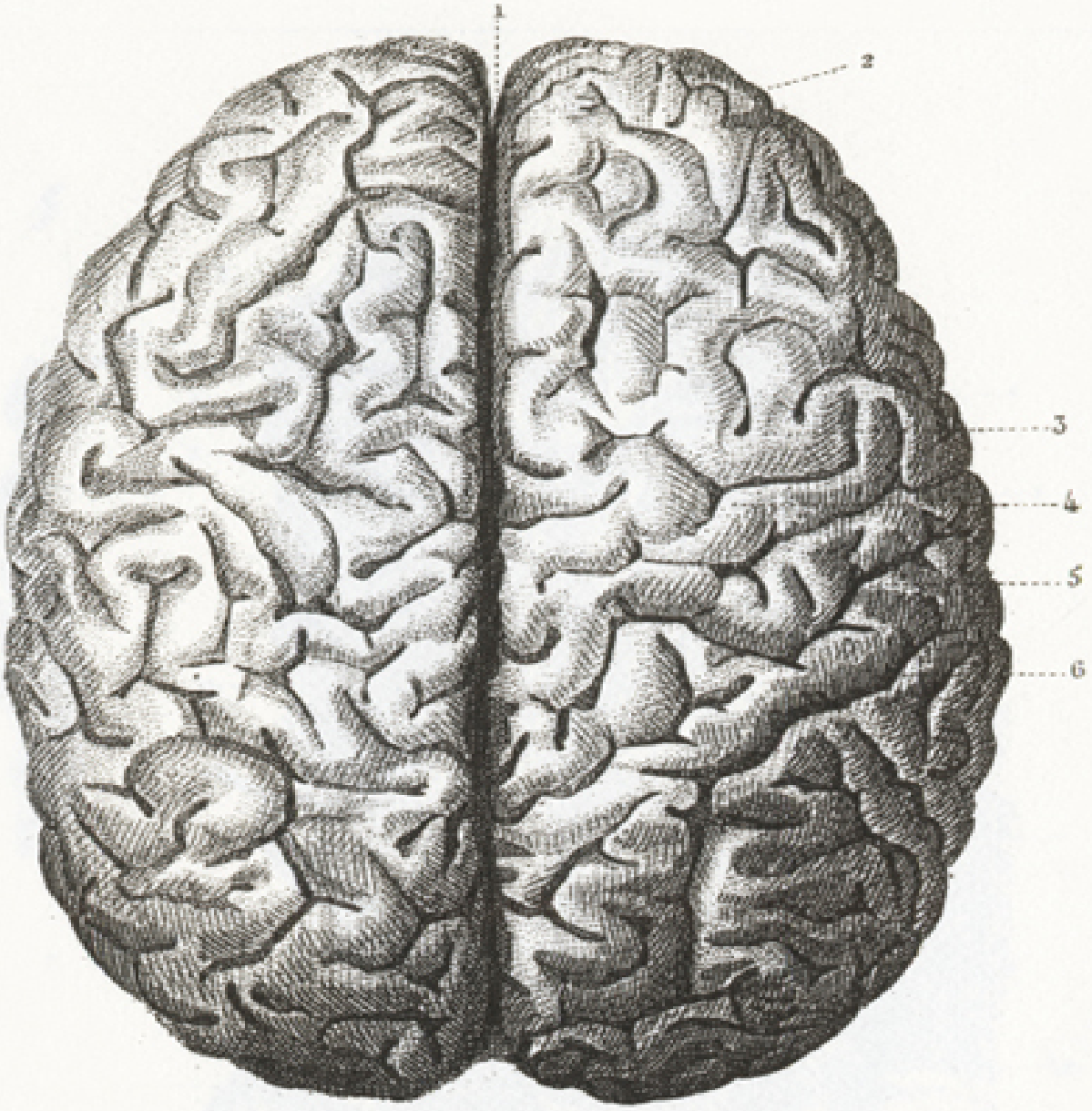
Treating Depression

MCT, CBT and Third Wave Therapies



Edited by Adrian Wells & Peter Fisher

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Preface

Depression is a very common psychological disorder, which affects over 120 million people worldwide. Approximately 10–15% of the population will be affected by depression during their lifetime. The personal, social, and economic burden of depression is profound, and depression is estimated to be the leading cause of disability worldwide. Fortunately there are effective psychological therapies for depression. One of the most studied interventions is cognitive behavioural therapy (CBT), which consists of cognitive and behavioural strategies. These two components of CBT, namely cognitive therapy (CT) and behavioural activation (BA), are equally effective. The high volume of empirical support for these approaches has led to their recommendation in healthcare guidelines as a first-line psychological treatment for depression. The pursuance of newer and alternative treatments has emerged in the last twenty years as a result of an increasing recognition of the limitations of CBT. Approximately half of the patients treated with CBT and behavioural methods recover from depression; this leaves the other half – a significant number of patients – with a partial response or with none at all. Furthermore, amongst those that do recover, there is a substantial rate of relapse that cannot be ignored. Only one third of the patients remain depression-free one year after the completion of psychological interventions.

It is evident that we need to add to the armoury of treatment approaches, with a view to providing a larger choice for both clinicians and patients. Moreover, we must find treatments that are more effective in the immediate term, and especially in the longer term. Fortunately the area has moved forward thanks to an influx of ideas and

new techniques from a range of backgrounds. This progress has led to the development of metacognitive therapy (MCT), mindfulness-based cognitive therapy (MBCT), and acceptance and commitment therapy (ACT). These approaches are based on different theoretical models of the causes and maintenance of depression. But we do not yet know whether these newer additions can be more effective than CBT or BA, and we are in danger of not being able to find out, because there is ambiguity concerning the distinctive features of these approaches.

Unfortunately the boundaries and integrity of these newer approaches are not always maintained. In clinical settings, we find that therapists have a tendency to draw on techniques and principles from any combination of CBT and these other approaches, often violating some of the basic principles of any one of the models. Why should this be of concern? Two reasons predominate: first, techniques drawn from different approaches are not always compatible and, when used together, may cancel each other out, annul the desired effects on causal mechanisms, and lead to reduced rather than improved efficacy. Second, when techniques are combined, we move away from the well-controlled evaluation of established, *bona fide* approaches that can be found in treatment manuals, which prevents the assessment of the absolute and relative efficacy of the newer therapies.

If psychological treatments for depression are to be delivered competently and with a high level of treatment fidelity, it is necessary for practitioners to recognize and understand the fundamental differences between, and distinctive features of, each approach. However, the difficulty of acquiring this level of knowledge is often underestimated, as some of the constructs contained in the therapeutic approaches sound similar but vary in the degree of conceptual refinement and specificity.

The present volume was conceived with the aim of addressing these above issues. First, we wanted to bring together in one place evidence-based, effective, and emerging approaches to treating depression. This would serve as a reference treatment manual for therapists and researchers. Second, we aimed to provide a vehicle for showcasing the important conceptual and practical differences that exist between these treatments. Third, we devised a format for the contributions that would allow scrutiny of the goodness of fit between psychological theory and the implementation of the different approaches.

Consequently the volume is divided into three sections. Section 1 comprises four chapters, which provide an overview of the nature of major depressive disorder, the clinical assessment of depression, a review of the effectiveness of CBT, and an account of the psychobiological processes and therapies of depression. Section 2 presents the theoretical foundations of the five psychological interventions and concludes with a critique of the five theories that highlights the similarities and differences between the models on which treatment is based. For Section 3, we have provided the proponents of each approach with a hypothetical case study (which constitutes the Introduction to this section). In response, they have produced a brief treatment manual to illustrate each stage of treatment – assessment, case formulation, and treatment methods – with reference to this same case study. The section concludes with a critique of the distinctive components of each treatment.

Treating Depression has been made possible by its distinguished participants, and we express our gratitude to them for sharing their knowledge and skills. Without their significant contributions we would not be standing on the edge of the next chapter, heralding our quest to resolve this state of significant human suffering.

Adrian Wells, PhD
Peter L. Fisher, PhD

Section 1
Assessment, Prevalence, and Treatment Outcomes

1
The Nature of Depression

Martin Connor, Adrian Wells, and Peter L. Fisher

Introduction

Sadness and despair are common experiences for many people, historically based descriptions reflecting the cultural context. Historical accounts indicate that the cause of severe mood disturbance was attributable to a physical illness for which the sufferer bore no responsibility. Symptoms of severe mood disturbance or melancholia included extreme sadness, an inability to function, and the frequent presence of delusions (Daly, 2007). Melancholia was thought to be caused by an imbalance of the 'bodily humours' (Daly, 2007; Akiskal & Akiskal, 2007). Conversely, accounts of less severe mood problems implied that the sufferer was ultimately responsible. In early Christian monastic settings a constellation of undesirable feelings and behaviours that interfered with devotional duties was known as the 'sin' of acedia (Jackson, 1981). This state was attributed to laziness or a 'lack of care' and was characterized by apathy, loss of hope, drowsiness, and a desire to flee the monastery (LaMothe, 2007). However, acedia was not considered equivalent to normal sadness, since the fourth-century monk John Cassian described it as a 'dangerous foe' that was 'akin to sadness' (Daly, 2007, p. 34). These historical descriptions of the 'symptoms' of melancholia and acedia loosely correspond to those of major depression as defined in modern diagnostic systems, which will be discussed in the next section.

Diagnosing Major Depressive Disorder

Major depression is a common but clinically heterogeneous disorder that is frequently comorbid with others. Current diagnostic methods rely on identifying constellations of psychological and behavioural symptoms through structured clinical interviews (see chapter 2 for a detailed account of assessment measures and processes). Major depressive disorder (MDD) is diagnosed according to either the current (fifth) edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V; APA, 2013) or the World Health Organization's International Classification of Diseases (ICD-10; WHO, 1993). Because major depression is a highly recurrent disorder (Boland & Keller, 2008), both systems operationalize it in terms of the occurrence of a single 'depressive episode' (WHO, 1992), also known as a 'major depressive episode' (MDE) (APA, 2013). The diagnostic criteria for a depressive episode are similar in both systems. Both DSM-V and ICD-10 define recurrent depression as the occurrence of two or more episodes that are separated by at least two months during which the criteria for a depressive episode are not met (APA, 2013; WHO, 1993). In DSM-V the term 'major depressive disorder' (MDD) is used to denote the occurrence of one or more major depressive episodes.

Major depression is a clinically heterogeneous disorder (Rush, 2007). The diagnostic criteria are designed to account for such heterogeneity, which means that depressed individuals with markedly divergent symptoms are assigned to the same diagnostic category (APA, 2013; Krueger, Watson, & Barlow, 2005). For example, two individuals diagnosed with a major depressive episode may both experience depressed mood and concentration

difficulties. However, one of them may have the accompanying symptoms of significant weight loss and insomnia, while the other may experience significant weight gain and hypersomnia. These differences may be important for the selection of appropriate treatment, and prognosis (APA, 2013; WHO, 1992; Rush, 2007), and therefore DSM-V enables the specification of depressive subtypes and of episode severity (APA, 2013).

Diagnostic Criteria for Major Depressive Disorder

The diagnosis of a major depressive episode requires that at least five of the symptoms listed in Table 1.1 are met for a period of at least two weeks. Importantly, one of the symptoms must be either a depressed mood or a loss of pleasure/interest in everyday activities. It is also necessary that the symptoms reach clinically significant levels, which typically compromise occupational and social functioning.

Table 1.1 Summary of DSM-V criteria for an episode of major depression.

1	depressed mood most of the time
2	loss of interest/pleasure in everyday activities
3	weight loss or weight gain, often accompanied by a reduced or increased appetite
4	sleep difficulties: sleeping too much or minimally
5	psychomotor agitation or retardation
6	tiredness, feeling fatigued, lacking energy
7	feelings of worthlessness or guilt
8	poor concentration, difficulty in making decisions
9	frequent thoughts of death, including thoughts and plans of suicide or suicide attempts

A closer inspection of the nine main symptoms of depression in Table 1.1 shows that individuals meeting diagnostic criteria for a depressive episode may have minimal overlapping symptoms. Nevertheless, researchers and clinicians have observed what appears to be relatively consistent constellations of depressive symptoms that may respond differently to treatment (Rush, 2007). Consequently, successive revisions of the DSM since version III have included specifiers that enable potentially important clinical characteristics of episodes to be recorded (APA, 2013). These episode specifiers relate to symptom severity, remission status, chronicity, and symptomatic features that may denote depressive subtypes.

The Epidemiology of Major Depression

Surveys of the prevalence of psychiatric disorders have been undertaken since the Second World War. However, estimates of prevalence varied widely, due to differences in methodology. Early estimates of the prevalence of MDD were derived from screening instruments that were not fit for purpose (Kessler et al., 2007). There were two main problems; (1) the screening instruments were prone to poor specificity or sensitivity (or both), which undermined confidence in the resultant prevalence estimates; and (2) the use of different instruments between surveys hindered the interpretation of results. This has become less of an issue since the World Health Organization commissioned the Composite International Diagnostic Interview (CIDI) in the 1980s (Kessler & Ustun, 2004) in order to compare psychiatric prevalence rates between countries according to standardized criteria (Kessler et al., 2007). The CIDI was based on the Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1981) and was designed to be administered by lay interviewers. It was also designed to support psychiatric diagnoses according to both ICD and DSM criteria. However, the original version of the CIDI was not designed to capture detailed demographic and clinical data. This meant that countries could only be broadly compared, in terms of overall prevalence rates (Kessler & Ustun, 2004).

The CIDI (version 3) was designed for the World Mental Health Survey Initiative (WMHS) (Kessler, 1999) for the purpose of facilitating the acquisition and comparison of psychiatric epidemiological data within the participating countries (Kessler & Ustun, 2004). In addition to enabling the quantification of lifetime and 12-month diagnoses according to both DSM-IV and ICD-10 criteria, the CIDI-3 also includes items that assess severity, demographic, quality-of-life, and disability data (Kessler & Ustun, 2004). Unlike previous versions, the CIDI-3 included interview

probe questions that increase the reliability of autobiographical recall. The methodological rigour used to produce different translations of the CIDI-3 has led to its being described as ‘state of the art’ for comparing epidemiological findings across participating WMHS countries (Alonso & Lepine, 2007). Two large-scale surveys within the WMHS framework have specifically examined the epidemiology of MDD. These are the European Study of the Epidemiology of Mental Disorders (ESEMeD) (Alonso et al., 2002) and the American National Comorbidity Survey Replication Study (NCS-R) (Kessler et al., 2003).

Overall prevalence rates

The NCS-R and ESEMeD surveys estimated that the 12-month prevalence of MDD according to DSM-IV criteria is 6.6 per cent in American adults and 4.1 per cent in European adults (Alonso et al., 2004; Kessler et al., 2003). In absolute terms, these results indicate that at least 13.1 million US adults experienced a major depressive episode in the preceding year (Kessler et al., 2003). In terms of lifetime rates, 16.2 per cent of Americans and 13.4 per cent of Europeans will experience at least one depressive episode.

In terms of DSM-IV symptomatology, the NCS-R results estimated that 10 per cent of the people identified within the 12-month prevalence time frame were mild, 39 per cent moderate, 38 per cent severe, and 13 per cent very severe according to the Quick Inventory of Depressive Symptomatology Self-Report (QIDS-SR) (Rush et al., 2003; Kessler et al., 2003). Thus, 51 per cent of people were classified as having severe or very severe clinical symptoms in the NCS-R sample, underscoring the significance of major depression as a major public health issue.

Prevalence rates by age and country

The ESEMeD study found that the 12-month prevalence for any psychiatric disorder is highest in the 18- to 24-year age group and lowest for individuals over 65 (Alonso & Lepine, 2007). Comparable results for the prevalence of MDE were found in the NCS-R, where 12-month and lifetime rates in the youngest cohort (18 to 29 years) were significantly higher than in those over 60 years (Kessler et al., 2003). However, the differences between age cohorts may be a function of hierarchical exclusion rules, which typically prohibit a diagnosis of MDE when there is physical comorbidity. The lower 12-month prevalence rate for older cohorts in the NCS-R may be artefactual, as higher levels of physical comorbidity in older adults may have precluded the diagnosis of a depressive episode (Kessler et al., 2010). To investigate this possibility, Kessler et al. (2010) re-analysed the WMHS data by omitting the hierarchical and organic exclusion rules that allowed depression comorbid with a physical disorder to be included. The results indicated that higher rates of physical comorbidity were not responsible for the lower rates of depression typically observed in older cohorts in developed countries (Kessler et al., 2010). An analysis across all the developed countries within the WMHS showed that the 12-month MDE prevalence was significantly lower for the oldest cohort than for the youngest cohort (Kessler et al., 2010). However, episode duration may increase with age. In developed countries, the mean episode in the youngest cohort lasted 25 weeks, by comparison to 31 weeks in the oldest cohort (Kessler et al., 2010).

Gender and prevalence of MDD

One of the most consistent epidemiological findings concerning MDD is that female prevalence rates are typically twice those registered in males (Boughton & Street, 2007). Both the ESEMeD and the NCS-R study

found that 12-month and lifetime MDD prevalence rates for females were approximately twice those for males. Higher female prevalence is known to emerge in adolescence and to continue into adulthood (Boughton & Street, 2007), although no significant gender differences have been found in terms of recurrence or chronicity (Kessler, Mcgonagle, Swartz, Blazer, & Nelson, 1993). However, the results of the United Kingdom's National Survey of Psychiatric Morbidity (NSPB) (Bebbington et al., 2003) have shown that the preponderance of female depression disappears after the age of 55, when there is a reduction in the prevalence of female depression. Boughton and Street (2007) reviewed numerous non-biological theories that have been proposed to explain the higher rates of depression seen in females. Some theories venture that higher levels of neuroticism or dependency in females increase the risk for depression, while others attribute differences to social restrictions imposed by the female role. Alternatively, the construct of major depression may itself be biased towards identifying disorder in females (Boughton & Street, 2007).

Many factors are likely to contribute to gender differences in the prevalence of depression, and there is increasing evidence that gender differences concerning emotional regulation are a key factor (Nolen-Hoeksema, 2012). Emotional regulation refers to activities that enable the individual to modify the nature of an emotional response (e.g. distraction: Nolen-Hoeksema, 2012). However, while females have been shown to employ a wider range of emotional regulatory behaviours than men (Tamres, Janicki, & Helgeson, 2002), it has been proposed that their greater tendency to ruminate on the causes and meaning of negative emotions places a higher proportion of them at risk of developing depression (Nolen-Hoeksema, 2012). Evidence that greater rumination in females may explain

their higher risk for MDD has been provided in studies that show rumination to be predictive of higher depression scores (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Aldao, 2011; Nolen-Hoeksema, Mcbride, & Larson, 1997).

Comorbidity

Major depressive disorder is highly comorbid with psychological (Rush et al., 2005) and somatic disorders (Schmitz, Wang, Malla, & Lesage, 2007). In the NCS-R study, 64 per cent of the 12-month MDD cases also met diagnostic criteria for another DSM-IV 12-month disorder (Kessler et al., 2003). However, while MDD was highly comorbid with other psychological disorders, it only preceded other 12-month disorders in 12.6 per cent of cases (Kessler et al., 2003). MDD is often comorbid with physical disorders ranging from 5 per cent to 10 per cent in primary-care settings, and from 8 per cent to 15 per cent in medical inpatient settings (Schmitz et al., 2007). Comorbid depression is associated with greater levels of disability and poorer prognosis for both psychological and physical disorders (Rush et al., 2005; Schmitz et al., 2007).

Where depression is comorbid with a physical disorder, the greatest impairments are found in those who experience chronic physical problems. The Canadian Community & Health Survey (Schmitz et al., 2007) revealed that the prevalence of functional disability in the two weeks prior to interview was significantly higher in respondents with chronic physical disorders and comorbid MDD (46 per cent) than in those with only chronic physical disorders (21 per cent) or only MDD (27.8 per cent). One of the most striking findings about the effect of comorbid depression and physical illness concerns cardiac mortality. In patients hospitalized for myocardial infarction, Lesperance, Frasure-Smith, Talajic, and Bourassa (2002) found a direct dose–response relationship between depressive

symptomatology on the Beck Depression Inventory (BDI) (Beck, Steer, & Brown, 1996) and the risk of cardiac mortality during a 5-year follow-up. Notably, the mortality rate in patients who scored 19 or more on the BDI was significantly higher than in those who scored less than 19 on the BDI – after controlling for cardiac disease severity (Lesperance et al., 2002). These results suggest that comorbid depression is associated with increased mortality during recovery from myocardial infarction.

Where another psychological disorder is comorbid with MDD, episodes of illness are typically more severe and last longer (Rush et al., 2005). As described earlier, there is evidence that comorbid dysthymia increases the duration of depressive episodes (Spijker et al., 2002). However, results from the naturalistic CDS study also indicated that comorbid panic (Coryell et al., 1988) or alcohol abuse (Mueller et al., 1994) reduce the likelihood of recovery from an MDE. Coryell et al. (1988) found that comorbid panic and MDD predicted significantly lower levels of recovery than non-comorbid cases (75 per cent versus 86 per cent respectively) over two years, while Mueller et al. (1994) found that comorbid alcoholism reduced the likelihood of recovery by 50 per cent over an observation period of ten years. However, neither of these two studies controlled for treatment differences in their analyses; but they provide evidence that comorbidity serves to increase episode duration and suggests that treatment efficacy will be lower in patients with comorbid conditions.

The moderating effect of comorbidity on treatment outcome has received relatively little attention (Carter et al., 2012; Hamilton & Dobson, 2002). However, there is consistent evidence that elevated anxiety symptomatology during an episode predicts poorer response to medication (Carter et al., 2012) and a lower probability of successful outcome following psychotherapy (Hamilton & Dobson,