Depression

Causes and Treatment

Second Edition

Aaron T. Beck, M.D., and Brad A. Alford, Ph.D.
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Preface to the Second Edition

The first edition of this book posed the question, “What has definitely been established regarding the nature, the causes, and the treatment of depression?” To answer it, Aaron Beck sifted through thousands of clinical and controlled studies and summarized representative research on the clinical, biological, psychological, and theoretical aspects of depression. Of greater significance, he described an original research program that, in retrospect, represented a breakthrough in understanding the cognitive components and treatment of depression.

Like the first edition, this one presents an update and overview of what is currently known about clinical depression, including developments that have taken place since the book was originally published 40 years ago and, also like that earlier volume, offers a historical perspective. Moreover, in Chapter 16 we review the randomized controlled trials that have built upon and elaborated cognitive theory and research.

What is new to the second edition? Definitions of the mood disorders have changed over the years, and new categories have been added. We now recognize major depression as the leading cause of disability worldwide, and it has received increased clinical and research attention. In the years since the book was first published additional types of bipolar disorder have been recognized, and research has been conducted on the relation between manic symptoms and life events. New drugs, such as the selective serotonin reuptake inhibitors, or SSRIs, have been developed. While comparable in efficacy (except in severe depression), they are chemically unrelated to tricyclic, heterocyclic, and other antidepressants discussed in the first edition, and they enjoy several advantages over those “first-generation” drugs. The newer medications can induce fewer adverse side effects and provide greater safety in case of overdose and improved tolerability and patient compliance. SSRIs may also be augmented with lithium, psychostimulants, and other agents.

There are even now many unresolved problems in pharmacotherapy. Drug treatment of depression—even using the newer SSRIs—still results in unwanted side effects, such as the sexual dysfunction that affects 60 percent of patients. There are potential lethal interactions between SSRI and MAOI drugs. Other unintended effects include gastrointestinal disturbance, nausea, and somnolence. Electroconvulsive therapy (ECT) causes side effects as well, and alternatives are under review, including transcranial magnetic stimulation (TMS). We describe the results and conclusions of preliminary studies on this new treatment.

Since this book first appeared we have made considerable progress in
understanding the biological basis of depression. Steps have been taken in identifying the genetic basis of the mood disorders, including schizoaffective disorder. Research on changes in hippocampal neurons and amygdala enlargement appears promising. “Neurotrophic” (keeping cells alive) and “neurogenesis” (stimulating growth of new cells) theories abound and are being tested.

Many of the biological aspects of depression still remain uncertain, though progress continues. One research area explores specific brain changes that correspond to the effective pharmacological and psychological treatments of depression. For example, studies have focused on differential effects in recovery for paroxetine (Paxil) therapy and cognitive therapy in modulating specific sites in limbic and cortical brain regions.

Researchers have continued to identify the pathophysiological aspects of major depressive disorder, including alterations in various monoamine brain systems. Neuropeptides such as corticotropin-releasing hormone are under investigation, as are hormonal variables such as glucocorticoid secretion. Dexamethasone nonsuppression of plasma cortisol has been suggested as a marker, although the same effects have been induced experimentally by sleep deprivation and dietary fasting.

Several studies have tested whether genetic markers can predict differential drug response, thus leading to the possibility of individualized pharmacologic treatment of depression. Response to paroxetine in relation to the serotonin transporter gene polymorphism (5-HTTLPR) have found reductions in depression ratings to be more rapid for certain genotypes than for others, despite equivalent paroxetine concentrations. Future studies in pharmacogenomics will continue to identify genetic markers in the hope of better predicting individual drug response, and the reasons for such response. The end result will be the possibility of individualized pharmacologic treatment of depression.

Clinical and psychosocial approaches to depression have made major strides. We now know a great deal more about cognitive vulnerability, the interaction of genetic predisposition with childhood and adult stress, and relapse than we did a generation ago. Most aspects of the cognitive theory of depression and suicide have been confirmed empirically, including negatively biased cognitions about the self, the importance of hopelessness as a predictor, content specificity of themes, and mood-congruent recall. Cognitive priming studies and studies utilizing longitudinal designs now support the theory of cognitive vulnerability in adults, and evidence is emerging for children as well.

Around the world, exciting research programs on clinical depression are underway. Cognitive therapies that target neurobiological mechanisms are being tested as adjuncts to conventional treatment. There is growing appreciation for the biopsychosocial nature of the mood disorders, along with an increased sophistication concerning the action of psychological and somatic therapies across multiple dimensions. The dichotomy between the phenomenological and the “biological” are increasingly understood to be, in reality, two sides of the same coin. For example, we review one report that found changes in thyroid hormone levels in response to cognitive therapy of major depression, consistent with the effect on the thyroid axis found in various somatic antidepressant treatments. Future studies are needed to test the effects of the cognitive and the somatic therapies on neurogenesis, particularly in
the granular cell level of the dentate gyrus (DG), the part of the hippocampus thought to be critical in laying down new cognitions.

As outlined above, depression research is vibrant and ever-changing. However, in addition to covering what is new, this Second Edition retains almost completely the original research and ideas of the First Edition. The basic theory of cognitive therapy was spelled out at that time. Part I, Clinical Aspects of Depression, keeps the naturalistic research on the cognitive aspects of depression (Chapter 2, “Symptomatology of Depression”). This work led to the cognitive content formulation which links the cognitive system to the affective, motivational, and physical phenomena of depression (Chapter 12, “Cognition and Psychopathology”). Part II, Experimental Aspects of Depression, includes the original tests of Freud’s theory that led to an “anomalous finding,” one that eventually generated a new system of treatment, cognitive therapy. This research is preserved also, as part of Chapter 10, including the dream study and the Negative Dreams (“Masochism”) Inventory (see Appendix).

Part III, Theoretical Aspects of Depression, contains from the First Edition the original idea of the negative cognitive triad in depression, and the theory of mania and other disorders, including anxiety, phobia, somatization, paranoia, obsessive compulsive disorders, and psychosis. Likewise, Chapter 13, “Development of Depression,” articulates the various causes of depression and has generated hundreds of studies. For these chapters, new sections add genetic findings, empirical support of the theory, and integrative theory that now underpins the general cognitive system of therapy. Thus, much of the first edition has been retained in the second, but the earlier work has been augmented and updated by the latest findings.

Part IV, Treatment of Depression, summarizes advances in somatic and psychological therapies. We review findings of randomized controlled trials, with special focus on comparisons between psychotherapy and antidepressant medication. Reviews of metaanalyses and conventional narrative reviews show certain psychological treatments and pharmacological therapy to be equally viable as clinical approaches to the mood disorders, with limited evidence suggesting the utilization of a combined approach. In addition, data now show the clear relapse prevention effect of cognitive therapy compared to medications. This includes group cognitive therapy for relapse of major depression, as well as for prevention of suicide reattempts in adults. Moreover, therapist experience with cognitive therapy is generally associated with better results.

Our comprehensive review of well-designed studies reveals that depressed patients treated with psychological interventions had a relapse rate of only 30 percent, compared to a relapse rate of 69 percent for patients treated with pharmacotherapy alone. We review studies that now support the routine use of maintenance treatment for depression. One major study calculated that half of all depression during the five years following a major depressive episode can be averted by using maintenance treatment, either cognitive behavior therapy or antidepressants.

In summary, where significant advances have occurred, we have incorporated these in this revision of Aaron Beck’s classic text. In cases where terminology is new, as in the case of the classification of disorders, current terms replace earlier ones or are
included alongside them. In the new edition, thus, we attempt to preserve the timeless material of the first edition and to distill all the timely advances that have occurred since then.

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Tables 1-2, 3-1, 4-1, 4-2, 4-3, 4-4, 6-1, 6-2, 6-3, 8-1 from American Psychiatric Association, Diagnostic and statitical manual of mental disorders, 4th ed., textual revisions (DSM-IV-TR)(Washington, DC: APA, © 2000).

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Part I

Clinical Aspects of Depression