Treatment of Comorbid Anxiety Disorders across the Lifespan

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The fourth edition of the *Diagnostic and Statistical Manual for Mental Disorders* (DSM-IV; American Psychiatric Association [APA], 2000) lists 12 diagnostic categories for anxiety disorders, leading to over 25 distinct anxiety diagnoses on Axis I (Norton, 2008). Decades of research on these disorders have found very high rates of comorbidity (i.e., co-occurrence of disorders within an individual), both for mood disorders and other anxiety disorders (Brown, Campbell, Lehman, Grishman, & Mancill, 2001; Kessler, Chiu, Demler, & Walters, 2005). In fact, a substantial body of research suggests that presentation of a single anxiety disorder, with no comorbidity, is the exception and not the norm (Brown, Di Nardo, Lehman, & Campbell, 2001; Davis, Barlow, & Smith, 2010), even for those disorders with the lowest comorbidity rates (Goisman, Goldenberg, Vasile, & Keller, 1995). This information is not completely surprising, given the overlap in both etiology and diagnostic symptoms across these disorders (see Lawrence & Brown, 2008 for a review).

The high rates of overlap among anxiety disorders have numerous implications across the research and practice realms. One of the most important is that many clinical trials for treatment of disorders restrict the types of participants, particularly excluding those with highly complex comorbid condition (e.g., psychosis or bipolar disorder) and instead focusing on “pure” clients. As a result, our current empirically supported treatments may not be generalizable to a large portion of clients – those with comorbid conditions (Goldenberg et al., 1996). Over the last 15 years, considerable effort has been put forth to examine how comorbidity influences treatment, as well as how treatment can influence comorbidity. Does the presence of a comorbid disorder make treatment more difficult? Will treating a primary disorder impact the

comorbid disorder? The focus of the current chapter will be to review this research, particularly focusing on how such knowledge can allow psychologists and other mental health practitioners to more effectively treat clients who present with multiple, comorbid anxiety disorders.

Anxiety Disorders and Comorbidity

As mentioned, epidemiological and community-based research have found high rates of comorbidity for persons with an anxiety disorder, even when compared to other categories of mental disorders (Toft et al., 2005). In terms of specific rates, research has shown differential patterns of comorbidity for the different anxiety disorders. For the below information, the first disorder named would be considered the primary diagnosis, with the following being the most typically comorbid diagnoses.

Generalized anxiety disorder (GAD) has shown high rates of comorbidity with several other anxiety disorders, with estimates from 66-83% (Goldenberg et al., 1996; Wittchen et al., 1994). For persons with primary GAD, the most commonly seen anxiety disorders are social and specific phobias (Borkovec, Abel, & Newman, 1995; Davis et al., 2010), panic disorder, and posttraumatic stress disorder (PTSD; Kessler et al., 2005). For panic disorder (with or without agoraphobia), the most commonly co-occurring anxiety disorders are specific phobias, social phobias, and generalized anxiety (Brown et al., 2001; Kessler, Chiu, Demler, & Walters, 2005). In persons with PTSD, high rates of social phobias are seen (Zayfert et al., 2002), as well as panic disorder (Kessler et al., 2005), although the most commonly co-occurring disorders are non-anxiety ones (e.g., major depression and substance abuse; Brady, Killeen, Brewerton, & Lucerini, 2000).

Social anxiety disorder (also known as social phobia) is highly comorbid with GAD, obsessive compulsive disorder (OCD; Davis et al., 2010), agoraphobia, specific phobias, and panic disorder (Kessler et al., 2005). Obsessive compulsive disorder has some of the highest comorbidity rates, with almost half of persons being diagnosed with a co-occurring anxiety problem (Weissman et al., 1994), most commonly panic disorder, phobias, or generalized anxiety (Brown & Barlow, 1992; Davis et al., 2010; Kessler et al., 2005). For those with a primary diagnosis of a specific phobia, much lower rates of comorbidity exist compared to other anxiety disorders (Brown et al., 2001); however, for those with a different primary anxiety disorder, specific phobias are the most common co-occurring disorder (Sanderson, Di Nardo, Rapee, & Barlow, 1990). Given the low incidence rates of persons with only a specific phobia presenting for treatment (Silverman & Kearney, 1992), though, it is more likely that that a specific phobia will be a comorbid, non-primary diagnosis in someone presenting for treatment.

Impact of Comorbidity on Disorder Severity

General clinical and research consensus is that, compared to someone presenting with only a single anxiety disorder, those persons who meet diagnostic criteria for multiple anxiety disorders tend to have more severe symptoms. In terms of course, single anxiety disorders have a later onset and are less likely to remit on their own than co-occurring disorders (Bruce, Machan, Dyck, & Keller, 2001; Goldenberg et al., 1996). Research examining social phobia has found more severe pretreatment symptoms in both the primary (social phobia) and comorbid disorders (Erwin, Heimberg, Juster, & Mindlin, 2002; Mennin, Heimberg, & Jack, 2000). Similar findings have been seen with generalized anxiety (Belzer & Schneier, 2004; Nutt, Argyopoulos, Hood, & Potokar, 2006; Wittchen et al., 1994). In one study examining PTSD in a non-combat

population, comorbid anxiety disorders were not related to PTSD severity (Zayfert et al., 2002), although a later study found that those with PTSD and a comorbid diagnosis of social phobia were more clinically severe than those with PTSD or social phobia alone (Zayfert, DeViva, & Hofmann, 2005).

There are, however, mixed outcomes in primary panic disorder diagnoses, with some studies finding higher symptoms (Allen et al., 2007) and some finding no differences in symptoms between persons who did and did not have comorbid disorders (Tsao, Mystkowski, Zucker, & Craske, 2005). One study in particular that studied comorbid GAD and panic disorder found that those with both had significantly less satisfaction in personal relationships, lower functioning, and lower emotional health (Massion, Warshaw, & Keller, 1993).

Anxiety Comorbidity and Treatment Impact

There has been a substantial body of research devoted to examining the treatment implications of comorbidity. Overall, presence of comorbidity does not appear to diminish the benefits of empirically supported treatments. For example, Brown, Antony, and Barlow (1995) found that presence of comorbid conditions in primary panic disorder did not impact short or long-term outcome for panic symptoms. Most research has yielded similar results about panic symptoms (e.g., Tsao, Mystkowski, Zucker, & Craske, 2005), but not all studies have confirmed such findings. One replication instead found comorbidity was associated with less likelihood of treatment response (Tsao, Lewin, & Craske, 1998).

Results are more mixed with other anxiety disorders. Treatment outcome for primary GAD with or without any comorbid conditions has shown no differences in short-term treatment response (Mennin et al., 2000), although others have shown comorbid anxiety

disorders to decrease remission rates in the long-term (Bruce et al., 2001). In contrast, GAD comorbid with OCD or panic disorder has been linked to attenuated treatment response (Steketee, Chambless, & Tran, 2001). Primary social phobia treatment outcome has not been negatively impacted by comorbid anxiety disorders (Brown et al., 1995).

Research on OCD is also mixed. Having primary OCD with comorbid PTSD has been found to decrease response rate (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002), while OCD and comorbid GAD was shown to increase dropout rates and decrease treatment response (Sketekee et al., 2001). In contrast, others have shown no negative impact on OCD treatment from comorbid anxiety problems in adults (Steketee et al., 1999; Storch et al., 2010) or children (Storch et al., 2008).

Treatment Approaches for Comorbid Anxiety Disorders

As discussed above, research has begun to examine the impact of treatment approaches for comorbid anxiety disorders with mixed results. As reviewed below, recent literature has demonstrated decreases in symptom severity and number of comorbid conditions following treatment for certain disorders, but not for others. Two distinct approaches to treating comorbid anxiety disorders have been developed and will be outlined below: transdiagnostic treatments and treatments designed to specifically treat specific combinations of disorders.

Transdiagnostic Approaches

Transdiagnostic treatments are predicated on three notions (Norton, 2008). First, that anxiety disorders may not be independent from one another; second, that treatments for specific anxiety disorders are robust; and finally, that some diagnoses may lead to other diagnoses. Given these ideas, treatment geared toward the primary diagnosis should then

generalize to the comorbid diagnoses as well. Due to the fact that there is no one "transdiagnostic treatment," but are instead treatments for specific disorders, below are summaries of research into transdiagnostic therapies, arranged by the primary diagnosis treated.

Social Phobia. One example of transdiagnostic treatment is cognitive-behavioral group therapy (CBGT; Juster & Heimberg, 1994; Heimberg, 1991; Heimberg & Juster, 1994). This type of treatment was first developed for use with individuals with social phobia and has been adapted for transdiagnostic groups as discussed below. Cognitive-behavioral group therapy begins with providing individuals with a cognitive framework for understanding the link between faulty beliefs about and anxiety during social interactions. Restructuring activities are then employed to help participants identify negative automatic thoughts, begin to classify them by cognitive distortion type, dispute negative thoughts using Socratic questioning and challenging of underlying assumptions, and finally developing coping self-statements. The behavioral component follows cognitive therapy. Individuals develop concrete goals (i.e., measurable, observable, attainable) for exposures. Further, participants are given "homework" assignments to complete exposures between sessions. Since CBGT is conducted in a group setting, this improves access to care and helps normalize symptoms of social phobia.

Mennin and colleagues (2000) examined the efficacy of CBGT in 122 adults with social phobia with and without GAD. Results indicated significant improvement in social phobia symptoms as well as the comorbid GAD symptoms. They proposed that pretreatment GAD symptoms exacerbate the avoidance, fear, and worry associated with social phobia, thus

treatment of social phobia may generalize to the overlapping symptoms in GAD (Menin et al., 2000). This seemed to be confirmed by their findings.

Specific Phobias. A one session cognitive behavioral treatment has been developed for specific phobias (Ost, 1989; 1997). It is predicated on the belief that individuals experience catastrophic thinking related to the feared stimuli which leads to avoidance. Therapy uses exposure to allow the individual to have positive experiences with the feared stimuli which allow individuals to collect experiences that contradict their faulty assumptions about the stimulus. Treatment is conducted in a single session that may last up to three hours. Participants are gradually exposed in vivo to the feared stimuli. Cognitive strategies are not used by the therapist during session but participants are encouraged to draw conclusions based on the positive experiences. With each step in the graduation, the participant must experience a reduction in anxiety of at least 50%.

In two studies, Ollendick et al. (2010) and Ost et al. (2001) report that youth undergoing this therapy experienced not only a significant drop in symptoms of the primary anxiety disorder (specific phobia) but also reductions in clinical severity of other comorbid phobias and anxiety disorders. They attribute this to increased self-efficacy following treatment of and "conquering" a specific phobia.

Generalized Anxiety Disorder. Different combinations of treatments have shown success in treating GAD and comorbid anxiety disorders. However, some common elements of treatment are shared by those found to be the most effective in reducing GAD and other comorbid diagnoses. Each of the proposed treatments involve self-monitoring of symptoms and cues, employing active strategies to reduce anxiety, and practice using skills between sessions

(Newman & Borkovec, 1995). For example, self-control desensitization therapy (Goldfried, 1971) requires individuals to practice relaxation skills in response to exposure to anxiety-producing stimuli and worries. Participants are taught to use pleasant imagery, diaphragmatic breathing, and progressive muscle relaxation. They are then instructed to use these skills when cued to by an anxiety provoking situation. Additionally, cognitive therapy also been examined. As discussed previously, cognitive therapy for anxiety involves identification of negative automatic thoughts, systematic evaluation of anxious thoughts, and restructuring techniques to make thoughts more adaptive. Specific to GAD, cognitive therapies typically include discussion of intolerance of uncertainty, poor problem-solving, and avoidance. Finally, CBT adds behavioral exposure to cognitive training. Following cognitive training, participants are encouraged to use skills in real world situations with clear goals for each exposure.

Newman and colleagues (2010) compared a treatment combination of self-monitoring, active anxiety reduction, and homework assignment in a sample with GAD and comorbid anxiety disorders. Results indicate that psychotherapy for primary GAD led to a reduction in number and severity of comorbid anxiety disorders at both post-treatment and follow-up. Borkovec et al. (1995) reported similar results in a sample of adults with GAD. Participants experienced a reduction in primary GAD as well as significantly fewer comorbid diagnoses following successful treatment of GAD. Ladouceur et al. (2000) examined a cognitive treatment for GAD. Results suggest that focusing on GAD specific elements of cognitive therapy lead to reductions in comorbid anxiety disorders. Specifically, they reported that participants had more than a 50% decrease in number of comorbid illnesses which were maintained at follow-up one year later.

Panic Disorder. Panic disorder treatment has also been shown to uimpact comorbid conditions. Typically, treatment of PD uses cognitive behavioral methods developed by Craske and Barlow (Panic Control Treatment; 1993). First, individuals are provided with a physiological explanation for panic symptoms; then, they are taught cognitive restructuring techniques to combat overestimations of risk related to these symptoms. Next, participants learn breathing retraining for use when hyperventilation occurs during a panic episode. Individuals are then gradually exposed to internal and external cues to panic using interoceptive (internal) and in vivo (external) exposure. Self-monitoring of anxiety and home practice are also required between treatment sessions.

Several authors have examined the effect of this treatment in PD with comorbid diagnoses (e.g., Brown, Antony, & Barlow, 1995; Tsao, Mystkowski, Zucker, Craske, 2005). Brown et al. (2000) reported a dramatic decrease in number and severity of comorbid conditions at post-treatment. However, in contrast to other research, participants had returned to pretreatment levels of comorbidity at two year follow up and were more likely to seek additional treatment between follow ups. Tsao and colleagues (2005) found that participants experienced significant reductions in both PD symptoms and comorbid diagnoses. Interestingly, the reduction in comorbid diagnoses in this sample was not significant until follow-up (9 months), which is a departure from other research. However, severity of comorbid diagnoses did decrease from pre- to post-treatment.

Specific Therapies

In contrast to transdiagnostic therapies, there are two therapies that have been designed to treat specific clusters of anxiety disorders. For example, multi-channel exposure

therapy (MCET) was developed to treat post-traumatic stress disorder (PTSD) and comorbid panic attacks (Falsetti, Resnick, & Davis, 2005). This treatment combines aspects of cognitive processing therapy (Resick & Schnicke, 1993) and panic control treatment (Barlow & Craske, 1994). Treatment in MCET targets symptoms of both PTSD and panic attacks through physiological, cognitive, and behavioral channels. Initially, participants in MCET are introduced to the idea that panic attacks and symptoms are conditioned responses to conditioned stimuli related to the initial unconditioned trauma. Following from this explanation treatment includes; (1) cognitive restructuring techniques to respond to catastrophizing and overestimation of trauma; (2) interoceptive exposure to physiological symptoms of panic and in vivo exposure to reminders (cues) to trauma; and (3) additional cognitive work involving exposure to the trauma via writing and reading aloud about the initial trauma.

Research has shown MCET to be effective over a waitlist control (Falsetti, 2003) as well as a minimal attention control (Falsetti, Resnick, & Davis, 2005). Falsetti, Resnick, Davis, and Gallagher (2001) used the above model in 22 women in a group format. They reported reductions in the number of women meeting diagnostic criteria for PTSD, panic attacks and symptoms, fear of having another attack, as well as reduction in the interference associated with attacks that were present and symptoms of depression. Additionally, Falsetti and colleagues (2003) reported improved function in a sample of female trauma victims following MCET. Participants experienced reduced symptoms of PTSD and panic attacks and experienced improvement in functioning in work, marriage, general functioning. Reductions were maintained at three and six month follow ups. Treatment gains have been shown to extend past the six month follow up as well (Falsetti, Resnick, & Davis, 2008).

A second specific therapy has been developed for individuals with GAD and comorbid panic disorder with agoraphobia (PDA; Labrecque, Dugas, Marchand, & Letarte, 2006). This treatment method is based on the notion that available cognitive-behavioral therapies of the individual disorders shared common components that could be used in treating both disorders together. Labreque et al. (2006) combined features from other available treatments for panic (Craske & Barlow, 1993; Marchand & Letarte, 1993) and GAD (Dugas & Ladouceur, 2000; Ladouceur et al., 2000). One major tenant of combining the treatments is to help individuals tolerate the uncertainty/discomfort that accompanies both GAD and PDA. Treatment components include: providing psychoeducation to participants about anxiety and specific diagnoses; information regarding symptoms and maintenance of panic; breathing retraining; cognitive restructuring; confronting beliefs; interoceptive and in vivo exposures; exposure to worries; training in problem orientation; and relapse prevention. Results of preliminary studies have demonstrated a reduction in symptom presentation of both GAD and PDA at post-treatment and follow up (Labrecque, Dugad, Marchand, & Letarte, 2006; Labrecque, Marchand, Dugas, & Letarte, 2007).

Case Study

Leah (a pseudonym) was a 16-year-old Caucasian female referred by her psychiatrist for evaluation and treatment of obsessive compulsive disorder, panic disorder without agoraphobia, and major depressive episode, recurrent. Specific symptoms of both anxiety disorders are listed below. Leah received her initial diagnoses from her psychiatrist according to the DSM-IV-TR (APA, 2000) at the age of 14 years. Leah was referred for treatment due to increased anxiety in numerous situations, ritualistic behaviors (i.e., excessive cleaning and

organizing), and impairment in social and academic functioning. Based on her symptoms, impairment, and results of clinical evaluation, Leah was given the above diagnoses. Leah was otherwise typically developing aside from hypothyroidism which was managed by a pediatric endocrinologist and stable at treatment entry. She was in a regular classroom and during periods of lower anxiety was performing above average academically and had several friends.

Assessment of Anxiety Symptoms

Leah began exhibiting a significant number of ritualistic and avoidant behaviors around the age of 14 years. Specifically, these included contamination concerns, avoidance of contaminated items (e.g., public areas), excessive cleaning/washing (i.e., items in the home such as books as well as her person), concerns about organization, excessive list making, and other just right phenomena (i.e., evening things out, repeating tasks until they felt just right). Leah reported feeling increasingly anxious when prevented from engaging in rituals. Her symptoms began interfering with academic, social, and family functioning at this time. Specifically, teachers noticed that assignments were completed only after long delays and that she was not spending time with peers during free periods. At home, Leah spent considerable amounts of time organizing and straightening her room. For example, each time she needed to put clean laundry away she would refold all clothes in the same container/drawer.

In terms of panic symptoms, Leah began having panic attacks (PA) once per month after her 15th birthday. She experienced her first PA at home while relaxing. This attack lasted approximately 15 minutes and involved increased heart rate, sweating, shaking, nausea, and derealization. In response to the PA, Leah became concerned with having another panic attack and worried that she was “going crazy.” Hence, she limited social interactions due to these

concerns. Leah also experienced symptoms of MDD, likely as a result of OCD and PD. Specifically, Leah reported feeling depressed mood and anhedonia. These mood disturbances appeared to result from her combination of OCD and PD, specifically her functional impairment (e.g., lack of social interactions and limited range of pleasurable activities). She was sleeping more than 11 hours per night, felt lethargic, and reported extreme indecisiveness. This was differentiated from indecisiveness related to anxiety by the nature of content (i.e., minor decisions rather than related to organizing or just right phenomena).

Treatment

Leah presented for 14 sessions of CBT with exposure and response prevention. Therapy was based on the model proposed by March and Mulle (1998) and validated in several studies (e.g., POTS, 2004; Storch et al., 2007). Treatment was conceptualized as a transdiagnostic approach and included additional sessions in which physiological symptoms of panic were targeted using CBT principles.

Sessions one and two focused on an introduction to therapy, psychoeducation, and fear hierarchy construction. Leah was asked to begin monitoring her thoughts between sessions and recording them using a thought record. Leah completed this easily and brought back a significant number of thought records between sessions. Hierarchy items included contamination related items (e.g., showering and cleaning excessively) and numerous organization/just right phenomena (e.g., list making, reorganizing bedroom and clothes). Leah's final hierarchy consisted of 25 items, the majority of which were related to avoidance and organizing. Initial cognitive work was begun in the second session; this involved having Leah begin to differentiate between "typical" and "OCD" worries and concerns.

Sessions three through eight primarily consisted of exposure and response prevention work. As an example of an exposure, Leah was required to “disorganize” her wallet, backpack, or computer files while in session. Exposures were continued until habituation to the feared stimuli was achieved and her anxiety was greatly decreased. Leah initially resisted some of the exposures, stating that they were not relevant to her treatment. However, over the course of treatment, Leah began to acknowledge that habituation in session and during homework was generalizing to more naturalistic, real world situations and, thus, was valuable. Exposures were conducted during each session with increasing difficulty. Due to Leah’s PA symptoms, particular attention was given to the physiological symptoms of anxiety during exposures. Other tasks during this portion of treatment involved reviewing homework from between sessions, which typically included additional exposures in the home as well as continued cognitive work. Leah also reviewed and practiced coping statements and cognitive restructuring in session.

Sessions nine through twelve focused on continued exposure with the introduction of interoceptive exposures for panic symptoms. As Leah had been primed to attend to physical symptoms in earlier exposures and to actively use coping statements, she was receptive to this type of exposure. Leah was assigned additional interoceptive exposures in places and at times which were related to her PA (e.g., at school, the mall). For example, one homework assignment was to go to a shopping center in the area and walk up and down the steps between floors until her heart rate was elevated at least three times during a shopping trip and remain at mall for a minimum of 30 minutes following exposure or until habituation occurs. We also continued to review cognitive strategies at the end of each session.

Relapse prevention and preparation for termination were covered in session twelve. Psychoeducation was provided regarding possible triggers for Leah's symptoms to return, for example, increased stress or less attention paid to returning symptoms. The importance of continued exposure and cognitive work at home was reinforced. Leah was asked to prepare any questions to be discussed with the therapist in the final session. Session 14 was conducted three weeks after session 13 to allow Leah to have additional time to work on symptoms at home. In the final session, termination was discussed and relapse prevention reviewed.

Assessment

Leah completed the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997) at pre- and post-treatment (session 13). At pre-treatment, Leah received a severity rating of 20, indicating moderately severe OCD symptoms. At the final session, Leah's CY-BOCS score had reduced to a 3, which is well within normal limits. Although Leah has not returned for a follow up assessment, per her psychiatrist she continues to maintain post-treatment levels of anxiety. Prior to treatment, Leah was experiencing monthly PA with worry about having one on a daily basis. After the first month of treatment, during which time Leah had one PA, Leah did not experience any further PAs. Worry about her attacks decreased over the course of treatment, so that at her 14th appointment, Leah reported only minimal worry (1 out of 10) and noted that this only occurred occasionally. Although there was no specific measure of MDD symptoms, anecdotally, Leah reported increased engagement in activities with friends, improved mood, decreased sleep, and she obtained a job at a coffee shop which she enjoyed.

Future Directions

Although much has been learned over the last 20 years regarding comorbidity in anxiety disorders, many questions are still unanswered. Currently, the most pressing need for research concerns finding the most effective and efficient treatment. As mentioned above, many studies investigating the treatment of anxiety disorders use participants without comorbid or complex presentations, undermining the ability of the results to generalized to the majority of clients (Goldenberg et al., 1996). Evidence suggesting that frequency and severity of comorbidity does not predict CBT treatment outcome (Davis, Barlow, & Smith, 2010; Storch et al., 2010) implies that exclusion criteria for CBT research studies no longer needs to include comorbid anxiety disorders.

Future research needs to establish whether focusing treatment on the primary diagnosis is superior to combining treatments for primary and comorbid diagnoses into one treatment (Labrecque et al., 2007). Studies have found treatments focusing on the primary diagnosis not only effectively treat the primary diagnosis, but also significantly decrease the frequency and severity of comorbid disorders (Newman, Przeworkski, Fisher, & Borkovec, 2010; Ollendick, Ost, Reuterkiold, & Costa, 2010). There is, however, evidence that combining treatments for both the primary and comorbid diagnosis is effective (Labrecque et al., 2007), which has led to the development of transdiagnostic treatments (such as those as reviewed above) that emphasize commonalities across disorders to treat the primary and comorbid diagnoses concurrently (McEnvoy, Nathan, & Norton, 2009). Future research should directly compare diagnosis-specific (e.g., multichannel exposure therapy) and transdiagnostic treatments to determine each method's effect on outcome variables such as diagnosis-specific symptoms, higher-order and common factors (e.g., negative affectivity, locus of control, emotion regulation), non-specific

factors (e.g., therapeutic alliance, group cohesion), treatment compliance and attrition, and duration of treatment effects and remission rates on primary and comorbid diagnoses (Storch et al., 2010). Research also needs to be undertaken to determine if treatments addressing both disorders simultaneously are superior to those treating disorders separately in discrete sessions (Reinecke & Hoyer, 2010).

A thorough understanding of the mechanisms that facilitate improvement of comorbid disorders may assist in determining whether diagnosis-specific or combined treatments are more effective. The answer may depend on the type and relatedness of the comorbid disorders (Reinecke & Hoyer, 2010) or the components within the treatment (Labrecque, Marchand, Dugas, & Letarte, 2007). Approaches that emphasize commonalities across disorders significantly reduce comorbid disorders (McEnvoy, Nathan, & Norton, 2009). For example, Labrecque and colleagues (2007) found that treatments that identified intolerance of bodily sensations for PDA and intolerance of uncertainty for GAD as vulnerability factors were preferred by participants. The authors suggest treatment models that address intolerance may be effective regardless of which disorder is the main diagnosis. Another study found that the more perceived control participants reported on a post-treatment anxiety control questionnaire, the greater the decrease in comorbidity (Craske et al., 2007). It is also possible, however, that decreases in comorbid disorders occur because clients apply techniques learned in treatment to their comorbid disorders (McEnvoy, Nathan, & Norton, 2009). Given that effective treatments for various anxiety disorders tend to share common features (e.g., exposure with response prevention, cognitive restructuring, relaxation training), this seems likely.

The optimal intensity of treatment also remains unclear, as one study reduced the clinical severity of comorbid anxiety disorders, which are typically treated with 12-16 sessions of CBT, using only one 3-hour session to treat youth with specific phobias (Ollendick, Ost, Reuterkiold, & Costa, 2010). It is unknown if other intensive treatments (e.g., multiple times weekly, extended therapy sessions) for different primary disorders would have the same impact. Future research also needs to identify the mechanisms leading to optimal outcomes and ascertain if the same effects occur for these disorders when they are more severe or when they are the primary disorder rather than the comorbid one.

Despite all that is known, more advances in the understanding of comorbid anxiety disorders need to be made. Najavits and colleagues (2008) speculate that future research will result in advances in the types of studies conducted, (e.g., an increase in randomized controlled trials), increases in training and dissemination, and a deeper understanding of the comorbidities themselves (e.g., rates, causal relationships, and prognosis).

Conclusions

Over the last two decades, our knowledge regarding anxiety disorders has grown immensely, especially in our understanding of issues surrounding comorbidity. We are now beginning to understand how different disorders present comorbidly, how those presentations impact both each other and treatment, and how to treat comorbid disorders. At the current time, there is strong evidence to support both transdiagnostic and specific therapies as effective, although much research remains before we have a full understanding of which works better. It may be that a transdiagnostic approach will work better for certain comorbid

combinations while a specifically developed therapy works better for others, or it could be that both are equally effective.

Regardless, today's practitioners should be aware that both methods have been shown to be useful in treating numerous combinations of anxiety disorders, as shown in both the above reviewed literature and the case study. Undoubtedly, given the enormous rates of comorbidity seen in those with anxiety disorders, most practitioners treating those with anxiety are likely to already be using some type of transdiagnostic or personally-developed specific therapies. It can be immensely beneficial to both researchers and clinicians to share this type of information with each other, particularly if more private practice clinicians were able to share knowledge gain from clinical experience and expertise. This could be done by engaging in systematic case studies regarding treatment methods and outcomes on typical, outpatient populations. Researchers should attempt to reach out to practitioners and form collaborations, supplying their expertise in data collection and analysis and possibly helping to secure grant funding so that the extra work involved in doing research in the context of a private practice would not be financially burdensome on practitioners.

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