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What Are the Biological Mechanisms of Obsessive-Compulsive Disorders (OCD)?

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ABSTRACT

In this paper, we sought to understand what are the biological mechanisms of Obsessive-Compulsive Disorders so that we could learn how to be aware of our obsessive-compulsive tendencies. Previous research has found that obsessive-compulsive disorder correlates with brain dysfunctions such as with frontal-parietal communication, brain activity in the right frontal pole, and levels of dopamine. In our first (correlational) study, we tested the strength of these relationships by examining naturalistic daily changes in their variables longitudinally over a one-week period. We measured parietal cortex functioning by performance in the Spatial Cueing Task, frontal cortex functioning by performance in the Berg's Card Sorting Test, levels of dopamine by number of eye blinks in one minute, and OCD by number of obsessive thoughts and compulsive behaviour in a day. Based on the strength of correlation found between parietal cortex functioning and levels of OCD symptoms in our correlational study, we then conducted a second (experimental) study to test for a causal relationship between these two variables. Over a one-week period, we randomly assigned participants each day to either a high parietal cortex functioning experimental condition or a low parietal cortex functioning control condition and measured the effect this manipulation had upon the levels of OCD symptoms. Data pooled across participants in our correlational study showed significant correlations of levels of OCD symptoms with parietal cortex functioning, but not with frontal cortex functioning or with levels of dopamine. However, the results of our experimental study failed to establish a causal role of the parietal cortex functioning upon levels of OCD symptoms. Therefore, the influences of specific brain structures and neurotransmitters on levels of OCD symptoms remains uncertain.

1. Introduction

1.1 Research Problem

Obsessive-Compulsive Disorder (OCD) causes personal suffering that hinders one to thoroughly enjoy life. Having obsessive-compulsive tendencies drains one's energy level and occupies one's mind in an unhealthy way. It triggers intrusive thoughts, irrational beliefs, and an excessive reliance on unproductive behaviours. As individuals who struggle with symptoms of obsessive-compulsive disorder and have family members who also face this condition, we would like to understand the physiological processes of this disorder in order to better cope with it and not be prisoners to our perfectionism.

1.2 Literature Review

One factor previously found to correlate with OCD is a deficient frontal-parietal communication. For example, in an experimental study by Liu et al. (2021), the researchers measured the brains' resting-state Functional Connectivity (rsFC) and Effective Connectivity (rsEC) of a group of unmedicated individuals diagnosed with OCD and a healthy control group through fMRI and MRI scans. They found that the interconnection between the frontal-parietal cortex and both the basal ganglia and the cerebellum was weaker in the OCD group than in the health control group. Based on these results, the researchers suggested that individuals with OCD have an impaired frontal-parietal interconnectivity.

Another factor previously found to correlate with OCD is increased activity in the right frontal pole. For example, in a study by Bohon et al. (2020), the Wisconsin Card Sorting Test (WCST) was utilized on the participants to measure perseverative errors in OCD participants. The participants were given instructions to match the cards to a certain rule that they were not informed about. However, through feedback, participants were able to figure out the sorting rule. Throughout the experiment researchers would change the rule without the participants knowing and again the participants would have to figure out the new sorting rule based on their feedback. The OCD participants showed increased activity in the brains' right frontal pole and increased perseverative errors, when compared to the healthy control group. Based on these results, the researchers suggested that mental skill tasks, such as the WCST, trigger more intense frontal brain activity in OCD participants.

A third factor previously found to correlate with OCD is increased levels of dopamine. For example, in a study by Viol et al. (2020), venipuncture produced an increase in dopamine levels in patients that met DSM-IV criteria for OCD after they underwent psychotherapy. Based on these results, the researchers suggested that, through psychotherapy treatment, increases in dopamine levels result in reduced OCD symptoms.

1.3 Hypotheses

Based on the above literature review, we predicted the following hypotheses:
Hypothesis #1: If parietal cortex functioning decreases then OCD symptoms will increase.
Hypothesis #2: If frontal cortex functioning decreases then OCD symptoms will increase.
Hypothesis #3: If dopamine levels increase then symptoms of OCD will decrease.

2. Methods

2.1 Participants

The two authors of this paper served as the participants in its studies. The participants ranged in age from twenty-one to thirty-three years old, with an average age of twenty-seven years, and included two cisgender females. The participants were all undergraduate students at Camosun College who completed the current studies as an assignment for Psych 215 ("Biological Psychology") and were grouped together due to their mutual interest in OCD.

2.2 Correlational Study Methods

We first performed a correlational study to test concurrently all of our hypotheses by examining naturalistic daily changes in their variables longitudinally. Each participant kept a study journal with them at all times

over this study's one-week period in order to record self-observations of the following four variables: (1) parietal cortex functioning, (2) frontal cortex functioning, (3) levels of dopamine, and (4) levels of OCD symptoms.

2.2.1 Parietal Cortex Functioning

To measure parietal cortex functioning, each participant did the Spatial Cueing Task every morning during the one-week study period. The Spatial Cueing Task was accessed in the "Psych Lab 101" app (https://www.neurobs.com/menu_presentation/menu_teaching/psych_lab_101).

2.2.2 Frontal Cortex Functioning

To measure frontal cortex functioning, each participant took the Berg's Card Sorting Task test each day first thing in the morning during the one-week study period. The Berg's Card Sorting Task test was was accessed in the "Psych Lab 101" app (https://www.neurobs.com/menu_presentation/menu_teaching/psych_lab_101).

2.2.3 Levels of Dopamine

To measure levels of dopamine, each participant measured how many times they blinked in one minute each day first thing in the morning during the one-week study period.

2.2.4 Levels of OCD Symptoms

To measure the levels of OCD symptoms, each participant recorded in a study journal the number of times they had obsessive thoughts and compulsive behaviours, and the types of obsessive thoughts and compulsive behaviours they experienced each day. We considered the types of obsessive thoughts to be every repetitive, irrational, and/or intrusive thoughts we would have, and the types of compulsive behaviours to be every ritualized behaviour we performed, such as dysfunctional cleaning, counting, rearranging, planning, and others. This process had to be completed in all seven days of the experiment.

2.3 Correlational Study Planned Analyses

To assess the strength and statistical significance of associations between variables predicted by our three hypotheses, we performed Pearson product moment correlations of their predictor variables (performance in the Spatial Cueing Test and Berg's Card Sorting Task, and eye blink counts) with their outcome variable (the amounts of times participants experience obsessive thoughts and compulsive behaviours in a day). For testing Hypothesis #1, we correlated each day's performance in the Spatial Cueing Task with that participant's observations of the number of obsessive thoughts and compulsive behaviours on that day. For testing Hypothesis #2, we correlated each day's performance in the Berg's Card Sorting Task with that participant's observations of the number of obsessive thoughts and compulsive behaviours on that day. For testing Hypothesis #3, we correlated the number of eye blinks in a minute every morning with that participant's observation of the number of obsessive thoughts and compulsive behaviours on that day. We performed all of the above correlations separately for each participant as well as using data pooled across all of the participants. For the correlations using pooled data, in addition to using the raw data, we also performed correlations after we had first transformed the data from each participant into *z*-scores in order to standardize differences in averages and variability seen between the participants in their data and thus make them more comparable. A correlation coefficient was considered statistically significant if the probability of its random occurrence (p) was $< .05$ (i.e., less than 5% of the time expected by chance alone).

2.4 Experimental Study Methods

Based on the strength of the correlation between parietal cortex functioning and levels of OCD symptoms found in our correlational study, we then chose to conduct an experimental study to test for a causal relationship between these two variables from Hypothesis #1.

We manipulated the independent variable, parietal cortex functioning, over a one-week period by randomly assigning - through flipping a coin - participants each day to either a high parietal cortex functioning experimental condition or a low parietal cortex functioning control condition using a blind procedure. On the high parietal cortex functioning experimental condition days, participants took the Spatial Cueing Task first thing in the morning, as previously described above in the Correlational Study Materials and Procedures. The duration of the test was approximately 5 minutes to complete. On the low parietal cortex functioning control condition days, participants followed their regular routine without taking the Spatial Cueing Task. In both conditions, the levels of OCD symptoms of the participants was measured on a obsessive-compulsive symptoms scale ranging from 0 to 10, with 0 describing neither obsessive thoughts, nor compulsive behaviours in one day, 5, as moderate obsessive thoughts and compulsive behaviours in one day, and 10, as numerous obsessive thoughts and compulsive behaviours in one day.

While it was impossible for participants to be unaware of what condition they were receiving and thus maintain a double-blind procedure, to help reduce bias in measurements each participant had a family member or roommate act as an assistant to measure the levels of OCD symptoms the participants presented during each day of the one-week period of experimentation. The participants did not disclose with the assistants whether they had taken the Task during the day. The order of conditions was randomly assigned each day. The dependent variable, levels of OCD symptoms, was then measured by the assistants on both experimental and control day at night using the same obsessive-compulsive symptoms scale described above in the correlational study.

2.5 Experimental Study Planned Analyses

To assess the statistical significance of differences seen in levels of OCD symptoms on the high parietal cortex functioning experimental days vs. the low parietal cortex functioning control days, Student's *t*-tests were performed. We performed *t*-tests separately for each participant as well as using data pooled across all of the participants. For the *t*-tests using pooled data, in addition to using the raw data, we also performed *t*-tests after we had first transformed the data from each participant into *z*-scores in order to standardize differences in averages and variability seen between the participants in their data and thus make them more comparable. An average difference between conditions was considered statistically significant if, using a one-tailed distribution (i.e., to determine if there is a difference between groups in a specific direction), the probability of its random occurrence (p) was $< .05$ (i.e., less than 5% of the time expected by chance alone).

3. Results

3.1 Correlational Study Results

As shown in Table 1, parietal cortex function was significantly correlated with levels of OCD symptoms; however, our predicted direction of relationship between

the variables - that decreased parietal cortex functioning would increase OCD symptoms - , was not confirmed. Although not being statistically significant for both participants (for one, $r = .56$ and $p = .20$, and for the other, $r = .93$ and $p = .0009$), frequency of OCD symptoms was significantly positively correlated with parietal cortex functioning using both pooled raw data ($r = .67$, $p = .006$; see Figure 3) and pooled standardized data ($r = .74$, $p = .001$; see Figure 4). In contrast, no statistically significant correlations were not found between levels of dopamine using both participant's data (for one, $r = -.16$ and $p = .74$, and for the other, $r = .98$ and $p = 1.42E-06$), pooled raw data ($r = .24$, $p = .41$; see Figure 1), or pooled standardized data (for levels of dopamine, $r = .41$, $p = .14$; see Figure 2). Similarly, frontal cortex functioning and frequency of OCD symptoms were not significantly correlated using any single participant's data (all $r \leq .69$, all $p \geq .08$), pooled raw data ($r = .23$, $p = .44$, see Figure 5), or pooled standardized data ($r = .49$, $p = .07$, see Figure 6). Based on a comparison of the correlation coefficients using either the pooled raw data or the pooled standardized data, parietal cortex functioning showed the strongest correlation with frequency of OCD symptoms.

3.2 Experimental Study Results

As shown in Table 2 & 3, no significant differences were found in levels of OCD symptoms between the high parietal cortex functioning experimental condition (Spatial Cueing Task) and low parietal cortex functioning control condition. Statistically significant differences between these conditions were not seen using any single participant's data (all $p \geq .15$), pooled raw data ($p = .23$; see Figure 7), ($p = .35$; see Figure 9), or pooled standardized data ($p = .35$; see Figure 8), ($p = .23$, see Figure 10).

4. Discussion

4.1 Summary of Results

Based on previous research, we hypothesized that decreases in parietal cortex functioning (Hypothesis #1), decreases in frontal cortex functioning (Hypothesis #2), and decreases in levels of dopamine (Hypothesis #3) would be followed by increases in OCD symptoms. Data pooled across participants in our correlational study showed a significant correlation between parietal cortex functioning and levels of OCD symptoms; however, it did not support the predicted direction of relationship between increased OCD symptoms and decreased parietal cortex functioning (Hypothesis #1). Additionally, the data of our correlational study did not show a significant correlation between levels of OCD symptoms and frontal cortex functioning (Hypothesis #2), or between levels of OCD symptoms and levels of dopamine (Hypothesis #3). And finally, the results of our experimental study were not able to establish a causal role of the parietal cortex functioning upon levels of OCD symptoms.

4.2 Relation of Results to Past Research

The strong relationship in our correlational study between OCD symptoms and parietal cortex functioning is in line with previous research; however, the direction of the variables found in our study is the opposite of the direction found in past studies, with increased parietal cortex functioning being associated with decreased levels of OCD symptoms. Liu et al. (2021) found that the interconnection between the

frontal-parietal cortex and both the basal ganglia and the cerebellum was weaker in their OCD group than in the health control group. Based on these results, the researchers suggested that individuals with OCD have an impaired frontal-parietal interconnectivity. While Liu et al. (2021) had two groups, participants that presented OCD symptoms and a control group, and utilized a fMRI scan to measure the whole-brain interconnectivity, in our correlational and experimental studies we longitudinally assessed parietal cortex functioning in two college student who were not OCD patients and measured/manipulated parietal cortex functioning indirectly through recording the average reaction time for the valid trial condition of the Spatial Cueing Test. Therefore, our measuring of the parietal cortex functioning, rather than its interconnectivity with other parts of the brain, might have influenced the differences in the results between our study and the research by Liu et al. (2012). Furthermore, taking the Spatial Cueing Task in our high parietal cortex functioning experimental condition might also have not been a strong enough stimulator of the parietal cortex, as the task takes a short period of time, and moderate effort to be completed; thus it is reasonable that taking the test in the experimental condition and not taking it in the control condition produced minimal effects on levels of OCD symptoms. Finally, having assistants with different levels of proximity with the participants measure the latter's levels of OCD, basing their ratings on subjective interpretations of the behaviours of participants, could have also influenced the lack of a causal relationship found in the experimental study. In future OCD studies, researchers should specifically measure the interconnectivity between the frontal and parietal cortex, as it was found to be correlated with OCD symptoms, rather than whole-brain connectivity, or the activity of the cortexes separately.

The lack of a relationship between frontal cortex activity and OCD symptoms found in our correlational study differs from previously published work. Bohon et al. (2020) found that increased activity in the brains' right frontal pole and increased perseverative errors were seen in OCD participants when compared to a healthy control group. Based on these results, the researchers suggested that mental skill tasks, such as the WCST, trigger more intense frontal brain activity in OCD participants. The methodology of our correlational study differed from that of the Bohon et al. (2020) study in two major ways that might account for the discrepant results. First, a difference between the studies is the number of other tests completed within one study. While our study used the Spatial Cueing Task, eye blink test and the Berg's Card Sorting Task, Bohon et al. (2020) administered just the WCST. Future studies could examine whether the effects of the test order, in which it was administered, could affect frequency of OCD symptoms. Secondly, the differences between the studies is the sample size, which could have affected their findings. Bohon et al. (2020) examined 14 adolescents with weighted restored Anorexia Nervosa and 11 adolescents with OCD in comparison to a healthy control group, while our correlational study had only a small sample size, being two participants, and a restricted variety of sample groups. We recommend that future studies test a larger sample size and ensure the tests' order is controlled for.

Our correlational study failed to confirm the relationship between frequency of OCD symptoms and dopamine levels found by previous research. Viol et al. (2020) found that venipuncture increased dopamine levels in patients that met DSM-IV criteria for

OCD to a greater extent after they had underwent psychotherapy. Based on these results, the researchers suggested that through psychotherapy treatment, OCD patients' dopamine levels increase. In contrast, our participants did not find that frequency of OCD symptoms affected dopamine level. The methodology of our correlational study differed from that of the Viol et al. (2020) study in two major ways that might account for the discrepant results. First, there were differences between the studies in how they measured dopamine levels. Our correlational study relied only upon self assessment. Future studies should test whether the objectively verified aspects of dopamine levels outlined by Viol et al. (2020), but not subjective self assessment, predict dopamine levels. Second, there differences between the studies in when they measured dopamine levels could affect their findings. While Viol et al. (2020) examined levels of dopamine in OCD participants before versus after receiving psychotherapy, while we measured natural fluctuations in dopamine levels across days.

4.3 Implications of Results

Possible practical applications of our current findings are that parietal cortex functioning, frontal cortex activity and dopamine levels are not responsible for OCD symptoms. For instance, increased parietal cortex functioning, decreased frontal cortex activity and increased dopamine levels did not result in decreased OCD symptoms. We originally conducted the current studies to find ways to reduce our obsessive-compulsive tendencies based on the knowledge of the biological mechanisms of OCD. Unfortunately, due to the non-significant results from our correlation and experimental studies, strategies to reduce these tendencies cannot be offered. It remains for future studies to determine all of the biological mechanisms involved in obsessive-compulsive disorders, and to eventually create more objective methods to measure OCD symptoms.

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Table 1

Correlations for Study Variables

Variables	Participant #1		Participant #2		Pooled raw data		Pooled standardized data	
	r	*n*	*r*	*n*	*r*	*n*	*r*	*n*
Levels of Dopamine & Levels of OCD Symptoms	-.16	7	.98*	7	.24	14	.41	14
Parietal Cortex Functioning & Levels of OCD Symptoms	.56	7	.93*	7	.67*	14	.74*	14
Frontal Cortex Functioning & Levels of OCD Symptoms	.29	7	.69	7	.23	14	.49	14

* $p < .05$.

Table 2

Descriptive Statistics for levels of OCD symptoms (measured by assistant) Across Different Parietal Cortex Functioning Conditions

Condition	Statistic	Participant #1	Participant #2	Pooled raw data	Pooled standardized data
High Parietal	*M*	6.0	2.5	4.2	-0.5
Cortex	*SD*	1.4	0.7	2.2	0.7
Functioning	*n*	2	2	4	4
Low Parietal	*M*	7.0	3.6	5.3	0.3
Cortex	*SD*	1.0	1.5	2.1	0.9
Functioning	*n*	3	3	6	6

Note. *M*, *SD*, and *n*, represent mean, standard deviation, and sample size, respectively. Levels of OCD symptoms were measured by the experimenter on a 0-10 scale of amount of symptoms (0 = no symptoms to 10 = multiple symptoms).

* $p < .05$ for comparison of high parietal cortex functioning condition with its respective low parietal cortex functioning condition.

Table 3

Descriptive Statistics for levels of OCD symptoms (measured by experimenter) Across Different Parietal Cortex Functioning Conditions

Condition	Statistic	Participant #1	Participant #2	Pooled raw data	Pooled standardized data
High Parietal	*M*	7.5	4.5	6.0	-0.2
Cortex	*SD*	0.7	2.1	2.1	1.0
Functioning	*n*	2	2	4	4
Low Parietal	*M*	8.0	5.0	6.5	0.2
Cortex	*SD*	1.0	1.0	1.8	0.9
Functioning	*n*	3	3	6	6

Note. *M*, *SD*, and *n*, represent mean, standard deviation, and sample size, respectively. Levels of OCD symptoms were measured by the experimenter on a 0-10 scale of amount of symptoms (0 = no symptoms to 10 = multiple symptoms).

* $p < .05$ for comparison of high parietal cortex functioning condition with its respective low parietal cortex functioning condition.

Figure 1

Association Between Levels of Dopamine and Levels of OCD Symptoms Using Pooled Raw Data

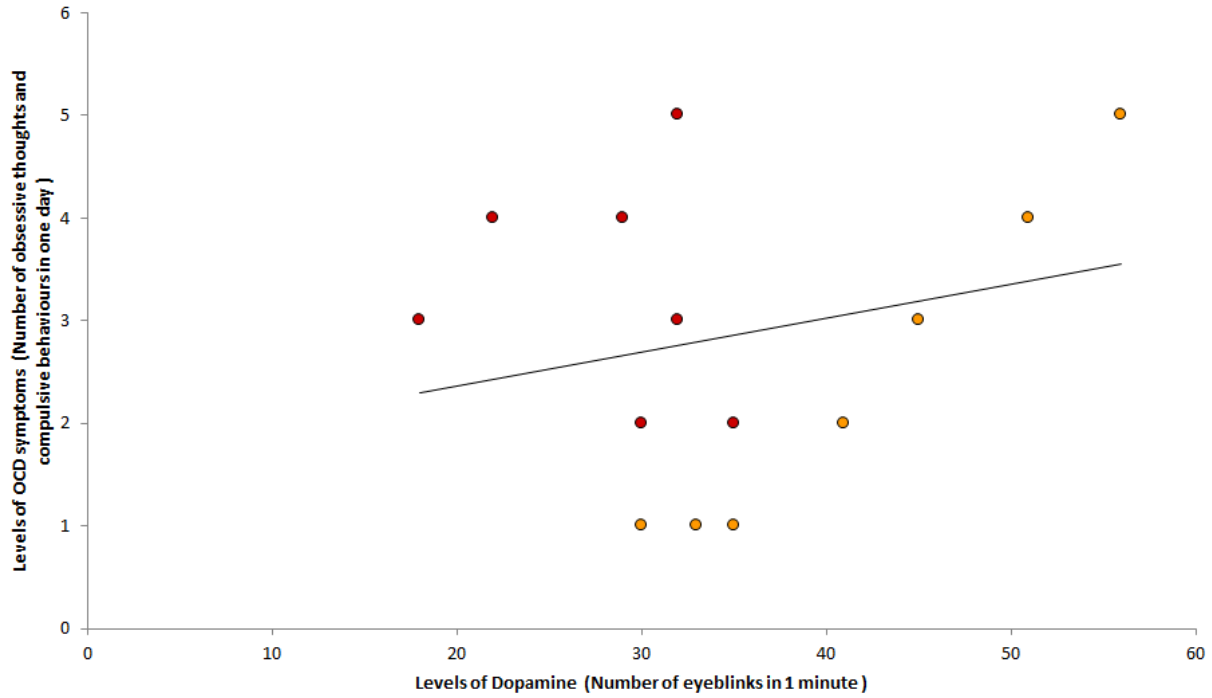


Figure 2

Association Between Levels of Dopamine and Levels of OCD Symptoms Using Pooled Standardized Data

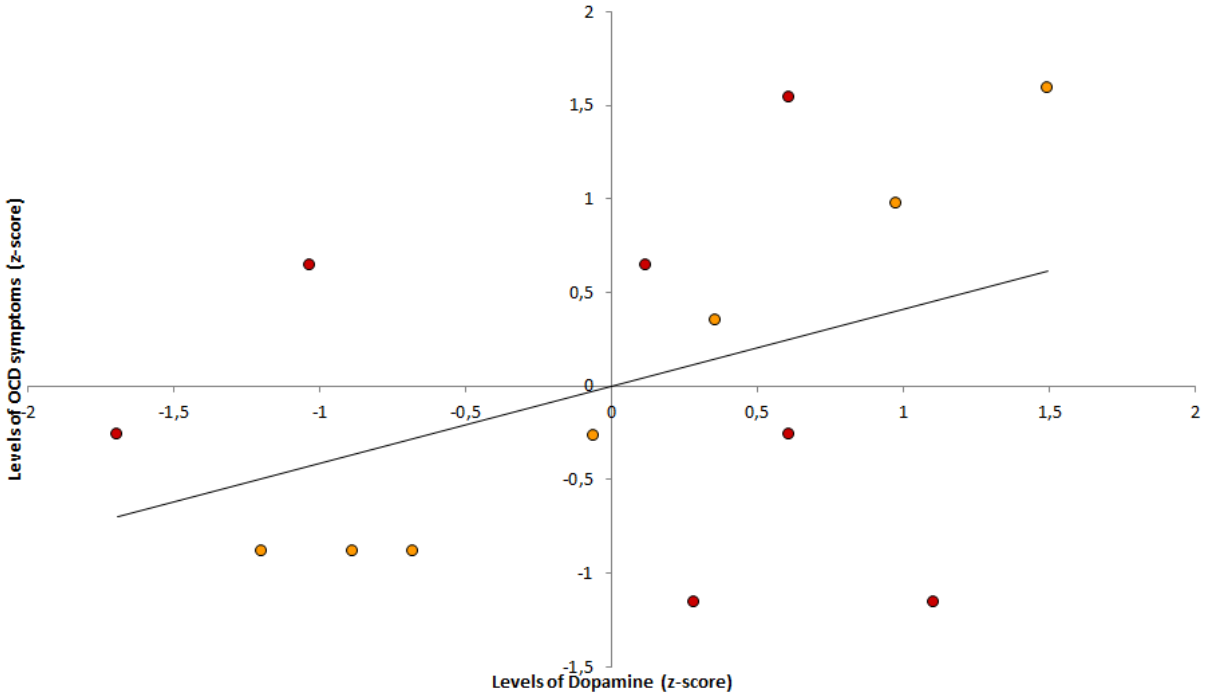


Figure 3

Association Between Parietal Cortex Functioning and Levels of OCD Symptoms Using Pooled Raw Data

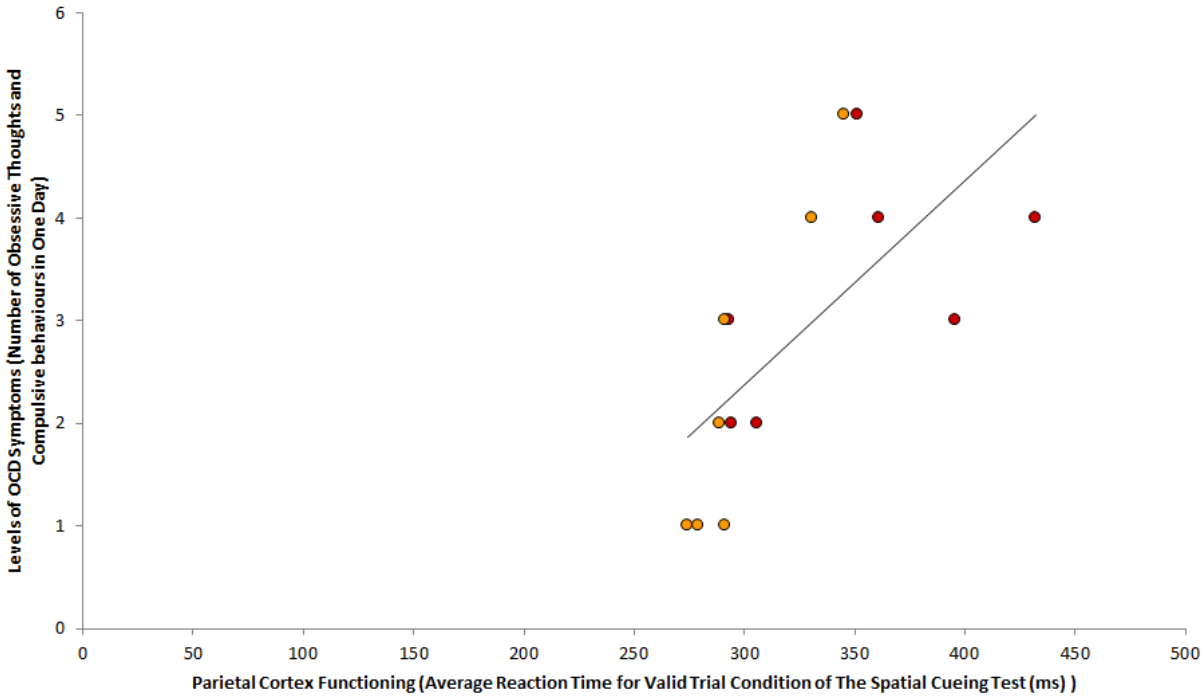


Figure 4

Association Between Parietal Cortex Functioning and Levels of OCD Symptoms Using Pooled Standardized Data

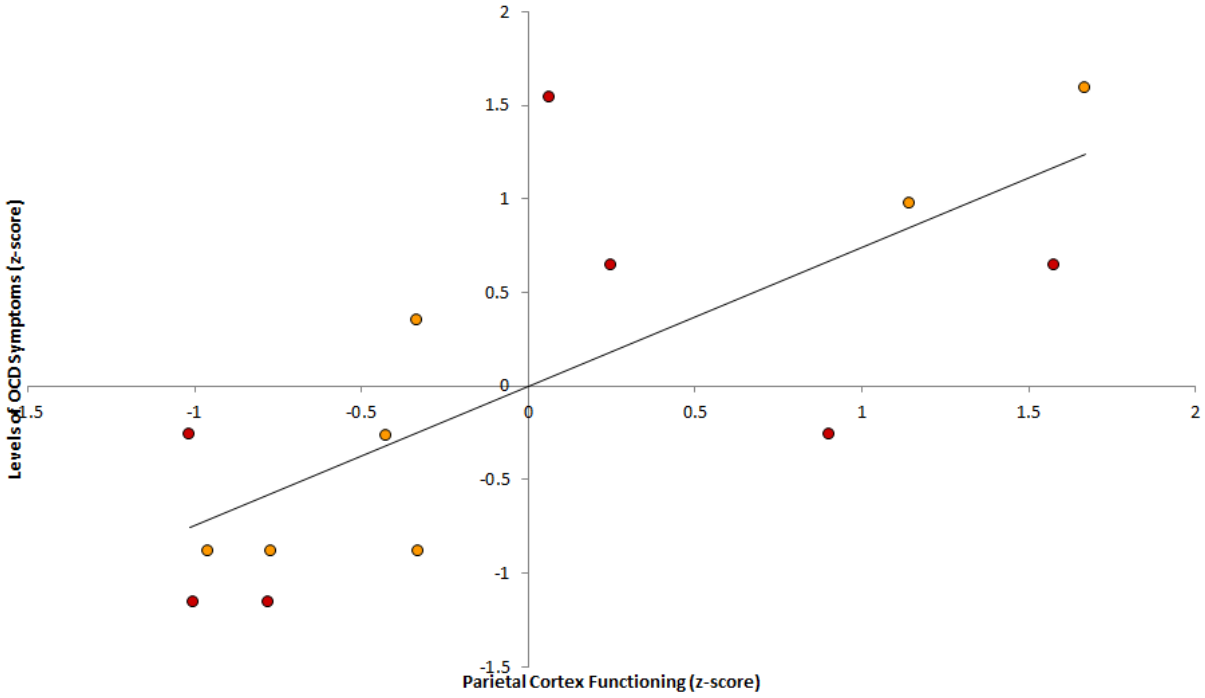


Figure 5

Association Between Frontal Cortex Functioning and Levels of OCD Symptoms Using Pooled Raw Data

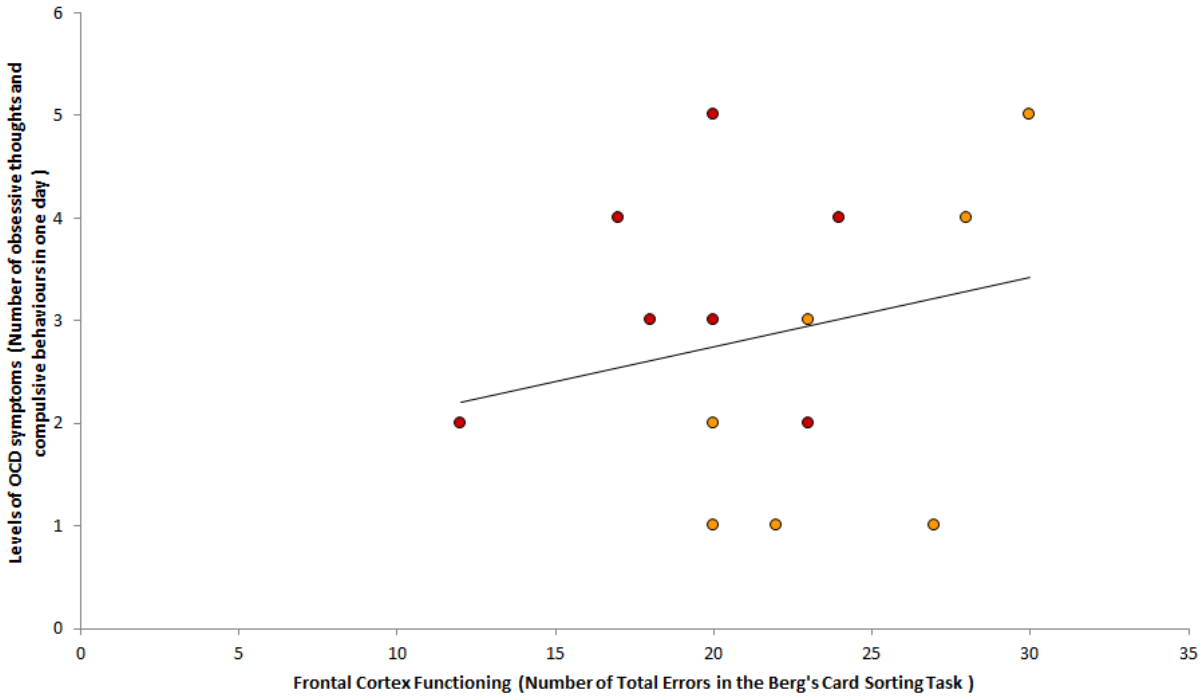


Figure 6

Association Between Frontal Cortex Functioning and Levels of OCD Symptoms Using Pooled Standardized Data

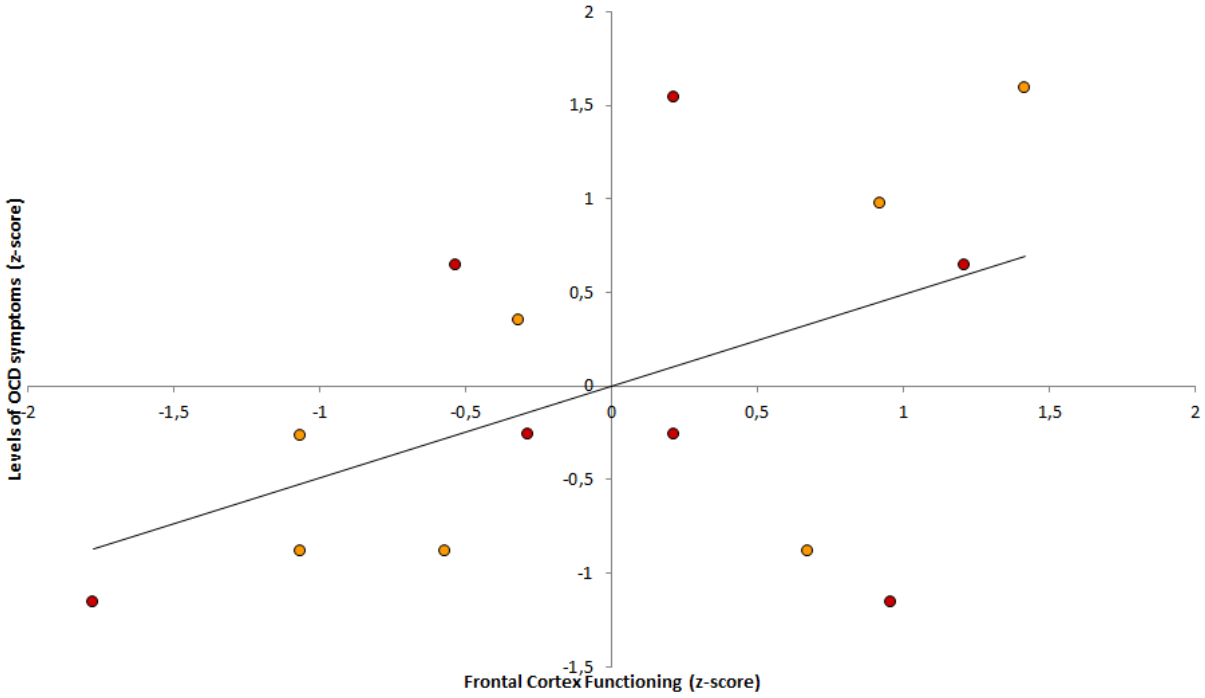
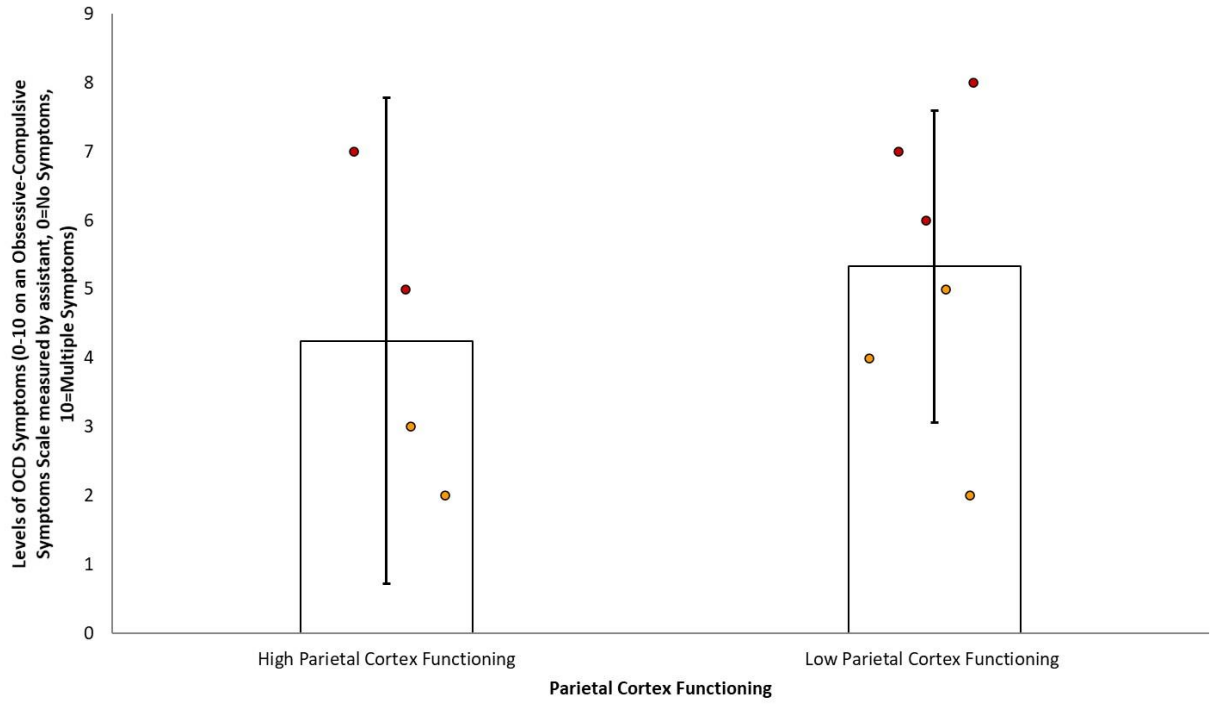


Figure 7

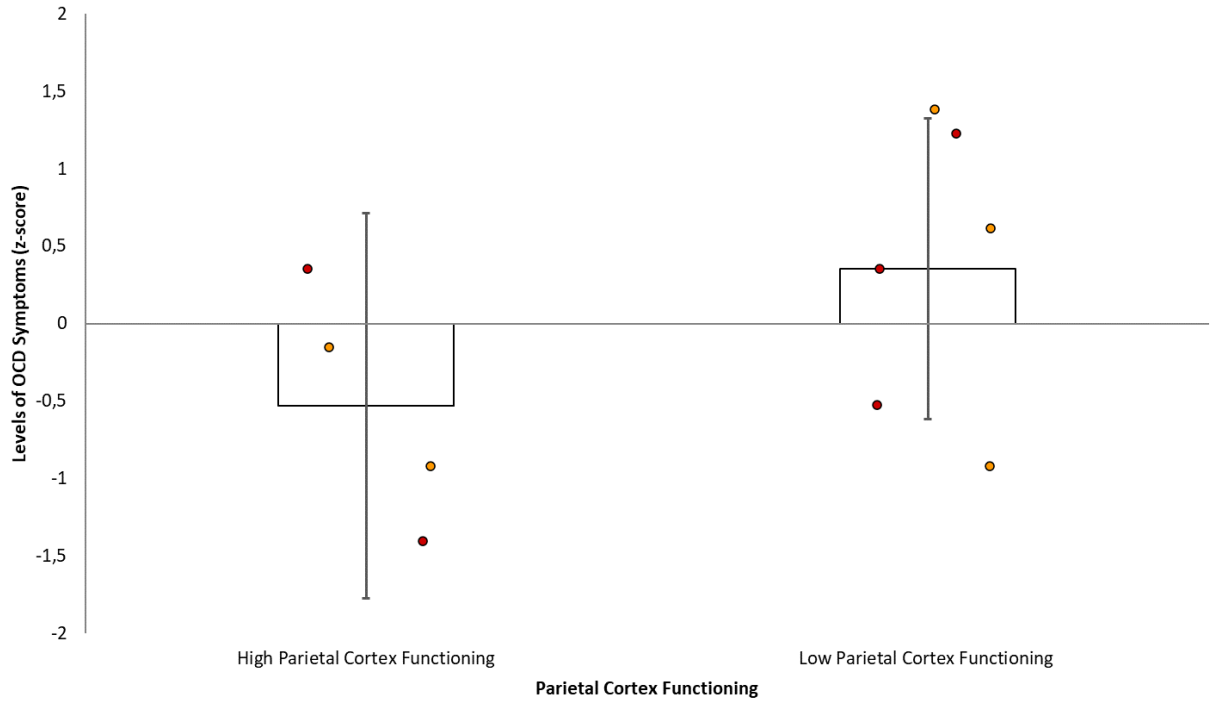
Average Levels of OCD Symptoms (Measured By Assistant) Across Different Parietal Cortex Functioning Conditions Using Pooled Raw Data



Notes. Levels of OCD symptoms scores are shown for high parietal cortex functioning and low parietal cortex functioning conditions using pooled raw data from all participants. Errors bars show ± 95% confidence levels. Overlapping scatterplot shows data from each participant. Marker colour differentiates participants: red = participant #1, and orange = participant #2.

Figure 8

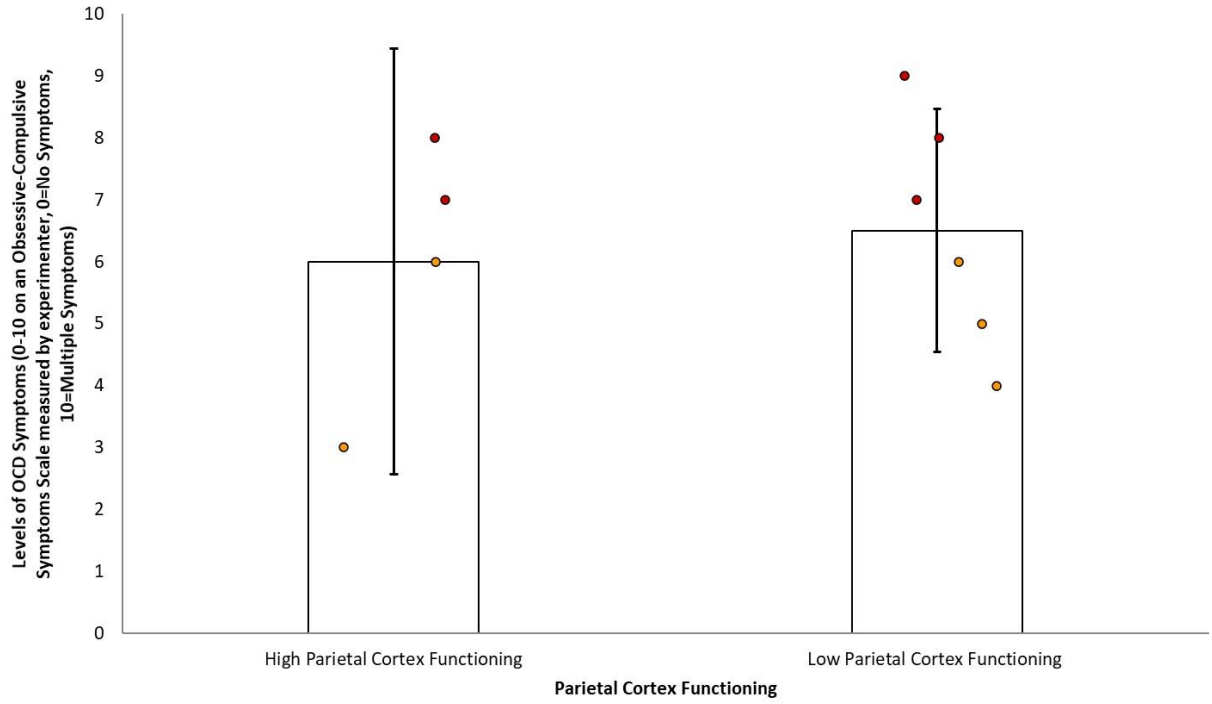
Average OCD Symptoms (Measured By Assistant) Across Different Parietal Cortex Functioning Conditions Using Pooled Standardized Data



Notes. Levels of OCD symptoms scores are shown for high parietal cortex functioning and low parietal cortex functioning conditions using pooled standardized data from all participants. Errors bars show ± 95% confidence levels. Overlapping scatterplot shows data from each participant. Marker colour differentiates participants: red = participant #1, and orange = participant #2.

Figure 9

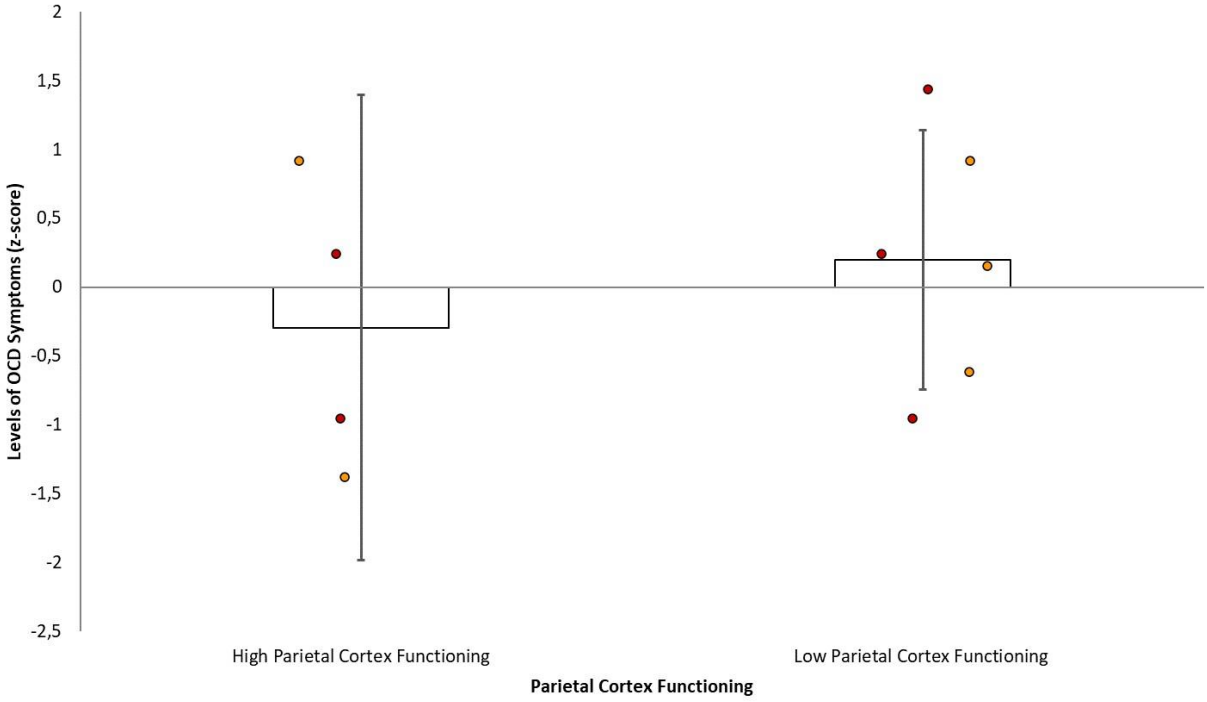
Average Levels of OCD Symptoms (Measured By Experimenter) Across Different Parietal Cortex Functioning Conditions Using Pooled Raw Data



Notes. Levels of OCD symptoms scores are shown for high parietal cortex functioning and low parietal cortex functioning conditions using pooled raw data from all participants. Errors bars show ± 95% confidence levels. Overlapping scatterplot shows data from each participant. Marker colour differentiates participants: red = participant #1, and orange = participant #2.

Figure 10

Average Levels of OCD Symptoms (Measured By Experimenter) Across Different Parietal Cortex Functioning Conditions Using Pooled Standardized Data



Notes. Levels of OCD symptoms scores are shown for high parietal cortex functioning and low parietal cortex functioning conditions using pooled standardized data from all participants. Errors bars show ± 95% confidence levels. Overlapping scatterplot shows data from each participant. Marker colour differentiates participants: red = participant #1, and orange = participant #2.