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# The Acoustic Startle Response in High- and Low-Hostiles as a Function of Pain-Stress[1]

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## SUMMARY

**Background:** Highly hostile individuals exhibit exaggerated physiological responses to stressors that are associated with cardiovascular disease and premature death. Evidence that high-hostiles have altered right hemispheric functioning has lead to a right hemisphere model of hostility. The Acoustic Startle Response (ASR) has been used to examine individual differences in emotional state and trait and can be potentiated by aversive stimuli. In this experiment, we hypothesized that high-hostiles would have larger ASRs when compared to low-hostiles, and would experience ASR potentiation following a cold pressor (CP).

**Material/ Methods:** Forty high- and low-hostile undergraduate men heard acoustic startle probes before and after a CP. ASRs were measured using electromyography (EMG) of the orbicularis occuli. A 2 X 2 mixed factorial ANOVA was conducted with Group (high- and low-hostile) as the between-subjects factor, Condition (pre- and post-CP) as the within-subjects factor, and EMG magnitude (mV) of the ASR as the dependent variable.

**Results:** A significant Group main effect indicated larger ASRs in high-hostiles. A significant Group X Condition interaction was found. There were no significant group differences before the CP, but low-hostiles' ASR decreased significantly after the CP, while high-hostiles' ASR did not change significantly.

**Conclusions:** High-hostiles' ASR failed to habituate, indicating a fixed and exaggerated physiological response to stressors on the reflex level. Low levels of hostility may be associated with a more flexible and reduced physiological response to stressors.

**Key words:** ASR, eye blink, cold pressor, right brain, hostility

[1] The authors report no conflicts of interest for this research. This research was approved by the Institutional Review Board of Virginia Polytechnic Institute & State University. This paper has not been published but is made available through Virginia Tech's Electronic Dissertation (ETD) Library.

# BACKGROUND

The use of the Acoustic Startle Response (ASR) as an index of emotion and neurologic impairment has gained considerable popularity for examining individual differences in emotional state and trait (Lang, 1995; Hoffman, 1997). The ASR is a defensive withdrawal behavior caused by an unexpected loud stimulus that induces a brainstem reflex evoking autonomic and involuntary muscle responses. Although these effects reflect automatic, subcortical mechanisms (Leitner, Powers, Stitt, & Hoffman, 1981), the startle magnitude appears to be sensitive to processes mediated at higher levels of the nervous system such as selective attention and habituation (Bohlin, Graham, Silverstein, & Hackley, 1981). ASR potentiation during aversive compared with pleasant and/or neutral conditions has been repeatedly demonstrated with pictures (Bradley, Cuthbert, & Lang, 1990, 1991, 1996*)*, film clips (Jansen & Frijda, 1994), mental imagery (Vrana & Lang, 1990; Cook, Hawk, Davis, & Stevenson, 1991; Hawk, Stevenson, & Cook, 1992), shock threat (Grillon, Ameli, Foot, & Davis, 1993), odors (Miltner, Matjak, Braun & Diekmann, 1994), and pheromones collected from subjects in a highly anxious state (Pause, Ohrt, Sojka, Ferstl & Prehn, 2006). The ASR magnitude has also been shown to vary with certain emotional dispositions such as non-clinical depression (Mneimne et al., 2008), generalized anxiety disorder (Ray, Molnar, Aikins, Newman, Borkovec, & Castonguay, 2009), inhibited adolescents with anxiety disorders (Reeb-Sutherland et al., 2009), defensiveness (LaRowe, 2003), and anxiety (Temple & Cook, 2007). In addition, the ASR has been shown to be sensitive to affective, cognitive, and arousal-related deficits resulting from neurological impairment (Morris, Bradley, Bowers, Lang, & Heilman; 1991).

Hostility is a personality trait with poor health outcomes such as cardiovascular disease and premature death (Everson et al. 1997), and is predictive of exaggerated physiological responses to stressors including heightened cardiovascular reactivity to stressful situations (Rhodes, Harrison, & Demaree, 2002; Smith, Glazer, Ruiz, & Gallo, 2004), and increased blood pressure and heart rate (Demaree & Harrison, 1997; Keefe, Castell, & Blumenthal, 1986). Research has shown that individuals with high levels of hostility (high-hostiles) experience a negative affect bias in the tachiscopic presentation of emotional stimuli (Shenal & Harrison, 2004), dichotic listening tests (Demaree & Harrison, 1997; Snyder & Harrison, 1997), affective verbal learning tests (Mollet & Harrison, 2006), and affective recognition (Harrison & Gorelczenko, 1990). High-hostiles have been shown to have altered right hemispheric functioning (Williamson & Harrison, 2003) with right cerebrum activation in response to pain stress (Demaree & Harrison, 1997). Evidence of an altered right hemisphere (Demaree, Everhart, Youngstrom, & Harrison, 2005; Demaree & Harrison, 1997; Everhart & Harrison, 1995; Williamson & Harrison, 2003) has lead this laboratory to propose a right hemisphere model of hostility suggesting that hostility may be derived from the diminished right frontal lobe regulatory capacity of autonomic and emotional functions while processing concurrent right frontal lobe stressors and demands (Fos-

ter, Williamson, and Harrison, 2005). Other researchers advocate that impaired function in the prefrontal cortex, be it from a lesion or metabolism as measured by regional cerebral blood flow (rCBF) is associated with hostile behaviors (Shapiro et al. (1994) .

Several authors provide evidence that the right hemisphere and particularly the central nucleus of the right amygdala, modulates the startle circuit's activity via an ipsilateral projection of the caudal amygdalofugal pathway to the nucleus reticularis pontis caudalis (NRPC) which projects to the facial motor nucleus that innervates the orbicularis oculi muscle (Grillon & Davis, 1995). This later circuit is proposed to be responsible for the execution of affective modulation of the acoustic startle response in humans. Affective startle modification is a robust phenomenon (Cook, 1999) and allows us to examine activity in the brain-stem circuit and possibly the cerebral asymmetries in its descending modulatory pathways that include frontal corticofugal pathways that are involved in habituation. For example, studies of the effects of human frontal lobe lesions on the habituation to peripheral vision suggest that the frontal cortex increases habituation of attention to redundant stimuli (Menemeier, Chatterjee, Watson, Wertman, Carter, & Heilman, 1994).

The purpose of this experiment is to extend previous findings of exaggerated physiological responses in high-hostiles to the ASR. Specifically, it is hypothesized that high-hostiles will have larger ASR magnitudes compared to low-hostiles due to diminished right frontal lobe regulatory control (Foster, Williamson, and Harrison, 2004). It is hypothesized that this diminished frontal lobe regulatory control interferes with the habituation to redundant stimuli, in this case acoustic startle probes. Because high-hostiles experience high levels of negative affect and this is conducive to valence modification of the ASR (Cook, 1997), it is also hypothesized that high-hostiles will show ASR potentiation after experiencing a negative stimulus, a cold pressor.

# METHODS AND MATERIALS

Twenty low-hostile and twenty high-hostile right-handed undergraduate males participants were recruited. They completed the Cook-Medley Hostility Scale (CMHS) (Cook & Medley, 1954) as part of the on-line screener survey. Participants who obtained a score of 19 or lower on the CMHS were classified as low-hostiles and participants who obtained a score of 29 or higher were classified as high-hostiles consistent with previous research (Williamson & Harrison, 2003; Shenal & Harrison, 2003; Herridge, et al., 2004; Rhodes et al., 2002). Participants completed the Coren, Porac, and Duncan Laterality Questionnaire as part of the on-line screener survey to determine sufficient right hemibody preference (CPD; Coren, Porac, & Duncan, 1979). Participants scoring +7 or above were included in this experiment. Participants completed the Medical History Questionnaire as part of the on-line screener survey. The Medical History Questionnaire assesses neurological trauma, psychiatric disorders, drug use, and major

medical disorders. Participants had to report no significant medical history to be included.

### Stimuli and EMG

The acoustic startle stimulus was a 50 millisecond, 100 dB (SPL; A scale) broadband white noise with instantaneous onset presented binaurally through headphones from a portable compact disc player. Acoustic startle probes were spaced pseudorandomly apart from each other in time (no longer than 15 seconds apart, standard deviation of 7.5 seconds) to reduce the possible effects of temporal learning. The laboratory chamber consisted of a chair facing into a desk. The cold pressor equipment was located out of view to the left of the participant. The experimenter and physiological recording equipment were located next to but out of view of the participant. The ice water for the cold pressor task was maintained in a small ice cooler at ($\pm$1 3 degrees Celsius). Electromyographical (EMG) measures of the acoustic startle response were recorded using the BioPac MP36 Bioamplifier and Acqknowledge acquisition software. Eyeblink startle responses were measured from both the left and the right orbicularis oculi using two pairs of 5 millimeter Ag/AgCl electrodes placed directly under the pupil in forward gaze. Raw EMG signals was sampled at 1000 Hz and filtered to minimize the noise that is above and below the EMG signal frequency band using a 1000 Hz low-pass filter and a 5 Hz high pass filter. Raw EMG (mV) was integrated and the peak EMG amplitude of each ASR was calculated by subtracting the mean EMG amplitude recorded during the baseline and then the natural log transformed prior to analyses.

### Procedure

Participants meeting the requirements previously stated were invited via e-mail to participate in the laboratory session of the experiment. During the first baseline recording EMG was recorded for two minutes while participants looked straightforward. Following baseline recording, participants were told that they were going to hear a series of sounds lasting approximately a minute and that they were to simply remain seated throughout. EMG was recorded continuously while the 10 startle probes were delivered through headphones. After the startle probes, a recovery period lasted one minute, after which an EMG was recorded again to serve as a baseline measure for the cold pressor condition that followed. Next, in the cold pressor condition, participants completed a cold pressor task for 45 seconds by placing their left hand in the ice water. Ten acoustic startle probes were then presented through the headphones. Participants were debriefed and thanked for their participation.

## RESULTS

All participants ($N$ = 40) were right-handed undergraduate male students age ($M$ = 19.3, $SD$ = 0.90). High hostiles had significantly higher scores on the CMHS

(*M* = 32.7, *SD* = 3.03) than low hostiles (*M* = 16.3, *SD* = 3.2), *t*(*1*,38) = 16.52, *p* < .0001. To analyze the ASR magnitudes, a 2 x 2 mixed-design analysis of variance (ANOVA) was conducted with EMG (mV) as the dependent variable, Group (high- and low- hostile) as the between-subjects factor, and Condition (pre and post cold pressor), as the within-subjects factor. There was a significant main effect for Group *F*(1,38) = 5.86, *p* < .05, with high hostiles having larger ASR magnitudes (*M* = 0.593) than low hostiles (*M* = 0.405). There was a significant main effect for Condition F(1.38) = 36.02, *p* < .0001, indicating larger ASR magnitudes in the pre cold pressor condition (*M* = 0.551) than in the post cold pressor condition (*M* = 0.446). There was a significant Group X Condition interaction *F*(1,38) = 7.27, *p* < .05. In order to probe the Group X Condition interaction, follow up one-way ANOVAs were conducted. A one-way ANOVA with Group as the between-subjects factor was conducted to compare the groups' ASR magnitudes in the pre cold pressor condition. The results approached significance F(1.38) = 3.19, *p* = .08, with high hostiles (*M* = 0.622) having a larger ASR magnitude than low hostiles (*M* = 0.481). A one-way ANOVA comparing the groups after the cold pressor was significant F(1.38) = 8.58, *p* < .01, indicating that high hostiles had a larger ASR magnitude (*M* = 0.564) than low hostiles (*M* = 0.329). In order to test the effect of the cold pressor task on each group individually, one-way ANOVAs were performed with Condition as the within-subjects factor. High hostiles' ASR magnitude did not change significantly from before (*M* = 0.622) to after the cold pressor task (*M* = 0.564), whereas the low-hostile's ASR magnitude decreased significantly from before (*M* = 0.481) to after the cold pressor (*M* = 0.329), *F*(1,19) = 3.71, *p* <.0001.

# DISCUSSION

The prediction that high-hostiles would exhibit a larger ASR than low-hostiles was supported and is consistent with the psychophysiological reactivity model (Smith, 1994) that links hostility to cardiovascular disease and negative health outcomes. According to this model, hostile persons are more vigilant for conflicts in their environment and have exaggerated physiological responses to a variety of stressors. Previous experiments have generally shown high hostiles, compared to low hostiles, to have increased cardiovascular reactivity to a stressor (cold pressor) (Demaree & Harrison, 1997), and this experiment provided evidence that the exaggerated physiological response occurs on a simple reflex level. Although hostility is considered a trait with a strong cognitive component, physiologically, high hostiles' reactivity to stressors is seen in the startle response, which occurs before the cognition and appraisal of the stimulus.

The hypothesis that the cold pressor would increase the ASR magnitude was not supported. Rather, a significant main effect for the condition indicated that startle responses decreased significantly after the cold pressor. There are several possible reasons for this finding. All participants completed the cold pressor task after the first series of startle probes. Thus, with the law of initial values in mind, it could be expected that startle response magnitudes would decrease over time

with repeated exposure to the stimulus. The significant ASR magnitude decrease may also be related to expectancies; after the cold pressor, participants were familiar with the stimulus and knew what to expect. Interestingly, the low-hostiles' ASR magnitude decreased significantly after the cold pressor while the high-hostiles' ASR magnitude did not change significantly. The latter case could be interpreted as a failure to habituate, which is also consistent with the hypothesis of diminished frontal lobe inhibitory capacity.

It is possible that the cold pressor recruited frontal systems in the low hostiles, leading to reflex inhibition and ASR habituation, whereas frontal lobe inhibitory capacity was exceeded by the aversive stimuli in the high-hostiles. If this is the case, it would be consistent with our capacity theory (Shenal & Harrison, 2004; Foster et al., 2005; see Carmona et. Al, 2009) which relates the profile of emotional bias and cardiovascular lability seen in high hostiles to diminished right frontal lobe capacity and its susceptibility to decompensation. Frontal systems, in particular the cingulate gyrus, mediate the startle response through corticofugal projections to the brainstem nuclei mediating the orbiculris occuli muscle contractions. The right hemisphere, and particularly the central nucleus of the amygdala, modulate the startle circuit's activity (Grillon & Davis, 1995) and high hostiles have been shown to have altered right hemispheric functioning (Williamson & Harrison, 2003), with right cerebrum activation in response to pain stress (Demaree & Harrison, 1997).

The ASR response pattern seen in high-hostiles may have several clinical implications. High hostiles' startle responses remained stable across conditions indicating a rigid and inflexible physiological response style that is identified as a risk factor for developing cardiovascular disease (Smith, 1994). Low-hostiles' startle responses decreased significantly after the cold pressor suggesting that they may employ effective strategies for coping with acute stressors whereas high-hostiles did not. Future investigations should examine how low-hostiles cognitively appraise stressful scenarios, and determine if those strategies can be employed by high-hostiles to improve their reactivity to stress.

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