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Tutorial Essays on Clinical Psychology

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

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

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1. ANTI-DEPRESSANTS FOR SCHIZOPHRENIA

Individuals with schizophrenia can show "positive" (type I) or "negative" (type II) symptoms. The former include hallucinations and delusions (similar to mania), while the latter are characterised by inactivity and flatness of emotions (similar to depression). With this last similarity in mind, anti-depressants have been used for the negative symptoms.

When prescribing a drug for any condition, it is important to establish that the drug is effective for that condition. This involves studies comparing the drug to a placebo, and after a number of studies, a meta-analysis of the pattern from the studies. Singh et al (2010) produced such a meta-analysis of studies on the use of anti-depressants with schizophrenia sufferers.

From a search of all randomised clinical trials on schizophrenia, twenty-two met the criteria for inclusion, which included a duration of illness of more than two years, randomisation of participants to drug or placebo, and standardised measures of schizophrenia severity. Because each study is slightly different in elements like duration or drug dose size (daily defined dose), direct comparison is difficult. Meta-analysis is a statistical technique that standardises the different sets of data to show the overall effects.

The total sample size from the 22 studies was 819 (416 given an anti-depressant and 403 a placebo). The trials ranged from four to twelve weeks. Anti-depressants along with anti-psychotics were found to significantly reduce the negative symptoms more than the placebo (figure 1.1). There were slight differences between the types of anti-depressants (table 1.1).

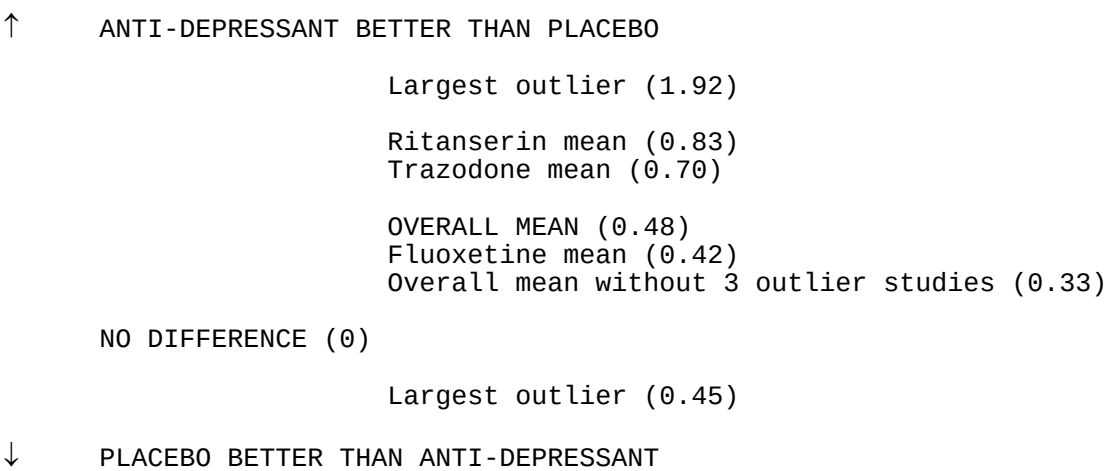

Figure 1.1 - Effect size of anti-depressants versus placebo.

TYPE OF ANTI-DEPRESSANT	NUMBER OF STUDIES SHOWING ANTI-DEPRESSANT BETTER THAN PLACEBO	NUMBER OF STUDIES SHOWING PLACEBO BETTER THAN ANTI-DEPRESSANT	NUMBER OF STUDIES SHOWING NO DIFFERENCE
Citalopram £ eg: Cipramil	1	-	-
Fluoxetine £ * eg: Prozac	4	-	-
Fluvoxamine £ eg: Faverin	2	-	-
Mianserin ^	1	-	1
Mirtazepine > eg: Zispin	3	1	-
Paroxetine £ eg: Seroxat	1	-	-
Reboxetine < eg: Edronax	2	1	-
Ritanserin ! *	2	-	-
Sertraline £ eg: Lustral	-	-	1
Trazodone ! *	2	-	-

(* = combined studies showed significant difference; & = tricyclic anti-depressant; ^ = tetra-cyclic; £ = selective serotonin re-uptake inhibitors; > = selective serotonin and noradrenaline re-uptake inhibitors; < selective noradrenaline re-uptake inhibitor; ! = other [1])

Table 1.1 - Studies of types of anti-depressants found by Singh et al (2010).

Though anti-depressants and anti-psychotics together were found to be effective, there is also the risk of an increased side-effect burden, unwanted drug interactions and consequent non-adherence, and higher cost (Singh et al 2010).

The meta-analysis was faced with two common problems with the method:

i) Publication bias - Negative studies (ie: placebo better than drug) or those finding no difference tend not to be published by pharmaceutical companies and researchers. So it is not clear how many such studies exist, and a large number of them would reduce the size of the effect. This is also called the "file-drawer problem".

ii) Outliers - Meta-analysis calculates the

[1] British National Formulary 50 (September 2005).

standardised mean difference, and so it is affected by outliers (individual studies showing very large effects).

REFERENCE

Singh, S.P et al (2010) Efficacy of anti-depressants in treating the negative symptoms of chronic schizophrenia: Meta-analysis British Journal of Psychiatry 197, 174-179

2. MYSTERY SHOPPING AND PSYCHIATRIC SERVICES - ROSENHAN RIDES AGAIN?

The "mystery shopper" idea is commonly used in the retail and service industries. This is where individuals pretend to be customers in order to assess the quality of the service or obtain information for research purposes (Walker and George 2010). It is now being applied to the health service, where individuals are instructed to use the service pretending to be patients and acting out a particular scenario.

Mystery shopping (MS) involves deception, which is particularly a problem when applied to the health service, even if it is less of an issue in the retail industry. Walker and George (2010) were concerned particularly about MS in the evaluation of psychiatric services. "One could, for example, test the effectiveness and quality of care provided by a drug treatment service by employing mystery shoppers to approach the service pretending to have a problem with drug misuse. They could then carry out their evaluation of the service as they proceed as patients through their assessments by the doctor and other relevant professionals. This whole process may involve several episodes, each of which may last for some time. At the end of the whole process the pretend patients would then report back on their experience of the service. These reports could then be used, along with other forms of evaluation, to plan improvements to, or implement changes in, the drug treatment service" (p121).

In this scenario the deception is more extensive involving the pretence of being a patient over a period of time. Such "pseudo-patient" behaviour is similar to Rosenhan's (1973) classic study (table 2.1) in which he and some of his psychiatry students pretended to be hearing voices in order to be admitted to psychiatric hospitals.

- AIMS: To get individuals, who were not mentally ill admitted to psychiatric hospitals, and for them to observe the behaviour of the staff towards the patients.

- METHOD: Eight "pseudo-patients" (Rosenhan and his students) between 1969-72 approached 12 psychiatric hospitals reporting that they were hearing voices that said "empty", "dull" or "thud". Otherwise, they told the truth (except for a pseudonym), and acted normally. All were admitted to a psychiatric hospital, and 95% were diagnosed as schizophrenic.

- RESULTS: The "pseudo-patients" recorded the behaviour of the staff towards the patients, and found an average of 6 minutes per day of interaction between the staff and patients. Only 2% of the staff paused to chat with the patients. The patients were made

powerless, and depersonalised or dehumanised as a condition not a person. Rosenhan called the hospitals "storehouses for the unwanted". 35 of 118 patients voiced suspicion that the "pseudo-patients" were not really mentally ill, but none of the staff. Eventually, the "pseudo-patients" were released with "schizophrenia in remission" still on their records (ie: the psychiatrists would not admit they were wrong).

- CONCLUSIONS: Two main conclusions are drawn from this study: (i) that the staff paid little attention to the patients, and (ii) the staff were not able to recognise a "pseudo-patient" from a real patient.

(Source: Brewer 2001)

Table 2.1 - Details of Rosenhan (1973).

Two questions were asked of the Rosenhan study, and they are applicable to MS of psychiatric services (Walker and George 2010):

a) Is deception the only way? When people know they are being observed and evaluated, their behaviour changes, and so it seems covert observation is required.

b) When does the end justify the means? The end is usually seen as the evaluation of the service in order to improve it. However, in Walker and George's scenario, the pseudo-patient is taking up resources and time that is denied to a "real" patient.

Furthermore, there is an undermining of trust and the effect upon the clinician in discovering, after the event, that a patient was pretending. A downside of the Rosenhan study was that, subsequently, 41 individuals suffering from a mental disorder were refused access to the hospitals as fakes (Brewer 2001). The hospitals did not want to get caught out again.

Walker and George (2010) concluded that "mystery shopping should only be used where independent scrutiny concludes that the importance of the aims of the evaluation is sufficient to justify the deception used" (p122).

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3. SOME REFLECTIONS ON GENETIC EXPLANATIONS OF BEHAVIOUR, AND SCHIZOPHRENIA

3.1. INTRODUCTION

When the first draft of the human genome was announced in 2000 by the Human Genome Project [2], publicity claims were made about finding the causes (and cures) for many diseases within ten years. But a decade later, such "medical promises" have not been kept (Hall 2010). Though, saying that, there has been undoubted progress, including a "gold rush" to find genes linked to disease and behaviour (Dupuis and O'Donnell 2007) [3].

One issue revolves around the theory of how genes cause disease (or behaviour). The main view has been the "common variant" hypothesis. Put simply, this approach looks for genetic variations between healthy and diseased individuals, and the difference must explain the disease. The focus is upon particular locations on the chromosomes (known as single-nucleotide polymorphisms; SNPs) where nucleotide pairs differ between individuals (ie: DNA "letters": A, C, T and G) [4]. Genome-wide association studies (GWAS), using the knowledge from the Human Genome Project, sought to find common variants. Many SNPs have been found related to many diseases, including schizophrenia, but the genetic differences only account for small variations in the heritability of diseases (Hall 2010). McClellan and King (2010) argued that searching for common variants in GWAS has limitations [5].

An alternative approach is that the relationship

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[2] Further details were reported in the International Human Genome Sequencing Consortium (2004) and the International HapMap Consortium (2005).

[3] Prior to the mapping of the human genome, the search for candidate genes (ie: those responsible) was limited to linkage and association studies. With linkage studies, families containing large numbers of sufferers are studied. A comparison is made, genetically, between sufferers and non-suffereres within the genetic family. Association studies compare sufferers and non-sufferers, but focus on specific areas of the genome known or suspected to be involved.

[4] With "copy number variants" the focus is upon a strand of DNA that may be different in sufferers due to duplication (repeat of a sequence) or deletion (sequence missing) (Williams et al 2009).

[5] For example, there is always the possibility of a false discovery (an apparent significant genetic association with a disease or behaviour). Thus the importance of the replication of studies (Dupuis and O'Donnell 2007).

between genes and diseases and behaviours is "heterogeneous". This means that "many different mutations in many different genes can produce the same disease" (Hall 2010).

The focus of these two approaches is upon differences in genes, but epigenetics has shown that the same genes may behave differently depending on chemical "tags" in the DNA. Epigenetics is another layer of understanding about inheritance.

3.1.1. Use of Animals

Many experiments to isolate the effect of particular genes use animals, and, in particular, genetically engineered ones (eg: transgenic or knockout mice). In such experiments the change in the gene (or genes) is viewed as the independent variable, with all other variables being held constant, leading to the change in behaviour (dependent variable).

But the other variables are not held constant as "different laboratories commonly employ their won idiosyncratic version of behavioural test apparatus and protocols, and any laboratory environment also has many unique features" (Crabbe et al 1999 p1670). For example, two laboratories found contradictory findings on the same transgenic mice's response to ethanol (Crabbe et al 1999).

Crabbe et al (1999) tested six mouse behaviours in three different laboratories in North America using the same inbred strains and a different strain. All variables related to the mice were the same including housing, feeding, shipping from suppliers, testing regime, and age. Crabbe et al (1999) noted that "despite our efforts to equate laboratory environments, significant and, in some cases, large effects of site were found for nearly all variable" (p1670). For example, in response to the same dose of cocaine, the same inbred mice were significantly more active at one laboratory.

Crabbe et al (1999) urged caution: "For behaviours with smaller genetic effects (such as those likely to characterise most effects of a gene knockout) there can be important influences of environmental conditions specific to individual laboratories, and specific behavioural effects should not be uncritically attributed to genetic manipulations such as targeted gene deletions" (p1672).

3.1.2. Genetics and "Race"

The Human Genome Project has increased the knowledge about human genetics, and has intensified the focus on

genetic variation among groups. The question is whether the differences can be grouped together in terms of "race". In other words, are genetic "races" the same as socially constructed ones based on skin colour etc? The answer is generally "no", but there is debate about racial categories and genetic data. For example, "Hispanic" is a single social category which hides the diversity of individuals within this group (Lee et al 2008).

Between 2003 and 2005 at Stanford University, California, scholars in different fields came together to discuss such issues (Koenig et al 2008). Subsequently, a series of statements (guiding principles) were devised, which included the following ideas (Lee et al 2008):

i) There is no scientific basis to claims of genetic support for racial superiority or inferiority.

ii) There is more genetic variation between individuals within the same socially constructed race as between races. However, migrations though human history have led to patterns of genetic variations linked to geographical areas.

iii) "We caution against making the naive leap to a genetic explanation for group differences in complex traits" (eg: intelligence) as the "contribution of any one gene to normal variation is small and these traits may be more fully explained by variation in environmental factors" (Lee et al 2008 p2).

Genetic "race" is also different to ethnic identity which "arises from a sense of shared difference and is expressed by the maintenance of fundamental, constantly shifting cultural symbols deemed essential to the survival of the group" (King 1999).

Ethnic identity among a minority group is influenced by many factors. King (1999) used the example of "Native Americans" in North America where the name itself can be disputed ("Indian", "Indian people"). Not only does the group have real historical events of victimhood (eg: forced removals in the nineteenth century - Modocs to Oklahoma in 1873), but also symbolic ones (eg: naming of products - "Washington Redskins" (American football team), "Apache helicopters"). Thus "Ethnicity, in multi-ethnic societies, is often situation-specific, multi-layered to ensure acceptability on varied occasions" (King 1999).

3.2. SCHIZOPHRENIA

Family and twin studies show a greater risk of

schizophrenia among relatives of sufferers than in the general population. This suggests that the disorder is inherited. But the question is how, in terms of the specific genes involved. It could be due to the interaction of several relatively common versions of genes (alleles) or specific rare alleles (Williams et al 2009). Another question is what do the altered genes do. They are likely to be related to brain development in specific ways, like normal functioning of neurons and synapses.

Modern genetic techniques and computing allows for large-scale analysis that has never been possible with twin and family studies in the twentieth century [6]. Here are three large-scale genetic studies that are ongoing.

1. SGENE-plus study [7].

Stefansson et al (2009) found 314 868 SNPs in a genome-wide scan of 2663 schizophrenic cases and 13 498 controls in eight European locations. Statistical techniques highlighted risk alleles on chromosome 6 related to the major histocompatibility complex (MHC) (immune system function), and on chromosomes 11 and 18 related to brain development, memory and cognition.

What this type of research also showed was that variants in a single gene can lead to a range of conditions. For example, a mutation in the gene on chromosome 18 (known as TCF4) can produce, at the extreme, Pitt-Hopkins syndrome (with severe motor and mental retardation), or be involved in schizophrenia.

2. International Schizophrenia Consortium study [8].

The International Schizophrenia Consortium (2009) sampled over one million SNPs from 3322 individuals with schizophrenia and 3587 controls in Europe. They found polygenic variations involving thousands of common alleles (ie: multiple genetic variations involved). Some of these alleles were shown with bipolar disorder sufferers, but not other mental disorders.

3. Molecular Genetics of Schizophrenia study [9].

Using genetic data from 8008 schizophrenic cases and

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[6] Details of studies at SZGene database (http://www.schizophreniaforum.org/res/sczgene/default.asp).
[7] Details at http://www.sgene.eu/Summary.php.
[8] Details at http://pngu.mgh.harvard.edu/isc/.
[9] Details at http://clinicaltrials.gov/ct2/show/NCT00006418.

19 077 controls from the USA, Shi et al (2009) confirmed that schizophrenia is associated with the MHC region of chromosome 6.

Modern genetic techniques applied to schizophrenia have "provided strong evidence that common alleles are actually involved in the disorder, and moreover, evidence that there are many of these, almost certainly hundreds and probably thousands" (Williams et al 2009 p71).

However much evidence is being collected about the genetic basis of schizophrenia, there are still limitations to bear in mind. The findings of these large-scale studies have "little value for individual risk prediction, meaning that application to clinical genetic testing for schizophrenia would be unwarranted" concluded the International Schizophrenia Consortium (2009 pp750-751). Furthermore, they said: "measures of polygenic burden, along with known risk loci and non-genetic factors such as season of birth, life stress, obstetrical complications, viral infections and epigenetics, could open new avenues for studying gene-gene and gene-environment interactions" (p751). Thus schizophrenia is caused by a combination of genetic predispositions and environmental factors.

Overall, there are two key issues related to genetic explanations of schizophrenia:

i) In evolutionary terms, genes that produce disorders that reduce fecundity (ie: presence and number of offspring) would be "phrased out" over subsequent generations (Williams et al 2009).

ii) The problem of diagnosing schizophrenia, particularly as there is no laboratory tests for it as there are with many diseases, and so diagnosis is based on "clinical grounds" (symptoms and signs) (Williams et al 2009).

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4. SHYNESS IN ARAB COUNTRIES

Abd Al Rasak et al (2006) distinguished between a culturally acceptable shyness that does not cause distress ("physiological shyness") and a problematic version linked to other mental disorders like social phobia ("pathological shyness") among students in two Arab countries. Seventy-six volunteers from Egypt studying medicine or literature, and 76 science and health students from Saudi Arabia were recruited.

Each participant completed Arabic versions of questionnaire about shyness, fear of negative evaluation, social phobia, avoidant personality disorder, and general mental health.

Overall, 15% of the total sample were categorised as "pathological shyness" (9.8% in Egypt and 18.4% in Saudi Arabia), and the reminder as "physiological shyness". The former group were based on high scores on the social phobia scale and the presence of avoidant personality disorder. Table 4.1 compares the prevalence with some other countries.

SOCIAL PHOBIA	• Egypt 13.9% (students) (2006 data) • Saudi Arabia 18.4% (students) (2006 data) • Canada (all ages) 3% (past year), 8% (lifetime) (2002 data) • Korea (all ages) 0.5% (lifetime) (1990 data) • Taiwan (all ages) 0.6% (lifetime) (1989 data) *
AVOIDANT PERSONALITY DISORDER	• Egypt 5.9% (students) (2006 data) • Saudi Arabia 11.8% (students) (2006 data) • USA (all ages) 2.36% *

(* all figures quoted in Abd Al Rasak et al 2006)

Table 4.1 - Comparison of rates of social phobia and avoidant personality disorder.

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5. EXPRESSED EMOTIONS AND RELAPSE

A social cause for mental illness is hotly debated because of the difficulty of establishing cause and effect. However, relapse can be predicted from the family behaviour. Brown et al (1972) showed that the expressed emotion (EE) at the time of admission to hospital by the relative with whom a schizophrenic lived predicted relapse of the schizophrenia within nine months of discharge from hospital.

EE was operationalised with three components to produce high EE or low EE:

- Number of critical comments made by relative when talking about the sufferer and the illness.
- Hostility towards the sufferer.
- Emotional over-involvement - excessive anxiety, over-concern, or over-protectiveness towards sufferer.

It was found that 58% of schizophrenics from high EE homes (ie: greater critical comments, hostility, and over-involvement) relapsed compared to 16% from low EE homes ($p<0.001$).

Vaughn and Leff (1976) replicated the Brown et al (1972) study using 37 individuals with schizophrenia and thirty with "neurotic depression" [10] in three hospitals in south-east London. Between 48-50% of the schizophrenics experiencing high EE relapsed within nine months out of hospital compared to 6-12% in the low EE group (figure 5.1) [11].

For individuals from high EE homes, drug treatment aided in reducing relapse, but had no affect for low EE homes (figure 5.2).

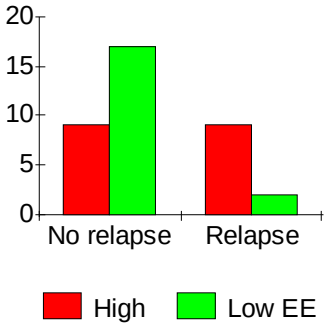

Figure 5.1 - Number of individuals with schizophrenia and relapse based on EE.

[10] This category does not exist today in the latest classification systems of mental disorders.

[11] The figures varied depending on the cut-off point for high and low EE (6+ or 7+ critical comments).

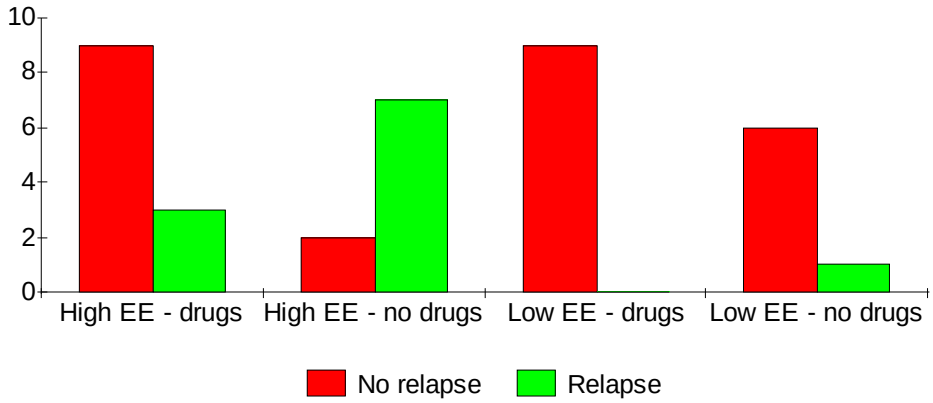

Figure 5.2 - Number of individuals with schizophrenia and relapse based on EE and drug treatment.

For depressed individuals, relapse rates were significantly higher in the high EE group (67%) than the low EE one (22%).

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6. THE URBAN ENVIRONMENT AND PSYCHOSIS

Psychosis is more common in urban than rural populations [12]. Is this because aspects of the urban environment cause psychosis, or do individuals with psychosis migrate to the city? This is not an easy question to answer.

Ellett et al (2008) attempted to establish a causal link between the urban environment and psychosis in a field experiment. Individuals with persecutory delusions were exposed to a busy inner London high street for a short period. It was predicted that this exposure would make them anxious, more negative about themselves and others, and increase the "jumping to conclusions" reasoning bias (Garety and Freeman 1999).

Thirty individuals with current persecutory delusions as part of their psychosis were recruited. These delusions included "other people are trying to damage my eyes", "there is a conspiracy against me - others might attack me", and "the police and government are spying on me and are out to get me". The participants were randomised to the experimental condition which involved walking accompanied down a busy shopping street in Camberwell (south London) for ten minutes, or a mindfulness condition (mindfulness relaxation in a quiet room for ten minutes) (figure 6.1). Thirty age and IQ matched non-psychotic individuals were a comparison group for the questionnaires.

1. Pre-experiment measures

- Positive and Negative Syndrome Scale (PANSS) (Kay 1991): 30 items about schizophrenia rated over the past three days on a seven-point scale.
- Psychotic Symptoms Rating Scale (PSYRATS) (Haddock et al 1999): 17 items related to delusions and hallucinations over the last week.
- Measure of IQ.
- Subjective rating of distress: "How anxious are you feeling right now?" and "How much are you feeling under threat from others?" (rated 0-10).

2. Randomisation

Clinical participants		Control participants
↓	↓	↓
Urban exposure	Mindfulness	Questionnaire completion

[12] Eg: rates of schizophrenia per 10 000 population: 2.5 in rural Scotland vs 3.0 in inner city south London (Brewer 2001).

3. Post-experiment measures

- Subjective rating of distress (as above).
- Brief Core Schema Scales (BCSS) (Fowler et al 2006): 24 items rated on a five-point scale on beliefs about the self and others.
- Brief Fear of Negative Evaluation Scale (BFNE) (Leary 1983): 12 items measuring the fear of receiving a negative evaluation on a five-point scale.
- Probabilistic reasoning task (individuals with delusions ask for less information to solve it - ie: "jumping to conclusions" bias).
- Beck Cognitive Insight Scale (Beck et al 2004): 15 items about insight on experiences (on four-point scale).
- State Social Paranoia Scale (SSPS) (Freeman et al 2007): 10 persecutory thoughts; eg: "someone stared at me in order to upset me".

Figure 6.1 - Details of the experiment.

Exposure to the urban environment for the clinical participants lead to significant increases in anxiety and paranoia, while the mindfulness group showed reductions (figure 6.2). Urban exposure also produced a more negative view of others on the BCCS, and greater "jumping to conclusions" bias on the probabilistic task. There were no significant differences between the clinical participants on the other measures.

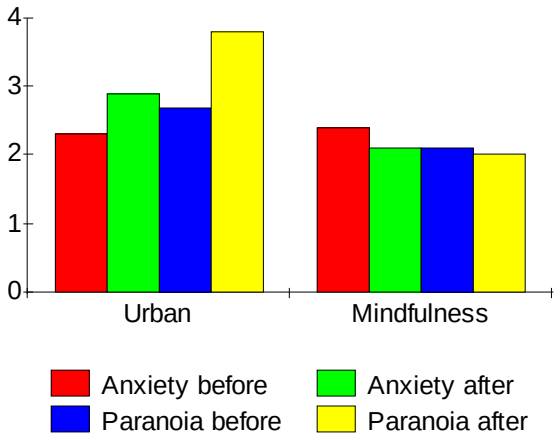

Figure 6.2 - Mean scores on anxiety and paranoia.

Evaluation of Ellet et al (2008)

1. This is a novel study of the effect of the urban environment on individuals already suffering from psychosis.

2. Though it was designed as a field experiment to test the effect of the urban environment, the study was a

quasi-experiment because the participants could not randomised to the experimental or control group (ie: the clinical participants were always in the experimental groups). This limits the ability to establish the causal relationships in the study.

3. The urban environment involves many variables, like sounds and sights, and it is difficult to isolate which variables were involved [13]. Laboratory experiments focusing on the individual aspects of the urban environment could do this.

4. The control group did not experience the experimental conditions. It would be used to see if their ratings on the post-experiment questionnaires changed after urban exposure.

5. Most of the questionnaires were self-rated, though they were standardised, psychometric questionnaires with established reliability and validity.

6. The urban exposure and mindfulness conditions were very different. A better comparison could have been between the busy urban environment and when it is quieter.

7. All the clinical participants had a diagnosis of non-affective psychosis (schizophrenia, delusional disorder, schizo-affective disorder), were 18-65 years old, and did not have alcohol or substance dependency or learning disabilities. The two experimental conditions contained clinical participants similar on demographic variables, like age, length of illness, and number of psychiatric hospital admissions.

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[13] Eg: high density of people, ethnic mix, or behaviour of others.

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7. THE GOOD SIDE OF DEPRESSION

Evolutionary explanations emphasise that behaviours which are evolutionary adaptive are the ones that pass from generation to generation. In the case of humans, it is behaviours that were evolutionary adaptive when humans first evolved as a species, not necessarily evolutionary adaptive today. In other words, behaviours that were advantageous in the "environment of evolutionary adaptation" [14].

Depression could be one such behaviour that gave evolutionary advantages: "a state of mind that brings real costs but that also brings real benefits" (Andrews and Thomson 2010) - namely, becoming more analytical and focused. Individuals with depression tend to think about their problems endlessly. This is seen as dwelling on the problem and as negative. But if this process is focused problem-solving, then it is a positive thing. Andrews and Thomson (2009) called it the "analytical rumination (AR) hypothesis" for depression. They observed that the fact that a large number of individuals suffer from depression at some point in their lives may suggest that it is "normal psychological functioning".

Andrews and Thomson (2009) made the following claims about the AR hypothesis:

i) Complex problems linked to evolutionary fitness-related goals trigger depression (ie: survival, reproductive). These are manifest today as depression over loss of a job or end of a romantic relationship.

ii) Depression produces changes in the body that promote rumination (eg: activates parts of the brain that enhance attention and focus).

Depression co-ordinates changes in the body that help the individual to focus on the problem-solving. For example, the desire for social isolation, and the loss of pleasure from normal activities.

iii) Depressive rumination aids problem-solving. For example, in a financial and economic games, Au et al (2003) found that participants whose mood was manipulated to be sad (with negative false feedback) made better decisions than happy or neutral participants.

iv) Depression is similar to fever, which is an

[14] "Environment of evolutionary adaptation" is not an easy concept to define: at one level, it is the hunter-gatherer lifestyle and environment of early humans, but, at another, it is "an abstract concept that attempts to put together the range of selective pressures that have given rise to human characteristics" (Foley 2002 p307).

evolutionary trade-off to aid the immune system. The cost is that the individual is immobile in bed with the fever, and the benefit is that the immune system has the resources to deal with the invading virus.

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8. HOARDING BEHAVIOUR

Hoarding behaviour (or "pathological collecting") is "the acquisition of, and unwillingness or inability to discard, large quantities of seemingly useless objects" (Samuels et al 2008 p836), or "unable to discard worn-out or worthless objects even when they have no sentimental value" (APA 1994). At the extreme, the clutter limits living space in the home, increases the risk of injuries from fire and falling, and of illnesses from poor sanitation (Samuels et al 2008).

Hoarding behaviour may have a biological basis as shown by the following evidence (Samuels et al 2008):

i) Case studies of hoarding behaviour developing after brain injury.

ii) Differences in brain activity in neuroimaging studies between individuals with obsessive-compulsive disorder (OCD) who do or do not hoard.

iii) Hoarding behaviour among different members of families with OCD.

iv) Hoarding behaviour by sufferers of genetic conditions like Prader-Willi syndrome and velocardiofacial syndrome.

On the other hand, there may be environmental causes to the behaviour - early material deprivation (and fear of future financial insecurity), and traumatic life events as children. For example, Hartl et al (2005) found that hoarders were more likely to have had something taken by force as a child, or have been forced to engage in sexual activity as a child or adult compared to non-hoarders.

Samuels et al (2008) investigated the prevalence of hoarding behaviour using participants in the Hopkins Epidemiology of Personality Disorder Study based in Baltimore, USA. The Hopkins study interviewed 3481 adults in east Baltimore in 1981-2, and 1920 of them were re-interviewed between 1993 and 1996. Samuels et al used 735 of them, who were asked: "Do you find it almost impossible to throw out worn-out or worthless things? If so, is that true even when they don’t have any sentimental value? Give me some examples. Is this a problem for you or for others? If so, tell me about it" (p838). Interviewers rated the presence of hoarding behaviour based on the responses on a scale of 0-2.

Twenty-seven individuals (3.7%) were as "2" on the scale ("pathological hoarding"). This figure was

calculated as 5.3% after adjustments for sampling representativeness. Table 8.1 give two examples of answers given by sufferers.

- "one participant, a 49-year-old man, said that 'My room is like a bomb hit it. I've got books and papers, stuff in the corner there. I don't want to throw nothing away. Old suits in my closet, I know I'll never wear again in my life. Old beat up tennis shoes, think I'll find a use for them. I never throw a book away. I like to keep articles, the whole paper; it starts building up on me in a hurry. Newspapers knee-high. I keep a whole drawer full of rubber bands; don't know why, but I do. Lots of junk'. Another participant, a 41-year-old woman, noted that she has saved 'old clothes from the 1970s, piled up clocks, iron, tiny television, picture frame, fans. My house sometimes looks like a junk shop. I argue with my fiance over throwing things away; he wants to get rid of all my good stuff; to me it's good stuff, to him it's junk. Stuff I've had for years, reminds me of my mother.. you know I'm not going to throw that away'" (Samuels et al 2008 p839).

Table 8.1 - Two examples of hoarders.

Hoarders were more likely to be older (than 55 years), male, not married or co-habiting (ie: living alone), not currently employed, and having lower income (figure 8.1).

	1			
AGE	34-44	45-54 (1.28)	55-94 (2.88)	
GENDER	female	male (2.22)		
MARITAL STATUS	married/ co-habit	separated/ divorced (1.14)	never married (1.76)	widowed (2.4)
LIVES ALONE	no	yes (1.50)		
CURRENTLY EMPLOYED	yes	no (1.98)		
HOUSEHOLD ANNUAL INCOME	>$49 999	$20 000 - 49 999 (2.89)	<$20 000 (4.51)	

(1 = base)

Figure 8.1 - Odds ratios for hoarding behaviour.

In terms of psychiatric problems, hoarders were over four times more likely to have had alcohol dependence in their lifetime and twice as likely to have it now. There was a greater risk of having any personality disorder, but, perhaps surprisingly, no evidence of obsessive-compulsive disorder (though obsessive-compulsive

personality disorder was more common).

Support for environmental influences on the development of hoarding behaviour came with the finding that sufferers reported significantly more often in childhood the following: either parent having psychiatric problems, experiencing home break-ins, and excessive physical discipline. Parental psychiatric problems was a stronger link to hoarding behaviour in women than in men (table 8.2).

	TOGETHER	MALE	FEMALE
PATERNAL	2.69	1.23	6.97
MATERNAL	2.73	1.91	4.68

Table 8.2 - Odds ratios for hoarding behaviour based on parent having psychiatric problems.

How do the findings of this study compare to previous research? Table 8.3 gives some examples of similarities and differences with other studies.

SIMILARITIES

- Sufferers more likely older/lower income
- Sufferers more likely alcohol dependence

DIFFERENCES

- Prevalence rate higher (vs eg: 0.4%)
- More men suffering than women (vs opposite)

(Steketee and Frost 2003; Wheaton et al 2008)

Table 8.3 - Similarities and differences between Samuels et al (2008) and previous studies.

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9. TOPIRAMATE AND ALCOHOL DEPENDENCE

Topiramate was originally designed as an anti-epileptic drug which reduces dopamine in the brain. It has also been used to treat alcohol dependence.

Johnson et al (2007) performed a fourteen-week trial of topiramate against a placebo at seventeen sites in the USA with 371 individuals diagnosed with alcohol dependence. For inclusion, the individuals had to be drinking more than 35 "standard drinks" per week for men, and 28 for women [15], and have a negative urine test for recreational drugs. All participants had expressed a wish to reduce their drinking.

There were fifteen exclusion criteria including having other mental disorders than alcohol dependence, taking psychotropic drugs, or were pregnant or lactating.

Baseline assessments were made prior to randomisation to the drug or placebo, and then weekly measures were made up to the end of the study (14 weeks). The participants were also given a manual to aid the adherence to treatment.

The primary outcome measure was the number of self-reported heavy drinking days (ie: five or more standard drinks per day for men and four for women). Other measures included self-reported non-drinking days. Physiological measures were also taken (eg: blood alcohol level).

At the end of 14 weeks, 256 individuals had completed the study (figure 9.1) [16]. It was found that topiramate was significantly better at reducing the number of heavy drinking days between baseline and week 14 than the placebo, and the number of abstinence days significantly increased (figure 9.2).

Johnson et al (2007) admitted three weaknesses with their study:

i) Side effects leading to study drop-out.

ii) Detailed inclusion and exclusion criteria produced participants who were healthier and more homogeneous than the general population of alcohol dependence.

iii) The study was relatively short with no longer term follow-up to see if relapse occurred.

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[15] A standard drink = 0.5 oz of absolute alcohol = 10 oz of beer = 4 oz of wine = 1 oz of 100-proof liquor (Johnson et al 2007).

[16] Drop-outs were assessed as relapse to the baseline.

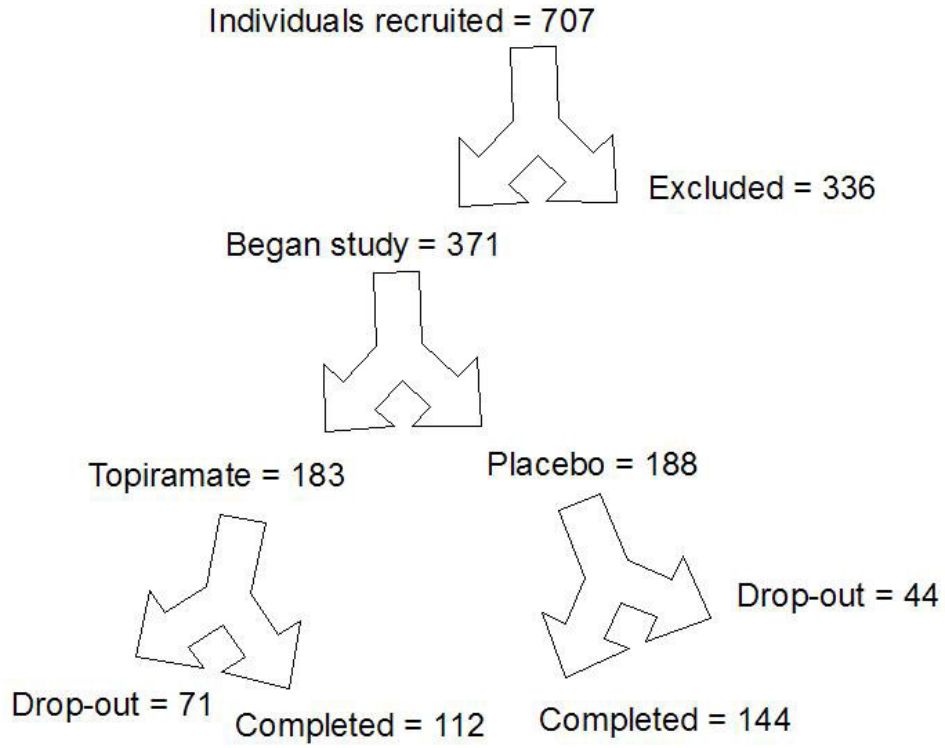
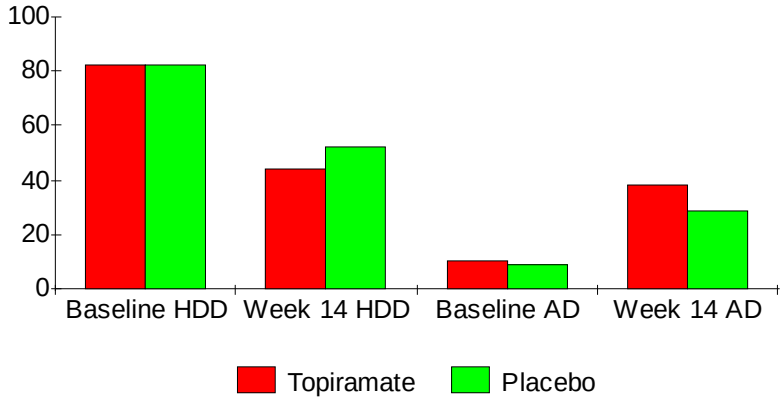


Figure 9.1 - Stages of Johnson et al (2007) study and number of participants.



HDD = heavy drinking days
AD = abstinence days

Figure 9.2 - Percentage of participants self-reporting heavy drinking days and abstinence days.

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10. THE INFLUENCE OF MEDIA REPORTING OF SUICIDE IN JAPAN

On 8th April 1986, young Japanese pop star, Yukiko Okada, committed suicide, and there was considerable media coverage in Japan. There was concern, particularly by the Japanese Suicide Prevention Association, that her fans may imitate her [17], and/or suicide may increase generally due to the media coverage. Was this the case?

For example, Yoshida et al (1991; quoted in Hagihara et al 2007) compared the actual and expected number of suicides among under 20s around the time of her death, and found that the actual number was significantly higher than expected. Kurusu (1992; quoted in Hagihara et al 2007) found an increase in suicides in the four days after 8th April 1986 among 10-14 year-old females.

Hagihara et al (2007) were interested in whether media coverage of suicide and the increased availability of suicide information on the Internet together explained suicide in Japan. For the period, 1st January 1987 to 31st March 2005, the researchers collected articles about suicide in four widely-read national newspapers, official monthly statistics of suicides, and the prevalence of Internet use by household.

The dependent variable (outcome) in the analysis was the number of suicides in a month, and the independent variables (cause) were the number of newspaper articles reporting suicide in the previous month, prevalence of household Internet use in the previous month, and national unemployment rate in the previous month.

For the study period, the average number of suicides per month was 1416 for males and 673 for females with an underlying trend of increasing over the eight years studied. It was found that, for males, all three independent variables significantly predicted suicide, but only increased number of newspaper articles about suicide led to increased suicide for females (figure 10.1).

The researchers' statistical modelling showed that an increase of 1 unit [18] in newspaper articles on suicide in one month led to a 0.01% increase in suicides in the next month, while a 1% increase in household Internet use produced a 0.16% rise in suicides, and a 1% increase in unemployment rate led to 12.7% more suicides for males.

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[17] The term "Yukko syndrome" was coined for this.

[18] 1 unit = (number of articles in newspaper A) x (circulation of newspaper A) + (newspaper B) + (newspaper C) + (newspaper D).

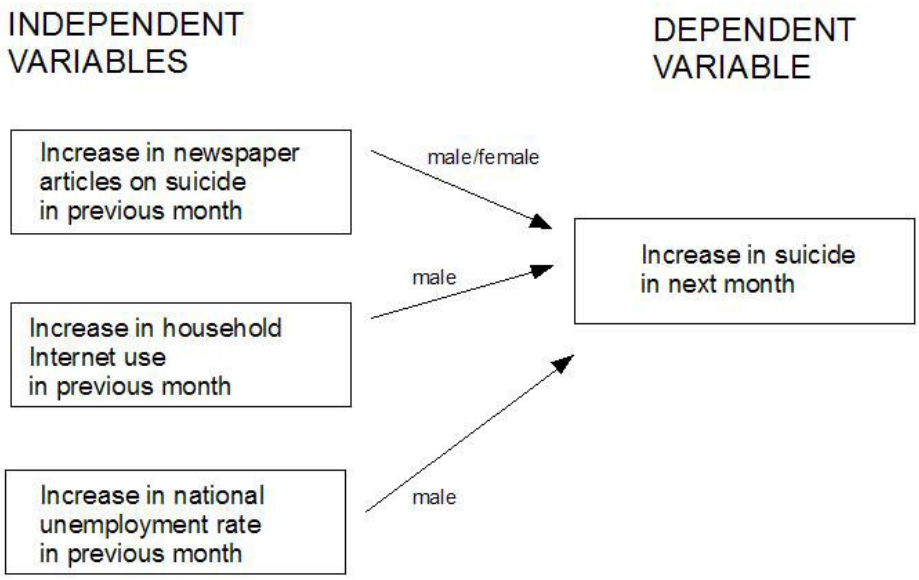

Figure 10.1 - Significant predictors of increased suicide.

This study was based on the analysis of statistics, and so has certain limitations:

i) It presents general patterns in Japan, but not the individual cases.

ii) "Household Internet use" does not mean that individuals who committed suicide had been using the Internet. Other members of the household may have been doing so.

iii) Internet use may not have been to read about news stories or to visit suicide web sites. Internet use covers many activities.

iv) Monthly suicide rates are not as sensitive as daily figures, which can be linked to specific days and newspaper coverage.

v) Suicides in the same month as the newspaper articles are missed by using the previous month as the independent variable.

vi) The newspaper articles were collected using the keyword, "suicide" in the database of newspaper articles. This may have missed articles that discussed suicide without including the term in the title or as a keyword.

vii) No account was taken of the amount of coverage of a suicide (ie: length of articles) or prominence (eg: front page with photographs of victim).

viii) Other media were not included. Television is a key source of news, and may have a greater influence than newspapers.

ix) Only four newspapers were used because they were available for the whole study period.

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