Shorter communication

An investigation of traumatic life events and obsessive-compulsive disorder

Kiara R. Cromer[a],*, Norman B. Schmidt[a], Dennis L. Murphy[b]

[a]*Department of Psychology, Florida State University, Tallahassee, FL 32306-1270, USA*
[b]*Laboratory of Clinical Science, NIMH, USA*

Abstract

Obsessive-compulsive disorder (OCD), like most other psychiatric disorders, is suspected of being influenced by an interaction between life events and genes, both with regard to onset and course of illness. To date, no specific genes have been identified as playing a frequent role, and only a relatively few empirical studies have assessed the association between stressful life events (SLEs) and OCD. The present study builds on past research by examining the potential contributions from traumatic life events (TLEs) on the severity and symptom features in 265 individuals with Structured Clinical Interview for DSM-IV (SCID)-diagnosed OCD. Of these participants 54% endorsed having experienced at least one TLE in their life time. The presence of one or more TLEs was associated with increased OCD symptom severity. This relationship remained significant despite controlling for key variables including age, OCD age-of-onset, comorbidity, and depressive symptoms. In addition, *obsessions/checking* and *symmetry/ordering* were two of four symptom factors that were specifically associated with the occurrence of TLEs. These results are generally supportive of a pathoplastic relationship between TLEs and OCD symptomatology and thus suggest the need for greater systematic consideration of life stresses in research focused on the nature and treatment of OCD.
© 2006 Published by Elsevier Ltd.

Keywords: Obsessive-compulsive disorder; Traumatic stress; Symptom dimensions

Introduction

Obsessive compulsive disorder (OCD), an anxiety disorder manifested by distressing and recurrent obsessions and compulsions, has been identified as one of the most chronic and costly forms of psychopathology (Dupont, 1993; Lopez & Murray, 1998). Historically considered a rare disorder, recent epidemiological findings have indicated a lifetime prevalence of approximately 1.6–3% (e.g., Kessler et al., 2005; Weissman et al., 1994). While research during the last few decades has expanded our understanding of the classification and treatment of OCD, many of the mechanisms and vulnerability factors involved in the etiology and maintenance of this disorder remain unknown.

---

*Corresponding author. Tel.: +1 850 644 1766; fax: +1 850 644 7739.
E-mail address: cromer@psy.fsu.edu (K.R. Cromer).

One putative vulnerability factor for psychiatric disorders is stressful life events (SLEs) including traumatic life events (TLEs). In his book *Hemmung, Symptom, und Angst*, Freud (1926) hypothesized that anxiety was a reaction to experiencing stressful events involving danger and loss. Subsequent empirical research has consistently associated SLEs with mood disorders (e.g., Bifulco, Brown, & Adler, 1991; Caspi et al., 2003) and anxiety psychopathology (e.g., Angst & Vollrath, 1991). Substantial evidence now exists identifying SLEs as a key factor in anxiety-related responding, and there are also experimental data to suggest that such events may act as catalysts for the onset of symptoms (Paykel & Dowlatshahi, 1988; Zvolensky, Eifert, & Lejuez, 2001). Although the impact of SLEs on the development and maintenance of psychiatric disorders has been extensively examined, their association with OCD has been only marginally investigated.

Three lines of research have provided preliminary evidence linking stress and OCD. Numerous case studies have suggested a potential association between SLEs (particularly trauma) and the development of OCD symptoms (Janet, 1903; Pitman, 1987). More systematic support for this connection comes from experimental investigations focusing on general stress in nonclinical populations, such as those finding increased intrusive thoughts in response to stressful and aversive stimuli (e.g., Horowitz, 1975; Jones & Menzies, 1998). Although the majority of these experimental investigations have primarily dealt with nonclinical OC symptoms and *generally* stressful experiences (as opposed to trauma), these findings highlight the potential impact the environment has in inducing obsessive impulses.

A wide range of correlational clinical investigations represents a third line of research underscoring an association between SLEs/TLEs and OCD. Studies of traumatized populations have found that in addition to the onset of post-traumatic stress disorder (PTSD), comorbid OCD can emerge following exposure to an extremely upsetting event (e.g., high-combat exposure; de Silva & Marks, 1999; Pitman, 1993), and that traumatized (versus nontraumatized) populations are more likely to meet diagnosis for OCD (Jordan et al., 1991; Solomon, 1993). For example, Saunders, Villeponteaux, Lipovsky, Kilpatrick, and Veronen (1992) found that individuals who had experienced childhood sexual abuse were approximately five times more likely to have OCD than nonabused individuals.

A small number of studies have attempted to elucidate the role of life stress in the onset of OCD. Initial reports found that the majority of patients in "obsessional states" believed that a specific event was responsible for their symptoms (Ingram, 1961; Lo, 1967; Pollitt, 1957); however, given methodological shortcomings, such as inconsistent diagnostic criteria, viable conclusions are limited. A more recent pediatric study found that patients reported significantly more life events in the year preceding onset of OCD when compared to healthy controls (Gothelf, Aharonovsky, Horesh, Carty, & Apter, 2004). Similarly, McKeon, Roa, and Mann (1984) assessed 25 adults with OCD using Paykel's Life Events Schedule, and found that compared to nonclinical controls, patients experienced more SLEs in the year preceding onset. Yet, other investigations have found no such differences making it difficult to draw conclusions (Maina, Albert, Bogetto, Vaschetto, & Ravizza, 1999). There is some indication that the post-partum period represents a potential risk factor for OCD in females, but again, findings have been inconsistent and birth as a stressor most likely is a unique and distinct SLE (e.g., Williams & Koran, 1997).

The relationship between stress and the *course* of OCD has received even less empirical attention. Although early investigations examining the relationship between SLEs and OCD considered the prognostic value of stress, the association with course was never systematically assessed (e.g., Pollitt, 1957). More recently, Rasmussen and Eisen (1991) found that patients reported a worsening of OCD symptoms after experiencing every-day SLEs. Similarly, a pediatric study found that children with Tourette's syndrome and/or OCD experienced significantly more daily stress when compared to nonclinical controls (Findley et al., 2003). The authors concluded that the higher numbers of SLEs may play a role in the waxing and waning of symptoms often seen in OCD.

The vast majority of investigations evaluating life events and OCD have typically described this relationship within a diathesis-stress framework. Yet, it is important to consider a broad range of associations that could capture the ways in which stress influences the disorder (Clark, Watson, & Mineka, 1994). In addition to a predisposition/diathesis-stress approach, the pathoplastic model may be a useful means of describing the association between SLEs and OCD. According to this model, individual differences in the experience of stressors could affect the expression of the clinical condition. In other words, regardless of the role stress may play in the development of OCD, the pathoplastic model proposes that SLEs may modify the manifestation of

OCD. As far as could be determined, there have been no explicit attempts to assess a pathoplastic relationship between SLEs and OCD.

The present study seeks to more fully examine the potential relationship(s) between TLEs and OCD within the context of a pathoplastic model in a large adult clinical sample. Our aim was to conduct a systematic analysis of the association between TLEs and a number of clinical facets of OCD including severity and symptomatology. Consistent with previous research, we hypothesized that TLEs would be related to a more complicated clinical expression of OCD as reflected by increased symptom severity.

Method

Participants

The sample consisted of 265 consecutive admissions to the Adult OCD Clinic at the National Institute of Mental Health (NIMH). These patients were part of a larger genetic investigation and were recruited through physician/psychologist referrals, websites, and advertisements in local newspapers. Inclusion criteria for participation included being at least 18 years old and having a primary OCD diagnosis based on the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2001). Exclusion criteria included active schizophrenia or psychosis, severe mental retardation that does not permit an evaluation to characterize OCD, or OCD symptoms that occur exclusively in the context of depression.

The mean age of the sample was 41 (SD = 15.02) and the majority of participants were female (62.9%) and Caucasian (95%, with 1.6% Hispanic, 1.1% African American, 1.1% Asian, and 1.1% other). The average symptom severity, as measured by the Yale-Brown Obsessive Compulsive Scale (Goodman et al., 1989), was 21.63 (SD = 9.34), and the mean age of OCD onset was 14.18 years (SD = 8.67; range 3–55 yr). Overall participants were well educated (67% obtained a college degree or higher) and over half (52%) had a history of marriage. With regard to comorbidity features, 90% of participants were diagnosed with at least one additional lifetime Axis I psychological disorder. The number of comorbid Axis I disorders ranged from 0 to 8 (M = 2.46, SD = 1.79), with the most frequently comorbid disorder being major depressive disorder (65%). The mean current BDI score was 14.85 (SD = 1.79; range 0–59).

Measures

Structured Clinical Interview for DSM-IV (SCID)

Participants were interviewed using the SCID-P for DSM-IV (First et al., 2001), which is a semi-structured interview designed to determine lifetime and current diagnoses of major DSM Axis I disorders. The SCID interviews were administered by trained and clinically experienced interviewers. In an effort to reach further reliability, each participant's SCID was re-assessed by two independent clinicians for blind diagnosing. SCID diagnoses in previous studies conducted by this group have demonstrated excellent reliability (LaSalle et al., 2004). The variable "psychological co-morbidity" was defined as the presence of any DSM Axis I disorder in addition to the primary diagnosis of OCD, including: major depressive disorder, dysthymia, bipolar I or II, social phobia, panic disorder, agoraphobia, specific phobia, generalized anxiety disorder, eating disorders, Tourette's syndrome, trichotillomania, and substance use disorders.

Yale-Brown Obsessive Compulsive scale (Y-BOCS)

The magnitude of obsessions and compulsions was measured with the Y-BOCS (Goodman et al., 1989), a frequently used assessment tool for the severity of OC symptoms. Specific symptoms were assessed using the Y-BOCS Symptom Checklist (Y-BOCS-SC) a symptom inventory that has demonstrated high reliability and validity (e.g., Leckman et al., 1997). The Y-BOCS-SC was used to derive factor scores for the symptom dimensions of OCD (Hasler et al., 2005). Specifically, principal component analysis was applied to the 13 a priori Y-BOCS categories and initial factor solutions were then rotated using the Varimax procedure (Hasler et al., 2005; Leckman et al., 1997). The factor analysis generated four factor scores for each subject representing the correlation of the symptom profile of the subject with each factor. The four factors included *obsessions/checking, symmetry/ordering, contamination/cleaning,* and *hoarding.* The factor scores were

standardized, with *mean* = 0 and SD = 1. These symptom dimensions have been shown to be temporally stable and are associated with differential treatment response (Mataix-Cols, Rosario-Campos, & Leckman, 2005) and comorbidity patterns (Hasler et al., 2005).

TLE measure

Life stress was assessed via diagnostic interview. Specifically, the information provided in the ''Traumatic Events List'' contained in the PTSD module of the SCID was used to assess TLEs. The PTSD section of the SCID begins by providing participants with a brief description of extremely upsetting, stressful or traumatic life experiences. Following this definition, the participants were asked an open-ended question regarding their own experiences with such events. The interviewer recorded all reported events. The participants were then asked if they were still troubled by any of the incidents they recounted. If so, the interviewer proceeded with the PTSD diagnosis module. This method insures that significant TLEs were documented regardless of whether or not an individual met criteria for PTSD. The various events reported were classified into 17 different categories. In the present report, TLE was indicated by a dichotomous variable indicating lifetime presence or absence of one or more TLE.

Beck Depression Inventory (BDI)

The BDI is a 21-item measure of global depression that assesses current mood. It is a reliable and validated measure of depressive symptomatology (Beck, Steer, & Garbin, 1988).

Results

General data analytic strategy

A series of regression analyses were utilized to examine the relationship between TLEs and OCD. Linear regression was used to determine if TLEs (independent variable) were associated with increased Y-BOCS scores (dependent variable), despite controlling for a number of other variables including age, OCD age-of-onset, and comorbidity. We next conducted a covariance strategy, as outlined by Kendall and Ingram (1989), to assess the predictive specificity of TLEs. That is, it would be important to ensure that any potential relationship between TLEs and OCD is not better explained by a relevant third variable. Depressive symptomatology was selected to test the uniqueness of the association between TLEs and OCD, given the extensive literature tying depression to the experience of stress (e.g., Caspi et al., 2003) and also the high comorbidity rates between OCD and depression. We first constructed an equation with Y-BOCS total score as the dependent variable, and BDI scores entered in Step 1, followed by TLEs in Step 2. For the second equation, BDI scores served as the dependent variable, while Y-BOCS total scores were entered as the covariate in Step 1. This approach allowed for the assessment of the relation of TLEs to OCD symptoms beyond the effects of depressive symptoms, as well as the assessment of the relation of TLEs to depression beyond the effects of OCD symptoms. Finally, a logistic regression analysis was used to evaluate the unique variance accounted for by each OCD symptom dimension on TLE, by regressing all four symptom factor scores simultaneously onto the TLEs measure.

Descriptive data and zero-order relations among theoretically relevant variables

Zero-order correlations among the key variables, in addition to means, standard deviations, and alpha internal consistency coefficients (when applicable) are presented in Table 1. It was found that 54% of the sample experienced at least one TLE and that the maximum number of TLEs experienced by any one person was five. It should be noted that the rate of endorsement for TLEs was considerably higher than the rate of PTSD (54% versus 10%). Of those events reported ($N = 238$), *accident participant* was the most frequent, with 16% (38 events) of events falling into this category. The next four most frequent events were: *nonviolent death witnessed* (10%; 24 events), followed by *crime witnessed* (9%; 22 events), *sexual abuse* (9%; 22 events), and *illness of self or loved one* (8%; 19 events).

Table 1
Intercorrelations between TLE and variables of interest

	Variable	1	2	3	4	5	6	7	8	9	10	*M*	SD	α
1	TLE	—	0.32	0.19	0.17	0.17	0.19	0.22	0.17	0.06	0.07	na	na	na
2	PTSD Dx		—	0.15	0.14	0.13	0.18	0.22	0.16	0.17	0.07	na	na	na
3	Y-BOCS Total			—	0.91	0.89	0.28	0.43	0.37	0.39	0.10	21.2	9.0	na
4	Y-BOCS Obsess				—	0.62	0.28	0.48	0.33	0.30	0.05	10.8	5.3	na
5	Y-BOCS Comp					—	0.21	0.28	0.34	0.40	0.13	10.5	4.8	na
6	BDI						—	0.49	0.20	0.33	0.22	14.6	10.9	na
7	Factor 1							—	0.44	0.40	0.22	na	na	0.71
8	Factor 2								—	0.40	0.23	na	na	0.73
9	Factor 3									—	0.11	na	na	0.75
10	Factor 4										—	na	na	0.71

Note: TLE = dichotomous measure of traumatic life events; (2) PTSD Dx = Post traumatic stress disorder diagnosis; (3) Y-BOCS total = Yale–Brown Obsessive-Compulsive Scale total score; (4) Y-BOCS obsess = Yale–Brown Obsessive-Compulsive Scale obsessions score; (5) Y-BOCS Comp = Yale–Brown Obsessive-Compulsive Scale compulsions score; (6) BDI = Beck Depression Inventory; (7) Factor 1 = Y-BOCS *obsessions/checking*; (8) Factor 2 = Y-BOCS *symmetry/ordering*; (9) Factor 3 = Y-BOCS *contamination/cleaning*; (10) Factor 4 = Y-BOCS *hoarding*.
na = not applicable.

Association between TLEs and OCD symptom severity

Our first hypothesis was that TLEs would be associated with increased OCD symptom severity. Consistent with expectation, analyses revealed that TLEs were associated with increased Y-BOCS total scores ($\beta = 0.19$, $t(241) = 3.06$, $p < 0.001$), as well as increased Y-BOCS obsessions ($\beta = 0.17$, $t(241) = 2.6$, $p < 0.001$) and compulsions ($\beta = 0.17$, $t(241) = 2.74$, $p < 0.001$), considered separately. Analyses were repeated with age and OCD age-of-onset as covariates. These variables were chosen because age-of-onset is associated with increased OCD symptom severity and age is associated with the likely experience of a TLE. Results indicated that despite controlling for these covariates, TLEs remained significantly associated with Y-BOCS total, obsessions, and compulsions scores.

In considering the association between OCD and TLEs we also felt it would be important to determine whether this relationship might be explained by greater overall severity. That is, TLEs might lead to increased levels of general psychopathology, and increased OCD symptom severity levels might thus simply reflect this association. We selected psychological comorbidity as a proxy for general psychiatric severity level, given that comorbidity rates are exceedingly high in OCD samples (92% of participants received one or more additional Axis I diagnoses) and have been tied to increased symptom severity (LaSalle et al., 2004) and poorer treatment response (Carrasco, Hollander, Schneier, & Liebowitz, 1992). The analyses were repeated adding psychological comorbidity as a covariate. Of note, individuals with comorbid PTSD were removed from the sample for these analyses since the TLE measure was derived from the PTSD module of the SCID. Results revealed that despite controlling for age, age-of-onset, and comorbid disorders, TLEs were still significantly associated with increased Y-BOCS total scores ($\beta = 0.16$, $t(241) = 2.52$, $p < 0.01$), as well as increased Y-BOCS obsessions ($\beta = 0.15$, $t(241) = 2.24$, $p < 0.05$) and compulsions ($\beta = 0.14$, $t(241) = 2.11$, $p < 0.05$).

Uniqueness of the association between TLEs and OCD symptom severity

In order to evaluate whether TLEs have a unique association with OCD, we conducted a covariance strategy with depression (i.e., BDI scores). Results, presented in Table 2, indicated that TLEs were a significant predictor of Y-BOCS total score beyond the effects of age, age-of-onset, and BDI scores ($\beta = 0.16$, $t(159) = 1.97$, $p < 0.05$). In contrast, the TLEs prediction of BDI scores controlling for age, age-of-onset, and Y-BOCS scores was a nonsignificant trend ($\beta = 0.15$, $t(159) = 1.79$, $p = 0.08$). This pattern of findings suggests that TLEs are more specifically related to a diagnosis of OCD than to depressive symptoms at the time of assessment.

Table 2
Summary of hierarchical regression analyses for TLE predicting OCD and depressive symptom severity

DV	Step	ΔR^2	B	SE (B)	β	t
Y-BOCS total	Step 1: Covariates					
	Age	0.04	−0.03	0.05	−0.05	−0.53
	Age-of-onset		−0.18	0.08	−0.19	−2.17*
	Step 2: MDD Symptoms					
	BDI	0.07	0.22	0.07	0.27	3.37**
	Step 3: Stress					
	TLE	0.02	2.83	1.44	0.16	1.97*
BDI total	Step 1: Covariates					
	Age	0.01	−0.01	0.06	−0.02	−0.18
	Age-of-onset		−0.13	0.1	−0.11	−1.25
	Step 2: OCD symptoms					
	Y-BOCS	0.07	0.33	0.1	0.27	3.37**
	Step 3: Stress					
	TLE	0.02	3.18	1.78	0.15	1.79 trend

Note: TLE = traumatic life event experienced yes/no; Y-BOCS = Yale–Brown Obsessive-Compulsive Scale total score; BDI = Beck Depression Inventory; age = age at time of study; age-of-onset = age at which OCD onset.
*$p < 0.05$, **$p < 0.01$, trend = $0.1 > p > 0.05$.

Table 3
Comparison of the four symptom dimensions in predicting TLEs

DV	Predictor	B	Wald	Exp (B)	95% CI for B
TLE	Factor 1	0.33	3.70*	1.31	1.0–1.9
	Factor 2	0.23	1.88	1.26	0.9–1.7
	Factor 3	−0.21	1.75	0.81	0.6–1.1
	Factor 4	0.01	0.01	1.01	0.8–1.4

Note: TLE = traumatic life event experienced yes/no; Factor 1 = Y-BOCS *obsessions/checking*; Factor 2 = Y-BOCS *symmetry/ordering*; Factor 3 = Y-BOCS *contamination/cleaning*; Factor 4 = Y-BOCS *hoarding*.
*$p < 0.05$.

Association between TLEs and symptom dimensions

We next conducted a series of exploratory analyses to determine whether certain symptom dimensions would be more strongly associated with TLEs. First, alpha internal consistency coefficients were calculated to evaluate whether the four Y-BOCS symptom dimensions had comparable reliabilities. Alpha coefficients (see Table 1) were fairly consistent across the four factors and were deemed acceptable (> 0.7). Zero-order correlations revealed that Y-BOCS *obsessions/checking* was significantly and positively correlated with TLEs ($r(242) = .22$, $p < 0.001$), as was s*ymmetry/ordering* ($r(242) = 0.17$, $p < 0.01$). In contrast, neither the *contamination/cleaning* ($r(242) = 0.06$, $p < 0.35$) nor the *hoarding* dimensions ($r(242) = 0.07$, $p < 0.25$) were significantly correlated with the experience of TLEs. We also examined the unique variance accounted for by each OCD symptom dimension with regard to TLEs. Specifically, using logistic regression we simultaneously regressed all four symptom dimensions onto TLEs, the dependent variable. Results are presented in Table 3. It was found that *obsessions/checking* explained the most variance, followed by *symmetry/ordering*, then *hoarding*, and finally *contamination/cleaning*.

Similar to the symptom severity analyses discussed above, we assessed the robustness of the relationship between TLEs and those factors that were significantly correlated with trauma. We found that the association between TLEs and *obsessions/checking* ($\beta = 0.16$, $t(242) = 2.14$, $p < 0.05$) and *symmetry/ordering* ($\beta = 0.15$, $t(242) = 2.39$, $p < 0.05$) remained significant despite controlling for age, age-of-onset, and total comorbidity.

Discussion

The current study investigated the potential relationship between TLEs and OCD symptomatology within a pathoplastic model and across important clinical domains. Data are supportive of such a relationship. Consistent with our hypothesis, TLEs were associated with a more severe clinical expression of OCD as reflected in Y-BOCS severity ratings. This finding corroborates previous investigations, such as the experimental studies on nonclinical samples (McLaren & Crowe, 2003) and the handful of clinical investigations that have assessed the impact of SLEs on OCD severity (e.g., Rasmussen & Eisen, 1991). Importantly, we demonstrated that this relationship remained despite controlling for a number of influential factors, including comorbidity and depressive symptomatology. We thus have established that TLEs are linked with OCD specifically, rather than with general severity. To our knowledge, this is the first investigation that has assessed the nonspuriousness of the association between TLEs and OCD.

To further examine this relationship, we assessed whether TLEs would be more strongly associated with specific OCD symptom dimensions. Results indicated that TLEs were significantly correlated with *obsessions/checking* and *symmetry/ordering* factors. Follow-up analyses revealed that higher scores on both the *obsessions/checking* and *symmetry/ordering* dimensions had a robust association with TLEs. Comorbidity may help explain our findings that the *obsessions/checking* and *symmetry/ordering* (versus *hoarding* or *contamination/cleaning*) symptom dimensions had the strongest association with TLEs. A recent investigation by Hasler et al. (2005) found that *obsessions/checking* and *symmetry/ordering* are especially associated with mood and anxiety disorders. Considering this finding in light of the extensive and robust evidence tying mood and other anxiety disorders to TLEs (Brown & Harris, 1993), a model emerges that may explain our results. Perhaps *obsessions/checking* and *symmetry/ordering* are associated with TLEs through their increased comorbidity with mood and anxiety disorders. Future studies should assess the link between TLEs and specific obsessions/compulsions taking into consideration comorbidity patterns.

Garber and Hollon (1991) have outlined three criteria for demonstrating that a certain variable, such as TLEs, acts as a vulnerability factor for a psychiatric disorder. According to their guidelines, it is necessary to demonstrate: (1) covariance between the vulnerability factor and an outcome; (2) a nonspurious relationship between the vulnerability factor and an outcome; and (3) the temporal precedence of a vulnerability factor. Due to our methodological constraints we are unable to assess the third criterion specified. We have, however, addressed the first two requirements. Results from our study thus provide initial support for a pathoplastic relationship and TLEs as a vulnerability factor for OCD. Further investigation of the role that TLEs play is vital though, considering that the current investigation is unable to rule out the possibility that the relationship between OCD and TLEs would better fit a predisposition (or other) vulnerability model.

Our findings should be considered in light of several limitations, such as the retrospective collection of data, a frequently discussed problem inherent in this design (Maina et al., 1999). Acknowledging this limitation, we were careful not to draw any conclusions about the relationship between TLEs and the *onset* of OCD. Our measure of TLEs is an additional constraint since it was based on the traumatic events listed in the PTSD SCID module. While this module has been thoroughly validated and consistently and reliably used to identify individuals with PTSD retrospectively, indices of reliability and validity for our specific measure of TLEs have not yet been established.

A clearer test of the pathoplastic vulnerability model would have been to only consider those TLEs that occurred following the onset of OCD. Unfortunately the exact age at which each TLE took place was not adequately assessed in our sample, and given the archival nature of our study we were therefore unable to focus solely on the events that took place post-onset. Future investigations should consider this approach when examining the pathoplastic relationship between TLEs and OCD. Finally, the possibility exists that individuals with greater OCD severity may be more sensitive to recalling TLEs. In other words, it may be that the more severe patients exaggerate the causal impact of certain life events that other individuals would consider minimally or not at all stressful. Our investigation lacks the prospective design which would be necessary to test this possibility. That being said, the questions regarding the experience of TLEs were built into the PTSD module of the SCID, not the OCD module. Moreover, there was no indication from the interviewer that the participant should think of events related in one way or another to their diagnosis of OCD. Examination of the types of events experienced provides further support that event exaggeration may

not be a substantial problem. Of the top five most frequently experienced events, three (accident participant, sexual abuse, and crime witnessed) are events that very few individuals would classify as *non*stressful, and are thus not likely to be influenced by a diagnosis of OCD.

The results generated from this study have multiple implications for future research in OCD. One avenue would be to clarify the link between TLEs, OCD and comorbidity, particularly that with depression. In addition, the distinction between a pathoplastic model and a predisposition model could be explored using experimental procedures utilizing laboratory stressors. Furthermore, it would be informative to conduct prospective clinical investigations to demonstrate the temporal association between TLEs and OCD. Regardless of whether or not TLEs are identified as a vulnerability factor for OCD, assessing the interaction between stress and a diathesis, such as specific gene susceptibility regions, could unearth potential pathogenic mechanisms.

References

Angst, J., & Vollrath, M. (1991). The natural history of anxiety disorders. *Acta Psychiatrica Scandinavica*, *84*(5), 446–452.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the beck depression inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100.

Bifulco, A., Brown, G. W., & Adler, Z. (1991). Early sexual abuse and clinical depression in adult life. *British Journal of Psychiatry*, *159*, 115–122.

Brown, G. W., & Harris, T. O. (1993). Aetiology of anxiety and depressive disorders in an inner-city population. 1. Early adversity. *Psychological Medicine*, *23*(1), 143–154.

Carrasco, J. L., Hollander, E., Schneier, F. R., & Liebowitz, M. R. (1992). Treatment outcome of obsessive compulsive disorder with comorbid social phobia. *Journal of Clinical Psychiatry*, *53*(11), 387–391.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-htt gene. *Science*, *301*(5631), 386–389.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, *103*(1), 103–116.

de Silva, P., & Marks, M. (1999). The role of traumatic experiences in the genesis of obsessive-compulsive disorder. *Behaviour Research and Therapy*, *37*(10), 941–951.

Dupont, R. L. (1993). Town Hall panel presentation. In Proceedings of *The Anxiety Disorders of America Conference*. Charleston, SC.

Findley, D. B., Leckman, J. F., Katsovich, L., Lin, H., Zhang, H., Grantz, H., et al. (2003). Development of the Yale children's global stress index (YCGSI) and its application in children and adolescents with Tourette's syndrome and obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*(4), 450–457.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2001). *Structured clinical interview for DSM-IV-TR Axis I disorders, research version, patient edition (SCID-I/P)*. New York: Biometrics Research, New York State Psychiatric Institute.

Freud, S. (1926). *Hemmung, symptom und angst*. Frankfurt, Germany: Fischer Taschenbuecher.

Garber, J., & Hollon, S. D. (1991). What can specificity designs say about causality in psychopathology research? *Psychological Bulletin*, *110*, 129–136.

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., et al. (1989a). The Yale–Brown obsessive compulsive scale. II. Validity. *Archives of General Psychiatry*, *46*(11), 1012–1016.

Gothelf, D., Aharonovsky, O., Horesh, N., Carty, T., & Apter, A. (2004). Life events and personality factors in children and adolescents with obsessive-compulsive disorder and other anxiety disorders. *Comprehensive Psychiatry*, *45*(3), 192–198.

Hasler, G., LaSalle-Ricci, V. H., Ronquillo, J. G., Crawley, S. A., Cochran, L. W., Kazuba, D., et al. (2005). Obsessive-compulsive disorder symptom dimensions show specific relationships to psychiatric comorbidity. *Psychiatry Research*, *135*(2), 121–132.

Horowitz, M. J. (1975). Intrusive and repetitive thoughts after experimental stress. A summary. *Archives of General Psychiatry*, *32*(11), 1457–1463.

Ingram, I. M. (1961). Obsessional illness in mental hospital patients. *The Journal of Mental Science*, *107*, 382–402.

Janet, P. (1903). *Les obsessions et la psychasthenie*. (2nd ed.) Vols. 1 & 2. Paris: Alcan.

Jones, M. K., & Menzies, R. G. (1998). Role of perceived danger in the mediation of obsessive-compulsive washing. *Depression and Anxiety*, *8*(3), 121–125.

Jordan, B. K., Schlenger, W. E., Hough, R., Kulka, R. A., Weiss, D., Fairbank, J. A., et al. (1991). Lifetime and current prevalence of specific psychiatric disorders among Vietnam veterans and controls. *Archives of General Psychiatry*, *48*(3), 207–215.

Kendall, P. C., & Ingram, R. E. (1989). Cognitive-behavioral perspectives: Theory and research on depression and anxiety. In P. C. Kendall, & D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features. Personality, psychopathology, and psychotherapy* (pp. 27–53). San Diego, CA: Academic Press.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602.

LaSalle, V. H., Cromer, K. R., Nelson, K. N., Kazuba, D., Justement, L., & Murphy, D. L. (2004). Diagnostic interview assessed neuropsychiatric disorder comorbidity in 334 individuals with obsessive-compulsive disorder. *Depression and Anxiety*, *19*(3), 163–173.

Leckman, J. F., Grice, D. E., Boardman, J., Zhang, H., Vitale, A., Bondi, C., et al. (1997). Symptoms of obsessive-compulsive disorder. *American Journal of Psychiatry*, *154*(7), 911–917.

Lo, W. H. (1967). A follow-up study of obsessional neurotics in Hong Kong Chinese. *British Journal of Psychiatry*, *113*(501), 823–832.

Lopez, A. D., & Murray, C. C. (1998). The global burden of disease, 1990–2020. *Nature Medicine*, *4*(11), 1241–1243.

Maina, G., Albert, U., Bogetto, F., Vaschetto, P., & Ravizza, L. (1999). Recent life events and obsessive-compulsive disorder: The role of pregnancy/delivery. *Psychiatry Research*, *89*(1), 49–58.

Mataix-Cols, D., Rosario-Campos, M. C., & Leckman, J. F. (2005). A multidimensional model of obsessive-compulsive disorder. *American Journal of Psychiatry*, *162*(2), 228–238.

McKeon, J., Roa, B., & Mann, A. (1984). Life events and personality traits in obsessive-compulsive neurosis. *British Journal of Psychiatry*, *144*, 185–189.

McLaren, S., & Crowe, S. F. (2003). The contribution of perceived control of stressful life events and thought suppression to the symptoms of obsessive-compulsive disorder in both non-clinical and clinical samples. *Journal of Anxiety Disorders*, *17*(4), 389–403.

Paykel, E. S., & Dowlatshahi, D. (1988). Life events and mental disorder. In S. Fisher, & J. Reason (Eds.), *Handbook of life stress, cognition and health*. New York: Wiley.

Pitman, R. K. (1987). Pierre Janet on obsessive-compulsive disorder (1903). Review and commentary. *Archives of General Psychiatry*, *44*(3), 226–232.

Pitman, R. K. (1993). Posttraumatic obsessive-compulsive disorder: A case study. *Comprehensive Psychiatry*, *34*(2), 102–107.

Pollitt, J. (1957). Natural history of obsessional states; a study of 150 cases. *British Medical Journal*, *32*(5012), 194–198.

Rasmussen, S. A., & Eisen, J. L. (1991). Phenomenology of OCD: Clinical subtypes, heterogeneity and coexistence. In J. Zohar, T. R. Insel, & S. Rasmussen (Eds.), *The psychobiology of obsessive-compulsive disorder*. New York: Springer.

Saunders, B. E., Villeponteaux, L. A., Lipovsky, J. A., Kilpatrick, D. G., & Veronen, L. J. (1992). Child sexual assault as a risk factor for mental disorders among women. *Journal of Interpersonal Violence*, *7*(2), 189–204.

Solomon, Z. (1993). *Combat stress reactions: The enduring toll of war*. New York: Plenum Press.

Weissman, M. M., Bland, R. C., Canino, G. J., Greenwald, S., Hwu, H. G., Lee, C. K., et al. (1994). The cross national epidemiology of obsessive compulsive disorder. The cross national collaborative group. *Journal of Clinical Psychiatry*, *55*(Suppl.), 5–10.

Williams, K. E., & Koran, L. M. (1997). Obsessive-compulsive disorder in pregnancy, the puerperium, and the premenstrum. *Journal of Clinical Psychiatry*, *58*(7), 330–334 (quiz 335–336).

Zvolensky, M. J., Eifert, G. H., & Lejuez, C. W. (2001). Offset control during recurrent 20% carbon dioxide-enriched air induction: Relation to individual difference variables. *Emotion*, *1*(2), 148–165.