

Reducing the Burden of Depression

Gavin Andrews, MD¹

Objective: To consider why the burden of depression persists.

Method: The epidemiology and disability associated with depression were reviewed to consider whether depression persists because: the causes are overwhelming, prevention is ineffective, the disease is difficult to detect or diagnose, the condition remits and recurs, treatments do not work, individuals do not seek treatment, or effective care is not provided when they do seek it.

Results: The first 5 possibilities were not considered significant reasons for the persistence of the burden.

Conclusion: The burden persists because individuals do not seek treatment for their depression when they relapse and effective proactive treatment is not always provided when they do seek it.

Can J Psychiatry 2008;53(7):420–427

Clinical Implications

- Routine screening of all primary care attenders would identify the patients who do not complain.
- Regular measurement of depression with instruments such as the Patient Health Questionnaire would screen for cases and indicate progress in treatment.
- Proactive care after diagnosis would increase adherence to medication and cognitive-behavioural therapy, and reduce relapse.

Limitations

- The current structure of practice is based around the acute care model that inhibits appropriate chronic care.
- The literature does not clearly differentiate between individuals who will only have a single episode of depression and those who have a history of recurrent depression and anxiety disorders.
- This review was limited to depression and should have discussed the anxiety and depressive disorders as one entity.

Key Words: prevalence, disability, persistence, treatment avoidance, proactive treatment

MDE is identified as prevalent, disabling, and a major determinant in the burden of disease. Nevertheless, waiting rooms are not full of individuals whose primary complaint is depression. How can this be? I will use data from the Australian NMHWS to illustrate some of the issues.

MDE is a syndrome of low mood, loss of interest, and other symptoms. There are no laboratory tests for MDE and diagnosis has depended on a trained clinician asking individuals about their symptoms. The DSM-IV¹ lists 5 criteria to satisfy the diagnosis. The first criterion lists 9 symptoms, 5 of which

must be present and at least 1 of the 5 must be depressed mood or loss of interest or pleasure for the diagnosis to be met. All must be judged to be significant by an experienced clinician in terms of severity, duration, abnormality, distress, and impairment. In addition, the diagnosis requires that the symptoms do not present as a mixed manic episode and MDE, do cause clinically significant distress and are not owing to substance use, general medical condition, bereavement, psychosis, and that there has never been a hint of mania. A total of 20 pieces of information is required before a clinician can safely make the diagnosis.

MD Epidemiology

Depressive symptoms in the population are common, but having symptoms is not the same thing as meeting criteria for a depressive disorder. In the NMHWS, 19% of adults reported at least 2 weeks of depressed mood or loss of interest in the past year, 6.3% met the full DSM-IV criteria at some point in the previous year, but only one-half of these were cases at the time of the NMHWS.²

Depression Is a Chronic Disorder. At the severe end of the spectrum, two 15-year prospective studies of individuals admitted to hospital with depression found these patients did not do well.^{3,4} Only a fifth of these 2 cohorts recovered and remained continuously well; three-fifths recovered but also had further episodes; a 10th were lost to suicide; and a 10th were always incapacitated by depression. A 12-year study in US specialist care, again presumably of people with more severe illnesses, showed that patients had symptoms in 60% of follow-up weeks and met full criteria for an MDE in 15% of those weeks.⁵ This is one of the few studies that have documented the significance of both subthreshold and threshold depression in a cohort followed for a substantial period. Cuijpers and Smit⁶ have assembled a series of studies showing that individuals with 1 to 4 symptoms have a 5-fold increase in risk of developing MD, compared with individuals with no symptoms.

Depression Remits and Recurs. The US National Comorbidity Survey showed that three-quarters of people aged 15 to 54 years who had ever met criteria for depression reported more than one episode. Their mean age was 34 years, and they reported an average of 4 MDEs in the 11 years since their first MDE.⁷ The World Health Organization's Global Burden of Disease 2000 study estimated a mean MDE duration of 26 weeks⁸ and the literature is consistent with this.⁹ The median duration of an MDE is less, around 13 weeks. If MDEs have a mean duration of about 6 months, some episodes will last weeks, others (perhaps 5% to 10%) will not remit for some years, and variation in duration is considerable. Episodes recur, with the average number of MDEs predicted from community survey data being around 8 in an individual's lifetime,

but again variation in recurrence is considerable. Thus the average individual with MD can expect to meet criteria for an MDE for 4 years in their lifetime. In addition, judging from the data on more severe cases, they will report symptoms of depression that do not meet criteria for a diagnosis but nevertheless are associated with some disability for 3 or 4 times as long, that is for 12 to 16 years in their lifetime. However, provided they do not commit suicide, they can expect 60 years in their lifetime without any depressive symptoms, including some 35 years of their working life.

Depression Occurs Throughout the Lifespan. In the US National Comorbidity Study, 17% remembered symptoms that met criteria for MD in their lifetime to date; however, their average age was only 34 years, therefore the rates were reduced because of failure to recall and because only part of the age of risk had passed.⁷ A modelling study, using Australian and Dutch data, and allowing for age of respondent and recall bias, estimated the lifetime risk of at least one MDE as 30% for male respondents and 40% for female respondents.¹⁰ On the basis of 3 prospective cohorts followed from birth to 26 years,¹¹ from 24 to 50 years,¹² and from 70 to 85 years¹³ we have estimated that at least one-half the population will experience at least one MDE during their lifetime.¹⁴ This explains why depression is such an important public health problem.

MD Disability and Burden

Moussavi et al¹⁵ studied a quarter of a million respondents in 60 countries and found that MD produces a greater decrement in health than other chronic diseases such as angina, arthritis, asthma, and diabetes.¹⁵ In addition they found that those with angina, arthritis, asthma, or diabetes also had increased risks of MD. When MD was comorbid with any of these diseases, the disability score was worse than with any other pair of these chronic physical diseases (Table 1).

The first Global Burden of Disease report¹⁶ came to a similar conclusion. Nonfatal burden for a disease was estimated from the product of prevalence multiplied by expert judgments about the average disability level. Many were surprised when MD was ranked as the leading determinant of disability in the world.¹⁶ Subsequent burden of disease studies have confirmed this finding, both in developed countries such as Australia and in the world at large.^{17,18} Mental health surveys have previously defined the prevalence of MD in different countries; however, the disability used in the burden of disease studies has remained dependent on expert judgments, an indirect and possibly erroneous measure. The Moussavi et al study¹⁵ is based on direct measures of disability and supports the finding that MD is associated with high levels of disability.

Abbreviations used in this article

CBT	cognitive-behavioural therapy
DSM	Diagnostic and Statistical Manual of Mental Disorders
ICD	International Classification of Diseases
MD	major depression
MDE	major depressive episode
NICE	National Institute for Health and Clinical Guidance
NMHWS	National Mental Health and Wellbeing Survey
PHQ-9	Patient Health Questionnaire

Table 1 Depression is the leading cause of disability

Top 5 causes of disability in Australia ¹⁷	Of total YLDs, %	Top 5 causes of disability in the world ¹⁸	Of total YLDs, %
Depression	7.9	Depression	9.4
Dementias	5.6	Hearing loss (adult)	5.5
Asthma	4.8	Cataracts	5.2
Osteoarthritis	4.8	Osteoarthritis	3.2
Hearing loss (adult)	4.1	Vision disorders (adult)	3.1

YLD = year lived with disability

MD is a chronic disorder that remits and recurs. Could a remitting disorder generate such disability? The point prevalence of MD is similar to the chronic physical diseases studied; however, the lifetime risk, the number of individuals who cycle in and out of depression, is 5 to 10 times greater than the lifetime risk of any of the other diseases studied. When it does occur, depression is more disabling than either of the 4 chronic physical diseases, angina, arthritis, asthma, or diabetes.

Why Does the Burden of a Disease Persist?

Why does the burden of any disease persist? The burden of any disease, we would argue, persists under numerous circumstances, including:

- causes are overwhelming (for example, in epidemics);
- prevention is ineffective (for example, in asthma);
- the disease is difficult to detect or diagnose (for example, in carcinoma of head of the pancreas);
- the condition remits and recurs (for example, in migraine);
- treatment is not effective, too expensive, unavailable, or unacceptable (for example, in chronic obstructive pulmonary disease);
- individuals do not seek treatment (for example, in hypertension);
- effective care is not provided.

Does this model explain why the burden of MD remains so great?

Do the Causes of Depression Overwhelm?

Probably Not. Genes and environmental stressors, and comorbidity with physical illness or owing to medications or substance abuse are the usual answers to what causes MD. If MD, worldwide, is the single largest determinant of disability it might be expected that the literature on causation would be

detailed and considerable. It is not. In a review and metaanalysis, Sullivan et al¹⁹ concluded that the point estimate of heritability of liability was 37%, probably the lowest for any of the internalizing disorders.²⁰ Adverse life events are one class of environmental events that precede depression; however, some of the relation is noncausal as individuals predisposed to, or with, MD put themselves into high-risk environments.^{21,22} Some adversities are associated with increased risks of MD many years later, child sexual abuse is one example.²³ The current interest is in gene and environment interactions in which adversity becomes salient in individuals with the requisite genetic vulnerability; however, these influences do not account for most MDEs.^{24,25}

The problem with the adversity literature is its lack of specificity—most individuals exposed to adversity do not develop a mental disorder, let alone MD. Further caution is required given that some high-trauma countries do not have high rates of depression.²⁶

Demographic correlates are not helpful. In the NIMHWS, MD was significantly more common (adjusted odds ratios) in women, less common in the elderly, and more common in those not married, with less education, and not in the labour force.² The last 3 variables are markers of deprivation; however, whether individuals are depressed because of their deprived status or have a deprived status because of recurrent depression is unclear.

If depression is a disorder, that is, a failure of adaptation that can affect one-half of the human population at some time, then it is difficult to discern a predominant cause. Nesse²⁷ argued that some episodes of depression might have evolutionary explanations and be adaptive. Others hope that causes (and treatments) may become clearer if we look at subtypes of depression.²⁸ Either way, it is difficult to see that the causes presently identified could overwhelm our ability to prevent or treat MD.

Table 2 Comparison of full and restricted DSM-IV Criterion A of MD in respondents meeting criteria for any anxiety, affective, or substance use disorder in the NSMHW (n = 1013)³⁴

Restricted criteria	Full criteria		Total, n
	Met criteria >4/9, n	Did not meet >4/9, n	
Met >2/5	315	8	323
Did not meet >2/5	24	666	690
Total	339	674	1013

Sensitivity = 0.93; specificity = 0.99; overall agreement = 0.97

Can Prevention Work?

Probably. We have argued that prevention of MD in young individuals is, or could be, effective.^{29,30} Cuipers et al³¹ came to a similar conclusion. If the median age of onset of MD is 32 years and the 25th percentile is 18 years, then it is imperative that we deliver universal prevention programs in high schools. We are presently delivering courses³² to help junior high school students manage stress, anxiety, and depression that fit within the demands of the high school curriculum. We have data that the course on stress management is effective and are gathering data on the effectiveness of the other 2 courses.³²

Is Depression Difficult to Detect or Diagnose?

Yes and no, but there are issues.

The Present Diagnostic Criteria Are Difficult to Remember and Could Be Shortened. One problem for a clinician is the difficulty of remembering the criteria. Humans can routinely recall no more than 5 to 9 items from a list. Zimmerman et al³³ reviewed evidence that the 9 symptoms in Criterion A in MD were poorly recalled. Depression is a common disease that all physicians should be able to screen for. It is difficult to screen a patient if you cannot remember the criteria (Table 2).

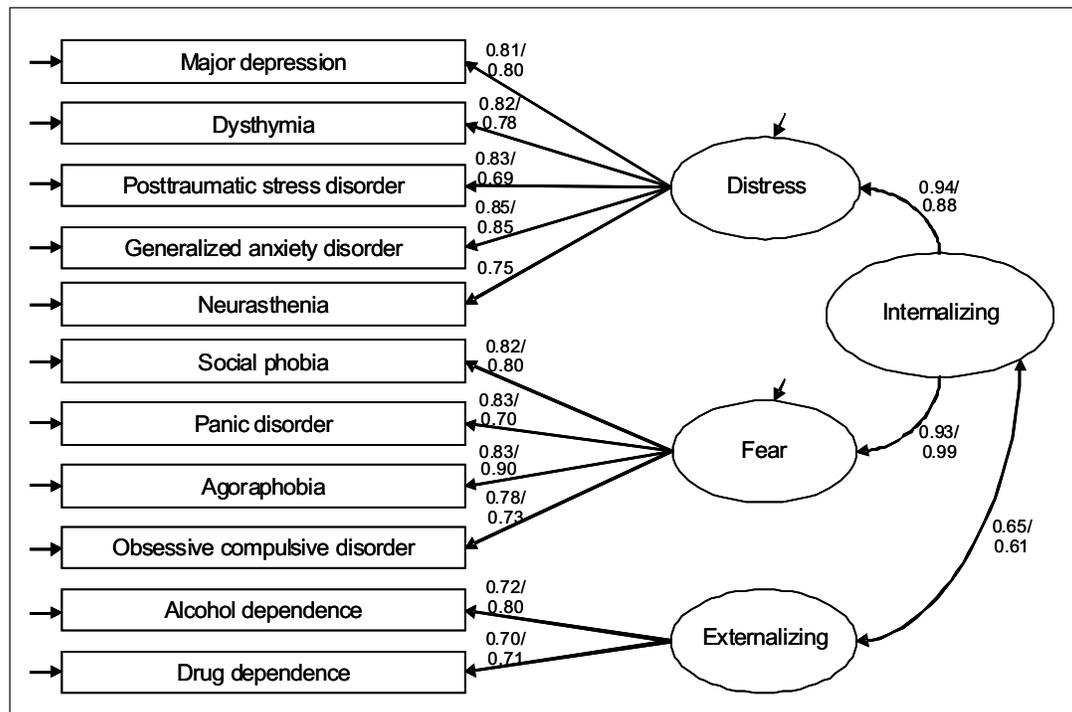
If it were possible to simplify the diagnostic criteria without loss of validity the traditional clinical interview may become more reliable. Zimmerman et al³³ concentrated on the 5 psychological symptoms contributing to Criterion A (depression, poor interest, worthlessness, poor concentration, and thoughts of death) and found that almost all patients who had met criteria for 5 or more of the 9 traditional symptoms met this new criteria for 3 of the 5 psychological symptoms. We confirmed his finding; 3/5 symptoms produced 97% agreement with the usual 5/9 criterion.³⁴ That one criterion could be simplified without loss of validity is understandable; however, could the 20 pieces of information and 5 criteria that form the diagnosis be simplified? In MD, 6 pieces of information from the NMHWs (5 symptoms of Criterion A combined with no history of mania and [or] hypomania) identified most

of the same individuals as had the full criteria that required 20 pieces of information.

Depression and Anxiety Disorders Are Internalizing Disorders. Are the individual depressive and anxiety disorders distinct? If this were the case then the rates of cooccurrence among these disorders would occur at chance levels. However, the rates of cooccurrence among these mental disorders are considerably higher than would be expected.^{35,36} It was suggested that such rates could reflect the existence of higher-order dimensions of psychopathology. Numerous studies have examined this and found similar groupings of mental disorders. Slade and Watson³⁷ identified a hierarchical 3-factor structure as the best fit to 10 common DSM-IV and 11 common ICD-10 mental disorders. This structure was characterized by correlated distress and fear factors (which were best considered lower-order facets of a broader internalizing factor) as well as an externalizing factor. As can be seen in Figure 1, the individual mental disorders that were characteristic of the distress factor were MD, dysthymia, generalized anxiety disorder, posttraumatic stress disorder, and neurasthenia (the latter in the ICD-10 model; in DSM-IV, neurasthenia would be coded as undifferentiated somatoform disorder 300.81). The mental disorders that were characteristic of the fear factor were social phobia, agoraphobia, panic disorder, and obsessive-compulsive disorder. In this study, the externalizing factor was best characterized by drug and alcohol dependence (Figure 1).

Depression Is a Dimension Not a Category. A diagnosis of MD is currently made when there are 5 or more of the 9 symptoms specified in Criterion A. Is there evidence of a natural break in the distribution of symptoms at or around this threshold? Numerous large studies have found that the relation between the number of symptoms and measures of impairment was linear, with no evidence of any natural discontinuity that would support the use of 5/9 symptoms as a critical diagnostic threshold.^{7,38,39} One of the implications is that the decision to treat can be made at any level on the continuum.⁴⁰ The frequent use of Depressive Disorder Not Otherwise

Figure 1 Best fitting model of the structure of 10 common DSM-IV and 11 common ICD-10 mental disorders from the Australian National Survey of Mental Health and Well-being, 1997. All parameter estimates (DSM-IV/ICD-10) are standardized and significant at $P < 0.05$.^a



Note: All parameter estimates, except for Neurasthenia, relate to DSM-IV and ICD-10. The single parameter estimate for Neurasthenia relates to ICD-10 only.

^aReprinted from Slade and Watson³⁷ with permission.

Specified⁴¹ makes it clear that clinicians often ignore the DSM-IV threshold that requires so many pieces of information. One of the reasons that the burden persists may be that our medical model of a treatment threshold obscures the need for stepped care to match treatment intensity to case severity.

Routine Screening. One response to the dimensional nature of MD is to routinely use a short screening scale. In our clinic, we have every patient complete the 9-item PHQ-9 at the registration desk each time they attend. The score is entered on the record by the receptionist. The PHQ-9 is a self-report instrument that parallels the symptom criteria for depressive disorders listed in DSM-IV. Depression severity is judged from the total score.⁴² Because of its brevity and sensitivity to change over time, the PHQ-9 can be used for screening new patients for depression as well as for routine evaluation of outcome and response to treatment. There are longer and more detailed scales available; however, the PHQ-9 is recommended as simple, acceptable to patients, and practical for clinicians to use. It is brief, reliable, and valid.⁴³

Depressed Patients May Not Complain. At the 15-year follow-up of a cohort of patients with MD, we discovered that only one-fifth of the patients had remained continuously well

after discharge from hospital.³ Few of the four-fifths who relapsed returned for treatment or sought other treatment. Depression seemed to rob individuals of their motivation to be well, despite the disability. In a similar vein, depressed patients in primary care with a normalizing attributional style were very unlikely to be detected unless one relied on a structured questionnaire; they accepted their depressed state but did not complain.⁴⁴ In a seminal primary care study, Kirmayer et al⁴⁵ noted that three-quarters of individuals with MD presented with somatic complaints, and that such presentations more than halved the rate at which clinicians recognized depression. MD may not be simple to diagnose unless the doctor asks or the clinic routinely uses a screening scale.

Depression That Remits and Recurs Leads to Treatment Difficulties

Yes, this is a problem. All chronic diseases wax and wane but depression can disappear completely for years and then return. Does this affect efforts to reduce the burden? If the median duration of an episode is 13 weeks, then by the time the average individual has thought of getting treatment, remission might have begun. In the NMHS 65% of individuals with depression at the time of interview reported at

least one mental health consultation in the past year, and 50% reported potentially effective treatment with medication or CBT. An equal number of individuals had met criteria during the year but had remitted by the time of interview. They reported similar rates (59%) of one or more mental health consultations but only 36% reported getting medication or CBT, as though the beginning of recovery from this episode reduced the need for effective treatment to be initiated; whereas, if this was not the first episode, treatment should have been maintained for at least 2 years.

Response to treatment is greeted with relief by patient and doctor, although the recovery may be due to natural remission and quite independent of treatment. Few clinicians are comfortable talking to patients about the prospect of relapse and the need for continuing treatment. When we argued that depression should be treated as a chronic disease, even in remission, numerous doctors wrote to say that they were relieved that they could stress the need for ongoing treatment and broach the possibility of relapse.⁹ Active treatment should be continued for 2 years in individuals with a history of recurrent depression, and patients should learn to recognize the early warning signs of relapse.

Treatment for Depression Is Difficult, Ineffective, Expensive, and Unavailable

No, treatment is none of these things. There are many sensible guidelines for practice.^{46,47} Antidepressant medication is widely prescribed, moderately effective, cost-effective, associated with reductions in suicide, and increasingly acceptable to the public. The evidence supporting CBT is similarly robust, although estimating rates of adequate CBT are much harder to measure than prescription rates. We paraphrase the simplest level in the NICE guidelines⁴⁶: For mild depression, provide advice about sleep hygiene, exercise, and problem solving plus self-help material, often in the form of computerized CBT, followed by review within 2 weeks. Antidepressants are not recommended, as the risk and benefit ratio does not justify such treatments. In moderate depression, antidepressants are strongly recommended, coupled with CBT, either face to face or via computer, problem solving, and behavioural activation. Fluoxetine or citalopram were favoured over all other antidepressants on the grounds of costs and safety. If one of these drugs does not work then transfer to the other would be indicated before referral for advice about use of the remaining antidepressants.⁴⁸ Treatment of severe depression is essentially the same⁴⁹ plus referral for specialist advice, including advice about the value of electro-convulsive therapy. Therefore, the issue is not what to do once the depression is recognized—there is already persuasive evidence that different treatments are appropriate for different degrees of depression severity—the issues are recognition and prudent continuing management of this chronic disease. When MD is

comorbid with physical disorders, the indicated treatments are a little different: treatment depends on the severity of depression, and caution regarding interactions between medications is necessary, and proactive care essential.

Individuals With Depression Do Not Come for Treatment

This is only partly true. In the NMHS, just less than one-half of the individuals who met criteria for MD during the year reported no mental health consultation. One-third of those saw no need for treatment, saying, “I prefer to manage myself”; however, two-thirds saw a need for treatment and most thought that information, counselling, or practical help would meet their needs but had not asked. However, only 1 in 6 individuals who had not consulted thought that medication or CBT would be appropriate. Patient education as to the therapies that are likely to benefit is clearly an issue in most individuals who do not seek treatment. Fortunately this appears to be changing.⁵⁰

The Organization of Care Itself Contributes to the Continued Burden

Could we provide more effective care? A demand-driven model of practice in which patients seek help only when they deem it necessary can never be appropriate for an episodic, but lifelong, condition that affects hope and volition, reduces compliance, and predisposes to suicide. When patients relapse they often perceive that they have failed their clinicians and their families and, therefore, decide to suffer in silence. We should discuss with them the chronic nature of their disorder and steps needed to manage it, exactly as we would discuss these issues with an individual with diabetes or asthma.⁹ Participatory self-management, predicated on a written action plan, is the hallmark of self-management programs that reduce morbidity. In asthma training programs this improvement in outcome began to be measurable because patients started to measure themselves. Patients using the PHQ-9 can do likewise.

Wagner et al⁵¹ describe a model for the management of chronic disease and Katon et al⁵² have applied this model to depression to prevent chronicity and relapse. There seem to be 4 components:

- Practice reorganization—establish a register of cases and proactively organize consultations according to acuity, including outreach to nonattenders. Routinely measure severity.
- Patient education—use booklet, the web, and family meetings to ensure that all concerned understand the nature of depression, its treatment, and the early warning signs that herald relapse.
- Expert systems—have stepped care protocols for diagnosis and management of complex cases, together

with criteria for specialist consultation and for sharing care with nurses and clinical psychologists.

- Computer support—use programs that record treatment and outcomes of all patients, and flag when progress is not as expected.

Proactive care, in accord with the Wagner criteria, produced better outcomes in the first year. Continued enhanced care is required; however, as individuals with MD generate twice the health care costs of other primary care attenders and while proactive care costs more than usual care, the cost per successfully treated patient is lower and the cost-effectiveness is higher, compared with patients receiving usual care.

Whitty and Gilbody⁵³ reviewed the United Kingdom's NICE guidelines and called for investment in case management and related organizational changes in primary care. They later reviewed the incremental cost-effectiveness of these case management and collaborative care enhancements, reporting, on the basis of 11 studies of 4600 patients, that the incremental cost-effectiveness ratios of enhanced primary care treatment was \$13 to \$25 per depression-free day.⁵⁴ If this means increased work force productivity then such strategies are indeed cost-effective. In a subsequent metaanalysis, they found that depression outcomes from collaborative care remained improved at 6 months and at 5 years. They related the improved effectiveness to supervision by experienced case managers that lead, among other changes, to improved medication compliance.⁵⁵

We might expect that the United Kingdom's National Health Service could implement the collaborative care that seems so effective in reducing the burden of depression; however, Whitty and Gilbody⁵³ doubted whether the required changes would be implemented. In the United States, some health care systems have been able to implement such programs; however, how widespread these programs are is uncertain.⁵⁶ Private practice fee-for-service models, whether insurance-based or not, do not encourage proactive collaborative care. Australia is edging toward subsidizing the work of practice nurses for specific tasks (for example, spirometry); however, despite the expansion of access of allied health staff and nurses to fee for service insurance payments, there is no provision for proactive collaborative care in the longer term. The problem is that the health system is still predicated on the acute care model—get sick, go to doctor, get treatment, become well—that is totally inappropriate in an environment in which the management of chronic diseases such as depression is a dominant activity. If we want to reduce the burden of depression we will need to change the way we treat individuals with depression, not necessarily change the treatments we use, but change the way we organize their treatment.

Funding and Support

The Canadian Psychiatric Association proudly supports the In Review series by providing an honorarium to the authors.

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington (DC): APA; 1994.
2. Andrews G, Henderson S, Hall W. Prevalence, comorbidity, disability and service utilisation. Overview of the Australian National Mental Health Survey. *Br J Psychiatry*. 2001;178:145–153.
3. Kiloh LG, Andrews G, Nielson MD. The long term outcome of depression. *Br J Psychiatry*. 1988;153:752–757.
4. Lee AS, Murray RM. The long-term outcome of Maudsley depressives. *Br J Psychiatry*. 1988;155:741–751.
5. Judd LL, Hagop SA, Maser JD, et al. A prospective 12 year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. *Arch Gen Psychiatry*. 1998;55:694–700.
6. Cuijpers P, Smit F. Subthreshold depression as a risk indicator for major depressive disorder: a systematic review of prospective studies. *Acta Psychiatr Scand*. 2004;109:325–331.
7. Kessler KS, Zhao S, Blazer DG, et al. Prevalence, correlates and course of minor depression and major depression in the national comorbidity survey. *J Affect Disord*. 1997;45:19–30.
8. Usten TB, Ayuso-Mateos JL, Chatterji S, et al. Global burden of depressive disorders in the year 2000. *Br J Psychiatry*. 2004;184:386–392.
9. Andrews G. Should depression be managed as a chronic disease? *BMJ*. 2001;322:419–421.
10. Kruijshaar ME, Barendregt J, Vos T, et al. Lifetime prevalence estimates of major depression: an indirect estimation method and a quantification of recall bias. *Eur J Epidemiol*. 2005;20:103–111.
11. Jaffee SR, Moffitt TE, Caspi A, et al. Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Arch Gen Psychiatry*. 2002;59:215–222.
12. Parker G. Is depression overdiagnosed? *Yes*. *BMJ*. 2007;335:328.
13. Palsson SP, Ostling S, Skoog I. The incidence of first-onset depression in a population followed from the age 70 to 85. *Psychol Med*. 2001;31:1159–1168.
14. Andrews G, Poulton R, Skoog I. Lifetime risk of depression: restricted to a minority or waiting for most? *Br J Psychiatry*. 2005;187:495–496.
15. Moussavi S, Chatterji S, Verdes E, et al. Depression, chronic disease and decrements in health: evidence from the World Health Surveys. *Lancet*. 2007;369:851–858.
16. Murray CJL, Lopez A, editors. The global burden of disease. Cambridge (MA): Harvard University Press; 1996.
17. Mathers C, Vos T, Stevenson C. The burden of disease and injury in Australia. Canberra (ACT): AIHW; 1999.
18. Mathers C, Lopez A, Stein C, et al. Deaths and disease burden by cause. CDPP working paper 18. Bethesda (MD): National Institute of Health; 2004.
19. Sullivan PF, Neale MC, Kendler KS. Genetic epidemiology of major depression: review and meta-analysis. *Am J Psychiatry*. 2000;157:1552–1562.
20. Gross C, Hen R. The developmental origins of anxiety. *Nature Reviews*. 2004;5:545–552.
21. Kendler KS, Karkowski LM, Prescott CA. Causal relationship between stressful life events and the onset of major depression. *Am J Psychiatry*. 1999;156:837–841.
22. Poulton RG, Andrews G. Change in danger cognitions in agoraphobia and social phobia during treatment. *Behav Res Ther*. 1996;34:413–421.
23. Andrews G, Corry J, Slade T, et al. Child sexual abuse. In: Ezzati M, Lopez AD, Rodgers A, et al, editors. Comparative quantification of health risks volume 2. Geneva (CH): World Health Organization; 2004. p 1851–1940.
24. Caspi A, Sugden K, Moffitt TE, et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science*. 2003;301:386–389.
25. Zimmerman P, Bruckl T, Lieb R, et al. The interplay of familial depression liability and adverse events in predicting the first onset of depression during a ten-year follow-up. *Biol Psychiatry*. 2008;63:406–414.
26. The WHO World Mental Health Survey Consortium. Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health surveys. *JAMA*. 2004;291:2581–2590.
27. Nesse RM. Evolutionary explanations for mood and mood disorders. In: Stein DJ, Kupfer DJ, Schatzberg AF, editors. American psychiatric publishing textbook of mood disorders. Washington (DC): American Psychiatric Publishing; 2006. p 159–175.
28. Parker G. Beyond major depression. *Psychol Med*. 2005;35:467–474.
29. Andrews G, Szabo M, Burns J. Preventing major depression in young people. *Br J Psychiatry*. 2002;181:460–462.
30. Andrews G, Wilkinson DD. The prevention of mental disorders in young people. *Med J Aust*. 2002;177:S97–S100.
31. Cuijpers P, Van Straten A, Smit F. Preventing the incidence of new cases of mental disorders: a meta-analytic review. *J Nerv Ment Dis*. 2005;193:119–125.

32. Andrews G, Van Vliet H, Wuthrich V. The reduction of anxiety in school children: preliminary results with www.climateschools.tv. In: Castle D, Hood S, Kyrios M, editors. Anxiety disorders: current controversies, future directions. Fitzroy (AUS): Australian Postgraduate Medicine; 2007. p 149–159.
33. Zimmerman M, Chelminski I, McGlinchey JB, et al. Diagnosing major depressive disorder X: can the utility of the DSM-IV symptom criteria be improved? *J Nerv Ment Dis.* 2006;194:893–897.
34. Andrews G, Slade T, Sunderland M, et al. Issues for DSM-V: simplifying DSM-IV to enhance utility: the case of major depressive disorder. *Am J Psychiatry.* 2007;164:1784–1785.
35. Andrews G, Stewart GW, Morris-Yates A, et al. Evidence for a general neurotic syndrome. *Br J Psychiatry.* 1990;157:6–12.
36. Andrews G, Slade T, Issakidis C. Deconstructing current comorbidity: data from the Australian National Survey of Mental Health and Well-being. *Br J Psychiatry.* 2002;181:306–314.
37. Slade T, Watson D. The structure of common DSM-IV and ICD-10 mental disorders in the Australian general population. *Psychol Med.* 2006;36:1593–1600.
38. Ustun TB, Sartorius N, editors. *Mental illness in general health care: an international study.* London (GB): John Wiley & Sons; 1995.
39. Sakashita C, Slade T, Andrews G. An empirical analysis of 2 assumptions in the diagnosis of DSM-IV Major Depression Episode. *Aust NZ J Psychiatry.* 2007;41:17–23.
40. Kraemer HC, Shrout P, Rubio-Stipec M. Developing the diagnostic and statistical manual V: what will “statistical” mean in DSM-V? *Soc Psychiatry Psychiatr Epidemiol.* 2007;42(4):259–267.
41. Andrews G, Anderson T, Slade T, et al. Classification of anxiety and depressive disorders: do you want to be right or want to be effective? *Depress Anxiety.* Forthcoming 2008 May.
42. Kroenke K, Spitzer RL. The PHQ-9: a new depression diagnostic and severity measure. *Psychiatr Ann.* 2002;32:1–7.
43. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med.* 2001;16:606–613.
44. Kessler D, Lloyd K, Lewis G, et al. Cross sectional study of symptom attribution and recognition of depression and anxiety in primary care. *BMJ.* 1999;318:436–440.
45. Kirmayer LJ, Robbins JM, Dworkind M, et al. Somatization and the recognition of depression and anxiety in primary care. *Am J Psychiatry.* 1993;150:734–741.
46. National Institute for Clinical Excellence. *Depression: management of depression in primary and secondary care—NICE guidance. Report CG23.* London (GB): National Institute for Health and Clinical Excellence; 2004.
47. AHCPR depression guideline panel. *Depression in primary care: detection, diagnosis, and treatment. Technical report no 5.* Rockville (MD): US Department of Health and Human Services, Public Health Service; 2000.
48. Rush JA, Maurizio FB, Wisniewski SR, et al. Sequenced treatment alternatives to relieve depression (STAR*D): rationale and design. *Control Clin Trials.* 2004;25:119–142.
49. Simon J, Pilling S, Burbeck R, et al. Treatment options in moderate and severe depression: decision analysis supporting a clinical guideline. *Br J Psychiatry.* 189:494–501.
50. Jorm AF. Mental health literacy: public knowledge and beliefs about mental disorders. *Br J Psychiatry.* 2000;177:396–401.
51. Wagner EH, Austin BT, Von Korff. Organizing care for patients with chronic illness. *Millbank Q.* 1996;74:511–543.
52. Katon W, Von Korff M, Lin E, et al. Population based care of depression: effective disease management strategies to decrease prevalence. *Gen Hosp Psychiatry.* 1997;19:169–178.
53. Whitty P, Gilbody S. NICE, but will they help people with depression? The new National Institute for Clinical Excellence depression guidelines. *Br J Psychiatry.* 2005;186:177–178.
54. Gilbody S, Bower P, Whitty P. Costs and consequences of enhanced primary care for depression. *Br J Psychiatry.* 2006;189:297–308.
55. Gilbody S, Bower P, Fletcher J, et al. Collaborative care for depression: a cumulative meta-analysis and review of longer-term outcomes. *Arch Intern Med.* 2006;166:2314–2321.
56. Dietrich AJ, Oxman TE, Williams JW, et al. Re-engineering systems for the treatment of depression in primary care: cluster randomized controlled trial. *BMJ.* 2004;329:602–607.

Manuscript received and accepted December 2007.

¹Director, Clinical Research Unit for Anxiety and Depression, School of Psychiatry, University of New South Wales at St Vincent’s Hospital, Sydney, Australia.

Address for correspondence: Dr G Andrews, 299 Forbes St, Darlinghurst, NSW, Australia 2010; gavina@unsw.edu.au

Résumé : Réduire le fardeau de la dépression

Objectif : Examiner pourquoi le fardeau de la dépression persiste.

Méthode : L’épidémiologie et l’incapacité associées à la dépression ont été examinées pour déterminer si la dépression persiste parce que : les causes sont accablantes, la prévention est inefficace, la maladie est difficile à détecter ou à diagnostiquer, l’affection entre en rémission et revient, les traitements ne fonctionnent pas, les personnes ne veulent pas de traitement, ou des soins efficaces ne sont pas dispensés quand elles en veulent.

Résultats : Les 5 premières possibilités n’ont pas été considérées comme étant des raisons significatives de la persistance du fardeau.

Conclusion : Le fardeau persiste parce que les personnes ne veulent pas de traitement pour leur dépression quand elles rechutent, et qu’un traitement proactif efficace n’est pas toujours dispensé quand elles en veulent.