

Cognitive Behavior Therapy for Depressed Adolescents

A Practical Guide to Management and Treatment

Randy P. Auerbach
Christian A. Webb
Jeremy G. Stewart



Cognitive Behavior Therapy for Depressed Adolescents

Cognitive Behavior Therapy for Depressed Adolescents provides clinicians, clinical supervisors, and researchers with a comprehensive understanding of etiological pathways as well as current CBT approaches for treating affected adolescents. Chapters guide readers from preparations for the first session and clinical assessment to termination and relapse prevention, and each chapter includes session transcripts to provide a more concrete sense of what it looks like to implement particular CBT techniques with depressed teens. In-depth discussions of unique challenges posed by working with depressed teens, as well as ways to address these issues, also are provided.

Randy P. Auerbach, PhD, ABPP, is a board-certified clinical psychologist and an assistant professor in the department of psychiatry at Harvard Medical School. At McLean Hospital, he is the director of clinical research for the division of child and adolescent psychiatry.

Christian A. Webb, PhD, is an instructor in the department of psychiatry at Harvard Medical School and an assistant neuroscientist at McLean Hospital.

Jeremy G. Stewart, PhD, is a postdoctoral fellow at McLean Hospital and a research fellow in psychology in the department of psychiatry at Harvard Medical School.

Cognitive Behavior Therapy for Depressed Adolescents

A Practical Guide to Management
and Treatment

Randy P. Auerbach

Christian A. Webb

Jeremy G. Stewart

First published 2016
by Routledge
711 Third Avenue, New York, NY 10017

and by Routledge
2 Park Square, Milton Park, Abingdon, Oxon, OX14 4RN

Routledge is an imprint of the Taylor & Francis Group, an informa business

© 2016 Randy P. Auerbach, Christian A. Webb, and Jeremy G. Stewart

The right of Randy P. Auerbach, Christian A. Webb, and Jeremy G. Stewart to be identified as the authors of this Work has been asserted by them in accordance with sections 77 and 78 of the Copyright, Designs and Patents Act 1988.

All rights reserved. No part of this book may be reprinted or reproduced or utilized in any form or by any electronic, mechanical, or other means, now known or hereafter invented, including photocopying and recording, or in any information storage or retrieval system, without permission in writing from the publishers.

Trademark notice. Product or corporate names may be trademarks or registered trademarks, and are used only for identification and explanation without intent to infringe.

Library of Congress Cataloging in Publication Data
Auerbach, Randy P.

Cognitive behavior therapy for depressed adolescents : a practical guide to management and treatment / Randy P. Auerbach, Christian A. Webb, Jeremy G. Stewart.

pages cm

Includes bibliographical references and index.

Depression in adolescence—Treatment. 2. Cognitive therapy for teenagers. I. Webb, Christian A. II. Stewart, Jeremy G. III. Title.

RJ506.D4A92 2016

616.85'2700835—dc23

2015029367

ISBN: 978-1-138-81613-8 (hbk)

ISBN: 978-1-138-81614-5 (pbk)

ISBN: 978-1-315-74630-2 (ebk)

Typeset in Baskerville
by Swales & Willis Ltd, Exeter, Devon, UK

Contents

<i>About the Authors</i>	viii
<i>Acknowledgments</i>	x
1 Introduction	1
<i>Epidemiology</i>	1
<i>DSM-5: Definitions and Subtypes</i>	3
<i>Cognitive Behavior Therapy</i>	5
<i>Goals for the Book</i>	7
<i>Intended Audience</i>	8
<i>Effective Use of the Book</i>	8
<i>Summary</i>	9
2 Models of Depression	10
<i>Life Stress and Adolescent Depression</i>	10
<i>Behavioral Models of Depression</i>	16
<i>Social Cognitive Learning Models of Depression</i>	18
<i>Cognitive Theories of Depression</i>	19
<i>Information-Processing Models of Depression</i>	24
<i>Summary</i>	29
3 Assessment	30
<i>Assessment Goals</i>	30
<i>Diagnostic Interviews</i>	32
<i>Self-Report Questionnaires</i>	35
<i>Observational Methods</i>	37
<i>Third-Party Reports</i>	38
<i>Suicide and Self-Harm Risk Assessment</i>	39
<i>Summary: An Ounce of Prevention is Worth a Pound of Cure</i>	41

4 Setting the Stage	43
<i>Psychoeducation</i>	44
<i>Collaborative Empiricism and Guided Discovery</i>	48
<i>Therapeutic Alliance</i>	49
<i>Alliance-Interfering Behaviors</i>	55
<i>Motivational Interviewing</i>	59
<i>Summary</i>	65
5 Starting	66
<i>Major Elements of Early Sessions</i>	66
<i>The Anatomy of Session 1</i>	79
<i>Session Content and Structure: Session 2 and Beyond</i>	84
<i>Behavioral Activation Strategies</i>	86
<i>Summary</i>	95
6 Working	96
<i>Identifying Negative Automatic Thoughts</i>	96
<i>Suggested Strategies to Elicit Negative Automatic Thoughts</i>	97
<i>The Bottom Line</i>	104
<i>Challenging Negative Automatic Thoughts</i>	105
<i>Suggested Strategies for Challenging Negative Automatic Thoughts</i>	105
<i>Between-Session Homework</i>	112
<i>Core Beliefs and Intermediate Beliefs</i>	113
<i>Communication Styles and Cognitions</i>	116
<i>Problem-Solving</i>	123
<i>Summary</i>	130
7 Maintaining Gains and Relapse Prevention	131
<i>Preparing for Treatment Termination</i>	131
<i>Reassessing Treatment Goals</i>	132
<i>Post-Treatment Competency in CBT Skills</i>	134
<i>Anticipating and Addressing Depression Triggers</i>	137
<i>Addressing Residual Symptoms</i>	138
<i>Addressing Underlying Vulnerability Factors</i>	141
<i>Additional Treatment</i>	142
<i>Parental Depression</i>	142
<i>Booster Sessions</i>	144
<i>Mindfulness-Based Interventions for Relapse Prevention</i>	145
<i>Summary</i>	158

8 Therapeutic Challenges and Comprehensive Care	160
<i>Family Involvement in Treatment</i>	160
<i>Cultural Considerations</i>	164
<i>Sleep</i>	174
<i>Comorbidity</i>	178
<i>Pharmacotherapy</i>	185
<i>Summary</i>	187
9 Addressing Suicidality	188
<i>Assessing Suicidality</i>	188
<i>Treatment of Suicidality</i>	199
<i>Summary</i>	203
10 Innovations and Future Directions in CBT	205
<i>Computer- and Internet-Based Interventions</i>	205
<i>Mobile Depression Interventions (Smartphones and Applications)</i>	210
<i>Providing CBT via Videoconferencing</i>	215
<i>Summary</i>	216
<i>References</i>	217
<i>Index</i>	243

About the Authors

Randy P. Auerbach, PhD, ABPP, is a board-certified clinical psychologist and an assistant professor in the Department of Psychiatry at Harvard Medical School. At McLean Hospital, he is the director of clinical research for the Division of Child and Adolescent Psychiatry as well as the director of the Child and Adolescent Mood Disorders Laboratory. Dr. Auerbach's program of research is aimed at identifying psychosocial, behavioral, and neurobiological factors that render certain children, adolescents, and young adults vulnerable to experience depressive symptoms and episodes, and the research also examines factors that contribute to successful cognitive behavioral therapy (CBT) interventions. This work is funded by grants from the National Institute of Mental Health, the Klingenstein Third Generation Foundation, the Dana Foundation, and several private foundations, and to date, it has resulted in over 50 published scientific papers and book chapters. Dr. Auerbach is the recipient of a number of awards, including the David Shakow Early Career Award for Distinguished Scientific Contributions in Clinical Psychology and the Society for Clinical Child and Adolescent Psychology Early Career Award.

Christian A. Webb, PhD, is an instructor in the Department of Psychiatry at Harvard Medical School and an assistant neuroscientist at McLean Hospital. Dr. Webb's primary area of research investigates the underlying psychosocial and neural mechanisms of symptom improvement in treatments for depression, with a particular focus on CBT. He also conducts research on the causes of depression, as well as studies low-cost, internet-based interventions for depression. He has received several awards for his research, including awards from the Anxiety and Depression Association of America, the Beck Institute for Cognitive Behavioral Therapy, the Canadian Psychological Association, and the Association for Behavioral and Cognitive Therapies. Dr. Webb's research has been supported through grants from the National Institute of Mental Health, the Social Sciences and Humanities Research Council of Canada, and the Klingenstein Third Generation Foundation.

Jeremy G. Stewart, PhD, is a postdoctoral research fellow at McLean Hospital and a research fellow in psychology in the Department of Psychiatry at Harvard Medical School. He completed his PhD in clinical psychology at Queen's University in Kingston, Ontario, Canada, which included a predoctoral internship at McLean Hospital. Dr. Stewart has devoted his career to better understanding factors that contribute to the development and maintenance of major depressive disorder in adolescents, with particular attention to the role of adverse experiences and stressful life events. Currently, Dr. Stewart is investigating the complex interplay among environmental, behavioral, and neural factors in predicting suicide risk in depressed adolescents. His research has been supported through awards from the Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council, and the Ontario Ministry of Training, Colleges and Universities.

Acknowledgments

My parents, Russell and Elyse Auerbach, have provided limitless support, and my wife, Tiffany Chantra, has showered me with warmth, laughter, and love, giving me the strength and courage to pursue that which seems out of reach. My mentorship team across the McGill, McLean, and Harvard community has nurtured my development as a clinical researcher and therapist, providing boundless opportunities to excel in the professional arena. Finally, I am indebted to the adolescents and families with whom I worked, as I learned invaluable lessons from all of you.

Randy P. Auerbach

To John Hunt: I am forever indebted to you for sparking my love of learning and teaching me to challenge fundamental assumptions. To my parents, Anthony and Micheline Webb: your sage advice and unwavering support over the years have provided me with immeasurable guidance and encouragement. To Renata: your love, laughter, and boundless *joie de vivre* inspire me each and every day. Finally, thank you to my many research and clinical mentors and colleagues, in particular at Penn and McLean Hospital: I am immensely grateful for your mentorship, wisdom, and the countless opportunities you have provided me.

Christian A. Webb

To my parents, Kent and Marianne Stewart: your patience and unconditional support throughout my education and training have allowed me to pursue my aspirations (and achieve some of them). To my wife, Stephanie Boyer: I am forever grateful for your generosity, warmth, and love. Your talents as a clinician and scientist continue to inspire my own work. To Kate Harkness: thank you for the opportunities you gave me, for fostering my creativity and for modeling an approach to clinical psychological science that I strive to emulate. To Michela David: thank you for sharing your substantial wisdom and skill and for approaching my CBT training with empathy and humor. Your teachings echo through the pages of this book. Finally, thank you to the exceptional group of clinical and research

mentors at both Queen's University and McLean Hospital that have supported and shaped my career, providing me with the necessary tools, inspiration, and opportunity to succeed.

Jeremy G. Stewart

1 Introduction

The prevalence of major depressive disorder (MDD) surges in adolescence and is associated with a range of negative downstream emotional, behavioral, interpersonal, and socioeconomic consequences (Greden, 2001; Greenberg et al., 2003). Despite relatively effective treatment options for depression in adolescence, the average length of a depressive episode is about 6 months and approximately 40–70% experience a recurrent episode within 5 years of the initial diagnosis (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015; Rao et al., 1996). Taken together, depression is both recurrent and debilitating, and, perhaps not surprisingly, it is one of the leading causes of disability and premature death worldwide (Kessler, 2012). Therefore, providing effective treatment for depressed adolescents, earlier in the disease course, is paramount.

Epidemiology

The most comprehensive epidemiological study completed on adolescent depression has been the National Comorbidity Study – Adolescent (NCS-A) Supplement. This project has amassed invaluable information about adolescent psychopathology, particularly as it relates to prevalence, age of onset, course, comorbidity, and treatment (e.g., Avenevoli et al., 2015; Nock et al., 2013). The study includes a large, ethnically diverse sample of adolescents ages 13–18 years ($n \sim 10,000$), and reports on clinical data (survey and interviews) collected over a span of 4 years (2001–2004).

Lifetime and 12-Month Prevalence

Our most recent estimates show that depression is a common and widespread problem. In school-aged children, prevalence rates range from 1% to 2% (for review, see Avenevoli, Knight, Kessler, & Merikangas, 2008); however, there is a dramatic increase during adolescence. Specifically, data from the NCS-A suggest that, by the end of adolescence,

2 *Introduction*

11% of teens will have experienced at least one major depressive episode (MDE), and moreover, 7.5% of adolescents met criteria for MDD in the previous year. Compared to males, female adolescents have a two- to threefold greater likelihood of experiencing MDD and are at four times greater risk for experiencing severe MDD. Similar effects are found when examining trends in age, as older adolescents are at twofold greater risk for MDD and a fourfold greater risk of reporting severe MDD relative to younger teens (Avenevoli et al., 2015). As a whole, these findings echo previous work that has shown that relatively older female adolescents are at greatest risk for experiencing MDD (Hankin, Mermelstein, & Roesch, 2007).

Twelve-Month Comorbidity

Depression rarely occurs in isolation, and results from the NCS-A suggest that approximately 64% of youth reporting MDD experience comorbid mental health disorders. Youth with MDD have a four times greater likelihood of reporting anxiety and behavioral disorders, and this comorbidity is associated with more severe depressive symptoms. Surprisingly, the pattern of comorbidity does not vary as a function of gender. This may reflect the fact that, among depressed youth the prevalence of oppositional defiant disorder, conduct disorder, and substance use disorders does not differ in males versus females (Avenevoli et al., 2015).

Socioeconomic Status, Race, and Ethnicity

In contrast to adults where depressive disorders are associated with a lower socioeconomic status (SES) (Kessler et al., 2003), reports in youth have been inconsistent (see Merikangas & Knight, 2009). Specifically, in a meta-analysis including 310 studies of youth, Twenge and Nolen-Hoeksema (2002) found no association between SES and depression. Nonetheless, in studies targeting the most impoverished individuals, a modest to moderate inverse association was found between SES and depression (Costello et al., 1996; Gilman, Kawachi, Fitzmaurice, & Buka, 2003).

There is limited research on racial and ethnic differences among depressed youth. Preliminary epidemiological investigations have not found between-group differences in the incidence of MDD among Caucasian and African American (Costello et al., 1998) or Native American (Costello, Farmer, Angold, Burns, & Erkanli, 1997) youth. Research examining differences in depressive symptoms has, however, indicated that Hispanic youth report modestly greater symptom severity relative to Caucasian and African American youth (Twenge & Nolen-Hoeksema, 2002). An important caveat to these findings is that the studies included relatively small samples, and therefore should be interpreted with caution.

Suicidality

Depression is intimately connected to suicidality; however, whereas depressive symptoms are predictive of increased suicidal ideation and a greater number of suicide plans, symptom severity is a weaker predictor of attempts (Nock, 2009). Twelve-month prevalence estimates from the NCS-A suggest that approximately 11% of depressed adolescents made an attempt, and this rate was nearly twofold greater among those with severe symptoms (~21%). Moreover, and strikingly, 75% of depressed adolescents make a suicide attempt in their lifetime (Nock et al., 2013).

DSM-5: Definitions and Subtypes

According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5: American Psychiatric Association, 2013) a MDE requires a patient to report five or more symptoms over a period of at least 2 weeks. At a minimum, patients must report depressed mood (or irritability in youth) and/or loss of interest or pleasure (i.e., anhedonia). Additional depressive symptoms include: weight change (i.e., 5% increase or decrease in body weight), sleep disturbance (insomnia or hypersomnia), psychomotor agitation or retardation, fatigue, feelings of worthlessness and/or excessive guilt, concentration difficulties, and recurrent thoughts of death.

To better define different *stages* of the MDE, consensus teams have agreed on definitions for the following terms: *episode*, *remission*, *response*, *recovery*, *relapse*, and *recurrence* (see Boland & Keller, 2009; Frank et al., 1991; for review, see Monroe & Harkness, 2011). Whereas an *episode* is operationalized as manifesting a set number of symptoms for a specific period of time, *remission* marks the end of a depressive episode when a person is either in *partial* or *full remission*. *Partial remission* is characterized by more than a minimal presentation of symptoms, but typically there is a reduction in the number and intensity of symptoms compared to within the episode. Conversely, when a patient is in *full remission*, he or she no longer meets diagnostic criteria for the disorder; however, it is not atypical for these individuals to experience some minimal symptoms. At its core, *remission* is an absence (or reduction) of symptoms; however, it does not assume that an intervention has been delivered. A *response*, by contrast, suggests that a course of treatment (e.g., psychotherapy, pharmacotherapy) has been applied, and that the depressive symptoms attenuated (e.g., >50% reduction) *as a result* of the intervention. *Recovery* connotes sustained improvement for roughly 4 months or that the underlying depressive episode has been resolved, and in most instances, a depressive episode is unlikely to surface in the near future. The difference between *relapse* and *recurrence* is important. A *relapse* is the return of a depressive

4 Introduction

episode after remission but before recovery, whereas *recurrence* refers to a new episode following recovery.

An important clinical distinction in diagnosing MDD in youth versus adults is the presence of irritability. Irritability is considered a cardinal symptom of depression among children and adolescents, and in fact, it is widely believed to be among the most frequently reported symptoms of moderate MDD in adolescents (see Crowe, Ward, Dunnachie, & Roberts, 2006). While adults often endorse irritability during subclinical and clinical depressive episodes, irritability in the absence of either sadness or anhedonia is not sufficient for a diagnosis of MDD. Similar to depressive episodes with strong anhedonic features (see Auerbach, Admon, & Pizzagalli, 2014; Loas, 1996), some research suggests that prominent irritability may reflect a subtype of MDD (Perlis et al., 2005). Specifically, Perlis and colleagues assert that irritability, as a feature of MDD, is cause for concern given that it may be associated with more severe symptoms, compromised functioning, and suicidality.

Persistent Depressive Disorder

An important change introduced in DSM-5 was merging chronic MDD and dysthymic disorder into persistent depressive disorder (PDD). PDD is characterized by depressive mood nearly all day, every day, for a period of at least 2 years. This depressive mood is accompanied by the presence of at least two of the following symptoms: appetite disturbance, sleep problems, low energy or fatigue, low self-worth, inattention/indecision, and hopelessness. If at any time an adolescent meets criteria for MDD (i.e., satisfying at least 5/9 symptoms, as described above), then a diagnosis of MDD is given in place of PDD. PDD also may be associated with anxious, atypical, and psychotic features.

Melancholia

A subtype of MDD that has received a great deal of clinical and research attention is melancholia (e.g., Curry et al., 2006). Melancholia requires the presence of anhedonia or lack of mood reactivity and at least three of the following symptoms: depressed mood, weight/appetite loss, psychomotor agitation/retardation, excessive guilt, and worse mood in the morning. Melancholia is believed to be biologically based, and studies have linked this subtype to hypothalamic–pituitary–adrenal axis overactivity as well as genetic factors (see Dinan & Scott, 2005; Kendler et al., 1996).

Specifiers

Although less frequently diagnosed, two cyclical subtypes of depression include seasonal affective disorder (SAD) and premenstrual mood

disorder (PMD). Blazer, Kessler, and Swartz (1998) found that approximately 1% of the population satisfy diagnostic criteria for SAD, and identified cases were more typical in winter months and more prevalent in northern versus southern latitudes (Blazer et al., 1998). Pearlstein and Stone (1998) indicated that mood changes occurring during the menstrual cycle are common; however, only 4–6% of women experience PMD (Sveindottir & Backstrom, 2000). A critical feature of PMD is the unequivocal recurrent onset–offset pattern of five or more depressive symptoms occurring in the majority of menstrual cycles for at least 1 year.

Additionally, the DSM-5 describes other specifiers such as recurrent brief depression, short-duration depressive episode (4–13 days), and depressive episode with insufficient symptoms. The empirical literature on these specifiers is sparse, but each connotes a shorter episode duration (i.e., recurrent brief depression, short-duration depressive episode) or subthreshold characteristics (i.e., depressive episode with insufficient characteristics).

Symptom Clusters

The manifestation of depressive symptoms is enormously heterogeneous, and consequently, the *experience* of MDD varies substantially from patient to patient. Importantly, there are preliminary data on how treatment may differentially impact symptom clusters. For example, Fournier and colleagues (2013) found that both antidepressant medication and cognitive behavior therapy (CBT) led to a greater reduction in cognitive- and suicide-related symptoms as compared to the placebo. Further, CBT was particularly effective in reducing atypical-vegetative symptoms (i.e., hypersomnia and weight gain) as compared to both antidepressant medication and the placebo. In contrast, Stewart and Harkness (2012) reported no differences; the authors concluded that antidepressant medication and CBT did not differentially impact cognitive versus somatic symptoms of depression. However, Harkness and Stewart (2009) reported that depressive symptom clusters may lead to the generation of different types of stressful life events. Whereas cognitive-affective symptoms led to higher levels of interpersonal-oriented events (e.g., conflicts with peers and parents), somatic symptoms generated more independent, or fateful life events (e.g., physical illness). As learning to effectively respond to, and cope with, life stressors is a central goal of CBT, attending to these differences may be of critical import when working collaboratively with youth.

Cognitive Behavior Therapy

CBT is the most empirically supported psychotherapeutic intervention for adolescent MDD (Spence & Reineke, 2003; Spirito, Esposito-Smythers,

6 Introduction

Wolff, & Uhl, 2011). Generally speaking, CBT provides a short-term (12–18 sessions), structured approach to systematically and strategically target dysfunctional interrelationships among thoughts, emotions, and behaviors (Figure 1.1). Patients learn about how negative cognitions and depressogenic information processing biases shape emotional experiences, particularly as this may relate to thoughts about the self, world, and future (i.e., the negative cognitive triad; Table 1.1). According to the cognitive behavioral model, *negative automatic thoughts* trigger negative emotions (e.g., sadness, anger, anxiety) and associated behaviors (e.g., isolating, self-harm). CBT helps patients identify patterns of distorted or biased thoughts, and teaches them skills to challenge these cognitions as a means of reducing emotional distress and maladaptive behaviors.

In addition to identifying and challenging depressogenic cognitions, CBT highlights the importance of *behavioral activation*. Depressed youth often isolate and avoid previously enjoyed activities (e.g., spending time with friends; playing sports), and consequently, a therapist uses a stepwise approach to help teens gradually re-engage with key sources of reinforcement and reward in their environment. Re-engaging is essential, as it also

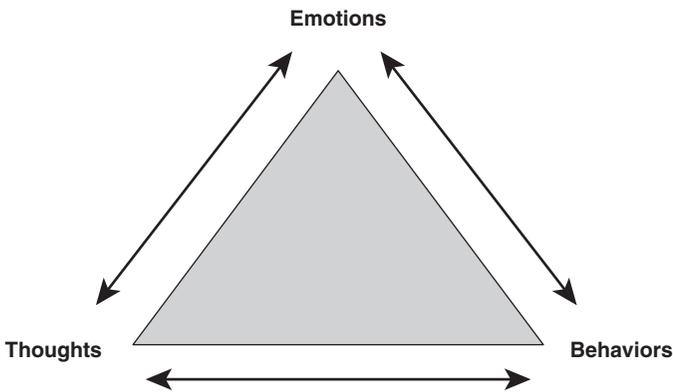


Figure 1.1 CBT Triangle: Thoughts, Emotions, and Behaviors

Table 1.1 Cognitive Triad

Domains	Definition	Example Negative Automatic Thought
Self	Self is worthless	I am unlovable
World	World is unjust	My parents and classmates don't want to spend time with me because I am unlovable
Future	Future is hopeless	I'll always be alone because I am unlovable

provides a testing ground to evaluate entrenched distorted patterns of thinking (e.g., Why bother going out? I won't have fun anyway), and the opportunity to experience pleasure.

Together, interventions aimed at challenging negative cognitions and reducing maladaptive behaviors are the bedrock for effective CBT. The therapy encourages an active and engaged adolescent patient, and, critically, necessitates an adaptable and flexible therapist. For patients who engage in treatment and persist with the exercises, optimal outcomes often ensue.

Goals for the Book

Before preparing this book, we brainstormed about the ideal components to include. After many hours of discussion, we decided it was important for readers to have essential information at their fingertips to target many important aspects of depression in adolescence. In the spirit of this aim, we sought to create a veritable *one-stop shopping* experience, including passages addressing the scope of problem (e.g., epidemiological data), background on prevailing models of depression (e.g., social-cognitive models), assessment (e.g., clinical interviews, narrow-band severity indicators), and treatment across all phases (i.e., early, middle, termination, and booster sessions). Although there are many books on CBT for depression (e.g., Beck, 1995, 2011; Friedberg & McClure, 2002), many are targeted toward younger people (i.e., children) or, most often, written with adult patients in mind. These books describe the array of cognitive and behavioral techniques available in CBT. Our goal is not to repeat the procedures and approaches detailed, but rather, to describe how to adapt CBT for adolescents. Given the unique developmental features of adolescence, we believe that tailoring one's approach may contribute to more effective outcomes. Adolescents are entangled in one of the most interesting and dynamic phases of human development, and it is a period rife with growth, discovery, angst, and uncertainty. Teens speak a language that is stridently different than that of children and adults. Thus, when administering CBT to depressed adolescents, it is essential to bear in mind these many differences and to tailor interventions and one's communication style accordingly. This book may serve as a *Rosetta Stone* for deciphering depressed adolescents, and by consequence, navigating potential pitfalls and obstacles that occur throughout treatment. Make no mistake, providing effective treatment for depressed adolescents is undeniably challenging, and at the same time, working with adolescents also may be among the most rewarding experiences. With all adolescents, there is a unique opportunity to guide them back on to a healthier developmental trajectory, which may have profound consequences across their lifespan.