Chapter 13
Anxiety Disorder and Substance Use Disorder Co-Morbidity: Common Themes and Future Directions

Sherry H. Stewart and Patricia J. Conrod

In this concluding chapter to the volume, we first provide a theoretical integration of the material contained in the initial two sections of the book. We review models, theories, and mechanisms to account for the high co-morbidity of anxiety and substance use disorders including notions involving self-medication, substance-induced anxiety, and third variable (e.g., anxiety sensitivity) explanations. Which particular pathway is most likely to be at play in explaining co-morbidity onset appears to vary as a function of the precise anxiety disorder involved as well as the specific substance being abused. We then move on to a consideration of processes involved in the maintenance of co-morbidity, as this knowledge may prove most useful in treatment. We consider recent evidence as to whether the presence of a co-morbid anxiety disorder impacts recovery from a substance use disorder. Regardless of the specific pathway to the onset of co-morbidity, once both disorders are present, they may serve to maintain one another or even exacerbate one another to create a vicious cycle such that the presence of one disorder can impede recovery from the other. In this chapter, we present an adaptation of Marlatt and Gordon's (1985) cognitive behavioral model to understand the factors and processes involved in the maintenance of anxiety disorder – substance use disorder co-morbidity. We conclude with a review of promising new approaches to the treatment and prevention of co-morbid anxiety and substance use disorders. We contrast sequential, parallel, and integrated approaches and present a theoretical argument for the superiority of integrated interventions, setting an agenda for future clinical trials in this area. Finally, various practical issues around the provision of treatment for co-morbid anxiety disorder – substance use disorder patients are considered.

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Causal Pathways

As noted in chapter 1, there are a variety of models, theories, and mechanisms that might explain the high degree of co-morbidity of anxiety disorders and substance use disorders observed in clinical and community samples alike. Three potential models of the onset of co-morbid anxiety and substance use disorder are illustrated in Fig. 13.1. The first two of these (top two panels in Fig. 13.1) posit a direct causal relation between the two disorders. For example, the model indicated in the top panel of Fig. 13.1 posits that anxiety disorder promotes the development of a substance use disorder. Theories that have been proposed to support this model include the self-medication (Khantzian, 1985), tension reduction (Greeley & Oei, 1999), and stress-response-dampening (Sher & Levenson, 1982) theories which hold in common the idea that anxiety disordered patients learn to use substances for the reinforcing effects that result from substance use (see review in Morris, Stewart, & Ham, 2005). A variety of mechanisms could be operative to explain this potential causal pathway (see chapte 2 and 3 for reviews of possible neurobiological, neuroendocrine, and psychophysiological mechanisms). For example, some drugs have negatively reinforcing anxiolytic, stress-response dampening, or depressant properties (e.g., alcohol) which could be particularly rewarding to an individual suffering from an anxiety disorder. Drugs might also exert their reinforcing effects via cognitive means (e.g., alcohol's dampening of the tendency to catastrophize the meaning of arousal-related bodily sensations among panic-prone individuals; see MacDonald, Baker, Stewart, & Skinner, 2000). A less obvious example of self-medication is through a process of enhancement of a sense of well-being induced by some drugs like cocaine and ecstasy, which may also be reinforcing for those with anxiety-related disorders.

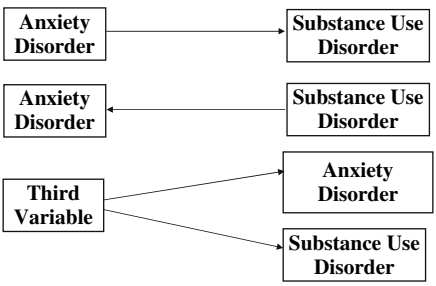

Fig. 13.1 *Illustration of three possible models of the onset of anxiety – substance use disorder co-morbidity*. The top panel is consistent with a self-medication theory of co-morbidity; the middle panel is consistent with a substance-induced induction of anxiety theory; and the bottom panel is consistent with a common third variable explanation (e.g., common personality or genetic predisposition causes both anxiety and substance use disorder, while there is no causal relation between anxiety and substance use disorder, *per se*)

A second potential model indicated in the middle panel of Fig. 13.1 posits that substance use disorder promotes the development of an anxiety disorder. Both psychological and neurobiological theories have been developed to explain this substance-induced anxiety enhancement model (see review in Sabourin & Stewart, in press). Suggested neurobiological mechanisms have included a 'kindling' process induced via multiple substance withdrawal experiences (see Kushner, Sher, & Beitman, 1990), or substance-induced disruptions in the stress-response system (see chapter 2). Chapter 10, for example, provides a solid theoretical articulation of the ways in which a history of smoking and nicotine dependence can contribute to the development of panic-related psychopathology – a theory which is consistent with this second model in Fig. 13.1 (see also Zvolensky, Schmidt, & Stewart, 2003). Chapter 5 provides a similarly useful review of why use of various other drugs might be panicogenic.

In terms of the support for these two direct causal models, one approach has been to examine relative order of onset of the two disorders in co-morbid cases. Although this approach alone does not establish causality, temporal order of onset consistent with the proposed direction of causality is a necessary (but not sufficient) condition for determining causation (Chilcoat & Breslau, 1998). Chapter's 13 review of the epidemiologic literature suggests that in at least 75% of cases of co-morbidity involving substance dependence, the anxiety disorder developed first. This means that substance-induced anxiety is a viable explanation for the onset of co-morbidity in only 25% of cases. Another method has been to use the methods outlined in the DSM-IV (American Psychiatric Association [APA], 1994) to distinguish between 'independent' and 'substance-induced' anxiety disorders not only by examining relative order of onset, but by examining whether the anxiety persists for at least four weeks after cessation of substance abuse and withdrawal. An epidemiologic study by Grant et al. (2004) strictly applied these criteria and showed that substance-induced anxiety was actually quite rare (see review in chapter 1). Taken together, these findings suggest that the self-medication theory (top panel of Fig. 13.1) is more consistent with the epidemiologic data on co-morbidity than the substance-induced anxiety theory (middle panel of Fig. 13.1).

However, which of the two causal hypotheses is best supported appears to vary as a function of the specific anxiety disorder in question as well as by the specific substance involved. For example, the fact that generalized anxiety disorder has been shown to be likely to resolve following substance use disorder treatment (Kushner et al., 2005) suggests that this particular anxiety disorder is likely to be substance-induced (middle panel of Figure 13.1) among co-morbid cases. In contrast, Kushner et al. (2005) have shown that co-morbid social phobia and post-traumatic stress disorder are unlikely to resolve with substance use disorder treatment, a pattern that is inconsistent with a substance-induced anxiety pathway to co-morbidity. Another approach has been to examine individuals' own perceptions of whether they are self-medicating their anxiety; this tendency has been shown to vary across anxiety disorders. For example, a

recent study by Bolton, Cox, Clara, and Sareen (2006) used data from the National Co-morbidity Survey (NCS) to examine self-reports of self-medication with alcohol/drugs among those with an anxiety disorder in a representative American sample. Consistent with theoretical speculation that self-medication is unlikely for certain anxiety disorders where substance use is not a socially acceptable coping response (e.g., Kushner, Abrams, & Borchardt, 2000), the lowest rates of self-medication were observed in those with the public speaking fear subtype of social phobia (i.e., where only 7.9% reported self-medicating). But, interestingly, the highest rates of self-medication were observed among those with generalized anxiety disorder (GAD) where a full 35.6% endorsed self-medicating with alcohol/drugs. Given the inconsistencies across methodological approaches regarding the direction of causality in GAD – substance abuse co-morbidity (e.g., Kushner et al., 2005 vs. Bolton et al., 2006), the nature of the relationship between GAD and substance abuse is clearly a topic deserving of further research attention. This dearth of knowledge is also evidenced by the absence of a specific chapter devoted to this topic in the current volume.

In terms of variable support of the self-medication vs. substance-induced anxiety models across specific drugs, the review in chapter 5 points out that studies examining order of onset of panic attacks and substance use have generally shown that, with the exception of alcohol use, the substance use generally precedes the development of panic attacks. This is inconsistent with a self-medication model of the development of co-morbidity. Unfortunately, most epidemiologic surveys comparing alcohol use disorders to 'other drug use disorders' as a group fail to provide more precise information about the types of drugs that are most likely fit to the self-medication pathway to co-morbidity development. We recommend that in the future, statistics be provided separately by drug class as was done in the Epidemiologic Catchment Area (ECA) survey (Regier et al., 1990) to allow for evaluation of important hypotheses such as that drugs with depressant or tranquillizing effects are most likely to fit the self-medication pathway to co-morbidity, while drugs with stimulant effects are most likely to fit the substance-induced anxiety pathway to co-morbidity.

A third possible model of the high co-morbidity of anxiety and substance use disorders is the 'third variable' model, illustrated in the bottom panel of Fig. 13.1. This model posits that a common underlying vulnerability (the third variable) contributes to the development of both disorders, while there is no direct causal relation between the two disorders themselves. Possible candidates for such third variables include a common personality predisposition (e.g., anxiety sensitivity; see reviews by Stewart & Kushner, 2001; Stewart, Samoluk, & MacDonald, 1999) or a common genetic basis to the two disorders. For example, family and twin studies have provided some evidence of possible common genetic contributions to the correlation between anxiety symptoms and alcohol consumption (e.g., Tambs, Harris, & Magnus, 1997). It should be noted that the genetic and personality vulnerability theories are not mutually exclusive in that a genetic predisposition could result in a specific personality vulnerability profile (e.g., demonstrated genetic contribution to anxiety

sensitivity; Stein, Jang, & Livesley, 1999), which in turn could predispose to the development of both anxiety and substance use disorders. Further research is needed on various third variable candidates before firm conclusions can be reached regarding the relative utility of the model at the bottom of Fig. 13.1 compared to the direct causal models presented in the upper portions of Fig. 13.1. If common third variables can be identified, this has important prevention implications as programs targeting the common risk factor (see chapter 11) can have a 'double impact' in preventing both anxiety and substance use disorders.

Maintenance

The factors involved in the maintenance of anxiety – substance use disorder co-morbidity need not be the same as those involved in co-morbidity onset. In fact, as suggested in the review above, there are likely multiple pathways to the development of a co-morbid anxiety disorder. But once the two disorders are present in a given individual, each may serve to maintain or even exacerbate the other. This 'vicious cycle' involved in the maintenance of anxiety disorder – substance use disorder co-morbidity is illustrated in Fig. 13.2. The figure makes clear how both the self-medication and substance-induced intensification of anxiety processes described earlier contribute to the maintenance of co-morbidity. For example, in chapter 5, Norton applies this mutual maintenance model to the understanding of panic disorder – alcohol dependence co-morbidity. In a patient with this form of co-morbidity, regardless of whether the panic attacks or alcohol abuse began first, once the two problems are established, the co-morbid patient may continue to use alcohol to manage his panic symptoms in the short term, with frequent experiences of alcohol withdrawal actually exacerbating panic symptoms in the longer term to ultimately create a vicious cycle between the symptoms of the two disorders.

The mutual maintenance model makes certain predictions. Most importantly for treatment, this model predicts that if one were to attempt to treat one of the two disorders without simultaneously treating the co-morbid problem, the individual would be at high risk of relapse to the treated disorder. For instance, in the case example above of the patient with co-morbid panic disorder and alcohol dependence, if we treated the patient's alcoholism without attending simultaneously to the panic disorder, the patient would be at high risk of relapsing to alcohol misuse, particularly during the withdrawal phase, since he would have no other means of coping with his untreated panic anxiety and his fear of interoceptive cues. We now turn our attention to data which has examined this prediction of the mutual maintenance model.

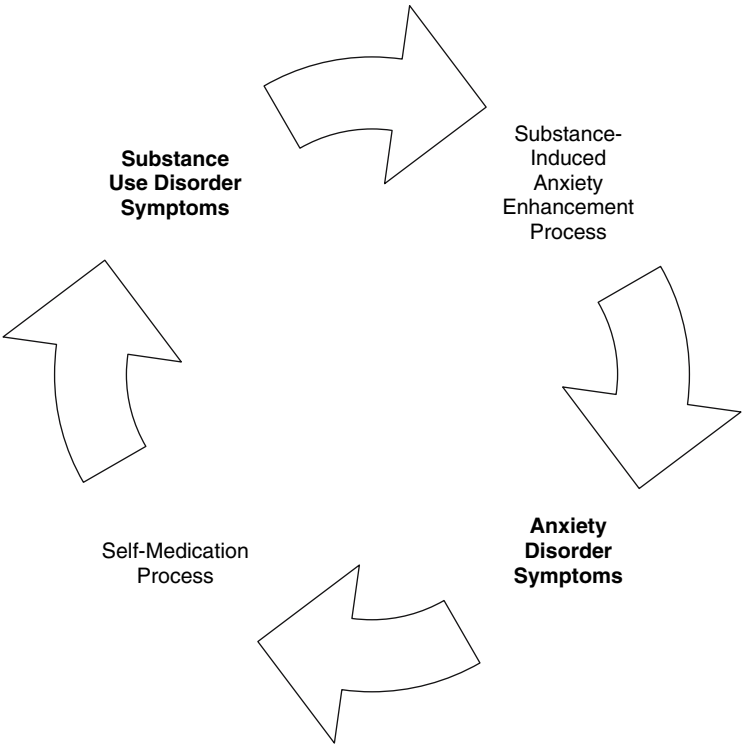

Fig. 13.2 *Illustration of the vicious cycle at play between anxiety disorder and substance use disorder symptoms in co-morbid individuals*. Note: This model is also referred to as the 'mutual maintenance' model

Impact of Co-morbid Anxiety on Recovery from a Substance Use Disorder

When individuals who suffer from co-morbid anxiety – substance use disorders enter treatment for either disorder alone, their treatment outcome is often affected negatively by the presence of the co-morbid disorder. For example, alcohol use disorders have been found to predict poorer anxiety disorder treatment outcomes for patients with panic disorder/agoraphobia, generalized anxiety disorder, social phobia (Bruce et al., 2005), and PTSD (Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003).

Co-morbid anxiety disorders have also been shown to increase the likelihood of relapse to substance misuse in treated or abstinent substance abusers (e.g. Driessen et al. 2001; Kushner et al., 2005; Willinger et al., 2002) as would be predicted by the mutual maintenance model. But not all studies have shown this relationship, however. For example, Marquenie et al. (2006) compared relapse rates for alcoholics with co-morbid panic disorder/agoraphobia or

social phobia to relapse rates for alcoholics with no co-morbid anxiety disorder, following standard alcoholism treatment. Inconsistent with predictions of the mutual maintenance model, the co-morbid anxiety disorders did not have a significant impact on either relapse rates or number of days to relapse. Nonetheless, Sabourin and Stewart (in press) have pointed out some methodological problems that may account for this long-term retrospective study's failure to support higher substance abuse relapse among treated alcoholics with co-morbid anxiety disorders.

In contrast to the results of Marquenie et al. (2006), several studies have reported higher substance use relapse rates in co-morbid anxiety disorder patients. For example, a study that examined the effects of trait anxiety on alcohol abuse relapse in among treated alcoholic patients showed that higher trait anxiety levels significantly predicted relapse to uncontrolled drinking (Willinger et al., 2002). Similarly, Driessen et al. (2001) showed that treated alcoholic patients with co-morbid anxiety had about 30% higher alcoholism relapse rates than did treated alcoholics without co-morbid anxiety. Most recently, Kushner et al. (2005) found that alcoholic patients with a co-morbid anxiety disorder (especially those with co-morbid panic disorder or social phobia) were significantly more likely to relapse to problem drinking than alcoholic patients with no co-morbid anxiety disorder. These findings are particularly convincing given the methodological soundness of the study (e.g., use of multiple criteria for drinking relapse; see review in Sabourin & Stewart, in press). Thus, it appears fairly safe to conclude that the presence of a co-morbid disorder impacts treatment outcome and relapse rates for the treated disorder, in a pattern consistent with mutual maintenance model predictions. This pattern strongly suggests that both disorders need to be addressed simultaneously to improve treatment outcome for co-morbid anxiety – substance use disorder patients.

Review of Promising New Approaches to Co-morbid Anxiety and Substance Use Disorder Treatment

Those who suffer from anxiety disorder – substance use disorder co-morbidity present a challenging population with respect to treatment. As we reviewed above, this population often suffers worse anxiety and substance use disorder treatment outcomes and appears at increased risk for relapse to substance misuse relative to those suffering from only one of these two disorders. Although the study of specific treatments for anxiety – substance use disorder co-morbidity is still in its infancy, this area is growing rapidly. There are now several promising approaches to treating co-morbid anxiety and substance use disorder as illustrated through each of the chapters in the treatment portion of this book. In this section, we briefly review the state of knowledge regarding effective treatments for anxiety – substance use

disorder co-morbidity in an attempt to integrate the material presented in the third section of this book.

The self-medication theory proposes that treatment of the 'underlying' anxiety disorder should have effects not only on the anxiety disorder, but also on symptoms of the substance use disorder. There have been mixed findings regarding the effects of pharmacological treatment for anxiety on substance use outcomes. Some studies have demonstrated improvements in substance use outcomes while other studies have found more mixed results (for more information, see review by Kushner et al., 2000). For example, Randall, Johnson, et al. (2001) attempted treatment of individuals with co-morbid social phobia and alcohol use disorder via paroxetine (a selective serotonin reuptake inhibitor [SSRI] that is established in the treatment of social phobia; see review by Marshall, this volume). As noted in the social phobia – SUD co-morbidity treatment chapter in the present book (see chapter 8), Randall, Johnson, et al. (2001) found that treating these co-morbid individuals with paroxetine did lead to improvements in anxiety and in the Clinical Global Index for alcohol, consistent with predictions of the self-medication theory. However, contrary to predictions of the self-medication theory, treatment with paroxetine did not result in significant decreases in drinking quantity and frequency. Two other similarly-designed studies by Kranzler et al. (1994) and Tollefson, Montague-Clouse, and Tollefson (1992) demonstrated that, consistent with self-medication theory predictions, successful treatment of anxiety with buspirone also led to a reduction in alcohol use. These findings are partially consistent with predictions of the self-medication hypothesis. Nonetheless, the paroxetine study (Randall, Johnson, et al., 2001) appears to suggest that there is more to the maintenance of problematic drinking behavior in socially phobic individuals than just the self-medication process.

More consistent with a mutual maintenance model, recent studies have examined the idea that treating both disorders may be the best approach to intervention in anxiety disorder – SUD co-morbid individuals. One way of classifying the various combined approaches is whether they are sequential (treating one problem and then the other), parallel (both treatments provided simultaneously but not necessarily in an integrated fashion), or integrated (Sabourin & Stewart, in press). Integrated treatments recognize the complex relationship between anxiety disorders and substance use disorders in co-morbid individuals (Zahradnik & Stewart, in press). The aim of integrated treatments is to create a hybrid of the treatments that are already known to work best for each disorder individually. Furthermore, truly integrated approaches explicitly include in the treatment strategy, an understanding of the reciprocal influences each disorder has on the other (Zahradnik & Stewart, in press). While sequential treatments continue to be the norm in clinical practice (e.g., the common practice of having an individual address their substance use disorder before they are accepted into anxiety disorder treatment), research has begun to address the utility of parallel and integrated approaches. Most studies to date

have focused on parallel approaches. The findings for parallel approaches thus far appear quite mixed with results varying from significant positive effects, to no group differences, to significant negative effects of combined treatments relative to control treatments.

For example, Randall, Thomas, and Thevos (2001) examined whether conducting two parallel cognitive behavioral therapy (CBT) treatments aimed at decreasing social phobia symptoms and at addressing problematic alcohol use would have additional benefits for social phobia – alcohol use disorder co-morbid individuals compared to co-morbid individuals treated for the alcohol use disorder alone. For the parallel treatment, the sessions consisted of CBT treatment for alcohol followed immediately by CBT for social phobia (i.e., the two treatments were offered simultaneously by the same therapist, but independently of each other). In direct contrast to predictions of the mutual maintenance model, Randall, Thomas, et al. (2001) found that patients who participated in the parallel treatment had *worse* drinking outcomes, as assessed by drinking quantity and frequency measures, than did those who participated in the alcohol only treatment. There are several possible explanations for these unexpected findings. Clients in the parallel treatment group may have participated in more social activities following their social phobia treatment, resulting in more opportunities to drink. Additional research needs to be conducted that would include other types of outcome measures that are not specifically linked to frequency or quantity of drinking. As suggested by Stewart, Morris, Mellings, and Komar (2006), coping drinking motives and problematic consequences of drinking are useful therapy targets for co-morbid social phobic – alcohol abuse patients. It is also possible that the lack of integration of the two treatments or the excessive demands of combining two already intensive treatments may have affected results in the Randall, Thomas, et al. (2001) study. The parallel treatment did in fact lead to somewhat higher drop out rates than the alcohol treatment alone, suggesting that the parallel treatment may have been too demanding for co-morbid patients to handle (Conrod & Stewart, 2005).

A similarly-designed study by Bowen, D'Arcy, Keegan, and Senthilselvan (2000) found *no* group differences in alcohol outcomes among panic disorder – alcohol use disorder co-morbid patients when patients were offered parallel CBT for panic disorder and standard alcohol treatment vs. alcohol treatment alone. Interestingly, though, both treatments resulted in significant reductions in anxiety. There are at least two possible interpretations of these findings. One explanation is consistent with the alcohol-induced anxiety theory of co-morbidity. Specifically, some might argue that additional panic-focused treatment is not necessary since anxiety will resolve once drinking levels are reduced. Another explanation, suggested by the authors, is that the relaxation training and stress management components of the standard alcohol treatment might have limited the ability to distinguish between treatments as these components may have been useful in targeting the co-morbid anxiety even in the alcohol alone treatment (i.e., control) condition. If so, then this study begs the question of what specific anxiety-management strategies are necessary and

sufficient for co-morbid anxiety disorder – SUD clients in combined treatment approaches.

A recent randomized, controlled trial conducted by Schade et al. (2005) compared standard alcohol treatment alone to standard alcohol treatment with anxiety treatment consisting of CBT plus optional fluvoxamine (an SSRI; see review in chapter 12) treatment (again, a parallel approach) in patients with a primary diagnosis of alcohol dependence and a co-morbid diagnosis of panic disorder, agoraphobia, or social phobia. There were no differences in alcohol outcome measures between the two groups of patients. The additional anxiety treatment did, on the other hand, improve anxiety symptoms. It can be speculated that improved anxiety scores are significant for this population, as decreased anxiety may serve as a protective factor for decreased risk for alcoholism relapse. The study examined outcome results 32 weeks after initial assessment, but did not look at longer-term outcomes in these patients. Future studies should examine longer-term treatment outcomes in co-morbid anxiety and substance use disorder patients to test the hypothesis that effective treatment of the co-morbid anxiety disorder serves a protective function in terms of risk for relapse to the substance use disorder in the longer-term.

Integrated Treatment Approaches. As briefly mentioned above, integrated treatment models recognize the complex relationship between anxiety disorders and alcohol use disorders and their possible mutual maintenance (Zahradnik & Stewart, in press). Furthermore, their aim is to create a hybrid of the treatments that work best for each disorder separately, and to also include in the treatment strategy an understanding of the reciprocal influences each disorder has on the other (Zahradnik & Stewart, in press). A sample integrated treatment was developed and tested for co-morbid panic disorder and alcohol use disorder by Kushner et al. (2006) (see also review in chapter 9). The treatment integrated CBT for panic disorder with content focusing on the interaction between alcohol use and panic symptoms. The integrated treatment was provided on top of treatment as usual (TAU) for the alcohol use disorder and compared to a group who received only the TAU. The trial showed promising results. The group receiving the integrated treatment showed better anxiety and alcohol outcomes than the TAU alcohol only treatment group. Furthermore, chapter 11 describes how personality-targeted treatments are promising as an early intervention model. Such interventions possess features of integrated treatments because they are designed and have been shown to indirectly impact both anxiety and substance-related symptoms by directly targeting a third variable, the underlying personality vulnerability.

It is hoped that integrated treatments will provide a more effective strategy in treating co-morbid anxiety disorder – substance use disorder patients. Integrated treatments appear to be the most recommended by 'expert opinion' and have been shown to be effective in the treatment of other patterns of co-morbidity (see review by Conrod & Stewart, 2005). However there have been relatively few randomized controlled trials, or even quasi-experimental designs,

testing the efficacy of truly integrated treatments in the anxiety – substance use disorder co-morbidity field (Watkins, Hunter, Burnam, Pincus, & Nicholson, 2005). This may be because there are some conceptual difficulties in fully integrating certain key aspects of each set of treatments (Conrod & Stewart, 2005). For example, one conceptual problem when integrating exposure-based treatment models for anxiety disorders with relapse prevention treatment for substance use disorder is that messages around exposure to high risk situations may be contradictory. This may not be a problem when CBT treatments for depression or bipolar disorders are integrated with relapse prevention models, due to greater theoretical overlap between CBT models for mood disorders and substance use disorders. Furthermore, even if a treatment could get around such conceptual incongruity, co-morbid clients may actually lack necessary coping skills required to tolerate or navigate through specific treatment components, such as exposure. More research is clearly needed to refine models of anxiety – substance use disorder co-morbidity in order to develop and test newer integrated treatment strategies. To conclude this section, we provide an example of how a classic cognitive behavioral model of substance use disorders (Marlatt & Gordon, 1985) could be adapted for treatment of substance use disorder clients with a co-morbid anxiety disorder, to facilitate further theoretical and empirical work in this area.

Adaptation of Marlatt and Gordon's (1985) Cognitive Behavioral Model to Understanding Co-Morbidity

Over two decades ago, Marlatt and Gordon (1985) developed a cognitive behavioral model to help explain the relapse process in substance abusers. This model has proven extremely useful in the prevention of relapse among treated substance abusers (see also Marlatt & Donovan, 2005). We present an adaptation of this model (Fig. 13.3) to highlight the how the presence of a co-morbid anxiety disorder can impact on each of the components of the relapse pathway in the original model.

The presence of a co-morbid anxiety disorder can impact the types of situations that are high risk situations for relapse. For those with co-morbid anxiety disorders, these are situations that are perceived as threatening in some way, although the precise situational triggers to heavy drinking/drug misuse may vary across the specific anxiety disorders. A patient with co-morbid panic disorder/agoraphobia is theoretically at high risk for relapse to heavy drinking/drug misuse in situations where he or she experiences feared bodily arousal sensations and situations where escape might be difficult or embarrassing if he or she were to have a panic attack. In contrast, a patient with social phobia is theoretically at high risk for relapse to substance misuse in social interaction or performance situations.

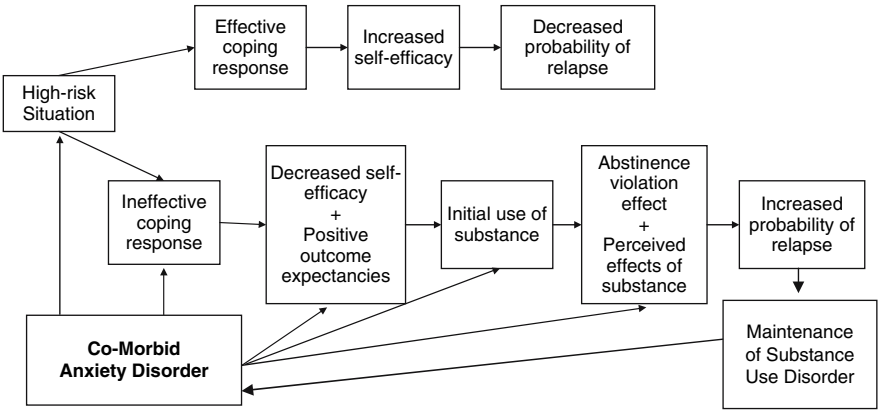

Fig. 13.3 Adaptation of Marlatt and Gordon's (1985) cognitive behavioral model of substance use disorder for explaining maintenance of anxiety disorder – substance use disorder co-morbidity [Adapted with permission from Marlatt, G.A., & Gordon, J.R. (1985). *Relapse prevention: Maintenance Strategies in the Treatment of Addictive Behaviors*. New York, Guilford Press (p.38)]. Reprinted with Permission of the Guilford Press

Those with co-morbid anxiety disorders are theoretically more likely than substance abusers without co-morbid anxiety disorders to choose ineffective coping responses to deal with the high risk (i.e., threatening) situations for relapse. This is particularly the case given the established tendency of anxious individuals to use avoidant coping strategies (Barlow, 2002). Substance misuse may simply be part of a more general pattern of avoidance of feared internal or external situations, among those substance abusers with co-morbid anxiety.

Co-morbid anxiety disorders can also impact the next step in the CBT model of relapse. Specifically, co-morbid anxiety can impact both self-efficacy (see review in chapter 4, this volume for the case of social phobia) and the particular positive outcome expectancies the substance abuser holds about the likely consequences of ingesting a given substance in this high risk situation. Across a variety of co-morbid anxiety disorders, general tension reduction expectancies may be particularly important predictors of substance use. But specific positive outcome expectancies may hold in the case of particular co-morbid anxiety disorders (e.g., social facilitation expectancies may be particularly important predictors of substance use for those with social phobia; see review in chapter 4). Recent data suggest that positive socially-related alcohol expectancies and low self-efficacy to avoid heavy drinking interact in predicting increased problematic drinking among socially anxious individuals (Burke & Stephens, 1997; Gilles, Turk, & Fresco, 2006).

Together their sensitivities to high risk situations involving threat, their tendency to engage in avoidant coping, their tendency to hold certain problematic positive outcome expectancies (e.g., tension-reduction expectancies), and their low self-efficacy to refrain from problematic substance use in high risk situations, make substance abusers with co-morbid anxiety much more likely

than other substance abusers to engage in an initial lapse to substance misuse when in a situation they find threatening. At this point, the individual crosses over the border from abstinence (or controlled use) to lapse (uncontrolled use episode) (Marlatt & Gordon, 1985). According to the original model, whether or not this initial lapse is followed by a total relapse depends largely on the patient's perceptions of the cause of the lapse and the patient's reactions to its occurrence. And again, it can be argued that substance use disorder patients with co-morbid anxiety are a highly vulnerable population for having an initial lapse turn into a full-blown relapse to their substance use disorder. This is because anxiety disorder patients have a cognitive tendency to catastrophize (Barlow, 2002) and they might thus be hypothesized to be likely to perceive the lapse as indicating a personal loss of control and a failure experience and thus to suffer guilt about the lapse incident. They are also theoretically more likely to notice and appreciate the desired negative reinforcing effects of the substance (at least with substances like alcohol or depressant/anxiolytic drugs). These factors would place a recovering substance abuser with a co-morbid anxiety disorder at heightened risk for continuing down the road toward relapse to a full-blown substance use disorder. Finally, we have added other components to the model (see Fig. 13.3), which accommodate the literature supporting the mutual-maintenance hypothesis, whereby anxiety symptoms can be further exacerbated by the substance use (see chapter 5).

The various predictions of this model could be tested in future research. If this adapted model is well supported though research, it can help explain why those substance abusers with co-morbid anxiety disorder seem to be at such elevated risk for relapse to substance abuse following treatment of their substance use disorder. This model would also have treatment implications in terms of providing several highly specific targets for therapy for co-morbid individuals in order to prevent substance abuse relapse. These targets would include not only each of the components in Marlatt and Gordon's (1985) original model, but also the co-morbid anxiety disorder which is seen to be driving the susceptibility to relapse at each level of the model.

Practical Issues in Treatment

There are several practical issues that need to be considered in the development and delivery of treatment approaches for co-morbid anxiety and substance use disorders. First and foremost are the challenges to integrated treatments presented by the continued tendency to separate addiction from mental health services in most health care delivery programs worldwide. This practice presents obstacles to the delivery of integrated treatments for co-morbid clients because practitioners in either service are typically not trained to assess and/or treat the other problem (i.e., addictions counselors rarely trained to identify and treat co-morbid anxiety disorders; mental health practitioners not usually trained to treat substance use

disorders). Parallel and sequential approaches require only that practitioners in each service be able to appropriately assess for the presence of a co-morbid substance use or anxiety disorder and then refer the co-morbid client to an expert for the treatment of the co-morbid disorder; in contrast, integrated approaches require that the practitioner also be appropriately trained in treatment of both disorders. It should be noted that it is theoretically possible that parallel approaches to treatment could accomplish many of the same goals as integrated treatments (e.g., simultaneously addressing the two inter-connected problems; decreasing the likelihood that the presence of the co-morbid disorder at the end of treatment of the 'index' disorder would serve as a risk factor for relapse of the 'index' disorder) provided that there is good communication between the two service providers (i.e., the 'case management' approach).

Nonetheless, even with excellent communication between service providers and outstanding coordination of services, there are still, theoretically, some limitations of the parallel approach relative to the integrated approach. For example, as we have noted elsewhere (Conrod & Stewart, 2005), parallel treatment requires the simultaneous provision of two empirically validated treatments and can be quite demanding of patients with complex problems, relative to a single integrated treatment. This factor may contribute to the high drop out rates observed in parallel approaches to treatment. As another example, relative to an integrated treatment delivered by a single well-trained therapist, parallel treatments create an artificial separation of the two disorders and fail to explicitly recognize and contend with the functional relations between the two disorders. This can be confusing to patients who perceive the symptoms of their anxiety disorder and substance use disorder to be functionally inter-related (e.g., Brown, Stout, & Gannon-Rowley, 1998; see also review by Stewart, 1996). Moreover, directly addressing the functional inter-relations between the two co-morbid disorders is a key ingredient in the treatment of co-morbid disorders from a theoretical perspective (Zahradnik & Stewart, in press). Finally, from an economic point-of-view, integrated treatments can be accomplished more efficiently than parallel treatments. Theoretically, then, integrated treatments should prove superior to parallel treatments which should prove superior to sequential treatments in terms of treatment outcome indices for both disorders and in terms of reducing risk for relapse of either disorder in the longer term. This hypothesis still requires empirical validation, however.

Another practical issue in treatment of co-morbidity concerns the increasing specialization required for the provision of empirically validated treatments for co-morbid patients. In general, the integrated treatments that are being developed and tested target very specific subtypes of co-morbidity (e.g., Kushner et al. [2006] integrated treatment for co-morbid panic disorder and alcoholism; Otto's et al. [1993] integrated treatment for benzodiazepine dependence in panic disorder's patients; Brady, Dansky, Back, Foa, and Carroll's [2001] integrated treatment of co-morbid PTSD and cocaine dependence). Although the establishment of clinics specializing in the integrated treatment of such very specific forms of co-morbidity are realistic in larger centers with an adequate population base,

such specialized services are not realistic in smaller centers where a smaller number of service providers must be prepared to deal with multiple forms of anxiety and substance use disorder co-morbidity. As more and more highly specific integrated programs are developed and validated, it becomes increasingly unrealistic to expect that service providers will be able to develop sufficient expertise in each new protocol to allow for efficient and effective service delivery (for an excellent discussion of this issue, see Kushner et al., 2006).

The personality-based approach described in chapter 11 provides a possible solution to this dilemma in that only two protocols would have to be mastered by clinicians (i.e., the anxiety sensitivity intervention which is relevant to many anxiety-related disorders and the introversion-hopelessness intervention which is relevant to certain anxiety disorders such as social phobia) in order to effectively treat the common factors contributing to various forms of co-morbid anxiety and substance use disorder. Nonetheless, as outlined in chapter 11, although this approach has been repeatedly been shown to reduce harmful substance use in both adults and adolescents (e.g. Conrod, Stewart, Comeau, & Maclean, 2006; Conrod et al., 2000; Watt, Stewart, Birch, & Bernier, 2006), more work is needed on establishing the efficacy of this approach in treating symptoms of the co-morbid anxiety disorder. An exception is a recent study by Castellanos and Conrod (2006) where it was shown that the anxiety sensitivity intervention was also effective in reducing panic attacks in youth.

There is also a movement afoot in the anxiety disorders field toward the development of more global 'broad-band' types of interventions that are effective in the treatment of anxiety disorders in general rather than focusing narrowly on protocols designed to treat only one anxiety disorder in particular (see Barlow, Allen, & Choate, 2004). For example, Westra and her colleagues have developed, manualized, and evaluated such a broad-band approach for the treatment of panic disorder with or without agoraphobia, social phobia, and generalized anxiety disorder patients. This treatment approach is 10 sessions in duration, is delivered in a group context, and includes psychoeducation, cognitive restructuring, and exposure components (see Westra, Dozois, & Marcus, 2007; Westra, Stewart, & Conrad, 2002, for program description and efficacy data). For practical reasons, it would be useful for future research to focus on the development and evaluation of a 'broad-band' integrated intervention designed to treat a variety of forms of anxiety disorder – substance use disorder co-morbidity. For example, the anxiety management protocol developed by Westra et al. (2002, 2007) could be adapted for suitability to the co-morbidity context in the same manner that Kushner et al. (2006) adapted Barlow and Craske's (2000) 'narrow-band' CBT for panic disorder for suitability as an integrated treatment for panic disorder – alcohol abuse co-morbidity. In fact, in their attempt to develop and test an integrated CBT for panic and alcohol dependence, Kushner et al. (2006) identified that an additional obstacle to implementing such a program was the presence of multiple co-occurring anxiety disorders within this co-morbid population. They, too, suggested that perhaps a broad-band integrated approach may be more suitable

for the treatment of co-morbid anxiety and substance use disorders. Future research would be needed to determine how much is sacrificed in terms of efficacy when moving to a more broad-band approach to co-morbidity treatment relative to the narrower protocols being investigated to date. And this would need to be weighed against the increased reach and efficiency of services that could be achieved with the broad-band approach.

A final practical issue pertains to applications of Prochaska and DiClemente's (1992) stages of change model to the treatment of anxiety disorder – substance use disorder co-morbidity. If a given co-morbid patient presenting for treatment is at an advanced stage of readiness for change for one disorder (e.g., in the action stage for the anxiety disorder) but at a very early stage of readiness for change for the (e.g., precontemplative for the substance use disorder), it would be tempting to take this as support for a sequential approach to treatment. Should therapy not simply focus first on the disorder where the patient is evidencing the greatest readiness to change? One must still consider the mutual maintenance model depicted in Fig. 13.2 and the revision of Marlatt and Gordon's (1985) model depicted in Fig. 13.3. Failure to simultaneously address the two disorders can leave the patient vulnerable to relapse of the treated disorder. But from a practical perspective, a therapist can engage the client in initially beginning to address the disorder where readiness to change is greatest, while providing psycho-education around the functional relations between the two disorders. Use of a motivational interviewing approach (Miller & Rollnick, 1991) can be helpful to work toward enhancing readiness to simultaneously address the co-morbid disorder through a more integrated approach to treatment. Indeed, this type of approach has been applied effectively to the treatment of co-morbid substance use and psychotic disorders (Barrowclough et al., 2001), but awaits formal evaluation in terms of its applicability to treatment of co-morbid anxiety and substance use disorders.

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