A TAXOMETRIC ANALYSIS OF PANIC DISORDER

A Thesis
by
CHRISTIAN A. HALL

Submitted to the School of Graduate Studies
at Appalachian State University
in partial fulfillment of the requirements for the degree of
MASTER OF ARTS

May 2021
Department of Psychology

A TAXOMETRIC ANALYSIS OF PANIC DISORDER

A Thesis
by
CHRISTIAN A. HALL
May 2021

APPROVED BY:

_____
Joshua J. Broman-Fulks, Ph.D.
Chairperson, Thesis Committee

_____
Shawn M. Bergman, Ph.D.
Member, Thesis Committee

_____
Christopher J. Holden, Ph.D.
Member, Thesis Committee

_____
Rose Mary Webb, Ph.D.
Chairperson, Department of Psychology

_____
Mike McKenzie, Ph.D.
Dean, Cratis D. Williams School of Graduate Studies

Abstract

A TAXOMETRIC ANALYSIS OF PANIC DISORDER

Christian A. Hall
B.S., Virginia Polytechnic Institute and State University
M.A., Appalachian State University

Chairperson: Joshua J. Broman-Fulks, Ph.D.

Panic-related suffering is associated with high individual costs and strain on medical resource utilization. Cognitive-behavioral interventions for panic disorder are effective, but obtaining a diagnosis often precludes access to such treatments. Evidence-based models suggest that panic disorder is a multi-dimensional construct, yet panic disorder is diagnosed categorically (i.e., "you have it, or you don't") in modern diagnostic manuals. Taxometric analyses, which test the dimensional or categorical latent structure of constructs, have consistently revealed dimensional latent structures when applied to other anxiety disorders and panic-related processes, but these analyses have never been applied to panic disorder. In this study, seven theoretically-relevant indicators of panic disorder were subjected to three nonredundant taxometric procedures to test the latent structure of panic disorder, and simulated comparison plots and objective fit indices were evaluated. The collective results provided consistent empirical support for a dimensional model of panic disorder. The implications of these findings for the measurement, assessment, diagnosis, and treatment of panic disorder are discussed.

Keywords: panic disorder, taxometric, latent structure, panic attack, anxiety

Acknowledgements

First, I would like to thank Dr. Josh Broman-Fulks for encouraging me to pursue scientific endeavors, giving me room to make mistakes along the way, and being willing to watch me grow from them as a result.

I would also like to thank to my committee members, Dr. Chris Holden and Dr. Shawn Bergman, both for their willingness to attend to this project and for their thoughtful contributions throughout the process.

Thank you as well to the faculty and graduate students of the Department of Psychology who, with patience and warmth, have challenged me to improve on nearly every front.

Finally, I am deeply appreciative of all of the past and present members of the Anxiety Lab at Appalachian State University, especially Elijah and Esther, for their invaluable ideas, inspiration, and unwavering support.

Table of Contents

List of Tables

List of Figures

A Taxometric Analysis of Panic Disorder

Panic attacks (PA) are conceptualized as discrete episodes of intense fear or discomfort that develop abruptly and peak in severity within minutes (American Psychiatric Association [APA], 2013). A variety of somatic and cognitive "symptoms" characterize PAs, including palpitations, sweating, shortness of breath, chest pain, lightheadedness, and fear of loss of control or death, and can range widely between individual PAs. PAs can occur with or without an identifiable cause. When a known stimulus (i.e., a "trigger") precipitates a PA, that PA is considered to be cued, whereas PAs that occur in absence of an identifiable stimulus are considered to be uncued. With the exception of an initial PA, most PAs are expected and precipitated by identifiable cues (Street et al., 1989). Beyond this distinction, expected and unexpected PAs do not demonstrably differ in either symptomatology (Kenardy & Taylor, 1999) or time of onset (Meuret et al., 2011).

PAs are thought to be normal and adaptive responses to perceived imminent threat, occurring in an estimated 13.2% – 28.3% of individuals at some point in their lifetime (e.g., de Jonge et al., 2016; Kessler et al., 2006). However, recurrent PAs are a risk factor for psychopathology, including panic disorder (PD) and other emotional and substance use disorders (Baillie & Rapee, 2005; Goodwin et al., 2004; Kinley et al., 2011). Among the estimated 8.8% of individuals who will experience recurrent PAs throughout their lives, only a fraction are expected to develop PD: estimates for the lifetime prevalence of PD in U.S. adults range from 1.6% – 4.8% (de Jonge et al., 2016; Kessler et al., 2006, 2012). In the small percentage of the population that develops clinically diagnosable PD, recurring PAs become accompanied by additional symptoms, including anxiety about future attacks and maladaptive attempts to manage or avoid those attacks.

PD is a mental disorder characterized in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5; APA, 2013) by the experience of recurrent unexpected panic attacks (PAs) accompanied by avoidance of perceived panic triggers and/or other maladaptive behaviors. The average age of onset of PD is estimated to occur from early-to-middle adulthood, later than most other anxiety disorders (Lijster et al., 2017; Weissman, 1997). The lifetime prevalence of PD is estimated to range from 1.6% – 4.8%, while its 12-month prevalence in adult and adolescent populations is estimated to be between 0.7% – 3.1% (Bandelow & Michaelis, 2015). Rates of PD are approximately twice as high for females as males, and sex differences in the prevalence and presentation of PD mirror those found in other anxiety disorders (Bandelow & Michaelis, 2015; Bekker & van Mens-Verhulst, 2007; Donner & Lowry, 2013). Women with PD bear an increased burden of illness and risk of comorbidities with alcohol dependence, depressive disorder, and personality disorders than men with PD (Chang et al., 2019; Kelly et al., 2006; McLean et al., 2011).

Compared to other anxiety disorders, PD is one of the least prevalent (Somers et al., 2006) and yet most severe and intensely experienced, with individual and societal burdens higher than those of many other emotional disorders (Batelaan et al., 2007; Konnopka et al., 2009). An estimated 45.3% of individuals with PD seek help with their experiences through mental health services, higher than any other emotional disorder (Mackenzie et al., 2011). However, in addition to concerns related to cost of treatment and lack of insurance coverage for treatment (Chartier-Otis et al., 2010), many people with symptoms of PD misperceive it as being a medical illness and fail to seek appropriate help (Coles & Coleman, 2010; Craske et al., 2005). If they manage to overcome barriers to treatment, patients with PD can receive effective treatments ranging from cognitive-behavioral therapy to pharmacological interventions (Bandelow & Baldwin, 2020;

Otto et al., 2010; Sánchez-Meca et al., 2010; Schwartze et al., 2017). Though PD is often chronic and, in approximately one third of cases, treatment-resistant (Chen & Tsai, 2016), a better understanding of the construct may allow clinicians to assess, diagnose, and treat PD with greater efficacy.

A wide array of risk factors have been implicated in the etiology and maintenance of PD, including female sex (Jalnapurkar et al., 2018), neuroticism (Forstner et al., 2019; Naragon-Gainey & Watson, 2018; Zugliani et al., 2017), anxiety sensitivity (Jurin & Biglbauer, 2018; Smits et al., 2019), experiential avoidance (Kämpfe et al., 2012; Spinhoven et al., 2014), intolerance of uncertainty (Carleton et al., 2013), and individual differences in fear conditioning (Lonsdorf & Merz, 2017; Pittig et al., 2018). Differentiating between emotional disorders can be particularly challenging given the high rates of comorbidity between such disorders and other diagnoses (Tilli et al., 2012; Zimmerman et al., 2008). For example, PD is often comorbid with other anxiety disorders (Goldstein-Piekarski et al., 2016), agoraphobia (Greene & Eaton, 2016), major depressive disorder (Dold et al., 2017), somatic symptom disorder (Newby et al., 2017), substance use disorders (e.g., Fullana et al., 2019), personality disorders (Navinés et al., 2016), medical illness (Meuret et al., 2017), and suicidality (Tietbohl-Santos et al., 2019), to list but a few such associations.

Finally, the wide range of overlapping symptoms and risk factors associated with PD and other anxiety disorders makes it difficult to separate these disorders into theoretically and clinically relevant categories (Allsopp et al., 2019; Asmundson et al., 2014). Common symptoms of anxiety disorders include worry, fatigue, restlessness, difficulty concentrating, muscle tension, sleep issues, irritability, avoidance behaviors, and, notably, PAs (APA, 2013). Conceptually, one of the primary distinctions made between PD and other anxiety disorders is that PD is associated

with recurrent unexpected PAs, whereas PAs associated with most other anxiety disorders tend to be provoked (Street et al., 1989). In addition, the diagnosis of PD requires that PAs not be better explained by another disorder, physical condition (e.g., cardiorespiratory issues), or substance use. For example, recurrent unexpected PAs experienced in response to cocaine or amphetamine intake would not qualify as meeting criteria for PD unless PAs were also experienced in the absence of substance use.

Panic and Anxiety

The sudden and intense occurrence of fear-based panic is often conceptualized as being a distinct type of experience from future-orientated worries or anxiety (Bouton et al., 2001; LeDoux & Pine, 2016). Supporting this theory, some have observed that the autonomic arousal associated with the panic response is present in PD, but not in most other disorders predominantly characterized by anxiety or worry (e.g., generalized anxiety disorder [GAD]; Brown & McNiff, 2009), and others have found that cardio-respiratory and vestibular anxiety symptoms were more closely associated with outpatients diagnosed with PD than with outpatients diagnosed with other anxiety disorders (Kenardy et al., 1992). Additionally, different developmental risk factors have been identified in the etiologies of PD and other anxiety disorders, suggesting that environmental factors may also play a role in differentiating between anxiety and panic. For example, Newman et al. (2016) found that PD and other phobic disorders (e.g., specific phobia, agoraphobia) were associated with childhood separation anxiety disorder, while GAD was associated with childhood agoraphobia and avoidant parental attachment. PD may also be differentiated from other anxiety disorders by its association with early childhood experiences of abuse and depression (Raskin et al., 1982).

Attempts to subtype PAs have revealed symptom clusters associated with cardio-respiratory, somatic, and cognitive features of PAs (e.g., Cox et al., 1994; Meuret et al., 2006; Schmidt et al., 2002), but more recent evidence challenges these findings and suggests that well-validated PA subtypes have yet to be identified (Kircanski et al., 2011). Instead, PAs may be best represented as an acute stress response that induces "fight-or-flight" behaviors, modulated by serotonin in response to an imminent threat (Del-Ben & Graeff, 2009; Johnson et al., 2004, 2014). The body's ready recruitment of the central nervous system during experiences of panic is thought to represent a distinct reaction to stress (van Oort et al., 2017).

Predatory Imminence Theory (PIT; Fanselow, 1994; Fanselow & Lester, 1988), or threat-imminence theory, offers an evolutionary explanation for the emergence of these distinct emotional systems by attributing anxiety and panic to opposite ends of a timeline of predatory threat responses. According to PIT, feelings of anxiety and related behaviors emerge in a pre-encounter with some perceived threat as an adaptive alarm mechanism, while panic uniquely manifests once the threat is imminent to engage the body for fight-or-flight defensive behavior. PIT has been adopted by theoretical models of panic disorder for both its explanatory and predictive power (e.g., Bouton et al., 2001; Hamm et al., 2014).

Neurobehavioral findings appear to lend support to PIT (see Perusini & Fanselow, 2015). For example, evidence suggests that serotonin-rich dorsal and medial raphe nuclei (DR and MR) in the brain stem signal to upstream regions of the brain to facilitate avoidance behaviors characteristic of both anxiety and panic (Zangrossi et al., 2020). However, the DR also innervates the amygdala and the dorsal periaqueductal gray area (dPAG) in the midbrain to provide additional signaling for explicit fight-or-flight behaviors (e.g., escape) representative of the circa-strike features of panic (Pobbe et al., 2011; Zangrossi & Graeff, 2014). Additionally, the

differential impact of reproductive hormones on these systems predicts some of the widely observed and reported sex differences present in PD and other anxiety disorders (Donner & Lowry, 2013). For example, decreases in serotonergic activity in the DR of female rats was observed after exposure to a stressful task, and a similar effect was observed in the MR of male rats (Domínguez et al., 2003). Other studies suggest that the expression of corticotropin releasing hormone receptors typically produced in the DR to inhibit connections to the dPAG and basolateral amygdala may be reduced in females during estrus (Donner & Lowry, 2013). These converging lines of research suggest that the distinction between panic and anxiety may usefully differentiate between PD and other anxiety disorders, but not necessisarily between PD and normal experiences with panic.

Etiology of Panic Disorder

Theories from multiple domains have been proposed to explain the causal processes behind PD. Etiological models of PD pathogenesis spanning biological, behavioral, and cognitive levels of analysis will be briefly reviewed.

Biological Theories

Genetic analyses and heritability studies of PD suggest that innate predispositions to PD exist across sex and aggregate within families, though the exact genes implicated in this association are still largely unknown (Hettema et al., 2001; Howe et al., 2016). Furthermore, Iurato et al. (2017) found inconclusive evidence for the role of epigenetic DNA methylation in PD patients compared to controls. Differences in cytokine signaling have been observed in patients with PD compared to both healthy controls (Quagliato & Nardi, 2018) and patients with GAD (Zou et al., 2020). PD has also been associated with dysfunction in a variety of neurotransmitter systems, including those regulating serotonin (Zangrossi et al., 2020),

norepinephrine (Lee et al., 2005), and opioids (Graeff, 2017), though speculative associations with others (e.g., GABAergic systems) are more tenuous (e.g., Schür et al., 2016).

In their neuroanatomic theory, Gorman and colleagues propose that a "fear network" in the brain, comprised of the amygdala, hippocampus, and medial prefrontal cortex, mediates the relationship between environmental factors (e.g., early childhood trauma and contextual learning processes) and panic (Gorman et al., 1989, 2000). In this model, those with PD are hypothesized to have overly sensitive fear networks and fearfully overreact to otherwise non-threatening stimuli compared to those without PD. Though the exact nature of this fear network sensitivity was never explicitly articulated, Gorman et al. (2000) suggest that it may encompass heightened sensitivities to CO_2, hypocapnia, separation, death-related thoughts, and interoceptive sensations, among previously mentioned individual differences in genetics and neurotransmission. Evidence affirms that PD patients may indeed demonstrate respiratory abnormalities (Grassi et al., 2013, 2014) and heightened interoceptive sensitivity (Domschke et al., 2010) when compare to healthy controls.

Behavioral Theories

Proponents of behavioral theories suggest that classical and operant conditioning mechanisms play a primary role in the development of clinical forms of anxiety, including PD (Lissek & Grillon, 2010; Nees et al., 2015). In behavioral paradigms, panic acts as a powerful unconditioned response to perceived threats in the environment, manifesting after the presentation of initial apprehension and anxiety towards a threat (Davey, 1992; Wolpe & Rowan, 1988). Learning theories of PD suggest that the salience of PAs enables external and internal contextual cues to become readily conditioned to the panic response, such that otherwise

innocuous stimuli become conditioned triggers for future attacks (Bouton et al., 2001; Mineka & Oehlberg, 2008).

As predicted by these theories, experimental studies have shown that interoceptive cues can become conditioned to the panic response, in addition to external cues (Acheson et al., 2007; de Cort et al., 2012, 2017; van Diest, 2019). Crucially, conditioned fear responses can overgeneralize to stimuli that are associated with previously conditioned stimuli, but that have never been paired with the fear response directly (Lissek et al., 2010, 2014; Neueder et al., 2019). This contextual fear conditioning is argued to be fundamental to the etiology and maintenance of PD, creating a "fear of fear" that conditions anxiety and other panic symptoms to the panic response itself (Bouton et al., 2001; Hamm et al., 2014). Finally, behavioral theories argue that fear and avoidance behaviors in PD are maintained by attempts to avoid, control, or suppress experiences with PAs and related stimuli. While such behaviors may result in the momentary abatement of fear and anxiety, they do little to reduce the frequency or intensity of such experiences in the long term (Barlow, 2004). Rather, the negative reinforcement of avoidance and escape behaviors leads to functional impairment and a restricting of behavioral repertoires (Hayes et al., 2011).

Cognitive Theories

Beck (1988) and Clark (1986) were the first to synthesize a cognitive model of PD. According to their theory, pre-anxious arousal sensations and cognitions arise in response to both external and internal stimuli, including thoughts, feelings, and interoceptive sensations (e.g., momentary chest pain). Catastrophic misinterpretations of these sensations increase apprehension of potential threats, which, in turn, engages threat response systems that generate additional anxiety-related stimuli. This process creates a "vicious cycle" that, in absence of

effective coping or reappraisal strategies, can lead to PD. This model was expanded by Casey et al. (2004), who borrowed from Bandura's (1988) self-efficacy theory to include low panic self-efficacy (or the individual perceptual capacity to deal with panic-related threats) as an antecedent to catastrophic misinterpretation.

Classification of Panic Disorder

PD is classified in the DSM-5 as a distinct diagnostic entity within the broader scope of anxiety disorders (APA, 2013). In fact, this sort of categorical classification extends to all mental health disorders listed in the manual. Proponents of categorical classification systems for mental disorders argue that communication about mental health issues, treatment decision making, and predictions about prognosis benefit from the clinical utility imparted from classification (First, 2010). Some claim that even when diagnosing dimensional constructs, categorical thresholds maximizing both sensitivity and specificity of prediction may still be useful for quickly and simply achieving the previously listed goals (Kamphuis & Noordhof, 2009).

However, others arguing against the DSM's categorical diagnostic system have remarked on its poor diagnostic reliability (e.g., Kalk & Young, 2017), inability to parse artifactual comorbidity from true comorbidity (e.g., Clark et al., 2017) and reliance on often unfounded causal etiologies for psychiatric disorders (e.g., Brendel, 2001; Pies, 2009). Indeed, Mayes and Horwitz (2005) suggest that the decision to formalize the categorization of mental disorders was influenced more by the political and economic pressures facing the field of psychiatry at the time than by compelling and available scientific evidence. A growing contingent of clinicians and researchers favoring dimensional diagnostic systems maintain that available neurobehavioral and transdiagnostic evidence refutes categorical conceptualizations of mental illness (e.g., Helzer et al., 2009) and argue that dimensional models of psychopathology demonstrate greater predictive

utility for disorder-related impairment than categorical models (e.g., Bjelland et al., 2009). In addition, dimensional models neither restrict nor reduce the information available to clinicians, making them preferable to categorical models insofar as they are interpretable (Kraemer, 2007).

The categorical approach to the classification of PD presented in the DSM-5 is potentially problematic for a variety of reasons. First, dichotomous criteria, like the expected/unexpected nature of recurring PAs, do not sufficiently discriminate between PD and non-clinical panic by themselves. For example, Wilson et al. (1992) discovered comparable rates of unexpected PAs in a sample of inpatients diagnosed with PD (21.4%) and a nonclinical sample of undergraduate students (17.8%). Such criteria also struggle to differentiate between PD and other anxiety disorders, as some have observed comorbidity rates as high as 98% between PD and other emotional and substance use disorders (Tilli et al., 2012). Second, continuous indicators comprising other diagnostic criteria for PD require that clinicians identify where along dimensions – or in which combinations – clinical patterns of disorder are expressed. For example, a diagnosis of PD requires that PAs be accompanied by four or more symptoms that cause "persistent" concerns about experiencing additional attacks or "a significant maladaptive change in behavior related to the attacks" (APA, 2013). These criteria can result in equivalent diagnoses based on totally disparate symptom profiles (Roberson-Nay & Kendler, 2011). The remaining diagnostic criteria are relatively ill-defined, specifying neither global nor individualized thresholds for "persistence" or "clinical significance." In combination with the complex and multifaceted etiology and expression of PD, these issues offer little guidance for anchoring accurate thresholds between clinical and normal levels of panic.

The debate between categorical and dimensional models of PD is consequential for those attempting to seek treatment for PA-related suffering and impairment. Failing to meet the DSM-5

criteria for PD diagnosis might mean the difference between receiving treatment and going untreated (Magruder & Calderone, 2000; Pierre, 2010). For example, it is common for prospective patients to report recurrent, unexpected panic attacks that generate persistent concerns and significant behavioral changes, yet only experience three panic-related symptoms. So called "subthreshold" cases of PD, where some, but not all, diagnostic criteria for PD are met, are estimated to affect between 1.9 – 2.7% of the general population (Batelaan et al., 2006; Skapinakis et al., 2011). In absence of empirically-derived evidence of the latent structure of PD, these cases represent either a substantial margin of error in the accurate assessment of pathological PD (assuming a categorical structural hypothesis), or a potential cohort of people suffering from PD symptoms that may be arbitrarily denied treatment by insurance companies who rely on DSM diagnostic status to determine service coverage (assuming a dimensional structural hypothesis).

Once criteria for clinically significant impairment has been empirically established, an understanding of latent structure may also inform which treatment goals are most appropriate. In a categorical model of PD, treatment efficacy would be defined by a reduction in symptoms below disorder threshold levels. For example, clients previously endorsing DSM-5 criteria for PD diagnosis who, through treatment, no longer meet those criteria would be considered "treated" or "cured" within the categorical model. However, dimensional models of PD imply more person-specific treatment goals and necessitate a relative and functional assessment of treatment progress. In dimensional models, tolerance or acceptance of fear triggers and PAs may be emphasized over the reduction or elimination of these experiences (e.g., Abramowitz et al., 2019; Hayes et al., 2011).

The measurement and classification of PD may also be informed by evidence of its latent structure. In theory, the aim of assessment and diagnostic measures for categorical constructs would be to sort individuals into their respective categories with as much accuracy and efficiency as possible. In contrast, the general aim of assessment instruments for continuous constructs would be to precisely identify an individual's location along all relevant dimensions. For example, a dimensional assessment tool for panic might aim to capture the extent to which an individual fears anxiety-related sensations (i.e., AS), the relative duration and intensity of their experiences with somatic sensations (e.g., rather than whether one experiences a PA or not), the frequency of avoidance or escape behaviors, and/or the level of panic-related distress and impairment experienced by the individual.

The economic burdens associated with PD are under-researched compared to those of other anxiety disorders (e.g., Konnopka & König, 2020), yet PD is associated with higher levels of health care and medical service utilization than many other emotional disorders (Horenstein & Heimberg, 2020). Effective psychological and pharmaceutical treatments for PD exist, yet for many individuals suffering from PD, the positioning of thresholds on dimensional assessments of the construct can mean the difference between receiving treatment and going untreated (Magruder & Calderone, 2000; Pierre, 2010). Contrary to the representation of PD as a polythetic-categorical construct in the DSM-5, no empirical evidence has been produced suggesting that risk factors or mechanisms specific to PD interact in such a manner as to categorically distinguish clinical from nonclinical or subthreshold levels of panic symptoms. In other words, current diagnostic thresholds for the assessment of PD may be arbitrarily designated, resulting in individuals with subthreshold scores on measures of PD being obstructed from receiving appropriate treatment (Ruscio, 2019). A more accurate understanding of the latent

structure of PD would not only clarify the appropriateness of its representation in the DSM-5 as a distinct diagnostic entity, but inform its assessment and treatment as well. Fortunately, there are empirical methods of determining whether PD, or any other psychological disorder, is most accurately conceptualized by a dimensional or categorical framework.

Taxometric Methods

Taxometrics refers to a set of statistical procedures that were pioneered by Paul Meehl (e.g., Meehl, 1995; Meehl & Yonce, 1994, 1996; Waller & Meehl, 1998) and elaborated upon by Ruscio and colleagues (e.g., Ruscio et al., 2007, 2010; Walters & Ruscio, 2009) to provide an empirical means of testing the latent categorical or dimensional structure of a variable (for a review and history of taxometric procedures, see Ruscio et al., 2011; Waller, 2006). Dimensional constructs vary continuously on a spectrum, while taxonic, or categorical, constructs exhibit a threshold between two (or more) classes. Taxometric methods are based on the premise that dimensional and categorial constructs can be discriminated based on the patterns of relationships between relevant indicators of the construct (Meehl, 1999).

For example, consider measuring theoretically-relevant indicators of sex (i.e., height, facial hair, hip width, musculature, etc.) to determine the latent structure of sex. Cases sorted along one indicator of sex (e.g., blood content of testosterone) are tested on other indicators of sex at varying cutoff points to see if marked differences in the remaining indicators emerge beyond a certain threshold, reflective of the categorical variance of the underlying construct. In theory, repeating this procedure for each indicator clarifies distinctions being the two putative male and female classes (referred to as the *taxon* and *complement*) even when one indicator alone might not reveal separate classes within its own distribution. The language of which group belongs to the compliment and which belongs to the taxon is not fixed (Meehl, 1999). However,

in cases where taxometric methods are applied to psychopathology, the taxon is typically represented by the disordered, extreme, or otherwise abnormal manifestation of the construct of interest.

Conducting a taxometric analysis involves subjecting indicators of a conjectured taxon to multiple nonredundant procedures, such as mean-above-minus-mean-below-a-cut (MAMBAC; Meehl & Yonce, 1994), maximum covariance (MAXCOV; Meehl & Yonce, 1996), maximum Eigen value (MAXEIG; Waller & Meehl, 1998), and latent mode factor analysis (L-Mode; Waller & Meehl, 1998). These procedures do not involve hypothesis testing. Instead, they generate support for a categorical or dimensional structural model through the use of multiple consistency tests (Meehl, 1995). Simulated plots of both dimensional and categorical models of the dataset provide points of comparison for the empirical data (Ruscio et al., 2011). Notably, these procedures are not designed for factor detection, and while they can identify the presence of a single categorical boundary within a construct, they do not discriminate well when applied to constructs containing more than two categories (McGrath & Walters, 2012; Ruscio et al., 2011).

Although researchers have yet to apply taxometric methodology to PD, taxometric analyses of other anxiety disorders have generally supported dimensional latent structures. For example, taxometric analyses of generalized anxiety (Kertz et al., 2014; Marcus et al., 2014), social anxiety (Boyers et al., 2017; Crome et al., 2010; Kollman et al., 2006; Ruscio, 2010), health anxiety (Ferguson, 2009; Longley et al., 2010), separation anxiety (Silove et al., 2007), post-traumatic stress (Broman-Fulks et al., 2006, 2009; Forbes et al., 2005; Ruscio et al., 2002), and agoraphobia (Slade & Grisham, 2009) have all yielded dimensional findings using indicators derived from valid measures obtained from large clinical, non-clinical, and mixed samples. Only

one taxometric study of anxiety disorders reported finding evidence of categorical structure (i.e., social anxiety disorder; Weeks et al., 2010), though the results may be attributable to implementation of a sampling procedure known to produce pseudo-taxonic results (Ruscio & Ruscio, 2004) and subsequent studies of social anxiety were unable to replicate this finding (e.g., Boyers et al., 2017).

Several studies have examined known risk factors and characteristics of PD using taxometric analyses. While knowing the latent structure of its risk factors does not directly inform whether PD itself is categorical or dimensional, evidence for the categorical structures of unique risk factors for PD may be suggestive of a common etiological origin and/or subsequent categorical manifestation of the PD construct. For example, if worries were expressed categorically in the population, then GAD – a disorder characterized by excessive worrying – may also have a categorical latent structure. However, taxometric analyses applied to vulnerability factors for PD have largely supported continuous models. For example, taxometric studies of worry, experiential avoidance, intolerance of uncertainty, neuroticism, fear of pain, fear of social evaluation, somatization, and alexithymia have yielded dimensional findings (Asmundson et al., 2007; Carleton et al., 2012; Jasper et al., 2012; Kertz et al., 2014; Kirk et al., 2021; Kleim et al., 2014; Longley et al., 2017; Mattila et al., 2010; Olatunji et al., 2010; Parker et al., 2008; Ruscio et al., 2001; Thomas & Locke, 2010; Weeks et al., 2009).

Anxiety sensitivity (AS) is the only PD-relevant vulnerability factor that has been subjected to taxometric analysis and produced some evidence of taxonicity. AS is a transdiagnostic risk factor for emotional disorders (Smits et al., 2019) that is thought to encompass dimensions of physical, cognitive, and social concerns related to anxiety (Deacon & Abramowitz, 2006; Taylor et al., 2007). Some cognitive models of PD suggest that AS plays a

role as both cause and consequence of PD (McNally, 2002), especially its dimensions related to physical (e.g., Jurin & Biglbauer, 2018) and cognitive (e.g., Ino et al., 2017) concerns. While such models largely align with the learning theory model of PD proposed by Bouton et al. (2001), they differ in their suggestion that AS represents a fundamental, trait-like fear of anxiety sensations that may exist independent of direct experiences with PAs (Reiss & McNally, 1985).

The latent structure of AS has arguably been scrutinized to a greater degree than any other anxiety-related construct, and yet findings to date have been mixed (Haslam et al., 2020). Schmidt et al. (2005) reported that a cognitive vulnerability to panic (operationalized as facets of AS and body vigilance) exhibited a discontinuous latent structure when subjected to multiple taxometric procedures. In addition, Bernstein, Zvolensky, and colleagues reported finding evidence of an AS taxon across multiple studies in the mid-2000's (e.g., Bernstein, Leen-Feldner, et al., 2006; Bernstein, Zvolensky, Kotov, et al., 2006; Bernstein, Zvolensky, Stewart, & Comeau, 2007; Bernstein, Zvolensky, Stewart, et al., 2006; Bernstein et al., 2005). However, other researchers were unable to replicate these findings and several subsequent investigations employing more rigorous methodology, including the use of an objective fit index, provided evidence for the competing dimensional model (Asmundson et al., 2011; Broman-Fulks et al., 2010). Thus, the evidence for either model (or both) remains inconclusive.

In summary, the vast majority of taxometric studies of anxiety disorders and anxiety-related constructs have identified latent dimensions. This pattern of results aligns with more expansive reviews and meta-analyses suggesting that the vast majority of methodologically-sound taxometric studies in the fields of personality and psychopathology have produced evidence favoring the latent dimensional conceptualization of these constructs (e.g., Haslam et

al., 2012, 2020). Although researchers have yet to analyze the latent structure of PD, multiple lines of evidence suggest that most PD-related constructs have yielded dimensional findings.

Indicators of Panic Disorder

To test the latent structure of PD, potential indicators of a conjectured PD taxon that are suitable for taxometric analysis must first be identified. In taxometric analyses, individually fallible and theoretically relevant indicators (e.g., measures of "symptoms") of a particular construct (e.g., a disease) can reveal, in combination, its latent structure when sorted and tested along scores of other such indicators (Meehl, 1995). It is a common misconception that indicators of a putative latent taxon need be discontinuous or bimodal, reflective of the binary presence or absence of the construct. Rather, continuous indicators may elucidate latent taxons just as well as discontinuous ones (Meehl, 1995). Importantly, nearly all of the symptoms and risk factors associated with PD that have been reviewed thus far revealed continuous latent structures in taxometric analyses, with the exception of biological sex and possibly AS. Though not a specific indicator of PD, female sex predicts PD and other anxiety disorders with double the likelihood of male sex (Jalnapurkar et al., 2018). Unfortuantely, sex is not an ideal indicator for most taxometric procedures, as it is dichotomously distributed in large samples. While some taxometric procedures have been adapted to work with dichotomous indicators as "last-resort" methods (e.g., the modified MAXCOV procedure), these indicators are not ideal for taxometric analysis due to their limited range (Ruscio, 2000). Instead, continuous indicators, or categorical indicators with four or more ordered categories, are recommended (Walters & Ruscio, 2009). With these qualifications in place, key indicators of PD will be reviewed in accordance with their prominence in current diagnostic and theoretical models of the construct.

Panic Attacks

The DSM-5 criteria for the diagnosis of PD necessitates the experience of recurrent, unexpected PAs (APA, 2013). This criterion is consistent with learning theories of PD that offer mechanisms of fear conditioning and stimulus generalization as core components in the etiology and experience of the disorder (Bouton et al., 2001; Lissek et al., 2010). Similarly, theories supporting the existence of trait-like tendencies or sensitivities to experience PAs (e.g., overactive fear networks, AS, panic self-efficacy, etc.) would predict that individuals with these features would be more likely to experience recurrent and spontaneous PAs than those without them, creating a positive feedback loop between PAs and either biological systems or antecedent cognitions (Casey et al., 2004; Gorman et al., 2000; McNally, 2002). Thus, items assessing the experience of recurrent and unexpected PAs would represent potential theoretically-sound candidate indicators of a PD taxon, were one to exist.

Panic Concerns

Following the experience of unexpected PAs, people often develop persistent concerns about experiencing additional subsequent panic attacks and their consequences, which is also represented in DSM-5 criteria for PD (APA, 2013). In addition, such concerns are consistent with cognitive models of PD which suggest that catastrophic misrepresentations of probability and severity of future PAs play a key role in the development and maintenance of PD. Similarly, behavioral models assert that PD is comprised, in part, of anxious responding to overgeneralized fear cues. Thus, measures of worries and concerns about future PAs would also represent DSM-5 and theoretically consistent candidate indicators of PD.

Behavioral Changes

The final criterion for the DSM-5 diagnosis of PD is the experience of significant maladaptive changes in behavior related to PAs (APA, 2013). Learning theories suggest that avoidance behaviors associated with PD are negatively reinforced as prospective PAs are avoided (Bouton et al., 2001; Mkrtchian et al., 2017), and cognitive models assert that avoidance leads to a failure to disconfirm catastrophic cognitions about panic attacks (Barlow, 2004). Both models suggest that avoidance behaviors often become maladaptive and increase the severity and frequency of PAs, thereby leading to increasing levels of impairment (Spira et al., 2004). Thus, candidate indicators of PD for taxometric analyses include measures of avoidance behaviors, especially as they are oriented towards the goal of limiting future PAs, and the impairment in functioning resulting from maladaptive behavioral responses to PAs.

Present Study

The present study represents the first empirical study to examine the latent structure of PD by applying taxometric methodology to theoretically and diagnostically relevant indicators of PD. Candidate indicators meeting criteria for taxometric analysis (e.g., Ruscio et al., 2011) were subjected to a base rate classification procedure that categorizes cases into putative taxon and compliment groups based on indicator scores and *a priori* base rate estimation (Ruscio, 2008). Correlation analyses were used to identify within-group correlations in the conjectured taxon and compliment groups. Candidate indicators demonstrating low within-group correlations (r's $\leq .30$), high validities (d's ≥ 1.25), four or more ordered categories (for non-continuous variables), and meeting other suitability criteria (e.g., $n > 300$; $n_{taxon} \geq 50$; $P_{taxon} \geq .10$) were analyzed using three nonredundant taxometric procedures (i.e., MAMBAC, MAXEIG, and L-Mode). In accordance with theoretical models of PD and its current classification in the

DSM-5, indicators capturing the recurrent and unexpected nature of PAs, worry and anxiety associated with potential future PAs, avoidance of identifiable cues associated with PAs, and PA-related impairment were selected as candidate indicators. Consistent with the findings of previous taxometric analyses of emotion disorder symptomology, it was hypothesized that PD would demonstrate a latent dimensional structure (e.g., Haslam et al., 2020).

Method

Sample Selection

Appropriate sample selection is critical for the generation and interpretation of taxometric results. Taxometric analyses require large sample sizes ($n \geq 300$) that include a modestly sized putative taxon ($n_{\text{taxon}} \geq 50$; $P_{taxon} \geq .10$). Also, any non-random sampling procedure could create discontinuity (i.e., pseudotaxonicity) in the data reflective of measurement artifacts rather than actual taxon or complement group membership (Ruscio & Ruscio, 2004).

Data for the present study was collected as part of a larger study examining predictors of panic disorder (Bergquist, 2015). Participants (n = 664) were recruited from Amazon Mechanical Turk (MTurk) and compensated $0.40 for completing a brief (~10-minute) survey that included demographic items and the Panic Disorder Severity Scale (PDSS). To be included in the present analyses, participants had to be native or fluent English speakers and at least 18 years of age, respond accurately to four validation items embedded in the survey (see Appendix A), and have at least a 95% approval rate for prior work on the MTurk platform. The sample was split between females (58.7%) and males (41.3%) and was predominately adult (M = 38.1; SD = 12.3), non-Hispanic white (69.9%), and living in the United States (81.9%) at the time of the study (see Table 1). The partial responses of one participant who did not complete the survey were excluded from analysis; all other participants fully completed each study measure (n = 663). The data

collection procedures for the present study were approved by the Institutional Review Board at Appalachian State University.

Measures

Panic Disorder. The Panic Disorder Severity Scale (PDSS; Shear et al., 1997) is a self-report measure of PD symptom severity composed of seven items representing the DSM criteria for PD (see Appendix B). The questions comprising the PDSS assess recent (i.e., "*During the past week*") experiences with panic frequency, panic distress, anticipatory anxiety, agoraphobic and interoceptive fear/avoidance, and work and social impairment/distress. Each item is rated on a 5-point Likert scale ranging from 0 (e.g., "*Not at all distressing*") to 4 (e.g., "*Extremely distressing*"); a total score ranging from 0 to 28 is summed from these items, with higher total scores representing PD symptom severity.

The PDSS is a reliable and valid measure of PD symptom severity that changes in response to PD treatment (Houck et al., 2002). A cut-off score of eight on the English version of the PDSS has been shown to detect PD patients with a sensitivity of 83.3% and a specificity of 64% (Shear et al., 2001). Furukawa et al. (2009) offer evidence-based guidelines for the interpretation of total PDSS scores – with scores greater than or equal to 6, 10, and 14 indicating "slightly", "moderately", and "markedly" ill respondents, respectively – and claim that even slightly ill scorers are "clearly diagnosable". Using these cutoffs as rough estimates of the rate of PD in the present sample, 35% of participants met criteria for being "slightly ill", 19% met criteria for being "moderately ill", and 11% met criteria for being "markedly ill", in terms of PD symptom severity. Finally, PDSS items were internally consistent in the present dataset (α = .94; ω = .94).

Indicator Selection

Indicator selection is one of the most important considerations in the taxometric method. Indicators should capture theoretically-relevant aspects of the construct of interest (Meehl, 1995) and must meet a set of minimum statistical criteria, including adequate range (e.g., at least four ordered categories for non-continuous indicators; Ruscio & Ruscio, 2004), high validity (i.e., a mean separation between conjectured taxon and complement groups of ≥ 1.25 standard deviations), and low within-group correlations (i.e., $r < .30$) for both the putative taxon and complement groups. Although only two indicators are necessary to run MAMBAC analysis, three or more indicators that meet these criteria are necessary to run MAXEIG and L-Mode analyses (Ruscio et al. 2010).

Based on their theoretical and clinical relevance to current conceptualizations of PD, the seven PDSS items were selected as candidate indicators of PD for the present study (see Appendix B for each item). Specifically, PDSS items assess PA frequency (PDSS item 1), PA-related distress (PDSS item 2), worry and anxiety about future PAs (PDSS item 3), fear and avoidance associated with PAs (PDSS items 4 and 5), and functional impairment associated with PAs (PDSS items 6 and 7). A primary indicator set was created using all of these items individually. In addition, consistent with the emphasis placed on replication of findings in taxometrics rather than traditional significance testing, an alternative set of four indicators was also constructed based on DSM-5 criteria. Specifically, indicator 1 represented DSM-5 diagnostic criterion A (i.e., requiring the experience of recurrent panic attacks involving intense fear; PDSS items 1 and 2 summed), indicator 2 represented diagnostic criterion B1 (i.e., PAs followed by persistent concern or worry about additional panic attacks; PDSS item 3), indicator 3 represented criterion B2 (i.e., PAs followed by maladaptive changes in behavior; PDSS items 4

and 5 summed), and indicator 4 represented the general DSM-5 criterion that symptoms cause clinically significant funcitonal impairment (i.e., PDSS items 6 and 7 summed). Both the individual and combined item indicator sets were evaluated and analyzed.

Data Analytic Strategy

Taxometric analyses were performed using the RTaxometrics package (Ruscio & Wang, 2020) in the RStudio programming environment (RStudio Team, 2020). Before checking whether the indicator sets met criteria for taxometric analysis, cases were assigned to putative taxon and complement groups based on empirically supported cutoff scores on the PDSS. Specifically, individuals who scored 6 or higher on the PDSS (i.e., meeting at least "slightly ill" cutoff for PD; Furukawa et al., 2009) were assigned to the putative taxon group, with the remaining cases assigned to the complement group.

After assigning cases to putative taxon and complement groups, the CheckData function in the RTaxometrics package was used to evaluate the suitability of the indicators for taxometric analysis (i.e., indicators with validities ≥ 1.25 *SD* and within group correlations ≤ .30). Indicators that met (or nearly met) these criteria were then subjected to MAMBAC, MAXEIG, and L-Mode taxometric procedures (RunTaxometrics), which in combination provide nonredundant evaluation of the latent structure of PD. Plots generated by each procedure from the empirical sample data were compared via parallel analysis to Monte Carlo simulations of 100 samples (n = 100,000) of parametrically similar data for both dimensional and categorical models.

MAMBAC. In the MAMBAC procedure, a "cut", representing a hypothetical classificatory threshold, is established at the lowest end of the score distribution of one indicator. The scores of a second indicator are then assigned to this distribution, and the mean differences of cases above and below the cut are plotted on the y-axis against the number of cases on the

x-axis. This procedure is repeated for a predetermined number of cuts further along the distribution of the input indicator until the final cut is established at the highest end of the distribution. In the present analyses, 25 cuts were made along each input variable. MAMBAC yields a plot averaged for every paired indicator combination of these mean differences (on the *y*-axis) set against cut values (on the *x*-axis). Applying the MAMBAC procedure to sample data allows for both objective and visual comparisons of fit between plots of sample data and plots of simulated data from both categorical and dimensional models derived from the parameters of the sample data. Peaked curves on the MAMBAC plot suggest that a categorical distinction exists at the cut below the peak, whereas flat curves suggest a dimensional structure.

MAXEIG. The MAXEIG procedure sorts cases along scores of a single indicator and then plots the maximum eigenvalue in the covariance matrix of the remaining indicators against a number of overlapping "windows" (i.e., sliding subsample score averages) of the sorting indicator. In the present analyses, overlap for windows was set to a default value of .90. The resultant MAXEIG plot averaged from each input indicator combination illustrates changes in the eigenvalue associations of indicators. MAXEIG curves can be interpreted as representing categorical structure when eigenvalues decrease at lower windows (i.e., compliment scores) and higher windows (i.e., taxon scores) relative to middle windows, producing a peaked curve; conversely, dimensional structure is revealed in a MAXEIG plot as a relatively flat curve.

L-Mode. The L-Mode procedure involves factor analyzing indicators using a weighted least squares approach and plotting distributions of factor scores from the first extracted factor. Categorical structures are revealed in the bimodality of this distribution, while dimensional structure assumes a unimodal distribution in an L-Mode plot.

Interpretation

Taxometric procedures generate a comparison curve fit index (CCFI; Ruscio et al., 2007) statistic that provides an objective comparison between the empirical data plots and the simulated taxonic and dimensional plots. The CCFI is a ratio of the root-mean-square distances of empirical data points from the dimensional comparison plot data points, over their total distance from both dimensional and categorical data points. This ratio ranges from 0 to 1, with lower scores (< .45) indicating that the empirical data aligns more closely with dimensional comparison data, and higher scores (> .45) indicating a closer fit with categorical comparison data. CCFI values between .45 and .55 are generally interpreted as ambiguous (Ruscio et al., 2010).

Objective fit indices and supplemental visual analyses were used to interpret the results of the present taxometric analyses. For visual analysis, the fit of empirical data plots was compared to simulated dimensional and categorical plots generated by each taxometric procedure (MAMBAC, MAXEIG, and L-mode). Peaked curves produced by MAMBAC / MAXEIG plots of the sample data, bimodal L-Mode plots of the sample data, and CCFI scores greater than .55 were interpreted as evidence for the categorical structure of PD. Conversely, flat curves produced by MAMBAC / MAXEIG plots of the sample data, unimodal L-Mode plots of the sample data, and CCFI scores less than .45 were interpreted as evidence for the dimensional structure of PD. In the absence of clear visual indications of model fit, or in cases where CCFI scores fell between .45 and .55, the results were interpreted as ambiguous.

Results

Preliminary Analyses

Based on the *a priori* threshold set for putative taxon membership (i.e., scoring 6 or higher on the PDSS), approximately 35% of the sample (n = 231) was assigned to the putative taxon group, while the remaining cases (n = 432) were assigned to the putative complement (Figure 1). Indicator validities (i.e., the standardized mean differences of indicators between taxon and complement groups) well-exceeded minimum requirements for each of the single-item (d = 2.24) and composite item (d = 2.51) indicators. In other words, there were differences of more than two standard deviations between mean scores of the putative taxon and complement groups on these indicators. Within-group correlations among the indicators were appropriately low for the putative complement group in both analyses ($r_{complement}$'s = .20 and .21), though the within-group correlations among the conjectured taxon group modestly exceeded the target recommendation of r = .30 in both indicator sets (r_{taxon}'s = .38 and .46). As noted in previous research (Meehl, 1995; Ruscio et al., 2007), minor violations of the targeted within-group correlations are unlikely to affect the interpretability of the resulting data plots, particularly when other indicator qualities (e.g., validity) are strong. Thus, each of the indicators selected for this study met minimum suitability criteria and were deemed appropriate for taxometric analysis. Detailed indicator validities and within-group correlations for each indicator set are presented in Table 3.

Taxometric Analyses

The individual PDSS item indicators were first subjected to MAMBAC, MAXEIG, and L-Mode, and the resulting data plots were examined relative to their consistency with simulated taxonic and dimensional plots generated from simulated data with similar distributional

characteristics to the empirical data (see Figures 2 and 3). Results indicated that the individual plots generated by MAMBAC, MAXEIG, and L-Mode analyses were largely consistent with the shape of the plots produced by simulated dimensional data. Specifically, the MAMBAC and MAXEIG curves appeared relatively flat and positively sloped, while the L-Mode curve appeared unimodal. This is in contrast with the typical inverse U-shape of MAMBAC and MAXEIG plots and bimodal distributional shape of L-Mode plots generated produced by taxonic data. The objective fit index (i.e., CCFI) score was .39, which is well-below the dimensional cutoff (i.e., .45), and thereby provided objective support for the dimensional interpretation.

Taxometric analysis of the composite-item indicator set generated similar results. Specifically, none of the empirical data plots produced by the MAXEIG, MAMBAC, and L-Mode procedures exhibited typical characteristics of taxonicity. Rather, all of the data plots appeared consistent with typical dimensional plots, and the CCFI score was .29, which provided strong objective support for a dimensional model.

Discussion

Empirically-supported theoretical models suggest that PD arises from a complex set of biological, behavioral, and cognitive processes (e.g., Pilecki et al., 2011), yet commonly accepted classification systems (e.g., DSM-5, ICD-10) treat PD as a categorical construct. The present study represents the first attempt to empirically test the latent structure of PD using taxometric methodology. Two sets of indicators derived from one of the most commonly used diagnostic measures of PD (i.e., the PDSS) were subjected to three non-redundant taxometric procedures: MAMBAC, MAXEIG, and L-Mode. As predicted, the collective results of the analyses, including CCFI scores and visual comparisons of data plots with simulated

dimensional and taxonic plots, provided consistent evidence for the latent dimensional structure of PD.

Implications

Previous research has identified factors and clusters within the symptom structure of PD (e.g., Cox et al., 1994; Kenardy et al., 1992), but little attention has been paid to the latent structure of the disorder itself. This study is the first to offer empirical evidence for the dimensional latent structure of PD, and it joins the collection of taxometric findings supporting the dimensional latent structures of most studied psychological disorders to date (Haslam et al., 2020). The dimensional results of the present study are consistent with empirically-supported theoretical models that suggest that PD is multidimensional phenomenon that appears to be caused and maintained by a complex array of biological, environmental, and behavioral factors (e.g., Bouton et al., 2001; McNally, 2002; Pilecki et al., 2011). Models that propose that PD results from a specific gene, environmental variable, or gene-environment interaction would be contraindicated by the present findings. Thus, future research would benefit from focusing on investigating the manner in which specific hereditory factors and life events influence the dimensional gradations of panic-related experiences. It is worth noting that, although the present findings suggest that PD is a dimensional construct, they do not necessarily speak to whether the experience of a panic attack, in and of itself, is a categorical or dimensional phenomenon, or whether the conceptualized distinctions between panic and anxiety (or other theoretically-relevant constructs) exist along a shared dimension (e.g., of predatory imminence; Fanselow, 1994) or as two discrete neurobehavioral responses (LeDoux & Pine, 2016). Additional research will be necessary to answer such empirical questions.

Counter to its representation within the DSM-5, available taxometric evidence suggests that a clearly identifiable boundary between nonclinical and clinical presentations of PD-related symptomology likely does not exist. If this finding is confirmed by future research, it would indicate that the practice of sorting individuals into disordered and non-disordered categories is inevitably creating arbitrary dichotimizations of a continuous construct. As such, researchers, health care providers, and insurance companies should be aware that current diagnostic thresholds, cutoffs, and criteria for PD likely represent unnatural and unnecessary distinctions that do not accurately reflect the dimensional nature of the PD construct. Further, individuals who experience panic-related symptomology but fall below the arbitrary threshold set by the current categorical diagnostic nomenclature (e.g., “subclinical” cases) may be unnecessarily limited or prevented from receiving services that they would likely benefit from as much as individuals scoring just above such thresholds. Likewise, treatment goals should be reconsidered in light of the dimesional latent structure of PD. Goals to eliminate PD or reduce symptoms down to a “subclinical” level may not be as appropriate as negotiating process-based ideographic treatment goals informed by identifying where along dimensions clients are suffering. Finally, evidence for the dimensionality of PD can be used to inform the development of psychometric measures, so as to more accurately, fully, and efficiently capture the construct for the purposes of assessment, progress tracking, and research.

Implementing these changes may not be possible in current diagnostic systems like the DSM-5, ICD-10, or RDoC. Accumulating evidence appears to be dissolving the boundaries between mental wellness and mental illness, and such boundaries are inherent presumptions for categorical diagnostic systems. When clinical classification shifts from categories to dimensions, the focus of clinical research must likewise shift from disease entities to the processes that move

individuals along dimensions of wellbeing, health, and functionality, while taking care not to rigidly categorize the resulting dimensions of change. Attempts to facilitate this process are underway. For example, Hayes et al. (2020) suggest that clinical diagnosis and intevention can be effectively informed by applying evolutionary principles of selection, variation, and retention, in context, to dimensions of change across various levels of analysis (i.e., psychological, physiological, sociocultural). This extended evolutionary meta-model (EEMM) allows for evidence-based processes of change to be applied flexibily and ideographically when placed in the context of philosophically consistent models of intervention (e.g., Acceptance and Commitment Therapy; Hayes et al., 2011). For example, an ideographic intervention for panic-related suffering within the EEMM can be accomplished through the functional analysis of an individual without the need for diagnostic cutoffs or criteria. Unlike in traditional systems of diagnosis, the variables and processes targetted by such an intervention would be determined by the individual and their context – not by the latent features of an underlying syndrome – and the goals of the intervention would be determined by the client in collaboration with the therapist. Since both the functional analysis and intervention goal-setting in EEMM-based interventions would be specific to each individual, it remains to be seen how such an approach would interact with contemporary economic, social, and legal systems (e.g., disability, insurance) that continue to function under outdated models of mental health.

Strengths and Limitations

Nearly all of the criteria pertaining to the suitability of indicators for taxometric analysis (e.g., Ruscio et al., 2011) were met in the present study, with the exception of mildly elevated within-group indicator correlations among the putative taxon group. Although near-zero nuisance correlations are an idealization that is often unobtainable in psychopathology, research has

suggested that taxometric procedures are robust to minor violations of this criteria, particularly when sample size is large and indicator validities well-exceed minimum cutoffs (Meehl, 1995; Ruscio et al., 2007). In fact, Ruscio et al. (2007) found that CCFIs produced by taxometric procedures accurately identified taxonicity in 100% of analyses of indicators of taxonic constructs that were simulated under varying parameters (include nuisance correlations). Further, many studies published in the empirical literature exceed the minimum recommended cutoffs for within-group correlations (e.g., Carleton et al., 2012; Kollman et al., 2006; Olatunji et al., 2008), and, if anything, elevated within group correlations would be expected to potentially bias results in favor of finding taxonic structure, even where it does not exist. Thus, the mild elevations in within-group correlations among the conjectured taxon group observed in the present study are unlikely to have influenced the interpretability of findings.

One potential explanation for the increased within-group correlations among the conjectured taxon pertains to the wording of PDSS items. The PDSS provides seven theoretically and clinically relevant indicators of PD, but responses to most items are largely affected by whether the respondent has experienced PAs (or limited symptom attacks) within the past week. Consequently, more than a third of the sample scored “0” on all items because they had not recently experienced a PA or limited symptom attack at the time of data collection. Restricted response range and standard deviation at the low scoring end of the present sample may account for why within-group correlations in the complement were not excessively high, as these qualities are known to reduce Pearson correlation coefficients (Goodwin & Leech, 2006; see Appendix C). Further, high within-group correlations in taxon groups can be understood in the context of the intensity of typical experiences with PAs. Recent experiences with PAs are likely

to be reported as severe across the range of all PDSS items, and it is unlikely that an individual would only report a few aspect of their PA as severe while considering the rest relatively mild.

Taxometric procedures are robust with respect to skewed indicators like those used in the present study (Cleland & Haslam, 1996; Haslam & Cleland, 1996), though positively skewed indicators have been noted to lead to taxonic misinterpretations of otherwise dimensional results (Ruscio & Ruscio, 2002; Ruscio et al., 2004). Despite the high levels of indicator skew observed among PDSS items potentially biasing analyses towards identifying categorical structures in the data, results consistently favored dimensional solutions. Ultimately, taxometric analyses depend on the quality of theoretically relevant indicators, and, while limited with respect to expected skew, the PDSS items used in the present study appear to be as effective as any in capturing theoretically and clinically important features of PD. Even so, future studies should aim to replicate these analyses using independent and varied indicators of PD, including biological (e.g., heart rate variabilty; Na et al., 2021) and behavioral measures (e.g., defensive responding; Benke et al., 2021), with particular efforts to ensure suitably low nuisance correlations and skew among indicators, if possible. In addition, the duration of experience with PAs beyond the most recent week, the expected/unexpected nature of PAs, biomarkers of fear generalization (see Asok et al., 2019), or other putative processes underlying PD would make for ideal indicators.

The sample used in the present study was relatively restricted with regard to demographic reprentation, consisting predominately of white, well-educated English speakers living in the U.S., a demographic about which much has already been said with respect to its overrepresentation in the psychological literature (e.g., Rad et al., 2018). Though research suggests that risk and resiliency factors associated with PD depend somewhat on demographic variation, the processes underlying PD symptomology are not thought to be specific to any

particular demographic. For example, prevelance rates of PD differ across ethnicity (Asnaani et al., 2010), sex (McLean et al., 2011), age (Olaya et al., 2018), and culture (Marques et al., 2011), yet the panic response and avoidance behaviors central to evidence-based theoretical and diagnostic models of PD are features of all humans (as well as other animal species). Nevertheless, additional research should examine the structure of PD using more diverse samples and indicators of PD relevent to its population-specific symptom presentations and risk/resiliency factors.

A large proportion (P = .35) of MTurk workers in the present sample reported PDSS scores meeting or exceeding evidence-based thresholds for "clearly diagnosable" PD, equivalent to a "slightly ill" level of clinical severity (Furukawa et al., 2009). However, the estimated prevalence of PD in the U.S. population is only 3.7% (Kessler et al., 2006) and 1.7% in the global population (de Jonge et al., 2016). Although research suggests that MTurk respondents tend to endorse significantly higher rates of emotional disorder symptomology relative to the general population (e.g., Arditte et al., 2016), the reported rate of PD in the current sample is remarkable considering the non-clinical nature of the sample. Thus, despite our efforts to ensure data integrity and protect against response biases via the implementation of inclusion criteria (e.g., restricting survey access to MTurk workers with a history of generating quality work based on approval rates > .95%) and imposition of multiple validation checks (Ophir et al., 2020), it remains possible that some of the participants may have provided poor quality data. However, even if participants had purposefully responded to PDSS items with all high or low scores to finish the survey faster, these cases would be unlikely to significantly impact the general integrity of our findings as taxometric analyses tend to focus on the middle of the response range to identify putative boundaries within latent structures. It is also possible that the sample

selection methodology used resulted in an overrepresentation of individuals with PD, given that such persons may be more likely to opt for relatively safe and remote online work (e.g., MTurk) than individuals without PD, due to well-documented increases in rates of avoidance of novel situations or places from which escape may be difficult were they to experience a panic attack.

Indeed, the mean PDSS score observed in the present sample (M = 4.7; SD = 5.8) was lower than those reported in similar nonclinical samples of MTurk workers (e.g., M = 5.5; SD = 6.9; Manning et al., 2021) as well as clinical samples of psychiatric outpatients (e.g., M = 9.0; SD = 6.6; Houck et al., 2002). Thus, it is possible that the methods used in the present study to estimate the base rate of a putative PD taxon were insufficient in properly discriminating between clinical and nonclinical cases (i.e., the guidelines proposed by Furukawa et al. [2009] may be overly liberal in their classification of moderately-scoring individuals as "clearly diagnosable" with PD). On the other hand, taxometric procedures like MAMBAC and MAXEIG assess for potentially taxonic boundaries within the full range of data, regardless of group assignment; in other words, though CCFIs would be impacted by the change, the empirical data curves produced by these procedures would have remained the same even if only 3% of the sample been assigned to taxon groups (see Appendix D). Future taxometric analyses of PD should aim to replicate these findings using more stringent criteria for determining class membership, such as indicators derived from diagnostic interviews. In addition, replication of the present findings in a sample of mixed-diagnosis outpatients would enhance confidence in the dimensional results observed in the current study.

References

Abramowitz, J. S., Deacon, B. J., & Whiteside, S. P. (2019). *Exposure therapy for anxiety: Principles and practice*. Guilford Publications.

Acheson, D. T., Forsyth, J. P., Prenoveau, J. M., & Bouton, M. E. (2007). Interoceptive fear conditioning as a learning model of panic disorder: An experimental evaluation using 20% CO2-enriched air in a non-clinical sample. *Behaviour Research and Therapy, 45*(10), 2280–2294. doi:10.1016/j.brat.2007.04.008

Allsopp, K., Read, J., Corcoran, R., & Kinderman, P. (2019). Heterogeneity in psychiatric diagnostic classification. *Psychiatry Research, 279*, 15–22. doi:10.1016/j.psychres.2019.07.005

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Publisher.

Arditte, K. A., Çek, D., Shaw, A. M., & Timpano, K. R. (2016). The importance of assessing clinical phenomena in Mechanical Turk research. *Psychological Assessment*, *28*(6), 684.

Asmundson, G. J. G., Collimore, K. C., Bernstein, A., Zvolensky, M. J., & Hadjistavropoulos, H. D. (2007). Is the latent structure of fear of pain continuous or discontinuous among pain patients? Taxometric analysis of the pain anxiety symptoms scale. *The Journal of Pain, 8*(5), 387–395. doi:10.1016/j.jpain.2006.10.007

Asmundson, G. J. G., Taylor, S., & A. J. Smits, J. (2014). Panic disorder and agoraphobia: An overview and commentary on DSM-5 changes. *Depression and Anxiety, 31*(6), 480–486. doi:10.1002/da.22277

Asmundson, G. J. G., Weeks, J. W., Nicholas Carleton, R., Thibodeau, M. A., & Fetzner, M. G. (2011). Revisiting the latent structure of the anxiety sensitivity construct: More evidence

of dimensionality. *Journal of Anxiety Disorders, 25*(1), 138–147. doi:10.1016/j.janxdis.2010.08.013

Asnaani, A., Richey, J. A., Dimaite, R., Hinton, D. E., & Hofmann, S. G. (2010). A cross-ethnic comparison of lifetime prevalence rates of anxiety disorders. *The Journal of Nervous and Mental Disease*, *198*(8), 551–555. https://doi.org/10.1097/NMD.0b013e3181ea169f

Asok, A., Kandel, E. R., & Rayman, J. B. (2019). The neurobiology of fear generalization. *Frontiers in Behavioral Neuroscience*, *12*, 329. https://doi.org/10.3389/fnbeh.2018.00329

Baillie, A. J., & Rapee, R. M. (2005). Panic attacks as risk markers for mental disorders. *Social Psychiatry and Psychiatric Epidemiology, 40*(3), 240–244. doi:10.1007/s00127-005-0892-3

Bandelow, B., & Baldwin, D. S. (2020). Pharmacotherapy for panic disorder. *The American Psychiatric Association Publishing Textbook of Anxiety, Trauma, and OCD-Related Disorders*, 385.

Bandelow, B., & Michaelis, S. (2015). Epidemiology of anxiety disorders in the 21st century. *Dialogues in Clinical Neuroscience*, *17*(3), 327–335.

Bandura, A. (1988). Self-efficacy conception of anxiety. *Anxiety Research*, *1*(2), 77-98.

Barlow, D. H. (2004). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. Guilford press.

Batelaan, N., De Graaf, R., Van Balkom, A., Vollebergh, W., & Beekman, A. (2006). Thresholds for health and thresholds for illness: panic disorder versus subthreshold panic disorder. *Psychological Medicine, 37*(02), 247. doi:10.1017/s0033291706009007

Batelaan, N., Smit, F., de Graaf, R., van Balkom, A., Vollebergh, W., & Beekman, A. (2007). Economic costs of full-blown and subthreshold panic disorder. *Journal of Affective Disorders*, *104*(1-3), 127–136. https://doi.org/10.1016/j.jad.2007.03.013

Beck, A. T. (1988). *Cognitive approaches to panic disorder: Theory and therapy.* In S. Rachman & J. D. Maser (Eds.), *Panic: Psychological perspectives* (p. 91–109). Lawrence Erlbaum Associates, Inc.

Bekker, M. H. J., & van Mens-Verhulst, J. (2007). Anxiety disorders: Sex differences in prevalence, degree, and background, but gender-neutral treatment. *Gender Medicine, 4*, S178–S193. doi:10.1016/s1550-8579(07)80057-x

Benke, C., Alius, M. G., Hamm, A. O., & Pané-Farré, C. A. (2021). Decreased defensive reactivity to interoceptive threat after successful exposure-based psychotherapy in patients with panic disorder. *Translational Psychiatry*, *11*(1), 1-11.

Bergquist, J. J. (2015). *Incremental validity of psychological flexibility, anxiety sensitivity, and mindfulness in the prediction of panic disorder symptomology and life satisfaction* (Doctoral dissertation, Appalachian State University).

Bernstein, A., Leen-Feldner, E. W., Kotov, R., Schmidt, N. B., & Zvolensky, M. J. (2006). *Test of a panic-relevant diathesis-stress model using a taxonic index of anxiety sensitivity.* In A. J. Sanfelippo (Ed.), *Panic disorders: New research* (p. 15–40). Nova Biomedical Books.

Bernstein, A., Zvolensky, M. J., Kotov, R., Arrindell, W. A., Taylor, S., Sandin, B., Cox, B. J., Stewart, S. H., Bouvard, M., Cardenas, S. J., & Eifert, G. H. (2006). Taxonicity of anxiety sensitivity: A multinational analysis. *Journal of Anxiety Disorders, 20*, 1-20.

Bernstein, A., Zvolensky, M. J., Stewart, S., & Comeau, N. (2007). Taxometric and factor analytic models of anxiety sensitivity among youth: Exploring the latent structure of

anxiety psychopathology vulnerability. *Behavior Therapy, 38*(3), 269–283. doi:10.1016/j.beth.2006.08.005

Bernstein, A., Zvolensky, M. J., Stewart, S. H., Comeau, M. N., & Leen-Feldner, E. W. (2006). Anxiety sensitivity taxonicity across gender among youth. *Behaviour Research and Therapy, 44*, 679-698.

Bernstein, A., Zvolensky, M. J., Weems, C., Stickle, T., & Leen-Feldner, E. W. (2005). Taxonicity of anxiety sensitivity: An empirical test among youth. *Behaviour Research and Therapy, 43*, 1131-1155.

Bjelland, I., Lie, S. A., Dahl, A. A., Mykletun, A., Stordal, E., & Kraemer, H. C. (2009). A dimensional versus a categorical approach to diagnosis: Anxiety and depression in the HUNT 2 study. *International Journal of Methods in Psychiatric Research*, *18*(2), 128-137.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, *108*(1), 4–32. doi:10.1037/0033-295x.108.1.4

Boyers, G. B., Broman-Fulks, J. J., Valentiner, D. P., McCraw, K., Curtin, L., & Michael, K. D. (2017). The latent structure of social anxiety disorder and the performance only specifier: A taxometric analysis. *Cognitive Behaviour Therapy, 46*(6), 507–521. doi:10.1080/16506073.2017.1338310

Brendel, D. H. (2001). Multifactorial causation of mental disorders: A proposal to improve the DSM. *Harvard Review of Psychiatry, 9*(1), 42–45. doi:10.1080/10673220127876

Broman-Fulks, J. J., Deacon, B. J., Olatunji, B. O., Bondy, C. L., Abramowitz, J. S., & Tolin, D. F. (2010). Categorical or dimensional: A reanalysis of the anxiety sensitivity construct. *Behavior Therapy, 41*(2), 154–171. doi:10.1016/j.beth.2009.02.005

Broman-Fulks, J. J., Ruggiero, K. J., Green, B. A., Kilpatrick, D. G., Danielson, C. K., Resnick, H. S., & Saunders, B. E. (2006). Taxometric investigation of PTSD: Data from two nationally representative samples. *Behavior Therapy*, *37*(4), 364-380.

Broman-Fulks, J. J., Ruggiero, K. J., Green, B. A., Smith, D. W., Hanson, R. F., Kilpatrick, D. G., & Saunders, B. E. (2009). The latent structure of posttraumatic stress disorder among adolescents. *Journal of Traumatic Stress, 22*(2), 146–152. doi:10.1002/jts.20399

Brown, T. A., & McNiff, J. (2009). Specificity of autonomic arousal to DSM-IV panic disorder and posttraumatic stress disorder. *Behaviour Research and Therapy*, *47*(6), 487–493. https://doi.org/10.1016/j.brat.2009.02.016

Carleton, R. N., Fetzner, M. G., Hackl, J. L., & McEvoy, P. (2013). Intolerance of uncertainty as a contributor to fear and avoidance symptoms of panic attacks. *Cognitive Behaviour Therapy, 42*(4), 328–341. doi:10.1080/16506073.2013.792100

Carleton, R. N., Weeks, J. W., Howell, A. N., Asmundson, G. J. G., Antony, M. M., & McCabe, R. E. (2012). Assessing the latent structure of the intolerance of uncertainty construct: An initial taxometric analysis. *Journal of Anxiety Disorders, 26*(1), 150–157. doi:10.1016/j.janxdis.2011.10.006

Casey, L. M., Oei, T. P., Newcombe, P. A., & Kenardy, J. (2004). The role of catastrophic misinterpretation of bodily sensations and panic self-efficacy in predicting panic severity. *Journal of Anxiety Disorders*, *18*(3), 325-340.

Chang, H. M., Pan, C. H., Chen, P. H., Chen, Y. L., Tai, M. H., Su, S. S., Tsai, S. Y., Chen, C. C., & Kuo, C. J. (2019). Sex differences in incidence and psychiatric comorbidity for alcohol dependence in patients with panic disorder. *Drug and Alcohol Dependence, 207*, 107814. doi:10.1016/j.drugalcdep.2019.107814

Chartier-Otis, M., Perreault, M., & Bélanger, C. (2010). Determinants of barriers to treatment for anxiety disorders. *Psychiatric Quarterly, 81*(2), 127–138. doi:10.1007/s11126-010-9123-5

Chen, M.-H., & Tsai, S.-J. (2016). Treatment-resistant panic disorder: Clinical significance, concept and management. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 70*, 219–226. doi:10.1016/j.pnpbp.2016.02.001

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy*, *24*(4), 461-470.

Clark, L. A., Cuthbert, B., Lewis-Fernández, R., Narrow, W. E., & Reed, G. M. (2017). Three approaches to understanding and classifying mental disorder: ICD-11, DSM-5, and the National Institute of Mental Health's Research Domain Criteria (RDoC). *Psychological Science in the Public Interest: A Journal of the American Psychological Society*, *18*(2), 72–145. https://doi.org/10.1177/1529100617727266

Cleland, C., & Haslam, N. (1996). Robustness of taxometric analysis with skewed indicators: I. A Monte Carlo study of the MAMBAC procedure. *Psychological Reports*, *79*(1), 243-248.

Coles, M. E., & Coleman, S. L. (2010). Barriers to treatment seeking for anxiety disorders: Initial data on the role of mental health literacy. *Depression and Anxiety, 27*(1), 63–71. doi:10.1002/da.20620

de Cort, K., Griez, E., Büchler, M., & Schruers, K. (2012). The role of "interoceptive" fear conditioning in the development of panic disorder. *Behavior Therapy, 43*(1), 203–215. doi:10.1016/j.beth.2011.06.005

de Cort, K., Schroijen, M., Hurlemann, R., Claassen, S., Hoogenhout, J., Van den Bergh, O., Goossens, L., Van Diest, I., & Schruers, K. (2017). Modeling the development of panic disorder with interoceptive conditioning. *European Neuropsychopharmacology, 27*(1), 59–69. doi:10.1016/j.euroneuro.2016.11.001

Cox, B. J., Swinson, R. P., Endler, N. S., & Norton, G. R. (1994). The symptom structure of panic attacks. *Comprehensive Psychiatry, 35*(5), 349–353. doi:10.1016/0010-440x(94)90274-7

Craske, M. G., Edlund, M. J., Sullivan, G., Roy-Byrne, P., Sherbourne, C., Bystritsky, A., & Stein, M. B. (2005). Perceived unmet need for mental health treatment and barriers to care among patients with panic disorder. *Psychiatric Services, 56*(8), 988–994. doi:10.1176/appi.ps.56.8.988

Crome, E., Baillie, A., Slade, T., & Ruscio, A. M. (2010). Social phobia: Further evidence of dimensional structure. *The Australian and New Zealand Journal of Psychiatry*, *44*(11), 1012–1020. https://doi.org/10.3109/00048674.2010.507544

Davey, G. C. (1992). Classical conditioning and the acquisition of human fears and phobias: A review and synthesis of the literature. *Advances in Behaviour Research and Therapy, 14*(1), 29–66. doi:10.1016/0146-6402(92)90010-l

Deacon, B., & Abramowitz, J. (2006). Anxiety sensitivity and its dimensions across the anxiety disorders. *Journal of Anxiety Disorders*, *20*(7), 837-857.

Del-Ben, C. M., & Graeff, F. G. (2009). Panic disorder: is the PAG involved? *Neural plasticity*, *2009*, 108135. https://doi.org/10.1155/2009/108135

van Diest, I. (2019). Interoception, conditioning and fear: The panic threesome. *Psychophysiology*, *56*(8).

Dold, M., Bartova, L., Souery, D., Mendlewicz, J., Serretti, A., Porcelli, S., Zohar, J., Montgomery, S., & Kasper, S. (2017). Clinical characteristics and treatment outcomes of patients with major depressive disorder and comorbid anxiety disorders - results from a European multicenter study. *Journal of Psychiatric Research, 91*, 1–13. doi:10.1016/j.jpsychires.2017.02.020

Domínguez, R., Cruz-Morales, S. E., Carvalho, M. C., Xavier, M., & Brandao, M. L. (2003). Sex differences in serotonergic activity in dorsal and median raphe nucleus. *Physiology & Behavior*, *80*(2-3), 203–210. https://doi.org/10.1016/j.physbeh.2003.07.012

Domschke, K., Stevens, S., Pfleiderer, B., & Gerlach, A. L. (2010). Interoceptive sensitivity in anxiety and anxiety disorders: An overview and integration of neurobiological findings. *Clinical Psychology Review, 30*(1), 1–11. doi:10.1016/j.cpr.2009.08.008

Donner, N. C., & Lowry, C. A. (2013). Sex differences in anxiety and emotional behavior. *Pflugers Archiv : European Journal of Physiology*, *465*(5), 601–626. https://doi.org/10.1007/s00424-013-1271-7

Fanselow, M. S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychonomic Bulletin & Review, 1*(4), 429–438. doi:10.3758/bf03210947

Fanselow, M. S., & Lester, L. S. (1988). *A functional behavioristic approach to aversively motivated behavior: Predatory imminence as a determinant of the topography of*

defensive behavior. In R. C. Bolles & M. D. Beecher (Eds.), *Evolution and learning* (p. 185–212). Lawrence Erlbaum Associates, Inc.

Ferguson, E. (2009). A taxometric analysis of health anxiety. *Psychological Medicine*, *39*(2), 277–285. https://doi.org/10.1017/S0033291708003322

First, M. B. (2010). Clinical utility in the revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM). *Professional Psychology: Research and Practice, 41*(6), 465–473. doi:10.1037/a0021511

Forbes, D., Haslam, N., Williams, B. J., & Creamer, M. (2005). Testing the latent structure of posttraumatic stress disorder: A taxometric study of combat veterans. *Journal of Traumatic Stress: Official Publication of The International Society for Traumatic Stress Studies*, *18*(6), 647-656.

Forstner, A.J., Awasthi, S., Wolf, C., Maron, E., Erhardt, A., Czamara, D., Eriksson, E., Lavebratt, C., Allgulander, C., Friedrich, N., Becker, J., Hecker, J., Rambau, S., Conrad, R., Geiser, F., McMahon, F. J., Moebus, S., Hess, T., Buerfent, B. C., … Schumacher, J. (2019). Genome-wide association study of panic disorder reveals genetic overlap with neuroticism and depression. *Molecular Psychiatry*. doi:10.1038/s41380-019-0590-2

Fullana, M. A., Tortella-Feliu, M., Fernández de la Cruz, L., Chamorro, J., Pérez-Vigil, A., Ioannidis, J., Solanes, A., Guardiola, M., Almodóvar, C., Miranda-Olivos, R., Ramella-Cravaro, V., Vilar, A., Reichenberg, A., Mataix-Cols, D., Vieta, E., Fusar-Poli, P., Fatjó-Vilas, M., & Radua, J. (2019). Risk and protective factors for anxiety and obsessive-compulsive disorders: an umbrella review of systematic reviews and meta-analyses. *Psychological Medicine*, 1–16. doi:10.1017/s0033291719001247

Furukawa, T. A., Katherine Shear, M., Barlow, D. H., Gorman, J. M., Woods, S. W., Money, R., Etschel, E., Engel, R. R., & Leucht, S. (2009). Evidence-based guidelines for interpretation of the Panic Disorder Severity Scale. *Depression and Anxiety, 26*(10), 922–929. doi:10.1002/da.20532

Goldstein-Piekarski, A. N., Williams, L. M., & Humphreys, K. (2016). A trans-diagnostic review of anxiety disorder comorbidity and the impact of multiple exclusion criteria on studying clinical outcomes in anxiety disorders. *Translational Psychiatry*, *6*(6), e847-e847.

Goodwin, L. D., & Leech, N. L. (2006). Understanding correlation: Factors that affect the size of r. *The Journal of Experimental Education*, *74*(3), 249-266.

Goodwin, R. D., Lieb, R., Hoefler, M., Pfister, H., Bittner, A., Beesdo, K., & Wittchen, H.-U. (2004). Panic attack as a risk factor for severe psychopathology. *American Journal of Psychiatry, 161*(12), 2207–2214. doi:10.1176/appi.ajp.161.12.2207

Gorman, J. M., Kent, J. M., Sullivan, G. M., & Coplan, J. D. (2000). Neuroanatomical hypothesis of panic disorder, revised. *American Journal of Psychiatry*, *157*(4), 493-505.

Gorman, J. M., Liebowitz, M. R., Fyer, A. J., & Stein, J. (1989). A neuroanatomical hypothesis for panic disorder. *The American Journal of Psychiatry, 146*(2), 148-161.

Graeff, F. G. (2017). Translational approach to the pathophysiology of panic disorder: Focus on serotonin and endogenous opioids. *Neuroscience & Biobehavioral Reviews*, *76*, 48-55.

Grassi, M., Caldirola, D., Di Chiaro, N. V., Riva, A., Daccò, S., Pompili, M., & Perna, G. (2014). Are respiratory abnormalities specific for panic disorder? A meta-analysis. *Neuropsychobiology, 70*(1), 52–60. https://doi.org/10.1159/000364830

Grassi, M., Caldirola, D., Vanni, G., Guerriero, G., Piccinni, M., Valchera, A., & Perna, G. (2013). Baseline respiratory parameters in panic disorder: a meta-analysis. *Journal of Affective Disorders*, *146*(2), 158–173. https://doi.org/10.1016/j.jad.2012.08.034

Greene, A. L., & Eaton, N. R. (2016). Panic disorder and agoraphobia: A direct comparison of their multivariate comorbidity patterns. *Journal of Affective Disorders, 190*, 75–83. doi:10.1016/j.jad.2015.09.060

Hamm, A. O., Richter, J., & Pané-Farré, C. A. (2014). When the threat comes from inside the body: A neuroscience based learning perspective of the etiology of panic disorder. *Restorative Neurology and Neuroscience*, *32*(1), 79–93. https://doi.org/10.3233/RNN-139011

Haslam, N., & Cleland, C. (1996). Robustness of taxometric analysis with skewed indicators: II. A Monte Carlo study of the MAXCOV procedure. *Psychological Reports*, *79*(3), 1035-1039.

Haslam, N., Holland, E., & Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: A quantitative review of taxometric research. *Psychological Medicine, 42*(05), 903–920. doi:10.1017/s0033291711001966

Haslam, N., McGrath, M. J., Viechtbauer, W., & Kuppens, P. (2020). Dimensions over categories: A meta-analysis of taxometric research. *Psychological Medicine*, 1–15. doi:10.1017/s003329172000183x

Hayes, S. C., Hofmann, S. G., & Ciarrochi, J. (2020). A process-based approach to psychological diagnosis and treatment:The conceptual and treatment utility of an extended evolutionary meta model. *Clinical Psychology Review*, 101908. doi:10.1016/j.cpr.2020.101908

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2011). *Acceptance and commitment therapy: The process and practice of mindful change*. Guilford Press.

Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H. U., Sirovatka, P. J., & Regier, D. A. (Eds.). (2009). *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V*. American Psychiatric Pub.

Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A review and meta-analysis of the genetic epidemiology of anxiety disorders. *American Journal of Psychiatry*, *158*(10), 1568-1578.

Horenstein, A., & Heimberg, R. G. (2020). Anxiety disorders and healthcare utilization: A systematic review. *Clinical Psychology Review*, 101894.

Houck, P. R., Spiegel, D. A., Shear, M. K., & Rucci, P. (2002). Reliability of the self-report version of the panic disorder severity scale. *Depression and Anxiety*, *15*(4), 183-185.

Howe, A. S., Buttenschøn, H. N., Bani-Fatemi, A., Maron, E., Otowa, T., Erhardt, A., Binder, E. B., Gregersen, N. O., Mors, O., Woldbye, D. P., Domschke, K., Reif, A., Shlik, J., Kõks, S., Kawamura, Y., Miyashita, A., Kuwano, R., Tokunaga, K., Tanii, H., Smoller, J. W., Sasaki, T., Koszycki, D., & De Luca, V. (2016). Candidate genes in panic disorder: Meta-analyses of 23 common variants in major anxiogenic pathways. *Molecular Psychiatry*, *21*(5), 665-679.

Ino, K., Ogawa, S., Kondo, M., Imai, R., Ii, T., Furukawa, T. A., & Akechi, T. (2017). Anxiety sensitivity as a predictor of broad dimensions of psychopathology after cognitive behavioral therapy for panic disorder. *Neuropsychiatric Disease and Treatment*, *13*, 1835.

Iurato, S., Carrillo-Roa, T., Arloth, J., Czamara, D., Diener-Hölzl, L., Lange, J., Müller-Myhsok, B., Binder, E. B., & Erhardt, A. (2017). DNA Methylation signatures in panic disorder. *Translational Psychiatry*, *7*(12), 1-10.

Jalnapurkar, I., Allen, M., & Pigott, T. (2018). Sex differences in anxiety disorders: A review. *Psychiatry, Depression & Anxiety, 4*. doi:10.24966/pda-0150/100011

Jasper, F., Hiller, W., Rist, F., Bailer, J., & Witthöft, M. (2012). Somatic symptom reporting has a dimensional latent structure: Results from taxometric analyses. *Journal of Abnormal Psychology, 121*(3), 725–738. https://doi.org/10.1037/a0028407

Johnson, P. L., Federici, L. M., & Shekhar, A. (2014). Etiology, triggers and neurochemical circuits associated with unexpected, expected, and laboratory-induced panic attacks. *Neuroscience and Biobehavioral Reviews*, *46 Pt 3*, 429–454. https://doi.org/10.1016/j.neubiorev.2014.07.027

Johnson, P. L., Lightman, S. L., & Lowry, C. A. (2004). A functional subset of serotonergic neurons in the rat ventrolateral periaqueductal gray implicated in the inhibition of sympathoexcitation and panic. *Annals of the New York Academy of Sciences, 1018*(1), 58–64. doi:10.1196/annals.1296.006

de Jonge, P., Roest, A. M., Lim, C. C., Florescu, S. E., Bromet, E. J., Stein, D. J., Harris, M., Nakov, V., Caldas-de-Almeida, J. M., Levinson, D., Al-Hamzawi, A. O., Haro, J. M., Viana, M. C., Borges, G., O'Neill, S., de Girolamo, G., Demyttenaere, K., Gureje, O., Iwata, N., Lee, S., … Scott, K. M. (2016). Cross-national epidemiology of panic disorder and panic attacks in the world mental health surveys. *Depression and Anxiety*, *33*(12), 1155–1177. https://doi.org/10.1002/da.22572

Jurin, T., & Biglbauer, S. (2018). Anxiety sensitivity as a predictor of panic disorder symptoms: A prospective 3-year study. *Anxiety, Stress, & Coping, 31*(4), 365–374. doi:10.1080/10615806.2018.1453745

Kalk, N. J., & Young, A. H. (2017). Footnotes to Kraepelin: changes in the classification of mood disorders with DSM-5. *BJPsych Open*, *3*(3), 1–3. doi:10.1192/bjpo.bp.117.004739

Kämpfe, C. K., Gloster, A. T., Wittchen, H.-U., Helbig-Lang, S., Lang, T., Gerlach, A. L., Richter, J., Alpers, G. W., Fehm, L., Kircher, T., Hamm, A. O., Ströhle, A., & Deckert, J. (2012). Experiential avoidance and anxiety sensitivity in patients with panic disorder and agoraphobia: Do both constructs measure the same? *International Journal of Clinical and Health Psychology, 12*(1), 5–22.

Kamphuis, J. H., & Noordhof, A. (2009). On categorical diagnoses in DSM-V: Cutting dimensions at useful points?. *Psychological Assessment*, *21*(3), 294.

Kelly, M. M., Forsyth, J. P., & Karekla, M. (2006). Sex differences in response to a panicogenic challenge procedure: An experimental evaluation of panic vulnerability in a non-clinical sample. *Behaviour Research and Therapy, 44*(10), 1421–1430. doi:10.1016/j.brat.2005.10.012

Kenardy, J., Evans, L., & Oei, T. P. (1992). The latent structure of anxiety symptoms in anxiety disorders. *The American Journal of Psychiatry*, *149*(8), 1058–1061. https://doi.org/10.1176/ajp.149.8.1058

Kenardy, J., & Taylor, C. B. (1999). Expected versus unexpected panic attacks: A naturalistic prospective study. *Journal of Anxiety Disorders*, *13*(4), 435–445. https://doi.org/10.1016/s0887-6185(99)00013-4

Kertz, S. J., McHugh, R. K., Lee, J., & Björgvinsson, T. (2014). Examining the latent structure of worry and generalized anxiety in a clinical sample. *Journal of Anxiety Disorders, 28*(1), 8–15. doi:10.1016/j.janxdis.2013.11.003

Kessler, R. C., Chiu, W. T., Jin, R., Ruscio, A. M., Shear, K., & Walters, E. E. (2006). The epidemiology of panic attacks, panic disorder, and agoraphobia in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *63*(4), 415-424.

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research, 21*(3), 169–184. doi:10.1002/mpr.1359

Kinley, D. J., Walker, J. R., Enns, M. W., & Sareen, J. (2011). Panic attacks as a risk for later psychopathology: results from a nationally representative survey. *Depression and Anxiety, 28*(5), 412–419. doi:10.1002/da.20809

Kircanski, K., Craske, M. G., Epstein, A. M., & Wittchen, H.-U. (2011). Subtypes of panic attacks: A critical review of the empirical literature. *FOCUS, 9*(3), 389–398. doi:10.1176/foc.9.3.foc389

Kirk, A., Broman-Fulks, J. J., & Arch, J. J. (2021). A taxometric analysis of experiential avoidance. *Behavior Therapy*, *52*(1), 208-220.

Kollman, D. M., Brown, T. A., Liverant, G. I., & Hofmann, S. G. (2006). A taxometric investigation of the latent structure of social anxiety disorder in outpatients with anxiety and mood disorders. *Depression and Anxiety*, *23*(4), 190-199.

Konnopka, A., & König, H. (2020). Economic burden of anxiety disorders: A systematic review and meta-analysis. *Pharmacoeconomics*, 1-13.

Konnopka, A., Leichsenring, F., Leibing, E., & König, H.-H. (2009). Cost-of-illness studies and cost-effectiveness analyses in anxiety disorders: A systematic review. *Journal of Affective Disorders, 114*(1-3), 14–31. doi:10.1016/j.jad.2008.07.014

Kraemer, H. C. (2007). DSM categories and dimensions in clinical and research contexts. *International Journal of Methods in Psychiatric Research*, *16*(S1), S8-S15.

LeDoux, J. E., & Pine, D. S. (2016). Using neuroscience to help understand fear and anxiety: A two-system framework. *American Journal of Psychiatry, 173*(11), 1083–1093. doi:10.1176/appi.ajp.2016.16030353

Lee, Y. J., Hohoff, C., Domschke, K., Sand, P., Kuhlenbäumer, G., Schirmacher, A., Freitag, C. M., Meyer, J., Stöber, G., Franke, P., Nöthen, M. M., Fritze, J., Fimmers, R., Garritsen, H. S., Stögbauer, F., & Deckert, J. (2005). Norepinephrine transporter (NET) promoter and 5′-UTR polymorphisms: Association analysis in panic disorder. *Neuroscience Letters*, *377*(1), 40-43.

Lijster, J. M., Dierckx, B., Utens, E. M., Verhulst, F. C., Zieldorff, C., Dieleman, G. C., & Legerstee, J. S. (2017). The age of onset of anxiety disorders. *Canadian Journal of Ppsychiatry. Revue Canadienne de Psychiatrie*, *62*(4), 237–246. https://doi.org/10.1177/0706743716640757

Lissek, S., Bradford, D. E., Alvarez, R. P., Burton, P., Espensen-Sturges, T., Reynolds, R. C., & Grillon, C. (2014). Neural substrates of classically conditioned fear-generalization in humans: a parametric fMRI study. *Social Cognitive and Affective Neuroscience*, *9*(8), 1134–1142. https://doi.org/10.1093/scan/nst096

Lissek, S., & Grillon, C. (2010). Overgeneralization of conditioned fear in the anxiety disorders: Putative memorial mechanisms. *Zeitschrift für Psychologie/Journal of Psychology, 218*(2), 146–148. https://doi.org/10.1027/0044-3409/a000022

Lissek, S., Rabin, S., Heller, R. E., Lukenbaugh, D., Geraci, M., Pine, D. S., & Grillon, C. (2010). Overgeneralization of conditioned fear as a pathogenic marker of panic disorder. *American Journal of Psychiatry, 167*(1), 47–55. doi:10.1176/appi.ajp.2009.09030410

Longley, S. L., Broman-Fulks, J. J., Calamari, J. E., Noyes, R., Wade, M., & Orlando, C. M. (2010). A taxometric study of hypochondriasis symptoms. *Behavior Therapy, 41*(4), 505–514. doi:10.1016/j.beth.2010.02.002

Longley, S. L., Miller, S. A., Broman-Fulks, J., Calamari, J. E., Holm-Denoma, J. M., & Meyers, K. (2017). Taxometric analyses of higher-order personality domains. *Personality and Individual Differences*, *108*, 207-219.

Lonsdorf, T. B., & Merz, C. J. (2017). More than just noise: Inter-individual differences in fear acquisition, extinction and return of fear in humans - Biological, experiential, temperamental factors, and methodological pitfalls. *Neuroscience & Biobehavioral Reviews, 80*, 703–728. doi:10.1016/j.neubiorev.2017.07.007

Mackenzie, C. S., Reynolds, K., Cairney, J., Streiner, D. L., & Sareen, J. (2011). Disorder-specific mental health service use for mood and anxiety disorders: Associations with age, sex, and psychiatric comorbidity. *Depression and Anxiety, 29*(3), 234–242. doi:10.1002/da.20911

Magruder, K. M., & Calderone, G. E. (2000). Public health consequences of different thresholds for the diagnosis of mental disorders. *Comprehensive Psychiatry, 41*(2), 14–18. doi:10.1016/s0010-440x(00)80003-6

Manning, K., Eades, N. D., Kauffman, B. Y., Long, L. J., Richardson, A. L., Garey, L., Zvolensky, M. J., & Gallagher, M. W. (2021). Anxiety sensitivity moderates the impact of

COVID-19 perceived stress on anxiety and functional impairment. *Cognitive Therapy and Research*, 1-8.

Marcus, D. K., Sawaqdeh, A., & Kwon, P. (2014). The latent structure of generalized anxiety disorder in midlife adults. *Psychiatry Research, 215*(2), 366–371. doi:10.1016/j.psychres.2013.12.011

Marques, L., Robinaugh, D. J., LeBlanc, N. J., & Hinton, D. (2011). Cross-cultural variations in the prevalence and presentation of anxiety disorders. *Expert Review of Neurotherapeutics*, *11*(2), 313-322.

Mattila, A. K., Keefer, K. V., Taylor, G. J., Joukamaa, M., Jula, A., Parker, J. D. A., & Bagby, R. M. (2010). Taxometric analysis of alexithymia in a general population sample from Finland. *Personality and Individual Differences, 49*(3), 216–221. doi:10.1016/j.paid.2010.03.038

Mayes, R., & Horwitz, A. V. (2005). DSM-III and the revolution in the classification of mental illness. *Journal of the History of the Behavioral Sciences*, *41*(3), 249–267. https://doi.org/10.1002/jhbs.20103

McGrath, R. E., & Walters, G. D. (2012). Taxometric analysis as a general strategy for distinguishing categorical from dimensional latent structure. *Psychological Methods, 17*(2), 284–293. https://doi.org/10.1037/a0026973

McLean, C. P., Asnaani, A., Litz, B. T., & Hofmann, S. G. (2011). Gender differences in anxiety disorders: prevalence, course of illness, comorbidity and burden of illness. *Journal of Psychiatric Research*, *45*(8), 1027–1035. https://doi.org/10.1016/j.jpsychires.2011.03.006

McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry*, *52*(10), 938-946.

Meehl, P. E. (1995). Bootstraps taxometrics. *American Psychologist, 50,* 266-275.

Meehl, P. E. (1999). Clarifications about taxometric method. *Applied and Preventive Psychology, 8*(3), 165–174. doi:10.1016/s0962-1849(05)80075-7

Meehl, P. E., & Yonce, L. J. (1994). Taxometric analysis I: Detecting taxonicity with two quantitative indicators using means above and below a sliding cut (MAMBAC procedure). *Psychology Reports, 74,* 1059-1274.

Meehl, P. E., & Yonce, L. J. (1996). Taxometric analysis II: Detecting taxonicity using covariance of two quantitative indicators in successive intervals of a third indicator (MAXCOV procedure). *Psychological Reports, 78,* 1091-1227.

Meuret, A. E., Kroll, J., & Ritz, T. (2017). Panic disorder comorbidity with medical conditions and treatment implications. *Annual Review of Clinical Psychology, 13*(1), 209–240. doi:10.1146/annurev-clinpsy-021815-093044

Meuret, A. E., Rosenfield, D., Wilhelm, F. H., Zhou, E., Conrad, A., Ritz, T., & Roth, W. T. (2011). Do unexpected panic attacks occur spontaneously?. *Biological Psychiatry*, *70*(10), 985–991. https://doi.org/10.1016/j.biopsych.2011.05.027

Meuret, A. E., White, K. S., Ritz, T., Roth, W. T., Hofmann, S. G., & Brown, T. A. (2006). Panic attack symptom dimensions and their relationship to illness characteristics in panic disorder. *Journal of Psychiatric Research, 40*(6), 520–527. doi:10.1016/j.jpsychires.2005.09.006

Mineka, S., & Oehlberg, K. (2008). The relevance of recent developments in classical conditioning to understanding the etiology and maintenance of anxiety disorders. *Acta Psychologica, 127*(3), 567–580. doi:10.1016/j.actpsy.2007.11.007

Mkrtchian, A., Aylward, J., Dayan, P., Roiser, J. P., & Robinson, O. J. (2017). Modeling avoidance in mood and anxiety disorders using reinforcement learning. *Biological Psychiatry, 82*(7), 532–539. doi:10.1016/j.biopsych.2017.01.017

Na, K. S., Cho, S. E., & Cho, S. J. (2021). Machine learning-based discrimination of panic disorder from other anxiety disorders. *Journal of Affective Disorders*, *278*, 1-4.

Naragon-Gainey, K., & Watson, D. (2018). What lies beyond neuroticism? An examination of the unique contributions of social-cognitive vulnerabilities to internalizing disorders. *Assessment, 25*(2), 143–158. doi:10.1177/1073191116659741

Navinés, R., Egmond, E., & Martín-Santos, R. (2016). Panic disorder and personality disorder comorbidity. In *Panic Disorder* (pp. 169-184). Springer, Cham.

Nees, F., Heinrich, A., & Flor, H. (2015). A mechanism-oriented approach to psychopathology: The role of Pavlovian conditioning. *International Journal of Psychophysiology, 98*(2), 351–364. doi:10.1016/j.ijpsycho.2015.05.005

Neueder, D., Andreatta, M., & Pauli, P. (2019). Contextual fear conditioning and fear generalization in individuals with panic attacks. *Frontiers in Behavioral Neuroscience, 13*, 152. https://doi.org/10.3389/fnbeh.2019.00152

Newby, J.M., Hobbs, M.J., Mahoney, A.E.J., Wong, S.K., & Andrews, G. (2017). DSM-5 illness anxiety disorder and somatic symptom disorder: Comorbidity, correlates, and overlap with DSM-IV hypochondriasis. *Journal of Psychosomatic Research, 101*, 31-37. https://doi.org/10.1016/j.jpsychores.2017.07.010

Newman, M. G., Shin, K. E., & Zuellig, A. R. (2016). Developmental risk factors in generalized anxiety disorder and panic disorder. *Journal of Affective Disorders*, *206*, 94–102. https://doi.org/10.1016/j.jad.2016.07.008

Olatunji, B. O., Broman-Fulks, J. J., Bergman, S. M., Green, B. A., & Zlomke, K. R. (2010). A taxometric investigation of the latent structure of worry: Dimensionality and associations with depression, anxiety, and stress. *Behavior Therapy, 41*(2), 212–228. doi:10.1016/j.beth.2009.03.001

Olatunji, B. O., Williams, B. J., Haslam, N., Abramowitz, J. S., & Tolin, D. F. (2008). The latent structure of obsessive-compulsive symptoms: A taxometric study. *Depression and Anxiety*, *25*(11), 956-968.

Olaya, B., Moneta, M. V., Miret, M., Ayuso-Mateos, J. L., & Haro, J. M. (2018). Epidemiology of panic attacks, panic disorder and the moderating role of age: Results from a population-based study. *Journal of Affective Disorders*, *241*, 627-633.

van Oort, J., Tendolkar, I., Hermans, E. J., Mulders, P. C., Beckmann, C. F., Schene, A. H., Fernández, G., & van Eijndhoven, P. F. (2017). How the brain connects in response to acute stress: A review at the human brain systems level. *Neuroscience & Biobehavioral Reviews, 83*, 281–297. doi:10.1016/j.neubiorev.2017.10.015

Ophir, Y., Sisso, I., Asterhan, C. S., Tikochinski, R., & Reichart, R. (2020). The turker blues: Hidden factors behind increased depression rates among Amazon's mechanical turkers. *Clinical Psychological Science*, *8*(1), 65-83.

Otto, M. W., McHugh, R. K., Simon, N. M., Farach, F. J., Worthington, J. J., & Pollack, M. H. (2010). Efficacy of CBT for benzodiazepine discontinuation in patients with panic disorder: Further evaluation. *Behaviour Research and Therapy*, *48*(8), 720–727. https://doi.org/10.1016/j.brat.2010.04.002

Parker, J. D. A., Keefer, K. V., Taylor, G. J., & Bagby, R. M. (2008). Latent structure of the alexithymia construct: A taxometric investigation. *Psychological Assessment, 20*(4), 385–396. doi:10.1037/a0014262

Perusini, J. N., & Fanselow, M. S. (2015). Neurobehavioral perspectives on the distinction between fear and anxiety. *Learning & Memory, 22*(9), 417–425. doi:10.1101/lm.039180.115

Pierre, J. M. (2010). The borders of mental disorder in psychiatry and the DSM. *Journal of Psychiatric Practice, 16*(6), 375–386. doi:10.1097/01.pra.0000390756.37754.68

Pies, R. W. (2009). Depression and the pitfalls of causality: Implications for DSM-V. *Journal of Affective Disorders*, *116*(1-2), 1–3. https://doi.org/10.1016/j.jad.2008.11.009

Pilecki, B., Arentoft, A., & McKay, D. (2011). An evidence-based causal model of panic disorder. *Journal of Anxiety Disorders*, *25*(3), 381-388.

Pittig, A., Treanor, M., LeBeau, R. T., & Craske, M. G. (2018). The role of associative fear and avoidance learning in anxiety disorders: Gaps and directions for future research. Neuroscience & Biobehavioral Reviews, 88, 117–140. doi:10.1016/j.neubiorev.2018.03.015

Pobbe, R. L., Zangrossi, H., Jr, Blanchard, D. C., & Blanchard, R. J. (2011). Involvement of dorsal raphe nucleus and dorsal periaqueductal gray 5-HT receptors in the modulation of mouse defensive behaviors. *European Neuropsychopharmacology: The Journal of the European College of Neuropsychopharmacology*, *21*(4), 306–315. https://doi.org/10.1016/j.euroneuro.2010.05.004

Quagliato, L. A., & Nardi, A. E. (2018). Cytokine alterations in panic disorder: A systematic review. *Journal of Affective Disorders*, *228*, 91-96.

Rad, M. S., Martingano, A. J., & Ginges, J. (2018). Toward a psychology of Homo sapiens: Making psychological science more representative of the human population. *Proceedings of the National Academy of Sciences*, *115*(45), 11401-11405.

Raskin, M., Peeke, H. V., Dickman, W., & Pinsker, H. (1982). Panic and generalized anxiety disorders: Developmental antecedents and precipitants. *Archives of General Psychiatry*, *39*(6), 687-689.

Reiss, S., & McNally, R. J. (1985). *Expectancy model of fear*. In S. Reiss & R.R. Bootzin (Eds.), *Theoretical issues in behavior therapy*. San Diego, CA: Academic Press.

Roberson-Nay, R., & Kendler, K. S. (2011). Panic disorder and its subtypes: a comprehensive analysis of panic symptom heterogeneity using epidemiological and treatment seeking samples. *Psychological Medicine, 41*(11), 2411–2421. doi:10.1017/S0033291711000547

RStudio Team (2020). RStudio: Integrated Development for R. RStudio, PBC, Boston, MA URL http://www.rstudio.com/.

Ruscio, A. M. (2010). The latent structure of social anxiety disorder: Consequences of shifting to a dimensional diagnosis. *Journal of Abnormal Psychology*, *119*(4), 662–671. https://doi.org/10.1037/a0019341

Ruscio, A. M. (2019). Normal versus pathological mood: Implications for diagnosis. *Annual Review of Clinical Psychology*.

Ruscio, A. M., Borkovec, T. D., & Ruscio, J. (2001). A taxometric investigation of the latent structure of worry. *Journal of Abnormal Psychology*, *110*(3), 413.

Ruscio, A. M., & Ruscio, J. (2002). The latent structure of analogue depression: Should the Beck Depression Inventory be used to classify groups?. *Psychological Assessment*, *14*(2), 135.

Ruscio, A. M., Ruscio, J., & Keane, T. M. (2002). The latent structure of posttraumatic stress disorder: A taxometric investigation of reactions to extreme stress. *Journal of Abnormal Psychology*, *111*(2), 290.

Ruscio, J. (2000). Taxometric analysis with dichotomous indicators: The modified MAXCOV procedure and a case-removal consistency test. *Psychological Reports*, *87*(3), 929-939.

Ruscio, J. (2008). A probability-based measure of effect size: Robustness to base rates and other factors. *Psychological Methods, 13*(1), 19–30. https://doi.org/10.1037/1082-989X.13.1.19

Ruscio, J., Ruscio, A. M., & Keane, T. M. (2004). Using taxometric analysis to distinguish a small latent taxon from a latent dimension with positively skewed indicators: the case of involuntary defeat syndrome. *Journal of Abnormal Psychology*, *113*(1), 145.

Ruscio, J., & Ruscio, A. M. (2004). Clarifying boundary issues in psychopathology: The role of taxometrics in a comprehensive program of structural research. *Journal of Abnormal Psychology, 113*(1), 24.

Ruscio, J., Ruscio, A. M., & Carney, L. M. (2011). Performing taxometric analysis to distinguish categorical and dimensional variables. *Journal of Experimental Psychopathology, 2*(2), jep.010910. doi:10.5127/jep.010910

Ruscio, J., Ruscio, A. M., & Meron, M. (2007). Applying the bootstrap to taxometric analysis: Generating empirical sampling distributions to help interpret results. *Multivariate Behavioral Research, 42*(2), 349–386. doi:10.1080/00273170701360795

Ruscio, J., Walters, G. D., Marcus, D. K., & Kaczetow, W. (2010). Comparing the relative fit of categorical and dimensional latent variable models using consistency tests. *Psychological Assessment, 22*(1), 5–21. doi:10.1037/a0018259

Ruscio, J., & Wang, S. B. (2020). RTaxometrics: Taxometric analysis. R package version 2.4.

Sánchez-Meca, J., Rosa-Alcázar, A. I., Marín-Martínez, F., & Gómez-Conesa, A. (2010). Psychological treatment of panic disorder with or without agoraphobia: A meta-analysis. *Clinical Psychology Review, 30*(1), 37 – 50. doi:10.1016/j.cpr.2009.08.011

Schmidt, N. B., Forsyth, J. P., Santiago, H. T., & Trakowski, J. H. (2002). Classification of panic attack subtypes in patients and normal controls in response to biological challenge: Implications for assessment and treatment. *Journal of Anxiety Disorders, 16*(6), 625–638. doi:10.1016/s0887-6185(02)00131-7

Schmidt, N. B., Kotov, R., Lerew, D. R., Joiner, T. E., & Ialongo, N. S. (2005). Evaluating latent discontinuity in cognitive vulnerability to panic: A taxometric investigation. *Cognitive Therapy and Research, 29*(6), 673–690. doi:10.1007/s10608-005-9632-y

Schür, R. R., Draisma, L. W., Wijnen, J. P., Boks, M. P., Koevoets, M. G., Joëls, M., Klomp, D. W., Kahn, R. S., & Vinkers, C. H. (2016). Brain GABA levels across psychiatric disorders: A systematic literature review and meta-analysis of 1H-MRS studies. *Human Brain Mapping*, *37*(9), 3337-3352.

Schwartze, D., Barkowski, S., Strauss, B., Burlingame, G. M., Barth, J., & Rosendahl, J. (2017). Efficacy of group psychotherapy for panic disorder: Meta-analysis of randomized, controlled trials. *Group Dynamics: Theory, Research, and Practice, 21*(2), 77–93. https://doi.org/10.1037/gdn0000064

Shear, M. K., Brown, T. A., Barlow, D. H., Money, R., Sholomskas, D. E., Woods, S. W., Gorman, J. M., & Papp, L. A. (1997). Multicenter collaborative panic disorder severity scale. *American Journal of Psychiatry*, *154*(11), 1571-1575.

Shear, M. K., Rucci, P., Williams, J., Frank, E., Grochocinski, V., Vander Bilt, J., Houck, P., & Wang, T. (2001). Reliability and validity of the Panic Disorder Severity Scale: replication and extension. *Journal of Psychiatric Research*, *35*(5), 293-296.

Silove, D., Slade, T., Marnane, C., Wagner, R., Brooks, R., & Manicavasagar, V. (2007). Separation anxiety in adulthood: Dimensional or categorical?. *Comprehensive Psychiatry*, *48*(6), 546-553.

Skapinakis, P., Lewis, G., Davies, S., Brugha, T., Prince, M., & Singleton, N. (2011). Panic disorder and subthreshold panic in the UK general population: epidemiology, comorbidity and functional limitation. *European Psychiatry*, *26*(6), 354-362.

Slade, T., & Grisham, J. R. (2009). A taxometric investigation of agoraphobia in a clinical and a community sample. *Journal of Anxiety Disorders*, *23*(6), 799–805. https://doi.org/10.1016/j.janxdis.2009.03.004

Smits, J. A., Otto, M. W., Powers, M. B., & Baird, S. O. (2019). Anxiety sensitivity as a transdiagnostic treatment target. In *The Clinician's Guide to Anxiety Sensitivity Treatment and Assessment* (pp. 1-8). Academic Press.

Somers, J. M., Goldner, E. M., Waraich, P., & Hsu, L. (2006). Prevalence and incidence studies of anxiety disorders: a systematic review of the literature. *Canadian Journal of Psychiatry*, *51*(2), 100–113. https://doi.org/10.1177/070674370605100206

Spinhoven, P., Drost, J., de Rooij, M., van Hemert, A. M., & Penninx, B. W. (2014). A longitudinal study of experiential avoidance in emotional disorders. *Behavior Therapy, 45*(6), 840–850. doi:10.1016/j.beth.2014.07.001

Spira, A. P., Zvolensky, M. J., Eifert, G. H., & Feldner, M. T. (2004). Avoidance-oriented coping as a predictor of panic-related distress: A test using biological challenge. *Journal of Anxiety Disorders*, *18*(3), 309-323.

Street, L. L., Craske, M. G., & Barlow, D. H. (1989). Sensations, cognitions and the perception of cues associated with expected and unexpected panic attacks. *Behaviour Research and Therapy, 27*(2), 189–198. doi:10.1016/0005-7967(89)90078-8

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., Abramowitz, J. S., Holaway, R. M., Sandin, B., Stewart, S. H., Coles, M., Eng, W., Daly, E. S., Arrindell, W. A., Bouvard, M., & Cardenas, S. J. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index-3. *Psychological Assessment*, *19*(2), 176.

Thomas, M. L., & Locke, D. E. C. (2010). Psychometric properties of the MMPI-2-RF Somatic Complaints (RC1) Scale. *Psychological Assessment, 22*(3), 492–503. https://doi.org/10.1037/a0019229

Tietbohl-Santos, B., Chiamenti, P., Librenza-Garcia, D., Cassidy, R., Zimerman, A., Manfro, G. G., Kapczinski, F., & Passos, I. C. (2019). Risk factors for suicidality in patients with panic disorder: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews, 105*, 34-38. doi:10.1016/j.neubiorev.2019.07.022

Tilli, V., Suominen, K., & Karlsson, H. (2012). Panic disorder in primary care: Comorbid psychiatric disorders and their persistence. *Scandinavian Journal of Primary Health Care, 30*(4), 247–253. doi:10.3109/02813432.2012.732471

Waller, N. G. (2006). Carving nature at its joints: Paul Meehl's development of taxometrics. *Journal of Abnormal Psychology, 115*(2), 210-215. https://doi.org/10.1037/0021-843X.115.2.210

Waller, N. G., & Meehl, P. E. (1998). *Multivariate taxometric procedures: Distinguishing types from continua.* Sage Publications, Inc.

Walters, G. D., & Ruscio, J. (2009). Where do we draw the line? Assigning cases to subsamples for MAMBAC, MAXCOV, and MAXEIG taxometric analyses. *Assessment, 17*(3), 321–333. doi:10.1177/1073191109356539

Weeks, J. W., Carleton, R. N., Asmundson, G. J., McCabe, R. E., & Antony, M. M. (2010). "Social Anxiety Disorder Carved at its Joints": Evidence for the taxonicity of social anxiety disorder. *Journal of Anxiety Disorders*, *24*(7), 734-742.

Weeks, J. W., Norton, P. J., & Heimberg, R. G. (2009). Exploring the latent structure of two cognitive components of social anxiety: Taxometric analyses of fears of negative and positive evaluation. *Depression and Anxiety, 26*(2), E40–E48. doi:10.1002/da.20414

Weissman, M. M. (1997). The cross-national epidemiology of panic disorder. *Archives of General Psychiatry, 54*(4), 305. doi:10.1001/archpsyc.1997.01830160021003

Wilson, K. G., Sandler, L. S., Asmundson, G. J., Ediger, J. M., Larsen, D. K., & Walker, J. R. (1992). Panic attacks in the nonclinical population: an empirical approach to case identification. *Journal of Abnormal Psychology*, *101*(3), 460–468. https://doi.org/10.1037//0021-843x.101.3.460

Wolpe, J., & Rowan, V. C. (1988). Panic disorder: A product of classical conditioning. *Behaviour Research and Therapy, 26*(6), 441–450. doi:10.1016/0005-7967(88)90138-6

Zangrossi, H., Del Ben, C. M., Graeff, F. G., & Guimarães, F. S. (2020). Serotonin in panic and anxiety disorders. *Handbook of the Behavioral Neurobiology of Serotonin,* 611–633. doi:10.1016/b978-0-444-64125-0.00036-0

Zangrossi, H., & Graeff, F. G. (2014). Serotonin in anxiety and panic: Contributions of the elevated T-maze. *Neuroscience & Biobehavioral Reviews, 46*, 397–406. doi:10.1016/j.neubiorev.2014.03.007

Zimmerman, M., McGlinchey, J. B., Chelminski, I., & Young, D. (2008). Diagnostic co-morbidity in 2300 psychiatric out-patients presenting for treatment evaluated with a semi-structured diagnostic interview. *Psychological Medicine*, *38*(2), 199–210. https://doi.org/10.1017/S0033291707001717

Zou, Z., Zhou, B., Huang, Y., Wang, J., Min, W., & Li, T. (2020). Differences in cytokines between patients with generalised anxiety disorder and panic disorder. *Journal of Psychosomatic Research*, *133*, 109975.

Zugliani, M. M., Martin-Santos, R., Nardi, A. E., & Freire, R. C. (2017). Personality traits in panic disorder patients with and without comorbidities. *The Journal of Nervous and Mental Disease, 205*(11), 855–858. doi:10.1097/nmd.0000000000000745

Tables

Table 1

Sample demographics

	Percentage	Frequency
Age (M = 38.1, SD = 12.3)		
Gender		
Female	58.7	390
Male	40.5	269
Other	0.8	5
Ethnicity		
White, non-Hispanic	69.9	464
Asian	14.6	97
Black, non-Hispanic	6.9	46
Hispanic	5.7	38
Native American	0.6	4
Other	2.3	15
Education		
GED	1.5	10
High School Diploma	19.9	132
Associate's Degree	18.2	121
Bachelor's Degree	42.0	279
Post-Graduate Education	18.4	122
Country of Residence		
United States	81.9	544
India	10.5	70
United Kingdom	1.7	11
Canada	1.4	9
Other	4.5	30
Total	100	664

Table 2

Descriptive statistics

(n = 663)	Pearson's *r* correlation coefficients						*M*	*SD*	Range	Skewness	Kurtosis
	1	2	3	4	5	6					
PDSS 1	1	-	-	-	-	-	1.72	0.94	0 – 4	1.22	0.91
PDSS 2	.81	1	-	-	-	-	1.77	1.07	0 – 4	1.21	0.51
PDSS 3	.71	.74	1	-	-	-	1.63	0.96	0 – 4	1.50	1.46
PDSS 4	.60	.64	.71	1	-	-	1.71	0.99	0 – 4	1.37	1.19
PDSS 5	.56	.57	.68	.71	1	-	1.54	0.89	0 – 4	1.73	2.53
PDSS 6	.65	.67	.70	.74	.69	1	1.63	0.92	0 – 4	1.45	1.53
PDSS 7	.64	.68	.72	.80	.68	.77	1.70	1.00	0 – 4	1.33	0.84
PDSS Total	.83	.86	.88	.87	.81	.87	4.70	5.79	0 – 28	1.29	1.05

Table 3

Indicator validities and nuisance correlations

	Validity	Within group Pearson's *r* correlation coefficients (taxon/complement)					
Analysis 1		1	2	3	4	5	6
1) PDSS 1	2.22	1	-	-	-	-	-
2) PDSS 2	2.31	.57/.67	1	-	-	-	-
3) PDSS 3	2.30	.44/.23	.48/.28	1	-	-	-
4) PDSS 4	2.32	.18/.04	.29/-.02	.40/.19	1	-	-
5) PDSS 5	1.88	.18/.05	.19/-.03	.45/.02	.47/.27	1	-
6) PDSS 6	2.27	.29/.12	.32/.12	.39/.15	.44/.33	.40/.32	1
7) PDSS 7	2.41	.24/.12	.37/.05	.41/.20	.60/.39	.40/.21	.49/.46
Analysis 2		1	2	3			
1) PDSS 1+2	2.30	1	-	-			
2) PDSS 3	2.54	.52/.28	1	-			
3) PDSS 4+5	2.48	.49/.14	.28/.01	1			
4) PDSS 6+7	2.72	.46/.20	.40/.13	.62/.47			

Note. Pearson's *r* correlation coefficients are presented for taxon and complement groups, separated by a '/'; validity is represented by Cohen's *d*, or the standardized mean difference between taxon and complement groups.

Table 4

Results of taxometric analyses

	Indicators		
	Single-Item	Composite	Recommendation
Sample size (*n*)	663	663	> 300
Taxon size (n_t)	231	231	> 50
Taxon base rate (*P*)	.35	.35	> .10
Number of indicators (*k*)	7	4	> 2
Mean indicator validity (*d*)	2.24	2.51	> 1.25
Mean nuisance correlations			
Taxon (r_{taxon})	.38	.46	< .30
Complement ($r_{complement}$)	.20	.21	< .30
CCFI scores			
MAMBAC	.47	.30	-
MAXEIG	.37	.27	-
L-Mode	.33	.29	-
Mean	.39	.29	-

Note. CCFI scores greater than .55 indicate support for the categorical structure of PD, whereas scores less than .45 indicate support for its dimensional structure; scores between .45 and .55 are considered ambiguous, favoring neither categorical nor dimensional models.

Figures

Figure 1

PDSS score frequency by clinical cutoff

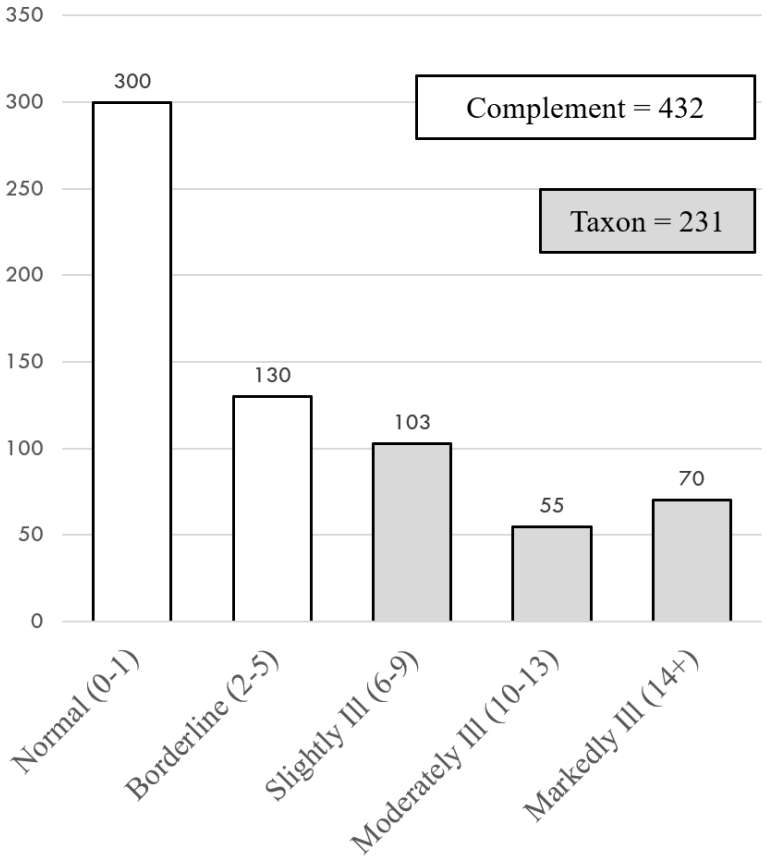


Figure 2

Taxometric plots (single-item indicators)

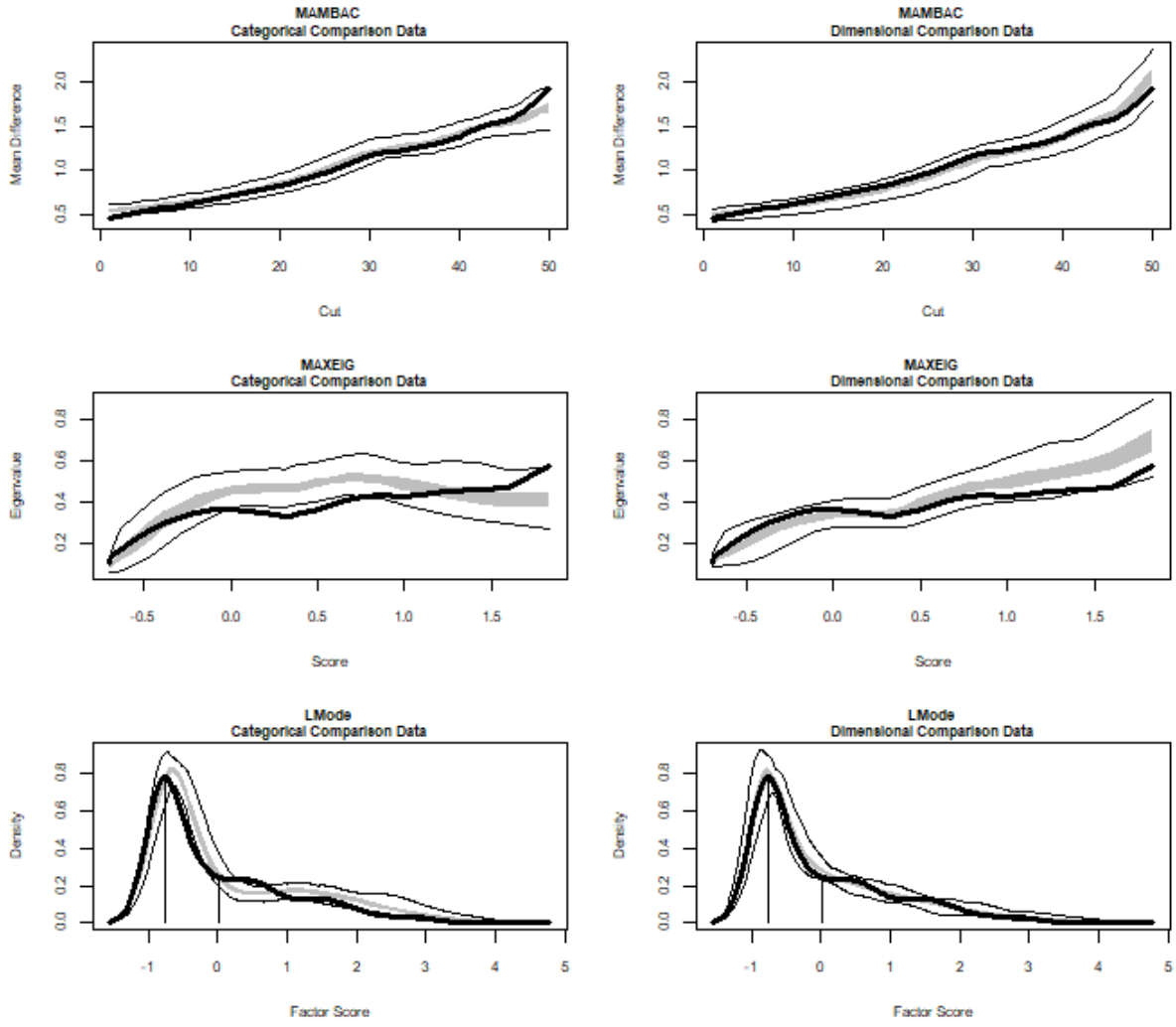
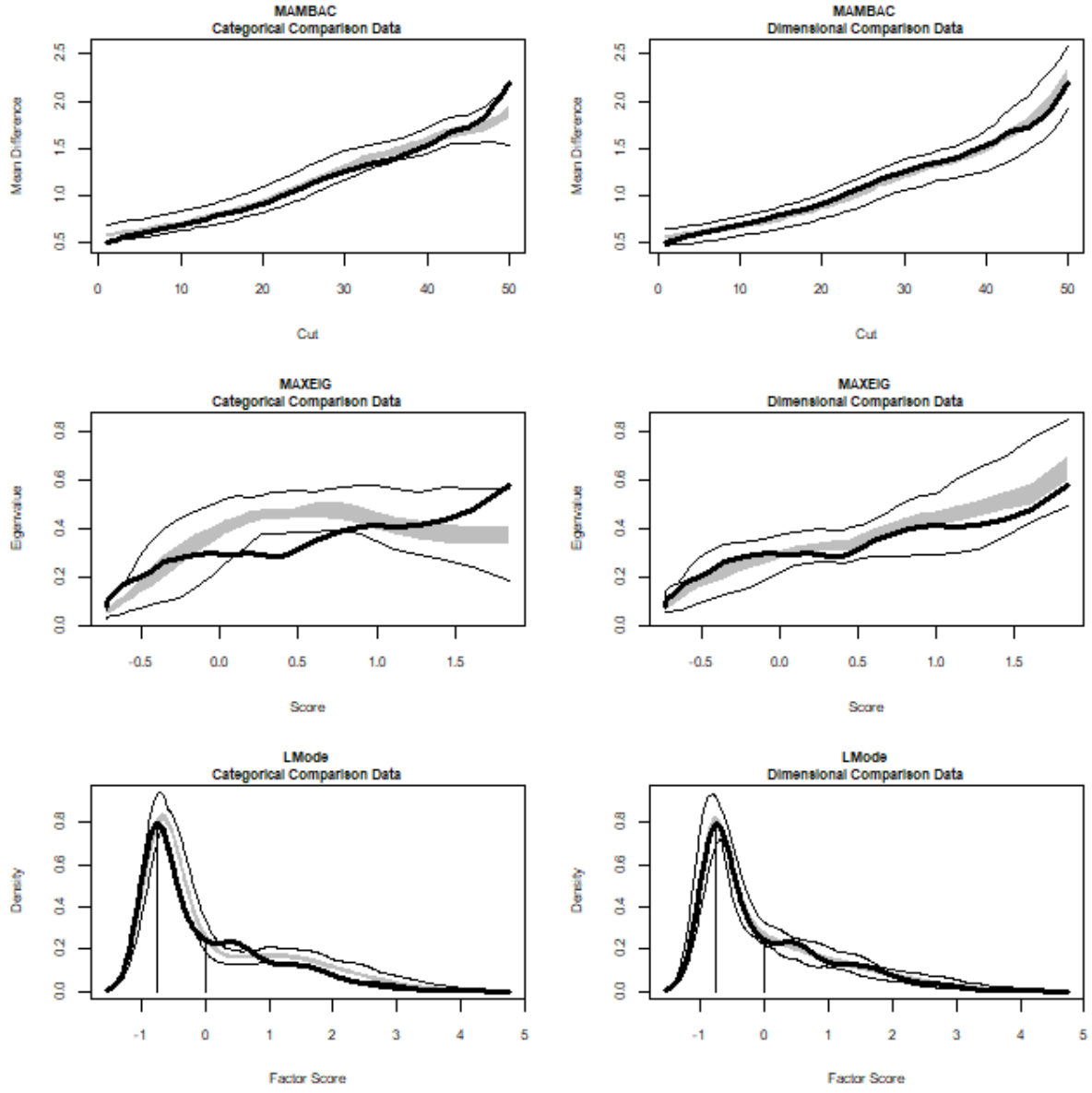


Figure 3

Taxometric plots (composite indicators)



Appendices

Appendix A – Validation Items

1. Please select "Always True" for this item.
2. Please select "Neither Agree nor Disagree" for this item.
3. [How often are you] Sick from drinking gasoline every morning for breakfast.
4. How often do you breathe water?
5. [To what extent do you agree with the statement] I have never used the internet.

Appendix B – Panic Disorder Severity Scale – Self Report Form

Several of the following questions refer to panic attacks and limited symptom attacks. For this questionnaire we define a panic attack as a sudden rush of fear or discomfort accompanied by at least 4 of the symptoms listed below. In order to qualify as a sudden rush, the symptoms must peak within 10 minutes. Episodes like panic attacks but having fewer than 4 of the listed symptoms are called limited symptom attacks. Here are the symptoms to count:

- Rapid or pounding heartbeat
- Chest pain or discomfort
- Chills or hot flushes
- Sweating
- Nausea
- Fear of losing control or going crazy
- Trembling or shaking
- Dizziness or faintness
- Breathlessness
- Feelings of unreality
- Fear of dying
- Feeling of choking
- Numbness or tingling

---

1. How many panic and limited symptoms attacks did you have during the week?

0 No panic or limited symptom episodes

1 Mild: no full panic attacks and no more than 1 limited symptom attack/day

2 Moderate: 1 or 2 full panic attacks and/or multiple limited symptom attacks/day

3 Severe: more than 2 full attacks but not more than 1/day on average

4 Extreme: full panic attacks occurred more than once a day, more days than not

2. If you had any panic attacks during the past week, how distressing (uncomfortable, frightening) were they while they were happening? (If you had more than one, give an average rating. If you didn't have any panic attacks but did have limited symptom attacks, answer for the limited symptom attacks.)

0 Not at all distressing, or no panic or limited symptom attacks during the past week

1 Mildly distressing (not too intense)

2 Moderately distressing (intense, but still manageable)

3 Severely distressing (very intense)

4 Extremely distressing (extreme distress during all attacks)

3. During the past week, how much have you worried or felt anxious about when your next panic attack would occur or about fears related to the attacks (for example, that they could mean you have physical or mental health problems or could cause you social embarrassment)?

0 Not at all

1 Occasionally or only mildly

2 Frequently or moderately

3 Very often or to a very disturbing degree

4 Nearly constantly and to a disabling extent

4. During the past week were there any places or situations (e.g., public transportation, movie theaters, crowds, bridges, tunnels, shopping malls, being alone) you avoided, or felt afraid of (uncomfortable in, wanted to avoid or leave), because of fear of having a panic attack? Are there any other situations that you would have avoided or been afraid of if they had come up during the week, for the same reason? If yes to either question, please rate your level of fear and avoidance this past week.

0 None: no fear or avoidance

1 Mild: occasional fear and/or avoidance but I could usually confront or endure the situation. There was little or no modification of my lifestyle due to this.

2 Moderate: noticeable fear and/or avoidance but still manageable. I avoided some situations, but I could confront them with a companion. There was some modification of my lifestyle because of this, but my overall functioning was not impaired.

3 Severe: extensive avoidance. Substantial modification of my lifestyle was required to accommodate the avoidance making it difficult to manage usual activities.

4 Extreme: pervasive disabling fear and/or avoidance. Extensive modification in my lifestyle was required such that important tasks were not performed.

5. During the past week, were there any activities (e.g., physical exertion, sexual relations, taking a hot shower or bath, drinking coffee, watching an exciting or scary movie) that you avoided, or felt afraid of (uncomfortable doing, wanted to avoid or stop), because they caused physical sensations like those you feel during panic attacks or that you were afraid might trigger a panic attack? Are there any other activities that you would have avoided or been afraid of if they had come up during the week for that reason? If yes to either question, please rate your level of fear and avoidance of those activities this past week.

0 No fear or avoidance of situations or activities because of distressing physical sensations

1 Mild: occasional fear and/or avoidance, but usually I could confront or endure with little distress activities that cause physical sensations. There was little modification of my lifestyle due to this.

2 Moderate: noticeable avoidance but still manageable. There was definite, but limited, modification of my lifestyle such that my overall functioning was not impaired.

3 Severe: extensive avoidance. There was substantial modification of my lifestyle or interference in my functioning.

4 Extreme: pervasive and disabling avoidance. There was extensive modification in my lifestyle due to this such that important tasks or activities were not performed.

6. During the past week, how much did the above symptoms altogether (panic and limited symptom attacks, worry about attacks, and fear of situations and activities because of attacks) interfere with your ability to work or carry out your responsibilities at home? (If your work or home responsibilities were less than usual this past week, answer how you think you would have done if the responsibilities had been usual.)

0 No interference with work or home responsibilities

1 Slight interference with work or home responsibilities, but I could do nearly everything I could if I didn't have these problems.

2 Significant interference with work or home responsibilities, but I still could manage to do the things I needed to do.

3 Substantial impairment in work or home responsibilities; there were many important things I couldn't do because of these problems.

4 Extreme, incapacitating impairment such that I was essentially unable to manage any work or home responsibilities.

7. During the past week, how much did panic and limited symptom attacks, worry about attacks and fear of situations and activities because of attacks interfere with your social life? (If you

didn't have many opportunities to socialize this past week, answer how you think you would have done if you did have opportunities.)

0 No interference

1 Slight interference with social activities, but I could do nearly everything I could if I didn't have these problems.

2 Significant interference with social activities but I could manage to do most things if I made the effort.

3 Substantial impairment in social activities; there are many social things I couldn't do because of these problems.

4 Extreme, incapacitating impairment, such that there was hardly anything social I could do.

Appendix C – Replication With High-Scoring Cases

To test whether the large presence of "non-panickers" within the sample influenced our results, primary analyses were replicated after cutting 20% (n = 133) of the lowest scoring cases from the sample, leaving 530 cases. After classification, the resulting taxon of individuals scoring 6 or higher on the PDSS comprised 44% of the sample (n = 231). Individual indicator validities exceeded recommended thresholds in both indicator sets (mean d's = 1.93 and 2.16), and within-group correlations were comparable to those observed in the primary analyses of single item (r_{taxon} = .38; $r_{complement}$ = .14) and composite item indicator sets (r_{taxon} = .46; $r_{complement}$ = .14). Mean CCFIs of .33 and .23 across procedures for these analyses echoed our initial findings of a dimensional latent structure for PD. Additionally, another analysis was conducted on this reduced sample using more a more stringent cutoff for taxon assignment. Seventy-one of the 530 participants scoring 14 or higher on the PDSS were assigned to the taxon group (P = .13) in this analysis, reflecting "Markedly Ill" cases within the sample (Furukawa et al., 2009). Indicator validities (mean d's = 2.37 and 2.66), but not nuisance correlations (r_{taxon}'s = .22 and .31; $r_{complement}$'s = .46 and 52), met recommendations for analyses in the single-item and composite item indicators, and mean CCFI's of .32 and .27 in these respective groups once again supported dimensionality.

Appendix D – Replication with Conservative Base Rate Classification

To test the extent to which CCFIs would change in response to more conservative classification procedures, both primary analyses were replicated using Furukawa et al.'s (2009) "Markedly Ill" criterion (PDSS scores ≥ 14) for determining taxon membership. The resulting taxon (n = 71) comprised 10.7% of the sample, just above the recommended threshold of 10% required for taxometric analysis (Ruscio et al., 2011). Individual indicator validities were high in both indicator sets (d's > 2.2), though within-group correlations were higher-than-recommended among both single item (r_{taxon} = .22; $r_{complement}$ = .52) and composite indicators (r_{taxon} = .31; $r_{complement}$ = .58). Consistent with our primary analyses, comparison plots favored dimensional models of PD, with CCFI's across procedures and indicator sets ranging from .27 to .40 (mean CCFI's = .34 and .29).

Vita

Christian Alexander Hall was born in Richmond, VA, to Amy Atkins and Steven Hall. He graduated from Mechanicsville High School in June 2012, and subsequently attended the Virginia Polytechnic Institute and State University in Blacksburg, VA, to study the natural sciences. In April 2016, he was awarded the Bachelor of Science degree in Biochemistry. After college, he travelled to Cixi, China, where he taught English as a second language to child and adolescent students from September 2016 to August 2017.

Upon returning to the U.S., Christian moved to Boone, NC, where he joined an Anxiety and Mindfulness research laboratory under the mentorship of Dr. Joshua Broman-Fulks. Encouraged by his mentor, Christian enrolled in the Experimental Psychology Master's program at Appalachian State University in Boone, where he began working as a research assistant, graduate teaching assistant, and research consultant. Since 2018, Christian has been involved in a variety of research projects, including mindfulness-based program evaluations, psychometric studies, and basic research on anxiety and fear-based suffering. He has contributed to a number of academic papers and a book chapter and presented his work at the Association for Behavioral and Cognitive Therapies Conventions in 2018 and 2020.

After earning the M.A. degree for his work at Appalachian State University in May 2021, Christian plans to pursue clinical training and research on interventions for anxiety and fear-based suffering in the Clinical Psychology Ph.D. program at Western Michigan University under the supervision of Dr. Brooke Smith. He will be moving from Boone to Kalamazoo in the autumn of 2021, where he hopes to stay active and in touch with his friends and family living across the country while he earns a doctoral degree in Clinical Psychology.