Journal of Research in Personality 43 (2009) 673–681

Contents lists available at ScienceDirect

Journal of Research in Personality

journal homepage: www.elsevier.com/locate/jrp

Trait displaced aggression and psychopathy differentially moderate the effects of acute alcohol intoxication and rumination on triggered displaced aggression

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ARTICLE INFO

Article history:
Available online 17 April 2009

Keywords:
Trait displaced aggression
Psychopathy
Triggered displaced aggression
Alcohol
Rumination

ABSTRACT

When angered, alcohol and rumination increase aggression toward the source of a subsequent minor annoyance. Little is known about individual differences that moderate this phenomenon. One hundred university students (47 men, 53 women) were provoked and given either alcohol or placebo and subsequently induced to ruminate or engage in distraction. Participants were then given the opportunity to aggress against a somewhat annoying fictitious participant by determining the amount of hot sauce the other participant must consume. Alcohol and rumination independently augmented aggressive behavior, and these effects were moderated by trait displaced aggression and psychopathy, respectively. These findings suggest alcohol use and rumination as targets of intervention, specifically for those high in trait displaced aggression and psychopathy.

1. Introduction

The causes of many acts of aggression are difficult to comprehend. For example, the excessive aggression commonly observed in cases of intimate partner violence and road rage are often instigated by fairly trivial triggering events. Research and theory within the triggered displaced aggression (TDA) paradigm can explain such perplexing instances of aggression that exceed normative tit-for-tat responding (Axelrod, 1984). The classic displaced aggression effect occurs when a person is provoked, is unwilling or unable to retaliate against the original provocateur and subsequently aggresses against a seemingly innocent target (Dollard, Doob, Miller, Mowrer, & Sears, 1939; Hovland & Sears, 1940; Marcus-Newhall, Pedersen, Carlson, & Miller, 2000). In contrast to classic displaced aggression, TDA occurs when the subsequent target is the source of a second, subjectively annoying provocation, (referred to as the trigger), and the aggressor responds with a disproportionate level of aggression (Pedersen, Gonzales, & Miller, 2000; Vasquez, Denson, Pedersen, Stenstrom, & Miller, 2005). To illustrate, a man who is scolded by his superior (e.g., "you really messed up that presentation") and does not retaliate, but then encounters a co-worker who is less than sympathetic (e.g., "well, maybe you should have tried harder") and subsequently shouts insults at the co-worker, has engaged in TDA.

A number of factors increase the severity of TDA. These include alcohol intoxication, rumination, and the stable dispositional tendency to displace aggression (Aviles, Earleywine, Pollock, Stratton, & Miller, 2005; Bushman, Pedersen, Vasquez, Bonacci, & Miller, 2005; Denson, Aviles, et al., 2008; Denson, Pedersen, & Miller, 2006). These effects can be understood within the context of the General Aggression Model (GAM; Anderson & Bushman, 2002). The GAM proposes that personal characteristics interact with situational features such as provocation and alcohol intoxication to activate aggressive cognition, angry affect, and/or physiological arousal. When sufficiently activated, these internal states bias appraisal and decision-making processes, which subsequently increase the likelihood and severity of aggressive behavior.

Recent research suggests that rumination increases all three of the internal antecedents to aggression specified in the GAM: angry affect, aggressive cognition, and physiological arousal (Kross, Ayduk, & Mischel, 2005; Ray, Wilhelm, & Gross, 2008; Rusting & Nolen-Hoeksema, 1998; Wimalaweera & Moulds, 2008). Miller and colleagues (Miller, Pedersen, Earleywine, & Pollock, 2003) theorized that prolonged rumination facilitates TDA because it maintains these routes to aggression over time, which in turn, increase hostile reactions to subsequent triggering events. The findings of recent TDA experiments are consistent with this theorizing.

Specifically, relative to distraction, rumination increases displaced aggression, but only when individuals are provoked and rumination is followed by a triggering event (Bushman et al., 2005; Denson et al., 2006; Denson, Spanovic, et al., 2008).

Only one previous experiment has simultaneously examined the effects of rumination and alcohol in eliciting aggression. Denson, Spanovic, et al. (2008) found that alcohol and self-focused

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doi:10.1016/j.jrp.2009.04.003

rumination independently augmented TDA. *Self-focused rumination* is defined as thoughts and feelings about one's thoughts, behavior, and negative emotions (Rusting & Nolen-Hoeksema, 1998). This is distinct from *provocation-focused rumination*, wherein the individual's thoughts and feelings are primarily externally focused on reliving the provoking incident and planning revenge (Denson et al., 2006). The current study is the first to simultaneously examine the effects of alcohol and provocation-focused rumination on aggression.

Researchers have proposed a number of psychological mechanisms to explain the aggression-augmenting effects of alcohol. One such mechanism, the *attention–allocation model* (also known as *alcohol myopia*), posits that alcohol consumption interferes with cognitive functioning by narrowing one's attention toward the most salient cues in the environment and steering attention away from less salient cues (Giancola & Corman, 2007; Steele & Josephs, 1990). This model is consistent with a recent TDA experiment which found that intoxicated participants were only able to pay attention to highly salient triggering events, whereas sober participants were able to allocate their attention to triggers that were both high and low in salience (Denson, Aviles, et al., 2008).

A second compatible mechanism is that of diminished inhibitory control. Giancola (2000, 2004) suggests that intoxicated aggression occurs as the result of temporary impairment of executive functioning, primarily via a reduction in the ability to control aggressive thoughts and actions. Such theorizing is consistent with neuroimaging research demonstrating that acute alcohol intoxication disrupts activity predominantly in the prefrontal cortex (Volkow, Wang, & Doria, 1995). Similarly, a recent study also reported alcohol dose-related increases in impulsive responses on a measure of inhibitory control (Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008).

2. Trait displaced aggression and psychopathy

Although the consistency of the alcohol–aggression relationship is robust, it is well established that alcohol does not facilitate aggression in everyone. Meta-analyses have shown that the magnitude of the alcohol–aggression link is moderate, supporting the notion that additional situational and dispositional variables are necessary to fully explain for whom and under what conditions alcohol increases aggression (Bushman & Cooper, 1990; Ito, Miller, & Pollock, 1996). Theorists have speculated that alcohol and rumination most likely prompt aggressive behavior only for persons who are already predisposed to behave aggressively (e.g., Caprara, Barbaranelli, & Comrey, 1992; Giancola, 2002b). Personality risk factors that moderate alcohol-induced aggression include low anger control, high trait anger, aggressive traits, low dispositional empathy, and narcissism (e.g., Barnwell, Borders, & Earleywine, 2006; Eckhardt & Crane, 2008; Foran & O'Leary, 2008; Giancola, 2002a, 2002b;). In the present research, we investigated two personality dimensions that have not yet been considered in experimental research on alcohol-induced aggression (viz. trait displaced aggression and psychopathy).

When provoked, individuals high in *trait displaced aggression* tend to respond with angry rumination rather than direct retaliation, and eventually end up 'taking it out' on undeserving others in the laboratory and in the real world (Denson et al., 2006). Interestingly, trait displaced aggression is correlated with behavioral inhibition. This is in contrast to trait general aggression, which is correlated with approach orientation and characterized by intense, immediate anger and direct retaliation following provocation (Buss & Perry, 1992). Because alcohol is associated with disinhibition (Giancola, 2002a), we expected that alcohol would augment TDA among those high in trait displaced aggression by weakening their normally adequate levels of inhibitory control. Thus, trait displaced aggression should moderate the effect of alcohol on TDA. By contrast, experimentally induced rumination might not have an additional effect on those high in trait displaced aggression, because these individuals have a natural tendency to engage in provocation-focused rumination (Denson et al., 2006).

This is the first experiment to examine *psychopathy* within the TDA paradigm (or any other form of acute alcohol-induced aggression). Psychopathy is a robust predictor of aggression and violence in forensic, psychiatric, and youth populations (e.g., Edens, Lilienfeld, Poythress, & Patrick, 2008; Porter & Woodworth, 2005; Skeem & Mulvey, 2001). In community and college samples, psychopathy has been linked with premeditated and reactive forms of aggression under both neutral and provoking conditions (e.g., Nouvion, Cherek, Lane, Tcheremissine, & Lieving, 2007; Reidy, Zeichner, & Martinez, 2008; Reidy, Zeichner, Miller, & Martinez, 2007).

In broad terms, there are two subtypes of psychopathy that are associated with aggression (see Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Hare, 1991; Harpur, Hakstian, & Hare, 1988; Levenson, Kiehl, & Fitzpatrick, 1995).[1] The first subtype is characterized by fearlessness, callousness, manipulativeness, lack of remorse, and low anxiety. In Lilienfeld and Widow's (2005) Psychopathic Personality Inventory-Revised (PPI-R), this relates to the factor known as Fearless Dominance (FD). The second subtype is characterized by extreme self-centeredness, impulsivity, proneness to boredom, poor behavioral control, poor goal-setting, and aggressive, anti-social behavior. In the PPI-R this relates to the factor known as Self-Centered Impulsivity (SCI). Both subtypes of psychopathy are correlated, with most imprisoned psychopathic individuals scoring high on both dimensions (Levenson et al., 1995). Impulsive behavior, which is most noticeable in SCI-type psychopaths, is considered to be the result of a breakdown in inhibitory control mechanisms. Indeed, studies with clinical samples indicate that adolescent psychopaths exhibit worse performance than normal controls on neuropsychological tests of executive functioning (Roussy & Toupin, 2000).

Because individuals high in psychopathy are dispositionally disinhibited, we expected that alcohol would not increase aggression among such individuals. Indeed, research with forensic and clinical samples has found that psychopaths and those with antisocial personality disorder were most likely to engage in violence when they were sober, despite higher overall levels of alcohol dependence than controls (Holcomb & Adams, 1985; Walsh, 1999). The authors suggested that alcohol lowers inhibition against violence only in non-psychopathic personalities. Thus, whereas alcohol lowers the threshold for aggression in low-psychopathy individuals, high-psychopathy individuals, who are often at or above this aggression threshold, tend to respond aggressively to even mild provocations, and the presence of alcohol should not appreciably alter this tendency. For this reason, alcohol intoxication was expected to interact with psychopathy in the current experiment such that alcohol would increase aggression only for those low in psychopathy. Furthermore, it was expected that provocation-focused rumination, which focuses attention on a highly-salient provocation, would exacerbate psychopathic tendencies toward blame externalization, self-centeredness, and exaggerated entitlement. As a result, rumination should elicit higher levels of TDA in high-psychopathy individuals than low-psychopathy individuals. Thus, we expected that psychopathy would interact with rumination to augment TDA.

[1] A third psychopathy factor, cold-heartedness, was not associated with aggression in prior research or the current study (Barry et al., 2007; Benning et al., 2005; Edens et al., 2008).