The Relation Between Obsessive–Compulsive Traits, Frontal Lobe Functioning, and Visual Recall

KELLI D. NELSON

NOAH V. CLAYTON

KEVIN R. BYRD*

University of Nebraska at Kearney

The authors of the current study administered the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993), the Maudsley Obsessional–Compulsive Inventory (MOCI; Hodgson & Rachman, 1977), and the Rey-Osterrieth Complex Figure (Lezak, 1995) to 32 undergraduate students in order to test a model in which the effects of frontal lobe functioning on visual recall are mediated by obsessive–compulsive traits. Frontal lobe functioning predicted scores on 1 of the 4 MOCI subscales (i.e., slowness) and visual recall. However, obsessive–compulsive traits did not mediate the relation between frontal lobe dysfunction and visual recall as predicted. We present a revised model of the relation between frontal lobe functioning, visual recall, and obsessive–compulsive traits.

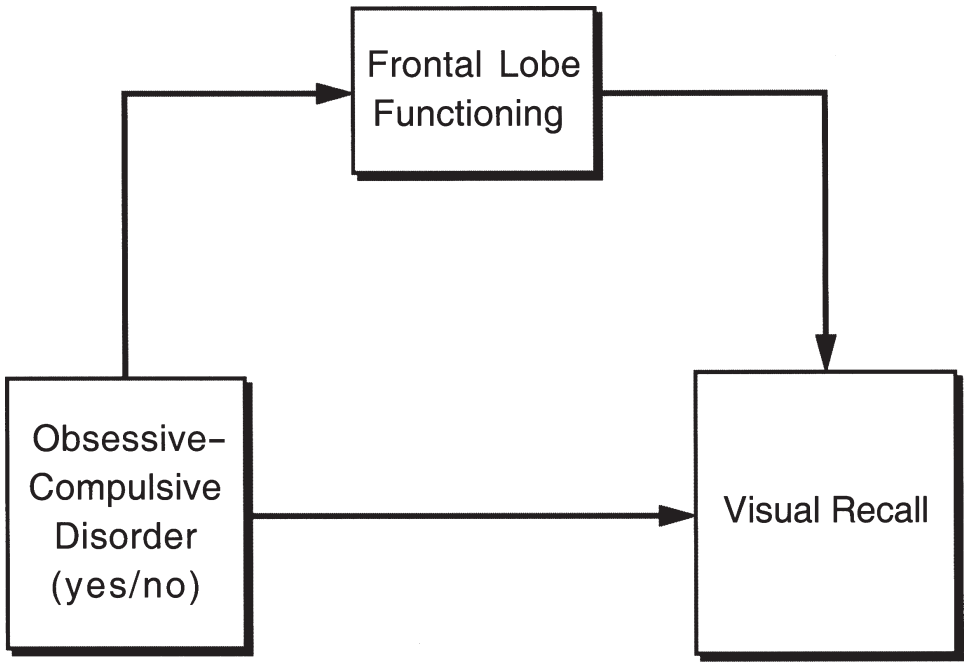
OBSESSIVE–COMPULSIVE DISORDER (OCD) consists of intrusive, anxiety-provoking thoughts or images (obsessions) accompanied by repetitive behaviors or mental acts (compulsions) that the person feels driven to perform in response to the obsessions, although there is little or no realistic connection between the two (American Psychiatric Association, 1994). Previous research suggests that abnormalities in frontal lobe functioning contribute to symptoms of OCD (Kaplan & Sadock, 1998; Rapaport, Gill, & Schafer, 1968), and that OCD is a source of visual recall deficits (Kozak, Foa, & McCarthy, 1988; Savage et al., 1999). The purpose of the present study was to test a model in which obsessive–compulsive traits mediate the relation between frontal lobe functioning and visual recall. One of the earliest reports to link OCD to frontal lobe deficits was presented by Rapaport et al. (1968) who noted that individuals with OCD are somewhat disadvantaged in their ability to change cognitive set or strategy, which was identified as a frontal lobe function by Milner (1963). More recently, Kaplan and Sadock (1998) reported an increase of metabolism and blood flow in the frontal lobes of individuals diagnosed with OCD.

Because OCD is associated with visual integration deficits as well as frontal lobe dysfunction, some researchers have attempted to account for the co-occurrence of these three factors through models by attributing the visual integration deficits to the underlying frontal lobe dysfunction. Meyer and Deitsch (1996) reported that individuals with OCD have a meticulous approach to the block design and object assembly subtests of the Wechsler Adult Intelligence Scale–Revised (i.e., a scale that assesses visual construction abilities). This approach occasionally leads to a loss of speed points but seldom results in inaccurate performance. This finding suggests that the lower scores of individuals with OCD are related more to performance strategy (ostensibly a frontal lobe function) than to neurological deficits in visual processing per se (parietal or occipital lobe functioning). In addition, Kozak et al. (1988) associated slowness on visual construction tests with difficulty in shifting spatial strategies, implicating frontal lobe dysfunction as a source of compulsive slowness, which may in

 *Faculty Supervisor

FIGURE 1

Mediated model of visual recall deficits associated with OCD, from Savage et al. (1999).



turn affect performance on visual construction tasks.

Savage et al. (1999) found that participants with OCD had significantly lower levels of recall on visual memory tests than a non-OCD control group. In agreement with Rapaport et al. (1968), these authors attributed the visual recall deficits to frontal lobe dysfunction, particularly with difficulties in shifting spatial set. On the basis of these findings, Savage et al. proposed a model of visual memory deficits in persons with OCD in which frontal lobe functioning mediates the relation between OCD and visual memory performance (see Figure 1). This model, which posits OCD as a source of frontal lobe deficits, unduly reifies a purely descriptive concept. A diagnostic label such as OCD has no explanatory power apart from the known pathogenesis and the individual symptoms to which it refers. The current authors therefore revised this model by positing frontal lobe dysfunction as a source of four specific, measurable, obsessive–compulsive traits, which in turn account for visual recall deficits because of the disruptive effect these traits have on processing and organizing visual information (see Figure 2).

Previous research that attributed OCD to frontal lobe deficits relied completely on informal or indirect indicators of frontal lobe functioning, such as unstandardized measures of "organizational strategies" (Savage et al., 1999). In order to test the revised model depicted in Figure 2, we used a more widely accepted measure of frontal lobe functioning, the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993). In addition, we assessed four obsessive–compulsive traits (slowness, checking, cleaning, and doubting) with the Maudsley Obsessional–Compulsive Inventory (Hodgson & Rachman, 1977). Following Savage et al. (1999), we assessed visual recall with the Rey-Osterrieth Complex Figure (Lezak, 1995).

The body of research previously cited led us to predict that frontal lobe function would be significantly related to one or more obsessive–compulsive traits. In accordance with the model adapted from Savage et al. (1999; see Figure 2), we predicted that obsessive–compulsive traits (i.e., checking, doubting, cleaning, and slowness) would mediate the relation between frontal lobe function and visual memory.

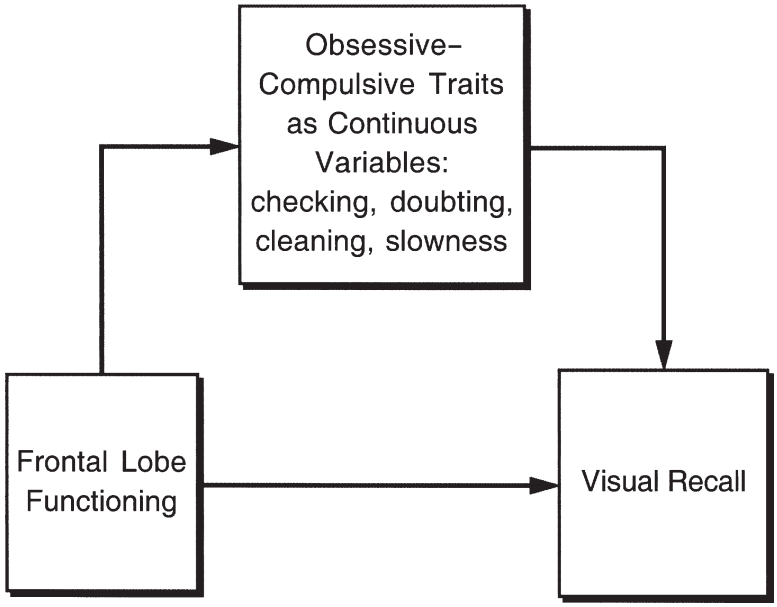
Method

Participants

Thirty-two students at the University of Nebraska at Kearney (16 men and 16 women) received extra credit in exchange for their participation in this study.

FIGURE 2

Revised model of visual recall deficits associated with OCD traits.



The age range of participants was from 18 to 45 years (M = 20.6).

Measures

Maudsley Obsessional–Compulsive Inventory (MOCI). The MOCI (Hodgson & Rachman, 1977) consists of 30 true-or-false questions from which four subscales are derived (i.e., checking, slowness, doubting, and cleaning). The reliability coefficients reported below are from Hodgson and Rachman (1977). The subscale of checking contains 7 items with a reported alpha of .70. A representative item is: "I do not check letters over and over again before mailing them" (reverse scored). The subscale of slowness consists of 7 items with a reported alpha of .70. A representative item is: "I do not usually count when doing a routine task" (reverse scored). The doubting subscale consists of 7 items with a reported alpha of .70. A representative item is: "I usually have serious doubts about the simple everyday things I do." The subscale of cleaning consists of 11 items with a reported alpha of .80. A representative item is: "I don't worry unduly about contamination if I touch an animal" (reverse scored).

Rey-Osterrieth Complex Figure (ROCF). The ROCF is a drawing that the participant reconstructs with paper and pencil while it is present and later from memory. It is comprised of 18 units that can be individually scored and summed to derive a measure of visual recall (Lezak, 1995). Two points are scored for each unit that is correctly placed and replicated. One point is scored for correct placement, even if it is poorly replicated. One point is also scored for correct replication, even if the unit is poorly placed. One half of a point is scored for units that are poorly placed and poorly replicated. Zero points are given for absent or unrecognizable units. This procedure yields a potential range of 0–36 points. Rapport, Charter, Dutra, Farchione, and Kingsley (1997) report an internal consistency coefficient (alpha) of .90 for the Lezak (1995) system of scoring the ROCF in a delayed-recall procedure (the procedure used in the current study and described below).

Wisconsin Card Sorting Test (WCST). The WCST (Heaton et al., 1993) is a measure of executive function that requires the individual to adjust his or her problem-solving strategy across changing stimulus conditions. The test consists of 4 stimulus cards and 128 response cards that depict figures of varying forms (i.e., crosses, circles, triangles, or stars), colors (i.e., red, blue, yellow, or green), and quantities (i.e., one, two, three, or four). The WCST challenges the participant to find and main-

TABLE 1

Psychometric Statistics

Variable	*M*	*SD*	Number of items	α
Slowness–revised	2.59	1.39	4	.72
Doubting	2.69	1.75	7	.65
Cleaning	4.47	2.70	11	.74
Delayed visual recall	21.64	6.60		
Total WCST errors	33.94	24.25		

tain the correct strategy for matching the response cards to the stimulus cards (according to form, color, or number) based on feedback from the examiner as to whether each attempted match is correct or incorrect. The correct sorting strategy changes intermittently without any signal to the participant, and the participant then must figure out the new, correct strategy. We operationalized frontal lobe functioning as the number of sorting errors on this test. This measure, which reflects the ability to shift cognitive strategies, has been shown to be sensitive to frontal lobe deficits (Heaton et al., 1993; Lombardi et al., 1999). Reliability is reported in terms of generalizability coefficients (Heaton et al., 1993, pp. 40–41). The coefficient for the measure used in this study (sorting errors) was .71. For a complete discussion of generalizability theory and procedures, see Shavelson, Webb, and Rowley (1989). For specific administration instructions see Heaton et al. (1993, pp. 5–7).

Procedure

After we obtained informed consent from the participants, the MOCI was administered. Once the participants indicated they were finished, we administered the ROCF. Participants were instructed to recreate the figure with pencil and paper while it remained in front of them. When the participant completed the drawing it was replaced with a blank piece of paper, and instructions were given to redraw the figure from memory. A final drawing of the figure was completed from memory after a 30-min delay, which was the sample scored for the data analyses. The WCST (Heaton et al., 1993) was administered during this 30-min interval.

Design

As previously stated, the purpose of this study was to test a model in which obsessive–compulsive traits mediate the relation between frontal lobe function and visual recall. Baron and Kenny (1986) proposed that in order to test for mediation, three regression equations are required: the mediator is regressed on the independent variable; the dependant variable is regressed on the independent variable; and the dependant variable is regressed on both the independent variable and the mediator. In order to establish mediation the following four conditions must be met:

TABLE 2

Correlation Matrix for Variables Used in Multiple Regressions

	1	2	3	4	5	6
1. Sex		–.39*	.38*	–.61**	–.22	–.15
2. Cleaning			–.02	.62**	.00	.24
3. Doubting				–.27	–.45**	.16**
4. Slowness					–.11	.42*
5. Delayed recall						–.45**
6. Total errors						

*$p < .05$. **$p < .01$.

TABLE 3

Multiple Regression for OCD Slowness (R^2 = .49)

Predictor	β	*B*	*SE B*	Significance
Sex	–.56	–1.65	.40	$p < .01$
WCST total errors	.34	.02	.01	$p < .02$

the independent variable must be significantly related to the mediator variable; the independent variable must be significantly related to the dependent variable; the mediator must be significantly related to the dependent variable; and the beta weight of the independent variable in the third equation must be less than that of the second equation.

We tested a separate mediated model for each obsessive–compulsive trait (posited as mediating variables): doubting, checking, cleaning, and slowness. The same "independent" and "dependent" variables were used for each of these models. The "independent" variable was the total number of errors on the WCST (frontal lobe functioning) and the "dependent" variable was the delayed recall score from the ROCF (visual recall; Lezak, 1995).

Results

Reliability (alpha) coefficients were calculated for each of the four subscales of the MOCI (doubting, checking, slowness, and cleaning). The coefficients in our sample (checking α = .41, cleaning α = .74, slowness α = .32, and doubting α = .65) were not found to be as high as those reported by Hodgson and Rachman (1977). In an attempt to increase the overall reliability of the slowness and checking subscales, we removed items that did not correlate significantly with the overall scale. This procedure increased the alpha coefficients to .72 for slowness and .57 for checking. Because the coefficient for checking remained unacceptably low, it was discarded from further analysis. For the psychometric properties of the scales used in this study, see Table 1.

A correlation matrix was created from the following variables: sex, cleaning, doubting, slowness, gender, delayed recall, and the WCST total errors (see Table 2). Because sex (coded *men* = 0; *women* = 1) correlated significantly with all three MOCI subscales, it was included as a control variable in the multiple regressions used to test the model.

In the doubting-mediated model, the first regression of doubting on total WCST errors was not significant ($p > .20$, β = .23). This nonsignificant finding ruled out a mediated model for the symptom of doubting according to the criteria of Baron and Kenny (1986). In the cleaning-mediated model, the first regression of cleaning on the total WCST errors was not significant ($p > .22$, β = .21). This nonsignificant finding also ruled out a mediated model for the symptom of cleaning.

In the slowness-mediated model, the first regression of slowness on WCST errors was significant ($p < .01$, β = .36; see Table 3). The second regression for testing the model, delayed visual recall on total WCST errors, was also significant ($p < .01$, β = –.51; see Table 4). In the third regression of delayed visual recall on total WCST errors and slowness, WCST errors was significant ($p < .01$, β = –.44), but slowness was not ($p > .48$, β = –.16; see Table 5). This nonsignificant finding for slowness in the third regression ruled out the proposed mediated model for the obsessive–compulsive trait of slowness.

These findings suggest that a relation exists between frontal lobe functioning and visual recall, and between frontal lobe functioning and slowness, but that slowness does not mediate the relation between frontal lobe function and visual recall. These findings led to the post hoc formation of a new model that

TABLE 4

Multiple Regression for Delayed Visual Recall Without the Mediating Variable (R^2 = .29)

Predictor	β	*B*	*SE B*	Significance
Sex	–.29	–3.81	2.10	$p > .07$
WCST total errors	–.49	–.13	.04	$p < .01$

TABLE 5

Multiple Regression for Visual Recall With the Mediating Variable (R^2 = .30)

Predictor	β	*B*	*SE B*	Significance
Sex	–.38	–4.98	2.65	$p > .07$
WCST total errors	–.44	–.12	.05	$p < .02$
OCD slowness	–.16	–.71	.98	$p > .47$

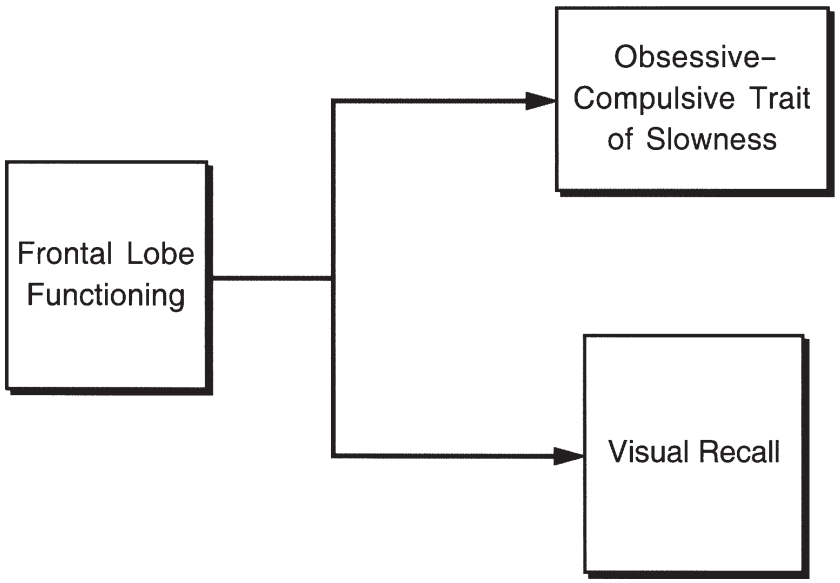
posits frontal lobe dysfunction, measured by the total number of errors on the WCST as a direct influence on both the obsessive–compulsive trait of slowness as measured by the MOCI and the visual recall deficits as measured by the Rey-Osterrieth Complex Figure (see Figure 3).

Discussion

We revised the mediated model proposed by Savage et al. (1999) due to the implausibility of positing the diagnosis of OCD as a causal variable for the prediction of frontal lobe dysfunction and visual recall deficits (see Figure 1). It was hypothesized that our results would be consistent with this revised model (Figure 2) in that the relation between frontal lobe dysfunction on visual recall deficits would be statistically mediated by obsessive–compulsive traits. Our results indicated that frontal lobe functioning predicts visual recall, as well as obsessive–compulsive slowness, but that slowness does not mediate the relation between frontal lobe measures and visual recall as predicted. This pattern of results suggests a nonmediated, direct-effect model in which frontal lobe function affects both obsessive–compulsive slowness and visual recall. Visual recall is not associated with slowness when the relation between visual recall and frontal lobe functioning is statistically controlled (see Table 5); therefore, slowness is not a mediator (see the criteria for mediation described above). It should be noted that the WCST taps only one frontal lobe

FIGURE 3

Final model of visual recall deficits associated with OCD traits.



function (knowing when to maintain or shift strategies), and that other frontal lobe operations might also influence obsessive–compulsive traits or symptoms.

A deficit in the ability to shift cognitive strategies as measured by the WCST is a form of perseveration, which is defined as the "persisting response to a previous stimulus after a new stimulus has been presented" (Kaplan & Sadock, 1998, p. 282). Perseveration, a symptom of frontal lobe dysfunction (Freedman, Black, Ebert, & Binns, 1998; Lombardi et al., 1999; Milner, 1963; Na et al., 1999), may partially account for obsessive–compulsive slowness through its interference with problem solving and the completion of tasks. At the same time, perseverative tendencies might also account for the relative deficits in visual recall performance among people with obsessive–compulsive tendencies. Multiple strategies are needed for complete reconstruction on visual recall tasks (e.g., looking at the whole, looking at the parts, looking at two parts in relation to one another, etc.); therefore, perseveration of any single reconstructive strategy would plausibly impair visual recall performance.

It should be noted that the use of a college population is a weakness in the current study. Our results are only suggestive, and the resulting model should not be applied to clinical populations without further research. There are several reasons for this caution. Primarily, there could very well be qualitative (not only quantitative) differences between our participants and actual individuals with OCD. Further, OCD was treated in the current study as a continuous variable, whereas standard clinical practice recognizes OCD as a dichotomous variable (i.e., as a diagnosis). The use of DSM-IV criteria for OCD also would have improved the external validity of this study. Therefore, the relevance of our study for actual individuals with OCD is questionable pending replication with clinically diagnosed participants.

Savage et al. (1999) initiated the experimental investigation of the relation between OCD, frontal lobe dysfunction, and visual recall deficits. Our results indicate that the relation between frontal lobe dysfunction and visual recall deficits is not mediated by obsessive–compulsive traits as predicted. Instead, the effect of frontal lobe dysfunction on visual recall appears to be a direct one, as is its effect on obsessive–compulsive slowness. These current findings show that future research should consider specific OCD symptoms in an actual clinical population.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182.

Freedman, M., Black, S., Ebert, P., & Binns, M. (1998). Orbitofrontal function, object alternation and perseveration. *Cerebral Cortex, 8,* 18–27.

Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin Card Sorting Test manual: Revised and expanded.* Odessa, FL: Psychological Assessment Resources.

Hodgson, R. J., & Rachman, S. (1977). Obsessional-compulsive complaints. *Behavior Research and Therapy, 15,* 389–395.

Kaplan, H. I., & Sadock, B. J. (1998). *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences/clinical psychiatry.* (8th ed.). Baltimore: Williams and Wilkins.

Kozak, M. J., Foa, E. B., & McCarthy, P. R. (1988). Obsessive-compulsive disorder. In C. G. Last (Ed.), *Handbook of anxiety disorders* (pp. 87–108). Elmsford, NY: Pergamon Press.

Lezak, M. D. (Ed.). (1995). *Neuropsychological assessment: Part 2. A compendium of tests and assessment techniques.* (3rd ed.). New York: Oxford University Press.

Lombardi, W. J., Andreason, P. J., Sirocco, K. Y., Rio, D. E., Gross, R. E., Umhau, J. C., & Hommer, D. W. (1999). Wisconsin Card Sorting Test performance following head injury: Dorsolateral fronto-striatal circuit activity predicts perseveration. *Journal of Clinical and Experimental Neuropsychology, 21,* 2–16.

Meyer, R. G., & Deitsch, S. E. (1996). *The clinician's handbook: Integrated diagnostics, assessment, and intervention in adult and adolescent psychopathology* (4th ed.). Boston: Allyn & Bacon.

Milner, B. (1963). Effects of different brain lesions on card sorting. *Archives of Neurology, 9,* 90–100.

Na, D. L., Adair, J. C., Kang, Y., Chung, C. S., Lee, K. H., & Heilman, K. M. (1999). Motor perseverative behavior on a line cancellation task. *Neurology, 52,* 1569–1576.

Rapaport, D., Gill, M. M., & Schafer, R. (1968). *Diagnostic psychological testing* (Rev. ed.). New York: International Universities Press.

Rapport, L. J., Charter, R. A., Dutra, R. L., Farchione, T. J., & Kingsley, J. J. (1997). Psychometric properties of the Rey–Osterrieth Complex Figure: Lezak–Osterrieth versus Denman scoring systems. *The Clinical Neuropsychologist, 11,* 46–53.

Savage, C. R., Baer, L., Keuthen, N. J., Brown, H. D., Rauch, S. L., & Jenike, M. A. (1999). Organizational strategies mediate nonverbal memory impairment in obsessive–compulsive disorder. *Biological Psychiatry, 45,* 905–916.

Shavelson, R. J., Webb, N. M., & Rowley, G. L. (1989). Generalizability theory. *American Psychologist, 44,* 922–932.