Anger and Stimulant Use in the Context of Addiction Treatment: From a Clinical Perspective

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In treatment settings, psychological distress due to displacement from substance use disorder patients’ standard routines, natural environment, loss of social supports, and removal of substances as a primary coping strategy can leave recovering “addicts” in a feedback loop of rumination and negative affective states. Thus for a portion of substance users, placement in treatment is a psychoemotional provocation that renders vulnerability to depletion of self-control associated with both increased anger and aggression (Denson, Pedersen, Friese, Hahn, & Roberts, 2011). Dependence and withdrawal symptoms such as restlessness, dysphoria, anxiety, irritability, stress, physical pain, lethargy, sleep disturbance, short-temperedness and moodiness can also cause anger and drive impulsive aggression.

In the context of the self-medication hypothesis (Khantzian, 1985, 1997), anger has a possible adaptive and functional role in stimulant use addiction and treatment recovery. The hypothesis suggests that cocaine and methamphetamine use share common reinforcing mechanisms with anger. Anger can therefore play a role in ameliorating the effects of addiction and withdrawal, and alleviating feelings of sadness, fear, guilt, self-doubt, embarrassment, shame, etc. Specifically, the mood-altering class of chemical stimulants that amplify central nervous system activity is the “drug of choice” to dissipate physical bodily sensation and psychological distress derived from pain, anhedonia, depression, and dysphoric emotional states of

Summary

While research does not support that substance use disorder patients have higher levels of trait anger than the general population, a subset of patients are likely to experience problems dealing with anger in treatment settings. The expression of anger may serve a self-protective function. Anger may also lead to heightened awareness, decision-making, or action planning. Patients may find it easier to function with anger because it makes them feel more in control relative to feeling impotent and weak.

Keywords

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of discomfort such as anergia or low self-worth (Alcaro, Huber, & Panksepp 2007; Moal & Koob, 2007). Thus, strong feelings of anger may be a neurobiological and physiological psychoactive stimulant for patients in addiction recovery, allowing for sensations of liveliness, excitement, pleasure, and even normalcy.

Anger’s self-reinforcing effects also provide a framework to understand why patients hold onto false beliefs that influence further expressions of anger and rationalize its consequences. For example, patients in treatment may believe (a) that venting anger is therapeutic, (b) that anger tendencies are always experienced at a conscious level and under complete volitional control of willpower, (c) that intense anger can cause a state of blackout, and (d) that their own anger genes would override any conscious choice in how it is expressed.

The cognitive appraisal of an event may support and influence the experience of anger and correlate with the expression of impulsive aggression. Thus patients’ belief systems can lead them to misinterpret anger management techniques as actually encouraging and perpetuating anger. This can plausibly lead a subgroup of patients to lose sight of the self-control strategies of anger management because they can seem meaningless, irrelevant, and not instantly gratifying.

In assisting individuals in substance use recovery, we have noted that anger often accompanies the initial stages of treatment. In an effort to determine what role anger might play in problem substance use and its treatment, we looked to the existing literature for insight. The goal of this analysis is to conceptualize the various ways anger manifest among cocaine and amphetamine users, and how a sample of the population comprehends that subjective experience. Hypotheses are offered from a clinical perspective underpinned by experiential observations, which correlate socio-psychological aspects of drug addiction treatment with neurophysiological facets of anger.

**Socio-Psychological Level of Analysis**

*Venting Anger Out of the System*

Suppressed anger can have negative consequences (Bridewell & Chang, 1997; Kassinove & Tafrate, 2002; Okifuji, Turk, & Curran, 1999). Angry expression that displays affect but finds basis in principles of assertive communication (i.e., venting) can be beneficial, by facilitating the processing of emotions and addressing its underlying sources (Busch, 2009; Kassinove & Sukhodolsky, 1995). Assertive venting, then, may positively effect a reduction in the intensity of anger and diminish other obstructive emotional states leading to a cathartic state in the therapeutic process (Busch, 2009).

However, it is not unusual for patients early on in their recovery, especially those with pre-existing “anger problems,” to aggressively express emotions. This is partially because (a) aggression is colloquially considered an effective anger management strategy, (b) patients may view less severe angry reactions (name-calling instead of lashing out) an accomplishment, and (c) patients may feel better afterwards. Overly aggressive expressions may feel good because it temporary lifts one’s mood, reduces tension and stress, and blunts physical pain (Bruehl, Chung, Burns, & Biridepalli, 2003). However, since aggressive expression can also promote endogenous morphine and dopaminergic function, reinforcing neural pathways associated with reward, anger might be strengthened overall. Such reinforcement is probably mediated via the mesolimbic dopaminergic (ML-DA) pathway projecting from the ventral tegmental area of the brain to the hypothalamus and limbic system (Davidson & Irwin, 1999; Nestler, Hyman, & Malenka, 2001; Niehaus, Murali, & Kauer, 2010). It is suggested that the neuromodulatory dopaminergic system is critically involved in the ‘motivation circuit’ that stimulates intense euphoric excitement and anticipation of reward.
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(Alcaro et al., 2007; Ikemoto & Panksepp, 1999; Salamone, Correa, Mingote, & Weber, 2003) is implicated in the emotional processing of anger (Reuter et al., 2009).

By being reinforced in such ways aggressive expression of emotion (anger) does not dissipate anger in the long run or diminish the inclination to express that emotion. It instead further conditions and encourages that affect (Bushman, 2002; Bushman, Baumeister, & Phillips, 2001). Retaining the belief that dysfunctional emotional expression will cause cathartic release despite previous failures may readily increase outbursts in the future as the patient anticipates a catharsis that will never come (Bushman, Baumeister, & Stack, 1999; Lohr, Olatunji, Baumeister, & Bushman, 2007). Under considerable stress, secondary negative consequences can include drug use, self-harm, injury to others during impulsive actions, damaged property, deteriorating relationships, or legal and employment difficulties (Deffenbacher, 2011; Deffenbacher, Oetting, Lynch, & Morris, 1996; Dahlen & Martin, 2006).

Provocation is one condition that can contribute to substance use disorder patients’ displaying aggression in light of its association with depletion of self-control (Denson, et al., 2011). There is sufficient evidence to support clinical approaches based on patient development of assertive communication skills and modifying beliefs about the nature of anger and aggression to successfully improve mood regulation (Lewis, 1992). Further research is required to determine if increasing patients’ understanding of neurochemistry and brain reward function as it relates to drug use and aggression is needed to help patients appropriately express or sublimate aggressive urges motivated by anger

Anger-Induced Blackout

Based on first-hand clinical experience, patients may believe that anger felt too intensely can cause blackouts, creating memory gaps. Anecdotal accounts from patients in clinical practice and related findings in the literature (Rosellini & Worden, 1997) provide some data for the presumption that uncontrollable and unstoppable anger can induce memory loss. This apparent memory impairment during a “blackout” state could be explained by the mood-state-dependent retrieval hypothesis (Bower, 1981), suggesting that memories formed in extreme anger could be accessible only in a similar emotional state (Swihart, Yuille, & Porter, 1999).

However, no matter how extreme or intense anger may be, this emotional experience seems insufficient to cause genuine memory loss (Tavis, 1982; Wolf, 1980). A more plausible explanation for anger-induced “blackout” is what Tavis (1982) calls a mental set, which includes: (a) the patient’s expectations about what will happen when anger is experienced, (b) personal payoffs and consequences as a result of expressing it, (c) the extent of self-control over anger, (d) the degree of responsibility for behavioral choices, and (e) the environmental setting that accompanies the angry mood. The patient’s mental set may therefore ultimately create the illusion of blacking out from extreme anger, when in actuality they are simply behaving stereotypically (Barnwell, Borders, & Earleywine, 2006; Bushman et al., 1999; Marlatt & Rohsenow, 1981; Sell, 2011).

In the blackout state, anger management patients report confronting a target they perceived as weaker or vulnerable who posed no real threat or caused little, if any, consequence to status, dominance, authority, employment, family, or personal safety (DiGiuseppe & Tafrate, 2007; Tavis, 1982). Patients’ autobiographic narratives of the events directly preceding anger-related memory loss typically end with patients “victorious” over the person or group they targeted (Kassinove & Tafrate, 2002). When patients claim they had no control over the violent expression of anger due to blacking out, they show a remarkable ability for a state of dissociation to exercise rational thinking to the extent of deciding who they will ultimately “black out” around and selecting what enraging event will cause the “black out” (Rosellini & Worden, 1997). Accordingly, how patients think about
anger-induced blackouts via activation of their ‘mental set’ matters more than the actual phenomenon itself (Bushman et al. 1999; Tavis, 1982)—similar to Sell, Tooby, and Cosmides’ (2009) recalibrational theory of anger.

Overall, patients who are convinced that intense anger can induce a state of partial amnesia may be least aware of their own physical sensations of anger arousal and the thoughts that can provoke that feeling. This failure to recognize emotional, cognitive, physical, and behavioral warning signs that signal anger buildup can lend credence to the idea of the limbic system to go into “overdrive” and induce a blackout (Goleman, 1995). This underscores the importance of mindfulness of the patient’s internal world and physical state in anticipating the acceleration of anger, and quickly interrupt it by consciously plugging in thoughts to bring the anger under control (Kassinove & Tafrate, 2002). Patients, in turn, are likely to feel more in charge over how they act out their anger as they learn how to recognize and modulate their own reaction patterns (Tavis, 1982). However, further research is required to determine if increasing patients’ understanding of hyper-arousal and the effect of anger and drugs on the brain can lower the credibility of belief in “blackout anger,” and eventually accept that it is a false belief.

Neurophysiological Level of Analysis

Mirror Neurons and the Social Programming of Anger

In treatment, stress, feelings of loss, apathy, and other emotional fluctuations related to withdrawal and the removal of mood-mind altering substances as a coping strategy can induce sensation-seeking behavior to feel more alive and experience excitement, especially early on in the recovery stage. Patients may invite arguments, verbal confrontations, and social drama that can provide entertaining distraction from confronting their own internal sensations and disappointing moods. Provoking interpersonal conflict can further generate temporary thrill due to the stimulation of neurochemicals, potentially cueing state-dependent drug-associated memory pathways.

Patients who are not the direct target of another’s angry expression, but observe it from the periphery, may process angry feelings without internal awareness. Conceptual understanding of emotional and social contagion as well as mirror neurons reveals non-conscious processes in the social transmission of angry feelings. Bennett (2011) described mirror neurons, cells in the frontal and parietal lobes, as “Brain antennae that pick up information about the intentions and emotions of others, enabling us to imitate behaviors and experience the feelings of others.” This suggests that the expression of anger co-occurs with observed Aggression. It is suggested that repeated activation of such mirror neurons could prime the amygdala and enterorhinal cortex circuits associated with anger to fire at a lower threshold, rendering the individual more prone to express angry feelings (Post, 2007; Rizzolatti & Craighero, 2004; Siever, 2008). To that point, Stosny adds, "anger and resentment are the most contagious of emotions. If you are near a resentful or angry person, you are more prone to become resentful or angry yourself" (cited in Restak, 2004, p. 37).

The priming effect of mirror neurons on the amygdala can lead to emotional contagion resulting in a tendency to tune in to others’ feelings and behaviors so that each experience of anger expression increases the risk to mirror the behavior (Hatfield, Cacioppo, & Rapson, 1994; Iacoboni, 2009; Rizzolatti, 2005; Todorov & Bargh, 2002). The result is that emotional diffusion is an agent of social contagion in that patient exposure to the behavior of others in the treatment milieu can influence similar behavioral change via observation or behavioral mimicry (Christakis & Fowler, 2007). In accordance, patients can believe they can exercise complete control over their cognitive and behavioral cues to anger and appraise provocation using appropriate anger management.
strategies and techniques, yet aversive stimuli and negative environmental cues still induce angry feelings. This is consistent with Berkowitz’s (1990) cognitive-neoassociationistic model of anger and aggression. Research is required to determine if increasing patient’s understanding that anger is to some degree a neurologically hard-wired response will enhance the ability to cope.

Discussion

Anger may provide short-term benefits that enhance patients’ coping capabilities, facilitating abstinence and withdrawal by “mobilizing psychological resources, energizing corrective behaviors, facilitating perseverance, protecting self-esteem, and communicating negative sentiment” (Wright & Howells, 2009, p. 396, citing Taylor & Novaco, 2005). However, intense and prolonged experiences of anger can hold destabilizing affect as it reinforces the modulation of anger that increases intrapsychic distress and interpersonal stressors. Given the high co-occurrence of psychiatric disorders such as post-traumatic stress, anxiety, bipolar disorder, depression, and borderline personality in the general substance use disorder population (Flynn & Brown, 2008), anger may represent a mood state (agitation) consistent with a distinct psychiatric symptom descriptor (emotional arousal in PTSD) or core feature of a physiological condition (chronic pain, insomnia) (Fernandez & Turk, 1995). When anger is a patient’s distinct psychiatric trait or symptomatic of an underlying physiological condition, the qualitative experience of that emotion can be differently comparable to patients for whom the hypothesis of anger as self-medication appears more valid.

Because this review is experientially informed by anecdotal accounts gathered through clinical intervention with stimulant users, it does not include quantitative data about the frequency and types of anger problems among this population. Also, it is unclear how the hypothesis that patients may substitute anger for stimulant drugs as a form of self-medication would apply to polysubstance use or those recovering from problematic opiate, alcohol, or anxiolytic use. Additionally, how neurophysiologic aspects of anger correlate with drug use categories to affect behavior has yet to be validated. It is also possible that anger acting as a medicating neurochemical stimulant similar to psychoactive drugs is as relevant to aggression as a thrilling addictive element.

However, anger management in drug treatment (see Reilly & Shopshire, 2002; Shopshire & Reilly, 2013) does not necessarily correspond with specific classifications of substances or cohere with the phenomenological experiences of users of those drug categories. Neurobiology-informed anger management interventions in relationship to drug-brain-anger specific interactions and alterations could potentially improve the comprehensiveness of anger management (Potter-Efron, 2012). Adding greater sensitivity as well as specificity to treatment for stimulant users in drug addiction programs could possibly augment patient motivation, receptivity, and adaptive exploration of anger management techniques.

References


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