Published in final edited form as:

Depress Anxiety. 2017 March ; 34(3): 301–306. doi:10.1002/da.22492.

The role of response inhibition in medicated and unmedicated obsessive-compulsive disorder patients: Evidence from the stop-signal task

Eyal Kalanthroff^{1,2}, Tobias Teichert³, Michael G. Wheaton^{1,2,4}, Marcia B. Kimeldorf², Omer Linkovski⁵, Susanne E. Ahmari³, Abby J. Fyer^{1,2}, Franklin R. Schneier^{1,2}, Gideon E. Anholt⁵, and H. Blair Simpson^{1,2}

¹Columbia University Medical Center, Department of Psychiatry

²New York State Psychiatric Institute, Division of Clinical Therapeutics

³University of Pittsburgh, Department of Psychiatry and the Center for the Neural Basis of Cognition

⁴Yeshiva University, Ferkauf Graduate School

⁵Ben-Gurion University of the Negev, Beer-Sheva, Israel, Department of Psychology

Abstract

Background—Numerous studies have investigated response inhibition in obsessive-compulsive disorder (OCD), with many reporting that OCD patients demonstrate deficits in response inhibition as compared to controls. However, reported effect sizes tend to be modest and results have been inconsistent, with some studies finding intact response inhibition in OCD. To date, no study has examined the effect of medications on response inhibition in OCD patients.

Methods—We analyzed results from a stop-signal task to probe response inhibition in 65 OCD patients (32 of whom were medicated) and 58 healthy controls.

Results—There was no statistically significant difference in stop-signal reaction time between the OCD group and the healthy control group, nor between the medicated and unmedicated OCD patients. However, variability was significantly greater in the medicated OCD group compared to the unmedicated group.

Conclusions—These results indicate that some samples of OCD patients do not have deficits in response inhibition, making it unlikely that deficient response inhibition underlies repetitive behaviors in all OCD patients. Future research is needed to fully elucidate the impact of

Correspondence concerning this article should be addressed to Eyal Kalanthroff, Columbia University Medical Center, New York State Psychiatric Institute, Unit #69, 1051 Riverside Drive, New York, NY 10032 unit 3205, Phone: 1646-774-8049, eyalkant@gmail.com.

Disclosures

Dr. Simpson has received research funds from Transcept Pharmaceuticals (2011–2013), and royalties from Cambridge University Press and UpToDate, Inc. Dr. Schneier has received research support from Forest Labs/Actavis and the Sycamore Fund, served on a scientific advisory board for Genentech, and received royalties from Cambridge University Press and UpToDate, Inc. The remaining authors declare no potential conflict of interest.

medication use on stop-signal performance. Implications for future research on the cognitive processes underlying repetitive thoughts and behaviors are discussed.

Keywords

Executive function; attention; cognitive manifestations; stop signal; psychopathology

Deficits in response inhibition (RI), the ability to suppress irrelevant actions or actions that are not appropriate in a specific context^[1], have been documented in patients with obsessive-compulsive disorder (OCD) and hypothesized to underlie the repetitive behaviors that characterize the disorder. In fact, some studies have suggested that a deficit in RI is an endophenotype of OCD^[2, 3, 4, 5]. For example, OCD patients exhibit diminished dorsal ACC activation during response inhibition tasks, which may underlie their inability to properly inhibit motoric behaviors^[6]. However, not all studies find an RI deficit in OCD, as highlighted in a recent meta-analysis^[7]. To date, no study has examined the effect of medication status on response inhibition in OCD patients.

The most consistent results showing an RI deficit use the *stop-signal* task (SST)^a, a common probe of RI^[8,9]. In this task, participants are asked to rapidly respond to visual stimuli (go signals) knowing that on some trials an auditory stimulus (stop signal) will be presented, requiring them to inhibit their already-initiated (pre-potent) response. This paradigm estimates the ability to inhibit pre-potent responses by calculating participants' stop-signal reaction times (SSRT) – an index of RI^[1]. Longer SSRTs have been found in OCD patients compared to healthy controls (HC)^[2, 3, 4, 5, 10, 11]. However, in a recent study, we found that unmedicated OCD patients did not have longer SSRTs relative to HCs (although patients exhibited a gradual slowing of reaction times over the course of the experiment, which reduced the likelihood of errors of commission on stop-trials)^[12]. Importantly, most studies that do report SSRT differences have had relatively small samples, recruited both medicated and unmedicated patients, and found modest effects^[13].

Two recent studies stand out for not mixing medicated and unmedicated patients: de Wit et al.^[14] compared 41 unmedicated OCD patients and 37 HC and found significantly elongated SSRT in the OCD group (group difference: $t(76) = 2.07$, $p = .04$; *Cohen's d* = .47), and comparable SDs across groups. Sohn et al.^[11] also reported modest differences in SSRT between 60 medicated OCD patients^b and 52 HC (group difference: $t(110) = 2.14$, $p = .04$; *Cohen's d* = .40). However, in this study SDs appeared notably larger in the medicated OCD group. Although replication is required, these data suggest that medication status may affect the variability of SSRT in OCD. Notably, serotonin reuptake inhibitors (SRIs; the only FDA-approved first line medication for OCD) have been found to have no effect on SSRT in rats^[15, 16] and healthy volunteers^[17, 18]. However, the effect of medication on SSRT in OCD patients has not before been directly tested.

^aThe *stop-signal* task is recommended by the Cognitive Systems Working Group of the NIMH Research Domain Criteria (RDoC) initiative (<http://www.nimh.nih.gov/research-priorities/rdoc>).

^bThese 60 patients, who had usable data came from a sample of 80 OCD patients in which 78 were medicated.

To examine the effect of medication status on SST performance, we capitalized on data from two academic sites that administered the *STOP-IT task* (a downloadable standardized version of the SST)^[19] to both medicated and unmedicated OCD patients and to HC. By combining data from two sites, we could analyze a much larger sample and evaluate the generalizability of our findings. Based on the literature, we predicted longer SSRTs in OCD patients. On the basis of rodent and healthy human work reviewed above, we did not expect mean SSRT differences between the medicated and unmedicated OCD groups. However, we explored whether the medicated group would have more variability on the basis of the descriptive results described above.

Method

Setting

SSRT data was collected as part of ongoing OCD studies at two academic sites: the New York State Psychiatric Institute/Columbia University Medical Center, New York, NY (referred to as NYPSI) and Ben-Gurion University of the Negev, Beer-Sheva, Israel (referred to as BGU). Subjects were recruited by clinical referral, advertisements, and at the BGU site also by the university online Psychology Experiments System. Each site's institutional review board (IRB) reviewed and approved the study. Participants provided written informed consent.

Participants

Participants included 65 individuals who met criteria for OCD as their principal diagnosis (as determined by a trained psychiatrist or psychologist during a clinical interview and confirmed by an OCD diagnosis on the Structured Clinical Interview for DSM-IV; SCID^[20]) and 58 healthy controls (HC). Demographic and clinical characteristics of the sample at each site are provided in Table 1. Thirty-two out of the 65 OCD patients were on medications for their OCD at the time of testing. None of the HC were on any psychiatric medication. Participants were excluded for psychotic disorders, alcohol/substance abuse or dependence, eating disorders, any serious neurological condition, current severe depression^c, or hoarding as a sole OCD symptom.

Procedure

Clinical Evaluation—At both sites, psychiatric diagnoses and eligibility were determined via clinical interview, conducted by trained clinicians (psychologists or psychiatrists) with expertise in anxiety, OCD and related disorders, using the SCID^[20]. OCD symptom severity was evaluated by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS)^[21] at the NYSPI site, and the Obsessive-Compulsive Inventory-Revised (OCI-R)^[22] at the BGU site. Depressed patients were also evaluated by a depression measure (Hamilton Rating Scale for Depression; HAM-D²³ at the NYSPI site, and Beck Depression Inventory-II; BDI-II²⁴ at the BGU site).

^cSevere depression was defined via benchmarks on the depressive symptoms measures used at each site: (at NYSPI: HAM-D²³>20 and at BGU: BDI-II²⁴>30)

SSRT Task—We used the “STOP-IT” program^[19], which uses a white square or circle on a black background as the go signal. The stop signal was an auditory tone (750 Hz, 75ms). The experiment included one practice block of 32 trials and three experimental blocks of 64 trials each. Each trial started with a 250ms fixation (a white plus sign in the center of a black screen), followed by a visual go stimulus. Participants were asked to press the “z” key if they saw a square and the “/” key if they saw a circle. The written and verbal instructions stated to press the correct key as quickly and accurately as possible, and emphasized not to wait for a potential stop signal. The visual stimulus stayed in view for 1,250ms regardless of the latency of the response. RT for no stop-signal trials (nsRT) was calculated from the appearance of the visual stimulus to the reaction and analyzed as an indication of task engagement. Each trial ended with a 2,000ms inter-trial interval. On a random selection of 1/4 of the trials, an auditory stop signal followed the go signal after a variable stop-signal delay (SSD) that was initially set at 250ms and adjusted by a staircase tracking procedure: after each successful stop the SSD was extended by 50ms and after each unsuccessful stop the SSD was shortened by 50ms. SSRT was defined as the time needed for successful inhibition at 50% of attempts, and was estimated by subtracting the mean SSD from the mean nsRT. During this interval, they received feedback on their performance in the previous block. For further details on the stimuli and procedure see Verbruggen et al.^[19]

Statistical Methods

To assess group differences in demographic and clinical variables, chi-square tests were used for categorical variables (gender and diagnosis of depression) and t-tests were used for continuous variables (age, OCD and depression symptom severity, and years of education). To compare OCD symptom severity across sites, we calculated Z-scores for Y-BOCS scores (NYSPI) and for OCI-R scores (BGU) separately. Analysis of nsRT was conducted via a one-way ANOVA with group (HC vs. medicated OCD vs. unmedicated OCD) as a between-subjects factor. To investigate our a priori prediction regarding the differences in SSRT between OCD patients (medicated and unmedicated combined) and HC, we conducted a planned contrast (1-tailed t-test). To investigate the effect of medication status on SSRT we conducted a planned contrasts between medicated and unmedicated OCD patients, and between each of the medication groups separately and HC (2-tailed t-tests). To explore differences in SD of SSRT and nsRT we used Levene’s test of homogeneity of variances. All analyses were repeated with site (BGU vs. NYSPI) as a covariate, but the general pattern of results did not change. Therefore in the results below we report on the combined BGU and NYSPI samples.

A power analysis using G*Power 3.1^[25], based on the effect sizes reported in previous studies^[11], indicated that the current sample allowed for examination of group differences in SSRT at a power > 90% to test small to medium size effects with a Type 1 error (α < 0.05).

Results

As shown in Table 1, OCD patients did not significantly differ from HC in age ($t(121) = 1.929$, $p > .05$), gender ($\chi^2(1) < 1$, Cramer’s $V < 1$, $p = .67$), or years of education ($t(121) <$

1, $p = .50$). There were also no significant differences between medicated and unmedicated OCD patients in any of these variables (age: $t(63) < 1$, $p = .9$; gender: $\chi^2(1) < 1$, Cramer's $V < 1$, $p = .37$; years of education: $t(63) = 1.51$, $p = .14$). As expected, OCD patients had higher OCD symptom severity scores than HC ($t(121) = 20.79$, $p < .000$). There was no significant difference between medicated and unmedicated OCD patients in OCD symptom severity ($t(63) < 1$, $p = .83$) or in the prevalence of depression (medicated patients: 5/32; unmedicated patients: 3/33; Cramer's $V < 1$, $p = .42$). There was no significant difference in nsRT between medicated OCD patients, unmedicated patients, and HC (means: $F(2, 120) = 1.233$, $MSE = 17,302.232$, $p = .3$; SD: $F(2, 120) = .26$, $p = .77$), indicating comparable task engagement between groups (Table 2).

No participant had significantly more or less than 50% successful inhibitions in the stop-signal task, suggesting that the staircase algorithm was successful (Table 2; estimation method by Verbruggen & Logan [19, 26]). The OCD group had slightly longer mean SSRT compared to HC; however this difference was not significant ($t(121) = 1.37$, $p = .09$, Cohen's $d = .25$; one-tailed); there was also no significant group difference in variance ($F(1, 121) < 1$, $p = .6$). SSRT did not significantly differ between medicated and unmedicated OCD patients ($t(63) < 1$, $p = .35$, Cohen's $d = .23$). However, the variance in the medicated group was significantly larger than in the unmedicated group ($F(1, 63) = 4.543$, $p < .05$). There was no significant difference in mean SSRT between HC and unmedicated patients ($t(89) < 1$, $p = .52$, Cohen's $d = .14$; two-tailed) and between HC and medicated patients ($t(88) = 1.64$, $p = .11$, Cohen's $d = .34$; two-tailed). The variance in the unmedicated group was comparable to HC ($F(1, 89) < 1$, $p < .40$) whereas the variance in the medicated group was significantly larger than in HC ($F(1, 88) = 3.878$, $p < .05$; Table 2).

Discussion

The current study investigated response inhibition (RI), as measured by the stop-signal task (SST), in a large sample of OCD patients. Although SSRTs were slightly longer in the OCD group compared to the HC group, this difference was not significant. This null result is similar to that of a previous investigation of unmedicated patients^[12] as well as a small medication-mixed sample that was recently reported^[27]. In addition, we found no significant differences in mean SSRT between medicated and unmedicated OCD patients nor between each of these groups and HC. However, medicated patients had significantly greater variability in SSRT as compared to unmedicated patients and to HC. These effects were specific to SSRT, as there were no significant group differences in either mean nsRT or its variability.

Inspection of data from prior studies indicates a tendency towards larger SDs in medicated OCD patients^[11] — yet the present study was the first to evaluate this formally. The finding of increased variability in medicated OCD patients has important implications for studies of response inhibition using the SST. Specifically, increased variance in medicated patients may indicate the presence of some unknown underlying factor that affects RI in OCD patients and is influenced by medication. If so, isolating this factor may clarify whether deficits in RI are a fundamental characteristic of OCD per se. For example, recent investigations have shown that anxiety impairs RI^[28, 29], and specifically that elevated

anxiety results in prolonged SSRTs^[30, 31]. Thus, perhaps medicated OCD patients have increased SSRT variability because of SRIs' varying effects on anxiety (e.g., improving anxiety in some patients but not others, helping alleviate anxiety to varying degrees in those it helps). This varying effect on anxiety might be mirrored by a varying effect on SSRT. Longitudinal work is needed to test this possibility (i.e., directly measuring SSRT before and after medication). Such data will allow us to conclude whether changes in anxiety levels, following medication treatment, correlate with the changes in SSRT in OCD patients, and could explain the increased variability in SSRT that we observed in medicated OCD patients.

Our failure to replicate the effect of longer SSRT in OCD patients coupled with the small effect sizes commonly found in the literature (as reviewed in the introduction) indicates that RI deficits are insufficient to explain the repetitive behaviors in all OCD patients. Two recent meta-analyses found small to medium effects sizes for RI deficits in OCD (effect sizes = .49^[7] and .37^[32]); however, authors on both studies reached different conclusions. While the authors of one study concluded that these deficits are small and unlikely, on their own, to explain the repetitive thoughts and behaviors in OCD^[7], the authors of the second study^[32] concluded that RI deficits are part of OCD patients' broad and stable impairment in executive control. However, it is also worth noting that studies that did find RI deficits in OCD subjects (either medicated or unmedicated) did not find a correlation between these deficits and OCD symptom severity^[10, 14]. This has spurred further study of neural processes that might better explain repetitive thoughts and behaviors. For example, Gillan et al.^[33, 34] have suggested that the repetitive behaviors in OCD are due to an imbalance between the executive system and the habitual system. Kalanthoff et al.^[35, 36], reported a deficit in OCD patients' task-control—an executive mechanism that is responsible for goal-directed task selection by suppressing irrelevant associative tasks^[37]. Tendency towards habit formation and difficulties executing control over irrelevant tasks, have both been shown to correlate with OCD symptom severity^[33, 37]. Future research is needed to determine whether these mechanisms are more directly related to OCD than RI deficits, and whether clinical interventions that directly target these processes prove beneficial.

Several limitations of the current study should be noted. First, even though the exact same task was used at both sites and diagnoses were determined by trained clinicians, formal cross-site reliability tests were not conducted. Second, although the SST is a widespread and validated task, future studies need to replicate the current results using other tasks measuring RI. Third, although OCD was the principal diagnosis for all our patients and we excluded comorbid psychiatric conditions previously linked to RI deficits, we do not have data regarding other comorbid disorders. We also do not have full data on which specific medications our patients were on. However, 100% of the patients for whom we do have medication data (56% of all medicated patients), were on SRIs, and the other participants were most likely as well (given that medicated patients were required to only be on medications prescribed specifically for OCD and SRIs are the only approved first-line medications for OCD at both sites). Finally, our study employed a single time point design. Future research using longitudinal methods is required to fully elucidate the relationship between medication use and SSRT in OCD.

To summarize, the current results indicate that RI is not significantly impaired in all OCD patients and that variance is greater in medicated patients. These data – together with the small effect sizes that characterize the literature – suggest that RI deficits are insufficient to explain the repetitive behaviors in all OCD patients. A 'second-hit' model, with anxiety mediating the relation between medication-status and RI is possible and requires further research. Our results highlight the importance of considering medication status when evaluating OCD patient performance on neurocognitive tasks.

Acknowledgments

This study was supported by the National Institute of Mental Health (R01MH091694 [PIs: H.B.S., F.R.S., A.J.F.], K24MH091555 [PI: H.B.S.], the New York State Office of Mental Hygiene, and donors to the New York-Presbyterian Hospital Youth Anxiety Center. Dr. Kalanthoff is supported by the Rothschild foundation, the Molberger Scholar award, and by The Israel Science Foundation (grant No. 79/15).

References

1. Verbruggen F, Logan GD. Response inhibition in the stop-signal paradigm. *Trends Cogn Sci.* 2008; 12:418–424. [PubMed: 18799345]
2. Chamberlain SR, Blackwell AD, Fineberg NA, et al. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neurosci Biobehav R.* 2005; 29:399–419.
3. Chamberlain SR, Fineberg NA, Menzies LA, et al. Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *Am J Psychiat.* 2007; 164:335–338. [PubMed: 17267798]
4. Menzies L, Achard S, Chamberlain SR, et al. Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain.* 2007; 130:3223–3236. [PubMed: 17855376]
5. Penades R, Catalan R, Rubia K, et al. Impaired response inhibition in obsessive compulsive disorder. *Eur Psychiat.* 2007; 22:404–410.
6. Fineberg NA, Potenza MN, Chamberlain SR, et al. Probing compulsive and impulsive behaviors, from animal models to endophenotypes: A narrative review. *Neuropsychopharmacol.* 2010; 35:591–604.
7. Abramovitch A, Abramowitz JS, Mittelman A. The neuropsychology of adult obsessive-compulsive disorder: A meta-analysis. *Clin Psychol Rev.* 2013; 33:1163–1171. [PubMed: 24128603]
8. Logan, GD. On the ability to inhibit thought and action: a user's guide to the stop signal paradigm. In: Dagenbach, D., Carr, TH., editors. *Inhibitory processes in attention, memory and language.* San Diego, CA: Academic Press; 1984. p. 189-239.
9. Logan GD, Cowan WB. On the ability to inhibit thought and action: A theory of an act of control. *Psychol Rev.* 1984; 91:295–327.
10. Boisseau CL, Thompson-Brenner H, Caldwell-Harris C, et al. Behavioral and cognitive impulsivity in obsessive-compulsive disorder and eating disorders. *Psychiat Res.* 2012; 200:1062–1066.
11. Sohn SY, Kang JI, Namkoong K, et al. Multidimensional measures of impulsivity in obsessive-compulsive disorder: Cannot wait and stop. *PLoS ONE.* 2014; 9:e111739. [PubMed: 25372136]
12. Ahmari SE, Leotti L, Teichert T, et al. Performance on the Stop Signal task is impaired in unmedicated adults with obsessive-compulsive disorder. Under review.
13. Lipszyc J, Schachar R. Inhibitory control and psychopathology: A meta-analysis of studies using the stop signal task. *J Int Neuropsych Soc.* 2010; 16:1064–1076.
14. de Wit SJ, de Vries FE, van der Werf YD, et al. Presupplementary motor area hyperactivity during response inhibition: a candidate endophenotype of obsessive-compulsive disorder. *Am J Psychiat.* 2012; 169:1100–1108. [PubMed: 23032388]
15. Bari A, Eagle DM, Mar AC, et al. Dissociable effects of noradrenaline, dopamine, and serotonin uptake blockade on stop task performance in rats. *Psychopharmacology.* 2009; 205:273–283. [PubMed: 19404616]

16. Eagle DM, Lehmann O, Theobald DE, et al. Serotonin depletion impairs waiting but not stop-signal reaction time in rats: Implications for theories of the role of 5-HT in behavioral inhibition. *Neuropsychopharmacol.* 2009; 34:1311–1321.
17. Clark L, Roiser JP, Cools R, et al. Stop signal response inhibition is not modulated by tryptophan depletion or the serotonin transporter polymorphism in healthy volunteers: Implications for the 5-HT theory of impulsivity. *Psychopharmacology.* 2005; 182:570–578. [PubMed: 16163530]
18. Eagle DM, Bari A, Robbins TW. The neuropsychopharmacology of action inhibition: Cross-species translation of the stop-signal and go/no-go tasks. *Psychopharmacology.* 2008; 199:439–456. [PubMed: 18542931]
19. Verbruggen F, Logan GD, Stevens MA. STOP-IT: Windows executable software for the stop-signal paradigm. *Behav Res Methods.* 2008; 40:479–483. [PubMed: 18522058]
20. First, MB., Spitzer, RL., Gibbon, M., Williams, JBW. Structured clinical interview for DSM-IV disorders (SCID-IV). Washington DC: American Psychiatric Press; 1997.
21. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown obsessive compulsive scale: Development, use, and reliability. *Arch Gen Psychiat.* 1989; 46:1006–1011. [PubMed: 2684084]
22. Foa EB, Huppert JD, Leiberg S, et al. The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychol Assessment.* 2002; 14:485–496.
23. Hamilton M. A Rating Scale for Depression. *J Neurol Neurosurg Psychiatry.* 1960; 23:56–62. [PubMed: 14399272]
24. Beck, AT., Steer, RA., Brown, GK. Beck depression inventory-II. San Antonio: 1996.
25. Faul F, Erdfelder E, Lang AG, Buchner A. G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods.* 2007; 39:175–191. [PubMed: 17695343]
26. Verbruggen F, Logan GD. Models of response inhibition in the stop-signal and stop-change paradigms. *Neurosci Biobehav R.* 2009; 33:647–661.
27. Rubia K, Cubillo A, Smith AB, et al. Disorder specific dysfunction in right inferior prefrontal cortex during two inhibition tasks in boys with attention deficit hyperactivity disorder compared to boys with obsessive-compulsive disorder. *Hum Brain Mapp.* 2010; 31:287–299. [PubMed: 19777552]
28. Berggren N, Richards A, Taylor J, Derakshan N. Affective attention under cognitive load: reduced emotional biases but emergent anxiety-related costs to inhibitory control. *Front Hum Neurosci.* 2013; 7:188. [PubMed: 23717273]
29. Derakshan N, Ansari TL, Hansard M, et al. Anxiety, inhibition, efficiency, and effectiveness: An investigation using the antisaccade task. *Exp Psychol.* 2009; 56:48–55. [PubMed: 19261578]
30. Kalanthoff E, Henik A, Derakshan N, et al. Anxiety, Emotional Distraction, and Attentional Control in the Stroop Task. *Emotion.* 2015 advanced online publication.
31. Pessoa L, Padmala S, Kenzer A, et al. Interactions between cognition and emotion during response inhibition. *Emotion.* 2012; 12:192–197. [PubMed: 21787074]
32. Snyder HR, Kaiser RH, Warren SL, Heller W. Obsessive-Compulsive Disorder Is Associated With Broad Impairments in Executive Function A Meta-Analysis. *Clin Psychol Sci.* 2015; 3:301–330. [PubMed: 25755918]
33. Gillan CM, Pappmeyer M, Morein-Zamir S, et al. Disruption in the balance between goal-directed behavior and habit learning in obsessive-compulsive disorder. *Am J Psychiat.* 2011; 168:718–726. [PubMed: 21572165]
34. Gillan CM, Morein-Zamir S, Urcelay GP, et al. Enhanced avoidance habits in obsessive-compulsive disorder. *Biol Psychiat.* 2014; 75:631–638. [PubMed: 23510580]
35. Kalanthoff E, Anhalt GE, Henik A. Always on guard: Test of high vs. low control conditions in obsessive-compulsive disorder patients. *Psychiat Res.* 2014; 219:322–328.
36. Kalanthoff E, Henik A, Simpson HB, et al. To do or not to do: Task control deficit in obsessive-compulsive disorder? Under review.
37. Kalanthoff E, Goldfarb L, Henik A. Evidence for interaction between the stop signal and the Stroop task conflict. *J Exp Psychol Human.* 2013; 39:579.

Table 1

Demographic and Clinical Characteristics of the Samples

	HC N=58		OCD N=65		OCD-M N=32		OCD-NM N=33	
	BGU N=35	NYSPI N=23	BGU N=30	NYSPI N=35	BGU N=14	NYSPI N=18	BGU N=16	NYSPI N=17
Age (years)	25.46 (3.8)	27.31 (5.6)	26.77 (7.4)	29.99 (8.25)	26.14 (5.9)	30.11 (9.88)	27.31 (8.7)	29.86 (6.4)
Females (%)	69%	65%	83%	60%	86%	50%	81%	71%
Education (years)	13.43 (0.81)	15.70 (2.08)	13.10 (0.40)	15.83 (2.20)	13.21 (0.58)	16.33 (2.22)	13 (0.5)	15.29 (2.11)
YBOCS	----	0.26 (1.25)	----	25.80 (3.65)	----	26.28 (3.98)	----	25.29 (3.31)
OCD-R	15.94 (3.28)	----	35.50 (9.47)	----	34.57 (8.34)	----	36.31 (10.56)	----
OCD Symptom severity Z-score	-0.76 (0.27)	-1.19 (0.97)	0.80 (0.7)	0.78 (0.28)	0.80 (0.7)	0.82 (0.31)	0.95 (0.88)	0.74 (0.26)
Depression (#)	0	0	6	2	3	2	3	0

BGU – Ben-Gurion University of the Negev, Israel. NYSPI – New-York State Psychiatric Institute, Columbia University Medical Center, NY. Standard Deviations are in brackets. Symptoms severity was measured using the Yale-Brown Obsessive Compulsive Scale (YBOCS) in the NYSPI site and using the Obsessive-Compulsive Inventory – Revised (OCI-R) in the BGU site. Z-scores for symptoms severity were calculated for each site separately. Depression is the number of participants meeting DSM-IV-TR criteria for MDD.

Table 2

Stop signal task

Measurement	HC (N=58)	OCD (N=65)	OCD medicated (N=32)	OCD unmedicated (N=33)
Stop-signal task				
SSRT (SD)	241 (45)	253 (55)	260 (66)	247 (43)
nsRT (SD)	588 (133)	575 (131)	550 (129)	599 (130)
p(r s)	0.49	0.49	0.50	0.48

* Results in the stop-signal task. SSRT = stop-signal reaction time; nsRT = reaction time for no stop-signal trials; p(r|s) = probability of correct stopping. Standard deviations (SD) are presented in brackets. All reaction-times are in milliseconds.