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Heritability of the neural response to emotional pictures: evidence from ERPs in an adult twin sample

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Affect-modulated event-related potentials (ERPs) are increasingly used to study psychopathology and individual differences in emotion processing. Many have suggested that variation in these neural responses reflects genetically mediated risk. However, to date, no studies have demonstrated genetic contributions to affect-modulated ERPs. The present study therefore sought to examine the heritability of a range of ERPs elicited during affective picture viewing. One hundred and thirty monozygotic and 124 dizygotic twin pairs passively viewed 30 pleasant, 30 neutral and 30 unpleasant images for 6 s each. The early posterior negativity was scored for each subject; in addition, the P300/late positive potential (LPP) was scored in multiple time windows and sites. Results indicate that the centro-parietal P300 (occurring between 300 and 600 ms) is subject to substantial genetic contributions. Furthermore, variance in the P300 elicited by affective stimuli was moderately heritable even after controlling for the P300 elicited by neutral stimuli. Later and more frontal activation (i.e. between 1000 and 3000 ms) also showed evidence of heritablity. Early parietal, and perhaps later frontal portions of the P300/LPP complex, may therefore represent promising neurobehavioral markers of genetically influenced processing of emotional information.

Keywords: heritability; emotion; neural response; twin studies

OVERVIEW

The ability to rapidly and accurately perceive motivationally salient cues in the environment is critical to survival. Detection of information pertaining to threat, food or reproduction (i.e. emotional stimuli) is therefore prioritized in the visual system: this content is detected more easily (Fox *et al.*, 2000; Öhman *et al.*, 2001) and more effectively captures and holds attention (Lang *et al.*, 1997; Armony and Dolan, 2002). As stimuli are registered as emotional, phasic physiological and behavioral responses (Gross and Thompson, 2007) are initiated that serve to mobilize the body's resources for action in service of survival (Frijda, 1986; Hajcak *et al.*, 2006; Lang and Bradley, 2010).

There are also marked individual differences in the amount of attentional resources that individuals allocate to affective stimuli (Koster *et al.*, 2005; Rottenberg *et al.*, 2005; Foti *et al.*, 2010; MacNamara and Hajcak, 2010), and the extent to which physiological resources are subsequently mobilized (e.g. Patrick, 1994; Grillon, 2002; McTeague *et al.*, 2011; Vaidyanathan *et al.*, 2011). Thus, emotional processing is a measurable characteristic with population-wide diversity that appears to be influenced by both environmental and genetic factors (Gabbay, 1992; Canli *et al.*, 2009; Coccaro *et al.*, 2011).

Despite enormous and growing interest in the molecular genetics of individual differences in emotion processing (e.g. McGuffin *et al.*, 1984; Kendler and Prescott, 1999; Hicks *et al.*, 2004), the complexity of these psychological constructs makes it difficult to directly link phenotypic characteristics to specific genes (Gottesman and Gould, 2003; Cannon and Keller, 2006). For this reason, identification of less complex intermediate neural phenotypes—which relate directly both to the underlying neurobiology and specific aspects of emotion processing—may prove useful in research directed at clarifying genetic determinants of normal and abnormal emotionality. Moreover, because perception of emotional stimuli entails an ongoing iterative series of processes that unfold over time and engage multiple neural systems (Gross and Thompson, 2007; Moratti *et al.*, 2011; Hajcak *et al.*, 2012, 2014), techniques with precise temporal resolution, capable of indexing this dynamic neural activity over time will also be crucial for investigating emotional processes that may be partially genetically determined.

Event-related potentials (ERPs), which directly reflect the response of underlying neuronal populations in real time, are well-suited for tracking rapidly changing brain activity. Moreover, emotion-modulated ERPs are emerging as useful neural metrics of both normative (Cuthbert *et al.*, 2000; Keil *et al.*, 2002; Bradley *et al.*, 2007; Codispoti *et al.*, 2009; Weinberg and Hajcak, 2011b; Hajcak *et al.*, 2012) and pathological emotional response (e.g. van de Laar *et al.*, 2004; Moser *et al.*, 2008; Mueller *et al.*, 2009; Foti *et al.*, 2010; MacNamara and Hajcak, 2010). Although evidence exists for genetic contributions to non-affective (i.e. cognitive) ERP components (e.g. Katsanis *et al.*, 1997; Polich and Bloom, 1999; Carlson and Iacono, 2006; Meyer *et al.*, 2012), there is little research examining the heritability of ERP components elicited by emotional stimuli (see, however, Anokhin *et al.*, 2010), and no articles examining the heritability of the emotional modulation of ERP components have appeared to date. If individual differences in neural correlates of emotion processing are determined in part by genes, then ERP components elicited by affective stimuli could represent neurocognitive mechanisms that mediate genetic influences on normal and pathological behavior (i.e. endophenotypes; Gottesman and Gould, 2003).

ERPS AND EMOTIONAL PROCESSING

Multiple ERP components are sensitive to the emotional content of stimuli (see Olofsson *et al.*, 2008 or Hajcak *et al.*, 2012 for reviews). However, there is increasing evidence that earlier and later components index distinct cognitive–affective processes (Olofsson *et al.*, 2008; Weinberg and Hajcak, 2010, 2011b; Wiens *et al.*, 2011; Hajcak *et al.*, 2012).

Comparatively early ERP components (e.g. those occurring up to 300 ms following stimulus onset) appear to reflect relatively obligatory engagement with emotional content (Olofsson *et al.*, 2008; Weinberg and Hajcak, 2011b; Hajcak *et al.*, 2012). For instance, the early posterior negativity (EPN), a temporo-occipital negativity maximal around 230 ms, is enhanced for emotional compared with

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neutral stimuli (Foti *et al.*, 2009; Schupp *et al.*, 2003b, 2006). Task parameters appear to have limited impact on the processing of emotional content reflected in this component: emotional modulation of the EPN is observed in passive viewing as well as more active tasks (Junghöfer *et al.*, 2001; Herbert *et al.*, 2008), and even when attention is directed away from affective stimuli or content (Kissler *et al.*, 2009; Schupp *et al.*, 2003a). Collectively, these results suggest that modulation of the EPN by emotional content reflects a relatively automatic process.

On the other hand, comparatively later ERP responses (e.g. those evident 300 ms or more after stimulus onset), such as the late positive potential (LPP), are thought to reflect more flexible and elaborated processing of stimulus content (Olofsson *et al.*, 2008; Weinberg and Hajcak, 2010, 2011b; Wiens *et al.*, 2011). The LPP, a positive-going slow wave that becomes evident at centro-parietal locations as early as 300 ms following stimulus onset, is enhanced for pleasant and unpleasant compared with neutral stimuli. This sustained positive wave is evident across the duration of picture presentation (Lang *et al.*, 1997; Cuthbert *et al.*, 2000; Junghöfer *et al.*, 2001; Foti and Hajcak, 2008) and appears to reflect multiple dynamic processes over time (Foti *et al.*, 2009; MacNamara *et al.*, 2009; Kujawa *et al.*, 2012; Weinberg and Hajcak, 2011b).

The initial portion of the LPP, occurring between 300 and 600 ms after stimulus onset, is morphologically and temporally similar to the target-elicited P300 observed in cognitive research that uses the oddball paradigm (Katsanis *et al.*, 1997; Carlson and Iacono, 2006; Polich, 2007, 2010; Olofsson *et al.*, 2008; Cano *et al.*, 2009). Consistent with the notion that emotional stimuli may be considered 'natural targets', the initial P300-like portion of the sustained LPP complex also appears sensitive to emotional content (Johnston *et al.*, 1986; Ferrari *et al.*, 2008; Foti *et al.*, 2009; Weinberg and Hajcak, 2011b). The marked similarities in topography and timing of this early component of the LPP suggest that it may reflect processes in common with the P300 observed in non-affective target detection tasks. We therefore use 'P300' to refer to this portion of the LPP.

Following the P300, a more sustained slow-wave component of the LPP is evident (Hajcak and Olvet, 2008). Although components that precede the LPP, like the EPN, are highly sensitive to the perceptual properties of stimuli (Wiens *et al.*, 2011), the slow-wave component is not, appearing to track stimulus content and meaning (i.e. emotionality) rather than stimulus complexity or size (De Cesarei and Codispoti, 2006; Wiens *et al.*, 2011). This sustained positivity exhibits a shift in scalp distribution over the course of affective picture processing, progressing from an early parietal to a more frontally maximal distribution (Foti *et al.*, 2009; MacNamara *et al.*, 2009). These data are in turn consistent with evidence suggesting that the LPP reflects coordinated activity of frontal-parietal attention networks (Sabatinelli *et al.*, 2007; Moratti *et al.*, 2011).

Others have suggested that affective modulation of the LPP is mediated by re-entrant processes from regions of the brain associated with the generation of emotions, like the amygdala, to the visual cortex (Lang and Bradley, 2010). Functionally, the LPP is thought to reflect processes associated with sustained engagement and continued elaboration of affective content (Dolcos and Cabeza, 2002;Azizian and Polich, 2007; Foti and Hajcak, 2008; Olofsson *et al.*, 2008; Dunning and Hajcak, 2009; MacNamara *et al.*, 2009; Weinberg and Hajcak, 2011b; Weinberg *et al.*, 2012). Additionally, this latter portion of the LPP appears uniquely related to memory for emotional pictures (Dolcos and Cabeza, 2002; Azizian and Polich, 2007), suggesting that increased emotional processing reflected in the LPP slow-wave component may also relate to encoding processes.

GENETIC INFLUENCE ON EMOTION-ELICITED ERP COMPONENTS

Twin and family studies have identified substantial genetic contributions to the P300 elicited by non-affective targets (Eischen and Polich, 1994; Katsanis *et al.*, 1997; Polich and Bloom, 1999; Iacono *et al.*, 2003; Hall *et al.*, 2006). To date, there has been only one published study demonstrating heritability of the P300 in response to affective stimuli (Anokhin *et al.*, 2010). However, this study used facial stimuli, which are relatively less emotionally evocative than complex emotional scenes (Britton *et al.*, 2006), and focused on genetic contributions to the overall amplitude of P300 response, rather than on genetic contributions to the 'emotional modulation' of the P300 (i.e. heritability of the 'increase' in P300 for affective compared with neutral faces).

Additionally, though molecular genetic studies have provided evidence for specific polymorphisms that may contribute to ERPs related to emotion processing (e.g.; de Rover *et al.*, 2012), they explain only a small portion of the observed variance and are not informative regarding the relative contributions of genes and environment to neural phenotypes. In contrast, the use of a sample with varying degrees of genetic relatedness [i.e. monozygotic (MZ) and dizygotic (DZ) twins] provides a way to effectively estimate the relative influence of differing etiological sources on a phenotype of interest. More specifically, using a twin design, variance in a phenotype can be decomposed into multiple sources: additive genetic effects (A); non-additive (dominant) genetic effects (D); shared environmental effects (i.e. the extent to which twins appear similar because of similar experiences in the environment, (C); and non-shared environmental effects (i.e. the extent to which twins appear different as a result of contrasting environmental experiences) along with measurement error (E). Operating from this perspective, the present study used data from a passive picture-viewing task administered to a sample of adult twins to clarify sources of etiologic influence underlying differing components of the ERP response to affective stimuli.

METHOD

Participants

Participants consisted of 510 (258 female, 252 male) twins (260 MZ, 248 DZ) recruited from the University of Minnesota Twin Registry and screened for hearing and visual impairments before testing. Thirty-one participants were excluded from current analyses owing to excessive artifact in the electroencephalogram (EEG) recordings ($n = 16$), discontinuation of participation ($n = 6$) or technical problems with the EEG collection ($n = 9$), resulting in 479 participants in the final analyses (244 MZ, 235 DZ; 242 males, 237 females). The mean age of study participants was 29.39 years (s.d. = 4.84; range = 22–38) and the racial composition was as follows: Caucasian, 96.5%; African American, 0.6%; Hispanic, 0.4%; Native American, 0.8%; mixed race, 0.8%; other/missing, 1.3%. Procedures for the study were approved by the University of Minnesota's institutional review board, and all participants provided informed written consent before testing. Data for the current report were collected as part of a larger protocol for which participants received $100 as compensation along with reimbursement for travel expenses.

Experimental stimuli and design

Each participant viewed a series of 90 pictures consisting of 30 pleasant, 30 neutral and 30 unpleasant scenes from the International Affective Picture System (IAPS; Lang *et al.*, 2008; Appendix A). Each picture was presented for 6 s followed by an intertrial interval of 12 s. Pleasant pictures included action, erotic and nurturant scenes. Unpleasant contents included scenes of physical injury and direct threat scenes. Neutral pictures consisted of scenes of inactive people, neutral human faces, household objects and kitchen utensils. The specific

pictures within the pleasant valence category differed somewhat for men and women (i.e. three opposite-sex nude scenes differed), to match picture contents for average normative ratings of valence and arousal across genders (Appendix A).

The 90 pictures were presented in eight different orders across participants of each gender. Within and between orders, pictures were counterbalanced such that valence categories (pleasant, neutral, unpleasant) were represented equally across orders at each serial position and no more than two pictures of a particular valence occurred consecutively within any stimulus order. For purposes of examining another effect of interest in the picture-viewing task, affect-modulated startle (cf. Lang *et al.*, 1997), noise probes (50 ms, 105 dB) were presented during most picture trials (81 of 90). However, because probes occurred at 3 s or later after picture onset, ERP responses within the 3 s time window of current analyses were unaffected by the probes.

Physiological data recording and reduction procedures

During the experiment, participants viewed the picture stimuli on a 21″ computer monitor situated ∼1 m away, at eye level, while seated in a comfortable recliner. Data collection was performed using two PC computers, one equipped with E-Prime software (MEL software, Inc) for stimulus delivery and the other with Neuroscan Acquire software for physiological data acquisition. ERP activity was recorded from 53 scalp sites positioned according to the 10–20 system (AF3,AF4, AFZ, C1, C2, C3, C4, C5, C6, CP1, CP2, CP3, CP4, CPZ, CZ, F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4,FCZ,FP1,FP2, FPZ, FT7, FT8, FZ, O1, O2, OZ, P1, P2, P3, P4, P5, P6, P7, P8, PO3, PO4, POZ, PZ, T7, T8, TP7 and TP8) using Neuroscan Quik-Caps with sintered Ag-AgCl electrodes. Electrodes were positioned above and below the left eye to monitor vertical electrooculographic activity and adjacent to the outer canthi of the left and right eyes to monitor horizontal electro-oculographic activity. All electrode impedances were kept below 10 kOhms. EEG signal activity was recorded using electrode site Cz as the online reference and applying an analog band pass filter of 0.05–200 Hz before digitization at 1000 Hz. The raw EEG data were rereferenced offline either to the average of the left and right mastoids (LPP) or to an overall electrode average (EPN; see below) and low-pass filtered with a cutoff of 30 Hz. Eye-blink and ocular-movement corrections were performed according to the procedures specified by Gratton *et al.* (1983).

Subsequent to these steps, a partially automated protocol was used to identify artifactual data for rejection. Data for individual channels were flagged for rejection if a voltage deflection of >50.0 μV occurred between sample points, if a deflection >300.0 μV occurred within a segment or if a voltage difference of <0.50 μV was evident within 100 consecutive milliseconds. Data for the remaining trials were inspected visually to detect any other artifacts warranting rejection.

EEG activity was examined across an interval extending from 200 ms before picture onset up to 3000 ms afterward. ERP waveforms were constructed by separately averaging pleasant, neutral and unpleasant picture trials. Each ERP average was baseline-corrected relative to activity in the 200 ms pre-stimulus window.

In line with extensive prior work, the LPP response was scored using the average-mastoid reference (e.g. Ito *et al.*, 1998; Cuthbert *et al.*, 2000; Schupp *et al.*, 2000; Weinberg and Hajcak, 2010). Because existing research (Foti *et al.*, 2009; Weinberg and Hajcak, 2011b; Weinberg *et al.*, 2012) has demonstrated that important information about the time course of emotional responding may be reflected in differences between early and later portions of the LPP, the LPP was scored as the average activity in four successive time windows following stimulus onset: 300–600 ms (i.e. the P300), 600–1000 ms, 1000–2000 ms and 2000–3000 ms. In addition, there is evidence that the LPP shifts in distribution from a relatively focal centro-parietal distribution early after stimulus presentation to a more diffuse and frontal distribution later in time (Hajcak *et al.*, 2010; Hajcak and Olvet, 2008; Olofsson *et al.*, 2008). Each time window of the P300/LPP complex was therefore scored at both Pz and Fz to capture this shifting distribution over time (Cuthbert *et al.*, 2000; Keil *et al.*, 2002; Foti and Hajcak, 2008; Foti *et al.*, 2009).

In contrast, most studies examining the EPN have used the average reference (e.g. Schupp *et al.*, 2003a, 2004; Wieser *et al.*, 2006). Affective modulation of this component is found to be maximal at occipital sites, and use of an average mastoid reference results in a substantial decrement in the EPN (Hajcak *et al.*, 2012). Accordingly, to effectively capture activity associated with the EPN and permit straightforward comparisons with prior published work, data were rereferenced to the average of all electrodes for the purposes of scoring the EPN. The EPN was scored from the resultant ERP waveform as the average activity at electrode site Oz, between 175 and 275 ms following picture onset (Schupp *et al.*, 2006; Foti *et al.*, 2009; Weinberg and Hajcak, 2011b; Hajcak *et al.*, 2012).

Statistical analyses

Analyses were conducted using SPSS General Linear Model software, version 17.0, and Mplus version 6.1. Separate one-way repeated-measures analyses of variance (ANOVAs) were conducted to examine the effects of picture valence (i.e. pleasant, neutral, unpleasant) on the EPN at Oz and the LPP at Pz and Fz for each of the four time windows. To account for the non-independence of twin scores, Twin ID was included as a clustering variable in our mixed-model ANOVA. This option adjusts standard errors within each group to account for groupwise correlation. Greenhouse-Geisser correction was applied to *p* values associated with multiple-df repeated-measures comparisons in instances involving violation of the assumption of sphericity. Following each ANOVA, two planned contrasts consisting of paired-samples *t*-tests were conducted to assess affective modulation effects, comparing unpleasant and pleasant against neutral. To examine emotional modulation of ERPs, we quantified modulation scores as residual values saved out from regression models in which the ERP for neutral pictures was used to predict the ERP for an affective picture average (i.e. residual scores reflected variance in the affective condition not accounted for by the neutral condition).

Residuals were used as a measure of variance unique to emotional processing rather than difference scores (DuBois, 1957; Cronbach and Furby, 1970). Subtraction methods relying on averages are somewhat ineffective in isolating variance unique to emotional images in that the resulting difference score remains correlated with both initial values (i.e. the average response to neutral and average response to emotional images); thus, the difference cannot be said to uniquely reflect activity associated with emotional processing. Residuals are also a difference score of sorts, but reflect the difference between an individual's observed average response to emotional images and the average that would be predicted from their neutral average; these residuals will be independent from the average response to neutral images, but correlated with the average response to emotional images. Residuals thus more successfully capture unique variance associated with emotional responding. Finally, forthcoming data from our labs suggest that residuals are a more reliable measure than difference scores, which are often unreliable (DuBois, 1957; Traub, 1967; Cronbach and Furby, 1970).

For each ERP variable of interest, twin correlations were examined for response magnitude scores and affective modulation effects, within MZ and DZ subsamples separately. Cross-twin correlations of

magnitude scores for each picture valence condition (i.e. pleasant, neutral, unpleasant) were examined. In addition, correlations were examined for the affective modulation scores for each emotional condition (i.e. variance specific to pleasant and unpleasant). Correlations were examined in terms of the intraclass correlation (ICC), as an index of score agreement. In twin samples, because each pair is measured in the same units (as opposed to, for example, measuring height and weight in a single individual), ICC uses a pooled scaling of covariance and is a more natural measure of agreement; moreover, while Pearson's *r* generates estimates of the consistency of scores, it does not capture absolute agreement (i.e. it is possible to have a high Pearson's *r* even if one variable is offset from the other by a constant value). ICC reflects the absolute agreement between variable values. For overall magnitude and condition effects associated with each ERP variable, one-tailed Z-tests for dependent correlations (Steiger, 1980) were used to test whether the ICC for MZ twins significantly exceeded the corresponding ICC for DZ twin.

On the basis of comparisons of the ICCs, suggesting a potential contribution of genetic influences to several components, we fit univariate biometric models to the raw data using Mplus software (version 6.1; Muthén and Muthén, 2010; Prescott, 2004). For each ERP variable and each condition, we fit either an ACE model (in cases where $r\text{MZ} < 2r\text{DZ}$) or an ADE model (where $r\text{MZ} > 2r\text{DZ}$) to the data. Then, to determine whether the A, C or D paths contributed significantly, we compared the goodness of fit for alternative AE, CE and E models with that of the ACE or ADE model using the χ^2 statistic, and Akaike's information criterion (AIC; computed as 2*logLikelihood + 2 *r*, where *r* designates the number of free model parameters), which considers model parsimony along with goodness-of-fit. Lower values of χ^2 and AIC were interpreted as indicative of better fit. In each instance, the most parsimonious model (i.e. model with the fewest parameters) was selected, provided that dropping a path did not significantly reduce fit (i.e. significantly increase χ^2). Further, in instances where $\text{rMZ} < 2r\text{DZ}$, suggesting potential shared environmental influences, we compared the goodness-of-fit between AE and CE models. If the fit of the selected model did not exceed that of a saturated model (i.e. model containing as many parameters as data values), as indicated by a non-significant χ^2 value, path coefficients for the model were estimated using the maximum likelihood method.

RESULTS

In this section, emotion modulation effects are first presented for each of the above-mentioned ERPs in the sample as a whole. Following this, cross-twin correlations for MZ and DZ twins, and estimates of the relative contributions of genetic and environmental influences based on these correlations, are presented for each ERP variable, as well as for residual scores reflecting variance in amplitude for pleasant and unpleasant picture conditions after controlling for the neutral condition.

Overall sample: modulatory effects of picture valence on EPN and LPP response

Table 1 presents average values by valence condition for the EPN and for each LPP score in the sample as a whole, and for MZ and DZ twin subsamples separately. Figures 1 and 2 present topographic maps for the EPN and LPP across all participants, depicting voltage differences (in microvolts) across the scalp for pleasant minus neutral pictures, and for unpleasant minus neutral pictures. Grand average stimulus-locked ERP waveforms for the EPN (at scalp site Oz) and the LPP (at sites Pz and Fz) are also presented in Figures 1 and 2, respectively. Analysis of variance results, highlighting emotion–modulation effects for each ERP variable, are also presented in Table 1.

Confirming patterns evident in Table 1 and Figures 1 and 2, scores for each ERP variable differed significantly by picture type, with both pleasant and unpleasant scenes eliciting larger amplitude responses than neutral scenes. Of note, the P300 scored at scalp site Pz between 300 and 600 ms, and the LPP between 600 and 1000 ms, displayed the most robust effects of emotion (pleasant/unpleasant > neutral). In later time windows, emotional modulation was diminished though not absent, at both Pz and Fz.

Biometric model fit

Biometric models were evaluated for each ERP component variable, in each time window, for each electrode site and valence condition. As noted earlier, goodness of fit was evaluated by comparing ACE (or ADE) models with AE, CE and E submodels, testing for a significant reduction in model fit after first dropping the C or D parameter, and then the A parameter (see Supplementary Material for tables containing goodness-of-fit statistics for each of the submodels in each time window). Parameters for best-fitting biometric models (i.e. estimated contributions of A, C, D, E, as appropriate) are presented in the right-most columns of Tables 2 and 3 for cases in which model fit did not exceed that of the saturated model, as evidenced by a non-significant χ^2. (Fit statistics for the alternative models tested are presented in Supplementary Table S1). As indicated by the presentation of model parameter estimates in Table 2, an AE model best accounted for the data in most cases where the fit of the saturated model was exceeded (i.e. instances in which dropping the C or D parameter resulted in a significant decrement in fit). There were a few exceptions in which a CE model fit the data best (Table 2). Additionally, as indicated by the presentation of estimates in Table 3, biometric models were less able to adequately account for residual scores; in those instances where the fit of the saturated model was exceeded, the AE model again generally exhibited the best fit, with one exception where a CE model exhibited the best fit.

MZ/DZ twin correlations and heritability estimates

ERP response magnitude

Table 2 displays ICC coefficients for MZ and DZ twins, respectively, as well as results of Z-score tests of differences between the two and ACE/ADE estimates for each ERP variable. Though MZ as compared with DZ twins tended to show higher correlations of electrocortical response across the time course of affective picture processing (beginning in the time window of the EPN), this effect was most pronounced—and consistently significant across all picture types—for the P300 during the 300–600 ms time window at scalp site Pz. Heritability estimates for the P300 during this time window ranged from 0.45 for the P300 elicited by neutral pictures to 0.55 for the P300 elicited by pleasant and unpleasant pictures. The corresponding heritability results in this time window at Fz were weaker, with the MZ/DZ difference achieving significance only for unpleasant pictures. The LPP to pleasant and neutral pictures during the 600–1000 ms window also appeared heritable at scalp site Pz, as did later, frontal (Fz) modulation of the LPP by unpleasant pictures (between 1000 and 3000 ms).

Affective modulation of ERPs

Table 3 shows that, after controlling for responsiveness to neutral pictures, the P300 elicited by unpleasant pictures between 300 and 600 ms was heritable at both Pz and Fz. The corresponding modulatory effect for pleasant scenes fell just short of significance. Additionally, residual variance in the LPP for unpleasant scenes during the 1000–2000 ms and 2000–3000 ms windows (i.e. after controlling for the corresponding LPP elicited by neutral pictures) also showed

Table 1 Means (in μV) and standard deviations for ERP variables for all subjects (left columns) and for MZ and DZ twins separately (middle and right columns)

ERP variable	All subjects		MZ		DZ		Main effect of emotion F (2, 910)	η_p^2	Pleasant *vs* neutral t(455)	Unpleasant *vs* neutral t(455)
	M	*SD*	*M*	*SD*	*M*	*SD*				
EPN (175–275 ms) Oz							195.66	0.30	19.84	12.81
Pleasant	1.55	4.77	1.62	4.73	1.48	4.82				
Neutral	3.55	4.65	3.57	4.59	3.53	4.71				
Unpleasant	2.21	4.90	2.29	4.83	2.13	4.98				
P300 (300–600 ms) Pz							697.43	0.65	33.68	33.86
Pleasant	10.18	5.62	10.37	5.57	9.99	5.68				
Neutral	4.60	4.91	4.59	4.90	4.61	4.93				
Unpleasant	10.98	5.83	11.00	5.94	10.96	5.72				
P300 (300–600 ms) Fz							369.64	0.44	24.17	22.60
Pleasant	2.71	7.29	2.99	7.43	2.41	7.15				
Neutral	−2.69	6.51	−2.65	6.73	−2.72	6.29				
Unpleasant	2.24	7.17	2.12	7.22	2.37	7.13				
LPP (600–1000 ms) Pz							683.48	0.66	29.77	35.64
Pleasant	7.36	4.74	7.37	4.54	7.36	4.95				
Neutral	2.01	4.01	1.92	4.19	2.11	3.81				
Unpleasant	9.59	5.32	9.54	5.05	9.64	5.60				
LPP (600–1000 ms) Fz							291.03	0.40	19.67	22.42
Pleasant	5.56	6.84	5.83	6.57	5.27	7.12				
Neutral	0.43	6.04	0.32	6.26	0.54	5.81				
Unpleasant	6.41	6.98	6.46	6.88	6.35	7.10				
LPP (1000–2000 ms) Pz							199.78	0.33	14.38	19.30
Pleasant	3.24	4.18	3.36	4.06	3.11	4.30				
Neutral	0.78	3.38	0.71	3.31	0.86	3.46				
Unpleasant	4.33	4.43	4.37	4.14	4.29	4.71				
LPP (1000–2000 ms) Fz							74.82	0.14	9.05	11.72
Pleasant	5.07	5.75	5.17	5.65	4.96	5.87				
Neutral	2.69	5.04	2.49	5.10	2.90	4.98				
Unpleasant	5.65	5.84	5.58	5.54	5.72	6.16				
LPP (2000–3000 ms) Pz							58.33	0.12	8.85	9.69
Pleasant	1.87	3.89	2.00	3.89	1.73	3.90				
Neutral	0.24	3.34	0.21	3.41	0.27	3.26				
Unpleasant	2.12	4.13	2.07	3.93	2.18	4.33				
LPP (2000–3000 ms) Fz							16.31	0.03	5.38	4.04
Pleasant	4.71	5.19	4.90	5.38	4.50	4.99				
Neutral	3.25	4.65	3.11	4.95	3.41	4.31				
Unpleasant	4.26	5.39	4.20	5.08	4.32	5.70				

Note: $P < 0.001$ for all comparisons. Also shown are analysis of variance results for effects of emotion on each ERP variable, at relevant scalp sites.

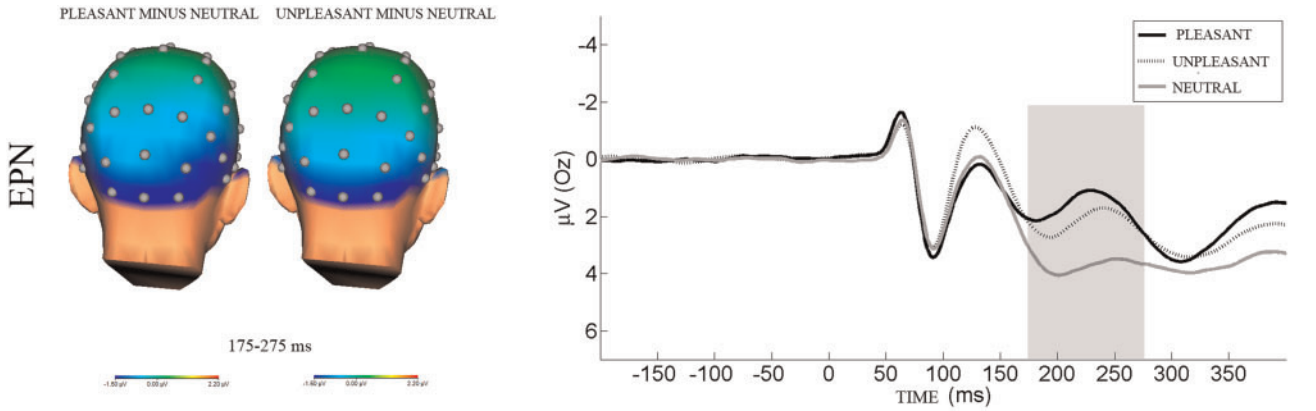

Fig. 1 Topographic maps for the time window of the EPN, depicting differences (in μV) in the sample as a whole for pleasant minus neutral (right) and unpleasant minus neutral (left) pictures. Also depicted are stimulus-locked ERP waveforms reflecting the average activity at Oz, referenced to an average of all electrodes. Stimulus onset is at time 0 and negative is plotted up; the time window of the EPN is highlighted in gray.

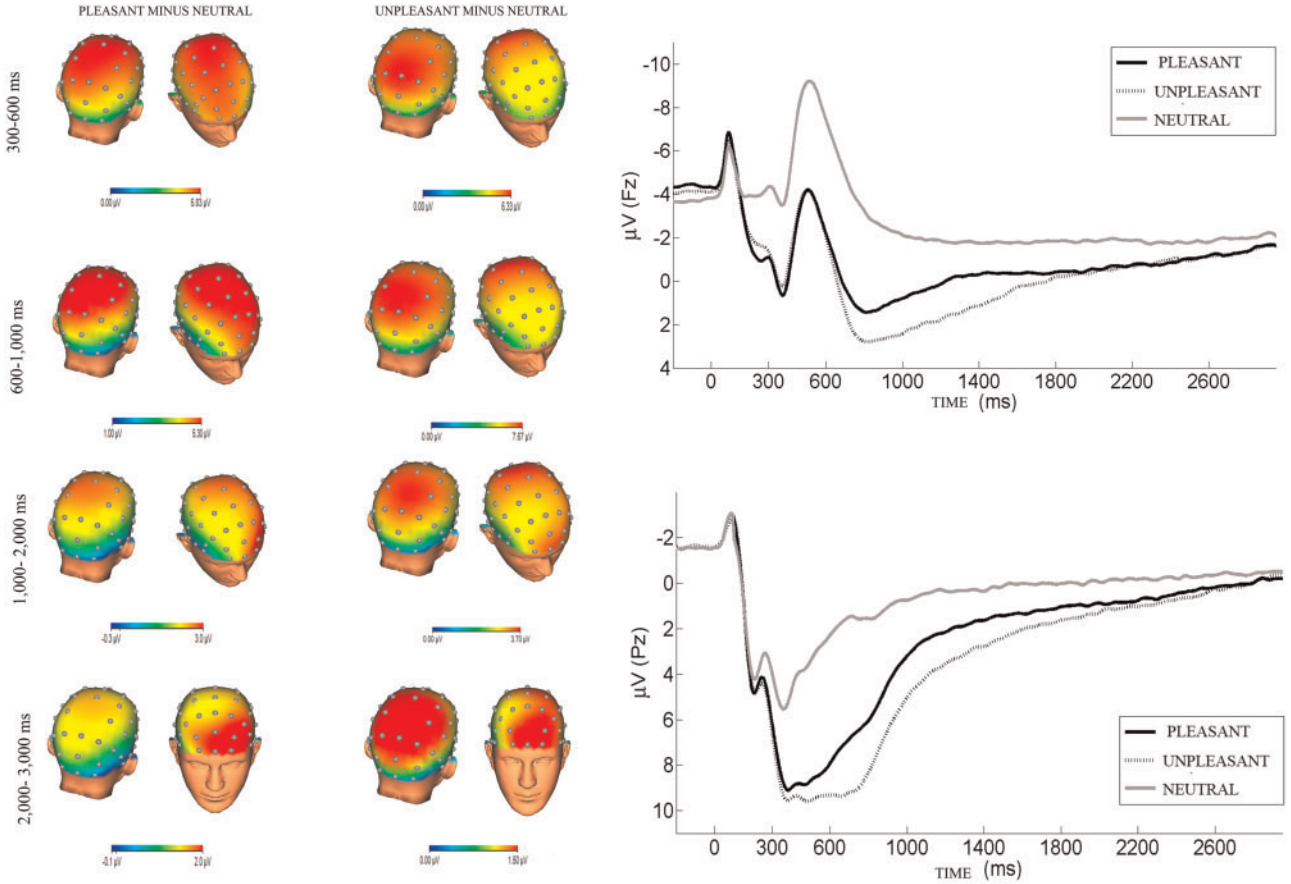

Fig. 2 Topographic maps for each of the four time windows of the LPP, depicting differences (in μV) in the sample as a whole for pleasant minus neutral and unpleasant minus neutral pictures; activity in each time window is depicted from two angles to display parietal (left) and frontal (right) effects. Also depicted are stimulus-locked ERP waveforms reflecting the average activity at Fz (top) and Pz (bottom), referenced to an average of the mastoids. Stimulus onset is at time 0 and negative is plotted up.

evidence of heritability at the frontal (Fz) recording site. Notably, no evidence was found for heritability of emotional modulation of the EPN, either for pleasant scenes or unpleasant scenes.

DISCUSSION

The present study sought to examine the contribution of genetic influences to multiple ERP components elicited by pleasant, neutral and unpleasant picture stimuli in a sample of adult MZ and DZ twins. Consistent with previous work (Cuthbert *et al.*, 2000; Olofsson *et al.*, 2008; Foti *et al.*, 2009; Hajcak *et al.*, 2012), robust emotional modulation of ERP components was evident across the time course of emotional picture processing, beginning as early as 175 ms and continuing up through 3 s following picture onset. However, there were important differences in the genetic contributions to different components, as we discuss below.

Heritable aspects of ERP responses to picture stimuli

We first considered the degree to which differing ERP components demonstrated heritability for pictures of each type (pleasant, neutral, unpleasant). Across picture types, the strongest and most consistent evidence of heritability was found for the centro-parietal P300 component observed between 300 and 600 ms. For this component, MZ twin correlations significantly exceeded DZ twin correlations for all picture types—indicating significant heritability for each. Our finding of robust heritability for the P300 response to affective and neutral picture stimuli is consistent with previously reported evidence for a substantial genetic contribution to the non-affective P300 elicited in oddball tasks (Katsanis *et al.*, 1997; Carlson and Iacono, 2006; Hall *et al.*, 2006), as well as with previous evidence from affective facial stimuli (Anokhin *et al.*, 2010). Indeed, the present data suggest that genes account for between 45 and 55% of variance in this component overall, which is comparable with previous reports of genetic contributions to personality trait characteristics (e.g. Blonigen *et al.*, 2005; Jang *et al.*, 2006, 2007). Thus, the magnitude of the picture-elicited P300 may represent a heritable trait-like index of stimulus processing.

In contrast, evidence for a genetic contribution to the EPN, presumed to reflect a more obligatory neural response associated with early perceptual registration, was more equivocal. Although for pictures of all types the correlation for MZ twins exceeded that for DZ twins, the difference in correlation values only reached significance for unpleasant images. However, the fact that MZ/DZ correlation differences for pleasant and neutral pictures were only somewhat lower than unpleasant, together with the fact that the heritable effect for unpleasant pictures was rendered null by controlling for its overlap with neutral picture EPN (see below), suggests that more compelling evidence of heritability of the EPN might well emerge for pictures of other types in samples of larger size.

Additionally, the present study is the first to demonstrate genetic contributions to later, more frontal, ERP components. Specifically, for the LPP between 2000 and 3000 ms, there was evidence of heritability for the response to unpleasant scenes, at scalp site Fz. Previous reports of an anterior shift in the distribution of the LPP over the course of

Table 2 Twin correlations and heritability estimates for each ERP variable, by picture condition

ERP variable	MZ	DZ	Comparison		Model-based estimates (90% CI)		
	ICC (95% CI)	ICC (95% CI)	*Z*	*P*	A^2	C^2	E^2
EPN (175–275 ms) Oz							
Pleasant	0.50* (0.34–0.62)	0.36* (0.18–0.52)	1.15	0.12		0.46 (0.40–0.55)	0.54 (0.45–0.64)
Neutral	0.54* (0.39–0.66)	0.44* (0.27–0.58)	1.12	0.13		0.50 (0.42–0.59)	0.50 (0.42–0.59)
Unpleasant	0.56* (0.41–0.67)	0.36* (0.18–0.52)	**1.92**	**0.03**	0.58 (0.49–0.67)		0.42 (0.34–0.52)
P300 (300–600 ms) Pz							
Pleasant	0.58* (0.44–0.69)	0.13 (0.05–0.32)	**3.85**	**<0.001**	0.54 (0.44–0.66)		0.46 (0.36–57)
Neutral	0.45* (0.28–0.59)	0.19** (0–0.38)	**2.21**	**0.02**	0.44 (0.33–0.57)		0.56 (0.44–0.68)
Unpleasant	0.58* (0.43–.69)	0.23** (0.05–0.41)	**2.99**	**<0.001**	0.54 (0.45–0.65)		0.46 (0.36–0.56)
P300 (300–600 ms) Fz							
Pleasant	0.40* (0.23–.55)	0.25**(0.06–0.42)	0.98	0.16	0.40 (0.29–0.53)		0.60 (0.49–0.72)
Neutral	0.44* (0.27–.58)	0.31** (0.13–0.48)	1.09	0.14		0.38 (0.39–0.48)	0.62 (0.53–0.72)
Unpleasant	0.55* (0.41–0.67)	0.28** (0.10–0.45)	**2.06**	**<0.05**	0.55 (0.49–0.66)		0.45 (0.36–0.55)
LPP (600–1000 ms) Pz							
Pleasant	0.37* (0.20–0.53)	0.13 (0.06–0.33)	**1.87**	**<0.05**	0.37 (0.25–0.52)		0.63 (0.50–0.77)
Neutral	0.47* (0.30–0.60)	0.17 (0.02–0.36)	**2.62**	**<0.05**			
Unpleasant	0.35* (0.17–0.51)	0.24** (0.06–0.41)	1.01	0.15			
LPP (600–1000 ms) Fz							
Pleasant	0.48* (0.32–0.61)	0.16 (0.03–0.35)	**2.40**	**<0.05**	0.48 (0.37–0.61)		0.52 (0.41–0.64)
Neutral	0.35* (0.18–0.51)	0.19** (0.01–0.37)	1.50	0.07	0.34 (0.23–0.48)		0.66 (0.54–0.79)
Unpleasant	0.44* (0.28–0.58)	0.22 (0.03–0.39)	**2.07**	**<0.05**	0.47 (0.36–0.60)		0.53 (0.42–0.65)
LPP (1000–2000 ms) Pz							
Pleasant	0.23* (0.05–0.41)	0.22** (0.01–0.38)	0.08	0.47	0.27 (0.15–0.43)		0.73 (0.59–0.87)
Neutral	0.42* (0.24–0.56)	0.21** (0.01–0.39)	**1.70**	**<0.05**	0.44 (0.32–0.58)	–	0.56 (0.44–0.69)
Unpleasant	0.17 (0.02–0.35)	0.09 (0.09–0.27)	0.45	0.32			
LPP (1000–2000 ms) Fz							
Pleasant	0.40* (0.23–0.55)	0.38* (0.20–0.53)	0	n/a			
Neutral	0.31* (0.14–0.47)	0.24* (0.04–0.41)	0.71	0.24	0.36 (0.24–0.50)		0.64 (0.52–0.78)
Unpleasant	0.35*(0.12–0.46)	0.15* (0.002–0.36)	1.56	0.06	0.33 (0.21–0.48)		0.67 (0.54–0.81)
LPP (2000–3000 ms) Pz							
Pleasant	0.25* (0.06–0.42)	0.13 (0.04–0.31)	0.90	0.19	0.25 (0.13–0.40)		0.75 (0.62–0.90)
Neutral	0.32* (0.13–0.48)	0.28* (0.08–0.45)	0.32	0.38		0.30 (0.20–0.41)	0.70 (0.60–0.81)
Unpleasant	0.17** (0.01–0.36)	0.14** (0.04–0.32)	0.39	0.34	0.20 (0.08–0.37)		0.80 (0.66–0.95)
LPP (2000–3000 ms) Fz							
Pleasant	0.24* (0.05–0.41)	0.29* (0.10–0.46)	−0.39	0.35		0.26 (0.16–0.38)	0.74 (0.64–0.85)
Neutral	0.09 (0.10–0.27)	0.26* (0.07–0.43)	−1.28	0.10		0.16 (0.06–0.29)	0.84 (0.73–0.96)
Unpleasant	0.35* (0.14–.47)	0.13 (0.01–0.28)	**1.77**	**<0.05**	0.31 (0.19–0.47)		0.69 (0.56–0.84)

Note: Z-scores are based on comparisons of ICCs for MZ and DZ twins. Significant *Z*-score values (and accompanying *P*s) are bolded. A^2 = heritability estimate/ additive genetic influence, C^2 = shared environmental influence, E^2 = non-shared environmental influence. Model-based estimates refer to estimates of genetic and environmental influence from best-fitting biometric models, in instances where fit matched or exceeded that for a saturated model. *$P < 0.01$, **$P < 0.05$.

picture presentation have suggested that this more frontal component reflects continued stimulus engagement and semantic elaboration (Hajcak and Olvet, 2008; Olofsson *et al.*, 2008; MacNamara *et al.*, 2009; Hajcak *et al.*, 2010, 2012, 2014). The implication is that the extent of this later elaborative processing is also subject to genetic influence; the question of whether this influence may be specific to emotional content is discussed below.

Heritable aspects of affective modulation of ERP components

Although a previous report demonstrated genetic contributions to amplitude differences in ERPs elicited by affective visual stimuli (Anokhin *et al.*, 2010), the present study provides the first evidence for a significant genetic contribution to affective modulation of ERPs to visual stimuli (i.e. the degree to which affective visual stimuli elicit larger ERPs than neutral stimuli). In particular, modulation of the P300 elicited by unpleasant scenes (i.e. the systematic variance in response to scenes of this type distinct from response to neutral scenes) was found to be modestly heritable. A trend-level effect for heritability of the P300 to pleasant *vs* neutral scenes was also evident for the 300–600 ms time window. Together, these results suggest that arousal-related increases in the P300 are in part heritable—and by extension, that this affect-modulatory effect might serve as an indicator of genetically transmitted variation in sensitivity to affective visual stimuli. From this perspective, the emotion-modulated P300 may hold potential as tool for the study of individual differences in early motivated attention (cf. Lang *et al.*, 1997).

Results for residual scores for the later, more frontal LPP were more equivocal. Although the MZ correlation exceeded the DZ correlation for the response to unpleasant *vs* neutral images at Fz between 1000 and 3000 ms, the fit of models for these time windows did not exceed the fit of a saturated model, precluding formal estimation of the level of genetic as compared with environmental influence. Nonetheless, model-based evidence for genetic influence specific to affective processes was evident for the full (non-residualized) late LPP scores. In particular, between 2000 and 3000 ms at scalp site Fz, there was evidence for genetic contributions to responses for unpleasant pictures, but not neutral. The implication is that later, more frontal neural activity is somewhat heritable, but perhaps only in the context of processing affective images. Further research will be necessary to corroborate and clarify this possibility.

On the other hand, residual scores for the EPN showed no reliable evidence of genetic influence, either for pleasant or unpleasant pictures.

Table 3 Twin correlations for ERP residual scores (pleasant controlling for neutral, and unpleasant controlling for neutral) reflecting variance attributable to emotion processing

ERP Variable	MZ	DZ	Comparison		Model-based estimates (90% CI)		
	ICC (95% CI)	ICC (95% CI)	*Z*	*P*	A^2	C^2	E^2
EPN (175–275 ms) Oz							
Pleasant	0.07 (.01–.25)	−0.02 (−0.21–0.17)	0.65	0.26			
Unpleasant	0.21* (0.03–0.39)	0.12 (−0.15–0.46)	0.67	0.25			
P300 (300–600 ms) Pz							
Pleasant	0.26** (.07–.43)	.05 (−0.15–0.25)	1.57	0.06	0.22 (0.11–0.37)		0.78 (0.65–0.92)
Unpleasant	0.35** (0.17–0.51)	0.09 (−0.11–0.27)	**2.00**	**<0.05**	0.29 (0.18–0.43)		0.71 (.59–.84)
P300 (300–600 ms) Fz							
Pleasant	0.11** (−0.09–0.29)	0.07 (−0.13–0.25)	0.29	0.39			
Unpleasant	0.33** (0.15–0.49)	0.11 (−0.09–0.29)	**1.69**	**<0.05**	0.30 (0.18–0.44)		0.70 (0.58–0.84)
LPP (600–1000 ms) Pz							
Pleasant	0.11 (−0.09–0.30)	0.08 (−0.12–0.28)	0.22	0.42			
Unpleasant	0.17 (−0.03–0.34)	0.16 (−0.04–0.34)	0.07	0.47			
LPP (600–1000 ms) Fz							
Pleasant	0.17 (−0.03–0.35)	0.20* (−0.003–0.38)	−0.22	0.41		0.19 (0.09–0.32)	0.81 (0.70–0.93)
Unpleasant	0.15 (−0.04–0.33)	0.12 (−0.08–0.31)	0.22	0.41			
LPP (1000–2000 ms) Pz							
Pleasant	0.09 (−0.10–0.28)	0.05 (−0.15–0.24)	0.29	0.39			
Unpleasant	0.01 (−0.18–0.20)	−0.10 (−0.27–0.10)	0.80	0.21			
LPP (1000–2000 ms) Fz							
Pleasant	0.29** (0.10–0.45)	0.17 (−0.04–0.34)	0.92	0.18			
Unpleasant	0.25** (0.07–0.41)	−0.07 (−0.23–0.14)	**2.21**	**<0.01**			
LPP (2000–3000 ms) Pz							
Pleasant	0.09 (−0.11–0.27)	0.05 (−0.15–0.25)	0.29	0.39			
Unpleasant	0.08 (−0.12–0.26)	0.10 (−0.16–0.22)	0.15	0.44			
LPP (2000–3000 ms) Fz							
Pleasant	0.19 (−0.06–0.31)	0.13 (−0.08–0.32)	0.46	0.32			
Unpleasant	0.34** (0.17–0.49)	−0.09 (−0.27–0.11)	**3.39**	**<0.001**			

Note: Z-scores are based on comparisons of ICCs for MZ and DZ twins. Significant *Z*-score values (and accompanying *P*s) are bolded. A^2 = heritability estimate/ additive genetic influence, C^2 = shared environmental influence, E^2 = non-shared environmental influence. Model-based estimates refer to estimates of genetic and environmental influence from best-fitting biometric models, in instances where fit matched or exceeded that for a saturated model. *$P < 0.05$, **$P < 0.01$.

Limitations and future directions

Though the current sample was large for an ERP study, it is modest in comparison with many studies that have used biometric modeling. Some of the marginal effects evident in the current work (e.g. evidence for heritability of the EPN across picture categories, or for affective modulation of the P300 by pleasant pictures) might well emerge as significant with increased statistical power in a larger sample.

In the present work, the strongest and most consistent evidence of heritability was found for the centro-parietal P300 component, consistent with previous studies demonstrating substantial heritability of the target-elicited P300 (Eischen and Polich, 1994; Katsanis *et al.*, 1997; Polich and Bloom, 1999; Iacono *et al.*, 2003; Hall *et al.*, 2006). This raises the question of whether the evidence for heritability in the affective viewing paradigm observed here reflects specific genetic influences on affective processing or instead general genetic influences on target processing. Because our study used a passive viewing paradigm, it is difficult to say definitively. Future studies in which target status and valence are fully crossed (e.g. Ferrari *et al.*, 2010; Weinberg *et al.*, 2012) will be needed to resolve this question. However, the evidence reported here for modest heritability of affective processing even after controlling for activity related to neutral images suggests some specificity of genetic contributions to emotion-related increases in the P300.

However, it is also worth noting that the estimates of genetic contributions to affective modulation of the P300 were more modest than those observed for the raw components. For example, in the 300–600 ms time window, estimates of additive genetic contribution to processing of neutral, pleasant and unpleasant images ranged from 0.45 to 0.55, whereas for modulation of the P300 by unpleasant images the estimate of heritability was 0.29. This may in part be due to the use of residual scores: While potentially preferable methodologically to subtraction-based difference scores, residuals likely still represent an imperfect method for isolating variance unique to affective processes (DuBois, 1957; Traub, 1967; Cronbach and Furby, 1970). Moreover, in either case, the use of neutral images as a non-affective comparison carries risks. For example, there is increasing evidence that arousal-related processes may be evident in the response to neutral images (e.g. Ito and Cacioppo, 2000; Weinberg and Hajcak, 2010; Ferri *et al.*, 2012). That is, the ERP response to neutral pictures may not represent a pure 'baseline'. Thus, factoring out variance related to responsivity to purportedly neutral images may result in a dilution of affect-related variance; to address this, future research might seek to replicate the current findings using alternative non-affective comparison conditions. Nonetheless, the current heritability estimates are comparable with those in other published research (e.g. Anokhin *et al.*, 2008, 2010), and even modest evidence for heritability of affective processing provides an impetus for further research along these lines.

Additionally, significant genetic contributions to emotional processing were most consistently observed for responses to unpleasant scenes. This might suggest specific genetic contributions to processing of unpleasant or threatening material, consistent with the notion that such material is more biologically relevant and motivationally salient than pleasant content (i.e. a negativity bias; Ito *et al.*, 1998). Processing of biologically salient material might also be more subject to selection pressures. However, we would caution against this interpretation. For one thing, the effects for pleasant pictures, although not significant, were consistently in the same direction and of similar magnitude as those for unpleasant scenes. Additionally, the pleasant category

included a subset of exciting/sports scenes (i.e. action), which are known to elicit ERP responses comparable with those for neutral images (Briggs and Martin, 2009; Weinberg and Hajcak, 2010). Inclusion of these exciting scenes may have diluted ERP responses to the pleasant category, making the results—particularly results concerning variance in pleasant picture response distinct from response to neutral scenes—somewhat more difficult to interpret. Inclusion of these scenes may also have contributed to the finding that unpleasant images elicited higher arousal ratings and larger LPPs than pleasant images, which might result in more reliable measurement. Future work could address this point by directly examining genetic contributions to ERPs elicited by pleasant and unpleasant picture stimuli equated for biological relevance (e.g. erotic and threat/mutilation images; Briggs and Martin, 2009; Weinberg and Hajcak, 2010; Weinberg *et al.*, 2012).

Nonetheless, the current results are novel and important in that they suggest heritability of individual differences in neural indices of emotion processing, both in terms of early, relatively obligatory allocation of attention and later, more elaborative processing. Given increasing contemporary interest in the neurogenetics of psychiatric disorders (Cloninger, 1987; Anokhin *et al.*, 2004; Cuthbert and Insel, 2010; Insel *et al.*, 2010; Bogdan *et al.*, 2012a, b; Yates, 2012), our findings indicate that the P300 and sustained LPP might serve as useful intermediate phenotypes in research investigating neurobiological and genetic underpinnings of a range of neuropsychiatric disorders. Measures of this type are likely to be particularly valuable because multiple psychiatric disorders are characterized by abnormalities in emotion processing, whether in terms of maladaptive early attention (Mogg *et al.*, 2004; Li and Luo, 2005; MacNamara and Hajcak, 2009, 2010; Van Strien *et al.*, 2009; Weinberg and Hajcak, 2011a), or deficits in sustained affective processing (e.g. Patrick *et al.*, 1993; Levenston *et al.*, 2000; Franken, 2003; Foti *et al.*, 2010; Dunning *et al.*, 2011; Weinberg and Hajcak, 2011a). Related to this, a wide array of psychiatric disorders display abnormalities in the magnitude of the emotion-modulated P300 and LPP, including phobias (Moser *et al.*, 2008; Leutgeb *et al.*, 2009; Mühlberger *et al.*, 2009), panic disorder (Pauli *et al.*, 1997), generalized anxiety disorder (MacNamara and Hajcak, 2010; Weinberg and Hajcak, 2011a), depression (Foti *et al.*, 2010), schizophrenia (Horan *et al.*, 2010) and substance abuse (Franken, 2003; Dunning *et al.*, 2011).

Furthermore, an extensive body of non-affective research using the oddball P300 documents a relationship between reduced P300 amplitude and externalizing disorders and tendencies (e.g. Brigham *et al.*, 1995; Iacono *et al.*, 2003, 2008; Bernat *et al.*, 2011) that appears to reflect shared genetic influence (Hicks *et al.*, 2006). However, no work to date has explored how genetic contributions to emotional modulation of the P300 might be associated with pathological emotional responding. The current results, which identify genetically transmitted variation in neural indices of emotional response, may therefore facilitate the identification of neurobiological markers for neuropsychiatric disorders, and thereby set the stage for future studies directed at identifying the contribution of genetic factors to the association between affect-modulated P300/LPP and disordered emotional response.

SUPPLEMENTARY DATA

Supplementary data are available at *SCAN* online.

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APPENDIX A

The 90 affective pictures, listed by their IAPS identification numbers, were as follows: pleasant, 5621, 8030, 8080, 8185, 8186, 8370, 8200, 8490, 8180, 4659, 4660, 4687, 4695, 4670, 4681, 1710, 2040, 2150, 2340, 1440, 2154, 2080, 2071, 2058, 2350, 1750, 2530 (males only: 4210, 4180, 4232; females only: 4572, 4542, 4538); neutral, 7004, 7010, 7020, 7041, 7175, 7185, 7000, 7187, 7035, 7179, 7491, 7705, 7100, 5740, 7050, 7150, 5510, 7059, 7510, 7700, 2038, 2190, 2480, 2840, 2393, 2890, 2102, 2280, 2397, 2215; and unpleasant: 3053,3102, 3080, 3120, 3130, 3000, 3060, 3010, 3071, 1525, 1050, 1205, 1300, 2811, 6230, 6250, 6260, 6300, 6370, 6830, 2692, 6200, 6210, 6213, 6243, 6244, 6570, 3064, 3280, 1220. Mean valence and arousal normative ratings (Lang et al., 2008) for each valence category by gender were as follows: for females, pleasant: valence (mean = 7.58, s.d. = 0.79); arousal (mean = 5.86, s.d. = 1.01); neutral: valence (mean = 4.96, s.d. = 0.34); arousal (mean = 2.71, s.d. = 0.51); and unpleasant: valence (mean = 2.13, s.d. = 0.72); arousal (mean = 6.77, s.d. = 0.68). For males, pleasant: valence (mean = 7.49, s.d. = 0.47); arousal (mean = 6.05, s.d. = 1.27); neutral: valence (mean = 4.94, s.d. = 0.30); arousal (mean = 2.71, s.d. = 0.62); and unpleasant: valence (mean = 2.93, s.d. = 0.87); arousal (mean = 6.17, s.d. = 0.62).