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Neural Correlates of Sensory Abnormalities Across Developmental Disabilities

Garrett J. Cardon

Department of Psychology, Colorado State University, Fort Collins, CO, United States

cardong@colostate.edu

Abstract

Abnormalities in sensory processing are a common feature of many developmental disabilities (DDs). Sensory dysfunction can contribute to deficits in brain maturation, as well as many vital functions. Unfortunately, while some patients with DD benefit from the currently available treatments for sensory dysfunction, many do not. Deficiencies in clinical practice surrounding sensory dysfunction may be related to lack of understanding of the neural mechanisms that underlie sensory abnormalities. Evidence of overlap in sensory symptoms between diagnoses suggests that there may be common neural mechanisms that mediate many aspects of sensory dysfunction. Thus, the current manuscript aims to review the extant literature regarding the neural correlates of sensory dysfunction across DD in order to identify patterns of abnormality that span diagnostic categories. Such anomalies in brain structure, function, and connectivity may eventually serve as targets for treatment.

1. INTRODUCTION

Sensory dysfunction is a common factor in a large proportion of individuals with a variety of developmental disabilities (DD; Engel-Yeger & Ziv-On, 2011; Ermer & Dunn, 1998; Levit-Binnun, Davidovich, & Golland, 2013; Liss, Mailloux, & Erchull, 2008), even though such symptomology is only recently becoming diagnostically relevant in some DDs (e.g., Autism Spectrum Disorder; ASD; American Psychiatric Association, 2013). Additionally, there is a great deal of overlap in the clinical presentation of sensory abnormalities across diagnostic lines (Acevedo, Aron, Pospos, & Jessen, 2018; Koziol, Budding, & Chidekel, 2011). Similarities or duplication in the manifestations of sensory symptoms across diagnostic categories may suggest common neural mechanisms that subserve these abnormalities, regardless of diagnosis. Furthermore, sensory irregularities can be extremely debilitating to patients and have the potential to alter development of the brain and affect fundamental behavioral domains such as communication, academics, social interaction, cognitive function, attention, memory, and emotional responsivity (Ashburner, Ziviani, & Rodger, 2008; Ben-Sasson, Carter, & Briggs-Gowan, 2009; Butler et al., 2009; Cascio, Moore, & McGlone, 2018; Cosbey et al., 2010; Engel-Yeger, Hardal-Nasser, & Gal, 2011; Hannant, Tavassoli, & Casidy, 2016; Linke et al., 2018).

Unfortunately, the majority of existing treatments for sensory abnormalities, while effective for some, are inconsistent and not backed by neurobiological evidence. Given the incidence and overlap of sensory abnormalities across developmental disabilities, their potential

impacts on patient outcome, and the need for better treatments, increasing our understanding about their neuroanatomic and -physiologic underpinnings could improve the basic conceptualization of these disorders and lead to advancements in clinical practice. This notion is particularly true, given the estimated rise in prevalence of DD by the Center for Disease Control (CDC; Zablotsky et al., 2017) and the emphasis the National Institute of Mental Health has put on understanding the biological bases of clinical symptoms, regardless of diagnosis (i.e., Research Domain Criteria (RDoC) initiative; Kozak & Cuthbert, 2016). Thus, the current manuscript aims to review the extant literature concerning the similarities and differences in the neural underpinnings of sensory dysfunction across DD in order to further understanding of the basic mechanisms that lead to these symptoms and identify potential targets for their treatment.

The structure of the current review includes, first, a discussion of several general principles vital to the topic of brain development and sensory processing. Sensory loss, and its effects on brain development, as well as Sensory Processing Disorders (SPDs), are both discussed in this section. Following this introductory segment, sensory processing findings along with their (possible) neural underpinnings are discussed for three other common DDs: Autism Spectrum Disorder (ASD), Attention Deficit Hyperactivity Disorder (ADHD), and Schizophrenia (SCHZ). Finally, a cataloguing and comparison of sensory processing results across DDs is undertaken. In order to review the various literature required to obtain information about the disparate topics that are integrated in the current review, PubMed searches using a host of search terms were performed. For instance, the full name or abbreviation for each of the aforementioned disorders, plus the terms ‘sensory’, ‘sensory abnormality’, ‘sensory dysfunction’, ‘sensory processing’, ‘sensory sensitivity’ were initially used to compile articles relevant to the topic of abnormal sensory processing for each disorder. During these searches, many articles were discovered that included neuroanatomical and/or -physiological methods. Further search terms and publications were derived from these articles via reuse of keywords and prominent terms and investigation of citations. Finally, a number of articles were found which formally compared results from behavioral, neurologic, or both types of method between two or more DDs. Where relevant to the above DDs, these articles were catalogued and used in determining patterns of similarities and differences in the neural correlates of abnormal sensory processing across these DDs. The results of this effort are summarized in Table 1.

1.1 The Role of Sensory Processing in Development: General Principles

Normal sensory function is fundamental to typical brain development. The initial organization of the cerebral cortex is driven primarily by intrinsic (e.g., genetic) factors (Pallas, 2001). However, following these early foundational steps, sensory input further motivates, informs, and refines cerebral maturation. For instance, extrinsic influences largely determine which connections survive the synaptic pruning in cortical development (Hebb, 1949; Huttenlocher & Dabholkar, 1997). Additionally, it appears that normal sensory activity is positively related to the onset and increase of inhibitory activity throughout the brain during development (Foeller, 2004), which is instrumental in establishing the proper balance of excitation and inhibition and the initiation and closing of sensitive periods needed for mature function (Dorrn, Yuan, Barker, Schreiner, & Froemke, 2010; Hensch, 2005;

Uhlhaas & Singer, 2012). One important aspect of brain development that appears to be mediated by inhibitory function is synchronization of neural oscillations within and between brain areas (esp. in the gamma oscillatory band; Uhlhaas & Singer, 2006, 2012; Uhlhaas et al., 2010). Lack of this temporal coordination has the potential to lead to malformation of canonical brain networks, such as the default mode network (DMN), which could lead to reduced coordination of global function, as well as isolation of local processors. Such processes could contribute to the development of pathological states. Due to factors such as these, intrinsically established neural pathways are not sufficient for maturation toward adult-like function.

Sensory input must be of normal amount and type in order for typical development to occur. Otherwise, atypical sensory experience has the potential to be associated with abnormalities in brain development and function. For instance, Sur et al., (1988) experimentally altered the pattern of sensory stimulation allowed to enter the auditory cortex by switching the inputs to the auditory thalamus from auditory to visual. That is, via the intact thalamo-cortical auditory pathways, the auditory cortices of their subjects ultimately received visual input. Following a short period of this manner of stimulation, the auditory cortices of these subjects began to exhibit structural and functional similarities typically specific to visual cortex. Thus, changes in sensory input have the potential to greatly alter various aspects of brain development.

One straightforward example of the developmental and functional impacts of sensory abnormalities in humans can be seen in individuals who present with partial or total sensory loss. People who experience both deafness and blindness exhibit alterations in brain function and organization, which, in turn, have been shown to be related to functional performance (Niemeyer and Starlinger, 1981; Neville & Lawson, 1987; Lepore et al., 1998; Rettenbach et al., 1999; Bavalier et al., 2000; Levänen, 2001; Bavelier & Neville, 2002; Merabet & Pascual-Leone, 2010). For instance, children who are born with hearing loss show delays in auditory cortical maturation, if auditory stimulation is not provided via auditory prosthetic devices (i.e., hearing aids and/or cochlear implants) within a sensitive period (Ponton, 1996; Sharma et al., 1997; Sharma et al., 2002a, 2002b, 2005). Additionally, numerous human and animal studies have shown that adults with profound deafness experience reorganization of the auditory cortex by both the visual and somatosensory systems—termed *cross-modal reorganization* (Buckley & Tobey, 2011; Doucet, Bergeron, Lassonde, Ferron, & Lepore, 2006; Fine, Finney, Boynton, & Dobkins, 2005; Finney, Clementz, Hickok, & Dobkins, 2003; Neville et al., 1983; Rebillard et al., 1977; Sadato, 2005). Similarly, both auditory and somatosensory stimuli (e.g., Braille reading) activate visual cortex in blind adults (Hyvarinen et al., 1981a, 1981b; Neville et al., 1983; Uhl et al., 1991 Kujala et al., 1995; Sadato et al., 1996; Cohen et al., 1997; Hamilton & Pascual-Leone, 1998; Roder et al., 1999). Recent reports expand on this notion by presenting evidence that children who are born with severe cases of hearing loss, but in whom auditory function is restored via auditory prostheses, exhibit cross-modal reorganization of the auditory cortex (Campbell & Sharma, 2016; Sharma & Glick, 2016; Sharma, Campbell, & Cardon, 2015). Moreover, children who are diagnosed with Auditory Neuropathy Spectrum Disorder (ANSD)—a disorder of auditory temporal processing—also present with abnormalities in cortical maturation (Campbell, Cardon, & Sharma, 2011; Cardon & Sharma, 2013; Cardon,

Campbell, & Sharma, 2012; Sharma & Cardon, 2015; Sharma et al., 2011). Also, Central Auditory Processing Disorder (CAPD), which is characterized by deficits in sound localization, discrimination, pattern recognition, temporal processing, and sound processing in background noise, in the absence of a peripheral hearing loss is also associated with abnormal cortical auditory responses (Koravand et al., 2017). Thus, it appears that both the amount and type of sensory input can affect maturation of the sensory cortices.

Sensory impairment, and associated brain abnormalities, have been shown to be highly correlated with patient outcome in a variety of domains, such as social/emotional functioning, speech and language development, cognitive abilities, theory of mind, academic performance, among others (Corina & Singleton, 2009; Peterson et al., 2016). In the aforementioned studies concerning the effects of hearing loss on cerebral cortex organization, the degree of cross-modal reorganization was negatively correlated with participants' ability to accurately perceive speech presented in background noise (Campbell & Sharma, 2016; Sharma & Glick, 2016; Sharma, Campbell, & Cardon, 2015), which is consistent with other related studies (Buckley & Tobey, 2011; Cardon & Sharma, 2018; Doucet et al., 2006). Deaf children also show more theory of mind delays and social/emotional difficulties (i.e., peer popularity) than their hearing peers (e.g., Peterson et al., 2016), as well as a greater propensity to exhibit impulsivity and distractibility (Dye, Hauser, & Bavalier, 2008; Parasnis, Samar, & Berent, 2003; Reivich & Rothrock, 1972). These behavioral issues could be mediated by neural consequences of sensory loss.

Another principle that is central to the issue of abnormal sensory processing and its role in development is the notion of *developmental diaschisis*. This concept holds that atypical structure/function in one part of the brain can contribute to abnormality in distant brain areas (Saré, 2016). Recent reports have provided compelling evidence that this process is indeed at work in the cerebral cortex and other parts of the brain (e.g., Feliciano, Su, Lopez, Platel, & Bordey, 2011; Ishii, Kubo et al., 2015). For instance, Ishii et al., (2015) showed that mice, in which somatosensory deficits had been induced in utero, eventually exhibited abnormalities in the medial prefrontal cortex (mPFC) and its associated behaviors, despite the lack of any apparent direct neural connections between these affected brain areas. When these investigators preferentially stimulated the neurons in the damaged part of the somatosensory cortex, the mPFC mediated behaviors improved. Others have proposed that developmental diaschisis may be at work in DD, such as Autism Spectrum Disorder (ASD). Wang et al., (2014), for example, argued that dysfunction of the cerebellum may disrupt the maturation of distant cortical networks. Using this same reasoning, one might reason that abnormality in areas involved in sensory processing could be associated with abnormal development of other, apparently unrelated, parts of the brain. Given the overlap in sensory symptoms observed across DD and the principle of developmental diaschisis, it might be reasoned that sensory abnormalities could be foundational to many cases of DD. Orefice et al., (2016), in fact, showed that prenatally altering peripheral somatosensory function (esp. GABAergic inhibition) caused somatosensory sensitivities, as well as social deficits. These same differences were not seen in adults in whom the same manipulations were carried out. Consistent with this line of thinking, differences in diagnosis, despite similarities in sensory symptoms, might be explained by genetic and environmental factors and their interaction. This notion is in keeping with the RDoC advocated by the NIMH, which includes sensory

perception (esp. auditory, visual, and olfactory/somatosensory/multimodal) as one of the fundamental components of functioning that might contribute to mental illness.

In addition to specific changes in brain development associated with sensory loss, many consider the presence of sensory abnormalities as a sign of overall brain vulnerability (Levit-Binnun, Davidovich, & Golland, 2013; Pitzianti et al., 2016). While sensory symptoms are often not included in the primary diagnostic criteria of DD, they are recognized as *secondary symptoms* or *neurological soft signs* (NSSs). This category of phenotypes includes deficiencies in motor function, sensory integration and processing, sleep, feeding, and self-regulation that are often more subtle than primary symptoms. It is thought that NSS are related to the loss of robust ability to adapt to perturbations in environmental stimuli that is associated with abnormal brain network architecture (Levit-Binnun, Davidovich, & Golland, 2013). This deficit in adaptability, in turn, leads to vulnerability of the brain during stressful events and greater likelihood for deviation from homeostasis. Decreased cerebral resilience and increased vulnerability could ultimately lead to psychopathology. Indeed, the sensory symptoms that are exhibited in many DDs may be indicators of abnormal brain architecture and vulnerability in these disorders. In fact, in numerous cases of DD, sensory and motor symptoms are recognized prior to official diagnosis via the observation of core symptoms. For example, diagnosis of Autism Spectrum Disorder (ASD), Dyslexia, Attention Deficit Hyperactivity Disorder (ADHD), and Schizophrenia have been shown to be preceded by sensory and motor abnormalities (Baranek, 1999; Bhat, Galloway, & Landa, 2012; Bryson et al., 2007; Cannon, Jones, Huttunen, Tanskanen, & Murray, 1999; Fliers et al., 2009; Kroes et al., 2002; Murray et al., 2006; Ozonoff et al., 2008; Schiffman et al., 2009; Viholainen et al., 2006). Thus, sensory abnormalities seem highly related to overall adaptive function of the brain.

1.2 Sensory Processing Disorders

The idea of a specific set of disorders negatively affecting normal behavioral responses to sensory stimuli was first advocated by Ayres (1972, 2005). This investigator originally conceptualized such dysfunction as a deficiency in sensory integration (i.e., sensory integration disorder; SID). Subsequently, the terms sensory processing disorder (SPD) and sensory modulation disorder (SMD) have been used seemingly interchangeably to describe peoples' difficulties with sensory stimulation (Koziol, Budding, & Chidekel, 2011). However, it could be argued that *processing* and *modulation* refer to specific challenges with sensory input. That is, sensory processing could describe the processing that occurs within distal sensory receptors, as well as higher order, even top-down, brain function related to perception and interpretation of sensory input. Also, sensory modulation seems to be more specifically associated with responsivity to sensory stimulation—either hypo- or hyperresponsiveness (Baranek, David, & Poe, 2006, 2007). People who exhibit hyporesponsivity to sensory input require more intense sensory stimulation to elicit a reaction, while those who present with hyperresponsivity often perceive otherwise innocuous stimuli as extreme or even threatening—the former appears to others as lack of responsiveness and the latter leads to evasive or defensive behaviors. Still others who are classified as having an SPD display sensory seeking behavior, in which they consistently pursue sensory stimulation in order to satisfy a physiologic craving for it, or to re-establish a

sense of homeostasis (Baranek et al., 2006). Because ‘processing’ is a more general term which might include both integration and modulation, it will be used throughout the remainder of this manuscript to describe general sensory dysfunction. More specific terms will be used as needed.

Currently, SPD/SID/SMD are not universally recognized as stand-alone disorders. While many (esp. occupational and physical therapists) commonly use the above terms and work with patients who have sensory difficulties, the fields of neurology, psychiatry, and neuropsychiatry generally do not distinguish these patients as a separate group (Koziol, Budding, & Chidekel, 2011). For instance, the Diagnostic and Statistical Manual, fifth Edition (DSM-5; APA, 2013), and previous editions, do not list SPD as a separate condition. This lack of recognition may be due to the variable nature in which sensory symptoms present and are diagnosed. In other words, sensory symptoms can be manifest in any sensory modality(i.e., auditory, visual, somatosensory, olfactory, gustatory, vestibular, and proprioception), or combination thereof, and can span basic sensory function to high level integration, interpretation, and comprehension.

Sensory abnormalities are diagnosed primarily via parent/caregiver questionnaires, such as the Sensory Profile (Dunn, 1997). While these instruments are generally effective at determining whether a given patient exhibits sensory abnormalities, they have several limitations. For example, they do not provide information about the possible neural underpinnings of SPD (Koziol, Budding, & Chidekel, 2011). Additionally, the symptoms of SPD overlap with many symptoms that are common to other, more generally accepted, disorders. Indeed, recently, the DSM-5 includes sensory abnormalities as a core feature of Autism Spectrum Disorder (ASD). Thus, the definition of SPD may not be sufficiently concrete for all to accept it as its own condition.

The decision of whether SPD is a disorder of its own, or not, is beyond the scope of this review. However, in considering sensory abnormalities across DDs, it is worthwhile to summarize the various sensory symptoms that can be exhibited and the manner in which some have attempted to define them. Due to the lack of consensus regarding SPD/SID/SMD, it is clear that more work is needed to fully understand and describe sensory abnormalities across DD. Specifically, though improving, there is a distinct shortage of reliable and reproducible information regarding the neural correlates of sensory dysfunction. One possible impact of this dearth of information and consensus is that the various treatments used to ameliorate the debilitating effects of sensory abnormalities have had inconsistent success (Case-Smith, Weaver, & Fristad, 2014; Lang et al., 2012; Leong et al., 2015). Presumably, if the neurobiological underpinnings of sensory dysfunction could be identified, one could use these as targets for more effective treatments. To this end, the efforts that have been put forth to understand sensory abnormalities across selected developmental disabilities and their neural underpinnings will be reviewed in the following sections.

2. SENSORY ABNORMALITIES WITHIN SELECTED DEVELOPMENTAL DISORDERS AND THEIR POSSIBLE NEURAL UNDERPINNINGS

The majority of the existing research publications related to sensory processing in developmental disabilities focus on a single diagnosis. Thus, the current section will review the basic patterns of sensory dysfunction and their possible neural underpinnings within DDs that commonly present with sensory abnormalities and for which there is a significant literature which attempts to describe their sensory abnormalities. These DDs include, Autism Spectrum Disorder (ASD), Attention Deficit Hyperactivity Disorder (ADHD), and Schizophrenia.

2.1 Autism Spectrum Disorder

2.1.1 Abnormal Sensory Processing in Autism Spectrum Disorder—Reports of sensory abnormalities have existed since very early characterizations of ASD (Asperger, 1944; Kanner, 1943). However, only recently has “hyper- or hyporeactivity to sensory input or unusual interests in sensory aspects of the environment” been added to the ASD diagnostic checklist (DSM-5; APA, 2013). Studies indicate that between 90% and 96% of patients present with sensory abnormalities (Baranek et al., 2006; Leekam et al., 2007; Marco et al., 2011; Tomchek & Dunn, 2007). Sensory processing abnormalities are increasingly being considered as foundational to ASD, contributing to the development of social, communication, and cognitive deficits that are characteristic of this condition (Baum, Stevenson, & Wallace, 2015; Wang et al., 2004, 2006). In fact, investigations have shown that sensory symptoms were present in young children prior to receiving an ASD diagnosis (Robertson & Baron-Cohen, 2017), and were correlated with social impairment (Wang et al., 2004, 2006). As such, some have argued that behavioral and physiologic correlates of sensory dysfunction could be used as early markers of ASD (Robertson & Baron-Cohen, 2017). Unfortunately, due to a high degree of variability in testing paradigms, stimuli, methods and the heterogeneous nature of the ASD population, there are many inconsistencies and even contradictions within the available body of research concerning the above. However, several patterns are emerging from the literature that provide insight into the sensory symptoms experienced by those with ASD and their neural underpinnings.

Persons with ASD might experience abnormal responsivity to sensory input in a host of ways and in response to stimuli from any sensory modality or combination thereof (Ben-Sasson, Cermak, & Orsmond, 2007; Ashburner et al., 2008; Klintwall et al., 2011; Marco et al., 2011; Baum et al., 2015; McCormick et al., 2015; Uljarević et al., 2017). Sensory reactivity in ASD seems to lie along a spectrum between extreme hyperreactivity and hyporeactivity (Baranek et al., 2006, 2009). Furthermore, sensory seeking behaviors are also common in ASD. On the other hand, while these categories of sensory reactivity are accepted by many, others submit that they are too simplistic (Robertson & Baron-Cohen, 2017). For instance, people with ASD tend to present with a visual search superiority in which they are able to locate a visual target amongst a set of other visual stimuli more quickly than typically developing (TD) peers (Baldassi et al., 2009; Joseph et al., 2009; Kéita et al., 2010; Keehn et al., 2008; O’Riordan et al., 2001; Plaisted, O’Riordan, & Baron-Cohen, 1998a, 1998b). In contrast, these same patients are less sensitive to the number of

visual distractors. Additionally, persons with ASD seem to be more tuned to the pixel-level or first-order (e.g., contrast, luminance) aspects of a visual scene, but less so with elements that involve objects or relationships, to which TD peers are preferentially attuned (Bertone, Mottron, Jelenic, & Faubert, 2005). Thus, those with ASD might exhibit different types of sensory preference or reactivity to separate components of a given sensory landscape. In all, one fundamental principle of sensory processing in ASD seems to be a preferential tuning to local, as opposed to global aspects of the environment (Robertson & Baron-Cohen, 2017).

2.1.2 Abnormalities in Sensory-Specific Cerebral Cortices in ASD—In an attempt to discover the neural origins of sensory abnormalities in ASD, studies have been performed examining sensory systems from periphery to central sites, using both behavioral and physiologic methods. Though varied, the sites of lesion identified in many of these investigations seem to be located centrally, rather than peripherally. For instance, a recent study of the hearing profiles of children with ASD indicated that a higher proportion of children with ASD (55%) presented with clinical auditory impairment, compared to only 6% of typically developing (TD) children (Demopoulos & Lewine, 2016). Further analysis revealed that most of the loci of these impairments originated at the level of the brainstem or higher, not in the cochleae.

Following this reasoning, many of the existing studies concerning sensory dysfunction in ASD have focused on primary sensory cerebral cortices. Atypical findings have been documented in these cortical regions, including studies that have found structural differences in several primary sensory cortical areas (e.g., Gage et al., 2009; Herbert et al., 2005; Rojas et al., 2002, 2005; Sparks et al., 2002). Also, EEG and MEG studies have shown significant differences in response latencies in the auditory, somatosensory, and visual cortices (Bruneau et al., 2003; Edgar et al., 2013; Marco et al., 2011; Miyazaki et al., 2007; Oram Cardy et al., 2008; Roberts et al., 2010; Vandenbroucke et al., 2008; Wilson et al., 2007). Abnormalities were also observed in the primary visual cortices together with typical function in higher-order areas of the brain associated with decision making in ASD (Mikami et al., 1986; Shadlen and Newsome, 1996; Snowden et al., 1991). Areas that tend to be involved in higher-order visual processing, such as the fusiform face area, have also been implicated in ASD (e.g., Lynn et al., 2018). A recent study showed decreased correlations between the structural characteristics (i.e., structural covariation) between a number of sensory-related brain structures in participants with ASD vs. control subjects (Cardon, Hepburn, & Rojas, 2017). Structural covariation findings such as these suggest abnormal connectivity between sensory brain regions and are consistent with previous studies showing non-sensory specific alterations in structural covariation in ASD (Balardin et al., 2015; Bethlehem, Romero-Garcia, Mak, Bullmore, & Baron-Cohen, 2017; Ray et al., 2014; Sharda et al., 2016).

2.1.3 Neural Correlates of Abnormal Multi-Sensory Processing in ASD—Many of the issues observed with sensory processing observed in ASD seem to be related to multi-sensory processing, rather than simple/unisensory function (Baum et al., 2015; Marco et al., 2011). In order to make sense of the complex sensory environments in which we typically find ourselves, we must be able to both integrate sensory stimuli that come from the same or

similar sources and separate those that come from different sources. People with ASD often have trouble with both of these functions. Several studies have shown that individuals with ASD do not benefit in the same manner, as their TD peers, from the addition of visual cues in a speech-perception-in-noise task (Foxe et al., 2015). This deficit may be related to other studies which have shown an altered window of temporal binding in ASD, leading to patients' decreased ability to integrate signals from different sensory modalities that come from the same source or eventdi.e., lip movement and speech sounds (Noel et al., 2017; Stevenson et al., 2016; Wallace & Stevenson, 2014). These behavioral multisensory deficits suggest that neural temporal processing shortcomings underlie sensory dysfunction in ASD. Direct evidence of dysfunctional temporal processing have been reported in the population. For instance, inter-trial coherence (ITC) can be used as a measure of the similarity in EEG (EEG) responses between trials (i.e., between evoked potential "sweeps"). A high degree of ITC is an index of strong cortical neuronal synchrony. Decreased inter-trial coherence in patients with ASD recorded in the auditory, visual, and somatosensory modalities suggests that the temporal pattern of cortical neuron activity over time (i.e., neural synchrony) may be lacking in ASD (Butler, Molholm, Andrade, & Foxe, 2017; Dinstein et al., 2012; Koldewyn et al., 2011; Milne, 2011). In other words, temporal processing seems to be less consistent or noisier in people with ASD (Robertson & Baron-Cohen, 2017).

This pattern of abnormal temporal processing in ASD may be related to the findings from numerous reports which provide evidence for altered balance of excitatory and inhibitory activity in the brains of people with ASD (Rubenstein & Merzenich, 2003). Investigators have theorized and shown evidence of an overabundance of Glutamate, the primary *excitatory* neurotransmitter in the cerebral cortex, and a scarcity of GABA, the principal *inhibitory* neurotransmitter in the brain, in ASD (Rubenstein, 2010). Excessive glutamate, paired with decreased GABA, could lead to hyperexcitability throughout the brains of those with ASD. In addition, temporal processing, local neural synchrony, and temporal coordination of disparate nodes of cortical networks (e.g., as in multisensory processing) are all mediated by GABAergic functioning and have all been shown to be defective in ASD (Klimesch et al., 2007; Thatcher et al., 2009).

2.1.4 Abnormal Supra-Modal Processing and Sensory Function in ASD—

Given the highly varied presentation of abnormal sensory processing across the ASD population, the neural mechanisms that mediate these processes are likely not relegated to sensory-specific cortices. Rather, if there are common neural underpinnings to sensory dysfunction in ASD, they would more logically be located in regions of the brain that had connections to sensory systems and modulated the activity therein, or might be made up of a networks of such sites. Indeed, supra-modal brain regions that have strong interconnections with sensory systems and processes have also been regularly identified as anomalous in ASD. One such brain area is the cerebellum. Converging data suggest that disruptions in the cerebellum contribute to the pathophysiology of ASD (Rogers et al., 2013). In fact, the most oft reported structural abnormalities in ASD are found in this structure (Courchesne & Allen, 1997; Courchesne, 1991; Courchesne, Yeung-Courchesne, Hesselink, & Jernigan, 1988; Courchesne, 1995; Kemper & Bauman, 1998; Kern, 2002; Pierce & Courchesne, 2001; Ritvo et al., 1986) as measured in both postmortem (Bailey et al., 1998; Bauman &

Kemper, 2005; DiCicco-Bloom et al., 2006) and *in vivo* imaging (Philip et al., 2012; Vargas et al., 2005) studies. Structural findings include, loss, degeneration, and decreased size of purkinje cells (Fatemi et al., 2002; Rogers et al., 2013), abnormalities in both grey and white matter, and cerebellar global volume, size differences in the vermis, and diffusion tensor imaging irregularities in the cerebellar peduncles (D'Mello & Stoodley, 2015; Catani et al., 2008; Courchesne et al., 2011; Hashimoto et al., 1995; Kemper & Bauman, 1998; Wang et al., 2014). Behavioral tests administered to people with ASD have also implicated the cerebellum (Gowen & Miall, 2005; Freedman & Foxe, 2017). Finally, functional neuroimaging data have revealed cerebellar dysfunction in ASD, including results that exhibit irregular connections to a number of sensory systems and frontal brain regions (Gowen & Miall, 2005; Hoppenbrouwers et al., 2008; Hanaie et al., 2013).

The cerebellum is highly involved in sensory processing, despite often being characterized as a motor structure. Anatomical and functional studies have shown that the cerebellum forms reciprocal connections with all sensory systems (Kern, 2002; Schmahmann, 1997) and has a high concentration of GABAergic neurons (Hanant et al., 2016). As such, the cerebellum has a modulatory relationship with the cerebral cortex, controlling the 'force' with which sensory stimulation is experienced (Koziol, Budding, & Chidekel, 2011). For instance, evidence from electrophysiological studies has shown that the cerebellum modulates input from auditory, somatosensory, and visual modalities—especially areas of the vermis heavily involved in sensory processing, such as lobules VI and VII (Kern, 2002). Because of its ubiquitous connections with sensory systems, the cerebellum is also a center of multisensory integration (Ronconi et al., 2017). A recent study that measured the structural covariation of 55 sensory-related brain areas, including several from the cerebellum, indicated that participants with ASD had decreased structural covariation between sensory areas in the cerebral cortex and the cerebellum, relative to controls (Cardon et al., 2017). These results suggest atypical connectivity between sensory cortices and the cerebellum.

Based on the above, the cerebellum may be implicated in sensory dysfunction in ASD, though its specific role remains elusive (Hannant, Tavassoli, & Casidy, 2016). However, one prominent theory about how the cerebellum interacts with functions typically tied to the cerebral cortex holds that it is central to generating internal models of aspects of the environment (Stoodley, 2012). These internal models are based on and adjusted by experience, and are ultimately used to make predictions about future events (i.e., Bayesian predictive coding; Baum et al., 2015; Ghajar & Ivry, 2009; Ito, 2008; Miall & King, 2008; Sinha et al., 2014). These predictions can be then used in preparing the necessary systems to respond to incoming sensory information, a function that allows for very flexible and smooth quotidian functioning. In contrast, inaccurate predictions about sensory input could also lead to unpleasant and maladaptive functioning, such as:1) becoming overwhelmed by sensory inputs due to decreased ability to determine/predict what environmental stimuli were of interest, while also filtering unimportant stimuli; 2) confusion about and aversion to sensory stimuli, because of its inherently unpredictable nature; and 3) overly literal perception and understanding of eternal input, resulting from lack of top-down contributions to interpretation (Lawson, Rees, & Friston, 2014; Miall & King, 2008; Pellicano & Burr, 2012). All of these possible outcomes of decreased predictive abilities could negatively

affect social functioning as well as prediction of constantly uncertain sensory events (Sinha et al., 2014). Such phenomena could also contribute to an intolerance of uncertainty (i.e., lack of predictive ability could motivate one to seek the most predictable situations), the result of which could be atypically aversive reactions to unpredictable stimuli (Gomot & Wicker, 2012; Neil et al., 2016). These types of behavioral patterns are observed regularly in people with ASD.

Overstimulation perceived as threatening could result in enhanced fear responses in ASD, which would likely be mediated by other non-sensory specific brain regions, such as the amygdala (Markram & Markram, 2010; Markram et al., 2007, 2008). It has been shown that the degree to which the amygdala is stimulated during a sensory event predicts the extent to which that sensory experience is deemed unpleasant or threatening (Zald, 2003). Additionally, populations of GABAergic neurons make up a signifi-cant proportion of human amygdalae, further implicating this structure in ASD (Hannant et al., 2016). Abnormalities of the amygdala, including larger and overactive amygdalae, have often been reported in ASD and animal models of ASD (Markram et al., 2008; Schumann et al., 2004; Sparks et al., 2002). Green et al., (2015) found human ASD subjects' amygdalae and primary auditory and somatosensory cortices to be overreactive during mildly aversive sensory stimuli, when compared to controls. This and a related study also both showed that the fMRI responses of ASD amygdalae were positively correlated with behavioral measures of sensory over-reactivity in these individuals (Green et al., 2013, 2015). These findings provide clear evidence of a link between function in the sensory cortices and the amygdala and this network's role in aversive reactions to sensory stimuli in ASD.

Numerous reports have also flagged the basal ganglia as contributing to sensory dysfunction in ASD (Koziol, Budding, & Chidekel, 2011; Hannant et al., 2016). As one is bombarded with countless sensory stimuli at any given time, one is faced with the challenge of selecting which of all of these stimuli are relevant and desired. Many investigators argue that the interaction of the cerebral cortex and basal ganglia is essential to resolve this problem. That is, the basal ganglia imposes a primarily inhibitory influence on the brain and, thus, aides as a selection mechanism for attention by releasing inhibition on the thalamus in response to sensory stimulation and allowing the appropriate areas of the cortex to become active (Koziol, Budding, & Chidekel, 2011). In this way, the basal ganglia acts as a sort of gate between lower brain levels and the cortex (Frank, Loughry, & O'Reilly, 2001; O'Reilly and Frank, 2006; Stocco et al., 2010). One way to measure this type of function is via sensory gating testing paradigms, in which EEG is recorded as paired sensory stimuli are presented. In control subjects, the cortical response to the initial stimulus is larger in amplitude than the that of the second stimulus. This suppression is taken as a marker of increased inhibitory activity in response to repetitive stimuli. Sensory gating abnormalities have been tied to both basal ganglia dysfunction (Prat et al., 2016) and excitation/inhibition imbalance (Orekhova et al., 2008; Madsen et al., 2015; Hannant et al., 2016). The available literature on sensory gating in ASD presents evidence that some with ASD exhibit sensory gating deficits, while others do not (Madsen et al., 2015; Oranje et al., 2013; Orekhova et al., 2008).

2.1.5 Higher Order Cognitive Processes and Large-Scale Brain Networks in Abnormal Sensory Processing in ASD—

Higher-order functions may also make

significant contributions to sensory dysfunction in ASD (Baum et al., 2015). Cognitive processes, such as attention have been implicated regularly throughout the literature as contributing to sensory abnormalities, given their role in modulating activity in sensory brain regions (Kastner et al., 1998; Robertson & Baron-Cohen, 2017; Silver et al., 2007). One area of the brain that may be of interest in attentional function in ASD is the fronto-insular cortex (FIC; Uddin & Menon, 2009; Uddin et al., 2013; Uddin, 2015). This area seems to be instrumental as an intermediary between the default mode network (DMN; i.e., task-negative network) and the central executive network (CEN; i.e., task-positive network). As such, the FIC is an important component of the salience network (SN)—interconnected brain regions instrumental in detecting environmental stimuli that are behaviorally relevant (Uddin & Menon, 2009; Uddin, 2015). The FIC is anatomically and functionally specialized to receive sensory information and relay it to the CEN, where decisions can be made about the stimuli. Given this role, if the FIC is dysfunctional, sensory information may never reach the ECN, or could be corrupted before its arrival, leading to alternate brain systems (e.g., limbic) becoming involved in response preparation, or erroneous decisions being made in the CEN due to inaccurate input (Sridharan et al., 2008). In fact, recent studies in both animal models of ASD and humans have presented evidence of abnormalities in the DMN, CEN, and SN in ASD, and connectivity between these networks (Abbott et al., 2016; Di Martino et al., 2009, 2014; Failla et al., 2017; Gogolla et al., 2014; Lynch et al., 2013; Uddin & Menon, 2009; Uddin et al., 2013). Germane to the current review, deficiencies in the functional connectivity between the DMN/CEN and the SN have been shown to be associated with abnormal sensory symptoms in ASD (Abbott et al., 2016).

Some have proposed that people with ASD present with hyper-connectivity within local brain regions, but long-range hypo-connectivity (Anderson et al., 2011; Courchesne & Pierce, 2005a, 2005b; Hull et al., 2017; Maximo et al., 2013; Paakki et al., 2010; Rudie and Dapretto, 2013). Indeed, abnormalities of white matter tracts of the brain have been shown regularly in ASD, especially in the anterior-posterior axis, which is relevant to the DMN and cerebellar-cortical connectivity (Maximo et al., 2013; Monk et al., 2009; Stein & Stanford, 2008). This notion might lead to isolated processing modules that don’t communicate effectively with other parts of the brain and could lead to behavioral symptoms such as preference for local aspects of sensory situations, at the expense of more global characteristics (Robertson & Baron-Cohen, 2017).

2.2 Attention Deficit Hyperactivity Disorder

2.2.1 Abnormal Sensory Processing in Attention Deficit Hyperactivity Disorder—

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common developmental disabilities, with an estimated 5% of all children in the United States receiving an ADHD diagnosis (APA, 2013). ADHD is generally characterized by a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development (DSM-5). In addition to symptoms of inattention, hyperactivity, and impulsivity, people with ADHD often present with sensory dysfunction. However, unlike ASD, sensory abnormalities are not included in the core diagnostic criteria for ADHD. Additionally, it appears that sensory symptoms are often present at birth in those who are eventually diagnosed with ADHD (Ben-Sasson, Soto, Heberle, Carter, & Briggs-

Gowan, 2014). In these ways, children with ADHD, ASD, and sensory processing disorders have some overlap (Yochman et al., 2013).

The most common finding surrounding sensory abnormality in ADHD has been that people with ADHD simply tend to have more sensory symptoms than typical controls (Ben-Sasson et al., 2014; Bijlenga, Tjon-Ka-Jie, Schuijers, & Kooij, 2017; Clince et al., 2015; Hern & Hynd, 1992; Iwanaga et al., 2006; Micoulaud-Franchi et al., 2015; Miller et al., 2012; Pfeiffer et al., 2014). Often these results are obtained via parent/caregiver report, such as the Sensory Profile (Cheung & Siu, 2009; Dunn & Bennett, 2002; Ermer & Dunn, 1998; Mangeot et al., 2001; Shimizu et al., 2014; Yochman et al., 2004, 2013). Persons with ADHD have presented with abnormalities in the vestibular (Bhatara et al., 1978, 1981; Dunn & Bennett, 2002; Grossman & Lithgow, 2012; Mulligan, 1996; Ren et al., 2014), tactile (Cermak, 1988; Parush et al., 1997; Schaughency, 1987), visual (Dunn & Bennett, 2002; Engel-Yeger & Ziv-On, 2011; Nazari et al., 2010; Ren et al., 2014; Schaughency, 1987), olfactory (Engel-Yeger & Ziv-On, 2011; Lorenzen et al., 2016; Romanos et al., 2008), and auditory (Bijlenga et al., 2017; Cheung & Siu, 2009; Engel-Yeger & Ziv-On, 2011; Ghanizadeh, 2009) modalities. In addition, multisensory processing seems to be challenging for many with ADHD (Panagiotidi et al., 2017). Also, people with ADHD have shown common sensory processing patters, such as hypersensitivity, hyposensitivity, and sensory seeking (Ghanizadeh, 2011). Furthermore, sensory over-responsivity (esp. tactile and auditory) has been shown to be associated with anxiety (as in ASD; Ghanizadeh, 2011), attentional deficits (Micoulaud-Franchi et al., 2015; Panagiotidi et al., 2018; Pitzianti et al., 2016), hyperactivity (Lin et al., 2013), and adaptive participation in daily life activities in ADHD. Thus, it is clear that patients with ADHD are very likely to present with some form of sensory atypicality and these challenges are closely linked to the other symptoms of ADHD. However, not unlike the variety of attentional and hyperactivity/impulsivity symptoms displayed across patients with ADHD, this group is heterogeneous in their presentation of sensory symptoms (Pfeiffer et al., 2014).

2.2.2 Sensory and Supra-Modal Brain Region Involvement in Sensory Dysfunction in ADHD—

To date, there are no definitive structural or functional brain characteristics that have been tied to sensory abnormalities in ADHD, though several are implicated. For example, several researchers have presented neurophysiological evidence of abnormalities in cortical visual evoked potentials, which were associated to both early visual sensory processing, as well as later attentional processing, such as filtering, orientation, and response inhibition (Kemner et al., 1996; Nazari et al., 2010; Perchet et al., 2001; Woestenburg et al., 1992; Yong-Liang et al., 2000). Furthermore, enhanced resting-state brain activity was shown in adolescents with ADHD, compared to age-matched controls, in primary or secondary visual, auditory, and somatosensory cortices and thalamus by Tian et al., (2008). Findings such as these suggest that sensory-specific cortices and subcortical sites are likely involved to some degree in abnormal sensory processing in ADHD.

The heterogeneous presentation of sensory abnormalities across ADHD suggests that supramodal brain regions are also involved in these symptoms. For instance, cortical thinning throughout the brain has been proposed as a marker of ADHD (Narr et al., 2009). While association cortices, where a great deal of sensory integration occurs, exhibited

cortical thinning, primary sensory regions were largely of typical thickness, suggesting a neural correlate of multisensory integration impairments in ADHD. In addition, areas that are connected to sensory systems and tend to have a modulatory influence over sensory cortices, such as the cerebellum (Cao, Shu, Cao, Wang, & He, 2014; Cherkasova & Hechtman, 2009; Davis et al., 2009; Goetz et al., 2014; Hong et al., 2014; Uddin et al., 2017), basal ganglia (Di Martino et al., 2013; Hong et al., 2014; Pereira et al., 2016), frontal cortices (Hong et al., 2014; Pereira et al., 2016; Uddin et al., 2017), and amygdala (Cocchi et al., 2012) are regularly shown to function abnormally in ADHD. While these structures have largely not been evaluated in terms of sensory abnormalities, one might reason that they could be involved in abnormal sensory processing in ADHD due to their connections to sensory systems, their role in higher order functions, such as prediction, inhibition, emotional reactivity, temporal processing, multisensory integration, and sensory filtering, and their likely involvement in sensory dysfunction in other disorders, such as ASD. Furthermore, the superior colliculus (SCs), which is important for sensory integration, appears to function atypically in those with ADHD (Panagiotidi et al., 2017). It is possible that problems integrating signals from multiple sensory modalities in the brainstem could lead to further abnormalities in multisensory processing at higher processing levels.

One pattern of sensory processing abnormality that has been investigated in ADHD (and ASD) is sensory overload (Micoulaud-Franchi et al., 2015). That is, many people with ADHD often feel overwhelmed by constant sensory stimulation, resulting from difficulty filtering out unwanted signals and attending to relevant or desired stimuli (Micoulaud-Franchi et al., 2015). This type of challenge could certainly contribute to inattention and hyperactivity. Some have argued that such deficits could be underpinned by sensory gating impairments, possibly mediated by basal ganglia dysfunction in ADHD (Holstein et al., 2013; Micoulaud-Franchi et al., 2015; Sable et al., 2012).

2.2.3 Patterns of Brain Connectivity Related to Abnormal Sensory Processing in ADHD—

In addition to alterations within structures, the connectivity between these brain regions may be abnormal in ADHD. For instance, functional connectivity between the amygdala and frontal cortices, as well as fronto-temporal-occipital, have both been shown to be impaired and associated with inattention and hyperactivity in ADHD (Cocchi et al., 2012). Large-scale intrinsic brain networks, such as the default mode network (DMN), salience network (SN), and central executive network (CEN) appear to function differently in participants with ADHD (Cao et al., 2014; Carmona et al., 2015; Pereira et al., 2016). As mentioned in the above section concerning ASD, dysfunction of any or all of the hubs or their network connections could lead to sensory processing deficits in ADHD.

Another pattern of sensory processing that has been noted in ADHD is a tendency toward local vs. global processing. This propensity may be sub-served by local hyperconnectivity, with decreased long-range connections throughout the brain (Cao et al., 2014; Cocchi et al., 2012). For instance, Ahmadlou, Adeli, and Adeli (2012) reported that participants with ADHD showed high interconnectivity between brain structures, but that the characteristic length of strong connections was very short. The DMN was shown to have decreased anterior-posterior connectivity in those the ADHD (Cao et al., 2014). Several studies have

presented diffusion tensor imaging (DTI; i.e., white matter tract integrity) evidence of abnormal connectivity in ADHD. For example, Cao et al., (2013) showed poor global connectivity with high degrees of local connectivity, which was theorized to be tied to similar functional deficits. Also, Hong et al., (2014) provided DTI evidence of deficient connectivity between frontal, striatal, and cerebellar regions, which would likely be considered long-range connections. Overall, persons with ADHD seem to be highly likely to present with sensory abnormalities, which tend to follow certain sensory processing patterns that seem to be mediated by dysfunction within subcortical and cortical structures, as well as their connections.

2.3 Schizophrenia

2.3.1 Abnormal Sensory Processing in Schizophrenia—Schizophrenia (SCHZ) is behaviorally characterized by cognitive and affective impairments, such as delusions, disorganized speech, hallucinations and disorganized or catatonic behavior, among others (DSM-5). SCHZ is most commonly diagnosed in adolescence or adulthood. However, there is a severe and rare form of SCHZ, which is diagnosed before the age of 13, termed childhood-onset SCHZ (COS; Bartlett, 2014). Sensory abnormalities, beyond hallucinations, are common in patients with Schizophrenia, though they are often ignored in practice (Javitt & Freedman, 2015). For instance, studies using the Sensory Profile (Dunn, 1997) have reported abnormally high scores in sensory sensitivity, sensation avoiding, and low registration in SCHZ (e.g., Brown, Cromwell, Filion, Dunn, & Tollefson, 2002; Halperin, 2018; McGhie & Chapman, 1961; Melle et al., 1996; Muntaner et al., 1993; Parham et al., 2017; Pfeiffer et al., 2014). Other studies have reported behavioral sensory abnormalities, such as hyperacusis (sounds seeming louder than they physically are), decreased auditory memory, misperception and distortion of sounds and visual stimuli, decreased acuity in processing dim, rapidly presented, or moving objects, increased pain thresholds, impaired two-point discrimination, and odor discrimination deficits (see Javitt, 2009 and Javitt & Freedman, 2015 for reviews). Furthermore, studies have shown that persons with SCHZ also present with deficits in multisensory integration. For instance, Ross et al., (2007) presented evidence of impairments in integrating visual and auditory information during a speech perception in noise task, even when the same participants exhibited no irregularities in unisensory speech perception. Thus, sensory abnormalities, though varied, are common findings in people with SCHZ, including children (Brown et al., 2002; David et al., 2011). While most believe that sensory abnormalities do not cause SCHZ, some argue that basic sensory difficulties may lead to some of the higher order cognitive symptoms experienced by those with the disorder (Javitt, 2009; Leitman et al., 2010; Rissling and Light, 2010).

2.3.2 Neuroanatomic and -Physiologic Correlates of Sensory Abnormalities in Schizophrenia—Numerous studies present evidence regarding the neuroanatomic and -physiologic underpinnings of some common sensory anomalies in SCHZ. For example, probably the most common sensory abnormality reported in SCHZ is atypical sensory gating (e.g., Braff, 1993; Brown et al., 2002; Cadenhead, Light, Geyer, & Braff, 2000; Hamilton et al., 2018; Javitt & Freedman, 2015; Javitt, 2009; Jin et al., 1998; McDowd et al., 1993; Smucny et al., 2013; Swerdlow et al., 2006). There are several different methods to measure sensory gating that have been used in SCHZ studies. One of these methods, pre-pulse

inhibition (PPI), occurs as two sounds are played sequentially, the first much quieter tn the second. The second sound is presented at a level that is loud enough to elicit a startle response from the participant, if it were played by itself. However, if the loud sound is preceded by a separate initial auditory stimulus, it will not cause typically developing participants to startle. Similarly, one can measure cortical auditory evoked potentials to paired auditory stimuli played at more comfortable listening levels. Presenting an auditory stimulus will elicit an evoked potential response that contains a positive-going waveform peak that occurs around 50 ms in adults, termed the P50. If one auditory stimulus is preceded by a similar auditory stimulus, the P50 (and N100 waveform component) response to the second stimulus will normally be suppressed (i.e., it will be of lesser amplitude than the P50 response to the first stimulus). PPI and P50 suppression have both been taken as evidence of sensory gating, or filtering of repetitive, irrelevant, or unwanted sensory information. This type of filtering, which seems to be based on inhibitory filtering and habituation, is important, because it allows our brains to be ready to process novel information (Javitt & Freedman, 2015). Deficits in this type of filtering could lead to sensory overload, which is often reported in SCHZ. Several brain regions appear to be involved in the filtering process, such as the reticular formation's rapidly habituating neurons, the thalamus, basal ganglia, hippocampus, dorso-lateral pre-frontal cortex (DLPFC) and superior temporal gyrus (STG; Hamilton et al., 2018). One replicable sensory finding in SCHZ has been deficient pre-pulse inhibition and P50 suppression, suggesting compromises in sensory gating mechanism, possibly due to atypicality in the structure and/or function of the above structures and their connections (Hamilton et al., 2018). Complimentary evidence of structural abnormalities in the thalamus (Gonthier and Lyon, 2004) and STG (Taylor et al., 2005) have been shown in SCHZ. In addition, P50 suppression and PPI impairments have been associated with a variety of cognitive processes—working memory, speed of processing, hypovigilance, attention—and are associated with clinical symptoms of SCHZ, such as frequency and intensity of auditory hallucinations (Arnfred and Chen, 2004; Erwin, Turetsky, Moberg, Gur, & Gur, 1998; Louchart-de la Chapelle et al., 2005; Freedman, Adler, Gerhardt, & Franks, 1987; Hamilton et al., 2018; Javitt & Freedman, 2015; Potter et al., 2006; Ringel et al., 2004; Yee et al., 1998). In contrast, some contradictory evidence has also been published (Adler et al., 1990; Light et al., 2000; Santos et al., 2010; Thoma et al., 2005).

One prominent notion is that sensory gating deficits are a result of inhibitory defects (Javitt, 2009). For example, it is thought that inhibitory gating stems from projections from the reticular formation that activate inhibitory interneurons in the hippocampus and other modulatory brain regions, ultimately leading to the inhibition of the cortical response to repetitive or irrelevant sensory stimuli (Frazier et al., 1998; Freedman, Adler, Waldo, Pachtman, & Franks, 1983; Javitt & Freedman, 2015). Consistent with this notion, studies using time/frequency analysis of EEG recordings from patients with SCHZ also present findings that suggest inhibitory deficits (Hong et al., 2004; Javitt, 2009; Spencer et al., 2003). For example, Hong et al., (2004) found that some patients with SCHZ and their first-degree relatives showed decreased gamma band (40 Hz) synchronization in response to a steady-state auditory stimulus. This is germane to the current discussion, because gamma synchronization has been shown to be subserved by both glutamatergic and GABAergic

function (Uhlhaas & Singer, 2006, 2012; Uhlhaas et al., 2010). Abnormality in neural synchrony suggests possible imbalance of excitatory and inhibitory brain activity in SCHZ (Hong et al., 2004; Javitt & Freedman, 2015; Spencer et al., 2003; Uhlhaas & Singer, 2006, 2012; Uhlhaas et al., 2010).

Gamma synchronization is needed for the binding of activity from different areas of the cerebral cortex and, thus, may be vital to successful multisensory integration. One prominent theory regarding the neural underpinnings of SCHZ is the disconnection hypothesis (Friston & Frith, 1995; McGuire & Frith, 1996). This theory states that SCHZ symptoms arise from disconnection of distant parts of the brain, even in the presence of intact local processing. That is, many believe that inhibitory mechanisms work to sync the activity of disparate parts of the brain in order to allow these distinct regions to work together. Given the above, it is highly possible that gamma dys-synchronization could be central to both abnormal sensory processing and the central causes of SCHZ.

Further evidence from network analysis of fMRI data has shown abnormalities that seem consistent with the disconnection hypothesis of SCHZ (e.g., Calhoun, Eichele, & Pearlson, 2009; Fornito, Zalesky, Pantelis, & Bullmore, 2012). For instance, Wotruba et al., (2014) presented evidence of aberrant connectivity of the central executive (CEN), default mode (DMN), and salience (SN) networks in participants with SCHZ, such that the SN and DMN were hypoconnected. Other studies have also shown abnormalities in connectivity between the cerebral cortex and cerebellum, as well as cerebellar structural anomalies (esp. vermis), in both adults and children with SCHZ (Keller et al., 2003; Konarski et al., 2005; Moberget et al., 2017). Additionally, Shi et al., (2012) found that graph theoretical analysis of the covariation of structural characteristics (e.g., cortical thickness) of 90 cortical and subcortical areas, as well as white matter connectivity, showed overall less correlation between these sites and tracts in neonates at high risk for developing SCHZ. One measure from this study showed that the central hubs of the networks in these infants exhibited overall less connections entering and leaving them than those of typically developing infants (see also Rubinov and Bullmore, 2013; Zugman et al., 2015; Palaniyappan et al., 2018). These converging results from different neuroanatomic and -physiologic methods lend credibility to the disconnection hypothesis of SCHZ and may contribute to our understanding of its sensory abnormalities. However, there are inconsistencies between the above and other studies' results (e.g., Greicius, 2008), which suggests that more work is needed to comprehensively understand neural connectivity in SCHZ and how it relates to behavioral outcome.

Still other abnormalities in neural processing related to sensory processing have been reported in SCHZ using EEG. For instance, the mismatch negativity (MMN) and P300 responses are both indices of the brain's ability to detect an anomalous stimulus embedded within a string of other repetitive stimuli (i.e., oddball paradigm). The MMN is elicited without a conscious response from the listener, while the P300 only occurs when a participant is asked to response (i.e., push a button) when he or she hears a target deviant stimulus interspersed within a string of frequent stimuli. People with SCHZ have regularly presented with significant differences in the amplitudes of their MMN and P300 response (Jahshan et al., 2012; Javitt, 2009; Leitman et al., 2010; Rissling and Light, 2010). Such

findings are present in those at-risk for Schizophrenia, as well as diagnosed patients, and have been associated with behavioral psychosocial performance. Overall, abnormalities in MMN and P300 may suggest deficits in the ability to detect and register stimuli that are salient to most people, and cold be related to behavioral function relevant to social performance (i.e., detection and interpretation of prosody, theory of mind, sarcasm; Javitt & Freedman, 2015). Additionally, the fact that the MMN is pre-conscious, while the P300 requires a participant response, is taken by some as evidence that people with SCHZ have deficits in both early and higher order sensory processing (Javitt, 2009; Leitman et al., 2010; Rissling and Light, 2010).

In addition to the auditory findings presented above, significant difference in visual processing have also been reported in SCHZ. These results follow a pattern that seems to be widely accepted in the field, which is that patients with SCHZ appear to present with deficits in processing in the visual magnocellular pathway, rather than the parvocellular pathway (e.g., Butler et al., 2007; Friedman, Sehatpour, Dias, Perrin, & Javitt, 2012; Martinez et al., 2008, 2012; Schechter et al., 2005). It is commonly held that the magnocellular pathway in the visual system processes information from the peripheral portion of the visual field. As such, it is primary concerned with visual stimuli that are presented in low light situations, that deal with movement, speed, and timing. On the other hand, the parvo-cellular visual pathway seems to be preferentially involved in processing details, shapes, sizes, colors, and contrast. Thus, the magnocellular system provides a sort of “frame” into which information from parvocellular information can be inserted (Javitt & Freedman, 2015). If persons with SCHZ have abnormal magnocellular processing, it is possible that inaccuracies about reality could be related to their lack of ability to create a context into which the details of their visual field (i.e., driven by parvocellular function) can be situated. This type of mismatch between magnocellular and parvocellular visual processing may also lead to feelings that images are fragmented, perception of visual illusions, and/or impaired emotional processing (Butler et al., 2009; Javitt, 2015). This notion presents one possible connection between abnormal sensory processing and the core symptoms of SCHZ.

3. COMMON PATTERNS IN THE NEURAL CORRELATES OF SENSORY DYSFUNCTION ACROSS DEVELOPMENTAL DISABILITIES

The above paragraphs have reviewed the most common findings related to sensory processing and their neural underpinnings for ASD, ADHD, and SCHZ. However, there are many other DDs in which sensory abnormalities are present, though, perhaps, not as abundant (e.g., Dyslexia and other Language Learning Disorders, Tourette Syndrome, Obsessive Compulsive Disorder, Oppositional Defiant Disorder, Intellectual Disability, among many others). Reviewing all of these conditions and their respective sensory processing characteristics is beyond the scope of this manuscript. However, in reviewing the details of sensory processing in ASD, ADHD, and SCHZ, it is apparent that there are many similarities and some differences between the aforementioned disorders. It is plausible that elucidating the similarities and differences in sensory abnormalities between DDs could lead to increased understanding of the neural mechanisms that underlie sensory processing deficits, and the disabilities themselves. Thus, the current section will attempt to catalogue,

compare, and contrast the current evidence regarding sensory abnormalities across the three reviewed developmental disabilities, especially focusing on studies in which two or more of these conditions are represented and statistically compared (see Table 1).

3.1 Single Brain Areas Are Insufficient to Comprehensively Define the Neural Correlates of Sensory Abnormalities in DD

The extant evidence suggests that sensory dysfunction does not stem from one single part of the brain in any DD. In fact, while it is intuitive to expect the primary sensory areas of the brain to be the main culprits in the hunt for the neural correlates of sensory dysfunction, this notion is not supported by the literature. Though these primary sensory areas often show some abnormalities in studies investigating sensory abnormalities in DD, there are other brain regions and networks that are implicated. Because of the variability in the manifestation of sensory abnormalities, even within a given diagnostic category, it is reasonable to argue that other, possibly non-sensory specific (i.e., supramodal), regions of the brain contribute to sensory dysfunction. In order for this type of area to be influence sensory processing, it would have to: i) be connected to one or more sensory systems and ii) affect these systems' function in some way. Throughout the current manuscript, several brain regions that fit these criteria have been mentioned in the context of multiple DDs. For instance, the cerebellum and basal ganglia are commonly found to be abnormal in ASD, ADHD, and SCHZ (Hong et al., 2014; Keller et al., 2003; Konarski et al., 2005; Koziol, Budding, and Chidekel, 2011; Moberget et al., 2017; Rogers et al., 2013), as well as other DDs, such as Dyslexia (Nicolson et al., 2001). Provided that the cerebellum has connections with all sensory systems, exerts a modulatory influence on them, is important for sensory integration, and is instrumental in forming predictions about sensory situations and preparing our systems for adaptive reaction to incoming sensory stimuli, cerebellar dysfunction could plausibly contribute to abnormal sensory processing (Koziol, Budding, and Chidekel, 2011). Though not focused specifically on sensory dysfunction, in their review of structural MRI in ASD and ADHD, Dougherty, Evans, Myers, Moore, and Michael (2015) (see Table 1) reported that both patients with ASD and ADHD consistently presented with decreased cerebellar volumes and abnormal structural connectivity within the cerebellum and between the cerebellum and other brain structures.

The basal ganglia is important for sensory gating and filtering of irrelevant sensory information (Koziol, Budding, and Chidekel, 2011). These functions allow the brain to attend to desired stimuli, while also blocking out a significant amount of input. Another recent study directly assessed and compared network connectivity of resting-state fMRI throughout the brain between children with ASD and ADHD (Di Martino et al., 2013; see Table 1). Consistent with the current discussion, this study found that children with ASD who also exhibited ADHD-like symptoms, and those with ADHD all presented with network abnormalities of the basal ganglia.

Koziol, Budding, and Chidekel (2011) proposed a model in which the cerebellum and basal ganglia are vital to sensory processing and disorders thereof. Within this model, the basal ganglia (primarily inhibitory) serves as a 'selection mechanism' that interacts with sensory cortices (primarily excitatory) to allow/disallow information to be processed at these higher

levels. The model also holds that the cerebellum controls the quality or intensity (i.e., force) with which sensory stimuli are perceived and plays a role in balancing excitatory and inhibitory influences over sensory perception. Thus, disordered basal ganglia function could lead to informational flooding (i.e., sensory overload). Furthermore, cerebellar dysfunction might result in hypo- or hyper-reactivity to sensory stimuli, abnormal temporal processing of sensory input, and/or poor multisensory integration. Together, these types of sensory abnormalities represent many of those observed in ASD, ADHD, and SCHZ. Since both the cerebellum and basal ganglia are often deficient in ASD, ADHD, and SCHZ, cerebellar- and basal ganglia abnormality-related sensory dysfunction could account for some of the overlap in sensory symptoms between these disorders.

Another supramodal brain structure that seems to have abnormal structural and/or functional properties across various DDs is the amygdala (Sparks et al., 2002; Schumann et al., 2004; Markram et al., 2008; Cocchi et al., 2012; Green et al., 2013, 2015). The amygdala is highly involved in emotional reactivity to sensory input. Thus, abnormalities of the amygdala could play a role in the atypical sensory reactivity that is often observed in various DDs, such as ASD and ADHD (Green et al., 2013, 2015). In comparing the resting-state network connectivity between children with ASD and ADHD, Di Martino et al., (2013) (see Table 1) found abnormal temporo-limbic connectivity was a characteristic that distinguished participants with ASD from all others. Dougherty et al., (2015) (see Table 1) reviewed structural MRI findings in people with ASD and ADHD. Comparison of the available findings resulted in the discovery of consistent reports of amygdala overgrowth in ASD, while those with ADHD tend to present with structurally normal amygdalae. On the other hand, other studies performed with just subjects with ADHD, as well as SCHZ, have shown amygdala abnormalities (e.g., Cocchi et al., 2012; Dzafic, Burianov a, Martin, & Mowry, 2018). It is possible that amygdala deficiencies are common across DDs, but that they are more pronounced in ASD. This pattern of results might explain the propensity of children in the ASD population to have atypical reactions to various types of sensory stimuli.

In addition to abnormalities in single brain structures, abnormalities have also regularly been reported in multiple DDs in large-scale brain networks, such as the default mode network (DMN), salience networks (SNs), and central executive network (CEN). These networks are highly related to sensory processing as they form the fundamental mechanisms for cognitive processing of sensory stimuli. That is, parts of the SN act as a sort of switch between the task-negative DMN and task-positive CEN. When sensory stimuli are relevant to a given subject, the SN allows information into/alerts the CEN where it can be processed (Sridharan et al., 2008; Uddin & Menon, 2009; Uddin, 2015). Thus, abnormalities of these large-scale brain networks could result in either hypo- or hyper-responsivity to sensory input, depending on the specific nature of the abnormality. These brain networks were discussed with respect to their abnormalities and the implications for sensory processing in all three of the DDs reviewed above. In addition, one study reviewed the available literature at the time of its publishing related to temporal coordination of activity in these large-scale brain networks in both ASD and SCHZ (Uhlhaas & Singer, 2012) and concluded that the temporal coordination in these networks was often abnormal in these DDs. While not included in this comparison, other studies have shown similar findings in ADHD (Cao et al., 2014; Carmona

et al., 2015; Pereira et al., 2016). Abnormal sensory processing might be among the widespread challenges caused by atypical function of the DMN, SN, and CEN.

3.2 Dysconnectivity

Many findings seem to point to dysconnectivity throughout the brain as being particularly important in understanding DD-related symptoms, including sensory processing abnormalities. Consistent with the above results regarding large-scale brain networks, many argue that people with various DD diagnoses present with alterations in local vs. long-range connectivity (Bethlehem et al., 2017; Cao et al., 2014; Geschwind & Levitt, 2007; Ray et al., 2014; Robertson & Baron-Cohen, 2017; see Table 1). For instance, those with ASD, ADHD, and SCHZ, as well as Tourette Syndrome, seem to show patterns of increased local connectivity, with decreases in long-range connectivity (Kern et al., 2015). In the aforementioned study by Uhlhaas and Singer (2012) (see Table 1), decreases in long-range temporal coordination of the DMN, SN, and CEN have been shown in SCHZ and ASD. Indeed, white matter connectivity abnormalities are also commonly reported across DDs (e.g., Baribeau & Anagnostou, 2013; Dougherty et al., 2015). For example, Dougherty found DTI similarities between those with ASD and ADHD. Additionally, Baribeau and Anagnostou (2013) (see Table 1) reviewed structural MRI in those with childhood onset SCHZ (COS) and ASD, in which they noted alterations in white matter maturation in these conditions. These findings are also consistent with other studies which present evidence of abnormal temporal processing across DDs (Butler et al., 2017; Dinstein et al., 2012; Koldewyn et al., 2011; Milne et al., 2011; Uhlhaas & Singer, 2012). For example, abnormalities in the 40 Hz steady-state response in ASD, ADHD, and SCHZ suggest gamma binding deficiencies in these disorders, which has implications for the temporal coordination of distributed brain regions (i.e., long-range connectivity).

Many argue that deficits in temporal coordination are related to abnormalities in inhibitory activity (Klimesch et al., 2007; Rubenstein & Merzenich, 2003; Rubenstein, 2010; Thatcher et al., 2009). That is, inhibitory neurons (esp. interneurons) play an important role in setting the rhythm of neural oscillations. Thus, if inhibitory neural activity is abnormal, oscillatory activity cannot be normal, which might lead to inability of disparate brain regions to temporally coordinate their activity. Abnormal temporal processing is often taken as evidence of imbalance of excitatory and inhibitory activity throughout the brain (Rubenstein & Merzenich, 2003). This type of imbalance is factored into many prominent theories regarding several DDs, including ASD, SCHZ, and ADHD (e.g., Rubenstein & Merzenich, 2003). Behavioral functions, such as processing sensory stimuli in the midst of challenging situations (e.g., speech perception in noise) and multisensory integration are highly dependent on inhibitory neural function. Deficits in these functions are widespread throughout DDs and suggest inhibitory dysfunction. Additionally, the propensity for people with ASD, ADHD, and SCHZ to show particular deficits in processing global (as opposed to more local) sensory information may be related to issues of deficient long-range connectivity and temporal coordination also seen in these disorders.

3.3 Central and Higher-Order Versus Peripheral Aspects of Sensory Processing

The relationship between sensory symptoms and their neural underpinnings is complex, stemming from an intricate interplay of both subcortical and cortical processes seems to be at work to produce the behavioral sensory symptoms that are so commonly observed in people with DD. In the preceding paragraphs, various subcortical structures (i.e., cerebellum, basal ganglia, amygdala, thalamus), as well as cortical structures (i.e., sensory cortices) and networks (i.e., DMN, SN, CEN) and white matter tracts, and their roles in sensory processing have been discussed. In addition to these notions, there are several top-down neural processes that may contribute to abnormal sensory detection, perception, and interpretation. One such process might be prediction (e.g., Bayesian predictive coding; Baum et al., 2015). Typical brains are set up to use previous experience to make predictions about the consequences of our own and others' actions, and other sensory input. These predictions afford us many advantages, such as fluidity of interaction with our environment, rapid assessment of relevance and threat of stimuli, and experience-dependent development of comprehension and response accuracy (Courchesne & Allen, 1997). On the other hand, inaccuracy of prediction could lead to problems, such as excessively literal understanding of input, due to a lack of top-down influences, dislike of unpredictable situations or rigidity, desire to artificially introduce predictability/routine into one's life, and inability to determine relevance of input (Gomot & Wicker, 2012; Neil et al., 2016). All of these deficits have implications for sensory processing and are commonly observed in a host of DDs, including those reviewed in the current manuscript. On the other hand, upon review, there are some discrepancies in the available data regarding some neural mechanisms related to one type of prediction—P50 suppression and PPI. That is, while P50 suppression and PPI deficits are very common in those with SCHZ, findings are inconsistent in ASD and ADHD, with some studies reporting no difference between the latter and TD populations (Magnée et al., 2009; Oranje et al., 2013; see Table 1). Thus, it appears there are many complex interactions and processes that combine to lead to abnormal sensory processing, many of which are yet to be discovered or confirmed. However, the information that is available at present suggests that there are many similarities in the underlying neural mechanisms responsible for sensory abnormalities across DD.

4. CONCLUSION

Sensory dysfunction is common in DD and can be extremely debilitating, often contributing to deficits in many essential functions. Unfortunately, consistently effective treatments for sensory dysfunction currently remain elusive. This discrepancy between need and solution may be due to a lack of understanding concerning the neural underpinnings of sensory dysfunction. The readily observable similarities in sensory symptoms between DDs lends credibility to the notion that there may exist shared neural mechanisms of sensory dysfunction across DDs. Therefore, the purpose of the current review was to survey the available literature regarding the neural correlates of sensory dysfunction across several DDs, in order to further understand the common biological mechanisms that lead to sensory abnormalities, regardless of diagnosis.

Based on this review of the literature, several conclusions can be made. First, it appears that several patterns concerning the neural correlates of sensory dysfunction that span diagnostic categories exist. The literature suggests that sensory dysfunction stems from abnormal structural and functional characteristics of central brain regions and networks, including both primary sensory, but also supramodal brain areas (i.e., cerebellum, basal ganglia, amygdala). Atypical connectivity throughout sensory and large-scale brain networks (e.g., DMN, SN, CEN), associated with abnormalities in temporal processing and inhibitory function, seems to be fundamental to abnormal sensory processing. These alterations of neural structure and function are likely associated with deficits in higher order brain functions, such as prediction and attention, which in turn can negatively affect sensory processing. It should be noted that, while these patterns are apparent upon survey of the literature, in many cases, replicable findings are the exception, rather than the rule. However, these patterns and their implications for improvement of clinical practice are viable and exciting avenues for future research.

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Table 1

Summary of Studies Comparing Behavioral and/or Neurobiologic Findings in Two or More Developmental Disabilities. Bold Text Denotes Studies Which Aimed to Specifically Investigate Sensory Function/Abnormality

Study	Disabilities Compared	Methods Used	Main Behavioral Findings	Main Neurobiologic Findings
Little et al., (2017)	**ASD-ADHD**	**Parent/guardian questionnaire (Child Sensory Profile 2; CSP-2)**	**Both ASD and ADHD groups presented with more sensory symptoms, compared to TD subjects. ASD and ADHD groups did not differ in sensory patterns. Children with ADHD showed greatest differences in visual processing, while those with ASD had more oral difficulties.**	**N/A**
Sanz-Cervera et al., (2015)	**ASD-ADHD**	**Parent/guardian questionnaire (Sensory Processing Measure; SPM)**	**Subjects with ADHD showed significantly more deficits in proprioception/body awareness, while those with ASD +ADHD received poorer scores in hearing and social interaction.**	**N/A**
Bethlehem et al., (2017)	ASD-ADHD	Graph theoretical analysis of anatomical MRI structural covariation (cortical thickness)	N/A	Both ASD and ADHD groups showed convergence of results, in that they presented with decreased covariation of cortical thickness from sites measured, as well as greater distance between significant network hubs. In contrast, each group overlapped more in their network characteristics with the TD group than with each other.
Dougherty et al., (2015)	ASD-ADHD	Structural MRI and DTI	N/A	Distinct findings between ASD and ADHD: total brain volume–ASD: increased volume, ADHD: decreased volume; amygdala–ASD: increased volume, ADHD: normal volume; internal capsule–ASD: unclear, ADHD: reduced FA in DTI. Similarities between ASD and ADHD included decreased cerebellar volume and FA in DTI and reduced FA in DTI of the longitudinal fasciculus
Ray et al., (2014)	Ray et al., (2014) ASD-ADHD	Graph theoretical analysis of structural and functional MRI	N/A	Participants with ASD and ADHD differed in overall connectivity patterns—ASD exhibited increase connectivity within networks, while those with ADHD presented with decreased connectivity within networks, but a higher number of connections outside these networks
Dehghan, Mirzakhany, AlizadeZarei, and Sartipizade (2014)	**ASD-ADHD**	**Parent/guardian questionnaire (Sensory Profile 2; SP-2)**	**Children with ASD differed in 9/9 sensory subtests, while the ADHD group showed differences in 7/9 subtests. Both groups exhibited increased sensory symptoms than TD children**	**N/A**
Di Martino et al., (2013)	ASD-ADHD	Graph theoretical analysis of resting-state functional connectivity (fMRI)	N/A	Subjects with ASD and ADHD showed abnormal network connectivity in precuneus; ASD - increases in bilateral temporo-limbic areas; ADHD - increased connectivity within right striatum/pallidum. Children with ASD who showed ADHD symptoms, presented with ADHD-specific connectivity patterns in basal ganglia.
Lane, Shelly, & Dumenci (2012)	**ASD-ADHD**	**Parent/guardian questionnaires and electrodermal response**	**Behavioral scores on measures of sensory overresponsivity were correlated with anxiety results**	**Behavioral sensory overresponsivity was not correlated with electrodermal response during sensory challenge**

Study	Disabilities Compared	Methods Used	Main Behavioral Findings	Main Neurobiologic Findings
Cheung and Siu (2009)	ASD-ADHD	Parent/guardian questionnaire (Chinese Sensory Profile; CSP)	Children with ASD and ADHD presented with significantly more sensory symptoms than TD children. Sensory results successfully distinguished clinical groups from TD group, but not from each other. Significant age effects were observed in all groups, such that sensory issues increased for TD and ADHD children, while the opposite was true for ASD	
Brieber et al., (2007)	ASD-ADHD	Parent/guardian questionnaire and structural MRI	Patients with ASD and ADHD did not differ in ADHD symptoms, but only ASD group showed social deficits	Both clinical groups showed decreased grey matter in left medial temporal lobe, with grey matter increases in left inferior parietal cortex. ASD group only exhibited abnormally high grey matter volumes in right supramarginal gyrus
Oranje et al., (2013)	**ASD-MCDD**	**Pre-pulse inhibition (PPI) and P50 suppression**	**N/A**	**No deficits in PPI or P50 suppression were found in any group (ASD, MCDD, TD). Results suggest that MCDD is dissimilar to SCHZ, because PPI and P50 suppression impairments are common in SCHZ**
Demopoulos et al., (2015)	**ASD-SPD**	**Performance-based auditory and somatosensory psychophysical tasks**	**Subjects with ASD and SPD were impaired in tactile processing (right-handed graphesthesia), while only the ASD group showed deficits in auditory processing (dichotic listening, temporal patterning, auditory discrimination). Auditory performance was correlated with communication skills. The SPD group showed higher tactile detection threshold than participants with TD or ASD.**	**N/A**
McCormick et al., (2015)	**ASD-DD**	**Parent/guardian questionnaire (Short Sensory Profile; SSP)**	**ASD group presented with more sensory symptoms, especially in smell, taste, and auditory modalities. TD group sensory symptoms decreased over time, while ASD and DD symptoms did not. Sensory symptoms were not predictive of adaptive functioning. Sensory symptoms are present early in the etiology of ASD and DD**	**N/A**
Baranek et al., (2006)	**ASD-DD**	**Parent/guardian questionnaire (Sensory Experiences Questionnaire; SEQ)**	**Sensory symptoms decreased with age. ASD presented with significantly more symptoms than TD or DD groups and showed a unique pattern of sensory responsivity–hyporesponsiveness. Hyperresponsiveness was consistent across ASD and DD groups, but more common in these groups than the TD group**	**N/A**
O'Donoghue et al., (2017)	SCHZ-BP	DTI (review)	N/A	(Review) Both disorders show frontal connectivity abnormalities. SCHZ shows greater differences in frontotemporal connectivity, while BP presents with limbic connectivity alterations
Karvelis et al., (2018)	**SCHZ-ASD**	**ASD and SCHZ traits questionnaires and visual statistical learning paradigm**	**ASD traits in healthy adults were associated with more literal sensory representations, with weaker influences of prior experience (i.e., priors), suggesting deficits in Bayesian inference (i.e., prediction) in sensory processing for this group. The same was not true for those with SCHZ traits**	**N/A**
Baribeau and Anagnostou (2013)	SCHZ-ASD	Structural and functional MRI (review)	N/A	ASD - brain overgrowth in early childhood with lack of maturation in adolescence; decreased long-range and increased local functional connectivity. COS -

Study	Disabilities Compared	Methods Used	Main Behavioral Findings	Main Neurobiologic Findings
				abnormalities in cerebral volume, cortical thickness, and white matter maturation in childhood and adolescence, leveling off in adulthood; impairments in local connectivity, with increased long-range connectivity. Connectivity findings consistent with reports of cortical thinning
Uhlhaas & Singer (2012)	SCHZ-ASD	Various methods measuring temporal coordination (review)	N/A	Temporal coordination of large-scale brain networks (e.g., DMN, SN, CEN) has been shown to be abnormal in SCHZ and ASD
Magnée et al., (2009)	**SCHZ-ASD**	**Auditory and audiovisual P50 suppression**	**N/A**	**No differences were seen between ASD and TD groups in audio/audiovisual P50 suppression. P50 suppression deficits were observed in SCHZ group.**
Karatekin & Asarnow (1999)	**SCHZ-ADHD**	**Eye-tracking**	**ADHD group showed shorter fixation times, while children with SCHZ looked at fewer relevant portions of pictures than TD group.**	**N/A**

Bolded rows represent studies that specifically investigated sensory processing via either behavioral or neurobiological methods, or both. Other rows present findings that may be relevant to the topic of neural correlates of sensory processing across DD. *Abbreviations*: ADHD-Attention-Deficit/Hyperactivity Disorder; ASD-Autism Spectrum Disorder; ASD+ADHD-Children with ASD who also exhibited ADHD symptoms; BP-Bipolar Disorder; CEN-central executive network; COS-Childhood Onset Schizophrenia; DD-other developmental disabilities; DMN-default mode network; DTI-diffusion tensor imaging; FA-fractional anisotropy; MCDD-multiple complex developmental disorders; MRI-magnetic resonance imaging; SCHZ-Schizophrenia; SN-salience network; SPD-Sensory Processing Disorder; TD-typically developing.