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BRIEF REPORT

MAOA T941G polymorphism and the time course of emotional recovery following unpleasant pictures

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Abstract

Difficulty down-regulating negative affect has been linked with anxiety and depression. In addition, recent studies have identified specific polymorphisms of the MAOA gene related to affective psychopathology. Here we examined whether genetic variation in MAOA was associated with the time course of responses to affective stimuli. Emotion-modulation of the startle blink response was measured during and after affective pictures. Women with the G/G genotype of the MAOA T941G single nucleotide polymorphism showed sustained reactivity to unpleasant stimuli, as evidenced by continued blink potentiation during the picture-offset period. These data suggest that the MAOA T941G polymorphism, which has been previously linked with mood disorders, is associated with a maladaptive pattern of affective responding in women.

Descriptors: Startle response, Individual differences, Genetics

Emotion dysregulation is a core feature of numerous forms of affective pathology. Difficulty down-regulating negative affect is particularly evident in depression, anxiety and a number of other Axis I and II disorders (American Psychiatric Association, 2000; Taylor & Liberzon, 2007). A substantial body of research has demonstrated that individuals with these disorders exhibit greater intensity of negative affect (Clark & Watson, 1991). In addition, recent literature indicates that individuals with depression, dysphoria, and anxiety also show longer lasting responses to negatively valenced stimuli (Deldin, Deveney, Kim, Casa, & Best, 2001; Larson, Nitschke, & Davidson, 2007; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002).

Sustained processing in the period following offset of an unpleasant stimulus has been observed in depressed individuals as indexed by event-related potentials, pupil dilation, and respiratory sinus arrhythmia (Deldin et al., 2001; Deveney & Deldin, 2004; Rottenberg, Kasch, Gross, & Gotlib, 2002; Siegle, Granholm, Ingram, & Matt, 2001; Siegle, Steinhauer, Carter, et al., 2003). Prolonged responses to negative stimuli in depression have also been associated with sustained activation of the amygdala (Siegle, Steinhauer, Thase, et al., 2002). Even at sub-clinical levels, anxious and dysphoric subjects show slower decay of negative affect (Larson et al., 2007). Neuroticism, a robust risk factor for internalizing disorders (Trull & Sher, 1994), is linked with sustained subjective experience of negative affect (Shuls, Green, & Hillis, 1998) and sustained medial prefrontal activation during a sadness induction (Haas, Constable, & Canli, 2008). Furthermore, potentiation of startle blink responses following the offset of unpleasant pictures is present among unselected subjects exhibiting a pattern of hemispheric asymmetry associated with distress disorders (Jackson, Mueller, Dolski, Dalton, Nitschke, et al., 2003; Larson & Davidson, 2001). As potentiation of startle blink has been shown to be mediated by amygdala influences on brainstem startle circuitry (Antoniadis, Winslow, Davis, & Amaral, 2007; Campeau & Davis, 1995), together these neuroimaging and psychophysiological studies implicate both the amygdala and medial prefrontal cortex in sustained negative affect. Thus, this brief snapshot of affective recovery, continuing to respond to unpleasant stimuli in the first few seconds following offset of unpleasant stimuli, may serve as a useful index of emotion dysregulation in distress disorders and negative affective style.

A separate line of research has begun to identify candidate genes associated with neural and behavioral emotion regulation mechanisms that confer risk for affective psychopathology (Canli & Lesch, 2007; Ebstein, 2006). One candidate gene implicated in risk for affective disorders is the monoamine oxidase A (MAOA) gene. Variation in this gene has been linked with depression (Du, Bakish, Ravindran, & Hrdina, 2004; Schulze, Muler, Krauss, Scherk, Ohlraun, et al., 2000) and anxiety (Samochowiec, Hajduk, Samochowiec, Horodnicki, Stepien, et al., 2004). The MAOA gene encodes the enzyme MAOA, which catalyzes the degradation of a number of neurotransmitters, including serotonin, norepinephrine, and dopamine. Inhibition of MAO activity is associated with improvement of symptoms of depression (Quitkin, Rifkin, & Klein, 1979) and anxiety (van Vliet, Westenberg, & Den Boer, 1993), possibly through promoting lasting increases in firing of midbrain dopamine neurons (Mercuri, Scarponi, Bonci, Siniscalchi, & Bernardi, 1997).

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PSYCHOPHYSIOLOGY

One specific polymorphism of the MAOA gene, the T941G SNP, has been linked with depression and anxiety (Tadic, Muller, Rujescu, Kohne, Stassen, et al., 2007; Tadic, Rujescu, Szegedi, Gigling, Singer, et al., 2003). The T-allele of this polymorphism is associated with lower MAOA enzyme activity (Hotamisligil & Breakefield, 1991), which results in decreased amine degradation and increased availability of neurotransmitters implicated in depression and anxiety. This idea is supported by findings that T-homozygotes show improved long-term affective recovery, exhibiting faster and more significant response to the antidepressant mirtazapine (Tadic et al., 2007). The G-allele of the T941G SNP, which is associated with higher MAOA enzyme activity, has the opposite effect, increasing amine degradation and decreasing the availability of neurotransmitters such as serotonin, dopamine, and norepinephrine.

In the current study, we examined whether the MAOA T941G polymorphism[1] is associated with more short-term emotion regulation processes that have been linked with trait negative affectivity and affective disorders. We used an emotion-modulated startle paradigm to assess whether variation in this polymorphism was associated with individual differences in affective recovery during the first several seconds following offset of emotionally laden stimuli. Given the advantage in recovery from depression conferred by the T/T genotype, we predicted that individuals with this variant would show faster recovery following unpleasant images. The sample was limited to women for both conceptual and practical reasons. First, internalizing disorders are more prevalent in women (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993), and second, the T941G SNP is located on the X-chromosome; therefore men have only one copy of either the T or G allele and the effects of heterozygosity cannot be assessed.

Method

Participants

Ninety-seven undergraduate women participated (mean age = 18.92, $SD = 0.85$). All were free of psychotropic medication and were native English speakers. These data were collected as part of a larger project in which electroencephalographic data were collected, so participants were free of head injury or neurological disorder and were right-handed. The sample was predominantly white ($N = 91$), and also included one African American, one Hispanic, and four Asian participants.[2] Median family annual income was rated as between $60,001 and $80,000.

Materials

Forty-two pictures of each of three valences (unpleasant, pleasant, and neutral) were selected from the International Affective Picture System[3] based on published norms for valence and arousal (Lang, Bradley, & Cuthbert, 2008). Unpleasant and pleasant pictures were selected to be toward the extreme ends of the bipolar valence rating scale and high on arousal. Neutral pictures were selected to be in the middle of the valence scale and low on arousal. Unpleasant pictures included mutilated bodies, physical assault scenes, guns aimed at the viewer, and accident scenes. Pleasant pictures depicted sports and adventure, nature scenes, money, and erotica (four pictures of romantic couples). The neutral pictures primarily consisted of household objects, but also included plants and neutral faces.

Procedure

Informed consent was obtained from participants upon arrival at the laboratory. Prior to picture presentation, electrodes for recording blink responses were applied. In order to familiarize the participants with the procedure and habituate them to the acoustic startle probe, they first viewed an introductory set of 9 pictures, during 8 of which startle probes were presented.

Pictures were presented on a 21-in LCD monitor in a quasi-random order (not more than two of each valence presented consecutively) for 6 s with a 12–18 s blank screen intertrial interval. The startle probe was a 50 ms 100 dB burst of white noise presented binaurally via pneumatic intra-ear headphones. Probes were presented during 36 pictures of each condition at one of four probe times: 1.5, 4.5, 7.5, or 9 s post-stimulus onset. Nine trials of each valence and probe time combination were presented. Stimuli were presented in six blocks with 21 trials per block (7 of each valence).

Startle Recording and Quantification

Electromyography (EMG) was recorded using two SensorMedics mini-electrodes (Conshohocken, PA) placed approximately 36 mm apart on the inferior left orbicularis oculi muscle (van Boxtel, Boelhouwer, & Bos, 1998) (impedance <20 Kohms). EMG signals were amplified 10,000 times and filtered with a bandpass of 1–200 Hz using bioelectric amplifiers (SAI Instrumentation, Caroga Lake, NY). A 30 Hz high pass filter was applied prior to integration and rectification of the raw EMG signals (time constant = 20 ms). A PC running SnapMaster software (HEM Data Corporation, Springfield, MI) and a 12-bit analog-to-digital board (Analogic Corporation, Wakefield, MA) was used to digitize signals at 500 Hz.

Blink magnitude, computed as the difference between magnitude at peak and at blink onset, was calculated for a window between 20 and 120 ms following probe onset. Trials with no perceptible eye-blink reflex were assigned a magnitude of zero and included in analysis. The mean number of no-response trials was 7.54 ($SD = 4.22$), representing 6.9% of the total number of trials. To correct for individual variance in blink magnitude,

[1]Based on mounting evidence (e.g., Hariri, Mattay, Tessitore, Kolachana, Fera, et al., 2002; Heinz, Braus, Smolka, Wrase, Puls, et al., 2005), we also hypothesized that individuals with the S-allele of the serotonin transporter promoter (5-HTTLPR) gene would show sustained reactivity to unpleasant affective stimuli. However, we did not find any significant effects for affect modulation of startle at any probe time as a function of this gene. Thus, for brevity's sake the full results for this gene are not reported here.

[2]Due to potential problems associated with population stratification, analyses were also run without the six non-Caucasian participants, but the findings were unchanged. Thus, participants from all ethnic groups were included in the final analyses.

[3]IAPS pictures used in this study were:
Unpleasant: 3000, 3010, 3015, 3030, 3051, 3053, 3060, 3071, 3080, 3100, 3102, 3120, 3130, 3140, 3150, 3168, 3170, 3266, 3350, 3400, 3500, 3530, 6212, 6230, 6260, 6312, 6313, 6350, 6360, 6510, 6560, 6570, 9040, 9252, 9410, 9500, 9560, 9570, 9800, 9810, 9910, 9921;
Neutral: 1670, 2620, 5510, 5520, 5531, 5532, 5533, 5534, 5731, 6150, 7000, 7002, 7006, 7009, 7010, 7025, 7030, 7034, 7035, 7040, 7050, 7060, 7080, 7090, 7100, 7130, 7140, 7150, 7170, 7190, 7207, 7217, 7224, 7233, 7234, 7235, 7490, 7500, 7700, 7710, 7920, 9210;
Pleasant: 1710, 2216, 2391, 4599, 4660, 4670, 4680, 5260, 5270, 5450, 5460, 5470, 5480, 5621, 5623, 5629, 5700, 5910, 7230, 7270, 7502, 8030, 8034, 8080, 8170, 8180, 8185, 8190, 8200, 8210, 8300, 8340, 8370, 8380, 8400, 8420, 8470, 8500, 8501, 8502, 8510, 8531.

reflex magnitudes were z-transformed within subject. Blinks greater than 3 standard deviations from each participant's mean were excluded. Approximately 12.9% of eyeblink reflexes were excluded (treated as missing values) due to an unstable baseline (50 ms preceding probe onset), or because reflex onset occurred prior to 20 ms following probe onset. All participants had at least three good startle responses of the nine possible for each cell (Picture Condition × Probe Time).

Genotyping

DNA was sampled using Oragene saliva collection kits (DNA Genotek, Ottawa, ON, Canada). The MAOA T941G SNP (position 106, exon 8, chromosome Xp11) was amplified using the following primers: forward (5′ GCT TCC AGC AGA GAG AAA CCA 3′) and reverse (5′ GGC CTC CTT GTA ATA CAT CAT GCA 3′). PCR was performed in a 5 μl volume (3 μl of master mix plus 2 μl of DNA) in a 384-well plate using the ABI 7900HT Fast Real-Time PCR System (Applied Biosystems, Foster City, CA). An initial denaturation step was run at 95°C for 10 min followed by 50 cycles at 92°C for 15 s and 60°C for 1 min. Finally, samples were held at 4°C. ABI SNP analysis software (Foster City, CA) was used to determine allele status. Four participants were dropped because the DNA could not be amplified, yielding a final sample of 93 participants.

Results

Allele Frequencies

Genotypic frequencies were 6.5% G/G ($N = 6$), 39.8% G/T ($N = 37$), and 53.8% T/T ($N = 50$), which is in keeping with Hardy-Weinberg equilibrium, $\chi^2 = 0.06$, $df = 1$, $p = 0.81$.

Emotion-modulated Startle Blink

A Genotype (G/G, G/T, T/T) × Picture Condition (pleasant, unpleasant, neutral) × Probe Time (1.5, 4.5, 7.5, 9 s) mixed model ANOVA was computed (Huyhn-Feldt correction applied to all ANOVA statistics). Most importantly for the hypotheses in the current study, the Genotype × Picture Condition × Probe Time interaction was significant, $F(12,540) = 2.074$, $p < .03$, $\eta^2 = .102$. To isolate the differences driving this interaction, Genotype × Picture Condition ANOVAs were conducted separately for each probe time. While the 1.5, 4.5, and 7.5 s probe times all yielded the expected main effect for Picture Condition ($ps < .002$), no interactions with Genotype emerged at any of these points in time ($ps > .69$). However, at the 9 s probe time, the Picture Condition × Genotype interaction was significant, $F(4,180) = 4.40$, $p < .005$, $\eta^2 = .089$. Post-hoc comparisons revealed significantly greater blink potentiation for unpleasant compared to neutral pictures for G/G compared to T/T, $t(54) = 3.40$, $p < .002$, and G/T participants, $t(41) = 2.74$, $p < .01$ (all post-hoc comparisons Bonferroni corrected). There was no difference between T/T and G/T individuals ($p = .70$). At 9 s, G/G women also showed potentiation of blinks for pleasant versus neutral pictures compared to G/T, $t(41) = 2.52$, $p = .01$, and T/T women, $t(54) = 3.68$, $p < .002$. For ease of interpretation, emotion modulation difference scores (unpleasant-neutral, pleasant-neutral) are presented in Figure 1. Table 1 presents the mean blink magnitudes for each cell in the design.

In addition to the findings for genotype and time course, the expected main effect for Picture Condition was significant, $F(2, 180) = 29.98$, $p < .001$, $\eta^2 = .364$, indicating that across probe time and genotype blinks were largest for unpleasant compared to pleasant, $p < .001$, and neutral, $p < .002$, pictures. Blink magnitudes for pleasant and neutral pictures did not differ ($p > .90$). In addition, there was a main effect for Probe Time, $F(3,270) = 5.425$, $p < .002$, $\eta^2 = .171$. Post-hoc comparisons indicated that blinks were larger at 9 s than at both 1.5, $p = .024$, and 7.5 s, $p < .001$. A significant Picture Condition × Probe Time interaction was also present, $F(6,540) = 4.059$, $p < .002$, $\eta^2 = .230$, and indicated that blink magnitudes were greater for unpleasant pictures compared to both neutral and pleasant at the 1.5, 4.5, and 7.5 s probe times, but not at the 9 s probe time.

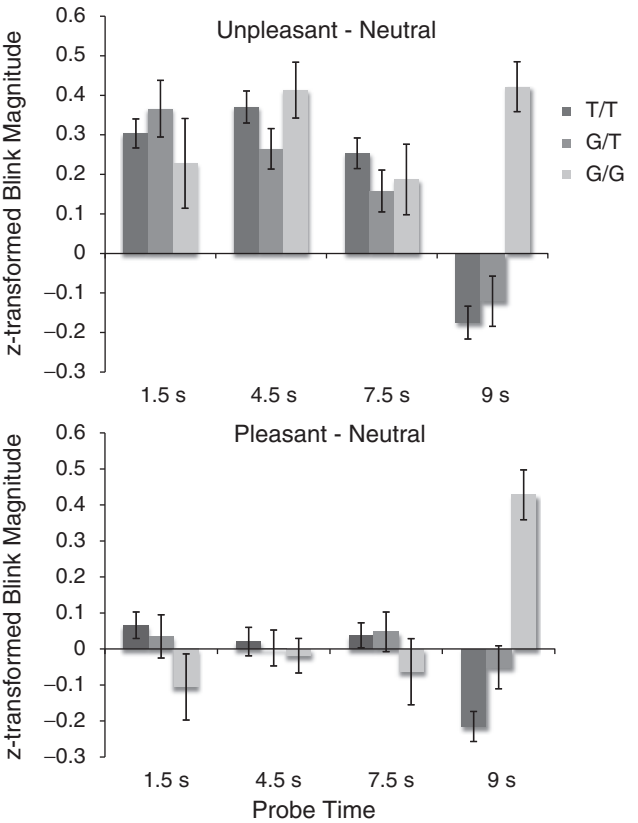

Figure 1. Mean z-transformed emotion-modulation blink difference scores at each probe time for the three MAOA T941G genotypes. Pictures were presented for 6 s, thus the last two probe times followed picture offset. The top panel depicts unpleasant minus neutral blink magnitudes. Positive numbers reflect greater potentiation to unpleasant pictures. The bottom panel depicts pleasant minus neutral blink magnitudes. Negative numbers reflect greater attenuation of the blink response to pleasant compared to neutral stimuli. Error bars represent standard error.

Following up on the significant main effect for Condition, at 1.5, 4.5, and 7.5 s for each probe time, blink reflexes were significantly larger for unpleasant compared to neutral pictures (all $ps < .001$). Furthermore, this held true for each of the three genotype groups (all $ps < .04$). However, significant modulation of blink responses to pleasant pictures, defined as significant attenuation of blink magnitudes for pleasant compared to neutral pictures, was not evident for any of the three probe times ($ps > .33$). Examining each of the three genotype groups separately also revealed no significant differences between pleasant and neutral pictures ($ps > .27$). Thus, for the first three probe

Table 1. *Mean z-Transformed Blink Magnitudes for Each Picture Condition, Probe Time, and MAOA T941G Genotype*

	Probe time			
	1.5 s	4.5 s	7.5 s	9 s
T/T				
Unpleasant	.12 (.32)	.29 (.34)	.12 (.27)	.01 (.28)
Neutral	−.19 (.22)	−.08 (.22)	−.14 (.24)	.19 (.29)
Pleasant	−.12 (.27)	−.06 (.21)	−.10 (.27)	−.03 (.29)
G/T				
Unpleasant	.20 (.39)	.22 (.24)	.05 (.26)	−.02 (.28)
Neutral	−.16 (.31)	−.04 (.27)	−.11 (.22)	.10 (.33)
Pleasant	−.13 (.28)	−.04 (.19)	−.06 (.28)	.05 (.28)
G/G				
Unpleasant	.12 (.24)	.27 (.18)	.09 (.26)	.28 (.19)
Neutral	−.10 (.24)	−.14 (.28)	−.10 (.32)	−.14 (.23)
Pleasant	−.21 (.37)	−.15 (.34)	−.16 (.22)	.29 (.23)

times, the typically observed potentiation of blink responses to unpleasant pictures was observed; however, blink attenuation to pleasant pictures was not evident (see Figure 1). Therefore, while a significant difference was present for modulation following offset of pleasant pictures at the 9 s probe time, the lack of modulation at earlier probe times makes this finding difficult to interpret. As such, strong conclusions based on the pleasant picture data seem unwarranted and are reported primarily for completeness.

Out of concern that the unequal sample sizes of the genotype groups may have affected the results, the emotion-modulation difference scores for the 9 s time point were further examined via Kruskal-Wallis test (case-level data for these difference scores are also presented in Figure 2). Results of this test supported the ANOVA findings. Genotype had a significant effect on blink magnitude for both unpleasant and pleasant compared to neutral pictures ($H(2) = 10.885$, $p < 0.01$; $H(2) = 12.69$, $p < 0.01$, respectively) at the 9 s time point. Post hoc comparisons of genotype groups were done using Mann-Whitney tests, Bonferroni corrected so that all significant effects are $p < .0167$. G/G women had significantly larger blinks for unpleasant compared to neutral pictures than did the G/T ($U = 31$, $p < 0.01$, $r = 0.43$) and T/T women ($U = 24$, $p < 0.01$, $r = 0.45$). Similarly, G/G women had significantly larger responses to pleasant compared to unpleasant pictures than did women with the G/T ($U = 29$, $p < 0.01$, $r = 0.44$) or T/T genotypes ($U = 30$, $p < 0.01$, $r = 0.42$). Finally, G/T and T/T women did not differ for either unpleasant compared to neutral ($U = 844$, $p = 0.49$, $r = 0.07$) or pleasant compared to neutral responses ($U = 724$, $p = 0.08$, $r = 0.19$).

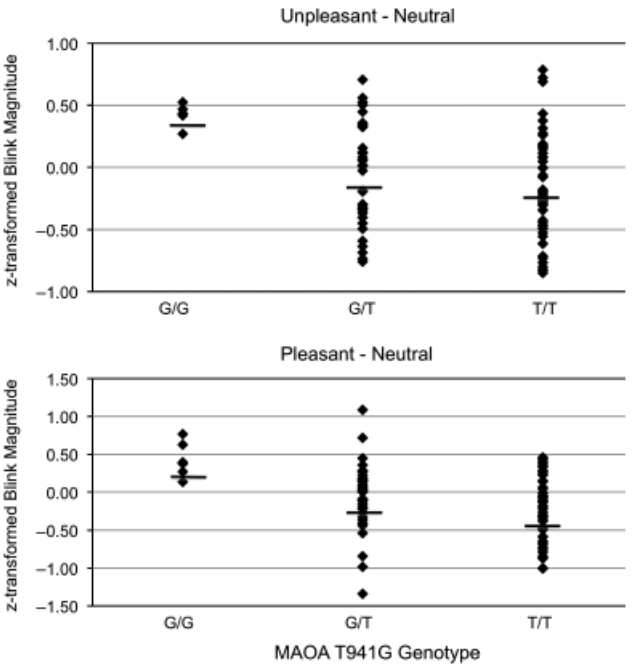

Figure 2. Mean z-transformed emotion-modulation blink difference scores for each participant at the 9 s probe time (3 s post-picture offset). The top panel represents unpleasant minus neutral blink magnitudes, and the bottom panel shows pleasant minus unpleasant magnitudes. The horizontal lines indicate the mean difference score for each genotype groups. Note that the *y*-axis scale is different for the top and bottom graphs.

Discussion

MAOA T941G was associated with recovery following affective stimuli in women. Specifically, women homozygous for the high-activity G-allele showed continued potentiation 3 s after the offset of unpleasant pictures, while women either homozygous or heterozygous for the low-activity T allele did not. The pattern of sustained potentiation following the offset of unpleasant pictures in women homozygous for the G-allele is consistent with that observed in anxious and dysphoric individuals (Larson et al., 2007).

There is also evidence from the pharmacogenetic literature supporting the idea that MAOA genotype may influence affective recovery. In comparison with G-carriers, homozygosity for the T allele of the T941G SNP has been previously linked with faster and more robust response to mirtazapine, an antidepressant acting on both serotonin and norepinephrine transmission (Tadic et al., 2007). Thus, women T-carriers appear to show faster affective recovery than women with the G/G genotype, both in terms of brief natural recovery following unpleasant stimuli, and in more long-term recovery from clinical depression. Similarly, Domschke and colleagues (2005) reported that women with high-activity genotypes of a 30 bp variable number tandem repeat (VNTR) in the promoter region of the MAOA gene exhibited a slower and less efficient response to antidepressant treatment. This provides additional evidence that individuals with low-activity MAOA genotypes may show enhanced recovery from depression in comparison to individuals with high-activity MAOA genotypes.

These findings are in concordance with what would be expected from the literature regarding the function of MAOA. MAOA degrades amine neurotransmitters, and medications that inhibit MAOA activity have been found to be effective in the treatment of internalizing disorders (Fitton, Faulds, & Goa, 1992; Papakostas & Fava, 2006). In addition, Meyer, Ginovart, Boovariwala, Sagrati, Hussey, et al. (2006) found that MAOA density was significantly elevated in individuals with depression, suggesting a possible cause of the significant loss of monoamines evident in depression. Studies examining other polymorphisms of the MAOA gene have found associations between high-activity

MAOA genotypes and depression (Schulze et al., 2000) and panic disorder (Deckert, Catalano, Syagailo, Bosi, Okladnova, et al., 1999) in women. Along similar lines, Hotamisligil and Breakefield (1991) found that the occurrence of a G at position 941 of the MAOA gene results in heightened levels of MAO activity, while a T results in lower levels of MAO activity. Because sustained negative affect has been observed in individuals with anxiety and depression and high MAOA activity has been associated with these disorders, the observation in the present study that women with the high-activity genotype of the T941G SNP exhibit sustained potentiation to unpleasant stimuli is consistent with previous literature.

While a growing body of research suggests that recovery from unpleasant stimuli in the seconds following stimulus-offset is linked with depression and likely anxiety (e.g., Larson et al., 2007; Siegle et al., 2001, 2003), the link between these brief snapshots of sustained negative affect and long-term mood pathology is less clear. One likely mechanism linking these phenomena is rumination, defined as perseverating on one's own sad or depressed mood (Nolen-Hoeksema, 1991). Rumination has been repeatedly shown to increase risk for depression, and prolong and intensify episodes of depression once they begin (Nolen-Hoeksema, 1991; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). The risk for depression conferred by trait negative affectivity appears to be mediated in part by rumination (Roberts, Gilboa, & Gotlib, 1998). Of more direct relevance, sustained amygdala response to negative words in depressed individuals is correlated with increased rumination (Siegle et al., 2003).

More broadly, these data show initial support for combining molecular genetic and psychophysiological techniques for understanding affective recovery and emotion regulation. While the combination of genetic and psychophysiological or neuroimaging techniques to understand emotion regulation and psychopathology is becoming more common, we are aware of only one other candidate gene study of emotion-modulation of startle blink. In this study, blink potentiation to unpleasant stimuli was observed in Met homozygotes of the catechol-O-methyltransferase Val158Met polymorphism (Montag, Buckholtz, Hartmann, Merz, Burk, et al., 2008); however, this was examined only during, not following, picture presentation. Together, the results of the present study and the results of Montag and colleagues' (2008) study provide an impetus for future examination of the relationship between genotype and performance on psychophysiological measures.

As noted in Footnote 1, we also examined the time course of affective responding as a function of the serotonin transporter promoter gene (5-HTTLPR), but found no significant effects. The lack of findings for this SNP may be in part due to one of the limitations of this or any other candidate gene association study. While candidate gene studies have become common and have made important contributions to understanding individual differences in complex behaviors and traits, recent work has also made clear that such studies are haunted by failures to replicate and Type I error concerns (Sullivan, 2007; van den Oord, 2008). Recently, authors have suggested selecting a two-tail alpha value of about 0.0005 rather than the traditional value of 0.05 in order to minimize the rate of false positive findings (Sullivan, 2007; van den Oord, 2008). While the *p*-values obtained in this study fall beneath the traditional 0.05 threshold, they are greater than the suggested value of 0.0005. Thus, while the results of the present study are promising, the potential for Type I error cannot be ignored, and replication of the findings will be important for determining the robustness of the observed effect.

A number of other limitations should also be taken into consideration. First, our sample was limited to women. While others who have examined the T941G SNP and other MAOA polymorphisms have found that MAOA genotype only has a significant effect in women, assessment of a sample of men is needed to rule out the possibility that T941G genotype affects startle potentiation in men as well as in women. Within our sample of women, the number of women homozygous for the G allele was also quite small. While the group differences in sustained blink potentiation are large and all of the G/G women showed clear potentiation at the 9 s probe time, the small sample size for this group also indicates a need for further replication. Finally, although our findings are consistent with much of the literature on MAOA functioning, the extant literature on the T941G SNP is small, and at least one study that we are aware of yielded seemingly contradictory results (Tadic et al., 2003). Thus, future work is needed to more precisely specify the role of the MAOA T941G SNP in relation to mechanisms conferring risk for psychopathology.

Overall, however, using the T941G SNP, the present study provides support for the idea that females with low-activity MAOA genotypes may show enhanced recovery from depression in comparison to females with high-activity MAOA genotypes. Specifically, it provides evidence that female T-carriers of the MAOA T941G SNP show faster recovery following negative affective stimuli compared to women with the G/G genotype. More broadly, it suggests that it may be useful to combine molecular genetic and psychophysiological measures in the study of affective recovery and emotion regulation.

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