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THE RELATION BETWEEN COGNITIVE INFLEXIBILITY AND

OBSESSIVE-COMPULSIVE PERSONALITY TRAITS IN ADULTS:

DEPRESSION AND ANXIETY AS POTENTIAL MEDIATORS

by

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Abstract

The current study quantitatively examined the relation between cognitive inflexibility and obsessive-compulsive personality traits and also examined whether depression and anxiety were mediators of that relation. A total of 56 individuals, ages 18 to 40, participated in the study and were recruited from the undergraduate and graduate student body of The University of Southern Mississippi as well as other participants from the University community. Participants completed self-report questionnaires on measures of cognitive inflexibility (Cognitive Flexibility Scale; CFS), depression and anxiety (Depression and Anxiety Stress Scale; DASS), and obsessive-compulsive personality traits (Personality Diagnostic Questionnaire; PDQ-4). It was hypothesized that cognitive inflexibility would relate to obsessive-compulsive personality traits (OCPT). Furthermore, it was predicted that anxiety symptoms (separate from those symptoms that are more core to OCPT) would fully mediate this relation. It was also expected that depression symptoms would at least partially mediate the relation between cognitive inflexibility and OCPT. Although the hypotheses were not fully supported, interesting relations (in the predicted directions) among variables did emerge. Cognitive inflexibility was related to anxiety and depression, the latter of which was further linked to OCPT. However, the full mediational model was not supported. The findings from the current study add to the current literature and have both theoretical and practical implications.

Keywords: cognitive inflexibility; obsessive-compulsive personality traits; depression; anxiety

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The Relation Between Cognitive Inflexibility and Obsessive-Compulsive Personality Traits in

Adults: Depression and Anxiety as Potential Mediators

Several studies have suggested the existence of a relation between executive dysfunction and obsessive-compulsive disorder (OCD; Denys, Sitskoorn, Westenberg, & De Geus, 2007; Fenger et al., 2005; Lawrence et al., 2006). According to Gapin and Etnier (2010), executive function is the cognitive process of maintaining the necessary problem solving skills to reach a desired goal. This process is involved in routine life, academics, and social functioning. An individual suffering from executive dysfunction would exhibit deficits in these areas. Whether the goal is to complete a school project or simply follow step by step directions, an individual with executive dysfunction will often struggle to do so (Fenger et al., 2005; Lawrence et al., 2006; Nakano et al., 2008). According to Lawrence et al. (2006), the ability to switch thought processes, known as set shifting, is a component of executive function and is also impaired in individuals with OCD. This deficit makes it extremely hard for the person to apply knowledge to a new situation as the individual will continue to rely on a previously used strategy to develop a solution, even if that strategy has proven to no longer be effective (Lawrence et al., 2006; Sanz, Molina, Calcedo, Martin-Loeches, & Rubia, 2001). Thus, they demonstrate a component of executive dysfunction often referred to as cognitive inflexibility.

Though a relation between executive dysfunction broadly—and cognitive inflexibility specifically—and OCD exists, a true causal relation is still quite unclear. A study done by M. Kim, Jang, and B. Kim (2009) suggests that executive dysfunction leads to the development of OCD. This particular study examined executive functioning in subclinical obsessive-compulsive individuals. It was found that the participants did in fact exhibit poor executive functioning when

asked to perform executive tasks (i.e., Wisconsin Card Sorting Test and Trails Making Test). The researchers inferred from the results that if executive function deficits are present in obsessive-compulsive individuals that are not diagnosed with the disorder, then perhaps it is executive dysfunction that leads to the disorder, OCD. In contrast, Aycicegi-Dinn, Dinn, & Caldwell-Harris (2009) suggest that the symptoms of executive dysfunction one experiences (obsessive or compulsive behaviors) develop to compensate for executive deficits, thus preceding the disorder. In respect to whether the symptoms or the disorder comes first, another question is what actually causes or has the potential to lead to executive dysfunction.

Some studies indicate that executive dysfunction, including cognitive inflexibility, results from neurological anomalies leading to functional impairments in the frontal lobe, affecting one's memory and reasoning (Fenger et al., 2005; Gu et al., 2008; Kuelz et al., 2006). In fact, these neurological differences may be heritable, with deficits in cognitive flexibility apparent among related individuals (Chamberlain et al., 2007). Others suggest that it may be due to traumatic brain injury, such as an instance in which an individual was not just hit in the head but was rendered unconscious, causing some sort of lesion in the frontal lobe (Coetzer, Stein, & Du Toit, 2001). There is also research that evaluates the role that age may play in the deterioration of executive function and cognitive flexibility. For example, Greenwood (2000) suggests that age affects the frontal lobes more quickly than it does any other region of the brain. Greenwood's study found that a significantly large decline in the blood flow of the frontal lobes occurs between the ages of 19 and 50. Based on these findings, the current study employed a sample of adults ages 18 to 45 to avoid any confounding by the extraneous variable of age. The projected sample age was also chosen to add to the scarcity of literature on young adults and executive dysfunction, as a large amount of literature on the elderly and executive dysfunction already

exists. Besides age, comorbidity with other disorders also plays a large role in the decreasing of one's executive functioning.

Comorbid disorders occur simultaneously in an individual and have the potential to multiply the severity of symptoms. Kloss et al. (2009) found that executive functioning in individuals presenting with symptoms of OCD and high levels of depression performed significantly worse on measures of executive function than those with low depression. The study by Kloss and colleagues suggests that OCD in comorbidity with another disorder leads to more severe executive dysfunction and that the severity is linearly related to symptom severity of the disorders. For example, Basso et al., (2007) found that individuals with major depressive disorder (MDD) comorbid with anxiety disorder performed significantly worse on executive tasks than individuals with only depression. It was also shown that the performance of those with only symptoms of depression was not significantly different from the control group, suggesting that having only mild symptoms of depression is not enough to impair cognitive performance. However, results of the study revealed that individuals with MDD suffer memory impairment, regardless of whether the disorder is comorbid with another disorder.

Among the literature, the question arose of whether individuals with OCD would regain cognitive deficits when their symptoms were under control. Bannon, Gonsalvez, Croft, and Boyce (2006) conducted a longitudinal study, a study done over time using the same subjects to monitor changes in the subjects, to determine whether an individual exhibits executive dysfunction only when he or she has symptoms of OCD or if executive dysfunction is always present). The second study was completed when the participants had reached a status of remittance for OCD. There were no changes on scores of executive functioning, suggesting that executive function is trait-related, despite symptom remittance. The findings of this study are

concurrent with those of Nakano et al. (2008). This particular study aimed at identifying executive dysfunction in medicated patients in remittance of major depressive disorder (MDD). Results show that executive dysfunction was still present after symptoms of MDD had subsided. The researchers believe this irreversibility may be due to deep, white matter vascular changes in the brains of the patients.

In particular, the cognitive inflexibility components appear to be theoretically linked to depression. That is, "cognitive inflexibility may play an important role in rumination, a risk factor for the onset and maintenance of depressive episodes" (Joormann, Levens, & Gotlib, 2011, p. 979). Anxiety has also been linked experimentally to problems with shifting sets, including shifting from a neutral to an emotional mental state (Johnson, 2009), underscoring the possibility that cognitive inflexibility is related to anxiety. However, little research has been done in this area. Thus, the focus of the current study was to examine cognitive inflexibility and its relation to OCPT, as well as how symptoms of depression and anxiety may mediate—and at least partially explain—that relation. In order to help us understand what cognitive inflexibility is, Martin and Rubin have provided a definition that was used as a construct in the current study.

According to Martin and Rubin (1995), cognitive flexibility is conceptualized as a person's "(a) awareness that in any given situation there are options and alternatives available, (b) willingness to be flexible to adapt to the situation, and (c) self-efficacy in being flexible" (p. 623). For the current study, the definition of cognitive flexibility was reversed to provide a definition for cognitive inflexibility. That is to say that those who are not aware of other options and alternatives available, unwilling to be flexible to adapt to the situation, and do not believe that he or she is actually able to adapt, are cognitively inflexible. This definition operationalizes cognitive inflexibility and its pertinence to OCPT is shown by the report that cognitive

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inflexibility tends to be positively correlated to rigidity, a large component in the symptoms of OCPT (Martin & Rubin, 1995).

Current Study

Among the OCD literature, most studies examining executive functioning or cognitive flexibility have been done with patients diagnosed with OCD. Not much research has been done to examine OCD and executive dysfunction/cognitive inflexibility among individuals who possess subclinical cognitive disabilities that do not require psychoactive medication but are serious enough to require the development of tactics to deal with the symptoms. To fill this gap in the literature, the current study examined OCD at subclinical levels in the form of obsessive-compulsive personality traits (OCPT). The current study also narrowed the executive function deficits to explicitly examine cognitive inflexibility. If an individual is afflicted with OCPT, he or she is characterized by engaging in perfectionistic behavior and strict adherence to rules. Aycicegi-Dinn, Dinn, and Caldwell-Harris (2009) suggested that this rigid rule-following may occur so that the individual is able to compensate for his or her cognitive deficits. As mentioned before, individuals with executive dysfunction have difficulties developing strategies and following rules to reach a goal. In contrast, individuals with OCPT are not cognitively impaired to the extent that they are unable to follow rules; in fact, they rely on them. Previous research has demonstrated that individuals with obsessive-compulsive disorder (OCD), as well as those with subclinical levels of OCPT, tend to demonstrate cognitive inflexibility on neuropsychological tasks (Aycicegi-Dinn, Dinn, & Caldwell-Harris, 2009). Other research has shown that this link may be partially explained by comorbidity with depression (Kloss et al., 2009). The researchers stated that evidence had been found in previous studies to justify their claim. There is also some belief that comorbid anxiety may also play a role in executive dysfunction; however, this aspect

requires further exploration. Thus, the current study used two mediation models as described by Baron and Kenny (1986) to determine if depression and/or anxiety would mediate the relation between cognitive inflexibility and OCPT.

Hypotheses

First, it was hypothesized that cognitive inflexibility would relate to OCPT. Second, it was hypothesized that anxiety would at least partially mediate the relation between cognitive inflexibility and OCPT. Third, it was hypothesized that depression would at least partially mediate the relation between cognitive inflexibility and OCPT.

Method

Participants

A total of 56 participants ranging from ages 18 to 40 years ($M = 20.46$, $SD = 3.51$) completed the survey in its entirety. Participants were recruited through word-of-mouth referrals as well as email announcements. Of the 56 participants, 44 (78.6%) were Caucasian, 9 (16.1%) were African American, 2 (3.6%) were Hispanic/Latino, and 1 was (1.8%) self-classified as "other." A total of 41 (73.2%) participants were female, and 15 (26.8%) were male. The sample was composed of 19 (33.9%) freshmen, 7 (12.5%) sophomores, 15 (26.8%) juniors, 13 (23.2%) seniors, and 9 (3.6%) graduate students and college graduates. The OCPD subscale from the Personality Diagnostic Questionnaire (PDQ-4) was used to determine which participants have a clinical level of symptoms of OCPD (score of 4 or higher). Participants had to be at least 18 years old to consent; otherwise, opportunity to participate in the study was open to all individuals, regardless of gender or race. Written consent was obtained from all participants.

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Participants were also required to complete a demographic form to record any history of psychological disorders, education, socioeconomic status, age, racial background, etc.

Measures

The Cognitive Flexibility Scale (CFS; Martin & Rubin, 1995) is a self-report questionnaire used to measure cognitive flexibility using the three components of communication, rigidity of attitudes, and personal habits. The scale is 12 items long and is on a 6-point Likert format. The original scoring format of the CFS was reversed so that results would yield a measure of cognitive inflexibility rather than cognitive flexibility. Internal consistency for the CFS was good to excellent, with $\alpha = .80$.

The Personality Diagnostic Questionnaire (PDQ-4; Hyler, 1994) is a self-administered measure of personality disorders. It was used to determine if a participant had OCPT as well as the severity of his or her symptoms. A score of 6 or higher qualified a participant for OCPT. Participants were asked to complete this brief questionnaire with complete honesty. An alpha coefficient is not a meaningful estimate of reliability in a scale of divergent criteria for personality disorder. Consistent with that, the OCPT scale of the PDQ-4 had a very low alpha coefficient (.05). However, item frequencies for the OCPT scale items indicated a range of responses and no significant skewness (skewness statistic = .33). Therefore, the scale was used as indicated.

The Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) is a self-report measure of anxiety, depression, and stress consisting of 42 items. This scale was developed for the purpose of measuring distinct symptoms of anxiety and depression, while keeping in mind the two constructs are dimensional, rather than categorical, clinical states. Items are presented in a scale format that ranges from (0) "did not apply to me at all" to (3) "applied to

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me very much, or most of the time." Excellent internal consistency for the DASS depression scale (Chronbach's α = .97), anxiety scale (Chronbach's α = .92), and stress scale (Chronbach's α = .95) has been reported (Antony, Bieling, Cox, Enns, & Swinson, 1998). The DASS has strong convergent and discriminant validity with both the Beck Depression Inventory and the Beck Anxiety Inventory (Antony et al., 1998; Lovibond & Lovibond, 1995). In the current sample, this measure showed strong internal consistency for depression, α = .85, and anxiety, α = .69.

Procedure

The measures were available in computer-survey form on a secure data collection website. Prior to the study, participants were informed about the experiment as well as its risks and informed of his or her right to discontinue at any time, and participants provided consent. Participants were administered the following measures in the order they were displayed: CFS, PDQ-4, and DASS. Participants were also asked to complete a demographic form. Data were analyzed using SPSS.

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Results

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Descriptive statistics for the variables of interest in the current study are presented in Table 1. An inspection of the skewness and kurtosis of anxiety and depression showed that the distribution of the scores for each was moderately skewed (2.05 for depression; 1.29 for anxiety) and kurtotic (5.35 for depression; 1.61 for anxiety). The skewness appeared to be due to a large number of participants reporting a very low level of these psychological symptoms. Because of the concern of these data violating the assumption of normality in linear regression, a linear transformation was performed on these two variables (taking a logarithmic function and adding a constant, given that the lowest score was 0; Howell, 2007; Tabachnick & Fidell, 2007). The two

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resulting variables were no longer skewed (-.19 for depression; -.28 for anxiety) or kurtotic (-.50 for depression; -.87 for anxiety). Therefore, these transformed variables were used in the correlation and regression analyses described below.

Correlation analyses among all variables of interest in the study were conducted to determine how they interrelated and to assist with the interpretation of further results (see Table 2). As indicated in the table, all variables were significantly positively intercorrelated with the exception of cognitive inflexibility and OCPT, which were not significantly related, $r = .21$, $p = .12$. Therefore, unfortunately, Hypothesis 1 was not supported.

Although a lack of relation between cognitive inflexibility and OCPT (i.e., no direct effect), there could be no mediation (i.e., the lack of a direct effect precluded an indirect effect). Nevertheless, the meditational models were examined, particularly to determine how depression and anxiety related to the predictor (cognitive inflexibility) and outcome (OCPT). Prior to these regression analyses, preliminary correlation analyses were conducted between key demographic variables and OCPT (outcome variable) as well as depression and anxiety (mediator variables, given that they are an outcome in part of the analyses to test for mediation) to determine if any demographics needed to be controlled in subsequent regression analyses. Age was significantly correlated with OCPT, $r = -.31$, $p = .02$, and depression, $r = -.36$, $p = .01$. Education was significantly correlated with depression, $r = -.39$, $p = .003$, and marginally correlated with OCPT, $r = -.24$, $p = .08$. Thus, as age and education increased, OCPT and depression symptoms decreased.

For the subsequent regression models testing the hypotheses, age and education were entered as control variables on Step 1 and the other variables were entered on Step 2. Two sets of a series of regression analyses were conducted to examine the relation between cognitive

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inflexibility and OCPT as well as to test for the potential mediational effect of anxiety (as well as depression in the second set of analyses), following the approach recommended by Baron and Kenny (1986). This approach involves conducting three regression analyses and examining four criteria for mediation. First, the criterion (OCPT) was regressed on to the predictor (cognitive inflexibility). Second, the mediator (anxiety) was regressed on to the predictor (cognitive inflexibility). Third, the criterion (OCPT) was regressed on to the predictor (cognitive inflexibility) and the mediator (anxiety) simultaneously. To test for the potential mediational effect of depression, the second and third regression analyses were conducted substituting depression as the mediator.

Mediation would be shown if: (1) cognitive inflexibility significantly predicted OCPT in the first regression analysis; (2) cognitive inflexibility significantly predicted anxiety in the second analysis; (3) anxiety significantly predicted OCPT, when controlling for cognitive inflexibility, in the third analysis; and (4) the relation between cognitive inflexibility and OCPT was significantly reduced in the third regression analysis (when controlling for anxiety) compared to the first regression analysis (when anxiety was not controlled).

First, when controlling for age and education, cognitive inflexibility did not significantly predict OCPT, $\beta = .19$, $p = .16$, as would be expected given their lack of relation at the zero-order correlation level. Thus, the first requirement for mediation was not met. Second, controlling for age and education, cognitive inflexibility significantly predicted anxiety, $\beta = .43$, $p = .001$, which did meet the second requirement for mediation. Third, anxiety did not significantly predict OCPT, when controlling for age, education, and cognitive inflexibility, $\beta = .23$, $p = .11$, which did not meet the third requirement for mediation. Finally, cognitive inflexibility and OCPT were not related after controlling for age, education, and anxiety, $\beta = .09$,

p = .55, but this was consistent with their lack of significant relation overall. Therefore, only one of the four requirements for mediation was met, and no post-hoc analyses were conducted.[1] See Figure 1 for the full model testing anxiety as a mediator.

For the second meditational model, there was no need to reexamine the relation between cognitive inflexibility and OCPT, which is known to be non-significant. However, as above, the remaining regression analyses were conducted to determine how depression related to cognitive inflexibility and OCPT. When controlling for age and education, cognitive inflexibility significantly predicted depression, β = .31, *p* = .01, which did meet the second requirement for mediation. Third, depression marginally predicted OCPT, when controlling for age, education, and cognitive inflexibility, β = .27, *p* = .08, which trended toward meeting the third requirement for mediation. Finally, cognitive inflexibility and OCPT were not related after controlling for age, education, and depression, β = .11, *p* = .45, but this was consistent with their lack of relation overall. Therefore, only one of the four requirements for mediation was met, whereas a second requirement was marginally met. Thus, no post-hoc analyses were conducted. See Figure 2 for the full model testing depression as a mediator.

Discussion

Findings of the current study support, though not substantially, only one of the three aforementioned hypotheses. Cognitive inflexibility did positively relate to OCPT. However, the magnitude of the relation found within the current study was not large enough to support a strong, direct relation between cognitive inflexibility and OCPT. Therefore, the hypotheses that anxiety and/or depression would mediate the relation between cognitive inflexibility and OCPT were not supported. In other words, neither anxiety nor depression was able to mediate the relation between cognitive inflexibility and OCPT because there was no significant direct

relation between cognitive inflexibility and OCPT, making the mediating variables (anxiety and/or depression) irrelevant in those models. Nevertheless, an examination of the complex interrelation of the variables of interest (i.e. cognitive inflexibility, anxiety, depression, and OCPT) within the models revealed significant findings.

Although cognitive inflexibility did not significantly relate to OCPT, it did significantly relate to both anxiety and depression, when controlling for age and education. Findings also revealed a marginal, but not significant, relation between depression and OCPT when controlling for cognitive inflexibility, age, and education. Anxiety, however, was not significantly related to OCPT when controlling for cognitive inflexibility, age, and education. At the zero-order correlation level (i.e., without other control variables), findings of the current study yielded evidence to suggest that cognitive inflexibility, anxiety, and depression were significantly inter-correlated.

Theoretical and Clinical Implications

It is thought that individuals who possess a strong tendency to cognitively simplify the world around them are often characterized as ''rigid'' or high in need for structure (Ciarrochi et al., 2005). The "rigidity" in thinking that accompanies OCPT is linked to an increase in emotional distress and poor problem solving (Neuberg & Newson, 1993). The intense relation found between cognitive inflexibility and anxiety could possibly be attributed to this rigidity. As changes in the environment, social life, occupation, etc. occur, one is required to adapt to them. Individuals who are cognitively inflexible tend to want things done a certain way. Therefore, change may make them increasingly anxious. Ciarrochi et al. (2005) found that intolerance of uncertainty about change, characterized as an increase in emotional distress when presented with unpreventable doubts of what will happen next, is also accompanied by symptoms of "rigidity"

and was moderately related to depression. The presence of rigidity in the intolerance of uncertainty may also give way to cognitive distortions, as it is impossible to avoid the uncertainties of an ever-changing society. These distortions may then lead to symptoms of depression.

OCPT and anxiety/depression are also related, although not uniquely related in the regression analyses. Anxiety and depression are located on Axis I in the DSM-IV (APA, 2000). OCPT relates to an Axis II disorder (i.e., OCPD). If an individual were to be afflicted with a disorder from both Axis I and II, as well as any other combination of problems from the remaining DSM-IV axes, he or she may experience a case of comorbidity. Comorbidity could yield a multiplicative rather than additive effect on symptoms of anxiety or depression if in conjunction with another personality disorder, health disorder, or significantly stressful event (Basso et al., 2007; Kloss et al., 2009).

Limitations and Directions for Future Research

Power proved to be an obstacle of great magnitude in the current study. The relatively small sample size when detecting small to moderate effect sizes probably precluded finding many significant results. The relation between cognitive inflexibility and OCPT may have been significant had the researcher had more participants and, therefore, more power to detect the moderate effect size. Future research should be conducted with larger samples.

The second largest limitation of the current study was that all data were obtained from self-report questionnaires rather than administered in a laboratory, allowing for rater bias. Participants may not have completed the surveys with honesty. They also may have become fatigued when completing the measures. Future researchers should employ the use of

neuropsychological testing in a laboratory for cognitive inflexibility to measure some of the constructs.

Another limitation of the study was a limited sampling method. The researcher was only able to recruit via email, word of mouth, and from the university's research participation website. Perhaps including a number of participants from the local community would have given way to an increase in sample diversity and a decrease in the skewness of depression reports. Participants were mostly students and were derived from a non-clinical sample. Although the researcher purposely examined students from a non-clinical sample, perhaps a more occupationally and racially diverse, clinical sample would yield greater findings.

Conclusions

The current study found significant relations between cognitive inflexibility and anxiety/depression and marginal significance between depression and OCPT. Surprisingly, the relation between anxiety and OCPT was not significant, even considering the assumption that an increase in anxiousness would result in an increase in rigidity of thinking, a characteristic of OCPT. It is imperative that future research employ a larger sample size; an increase in power may have the potential to yield a greater magnitude in the effect size between anxiety and OCPT, as well as depression and OCPT.

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Table 1

Descriptive Statistics of Predictor, Mediator, and Outcome Variables

Variable of Interest	Minimum	Maximum	Mean	Standard Deviation	Skewness	Kurtosis
Cognitive Inflexibility	12	43	26.04	6.84	-.02	-.36
Depression	0	38	6.82	7.41	2.05	5.35
Anxiety	0	28	6.29	6.25	1.29	1.61
OCPT	1	8	3.96	1.29	.33	.58

Note. N = 56. OCPT = Obsessive-Compulsive Personality Traits.

Table 2

Inter-correlations Among Predictor, Mediator, and Outcome Variables

Variable of Interest	Cognitive Inflexibility	Depression	Anxiety	OCPT
Cognitive Inflexibility	--			
Depression	.37**	--		
Anxiety	.43**	.54***	--	
OCPT	.21	.37**	.32*	--

Note. N = 56

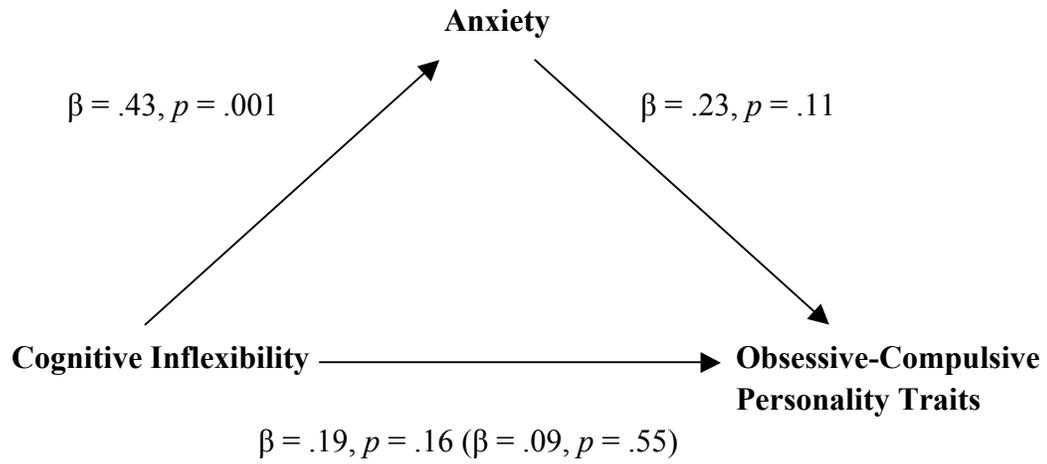* *p* < .05. ** *p* < .01. *** *p* < .001.

Figure 1. Model testing anxiety as a mediator in the relation between cognitive inflexibility and obsessive-compulsive personality traits (controlling for age and education).

Depression

β = .31, *p* = .01   β = .27, *p* = .08

Cognitive Inflexibility ──────────────▶ Obsessive-Compulsive
                                         Personality Traits

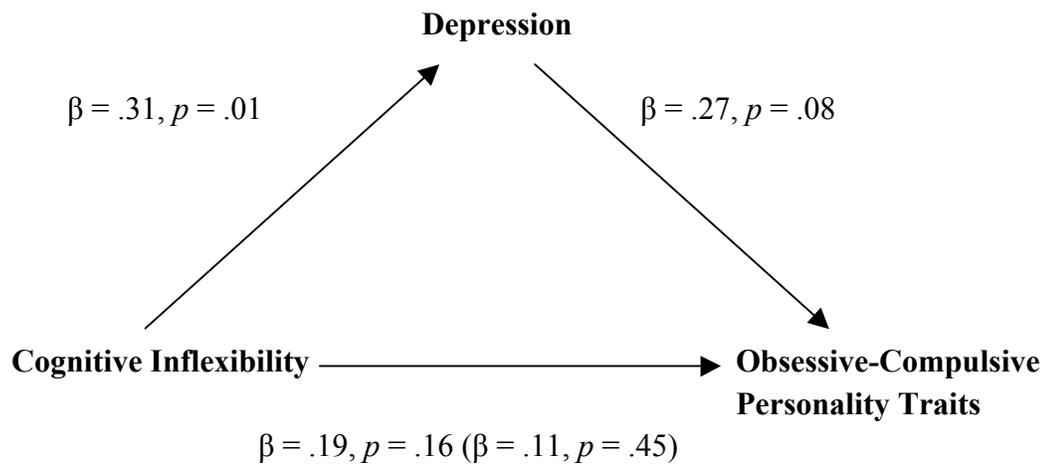β = .19, *p* = .16 (β = .11, *p* = .45)

Figure 2. Model testing depression as a mediator in the relation between cognitive inflexibility and obsessive-compulsive personality traits (controlling for age and education).

[1] If the models had supported a potential mediation (i.e., indirect effect between cognitive inflexibility and OCPT carried by anxiety or depression), post-hoc Sobel tests would have been conducted to compare the relation between cognitive inflexibility and OCPT in the first and third regression analyses (i.e., comparing the relation not controlling for anxiety/depression to the relation when controlling for anxiety/depression) to determine if the reduction in the relation (based on Beta weights) was significant. If so, such a finding would have supported at least partial mediation. Furthermore, if the Sobel test would have been significant and the relation between cognitive inflexibility and OCPT in the third regression analysis (controlling for anxiety/depression) was non-significant and reduced close to zero, it would have been evidence to support full mediation. Finally, a post-hoc analysis using nonparametric bootstrapping methods (Preacher & Hayes, 2004, 2008) to estimate a confidence interval of the indirect effect would have been evaluated to determine the significance of the mediation.