Emotional Catharsis and Aggression Revisited: Heart Rate Reduction Following Aggressive Responding

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The authors tested two components of the catharsis theory of aggression: physiological tension reduction and aggressive drive reduction. On the basis of work in the stress–aggression literature, they also examined the moderating effect of impersonal stress exposure on cathartic reductions in heart rate following aggressive responding. Participants were instructed to administer nonaggressive (correct button) or aggressive (shock button) responses to a frustrating confederate in a laboratory aggression paradigm, and half the participants were exposed to an impersonal stressor (aversive air blasts) during the procedure. Heart rate was recorded before and after the participants administered the aggressive or nonaggressive response. Analyses revealed that participants exhibited reductions in heart rate following aggressive but not nonaggressive responding, but this was the case only for those not exposed to the impersonal stressor. Heart rate reductions during the experimental blocks actually predicted the most intense aggression in a subsequent block of trials. The results are considered in light of different theories of aggression by J. E. Hokanson (1974) and L. Berkowitz (1990) and have implications for interventions with anger-prone individuals.

Keywords: aggression, stress, tension reduction, catharsis, heart rate

Popular advice suggests that the individual outwardly express frustration (e.g., hitting a pillow, taking up boxing) to abate feelings of anger as well as to prevent the possibility of future aggressive acting out. Stemming from classic Greek literature, the dramatic idea of catharsis was that experiencing strong emotions such as rage, grief, or pity while watching a tragic play was emotionally and spiritually cleansing for the individual (Schaar, 1961). In modern psychology, the clinical conception of catharsis hails back to Breuer and Freud (1957), who, through their work with patients who suffered from “hysteria,” described catharsis as a feeling of relief or release following emotional expression of anger that resulted in the diminishment, if not complete abatement, of emotional symptoms. Nonetheless, the results of systematic experimental research investigating the mechanisms and supposed benefits of catharsis have been mixed, especially in regards to whether outward expressions of aggression lead to emotional tension reduction as well as aggressive drive reduction (Geen & Quany, 1977). However, few researchers have examined whether tension reduction that follows from behavioral expressions of frustration actually predicts the probability of future aggression. Current studies also have not integrated newer models that suggest that emotional distress resulting not only from direct insult by a provocateur but from everyday experiences of acute and chronic stress may also influence aggressive responding (Berkowitz, 1990). In this research, we reexamined the case for catharsis of aggression by studying (a) whether engaging in an aggressive act against a frustrating target actually produces physiological tension reduction (i.e., decreases in heart rate), (b) whether exposure to a concurrent impersonal stressor (air blast) moderates the tension reduction afforded by engaging in aggressive action, and (c) whether tension reduction following aggression predicts reductions or increases in aggressive drive (i.e., future aggression).

Catharsis Theory: Empirical Support for Tension Reduction and Drive Reduction

Drawing heavily from the psychoanalytic conceptualization of catharsis, Buss (1961) proposed the classic hydraulic model of aggression. According to this model, emotions that cause aggression “build up” in an individual, creating tension; when released via behavioral expression, typically through an aggressive act, the associated tension is reduced (Buss, 1961). Taking this model one step further, catharsis theory suggests that the drive to commit subsequent aggressive acts will also be reduced (Schafer, 1970). Thus, according to catharsis theory, engaging in an aggressive act in response to frustration should lead to both (a) tension reduction and (b) aggressive drive reduction.

Building on the work of Buss (1961), early studies by Hokanson tested these two components of catharsis theory: physiological (tension reduction) and behavioral (aggressive drive reduction) effects of cathartic venting (Hokanson, 1974). For example, Hokanson and colleagues found that in experimental conditions involving interpersonal provocation, male (but not female) participants demonstrated a rapid reduction in systolic blood pressure when allowed to respond in an aggressive manner, whereas participants who responded in a nonaggressive manner or who were

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not permitted to respond at all evidenced slower reductions in their blood pressure (Hokanson & Edelman, 1966; Hokanson & Shetler, 1961). In a study following up on Hokanson’s work, Geen, Stonner, and Shope (1975) found that, in addition to the physiological changes following an aggressive response, those who initially responded with aggression became more likely to respond with aggression in the future. This finding, along with other studies that directly contradicted earlier theorizing that aggressive actions result in a diminishment of aggressive drive, was interpreted by Hokanson as evidence that reduction in tension following an aggressive act actually serves as a reinforcer for future aggressive behavior (Hokanson, 1974; Hokanson, Willers, & Koropsak, 1968). This model is illustrated in Figure 1. As per Figure 1, the link between retaliating aggressively and physiological tension reduction has been tested empirically, as has the link between retaliating aggressively and future aggressive tendencies. However, existing studies have not tested the direct relationship between physiological tension reduction and subsequent aggressive behavior (dashed line in Figure 1).

With a few exceptions (Engebretson, Matthews, & Scheier, 1989; Lai & Linden, 1992), experimental investigations into catharsis and aggression have languished until the past few years, when Bushman and colleagues began reexamining the mechanisms of catharsis in light of popular psychology and media endorsements of catharsis as useful and healthy. In one study examining the effect of expectancies on cathartic benefits and aggressive responding, Bushman, Baumeister, and Stack (1999) found that participants who read a procatharsis newspaper article (i.e., “Research Shows That Hitting Inanimate Objects is an Effective Way to Vent Anger”) were more likely to select an aggressive activity (hitting a punching bag) after receiving negative feedback on an essay than were those who read an anticatharsis article. Even further, among those who read the procatharsis article, the participants who also hit the punching bag subsequently engaged in more aggressive interpersonal responses (both in terms of strength and duration of a noise blast directed at another individual) than those who did not hit the punching bag (Bushman et al., 1999). This provided additional support for Hokanson’s early contention that venting anger may actually reinforce engagement in future aggression. Bushman and colleagues (Bushman, 2002; Bushman, Baumeister, & Philips, 2001) have concluded that participants who believe that venting anger is useful as an emotion-regulation strategy are more likely to use cathartic venting (i.e., punching a bag) and to engage in more aggressive acts in subsequent interactions. Bushman’s studies highlight that cognitive expectancies about catharsis will affect their reactions to stress or frustration. More relevant, these studies do not provide support for the drive reduction predictions of catharsis theory; that is, venting anger, which would presumably lead to tension reduction, results in an increase, not a decrease, in aggression.

As a way of elaborating on previous investigations of catharsis, we examined physiological tension reduction following aggressive versus nonaggressive actions toward a frustrating target. With previous studies suggesting that tension reduction plays an important role in the potential for future aggression, the question of whether physiological tension reduction actually predicts subsequent aggression remains of central importance (see Figure 1), despite the fact that this question has yet to be systematically examined in modern catharsis research. Thus, one contribution of the current study includes that we incorporated a physiological measure of tension reduction from pre- to postaggressive responses. Another contribution involves the analysis of the moderating effects of impersonal stress exposure on physiological tension reduction involving aggression, as discussed next.

**Stress and Physiology–Aggression Relationships**

Although there has been no recent research into the physiological aspects of catharsis and aggression, there have been substantial advances in our understanding of the physiology of aggression (see Lorber, 2004; Patrick & Verona, 2007). One relevant theory has been advanced by Berkowitz (1990), in his neoassociationistic model of aggression. He postulated that an aversive event (i.e., a stressor) primes an individual for engaging in aggressive behaviors by activating relevant physiological systems and memory areas that have been linked to aggression. In other words, stress-related physiological arousal leads to aggressive behavior because the physiological systems and the behavioral responses are both connected to an associative network that governs defensive responses to threat. An important point is that Berkowitz’s model suggests that the activating aversive event need not be interpersonal (insults, negative feedback by another) in nature. Indeed, numerous studies have found that impersonal stressors, such as uncomfortable room temperatures (Anderson, Anderson, & Deuser, 1996), unpleasant odors (Rotton, 1979), and annoying air blasts (Verona, Patrick, & Lang, 2002), lead to increases in aggressive behavior. However, some recent work has revealed that individual differences, particularly gender, moderate the effects of stress and physiological activation on aggression (Verona & Curtin, 2006; Verona & Kilmer, 2007). Interestingly, Hokanson (1974) reported that physiological tension reduction after aggression occurs in men but not women.

One of the goals of the current study was to integrate catharsis–aggression research with associative network models that suggests that impersonal stressors can increase risk for aggressive behaviors.

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**Figure 1.** Pictorial representation of two components of catharsis theory of aggression.
Research on catharsis suggests that engaging in aggressive behavior serves to reduce the physiological arousal elicited by interpersonal frustration. However, it is unclear whether the tension-reducing effect of aggressing against a target is limited to situations in which the negative affect is only evoked by interpersonal provocation from that target. In the current study, we investigated whether impersonal stress exposure (i.e., intermittent air blasts directed at the throat) would moderate the physiological tension-reducing benefits of engaging in aggressive behavior against a frustrating target. Bushman’s work (Bushman, 2002; Bushman et al., 1999, 2001) on expectancies would suggest that the potential benefits of catharsis may be experienced only by those who believe that the aggressive behavior could help relieve their tension. Berkowitz (1962) hypothesized that a frustrating situation leads to an intent to injure the source of the negative affect; thus, tension reduction after aggressing against a frustrating target probably occurs because the aggressor feels that her or his intent was satisfied. In the case of participants whose negative affect is initiated by an external stressor that is unrelated to the actions of another person, they may not experience the physiological benefits of aggressing against that person. This hypothesis is of particular importance in today’s society, as instigators for aggression are often a combination of noninterpersonal (e.g., financial strain, unemployment) and interpersonal (e.g., insult, apathy) stressors.

Current Study

Despite the resurgence of interest in catharsis and aggression within both popular culture (“Thwack! Kerpow! I’m purging my anger,” 2007) and psychological research (Bushman, 2002; Bushman et al., 1999, 2001), the physiological processes implicated in catharsis theory and their relations to future behavior remain unclear. The current study is the first to collectively study the relations between engaging in an aggressive act, heart rate reductions following an aggressive act (tension reduction), and the likelihood of engaging in subsequent aggressive behavior (drive reduction). In addition, because recent research has demonstrated that aggressive behaviors may result from situations where there is no direct interpersonal provocation (e.g., noxious smells, uncomfortable temperatures), another goal of the current study was to examine whether exposure to an impersonal stressor moderated the potential physiological benefits of aggressing. Finally, on the basis of previous work suggesting gender differences in tension reduction following aggression (Hokanson & Edelman, 1966; Lai & Linden, 1992), equal numbers of men and women were included in this study.

The tension reduction and drive reduction components of catharsis theory in both men and women were tested within a laboratory aggression paradigm in which the target of aggression (i.e., confederate) performed poorly on a task, with implications for the participant (i.e., participant could not receive money because of confederate’s poor performance). During the aggression procedure, half the participants were exposed to intermittent air blasts directed at their throats (high stress) and half were not (low stress) while heart rate (HR) activity was monitored. The first goal of the study was to test the tension reduction component of catharsis theory (Geen & Quany, 1977). The hypothesis was that engaging in an aggressive versus a nonaggressive response against the frustrating target would relieve physiological tension by reducing HR activity from pre- to postresponse, mostly in male participants (Hokanson & Edelman, 1966). The second goal was to examine the effects of the concurrent impersonal stressor on tension reduction following an aggressive response.

One hypothesis was that the physiological arousal associated with the air blast stressor in the high-stress condition would not be reduced following aggressive responding toward the target because the negative affect experienced was partly unrelated to the interpersonal situation (Ellsworth & Smith, 1988). Thus, we expected that stress condition would moderate the effects of aggressive versus nonaggressive responding on HR reduction pre- to postresponse. The third goal was to assess the drive reduction part of the theory, or whether HR reductions following aggression actually predict future aggressive behavior. On the basis of previous theorizing (Hokanson, 1974), we expected that reductions in HR following aggressive responses would predict increases and not decreases in subsequent aggression.

Method

Participants

Participants were 110 volunteers (54 women) recruited through psychology classes and via fliers posted around campus, and they were scheduled for participation via telephone or email. They received course credit and $10 compensation for their participation. Participants who reported hypertension or cardiovascular illness were excluded from participation. The mean age of the participants was 21 years (SD = 5.7). Participants were mostly Caucasian Americans (n = 95; 89%), whereas the rest identified as African American or another ethnicity. Of the 110 participants, 3 were missing HR data, and 2 were missing postrecovery shock intensity data because of equipment malfunction. Thus, these participants were not included in analyses involving these variables.

Procedure

After signing informed consent, participants were introduced to a same-sex student (actually a confederate of the experimenter), and both were told that the study involved an investigation of the effects of distraction on supervisor and employee performance. The real participants were always assigned the role of supervisor. The supervisor (i.e., real participant) and employee (i.e., confederate) were then escorted to separate rooms.

Electrode hook-up and baseline physiological assessments.

Before the instructions were provided, the participants were seated in a recliner positioned in front of a 21-in. monitor, from which they could view the employee’s “answers” later in the experiment. Participants in all conditions were fitted with electrodes to record eyeblink startle reflex (to establish the efficacy of the stress manipulation) and HR (to test tension reduction). The eyeblink component of the startle reflex was recorded from the orbicularis oculi muscle beneath the left eye, using 4-mm electrodes (Med Associates, St. Albans, VT), and HR activity was recorded from 8-mm electrodes positioned on the right and left inner forearms. Elec-

1 We introduced participants to confederates of their same gender to optimally streamline analyses and presentation of results. Prior research has indicated that interactions between target and perpetrator gender in laboratory aggression paradigms mostly reflect the fact that men are less willing to aggress against a female target than a male target (see Frodi, 1978; Schnake, Ruscher, Gratz, & O’Neal, 1997), and male-on-female aggression is considered especially prohibitive (Hilton, Harris, & Rice, 2003). Given this literature, this confound will probably have a limited impact on the interpretation of our findings.
trode placement procedures followed established guidelines (e.g., Blumenthal et al., 2005). Following electrode hook-up, participants were instructed to relax, and a 5-min HR baseline was obtained (Beauchaine, Gartner, & Hagen, 2000). Then, a pretest startle assessment was conducted (Verona et al., 2002). The startle reflex was elicited by a 50-ms presentation of a 105-dB burst of white noise (i.e., a startle “probe”) with an instantaneous rise time. Seven startle probes were presented through participants’ earphones, separated by intervals of 11–13 s, to elicit baseline startle reflex. The raw electromyographic and HR signals were obtained with Neuroscan amplifiers and digitized at 2000 Hz.

Instructions and cover story. The real participants were told that the employee was performing a digit memory task in the next room, and they would view the confederate’s answers on the monitor positioned in front of them. They were to provide accuracy feedback to the employee via a “correct” button if the answer was correct, or a “shock” button if the answer was incorrect. Participants were told that, as supervisors, they would administer shocks to the employee to simulate “criticisms” of job performance, as in a work situation, and they were free to choose and vary the level of shock to administer across the trials. This cover story was used to assure that participants’ shock responses could be interpreted as punishment for negative performance and not as helping behavior as in the teacher–learner paradigm. The participants were led to believe that they were administering actual shocks to the employee; in actuality, no real shock was delivered.

Participants were told in addition that they would split $20 with the employee ($10 for the participant) if the employee performed the task effectively. The experimenter manipulated the employee’s answers so that he or she made a large number of errors during each block and across the whole experiment. Thus, the participant could not earn any money at the end of the experiment. After each block, all participants received feedback on their monitor screen as to the number of incorrect answers made by the employee during that block. This procedure ensured participants’ frustration with employee performance, and this has been confirmed in our previous work (Verona & Curtin, 2006).

Stress conditions. Following the cover story instructions, participants were randomly assigned to stress conditions in a gender-balanced way: low stress (27 women, 29 men), high stress (27 women, 27 men). As per the cover story, they were told that the study examined the impact of environmental distractions on participants’ decision making and performance and that they would be randomly assigned to either receive a “distraction” (high stress) or no distraction (low stress). Those assigned to high stress were told that they would receive brief, intermittent blasts of compressed air to the throat throughout the procedure. The air blasts, which were 100 psi and 50 ms in duration, were delivered via a tube positioned through a harness placed around the participants’ waist and chest and directed at the throat at the level of the larynx (Grillon & Ameli, 1998; Verona & Curtin, 2006). The participants were assured that the employee was not at all responsible for these air blast administrations. Participants in low stress were not fitted with the harness. They were told that they were in the control condition and that they would not receive a distraction during the procedure.

Experiment Structure

Block structure. Before the beginning of the actual experimental blocks, participants completed an initial block of trials (practice block) so that they could get accustomed to the aggression paradigm and to obtain baseline aggression scores. Participants provided feedback (shock or correct responses) to the confederate without the administration of air blasts (even if they were in the high stress condition) during this practice block. This was an assessment of preexisting differences in tendencies toward aggression in the laboratory.

The actual experiment followed and consisted of a total of four experimental blocks. During the experimental blocks, participants in high stress were administered a total of 16 air blasts (4 within each block) across the procedure. Following the four experimental blocks, participants were allowed to relax for 5 min during a recovery period. Then, they were asked to complete a final postrecovery block (in which there was no stress exposure for either stress group). Participants’ aggression scores during this final block allowed us to examine to what extent HR reductions following aggressive responding during the four experimental blocks actually predicted future aggression (during postrecovery block). See Figure 2 for pictorial presentation of the experiment and trial structure.

Trial structure. Each block in the experiment consisted of 10 trials. For 50% of the trials, the employee responded incorrectly, requiring a shock button response from the participant (aggression trials); the other half of the trials required “correct” button feedback to the employee (nonaggression trials). Across the whole experiment, incorrect trials were followed by the same number of correct and incorrect trials to ensure that no contingency was established between the delivery of shocks by the participant and the employee’s purported performance on subsequent trials. As illustrated in Figure 2, trials within blocks involved a prereseponse interval, while the participants waited for the employee’s answer to come up on the screen. After 6 to 8 s, the confederate’s alleged digit recall answer appeared on the screen, followed by the participant’s button press, either an aggressive (shock buttons 1–10) or nonaggressive (correct button) response. An 8- to 10-s postresponse interval followed the participant’s response (see Figure 2). HR recordings were made continuously during the prereseponse period and during the postresponse period of each trial. Four startle probes were delivered to elicit startle blink reactions in each of the blocks.

Physiological Measures

Startle sensitization. Offline data processing included signal epoching (sample window includes 50 ms preprobe to 250 ms postprobe presentation), rectification, smoothing (30 Hz lowpass filter, 24 dB/octave), and baseline correction. The startle reflex

2 We recorded startle reactivity in the two stress conditions to more directly assess the efficacy of the stress exposure to prime negative emotion. Even though we had HR recordings, cardiac change does not directly index the valence of the response and partly reflects the metabolic demands of stimulus processing, attention, and orienting (Lacey, 1967). Although HR is a good index of arousal, HR reactivity differences between the two conditions may be obscured in our task, which involved substantial attention and orienting to various stimuli.
was scored offline as peak magnitude of the eyeblink reaction for each probe presentation. Tonic startle reactivity during the test procedure was operationalized in terms of startle sensitization—that is, magnitude of the startle reflex during the experiment as a proportion of baseline startle scores (Grillon & Davis, 1997; Verona & Curtin, 2006). Startle sensitization scores across the experimental blocks were used to examine the efficacy of the stress manipulation. Startle scores could not be calculated separately for preresponse and postresponse periods during trials because there were too few startle recordings during the postresponse period (only one in each block) to generate a reliable measure of reductions in startle from pre- to postresponse separately for aggression and nonaggression trials.

Heart rate. Offline processing of HR involved automatic detection of R-spikes in the continuous HR data as well as visual inspection. The data were reduced to HR in beats per minute for each half-second period during the baseline and experimental blocks. HR levels were averaged across the baseline period to create a HR baseline score. During experimental blocks, HR was averaged across the first 5 s of the preresponse interval and across the first 5 s in the postresponse interval before air blasts or startle probes were to be presented during trials that included them. A trial pre–postresponse HR change score was calculated as the difference between HR levels from pre- to postresponse intervals during experimental blocks, with negative scores indicating reductions in HR following participants’ responses. These pre–post HR change scores were used in main analyses of HR tension reduction below.

Aggression Paradigm Checks and Debriefing

Participants were administered the Multidimensional Personality Questionnaire—Brief Form (MPQ–BF; Patrick, Curtin, & Tellegen, 2002), which is a 155-item version of Tellegen’s (1982) MPQ. As a test of the validity of the aggression paradigm, we examined relationships between scores on the Aggression subscale of the MPQ–BF and participants’ shock intensity behavior in the subsequent aggression task. Only 105 participants (54 women, 51 men) had usable MPQ–BF data.

Following the experiment, participants completed a poststudy questionnaire (Verona et al., 2002) and were interviewed. Participants were asked to rate their impressions of the employee (1 = extremely unfavorable and 10 = extremely favorable). Two other 10-point items assessed the extent to which participants were motivated to increase shock intensities for instrumental (“to encourage better performance in the employee”) and hostile reasons (“upset at the employee”). Items on the poststudy questionnaire were used to examine the efficacy of the aggression paradigm.

During the poststudy interviews, six participants expressed suspicions about the cover story and, therefore, were excluded from analyses (i.e., not included in the sample size presented above). They either were not convinced that they were actually shocking another participant or realized that we wanted to instigate their aggression. These participants did not differ on demographic variables from the participants retained in the sample. During scheduled debriefing sessions, participants were informed of the true purposes of the study and allowed to voice any concerns and ask questions.

Results

Validity of Shock Intensity (Aggression) Measure

Overall mean shock intensities delivered across the practice and experimental blocks were positively related to the Aggression
subscale of the MPQ–BF \((r = .27, p < .01)\). Shock intensity was also correlated with poststudy questionnaire ratings of lower impressions of the employee \((r = -.28, p < .01)\) and with their self-reported instrumental and hostile motives \((rs = .54 \text{ and } .56, \text{ respectively}, ps < .001)\) for increasing shock responses. These correlations did not differ by gender. Thus, participants who administered the highest levels of aggression were the ones most frustrated by the employee and by the employee’s performance on the task, confirming the validity of the aggression paradigm.

Physiological Validation Checks

*Increases in tension during aggression procedure.* We wanted to establish that participants did experience an increase in autonomic tension during the aggression procedure (as a function of the frustrating confederate), as indicated by HR increases from baseline to the preresponse interval during the experiment. A mixed-model repeated measures analysis of variance (ANOVA) was conducted on average HR with time (baseline vs. preresponse interval) as a within-subjects factor and stress (low vs. high stress) and gender (female vs. male) as between-subjects factors. These analyses revealed a significant increase in HR from baseline to the preresponse interval, \(F(1, 103) = 14.9, p < .001, M_s = 71.3 \text{ and } 73.2\). There was also a significant main effect of gender, \(F(1, 103) = 4.6, p < .05\). Women exhibited higher average HR across baseline and preresponse intervals compared with men, but gender did not interact with time, so HR reactivity from baseline to preresponse intervals was similar for both genders: women: \(M_s = 73.3 \text{ and } 75.1\); men: \(M_s = 69.3 \text{ and } 71.5\). In effect, we confirmed that participants experienced increased emotional tension during the aggression procedure, which, according to catharsis theory, would purportedly be reduced following aggressive responding.

**Impersonal stress manipulation.** A Stress \(\times\) Gender ANOVA on startle sensitization revealed a significant main effect of stress condition, \(F(1, 109) = 31.20, p < .001\), with greater startle sensitization during high stress \((M = 1.12, SD = 0.60)\) than low stress \((M = 0.63, SD = 0.28)\) conditions. No gender main effect or interaction was observed for startle sensitization. This confirmed that the stress manipulation successfully primed negative affect in both male and female participants.

Goals 1 and 2: Tension Reduction and Impersonal Stress Exposure

The first goal of the study was to test the tension reduction component of catharsis theory, and the second goal was to examine whether tension reduction following aggression occurred when participants were concurrently exposed to an impersonal stressor (unrelated to the target of the aggression) during the aggression procedure. As per these goals, we conducted a Stress \(\times\) Gender \(\times\) Trial Type (aggression vs. nonaggression trials) mixed-model analysis of covariance (ANCOVA) on HR change from pre- to postresponse during the experimental blocks, with baseline HR entered as a covariate in analyses. Analyses revealed a significant Stress \(\times\) Trial Type interaction for pre–post response HR change scores, \(F(1, 101) = 4.05, p < .05\). Simple effects were first conducted within each trial type (aggression and nonaggression trials). For aggression trials (shock button response trials), we observed significant reductions in HR for the low-stress relative to high-stress group, \(F(1, 103) = 5.44, p < .02\), and the high-stress group exhibited an increase in HR following aggressive responding (see Figure 3). In contrast, simple effects test conducted on HR change scores following nonaggression responses (correct button response trials) did not reveal any effects of stress, \(F(1, 103) =

![Figure 3](image-url) Changes in heart rate from pre- to postresponse intervals during nonaggression and aggression trials as a function of stress group.
0.07. Follow-up analyses conducted within each stress condition failed to reveal a main effect of trial type in either stress condition. This suggests that HR changes following aggression trials were not significantly different from HR changes following nonaggression trials in either stress condition. Contrary to previous work (Hokanson & Edelman, 1966), gender did not interact with any of these effects.

To confirm that differences in participants’ general HR arousal during the two types of trials (aggression vs. nonaggression trials) did not account for the results obtained, we conducted a Stress × Gender × Trial Type (aggression vs. nonaggression) ANCOVA with HR baseline included as a covariate separately on preresponse and postresponse interval HR scores (not change scores). No effects of trial type were found for HR scores during the preresponse interval. Instead, analyses revealed a marginally significant Stress × Trial Type interaction for the postresponse interval, $F(1, 101) = 3.14, p = .08$, which showed that postresponse HR was reduced postaggression in low- versus high-stress participants. No other main or interaction effects were found. Thus, general HR arousal was not different for preresponse aggression versus nonaggression trials, suggesting that HR levels following aggression response intervals accounted for the HR change effects noted above.

In summary, HR was reduced following aggression trials in those in the low-stress relative to the high-stress condition. Thus, aggressing against the target produced significant reductions in HR during low stress relative to when an impersonal stressor was present. Stress condition did not affect HR change following nonaggressive responses. In fact, nonaggression responses seemed to have little impact on changes in HR pre- to postresponse (scores were close to zero) in both stress conditions (see Figure 3).

**Goal 3: Does Tension Reduction Predict Future Aggressive Behavior (Drive Reduction)?**

The third goal of the study was to test the drive reduction component of catharsis theory. In other words, do HR reductions following aggression (confirmed in the above analyses for the low-stress group) predict the (increased or decreased) probability of further aggression? To answer this question, we conducted hierarchical regression analyses on shock intensity (aggression) recorded from participants separately during the experimental blocks (concurrent aggression) and during the postrecovery block (subsequent aggression). Baseline HR and practice block shock intensity scores were entered in the first step of the analyses to covary participants’ preexisting differences in HR and aggressive tendencies from other effects on aggression. Gender and stress condition were entered in the second step of the analyses because of their a priori importance in the research questions. In the third step, we entered the HR change scores from pre- to postaggression response (only aggression trials) during experimental blocks as predictors of concurrent and subsequent aggression (shock intensity during experimental and postrecovery blocks, respectively).

The results of these analyses are summarized in Table 1. Regression analyses revealed that the first step of the analyses (particularly practice block aggression scores) accounted for a substantial portion of the variance in concurrent and subsequent aggression intensity (57% and 50%, respectively), as would be expected. For subsequent aggression intensity, male gender entered in the second step was a marginally significant predictor. Finally, HR change following aggression was a significant predictor of concurrent and subsequent aggression intensity, and this third step contributed a significant (albeit small) portion of the variance above other steps in both analyses. This result showed that, contrary to the drive reduction component of catharsis theory but consistent with other theorists (Hokanson, 1974), those who experienced the greatest reductions in HR following aggressive responding (relative to preresponding) delivered the highest intensity shocks to the confederate (concurrent aggression) and showed the highest aggression intensity in a future block (subsequent aggression). This result for subsequent aggression was similar for both low- and high-stress groups ($βs = −.11$ vs. $−.16$, respectively) and in the same direction for both men and women, although the beta for women was stronger than for men (but not significantly; $βs = −.26$ vs. $−.06$, respectively).

**Discussion**

The results of this research supported some portions but not others of the catharsis theory of aggression and extended previous work. Decreases in heart rate were observed in low- relative to high-stress conditions following aggressive responding but not following nonaggressive responding. This partly supports the emotional tension reduction component of catharsis theory. Exposure

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**Table 1**

Hierarchical Regression Analyses Predicting Shock Intensity During Experimental and Postrecovery Blocks From Covariates, Stress Condition, Gender, and Heart Rate Change Following Aggression Responding

<table>
<thead>
<tr>
<th>Variable</th>
<th>Concurrent aggression</th>
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<th>Subsequent aggression</th>
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<td>R²</td>
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<td></td>
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<tr>
<td>Baseline heart rate</td>
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<td>—</td>
<td>.51***</td>
<td>—</td>
</tr>
<tr>
<td>Practice block shock intensity</td>
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<td>.05</td>
<td>.74***</td>
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<tr>
<td><strong>Step 2</strong></td>
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</tr>
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<td>.52***</td>
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<tr>
<td>Heart rate change following aggression</td>
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<td>.15***</td>
</tr>
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*p < .10.  **p < .05.  ***p < .01.

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**Note:**

*aShock intensity during experimental blocks.  bShock intensity during postrecovery block
to an impersonal stress (low-stress condition) impeded heart rate reductions following aggression. Instead, heart rate arousal levels were maintained or increased from pre- to postaggression when participants were simultaneously exposed to an impersonal air blast stressor during the aggression experiment (high stress). In terms of the drive reduction component of catharsis theory, heart rate reductions following aggressive responding were associated in this study with the probability of increased (not decreased) aggression. Thus, the putative relief experienced following aggression does not lead to later cathartic reductions in aggressive drive; the drive reduction component of catharsis theory was not supported.

These data may speak to at least two different pathways through which an individual’s physiological reactions may lead to aggressive acting out, referred to here as the physiological reinforcement and physiological activation models of aggression. The first pathway is consistent with Hokanson’s (1974) learning model, in which physiological reductions following aggression reinforce subsequent aggressive acts. Two pieces of data from this study substantiate this model. First, engagement in aggression was associated with reductions in heart rate in low stress (relative to high stress), although engagement in a nonaggressive button press was not. This supports the idea that tension reduction can often follow aggressive responding when tension can be solely attributed to the target of the aggression. Second, those who showed the largest decreases in heart rate following aggressive responses were the ones who exhibited the most intense aggressive responding concurrently and in a subsequent block. Therefore, it can be suggested that relief from emotional tension reinforced more intense aggression in the participants. Of course, we cannot make cause-and-effect conclusions. It is possible that the most aggressive individuals coming in to the experiment were the ones who experienced the greatest reduction in heart rate after responding with aggression (instead of the other way around). However, we were able to show in our analyses that, even when practice block (preexisting) aggression scores were covaried, heart rate reductions following aggressive responding during the experimental blocks still predicted the most extreme aggression in the postrecovery block (subsequent aggression). Thus, it is likely that the tension reduction experienced by the participants during the experiment had an effect on the amount of aggression they later delivered to the confederate.

Despite previous work (Hokanson & Edelman, 1966; Lai & Linden, 1992), we did not detect gender differences in the effects of aggressive versus nonaggressive responding on heart rate reductions. There may be several reasons for this. It is possible that gender norms may have changed enough in the last few decades since Hokanson’s (1974) classic studies so that mild aggression in women may be reinforced (and reinforcing) in today’s society. Newer generations of women have potentially been rewarded (e.g., obtain better job positions) for demonstrating assertiveness and more direct confrontational behaviors. This explanation is plausible in light of the fact that endorsements of the psychological benefits of cathartic anger expression in popular culture are typically published in self-help books and publications geared toward women. A methodological reason could be that the frustrating aggression paradigm used in the current study (monetary goal-blocking by confederate) may have similar effects on both genders in terms of tension reduction following aggressive acts. A different interpersonal situation, including direct provocation or insult used in earlier studies, may result in less tension reduction following aggression among women than men (Lai & Linden, 1992), partly because women may experience other types of emotions (e.g., depression or anxiety) along with anger in these provoking situations (Verona, Reed, Curtin, & Pole, 2007). This should be investigated in future research on this topic.

It was interesting that in the current study we found that high-stress exposure (the impersonal stressor) prevented heart rate reductions following aggressive responding. A possible explanation for this moderating effect of impersonal stress is that when arousal can be attributed to a stressor unrelated to the interpersonal exchange, aggressive responding against the target does not help reduce physiological tension, at least in terms of heart rate reductions. This interpretation is consistent with Bushman’s work on catharsis expectations (Bushman, Baumeister, & Phillips, 2001). Participants in low stress who attributed their emotional tension solely to the frustrating confederate expected the aggressive act against the target to relieve tension, and so it did. On the other hand, those concurrently exposed to high stress may not have expected aggressive retaliation against the frustrating confederate to influence their negative affect given that their distress was partly elicited by the air blast stressor; thus, they did not experience the physiological benefits of aggression against the target. Berkowitz (1962) also has written that doing injury to the source of one’s negative arousal or frustration can lead to pleasant relief because one has gratified one’s initial goal (aggressive instigation). In high stress, the participants were aware that their aggressive responses were not going to hurt the source of their negative affect, thus preventing them from experiencing gratification from their aggression. The present findings then suggest that in real-world situations involving instigating contexts that include impersonal stressors, individuals should not expect aggressive reactions directed at others to produce much relief during these everyday experiences of stress (e.g., too much work to do).

On the basis of these findings involving stress condition, we contend that a second pathway for aggression involves mutual physiological activation. This pathway is consistent with Berkowitz’s (1990) neoassociationistic model of aggression, in which he stipulated that stress and the resulting emotional distress prime aggressive behaviors in individuals when no other recourse is available to them. This is because both negative affect and aggression are parts of a broader defensive motivation system that governs responses to threat. Thus, activation of the negative affective system will elicit concomitant activation in the behavioral system, and vice versa. In our study, we found that the high-stress group did not show reductions in heart rate, and nonsignificant increases in heart rate, from pre- to postaggressive response. On the basis of Berkowitz’s model, this finding indicates that not only does negative affect and physiological reactivity lead to aggression, but engagement in aggression leads to increases in negative affect and physiological reactivity that have already been primed by an existing stressor. Indeed, recent work by Haller, Kruk, and colleagues (Halasz, Liposits, Kruk, & Haller, 2002; Kruk, Halasz, Meelis, & Haller, 2004) has indicated that stimulating attack-related centers in the rat brain simultaneously induced strong hypothalamic–pituitary–adrenal axis and amygdala activation, implying that stimulation of aggressive centers can also activate stress responses. The study of this feedback loop between affective–stress centers and behavioral responses (stress reactivity can prime aggression, which in turn maintains negative
affective state) in humans should be a priority of future research in the aggression area (cf. Verona & Kilmer, 2007).

**Strengths, Limitations, and Implications**

The current study has several strengths. These include testing both tension reduction and drive reduction components of catharsis theory, as well as examining the relationship between heart rate reductions at one time point and aggressive behavior exhibited in a subsequent portion of the study. Prior to this study, these types of analyses had not been conducted on catharsis theory. Another strength of the current study includes the integration of classic models of catharsis and newer models of stress-aggression relationships that create a more nuanced picture of physiology-aggression relationships. Nonetheless, there are limitations to this study that warrant further research. First, the laboratory context and manipulations may not adequately reflect the situations encountered by men and women in their everyday lives. For example, only overt physical aggression was measured in this experiment, and this could have implications for finding gender differences. It is possible that women versus men would show even more heart rate reductions following aggression if verbal aggression or more covert forms of aggression (including social manipulation) more commonly used by women would have been measured (Bjorkqvist, Larsen, & Kaukiainen, 1992). Another limitation of this study is that we included only one physiological measure of tension or arousal (heart rate). The acoustic startle measure was used only to validate the stress manipulation and was not included in the analyses as a dependent variable. It is likely that different measures of autonomic activity may yield a different set of results. At least, if tension reduction afforded by aggressive responding is fairly generalized, it would be helpful to replicate these results using blood pressure or vagal tone.

Despite some limitations, these data have implications for our understanding of aggression instigation. The data suggest that the relationship between physiological reactvity and aggression is a complex one and that different aspects of the instigating context (interpersonal and impersonal components) affect the interaction between physiological and behavioral systems. It also suggests that, although engaging in mildly aggressive behavior may be associated with short-term relief, it may have detrimental long-term effects in terms of aggression potential. This has implications for treatment and prevention of aggression. Some therapists and educators continue to encourage aggressive expressions of negative affect in therapy or through sports activities for children (Bennett, 1991; Schaefer & Mattei, 2005). However, as stipulated by Bushman and colleagues (Bushman, 2002; Bushman et al., 2001), engaging in anger-venting activities including hitting pillows or punching bags may not help reduce aggressive drives. In truth, our data are the first to demonstrate that it is those who experience the most emotional tension reduction following these anger-venting activities that may be at highest risk for subsequent aggression. It may be more fruitful to help these individuals develop other strategies (distraction) to deal with angry feelings (Lewis & Bucher, 1992). Individuals can be taught to reward themselves for engaging in alternative strategies; parents and mental health providers can systematically reinforce children or clients with anger problems for using distraction or healthy communica-

**References**


Halasz, J., Liposits, Z., Kruk, M. R., & Hailer, J. (2002). Neural background of glucocorticoid dysfunction-induced abnormal aggression in...


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