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Research report

Differential effects of escitalopram challenge on disgust processing in obsessive–compulsive disorder

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ABSTRACT

Introduction: Literature on the ability of patients with obsessive–compulsive disorder (OCD) to recognize *static* facial expressions of disgust is not consistent. We aimed to investigate whether OCD is associated with deficits in the recognition of disgust in a *dynamic* task, and if so, whether the acute administration of the selective serotonin reuptake inhibitor (SSRI) escitalopram would result in the normalization of such deficits.
Methods: OCD patients ($n = 20$) and matched healthy controls ($n = 20$) received a single dose of escitalopram 20 mg on one day, and a single dose of placebo on another day, in randomized order, under double-blind conditions. Accuracy (i.e. the percentage of correct answers) and sensitivity to disgust stimuli (defined as the lowest level of emotional intensity expressed on the photo image after which no errors were made in the recognition of disgust for subsequent trials of increasing intensity) were compared in OCD patients and controls, with a repeated measures analysis of variance using a mixed model approach.
Results: On placebo, the accuracy of, and sensitivity to, disgust stimuli were similar across groups. OCD patients had more accurate and more sensitive recognition of disgust after acute SSRI administration than after placebo, while controls had less accurate recognition and less sensitive recognition of disgust after acute SSRI administration than after placebo.
Conclusions: The use of a dynamic facial recognition task demonstrated altered responses to disgust in OCD patients compared to healthy controls after a pharmacological challenge with escitalopram. These findings suggest that the serotonergic system plays a role in disgust recognition.

1. Introduction

Facial expressions convey important emotional information and their accurate recognition is fundamental to successful social interaction. Deficits in the ability to recognize facial expressions accurately have been linked to different forms of psychopathology, although the specific nature and underpinnings of these deficits is still poorly understood [1]. Some studies suggest that impaired recognition of facial expressions is common to a number of different disorders (e.g. depression [2–4]) and schizophrenia (e.g. [5,6]). However, other work suggests that particular psychiatric disorders are characterized by specific deficits in recognizing facial expressions (e.g. [7,8]).

Although obsessive–compulsive disorder (OCD) is classified as an anxiety disorder, many intrusive thoughts (obsessions) and repetitive, ritualistic actions (compulsions), may also involve the emotion of disgust [9,10]. There is evidence to suggest that OCD may be characterized by specific impairment in the recognition of disgusted facial expressions [11]. Recent work has indicated that less accurate recognition of disgust in OCD is associated with increased OCD severity [1,12] and increased functional impairment [1]. Studies in non-clinical samples (e.g. [13,14]) have also found an association between OC symptoms and disgust sensitivity.

Nevertheless, not all data on disgust recognition in OCD are consistent. Some researchers have suggested that changes may occur only amongst a subset of individuals with OCD—for example, in those with contamination concerns [15–17]. Furthermore, there are several studies that have failed to find a deficit in disgust

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recognition in OCD (e.g. [18,19]). Notably, the bulk of this work has relied on static facial expressions, rather than on dynamic tasks which may provide additional information in terms of the accuracy, sensitivity, and timing of responses. Also, most of these studies either included OCD patients with clinically significant comorbidity (e.g. [1]) or did not mention comorbidity at all (e.g. [11]) which may have contributed to the inconsistencies [18,19].

The underlying neurobiology of disgust recognition is not well characterized [10]. Several lines of evidence point to the involvement of the serotonergic system in OCD. For example, a small number of neurobiological studies have indicated that rare functional abnormalities in specific genes in the serotonin system are associated with OCD (e.g. [20,21]), while a large number of treatment studies have demonstrated that OCD responds fairly selectively to serotonin reuptake inhibitors (SRIs) (e.g. [22–24]). It may therefore be hypothesized that deficits in disgust recognition in OCD might respond to administration of an SRI. Although OCD treatment requires chronic treatment, single doses of antidepressant medication have been shown to alter emotional processing [25,26].

The current study thus aimed to investigate whether OCD patients without comorbid psychiatric disorders showed deficits compared to healthy controls in the recognition of disgust in a dynamic task and if so, whether single administration of the SRI escitalopram (which is highly selective for the serotonin system (e.g. [27]), would result in the normalization of such deficits. The emotion recognition task was completed 3 h after administration of the pharmaceutical agent (a time period seen as ideal to ensure optimal plasma levels [28]). We also investigated the moderating effect of a number of variables, i.e. affect (i.e. levels of depressive and anxiety symptoms) and OCD severity, on disgust recognition.

2. Materials and methods

2.1. Participants

Participants with OCD were recruited from a wide range of sources (e.g. the OCD Association of South Africa, community based primary care practitioners, and psychiatrists and psychologists). Gender-, age- and IQ-matched controls were recruited from the community and our university campus through media advertisements. Participants were included if they were between the ages of 18 and 65 years.

Patients were included if they had a primary diagnosis of OCD and no other comorbid psychiatric conditions of clinical significance (i.e. some OCD patients with comorbid specific phobia ($n = 2$) were allowed to participate if this did not significantly interfere with their daily functioning) and if they were either psychotropic medication free or on psychotropic medication that was (1) limited to a single psychotropic medication from the selective SRI-class of agents, (2) administered at a steady dose that was not higher than the optimal dose for OCD for the particular agent (e.g. 60 mg of fluoxetine), (3) taken for at least 2 months (8 weeks), and (4) that was stabilized according to the treating psychiatrist. Referring clinicians were contacted to help establish, where possible, a longitudinal expert assessment and diagnosis. Controls were included if they had no current DSM-IV disorder.

The institutional review board of the University of Stellenbosch (South Africa) approved the protocol, and all participants provided written informed consent after being presented with a complete description of the study.

2.2. Procedures

2.2.1. Screening and clinical assessment

Prospective participants were screened telephonically and subsequently interviewed by a clinical psychologist. Demographic data were collected. The Mini International Neuropsychiatric Interview Plus (MINI Plus) – version 5 [29], a structured diagnostic interview developed for DSM-IV and ICD-10 psychiatric disorders, was used for diagnostic purposes. All patients were also interviewed with the Structured Clinical Interview for Obsessive–Compulsive Spectrum Disorders (SCID-OCSD) to determine the presence of comorbid putative obsessive–compulsive spectrum disorders [30]. The assessment also addressed the presence/absence of tics (current and/or past). Patients with any psychiatric comorbidity were excluded from participation in the brain imaging component of the study.

Patients with a primary diagnosis of OCD were included if they were at least moderately symptomatic on the Yale–Brown Obsessive–Compulsive Severity Scale (YBOCS), i.e. if they had a YBOCS total score >16 [31]. The YBOCS is a clinician-rated 10-item measure of the severity of symptoms of OCD; each item is rated from 0 (no symptoms) to 4 (extreme symptoms), with a total range of 0–40 and separate subtotals for the severity of obsessions and compulsions. The scale has good inter-rater reliability, validity and a high degree of internal consistency among all items [32]. Controls were included if they had no significant current DSM-IV Axis I or II mental disorder or past history of any substance or alcohol abuse/dependency.

In addition to the MINI Plus, the Montgomery–Asberg Depression Rating Scale (MADRS) was used to ensure that persons with clinically depressed mood were excluded. The MADRS is a 10-item scale used to assess patients' mood, feelings of unease, sleeping pattern, appetite, ability to concentrate, ability to take initiative, emotional involvement, pessimism and zest for life [33]. Each item is rated on a scale of 0–6, with a total score ranging between 0 and 60. There is good evidence of the scale's reliability and validity [34]. Participants with a MADRS score >20 (i.e. possibly indicating clinical depression) were excluded from the study. The severity of participants' anxiety was assessed using the 14-item Hamilton Anxiety Rating Scale (HAM-A) [35]. Each item is scored on a scale of 0 (not present) to 4 (severe), with a total score range of 0–56; scores <17 suggest mild anxiety, 18–24 mild to moderate anxiety, and 25–30 moderate to severe anxiety.

The Wechsler Abbreviated Scale of Intelligence (WASI) [36], a valid screening measure of verbal, performance, and general intellectual ability (e.g. [37]) was employed in order to obtain an estimate of intelligence. The WASI is a battery consisting of 4 subtests (vocabulary, similarities, matrix reasoning and block designs) used to derive both verbal and performance quotients, as well as a global intelligence quotient (IQ).

2.2.2. Pharmacological challenge

Participants were administered a single dose of escitalopram (20 mg) or placebo, in randomized order, on the two separate testing days, approximately 1 week apart. The use of escitalopram as a pharmacological tool for exploring the role of serotonin in modulating normal emotional processing and in the pathophysiology of anxiety and depressive disorders has been suggested [38].

2.2.3. The emotion recognition task

A modified version of Montagne et al.'s [39] emotion recognition task was used; modifications include using a different set of facial stimuli (the sources of facial stimuli are described below), and reducing the number of emotions from the 6 primary emotions to 5 (i.e. angry, disgusted, fearful, happy or sad). Two components were assessed using the emotion recognition task, i.e. (1) the accuracy in recognizing disgust (i.e. the percentage of correct answers for each expression shown for all intensities), and (2) the sensitivity for disgust (defined as the lowest level of emotional intensity expressed on the photo image after which no errors were made in the recognition of disgust for subsequent trials of increasing intensity). The facial stimuli used in the emotion recognition task consisted of gray-scale images of the faces of two males and two female actors mimicking emotional and neutral expressions. Three of the faces were selected from the Directed Emotional Faces libraries [40], and one from the Ekman [41] library of facial stimuli. The faces were selected on the basis of mean ratings (9-point scale) by 12 subjects for each emotion. Winmorph, an interactive computer program devised by Murray and colleagues (Murray LM, Facial expression perception in different psychiatric groups, Unpublished doctoral dissertation, University of St Andrews, 1999) was used to create intermediate morphed images between a neutral face (0% emotion) and a extremely expressive facial expression (starting with 20% intensity levels and ending with a full-blown or 100% expression), in increments of 2% changes in intensity (Fig. 1a and b).

The tests were presented to each subject in a fixed order. Participants read through a set of instructions displayed on a computer screen asking them to identify the 5 emotional expressions as angry, disgusted, fearful, happy or sad. During the experiment, the participants were presented with images of actors' faces morphing from neutral to one of 9 different intensity levels (i.e. 20%, 30%, 40%, 50%, 60%, 70%, 80%, 90% and 100%). Blocks of trials were presented in increasing intensity, with each block containing faces for each combination of actors and emotions. Accordingly, each block consisted of a total of 20 trials, presented in random order, for a total of 180 trials for each session of the task. After the morphing sequence for each face was completed, the participants were required to identify the emotion expressed by pressing a number on the keyboard that corresponded to the order in which the five emotion labels appeared beneath the face. The next trial began following the participant's response. There were no time restrictions but participants were instructed to respond to each stimulus as fast as they could. The task was presented using the *E*-prime software package (Psychology Software Tools Inc., 2002). For the current investigation, our data analyses focused on responses to disgust stimuli only.

2.3. Data analyses

Performance on the emotion recognition task was analysed by means of a repeated measures analysis of variance using a mixed model approach (including a restricted maximum likelihood test, or REML). All data were analysed using Statistica 9.0 for Windows (Statsoft, Tulsa, Oklahoma). The following factors were included as grouping variables in the analyses: testing session (session 1 or 2), diagnosis (OCD or control), the order in which the pharmacological challenge was given (i.e. placebo on the first test day, and escitalopram on the second, or escitalopram on the first and placebo on the second test day), and intensity levels of the stimuli. In the total sample, gender and level of comorbid depressive symptoms were included

Fig. 1. Example of morphing during the emotion recognition task.

as covariates in the analyses. Based on the known association between depressed mood and OCD [42,43], and the very strong correlation found here between the totals of the MADRS and the HAM-A (Spearman $r = 0.81$, $p < 0.001$) in OCD and controls, only the MADRS-total was included as covariate. In the OCD subset, the severity of OCD (YBOCS-total) and depressive symptoms (MADRS-total) were also investigated. Second order interaction analyses included the interaction between diagnosis and pharmacological challenge, and diagnosis and intensity, as well as intensity and pharmacological challenge. Third order interactions (i.e. between intensity [from 0 to 100%], pharmacological challenge (placebo/control) and diagnosis (OCD/control) were also assessed. We subsequently repeated all of the above mentioned analyses across three groups, i.e. comparing OCD patients who were receiving treatment with a psychotropic medication (an SSRI) with those not on any psychotropic medication at the time of the assessments and controls.

We employed a 5% threshold as guideline for determining significant differences.

3. Results

3.1. Demographics and clinical characteristics

Demographic data are presented in Table 1. Twenty patients with a primary diagnosis of OCD ($n = 20$; 11 male, 9 female) and 20 gender-, age- and IQ-matched controls (9 male, 11 female) took part in the study.

Thirty seven ($n = 37$) patients with "possible" OCD and 25 controls were screened for participation. In both groups, reasons for exclusion were one or more of the following: comorbid psychiatric conditions, no OCD or mild OCD (i.e. YBOCS scores lower than 17), being left-handed or participant withdrawal due to time constraints or not being interested to proceed with participation anymore ($n = 4$). One of the OCD patients was diagnosed with comorbid major depressive disorder on the MINI Plus but was not excluded given that his MADRS-score was far below the cut-off score for exclusion (MADRS = 12). Another patient was diagnosed with comorbid specific phobia; however, since this condition was secondary to his OCD and not functionally impairing on a daily basis, it was decided to include him in the study as well. Within the control group, two participants had a current psychiatric disorder on the MINI Plus (i.e. specific phobia ($n = 1$) and premenstrual dysphoric disorder ($n = 1$)) but they were included given that these conditions were not present and functionally impairing on a daily basis.

Eleven out of the 20 OCD patients were receiving SSRI treatment at the time of study participation: 3 were on sertraline (150 mg, 125 mg, 300 mg), 5 on fluoxetine (2 on 20 mg, 2 on 40 mg and 1 on 60 mg), 1 on escitalopram (5 mg), 1 on citalopram (20 mg) and 1 OCD patient on paroxetine (40 mg). None of these participants received more than one drug concurrently at the time of the assessments. According to participant reports and feedback from treating psychiatrists, the treatment effects had stabilized for all of these participants. Of note, however, is that at the time of the screening interview, all patients were symptomatic enough in terms of their OCD to be included in the study (i.e. YBOCS total score >16).

The YBOCS- (total OCD severity) scores in the OCD group ranged from 17 to 32 (with a mean [SD] YBOCS-score of 23.0 [3.96]), suggesting moderate to severe OCD. Patients with OCD had significantly higher levels of depressive and anxiety symptoms (both $p < 0.001$) than controls (Table 1). Male and female patients did not differ significantly in terms of severity of OCD, or levels of depressive or anxiety symptoms. In the OCD group, depressive symptom scores were significantly positively correlated with OCD severity (Spearman $r = 0.6$, $p = 0.01$). There also was a tendency for the levels of anxiety to be positively correlated with OCD severity (Spearman $r = 0.4$, $p = 0.058$).

3.2. Performance on the emotion recognition task

(All figures presented here depict least squares (LS) means whereas the arithmetic means are presented in the text.)

3.2.1. Accuracy in recognizing disgust

In the total sample (OCD + controls), the accuracy of disgust recognition increased significantly from session 1 to session 2 ($F = 19.1$, $p < 0.001$). Increased accuracy in the total sample was significantly associated with faces presented at higher intensity levels ($F = 18.7$, $p < 0.001$).

There was a significant interaction effect between the intensity level at which faces was presented and gender in terms of the accuracy of disgust recognition. Specifically, although both genders were more accurate at higher intensity levels, females significantly

Table 1
Demographics and clinical characteristics of participants.

Variable	OCD patients ($N = 20$)	Controls ($N = 20$)	Statistics
Gender	9 female, 11 male	11 female, 9 male	$\chi^2 = 0.4$, $p = 0.5$
Age (mean [SD])	34.1 [11.0] years	34.75 [10.76] years	$t = -0.2$, $p = 0.9$
IQ (mean [SD])	117.3 [12.0]	119.1 [13.0]	$t = -0.4$, $p = 0.7$
MADRS total score	10.0 [5.0]	3.5 [3.1]	$t = 5.0$, $p < 0.001$
HAM-A total score	9.8 [4.3]	4.6 [3.8]	$t = 4.0$, $p < 0.001$

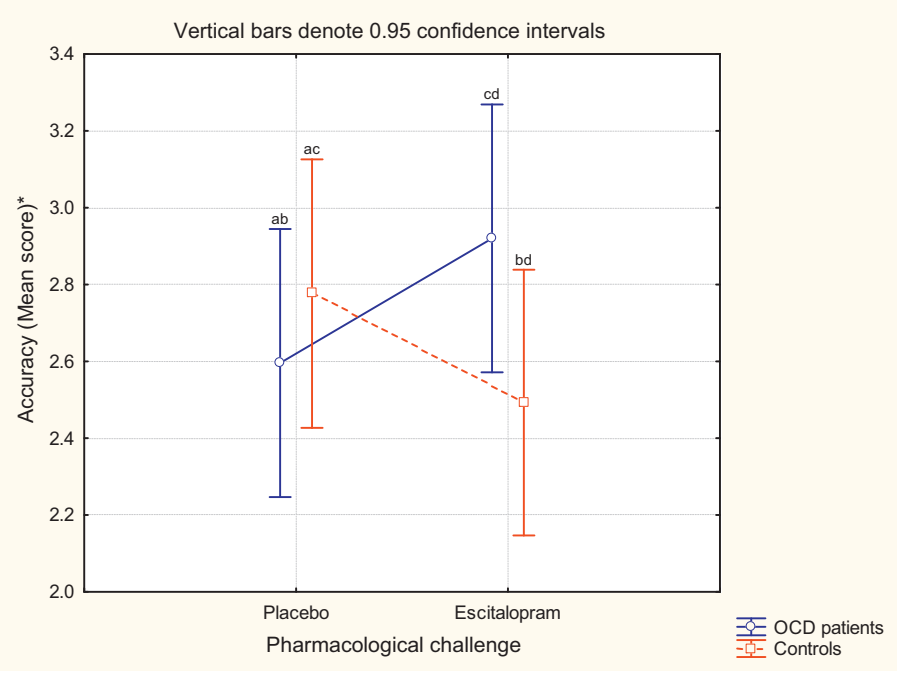

Fig. 2. Diagnostic group × Pharmacological challenge interaction in terms of the accuracy of disgust recognition. * The mean scores refer to the least squares (LS) means.

outperformed males ($F = 2.0$, $p < 0.05$). There also was a significant interaction between diagnostic group (i.e. controls/OCD patients) and pharmacological challenge (i.e. placebo/escitalopram) ($F = 20.3$, $p < 0.001$): On placebo, OCD patients (mean: 2.6, SD: 1.1) performed similarly to control subjects (mean: 2.8, SD: 1.1). OCD patients showed significantly increased accuracy in terms of disgust recognition after escitalopram (mean: 2.9, SD: 1.1; $p < 0.01$) compared to when they were on placebo. Conversely, controls on escitalopram (mean: 2.5, SD: 1.2; $p < 0.01$) showed significantly decreased accuracy in terms of disgust recognition compared to placebo (Fig. 2).

Subsequent analyses of data from the OCD patient group, with YBOCS- and MADRS-totals included as covariates, showed a main effect approaching significance for OCD severity to be associated with accuracy of performance in terms of disgust stimuli in the facial morphing task ($F = 3.7$, $p = 0.06$). Investigation of the interaction between accuracy and OCD severity showed that patients with increased OCD severity were significantly less accurate in their responses on both placebo ($r = -0.2$, $p < 0.01$) and escitalopram ($r = -0.3$, $p < 0.01$).

When accuracy of responses was compared between OCD patients on and those not on chronic SSRI treatment, a significant interaction between diagnostic group (i.e. controls/OCD patients on SSRI treatment/OCD patients not on SSRI treatment) and pharmacological challenge (i.e. placebo/escitalopram) was found in terms of the accuracy of disgust recognition ($F = 12.2$, $p < 0.001$; Table 2): On placebo, accuracy was similar across groups. Compared to the other two groups, OCD patients on SSRI treatment performed significantly better in terms of accuracy after escitalopram challenge ($F = 12.2$, $p < 0.001$). After escitalopram challenge, OCD patients on SSRI treatment showed significantly increased accuracy in terms of disgust recognition compared to when they were on placebo. In contrast, the accuracy of OCD patients not on SSRI treatment did not change significantly after escitalopram challenge compared to when they were on placebo (Fig. 3).

3.2.2. *Sensitivity for facial expressions of disgust ("intensity")*

Participants, i.e. both patients and controls, were more sensitive to disgust at the second session ($F = 7.1$, $p = 0.01$).

There also was a main effect for diagnosis in terms of sensitivity to disgust: OCD patients (mean: 44.1, SD: 11.3) were significantly more sensitive to disgust than controls (mean: 46.5, SD: 7.1; $F = 4.3$, $p < 0.05$). The interaction between diagnostic group (i.e. controls/OCD patients) and pharmacological challenge (i.e. placebo/escitalopram) in terms of sensitivity towards disgust was not significant however ($F = 1.7$, $p = 0.2$).

Subsequent analyses of OCD data only, with YBOCS- and MARDS-totals added as covariates, demonstrated that the severity of OCD symptoms did not significantly influence patients' sensitivity to disgust ($F = 0.3$, $p = 0.6$).

When sensitivity to disgust was investigated across three groups, a significant interaction between diagnostic group (i.e. controls/OCD patients on SSRI treatment/OCD patients not on SSRI treatment) and pharmacological challenge (i.e. placebo/escitalopram) was found in terms of the sensitivity to

Table 2
Accuracy of disgust recognition: 3-group comparison.

Challenge	Controls		OCD patients				Statistics
	Accuracy mean score*	SD	On chronic SSRI-treatment		Not on chronic SSRI-treatment		
			Accuracy mean score*	SD	Accuracy mean score*	SD	
Placebo	2.8	1.1	2.8	1	2.3	1.2	$F = 12.2$,
Pharmacological challenge	2.5	1.2	3.2	0.9	2.5	1.3	$p < 0.001$

* Arithmetic mean scores.

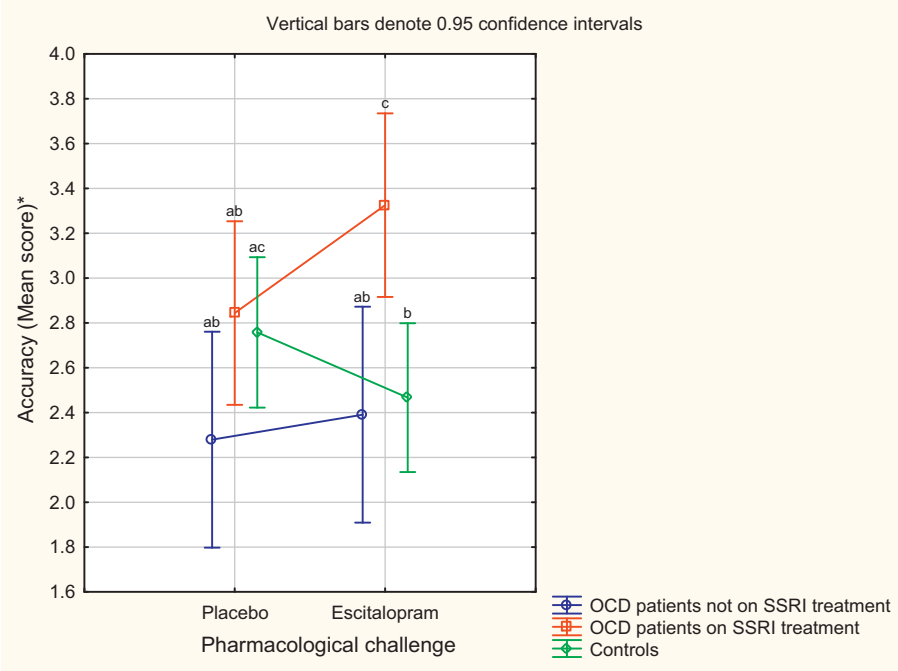

Fig. 3. Diagnostic group × Pharmacological challenge interaction in terms of the accuracy of disgust recognition. * The mean scores refer to the least squares (LS) means.

disgust ($F = 40.9$, $p < 0.001$): Compared to controls, all OCD patients (whether on SSRI treatment or not) were significantly more sensitive to disgust after escitalopram challenge. On placebo, sensitivity to disgust was similar across groups whereas after escitalopram challenge, OCD patients on SSRI treatment as well as those not on SSRI treatment showed significantly increased sensitivity to disgust compared to when they were on placebo. Controls' sensitivity to disgust decreased significantly after escitalopram challenge (Table 3; Fig. 4).

Subsequent analyses of OCD data only, with OCD patients who were on SSRI treatment and those not on any psychotropic medication at the time of the assessments separated, and with YBOCS- and MARDS-totals added as covariates, demonstrated that the severity of OCD symptoms did not significantly influence patients' sensitivity to disgust in either group ($F = 0.3$, $p = 0.6$).

4. Discussion

Our findings suggested that on placebo, the accuracy of disgust recognition in OCD patients and controls were similar. Our findings, based on the administration of a dynamic task that potentially provides greater power to differentiate groups, are consistent with previous work using static facial expressions which have failed to find a deficit in disgust recognition in OCD at baseline (e.g. [18,19]). However, the acute administration of escitalopram had a significantly different effect on OCD patients and controls in terms of the accuracy of their recognition of disgust in a dynamic facial expression recognition task. OCD patients had more accurate and more sensitive recognition of disgust after SSRI administration than after placebo, while controls had less accurate recognition and less sensitive recognition of disgust after SSRI administration than after placebo. OCD patients who were on SSRI treatment were particularly accurate at disgust recognition after acute SSRI administration (compared to untreated OCD patients and health controls).

Previous work has found that changes in emotional processing can be seen after acute administration of an SSRI. For example, single dose citalopram in healthy subjects improved detection of fearful and happy facial expressions [25,44] and attentional bias towards positive stimuli [44]. In our study controls showed significantly decreased accuracy and decreased sensitivity in terms of disgust recognition after single-dose escitalopram. This is consistent with other studies that have also found attenuated responses to aversive stimuli in healthy controls after acute SSRI administration, including, for example, several fMRI studies that found attenuated responses to aversive stimuli especially in the amygdala region after single-dose administration in health volunteers [45–48].

OCD patients, on the other hand, were more accurate and more sensitive in disgust recognition after an SSRI challenge. This is consistent with data that has indicated that after acute administration of an SSRI there are increased anxiety responses (e.g. [44]). More importantly, however, the contrast between the responses of OCD and healthy controls in disgust recognition after acute SSRI administration suggests that abnormal disgust processes in OCD are mediated by the serotonergic system. In particular, it would seem in particular contexts (perhaps those in

Table 3
Sensitivity to disgust recognition: 3-group comparison.

Challenge	Controls		OCD patients				Statistics
	Intensity level mean score*	SD	On chronic SSRI-treatment		Not on SSRI-treatment		
			Intensity level mean score*	SD	Intensity level mean score*	SD	
Placebo	45.6	6.9	44.5	8.4	45.6	9.9	$F = 40.9$,
Pharmacological challenge	47.3	7.2	43.2	13.4	43.1	12.0	$p < 0.001$

* Arithmetic mean scores.

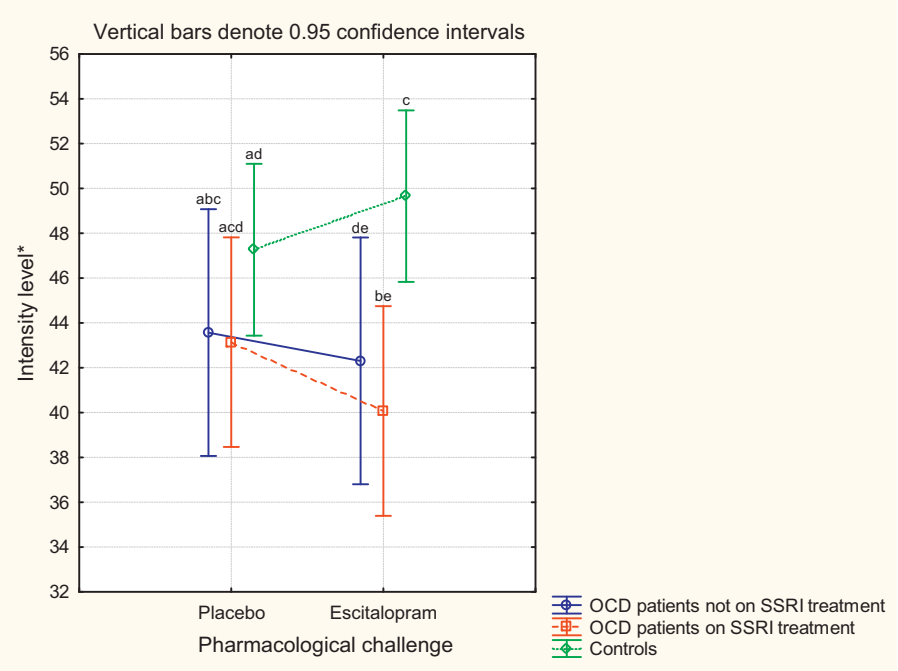

Fig. 4. Diagnostic group × Treatment interaction in terms of the sensitivity to disgust. * The mean scores refer to the least squares (LS) means.

which the serotonin system is activated, e.g. when the organism is under threat [49]), that OCD patients respond with a hyper-alert response to disgust, whereas healthy controls have attenuated responses.

It is notable that after chronic treatment of OCD with SSRIs, subjects were even more responsive to disgust. One may well have hypothesized that after appropriate management, OCD patients would demonstrate normalization of disgust recognition. However, it may be that this is a trait variable that does not alter, even with pharmacotherapy.

Performance on the facial morphing task of both OCD patients and controls was not significantly influenced by comorbid depressive (and by implication, anxiety) symptoms. We did however find a significant interaction between OCD severity and accuracy; patients with worse OCD were significantly less accurate in their responses to disgust stimuli on both placebo and escitalopram. This is consistent with previous studies that have also reported that increased severity of OCD may impact negatively on recognition of disgust. In particular, Corcoran et al. [1] found that participants with OCD who showed poor recognition of disgust reported more severe OCD symptoms than did those who performed "normally" on the task. Parker et al. [19] also reported that the one OCD patient who showed disgust recognition deficits had the most severe OC symptoms in their sample.

Patients and controls performed significantly better on all counts on the second assessment day suggesting that there may be a learning effect associated with this facial recognition task. In most cases however, the second assessment day was at least 1 week after the first, arguably buffering against a learning effect.

Limitations of the study include the presence of OCD patients with some, albeit minimal, comorbidity. Also, our OCD sample was heterogeneous in terms of their OC symptoms. The fact that patients with specific OC symptom subtypes may be more prone to impaired disgust recognition (see review by Berle and Phillips, 2006 [50]) warrants investigation of the influence of OCD subtype on task performance, in a larger sample. One of the strengths of the present analysis, however, is that we also investigated the possible influence of chronic SSRI treatment on disgust recognition in patients with OCD.

In summary, there are very few studies that have investigated the effects of acute SSRI administration on emotional processing in humans, and most of these have focused on healthy volunteers. Our study is, to our knowledge, the first to investigate the effects of acute SSRI administration on disgust recognition in OCD. The finding that OCD patients were more accurate and more sensitive to disgust after SSRI, while controls were less accurate and less sensitive, indicates that abnormalities in disgust recognition in OCD may be particularly evident in contexts characterized by serotonin release (perhaps threat-evoking situations). Putative serotonin-mediated hyper-responsivity to disgust cues appears to be a trait characteristic of OCD, as it is not diminished by treatment with SSRIs. Future directions should include investigation of brain activation in OCD patients and healthy controls on placebo/escitalopram during administration of an emotion recognition task using fMRI.

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