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# 6 Sensory Dysregulation in Obsessive-Compulsive-Related Disorders

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## Introduction

Obsessive-compulsive related disorders (OCDs) are generally conceptualized as fear-based and impulse-control disorders, in which the pathology is attributed to maladaptive cognitions, difficulty regulating emotions, and impaired self-control. These “top-down” conceptualizations dominate most theoretical models of OCD. However, potentially important “bottom-up” processes have been identified, which may help inform neurodevelopmental models of OCD. From as far back as 1903, Pierre Janet’s early account of obsessive-compulsive disorder (OCD) described the foundation of the illness as a psychasthenic state characterized by feelings of ‘incompleteness’ (Pitman, 1987). Janet depicted incompleteness as a chronic sense of perceptual unease in which the imperfections of the world feel intolerable and states of uniformity and order are constantly sought. While Janet’s model fell out of favor among theorists for nearly a century, findings from recent research warrant reconsideration. A growing body of research has indeed documented abnormal sensory phenomena in OCDs including OCD as well as chronic tic disorder (CTD), body-focused repetitive behaviors (BFRBs) (e.g., trichotillomania, excoriation disorder), body dysmorphic disorder (BDD), and health anxiety disorder (HAD). Evidence suggests these experiences are not epi-phenomenal, but rather that dysregulation of sensory and perceptual processes may precede the clinical onset of OCDs, have significant influence on symptom expression, and therefore may represent a key neurodevelopmental component of OCD etiology.

## Premonitory urges

Although many compulsive behavior symptoms of OCDs are preceded by cognitive or affective events, some are completed without any explicit reason or justification. This is especially the case in children and adolescents, who are still developing insight and meta-cognition (Lewin et al., 2010), or the ability to identify and describe their own thoughts. Instead,

affected persons describe a vague somatic “urge”, “craving”, or “desire” to engage in the compulsion, which is often difficult to tolerate or resist. This is most clearly seen in CTD, as tics are purposeless behaviors that lack obsessional precursors. Persons with CTDs frequently describe “premonitory urges” that precede tics and are temporarily alleviated upon tic execution (Banaschewski et al., 2003; Leckman et al., 1993). Accordingly, theorists posited that premonitory urges may act as negative reinforcers and represent a key factor maintaining tic symptoms. Since the identification of premonitory urges as potentially important aspects of CTDs, research has likewise identified similar premonitory urge phenomena in other OCRDs. These will be discussed in the following sections.

### **Obsessive-Compulsive Disorder (OCD)**

Many individuals with OCD report experiencing abnormal sensory phenomena and premonitory urges that instigate symptoms. In fact, even “cognitive” obsessions in OCD are known to often contain rich sensory features (Porth & Geller, 2018), such as in cases where contamination fears and washing compulsions are brought on, owing to physical sensations that one’s hands are “dirty” or “greasy” (Lapidus et al., 2014; Olatunji et al., 2007; Shapira et al., 2003; Stein et al., 2001). Explicit sensory experiences and urges that occur outside of cognitive obsessions are described in a variety of ways, including “not just right” experiences (Coles et al., 2003; Diniz et al., 2006; Ecker & Gönner, 2008; Ghisi et al., 2010; Leckman et al., 1994; Lee et al., 2009; Moretz & McKay, 2009; Summers et al., 2014), feelings of “incompleteness” (Ecker & Gönner, 2008; Ghisi et al., 2010; Pietrefesa & Coles, 2009; Pitman, 1987; Summerfeldt, 2004), and somatic urges for action (da Silva Prado et al., 2008; Ferrao et al., 2012; Miguel et al., 2000). “Not just right” experiences (NJREs) reflect an inner feeling that something is wrong, uncertain, or not under control, which must be corrected. “Incompleteness” experiences are similar to NJREs, often reflecting an inner feeling that something is wrong and that one must perform a compulsion or ritual until achieving a sense of completion (Pietrefesa & Coles, 2009; Pitman, 1987). Premonitory urges in OCD come in many forms and resemble those from CTDs, having been described as aversive bodily sensations (e.g., tension) or energy surges (da Silva Prado et al., 2008; Ferrao et al., 2012; Miguel et al., 2000; Rosario et al., 2009). These different types of sensory and/or urge phenomena are often referred to interchangeably. Although NJREs are common among non-clinical, healthy individuals (Coles et al., 2003; Ghisi et al., 2010) and are found in non-OCRD pathologies such as anxiety disorders (Fergus, 2014), most evidence suggests a strong relationship between NJREs and OCD (Cervin et al., 2020; Coles et al., 2003; Sica et al., 2015; Summers et al., 2014). Indeed, one study found that NJREs were the strongest feature differentiating youth with OCD from

youth with anxiety disorders (Cervin et al., 2021). There is mixed data regarding the prevalence of NJREs and similar sensory phenomena in adult OCD patients (Ghisi et al., 2010; Leckman et al., 1994; Shavitt et al., 2014; Sibrava et al., 2016). However, some evidence suggests NJREs are more common in childhood OCD than adult-onset OCD (Ferrao et al., 2012). It is important to note that OCD patients are significantly bothered by sensory-related obsessions and premonitory urges, as many report greater distress and impairment from these experiences as compared to cognitive obsessions and compulsive behaviors (da Silva Prado et al., 2008; Ferrao et al., 2012).

### **Chronic Tic Disorders (CTD)**

As previously mentioned, premonitory urges are widely recognized as important contributors to CTDs. Evidence shows that most CTD patients (~90 percent) report some type of premonitory urge experience, however children report urges less frequently (Leckman et al., 1993) and the prevalence of urges increases with age (Sambrani et al., 2016) (discussed later in Chapter). The urge often occurs in the same body area as the tic it precedes, but they can be felt as generalized across the whole body (Miguel et al., 1995). A significant portion of persons with CTDs also report NJREs and feelings of incompleteness, with estimates ranging between 30–90 percent (Leckman et al., 1994; Miguel et al., 2000; Neal & Cavanna, 2013).

### **Body-Focused Repetitive Behaviors (BFRBs)**

Tracing back to early behavior analytic studies in youth, BFRBs have long been understood to be triggered by sensory experiences and maintained by sensory feedback (Miltenberger et al., 1998; Rapp et al., 1999; Williams et al., 2007). Sensory cues for hair pulling and skin picking include visual and tactile sensations, such as undesired colors (e.g., coarse hairs, blemishes) or other aesthetic qualities (e.g., rough skin imperfections or “out of place” hair) (Grant et al., 2007; Mansueto et al., 1997; Snorrason et al., 2015; Snorrason et al., 2019; Snorrason et al., 2010; Wilhelm & Margraf, 1993). Additionally, a recent study documented heightened incompleteness experiences in persons with skin picking disorder (Ricketts et al., 2021). Upon symptom completion, most individuals report experiencing a sense of pleasure, gratification, or relief (Bohne et al., 2005; Christenson et al., 1991; Keuthen et al., 2000; Meunier et al., 2009; Tucker et al., 2011; Woods et al., 2006). Our understanding of premonitory urge phenomena in BFRBs is limited, particularly in children. However, case reports show that affected youth experience urge phenomena (Pinto et al., 2017; Swedo & Rapoport, 1991), and the incidence of affected youth reporting urge phenomena appears to increase with age (Panza

et al., 2013; Schumer et al., 2015) until the experience is nearly universal (~80 percent) in affected adults (Dieringer et al., 2019).

### **Body Dysmorphic Disorder (BDD)**

BDD is characterized by a disruption of healthy body image wherein affected individuals obsess over a perceived flaw in their physical appearance and engage in compulsive rituals designed to disguise or eliminate the perceived flaw (e.g., mirror checking, excessive grooming, make-up application, reassurance seeking). BDD typically onsets during early adolescence (Phillips et al., 2005), coinciding with developmentally appropriate increases in interest toward sexual attraction and body aesthetics. Urges to engage in appearance checking or modification have not been identified in BDD, but persons with BDD do exhibit visual abnormalities related to distortions of self-perception (Li et al., 2013). BDD has also been associated with NJREs and feelings of incompleteness. A study by Summers and colleagues found that (a) persons with BDD experience more severe NJREs than healthy individuals; (b) there is a positive correlation between severity of incompleteness/NJREs and BDD symptoms; and (c) physiological reactivity in response to a task designed to elicit body concerns was positively correlated with incompleteness severity (Summers et al., 2017). Furthermore, that research group found that individuals with BDD exhibit greater discomfort than healthy controls when exposed to appearance-related (i.e., drawing of a man with crooked facial features) and non-appearance-related visual NJRE stimuli (i.e., a cluttered table).

### **Health Anxiety Disorder**

Health anxiety disorder (HAD) is characterized by persistent preoccupation with bodily sensations, which are deemed abnormal or pathogenic, and a resulting conviction that one has or might have a serious medical problem. Very little research has been conducted on HAD in youth, but the disorder certainly affects persons across the lifespan (Wright & Asmundson, 2003). Urge phenomena have not been documented in HAD. However, studies have consistently linked HAD to excessive focus on and misinterpretation of bodily sensations, with affected persons experiencing normal body sensations as more intense or more aversive than healthy individuals (Barsky & Klerman, 1983). Theorists have speculated that the somatic amplification of body signals in BDD is due to misattribution (i.e., stomach tension being misinterpreted as pain), or cognitive biases toward illness. However, there may also be some perceptual abnormality that supports amplification of body signals in HAD.

## **Altered Sensation and Perception in OCRDs**

### ***Interoception***

Interoception refers to the process by which one detects, integrates, and interprets internal body sensations (Craig, 2002; Khalsa et al., 2018; Tsakiris & Critchley, 2016). Appropriate awareness of body sensations is critical for maintaining homeostasis as well as selecting and executing adaptive behaviors. Indeed, interoceptive dysfunction is thought to exert widespread negative effects on a variety of psychological processes such as attention, decision-making, and emotion (Craig, 2002). In children, abnormally increased cardiac sensitivity has been linked to anxiety symptoms (Eley et al., 2004), while decreased cardiac sensitivity has been linked to obesity and picky eating (Koch & Pollatos, 2014; Mata et al., 2015). Research has furthermore documented a range of abnormal interoceptive processes across psychiatric populations that overlap and co-occur with OCRDs, including autism spectrum disorder, attention-deficit/hyperactivity disorder, feeding and eating disorders, and oppositional defiant disorder (Brewer et al., 2021; Gourley et al., 2013; Pollatos et al., 2008).

In OCRDs, a vast majority of research has been conducted in adults, but interoceptive abnormalities have been reported consistently. Objective investigations using heartbeat detection tasks and similar methods have consistently found reduced interoceptive accuracy across OCRDs (Barsky et al., 1995; Demartini et al., 2021; Ganos et al., 2015; Krautwurst et al., 2014; Krautwurst et al., 2016; Kunstman et al., 2016; Lazarov et al., 2014; Pile et al., 2018; Pratt, 2014; Rae et al., 2019; Schultchen et al., 2019; Tyrer et al., 1980). Obsessive-compulsive traits in non-clinical populations have also been linked to reduced propensity to utilize interoceptive signals to gauge arousal (Lazarov et al., 2010), suggesting OCRDs may be associated with deficient interoceptive accuracy and increased reliance on external proxies. In contrast, research generally indicates that persons with OCRDs appraise bodily sensations as overly intense or threatening (Deacon & Abramowitz, 2006; Eng et al., 2020), with self-report studies documenting perceived increases in interoceptive sensitivity in OCD (Eng et al., 2020; Jokić & Purić, 2021), CTD (Eddy et al., 2014; Rae et al., 2019), BFRBs (Teng et al., 2002; Woods et al., 1996), BDD (Grunewald et al., 2023), and HAD (Krautwurst et al., 2016). These findings suggest that certain, danger-related interoceptive signals may be exaggerated in OCRDs, owing possibly to hyperarousal and/or fear-related attentional bias.

### ***Exteroception***

The perception of environmental stimuli, or exteroception, has received less attention than interoception for its role in OCRDs and other child psychopathologies. Still, extant data generally indicate that some children

exhibit markedly elevated reactivity to and avoidance of benign environmental stimuli (e.g., sock seams, perfumes) (Dunn & Westman, 1997). Children who exhibit sensory hypersensitivity appear to have elevated risk for psychiatric disorders, particularly anxiety disorders, attentional disorders, and disruptive behavior disorders (Andersson et al., 2008; Bar-Shalita et al., 2008; Conelea et al., 2014; Gourley et al., 2013; Gouze et al., 2009; Keuler et al., 2011; Perez-Robles et al., 2013). However, there is at least one conflicting report showing no association between childhood sensory abnormalities and psychopathology (Van Hulle et al., 2012).

Similar to findings related to self-reported interoception, research has consistently revealed self-reported increases in sensitivity to external stimuli in OCRDs including OCD (Ben-Sasson & Podoly, 2017; Cervin, 2023; Dar et al., 2012; Lewin et al., 2015; Rieke & Anderson, 2009), CTDs (Cohen & Leckman, 1992; Isaacs et al., 2020; Ludlow & Wilkins, 2016; Soler et al., 2019), BFRBs (Falkenstein et al., 2018; Houghton et al., 2018), and HAD (Barsky & Wyshak, 1990; Barsky et al., 1990). To my knowledge, no study has examined external sensory sensitivity in BDD, but there is evidence of enhanced aesthetic sensitivity and attention to detail (Kaplan et al., 2013; Madsen et al., 2013). There also appears to be a high degree of overlap between emergent conditions characterized by sensory intolerance (e.g., Misophonia, “Sensory Processing Disorder”) and OCRDs, as studies have documented elevated rates of OCRDs among samples of patients with sensory intolerance (Smith et al., 2022; Taylor et al., 2014; Wu et al., 2014), including among childhood samples (Van Hulle et al., 2019). Yet, investigations of external sensory sensitivity utilizing objective measurement tools largely mirror those from investigations of interoceptive sensitivity. Several studies found no evidence of abnormal sensory thresholds in OCRDs (Barsky et al., 1995; Belluscio et al., 2011; Güçlü et al., 2015; Haenen et al., 2010; Puts et al., 2015) or altered pain thresholds/tolerance (Blum et al., 2017; Grant et al., 2017; Lautenbacher et al., 1998). However, a minority of studies have reported anomalous findings such as decreased tactile detection thresholds in BFRBs (Houghton et al., 2019) and HAD (Rodic et al., 2016), poor olfaction in OCD (Segalas et al., 2011) and CTD (Kronenbuerger et al., 2018), as well as increased physiologic reactivity and slow habituation to aversive external stimulation in OCD (Janik et al., 2018; Podoly et al., 2022).

### **Models of sensory dysregulation in OCRDs**

Currently available evidence shows that symptoms of OCRDs are frequently maintained by sensory phenomena such as premonitory urges, that persons with OCRDs perceive themselves to be abnormally sensitive to interoceptive and exteroceptive signals, but that there is little evidence for objective physiological sensory abnormalities. Given these mixed

findings, it is presently unclear whether abnormal sensory experiences in OCRDs are functionally significant and related to a causal etiologic mechanism. However, there are several promising explanations for various sensory phenomena in OCRDs, which collectively point to disruptions in sensory processing and integration between top-down cognitive influences and bottom-up sensory afferents.

### ***Sensory Gating***

Although few studies have identified objective sensory abnormalities in OCRDs such as reduced detection thresholds or heightened interoceptive awareness, research has consistently identified one objective sensory deficit among OCRDs. Sensory gating is an adaptive process in which some sensory information is selectively inhibited before reaching conscious awareness, which is believed to sharpen perception, conserve attentional resources, and enhance focus on relevant sensory information while mitigating background noise. Indeed, the peripheral nervous system has an immense sum of sensory receptors that are rarely, if ever, at complete rest, meaning they are constantly conveying afferent sensory messages to the brain. The brain then must decipher information from torrents of afferent sensory inputs that occur in complex temporal patterns. If some filtering of sensory inputs did not occur, one would be overwhelmed with cascades of mostly irrelevant information, which would likely impede perception, attention, cognitive operations, and adaptive behavior. Furthermore, adaptive sensory gating appears to mature as children age (Davies et al., 2009), suggesting that developmental adversities may hinder gating efficiency and increase risk for psychopathology.

Faulty sensory gating has been closely tied to psychotic-spectrum disorders (McGhie & Chapman, 1961; Patterson et al., 2008), attentional disorders (Micoulaud-Franchi et al., 2019; Micoulaud-Franchi et al., 2015), as well as “sensory processing disorders” (SPDs) (Davies et al., 2009). SPDs are a purported childhood-onset disorder characterized by excessive (or reduced) exteroceptive sensitivity (Houghton et al., 2020) (discussed below). Reduced gating has also been documented in OCRDs, including OCD (Hashimoto et al., 2008; Rossi et al., 2005; Xiao et al., 2010), CTDs (Castellanos et al., 1996; Swerdlow et al., 2001; Zebardast et al., 2013), BFRBs (Houghton et al., 2019), and BDD (Giannopoulos et al., 2021; Kapsali et al., 2020). No research has yet linked sensory gating to HAD, but experts have posited a role for deficient gating among persons with HAD and similar somatoform disorders (Boutros & Peters, 2012). Impaired sensory gating may contribute to symptoms of OCRDs by allowing excessive irrelevant sensory afferent information to reach conscious awareness, which results in feelings of over-inundation (i. e., hypersensitivity) and difficulty habituating to stimulation. Overstimulation does tend to elicit compensatory behaviors designed to



distract from aversive inputs and replace such discomforts with more pleasurable stimuli (Dunn, 2001), and others have suggested that faulty sensory gating could disrupt action selection and suppression of unwanted behaviors (Koziol et al., 2011).

### **Models of premonitory urges**

Substantial evidence indicates that premonitory urges are governed by activity in limbic sensory and motor areas, specifically the insular and cingulate cortices (Jackson et al., 2011). Indeed, Jackson and colleagues demonstrated that neural substrates of premonitory urges in CTDs align closely with those of normal urges for action from our everyday lives (e.g., urge to yawn). The role of the insula in generating urges for action, as well as pathological urges to engage in compulsive behavior, was most clearly demonstrated in a study that found insula lesions among nicotine addicts were associated with a complete elimination of the urge to smoke (Naqvi & Bechara, 2009; Naqvi et al., 2007). Unfortunately, this neurobiological account for premonitory urges fails to explain the development of premonitory urges in OCRDs.

Behavioral models of the development of premonitory urges are generally based on the notion that tension reduction, or negative reinforcement, maintains the urge-tic relationship. This is generally supported by reinforced tic suppression studies in which tic rate increases when urges are strong, urge strength increases during tic suppression, and urge strength attenuates during free-to-tic conditions (Brandt et al., 2016; Houghton et al., 2014; Langelage et al., 2022). However, the negative reinforcement hypothesis does not account for the fact that premonitory urges are not always present when tics first emerge in childhood (Sambrani et al., 2016; Woods et al., 2005). Tic typically onset around ages 6–7, whereas awareness of premonitory urges can emerge up to three years after tic onset (Banaschewski et al., 2003; Leckman et al., 1993). Woods and colleagues proposed that urges may be present in young children with CTDs, but that younger children cannot reliably notice and describe the urge experience (Woods et al., 2005). This is supported by research showing that interoceptive abilities typically do not fully develop until the ages of 5.8–7.8 years (Sigmundsson et al., 2000). Indeed, recent findings suggest that 80–95 percent of children with CTDs experience some degree of a premonitory urge experience (Openneer et al., 2020). Because sensory experiences can be made conscious or more salient through learning (Cameron, 2001), perhaps the sequelae of tics (e.g., change in proprioception, distraction, pain, teasing) provide the feedback necessary to increase awareness toward private sensations that precede tics. Upon becoming aware that some internal sensation exists prior to the tic, the urge then transitions from a benign experience with no functional significance to one that is functionally linked to the tic and tic-related

consequences (e.g., discomfort, distraction, teasing). Finally, just as other benign stimuli can acquire aversive valence when such stimuli predict a negative outcome (Kamin et al., 1963; Rescorla & Solomon, 1967), the urge then acquires an aversive valence, and escape from the urge becomes reinforcing. This conceptualization is supported by studies showing that premonitory urges are associated with neural activation of regions involved in negative affect and punishment-based learning (Wang et al., 2011), as well as data showing that urge severity is associated with indices of functional and social impairment (Woods et al., 2005) and peer victimization (Zinner et al., 2012).

Taken together, premonitory urges for compulsive behaviors may be universally present as signals of impending action, but as compulsive behavior symptoms become intrusive and individuals attempt to predict and suppress such actions, urges acquire aversive valence and become functionally tied to symptoms. Premonitory urges can thus be considered “normal”, in that they mirror common urges for action, but only because they signal the emergence of undesired symptoms do they acquire aversive valence. In the next section, the process through which benign interoceptive signals can intensify and become aversive is discussed.

### **The “alliesthesia” and somatosensory amplification models**

Some have suggested that abnormal sensory experiences in OCDs could be caused by a pathogenic integration between higher-order cognitive processes and lower-order perceptual afferents (Paulus & Stein, 2010). Specifically, Paulus and Stein argued that fear-based disorders (e.g., anxiety, OCD, BDD, HAD) are characterized by negatively biased internal cognitive models that facilitate hypervigilance to potential threats (Stein & Nesse, 2011), translating to hyper-attention toward sensory stimuli that may signal danger. Similarly, persistent hypervigilance to threat appears to be tied to evolutionary processes related to early threat detection and precautionary responses. By means of a process known as alliesthesia (Cabanac, 1971), the internal condition of an individual is intertwined with external stimuli that impact the internal environment, causing stimuli to acquire an emotional significance based on their perceived pleasantness or unpleasantness. The central and peripheral nervous systems of the body continuously transmit a stream of noisy and unclear sensory inputs that require integration and interpretation. Individuals who are vigilant for threats tend to perceive stimuli with negative emotional impact as more intense, as these stimuli could potentially indicate danger. Coupled with faulty sensory gating and an increase in the amount of irrelevant information reaching conscious awareness, affected persons may constantly struggle to interpret the threat relevance of various ambiguous suprathreshold stimuli. Consequently, ambiguous stimuli have the potential to trigger anxious catastrophizing, whereby a seemingly harmless

sensory encounter (e.g., a buzzing noise) is perceived as unusually intense or even catastrophic (e.g., “There might be a swarm of bees in this room!”).

Perhaps this model could be extended beyond the mischaracterization of threat-related stimuli in anxiety/OCD/HAD/BDD and into other OCRDs such as BFRBs and CTDs. Indeed, arguments have been made for the role of alliesthesia, threat prediction, and reward anticipation in similar compulsive behavior disorders, drug addiction. Paulus and Stewart (Paulus & Stewart, 2014) argued that persons with substance use disorders encounter an “embodied” experience, similar to craving, that is subserved by frequent errors between their predicted versus actual body state (e.g., “elated” versus “tense”). Furthermore, an innovative study utilizing a bayesian computational model of neurophysiological data from individuals with various psychopathologies (i.e., anxiety, depression, substance use disorders, and eating disorders) found increased interoceptive error rates across all pathologies as compared to controls during interoceptive challenge (a combined breath-holding and heartbeat-counting task) (Smith et al., 2020). It could therefore be that compulsive tics and BFRBs are associated with a similar interoceptive reward-prediction error, specifically related to proprioceptive, visceral, and dermatological sensations.

Unfortunately, the models proposed by Paulus and colleagues have been concerned with interoception and do not deliver an account of exteroceptive hypersensitivity. Yet, from the perspective of health anxiety and similar unexplained medical symptom disorders (i.e., conversion disorder, somatization disorders, psychogenic non-epileptic seizures), experts have explored how the intensity of both internal and external sensory experiences can be amplified, even in the absence of objective differences in sensory thresholds (Barsky & Wyshak, 1990; Köteles & Witthöft, 2017). Somatosensory amplification refers to the notion that a combination of body hypervigilance and strong emotional reactivity to sensation can result in the intensification of certain sensory experiences. This concept shares many similarities with other models of medically unexplained symptoms such as central sensitization in chronic pain. Because external stimuli can affect the internal milieu, it stands to reason that certain environmental exposures (i.e., a strange smell) can trigger health worries and undergo amplification. This has important theoretical implications for emerging conditions that overlap with OCRDs, namely SPDs and “idiopathic environmental intolerances” (IEI). As has been discussed in other reviews (Houghton et al., 2020), children with sensory processing disorders almost universally express elevated symptoms of OCRDs and anxiety, thus suggesting that environmental hypersensitivity in sensory processing disorders could result from top-down amplification of sensory inputs. Likewise, controversial conditions characterized by IEI such as misophonia (selective sound sensitivity), multiple chemical (olfactory)

sensitivity, and electromagnetic sensitivity share many characteristics with OCRDs and could potentially be conceptualized under this model. Support for this notion comes from data indicating that top-down belief systems significantly influence IEI symptom expression. For example, in persons with purported electromagnetic sensitivity, their actual exposure to electromagnetism does not influence symptom severity (Rubin et al., 2010), but perceived electromagnetism exposure does influence symptoms (Szemerszky et al., 2015; Witthöft & Rubin, 2013)

### ***Issues for further investigation***

This chapter has provided evidence for premonitory urge phenomena as well as subjective increases in sensitivity to interoceptive and exteroceptive stimuli. I also discussed several preliminary models for sensory dysregulation in OCRDs. Yet, this area of research is still early stages, with many unanswered questions and untested assumptions. The following sections will detail several important avenues for further research.

### **Sensory processing disorders?**

As discussed previously and in other reports (Houghton et al., 2020), researchers and clinicians have increasingly argued for the existence of SPDs. These conditions are not recognized by DSM-5 and ICD-11, and it is presently unclear whether these conditions represent valid diagnoses or are best conceptualized as symptom clusters that often co-occur with anxiety-related pathologies and autistic traits. For instance, many children exhibit select hypersensitivities and strong responses to certain stimuli (e. g., food textures, smells, clothing material), which do not necessarily impair functioning and may vanish with age. However, there is some evidence that children with SPDs have objective sensory abnormalities (Bar-Shalita et al., 2009; McIntosh et al., 1999; Schaaf et al., 2003), potentially supporting the validity of SPD as a unique pathology. However, other research points to symptoms of SPDs in children as representing early risk factors for anxiety disorders and related pathologies (Carpenter et al., 2019). For example, a longitudinal twin study in 1,613 children found that the number of tactile or auditory sensory over-responsivity symptoms was positively correlated with likelihood of OCD diagnoses (Van Hulle et al., 2019).

In order to significantly advance research on SPDs and their relationships with OCRDs, future studies ought to take certain steps to maximize impact and interpretability. First, it is crucial that scientists establish consensus in defining the core constructs of sensory psychopathology. Once clear definitions of pathological sensory constructs (e.g., “sensory over-responsivity”) and the diagnostic validity of sensory processing disorders have been established, researchers can develop psychometrically

valid assessment strategies. Relatedly, future studies ought to utilize both subjective and objective assessment methods and clearly differentiate between the two processes. Objective measures of sensory function sometimes align with self-reported perception (Bar-Shalita et al., 2009), but this is not always the case (Kozak & Miller, 1982). Careful attention to both components of the large sensory processes apparatus should enable more precise conclusions regarding the interplay between conscious feeling states, beliefs, physiological responses to stimulation, and behavior. Finally, the neurophysiological deficits that purportedly underlie SPDs may interact significantly with various environmental factors and comorbidities, such as physical health (e.g., nutrition), neuroendocrine/hormonal processes, motor development, and cognitive and social experiences (i.e., exposure to diverse, highly stimulating environments).

## **Treatment**

While sensory dysregulation has garnered attention as a potential OCRD mechanism, there is little empirical guidance on effective treatments, and the limited but existing evidence suggests the need for further refinement of existing treatments. There are behavioral treatment strategies aimed at children with purported sensory processing disorders, such as “sensory integration therapy”. Unfortunately, sensory integration therapy has not undergone rigorous clinical trials, and existing evidence points to questionable efficacy (Complementary et al., 2012). Cognitive-behavioral therapies for OCRDs, which boast considerable empirical support, have been investigated regarding effects on patients who exhibit sensory dysregulation. A meta-analysis of CBT for incompleteness symptoms in OCD found that incompleteness was modestly improved over the course of treatment, with stronger effect sizes noted for treatments that explicitly targeted incompleteness (Schwartz, 2018). In contrast, other studies have shown that NJREs and incompleteness symptoms are associated with poor OCD treatment outcomes (Cervin & Perrin, 2021; Nissen & Parner, 2018). Given these data, there is a clear need to further investigate the efficacy of CBT-based interventions for sensory components of OCRDs.

There is perhaps room for innovative new treatment methodologies that are agnostic to diagnosis but instead aim directly at sensory dysregulation. One example of such an approach is known as Flotation-REST (Reduced Environmental Stimulation Therapy), which involves using sensory deprivation in a saltwater floatation chamber to encourage mindfulness meditation and relaxation. Early data point to robust anxiolytic effects of Flotation-REST (Feinstein et al., 2018), as well as improvements in interoceptive awareness (Feinstein et al., 2018) and decreased functional connectivity within somatomotor and default mode networks (Al Zoubi et al., 2021). Other potential treatment avenues include non-invasive neuromodulation, particularly targeting the insula

(Ibrahim et al., 2019). However, preliminary investigation of insular neuromodulation via transcranial magnetic stimuli showed treatment-related decreases in interoceptive accuracy but increases in perceptual confidence (Pollatos et al., 2016). It is currently unclear how such treatment effects would translate into OCD patients and whether approaches that are suitable for adults would exhibit similar efficacy in youth.

## Summary

Research reviewed in the current chapter demonstrates that symptoms of many OCDs are instigated by sensory phenomena, affected individuals exhibit subjective sensory abnormalities, but that sensory abnormalities are not necessarily caused by objective sensory deficits. However, there is evidence for more subtle sensory gating abnormalities and disrupted sensory integration processes in OCDs. Taken together, this evidence shows that sensory dysregulation is a potentially important component of OCD psychopathology. It is therefore unsurprising that research in this area is growing at a rapid pace, and hopefully we will receive significant new insights that will help develop a more comprehensive understanding of how sensory dysregulation interacts with other risk factors and etiological mechanisms. Within these efforts, there is a clear need for additional research on targeted and innovative treatment approaches for sensory aspects of OCDs. Such research would significantly advance our ability to provide comprehensive care for individuals with OCDs and may also have significant implications for conditions similar to OCDs that also exhibit similar sensory abnormalities (Khalsa et al., 2018).

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