Living with mental illness, dying of disease: The impact of stress, depression and the self-defeating attitudes on physical health.

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Abstract

The current research explores stress, personality, and the mortality and causes following a depressive illness in a series of seven studies. Study 1 found a positive relationship between the personality dimension of psychoticism and stress in those patients attending a psychiatric day unit who failed to benefit from the treatment. Study 2 explored this relationship further with a nonclinical sample (i.e., commercial airline pilots) and again found a link between personality and stress. Next, the research sought to explore depression more fully in a large data base of patients who had been treated for depression in the early 1960's followed up many years later. Study 3 investigated the pathways that might link depression and early mortality. This study found such pathways were long term, as well as short-term. Study 4 sought to develop this work further by examining the database for suicide rates. The suicide rates were significantly higher for men in the general population, and for depressed patients of both sexes. However, the results also showed that depression may be a particularly strong predictor of suicide risk among women. Study 5 further explored the data base for physical outcomes of depression, in this case, ischaemic heart disease. Significantly higher rates of death from ischemic heart disease before the age of 70 were found among males with endogenous depression. Study 6 furthered this work by exploring the data base for strokes following depression. The findings showed a positive relationship between depression and strokes later in life and suggested that the identification of depressive symptoms at younger ages may have an impact on the primary prevention of having a stroke in later life. The final study returns in part to the earlier work (studies 1 &2) and explores depression with personality (Neuroticism) and self-perception. Higher levels of neuroticism were statistically significantly related to more negative self perceptions, more stringent self expectations, and greater discrepancy between actual and ideal self perception across

multiple domains of behaviour and feelings. Overall, the results of this research program show how personality can influence stress and depression, and that there are numerous long-term health (including mental health) consequences to suffering from depression.

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Commentary:

In the 1959 I became a state registered physiotherapist who wanted to better understand the efficacy of the treatments I was delivering. However, at the time of my training physiotherapy treatment efficacy was measured subjectively as we were not trained in research methods. For me, that would change in 1979 as I enrolled in a university course majoring in management and research. This course was a watershed and an attempt to remedy these deficits by offering a postgraduate course which sought to train senior therapists to both manage and to seek objectivity by learning research techniques. The research aim included learning about the formulation of hypotheses, the need to place the hypotheses in the context of the wider field, statistical analysis, and finally the interpretation of the results.

At the time of completing this research I had worked extensively in general rehabilitation, then neurology, followed by psychiatry, and it was in a psychiatric day unit that I would conduct this first research project. This first research (appendix 1) was produced at a time of rapid change in psychiatry. In 1980, the need to save money on health and welfare necessitated treatments to be evaluated. This initiative was aimed at replacing the bottomless purse of expenditure in the National Health Service with objectivity and efficiency. This stratagem heralded Community Care (despite the fact that some questioned the very existence of the community). Asylums were being closed, and planning was needed to provide for all psychiatric care: those acutely mentally ill and the many who had spent years in wards for the chronically ill. Psychiatric Day Units were part of the alternative provision to cater for the vast numbers of hospital beds occupied by the acute and chronically mentally ill Killaspy, (2006).

The relationship of bodily symptoms to psychological adjustment (please see appendix 1) was an attempt to discover to what extent mental illness is camouflaged by physical symptoms. As a physiotherapist who had worked first in physical rehabilitation, I had seen that the results for patients were varied; however, if a patient failed to improve they were prescribed more of the same. Given the opportunity to dig

deeper by formulating and testing hypotheses was both urgently necessary and personally very appealing. This initiative was especially pertinent as I had recently begun working in the Psychiatric day units. It became noticeable that neither specialties (physical rehabilitation and psychiatry) overlapped regarding treatment: a dichotomy existed between psyche and soma. However for the patients their minds, bodies, lives their disorders were one. Ideally for the assessment and evaluation of the patients, their disorders and diagnoses would include all relevant information.

In my first (unpublished dissertation) I surveyed the psychiatric day unit patients using the Eysenck (1975) Personality Questionnaire (EPQ); the Holmes Rahe (1967) Social Readjustment Rating Scale (SRRS) which was used to measure stress in the year prior to their psychiatric unit admission; the Hugh Bell Adjustment Inventory (1934); and the Bodily Symptom Questionnaire, which was designed by the present author for this piece of research.

The results of this study confirmed as Eysenck (personal communication) pointed out "many anxiety neuroses are camouflaged by physical symptoms and may be seen in other departments." The results corroborated by the case notes demonstrated that some patients had wasted time being treated for physical symptoms or languishing alone before eventually being referred to the psychiatric service. Expensive and scarce resources had been utilized while the patients were passed around from one specialty to another before finally being referred to the psychiatric service. Indeed, the psychiatric day unit proved to be effective in bringing about change in the majority of the mentally ill patients. Eight weeks after discharge from the day unit patients showed overall adjustment in four separate variables: bodily symptoms, the ability to function better: emotionally, in the home, and within society.

One of the conclusions and recommendations following this research was the use of the Eysenck Personality Questionnaire (EPQ) as a relatively easy tool for screening patients when their symptoms were many and potentially suggesting the somatisation of a primarily psychological disorder. This first study provoked further questions: what further clinical phenomena are relevant to having a more comprehensive understanding of how depressed and mentally ill individuals function? Are individual differences in personality relevant? and what were the antecedents of the mental

illness, that is, what made the patients sick? By attempting to determine more precisely the precipitating factors would enable treatment to become more targeted and therefore, more efficient and effective.

However, two central concepts stood out and they would be the focus of my publication, *Stress and Psychoticism* (1981). The initial dissertation had used the Holmes Rahe (1967) Social Readjustment Rating Scale (SRRS). The SRRS was a simple questionnaire which was used to measure life events in the year before admittance to the psychiatric day unit. Eminent authors were pioneering tracks into the hinterland between the mind and body loosely described as links to psychosomatic disorders (e.g. Canon, 1939; Claude Bernard, 1945; Cassel, 1976; and Hinkle & Wolffe, 1957). Cross referencing their publications provided me with the knowledge and confidence to pursue hypotheses which went beyond a diagnosis. The research explored the possible antecedents of conditions. It was the beginning of the biopsychosocial approach that permeates the papers presented in this commentary.

The inclusion of the SRRS to measure stress was because of the strong belief that disorder is an integral part of a complex adaptive system (for a brief review of the science of complexity, see appendix 2). Stress in this research is conceived as part of a nonlinear complex adaptive system which includes a feedback mechanism.

I became particularly interested in stress (Selye, 1974) as I had been running an adaptation of psychoprophylaxis classes in the asylum. When I first worked in the asylum and asked what was most needed I was told that the pressing need was to reduce anxiety. Clearly to make inroads into alleviating anxiety, classes would need to be run to be of any significance impact in such a large hospital. I had attended a course on psychoprohylaxis which I modified as 'relaxation classes'. The aim was a progressive treatment to which all (initially) acute patients were invited to attend, not just those with a diagnosis of anxiety.

Patients would enter my department, remove their shoes, lie supine on a blanket on the floor with a pillow to slightly raise their heads for comfort and cover themselves with a blanket and close their eyes. Teaching them the technique would take half an hour. This was followed by directed imagery to the accompaniment of background

music. After half an hour the patients would be very reluctant to be roused. The remaining time would be spent with the patients sitting round the room on the floor leaning against the walls recalling where they had travelled, what they has seen, felt and smelled during the directed imagery. They frequently gained insight during these times, interpreting the significance of the imagery, sometime crying, sometimes laughing.

The group would collaborate and try to help each other with interpretations which were seemingly elusive. The relaxation success was dependent on repetition - the more they practiced, the faster they could progress and attain a relaxed state. From lying on the floor, they were progressed to relaxing during talking, sitting, and walking. Those patients who had panic attacks were encouraged to counter their irrational fears with the technique.

The success of these classes was patient driven. The physiotherapy department was limited in terms of floor space and the patients would compete for positions. They would lie under the treatment couches, packed in like sardines. Late comers had to be turned away. This enthusiasm spread, everyone appeared to want to join the bandwagon which resulted in other therapists: occupational therapists, nurses, psychiatrists seeking me out wanting to participate to learn the technique. Classes were conducted elsewhere to train others. The next step was the production of a relaxation tape enabling patients to continue the technique, if they went home on leave and when they were discharged.

It began as a means of reducing anxiety but what was it doing in addition? I knew it was doing more: I received feedback from the patients, the ward sisters and the psychiatrists. It was empowering the patients with something tangible to do at a time when they were most vulnerable: in an asylum suffering the stigma and impotence associated with being in an asylum. They were receiving drugs, and ECT as the treatment appropriate for their diagnoses. However, relaxation was under their own control and the more they practiced, the better and more control they had (as well as the reduced need for sleeping medication). The emotions associated with anxiety and being mentally ill instead of being all consuming could be understood managed and eventually alleviated.

I would teach them about the reciprocal nature of the sympathetic and parasympathetic nervous system and explain that stress was natural and healthy but distress was akin to living with one foot on the accelerator all the time, causing wear and tear. This metaphor made perfect sense to them: They were learning to take their foot off the accelerator. The patients were no longer passive recipients of "treatment". They were also helping each other comparing notes of their experience with the imagery and excited to make claims of their progress.

Stress

The concept of stress began in the physical world of materials and the need to understand structure and deformation. Hooke in the 17th century was concerned with the design of structures and the need to calculate load, stress and strain, particularly with the construction of bridges. (https://en.wikipedia.org/wiki/File:Hookeslaw). Hooke's work has been used primarily as a metaphor to influence the concept of stress in the 20th century in various disciplines including psychology.

After the World War II, stress became associated with shell shock (Grinker & Spiegal, 1945), with the need to select recruits who were stress resistant. Selye, (1974) was concerned with external stressors and the physiological reactions, thus, stress was conceived as an external load on a biopsychosocial system. As seen in Table 1, my work (Thomson, 1980) would follow on from this, including internal stressors.

Table 1: Changes in the concept of stress

Hooke - the 17th Century	Selye & Lazarus 1966 - 1974	Thomson 1980
concerned with materials: load, stress & strain. while building bridges & structures. Deformation was concerned with deformation of structure caused by interplay between load & stress differences in metals.	Selye 1974 Concerned with physiological reactions to external stressors ..	Internal & External Stress. Individual differences personality and adaptation, psychoprohylaxix / relationation
Grinker & Spiegal [1945] concerned with the psychodynamic s of breakdown & battle fatigue.	Lazarus c 1966 concerned with appraisal of harm, threat, challenge , and emotions.	

Solomon (1964) was concerned with how neuroimmune interaction occurs. Solomon speculated in a landmark paper: *Emotions, immunity, and disease: a speculative theoretical integration* that the brain and the immune system represent a single, integrated system of defense. Immune alterations in psychotic individuals were reported, including lower numbers of lymphocytes and poorer antibody response to the pertussis vaccination when compared with non-psychiatric control subjects. Later Ader & Cohen (1975) demonstrated classical conditioning of the immune function, and advanced psychoneuroimmunology. They discovered a network of nerves leading to blood vessels as well as cells of the immune system and also found nerves in the thymus and spleen terminating near clusters of lymphocytes, macrophages, and mast cells, all of which help control immune function. However, it was Hans Selye (1974, page 39) an endocrinologist working in Montreal who would pioneer the fuller understanding of stress.

 Selye developed his theory of the three stages of the general adaptation syndrome (GAS). (In his book, *Stress without distress*, where he describes the manifestation of stressors in the whole body, as they develop in time.) The general adaptation

syndrome evolves in three distinct stages: alarm reaction, stage of resistance, stage of exhaustion. He explained the hypothalamic-pituitary-adrenal axis (HPA axis) system which prepares the body to cope with stress. Further, he explored a local adaptation syndrome that includes the inflammatory response and repair processes which occur at the local site of tissue injury as in small, topical injuries, such as contact dermatitis that may lead to GAS if the local injury is severe enough. Here are his three stages:

 "Stage 1: Alarm

Upon encountering a stressor, the body reacts with "fight-or-flight" response and the sympathetic nervous system is activated.

Hormones such as cortisol and adrenaline are released into the bloodstream to meet the threat or danger. The body's resources are now mobilized.

Stage 2: Resistance

The parasympathetic nervous system returns many physiological functions to normal levels while the body focuses resources against the stressor. Blood glucose levels remain high; cortisol and adrenalin continue to circulate at elevated levels, but outward appearance of the organism seems normal. Increase HR, BP, breathing, the body remains on red alert.

Stage 3: Exhaustion

If a stressor continues beyond the body's capacity to cope deprived of resources, the organism becomes susceptible to disease and death."

Like stress, Psychoticism was also barely formulated in 1979. Indeed, Eysenck & Eysenck (1975 page 11) explained that the nature of the 'P' (psychoticism variable in his personality scale:

"can only be guessed at, a high scorer may be described as being solitary, not caring for people; he is often troublesome, not fitting in anywhere. He may be cruel and inhumane, lacking in feeling and empathy, and altogether insensitive. He is hostile to others, even to his kith and kin, and aggressive, even to loved ones. He has an odd liking for unusual things and a disregard for danger; he likes to make fools of other

In 1979, the two subjects of stress and psychoticism were most unlikely bedfellows. Both were comparatively new. Stress was concerned with reactions to external deformation via Selye's (1974) stressors and Psychoticism was a personality trait showing a particular pattern of behaviour involving individual differences among

people. The benefits of neither were known. Since then, much has been discovered, for example, Eysenck (1995) found that those high on psychoticism are indispensable during war and can be relied upon to act with feats of heroism. Indeed, Grinker & Spiegal (1945) who were concerned with the psychodynamics of breakdown and battle fatigue might have done well to embrace the trait of psychoticism. However, if society was looking for higher sociability, those high in psychoticism would be the last chosen.

Publication 1: Stress and Psychoticism (Evans, 1981)

Thus, when the paper on stress and psychoticism was first published it was novel not only for combining the two subjects of stress and psychoticism but also because stress, in this paper, was conceived as being initiated internally. This finding made sense particularly as Lazarus (1994) and Antonosky (1979) failed to solve concerns which were focussed on causes of stress external to the individual. It was not until individual differences were taken into account (Evans, 1980) that personality traits differentiated people in their perception and ability to cope with stressors.

All the data were examined, and high scores on stress were significantly correlated with high scores on psychoticism. Stress and personality had never been linked before. So what did this mean? Correlation is not causation. From the patient's point of view it meant that as a group they did not benefit from attending the psychiatric day unit, it even made them worse. But why? Although many patients did indeed improve in the psychiatric day unit, the next step was to investigate which patients did not adjust following treatment and to determine what, if anything, they had in common.

The explanation which appeared to make sense was that in those patients who became worse, the 'tough-minded' high P scorers did not benefit from the intense pressure generated by the day unit group to conform and the demand to empathize: They had no point of reference. It appeared that the very characteristics, which may have played a causative role in their becoming ill were merely reinforced by the dynamics operating within the group.

At that stage, it was not known if stress and psychoticism were indeed related or whether it was merely a chance finding. However, if accepted, the results found begged the question what was the treatment of choice for the treatment or management of the patients with these specific characteristics? It would need to be one which took account of the personality characteristics of the patient with high psychoticism scores. Indeed, the results found were counterintuitive: why was the typical tough minded high P scorer vulnerable to stress? The life events (SRRS, 1967) had a causal connection with mental illness. Examination of the results showed a preponderance of relationship difficulties: trouble with the law, with neighbours, with spouse.

Although this publication was exciting, it was unpopular because it overturned accepted theory and practice. Even the day unit where the research had been carried out did not consequently adjust the treatment as a result. I would need to move on from this sample. However, at an international conference on psychosomatic disorders in London, the paper was embraced. The demand for reprints internationally was high.

Publication 2: Personality and Stress (Evans, 1985)

The next step following *Stress and Psychoticism* (Evans, 1980) was to examine the connection between personality and stress in a normal group. The author cast around to see where there might be a regular homogenous group. Since there were personal connections with flying, it was known that pilots needed to take regular health screening checks. This intimate knowledge of flying contributed to the design of a questionnaire aimed to find out what caused pilots stress during flights. The saying goes that "There are no old bold pilots" implying that the pilots, for the most part, possess characteristics that make them reliable and stable. The occupational questionnaire included questions that asked the pilots to assess the extent to which various conditions and procedures caused them stress before and during a flight.

The research was conducted in a pilot screening clinic in London. This paper confirmed the connection between personality and stress using a normal cohort of commercial pilots working for various airlines. As a result, this paper was important

as it confirmed the correlation between personality and stress in a healthy control population.

MPHIL (see appendix 3) For Thomson, nee Evans, 1996)

On the strength of the research published so far, the author was accepted to take an MPhil part-time at the Department of Psychology at Surrey University. The MPhil was to be on Stress and Personality. The clinicians who had been involved with the first research asked that the author aid them first in the clinicians' research, claiming it would only take "weeks". The weeks turned into months and the author thus became involved in research into depression.   Running out of time, the author requested that the title of the MPhil thesis should be changed to **Psychosomatic Factors in Premature Mortality Following Depression. A Prospective mortality study of 685 patients diagnosed as suffering from a depressive illness.** This alternative title was accepted.

The connection between stress, personality, depression and mortality does not immediately become apparent. The work of Selye (1974) found that stress whether due to strong emotions like depression or extremes of temperature draws on the adaptive capacity of the body. Further, social factors have also been linked with premature mortality. For example, Berkman and Syme (1979) showed that people who lack social and community ties are more likely to die than those with more extensive contacts. Death during a nine-year follow-up was due to four principle causes: ischaemic heart disease, cancer, cerebrovascular accidents and circulatory disease. These authors concluded that the isolation was an indirect factor in the deaths. However, an important consideration was why were these people isolated? This raised the question that personality and the propensity to live in isolation might relate to premature death.

The connections following these studies between stress, personality, and depression and premature mortality became convincing and opened up the possibility of further research. It is interesting to note that in a recent prospective study Anderson et al, (2015) depression was found to relate to an increased risk of infection. This finding is particularly pertinent to the MPhil (which was published as Type of depression and results of mortality, Thomson (nee Evans), 1996 (appendix 3) where respiratory

disease was a significant cause of mortality. But also because it adds credence to the biopsychosocial model which this work is so committed to promoting.

Although the MPhil data are not included as part of this thesis, the initial study became the foundation for twenty years work in exploring and expanding this data set. Since the resulting publications after the initial analysis and publication are included in this thesis, a detailed explanation of the data set will be explained here.

The author was very privileged to be allowed access to data collected at the Medical Research Unit (MRC) at Graylingwell Hospital by the late director Dr. Peter Sainsbury. This permission was as a result of the unpublished paper which Dr. Sainsbury read (please see Appendix 1). Availability of these data enabled a prospective follow-up study of depressed patients to investigate premature death following clinically diagnosed cases of depression. Although the M.R.C data were of the highest quality, at the time of gathering the data the M.R.C was in mothballs. There was a wealth of data available for use in many dusty filing cabinets. Gathered before the era of computers by eminent researchers in the field and before statistical packages such as SPSS. All data were on sheets containing data for each patient gathered in 1960 - 61.

The data needed to be transferred and coded using binary coding for each patient separately, 685 patients in all. The statistician at Wessex Regional Health Service worked with the author over many days to achieve this. Over the years, different classifications of illnesses had been in operation. The original patients dying in 1960 were classified using the 7th World Health Organisation coding, whereas the code in operation in 1986 was for the 9th. Accordingly every cause of death had to be reclassified. This recoding was a time-consuming activity but essential for the accuracy of the research. Every cause of death listed in the data was checked by a medical consultant. In some cases, it was necessary to reallocate the death code. All the control population were classified according to the 9th classification.

The National Health Service Central Register (N.H.S.C.R) was set up to help local Family Practitioner Committees (F.P.C.) carry out registration work efficiently by

maintaining a central register of all National Health Service patients in England and Wales. Each patient is identified by a number, name, sex, and date of birth, and a symbol that denotes the area in which the person is currently on an N.H.S doctors list. Removal from the list due to death and leaving the UK is routinely noted against an individual's name. The N.H.S.C.R. can thus be used as a tracking device for follow-up studies. The researcher made an application to the N.H.S.C.R with the submission of a protocol. Once the protocol was approved information had to be supplied on index cards for each patient in the study. One card was retained and a second was sent to the N.H.S.C.R until in this case death occurred and the card was returned to the researcher.

The MPhil research was designed to determine whether premature death could distinguish between the two diagnoses, reactive and endogenous depression. At the time, The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-4) in 1987 was in use. The controversy at the time was concerned with whether there are one or two types of depression. The traditional view was that there was a neurotic or reactive depression following precipitating causes whereas endogenous depression was an illness that appears for no apparent reason in patients with well-balanced premorbid personalities. Endogenous depression was considered to be caused by some internal metabolic imbalance; this process distorted mood and often reality leading to psychotic symptoms. In reactive depression, there was no distortion of reality. The results of the MPhil study confirmed that endogenous depression carries the greatest risk of dying prematurely. This result gave credence to the two diagnoses but sadly not before the decision was taken by DSM 1V and ICD 10 revisions to consider depression as one discrete category rather than two separate entities. Thomson (1996, appendix 3) concluded by recommending that the patients should be assessed before discharge and to consider the period after discharge as a crucial and a vulnerable time for the patient. However another concern following the results was that premature mortality was not yet accepted as having a causal relationship with a depressive illness. If the seriousness of the results had been taken as part of the natural history of a depressive illness, then depression could expect to be treated in a similar vein to any life-threatening illness such as heart disease or cancer with the accompanying resources.

Indeed, it is only recently that this issue has been taken seriously. For example, the following is a quote from Lépine and Briley (2011, pp. 3) on the increasing burden of depression:

> The mortality of depression as a research subject is becoming even more topical. Epidemiological surveys conducted in general populations have found that the lifetime prevalence of depression is in the range of 10% to 15%. Mood disorders, as defined by the World Mental Health and the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, have a 12-month prevalence which varies from 3% in Japan to over 9% in the US. A recent American survey found the prevalence of current depression to be 9% and the rate of current major depression to be 3.4%. All studies of depressive disorders have stressed the importance of the mortality and morbidity associated with depression. The mortality risk for suicide in depressed patients is more than 20-fold greater than in the general population. Recent studies have also shown the importance of depression as a risk factor for cardiovascular death. The risk of cardiac mortality after an initial myocardial infarction is greater in patients with depression and related to the severity of the depressive episode. Greater severity of depressive symptoms has been found to be associated with significantly higher risk of all-cause mortality including cardiovascular death and stroke. In addition to mortality, functional impairment and disability associated with depression have been consistently reported. Depression increases the risk of decreased workplace productivity and absenteeism resulting in lowered income or unemployment. Absenteeism and presenteeism (being physically present at work but functioning sub optimally) have been estimated to lead to a loss of $36.6 billion per year in the US. Worldwide projections by the World Health Organization for the year 2030 identify unipolar major depression as the leading cause of disease burden.

Death may now be considered as part of the natural history of depression (Neeleman, 2001). The risk factors associated with depression including cardiovascular disease and stroke are also now recognized by W.H.O. (Silverstein & Angst, 2015). Since embarking on this research in 1988 and then in 1996 neither of these points was the

accepted view therefore it is hoped that my research made a strong contribution to the recent progress made.

The next step was to revisit the data and conduct a 49 year follow up. Very few prospective studies of clinical depression extend beyond 20 years. It was felt incumbent upon the author to take the next step.

Publication 3: Lifting the shroud on depression and premature mortality: A 49 year follow - up study (Thomson, 2011)

The above publication followed-up patients in the mortality MPhil study previously submitted as an MPhil thesis, which was a 24-year follow-up study.

Thomson (2011) was among the few studies to follow patients prospectively for an extended follow-up of 49 years. The paper concluded confirming the short - and long-term association between depression and premature mortality. It also admitted the pathways linking depression and premature death were likely to be complex and multifactorial in nature. This paper continues to attract widespread interest since its publication.

Following Thomson (2011), the opportunity and enthusiasm engendered by its success energized the following articles on causation of various physical conditions (such as, stroke, ischaemic heart disease, and suicide) Thus, although the papers originated from the same cohort, the papers were focused on different aspects of this rich data set. Each cause of death demanded the need to revisit the data and re-analyse the results.

Publication 4: Long - term follow up of suicide in a clinically depressed community sample (Thomson, 2011)

This work is concerned with the need to see the patient and his/her disorder from a biopsychosocial point of view. In his book *Suicide: A Study in Sociology*, Emile Durkheim (1952) categorized suicide into egoistic, altruistic, and anomic suicide. Egotistic suicide follows a lack of integration into society, thus when society must 'pull together' (such as, in crises) the suicide rate falls, for example, during war. Altruistic suicide occurred where demands for benevolence and discipline for

obedience are high, for example, prison military and religious organizations. Anomic suicide is characterized by having too little control, for example, any powerless situation.

This paper on suicide was interesting as depression was significantly higher in this group than in the general population for both men and women and also depression was an especially strong predictor of suicide in women. Of the sixteen women who committed suicide, eleven took their lives in the first decade following admission to hospital for depression. As Durkheim's sociological concept proposes the lack of integration as symptomatic of his egotistical suicide as well as Berkman and Syme (1979) report loneliness as a predictor of depression, these together may suggest that admission to hospital followed by death relatively soon after discharge carried the additional burden of stigmatization, and loneliness. Clearly there is a need to identify those patients most at risk of dying prematurely from suicide. The women who committed suicide had all been in   hospital and had received treatment but despite this these patients remained mentally ill. The deaths of these patients even begged the question did therapy and hospitalization play a causative part in premature death?

Cameron & McGoogan (1981) advised some caution in using death certificates when individual death certificates were used to confirm a particular cause of mortality. However, whatever the causation, the overall mortality was alarming. Further, it is important that depression is not seen in isolation here, the more serious the depression, the more stress, the more drugs, perhaps the more familial conflict etc.

Publication 5: The Head Stands Accused by the Heart!. Depression and Premature Death from Ischaemic Heart Disease (Thomson, 2014)
This paper explored ischaemic heart disease as a cause of death following a depressive illness. Both illnesses are very significant contributors to the global burden of disease with 7.3 million deaths due to heart disease annually and depression affecting annually 350 million people globally (W.H.O, 2008). The study found clear evidence for clinical depression and premature mortality from Ischaemic heart disease in the context of sex and type of depression. Men with endogenous depression appear to be at particularly elevated risk for premature mortality from IHD.

Publication 6: The rate of Stroke Death after Depression: A 40 - year Longitudinal Study Extension of the Chichester / Salisbury Catchment Area Study (Thomson, 2014)

The next pressing need was to determine the underlying causal mechanisms after 40 years follow up. To this end, the data and death certificates were reanalysed to determine the cause of mortality. The conclusion drawn was that depressive symptoms at younger ages may have an impact on the occurrence of stroke later in life. The notion that depression has stronger effects over a long period is consistent with the view that severe clinical depression and physical illness occur concurrently, one exacerbating the other, and health is degraded through slow acting cumulative processes.   This rationale is consistent with Selye's stress hypothesis, and the third stage of the G.A.S.

Overall, the large prospective data-base of the 685 clinically diagnosed depressed patients was a rich source of information, but was not without its limitations. There were five main issues (social change, gender bias, medication, age differences between diagnoses and accidental death) that need to be considered relating to the circumstances at the time the data was collected and changes which have taken place in the intervening years.

First, Social change. In 1960 -1961 when the data were originally collected in England, experiences and memories of the Second World War were still vivid for the older generation. The war had changed society, economics and the minds of its people. Many men had lost their lives and many others returned home mentally and physically damaged by the horrors they had to endure. Adjustment once they did return was not always easy. Women before the war were usually housewives dependent on the husband as the breadwinner. But during the war they showed their resilience and their ability: they both could earn and run the home. When men returned home, women's position in the household was expected to return to pre-war status. For some this was not possible. By the early 1960's women's roles in society were beginning to be challenged, as were issues of racism resulting in social change that would not fully be realised until much later.

Second, gender bias. Gender bias is acknowledged by the W.H.O: Mental Health (2008). Doyal (2001) Doctors are more likely to diagnose depression in women compared with men, even when they have similar scores on standardized measures of depression or present identical symptoms". This bias was much more prevalent in 1960's when this research data was collected. Qin (2000) maintains the risk factors of suicide differed according to gender. Unemployment played a more important role in predicting suicide for men. But a history of hospitalized mental illness was the most marked risk factor for suicide for both men and women, which Hawton (2000) considers warrants more research and initiatives to develop gender specific approaches in order to guide clinical practice and prevention strategies in both genders. Thus, the hospitalisation of women with depression, may also have increased their feelings of hopelessness and lack of control over their lives thus, amplifying the depression, which may have appeared to confirm to the diagnosing psychiatrists that they were correct in their initial diagnosis, when in fact their actions had created the level of disorder.

The older antidepressants tricyclic's (TCAS) and monoamine oxidase inhibitors (MAOI's) were in use in 1960. Feighner JP (1999):

> "The psychopharmacology of depression is a field that has evolved rapidly in just under 5 decades. Early antidepressant medications--tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs)--were discovered through astute clinical observations. These first-generation medications were effective because they enhanced serotonergic or noradrenergic mechanisms or both. Unfortunately, the TCAs also blocked histaminic, cholinergic, and alpha1-adrenergic receptor sites, and this action brought about unwanted side effects such as weight gain, dry mouth, constipation, drowsiness, and dizziness. MAOIs can interact with tyramine to cause potentially lethal hypertension and present potentially dangerous interactions with a number of medications and over-the-counter drugs. However, these drugs were associated with numerous side effects, and in some cases dangerous dietary interactions".

Currently, the first treatment of choice for depression is Selected Serotonin Reuptake Inhibitors (SSRI's), and Serotonin Norepinephrine Reuptake Inhibitors (SNRI's) which carry less side effects when compared with the first generation drugs. Indeed,

the treatments for depression in the 1960's also included far greater use of electric convulsive treatment, especially when admitted to hospital and these older generation of drugs, the ECT and hospital admissions may have played a causal role in premature deaths of my participants.

Fourth, the average age differences between the reactive and endogenous depressive patient at referral were 44 years and 58 years respectively. At first glance, it may appear that the reason endogenous depressives had higher mortality rates was that they were indeed older, and therefore more likely to die. However, the research utilized actuarial information about age-adjusted death rates. Expected death rates were computed separately by age grouping and gender for each year of the research.

Fifth, and finally, accidental death rates. When exploring suicide rates, often women are over-represented as their deaths can be more easily classified as suicides, whereas men may kill themselves in, for example, single vehicle accidents, and their death would be classified as accidental death. Thus, accidental death rates may be useful to examine along with suicide rates. Further, Holding & Barraclough (1974) report the importance of considering accidental deaths when the mortality of mental illness is studied. Of 110 accidental deaths, 60% were diagnosed as mentally ill.

Next, I moved away from this large data set and started focusing more on why patients died following hospital discharge not only by suicide but from natural causes. Furthermore, what part did personality and individual differences play? Intuitively my thoughts were turning to a hypothesis which considered the possibility that the premature death following depression could be via two routes: One unnatural and intentional death, the other invoking an unconscious mechanism via death from natural causation. Selye hypothesized that the third stage in his G.A.S resulted in death by exhaustion. Was this the cause of the premature mortality? Was the premature mortality the outcome of patients becoming exhausted by their mental illness and giving up? Or were there personality issues, such as self-defeating attitudes, feelings and behaviours that aided this 'giving up'.

Publication 7: Depression, Neuroticism, and the Discrepancy between Actual and Ideal Self (Thomson, 2016)

To explore this in more depth, the present author created a questionnaire to measure self-defeating attitudes, feelings and behaviours. It is called The Self-Defeating Quotient (SDQ, please see appendix 5). The SDQ was designed as a biopsychosocial questionnaire to test hypotheses and to elucidate further the questions raised by the research conducted so far. The main aim of the SDQ is to explore actions, attitudes, feelings and behaviour which personally sabotage and are self-detrimental.

Baumeister (1988) describes three models of self-defeating behaviours. First, the primary self-destruction deliberate and intentionally hurting themselves. Second, the conceptual model known as 'trade off when people will deliberately do something that will harm them so that if they fail they are able to blame their failure on the bad choice they previously made. Third, self-destructiveness which includes counterproductive strategies. The person neither desires nor foresees the harm to self. In this instance a person is pursuing a desirable outcome but chooses a strategy or approach that backfires and produces the opposite of the desired result.

Twenge et al (2002, p 59 - 71) "Factors that contribute to self – destructive behaviours include low-egos, low self-esteem and seclusion. Results from various studies propose that a strong feeling of social inclusion is important for enabling the individual to use human capacity for self-regulation in ways that will preserve and protect the self and promote the self's best long term interests and well-being."

Currently many psychiatrists use the Maudsley History Taking Assessment to assess patients and to arrive at a diagnosis. Every new patient referred to a psychiatric service can be expected to be examined by a psychiatrist for approximately an hour. Though this may depend on a particular service, area, and the training and preferences of the psychiatrist. That time is used in an assessment which includes:
1. Presenting Complaint
2. Family History
3. Personal History
4. a) Education. b) Employment.
5. Past medical history.
6. Personality including pre-morbid
7. Formulation

8. Diagnosis

9. Treatment

The rest of the hour can be spent writing to the patient's referral source. Thereafter patients received approximately 20 minutes of time depending on the resources available.

The SDQ was designed to: augment the clinical assessment, inform the patients and clinicians and to provide more detailed information in aspects of the patients lives. Fortunately, two psychiatrists who were interested in the research agreed to provide patients to enable the pilot study to take place. When the questionnaires were scored (invariably on the evening of the appointment) the results sometimes rang alarm bells sufficient to alert the psychiatrist. On some occasions, the psychiatrist would recall the patient.   He would also sometimes decide to change the diagnosis or change the medication. The SDQ appeared to be picking up valuable information which the Maudsley Assessment did not.

There are many theories of depression, but Lazarus (1994) describes the way in which depression provokes emotions. The results of the SDQ in a depressed population confirmed that emotions have a causal relationship with a depressive illness. This is a potentially fertile area of study and the utilization of the SDQ.

This paper is the first validation paper of the SDQ. Further, the author was invited by the American Psychological Association to include the SDQ questionnaire in their online test database. It is hoped it will become a tool for use in clinical situations as an assessment, therapeutic aid, and also for research purposes. But even more important the SDQ could play an informative and preventative role providing potential patients with personal insight.

Concluding Reflections:

At the outset of my career as a therapist and then as a researcher, a dichotomy existed between the mind and body psyche and soma. The psyche was the domain of psychiatry and the body was the domain of the rest of medicine. As my understanding evolved, it became obvious that these were artificial and arbitrary distinctions that did

not represent the patient / client. 15 years after my first research (Appendix 1) when I had first began to question the then existing medical model of disease the diagnostic criteria for psychosomatic research was proposed. An international group of investigators based on the recognition from a wide body of evidence accumulated in psychosomatic medicine. This evidence included the concepts of quality of life, stressful life events, somatization, and personality disorders (DCPR). Fava et al (1995) stated that "The DCPR rationale was to expand the traditional domains of the disease model by translating psychosocial variables that derived from psychosomatic research into operational tools. Since that time DCPR have undergone extensive validation. Fava et al (2012) went further to say that "the lesson from the DCPR literature points to the clinical utility of expanding the traditional symptoms based psychiatric taxonomy or biomedical disease model. Future challenges concern the joint use of the DCPR and neurobiological investigations to fill the gap between bio markers and clinical judgment and the application of specific treatments to the DCPR, improving Quality of life and clinical outcomes".

What my research has shown is that while there are certainly psychiatric illnesses and physical illnesses, they may each cause the other to arise. I conclude that the significance of the work presented here is not the research papers relating to the individual causes of death (important as they are), but of the overall mortality and morbidity. What has become increasingly apparent to me is the combination of all factors and variables relating to the human condition discussed in this body of research. As this work demonstrates from the earliest paper reported (Stress and Psychoticism, Evans, 1981) to the latest (Depression, Neuroticism, and the Discrepancy Between Actual and Ideal Self-Perception, Thomson, 2016), individual differences are so central to the lifestyle choices open to the individual that the focus must be on avoiding physical and mental illness by being selective and choosing wisely and judiciously between lifestyle choices. It is the impact that cognition and the choices made which, in turn, impact on physical and mental health.

For the researcher the responsibility lies in presenting the facts with honesty and integrity. The model most likely to combine all these is by using the new science of complexity which may help put the individual at the centre. In the commentary I touched briefly on the new science of complexity (Appendix 2) as a contender for a

new non-linear model of order and disorder. This model has the capacity of embodying all contributing diagnostic information thus reducing error. It also has the capacity to combine the traditional with any new and future neurobiological or genetic research to bridge the gap between clinical judgment and the application of specific treatments.

This bridge between clinical judgement, treatment and research is sorely needed. As a researcher I have been disenchanted by research based on meta-analysis whereby well designed research can be excluded according to the whims of the researcher. Where retrospective and prospective research are given equal weight.

As a therapist I have learnt to listen to my patients to fit the treatment to them. I have also been saddened to realize that some physical and mental disorders, now that the major infections have been eliminated, are self-inflicted and therefore could be prevented.

Bearing all this in mind it appears to me that to prevent and reduce 'disorder' in its widest sense may be the remit for educators, to provide children throughout their educative and formative years with a curriculum aiming to prepare them for life. This is not to assume control and take away the responsibility of the parents but rather to work with the parents while also helping those children who lack parental concern and control. Enabling children to make informed decisions about living in society in a harmonious and orderly manner and for them to be aware of the consequences if they do not.

In my work as a family therapist I was alarmed that family dynamics operated to scapegoat children at very young ages, driving them to become involved with substitute family peer groups into delinquency and future failures. It felt like witchcraft in the twentieth century - sinister in its destructive influence on children as young as five. A well-designed curriculum could make sense of the negative impact the subjection of these influences has on young lives. Untangling such influences in therapy in later life when they have impacted negatively is an option but prevention is much to be preferred.

For all those involved with the treatment of disorder, they have the responsibility and accountability to treat using all information available. However, individual differences do need to be recognized, ideally treating the patient as an individual. Policy makers concerned with cost effectiveness will no doubt argue the economic case that this is too expensive. My experience has influenced my opinion that a health service where the individual is the focus would be cost effective.

Since embarking on the current work, two single-author research papers have been accepted for publication, both further exploring depression (one in Journal of Depression & Anxiety and the other in Journal of Clinical Depression). There are two further research papers awaiting acceptance: a) A research paper proposing a self-defeating trait, and b) a paper exploring a new model of disorder: embracing complexity theory. A book protocol has been submitted to Karnac Publishers (titled 'Family Matters: Making psychic pain tangible'). I have also mindful of the correlation between mirror neurons and lack of empathy while presenting this work. I am interested to pursue this line of investigation revisiting my work on stress and personality. I would like to research the connection between stress, psychoticism, mirror neurons, and suicide. I think this is an exciting area of research. I am optimistic that if my hypothesis is correct then at last it may be possible to predict those most at risk of suicide.

In summary there is a lot to be done, but hopefully I am wiser than I was when I first caught the research bug back in my youth. Furthermore perhaps I can avoid the mistakes and regrets of the past and move on with confidence to improve the commitment to progress and making a difference in the lives of not just those struggling with disorder but also helping to prevent disorder.

Notes on the Appendices.

Appendix 1 is an opinion expressed by Dr. Peter Sainsbury he was the director of the Medical Research Unit based in Graylingwell Hospital Chichester. I felt I wanted to include this letter because it was my first introduction to Peter and because of my research he allowed me to use the data which became the basis of the mortality studies.

Appendix 2 this is a very brief explanation of the science of complexity and adaptive systems. Once I read about the new science of complexity it had a profound effect on the way I perceived nonlinear systems theory. I feel very strongly that is this was applied to the problems associated with disorder then the likelihood of solutions would be enhanced.

Appendix 3

The following paper was placed in the appendix because it couldn't be included in this thesis.

Wendy Thomson (1996) Types of depression and results of mortality Pers. & Indiv. Diff. V0l. 21 No. 4, pp. 613-615, 1996

Appendix 4

World Health Organization Information sheet

Premature death among people with severe mental disorders1 Key facts

When I first began research my research confirmed hypothesis I made at the time they not accepted generally. This paper shows that in later years they became generally accepted. It is a vindication of my work.

Appendix 5

The Self Defeating Quotient is a full copy of the questionnaire discussed in the paper Wendy Thomson (2016) Depression, Neuroticism, and the Discrepancy Between Actual and Ideal Self-Perception Personality and Individual Differences Volume 88, January 2016, Pages 219–224

References for the commentary.

Ader, R., Felten, D. L. & Cohen, N. (2006). *Psychoneuroimmunology, 4th edition*. New York: Academic Press.

Ader, R., & Cohen, N. (1975). Behaviourally conditioned immunosuppression. *Psychosomatic Medicine, 37* (4), 333 – 340.

Antonovsky, A. (1979). *Health, Stress and Coping*. San Francisco: Jossey – Bass.

Ash, G. M., (1998). *Gestalt Psychology in German Culture. 1890 - 1967: Holism and the Quest for Objectivity*. Cambridge: Cambridge University Press.

Barraclough, B.M., Bunch, J., Nelson, B. & Sainsbury, P. (1974). A hundred cases of suicide: Clinical aspects. *British Journal of Psychiatry, 125*, 355 – 373.

Berkman, L.F. & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of the Almada County residents. *American Journal of Epidemiology, 109,* 186 – 204.

Bernard, L., Busfield, JR., Wechsler, H., & Barnum, W.J. (1961). Physiological differentiation of reactive and endogenous depression. *Archives of General Psychiatry, 1 (5)*, 472-477.

Baumeister, R.F., & Scher, S. (1988). Self - defeating behaviour patterns among normal individuals: Review and analysis of common self – destructive tendencies. *Psychological Bulletin, 104* (1), 3 – 22.

Cameron, H. M. & McGoogan, E. (1981). A prospective study of 1152 hospital autopsies: 1 Inaccuracies in clinical diagnoses and their significance. *The Journal of Pathology, 133* (4), 285 - 300.

Canon, W.B. (1939). *Wisdom of the Body*. New York: Norton.

Cassel, J. (1976). The contribution of the social environment to host resistance. *American Journal of Epidemiology, 104* (2), 107 -123.

Copeland, J. R. M. (1984). Reactive and endogenous depressive illness a five-year outcome. *Journal of Affective Disorders, 6,* 153 – 162.

Checkley, S.A., Glass, I.B., Thompson, C., Corn, T. & Robinson, P. (1984). The GH response to clonidine in endogenous as compared with reactive depression. *Psychological Medicine, 14* (4), 773-777.

Doyal, L. (2001). Sex, gender, and health: the need for a new approach. *British Medical Journal, 323,* 1061 – 3.

Durkheim, E. (1952). *Suicide: A Study in Sociology*. London: Routledge.

Evans, W.R. (1980). Stress & psychoticism. *Personality and Individual Differences, 2,* 21-24.

Evans, W.R. (1985). Personality & stress. *Personality and Individual Differences, 7,* 51-253.

Eysenck, H.J. (1967). *The Biological Basis of Personality.* Springfield Illinois: C. Thomas.

Eysenck, H.J. & Eysenck S.B.G. (1969). *Personality Structure and Measurement.* London: Routledge.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire (Junior and Adult).* Kent, UK: Hodder & Stoughton.

Fava, G.A., Freyberger, H.J., Bech, P., Chrisodoulou, G., Sensky, T., Theorell, T., & Wise, T.N. (1995). Diagnostic criteria for use in psychosomatic research *Psychotherapy Psychsom*, 63, 1-8.

Fava, G.A., Sonino, N., &Wise, T.H. (2012). *The Psychosomatic Assessment.*

Strategies to Improve Clinical Practice. Basel: Karger.

Feighner, J.P. (1999). Mechanism of action of antidepressant medications. *Journal of Clinical Psychiatry, 60* (4), 12-13.

Hawton, K. (2000). Sex and suicide: Gender differences in suicidal behaviour. *British Journal of Psychiatry, 177*, 484-485.

Hinkle, L. E. & Wolff, H. G. (1957). The nature of man's adaptation to his total environment & the relation of this to illness. *Archives of Internal Medicine, 99,* 442.

Hirschfield, M.A. (1980). Situational depression: validity of the Concept. Presented at the World Psychiatric Association Symposium: What is a case? Institute of Psychiatry London.

Hooke's Law *retrieved from* https://en. Wikipedia.Org/wiki/File:Hookeslaw.ogg

Hyman, SE. (2012). Revolution stalled. *Science Translational Medicine, 4,* 155cm11.

Jung, C. G. (1969).*The Nature of the Psyche*. London: Routledge.

Killaspy, H. (2006). From the asylum to community care: learning from experience. *British Medical Journal, 79-80* (1), 245-258.

Lader, M. H. (1981). *Focus on Depression*. Bencard

Lancaster, M.A. & Knoblich, J.A. (2014). Generation of cerebral organoids from human pluripotent stem cells. *Nat Protoc,* 9(10), 2329-2340.

Lazarus, R. S. & Lazarus, B.N. (1994). *Passion & Reason: Making Sense of our emotions.* Oxford: Oxford University Press.

Lépine, J.P. & Briely, M. (2011). The increasing burden of depression.

Neuropsychiatric Disease and Treatment, 7 (1), 3–7.

Lewin, R. (1992). *Life at the Edge of Chaos*. New York: Collier Books.

Lovelock, J. (1988). *The Ages of Gaia: A biography of our living earth.* Oxford: Oxford University Press.

Munos, B. H. (2013). Pharmaceutical innovation gets a little help from new friends. *Science Translational Medicine, 5,* 168.

Qin, P., Agerbo, E., Westergard-Nielson, N., Eriksson, T., & Mortensen, P. (2000). Gender differences in risk factors for suicide in Denmark. *British Journal of Psychiatry*, *177,* 546-550.

Querido, A. (1959). Forecast and Follow – up. *British Journal of Preventative Social Medicine, 13,* 33.

Rutter, M. (1972). *Maternal Deprivation Reassessed*. London: Penguin.

Solomon, J.P. & Moos, RH. (1964). Emotions, immunity, and disease: a speculative theoretical integration. *Archives of General Psychiatry. 11*, 657 – 74.

Selye, H. (1974) *Stress without distress*. London: Hodder & Stoughton.

Twenge, J., Cantanese, K. & Baumeister, R.F. (2002). Social exclusion causes self – defeating behavior. *Journal of Personality and Social Psychology, 83(3),* 606-615.

Sullivan P.F., Daly. M.J., & O' Donovan, M. (2012). Genetic architecture of psychiatric disorders; the emerging picture and its implications. *Nature Rev. Genet.* 13, 537 – 551.

www.who.int/mentalhealth/management/infosheet.pdf

Chapters

1.   Wendy Evans (1980). Stress and psychoticism   *Journal of Personality and Individual Differences, 2,* 21-24.

2.   Wendy Evans (1985). Personality and stress. *Journal of Personality and Individual Differences, 7*(2), 251 -253.

4.   Wendy Thomson (2011) Lifting the Shroud on Depression 2011 Journal of affective disorders. 130 pp. 60-65

5.   Wendy Thomson (2012) Long-term follow-up of suicide in a clinically depressed community sample. Journal of affective disorders 139 pp. 52 -55.   (This paper accepted for presentation at the International Society of Affective Disorders).

6.   Wendy Thomson (2014) The head stands accused by the heart! Depression and premature death from ischaemic heart disease (ID: 2720024) *Open Journal of Depression, 3,* 33-40.

7.   Wendy Thomson (2014) Rate of stroke death following depression: a 40-year longitudinal study extension of Chichester / Salisbury Catchment Area Study. *Journal of Stroke & Cardiovascular Disease, 23*(7), 1837 -1842.

 8 Wendy Thomson (2016) Depression, neuroticism, and the discrepancy between actual and ideal self-perception. *Personality and Individual Differences, 88,* 219–224.

STRESS AND PSYCHOTICISM

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Summary—The EPQ, a stress questionnaire, and a bodily symptom questionnaire were completed by 21 female and 12 male psychiatric patients. The P score correlated with a high stress score, and certain stress items differentiated extreme high and low P scorers. In addition, high P scorers were found to have a tendency to increase, rather than decrease some of their bodily symptoms after treatment. A different approach to treatment for high P scorers is suggested.

INTRODUCTION

The importance of personality in the determinant of disease is rarely recognised outside the realm of psychology, and psychiatry. Even within psychiatry criticism can be levelled at the blanket approach to treatment, without due regard to the part individual personality dimensions play in the way an individual reacts to stress. There is evidence, that disease can reflect personality; for example Sainsbury (1960) found that patients attending clinics at a general hospital, showed significant differences in personality variables: patients receiving treatment for warts were found to be significantly more neurotic, and more extraverted than controls, reflecting a hysterical pattern, whereas cases of prolapse, reflected the dysthymic personality (high neuroticism, low extraversion). The fracture clinic was particularly interesting; the mean extraversion score was significantly high, while the mean neuroticism score was low. Thus it was reasonable for Sainsbury (1960) to conclude, that the existence of certain personality dimensions may make one accident prone (see also Shaw and Sichel, 1971).

A relatively new dimension of personality, the psychoticism, or tough-mindedness dimension, may also be relevant (Eysenck and Eysenck, 1975). This dimension is quite independent of extraversion and neuroticism and it is believed that genetic factors contribute more to individual differences, than do environmental factors. High 'P' scorers as described by Eysenck are: solitary individuals, not caring for people, often troublesome, not fitting in anywhere, lacking in feeling and empathy, insensitive, hostile, aggressive and impersonal sexually. Criminals and psychotics have high P scores, while the psychiatric disorders which typify the above behaviour patterns are: drug addicts, alcoholics, schizoid, psychopathic and behaviour disorders.

It is generally thought that stress is generated within society, which in turn generates strain in the individual, and that different personality types differ in their vulnerability to stress. What might be extremely stressful to one might not be so to another. Another way of looking at stress is to hypothesize that certain individuals, such as those scoring high on P, actually generate their own stress. Their life-style eventually catches up with them. They are surrounded by the results of their own behaviour and their need for arousal. They act with some disregard for danger, as their impulsivity supplants concern for the consequences of their actions, to themselves or to others.

An opportunity arose to test this hypothesis, while the author was exploring the relationship of bodily symptoms to psychological adjustment, in a psychiatric day hospital.

PROCEDURE

Permission was obtained to invite each new day-patient at a small psychiatric day hospital, to participate in the inquiry. All admissions to the day hospital between October 1978 and May 1979 were approached, with the exception of a group of ten

Table 1

	P	Stress
Female 21		
Mean	3.14	180
S.D.	3.3	118
Correlation (P and Stress)	0.459	
Male 21		
Mean	6.16	261.1
S.D.	5.09	118.8
Correlation (P and Stress)	0.539	

patients who were admitted during a time when for personal reasons, the author was not available.

The day hospital where this study took place, does not adhere to any one particular method of treatment. It maintains a somewhat ecletic approach, but with a psychothera-peutic bias. On admission, the patient becomes a member of a group, which operates to closely resemble a family. This facilitates depth of feeling, sharing, developing loyalties, and if necessary inflicting sanctions, on those within its influence. A treatment pro-gramme is planned, in which all the members of the group participate. Thus, in this treatment setting, all are accountable for their own behaviour, with overall accountability to the psychiatrist in charge.

Every new patient, on the second day at the Psychiatric Day Hospital, was asked if he would take part in a study. The patient was then given three questionnaires.

(1) The Eysenck Personality Questionnaire (1975).
(2) The Holmes–Rahe Life Event Score (1967). This provides a social adjustment rating scale (S.A.R.S.), which indicates stress due to life events, one year prior to admission. A score of 200 or more indicates vulnerability to mental illness.
(3) Bodily Symptom Questionnaire. This questionnaire was developed especially for this study by the author. It aims to measure a number of symptoms, the majority of which are due to autonomic arousal.

The overall sample comprised 21 females and 12 males whose ages ranged from 20–70 yr. The sexes were analysed separately, because the mean P score for normal males is higher than for normal females. In some calculations in the tables there are less than the full sample, because some patients failed to complete all questionnaires.

RESULTS

Table 1 shows that a relationship exists between P and stress in both the males and females significant at the $0.05 > p < 0.02$ level.

In order to further examine the relationship the samples were divided into extreme high P, and low P scorers.

Table 2 shows the comparison of the 14 high P scorers with the 11 low P scorers. The differences in stress scores are highly signficant.

The 43 items which were weighted in the S.A.R.S. and designed to measure stress, were then scrutinized to find which items were most typical and most often endorsed in the high P group. When the 12 items selected (Table 3) were scored for the two extreme groups, males and females together, the correlation with the P score was found to be 0.634. It would then appear that these 12 items from the S.A.R.S. indicate the type of stress likely to be found in the high P group.

Of additional interest is the question of bodily symptoms. Both the high and low P groups showed high levels of autonomic arousal, as measured by the bodily symptom questionnaire. There was, however, an important difference; whereas the low P group showed a positive response to treatment at follow-up there being fewer bodily symptoms

Table 2.

	Female						Male				
	P	E	N	L	Stress		P	E	N	L	Stress
Low P (8)						Low P (3)					
Mean	0.12	5.6	16	12.2	97	Mean	1	6	18.6	7.6	156
S.D.	0.3	4.6	4.8	4.73	46.4	S.D.	0.81	1.6	0.9	3.7	71.6
High P (7)						High P (7)					
Mean	7.2	10.8	21.2	5.7	250	Mean	9.1	9.4	19.7	6.4	324.8
S.D.	2.0	5.11	1.03	2.7	150	S.D.	4.6	4.1	2.2	6.4	102.3

P = Toughminded score.
N = Neuroticism.
E = Extroversion/Introversion score.
L = Lie score.

than on admission, this was not so with some of the high P scorers who collected extra bodily symptoms. A correlation between P and extra bodily symptoms resulted in $r = 0.342$.

In Table 2 it will be seen that those patients in the low P category are also exceptionally high on L, particularly the females. This might be worth investigating further in a subsequent study, because one possible effect of this could be that dissimulators (who score high on L and exceptionally low on N) would claim fewer symptoms both before and after treatment than they really possess. This argument could also account for the very considerable difference between the stress score of high and low P scorers.

DISCUSSION

A positive relationship has been demonstrated between the P factor and stress, it must now be considered whether this is likely to be causal or coincidental. Does for example, the presence of stress lead to high P or *vice versa?* Of particular interest in this connection were the 12 items of additional stress identifiable within the high P group. Having isolated these items it was then not difficult to see how the particular traits of high P scorers mentioned in the introduction, can give rise to the type of stress most prevalent in that group.

Change in sleeping, eating, sexual difficulties, change in living conditions, trouble with in-laws, reflect the lack of harmony within the home, and interpersonal relationships. Perhaps most fundamental to a harmonious life is to feel deeply, foresee the consequence of our actions and have the ability to empathize. Business adjustment, change in financial state, change in responsibility, trouble with the boss, violation of the law, indicate the type of stress outside the home to which high P scorers are particularly vulnerable.

Mowbray *et al.* (1961) found psychiatric patients were referred not on the basis of clinical diagnosis, but because of abnormalities of conduct, social problems or inappropriate responses to medical attention. Approximately a third of the whole sample in this study came within the high P criteria which would correspond to the referral characteristics of Mowbray.

Table 3.

1. Divorce
2. Sex difficulties
3. Business adjustment
4. Change in financial state
5. Change in responsibility at work
6. Trouble with in-laws
7. Change in living conditions
8. Trouble with the boss
9. Change in sleeping habits
10. Change in eating habits
11. Christmas
12. Violation of the law.

The bodily symptom questionnaire revealed a high level of symptoms associated with autonomic arousal, in both groups. There was, however, an important difference: whereas the overall group showed a positive response to treatment in the bodily symptoms at follow-up, i.e. there were fewer symptoms than on admission, this was not so with the high P scorers, who appeared to have collected extra bodily symptoms during treatment. This suggested that high P scorers do not benefit from a treatment regime which relies heavily on group work. It is possible that the solitary non-feeling P scorer has no point of reference from which to draw, in order to benefit from the strong feelings generated within a group. He may even feel increased isolation. This raises the question 'Should these patients receive treatment alongside patients whose problems are of different complexity?' The evidence would seem to suggest, that at the very least, the treatment team should be aware of the implications. Treatment of those with high P scores might perhaps utilize a more educative approach, whereby behavioural methods are employed to enforce the notion that stress is the result of misapplied behaviour. Thus, personality and attitudes to health must be recognised as potentially important determinants of treatment outcome.

REFERENCES

EYSENCK H. J. and EYSENCK S. B. G. (1975) *Manual of the Eysenck Personality Questionnaire.* Hodder & Stoughton, London.
HOLMES T. H. and RAHE R. H. (1967) The Social Readjustment Rating Scale. *J. psychosom. Res.* **11**, 213–218.
MOWBRAY R. M., BLAIR J. and CLARK (1961) The General Practitioner's attitude to psychiatry. *Scottish med. J.* **6**, 314.
SAINSBURY P. (1960) Psychosomatic Disorders and Neurosis in out-patients attending a general hospital. *J. psychosom. Res.* **4**, 261–273.
SHAW L. and SICHEL H. (1971) *Accident Proneness.* Pergamon Press, London.

Personality and stress

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Summary—Commercial airline pilots were investigated to determine the relationship between stress, personality and life events in a normal, i.e. non-clinical population. Pilots were asked to complete the Eysenck Personality Questionnaire, the Social Adjustment Rating Scale and a specially devised Occupational Stress Inventory. The results obtained support the hypothesis that personality factors rather than the environment play a causal role in the generation of a stress reaction within individuals.

INTRODUCTION

This article is concerned with the interaction of personality and stress. Stress is viewed by society as having only a negative influence on its members, causing sometimes a stress-induced illness. Stress-induced change leading to benefit rather than illness is a long-term concept and as such is neglected. Society takes responsibility for the generation of stress, which is then imposed on the individual, and thus the implication is that stress comes from without not from within. The present author (Evans, 1981) obtained results which implied that stress is generated in part at least by the individual by virtue of his personality. This study was conducted on unselected psychiatric patients attending a psychiatric day unit; and showed how stress precipitated the illness. High scores on a life-event scale (Holmes and Rahe, 1967) correlated significantly with high scores on P (Eysenck and Eysenck, 1975). It was hypothesized that it was personality in addition to environment which played a causal role in the generation of a stress reaction within individuals. The next step taken was to examine a healthy coping cohort, and to see if the personality and stress hypothesis still held. Aviation pilots were chosen. The mean age of the 43 pilots was 42.9 ± 10.2 yr. The pilot possesses certain qualities, which probably operate to play a selective part in his choice of career. Commercial pilots are a self-selected as well as a chosen professional group, and as such one would expect them to have certain personality characteristics in common. Early aviators were associated with feats of daring and courage, and the present-day aviator still has to experience the first solo flight, accept command responsibility and the possibility of dealing with emergencies. Selye (1974) showed that physiological stress is evoked irrespective of whether the exposure is exhilarating or frightening. The stress response is non-specific, physiological stress.

METHOD

Commercial pilots have to attend a mandatory medical examination every 6 months and failure to pass the examination means withdrawal of their licence to fly. The pilot attends an aviation specialist who is responsible for testing the pilots. Commercial pilots were selected because they form a homogeneous group for sex, intelligence, maturity and responsibility and from the point of view of this study it was important that they were not a sick population. An approach was made to The Institute of Aviation Medicine who proved to be extremely helpful and introduced the author to a large private practice in London specializing in aviation medicine. The Director showed considerable interest and was willing to allow the author to question the unselected pilots attending for examination.

Medical screening is a highly emotive subject to a pilot. His livelihood depends on maintaining health to a required standard. It was therefore to be expected that high degrees of dissimulation would occur. Hence it was necessary to allay these reasonable fears by reassuring the pilots in the initial instructions as to the complete anonymity of the study.

Dissimulating conditions can be discovered by correlating the L score with the N score of the EPQ. Michaelis and Eysenck (1971), found that when conditions provided high degrees of motivation to dissimulate, N and L correlated approx. 0.4 or above. Where conditions provided low motivation to dissimulate, the correlation between N and L was low.

From what is already known the presence of a high P score might mitigate against pilot safety: some correlates of P, i.e. lack of vigilance and attention set, abnormality of perceptual judgement and impulsivity would be negatively related to the skills needed by pilots. It was therefore considered particularly interesting to see if there is an abnormal degree of P in the population to be studied. An Occupational Stress Questionnaire (OSQ) was designed especially for this study and is given in the Appendix.

Procedure

As each pilot attended for the medical examination, he was asked if he would be willing to take part in an investigation, and was given an envelope containing the following questionnaires:

1. The Eysenck Personality Questionnaire (EPQ, Eysenck and Eysenck, 1975).
2. The Social Adjustment Rating Scale (SARS; Holmes and Rahe, 1967).
3. An Occupational Stress Questionaire (OSQ; especially designed for this project).

The pilots were supplied with a stamped and addressed envelope. Included also was a letter explaining the nature of the study and requesting their help. The data was then collected and analysed to test the hypotheses.

Table 1. The mean personality scores of the pilots

	P		E		N		L	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
Pilots	2.5	2.3	7.3	6.2	7.5	5.8	7.5	5.4
EPQ norms	3.78	3.09	13.19	4.91	9.83	5.18	6.80	4.17

Table 2. Correlations between personality dimensions and occupational and life stress

	P	E	N	L
Occupational stress	NS	NS	NS	0.3*
Life stress	0.47***	0.57***	0.64***	0.66***

*Significant.
***Highly significant.

RESULTS

The first important analysis in this group of aviators was the test for dissimulation. Michaelis and Eysenck (1971) advised correlating N and L when conditions for dissimulation were considered to be high. The correlation found was −0.25. This was a reassuring finding because under conditions of little motivation to dissimulate, results on the other scales may be accepted as valid.

Table 1 compares the mean scores of pilots on the EPQ, with the male norms of the general population. As can be seen these pilots when compared with normal males, have low scores on P, E and N. In addition to measuring dissimulation the L score also measures a stable personality factor which may denote some degree of conformity.

Correlations were used to test the hypothesis that the P score would correlate with stress from life events (see Table 2). This correlation was found to be highly significant. However, unlike the findings in the preliminary study (Evans, 1981), in this presumably healthy group of aviators all personality dimensions correlated with stress engendered by life events to a highly significant degree. P, E and N did not correlate with occupational stress, but the L score did.

DISCUSSION

The hypotheses that personality correlates with stress in both a patient group and in a healthy group of aviators is upheld. The pilots had low scores in both psychoticism and stress, as compared with the patient group, and the pilots presented as an extremely stable group. It is likely that peculiarities of personality are important factors determining the choice of career as a pilot.

REFERENCES

Evans W. R. (1981) Stress and psychoticism. Person. individ. Diff. 2, 21–24.
Eysenck H. J. and Eysenck S. B. G. (1975) Manual of the Eysenck Personality Questionnaire. Hodder & Stoughton, London.
Holmes T. H. and Rahe R. H. (1967) The Social Adjustment Rating Scale. J. psychosom. Res. 11, 213–218.
Michaelis W. and Eysenck H. J. (1971) The determination of personality inventory factor patterns and intercorrelations by changes in real life motivation. J. genet. Psychol. 118, 223–234.
Selye H. (1974) Stress Without Distress. Hodder & Stoughton, London.

See Opposite for Appendix

APPENDIX

Strictly Confidential

Please place a cross on the following lines to indicate in your judgement the amount of stress reaction you experience due to or in the undermentioned situations, for example:

0%   100%
No stress at all └─────────────×──────────────────────┘ Very considerable stress
                          Take off
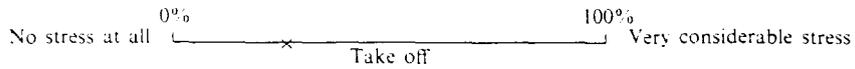
Flying:

1. Monotony of Mission
2. Limited Visibility
3. Instrument Flying
4. Seating Comfort
5. Aircraft Vibration
6. Night Flying
7. Stacking
8. Landing
9. Take-off
10. Aircraft Reliability
11. Seat-belts
12. Field of View
13. Cabin Lighting
14. Aircraft Instruments
15. Navigation Instruments
16. Avionics (Radios)
17. Interest in Mission
18. Glare
19. Adequacy of Ground Support
20. Hunger and Thirst

21. Noise
22. Temperature Regulation
23. Economic Cutbacks Which Affect Safety etc.
24. Administration; Paperwork
25. Meteorological Checks
26. Taxiing
27. Personnel Relationships
28. Social Involvement
29. Recreational Outlet
30. Morale
31. Command Relationships
32. Medical Checks
33. Type Testing
34. Sleep
35. Lag
36. Fatigue; Job Overload
37. Thwarted Ambition
38. Emergency

Please list any additional stresses:

Research report

Lifting the shroud on depression and premature mortality: A 49-year follow-up study

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A B S T R A C T

Numerous studies have shown higher rates of death from natural causes in the years immediately following an episode of clinical depression. The longer term relationship of depression to excess mortality is less clear because relatively few studies have followed the same cohort of patients for more than 10 years. The present paper reports on the findings following the same cohort of patients 49 years after discharge.
Method: Patients who were diagnosed with depression in the Chichester/Salisbury Catchment Area Study were followed for 49 years. The incidence of death from natural causes in the clinical population was compared with population rates adjusted for age and sex.
Results: The results suggest that clinical depression may have enduring effects on physical health that emerge later in life, the significance of which have so far remained undetected. Further analyses of death rates by age and sex suggest that:

  a.) Depression has a stronger impact on mortality among women.
  b.) Excess mortality starts to emerge at an earlier age among women.

Discussion: Because prior studies of depression and mortality have typically followed patients for fewer than 25 years, the long-term impact of major depression has remained undetected. The results are consistent with the view that depression has a negative impact on health that spans multiple decades.
Limitations: The association between mortality and depression could arise if prolonged treatment with antidepressant medication increases mortality. The association found between depression and mortality might also reflect differences in the quality of medical care that is provided to clinically depressed individuals. The present study does not control for factors that are confounded with depression (e.g., diet, exercise), nor does it utilize a matched control group.
Conclusion: The pathways linking depression and mortality are likely to be complex and multifactorial in nature. The major implication of the present work is to suggest that such pathways link depression with long-term, as well as short-term differences in mortality.

1. Introduction

A growing body of longitudinal research suggests that patients with a history of major depressive disorders face an increased risk for premature mortality. There are many studies, which investigate the outcome of different types of depression, (Lee and Murray 1988) who examined the same type of data. Eaton and colleagues (Kouzis et al., 1995) examined the association between depression and mortality utilizing data from five large-scale epidemiologic catchment area studies conducted in the United States. The results of this investigation suggested that depressed patients were 2.6 times more likely to die over a 1-year period than non-depressed patients. An 18-year follow-up of clinically depressed patient found mortality rates double that of the

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general population (Lee and Murray, 1988). In a 24-year follow-up study of clinically depressed patients from the Chichester/Salisbury Catchment Area studies, Thomson (1996) found significantly higher rates of premature mortality compared with death rates in the general population.

While some of the excess in premature mortality among depressed patients may be explained by suicide (Tsuang and Woolson, 1978), numerous studies find that depressed patients are more likely to develop serious physical illness and to die more frequently from natural causes. Illustratively, Thomson (1996) reports higher rates of premature mortality from natural causes, distinct from accidents and suicides. A 13-year prospective study found that patients with a history of major depression had an increased risk of suffering from myocardial infarction (Pratt et al., 1996) and stroke (Larson et al., 2001).

Many of the initial studies of excess mortality and depression have recruited their samples from clinical treatment facilities, thereby raising an important methodological issue. The use of clinic-based samples might create the appearance of an association between depression and excess mortality when one does not exist. Mortality rates in clinic-based samples may be higher than those found in the general population due to sampling bias, rather than the effects of depression, because patients referred for inpatient treatment may be in poorer health or may be exposed to more hazardous conditions in the community (Berkson, 1946). Community based studies can circumvent the potential for sampling bias in clinical samples by findings cases of depression in the community that are not receiving treatment. The results of numerous community-based studies suggest that the association between depression and mortality is not a spurious by-product of sampling bias. Higher rates of mortality in depressed individuals have been found in community-based investigations conducted in rural Canada (Murphy et al, 2008), New Haven (Bruce et al, 1994), multiple epidemiological catchment area studies in the United States (Kouzis et al, 1995; Zheng et al, 1997), and the United Kingdom (Surtees et al, 2008). The results from a large-scale longitudinal study of 60,000 residents of Norway suggests that the effects of depression on mortality may be as strong as those found for long-term cigarette smoking (Mykletun et al, 2009).

A major issue awaiting further investigation is the degree to which major depression has an enduring effect on physical health across the lifespan. Some studies suggest the opposite: that the effects of depression on mortality are relatively acute and short-term. Typically, prospective studies have found marked differences in death rates between depressed patients and the normal population in the years immediately following referral for treatment. Numerous studies have found that the effects of depression on mortality are most pronounced during the initial years of follow-up (Black et al., 1985; Black et al., 1987; Brodaty et al., 1997; Surtees and Barkley, 1994; Tsuang et al., 1980). Some investigators have suggested that it is the presence of a *recent* episode of major depression that is associated with increased risk of mortality, rather than a past history of clinical depression (Bruce et al, 1994). However, these findings should be interpreted with caution. Relatively few studies have followed patients for more than 10 years (e.g., Pratt et al, 1996; Larson et al, 2001), and only a handful of studies have followed patient for more than 20 years (e.g., Murphy et al, 2008; Thomson, 1996;

Tsuang and Woolson, 1978). It is not clear that even a 25-year follow-up period is long enough to detect longer term effects of major depression on longevity. Even in some of the longer term follow-up studies, excess mortality among depressed patients tends to occur in the years following referral, rather than ten to 25 years later (e.g., Thomson, 1996). The exception to this pattern is the Stirling County Study (Murphy et al, 2008), which employed a 40-year follow-up to examine the association between depression and mortality. In this study, connections between depression, health risk behavior, and mortality began to emerge 16 years after the baseline assessment. A statistically significant excess of mortality at the 40-year follow-up was found for depressed men, although not for depressed women. The pattern of findings reported by Murphy et al (2008) suggests that depression may have a long-term sleeper effect, as well as a short-term impact, on mortality rates. Among depressed men, excess mortality was found at the time of a 16-year follow-up, and not at the 24-year follow-up. The pattern of increased mortality among depressed men then re-emerged at the 40-year follow-up.

In order to more fully understand the linkage between major depression and premature mortality, greater attention should also be given to possible sex differences in the nature and impact of major depression. Initial investigations suggest that the effects of major depression on mortality may be greater for women, as higher mortality rates for depressed women have been reported in multi-year, follow-up studies. Illustratively, two clinic-based studies (Black et al., 1987; Brodaty et al, 1997) found higher mortality rates for women than for men. While these studies suggest that women are more vulnerable to the effects of depression on their physical health, higher mortality rates for men have sometimes been found in smaller investigations that focus on men who are middle aged or older (e.g., Brodaty et al, 1993; Evans and Whitlock, 1983), as well as in the large-scale Stirling County study (Murphy et al, 2008). These findings suggest that trends in premature mortality should be analyzed separately by sex, and that attention should be given to potential differences between men and women in the age distribution of premature deaths.

By utilizing a long follow-up period (49 years), the present work will examine the possibility that major depression has more enduring effects on patient's mortality that have yet to be identified by studies that use shorter follow-up periods. In addition, the present work will attempt to replicate and extend the work of the 40-year Stirling County follow-up study (Murphy et al, 2008) in several ways. While the Stirling County findings offer a unique long-term perspective on excess mortality, these results need to be replicated in a community context that more closely represents conditions in contemporary industrialized societies. As Murphy et al. (2008) note, Stirling County was a very remote and undeveloped rural area, particularly during the early years of the project when electricity was not universally available. In addition, the present study will examine the timing of premature deaths more closely. Murphy et al (2008) report the death rates found at 16, 24, and 40-year follow-ups. However, the assessment of deaths at the later follow-up times is cumulative: e.g., the count of deaths at the 40-year follow-up includes the deaths that have occurred at the early

follow-up periods. A more fine-grained approach to analyzing the timing of excess deaths would compile a count of observed and expected deaths that took place during specific intervals following the initial assessment of clinical severity, and conduct inferential tests to determine whether a significant increase in mortality occurs in each of those follow-up intervals. This analytic strategy has the potential to show whether mortality rates actually exhibit a sleeper effect, such that mortality rates for depressed patients initially increase, then fall to a level approximating the normal baseline death rate, before increasing markedly for depressed patients decades after the diagnosis of depression is made. Lastly, the present study will give greater attention to excluding cases in which patients have died due to suicide or accidents (which may be also include unrecognized cases of intentional self-destruction).

2. Methods

2.1. Participants

The sample for the present study is a longitudinal extension of the one that was utilized in the Thomson (1996) 24-year follow-up study of depression and premature mortality. The present work extended the timeframe for the investigation to 49 years. Permission was granted to use the data collected by Sainsbury et al. (1966) to evaluate community care in two distinct health authorities in England. Participants in this study comprise a random sample of cases that were referred to community care services by a mental health professional. The sample should be regarded as a clinic-based study, rather than as a random sample of members of the community. The present study utilizes data from subjects who have been formally diagnosed with depression by senior psychiatrists: rather than being categorized as depressed based on self-report survey screening measures, clinical severity was the basis for inclusion in the sample. The sample encompassed patients with a primary diagnosis of depression, and not patients who had depression secondary to a health problem. The total population of patients referred to community care services in the two catchment areas was 1413, of whom 685 were diagnosed as depressed. Males formed 33.3% of the total cohort, with a mean age at referral of 58.1 years. Females formed 67.7% of the total population, with a mean age at referral of 51.2 years. Because the present study utilized actuarial information about age-adjusted death rates, cases were included only if the date of birth and death could be ascertained. In addition, cases younger than 16.5 years of age at the start of the study were not included. Of the 685 cases that formed the original cohort, 566 were utilized for the present study based on the availability of birth and death dates, as well as meeting the age criterion for inclusion.

2.2. Measures and procedures

A consultant to the research unit then made an independent diagnosis of each case on a separate visit. These diagnoses have been found to have high levels of concordance between diagnosticians (Kreitman et al., 1961). Observed death rates in this cohort of patients were compared with normative data from the entire population of England and Wales that were collected and analyzed by the Office of Populations and Surveys (OPCS). These tables provided information on the national death rate per million by age and sex for each year of the study. Because the sample of depressed patients is drawn from the South of England, where mortality rates are below the national average (Shaw et al., 1998), the use of national death rates may slightly overestimate the expected death rate in the general population, making it less likely that the present study will detect excess mortality among depressed patients. However, the use of national data provides historical data on age-adjusted death rates from the start of the follow-up period (1960) to the present.

It should also be noted that the expected death rate figures are based on the general population, which includes some individuals who are suffering from clinical depression like the clinic sample in the present study: the general population should not be thought of as an entirely non-depressed sample. The inclusion of some depressed individuals in the computation of general population death rates leads to slightly more conservative test of the hypothesis that clinical depression increases the risk of premature death from natural causes.

Computation of the expected number of deaths was based upon the distribution of cases by age and sex in each year. Information regarding the date and cause of death was collected through the National Health Service Case Register (NHSCR). A protocol was submitted to the NHSCR to obtain permission to use these data in the study. Once the protocol was accepted, information from each patient was put on two cards. One card was sent to the NHSCR and the other was retained. When a death occurred a card was returned with the data and cause of death together with the International Classification of Disease code.

3. Results

Table 1 shows the number of predicted and observed deaths over the course of the 49-year follow-up, grouped in increments of 5 years. The left hand columns show the results for all deaths, while the right hand columns exclude suicides and accidents. A Poisson test (Rosner, 2005) was employed to test the null hypothesis that the observed frequency of deaths over a 5-year period was the same as a proportion of cases that would die in the general population (assuming the same sex and age distribution as was found in the sample). Considering the data for all deaths, significantly higher levels of mortality were found in the decade immediately following referral (1960–1969), as well as in follow-ups that were conducted more than 24 years after referral (i.e., 1985–1989, 1995–2004). These deaths in later decades may represent a "sleeper effect" of depression on mortality that has heretofore not been observed because almost all studies of depression and mortality employ a shorter time for follow-ups.

The right hand columns of Table 1 show the number of predicted and observed deaths excluding cases that died from suicide, accidents, and open verdicts. Predicted deaths were not adjusted to exclude these causes of death, as they are not as prevalent in the general population. The resulting comparison yields a somewhat more stringent test of the hypothesis that depressed patients have higher mortality

Table 1
Predicted and observed frequency of death.

Years	All Deaths		Excluding suicides and accidents	
	Predicted deaths	Observed deaths	Predicted deaths	Observed deaths
1960–1964	45.2	79 ***	45.2	70 ***
1965–1969	43.8	70 ***	43.8	61 **
1970–1974	45.4	37	45.4	34 *
1975–1979	51.4	40	51.4	40
1980–1984	50.5	60	50.5	57
1985–1989	45.6	71 ***	45.6	69 ***
1990–1994	46.0	56	46.0	51
1995–1999	33.5	51 ***	33.5	51 **
2000–2004	26.2	38 ***	26.2	37 *
2005–2008	18.6	24	18.6	24

  * p<.05.
 ** p<.01.
*** p<.001.

from natural causes. Exclusion of suicides and accidents did not change the results of the analyses. Deaths from natural causes happened significantly more often among depressed patients in the decade following referral as well as in follow-ups that were carried out more than 24 years after referral (i.e., 1985–1989, 1995–2004). These findings suggest that the "sleeper effect" of depression on mortality is not the product of increased risk for suicide or accidental death.

Further analyses examined the relationship between depression and premature mortality separately for men and women. Predicted and observed deaths are shown for men and women, respectively, in Table 2. Deaths from suicides and accidents are excluded. For males, the number of actual deaths significantly exceeds the number of predicted deaths for the first 5 years of the follow-up period (1960 to 1964), as well as for the decade between 1990 and 1999. For the other follow-up periods, the number of observed deaths follows the predicted number quite closely.

Females showed a statistically significant excess of mortality at numerous follow-up points. Specifically, the number of observed deaths was significantly greater than the number of predicted deaths for the first decade of the follow-up study (1960–1969), the decade between 1980 and 1989,

Table 2
Predicted and observed frequency of death excluding suicides and accidents.

Years	Males		Females	
	Predicted deaths	Observed deaths	Predicted deaths	Observed deaths
1960–1964	21.1	36 **	24.0	34 *
1965–1969	16.6	22	22.4	39 ***
1970–1974	19.8	17	25.6	17 *
1975–1979	21.3	15	30.1	25
1980–1984	22.1	19	28.4	38 *
1985–1989	17.3	20	28.2	49 ***
1990–1994	14.8	22 *	31.2	29
1995–1999	9.6	17 *	24.0	34 *
2000–2004	6.5	6	19.7	31 *
2005–2008	4.6	4	14.0	20

  * p<.05.
 ** p<.01.
*** p<.001.

and the decade between 1995 and 2004. In addition, women showed a significantly lower incidence of death for the period between 1970 and 1974. When there were significant differences between observed and predicted deaths, the discrepancy between the two was quite large. Illustratively, between 1960 and 1969, and again between 1985 and 1989, the actual death rate for female patients was nearly double the predicted rate. Between 1995 and 2004, the observed death rate was almost one and a half times the predicted rate. Cumulatively, the analyses of mortality by sex suggest that while depression is associated with increased mortality for men and women, the effects of major depression on mortality are stronger and more pervasive for women.

Additional analyses examined the issue of whether excess mortality from natural causes is more prevalent during particular age periods during the patient's lifespan. Because predicted death rates are based on population mortality for specific age ranges, it is possible to compute predicted deaths for specific age ranges across all years of the study. The question addressed in this analysis is whether the distribution of observed deaths across age ranges follows the same relative proportions as the distribution of predicted deaths. Table 3 shows the predicted and observed distribution of deaths summed across the 49-year course of the study, broken down by age grouping and sex. A Chi-squared goodness of fit test was employed to evaluate the degree to which the observed age distribution of deaths differed from the distribution that would be expected based on normal population mortality statistics. Analyses were conducted separately for males and females, as men tend to die earlier than women. The results of these analyses revealed a significant lack of fit between the observed and predicted age of death distributions for men (Chi-squared $(7) = 32.976$; $p<.001$) and for women (Chi-squared $(7) = 80.673$; $p<.001$). For men, the surplus of mortality appears to start at the age of 55, when the proportion of observed deaths in the sample is almost twice the rate that would be expected if the proportion of deaths followed the distribution of predicted deaths. For women, excess mortality starts to emerge at a younger age than for men. Specifically, among women

Table 3
Distribution of predicted and actual death by age and sex (excluding suicides and accidents).

Ages	Males		Females	
	Predicted distribution	Observed distribution	Predicted distribution	Observed distribution
20–24	0.1	0.1	0.1	0
25–34	0.3	3	0.5	4
35–44	1.0	1	2.6	13
45–54	5.2	6	9.1	17
55–64	19.3	37	26.3	34
65–74	41.6	59	70.4	68
75–84	46.1	54	117.8	123
85+	36.5	17	89.2	57
Total	178	178	316	316

Note
Chi-squared goodness of fit between age distribution of expected and observed deaths (cells for age 20–24 through 25–44 combined so that all expected cell frequencies exceed five cases).
Men: Chi-squared $(7) = 32.976$; $p<.001$.
Women: Chi-squared $(7) = 80.673$; $p<.001$.

between the age of 35 and 54, the proportion of observed deaths is three times the level that would be expected if the distribution of observed and predicted deaths were the same. This sex difference may in part reflect earlier age of referral to the treatment program for women than for men (51.2 years versus 58.1 years). Cumulatively, these findings suggest that the high levels of premature mortality found among women with a history of depression may reflect the early onset of death in early middle age.

4. Discussion

The most significant finding of the present work is a possible "sleeper effect" linking the diagnosis of depression with increased mortality decades later. Like earlier studies, the present investigation found markedly higher levels of mortality in the years immediately following treatment. Depressed cases did not show significantly higher rates of mortality from 10 to 24 years following referral. However, mortality rates were significantly higher beyond the 24-year follow-up point. It is noteworthy that the association between depression and mortality in the present study re-emerges 25 years following referral and diagnosis. Even those studies with the longest follow-up periods (e.g., Berglund and Nilsson, 1987; Brodaty et al, 1997; Rorsman et al., 1982; Thomson, 1996) generally do not collect follow-up data after 25 years. Excess mortality was found even when cases of suicide, accidents, other non-natural causes were excluded from the analysis. These findings are consistent with those of earlier work suggesting that depression is associated with higher levels of death from natural causes.

Potential explanations of the long-term association between depression and elevated mortality should consider a number of factors. While the results of the present study do not conclusively prove that depression has a negative impact on physical health, the results of the present study are at least consistent with this view. A number of mechanisms may serve as mediators between depression and long-term health outcomes. The sleeper effect may reflect the adverse effects of behavioral patterns that have a cumulative effect on physical health over time (e.g., smoking, obesity, lack of physical activity), which are often found among patients with a history of major depression (Murphy et al, 2003; Simon et al, 2006). Future research in this area should give further attention to investigating the specific disease processes that are implicated in the association between depression and mortality. Illustratively, behavioral factors such as smoking, diet, and activity level may mediate the relationship of depression to specific physical conditions such as cardiovascular disease (Larson et al., 2001) and diabetes (Meyer et al., 2004). In addition, negative affectivity may produce physiological changes that impair cardiovascular functioning and have a direct impact on risk for cardiovascular disease, even after controlling statistically for the effects of smoking, diet, and physical activity (Pratt et al, 1996; Larson et al., 2001).

While excess mortality was found for both men and women, the association between major depression and premature mortality appears to be particularly strong for women. This finding is consistent with the pattern of results reported in other large-scale prospective investigations (e.g., Black et al, 1987; Brodaty et al, 1997). The results suggest that the impact of depression on women's health is exhibited not only in the period following initial referral, but also during assessments that occur long after referral. The impact of depression on mortality is noticeable even among women who are relatively young. The greatest excess of mortality found among women in the present study affected women particularly in early middle age (aged 35 to 54 years). Further investigation should consider the ways in which depression and other risk factors interact differently in shaping the health outcomes of men and women across the life course.

The results of the present study provide a partial replication of the findings of the Stirling County 40-year follow-up study (Murphy et al, 2008). Consistent with the findings of the Stirling County study, higher mortality rates were found among depressed subjects, and evidence was found for a sleeper effect, such that additional cases of excess mortality would only be found by following a cohort for 25 years or more. By contrast, with the Stirling County study, excess mortality was found among women as well as men. Indeed, depression appears to have had a stronger effect on mortality among women in the present study. This pattern may reflect contextual differences affecting the roles of women and their health differently in remote areas of Canada versus small towns in the United Kingdom that are more fully integrated with the modern economy.

Limitations to the conclusions of the present study should be noted. The findings of the present study are open to alternative explanations. The association between mortality and depression is consistent with the hypothesis that prolonged treatment with antidepressant medication is associated with heightened mortality. However, it is worth noting that antidepressant treatment has, in some circumstances, been found to be associated with positive health outcomes (Avery and Winokur, 1976; Sauer et al, 2001). To the extent that antidepressant medications have a neutral or even a benign impact on physical health, the argument that the findings of the present study reflect an iatrogenic effect of antidepressant medications is less plausible. The association found between depression and mortality might also reflect differences in the quality of medical care that is provided to clinically depressed individuals. Illustratively, doctors who are treating depressed patients may overlook symptoms of physical illness to the extent that such symptoms are attributed instead to the patient's depressive condition (Mykletun et al, 2009). The present study also does not control for potential confounds, nor does it utilize matched control subjects. Therefore, it is possible that the increased rates of mortality in the present sample may also reflect the impact of uncontrolled factors, such as SES or initial levels of physical health prior to the onset of depression, that are associated with long-term health outcomes.

The pathways linking depression and mortality are likely to be complex and multifactorial in nature. The major implication of the present work is to suggest that such pathways link depression with long-term, as well as short-term differences in mortality. Studies that have started out with a case control design may yield interesting findings if participants can be followed for a period of time extending beyond 25 years.

References

Avery, D., Winokur, G., 1976. Mortality in depressed patients treated with electroconvulsive therapy and antidepressants. Archives of General Psychiatry 33, 1029–1037.

Berglund, M., Nilsson, K., 1987. Mortality in severe depression: a prospective study including 103 suicides. Acta Psychiatrica Scandinavica 76, 372–380.

Berkson, J., 1946. Limitations of the application of fourfold tables to hospital data. Biometrics Bulletin 2 (3), 47–53.

Black, D.W., Warrack, G., Winokur, G., 1985. The Iowa record-linkage study. III Excess mortality among patients with "functional" disorders. Archives of General Psychiatry 42 (1), 82–88.

Black, D.W., Winokur, G., Nasrallah, A., 1987. Mortality in patients with primary unipolar depression, secondary unipolar depression, and bipolar affective disorder: a comparison with general population mortality. International Journal of Psychiatry in Medicine 17, 351–360.

Brodaty, H., Harris, L., Peters, K., Wilhelm, K., Hickie, I., Boyce, P., Mitchell, P., Parker, G., Eyers, K., 1993. Prognosis of depression in the elderly. British Journal of Psychiatry 163, 589–596.

Brodaty, H., MacCuspie-Moore, C.M., Tickle, L., Luscombe, G., 1997. Depression, diagnostic sub-type, and death: a 25-year follow-up study. Journal of Affective Disorders 46, 233–242.

Bruce, M.L., Leaf, P.J., Rozal, G.P., Florio, L., Hoff, R.A., 1994. Psychiatric status and 9-year mortality data in the New Haven epidemiologic catchment area study. American Journal of Psychiatry 151, 716–721.

Evans, N.J.R., Whitlock, F.A., 1983. Mortality and late-onset affective disorder. Journal of Affective Disorders 5, 297–304.

Kouzis, A., Eaton, W.W., Leaf, P.J., 1995. Psychopathology and mortality in the general population. Social Psychiatry and Psychiatric Epidemiology 30 (4) 175-70.

Kreitman, N., Sainsbury, P., Morriset, J., Towers, J., Scrivener, J., 1961. The reliability of psychiatric assessment: an analysis. Journal of Mental Science 107, 887–908.

Larson, S.L., Owens, P.L., Ford, D., Eaton, W., 2001. Depressive disorder, dysthmia, and risk of stroke: thirteen-year follow-up from the Baltimore Epidemiologic Catchment Area Study. Stroke 32, 1979–1983.

Lee, A.S., Murray, R.M., 1988. The long-term outcome of Maudsley depressives. British Journal of Psychiatry 153, 741–751.

Meyer, C.M., Armenian, H.K., Eaton, W.W., Ford, D.E., 2004. Incident hypertension associated with depression in the Baltimore Epidemiologic Catchment area follow-up story. Journal of Affective Disorders 83, 127–133.

Murphy, J.M., Burke, J.D., Monson, R.R., Horton, N.J., Laird, N.M., Lesage, A., Sobol, A.M., Leighton, A.H., 2008. Mortality associated with depression: a forty year perspective on the Stirling County Study. Social Psychiatry and Psychiatric Epidemiology 43 (8), 594–601.

Murphy, J.M., Horton, N.J., Monson, R.R., Laird, N.M., Sobol, A.M., Leighton, A.H., 2003. Cigarette smoking in relation to depression: historical trends from the Stirling County study. American Journal of Psychiatry 160, 1663–1669.

Mykletun, A., Bjerkeset, O., Øverland, S., Prince, M., Dewey, M., Stewart, R., 2009. Levels of anxiety and depression as predictors of mortality: the HUNT study. The British Journal of Psychiatry 195, 118–125.

Pratt, L.A., Ford, D.E., Crum, R.M., Armenian, H.K., Gallo, J.J., Eaton, W.W., 1996. Depression, psychotropic medication, and risk for myocardial infarction: prospective data from the Baltimore ECA follow-up. Circulation 94, 3123–3129.

Rosner, B.R., 2005. Fundamentals of Biostatistics. Brooks Cole, New York.

Rorsman, B., Hagnell, O., Lanke, J., 1982. Mortality in the Lundby study: natural death in different forms of mental disorder in a total population investigated during a 25-year period. Neuropsychobiology 8, 188–197.

Sainsbury, P., Walk, D., Grad, J., 1966. Evaluating the Grayingwell Hospital community psychiatric service in Chichester. Millbank Memorial Fund Quarterly 44, 243–245.

Sauer, W.H., Berlin, J.A., Kimmel, S.E., 2001. Selective serotonin reuptake inhibitors and myocardial infarction. Circulation 104, 1894–1898.

Shaw, M., Dorling, D., Brimblecombe, N., 1998. Changing the map: health in Britain 1951-91. In: Bartley, M., Blane, D., Smith, G.D. (Eds.), The Sociology of Health Inequalities. Blackwell, Oxford.

Simon, G.E., Von Korff, M., Saunders, K., Miglioretti, D.L., van Belle, G., Kessler, R.C., 2006. Associations between obesity and psychiatric disorder in the US adult population. Archives of General Psychiatry 63 (7), 824–830.

Surtees, P.G., Barkley, C., 1994. Future imperfect: the long-term outcome of depression. British Journal of Psychiatry 164, 327–341.

Surtees, P.G., Wainwright, N.W., Luben, R.N., Wareham, N.J., Bingham, S.J., Khaw, K.T., 2008. Depression and ischemic heart disease mortality: evidence from the EPIC-Norfolk United Kingdom prospective cohort study. American Journal of Psychiatry 165 (9), 1204.

Thomson, W., 1996. Type of depression and results of mortality. Personality and Individual Differences 21 (4), 613–615.

Tsuang, M.T., Woolson, R.F., 1978. Excess mortality in schizophrenia and affective disorders. Do suicides and accidental deaths solely account for this excess? Archives of General Psychiatry 35, 1181–1185.

Tsuang, M.T., Woolson, R.F., Fleming, J.A., 1980. Causes of death in schizophrenia and manic-depression. British Journal of Psychiatry 136, 239–242.

Zheng, D., Macera, C.A., Croft, J.B., Giles, W.H., Davis, D., Scott, W.K., 1997. Major depression and all-cause mortality among white adults in the United States. Annals of Epidemiology 7, 213–218.

Research report

Long term follow up of suicide in a clinically depressed community sample

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ARTICLE INFO

ABSTRACT

Background: The purpose of this study was to examine how sex differences in suicide rates unfolded in a long-term follow up of patients who had been diagnosed with major depression.
Method: Patients who were diagnosed with major depression in the Chichester/Salisbury Catchment Area Study were followed for 49 years. Recorded deaths from suicide were compared with rates that were predicted from historical data on suicide mortality rates from 1960 onwards.
Findings: An overall suicide rate of 3.4% was found in the present sample. Sixteen women and three men died from suicide. Women's suicide rates were significantly higher than the level predicted based on general population trends. Men showed a barely non-significant trend in the same direction. The diagnosis of clinical depression was associated more strongly with increased risk for suicide among women compared with men. Of the female suicides, 13 had been diagnosed with endogenous depression.
Conclusions: While suicide rates are significantly higher for men in the general population, and for depressed patients of both sexes, the depression may be a particularly strong predictor of suicide risk among women.
Limitations: The dataset does not provide information about processes that mediate the relationship between depression and suicide mortality.

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1. Introduction

In most industrialized societies, men have shown a consistently higher rate of suicide mortality than women (Hawton, 2000). In the United Kingdom, suicide rates during the latter part of the 20th Century fell for women and increased for men (Kelly and Bunting, 1998). Higher suicide rates have also been found among individuals who have been diagnosed with clinically severe depression (Neeleman, 2001; Wulsin et al., 1999). What is not clear is the extent to which the diagnosis of depression may be differentially predictive of suicide in men and women. Initial studies suggest that the predictors of suicide may differ between men and women: Depressive symptoms may be

more closely associated with suicidal behaviour among women than among men (Oquendo et al., 2007). A substantially larger portion of female suicides have been diagnosed with depression compared with male suicides (Henriksson et al., 1993). On the other hand, longitudinal studies suggest that major depression increases the risk of suicide more for men than for women (Blair-West et al., 1999; Bradvik et al., 2008; Wulsin et al., 1999).

The present study also utilized an extended follow up period to assess potential long-term associations between depression and suicide that may be overlooked when shorter follow up periods are employed. With some exceptions (e.g., Bradvik et al., 2008), most studies of depression and suicide have used either retrospective psychological post-mortems, or else prospective designs using a relatively brief follow up frame (between 1 to 5 years). Prospective studies that use such a relatively brief time frame may be more sensitive to short term, acute effects of crises on depression and suicide, and may miss trends in depression that unfold more

slowly over decades. The present study tested the following hypotheses:

Hypothesis 1. Clinical depression is associated with increased suicide mortality among men and women.

Hypothesis 2. There are differences between males and females in the strength of the relationship between major depression and suicide.

Attention will also be given to the incidence of suicides in the later years of the follow-up period.

2. Methods

2.1. Participants

The sample for the present study is a longitudinal extension of the one that was utilized in the Thomson (1996) 24 year follow-up study of depression and premature mortality (Thomson, 2011). In the present work, the timeframe for the investigation was extended from twenty-four to forty-nine years. Permission was granted to use the data collected by Sainsbury et al. (1966) to evaluate community care in two distinct health authorities in England. The present study utilizes data from subjects who had been formally diagnosed with depression by psychiatrists: clinical severity, rather than being categorized as depressed based on self-report survey screening measures, thereby implying severity. The sample included patients who were hospitalized at intake as well as those who were treated through outpatient services. The present sample is a community sample in the sense that it captures all of the cases in a given catchment area from a specific time frame (1960), not in the sense that all members of the sample maintain continuous residency in non-institutional settings. The total population of patients referred to the two catchment areas was 1413, of whom 685 were diagnosed as depressed (480 were diagnosed as having endogenous depression, and 205 were diagnosed as having neurotic reactive depression). The mean age of patients with reactive depression was 44 compared with 58 years for the patients with endogenous depression. Males formed 33.3% of the total cohort, with a mean age at referral of 58.1 years. Females formed 67.7% of the total population, with a mean age at referral of 51.2 years. Because the present study utilized actuarial information about age adjusted death rates, cases were included only if the date of birth and death could be ascertained. In addition, cases younger than 16.5 years of age at the start of the study were not included. Of the 685 cases that formed the original cohort, 566 were utilized for the present study based on the availability of birth and death dates, as well as meeting the age criterion for inclusion. The ratio of males to females (1:2) was the same in the selected sample.

2.2. Measures and procedures

Patients were diagnosed as depressed following rigourous procedures and criteria. Diagnoses of depression were made by four research psychiatrists. A consultant to the research unit then made an independent diagnosis of each case on a separate visit. The resulting diagnoses have been found to be reliable and

to possess high levels of diagnostic convergence (Kreitman, Sainsbury, Morrissey, Towers and Scrivener, 1961). Further details on the assessment procedures have been published previously (Sainsbury et al., 1966). The Chichester and Salisbury samples focused on patients with a primary diagnosis of depression, not depression secondary to physical illness. The present study compared observed suicide death rates in this cohort of patients with normative data from the entire population of England and Wales. Normative data were based on figures reported by the Office for National Statistics. Historical data from this series have been compiled by McClure (2000). Expected death rates were computed separately by age grouping and gender for each year of the study. As noted by McClure (2000), overall suicide rates vary by gender and age, as do trends in suicide rates over time. Illustratively, the suicide rate for men under the age of 45 increased between 1960 and 1990, while it decreased for older men. Therefore, predicted levels of suicide must be adjusted by age and gender in different ways in the 1960s and 1990s.

Computation of the expected number of deaths was based upon the distribution of cases by age and sex in each year, using the historical incidence data provided by McClure (2000). To obtain a predicted number, the number of cases in a specific gender and age category was multiplied by the appropriate incidence rate for the year for which the predicted rate was calculated. The actual number of deaths from suicide and other causes noted below for each year, gender, and age group in the sample was compiled from official death records. Information regarding the date and cause of death was collected through the National Health Service Register (NHSCR). A protocol was submitted to the NHSCR to obtain permission to use these data in the study. Once the protocol was accepted, information from each patient was put on two cards. One card was sent to the NHSCR the other was retained. Records of each patient were returned, with the data and cause of death together with the International Classification of Disease code. Cases were coded as suicide deaths according to the ICD-8 and ICD-9 codes 430–438, or equivalent ICD-7 or ICD-10 codes. Open verdicts, undetermined deaths, poisoning, and misadventure were included in the count of suicides, but motor vehicle accidents were not.

3. Results

The main analyses of the present study examined the frequency of suicide by sex and type of depression in relation to expected levels of suicide based on general population statistics. Predicted and observed rates of death from suicide and other non-natural causes were compiled for each year of the study by sex. Because almost all of the male patients had died the fortieth year of the study, analyses were based on data from 1960 through 1999. Follow up analyses examined death rates for patients who had been diagnosed as reactive or endogenous depressives, as well as the distribution of age of death by gender among all depressed patients.

The main analyses of the present study computed predicted suicide rates for men and women in the present sample. Table 1 shows the number of predicted and observed suicides for men and women between 1960 and 1999. To determine whether the observed frequency of suicide is

Table 1
Observed and expected deaths from suicide by sex (40 year follow up).

Type of Sex	Observed	Expected	p	SMR
Male	3	0.83	.052	3.60
Female	16	1.24	.001	12.85

significantly higher than the predicted frequency, the Poisson test (Rosner, 2005) was employed. The null hypothesis stated that the observed frequency of suicide over the forty year period was the same as a proportion of cases that would die in the general population based on the sex and age distribution of the sample in each year of the study. The observed incidence of suicide for men was not significantly higher than the predicted incidence, although this figure bordered on statistical significance. For women, the observed incidence of suicide was significantly higher than the predicted incidence. The Standardized Mortality Ratio (SMR) for women was 12.85.

Suicide among women was most common during the decade following initial intake. Of the 16 suicides, 11 occurred in the first decade of the follow-up study, 2 during the second decade, and 3 more than twenty years after the start of the study. This finding suggests that longitudinal studies that use relatively short follow-up periods may slightly understate the increased long-term rate of suicide among depressed patients. The three patients who committed suicide in the later decades of the follow-up study were older at the start of the study than the eleven who committed suicide in the first decade. Specifically, when the study started, the average age of the late-suicide group was 58, while the average age of the early suicide group was 53 years old. The additional years covered by the extended longitudinal follow-up period in the present work revealed higher rates of suicide among elderly women. The average age of death in the late suicide group was 86, compared with 55 for the early suicide group.

Additional exploratory analyses examined the clinical characteristics of women who committed suicide in the present sample. Patients in this study were diagnosed as having reactive or endogenous depression. In the present sample, suicide was more prevalent among women with endogenous depression than women with reactive depression. Specifically, of the 16 women who committed suicide, 13 had been initially diagnosed as having endogenous depression, and 3 were diagnosed as having reactive depression.

4. Discussion

The long term follow up period employed in the present study may provide a more accurate estimate of the long-term risk for suicide among patients who are suffering from major depression. The overall suicide rate in the present sample of 3.4% is close to the 3.5% lifetime rate for patients with major depression estimated by Blair-West et al. (1997). The rate obtained in the present study is only slightly higher than the figure of 2.4% for affective disorders, combining inpatient and outpatient populations, offered by Bostwick and Pankratz (2000), and slightly lower than the lifetime risk of 6% estimated by Inskip et al. (1998)). This finding supports

the view that a lower figure for lifetime suicide rates (3.5%) may provide a more accurate estimate of lifetime risk for these patients that the widely circulated estimate of 15% (Guze and Robins, 1970; Miles, 1977).

The results of the present study are consistent with the view that depression is a particularly strong indicator of risk for suicide among women. The suicide rate observed was 1.6% for men and 4.2% for women. While male depressives exhibited a trend toward higher suicide rates, the SMR for women was markedly higher. The higher suicide rate for males in the general population may reflect suicides by men who have not been diagnosed with clinical depression. Compared with women, male suicides are more often diagnosed with substance abuse, conduct disorders, or other conditions.

The higher rate of suicide among depressed women may have been more evident because an extended follow up period was utilized. By following women for more than ten years, it was possible to observe differences in suicide rates that might have been overlooked in studies that utilize a shorter follow up period. However, it should be noted that other long-term longitudinal studies have found a higher rate of suicide mortality among men with major depression compared with women (e.g., Blair-West et al., 1999; Bradvik et al., 2008). Thus, the sex differences found in the present study must be interpreted with caution. The distribution of depression across decades suggests some potential heterogeneity in the mechanisms linking depression with suicide. The majority of female suicides died in the first decade after the clinical diagnosis of depression, suggesting a more acute process, while other suicides occurred more than ten years after the diagnosis, suggesting a more chronic and cumulative process.

In the present study, almost all of the women who had committed suicide (13/16) had been diagnosed with endogenous depression. While the endogenous-reactive distinction has fallen out of favour in the diagnosis of affective disorders, for the purposes of the present study the diagnosis of endogenous depression may be regarded as an indicator of severity. The findings of the present study concerning endogenous depression may be consistent with other studies (Bradvik et al., 2008) suggesting that the association between depression and suicide is particularly strong among individuals who are suffering from more severe depression.

Certain limitations in the present study must be acknowledged. The data set does not provide information on factors that might be associated with depression and suicidality, such as social and demographic variables, comorbid conditions, or risk and protective factors. The use of national statistics for the computation of expected suicide rates may result in an overestimate of predicted suicides for the locations from which this sample was drawn. The sample of depressed patients in the present study is drawn from the South of England, where psychiatric morbidity is lower than the national average (Lewis and Booth, 1992). Further, the sample was collected outside of the major urban centres of Great Britain. Though suicide rates have been increasing in rural areas, across the time period covered by the present study suicide rates have been higher in urban areas (Middleton et al., 2003). To the extent that the predicted suicide rate is over-estimated, the present study might

underestimate the strength of the relationship between suicide and clinical depression. However, the national dataset is the sole source of historical data on suicide rates adjusted for age and gender spanning multiple decades.

References

Blair-West, G.W., Mellsop, G.W., Eyeson-Annan, M.L., 1997. Down-rating lifetime suicide risk in major depression. Acta Psychiatrica Scandinavica 95, 259–263.

Blair-West, G.W., Cantor, C.H., Mellsop, G.W., Eyeson-Annan, M.L., 1999. Lifetime suicide risk and depression: sex and age determinants. Journal of Affective Disorders 55, 171–178.

Bostwick, J.M., Pankratz, V.S., 2000. Affective disorders and suicide risk: a re-examination. The American Journal of Psychiatry 157, 1925–1932.

Bradvik, L., Mattison, C., Bogren, M., Nettelbladt, P., 2008. Long-term suicide risk of depression in the Lundby cohort 1947–1997. Acta Psychiatica Scandinavica 117, 185–191.

Guze, S.B., Robins, E., 1970. Suicide and primary affective disorders. The British Journal of Psychiatry 117, 437–438.

Hawton, K., 2000. Sex and suicide: gendered differences in suicidal behavior. The British Journal of Psychiatry 177, 484–485.

Henriksson, M.M., Aro, H.M., Marttunen, M.J., Heikkeinen, M.E., Isometsa, E.T., Kuoppasalmi, K.I., Lounqvist, J.K., 1993. Mental disorders and comorbidity in suicide. The American Journal of Psychiatry 150, 935–940.

Inskip, H.M., Harris, E.C., Barraclough, B., 1998. Lifetime risk for suicide for affective disorder, alcoholism, and schizophrenia. The British Journal of Psychiatry 172, 35–37.

Lewis, G., Booth, H., 1992. Regional differences in mental health in Great Britain. Journal of Epidemiology and Community Health 46, 608–611.

McClure, G.M.G., 2000. Changes in suicide in England and Wales, 1960–1997. The British Journal of Psychiatry 176, 64–67.

Middleton, N., Gunnell, D., Frankel, S., Whitley, E., Dorling, D., 2003. Urban–rural differences in suicide trends among young adults: England and Wales, 1981–1998. Social Science & Medicine 57, 1183–1194.

Miles, C.P., 1977. Conditions predisposing to suicide: a review. The Journal of Nervous and Mental Disease 164, 231–246.

Neeleman, J., 2001. A continuum of premature death. Meta-analysis of competing mortality in the psychosocially vulnerable. International Journal of Epidemiology 30 (1), 154–162.

Oquendo, M.A., Bongiovi-Garcia, M.E., Galfaqlvy, H., Goldberg, P.H., Grunebaum, M.F., Burke, A.K., Mann, J.J., 2007. Sex differences in clinical predictors of suicidal acts after major depression: a prospective study. The American Journal of Psychiatry 164, 134–141.

Rosner, B.R., 2005. Fundamentals of biostatistics. Brooks Cole, New York.

Sainsbury, P., Walk, D., Grad, J., 1966. Evaluating the Graylingwell Hospital community psychiatric service in Chichester: suicide and community care. The Milbank Memorial Fund Quarterly 44 (1:2), 231–242.

Thomson, W., 1996. Type of depression and results of mortality. Personality and Individual Differences 21 (4), 613–615.

Thomson, W., 2011. Lifting the shroud of depression and mortality: a 49 year follow-up study. Journal of Affective Disorders 130, 60–65.

Wulsin, L.R., Vaillant, G.E., Wells, V.E., 1999. A systematic review of the mortality of depression. Psychosomatic Medicine 61 (1), 6–17.

Scientific Research

The Head Stands Accused by the Heart!

—Depression and Premature Death from Ischaemic Heart Disease

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Abstract

Background: The purpose of this study was to examine whether clinical depression was associated with higher risk of premature death from ischemic heart disease (IHD). Risk for IHD was examined separately by sex and sub-type of depression in a long-term follow-up study spanning 49 years. Method: Patients who were diagnosed with depression in the Chichester/Salisbury Catchment Area Study were followed for 49 years. Observed deaths from IHD prior to the age of 70 were compared with rates that were predicted from historical data on mortality rates from 1960 onwards. Results: Significantly higher rates of death from IHD before the age of 70 were found among males with endogenous depression. Conclusions: The results are discussed in terms of the broader literature on mortality from natural causes among patients with clinical depression. In terms of prevention, the results indicate that patients diagnosed with severe clinical depression particularly men at the very least warrant risk assessment with regard to IHD.

1. Introduction

Heart disease and depression are both very significant contributors to the global burden of mortality and morbidity. WHO currently estimates that 7.3 million deaths are due to heart disease annually and that depression affects 350 million people globally in any one year. Mounting evidence from clinic-based and community samples suggest that individuals suffering from severe depression are at increased risk for death from natural causes. Higher rates of mortality from natural causes in depressed individuals have been found in large-scale community-based investigations conducted in rural Canada (Murphy, Monson, Oliver, & Leighton, 1987), the United

States (Bruce, Leaf, Rozal Florio, & Hoff, 1994; Kouzis, Eaton, & Leaf, 1995; Zheng, Macera, Croft, Giles, Davis, & Scott, 1997), Norway (Mykletun, Bjerkeset, Øverland, Prince, Dewey, & Stewart, 2009), and the United Kingdom (Surtees, Wainwright, Luben, Wareham, Bingham, & Khaw, 2008; Thomson, 1996; Thomson. 2011). In order to more fully understand the processes linking clinical depression with mortality, investigators have examined the association of depression with specific diseases. Particular attention has been given to the relationship of depression with death from cardiovascular illness. Systematic reviews and meta-analyses of research suggest that depression substantially increases the risk of death from cardiovascular disease among individuals who initially do not show overt symptoms of cardiovascular impairment (Nicholson, Kuper, & Hemingway, 2006; Rugulies, 2002; Wulsin, Evans, Ramachandran, Murabito, Kelly-Hayes, & Benjamin, 2005; Wulsin, Valliant, & Wells, 1999), even when the effects of smoking, obesity, and other risk factors are controlled (Surtees, Wainwright, Luben, Wareham, Bingham, & Khaw, 2008; Aromaa, Raitasalo, Reunanen, Impivaara, Heiovaara, Knect, Lehtinen, Joukamaa, & Naatreka, 1994; Pratt, Ford, Crum, Armenian, Gallo, & Eaton, 1996; Wulsin, & Singal, 2003). The present study will seek to increase our understanding of the association between clinical depression and premature death from IHD by considering the degree to which the strength of this relationship may differ by sex and depressive subtype, and by utilizing a long-term follow-up period spanning 49 years.

The present study examines potential differences in the strength of the association between clinical depression and IHD death by sex. While studies of depression as a risk factor for IHD often control sex differences in depression and IHD, they rarely examine the possibility that the association between depression and IHD may be stronger for men or for women. The preponderance of available evidence suggests that the effects of depression and anxiety disorders on IHD mortality may be stronger for men than that for women (Murphy, Monson, Oliver & Leighton, 1987; Aromaa, Raitasalo, Reunanen, Impivaara, Heiovaara, Knect, Lehtinen, Joukamaa, & Naatreka, 1994; Coryell, Noyes, & Clancy, 1982; Haugland, Craig, Goodman, & Siegel, 1983; Hoyer, Mortensen, & Olesen, 2000; Lawrence, Holman, Jablensky, & Hobbs 2003; Rorsman, 2007; Week & Vaeth, 1986), although this finding has not been replicated in all studies (Angst, Stassen, Clayton, & Angst, 2002; Osby, Brandt, Correia, Ekborn, & Sparen, 2011; Tsuang, Woolson, & Fleming, 1980).

The present study will also examine the possibility that certain types of depression are more closely associated with increased risk for premature death. In the broader literature on depression and premature death, higher rates of death from natural causes have been found among patients with endogenous depression (Thomson, 1996), but not those with reactive depression. By contrast, in the literature on IHD mortality, potential differences in mortality between patients with reactive and endogenous forms of depression have not received attention. Rather, in IHD research, consideration of the heterogeneity of depression has focused on differences between unipolar and bipolar patients (Angst, Stassen, Clayton, & Angst, 2002), or differences in mortality associated with the duration or severity of depressive symptoms (Wulsin, Valliant, & Wells, 1999; Coryell, Noyes, & Clancy, 1982).

The present study will focus on premature death from IHD mortality across an extended follow-up period. In the context of trends in life expectancy and IHD mortality in the past 50 years, death from IHD before the age of 70 years merits attention. Between the 1970's and 2000, mortality rates from IHD for individuals under the age of 70 declined (Allender, Scarborough, O'Flaherty, & Capewell, 2008). To the extent that depression is associated with increased risk of premature death from IHD, we would expect to find that rates of IHD death among depressed individuals are higher than the rate predicted by declining trends. To more adequately assess the incidence of premature IHD death, and its association with depression, the present study will utilize an extended follow-up period. Characteristically, investigations of IHD mortality have followed subjects for fewer than ten years. However, initial investigations that have employed a longer follow-up period (Murphy, Monson, Oliver, & Leighton, 1987; Angst, Stassen, Clayton, & Angst, 2002) suggest that the association between clinical depression and IHD mortality may span multiple decades. The present investigation will follow IHD deaths up to 49 years after the initial diagnosis of depression. Such an extended time frame enables the investigator to detect such phenomena as premature IHD death in middle age among patients who have been diagnosed with depression in young adulthood. The focal hypotheses to be tested in the present investigation posit the following:

Hypothesis 1: Men with endogenous depression will be at higher risk of premature IHD death than men of comparable age in the general population.

Hypothesis 2: The association between depression and premature IHD death will be stronger among patients who have been diagnosed with endogenous than reactive depression.

2. Methods

2.1. Participants and Procedures

The sample for the present study is a longitudinal extension of the one that was utilized in the Thomson (Thomson, 1996) 24-year follow-up study of depression and premature mortality. In the present work, the timeframe for the investigation was extended from 24 to 49 years. Permission was granted to use the data collected by Sainsbury and colleagues (Sainsbury, Walk, & Grad, 1966) to evaluate community care in two distinct health authorities in England. The present study utilizes data from subjects who have been formally diagnosed with depression by psychiatrists: clinical severity, rather than being categorized as depressed based on self-report survey screening measures, thereby implying severity. The total population of patients referred to the two catchment areas was 1413, of whom 685 were diagnosed as depressed (480 were diagnosed as having endogenous depression, and 205 were diagnosed as having neurotic reactive depression). The mean age of patients with reactive depression was 44 compared with 58 years for the patients with endogenous depression. Males formed 33.3% of the total cohort, with a mean age at referral of 58.1 years. Females formed 67.7% of the total population, with a mean age at referral of 51.2 years. The theory that depression was either endogenous or reactive in origin was still prevalent in 1960 when the original data was collected, this theory has since lost support. It is now commonly believed that both environmental and genetic history play a part. Because the present study utilized actuarial information about age adjusted death rates, cases were included only if the date of birth and death could be ascertained. In addition, cases younger than 16.5 years of age at the start of the study were not included. Of the 685 cases that formed the original cohort, 566 were utilized for the present study based on the availability of birth and death dates, as well as meeting the age criterion for inclusion.

2.2. Measures and Procedures

Four research psychiatrists made diagnoses of reactive and endogenous depression. A consultant to the research unit then made an independent diagnosis of each case on a separate visit. The resulting diagnoses have been found to be reliable and to possess high levels of diagnostic convergence (Kreitman, Sainsbury, Morriset, Towers, & Scrivener, 1961). Further information concerning the diagnostic procedures has been reported earlier (Sainsbury, Walk, & Grad, 1966). The Chichester and Salisbury samples focused on patients with a primary diagnosis of depression, not depression secondary to physical illnesses such as IHD.

2.3. Endpoint

Information regarding the date and cause of death was collected through the National Health Service Register (NHSCR). A protocol was submitted to the NHSCR to obtain permission to use these data in the study. Once the protocol was accepted, information from each patient was put on two cards. One card was sent to the NHSCR the other was retained. Records of each patient were returned, with the data and cause of death together with the International Classification of Disease code. Cases were coded as IHD deaths using the same ICD codes as those published by Allender and colleagues (Allender, Scarborough, O'Flaherty, & Capewell, 2008). Individuals whose cause of death included IHD or IHD and another cause of death were counted as IHD Deaths.

2.4. Analysis

The present study compared observed IHD death rates in this cohort of patients with normative data from the entire population of England and Wales that was collected by the Office of Populations and Surveys (OPCS) and subsequently analyzed in a study of trends in IHD deaths in the past fifty years (Allender, Scarborough, O'Flaherty, & Capewell, 2008). These tables provided information on the national rate of IHD death by age and sex for each year of the study. Particular attention was given to death rates for individuals under the age of seventy. For each year of the study, the expected number of deaths was computed based upon the distribution of cases by age and sex.

3. Results

Analyses were conducted in two stages. Exploratory analyses examined the age of death from IHD and other natural causes among men and women with reactive and endogenous depression, as well as the relative fre-

quency of death from IHD and other natural causes among individuals under the age of seventy. These exploratory analyses suggest that premature IHD death may be more common among men with endogenous depression. The main analyses of the present study examined the frequency of premature IHD death by sex and type of depression in relation to expected levels of IHD death in the general population. Predicted and observed rates of IHD death were compiled for each year of the study by sex for patients who were initially diagnosed as having reactive or endogenous depression. Because almost all of the patients diagnosed with endogenous depression had died or reached the age of seventy by the fortieth year of the study, a relatively small sample was available between 2000 and 2009. Hence, the analyses focused on data from 1960 through 1999.

3.1. Exploratory Analyses

Exploratory analyses examined the timing of IHD death, and the relative incidence of IHD death, by sex and type of depression. **Table 1** shows the number of deaths from IHD and all other causes for men and women with reactive and endogenous depression over the course of the forty-year follow-up. While deaths from IHD occur frequently across all groups, the age of death from IHD is lower among men with endogenous depression (M = 69.5) than the age of death from other natural causes. **Table 2** shows the number of deaths before the age of 70 from IHD and all other natural causes for men and women with reactive and endogenous depressives. IHD is the leading cause of IHD death among men with endogenous depression: half of deaths arise from IHD. By contrast, IHD accounted for only 18.7% of the premature deaths among women with endogenous depression, and 19% of the premature deaths among men with reactive depression. While these findings are consistent with the view that risk for premature death is higher among men with endogenous depression, more rigorous analysis is needed to determine whether premature IHD death rates are higher in this group than in the general population. This question will be addressed in the following section.

3.2. Main Analyses: Observed and Expected IHD Mortality Rates

The main analyses of the present study computed predicted IHD death rates before the age of 70 for this sample. As described above, predicted death rates were computed based upon a published analysis of mortality data (Allender, Scarborough, O'Flaherty, & Capewell, 2008). **Table 3** shows the number of predicted and observed deaths for reactive and endogenous depressives between 1960 and 1999. To determine whether the observed frequency of deaths is significantly higher than the predicted frequency, the Poisson test (Rosner, 2005) was employed. The null hypothesis stated that the observed frequency of deaths over this forty year period was the same as a proportion of cases that would die in the general population based on the sex and age distribution of the sample in each year of the study.

Consistent with Hypothesis 1, the association between depression and premature IHD death was stronger for men than for women. The Standardized Mortality Ratio (SMR) for men was 1.97, while the SMR for women was 1.15. Hypothesis 2 was partially supported. The incidence of IHD death was significantly higher than for males with endogenous depression than it was for men of the same age. However, the incidence of IHD death was not significantly higher for women with endogenous depression, or for patients with reactive depression.

4. Discussion

The results of the present study support the hypothesis that endogenous depression is associated with higher risk for IHD among men. Men in this group have almost twice as much risk of dying from IHD before the age of seventy than their counterparts in the general population. These findings further suggest that the pathways linking depression and IHD mortality may vary depending upon sex and the type of depression: men with endogenous depression were particularly prone to premature death from IHD. The finding of elevated levels of premature IHD mortality among men with endogenous depression is consistent with earlier findings of elevated IHD mortality among men who have suffered from clinical depression (Murphy, Monson, Oliver, & Leighton, 1987; Aromaa, Raitasalo, Reunanen, Impivaara, Heiovaara, Knect, Lehtinen, Joukamaa, & Naatreka, 1994; Coryell, Noyes, & Clancy, 1982; Haugland, Craig, Goodman, & Siegel, 1983; Hoyer, Mortensen, & Olesen, 2000; Lawrence, Holman, Jablensky, & Hobbs, 2003; Rorsman, 2007; Week & Vaeth, 1986), although it also raises the question of whether failures to replicate this finding (Angst, Stassen, Clayton, & Angst, 2002; Osby, Brandt, Correia, Ekborn, & Sparen, 2011; Tsuang, Woolson, & Fleming, 1980) might be explained in part by differ-

Table 1. Lifespan by sub-type of depression, sex, and cause of death (excluding suicides).

Type of Depression	Sex	Cause of Death	Lifespan (Years)		
			M	SD	n
Reactive	Male	IHD	71.9	10.91	7
		Other	69.6	8.95	20
	Female	IHD	76.9	8.44	14
		Other	74.7	10.47	34
Endogenous	Male	IHD	69.5	10.59	34
		Other	75.1	10.12	83
	Female	IHD	77.5	9.57	35
		Other	76.8	11.10	172

Table 2. Frequency of death before age of 70 from IHD and other natural causes by sub-type of depression and sex.

Type of Depression	Sex	Cause of Death			
		IHD		Other Natural Causes	
		n	%	n	%
Reactive	Male	2	19.2%	9	81.8%
	Female	3	25.0%	9	75.0%
Endogenous	Male	18	50.0%	18	50.0%
	Female	6	18.7%	26	81.3%

Table 3. Observed and expected IHD deaths before age of 70 by type of depression and sex (40-year follow-up).

Type of Depression	Sex	Observed	Expected	p	SMR
Reactive	Male	2	2.49	0.710	0.80
	Female	3	2.00	0.323	1.50
Endogenous	Male	18	9.12	0.006	1.97
	Female	6	5.23	0.424	1.15

ences in the sub-types of depression that are included in the study. The results of the present study suggest that sex differences are not as evident among patients who would be characterized as having reactive depression. The finding of elevated mortality among men with endogenous depression is also of interest given the findings of research on death from natural causes in this cohort (Thomson, 2011). This broader study found that clinical depression was associated with higher rates of premature mortality from natural causes for men and women, but that this association was stronger for women than for men (Thomson, 2011). This pattern of findings suggests that the linkage between depression, sex, and premature mortality from natural causes may differ depending upon the specific cause of death that is under consideration. Premature death from IHD may be a greater concern for depressed men, while levels of premature death from other natural causes may be more prevalent among depressed women.

The results of the present study illustrate the value of using long-term follow-up periods to investigate the association between depression and mortality from specific causes. The potential benefit to using a thirty or forty-year follow-up period is the opportunity to record the causes of mortality for almost all of the members of a cohort, thereby gaining more data on the health consequences of depression (Murphy, Monson, Oliver, & Leighton, 1987; Angst, Stassen, Clayton, & Angst, 2002). Research conducted with briefer follow-up periods may underestimate the effects that chronic depression has on health problems that emerge decades after the initial diagnosis of depression is made.

The results of the present investigation suggest further directions for future research. A logical next step in accounting for the connection between endogenous depression and premature IHD death among men would in-

volve examining the incidence of IHD risk factors in this patient population. To the extent that men with endogenous depression are more likely to engage in unhealthy behavior (excess eating, drinking, smoking; overeating and obesity; poor compliance prescribed medications or other treatments to address cholesterol levels), the link between endogenous and depression and premature IHD death may be explained, and addressed by these behavioral factors. While increased levels of these risk factors have been found among depressed individuals (Murphy, Monson, Oliver, & Leighton, 1987; Simon, Von Korff, Saunders, Miglioretti, van Belle, & Kessler, 2006), further consideration could be given to the extent to which risk factors might be particularly high for males with endogenous depression. In addition to health risk behaviors, cognitive and affective patterns associated with depression may have an adverse effect on cardiovascular functioning (Pratt, Ford, Crum, Armenian, Gallo, & Eaton, 1996). A further potential line for investigation might consider the ways in which endogenous depression and related stressors might have a direct physiological impact on cardiovascular functioning (Selye, 1956). Such research holds the potential of directing efforts at IHD prevention to depressed patients through modifications in health-related behaviors and cognitions.

Several limitations of the present study should be noted. The associations found do not necessarily entail that endogenous depression has a causal role in the etiology of IHD. An alternate explanation of the findings is that the provision of health care for men with endogenous depression is less adequate than it is for the general population (Lawrence, Holman, Jablensky, & Hobbs, 2003). Differential levels of care could possibly arise because treatment focuses heavily on the symptoms of depression, rather than physical illnesses. In addition, among severely depressed patients, physical symptoms may also be misattributed to depression (Mykletun, Bjerkeset, Øverland, Prince, Dewey, & Stewart, 2009). A further limitation to the present study arises from regional differences in IHD death rates. Because the sample of depressed patients in the present study was drawn from the South of England, where IHD death rates are below the national average (Selye, 1956), the use of national IHD death rates may slightly overestimate the expected IHD death rate in the non-depressed population. To the extent that the predicted IHD death rate is over-estimated, the present study might slightly underestimate the association between clinical depression and IHD. National IHD death rates were nonetheless used because they provided a more comprehensive view of trends in IHD death by age and sex over the past 50 years. A third major limitation to the present study arises from the lack of information concerning participants' health status prior to death. We do not know whether depressed patients were more likely to have suffered from IHD attacks, or whether they were simply less likely to survive and recover from IHD. As noted earlier, the data do not allow us to control for the effects of other health behaviors and conditions that are often confounded with depression.

As noted the dichotomy that existed in 1960 between endogenous and reactive depression no longer has support nevertheless it is particularly interesting that these results do provide support for a dichotomy.

5. Conclusion

In summary, the results of the present study are consistent with those of other investigations that have found an increased rate of premature IHD death among depressed individuals. The present study shows that the relationship between depression and increased risk of premature IHD death holds when the assessment of depression is based on a formal and rigorous psychiatric assessment, and when IHD mortality rates are established over an extended follow-up period. The results of the present study further suggest that the associations between clinical depression and premature IHD mortality should be considered in the context of sex and type of depression. Men with endogenous depression appear to be at particularly elevated risk for premature death from IHD, and may be in particular need of clinical interventions to assess and modify other IHD risk factors.

Acknowledgements

References

Allender, S., Scarborough, P., O'Flaherty, M., & Capewell, S. (2008). Patterns of Coronary Heart Disease Mortality over the 20th Century: Possible Plateaus in the Rate of Decline. *BMC Public Health, 8,* 148-160. http://dx.doi.org/10.1186/1471-2458-8-148

Angst, F., Stassen, H. H., Clayton, P. J., & Angst, J. (2002). Mortality of Patients with Mood Disorders: Follow-Up over 34 to 38 Years. *Journal of Affective Disorders, 68,* 167-181. http://dx.doi.org/10.1016/S0165-0327(01)00377-9

Aromaa, A., Raitasalo, R., Reunanen, A, Impivaara, O., Heiovaara, M., Knect, P., Lehtinen, V., Joukamaa, M., & Naatreka, J. (1994). Depression and Cardiovascular Diseases. *Acta Psychiatrica Scandinavica, 377,* 77-82. http://dx.doi.org/10.1111/j.1600-0447.1994.tb05807.x

Bruce, M. L., Leaf, P. J., Rozal, G. P., Florio, L., & Hoff, R. A. (1994). Psychiatric Status and 9-Year Mortality Data in the New Haven Epidemiologic Catchment Area Study. *American Journal of Psychiatry, 151,* 716-721.

Coryell, W., Noyes, R., & Clancy, J. (1982). Excess Mortality in Panic Disorders. *Archives of General Psychiatry, 39,* 701-703. http://dx.doi.org/10.1001/archpsyc.1982.04290060051010

Haugland, G., Craig, T. J., Goodman, A. B., & Siegel, C. (1983). Mortality in the Era of Deinstitutionalization. *American Journal of Psychiatry, 136,* 848-852.

Hoyer, E. H., Mortensen, P. B., & Olesen, A. W. (2000). Mortality and Causes of Death in a Total National Sample of Patients with Affective Disorders Admitted for the First Time between 1973 and 1993. *British Journal of Psychiatry, 176,* 76-82. http://dx.doi.org/10.1192/bjp.176.1.76

Kouzis, A., Eaton, W. W., & Leaf, P. J. (1995). Psychopathology and Mortality in the General Population. *Social Psychiatry and Psychiatric Epidemiology, 30,* 175-70. http://dx.doi.org/10.1007/BF00790655

Kreitman, N., Sainsbury, P., Morriset, J., Towers, J., & Scrivener, J. (1961). The Reliability of Psychiatric Assessment: An Analysis. *Journal of Mental Science, 107,* 887-908.

Lawrence, D. M., Holman, C. D., Jablensky, A. V., & Hobbs, M. S. (2003). Death Rate from Ischaemic Heart Disease in Western Australian Psychiatric Patients 1980-1998. *British Journal of Psychiatry, 182,* 31-36. http://dx.doi.org/10.1192/bjp.182.1.31

Murphy, J., Monson, R. R., Oliver, D. C., & Leighton, A. H. (1987). Affective Disorders and Mortality. *Archives of General Psychiatry, 44,* 473-80. http://dx.doi.org/10.1001/archpsyc.1987.01800170095012

Mykletun, A., Bjerkeset, O., Øverland, S., Prince, M., Dewey, M., & Stewart, R. (2009). Levels of Anxiety and Depression as Predictors of Mortality: The HUNT Study. *British Journal of Psychiatry, 195,* 118-125. http://dx.doi.org/10.1192/bjp.bp.108.054866

Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an Aetiolgic and Prognostic Factor in Coronary Heart Disease: A Meta-Analysis of 6362 Events among 146538 Participants in 54 Observational Studies. *European Heart Journal, 27,* 2763-2774. http://dx.doi.org/10.1093/eurheartj/ehl338

Osby, U., Brandt, L., Correia, N., Ekborn, A., & Sparen, P. (2011). Excess Mortality in Bipolar and Unipolar Disorder in Sweden. *Archives of General Psychiatry, 58,* 844-850. http://dx.doi.org/10.1001/archpsyc.58.9.844

Pratt, L. A., Ford, D. E., Crum, R. M., Armenian, H. K., Gallo, J. J., & Eaton, W. W. (1996). Depression, Psychotropic Medication, and Risk of Myocardial Infarction. *Circulation, 94,* 3123-3129. http://dx.doi.org/10.1161/01.CIR.94.12.3123

Rorsman, B. (2007). Mortality among Psychiatric Patients. *Acta Psychiatrica Scandinavica, 50,* 354-375. http://dx.doi.org/10.1111/j.1600-0447.1974.tb08221.x

Rosner, B. R. (2005). *Fundamentals of Biostatistics*. New York: Brooks Cole.

Rugulies, R. (2002). Depression as a Predictor for Coronary Heart Disease: A Review and Meta-Analysis. *American Journal of Preventative Medicine, 23,* 51-61. http://dx.doi.org/10.1016/S0749-3797(02)00439-7

Sainsbury, P., Walk, D., & Grad, J. (1966). Evaluating the Grayingwell Hospital Community Psychiatric Service in Chichester. *Millbank Memorial Fund Quarterly, 44,* 243-245. http://dx.doi.org/10.2307/3349082

Selye, H. (1956). *The Stress of Life*. New York: McGraw-Hill.

Simon, G. E., Von Korff, M., Saunders, K., Miglioretti, D. L., van Belle, G., & Kessler, R. C. (2006). Associations between Obesity and Psychiatric Disorder in the US Adult Population. *Archives of General Psychiatry, 63,* 824-830. http://dx.doi.org/10.1001/archpsyc.63.7.824

Surtees, P. G., Wainwright, N. W. J., Luben, R. N., Wareham, N. J., Bingham, S. A., & Khaw, K. T. (2008). Depression and Ischemic Heart Disease Mortality: Evidence from the EPIC-Norfolk United Kingdom Prospective Cohort Study. *American Journal of Psychiatry, 165,* 515-523. http://dx.doi.org/10.1176/appi.ajp.2007.07061018

Thomson, W. (1996). Type of Depression and Results of Mortality. *Personality and Individual Differences, 21,* 613-615. http://dx.doi.org/10.1016/0191-8869(96)00104-3

Thomson, W. (2011). Lifting the Shroud of Depression and Mortality: A 49-Year Follow-Up Study. *Journal of Affective Disorders, 130,* 60-65. http://dx.doi.org/10.1016/j.jad.2010.09.028

Tsuang, M. T., Woolson, R. F., & Fleming, J. A. (1980). Causes of Death in Schizophrenia and Manic-Depression. *British Journal of Psychiatry, 136,* 239-242. http://dx.doi.org/10.1192/bjp.136.3.239

Week, A., & Vaeth, M. (1986). Excess Mortality of Bipolar and Unipolar Manic-Depressive Cardiovascular Death and Manic-Depressive Psychosis. *Journal of Affective Disorders, 11,* 227-234. http://dx.doi.org/10.1016/0165-0327(86)90074-1

Wulsin, L. R., & Singal, B. M. (2003). Do Depressive Symptoms Increase the Risk for the Onset of Coronary Disease? A Systematic Quantitative Review. *Psychosomatic Medicine, 65,* 201-210. http://dx.doi.org/10.1097/01.PSY.0000058371.50240.E3

Wulsin, L. R., Evans, J. C., Ramachandran, S. V., Murabito, J. M., Kelly-Hayes, M., & Benjamin, E. J. (2005). Depressive Symptoms, Coronary Heart Disease, and Overall Mortality in the Framingham Heart Study. *Psychosomatic Medicine, 67,* 697-702. http://dx.doi.org/10.1097/01.psy.0000181274.56785.28

Wulsin, L. R., Valliant, G. E., & Wells, V. E. (1999). A Systematic Review of the Mortality of Depression. *Psychosomatic Medicine, 61,* 6-17.

Zheng, D, Macera, C. A., Croft, J. B., Giles, W. H., Davis, D. & Scott, W. K. (1997). Major Depression and All-Cause Mortality among White Adults in the United States. *Annals of Epidemiology, 7,* 213-218. http://dx.doi.org/10.1016/S1047-2797(97)00014-8

Rate of Stroke Death after Depression: A 40-year Longitudinal Study Extension of Chichester/Salisbury Catchment Area Study

Wendy Thomson, PhD

Background: This study examined clinically diagnosed depression as a risk factor for incidence of death by stroke in a prospective clinically based design study. Risk for stroke was examined separately by sex in a long-term follow-up study spanning 40 years. *Methods:* Patients who were diagnosed with depression in the Chichester (population 100,000)/Salisbury (population 85,000) Catchment Area Study were followed up for 40 years. Death certificates were used to determine the cause of death in the cohort. Death rates in the general population, adjusted for age, gender, and year, were used as a control. *Results:* Clinical depression was found to be a risk factor for subsequent death from stroke in men but not in women. Death by stroke was a statistically significant cause of death in the men with diagnoses of endogenous depression but not in those men diagnosed with reactive depression. The strength of the relationship of depression with stroke increased over time. *Conclusions:* These findings suggest that the identification of depressive symptoms at younger ages may have an impact on the primary prevention of stroke in later life. The notion that depression has stronger effects over a long period is consistent with a view that severe clinical depression and physical illness occur concurrently, one exacerbating the other, and health is degraded through slow-acting, cumulative processes. Data were unavailable for the type of stroke or the health-risk behaviors (smoking, diet, and so forth) in the cohort which constituted a limitation of the study. Neither is it known what proportion of the cohort suffered a nonlethal stroke nor to what extent the treatment of clinical depression militates against suffering a lethal stroke. **Key Words:** Mortality—depression—stroke—sex differences—prevention.
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Severe Depressive Illness Preceding Death by Stroke

The World Health Organization (WHO) reports[1] that "globally; cerebrovascular disease (stroke) is the second leading cause of death. It is a disease that predominantly occurs in mid-age and older adults. WHO estimated that in 2005, stroke accounted for 5.7 million deaths world wide, equivalent to 9.9 % of all deaths. Over 85% of these deaths will have occurred in people living in low and middle income countries and one third will be in people aged less than 70 years." In response to the need for improvements in stroke data collection, prevention, and treatment, WHO has developed an international stroke surveillance system.[1]

Numerous studies suggest that individuals suffering from severe depression are at increased risk for death from natural causes.[2-9] Particular attention has been given to the relationship of clinical depression with death from stroke. Long-term follow-up studies suggest that the onset of major depression is followed by an increased probability of having a stroke[10] and death from stroke.[6,11,12] The association between depression and increased risk of death from stroke persists even when other cardiovascular risk factors and sociodemographic variables are controlled statistically.[2,13-18] The present

study will seek to increase our understanding of the association between major depression and premature death from stroke by considering the degree to which the strength of this relationship may differ by sex and by using a long-term follow-up period spanning 40 years.

In the broader literature on depression and premature mortality, attention has been given to sex differences in the strength of the relationship between major depression and premature mortality. For example, Thomson[7] found that the association between major depression and premature mortality from natural causes was statistically significant for men and women, yet was also significantly stronger for women compared with men. By contrast, relatively little attention has been given to potential sex differences in the size of the association between depression and risk for death from stroke. The results of long-term follow-up studies of depression and death from stroke have yielded inconsistent findings concerning possible sex differences. Angst et al[19] found a significant association between depression and death from stroke among males but not females. By contrast, Osby et al[20] found that depression was associated with increased risk of death from stroke for males and females. Shah et al[12] found increased risk of early stroke mortality for both sexes, although the effects of depression were stronger for women.

The present study will examine the association between depression and stroke mortality over a long follow-up time frame (40 years). With few exceptions, studies of depression and risk of death from stroke have followed patients for less than 10 years. Notable exceptions to this general trend have followed study participants for 14 to 38 years.[12-15,19,20] To the extent that depression has very long-term effects on stroke mortality that emerge over decades, such effects may be overlooked when follow-up assessments end after 10 to 15 years. The present work will follow a cohort of patients who have been diagnosed with major depression and will use a follow-up interval that is rarely found in the literature on depression and mortality.

Methods

Participants and Procedures

The sample for the present study is a longitudinal extension of the one that was used in the Thomson[7] follow-up study of depression and premature mortality. In the present work, the time frame for the investigation was extended from 24 to 40 years using procedures described in a broader study of depression and premature mortality from all natural causes.[8] Permission was granted to use the data collected by Sainsbury et al[21] to evaluate community care in 2 distinct health authorities in Southern England. The present study uses data from subjects who were formally diagnosed with depression of clinical severity in 1960 by senior psychiatrists under the strict criteria of the Medical Research Council.[22] The particular strength of this study is the diagnostic rigor used to include patients in the study cohort. Senior psychiatrists collected standardized data from all referrals from a total population of 2 census areas in the United Kingdom. The 2 areas selected were Chichester and Salisbury, clearly defined areas in the South of England with responsibility for the provision of all medical and health-related services. The services were selected for study because they had similar demographic population structures. A strength of the research design was that it was a total sample of patients diagnosed as depressed by senior psychiatrists. To this extent, the cohort was representative of the large majority of English regional senior psychiatrist-diagnosed cases of depression. A reliability study was conducted, which showed an acceptable level of concordance between the psychiatrists making the diagnosis.[23]

The total population of patients referred to the 2 catchment areas was 1413, of whom 685 were diagnosed as depressed: 480 were diagnosed as having endogenous depression, and 205 were diagnosed as having neurotic reactive depression. The mean age of patients with reactive depression was 44 compared with 58 years for the patients with endogenous depression. Males formed 33.3% of the total cohort, with a mean age at referral of 58.1 years. Females formed 67.7% of the total population, with a mean age at referral of 51.2 years. Because the present study used actuarial information about age-adjusted death rates, cases were included only if the date of birth and death could be ascertained. In addition, cases younger than 16.5 years of age at the start of the study were not included. Of the 685 cases that formed the original cohort, 566 were used for the present study based on the availability of birth and death dates, as well as meeting the age criterion for inclusion. The data obtained from the 2 catchment areas were pooled for purposes of analysis.

Measures and Procedures

Four senior psychiatrists made diagnoses of reactive and endogenous depression at the time of referral to either Chichester or Salisbury psychiatric service areas; information concerning the diagnostic procedures has been reported earlier.[21] The resulting diagnoses have been found to be reliable and to possess high levels of diagnostic convergence concerning depression.[23] Illustratively, diagnosticians exhibited 85% agreement concerning the severity of symptoms of depression. The Chichester and Salisbury samples focused on patients with a primary diagnosis of depression, not depression secondary to physical illnesses such as stroke. A strength of the research design was that it involved a total sample of patients referred to the services at that time. There is no reason to believe that the criteria for diagnosing

Table 1. *The original population begun in 1960 consisting of all referrals to 2 psychiatric services: Chichester and Salisbury*

Population	All referrals to the psychiatric services	Cohort of depressives
Endogenous depressives	480	480
Reactive depressives	205	205
Other diagnoses	728	
Total	1413	685

depression used then have changed significantly since 1960. To this extent the cohort is representative of the large majority of psychiatrist-diagnosed cases of depression.

Information regarding the date and cause of death was collected through the National Health Service Register (NHSCR).[24] A protocol was submitted to the NHSCR to obtain permission to use these data in the study. Once the protocol was accepted, information from each patient was put on 2 cards. One card was sent to the NHSCR, and the other was retained. Records of each patient were returned to the researcher with the data and cause of death together with the International Classification of Disease (ICD) code. Cases were coded as stroke deaths according to the ICD-8 and ICD-9 codes 430-438 or equivalent ICD-7 or ICD-10 codes. Individuals who died from stroke and another cause were counted as stroke deaths.

Analysis

The present study compared observed stroke death rates in this cohort of patients with normative data from the entire population of England and Wales collected by the Office of Populations and Surveys. The Office of National Statistics[25] used these data to compile information on the national rate of stroke death by age and sex for each year of the study between 1968 and 1999 for individuals 45 years of age. For the present study, expected death rates for the years 1960-1967 were calculated based on the data from 1968.

Results

The main analyses of the present study examined the frequency of stroke death by sex and type of depression in relation to expected levels of stroke death in the general

population. Predicted and observed rates of stroke death were compiled for each year of the study by sex. Tracking of patients' mortality extended for a 49-year period from 1960 to 2008, inclusive.[7] However, because almost all the male patients had died by the year 2000, analyses were based on 40 years of data from 1960 through 1999. Follow-up analyses examined death rates for patients who had been diagnosed as reactive or endogenous depressives and the distribution of age of death by gender among all depressed patients.

The main analyses of the present study computed predicted stroke death rates for men and women 45 years of age and older in the present sample. As described earlier, predicted death rates were computed based on analyses of mortality data from the Office of National Statistics.[25] Table 1 shows the number of predicted and observed deaths for men and women between 1960 and 1999. To determine whether the observed frequency of deaths is significantly higher than the predicted frequency, the Poisson test[26] was used. The null hypothesis stated that the observed frequency of deaths over the 40-year period was the same as a proportion of cases that would die in the general population based on the sex and age distribution of the sample in each year of the study. The observed incidence of death from stroke was significantly higher for men but not for women. The standardized mortality ratio (SMR) for men was 1.62, whereas the SMR for women was 1.09.

Follow-up analyses examined the death rate from stroke among patients who were initially diagnosed as having endogenous or reactive depression. Men had elevated death rates, and women did not, in both subsamples. To more fully understand when the surplus of stroke mortality among men occurred, their age distribution of death from stroke was analyzed. The observed age distribution of stroke death among men was compared with the distribution that would be predicted based on population mortality rates. The expected frequency of death was computed for specific age groupings, shown in Table 2, for each year of the study. For depressed men in this sample, 10 stroke deaths occurred between the ages of 65 and 74. By contrast, as shown in Table 3, based on general population death rates, one would expect 3.6 deaths to have occurred among men between the ages of 65 and 74 over the course of the study. A Poisson test was used to test the significance of the difference between the observed and expected death frequency. The probability of observing 10 stroke deaths when 3.6 were predicted is less than .001. The standardized mortality rate for this

Table 2. *Observed and expected stroke deaths older than the age of 45 by sex (40-year follow-up)*

Sex	Observed	Expected	*P* value	Standardized mortality ratio	95% Confidence interval
Male	20	12.31	.027	1.62	1.02-2.46
Female	32	29.35	.336	1.09	.76-1.52

Table 3. *Observed versus expected age of death for males*

Age of death for males	45-54 y	55-64 y	65-74 y	Above 75 y
Observed, n (%)	0 (0)	1 (5)	10 (50)	9 (45)
Expected, n (%)	0.2 (1.3)	1.1 (8.8)	3.6 (29.3)	7.5 (60.6)

age group was 2.78 (95% confidence interval: 1.41-4.95). This finding is consistent with the hypothesis that depressed men have an elevated risk of premature death from stroke before the age of 75.

The extension of the follow-up period to 40 years appeared to have increased its ability to detect stroke mortality. In the first 20 years of the follow-up period, 10 males died from stroke when 7.6 deaths were predicted based on age-adjusted population rates. By contrast, in the later 20 years of the follow-up period, 10 males died from stroke when 4.7 deaths would have been expected based on population rate. Applying the Poisson test, the probability of observing 10 deaths when 4.7 are expected is .014. The SMR for deaths in the later 20 years of the study is 2.13 (95% confidence interval: 1.08-3.79).

Discussion

The findings of the present sample extend the earlier findings of Thomson[7] by demonstrating excess stroke mortality after a 2 to 3 decade interval. The findings are also consistent with a growing body of evidence suggesting that major depression is a risk factor for increased stroke mortality. The results of the present study raise the question of whether the effects of depression on stroke may be clearer in long-term follow-up studies than in relatively short-term follow-up periods. In the present work, stroke death rates for males were significantly higher than the population base rate when patients were followed for more than 20 years. Although the findings of the present work on this point are merely suggestive, further analysis of this issue in data sets that have followed patients for more than 20 years[14,19,20] may shed more light on the impact of depression on stroke mortality with time. If this trend is confirmed, then studies using a shorter follow-up period might be underestimating the strength of the association between depression and stroke mortality.

The finding of elevated stroke risk for men raises an issue that might be addressed by further analysis of data that has been collected in existing long-term longitudinal data sets. Specifically, the findings of the present study raise the question of whether depression has a stronger effect on stroke mortality for men than

for women. Although long-term field studies have often controlled for the effects of sex on risk for stroke mortality, little attention has been given to the possibility that sex moderates the strength of the relationship between depression and stroke mortality. The findings of the present study, in agreement with those of Angst et al,[19] are consistent with the view that depression has a greater impact on stroke mortality among men than among women. Examination of the potential moderating effect of sex in larger, long-term, longitudinal data sets would permit firmer conclusions to be drawn.

The finding of a significant effect of major depression on stroke mortality for men should also be interpreted within the broader context of research on depression and premature mortality from natural causes. This finding is of interest given the results of research on overall death rates from natural causes in this cohort, which found that the effect of major depression on rates of premature mortality from all natural causes was stronger for women than for men.[7] By contrast, the present study found stronger effects of depression on death from stroke for men in the same cohort. This pattern of findings suggests that the linkage between depression, sex, and premature mortality may differ depending on the specific cause of death that is under consideration. Premature death from stroke maybe a greater concern for depressed men, whereas depressed women may encounter increased levels of premature death from natural causes other than from stroke.

The precise mechanisms that link depression with increased risk for stroke mortality require further attention[27,28] Carod–Artal,[28] suggests that, "Depressive symptoms maybe associated with stroke through the development of hypertension because it has been reported that depressive symptoms predict the incidence of later hypertension." This view of the mediating role of hypertension is consistent with studies reporting increased levels of hypertension among depressed community residents.[29] Other potential mediating factors include behavioral changes that increase the likelihood of hypertension and stroke, such as smoking, poor diet, and inactivity.[13-18]

Some limitations to the present study should be acknowledged. It would be particularly valuable to have information on a number of variables including prescribed treatment and medication, relapse, and so forth. However, 40 years ago the research design was focusing on then current issues within psychiatry, for example, 1 issue that was debated was whether there were 1 or 2 types of depression: reactive or endogenous depression. This explains why there was a meticulous concern with establishing and evaluating the diagnosis at the time. Nevertheless, the quality of the data analyzed provide reliability with respect to the association

between the incidence of clinical depression and death from stroke.

Because the sample of depressed patients in the present study is drawn from the South of England, where stroke death rates are below the national average,[30] the use of national stroke death rates might have slightly overestimated the expected stroke death rate in the nondepressed population. To the extent that the predicted stroke death rate was overestimated, the present study would underestimate the amount of excess mortality from stroke among depressed patients. National data was used because it offers historical age-adjusted stroke death rates spanning multiple decades. Another significant limitation of the present research is the absence of data concerning other cardiovascular risk factors and behaviors that might mediate the relationship between major depression and stroke death. A related limitation is the absence of information on patient's psychiatric status over time. Illustratively, it is not known how many of the patients might have exhibited a different pattern of symptoms later in the study, such as changing from major unipolar depression to bipolar manic depression or other conditions. It is also not clear whether any patients showed long-term remission from major depression later in life.

In summary, the results of the present study are consistent with those of other investigations that have found that individuals who suffer from major depression have an elevated risk of death from stroke later in life. The present study shows that the relationship between depression and increased risk of stroke death holds when the assessment of depression is based on a formal and rigorous psychiatric assessment and may indeed be stronger when stroke mortality rates are examined over an extended follow-up period. The results of the present study further suggest that the associations between major depression and stroke mortality should be considered in the context of sex differences. Men with endogenous depression appear to be at particularly elevated risk for premature death from stroke and may be in particular need of clinical services to reduce other risk factors for stroke. More extensive analysis of potential sex differences in existing long-term longitudinal data sets may help to provide a more adequate test of this hypothesis. The recommendations of the WHO STEPwise Stroke surveillance programme[1] make provision for further collaborative research, on which preventative stroke strategy, treatment, and rehabilitation can be planned and executed. The WHO initiative is comprehensive and forward looking and clinical depression could be considered to be included as risk factors for stroke with implications for preventative measures.

References

1. The W.H.O STEPwise approach to stroke surveillance. Noncommunicable Diseases and Mental Health, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland.
2. Bruce ML, Leaf PJ, Rozal GP, et al. Psychiatric status and 9-year mortality data in the New Haven Epidemiologic Catchment Area Study. Am J sychiatr 1994;151:716-721.
3. Kouzis A, Eaton WW, Leaf PJ. Psychopathology and mortality in the general population. Social Psychiatry and Psychiatric Epidemiology 1995;30:165-170.
4. Murphy J, Monson RR, Oliver DC, Leighton AH. Affective disorders and mortality. Archives of General Psychiatr 1987;44:473-480.
5. Mykletun A, Bjerkeset O, Øverland S, et al. Levels of anxiety and depression as predictors of mortality: the HUNT study. British J Psychiatr 2009;195:118-125.
6. Surtees PG, Wainwright NWJ, Luben RN, et al. Depression and ischemic heart disease mortality: evidence from the EPIC-Norfolk United Kingdom Prospective Cohort Study. Am J Psychiatr 2008;165:515-523.
7. Thomson W. Type of depression and results of mortality. Personality and Individual Differences 1996;21:613-615.
8. Thomson W. Lifting the shroud of depression and mortality: a 40-year follow up study. J Affective Disorders 2011;130:60-65.
9. Zheng D, Macera CA, Croft JB, et al. Major depression and all-cause mortality among white adults in the United States. Ann Epidemiology 1997;7:213-218.
10. Larson SL, Owens PL, Ford D, Eaton W. Depressive disorder, dysthmia, and risk of stroke: thirteen-year follow-up from the Baltimore Epidemiologic Catchment Area Study. Stroke 2001;32:1979-1983.
11. Ohira T, Iso H, Satoh S, et al. Prospective study of depressed symptoms and risk of stroke among Japanese. Stroke 2001;32:32903-32908.
12. Shah AJ, Veledar E, Hong Y, et al. Depression and history of attempted suicide as risk factors for heart disease mortality in young individuals. Archives of General Psychiatry 2011;68:1135-1142.
13. Carney RM, Freedland KE. Psychological distress as a risk factor for stroke-related mortality. Stroke 2002;33:5-6.
14. Everson SA, Roberts RE, Goldberg DE, Kaplan GA. Depressive symptoms and increased risk of stroke mortality over a 29-year period. Archives of Internal Medicine 1998;158:1133-1138.
15. Gump BB, Matthews KA, Eberly LE, Chang Y. Depressive symptoms and mortality in men. Stroke 2005;36:98.
16. Jonas BS, Mussolino ME. Symptoms of depression as a prospective risk factor for stroke. Psychosomatic Medicine 2000;62:463-471.
17. Salaycit KJ, Kelly-Hayes M, Beiser A, et al. Depressive symptoms and risk of stroke: the Framingham Study. Stroke 2007;38:16-21.
18. Simons LAI, McCalum J, Friendlander Y, Simons J. Risk factors for ischemic stroke: Dubbo Study of the Elderly. Stroke 1998;29:1341-1346.
19. Angst F, Stassen HH, Clayton PY, Angst J. Mortality of patients with mood disorders: follow-up over 34-48 years. J Affective Disorders 2002;68:167-181.
20. Osby U, Brandt L, Correia N, et al. Excess mortality in bipolar and unipolar disorder in Sweden. Archives of General Psychiatry 2001;58:844-850.
21. Sainsbury P, Walk D, Grad J. Evaluating the Graylingwell Hospital community psychiatric service in Chichester:

suicide and community care. Milbank Memorial Fund Quarterly 1966;44:231-242.

22. The Medical Research Centre's mission, as set out in a Royal Charter, is to encourage and support research to improve human health. Produce skilled researchers. Advance and disseminate knowledge and technology to improve the quality of life and economic competitiveness of the UK. Promote dialogue with the public about medical research.

23. Kreitman N, Sainsbury P, Morrissey J, et al. The reliability of psychiatric assessment: an analysis. British J Psychiatr 1961;107:887-908.

24. The role of The National Health Service Central Register (NHSCR) compiles and maintains for the Department of Health a computerized record of NHS patients. NHS.

25. Office for National Statistics. 20th century mortality (England and Wales 1901-2000) CD ROM. Office for National Statistics: London. Table retrieved from British Heart Foundation Statistics Website (http://www.heartstats.org/datapage.asp?id=8206) on July 4, 2010.

26. Rosner BR. Fundamentals of Biostatistics. New York: Brooks Cole 2005.

27. Pan A, Sun Q, Okerke OI, et al. Depression and risk of stroke morbidity and mortality: a meta-analysis and systemic review. J Am Med Association 2011;306:1241-1249.

28. Carod- Artal FJ. Are mood disorders a stroke risk factor? Stroke 2007;38:1.

29. Meyer CM, Armenian HK, Eaton WW, Ford DE. Incident hypertension associated with depression in the Baltimore epidemiologic catchment area follow-up. J Affective Disorders 2004;83:127-133.

30. Barker DJP, Osmond C. Death rates from stroke in England and Wales predicted from past maternal mortality. British Med J 1987;295:83-86.

Depression, Neuroticism, and the Discrepancy Between Actual and Ideal Self-Perception

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ABSTRACT

Relationships between personality dimensions, depression, and self-perception were investigated in a sample of 95 subjects. Higher levels of neuroticism were significantly related to more negative self-perceptions, more stringent self-expectations, and greater discrepancy between actual and ideal self-perception across multiple domains of behaviour and feelings. Clinically depressed subjects also had more negative perceptions of Emotional Well-Being. No significant differences were found between depressed and normal controls concerning self-evaluation standards

1. Introduction

The relationship between psychological distress and physical health has received a great deal of attention over the past decade. In particular, clinical depression has been found to be associated not only to illness, but also to premature death. Illustratively, in a 24-year longitudinal follow-up study of a cohort of clinically depressed patients and matched controls, significantly higher rates of premature death were found among depressed subjects (Thomson, 1996). While it is widely accepted that clinically depressed patients have a higher risk for death from suicide (Jamison, 1999), this study suggests that depressed patients are also at higher risk for premature death from physical illnesses, including cerebro-vascular disease and respiratory disease, as well as suicide and accidents. While this study suggests that there is a link between depression and mortality, further investigation is needed to elucidate the mediating factors that account for this relationship. One potentially important pathway through which depression may affect mortality is through its effect on self-criticism and self-derogatory ideation. Intense self-criticism and perfectionism has been identified as risk factors for suicide among clinically depressed individuals (Blatt, 1995). Excessively high self-criticism may have broader effects on health as well. Efforts to attain self-validation in the face of threats to self-worth may have deleterious effects on self-control, health maintenance habits, and physical health (Crocker & Park, 2004).

Numerous studies suggest that depression is closely linked with negative self-evaluation. Clinically depressed individuals tend to evaluate their competencies, behaviour, and self-worth more negatively than the general population (Blatt, 1995; Kovacs & Beck, 1978). Intense self-derogation in childhood and adolescence may be marker of increased

risk for depression and other disorders in adulthood (Dubois & Tevendale, 1999). Negative self-evaluations are thought to stem both from negative self-perceptions and from unrealistically high self-expectations (Kovacs & Beck, 1978). Clinically depressed individuals may have negative self-perceptions due to distortions in information processing. For example, individuals who have suffered from clinical depression are more likely to recall experiences and events that are congruent with their negative mood (Haaga, Dyck, & Ernst, 1991). Threats to self-worth may also receive excessive attention (Mathews, Ridgeway, & Williamson, 1996). In addition to such cognitive distortions, negative self-perceptions may in part accurately reflect the diminished energy, cognitive skill, and coping capacity that characterize episodes of severe depression (Dobson & Franche, 1989). In addition, according to cognitive models of depression (e.g., Kovacs & Beck, 1978), a core element of clinically severe depression may be the rigid adherence to unrealistic self-expectations. Failure to fulfill unrealistic self-expectations, in turn, exacerbates depressive symptoms. Perfectionistic thinking, in particular, is thought to reduce flexibility and resourcefulness, and increase distress, in the face of stressful life events (Hewitt & Flett, 2002). However, while the association between clinical depression and negative self-perception has been well documented, the proposed link between depression and heightened self-expectations has not received consistent support from empirical research (Coyne & Gotlib, 1983).

The linkage between depression and self-derogation may arise, in part, from heightened levels of neuroticism among many clinically depressed patients. According to Eysenck's (1967) model of personality, physiological differences in the sympathetic nervous system give rise to individual differences in emotional stability versus neuroticism. Individuals who have a more neurotic temperament are more likely to experience negative affect, including depression. Individuals who are high on neuroticism are also more likely to engage in self-derogatory

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thinking, due in part to negative self-perception, as well as maintaining stringent and unrealistic self-expectations. Neuroticism can produce negative self-perception in part due to distortions in information processing. For example, anxiety can distort attentional processes, as perceived threats receive excessive amounts of attention (Mogg, Mathews, & Eysenck, 1992). Individuals who are high on neuroticism often have more stringent and demanding self-expectations. Maintaining unrealistically high self-expectations can increase levels of anxiety, and may account for the negative effects of neuroticism on coping with stressful life events and adjustment (Enns, Cox, & Clara, 2005; Laurenti, Bruch, & Haase, 2008).

The present research will investigate the following hypotheses concerning the relationships of neuroticism and depression with self-perception and self-evaluation:

1. Higher levels of Neuroticism will be associated with more negative perceptions of behaviours and feelings.
2. Higher levels of Neuroticism will be associated with more stringent standards for evaluating behaviour and feelings.
3. Higher levels of Neuroticism will be associated with higher levels of discrepancy between subjects' self-description and their ideal standards.
4. Clinically depressed subjects will exhibit more negative perceptions of behaviours and feelings.
5. Clinically depressed subjects will have more stringent standards for evaluating behaviour and feelings.
6. Clinically depressed subjects will exhibit higher levels of discrepancy between subjects' self-description and their ideal standards.

2. Methods

2.1. Sample

The present study utilized data from 159 participants. A substantial portion of the sample received psychiatric care for depression: 34.2% of the sample received treatment for depression, while the 65.8% of the sample did not receive psychiatric treatment for depression. Females comprised 64% of the sample, while 36% were male. The median age of the study participants was 39 years old. With respect to employment status, 40.9% of the sample was employed full-time, 30.8% were employed part-time, and 1.9% were self-employed. A further 6.3% were full-time students without employment, 10.1% were unemployed, 5.0% were disabled, 2.5% were retired, and 2.5% were homemakers. Pre-existing medical conditions were present in 35.1% of the sample.

2.2. Procedures

The treated group consisted of patients referred to a psychiatrist and diagnosed as depressed in an outpatient department. The questionnaires were enclosed in a stamped addressed envelope and accompanied by an information sheet that explained the purposes of the study. Potential participants were informed that their involvement in the study was voluntary, that they could withdraw from the study at any time after they started, and that responses to the survey would be anonymous. Every patient who was referred as possibly depressed by their General Practitioner was invited to complete a questionnaire while they awaited the consultation with the psychiatrist. Control subjects were not being treated for mental illness.

2.3. Instruments

2.3.1. Eysenck personality inventory

The Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975) consists of 90 yes-no items that are designed to measure three dimensions of personality: Neuroticism, extraversion, and psychoticism. The measure also includes a Lie scale to screen out respondents who

give distorted answers to appear socially desirable. The EPQ scales have shown high levels of reliability, both in terms of internal consistency and test-retest reliability coefficients (Eysenck & Eysenck, 1975). Alpha coefficients and test-retest correlations for the EPQ scales are higher than .8 across demographic sub samples. The dimensional structure of the EPQ has proved to be robust in numerous factor-analytic studies. Illustratively, a simple structure factor rotation yields three dimensions that are comprised, respectively, by the Neuroticism, Extraversion, and Psychoticism items (Barrett & Kline, 1980). Further, these three dimensions appear to underlie the factor structure of many other widely used personality inventories (Kline & Barrett, 1983). Considerable evidence for the external validity of the EPQ dimensions has been provided by numerous studies relating differential performance on experimental tasks, as well as behavioural patterns in real-world settings, to levels of Neuroticism, Extraversion, and Psychoticism (Eysenck, 1967).

2.3.2. Self defeating quotient

The self defeating quotient (SDQ) was developed by the author for the purposes of the present investigation. Items for the SDQ were piloted with patients who were undergoing treatment for depression, and were revised in consultation with treating psychiatrists. The SDQ consists of 33 statements describing elements of the respondents' behaviour and feelings, and is administered in two parallel forms: one describing the extent to which the statement describes the actual behaviour or feelings of the respondent (the Now form), and the other indicating the ideal level of each item (the Ideal form). Illustratively, the Control item asks participants to indicate how much control they have over "things that made them feel optimistic and content." Participants responded to this item by indicating whether they had Total Control or No Control (Appendix A shows the full set of SDQ Now and Ideal items). Subjects were asked to mark their response to each item on a scale that ranged from 0 to 100. At the one extreme, the preferential state or behaviour was represented by a score of 0, while a negative response was indicated by a score of 100. The SDQ Now Total is scored by computing the average SDQ Now rating items. The SDQ ideal total score is computed as the average Ideal rating of the items. The SDQ total discrepancy score is computed by subtracting the Ideal score from the Now score. Higher discrepancy scores indicate a greater difference between ideal and perceived behaviours and feelings, and are interpreted as an indicator of increased risk for self-harm. The factor structure of the SDQ will be examined in the present investigation.

3. Results

Preliminary analyses examined the mean response of subjects to the EPQ scales and SDQ items. The main analyses of the present study then proceeded in three stages. The first stage of the analyses sought to identify the underlying dimensional structure of the SDQ through factor analysis, and to develop factorially based scales for scoring the SDQ items. The second stage of the analyses examined the relationship of specific SDQ sub-scales with differential levels of psychoticism, neuroticism, and extraversion. The final stage of analysis focused on whether clinically depressed patients and normal controls could be differentiated using the SDQ scales.

3.1. Sample descriptives

3.1.1. Eysenck personality inventory

Of the 159 subjects who participated in the present study, 125 provided complete data on the EPI. Mean scores for the sample on the EPQ scales are shown in Table 1. Compared with the EPQ norms (Eysenck & Eysenck, 1975), scores on the Neuroticism scale are notably higher, as would be expected in a sample that is comprised predominantly of individuals with clinical depression.

Table 1
Descriptive statistics for EPQ scales.

Scale	Mean	S.D.
Neuroticism	14.2	5.1
Extraversion	11.1	4.8
Psychoticism	3.2	2.3
Dissimulation	7.2	3.2

3.1.2. Self-defeating quotient

Mean scores for the SDQ items in Now and Ideal forms are shown in Table 2. With few exceptions, ratings of items on the Now form are higher than ratings on the Ideal form, indicating that, on average, subjects rate their actual behaviour and feelings as less than ideal. Two exceptions to this general trend concern the Weight and Drugs items. For the Weight item, neither the Now nor the Ideal ratings were very positive. For the Drugs items, both the Now and the Ideal ratings were extremely positive, indicating that participants did not use non-prescription drugs or value drug use very highly.

3.2. Structure of the SDQ

3.2.1. Factor analysis of the SDQ

To investigate the underlying dimensional structure of the SDQ, factor analyses were conducted using principal components analysis. The scree plot of the eigenvalues (Cattell, 1966) suggested that four factors should be retained for analysis. Both varimax and oblique rotations of the factors were conducted (Harman, 1976). The results of both rotations were consistent and interpretable. Findings from the varimax

rotation are presented below. The results of the factor analysis for the SDQ Now are shown in Table 3. Loadings below .3 have been omitted for the sake of clarity. In the analysis of the SDQ Now form, the first factor has high loadings on a broad range of issues pertaining to emotional well-being, personal habits, and community affairs. The second factor loads on items pertaining to social control. The third factor has high loadings for items related to social contexts that foster human development. The fourth factor has high loadings for items dealing with the protection of property and the environment.

3.2.2. Scoring of the SDQ scales

The results of the four-factor solution for the SDQ-Now scales were utilized to develop the scoring framework for the SDQ scales. First, items with high loadings on the first factor comprised the emotions, habits, and community scale. This incorporated the following items: Control, initiative, contentment, stress, problems, temper, jealousy, elections, neighbours, country, community, diet, weight, and debt. Results from the second factor shaped the scoring of the social control scale, which was computed as the average of items related to honesty, caring, aggression, conservation, exercise, vandalism, and destruction, while items with high loadings on the third factor comprised the Developmental Contexts scale. Specifically, this scale was computed as the average rating of items pertaining to early education, adult learning, colleagues, childhood, work, family, family time, law, and altruism. Finally, the final scale, drugs, alcohol, smoking, and frustration, contained the corresponding items which had high factor loadings on this scale, which consisted of these four constituent items. Optimism and philosophy were removed as these items did not load strongly on any of these four factors.

Table 2
Descriptive statistics for EPQ Now and ideal items.

	Now		Ideal	
SDQ item	Mean	S.D.	Mean	S.D.
Diet	38.8	22.8	15.1	17.6
Weight	38.3	19.3	45.3	17.5
Alcohol	35.3	23.7	27.7	20.1
Exercise	51.3	28.2	18.3	20.2
Drugs	4.8	11.7	6.5	16.6
Smoking	24.4	29.9	6.2	14.2
Care	34.5	21.5	10.9	13.3
Stress	60.1	26.0	19.2	25.6
Contentment	42.3	26.4	7.8	11.4
Jealousy	34.8	26.9	8.8	14.8
Temper	39.5	28.8	13.5	19.6
Debt	25.0	27.8	5.7	10.4
Control	40.1	27.4	9.1	13.3
Family	12.4	15.5	9.7	16.2
Initiative	30.8	28.6	7.6	12.6
Aggression	25.7	28.1	6.5	13.6
Family time	35.8	28.1	9.6	17.4
Neighbours	29.6	28.7	9.5	15.7
Work	33.3	24.1	7.0	12.3
Colleagues	24.9	16.4	10.1	13.9
Country	42.1	26.9	17.9	18.3
Community	56.6	27.9	21.8	24.0
Law	23.1	27.5	13.8	23.2
Conservation	26.8	25.2	12.0	20.4
Vandalism	13.4	15.9	9.5	16.8
Honesty	13.4	19.0	7.1	14.2
Elections	25.6	29.5	17.3	24.1
Altruism	26.1	21.0	13.3	16.5
Early education	36.2	26.6	13.1	18.5
Adult training	40.0	27.6	13.3	20.6
Childhood	45.6	30.2	12.7	19.7
Problems	52.9	31.7	15.1	18.0
Destruction	24.9	28.9	9.4	15.5
Frustration	50.8	29.1	15.4	23.6
Optimism	50.0	26.3	15.9	19.5
Philosophy	35.0	31.3	27.2	31.6

Table 3
SDQ factor structure (Now form).

Rotated factor matrix	Factor			
	1	2	3	4
Control	.711			
Stress	.682			
Problems	.604			
Initiative	.606	.357		
Debt	.625			
Contentment	.576			
Temper	.601	.423		
Neighbour	.484	.532		
Country	.511	.440		
Diet	.601			
Elections	.611			
Jealousy	.505		.301	
Community	.433			
Weight	.498			
Adult Learning			.793	
Family Time			.650	.347
Colleagues			.580	.346
Law		.398	.438	.332
Childhood			.594	
Family			.511	
Education			.662	
Work	.389		.476	
Drugs				.691
Altruism			.321	.329
Alcohol				.519
Smoking				.633
Honesty		.754		.397
Vandalism			.679	
Care		.588		.321
Aggression		.664		
Conservation		.534		
Exercise		.428		
Destruction		.518	.407	
Frustration	.519			.576
Optimism	.404			
Philosophy	.343			

3.2.3. SDQ Now and personality

In order to examine the association between SDQ Now dimensions and personality, correlational analyses were conducted (see Table 4). These analyses found that participants with higher psychoticism had higher average scores on all SDQ scales, while those with higher dissimulation had lower average scores on all four scales. Additionally, those with higher extraversion had significantly lower scores on the developmental contexts scale as well as higher scores on the drugs, alcohol, smoking, and frustration scale. Finally, individuals with higher neuroticism had significantly higher scores on the emotions, habits, and community scale as well as the developmental contexts and the drugs, alcohol, smoking, and frustration scale.

3.2.4. SDQ Ideal and personality

Correlational analyses were conducted to examine the association between the SDQ Ideal scales and the EPQ (see Table 5). In these analyses, individuals with higher psychoticism were found to have significantly higher scores on the drugs, alcohol, smoking, and frustration scale, while those with higher scores on dissimulation had significantly reduced scores on the developmental context as well as the drugs, alcohol, smoking, and frustration scale. Finally, individuals with higher neuroticism were found to have significantly lower scores on the first three scales.

3.2.5. Discrepancy on SDQ subscales and personality

In order to examine the relationship between Now-Ideal discrepancy scores on the SDQ and personality, correlational analyses were conducted (see Table 6). These analyses found that individuals with higher psychoticism or neuroticism had significantly higher discrepancies on all four scales. Additionally, individuals with greater extraversion had significantly lower discrepancies on the developmental contexts scale, and significantly higher discrepancies on the drugs, alcohol, smoking, and frustration scale. Finally, those with greater dissimulation had significantly reduced scores on all scales with the exception of developmental contexts.

3.3. SDQ scores and psychiatric status

Having found significant relationships between Neuroticism and the SDQ dimensions, the next stage of the analyses examined the question of how well the SDQ differentiated clinically depressed and normal control subjects.

3.3.1. SDQ Now scores for depressed and control subjects

Scores on the SDQ-Now for clinically depressed and control subjects are shown in Table 7. T-tests were conducted to test the statistical significance of differences between groups. Clinically depressed subjects had significantly higher mean scores on the emotions, habits, and communications scale of the SDQ. This mean difference had an effect size of $d = .3949$, indicating a small to moderate effect size.

3.3.2. SDQ ideal scores for depressed and controls subjects

SDQ-Ideal scores for clinically depressed and control subjects are shown in Table 8. No significant differences were found.

3.3.3. SDQ discrepancy scores for depressed and control subjects

SDQ Discrepancy scores for clinically depressed and control subjects are shown in Table 9. No significant differences were found in this set of analyses.

4. Discussion

The present work was undertaken to test four focal hypotheses concerning relationships between neuroticism, depression, and discrepancies between perceived and ideal self-descriptions. The first hypothesis proposed that higher levels of Neuroticism would be associated with more negative appraisals of behaviours and feelings. This hypothesis was strongly supported by the results of the present work. Subjects with higher levels of Neuroticism tend to have significantly more negative appraisals of their emotional well-being, community affairs, personal habits, and developmental contexts. The cumulative pattern of these findings suggests that neuroticism is associated with a pervasive negative view of multiple life domains. The second hypothesis proposed that higher levels of neuroticism would be associated with more stringent standards for evaluating one's own behaviour and feelings. This hypothesis received support to the extent that higher levels of Neuroticism were associated with more stringent and demanding ideals with regard to emotional well-being, community affairs, personal habits, developmental contexts, and social control.

The third hypotheses proposed that higher levels of Neuroticism would be associated with higher levels of discrepancy between subjects' self-description and their ideal standards. This hypothesis also received support from the results of the present study. Subjects with higher Neuroticism scores had significantly higher discrepancy scores for emotional well-being, community affairs, personal habits, developmental

Table 5
Correlation of SDQ Ideal and EPQ Scales (n = 125).

	EPQ scale			
SDQ ideal scale	Psychoticism	Extraversion	Neuroticism	Dissimulation
Emotions, habits, commun.	.055	.083	−.333***	−.078
Social control	.077	.101	−.422***	−.066
Developmental contexts	.155	−.130	−.192*	−.318***
Drugs, alc., smoking, frus.	.340***	−.036	−.061	−.258**

* p < .05.
** p < .01.
*** p < .001.

Table 4
Correlation of SDQ Now and EPQ Scales (n = 125).

	EPQ scale			
SDQ Now scale	Psychoticism	Extraversion	Neuroticism	Dissimulation
Emotions, habits, commun.	.445***	−.085	.515***	−.295**
Social control	.252**	.026	−.020	−.320***
Developmental contexts	.399***	−.340***	.241**	−.244**
Drugs, alc., smoking, frus.	.448***	.202*	.257**	−.458***

* p < .05.
** p < .01.
*** p < .001.

Table 6
Correlation of SDQ discrepancy and EPQ scales (n = 125).

	EPQ scale			
SDQ now-ideal	Psychoticism	Extraversion	Neuroticism	Dissimulation
Emotions, habits, commun.	.355***	−.111	.608***	−.231***, ***
Social control	.208*	−.050	.324***	−.280**
Developmental contexts	.287**	−.262**	.430***	.048
Drugs, alc., smoking, frus.	.198*	.217*	.284**	−.261**

* p < .05.
** p < .01.
*** p < .001.

Table 7
SDQ Now Scores Among Clinically Depressed and Control Subjects (n = 157).

| SDQ Now scale | Depressed | Control | |
	Mean	Mean	t-value
Emotions, habits, commun.	43.7	37.7	2.101* (142)
Social control	24.5	27.5	1.170***** (143)
Developmental contexts	31.3	29.9	−.501 (135)***
Drugs, alc., smoking, frus.	29.6	28.5	−.289 (122)

* p < .05.
** p < .01.
*** p < .001.

Table 9
SDQ discrepancy scores among clinically depressed and control subjects.

| SDQ Now-ideal | Depressed | Control | |
	Mean	Mean	t-Value
Emotions, habits, commun.	26.9*	23.2	1.084 (136)
Social control	14.5**	17.4	−1.149 (140)
Developmental contexts	17.6***	16.0	.589 (130)
Drugs, alc., smoking, frus.	13.2	14.5	−.339 (121)

* p < .05.
** p < .01.
*** p < .001.

contexts, and social control Higher levels of Neuroticism were related to higher discrepancy scores in part because this personality dimension was associated with more negative self-perceptions, as noted above, across multiple SDQ dimensions. However, a further contributing factor was the association of Neuroticism with more stringent and demanding ideals in multiple life domains. Consistent with the first, second, and third hypothesis, the Neuroticism scale was the most important predictor of the SDQ scales and discrepancies between perceived and ideal levels of the SDQ dimensions.

The dimension of Extraversion was also associated with the SDQ Now and discrepancy scores. Extraverts tend to possess more benign views of developmental contexts that they occupied in the past and the present, both in terms of how they perceive these contexts and in terms of how these perceptions compare with their ideals. Extraverts also reported a more positive view of community affairs. These findings may reflect the degree to which extraverts experience higher levels of social integration across school, work, and family contexts. On the other hand, extraverts also had a more negative view of their personal habits and substance use. Among extraverts, ratings of substance use were higher overall and were more discrepant with the subjects' stated ideals. Issues with personal habits and substance use may reflect the levels of impulsivity and stimulus-seeking that are often associated with the personality trait of Extraversion (Eysenck, 1967).

While the primary hypotheses of the present study focused on neuroticism and depression, a number of serendipitous finding emerged with regard to Psychoticism. Subjects with higher Psychoticism scores had higher discrepancy scores across multiple scales. Subjects with higher Psychoticism scores also reported greater substance use and more lenient attitudes to substance use. These findings suggest that the SDQ dimensions may have implications for broader areas of mental health in addition to neuroticism and depression.

Subjects who dissimulate, or give socially desirable answers, also tend to portray themselves in a more positive light on all of the SDQ Now dimensions, and profess to hold more stringent standards concerning substance use on the SDQ Ideal form. This pattern of findings is consistent with the view of dissimulation as a trait that leads subjects to value a positive social presentation, regardless of the specific content area that is being measured.

Turning to the findings for clinical depression, the fourth hypothesis stated that clinically depressed subjects would exhibit more negative appraisals of behaviours and feelings. This hypothesis received partial support from our findings. Clinically depressed subjects provided significantly more negative evaluations of their emotional well-being.

Table 8
SDQ Ideal Scores Among Clinically Depressed and Control Subjects.

| SDQ Ideal Scale | Depressed | Control | |
	Mean	Mean	t-Value
Emotions, habits, commun.	15.7	14.9	−.485 (140)
Social control	11.2	10.1	−.575 (144)
Developmental contexts	13.8	14.1	.126 (136)
Drugs, alc., smoking, frus.	16.5	14.0	−.896 (121)

However, ratings of other SDQ Now dimensions were not significantly different between depressed and control subjects. The fifth hypothesis proposed that clinically depressed subjects would have more stringent standards for self-evaluation. This hypothesis was not supported by the findings of the present study. Indeed, depressed subjects had more lenient standards concerning community affairs. The findings of the present study are more consistent with the view that higher standards do not characterize the majority of patients with clinical depression (Coyne & Gotlib, 1983). The sixth hypothesis stated that clinically depressed subjects would exhibit higher levels of discrepancy between their self-description and their ideal standards. This hypothesis was not supported. The cumulative pattern of these findings is consistent with the view that clinically depressed patients are prone to self-derogatory thinking, and that self-derogatory thought patterns arise primarily from negative self-perception, rather than from having different ideals or performance standards.

Overall, the findings of the present study are consistent with the view that neuroticism has a pervasive relationship with self-derogatory and self-critical thinking, resulting from negative self-perceptions as well as higher evaluation standards. Neuroticism appears to play a unique and powerful role in sustaining these thought patterns, distinct from other dimensions of personality. Clinical depression is also associated with marked discrepancies between perceived and ideal levels of emotional well-being, primarily due to negative self-perceptions, rather than evaluation standards. The combination of heightened neuroticism with clinical depression may merit particular attention in further investigation of the relationship between psychopathology and self-derogatory thinking. Further investigation of the relationship between neuroticism, depression, and self-perception should adopt a multidimensional perspective on the assessment of self-perception as well as the discrepancy between real and ideal self-concept. The results of the factor analysis suggest that self-concept is a multidimensional concept. Further, the relationship of neuroticism and depression to self-perception differs according to which dimension of self-concept is under consideration. Both neuroticism and depression are related to negative perceptions of emotional well-being. However, neuroticism has a stronger relationship with self-perception across a wider set of domains that are not associated as strongly with clinical depression.

While Neuroticism was found to have a pervasive pattern of relationships with SDQ dimensions, Extraversion was also found to be related to multiple dimensions of self-perception and evaluation. Further investigation should consider the extent to which combinations of Neuroticism and Extraversion have differential effects, as suggested by Eysenck (1967).

The results of the present study should be interpreted with caution. The design of the investigation was cross-sectional, and so does not provide us with a clear picture of the temporal precedence, much less the causal structure, of neuroticism, depression, and self-destructive ideation. Further, the sample for the present study was drawn from a single clinic, and so the generalizability of these findings to a wider and more diverse population is not known. Nonetheless, the cumulative pattern of our findings provides some initial evidence for a link between Neuroticism and Depression and self-destructive ideation.

Appendix A. Sample SDQ Now items

Item	Positive pole	Negative pole
How do you feel about yourself and your family? (family)	Very good	Very bad
How much control do you feel you have over things that make you feel optimistic and contented? (control)	Total control	No control
How often do you feel really angry? (angry)	Very seldom	Very often
How do you feel when you are around people at work? (work)	Very comfortable	Very uncomfortable
To what extent do you think that you create your own stress? (stress)	Not at all	I create all my own stress

References

Barrett, P. T., & Kline, P. (1980). Personality factors in the Eysenck personality questionnaire. *Personality and Individual Differences, 1,* 317–333.

Blatt, S. J. (1995). The destructiveness of perfectionism: implications for the treatment of depression. *American Psychologist, 50,* 1003–1020.

Cattell, R. B. (1966). The Screen test for the number of factors. *Multivariate Behavioral Research, 1,* 245–276.

Coyne, J. C., & Gotlib, I. H. (1983). The role of cognition in depression: A critical appraisal. *Psychological Bulletin, 94,* 472–505.

Crocker, J., & Park, L. E. (2004). The costly pursuit of self-esteem. *Psychological Bulletin, 130,* 392–414.

Dobson, K., & Franche, R. -L. (1989). A conceptual and empirical review of the depressive realism hypothesis. *Canadian Journal of Behavioural Science, 21,* 419–433.

DuBois, D. L., & Tevendale, H. D. (1999). Self-esteem in childhood and adolescence: Vaccine or epiphenomenon. *Applied and Preventative Psychology, 8,* 103–117.

Enns, M. W., Cox, B. J., & Clara, I. P. (2005). Perfectionism and neuroticism: A longitudinal study of specific vulnerability and diathesis-stress models. *Cognitive Therapy and Research, 29,* 463–478.

Eysenck, H. J. (1967). *The biological basis of personality.* Springfield, IL: Thomas.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck personality questionnaire.* London: Hodder and Stoughton.

Haaga, D. A., Dyck, M. J., & Ernst, D. (1991). Empirical status of a cognitive theory of depression. *Psychological Bulletin, 110,* 15–236.

Harman, H. (1976). *Modern factor analysis.* Chicago: University of Chicago Press.

Hewitt, G. L., & Flett, P. L. (Eds.). (2002). *Perfectionism: Theory, Research, and Treatment.* Washington, D.C.: American Psychological Association.

Jamison, K. R. (1999). *Night Falls Fast: Understanding Suicide.* New York: Alfred A. Knopf.

Kline, P., & Barrett, P. T. (1983). The factors in personality questionnaires. *Advances in Behaviour Research and Therapy, 5,* 141–202.

Kovacs, M., & Beck, T. (1978). Maladaptive cognitive structures in depression. *American Journal of Psychiatry, 135,* 525–533.

Laurenti, H. J., Bruch, M. A., & Haase, R. F. (2008). Social anxiety and socially prescribed perfectionism: unique and interactive relationships with maladaptive appraisal of interpersonal situations. *Personality and Individual Differences, 45,* 55–61.

Mathews, A., Ridgeway, V., & Williamson, D. A. (1996). Evidence for attention to threatening stimuli in depression. *Behaviour Research and Therapy, 34,* 695–705.

Mogg, K., Mathews, A., & Eysenck, M. (1992). Attentional bias to threat in clinical anxiety states. *Cognition and Emotion, 6,* 149–159.

Thomson, W. (1996). Type of depression and results of mortality. *Personality and Individual Differences, 21*(4), 613–615.

Appendices

Appendix 1 comments from Dr. Peter Sainsbury.

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 "The Relationship of Bodily Symptoms to Psychological Adjustment"

I thought this was a very nicely written and thought out dissertation for a degree. As it stands it is less satisfactory for publication as a paper because (1) It falls halfway between an essay and a bit of original psychosomatic research ; (2) there are defects in the design, e.g. to test her hypotheses Mrs. Evans might have done better to select a consecutive series of G.P.s' patients who scored high on the symptom inventory, and then randomly allocated cases to psychiatric management and to conventional G.P. supervision; (3) the number of cases is small and it was not clear where the "multivariate analysis" came in. So some reference to statistical method is required in the Method Section. (4) I suppose since Holmes and Rahe Life-event scale is used, the Review Section should also refer to the literature on life-events and bodily symptoms, e.g. Holmes and Wolf.

In any case it is a very nice and interesting paper. Why not try the Journal of Psychosomatic Research ?

Appendix 2

What is complexity?

Complexity theory maintains that all complex systems from the micro to the macro, from molecules to nation states, and the balance of nature, lies a set of rules that when identified will yield unification of science. The search for those rules is the concern of those who believe in this emerging science.

Complex Adaptive Systems are a model for thinking about the world around us not a model for predicting what will happen. I have found that in nearly all situations I can view what is happening in Complex Adaptive Systems terms and that this opens up a variety of new options which give more choice and more freedom. The research presented here has been

influenced by complexity theory and the gestalt concept. Viewing man as a complex adaptive system with disorder and order (homeostasis) and the interplay between the two.

3   Wendy Thomson (1996) Types of depression and results of mortality Pers. & Indiv. Diff. V0l. 21 No. 4, pp. 613-615, 1996

Type of depression and results of mortality

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Summary—The object of this study was to discover if a mortality study could distinguish between reactive/neurotic and endogenous depression; a reoccurring concern. Research has so far been inconclusive and psychiatrists still use the two diagnoses; reactive and endogenous depression. A method was designed using a cohort selected 24 years previously and that followed the strict criteria of a Medical Research Unit in the U.K. Each patient was given the diagnosis of either reactive or neurotic at that time. The cohorts were followed prospectively for 24 years. The results were computed to test the statistical significance of premature mortality for both diagnosis and to compare the results with a control group. The results showed endogenous depression was associated with premature mortality, from both natural and unnatural death, particulary in the years immediately after discharge. The results give some support to the distinction between endogenous and reactive depression, and demonstrate that a diagnosis of endogenous depression is related to a higher risk of premature mortality in some patients. Copyright © 1996 Elsevier Science Ltd.

INTRODUCTION

Lewis (1934), Paykel, Prusoff and Klerman (1971), Hirschfield (1980) and Copeland (1984) all concluded that there is no separate and distinct clinical picture associated with reactive and endogenous depression respectively. They also maintain that neurosis and psychosis should be viewed as a continuum. Lader (1981) suggested severity of depression is perhaps a more important distinction. Studies conducted by Eysenck (1956) support a depression dichotomy, as does Eysenck (1970). In psychiatry there is a lack of physiological tests to help substantiate a diagnosis. In spite of doubts, psychiatrists continue to use the endogenous/reactive dichotomy extensively. A study by Kerr, Schapira and Roth (1969) suggested the possibility that mortality data might throw light on the problem.

METHOD

1. To select a cohort of patients referred to a psychiatrist and given a diagnosis of reactive or endogenous depression, to collect relevant data.
2. To follow the patients for 24 years and to calculate the number dead, and the age, sex, diagnosis and causes of death.
3. To compare the number dead and the data with a suitable control population.
4. To calculate the statistical significance of the results obtained.

Permission was granted to use the data collected by Sainsbury and Grad (1996) to evaluate community care in two distinct health authorities in England. Standardized data were collected by Sainsbury and Grad on all referrals made to the psychiatric service, using an interview schedule.

A reliability study was conducted to evaluate the study by Kreitman, Sainsbury, Morrissey, Towers and Scrivener (1961). The diagnoses were made by four research psychiatrists; the research unit's consultants then independently diagnosed the cases on a second visit. A strength of the research design was that it involved a total sample of patients referred to the service at that time. There is no reason to believe that the criteria for diagnosing depression used then have changed significantly since 1960. To this extent the cohort is representative of the large majority of psychiatrist-diagnosed depression.

Selection of a control group

Normative data from the entire population of England and Wales were collected and analysed by the Office of Populations and Surveys (OPCS). These tables were used to calculate:

• The national death rate per million by age and sex.
• The national death rate for each year of the study.
• The national death rate per million by age, sex, and principal cause of deaths, all remaining deaths were calculated as 'other deaths'.

The National Health Service Register (NHSCR) collated the actual death data from the entire population. A protocol had to be submitted to the NHSCR to obtain permission to use this information in the study. Once the protocol was accepted information from each patient was put on two cards, one card was sent to the NHSCR the other was retained. Records of each patient were returned, with the data and cause of death together with the International Classification of Disease code.

Appendix 4

World Health Organization Information sheet

Premature death among people with severe mental disorders1 Key facts

- People with severe mental disorders on average tend to die earlier than the general population. This is referred to as premature mortality. There is a 10-25 year life expectancy reduction in patients with severe mental disorders.

- The vast majority of these deaths are due to chronic physical medical conditions such as cardiovascular, respiratory and infectious diseases, diabetes and hypertension. Suicide is another important cause of death.

- Mortality rates among people with schizophrenia is 2 to 2.5 times higher than the general population.

- People with bipolar mood disorders have high mortality rates ranging from 35% higher to twice as high as the general population.

- There is a 1.8 times higher risk of dying associated with depression. People with severe mental illness do not receive the same quality of physical health care as the general population.

- The majority of deaths of patients with severe mental illness that are due to physical medical conditions are preventable with more attentive checks for physical illness, side effects of medicines and suicidal tendencies-.

- Interventions exist to promote the mental and physical health of individuals with severe mental disorders. There is a need for increasing access to quality care for patients with severe mental disorders, and to improve the diagnosis and treatment of coexisting physical conditions. The integration of mental and physical health care could facilitate this.

Overview

The lifespan of people with severe mental disorders is shorter compared to the general population1.

Cardiovascular disease, which includes coronary heart disease, atherosclerosis, hypertension and stroke, is one of the leading causes of death among people with severe mental disorders. People with severe mental disorders also have higher than expected rates of Type II diabetes, respiratory diseases, and infections such as HIV, hepatitis and tuberculosis.

1 The severe mental disorders to which this information sheet refers are psychosis, bipolar mood disorder and moderate-severe depression.

The medical conditions experienced by this group are associated with preventable risk factors, such as smoking, physical inactivity, obesity, and side effects of psychiatric medication.

People with severe mental disorders are also more likely to receive lower quality health and social care than the general population. One of the central issues around healthcare access for people with a severe mental disorder is the stigma and discrimination associated with mental illness. Strategies to improve health and life expectancy must focus not only on modifying individual risk factors but also on improving access to quality health care and eliminating the stigma associated with severe mental disorders.

Factors contributing to premature death

Chronic physical conditions

People with severe mental disorders have a higher prevalence of many chronic diseases and are at a higher risk for premature death associated with these diseases than the general population. The excess mortality among this group largely related to cardiovascular, respiratory and metabolic diseases. Metabolic disease is a collective term referring to diabetes, hypertension and weight gain.

The prevalence of diabetes in people with schizophrenia is 2-3 times higher than the general population. This is in part due to lifestyle and health risk factors, but it is also partly due to unmonitored antipsychotic treatment, which can lead to weight gain. Significant weight gain is one of the main reasons patients do not want to take prescription medication. Weight gain in this population also poses a significant risk of lipid abnormalities and cardiovascular complications.

Infectious diseases

People with severe mental disorders also have higher rates of infectious diseases such as HIV and hepatitis infection. Studies have indicated that people with severe mental disorders are often at a socioeconomic disadvantage and have a greater prevalence of risky behaviours such as intravenous substance abuse and risky sexual practices.

Patients with schizophrenia have been found to be at higher risk for tuberculosis than the general population due to factors such as a history of substance abuse, poor nutrition, homelessness, or previous time spent in an institution or prison.

Suicide

Severe mental disorders are associated with elevated suicide rates. For example, the mortality rate due to suicide is estimated to be over 12 times greater among people with schizophrenia compared to the general population. A history of suicide attempts, depression, not taking

medications as prescribed, and drug and alcohol misuse are risk factors for suicide among patients with schizophrenia and bipolar disorder.

Lifestyle and health risk behaviours

Behaviours leading to poor self-care, such as tobacco use and lack of exercise are associated with depression, schizophrenia and bipolar disorder and can lead to chronic illnesses such as coronary heart disease and Type 2 diabetes. Patients with schizophrenia are more likely to smoke .The prevalence of smoking among them is about three times more than that in the general population.

Once chronic illness has developed, the severe mental disorders associated with poor self-care can lead to worse health outcomes and higher mortality rates.

Symptoms of the mental disorders themselves can cause barriers to seeking care, as well as difficulty with following medical advice. Barriers to include: the attitudes of health-care workers, disregard for physical health concerns by caregivers, and a lack of communication between health-care professionals providing physical and mental health care. Social and economic consequences of severe mental disorders include increased risk of poverty, unemployment, social isolation and social stigma. These factors can increase psychological stress and unhealthy behaviours (such as smoking), which in turn increase the risk of chronic illness.

Physical activity can have a protective effect against the effects of severe depression and psychological stress on cardiovascular disease.

Other service-related factors

There is some evidence that people with a severe mental disorder do not receive the same levels of care and treatment for their physical health as the general population. In the majority of cases, people with mental disorders are often at a disadvantage as compared with the general population due to unemployment, living in institutions, isolation and exclusion, as well as socioeconomic status – all risk factors that can prevent recovery as well as lead to poor health and premature mortality.

Key actions

Actions that can be taken to improve both the physical and mental health of people with severe mental disorders:

1. 1) Create protocols for both the physical and mental health needs of patients with severe mental disorders in the following areas: prevention, identification, assessment and treatment.

2. 2) Improve access to general health services through the integration of physical and mental health services.
3. 3) Work to overcome the stigma often associated mental illness and end discrimination that sufferers often endure.

WHO response

WHO advises its Member States on developing and implementing effective policies, strategies and plans to improve the health, both physical and mental, of people living with severe mental disorders.

The WHO Comprehensive Mental Health Action Plan, endorsed by the World Health Assembly in 2013, outlines the need to improve access to and quality of care for people with severe mental disorders, with the specific inclusion of general physical health care. The Action Plan promotes integrated health-care delivery that promotes mental health alongside physical health at all levels of care.

The mental health GAP Action Programme (MH GAP) aims at scaling up services for mental, neurological and substance use disorders for countries, particularly low- and middle-income countries. The Programme refers to the importance of monitoring the physical health of people with mental illness.

WHO Quality Rights Project aims to improve the quality of care and human rights conditions in mental health and social care facilities and to empower organizations to advocate for the health of people with mental disorders. The WHO Quality Rights toolkit includes a standard framework for ensuring that mental health facilities are equipped with adequate services for general and reproductive health.

Appendix 4

SDQ Survey

1. What is your gender?

◯ Female

◯ Male

2. What is your age? Please enter as a whole number.

[   ]

3. Are you White, Black or African-American, American Indian or Alaskan Native, Asian, Native Hawaiian or other Pacific islander, of multiple races, or some other race?

◯ White

◯ Black or African-American

◯ American Indian or Alaskan Native

◯ Asian

◯ Native Hawaiian or other Pacific Islander

◯ From multiple races

Other (please specify)

[   ]

4. What is your employment status?

◯ I work full-time

◯ I work part-time

◯ I am unemployed

◯ I am retired

◯ I am disabled and unable to work

5. Are you currently receiving treatment for a medical illness?

◯ I am not receiving treatment for a medical illness

◯ I am receiving treatment for a medical illness

6. Please list your medical illnesses

[   ]

7. Are you currently receiving treatment for a psychological condition or illness?

◯ I am not receiving treatment for a psychological condition or illness

◯ I am receiving treatment for a psychological condition or illness