



## Research paper

# Disgust proneness predicts obsessive-compulsive disorder symptom severity in a clinical sample of youth: Distinctions from negative affect



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## ABSTRACT

**Background:** Although studies have linked disgust proneness to the etiology and maintenance of obsessive-compulsive disorder (OCD) in adults, there remains a paucity of research examining the specificity of this association among youth.

**Method:** The present study employed structural equation modeling to examine the association between disgust proneness, negative affect, and OCD symptom severity in a clinical sample of youth admitted to a residential treatment facility ( $N = 471$ ).

**Results:** Results indicate that disgust proneness and negative affect latent factors independently predicted an OCD symptom severity latent factor. However, when both variables were modeled as predictors simultaneously, latent disgust proneness remained significantly associated with OCD symptom severity, whereas the association between latent negative affect and OCD symptom severity became nonsignificant. Tests of mediation converged in support of disgust proneness as a significant intervening variable between negative affect and OCD symptom severity. Subsequent analysis showed that the path from disgust proneness to OCD symptom severity in the structural model was significantly stronger among those without a primary diagnosis of OCD compared to those with a primary diagnosis of OCD.

**Limitations:** Given the cross-sectional design, the causal inferences that can be made are limited. The present study is also limited by the exclusive reliance on self-report measures.

**Conclusions:** Disgust proneness may play a uniquely important role in OCD among youth.

## 1. Introduction

Obsessive-compulsive disorder (OCD) consists of recurrent obsessions and/or compulsions that interfere with daily functioning (DSM-5; American Psychiatric Association [APA], 2013). Obsessions are characterized by intrusive, repetitive thoughts, images, or impulses. Compulsions, however, are purposeful, repetitive behaviors or rituals performed in an effort to relieve distress associated with obsessions. It is widely accepted that the development of OCD may be attributed to a complex interaction of genetic, environmental, and psychological processes (Abramowitz et al., 2009). Although the origin of OCD is likely to be multifactorial, a growing body of research suggests that obsessions/compulsions observed among those with OCD may be partially the result of excessive disgust proneness (Olatunji et al., 2010a). Disgust proneness is defined as a personality trait that reflects the tendency to experience disgust frequently and intensely (van

Overveld et al., 2006). Available research suggests that disgust proneness is present to a greater or lesser extent in all individuals (Olatunji et al., 2007) and is relatively stable over time (de Jong et al., 1997). The origins of disgust proneness have historically implicated environmental factors (Rozin and Millman, 1987). Indeed, research has shown that disgust propensity may be transmitted inter-generationally beginning as early as infancy with a mother's verbal and nonverbal display of disgust in the presence of her child (Muris et al., 2013). However, recent work suggests the origins of disgust proneness likely reflects the interaction of genetics (Sherlock et al., 2016) and childhood socializing experiences where disgust responses are modeled excessively (Stevenson et al., 2010).

The link between disgust proneness and OCD has been consistently observed in adult samples (Cisler et al., 2009; Olatunji et al., 2010a, 2010b). Although this link appears to be most robust for the contamination variant of OCD (Deacon and Olatunji, 2007; Olatunji et al.,

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2004; Tolin et al., 2006), self-report measures of disgust proneness have been found to correlate moderately with hoarding, neutralizing, and ordering symptoms of OCD (Olatunji et al., 2010a, 2010b). Disgust proneness was also found to demonstrate small but significantly correlations with religious obsessions even after controlling for general fearfulness and cleanliness fears (Olatunji et al., 2005). Similarly, Olatunji et al. (2011a, 2011b) found that disgust proneness predicted an OCD symptom latent factor that consisted of washing concerns, checking and doubting, obsessing, neutralizing, ordering, and hoarding even when controlling for negative affect. Furthermore, there is now strong evidence that the association between negative affect and OCD symptoms is mediated by disgust proneness (Olatunji et al., 2007; Olatunji et al., 2010a, 2010b). These findings are consistent with the view that “OCD may represent a dysfunction in the appraisal and processing of disgust” (Husted et al., 2006, p. 390).

Disgust proneness may contribute to the development and maintenance of anxiety and related disorders early in development (Muris, 2006). Theoretical models posit that disgust proneness may contribute to the development of OCD may reinforcing disease-avoidance motives (Olatunji et al., 2011a, 2011b). Consistent with this view, research has shown that the experience of disgust contributes to the development of fear-related beliefs and subsequent avoidance behavior among youth (Muris et al., 2009). Research with children has also shown that the experience of disgust results in an increased inclination to interpret ambiguous situations in a more negative way (Muris et al., 2012). Despite basic findings that highlight potential mechanisms by which disgust proneness may confer risk for OCD in children, very few studies have actually examined the link between disgust proneness and OCD among youth. In an initial study, Muris et al. (1999) found a moderate association ( $r = .30$ ) between disgust proneness and OCD symptoms in a nonclinical sample of children. However, this relationship became nonsignificant when controlling for trait anxiety. A subsequent study also found small, but significant, correlations ( $r$ 's = .20 for boys and .23 for girls) between a behavioral measure of disgust proneness and OCD symptoms in a nonclinical sample of children when controlling for neuroticism (Muris et al., 2008). Although controlling for more broad indicators of negative affect like neuroticism is likely to yield more robust effects between disgust proneness and OCD than when controlling for more specific indicators like trait anxiety, the modest associations observed in the limited research with youth suggests that it may be premature to conclude that disgust proneness in children is related to the development of OCD.

The modest association between disgust proneness and symptoms of OCD among youth may be the product of measurement limitations. Indeed, the available research linking disgust proneness and symptoms of OCD among youth has failed to employ an age-appropriate measure of disgust proneness that is designed specifically for children. Unfortunately, such studies have used simplified or age-downward extensions of adult measures, which may fail to capture important developmental nuances in disgust proneness. The Child Disgust Scale (CDS; Viar-Paxton et al., 2015) was recently developed to fill this important gap in the literature, and a comprehensive examination of psychometric properties suggests that the scale is a developmentally appropriate measure of disgust proneness that yields reliable and valid scores with children. A more recent study found that the CDS displays strong psychometric properties and is developmentally appropriate for use in pediatric clinical populations with OCD and anxiety disorders (Nadeau et al., 2017). Using the CDS, Viar-Paxton and colleagues (2015) found a strong association ( $r = .40$ ) between disgust proneness and symptoms of OCD in a nonclinical sample. Nadeau and colleagues also found that scores on the CDS were significantly associated with OCD symptom severity ( $r = .30$ ) in a clinical sample of youth.

Another important question is the extent to which disgust proneness is uniquely related to symptoms of OCD above and beyond the effects of negative affect. Negative affect may be defined

as the proneness to experience an array of negative emotional states as well as the proneness to activate defensive motivational systems (Craske, 2003). Importantly, various indicators of negative affect have been implicated in the development of OCD. For example, symptoms of depression have been found to robustly predict symptoms of OCD (Kim et al., 2012). In fact, previous research suggests that OCD and depressive symptoms co-occur primarily due to shared genetic factors (Bolhuis et al., 2014). Perhaps, as suggested by Rachman (1997), there is a bi-directional relationship between OCD and depression. To the extent that indicators of negative affect leads to the development of OCD, it remains unclear if disgust proneness confers risk for OCD above and beyond negative affect. Given that negative affect may represent a higher order generalized vulnerability factor for psychopathology more broadly whereas disgust proneness is viewed as proximal lower-order vulnerability for OCD specifically (Olatunji et al., 2011a, 2011b), unique effects for disgust proneness in predicting OCD symptoms may be expected. Examination of the extent to which disgust proneness does confer risk for the development of OCD above and beyond negative affect may have important implications for conceptualizing disgust proneness as a unique mechanism that should be the focus of treatment and prevention efforts.

The limited literature is inconsistent concerning the association between disgust proneness and OCD symptoms among youth. Given the recent availability of a developmentally appropriate measure of disgust proneness, the present study employs structural equation modeling (SEM) to examine the relation between disgust proneness and negative affect in the prediction of OCD symptom severity in a clinical sample of youth. Importantly, no study to date has examined the unique associations between disgust proneness and OCD symptom severity among youth using a clinical sample. It was predicted in the present study that disgust proneness would remain significantly associated with OCD symptom severity when both disgust proneness and negative affect are simultaneously modeled as predictors. It was also predicted that disgust proneness (proximal lower-order vulnerability) would mediate the association between negative affect (distal higher order generalized vulnerability) and OCD symptom severity. Lastly, it was predicted that the path from disgust proneness to OCD symptom severity when controlling for negative affect would be stronger among those with a primary diagnosis of OCD compared to those without a primary diagnosis of OCD.

## 2. Methods

### 2.1. Participants

The sample consisted of 471 participants (51.6% female) who presented for admission for treatment at the same residential facility. Participants that provided informant consent for their data to be used for research purposes were included. No participants were excluded. The mean age of the participants was 15.58 years ( $SD = 1.18$ ), ranging from 12 to 18 years. The ethnicity composition was as follows: Caucasians ( $n = 426$ ; 90.4%), Multiethnic ( $n = 1$ ; .2%), Hispanic ( $n = 13$ ; 2.8%), Black ( $n = 3$ ; .6%), Asian ( $n = 24$ ; 5.1%), Indian ( $n = 1$ ; .2%), and bi-racial, including Caucasian Chinese ( $n = 3$ ; .6%). The majority of participants' parents were married ( $n = 351$ ; 74.5%), followed by divorced ( $n = 88$ ; 18.7%) and single ( $n = 15$ ; 3.2%). Seventeen participants (3.6%) did not report parental marital status. Fifty-one percent of the sample had a primary diagnosis of OCD, while 24% had a primary mood disorder and 18% had a primary anxiety disorder. Seven percent of the sample had another primary diagnosis, such as an eating disorder or attention deficit hyperactivity disorder (ADHD). Table 1 displays the rates of primary diagnoses in the sample. Diagnoses were derived via unstructured interviews conducted by child and adolescent psychiatrists specializing in OCD. This diagnostic approach has been shown to be reliable in previous research (Leonard et al., 2015).

**Table 1**  
Frequency of primary diagnoses.

Group	Primary diagnosis	n	% of Sample	
OCD	OCD	243	51.5	
Anxiety	Panic disorder	4	.8	
	PTSD	9	1.9	
	Social anxiety	24	5.1	
	Generalized anxiety disorder	29	6.1	
	Anxiety disorder not otherwise Specified	18	3.8	
Mood	Major depressive disorder	76	16.1	
	Dysthymia	1	.2	
	Bipolar disorder	5	1.1	
	Mood disorder not otherwise Specified	29	6.1	
Other	Autism	1	.2	
	ADHD	9	1.9	
	Tic disorder/Tourette's	9	1.9	
	Trichotillomania	5	1.1	
	Body dysmorphic disorder	1	.2	
	Substance use disorder	1	.2	
	Anorexia nervosa restricting Subtype	1	.2	
	Eating disorder not otherwise Specified	1	.2	
	Other	6	1.3	
	Total		472	100.0

## 2.2. Procedure

Participants completed all measures (outlined below) during their admission to the residential treatment facility as part of larger test battery. Use of these data for research purposes and the informed consent process was approved by the institutional review board.

## 2.3. Measures

*Children's Yale-Brown Obsessive Compulsive Scale* (Y-BOCS; Scahill et al., 1997). The Y-BOCS was used to assess the severity of OCD symptoms. This 10-item questionnaire measures the following five parameters of obsessions (items 1–5) and compulsive rituals (items 6–10): (a) time occupied/frequency, (b) interference, (c) distress, (d) resistance, and (e) perceived control. Each item/symptom is rated on a five-point Likert-type scale from 0 (no symptoms) to 4 (severe symptoms) and severity of symptoms are rated using a 0- to 4-point rating scale, with 0= none, 1= mild, 2= moderate, 3= severe, and 4=extreme. The reliability and validity of this measure are strong (Storch et al., 2006). The Y-BOCS had an alpha of .96 in the present study.

*Child Disgust Scale* (CDS; Viar-Paxton et al., 2015). The CDS measures disgust propensity in children. Items are rated on a 3-point responses scale (i.e., 0= Always, 1= Sometimes, 2= Never). The CDS includes a Disgust Avoidance subscale and Disgust Affect subscale. The reliability, validity and factor structure of the CDS were found to be adequate based on a school-based sample and clinical sample (Viar-Paxton et al., 2015). The CDS had an alpha of .81 in the present study.

*Anxiety Sensitivity Index* (ASI; Reiss et al., 1986). The ASI is a 16-item questionnaire that measures fear of anxiety sensations and beliefs that the sensations are potentially harmful across the dimensions of Physical Concerns, Mental Incapacitation, and Social Concerns. Items are rated on a 5-point responses scale (i.e., 0=very little, 1= little, 2= average, 3=much, 4=very much). Reliability of this measure has been found to be good (Peterson and Reiss, 1992). The ASI had an alpha of .91 in the present study.

*Moods and Feelings Questionnaire* (MFQ; Costello et al., 1991). The MFQ is a 34- item questionnaire designed for children and adolescents. It includes questions about neuro-vegetative symptoms and feelings of loneliness, and feeling unloved and ugly. Severity of symptoms are rated on a three-point scale (i.e., 0=not true, 1=sometimes true and 2=true). The psychometric properties of this measure

were found to be strong, including acceptable reliability and significant correspondence with clinical diagnoses of depression (Wood et al., 1995). The MFQ had an alpha of .94 in the present study.

*The Screen for Child Anxiety Related Emotional Disorders—Children* (SCARED—Birmaher et al., 1999). The SCARED—C is a 66-item measure of seven domains of anxiety disorder symptoms. Severity of symptoms are rated using a 0- to 2-point rating scale, with 0=not true or hardly ever true, 1=sometimes true, and 2=true or often true. The reliability, validity and factor structure of this measure are well-supported. The SCARED—C had an alpha of .95 in the present study.

## 2.4. Data analytic approach

### 2.4.1. Missing data

The multiple-missing-data test (also known as Little's MCAR test; Little and Rubin, 1987) was used to examine missing data patterns for each set of indicators used in our factor models. With Little's MCAR test, the null hypothesis for this test is that data are 'missing completely at random' (MCAR).

For the *Obsession Compulsive Disorder* (OCD) symptom severity factor, the Y-BOCS Obsession subscale and the Compulsion subscale were used as the two observed indicators. All missing items were missing completely at random (MCAR),  $\chi^2(62) = 76.50, ns$ .

For the *Disgust Proneness* (DP) factor, the Avoid and Affect subscales of the CDS were used as the two observed indicators. All missing items were again missing completely at random (MCAR),  $\chi^2(8) = 8.64, ns$ .

For the *Negative Affect* (NA) factor, the ASI total score, MFQ total score, and the SCARED—C total score were used as the three observed indicators. Previous research has shown that there is an overarching negative affectivity factor that is largely comprised of fear and distress components (Ebesutani et al., 2011). More direct assessments of negative affect, like the MFQ, tend to assess the distress component. Given the nonspecificity of the distress component, including measures of the fear component like the ASI and SCARED—C may allow for a more rigorous test of the association between disgust proneness and OCD symptoms. Based on Little's MCAR test, all missing items were missing completely at random (MCAR),  $\chi^2(8) = 7.15, ns$ .

The MCAR nature of the missing data patterns noted above allowed us to employ the recommended full information maximum likelihood (FIML) data imputation method. The FIML imputation procedures is among the best and most recommended approach to handling missing data due to (a) utilizing all available data in the covariance matrix to estimate model parameters and (b) produced less biased scores and efficiently estimates standard errors compared to other procedures such as the pairwise or listwise approach (Arbuckle, 1996; Wothke, 2000).

### 2.4.2. Measurement model

Prior to conducting SEM, the fit of the measurement model we examined using Mplus version 7.11 (Muthén and Muthén, 2012) and the MLR estimator. There were three factors in this measurement model: (1) OCD symptom severity (identified by the Obsession and Compulsion subscales of the Y-BOCS), (2) DP (identified by the Avoidance and Affect subscales of the CDS), and (3) Negative Affect (identified by the ASI, MFQ and SCARED—C total scores).

The fit of the measurement model was examined via the Comparative Fit Index (CFI), the Root Mean Square Error of Approximation (RMSEA), and the Standardized Root Mean Square Residual (SRMR). Cut-offs for good fit included the following: CFI > .90 (Bentler, 1990), and RMSEA and SRMR < .08 (Browne and Cudeck, 1993).

## 2.5. Structural equation modeling

Using SEM, the extent to which the DP factor significantly predicted OCD symptom severity independent of the NA factor was examined. To

examine whether DP significantly mediates the relationship between NA and OCD symptom severity, a combination of tests as recommended by MacKinnon et al. (2002) was employed. First, Baron and Kenny's (1986) causal link test which requires four conditions to be met to support the presence of significant mediation was examined: (1) the independent variable (i.e., NA) significantly predicts the outcome variable (i.e., OCD symptom severity), (2) the independent variable (i.e., NA) significantly predicts the proposed mediator (i.e., DP), (3) the proposed mediator significantly predicts the dependent variable (i.e., OCD symptom severity) while controlling for the independent variable (i.e., NA), and (4) the significant relationship between the independent variable and the dependent variable becomes non-significant once the proposed mediator is included in the model.

Second, the significance of the indirect (intervening) pathway through the mediator was examined. We also compared the following models (using the Satorra-Bentler Scaled Chi-Square Difference Test; Satorra and Bentler, 1994): (a) the model whereby the direct effect ( $c'$ ) path was constrained to zero and (b) the model whereby the direct effect ( $c'$ ) path was freely estimated. If chi-square does not significantly degrade when testing the constrained model (to zero) relative to the freely estimated model, then this would support full mediation of the mediation model (i.e., that the direct path drops to 0 upon including the mediator in the model).

### 2.5.1. Path invariance analysis

The present study also examined the invariance of the structural model (i.e., invariance of the gamma [ $\zeta$ ] paths from DP to OCD symptom severity) across participants with and without a primary diagnosis of OCD. We were interested in conducting this specific test (of the pathway between DP and OCD symptoms) because of the a priori hypothesis specifically linking DP to OCD. Rate of comorbid diagnoses did not differ between the two groups. To examine invariance of the gamma path coefficients in the structural model, the gamma paths from NA to OCD symptom severity, and DP to OCD symptom severity were freely estimated across groups. In the next 'constrained' structural model, that same gamma path between DP and OCD symptom severity was constrained to be equivalent across groups. A non-significant  $\chi^2$  difference tests would indicate that the equality constraint imposed across groups does not significantly degrade overall model fit, thereby supporting equality of the constrained parameter (e.g., equal gamma path coefficients) across groups.

All reported path coefficient values are standardized coefficients, which may also be interpreted as effect-size estimates.

## 3. Results

### 3.1. Measurement model

The fit of the three-factor measurement model was good (i.e., RMSEA=.054, SRMR=.033; CFI=.99, TLI=.98). As shown in Fig. 1, all factor loadings were significant ( $p < .01$ ). The means, standard deviations, and correlations among the observed indicators are shown in Table 2.

### 3.2. Structural equation modeling

As seen in Fig. 2, the DP factor significantly predicted the OCD symptom severity factor ( $\zeta=.31, z=2.28, p < .05$ ), while NA did not significantly predict the OCD symptom severity factor ( $\zeta=.19, z=1.89, p=.06$ ).

### 3.3. Mediation model

The Baron and Kenny's (1986) causal link test supported DP as a significant mediator in the relationship between NA and OCD symptom severity (see Fig. 3). First, NA significantly predicted OCD symptom

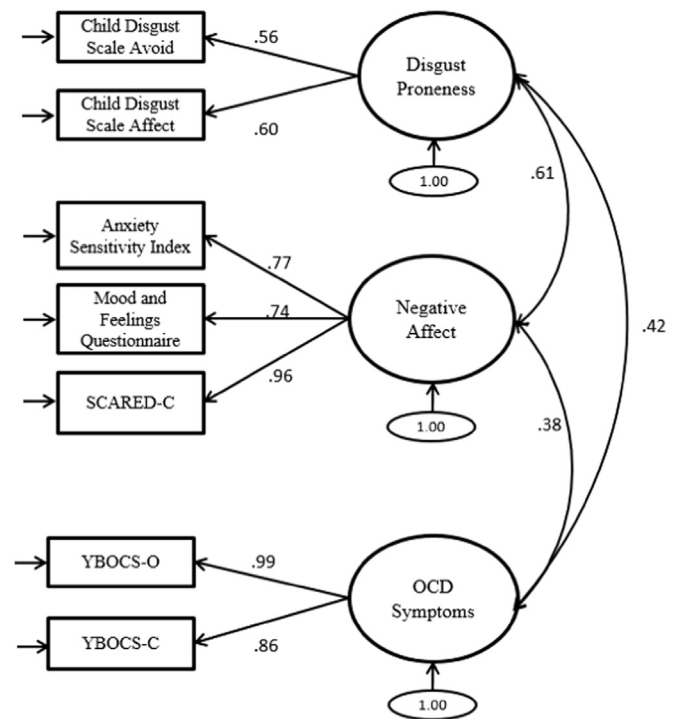


Fig. 1. Measurement model of the association between disgust proneness, negative affect, and OCD symptom severity. Note. SCARED-R = Screen for Child Anxiety Related Emotional Disorders-Revised, YBOCS-O = Yale-Brown Obsessive Compulsive Scale Obsession Subscale, YBOCS-C = Yale-Brown Obsessive Compulsive Scale Compulsion Subscale.

severity ( $\zeta=.37, z=7.90, p < .001$ ); second, the independent variable (i.e., NA) significantly predicted the DP mediator ( $\zeta=.74, z=6.09, p < .001$ ); third, the proposed mediator significantly predicted the dependent variable (i.e., OCD symptom severity;  $\zeta=.31, z=2.29, p < .05$ ) while controlling for the independent variable (i.e., NA); lastly, (4) the significant relationship between the independent variable and the dependent variable (i.e.,  $\zeta=.37, z=7.90, p < .001$ ) became non-significant (i.e.,  $\zeta=.19, z=1.89, ns$ ) once the proposed mediator (i.e., DP) was included in the model.

Second, the significance of the indirect (intervening) pathway through the proposed mediator was examined. The total standardized direct effect (between NA and OCD symptom severity) was .38,  $p < .001$ . The indirect (intervening) pathway (between NA and OCD symptom severity through DP) was significant ( $\zeta=.19, z=2.09, p < .05$ ), accounting for approximately 50% of the total direct effect. The chi-square difference test between the constrained model (whereby the direct path was constrained to 0) and the freed model (whereby the direct path was freely estimated) was then conducted. The Satorra-Bentler Scaled Chi-Square Difference Test between the constrained model [ $\chi^2(40)=196.45$ , scaling factor =1.0726] and the freed model [ $\chi^2(39)=193.48$ , scaling factor =1.0734] was 2.85,  $ns$ . The non-significant finding also supports the presence of full mediation.

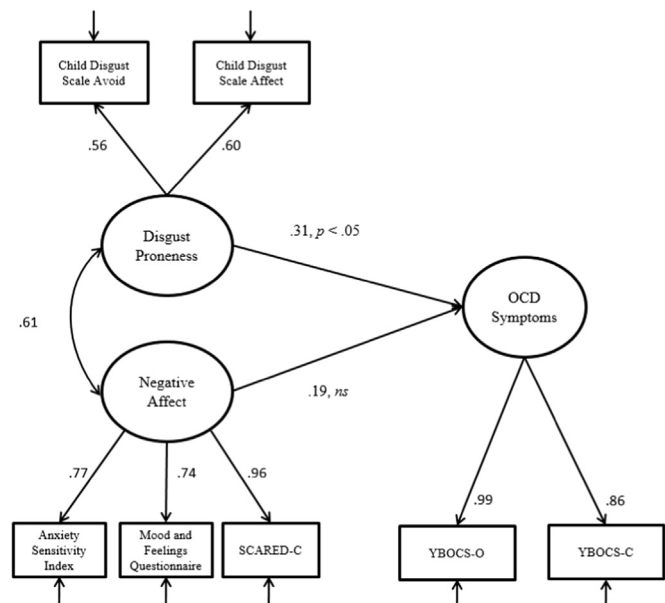
### 3.4. Path invariance analysis

The invariance of the DP to OCD symptom severity path for those with a Primary OCD Diagnosis and those without a Primary OCD Diagnosis was then examined. Table 3 shows differences between the two groups on the various latent indicators. The table shows that those with a primary diagnosis of OCD reported more OCD symptom severity and more disgust proneness than those without a primary diagnosis of OCD. In contrast, those without a primary diagnosis reported more depressive symptoms than those with a primary diagnosis of OCD. The structural models and their associated parameters for the two groups

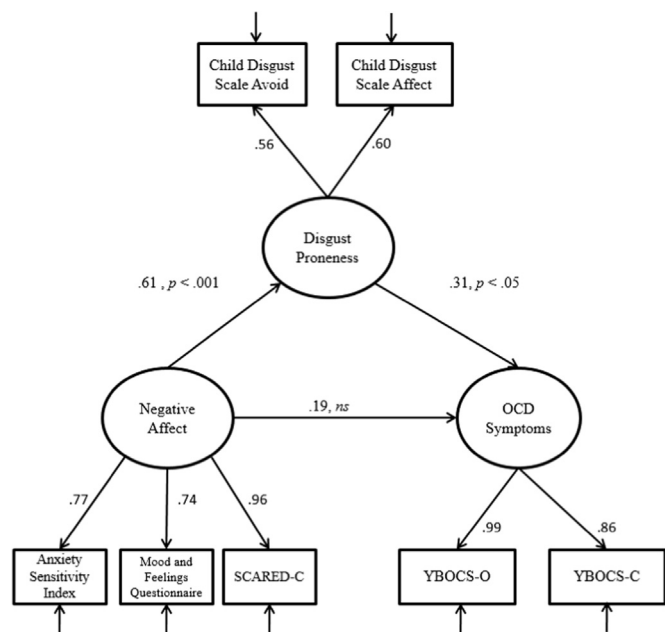
**Table 2**  
Correlations, means, and standard deviations for the observed variables.

	1	2	3	4	5	6	7
1. Anxiety sensitivity index							
2. Mood and feelings questionnaire	.56						
3. Screen for child anxiety related emotional disorders-children	.74	.71					
4. Child disgust scale - disgust avoidance subscale	.26	.17	.30				
5. Child disgust scale - disgust affect, subscale	.33	.28	.38	.33			
6. Yale-Brown obsessive compulsive scale - obsession subscale	.34	.26	.35	.30	.19		
7. Yale-Brown obsessive compulsive scale - compulsion subscale	.28	.21	.29	.31	.17	.86	
Mean	19.40	24.61	31.97	10.65	3.39	9.22	8.77
SD	13.48	15.87	18.35	4.19	2.57	5.54	5.61

Note. All correlations were significant ( $p < .001$ ).



**Fig. 2.** Structural model: disgust proneness and negative affect predicting OCD symptom severity.



**Fig. 3.** Mediation model: disgust proneness as a mediator of the relationship between negative affect and OCD symptom severity.

appear in Fig. 4. The invariance of this structural model was examined to determine whether the path from DP to OCD symptom severity differed significantly across the ‘Primary OCD Diagnosis’ group ( $\zeta = .26$ ,  $z = 1.21$ , *ns*) and the ‘No Primary OCD Diagnosis’ group ( $\zeta = .61$ ,  $z = 2.10$ ,  $p < .05$ ). Freeing the gamma path coefficient (from DP to OCD symptom severity) across groups led to significant improvement to model fit,  $\chi^2(1) = 5.477$ ,  $p < .05$ , supporting that the path coefficients from DP to OCD symptom severity differed significantly across groups.

#### 4. Discussion

Although disgust proneness has been consistently implicated in OCD among adult samples (Olatunji et al., 2005; Schienle et al., 2003), such a link has not been consistently observed among youth (Muris et al., 1999). Furthermore, no study to date has examined the unique links between disgust proneness and OCD symptoms among youth in a clinical sample. The present study was designed to address this important limitation. Consistent with prior research with adults (e.g., Olatunji et al., 2011a, 2011b; Thorpe et al., 2003), the present study found that a latent disgust proneness factor was significantly positively associated with a latent OCD symptom severity factor in a clinical sample. The present study also found that a latent negative affect factor consisting of anxiety sensitivity, depression and anxiety was significantly positively associated with OCD symptom severity in the clinical sample. These findings suggest that among youth, disgust proneness and negative affect may play a role in OCD.

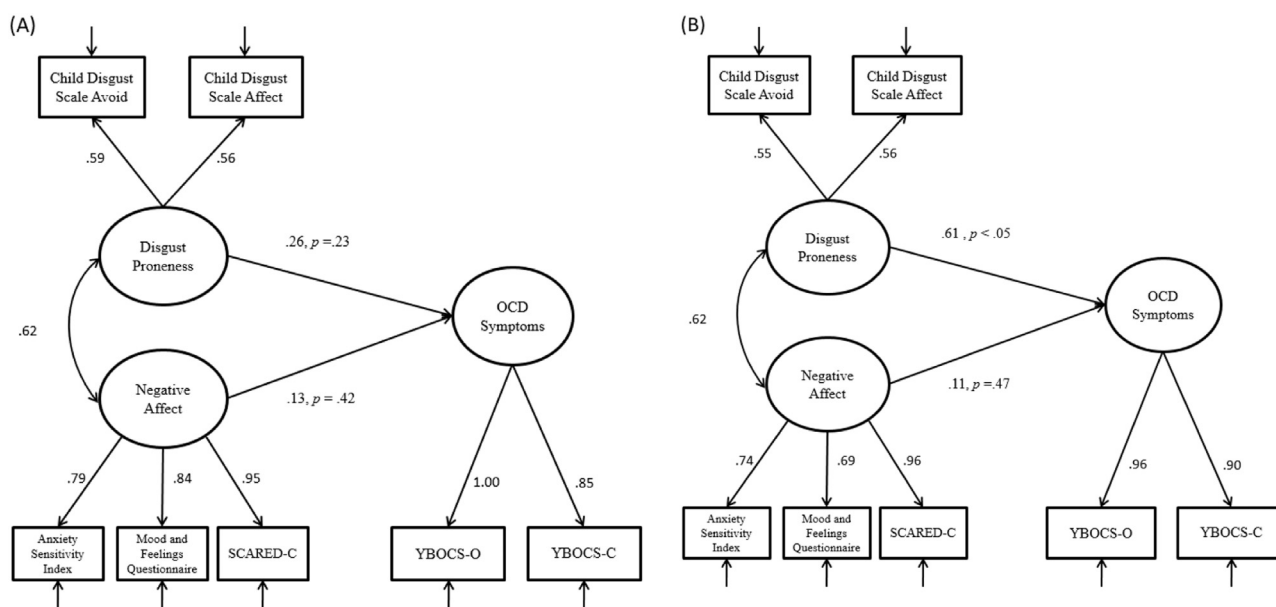
The present study also found that disgust proneness is significantly associated with negative affect. This suggests that the relationship between latent disgust proneness and OCD symptom severity may be a mere artifact of the shared variance between disgust proneness and negative affect. Consistent with this view, Muris and colleagues (1999) found the association between disgust proneness and OCD symptoms in a nonclinical sample of children became nonsignificant when controlling for trait anxiety. Findings of this sort highlight the importance of assessing concurrent levels of negative affect when examining the relationship between disgust proneness and OCD symptoms (i.e., Davey and Bond, 2006). This is necessary to ensure that the association between disgust proneness and OCD symptoms is not due to concurrent levels of negative affect. Contrary to the findings of Muris et al. (1999), results of the present study revealed that when latent disgust proneness and negative affect were simultaneously modeled as predictors, disgust proneness remained significantly associated with OCD symptom severity. This finding suggests that in a clinical sample of youth, the association between disgust proneness and OCD symptom severity is not explained by negative affect.

In the present study, the causal link test, the test of indirect effect via the mediator, and the Satorra-Bentler Scaled Chi-Square Difference Test indicated that disgust proneness is a significant intervening and mediating variable between negative affect and OCD symptom severity. Research has shown that negative affect at one point in time predicts symptoms of emotional distress at later points in peoples’ lives (Costa

**Table 3**

Means (and standard deviations) of all observed variable total scores across participants with a primary OCD diagnosis (n=243) and without a primary OCD diagnosis (n=228).

Latent Indicators	Primary OCD Diagnosis Mean (S.D.)	Non-Primary OCD Diagnosis Mean (S.D.)	t-test	p-value
1. Anxiety sensitivity index	19.48 (12.30)	19.32 (14.65)	.13	.893
2. Mood and feelings questionnaire	22.41 (14.78)	26.97 (16.67)	-3.13	.002
3. Screen for child anxiety related emotional disorders-children	31.45 (18.29)	32.52 (18.43)	-.63	.530
4. Child disgust scale - disgust avoidance subscale	11.17 (4.08)	10.09 (4.24)	2.79	.006
5. Child disgust scale - disgust affect, subscale	3.52 (2.55)	3.26 (2.59)	1.08	.282
6. Yale-Brown obsessive compulsive scale - obsession subscale	11.88 (3.91)	6.38 (5.62)	12.41	.000
7. Yale-Brown Obsessive compulsive scale - compulsion subscale	11.50 (4.11)	5.86 (5.54)	12.57	.000



**Fig. 4.** Invariance model: path invariance analysis between those (A) with a primary OCD diagnosis and (B) without a primary OCD diagnosis.

and McRae, 1980; Levenson et al., 1988). When negative affect enhances the propensity to experiencing disgust (versus other emotions), this may be a specific path that contributes to the development of OCD. This enhancement may take the form of excessive vigilance for disgusting stimuli in the environment. Processes of associate learning may also be facilitated by negative affect, particularly concerning the overgeneralization of disgust-related associations. Lastly, processes of habituation may be impeded by negative affect so that reactivity to disgusting stimuli is maintained overtime. These various mechanisms may explain how negative affect, as a generalized vulnerability to emotional disorders (e.g., Barlow, 2000), confers risk for disgust proneness, which then operates as a specific vulnerability factor for OCD.

Based on the disease-avoidance model of disgust (Davey, 2011), theoretical models posit that disgust proneness may play a larger role in OCD relative to other disorders. Consistent with this view, Olatunji et al. (2011a, 2011b) found that compared to nonclinical controls and individuals with generalized anxiety disorder, those with OCD more strongly endorsed disgust propensity. Accordingly, the presence or absence of a primary diagnosis of OCD may be expected to moderate the structural association between disgust proneness, negative affect, and OCD symptom severity. Path invariance analysis revealed that the path coefficients from latent disgust proneness to latent OCD symptom severity differed significantly between the groups. Contrary to predictions, the path from latent disgust proneness to OCD symptom severity in the structural model was significant among those without a primary diagnosis of OCD and nonsignificant among those with a primary diagnosis of OCD.

It is unclear why the path from the latent disgust proneness to OCD symptom severity would be stronger for those without OCD. One

possible explanation may be the distribution of OCD symptom severity and disgust proneness. Prior taxometric research has shown that symptoms of OCD and disgust proneness are present to a greater or lesser extent in all individuals (Olatunji and Broman-Fulks, 2007; Olatunji et al., 2008). However, the range of OCD symptom severity and disgust proneness may be more restricted among those with a primary diagnosis of OCD and this may partially explain why disgust proneness is a less robust predictor in this youth residential sample. This speculation is likely to be incorrect however, given that a Levene Test for equality of variances on the CDS total score and the CYBOCS total score revealed that the variance of the scores were not found to be significantly different across groups. An alternative explanation may be that disgust proneness is not a robust predictor of OCD symptom severity among the primary OCD group because other vulnerabilities, such as genetic factors, may be more salient in determining the severity of symptoms for those with OCD. In other words, it's possible that the genetic contributions to symptom severity are larger in the primary OCD group than in the non-primary OCD group, in which case there would be less variance left to be explained by disgust proneness in the primary OCD group. However, if genetic factors contribute less (and thus explains less of the variance) in the non-primary OCD group, then it's possible that other factors like disgust proneness may play a larger role in determining severity of symptoms in the non-primary OCD group.

The present findings build upon the existing literature in demonstrating that disgust proneness predicts OCD symptom severity in a clinical sample of youth. However, limitations of the study should be considered when interpreting these findings. For example, given the cross-sectional design, the causal inferences that can be made are

limited. The mediational model assumes that negative affect causes disgust proneness, which in turn causes OCD symptom severity. In order to establish this temporal precedence, it will be important for future research to employ a longitudinal design to test the sequencing of these predicted effects. Future research examining the extent to which the effects of negative affect are mediated by disgust proneness will benefit from a prospective approach where multiple measurement points (with multiple measures of the same construct) can be obtained. Although one important strength of the present study is the use of a newly developed measure of disgust proneness that is designed specifically for children, the present study is limited by the exclusive reliance on self-report measures. Consequently, relationships between negative affect, disgust proneness, and OCD symptoms may be inflated due to questionnaire-specific method variance (i.e., the self-report rating method). The path invariance analysis as a function of OCD diagnosis should also be interpreted with caution given the absence of structured diagnostic interviews.

Despite study limitations, the present study suggests that disgust proneness in children is related to OCD (Olatunji et al., 2011b; Taboas et al., 2015). The present findings suggest that there may also be value in incorporating the assessment of disgust proneness in treatment planning for those with OCD. Treatment planning may then require the incorporation of exposures that are specifically targeted towards improving disgust tolerance. However, a major aim of future research will be to leverage emerging research on genetic (Sherlock et al., 2016) and environmental (Stevenson et al., 2010) determinants of disgust proneness to better understand the precise mechanisms that may function as risk and protective factors in the etiology of OCD. Future longitudinal research along these lines where multiple assessment modalities are employed may then allow more definitive inferences to be made regarding the role of disgust proneness in the development and maintenance of OCD in youth.

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