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ASPECTS OF DEPRESSION

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

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

CONTENTS

1. SEX AND ETHNIC DIFFERENCES IN PREVALENCE OF DEPRESSION AND ANXIETY

The rates of mental disorders like depression and anxiety disorders varies between men and women. But is this because of biological differences between the sexes, social differences (eg: expectations in society), or an artefact of the study (eg: women more willing to seek help)? Answering this question is difficult, but a large-scale cross-cultural study will help.

Gater et al (1998) reported details of the World Health Organisation Study of Psychological Problems in General Health Care across fifteen countries (table 1.1). Stratified random samples of individuals attending primary care clinics in these countries were interviewed and diagnosed using ICD-10 criteria for depression and anxiety disorders (n = 25 916).

- Ankara, Turkey
- Athens, Greece
- Bangalore, India
- Berlin, Germany
- Groningen, Netherlands
- Ibadan, Nigeria
- Mainz, Germany
- Manchester, England
- Nagasaki, Japan
- Paris, France
- Rio de Janeiro, Brazil
- Santiago, Chile
- Seattle, Washington
- Shanghai, China
- Verona, Italy

Table 1.1 - Sites in the World Health Organisation Study of Psychological Problems in General Health.

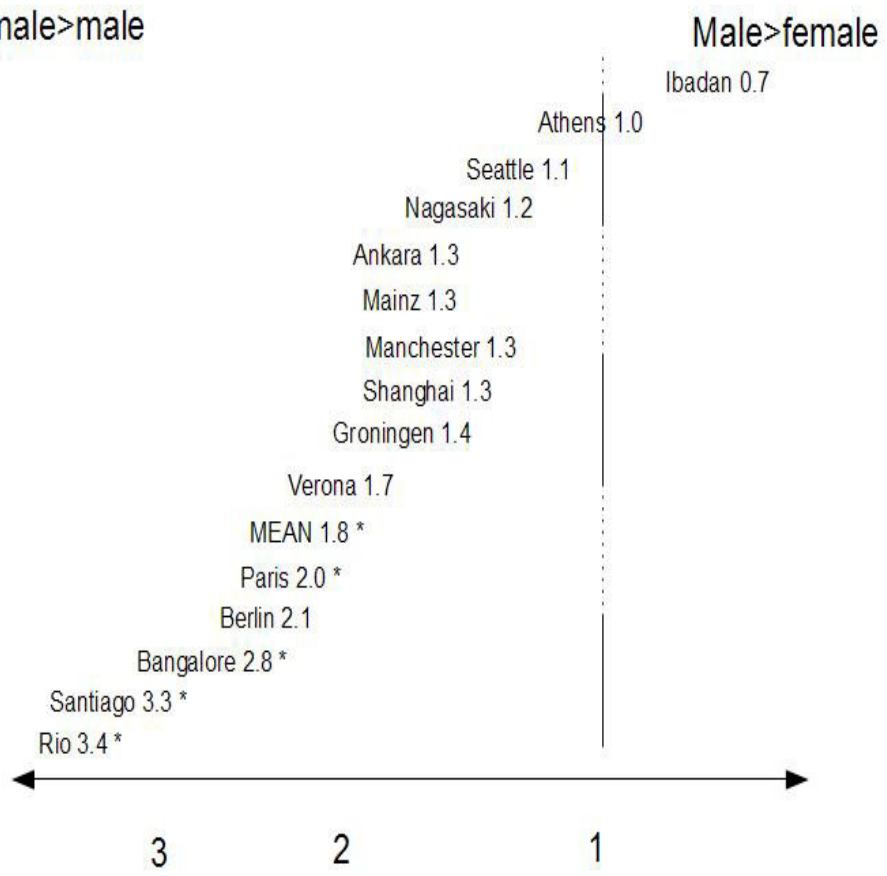
The mean prevalence of current depression was 7.1% for men and 12.5% for women (sex ratio - 1.8 female: 1 male; $p<0.01$ [1]), but this varied between countries (from 3.4 in Brazil to 0.7 in Nigeria) (figure 1.1).

The mean prevalence of current generalised anxiety disorder was 5.7% for men and 9.2% for women (sex ratio - 1.6; $p<0.01$), and 1.5% and 2.8% respectively for current agoraphobia and panic disorder (sex ratio - 1.9; $p<0.01$).

[1] Traditionally a p-value below 0.05, like this, is viewed as statistically significant after the data have been analysed with statistical tests. There is the risk that the results will be statistically significant approximately one in twenty times "by chance". Studies often do many statistical comparisons, and thus are "virtually guaranteed to find statistical significance in results that are meaningless statistical flukes" (Seife 2011).

There were differences between countries also (figure 1.2).

The authors were cautious about the reasons for the se differences, and accepted that symptom reporting differences may explain them.

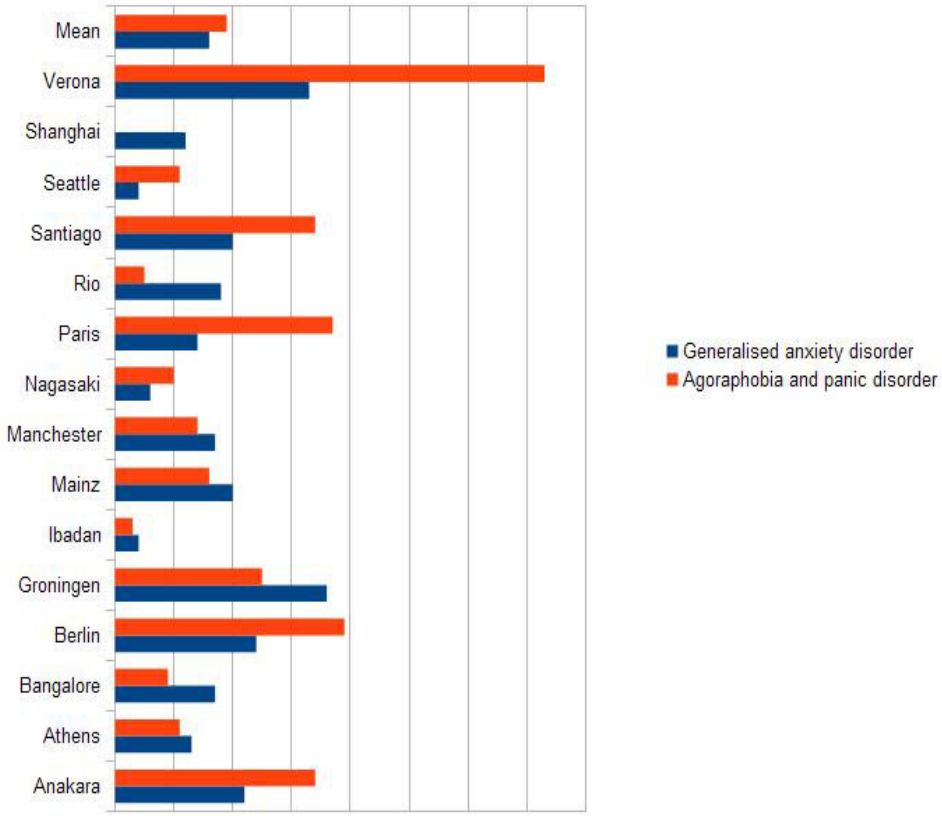


(* = significant difference at p<0.01)

(Data from Gater et al 1998 table 3 pp410-411)

Figure 1.1 - Sex ratios for depression in fifteen countries.

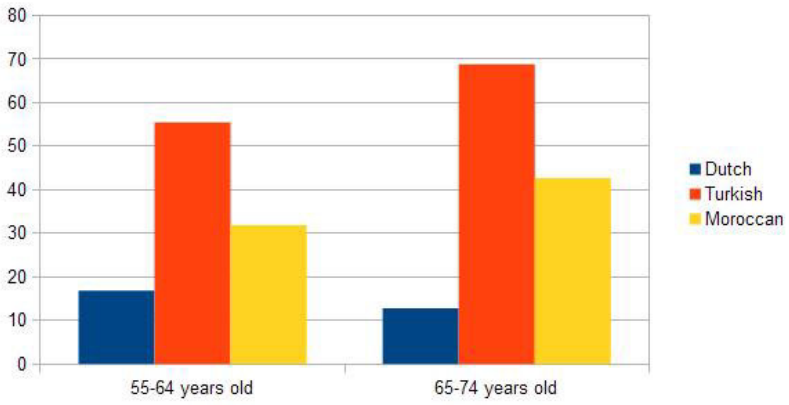
A general prevalence figure hides the diversity of rates among sub-groups within the population (eg: age, ethnicity). For example, van der Wurff et al (2004) found a rate of 14.5% for depression among older native Dutch adults (55-74 years old) in Amsterdam, but among Turkish and Moroccan ethnic minorities the rate was 61.5% and 33.6% respectively (figure 1.3). Depression was more common among women, and individuals with chronic health problems and physical disabilities in the whole sample.



(Sex ratio >1 = more women than men suffer)

(Data from Gater et al 1998 table 3 pp410-411)

Figure 1.3 - Sex ratios for generalised anxiety disorder, and agoraphobia and panic disorder in fifteen countries.



(Data from van der Wurff et al 2004 table 2 p37)

Figure 1.3 - Different rates of depression based on age and ethnicity.

Depression was measured using the Center for Epidemiologic Depression Scale (CES-D) (Radloff 1977) which produces a score between 0-60 (with 16 or above as the cut-off for depression) [2].

2. MEASURING DEPRESSION IN OLDER ADULTS

Depression is the most common psychiatric disorder among older adults (approximately 1 in 6), the leading cause of suicide for them, and an independent predictor of mortality (Baldwin and Wild 2004). Katona (1996) stated that depression in older adults is more likely to be chronic with a high risk of relapse.

Late-life depression can manifest symptoms that are different to earlier life (eg: less complaint of sadness and more somatic complaints) (Baldwin and Wild 2004). There are also a variety of physical disorders that older adults may experience which can cause organic depression, like Alzheimer's disease and stroke. Vascular depression has been proposed as a new sub-type due to stroke-induced while matter changes (Baldwin and O'Brien 2002). There is less depressive thinking (eg: guilt, unworthiness) compared to depression generally, and more prominent symptoms like apathy, and poor executive functioning (Baldwin and Wild 2004).

The most commonly used self-reporting assessment of depression in older adults is the Geriatric Depression Scale (GDS) (Yesavage et al 1983). Originally developed with thirty yes/no items [3], but 1, 4-, 5-, 10- and 15-item versions are also used (table 2.1).

Montorio and Izal (1996) reported a test-retest reliability (table 2.2) of the GDS-30 of 0.94. The validity of GDS-30 is based on individuals living independently in the community, and the use with other groups (eg: psychiatric inpatients) has produced different findings, for example, in relation to a cut-off

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[2] Depression can be viewed in two ways:
i) A continuum from mild to severe with a cut-off point for classification as clinical (and thus needing help) (eg: CES-D cut-off point of 25 often used).
ii) Qualitatively different to low mood and normal feelings (eg: presence of six or more symptoms as a boundary between major depression and distress; Klein 1990).

Alternatively, it may be a combination of both. Some symptoms of depression are qualitatively different (eg: somatic symptoms) while others are severe points on a continuum (eg: overall severity). Put in a slightly different way, "symptoms exist on a severity continuum, but once they reach a certain threshold on this continuum, a qualitatively distinct variant of depression occurs" (Baldwin and Shean 2006 p104).

On the other hand, the experience of depression could be unique for each sufferer, such that for some individuals it is a continuum of behaviour while for others it is a distinctively different thing. The uniqueness will also include a different symptom-mix for each sufferer.

[3] Brink et al (1982) collected 100 items useful in distinguishing depression from non-depression, which they reduced to thirty (that correlated best with total score) with 100 volunteers.

ITEM	YES	NO
1-item version (GDS-1): • Are you basically satisfied with your life?	0	1
4-item version (GDS-4): • Are you basically satisfied with your life? • Have you dropped many of your activities and interests? • Are you afraid something bad is going to happen to you? • Do you feel happy most of the time?	0 1 1 0	1 0 0 1
5-item version (GDS-5): • Are you basically satisfied with your life? • Do you often get bored? • Do you often feel helpless? • Do you prefer to stay at home, rather than going out and doing new things? • Do you feel pretty worthless the way you are?	0 1 1 1 1	1 0 0 0 0
10-item version (GDS-10) (GDS-5 plus): • Are you in good spirits most of the time? • Have you dropped many of your activities and interests? • Do you feel happy most of the time? • Do you feel full on energy? • Do you think most people are better off (in their lives) than you are?	0 1 0 0 1	1 0 1 1 0
15-item version (GDS-10 plus): • Do you feel your life is empty? • Do you feel you have more problems with your memory than most? • Do you think it is wonderful to be alive now? • Do you feel that your situation is hopeless? • Are you afraid that something bad is going to	1 1 0 1 1	0 0 1 0 0

(Source: Baldwin and Wild 2004 box 4 p133; Almeida and Almeida 1999 table 2 p861)

Table 2.1 - Items in Geriatric Depression Scale.

RELIABILITY - Consistent both within itself (internal reliability) and over time (external reliability).
Internal reliability established by the split-half method, Kuder-Richardson method, or Cronbach's alpha. External reliability by test-retest or parallel forms method.

VALIDITY - Measure the behaviour that it claims to measure. The main types of validity are face, content, criterion (made up of concurrent, and predictive validity), and construct validity.

DISCRIMINATION - Individual items and the test as a whole discriminate between high and low scorers; eg: individual with severe depression should answer items different to individual not depressed. Item analysis is the main technique used here.

Table 2.2 - Criteria for good psychometric questionnaire.

point for diagnosis. Any cut-off point needs to give high sensitivity (accurate diagnosis of individuals with depression) and specificity (those individuals without depression) (table 2.3).

STUDY	DEPRESSION (SENSITIVITY)	NO DEPRESSION (SPECIFICITY)
Yesavage et al (1983) community sample cut-off = 11	84% (true positive) 16% (false negative)	95% (true negative) 5% (false positive)
Hickie & Snowden (1987) mixed sample * cut-off = 14	88% (true positive) 12% (false negative)	84% (true negative) 16% (false positive)
Hickie & Snowden (1987) cut-off = 15	88% (true positive) 12% (false negative)	100% (true negative)
Rapp et al (1988) inpatients cut-off = 14	65% (true positive) 35% (false negative)	93% (true negative) 7% (false positive)

(* sample from day centres, nursing homes, psychiatric out-patients and inpatients)

(True positive = individual with depression diagnosed as depressed by GDS-30; false positive = individual without depression diagnosed as depressed; true negative = individuals without depression correctly diagnosed; false negative = individual with depression diagnosed as without)

Table 2.3 - Three studies and cut-off points for GDS-30.

Chiu et al (1994) reported the translation of the GDS-30 into Chinese to use with eighty depressed and 113 non-depressed 60-87 year-olds in Hong Kong. Some items were changed slightly to fit Chinese culture (eg: "Do you find life exciting?" to "Do you find life full and colourful?"), or enhanced with examples (eg: "Do you prefer to stay at home, rather than going out and doing new things?" like "going out to have dinner in a new restaurant with one's relatives or friends").

Test-retest reliability was established by thirty participants completing the questionnaire a second time one week later (figure 2.1). The correlation of total scores for both questionnaires by each individual was 0.84 with a median score of 0.59 for the correlation of each item on both occasions.

The discriminant analysis was 97.5% (correctly classified 78 of 80 depressed individuals) ad 87.5% (correctly classified 98 of 113 non-depressed individuals).

The concurrent validity was 0.88 for the correlation of an individual's score on the GDS-30 and the Chinese version of the Zung Self-Rating Depression Scale (figure 2.2).

A cut-off point of 15 was found to produce the best

balance of true/false classifications (ie: 96.3% true positive/3.7% false negative and 92.0% true negative/8.0% false positive.

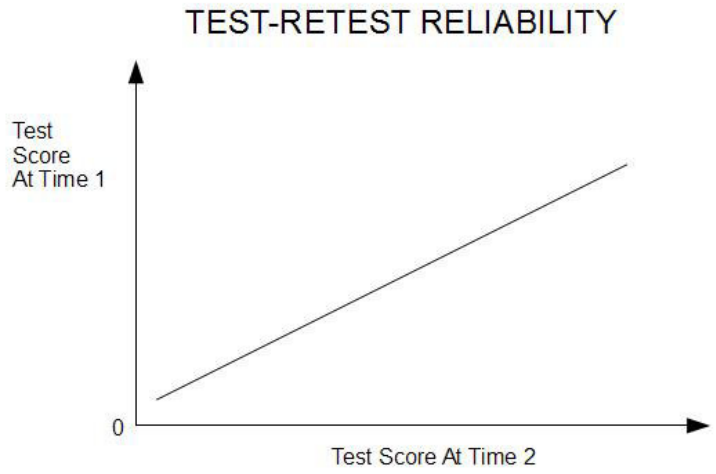

Figure 2.1 - Hypothetical scattergram showing a positive correlation for test-retest reliability.

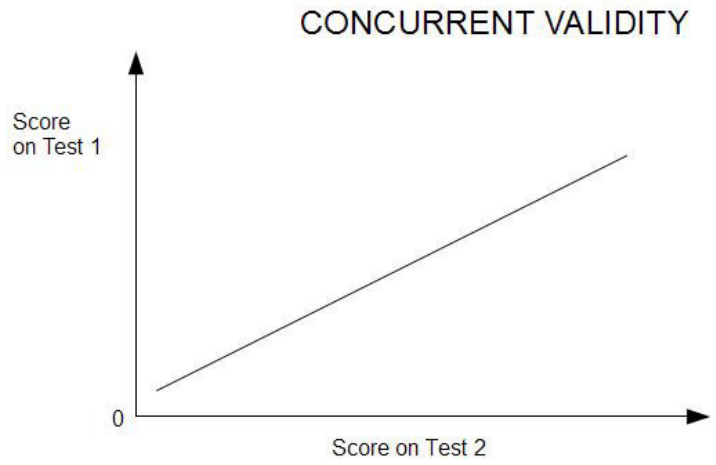

Figure 2.2 - Hypothetical scattergram showing a positive correlation for concurrent validity.

The validity of the GDS-15 has been established by comparing the scores with other established methods of diagnosis of depression (construct validity). For example, Almeida and Almeida (1999) collected data from 64 60 year-old and above adults attending a medical school in Sao Paulo, Brazil. Diagnosis using ICD-10 and DSM-IV [4] were compared to the GDS scores - average scores

[4] There was 87.5% agreement of diagnosis of the sample between ICD-10 and DSM-IV criteria.

on GDS-15 were 4 (not depressed), 7 (mild depression), 8 (moderate depression), and 12 (severe depression). A cut-off of 4-5 on GDS-15 correctly diagnosed 93% of individuals according to ICD-10 and 97% with DSM-IV. On the GDS-10, similar results were found with a cut-off of 3-4, but only 80% figures for 2-3 with GDS-4. GDS-1 only diagnosed 60% of cases. The authors felt that this suggested that it was not a "useful strategy to investigate the presence of depression in clinical practice" (p862).

Almeida and Almeida (1999) found a correlation of 0.82 between scores on GDS-15 and the Montgomery-Asberg Rating Scale (MADRS) (concurrent validity) [5]. Montorio and Izal (1996) reported a correlation of 0.78 between the GDS-30 and the Beck Depression Inventory (BDI).

Depression may be a risk factor for later dementia.

Chen et al (2008) interviewed adults over 65 years old in Anhui Province, China, and Liverpool, England, without dementia at baseline. Depression at baseline was compared with dementia at follow-up (1-4 years later). The risk of dementia was only associated with the most severe depression (2-5 times greater risk than non-depressed individuals).

3. UNDETECTED DEPRESSION

One problem with major depression is that it can go undiagnosed and undetected, and thus untreated. Maybe half of such cases are undetected by regular healthcare (Gilbody et al 2005). One solution is screening in general practice, particularly among high-risk individuals. This involves individuals being specially selected for a diagnostic interview and the offer of treatment.

Baas et al (2009) reported the limited success of such a screening programme at three health centres in the Netherlands in 2005-6. From GP records three high-risk groups were defined and invited for interview: individuals with current mental health problems, individuals with unexplained somatic complaints (ie: no apparent biological explanation to GP), and individuals who frequently visited their GP (top 10%).

This produced a total of 2005 individuals, which was reduced to 1687 with exclusions (eg: already known to have major depression). Eventually after refusals and whittling down with a preliminary questionnaire, 173

[5] Yesavage et al (1983) correlated the scores from the GDS-30 with those of the Zung Self-Rating Scale for Depression (SDS) and the Hamilton Depression Rating Scale (HAMD) using sixty depressed and 40 non-depressed older adults.

individuals were interviewed as potential risks, of which 71 were diagnosed as having major depression (but only 35 were new cases). Of the 35, 17 individuals agreed to treatment. Statistically, this gave the number needed to screen to treat one undiagnosed case of major depression as 118 (17/2005).

Undiagnosed and unrecognised mental illness can be a problem, particularly with older adults living alone. This is not helped by assessment measures that are difficult to use and require training.

The Global Mental Health Assessment Tool - Primary Care Version (GMHAT/PC) was designed to overcome these problems (Sharma etal 2004). It is a computer-based assessment used in the context of a semi-structured interview. The interviewer follows the instructions on each screen to question the interviewee and input the answers. Each screen concentrates on specific symptoms including worries, suicidal risk, sleep, appetite, and psychotic symptoms. At the end of the interview a diagnosis is suggested by the computer programme based on ICD-10 criteria.

Sharma et al (2010) reported the use of GMHAT/PC with 169 over 60s at three hospitals in the UK. Eighty-six individuals already had an ICD-10 diagnosis of a mental disorder, and eighty-three participants were the healthy controls. The sensitivity (ie: correctly recognising cases) of the GMHAT/PC was 77%, and the specificity (ie: correctly recognising non-cases) was 96%. Put another way, nineteen individuals with a mental disorder were "missed" by GMHAT/PC, and three individuals without were wrongly diagnosed (table 3.1).

	ICD-10 cases (n=86)	ICD-10 non-cases (n=83)
GMHAT/PC cases	67 (true positive)	80 (true negative)
GMHAT non-cases	19 (false negative)	3 (false positive)

Table 3.1 - Number of cases and non-cases.

4. LONG-TERM COURSE OF DEPRESSION

The long-term course of major depression can vary depending on a number of factors. Szadoczky et al (2004) assessed the two-year outcome of 117 in-patients in Hungary. They were interviewed in detail at entry to hospital (baseline), and evaluations of the depression were made after six weeks of treatment and two years later.

Thirty-four individuals had no remission of their depression during the two-year period, and these patients

were different to the others in having lower levels of education, more severe self-reported depression at baseline, were more anxious, and had less social support.

The severity of depression after six weeks of treatment (not at baseline) predicted the severity of depression at two years. In other words, individuals who did not respond to the six weeks of treatment in hospital suffered more after two years, and those who responded to treatment were more likely to have remission in the long-term.

When individuals first contact psychiatric services about their problems, they will be treated as an outpatient (eg: seen by psychiatrist at clinic) or as an inpatient admitted to a psychiatric hospital. Does the type of first contact predict subsequent hospitalisations or improvements in the disorder?

Kessing and Munk-Jorgensen (2004) attempted to answer this question for depression and bipolar disorder using data from Denmark. The Danish Psychiatric Central Research Register (DPCRR) includes details of all psychiatric admissions or consultations, except those by private psychiatrists. From the DPCRR, data about first contact between 1995 and 1999 were analysed.

There were 13 193 patients with depression (66% treated as outpatients and 34% as inpatients) and 1005 patients with bipolar disorder (39% treated as outpatients and 61% as inpatients). Those individuals whose first contact was inpatient treatment were twice as likely to have subsequent hospitalisations, and those with depression were four times more likely to commit suicide.

But it could be that the individuals treated as inpatients at first contact were more severe than the outpatients, and so the groups were not comparable. After adjusting for this, the difference was still there.

Kessing and Munk-Jorgensen interpreted the results as showing that the severity of the disorder at first contact predicts improvement or not. The more severe (and consequently inpatient treatment) at first contact, the more likely poor outcome (and subsequent hospitalisation, for example).

"Suicide ideation" is a term used to refer to thoughts about suicide and/or how to commit suicide. It is usually associated with depression, and with suicidal behaviour.

Rates of suicidal ideation vary from 1-22% of the sample studied depending on the age, for example (Pfaff and Almeida 2004).

In a community sample of Western Australian over 60s, 6.3% (32 of 504) admitted to current suicidal ideation (ie: past week), of which three-quarters were classed as depressed, and about one-fifth had a history

of suicide attempts. Depressed individuals were three times more likely to have suicidal ideation than non-depressed individuals, and those with a history of suicide attempts fifteen times more likely (than no history) to have such thoughts (Pfaff and Almeida 2004).

5. ATTRIBUTION AND DEPRESSION

The relationship between negative life events and depression is not straightforward because not everybody experiencing the former suffers from the latter. There seems to be an intervening variable, which is negative cognitive style (ie: a negative way of thinking about the world) (figure 5.1).

But what are the elements of this negative cognitive style? Attribution of cause is key. Abramson et al (1978) highlighted three dimensions of attribution of negative events that lead to depression:

i) Stable (vs unstable) - the cause is unchanging and/or unlikely to change.

ii) Global (vs specific) - the cause applies to all situations.

iii) Internal (vs external) - the cause is related to the individual (rather than the situation).

Using the example of a pupil failing a mathematics test. The negative cognitive style leads to the attribution of cause of failure as due to the individual being poor at mathematics (not that the test was difficult) (internal), being poor at all subjects (rather than just mathematics) (global), and these will not change (stable).

Hilsman and Garber (19950 showed this attributional process in a study with over 400 11-12 year-olds in the USA. Details of their cognitive styles were taken one week before report cards were due using various psychometric questionnaires. The children completed a questionnaire about their mood the next day after the report cards were distributed, and one week later. The negative life event was receiving grades lower than expected.

Negative cognitive style predicted depression the day after the report cards, but not one week later. Also the stronger the negative cognitive style, the more the depressive symptoms.

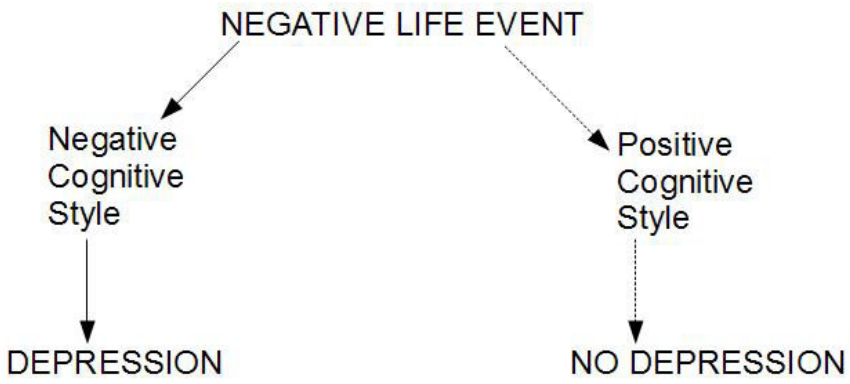

Figure 5.1 - Relationship between negative life events, cognitive style, and depression.

6. DEPRESSION AND PREGNANCY

There are situations where pharmacological treatments are not possible or advisable, like major depression in pregnancy [6]. Pharmaceutical companies advise against the use of anti-depressants here because the drugs can pass into the placenta (as in breast milk; table 6.1) [7]. Thus there is a need for non-pharmacological treatments, like therapy.

Another alternative for pregnant women is acupuncture which Manber et al (2004) assessed in a small randomised controlled trial (RCT) of sixty-one women in the USA. There are key elements to RCTs:

Breast-feeding mothers prescribed anti-depressants, for example, can pass the substance onto their babies. Yoshida et al (1998) reported this from a case study [8] of four mothers at the Mother and Baby Unit in the Bethlem Royal Hospital, London, who were taking the anti-depressant, fluoxetine. The breast milk was sampled for fluoxetine and its metabolite norfluoxetine in the morning 12-15 hours after the last dose of the drug.

As to the effect of the ingested substance for the infant, Yoshida et al (1998) reported that they showed no developmental abnormalities at one year old.

Table 6.1 - Breast-feeding and anti-depressants.

[6] Major depression during pregnancy ranges from 3-14% of women (Manber et al 2010).

[7] However, anti-depressant use during pregnancy doubled between 1999 and 2003 (Manber et al 2010).

[8] This method gives detailed information about a small number of individuals, but the findings cannot be generalised because of the small sample size.

i) Random assignment of participants to conditions - active acupuncture adapted for depression, general acupuncture (control acupuncture condition), or massage (control condition) for twelve sessions over eight weeks.

ii) Double blind - the individuals performing the acupuncture did not know details of the research, while the women did not know which acupuncture group they were. Obviously, the women in the massage group knew this, but they were not aware of the research purpose.

iii) Standardised measure of outcome ("responders") - Hamilton Rating Scale for Depression ($HRSD_{17}$) reduction of at least 50% from baseline and total score of less than 14.

At the end of the eight weeks of the trial, 68.8% of the active acupuncture group were classed as responders, 47.4% of the acupuncture control, and 31.6% of the massage control. The active acupuncture group was significantly higher than the massage control only. The authors felt that this study showed the promise of acupuncture for treating depression.

But there were limitations to this pilot study:

- Women entered the study at different points in the pregnancy between weeks 11 and 28.
- A small sample and seven women dropped out leaving 54 completing the study.
- The comparability of massage (shortened to twenty minutes to equal the length of acupuncture sessions) to acupuncture.
- Sample bias - majority of women were Caucasian, with high levels of education and income. This is because the study was volunteer-based from advertisements in obstetric clinics and local parent and baby magazines.
- To aid blinding, the treatment was carried out by one group and the assessment by the researchers. Though methodological controls were used (eg: audiotaping acupuncture sessions of which a sample were listened to), there was no guarantee that the conditions were standardised.
- $HRSD_{17}$ is a standardised measure of depression, but it is self-rated, thus depending on the "honesty" and accuracy of replies.

Manber et al (2010) reported the same design of RCT with 150 pregnant women. The response rate was 63% for

the specific acupuncture group, which was significantly more than the other two groups combined (44.3%).

7. INSOMNIA AND DEPRESSION

Insomnia can cause depression, and depression can cause insomnia, but which comes first? van Moffaert (1994) called this a "chicken and egg situation" [9]. Ford and Kamerow (1989) was a key study in the use of longitudinal method to establish the temporal order (ie: insomnia before depression [10]) and thus a causal direction [11]. Individuals reporting insomnia at baseline were early forty times more likely to have major depression one year later. This relationship has been replicated in over a dozen studies of different cohorts and lengths of time (Pigeon 2010).

So the preferred method of research the longitudinal study is able to record the presence of insomnia over time (from baseline to follow-up) and the presence or absence of depression at follow-up.

Okajima et al (2012) used a two-year period in their study of adults in rural Japan. The baseline was established with a self-rated questionnaire in November 2005-January 2006, and follow-up was in November-December 2007 in the town of Daisen (Tottori Prefecture) (figure 7.1). All adults over 20 years old in the town (n = 5528) received the baseline questionnaire, and 2825 of them completed it (51% response rate), and then 1577 of them completed the follow-up questionnaire (56% response rate) (table 7.1). Anonymity was maintained by the use of unique code numbers for each participant.

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[9] The relationship is also bidirectional. Buysse et al (2008) found that insomnia predicted future major depression, and major depression then predicted future insomnia.

[10] This is key evidence, but also studies showing that treating insomnia helps depression (eg: Morawetz 2003).

[11] Turek (2005) noted the use of the word "association" between insomnia and depression by researchers and a reluctance to use "cause". There is an alternative relationship between insomnia and depression with a common biological basis to both, and when this is altered this produces both behaviours ("duality-of-effect hypothesis") (Turek 2005).

(Drawn with MapCreator2)

Figure 7.1 - Position of Tottori Prefecture in Japan.

	TOTAL	MALES	FEMALES
Eligible for questionnaire	5528	2521	3007
Completed baseline questionnaire	2824	1220	1604
Completed follow-up questionnaire	1577	684	893

(Source of data: Okajima et al 2012 figure 1 p378)

Table 7.1 - Numbers of respondents.

The questionnaire included a Japanese version of the Pittsburgh Sleep Quality Index (PSQI) (Doi et al 2009) [12]. This measures seven aspects of sleep in the last month of a scale of 0-3:

- Sleep quality (how well individual feels they slept).
- Sleep onset latency (perceived time to fall asleep).
- Sleep duration (estimated number of hours of sleep per night).
- Sleep disturbance (eg: early morning awakening).
- Habitual sleep efficiency (amount of time spent in bed

[12] Originally developed by Buysse et al (1989).

relative to time sleeping).

- Use of sleeping medication.

- Daytime dysfunction (eg: difficulty concentrating).

A PSQI score of six or greater was categorised as an insomnia sufferer for this study.

Depression was measured with a shortened version of the Center for Epidemiologic Studies Depression Scale (CES-D) (Poulin et al 2005). There were twelve items and each one was scored from 0 ("never or rarely") to 3 ("always"). A score of twelve or more was categorised as depressed.

Based on the results the respondents were divided into groups (table 7.2) and sub-groups (table 7.3).

	CES-D <12	CES-D ≥12
PQSI <6	No insomnia No depression N = 874	No insomnia Depression N = 188
PQSI ≥6	Insomnia No depression N = 216	Insomnia Depression N = 160

(Source of data: Okajima et al 2012 table 2 p380)

Table 7.2 - Four main groups for analysis.

The presence of insomnia at baseline was significantly associated with the reporting of depression at follow-up (ie: twice as likely as non-insomniacs). Further analysis revealed that poor quality sleep, sleep onset latency, sleep disturbances, use of sleeping medication, and daytime dysfunction were individually significantly associated with depression, but not sleep duration and habitual sleep efficiency.

INSOMNIA
i-1: No - both (baseline/follow-up) (N = 762)
i-2: Yes (follow-up only) (N = 117)
i-3: Yes (baseline only) (N = 128)
i-4: Yes - both (N = 171)

DEPRESSION
d-1: No - both (baseline/follow-up)
d-2: Yes (follow-up only)
d-3: Yes (baseline only)
d-4: Yes - both

(Source of data: Okajima et al 2012 table 1 p379)

Table 7.3 - Sub-categories of respondents.

Individuals reporting insomnia in both questionnaires (category i-4) were seven times more likely to have depression at follow-up than non-insomniacs (category i-1) (adjusted odds ratio) (table 7.4).

Category	Follow-up	Baseline & Follow-up
i-1 (no insomnia)	6.3	5.3
i-2 (follow-up only)	33.3	13.7
i-3 (baseline only)	7.8	12.5
i-4 (both)	17.0	31.6

(Source of data: Okajima et al 2012 table 4 p381)

Table 7.4 - Percentage of respondents reporting depression based on insomnia.

Evaluation

i) Use of self-reports to a validated questionnaire to diagnose insomnia. Other studies have used structured interviews (eg: Buysse et al 2008; appendix 7A).

ii) Insomnia and depression measured at two points two years apart, and no details of the symptom levels inbetween. More regular assessments would be recommended (Morin et al 2009).

iii) Few studies have investigated the individual elements of insomnia that predict depression. For example, Chang et al (1997) found that poor quality sleep and less than seven hours per night at university was associated with depression 30 years later (longitudinal study) (table 7.5). While in a cross-sectional study, difficulty falling asleep was most important, then difficulty maintaining sleep, and early morning awakening (Kaneita et al 2006; appendix 7B).

iv) An attempt to study a whole town, though only 28.5% of eligible residents completed the study.

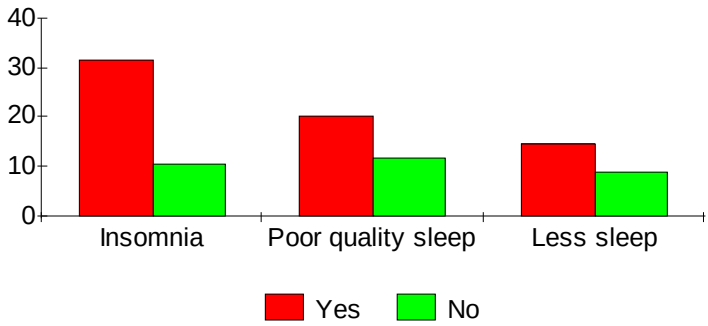
v) A study in rural Japan, whereas many other studies have taken place in Western countries and/or urban settings.

vi) Individuals who dropped out of the study before follow-up were slightly younger than those who completed.

Details of the sleep habits of 1024 male students at the John Hopkins University School of Medicine in Baltimore, USA, were collected between 1948 and 1964 as part of the standard medical examination given to students.

In a follow-up of the men over 30 years later, 101 reported clinical depression. These men were over twice as likely to have had insomnia as a student than non-depressed men as well as poor quality sleep and less of it as a student.

The information on sleep was based on simple questions like, "Do you ever have insomnia?" or "Do you sleep well?". Depression was also measured by self-reports for any time after graduation until the end of 1993 (eg: use of anti-depressant medication).



(Data from Chang et al 1997 table 2 p109)

Percentage of men reporting clinical depression at follow-up based on sleep as a student.

Table 7.5 - Details of Chang et al (1997)

APPENDIX 7A - BUYSSE ET AL (2008)

Buysse et al (2008) used 591 young adults [13] in Switzerland as part of the Zurich Study began in 1978 [14] [15]. Over twenty years these individuals were interviewed six times [16] [17] [18].

Insomnia was measured by the question - "Have you experienced disruptions in your sleep pattern during the last twelve months, eg: inability to fall asleep, waking

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[13] Evaluation - Age group restricted to 20-40 years old, which limits the generalisability of the findings.

[14] Details in Angst et al (1984).

[15] Evaluation - Two-stage stratified sampling used to gain a representative sample of young adults in the canton of Zurich. Stage 1 involved sending a questionnaire about psychiatric health (Symptom Checklist 90-R; SCL-90-R to all males aged 19 years old and females aged 20 years old in the area. From the respondents 292 males and 299 females were chosen in stage 2. These were two-thirds of the high scorers (above 85th percentile) and one-third randomly below the 85th percentile. This process was done for the Zurich Study.

[16] Evaluation - Interviews in 1979, 1981, 1986, 1988, 1993, and 1999. Is this too many or too few in a twenty-year period?

[17] Evaluation - 47% (278) of the sample completed all the interviews, but 91.4% (540) completed at least two interviews, 63% (372) at least five, 74% (435) at least four, and 82% (486) at least three interviews.

[18] Evaluation - A 20-year longitudinal study is a good length of time.

up in the night, or waking up too early in the morning?" [19]. Answers were categorised into four periods - at least one month in duration (24.4% of participants [20]), 2-3 weeks at least once (13.2%), less than two weeks but recurring at least monthly (recurrent brief insomnia) (24.2%), and less than two weeks occurring less than monthly (occasional brief insomnia) (15.9%) - and no insomnia (22.3%) [21].

Between 17-50% of participants without depression reporting insomnia lasting two weeks or longer at one interview had major depression at a later interview [22]. While 8-29% of individuals with depression at interview had experienced the insomnia previously [23] [24] [25].

APPENDIX 7B - KANEITA ET AL (2006)

Kaneita et al (2006) calculated a nationwide prevalence rate of 23.5% for insomnia among over 100 000 adolescents (12-18 years old) in Japan. This was similar to 21.4% found in 3000 Japanese adults (Kim et al 2000).

While in Europe (France, Great Britain, Germany and Italy), the rate was 25.7% among over 1100 adolescents (Ohayon et al 2000), but 34% in the USA (Roberts et al 2000). In a study in China, 16.9% of 1365 adolescents reported insomnia (Liu et al 2000).

Kaneita et al (2006) defined insomnia as the presence of one of the following in the past month:

- Difficulty initiating sleep (DIS) - "Do you have difficulty falling asleep at night?".
- Difficulty maintaining sleep (DMS) - "Do you wake up during the night after you have gone to sleep?".
- Early morning awakening (EMA) - "Do you wake up too early in the morning and have difficulty getting back

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[19] Evaluation - Sleep-wake diaries may be more accurate than a general self-report statement like this, while polysomnographic measures would be best of all (eg: EEG).

[20] This is a cumulative percentage (ie: individuals categorised based on at least one interview not the same in every interview).

[21] Evaluation - The reliability of these categories is open to question. A measure like number of nights per week or per month might have been better (Buysse et al 2008).

[22] Evaluation - Depression (and other psychiatric disorders) were diagnosed for the past year by a semi-structure interview lasting 2-3 hours conducted by trained professionals using the Structured Psychopathological Interview and Rating of Social Consequences of Psychic Disturbances for Epidemiology (SPIKE). SPIKE has inter-rater reliability of 0.89-0.91, and validity was established by comparison with medical records (Buysse et al 2008).

[23] Range of figures given because of the six interviews, and average not given.

[24] Evaluation - The diagnostic criteria for depression changed during the period of the study (from DSM-III-R to DSM-IV).

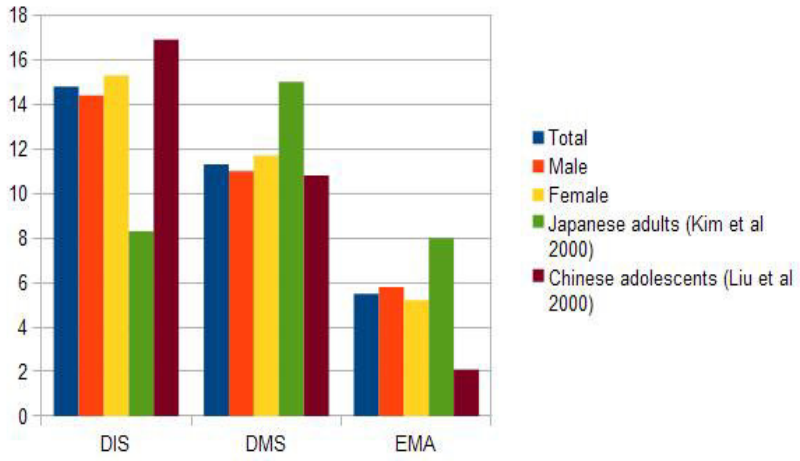
[25] Evaluation - The study did not take account of current medication use (Buysse et al 2008).

to sleep?".

Participants were offered five response options for each question varying from "never" (0) to "often" (3) and "always" (4), with the latter two replies classed as the presence of the behaviour.

In terms of the individual symptoms of insomnia, significantly more female students experienced DIS and DMS, but significantly more male students reported EMA (all $p<0.01$) (figure 7.2).

Based on other questionnaire information collected about the students, insomnia was significantly associated with being male, having poor mental health, skipping breakfast, drinking alcohol, smoking, not participating in extra-curricular activities, and late bedtime (after midnight) (with no intention to go to university a factor for the older adolescents).



(Data from Kaneita et al 2006 tables 1-3 pp1545-6)

Figure 7.2 - Percentage of Japanese adolescents reporting the symptoms of insomnia ("often" and "always" responses).

In terms of the individual symptoms of insomnia and older adults, Yokoyama et al (2010) used a nationally representative sample of Japanese adults aged 65 years and above (n = 4997) sampled in 1999 (baseline), and interviewed subsequently in 2001, 2003, and 2006.

DIS reported in the interview in 2003 was a significant predictor of depression in 2006 [26] [27].

The study also found a weak correlation (Pearson correlation coefficient <0.4) between the sub-types of

[26] 11.1% of the sample reported DIS and 34.8% of them had depression later, compared to 22.9% and 20.2% for DMS and 11.5% and 29.1% for EMA respectively. Other studies have found EMA to be more common (Pigeon 2010).

[27] The researchers controlled for sleep duration and excessive daytime sleepiness (Pigeon 2010).

insomnia suggesting that they are distinct.

But the sub-types vary over time. For example, Hohagen et al (1994) found low stability of sub-types over four months among 328 GP patients in Mannheim, Germany. Of these individuals who reported DIS at baseline, only 51% reported it at the second interview, 17% for DMS, and 45% for EMA. The authors concluded that sub-dividing insomnia may not be a "useful tool".

The Yokoyama et al (2010) study did not ask about a history of depression, and the authors admitted: "we may be examining the relationship between insomnia sub-types and recurrence of depression rather than between insomnia sub-types and initial onset of depression" (Yokoyama et al 2010 p1701). Furthermore, no information was collected on depression (eg: severity, remission) other than the self-reports at points of time.

8. TREATING DEPRESSION IN CHRONIC ILLNESS

Depression often develops with chronic illness, and, in one sense, there is a reason to be depressed in these circumstances. However, it is still possible to treat the depression as such symptoms should never be viewed as normal (Markowitz et al 1998).

Markowitz et al (1998) compared four interventions for depression among individuals with HIV-positive status for longer than six months in New York. The 101 volunteers were randomly divided into four conditions for the sixteen weeks of the study:

i) Interpersonal psychotherapy (IP) [28] - concentrates upon changes in mood in relation to life events with the focus on what the patient wants now and how to achieve it.

ii) Cognitive behaviour therapy (CBT) [29] - concentrates upon irrational and negative thoughts underlying moods, and how to change them for the better.

iii) Supportive therapy - the therapist listened to the client's concerns, but did not attempt to change anything.

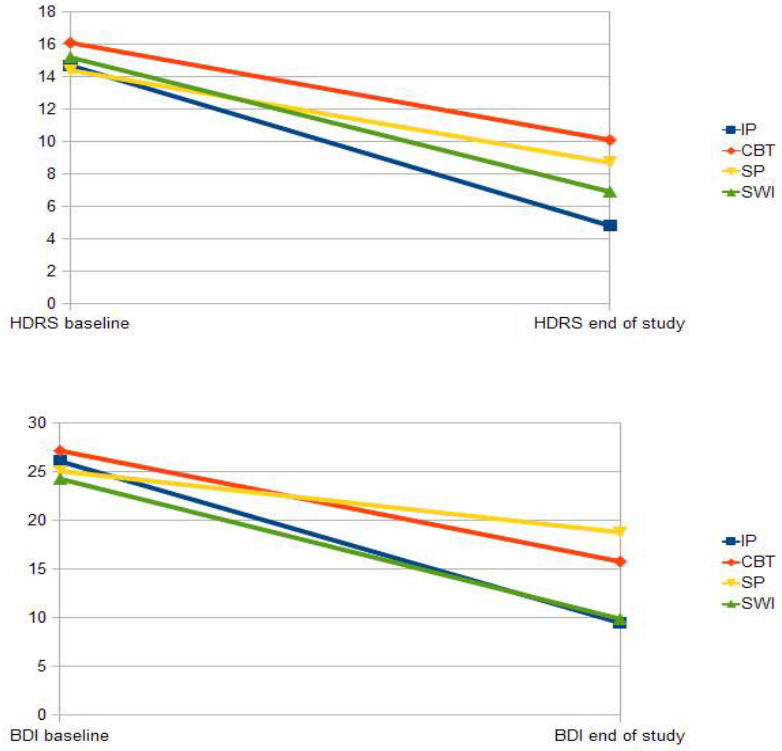
iv) Supportive therapy and imipramine (anti-depressant drug) (SWI).

Baseline and outcome measures of the severity of depression were taken using the 17-item Hamilton

[28] Based on principles in Klerman et al (1984).
[29] Based on the principles in Beck et al (1979).

Depression Rating Scale (HDRS) (Hamilton 1960) and the Beck Depression Inventory (BDI) (Beck 1978). Sixty-nine participants completed the study.

All completers showed an improvement between baseline and end of the study, but the greater decline in depression occurred with IP and SWI (figure 8.1).



(Lower score = less severe depression; HDRS = Hamilton Depression Rating Scale; BDI = Beck Depression Inventory)

(Data from Markowitz et al 1998 tables 2 and 3 p456)

Figure 8.1 - Mean scores for completers on two measures of depression severity.

Musselman et al (1998) reviewed the relationship between depression and heart problems (cardiovascular disease). About one in five cardiovascular disease patients are diagnosed with depression. But what is the relationship between the two conditions?

a) Depression is a risk for future heart disease - controlling for other risk factors, individuals with depression have a greater risk of cardiovascular disease than non-depressed individuals (men over twice the risk in some studies). Physiological changes occur in major depression which make the body vulnerable to heart problems.

b) Cardiovascular disease is a risk factor for depression - as with many chronic illnesses, sufferers

can develop depression, though a higher rate of depression is not found with more severe cardiovascular disease.

c) Depression and cardiovascular disease together predict a poorer outcome for the latter - eg: individuals with coronary artery disease and depression have more future "cardiac events" (eg: heart attacks) and earlier death than non-depressed coronary artery disease sufferers.

Though stress (including psychological problems) tends to lead to higher blood pressure (hypertension), there are contradictory results about anxiety and depression (figure 8.2). But a large-scale longitudinal study has found that anxiety and depression are associated with lower blood pressure. Using data from the Nord-Trondelag Health Study (HUNT study) in Norway, Hildrum et al (2008) reported that higher symptom scores of anxiety and depression in 1984-6 (baseline) predicted lower systolic blood pressure eleven years later (negative correlation).

The strengths of this study were the sample size (n = 36 530), the age range of the participants (20-89 years old), and its length. But it used self-report measures of anxiety and depression, and involved few ethnic minority participants.

Anxiety/depression	→	Increased blood pressure (eg: Jonas and Lando 2000)
Anxiety/depression	→	No effect (eg: Yan et al 2003)
Anxiety/depression	→	Lower blood pressure (eg: Hildrum et al 2008)

Figure 8.2 - Contradictory findings about anxiety and depression and blood pressure.

9. GENETICS AND DEPRESSION

If identical (monozygotic; MZ) twins have a higher concordance rate (ie: both twins suffer) for a mental disorder than non-identical (dizygotic; DZ) twins, then it is assumed that the condition has a heritability component. This is because MZ twins share 100% of their genes, whereas DZ twins only 50% as in any siblings.

Registers of twins are thus praised research tools. For example, in the USA, the Vietnam Era Twin Registry includes male-male twin pairs born between 1939 and 1957 where both twins served in the US military during the Vietnam War (1965-1975).

Lyons et al (1998) used this registry for a study of depression. Twins were telephone-interviewed, and diagnosed with major depression using DSM-III-R criteria. Among 1874 pairs of MZ and 1498 pairs of DZ twins, 9.2% were diagnosed with lifetime major depression (ie: suffered at some point in their lives, but not necessarily currently). The concordance rate for MZ twins was 22.5% (68 of 302 pairs both suffered) which was significantly higher than 14.0% for DZ twins (40 of 286).

Table 9.1 assesses the methodological issues of this study.

Eight out of ten research groups have confirmed that individuals with the s/s version of the serotonin transporter gene (5-HTTLPR) are more prone to depression after multiple stressful life events (more emotionally reactive) than individuals with the s/l (lowest reactivity) and l/l (intermediate) versions (Wilhelm et al 2009).

But would individuals want to know that they had that version of the genotype? Wilhelm et al (2009) offered the opportunity to 128 Australians (part of a 25-year longitudinal study) who had given genetic material previously. Eighty-eight individuals chose to receive their personal results.

Of these individuals, those with the s/s genotype were significantly more distressed in the following three months, but none regretted learning their result.

Interpersonal dependency (ID) is "an over-reliance on other people for emotional needs" (O'Neill and Kendler 1998). It is measured by the Interpersonal Dependency Inventory (IDI) (Hirschfeld et al 1977) [30]. There are three sub-scales: "emotional reliance on another person", "lack of self-confidence", and "assertion of autonomy". ID, particularly the "emotional reliance on another person" element, has been linked to disorders like depression, alcoholism, and eating disorders. For example, IDI scores positively correlate with Symptom Checklist (SCL) (Derogatis et al 1973) [31] scores; +0.53 among female twins on the Virginia Twin Registry (O'Neill and Kendler 1998) [32].

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[30] Items eg: "I feel that I never really get what I need from people".

[31] This lists a number of symptoms (ninety in SCL-90-R) like headaches, feeling critical of others, other people being aware of your private thoughts, and having to check and double-check what you do. Individuals are asked, "how much were you distressed by" each symptom with a choice of responses varying from "not at all" (0) to "extremely" (4).

[32] Spearman (1904) introduced correlation based on ranks (ie: Spearman's rho). This reduced "accidental error" (ie: influence of outliers), and "eliminates any disparity between the two characteristics compared" (ie: different scored variables).

ISSUE	POSITIVE ASPECT	NEGATIVE ASPECT
Telephone interviewing.	Face-to-face not feasible because of geographical dispersion and size of sample.	Lose visual cues of face-to-face interviewing.
Self-reported information.	Asks individual, who may not have sort treatment.	No independent verification of information given.
Sample size.	Large.	Not representative of all twins.
Response rate.	79.6% of twins agreed to participate.	Who were non-responders?
Zygosity (ie: MZ or DZ).	Established by questionnaire and blood group typing.	Only 95% accurate.
Lifetime depression.	More severe depression recalled accurately.	Unreliability of recall.
Interviewers.	Experienced and trained.	Close-ended options directly typed into computer database, which limited information to record.
Cohort.	Similar experiences.	Age range limited to 36-55 years old.

Table 9.1 - Methodological issues with the Lyons et al (1998) study.

10. BIOCHEMISTRY OF SEVERE DEPRESSION

Using a broad definition of severe depression, lifetime rates in the USA are 12% for men and 20% for women (Belmaker and Agam 2008).

There is a genetic component to severe depression, though it is complex and not related to a single gene, particularly for early-onset and recurrent depression. Twin studies suggest a heritability of about 37% (Belmaker and Agam 2008).

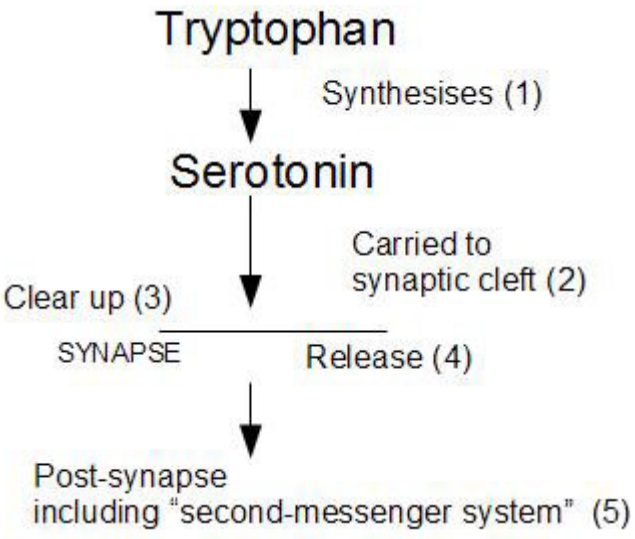
What is inherited is possibly changes to the neurotransmitters in the brain, particularly noradrenaline and serotonin (sometimes called the monoamine-deficiency hypothesis) (figure 10.1) (Belmaker and Agam 2008). This has been studied in different ways:

i) Metabolites (post-synapse by-products) of the neurotransmitters in the blood, urine, or cerebrospinal fluid of currently depressed individuals.

ii) Post-mortem studies of the brains of depressed individuals - eg: less p11 (a protein that works with serotonin receptors) (Belmaker and Agam 2008).

iii) Neuroimaging of live patients - eg: reduced tryptophan hydroxlase (TPH-2) (Belmaker and Agam 2008).

iv) Experimental manipulation of neurotransmitter levels - eg: a drink containing all amino acids except tryptophan stimulates the liver to synthesise proteins which remove tryptophan from the blood. This causes a relapse among individuals with a history of depression, but has no effect on non-depressed individuals (Belmaker and Agam 2008).



(After Belmaker and Agam 2008 figure 1 p59).

Figure 10.1 - Five potential differences in the synapse in depressed individuals.

Jans et al (2007) proposed "serotonergic vulnerability" as an explanation. This is the disruption of the serotonin neurotransmitter system including its manufacture, transportation, release at the synapse, and post-release.

Hormonal changes occur after childbirth that could produce this vulnerability based on the version of a gene the woman carries, and this could explain post-natal depression. Sanjuan et al (2008) found that the 12.7% of 1804 Spanish women who experienced such depression were more likely to have a high-expression version of a 5-HTT genotype than non-depressed women.

Recent studies have suggested that the malfunction in brain chemistry is in the "second-messenger system" (which carries information inside the cell). For example, cyclic AMP response element-binding protein (CREB) has

been altered in rats in an animal model of depression (Belmaker and Agam 2008).

When a biochemical basis is established, than an anti-depressant version can be developed, usually initially with animals. Depression-like behaviour is either selectively bred in rats or created by learned helplessness situations like the forced swim test. In this test, a rat is placed in a cylinder with no escape that fills with water. The rat struggles for a while and then floats passively (learned helplessness). Anti-depressant compounds increase the length of time of struggling (table 10.1). Genetically engineered mice with a gene knockout (ie: "turned off") are also used. "However, no animal model of depression captures the periodic change of behaviour into and out of depression that is seen in patients with depression" (Belmaker and Agam 2008 p57).

VARIABLE	LENGTH OF STRUGGLING TIME
Prior injection of anti-depressant	Increases
Serotonin-re-uptake-transporter knockout mouse	Decreases
Rat pups separated from mother/abused	Decreases
Genetically engineered mice to increase physiological stress reaction	Decreases

Table 10.1 - Example of results from forced swim test.

Other biochemistry that is involved in severe depression includes elevated cortisol (part of the physiological reaction to stress known as hypothalamic-pituitary-adrenal axis). For example, experiments that induce mild stress through simulated public speaking lead to greater changes in cortisol levels in the blood of depressed than non-depressed individuals (Belmaker and Agam 2008).

Belmaker and Agam (2008) pointed out that "A major liability of the hypothalamic–pituitary–adrenal axis theory of depression is the difficulty of defining the relationship of stress to depression. Some patients have a single lifetime depressive episode, whereas a larger proportion have a recurrent or even chronic course. Various types of acute stress, early childhood trauma, or long-term psychosocial problems may be involved and may lead to different responses of the stress system. Stress may be causative in some cases and secondary to depressed mood in others". Furthermore, "Most patients treated for depression have no evidence of hypothalamic–pituitary–adrenal dysfunction, just as most such patients have no direct evidence of brain monoamine deficiency" (p63).

There are many other hypotheses for the physiological basis to depression, but the evidence for these can be limited. Belmaker and Agam (2008) were open-minded:

> Avoidance of premature closure on any one scientific theory of the mechanism of depression will best serve the search for new, more effective treatments. It is likely that the pathogenesis of acute depression is different from that of recurrent or chronic depression, which is characterised by long-term declines in function and cognition (p65).

In conclusion, they said: "Major depressive disorder is likely to have a number of causes. Middle-aged or elderly patients presenting with depression may have a disorder related to cardiovascular disease and originating from endothelial dysfunction. Patients in their late teens or early 20s who have severe depression may have important genetic risk factors and a high risk of manic episodes. In patients with an anxious and depressive personality, depression may be due to genetically determined personality factors or adverse childhood experiences" (Belmaker and Agam 2008 p65).

11. STARTLE REFLEX TO PREDICT DEPRESSION

The startle reflex is an involuntary response to a sudden stimulus like a loud noise. This response is exaggerated if the stimulus is unpleasant and suppressed when the stimulus is pleasant. But this general principle is altered by the mood of the individual, and the affect-startle paradigm (Vrana et al 1988) was developed to study this.

Kaviani et al (2004) presented 22 in-patients at the Royal Bethlem Hospital, London, and 22 healthy controls with short film clips, which were either pleasant (comedy; ice skating), neutral (street scene; household objects), or unpleasant (toe surgery; gangsters dragging terrified victim into forest). During each film clip a burst of noise for 50 ms was used to produce the startle response. The startle response was measured by changes in electrical activity in the muscles of the eyes (ie: eyes widen during the startle response).

Overall, the strongest startle response accompanied the unpleasant film clips. Patients with lower depression scores showed greater startle responses than high depressive individuals, while the same was the case for low anhedonia (loss of pleasure) over high anhedonia individuals, and for high anxiety over low anxiety individuals.

This study showed that high levels of depression

and/or anhedonia compromises the startle response, while high anxiety is associated with hyperstartle responding.

Depression also affects the processing of facial expressions. Depressed individuals are slower to process positive emotion expressing faces. This is usually tested in the laboratory with the "face-in-the-crowd" task (Suslow et al 2001). In an array of faces showing the same expression, there will be one that is different (eg: smiling face among five neutral expressions), and the time taken to find it is measured.

Suslow et al (2004) compared eleven patients with major depression, eleven with major depression and anxiety disorders, and 22 never-depressed controls in Germany on the "face-in-the-crowd" task. The individuals with depression and anxiety were significantly slower than controls to find a happy face among neutral ones (in arrays of 2, 4 or 6 faces). Both depressive groups were slower than the controls in finding a neutral face among other expressions, but there was no difference between the groups for finding a negative expression among neutral faces.

12. QUALITATIVE RESEARCH ON SELF-HARM

Qualitative methods allow the researcher to explore the meaning of the behaviour to the participant.

Oldershaw et al (2008) used semi-structured interviews lasting one hour with twelve parents of adolescents receiving treatment for self-harm [33] [34]. The questions were open-ended to encourage the parent to talk, like "Describe how and when you first found out about your son/daughter's self-harm behaviour?".

From analysis of the verbatim transcripts four key themes emerged:

i) "The process of discovery" - The parents had gradually developed suspicions about the self-harming until it could not be ignored and help was required. For example, "Mr.T" said: "It's basically just grown. It's got worse. Whereas we, you know, finger crossed, everything crossed, you hope it's going to stop" (p141). While "Mrs.B" said about confronting her daughter - "She'd told them that it was the rats - she had two pet

[33] Nine mothers, two fathers and one grandmother with a maternal role of adolescents aged 13-18 years old attending Child and Adolescent Mental Health Services (CAMHS) in south London. In terms of ethnicity, nine were White, 2 Black African, and the other British Indian. Three of the parents were single parents, eight were married, and the other was co-habiting.

[34] Self-harm was defined as a "non-fatal self-injurious act purposefully carried out regardless of underlying intent" (Oldershaw et al 2008 p140).

rats at that stage - erm, so I asked her about it and she said 'yes it's the rats', so I thought well you know, sounds a reasonable explanation, I'll accept that" (p143).

This theme was sub-divided into three - "confronting an adolescent over the signs of self-harm", "parent reaction to disclosure", and "influence of outside agencies (schools or GPs) in timing of help-seeking".

ii) "Making sense of self-harm" - Two sub-themes here were "self-harm as a 'fashion', 'phase' or a deliberate choice", and "inability to empathise". "Mrs.H" saidin the case of the former, "First of all, my immediate reaction was erm she's just copying. She's just copying her friend", and for the latter, "I don't understand it, I mean, I know why, but I don't understand" (p143).

Parents tried to make sense by looking for causes of the behaviour, like coping with negative emotions, bullying, or to provide control. For example, "Mrs.M" said: "I can understand that it's some way of you having some sort of control over your pain, over your life, because you feel totally out of control when you're feeling so depressed or vulnerable or whatever" (p142).

iii) "Psychological impact of self-harm on parents" - Three sub-themes were "sadness and loss" (eg: "I miss [gets tearful] my little girl and that's that's quite hard"; "Mr.J"; p143), "loss of control and helplessness" (eg: "It was a case of, 'crap, we have lost control. I don't know what it is to do'"; "Mrs.L"; p144), and "influence of outside agencies on the psychological impact" (eg: "It just helped to have somebody to sound off and you know, am I doing the right thing"; "Mrs.E"; p144).

iv) "Effect of self-harm on parenting and family" - The nature of the relationship with the adolescent changed. For example, "Mrs.M" said: "She'd get annoyed with me or if I tell her off about something I'd then think 'oh is she going to go and run upstairs and...'" (p144) (sub-theme of "walking on eggshells").

Other sub-themes here were "denying own needs", "imbalance in parental involvement between siblings", and "positive effects on family life". In the first case, "Mrs.P" admitted: "I've put off going back to work because of what's been happening with her" (p144).

Any study needs to be aware of ethical issues and to gain ethical approval before going ahead, but when the topic is very sensitive this is very important:

- Confidentiality/privacy - Use of pseudonyms for parents and children.

- Consent - Though formal consent was not required from the adolescents, the researchers recommended that the parents discussed their participation in the study with their children.

- Distress - It was a difficult topic and emotionally stressful for the parents, so some distress could not be avoided.

- Right to withdraw/non-participate - Nine other parents declined to participate at different stages of the project.

- Participants were sent summaries of the analysis for feedback and two parents responded with satisfied comments.

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