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Clarifying relations between dispositional aggression and brain potential response: Overlapping and distinct contributions of impulsivity and stress reactivity

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ABSTRACT

Impulsive-aggressive individuals exhibit deficits in amplitude of the P3 brain potential response, however, it remains unclear how separable dispositional traits account for this association. The current study sought to clarify the basis of this association by examining contributions of trait impulsiveness and stress reactivity to the observed relationship between dispositional aggression and amplitude of the P3 brain potential response in a visual novelty-oddball procedure. A significant negative association was found between aggressiveness and amplitude of P3 response to both target and novel stimuli over frontal–central scalp sites. Impulsivity showed a parallel inverse relationship with P3 amplitude, attributable to its overlap with dispositional aggression. In contrast, stress reactivity did not exhibit a zero-order association with P3 amplitude, but modestly predicted P3 in a *positive* direction after accounting for its overlap with aggression. Results are discussed in terms of their implications for individual difference variables and brain processes underlying impulsive-aggressive behavior.

1. Introduction

Research on the neurobiological bases of aggression has yielded evidence that individuals prone to aggressive behavior exhibit deficits in electrocortical responses such as P3 amplitude (e.g., Barratt et al., 1997). Theoretical perspectives on aggressive behavior (e.g., Davidson et al., 2000; Giancola, 1995; Moffitt, 1993) have emphasized the role of abnormalities in frontal brain systems governing affective and behavioral regulation—suggesting that reduced P3 response in aggressive individuals should be frontally based. Although some studies have reported this (e.g., Costa et al., 2000; Gerstle et al., 1998), findings as a whole have been inconsistent. A potential explanation is that aggressive behavior is complexly determined (Barratt et al., 1999; Berkowitz, 1990; Patrick and Zempolich, 1998), with reduced P3 amplitude at particular sites mediated by differing constituent dispositions. At a broad level, for example, angry/reactive aggression is reliably associated with P3 amplitude reduction, whereas instrumental/proactive aggression is not (Patrick, 2008). At a finer-grained level, individual differences in impulsiveness and distress proneness, which have been posited to contribute to angry/reactive aggression (Davidson et al., 2000), are viewed as distinctive trait dispositions (Tellegen & Waller, 2008) with differing neurobiological substrates (Gray, 1987). Based on these considerations, we hypothesized that traits of impulsivity and stress reactivity would play distinctive roles in the aggression-P3 amplitude association. Specifically, the present study utilized a task designed to examine the processing of target stimuli in addition to emotional and neutral novel stimuli in conjunction with independent measures of aggression and related trait dimensions of impulsivity and stress reactivity to evaluate contributions of these differing dispositional variables to P3 amplitude variations.

1.1. Aggressiveness and P3 response

Several studies have found that aggression – in particular, the type variously described as "impulsive," "reactive," or "angry" aggression – is associated with reduced amplitude of the P3 (P300) event-related potential (ERP) response.[1] The P3 is the predominant

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[1] The term P3 as used here refers to a family of ERP components including the P3 response to attended target stimuli in a frequent/infrequent "oddball" task (aka "P300," or "P3b"), and the P3 response to unexpected novel events (aka "novelty P3," or "P3a").

positive undulation of an ERP waveform time-locked to an attended stimulus, and tends to have maximal amplitude over parietal scalp recording sites. The best-known variant of the P3 is the oddball-target P3, evoked by infrequent, task-relevant events in a stimulus sequence. The term "P3b" is sometimes used for this frequency-sensitive variant, which is theorized to reflect later attentional and memory processing (Polich, 2007). This variant of the P3 has a posterior scalp distribution; its likely neural generators include temporal and parietal cortices (Dien et al., 2003; Polich, 2007).

Another variant of the P3 is the "novelty P3" (Courchesne et al., 1975) or "P3a" (Squires et al., 1975), which occurs in response to unexpected rare non-target stimuli in a sequence within three-stimulus or "novelty" oddball tasks. This variant of the P3 has been conceptualized as a neural indicator of attentional orienting (Courchesne et al., 1975; Polich, 2007; Squires et al., 1975). The scalp topography of the novelty P3 shifts from being maximal at posterior scalp sites to central and anterior sites as the target/novel discrimination becomes more difficult, presumably owing to increased attentional demands (Polich, 2007). Consistent with this, investigations of patients with focal lesions in frontal brain regions (Knight, 1984, 1997), data from dense-array ERP source localization studies (Dien et al., 2003), and functional magnetic resonance imaging studies (Yamaksi et al., 2002; Fichtenholtz et al., 2004) point to an important direct role of anterior brain regions (i.e., prefrontal and anterior cingulate cortices) in the allocation of attention to novel stimuli (Polich, 2007).

A number of published studies have compared target P3 amplitude in aggressive versus non-aggressive individuals. Barratt et al. (1997) examined differences in P3 amplitude between non-aggressive community controls and male inmates classified via interview as instrumentally or impulsively aggressive in a standard two-stimulus visual oddball task. Inmates classified as impulsively aggressive (and not those classified as instrumentally aggressive) evidenced reduced amplitude of P3 responses to target stimuli over central and parietal recording sites relative to the other two groups. Of interest from the standpoint of aggressiveness as a dispositional construct, Barratt et al. (1997) also found that P3 amplitude correlated negatively with self-report measures of anger and impulsivity, but the unique contribution of these dispositional measures in the prediction of reduced P3 amplitude was not evaluated. More recently, Bernat et al. (2007) reported that P3 amplitude reduction in a standard two-stimulus visual oddball task was related to frequency of violent offending in a male prisoner sample, whereas P3 amplitude was unrelated to frequency of non-violent offending. Similar findings have been obtained in non-incarcerated (student) samples. For example, Gerstle et al. (1998) found that self-reported impulsive aggression assessed via interview was negatively associated with anterior P3 amplitude for both rare and frequent stimuli (participants were instructed to silently count the rare tones) elicited during a two-stimulus auditory oddball task.

The novelty P3 has been investigated to a more limited degree as a brain response indicator of aggressive tendencies. One study of this type (Mathias and Stanford, 1999) reported P3 amplitude reductions in impulsively aggressive students for target stimuli over parietal recording sites and for novel stimuli over central and temporal scalp recording sites during a three-stimulus visual oddball task. One aim of the present study was to further investigate associations of dispositional aggression with novelty P3 response, along with target P3.

Taken together, these findings suggest that aggression is related to deficits in post-perceptual processing of task-relevant stimuli. This interpretation is broadly consistent with theory and research indicating an association between aggressive behavior and impairments in anterior brain function (Davidson et al., 2000; Morgan & Lilienfeld, 2000; Seo et al., 2008). In this regard, the three-stimulus or "novelty" oddball task procedure may be especially sensitive to brain deficits reflecting hypothesized anterior neural sources. The current study extended prior aggression-P3 research by using more ecologically-valid visual stimuli (affective and neutral pictures) as novel stimuli in place of non-emotional cues such as tones or geometric shapes. As described in the next section, the study sought to clarify the basis of the aggression-P3 relationship by evaluating associations of target and novelty P3 amplitude response with traits that have close conceptual and empirical ties to aggressive behavior—namely, impulsiveness and stress reactivity.

1.2. Aggression, personality, and P3 response amplitude

Recent years have seen rising interest in the utility of personality constructs in the prediction and explanation of aggressive and antisocial behavior. This trend has accompanied the increasing popularity and acceptance of structural models of personality. Research using the Five Factor Model (FFM) of personality, for example, has indicated that a personality profile consisting of low Agreeableness (reflecting a tendency to experience conflict with others) and low Conscientiousness (reflecting a lack of regard for order and control) is associated with antisocial behavior in community (Lynam et al., 2003; Miller et al., 2003) and psychiatric samples (Trull, 1992). The three-factor structural model of personality embodied in Tellegen's Multidimensional Personality Questionnaire (MPQ; Patrick et al., 2002; Tellegen, 1982; Tellegen & Waller, 2008) has also been examined in relation to aggressive tendencies. This model encompasses three higher-order dimensions of personality: Constraint, which reflects tendencies toward behavioral control versus disinhibition; Negative Emotionality, which reflects tendencies toward emotional distress, alienation from others, and hostility/aggression; and Positive Emotionality, reflecting tendencies to experience states of positive affect through interpersonal engagement (agency, affiliation). The three higher-order MPQ dimensions are indexed through scores on various lower-level ("primary") trait scales.

Of particular relevance to the investigation of aggressive tendencies are the lower-order MPQ trait scales Aggression, Stress Reaction, and Control (impulsivity reverse-keyed). The Aggression scale directly indexes proclivities toward hostility, belligerence, and angry retaliation. Scores on this scale are robustly associated with levels of self-reported delinquency in epidemiological samples (Krueger et al., 1994) and have been shown to prospectively predict violent offense behavior (Krueger et al., 2000) and persistence of antisocial deviance more broadly (Krueger, 1999b). The Stress Reaction scale assesses tendencies toward negative emotional reactivity in the face of provocation or uncertainty. Scores on this scale can be viewed as indexing the core affective-experiential component of Negative Emotionality (Tellegen & Waller, 2008). The Control scale taps tendencies to be planful versus spontaneous, cautious versus careless, and reflective as opposed to unreflective. Low scores on this scale index the impulsive, present-centered orientation of individuals low in Constraint.

From the perspective of aggression as unrestrained negative affect (e.g., Davidson et al., 2000; Krueger, 2002), dispositional aggression entails heightened levels of both negative emotional reactivity and impulsiveness. Consistent with this, factor analyses of the MPQ trait scales (Patrick et al., 2002; Tellegen & Waller, 2008) have demonstrated loadings of the Aggression scale on both the higher-order Negative Emotionality and Constraint factors. The loading of Aggression on Constraint, while lower (in an inverse direction; −.3 to −.4) than its loading on Negative Emotionality (∼.6), is nonetheless substantial enough to be meaningful. Notably, the broad construct of externalizing – of which impulsive aggression can be considered one facet (Krueger et al., 2007; Patrick &

Bernat, 2009) – is also associated jointly with these two broad personality factors (Krueger, 1999b, 2002).

These conceptual and empirical linkages point to affiliated traits of stress reactivity and impulsiveness as potentially important to an understanding of brain reactivity differences in aggressive-externalizing individuals. In this regard, some evidence exists to indicate that impulsive personality traits predict impairments in brain response known to be associated with externalizing. For example, Justus et al. (2001) reported negative associations between frontal oddball P3 amplitude and personality traits reflecting impulsivity and excitement seeking. Similarly, Moeller et al. (2004) reported that scores on a well-established measure of impulsivity (Barratt, 2000) predicted reduced P3 amplitude in a sample of cocaine-dependent participants. However, the question of whether elevations in impulsivity *per se* might account for reduced brain potential response in high aggressive individuals – or high externalizing individuals more broadly – has not been addressed to date and it remains unclear whether trait dimensions of impulsivity and aggression are independently associated with reduced P3.

With regard to stress reactivity and affiliated traits and disorders, evidence pertaining to amplitude of the P3 brain response is more mixed. Some studies have reported *enhanced* P3 amplitude in individuals scoring high in trait anxiety (e.g., Karch et al., 2007) and in patients diagnosed with "pure" cases of anxiety-related disorders (i.e., cases without comorbid psychiatric problems; Enoch et al., 2008). In addition, some studies have reported increased amplitude of ERP components other than P3 in anxiety disorder patients (e.g., Iwanami et al., 1997). In contrast, other work has yielded evidence of *reduced* P3 response in participants high in negative affectivity. For example, two studies by Gurrera et al. (2001, 2005) reported diminished auditory P3 amplitude in individuals scoring high on the FFM construct of neuroticism. Other studies have reported evidence of *reduced* P3 amplitude in individuals diagnosed with major depression (e.g., Blackwood et al., 1987; Karaaslan et al., 2003; Yanai et al., 1997). However, along with increased negative affectivity, depression also entails a distinct element of low positive affect (Clark & Watson, 1991) that could account for P3-related processing impairments. Indeed, there is evidence that P3 amplitude tends to normalize once active depressive symptoms remit, suggesting that P3 operates as an episode marker of depression rather than a dispositional indicator (e.g., Blackwood et al., 1987).

1.3. Current study aims and hypotheses

The major objective of the present study was to replicate and extend prior research demonstrating impaired brain potential response in aggressive individuals by examining P3 amplitude in relation to dispositional aggression and affiliated personality traits of impulsiveness and stress reactivity. To our knowledge, this is the first study to evaluate the distinctive contributions of aggressive vs. impulsive trait dispositions to reductions in P3 amplitude. Specifically, we assessed these dispositional variables using the MPQ in a primarily undergraduate sample, and evaluated their overlapping and distinctive contributions to prediction of P3 response to target and novel stimuli in a three-stimulus visual oddball task. The personality-based approach to the study of aggression is advantageous in that it focuses on carefully operationalized dimensional constructs rather than discrete behavioral variables (e.g., history of violent offending).

Based on the foregoing literature review, primary hypotheses for traits of aggression and impulsivity were as follows: (1) higher dispositional aggressiveness as indexed by scores on the MPQ Aggression scale would be associated with reduced amplitude of P3 response to oddball task stimuli; (2) the relationship between aggression and P3 response would be stronger at anterior (frontal and central) scalp sites than at posterior (parietal) sites, particularly in the case of novel task stimuli; (3) higher impulsiveness as indexed by the MPQ Control scale (reversed) would be associated with reduced amplitude of P3 response, and this association would overlap with (i.e., be accounted for at least in part by) the relationship with aggressiveness. Hypotheses for the trait of stress reactivity were somewhat more tentative. On one hand, as noted, there is some evidence that negative affective traits are associated with increased P3 amplitude. On the other hand, the tendency of stress reactivity to co-occur with aggression could offset positive relations between stress reactivity and P3 amplitude; suppressive effects of co-occurring aggression could in fact account for null relations between negative affective traits/disorders and P3 in some published reports. Based on these considerations, we tentatively hypothesized that: (1) stress reactivity (in contrast with traits of aggression and impulsivity) would show evidence of a positive association with P3 amplitude, and (2) this positive association would emerge most clearly after statistically controlling for the overlap of stress reactivity with aggression.

2. Methods

2.1. Participants

Eighty-two participants were recruited from psychology classes and through advertisements in the University of Minnesota Daily newspaper. Participants were screened to be free from hearing and visual impairments. Four participants were excluded due to invalid MPQ profiles, and two due to equipment failure, resulting in 76 participants for analysis (45 female; age range = 18–50, $M = 21.04$, $SD = 5.19$). Procedures for the study were evaluated for compliance with ethical guidelines by the University of Minnesota's Institutional Review Board (IRB), and all participants provided informed written consent prior to testing. Participants received course credit and/or $7.50 per hour for compensation.

2.2. Measurement of personality

Participants completed an abbreviated, 155-item version of the MPQ (MPQ-bf; Patrick et al., 2002), a questionnaire developed by Tellegen (1982) to assess a broad spectrum of personality traits in normal populations. As noted, the focus of the current study was on MPQ trait scales related to aggressive tendencies: Aggression, Impulsivity (reverse-keyed Control), and Stress Reaction. The Aggression scale measures tendencies toward vindictiveness, angry retaliation, and hurting others to achieve ends. High scorers are hostile, physically aggressive, and deliberately intimidating toward others. The Impulsivity scale indexes spontaneity and impulsive tendencies, in particular, a lack of planful control or calculating consequences in decision making. Extreme scorers tend to be reckless and careless do not like to plan activities, and prefer to "play things by ear." The Stress Reaction scale measures anxious tendencies and propensities toward states of negative affect. High scorers are easily upset and irritable, prone to worry, and have frequently changing moods. Substantial evidence exists for the validity of the Aggression, Impulsivity, and Stress Reaction traits as measured by the MPQ, including the fact that scores on these self-report scales show robust convergence with information provided by parental and peer raters (Harkness et al., 1995).

2.3. Experimental stimuli and task

The current study employed a 3-stimulus oddball paradigm. The task extended the rotated-heads oddball task designed by Begleiter et al. (1984), by adding affective photographic stimuli as the third (novel) stimulus category. Simple oval shapes served as the frequent non-target (standard) stimuli, which comprised 70% ($N = 168$) of the total stimuli presented. The target stimuli were schematic heads, each consisting of the same oval shape accompanied by a stylized nose and ear (15% of all stimuli presented, $N = 36$). For each target trial, the task was to press the left or right button on a button-box, with either the left or right hand respectively, to indicate whether the ear was on the left or right side of the head. For 18 of the targets (50% of target trials) the nose was pointed up. For the other 18 targets, the nose was pointed down, requiring that participants recognize, for example, that the ear was on the right side of the head even though it was on the left side of the screen, and vice versa. Participants were required to achieve 85% accuracy during a practice session (including only target and non-target stimuli) in order to begin the test session.

The novel stimuli were pleasant, neutral, and unpleasant pictures selected from the International Affective Picture System (IAPS; Lang et al., 2008). IAPS pictures, comprising 15% ($N = 36$) of the total stimulus set, were randomly interspersed throughout the task. Pleasant pictures included action (e.g. skydiving, river rafting) and erotic scenes (e.g. opposite-sex nude individuals and intimate couples). Unpleasant contents included scenes of victimization (e.g. acts of aggression, physi-

cal brutality, and combat) and scenes of threat (e.g. pointed guns, looming attackers). Neutral pictures consisted of scenes of inactive people, neutral human faces, household objects, and kitchen utensils. The specific pictures within each content category differed somewhat for men and women,[2] in order to match picture contents in terms of average normative ratings of valence and arousal across gender subgroups.[3]

2.4. Stimulus delivery and recording procedure

Stimuli were presented using an IBM-compatible computer running E-Prime (MEL Software, Inc). During the experiment, participants viewed the stimuli on a 21″ computer monitor while seated in a comfortable recliner, and made responses using a serial response box. The monitor was situated 1 m from participants' eyes, and the response box was positioned on their laps. All task stimuli (ovals, target heads, and novel pictures) were displayed within a rectangular frame, filled with dark gray, which appeared against a black background. The frame size was $5^\circ \times 6.67^\circ$ visual angle (height × width; 223 pixels × 297 pixels respectively on a 1024 × 768 bitmap image). Oval and head stimuli were displayed within this frame at a $3.75^\circ \times 3.50^\circ$ visual angle (height by width; 169 pixels × 152 pixels), while IAPS picture stimuli filled the frame. Stimuli were presented for 100 ms each, with a variable inter-trial interval of between 1.5 and 3.0 s. Picture presentation was counterbalanced across participants using 12 different stimulus orders, in which the presentation of oval, target head, and novel stimuli was randomized with constraints (i.e., no more than four oval stimuli appeared consecutively, no two target types appeared consecutively, and no two novels appeared consecutively). Data acquisition was coordinated by a second IBM-compatible computer with Neuroscan amplifiers and software (Neuroscan, Inc.).

2.5. Physiological measurement and data reduction

ERP activity was recorded from 51 scalp sites (AF3, AF4, C1, C2, C3, C4, C5, C6, CP3, CP4, CPZ, CZ, F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FCZ, FP1, FP2, FT7, FT8, FZ, O1, O2, OZ, P1, P2, P3, P4, P5, P6, P7, P8, PO3, PO4, PO5, PO6, POZ, PZ, T7, T8, TP7, TP8) using Neuroscan Quik-Caps with sintered Ag-AgCl electrodes. Electrodes were positioned above and below the left eye to monitor vertical electrooculogram (VEOG) activity. All electrode impedances were kept below 10K Ohms. EEG signals were digitized on-line at 1000 Hz during data collection with an analog band pass filter of .05–200 Hz. Data were referenced to electrode site Cz during on-line data collection, and off-line were arithmetically re-referenced the average of left and right mastoid electrodes for subsequent processing and analysis. Data epochs from −1000 ms to 2000 ms were extracted from the continuous EEG recordings using Neuroscan EDIT software (version 4.3, Neuroscan Inc.), and corrected for eye movements using the algorithm developed by Semlitsch et al. (1986), as implemented within the EDIT software. The epoched and eye-blink corrected EEG data were imported to Matlab (Mathworks, Inc.) for subsequent data processing. Data were resampled to 128 Hz using the Matlab resample command (which applies a low pass anti-aliasing filter before downsampling). Trials during which activity exceeded a range of ±100 μV, relative to a 500 ms baseline, were excluded from further processing. Data were averaged within each of the stimulus conditions (non-target ovals, target heads, novel pictures).

Using the grand average across participants to guide window selection, target P3 amplitude was scored as the maximum value between 295 and 656 ms post stimulus, whereas the shorter latency P3 for novel picture stimuli was defined as the maximum value between 273 and 602 ms. To facilitate presentation, results for representative midline electrode sites (Fz, Cz, Pz) are reported in the main analyses (see next section). However, as a supplement to this, topographic maps are presented depicting results across all available electrode locations.

2.6. Data analyses

Pearson correlations were computed to characterize relations among traits of Aggression, Impulsivity, and Stress Reaction, and to evaluate simple (zero-order) associations between each trait variable and P3 amplitude to target and novel stimuli. Zero-order correlations were supplemented with partial correlations to further elucidate the nature of interrelations among traits, and to clarify overlap among the three traits in terms of their associations with P3 amplitude. In addition, general linear model (GLM) analyses were conducted to evaluate effects of Stimulus Type (target, novel)[4] and Electrode Site (Fz, Cz, and Pz)[5] on P3 amplitude and relations of trait variables of interest with P3 amplitude (see Pedhazur, 1997, for details of statistical analyses that allow the inclusion of both categorical within-subjects and continuous between-subjects variables). All analyses incorporated P3 amplitude as a continuous dependent measure. GLM statistics were Greenhouse-Geiser adjusted and reported p values represent the adjusted statistic. Partial eta square (η^2) values are also reported as an index of effect size.

To avoid redundancy in presentation, findings from a two-way, Stimulus Type × Electrode Site repeated measures ANOVA are first presented to document main effects for these basic within-subjects variables as well as their interaction. This is followed by presentation of results from three-way GLMs, conducted separately for traits of Aggression, Impulsivity, and Stress Reaction, in which scores on the trait of interest were included as a continuous between-subjects factor along with Stimulus Type and Electrode Site as within-subjects factors. These GLMs provided for an evaluation of the relative magnitude of trait/P3 associations for target and novel stimuli at differing scalp locations. In addition, to more thoroughly characterize the scalp topography of trait/P3 associations for stimuli of each type (targets, novels), topographic maps are provided that depict correlations for each of the three traits of interest with P3 amplitude at all available scalp sites.

A further set of analyses was undertaken to evaluate the unique contribution of each trait to the prediction of P3 response amplitude, controlling for variance in common with the other traits. Specifically, for each of the MPQ trait variables (Aggression, Impulsivity, Stress Reaction), partial correlations were computed – for target and novel stimuli, separately – to assess the relationship between the *unique variance* in that trait variable and P3 amplitude after controlling for the influence of the other two trait variables. Partial correlation results for stimuli of each type (target, novel) are presented as topographic maps depicting statistical significance from analyses for each trait variable and P3 amplitude at all available scalp sites. This approach to controlling for overlap among variates is equivalent (in terms of resulting p values and interpretations) to a stepwise hierarchical regression approach in which the association for the variate of interest is examined in step 2, after entering variates to be controlled for in step 1.

Table 1
Correlations among Multidimensional Personality Questionnaire (MPQ) trait scales relevant to aggressive behavior.

Scale	Aggression	Stress Reaction
Aggression	–	
Stress Reaction	.37**	–
Impulsivity	.53**	.24*

Note: $N = 76$.
* $p < .05$.
** $p < .01$.

3. Results

3.1. Associations among traits of Aggression, Impulsivity, and Stress Reaction

Table 1 depicts correlations among target trait scales from the MPQ (Aggression, Impulsivity, and Stress Reaction). Consistent with the idea that these scales index distinct but interrelated con-

[2] The 36 novel pictures, listed by their IAPS identification numbers, were as follows: For females: erotic - 4572, 4656, 4660, 4670, 4677, 4687; action - 5629, 8043, 8161, 8180, 8300, 8400; threat - 1101, 1114, 1932, 6190, 6250, 6410; victim - 3022, 6211, 6530, 8480, 9230, 9250; neutral - 2190, 2393, 2570, 2890, 7000, 7010, 7020, 7036, 7100, 7130, 7175, 7500. For males: erotic - 2030, 4320, 4666, 4669, 4672, 4770; action - 5626, 5629, 8034, 8200, 8340, 8490; threat - 1525, 6250, 6300, 6370, 6510, 6930; victim - 6315, 6540, 8485, 9050, 9250, 9600; neutral - 2480, 2870, 2890, 5390, 7004, 7010, 7020, 7090, 7100, 7491, 7595, 9700.

[3] Mean valence and arousal normative ratings (Lang et al., 2008) for each picture content (across gender subgroups) were as follows: Erotic: valence (mean = 6.89, SD = 1.75); arousal (mean = 6.40, SD = 2.05). Action: valence (mean = 6.95, SD = 1.70); arousal (mean = 6.34, SD = 2.16). Threat: valence (mean = 3.08, SD = 1.79); arousal (mean = 6.37, SD = 2.25). Victim: valence (mean = 2.98, SD = 1.76); arousal (mean = 6.32, SD = 2.08). Neutral: valence (mean = 4.96, SD = 1.12); arousal (mean = 2.64, SD = 1.82).

[4] Supplemental analyses were conducted to examine effects for novel picture stimuli of differing valences (pleasant, neutral, unpleasant). Associations of traits of interest with P3 response were not found to differ as a function of stimulus valence, and thus for the sake of brevity and ease of interpretation, findings are reported for GLMs incorporating novel picture stimuli as a whole (i.e., averaged across valence types).

[5] Reported GLM analyses focused on sites Fz, Cz, and Pz because we were specifically interested in differential effects at anterior as compared to more posterior scalp sites. As a supplement to this, simple effects (correlational and regression) for all 51 scalp sites are depicted as interpolated topographical head maps in Fig. 2. In addition, we conducted supplemental GLM analyses that included P3 amplitude for lateral scalp sites (F3, F4, C3, C4, P3, and P4) along with midline sites (Fz, Cz, Pz). We found a significant Laterality (left, right, midline) effect such that P3 amplitude was larger at midline sites than at left or right sites, $F(2,74) = 8.18$, $p < .001$, $\eta^2 = .10$. However, this effect did not interact with any of the trait variables of interest, and thus the presentation focuses on results for the midline sites where P3 amplitude response was strongest.

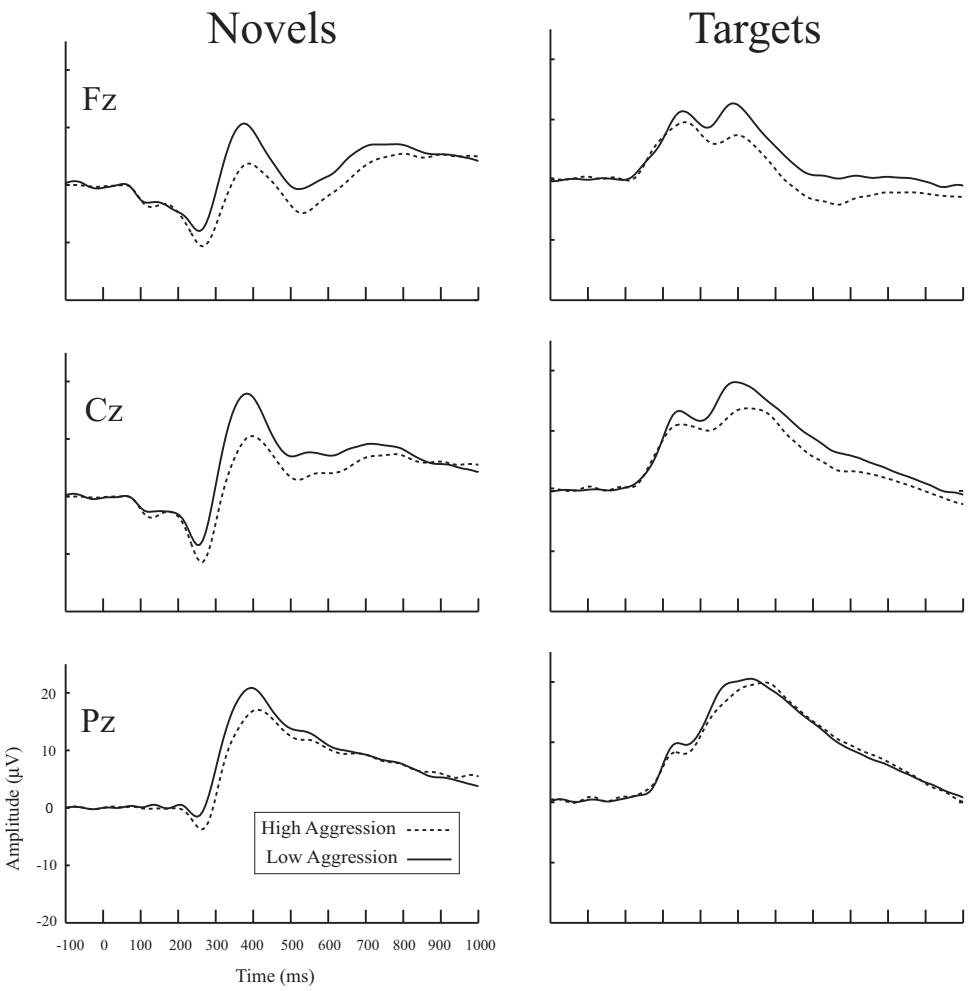

Fig. 1. Average waveform plots of ERP data obtained from novel and target stimuli at electrode sites Fz, Cz, and, Pz. The group split is presented for illustrative purposes to depict the direction of effects that are modeled statistically as continuous measures. Waveforms represent averaged ERP response from high and low scores (median split) on the brief Multidimensional Personality Questionnaire (MPQ-bf; Patrick et al., 2002) aggression scale.

structs, significant correlations of modest to moderate magnitude were observed among the three. Correlations between Aggression and the other two trait variables appeared more robust than the correlation between Impulsivity and Stress Reaction, and Fisher-*Z* comparisons confirmed that the *r* between Aggression and Impulsivity was significantly higher than the *r* between Stress Reaction and Impulsivity, $Z = 2.09$, $p < .05$. Moreover, the association between Impulsivity and Stress Reaction became negligible after controlling for scores on the Aggression scale (partial $r = .05$, $p = .66$). The implication is that the modest correlation between traits of Stress Reaction and Impulsivity was due entirely to their mutual association with the trait of Aggression.

3.2. Associations of Aggression and related traits with P3 response amplitude

In the initial two-way repeated measures ANOVA, there was no main effect found for Stimulus Type, $F(1,75) = 2.28$, $p = .135$, $\eta^2 = .03$, but the main effect of Electrode Site was found to be significant $F(2,148) = 3.92$, $p = .036$, $\eta^2 = .05$, reflecting greater P3 amplitude parietally (Pz scalp site) as compared to frontally (Fz scalp site). The Stimulus Type × Electrode Site interaction was significant, $F(2,150) = 3.84$, $p = .029$, $\eta^2 = .05$, reflecting generally larger P3 amplitude for target stimuli at the frontal site than for novel stimuli. P3 amplitude being maximal over posterior sites can be accounted for given the novel stimuli in the present study are perceptually quite different from targets, thus making target/novel discriminations relatively easy (Polich, 2007).

Several effects of interest emerged from the three-way GLMs in which MPQ trait scores (Aggression, Impulsivity, or Stress Reaction) were included as a continuous between-subjects factor (in separate analyses) along with Stimulus Type and Electrode Site as within-subject factors. A robust main effect of Aggression was found in the first GLM, $F(1,74) = 17.28$, $p < .001$, $\eta^2 = .19$, along with significant two-way interactions for Aggression × Stimulus Type, $F(1,74) = 5.27$, $p = .025$, $\eta^2 = .07$, and Aggression × Electrode Site, $F(2,148) = 8.21$, $p = .002$, $\eta^2 = .10$. The three-way Aggression × Stimulus Type × Electrode Site interaction was not significant, $F(2,148) = 0.47$, $p = .60$, $\eta^2 = .01$.[6] As a graphic illustration of the direction for the main effect of dispositional aggression on P3 amplitude, Fig. 1 presents waveform plots for participant subgroups consisting of individuals scoring above and below the median on the Aggression scale of the MPQ. To clarify the nature of the two-way interactions, P3 values were collapsed across either Stimulus Type or Electrode Site, and the correlation between Aggression scores and P3 amplitude was examined for Stimulus

[6] At the suggestion of an anonymous reviewer, we tested for a possible influence of gender on the Aggression/P3 relationship by including it as an additional factor in the analysis; no evidence of a Gender × Aggression interaction was found, $F(1, 72) = 0.82$, $p = .37$, indicating a similar relationship across men and women.

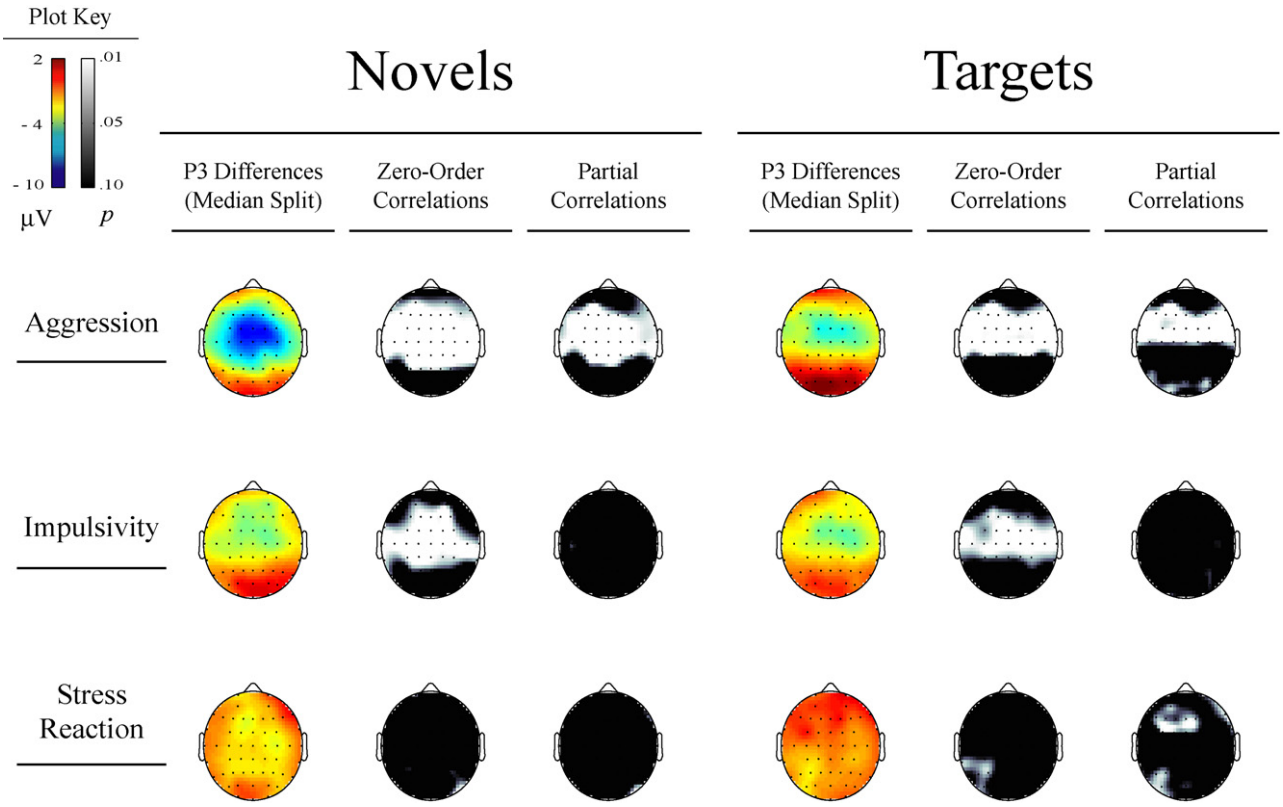

Fig. 2. Topographical maps depicting differences in P3 amplitude between high and low scores (above and below the median presented for illustrative purposes) on aggression, impulsivity, and stress reaction personality scales of the Multidimensional Personality Questionnaire (MPQ-bf; Patrick et al., 2002). Statistical effects (correlational *p* values) from continuous trait scores are presented. Zero-order results present Pearson's correlations between P3 amplitude across scalp sites and aggression, impulsivity, and stress reactivity traits. Partial correlation analyses examined the association between P3amplitude and the unique variance of each trait while controlling for the influence of the other two.

Type overall (collapsing across electrode sites) and for each electrode site (collapsing novel and target stimuli). For the Stimulus Type variable, collapsing across electrode sites, follow-up analyses revealed a stronger negative correlation between Aggression and P3 amplitude for novel stimuli ($r = -.47$, $p < .001$) as compared to target stimuli ($r = -.31$, $p = .006$). For the electrode site variable, collapsing across Stimulus Type, follow-up analyses revealed stronger negative relationships between Aggression and P3 amplitude at frontal ($r = -.48$, $p < .001$) and central ($r = -.49$, $p < .001$) sites than at the parietal site ($r = -.17$, $p = .12$).

In the second GLM, a significant main effect was found for Impulsivity, $F(1,74) = 9.13$, $p = .003$, $\eta^2 = .11$, with P3 amplitude diminished for individuals higher in impulsiveness. In this case, however, no interaction effects emerged as significant. In the third GLM, the main effect of Stress Reaction was found to be non-significant, $F(1,74) = 0.81$, $p = .37$, $\eta^2 = .01$, and no interaction effects emerged as significant. To facilitate comparison of effects for the three MPQ traits of interest, Fig. 2 presents results from zero-order correlations (Pearson's *r*) with P3 amplitude for each trait variable, by Stimulus Type (target, novel) across all electrode sites.

To provide a more complete depiction of results for each trait variable across the full array of available EEG recording sites, topographic maps are presented in Fig. 2 depicting the scalp distribution of P3 amplitude differences for subgroups consisting of participants above and below the median on each trait of interest. Median split group differences in P3 amplitude are presented for illustrative purposes while statistical maps depict simple (zero-order) correlations for each trait variable as continuous measures. Consistent with follow-up analyses reported above, Fig. 2 shows that Aggression-related reductions in P3 response to target and novel stimuli were maximal over anterior recording sites (*r*s for novel/target stimuli at site Fz = −.47/−.41 and at site Pz = −.27/−.05). Effects for the trait of Impulsivity were weaker for novel and target stimuli than those for Aggression and less focal in terms of scalp topography, consistent with the aforementioned absence of an Impulsivity × Electrode Site interaction (*r*s for novel/target stimuli at site Fz = −.32/−.30 and rs at site Pz = −.19/−.13).

Fig. 2 also depicts partial correlational results for novel and target stimulus types, reflecting the unique associations of each trait variable with P3 amplitude at differing scalp sites after controlling for the influence of the other two traits on P3. From these data, it clear that the unique variance associated with the trait of Aggression (i.e., that unrelated either to Impulsivity or to Stress Reaction) was predictive of reduced P3 amplitude over frontal–central scalp sites (partial *r*s for novel/target stimuli at Fz = −.38/−.37, Cz = −.43/−.28, and Pz = .06/−.18). In contrast, the unique variance in Impulsivity was not significantly associated with P3 amplitude at any scalp site. Thus, it appears that the aforenoted negative zero-order relationship between Impulsivity and P3 amplitude was attributable to variance shared between Impulsivity and Aggression. A further finding to emerge from the partial correlations pertained to the trait of Stress Reaction. Whereas Stress Reaction showed no association with P3 amplitude at the zero-order level, the partial correlation of Stress Reaction with P3 for target stimuli at frontal scalp sites was *positive* as opposed to negative (Fig. 2, bottom right). That is, after controlling for variance in common with traits of Aggression and Impulsivity (see Table 1), higher stress reactivity was associated with *increased* amplitude of P3 response (partial *r* at Fz = .27). To clarify this result further, supplementary partial correlations controlling for either Aggression or Impulsivity (rather than both) were computed. After controlling for overlap with Aggression alone, a significant positive

association between Stress Reaction and P3 emerged at electrode site Fz, $r = .26$, $p < .05$. In contrast, after controlling for Impulsivity alone, the correlation between Stress Reaction and P3 was not significant, $r = .15$, $p = .19$, indicating a suppressive effect of Aggression on the Stress Reaction/P3 association. Suppression is a case of mediation in which the magnitude of relationship between two variables increases when a third variable is included and controlled for in a statistical model (see MacKinnon et al., 2000). In the current instance, the trait of Aggression exerted a suppressive effect on the relationship between Stress Reaction and P3—rendering it null, when otherwise it would have been positive.[7]

4. Discussion

Previous research has consistently found evidence of an association between aggression and reduced amplitude of the P3 brain response. However, contrary to theory and research suggesting that aggression is primarily associated with frontal brain deficits, these reported effects have been inconsistent with regard to spatial topography—that is, P3 amplitude reductions have not consistently been specific to anterior scalp sites. Furthermore, prior research has not systematically accounted for the intersection of trait aggressiveness with the related traits of impulsivity and stress reactivity, both of which may contribute uniquely to the aggression-P3 relationship. Thus, in the present study, we sought to evaluate the distinct contributions of trait aggression – and the overlapping dimensions of impulsivity and stress reactivity – to reductions in P3 amplitude to stimuli in a task (the novelty oddball paradigm) designed specifically to engage anterior brain processes. To our knowledge, this is the first study to evaluate the distinctive contributions of aggressive vs. impulsive trait dispositions to reductions in P3 amplitude. In addition, the present study further extended previous research by utilizing affective cues (emotional pictures) as novel stimuli, in a modification to the standard three-stimulus oddball paradigm, thus adding incremental ecological validity to the experimental task.

We replicated previous findings indicating that aggressive individuals exhibit reductions in P3 amplitude to task-relevant target stimuli in the oddball task, and also found evidence for reduced P3 amplitude to task-irrelevant novel stimuli consisting of affective and neutral pictures. Also consistent with prior research, the present study found that trait impulsivity was related to diminished P3 amplitude; however, this effect was rendered non-significant when controlling for trait aggression. This study is the first to demonstrate that the effect of impulsivity on P3 amplitude is largely mediated by tendencies toward aggressiveness that commonly co-occur with impulsivity. Notably, the reduction in P3 amplitude that was associated uniquely with the trait of aggression (and to a lesser degree, variance in common with impulsivity) was especially pronounced over anterior (fronto-central) scalp sites. Additionally, the present study found that high levels of stress reactivity, while often characteristic of aggressive individuals, played no role in P3 amplitude reduction. Rather, the trait of stress reactivity evidenced a contrasting modest *positive* association with P3 response amplitude after controlling for its overlap with the trait of aggression. This enhancement of P3 response associated with the unique component of stress reactivity (i.e., variance unrelated to aggressiveness) was evident specifically for target stimuli, and was specific to frontal electrode sites.

4.1. Distinguishing effects related to stress reactivity and impulsivity from effects of aggression

Our finding of a moderate positive relationship between the variance unique to the trait of stress reactivity (controlling for aggression and impulsivity) and amplitude of the P3 response to target stimuli over anterior recording sites is notably *opposite* to the relationship evident for the trait of aggression. The specificity of this effect to target stimuli indicates enhanced allocation of resources to the primary instructional requirement of the task (i.e., to detect and discriminate "head" stimuli). Together, this pattern suggests greater directed attention toward task relevant stimuli among individuals with higher levels of dispositional stress reactivity (after controlling for affiliated aggressive and impulsive tendencies). However, this effect should be interpreted as tentative and warrants replication.

Whereas some prior studies have similarly reported enhanced P3 amplitude in individuals high in anxiety *per se* (e.g., Karch et al., 2007; Enoch et al., 2008), other work has yielded evidence of *reduced* P3 in subjects scoring high in dispositional neuroticism (Gurrera et al., 2001, 2005). These inconsistent results are likely attributable to the fact that the construct of neuroticism (also referred to as negative emotionality or negative affectivity; Tellegen & Waller, 2008) is broader than the construct of anxiousness—encompassing tendencies toward dysphoria, alienation, and anger/hostility in addition to anxiousness. In parallel with the current findings for P3 amplitude, there is evidence that that the amplitude of the error-related negativity (ERN) – a brain response that occurs following the commission of errors in a speeded performance task – is increased in individuals with negative-affect related disorders (i.e., anxiety or depression; Olvet & Hajcak, 2008). The implication is that mildly elevated levels of anxiety related neuroticism or negative affectivity may be associated with *enhancement* of basic brain responses such as P3 or ERN in laboratory tasks, whereas higher elevations of anxiety related neuroticism reflecting more severe psychopathology (e.g., significant elevations, and/or when comorbid with other psychiatric problems including depression and substance use disorders) is related to *reductions* of such brain responses (Enoch et al., 2008).

In contrast, higher levels of impulsivity in the current study were associated with reduced P3 for both novel and target stimuli, across scalp recording sites. Notably, reductions in ERN response have also been reported among individuals high in impulsivity or disinhibition (Hall et al., 2007; for a review see Olvet & Hajcak, 2008). However, supplementary correlational analyses revealed that this broadly-distributed effect was mostly accounted for by variance in impulsivity that overlapped with the trait of aggression. This finding can be interpreted in relation to the idea that a general disinhibitory ("externalizing") factor (Krueger, 1999a; Krueger et al., 2002, 2007) underlies the P3 amplitude reductions seen in varying types of impulse-related problems—including child and adult antisocial deviance along with problems involving alcohol and other drugs (Justus et al., 2001; Patrick et al., 2006). Prior work has shown that traits in the domains of negative affectivity and impulsiveness/low-constraint operate as indicators of this general externalizing factor (Krueger, 1999b; Sher & Trull, 1994); the trait of aggression, which lies at the intersection of negative affectivity and low constraint, emerges as particularly robust (e.g., Krueger, 1999b; Krueger et al., 1996, 2007). This may be because overt aggressive tendencies are indicative of greater underlying proneness to externalizing problems than tendencies toward spontaneity and lack of planfulness. That is, from the standpoint of item response theory (e.g., Muthen, 1996), aggressive tendencies may be discriminating at higher levels of the underlying liability dimension than impulsive tendencies *per se*.

[7] An alternative analytic approach to assess for suppressive effects is the inconsistent mediator test described by MacKinnon et al. (2000). Analysis of the current data using this approach yielded positive results, confirming our conclusion that Aggression operated to suppress an otherwise *positive* relationship between Stress Reaction and target P3.

From this standpoint, the finding that diminished P3 amplitude is more closely linked to dispositional aggression than to impulsivity *per se* suggests that P3 amplitude reduction may be indicative of more severe underlying externalizing vulnerability. Consistent with this idea, recent work has demonstrated a heritable basis to the relationship between diminished P3 amplitude and externalizing propensity as defined by levels of disorder symptoms (Hicks et al., 2007).

4.2. Dispositional aggression and diminished anterior brain response

In primary GLM analyses, the trait of aggression was associated with reduced P3 for both novel and target stimuli; this effect was notably stronger for novel stimuli, and at frontal and central scalp recording sites. Supplemental analyses indicated that, even after controlling for variance in common with traits of impulsivity and stress reaction, the effect of aggression on P3 amplitude remained significant over frontal–central sites. This pattern indicates a unique predictive association for aggression with respect to fronto-central brain processing of novel as well as target stimuli. Diminished P3 to stimuli of both types is indicative of deficits in attentional orienting to salient non-task-relevant stimuli along with deficits in post-perceptual processing of task-relevant stimuli. The localization of these effects to anterior scalp sites is consistent with dysfunction in frontal brain circuitry. In particular, the P3 response to novel stimuli has been demonstrated to reflect activity in anterior brain regions including prefrontal cortex (Knight, 1984) and anterior cingulate cortex (Dien et al., 2003). The current findings are thus consistent with prior work documenting impairments in anterior brain structure and function in aggressive individuals (e.g., Morgan & Lilienfeld, 2000; Raine, 2002).

What specific brain systems are dysfunctional in aggressive individuals? Davidson et al. (2000) reviewed various lines of evidence pointing to deficits in systems involved in regulating emotion, particularly negative affect, in impulsively violent individuals. Specifically, deficits in regions of the prefrontal cortex (orbital, ventromedial, dorsolateral) and the anterior cingulate cortex were highlighted by these authors. Neurochemically, there is evidence that anterior brain dysfunction in aggressive individuals involves disruptions in serotonergic function, particularly hypo-function in the ventral subdivision of the prefrontal cortex (Seo et al., 2008). In turn, dysfunctions at both levels (neurochemical, neurophysiological) are hypothesized to result from constitutional liability in conjunction with adverse environmental events that potentiate this liability (see, e.g., Caspi et al., 2002).

4.3. Limitations and future directions

Some limitations of the current study can be noted that highlight important directions for future research. One limitation was the study's exclusive focus on P3 response amplitude in an oddball task to index brain reactivity. Although the current study did extend prior work by examining relations for two variants of the P3 (responses to both target and novel stimuli), measurement of other components of brain response in addition to P3 amplitude across multiple tasks would permit stronger inferences to be drawn about processing deviations among individuals high in aggression and/or impulsiveness. As an example, measurement of ERN response (also known to be reduced in individuals with disinhibitory tendencies; see, e.g. Hall et al., 2007) would be informative because it reflects a more specific task-relevant process (performance monitoring) linked to a distinctive neural source (anterior cingulate cortex; for a review see Olvet & Hajcak, 2008).

Another limitation of the current study pertains to the approach used to partition sources of variance among individual difference characteristics and P3 amplitude. Specifically, partial correlations were used to evaluate contributions of non-overlapping variance among individual manifest (i.e., scale score) measures of each target trait. This approach could be extended in future research by collecting multiple measures of each trait construct and using structural equation analysis to evaluate the distinctive contributions to P3 amplitude of traits modeled as latent variables. This approach is advantageous in that it specifically accounts for measurement error and provides for more accurate estimation of relations among variables (in this case trait constructs) within the model (see Krueger et al., 2007 for an example of modeling externalizing tendencies). This approach could be extended further by incorporating additional measures of brain response along with P3 to permit modeling of anterior brain reactivity as a latent variable, along with trait constructs of interest.

As a supplement to statistical modeling of variance terms, it will also be valuable in future research to examine P3 and other affiliated brain measures in subgroups of participants selected for elevations on one or more specific trait constructs of interest. For example, participants high in both impulsivity and aggression could be compared to groups high in one or the other trait alone. This would provide another means of evaluating whether impulsiveness *per se* predicts reduced P3 response, independently of aggression—and if so, whether the parameters of this association (e.g., scalp topography; task/stimulus contexts in which reductions occur) differ from those for the trait of aggression.

One further limitation of the present study that warrants mention was its exclusive focus on relations between normative personality traits and brain response. Although substantial evidence exists that pathological tendencies lie on common continua with normative personality traits (Clark, 2005; Krueger et al., 2007; Mineka et al., 1998), permitting some extrapolation from the current findings to the domain of psychopathology, it will be valuable in future research to directly assess mental disorder symptoms along with affiliated personality traits in order to test assumptions about continuity. Related to this, the prevalence and severity of aggressive tendencies among participants in the current study was necessarily limited by the non-clinical nature of the sample. Recommended strategies for participant recruitment in future research include sampling to ensure adequate representation of participants scoring across the continuum of measurable aggressive traits tendencies (including those at the extremes), use of supplemental indices of aggressive behavior (e.g., reports or recorded instances of specific violent acts such as assault), and recruitment of participants from settings where severe aggressive deviancy is common (e.g., prisons; inpatient forensic facilities).

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