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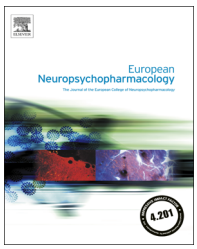

Using a partner's facial emotion to elucidate social dominance motivation induced by an SSRI

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Abstract
Previous studies independently showed that acute treatment with a selective serotonin reuptake inhibitor (SSRI) enhanced happy face recognition, and dominance behaviors which might reflect enhancement of reward sensitivity. The present study aimed to determine whether such a mechanism would be related to social resource acquisition induced by an SSRI. Forty healthy subjects were recruited for the experiment. A randomized, double-blind, placebo-controlled crossover nested within confederate type (happy, fearful, or sad) trial of a single-dose of 10 mg escitalopram versus placebo was conducted with a two-week washout period. In each of the treatment groups, the subjects interacted socially with one of the three types of confederate in a waiting room for 3-minute. Then, they went to an individual laboratory and were led to believe that they played the Mixed-motive game with the confederate. The game measures punitive/cooperative behaviors by how participants allocate higher/lower game scores to the confederate and communicate cooperation/ingratiation/helplessness/sadness/blaming/extrapunitive, messages to the confederate. Significant treatment-by-confederate type interactions were observed through game score distributions and ingratiation messages to the confederate and attentive eye gaze. In the happy confederate condition, escitalopram increased ingratiation messages and lowered points awarded to the confederate. In the fearful confederate condition, escitalopram increased ingratiation messages and reduced time spent looking away from the confederate. No changes in these measures were found in the sad confederate condition. Therefore acute escitalopram treatment enhances

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reward sensitivity to the facial emotions of social partners which in turn increases social resource acquisition and social dominance towards happy but not fearful social partners.

1. Introduction

Serotonin transmission is related to many different behavioral changes including reward sensitivity (Tanaka et al., 2007) and social dominance (Gilbert, 2006). In a series of psychopharmacological and genetic studies in humans, convergent evidence suggests that serotonin transmission influences sensitivity to reward. Depletion of serotonin by tryptophan depletion resulted in impairment on selecting the option for financial reward over pain in a game and the neural representation of reward outcome value (Seymour et al., 2012). In contrast, enhancement of serotonin transmission resulted in an increase in the neural pathway related to reward (Tanaka et al., 2007). Patients diagnosed with Major Depressive Disorder with the 5-HTTLPR ll allele, reflecting higher serotonin transmission, were more likely to select the advantageous deck in the computerized Iowa gambling task than those without this allele (Must et al., 2007). This evidence suggests that serotonin plays a crucial role in reward sensitivity. However, the effects of serotonin on reward sensitivity are complicated. McCabe et al. (2010) showed that 7 days treatment with citalopram could decrease reward sensitivity to a primary reinforcer (i.e. chocolate smell) and decrease reactivity to an aversive stimuli (i.e. Mold strawberry smell). Similarly, Alber et al. (2012) showed that 7 days paroxetine treatment down-regulated the responsiveness of the primary reward system. They suggested that such down-regulation to primary reinforcer signals allows individuals to exercise cognitive control to choose the immediate reward and reduce risk-taking behaviors. Using the probabilistic reversal learning task, Bari et al. (2010) revealed that rats that received an acute high dose of citalopram showed reduced sensitivity to negative feedback by selecting less lose-shift choices in the game. Those that received repeated doses of citalopram showed increased reward sensitivity by selecting more win-stay choices in the game. These findings were consistent with the proposal that long term treatment with a serotonergic antidepressant helps patients to re-learn from their environment to be less sensitive to punishment and more sensitive to reward signals (Harmer, 2008) thus developing their ability to exercise control to select the best possible choices within a given situation.

According to the social rank theory proposed by Price and Sloman (1987), animals that experience involuntary submission after social defeat would show biochemical and behaviors resembling depressive symptoms in humans including lower serotonergic transmission, social motivation, and affiliative behavior and higher levels of stress and sleeping problems. SSRI treatment was found to reverse depressive symptoms in these animals (Raleigh et al., 1991). In line with these preclinical findings, Tse and Bond (2002a) showed that two weeks of citalopram treatment enhanced expression of dominant social behaviors on a number of different measures (e.g. eye gaze behavior and communication). Moskowitz et al. (2001) also reported that 12 days' treatment with 1 g tryptophan enhanced dominance in friendship as reported by participants' friends. This evidence suggests that increased social dominance could also be a potential therapeutic mechanism for serotonergic antidepressants. Terburg and van Honk (2013) suggest that social dominance behaviors are used in pursuit of something desirable. Socially dominant individuals acquire tactics (e.g. coercive or pro-social behavior) to exert control over resources in the belief that on one hand, these tactics promote their ability to acquire resources and on the other hand, they minimize the personal cost of conflict (Hawley, 1999). Thus, before confronting another person to compete for social status, the individual needs to know their relative ability to compete with that person and be able to read the social signals indicating a less retaliative person. Such knowledge might have been gained before the engagement in competition.

Detection of facial expressions has evolved to serve as a cue to understand the emotional state of others. These cues help people to regulate their behavior to minimize costly conflict and maximize collection of resources. Smile is an important non-verbal signal indicating friendliness. Strangers displaying smile, regardless of their other nonverbal behavior, were rated as more approachable (Miles, 2009). The happy face of a social partner also suggests that they are more willing to share their resources with others (Scharlemann et al., 2001). Therefore, sensitivity to detect happy facial expression helps one to identify a potential "donor" of resources. Fearful facial expression serves as a signal of distress. This would call for the prosocial behavior of observers and increase affiliative behaviors (Marsh and Ambady, 2007). A single dose (not the chronic dose) of SSRI was found to increase sensitivity to detect fearful facial expression (Harmer et al., 2003). Therefore, it is conceivable to postulate that a single dose of SSRI would also increase affiliative behavior to people expressing fearful facial emotion.

The computerized facial emotion recognition task provides a paradigm to detect the effects of SSRIs on the early processes of social interaction, namely social perception. However, it does not tell us anything about social behavior i.e. how participants would behave after viewing a happy face. However, the laboratory task using the mixed motive game and confederates to study social behavior (Tse and Bond, 2002a) could incorporate the facial emotion of the confederate and the social behavior of participants. Examining the interaction between the facial emotion of a confederate and the social behavior expressed by participants should help us to understand the relationship between the findings from facial emotion recognition and social behavior studies of SSRIs and close this knowledge gap.