Emotion appraisal is modulated by the genetic polymorphism of the serotonin transporter

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Abstract We investigated the relationship between the genetic polymorphism of the promoter of the serotonin transporter gene (SLC64A) and emotion appraisal profiles for fear, sadness, and joy in 114 healthy volunteers. Results revealed that carriers of the s-allele achieved higher scores than non-carriers for unpleasantness and goal-hindrance but scored lower for coping ability in the case of fear and sadness. There were no such differences in the case of joy. These results indicate that the s-allele of the serotonin transporter gene is associated with a vulnerable cognitive style related to the appraisal of negative emotions.

Keywords Serotonin transporter · Polymorphism · Genetics · Emotion · Appraisal

Introduction

Various lines of evidence suggest that an allelic variation of the upstream regulatory region of the serotonin transporter gene (SLC64A) is related to anxiety- and depression-related traits, which may indicate dysfunctional emotion regulation (Canli and Lesch 2007). The short (s) variant of the gene produces less transporter protein than the long (l) variant and is associated with higher anxiety (neuroticism) (Lesch et al. 1996), more pronounced self-reported depressive symptoms (Gonda et al. 2005), and increased activation of the amygdala during the viewing of fearful facial expressions (Hariri et al. 2002). However, meta-analytic evidence provided a less consistent picture regarding the association between SLC64A polymorphism and anxiety-related personality traits (Schinka et al. 2004; Sen et al. 2004; Munafo et al. 2005).

One of the most important aspects of emotion regulation is how we interpret and reflect to the experience of an emotion (Scherer 1997). In this respect, there is a considerable variance in the population. For example, one may appraise sadness more unpleasant, less controllable, and excessively demanding than others. Despite its importance, to date, no studies have been investigated the effect of the allelic variation of the SLC64A gene on emotion appraisal.

In this study, we genotyped the SLC64A gene of 114 healthy individuals recruited from the community and compared emotion appraisal profiles in the case of negative (fear and sadness) and positive (joy) emotions as a function of serotonin transporter gene allelic variations.

Methods

The demographic details of the participants are shown in Table 1. The study has been approved by institutional ethics committee and has been performed in accordance with the Declaration of Helsinki. All persons gave their informed consent. The Mini-International Neuropsychiatric Interview (Sheehan et al. 1998) was used to exclude psychiatric disorders. Genotyping was performed as described previously (Lesch et al. 1996). Emotion appraisal was assessed using Scherer’s appraisal questionnaire (Scherer 1997). The participant was asked to recall a situation in which he/she had recently experienced a strong emotion. The questionnaire for fear, sadness, and joy consisted of four parts: (1) situation description, (2) subjective feeling state, (3) physiological symptoms and expressive reactions, and (4) appraisal. Questions were posed on the following
types of emotion appraisal: novelty/expectation (“Did you expect this situation to occur?”), pleasantness (“Did you find the event itself pleasant or unpleasant?”), goal-conduciveness (“How important was the event for your goals, needs, or desires at the time it happened? Did it help or hinder you to follow your plans or achieve your aims?”), fairness (“Would you say that the situation or event that caused your emotion was unjust or unfair?”), responsibility/causation (“Who do you think was responsible for the event in the first place?”), coping ability (“How did you evaluate your behavior to act on or to cope with the event and its consequences when you were first confronted with this situation?”), morality, (“If the event was caused by your own or someone else’s behavior, would this behavior itself be judged as improper or immoral by your acquaintances?”), and relationship to self-concept (“How did this event affect your feelings about yourself, such as your self-esteem or your self-confidence?”).

Ratings for each emotion appraisal were converted to Z-scores. Z-scores were the deviation of a value for a specific emotion from the mean over all emotions investigated, as described by Scherer (1997). The group mean of these Z-scores was calculated for fear, sadness, and joy. The general linear models panel of the STATISTICA 7.0 software (StatSoft Inc., Tulsa) was used to conduct repeated measures analyses of variance (ANOVAs) and post-hoc Tukey HSD tests.

Results

S-carriers included participants with s/s (n = 27) and s/l (n = 32) genotype, whereas non-carriers included participants with l/l (n = 35) genotype. S-carriers and non-carriers did not differ in age, education, and socioeconomic status (t test, P > 0.1) (Table 1). This distribution of the serotonin transporter genotypes did not deviate from the Hardy–Weinberg equilibrium (Chi-square test, P > 0.05).

First, we investigated the effect of gender. This ANOVA revealed no significant main effect of gender (F < 1, P > 0.1), and, therefore, data from male and female participants were collapsed in further analysis. The genotype (s-carriers vs. non-carriers) by emotion and by appraisal type three-way ANOVA revealed significant main effects of emotion [F(2,224) = 365.21, P < 0.0001, η² = 0.77, power = 1.0] and appraisal [F(7,784) = 126.61, P < 0.0001, η² = 0.53, power = 1.0]. There was a two-way interaction between emotion and appraisal [F(14,1,586) = 446.92, P < 0.0001, η² = 0.80, power = 1.0]. Most critically, there was a two-way interaction between genotype and appraisal [F(7,784) = 7.32, P < 0.0001, η² = 0.06, power = 0.99], and a three-way interaction between genotype, emotion, and appraisal [F(14,1,568) = 2.69, P < 0.001, η² = 0.02, power = 0.99].

In the case of fear, there was a significant two-way interaction between genotype and appraisal [F(7,784) = 11.41, P < 0.0001, η² = 0.09, power = 1.0]. The post-hoc tests revealed that s-carriers achieved higher scores than non-carriers for unpleasantness (P < 0.0001) and goal-hindrance (P < 0.01). In contrast, s-carriers achieved lower scores than non-carriers for coping (P < 0.0001) (Fig. 1). In the case of sadness, a similar two-way interaction was found between genotype and appraisal [F(7,784) = 6.68, P < 0.0001, η² = 0.06, power = 0.99]. The post-hoc tests revealed that s-carriers achieved higher scores than non-carriers for unpleasantness (P < 0.05) and goal-hindrance (P < 0.01). S-carriers achieved lower scores than non-carriers for coping (P < 0.05) (Fig. 1). Finally, in the case of joy, there was no interaction between genotype and appraisal (F < 1, P > 0.1) (Fig. 1).

Discussion

The results of this study are consistent with previous findings suggesting that the s-variant of the serotonin transporter gene is associated with anxiety- and depression-related traits (Canli and Lesch 2007). Our data further clarifies these findings, demonstrating that participants with the s-variant experience negative emotions more unpleasant, more influential, and disruptive on personal goals, and feel less able to cope with these emotions. The possible explanation of these findings is that s-allele carriers experience negative events differently than non-carriers, or, alternatively, they recall those events differently. It was remarkable that the effect of serotonin transporter polymorphism was confined to the appraisal of negative emotions (fear and sadness) and to specific appraisal types (unpleasantness, goal-hindrance, and coping). Our finding demonstrating less subjective coping ability in s-carriers is consistent with the results of Wilhelm et al. (2007) who found that the s-variant of the serotonin transporter gene is associated with the use of fewer problem-solving strategies and less efficient coping with stressful situations. In
addition, several previous studies highlighted the association between serotonin transporter polymorphism and negative emotions (Caspi et al. 2003; Kendler et al. 2005; Wilhelm et al. 2006).

The possible neuroanatomical substrate of emotion regulation and appraisal may include the interaction between cingulate cortex and amygdala (Kalisch et al. 2006), which is significantly modulated by the polymorphism of the serotonin transporter gene (Pezawas et al. 2005). Specifically, s-allele carriers had smaller gray matter volume in brain areas critical for processing of negative emotion (perigenual cingulate cortex and amygdala). The functional connectivity between these brain regions is essential for the extinction of negative affect. It is interesting that s-allele carriers showed relative uncoupling of this circuit, which inversely predicted almost 30% of variation in temperamental anxiety (Pezawas et al. 2005).

The most important limitation of the present study is similar to that of others investigating the effect of single gene polymorphisms on complex and continuously distributed traits. In such cases, the influence of a single gene is at best modest (Plomin et al. 1994). However, our data revealed a similar pattern of appraisal for fear and sadness as a function of serotonin transporter polymorphism, which suggests that differences between s-carriers and non-carriers are consistent and are not due to chance. In addition, despite the relatively small sample size, the analysis demonstrated high statistical power.

The effect of adverse life events on emotion appraisal and its interaction with serotonin transporter polymorphism was not assessed in the present study. It is an especially important question for future studies given that s-carriers show increased sensitivity to stress and adverse life events (Caspi et al. 2003; Kendler et al. 2005; Wilhelm et al. 2006), which may affect the appraisal of negative emotions.

In conclusion, our data indicate a significant relationship between genetic traits and cognitive style that is involved in emotion appraisal. These mechanisms may shed new light on the neurobiology of emotion regulation and its disorders.

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References


