

Uncertainty as a mechanism in obsessive-compulsive disorder and its treatment

By

Kelly A. Knowles

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Approved:

Bunmi O. Olatunji, Ph.D.

Jennifer U. Blackford, Ph.D.

David A. Cole, Ph.D.

Steven D. Hollon, Ph.D.

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To anyone struggling with life's uncertainties

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At some point during the process of writing this dissertation, I stumbled upon my family's genealogy. Its introduction, with slight modifications, fits well here:

“The preparation of this work has cost me the labor of several years...I have travelled hundreds of miles [to conferences and interviews], searched [many databases], written innumerable [papers, chapters, abstracts, and grant applications], requiring, in all, a degree of labor at which I am astonished myself, as I look back upon it.” – Rev. David D. Field, *A Genealogy of the Brainerd Family in the United States*

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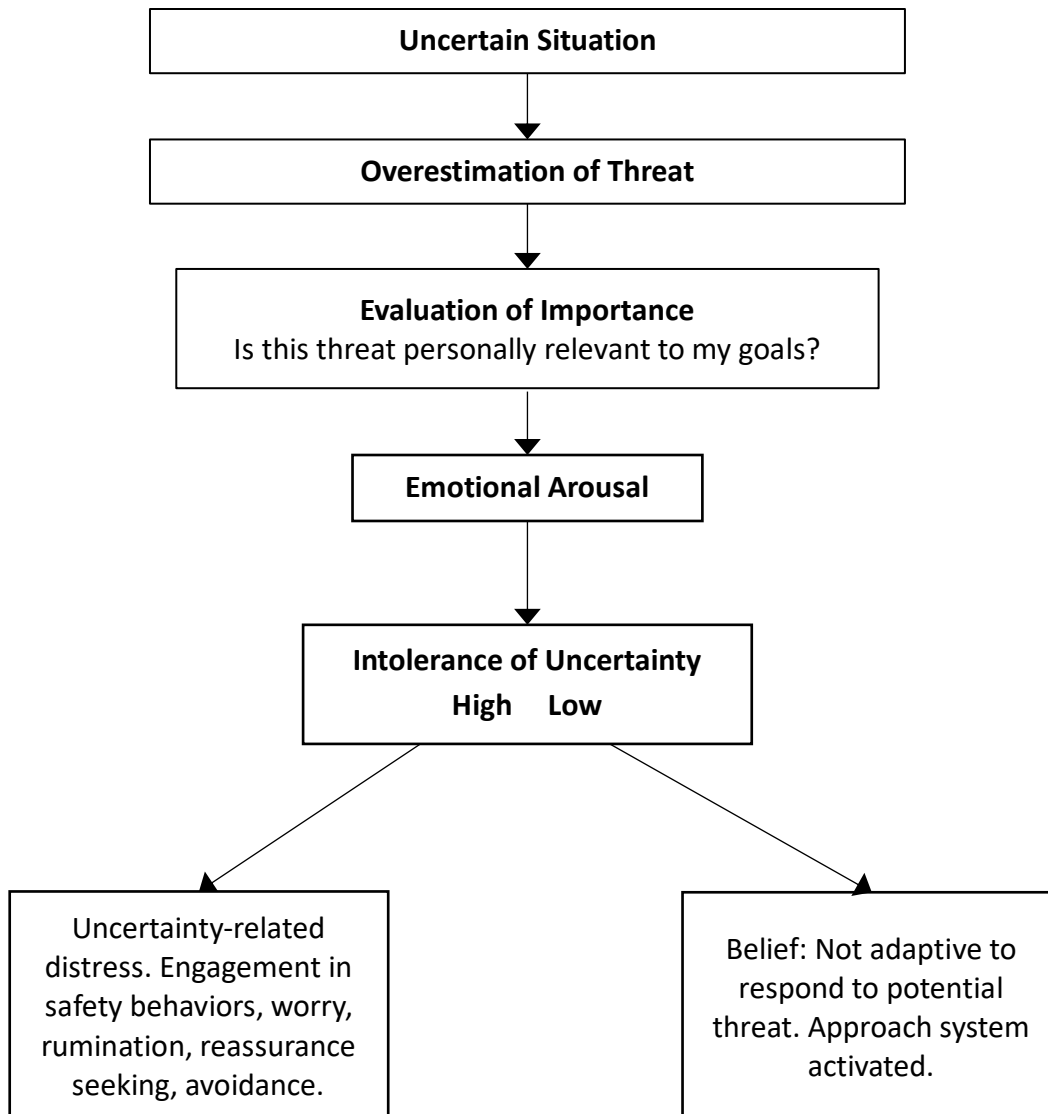
## CHAPTER 1

*“A healthy person is someone who can manage themselves in the uncertain, unpredictable world around them, where novelty and change are the norm rather than the exception.”* (Kashdan & Rottenberg, 2010, pp. 875–876)

### **1. What is intolerance of uncertainty?**

Intolerance of uncertainty (IU) is defined as “a dispositional characteristic that results from a set of negative beliefs about uncertainty and its implications and involves the tendency to react negatively on an emotional, cognitive, and behavioral level to uncertain situations and events” (Buhr & Dugas, 2009, p. 216). Difficulty in tolerating uncertainty can lead to avoidance of uncertain situations and distress about the potentially negative outcomes of uncertain situations. Given that many aspects of life are inherently uncertain, an overarching tendency to avoid or respond negatively to uncertainty can lead to the development of psychopathology (Einstein, 2014; Shapiro et al., 2020). Although the original conceptualization of IU largely explained the function of the worry commonly observed in generalized anxiety disorder (GAD) (Freeston et al., 1994), IU is considered a transdiagnostic factor for several emotional disorders (McEvoy et al., 2019; Rosser, 2019). Consistent with this view, a recent meta-analysis conducted by McEvoy et al. (2019) demonstrated effect sizes ranging from  $r = .40$ – $.57$  between IU and symptoms of eating disorders, depression, and anxiety-related disorders, including generalized anxiety disorder (GAD), social anxiety disorder, panic disorder, agoraphobia, and obsessive-compulsive disorder (OCD).

Although much remains unknown about the mechanisms that explain how IU contributes to the development of various disorders, Einstein (2014) outlined a theoretical model describing the process by which IU may confer risk for psychopathology. As depicted in Figure 1, in



**Figure 1.** Model of the role of IU in psychopathology adapted from Einstein 2014.

uncertain situations, anxious individuals tend to overestimate the likelihood or severity of potential threat (Morriss et al., 2016; Pepperdine et al., 2018). When personally relevant, perceived threats generate emotional arousal (e.g., Helzer et al., 2009; Riemann & McNally, 1995). If the individual has a high need for predictability, they may experience additional arousal associated with uncertainty (Bar-Anan et al., 2009; Nelson & Shankman, 2011). Individuals with difficulty tolerating this uncertainty arousal may initiate behaviors to reduce the associated

arousal, including active information-seeking, excessive worry, or avoidance of the situation and associated thoughts and emotions (Rosen & Knäuper, 2009; Toffolo et al., 2013; Yao et al., 2022). Individuals low in the need for predictability experience less arousal and have the cognitive capacity to recognize that the threat is not yet realized, leading to more adaptive emotional and behavioral responses in service of the individual's goals (Grupe & Nitschke, 2013; Pushkarskaya et al., 2015; Wild et al., 2014).

The model depicted in Figure 1 may be especially useful for conceptualizing the development and maintenance of OCD. OCD is characterized by unwanted, distressing, intrusive thoughts (obsessions) and attempts to reduce the anxiety associated with these thoughts through repetitive behaviors or mental rituals (compulsions). Individuals with OCD frequently overestimate threat, especially when personally relevant (Moritz & Pohl, 2009; Taylor et al., 2010). Desire for predictability also explains unique variance in OCD symptoms (McEvoy & Mahoney, 2012). Compulsions function to reduce uncertainty arousal by increasing the perception of control over future outcomes (i.e., Greco & Roger, 2003). For example, individuals with OCD may engage in excessive checking ("I can't be sure I locked the door, so I better go back and check"), reassurance-seeking ("Are you sure I didn't hit someone while I was driving?"), washing and cleaning rituals ("There may be germs on this doorknob, so I need to wipe it down repeatedly throughout the day to prevent anyone from coming into contact with a dangerous disease"), or other mental compulsions ("I'll repeat the Lord's Prayer at least one more time through, so I can make sure God heard me and will absolve me of my sin") in order to reduce the distress associated with uncertainty. Given the proposed role of IU in the development of OCD, further investigation into the development of IU, its contributions to OCD, and the ability to modify IU within the treatment of OCD is warranted. As noted by Campbell and Fiske

(1959), however, before examining the associations between a specific trait and symptoms of psychopathology, we must first have confidence in the measures of that trait, established through evidence of reliability and convergent and discriminant validity.

### **1.1. Measuring IU**

Much of the existing knowledge implicating IU in emotional disorders has been facilitated by the development of the *Intolerance of Uncertainty Scale* (IUS-27; Freeston et al., 1994). In the initial psychometric examination of the English version of the IUS-27 (Buhr & Dugas, 2002), the scale demonstrated excellent internal consistency ( $\alpha = .94$ ) and good test-retest reliability ( $r = .74$ ) over five weeks. As evidence of convergent and discriminant validity, the IUS-27 was strongly correlated with worry, anxiety, and depression ( $r_s = .55-.60$ ), with the association between worry and IU remaining significant after controlling for anxiety and depression (partial  $r = .30$ ). As evidence of known groups validity, IUS-27 scores differed significantly by diagnostic status; individuals with GAD scored significantly higher than individuals with only some symptoms of GAD, and individuals with some GAD symptoms scored higher than those who did not endorse any GAD criteria. The English version of the IUS-27 demonstrated a four-factor structure: (1) uncertainty leads to the inability to act, (2) uncertainty is stressful and upsetting, (3) unexpected events are negative and should be avoided, and (4) being uncertain about the future is unfair.

An abbreviated version of the IUS-27 has also been developed to facilitate more time efficient administration (IUS-12; Carleton et al., 2007). The IUS-12 was very highly correlated with the original IUS-27 ( $r = .96$ ) and also demonstrated excellent internal consistency ( $\alpha = .91$ ) in an initial investigation of its psychometric properties. The IUS-12 also demonstrated evidence of convergent and discriminant validity, with strong correlations with measures of anxiety,

depression, worry, and GAD symptoms ( $r_s = .57-.64$ ) and unique variance in worry and GAD symptoms accounted for by the IUS-12 after controlling for anxiety and depression. The IUS-12 has two stable factors. The first, Prospective IU, refers to the tendency toward active information-seeking to reduce uncertainty. The second, Inhibitory IU, refers to avoidance-oriented responses to uncertainty. Several studies have examined the factor structure of the IUS-12, with a bifactor model typically providing the best fit (Hale et al., 2016; Shihata et al., 2018). In a bifactor model, items load onto both a general factor (e.g., general IU) and a specific factor (e.g., either Prospective or Inhibitory IU); the general factor and specific factors are uncorrelated. Most studies using the IUS-12 only report total scores; however, there is additional utility in reporting subscale scores as well. For example, Prospective and Inhibitory IU are differentially associated with symptoms of emotional disorders (McEvoy & Mahoney, 2011, 2012), with Prospective IU uniquely associated with OCD and GAD, and Inhibitory IU uniquely associated with social anxiety disorder, panic disorder, agoraphobia, and depression.

Although the IUS-27 and IUS-12 are the most commonly used measures to assess IU, other measures have been used in the psychopathology literature, including the Perfectionism/Certainty subscale of the *Obsessive Beliefs Questionnaire* (OBQ-PC; Obsessive-Compulsive Cognitions Working Group, 2001), which also includes items relevant to perfectionism. The OBQ was designed to examine beliefs, including IU, related to OCD symptoms, while the IUS-27 and IUS-12 were originally conceptualized to measure IU associated with worry. Use of these two measures is often dependent on the population of interest. Gentes and Ruscio (2011) provide an excellent overview of these two measures and their relations with emotional disorders, concluding that correlations between symptoms of psychopathology and IU are largely consistent between the two measures. Most studies of IU in

OCD use the OBQ-PC subscale. However, of the 16 items that make up the OBQ-PC subscale, only four were conceptualized as representing IU, while the remainder relate to perfectionism (Myers et al., 2008). While perfectionism may be implicated in some OCD subtypes, nearly all symptoms of OCD are characterized by an inability to tolerate uncertainty: uncertainty regarding dirt or disease transmission in contamination-related OCD, uncertainty about potential negative outcomes in harm-related OCD and OCD focused on symmetry or exactness, uncertainty over one's own mental state, character, or morality in OCD involving unacceptable or taboo thoughts. Thus, the OBQ-PC subscale, which is heavily weighted towards perfectionism, may underestimate the significance of the link between IU and OCD symptoms. Unlike the OBQ-PC subscale, items on the IUS-12 also do not reference specific symptoms of psychopathology, such as a tendency to worry, experience somatic anxiety, or perform rituals in response to feelings of uncertainty, making the more IUS suitable for use across multiple presentations of psychopathology, including OCD.

## **1.2. Examining IU within a larger personality framework**

A number of conceptual approaches have described IU as a specific component of a broader personality trait. For example, IU in adulthood may originate from a behaviorally inhibited temperament in childhood. Within the developmental psychopathology literature, behavioral inhibition is the tendency to respond fearfully or to withdraw from novel or unfamiliar situations, objects, or people (Fox et al., 2005). Two longitudinal studies have found a significant relation between behavioral inhibition observed in early childhood and later self-reported IU (Hawes et al., 2021; Zdebik et al., 2018). Another view suggests that IU may be a specific component of neuroticism, the tendency to experience negative emotions, especially in response to stressors (Barlow et al., 2014). This view is supported by research that finds a direct

effect of neuroticism on IU as well as specific contributions of IU on anxiety symptoms in both clinical and non-clinical samples (McEvoy & Mahoney, 2012; Sexton et al., 2003), and a meta-analytic study that found that IU, among other cognitive vulnerabilities for emotional disorders, had the strongest factor loading onto a core neuroticism factor (Hong & Cheung, 2015). Carleton (2016a) considers IU to be an indicator of a fundamental “fear of the unknown” underlying anxiety and neuroticism, with fear of the unknown defined as “an individual’s propensity to experience fear caused by the perceived absence of information at any level of consciousness or point of processing” (p. 31). Finally, IU has also been discussed as a lower-order component of distress tolerance, or the perceived capacity to withstand aversive states (Leyro et al., 2010; Zvolensky et al., 2010). Other lower-order forms of distress (in)tolerance include intolerance of ambiguity, frustration, physical sensations, and negative emotional states more generally.

Importantly, these conceptual models all assume that IU is a relatively stable trait that arises early in life. This claim is an especially important component of understanding whether IU can be considered a cognitive vulnerability for a given emotional disorder. Koerner and Dugas (2008) outline four criteria to consider a construct as a cognitive vulnerability for psychopathology. First, when a set of beliefs (cognitions) is present, it must heighten the risk that psychopathology will develop. Second, the cognitive vulnerability must be a causal risk factor, influencing the etiology of psychopathology either directly or indirectly. Third, the cognitive vulnerability must be stable and trait-like. Finally, the cognitive vulnerability must be malleable to intervention.

### **1.3. IU as a cognitive vulnerability for OCD**

Although there is a significant body of literature suggesting a robust relation between IU and OCD symptoms, such information alone is not sufficient to determine if IU can be



considered a cognitive vulnerability for OCD. Some researchers have proposed that IU is at the core of almost all presentations of OCD (e.g., Grayson, 2010). Despite this assertion, limited research is available examining IU as a vulnerability factor for the development and maintenance of OCD or the role of increasing tolerance of uncertainty as a *mechanism* for effective OCD treatment. In an effort to move the field forward, the next sections will examine evidence for each of Koerner and Dugas's (2008) criteria for determining if IU may be conceptualized as a cognitive vulnerability for OCD. This includes the following:

- (1) Evidence for a robust association between IU and OCD symptoms,
- (2) Evidence for IU as a causal risk factor that influences the development of OCD directly or indirectly,
- (3) Evidence that IU is stable and trait-like, and
- (4) Evidence that IU is malleable to intervention.

### **1.3.1. Evidence for a relation between IU and OCD symptoms**

Tolin and colleagues (2003) proposed that IU may be a central component of the etiology of OCD. Specifically, Tolin and colleagues found that IU was elevated in OCD patients with checking compulsions compared to nonanxious controls ( $\eta_p^2 = .28$ ) and hypothesized that this relation was due to the role of heightened pathological doubt, an obsessional lack of confidence in one's memory of performing an action such as locking a door or turning off the stove. Since then, a number of studies have found evidence of an association between IU and OCD symptoms in both clinical and non-clinical samples. To summarize such findings, two meta-analyses examined the relation between IU and OCD symptoms throughout the empirical literature. Across 33 studies, Gentes and Ruscio (2011) found a mean correlation between .42 and .50 between IU and OCD, depending on the measure used. Similarly, McEvoy and colleagues (2019)

observed moderate associations between IU and OCD (mean  $r = .42$ ) across 69 studies.

Together, these two meta-analyses suggest that IU is moderately to strongly associated with OCD symptoms. Although meta-analysis is a useful tool to quantitatively evaluate a body of research in a given area (Thacker, 1988), the two meta-analyses described above included only self-report measures of IU and OCD symptoms, and only provide cross-sectional data. Thus, additional research examining multimodal and prospective relations between IU and OCD symptoms is reviewed below.

In an examination of IU and obsessive-compulsive symptoms using both self-report measures and behavioral tasks, Sarawgi, Oglesby, and Cogle (2013) administered measures of IU and obsessive-compulsive symptoms and tasks related to either ordering and arranging, checking, washing, contamination avoidance, or neutralization to a large sample of undergraduate students. The findings showed that IU was significantly related to each self-report measure of the obsessive-compulsive symptom domains,  $r_s = .42-.58$ . Further, IU predicted performance on all *in vivo* task domains except neutralization/harm, including urge to check in a stove checking task ( $r = .22$ ), avoidance ( $r = .22$ ), urge to wash ( $r = .42$ ), and washing duration ( $r = .30$ ) in a contamination task, and urge to arrange ( $r = .30$ ) and arranging duration ( $r = .17$ ) in an ordering and arranging task.

Fewer studies have examined the prospective relation between IU and OCD symptoms. In a retrospective study, individuals reported that IU typically preceded the onset of full OCD by approximately 3 years (Coles et al., 2012). Abramowitz and colleagues (2006) found that multiple domains of obsessive beliefs, including IU, predicted postpartum OCD symptoms in expectant parents,  $\beta = 0.31$ , but did not examine the relative contributions of IU compared to other common obsessive beliefs, such as overestimates of threat and responsibility for harm or

the importance and control of intrusive thoughts. In a prospective study, Pozza and colleagues (2019) found that, after controlling for depressive symptoms, endorsement of perfectionism and high IU beliefs predicted the severity of OCD symptoms one year later in both children and adolescents,  $\beta = 0.17$ , accounting for 7% of the variance in OCD symptom severity. Although these studies provide some evidence suggesting a prospective association between IU and OCD symptoms, the existing literature is relatively limited, as none of the existing prospective studies used a robust measure of IU (such as the IUS) to examine changes in IU over time, potentially conflating IU and perfectionism or other obsessive beliefs.

### **1.3.2. Evidence for IU as a causal risk factor in the etiology of OCD**

Kraemer and colleagues (1997) note that a *causal risk factor* must meet the following criteria: (1) temporal precedence, such that the risk factor must precede the hypothesized outcome; (2) the ability to be manipulated; and (3) when manipulated, the risk factor must change the risk of the outcome. Compared to correlational studies, fewer studies have examined IU as a causal risk factor for OCD.

#### **1.3.2.1. Temporal precedence**

Evidence that IU temporally precedes OCD symptoms is mixed. In a study of cognitive therapy for OCD, individuals who experienced above-average decreases in IU during treatment had significantly greater decreases in obsessions and compulsions compared to individuals who experienced below-average changes in IU,  $d = 0.77$ , with changes in IU preceding changes in OCD symptoms (Wilhelm et al., 2015). Next, in a study of exposure and response prevention for OCD, changes in IU did not precede changes in OCD symptoms (Su et al., 2016). Of note, however, both studies used the OBQ-PC subscale, meaning that these findings do not isolate changes in IU from changes in perfectionism. Using the IUS-12, Shapiro and colleagues (2020)

did not find evidence of the temporal precedence of IU over a one-year period, as baseline IU did not predict incidence of new internalizing diagnoses or changes in symptoms of emotional disorders, including OCD, among undergraduates with elevated IU (1.5 SD above the sample mean). In addition, although changes in IU over one year were associated with changes in symptoms of other emotional disorders, changes in IU did not predict OCD symptoms. However, there was a high rate of attrition in this study (from  $N = 138$  at baseline to  $N = 42$  at one-year follow-up), and the use of a restricted range of IU scores may have weakened the correlation between IU and OCD symptoms.

### **1.3.2.2 Experimental induction of IU**

A number of studies have examined the effects of inducing IU within an experimental context. Ladouceur, Gosselin, and Dugas (2000) induced IU by telling participants playing a roulette game that they had either a worse chance of winning compared to participants in a previous study (higher IU) or a better chance compared to past participants (lower IU). The stakes of the study were raised in that participants were told that \$100 would be donated to a fictitious foundation if and only if they drew even or won the game. Participants in the increased IU group reported higher levels of worry about the fictional foundation compared to participants in the decreased IU group,  $d = 1.70$ . However, the experimental induction of IU in this study, as well as the perceived consequence, was not personally relevant to participants; inductions of IU may be more ecologically valid when participants are personally invested in the outcome (Mosca et al., 2016). Next, Grenier and Ladouceur (2004) asked participants to visualize and pretend they had ingested a medication that caused an unpredictable effect, then read a script that either emphasized high IU (e.g., “It’s frustrating that I don’t know what’s going to happen to me. It’s out of my control”) or low IU (e.g., “I have to live one day at a time. No one is able to plan and

organize everything in advance”). Participants low in state anxiety at baseline reported higher worry after the increased IU manipulation,  $d = 1.30$ , and lower worry after the decreased IU manipulation,  $d = -0.84$ , suggesting that the effects of experimentally manipulated IU may be limited to individuals in a non-anxious state. While this may suggest a potential ceiling effect of IU manipulation, it is also possible that individuals who are anxious at baseline may already feel somewhat uncertain, with additional uncertainty not contributing to their anxiety. In other words, individuals may have a threshold for tolerating uncertainty, rather than experiencing a dose-response effect of uncertainty on anxiety.

Mosca and colleagues (2016) expanded upon the work of Grenier and Ladouceur (2004) by asking participants to select a personally relevant potential negative event before reading a list of uncertainty-related beliefs, without imagining that any feelings that arose were attributable to an imagined medication. Participants in the increased IU condition reported greater IU, worry, and negative affect than those in the decreased IU condition,  $\eta_p^2 = .09-.13$ , while individuals in the decreased IU condition did not differ from those in a control condition who did not read statements about uncertainty beliefs. Thus, it may be easier to experimentally increase IU than it is to decrease IU. In another study that manipulated individual beliefs about uncertainty using a personally relevant event, participants exposed to negative beliefs about uncertainty reported a greater likelihood of feared consequences to personal worries,  $\eta_p^2 = .08$  (Deschenes et al., 2010), suggesting a potential cognitive bias as a consequence of experimentally induced IU, in addition to its purported effects on anxiety and worry. Finally, Rosen and Knäuper (2009) provided individuals with false feedback regarding whether they demonstrated low or high IU after the administration of a doctored IU questionnaire, which increased or decreased the probability that an item would be endorsed. Individuals who received high IU feedback in a high situational

uncertainty condition reported increased worry,  $d = 1.05$ , and sought more information about a fictional sexually transmitted infection,  $d = 0.80$ , compared to individuals who received low IU feedback in a low situational uncertainty condition, demonstrating a potential behavioral consequence of increased IU.

### **1.3.2.2. Effects of experimentally induced IU on OCD symptoms**

Though the existing evidence suggests that IU can be induced within an experimental context, only two studies have examined the effects of induced IU on obsessive-compulsive symptoms. First, a recent study expanded upon Rosen and Knäuper's experimental manipulation of IU (Geok et al., 2022). In addition to providing undergraduate participants with false feedback regarding their ability to tolerate IU, participants were asked to keep a daily diary of situations where they tolerated uncertainty well (low IU condition) or poorly (high IU condition) over the course of a week. Geok and colleagues found a significant interaction between IU condition and baseline OCD symptom severity,  $\beta = 0.64$ , such that individuals with high baseline OCD symptoms demonstrated a greater decrease in OCD symptoms after the low IU induction compared to individuals in the high IU induction, suggesting a beneficial effect of low IU on OCD symptoms. Next, in an experimental study, undergraduate students provided with false feedback suggesting high IU reported significantly higher threat perceptions about a personally relevant intrusive thought compared to individuals who were told they were more tolerant of uncertainty,  $d = 0.73$ . However, this manipulation did not affect performance in a checking task (Faleer et al., 2017). Thus, while experimentally induced IU may impact OCD symptoms, the effects may be limited to self-reported OCD symptoms or specific OCD symptom domains, and additional research is needed to determine if these effects are as replicable as the effects of experimentally induced IU on worry.

### 1.3.3. Evidence that IU is stable and trait-like

Although the extent to which IU is trait-like has not yet been thoroughly investigated, examinations of the test-retest reliability of its most commonly used measures may provide some insights into the stability of the construct. For example, Buhr and Dugas (2002) reported a five-week test-retest reliability coefficient of  $r = .74$  for the original IUS-27. Carleton and colleagues (2014) also found good test-retest reliability over two weeks for the abbreviated IUS-12,  $r = .77-.83$ . Though test-retest reliability is an important aspect of measurement stability, both studies only examined reliability over a relatively short period of a few weeks. In addition, an examination of the stability of the measures of IU is an incomplete test of whether the construct is stable and trait-like, as such studies typically only report correlations on a measure at two timepoints. In addition, test-retest coefficients underestimate the true stability of a given trait as they are attenuated by measurement error (Costa & McRae, 1988); models that can account for measurement error may provide a more accurate examination of stability (Kenny & Zautra, 1995; Cole et al., 2005).

One study did examine the stability of IU over a longer period, during the context of acute stress. IU was relatively stable among parents three months after their child's treatment for cancer, with a case-by-case analysis revealing no changes in IU over three months (Vander Haegen & Etienne, 2018). Thus, even the stress associated with a child's cancer may not have an impact on IU, lending support to considering IU as a stable personality trait. However, information about parental IU before the child's cancer diagnosis was not available, providing only a brief snapshot of the stability of IU in response to stress without baseline information.

There is some evidence for the stability of IU within child and adolescent samples. In children, IU demonstrated moderate stability (stability coefficient = .32) from ages 8 to 11 (Hong

et al., 2017), providing some evidence that IU remains a stable trait through at least early adolescence. Another longitudinal study found that mothers' ratings of children's shyness ( $B = 0.84$ ) and observed dysphoria during a laboratory behavioral task ( $B = 0.87$ ) at age 3 predicted higher self-reported IU in early to mid-adolescence, suggesting a potential relationship between observed negative emotionality in young children and later IU (Hawes et al., 2021). Similarly, behaviorally inhibition ( $\beta = .24$ ) and insecure attachment ( $\beta = .32$ ) at age 6 predicted high IU above and beyond neuroticism in young adults followed for 15 years (Zdebik et al., 2018). Behavioral inhibition and IU are highly related concepts, and behavioral inhibition is a stable characteristic of childhood temperament with strong predictive value for the development of anxiety-related disorders in childhood, adolescence, and adulthood (Fox et al., 2005; Kagan & Moss, 1962). Given that IU likely requires an understanding of the unknown and future potential threats, it may be the product of behavioral inhibition, which describes behavior around novel or unfamiliar situations or people. It is important to note, however, that measures of IU have been developed for use in youth ages 7 to 17 (Comer et al., 2009), and a parental report adaptation has been used to assess IU in children ages 3 to 10 (Sanchez et al., 2017). At this time, no studies have concurrently examined correlations between parent-report measures of IU in young children and children's behavior in established behavioral inhibition paradigms.

#### **1.3.4. Evidence that IU is malleable during treatment**

Modifying client beliefs about their ability to manage uncertainty may contribute to better OCD treatment outcomes (Grayson, 2010; Jacoby & Abramowitz, 2017). A number of studies have examined changes in IU within a treatment context. In most studies, IU is examined as one potential treatment outcome, often as a correlate of symptom improvement. In a few studies, IU



is designated as a specific treatment target, with intervention designed to improve clients' ability to tolerate uncertainty.

#### **1.3.4.1. Effects of OCD treatment on IU**

Several studies have found improvements in IU over the course of treatment for OCD. For example, Belloch and colleagues (2010) reported that IU decreased significantly after 18 sessions of cognitive therapy for OCD, though IU decreased more for individuals with autogenous (ego-dystonic; personally unacceptable and unrealistic, threatening thoughts) obsessions,  $d = 2.32$ , compared to individuals with reactive (realistic, aversive thoughts with threatening consequences) obsessions,  $d = 0.95$ . In both groups, changes in IU were maintained at one-year follow-up. Su and colleagues (2016) reported that perfectionism and IU decreased after 8 weeks of twice-weekly exposure and response prevention for OCD,  $d = 0.60$ , and continued to decrease from baseline to 24-week follow-up,  $d = 0.91$ . Although most studies of OCD that demonstrate effects on IU focus on cognitive-behavioral treatments, Mathur and colleagues (2021) found that both perfectionism and IU significantly decreased among OCD patients undergoing mindfulness-based therapy (Cohen's  $f^2 = .45$ ).

The evidence demonstrating that IU decreases after OCD treatment provides an initial test of the malleability of IU in response to treatment. However, fewer studies report an association between changes in IU and changes in OCD symptoms during treatment. In the first such study, Overton and Menzies (2005) assessed idiosyncratic cognitive beliefs among individuals with primary checking-type OCD. To measure IU, individuals rated distress about uncertainty related to their most common checking compulsion, such as "How distressing is it for you if you cannot be certain that your front door is locked?" After 12 sessions of exposure and response prevention, individuals demonstrated a large change in IU related to their target

compulsion,  $d = 2.43$ , and changes in uncertainty beliefs were strongly correlated ( $r = .75$ ) with changes in OCD symptoms. While this study provides compelling evidence that changes in IU and OCD symptoms are strongly linked during treatment for compulsive checking, only a specific form of IU was assessed which limits broader inferences.

Using the OBQ-PC subscale, Wilhelm and colleagues (2015) found that individuals who reported above-average decreases in perfectionism and IU during cognitive therapy for OCD reported significantly greater decreases in obsessions and compulsions compared to individuals who reported below-average changes in perfectionism and IU during treatment,  $d = 0.77$ . Similarly, after 16 sessions of cognitive-behavioral therapy for OCD, Kyrios and colleagues (2015) observed significant changes in perfectionism and IU (Hedges's  $g = 1.10$ ), which were maintained at 6-month follow-up. In addition, greater changes in perfectionism and IU predicted better post-treatment OCD symptoms,  $\beta = -.57$ . Pinciotti, Riemann, and Wetterneck (2020) found that improvements in IU partially explained symptom improvement ( $\beta = .06$ ) among patients undergoing residential treatment for OCD, suggesting that changes in IU are associated with symptom changes in individuals with the most severe OCD symptoms. Most recently, Donegan and colleagues (2022) found that changes in IU significantly mediated reductions in anxiety among peripartum women who received group cognitive-behavioral therapy. The majority of participants in the study (86.7%) met criteria for primary GAD, with 36% meeting diagnostic criteria for OCD or a secondary anxiety-related disorder. Given that the intervention addressed excessive use of safety behaviors that is commonly observed in OCD (i.e., reassurance seeking, checking), this evidence supports the importance of targeting IU in the treatment of OCD.

#### **1.3.4.2. Direct interventions targeting IU**

A few studies have attempted to directly reduce IU during treatment. Ladouceur, Dugas and colleagues (2000) designed a 16-week therapeutic treatment specifically targeting IU for individuals with GAD. Treatment elements included awareness training, cognitive modification of maladaptive beliefs about worry, imaginal exposure, and problem-orientation training; throughout all treatment components, the ability to tolerate uncertainty was emphasized. Individuals in the treatment condition demonstrated substantial reductions in IU from pre-treatment to post-treatment,  $d = 1.83$ , and gains were maintained through 12-month follow-up,  $d = -0.12$ . Thus, IU demonstrated substantial malleability in response to the intervention within a GAD sample.

Whittal and McLean (2002) describe a group CBT intervention for OCD that includes challenging IU. Specifically, patients are asked to normalize uncertainty by surveying 10 friends or co-workers about whether they remember locking the door the last time they left the house, and how certain they were that the door was locked. In a recent study, a group-based treatment designed to reduce IU (“Making Friends with Uncertainty”; Mofrad et al., 2020) was piloted among 24 individuals with mixed emotional disorders, including OCD, GAD, and depression. This intervention included psychoeducation about uncertainty, encouragement to experiment with uncertainty in low and higher stakes situations, and discussion aimed to help participants apply this learning to threatening scenarios. Overall, 45% of participants showed reliable change in IU, and 80% demonstrated reliable change in anxiety or depression. Although individuals with OCD symptoms were included in the group, this study did not examine the impact of the intervention on OCD symptoms.

Finally, in a single-session intervention, Oglesby and colleagues (2017) implemented a cognitive bias modification intervention for individuals who demonstrated a high IU interpretation bias, such that they were more likely to endorse a negative interpretation (e.g., “I have a terrible disease”) as related to an ambiguous prime (e.g., “doctor called”) compared to a more neutral interpretation (e.g., “appointment reminder”). During the intervention, individuals were provided with feedback suggesting that neutral interpretations were “correct,” while negative interpretations were “incorrect.” Compared to individuals in the control condition (a phrase-pairing task not relevant to IU), there was a significantly greater decrease in IU among individuals who received the cognitive bias modification intervention,  $B = 5.10$ . As evidence of a specific mechanism, there was a significant indirect effect of the IU cognitive bias modification condition on change in IU from baseline to one-month follow-up through change in IU interpretation bias. Thus, even a brief, single-session intervention can reduce a cognitive bias associated with IU, which reduced self-reported IU. However, no effect of this intervention on OCD symptoms was examined.

#### **1.4. Redux: Is IU a cognitive vulnerability for OCD?**

The existing evidence suggests a strong possibility that IU is a cognitive vulnerability for OCD. First, there is evidence of a strong association between IU and OCD symptoms in both cross-sectional and prospective studies. Second, the evidence suggests that IU may be a causal risk factor for OCD, though the evidence is more mixed. Although one study suggests that changes in IU temporally precede changes in OCD symptoms during treatment (Wilhelm et al., 2015), two others did not find evidence of temporal precedence (Shapiro et al., 2020; Su et al., 2016). However, it may be difficult to select the appropriate time frame during which high IU is evident before the development of OCD symptoms, and most studies are not designed for this

kind of longitudinal examination. Next, across a number of experimental studies, IU has been demonstrated to be manipulable, though the duration of such changes in IU is unclear. Two experimental studies find evidence for an effect of experimentally inducing IU on OCD symptoms (Faleer et al., 2017; Geok et al., 2022), though this effect may only be evident in self-reported changes in OCD symptoms and not robust during behavioral tasks and may be specific to some domains of OCD symptoms and not others.

Third, there is variable evidence that IU is stable and trait-like, with existing studies examining stability for short-term investigations (weeks) as well as within the context of much longer studies of childhood temperament. However, no studies to date have attempted to isolate the trait and state components of IU, as has been identified for other personality constructs, such as anxiety (Cattell, 1966; Eysenck, 1983). Fourth, there is significant evidence that IU improves during treatment for OCD and is associated with change in OCD symptoms during treatment. The magnitude of change in IU during treatment varies, with effect sizes ranging from moderate to vary large depending on the type of intervention and whether the target is general IU or the ability to tolerate an idiosyncratic uncertainty. Finally, while a few interventions targeting IU specifically have been tested, none of these studies have specifically examined the association between changes in IU and OCD symptoms.

### **1.5. Next steps in IU and its association with OCD**

In the following chapter, I present an analysis of the time-varying (state) and time-invariant (trait) aspects of IU within a community sample in order to build upon the existing literature suggesting that IU is a stable trait that is associated with OCD symptoms. Next, in Chapter 3, I will highlight that increasing tolerance of uncertainty is an important approach in cognitive-behavioral approaches to OCD treatment, and that variable exposure is one method by

which increased tolerance of uncertainty might be achieved. An original empirical study examining changes in uncertainty during a variable exposure intervention for individuals with symptoms of contamination-focused OCD, will be reported. This study contributes to the literature on the malleability of perceptions of uncertainty and their association with OCD symptoms. In the final chapter, I review the contributions of this research to the conceptualization of IU as a cognitive vulnerability for OCD along with potential implications for the role of tolerating uncertainty as a mechanism in effective OCD treatment.

## CHAPTER 2

### **2. Is IU a Stable Trait? An Analysis of the Time-Varying (State) and Time-Invariant (Trait) Aspects of IU and Its Association with Obsessive-Compulsive Symptoms**

#### **2.1. Introduction**

A central question in conceptualizing the link between IU and OCD is the extent to which IU can be considered a stable trait that predicts obsessive-compulsive symptoms. Such an approach may be informed by the trait-state distinction that can be made using a longitudinal approach that utilizes repeated measures. This model suggests that personality traits consist of two separate components: time-invariant (TI) traits and time-varying (TV) states (Cattell, 1966; Eysenck, 1983). Extensions of the model contend that personality characteristics may consist of both trait and state components (Hertzog & Nesselroade, 1987) or even a “continuum of traitness” (Kenny & Zautra, 2001). According to this view, the TV component of IU may have temporal stability to the extent that state-specific causes are relatively consistent over time. As the time interval increases, the stability of these causes decreases. In contrast, the TI component of IU is completely stable over time, showing no degradation. The longitudinal structure of the IUS-12 may therefore consist of (a) a completely stable, TI component and (b) a situational, TV component. If the causes of IU remain completely consistent over certain periods of development, the IUS-12 will be entirely TI. However, if some causes of IU vary across time, at least part of the IUS-12 will be TV. In addition, prior research suggests that cognitive therapy may be less effective for patients whose depression is attributed to more stable, trait negative affect, rather than more malleable state-like processes (Vittengl et al., 2014). Thus, understanding the trait-state dimensions of the IUS-12 may have important implications for understanding the effects of interventions on IU; if measures of IU demonstrate changes in both

state and trait variance after treatment, this may be evidence for a specific intervention effect rather than changes due to incidental state fluctuations (Roberts et al., 2017).

IU likely consists of both TV and TI components: the TV component of IU reflects situation-specific responses characterized by behavioral avoidance of uncertainty or certainty-seeking, negative judgments about the situation, and a negative emotional reaction. In contrast, the TI component reflects a relatively stable tendency to respond negatively to uncertainty and ambiguity. No study to date has examined the extent to which the longitudinal structure of any measure of IU reflects TI versus TV factors. Accordingly, the present study examines the TI and TV components of the IUS-12 by applying Cole et al.'s (2005) latent trait-state model to data from a sample of community adults in a 6-wave, 5-month longitudinal design.

While IUS-12 total scores are frequently used, research supports a bifactor model of the IUS-12 over unidimensional models (Hale et al., 2016; Shihata et al., 2018), with a strong general IU factor as well as Prospective and Inhibitory factors. Examination of the Prospective and Inhibitory components of the IUS-12 suggests that the two factors may be differentially related to psychopathology. For example, Prospective IU is associated with worry and OCD symptoms, while Inhibitory IU is more strongly associated with panic disorder, agoraphobia, social anxiety, and depression (McEvoy & Mahoney, 2011). Given potential differences between Prospective and Inhibitory IU and their differential relations with symptoms of psychopathology, we modeled both factors separately<sup>1</sup>. We predicted that the longitudinal structure of both IU factors would be characterized by significant TI and TV factors, with the TI factor accounting for

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<sup>1</sup> In the present sample, a confirmatory factor analysis of the IUS-12 suggested that a unidimensional solution demonstrated poor fit, CFI = .77, TLI = .72, RMSEA = .17. Taken together with potential differential associations with different forms of psychopathology, we elected to model both lower-order factors of IU separately. Results do not differ significantly using a single IU factor in the models (Appendix Tables 1, 2, and 3).



a greater portion of the variance. The present study also examined the association between the TI and TV components of the IUS-12 and OCD symptoms. We predicted that the TI component of the IUS-12 would be more strongly associated with OCD symptoms compared to the TV component. This prediction is consistent with research demonstrating that the TI components of personality risk factors are uniquely associated with anxiety and related disorders (Naragon-Gainey et al., 2013; Olatunji et al., 2020). To determine the specificity of these associations, the present study also examined TI and TV components of the IUS-12 as predictors of depressive symptoms alone and with depressive symptoms as a covariate in models predicting OCD symptoms. Given that many compulsions are active attempts to achieve certainty (i.e., checking, reassurance-seeking), it was predicted that the TI component of Prospective IU may be more strongly associated with OCD symptoms than Inhibitory IU.

## **2.2. Method**

### **2.2.1. Participants**

A total of 1280 participants (87% female, 13% male, 0.2% declined to respond) completed at least one survey as part of the present study. The mean age of the participants was 42.6 years ( $SD = 13.6$  years), ranging from 18 to 71 years. The racial/ethnic composition was as follows: White/Caucasian ( $n = 1118$ ; 87.3%), Black/African American ( $n = 49$ ; 3.8%), Asian/Asian American ( $n = 31$ ; 2.4%), Hispanic/Latino ( $n = 47$ ; 3.7%), biracial or multiracial ( $n = 17$ ; 1.3%), Native American/American Indian ( $n = 3$ ; 0.2%), Other ( $n = 8$ ; 0.6%), declined to respond ( $n = 7$ ; 0.5%). At baseline, 20.7% of participants reported being in psychotherapy, and 36.3% reported taking medication for psychiatric or psychological problems. Of note, the present sample reported high mental health service utilization compared to adults in the 2019 National

Health Interview Survey, in which 15.8% took prescription medication for mental health and 9.5% received counseling or therapy in the last 12 months (Terlizzi & Zablotsky, 2020).

### **2.2.2. Procedure**

Participants were recruited through ResearchMatch as part of a larger study on the relation between sleep, stress, and repetitive negative thoughts<sup>2</sup>. ResearchMatch is a national health volunteer registry supported by the U.S. National Institutes of Health as part of the Clinical Translational Science Award program. ResearchMatch has a large population of volunteers who have consented to be contacted by researchers about health studies. Participants received a link to complete an online battery of questionnaires and were informed that they would be able to enter a drawing for a \$25 gift card after completing each survey, for a total of six opportunities to win one of six \$25 gift cards. Participants received a total of six identical survey batteries over a five-month period, approximately one month apart. Observations were coded as missing if a participant did not complete the survey within seven days after receiving the survey invitation. To collect and manage the data, we used Research Electronic Data Capture (REDCap; Harris et al., 2009). REDCap is a secure, web-based application designed to support data capture for research studies and is supported by UL1 TR000445 from NCATS/NIH. The university Institutional Review Board approved this study and all procedures, and informed consent was obtained from all participants.

### **2.2.3. Measures**

The *Intolerance of Uncertainty Scale-12* (IUS-12; Carleton et al., 2007) is a 12-item self-report scale of the perceived ability to tolerate the possibility of unpredictable negative events. Items on the IUS-12 are rated on a Likert scale from 1 (*not at all characteristic of me*) to 5

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<sup>2</sup> All waves of the study were completed before the COVID-19 pandemic.

(entirely characteristic of me), and higher scores indicate greater IU. The IUS-12 consists of two subscales, Prospective IU and Inhibitory IU. In the present sample, the Inhibitory IU subscale demonstrated excellent internal consistency across all waves ( $\alpha$ s = .91–.92), and the Prospective IU subscale demonstrated good internal consistency ( $\alpha$ s = .85–.89). Across the six waves of the study, the correlation between Prospective and Inhibitory IU ranged from  $r = .58$ –.63.

The *Obsessive-Compulsive Inventory Revised* (OCI-R; Foa et al., 2002) is an 18-item self-report measure of OC symptoms experienced in the past month. The OCI-R consists of six subscales measuring specific categories of OC and related symptoms (washing, checking, ordering, neutralizing, obsessing, hoarding). In the present study, the hoarding subscale was excluded, as hoarding is now considered distinct from OCD (Wheaton et al., 2011). Though the sample was unselected, 21.9% of participants scored at or above 4 on the OCI-R Obsessing subscale at baseline, the cutoff score used to differentiate OCD patients from community controls without a psychiatric diagnosis (Foa et al., 2002). This rate of significant OCD symptoms is higher than the estimated prevalence of clinical OCD within the general population (1–2%; American Psychiatric Association, 2013). The OCI-R demonstrated good to excellent internal consistency ( $\alpha$ s = .89–.91) at all waves in the present sample.

The *Depression Anxiety Stress Scales, 21-item version* (DASS-21; Antony et al., 1998; Lovibond & Lovibond, 1993) is a 21-item self-report measure of depression, physical arousal, and psychological tension and agitation. Only the seven-item Depression subscale was used in the present study. The DASS-Depression subscale demonstrated excellent internal consistency ( $\alpha$ s = .91–.94) across all six waves of the study. In general, the present sample was low in depression ( $M = 5.02$  at Time 1), and only 5.9% of the sample scored at or above the mean of the clinically depressed group ( $M = 14.98$ ) found in Antony et al. (1998). For comparison, in a recent

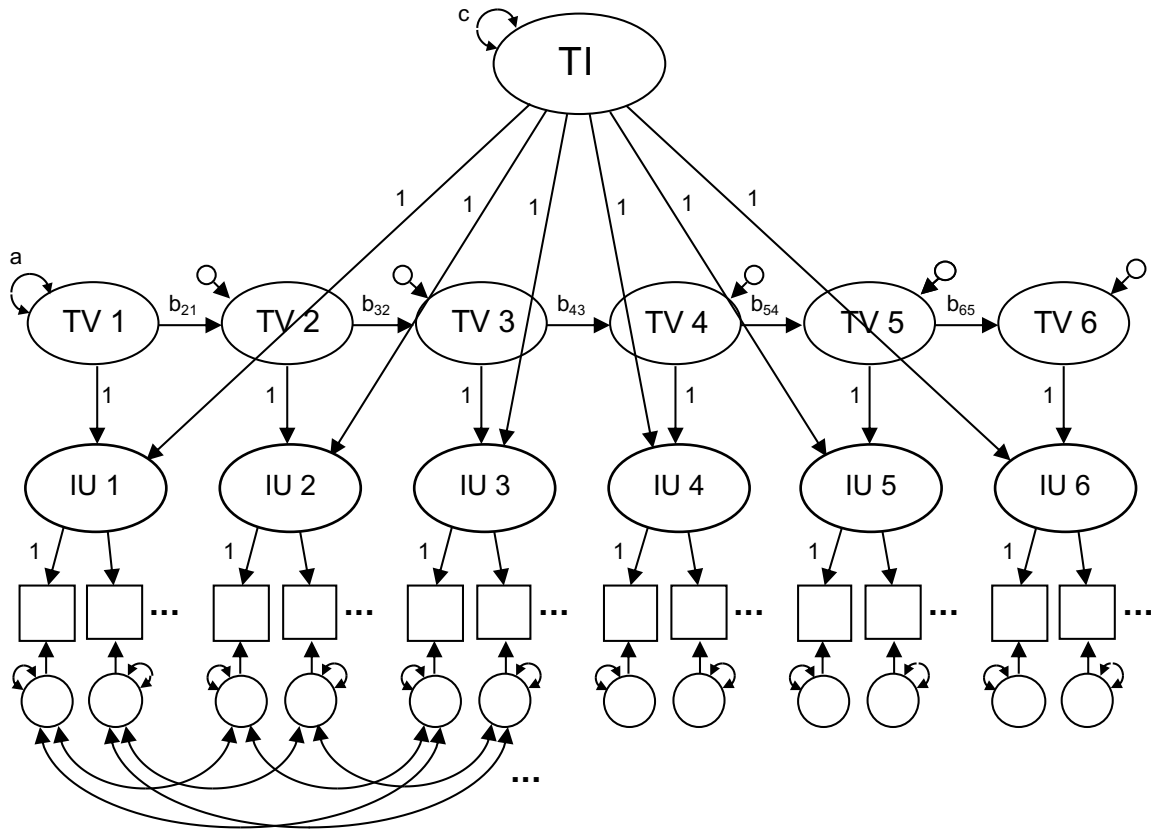
large, nationally representative survey, 10.4% of adults met criteria for Major Depressive Disorder within a 12-month period (Hasin et al., 2018). As evidence of discriminant validity, the OCI-R and DASS-Depression scales were only moderately correlated at each wave ( $r_s = .25-.41$ ), suggesting that each measure captured different symptoms within the present sample.

#### **2.2.4. Data Analysis**

All analyses were conducted using Mplus Version 8 (Muthén & Muthén, 2017). First, measurement models for Prospective and Inhibitory IU were examined. Each model consisted of six oblique latent variables (one for each wave) representing the target construct at each of the six waves. To account for shared method variance across waves, within-indicator, cross-wave residual covariances were free (LaGrange & Cole, 2008), similar to Kenny and Kashy's (1992) recommendations for multitrait-multimethod models. The same measurement models were embedded in the TI-TV models for Prospective and Inhibitory IU.

TI-TV analyses were conducted for each dimension of the IUS-12; a generic path diagram for these models is presented in Figure 2. Each model consisted of six latent variables, one for each wave of the targeted latent construct (Prospective or Inhibitory IU). At each wave, the variances of these latent IU variables were partitioned into a TV factor and an orthogonal TI factor, which sum to form the composite IU factor. Autoregressive paths allow for a degree of cross-wave stability for the TV factor. The Prospective IU model contained seven indicators, the seven items that make up the Prospective subscale of the IUS-12, and the Inhibitory IU model contains five indicators, the five items that make up the Inhibitory subscale of the IUS-12.

To identify the measurement and TI-TV models, the loading for the first indicator variable was fixed to one for each latent variable. Across waves, all loadings and stability coefficients were constrained to be equal to their counterparts. Factor loadings connecting latent

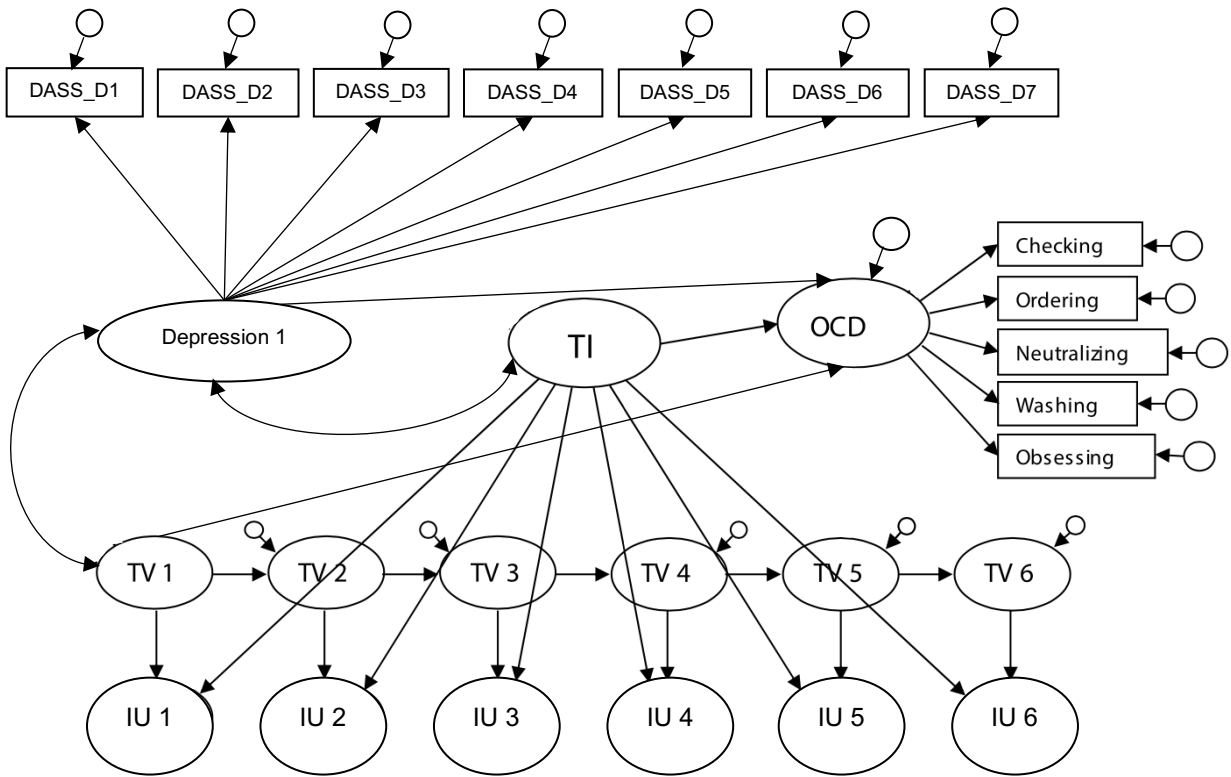


*Note.* The number of indicator variables varies from model to model. To avoid visual clutter, not all correlations between residuals for repeated items are depicted. IU = intolerance of uncertainty, TI = time-invariant factor; TV = time-varying factor.

**Figure 2.** Generic path diagram for the TI-TV model from Knowles et al., 2022.

IU variables to TI and TV factors were also set to one, constraining them to be equal across time. Fixing these factor loadings to one identifies the model, allowing the TV and TI variances ( $a$  and  $c$  in Figure 2, respectively) to be estimated. These constraints align with the theory that psychological variables consist of constant trait variance plus additional state-dependent variance (Cole et al., 2005; Kenny & Zautra, 1995; Steyer et al., 2015). Within-item, cross-wave residual covariances were free to account for item-level shared method variance across waves (LaGrange & Cole, 2008). Variance components and autoregressive coefficients were also free.

Next, TI and TV components of Inhibitory and Prospective IU were examined in separate models as predictors of latent OCD Symptoms across the six waves of measurement. As an examination of specificity, latent Depressive Symptoms were added as a covariate in the TI-TV model predicting latent OCD Symptoms (Figure 3). As a comparison, models examining the relation between TI and TV components of Inhibitory and Prospective IU and Depressive Symptoms were also constructed, as well as with the addition of OCD Symptoms as a covariate.



*Note:* Depressive Symptoms were treated as a covariate across all components of the model (OCD Symptoms, Time-Invariant IU, and Time-Varying IU at Wave 1). Models were also constructed in which latent Depressive Symptoms, controlling for OCD Symptoms, were regressed onto the time-invariant and time-varying components of IU at each wave in a similar manner.

**Figure 3.** Example path diagram in which latent OCD Symptoms, controlling for latent Depressive Symptoms, is regressed onto the time-invariant (TI) and time-varying (TV) components of IU from Knowles et al., 2022.

Maximum likelihood estimation with robust standard errors was used to utilize cases with partial data. Maximum likelihood estimation assumes only that the data are missing at random (not missing completely at random, like listwise or pairwise deletion) and can yield robust estimates even when this assumption is violated (Little & Rubin, 1987; Muthén et al., 1987; Schafer, 1997). In addition, maximum likelihood estimation with robust standard errors is robust to non-normality, and both OCD and depressive symptoms were positively skewed in the present sample (OCD skewness = 1.63–2.12; depressive skewness = 1.25–1.60).

## **2.3. Results**

### **2.3.1. Descriptive Statistics and Correlations of the IUS-12 Subscales**

Means, standard deviations, and correlations for the IUS-12 subscales, OCI-R, and DASS-Depression scores across waves appear in Table 1. As expected, correlations between waves generally decreased as the lag between waves increased from one month to five months. For Inhibitory IU, correlations ranged from .68 to .83. For Prospective IU, correlations ranged from .69 to .84. For OCD symptoms, correlations ranged from .74 to .92. For depressive symptoms, correlations ranged from .67 to .86.

### **2.3.2. Inhibitory IU and Prospective IU Measurement Models**

Measurement models for both Inhibitory and Prospective IU fit the data well (Appendix Table 1). Although chi-square tests were significant for both models, all other goodness-of-fit indices indicated good fit according to Hu and Bentler's (1999) criteria.

Variable	<i>N</i>	Correlations					Means	<i>SD</i>
Inhibitory IU								
Wave 1	1190						10.29	5.01
Wave 2	741	.76					10.16	4.94
Wave 3	570	.73	.76				9.76	4.81
Wave 4	471	.72	.71	.82			9.56	4.86
Wave 5	458	.73	.73	.77	.80		9.09	4.47
Wave 6	410	.68	.71	.79	.82	.83	9.34	4.79
Prospective IU								
Wave 1	1188						20.82	5.97
Wave 2	742	.77					20.63	5.98
Wave 3	570	.77	.80				20.17	5.99
Wave 4	470	.75	.79	.83			20.01	6.24
Wave 5	457	.73	.76	.77	.82		19.68	6.21
Wave 6	410	.69	.77	.78	.84	.82	19.78	6.50
OCI-R								
Wave 1	1151						9.50	8.82
Wave 2	713	.85					8.06	8.34
Wave 3	548	.74	.83				7.75	8.41
Wave 4	457	.76	.83	.78			7.44	8.02
Wave 5	443	.74	.85	.88	.89		6.94	7.77
Wave 6	389	.74	.87	.88	.88	.92	6.94	7.78
DASS-Depression								
Wave 1	1196						4.96	5.10
Wave 2	741	.75					4.44	4.62
Wave 3	570	.73	.76				4.15	4.92
Wave 4	471	.73	.72	.83			4.13	4.90
Wave 5	456	.74	.72	.79	.85		4.11	5.19
Wave 6	409	.73	.67	.79	.79	.86	4.33	5.13

*Note.* All correlations significant at  $p < .001$ . IU = intolerance of uncertainty; OCI-R = Obsessive-Compulsive Inventory Revised; DASS = Depression Anxiety Stress Scales

**Table 1.** Means, standard deviations, and correlations for IU, OCD symptoms, and depressive symptoms from Knowles et al., 2022.



### 2.3.3. Inhibitory IU and Prospective IU TI-TV Models

Parameter estimates for the Inhibitory and Prospective IU TI-TV models appear in Table 2. For the Inhibitory IU TI-TV model, estimates of both TI and TV factor variance were significant; 75.5% of the explained variance was attributable to the TI factor. The TV factor was somewhat stable across waves; TV factor stability was statistically significant,  $p < .001$ , unstandardized path coefficient = .292, standardized coefficients ranged from .292 to .305. For the Prospective IU TI-TV model, both the TI and TV factors contributed significant variance to the target factor; 80.4% of the explained variance was attributable to the TI factor. TV factor

Parameter	Inhibitory IU		Prospective IU	
	Estimate	SE (range)	Estimate	SE (range)
Factor Variance				
Total variance	.823		.606	
TI factor variance	.621	.040	.487	.035
TV factor variance	.202	.015	.119	.009
Proportion of variance attributed to TI factor	.755	(.755-.843)	.804	(.804-.843)
Proportion of variance attributed to TV factor	.245	(.157-.245)	.196	(.157-.196)
TV factor stability				
Unstandardized	.292	.032	.212	.037
Standardized	.292-.305		.212-.217	
Indicator variable factor loadings onto the latent IU factor				
Unstandardized	0.875-1.142	.023-.027	0.902-1.169	.033-.040
Standardized	.720-.915		.631-.766	
Loadings of the latent IU factor onto the TI factor				
Unstandardized	Fixed at 1.0		Fixed at 1.0	
Standardized	.869-.902		.896-.911	
Loadings of the latent IU factor onto the TV factor				
Unstandardized	Fixed at 1.0		Fixed at 1.0	
Standardized	.432-.495		.412-.443	

Note: IU = Intolerance of uncertainty, TI = Time invariant, TV = Time varying. All estimates significant,  $p < .001$ . Estimates are based on wave 1 variance estimates; range reflects slight variability in TV variance estimates across waves. Proportion of variance =  $\frac{var(TI)}{var(TI)+var(TV)}$ .

**Table 2.** Parameter estimates for IU TI-TV models from Knowles et al., 2022.

stability was significant,  $p < .001$ , unstandardized path coefficient = .212, standardized coefficients ranged from .212 to .217.

#### **2.3.4. TI-TV Models in the Prediction of OCD Symptoms**

In examining TI and TV components of the IUS-12 as predictors of OCD symptoms, a latent OCD Symptom variable was extracted from the OCI-R subscales of obsessing, checking, ordering, washing, and neutralizing symptoms, which was regressed onto the TI and TV components of Inhibitory and Prospective IU in separate models. Each model was tested six times<sup>3</sup>, once at each wave of data (e.g., OCD symptoms at Wave 3 with the TV component of Prospective IU at Wave 3). All models fit the data well (Appendix Table 1). In both the Inhibitory and Prospective IU models at each wave, the regression weight for the TI factor was significant,  $p < .001$ ; standardized coefficients ranged from .477 to .590 for Inhibitory IU and .540 to .570 for Prospective IU. The TV components of both Inhibitory and Prospective IU were not consistently significant predictors of OCD symptoms, standardized coefficients ranged from -.019 to .195 for Inhibitory IU and -.019 to .155 for Prospective IU (Table 3).

To compare the relation between OCD Symptoms and each TI IU factor to the relation between OCD Symptoms and each TV IU factor, the models were tested again with the variance of the TV and TI IU factors constrained to be equal. These constrained models were compared to the unconstrained models using the Satorra-Bentler Scaled Chi-Square Difference Test (Satorra & Bentler, 2010). In all six waves, the constrained model demonstrated worse fit than the unconstrained model (scaled  $\Delta\chi^2 = 55.21-132.84$ ) for both Inhibitory IU and Prospective IU, suggesting that the TI and TV IU factors do not equally predict OCD symptoms. For both

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<sup>3</sup> Given the complexity of each TI-TV model and sample size limitations, it was not feasible to predict all six waves of OCD symptoms in a single model. Cross-lagged models attempting to model the data prospectively did not converge. However, results were largely consistent across the single-wave models.

Dependent variable	Wave	TI factor			TV factor		
		B	SE (B)	$\beta$	B	SE (B)	$\beta$
OCD Symptoms							
Inhibitory IU $\rightarrow$ OCD Symptoms							
Symptoms	1	1.043	.104	.534***	0.151	.222	.044
	2	1.003	.121	.567***	0.119	.210	.038
	3	0.926	.135	.477***	0.700	.253	.195**
	4	0.984	.123	.590***	-0.060	.205	-.019
	5	0.942	.122	.587***	0.563	.243	.174*
	6	0.922	.141	.555***	0.362	.337	.097
Prospective IU $\rightarrow$ OCD Symptoms							
	1	1.236	.098	.557***	-0.086	.250	-.019
	2	1.068	.107	.562***	0.532	.251	.134*
	3	1.198	.145	.570***	0.267	.286	.059
	4	0.973	.122	.540***	0.116	.246	.029
	5	0.974	.114	.566***	0.603	.269	.155*
	6	1.008	.140	.557***	0.279	.343	.066

Note. \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ ; OCD = obsessive-compulsive disorder; TI = time-invariant factor; TV = time-varying factor; IU = intolerance of uncertainty

**Table 3.** Regression of latent OCD Symptoms at six waves onto TI and TV latent variables extracted from IU from Knowles et al., 2022.

Inhibitory and Prospective IU, the TI factor was a consistently and significantly stronger predictor of OCD Symptoms compared to the TV factor (Table 4).

Model	$\chi^2$	df	$\Delta\chi^2$	Model	$\chi^2$	df	$\Delta\chi^2$
<i>Inhibitory IU</i>				<i>Prospective IU</i>			
Wave 1	1020.81	506	55.21	Wave 1	2267.32	960	132.84
Wave 2	981.98	505	72.55	Wave 2	2260.59	959	111.95
Wave 3	977.83	505	76.31	Wave 3	2262.08	959	100.18
Wave 4	980.53	505	93.68	Wave 4	2262.26	959	99.73
Wave 5	1039.28	505	105.10	Wave 5	2312.33	959	96.05
Wave 6	1056.92	506	108.51	Wave 6	2328.49	960	90.71

Note. In models with equality constraints, the variance of the time-invariant and time-varying IU factors were constrained to be equal. This constraint increased the degrees of freedom by 1 in each model. All Satorra-Bentler scaled chi-square tests were significant at  $p < .001$ . IU = intolerance of uncertainty

**Table 4.** Goodness of fit indices for TI-TV models with OCD symptoms with equality constraints from Knowles et al., 2022.

### 2.3.5. TI-TV Models in the Prediction of Depressive Symptoms

To determine the specificity of the associations between TI and TV IU on OCD symptoms, comparison models were constructed examining whether TI or TV components of IU predicted depressive symptoms. A latent Depressive Symptom variable was extracted from the DASS-Depression indicators, which were regressed onto the TI and TV factors of Inhibitory and Prospective IU in separate models. Each model was tested six times, with outcomes selected from each wave of data (e.g., Depressive Symptoms at Wave 3 with the TV component of Prospective IU at Wave 3). All models fit the data well (Appendix Table 1). In both the Inhibitory and Prospective IU models at each wave, the regression weight for the TI factor was significant,  $p < .001$ ; standardized coefficients ranged from .491 to .598 for Inhibitory IU and .338 to .441 for Prospective IU. The TV components of both Inhibitory and Prospective IU were not consistently significant predictors of Depressive Symptoms, with standardized coefficients ranging from .009 to .166 for Inhibitory IU and .031 to .191 for Prospective IU (Table 5).

Dependent variable	Wave	TI factor			TV factor		
		B	SE (B)	$\beta$	B	SE (B)	$\beta$
Depressive Symptoms	Inhibitory IU → Depressive Symptoms						
	1	0.482	.034	.540***	0.107	.081	.067
	2	0.420	.035	.529***	0.188	.081	.133*
	3	0.441	.042	.491***	0.255	.116	.153*
	4	0.438	.040	.523***	0.269	.119	.166*
	5	0.520	.045	.545***	0.017	.144	.009
	6	0.546	.045	.598***	0.299	.134	.154*
	Prospective IU → Depressive Symptoms						
	1	0.390	.035	.394***	0.062	.101	.031
	2	0.288	.035	.338***	0.341	.122	.191**
	3	0.359	.042	.371***	0.343	.134	.165**
	4	0.356	.042	.395***	0.182	.126	.091
	5	0.362	.050	.357***	0.127	.161	.055
	6	0.432	.054	.441***	0.219	.148	.096

Note. \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ . TI = time-invariant factor; TV = time-varying factor; IU = intolerance of uncertainty

**Table 5.** Regression of latent Depressive Symptoms at six waves onto TI and TV latent variables extracted from IU from Knowles et al., 2022.

To compare the relation between Depressive Symptoms and the TI IU factor to the relation between Depressive Symptoms and the TV IU factor, the models were tested again with the variance of the TV and TI IU factors constrained to be equal and compared using the Satorra-Bentler Scaled Chi-Square Difference Test. At all six waves, the constrained model demonstrated worse fit than the unconstrained model (scaled  $\Delta\chi^2 = 62.72-138.33$ ) for both Inhibitory and Prospective IU, suggesting that the TI IU factor and TV IU factor are not equal predictors of Depressive Symptoms. For both Inhibitory and Prospective IU, the TI IU factor was a consistently and significantly stronger predictor of Depressive Symptoms compared to the TV IU factor (Table 6).

Model	$\chi^2$	df	$\Delta\chi^2$	Model	$\chi^2$	df	$\Delta\chi^2$
<i>Inhibitory IU</i>				<i>Prospective IU</i>			
Wave 1	1330.29	575	62.72	Wave 1	2699.96	1053	138.33
Wave 2	1190.42	574	68.52	Wave 2	2566.35	1052	119.75
Wave 3	1185.58	574	75.44	Wave 3	2511.92	1052	103.08
Wave 4	1260.85	574	91.41	Wave 4	2664.03	1052	100.96
Wave 5	1244.95	574	96.37	Wave 5	2596.07	1052	94.95
Wave 6	1181.31	575	110.26	Wave 6	2542.34	1053	92.14

*Note.* In models with equality constraints, the variance of the time-invariant and time-varying IU factors were constrained to be equal. This constraint increased the degrees of freedom by 1 in each model. All Satorra-Bentler scaled chi-square tests were significant at  $p < .001$ . IU = intolerance of uncertainty

**Table 6.** Goodness of fit indices for TI-TV models with depressive symptoms with equality constraints from Knowles et al., 2022.

### 2.3.6. Specificity of TI-TV Models of IU in the Prediction of OCD and Depressive Symptoms

After separately examining the TI and TV components of Prospective and Inhibitory IU in the prediction of both OCD and Depressive Symptoms, covariates were introduced into the

models as a test of specificity (Figure 3). First, the associations between TI and TV Prospective and Inhibitory IU and OCD Symptoms were examined with Depressive Symptoms as a covariate. Next, the associations between TI and TV Prospective and Inhibitory IU and Depressive Symptoms were examined with OCD Symptoms as a covariate. Again, the models were examined at each wave of data (e.g., Depressive Symptoms at Wave 3, controlling for OCD Symptoms at Wave 3, with the TV component of Prospective IU at Wave 3). Model indicators suggested good fit for each model (Appendix Table 1). In the Inhibitory IU models at each wave, the regression weight for TI variance was significant in the prediction of OCD Symptoms,  $p < .001$ , with standardized coefficients ranging from .463 to .579, as well as in the prediction of Depressive Symptoms,  $p < .001$ , standardized coefficients range from .410 to .589 (Table 7). The TV component of Inhibitory IU was not a consistently significant predictor of either OCD Symptoms or Depressive Symptoms; standardized coefficients ranged from -.026 to .188 for OCD Symptoms and -.001 to .167 for Depressive Symptoms.

Dependent variable	Wave	Depressive Symptoms			TI factor			TV factor			
		B	SE (B)	$\beta$	B	SE (B)	$\beta$	B	SE (B)	$\beta$	
OCD Symptoms		Inhibitory IU → OCD Symptoms									
	1	0.303	.096	.139**	0.895	.116	.463***	0.107	.211	.031	
	2	0.446	.138	.201**	0.815	.125	.465***	0.066	.196	.021	
	3	0.056	.112	.026	0.904	.141	.468***	0.678	.250	.188**	
	4	0.050	.124	.025	0.960	.134	.579***	-0.085	.202	-.026	
	5	0.103	.119	.062	0.886	.142	.555***	0.547	.245	.171*	
	6	0.013	.174	.007	0.909	.168	.553***	0.365	.331	.104	
			Prospective IU → OCD Symptoms								
	1	0.436	.094	.198***	1.049	.099	.485***	-0.118	.233	-.027	
	2	0.604	.137	.274***	0.890	.103	.475***	0.348	.240	.088	
	3	0.148	.105	.069	1.149	.143	.553***	0.208	.277	.046	
	4	0.258	.125	.130*	0.887	.124	.497***	0.055	.235	.014	
5	0.236	.099	.141*	0.871	.116	.517***	0.565	.260	.148*		
6	0.186	.144	.102	0.920	.146	.519***	0.235	.329	.057		

		OCD Symptoms			TI factor			TV factor		
Depressive Symptoms	Inhibitory IU → Depressive Symptoms									
	1	0.064	.021	.140**	0.416	.044	.467***	0.090	.078	.057
	2	0.095	.032	.210**	0.324	.048	.410***	0.173	.077	.123*
	3	0.010	.025	.022	0.432	.050	.482***	0.245	.117	.147*
	4	0.028	.036	.056	0.410	.058	.490***	0.270	.121	.167*
	5	0.033	.048	.055	0.490	.067	.514***	-0.002	.141	-.001
	6	0.008	.049	.014	0.539	.069	.589***	0.299	.134	.154*
	Prospective IU → Depressive Symptoms									
	1	0.115	.024	.254***	0.253	.046	.257***	0.050	.094	.025
	2	0.167	.036	.369***	0.112	.051	.132*	0.240	.118	.135*
	3	0.047	.030	.101	0.304	.058	.316***	0.324	.133	.157*
	4	0.080	.038	.158*	0.280	.060	.313***	0.167	.122	.084
	5	0.113	.053	.189*	0.255	.073	.253***	0.065	.154	.029
6	0.072	.054	.132	0.358	.083	.368***	0.204	.149	.090	

Note. \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ ; IU = intolerance of uncertainty; OCD = obsessive-compulsive disorder; TI = time-invariant factor; TV = time-varying factor

**Table 7.** Regression of latent OCD Symptoms controlling for latent Depressive Symptoms (top) and latent Depressive Symptoms controlling for OCD Symptoms (bottom) at six waves onto TI and TV latent variables extracted from Inhibitory and Prospective IU from Knowles et al., 2022.

In the Prospective IU models at each wave, the regression weight for TI variance was significant in the prediction of OCD Symptoms,  $p < .001$ , standardized coefficients ranging from .475 to .553. In the prediction of Depressive Symptoms, TI Prospective IU was significant across all waves except Wave 2,  $p < .05$ , standardized coefficients ranging from .132 to .368. As with Inhibitory IU, the TV component of Prospective IU was not a consistently significant predictor of either OCD or Depressive Symptoms; standardized coefficients ranged from -.027 to .148 for OCD Symptoms and .025 to .157 for Depressive Symptoms (Table 7).

The specificity models were re-tested with the variance of the TV and TI IU factors constrained to be equal and compared using the Satorra-Bentler Scaled Chi-Square Difference Test. In the prediction of both OCD Symptoms and Depressive Symptoms at all six waves, the constrained model demonstrated worse fit than the unconstrained model, scaled  $\Delta\chi^2 > 60.35$  for both Inhibitory IU and Prospective IU, suggesting that the TI and TV IU factors do not equally

predict Depressive Symptoms (when controlling for OCD Symptoms) or OCD Symptoms (when controlling for Depressive Symptoms). Overall, TI Prospective IU was a stronger predictor of OCD Symptoms compared to Depressive Symptoms (Table 8).

Model	$\chi^2$	df	$\Delta\chi^2$	Model	$\chi^2$	df	$\Delta\chi^2$
TI-TV models with OCD symptoms, controlling for depressive symptoms							
<i>Inhibitory IU</i>				<i>Prospective IU</i>			
Wave 1	1877.60	762	60.35	Wave 1	3384.70	1300	134.81
Wave 2	1638.95	760	69.75	Wave 2	3144.14	1299	117.42
Wave 3	1568.95	760	78.20	Wave 3	3001.64	1298	104.23
Wave 4	1562.21	760	98.96	Wave 4	3053.33	1298	101.79
Wave 5	1576.27	760	100.26	Wave 5	3016.73	1298	94.60
Wave 6	1519.84	761	113.12	Wave 6	2974.52	1299	97.22
TI-TV models with depressive symptoms, controlling for OCD symptoms							
<i>Inhibitory IU</i>				<i>Prospective IU</i>			
Wave 1	1877.40	762	65.54	Wave 1	3389.31	1300	148.52
Wave 2	1637.48	760	74.50	Wave 2	3128.90	1298	121.11
Wave 3	1564.35	760	84.06	Wave 3	2999.92	1298	97.37
Wave 4	1553.30	760	99.36	Wave 4	3051.35	1298	101.60
Wave 5	1582.51	760	96.66	Wave 5	3015.25	1298	94.49
Wave 6	1521.13	761	108.03	Wave 6	2969.25	1299	91.67

*Note.* In models with equality constraints, the variance of the time-invariant and time-varying IU factors were constrained to be equal. This constraint increased the degrees of freedom by 1 in each model. All Satorra-Bentler scaled chi-square tests were significant at  $p < .001$ . IU = intolerance of uncertainty; OCD = obsessive-compulsive disorder; TI = time-invariant factor; TV = time-varying factor

**Table 8.** Goodness of fit indices for TI-TV specificity models with equality constraints from Knowles et al., 2022.

## 2.4. Discussion

Although descriptive and experimental studies suggest that IU is a personality trait that may contribute to the etiology and maintenance of OCD (Calleo et al., 2010; Sarawgi et al., 2013), the extent to which IU is “trait-like” is yet to be examined in the literature. To address this gap, the present study applied a latent trait-state model to the Inhibitory and Prospective factors



of the IUS-12 over a five-month period. The findings showed that although estimates of TI factor variance and TV factor variance were significant in models of Prospective and Inhibitory IU, the amount of TI variance was greater than the amount of TV variance. More specifically, the TI component of both Prospective and Inhibitory IU was significantly larger than the TV component over the five-month period, with 76 to 84 percent of the variance in the Inhibitory IU model attributable to the TI component and 80 to 84 percent of the variance in the Prospective IU model attributable to the TI component. Furthermore, while TV factor stability was statistically significant for both Inhibitory and Prospective IU, these associations were not robust. Consistent with existing theory (Cattell, 1966; Eysenck, 1983; Hertzog & Nesselroade, 1987), the present findings suggest that both factors of the IUS-12 consist of a stable trait component and, to a lesser extent, a time-varying state component. The present findings also converge with research showing that various personality factors generally have larger stable trait variance than state-specific variance (Olatunji et al., 2020; Wagner et al., 2019).

The present study suggests that IU, as assessed by the IUS-12, largely consists of a TI component. However, the origins of this TI component are unclear. The TI component of the IUS-12 may be the product of neuroticism, a trait that is defined as the tendency to experience frequent, intense negative emotions associated with a sense of uncontrollability in response to stress (Barlow et al., 2014). Heightened neuroticism may result in negative beliefs about uncertainty and its implications, including a sense of uncontrollability. The proposed link between neuroticism and the development of IU is consistent with recent research. For example, Hawes and colleagues (2021) found that high neuroticism and low extraversion at age 3, as measured through laboratory observation and parent report, predicted self-reported and parent-reported IU at ages 12 and 15. Self-reported IU also significantly increased between ages 12 and

15. This finding offers some important insight into a developmental window of the TV dimension, where IU does not appear to be perfectly stable. Future longitudinal research across the developmental spectrum and, when possible, across multiple informants (i.e., self, parent, partner, teacher) is needed. Indeed, different sources of information about one's level of IU may be differentially sensitive to the TI and TV dimensions (e.g., Cole et al., 2017).

IU may function as a specific cognitive risk factor by which the broader underlying vulnerability of trait neuroticism has its effects on the development of various emotional disorders (Norton & Mehta, 2007; Sexton et al., 2003). However, it is unclear the extent to which the effect of IU on emotional disorders is a function of the observed TI or TV component. The present study found that the TI components of both Inhibitory and Prospective IU were significantly associated with latent OCD Symptoms across all waves. In contrast, the TV components of Inhibitory and Prospective IU were not reliably associated with OCD Symptoms. Furthermore, in models where the TV component of IU demonstrated significant associations with OCD Symptoms, the relative weights of the TI component were significantly larger; models constraining the two predictors to be equal demonstrated worse fit, suggesting that the TI component of IU has a greater impact on OCD Symptoms than the TV component. These findings may have important implications for etiological models of OCD. More specifically, the determinants of IU that appear to remain relatively consistent over time may confer risk for the development of OCD, rather than the determinants of IU that vary over time.

A major aim of the present study was to examine the extent to which the TI and TV components of IU are specific to OCD symptoms compared to depressive symptoms. Consistent with the conceptualization of IU as a transdiagnostic risk factor (Gillett et al., 2018; McEvoy et al., 2019), the present study found that the TI component of both Inhibitory and Prospective IU

were significantly associated with latent Depressive Symptoms. Previous research has shown that IU may confer risk for depression (Jensen et al., 2016), perhaps by increasing distress in uncertain situations. However, the present study is the first to show that the determinants of IU that are relatively stable over time may contribute to depressive symptoms. From the perspective that IU is a specific social-cognitive vulnerability related to neuroticism (Naragon-Gainey & Watson, 2018), and that such patterns of negatively interpreting and responding to negative emotions increase and maintain symptoms of emotional disorders (Barlow et al., 2014), a link between IU and depression may be expected, especially given the high comorbidity between anxiety and depression (Jensen et al., 2016; Yook et al., 2010). Not unlike the development of OCD, the TI component of IU may be one mechanism by which neuroticism confers risk for depression. The TI component of IU may also be characterized by repetitive negative thinking, conferring specific risk for depression (McEvoy & Mahoney, 2013). For example, recent research suggests that the relation between IU and depressive symptoms may be mediated by rumination (Huang et al., 2019). Among individuals high in IU, rumination may be a common response to situations that are ambiguous and uncertain, leading to reduced problem-solving, increased distress, and the development of depression. However, additional research examining other symptoms of psychopathology (e.g., worry, panic, etc.) is needed to determine if both the TI and TV components of IU are transdiagnostic.

As a more rigorous test of specificity, the present study found that TI IU was significantly associated with OCD Symptoms across all waves when controlling for Depressive Symptoms. The TI component was also generally associated with Depressive Symptoms when controlling for OCD Symptoms. However, a different pattern of associations between OCD Symptoms and Depressive Symptoms emerged when examining the Inhibitory and Prospective factors of IU. In

particular, TI Prospective IU appears to be more strongly related to OCD Symptoms (when controlling for Depressive Symptoms) compared to Depressive Symptoms (when controlling for OCD Symptoms); standardized path coefficients in these models are larger across all waves for the TI component of Prospective IU and OCD Symptoms ( $\beta$  range .465-.542) than for the TI component of Prospective IU and Depressive Symptoms ( $\beta$  range .104-.366)<sup>4</sup>. Whereas Inhibitory IU refers to avoidance-oriented responses to uncertainty, Prospective IU is the tendency toward active information seeking in order reduce uncertainty. Importantly, Prospective IU is associated with approach-oriented strategies to cope with uncertainty (Birrell et al., 2011). Although depressed individuals may also experience discomfort with uncertainty, approach-oriented attempts to cope are less characteristic of the disorder. Consistent with previous research demonstrating that Inhibitory IU is more strongly associated with depressive symptoms than Prospective IU (Saulnier et al., 2019), the commonly observed transdiagnostic effects of IU may largely be accounted for by the TI component of Inhibitory IU.

To our knowledge, the present study is the first to examine TI and TV components of IU and their association with symptoms of OCD and depression using a longitudinal design. A large proportion of IU was time-invariant, and this trait-like feature is strongly associated with OCD symptoms (and depressive symptoms to a lesser degree). However, the present findings should be considered in light of several limitations. For example, these analyses rely exclusively on self-report measures. Recent research efforts have focused on the development of behavioral measures of IU (Osmanağaoğlu et al., 2021), and future research should expand upon existing work by incorporating multimodal measurement, including reliable behavioral and biological

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<sup>4</sup> Given that these non-nested models include the same number of parameters, standard frequentist methods of comparing model fit, such as by comparison of AIC and BIC values, are not available (Myung & Pitt, 1997).

indicators of IU. A multi-method approach that also incorporates measurement of specific stressors or utilizes ecological momentary assessment may also provide much needed insight into the TV component of IU. In addition, clinician-administered symptom measures may provide a more precise estimation of the link between IU and various forms of psychopathology. Similarly, because the present study only included a single measure of IU, the results of this study should be interpreted as relevant only to IUS-12 scores. Indeed, time-varying and time-invariant components of IU, and their relation with OCD and depressive symptoms, may vary dependent on the measure used. It is also important to note that the use of a 2-factor model of the IUS-12, though demonstrating best fit within the present sample, differs from recommendations made in recent factor analytic studies of the IUS-12 (Hale et al. 2016; Shihata et al., 2018). Solidifying the factor structure of the IUS-12 in future research may have implications for decoupling state and trait components of IU.

Although this study employs a longitudinal design, five months may be too short to fully capture the TI and TV components of IU. Future longitudinal research should examine the stability of IU over a longer duration in both clinical and community samples, including in samples of patients diagnosed with OCD and other forms of psychopathology. Another important limitation is that the present study employed a predominantly white, female sample, which limits the generalizability of the findings. Notably, the existing literature suggests that the IUS-12 has a similar factor structure and psychometric properties across gender and racial/ethnic backgrounds (Hale et al., 2016; Norton, 2005). However, further exploration of differences in the trait-state structure of the IUS across gender and ethnicity is warranted. Future research investigating the TI and TV components of IU using multiple measures and across diverse

samples will be invaluable in more clearly delineating the underlying mechanisms that explain the relative contributions of uncertainty aversion in OCD and other forms of psychopathology.

## CHAPTER 3

### **3. Changes in Uncertainty During a Variable Exposure Intervention for Individuals with Contamination-Focused OCD Symptoms**

#### **3.1. Introduction**

The ability to tolerate distress associated with uncertainty is a key feature of individuals with obsessive-compulsive disorder (OCD). Descriptive research shows that high intolerance of uncertainty (IU) distinguishes individuals with OCD from nonanxious individuals (Tolin et al., 2003) and is correlated with symptom severity among OCD inpatients (Calleo et al., 2010). Furthermore, compulsive rituals often function to reduce distress in response to uncertainty and increase one's perception of control (Greco & Roger, 2003). For example, individuals with high obsessive-compulsive (OC) symptoms respond with more checking behaviors in mildly uncertain situations compared to individuals with low OC symptoms (Toffolo et al., 2013), and IU was correlated with engagement in behavioral tasks associated with various OC symptom dimensions (Sarawgi et al., 2013).

The gold standard treatment for OCD is exposure and response prevention (Abramowitz, 1997; Foa & McLean, 2016; Rosa-Alcázar et al., 2008). Traditionally, exposure therapy encourages patients to approach feared stimuli and situations to gradually habituate to the anxiety experienced in their presence. The key mechanism of exposure is believed to be fear extinction, in which learned associations between feared situations and aversive outcomes are extinguished over time (Moscovitch et al., 2009). Importantly, fear extinction is affected by high IU. Experimental research on fear extinction has shown that, compared with those low in IU, individuals high in IU do not discriminate between learned threat and safety cues (Morriss et al., 2015, 2016). Thus, designing exposure interventions in a way that teaches patients to tolerate

uncertainty may lead to improved exposure outcomes. A new theoretical approach which suggests that exposure works based on the principles of inhibitory learning, in which individuals incorporate new, competing safety information about their fears (Craske et al., 2008; Jacoby & Abramowitz, 2016) may offer some important insights into targeting uncertainty during treatment. According to the inhibitory learning model, during exposure therapy, the associations between the feared stimulus and negative outcomes are weakened, while the associations between the feared stimulus and safety or more neutral outcomes are strengthened. Thus, new learning is emphasized instead of the extinction of previously learned fear associations. In line with the inhibitory learning framework, increasing variability in the exposure hierarchy may improve the efficacy of exposure-based treatments by increasing tolerance of uncertainty (Craske et al., 2014; Knowles & Olatunji, 2019).

Several potential mechanisms have been hypothesized to lead to improved outcomes in variable exposure. First, increased variability may create additional cues for memory retrieval, enhancing an individual's ability to store new information in memory (Bjork & Bjork, 1992, 2006). Next, variable progression through an exposure hierarchy, in which participants face situations of varying difficulty in a non-predictable order, may maximally violate client expectancies, which also improves learning (Rescorla & Wagner, 1972). To promote adaptive learning, exposure-based interventions should be designed to provide a strong mismatch between one's expectation of the likelihood of an aversive outcome and the actual outcome. From this perspective, the more one's expectancy can be violated during exposure trials, the stronger the inhibitory learning. Indeed, research has shown that expectancy violation is a strong predictor of the efficacy of exposure-based treatment for fear-based disorders (i.e., Wannemueller et al, 2019). Varying the exposure hierarchy may maximize the mismatch between one's threat



expectancies and the actual outcome. Finally, rather than proceeding up an exposure *hierarchy* in a linear fashion where certainty regarding the next threat is available, a more variable approach where exposure items are randomly presented may provide the necessary opportunity to tolerate uncertainty.

A variable approach to exposure may not only extinguish previously learned threat associations to potentially contaminated objects, but this approach may also emphasize new safety learning in the context of uncertainty. This safety learning may also generalize to other threat-relevant emotional processes. For example, research has consistently shown that disgust plays a central role in contamination-focused OCD (Knowles et al., 2018) and habituates more slowly during exposure (Olatunji et al., 2009). Previous research has also found that individuals with contamination-focused OCD have higher IU than non-anxious individuals, as well as specific high IU associated with cleanliness (Jensen & Heimberg, 2015). This suggests that individuals high in contamination fear likely have difficulty tolerating the uncertainty associated with touching a potentially contaminated object (i.e., how can I be sure that I won't get sick from touching this doorknob?). Accordingly, an inhibitory-learning focused approach may specifically benefit individuals high in contamination fear who have difficulty tolerating the experience of disgust. A variant of exposure delivery that incorporates uncertainty in the order of presentation of feared stimuli may thus confer additional benefit for individuals with elevated contamination fear.

Varying the order of hierarchy items during exposure therapy may also lead to increased emotional variability as individuals encounter stimuli with different levels of perceived threat. Previous studies have found that increased variability in the level of fear experienced during an exposure is associated with improved exposure outcomes (Culver et al., 2012; Kircanski et al.,

2012; Waters et al., 2015), though fear variability is not consistently associated with symptom improvement (Benito et al., 2018; Jacoby et al., 2019). Several potential mechanisms for the effect of emotional variability have been proposed. First, increased emotional variability may create additional retrieval cues for extinction learning. Internal states, such as emotions, can serve as contexts within exposure (Mystkowski et al., 2003), and the use of multiple contexts in exposure allows for greater generalization of extinction learning (Bandarian-Balooch et al., 2015; Hermann et al., 2020). After exposure, an individual may then more easily recall this learning within the various emotional contexts of daily life. Second, emotional variability during exposure may promote greater distress tolerance as individuals learn that they can handle intense emotions without needing to prepare in advance (Craske et al., 2008; Knowles & Olatunji, 2019).

Three empirical studies to date have compared the efficacy of hierarchical and variable exposure. In a study of individuals with a fear of heights, no differences were found between random, variable exposure to heights compared to graded exposure, but individuals in the variable exposure condition reported greater reductions on a more general measure of anxiety (Lang & Craske, 2000). In a study with individuals with contamination fear, individuals who completed random, variable exposure did not differ from those who completed traditional graded exposure, but greater variability in fear predicted better outcomes at follow-up (Kircanski et al., 2012). Most recently, Jacoby and colleagues (2019) compared individuals with moderately distressing unacceptable thoughts after four sessions of either gradual exposure or variable exposure. While there were again no significant outcome differences after the intervention, after a three-month follow-up, 81% of the participants in the variable exposure condition exhibited clinically significant and reliable change in OCD symptoms, compared to only 37% in the gradual exposure condition. Unlike in previous studies, fear variability did not predict

intervention outcomes. Despite the proposed role of increased opportunities to tolerate uncertainty during variable exposure, none of these studies measured changes in uncertainty during exposure interventions.

Although variable exposure may confer additional benefit after treatment compared to standard, hierarchical exposure, the mechanism by which these improvements are maintained over time is not yet clear. In addition, it is not yet clear for whom variable exposure may be more beneficial than hierarchical exposure, or if variable exposure differentially impacts IU compared to standard, hierarchical exposure. The present study examined the effects of a single-session exposure intervention using a standard hierarchy compared to that of a variable exposure intervention for participants with elevated contamination fear and examined both emotional variability and changes in uncertainty as potential mechanisms for differential outcomes. It is hypothesized that individuals in both the standard hierarchical and variable conditions will report lower anxiety and disgust during a behavioral approach task (BAT), experience lower physiological activity during the BAT, and perform a greater number of steps in the BAT at one-week follow-up. However, those in the variable exposure condition were predicted to outperform those in the standard hierarchy condition on these one-week follow-up outcomes. It was also predicted that those in the variable exposure condition would report more uncertainty as well as more variability in anxiety, disgust, and uncertainty during exposure than those in the standard hierarchy condition. Variability in uncertainty and affective processes were also predicted to predict better outcomes. Lastly, in a set of exploratory analyses, IU and disgust propensity were examined as potential moderators to determine if specific symptom characteristics differentially predict outcomes in the standard and variable exposure conditions.

## 3.2. Method

### 3.2.1. Participants

Undergraduate students and community adults with high contamination fear were recruited for the present study. Individuals who scored above 13 on the Padua Inventory Contamination Scale (PI; Burns et al., 1996) were eligible to participate; the cutoff score on this measure is in line with the mean PI score among clinical samples and has been used in previous studies (e.g., Olatunji, Lohr, et al., 2007). Participants were excluded if they endorsed any heart, respiratory, or neurological condition (excluding controlled asthma) or if they were pregnant, as these conditions would affect the quality of the physiological data gathered during the study. A total of 77 participants were enrolled in the study, of whom 73 were randomly assigned to either the standard or variable exposure intervention; four participants withdrew from the study or were lost to follow up before random assignment. After random assignment to exposure condition, two participants withdrew from the study before the third visit and one withdrew before the final visit, citing scheduling difficulties. Table 9 displays demographic information for the full sample.

	Standard		Variable		Total		<i>F</i> / $\chi^2$	<i>p</i>
	<i>(n</i> = 35)		<i>(n</i> = 38)		<i>(n</i> = 73)			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
% female	88.6		78.9		83.6		1.229	.268
% white	65.7		60.5		63.0		.210	.646
Age	29.34	15.11	27.08	12.90	28.16	13.95	.476	.492
PI Contamination	21.17	5.55	22.63	6.48	21.93	6.06	1.060	.307
OCI-R Washing	5.06	2.90	5.47	3.38	5.27	3.14	.317	.575
DPSS-R Propensity	26.82	5.23	26.61	4.74	26.71	4.95	.035	.852
IU	34.03	10.57	33.24	10.39	33.62	10.41	.104	.748

*Note:* PI = Padua Inventory, OCI-R = Obsessive-Compulsive Inventory Revised, DPSS-R = Disgust Propensity and Sensitivity Scale Revised, IU = Intolerance of Uncertainty

**Table 9.** Demographic and baseline information.

### 3.2.2. Measures

The *Padua Inventory Contamination Subscale* (PI; Burns et al., 1996) is a 10-item self-report questionnaire designed to assess fear of contamination. Items are rated on a Likert scale from 1 (not at all) to 5 (very much). Higher scores on this inventory indicate more contamination fear. In the present sample, the PI was found to have acceptable internal consistency ( $\alpha = .70$ ).

The *Obsessive-Compulsive Inventory-Revised* (OCI-R; Foa et al., 2002) is an 18-item self-report measure of OC symptoms in the past month. The OCI-R consists of 6 subscales measuring specific categories of OC symptoms (washing, checking, ordering, neutralizing, hoarding, obsessing). Participants rate items based on how much the experience has bothered them during the past month from 0 (not at all) to 4 (extremely). Only the washing subscale is reported in the present study ( $\alpha = .79$ ).

The *Intolerance of Uncertainty Scale-12* (IUS-12; Carleton et al., 2007) is a 12-item self-report scale of the perceived ability to tolerate the possibility of unpredictable negative events. Items on the IUS-12 are rated on a Likert scale from 1 (not at all characteristic of me) to 5 (entirely characteristic of me), and higher scores indicate greater IU. In the present sample, the IUS-12 demonstrated excellent internal consistency ( $\alpha = .92$ ).

The *Disgust Propensity and Sensitivity Scale-Revised* (DPSS-R; Olatunji, Cisler, et al., 2007; van Overveld et al., 2006) is a 16-item self-report measure of individuals' reactions to disgust. Respondents answer how often each item is true for them on a Likert scale from 1 (never) to 5 (always). The DPSS-R is made up of two subscales: The Disgust Propensity scale measures how frequently individuals experience disgust, and the Disgust Sensitivity scale measures the emotional impact of experiencing disgust. Only the Disgust Propensity subscale is reported in the present study ( $\alpha = .84$ ).

### 3.2.3. Apparatus

Skin conductance level (SCL) was measured using a Biopac MP150 and two non-invasive Ag-AgCl electrodes placed on the middle phalanges of the second and third fingers on the participant's non-dominant hand. The equipment allowed for participants to move freely around the room and was worn during the BAT and exposure intervention. Due to technical problems with the equipment, 59 participants had complete SCL baseline data, 62 participants had complete SCL data during the exposure, 61 participants had complete SCL data at the post-intervention visit, and 51 participants had complete SCL data at the one-week follow-up.

### 3.2.4. Procedure

Following informed consent, participants completed four laboratory sessions (baseline, intervention, post-intervention, one-week follow-up). At the first visit, participants completed measures of current OC washing symptoms and related cognitive vulnerabilities. Baseline skin conductance was also collected during a five-minute waiting period. At the baseline, post-intervention, and one-week follow-up visits, participants took part in a BAT in a public bathroom and performed a series of steps by touching objects in the bathroom with increasing difficulty. At each step, participants provided ratings of anxiety and disgust on a scale from 0 (“no anxiety/disgust”) to 100 (“the most anxiety/disgust I've ever felt”). The BAT ended once a participant chose not to perform a given task. Participants also wore mobile psychophysiological equipment during the task to continuously measure physiological responses.

At the second visit, participants were randomly assigned to complete either an exposure intervention (hierarchical or variable) in a *different* bathroom location (new context). In both exposure conditions, participants received brief psychoeducation regarding the rationale for the exposure intervention. Participants in the standard exposure condition ( $n = 35$ ) were asked to

complete a series of 17 steps in a predetermined order that ranged from easiest to most difficult based on pilot testing (see Table 10). Participants in the random, variable condition ( $n = 38$ ) completed the same steps as participants in the standard condition, but step progression was determined randomly. Disgust and anxiety ratings were also assessed at each step in the exposure intervention on the same scale used during the BAT. Before each exposure step, perceived uncertainty was assessed by asking participants to rate how uncertain they felt about being able to do the upcoming, unrevealed task on a scale from 0 (“completely certain”) to 100 (“completely uncertain”). Participants could refuse to complete any step in the exposure intervention; if they refused, they were offered the opportunity to complete the next step so that all participants had the opportunity to receive the same “dose” of the intervention. Participants were coached and encouraged to complete as many steps as they could and were asked to commit to response prevention by resisting handwashing for two hours after the end of the session<sup>5</sup>. All participants were debriefed at the end of the fourth session and received either course credit or \$50 for their participation. All procedures were approved by the university IRB.

### **3.2.5. Data Analytic Overview**

First, a series of  $t$ -tests were conducted to examine differences in baseline characteristics by condition. Process-level differences were examined between the two exposure conditions, including mean and peak anxiety, disgust, and uncertainty and within-subject variability in anxiety, disgust, and uncertainty during exposure. Variability is reported as intra-individual standard deviation (ISD). Separate growth curve models were constructed to more closely

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<sup>5</sup> Participants were largely compliant with response prevention instructions, with most (57.1%) able to resist washing their hands for at least one hour after leaving the laboratory, and only two (2.9%) reporting handwashing within 15 minutes of leaving the laboratory. There were no differences among the conditions in the distribution of response prevention compliance, Mann-Whitney  $U = 606.5$ ,  $p = .95$ . Median response prevention time did not differ by condition,  $p = .68$ .

examine individual trajectories of anxiety, disgust, and uncertainty during the exposure intervention (Level 1 = Exposure Step, Level 2 = Participant). The software package *R* (Version 4.04) was used for all multilevel and growth curve models.

Step	BAT steps (Bathroom Location #1)	Exposure steps (Bathroom Location #2)
1	Outside panel of bathroom door	Wall
2	Inside handle of bathroom door	Door
3	Outside handle of stall door	Door handle
4	Inside lock of stall door	Soap dispenser
5	Bathroom counter	Edge of sink
6	Soap dispenser	Faucet
7	Inside sink	Support rail
8	Outside trash can	Inside sink
9	Wall inside stall	Outside trash can
10	Floor in front of sink	Floor in front of sink
11	Back of toilet	Toilet handle
12	Toilet seat	Floor in front of toilet
13	Floor in front of toilet	Back of toilet
14	Toilet rim (seat raised)	Toilet seat
15	Inside toilet	Toilet rim (seat raised)
16	Arm and chest (after all previous items)	Inside toilet
17	-	Arm and chest (after all previous items)

*Note.* BAT = Behavioral approach task. Items in the BAT were presented in the same order at each session; if a participant refused a given step in the BAT, the task was ended. For the exposure session, individuals in the standard exposure condition received the items in the order listed. Individuals in the variable exposure condition were first presented with items 1 and 2, then steps 3-16 in a randomized order, then step 17. During the exposure, individuals were given the opportunity to complete all steps regardless of prior step refusal.

**Table 10.** Steps in the BAT and exposure intervention.

Next, multilevel models (Level 1 = Session, Level 2 = Participant) were constructed to examine primary intervention outcomes, including changes in anxiety and disgust ratings,



changes in the number of steps completed in the BAT, and changes in peak SCL<sup>6</sup> during the BAT from baseline to the one-week follow-up visit. Exposure condition and session (baseline, post-intervention, one-week follow-up) as well as their interaction were treated as fixed predictors in the model. Next, mean uncertainty and variability in uncertainty during exposure were entered as predictors in the multilevel model for each hypothesized primary outcome. All models included a random intercept to account for clustering.

Potential moderators of intervention outcome were also explored, including IU and disgust propensity. These Level 2 predictors were mean-centered and entered in separate multilevel models. Three-way interactions between symptom measures, exposure condition, and session were tested.

### **3.2.6. Power Analysis**

With a total sample size of  $N = 73$ , we had sufficient power (.80) to detect moderate to large differences ( $d = .67$ ) between the two conditions. With the reduced sample size ( $N = 59$  at baseline) for SCL analyses due to missingness, we had sufficient power (.80) to detect a slightly larger difference ( $d = .74$ ) between the two conditions.

## **3.3. Results**

### **3.3.1. Baseline Characteristics**

There were no significant differences between the two exposure conditions in contamination fear, IU, disgust propensity, or baseline OC washing symptoms (Table 11). During the first BAT, participants completed an average of 11 steps, refusing to touch the toilet seat (Table 10; Standard Exposure,  $M = 11.86$ ,  $SD = 3.12$ ; Variable Exposure,  $M = 11.53$ ,  $SD =$

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<sup>6</sup> Peak SCL during the BAT is reported as change from an individual's mean SCL during the five-minute baseline period at the first visit.

	Standard		Variable		<i>t</i>	<i>p</i>
	<i>(n = 35)</i>		<i>(n = 38)</i>			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
<b>Baseline BAT</b>						
Steps completed	11.86	3.12	11.53	3.95	0.40	.69
Peak anxiety	55.17	32.57	62.81	25.17	-1.12	.27
Peak disgust	64.40	30.69	70.84	25.70	-0.97	.34
Mean SCL	4.83	2.49	5.50	2.69	-1.00	.32
Peak SCL	5.98	3.35	7.29	3.59	-1.46	.15
<b>Exposure</b>						
Steps completed	15.37	1.80	15.53	1.74	-0.37	.71
Baseline distress	36.10	19.18	44.89	26.12	-1.65	.10
Mean anxiety	35.10	18.45	34.55	16.19	0.14	.89
Peak anxiety	68.43	23.76	75.13	20.84	-1.28	.20
ISD anxiety	24.47	7.97	25.80	8.55	-0.68	.50
Mean disgust	40.70	17.05	40.74	15.93	-0.01	.99
Peak disgust	75.57	21.58	83.03	19.05	-1.57	.12
ISD disgust	27.45	6.95	29.37	7.93	-1.10	.28
Mean SCL	3.26	3.56	2.28	3.61	1.04	.30
Peak SCL	6.25	4.89	5.11	4.64	0.92	.36
Mean uncertainty	20.43	21.50	20.39	26.44	0.01	>.99
ISD uncertainty	22.69	7.57	16.19	9.03	3.31	.001**
<b>Post BAT</b>						
Steps completed	12.94	2.71	13.22	2.74	-0.43	.67
Peak anxiety	46.21	27.44	53.53	26.58	-1.13	.26
Peak disgust	56.38	27.82	66.25	24.15	-1.59	.12
Mean SCL	2.04	5.22	1.28	2.88	0.70	.49
Peak SCL	3.26	6.14	2.88	3.65	0.30	.77
<b>Follow-up BAT</b>						
Steps completed	12.94	2.82	13.29	2.55	-0.53	.60
Peak anxiety	45.86	28.58	47.71	26.58	-0.28	.78
Peak disgust	57.80	28.67	59.00	23.57	-0.19	.85
Mean SCL	1.41	3.94	1.27	2.74	0.00	.89
Peak SCL	2.79	5.14	2.79	2.93	0.14	>.99

*Note.* BAT = behavioral approach task; SCL = skin conductance level

**Table 11.** Differences between exposure conditions in the exposure and BATs.

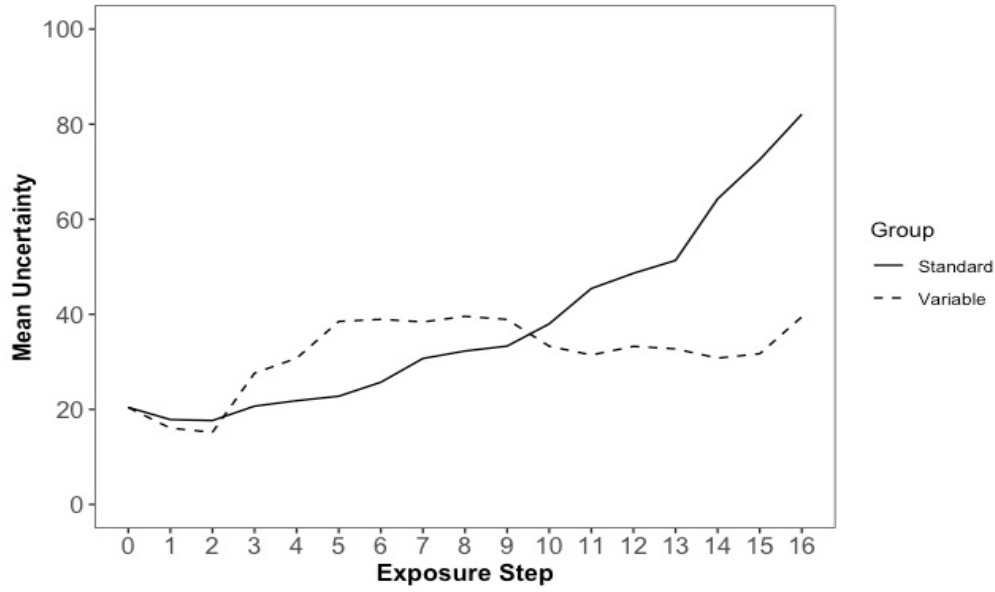
3.95,  $t(71) = 0.40, p = .69$ ). Participants reported equivalent peak anxiety and disgust during the first BAT, and peak SCL did not differ between the conditions. Baseline SCL also did not significantly differ between the two conditions (Table 11).

### **3.3.2. Behavioral, Subjective, and Physiological Responses during Exposure**

There were no differences in baseline distress immediately before exposure began. On average, participants completed 15 out of 17 steps during the exposure; steps completed during exposure did not differ between conditions. There were also no significant differences between the conditions in participants' peak SCL during exposure after accounting for individuals' baseline SCL. There were no significant differences between the two exposure conditions in mean level of anxiety or disgust during exposure, peak anxiety or disgust during exposure, or variability in anxiety or disgust during exposure (Table 11).

#### **3.3.2.1. Uncertainty During Exposure**

There were no significant differences by exposure condition in reported uncertainty at the beginning of the exposure (Standard  $M = 20.43, SD = 21.50$ ; Variable  $M = 20.39, SD = 26.44$ ),  $t(71) = .01, p > .99$ . Similarly, participants' mean reported uncertainty during exposure did not differ between the conditions (Standard  $M = 37.97, SD = 19.92$ ; Variable  $M = 31.59, SD = 21.43$ ),  $t(71) = 1.32, p = .19$ . However, variability in uncertainty did significantly differ between the exposure conditions, with individuals in the variable exposure condition (Variable  $M$   $ISD = 16.19, SD = 9.03$ ) reporting lower levels of variability compared to individuals in the standard exposure condition (Standard  $M$   $ISD = 22.69, SD = 7.57$ ),  $t(71) = 3.31, p = .001, d = .78$ . Figure 4 displays mean changes in uncertainty at each step of the exposure by condition.



**Figure 4.** Mean uncertainty during exposure.

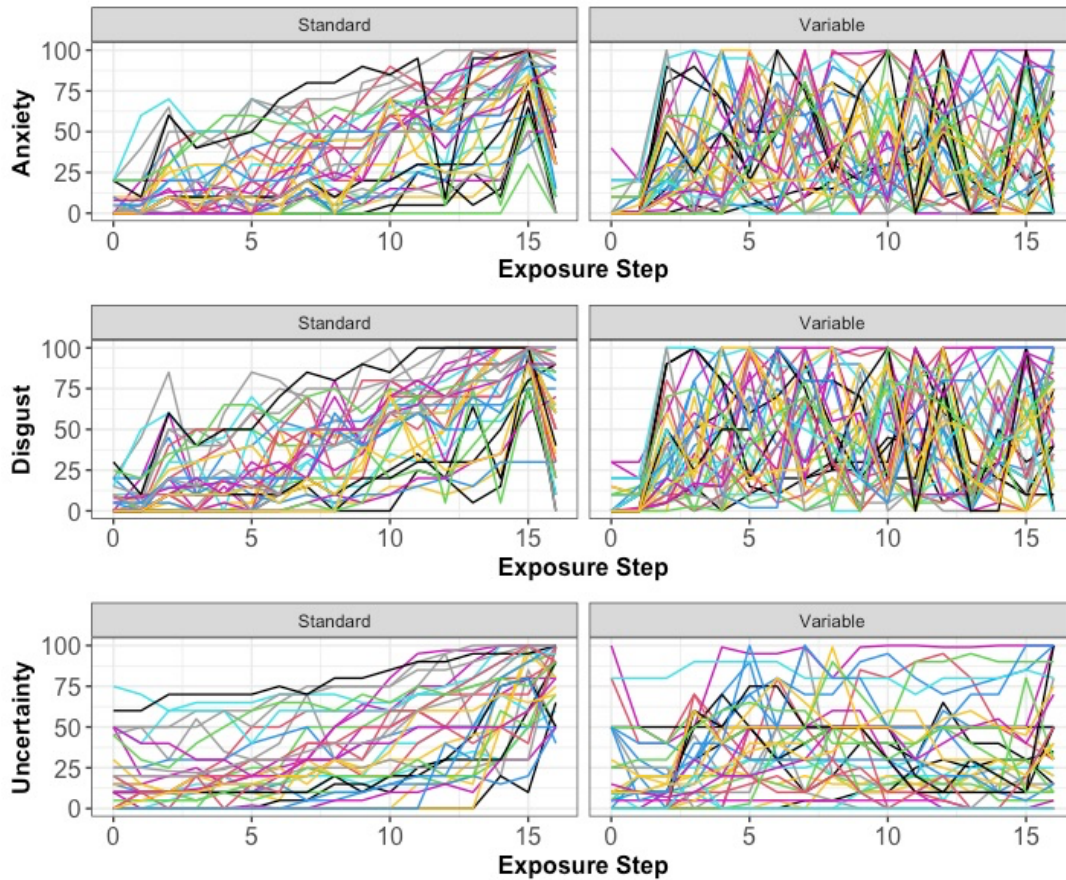
### 3.3.3. Individual Trajectories of Uncertainty, Anxiety, and Disgust during Exposure

Individual trajectories of uncertainty, anxiety, and disgust were then examined. Growth curve modeling parameters are displayed in Table 12. Individual trajectories of anxiety, disgust, and uncertainty during the exposure intervention, separated by exposure condition, are displayed in Figure 5.

Model	ICC	$\Delta\chi^2$	Fixed Effect (SE)				Random Effect [95% CI]		
			Intercept	Condition	Linear Time	Quadratic Time	Intercept	Linear Time	Quadratic Time
<b>Anxiety</b>	0.26	14.57***	3.04 (2.94)	10.67 (3.61)*	4.46 (0.51)***	-0.11 (0.03)***	13.53 [10.39, 17.62]	1.69 [1.35, 2.11]	
<b>Disgust</b>	0.20	26.20***	3.81 (3.02)	10.37 (3.57)**	5.70 (0.55)***	-0.16 (0.03)***	13.52 [9.92, 18.42]	1.63 [1.27, 2.09]	
<b>Uncertainty</b>	0.47	235.41***	18.36 (2.35)***		1.60 (0.60)**	0.04 (0.04)	18.39 [15.18, 22.28]	4.57 [3.74, 5.60]	0.33 [0.27, 0.40]

Note. \*  $p < .05$ , \*\*  $p < .01$ , \*\*\*  $p < .001$ . Missing parameters indicate that models including those parameters either did not fit as well as the best-fitting model or that the model including that parameter did not converge.

**Table 12.** Growth curve modeling parameters for anxiety, disgust, and uncertainty during exposure.



*Note.* Each line represents a single individual’s reported anxiety, disgust, or uncertainty during the exposure intervention. Trajectories are grouped by exposure condition.

**Figure 5.** Individual trajectories of anxiety, disgust, and uncertainty during exposure.

### 3.3.3.1. Uncertainty

47% of the variance in reported uncertainty during exposure can be attributed to between-subjects effects. The model indicating best fit included a significant linear effect of time, indicating that with each step in the exposure, participants reported increased uncertainty. Adding a fixed effect of condition did not improve model fit, suggesting that incorporating differences between the conditions on the rate of change in uncertainty into the model did not significantly improve the fit of the model. Thus, the effect of condition was not included in the final model of uncertainty trajectories. The random intercept, random linear effect of time, and

random quadratic effect of time were significant, suggesting that participants' reported uncertainty and slope of change in uncertainty varied with time.

### **3.3.3.2. Anxiety**

26% of the variance in anxiety during exposure can be attributed to between-subjects effects. The best-fitting model for anxiety during exposure had significant fixed linear and quadratic effects of time (i.e., step in the exposure), as well as a significant effect of condition. Overall, participant anxiety increased more quickly at the beginning of the exposure and decelerated throughout exposure. Participants in the variable exposure condition experienced steeper increases in anxiety than participants in the standard exposure condition. The random intercept and random linear effects of time were significant, suggesting that individuals differed in their initial anxiety and trajectories throughout exposure.

### **3.3.3.3. Disgust**

20% of the variance in disgust during exposure can be attributed to between-subjects effects. The best-fitting model for disgust during exposure had significant fixed linear and quadratic effects of time as well as a significant effect of condition. Overall, participant disgust increased more quickly at the beginning of the exposure and decelerated throughout exposure. Participants in the variable exposure condition experienced a steeper increase in disgust compared to participants in the standard exposure condition. The random intercept and random linear effects of time were significant, suggesting that individuals differed in their initial disgust and trajectories throughout the exposure.

### **3.3.4. Behavioral, Subjective, and Physiological Changes from Baseline to One-Week**

#### **Follow-Up**

Individuals in both exposure conditions completed additional steps during the BAT from baseline to follow-up,  $F(2, 141.83) = 15.11, p < .001$ . Number of BAT steps completed did not differ by exposure condition,  $F(1, 73.69) = 0.01, p = .92$ , and the condition by session interaction was not significant,  $F(2, 141.83) = 0.69, p = .50$ . Participants also experienced lower levels of peak anxiety during the BAT from baseline to one-week follow-up,  $F(2, 139.43) = 11.29, p < .001$ , as well as lower peak disgust,  $F(2, 139.77) = 6.97, p = .001$ . Neither peak anxiety nor peak disgust varied by exposure condition (Anxiety:  $F(1, 71.96) = 1.11, p = .30$ ; Disgust:  $F(1, 72.44) = 1.12, p = .29$ ), and the condition by session interactions were not significant (Anxiety:  $F(2, 139.43) = 0.94, p = .39$ ; Disgust:  $F(2, 139.77) = 1.32, p = .27$ ). Peak SCL during the BAT did not significantly change from baseline to one-week follow-up,  $F(2, 111.66) = 0.09, p = .92$ . Peak SCL also did not differ by exposure condition,  $F(1, 60.6) = 0.01, p = .91$ , and the exposure condition by session interaction was not significant,  $F(2, 111.66) = 0.22, p = .81$ .

### **3.3.5. Effects of Variability in Uncertainty, Anxiety, and Disgust on Intervention**

#### **Outcomes**

#### **3.3.5.1. Variability in Uncertainty**

There was a significant effect of variability in uncertainty on change in peak anxiety experienced during the BAT from baseline to follow-up,  $t(135) = 2.01, p = .047$ . Participants who experienced low variability in uncertainty during exposure (-1 SD) reported lower anxiety during the BAT at the post-intervention visit (estimated marginal mean difference = 11.9, SE = 2.27,  $t(135) = 5.23, p < .001$ ) and at one-week follow-up (estimated marginal mean difference = 23.8, SE = 4.54,  $t(135) = 5.23, p < .001$ ) compared to baseline. Participants who experienced

mean variability in uncertainty during exposure also reported significantly lower anxiety during the BAT at the post-intervention visit (estimated marginal mean difference = 5.8, SE = 1.55,  $t(135) = 3.71, p < .001$ ) and at one-week follow-up (estimated marginal mean difference = 11.5, SE = 3.11,  $t(135) = 3.71, p < .001$ ) compared to baseline. Participants who experienced high variability in uncertainty (+1 SD) did not experience significant changes in peak anxiety during the BAT from baseline to the follow-up visit,  $t(135) = -0.16, p = .87$ . There were no significant interactions between variability in uncertainty and exposure condition on any other primary outcomes.

### **3.3.5.2. Variability in Anxiety**

Individual variability in anxiety did not significantly predict changes in primary study outcomes, including change in peak anxiety during the BAT, change in number of steps taken in the BAT, or change in peak SCL during the BAT, all  $ps > .27$ . There were also no significant interactions between variability in anxiety and exposure condition on any primary outcomes.

### **3.3.5.3. Variability in Disgust**

There was a significant effect of variability in disgust on change in peak disgust experienced during the BAT from baseline to follow-up,  $t(135) = 2.21, p = .029$ . Participants who experienced low variability in disgust during exposure (-1 SD) reported lower disgust during the BAT at the post-intervention visit (estimated marginal mean difference = 7.1, SE = 1.85,  $t(135) = 3.83, p < .001$ ) and at one-week follow-up (estimated marginal mean difference = 14.2, SE = 3.70,  $t(135) = 3.83, p < .001$ ) compared to baseline. Participants who experienced mean variability in disgust during exposure also reported significantly lower disgust during the BAT at the post-intervention visit (estimated marginal mean difference = 4.5, SE = 1.33,  $t(135) = 3.39, p < .001$ ) and at one-week follow-up (estimated marginal mean difference = 9.0, SE = 2.66,



$t(135) = 3.39, p < .001$ ) compared to baseline. Participants who experienced high variability in disgust (+1 SD) did not experience significant changes in peak disgust during the BAT from baseline to the follow-up visit,  $t(135) = 1.01, p = .32$ . There were no significant interactions between variability in disgust and exposure condition on any other primary outcomes.

### **3.3.6. Individual Differences Predicting Exposure Response**

IU and disgust propensity were examined as potential moderators of intervention response to determine if specific symptom characteristics predicted the likelihood of benefiting from one exposure condition or another. There were no significant differences between the exposure conditions in steps taken during the BAT, anxiety, and disgust ratings at the post-intervention visit or one-week follow-up as a function of IU or disgust propensity ( $ps > .05$ ).

## **3.4. Discussion**

The present study compared the effectiveness of a single-session exposure intervention using a standard hierarchy to that of a variable exposure intervention for those with heightened contamination fear. Consistent with predictions, those in both exposure conditions reported lower anxiety and disgust during the BAT and performed more BAT steps at the one-week follow-up. However, the two exposure conditions did not significantly differ from each other on any primary outcome measure at the one-week follow-up. These findings are consistent with previous research that also failed to support that the hypothesis that introducing variability in exposure intensity would improve long-term outcomes relative to traditional gradual (hierarchical) exposure (Jacoby et al., 2019; Kirkanski et al., 2012). One interpretation of these findings is that the traditional exposure approach that utilizes a gradual hierarchy is likely to be sufficient, but not necessary, to produce clinical improvement in anxiety-related disorders (Kirkanski et al., 2012). Although some clinicians may express hesitation regarding a non-

hierarchical approach to exposure, it is important to note that there were no differences in average or peak anxiety and disgust during exposure or in the number of steps completed in the present study, suggesting that participants in the variable exposure condition neither experienced increased distress nor “opted out” of difficult exposures due to their random, unpredictable nature. In addition, equivalent outcomes suggest that a variable exposure approach may be used with similar effectiveness to standard, hierarchical exposure.

The present study also examined specific processes underlying single-session standard and variable exposure interventions for contamination fear, including changes in anxiety, disgust, and uncertainty. The findings revealed no significant group differences in any of the process indicators during exposure. Contrary to predictions, however, participants assigned to the variable exposure condition reported *less* overall variability in uncertainty compared to those assigned to standard exposure. This pattern of findings in the variable condition reflects participants reporting relatively moderate and steady levels of uncertainty throughout the exposure (see Figure 4). This lack of variability in participants’ perception of how uncertain they felt about being able to do the upcoming task in the variable exposure condition may reflect an inability to predict the difficulty of the upcoming step. The lack of variability in uncertainty in the variable exposure condition may also reflect an increasing sense of self-efficacy in their ability to do the upcoming task. Increased self-efficacy can occur when one exceeds their own expectations of coping with feared stimuli (Biran & Wilson, 1981). In contrast, those in the exposure intervention using a standard hierarchy experienced steadily increasing uncertainty, which likely reflects the predictable increasing difficulty of the exposure tasks.

Although there were no differences by condition in average or peak anxiety and disgust during exposure, analysis of the individual trajectories did reveal important group differences.

Overall, participant anxiety and disgust increased more quickly at the beginning of the exposure and decelerated throughout the exposure. However, those in the variable exposure condition experienced steeper increases in anxiety and disgust than those in the standard exposure condition. The steeper increases in anxiety and disgust in the variable exposure condition suggests that random presentation of hierarchy items may initially increase distress during exposure treatment. However, this steep increase in distress does not appear to translate to poorer outcomes given that no significant differences were found for both exposure conditions in BAT steps at the one-week follow-up. While there were significant group differences in overall variability in uncertainty in the present study, adding an effect of condition did not improve upon a model explaining variance in the rate of change in uncertainty. This suggests that the rate at which participants reported on how uncertain they felt about being able to do the upcoming hierarchy item was relatively similar in both exposure conditions. This pattern of findings highlights the importance of examining both group-level and individual-level change processes in more robust randomized trials as such processes may differentially relate to treatment outcome (e.g., Benito et al., 2018).

The present study did find that uncertainty variability during exposure predicted exposure outcomes independent of treatment condition. Indeed, variability in uncertainty predicted reductions in anxiety during the BAT. Specifically, participants who experienced low or moderate variability in uncertainty during exposure had significantly greater reductions in peak anxiety during the BAT from baseline to follow-up compared to individuals who experienced high levels of uncertainty variability during exposure. This finding converges with previous research highlighting the significance of uncertainty with regard to treatment outcome in anxiety and related disorders (Bomyea et al., 2015; Boswell et al., 2013). To the extent to which a lack of

variability in perceptions of uncertainty about completing the upcoming exposure task reflects increased self-efficacy, then individuals with such perceptions would be expected to benefit more from exposure therapy. Indeed, self-efficacy mediates symptom improvement after exposure therapy (e.g., Breuninger et al., 2019) and may also predict who benefits from exposure (Böhnlein et al., 2020). Interestingly, individual differences in trait measures of IU and disgust propensity were unrelated to treatment outcome in the present study. However, assessment of other traits (i.e., distress intolerance) may reveal important prescriptive indicators that identify who may benefit more from a variable exposure approach relative to the standard approach.

In contrast with previous research (Kircanski et al., 2012), but consistent with more recent findings (Benito et al., 2018; Jacoby et al., 2019), the present study did not find a significant association between anxiety variability and exposure outcomes. However, participants who experienced low or moderate variability in disgust had significantly greater reductions in disgust during the BAT from baseline to follow-up compared to individuals who experienced high levels of disgust variability during exposure. The finding of a link between disgust, but not anxiety, variability may reflect previous research showing that disgust is a more prominent emotional response than fear/anxiety in contamination-based OCD (Olatunji, Lohr, et al., 2007). The present findings also complement previous research showing that changes in disgust during exposure-based treatment for both fear-based disorders specifically (Olatunji, Huijding, et al., 2011) and OCD more broadly (Olatunji, Tart, et al., 2011) is linked to treatment outcome. Theoretical models describe disgust as a psychological process that motivates disease avoidance (Oaten et al., 2009). Low to moderate disgust variability during exposure may reflect increased tolerance of the distress associated with coming into contact with potential contagion.

Such distress tolerance may then explain reductions in disgust when confronted with a perceived contaminant.

Previous research suggests that rather than mean or peak levels of treatment process indicators, variability in process indicators may be associated with improved exposure outcomes (Culver et al., 2012; Kircanski et al., 2012; Waters et al., 2015). For example, Guzick and colleagues (2020) found that youth with OCD who reported more variable prediction accuracy experienced more rapid symptom reduction during exposure-based treatment. According to the inhibitory learning model, variability in various processes during exposure may facilitate the generalization of safety learning (Knowles & Olatunji, 2019). However, the present findings suggest that the link between distress variability during exposure and treatment outcomes may be more complex. Indeed, some studies have found that increased variability in the level of fear experienced during an exposure is associated with improved exposure outcomes (Culver et al., 2012; Kircanski et al., 2012; Waters et al., 2015) and others have not found such an association (Benito et al., 2018; Jacoby et al., 2019). Given that those who reported a high level of variability in disgust and uncertainty experienced less favorable outcomes, there may be an optimal level of fear variability during exposure-based treatment that produces more favorable outcomes in anxiety and related disorders.

Although the present study highlights how the processes of uncertainty and variability may be linked to treatment outcome using an analogue design, these findings must be considered in light of design limitations. First, the study investigates only a single-session exposure intervention, limiting its therapeutic power and generalizability to standard trials of exposure therapy. Although the single-session design allows for examination of potential within-session symptom changes, it is silent with regard to between-session changes. Future research is needed

to examine variable exposure interventions in which hierarchy items are randomly presented in repeated sessions to more directly examine important differences in within-session trajectories and how they may compare to between-session trajectories. The present study is also limited by use of an analogue sample of individuals with elevated contamination fear. Analogue samples are routinely used in OCD research and provide high-quality information regarding causal mechanisms given the dimensional nature of OCD symptoms in the population (Abramowitz et al., 2014). That being said, the generalizability of these findings does require replication with participants with a diagnosis of OCD. Despite these limitations, the present study provides an important test of a variable exposure approach in which hierarchy items are randomly presented. Future research along these lines that examines the mechanisms of modifications to exposure therapy informed by the inhibitory learning model may lead to more efficient and effective treatments for those with anxiety and related disorders.

## CHAPTER 4

### **4. Evaluating new evidence: Is IU a cognitive vulnerability for OCD?**

The two studies presented here have important implications for conceptualizing IU as a cognitive vulnerability for OCD (Table 13). First, in Chapter 2, partitioning IU into trait and state components revealed a substantial time-invariant component of IU, accounting for between 76 and 84% of the total variance in IU as measured by the IUS-12. Contributing to the conceptualization of IU as a stable, trait-like cognitive vulnerability for OCD, the time-invariant component of IU predicted obsessive-compulsive symptoms over time. Consistent with previous studies (Gentes & Ruscio, 2011; McEvoy et al., 2019) and conceptualizations of IU as a transdiagnostic cognitive vulnerability, this association does not appear to be specific to OCD, with evidence of the time-invariant component of IU also predicting depressive symptoms. However, this study provides important new evidence suggesting that it is the stable, time-invariant aspect of IU that has a significant influence on OCD symptoms, rather than the time-varying components of IU that may shift based on external factors, though evidence is currently limited to a single measure of IU. Findings along these lines support the view of the trait component of IU as a nonspecific risk factor for the etiology of OCD.

Although the trait component of IU appears to convey risk for the development of OCD, the state component may be more readily modified during treatment. Indeed, Chapter 3 demonstrates the impacts of a variable (non-hierarchical) approach to exposure intervention on perceptions of uncertainty and intervention outcomes within an analogue OCD sample. This study examined trajectories of perceived uncertainty during exposure and its effect on anxiety and disgust to determine if changes in perceived uncertainty can be considered a mechanism of effective exposure interventions for OCD. Although the study did find evidence that changes in

	Type of evidence	Example citations	Limitations
Robust association between IU and OCD symptoms	<ul style="list-style-type: none"> <li>• Strong association across multiple studies (meta-analysis)</li> <li>• Specific relationship after accounting for general factors (e.g., neuroticism)</li> <li>• Prospective associations</li> <li>• Associations across multiple levels of analysis</li> </ul>	<ul style="list-style-type: none"> <li>• Gentes &amp; Ruscio, 2011; McEvoy et al., 2019</li> <li>• Boelen &amp; Reijntjes, 2009</li> <li>• Pozza et al., 2019</li> <li>• Sarawgi et al., 2013</li> </ul>	<ul style="list-style-type: none"> <li>• Majority of studies are cross-sectional</li> <li>• Few studies examine associations across multiple levels of analysis</li> </ul>
IU as a causal risk factor that influences the development of OCD	<ul style="list-style-type: none"> <li>• Temporal precedence (changes in IU precede changes in OC symptoms within an intervention context)</li> <li>• Experimental manipulation leads to changes in OC symptoms</li> </ul>	<ul style="list-style-type: none"> <li>• Wilhelm et al., 2015</li> <li>• Faleer et al., 2017; Geok et al., 2022</li> </ul>	<ul style="list-style-type: none"> <li>• Mixed evidence of temporal precedence</li> <li>• Unclear duration of effects of manipulated IU</li> <li>• Longitudinal studies needed over long durations (e.g., early childhood to first manifestation of symptoms)</li> </ul>
IU is stable and trait-like	<ul style="list-style-type: none"> <li>• Longitudinal studies in children/adolescents</li> <li>• Longitudinal research partitioning variance into trait and state components</li> </ul>	<ul style="list-style-type: none"> <li>• Hawes et al., 2021; Zdebik et al., 2018</li> <li>• Knowles et al., 2022</li> </ul>	<ul style="list-style-type: none"> <li>• Association between IU and OC symptoms examined in a community sample; needs replication in clinical samples</li> <li>• Limited to the IUS-12 and IUSC; further studies across multiple levels of analysis are needed</li> </ul>
IU is malleable to intervention	<ul style="list-style-type: none"> <li>• Direct interventions modifying IU</li> <li>• Changes in IU after OCD treatment</li> <li>• Changes in IU associated with changes in OC symptoms after treatment</li> </ul>	<ul style="list-style-type: none"> <li>• Ladouceur et al., 2000; Mofrad et al., 2020; Oglesby et al., 2017</li> <li>• Belloch et al., 2010; Mathur et al., 2021; Su et al., 2016</li> <li>• Pinciotti et al., 2020; Wilhelm et al., 2015</li> </ul>	<ul style="list-style-type: none"> <li>• No IU-specific interventions with measured effects on OC symptoms; changes in IU and OC symptoms may be attributed to an unspecified third variable</li> </ul>

**Table 13.** Evaluating the evidence for IU as a cognitive vulnerability for OCD.

state uncertainty (operationalized as variability in uncertainty) impacted peak anxiety during a behavioral approach task, it was participants who experienced low or moderate variability in uncertainty during exposure that had greater reductions in anxiety, rather than individuals who tolerated high variability during the exposure. The results of Chapter 3 provide preliminary evidence for the malleability of state uncertainty during exposure and a potential link between



uncertainty and exposure outcomes, however additional research will be needed to examine changes in both state and trait levels of IU during exposure-based treatment for OCD (Pinciotti et al., 2020; Su et al., 2016; Wilhelm et al., 2015), rather than in the context of single-session interventions. Increased tolerance of uncertainty may be a mechanism that facilitates effective treatment for OCD, but the evidence to date is relatively non-specific (e.g., IU improves after cognitive-behavioral interventions for OCD that include an exposure component).

The literature summarized in Table 13 suggests a strong and specific association between IU and OCD symptoms across multiple studies, including both prospective associations between IU and later development of OCD symptoms, as well as significant associations across multiple levels of analysis. The existing literature also suggests that IU is a causal risk factor of OCD, with evidence of temporal precedence (changes in IU preceding changes in OC symptoms during an intervention), evidence that IU can be affected by experimental manipulation, and evidence that experimentally induced IU leads to changes in OCD symptoms. Longitudinal studies (in both children, adolescents, and adults) also demonstrate that IU is a stable, trait-like characteristic. Lastly, IU is malleable in response to both direct interventions and more general interventions targeting OCD. Based on the evidence to date (Table 13), IU is most likely a cognitive vulnerability for OCD. However, it remains unclear if improving tolerance of uncertainty is a necessary and sufficient mechanism of effective OCD treatment (e.g., Grayson, 2010).

#### **4.1. Future directions in the study of IU and OCD**

##### **4.1.1. Longitudinal research on IU and OCD**

Though prospective research on IU and OCD is increasing, there are still many unanswered questions regarding the causal nature of the association between IU and OCD. In

particular, establishing the temporal precedence of IU in predicting OCD symptoms has been difficult, with mixed results in two treatment studies and null results in an observational “high-risk” sample. Establishing evidence of temporal precedence within the treatment context is one way to determine if IU is a mechanism for effective treatment for OCD, but more methodologically rigorous long-term studies are needed to examine the developmental trajectories of IU and OCD. Although mean age of onset of OCD occurs in late adolescence (Brakoulias et al., 2017; Ruscio et al., 2010), a distinct early onset phenotype has been reported, demonstrating more severe symptoms and a faster progression from the first appearance of symptoms to full OCD (Anholt et al., 2013; Sobin et al., 2000). Longitudinal studies should ideally measure IU prior to this early onset OCD phenotype with extended follow-up across development.

#### **4.1.2. Experimental research on IU and OCD**

Research has shown that IU can be experimentally induced to influence OCD symptoms (Faleer et al., 2017; Geok et al., 2022). However, it is not yet known how long the effects of induced IU last, which has important implications for research on interventions aiming to change IU. For example, studies demonstrating that IU can be reduced within a brief experimental context do not inform potential therapeutic interventions, where lasting changes in IU are a specific goal. Thus, future studies should include follow-up measurement to determine if the effects of induced IU are enduring, even over a shorter period of time. In addition, only one study of experimentally induced IU included a control condition in which IU was not induced (Mosca et al., 2016); this study only found an effect on experimentally increased IU, but not experimentally decreased IU, compared to individuals who did not receive an IU induction. It is possible that IU is easier to increase than decrease within a brief experimental context, which

may have consequences on IU-focused interventions, suggesting that single-session interventions may be insufficiently powered to decrease IU. Though associations between changes in IU and changes in OCD symptoms within treatment studies have been reported (Pinciotti et al., 2020; Su et al., 2016; Wilhelm et al., 2015), the effects of specific interventions targeting IU have not been examined within clinical OCD samples.

#### **4.1.3. Multimodal measurement of IU**

In the majority of research reviewed, IU was most frequently measured by self-report. However, self-report measures are often biased toward socially desirable responding and can only capture aspects of behavior that individuals are consciously aware of and choose to report. Thus, researchers have examined correlates of IU using behavioral tasks and psychophysiological measurement to establish a broader nomological network that helps define the construct of IU. According to Cronbach and Meehl (1955), “A necessary condition for a construct to be scientifically admissible is that it occur in a *nomological net*, at least some of whose laws involve observables.” Given that the concept of IU, like many psychological traits, is not directly observable, researchers must establish reliable correlates that can be directly observed. Examining IU across multiple levels of analysis also allows for better understanding of the underlying mechanisms (Bilder et al., 2013; Cicchetti & Dawson, 2002).

One example of a framework for researching psychological constructs across multiple levels of analysis is the National Institute of Mental Health’s Research Domain Criteria Initiative (RDoC; Insel et al., 2010; Sanislow et al., 2010). Within this framework, the *Intolerance of Uncertainty Scale* has been identified as a self-report measure of Potential Threat, defined by NIMH as “activation of a brain system in which harm may potentially occur but is distant, ambiguous, or low/uncertain in probability, characterized by a pattern of responses such as

enhanced risk assessment (vigilance).” The current RDoC matrix

(<https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/constructs/rdoc-matrix>)

suggests a number of other tasks, measures, and circuits implicated in Potential Threat, including physiological measures such as potentiated startle and measures of adrenocorticotrophic hormone (ACTH), cortisol, and corticotropin releasing factor (CRF); neural circuits including the bed nucleus of the stria terminalis (BNST); and paradigms such as the no, predictable, and unpredictable threat task (NPU-threat task; Schmitz & Grillon, 2012), in which participants’ responses to predictable and unpredictable threat can be directly compared. The RDoC approach provides a framework by which IU can be modeled across multiple levels of analysis in future research efforts aimed at identifying etiological mechanisms that can be translated into clinical interventions for OCD (e.g., Fineberg et al., 2011).

#### **4.1.3.1. Behavioral measurement of IU**

Behavioral measures of IU have been developed to complement self-report measures. Ladouceur and colleagues (1997) proposed a probabilistic inference task in which participants were asked to draw as many marbles as they choose from a bag until they wanted to guess whether the bag contained mostly black or mostly white marbles. Participants also rated their level of certainty associated with their response. Within a moderately ambiguous condition (with a ratio of 85:15 white to black marbles or vice versa), the number of marbles drawn by a participant before they reached a decision was moderately correlated with self-reported IU (Spearman’s  $\rho = 0.43$ ). Jacoby and colleagues (2014) referred to this task as the Beads Task and found that, among individuals with anxiety disorders, self-reported IU was associated with the number of draws to reach a decision as well as distress experienced during the task, but only as measured by the OBQ-PC ( $r_s = .34-40$ ); correlations with the IUS-12 were not significant.

Given that individuals with OCD often repeat compulsions to achieve feelings of certainty (Rachman, 2002; Wahl et al., 2008), number of draws to a decision in this task could serve as a useful analogue to compulsive behavior.

Jacoby and colleagues continued to refine the Beads Task to increase its ecological validity by first incorporating a cold-pressor task as a threat for incorrect responses (Jacoby et al., 2016) and then recruiting a confederate who would receive the same “punishment” if the participant made an incorrect response (Jacoby et al., 2019). Results demonstrated a significant relationship ( $r = .48$ ) between Prospective IU and task-related distress in the solo version of the task, and a significant relationship ( $r = .31$ ) between Inhibitory IU and task-related distress when using a confederate, suggesting different potential processes by which IU impacts distress related to responsibility for harm to oneself or others. Responsibility for harm is a key belief underlying some presentations of OCD (e.g., Salkovskis, 1999; Wheaton et al., 2010); indeed, Jacoby and colleagues found that the number of draws to a decision in the partnered version of the Beads Task was moderately associated ( $r = .38$ ) with the unacceptable thoughts dimension of OCD symptoms. The Beads Task has also been validated for use with children, without a threatened punishment for incorrect responses (Osmanağaoğlu et al., 2021).

A related behavioral IU task is the PACT Anagram Task (Beadel et al., 2014; O’Bryan et al., 2021). Participants are briefly presented with anagrams and told that their ability to solve them is a measure of verbal intelligence. Participants select one of five potential answers, then rate their confidence in their response as well as their level of distress during the task. Participant distress was moderately associated with self-reported IU,  $r = .36$ , and obsessive-compulsive symptoms,  $r = .30$  (O’Bryan et al., 2021). Though both the Beads Task and PACT Anagram Task demonstrate significant correlations with self-reported IU, the associations were moderate

in strength and were based on participant-reported distress, rather than specific behavioral indicators. As noted by O’Byran and colleagues (2021), finding behavioral indicators of IU has been challenging, especially ones that can be standardized and performed in a laboratory context.

#### **4.1.3.2. Physiological correlates of IU**

The NPU-threat task (Schmitz & Grillon, 2012) can be used to assess physiological responses to uncertainty. During this task, participants see various cues that indicate either no threat (no shock will be given), predictable threat (shock will be given only when this cue appears), and unpredictable threat (shock could occur at any time during this block of the trial). Variations of the task use a startle probe such as a loud noise or aversive scream instead of a physical shock. Nelson and Shankman (2011) reported that individuals high in IU had an attenuated startle response during the unpredictable threat condition, with these results largely driven by Inhibitory IU. In contrast, Chin and colleagues (2016) found that individuals high in IU demonstrated increased startle potentiation when shocks followed a specific cue 50% of the time, but not in a less uncertain condition when shocks followed a cue 75% of the time. However, no version of the NPU-threat task has been used in an analogue or clinical OCD sample, an important direction for future research. Other physiological correlates of IU during unpredictable threat contexts include event-related potentials, including an enhanced tactile P300 response (Ferry & Nelson, 2021) and error-related negativity (ERN; Jackson et al., 2016). Interestingly, Jackson and colleagues identified different electrophysiological responses associated with the Inhibitory and Prospective components of IU, such that Prospective IU was associated with a larger error-related negativity response and Inhibitory IU was associated with smaller error-related negativity response. This finding suggests additional utility in exploring differences among approach and avoidance strategies in response to uncertainty. Although

Jackson and colleagues note that an enhanced ERN has been found in OCD, no studies to date examine the associations between ERN, IU, and OCD symptoms, or the association between ERN and IU in an OCD sample.

Other studies have examined the relation between IU and psychophysiological responding within the context of threat conditioning and extinction paradigms. In a recent meta-analysis, Morriss and colleagues (2021) found a consistent small to medium association (Hedges'  $g = 0.28-0.29$ ) between IU and delayed extinction to threat cues as indexed by skin conductance responses, suggesting that individuals high in IU had difficulty updating learned threat associations (“This cue no longer means there will be a shock, but I can’t be too careful”). In addition, this association was robust after controlling for trait anxiety, suggesting evidence of a specific association between delayed threat extinction and IU. Thus, within multiple threat paradigms, there is evidence for physiological response patterns associated with IU. Similar studies have found evidence of impaired updating of learned threat associations, as measured by skin conductance, between individuals with OCD and healthy individuals (Apergis-Schoute et al., 2017); again, however, studies examining associations between IU and physiological responses to threat cues have not been conducted within OCD samples.

#### **4.1.4. Expanding the nomological network of IU and OCD symptoms**

To understand IU and its role in OCD, the nomological network in which it occurs should be more robustly defined (Cronbach & Meehl, 1955). In other words, understanding IU as a cognitive vulnerability for OCD requires situating IU along with other relevant constructs, such as negative affectivity and behavioral inhibition, to clarify points of convergence and divergence. For example, Hong and Lee (2015) found that the IUS demonstrated convergent validity with relevant cognitive vulnerabilities (e.g., neuroticism/negative affectivity). They also established

evidence of discriminant validity between prospective and inhibitory IU, with inhibitory IU demonstrating stronger associations with fear of negative evaluation, anxiety sensitivity, looming cognitive style, and rumination compared to prospective IU. From a nomological network perspective, this distinction between inhibitory and prospective IU suggests etiological pathways that may differ across disorders and differentially respond to treatment, with preliminary evidence suggesting that inhibitory IU may be more closely related to other vulnerabilities for psychopathology and thus may be more maladaptive.

Although Hong and Lee (2005) examined relations between IU and symptoms of psychopathology within a nomological network, their study did not include a measure of OCD symptoms. Understanding how OCD symptoms fit into the nomological network of IU, and whether there are differential relations between IU and OCD and IU and other psychological disorders, will be an important next step in understanding the broader construct of IU and the extent to which its association with OCD symptoms is distinct or a function of other related constructs. Continuing to build out this nomological network across multiple levels of analysis will also improve our understanding of the underlying mechanisms of IU within the context treatments for emotional disorders. For example, Hong and Lee (2015) speculate that, given differential relations between prospective and inhibitory IU and various psychological disorders, individuals with GAD and OCD may benefit most from interventions that target prospective IU, such as cognitive restructuring to change threat perceptions, while individuals with social anxiety, panic disorder, and depression with greater inhibitory IU may benefit more from active engagement with uncertainty, perhaps through exposure-based interventions that encourage active approach. Additional dismantling studies may provide insights into the utility of specific cognitive and behavioral interventions on IU, which could reasonably differ by disorder or



individual levels of prospective vs. inhibitory IU. Although incorporating exposure to uncertainty in a variable exposure intervention did not produce significant improvements compared to standard exposure in an analogue OCD sample, these results may differ when applied to other fear-based disorders in which inhibitory IU is a more prominent feature.

## **4.2. Conclusions**

As explored above, there is consistent evidence of a strong association between changes in IU and changes in OCD symptoms, as well as associations between cognitive-behavioral treatments for OCD and changes in IU. However, evidence for a *specific* association (e.g., other proposed mechanisms do not account for therapeutic change after controlling for changes in IU), has not yet been established. Furthermore, to establish that increased tolerance of uncertainty is a mechanism within effective treatment for OCD, additional evidence is needed. For example, Kazdin (2007) outlines the following criteria to identify a purported mechanism of a particular intervention: a strong, specific association; consistency across multiple studies and samples; direct manipulation of the mechanism; evidence of temporal precedence within the intervention context; evidence of a gradient or dose-response effect; and plausibility or coherence with the broader scientific literature. Given the evidence for IU as a cognitive vulnerability for OCD, as well as clinical observations of the importance of learning to tolerate uncertainty for individuals with OCD, increased tolerance of uncertainty may be an important mechanism of effective OCD treatment. Future research that builds on the findings presented in Chapters 2 and 3 will be well-positioned to advance current knowledge on IU that will inform models of the development and evidence-based treatment of OCD.

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**Appendix Table 1.** Goodness of fit indices for primary models ( $N = 1280$ ) from Knowles et al., 2022.

Model	$\chi^2$	N	df	CFI	TLI	RMSEA	
						Estimate	90% CI
Measurement models							
Total IU	7354.50	1244	2344	.870	.858	.041	.040-.043
Inhibitory IU	661.65	1244	335	.982	.976	.028	.025-.031
Prospective IU	1647.89	1244	729	.951	.943	.032	.030-.034
TI-TV models							
Total IU	7406.77	1244	2362	.869	.858	.041	.040-.043
Inhibitory IU	713.47	1244	353	.980	.975	.029	.026-.032
Prospective IU	1709.16	1244	747	.949	.941	.032	.030-.034
TI-TV models with OCD symptoms							
<i>Total IU</i>							
Wave 1	7571.60	1244	2724	.865	.855	.038	.037-.039
Wave 2	7461.06	1244	2724	.865	.855	.037	.036-.038
Wave 3	7381.69	1244	2724	.866	.856	.037	.036-.038
Wave 4	7338.61	1244	2724	.867	.857	.037	.036-.038
Wave 5	7344.34	1244	2724	.866	.856	.037	.036-.038
Wave 6	7325.11	1244	2725	.867	.857	.037	.036-.038
<i>Inhibitory IU</i>							
Wave 1	932.50	1251	505	.970	.964	.026	.023-.029
Wave 2	857.64	1244	504	.974	.969	.024	.021-.026
Wave 3	815.21	1244	504	.976	.972	.022	.019-.025
Wave 4	773.84	1244	504	.979	.975	.021	.018-.024
Wave 5	794.74	1244	504	.978	.974	.022	.019-.024
Wave 6	781.82	1244	505	.978	.975	.021	.018-.024
<i>Prospective IU</i>							
Wave 1	2087.57	1251	959	.940	.933	.031	.029-.032
Wave 2	2011.42	1244	958	.942	.935	.030	.028-.032
Wave 3	1949.51	1244	958	.945	.938	.029	.027-.031
Wave 4	1899.36	1244	958	.947	.940	.028	.026-.030
Wave 5	1913.43	1244	958	.946	.939	.028	.026-.030
Wave 6	1904.08	1244	959	.946	.940	.028	.026-.030

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TI-TV models with Depression

*Total IU*

Wave 1	7967.95	1280	2877	.873	.864	.037	.036–.038
Wave 2	7718.79	1258	2876	.872	.863	.037	.036–.038
Wave 3	7640.19	1257	2876	.874	.865	.036	.035–.037
Wave 4	7734.51	1256	2876	.869	.860	.037	.036–.038
Wave 5	7648.31	1255	2876	.872	.862	.036	.035–.037
Wave 6	7529.54	1253	2877	.874	.865	.036	.035–.037

*Inhibitory IU*

Wave 1	1237.81	1280	574	.963	.957	.030	.028–.032
Wave 2	1065.73	1258	573	.969	.964	.026	.024–.029
Wave 3	1019.45	1257	573	.972	.967	.025	.022–.027
Wave 4	1052.33	1256	573	.969	.963	.026	.023–.028
Wave 5	100.05	1255	573	.972	.967	.024	.022–.027
Wave 6	895.75	1253	574	.978	.975	.021	.018–.024

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*Prospective IU*

Wave 1	2521.56	1280	1052	.937	.929	.033	.031–.035
Wave 2	2309.28	1258	1051	.940	.933	.031	.029–.033
Wave 3	2202.71	1257	1051	.945	.938	.030	.028–.031
Wave 4	2303.55	1256	1051	.938	.931	.031	.029–.033
Wave 5	2202.47	1255	1051	.943	.937	.030	.028–.031
Wave 6	2116.47	1253	1052	.947	.940	.028	.027–.030

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Total IU → OCD, controlling for latent depression

Wave 1	8925.91	1280	3274	.868	.859	.037	.036–.038
Wave 2	8547.39	1258	3272	.867	.858	.036	.035–.037
Wave 3	8353.80	1257	3272	.870	.862	.035	.034–.036
Wave 4	8357.79	1256	3272	.867	.858	.035	.034–.036
Wave 5	8298.31	1255	3272	.869	.860	.035	.034–.036
Wave 6	8188.10	1253	3273	.870	.862	.035	.034–.036

Inhibitory IU → OCD, controlling for latent depression

Wave 1	1782.28	1280	761	.949	.942	.032	.030–.034
Wave 2	1508.50	1258	759	.957	.952	.028	.026–.030
Wave 3	1395.41	1257	759	.963	.958	.026	.024–.028
Wave 4	1343.31	1256	759	.964	.959	.025	.023–.027
Wave 5	1331.89	1255	759	.965	.960	.025	.022–.027
Wave 6	1231.37	1253	760	.970	.966	.022	.020–.024



Prospective IU → OCD, controlling for latent depression							
Wave 1	3201.50	1280	1299	.926	.918	.034	.032–.035
Wave 2	2887.71	1258	1298	.929	.922	.031	.030–.033
Wave 3	2685.80	1257	1297	.938	.931	.029	.028–.031
Wave 4	2685.87	1256	1297	.935	.928	.029	.028–.031
Wave 5	2620.81	1255	1297	.938	.931	.029	.027–.030
Wave 6	2543.11	1253	1298	.940	.934	.028	.026–.029
Total IU → Depression, controlling for latent OCD							
Wave 1	8925.67	1280	3274	.868	.859	.037	.036–.038
Wave 2	8544.78	1258	3272	.867	.858	.036	.035–.037
Wave 3	8348.13	1257	3272	.870	.862	.035	.034–.036
Wave 4	8352.15	1256	3272	.867	.859	.035	.034–.036
Wave 5	8301.28	1255	3272	.868	.860	.035	.034–.036
Wave 6	8189.91	1253	3273	.870	.862	.035	.034–.036
Inhibitory IU → Depression, controlling for latent OCD							
Wave 1	1781.38	1280	761	.949	.943	.032	.030–.034
Wave 2	1506.71	1258	759	.958	.952	.028	.026–.030
Wave 3	1392.29	1257	759	.963	.958	.026	.024–.028
Wave 4	1339.29	1256	759	.964	.960	.025	.022–.027
Wave 5	1336.01	1255	759	.965	.960	.025	.022–.027
Wave 6	1233.37	1253	760	.970	.966	.022	.020–.025
Prospective IU → Depression, controlling for latent OCD							
Wave 1	3205.19	1280	1299	.925	.918	.034	.032–.035
Wave 2	2878.77	1258	1297	.930	.923	.031	.030–.033
Wave 3	2682.08	1257	1297	.938	.931	.029	.028–.031
Wave 4	2684.53	1256	1297	.935	.928	.029	.028–.031
Wave 5	2619.97	1255	1297	.938	.931	.029	.027–.030
Wave 6	2541.30	1253	1298	.940	.934	.028	.026–.029

*Note.* All chi-square tests significant at  $p < .001$ . CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation; IU = intolerance of uncertainty; OCD = obsessive-compulsive disorder; TI = time-invariant; TV = time-varying

**Appendix Table 2.** Parameter estimates for total IU TI-TV models from Knowles et al., 2022.

Parameter	Total IU	
	Estimate	SE (range)
Factor Variance		
Total variance	.953	
TI factor variance	.782	.045
TV factor variance	.171	.012
Proportion of variance attributed to TI factor	.821	(.821–.860)
Proportion of variance attributed to TV factor	.179	(.140–.179)
TV factor stability		
Unstandardized	.241	.033
Standardized	.241–.248	
Indicator variable factor loadings onto the latent IU factor		
Unstandardized	0.574–1.010	.020–.027
Standardized	.410–.869	
Loadings of the latent IU factor onto the TI factor		
Unstandardized	Fixed at 1.0	
Standardized	.906–.923	
Loadings of the latent IU factor onto the TV factor		
Unstandardized	Fixed at 1.0	
Standardized	.384–.424	

*Note:* IU = Intolerance of uncertainty, TI = Time invariant, TV = Time varying. All estimates significant,  $p < .001$ . Estimates are based on wave 1 variance estimates; range reflects slight variability in TV variance estimates across waves. Proportion of variance =  $\frac{var(TI)}{var(TI)+var(TV)}$ .

**Appendix Table 3.** Regression models with total IU from Knowles et al., 2022.

Dependent variable	Wave	TI factor			TV factor					
		B	SE (B)	$\beta$	B	SE (B)	$\beta$			
Total IU $\rightarrow$ OCD Symptoms										
OCD Symptoms	1	1.022	.084	.583***	0.172	.230	.046			
	2	0.957	.102	.604***	0.262	.213	.077			
	3	0.953	.123	.546***	0.749	.272	.195**			
	4	0.944	.109	.627***	-0.015	.204	-.004			
	5	0.901	.109	.633***	0.666	.216	.200**			
	6	0.893	.126	.598***	0.362	.322	.101			
Total IU $\rightarrow$ Depressive Symptoms										
Depressive Symptoms	1	0.434	.028	.544***	0.115	.082	.067			
	2	0.363	.029	.519***	0.252	.087	.165**			
	3	0.400	.035	.506***	0.344	.113	.193**			
	4	0.395	.034	.534***	0.300	.112	.175**			
	5	0.456	.038	.544***	0.062	.138	.031			
	6	0.493	.040	.612***	0.253	.119	.127*			
Total IU $\rightarrow$ OCD Symptoms controlling for Depressive symptoms										
OCD Symptoms		B	SE (B)	$\beta$	B	SE (B)	$\beta$	B	SE (B)	$\beta$
	1	0.920	.094	.529***	0.127	.219	.034	0.229	.093	.105*
	2	0.807	.102	.524***	0.176	.204	.052	0.383	.133	.173**
	3	0.972	.128	.570***	0.757	.274	.196**	-0.060	.109	-.028
	4	0.935	.118	.636***	-0.039	.202	-.011	-0.011	.123	-.005
	5	0.880	.123	.629***	0.655	.218	.197**	0.035	.106	.021
6	0.919	.146	.631***	0.373	.317	.103	-0.076	.165	-.042	
Total IU $\rightarrow$ Depressive Symptoms controlling for OCD symptoms										
Depressive Symptoms		B	SE (B)	$\beta$	B	SE (B)	$\beta$	B	SE (B)	$\beta$
	1	0.380	.038	.476***	0.095	.079	.055	0.055	.022	.119**
	2	0.275	.044	.395***	0.225	.083	.148**	0.092	.034	.203**
	3	0.413	.045	.522***	0.351	.116	.197**	-0.013	.025	-.029
	4	0.386	.053	.523***	0.300	.113	.175**	0.009	.037	.018
	5	0.450	.062	.537***	0.059	.135	.029	0.007	.049	.011
6	0.511	.063	.634***	0.264	.118	.132*	-0.020	.048	-.037	

*Note.* Top: Regression of latent OCD and Depressive Symptoms at six waves onto TI and TV latent variables extracted from IU. Bottom: Regression of latent OCD Symptoms controlling for latent Depressive Symptoms and latent Depressive Symptoms controlling for OCD Symptoms at six waves onto TI and TV latent variables extracted from IU.