The Role of Negative Affect in Eating Disorders and Substance Use Disorders

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Abstract

Affect regulation is one mechanism that has been implicated in the development and maintenance of both eating disorders and substance use disorders. Specifically, the affective processing model of negative reinforcement posits that negative affect, as a symptom of withdrawal, is the main impetus in substance use disorder development and maintenance. Similarly, a recent transactional model of emotion dysregulation posits that individuals with eating disorders display heightened emotional sensitivity and reactivity, which in turn predisposes these individuals to eating disorder behaviors (e.g., binge eating, purging, etc.) as a means of attempting to modulate heightened negative affect. While affect regulation is similar in eating disorders and substance use disorders, differences in precursors of negative affect, cognitions, and withdrawal symptoms are present in these two forms of psychopathology. Despite these differences, affect regulation models in both eating and substance use disorders have begun to influence treatment. Thus, understanding the role of negative affect may be a key component of treating substance use disorders and eating disorders independently, as well as the co-occurrence of these disorders.

Keywords

Affect regulation • Emotion dysregulation • Eating disorders • Substance use disorders

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T.D. Brewerton and A.B. Dennis (eds.), *Eating Disorders, Addictions and Substance Use Disorders*, DOI 10.1007/978-3-642-45378-6_16,

16.1 The Role of Negative Affect in Eating Disorders and Substance Use Disorders

Substance use disorders (SUD) and eating disorders (ED) share many clinical similarities. For example, both disorders typically begin in adolescence or early adulthood, include behaviors which may function to maintain the disorder despite harmful consequences, have a high tendency to relapse, and alter the way the individual relates to others (Goodman, 2008). Such similarities suggest that these two disorders may share common mechanisms of development and maintenance. Accordingly, several etiological models have been presented that may explain the co-occurrence of ED and SUD, including neurobiological (see Chap. 3), personality (see Chap. 6), and genetic (see Chap. 5) theories which highlight factors common to both disorders. While each of these theories elucidates possible mechanisms of etiology, they often fail to identify common mediating variables which may clarify the process of SUD and ED development (Harrop & Marlatt, 2010). Affect regulation is one mechanism that has been implicated in the development and maintenance of both ED (Haynos & Fruzzetti, 2011) and SUD (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004) and has been identified as a mediator of both disorders (Gadalla & Piran, 2007; von Ranson, McGue, & Iacono, 2003). Thus, understanding the role of negative affect may be a key component of treating co-occurring SUD and ED.

Examining affect regulation as a possible mechanism of both SUD and ED broadly fits the conceptualization typically referred to as the self-medication hypothesis of SUD. Simply stated, the self-medication hypothesis posits that individuals use drugs or alcohol in an attempt to relieve symptoms of mental disorders (Khantzian, 1985). The simplicity of this hypothesis is broadly appealing but fails to identify specific biobehavioral processes that substance use is thought to influence (Henwood & Padgett, 2007; Lembke, 2012). Negative affectivity may be a clinically and scientifically useful behavioral process to account for the commonalities between SUD and ED, and alleviation of negative affect has been thoroughly researched as a vital component of both SUD (Baker et al., 2004; Measelle, Stice, & Springer, 2006) and ED (Engel et al., 2013; Haynos & Fruzzetti, 2011; Smyth et al., 2007).

This chapter will review two clinically oriented models that include affect as a key construct. First, we will review the role of affect regulation as a key component of a negative reinforcement model of SUD (Baker et al., 2004). Second, we will discuss a model that posits that emotion dysregulation is key in understanding the development and treatment of ED (Haynos & Fruzzetti, 2011). We will focus our discussion of each model on relevant research with a particular emphasis on ecological momentary assessment (EMA) studies. EMA entails multiple assessments conducted "in the moment" in a naturalistic environment (Shiffman, Stone, & Hufford, 2008). The main advantages of this type of data collection are that EMA avoids the problems associated with retrospective recall bias in self-report assessments when they are not conducted in real time and allows detailed study of antecedents and consequences of behavior, which is important for the study

of affect regulation. Moreover, EMA has been successful in examining relationships among affect in SUD (Shiffman, 2009) and ED (Smyth et al., 2007). Finally, we will compare and contrast the SUD and ED literature that has examined the role of affect in the etiology of these disorders.

16.2 Substance Use Disorders

Negative affect regulation has been a widely studied antecedent to substance use and has been included in multiple models of SUD development and maintenance. The initial models that included affect as an explanatory variable suggested that withdrawal syndrome symptoms, including negative affect, appear after a single or very few instances of substance use. Consequently, as the level of the ingested substance begins to fall, withdrawal symptoms increase, and subsequent substance use represents an attempt to manage or alleviate such symptoms (Hull, 1943). Subsequent models have continued to consider the link between negative affect and substance use. For example, the fundamental assertion of Solomon's (1980) opponent-process model of SUD is that every behavioral process has a pleasant or unpleasant affective valence which is followed by an opponent process. Thus, substance use (A process) produces a pleasurable affective state, but when withdrawal symptoms including negative affect (B process) increase, the substance use is repeated to maintain the magnitude of the A process over the B process. Moreover, negative affect is also implicated in theories that emphasize external factors and cues to substance use. For example, social learning theory posits that when negative affect is high, self-efficacy for dealing with external problems (e.g., interpersonal stressors, etc.) is low. Consequently, substance use to alleviate underlying tension may be triggered, in part, by self-regulatory-related expectancies regarding an inability to inhibit drinking (Jung, 1994).

The inclusion of negative affect in each of these theories suggests that affect regulation is a robust phenomenon that may play a pivotal role in SUD etiology. Interestingly, few models have focused primarily on affect regulation as the key factor in understanding the development of SUD. One notable exception, however, is the affective processing model of negative reinforcement (Baker et al., 2004). Simply stated, this model conceptualizes the etiology of SUD in the following four steps. First, the initial instance of substance use is performed in an attempt to experience the desirable effects elicited by the substance (e.g., drinking alcohol produces calming and euphoric effects). Second, as the amount of substance ingested is metabolized and eliminated by the body, unpleasant symptoms of withdrawal begin to present themselves (e.g., hangover symptoms such as anxiety, nausea, headache, etc.). Third, the individual wants to alleviate these unpleasant withdrawal symptoms and therefore uses the substance again in an attempt to elicit the pleasurable effects previously experienced (e.g., the individual drinks more alcohol (aka "hair of the dog") to feel better). Fourth, the individual learns that substance use alleviates the unpleasant withdrawal symptoms and may also alleviate other similar unpleasant experiences (e.g., drinking in response to negative

affect resulting from interpersonal conflicts). Thus, this model posits that symptoms of withdrawal syndrome act to motivate continued substance use and that negative affect is the primary symptom responsible for this process.

Overt physical symptoms of withdrawal (e.g., tremors, insomnia, shaking, etc.) are commonly associated with SUD. Including these symptoms in explanations of SUD becomes problematic because each class of substance may produce very different physical symptoms during withdrawal. In other words, withdrawal symptoms seem to be specific to the type of substance used (WHO, 1992). Therefore, any etiological explanation that includes withdrawal symptoms must focus on symptoms that are universal to all substance use. Negative affect is one symptom that is universal to all substance use (Marlatt & Gordon, 1980). Thus, the universality of negative affect as a withdrawal symptom is the crucial supposition of the affective processing model of negative reinforcement.

While negative affect in general is identified as the key component of Baker and colleagues' (2004) model, it may be more appropriate to say that the intensity and awareness of negative affect are the key considerations in understanding SUD. Specifically, negative affect is a symptom of withdrawal that is likely to be present after the very first substance use episodes. At this point in the development of substance use problems, the level of awareness of negative affect is most likely very low, often only at a preconscious level of awareness, and acts primarily as an interoceptive (internal) cue. For example, an individual may not consciously recognize their current unpleasant emotional state as negative affect resulting from substance use, but is able to detect that additional substance use alleviates this feeling. Furthermore, negative affect intensity may increase and enter conscious awareness as a result of abstinence from substance use or as a consequence of other general stressors (external cues). Thus, the negative affect may be increased by the combination of interoceptive and exteroceptive cues, which increases the number of cues for substance use.

Baker and colleagues (2004) present three main arguments for why the rise in negative affect intensity is a key component to understanding its role in SUD development and maintenance. First, negative affect intensity is related to hot and cool processing of motivational reasoning (Metcalfe & Mischel, 1999). Simply stated, cool processing is best described as a nonemotional basis for motivating behavior, while hot information processing is influenced by emotional memories and less able to be modified by declarative statements (Öhman & Mineka, 2001). Cool processing predominates at low levels of awareness of negative affect but gives way to hot processing as negative affect awareness and intensity increase. Thus, as negative affect intensity increases, the individual not only becomes more aware of the unpleasant emotional state they are experiencing but also recalls the memory of the pleasant emotional state induced by previous substance use episodes. Consequently, the individual is motivated to use substances by the emotional memory of the pleasurable state experienced during previous substance use episodes and the expectancy that it will produce the same outcome.

Second, Baker and colleagues (2004) argue that affect intensity influences an individual's ability to maintain information, set and accomplish goals, make plans,

and follow instructions. Tasks such as these are referred to as cognitive control. At very low levels of negative affect, the individual is not influenced by affective state, and cognitive control is not necessary to prevent the engagement in substance use. Alternatively, when negative affect is very high, cognitive control is impaired because information processing is negatively impacted by emotional factors which influence adaptive decision making. Thus, one major difference between low and high levels of awareness of negative affect is that cognitive control is significantly impaired by the appetitive emotional value of the substance being used at high levels of negative affect.

Third, substance use is not an entirely individual activity. For example, awareness of future unavailability of a drug, social cues for substance use, and interpersonal stressors are largely related to factors that are out of the control of an individual. These motivations for substance use are referred to as modulators. Baker and colleagues (2004) state that at low levels of negative affect, the use of substances tends to be proceduralized and at high levels decision making is based primarily on emotional factors. That is, the individual may be unaware of the external motivational impetus for substance use at low and high levels of negative affect but is aware of the availability of drugs. Therefore, individuals may be most susceptible to modulators at moderate levels of negative affect.

The affective processing model presents the fundamental groundwork to argue that SUD are learned responses to withdrawal-induced negative affect cues which produce a negatively reinforcing reduction in negative affect. Motivation for substance use may arise from interoceptive (e.g., recognition of increased negative affect) or exteroceptive (e.g., modulators) sources. As the unpleasant stimuli are presented, substances are used to ameliorate negative affect. Central to this learning process is that negative affect, whether arising from withdrawal or other external factors unrelated to substance use, is reduced when substance use is performed. This reduction of unpleasant affect is the consequence that would be thought to maintain the disorder. In other words, it is rewarding.

16.3 Ecological Momentary Assessment Research in Substance Use Disorders

Research cited by Baker and colleagues (2004) to support each aspect of the affective processing model of negative reinforcement provides a sound justification supporting the model and adequately addresses many criticisms of affective regulation models. However, recent advances in EMA may provide another method of elucidating the role of affect in SUD (Shiffman, 2009). Interestingly, EMA studies of smoking, alcohol, and other substances have provided new insights into affect regulation that may be specific to each type of substance.

Cross-sectional and retrospective designed studies have consistently supported the role of negative affect regulation in smoking studies (Shiffman, 1993), in spite of some inconsistency (Shiffman, 2009). More recent EMA research has indicated that mood and craving predicted smoking 4 h prior to the behavior, but not 2 h prior

(Berkman, Dickenson, Falk, & Lieberman, 2011), and that smokers that abstained from smoking on their chosen quit day experienced greater increases in negative affect than those who failed to quit (Yeh, McCarthy, & Baker, 2012). Collectively, these EMA studies may point to temporal relationships among affect and smoking that are currently not fully understood.

Similarly, EMA research examining alcohol use has yielded encouraging results that may support the role of affect regulation in substance use. The EMA literature appears to consistently suggest that affect regulation may play a role in several different drinking scenarios (1) when negative affect is experienced early in the week and drinking occurs on the weekend, (2) negative affect is increased early in the day and drinking occurs later that same evening, and (3) drinking is assessed using binge-drinking criterion or only first drink of a drinking episode as the operational definition of drinking (Shiffman, 2009). Moreover, EMA studies that have looked at drinkers who also smoke have found that alcohol is appraised as more negatively reinforcing than cigarettes and that drinking, but not smoking, decreases negative affect (Piasecki et al., 2011). Thus, Baker and colleagues' (2004) associations of affect regulation and negative reinforcement-based learning may be supported.

EMA examinations of other substance use have also revealed interesting findings. Specifically, Carrico and colleagues (2013) report that negative affect was not associated with any measure of methamphetamine use, but positive affect was associated with coping and self-efficacy for managing drug use. Similarly, negative affect is not directly associated with cocaine and heroin use, but is associated with temptation to use (Waters, Marhe, & Franken, 2012), stress preceding use (Preston & Epstein, 2011), and a gender difference indicating women experience greater negative affect after use than do men (Kennedy, Epstein, Phillips, & Preston, 2013). Finally, in a series of EMA studies, Buckner, and colleagues (Buckner, Crosby, Silgado, Wonderlich, & Schmidt, 2012; Buckner, Crosby, Wonderlich, & Schmidt, 2012; Buckner, Zvolensky, & Eckera, 2013; Buckner et al., 2011) report that social anxiety predicted cannabis use. The motivational role of modulators and exteroceptive factors outlined in Baker and colleagues' (2004) model may also be supported by Buckner, Crosby, Wonderlich, and Schmidt (2012) finding that the presence of other cannabis users facilitated marijuana use.

Taken together, SUD studies that have employed EMA designs have found support for the basic assertion that affect regulation plays a pivotal role in substance use while also suggesting some differences that were previously unable to be detected with cross-sectional or retrospective designs. Recent advances in statistical analyses, mobile technology such as GPS-enabled smartphones, and the ability to collect real-time physiological data may advance our understanding of the nuances of affect regulation in SUD (Shiffman, 2009). Thus, EMA designs have the potential to further evidence the associations identified by previous studies and advance our understanding of SUD through better measurement of variables that may not yet be included in conceptual and/or theoretical models. Future EMA research is encouraged to continue exploring and examining the role of affect in SUD.

16.4 Negative Affect in Eating Disorders

A substantial body of empirical research suggests that negative affect is an important factor to consider in relation to ED psychopathology. For example, evidence from both population-based studies and research using clinical samples suggests that mood and anxiety disorders commonly co-occur with ED (Hudson, Hiripi, Pope, & Kessler, 2007; Kaye, Bulik, Thornton, Barbarich, & Master, 2004). Furthermore, beyond diagnostic co-occurrence, evidence suggests that those with ED tend to display elevated subthreshold symptoms of anxiety and depression (e.g., Kaye et al., 2004; Wagner et al., 2006), as well as higher levels of various forms of negative affect (e.g., anger, guilt, hostility; Allen, Scannell, & Turner, 1998; Waller et al., 2003).

Additional support for the relevance of negative affect to ED is provided by research suggesting that those with ED display a pattern of co-occurring personality disorders and traits associated with negative emotionality, including borderline personality disorder features and obsessive-compulsive personality disorder features, as well as broader personality constructs such as neuroticism (e.g., Cassin & von Ranson, 2005). Finally, numerous recent studies have focused on examining emotion-based constructs that are highly associated with negative affect. Among these are negative urgency, a construct defined by the tendency to act rashly in the face of negative affect (Cyders & Smith, 2008), and distress tolerance, which refers to the ability to experience and tolerate negative affective states (Simons & Gaher, 2005). Consistent with the findings noted previously, evidence suggests that elevated negative urgency and poor distress tolerance are associated with ED symptoms, particularly binge eating (Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007; Fischer, Settles, Collins, Gunn, & Smith, 2012). Taken together, these distinct but intersecting lines of research point to the importance of considering the role of negative affect in the etiology, maintenance, and treatment of ED.

The specific role of negative affect in ED has been emphasized to varying degrees in existing etiological/maintenance and treatment models. For example, the escape theory of binge eating (Heatherton & Baumeister, 1991) posits that binge eating behavior is motivated by a desire to divert attention from the negative affective experience associated with aversive self-perceptions. In another model that is focused on a more specific facet of negative affect, Strober (2004) highlighted the role of fear and anxiety in AN, suggesting that individuals with AN exhibit a propensity toward anxiety and fear learning processes, as well as a greater resistance to extinction of learned fear responses. Additionally, although emotion processes are not a primary focus in the model, Fairburn, Cooper, and Shafran's (2003) transdiagnostic theory of ED maintenance addresses the role of mood intolerance, particularly with regard to associations with binge eating and compensatory behaviors.

Numerous models of ED psychopathology thus address the role of negative affect. Accordingly, numerous recent emotion-based psychotherapeutic treatments for ED have emerged. Included among these interventions are dialectical behavior therapy (DBT; Safer, Telch, & Chen, 2009), integrative cognitive-affective therapy

(ICAT; Wonderlich et al., 2014), and emotion acceptance behavior therapy (EABT; Wildes & Marcus, 2011). Each of these interventions focuses heavily on the role of negative affect in precipitating and maintaining ED behaviors, with a particular emphasis on providing skills for managing negative affective states. In sum, existing theoretical and treatment models of ED psychopathology vary to some extent in the role or relative emphasis on negative affect, although more recent theoretical models and emerging treatments focus more heavily on the importance of negative affect and related emotion constructs.

16.5 Emotion Dysregulation Model of Eating Disorders

Although several of the ED psychopathology models discussed above address negative affect as a relevant factor, a recent model proposed by Haynos and Fruzzetti (2011) provides a useful framework for conceptualizing the role of negative affective states and related emotion constructs in ED. This proposed transactional model of emotion dysregulation focuses specifically on AN. However, it can also be applied to ED more broadly, and thus the discussion of the model here will focus on ED psychopathology as a whole. The basic theory of this model is that individuals with ED display an underlying emotional vulnerability characterized by elevated emotional sensitivity and reactivity, which in turn predisposes these individuals to frequently experience elevated emotional arousal in response to various stimuli (e.g., cognitions, environmental stressors, etc.). This frequent experience of heightened negative affect promotes ED behaviors (e.g., binge eating, purging, etc.) as a means of attempting to modulate affect, thus functioning as a form of maladaptive emotion regulation. Additionally, variables are also posited to influence the underlying emotional vulnerability. Specifically, both weight loss/starvation state and invaliding responses from others (which result due to deficits in emotional expression by the eating disordered individual) are also proposed to contribute to and exacerbate emotional vulnerability, thus perpetuating the cycle and maintaining ED symptoms over time. The focus of this brief review of the literature relevant to this model will be on the primary emotion components within the model (for further explanation of the inaccurate expression and invalidating environmental components of the model, see Haynos & Fruzzetti, 2011).

There is growing evidence suggesting that individuals with ED display a broad pattern of deficits in various dimensions of emotion regulation (Harrison, Sullivan, Tchanturia, & Treasure, 2010; Racine & Wildes, 2013), and existing empirical findings lend support to the various associations addressed in the Haynos and Fruzzetti (2011) emotion dysregulation model of ED psychopathology. For example, evidence for an underlying emotional vulnerability can be found in research suggesting that those with ED display an attentional bias to emotionally evocative cues (Shafran, Lee, Cooper, Palmer, & Fairburn, 2007), individuals who binge eat display greater fluctuations in anxiety and depression than those who do not binge eat (Lingswiler, Crowther, & Stephens, 1989), and days characterized by

fluctuating levels of negative affective states are common in bulimia nervosa (BN; Crosby et al., 2009) and anorexia nervosa (AN; Lavender et al., 2013).

Additionally, as noted previously, the presence of heightened negative emotional arousal in ED is evidenced by the high rates of co-occurring affective disorders (Hudson et al., 2007; Kaye et al., 2004), as well as elevated negative affective states and symptoms (Allen et al., 1998; Wagner et al., 2006; Waller et al., 2003). Perhaps most importantly, an extensive literature supports an association between these negative affective states and ED symptoms, particularly behaviors such as binge eating, purging, dietary restriction, etc. For example, negative affect has been identified as a common antecedent for binge eating behaviors (Haedt-Matt & Keel, 2011), and evidence suggests that the likelihood of various ED behaviors tends to coincide with elevations in negative affect and anxiety (Crosby et al., 2009; Lavender et al., 2013). Furthermore, studies utilizing mood induction techniques in conjunction with feeding laboratory paradigms also provide evidence for an association between negative affect and ED behavior (Telch & Agras, 1996). Finally, consistent with the Haynos and Fruzzetti model, several studies also suggest that negative affect may decrease (at least temporarily) subsequent to certain ED behaviors (Smyth et al., 2007; Engel et al., 2013), although this remains a source of debate (Haedt-Matt & Keel, 2011).

16.6 EMA Studies of Negative Affect in Eating Disorders

EMA methods have been applied in a variety of ED studies, with many studies focusing on elucidating the associations between negative affect and ED behaviors. As noted previously, EMA methods have the benefit of reducing retrospective recall biases, contrasting with standard assessments that rely on recall of ED behaviors over an extended period of time (e.g., 28 days in eating disorder examination, the gold-standard interview-based assessment of ED psychopathology; Fairburn, 2008). Additionally, the collection of momentary emotion and behavior data across multiple time points within a day, as well as across days, allows for a more precise examination of the temporal nature of the emotion-behavior association in ED. The following is a brief review of findings regarding the association between negative affective states and ED symptoms derived from studies that utilized EMA.

The majority of EMA studies in the ED literature have been conducted with samples of individuals with BN, binge eating disorder (BED), or related groups (e.g., obese individuals who binge eat). In a sample of individuals with subclinical binge eating behavior who completed a 2-week EMA protocol, Wegner and colleagues (2002) found that several indices of negative affect were worse on days during which a binge eating episode occurred versus days in which no eating binges occurred. In a study that included both women with BN and women with BED, Hilbert and Tuschen-Caffier (2007) found that both ED groups reported a more negative mood prior to binge eating than prior to regular eating. Furthermore,

pre-binge mood was worse among those with BN compared to the BED group, and both ED groups were found to display a worse mood subsequent to the binge.

In another study, a sample of women with BN completed a 2-week EMA protocol (Smyth et al., 2007). Results revealed that negative affect was higher on days in which binge eating or self-induced vomiting episodes were reported. Furthermore, a trajectory of increasing negative affect prior to binge eating and vomiting behaviors was observed, while a trajectory of decreasing negative affect subsequent to the behaviors was found. A recent study using data from this study reported on specific facets of negative affect (fear, guilt, hostility, sadness) in relation to the occurrence of binge eating and purging behaviors (Berg et al., 2013). Results revealed a similar pattern of increasing negative affect facets prior to both binge eating and purging, and the facet of guilt in particular was found to increase prior to and decrease subsequent to the behaviors, even when controlling for the other negative affect facets. Finally, Crosby and colleagues (2009) examined types of days characterized by patterns of negative affect in the same sample of women with BN. Results revealed nine distinct types of days that were characterized by different patterns of negative affect (e.g., stable high negative affect across the day, negative affect that increased late in the day, negative affect that decreased late in the day, etc.). The likelihood of binge eating and purging behaviors was found to differ between days characterized by stable low negative affect and several other types of days, providing further evidence for an association between negative affect and ED symptoms.

More recently, studies have also used EMA methods to examine associations between emotion and ED behaviors in AN. In one of the earliest such studies, Engel and colleagues (2005) reported on data collected from 10 women with AN who completed 2 weeks of EMA. Results revealed substantial variables of mood across individuals, as well as across the day within individuals. Mood variability was also found to be positively associated with restrictive and ritualistic eating behaviors. In a larger, more recent study using EMA in a sample of women with AN, Engel and colleagues (2013) examined negative affect antecedent and consequent to a variety of ED behaviors using two methods. Using a method in which multiple data points prior to and following the behaviors were used to model pre- and post-behavior trajectories of negative affect, significant increases in negative affect were observed prior to loss of control eating, purging, and weighing, while significant decreases in negative affect were found following the behaviors. However, using only a single rating immediately before and after the behaviors, results revealed a significant increase in negative affect after loss of control eating, purging, and weighing, while a significant decrease in negative affect was observed after exercise and drinking fluids to curb appetite. These disparate findings thus suggest that further research is necessary to clarify the affective changes that occur in response to ED behaviors.

Finally, in a second study using data from the same EMA study of women with AN, Lavender and colleagues (2013) examined daily patterns of anxiety in relation to the occurrence of various ED behaviors. Results revealed seven distinct types of days characterized by varying patterns of anxiety across the course of the day. Certain ED behaviors (e.g., binge eating, vomiting, dietary restriction) were found

to differ between days characterized by stable low anxiety and other types of days. Furthermore, an examination of the timing of ED behaviors within each type of day revealed that frequently, ED behaviors were more likely to occur during times of elevated anxiety. Taken together, results from the small but growing body of literature comprised of EMA studies with AN samples provide support for a similar association between emotion and ED symptoms that have been found in other ED samples.

Concluding Comments on Affect Regulation in Eating and Substance Use Disorders

We have provided a brief review of affect regulation theory and research in both SUD and ED. Clearly, there has been scientific and clinical growth in this topic in both areas of psychopathology. However, there are also differences between the studies of affect regulation in these two distinct areas of psychopathology. For example, precursors of negative affect in the substance use domain are significantly influenced by withdrawal tied to the drug of use or abuse. As Baker and colleagues (2004) point out, it is the negative affect emanating from the withdrawal experience which appears to serve as the critical antecedent for the behavioral use of the substance. On the other hand, the concepts of withdrawal in ED literature are not well understood or developed. Other than some understanding that starvation may impact affect (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950), the origins of negative emotional experience in the ED seem to reside more in the interface of the individual's environment and their internal propensities and resources (e.g., personality and coping resources). Clearly, a careful consideration of the precipitants of negative affect in both ED and SUD may help to integrate and advance this field of study. For example, it is unclear how an inability to engage in ED behaviors may produce a "withdrawal-like" phenomenon in ED which is similar to SUD. Similarly, the contributions of environmental- and person-based factors in the elicitation of negative affect and its role as a precipitant of episodes of substance abuse remain unclear. Baker and colleagues (2004) acknowledge the role of modulators in their theory, but this aspect of affect generation in the model seems less well developed than negative affectivity associated with withdrawal from substances.

Another factor to consider in the comparison and contrast of SUD and ED in terms of affect regulation is the role of cognitive processes. Much research in ED has focused on cognitions surrounding eating, shape, and weight concerns. This comes out of a significant influence of cognitive-behavioral therapy in ED (Fairburn, 2008) and has resulted in several empirically supported treatments which focus on cognitive factors. An alternative approach to cognition, which is seen in the substance use literature, is the consideration of cognitive constructs, such as expectancy. Specifically, expectancies regarding the possible function of engaging in the use of substances at any given point in time have been shown to be a significant predictor of substance use episodes (Fisher, Smith, Anderson, & Flory, 2003; Fulton, Krank, & Stewart, 2012). For example, the expectation that a certain substance will improve negative mood has been shown to be a robust

predictor of engagement in utilization of that substance (Kuntsche, Knibble, Gmel, & Engels, 2005). Paralleling this literature, there has been an increased consideration of expectancies in ED behavior (Fischer et al., 2012). In particular, there is some evidence that expectations that dieting will result in thinness have a powerful impact on anorexic-like behaviors. Similarly, the expectation that binge eating will reduce negative mood has been found to be associated with bulimic-type presentations (Hohlstein, Smith, & Atlas, 1998). Clearly, in both areas of psychopathology, a consideration of the role of the specific types of cognition needs to be carefully considered to provide a truly integrative and comprehensive affect regulation model.

Finally, affect regulation models in both the ED and SUD have begun to influence treatment. For example, recent empirical work in the SUD suggests that mindfulness-based approaches to treatment (e.g., dialectical behavior therapy, DBT) reduce the use and abuse of substances by patients (Dimeff & Linehan, 2008). Similarly, broad-based cognitive-behavioral strategies which emphasize affect regulation have also been shown to have promise in treating individuals with SUD (Kadden, Carbonari, Litt, Tonigan, Zweben, 1998). Similar observations could be made about ED treatment. For example, both mindfulness-based approaches (Kristeller, Baer, & Quillian-Wolever, 2006) and DBT-oriented approaches have been found to have efficacy in ED treatment (Safer et al., 2009). However, there are also other emerging ED treatments that have evolved more directly from explicit affect regulation models of ED psychopathology. For example, emotional avoidance behavior therapy (EABT; Wildes & Marcus, 2011) and integrative cognitive-affective therapy (ICAT; Wonderlich et al., 2014) are examples of new ED treatments emerging explicitly from affect regulation theories of ED behavior. Future treatments that target factors which increase affective intensities or affect dysregulation, promote greater awareness and tolerance of emotional states, and support inhibition of impulsive or reckless behaviors when affectively aroused hold considerable promise for treatments in both SUD and ED domains.

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