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Anger Style, Psychopathology, and Regional Brain Activity

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Abstract

Depression and anxiety often involve high levels of trait anger and disturbances in anger expression. Reported anger experience and outward anger expression have recently been associated with left-biased asymmetry of frontal cortical activity, assumed to reflect approach motivation. However, different styles of anger expression could presumably involve different brain mechanisms and/or interact with psychopathology to produce various patterns of brain asymmetry. The present study explored these issues by comparing resting regional electroencephalographic activity in participants high in trait anger who differed in anger expression style (high anger-in, high anger-out, both) and participants low in trait anger, with depression and anxiety systematically assessed. Trait anger, not anger-in or anger-out, predicted left-biased asymmetry at medial frontal EEG sites. The anger-in group reported higher levels of anxious apprehension than did the anger-out group. Furthermore, anxious apprehension moderated the relationship between trait anger, anger-in, and asymmetry in favor of the left hemisphere. Results suggest that motivational direction is not always the driving force behind the relationship of anger and left frontal asymmetry. Findings also support a distinction between anxious apprehension and anxious arousal.

Emotional disturbances involving anger are associated with adverse correlates, course, and outcomes of mood and anxiety disorders (e.g., Erwin, Heimberg, Schneier, & Liebowitz, 2003; Fava, Anderson, & Rosenbaum, 1990; Foa, Riggs, Massie, & Yarczower, 1995; McElroy, 1999). Anger dysregulation could serve to maintain or exacerbate symptoms of depression and anxiety, leading to relapse, increased distress, and/or reductions in social and occupational function (Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003). Anger and related constructs such as hostility and aggression are distinguishable but are often used interchangeably (Martin, Watson, & Wan, 2000). In order to further explore the relationship between anger and psychopathology, it is important that anger experience and expression be appropriately distinguished. Anger experience is commonly defined as a feeling that is evoked when individuals perceive a situation as involving injustice or when they believe that they are being treated badly or unfairly (Averill, 2001). Depression and anxiety are associated with high levels of trait anger experience (e.g., Deffenbacher, Oetting, Lynch, & Morris, 1996a; Koh, Kim, & Park, 2002; Riley, Triber, & Woods, 1989), and in recent years clinicians have suggested anger-management strategies to improve treatment outcome for

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depressed and anxious individuals (e.g., Cahill, Rauch, Hembree, & Foa, 2003; Eckhardt, 1999).

People who experience anger differ, however, in their style of anger expression, specifically whether anger is directed toward others. Spielberger (1988) developed the State-Trait Anger Expression Inventory (STAXI) in order to quantify multiple facets of anger expression and the experience of state and trait anger. To quantify anger expression, the STAXI measures self-report of anger-out expression (the expression of angry feelings in the form of aggressive verbal or motor behavior directed toward people or objects) and anger-in expression (the suppression or holding in of angry feelings). Anger-out and anger-in are not conceived as opposite poles of a single continuum but as orthogonal and potentially co-occurring (e.g., Deffenbacher, 1992; Knight, Chisolm, Paulin, & Waal-Manning, 1988).

Differentiation of anger expression styles is important when exploring the relationship of anger to psychopathology, since these styles are associated with divergent implications for both physical and mental health. For example, a meta-analytic review has indicated that anger-in is associated with higher resting blood pressure, worsened cardiovascular disease severity, and increased cardiovascular mortality (Jorgensen, Johnson, Kolodziej, & Shreer, 1996). Anger-out has been associated with extreme levels of narcissism and self esteem (Papps & O'Carroll, 1998), drug addiction (De Moja & Spielberger, 1997) and binge-eating and impulsivity (Penas-Lledo, Fernandez, & Waller, 2004). Strong associations have been reported between depression and anger-in (e.g., Brody, Haaga, Kirk, & Solomon, 1999; Deffenbacher et al., 1996b) and between anxiety and anger-out (Feeny, Zoellner, & Foa, 2000; Hull et al., 2003; Spielberger, Reheiser, & Sydeman, 1995), whereas evidence for associations between depression and anger-out or between anxiety and anger-in is inconsistent (e.g., Bridewell & Chang, 1997; Brody et al., 1999; Hull et al., 2003; Koh et al., 2002; Riley et al., 1989; Spielberger et al., 1995; Whiteside & Abramowitz, 2004).

In recent years, considerable research has been devoted to the critical roles motivational systems play in the experience and expression of emotions such as anger, sadness, and fear (e.g., Cacioppo & Bernston, 1999; Carver, 2001, 2004; Harmon-Jones, 2003a; Lang, Bradley, & Cuthbert, 1990; Wacker, Heldmann, & Stemmler, 2003). It has been argued that a behavioral activation system supports positive emotions, responds to rewarding stimuli, and leads to approach behavior and active avoidance, whereas a behavioral inhibition system supports anxiety, responds to punishing stimuli, and leads to inhibition of action, passive avoidance, and heightened arousal (Gray, 1982, 1987; Gray & McNaughton, 1996). Models of emotion have been developed that relate aspects of these systems to specific patterns of regional brain activity. Davidson (1983, 1984, 1995) asserted that an approach system is linked to greater left than right frontal activity and that a withdrawal system associated with withdrawal from aversive stimuli is associated with greater right than left frontal activity in right-handed subjects. Research has provided support for this model, demonstrating that left frontal resting electroencephalogram (EEG) asymmetry is associated with approach motivation (Coan & Allen, 2003; Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997), whereas right frontal resting EEG asymmetry is associated with withdrawal motivation (Sutton & Davidson, 1997; but see Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2004, for discrepant results).

Unlike fear and sadness, which are often conceptualized as negative emotions involving withdrawal motivation, anger is commonly construed to combine negative emotion or unpleasant valence with approach motivation (Harmon-Jones, 2003a, 2003b), although in fact anger may sometimes have important appetitive qualities rather than being exclusively negative in valence. EEG studies have indicated that trait anger experience (Harmon-Jones & Allen, 1998), anger-out (Hewig et al., 2004), and approach-related state anger (Harmon-

Jones & Sigelman, 2001; Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003; Wacker et al., 2003) are associated with greater left than right frontal activity, consistent with research linking approach motivation to asymmetry in favor of the left hemisphere (e.g., Harmon-Jones & Allen, 1997). However, not all studies inducing state anger have found this asymmetry. Waldstein et al. (2000) found comparable amounts of left and right frontal activation during state anger. In addition, not all anger experience is necessarily associated with anger expression in the form of approach motivation. Anger-in involves withdrawal motivation in the form of outward suppression of angry feelings and therefore could be associated with frontal asymmetry in favor of the right hemisphere. However, one study exploring the relationship between trait anger-in and EEG asymmetry found no relationship between anger-in and frontal asymmetry in either direction (Hewig et al., 2004), and another study examining state anger found greater left than right frontal EEG activation in response to an imagined anger-inducing event involving withdrawal from the target of anger (Wacker et al., 2003). Although there are exceptions (e.g., Hewig et al., 2004; Wacker et al., 2003), appropriate distinctions have not consistently been made between anger experience and anger expression style in this literature. Previous EEG asymmetry studies have not explicitly selected participants on the basis of high trait anger, anger-out, and/or anger-in in order to represent the amount of anger experience and expression typical of that seen in individuals with psychopathology. More research is needed to identify the relationships among styles of anger expression, regional brain activity, and depression and anxiety.

Since high levels of anger experience and expression are highly comorbid with depression and anxiety, frontal asymmetry arising in association with different facets of anger might be differentially moderated by co-occurring depression and/or anxiety (see Coan & Allen, 2004, for discussion of moderators of EEG asymmetry). Individuals with past or current depression sometimes display a rightward shift in resting frontal asymmetry (e.g., Gotlib, Ranganath, & Rosenfield, 1998; Henriques & Davidson, 1990, 1991). Some evidence suggests that this pattern of results does not change with fluctuating symptom severity (e.g., Allen, Urry, Hitt, & Coan, 2004b; but see Debener et al., 2000). Decreased left frontal activity (rightward asymmetry) may be a trait-like biological diathesis for depression consistent with reduced approach motivation (Davidson, Pizzagalli, Nitschke, & Putnam, 2002). However, several EEG studies have not observed left frontal hypoactivity in depression (e.g., Harmon-Jones et al., 2002; Kentgen et al., 2000; Metzger et al., 2004; Nitschke, Heller, Palmieri, & Miller, 1999; Reid, Duke, & Allen, 1998). Inconsistent results may be due to several factors, including methodological differences such as choice of EEG reference (e.g., Debener et al., 2000; Metzger et al., 2004) or diagnostic heterogeneity in depressed groups (see Davidson, 1998).

Heterogeneity in depressed groups may also be attributable to varying amounts or types of comorbid anxiety (Heller, Koven, & Miller, 2003; Heller & Nitschke, 1998; Mineka, Watson, & Clark, 1998; Nitschke, Heller, Imig, McDonald, & Miller, 2001). The comorbidity of mood and anxiety disorders is critical to take into account, since lifetime diagnoses of anxiety and depression co-occur in up to 40-75% of individuals with either diagnosis (Clark, 1989; Kessler, 1995). Researchers have typically not assessed any type of anxiety in studies of EEG asymmetry in depression. Conversely, research on frontal asymmetry in anxiety has suffered from methodological problems similar to those seen in the depression literature, including the uncontrolled heterogeneity of anxious groups (e.g., Wiedemann et al., 1999) and failure to measure comorbid depression. Heller, Miller, and colleagues (Heller, Etienne, & Miller, 1995; Heller et al., 2003; Heller & Nitschke, 1998) have argued that distinct types of anxiety are differentially associated with depression and hemispheric asymmetry. Various symptoms such as sympathetic nervous system hyperactivity and panic are components of the construct of anxious arousal (Clark & Watson, 1991; Heller et al., 1995; Heller & Nitschke, 1998; Heller, Nitschke, Etienne, &

Miller, 1997; Nitschke et al., 2001; Watson et al., 1995). In contrast, worry and anticipation of future threat are components of the construct of anxious apprehension (Barlow, 1988, 1991). Heller, Miller, and colleagues have hypothesized that these anxiety types are distinguishable in brain asymmetry (e.g., Heller, 1993; Nitschke et al., 1999). Converging evidence (e.g., Engels et al., 2007; Nitschke et al., 1999) suggests that anxious arousal is associated with an asymmetry in favor of the right hemisphere for frontal as well as for posterior regions, consistent with hypotheses that the right hemisphere is involved in modulation of peripheral and behavioral arousal (Heller, 1993; Heller et al., 1997) and threat response (Nitschke & Heller, 2002; Nitschke, Heller, & Miller, 2000). In contrast, anxious apprehension has been associated with increased left-hemisphere activity, consistent with the considerable linguistic processing likely to be involved in worry (Engels et al., 2007; Heller et al., 1997; Nitschke & Heller, 2002; Nitschke et al., 2000; Nitschke et al., 1999).

The present study examined whether anger expression style predicts differential resting frontal EEG asymmetry as a function of anger-out, anger-in, or a combined style of anger expression. The study also examined whether depression, anxious apprehension, and anxious arousal moderate relationships between anger experience or expression and brain asymmetry. Positive and negative affect as well as approach and withdrawal motivation were assessed in order to identify possibly divergent relationships with anger expression styles that could explain distinct patterns of regional brain activity.

Primary hypotheses were: 1) Participants with high trait anger have more left frontal asymmetry than participants with low trait anger, consistent with previous research (e.g., Harmon-Jones & Allen, 1998). 2) In high trait anger individuals, an anger-out expression style is associated with more leftward lateralization than is an anger-in expression style, since anger-out is an approach strategy used in the context of anger (Hewig et al., 2004). It was predicted that an anger-in style would instead be associated with greater right than left frontal EEG activity, since it is a withdrawal strategy.

In addition to the primary hypotheses about subtypes of anger, depression and anxiety were assessed for possible moderation of relationships between anger expression style and patterns of brain activity. For example, the combined anger-in / anger-out group could show any of several patterns, in part as a function of co-occurring depression or anxiety on either a group or individual basis.

Method

Participants

Laboratory participants were 65 undergraduates (30 male, 35 female) enrolled in an introductory psychology class. They were chosen from individuals tested in large groups with a battery of questionnaires. Selected participants were right-handed as determined by the Edinburgh Handedness Inventory (Oldfield, 1971).

Laboratory participants for a two-session EEG protocol were selected on the basis of responses on the Trait Anger, Anger Expression-Out, and Anger Expression-In subscales of the State Trait Anger Expression Inventory (STAXI-2; Spielberger, 1999). STAXI-2 Trait Anger is a ten-item scale that has participants rate how they generally feel about statements regarding anger experience, whereas STAXI-2 Anger Expression-Out and Anger Expression-In are each eight-item scales in which participants rate how they generally react or behave when angry or furious (1=almost never, 2=sometimes, 3=often, 4=almost always). Students who left more than 5% of the items in the battery of questionnaires blank were excluded.

Participants were recruited for four groups that differed in amounts of trait anger and anger expression style. A similar group selection procedure has been used in other anger studies (e.g., Suchday & Larkin, 2001). Those scoring below the 50th percentile on all three STAXI-2 subscales met criteria for the potential low anger group. Those scoring at or above the 90th percentile on both STAXI-2 Trait Anger and Anger Expression-In subscales and below the 50th percentile on the STAXI-2 Anger Expression-Out subscale met criteria for the anger-in group. Students scoring at or above the 90th percentile on both STAXI-2 Trait Anger and Anger Expression-Out subscales and below the 50th percentile on the STAXI-2 Anger Expression-In subscale met criteria for the anger-out group. Individuals scoring at or above the 90th percentile on all 3 STAXI-2 subscales met criteria for the combined anger group.

Of over 2000 right-handers completing questionnaires, 594 met criteria for the low anger group, 25 for anger-in, 64 for anger-out, and 37 for the combined anger group. All those meeting criteria for the three anger groups and a random subset of the low anger group were invited to participate in two laboratory EEG sessions. Potential participants were excluded if they reported a history of epilepsy or loss of consciousness greater than 10 minutes. In addition, participants were screened but not excluded for prescribed medications. One male participant reported fluoxetine use, whereas one female participant was taking propranolol. Analyses were performed with and without these participants to assess whether psychotropic medication affected questionnaire or EEG results. Since all results did not change with and without their inclusion in analyses, these participants were retained in the sample. The 65 participants completing the first EEG session were 20 (11 women) low anger, 11 (4 women) anger-in, 25 (13 women) anger-out, and 9 (7 women) combined anger participants. Individuals were age 18 to 21 (mean 18.66 years, $SD=0.83$) and 77% European American. Table 1 provides additional information about age, handedness, gender, and ethnicity, none of which distinguished the four groups. Nine of the 65 participants declined to participate in the second EEG session, but the number of participants who discontinued the study did not differ as a function of group, $\chi^2(3) = .58, p > .90$.

To verify that the four groups could still be psychometrically distinguished at the time of EEG recording, STAXI-2 scores obtained at the first and second EEG session were examined and compared to scores from the screening session. As shown in Table 2, the intended group similarities and differences remained robust, with no substantial, consistent regression to the mean.

Apparatus and Physiological Recording

For psychophysiological recording, the participant was seated in a comfortable chair in a quiet room connected to the adjacent equipment room by intercom. Recording procedures generally followed the recommendations of Picton et al. (2000). Electrode placement was based on the International 10-20 System (Jasper, 1958). A Lycra stretchable cap (Electro-Cap International, Eaton, OH) with tin electrodes was used for midline (Fz, Cz, Pz), left and right frontopolar (Fp1, Fp2), midfrontal (F3, F4), lateral frontal (F7, F8), central (C3, C4), anterior temporal (T3, T4), posterior temporal (T5, T6), parietal (P3, P4), occipital (O1, O2) and mastoid (A1, A2) sites, with A1 as the reference for all other sites. Tin electrodes near the outer canthus of each eye and above and below the left eye referenced to A1 provided four horizontal and vertical electrooculogram (EOG) channels for offline eye movement artifact correction of the EEG data. Electro-Gel (Electro-Cap International, Eaton, OH) served as the conductive gel for all sites. Mastoid and EOG sites were prepared with rubbing alcohol and gently abraded with NuPrep (Weaver and Company, Aurora, CO) via cotton swabs. EEG sites were prepared via gentle abrasion using cotton swabs and Electro-Gel. EEG activity recorded from the right mastoid was used to compute an average mastoid

reference offline (Miller, Lutzenberger, & Elbert, 1991; Nunez, 1981). Electrode impedance was below 20 K ohms. Homologous electrodes were within 5 K ohm of each other.

Stimulus presentation and onset of physiological data collection were controlled by STIM software (James Long Company, Caroga Lake, NY) run on a PC. Physiological data were collected by SNAPSHOT software (HEM Data Corporation, Southfield, MI). Recordings were amplified by a 64-channel Isolated Bioelectric Amplifier (James Long Company, Caroga Lake, NY, and SA Instrumentation Company, San Diego, CA) with EEG channels set to a gain of 10 K, EOG channels set to a gain of 2 K, and half-amplitude frequency cutoffs at 0.1 and 100 Hz. Analog signals were digitized continuously during each recording period at 250 Hz using a 12-bit A/D converter.

Procedure

In the first of two EEG sessions, a tour of the laboratory was conducted, a detailed description of the study was presented, and written informed consent was obtained. The remaining procedure was identical for both sessions. If EEG asymmetry is to be considered an index of a traitlike construct of affective reactivity, two important psychometric criteria it should possess are internal consistency and stability over time (e.g., Hagemann, Naumann, Thayer, & Bartussek, 2002; Tomarken & Keener, 1998). EEG asymmetry has been found to have excellent internal consistency reliability but only moderate stability ($r=.60$) over a period of three weeks (Tomarken, Davidson, Wheeler, & Kinney, 1992). To address this problem, Davidson (1998) recommended either screening for subjects that possess stable or extreme scores on a particular trait or testing individuals on two occasions approximately six weeks apart, then averaging these assessments to obtain a reliable asymmetry index for each subject. The present study employed the latter strategy, with subjects assessed twice, two months apart. Experimenters in both laboratory sessions were blind to group membership of participants.

After electrode application and amplifier calibration check, participants were instructed to relax during eight 1-min resting baseline periods during which EEG was recorded. Prior to the start of each period, participants saw directions (“eyes open” or “eyes closed”) on a computer screen in front of them. During the eyes-open recording, participants were instructed to focus their eyes on a small cross in the center of the computer screen. After each eyes-closed recording was completed, via intercom the experimenter instructed participants to open their eyes. Per the recommendations of Tomarken et al. (1992), there were four eyes-open (O) and four eyes-closed (C) baseline periods, ordered COCCOCCO for half the subjects and OCCOCCOOC for the other half.

Participants completed the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) to index state levels of positive and negative emotion immediately after cessation of physiological recording. Participants then completed a battery of questionnaires, including the STAXI-2 Trait Anger, Anger Expression-Out, and Anger Expression-In scales (Spielberger, 1999) to confirm group membership, the STAXI-2 Anger Control-In and Anger Control-Out scales (Spielberger, 1999) and The Aggression Questionnaire (Buss & Perry, 1992) to characterize subjects as some past anger research has done (e.g., Harmon-Jones & Allen, 1998; Hewig et al., 2004), and the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, and Borkovec, 1990) to measure anxious apprehension. The Anger Control-In scale measures attempts to control angry feelings by calming down (e.g., “I take a deep breath and relax”), whereas the Anger Control-Out scale measures the control of angry feelings by preventing aggression toward other people or objects (e.g., “I control my temper”). The Anhedonic Depression scale of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995) was used, because it is much less correlated with anxiety questionnaires than previous self-report measures of depression (Nitschke et

al., 2001) and thus appears to be a better candidate for use in asymmetry studies specifically measuring depression. In particular, a subscale of 8 items was used that distinguishes depression from other items reflecting low positive affect more generally (Nitschke et al., 2001). The BIS/BAS scales (Carver & White, 1994) were used to assess approach and withdrawal motivation, and the Anxious Arousal scale from the MASQ was administered to measure anxious arousal. The PSWQ and MASQ Anxious Arousal scales have low correlations with each other as well as with MASQ Anhedonic Depression and other more general measures of anxiety and depression (Nitschke et al., 2001).

Data Reduction and Analysis

For each session, EEG data were converted to microvolts and rereferenced using the computed linked mastoids derivation so that each channel reflected the voltage between the relatively inactive mastoids and an active scalp site. Although source localization analysis was not undertaken in the present study, it is reasonable to attribute the resulting voltage, particularly alpha band activity, to regions underlying the scalp sites, with regional brain activity inversely proportional to regional alpha power (Davidson, 1988; Shagass, 1972). “Activity” is used hereinafter to refer to brain activity (not its inverse, amount of alpha activity). An eye-movement correction program processed 1-s segments to remove EOG artifact from the EEG (Gratton, Coles, & Donchin, 1983; Miller, Gratton, & Yee, 1988), allowing retention of nearly all EEG epochs. Using software locally adapted from that of J. Senulis and R. Davidson (personal communication, November 17, 1994), a Hamming window was applied to 1.024-s epochs of continuous EEG. Epochs were overlapped by 50% to capture data at the ends of the epochs that would otherwise be attenuated by the Hamming windowing. The power spectrum, including alpha band activity, was obtained by applying a fast Hartley transform (FHT; Bracewell, 1984) to each epoch and then averaging all the power spectra across the 119 epochs within each 60-s baseline.

To obtain alpha values for each baseline, FHT results were output in 0.977-Hz bins for activity between 8 and 12 Hz. Power density in the alpha band was obtained by averaging across four bins (8.789-9.766 Hz, 9.766-10.742 Hz, 10.742-11.719 Hz, 11.719-12.696 Hz). The average alpha power was calculated separately for each baseline to examine internal consistency, averaged across baselines within session to examine test-retest reliability across sessions, and then averaged across sessions for each of the 19 electrode sites for further analysis. Because the distributions for the alpha values at the various EEG sites were consistently skewed, natural log transformations were performed, and normal distributions were obtained. ANOVAs were carried out using alpha power scores from each electrode site to examine the relationship between anger style and hemisphere differences.

For regression analyses incorporating anxiety and depression measures, asymmetry scores were computed as the difference of the natural-log-transformed alpha power density scores that had symmetrical left and right locations (Fp1 and Fp2, F3 and F4, F7 and F8, C3 and C4, T3 and T4, T5 and T6, P3 and P4, and O1 and O2). For the asymmetry score ($\ln[\text{Right}] - \ln[\text{Left}]$), more positive values indicate more right-hemisphere alpha and thus more left-hemisphere brain activity. The asymmetry score provides a degree of correction for overall alpha power, which could potentially be confounded with the magnitude of asymmetry, and mitigates the impact of individual differences in skull thickness, etc. that could affect EEG signal amplitude (Allen, Coan, & Nazarian, 2004a).

Results

EEG and questionnaire results, averaged across the two laboratory sessions, are presented only for participants who completed both sessions, for a final N of 56 participants.

Questionnaire Analysis of Anger Groups

Questionnaire scores averaged across session were used as dependent variables to examine group differences in anxious apprehension, anxious arousal, anhedonic depression, approach and withdrawal motivation, positive and negative affect, aggression, anger, hostility, and anger control. An omnibus, doubly multivariate MANOVA was computed to determine whether groups differed on questionnaire measures. An overall effect for group emerged from this analysis, Wilks Lambda=.06, $F(51, 108) = 3.38$, $p < .001$. To follow up on this main effect, ANOVAs were performed for each questionnaire separately (see Table 3 for group comparisons as well as means and standard deviations for each questionnaire). Significant between-subjects effects for individual scales were followed up with Newman-Keuls tests conducted at the .05 level. No participants displayed questionnaire scores greater than 3 standard deviations from the mean.

As anticipated in the Introduction, groups differed in reported psychopathology, specifically anxious apprehension and anhedonic depression (see Table 3). Anger-in and combined groups had higher PSWQ scores than did anger-out and low anger groups. Anger-in and combined groups displayed higher scores than low anger participants on Anhedonic Depression. Anger-in participants also had less positive affect and higher withdrawal motivation than anger-out and low anger groups. Group differences on The Aggression Questionnaire and STAXI anger control (reported in Table 3) were examined because of significant correlations reported between the subscales and anterior asymmetry (Harmon-Jones & Allen, 1998; Hewig et al., 2004).

Hemispheric EEG Analysis of Anger Groups

Group differences in hemispheric activity were examined using an omnibus univariate ANOVA design, with group (anger-in, anger-out, combined, and low anger) as the between-subjects variable and hemisphere (left, right) and region (frontopolar, medial frontal, lateral frontal, anterior temporal, central, posterior temporal, parietal, and occipital) as within-subjects variables. The dependent variables were EEG alpha power at non-midline electrode sites averaged across sessions (Fp1, Fp2, F3, F4, F7, F8, T3, T4, C3, C4, T5, T6, P3, P4, O1, O2). Huynh-Feldt corrections were used to adjust degrees of freedom due to sphericity violations where appropriate. One participant from the anger-out group with alpha power scores greater than three standard deviations from the mean was excluded (resulting $N = 55$). A main effect of region indicated that parietal and occipital regions had the highest levels of alpha power (and the lowest levels of brain activity), whereas anterior temporal region displayed the lowest alpha power (and the most brain activity), $F(7, 357) = 84.42$, $p < .001$, $\epsilon = .33$, $\eta^2 = .62$. The Hemisphere x Region interaction demonstrated that medial and lateral frontal regions had greater right than left brain activity, whereas the posterior temporal region displayed more left than right activity, $F(7, 357) = 6.38$, $p < .001$, $\epsilon = 1.00$, $\eta^2 = .11$. These results were qualified by the Group x Hemisphere x Region interaction, which tested the prediction that the four groups would differ in frontal asymmetry, $F(21, 357) = 2.32$, $p = .001$, $\epsilon = 1.00$, $\eta^2 = .12$. The analysis was repeated adding gender as a covariate (see Miller & Chapman, 2001) to ensure that gender was not driving this interaction. The Group x Hemisphere x Region interaction remained significant when variance related to gender was removed, $F(21, 357) = 1.80$, $p = .035$, $\epsilon = .72$, $\eta^2 = .10$. Group x Hemisphere ANOVAs were then computed for each region in order to interpret group differences in hemispheric activity.

Figure 1 illustrates Group x Hemisphere interactions for medial frontal, central, and posterior temporal regions. For the medial frontal (F3/F4) region, $F(3, 51) = 3.83$, $p = .015$, $\epsilon = 1.00$, $\eta^2 = .18$, Newman-Keuls indicated that the low anger group displayed asymmetry in favor of less left than right medial frontal activity, whereas the other groups were not

lateralized. For central (C3/C4) and posterior temporal (T5/T6) regions, $F(3, 51) = 2.73$, $p = .053$, $\varepsilon = 1.00$, $\eta^2 = .14$, and $F(3, 51) = 4.53$, $p = .007$, $\varepsilon = 1.00$, $\eta^2 = .21$, respectively, the anger-in group displayed more left- than right-hemisphere activity, whereas the other groups were not lateralized. The occipital Group x Hemisphere interaction was marginally significant, $F(3, 51) = 2.47$, $p = .072$, $\eta^2 = .13$, and Newman-Keuls tests revealed no significant group differences for this region.

Anger and Questionnaires Predicting EEG Asymmetry

Due to anger group differences in psychopathology, affect, and motivation questionnaires, these questionnaires could not be used as covariates in an analysis of covariance to examine hemispheric differences in regional brain activity among anger groups (see Miller & Chapman, 2001, for an explanation). Anger group comparisons, questionnaire scores, and their interactions were instead used as predictors in hierarchical linear regressions in order to determine whether they contributed significant variance to EEG asymmetry scores. These analyses addressed whether questionnaire scores, especially those reflecting anxious apprehension, anxious arousal, and anhedonic depression, were moderators of the relationship between aspects of anger and asymmetry scores. Participants with asymmetry scores greater than 3 standard deviations from the mean for a specific region (e.g., F4 - F3) were excluded from regression analyses for that particular region. One outlier met this criterion for each region (leaving $N = 55$) with the exception of T6 - T5 ($N = 56$).

Anger group comparisons (via three orthogonal dummy-coded variables) were entered in the first step to determine how different types of anger (e.g., trait anger, anger style) contributed to EEG asymmetry. The variables representing the four groups were: 1) anger-in group vs. anger-out group, 2) anger-in and anger-out groups vs. combined group, and 3) high anger groups vs. low anger group. The first comparison addressed whether the style of anger expression predicts asymmetry. The second comparison addressed whether pure anger expression differs from combined anger expression. The third comparison addressed whether trait anger experience (regardless of anger expression style) predicts asymmetry. In combination, the three group comparisons indeed accounted for significant lateralization in F4 - F3 and T6 - T5 asymmetry scores, marginally significant for C4 - C3. Table 4 demonstrates that high trait anger (vs. low trait anger) and an anger-in style (vs. anger-out style) were associated with greater left- than right-hemisphere activity (indicated by positive t values) for these regions. Pure anger expression (vs. combined anger expression) did not predict asymmetry for any of the 8 scores ($p > .18$).

One questionnaire at a time (PANAS PA, PANAS NA, BAS, BIS, PSWQ, MASQ-AA, MASQ-AD) was entered in the second step to see whether psychopathology, affect, or motivation contributed significant additional variance in predicting asymmetry. For the PANAS and BIS/BAS questionnaires, regressions were performed only for the three frontal regions (Fp2 - Fp1, F8 - F7, and F4 - F3), because relevant asymmetry findings in previous research were specific to these regions (e.g., Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Since hemispheric differences have been implicated more broadly (not confined to frontal regions) in anxious apprehension, anxious arousal, and anhedonic depression, regressions for the PSWQ, MASQ-AA, and MASQ-AD were performed for all eight regions. Table 5 illustrates that only the MASQ-AA contributed significant unique variance to asymmetry scores in the full model, with higher scores associated with greater right- than left-hemisphere activity (indicated by negative t values).

Finally, the interactions between anger dummy comparisons and questionnaire scores were entered in a third step, to evaluate whether questionnaire scores moderated the relationship between aspects of anger and EEG asymmetry scores (see Coan & Allen, 2004, for discussion of moderators and mediators of EEG asymmetry). Table 6 indicates that the full

model including the anger comparisons, the PSWQ, and their interactions was significant for T6 – T5 and marginally so for C4 – C3. The PSWQ interacted with trait anger and anger-in style to predict asymmetry such that higher scores were associated with more left-hemisphere activity (indicated by positive t values). No significant interactions were found for BIS/BAS and PANAS scales in the frontal regions nor MASQ-AA and MASQ-AD in the eight regions.

Discussion

The present study investigated whether different anger expression styles are associated with distinct patterns of baseline hemispheric activity. A second goal was to determine the extent to which anxiety or depression moderates the relationship between anger expression and frontal asymmetry. Four substantial findings emerged that elucidate the relationship between anger style, psychopathology, and functional asymmetry. First, it was predicted that participants with high trait anger, regardless of anger expression style, would display higher left frontal asymmetry than participants low in trait anger, consistent with previous research (e.g., Harmon-Jones & Allen, 1998). Regression analyses supported this hypothesis. Second, in line with motivational models of emotion (e.g., Davidson, 1983, 1984, 1995; Sutton & Davidson, 1997), it was predicted that in high trait anger participants an anger-out style would be associated with greater left frontal asymmetry than would an anger-in style, as left frontal activity has been interpreted as part of approach motivation. This hypothesis was not supported. The two remaining findings differentiate types of anxiety on the basis of their differential relationships to anger and regional brain asymmetry. Anxious arousal predicted right frontal asymmetry, replicating previous research and theory that has sometimes pointed to the right frontal quadrant and sometimes to the right hemisphere more broadly (e.g., Engels et al., 2007; Heller et al., 1997; Nitschke et al., 1999; Nitschke et al., 2000; Nitschke & Heller, 2005; Shackman et al., 2006), whereas anxious apprehension strengthened the relationship between trait anger / anger-in and leftward asymmetry for central and posterior temporal sites. It appears that anxious apprehension is more associated with certain types of anger expression (anger-in and combined types), whereas anxious arousal does not differentiate anger groups from each other or from the low anger group.

In participants with high trait anger, anger-in differed from anger-out for questionnaire measures as well as for brain activity in regions other than frontal cortex. Anger-in was associated with higher levels of withdrawal motivation than anger-out, which validates the present proposal that anger-in is a withdrawal strategy used in response to angry feelings. In addition, anger-in was associated with lower levels of positive affect, consistent with previous research on emotion suppression (Gross & John, 2003). Previous research examining relationships among anger expression style, anxiety, and depression did not differentiate types of anxiety or examine anhedonic depression, a facet of depression that is more easily distinguishable from various types of anxiety (Nitschke et al., 2001). In the present study the anger-in group endorsed higher levels of anxious apprehension than did the anger-out group, but the groups did not differ on anxious arousal or anhedonic depression. These differences have implications for treatment conceptualizations for individuals high in an anger-in versus an anger-out style of expression. Anger-in could serve to maintain worry and lower positive affect, and vice versa, making it an important issue to assess and address during therapy. Anger-out, in contrast, is specifically associated with high levels of physical and verbal aggression, making it less relevant to worry but important to assess and address in externalizing disorders.

In participants with high trait anger, anger-in was associated with asymmetry in favor of the left hemisphere in central and posterior temporal brain regions. Anger induction paradigms used in PET studies have elicited temporal lobe activation in response to angry narratives

and imagery (Damasio et al., 2000; Dougherty et al., 1999, 2004; Kimbrell et al., 1999) and more specifically in response to imagined restraint of anger (Pietrini et al., 2000). However, for all but one of these studies it is impossible to disentangle feelings of anger from an anger-in style, since participants were immobilized, and their facial expressions and behavior were not monitored for signs of anger expression or suppression. It could be that state-induced temporal lobe hyperactivity is a more trait-like phenomenon in individuals who display an anger-in style.

The leftward asymmetry in central and temporal regions in the anger-in group could reflect increased language processing similar to that displayed in anxious apprehension, since the anger-in group, as well as the combined group (which shares an anger-in expression style), reported more anxious apprehension than the anger-out and low anger groups. In addition, anxious apprehension moderated the relationship between trait anger and anger-in and leftward asymmetry for these regions. This is not surprising, since neuroimaging research demonstrates a strong relationship between anxious apprehension and left hemisphere asymmetry (Engels et al., 2007; Nitschke et al., 2000; Nitschke & Heller, 2005). Thus, anxious apprehension and anger-in might share characteristics that explain higher left than right hemisphere activity in specific brain regions. Anxious apprehension has been conceptualized as a future-oriented, verbally mediated cognitive process (Borkovec, 1994) thought to depend on the left hemisphere (e.g., Nitschke et al., 2000). Thus, both anxious apprehension and anger-in may foster activity in left-hemisphere language production areas (such as Broca's Area) reflective of verbal rehearsal (Borkovec, 1994; Borkovec & Inz, 1990; Engels et al., 2007; Freeston, Dugas, & Ladouceur, 1996) and / or sustained language reflection associated with Wernicke's area, in addition to increased phonological memory storage in left posterior brain regions (Gupta & MacWhinney, 1997).

Trait anger, regardless of motivational direction (i.e., collapsed across anger-in and anger-out), was associated with leftward frontal asymmetry, a finding consistent with previous research on anger and left frontal asymmetry (Harmon-Jones & Allen, 1998; Wacker et al., 2003). These results provide evidence that motivational direction (e.g., approach, withdrawal) need not be the driving force behind the relationship of anger and left frontal asymmetry. In the present study, trait anger results cannot be explained by group differences on the BAS, since the low anger group did not differ from the anger groups in BAS scores. BAS and BIS scores also did not moderate the relationship between trait anger and left frontal asymmetry in regression analyses.

In addition, trait anger asymmetry results cannot be explained by group differences in anhedonic depression. If the pattern were explained by depression scores, one would expect that the anger-in and combined groups would have had lower levels of depression, since previous research has shown reduced left frontal asymmetry in depression (e.g., Allen et al., 2004b; Gotlib et al., 1998). However, the anger-in and combined groups had the highest levels of depression. In fact, the low anger group displayed frontal asymmetry in favor of the right hemisphere when compared to the high trait anger groups, which might appear to be at odds with research showing greater left frontal asymmetry for control participants than for depressed participants. However, not all depression studies have consistently found leftward asymmetry in their control participants (e.g., Bell et al., 1998; Bruder et al., 1997; Kentgen et al., 2000; Nitschke et al., 1999; Reid et al., 1998). In addition, a recent study found that individuals low in aggression displayed frontal asymmetry in favor of the right hemisphere, consistent with present results (E. Verona, personal communication, March 6, 2007).

What could be driving this frontal asymmetry in favor of the left hemisphere for trait anger? One possibility is anger rumination. Rumination has been broadly conceptualized as the experience of repetitive, intrusive, and negative cognitions. Anger rumination has been

defined as unintentional and recurrent cognitive processes that emerge during and continue after an episode of anger experience that is related to the duration of anger experience and the tendency to dwell on one's anger experiences (Sukodolsky, Golub, & Cromwell, 2001). Research indicates that rumination increases angry feelings and maintains angry moods (Bushman, 2002; Rusting & Nolen-Hoeksema, 1998), and anger rumination is highly correlated with trait anger, anger-out, and anger-in (Sukodolsky et al., 2001). The limited research on neural mechanisms involved in rumination indicates that it, like anger, is associated with left frontal activity. Trait rumination predicts left frontal activity when individuals are instructed to look at negative rather than neutral pictures (Ray et al., 2005). In addition, in women rumination is associated with greater left frontal asymmetry than is distraction (Blackheart & Kline, 2005). Rumination is considered a maintenance factor of depression and is also common in anxiety (e.g. Nolen-Hoeksema, 2000). It is possible that rumination involving one type of negative affect could maintain other negative feelings. For example, research indicates that anger rumination is associated with depression (Gilbert, Cheung, Irons, & McEwan, 2005). More research is needed to address the moderating or mediating effects rumination might have for brain regions involved in the implementation of negative emotion and psychopathology.

The present study did not replicate a relationship between anger-out and frontal asymmetry in favor of the left hemisphere demonstrated in one study (Hewig et al., 2004), which found the strongest evidence for this association in analyses with a current source density (CSD) reference. CSD is a spatial high-pass filter that is relatively insensitive to deeper and more distributed sources. In the absence of consensus on the depth of sources contributing to frontal alpha asymmetry, it is unclear whether a high-pass filter provides more accurate information than a linked-mastoid reference, which would generally provide more sensitivity to deeper or more distributed activity. Hewig et al. (2004) performed analogous GLM hemispheric analyses with a CSD reference and a linked-earlobe reference similar to the linked-mastoid reference used in the present study. They did not find a strong relationship between anger-out and left frontal asymmetry with the linked-earlobe reference, although they did find a significant correlation between a leftward frontal asymmetry score and anger-out. Interestingly, in that study, the correlations between trait anger and left frontal asymmetry scores were very similar to those seen for anger-out but did not meet the .05 p-value. For example, a positive correlation of .16 emerged between trait anger and an AF4-AF3 asymmetry score (whereas for anger-out the correlation was .22), and another positive correlation of .16 emerged between trait anger and an Fp2-Fp1 asymmetry score (whereas for anger-out the correlation was .18). These results, although not significant, are consistent with findings of the present study, indicating that trait anger is associated with frontal asymmetry in favor of the left hemisphere.

These results support but in some ways narrow existing findings. In developing a taxonomy of anger and its neural implementation, present results argue, first, for distinctions among trait anger, anger-out, and anger-in styles. Although correlated, they have distinguishable relationships to measures of lateralized regional brain activity. Second, in considering frontal activity specifically, present data argue against a general assumption that anger is always associated with leftward asymmetry or approach motivation. This assumption appears to be true for trait anger but not for anger-out or anger-in. Finally, results support a distinction between anxious apprehension and anxious arousal, which showed distinct relationships with anger styles and frontal asymmetry.

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Figure 1. Group differences in alpha power at individual electrode sites (error bars indicate standard error). The x-axis denotes electrode position, and the y-axis denotes log alpha score, for which more negative values indicate more brain activity.

Table 1

Participant Demographics

Demographic	Anger-In (n=11)	Anger-Out (n=25)	Combined (n=9)	Low Anger (n=20)	Test	p
Age (years)						
M	18.82	18.60	18.89	18.55	F(3,61) = 0.50	.681
SD	.98	.91	.78	.69		
Gender ¹	4/7	13/12	7/2	11/9	χ^2 (3) = 3.47	.324
Ethnicity ²	1/0/3/6/1	1/1/3/20/0	0/0/1/8/0	3/0/1/16/0	χ^2 (12) = 12.88	.378

¹ Number of women / number of men.

² Number of each ethnic group: Asian / Latino(a) / African American / European American / Biracial.

Table 2

Group Values for STAXI-2 Subscales

Anger-In		Anger-Out		Combined		Low Anger		Effect Size			
M SD		M SD		M SD		M SD		η^2	Group Comparison		
<u>Screening session</u>											
Trait Anger		26.18	1.89	26.60	1.98	29.11	2.98	16.95	2.14	.84	F(3,61) = 102.56
Anger-Expression Out		15.09	3.11	23.12	1.83	22.33	2.55	14.50	1.91	.79	F(3,61) = 74.74
Anger-Expression In		25.45	2.84	15.48	2.71	24.78	2.39	15.15	1.66	.79	F(3,61) = 76.61
<u>First session</u>											
Trait Anger		23.09	6.66	25.40	4.56	26.44	4.50	17.45	2.65	.41	F(3,61) = 14.17
Anger-Expression Out		14.82	3.66	21.40	2.66	19.33	2.29	15.95	3.28	.47	F(3,61) = 18.23
Anger-Expression In		22.82	3.22	16.60	3.43	24.00	2.78	15.40	3.36	.53	F(3,61) = 23.25
<u>Second session</u> ^I											
Trait Anger		23.22	5.02	24.71	4.30	27.88	4.70	16.56	3.07	.52	F(3,52) = 19.00
Anger-Expression Out		15.89	3.41	20.57	3.47	21.38	1.06	14.39	2.81	.52	F(3,52) = 18.37
Anger-Expression In		22.00	5.24	16.90	4.06	23.00	2.45	15.50	3.47	.38	F(3,52) = 10.46

^INote. All p-values < .001. Nine of the 65 participants completing the first EEG session declined to participate in the second EEG session.

Table 3

Group Values for Questionnaire Scales

	Anger-In (n=9)	Anger-Out (n=21)	Combined (n=8)	Low Anger (n=18)	Group Comparison		
	M (SD)	M (SD)	M (SD)	M (SD)	F	p	η^2
MASQ-AA	25.50 (5.07)	24.90 (6.02)	27.44 (4.02)	22.17 (3.34)	2.52	.068	.13
MASQ-AD	19.89 (4.77)	17.67 (4.09)	19.50 (5.48)	14.47 (3.14)	4.76	.005	.22
PSWQ	58.83 (13.32)	43.60 (13.54)	59.44 (10.43)	43.33 (11.62)	6.17	.001	.26
PANAS PA	20.94 (6.66)	27.29 (6.07)	23.56 (4.72)	29.47 (5.60)	5.05	.004	.23
PANAS NA	14.94 (4.50)	15.69 (3.47)	14.13 (3.81)	13.64 (2.83)	1.19	.322	.06
BAS	41.06 (5.13)	43.05 (2.78)	43.63 (5.14)	41.64 (3.51)	1.08	.365	.06
BIS	23.78 (3.87)	19.19 (4.33)	19.75 (2.65)	18.44 (2.46)	4.90	.004	.22
BP Physical Aggression	21.50 (5.56)	24.17 (6.49)	20.69 (7.44)	17.56 (5.33)	3.77	.016	.18
BP Verbal Aggression	13.67 (1.79)	17.14 (3.91)	14.69 (3.53)	11.67 (3.15)	8.77	<.001	.34
BP Anger	17.11 (4.59)	19.90 (3.38)	19.63 (6.13)	11.89 (2.25)	16.29	<.001	.48
BP Hostility	21.44 (6.87)	21.07 (5.64)	22.56 (4.56)	15.64 (4.15)	5.12	.004	.23
STAXI ACI	19.78 (3.28)	17.95 (4.32)	16.50 (3.63)	22.69 (4.36)	6.07	.001	.26
STAXI ACO	22.61 (3.63)	18.69 (4.17)	16.44 (3.46)	24.58 (4.17)	5.12	.004	.39

Note. Numerator degrees of freedom were 3 and denominator degrees of freedom were 52. MASQ = Mood and Anxiety Symptom Questionnaire. AA = Anxious Arousal scale. AD = Anhedonic Depression scale. PANAS = Positive and Negative Affect Schedule. PA = Positive Affect. NA = Negative Affect. BAS = Behavioral Activation Scale. BIS = Behavioral Inhibition Scale. BP = The Buss and Perry Aggression Questionnaire. STAXI = State Trait Anger Expression Inventory. ACI = Anger Control-In scale. ACO = Anger Control-Out scale.

Table 4
Hierarchical Linear Regressions with Anger Comparisons Predicting EEG Asymmetry

DV		Full Model			Predictor		
		R ²	F	p	β	t	p
F4-F3	Group	0.16	3.14	.033			
	Anger-In vs. Anger-Out				0.16	1.20	.237
	High vs. Low Anger				0.39	2.95	.005
C4-C3	Group	0.13	2.61	.061			
	Anger-In vs. Anger-Out				0.31	2.28	.027
	High vs. Low Anger				0.21	1.54	.130
T6-T5	Group	0.21	4.56	.007			
	Anger-In vs. Anger-Out				0.41	3.18	.002
	High vs. Low Anger				0.28	2.20	.033

Note: DV = dependent variable. In the regression analysis for each asymmetry score, 3 dummy variables representing anger group comparisons were entered as predictors. Numerator degrees of freedom were 3, and denominator degrees of freedom were 51 or 52 (an outlier was removed from analyses of F4-F3 and C4-C3).

Table 5
Hierarchical Linear Regressions with Anxious Arousal Predicting EEG Asymmetry

DV	Full Model				Predictor		
	Predictor added 2 nd	R ²	F	p	ΔR^2	t	p
F4-F3	MASQ-AA	.23	3.71	.010	.07	-2.18	.034
C4-C3	MASQ-AA	.23	3.76	.009	.10	-2.53	.015
T4-T3	MASQ-AA	.09	1.27	.029	.08	-2.09	.041

Note: DV = dependent variable. MASQ-AA = Mood and Anxiety Symptom Questionnaire, Anxious Arousal subscale. In the regression analysis for each asymmetry score, group dummy scores and one questionnaire measure were entered in separate steps as predictors, constituting the full model. The unique contribution of each questionnaire was evaluated via the δR^2 obtained when it was added second in the hierarchical regression. Numerator degrees of freedom were 3 for group and 1 for the questionnaire. Denominator degrees of freedom were 51 for group and 50 for MASQ-Anxious Arousal.

Table 6
Hierarchical Linear Regressions with Interactions Predicting EEG Asymmetry

DV	Predictor added 3 rd	Full Model			Predictor		
		R ²	F	p	ΔR^2	t	p
C4-C3	(Anger-In vs. Anger-Out) x PSWQ	.23	2.05	.069	.10	1.82	.074
T6-T5	(High vs. Low Anger) x PSWQ	.31	3.14	.008	.08	2.13	.038

Note: DV = dependent variable; PSWQ = Penn State Worry Questionnaire. In the regression analysis for each asymmetry score, group was entered first, one questionnaire was entered second, and the interaction between group and the questionnaire was entered third. The unique contribution of the interactions was evaluated via the ΔR^2 and t values obtained. Numerator degrees of freedom were 1 and denominator degrees of freedom were 47 for the test of each interaction.