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Transdiagnostic Emotional Vulnerabilities Linking Obsessive-Compulsive and Depressive Symptoms in a Community-Based Sample of Adolescents

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Abstract

Background: Transdiagnostic emotional vulnerabilities are suspected to underlie psychopathologic comorbidity but have received little attention in adolescent emotional pathology literature. We examined distress tolerance, anxiety sensitivity, and anhedonia as concomitant transdiagnostic mechanisms that account for (i.e., statistically mediate) the covariance between adolescent obsessive-compulsive disorder (OCD) and major depressive disorder (MDD) symptoms. **Method**: Data on MDD, OCD, and the three aforementioned transdiagnostic vulnerabilities were collected from a community-based sample of 3,094 9th graders in a large metropolitan area and analyzed using mixed effects modeling to evaluate mediation effects. **Results**: Individually and when controlling for each other, all three transdiagnostic vulnerabilities mediated the relation between OCD and MDD symptoms both before and after adjusting for demographics. Conclusions: Distress tolerance, anxiety sensitivity, and anhedonia may be unique mechanisms accounting for comorbidity between OCD and MDD symptoms in youth. Longitudinal evaluation of these candidate transdiagnostic emotional vulnerabilities in adolescent OCD-MDD comorbidity is warranted.

Transdiagnostic Emotional Vulnerabilities Linking Obsessive-Compulsive and Depressive Symptoms in a Community-Based Sample of Adolescents

Recent perspectives in psychopathological science propose that cross-cutting dimensions and biological pathways underlie and account for the presentation and co-presentation of various mental disorders(Cuthbert & Insel, 2013; Dozois, Seeds, & Collins, 2009; Sauer-Zavala et al., 2012). In one such conceptualization, the underlying cause of various emotional symptoms and disorders, as well as their comorbidity, may be underpinned by a smaller set of *reactive* transdiagnostic vulnerabilities (i.e., characteristic patterns of maladaptive responses to emotional stimuli and states) (e.g., Leventhal & Zvolesnky, 2015). By enhancing or diminishing the response to emotion beyond normative functioning, reactive vulnerabilities can increase risk of emotional disturbances manifested in various ways (e.g., sadness, fear, withdrawal, disgust). By doing so, a small set of reactive vulnerabilities can precede and confer risk for various individual or combinations of clinical phenotypic expressions, including but not limited to MDD and OCD, and therefore be a root cause of psychopathological comorbidity.

A transdiagnostic emotional vulnerabilities framework may be useful for conceptualizing the high degree of overlap between obsessive-compulsive disorder (OCD) symptoms and major depressive disorder (MDD) symptoms (Watson, 2009). To this end, three putative vulnerabilities, highlighted as part of extant transdiagnostic emotion models (e.g., Leventhal & Zvolensky, 2015), are evaluated in the current study as mechanisms accounting for the overlap between OCD and MDD: distress tolerance (i.e., ability to withstand distressing states), anxiety sensitivity (i.e., fear of anxiety-related sensations), and anhedonia (i.e., inability to experience pleasure). Evidence from various clinical and non-clinical participant populations link poor

distress tolerance with both OCD (Cougle et al. 2011; Cougle et al. 2012; Cougle et al. 2013; Macatee et al. 2013) and depressive (Ellis et al. 2013; McHugh et al. 2014; Ali et al. 2015; Buckner et al. 2007; Gorka et al. 2012, Magidson et al. 2013, Tull & Gratz 2013; Brandt et al. 2013, O'Cleirigh & Ironson 2007) symptoms. Much like distress tolerance, anxiety sensitivity has also been implicated in clinical and non-clinical samples with OCD (Calamari et al. 2008; Olatunji & Wolitzky-Taylor 2009; Raines et al. 2014; Storch et al. 2013; Wheaton et al. 2012), as well as depression in clinical and non-clinical samples (Otto et al. 1995; Rector et al. 2007, Taylor et al. 1996; Tull & Gratz 2008; Zavos et al. 2012). Moreover, anhedonia demonstrates a similar relationship with depression and OCD, as it is a diagnostic criterion for the former (APA 2013), and it was recently shown to positively correlated with OCD severity after controlling for depression in an online sample of adults with OCD (Abramovitch et al. 2014). Although evidence of the link between anhedonia and OCD is limited, anhedonia may represent an important vulnerability for OCD given the emerging evidence implicating the reward pathway in the pathophysiology of OCD (Abramovich et al., 2014), such as abnormal activation patterns in the ventral striatum and insula (e.g., Figee et al., 2011).

Very little work has empirically tested whether reactive emotional vulnerabilities are transdiagnostic mechanisms of comorbidity (c.f., Wolitzky-Taylor et al., 2015; 2016; Zvolensky, Farris, Leventhal, & Schmidt, 2014), and no existing study has investigated anhedonia, distress tolerance, and anxiety sensitivity in tandem as mechanisms accounting for the relationship between OCD and MDD symptoms. To address this gap, the current cross-sectional correlational report in community-based sample of high school freshman, tested the hypothesis that all three transdiagnostic vulnerabilities (i.e., mediators) in isolation (i.e., each evaluated as a unique mediator in the absence of the other proposed mediators) and in combination (i.e., all mediators

tested simultaneously as predictors in a single mediation model) would account for a significant proportion of the relation between OCD symptoms and depressive symptoms. By testing these hypotheses, this study is positioned to provide evidence of possible mechanisms of co-occurrence of two debilitating psychiatric syndromes in a vulnerable developmental period (i.e., mid-adolescence), which could advance theoretical models of the etiology of this comorbidity and inform treatments for OCD and MDD phenomena.

Method

Participants and Procedures

For the purposes of this study, a community-based sample of 9th grade students was collected from ten public high schools across the greater Los Angeles metropolitan area as part of a larger longitudinal investigation of adolescent health and substance use (the current study uses responses from the first timepoint of data collection). These schools were chosen to sufficiently represent a diverse demographic: the percent of students who were eligible for free lunch within each school (i.e., students whose parental income $\leq$ 185% of the national poverty level) was 31.1% (*SD* = 19.7, range: 8.0% - 68.2%) among the ten schools. Students were ineligible if they were enrolled in special education (e.g., learning disabilities) or English as a Second Language Programs; a total of 4,100 students were deemed eligible to participate in the study. Assent to participate was collected from 3,874 (94.5%) students; of those, 3,383 (82.5%) provided written parental consent to participate in the study. After excluding students with substantial missing data on key variables from the analyses (*n* = 289), a total of 3,094 participants were used for the study at hand. For the sample, mean age was 14.57(*SD*=.40), mostly female (54.3%), and diverse (e.g., 45.3% Hispanic, 16.5% Asian, 16% non-Hispanic White) (see Table 1 for more demographic details).

Paper-and-pencil surveys were distributed in Fall 2013 during two separate one-hour in-class survey sessions less than two weeks apart. Each school that participated was compensated $2,500; students who completed the surveys were entered into a raffle for a gift card worth $50 as a means of compensation. The study was approved by the University of Southern California Institutional Review Board.

Measures

Covariates and Sample Descriptive Measures. To describe the sample population and include potential covariates, we assessed for demographic characteristics (e.g., age, gender, race/ethnicity [coded as nominal variable to reflect the 8 categories listed in Table 1]). Median neighborhood income level was also computed utilizing self-reported zip code information based on 2010 U.S. Census data (United States Census Bureau/American FactFinder, 2013).

Anhedonia. The Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995) is a 14-item self-report measure in which participants rate the degree of agreement with statements regarding hedonic experience based on how they had been feeling in the past few days. Items use a 4-point Likert scale (rated 1-4) that are scored based on the algorithm from Snaith et al. (1995), in which item responses were dichotomized so that strongly agree and agree equaled zero and strongly disagree and disagree equaled one. Higher scores reflecting more anhedonia. Prior research has found the SHAPS to exhibit sufficient internal consistency and construct validity in adolescent populations (Leventhal et al., 2015).

Anxiety Sensitivity. The Childhood Anxiety Sensitivity Index (CASI; Silverman et al., 1991) includes 18 items assessing fear or concern in response to anxiety-related physiological sensations, mental states, and social situations. Responses are based on a 3-point scale and are summed, with higher scores indicating higher anxiety sensitivity. The CASI has demonstrated

adequate two-week test-retest reliability and satisfactory criterion-related validity in non-clinical adolescent samples (Weems et al., 2001; Silverman, Ginsburg, & Goedhart, 1999).

Distress Tolerance. The 15-item Distress Tolerance Scale (DTS; Simons & Gaher, 2005) includes statements regarding typical responses indicative of intolerance of distressing emotional states. The measure requires averaging the responses to each item, which each use a 5-point Likert scale, with higher average scores reflecting lower levels of distress tolerance. The DTS has been shown to have excellent psychometric properties, including high test-retest reliability and appropriate convergence with other relevant measures assessing affective distress and regulation (Simons & Gaher, 2005).

Obsessive Compulsive Disorder Symptoms. The OCD symptoms subscale of the Revised Children's Anxiety and Depression Scale (RCADS; Chorpita et al., 2000) was used to measure OCD symptoms. This subscale consists of 6 items based on a 4-point Likert scale (scored 0-3) indicating relative frequency of occurrence. Item results are added together, yielding a sum score, with higher scores indicating higher OCD symptom frequency. Prior work has demonstrated the RCADS to display good reliability and sufficient convergent and discriminant validity in adolescent samples (Ferdinand et al., 2006; Chorpita et al., 2000). A sum on the OCD subscale of greater than 5 considered in the clinical range, yielding a specificity of .65 and sensitivity of .70 for a diagnosis of OCD among adolescents (Chorpita, Moffitt, & Gray. 2005).

Depressive Symptoms. The Center for Epidemiologic Studies Depression Scale (CESD; Radloff, 1977) is a 20-item measure of depressive symptoms during the preceding week. Responses are based on a 4-point Likert scale and are summed, with higher numbers indicating higher frequency. The CESD has sufficient psychometric characteristics, and has been validated in samples of high school students as well as ethnically-diverse adolescent populations (Edman

et al., 1999; Radloff, 1991). A score greater than 16 is considered in the clinical range (Radloff, 1977) with a score higher than 26 falling in the severe range.

Analytic Approach

Hypotheses were tested using mixed effects modeling (*df*'s based on Satterthwaite), with the first level of the model including all covariates (i.e., age, income, gender, and minority status) and mediator variables and the second level containing a school identity variable to account for participant nesting within schools.

We examined the direct effects (depressive symptoms regressed on OCD symptoms controlling for the mediators), indirect effects (association between OCD symptoms and depressive symptoms through the mediators), and total effects (depressive symptoms regressed on OCD symptoms not controlling for the mediators). We calculated the relative indirect effect (i.e., ratio of the indirect effect and total effect as an index of the proportion of the total effect accounted for by mediators; Preacher & Kelley, 2011) as an effect size. The mediational analyses were performed by first investigating each mediator in isolation from each other (Figure 1). We then calculated a multiple mediator model in which all three mediators were simultaneously included to control for overlapping variance in mediational paths and to determine which pathways may exhibit empirically unique relations relative to the others (Figure 2). All models were tested both unadjusted and after adjusting for demographic covariates. Results are reported as unstandardized path coefficients and relative indirect effects with bootstrapped 95% CIs in Tables 2-3, with standardized coefficients reported in Figures 1-2. Interpretation of the standardized path coefficients was based on Cohen's (1988) rule of thumb for small (.1), medium (.30), and large (.50).

Mediation analyses can be used for determining a sequential causal model, but the current study makes use of this approach as a tool for estimating the degree to which the transdiagnostic factors statistically account for the covariance between OCD and MDD symptoms (MacKinnon, Fairchild, & Fritz, 2007). There is precedent in the research literature for using mediation in this way to evaluate putative transdiagnostic factors (e.g. Wolitzky-Taylor et al. 2015; Wolitzky-Taylor et al., 2016). Missing data in the final sample were handled using multiple imputation. Inflated error rates were addressed by applying a False Discover Rate procedure— $\alpha_{critical} = .028$ (Benjamini & Hochberg, 1995).

Results

Preliminary Analysis and Total Effects

Descriptive statistics and Cronbach's alphas for the sample are provided in Table 1. Based on abovementioned cutoff scores, 33% of the sample scored in the clinical range for OCD symptom severity, and 33% and 16% in the clinical and severe ranges for MDD symptom severity, respectively. There were significant total effects for the positive relation of OCD and depressive symptoms in models unadjusted, *b*(95% bootstrapped CI) = 1.39(1.29, 1.49), $p < .0001$, and adjusted by covariates, *b*(95% CI) = 1.33(1.24, 1.43), $p < .0001$. Standardized path coefficients indicate a medium-to-large effect for OCD on depressive symptoms (see Figure note).

Emotional Vulnerabilities as Mediators

Greater OCD symptoms were associated with poorer distress tolerance, anxiety sensitivity, and anhedonic symptoms (Tables 2-3), with medium effect sizes for distress tolerance and anxiety sensitivity, and a small effect size for anhedonia (see Figure 1) . Individual analyses of each mediator separate from one another ($a \times b$ paths) indicated that the OCD-MDD association was significantly mediated by each transdiagnostic mediator in the expected direction

(see Tables 2-3). Distress tolerance and anxiety sensitivity demonstrated a similar magnitude of effect size as indicated by the standardized indirect effect (see Figure 1) and relative indirect effect (see Tables 2 and 3).

Similarly, to test whether the mediational paths were empirically unique or redundant, the three mediators were entered into the same model in tandem. Similar to the models with a single mediator, when the mediators controlled for each other in the analyses, all three were significant, and the effect sizes for anxiety sensitivity and distress tolerance were similar in magnitude (see Tables 2 and 3 for full results). The standardized total indirect effect was small-to-medium in size (see Figure 2).

Supplemental Mediation Analyses Reversing the Ordering of MDD and OCD Symptoms

The transdiagnostic vulnerabilities framework applied here proposes that emotional vulnerabilities precede the onset of both MDD and OCD symptoms and that the ordering of OCD and MDD in the statistical modeling may be arbitrary. To address the possible bi-directionality of OCD and MDD symptoms, we tested the *alternate* ordering in which the abovementioned adjusted models were re-tested with OCD symptoms as the dependent variable and depressive symptoms as the independent variable. The pattern of findings paralleled those reported in the primary analysis—all three factors were significant mediators in the multiple-mediator models and the magnitudes of relative indirect effects were stronger for distress tolerance and anxiety sensitivity compared to anhedonia (results available upon request to the first author).

Discussion

In a community-based sample of adolescents, this study provides new evidence indicating that anhedonia, anxiety sensitivity, and distress tolerance each accounted for a significant portion of the relation between OCD and depressive symptoms (i.e., indirect effect was 55% and 50% of the total effect for adjusted and unadjusted analyses, respectively). The results were similar when statistical models included adjustments for demographics. Altogether, the evidence provides support that all three vulnerabilities may reflect transdiagnostic mechanisms of OCD-MDD comorbidity in adolescents. Given that each of these mediators remained robust after controlling for each other in the multiple mediation model, each of these putative vulnerabilities may represent unique causal pathways linking OCD and MDD symptoms.

The results of the current investigation align with transdiagnostic vulnerability models of emotional comorbidity (e.g., Leventhal & Zvolesnky, 2015; Fairholme, Boisseau, Ellard, Ehrenreich, & Barlow, 2010), which purport that OCD and MDD symptoms are not associated *merely* because OCD causes MDD or vice versa. Transdiagnostic models propose that in a significant portion of comorbid cases these vulnerabilities precede OCD and MDD symptoms and increase the risk of both disorders. The order onset of OCD relative to MDD may be arbitrary in cases whereby the comorbidity originates from a common transdiagnostic mechanism. Consistent with this model, when OCD and MDD symptoms were swapped as independent and dependent variables in a supplemental analysis, result patterns were comparable. In addition, the *b* path models from the original and swapped analyses suggest that these three emotional vulnerabilities independently associate with depressive symptoms (while controlling for OCD symptoms), as well as OCD symptoms (while controlling for depressive symptoms). Given the cross-sectional and non-experimental nature of the study design, however,

such alternate directional results provide only indirect support for the ordering of the vulnerabilities relative to MDD and OCD.

A transdiagnostic vulnerabilities model is not the only explanation for the current findings. The relation between OCD symptoms and depression remained after controlling for the mediators in the models, suggesting partial mediation. In addition, according to standardized path coefficients, most effects were small (e.g., anhedonia) or small-to-medium (e.g., total indirect effect). Thus, other factors may also explain the relation between OCD and MDD symptoms, in addition to the three transdiagnostic processes studied herein. Future studies should examine other factors that may account for the link between OCD and MDD, including psychological constructs (e.g., intolerance of uncertainty), biological factors (e.g. genetic variants in the serotonergic system), and environmental risk factors (e.g., trauma).

In contrast to a transdiagnostic vulnerabilities model, the causal cascade model whereby one disorder causes another through a reactive vulnerability mediating mechanism is another plausible account of why reactive vulnerabilities may account for OCD-MDD covariance. For example, individuals with OCD symptoms may experience high anxiety sensitivity (e.g., Calamari et al. 2008), triggering safety behaviors or avoidance (i.e., Mowrer, 1947) due to low distress tolerance (e.g., Cougle et al. 2011). In turn, these safety behaviors may cause the individual to withdrawal and therefore reduce positive reinforcement, eliciting high anhedonia (e.g., Hatzigiakoumis et al., 2011), which is a risk factor for depression (e.g., Lewinsohn, 1974). This is one plausible causal account of the comorbidity between OCD and MDD symptoms in addition to the transdiagnostic vulnerability model.

In addition to providing novel evidence of transdiagnostic processes, this study also adds to emerging literature on the role of reactive emotional vulnerabilities psychopathology that is

primarily limited to adult samples. Results in this adolescent sample are consistent with and build on previous adult investigations linking OCD to distress tolerance (Cougle et al. 2011, Cougle et al. 2012, Cougle et al. 2013; Macatee et al. 2013; Robinson & Freeston 2014) and anxiety sensitivity (Calamari et al. 2008; Olatunji & Wolitzky-Taylor 2009; Raines et al. 2014; Storch et al. 2013; Wheaton et al. 2012), as well as studies associating depression with distress tolerance (e.g., Ellis et al. 2013; McHugh et al. 2014) and anxiety sensitivity (Otto et al. 1995; Rector et al. 2007, Taylor et al. 1996; Tull & Gratz 2008; Zavos et al. 2012). Our results advance the literature by documenting distress tolerance and anxiety sensitivity as possible explanation of the comorbidity between OCD and MDD symptoms in adolescents.

Only recently has evidence emerged for a link between anhedonia and OCD (Abramovitch et al., 2014). The current study extends this finding to a large, non-clinical community-based high school sample and suggests anhedonia may have a mediating role in the relation between OCD symptoms and depression that is empirically distinct from mediation by the other transdiagnostic factors. This finding is consistent with emerging research on the pathophysiology of OCD implicating brain reward pathways , such as the ventral striatum and nucleus accumbens (e.g., Figee et al., 2011), that may underpin anhedonia (e.g., Nestler & Carlezon, 2006).

From a clinical perspective, the current results suggest that practitioners providing evidence-based psychosocial treatments for OCD and depression could benefit from targeting these transdiagnostic vulnerabilities. Specifically targeting low distress tolerance, anxiety sensitivity, and anhedonia is not a defined feature of exposure and response prevention, the gold standard psychosocial treatment for OCD (Jenike, 2004). Comorbid depressive symptoms reduce the efficacy of behavior treatments for OCD (Abramowitz & Foa, 2000; Foa et al.; 1983), and

individuals with OCD and depressive symptoms tend to have higher levels of anxiety, more psychiatric comorbidities in general, increased rates of unemployment, and more functional disability (Abramowitz, Storch, Keeley, & Cordell , 2007; Hong et al. 2004; Ricciardi & McNally, 1995; Tukel, Meteris, Koyuncu, Tecer, & Yazici, 2006; Tukel, Polat, Ozdemir, Sksut, & Turksoy, 2002). Thus, incorporating such transdiagnostic vulnerabilities into a behavioral case conceptualization for OCD, particularly in the presence of comorbid depressive symptoms, may facilitate patient recovery.

One limitation of the study is the cross-sectional and correlational design, which limits causal inference about the mediating effects of the transdiagnostic vulnerability factors. This complicates drawing conclusions about temporal patterns, such as whether or not OCD or MDD are better modeled as predictors or outcomes. This cross-sectional limitation also precludes ruling out anhedonia as just a diagnostic feature of depression and not a vulnerability factor, although research and theory has supported the latter (Lewinsohn, 1974; Loas, 1996). As another limitation, the self-report nature of the data may suggest that a portion of the effects in the study stemmed from shared method variance. Similarly, there were no clinical diagnostic tools included in data collection to corroborate self-reports. The current study included a measure of anhedonia that measures feelings in a short and recent window of time (i.e., the last few days). Although the measure has demonstrated good temporal stability estimates (ICC r = .70; Franken et al., 2007), anhedonia as operationalized in this study may reflect more of a state construct that a trait construct. Future research would also benefit from enhanced generalizability by including a wider range of adolescent ages from other geographic regions, as well as a clinically diagnosed MDD and OCD sample. Studying this research question in a clinical sample, for example, could

help account for other possible mechanisms linking these two conditions, such as medication usage and past treatments.

Conclusion

This is the first study to demonstrate jointly and independently the mediating effect of distress tolerance, anxiety sensitivity, or anhedonia in the relation between OCD symptoms and depression in a large, community-based adolescent sample. This is a group that is rarely studied and an age range that may be particularly pertinent for examining risk factors for the development of MDD and OCD, given that puberty is a common age at onset for MDD and most cases of OCD onset in adolescence or early adulthood (APA, 2013). Thus, future research corroborating results from the current study may suggest avenues for preventing MDD and OCD comorbidity by targeting important transdiagnostic emotional vulnerabilities.

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Figure Legends

Figure 1. Standardized Path Coefficients for Individual Mediational Models.

Note. Separate mediational model sets were conducted for each transdiagnostic mediator. a = path from the independent variable (i.e., OCD symptoms) to the mediator. b = path from the mediator (i.e., distress tolerance, anxiety sensitivity, anhedonia) to the dependent variable (i.e., depression) after controlling for the effect of the independent variable. a × b = the indirect effect of the independent variable on the dependent variable that occurs through the mediator, which equals the product of the "a" path and "b" path. c′ = the direct effect of the independent variable on the dependent variable that is not carried through the mediator, which equals the effect of the independent variable on the dependent variable after controlling for the mediator. The total effect = the sum of the indirect effect (a × b) and direct effect (c′), which equals the effect of the independent variable on the dependent variable not adjusting for the mediators. Unadj = unadjusted *B*(95% CI); Adj = adjusted *B*(95% CI). Unadjusted C path β (95% *CI*) = .45 (.42, .48). Adjusted C path β (95% *CI*) = .43 (.40, .46).

Figure 2. Standardized Path Coefficients for Combined Mediational Models.

Note. A single mediational model set was conducted, which included all mediators simultaneously modeled. a = path from the independent variable (i.e., OCD symptoms) to the mediator. b = path from the mediators (i.e., distress tolerance, anxiety sensitivity, anhedonia entered as simultaneous predictors) to the dependent variable (i.e., depression) after controlling for the effect of the independent variable. $a \times b$ = the indirect effect of the independent variable on the dependent variable that occurs through a mediator controlling for the mediation occurring through the other two variables, which equals the product of the "a" path and "b" path for that specific mediator. c′ = the direct effect of the independent variable on the dependent variable that is not carried through the mediators, which equals the effect of the independent variable on the dependent variable after controlling for all three mediators. The total effect = the sum of the indirect effect ($a \times b$) of the three mediators and the direct effect (c′), which equals the effect of the independent variable on the dependent variable. For the "a" path estimates, please see Figure 1. Unadj = unadjusted *B*(95% CI); Adj = adjusted *B*(95% CI). Unadjusted C path β (95% *CI*) = .45 (.42, .48). Adjusted C path β (95% *CI*) = .43 (.40, .46).

Table 1. Sample Characteristics and Variable Descriptive Statistics

Variable: *M*(*SD*) or %	Overall Sample (*N* = 3,094)	α
Age	14.57 (.40), range 12.83-16.24]	
Gender		
Female	54.3	
Male	45.7	
Race/Ethnicity[a]		
American Indian or Alaskan Native	1.0	
Asian	16.5	
Black or African American	4.8	
Native Hawaiian or Pacific Islander	3.4	
Hispanic or Latino	45.3	
White	16.0	
Multiracial	5.6	
Other	5.7	
Median Income by Zipcode	$69,469 ($13,433)	
Symptoms		
RCADS OCD Subscale	4.48 (3.86) [range 0-18]	.82
CESD	14.66 (11.92) [range 0-60]	.81
DTS	2.58 (0.85), [range 1-5]	.91
CASI	30.48 (7.04), [range 18-54]	.88
SHAPS	1.71 (2.40), [range 0-14]	.89

Note. [a]Missing race/ethnicity data for 1.7% of participants. RCADS OCD Subscale = Revised Children's Anxiety Scale Obsessive-Compulsive Disorder Subscale . Center for Epidemiologic Studies Depression Scale . DTS = Distress Tolerance Scale . CASI = Childhood Anxiety Sensitivity Index . SHAPS = Snaith-Hamilton Pleasure Scale . Data from 9th grade students in Los Angeles, California, USA collected in Fall 2013. α = Cronbach's α internal consistent estimate.

Table 2. Unadjusted Effects of OCD Symptom Severity on Depressive Symptom Severity and Mediation by Distress Tolerance, Anxiety Sensitivity, and Anhedonia

	Component Paths		Mediation: OCD symptoms → Mediator(s) → MDD symptoms		
	OCD symptoms →Mediator(s)	Mediator(s) → MDD symptoms Controlling for OCD symptoms	Indirect effect	Direct effect	Relative Indirect Effect
	A Path (95% CI)	*B Path (95% CI)*	*AB Path (95% CI)*	*C' Path (95% CI)*	*(95% CI)*
Individual Analyses					
Distress Tolerance	.10 (.10, .11)†	3.95 (3.46, 4.44)†	.40 (.36, .43)†	.98 (.87, 1.09)†	.287 (.25, .34)
Anxiety Sensitivity	.87 (.81, .93)†	.47 (.41, .53)†	.41 (.35, .47)†	.98 (.87, 1.09)†	.294 (.25, .35)
Anhedonia	.06 (.04, .08)†	1.54 (1.40, 1.69)†	.11 (.06, .13)†	1.30 (1.20, 1.39)†	.066 (.04, .10)
Combined Analyses				.69 (.58, .79)†	
Distress Tolerance	-	2.66(2.16, 3.15)†	.27(.21, .32)†	-	.194 (.16, .24)
Anxiety Sensitivity	-	.38 (.32, .44)†	.33 (.28, .39)†	-	.237 (.20, .29)
Anhedonia	-	1.60(1.46, 1.74)†	.10 (.06, .13)†	-	.072 (.04, .10)

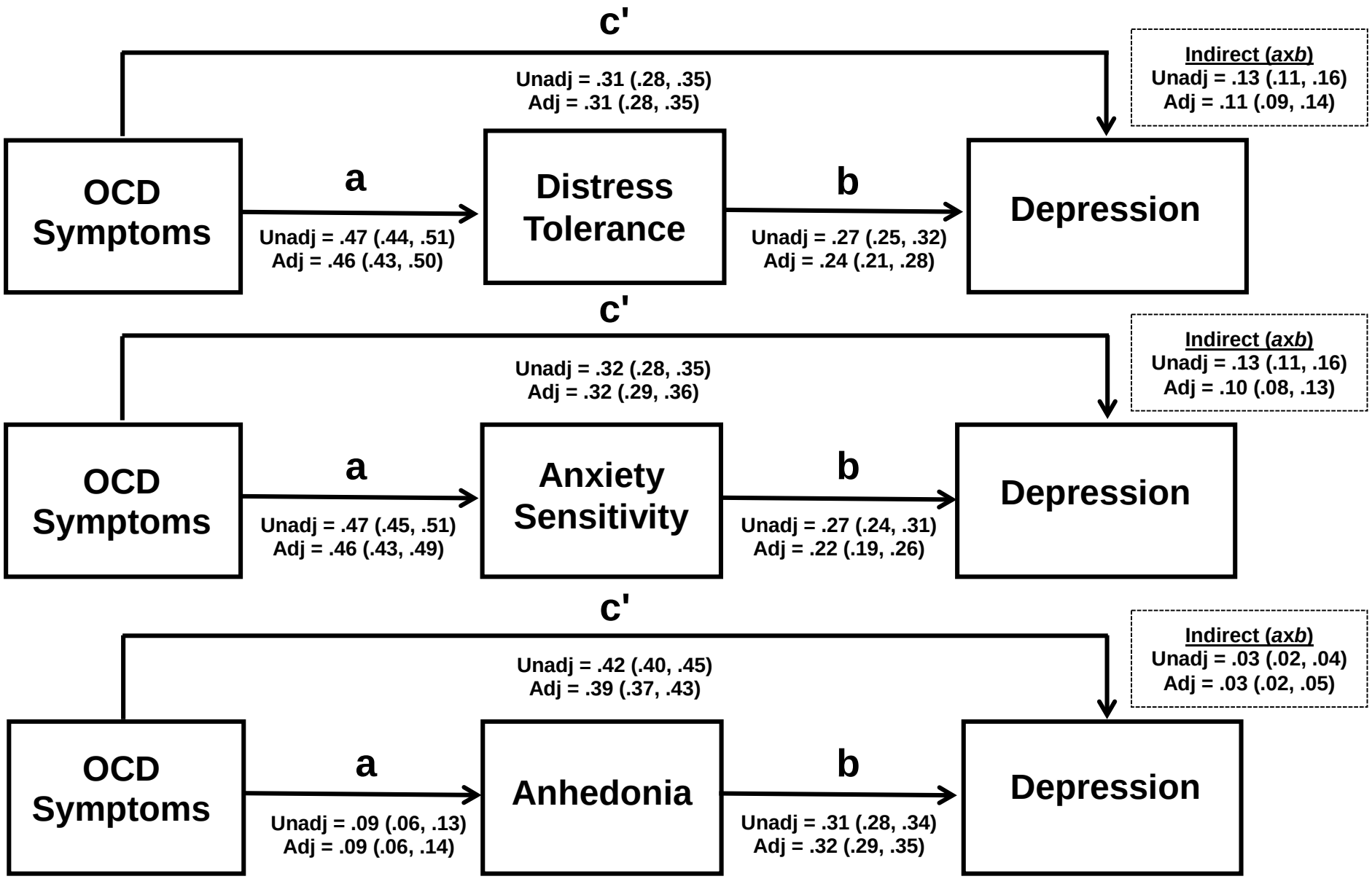
Note. N = 3,094. Data from 9th grade students in Los Angeles, California, USA collected in fall 2013. OCD = Obsessive Compulsive Disorder symptom severity. MDD = depressive symptom severity. A path = unstandardized path coefficient from the respective mediator regressed on OCD symptoms. B Path = unstandardized path coefficient from depression severity regressed on the mediator(s). C' Path = unstandardized path coefficient for OCD symptoms for the model in which depression severity was simultaneously regressed on the mediator(s) and OCD symptoms (i.e., relation of OC to depression after controlling for mediator[s]). C Path = unstandardized path coefficient from depression severity regressed on OCD symptoms—total effect; C-path (95% CI) = 1.39 (1.29, 1.49)†. Relative indirect effect is an effect size commonly interpreted as the proportion of the total effect accounted for by mediation (Preacher & Kelley, 2011). †$p < .0001$.

Table 3. Adjusted Effects of OCD Symptom Severity on Depressive Symptom Severity and Mediation by Distress Tolerance, Anxiety Sensitivity, and Anhedonia

	Component Paths		Mediation: OCD symptoms → Mediator(s) → MDD symptoms		
	OCD symptoms →Mediator(s)	Mediator(s) → MDD symptoms Controlling for OCD symptoms	Indirect effect	Direct effect	Relative Indirect Effect
	A Path (95% CI)	*B Path (95% CI)*	*AB Path (95% CI)*	*C' Path (95% CI)*	*(95% CI)*
Individual Analyses					
Distress Tolerance	.10 (.09, .11)†	3.49 (3.01, 3.98)†	.35 (.30, .41)†	.98 (.88, 1.09)†	.262 (.23, .32)
Anxiety Sensitivity	.84 (.78, .89)†	.38 (.32, .44)†	.32 (.26, .37)†	1.02 (.91, 1.12)†	.240 (.20, .30)
Anhedonia	.06 (.04, .09)†	1.60 (1.46, 1.74)†	.10 (.06, .13)†	1.24 (1.14, 1.33)†	.072 (.04, .11)
Combined Analyses				.73 (.63, .84)†	
Distress Tolerance	-	2.54(2.06, 3.02)†	.25 (.20, .31)†	-	.191 (.16, .24)
Anxiety Sensitivity	-	.29(.23, .35)†	.24 (.19, .30)†	-	.183(.14, .24)
Anhedonia	-	1.62(1.49, 1.76)†	.10 (.06, .13)†	-	.073 (.05, .11)

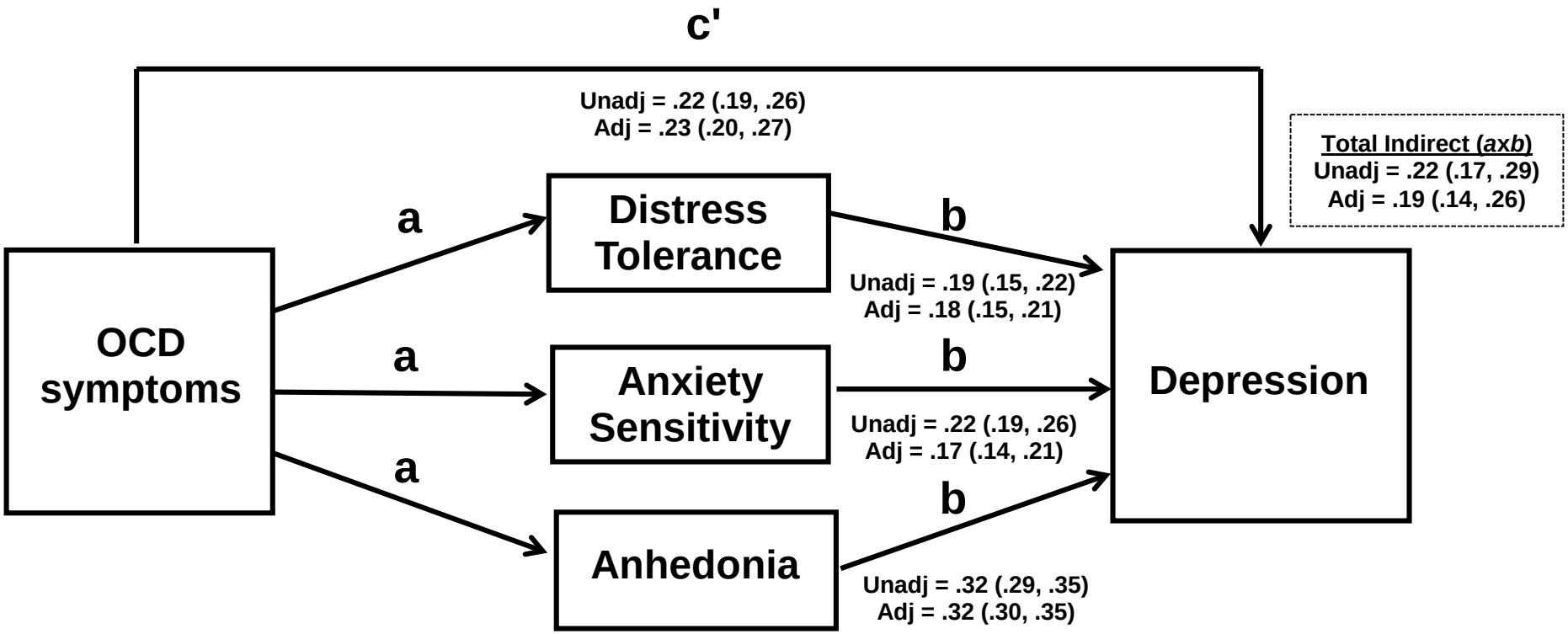
Note. *N* = 3,094. Data from 9th grade students in Los Angeles, California, USA collected in fall 2013. OCD = Obsessive Compulsive Disorder symptom severity. MDD = depressive symptom severity.. A path = unstandardized path coefficient from the respective mediator regressed on OCD symptoms plus covariates. B Path = unstandardized path coefficient from depression severity regressed on the mediator(s) plus covariates. C' Path = unstandardized path coefficient for OCD symptoms for the model in which depression severity was simultaneously regressed on the mediator(s), OCD symptoms (i.e., relation of OC to depression after controlling for mediator[s]) plus covariates. C Path = unstandardized path coefficient from depression severity regressed on OCD symptoms plus covariates —total effect; C-path (95% CI) = 1.33 (1.24, 1.43)†. Covariates for the models include gender, minority status (non-Hispanic White vs. not non-Hispanic White), age, and income. Relative indirect effect is an effect size commonly interpreted as the proportion of the total effect accounted for by mediation (Preacher & Kelley, 2011). †$p <$ .0001.

Figure 1. Standardized Path Coefficients for Individual Mediational Models



Note. Separate mediational model sets were conducted for each transdiagnostic mediator. a = path from the independent variable (i.e., OCD symptoms) to the mediator. b = path from the mediator (i.e., distress tolerance, anxiety sensitivity, anhedonia) to the dependent variable (i.e., depression) after controlling for the effect of the independent variable. a × b = the indirect effect of the independent variable on the dependent variable that occurs through the mediator, which equals the product of the "a" path and "b" path. c′ = the direct effect of the independent variable on the dependent variable that is not carried through the mediator, which equals the effect of the independent variable on the dependent variable after controlling for the mediator. The total effect = the sum of the indirect effect (a × b) and direct effect (c′), which equals the effect of the independent variable on the dependent variable not adjusting for the mediators. Unadj = unadjusted *B*(95% CI); Adj = adjusted *B*(95% CI). Unadjusted C path β (95% *CI*) = .45 (.42, .48). Adjusted C path β (95% *CI*) = .43 (.40, .46).

Figure 2. Standardized Path Coefficients for Combined Mediational Models



Note. A single mediational model set was conducted, which included all mediators simultaneously modeled. a = path from the independent variable (i.e., OCD symptoms) to the mediator. b = path from the mediators (i.e., distress tolerance, anxiety sensitivity, anhedonia entered as simultaneous predictors) to the dependent variable (i.e., depression) after controlling for the effect of the independent variable. $a \times b$ = the indirect effect of the independent variable on the dependent variable that occurs through a mediator controlling for the mediation occurring through the other two variables, which equals the product of the "a" path and "b" path for that specific mediator. c′ = the direct effect of the independent variable on the dependent variable that is not carried through the mediators, which equals the effect of the independent variable on the dependent variable after controlling for all three mediators. The total effect = the sum of the indirect effect ($a \times b$) of the three mediators and the direct effect (c′), which equals the effect of the independent variable on the dependent variable. For the "a" path estimates, please see Figure 1. Unadj = unadjusted *B*(95% CI); Adj = adjusted *B*(95% CI). Unadjusted C path β (95% *CI*) = .45 (.42, .48). Adjusted C path β (95% *CI*) = .43 (.40, .46).