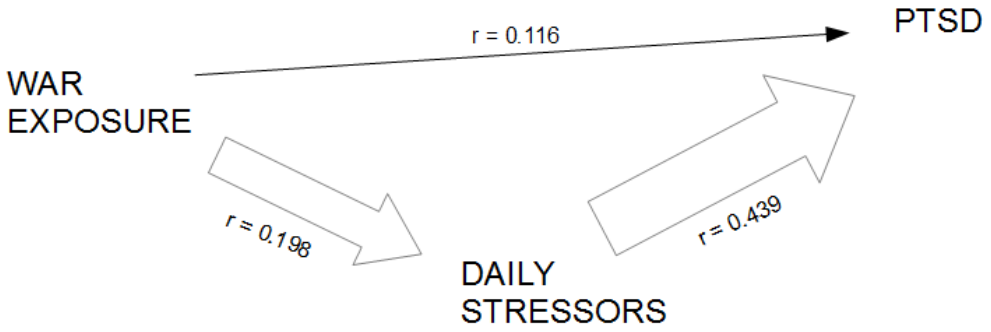
- Childhood War Trauma Questionnaire (Macksoud and Aber 1996) - 55 items, rated yes or no, about violence witnessed, experienced or perpetrated, and bereavement due to war.

- Post-War Adversities Index (Layne et al 2010) - 30 items about post-conflict current stressors (eg: "during the past six months, have people you live with had serious conflicts with each other (arguing, shouting, screaming)?").

- UCLA Post-Traumatic Stress Disorder Reaction Index (PTSD-RI) (Pynoos et al 1998) - The frequency of twelve symptoms in the last month, scored as "never" (0), "sometimes" (1), or "often" (2).

- Hoskins Symptom Checklist (HSCL-25) (Lavik et al 1999) - 25 items about depression and anxiety rated from 0 to 3 ("not at all" to "extremely").

Table 2.1 - Main questionnaires used by Newnham et al (2015) [21].



(Thickness of line = strength of relationship)

(Based on Newnham et al 2015 figure 1 p118)

Figure 2.1 - Relationship between war exposure and PTSD.

This finding supports other studies in post-conflict regions. For example, among Darfuri adult refugees in temporary camps in Chad, psychological distress was related to current concerns about safety and basic needs rather than war trauma (Rasmussen et al 2010). Also in non-war situations. Fernando et al (2010), for instance, found that student victims of the 2004 tsunami in Sri Lanka were more affected by current material deprivation, say, that the disaster.

[21] Fontana and Frey (2000) observed: "Asking questions and getting answers is a much harder task that it may seem at first. The spoken or written word has always a residue of ambiguity, no matter how carefully we word the questions and how carefully we report or code the answers" (p645).

2.2. LONG-TERM EFFECTS

Very long-term studies of PTSD are rare, partly because PTSD was only included as a diagnostic category in 1980 (in DSM-III).

The National Vietnam Veterans Readjustment Study (NVVRS) was begun in the mid-1980s in the USA. At about a decade after the Vietnam War ended, 15.2% of men and 8.5% of women were diagnosed as having current PTSD, and about 30% of all the sample lifetime PTSD (Kulka et al 1990). The National Vietnam Veterans Longitudinal Study (NVVLS) followed-up this study.

Marmar et al (2015) reported work with around 2000 individuals on the NVVLS in 2013 (ie: 40 years after the Vietnam War). Participants completed questionnaires and telephone interviews, and a sub-sample had a clinical interview. Individuals were classed as theatre-veterans (who had seen combat) or era-veterans (who did not experience combat).

PTSD was assessed in a number of ways:

- Mississippi Scale for Combat-Related Post-Traumatic Stress Disorder (M-PTSD) (Keane et al 1988) - This has 35 items covering re-experiencing and avoidance, withdrawal and numbing, arousal and emotion control, and self-persecution or survivor guilt.

- PTSD Checklist for DSM-5 (PCL-5) (Weathers et al 2013) - This covers the twenty symptoms of PTSD in DSM-5.

- Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) (Blake et al 1995).

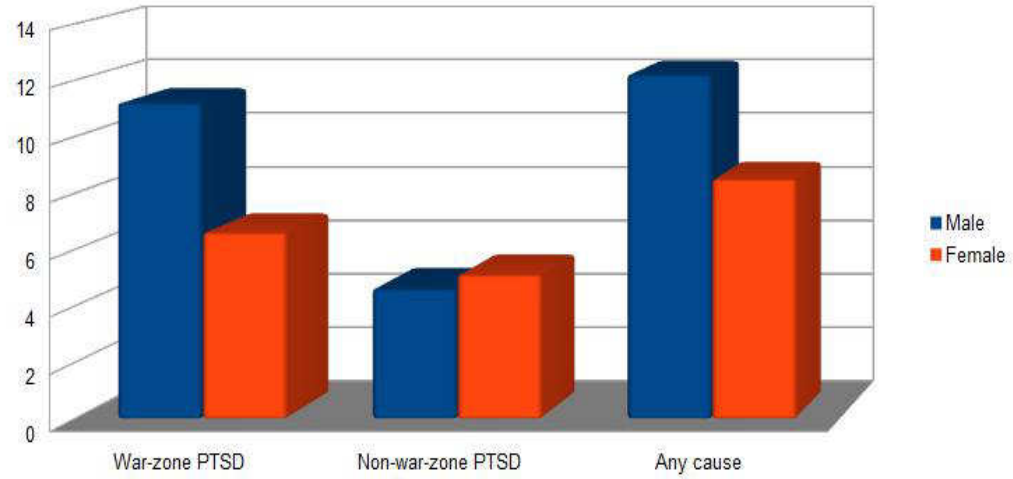
- Structured Clinical Interview for DSM-IV, Non-Patient Version (SCID-NP) (First et al 2002).

Based on CAPS-5, 4.5% of male theatre-veterans were classed as having current war-zone PTSD, with a 17% lifetime prevalence, and among women the figures were 6.1% and 15.2% respectively. From PCL-5 the current prevalence was 11.2% for men and 6.6% for women (figure 2.2).

Mean M-PTSD scores of over nine hundred theatre-veterans who completed the NVVRS and NVVLS showed a modest increase over time [22], while era-veterans' (n = 409) scores were low and stable.

From the SCID-NP, it was found that one-third of theatre-veterans with PTSD had a co-morbidity of major depressive disorder.

[22] 16% had an increase of more than 20 points, and 7.6% a decrease of the same amount.



(Data from Marmar et al 2015 table 2 p879)

Figure 2.2 - Prevalence rates for PTSD using PCL-5.

Marmar et al (2015) summed up: "An important minority of Vietnam veterans are symptomatic after four decades, with more than twice as many deteriorating as improving" (p880).

A key strength of the study was multiple measures of PTSD, both self-reports and clinician-based, but the "lengthy interval data collection points precludes a refined analysis of remitting and relapsing symptom patterns" (Marmar et al 2015 p880).

How do the findings of Marmar et al (2015) compare to other long-term studies?

1. US Vietnam veterans - Koenen et al (2003) found a decline in PTSD prevalence up to fourteen years after the war, while Schurr et al (2005) reported delayed-onset PTSD.

2. Other wars - eg: an increase in PTSD prevalence up to twenty years after the 1973 Yom Kippur War is Israel (Solomon 1993), and an intensification of stress symptoms among World War II veterans at twenty years (Archibald and Tuddenham 1965).

2.3. NATURAL DISASTERS

Hurricane Katrina made landfall on 29th August 2005

in the New Orleans area [23]. As well as the obvious physical effects, there were lasting psychological ones. Research on survivors of such natural disasters have produced a number of findings about PTSD.

1. Mental health problems worsen over time - eg: the prevalence of PTSD was 15% among 815 survivors 5-8 months after Katrina, but at 21% one year after (Kessler et al 2008).

2. Social support aids resilience to psychological trauma - both pre- and post-disaster (Chan et al 2015).

3. "Post-traumatic growth" vs PTSD - Rhodes et al (2010) used data collected in 2003 in New Orleans on 1019 low-income parents as a baseline to compare 392 of them one year after Katrina (known as the Resilience in Survivors of Katrina (RISK) project). About half the participants had symptoms of PTSD, and serious mental illness had doubled to 14% since baseline.

But one-third of the sample reported "post-traumatic growth" ("the feeling that surviving the disaster made them stronger, even if they simultaneously experienced from mental illness"; Reardon 2015).

Three years after Katrina, two-thirds of low-income women showed no signs of psychological distress (Lowe and Rhodes 2013; appendix 2C).

2.4. INTERNATIONAL COMPARISONS

When investigating lifetime PTSD, individuals are often asked to nominate their worst trauma and PTSD is assessed in relation to that. "Because worst traumas are not the most commonly experienced events in the population and, presumably, have a higher risk of PTSD than typical traumas, this approach over-estimates conditional risk of PTSD" (Atwoli et al 2015 p307).

The World Mental Health (WMH) surveys compare PTSD from a randomly chosen lifetime trauma to the nominated worst trauma, and produce a weighted average (Atwoli et al 2015). Thus, the lifetime prevalence of PTSD are calculated in the WMH surveys as 2.3% in South Africa (Atwoli et al 2013), 2.2% in Spain (Olaya et al 2015), and 1.3% in Japan (Kawakami et al 2014), for example. These figures are lower that studies using just the worst event (Atwoli et al 2015).

Breslau et al (2004) found a lifetime prevalence of

[23] Nearly 2000 people died and over 650 000 individuals were displaced, with low-income and African-American communities suffering disproportionately more, particularly due to poor quality housing and the areas where they lived. Such groups also experienced more post-event stressors, like living in shelters, and unemployment (Lowe and Rhodes 2013).

PTSD of 13.6% with the worst event method compared to 9.2% using the WMH survey method. Beals et al (2013) found that asking two Native American samples about their single worst trauma produce PTSD of 6-15%, but rates of 9-20% when asking about their three worst traumas.

The conditional risk of PTSD is the prevalence among those exposed to trauma as opposed to prevalence across the whole population. This was 3.3% in South Africa and 3.3% in Spain, for instance (Atwoli et al 2015) [24].

Another problem with international comparisons of PTSD rates is that there are different ways of classifying traumatic events. The WMH surveys distinguished twenty-nine different types, which were categorised into eight groups - war events, physical violence, sexual violence, accidents, unexpected death of a loved one, social network events, witnessing trauma, and other/private (Atwoli et al 2015).

Lifetime exposure to a traumatic event varies between 74% in South Africa and 54% in Spain, while the type of trauma is also different. In South Africa, physical violence and witnessing trauma are most common, but accidents and unexpected death are most reported in Europe. These categories of trauma were linked to the conditional risk of PTSD in those countries (Atwoli et al 2015).

Socio-demographic factors linked to trauma exposure vary between countries. In South Africa, being employed is a significant risk (as compared to homemaker), while being male is common in many countries, for instance. But being female is a greater risk for PTSD (Atwoli et al 2015).

2.5. MEDICALISATION

Behrouzan (2015) stated: "Most debates on post-war mental health focus on clinical evaluations of veterans' and civilians' individual experiences of wartime 'trauma'. But the psychological afterlife and the social discord that wars create cannot be reduced to a clinical artifact of individual trauma or be divorced from the historical and cultural meanings that it carries" (p40).

She focused on the "medicalisation" [25] of memories of

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[24] Behrouzan (2015) urged caution: "epidemiological data are useful, but they need to be interpreted within proper conceptual frameworks to ask what they reveal and what they mask. What is necessary is a critical examination of the cultural meanings that shape individuals' experiences of diagnostic categories and the standards and models on which they rely" (p41).

[25] "Medicalisation" describes how "previously non-medical phenomena are brought under the purview of biomedicine and often turned into disorders" (Behrouzan 2015 p41). Clarke et al (2010) used the term "biomedicalisation" to refer to "the increasingly science-based expansion of medicine into more

the Iran-Iraq War for Iranians. Quoting "Sara", who had been a child at the time, about receiving a diagnosis of PTSD and depression in adulthood: "On the one hand, I wasn't sure how I felt about the stigma (appendix 2D) because it meant I was not a normal person, but on the other hand, I was relieved. I knew I wasn't crazy or weird. It made sense. After all, I grew up in the shadow of a bloody war" (p46). Behrouzan (2015) called "these modes of self-creation 'psychiatric subjectivities', and described them as a performative embodiment of the biomedical discourse as part of one's articulation and interpretation of lived life" (p46).

Behrouzan (2015) summed up: "For Sara, the war is an integral element of her identity, as well as that of her generation. On the one hand, drawing on the psychiatric language that became normalised in society since the 1990s, she clinically diagnoses and medicalises life experiences that are socially and historically structured" (pp46-47).

2.6. APPENDIX 2A - TYRANNY

The mental health of the individual is affected by their environment, particularly where it is aversive as in a totalitarian state. Here tyranny [26] [27] is used to govern the people - ie: physical and psychological coercion.

Abed (2004) outlined four ways in which the totalitarian state damages mental health:

i) Directly - eg: torture.
ii) Indirectly - eg: disempowerment, normalising of violence.
iii) No resources for mental health services.

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and more aspects of life, including the absence of disease" (p405). This includes the emphasis on risk factors for future illnesses, which has created individuals without current disease as in a "state of temporary undisease" rather than being healthy.

This idea also fits with the future perceived as "owned" because of its predictability. But when the future is not as expected, there can be a feeling of betrayal or of being cheated.

[26] Arendt (1968) defined tyranny as "arbitrary power, unrestricted by law, wielded in the interest of the ruler and hostile to the interests of the governed" (quoted in Abed 2004).

[27] Abed (2004) distinguished three forms of tyranny:
i) Authoritarian - the state maintains the social order, but does not change civil society (eg: military rule in Argentina and Chile in 1970s and 1980s).
ii) Sultanistic - society is subverted to the will of the leader, though parts of civil society unaffected (eg: Iran under the Shah, pre-1979).
iii) Totalitarian - "Society is re-engineered under the guiding principles of the state ideology (communism, nationalism, fundamentalism etc) in a process that frequently leads to intense suffering, mass murder and a variety of mass human rights violations. The totalitarian regime relies on the loneliness of the individual; a reality that it actively promotes through its policy of atomisation of society into isolated (ineffectual) individuals, its imposition of a state ideology and its systematic use of terror" (Abed 2004 pp3-4).

iv) The effect on the perpetrators of the state violence.

In one survey in Iraq under Saddam Hussein and the Ba'th Party, about half the households questioned reported torture, killing, beating, and disappearance among other abuses (Amowitz et al 2004). The most common psychiatric conditions related to living in such a situation are post-traumatic stress, depression, anxiety, and paranoia (Abed 2004).

2.7. APPENDIX 2B - THERESA BETANCOURT

Betancourt (2015) distinguished two common views about the effect of exposure to violence in childhood:

i) "Violence begets violence" - children exposed to violence become violent adults and/or experience other problems. For example, war-affected youth show a "foreshortened sense of the future" (ie: concern with current gratification) (Betancourt 2015).

ii) "Resilience hypothesis" - coping is aided by the right type of support, and violence in adulthood is not inevitable.

Betancourt et al (eg: 2013a) found evidence of both possibilities among the war-affected youth in Sierra Leone six years after the civil war. Around one in ten of the sample had high levels of mental health symptoms which could lead to option (i) above, but the remainder, despite higher levels of symptoms than US samples, showed improvement fitting with option (ii) above.

Individuals exposed to violence have been found to show negative parental behaviours, which has consequences for their children who were not exposed to the violence (ie: intergenerational effect), in studies with refugees, war veterans, and Holocaust survivors (Betancourt 2015).

2.8. APPENDIX 2C - LOWE AND RHODES (2013)

Lowe and Rhodes (2013) were able to identify six distinct groups in terms of the longer-term psychological response to Katrina.

They used a group of 492 low-income [28] parents who attended three community colleges in New Orleans in 2004-5 for another study as a baseline (Time 1). Then twelve months after Katrina, 402 were found for a first follow-

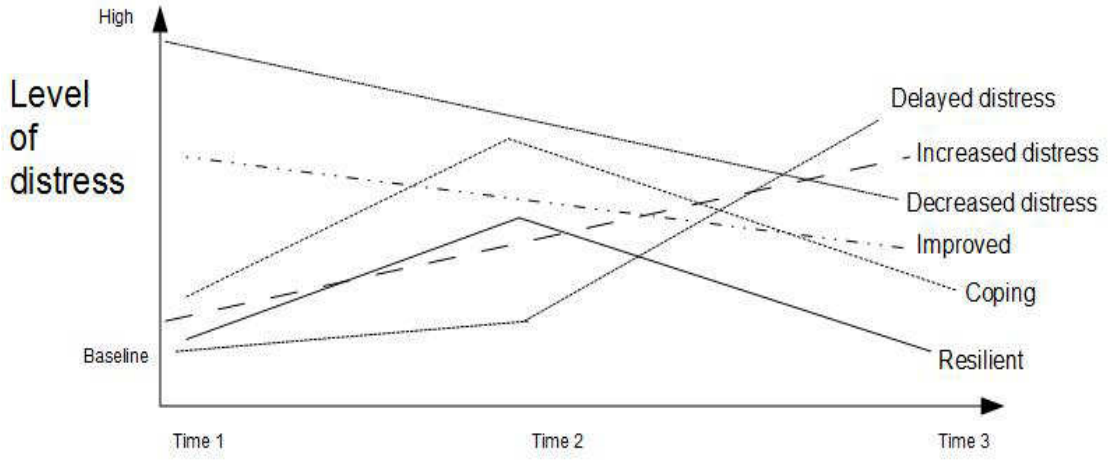
[28] Low income was defined as household income under 200% of US government-defined poverty level.

up (Time 2), and 386 (all female [29]; 85% African-American) were followed up three years after Katrina (Time 3).

A number of measures were taken including:

- General psychological distress - eg: "During the past thirty days, about how often did you feel so depressed that nothing could cheer you up?".
- Post-traumatic stress - eg: "Any reminders brought back feelings about it [traumatic event]".
- Hurricane-related stressors - eg: "lacked enough fresh water to drink".
- Social and material resources - eg: "There are people I know will help me if I really need it".

The scores and variables were grouped using a technique called latent class growth analysis (LCGA) [30]. The six trajectory groups were (figure 2.3):



(Based on Lowe and Rhodes 2013 figure 1 p404)

Figure 2.3 - Six trajectory groups and psychological distress over time.

1. Resilient (62.4% of participants; figure 2.4) - Increased mild or moderate distress after Katrina, but a decline to baseline levels at three-year follow-up.

[29] There were sixteen men who were removed from the analysis at this point.

[30] Nandi et al (2009), for example, used LCGA to distinguish five depression trajectories among New York residents in the 2-3 years after the 9/11 (September 11th 2001) terrorist attack - no symptoms, mild increase since event, severe increase since event, decrease, and chronic symptoms throughout.

2. Coping (22.2%) - Similar to "resilient", but greater distress and less of a decline.

3. Increased distress (3.9%) - Increase in mild/moderate distress after Katrina.

4. Delayed distress (4.5%) - Distress developed by one and three years post-Katrina.

5. Decreased distress (3.9%) - High distress at baseline that declined over time.

6. Improved (3.2%) - Mild/moderate distress at baseline that declined over time.

Allocation to the group was influenced by degree of exposure to hurricane-related stressors, experiences of bereavement (human and/or pet), perceived social support, and socio-economic status. The "resilient" group, for instance, experienced significantly less stressors and bereavement than the other groups, but had more social support than them. The "increased distress" group had most pet loss, and less social support than the "resilient" group, while the "decreased distress" group had lowest socio-economic status (eg: most welfare benefits).

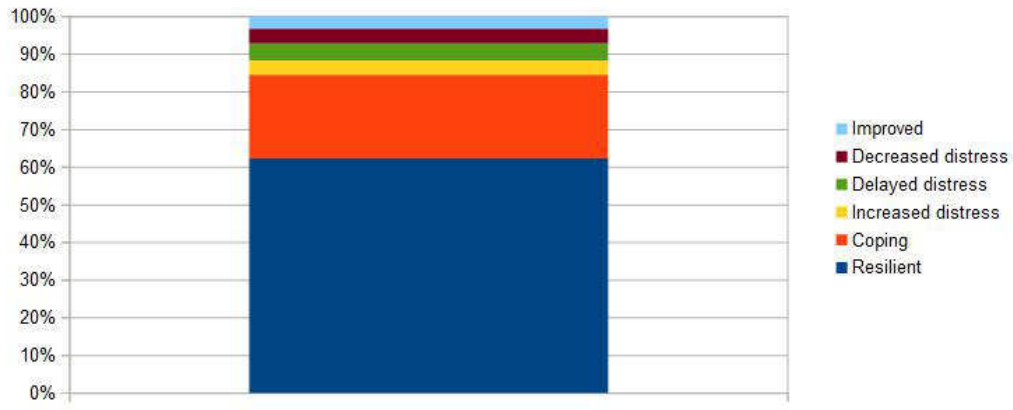


Figure 2.4 - Percentage of participants allocated to each trajectory group.

Lowe and Rhodes (2013) admitted that there were four key limitations to the generalisability of their study:

- Though the participants were low-income mothers, they were all enrolled in college at baseline.

- There were gaps between the follow-ups which meant that the researchers were not able to "understand the

complex patterns of change" (p409).

- For the group names, "although attempts were made to choose labels representative of the trajectory shapes, the names selected are not value-neutral" (p409).

- Self-reported measures of distress used.

2.9. APPENDIX 2D - STIGMA

The concept of stigma in the social sense is attributed to Goffman's (1963) work. Subsequently, researchers, particularly in the 21st century, have "begun to look more closely at the complexity of stigma; at comparisons across time, place, and substantive cases; and at novel uses" (Pescosolido and Martin 2015 p88). Beyond mental illness, stigma has been applied to a variety of phenomenon, from health issues to non-native accents or voluntary childlessness (Pescosolido and Martin 2015).

The idea of stigma is not without its critics - eg: "some persons with lived experience (ie: have an illness, an event, a status, or a relationship that predisposes them to disdain) find that 'stigma' does not convey the harshness of its impact, preferring 'discrimination'" (Pescosolido and Martin 2015 p88). While Manzo (2004) called it an "underdefined and overused concept".

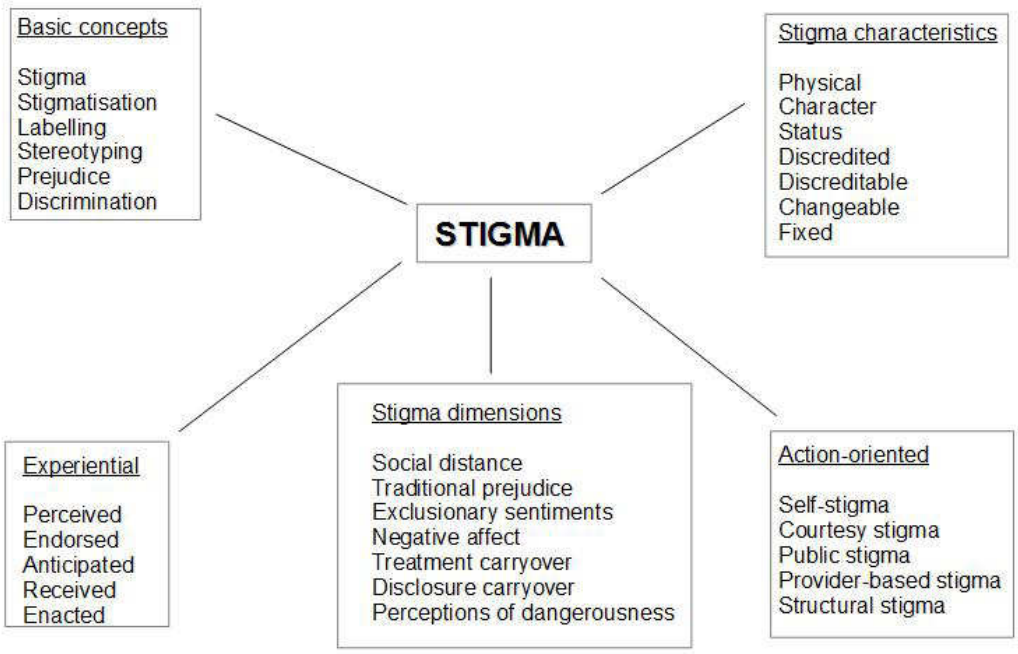
What that means is that stigma has become "a global referent category" (Pescosolido and Martin 2015). Simply, stigma is the "mark of shame" [31], but it is associated with stigmatisation (the social process related to stigma), and includes labelling, stereotypes, prejudice and discrimination, as well as the experience of stigma (eg: perceived, anticipated, enacted, or self-stigma) (Pescosolido and Martin 2015) [32].

Pescosolido and Martin (2015) summed up: "a difference is translated into a marked, devalued distinction. Through a culturally constituted process, the mark works to advantage or disadvantage others through social interactions with individuals, groups, organisations, and institutions. Stigma is conferred through labels, which are officially sanctioned terms

[31] The mark can be physical, character (eg: moral weakness), or status, for example (Pescosolido and Martin 2015).

[32] There is also "courtesy stigma" (or "stigma by association"; Ostman and Kjellin 2002). "Although individuals themselves do not have the mark, they live with, work with, or otherwise have a proximate social relationship with individuals or groups that do. Because of this social proximity, they too are subjected to devaluation through suspicion of having played a role in cause or the lack of ability to help. Both individuals who have strong ties to stigmatized groups or individuals and those that have occasional interactions are subject to this contamination" (Pescosolido and Martin 2015 p94).

encoded by official agents of social control (eg: 'mentally ill' by the medical profession, 'criminal' by the justice system) or by others informally (eg: 'fat'). These labels produce stereotypes (ie: negative beliefs and attitudes assigned to social entities thus labelled), with variable levels of negative social consequences of prejudice (endorsement of stereotypes) and discrimination (differential and disadvantaged treatment of the stigmatised). In other words, stigma represents the intersection of cultural differentiation, identity formation through social interaction, and social inequality" (p93) (figure 2.5).



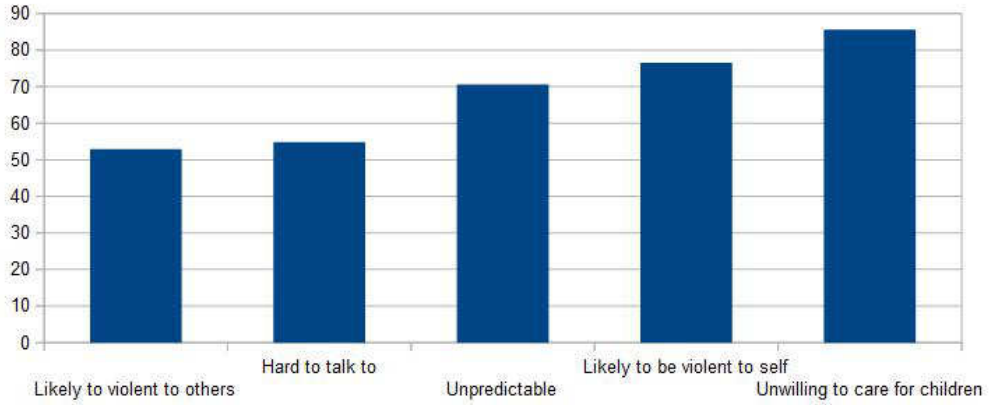
(Based on Pescosolido and Martin 2015 table 1 p92 and table 2 p97)

Figure 2.5 - Different concepts related to stigma, according to Pescosolido and Martin (2015).

Pescosolido and Martin (2015) preferred to talk about "the stigma complex", which they defined as "the set of inter-related, heterogeneous system structures, from the individual to the society, and processes, from the molecular to the geographic and historical, that constructs, labels, and translates difference into marks. In turn, reactions from the internal to individual to those by even remote association, to a cultural bundle of prejudice (ie: values, beliefs, attitudes, intentions) and discrimination (from other individuals, organisations, and institutions) are produced. This cultural bundle both shapes and is shaped by larger contexts that attempt to reduce them and subject them to

larger, often unacknowledged, feedback loops, as well as intended and unintended consequences" (Pescosolido and Martin 2015 p101).

Concentrating on schizophrenia, the Stigma in Global Context-Mental Health Strategy (SGC-MHS) (eg: Pescosolido et al 2008) explored stigma in sixteen countries. Though there were national differences in the endorsement of twenty-seven items after reading a vignette about an individual with schizophrenia, overall three-quarters of respondents agreed with "likely to be violent to self" and "unwilling to care for children" (figure 2.6).



(Data from Pescosolido and Martin 2015 figure 1 p100)

Figure 2.6 - Mean agreement in sixteen countries with selected items after reading vignette about an individual with schizophrenia.

Interventions have been designed to reduce mental health-related stigma. Mehta et al (2015) reported evidence for the effectiveness of such interventions in increasing knowledge and reducing stigmatising attitudes. The interventions included direct contact with service users, mental health education, internet courses, and video materials. The target of the interventions included students, the general public, and healthcare professionals.

Mehta et al (2015) found eighty studies between 1980 and 2013 in their literature review. Most of the studies had a follow-up of at least four weeks.

Meta-analysis of the data found a medium effect in increased knowledge [33], and a small reduction in stigmatising attitudes and behaviours [34].

[33] Standardised mean difference (SMD) of +0.54.
[34] SMD of -0.26 (attitudes).

A number of methodological issues were raised about the studies:

- Different outcome measures (136 in 80 studies).
- Validity of measures of stigma (55 different scales used).
- Sample size (59 - 753).
- Length of follow-up (1 - 18 months).
- Control group or not.
- Randomisation of participants or not.
- Sample recruitment and selection bias.
- Drop-out/attrition rate.

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3. DRUG TREATMENT

3.1. POST-TRAUMATIC STRESS DISORDER

PTSD can affect between 5-15% of individuals who have experienced a traumatic events (Hoskins et al 2015). There are no specific drugs for treating it, but other types like anti-depressants are used.

Do such drugs work for PTSD? The medical opinion is divided. The National Institute for Health and Clinical Excellence (NICE) in the UK, for example, found that certain anti-depressants are better than placebos (National Collaborating Centre for Mental Health 2005), while the Institute of Medicine (2008) in the USA had inadequate evidence to agree (Hoskins et al 2015).

The World Health Organisation (WHO) felt the need to deal with the inconsistent findings of previous reviews. Thus Hoskins et al (2015) reviewed the randomised controlled trials (RCTs) (appendix 3A) of pharmacological treatments (compared with placebo) to reduce traumatic stress symptoms in PTSD sufferers. They included fifty-one studies (eg: adults; double-blind; PTSD diagnosed with ICD or DSM criteria), of which fifteen were new RCTs to previous reviews of the evidence [35].

Selective serotonin reuptake inhibitor (SSRI) anti-depressants [36] generally, and particularly fluoxetine, paroxetine and venlafaxine, were found to be statistically superior to placebo, but with a small effect (ie: less than psychological treatment over waiting list or usual treatment control group; Bisson et al 2007). Also there was not enough evidence for other types of anti-depressants.

3.2. APPENDIX 3A - RANDOMISED CONTROLLED TRIALS

RCTs [37] are a key part of evidence-based medicine

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[35] National Collaborating Centre for Mental Health (2005), ACPMH (2007), and Stein et al (2004).

[36] SSRIs are used to treat social anxiety, for instance. This affects one in twenty individuals, and involves the avoidance of situations that include scrutiny or perceived risk of embarrassment (Stein and Andrews 2015). SSRIs reduce serotonin in the brain, and Frick et al (2015) confirmed increased serotonin activity in social anxiety in a positron emission tomography (PET) study.

[37] Example of RCT (appendix 3B).

(EBM), which Sackett (1996) defined as "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients" (quoted in Wahlberg and McGoey 2007). However, Wahlberg and McGoey (2007) preferred to describe EBM as "a strategy of rationalisation whereby certain individuals are delegated the task of keeping abreast of and collating latest advances, trial evidence and observational studies" (p3) [38]. This is a "social construction of the evidentiary process" (Petryna 2007).

This idea fits with Rose's (1999) point: "Where do [forms of knowledge/evidence] emerge? Which are the authorities who are able to pronounce upon them? Through what concepts and explanatory regimes are they investigated?" (quoted in Wahlberg and McGoey 2007).

Consequently, Wahlberg and McGoey (2007) asked: "Are we sure that randomised controlled trials are the 'best we've got' when it comes to evaluating whether or not a treatment does more good than harm? There are a number of components to this question of whether, and when the RCT is properly seen as the gold standard of EBM [39]. On the one hand, there are debates concerning the 'taintedness' of the evidence-producing process, which often point to the distorting role of the pharmaceutical industry when lobbying for a relaxation of safety criteria, or when selectively publishing trial data in order to present a 'rosier' picture of safety and efficacy of their drugs" (p5).

On the other hand, Cartwright (2007) noted that there is no "gold standard": "Gold standard methods are whatever methods will provide (a) the information you need, (b) reliably, (c) from what you can do and from what you can know on the occasion" (p11).

Kendall quoted in Kendall and McGoey (2007) noted that "when we are deciding about which treatments work best, as compared to others - which is an important clinical question - then, at this point in time, RCTs are probably the nest way of answering that question. There are too many flaws in uncontrolled, non-randomised research. So, I am in favour of RCTs. No matter how problematic they are in the way they're conducted, even when they're done on a shoestring (which they often are if they're not funded by a drug company), they are

[38] EBM has not necessarily removed doubt from medicine because RCTs need to be interpreted, and this has become "the area for dispute and contest" between professionals, patients, and the state (Armstrong 2007) (what Johnson (1972) called indeterminancy, and May et al (2006) "technogovernance"). EBM can be viewed as positive (finding the best evidence) or negative ("a new form of dogmatic authoritarianism" [Feinstein and Horwitz (1997] (Jensen 2007).

[39] Cochrane's (1972) "Effectiveness and Efficacy" is viewed as crucial in RCTs becoming seen as the "gold standard" method (Armstrong 2007). The first RCT in psychiatry may have been in 1954 on chlorpromazine for treating schizophrenia (as it is not entirely clear) (Wessely 2007).

probably the best way that we currently have of deciding which treatments work and which ones don't" (p138).

Abraham (2007) argued that "the quality of the drug trial evidence base greatly depends on the how ethical, commercial and political priorities are set, and not merely on standardised techniques of data processing", and these priorities/standards "reflect a regulatory culture of neoliberal corporate bias, and have been powerfully shaped by the commercial and political interests of the pharmaceutical industry and regulatory institutions" (p41).

Two issues are debated about RCTs for drugs generally (Abraham 2007):

a) The size of the trial - Small differences between the drug being tested and the comparator require a large number of participants to be detected.

b) The comparator - ie: placebo or already-approved drug for the same condition.

Together these issues have implications as in this example with anti-depressants. If a drug produces a positive response in 70% of participants compared to in 30% of the placebo group (table 3.1), then fifty individuals are needed on the trial for a significant difference [40], but if the drug being tested has a 60% response compared to 70% for an already-approved drug comparator, eight hundred individuals would be needed for a statistically significant result (Abraham 2007). Also, in the latter scenario, "if no statistically significant difference between the drugs is found in a head-to-head trial, then it will not be clear whether this is because both drugs have performed equally effectively or ineffectively in that trial" (pp43-44).

Cartwright (2007) distinguished between "clinchers" and "vouchers" in establishing causality. RCTs as a method are "clinchers" - ie: they "clinch the conclusion but are narrow in their range of application" - whereas "voucher" methods "merely vouch for the conclusion but are broad in their range of application" (eg: qualitative comparative analysis as in a literature review).

"Clinchers" are deductive: ie: "if all the assumptions for the correct application are met, then if evidence claims of the appropriate form are true, so too

[40] Small placebo trials may not have a representative sample, and, in some cases, pharmaceutical companies combine (or "pool") the data from different such trials for the same drug. "However, this is a very malleable technique, within which it can be very difficult to know whether commercial interests have produced valid results or claims that reflect narrow sectional goals contrary to those of the potential users of the drug" (Abraham 2007 p46).

- Anti-depressant trials also face a high placebo response rate (varying from 15 to 70% of participants as compared to 30 to 70% for anti-depressants) (Lakoff 2007). This is a problem for drug researchers, who, Lakoff (2007) reported, distinguish between artifactual and real placebo responses. Artifactual placebo responses can be of two kinds - (i) baseline measures of depression are inflated and so later measures after placebo show improvement, and (ii) "regression to the mean". When a condition is fluctuating, the time of the baseline measure could be a "bad day", for instance, and subsequent measures will be seen in relation to that. Real placebo responses include "covert psychotherapy" (ie: the drug administrators listening to or helping the patient beside the treatment being tested). Montgomery (1999) went as far as saying that individuals "who are overtly sensitive to reassurance need to be identified and if possible excluded" (from RCTs) (quoted in Lakoff 2007).

- There is an incentive for drug researchers to screen out placebo-responders from RCTs. For example, the search for personality types of responders. In the 1970s, it was felt that such individuals were high on social acquiescence (ie: more likely to agree with social norms), but now it is agreed that there is no consistent personality type of placebo-responders (Lakoff 2007).

- But drug researchers have found a way to identify placebo-responders. Analysis of anti-depressant trial data by the pharmaceutical company Eli Lilley found that placebo-responders had a stronger improvement in blinded than open-label conditions (ie: when they did not know if they were in the placebo or drug condition). In fact, when the trial was double-blinded (ie: both doctor and patient did not know which group), placebo-responders improved most. This seems to be the opposite of common sense. It appears to be "the doctor's uncertainty" that encourages the placebo response. The upshot is that pharmaceutical companies can identify placebo-responders. RCTs for anti-depressants often begin with a "single-blind placebo run-in period" before the full double-blind stage. Placebo-responders will show a sharp improvement in the placebo condition of the latter stage, and their data can be removed from analysis of the efficacy of the drug (Lakoff 2007).

Table 3.1 - Placebo-responders.

will the conclusions be true" (Cartwright 2007 p12). However, Cartwright (2007) pointed out the difference between the "ideal RCT" (following this logic) and the "real RCT" (with its weaknesses).

Wessely (2007) argued that randomisation is an important strength of RCTs, and it combats confounders like selection bias. But Will (2007) countered that "there is considerable play of judgment around the choice of the object of randomisation" (p87) [41]. RCTs also have

[41] Kraemer (2015) noted a problem with randomisation. If a sample of participants is randomised to treatment or control groups, there is no guarantee that the groups will be matched for relevant characteristics. "When a few baseline variables significantly differentiate the 2 groups at the 5% level, researchers often propose to adjust for those covariates in testing the treatment effect. This is post hoc testing (like offering to bet at pre-race odds on a horse as it approaches the finish line), which frequently leads to false-positive results" (p961).

another control in the form of protocols (ie: inclusion and exclusion criteria), which if researchers follow may lead to "serious difficulties of recruitment" (Will 2007)[42].

Will (2007) referred to the "alchemy of the RCT" "characterised by the ritual invocation of randomisation and control as tools to transform those imperfect materials [science, research and practice] into the stuff of certainty" (p97).

Wessley (2007) argued that another strength of RCTs is the production of evidence that is contrary to common sense and popular opinion. He used the example of single-session psychological debriefing soon after trauma to prevent later PTSD. RCTs found that such debriefing increased the risk of PTSD (eg: Wessely et al 2000) despite the popular belief in the benefit.

RCTs, like those for anti-depressants (table 3.2), use symptom rating scales to "translate subjective experience into quantitative cut-off points and outcome measures, making it possible to assemble and compare groups of patients across sites and between evaluators" (Lakoff 2007 p58). But the success of such rating scales depends on the agreement "on what the salient characteristics of the illness are" (Lakoff 2007).

Since the unexpected "success" of Prozac at the turn of the century, anti-depressants have become the second most prescribed class of drugs in the USA, for example, from "a small, niche market at best" in the 1960s (Lakoff 2007). One explanation for the growth in sales of anti-depressants is "an already-existing but long-ignored group of patients was finally being recognised and treated" (Lakoff 2007). Others preferred to emphasise increased political and economic insecurity which produced emotional distress and the demand for help. Alternatively, it could be the marketing efforts of pharmaceutical companies which "in some sense, 'produced' an epidemic of affective illness" (Lakoff 2007).

Drug development and testing is based in the biomedical model of disease. "According to this model, illnesses are understood as stable entities that exist outside their embodiment in particular individuals, and which can be explained in terms of specific causal mechanisms that are located within the sufferer's body. From the vantage of drug regulation, experimental interventions should show targeted effects: a given drug should work directly on specific disease. Thus an 'anti-depressant' should directly treat 'depression'. The problem, in the case of trials focused on psychiatric illness, is that there are neither agreed-upon inclusion criteria, nor end-points that can reliably indicate when drug efficacy has been achieved" (Lakoff 2007 p59).

Table 3.2 - Growth in anti-depressant use.

[42] There is always a risk of what Cowgill (2015) called "sampling vagaries".

In defending RCTs, Wessely (2007) emphasised what RCTs can and cannot tell us. They can tell if treatment A does more good than harm than treatment B (or no treatment) in condition C. They do not tell if treatment A will absolutely work for a specific patient, if treatment A works on condition D, nor if treatment A works with a patient very different to the sample of the RCT.

Wessely (2007) stated: "The systematically acquired information that results can be used to help future patients, without forgetting that what is truly unique about a patient (and so cannot be studied in a clinical trial) still has to be taken into account when caring for the patient, and for this the treating clinician will often need large amounts of intuition, experience and empathy" (p122).

Petryna (2007) reported on the "offshoring" of clinical trials to middle- and low-income countries (eg: 400 new trials per year in Poland), partly because of "treatment-saturation" in the West (ie: a shortage of other medicine-free volunteers). But also the large number of trials being run as the "advent of blockbuster drugs with sales of over a billion dollars annually has led to the profitable 'me-too drugs' business" (p25), and the demands of regulators.

Petryna (2007) observed: "The increasing choice of Third World citizens to be subjects of global drug trials parallel their poverty status. Even if the trend in drug trial expansion can be justified in terms of potential health benefits, pursuing disadvantaged populations that have (as yet) little or no legal recourse in case of harm involves troublesome ethics" (p28).

3.3. APPENDIX 3B - EXAMPLE OF RECENT RANDOMISED CONTROLLED TRIAL

Binge-eating disorder (BED) appears in DSM-5, and involves recurrent episodes of excessive eating (binges) with a sense of loss of control and psychological distress, but no compensatory weight-loss behaviours (eg: vomiting) as in bulimia nervosa (McElroy et al 2015).

Treatments available include cognitive-behavioural therapy, interpersonal psychotherapy, and drugs. Anti-depressants, for example, reduce the frequency of binges, but do not help with weight loss (which anti-obesity drugs like sibutramine hydrochloride do, but these have safety concerns). Anti-epileptics work on binges and weight loss, but affect cognition leading to discontinuation (McElroy et al 2015).

McElroy et al (2015) reported the benefits of

lisdexamfetamine [43] (usually prescribed for attention-deficit hyperactivity disorder) in a RCT. Thirty-one US sites were involved in the double-blind, placebo-controlled trial over eleven weeks, giving a total of 260 individuals at the start (with 58 not finishing; table 3.3) [44]. Inclusion was based on BED with at least three binge days per week in two weeks prior to baseline, and a body mass index between 25 and 45. There were a number of exclusion criteria (table 3.4).

	Placebo	30 mg/d	50 mg/d	70 mg/d
Total	**17**	**15**	**13**	**13**
Adverse effects	0	3	1	3
Protocol violation	7	2	2	5
Randomised by participant did not provide information	6	4	4	2
Lost to follow-up	4	6	3	2
Other	0	0	3	1

(Data from McElroy et al 2015 figure 1 p238)

Table 3.3 - Reasons for discontinuation based on group.

- Current bulimia nervosa, anorexia nervosa, ADHD, depression, or another psychiatric disorder.
- Lifetime history of bipolar disorder, psychosis, or other psychiatric conditions that may confound results.
- Psychological or weight-loss intervention (current or in last three months).
- Use of psychostimulants in last six months.
- Personal or family history of cardiovascular disease.
- Current or lifetime substance abuse.
- Current drug treatment for psychiatric conditions or weight-loss (in last sixty days).

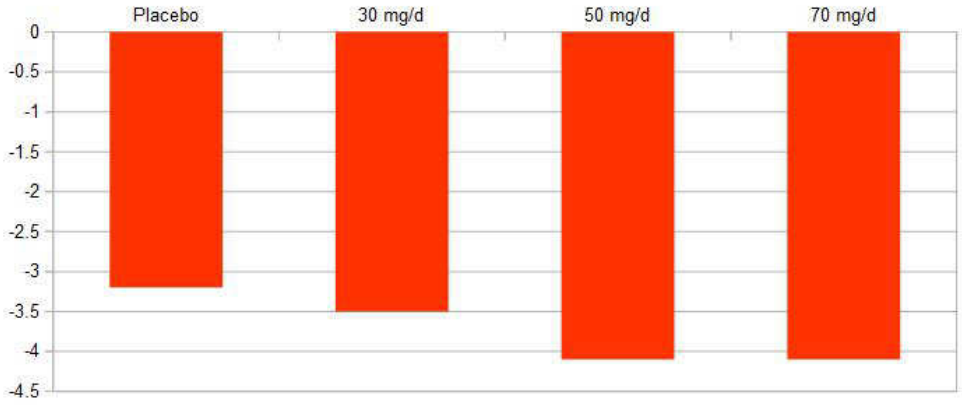
Table 3.4 - Exclusion criteria used by McElroy et al (2015).

[43] Lisdexamfetamine is a dextroamphetamine, which inhibit reuptake of dopamine (ie: reduce the amount in the brain), and individuals with BED have been reported to have higher levels of dopamine than non-sufferers (McElroy et al 2015).

[44] Most participants were female (82%) and/or White (78%).

The participants were randomised to one of four groups - placebo, or three different daily doses of lisdexamfetamine (30, 50, or 70 mg) - for eleven weeks [45]. The study was blinded by making the placebo and drug identical in size, weight, shape and colour. The primary outcome measure was number of binge-eating days based on self-reported diaries.

The number of binge-eating days at week 11 was significantly reduced in the two higher dose groups (figure 3.1). There was a significant weight loss in the drug groups compared to placebo (figure 3.2).



(Data from McElroy et al 2015 table 2 p241)

Figure 3.1 - Mean change in number of binge-eating days between baseline and week 11.

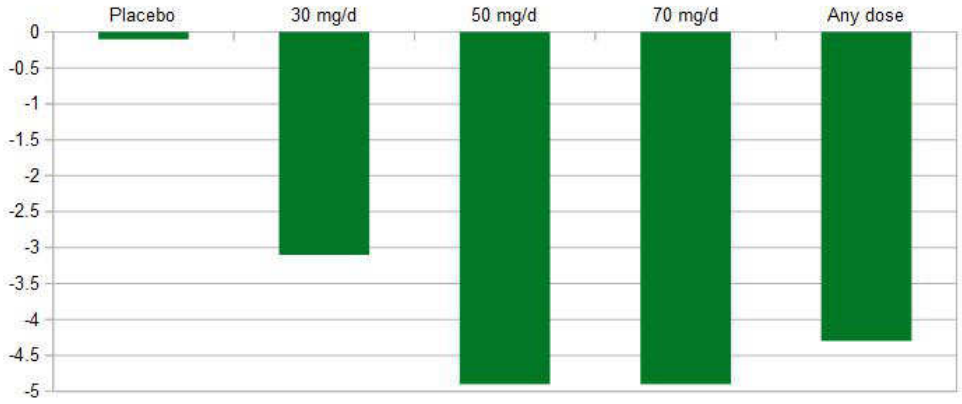


Figure 3.2 - Mean change in body weight from baseline (kg).

[45] Adherence was defined as at least 80% of pills taken, based on counting remaining pills in packet.

Severe side effects were limited, and similar to adults with ADHD taking lisdexamfetamine [46].

The researchers concluded that lisdexamfetamine was worth "further analysis" as a treatment option for BED.

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[46] McElroy et al (2015) stated: "The safety profile of lisdexamfetamine was generally consistent with that seen in studies of lisdexamfetamine in adults with ADHD in the types and frequency of adverse effects. In the present study, small mean increases in heart rate were noted with lisdexamfetamine treatment, consistent with other studies of psychostimulants. Discontinuation owing to lack of efficacy (none) and adverse events (7 of 259 participants) was comparable with results of selective serotonin reuptake inhibitor studies and somewhat lower than that seen in topiramate trials.

One participant died during the study. Post-mortem toxicology analysis reported that methamphetamine/amphetamine levels were consistent with a methamphetamine overdose. This was, to our knowledge, the first such report of a methamphetamine overdose during the course of a lisdexamfetamine clinical trial, because no previous publications of lisdexamfetamine clinical trials across multiple indications have reported methamphetamine overdose" (p243).

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4. OVER- OR UNDER-TREATMENT OF MENTAL DISORDERS

4.1. INTRODUCTION

Some studies (eg: Druss et al 2007) have suggested that up to half of individuals treated for mental disorders worldwide do not have a formal diagnosis (Bruffaerts et al 2015). In other words, the "worried well".

Bruffaerts et al (2015) challenged this conclusion:

i) Individuals may need treatment for reasons other than a formally diagnosed mental disorder (eg: suicidal behaviour).

ii) Previous studies did not distinguish the types of treatment (eg: bereavement counselling, self-help groups).

iii) Most studies were carried out in the West.

Bruffaerts et al (2015) rectified the last problem by the use of data from the World Health Organisation's (WHO) World Mental Health surveys in 23 countries between 2000 and 2009 (with over 62 000 respondents). These involved face-to-face interviews and formal diagnosis of mental disorders using DSM-IV criteria. Of the sample, 9.2% of individuals had received treatment in the last year, and among them, 16% did not need treatment (ie: no current or lifetime diagnosis of mental disorder or other relevant problem like sub-threshold disorders) [47].

This is an issue because many cases of mental disorders go untreated through lack of resources. Buffaert et al (2015) stated: "After all, in times of economic restraints and limited financial resources, more emphasis may be given to (re)allocate resources to those with the highest needs or people who are especially at risk for developing mental disorders like younger cohorts, females or those with lower educational

[47] But this group only made up 10% of visits to treatment, and less than 8% were seen by psychiatrists.

attainment. Instead, we found that those treated were older, higher-educated females who are in general at lower risk for developing serious emotional problems. This suggests that at least a part of the treatment resources may be misallocated and that this may be more pronounced in high-income countries. However, this is not a suggestion to omit mild emotional problems from mental healthcare but rather a suggestion that formal healthcare for mild disorders could be more focused on those who are at risk of developing more serious emotional problems" (p107).

4.2. ANTI-DEPRESSANTS

In Europe as a whole, anti-depressant prescriptions have increased by about 20% per year between 2000 and 2010 [48], but this hides great variety among individual countries (eg: over six times more prescriptions per person in Iceland than Estonia) (Lewer et al 2015) [49]. Why the difference between countries in Europe?

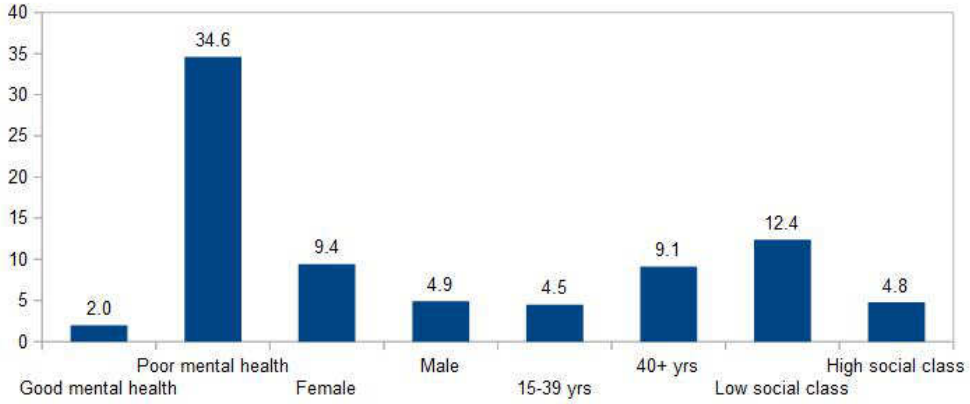
Lewer et al (2015) used data from Eurobarometer 2010 to answer this question. Eurobarometer is a face-to-face survey of around one thousand individuals in each of twenty-seven European Union countries. The outcome variable (or dependent variable) was measured by the question: "Have you taken any anti-depressants in the last twelve months?", with four options for reply. Socio-demographic information and attitudes towards mental illness were collected in the survey and used as the independent variables in statistical analysis.

Overall, in 2010, 7.2% of respondents (n = 1995) had used anti-depressants in the past year (12-month prevalence) [50], with a range of 15.7% in Portugal to 2.7% in Greece. The likelihood of being in this group was linked to poorer mental health, being female, being older (over 40 years old), not in paid employment (including looking after the home), lower perceived social class, and in financial difficulties (figure 4.1).

Anti-depressant use was also higher in countries where individuals with mental illness were perceived as dangerous by residents, or in countries where recovery was considered more likely or individuals were not blamed for their mental illness.

Of the 1995 respondents who had taken anti-depressants, 58% reported regular use (varying from 89% in Sweden to 19% in Bulgaria). The key variables here were poorer mental health, being younger (below 40 years

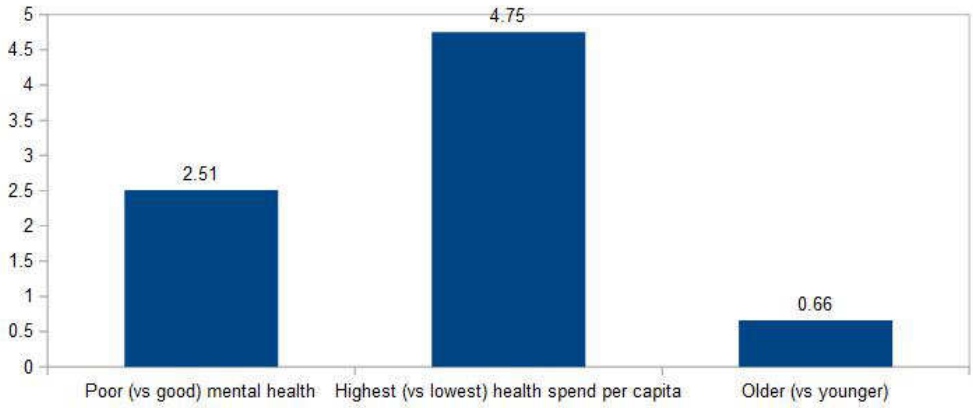
[48] The pharmaceutical industry has a role to play here (appendix 4A).
[49] Prescriptions have also increased for children and adolescents in recent years (appendix 4B).
[50] 3.7% in 2000 (Alonso et al 2004).



(Data from Lewer et al 2015 table 1 p222)

Figure 4.1 - Percentage using anti-depressants in past year.

old), and comfortable talking to someone with a mental health problem (figure 4.2). In terms of country-wise, regular anti-depressant use was associated with high healthcare spending per capita, where individuals with mental illness were considered to be unpredictable, but likely to recover. The researchers admitted that these "results relating to stigmatising attitudes are complex and merit some discussion... [for example]... the idea that medicalised views of mental illness may act as a 'double-edged sword', leading to higher rates of treatment but also greater social distance..." (Lewer et al 2015 p225).



(Data from Lewer et al 2015 table 2 p224)

Figure 4.2 - Significant odds ratios for regular use of anti-depressants.

4.2.1. Anti-Depressants and Suicide

The association between anti-depressant use and suicide is inconsistent in the research literature. Increasing anti-depressant use and decreasing suicide would suggest that anti-depressants are beneficial, while increasing anti-depressant use and increasing suicide could be evidence of their ineffectiveness.

In the former case, at an individual level, Gibbons et al (2007), for instance, found lower suicide at six months among 200 000 adults on the US Veterans Affairs Medical System taking anti-depressants than not. Other studies, particularly with young people, find that anti-depressant use increases suicidal behaviour (eg: Bridge and Axelson 2008).

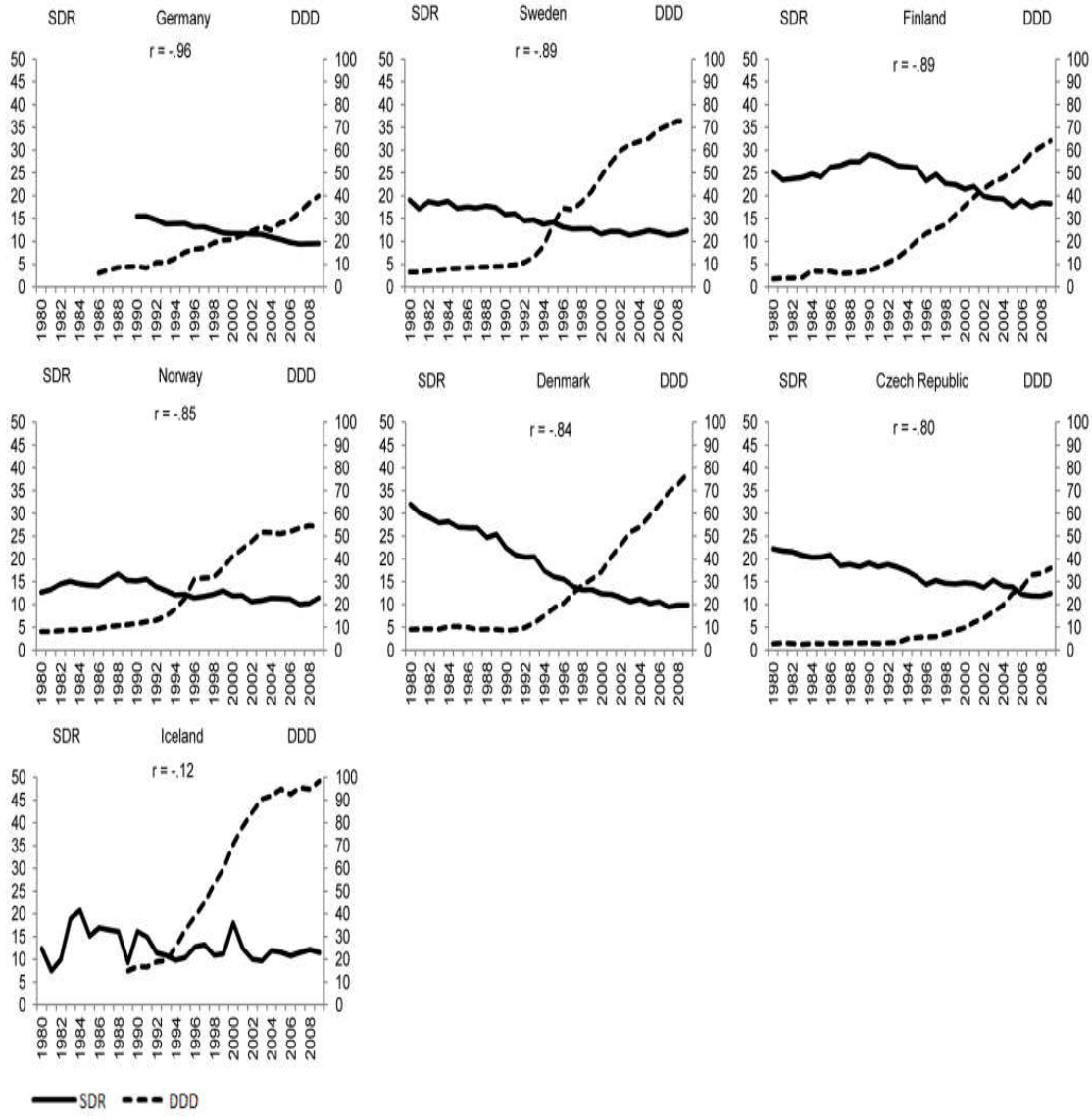
Gusmao et al (2013) observed: "Studies vary in basic terminology, definition of outcomes and time periods considered, drugs and other interventions assessed, and statistical methods, leading to seemingly contradictory results".

At a national level, Ludwig and Marcotte (2005) calculated that an overall increase in sales of one anti-depressant pill per capita was associated with a 2.5% decline in the suicide rate, using data for selective serotonin reuptake inhibitors (SSRIs) from the USA, Canada, Australia, and twenty-four European countries between 1980 and 2000. "However, they acknowledged that this finding was qualified by SSRI sales data having to be imputed prior to 1990 due to a lack of sales data. Nonetheless, their finding suggests that greater utilisation of SSRI, particularly for adults, might be a cost effective strategy from a public health perspective, with one suicide averted for every 300,000 pills sold" (Gusmao et al 2013).

Gusmao et al (2013) found that "suicide rates have tended to decrease more in European countries where there has been a greater increase in the use of anti-depressants". They used data from twenty-nine European countries to calculate "defined daily dosage" per thousand individuals per day (DDD/1000/day) for the period 1980 to 2009. Completed suicide rates came from World Health Organisation (WHO) data, giving a standardised death rate (SDR) for suicide. In the statistical analysis, gross domestic product (GDP), alcohol consumption, unemployment, and divorce rates were controlled for.

During the study period, DDD/1000/day had risen from an average of 13.69 to 43.91 (average growth per annum of 20%), though there was variation between countries (eg: 3% growth p.a in Switzerland versus 34% in Sweden). SDR had declined from 19.06 to 12.93 during the study period (again with country variations).

Overall, "there is an inverse statistically significant correlation [51], with an increasing use of anti-depressants and greater reductions in the suicide SDR... In almost all countries, an increase of DDD/1000/day seems to correspond with a decrease in suicide SDR, although in countries where suicide rates are already low, anti-depressants appear to have less impact" (Gusmao et al 2013) (figure 4.3).



(Source: Gusmao et al 2013 figure 1)

Figure 4.3 - Suicide rate (SDR) and use of anti-depressants (DDD) for seven countries with longest records in the study (nineteen years of continuous data).

[51] Portugal was the only exception with a positive correlation. "This can possibly be explainable by the lack of precision of suicide register and over-estimation of undetermined violent deaths concealing suicides" (Gusmao et al 2013).

The researchers made the following calculation: "If 10 DDD/1000/day corresponds approximately to 1% of population treatment point prevalence, our data suggests that there could have been an increase in treatment for depression of 4% of this population correlating with a saving of 31.670 deaths by suicide in the last year covered, equivalent to 650 people treated for each life saved, per year" (Gusmao et al 2013).

Gusmao et al (2013) were aware of the main limitations: "Because this is an ecological study, we emphasise that we cannot depict causal links and therefore these results must be interpreted with great caution". Other limitations of the analysis including reliance on the accuracy of data used (eg: definition of death as suicide or undetermined), lack of information at an individual level (eg: whether individuals take the drugs prescribed), and reason for prescription of anti-depressants (Gusmao et al 2013).

4.3. ANTI-PSYCHOTICS AND LEARNING DISABILITIES

Sheehan et al (2015) found that the "proportion of people with intellectual disability who have been treated with psychotropic drugs far exceeds the proportion with recorded mental illness". They analysed the data of 3.7 million active patients in 571 general practices in the UK in The Health Improvement Network (THIN) database for the period 1999 to 2013. The focus was upon 32 306 adults with intellectual disability or learning disabilities (LD) (defined as "a significant deficit in cognitive and adaptive function with onset during the developmental period"; Sheehan et al 2015). The researchers looked for records of diagnosis of serious mental illness, challenging behaviour (eg: self-injury, aggression, arson), and prescription of psychotropic drugs.

Of the sample, 21% had a diagnosis of mental illness, 25% a history of challenging behaviour, and 49% a history of prescription of psychotropic drugs at the beginning of the study. By the end of the study, 63% had been prescribed such drugs. This suggested that psychotropic drugs were prescribed to individuals without a diagnosis of mental illness.

People with a record of challenging behaviour were over twice as likely to be prescribed anti-psychotics than non-challenging behaviour individuals (237 vs 96 per 10 000 person years).

The rate of prescription of anti-psychotics was nearly twice that of the general population (132 vs 61-70 per 10 000 person years).

Put another way, 70% of those with LD prescribed an anti-psychotic drug did not have a diagnosis of severe

mental illness on their medical record, which compares to around three-quarters in a Norwegian study (Holden and Gitlesen 2004). In the UK general population, it is around half (Marston et al 2014).

Sheehan et al's (2015) study, like any using medical records, depends upon the accuracy of the data recorded, and only covers information that comes to the notice of doctors. The researchers made the following comments on the data:

> The results are of recorded diagnoses, which may not correspond to the true rate of illness in the population. Some diagnoses may have been entered in the free text of the computer system, which we did not interrogate. We restricted our mental illness code lists to the most common mental disorders and excluded categories such as personality disorder and substance misuse; further work is necessary to report on a wider range of illnesses. The challenging behaviour code list has not been externally validated, and we cannot report its sensitivity or specificity. However, it showed the expected associations with conditions known to increase the risk of challenging behaviour and is therefore likely to adequately represent the concept. Certain characteristics, such as degree of intellectual disability, are not always well recorded in THIN, and we have not been able to extend our interpretations to include analysis of these variables. We did not include our own comparison group without intellectual disability and relied on previously published data to put our results in context. Although prescription of drugs is well recorded in THIN, we will have missed the minority of prescriptions issued in secondary care (Sheehan et al 2015 pp7-8).

4.4. APPENDIX 4A - PHARMACEUTICAL INDUSTRY

In reference to the pharmaceutical industry ("pharma"), Matheson (2008) referred to their research as "product canons" that "integrate scientific truth-claims and commercial positioning, generating knowledge with implicit commercial functionality. From this platform, pharma stamps consensus-building 'narratives' into medical-scientific discourse, in which 'problems' arise and are 'solved' by drugs" (p355).

Matheson (2008) noted the processes used by the pharmaceutical industry, like highlighting an emerging or growing problem, an "unmet need", or expanding a "disease's range" (table 4.1). For example, emphasising the previous under-diagnosis of bipolar disorder generally (highlight size of problem), or the inclusion

of children in diagnosis (expand disease's range) (Healy 2006). "'Problems' are complemented by the promotion of drugs as 'solutions'" (Matheson 2008 p365).

STRATEGY	PURPOSE
Highlight emerging therapeutic issues	Prepare and establish new market
Highlight size of problem	Expand current market
Highlight "unmet needs"	Establish new market. and/or expand current market
Expand disease's range	Prepare and establish new market, and/or expand current market
Promote new diseases	Open new market, and/or expand current market
Promote drug solutions	Protect and/or expand market share

(Based on Matheson 2008 table 2 p364)

Table 4.1 - Some strategies used by the pharmaceutical industry to influence diagnosis of "problems".

"Drug narratives" are produced to persuade, using techniques like the volume of data, authority, or even subterfuge (eg: embed sponsored content in non-sponsored content) (table 4.2) (Matheson 2008).

TECHNIQUE	EXAMPLE
Volume of data	Many published studies
Authority	"Key opinion leaders"
Different values	Emotional appeal to patients and caregivers
Rhetoric	Exaggerate benefits
Incentives	Money
Subterfuge	Downplay or conceal influence of pharmaceutical companies in promoting drug

(Based on Matheson 2008 table 3 p368)

Table 4.2 - Some techniques used by the pharmaceutical industry to persuade.

Matheson (2008) concluded: "While the establishment of a more scientific basis for medicine is to be welcomed, it must be acknowledged that this is proceeding to at least some degree on pharma terms. Pharma's influence within the overall dispositif of biomedical knowledge production is pervasive, reaching beyond

research per se into the networks, institutions, cultures and mind-sets of academic medicine and science—though many within these communities may be but dimly aware of such influence. Pharma's contribution to medical-scientific knowledge is manifest on many levels, including the dominance of the biomechanistic model of human health, the choice of what problems are investigated, the drugs themselves and associated clinical and mechanistic research, and the way specific aspects of human biology, pathology and medicine are constructed" (p377).

Applbaum (2010) talked of a "shadow science" as the pharmaceutical company Eli Lilly tried to "drown out" reports of serious side effects of their anti-psychotic medication "Zyprexa". Thus, the company "focused their scientific and sales attention not on the clinical characteristics of their drug as determined by their own research or that of external researchers, but on how prescribing physicians perceived those characteristics. In other words, the company treated the medical concerns associated with their drug as a relative and fungible truth - in short, as a brand truth that they had the right and resources to control" (Applbaum 2010 pp237-238). So every time a negative study was published about the drug, Lilly sponsored a contradictory study.

Applbaum (2010) summed up: "I suggest that what we are witnessing is not a subtle process of the commercialisation of scientific exploration, but the reconfiguration of the infrastructure for knowledge management in the service of promoting brand values" (p251).

4.5. APPENDIX 4B - CONTEXTUALISING PAEDIATRIC DEPRESSION

In an article about the use of selective serotonin reuptake inhibitor anti-depressants (SSRIs) with depressed children and adolescents, Wilson (2011) was interested in "the neuro-pharmaco-ideo-affective affiliations that are the stuff of depressive states in treatment [ie] the systemic (dynamic, entangled, indebted) relations between the neurology of depressed moods and the pharmaceuticals that are deployed to regulate them" (p277). She was seeking to understand the complexity of "paediatric depressions" rather than whether SSRIs were effective or not.

Thus, "depressed states are not simply the effects of isolated parts of the body (gene, hormone, neurotransmitter, synapse); nor are they the effects only of a large and complex neurological system (central-enteric-peripheral); nor are they the effects of the interactions of neurological events with their environments... Instead,... depressions are the phenomena

that neuro-serotonergic-ideational intra-actions may become" (Wilson 2011 p290) [52].

Barad (2007) defined intra-action, "in contrast to the usual 'interaction', which assumes that there are separate individual agencies that precede their interaction, the notion of intra-action recognises that distinct agencies do not precede, but rather emerge through, their intra-action" (quoted in Wilson 2011).

This idea of a wider understanding of depression can be linked to Miller (2007) "dynamic adaptive systems framework", "in which brain reward systems, gender, the biological changes of puberty, the development of the capacity for abstract thought, cortical regulation of emotion and attentional impairment together breed the conditions out of which paediatric depressions arise" (Wilson 2011 p292).

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[52] This fits with "Gut feminism" (Wilson 2004) and "the systemic nature of depression" - ie: depressed states "are caught up not simply with the brain, nor with social events, but also with the biological particularities of gut action" (Wilson 2011 p283).

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5. GENE-ENVIRONMENT INTERACTION IN PSYCHIATRIC DISORDERS

5.1. END OF EITHER/OR DEBATE

The simple either/or debate of nature and nurture has been replaced by the acceptance of gene-environment interactions as explaining behaviour [53].

Three such interactions have received much interest in psychiatry, though Zammit et al (2010) have questioned the replicability of the findings:

i) Short version of a serotonin transporter gene (5-HTTLPR) and stressful life events increasing the risk of depression compared to the long version of the gene and stress, and the short version of the gene or stressful life events by themselves (Caspi et al 2003).

ii) Low-activity variant of monoamine oxidase A gene (MAOA) and childhood maltreatment leading to anti-social and violent behaviour in males ((Caspi et al 2002).

iii) Variant of COMT gene and cannabis use leading to psychosis (Caspi et al 2005).

Zammit et al (2010) also raised concerns about the understanding of the interaction in the studies. The authors distinguished two definitions of interaction:

- Definition 1 - "Many different combinations of risk factors will exist for a disease in a population... Some of these risk factors are likely to be genetic, and some non-genetic, and both types almost certainly play a contributory role in causing most, if not all, cases of disease" (p65).

- Definition 2 - "Statistical interaction occurs when the risk of disease if exposed to both the gene (A) and the environmental exposure (B) is different from that predicted by the statistical model being used" (p65).

[53] This either/or dichotomy is also challenged by epidemiology (appendix 5A) and epigenetics (appendix 5B).

The authors were concerned with the latter definition, particularly when statistical models vary. Zammit et al (2010) stated that the "debate over whether gene–environment interactions 'exist' is not helpful. The more useful questions are as follows: Can we reliably describe the patterns of disease risk for joint exposure to two (or more) risk factors? And, most importantly, is doing so likely to help us understand more about disease aetiology or prevention?" (p65).

5.2. VOLUNTEERS

Modern research on the complexity of the gene-environment interaction requires volunteers for the databases, including their blood and tissue samples, and details about their lifestyles. Svendsen and Koch (2008) were interested in the recruitment of such volunteers by pharmacogenomic researchers (interviewed in Denmark). The researchers used "specific devices (letters, informed consent forms, telephones), actualise specific norms (bioethical frameworks) and perform specific acts (send out invitations, make telephone calls) to make patients see themselves as participants in research" (Svendsen and Koch 2008 p403). Writing to individuals with a specific psychiatric diagnosis, say, asking them to volunteer made them feel "special" (in the sense of select), and offered the opportunity of a positive moral choice.

"In pharmacogenomic research the individual patient is both the resource for and the target of the development of personalised medicine. Yet the patient who acts as the resource in the present is not likely to be the person who will eventually receive personalised medicine, if this goal is ever realised. Recruitment asks the individual patient to metaphorically 'undress'— not to get the tailored medicine herself, but to make it possible for a collective of future citizens. In this sense participation in the pharmacogenomic research project... may be conceived as a form of biological citizenship (Rose and Novas 2005) and interpellation [54] as the process through which patients (may) come to recognise themselves as important resources of future public health care" (Svendsen and Koch 2008 p415).

Baart (2010) summed up the optimism of genetics [55]: "The discovery in 1993 that Huntington's disease is caused by an inherited defective gene fuelled optimism for the role played by genetics in mental disorders in

[54] Althusser (1971) used the term "interpellation" to refer to "the process of being addressed, hailed in a particular way, and of coming to recognise oneself as a specific object" (Svendsen and Koch 2008 p403).

[55] This is also seen in synthetic biology (appendix 5C).

general. Many people both within and outside the scientific community thought that it was only a matter of time before the genes responsible for schizophrenia, depression and anxiety disorders would also be discovered... It was felt that once the pathway taken by genes in malfunctioning brains had been mapped, a cure would present itself and the war on mental illness could finally be won, just as the war on infectious disease had been" (p256).

On the other hand, she said, many "feared the future, as they envisaged full medicalisation, where psychiatric subjects would be stigmatised as being chronically ill with diseased brains, and therefore ineligible for social intervention and psychological help... This perspective also rested on the firm belief in a future where genes would function as the causal factors in psychiatric disorders" (p257).

But reality is neither of these: "To date, psychiatric genomics has yet to turn this future into reality and it is currently doubtful if it ever will. The general feeling is that genomics will not lead to genetic determinism in psychiatry because psychiatric disorders emerge as polygenetic and multi-factorial products of genetic and environmental factors" (Baart 2010 p257).

Saying that, Hedgecoe (2001) worried about "geneticisation by stealth" as the "narrative" of genetics would come to dominate discussion about schizophrenia, say. Baart's (2010) interviews with scientists, policymakers, and patients involved in genetics research in Holland, however, suggested the possibility of "different psychiatric futures".

5.3. APPENDIX 5A - EPIDEMIOLOGY

The "epidemiology wars" (Poole and Rothman 1998) describe disputes between epidemiologists and researchers over issues like the causation of disease in a population, particularly when it is multiple and complex. Explanations can be grouped into three - evolutionary factors, life course, and lifestyle and community - which can be overlapping, complementary and combinable (Shim and Thomson 2010).

Multi-causality (ie: a disease had numerous determinants) has defused the "epidemiology wars", and "affirms that causal pathways occurring on different levels of causation operate simultaneously and synergistically to determine the incidence and distribution of chronic disease in complex ways" (Shim and Thomson 2010 p163).

However, researchers struggle with the practicalities of such ideas. In the field of epidemiology, Shim and Thomson (2010) distinguished a

divide in resolving this problem:

i) "Inductive ethic" - collect data and then develop models of causation (with an emphasis on the biological).

ii) "Deductive ethic" - theory-drive (ie: before data collection) (with an emphasis on the social).

Shim and Thomson (2010) summed up: "Those who espouse the deductive ethic tend to black box the genetic composition of the body and focus instead on variations in the environment and its interactions with and impacts upon the body. In contrast, others working within the inductive approach tend to black box the environment, and concentrate instead on genetic variation and its associations with disease risk and susceptibility" (p173).

5.4. APPENDIX 5B - EPIGENETICS

Experiences can change epigenetic marks (eg: chemicals) on chromosomes, which influence gene activity without changing the gene. One analogue is that genetic mutations alter the meaning whereas epigenetic changes alter activity (Nestler 2011).

Nestler (2011) described this process in a mouse model of depression [56]. Pairs of male mice were placed together in a cage for ten days, of which one mouse was highly aggressive and the other not. The latter was consequently bullied, and showed the signs of depression (eg: withdrawn, anxious, no long enjoy pleasurable activities). At the DNA level of the animals, epigenetic modifications were found in 2000 genes in the reward area of the brain. "So it seems that depression may shut down genes important to activating the part of the brain that allows an animal to feel good, creating a sort of 'molecular scar'" (Nestler 2011 p61).

But about one-third of bullied males did not develop depressive signs. These individuals showed additional epigenetic changes in the brain's reward area. "The findings suggest that this alternative pattern of modification is protective and that resiliency is more than just an absence of vulnerability; it involves an

[56] Animal models are founded on the hope of "medical breakouts and better health for humans", but "the overall success rate translating knowledge from the animal laboratory into clinical practice remains low..., creating moral as well as economic legitimacy problems..." (Dam and Svendsen 2015).

Translational medicine or translational research is the attempt to develop new treatments for humans from basic animal research. Dam and Svendsen (2015) referred to the "patientisation" of animals as they are used in research, and also the concern about making them "sufficiently human" (Thompson 2013) in a biological sense.

active epigenetic programme that can be called on to combat the effects of chronic stress" (Nestler 2011 p61).

5.5. APPENDIX 5C - BIOTECHNOLOGY

Synthetic biology is an example of "new emergencies in the life sciences" (Sunder Rajan 2006) [57]. It has applied "engineering design principles" to biology in three ways - "device-based standardised construction", the "re-engineering of microbes as biotechnologies", and the "whole genome engineering of cellular 'chassis' production" (MacKenzie 2010). Put simply, biological substances are treated as any material that can be engineered or designed.

MacKenzie (2010) challenged the assumption that these processes can be done, not from a technical point of view, but that there is "an aggregate of practices" behind them. "Above all, unquestioning acceptance of the principles offers little insight into how synthetic biology becomes credible and contagiously desirable as a way of working with biological substance" (MacKenzie 2010 p182).

MacKenzie (2010) distinguished two concepts underlying the design process in synthetic biology - "meta-technique" and "meta-material". The former refers to how techniques are assembled through collaboration and standardisation (eg: computerised "design toolbox"), and "meta-material" "suggests ways of thinking about the dynamism of living things infused by models, constructs and layered work-processes" (MacKenzie 2010 p180). Pottage (2006) stated that "what is significant about synthetic biology is not (just) that is suspends evolutionary genealogies, but that it collects biological elements into digital media and modes of organisation" (quoted in MacKenzie 2010 p189).

There is also a wider context to the development of synthetic biology, namely neoliberal capitalism and biotechnology companies ("biocapital"), and as Sunder Rajan (2006) pointed out: "understanding biocapital involves analysing the relationship between materiality and modes of abstraction that underlie the co-emergencies of new forms of life science with market regimes for the conduct of such science" (quoted in MacKenzie 2010 p194).

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[57] Genetic-related technologies are "introduced in new social contexts and cultures, they produce or engage with alternate or conflicting meanings and practices that simultaneously differ while also sometimes appealing to or repeating previous articulations or primary intended effects" (Gibbon et al 2010 p442). The authors linked this to Derrida's (1982/1968) idea of "iterability".

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6. DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS (DSM) AND ITS CHANGING DEFINITIONS

6.1. CHANGING DEFINITIONS

The diagnosis and classification of mental problems as disorders or illness using categorical criteria as in the "Diagnostic and Statistical Manual of Mental Disorders" (DSM) has been criticised in a number of ways (Green 2014):

i) The labels used heighten social stigma towards sufferers.

ii) The "apparent medical nature of the process" justifies the questionable practices associated with diagnosis (eg: the power to detain individuals).

iii) The focus on biological and individual explanations for mental problems.

iv) "Diagnostic categories are said to give rise to tautological 'pseudo explanations' (that is, 'he is unhappy because he has Depression'), entrapping people in a sort of false consciousness about their emotions" (Green 2014 p362).

v) The use of the term "mental disorder" suggests a personal flaw with the individual.

Alternative concepts than illness have been proposed, like "reaction patterns", "problems of living", or "complaints" (Green 2014). Could psychiatry do without a classification system?

The use of diagnostic criteria allows communication about a specific phenomenon, and even if DSM were not used, Green (2014) observed, "In a field which specialises in the cognitive and emotional problems faced by humans, it seems unlikely that we can get by without having some way of discussing those problems in themselves. Unless we believe there are no interesting similarities from case-to-case classification..." (p366).

Classification also facilitates the development of knowledge about a phenomenon, and subsequently, the application of evidence-based treatments. Green (2014) argued that a system of classification is inevitable,

"the problem is not their very existence, but the absolute privileging of one set of categories over another" (p369).

If this is the case, how to overcome the labelling (and associated stigma) of any system of classification? One answer is "identity disregard" (Sen 2007) (ie: individuals have more than one identity). So, "people who get designated 'patients' also continue to be sons, mothers, sports fans, professionals. However, health-care professionals would also do well to stay ever aware that even when someone very strongly identifies (or, more often) is identified, as belonging to a particular group (patient, sufferer of Depression, Autistic), there remain other identities and affiliations of greater importance" (Green 2014 p370).

A further issue is that "once someone has been labelled by a doctor, there is a tendency for this fact to take on a power which facilitates the mistreatment and stigma identified by the critics of diagnosis" (Green 2014 p371). In other words, a constructed category becomes cement-hard true when used by an expert (ie: reification). Green (2014) proposed a solution to this problem: "Clinicians should be obliged to inform patients that psychiatric diagnosis differs from physical diagnosis in key ways. One obvious fact... is that there exist no physical 'biomarkers' to define many putative mental 'illnesses... Clinicians should also emphasise rather than obscure the political- and committee-led nature of systems such as DSM or its potential replacements" (p373).

In conclusion, Green (2014) said: "Any psychiatric language or system of identities should be viewed as being one vocabulary among many. If it can recognise and accommodate the plural affiliations and commitments of its service users and also remain 'ironic enough' (Rorty 1989) about the use of official languages, the seeds can be sown for a genuinely participatory and liberating clinical practice" (p374).

6.2. EATING DISORDERS

In DSM-IV (APA 2000), anorexia and bulimia nervosa were the main categories of eating disorders, along with "eating disorders not otherwise specified" (EDNOS). This latter category included binge eating disorder, which became a separate diagnosis in DSM-5, and EDNOS was replaced by "other specified feeding or eating disorder" (OSFED) and "unspecified feeding or eating disorder" (UFED) in DSM-5. The diagnostic criteria for anorexia and

bulimia were changed slightly between DSM-IV and DSM-5 [58].

What are the implications of these changes for the prevalence rates of eating disorders? For example, Fairburn and Cooper (2011) reclassified DSM-IV diagnoses using DSM-5 criteria [59], and anorexia increased while bulimia diagnoses were stable. This was supported by Mancuso et al's (2015) study of 117 individuals at the Body Image and Eating Disorder Treatment and Recovery Service in Melbourne, Australia. All participants completed the 28-item Eating Disorder Examination - Questionnaire (EDE-Q) (Fairburn and Beglin 1994) [60].

However, Sysko et al (2012) [61] and Machado et al (2013) [62] found that both anorexia and bulimia diagnoses increased with DSM-5.

There were decreases in the "residual" categories (ie: EDNOS to OSFED/UFED) in all four studies (table 6.1). "These data suggest that the DSM-5 criteria may reduce the prevalence of EDNOS by increasing the proportion of individuals who meet diagnostic criteria for a full-threshold eating disorder, particularly anorexia nervosa and binge eating disorder" (Mancuso et al 2015 p519).

6.3. CULTURAL CONCEPTS OF DISTRESS

DSM-5 (APA 2013) distinguished a number of "cultural concepts of distress" including:

- Ataque de nerviois (Latino: "attack of nerves").
- Dhat syndrome.
- Khyal cap ("wind attacks"; Cambodian; similar to panic

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[58] Other key changes between DSM-IV and DSM-5 include:

- Schizophrenia - at least one symptom must be delusions, hallucinations or disorganised speech in DSM-5, as well as removal of sub-types.
- Obsessive-compulsive disorder (OCD) move from "Anxiety Disorders" to "Obsessive-Compulsive and Related Disorders" (which includes hoarding disorder).
- Post-traumatic stress disorder (PTSD) and acute stress disorder moved from "Anxiety Disorders" to "Trauma- and Stressor-Related Disorders". PTSD has three major symptoms in DSM-IV, but four in DSM-5 - re-experiencing, arousal, avoidance, and numbness (now separate symptom).

[59] Approximately 150 consecutive patients at their UK clinic for eating disorders.

[60] Eg: "Over the past four weeks have you been consciously trying to restrict (cut back) the overall amount that you eat, whether or not you have succeeded?". Scored as:

- 0 (no attempt at restraint)
- 1 (attempted to exercise restraint on 1 to 5 days)
- 2 (attempted to exercise restraint on less than half the days; 6-12 days)
- 3 (attempted to exercise restraint on half the days; 13-15 days)
- 4 (attempted to exercise restraint on more than half the days; 16-22 days)
- 5 (attempted to exercise restraint almost every day; 23-27 days)
- 6 (attempted to exercise restraint every day).

[61] Participants at Columbia Center for Eating Disorders in USA.

[62] This study took place in Portugal.

	Fairburn & Cooper (2011)	Sysko et al (2012)	Machado et al (2013)	Mancuso et al (2015)
Anorexia: DSM-IV DSM-5	 8 29	 33 41	 15 18	 35 55
Bulimia: DSM-IV DSM-5	 39 39	 27 33	 12 15	 19 19
Binge eating disorder (DSM-5)	7	13	6	5
EDNOS OSFED/UFED	53 25	40 14	73 51	46 29

(Data from Mancuso et al 2015)

Table 6.1 - Reclassification of individuals (%) using DM-IV and DSM-5 criteria in four studies.

attack).
- Kufungisisa ("thinking too much"; Shona of Zimbabwe; similar to depression).
- Maladi moun ("humanly caused illness" or "sent sickness"; Haitian).
- Nervios (Latino).
- Shenjing shuairuo ("weakness of nervous system"; Mandarin; similar to neurasthenia).
- Susto ("fright"; Latino).
- Taijin kyofusho ("interpersonal fear disorder"; Japan; similar to social anxiety).

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7. MENTAL ILLNESS AND BEING A VICTIM

7.1. INTIMATE PARTNER VIOLENCE

Intimate partner violence (IPV) covers emotional abuse, threatening behaviour, and physical and sexual violence by current or former sexual partners (Khalifeh et al 2015). In Western countries, about one-fifth of women and one in ten men report experiencing IPV at some point in their lives (lifetime prevalence), with the figures being 5% and 3% respectively for current prevalence (ie: in the last twelve months) (Khalifeh et al 2015) [63].

But individuals with serious mental illness have a higher risk of IPV, Khalifeh et al (2015) found in their British study. The researchers analysed data from the 2010-11 British Crime Survey (BCS), which covered over 23 000 individuals in this UK government-funded national representative survey. The BCS concentrated on IPV in the past year (table 7.1), and used the concept "chronic mental illness" (CMI) to cover "'any long-standing mental health condition, such as depression', which has lasted for twelve months or more and which limits day-to-day activities" (Khalifeh et al 2015 p208).

Overall, 692 respondents self-reported CMI, and they were compared to 12 309 respondents who had no CMI. Significantly more individuals with CMI reported any IPV than the comparison group (16% vs 5%), and women with CMI experienced significantly more than men with CMI (21% vs 10%) (figure 7.1). In terms of odds ratios, women with CMI were 3.5 times more likely to have experienced any IPV in the past year than the comparison group and men with CMI twice as likely.

Experiencing IPV also led to health problems, physical injury and illness, mental and emotional problems, and suicide attempts for all victims, but especially victims with CMI for mental and emotional problems, and suicide attempts (eg: five times more

[63] The range in other studies around the world varies between 15-71% (Devries et al 2013).

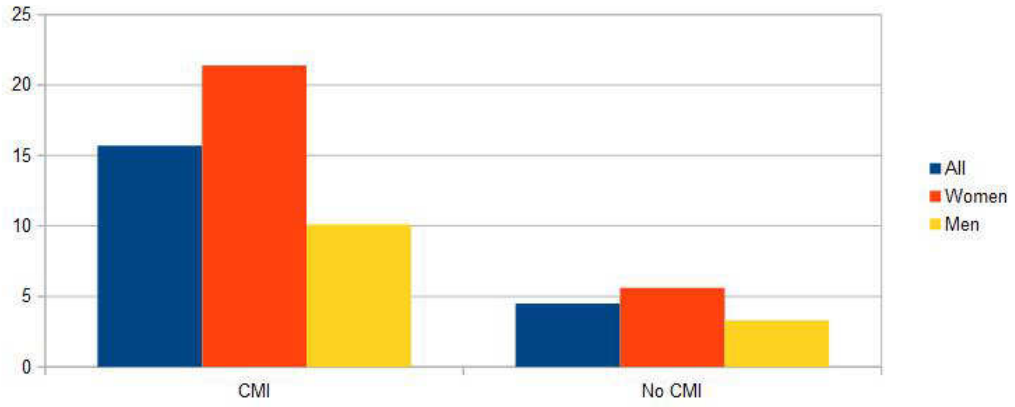
1 Emotional abuse: partner did any of the following
- Prevented them from having fair share of money
- Stopped them from seeing friends or relatives
- Repeatedly belittled them so they felt worthless
- Threatened to hurt them or someone close to them

2 Physical abuse: partner did any of the following
- Pushed them, held them down or slapped them
- Kicked, bit or hit them, or threw something at them
- Choked or tried to strangle them
- Threatened them with a weapon or threatened to kill them
- Used some other kind of force against them

3 Sexual abuse: partner did any of the following in a way that caused fear, alarm or distress
- Indecently exposed themselves to them
- Touched them sexually when they did not want it (eg: groping, touching of breasts or bottom, unwanted kissing)
- Sexually threatened them (e.g. demanded sex when they did not want it or followed or cornered them in a sexually threatening way)
- Forced them to have sexual intercourse, or to take part in some other sexual act, when they made it clear that they did not agree or when they were not capable of consent

(Source: Khalifeh et al 2015 p208).

Table 7.1 - Definition of IPV used by BCS.



(Data from Khalifeh et al 2015 table 2 p210)

Figure 7.1 - Standardised prevalence of IPV in past year based on CMI.

likely than individuals without CMI and IPV) [64].

[64] A common way of measuring suicide attempts in studies like this is the Suicide Intent Scale (Beck et al 1974). Individuals score themselves on twenty items about their attempt - eg: Active preparation for attempt: none (0), minimal to moderate (1), extensive (2); Suicide note: absence of note (0), note written but torn up, or thought about (1), presence of note (2) (Redfield 1999).

Table 7.2 lists the main strengths ans weaknesses of the study by Khalifeh et al (2015).

Strengths:

- Large and nationally representative sample.
-
- Clearly defined concepts.
- Specially designed measure of IPV used in British Crime Surveys, including self-completed (vs interviewer-completed), and questions about specific acts of IPV (vs general terms like "domestic violence").
- Controlled for socio-demographic confounders (eg: gender).

Weaknesses:

- Self-reported data from volunteers.
- IPV measure not validated against other psychometric measures.
- No data on context, nature and frequency of IPV "which would allow a distinction between isolated acts of violence versus prolonged, severe and controlling violence" (Khalifeh et al 2015 p211).
- No details about CMI.
- "It is possible that people with and without mental illness had differential recall or reporting of IPV, although there is some evidence that self-reported victimisation among people with mental illness is reliable" (Khalifeh et al 2015 p211).
- Unable to establish direction of causality: "In this study, the definitions of 'CMI' (duration more than 1 year) and 'recent IPV' (within the past year) mean that mental illness would have preceded 'recent IPV', except where there was measurement error due to reporting or recall bias. Nonetheless, the onset of IPV and CMI was not measured, and some participants may have experienced IPV before the onset of their mental illness (where IPV may have causally contributed to their CMI). The association found in this study between CMI and IPV could plausibly explained by two pathways: mental illness could be a risk factor for IPV and/or historical IPV could be a risk factor for both CMI and recent IPV. Both of these pathways are supported by a recent systematic review of longitudinal studies, which found evidence for a bidirectional causal relationship between common mental disorders and IPV" (Khalifeh et al 2015 p211).

Table 7.2 - Strengths and weaknesses of Khalifeh et al(2015).

7.2. ESTABLISHING CAUSALITY

Devries et al (2013) stated that "IPV experience is strongly and consistently associated with depression, including depressive symptoms and depressive disorders, and suicide in cross-sectional studies of women in both high- and lower-income settings. There is less research

on men, but cross-sectional studies also show that depressive symptoms are associated with IPV experience".

Cross-sectional studies cannot establish causality, and there are three possible ways that IPV and depression may be associated (Devries et al 2013):

1. IPV experienced causes depression (via traumatic stress from IPV, for example).

2. Depression causes IPV (eg: depression preceded the first incidence of dating violence in US teens; Lehrer et al 2006).

3. Both IPV and depression are caused by common risk factors (eg: early life experiences).

Devries et al (2013) performed a systematic review and meta-analysis to establish which of these associations is evidence-based. Sixteen longitudinal studies up to February 2013 met the inclusion criteria (with the majority from the USA).

IPV was self-reported for specific categories of violence, and depression was measured mostly self-reported scales rather than interviewer diagnosis.

It was found that the experience of IPV increased the likelihood of depressive symptoms and suicide attempts by women, and that depression increased the risk of IPV. There were few studies with men. The authors admitted: "our ability to draw firm conclusions is limited by the quality of the available studies, in particular the lack of adjustment for common risk factors... The different scales of measurement (binary or continuous) employed across various studies meant that we were unable to combine all measures of effect, which limited the number of studies in our meta-analyses. However, studies that we could not include in meta-analyses showed a positive direction of effect consistent with that of the studies included in the meta-analyses. Too few studies met the inclusion criteria to meaningfully assess publication bias" (Devries et al 2013).

Other limitations included "lack of comprehensive control of potential confounders" (eg: alcohol use), the measurement of depressive symptoms (self-reports) rather than diagnosis of a depressive disorder, emotional violence not covered, all but two studies in high-income countries, and exposure to IPV was coded as present or absent (as opposed to amount or regularity violence) (Devries et al 2013).

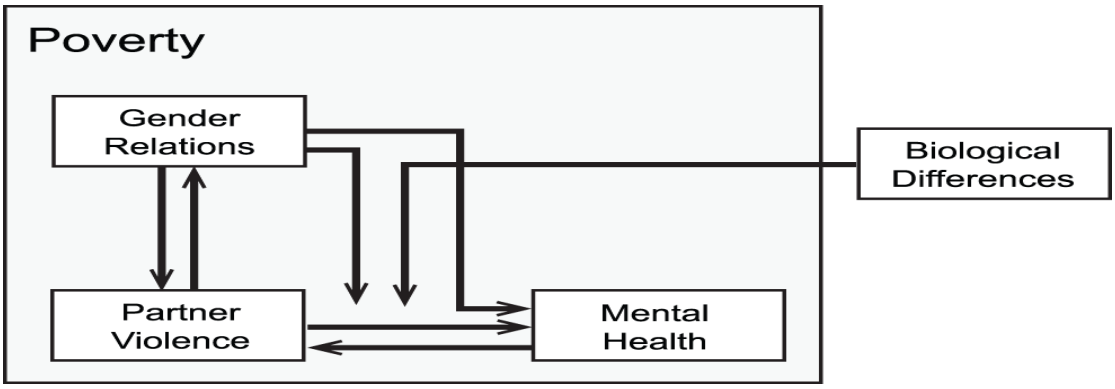
Devries et al (2013) summed up: "it seems that the relationship between IPV and depression is bidirectional, with women who are exposed to IPV being at increased risk

of depression symptoms, and women ho report depressive symptoms being more likely to subsequently experience IPV".

Tsai (2013) evaluated the Devries et al (2013) study in the following way:

- Longitudinal studies allow examination of "temporal relationship" between IPV and depression.
- "Although the literature contained few studies examining the impacts of violence on incident depressive disorders, the authors identified enough studies to support a conclusion that violence was associated with incident symptoms of depression".
- Lack of data about relationship between IPV and depression among men.
- An unmeasured confounding variables like child abuse "could be causally related to both irrespective of their temporal relationship with each other, inducing a spurious association that could be mistakenly interpreted as bidirectional". This could be partially overcome by measuring baseline depressive symptom severity.

Tsai (2013) emphasised the role of gender: "The extent to which exposure to violence results in poor mental health outcomes is modified by gendered norms governing how men and women safely negotiate situations of potential danger as well as by biological differences in physical strength. And finally, poverty offers a hospitable environment for gender-unequal norms, violence against women, and psychological distress to thrive" (figure 7.2).



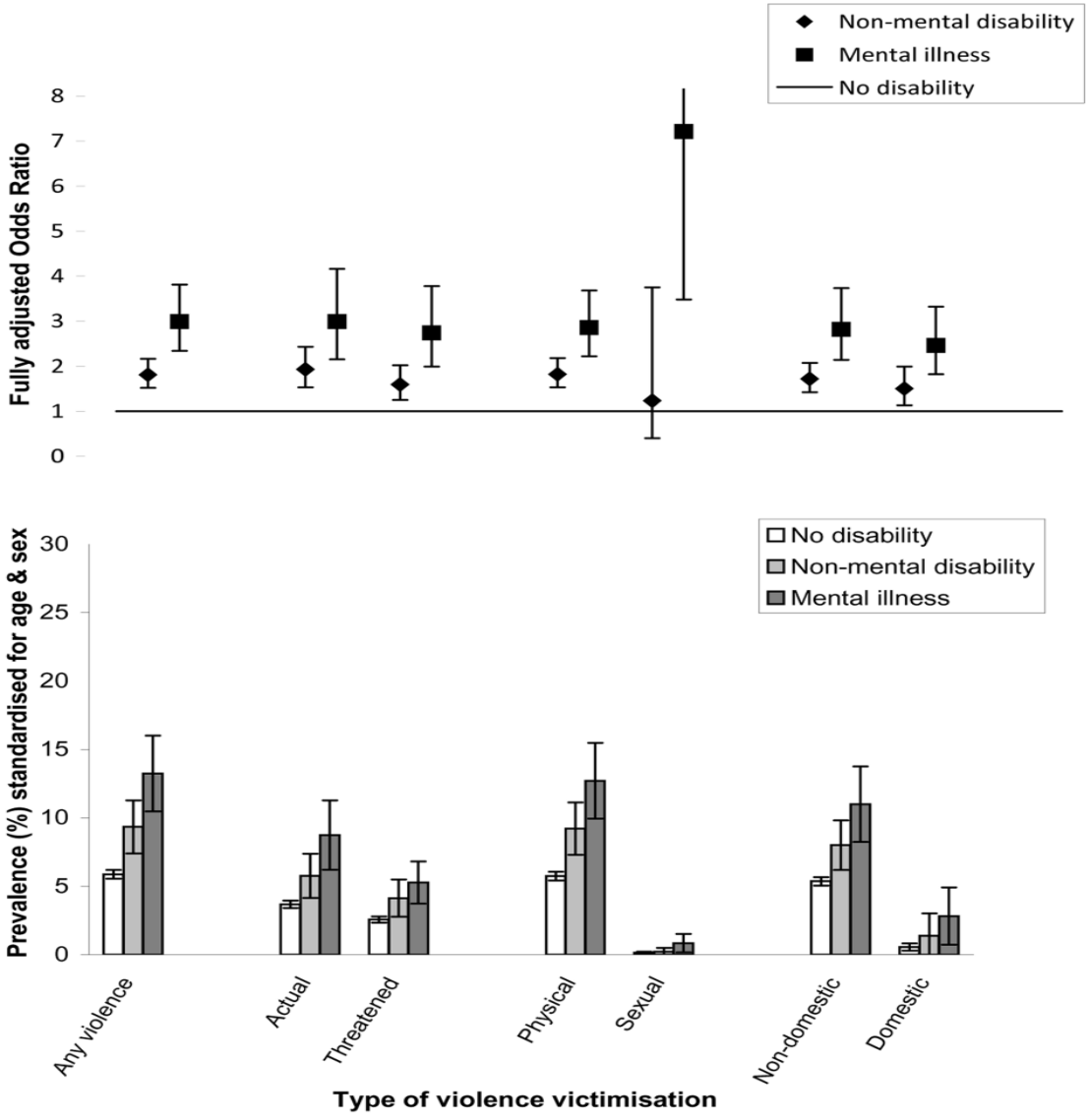
(Source: Tsai 2013 figure 1)

Figure 7.2 - Model of connections between IPV and mental health taking account of gender.

7.3. GENERAL VIOLENCE

More widely, violence was experienced by about one-quarter of individuals with mental illness in the past year, which was about four times more than individuals without mental illness (Hughes et al 2012).

Khalifeh et al (2013) used the data from the 2009-10 BCS, which included a measure of "disability" for the first time. Just over 16% of 44 000 respondents reported a "disability". The risk of being a victim of actual or threatened violence in the past year was two and a half times greater with a mental illness or long-term physical illness, and twice as likely with a mobility problem as compared to individuals without a disability (figure 7.3).



(Source: Khalifeh et al 2013 figure 1)

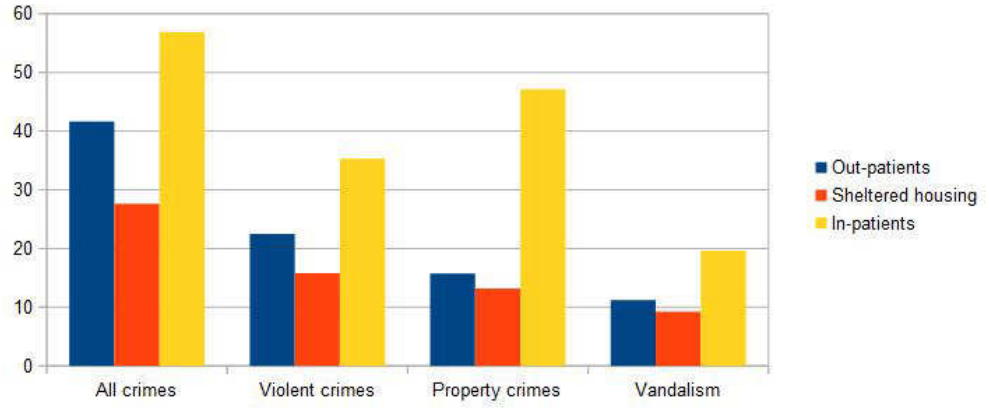
Figure 7.3 - Prevalence and odds of violence by sub-types for respondents aged sixteen years and above.

7.4. OVERALL CRIME VICTIMISATION

Individuals with severe mental illness (SMI) are more likely to be victims of crime than the general population [65]. For example, eight times more likely for violent crime (Kamperman et al 2014; appendix 7A), and over three times for non-violent crime in the last year (Teplin et al 2005).

de Mooij et al (2015) found that the type of care experienced by individuals with SMI was important in a study that compared out-patients, in-patients, and those in sheltered housing in the Netherlands. Data for 2011 were used from a self-report measure of different crimes categorised into violent crimes, property crimes, and vandalism [66]. Two hundred and sixteen individuals aged 27-76 years old with SMI (defined as schizophrenia, psychotic disorder, substance use disorder, or severe mood or anxiety disorder) were compared to 10 865 residents of Amsterdam and the surrounding area.

In-patients experienced significantly more crime victimisation overall than the other two groups of individuals with SMI (figure 7.4). Individuals with SMI were over twice as likely to be victims of violent crime than the general population (figure 7.5), though overall victimisation rates were similar.



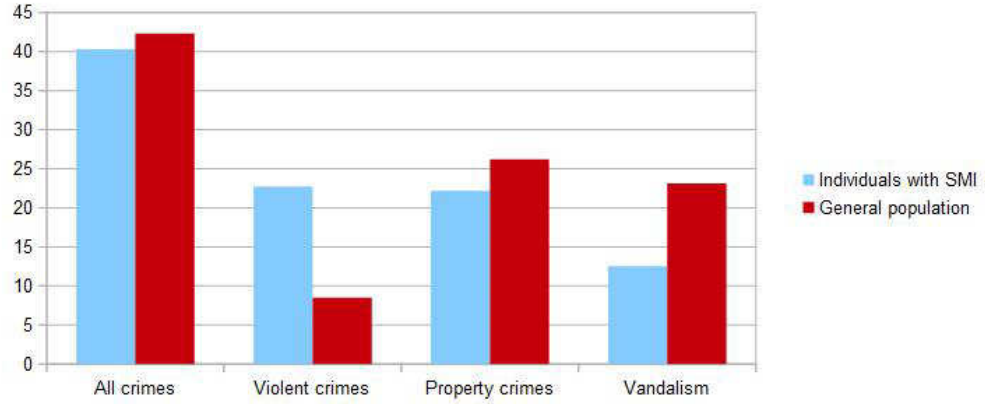
(Data from de Mooij et al 2015 table 2 p518)

Figure 7.4 - Prevalence of crime victimisation (%) among individuals with SMI.

Younger individuals with SMI were most likely to be victims. "This can be explained by the fact that

[65] There is also fear of crime (appendix 7B).

[66] Violent crimes = sexual crimes, threats and assaults. Property crimes = burglary, theft of a bike, theft of a car, pickpocketing, theft of other property). Vandalism = vandalisation in general and of a car.



(Data from de Mooij et al 2015 table 2 p518)

Figure 7.5 - Prevalence of crime victimisation (%) among individuals with SMI and the general population.

younger people are more active in their daily lives than older people and therefore more exposed to dangerous situations. Another possible explanation could be that younger patients may have fewer skills to cope with, or to avoid involvement in, a criminal event than older people, who have more like experience with victimisation" (de Mooij et al 2015 p520).

Disorganisation in thinking and behaviour was another risk factor for individuals with SMI, but not being female (as in previous studies).

There were a number of methodological issues, and these may explain the difference in findings to previous studies:

1. Cross-sectional data collected at one point in time. This meant that causality could not be established.

2. Certain predictors of victimisation were not included - eg: previous victimisation.

3. The levels of property crime may have varied between patients and the general population because property ownership was lower in the former group (eg: ownership of car: 6% vs 75% of general population; de Mooij et al 2015).

4. No details of mental health problems of comparison group.

5. No details of victimisation among homeless individuals with SMI (as homelessness is known to be a key risk factor for victimisation).

6. No inclusion of individuals with SMI younger than 27 years old.

7. Individuals with SMI needed to have the previous two years continuous intensive mental healthcare for inclusion.

8. Study based in Amsterdam area.

9. A self-report measure of victimisation used by trained interviewers, though it was a standardised measure used in Dutch crime surveys.

10. Certain crimes not included - eg: "white-collar crimes".

7.5. APPENDIX 7A - KAMPERMAN ET AL (2014)

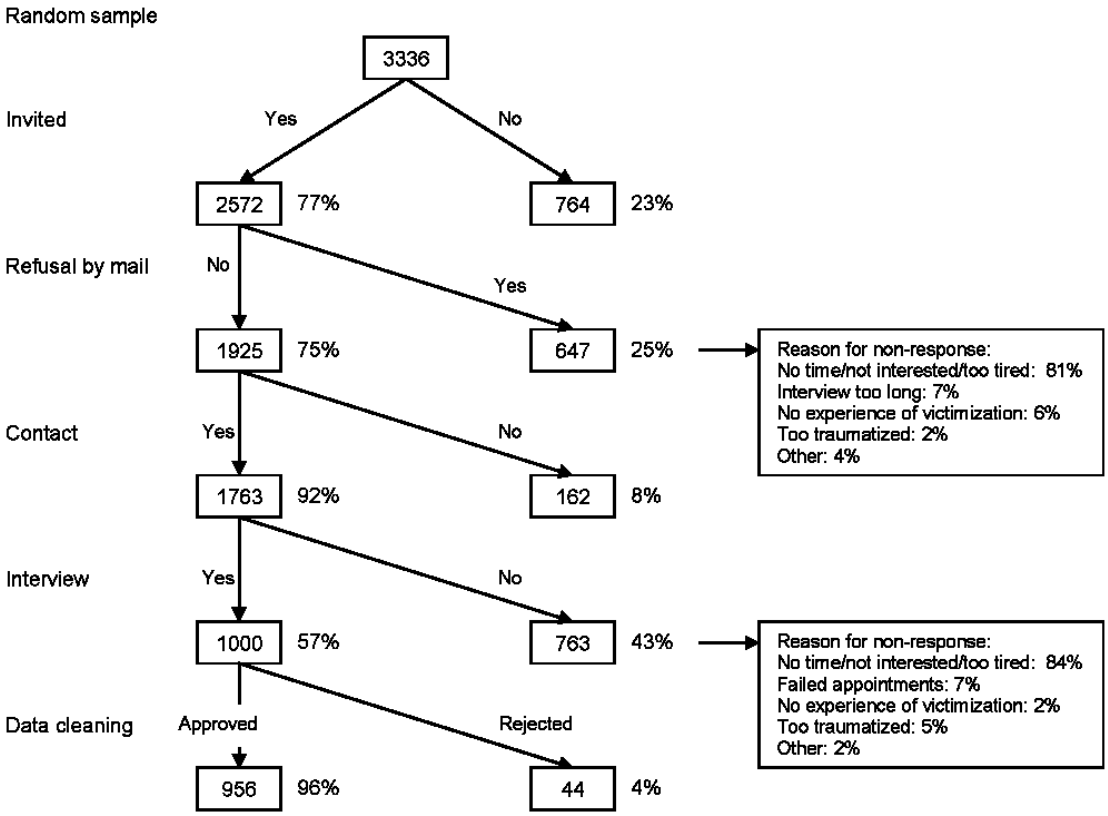
Nine hundred and fifty-six psychiatric out-patients aged 18 to 65 years in the Netherlands were interviewed about their experience of crime victimisation in the last year.

The sample was made up of volunteers who had responded to a letter sent to 3 336 randomly chosen out-patients with chronic [67] psychotic, bipolar, or major depressive disorder at one mental healthcare institution. Figure 7.6 shows the stages of selection of this sample. A comparison sample of 38 227 Dutch residents were used.

The annual prevalence of crime victimisation was 46.5% in the out-patient sample compared to 32% in the general population (figure 7.7).

Out-patients were more likely to experience polyvictimisation (ie: more than one incident of victimisation of the same offence in the previous year) (figure 7.8). Female out-patients had a higher risk of crimes like assault and sexual harassment than males.

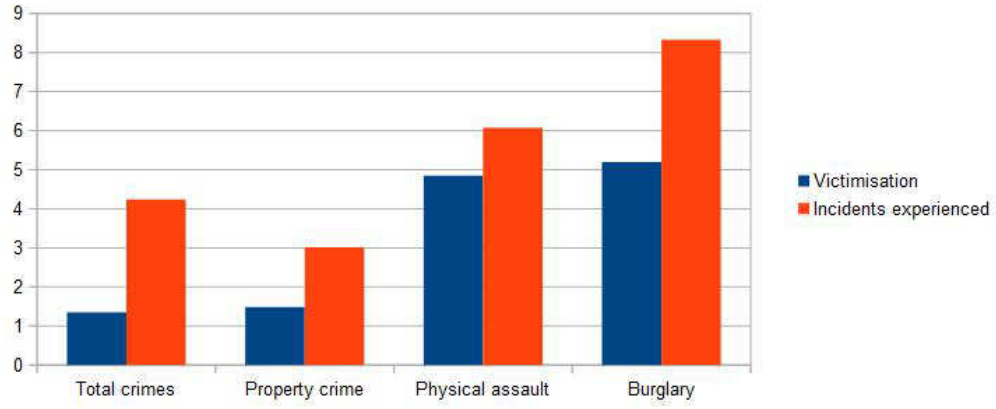
[67] For at least years, and using DSM-IV criteria.



(Invited = eligible; contact = agreed to participate by letter, but could not be contacted by telephone; interview = agreed on telephone to be interviewed, but no interview took place)

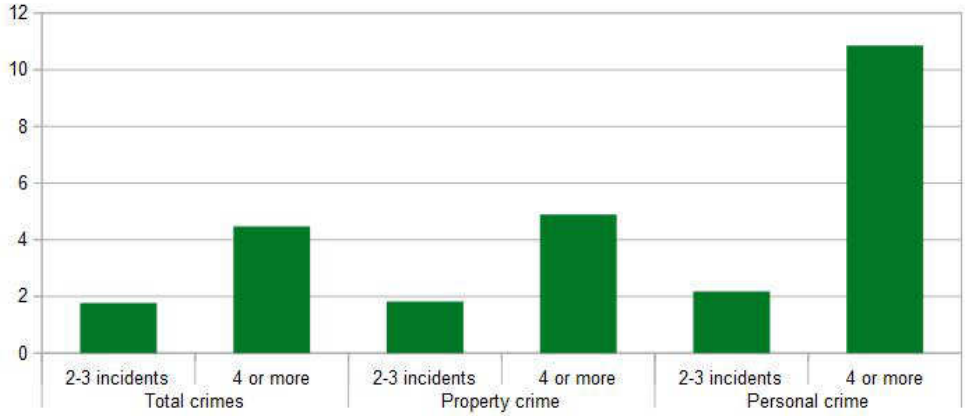
(Source: Kamperman et al 2014 figure 1)

Figure 7.6 - Flow-chart showing recruitment process.



(Data from Kamperman et al 2014 tables 2 and 6)

Figure 7.7 - Relative risk of being a victim and experiencing incidents of selected crimes for out-patients compared to the general population (where 1 = risk for general population).



(Data from Kamperman et al 2014 table 3)

Figure 7.8 - Relative risk of polyvictimisation for out-patients compared to the general population (where 1 = risk for general population).

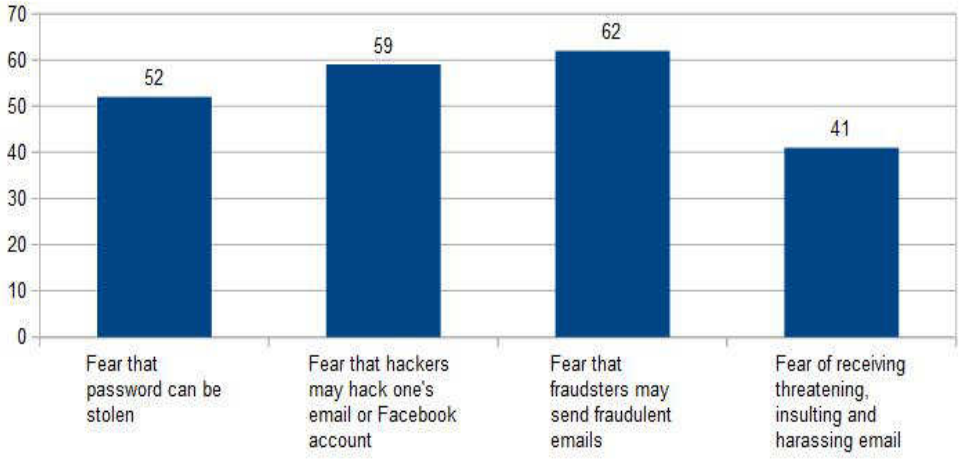
7.6. APPENDIX 7B - FEAR OF BEING A VICTIM

"Criminal victimisation is a horrible experience. It creates fear in the minds of the primary victims as well as secondary victims and third-parties like the victims' significant and generalised others. The net effect is a wide spread feeling of insecurity among these people, culminating into some personal efforts to protect themselves from criminal victimisation or repeat victimisation" (Rada and Ndubueza 2013 p36).

Weis and Milakovich (1974) observed that the "fear of crime, reports about it, and the political misuse of that fear is a problem as serious as crime itself" (quoted in Rada and Ndubueze 2013).

In the past, "university campuses were seen as sanctuaries, fortified against crime of the larger society" (Rada and Ndubueze 2013). Since the 1980s this has been challenged with high profile cases on university campuses, and subsequently with researchers' interest in students' fear of crime. This is more so with the growth of the Internet.

Rada and Ndubueze (2013) sampled over six hundred students at two universities in Nigeria with a questionnaire that included questions about the fear of on-line victimisation. Over half the respondents were afraid that their passwords could be stolen, or that their email or Facebook account might be hacked, or that they would receive threatening, insulting or harassing emails (figure 7.9).



(Data from Rada and Ndubueze 2013 table 3 p40 and table 4 p41)

Figure 7.9 - Percentage of respondents reporting fear of on-line victimisation.

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8. SOCIAL CONTEXT

The social context and dominant discourses in society are a background to potential mental health issues, but there are often ignored and downplayed because of the emphasis on the individual.

Carney et al (2014) distinguished three characteristics of the "individualisation of society under capitalist regimes":

i) "Risk society" (Beck 1992) - material wealth but insecurity (eg: job).

ii) "Winner-take-all-society" (Hacker and Pierson 2010) - high levels of income equality in societies with political equality [68] [69].

iii) Encouraging consumerism via spending on credit (Rajan 2010) "to perpetuate the myth that capitalism makes everyone rich" (Carney et al 2014).

Neoliberalism is another term described modern Western societies. It aims to "down-size the state and open up public services to market competition" (Corbett and Walker 2013 quoted in Ishkanian 2014) [70].

Using the ideas of Michel Foucault, Cromby and Willis (2014) pointed out that "rather than govern by dictating rights and responsibilities, neoliberalism proceeds by harnessing desires for independence and creativity to the interests of business, reconfiguring the social relations of capitalism to emphasise competition, not between workers and capitalists, but

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[68] Wacquant (2009) outlined "'strategies to treat the conditions and conducts that [contemporary societies] deem undesirable, offensive, or threatening': socialisation, medicalisation and penalisation" (Baillergeau 2014 pp355-356).

[69] Palmer (2015) offered two warning signs for the financial industry:
i) The sudden rapid growth of a new financial technique or product.
ii) The assumption of safety.

[70] A commodity is a key aspect of this system, and it is any "thing" that can be bought or sold. The process of commodification requires "a commodity to be objectified and isolable, which means that it must be reduced or fragmented" (Calvert 2008 p384). Callon (1998) used the term "disentanglement" to describe how commodities are "extracted from the network of relationships in which they are already embedded" - "an apparently stable object" (Calvert 2008). This has implications for biotechnology and biology when "the economic pressures to commodify may prove irresistible" (Calvert 2008).

"Commodities are easily and frequently commuted into personal possessions which excite commitment, investment, and meaning" (Warde 2015 p121), and "we learn, define, and remind ourselves of who we are by our possessions" (Belk 1988).

Cohen (2000) distinguished between citizen-consumers and customer-consumers, with the former being "consumers who take on the political responsibility we usually associate with citizens to consider the general good of the nation through their consumption, and the latter being consumers who seek primarily to maximise their personal economic interests in the market place" (quoted in Warde 2015).

between workers themselves" (p243).

Furthermore: "Foucault's (2008) analysis of neoliberalism highlighted the way in which it represents a reconfiguration of human nature and the social order in accord with the dictates and demands of the market, and that in so doing it implied a new kind of subject. It is in this sense a particular instance of governmentality, ie: 'a particular mentality, a particular manner of governing, that is actualised in habits, perceptions and subjectivity' (Read 2009 p34). Neoliberalism, then, implies a technology of the self (Foucault 1998). a mode of subjectification, in the double sense of both being subjected to a power relation and, in so doing, working to reflexively understand one's self as a particular kind of subject" (Cromby and Willis 2014 p251).

In relation to the voluntary sector, the upshot is that organisations with specialist knowledge, in say domestic violence, are replaced by generic organisations that win the government contracts to provide services (and then sub-contract) [71]. One of Ishkanian's (2014) interviewees from a women's voluntary organisation in 2013 described it thus: "In the past, domestic violence services were informed and shaped by feminist thinking. Now, by placing value on gender neutrality and thinking that 'anybody can deliver', the policy landscape has been completely altered. It's becoming more corporate and you have the big players, the generic providers who may have a social purpose but who operate like a business. In the area of refuges, there are massive housing associations that don’t have that ethos, history or expertise of working on domestic violence, but they are very good at winning contracts. These organisations become the gatekeepers who win the big contracts and then sub-contract to smaller organisations. Smaller organisations, which have more expertise, but less experience winning contracts are forced into sub-contracting. What happens is that they have less autonomy, but if they didn't sub-contract they'd be out of it entirely" (pp342-343).

Dependence on such funding was described by another interviewee as "walking on a thin double edged sword. We can't be too vocal in our criticism of policies because we rely on statutory funding, so we have to operate with tact and diplomacy" (Ishkanian 2014 p344).

Exploring experiences of austerity after 2008 in the Republic of Ireland with 100 interviewees, Carney et al (2014) outlined four different responses based on age and socio-economic status (SES):

[71] Neville (2014) noted how "an individualistic and depoliticised construction of volunteering" placed the focus on the individual and ignored the "dismantling of the welfare state that is currently occurring under the guise of austerity".

a) "Surviving day-to-day" - young, low SES adults suffer most directly from the austerity programme of the Government.

b) "Planning to emigrate" - lower and mid-SES young adults had this as a back-up plan if circumstances became intolerable, and it follows historical precedents [72].

c) "Using private resources to support family" - high SES adults have this option for themselves and their children.

d) "Viewing present as favourable to past" - oldest, low SES adults focused on the general improvements since their childhood.

TEENAGE PREGNANCY

There is a dominant discourse in the UK which constructs "teenage pregnancy and motherhood as a mistake and a cause of social exclusion" (Rudoe 2014) [73]. "Teenage pregnancy" is also used to refer to working-class women.

Rudoe (2014) interviewed sixteen pregnant young women and mothers (aged 16-20 years old), and found that they "did not by any means embrace the idea of teenage pregnancy and motherhood being a 'mistake' or a 'catastrophe', but instead reflected on the issue from a much broader and more varied perspective" (p308).

For example, "Claudia" observed: "I don't think it's ever a mistake to have a baby... but I would tell young girls to wait a while, live a bit, before you have your child, because it does change your whole life. But like some people it changes them in a good way, like there's kids, like young girls I know, who's been like really street, like getting arrested, whatever, parties, then they had their daughter and now they're just, like, a homely person, so in a way it is good – there's good

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[72] Pande (2014) described migrant domestic workers who work "illegally" as "global exiles" because of "their displacement and permanent statelessness". In particular, individuals from Africa and Asia working illegally in Lebanon who cannot return home, and those from the Philippines who are banned by their government from working in Lebanon but do. In focus groups, they described themselves as "prisoners" in the country, and the consequent effect on their mental health.

[73] Speaking specifically about homelessness, but applicable to other aspects of social welfare, Dwyer et al (2015) said: "Scanlon and Adlam (2008, 2011) refer to as an 'essentially stubborn and dangerous societal refusal' (2011 p131) to face up to the complex social causes underpinning the problems of many marginalised people and the ways in which this 'systematic refusal' routinely perpetrates the problems that policymakers and service providers are ostensibly trying to alleviate. Within the wider context of public expenditure cuts, systemic welfare retrenchment and the promotion of a populist 'politics of resentment' by the UK Coalition government (Hoggett et al 2013), it should perhaps not be too surprising to find that those whose homelessness is judged to be due to their 'irresponsible' and/or anti-social behaviour routinely find their applications rejected and themselves often, quite literally, left out in the cold" (p5)

changes, there's bad changes. If it's the right time I think you should do it, but if not then, you know, just wait" (p298).

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9. MAJOR DEPRESSIVE DISORDER AND ASSISTED DYING

9.1. ASSISTED DYING

Schuklenk and van de Vathorst (2015) argued that assisted dying (AD) should be available to individuals with treatment-resistant major depressive disorder (TRD - treatment-resistant depression) [74]. But this view varies with public opinion. For example, three-quarters of those surveyed in a Canadian city were against AD in such a psychiatric condition (Schuklenk et al 2011).

While in a Dutch study using a case study of a middle-aged woman with recurrent TRD, 52% disagreed with euthanasia, 28% agreed, and the remainder were uncertain (Van Delden et al 2011 quoted in Schuklenk and van de Vathorst 2015).

Bolt et al (2015) surveyed over 1400 physicians in the Netherlands about their experiences and attitudes towards AD for psychiatric illness. Around one-third found it conceivable and the remainder inconceivable "they would perform EAS" (euthanasia and physician-assisted suicide), whereas the figure of conceivability was over 80% for a physical illness.

Gauthier et al (2015) found that fourteen of 611 cases of "suicide tourists" in Switzerland were categorised as mentally ill (excluding dementia). These are non-Swiss residents who visited the country between 2008 and 2012 for AD.

Assisted suicide is available in the Netherlands for TRD under a set of particular criteria, including an explicit, voluntary, and well-considered request [75], where the "suffering is unbearable and there is no prospect of improvement", and there are no "reasonable alternatives" (Schuklenk and van de Vathorst 2015).

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[74] It is estimated that up to one-third of individuals with depression have a treatment-resistant version (ie: "failure to achieve remissions despite adequate treatment"; Olchanski et al 2013) (Schuklenk and van de Vathorst 2015). Broome and de Cates (2015) were worried that there was no universal definition of TRD, and that remission varies between studies. For example, some studies see the that as "full recovery", while others use a reduction in the Hamilton Rating Scale of Depression.

[75] Cowley (2015) raised concerns over the individual's competence to choose. den Hartough (2015) stated: "Death wishes are one common expression of this mood disturbance. That is one reason to doubt whether the patient's decision can be considered sufficiently competent to require to be respected (Or even as 'voluntary' enough...)" (p588).

Table 9.1 list some arguments against AD for TRD and Schuklenk and van de Vathorst's (2015) counter-arguments.

ARGUMENTS AGAINST	COUNTER-ARGUMENTS
Successful treatments might be developed in the future (appendix).	But how long to wait, if ever.
TRD is not diagnosed with absolute certainty by psychiatrists.	Schuklenk and van de Vathorst (2015) countered that "patients should not reasonably be expected to continue lives they do not consider worth living while psychiatry treatments' nirvana remains elusive" (p581).
Put more money in care rather than AD.	In reality, financial restraints limit this.
Offering AD reinforces loss of hope.	"In some cases, however, hope reaches its limits and may become illusory" (Schuklenk and van de Vathorst 2015 p582).

Table 9.1 - Arguments for and against AD for TRD.

Sagan (2015) noted the unforeseen consequences if individuals with TRD try to commit suicide, including the train driver who sees the person jump out in front of them, or neighbours in the vicinity of a gassing that results in an explosion.

9.2. TREATMENT FOR DEPRESSION

Poor outcomes of treatment for depression is linked to poor treatment adherence, high drop-out, unnecessary and/or frequent changes, and inconsistent treatment strategies (Guo et al 2015).

Many of these issues can be dealt with using measurement-based care (MBC) (Trivedi et al 2006), which involves individual treatment for patients based on severity of symptoms and tolerance of medication, and includes patient self-reports.

Guo et al (2015) performed a randomised clinical trial of MBC for 120 individuals with depression in Beijing, China. All participants had a DSM-IV diagnosis of Major Depressive Disorder (MDD), and a score of seventeen or above on the Chinese version of the Hamilton Depression Rating Scale (HAM-D). The MBC group received an anti-depressant for 24 weeks based on a personalised schedule that varied dosage depending on response and side effects, while the standard care (control) group had a standard dosage throughout. The outcomes measures at

the end of the trial were based on changes in HAM-D - a score of seven or less defined as "remission", and 50% or more decline in baseline score was "response".

Significantly more participants in the MBC than control group showed a response ($p = 0.002$) and remission ($p<0.001$) (figure 9.1). Note that the dosage of AD was higher in the MBC group throughout.

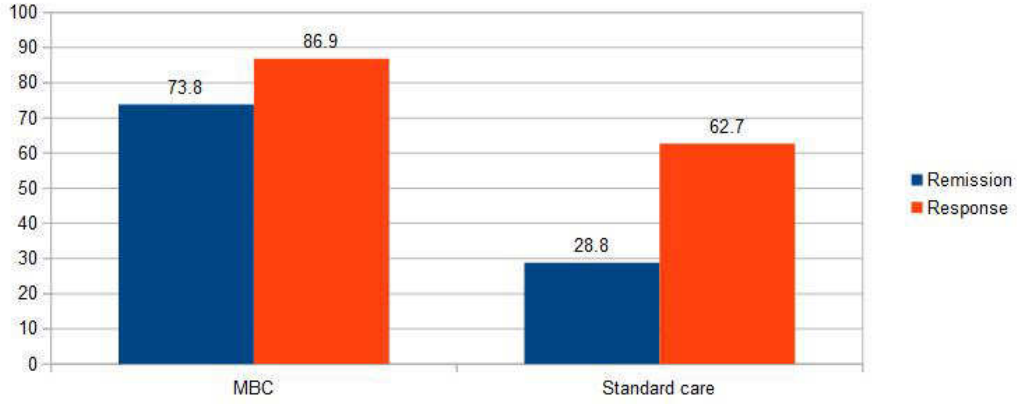

Figure 9.1 - Percentage of participants showing remission and response at 24 weeks.

The researchers recognised nine limitations to their study:

i) It was an open-label study - ie: it was known to the patients and doctors which AD was being given and how much.

ii) Only two ADs were offered - paroxetine and mirtazapine.

iii) Clinical research co-ordinators monitored the MBC group to see that the trial's protocols were not violated, but not the control group.

iv) The study took place at one hospital in China.

v) The reasons for treatment discontinuation by participants were not assessed.

vi) The number of appointments with a doctor was controlled in the MBC group, but not in the control group.

vii) Pill counts, as a measure of treatment adherence, were performed in both groups, which could have increased adherence in the control group.

viii) There was no structured interview to diagnose MDD, only the use of a symptom checklist.

ix) The study was only 24 weeks long.

9.3. MINDFULNESS-BASED COGNITIVE THERAPY

Residual depressive symptoms and the risk of relapse exist for individuals who recover from episodes of MDD. But these risks decrease "if patients in partial or full remission can learn, first, to be more aware of negative thoughts and feelings at times of potential relapse/recurrence, and, second, to respond to those thoughts and feelings in ways that allow them to disengage from ruminative depressive processing" (Segal and Walsh 2016 pp7-8).

Mindfulness-based cognitive therapy (MBCT) can help here. It is a group-based therapy that combines the focus on the now (attentional training [76]) of mindfulness meditation, and the strategies to deal with negative thoughts in cognitive-behavioural therapy. "Patients are taught to become more aware of thoughts and feelings, and to relate to them in a wider 'decentered' perspective as 'mental events', rather than as aspects of the self or as accurate reflections of reality" (Segal and Walsh 2016 p8).

But is it effective in preventing relapse? Kuyken et al (2015) (table 9.2) found so in a large study. Over four hundred individuals with recurring depression received MBCT or maintenance-dose anti-depressants. At two years, there was no difference in rates of relapse between the two groups, which suggested that "MBCT provided relapse protection on par with maintenance anti-depressant pharmacotherapy" (Segal and Walsh 2016) [77].

In fact, MBCT was more effective for victims of childhood abuse in the study. Though this finding was from secondary analysis of the data, and thus tentative, "it is possible that in MBCT, patients learn how to approach and observe their painful memories rather than ruminating about them" (Segal and Walsh 2016 p8).

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[76] So, "attention is trained to shift away from cognitive habits that link external sensations to conceptual manipulation and elaboration. Instead, mindfulness teaches individuals to direct attention internally, to representations of the visceral components of emotion, as supported by a distinct neural pathway for interoception, the sensation of the body's internal state, including signals from the breath" (Segal and Walsh 2016 p10).

[77] Kuyken et al (2015) offered the following interpretation of their findings: "We found no evidence that MBCT-TS is superior to maintenance anti-depressant treatment for the prevention of depressive relapse. However, when considered in the context of the totality of randomised controlled data, we found evidence from this trial to support MBCT-TS as an alternative to maintenance anti-depressants for prevention of depressive relapse or recurrence at similar costs. It allows such individuals to stay well and maintain good quality of life" (p72).

- MBCT-TS (MBCT and taper support to reduce anti-depressants) vs maintenance-dose anti-depressants (control).
- Participants from four areas in south-west England.
- DSM-IV diagnosis of recurrent MDD in full or partial remission.
- Three or more episodes of MDD in adult lifetime.
- Eight weeks of MBCT.
- Assessment at six points - baseline, and 1, 9, 12, 18 and 24 months post-MBCT.
- Outcome measures - time to relapse, and number of depression-free days.
- 212 participants in each group began study, and 183 in each group completed.
- Relapse: 44% (MBCT) vs 47% (control).

Table 9.2 - Details of Kuyken et al (2015).

Compared to a placebo, MBCT reduces the risk of relapse by one-third, according to a systematic review and meta-analysis of six randomised controlled trials (Piet and Hougaard 2011).

Practising mindfulness meditation away from group therapy is important (Segal and Walsh 2016).

Dimidjian et al (2014) has developed an online version called Mindful Mood Balance (MMB) with eight self-administered sessions.

MBCT has been reported as effective with currently depressed individuals, and those who cannot tolerate anti-depressants, as well as other psychiatric conditions (eg: eating disorders), and in changing pain-related cognitions (eg: web-based "mindfulness in action") (Segal and Walsh 2016).

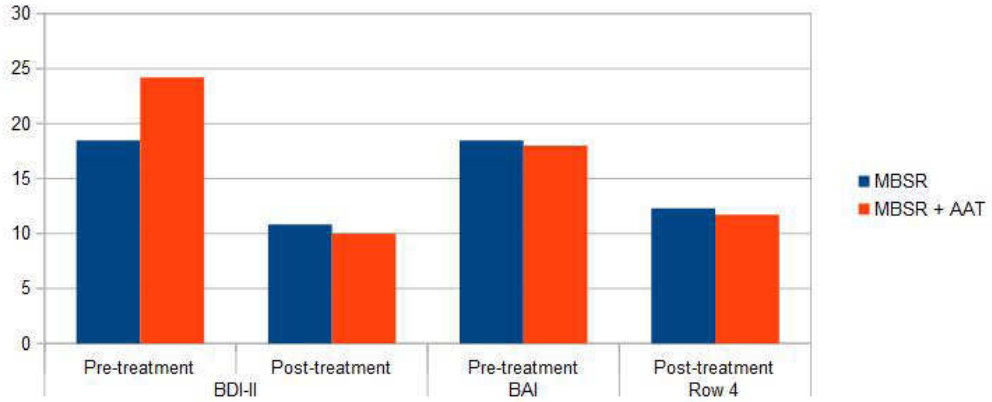
The next question is what about MBCT is effective. A number of studies have confirmed five aspects - mindfulness, reduced rumination, cognitive flexibility/decentering, cognitive reactivity, and self-compassion (Segal and Walsh 2016).

Henry and Crowley (2015) investigated mindfulness-based stress reduction (MBSR) combined with animal-assisted therapy (AAT). MBSR (Kabat-Zinn 1990) "teaches non-judgmental awareness of the present moment to alleviate psychological distress", while AAT is "a goal-directed intervention in which an animal that meets specific criteria is an integral part of the treatment process" (Henry and Crowley 2015 p386).

Eighty-two volunteers at a US university were recruited because they reported at least mild psychological distress, and had a positive attitude towards dogs. Only thirty-three were eligible to start the study, and 21 completed it. The participants were randomly assigned to MBSR or MBSR + AAT for six 50-minute sessions. In the latter group, participants could

interact with the dog as they wanted during the sessions.

Both groups showed significant reductions in anxiety and psychological distress from baseline to end of sessions (figure 9.2). So, "the addition of AAT did not significantly enhance or hinder the desired treatment outcomes; however, results suggest that AAT increased some aspects of client satisfaction, particularly perception of therapist efficacy, recommendation of the intervention, and future participation in similar interventions" (Henry and Crowley 2015 p397).



(Data from Henry and Crowley 2015 table 1 p392)

(BDI-II = Beck Depression Inventory-II (Beck et al 1996); BAI = Beck Anxiety Inventory (Beck and Steer 1990))

Figure 9.2 - Mean scores pre- and post-treatment.

The researchers noted that the "effectiveness of MBSR may have created a 'ceiling effect' with participants experiencing only mild distress. Thus, any potential benefits of AAT may not have been seen" (Henry and Crowley 2015 p397). On the other hand, the "dog was intentionally used as an icebreaker to provide a topic of discussion and build rapport. Also, participants responded to the dog's behaviours and appearance, laughing at the dog's profuse tail wagging or commenting on his 'smile'" (Henry and Crowley 2015 pp397-398).

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10. FOUR DIFFERENT AREAS OF STUDY OF SUICIDE

10.1. US MILITARY

Traditionally suicide rates in the US military have been lower than the general population, but they have increased in recent years (Reger et al 2015).

Accurate data collection can be a problem (Reger et al 2015):

- Department of Defense records only the cover the period of military service, and not after (separation from the US military).

- Department of Veterans Affairs only deals with veterans who seek help (approximately one-third of all veterans).

- The use of death certificates depends on the accuracy of information recorded by, say, funeral directors.

Ursano et al (2015) used official data in the form of Department of Defense Suicide Event Report records in their study to assess the high risk individuals. Between 2004 and 2009 [78], there were 9791 documented suicide attempters [79] among active US soldiers in the Army Study to Assess Risk and Resilience in Servicemembers (Army STARRS) [80].

The majority of cases were enlisted soldiers (98.6%), which converted into a suicide attempt rate of 377.0 per 100 000 person-years. Among officers, the figure was 27.9. This compares to 213.8 for men and 273.6 for women aged 18-34 years in the US population for non-fatal self-injury in the same time period (Ursano et al 2015) [81].

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[78] This period covered active duty in Afghanistan and Iraq.

[79] Diagnosis based on International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) categories covering self-inflicted poisoning or injury with suicidal intent (codes E950-958).

[80] It is a longitudinal, retrospective cohort study of 1.66 million soldiers. Details at http://www.armystarrs.org/.

[81] These data from the Centers for Disease Control and Prevention are based on US hospital emergency departments, and may be an underestimation (Ursano et al 2015).

Table 10.1 details the socio-demographic variables associated with a greater or lesser risk of a suicide attempt (figure 10.1). Many of these variables among enlisted soldiers are common to the general population from previous studies - namely, increased risk for women, younger individuals, Whites, those with lower educational qualifications, and individuals with previous mental health problems (Ursano et al 2015).

- Female: 2.4
- Entered Army at 25 years or older: 1.6 (vs entering before 21 years old: 0.7)
- Younger than 21 years old at time of attempt: 5.6 (vs older than 40 years old: 0.5)
- Did not complete high school: 2.0 (vs completed college: 0.6)
- Ethnic minority: 0.7
- Length of service - 1-2 years: 2.4 vs >10 years: 0.5
- Never deployed: 2.8; previously deployed: 2.6
- Previous mental health problems in last month: 18.2

(1 = average risk; >1 = higher than average risk; <1 = lower than average risk)

Table 10.1 - Odds ratios for suicide attempts among US enlisted soldiers.

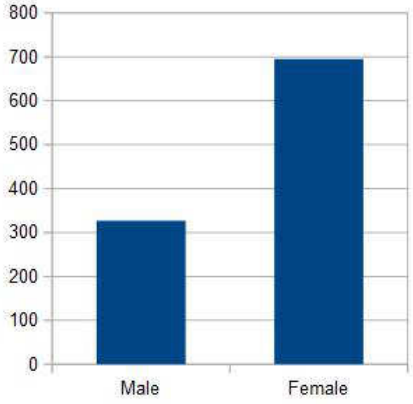
Overall, during the study period, other studies have found that suicide attempts and deaths in the US Army has risen, and relative to other branches of the US military (Ursano et al 2015).

Reger et al (2015) retrospectively analysed the data for all uniformed service personnel in the US military at any time between 7th October 2001 and 31st December 2007 [82] (n = 3 945 099). Data on mortality was collected for a further two years from the National Death Index.

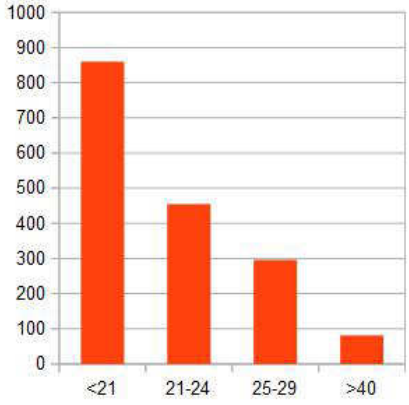
Of 31 962 deaths, 5041 were identified as suicide [83]. Higher rates of suicide were among younger personnel, males, not married, and White individuals (figure 10.2). Shorter length of service was also a risk factor (figure 10.3).

[82] This was the period covering "Operation Enduring Freedom" and "Operation Iraqi Freedom".

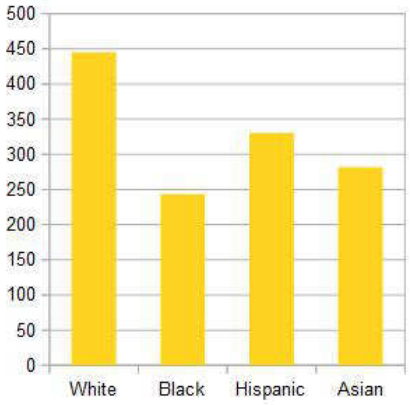
[83] Reger et al (2015) admitted: "It is possible that suicides were underestimated because some service members with suicidal intention may place themselves in harm's way" (p568).



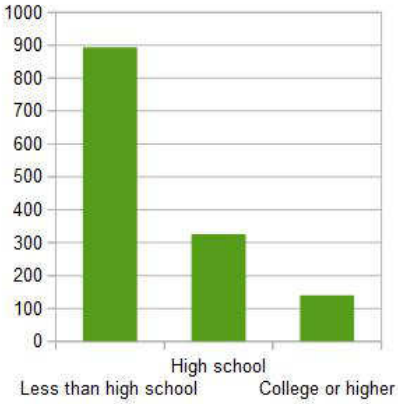
(a) Gender



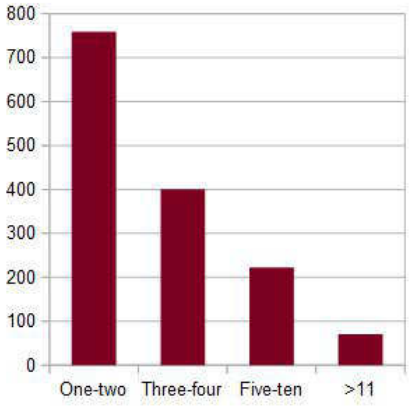
(b) Current age (years)



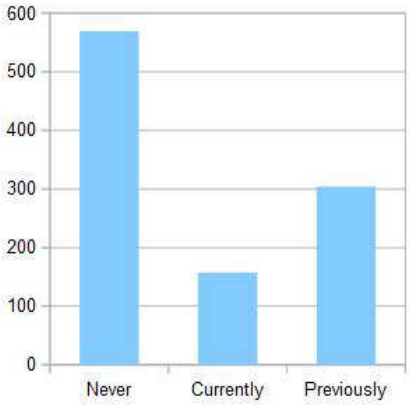
(c) Ethnicity



(d) Educational level



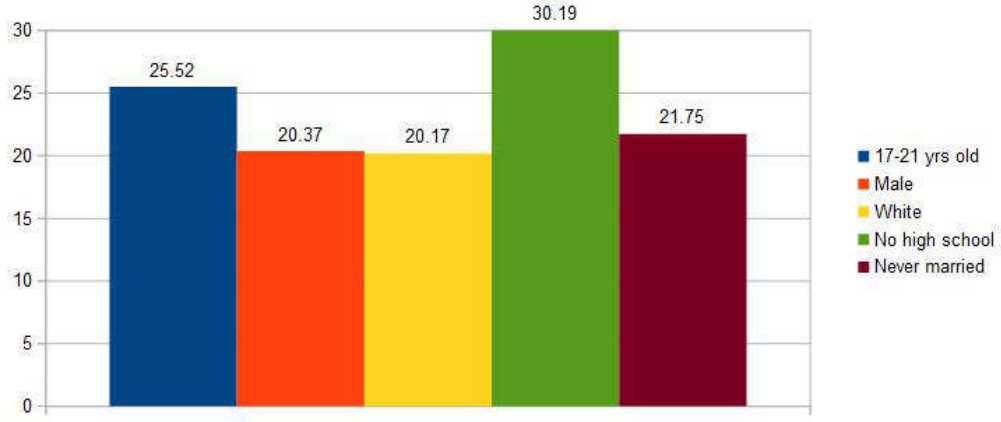
(e) Years of service



(f) Deployment status

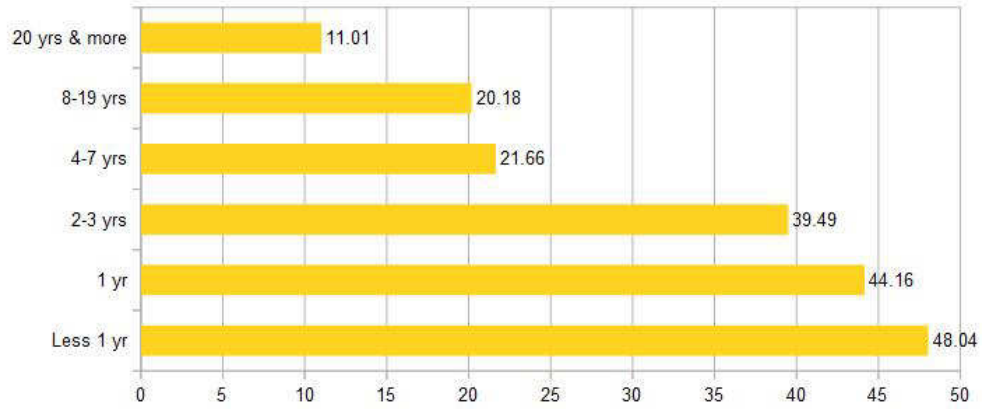
(Data from Ursano et al 2015 table 1 p920 and table 3 p922)

Figure 10.1 - Rates per 100 000 person-years among enlisted soldiers for selected socio-demographic variables.



(Data from Reger et al 2015 table 1 p564)

Figure 10.2 - Highest rates of suicide (per 100 000 person-years) based on selected socio-demographic variables.



(Data from Reger et al 2015 table 4 p567)

Figure 10.3 - Suicide rate (per 100 000 person-years) based on length of service.

The risk did not vary between deployed and non-deployed personnel (where deployment was defined as 30 days active), but it increased after separation from the military (figure 10.4).

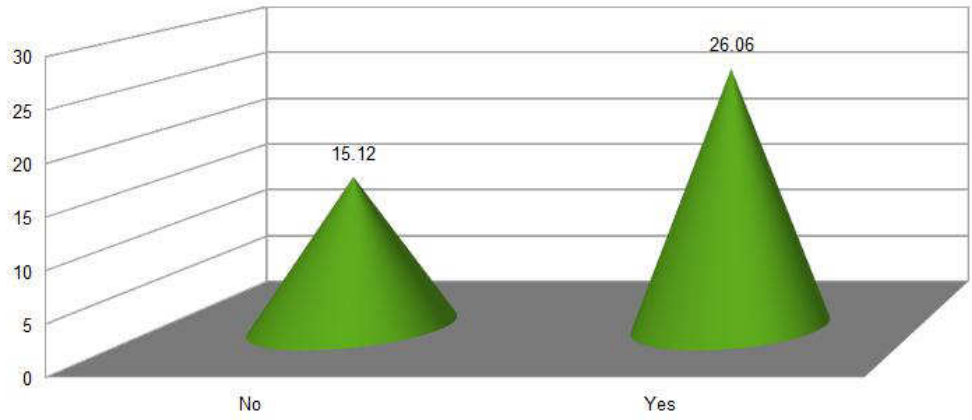
The possible reasons for this included (Reger et al 2015):

a) The transition to civilian life including loss of shared military identity, problems developing new social support, or "unexpected difficulties finding meaningful work may contribute to a sense that the individuals do not belong or are a burden to others" (Reger et al 2015

p567).

b) Individual factors that existed before military services.

c) Factors that led to early military discharge (eg: those given a dishonourable discharge cannot access veterans' services). Iversen et al (2005) found that veterans with mental health problems were more likely be discharged early from the British Army, and to experience unemployment subsequently.



(Data from Reger et al 2015 table 2 p565)

Figure 10.4 - Suicide rate (per 100 000 person-years) based on separation from military.

The difference in findings between Ursano et al (2015) and Reger et al (2015) can be explained by methodological differences, in particular:

- Services covered - US Army (Ursano et al 2015) vs military.
- Period covered - 2004-9 (Ursano et al 2015) vs 2001-9.
- When suicide occurred - Ursano et al (2015) did not cover post-military.
- Data source - eg: veterans who seek help from Veterans Administration, but they "likely represent a vulnerable sub-population" (Reger et al 2015).

10.2. BENEFIT CLAIMANTS

Suicides have increased in the UK in the 21st century to peak in 2013 (Barr et al 2015a), and this has

been linked to the 2008-10 recession, in part (Barr et al 2012). Another contributing factor is the Work Capability Assessment (WCA), which reassesses individuals on out-of-work disability benefit. The experience is very as stressful as claimants can be denied disability benefit if they are classed as fit for work (Barr et al 2015a).

A survey of psychiatrists in Scotland found an increased frequency of appointments, medication, and self-harm among individuals with mental health problems after their WCA (quoted in Barr et al 2015a). However, this was only a survey.

Barr et al (2015a) used data sets on suicides, self-reported mental health problems, and anti-depressant usage from official sources (ie: the Office for National Statistics). Suicide and injury of undetermined cause in the working age population (18-64 years) between 2004 and 2013, anti-depressant prescribing rates per 100 000 population for 2010 to 2013, and self-reported mental health problems per 100 000 working age population between 2004 and 2013. The information was analysed for 149 local authority areas in England, along with the proportion of the working age population that received an outcome from a WCA (per 100 000 population) between 2010 and 2013. Level of deprivation per local authority area was controlled for.

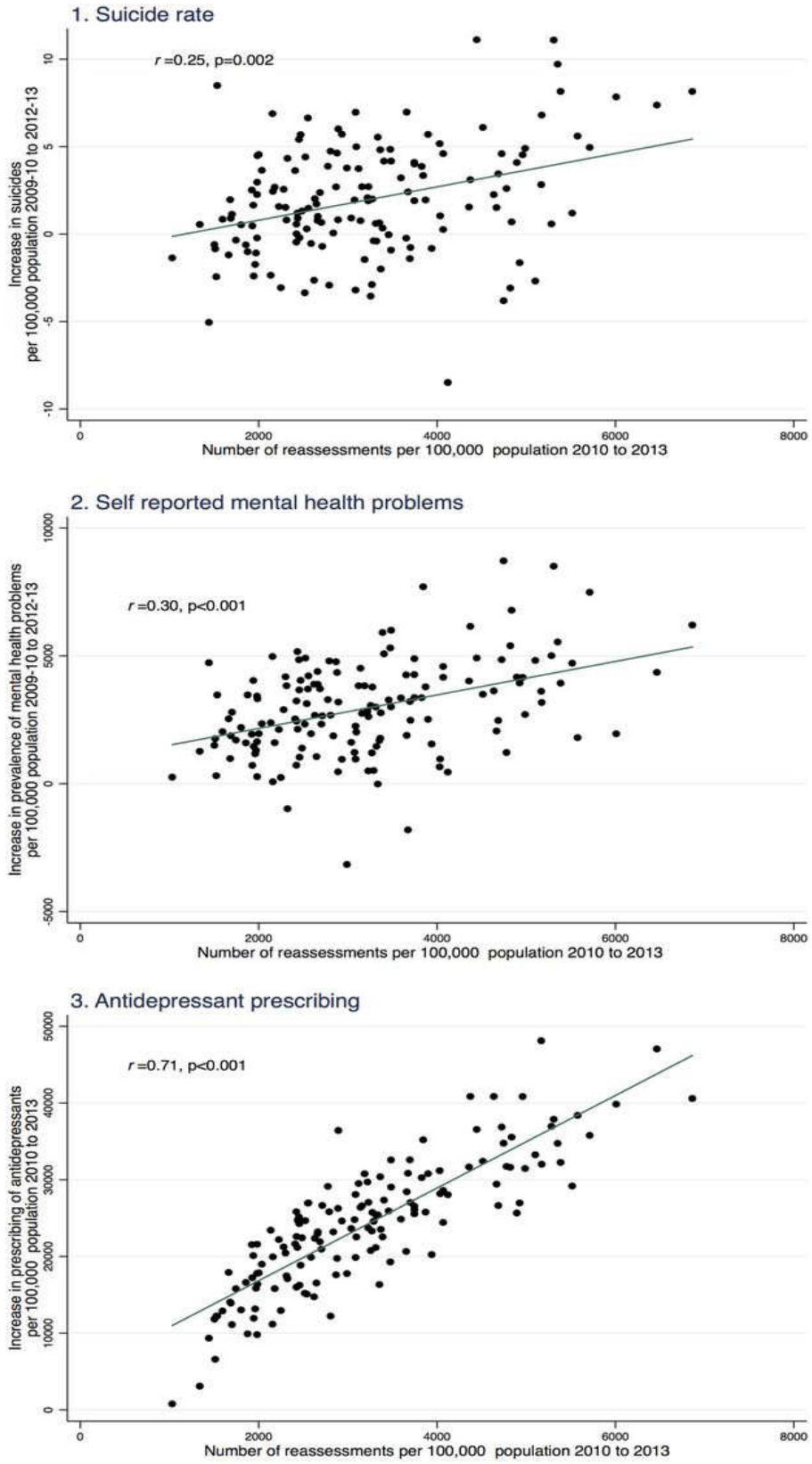
Overall, 1.03 million claimants were reassessed with a WCA, which is 3010 people per 100 000 working population. After controlling for depression, in areas where reassessments were higher, there was a greater increase in suicides, self-reported mental problems, and anti-depressant prescribing (figure 10.5).

Statistical modelling estimated that for every 10 000 people reassessed, there were an additional six suicides ($p = 0.002$), 2700 cases of reported mental health problems ($p = 0.014$), and 7020 items of anti-depressants prescribed ($p<0.001$).

Barr et al (2015a) calculated that the WCA process between 2010 and 2013 in England was associated with 590 additional suicides (ie: 5% above the average level), 279 000 additional cases of self-reported mental health problems (ie: 11% above average), and 725 000 additional anti-depressant items (ie: 0.5% above average).

These findings are critical of the UK Government's policy of WCA, and so there has been a political reaction to the research. For example, the Government department involved (Department for Work and Pensions; DWP) described the study as "wholly misleading" (McVeigh 2015a). Barr et al (2015b) countered that the DWP had not taken the concerns raised seriously. Jed Boardman (a consultant psychiatrist) is quoted as saying that individuals with mental health problems will relapse because of the anxiety caused by WCA (McVeigh 2015b).

With such debate, it is important to understand the



(Source: Barr et al 2015a figure 1)

Figure 10.5 - Associations between WCA and mental health outcomes per local authority area.

study's methodological strengths and weaknesses (table 10.2).

STRENGTHS	WEAKNESSES
1. Three separate mental health outcomes from independent data sources all showing the same association, and thus "reducing the likelihood that the results are due to spurious correlations" (Barr et al 2015a). 2. The associations were statistically significant, even after controlling for variables like area deprivation: suicide ($r = 0.25$; $p = 0.002$), mental health problems ($r = 0.30$; $p<0.001$), and anti-depressants ($r = 0.71$; $p<0.001$). 3. The findings were time-lagged (ie: WCA before mental health outcome), which makes reverse causality unlikely.	1. The data were aggregated (ie: totals) and it was not possible to identify "whether the additional people experiencing the adverse mental health outcomes are the same people who have undergone reassessment" (Barr et al 2015a). 2. Unobserved confounding factors could explain the associations. Barr et al (2015a) defended themselves: "We... found no association with trends in heart conditions in the working age population, or trends in prescribing of cardiovascular drugs. (ie: health conditions that would not plausibly be affected by the WCA reassessment process, in the short term at least). These test results suggest that the observed association between the reassessment process and mental health outcomes in the working-age population is not due to unobserved confounding...". 3. The nature of the data - eg: self-reports of mental health problems; inclusion of injuries of undetermined cause. But Barr et al (2015a) argued that "such biases are probably relatively constant over time" and have little influence on the main findings.

Table 10.2 - Key strengths and weaknesses of methodology of Barr et al (2015a).

10.3. SUICIDE NOTES

Suicide notes are left by up to one-third of individuals (Fernandez-Cabana et al 2015). These notes have been analysed by researchers in two ways (Fernandez-Cabana et al 2015):

1. A profile of individuals who leave notes, and comparison with those who do not.

For example, note writers in an Indian study of forty notes were young males who committed suicide by hanging at home, and showed signs of depression (Bhatia et al 2006).

In the USA, a study of 621 notes found that note writers lived alone more than non-writers, but were less

likely to have made previous suicide threats (Callanan and Davis 2009). While in an Australian study of 1051 notes, note writers were younger than non-writers, divorced or living alone, severely stressed, for instance, but less psychotic (Haines et al 2011).

2. Content analysis of the suicide notes.

Foster (2003), for example, looked for common themes, which included most often "apology/shame" (74% of notes), "love for those left" (60%), and "life too much to bear" (48%). Leenaars (1996) distinguished between intrapsychic variables in the notes (eg: unbearable psychological pain) and interpersonal variables (eg: problems with relationships).

Content analysis has also been made of real and fake notes. Anger and guilt appear more frequently in real suicide notes (Fernandez-Cabana et al 2015).

Content analysis tends to be linguistic analysis, using techniques like the Linguistic Inquiry and Word Count (LIWC) (Pennebaker et al 2001), which is software that analyses 72 variables (eg: number of pronouns, positive emotions, words related to time). Suicide notes from attempters have fewer positive emotions, for example, than from completers, while real notes have more pronouns and are longer than fakes (Fernandez-Cabana et al 2015).

Fernandez-Cabana et al (2015) pointed out that LIWC had only been used on suicide notes in English. The researchers rectified this problem by a study of twenty-three notes from deaths in Ourense, Spain between 2006 and 2009. There were 121 other cases of suicide without a note used as a comparison group. Personal information about the whole sample came from the records of the medical examiner, court files, and interviews with relatives, friends and neighbours.

In terms of the differences between those who left a note and those who did not, the former were younger (54 vs 68 years old), more likely to be single, divorced, separated or widowed, to use the workplace as the site of suicide, and report emotional troubles as the trigger (compared to mental illness for non-writers [84]). There were no differences between writers and non-writers in gender, residence (rural or urban), previous attempts, and method used.

Using LIWC, suicide notes by women were longer, and included more positive emotions, for instance, than by men. There were some differences based on age (eg: use of

[84] "However, this variable should be observed with caution because it was subject to informant bias and it could not be collected in up to 35.42% of total cases" (Fernandez-Cabana et al 2015 p152).

exclamations by younger writers), and residence (eg: more punctuation marks by urban writers).

10.4. MEDIA

Redfield (1999) summarised the characteristics of general media coverage that can encourage "suicide contagion" [85]:

- Presenting simplistic explanations for suicide.
- Excessive reporting of suicide.
- Sensational coverage.
- Including "how-to" information.
- Presenting suicide as a means to achieve certain goals.
- Glorifying those who have committed suicide.
- Focusing on suicide completer's positive characteristics.

Today, however, Internet activity can be as influential as "traditional" media coverage. Westerlund et al (2015) analysed the posts on a Swedish Internet forum in October 2010 where a young adult male user ("thread starter"; TS) started a thread called "Hanging", and subsequently hanged himself streamed live on webcam [86]. Westerlund et al (2015) observed: "With the massive communication possibilities that the internet has brought, problematic subjects like suicide have become more accessible to individuals than ever before. Today, the internet has become the main platform for information and communication about suicide. Conversations and disclosures regarding suicide occur on a large variety of internet forums, and it has been debated if online communication about suicide primarily provides opportunities or poses a serious threat for those who communicate about suicide-related issues" (p476).

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[85] Based on report of a workshop at the Centres for Disease Control (O'Carroll and Potter 1994).

[86] Other such cases include a teenage boy in Miami in November 2008, and a British adult in March 2007 (Westerlund et al 2015).

In terms of the live streaming, Westerlund et al (2015) observed: "Of course there can be many reasons why an individual wants to take their own life in public. One can be about loneliness. Perhaps the knowledge that other participants were watching the suicide gave the TS a sense of community, so he would not have to die alone... When the TS chooses to 'share' his suicide with the other participants, it can be interpreted as a final attempt to break the social isolation and loneliness, although it may seem contradictory.

A suicide is not just about dying. A suicide also communicates something to the world around the person, such as that the person feels unloved and outcast. The suicidal person achieves something with the act, something that the recipients cannot escape. It is a way to regulate the social environment. The suicidal act can be seen as a powerless person's weapon to influence the outside world in a way in which the recipients are deprived of the ability to speak back, which is a fundamental point" (p480). Ozawa-De Silva (eg: 2010) reported the sense of sociality of Internet-based suicide pacts in Japan.

Harris et al (2009) found that individuals who search for suicide-related material on the Internet score high on suicide-risk variables (eg: suicidal thoughts; living alone; history of psychiatric diagnosis; less perceived social support from family and friends). "Suicide-related online users find open, anonymous, unmoderated and peer-to-peer internet forums to be supportive and useful for their perceived needs – a 'place' where they could find participants similar to themselves – and that communication with family, healthcare professionals and help sites is less satisfying" (Westerlund et al 2015 p476).

Owen et al (2012) defined "suicide communication" as the "set of circumstances in which a person expresses suicidal feelings, thoughts, intentions or plans, either directly or indirectly, in interaction with other people in their social environment" (quoted in Westerlund et al 2015). About one-third to half of individuals communicate directly beforehand about their intentions, and up to 80% indirectly (Westerlund et al 2015).

The case study by Westerlund et al (2015) consisted of 638 messages in the thread on the 11th October 2010 between 11.51 hours (1st posting) and 17.26 h (when closed by the moderator) (while the suicide act occurred at 13.00 h).

Firstly, thirty posts before the suicide act were analysed, of which nine were from the TS. Of the twenty-one posts from other users, about half were encouraging or inciting suicide, and the others were discouraging (table 10.3).

- Thread starter - I have now decided to kill myself by hanging. I have softly tried to strangle myself and saw how that feels. Took some painkillers a few minutes ago (100mg dexofen and 1500mg paracetamol), now waiting for it to start working. Have turned on my webcam with a program that makes a screenshot every 2 seconds and put up an FTP [file transfer protocol, a standard way of transferring computer files on the internet] where the images will be available, will post the IP, port and login details before I do it.

- First two replies - Good luck then!.
  - It can't be that bad... When everything is at its worst it can only get better.

- Encouraging/inciting posts - Stupid fuck, strangulation is no pleasure. Don't you have a car... carbon monoxide rules.
  - In the way you write, one can see that you're just a faker, go and hang yourself.

- Discouraging posts - Can't you tell us a little about your life TS?
  - Don't do it, there are other solutions.

(Source: Westerlund et al 2015 p478)

Table 10.3 - Examples of posts before suicide.

The second analysis period covered 608 posts during and after the suicide. Postings questioned the authenticity of TS, including about 10% after the event who still thought it was fake. In a ratio of two to one, users expressed the attitude that suicide is "tragic, terrible or shocking" compared to "exciting, interesting or funny" (table 10.4).

In terms of prevention, more postings felt that it could or should have been prevented compared to could or should not. The question of responsibility was perceived as divided between TS and those who incited him.

- "Tragic" posting - Horrible. Moreover, this kind tends to rub off and inspire others. Hope that TS is in a better place now.
- "Exciting" posting - Call me sick, but have never laughed so much in my life lol ["laugh out loud"].
- Prevention - However, we could have acted faster. He could have survived if we had called him [versus] Ha-ha, awesome, if you want to kill yourself it's your own decision, no one should interfere.
- Anger at "inciters" - All these disgusting idiots on [forum name] who incited him to do it. Hope you will suffer for the rest of your lives. Filthy bastards!

(Source: Westerlund et al 2015 p478-479)

Table 10.4 - Examples of posts during and after suicide.

Westerlund et al (2015) commented on the reaction to TS:

> Even if people want to help, lay people are often hesitant when confronting a person in a suicidal crisis, because of a lack of knowledge and the fear, anger and anxiety that such communication acts can awaken. A suicide communication event can be a highly face-threatening situation, both for the suicidal person and other people involved in the communication. The jokes and the ironic comments made by the participants, and the fact that some did not take the TS's posts seriously, could partly be understood by the taboo and stigma that surround suicide. Also, popular myths about suicide, such as 'people who talk about suicide don't do it' and 'asking about suicidal thoughts may create suicidal ideas' may discourage lay intervention. This could be a problem on internet forums like [forum name] where many participants are rather suspicious because of the high level of deceptive posts and trolling. In this environment inaccurate assumptions and myths can continue to flourish (p480).

The case study raised a number of ethical issues, which Westerlund et al (2015) were aware of:

a) The study was published five years after the

event (ie: consideration for relatives and participants).

b) Consent not required as postings in public domain.

c) Though most postings were anonymous or hidden with username, any identifying information was removed by the researchers.

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11. ADDICTION

11.1. MAKING SENSE OF ADDICTION

Addiction [87] is often studied by different disciplines with no common ground. "Scientists tend to reduce addiction to the domain of pathological body, whereas social scientists often produce analyses that seem to overlook that drugs are chemical substances, that drug users have bodies, and that the contemporary sciences are altering the ways that we understand and act upon substances and bodies" (Dunbar et al 2010 p2) [88].

The biological approach [89] emphasises how prolonged substance use changes the brain in an irreversible way [90]. This has been called "the NIDA paradigm" [91] after Alan Leshner, director of the National Institute for Drug Abuse (NIDA) in the USA who popularised this position (Dunbar et al 2010) [92] [93]. The key behavioural

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[87] For convenience, and accepting exceptions, Kuhar (2010) defined drug abuse/dependence/addiction as "(1) a biological brain disorder, (2) a long-lasting and relapsing syndrome, (3) sometimes powerful and (4) treatable" (p25).

[88] Vrecko (2010) noted that "addiction has long been (and remains) a 'hybrid' entity (Latour 1993) that combines a mixture of scientific, psychological, moral, technological, social and spiritual elements" (p37).

[89] Kuhar (2010) summed up the knowledge gained from "basic science" about addiction:
1. Animal studies show a biological basis to addiction, as opposed to "solely a moral or character failing of certain people".
2. Drugs activate brain areas related to survival.
3. The hi-jacking of such brain areas make drugs so powerful in controlling behaviour. (The idea that addiction hi-jacks the brain is a metaphor, not an explanation, stated Acker (2010): "'Hi-jack' is a volatile term , resonant with the greatest fears we have about the world today" (p72).
4. At a biochemical level drugs change the brain. "The brain on drugs really is a different brain!" (p33).
5. Drugs produce instability in the biochemistry of the brain.
6. Substances that target the biochemistry targeted by drugs could offer a treatment for addiction.
7. Drug-induced brain changes are long-lasting and so should treatment be.
8. Drugs affect brain areas involved in decision-making, for example.

[90] Scientist can disagree, including over whether behavioural cues and reinforcers are important in giving drugs their "power" (Acker 2010).

[91] Also chronic, relapsing brain disease (CRBD) model (Campbell 2010). This is an "ideological code" - a "schema that replicates its organisation in multiple and various sites" (Smith 1999 quoted in Campbell 2010). Redefining addiction as a CRBD "rendered it tractable to neuroimaging techniques" (Campbell 2010).

[92] Acker (2010) noted that "the laboratory, far from being an isolated and impermeable space, is intimately linked with the social world around it. Influence flows in both directions" (p71).

[93] A difference between addiction and habituation, as in early definitions of addiction (eg: Tatum and Seevers 1931), allowed the distinction between "good drugs" and "bad drugs". Addiction is seen in the need for the drug to avoid withdrawal (as in morphine), while habituation is a desire for continuing drug use without withdrawal (eg: stimulants, cigarettes). Rasmussen (2010) pointed out the consequences of

characteristics are "uncontrollable, compulsive drug craving, seeking, and use, even in the face of negative health and social consequences" (Alan Leshner quoted in Courtwright 2010) (appendix 11A).

But "the claim that addiction is a brain disease requires contextualisation" (Kushner 2010 p9). For example, the NIDA paradigm "is not new, but rather is a restatement of long-held views repackaged in the current vocabulary of neuroscience" (Kushner 2010 p9). Kushner (2010) felt that nineteenth century neurologists would have found the idea of addiction as brain damage familiar as current views resemble past views, like degeneration theory. This sought a hereditarian explanation for disorders, and saw degeneration as organic, "but much like addiction therapies today, treatment revolved around an array of psychological and moral interventions under the rationale that alterations in habits had a direct physiological influence on the nervous system" (Kushner 2010 p10).

A key aspect of the contextualisation of addiction relates to social variables, like social class or ethnicity. For example, Hickman (2004) referred to the "double meaning of addiction" as wealthy cocaine users are treated as "chemically dependent" (and given rehabilitation), while the poorer users are seen as criminals and imprisoned. Both groups purchase their substances from the same place.

If the focus is upon individual brains, "how do we understand disproportionate concentrations of sever drug problems in particular groups", asked Acker (2010)? She used the example of the "crack epidemic" in the 1980s in inner-city areas of the USA. "That many diseases concentrate in the poorest, least employed, most nutritionally deprived, and most socially and culturally isolated population groups in American society suggests that it is these conditions, not the power of a drug or a bacterium, that accounts for these patterns. While the chronic relapsing brain disease model offers an explanation of the impact of crack use on individuals, it is less forthcoming on the issue of rates of addiction in

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such a distinction: "For so long as the stigma of addiction was confined to morphine congeners, the 'merely habituating' stimulant drugs could be legitimately and profitably sold" (p119). Put into figures, 200 000 - 300 000 "pariahs injecting opiates in post-war America" compared to "some 72 million American cigarette smokers" (Rasmussen 2010).

specific population groups" (Acker 2010 pp82-83).

Kushner (2010) also emphasised the context of prescription drugs. These substances are being promoted in relation to mental health, say. So, the brain changes induced by a licit substance, like "Ritalin" (an amphetamine-like basis) are seen as positive for ADHD sufferers [94].

Kushner (2010) described addictions as "syndromes of dependence, informed and 'enabled' by an interaction of culture and biology" (p19). This gives us a "cultural biology of addiction" which sees addiction as a combination of both culture and biology (Kushner 2010).

The inclusion of behaviours, and more of them, into the concept of addiction "sheds light on how tropes of addiction are increasingly used in modern societies to demand more self-control from individuals who experience fewer societal constraints on their lives and behaviours; on how individuals identified as addicts are expected to work on themselves in an ever-expanding array of 12-step programmes and self-help groups; and on how even those of us who currently consume and behave in non-compulsive, unproblematic ways may find ourselves subjects of pre-emptive regimes of monitoring and control, as when we more or less constantly reflect on the nature of our behaviours and our risks of becoming one or another type of addict" (Vrecko 2010 p39). Thus, the role of "addiction treatments" is as "civilising technologies" - "to produce better citizens, rather than to cure biological diseases" (Vrecko 2010).

Rose (2007) described how "the 'molar' biology of the visible body, of organs, tissues and cells, has lost its prominence in the face of the molecularised biology of coding sequences, intracellular elements and neurotransmitters" (Keane and Hamill 2010 p52). This changes "shapes and establishes the very object of explanation" (Rose 2007). For example, naltrexone treatment for heroin addiction blocks the opiate receptors and thereby reduces the craving/desire for drugs. There is no need to treat or reform the whole person, and "the moral significance of addiction is profoundly altered by the shift from disordered subject to neural anomaly" (Keane and Hamill 2010 p53).

This seems to remove the "moral weight" associated with addiction in the past. But the idea of the "disordered drug abuser" is not far away as drugs produce

[94] Windle (2010) proposed a multi-level developmental context approach to addiction which included multi-factor influences (including genetic, biochemical, social, and societal), and factors related to lifespan development (eg: onset in adolescence). This was a sharp contrast to single-factor disease models.

the "permanent" change to the brain (Keane and Hamill 2010).

Addiction as a brain disease or disorder is different to the understanding of addiction in pain medicine, which distinguishes that from physical dependence. Physical dependence is seen as a "normal" part of extended (appropriate) use of painkillers (eg: opiates), while addiction is a psychological condition (recognised by out-of-control behaviours and compulsive drug use) (Keane and Hamill 2010). Bell and Salmon (2008) noted the distinction here between the "deserving pain patient" and the "undeserving addict". However, pain medicine does accept the pseudo-addict" with "desperate drug-seeking, produced not by true drug addiction, but by the undertreatment of pain" (Keane and Hamill 2010).

"The management of a patient with addiction and chronic pain is complex not just because it involves co-morbidity, but because it represents the convergence of two different models of substance dependence, two different evaluations of the harm of addictive drugs and two differently valued drug-using identities" (Keane and Hamill 2010 p62).

Keane and Hamill (2010) argued that pain medicine has remained at the level of "molar biology" while addiction science is "molecular". But the "upsurge in painkiller abuse" in the USA in recent years challenges any distinction because "opiates are addictive and dangerous, whatever the context of their use" (Keane and Hamill 2010 p65).

11.2. BIPOLAR DISORDER AND PROBLEM GAMBLING

"Problem gambling" is defined as "excessive gambling behaviour that causes harm to the individual, their family and friends or to the wider community" (Jones et al 2015). Its prevalence is estimated at 0.7% of the general population in the UK in 2010 (Wardle et al 2011). But that figure is higher among individuals with bipolar disorder (Jones et al 2015).

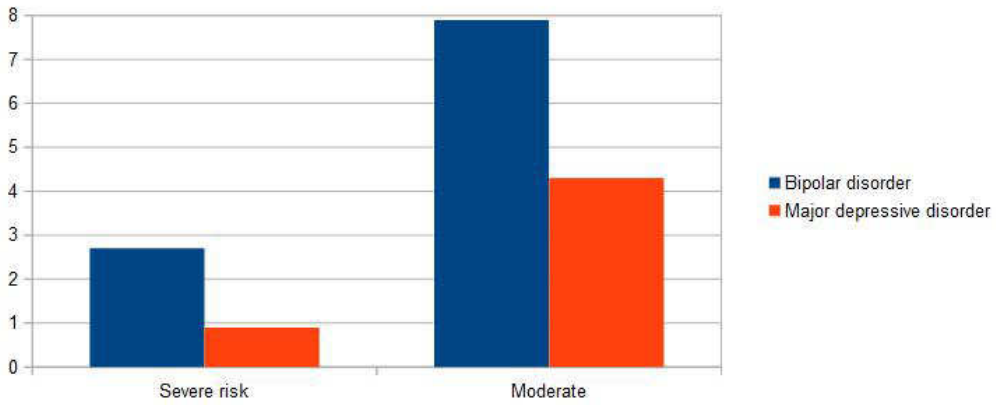
Jones et al (2015) drew participants from the Bipolar Disorder Research Network (BDRN), which covers adults with a lifetime diagnosis of affective disorder in the UK. Seven hundred and fifty volunteers responded to a mailing, and were assessed for gambling behaviour by the self-report Problem Gambling Severity Index (PGSI) (Ferris and Wynne 2001). This has nine items (table 11.1), each scored 0-3, for the last twelve months. Total scores of eight or more (out of 27) are classed as "severe risk", and 3-7 as a "moderate risk".

- Have you ever bet on more than you could afford to lose?
- Have you ever felt guilty about the way you gamble?
- Any health problems due to gambling?

(Source: Jones et al 2015 table 2 p330)

Table 11.1 - Example of items from PGSI.

Of the volunteers, 635 had a diagnosis of bipolar disorder and the remainder Major Depressive Disorder. The prevalence of severe risk of gambling problems was 2.7% of the former, and 0.9% of the latter (figure 11.1).



(Data from Jones et al 2015 table 1 p330)

Figure 11.1 - Prevalence (%) of problem gambling [95].

Comparing the moderate or severe risk bipolar group to the rest of the bipolar disorder sample, the former were significantly younger (median age 40 vs 46 years), and were more likely to work in the service industry. There was no difference in gender [96], education, and marital history between the two groups.

Problem gambling was significantly associated with diagnosis of type 2 bipolar disorder [97], with a younger age of onset, a history of rapid cycling (ie: four or more episodes of mania or hypomania in one year), and a history of suicidal ideation and/or attempt.

Jones et al (2015) found that the Internet was involved in about a quarter of bipolar disorder

[95] Kennedy et al (2010) reported a prevalence of 12% for moderate and severe risk among bipolar individuals in Canada.

[96] Other studies of the general population and individuals with bipolar disorder found more men had problem gambling than women (Jones et al 2015).

[97] This is characterised by hypomania (low mood) rather than mania compared to type 1, and no psychotic symptoms.

individuals with problem gambling. Lloyd et al (2010) reported that online gambling problems were associated with hypomanic experiences. "Therefore, these data suggest that the characteristics of mild mood elevation involving enhanced reward focus, sleeplessness and distractibility constitute particular risk factors for problematic use of gambling services" (Jones et al 2015 p331).

There are four main methodological issues with this study:

1. Self-report measure of gambling behaviour - risk of recall bias or social desirability versus anonymity and confidentiality of a postal questionnaire.

2. Response rate of 23% - ie: risk of response bias. "It is difficult to know whether this bias over- or underestimates the prevalence of gambling problems. People who are currently gambling might be more likely to be interested in the research and complete the questionnaire; conversely, they might prefer not to disclose their gambling behaviours and thus not respond. However, the PGSI was included in a mail-shot with a number of other questionnaires, and responders completed all questionnaires, which reduces the likelihood that the decision to respond was particularly influenced by the inclusion of the PGSI" (Jones et al 2015 p332).

3. The sample size of the severe risk problem gambling bipolar disorder group was very small (n = 17), which limited any further analysis, and thus the combination with moderate risk. But severe and moderate risk individuals may have differences as well as similarities.

4. Not possible to establish causality (ie: bipolar disorder leads to problem gambling) as it was a cross-sectional study finding associations/correlations only.

11.3. APPENDIX 11A - KEY BEHAVIOURS

Underlying addictive disorder are the processes of novelty-seeking, attentional bias, and cue-conditioning. Novelty- or sensation-seeking has been found to be associated with compulsive cocaine-seeking behaviours in rats, and binge drinking in human adolescents (Banca et al 2016).

Cue-conditioning is where certain neutral stimuli become associated with an addictive behaviour, and subsequently are triggers for that behaviour (eg: places or friends associated with drug use). Attentional biases involve "the tendency to preferentially process cues

related to the object of addiction" (Banca et al 2016).

Banca et al (2016) explored these three processes in relation to online explicit sexual material among individuals with compulsive sexual behaviour (CSB). Twenty-two heterosexual males with CSB in eastern England were recruited via the Internet and therapist referrals, along with forty age-matched male volunteers as controls. All participants completed a number of questionnaires before testing at the University of Cambridge.

1. Novelty preference task - Participants were shown three categories of images to become familiar with (sexual, neutral human, and neutral object). Then there was a choice-discrimination test where a familiar and a novel image were presented, and the aim was to spot the latter. The CSB group were better at spotting the novel sexual images as compared to neutral images, whereas the controls were better at choosing the neutral human over the neutral object images.

2. Conditioning preference task - Participants were conditioned to associate two random visual patterns with an image of an undressed woman or a neutral grey box (sexual condition), or a £1 coin or a neutral grey box (monetary condition). In the testing phase, participants were shown the visual pattern and asked to guess what type of image would follow. The CSB group did better where the pattern was associated with either sexual or monetary images.

Together these two types of tasks showed an attentional bias towards sexual stimuli for CSB individuals.

3. Neuroimaging task - A conditioning task similar to the previous one was done with the participants undergoing functional magnetic resonance imaging (fMRI) to show the areas of the brain active. The CSB group had a difference in the dorsal cingulate region, but it was limited.

The tasks together showed that individuals with CSB preferred novel images more than controls, and were conditioned to related cues more than them, "possibly mediated by greater cingulate habituation along with a generalised enhancement of conditioning to rewards" (Banca et al 2016 p91).

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12. SUCCESSFUL PSYCHOPATHS

The "successful psychopath" (SP) (or adaptive or sub-clinical psychopath) is "an individual who displays many of the core features of psychopathic personality (psychopathy) while achieving success" (Lilienfeld et al 2015). This contrasts with psychopaths "ensconced safely behind prison walls, most of whom are presumably unsuccessful" (Lilienfeld et al 2015), who Hare (1993) called "the tip of a very large iceberg".

It is popularly believed that SPs are over-represented in areas like politics and business, whereas the actual research on the concept is limited and controversial (Lilienfeld et al 2015).

Widom (1977) was one of the first people to study SPs after placing advertisements in "underground newspapers" in Boston for "charming, aggressive, carefree people who are impulsively irresponsible but are good at handling people and looking out for number one" (quoted in Lilienfeld et al (2015). Twenty-eight relevant individuals came forward with autobiographical descriptions. Widom's analysis of these descriptions found similarities to psychopaths in prison (eg: low empathy), but also differences (eg: able to delay gratification). However, two-thirds have been arrested at least once. Widom and Newman (1985) found only 40% had been arrested in a similar study in Indiana.

But this research did not have a comparison group, and the advertisements "may have recruited psychopathic individuals who managed to stay out of prison rather than those who achieved social success" (Lilienfeld et al 2015). However, the advertisement method has been used in other studies.

So, researching SPs is limited by the recruitment process, how "successful" is defined, and the way of measuring psychopathy. For example, Ishikawa et al (2001) recruited males in Los Angeles from temporary-employment agencies, used the Psychopathy Checklist-Revised (PCL-R) (Hare 2003) (the standard measure), and classed successful as having not been convicted of a crime.

While Mullins-Sweatt et al (2010) asked attorneys and clinical psychologists to nominate one person they knew who was "a charming and guiltless social predator, who had achieved personal success" (Lilienfeld et al 2015). Personality tests of the nominated individuals found them to be higher on conscientiousness traits (eg: self-discipline), for instance, than psychopaths generally.

Lilienfeld et al (2015) presented three ways of conceptualising SPs:

i) Differential-severity model - This assumes that psychopathy is a unitary construct, and the SP is a mild version of clinical psychopathy.

ii) Moderated-expression model - The SP is an untypical version of psychopathy as the individual has been tempered by protective factors, like effective parenting, or superior executive functioning. This also assumes that psychopathy is a unitary construct.

iii) Differential-configuration model - Psychopathy is seen as a combination of personality traits, and the SP has more positive than negative traits, or different traits, to clinical psychopaths.

The last model has support from work on a personality dimension called "fearless dominance" (eg: boldness, physical fearlessness, emotional resilience). High levels of fearless dominance has been found in studies of SPs, including of US Presidents by their biographers (Lilienfeld et al 2012), and of business leaders and employees in high-risk occupations (eg: fire service) (Lilienfeld et al 2014).

But not all researchers agree with this work. Lilienfeld et al (2015) admitted two caveats:

a) It could be that "fearless dominance is only peripherally relevant to psychopathy... One possibility is fearless dominance is a protective variable that helps to differentiate successful from unsuccessful psychopathy, with the former condition characterised by charisma and venturesomeness. If so, the aforementioned findings on fearless dominance may be better explained by a moderated-expression model than by a differential-configuration model" (Lilienfeld et al 2015 p301).

b) Conscientiousness traits seem to be part of the SP. "Because fearless dominance is largely unrelated to conscientiousness, the former dimension may be insufficient to explain successful psychopathy; if so, a full account of successful psychopathy may require multiple traits" (Lilienfeld et al 2015 p301).

Patrick et al (2009) proposed the triarchic model, which fits with the differential-configuration view. Psychopathy is a combination of three personality dimensions - boldness (or fearless dominance), disinhibition, and emotional coldness (meanness). The SP has higher boldness and lower disinhibition than clinical psychopaths, but is similar in terms of emotional coldness.

This idea fits with the "findings that (a) fearless dominance is associated with adaptive behaviours and (b)

successful psychopathy is associated with elevated conscientiousness and intact or superior executive functioning, both of which are tied to low disinhibition" (Lilienfeld et al 2015 p302).

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13. ESTABLISHING THE ADVERSE EFFECTS OF CANNABIS

Appendix 13A - Low birth weight
Appendix 13B - Car accidents
Appendix 13C - Mental disorders
Appendix 13D - Fergusson et al (2003)
Appendix 13E - Meier et al (2012)
Appendix 13F - Science

The use of cannabis/marijuana is increasing [98], and this is, in part, due to the reduction of legal penalties related to its use. For example, thirty-nine US states and Washington DC allow medicinal use, while four states have legalised recreational use, including Colorado which has legalised the "whole production chain" (Cressey 2015). "The popular notion seems to be that marijuana is a harmless pleasure, access to which should not be regulated or considered illegal" (Volkow et al 2014 p2219). However, "the evidence for marijuana's effects on health and behaviour is limited and at times conflicting" (Cressey 2015 p281) [99].

Earlier studies (ie: pre-1990s) tended to be with animals, or human (students usually) laboratory studies (Hall 2015). Hall et al (1994) attempted to establish causal inferences about the health effects of cannabis use from three types of studies - case-control, prospective, and experimental - as well as determine cannabis use before outcome, and control for potential confounding variables.

Providing evidence for the Australian government, Hall et al (1994) identified the following adverse effects:

- Anxiety, and paranoia (especially for naive users)
- Cognitive impairment
- Psychomotor impairment
- Risk of psychotic symptoms with high doses
- Risk of low birth weight for babies born to cannabis users during pregnancy (appendix 13A).

Hall (2015) reflected on the research since 1993, and the conclusions today.

The risk of overdose is "extremely small" and this is an "uncontroversial conclusion", as up to 70 g may be

[98] Around 12% of 12 year-olds and above in the USA report use in the past year (Volkow et al 2014). In fact, recreational cannabis use is almost as common as tobacco use among adolescents and young adults, with regular users smoking 3-5 "joints" per day (Hall 2015).
[99] The emphasis is always on science (appendix 13F).

required for a fatal dose (Hall 2015).

In terms of the short-term effects, impairment of memory and co-ordination are certainties, as seen in the increased risk of car crashes (appendix 13B) (eg: Ramaekers et al 2004), while paranoia and psychosis (appendix 13C) are a possibility (Cressey 2015).

In the longer term, addiction/dependency occurs in small number of users, though there is disagreement and problems with establishing other effects (Cressey 2015). The risk of dependency is around one in ten users (as compared to one in seven for alcohol and one in four for heroin) (Hall 2015). The risk is higher for adolescent initiation of use (1 in 6) and daily users (one in two) (Hall 2015).

The "smoking pot" method of intake could increase the risk of lung cancer (eg: Aldington et al 2008) (table 13.1) [100]. Other studies in New Zealand suggested psychotic symptoms (eg: Fergusson et al 2005), poor educational performance (eg: Fergusson et al 2003; appendix 13D), and declines in IQ (eg: Meier et al 2012; appendix 13E).

- This was a case-control study in New Zealand [101] with 102 adult cases (below 55 years old) with a lung cancer diagnosis between 2001 and 2005 taken from hospital databases, and compared to 493 random controls. All individuals, who agreed to participate (79 cases and 324 controls) were interviewed face-to-face about their lifetime use of cannabis and tobacco.

- The risk of lung cancer increased by 8% for each joint-year of cannabis (ie: 1 joint per day for 1 year) [102], and by 7% for each pack-year of cigarettes (ie: 1 pack per day for 1 year). The risk of lung cancer was over five times higher for the highest third of cannabis users (greater than 10.5 joint-years) as compared to non-users.

- Key methodological issues:
  * Retrospective study (ie: working backwards from lung cancer to discover cannabis use).
  * Self-reports - depends on honesty of replies and accuracy of recall.
  * Small number of cases.
  * "Non-users" defined as less than 20 joints in lifetime.

Table 13.1 - Details of Aldington et al (2008)

[100] Cannabis smoking has some similarities to tobacco smoking, and some differences (eg: greater absorption of carbon monoxide as smokers inhale more deeply) (Aldington et al 2008).
[101] Cannabis use is high in New Zealand, and cannabis is rarely mixed with tobacco (Aldington et al 2008).
[102] This was a standard measure that combined the intensity (amount and frequency) and duration of use (Aldington et al 2008).

Focusing on cognitive performance, studies often do not match users and non-users for intellectual function, or include measures of cognitive performance prior to cannabis use (Hall 2015). Better-controlled studies find that regular users have deficits in verbal learning, memory and attention, but it is not clear if these cognitive functions recover after cessation of long-term use (Hall 2015).

Regular cannabis use during adolescence can impair neural connectivity, particularly in the precuneus (related to self-conscious awareness) and the fimbria in the hippocampus (related to memory) (Volkow et al 2014).

More widely, earlier initiation of cannabis use was associated with early school-leaving, even controlling for parental social class, for instance (eg: Ellickson et al 1998). But whether this was causation and not just association, Hall (2015) was unsure: "It is plausible that educational outcomes in regular cannabis users are impaired as a result of a combination of: a higher pre-existing risk of educational problems in those who become regular cannabis users, the adverse effects of regular cannabis use on learning in school, increased affiliation of regular cannabis users with other cannabis-using peers who reject school and a strong desire among younger cannabis users to make a premature transition to adulthood by leaving school" (p24).

There are a limited number of cohort studies suggesting possible long-term effects for children of cannabis smoking pregnant mothers. For example, Day et al (1994) followed a cohort of 655 low-income women in Pittsburgh who gave birth between 1990 and 1995. At age three years, children of regular cannabis users during pregnancy had lower memory scores on an IQ test, more problem behaviours and lower educational achievement at age ten, and poorer school performance at fourteen years old (Hall 2015). But the study was not able to control for genetic differences in IQ (Hall 2015).

There is concern that cannabis use is a gateway to the use of other illicit drugs. This may be due to the opportunity to get these substances (eg: from cannabis dealer), or the chemical properties of cannabis increase the desire for other substances [103]. However, individuals who are risk-takers may simply try novel substances (Hall 2015). Hall (2015) summed up: "the relationships between regular cannabis use and other illicit drug use have persisted after statistical adjustment for the effects of confounding variables in both longitudinal studies and

[103] Animal studies (eg: rats) find reduced activity in the brain's reward areas after adolescent exposure to cannabinoids (ie: the brain is "ripe" for other substances) (Volkow et al 2014).

discordant twin studies" (p25).

There are a number of general problems with the studies:

i) Many confounding variables make it difficult to tease apart correlation and causation. For example, some cannabis users also consume large amounts of alcohol. "Attributing the effects of one particular substance or behaviour is therefore very difficult" (Cressey 2015 p281). Macleod et al (2004) has raised doubts about statistical analyses that control for confounders.

ii) In the case of correlations or associations, the relationship may be two way. For instance, with the correlation between cannabis use and schizophrenia, individuals developing the condition may self-medicate with cannabis, but this is difficult to prove (Cressey 2015).

iii) Problems in establishing the amounts of chemicals, like tetrahydrocannabinol (THC) [104], in cannabis as potency varies. This also means the older studies with lower-potency cannabis have limited application to high-potency cannabis today (eg: three-fold increase in potency between 1990s and 2010s using official US data) (Cressey 2015) [105]. However, Di Forti et al (2015) did find a link between the risk of psychosis and high-potency cannabis use (eg: "skunk" with high THC), but not with lower-potency use (eg: "hash" (resin)) (table 13.2).

iv) In self-report studies, categories vary - for example, daily use can classed as "heavy" or "regular" use (Hall 2015).

Hall (2015) summarised the findings of studies in the last quarter of a century:

- Greater risk of car accident if driving when intoxicated by cannabis (and even greater with alcohol as well).
- Cannabis use during pregnancy "modestly reduces birth weight".
- Dependence is possible with regular use, but not fatal overdose.

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[104] The cannabidiol (CBD) level can modulate the effect THC (Hall 2015).

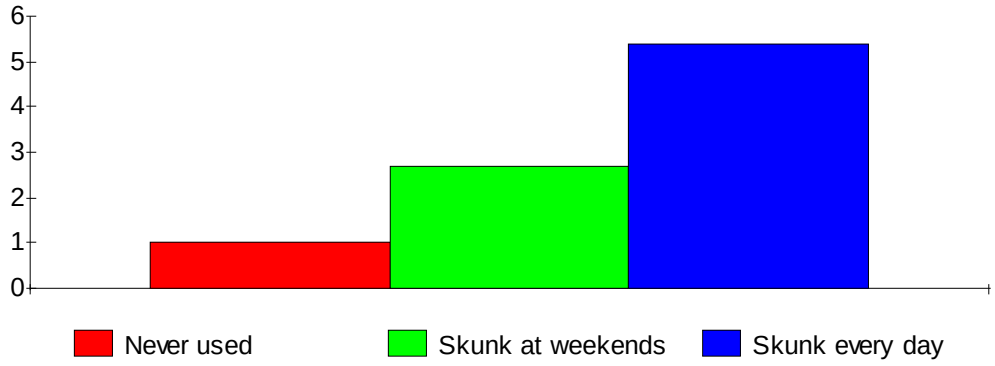
[105] "Some argue that the effects will be minimal, because users titrate their doses of THC to achieve the desired level of intoxication, but recent evidence suggests that regular cannabis users titrate their THC doses incompletely when given more potent cannabis products" (Hall 2015 p29).

Di Forte et al (2015) performed a case-control study using 410 individuals with first-episode psychosis admitted to a psychiatric hospital in south London between 2005 and 2011, as compared to 370 population controls (who volunteered in response to Internet and newspaper advertisements, and leaflets in the local area). The lifetime frequency of cannabis use, and potency of the cannabis used were measured, and combined into a cannabis exposure score, where 1 was "hash less than once per week every week" and 6 was "skunk every day". Cannabis use and potency were based on self-reports and estimates.

The cases were no more likely to have ever used cannabis than the controls, but they were more likely to use high potency cannabis regularly. Individuals who used skunk every day were five times more likely to be diagnosed with a psychotic disorder than individuals who had never used cannabis in their lifetime (figure 13.1).

Di Forti et al (2015) stated: "If causality is assumed, this finding suggests that skunk alone was responsible for the largest proportion of new cases (24%) [106] of psychotic disorder in the study population, an effect driven by its high prevalence among patients with first-episode psychosis who used cannabis (218 [53%] of 410 patients)" (p236).

In terms of reverse causality, the researchers commented: "A theoretical explanation of why skunk might have been preferred by patients with first-episode psychosis is that, when they began to experience their illness prodrome, these individuals might have sought increased concentrations of THC to self-medicate. However, experimental studies show that THC induces psychotic symptoms, while cannabidiol ameliorates them and reduces anxiety. That people who already have prodromal symptoms would choose a type of cannabis that is high in THC and has little cannabidiol (such as skunk), which might exacerbate their symptoms, rather than a cannabidiol-containing type (such as hash), would seem counterintuitive" (p237).



(Based on Di Forti et al 2015 figure 2 p237)

Figure 13.1 - Relative risk of psychotic disorder based on cannabis use and potency.

Table 13.2 - Di Forti et al (2015).

[106] This is known as the population attributable fraction (PAF), and compares to 8% in a New Zealand study and 13.3% in Holland (Di Forti et al 2015).

- Greater risk of psychosis, particularly for vulnerable individuals (eg: family history of psychotic disorders).

- Regular use linked to lower educational performance, use of other illicit drugs, and cognitive impairment.

APPENDIX 13A - LOW BIRTH WEIGHT

Animal studies show that high doses of cannabis extract for pregnant females produces growth retardation and birth malformations in offspring (eg: Bloch 1983). In relation to humans, there are problems with confounding variables - eg: pregnant cannabis users are less likely to seek antenatal care, and have poorer nutrition than non-users, as well as separating the effects of cigarette smoking (Hall 2015). Cannabis use during pregnancy is generally low (2-6% of women self-report) (Hall 2015).

While Zuckerman et al (1989), for example, actually found no increased risk of birth defects in a large sample. "None the less, there is a good case on the grounds of prudence for recommending that women should avoid using cannabis while pregnant, or while attempting to become pregnant" (Hall 2015 p22).

APPENDIX 13B - CAR ACCIDENTS

Culpability studies distinguish between drivers who were responsible for their crash and those who were not. Compared to drug- or alcohol-free cases, the odds ratio of being culpable in a fatal or non-fatal traffic accident is up to 6.6 (with heavy cannabis intoxication), similar for alcohol, and 11.5 for alcohol and cannabis, depending on the study (Ramaekers et al 2004).

One study in Quebec, Canada, found that cannabis and alcohol together increased the risk of a crash by eighty times, and cannabis and benzodiazepines (tranquillizer) by twenty-fold (Dussault et al 2002).

Culpability studies depend on accurate classification of responsibility, and assume that "drug free drivers involved in crashes are representative for the driving population at large" (Ramaekers et al 2004 p116).

In the case of earlier studies of fatal car crashes, cannabis metabolites [107] in the body "indicated only that cannabis had been used in the days before the accident; they did not show that the drivers were cannabis-impaired

[107] Eg: THC-COOH (an inactive carboxy metabolite of THC) (Ramaekers et al 2004).

at the time of the accident. Moreover, in many of these studies a substantial proportion of drivers with cannabis in their blood also had high blood alcohol levels, making it difficult to distinguish between the effects of cannabis and alcohol on accident risk" (Hall 2015 p21).

Other epidemiological methods of study include anonymous random telephone surveys, and case-control studies using individuals hospitalised after a car accident, say.

In a meta-analysis of case-control studies, the better-designed ones together showed the risk of car crash was doubled for recent cannabis use (Asbridge et al 2012). However, the risk from driving while intoxicated with alcohol is 6-15 times greater, and using French data, while cannabis use accounts for an estimated 5% of traffic fatalities, 29% were linked to alcohol (Hall 2015). Alcohol and cannabis together increase the risk of car accident substantially (Hall 2015).

More generally, Gerberich et al (2003), using data from 65 000 patients, found that cannabis users had higher rates of hospitalisation for any type of injury than former users and non-users.

Experimental studies allow a controlled assessment of the effect of cannabis on driving-related tasks or driving simulation. In the former case, tracking-reaction time, perception, eye-hand co-ordination, and divided and sustained attention, for example, showed the highest impairment in the first hour after smoking cannabis and 1-2 hours after oral intake, but the impairment had disappeared by 3-4 hours after use (except for high doses) (Ramaekers et al 2004). "A potential disadvantage of experimental laboratory studies is that it is often unknown whether tests of skills related to driving serve as a good model for the driving task as a whole. Many tests are short and relatively simple and do not necessarily reflect performance in the real world. Driving is probably one of the most complex psychomotor tasks" (Ramaekers et al 2004 p113).

Studies using driving simulators are better, but still limited compared to closed-course driving, and "can often measure only parts of the total driving behaviour" (Ramaekers et al 2004). Driving simulation studies have found compensatory behaviour (eg: maintaining longer distance to vehicle ahead) as well as impairments (eg: hitting roadway obstacles). "However, this compensatory behaviour was never sufficient to fully overcome the overall impairing effect of cannabis" (Ramaekers et al 2004 p114). There are few closed-course driving studies (Ramaekers et al 2004).

Klonoff (1974) was the first to perform a field experiment (ie: experimental dosage of cannabis while

driving in actual traffic). Thirty-eight individuals drove for forty-five minutes in Vancouver with a driving test examiner after a placebo or 4.9 mg or 8.4 mg of oral THC. A series of field experiments have been done in the Netherlands (eg: Ramaekers et al 2000). This type of study has high ecological validity, but it does raise ethical issues in terms of the risk to other drivers.

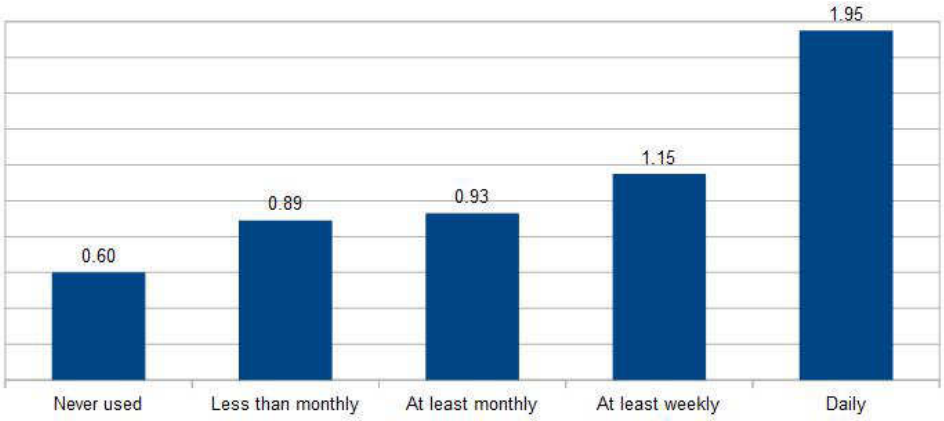
Ramaekers et al (2004) highlighted an important point:

> Experimental studies furthermore indicate that not all driving tasks are equally sensitive to the detrimental effects of THC. Performance was always worst in tests measuring driving skills at the operational level, ie: tracking and speed adjustment, as compared to performance in tests measuring driving performance at the manoeuvring level, ie: distance keeping and braking, and the strategic level, ie: observation and understanding of traffic, risk assessment and planning. Strategic and manoeuvring levels are particularly demanding of resources in that they require effortful processing and attention. Thus, processing is relatively slow and flexible. In contrast, the operational level is considered to be an automatic, routine process, which is fast and relatively inflexible. Drivers may be particularly vulnerable to detrimental effects of THC in traffic situations were they specifically employ driving skills that are operated at lower automated levels, such as during highway driving. The implication might be that drivers under the influence of THC might be more likely to be involved in specific types of traffic accidents such as single vehicle crashes. Culpability studies by definition have neglected this possibility, since drivers involved in this type of accident are practically always responsible, irrespective of drug use (p117).

APPENDIX 13C - MENTAL DISORDERS

Fergusson et al (2005) used data from the Christchurch Health and Development Study (CHDS), which is a cohort study of 1265 children born in four months in the middle of 1977 in Christchurch, New Zealand. By 25 years old 1055 participants remained on the study. At 18, 21 and 25 years old, the sample were given a comprehensive mental interview, and their cannabis use was categorised annually on a five-point scale, from non-user (1) to used on a daily basis (5). Prior history of cannabis use or psychotic symptoms were the main confounders controlled for. The CHDS database contained information about family, socio-economic factors, and the personality of the individual.

There was a statistical significant association between cannabis use and psychotic symptoms, with daily users reporting 2-3 times more symptoms than non-users (figure 13.2) (known as a dose-response relationship). After adjusting for confounding variables, this difference was reduced to 1.6-1.8 times.



(Data from Fergusson et al 2005 table 1 p362)

Figure 13.2 - Mean number of psychotic symptoms reported by frequency of cannabis use in past year at age 25 years.

In a fifteen-year prospective study of over 50 000 Swedish male army recruits, Andreasson et al (1987) found that cannabis users before age eighteen were over twice as likely to be diagnosed with schizophrenia by the end of the study. This study adjusted for a history of psychosis, and parental divorce, and the findings were confirmed at twenty-seven years follow-up (Zammit et al 2002).

"Researchers who remain sceptical about a casual explanation often argue that a causal hypothesis is inconsistent with the absence of any increase in the incidence of schizophrenia, as cannabis use has increased among young adults" (Hall 2015 p26).

It is estimated that the risk of psychosis doubles from 7 in 1000 in non-users to 14 in 1000 for regular cannabis users (Hall 2015). Put another way, one case of schizophrenia in the UK would be prevented if 4700 young men did not smoke cannabis (Hall 2015). The highest risk is always for individuals who already experience psychosis, as is the case with other mental disorders (eg: increased suicide risk among those cannabis users with pre-existing risk before use) (Hall 2015).

The relapse rate for individuals with schizophrenia, however, is higher with cannabis use (eg: Linszen et al 1994).

Two key problems exist in establishing that cannabis causes psychosis, and statistical techniques are used to deal with them (Fergusson et al 2005):

a) Uncontrolled confounding factors - Fixed effects regression models are used to combat these.

b) Reverse causality - ie: developing psychosis leads to cannabis use. Structural equation models are used to deal with this.

APPENDIX 13D - FERGUSSON ET AL (2003)

Adolescent who use cannabis tend to have lower educational grades, negative attitudes towards school, higher school absenteeism, higher rates of expulsion and drop-out, and subsequently, more unemployment than non-users (Fergusson et al 2003).

Fergusson et al (2003) offered two possible causal pathways (alone or together) for these findings:

i) The "regular use of cannabis requires that the individual develops a network of peers and contacts in order to obtain and use cannabis. It may be suggested that, as a result of contact with networks of substance-using peers and others, young people who use cannabis heavily or regularly acquire what Kandel et al (1986) have described as 'anti-conventional' attitudes. These attitudes may encourage young people to avoid making a personal investment in education and to choose an alternative lifestyle" (Fergusson et al 2003 p1682).

ii) Heavy cannabis use produces cognitive impairment or "amotivational syndrome" (Lynskey and Hall 2000) that leads to educational under-achievement.

On the other hand, cannabis use and educational under-achievement may be caused by a common factor (eg: social disadvantage). "If this were the case, the association between cannabis use and educational achievement may simply reflect the fact that the risk factors and life processes that encourage the use of cannabis may also encourage educational under-achievement" (Fergusson et al 2003 p1682).

Yet another possibility is reverse causality - educational under-achievement leads to cannabis use. Fergusson et al (2003) sought to establish the direction of causality using data from the CHDS [108]. "Evidence showing that young people who used cannabis heavily, prior to school-leaving, were at increased risk of high school dropout, would support the view that heavy cannabis use increases risks of educational under-achievement whereas evidence showing an increase of cannabis use following high school dropout would suggest a reverse causal process in which school dropout

[108] This is a longitudinal study, and this method is able to show the temporal sequence of events and development, but has the problem of sample attrition, and consequently, potential bias.

encourages cannabis use" (Fergusson et al 2003 p1682) (figure 13.3).

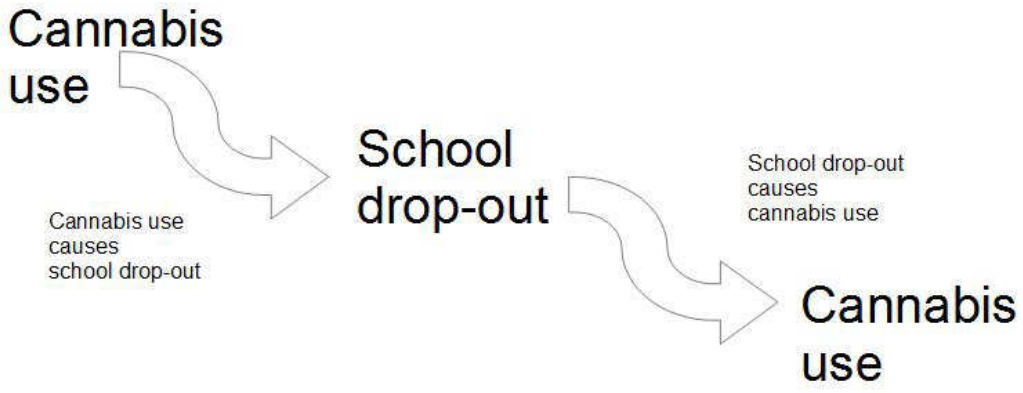
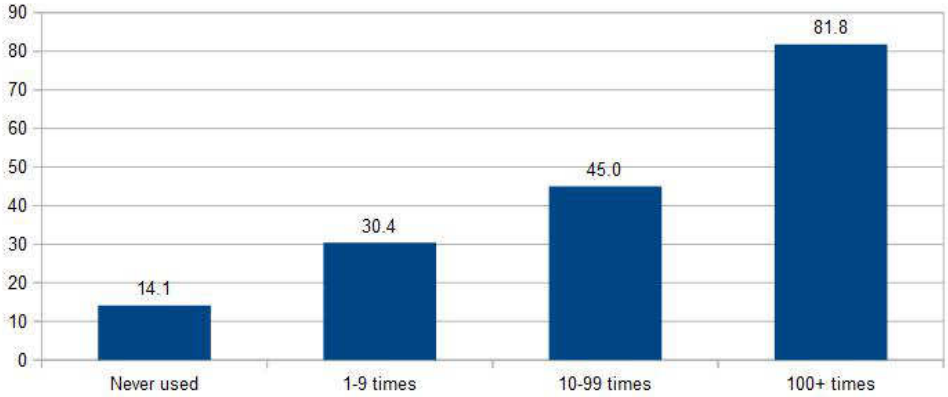


Figure 13.3 - Direction of causality between cannabis use and educational under-achievement.

Data on cannabis use annually between fourteen and twenty-five years old were amassed for around nine hundred participants, along with details of educational qualifications. Controlled confounding factors included socio-demographic information, and cognitive abilities of the individual at 8-11 years old.

Adolescents who had used cannabis on more than 100 occasions left school with no qualifications six times higher than non-users (figure 13.4). This difference was reduced to four times greater after controlling for confounding variables. There was no evidence for reverse causality after adjusting for background factors and pre-existing levels of cannabis use.



(Data from Fergusson et al 2003 table 1 p1687)

Figure 13.4 - Percentage leaving school without qualifications based on cannabis use in lifetime.

Fergusson et al (2003) confirmed the link between early use of cannabis and high school drop-out:

i) Dose-response relationship - ie: greater use and higher rate of drop-out.

ii) The relationship persisted after controlling for confounding factors.

iii) Low cognitive abilities at 8-11 years old did not predict cannabis use.

iv) The temporal sequence confirmed cannabis use before school drop-out.

Fergusson et al (2003) felt that their findings "suggests that the probable mechanism by which cannabis may influence educational achievement is through the social context by which cannabis is acquired and used, fostering attitudes and values that encourage school dropout and limit subsequent educational opportunity" (p1691).

APPENDIX 13E - MEIER ET AL (2012)

Neuropsychological impairments (eg: decline in IQ) can persist after the intoxication period and after abstinence, but the magnitude and persistence of these impairments depends on the quantity, frequency, duration, and age-of-onset of cannabis use (Meier et al 2012).

Meier et al (2012) reported a longitudinal study of 1037 individuals in Dunedin, New Zealand. Neuropsychological testing was done before cannabis use and compared to after (in 2010-12). The Dunedin Multi-Disciplinary Health and Development Study involved consecutive births between 1st April 1972 and 31st March 1973. Assessments were made at 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and 38 years old. This last assessment involved 1004 participants, and included neuropsychological tests, which were compared to scores at 7, 9, 11 and 13 years old. Cannabis dependence and use were measured at 18, 21, 26, 32 and 38 years old. The total number of days in the past year were scored (0-365), and cannabis use was categorised as "never used", "used but never regularly", "used regularly at one wave" (assessment), "used regularly at two waves", and "used regularly at three or more waves" (figure 13.5).

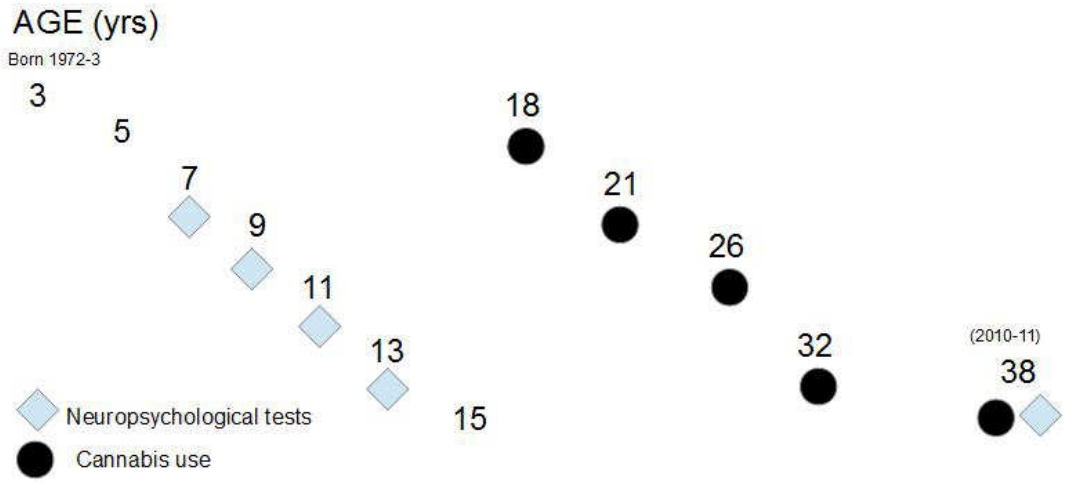
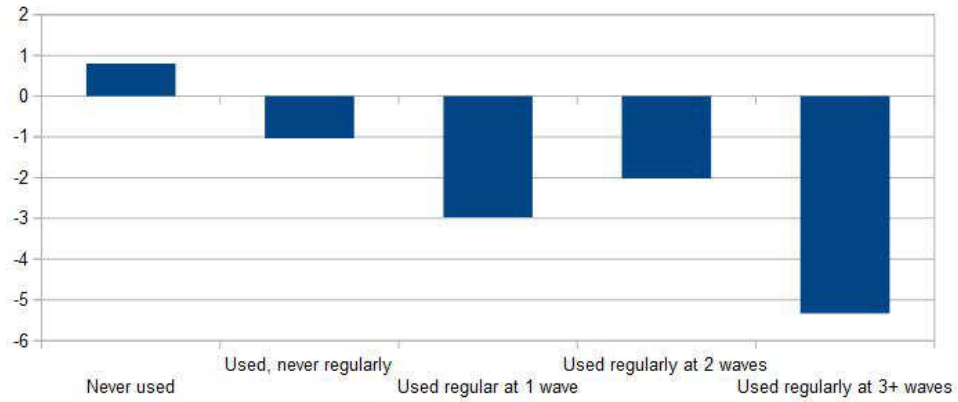


Figure 13.5 - Assessments in Dunedin Multi-Disciplinary Health and Development Study.

Six hypotheses were tested by the researchers:

1. Persistent cannabis users have greater declines in cognitive performance from childhood to adulthood than non-users (known as the "cognitive decline" hypothesis) - This was tested by comparing within-person test scores before and since cannabis use. Individuals reporting cannabis use showed declines in IQ test scores equivalent to six IQ points for the heaviest users, while non-users had a small increase in 25 years (figure 13.6).



(Data from Meier et al 2012 table 1 pE2658)

Figure 13.6 - Mean changes in IQ score between ages 13 and 38 years based on cannabis use.

2. Whether the cognitive impairment is specific or global (known as the "specificity" hypothesis - This was investigated by using multiple tests of intelligence and

cognitive abilities, including memory, and verbal comprehension. Cannabis users showed declines in all areas.

3. Persistent cannabis users show cognitive declines because they leave school earlier than non-users (known as the "education" hypothesis - Years of education were controlled for in the statistical analysis, and the decline in IQ still persisted for regular cannabis users.

4. Whether cannabis-induced neuropsychological impairments are evident in everyday life (known as the "everyday cognition" hypothesis) - Third-party informants (nominated by the participants because they "who knew them well") were asked about attention and memory problems of the individual in a mailed questionnaire at age 38 years. There were four items about attention (eg: "can't concentrate, mind wanders") and three items about memory (eg: " has problems with memory"). Significantly more problems were reported for individuals with persistent cannabis dependence. Meier et al (2012) stated: "Cognitive problems among persistent cannabis users were apparent to the 'naked eye'" (pE2661).

5. Earlier age-of-onset of cannabis use produces greater impairment (known as the "developmental vulnerability" hypothesis) - Adolescent-onset cannabis users showed greater IQ decline that adult-onset users, and the latter "did not appear to experience IQ decline as a function of persistent cannabis use" (Meier et al 2012 pE2661).

6. Whether neuropsychological declines can be reversed by abstinence or reduced cannabis use (known as the "recovery" hypothesis - Cessation of cannabis use for one year or more did restore cognitive declines for adult-onset users, but not for adolescent-onset persistent users.

The authors admitted that "although we were able to rule out a set of plausible alternative explanations for the association between persistent cannabis use and neuropsychological functioning, such as pre-morbid neuropsychological deficit and hard-drug and alcohol dependence among persistent cannabis users, our data cannot definitively attest to whether this association is causal. For example, there may be some unknown 'third' variable that could account for the findings" (Meier et al 2012 pE2661). Nor did the study show the mechanism by which persistent cannabis use was associated with neuropsychological impairment.

As with most studies, cannabis use was self-reported with no external verification. The researchers said:

"Under-reporting of cannabis use due to concerns about admitting to using an illegal substance is unlikely, however, because study members, interviewed repeatedly over 38 y about a number of illegal activities, have learned to trust the Dunedin Study's confidentiality guarantee" (Meier et al 2012 pE2662).

The generalisability of the findings is limited because of the specifics of the cohort (born in one area of New Zealand in the 1970s), and the prevalence of cannabis use is higher in New Zealand than other equivalent countries (Meier et al 2012).

APPENDIX 13F - SCIENCE

Ludwik Fleck writing in 1948 was very forward-looking: "In the near future, new, remarkable developments will appear. After blood transfusion will come organ grafts and then genetic manipulations. Even the 'surgery of the soul' is no longer a mere fantasy" (quoted in Lowy 2015a). He was concerned with the "possibility of abuses" after experiencing close at hand the Nazi medical "experiments" in Buchenwald concentration camp during the Second World War [109]. There was a debate subsequently as to whether data from such research could be used (Lowy 2015a).

Fleck linked these concerns to his analysis of the production of "scientific facts" [110], which he argued were "initially generated within a relatively homogenous thought collective that shares the same thought style: similar theoretical and practical training, agreement on legitimate scientific questions, methods that should be used to answer these questions, and criteria for the validation of results. Such agreement makes possible the collective production of knowledge, through which: 'a set of findings meanders through the community; becoming polished, transformed, reinforced or attenuated, while influencing other findings, concept formation, opinions and habits of thought' (Fleck 1935)" (lowy 2015b). It is not a question of science being the inappropriate method to study the natural world (and to a lesser extent the social world), but that science needs to be understood in a context.

It is possible that a "thought collective" can produce, what Fleck called, a "harmony of illusion" (ie: "collective production of inaccurate scientific facts"; Lowy 2015b; eg: alchemy in the past).

Natural science is different social science, though, because "an atom, a chemical compound, or an enzyme is

[109] "He survived Buchenwald mainly because he became a member of a prisoners' team that produced anti-typhus vaccine for the German army — and sabotaged this production" (Lowy 2015b).
[110] This linking has been criticised (Lowy 2015a).

indifferent to scientists' pronouncements about its nature, but a diagnosis of coronary heart disease, asthma, or schizophrenia can deeply affect the person receiving this diagnosis, via a 'looping effect' (Hacking 2002)" (Lowy 2015b).

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14. CONTAGION AND MASS SHOOTINGS

The FBI (2014) noted that mass shooting incidents had increased over the 21st century in the USA. The factors implicated in this trend are access to firearms, mental health problems, and media exposure of such incidents.

Towers et al (2015) focused on the latter factor and the idea of contagion (ie: "mass media attention given to sensational violent events may promote ideation in vulnerable individuals"). The nature of the large media attention means that the very small number of individuals likely to copycat are reached (Adee 2015).

The researchers used data from two sources (table 14.1; figure 14.1):

i) The newspaper "USA Today" on mass killings (defined as murder of at least four persons) in the USA between 2006 and 2013 (n = 232, of which 176 involved firearms). The newspaper used FBI reports, police documents, and media reports.

ii) The Brady Campaign to Prevent Gun Violence on school shootings between 1998 and 2013 (n = 188), and mass shootings (defined as three or more persons shot, but not necessarily killed) from February 2005 to January 2013 in the USA (n = 477). They used media reports.

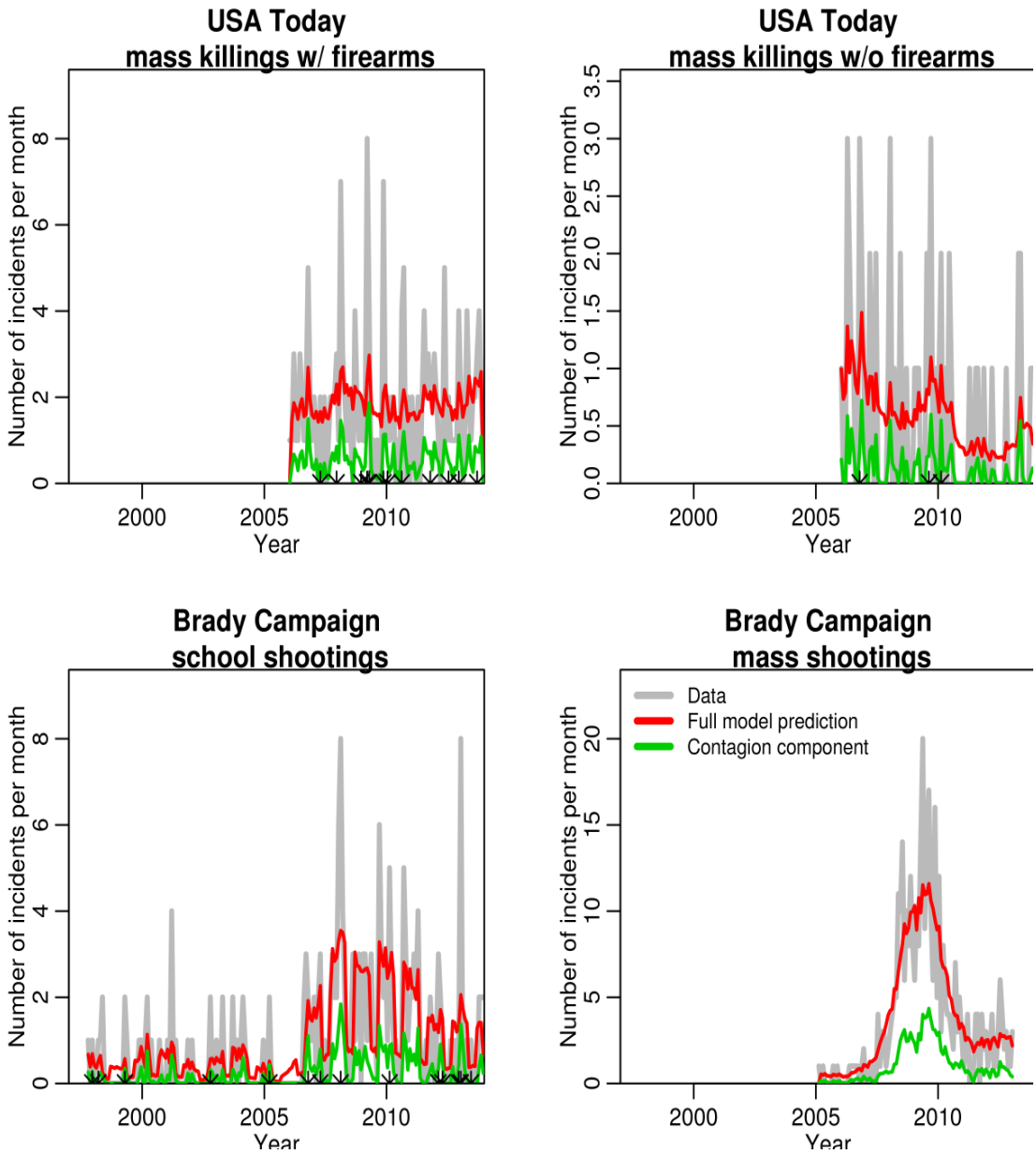
	Mass killings	School shootings	Mass shootings
Number in dataset	232	188	477
Significant association	Saturdays	September-April; weekdays	Saturday or Sunday
Killer suicide (or "suicide by cop") (%) *	46	18	17

(* Compared to 5-10% of all murders; Lester 2010)

Table 14.1 - Data used by Towers et al (2015).

The data were analysed using a self-excitation contagion statistical model, which assumes that an event increases the probability of a similar event occurring.

The data showed a mass killing on average every 12.5 days after one, and a school shooting every 31.6 days after one. Thus, another event was significantly more likely to happen in the future for mass killings and school shootings, but not mass shootings, than when no event had occurred.

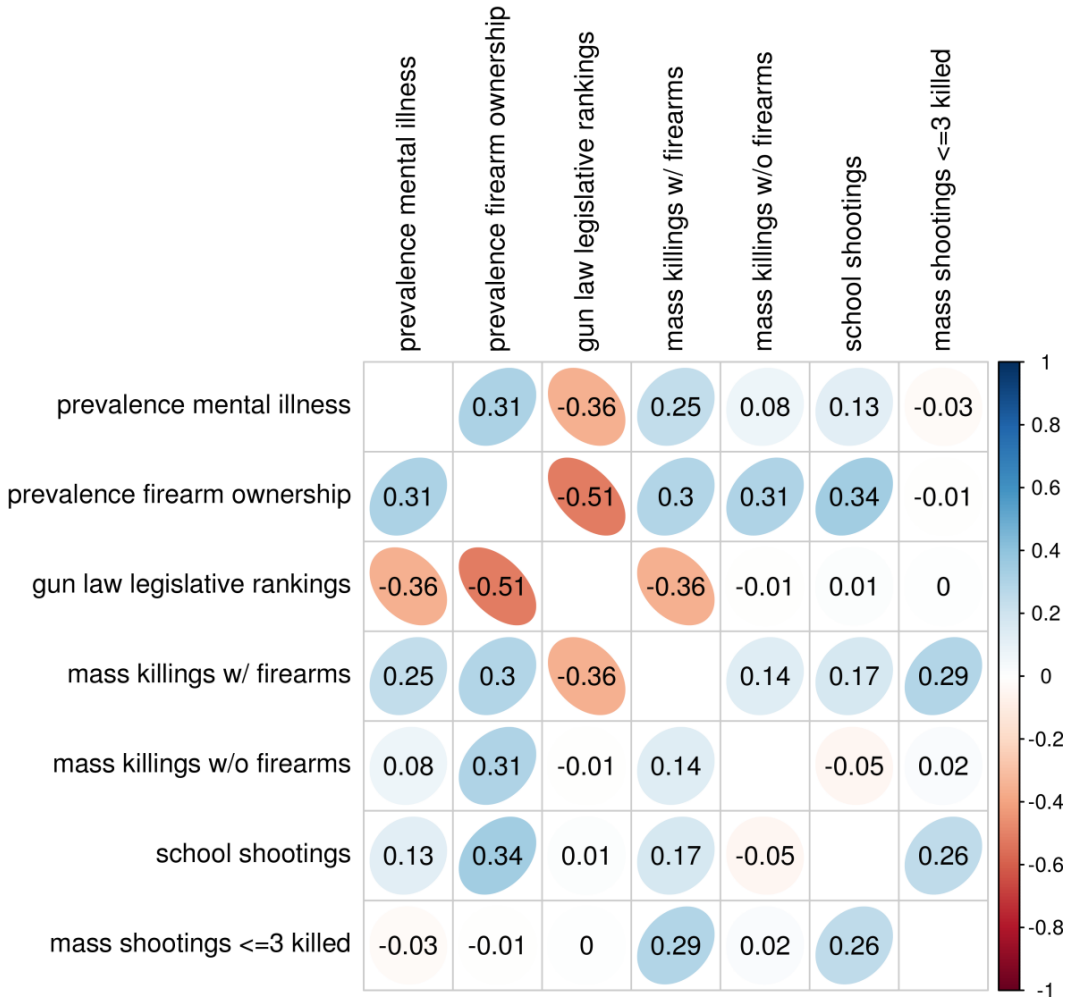


(Source: Towers et al 2015 figure 1)

Figure 14.1 - Number of events used in analysis.

In terms of other variables, the prevalence of firearm ownership by US state [111] was significantly associated, but not state prevalence of mental illness [112]. Figure 14.2 summarises the correlations between different variables.

[111] Data from Brady Campaign.
[112] Data from National Alliance on Mental Illness.



(Source: Towers et al 2015 figure 2)

Figure 14.2 - Relationship of different variables and events.

Towers et al (2015) admitted: "what our analysis tests is whether or not temporal patterns in the data indicate evidence for contagion, by whatever means. In truth, and especially because so many perpetrators of these acts commit suicide, we likely may never know on a case-by-case basis who was inspired by similar prior acts, particularly since the ideation may have been sub-conscious".

In relation to the data sources, the authors pointed out: "It is also unclear whether or not the patterns we have observed in our analysis are perhaps spuriously caused by potential unaccounted-for biases in the data samples that were compiled by private organisations. To further the understanding of the underlying root causes of these events, and to confirm whether or not contagion truly plays a role, an official comprehensive detailed, accurate, and publicly available federal database of incidents of all mass killings and school shootings in

the US is necessary. A database that includes, at a minimum, details on the background events, mental health status and access to mental health treatment of the perpetrators, exactly what kinds of weapons were used, where the perpetrators obtained their weapons, and whether they did so legally or illegally" (Towers et al 2015).

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15. PATERNALISM? TWO BRIEF EXAMPLES

15.1. PLACEBO TREATMENTS

It has been argued that prescribing a placebo treatment for a patient (ie: not as part of a clinical trial) raises an ethical problem as the patient has not given informed consent, and is thus being deceived (eg: American Medical Association). However, physicians have and do prescribe placebos, and do not see an ethical problem. For example, two-thirds of over 700 general practitioners in the UK had no problem, and 12% of them admitted to prescribing at least once in their lives (Howick et al 2013).

Glackin (2015) defended the use of placebo treatment against the charge of deception. He argued that patients do not receive complete information with any treatment because they are not told the biochemical details, for example (ie: not all information is needed for informedness). Also the doctor decides what is best for the patient, and for the patient to know. This is "a relatively innocuous, second-order or 'epistemic' form of paternalism" (Glackin 2015 p672).

A statement like, "I would like to offer you a pill which I believe can help lessen your suffering. I do not know exactly how it works. I have other pills to offer whose mechanism is clearer, but I am not sure that they will work better for you, and they may also entail more serious side effects", would be sufficient (Glackin 2015 p671).

Barnhill and Miller (2015) countered: "We accept a view of informed consent according to which informed consent requires giving patients all the information that reasonable patients would want to be given, and we assume that reasonable patients would want to be informed about whether a treatment is a placebo" (p674).

But what does a "reasonable patient" want? For example, nearly two-thirds of a Swedish population sample agreed that it was acceptable to give a placebo to a dying cancer patient (Lynoe et al 1993). While Hull et al (2013), in a US survey, found that half of respondents agreed and half disagreed with giving placebo treatment described as "a real medicine".

15.2. BICYCLE HELMETS

Making a behaviour compulsory by legislation for health reasons requires that the behaviour is shown to be beneficial. One example of this is mandatory bicycle helmet wearing by adults, as in Finland, New Zealand and Australia (Biegler and Johnson 2015). How to establish the benefits of helmet wearing over non-wearing? Three main sets of data are used.

1. Biomechanical data - Using data related to the physics of crashes, Hynd et al (2009) reported that a helmeted head can fall four times further than an non-helmeted one for the same injury.

- But laboratory-based studies of crash-test dummies.

2. Hospital admissions data - The injuries of helmeted riders are compared with non-helmeted riders for similar crashes. For example, a reduced risk of head and brain injury of about 70% for the former (Thompson et al 1996).

Heng et al (2006) found that 6% of helmeted riders and 40% of non-helmeted riders visiting an emergency department in Singapore had a head injury (eg: skull facture) (Tregouet 2015).

- But only includes those attend hospital.
- But depends on the accuracy of record kept by hospital.

3. Police reports data

- But over-representative of high impact, multi-vehicle crashes, and/or chest injuries, which helmets cannot protect against (Biegler and Johnson 2015).
- But cyclist-only crashes under-represented (Biegler and Johnson 2015).

15.3. REFERENCES

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16. ATTITUDES TOWARDS CLIMATE CHANGE

16.1. OVERVIEW

Capstick et al (2015) performed a systematic literature review covering 1980 to 2014 of articles and publications about public perceptions of climate change (CC). Fifty-seven items were found to be appropriate to include.

The researchers distinguished four phases of public attitudes during their studied time period:

i) 1980 to early 1990s - CC was beginning to feature in public perceptions of environmental risks generally - for example, in relation to the "ozone hole" in the atmosphere (ie: CC caused by ozone depletion). At this point, public understanding was "seriously at variance with the scientific models of global warming" (Kempton 1991 quoted in Capstick et al 2015).

ii) Mid-1990s to mid-2000s - Growing public concern about CC versus variability in opinion (eg: in USA, Democratic vs Republican respondents).

iii) Mid- to late-2000s - Declining public concern and growing scepticism. For example, Whitmarsh (2011) argued that the "politicisation of climate change" significantly influenced these changes, particularly in the USA, where "following the election of Barak Obama, climate change came to constitute a 'litmus test', which conservative Republicans aligning with climate sceptic positions as a means of differentiating themselves from Democrats" (Capstick et al 2015 p44).

iv) 2010s - Stabilisation of public attitudes. For example, surveys in the EU between 2009 and 2013 found that the relative importance of CC had remained consistent (Capstick et al 2015).

Measures of and changes in attitudes are influenced by certain factors (Capstick et al 2015):

1. Significant CC related events - eg: Kyoto conference on CC 1997; "Climategate" at the University of East Anglia 2009.

2. Weather - eg: unusually cold weather in late

2000s in Europe and the USA, and increased scepticism of "global warming".

3. Social and economic events - eg: association between unemployment and scepticism about CC. The "finite pool of worry" hypothesis (Weber 2010) "predicts that concern about one issue - climate change - will decrease as concern about another - in this case, economic circumstances - becomes more salient" (Capstick et al 2015 p49).

4. Design of quantitative surveys - Different measures of attitudes have been used in different surveys (eg: "seriousness" of CC, or "concern" about CC, or "certainty" of CC).

5. Cross-sectional versus longitudinal studies - The former measure attitudes at one point in time, whereas the latter can track attitude change.

Capstick et al (2015) concluded: "After a period of rising concern to around 2007, polls show a sharp decline in belief and concern across many developed countries, followed by stabilisation in some parts of the world since about 2010. While experiences of anomalous weather and other events (eg: "The Day after Tomorrow"; Fukushima; Climategate) appear to have exerted some influence on public perceptions (or, most often, perceptions of a sub-group of the population), time series analyses that take into account a range of possible explanatory factors (eg: weather, socio-economic factors, and political actions) indicate the economic downturn and political events have been particularly influential" (p51).

16.2. LEARNING FROM PSYCHOLOGY

van der Linden et al (2015) listed five insights from psychology to improve public engagement with climate change:

i) Concrete experience rather than the abstract - Statistical information about climate change (abstract) is less effective in motivating individuals to change than personal experience of, say, an extreme weather event.

ii) Importance of group norms - eg: individuals reduce their energy consumption when told that their neighbours have done so.

iii) "Psychological distance" - Events that feel distant in terms of time (ie: in the far future) or space

(eg: happening to someone else) are not integrated into current decision-making. Policymakers need to emphasise the risk to the individual here and now, and how current decisions are important.

iv) Emphasise gains rather than losses - Kahneman and Tversky (1979) found that when faced with a loss, individuals take more risks that when it is a gain. "In other words, when climate change impacts are framed as potential (ie: uncertain) losses in the distant future, whereas climate change solutions are framed as certain losses for society at present, it encourages people to conclude that maintaining the status quo may be 'worth the gamble'" (van der Linden et al 2015 p760).

v) Use intrinsic rather than extrinsic motivation - Extrinsic motivation is where external incentives are involved (like money), whereas intrinsic motivation is personal (eg: self satisfaction). Intrinsically motivated pro-environmental behaviour will be more effective in the longer term than using extrinsic rewards.

16.3. CHALLENGES

Global warming will bring a number of challenges for modern societies.

1. Rising urban temperatures - The "urban heat island effect" describes how buildings and pavements cause cities to be warmer than their surrounding areas. This increases the risk of "heat-related deaths". For example, over 700 000 deaths from conditions like dehydration, hyperthermia, heat stroke, and respiratory problems in Europe in the heatwave of August 2003, while 700 died in a five-day heatwave in 1995 in Chicago (Hoag 2015).

2. Spread of tick-borne illnesses - For example, the reported cases of Lyme disease (caused by the bacterium Borrelia burgdorferi carried by blacklegged ticks) in the USA have tripled since 1992, "although some of the increase could be due to heightened awareness" (Wenner Moyer 2015).

There is also a similar issue with vector-borne disease (eg: malaria) (Parham et al 2015).

3. Extreme drought - For example, since 2012 California has been undergoing the worst drought in a century. Though California's climate is warming, "it is not clear whether California's current drought is a temporary weather condition or is the emergence of a 'new normal'" (AghaKouchak et al 2015 p410). Anthropogenic effects (ie: role of humans) will compound the situation

with increased demand for energy.

Changes in individual water use is key, as seen with Australia's 1997-2007 Millennium Drought (AghaKouchak et al 2014).

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