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TREATMENT OF GENERALIZED ANXIETY DISORDER

Therapist Guides and Patient Manual

GAVIN ANDREWS, ALISON E.J. MAHONEY,
MEGAN J. HOBBS, AND MARGO R. GENDERSON

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Preface

When scared, our hearts beat fast, we feel short of breath, we break out in a cold sweat, we tremble and shake, and less noticeably, our pupils dilate and attention focuses. These physical changes are adaptive and facilitate the fight or flight response. If the threat is imminent but not yet upon us, we feel aroused as we prepare to fight or flee, and the same body changes occur. In short, we are anxious and we have little capacity to attend to other matters.

If the threat is in the future, not imminent, we worry about it, thinking about ways to neutralize it. At best, this evokes images and thoughts of strategies whereby the problem can be solved, sometimes realistically and sometimes unrealistically. The focus of our worry is on appraisal of the threat and its solution, not on how the problem could escalate. Flight or fight arousal symptoms are present but the changes are commensurate with the degree of the immediate threat. We are worried, but we can put the worry aside to attend to everyday tasks.

Some people, in the absence of danger, see threats everywhere and worry day after day. As the reality of the threat is somewhat tenuous, the thought “what if I’ve forgotten something the kids need for school?” is justified by the worrying thought “I’ll look like a bad mother” and so on, into the future, until the thought “and they’ll never get a good job” seems unreasonable. In such people, the worrying thoughts are verbal, the excessive and uncontrollable escalation is future-oriented, physical arousal is chronic but constrained, and there is surprisingly little rehearsal of problem-solving strategies. Everyday tasks are interfered with. The behavior is not normal problem-oriented worrying.

People who are anxious and worry in a maladaptive way benefit from good, proactive treatment. That is the focus of this book. Across the board, the book will be valuable to all proponents of the scientist–practitioner model. We begin by tracing the history of generalized anxiety disorder (GAD). We then look at the effectiveness of

pharmacological and psychological treatments, favoring the latter. In Chapter 4, we list contemporary models of GAD and explore new developments in cognitive behavior therapy. This chapter may be particularly applicable to the difficult-to-get-better patient. We then present a clinician's guide to treatment that covers assessment, formulation, and the beneficial and problematic steps in cognitive behavioral therapy. Finally, there is a patient treatment manual that can be used as a curriculum for individual or group therapy, or copied and provided to patients to work through on their own.

The first half of the book contains academic reviews that will be of most interest to researchers. The second half of the book contains practical advice that will be of most interest to clinicians and their patients. Now read on.

Disclaimer

GA and MH contributed to the DSM-5 proposals for the revision of the GAD classification. The views contained in this book are those of the authors and do not express the views or opinions of the American Psychiatric Association.

Contents

List of figures *xi*

List of tables and forms *xiii*

1 DSM-5 generalized anxiety disorder: the product of an imperfect science *1*

Introduction *1*

Creating a useful and reliable psychiatric classification *2*

Balancing tradition and validity *5*

Designing a classification that is valid but withstands
media sound bites *11*

Conclusions *16*

Questions for future research *16*

2 Generalized anxiety disorder assessment measures *19*

Introduction *19*

Structured diagnostic interviews *20*

Symptom measures *21*

3 Treatment effectiveness *23*

Pharmacological treatments *23*

Psychological treatments *26*

Questions for future research *33*

4 Next steps for improving treatment effectiveness *35*

Introduction *35*

Contemporary models of generalized anxiety disorder *36*

Effectiveness of treatment based on contemporary models *45*

The interaction between patient characteristics
and treatment approach *52*

Questions for future research *53*

5 Clinical guides to treatment 55

Physicians' guide 55

Clinicians' guide 57

Summary 102

6 Patient treatment manual 103

Section 1. Understanding generalized anxiety disorder 104

Section 2. Understanding what keeps your worry going 112

Section 3. Managing physical symptoms 124

Section 4. Managing thinking symptoms 129

Section 5. Advanced skills for managing worry thoughts 146

Section 6. Dealing with behaviors that affect worry 161

Section 7. Advanced skills for facing fears 168

Section 8. Putting it all together and staying well in the longer term 173

Record forms for patients 179

Progressive muscle relaxation exercises 180

Finding answers to common questions 189

References 191

Index 215

List of Figures

- 5.1 Contents of the clinicians' guide 58
- 5.2 Metacognitive formulation of Tom's worry 71
- 5.3 Intolerance of uncertainty formulation of Jenny's worry 74
- 5.4 Avoidance-based formulation of Petra's worry 76
- 6.1 Liz's worry cycle 113
- 6.2 Setbacks are normal 129
- 6.3 The ABC Model 130
- 6.4 Biased thinking can maintain anxiety 132
- 6.5 Avoidance maintains anxiety 162
- 6.6 Anxiety reduces when you stop avoiding 162
- 6.7 Desensitization over time 163
- 6.8 Liz's stepladders 165
- 6.9 The GAD cycle 173
- 6.10 Setbacks are normal 174

List of Tables and Forms

Tables

- 4.1 Outcomes of randomized controlled trials for treatments based on contemporary models of GAD 31
- 6.1 Liz's worry log 124
- 6.2 Thoughts influence how you feel and what you do (example 1) 130
- 6.3 Thoughts influence how you feel and what you do (example 2) 131
- 6.4 Thoughts can become self-fulfilling 131
- 6.5 Liz's thought-challenging record 137
- 6.6 Worry experiments 138
- 6.7 Liz's structured problem-solving worksheet 144
- 6.8 Liz's disadvantages and advantages of worry 147
- 6.9 Liz challenges a negative belief about worry (example 1) 149
- 6.10 Liz challenges a negative belief about worry (example 2) 150
- 6.11 Experiments to test negative beliefs about worry 152
- 6.12 Liz challenges a positive belief about worry 154
- 6.13 Experiments to test positive beliefs about worry 155
- 6.14 Liz's imaginal exposure 170
- 6.15 Repeating imaginal exposure 172
- 6.16 Early warning signs of relapse 176
- 6.17 Finding answers to common questions 189

Forms

- 6.1 What areas of your life does GAD interfere with? 107
- 6.2 Your worry cycle 122
- 6.3 Worry log 179
- 6.4 Monitoring relaxation exercises 182
- 6.5 Thought-challenging record 183

6.6	Structured problem-solving worksheet	184
6.7	Thought record for negative beliefs about worry	185
6.8	Thought record for positive beliefs about worry	186
6.9	Exposure stepladders	187
6.10	Imaginal exposure log	188

DSM-5 generalized anxiety disorder: the product of an imperfect science

Introduction

Psychiatric classification is difficult. Nosologists need to strike a balance between utility and reliability, between tradition and validity, creating an evidence-based manual that withstands media sound bites. The generalized anxiety disorder (GAD) classification has proven more difficult than most. Introduced in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) as a residual diagnosis, the GAD criteria had one of the lowest diagnostic reliabilities of the mood and anxiety disorders. The revised DSM-III breathed new life into the GAD criteria, identifying chronic apprehensive anxiety about multiple aspects of life as the defining feature of GAD. This innovation improved the reliability of GAD symptoms but concerns about reliability and validity of the diagnosis led some experts to suggest that GAD should be excluded from DSM-IV. This did not occur. After further revision in DSM-IV, the GAD criteria were established as an independent diagnosis. Some 30 years since the introduction of GAD in DSM-III, more recommendations were made to further improve the validity of the classification. Despite these recommendations and in the face of media skepticism about psychiatry as a science, none of these recommendations influenced the DSM-5 GAD classification. In this chapter, we trace the balancing act that has been the evolution of the GAD classification.

Creating a useful and reliable psychiatric classification

Clinicians need to use the DSM to distinguish between cases and non-cases of mental disorder and to distinguish between different types of mental disorder. Researchers need to use the classification for the same reason. Ideally, the way that the DSM defines these diagnostic boundaries is useful and reliable. Here, psychiatric classification becomes tricky. A useful classification is one that facilitates clinical and academic decision making and the communication of these decisions. Clinicians and academics should be able to read the DSM and understand the core clinical features of each disorder and recall these features when assessing cases. A reliable classification, however, is achieved by specifically defining the clinical features of the disorder by objective criteria. More often than not, the greater the specification, the greater the reliability of the diagnosis. Balancing utility with reliability is therefore a thin-edged sword. An unreliable psychiatric classification is useless. However, the more criteria that are used to define a disorder, the larger and more unwieldy the classification becomes. A psychiatric classification therefore needs to balance the need for useful diagnoses, which would include the minimum amount of detail needed to capture the core problem of each disorder, with reliable diagnoses, which use many criteria to specify disorders.

Generalized anxiety disorder (GAD) is an excellent example of how nosologists, who can improve diagnostic reliability, can also improve the utility of a diagnosis.

DSM-III

In DSM-III, GAD had no defining features. GAD had the lowest test–retest reliability estimate (other than simple phobia) with a kappa of 0.47 (Di Nardo et al., 1983). Inter-rater reliability estimates of the DSM-III GAD classification were only slightly higher, with fair agreement between experienced clinicians diagnosing a current episode of GAD (kappa = 0.57) (Barlow, 1985). Indeed, Barlow et al. (1986a) examined if GAD could be differentiated from depression and the other anxiety disorders based on the severity of patients' motor tension, autonomic hyperactivity, and vigilance scanning. No reliable distinctions

were identified, and the authors concluded that there was little need to include GAD in the DSM as a residual diagnosis.

DSM-III-R

GAD was specified further in DSM-III-R. Repetitive thinking about the potential negative outcomes of future events concerning multiple aspects of life became the defining feature of the classification (Barlow et al., 1986b). Patients' anxiety and worry was identified as excessive, unrealistic, and difficult to control (Craske et al., 1989; Sanderson and Barlow, 1990). GAD was now classified as a chronic disorder (Barlow et al., 1986a). Eighteen associated symptoms were itemized.

Despite these changes to the classification, the reliability of DSM-III-R-defined GAD was still not ideal, yielding estimates comparable to DSM-III, and DSM-III-R GAD was associated with low test-retest lifetime estimates ($\kappa = 0.39$) (Mannuzza et al., 1989). Inter-rater reliability was slightly better. There was an improved degree of inter-rater reliability for current cases of DSM-III-R-defined GAD (κ of 0.56) (Williams et al., 1992) and for GAD as a principal diagnosis (κ of 0.57) (Di Nardo et al., 1993). Although the diagnosis now had a unique and defining clinical feature, it was unclear why reliability estimates were not higher. Some experts hypothesized that it was difficult for clinicians to distinguish between the excessive worry of GAD and the obsessions of obsessive compulsive disorder (OCD). However, no inter-rater disagreements about whether patients' principal diagnosis was GAD or OCD could be identified (Brown et al., 1993).

Although the reasons for the relatively low reliability estimates remained elusive, the DSM-III-R GAD symptoms of worry could be assessed reliably. Sanderson and Barlow (1990) showed that individuals with GAD worried most about family, finances, work, and illness, and clinical assessments of the excessiveness and/or unrealistic nature of these worries had excellent reliability ($\kappa = 0.90$) (Sanderson and Barlow, 1990). Craske and colleagues also found high agreement between raters about the types of things that individuals with GAD worry about (91.2%) (Craske et al., 1989). Nevertheless, DSM-III-R-defined GAD remained one of "the more conceptually challenging [diagnoses] in psychiatric nosology."

The DSM-III-R GAD diagnosis had fair reliability at best and, at worst, the classification was of little use, for clinicians or for academics. At least, this is what some clinicians and academics argued. The frequent co-occurrence of DSM-III-R-defined GAD with other mood and anxiety disorders further complicated the future of the GAD classification. Debate about merging GAD with another mood or anxiety disorder ensued (Brown, Barlow, and Liebowitz, 1994). Indeed, some DSM-IV Anxiety Disorders Work Group members suggested that the empirical basis of GAD was not sufficient to warrant the inclusion of GAD in DSM-IV. An alternative was floated. GAD could be removed from the main text of the DSM-IV and included in the Appendix.

DSM-IV

Despite this debate, GAD survived in DSM-IV. This decision was made because even though comorbidity with other anxiety and depressive disorders was common, there was a proportion of patients who met the diagnostic threshold for GAD but did not meet threshold criteria for an additional mood or anxiety disorder. In defense of the disorder, it was noted that disorders other than GAD also had high rates of comorbidity (Brawman-Mintzer et al., 1993; Brown and Barlow, 1992), and that GAD was associated with a different age of onset than other anxiety disorders (Brown, Barlow, and Liebowitz, 1994).

With the publication of DSM-IV, in which the long list of associated symptoms was reduced to a manageable number, the balance between reliability and utility came down on the side of utility. The reliability of the GAD classification may not have been excellent, but there appeared to be a group of patients whose core issue was excessive and generalized worry, and clinicians therefore saw a continued use for the classification.

The GAD classification may have avoided relegation to the Appendix of the DSM-IV but the classification did undergo a series of revisions before inclusion in DSM-IV. Excessive, difficult-to-control, and chronic generalized worry remained the defining feature of GAD. The classification however was shortened in other ways. The “unrealistic” criterion was omitted from the classification. The list of 18 associated symptoms was also cut and the six most frequently endorsed associated symptoms remained. There were, of course, people who endorsed the symptoms

that were dropped. The job of a psychiatric classification is not to capture all variants of mental health problems. Instead, the classification needs to be pragmatic and to use the minimum number of symptoms to define each disorder, and to provide clinicians and researchers with sufficient detail to decide who meets the criteria for which disorder and who does not.

Reducing the number of symptoms in DSM-IV appears to have struck a good balance between detail and utility. The inter-rater reliability of the DSM-IV GAD classification trumped earlier classifications. There is good agreement about current ($\kappa = 0.65$) and lifetime ($\kappa = 0.65$) cases of DSM-IV GAD (Brown et al., 2001b; Lobbetael, Leurgans, and Arntz, 2011; Zanarini et al., 2000).

Balancing tradition and validity

Creating a useful and reliable classification may be difficult, but creating a reliable classification that is valid continues to challenge nosologists. The frequent co-occurrence of GAD with other mood and anxiety disorders, and what this co-occurrence means for the validity of the diagnosis, was a ubiquitous theme in the early GAD literature. It is more common in clinical practice to see people who experience GAD with other disorders than as a sole diagnosis (Brown and Barlow, 1992; Brown et al., 2001a). GAD also co-occurs with mood and other anxiety disorders in the community, albeit to a lesser degree than in clinical samples. Two thirds of current cases of GAD will experience at least one additional diagnosis (Hunt, Issakidis, and Andrews, 2002). Comorbidity is not reserved to GAD. All mood and anxiety disorders co-occur more often than not (Hettema, Prescott, and Kendler, 2003; Moffitt et al., 2007; Pine et al., 1998). More than half of the general population who experience a common mental disorder in their lives will experience more than one mental disorder (Kessler et al., 2005a; Slade et al., 2009).

To put fears about comorbidity and GAD to rest, the questions were not: does GAD co-occur with other disorders and is GAD secondary to them? Instead, the real questions to be answered were: are there people who meet criteria for GAD and regard it as more distressing and disabling than the other disorders, and are there people who meet criteria for GAD that have no other mental disorder and have significant

distress and impairment as a result? There is, and they do (Kessler et al., 2002; Wittchen et al., 2000). So perhaps co-occurrence of GAD with other disorders should not have been a problem for the GAD classification. Hindsight is always 20/20.

Psychiatric classification may be difficult but Barlow, Brown, and their DSM-IV Work Group contemporaries crafted increasingly useful and reliable criteria for GAD. Specifically, the evolving classification has progressively facilitated clinical distinctions to be made between GAD and adaptive worry, and between GAD and other mood and anxiety disorders. Academics have also been able to grow the empirical basis about the prevalence of GAD and about the risk factors and clinical correlates of the disorder.

DSM-IV-defined GAD is common. One in twenty adults experience GAD in their lives (4.1% to 6.0%) (Grant et al., 2005; Kessler et al., 2005b; Kessler and Wittchen, 2002; McEvoy, Grove, and Slade, 2011) and half of these adults report that they have experienced GAD in the past year (1.5% to 3.6%) (Carter et al., 2001; Hunt, Issakidis, and Andrews, 2002; Kessler, 2005a). Less data are available with respect to the population prevalence of GAD in childhood and adolescence. Estimates range between 0.8% and 2.2% for lifetime estimates and between 0.5% and 1.1% for past-year estimates (Kessler et al., 2012a, b; Wittchen, Nelson, and Lachner, 1998). The prevalence varies by gender and age. Females are twice as likely to experience GAD as males (Carter et al., 2001; Grant et al., 2005; Maier et al., 2000). GAD peaks in prevalence in middle age and declines in prevalence in the later years of life (Carter et al., 2001; Grant et al., 2005; Hunt, Issakidis, and Andrews, 2002; Kessler et al., 2005b).

GAD may be common but, as with most psychiatric disorders, the necessary and sufficient causes of GAD are not known. What is known is that one third of the liability to develop GAD is under genetic control (Hettema, Prescott, and Kendler, 2004; Kendler et al., 1994, 2003) and that the within-familial environment does not contribute substantially to developing GAD (0–4% of the liability) (Hettema et al., 2006; Kendler et al., 1992). This means that individual non-family environmental factors are the greatest predictor of GAD, and these are only modestly related to those factors associated with experiencing major depressive

disorder (MDD) (Hettema et al., 2006; Hettema, Prescott, and Kendler, 2004; Kendler et al., 1995, 2003, 2007). The special environmental triggers for GAD, however, are unclear. Childhood adversities are known to increase the likelihood of the development and persistence of all of the common mental disorders (Green et al., 2010; McLaughlin et al., 2010) but are neither necessary nor sufficient for, or specific to, GAD.

Some dose relationship between environmental factors and GAD may explain the relatively late age of first onset of GAD, which is in the early 30s (Grant et al., 2005; Kessler et al., 2005b). GAD may begin early in life but manifest as an anxious or neurotic temperament which, when paired with environmental triggers, results in acute episodes of anxiety (Akiskal, 1998; Kagan and Snidman, 1999). These acute periods of anxiety may increase in duration across the lifespan and, therefore, people who do not meet the criteria for their first episode of DSM-IV-defined GAD until their 30s may have experienced increasing periods of anxiety and worry prior to this (Kessler and Wittchen, 2002; Starvcevic and Bogojevic, 1999). This hypothesis is consistent with the slow waxing and waning course of GAD (Angst et al., 2009; Ballenger et al., 2001). GAD is characterized by periods of worrying that tend to last for months or years rather than days or hours (Grant et al., 2005; Yonkers et al., 2003).

Despite the chronic course of GAD, less than half of people who experience GAD seek treatment. Those who seek treatment wait more than ten years after the onset (Wang et al., 2005). Even then, treatment tends to be sought for comorbid somatic panic and depressive symptoms rather than anxiety (Judd et al., 1998; Kessler and Wittchen, 2002). A low number of people with GAD seek treatment from specialized mental health services, preferring to consult primary care physicians rather than psychologists or psychiatrists, and GAD is the most common anxiety disorder in primary care (Maier et al., 2000; Üstun and Sartorius, 1995; Wittchen, 2002).

The balance that nosologists have iteratively struck between the reliability, utility, and validity of the classification has delineated the prevalence and some of the risk factors and clinical correlates of GAD. These data show that GAD can be differentiated from other disorders. We said that, at one level, these data support the validity of the

GAD diagnosis. However, at another level, the traditional approach to classifying GAD as a categorical disorder may be limited. There are increasing structural data that show that many different disorders are experienced to different degrees. The diagnostic boundaries between adaptive cognitions and behaviors and mental disorders are porous. Most mental disorders exist as continuous phenotypes; most mental disorders are dimensional. For instance, people can be sad, but fleetingly so. People can endorse only a few of the symptoms of MDD and be below threshold, and those above threshold can have mild, moderate, or severe MDD. Exactly the same dimension occurs in GAD, from short periods of excessive worry to chronic and excessive worry that produces significant distress and impairment. The use of diagnostic symptoms and thresholds to define disorders do not, therefore, identify discrete diseases that are independent of healthy and adaptive cognitions and behaviors (Kendell and Jablensky, 2003). Instead, DSM symptoms index varying degrees of diagnostic severity; the diagnostic criteria define the point at which the disorder is identified as likely to require treatment.

No structural data have examined the relative validity of treating GAD as a categorical or dimensional phenotype. However, people worry to varying degrees (Ruscio, Borkovec, and Ruscio, 2001) and, as a core feature of GAD, this suggests GAD may also be dimensional. Epidemiological data about GAD suggest as much. If subthreshold and threshold cases of GAD were discrete, then it would be expected that these groups of individuals would be qualitatively distinct with respect to their risk factors, clinical correlates, and/or associated disability. However, epidemiological studies that have examined the risk factors and correlates of subthreshold and threshold cases of GAD show that these groups of individuals are not qualitatively distinct. Instead, individuals who endorse all the diagnostic criteria for GAD except, for example, that their anxiety was not excessive or that their symptoms lasted for less than six months, are similar with respect to risk and clinical correlates to individuals who endorsed all the criteria for GAD (Angst et al., 2009; Bienvenu, Nestadt, and Eaton, 1998; Lee et al., 2009; Maier et al., 2000; Ruscio et al., 2005; Slade and Andrews, 2001). These data suggest that there is a degree of continuity between subthreshold

and threshold cases of GAD. The balance that the DSM strikes between its categorical heritage and what the data say is convenient but may be misleading.

The traditional categorical approach in psychiatric classification of identifying distinct types of disorder is also problematic. Contrary to the traditional notions of psychiatric validity (Robins and Guze, 1970), the data have simply not proven that mental disorders are independent from each other. Instead, the comorbidity problem keeps rearing its head. There are consistent patterns that characterize the co-occurrence of the common mental disorders. For example, individuals who experience a unipolar mood or anxiety disorder are more likely to experience other anxiety disorders and mood disorders than substance use disorders, and people who experience substance use disorders are more likely to experience multiple substance use or conduct disorders than mood and anxiety disorders (Kessler et al., 2012b; Teesson, Slade, and Mills, 2009).

Numerous hypotheses about the reasons for diagnostic comorbidity exist. For example:

1. Comorbidity could result by chance.
2. The occurrence of one diagnosis could increase the risk of developing additional diagnoses.
3. Comorbidity could indicate that the respective disorders share a common diathesis.
4. Comorbidity could be a result of incorrectly lumping and splitting symptoms that index a shared pathology (Andrews, 1996; Hyman, 2007; Maser and Patterson, 2002).

Given that diagnoses co-occur at rates far exceeding chance levels, the first explanation is unlikely. There are, however, robust data that show that two latent factors explain the co-occurrence of the common mental disorders: the *internalizing* and the *externalizing* factors (Cox, Clara, and Enns, 2002; Krueger, 1999b; Krueger et al., 1998, 2003; Krueger and Markon, 2006; Lahey et al., 2008; Markon, 2010; Slade and Watson, 2006; Vollebergh et al., 2001). The internalizing factor explains the co-occurrence of the unipolar mood and the anxiety disorders. The externalizing factor explains the co-occurrence of the substance use and antisocial disorders.

Some studies have also found support for a division within the internalizing disorders, with the co-occurrence of GAD, MDD, dysthymia, and post-traumatic stress disorder (PTSD) explained by an *anxious/misery* subfacet of the internalizing factor, whereas the co-occurrence of panic and some of the phobic disorders is explained by reference to a *fear* subfacet of the internalizing factor (Krueger, 1999b; Slade and Watson, 2006). There is robust support for this internalizing–externalizing factor structure of the common mental disorders across age groups (Eaton, Krueger, and Oltmanns, 2011; Krueger et al., 1998; Lahey et al., 2008), ethnicities (Eaton et al., 2012), and countries (Fergusson, Horwood, and Boden, 2006; Kessler et al., 2005a; Krueger et al., 2003; Slade and Watson, 2006; Vollebergh et al., 2001). The internalizing–externalizing model of the occurrence of the common mental disorders has also been extended to explain the occurrence of psychotic-like experiences (Laurens et al., 2012; Wright et al., 2013). As well as explaining the phenotypic covariance of the unipolar mood and anxiety disorders, the internalizing factor explains the genotypic covariation and the shared environmental risk factors of these disorders (Hettema, Neale, and Kendler, 2001; Kendler et al., 2011; Kendler, Davis, and Kessler, 1997; Kendler et al., 2003). This common genetic covariation also overlaps with the genetic risk of being neurotic (Hettema et al., 2006). These patterns of comorbidity, risk factors, and clinical correlates challenge the validity of the notion that the DSM is “carving nature at its joints” and identifying discrete diseases by applying the categorical approach to classification (Kendell and Jablensky, 2003).

In an attempt to bring tradition into accordance with reality, proposals were made to reconfigure the DSM-5 chapters. Based on the risk factor similarities between many disorders, five disorder chapters were proposed for DSM-5: the neurodevelopmental, neurocognitive, internalizing, externalizing, and psychotic-like disorders (Andrews et al., 2009a, b; Carpenter et al., 2009; Goldberg et al., 2009; Krueger and South, 2009; Sachdev et al., 2009). To some extent, the organization of DSM-5 follows this arrangement (American Psychiatric Association, 2013). The important relationships within the internalizing disorders and within the externalizing disorders, and between both clusters of