American Journal of Clinical Hypnosis, 55: 249–271, 2013

ISSN: 0002-9157 print / 2160-0562 online
DOI: 10.1080/00029157.2012.740607

Routledge
Taylor & Francis Group

The Power of Belief and Expectancy in Understanding and Management of Depression

Assen Alladin
University of Calgary, Calgary, Alberta, Canada

This article examines how beliefs can influence the definition, classification, understanding, and treatment of depression. It is organized in five parts: The first part critically reviews the definition of depression; the second part explores the medicalization of depression; the third part examines the role of the pharmaceutical industry in the promotion and marketing of antidepressant medications; the fourth part surveys the psychological therapies for depression and examines the role of expectancy in outcome; and the last part looks at the mechanisms involved in the placebo effect. A list of evidence-based strategies, including hypnosis, are discussed in the context of cognitive hypnotherapy for depression to illustrate how expectancy effect can be maximized in psychotherapy.

Keywords: antidepressant, cognitive therapy, depression, expectancy, hypnotherapy, pharmacotherapy, placebo

Depression is a pervasive condition and the problem seems to be on the increase both in adults (Fombonne, 1994, 1999; Goodwin, Jacobi, Bittner, & Wittchen, 2006; Kessler & Wang, 2009) and adolescents (Rudoph, Hammen, & Daley, 2006). Major depressive disorder (MDD), major depression, mood disorder, depression, affective disorder, or depressive disorder will be used interchangeably in the present article to denote MDD. DSM will be used to refer to all editions of the Diagnostic and Statistical Manual of Mental Disorders—citation of a specific edition will be dated. The World Health Organization (WHO) has estimated that approximately 121 million people in the world suffer from depression at any one time and it is the fourth leading cause of burden among all diseases (WHO, 2002).

Depression is also on the increase (Fombonne, 1994, 1999; Goodwin et al., 2006; Wang & Kessler, 2006). A review of 22 national and international epidemiologic studies of depressed adults (Goodwin et al., 2006) concluded that there is a "remarkable and consistent increase in prevalence rates of major depression across studies . . . over the past three decades" (p. 39). For example, the 1-year and the lifetime prevalence rates of 2.7% and 4.9% respectively from 1991 (Weissman, Bruce, Leaf, Florio, & Holzer, 1991) increased to 6.6% and 16.2% respectively in 2003 (Kessler et al., 2003; Kessler

Address correspondence to Dr. Assen Alladin, University of Calgary, Psychiatry, 1403 29 Street NW, Calgary, Alberta T2N 2T9, Canada. E-mail: assen.alladin@albertahealthservices.ca

et al., 2005). The WHO Global Burden of Disease Study has projected that by the year 2020 the prevalence of depression will increase and it will become the second leading cause of Disability Adjusted Life Years lost. There is also ample evidence that the rates of depression in adolescents have increased in recent years compared to those of previous decades (Cross-National Collaborative Group, 1992; Kessler, Avenevoli, & Merikangas, 2001; Rudolph et al., 2006).

Causes of the Increase in Depression

Many factors—biological, psychological, and social—or a combination of these factors (Gilbert, 2004) might have contributed to the increase in depression over the past few decades. Recently, some writers (e.g., Double, 2006; Gordon, 2008; Greenberg, 2010; Horwitz & Wakefield, 2007; Kirsch, 2010; Leventhal & Martell, 2006; Moncrieff, 2008; Yapko, 2009, also this issue) have been advancing alternative explanations (as opposed to traditional views; e.g., methodological artifacts, the measures used, etc.) for the increase in prevalence of depression. Some of these writers find it paradoxical that despite major developments in medical and psychological understanding of depression and significant increase in the number of antidepressants, psychological interventions, mental professionals, and treatment facilities, the rate of depression continues to rise. For example, Greenberg (2010) believes the prevalence rates for depression reported by clinicians over the past decade are highly inflated because, in most cases, he thinks the diagnosis does not represent depression as mental illness but unhappiness labeled as a disease.

This article will look at three main factors that might have contributed to the increase in diagnosis and alleged overuse of drug treatment for depression, namely (1) definition of depression, (2) medicalization of depression, and (3) marketing of antidepressant (AD) medications. These three factors, particularly the medicalization of depression and the marketing of ADs, are selected for review here because (1) they offer alternative explanations that are not usually covered in regular textbooks; (2) they question the validity of the concept of depression; (3) they critically examine the effectiveness of ADs and psychotherapy for depression; (4) they illustrate how mass media and advertizing can shape beliefs and expectancy about depression and its treatment; and (5) they offer additional strategies (e.g., maximizing expectancy) for enhancing the effects of ADs and psychotherapy with depression.

The critical examination of these three factors in this article is not intended to dismiss the concept of depression nor undermine the suffering that depressed patients endure, or knock down the importance of ADs and psychotherapy in the management of depression. The purpose of this article is to encourage clinicians not to reflexively dismiss the growing literature on alternative or non-traditional views of depression, but to study them seriously. Some of these ideas may be helpful or easily integrated with their own

model of practice. For example, cognitive hypnotherapy (CH) for depression, which is described later, routinely utilizes hypnotic phenomena to enhance positive expectancy in depressed patients (Alladin, 2007). After reviewing these three factors, the effectiveness of cognitive behavior therapy (CBT) and interpersonal psychotherapy (ITP) with depression will be critically evaluated, and the important role of the placebo response in outcome will be highlighted. Finally, a list of evidence-based strategies, including hypnosis, will be discussed in the context of CH for depression to illustrate how expectancy effect can be maximized in psychotherapy.

Definition of Depression

Contemporary textbooks of mental illness or depression do not offer a working definition of depression; instead they list a range of symptoms. The concept of depression is briefly discussed here to highlight some of the arguments the opponents of DSM have advanced to underline the difficulties associated with the current DSM (DSM-IV-TR; American Psychological Association: APA, 2000) description and classification of depression. Before reviewing the criticisms, the DSM-IV-TR criteria for depression are listed and discussed below.

DSM-IV-TR Criteria for Major Depressive Episode

The current official diagnosis of MDD is based on the criteria laid down by the Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR; APA, 2000). To diagnose a person with a major depressive episode, five or more of the following nine symptoms must be present as described below:

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report or observation made by others (in children and adolescents it can be irritable mood).
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (based on self-report or observation made by others).
3. Significant weight loss (not due to dieting), or decrease or increase in appetite nearly every day.
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (self-report corroborated by others).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt nearly every day.
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (self-report or observed by others).

9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide (adapted from DSM-IV-TR: APA, 2000, p. 356).

The symptoms must have been present during the same 2-week period. This must represent a change from previous functioning and at least one of the symptoms ought to be either depressed mood (symptom 1) or loss of interest or pleasure (symptom 2).

In addition, the symptoms should cause clinically significant distress or impairment in social, occupational, or other important areas of functioning and should not meet criteria for a "mixed episode" (manic and depressive symptoms) and they should not be due to the direct physiological effects of a substance, or accounted for by bereavement.

Although with each revision of the DSM (DSM-I published in 1952; APA, 1952) the authors have incorporated the most current knowledge about depressive disorders and had relied on empirical methods to increase the reliability and validity of the diagnosis, there has been little change in the description of MDD since DSM-III (APA, 1980). In spite of the significant and revolutionary departure of DSM-III from DSM-II (APA, 1968) in the classification and description of mood disorders, only minor changes have occurred in the description and criteria of a major depressive episode from DSM-III through to DSM-IV-TR (Mayes & Horwitz, 2005; Parker, 2005; Pilgrim & Bentall, 1999). The initial DSM-III criteria for a major depressive episode were based on the influential work by Feighner and colleagues. (1972) and Spitzer, Endicott, and Robins (1978). Feighner's criteria for "primary" depression (see Goldstein & Anthony, 1988, p. 189) are almost the same as for DSM-III (DSM-R: APA, 1987, p. 217) through to DSM-IV-TR (APA, 2000, p. 356). Interestingly, the pre-published criteria for a major depressive episode in the awaited DSM-V are nearly the same (DSM-5: APA, 2012). Critics of the DSM, therefore, argue that little development has occurred in the classification of mood disorders since 1980 (e.g., Parker, 2005; Pilgrim & Bentall, 1999).

Criticisms of DSM-IV Definition of MDD

As the DSM criteria for depression is based solely on symptoms and not on signs (Parker, 2005), many clinicians and writers (e.g., Gordon, 2008; Moncrieff, 2007a) have questioned the validity of the current DSM (DSM-IV-R) concept of major depression. The main criticisms of the DSM revolve around reliability and validity.

The DSM Concept of Depression Lacks Validity

Gordon (2008) and others (e.g., Horwitz & Wakefield, 2007; Parker, 2005) believe the current classification of depression is less reliable and scientific than one is led to believe by the DSM-IV-TR (APA, 2000). They argue that the inclusion or exclusion of a psychiatric disorder in the DSM is not always based on science or developments in medicine. For example, in 1974, the diagnosis of "sexual orientation disturbance" (homosexuality)

was removed and replaced by "sexual disorder not otherwise specified" in the seventh printing of the DSM-III (Mayes & Horwitz, 2005; Spitzer, 1981) due to active lobbying and political campaigns (McCommon, 2006; Rissmiller & Rissmiller, 2006). On the other hand, the diagnosis of posttraumatic stress disorder was added to the DSM-III in response to pressure from Vietnam veterans (Halgin, 2003). These two examples illustrate that there is some subjectivity involved in the classification of a psychiatric disorder.

Proponents of DSM-IV-TR have challenged these criticisms (e.g., Ravindran & Kennedy, 2007b). For an informative debate between opponents (Moncrieff, 2007a, 2007b) and proponents (Ravindran & Kennedy, 2007a, 2007b) of the current DSM concept of depression see *Canadian Journal of Psychiatry* (2007, Vol. 52, No. 2, pp. 96–102). In the debate, Ravindran and Kennedy (2007b) assert that there is ample "evidence to support a disease-centred model of depression" (p. 98), while Moncrieff (2007a) contends that the evidence for such a model is inconsistent and non-specific. The debate illustrates the division in opinions of what constitutes depression and this has significant implications for diagnosis and treatment. For example, the practitioners who believe in the medical model of depression are more likely to take a medical approach to diagnosing and treating depression. On the other hand, practitioners who ascribe to a non-biological model of depression are more likely to advocate for a non-pharmacological approach to treatment. To illustrate this point, Pilgrim and Bentall (1999) have compared and contrasted the attitudes and beliefs that psychiatrists and psychologists hold about depression. They found each professional group to ascribe to a wide range of theories and models of depression. Their findings demonstrate that the understanding of depression is not based solely on science, but can also be influenced by training and professional loyalty. Proponents of DSM are aware of these flaws and limitations, but they believe in its usefulness for making a diagnosis (Frances, First, & Pincus, 1995) and they trust that with advances in etiology and treatments of depression, the classification of MDD will become more reliable and valid (APA, 2000; Frances et al. 1995; Halgin, 2003).

The Comorbidity of Anxiety With Depression

The relationship between depression and anxiety has been a contentious issue in the DSM-IV-TR classification of depression (e.g., Brown & Leyfer, 2010; Huppert, 2009). As there is a significant overlap of symptoms between anxiety and depression (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Dobson, 1985; Dozois & Westra, 2004), skepticism has been raised as to whether anxiety and depression are empirically distinct entities (Brown & Leyfer, 2010; Dozois & Westra, 2004). Huppert (2009) believes the commonalities between anxiety disorders and depression are an artifact of the DSM-IV-TR classification since the "symptom criteria for GAD, SAD, and PTSD . . . are also symptom criteria for MDD or dysthymia" (p. 578). Goldberg and Huxley (1992)

and Tyrer (1989) favor a general neurotic syndrome that includes varying degrees of anxiety and depressive symptoms. In accordance, the 10th edition of the International Classification of Diseases (ICD-10) has included "mixed anxiety and depressive disorder" in the classification of depressive disorders (WHO, 1993, p. 132). There are also disputes about the dividing line between depression and bipolar disorder and the relationship between depression and physical disease (Parker, 2005; Pilgrim & Bentall, 1999).

From the above review, it is apparent that there is no clear definition of depression. What constitute depression appears to be, to some extent, shaped by professional fidelity and allegiance. Moreover, because of the overlap with other disorders (particularly anxiety), the validity of the concept of depression is questioned. Furthermore, as it is not clear what constitutes depression and since there is no biological marker for depression, there may be some elements of subjectivity involved in the diagnosis. These concerns have led many clinicians and writers to question the validity of the DSM concept of depression (e.g., Gordon, 2008; Nesse & Stein, 2012). The next two sections explore the medical and pharmaceutical influences that might have been involved in the medicalization of the concept of depression.

Medicalization of Depression

Recently, many writers, including psychiatrists (e.g., Curtiss, 2001; Double, 2006; Gordon, 2008; Greenberg, 2010; Horwitz & Wakefield, 2007; Kirsch, 2010; Leventhal & Martell, 2006; Moncrieff, 2008; Parker, 2005; Yapko, 2009, also this issue), have questioned the validity of depression as a *disease*. They believe the DSM-IV-TR (APA, 2000) concept of depression fails to differentiate between depression as an illness and depression as a natural reaction to negative life-events. For example, James Gordon (2008), Harvard-educated psychiatrist and director of The Center for Mind–Body Medicine, who himself had a history of depression, does not regard depression to be a medical illness. He and others (e.g., Curtiss, 2001; Leventhal & Martell, 2006) see it simply as a mood in reaction to a stressor. In this sense they perceive depression as a natural and healthy response to an inability to cope with certain life circumstances. This inability to cope and the concomitant distress can manifest as what may appear to be symptoms of depression to some mental health professionals and physicians (Gordon, 2008; Hagen, 2011; Nesse, 2011), especially when the cause of the distress is not investigated. Because of DSM-IV-TR's categorical method of classification and the broader symptomatic description of depression, Horowitz and Wakefield (2007) and Moncrieff (2007a) believe some mental professionals can easily confuse normal sadness with MDD. Also, as the DSM-IV-TR diagnosis of MDD is based on the assumption that symptoms alone can designate the presence of a disorder (Pilgrim & Bentall, 1999); it allows normal responses to stressors to be mischaracterized as symptoms of depressive disorder (Parker, 2005).

This approach to diagnosis concerns Gordon (2008) and Nesse (2011) as it is based on the subjective judgment of the physician or mental health practitioner to make a diagnosis. Moreover, it poses the risk that the primary cause of the disturbance will be attributed to some somatic change and consequently the person will be treated with AD medication as the first line of intervention. Gordon and Nesse both consider this attempt to "cure" depression by methods unrelated to the real causes as inappropriate and superficial. Horowitz and Wakefield (2007) and Moncrieff (2007a) believe this confusion between depression and normal distress has important implications not only for psychiatry and its patients, but also for society in general. In the next section of the article I will explore the impact of medicalization of depression on the pharmaceutical industry.

Marketing of Antidepressant Medications

Drug treatment plays a central role in modern management of depression. Over the last 30 years AD drugs have not only become more widely prescribed, but also very familiar to the general public, largely due to aggressive marketing and advertisement by the pharmaceutical industry (e.g., Kirsch, 2010; Horowitz & Wakefield, 2007; also see Yapko, this issue). AD such as Prozac have become household names and books about them have become best sellers (e.g., Kramer, 1993). In the United Kingdom, between 1991 and 2001, the prescriptions for antidepressants rose by 173% and the cost for them increased by 700% (Department of Health, 2002). In the United States 189 million prescriptions for ADs were written in 2005 (Gordon, 2008; Smith, 2012, also see Yapko, this issue). Many of these researchers believe the massive increase in AD use could have been influenced by the intensive publicity campaigns by the drug industry (e.g., Horowitz & Wakefield, 2007; Kirsch, 2010).

In tandem with the medicalization of depression and the loosening of the DSM-III criteria for depression, many writers believe the pharmaceutical industry has cashed in (e.g., Gordon, 2008; Kirsch, 2010). Previously, Horowitz and Wakefield (2007) argued that although the DSM-III (1980) criteria for depression had become more restrictive compared to the criteria listed in DSM-II (APA, 1968), the criteria listed in the DSM-III—and we can add, even now, in DSM-IV-TR—are still too broad.

Gordon (2008) and Moncrieff (2007a) also think that the media and the popular literature have led the public to regard depression to be a medical disorder. Gordon is alarmed that the movement toward the medicalization of depression was highly lauded, although there was no solid evidence that depression is a disease. Moreover, it was very concerning to Gordon and others (e.g., Curtiss, 2001; Double, 2006; Gordon, 2008; Greenberg, 2010; Horwitz & Wakefield, 2007; Kirsch, 2010; Leventhal & Martell, 2006; Moncrieff, 2008; Yapko, 2009, also this issue) that ADs are overprescribed. It is quite likely that this movement must have influenced the public and some health professionals. Moncrieff (2007a, p. 100) states:

> We do indeed live in an age characterized by an 'epidemic of psychological disorders.' . . . However, the mass prescribing of antidepressants and the concomitant message that depression is a brain disease have helped to create this situation, not to improve it.

As nearly 80% of antidepressant prescriptions are written by physicians who are not psychiatrists (Mark, Levit, & Buck, 2009; Olfson & Marcus, 2009), Gordon (2008) and Moncrieff (2007a) believe the above movement might have indirectly influenced some physicians to prescribe ADs to any emotional condition that resembles depression.

These researchers (e.g., Bremmer, 2008; Gordon, 2008; Kirsch, 2010; Horwitz & Wakefield, 2007; Smith, 2012) are even more alarmed that, in the present climate, if a depressed person does not view his or her depression as a medical disease or refuses to take antidepressant, the person may be labelled as resistant or uncooperative. Similarly, physicians or psychiatrists who refuse to prescribe antidepressants are likely to be accused of malpractice. In 1998, Alan Leshner, then director of the National Institute on Drug Abuse, stated that "My belief is that today, in 1998, you should be put in jail if you refuse to prescribe SSRIs for depression" (from Gordon, 2008, p. 10).

Moreover, the opponents of the medicalization of depression argue that there is little evidence that ADs are more effective than a variety of other available therapeutic approaches for depression (e.g., Kirsch, 2010; Moncrieff, 2006, 2007b, 2008). Hence they have expressed concern about the role of pharmaceutical industry in promoting ADs and the chemical model of depression (e.g., Kirsch, 2010). Furthermore, they are concerned about the pressure exerted by managed care industries to promote pharmacologic treatment of depression (e.g., Double, 2006; Moncrieff, 2006, 2007b, 2008).

Proponents of ADs contend that although comparable outcomes have been reported for AD medications and evidence-based psychotherapies, ADs are the most available first-line treatments for moderate-to-severe depression (Ravindran & Kennedy, 2007b). Moreover, these proponents believe that the biological explanation of depression and the concomitant drug treatment reduce stigma of mental illness (e.g., Kramer, 1993). Castaldelli-Maia, Scomparini, Andrade, and Bhugra (2011), from their review of 32 articles on "stigma, antidepressants, and depression," they found the stigma of using an AD increases stigma of mental illness in depressives rather than reducing it and it minimizes positive expectancy of using AD medication.

From the preceding discussion it is apparent that the contemporary concept of depression and the approach to treatment is challenged. Particularly, some researchers are critical of (1) the disease theory of depression, (2) the increase in use of AD medication for treating depression, and (3) the evidence upon which their prescriptions is based. However, these writers are not saying that depressed patients should not use antidepressants. They are proposing that medications should be used rarely, for brief periods of time, and in occasional life-threatening emergencies, or when all other less potentially harmful approaches such as psychotherapy have been tried for a reasonable length of time without success. The next section reviews the effectiveness of psychotherapy with depression.

Recent Controversies About the Treatment of Depression

Most people with depression do not receive treatment. Of those who do receive treatment, about one third do not respond to the current interventions and over half of those who experience a first onset of a major depressive episode will experience further recurrences in the future (Munoz, Cuijpers, Smit, Barrera, & Leykin, 2010). Nevertheless, in the past 20 years there has been significant development and innovations in the pharmacological and psychological treatments of depression. For the present purpose, psychological treatment, namely CBT and ITP will be reviewed.

There are a variety of psychological approaches for the management of depression. CBT and ITP are chosen for review here as they represent the two most widely used evidence-based psychotherapies for depression. Moreover, they formed part of one of the largest and most carefully controlled studies to assess the effectiveness of different treatments for depression (discussed in detail below), including AD medication. CBT for depression is largely based on Beck's (1967) observation that depressed patients are preoccupied with stereotypic negative thoughts about themselves, the world, and the future. These negative thoughts or cognitive distortions lead to the formation of depressive symptoms. Beck developed CBT for depression (Beck, Rush, Shaw, & Emery, 1979) as a method for modifying these automatic thoughts. Teaching patients to recognize and examine their negative beliefs and information-processing proclivities have been found to produce relief from their symptoms and enable them to cope more effectively with life's challenges. Over 80 controlled trials have consistently demonstrated CBT to be effective with depression and it compares favorably with pharmacological treatment (Hollon & Dimidjian, 2009). CBT usually outperforms pill placebo or waiting-list control groups. However, CBT therapists with more experience and training have been shown to have superior success with depression (DeRubeis et al., 2005).

IPT was developed by Klerman and colleagues (Klerman, Weissman, Rousainville, & Chevron, 1984) and it follows a different tack to describe depression. This conceptualization traces the essence of depression in the interpersonal relationships of the depressed person. Depression is seen to result from disturbances in these roles. These disturbances may be recent (e.g., marital separation) or rooted in long-standing patterns of interactions the depressed person had with important others. The theory draws from Bowlby's (1982) attachment theory to support the hypothesis that childhood insecure attachment with care-givers sets the stage for all future disturbed relationships. IPT has been found to be an effective treatment for depression (Western & Morrison, 2001) and its effect is comparable to AD (imipramine: Beach, Jones & Franklin, 2009; Elkin et al., 1989).

Which Treatment for Depression Is More Effective?

If CBT, ITP, and ADs are all effective with depression, then which treatment is most effective? The National Institute of Mental Health Treatment of Depression Collaborative Research Program (Elkin et al., 1989), referred as TDCRP, tried to answer

this question. TDCRP is the largest multicenter comparative clinical treatment trial in psychotherapy ever conducted to date (Scott & Solovey, 2007). The study compared imipramine (AD) and clinical management with CBT, IPT, and placebo with clinical management. The results showed all three active treatments were equally effective in reducing depression at post-treatment. For example, the percentages of patients who reduced their depression below the clinical level (6 or less on the Hamilton Rating Scale for Depression) for each active condition was CBT = 36%, IPT = 43%, and AD + clinical management = 42% respectively.

The findings from the above study provide strong evidence that psychotherapy is as effective as AD in the treatment of depression. Moreover, the study demonstrated that none of the treatment—pharmacological or psychotherapeutic—fared better than the placebo (Shea et al., 1992). If they are all equally effective, including the active placebo, how can this be so? How can such apparently diverse treatments be equivalent in reducing depressive symptoms? It is even more intriguing that other dissimilar treatments such as St. John's wort (Linde, Berner, Egger, & Mulrow, 2005), L-methylfolate (Papakostas, Cassiello, & Iovieno, 2012), acupuncture (Wu, Yeung, Schnyer, Wang, & Mischoulon, 2012), transcranial magnetic stimulation (Hermann & Ebmeier, 2006), behavioral activation (Dimidjian et al., 2006), mindfulness-based cognitive therapy (Hofmann, Sawyer, Witt, & Oh, 2010; Mathew, Whitford, Kenny, & Denson, 2010), and exercise (Stathopoulou, Powers, Berry, Smits, & Otto, 2006) are all effective with depression and some of them have comparable or better effect than medication. Again, the effects of some of these therapies were comparable to the placebo group. For example, a meta-analysis of 207 clinical studies of the effectiveness of acupuncture with MDD, concluded that the "efficacy of acupuncture monotherapy was comparable to ADs [antidepressants] alone in improving clinical response and alleviating symptom severity of MDD, but the review also found that sham (control treatment) acupuncture had comparable effects on depression" (Wu et al., 2012, p. 399).

However, a recent meta-analysis of 6 studies with major depression (Fournier et al., 2010) found ADs to be effective with severe depression but comparable to a placebo with mild and moderate depression. On the other hand, Imel, Malterer, McKay, and Wampold (2008) from their meta-analysis of 29 studies comparing the effect of ADs with psychotherapy in the treatment of severe depression found psychotherapy to be equally effective with severe depression and, at long-term, psychotherapy demonstrated a prophylactic effect. Although Kirsch, Deacon, Huedo-Medina, Scoboria, Moore, and Johnson (2008) have argued that the effect of DA on severe depression could be due to the nocebo effect of the drugs, at this point based on all the evidence, it can be concluded that mild and moderate depressions respond to a variety of therapies, but not to ADs.

Common Factors in Psychotherapy

The comparable results of such diverse therapies with depression can be explained in terms of the "common factors" theory of change in psychotherapy. Common factors are

considered to be the true sources of therapeutic change and these elements are deemed to be shared by all or most forms of psychotherapy (Ingram, Hayes, & Scott, 2000). Based on decades of research, psychotherapy outcome, irrespective of theoretical orientation, can be attributed to the following four common factors (the figures represent portion of outcome variances: Lambert, 1992; Norcross & Lambert, 2011): (1) 15% expectancy or placebo effect; (2) 15% treatment techniques or strategies; (3) 30% therapeutic relationship; and (4) 40% extratherapeutic variables such as self-change, spontaneous remission, social support, and fortuitous events.

What is most impressive from these findings is that extratherapeutic factors (40%) have the greatest impact on change, while the total portion of outcome variance attributable to treatment is only 15%, which is comparable to the placebo effect (expectancy). Based on these findings, Wampold (2001) stated that the medical model, implicit in the evidence-based movement, which assumes that the outcomes of therapy originate in the specific ingredients built into a given treatment, is out of step with the facts. These findings led him to propose the *contextual model*, which upholds the importance of common factors in promoting positive change. Wampold also emphasized that there is no connection between the supposed underlying causes of psychological suffering and the treatments that effectively ameliorate it. Recently Sparks, Duncan, and Miller (2008) extended the common factors explanation to explain the effect of antidepressants with depression as they do not fare better than psychotherapy or other interventions, at least in mild and moderate depression. Kirsch, Moore, Scoboria, and Nicholls (2002) demonstrated that 82% of the drug response is due to placebo effects and only 18% of the drug response is due to the pharmacological agent in the medication. Similarly, Fisher and Greenberg (1989) in their review of the antidepressant effects of the tricyclics and the MAOIs, found a 15% improvement advantage over the placebo. It is interesting that the techniques effect (15%) of psychotherapy is almost comparable to the drug effect and the placebo effect.

The Power of Placebo

From the foregoing discussion it is evident that the power of placebo is as potent as drug or psychotherapy effect and depression seems to be highly sensitive to the placebo effect (Buck, 2012; Moncrieff, 2008; Raz, 2011; Yapko, this issue). The review by Fisher and Greenberg (1989) clearly demonstrated that in studies that made use of *active placebos*, whatever superiority had been reported for the drug effect previously over placebo disappeared. This section of the article will examine the mechanisms of the placebo response.

The TDCRP study described above also examined patient's expectancy about the treatment. Those patients who expected to feel better improved the most and those who did not expect to feel better derived least benefit from the treatment. Moreover, the expectancy effect on treatment outcome was independent of which treatment the patients

received. Those patients who expected to get better showed the most improvement, regardless of the patients treated with antidepressants, CBT, ITP, or placebo (Sotsky et al., 1991).

Placebo studies have raised awareness about the vital importance of expectancy effects in medical and psychotherapeutic interventions. There are overwhelming data attesting to the power of expectancies in medical and psychological treatments (Weinberger & Eig, 1999). Expectancy is an extremely powerful variable in human health functioning. Psychotherapy is more effective when the patient has positive expectations (Kirsch, 1985, 1990, 2010). Expectancies are critical to outcome (Weinberger & Eig, 1999). Positive expectancy can be regarded as a form of positive belief, positive self-talk, or sense of self-efficacy. Before describing strategies for maximizing expectancy effect with depression, the power of the nocebo effect is briefly described.

The Power of the Nocebo in Depression

Just as positive beliefs or positive expectancy can produce desirable outcome, expectations of sickness and symptoms may produce sickness and symptoms in the expectant individual. In parallel with the healing "placebo" effects of positive expectations, the effect of negative expectations on what is expected has been referred to as the nocebo effect (Hahn, 1999; Kennedy, 1961). Nocebo can produce a variety of physical and psychological symptoms ranging from minor discomfort to named sicknesses, disabilities, injuries, mass psychogenic illness, and even death (Hahn, 1995; 1999). There is extensive evidence to support the hypothesis that negative suggestions produce nocebo effects (negative experience or negative symptoms: see Hahn, 1999 for review).

In the context of anxiety, Schoenberger (1999) has described how "anxiety expectancy" can produce feelings of anxiety and fear and avoidance behavior. Anxiety expectancy is a belief about the occurrence of anxiety, or it is the anticipation of one's autonomic anxiety reaction to a particular situation. Several research studies have demonstrated the relationship between anxiety expectancy and experienced fear and avoidance behavior (see Schoenberger, 1999). Similarly, expectation of depression or hopelessness can lead to depressive affect (Kirsch, 2006). Schoenberger's (1999) model of anxiety expectancy can be easily extended to depression. According to the cognitive theory, emotional disorders are caused by cognitive distortions (Beck, 1967, 1976). Alladin (1994, 2007) and Araoz (1981, 1985) have labeled these dysfunctional styles of thinking *negative self-talk* or *negative self-hypnosis* (NSH).

Araoz (1981, 1985) proposed the concept of NSH to explain the perpetuation of emotional disorders in some patients. According to Araoz, NSH consists of non-conscious (automatic) rumination with negative statements and defeatist mental images that the person indulges in, encourages, and often works hard at fostering. Alladin (1992, 1994, 2006) drew the parallel between NSH and depression and conceptualized unipolar nonendogenous depression as a form of dissociation (Alladin, 1992, 1994, 2006, 2007). This model considered NSH as one of the major factors in the causation, aggravation,

and maintenance of clinical depression. Recently Alladin (2007) revised the model and replaced NSH with *rumination* as this concept incorporates over 20 years of research with depression (Nolen-Hoeksema, 2002). There is extensive evidence that negative rumination exacerbates and prolongs symptoms of depression and aggravates moderate symptoms of depression into major depressive episode (Papageorgiou & Wells, 2004). Yapko (1992, 1997) also regards the depressive affect produced by cognitive distortions to be a form of "symptomatic trance."

In summary, Beck's cognitive model provides a framework for understanding the role of beliefs and expectancy in the development of depression. In the context of expectancy, depressogenic cognitions can be regarded as nocebos for depression. Moreover, Beck and his colleagues (Beck et al., 1979) have developed cognitive therapy to help patients change their beliefs that produce their depression. There is extensive evidence in support of the effectiveness of cognitive therapy with depression (e.g., Hollon & Dimidjian, 2009; Teasdale, 2005). In other words, replacement of negative beliefs or negative expectations with realistic or positive expectations is shown to be effective in reducing depressive symptoms. The next section describes various strategies for maximizing expectancy effect in the management of depression.

Strategies for Maximizing Expectancy Effect in Psychological Treatment of Depression

From the foregoing reviews and discussion it is apparent that the definition, diagnosis, and treatments of depression are not entirely satisfactory. Many researchers (e.g., Gordon, 2008; Parker, 2005) have argued for a paradigm shift in modelling and classifying the depressive disorders. Some authors (e.g., Curtiss, 2001; Gilbert, 2007; Gordon, 2008; Leventhal & Martell, 2006) and clinicians have gone beyond that and have actually proposed alternative models of understanding and treating depression. While this trend is commendable and necessary, one should not be distracted from the fact that mental health workers and primary care physicians, especially the front-line workers, are dealing with millions of depressives on a daily basis world-wide. This article will thus focus on how to maximize the benefits of currently available psychotherapy, specifically cognitive hypnotherapy (CH), for depression.

In recent years, new research has led to considerable progress in the understanding of the mechanisms of placebo. This research reveals that the study of placebo is essentially the study of the psychosocial milieu of every treatment (Koshi & Short, 2007). Hence several researchers view the term "placebo effect" to be a misnomer because by their very definition placebos have no effect at all. According to Moerman (2006), a more accurate term would be "meaning response" because this highlights the meanings that are invested in the therapeutic context or doctor–patient relationship. Moreover, Greenberg, Constantino, and Bruce (2006) and Kirsch (1990) view psychotherapy to be inextricably linked with the manoeuvring and revision of patients' expectations.

Constantino, Glass, Arnkoff, Ametrano, and Smith (2011), Greenberg et al. (2006) and Kirsch (1990) have reviewed the extensive literature on the role of expectations in psychotherapy and they have come up with a list of recommendations for maximizing the expectancy effect in psychotherapy. Similarly, Gfeller and Gorassini (2010) and Lynn, Kirsch and Hallquist (2008) have offered several recommendations for enhancing the effect of hypnotherapy. These recommendations for maximizing the expectancy effect in psychotherapy and hypnotherapy are discussed below in the context of CH for depression. CH is an evidence-based multimodal treatment for depression, which can be applied to a wide range of patients with depression (Alladin & Alibhai, 2007). CH uses hypnosis to amplify the effect of CBT by maximizing concentration, facilitating divergent thinking and experiences, and enhancing access to unconscious processes (Alladin, 2012). CH was empirically validated by Alladin and Alibhai (2007) and the results showed an additive effect of combining hypnosis with CBT, that is, the effect size for hypnosis was larger than the effect size for CBT. The authors suggested that one of the reasons for the larger effect size in the hypnosis group was due to the placebo effect.

Strategies for Maximizing Expectancy Effect in CH for Depression

The strategies for maximizing the expectancy effect in CH discussed here are not meant to be used in the sequence as they are listed. Any of these strategies can be used when the need arises. Although the strategies are discussed in the context of CH, the treatment protocol is not detailed here as it is fully described elsewhere (see Alladin, 1994, 2006, 2007, 2008, 2009, 2010, 2012a, 2012b).

1. Assess patients' prognostic expectations at the beginning of treatment. If needed verify, validate, or modify patient's beliefs and level of optimism. If an overly optimistic patient with chronic depression asks, "How long would it take for me to feel better?" the therapist may state: "Research indicates that it takes about 15 to 20 sessions, or about 6 months, for a person with long-term depression to get better. As you appear to be very motivated to get better, it's more than likely that you will get better within this time. It may take this long because depression seems to be cyclical—even if a patient is doing well, we often notice some ups and downs during the course of treatment."
2. Conduct a careful analysis of patient's attitudes, beliefs, and expectancies about CBT and hypnosis. Clarify misunderstandings and misconceptions and emphasize the collaborative nature of the therapy.
3. Socialize the patient to a common language of communication. This can be achieved by asking the patient to read certain prescribed material. For example, after the first session of CBT, the patient is encouraged to read the first three chapters from *Feeling Good: The New Mood Therapy* (Burns, 1999). The patient is told to pay particular attention to Chapter Three, which describes 10 types of cognitive distortions, and to identify how many of these apply to the patient. In future

sessions when reference is made to, for example, "All-or-Nothing Thinking", the patient is able to recognize the characteristics of this kind of negative thinking.

4. Portray hypnosis as a cognitive experience not far removed from many everyday life phenomena: "Hypnosis is focused attention or a form of self-absorption, which we all experience, for example, when we are watching a movie." If the therapist is more oriented toward the neo-dissociative theory of hypnosis (Hilgard, 1977), words like "dissociation" or "trance" can be used, as long as these concepts are explained to the patient.
5. Discuss the concept of goal-directed fantasy or imaginative involvement and its role in hypnotic experience. In CH, a connection is made between "cognitive distortions" and "negative self-hypnosis." Indicate that: "Cognitive distortions are a form of negative self-hypnosis. The more you get involved in negative self-talk, the more depressed you become. On the other hand, the more absorbed you become in positive self-talk, the better you feel."
6. Elaborate on the importance of absorption and suspension of reality orientation for facilitating optimal hypnotic responsiveness. One way to achieve this is to induce eye or body catalepsy, which I (Alladin, 2010) routinely use in cognitive hypnotherapy:

> To ratify the credibility of hypnosis and demonstrate the power of the mind to influence the body, eye and body catalepsies can be hypnotically induced. This procedure can reduce skepticism about hypnosis, can foster positive expectancy, and can instill confidence in depressed patients that they can tap on personal resources in new ways to produce substantial behavioral and emotional changes. (p. 171)

The induction of catalepsy demonstrates to the patient that they have the ability to produce changes in their body by utilizing the power of their mind. This procedure helped Bob, a 55-year-old electronic engineer, with a 6-year history of moderate depression and social phobia, reduce his skepticism about hypnosis (see Alladin, 2006). Bob strongly believed that his anxiety and depression were biochemical disorders, inherited from his father, who suffered with anxiety and depression throughout his adult life. Bob did not show good response to ADs, but he was convinced that CH would help him, although he was skeptical of hypnosis. Five sessions of hypnosis were needed to help Bob acquire a positive image of hypnosis. Because Bob was so preoccupied with the biological cause of his anxiety and depression, it was decided to devote several sessions of hypnotherapy at the initial stage of his therapy. The sessions were devoted for ego-strengthening, positive mood induction, expansion of awareness, and demonstration of the power of his mind over his body by producing eye and body catalepsy. Following these sessions, Bob became fascinated with hypnosis and started reading books on it. He was intrigued that he could not open his eyes or get out of the chair, which reinforced his belief that he could change and strengthen his mind and body. He started to show significant improvement and indicated to me that he liked coming

to therapy and he looked forward to his "fascinating sessions." This case illustrates the unique potential of hypnosis to produce dramatic cognitive, emotional, and somatosensory changes in depressed patients.

7. Explain that hypnosis is an active process that can be learned by the patient as a coping skill. This can be facilitated by teaching patients self-hypnosis and providing a pre-recorded self-hypnosis CD for home-practice.
8. Debunk therapy-interfering myths about hypnosis (e.g., people lose control, fall asleep, reveal secrets, get stuck in it, etc.).
9. Assess for the presence of minimal suggestibility by administering easy to pass suggestions at the outset of treatment with hypnosis. It is advisable to start with relaxation suggestions first and then move on to induce somatosensory (e.g., feeling light, heavy, detached, warm, etc.) changes. If the patient is successful, then proceed to eye catalepsy, body catalepsy, and other more challenging suggestions as required to enhance the expectancy effect.
10. Select tasks that are graduated and that are likely to be accomplished by the patient. For example, in the initial stage of CBT, when prescribing behavioral activities, do not select more than five tasks to be carried out at home. These tasks should be selected in collaboration with the patient and they should be easily accomplished (e.g., shower, brush your teeth, make your own tea or coffee, peruse the daily newspaper, and watch TV for 30 minutes).
11. Structure expectations so that even small improvements are seen as steps toward achieving desired goals.
12. Adopt a permissive therapeutic stance and be prepared to bypass an agenda. Even if the agenda has been set at the beginning of a session or in previous session, the therapist should be flexible enough to be able to step back and focus on any concern that the patient may express later in the session.
13. Reinterpret failures to respond to hypnotic suggestions as successes. For example, if a patient fails to produce eye catalepsy, the therapist can indicate: "This shows that you are in control, you were able to open your eyes when you chose to do so."
14. Present a convincing rationale for therapy and label the procedures as relaxation, or imagination if the patient rejects hypnosis.
15. When using strategies to enhance outcome expectations, be modest but emphatic. Make concerted effort to use hope-inspiring statements, but be attentive of patient's belief system or sense of self and avoid promise of unrealistic degree or speed of change. In the beginning keep the statements more general, e.g., "It makes sense that you sought treatment for your depression" or "I specialize in cognitive hypnotherapy which is very effective with depression." In addition, the therapist can make statements to express confidence and competence: "I am confident that by working together we will be able to overcome your depression." However, the therapist should be conscious of the fact that the patient may not believe the therapist initially and this may have to be repeated during several sessions.

16. Personalize expectancy-enhancing statements based on patient's strengths, assets, or experiences: "You have already overcome two major obstacles. First, you have admitted to yourself that you have depression. Secondly, you sought help for it. These are not easy things to do. This indicates to me that you want to get better, although you may have questions about whether you will get better." If from the history-taking the therapist is aware of the patient's strengths in terms of achievements, the therapist can say: "You strike me as someone who has great determination. I get the impression that once you are committed to something you see it through."
17. Offer non-technical review of research findings on the intended treatment: "Research has shown that depressed people do much better with CBT or hypnosis than when they try to deal with their depression on their own."
18. Explain the occurrence of fluctuations in mood and the likelihood of small setbacks: "Depression is known to be cyclical, the mood often fluctuate, so it is important not to get discouraged when you feel down sometimes. Soon you will be able to get out of this, so it's important to continue with your therapy even if you are not feeling well at times. It's normal to have ups and downs during healing."
19. Monitor therapeutic expectancies over the course of treatment and if the needs arise modify patient's beliefs and level of optimism.

Conclusions

This article critically examined the definition, diagnosis, and treatments of depression from an "alternative" perspective. This selective review indicates that depression is (1) poorly defined, (2) often misdiagnosed or over-diagnosed, (3) overly medicalized, (4) permissively treated with drugs, and (5) it is equally (in most cases) responsive to diverse assortments of therapies and placebos. The equivalent responses of depression to assortments of treatments indicate that MDD is highly receptive to the placebo effect. This article highlighted the importance of maximizing the expectancy effect in the psychological management of depression. It is recommended that psychotherapists, based on research evidence, acknowledge that it is not the therapy techniques that produce meaningful changes in the depressed person, but it is the common factors, specifically patient's expectancy. This acknowledgement will not only be the first step in the right direction, but will also enhance both evidence-based practice and treatment outcome.

References

Alladin, A. (1992). Depression as a dissociative state. *Hypnos: Swedish Journal of Hypnosis in Psychotherapy and Psychosomatic Medicine*, *19*, 243–253.

Alladin, A. (1994). Cognitive hypnotherapy with depression. *Journal of Cognitive Psychotherapy: An International Quarterly*, *8*, 275–288.
Alladin, A. (2006). Cognitive hypnotherapy for treating depression. In R. Chapman (Ed.), *The clinical use of hypnosis with cognitive behavior therapy: A practitioner's casebook* (pp. 139–187). New York, NY: Springer Publishing Company.
Alladin, A. (2007). *Handbook of cognitive hypnotherapy for depression: An evidence-based approach*. Philadelphia, PA: Lippincott, Williams & Wilkins.
Alladin, A (2008). *Cognitive hypnotherapy: An integrated approach to treatment of emotional disorders*. Chichester, UK: John Wiley & Sons Ltd.
Alladin, A (2009). Evidence-based cognitive hypnotherapy for depression. *Contemporary Hypnosis*, 26, 245–262.
Alladin, A. (2010). Evidence-based hypnotherapy for depression. *International Journal of Clinical and Experimental Hypnosis*, *58*, 165–185.
Alladin, A. (2012a). Cognitive hypnotherapy for major depressive disorder. *American Journal of Clinical Hypnosis*, *54*, 275–293.
Alladin, A. (2012b): Cognitive hypnotherapy: A new vision and strategy for research and practice, *American Journal of Clinical Hypnosis*, *54*, 249–262.
Alladin, A., & Alibhai, A. (2007). Cognitive-hypnotherapy for depression: An empirical investigation, *International Journal of Clinical and Experimental Hypnosis*, *55*, 147–166.
American Psychiatric Association (APA). (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: Author.
American Psychiatric Association (APA). (1968). *Diagnostic and statistical manual of mental disorders* (2th ed.). Washington, DC: Author.
American Psychiatric Association (APA). (1980). *Diagnostic and statistical manual of mental disorders* (3th ed.). Washington, DC: Author.
American Psychiatric Association (APA). (1987). *Diagnostic and statistical manual of mental disorders* (3th ed., revised). Washington, DC: Author.
American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
American Psychiatric Association (APA). (2012). APA DSM-5. Retrieved from http://www.dsm5.org/proposedrevisions/pages/proposedrevi...2012-10-02e.html
Araoz, D. L. (1981). Negative self-hypnosis. *Journal of Contemporary Psychotherapy*, *12*, 45–52.
Araoz, D. L. (1985). *The new hypnosis*. New York, NY: Brunner/Mazel.
Beach, S. R. H., Jones, D. J., & Franklin, K. J. (2009). Marital, family, and interpersonal therapies for depression in adults. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed.) (pp. 624–641). New York, NY: Guilford Press.
Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects*. New York, NY: Hoeber.
Beck, A. T. (1976). *Cognitive therapy and emotional disorders*. New York, NY: International University Press.
Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.
Bowlby, J. (1982). *Attachment and loss* (2nd ed.). New York, NY: Basic Books.
Bremner, J. (2008). *Before you take that pill: Why the drug industry may be bad for your health*. New York, NY: Avery.
Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology*, *110*, 585–599.
Brown, T. A., & Leyfer, O. (2010). Classification of anxiety disorders. In D. J. Stein, E. Hollander, & B. O. Rothbaum (Eds.), *Textbook of anxiety disorders* (2nd ed., pp. 17–33). Washington, DC: American Psychiatric Publishing, Inc.

Buck, M. (2012). The placebo response in pediatric clinical trials. *Pediatric Pharmacology*, *18*, 3. Retrieved from http://www.medscape.com/viewarticle/761908_7.

Burns, D. D. (1999). *Feeling good: The new mood therapy*. New York, NY: Avon Books.

Castaldelli-Maia, J. M., Scomparini, L. B, Andrade, A. G., & Bhugra, D. (2011). *Journal of Nervous & Mental Disease*, *199*, 866–871.

Constantino, M. J., Glass, C. R., Arnkoff, D. B., Ametrano, R. M., & Smith, J. Z. (2011). Expectations. In J. C. Norcross (Ed.), *Psychotherapy relationships that work: Evidence-based responsiveness* (2nd. ed., pp. 353–376). New York, NY: Oxford University Press.

Cross-National Collaborative Group (1992). The changing rate of major depression: Cross-national comparisons. *Journal of the American Medical Association*, *268*, 3098–3105.

Curtiss, A. B. (2001). *Depression is a choice: Winning the battle without drugs*. New York, NY: Hyperion.

Department of Health (2002). *Prescription cost analysis 2001*. London, UK: Stationery Office.

DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon R. M., . . . Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry*, *62*, 409–416.

Dimidjian, S., Hollon, S. D., Dobson, K. S., Schmaling, K. B., Kohlenberg, R. J., Addis, M., . . . Dunner, D. L. (2006). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the acute treatment of adults with major depression.*Journal of Consulting and Clinical Psychology*, *74*, 658–670.

Dobson, K. S. (1985). The relationship between anxiety and depression. *Clinical Psychology Review*, *5*, 307–324.

Double, D. B. (Ed.). (2006). *Critical psychiatry: The limits of madness*. New York, NY: Palgrave Macmillan.

Dozois, D. J. A., & Westra, H. A. (2004). The nature of anxiety and depression: Implications for prevention. In D. J. A. Dozois & K. S. Dobson (Eds.), *The prevention of anxiety and depression: Theory, research, and practice* (pp. 9–41). Washington, DC: American Psychological Association.

Elkin, I., Shea, M. T., Watkins, J. T., Imber, S. D., Sotsky, S. M., Collins, J. F., . . . Parloff, M. B. (1989). National Institute of Mental Health Treatment of Depression Collaborative Research Program: General effectiveness of treatments, *Archives of General Psychiatry*, *46*, 971–982.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A. Jr., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry*, *26*, 57–63.

Fisher, S., & Greenberg, R. (Eds.). (1989). *The limits of biological treatments for psychological distress*. Englewood Cliffs, NJ: Lawrence Erlbaum.

Fombonne, E. (1994). Increased rate of depression: Update of epidemiological findings and analytical problems. *Acta Psychiatrica Scandinavica*, *90*, 145–156.

Fombonne, E. (1999). Time trends in affective disorders. In P. Cohen, C. Slomkowski, & L. Robins (Eds.), *Historical and geographical influences on psychopathology* (pp. 115–140). Mahwah, NJ: Erlbaum.

Fournier, J. C., DeRubeis, R. J., Hollon, S. D., Dimidjian, S., Amsterdam, J. D., Shelton, R. C., & Fawcett, J. (2010). Antidepressant drug effects and depression severity: A patient-level meta-analysis. *Journal of American Medical Association*, *303*, 47–53.

Frances, A., First, M. B., & Pincus, H. A. (1995). DSM-IV: Its value and limitations. *Harvard Medical Health Letter*, Vol. II, No. 12.

Gfeller, J. D., & Gorassini, D. R. (2010). Enhancing hypnotizability and treatment response. In S. J. Lynn, Rhue, J. W., & Kirsch, I. (Eds.), *Handbook of clinical hypnosis* (2nd ed., pp. 339–356). Washington, DC: American Psychological Association.

Gilbert, P. (2004). Depression: A biopsychosocial, integrative, and evolutionary approach. In M. Power (Ed.), *Mood disorders: A handbook of science and practice* (pp. 99–142). West Sussex, UK: John Wiley.

Goldberg, D., & Huxley, P. (1992). *Common mental disorders: A bio-social model*. London, UK: Routledge.

Goldstein, W. N., & Anthony, R. N. (1988). The diagnosis of depression and the DSMs. *American Journal of Psychotherapy*, *17*, 180–196).

Goodwin, R. D., Jacobi, F., Bittner, A., & Wittchen, H. (2006). Epidemiology of mood disorders. In D. J. Stein, D. J. Kuffer, & A. F. Schatzberg (Eds.), *Textbook of mood disorders* (pp. 33–54). Washington, DC: American Psychiatric Publishings, Inc.

Gordon, J. S. (2008). *Unstuck: Your guide to the seven-stage journey out of depression*. New York, NY: Penguin Group.

Greenberg, G. (2010). *Manufacturing depression: The secret history of a modern disease*. New York, NY: Simon & Schuster.

Greenberg, R. P., Constantino, M. J., & Bruce, N. (2006). Are expectations still relevant for psychotherapy process and outcome? *Clinical Psychology Review*, *26*, 657–678.

Hagen, E. H. (2011). Evolutionary theories of depression: A critical review. *Canadian Journal of Psychiatry*, *56*, 716–726.

Halgin, R. P. (2003). *Taking sides: Clashing views on controversial issues in abnormal psychology* (2nd ed.). Guilford, CT: McGraw-Hill/Dushkin.

Hahn, R. A. (1995). *Sickness and healing: An anthropological perspective*. New Haven, CT: Yale University Press.

Hahn, R. A. (1999). Expectations of sickness: Concept and evidence of the nocebo phenomenon. In I. Kirsch (Ed.), *How expectancies shape experience* (pp. 333–356). Washington, DC: American Psychological Association.

Herrmann, L. L., & Ebmeier, K. P. (2006). Factors modifying the efficacy of transcranial magnetic stimulation in the treatment of depression: A review. *Journal of Clinical Psychiatry*, *67*, 1870–1876.

Hilgard, E. R. (1977). Divided consciousness: Multiple controls in human thought and action. New York, NY: John Wiley & Sons.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., Oh, D. (2010). The effect of mindfulness-Based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *78*, 169–183.

Hollon, S. D., & Dimidjian, S. (2009). Cognitive and behavioral treatment of depression. In I. H. Gotlib & C. L. Hamen (Eds.), *Handbook of depression* (2nd. ed., pp. 586–603). New York, NY: Guilford Press.

Horwitz, A. V., & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder*. New York, NY: Oxford University Press.

Huppert, J. D. (2009). Anxiety disorders and depression comorbidity. In M. M. Anthony & M. B. Stein (Eds.) *Oxford Handbook of anxiety and related disorders* (pp. 576–586). New York, NY: Oxford University Press.

Imel, Z. E., Malterer, M. B., McKay, K. M., & Wampold, B. E. (2008). A meta-analysis of psychotherapy and medication in unipolar depression and dysthymia. *Journal of Affective Disorders*, *110*, 197–2006.

Ingram, R. E., Hayes, A., & Scott, W. (2000). Empirically supported treatments: A critical analysis. In C. R. Snyder & R. E. Ingram (Eds.), *Handbook of psychological change: Psychotherapy processes & practices for the 21st century*. (pp. 40–60). New York, NY: John Wiley & Sons.

Kennedy, W. P. (1961). The nocebo reaction. *Medical World*, *91*, 203–205.

Kessler, R. C., Avenevoli, S., & Merikangas, K. R. (2001). Mood disorders in children and adolescents: An epidemiologic perspective. *Biological Psychiatry*, *49*, 1002–1014.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., . . . Wang, P. S. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *Journal of American Medical Association*, *289*, 3095–3105.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 593–602.

Kessler, R. C., & Wang, P. S. (2009). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression*. (2nd ed., pp. 5–22). New York, NY: Guilford Press.

Kirsch, I. (1985). Response expectancy as a determinant of experience and behavior. *American Psychologist*, *40*, 1189–1202.

Kirsch, I. (1990). *How expectancies shape experience*. Washington, DC: American Psychological Association.

Kirsch, I. (2006). Medication and suggestion in the treatment of depression. In M. D. Yapko (Ed.), *Hypnosis and treating depression: Applications in clinical practice* (pp. 271–280). New York, NY: Routledge.

Kirsch, I. (2010). *The emperor's new drugs: Exploding the antidepressant myth*. New York, NY Basic Books.

Kirsch, I., Deacon, B. J., Huedo-Medina, T. B., Scoboria, A., Moore, T. J., & Johnson, B. T. (2008). Initial severity and antidepressant benefits: A meta-analysis of data submitted to the Food and Drug Administration. *PLoS Medicine*. Retrieved from http://medicine.plosjournals.org/perlserv/request=get.document&doi=10.1371/journal.pmed.0050045

Kirsch, I., Moore, T. J., Scoboria, A., & Nicholls, S. S. (2002). The emperor's new drugs: An analysis of antidepressant medication data submitted to the U.S. Food and Drug Administration. *Prevention and Treatment*, 5. Retrieved December 3, 2005, from http://www.journals.apa.org/prevention/volume5/pre0050023a.html.

Klerman, G. L., Weissman, M. M., Rounsaville, B., & Chevron, E. (1984). *Interpersonal Psychotherapy of Depression*. New York, NY: Basic Books.

Koshi, E. B., & Short, C. A. (2007). Placebo theory and its implications for research and clinical practice: A review of the recent literature. *Pain Practice*, *7*, 4–20.

Kramer, P. (1993). *Listening to Prozac: A psychiatrist explores antidepressant drugs and the remaking of the self*. New York, NY: Viking.

Kramer, P. (2005). *Against depression*. New York, NY: Viking.

Lambert, M. J. (1992). Psychotherapy outcome research: Implications for integrative and eclectic therapists. In J. C. Norcross & M. R. Goldfried (Eds.), *Handbook of psychotherapy integration* (pp. 94–129). New York, NY: Basic Books.

Leventhal, A. M., & Martell, C. R. (2006). *The myth of depression as disease: Limitations and alternatives to drug treatment*. Westport, CT: Praeger.

Linde, K., Berner, M., Egger, M., & Mulrow, C. (2005). St John's wort for depression: Meta-analysis of randomised controlled trials. *British Journal of Psychiatry*, *186*, 99–107.

Lynn, S. J., Kirsch, I., & Hallquist, M. N. (2008). Social cognitive theories of hypnosis. In M. R. Nash & A. J. Barnier (Eds.), *The Oxford handbook of hypnosis: Theory, research and practice*, (pp. 111–139). Oxford, UK: Oxford University Press.

Mark, T., Levit, K., & Buck, J. (2009). Datapoint: Psychotropic drug prescriptions by medical specialty. *Psychiatric Services*, *60*, 1167.

Mathew, K. L., Whitford, H. S., Kenny, M. A., & Denson, L. A. (2010). The long-term effects of mindfulness-based cognitive therapy as a relapse prevention treatment for major depressive disorder. *Behavioural Cognitive Psychotherapy, 38*, 561–576.

Mayes, R. & Horwitz, A. V. (2005). DSM-III and the revolution in the classification of mental illness. *Journal of History of Behavioral Science*, *41*, 249–67.

McCommon, B. (2006). Antipsychiatry and the gay rights movement. *Psychiatric Services*, *57*, 1809.

Moerman, D. E. (2006). The meaning response: Thinking about placebos. *Pain Practice*, *6*, 233–236.

Moncrieff, J. (2006). The politics of psychiatric drug treatment. In D. B. Double (Ed.), *Critical psychiatry: The limits of madness* (pp. 115–132). New York, NY: Palgrave Macmillan.

Moncrieff, J. (2007a). Rebuttal: Depression is not a brain disease. *Canadian Journal of Psychiatry*, *52*, 100–101.

Moncrieff, J. (2007b). Are antidepressants as effective as claimed? No, they are not effective at all. *Canadian Journal of Psychiatry*, *52*, 96–97.

Moncrieff, J. (2008). *The myth of the chemical cure*. Basingstoke, UK: Palgrave Macmillan.

Muñoz, R. F., Cuijpers, P., Smit, F., Barrera, A. Z., & Leykin, Y. (2010). Prevention of major depression. *Annual Review of Clinical Psychology*, *6*, 181–212.

Nesse, R. M. (2011). Why has natural selection left us so vulnerable to anxiety and mood disorders? *Canadian Journal of Psychiatry*, *56*, 705–706.

Nesse, R. M., & Stein, D. J. (2012). Towards a genuinely medical model for psychiatric nosology. *BMC Medicine*, *10*, doi:10.1186/1741-7015-10-5

Nolen-Hoeksema, S (2002). Gender differences in depression. In I. H. Gotlib & C. C. Hammen (Eds.), *Handbook of Depression* (pp. 492–509). New York, NY: Guilford Press.

Norcross, J. C., & Lambert, M. J. (2011). Evidence-based therapy relationships. In J. C. Norcross (Ed.), *Psychotherapy relationships that work* (2nd ed., pp.3–21). New York, NY: Oxford University Press.

Olfson, M., & Marcus, S. (2009). National patterns in antidepressant medication treatment. *Archives of General Psychiatry*, *66*, 848–856.

Papageorgiou, C., & Wells, A. (2004). *Depressive rumination: Nature, theory and treatment*. Chichester, UK: John Wiley & Sons.

Papakostas, G. I., Cassiello, C. F., & Iovieno, N. (2012). Folates and s-adenosylmethione for major depressive-disorder. *Canadian Journal of Psychiatry*, *57*, 406–413.

Parker, G. (2005). Beyond depression. *Psychological Medicine*, *35*, 467–474.

Pilgrim, D., & Bentall, R. (1999). The medicalisation of misery: A critical realist analysis of the concept of depression. *Journal of Mental Health*, *8*, 261–274.

Ravindran, A. V., & Kennedy, S. H. (2007a). In debate: Response to Dr. Moncrieff. *Canadian Journal of Psychiatry*, *52*, 102.

Ravindran, A. V., & Kennedy, S. H. (2007b). Are antidepressants as effective as claimed? Yes, but . . . *Canadian Journal of Psychiatry*, *52*, 98–99.

Raz, A. (2011). Placebo science: Research you can believe in. *Journal of Mind-Body Regulation*, *1*, 41–43.

Rissmiller, D. J., & Rissmiller, J. (2006). Letter in reply. *Psychiatric Services*, *57*, 1809–1810.

Rudolph, K. D., Hammen, C., & Daley, S. E. (2006). Mood disorders. In D. A. Wolf & E. J. Mash (Eds.), *Behavioral and emotional disorders in Adolescents: Nature assessments, and treatment* (pp. 300–342). New York, NY: Guilford Press.

Schoenberger, N. E. (1999). Expectancy and fear. In I. Kirsch (Ed.), *How expectancies shape experience* (pp. 125–144). Washington, DC: American Psychological Association.

Scott, F. J., & Solovey, A. D. (2007). *Second order change in psychotherapy: The golden thread that unifies effective treatments*. Washington, DC: American Psychological Association.

Shea, M. T., Elkin, I., Imber, S. D., Sotsky, S. M., Watkins, J. T., Collins, J. F., . . . Dolan, R. T. (1992). Course of depressive symptoms over follow-up: Findings from the National Institute of Mental Health Treatment of depression collaborative research program. *Archives of General Psychaitry*, *49*, 782–787.

Smith, B. (2012). Inappropriate prescribing. *Monitor on Psychology*, *43*, 36–40.

Sotsky, S. M., Glass, D. R., Shea, M. T., Pilkonis, P. A., Collins, J. F., & Elkin, I, . . . Oliveri, M. E. (1991). Patient predictors of response to psychotherapy and pharmacotherapy: Findings in the NIMH Treatment of Depression collaborative research program. *American Journal of Psychiatry*, *148*, 997–1008.

Sparks, J. A., Duncan, B. L., & Miller, S. D. (2008). Common factors in psychotherapy. In J. L. Lebow (Ed.), *Twenty-first century psychotherapies* (pp. 483–497). Hoboken, NJ: Wiley.

Spitzer, R. L. (1981). The diagnostic status of homosexuality in DSM-III: A reformulation of the issues. *American Journal of Psychiatry*, *138*, 210–215.

Spitzer, R., Endicott, J., & Robins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Archives of General Psychiatry*, *35*, 773–782.

Stathopoulou, G., Powers, M. B., Berry, A. C., Smits, J. A., & Otto, M. W. (2006). Exercise interventions for mental health: A qualitative review. *Clinical Psychology: Science and Practice*, *13*, 179–193.

Teasdale, J. D. (2005). Multi-level theories of cognition-emotion relations. In T. Dalgleish & M. J. Power (Ed.), *Handbook of Cognition and Emotion* (pp. 665–681). New York, NY: Wiley.

Tyrer, P. (1989). *Classification of neurosis*. Chichester, UK: Wiley.
Wampold, B. E. (2001). *The great psychotherapy debate: Models, methods, and findings*. Mahway, NJ: Erlbaum.
Wang, P. S., & Kessler, R. C. (2006). Global burden of mood disorders. In D. J. Stein, D. J. Kuffer, & A. F. Schatzberg (Eds.), *Textbook of mood disorders* (pp. 55–67). Washington, DC: American Psychiatric Publishings, Inc.
Western, D., & Morrison, K. (2001). A multidimensional meta-analysis of treatments for depression, panic, and generalized anxiety disorder: An empirical examination of the status of empirically supported therapies. *Journal of Consulting and Clinical Psychology*, *69*, 875–899.
Weinberger, J., & Eig, A. (1999). Expectancies in psychotherapy: The ignored common factor. In I. Kirsch (Ed.), *How expectancies shape experience* (pp. 357–382). Washington, DC: American Psychological Association.
Weissman, M. M., Bruce, L. M., Leaf, P. J., Florio, L. P., & Holzer, C. (1991). Affective disorders. In L. N. Robins & D. A. Regier (Eds.), *Psychiatric disorders in America: The epidemiologic catchment area study* (pp. 53–80). New York, NY: Free Press.
World Health Organization (2002). *The world health report 2002: Reducing risks, promoting healthy life*. Geneva: Author.
World Health Organization (1993). *International Classification of Diseases* (10th ed.). Geneva: Author.
Wu, J., Yeung, A. S., Schnyer, R.,Wang, Y., & Mischoulon, D. (2012). Acupuncture for depression: A review of clinical applications. *Canadian Journal of Psychiatry*, *57*, 397–405.
Yapko, M. D. (1992). *Hypnosis and the treatment of depressions: Strategies for change*. New York, NY: Brunner/Mazel.
Yapko, M. (1997). *Breaking the patterns of depression*. New York, NY: Random House/Doubleday.
Yapko M. (2009). *Depression is contagious: How the most common mood disorder is spreading around the world and how to stop it*. New York, NY: Free Press.
Yapko, M. D. (this issue). Treating depression with antidepressants: Drug-placebo efficacy debates limit broader considerations. *American Journal of Clinical Hypnosis*, *55*, 272–290.