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Genetic moderation of sensitivity to positive and negative affect in marriage

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Abstract

Hypothesizing that genetic factors partially govern sensitivity to interpersonal cues, we examined whether a polymorphism (5-HTTLPR) in the serotonin transporter gene would moderate spouses' sensitivity to positive and negative partner affect. Before and after marital discussions, participants from seventy six couples (total $n = 150$) reported their affective states. Spouses carrying the short allele of the 5-HTTLPR were more responsive to their partner's pre-interaction positive affect and anxiety/nervousness, compared to spouses with two long alleles. These data support the contention that the serotonin system influences affective responses to social stimuli. In contrast to the view that the 5-HTTLPR primarily affects response to adverse experiences, these results suggest that this polymorphism moderates sensitivity to positive as well as negative affect.

Keywords

Affect sensitivity; Emotional transmission; 5-HTTLPR; Marriage

Many of our emotions are experienced and regulated in the context of personal relationships (Bradbury & Karney, 2010; Butler, 2011). Close partners influence one another's emotional states, and several studies illustrate how interactions between partners, and partners' personal characteristics, shape the emotional dynamics in a relationship. For example, the emotions that one partner feels at the end of the workday can influence the feelings of the partner after they reunite (Schoebi, 2008), and factors like attachment style (Butner, 2007), or cultural values (Schoebi, Wang, Ababkov & Perrez, 2010) moderate such emotional transmission. The present study aims to extend understanding of emotional interdependence in intimate dyads by examining genetic moderation of emotion transmission from before to after marital interaction. Doing so allows us to investigate the biological basis of emotional experiences in marriage, while also addressing hypothesized genetic influences on sensitivity to social behavior.

One particularly intriguing candidate for moderating the transmission of affect is variation in the promoter region of the serotonin transporter gene (5-HTTLPR). At this locus, two

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principal alleles, short and long, appear to differentially affect emotional sensitivity to life events. This has been documented extensively in studies of depression, where the mood of 5-HTTLPR short-allele (S) carriers is more affected by stressful events than individuals with two long alleles (L; Uher & McGuffin, 2010).

Greater sensitivity of S-allele carriers appears to extend to positive experience as well (Belsky & Pluess, 2009; Homberg & Lesch, 2010). This research suggests that the S-allele is not functioning solely as a marker of vulnerability, but is instead a general marker for sensitivity to life experiences, regardless of valence. However, because the dependent measures in these studies typically assess psychopathology, they can only demonstrate that positive social experiences reduced psychopathology (Belsky et al., 2009). Whether this 5-HTTLPR-related sensitivity to positive experiences affects positive outcomes remains unknown. As noted by Belsky and Pluess (2009), this restricted range of dependent measures hinders understanding of the mechanisms by which the 5-HTTLPR influences psychological processes. Studies assessing positive and negative outcomes are therefore needed to clarify whether the 5-HTTLPR is only a marker of vulnerability for psychopathology or a general marker for sensitivity to life experience.

Because the effects of the 5-HTTLPR are particularly likely to operate in the social domain (Way & Gurbaxani, 2008), the transmission of emotion between interacting spouses represents a promising paradigm for evaluating the effects of the 5-HTTLPR upon emotional sensitivity. Emotions are signals that guide social interactions, providing individuals with information about their partners' motives (Keltner & Haidt, 2001). Affect transmission reflects the degree to which a person is sensitive to partner emotions and responds to those with feelings according to the signals perceived: Positive affect signals benevolence and is likely reciprocated, anger and hostility signal threat and may elicit anxiety or angry resistance. With respect to other emotions, however, the response may be complementary instead (e.g., Van Kleef, 2008). For example, as a potential signal of weakness, anxiety may reduce aggression and foster positive emotions in a caring partner.

In this study, spouses reported their positive and negative affect before and after laboratory-based interactions. With statistical models that adjusted for the interdependence between spouses, we (a) used partner affect ratings before the interactions as predictors of changes in the mate's affect ratings and (b) examined whether any such effects would be stronger among S-allele carriers (including assessment of the polymorphism rs25531, which lies upstream of the 5-HTTLPR (Wendland et al., 2006) and may modulate its effects on serotonin transporter gene expression (Hu et al., 2006)). We sought to discriminate between two interpretations of the effects of the 5-HTTLPR: the possibility that S-allele carriers were primarily sensitive to partner negative affect, versus the possibility that this sensitivity included positive as well as negative partner emotion, consistent with theories of serotonin as a modulator of stimulus reactivity (Spoont, 1992; Tops, Russo, Boksem, & Tucker, 2009).

Method

Participants

Participants were 76 couples recruited from marriage licenses in Los Angeles County between May 1993 and January 1994 to participate in a longitudinal study of marriage, and who were still participating in the study after 12 years of marriage (original N = 172 couples). Men averaged 27.9 years of age at the first assessment, SD = 4.0; wives averaged 26.4 years, SD = 3.7; 67% were Caucasian, 13% Hispanic, 12% Asian-American, 4% African-American, 4% other.

Procedure

Three times over the first 8 years of marriage (at 6 and 18 months after the wedding, and 8 years later), couples participated in four 10-minute lab-based interaction tasks, for a total of 120 minutes of interaction. In two interactions, couples discussed a topic of disagreement in their relationship, with each spouse bringing up one marital concern. In two interactions designed to elicit support, one partner brought up a personal issue that he/she wanted to change while the other was instructed to respond in whatever way she/he ordinarily would if this topic came up; roles were reversed in a second conversation (see Pasch & Bradbury, 1998).

Positive and Negative Affect

Immediately before and after each interaction, spouses independently completed items adapted from the Positive and Negative Affect Scale (Watson, Clark, & Tellegen, 1988). Positive affect was assessed with 3 items (feeling enthusiastic, excited, interested). Negative affect was assessed with 7 items. As in prior work (Schoebi, 2008), we differentiated between negative affect signaling weakness or submission (nervous, afraid, scared, jittery) and negative affect signaling dominance (irritable, hostile, upset). Items were rated on 5-point scales (1= not at all, 5 = very much), and an average score was computed for each scale to reflect positive affect (PA), negative dominant affect (NDA), and negative submissive (NSA) affect. Cronbach's alphas exceeded .69 at each assessment (*Mdn*= .82).

Genotyping

DNA was collected from saliva and extracted according to manufacturer recommendations (DNA Genotek). All samples were genotyped for the 5-HTTLPR using the protocol described in Way and Taylor (2010) as well as that described in Anchordoquy et al. (2003). The latter was used for phase-certain genotyping of rs25531, which used 4μl of PCR reaction product digested with MspI (4 units; New England Biolabs, Ipswhich, MA) in a 10μl reaction assay with 1x NEB Buffer 4 at 37°C for 3 hrs, 65°C for 20 min and held at 4°C. Four μl of restriction enzyme solution was analyzed on an ABI 3730 DNA Analyzer (Applied Biosystems, Carlsbad, California). From 163 saliva samples collected from 82 couples, thirteen could not be genotyped for the 5-HTTLPR (4 samples) or the rs25531 (9 samples), leaving 150 genotyped samples from 76 couples. For two of these 76 couples, only the man's genotype data was available.

Data Analysis

To assess the potential role of the 5-HTTLPR/rs25531 haplotype, the G-allele in the presence of the L-allele (Lg) was defined as functionally equivalent to the S-allele, according to Hu et al. (2006). Thus, SaSa (n=36), LgLg (n=2), SaLg (n=10) SgSa (n=1), SaLa (n=65), LgLa (n=10) genotypes were scored as S′ carriers and LaLa genotypes (n=26) were scored as L′/L′. The allele distributions of the 5-HTTLPR (p=.89) and rs25531 (p=. 19) did not deviate from Hardy-Weinberg equilibrium (exact test in Haploview 3.32; Barrett, Friy, Maller, & Daly, 2005). Analyses were conducted with both coding schemes to allow assessment of the potential contribution of rs25531. Hypotheses were tested using dyadic multilevel models to account for nonindependence (multiple interactions per person and couple), using the multivariate application of the MLwiN software and a two-tailed significance level of .05. Descriptives of affect ratings are shown in Table 1. Within-person correlations between affect ratings were moderate (r <.52).

We centered predictors at each person's mean to model within-couple affect contingencies. To test sensitivity to the partners' PA, we used a cross-lagged design where post-interaction affect reports were predicted by the individual's own, and by the partner's pre-interaction

PA (cf. Kenny & Cook, 1999). The level-1 equation (1) for positive affect of one spouse can be written as:

$$\text{POST PA}_{ij}=\beta_0(\text{intercept})+\beta_1(\text{PA})_{ij}+\beta_2(\text{Partner PA})_{ij}+r_{ij} \quad (1)$$

In this equation, β_2 reflects the extent to which the partner's pre-interaction PA is associated with fluctuations in PA. For the prediction of negative affect, we used a similar strategy, with the exception that we simultaneously used NDA and NSA as predictors, to adjust for covariation between NDA and NSA.

We examined genotype effects using dummy variables to contrast S-allele carriers from L/L individuals. Interactions of these predictors with the level-1 predictors capture the extent to which the coefficients of S-allele carriers differ from those of the L/L genotype and, thus, the extent to which genotype moderates sensitivity to partner affect.

Results

The results suggested stability in PA during the interactions (husbands: β=.474, p<.001; wives: β=.502, p<.001), and the partner's prior PA predicted the wives' (β=.087, p=.015) and the husbands' (β=.105, p=.001) post-interaction PA. Negative affect ratings were similarly stable during the interactions (husbands NDA: β=.402, p<.001, wives NDA: β=.387, p<.001; husbands NSA: β=.346, p<.001, wives NSA: β=.236, p<.001). Husbands' NSA predicted drops in their wives' NSA (β=−.067, p=.047), but the equivalent effect for wives was nonsignificant (β=−.041, p=.281). Effects for husbands' NSA predicting wives' NDA (β=−.057, p=.092), and for wives' NSA predicting husbands' NDA (β=−.049, p=.114) were nonsignificant. Partner NDA did not predict subsequent NDA (husbands: β=.067, p=.136; wives: β =−.005, p>.5), or NSA (husbands: β =−.025, p>.5; wives: β =−.070, p=.139). Model comparisons yielded no reliable sex differences when predicting PA (χ^2 (3) =.39, p> .5), NDA (χ^2 (5) =1.82, p>.5), or NSA (χ^2 (5) = 9.13, p>.1). We therefore report only sex-constrained models.

Table 2 shows the partner effects of PA, NDA and NSA, as moderated by the 5-HTTLPR genotype. Results indicate that genotype moderated sensitivity to partner PA. We found no significant sensitivity to PA for L/L individuals (ES =−.014; standardized coefficient based on pooled within-person variance), but PA changes in spouses with the S-allele was significantly associated with the partner's pre-interaction PA (ES =.102). Similarly, when data were reanalyzed to account for variation at rs25531, PA change in S′ individuals (ES =.082), but not L′/L′ individuals (ES =.001) were significantly associated with their partner's pre-interaction PA. In this reanalysis, however, moderation was not significant (p= .11), and the model fit was poorer (χ^2 (1) = 4.69, p=.030).

We found no differences in effects of the partner's NDA between genotypes. Sensitivity to the partner's NSA, however, differed significantly between L/L and S-allele carriers with respect to NDA. S-allele carriers' changes in NDA (ES =−.090) were significantly associated with the partner's pre-interaction NSA, but there was no such association for L/L spouses (NDA: ES =.057). Therefore, when their partner reported anxiety before the interaction, S-allele carriers dropped in irritability. When assessed as a function of the 5-HTTLPR/rs25531 haplotype, sensitivity to the partner's NSA differed significantly regarding NDA, and marginally regarding NSA. S′ individuals' changes in NDA (ES =−.067) were associated with the partner's pre-interaction NSA, whereas a nonsignificant association resulted for L′/L′ spouses (ES =.079). This model fit the data marginally worse (χ^2 (1) = 3.46, p=.063).

Exploratory analyses suggested no significant effects of the partner's genotype or interactions between spouses' genotypes. Testing contrasts for S/L individuals suggested no significant differences between S/S and S/L individuals (also true for S′/S′ and S′/L′ individuals), nor did discussion topic (conflict vs. support) moderate changes in affect. To control for potential population stratification artifacts, ethnicity was tested as Level-2 covariate; no significant effects emerged and results did not change appreciably.

Discussion

These findings are consistent with the view that 5HTTLPR genotype influences sensitivity to the partner's positive and negative emotions during marital interactions. S-allele carriers were more sensitive to their partner's positive affect than were L/L individuals. This finding corroborates research suggesting that the 5HTTLPR moderates sensitivity to positive stimuli (Belsky & Pluess, 2009), especially in the social domain (Way & Taylor, 2010). There was also a 5-HTTLPR-related difference in sensitivity to negative affect signaling weakness or submission, with significant effects for S-allele carriers but not for L/L individuals. These results extend prior work on the transmission of emotion between intimate partners, and they suggest that the greater sensitivity to partner emotion demonstrated by S-allele carriers is not specific to positive or negative affect. Reanalysis of the data to include the rs25531 polymorphism led to qualitatively similar results. The significance of the moderation was reduced, though, presumably due to the smaller sample size within the L′/L′ group.

Our findings indicate that higher pre-interaction levels of NSA (i.e., higher anxiety/nervousness) in partners of S-allele carriers resulted in *lower* NSA and NDA for those carriers. These inverse associations may provide insight into the interpersonal mechanisms by which the 5-HTTLPR influences emotional sensitivity. A process involving emotional contagion would lead to changes in the same direction (Hatfield, Cacioppo, & Rapson, 1993) and could only explain the contingencies we observed for PA. Such a mechanism, however, cannot account for associations with partner NSA. Rather, the NSA effects are more consistent with an emotional complementarity explanation, whereby, in S-allele carriers, the partner's signals evoke corresponding feelings, presumably of benevolent quality, leaving them less hostile and anxious in response.

How might the 5-HTTLPR affect such processes? A contributing factor to differences in affect transmission might be greater attunement to affective signals in S-allele carriers. In studies of attention, S-allele carriers exhibit an attentional bias to anxious (Thomason et al., 2010) and angry faces (Pérez-Edgar, et al., 2010). Evidence for a bias toward positive stimuli is more equivocal, though eye-tracking data show that S-allele carriers allocate increased attention to positive images (Beevers, Ellis, Wells, & McGeary, 2010).

5-HTTLPR-related differential emotional responses could also result from greater responding of S-allele carriers to the same affective cues. Indeed, a meta-analysis of functional neuroimaging studies found greater amygdala reactivity to emotional faces and stimuli in S-allele carriers than L/L individuals (Munafò, Brown, & Hariri, 2008).

Although the assessment of within-person differences in responsivity to affective signals is a particularly sensitive design, compared to the more common between-person comparisons (e.g., Caspi, et al., 2003), the current analyses did not detect 5-HTTLPR-related sensitivity to aggression or dominance (NDA). This may indicate that the 5-HTTLPR is more associated with sensitivity to NSA than NDA. More likely, however, is that the current paradigm is not well-suited for the assessment of feelings such as hostility; such hot feelings might arise during interactions rather than in the pre-interaction phase examined here. It is

also possible that the current sample is biased toward sensitivity for benevolence, as only couples who were still married several years after the wedding provided data.

In conclusion, genetic variation may affect the magnitude of emotional interdependence between spouses. The emotions a spouse feels following his or her marital conversations are predicted in part by the emotions of the partner prior to those conversations, and the magnitude of this prediction is greatest for S-allele carriers. The 5-HTTLPR appears to affect sensitivity to positive and negative affect, and our finding that more negative pre-interaction emotions by one spouse covary with less negative post-interaction emotion by the partner underscores the importance of assessing the social context in order to understand the psychological effects of the 5-HTTLPR.

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Table 1

Descriptive statistics of the study variables: Positive and negative affect reported before and after the interactions

	5HTTLPR			
	S		L/L	
Variable	***M***	***SD***	***M***	***SD***
Husbands	*n* = 58		*n* = 18	
PA_pre	3.36	0.87	3.36	0.83
NDA_pre	1.32	0.56	1.38	0.59
NSA_pre	1.57	0.78	1.71	0.93
PA_post	3.45	0.96	3.27	0.90
NDA_post	1.34	0.59	1.41	0.64
NSA_post	1.42	0.66	1.60	0.81
Wives	*n* = 54		*n* = 20	
PA_pre	3.38	0.89	3.12	0.92
NDA_pre	1.29	0.49	1.40	0.61
NSA_pre	1.50	0.71	1.56	0.84
PA_post	3.46	0.97	3.18	1.03
NDA_post	1.33	0.60	1.28	0.51
NSA_post	1.31	0.56	1.33	0.59

Note. PA = positive affect, NDA = negative dominant affect, NSA = negative submissive affect. S = short allele carriers, L/L = long allele homozygotes. Coefficients reflect mean scores across the three laboratory sessions. No significant differences existed in spouses' affect ratings between genotypes or across lab sessions, no systematic trends in affect ratings existed across time, and genotypes were not associated with individuals' trends across the laboratory sessions.

Table 2

5-HTTLPR genotype interacting with pre-interaction partner affect to predict post-interaction affect

5-HTTLPR genotype	Coefficients			Genotype difference (LL vs. S carriers)	
	β	*SE*	*ES*	γ	*SE*
Partner PA predicting PA					
S	.113***	.031	.102	−.127*	.056
L/L	−.014	.053	−.028		
Partner NDA predicting NDA					
S	.035	.038	.031	−.035	.064
L/L	.000	.056	.000		
Partner NSA predicting NDA					
S	−.070**	.026	−.090	.114*	.051
L/L	.044	.051	.057		
Partner NDA predicting NSA					
S	−.051	.041	−.033	.038	.071
L/L	−.047	.051	−.038		
Partner NSA predicting NSA					
S	−.065*	.028	−.075	.078	.056
L/L	.058	.057	.066		

Note. ES = Effect size; standardized coefficient based on pooled within-person variance parameters. PA = positive affect, NDA = negative dominant affect, NSA = negative submissive affect, S = short allele carriers, L/L = long allele homozygotes.

* $p < .05$;

** $p < .01$;

*** $p < .001$.