Journal of Abnormal Psychology
2009, Vol. 118, No. 1, 131–145

0021-843X/09/$12.00 DOI: 10.1037/a0014376

Stress-Induced Asymmetric Frontal Brain Activity and Aggression Risk

Edelyn Verona and Naomi Sadeh
University of Illinois at Urbana-Champaign

John J. Curtin
University of Wisconsin—Madison

Impersonal stressors, not only interpersonal provocation, can instigate aggression through an associative network linking negative emotions to behavioral activation (L. Berkowitz, 1990). Research has not examined the brain mechanisms that are engaged by different types of stress and serve to promote hostility and aggression. The present study examined whether stress exposure elicits more left than right frontal brain activity implicated in behavioral approach motivation and whether this lateralized brain activity predicts stress-induced aggression and hostile/aggressive tendencies. Results showed that (a) participants in the impersonal (assigned to stress by a computer) and interpersonal (assigned to stress by a provoking confederate) stress conditions both showed more left than right frontal electroencephalogram activity after condition assignment and stress exposure and (b) the 2 stress groups exhibited subsequent increases in aggression relative to the no-stress group. Importantly, left frontal asymmetry in response to stress exposure predicted increases in subsequent aggressive behavior, a finding that did not emerge in the no-stress condition. Thus, both the interpersonal and impersonal stressors impacted state changes in brain activity related to behavioral approach, suggesting that stress reactivity involving approach activation represents risk for behavioral dysregulation.

Keywords: aggression, EEG frontal asymmetry, stress, approach motivation, hostility

Supplemental materials: http://dx.doi.org/10.1037/a0014376.supp

Stress exposure often enhances hostile reactions and behavioral disinhibition and instigates aggressive behavior in humans and nonhuman animals (Berkowitz, 1990; Lutz, Marinus, Chase, Meyer, & Novak, 2003). This effect is robust in situations involving direct provocation or intrusion by another organism (Bettencourt & Miller, 1996; Bushman & Baumeister, 1998); however, other work has highlighted the role of seemingly impersonal stressors (e.g., hot rooms, pain, cold water immersion) in eliciting hostile and aggressive reactions (Berkowitz, 1990; Verona & Kilmer, 2007). Few studies have examined the brain mechanisms activated by different forms of stress exposure that may serve to increase risk for aggression, although animal research has linked stress-related release of glucocorticoids with attack behaviors in rats (Kruk, Halasz, Meelis, & Haller, 2004). In the human literature, recent work has shown that behavioral approach motivation, as measured by frontal electroencephalogram (EEG)-alpha activity, is elicited by anger inductions (for a review, see Harmon-Jones, 2004). No research thus far has examined whether impersonal and interpersonal stressors elicit similar or distinct patterns of brain activity. The current study investigated whether (a) interpersonal and impersonal stressors elicit similar or different patterns of frontal alpha activity that relate to emotional and aggressive reactions and (b) whether approach-related brain activity, regardless of the type of stressor, predicts stress-induced behavioral reactions in the form of aggression. This work is important in that it has the potential to identify mechanisms by which stress induces maladaptive behavioral-approach reactions, which have implications for the study of aggressive and other dysregulated behaviors that occur in response to negative emotional states.

Stress, Behavioral Priming, and Aggression

Acute exposure to stressors and concomitant negative emotional experiences have been linked to engagement in dysregulated behaviors, including aggressive behaviors (Berkowitz, 1990; Verona, Patrick, & Lang, 2002) and alcohol/drug urges and binges (Nesic & Duka, 2006; Stewart, 2003). To explore the mechanisms underlying the association between acute stress and dysregulated behaviors, researchers have used laboratory paradigms that induce aversive instigation and measure aggressive behavior. In most studies (e.g., Bettencourt & Miller, 1996; Bushman & Baumeister, 1998), the aversive context involves interpersonal provocation, which is defined by a direct insult or offense by another person. Indeed, the most reliable instigation of aggression appears to occur in response to interpersonally provoking situations (Bettencourt & Miller, 1996) and the attribution of threat to another person (Dill & Anderson, 1995; Ellsworth & Smith, 1988). However, Berkowitz (Berkowitz, 1990; Berkowitz & Harmon-Jones, 2004) postulated that impersonal stressors, defined as stressors that lack direct provocation by an instigator, evoke hostility in individuals and subsequent aggression toward an innocent target.

Edelyn Verona and Naomi Sadeh, Department of Psychology, University of Illinois at Urbana-Champaign; John J. Curtin, Department of Psychology, University of Wisconsin—Madison.

Naomi Sadeh was supported by National Research Service Award MH19554 from the National Institute of Mental Health. The authors would like to thank Becky Levin, Greg Miller, and Jennifer Stewart for their comments on an earlier version of the article and Keith Bredemeier, Lindsey Francke, Ashley Kilmer, Carly Parnitzke, and Matt Pfaff for their help with data collection.

Correspondence concerning this article should be addressed to Edelyn Verona, University of Illinois at Urbana-Champaign, Department of Psychology, 603 East Daniel Street, Champaign, IL 61820. E-mail: everona@illinois.edu

Evidence that acute stress exposure primes the initiation of aggressive behaviors, particularly in predisposed individuals, stems partly from work by Lang and colleagues (Lang, 1979; Lang, Bradley, & Cuthbert, 1990), who argue that negative emotional states are evolutionarily adaptive in that they promote survival by priming defensive behavior under threatening circumstances. Thus, negative emotional states can increase risk for aggressive behavior via an associative network that primes individuals for defensive action. Support for the associative network model stems from research that indicates that aversive stimulation does not necessarily have to involve direct provocation to prime hostility and aggression. For example, impersonal stressors such as foul odors (Rotton, 1979), hot rooms (Anderson, Anderson, & Deuser, 1996), cold water (Berkowitz, Cochran, & Embree, 1981), and air blasts (Verona et al., 2002) also result in hostile judgments of a stranger or delivery of electric shock to a confederate within a laboratory paradigm. Thus, the extant literature suggests that acute stress exposure instigates hostility and aggression regardless of the impersonal or interpersonal nature of the context. The brain processes responsible for these outcomes in the two types of stress require further clarification. Given that anger and aggression have been linked to approach motivational tendencies (e.g., Harmon-Jones, 2003), an extension of the associative network models of aggression suggests that stress exposure induces prefrontal processes related to approach activation that in turn increase the risk for engaging in aggression under stress. This hypothesis has never been tested. The present study was developed to directly compare brain activity induced by impersonal stress with that induced by interpersonal stress and to examine whether either type of stress leads to aggression as a result of similar motivational processes.

It has been suggested in other literatures that, in contrast to associative network models that suggest both types of stress lead to behavioral activation (approach-motivation hypothesis), stress effects are context-specific (stress-specific hypothesis; Ellsworth & Smith, 1988). In the latter, the defensive behavior primed by the experience of negative emotions is said to differ depending on the context in which the emotion is elicited (Berkowitz, 1989). Specifically, stress induced by a provoking target may prime individuals to respond with an approach response (e.g., fight), whereas aversive contexts not involving a provoking target may elicit a withdrawal response (e.g., flight). Given the activation of competing approach-versus-withdrawal behavioral tendencies that can be triggered, individuals are likely to experience different types of subjective emotions in response to the different types of stress. For instance, the experience of fear is thought to occur in situations that evoke a strong withdrawal motivation, whereas feelings of hostility are thought to emerge in contexts that evoke a strong approach motivation (Berkowitz, 1989). This stress-specific hypothesis for differential behavioral motivations requires empirical exploration, particularly since the research on impersonal stress effects on aggression cited above is not supportive of this contention. The present study is the first to examine whether different forms of stress exposure induce similar or distinct brain responses and subjective emotions and whether these indexes of approach or withdrawal motivational states result in differential risk for aggressive reactions.

Behavioral Approach, Frontal Alpha Activity, and Hostile Reactions

In prior work, our research team proposed that stress exposure, even if impersonal in nature, activates brain mechanisms involved in approach motivation, and it is this approach motivational response to stress that explains risk for stress-induced aggression in certain individuals (see Verona & Kilmer, 2007). Approach motivation typically refers to action tendencies directed at moving toward a target (Wacker, Heldmann, & Stemmler, 2003). For decades, researchers have conceptualized problems with externalizing behaviors and aggression partly as a consequence of a hyperactive behavioral approach system on the basis of evidence that a positive relationship exists between aspects of approach (e.g., activity level) and anger responses among young children (Putnam, Ellis, & Rothbart, 2001). Depue and Iacono (1989) indicated that the behavioral facilitation system (akin to Jeffrey Gray's, 1982, behavioral activation system) governs responses to frustration and reward blocking, which can take the form of aggressive behavior toward the obstacle or threat. Researchers have long recognized that, more pertinent to psychopathology, tendencies toward negative affect combined with thrill seeking are personality risk factors for dysregulated behavior, including antisocial deviance, aggression, and impulsive self-harm (Krueger, 1999; Miller & Lynam, 2006; Sher & Trull, 1994; Verona, Patrick, & Joiner, 2001). Prior work has not directly assessed stress-induced changes in brain mechanisms related to approach motivation and how these patterns of brain activity contribute to the experience of hostility or manifestation of dysregulated behaviors, which was a goal of the present study.

Recent research on motivational systems has emerged from work conducted with electroencephalogram (EEG) recordings of prefrontal brain activity that has linked tendencies toward behavioral approach (e.g., higher sensitivities to reward, hypomania, and reward-seeking traits) to relatively greater left than right anterior brain activity (Coan & Allen, 2003; Davidson, 1983; Pizzagalli, Sherwood, Henriques, & Davidson, 2005; Sutton & Davidson, 1997). Important for the present study, Harmon-Jones and colleagues (e.g., Harmon-Jones, 2004; Harmon-Jones & Allen, 1998) have argued that more left than right frontal activity is indicative of behavioral approach, regardless of the valence of the emotional state. He demonstrated this empirically by inducing the emotion of anger, which is commonly assumed to be negative in valence but has approach motivational properties and elicits more left than right frontal activity. The link between anger/aggression and relatively greater left than right prefrontal activity as seen by EEG has been replicated with measures of anger, aggressive behavior, and externalizing symptoms in children across research labs (Forbes et al., 2006; Fox & Davidson, 1988; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001). However, this stress–EEG–behavior link has not been experimentally manipulated and tested directly in prior work. Research on the effects of stress on approach-related brain activity in the prefrontal cortices and its relationship to aggressive behavior is timely, given the demonstrated empirical association between approach motivation and aggressive responding (Harmon-Jones, 2004).

Emotional Challenges and Frontal EEG Asymmetry

A large body of literature spanning more than 2 decades has investigated the role of frontal alpha activity with EEG. Davidson (1983, 2004) and other theorists (Heller, 1990, 1993; Heller & Nitschke, 1998; Tucker, 1981) have suggested that prefrontal regions of the brain are asymmetrically involved in the regulation of emotional states. More relative right prefrontal activation, as measured by EEG, is implicated in the risk for affective disorders (e.g., depression, anxiety) and withdrawal emotions (e.g., disgust, sadness), whereas more left prefrontal activity is generally associated with appetitive emotions (e.g., excitement, happiness). Some of this work has relied upon a resting state for EEG recordings (also see Harmon-Jones, Lueck, Fearn, & Harmon-Jones, 2006), which may have led to inconsistent findings across studies. As outlined by Hagemann (2004), it is not uncommon for inconsistent findings to emerge across studies that employ resting EEG, an outcome that he attributes to several factors, including the recording methodology used, the treatment of artifacts, and the presence of both state and trait variability in the EEG. Coan, Allen, and McKnight (2006) advocate for a "capability model" of prefrontal activity, positing that "individual differences [in frontal alpha asymmetry] are best thought of as interactions between the emotional demands of specific situations and the emotion-regulatory *abilities* individuals bring to those situations" (p. 198; italics in original). Indeed, Coan et al. (2006) observed more reliable relationships between EEG asymmetry and criterion variables during emotional challenges compared with those involving resting EEG recordings, even when they instituted methodological variations in the measurement of EEG asymmetry (Coan et al., 2006). For this reason, the present study used emotional challenges to elicit changes in brain activity and examined their relationship to aggressive behavior.

Present Study

The present study sought to integrate research on the stress–aggression relationship with recent work emphasizing motivational systems and brain processes that promote anger and aggression. Specifically, we examined whether different types of stress exposure, either interpersonal or impersonal in nature, would lead to distinct or similar responses in frontal brain activity, reports of emotion, and aggressive behavior. To test these hypotheses, we created equivalent stress conditions but varied whether participants attributed the stress exposure to an interpersonal or impersonal situation.

The first major goal of the study was to clarify whether impersonal and interpersonal stressors are associated with similar or distinct prefrontal activity, subjective emotions, and aggressive behavior. Although associative network models of aggression would suggest similar processes underlie increases in aggression under either type of stress, this analysis has yet to be conducted. The *approach-motivation hypothesis* of stress-induced aggression (Hypothesis 1) predicts that either type of stressor will foster (a) left lateralized frontal brain activity indicative of approach motivation, (b) self-reports of hostility, and (c) heightened aggressive behavior directed at a target. The alternative *stress-specific hypothesis* (Hypothesis 2) predicts that the two stress manipulations will activate different motivational states depending on participants' attributions about the stressor (Berkowitz, 1990; Lang, 1979). That is, the stress-specific hypothesis posits that the impersonal stressor will lead to behavioral withdrawal, since it does not involve direct provocation, and the interpersonal stressor will result in behavioral approach, as it involves a provoking target. In contrast to the approach-motivation hypothesis, the stress-specific hypothesis predicts greater right than left frontal brain activity and increases in fear, but not anger or aggression, in the impersonal relative to the interpersonal stress group. On the basis of evidence that both impersonal and interpersonal stressors elicit hostile and aggressive reactions in laboratory experiments (Berkowitz, 1990), we expected our results to support the approach-motivation rather than the stress-specific hypothesis.

The second major goal of the study was to examine whether among participants exposed to stress, state changes in frontal alpha asymmetry (approach activation) would predict increases in subsequent aggressive behavior and show positive associations with aggressive personality traits. These analyses would help establish that stress exposure and frontal processes related to approach activation serve as interactive risk factors for aggressive behavior and hostile dispositions.

Method

Participants

The sample consisted of 135 volunteers (61 women) recruited from a university psychology department participant pool and flyers posted on campus and in the community. Participants ranged in age from 18 to 40 years ($M = 24.6$, $SD = 6.46$), with the majority of participants identifying as Caucasian ($n = 80$; 60.6%) and the others identifying as African American ($n = 16$; 12.1%), Asian ($n = 14$; 10.6%), Hispanic ($n = 12$; 9.1%), or other ($n = 10$; 7.6%). The sample was composed mostly of current undergraduate college students ($n = 71$; 52.6%), with the remaining participants identifying as nonpsychology graduate students ($n = 24$; 17.8%) or community residents ($n = 40$; 29.6%). Of the community residents, 6 had a high school diploma (4.5%), 13 attended some college (9.8%), 16 completed a bachelor's degree (12%), and 3 obtained a degree in higher education (2.3%). Three people did not report ethnicity, and 2 people did not report level of education. Individuals recruited from the participant pool and those recruited broadly from campus or the community received course credit and $10/hr compensation, respectively, for their participation. Prior to participation, individuals were screened with the following inclusion criteria: 18–40 years old, right-handed, and normal hearing. Informed written consent was obtained from all individuals prior to the study.

Due to missing data resulting from equipment failure, 13 participants were excluded from analysis of the aggression task but were included in all other analyses. EEG data on 7 individuals were missing due to equipment failure or excessive artifacts in their signal, and 1 participant had alpha power density values greater than 3 *SD*s above the mean (i.e., values greater than 3.0; Zinser et al., 1999). These 8 participants were excluded from the EEG analyses but were included in all other analyses. The descriptive statistics reported above are based on the total sample of 135 volunteers.

Self-Report Measures

Measures of personality. Following informed consent procedures, participants completed personality questionnaires to examine the correlates of our measures of frontal alpha asymmetry and laboratory aggression. First, participants completed the Aggression Questionnaire (AQ; Buss & Warren, 2000) and the BIS/BAS Scales (Carver & White, 1994). The AQ is a 34-item measure of aggressive attitudes and behaviors that asks participants to rate the extent to which they feel or act in the manner described by the items from 1 (*Not at all*) to 5 (*Extremely*) and yields five subscales including Physical Aggression, Verbal Aggression, Indirect Aggression, Hostility, and Anger. The BIS/BAS Scales were used to assess the appropriateness of using the EEG hemispheric asymmetry score as a measure of behavioral approach activation. The BIS/BAS is a 20-item questionnaire with four subscales: BIS (7 items), BAS–Reward Responsiveness (5 items), BAS–Drive (4 items), and BAS–Fun Seeking (4 items). Each item is rated on a 4-point scale ranging from 1 (*Strongly Agree*) to 4 (*Strongly Disagree*). The BIS subscale assesses sensitivity to aversive stimuli, whereas the BAS–Reward Responsiveness, –Drive, and –Fun Seeking subscales measure sensitivity to anticipated/acquired rewards, motivation to achieve desired goals, and willingness to approach new appetitive stimuli, respectively.

Participants also completed a trait version of the 17-item Anxious Arousal subscale of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995) and the 12-item Rumination subscale from the Rumination–Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999). These symptoms have been associated with lateralized frontal activity (Heller, Nitschke, Etienne, & Miller, 1997; Henriques & Davidson, 1991), and thus the measures were administered to examine their associations with frontal asymmetry and to help interpret frontal asymmetry changes in response to stress conditions.

Mood changes across the experiment. Participants completed a state version of the 60-item Positive and Negative Affect Schedule–Expanded Form (PANAS-X; Watson & Clark, 1994) at three time points: the beginning of the experiment (baseline), following condition assignment (post–condition assignment), and following actual stress exposure (post–stress exposure). The PANAS-X data were used to examine changes in self-reported mood across the experiment, and the three time points were selected based on predictions about when relevant changes in mood would occur. Analyses were conducted on the Fear and Hostility subscales to assess how fluctuations in these mood states relate to frontal brain activity. The Hostility subscale indexed feelings of irritability, annoyance, and anger.

Cover Story and Baseline Assessments

Figure 1 illustrates the experimental procedures across stress groups. Upon arrival, participants completed informed consent forms, a demographic questionnaire, and a 14-item version of the Handedness Questionnaire (Raczkowski, Kalat, & Nebes, 1974) to confirm they were right-handed. The participant and a same-sex confederate drew pieces of paper out of a cup for a rigged role-assignment procedure involving a simulated work situation. The real participant was assigned to first complete an essay (which would or would not be evaluated by the other person) and then to be the supervisor in a subsequent task (where he/she would provide feedback to the confederate). Thus, participants anticipated that they would be able to provide feedback to their confederate (i.e., engage in action) later in the experiment, a procedure that helps ensure increases in approach motivation in response to mood inductions (Harmon-Jones et al., 2006). Following role assignments, the confederate and participant were led to separate rooms, where each purportedly received independent instructions for the remainder of the experiment.

Baseline measures. Participants completed trait questionnaires and the baseline PANAS-X mood rating, as described above. The participant was seated in a recliner positioned in front of a 21-in. monitor. After the physiological hookup, participants were instructed to sit and relax during an 8-min resting EEG recording. Each participant completed four blocks of eyes open (O) and eyes closed (C), with half of the participants in each stress condition assigned to one presentation order of eyes open/eyes closed (COOCOCCO) and the other half assigned to an alternate order (OCCOCOOC). The

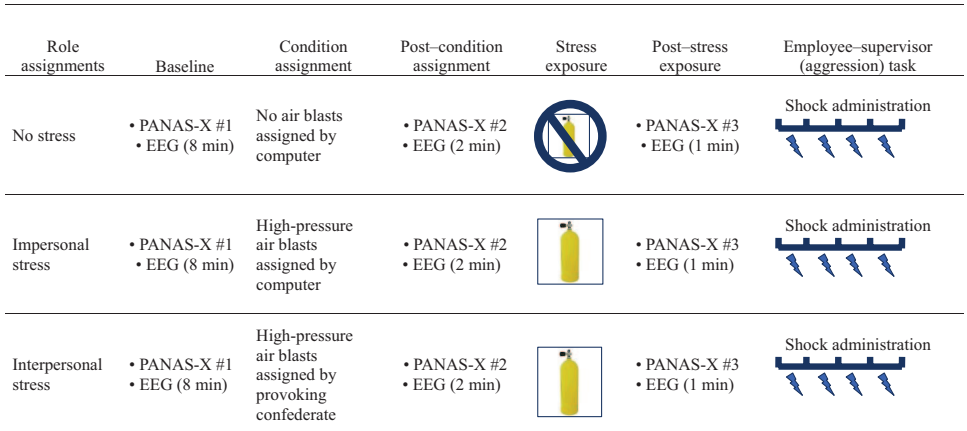

Figure 1. Study procedures and time points. PANAS-X = Positive and Negative Affect Schedule–Expanded Form (Watson & Clark, 1994); EEG = electroencephalogram. A color version of this figure is available on the web at http://dx.doi.org/10.1037/a0014376.supp

average alpha power recorded during the 8-min resting EEG recording served as the baseline measure of EEG activity.

Stress Manipulation (Condition Assignment and Stress Exposure)

Following the baseline EEG data collection, all participants completed an essay in which they described themselves and their strengths and weaknesses. They were told that the essay served the purpose of obtaining information about their personalities. The no-stress and impersonal (IMP) stress conditions were adapted from Verona and Curtin (2006), and the interpersonal (INT) stress manipulation was modified from Edguer and Janisse (1994). After completing the essay, participants were notified of their condition assignment. Participants in no-stress ($n = 49$; 26 men) were told that they were randomly assigned by a computer to receive no air blasts (low distraction) during the subsequent task. Those in the IMP stress condition ($n = 41$; 22 men) were told that they were randomly assigned by a computer to receive high-pressure air blasts directed at their throat (high distraction). Finally, participants in the INT stress condition ($n = 45$; 26 men) were told that their essays were going to be evaluated by the other participant they met (confederate). Depending on the confederate's evaluation of the essay, the confederate would determine whether the participant would receive low-, medium-, or high-pressure air blasts during a subsequent task. During condition assignment, participants in the INT stress condition were given an evaluation form by the confederate that indicated the participant should receive high-pressure air blasts due to a poorly written essay. Aside from manipulating who or what was presumably responsible for their assignment to high stress (confederate or computer), participants in the IMP and INT stress conditions were given the same description of the air blast stressor ("high pressure" and "strong"). A 2-min eyes-open EEG recording was conducted following condition assignment for all groups. Participants then completed a second PANAS-X (post–condition assignment) to rate their mood at that moment (see Figure 1).[1]

As illustrated in Figure 1, participants in the IMP and INT stress conditions were then exposed to the same stressor (2 min of intermittent and unpredictable high-pressure air blasts), although presumably their interpretation of that stressor was different. Participants in the no-stress condition were not exposed to air blasts at this time period. The air blasts were 50 ms in duration and were generated by a tank filled with compressed breathable air connected to a regulator that reduced the output to a constant flow pressure of 100 psi (7.03 kgf/cm^2). The air blasts were delivered via a tube that was directed at the throat at the level of the larynx (Grillon & Ameli, 1998). After stress exposure, the participants in all three groups completed a third (1-min) eyes-open EEG recording and a third PANAS-X questionnaire to assess their mood (post–stress exposure).

Aggression Procedure

Finally, participants were introduced to the employee–supervisor task (the aggression procedure). The aggression paradigm was adapted from Buss (1961) and was modified on the basis of more recent work in our laboratory. Some aggression researchers use alternative aggression paradigms, especially Taylor's (1967) competitive reaction time paradigm, because frustration and provocation are inherent in the aggression procedure (i.e., when the participant loses, he/she receives a shock from the other participant). The present experiment utilized a more classic paradigm, because it allows the researchers to induce negative affect (interpersonal and impersonal) unrelated to performance in the aggression procedure.

Participants were told that the employee (confederate) would perform a digit span task involving digit memorization and recall the series of digits by typing them on a keyboard. Following each number trial, the participant was to view the employee's response on his/her monitor and provide feedback, as quickly as possible, regarding the correctness of the answer. As supervisor, the participant was instructed to press a *correct* button if the response was correct or 1 of 10 *shock* buttons (representing increasing intensities of shock) if the employee's response was incorrect to simulate criticism of job performance, as in a work situation. They were free to choose any of the shock intensities for incorrect responses across the trials. The confederate's performance did not affect the participants' compensation in the study. In actuality, no shocks were administered to the confederate during the experiment, but participants were led to believe that they were administering real shocks.

A shock demonstration was conducted prior to beginning the aggression task to enhance the credibility of the cover story. To control for individual differences in shock sensitivity, which might affect willingness to deliver increasing levels of shock during the task, we administered three electric shocks of increasing intensity to each participant and rated each for perceived aversiveness on a scale of 1 (*not at all painful*) to 100 (*extremely painful*). The experimenter's description of shock levels to be delivered to the employee was calibrated to each participant's ratings of these pretest shocks.

The actual experiment consisted of a total of four task blocks, with 10 trials per block. Between 40% and 60% of the trials across blocks involved an incorrect response from the employee (confederate), calling for a shock button response from the supervisor (actual participant). To maintain the efficacy of the manipulations, we administered intermittent air blasts to the participants in the IMP and INT conditions during the actual aggression procedure (four air blast administrations per block).

EEG Recordings and Alpha-Asymmetry Measure

To record EEG, we placed a lycra stretchable cap (Electrocap International, Eaton, OH) with silver/silver chloride electrodes on each participant's head. The scalp was mildly abraded, and electrode paste was applied to each electrode site. The International 10–20 System (Jasper, 1958) was used for electrode placement, and electrode impedance for all channels was below 5 KΩ. To record eye movements for the purpose of offline blink correction, we placed two miniature 5 mm silver/silver chloride electrodes

[1] Although participants were randomly assigned to stress conditions, we examined differences in trait measures of aggression, anxiety, and rumination between the three conditions with a univariate analysis of variance and total scores on the AQ, MASQ Anxious Arousal, and RRQ scales, respectively. No significant differences between the stress groups emerged ($ps > .34$).

above and below the pupil of the right eye to serve as a bipolar channel to measure vertical electrooculogram (EOG). During eyes-open recording segments, participants were instructed to focus on a fixation cross located in the center of the monitor to avoid eye movement artifact. During recording, the left mastoid served as the reference electrode for all other sites, specifically midline (Fz, Cz, Pz), lateral frontal (F8/F7), midfrontal (F4/F3), central (C4/C3), anterior temporal (T4/T3), posterior temporal (T6/T5), and midparietal (P4/P3). EEG and EOG were amplified using Neuroscan Synamps2 (Neuroscan Compumedics, Charlotte, NC) and band pass filtered from 0.1 to 100 Hz. Analog signals were digitized online at 2,000 Hz using a 24-bit analog-to-digital converter.

After data collection, the EEG was manually checked for movement artifact and submitted to a regression-based blink-correction procedure in Neuroscan Edit software version 4.3. Data containing artifacts were rejected. Less than 1% (0% to 52% across participants) of epochs were rejected due to artifact. Due to a problem with the left mastoid connection for some participants, EEG for all participants was re-referenced off-line using an average head reference derivation (based on all of the electrode sites), rather than an average mastoid reference, to avoid losing participant data. Thus, each channel of EEG reflected the voltage between the average of all of the available scalp sites on the head and an active scalp site. We also re-referenced a second time to Cz, a site used in past research on EEG hemispheric alpha activity (Harmon-Jones & Allen, 1998) to examine the reliability of findings across reference schemes.

Artifact-free epochs of 2,047.5 ms (4,096 samples) were extracted, with continuous epochs overlapping by 50%. The power spectra were derived using the fast Fourier transform method with a Hamming window for each epoch. Total power for the alpha band (8–13 Hz) was calculated for the baseline measurement by averaging the 8-min eyes-open and eyes-closed segments, whereas the post–condition assignment and post–stress exposure measurements consisted of the average alpha power collected from those two respective eyes-open segments (2 min and 1 min, respectively). Alpha power was log-transformed to normalize the data.

On the basis of previous research that indicates that alpha power is inversely related to brain activity (Cook, O'Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998), the term *activity* in this article refers to brain activity, or to decreased alpha. On the basis of previous work, EEG hemispheric asymmetry scores were calculated by subtracting alpha power in the left hemisphere from alpha power in the right hemisphere, for example, ln(F8) – ln(F7). Thus, higher frontal asymmetry scores indicated less alpha activity in the left hemisphere and more relative left frontal brain activity. Primary analyses were conducted on lateral frontal (F8/F7) sites to directly compare with previous work that has examined this frontal site (e.g., Harmon-Jones, 2007; Harmon-Jones et al., 2006).

Laboratory Aggression Measure

As in previous research, aggression was operationalized as the mean level of shock that participants administered to the confederate during each of the four task blocks and across the whole experiment. We also calculated the percentage of shock responses that involved extreme aggressive responding (shock levels greater than or equal to 5) to analyze the context of extreme levels of aggression. Increases in aggression across the employee–supervisor task blocks were of interest because this effect has been emphasized in experimental research on aggression (Goldstein, Davis, & Herman, 1975; Verona & Curtin, 2006; Verona & Kilmer, 2007).

Debriefing and Manipulation Check

Following the experiments, participants completed a poststudy questionnaire and were interviewed. On the poststudy questionnaire, participants used a 10-point rating scale to indicate the extent to which they were motivated to increase shock intensities due to instrumental motives ("to encourage better performance in the employee"; Verona et al., 2002) and due to hostile motives ("because upset at the employee"). Items on the poststudy questionnaire were used partly to examine the efficacy of the aggression paradigm. During poststudy interviews and debriefings, 10 participants (7 men; 5 no-stress, 1 IMP stress, and 4 INT stress) expressed suspicions about the cover story and were replaced by other participants (i.e., not included in the sample size presented above). They were not convinced either that the confederate was a real participant or that they were actually shocking another participant. These participants did not differ on demographic variables from the participants who were kept in the sample. All participants were fully debriefed. They were given an explanation of the true intentions and hypotheses of the experiment, were provided justification for using deception in aggression studies, and were encouraged to voice any concerns to the experimenters. In all, the experiment took approximately 2.5 hr.

Results

Construct Validity of the Aggression Paradigm and EEG Asymmetry Measure

Aggression paradigm. We analyzed the construct validity of our measure of laboratory aggression, specifically mean shock intensity during the employee–supervisor task, by conducting correlations with relevant indexes of aggressive traits and attitudes. As expected, the mean shock intensity administered by participants was related significantly to AQ total score ($r = .22$, $p < .01$) and the AQ Physical Aggression and Hostility subscales (rs $= .32$ and .19, ps $< .01$ and .05, respectively). This indicates that individuals who administered more intense shocks reported higher overall levels of trait aggression, particularly physical aggression, than did those who administered less intense shocks, demonstrating that the laboratory aggression measure used in this study correlates as expected with relevant criterion variables. No gender differences emerged for these correlations.

Overall, participants reported higher levels of poststudy questionnaire ratings of instrumental (i.e., using shock to improve the confederate's performance) than hostile (i.e., using shock in response to feeling upset at the confederate's performance) motives for shock intensities (instrumental: $M = 5.2$, $SD = 2.6$; hostile: $M = 3.5$, $SD = 2.8$), $t(127) = 7.0$, $p < .001$. However, ratings of instrumental and hostile motives were significantly correlated with each other ($r = .45$, $p < .001$), confirming that aggression is simultaneously motivated by different reasons (Bushman & Anderson, 2001). Indeed, mean shock intensity showed similar positive relationships with participants' ratings of instrumental and

hostile motives for aggression (rs = .41 and .47, respectively, $p <$ .001). To confirm that hostile intentions were predominantly responsible for participants' aggressive behavior in the experiment, we conducted a hierarchical regression on mean shock intensity, with the instrumental motives item entered as a predictor in the first step and the hostile motives item entered in the second step. As expected from the zero-order correlations, both instrumental and hostile motives predicted shock intensity scores in the second step, $F(2, 114) = 20.3$, $p < .001$, $R^2 = .25$, but the unique contribution of hostile motives was larger (βs = .25 and .35, ps < .01). Additionally, hostile motives uniquely accounted for 10% of the variance in shock intensity scores above instrumental motives, $\Delta F(1, 112) = 14.5$, $p < .001$. When these analyses were repeated with hostile motives entered first and instrumental motives entered second, the instrumental motives item accounted for only an additional 5% of the variance in aggression above hostile motives, $\Delta F(1, 112) = 7.6$, $p < .01$. These results confirm that shock intensity selections during the aggression procedure were indeed influenced by hostile intentions and being upset at the employee.

Frontal alpha asymmetry. Motivation-based models of frontal alpha asymmetry posit that greater left than right activity reflects the activation of approach motivational tendencies. To establish the construct validity of frontal alpha asymmetry in our data set, we regressed asymmetry scores simultaneously on the BIS and BAS scales to examine the unique relationships among each of these scales and frontal asymmetry. Separate regressions were conducted on the entire sample at the three time periods that frontal asymmetry was measured (baseline, post–condition assignment, and post–stress exposure). A positive relationship was observed between BAS reward and frontal asymmetry at baseline ($\beta = .24$, $p = .02$) and post–condition assignment ($\beta = .20$, $p =$ .06), although the latter correlation was a nonsignificant trend. These results suggest that tendencies toward behavioral approach were generally associated with increased left frontal asymmetry. In contrast, BIS was not correlated significantly with brain activity at any time measurement (ps > .27), which is consistent with previous research on behavioral activation and frontal asymmetry (Coan & Allen, 2003).

Other research has suggested that fear and verbal rehearsal influence frontal alpha asymmetry (Heller et al., 1997; Hofmann et al., 2005). To test these relationships, we used MASQ anxious arousal and RRQ rumination as simultaneous predictors in a regression conducted on frontal asymmetry scores in the entire sample. No significant effect for MASQ anxious arousal (ps > .41) or RRQ rumination (ps > .58) were observed for frontal asymmetry scores at any time period. These analyses indicate that changes in EEG frontal asymmetry in our particular paradigm cannot be accounted for by symptoms of anxiety or verbal rehearsal, although this may be the case in other contexts (Heller et al., 1997; Hofmann et al., 2005).

Goal 1: Effects of Stress

Analytic strategy. Consistent with a priori hypotheses, planned orthogonal comparisons (POCs) of stress condition effects were utilized in analyses. Specifically, stress condition was decomposed into orthogonal stress versus no-stress (IMP/INT vs. no-stress) and IMP versus INT contrasts to examine whether (a) stress in general produces changes in brain activity, mood, and aggression (Hypothesis 1: approach-motivation hypothesis) and (b) the two types of stress produce differential effects on these dependent measures (Hypothesis 2: stress-specific hypothesis), respectively. Thus, frontal alpha asymmetry scores (F7/F8) and PANAS mood ratings (for fear and hostility) were analyzed separately within mixed-model multivariate analysis of variance (MANOVA), with time (baseline, post–condition assignment, post–stress exposure) as a within-subject variable and stress condition POCs (stress vs. no-stress, IMP vs. INT) as between-subjects variables. Analysis of shock intensity also involved a mixed-model MANOVA with task block (1–4) as the within-subject variable and stress condition POCs as the between-subjects variables. Gender effects or interactions were not observed in the analyses of frontal alpha asymmetry or shock intensity, except for the ubiquitous gender main effect for shock intensity (i.e., men responded with more intense aggression than did women). For this reason, gender was not included as a factor in the analyses reported. We report, in addition to p values, effect size with partial eta squared (i.e., equivalent to ΔR^2 from multiple regression models).

Asymmetric frontal brain activity. Means and standard deviations for lateral frontal (F7/F8) sites EEG alpha power is provided by stress condition, hemisphere, and time in Table 1. As mentioned above, frontal alpha asymmetry scores were calculated as the difference between left (F7) and right (F8) hemispheres such that higher scores reflected greater left frontal activity. As predicted, the Stress vs. No Stress × Time interaction was significant, $F(2, 123) = 5.98$, $p < .01$, $\eta_p^2 = .09$ (see Figure 2). To decompose this interaction, follow-up analyses of the time effect were conducted separately in the combined stress (IMP and INT) versus no-stress conditions. As expected, no significant effect of time was observed in the no-stress condition ($p = .43$), but the effect of time was significant in the combined stress conditions, $F(2, 81) = 9.63$, $p <$ .001, $\eta_p^2 = .19$. As illustrated in Figure 2, shifts to greater left frontal asymmetry were observed in the two stress groups from baseline to both post–condition assignment, $F(1, 82) = 17.50$, $p <$ .001, $\eta_p^2 = .18$, and post–stress exposure, $F(1, 82) = 7.86$, $p < .01$, $\eta_p^2 = .08$, but no differences in frontal asymmetry were observed between the two latter time points ($p = .44$). In contrast, the IMP vs. INT × Time interaction was not significant, $F(2, 123) = .20$, $p = .82$, $\eta_p^2 = .03$. This indicates that shifts to left frontal asymmetry following the manipulations were comparable in the IMP and INT stress conditions.[2]

To confirm that the above-reported time effect in the stress conditions was produced primarily by changes in left hemisphere frontal activation, we conducted separate analyses of the time effect for left and right frontal alpha power for participants in the combined stress conditions. As expected, the time effect was significant for left frontal alpha power, $F(2, 81) = 3.73$, $p < .03$, $\eta_p^2 = .08$, but not for right frontal alpha power ($p = .65$).

[2] Repeated measures analyses were also conducted with the trichotomous stress group variable (no-stress, IMP, INT) instead of the POCs as the between-subjects variable in analyses conducted on EEG–alpha asymmetry scores. We found, consistent with the analyses using the POCs, a Stress Group × Time interaction, $F(4, 248) = 2.96$, $p < .02$. The time effect was significant for the IMP stress group, $F(2, 38) = 5.8$, $p < .01$, and the INT stress group, $F(2, 41) = 3.9$, $p < .05$, but not the no-stress group, $F(2, 42) = 0.9$, $p =$ .43. Given the similarity in results, we chose to report the POC analyses in the text, as they directly map onto our a priori hypotheses.

Table 1
Alpha Power for Lateral Frontal Sites (F8/F7), PANAS-X Mood Scores, and Mean Shock Intensity by Stress Condition

	No stress			Impersonal stress			Interpersonal stress		
Variable	Baseline *M* (*SD*)	Post–condition assignment *M* (*SD*)	Post–stress exposure *M* (*SD*)	Baseline *M* (*SD*)	Post–condition assignment *M* (*SD*)	Post–stress exposure *M* (*SD*)	Baseline *M* (*SD*)	Post–condition assignment *M* (*SD*)	Post–stress exposure *M* (*SD*)
Right alpha power (F8)	−0.70 (0.70)	−0.71 (0.71)	−0.86 (0.73)	−0.51 (0.90)	−0.54 (0.92)	−0.54 (0.91)	−0.75 (0.77)	−0.78 (0.70)	−0.84 (0.78)
Left alpha power (F7)	−0.72 (0.74)	−0.65 (0.88)	−0.86 (0.81)	−0.48 (0.90)	−0.61 (0.97)	−0.60 (0.92)	−0.75 (0.76)	−0.87 (0.73)	−0.90 (0.77)
PANAS-X Fear	9.7 (3.7)	8.5 (2.9)	8.4 (2.7)	9.3 (4.3)	8.6 (3.8)	10.0 (5.1)	8.0 (2.5)	8.0 (2.9)	8.1 (3.9)
PANAS-X Hostility	7.5 (2.6)	7.7 (2.9)	8.1 (4.1)	8.1 (3.6)	7.9 (3.2)	8.8 (4.7)	7.1 (1.8)	8.4 (2.8)	9.2 (3.9)
	M (*SD*)	Min/max	% shocks ≥5	*M* (*SD*)	Min/max	% shocks ≥5	*M* (*SD*)	Min/max	% shocks ≥5
Shock intensity	2.9 (1.6)	1.0/6.0	34.6	3.4 (2.2)	1.0/8.4	37.3	4.0 (1.6)	1.7/7.4	41.6

Note. No stress: $n = 44$; impersonal stress: $n = 40$; interpersonal stress: $n = 43$. Min/max = the minimum and maximum of the mean shock intensities administered by participants in each group. All groups included participants who used the lowest and highest shock levels possible (from 1 to 10). PANAS-X = Positive and Negative Affect Schedule–Expanded Form (Watson & Clark, 1994); Min = minimum; max = maximum.

Follow-up tests indicated, consistent with the previous analyses, greater shifts in left frontal activation in the two stress conditions from baseline to both post–condition assignment, $F(1, 82) = 6.58$, $p < .02$, $\eta_p^2 = .07$, and post–stress exposure, $F(1, 82) = 4.88$, $p < .03$, $\eta_p^2 = .06$, but the two latter time points did not differ from each other ($p = .89$).[3]

To examine the reliability of brain activity findings across different EEG reference schemes, we reconducted analyses after re-referencing the EEG data to the Cz electrode. As expected, the effect of time (baseline, post–condition assignment, post–stress exposure) was significant for the combined stress groups, $F(2, 81) = 3.14$, $p = .048$, $\eta_p^2 = .07$, but not for the no-stress group ($p > .64$). As with the average head reference, an effect of time emerged specifically for left hemisphere activity in the two stress groups, $F(2, 82) = 5.51$, $p = .006$, $\eta_p^2 = .12$, with a significant increase in left frontal activity from baseline to post–condition assignment, $F(1, 82) = 10.3$, $p = .002$, $\eta_p^2 = .11$, and baseline to post–stress exposure, $F(1, 82) = 4.64$, $p = .034$, $\eta_p^2 = .05$. No time effect was found for the right hemisphere in either of the two stress groups or the no-stress group ($ps > .15$). Since inconsistent findings across reference schemes are a common problem in the EEG alpha asymmetry literature, the similar pattern of findings that emerged across reference schemes supports the reliability of the present findings.[4]

In summary, analyses of lateral frontal sites confirmed that the two stress conditions elicited significant shifts in frontal brain activity, specifically involving increases in left hemisphere activity, often interpreted as approach motivation. This is the first study to show that changes in EEG frontal asymmetry are similar across IMP and INT stress exposure.

Aggressive responding. Next, we examined whether the stress manipulations affected subsequent aggressive responding in a manner concordant with the lateralized frontal activity. Means, standard deviation, and range of shock intensity responses, as well as the percentage of extreme aggressive responses (≥Level 5 shock) administered by participants during the subsequent employee–supervisor task are provided by stress condition in Table 1. Shock intensity was analyzed within a mixed model ANOVA with stress condition POCs and task block (1–4) as factors in analyses.

Analysis of mean shock intensity revealed a main effect of block, $F(3, 116) = 6.47$, $p < .001$, $\eta_p^2 = .14$, and a stress versus no-stress contrast effect, $F(1, 118) = 3.90$, $p = .05$, $\eta_p^2 = .03$. However, these main effects were modified by a Stress vs. No Stress × Block interaction, $F(3, 116) = 3.12$, $p < .05$, $\eta_p^2 = .08$ (see Figure 3). Follow-up analyses conducted separately in the stress (IMP and INT) versus no-stress conditions revealed no significant effect of block in the no-stress condition ($p = .28$), but a significant effect of block in the combined stress conditions emerged as expected, $F(3, 75) = 9.18$, $p < .001$, $\eta_p^2 = .27$. Specifically, simple contrasts revealed that participants in the two stress groups increased shock intensity from Block 1 to Blocks 2, 3, and 4, $Fs(1, 77) = 17.92$, 7.14, and 20.88, respectively, $ps < .01$. Thus, only participants in the two stress conditions administered increasing shock intensities from the first to subsequent task

[3] To examine whether the pattern of effects we obtained were specific to frontal regions, we reconducted the repeated measures MANOVA analysis with time as the within-subjects variable and stress condition POCs (stress vs. no stress, IMP vs. INT) as the between-subjects variable on posterior sites (P4/P3), which have been the focus of research in asymmetry studies on arousal (Heller et al., 1997). Evidence that the effect of stress condition on changes in alpha asymmetry was specific to frontal regions emerged. Specifically, the analysis did not produce a significant interaction involving either stress condition POC ($p > .20$), indicating that stress condition did not differentially influence changes in posterior asymmetry.

[4] When analyses of frontal asymmetry were conducted with midfrontal (F3/ F4) sites, simple effect tests revealed a nonsignificant leftward shift in frontal activity for the two stress groups from baseline to post–stress exposure, $F(1, 82) = 3.16$, $p < .08$, but not post–condition assignment ($p = .77$). This suggests that results were broadly consistent but not robust across the lateral frontal and midfrontal sites.

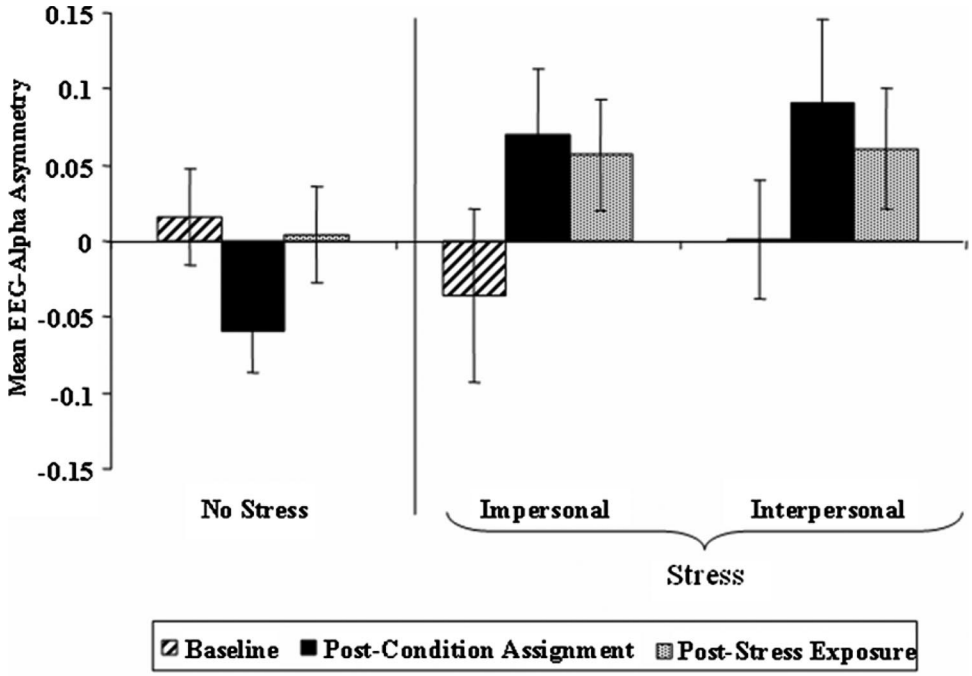

Figure 2. Electroencephalogram (EEG) lateral frontal asymmetry at baseline, post–condition assignment, and post–stress exposure by stress condition. $n = 44$, 40, and 43 for the no-stress, impersonal stress, and interpersonal stress conditions, respectively. Error bars represent ±1 standard error.

blocks, whereas no-stress participants did not alter their choice of shock intensity over blocks. The IMP vs. INT × Time interaction was not significant, $F(3, 116) = 1.28$, $p = .29$, $\eta_p^2 = .03$, indicating that the block effects were comparable in the IMP and INT stress conditions. Figure 3 suggests slightly higher intensity shocks administered by the INT than the IMP stress group (see Table 1), but the mean shock intensity did not differ significantly between the two stress groups (IMP vs. INT POC was not significant $p = .17$).

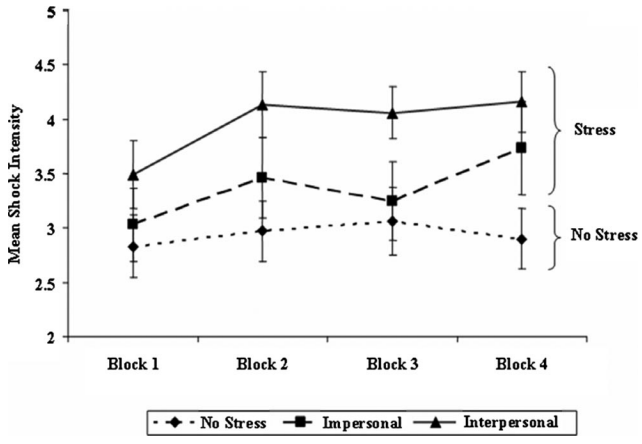

Figure 3. Mean shock intensities delivered across block by participants in each stress condition. $n = 41$, 36, and 39 for the no-stress, impersonal stress, and interpersonal stress conditions, respectively. Error bars represent ±1 standard error.

When analyses were conducted with a measure of extreme aggression (percentage of shock levels ≥5), the results were similar although slightly stronger than those with mean shock intensity. Specifically, the analysis revealed main effects of block, $F(3, 116) = 10.25$, $p < .001$, $\eta_p^2 = .21$, and a Stress vs. No Stress × Block interaction, $F(3, 116) = 3.96$, $p = .010$, $\eta_p^2 = .09$. Simple effects showed that, relative to the first block, participants in the stress conditions continuously increased their use of extreme aggression across Blocks 2, 3, and 4, Fs(1, 77) = 20.7, 21.6, and 11.2, respectively, ps < .001, while participants in the no-stress condition did not (ps > .06). The average percentage of extreme aggression used in the no-stress and stress conditions across the four blocks was 8.9, 9.6, 12.3, 6.5 and 10.0, 17.3, 18.4, 14.7, respectively.

In summary, participants exposed to one of two types of stress demonstrated increases in aggression across blocks and delivered more extreme shock responses to the confederate relative to those not exposed to stress. Thus, participants in the two stress conditions who showed stress-related shifts in left frontal brain activity earlier in the experiment also displayed subsequent increases in aggression across time during the employee–supervisor task.

Changes in mood across time. Means and standard deviations for self-reported fear and hostility are displayed by stress condition and time in Table 1. Self-reported fear and hostility were each analyzed separately in mixed model ANOVAs with stress condition POCs as a between-subjects variable and time as a within-subject variable. The analysis examining fear and hostility was important given the impersonal and interpersonal nature of the two stress conditions. For fear, a significant main effect of time was observed, $F(2,$

130) = 7.77, $p < .001$, $\eta_p^2 = .11$, which was moderated within both Stress vs. No Stress × Time, $F(2, 130) = 5.66$, $p < .01$, $\eta_p^2 = .08$, and INT vs. IMP × Time interactions, $F(2, 130) = 5.83$, $p < .01$, $\eta_p^2 = .08$, indicating that the pattern of fear response across time was different in all three stress conditions (see Figure 4). To clarify these interactions, we conducted time analyses separately in all three conditions. In the no-stress condition, analyses revealed a significant time effect, $F(2, 47) = 7.08$, $p < .01$, $\eta_p^2 = .23$, with follow-up analyses revealing a decrease in fear from baseline to both post–condition assignment, $F(1, 48) = 10.27$, $p < .01$, $\eta_p^2 = .18$, and post–stress exposure, $F(1, 48) = 14.21$, $p < .001$, $\eta_p^2 = .23$. In the IMP stress condition, the time effect was significant, $F(2, 38) = 10.47$, $p < .001$,

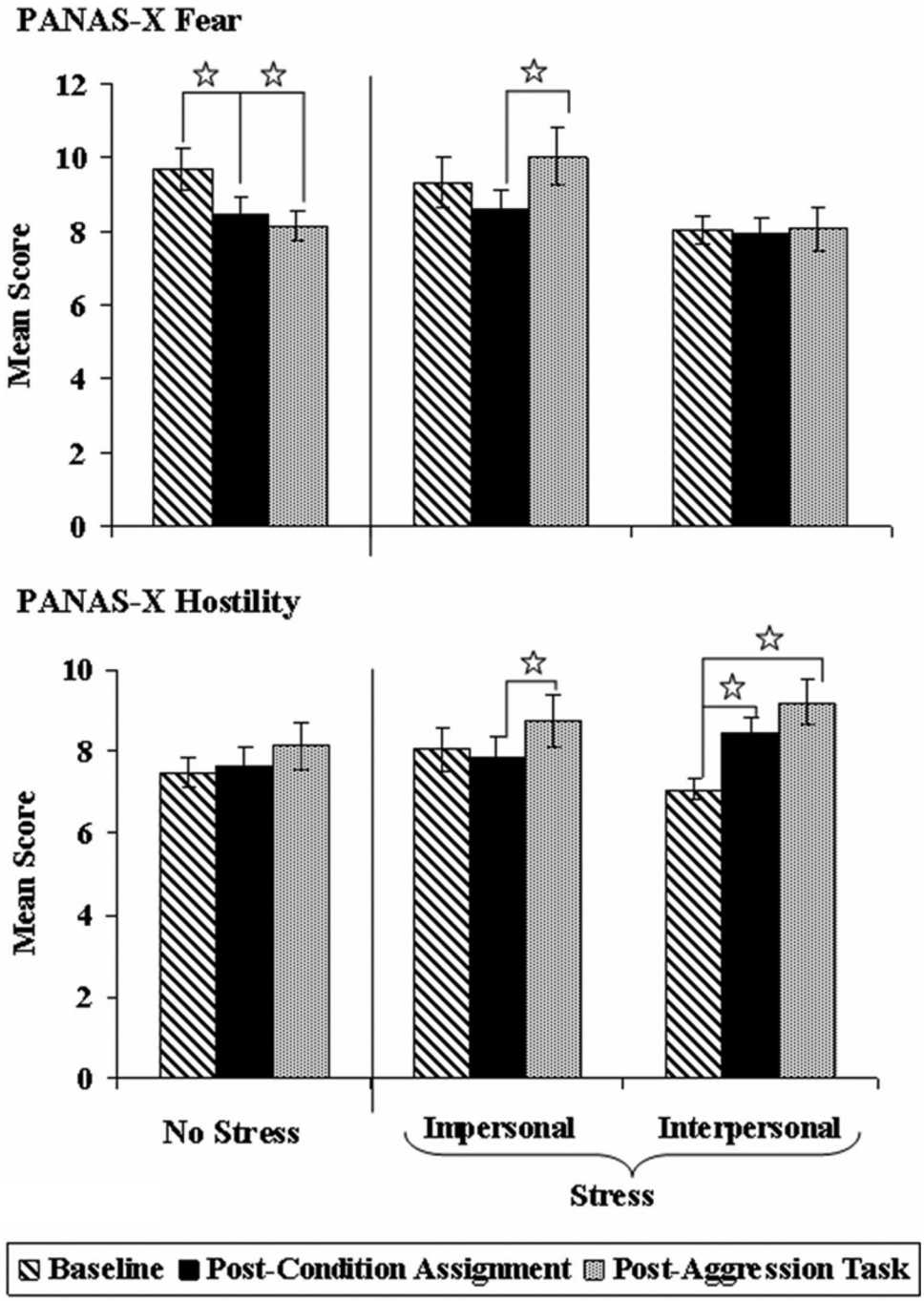

Figure 4. Self-reported changes in PANAS-X fear and hostility across time by stress condition. All PANAS-X mood scores represent change from baseline. $n = 44$, 40, and 43 for the no-stress, impersonal stress, and interpersonal stress conditions, respectively. PANAS-X = Positive and Negative Affect Schedule–Expanded Form (Watson & Clark, 1994). Error bars represent ±1 standard error. Stars indicate $p < .05$.

$\eta_p^2 = .36$, and follow-up tests indicated that fear increased selectively after stress exposure relative to self-reported fear after condition assignment, $F(1, 39) = 13.02$, $p < .001$, $\eta_p^2 = .25$. No effect of time on fear was found in the INT stress condition, $F(2, 43) = .14$, $p = .87$, $\eta_p^2 = .01$.

For hostility, a main effect of time was observed, $F(2, 130) = 6.04$, $p < .01$, $\eta_p^2 = .09$, which was moderated within an IMP vs. INT × Time interaction, $F(2, 130) = 3.41$, $p < .05$, $\eta_p^2 = .05$ (see Figure 4). To decompose this interaction, we conducted separate time analyses within the INT and IMP stress conditions. Analyses for the INT stress condition revealed a time effect, $F(2, 43) = 7.52$, $p < .01$, $\eta_p^2 = .26$. Follow-up analyses revealed that hostility in the INT stress condition increased significantly from baseline to both post–condition assignment, $F(1, 44) = 10.10$, $p < .01$, $\eta_p^2 = .19$, and post–stress exposure, $F(1, 44) = 12.82$, $p < .001$, $\eta_p^2 = .23$. Reports of hostility at the two latter time points did not differ ($p = .15$), suggesting that heightened hostility was maintained across these two periods in the INT stress group. The effect of time was also significant in the IMP stress condition, $F(2, 38) = 3.79$, $p < .05$, $\eta_p^2 = .17$. Follow-up tests revealed that the IMP stress group selectively showed an increase in hostility only after stress exposure relative to participants' self-reported hostility after condition assignment, $F(1, 39) = 7.10$, $p < .01$, $\eta_p^2 = .15$.

Thus, consistent with the pattern of stress-related left frontal activity, both stress groups reported increases in hostility, although the effect was consistent across time only for the INT stress group. For the IMP stress group, self-reported hostility was not evident until participants were directly exposed to air blasts (not after condition assignment when they were threatened with the future presentation of air blasts), at which point they also reported significant increases in fear (see above). Thus, the subjective experience of the stressor in the IMP group involved a more complex set of negative emotions than in the INT group.

Goal 2: Stress-Induced Frontal Asymmetry Predicts Aggressive Behavior and Relates to Hostile Personality

Our final goal was to examine whether approach-related brain activity predicts subsequent aggressive behavior and is associated with hostile or aggressive tendencies, particularly for participants exposed to stress. First, we conducted regression analyses on mean shock intensity, with the frontal asymmetry index (higher scores signifying more left than right frontal activity) at each time point serving as a predictor in separate analyses. The stress versus no-stress and IMP versus INT POCs were also included as predictors, along with their interactions with mean centered frontal asymmetry at each time point. Analyses with frontal asymmetry at post–stress exposure as a predictor revealed a significant Stress vs. No Stress × EEG Frontal Asymmetry interaction, $\beta = .20$, $t(108) = 2.11$, $p < .05$, indicating that the relationship between post–stress exposure frontal asymmetry and aggression was different for the combined stress groups relative to the no-stress group. Follow-up correlations showed that, whereas more left frontal asymmetry at post–stress exposure predicted less aggression in the no-stress condition ($r = –.24$), it predicted more intense aggression for the combined stress groups ($r = .20$). Thus, shifts to left frontal asymmetry promote aggression only when individuals are exposed to stress. EEG frontal asymmetry at post–stress exposure did not interact with the IMP versus INT ($p = .85$), indicating that the relationships between frontal asymmetry and aggression were similar across the two stress groups. Analyses that included frontal asymmetry at baseline and post–condition assignment as predictors did not yield significant effects or interactions involving frontal asymmetry.

Second, we conducted partial correlation analyses (covarying out baseline EEG asymmetry scores) to examine whether left frontal activity in response to stress was associated with aggressive and hostile personality traits, as measured by the AQ subscales. These analyses revealed that left frontal activity following stress exposure was positively correlated with the combined AQ Anger and Hostility subscales only among participants in the INT stress condition ($r = .31$, $p < .05$) but not the IMP or no-stress groups (rs = –.05 and .08, respectively). No effects were found for the other AQ subscales or for frontal asymmetry following condition assignment. Thus, those who showed the most left-frontal activity in response to stress administered the most intense aggression to the confederate and, among those exposed to interpersonal stress in particular, reported higher levels of anger and hostility in their everyday lives.

Discussion

Stress and Approach-Related Behaviors

According to Berkowitz (1990), a variety of stressful contexts can prime an individual to engage in aggressive behavior. Given that most forms of aggression and acting out require the aggressor to approach the target in order to inflict harm (e.g., physical aggression), aggressive acts are hypothesized to be influenced by the activation of behavioral approach systems. In support of this contention, the literature on motivational states indicates that anger is associated with more relative left than right frontal activity, which is consistent with research linking anterior leftward asymmetry with behavioral approach (Sutton & Davidson, 1997). An important contribution of the present study is the link it provides between the extant literatures on stress-induced aggressive behavior and research on approach-related brain activity. Specifically, results were consistent with the approach-motivation hypothesis of stress-induced aggression in that exposure to either an impersonal or interpersonal stressor initiated approach-related brain activity and aggressive behavior. In particular, individuals in the two stress conditions evidenced shifts to more left than right hemisphere activity following condition assignment and stress exposure, subsequent increases in aggression intensity, and a higher proportion of extreme aggressive responses. Even more illuminating, regression analyses indicated that individuals who showed more left than right frontal activity in response to stress exposure were also the ones who administered the most intense shock responses to the employee and reported the most anger/hostility in their everyday lives. These data suggest that the type of stressor plays less of a role in determining lateralized frontal brain activity and its relationship to dysregulated behaviors than posited by the stress-specific hypothesis, given that both forms of stress elicited comparable changes in brain activity. As well, shifts to left frontal activity predicted heightened aggression in both stress groups.

The present findings help clarify the links between affective states and particular motivational tendencies by providing evidence that negative affective contexts can induce brain activity

consistent with behavioral approach models. Carver (2004) reported that tendencies toward a hyperactive behavioral activation system, which is typically implicated in appetitive and pleasant activities, was associated with increases in self-reported negative affective states in goal-impeding situations. The results of the present study complement those of Carver (2004), since we found evidence that negative affective states activate behavioral approach systems in contexts where future action is anticipated and there is an emotional induction. For instance, we found that frontal asymmetry predicted less aggression under a neutral condition (no stress), which is consistent with Davidson's (2004) conceptualization of resting levels of left lateralized frontal activity being associated with better adjustment. However, shifts to more left lateralized frontal activity in response to stress predicted increases in aggression, which signifies that approach activation in stressful contexts serves to increase risk for aggression.

These findings have implications for continued research on contextual and brain factors that promote dysregulated behaviors, including aggression, risk taking, and substance use. Considerable evidence suggests that some individuals engage in risky or impulsive activities or use substances as a way of coping with negative affective states (Lightsey & Hulsey, 2002; Richman & Flaherty, 1990; Tice, Bratslavsky, & Baumeister, 2001). Nigg (2006) has implicated excessive approach tendencies as a temperamental route to youth conduct disorder and *frustrative aggression* (involving both approach and stress systems). In light of the present study, it seems that stress reactivity involving activation of approach-related frontal processes may represent a mechanism for the emergence of a spectrum of dysregulated behaviors. Animal research suggests that stress-related brain systems (HPA axis, extended amygdala) influence dopaminergic pathways implicated in approach and reward, which in turn innervate the anterior cingulate that governs response monitoring and control (Holroyd & Coles, 2002). Indeed, researchers have theorized that adaptations in these connections over time are implicated in the development of dysregulated syndromes including substance use disorders (Koob & Le Moal, 2001). Animal researchers also have found that stimulating attack centers in the rat brain (hypothalamus) simultaneously activates stress-related glucocorticoid activity and vice versa, indicating that stress/emotion circuits and behavioral approach centers are mutually influential (Kruk et al., 2004). This work can be extended to the human literature to investigate alterations in brain activity and behavior under stress among individuals with histories of mood-related risk taking or syndromes marked by anger and aggression, including borderline personality disorder or antisocial/externalizing tendencies.

Impersonal Versus Interpersonal Stress

Although the two types of stress elicited brain activity associated with approach motivation, the mood data indicated that participants in the two stress groups reported different subjective experiences. That is, despite the fact that the two stress groups did not differ significantly from each other on aggression, it was only interpersonal stress that elicited consistent increases in hostility across the experimental manipulations (cf. Ellsworth & Smith, 1988). In addition, shifts to left frontal asymmetry in response to interpersonal but not impersonal stress were related to hostile personality traits. Impersonal stress prompted increases in hostility but, in contrast to interpersonal stress, it also elicited fear following stress exposure, which suggests that the IMP stress group experienced simultaneous activation of behavioral approach and behavioral withdrawal systems (anger and fear). This dual activation is perhaps reflected in the inconsistent increases in hostile mood and intermediate levels of aggression in the IMP stress group (see Figure 3). Given participants' reports of fear and hostility, we would have expected that impersonal stress would lead to increased bihemispheric frontal activity, per Davidson's (1983) model of frontal brain asymmetry. This was not the case in our study. The results are consistent nonetheless with Wacker et al. (2003), who reported that contexts eliciting both fear and anger resulted in more left frontal asymmetry. In their study and in the present study, the experience of anger seems to have overwhelmed that of fear in influencing changes in EEG frontal brain activity.

Nonetheless, the present analysis implies that the distinct stressful contexts resulted in few differential effects on emotional and motivational responses in the present study. Brain activity and aggression were ultimately similar across stressful contexts. Unfortunately, the present study was not designed to uncover the cognitive or attributional processes that influenced aggressive behavior in the different stress groups (cf. Anderson et al., 1996). In future work, participants' cognitions should be measured to help elucidate the combined influences of approach motivation, emotional response, and subsequent cognitive processing in determining the risk for aggression under different stress conditions.

Strengths, Limitations, and Implications

The present study has a number of strengths, including the integration of a unique set of stress inductions and multiple methodologies (electrophysiological, overt behavior, self-report mood) into a unified experimental paradigm. Additionally, measures of brain activity and state mood were collected over a series of time points, allowing us to examine shifts in frontal brain activity and how these patterns of brain activity were maintained over time. Importantly, the present study did not merely rely on self-reported aggression in order to assess and draw inferences about the relationship between aggressive behavior and patterns of brain activity, and this complements previous EEG studies that have used other laboratory paradigms or questionnaires to examine the relationship between frontal asymmetry, anger, and aggression.

The use of a laboratory aggression task may not be comparable to the contexts in which aggression manifests in the real world, but studying this phenomenon in a controlled environment increased our ability to attribute the present results to the stress manipulations experienced by participants. We selected a task involving only overt physical aggression, because we were interested in how stress influences violent and physically destructive behaviors and wanted to apply the results to the manifestation of these types of aggression. Given that the two forms of stress used in the present study may differentially affect aggressive responding among different individuals, future research should investigate whether other forms of aggression, such as verbal or relational aggression, are affected by the stressors we used or are more relevant for stress-induced aggression in particular subgroups, such as men versus women (Bjorkqvist, Lagerspetz, & Kaukiainen, 1992; Crick & Grotpeter, 1995; Verona, Reed, Curtin, & Pole, 2007).

Further research is needed to understand individual differences in relation to stress-induced approach behavior and aggression. We found preliminary evidence that approach-related brain activity was associated with trait anger and hostility only in the INT stress condition. Future research could investigate individual difference moderators of stress effects, including the influence of gender. Previous work in our lab has shown gender differences in behavioral reactions to stress (Verona & Curtin, 2006; Verona & Kilmer, 2007), but the present study was not powered to examine gender moderation, due to modest sample sizes in each stress/gender group. This program of research would be fruitful for uncovering who is at risk for aggression under particular stressful contexts (see Patrick & Verona, 2007) and would benefit from including markers of brain activity to further understand risk for stress-induced aggression.

Although we interpreted the shifts to more left frontal brain activity in response to stress as indicative of approach motivation, per Harmon-Jones (2004; Harmon-Jones & Allen, 1998), other interpretations are plausible. Heller et al. (1997), as well as Hofmann et al. (2005), have reported that anxious apprehension or worry is related to relatively more left than right frontal activity, as a function of the verbal rehearsal that is implemented by the left hemisphere. A recent study conducted with fMRI confirmed increased activation in the left hemisphere around Broca's area in participants scoring high on anxious apprehension (Engels et al., 2007). Thus, it is possible that shifts to left frontal activity in the two stress groups reflect apprehension, worry, or rumination related to the threat of air blasts. However, correlational analyses in the present study revealed no relationships between shifts in EEG frontal asymmetry and trait measures of anxiety and rumination. Instead, EEG asymmetry was positively correlated with measures of approach motivational tendencies. Nonetheless, it is possible that state changes in worry or rumination may have contributed to the brain activity observed, a possibility that can be investigated in future studies. Finally, the group differences in lateral frontal activity were not fully replicated with midfrontal EEG sites (see footnote 3); thus, the potential specificity of the effect to lateral frontal sites warrants further investigation.

Despite these limitations, the present results advance the research literature on motivational states and aggression by elucidating the stressful contexts and precipitants that can instigate aggressive responding. Furthermore, the EEG findings have implications for the continuing debate over the role of lateralized frontal asymmetry in the regulation of affective states and behaviors and in particular contexts (Davidson, 2004; Harmon-Jones, 2004), including the elicitation of approach behaviors by negative affective states. Parallel patterns of brain activity were elicited by the IMP and INT stress conditions, and stress exposure moderated the link between left frontal activity and aggressive behavior. Thus, the present results indicate that stress reactivity involving approach-related prefrontal brain activity may be an immediate precursor to aggression (cf. Verona & Kilmer, 2007), and the combination of tendencies toward heightened emotional reactivity and behavioral approach may be trait risk factors for chronic aggression and externalizing disorders (Patrick & Verona, 2007). Additional work is needed before the biological and environmental contributors to aggressive behavior will be fully understood. However, the present study sheds insight on a typically understudied pathway to chronic aggression, one that is initiated by heightened reactivity to stress and environmental irritants. Continued research on stress-induced pathways to aggression is important for the development of intervention strategies that will be effective for reducing aggressive reactions to stressful contexts, both impersonal and interpersonal in nature.

References

Anderson, C. A., Anderson, K. B., & Deuser, W. E. (1996). Examining an affective aggression framework: Weapon and temperature effects on aggressive thoughts, affect, and attitudes. *Personality and Social Psychology Bulletin, 22,* 366–376.

Berkowitz, L. (1989). Frustration-aggression hypothesis: Examination and reformulation. *Psychological Bulletin, 106,* 59–73.

Berkowitz, L. (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. *American Psychologist, 45,* 494–503.

Berkowitz, L., Cochran, S., & Embree, M. (1981). Physical pain and the goal of aversively stimulated aggression. *Journal of Personality and Social Psychology, 40,* 687–700.

Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion, 4,* 107–130.

Bettencourt, B. A., & Miller, N. (1996). Gender differences in aggression as a function of provocation: A meta-analysis. *Psychological Bulletin, 119,* 422–447.

Bjorkqvist, K., Lagerspetz, K. M., & Kaukiainen, A. (1992). Do girls manipulate and boys fight? Developmental trends in regard to direct and indirect aggression. *Aggressive Behavior, 18,* 117–127.

Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? *Psychological Review, 108,* 273–279.

Bushman, B. J., & Baumeister, R. F. (1998). Threatened egotism, narcissism, self-esteem, and direct and displaced aggression: Does self-love or self-hate lead to violence? *Journal of Personality and Social Psychology, 75,* 219–229.

Buss, A. H. (1961). *The psychology of aggression.* New York: Wiley.

Buss, A. H., & Warren, W. L. (2000). *The Aggression Questionnaire.* Los Angeles: Western Psychological Services.

Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion, 4,* 3–22.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319–333.

Coan, J. A., & Allen, J. J. B. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology, 40,* 106–114.

Coan, J. A., Allen, J. J. B., & McKnight, P. E. (2006). A capability model of individual differences in frontal EEG asymmetry. *Biological Psychology, 72,* 198–207.

Cook, I. A., O'Hara, R., Uijtdehaage, S. H., Mandelkern, M., & Leuchter, A. F. (1998). Assessing the accuracy of topographic EEG mapping for determining local brain function. *Electroencephalography and Clinical Neurophysiology, 107,* 408–414.

Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development, 66,* 710–722.

Davidson, R. J. (1983). Hemispheric specialization for cognition and affect. In A. Gale & J. Edwards (Eds.), *Physiological correlates of human behavior* (pp. 203–216). London: Academic Press.

Davidson, R. J. (2004). What does the prefrontal cortex "do" in affect: Perspectives on frontal EEG asymmetry research. *Biological Psychology, 67,* 219–233.

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Reviews of Psychology, 40,* 457–492.

Dill, J. C., & Anderson, C. A. (1995). Effects of frustration justification on hostile aggression. *Aggressive Behavior, 21,* 359–369.

Edguer, M., & Janisse, M. P. (1994). Type A behaviour and aggression: Provocation, conflict and cardiovascular responsivity in the Buss teacher-learner paradigm. *Personality and Individual Differences, 17,* 377–393.

Ellsworth, P. C., & Smith, C. A. (1988). From appraisal to emotion: Differences among unpleasant feelings. *Motivation & Emotion, 12,* 271–302.

Engels, A. S., Heller, W., Mohanty, A., Herrington, J. D., Banich, M. T., Webb, A. G., & Miller, G. A. (2007). Specificity of regional brain activity in anxiety types during emotion processing. *Psychophysiology, 44,* 352–363.

Forbes, E. E., Shaw, D. S., Fox, N. A., Cohn, J. F., Silk, J. S., & Kovacs, M. (2006). Maternal depression, child frontal asymmetry, and child affective behavior as factors in child behavior problems. *Journal of Child Psychology and Psychiatry, 47,* 79–87.

Fox, N. A., & Davidson, R. J. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month-old infants. *Developmental Psychology, 24,* 230–236.

Goldstein, J. H., Davis, R. W., & Herman, D. (1975). Escalation of aggression: Experimental studies. *Journal of Personality and Social Psychology, 31,* 162–170.

Gray, J. A. (1982). *The neuropsychology of anxiety.* New York: Oxford University Press.

Grillon, C., & Ameli, R. (1998). Effects of threat and safety signals on startle during anticipation of aversive shocks, sounds, or airblasts. *Journal of Psychophysiology, 12,* 329–337.

Hagemann, D. (2004). Individual differences in anterior EEG asymmetry: Methodological problems and solutions. *Biological Psychology, 67,* 157–182.

Harmon-Jones, E. (2003). Anger and the behavioral approach system. *Personality and Individual Differences, 35,* 995–1005.

Harmon-Jones, E. (2004). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology, 67,* 51–76.

Harmon-Jones, E. (2007). Trait anger predicts relative left frontal cortical activation to anger-inducing stimuli. *International Journal of Psychophysiology, 66,* 154–160.

Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and prefrontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology, 74,* 1310–1316.

Harmon-Jones, E., Lueck, L., Fearn, M., & Harmon-Jones, C. (2006). The effect of personal relevance and approach-related action expectation on relative left frontal cortical activity. *Psychological Science, 17,* 434–440.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality & Social Psychology, 80,* 797–803.

Heller, W. (1990). The neuropsychology of emotion: Developmental patterns and implications for psychopathology. In N. Stein, B. L. Leventhal, & T. Trabasso (Eds.), *Psychological and biological approaches to emotion* (pp. 167–211). Hillsdale, NJ: Erlbaum.

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology, 7,* 476–489.

Heller, W., & Nitschke, J. B. (1998). The puzzle of regional brain activity in emotion: A framework for understanding cognition in depression. *Cognition and Emotion, 12,* 421–447.

Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology, 106,* 376–385.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology, 100,* 535–545.

Hofmann, S. G., Moscovitch, D. A., Pizzagalli, D. A., Litz, B. T., Kim, H., & Davis, L. L. (2005). The worried mind: Autonomic and prefrontal activation during worrying. *Emotion, 5,* 464–475.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109,* 679–709.

Jasper, H. (1958). The 10–20 electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology, 10,* 371–375.

Koob, G. F., & Le Moal, M. (2001). Drug addiction, dysregulation of reward, and allostasis. *Neuropsychopharmacology, 24,* 97–129.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56,* 921–926.

Kruk, M. R., Halasz, J., Meelis, W., & Haller, J. (2004). Fast positive feedback between adrenocortical stress response and a brain mechanism involved in aggressive behavior. *Behavioral Neuroscience, 118,* 1062–1070.

Lang, P. J. (1979). A bio-informational theory of emotional imagery. *Psychophysiology, 16,* 495–512.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review, 97,* 377–398.

Lightsey, O. W., & Hulsey, C. D. (2002). Impulsivity, coping, stress, and problem gambling among university students. *Journal of Counseling Psychology, 49,* 202–211.

Lutz, C., Marinus, L., Chase, W., Meyer, J., & Novak, M. (2003). Self-injurious behavior in male rhesus macaques does not reflect externally directed aggression. *Physiology & Behavior, 78,* 33–39.

Miller, J. D., & Lynam, D. R. (2006). Reactive and proactive aggression: Similarities and differences. *Personality and Individual Differences, 41,* 1469–1480.

Nesic, J., & Duka, T. (2006). Gender specific effects of a mild stressor on alcohol cue reactivity in heavy social drinkers. *Pharmacology Biochemistry, and Behavior, 83,* 239–248.

Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 47,* 395–422.

Patrick, C. J., & Verona, E. (2007). The psychophysiology of aggression: Autonomic, electrocortical, and neuro-imaging findings. In D. Flannery, A. Vazsonyi, & I. Waldman (Eds.), *The Cambridge handbook of violent behavior and aggression* (pp. 111–150). New York: Cambridge University Press.

Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: A source-localization study. *Psychological Science, 16,* 805–813.

Putnam, S. P., Ellis, L. K., & Rothbart, M. K. (2001). The structure of temperament from infancy through adolescence. In A. Eliasz & A. Angleitner (Eds.), *Advances in research on temperament* (pp. 164–182). Lengerich, Germany: Pabst Science.

Raczkowski, D., Kalat, J. W., & Nebes, R. (1974). Reliability and validity of some Handedness Questionnaire items. *Neuropsychologia, 12,* 43–47.

Richman, J. A., & Flaherty, J. A. (1990). Alcohol-related problems of future physicians prior to medical training. *Journal of Studies on Alcohol, 51,* 296–300.

Rotton, J. (1979). The air pollution experience and physical aggression. *Journal of Applied Social Psychology, 9,* 397–412.

Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology, 103,* 92–102.

Stewart, J. (2003). Stress and relapse to drug seeking: Studies in laboratory animals shed light on mechanisms and sources of long-term vulnerability. *American Journal on Addictions, 12,* 1–17.

Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8,* 204–210.

Taylor, S. P. (1967). Aggressive behavior and physiological arousal as a

function of provocation and the tendency to inhibit aggression. *Journal of Personality, 35,* 297–310.

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: If you feel bad, do it! *Journal of Personality and Social Psychology, 80,* 53–67.

Trapnell, P. D., & Campbell, J. D. (1999). Private self-consciousness and the five-factor model of personality: Distinguishing rumination from reflection. *Journal of Personality and Social Psychology, 76,* 284–304.

Tucker, D. M. (1981). Lateral brain function, emotion, and conceptualization. *Psychological Bulletin, 89,* 19–46.

Verona, E., & Curtin, J. J. (2006). Gender differences in the negative affective priming of aggressive behavior. *Emotion, 6,* 115–124.

Verona, E., & Kilmer, A. (2007). Stress exposure and affective modulation of aggressive behavior in men and women. *Journal of Abnormal Psychology, 116,* 410–421.

Verona, E., Patrick, C. J., & Joiner, T. E. (2001). Psychopathy, antisocial personality, and suicide risk. *Journal of Abnormal Psychology, 110,* 462–470.

Verona, E., Patrick, C. J., & Lang, A. R. (2002). A direct assessment of the role of state and trait negative emotion in aggressive behavior. *Journal of Abnormal Psychology, 111,* 249–258.

Verona, E., Reed, A., Curtin, J., & Pole, M. (2007). Gender differences in emotional and overt/covert aggressive responses to stress. *Aggressive Behavior, 33,* 261–271.

Wacker, J., Heldmann, M., & Stemmler, G. (2003). Separating emotion and motivational direction in fear and anger: Effects on frontal asymmetry. *Emotion, 3,* 167–193.

Watson, D., & Clark, L. A. (1994). *The PANAS-X: Manual for the Positive and Negative Affect Schedule–Expanded Form.* Unpublished manuscript, University of Iowa.

Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, M. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104,* 3–14.

Zinser, M. C., Fiore, M. C., Davidson, R. J., & Baker, T. B. (1999). Manipulating smoking motivation: Impact on an electrophysiological index of approach motivation. *Journal of Abnormal Psychology, 108,* 240–254.

Received April 17, 2008
Revision received August 21, 2008
Accepted August 27, 2008 ■