Sensory Over Responsivity

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Sensory over responsivity (SOR) is characterized by extreme or atypical negative reactions to sensory stimuli across one or more sensory domains (Dunn, 1999; Interdisciplinary Council on Developmental and Learning Disorders [ICDL], 2005; Lane, 2002; Parham & Mailloux, 2005; Reynolds & Lane, 2008). Broadly, there are several hypothesized models that explain the neurological and behavioral underpinnings of atypical sensory processing. SOR is one particular manifestation of sensory dysfunction, often co-occurs with psychiatric disorders, (i.e., attention-deficit/hyperactivity disorder, generalized anxiety disorder, oppositional defiant disorder) (Ben-Sasson, Soto, Heberle, Carter, & Briggs-Gowan, 2014; Carter, Ben-Sasson, & Briggs-Gowan, 2011; Conelea, Carter, & Freeman, 2014), and is characterized as a possible symptom of autism spectrum disorder (ASD) within the repetitive and restrictive behavior criteria of the DSM-5 (American Psychiatric Association, 2013). SOR also occurs and is associated with clinical impairment without co-morbid psychiatric or neuro-cognitive disorders (Carter et al., 2011). This chapter reviews the theoretical orientations that have framed SOR as a disorder, current developmental and neurocognitive understandings of sensory processing within and across the senses (i.e., multimodal processing), and discusses potential future directions for research.

Theoretical Orientations of Sensory Processing

Sensory processing issues were first described in 1963 by Anna Jean Ayres, who was trained as an occupational therapist and educational psychologist, to explain, assess and treat the behavioral and learning challenges experienced and expressed by children with various disabilities by relating neurological processing of and behavioral responses to sensory stimuli (Miller, Anzalone, Lane, Cermak, & Osten, 2007). Ayres used the term sensory integration (SI) to describe these phenomena (Ayres, 1985). Underlying the developmental component of SI

theory is the assumption that learning stems from one's ability to detect, process and use sensory information from the environment and from one's movement to plan and organize behavior (Bundy, Lane, & Murray, 2002). More specifically, individuals differ in their way of processing information from the visual, auditory, tactile, vestibular, proprioceptive, gustatory, and olfactory senses (Huebner and Dunn, 2001). Since Ayres' initial work in sensory integration, theoretical models and research evidence for this construct have been further developed and through the advancement of such models the operationalization and clinical characterization of SOR has become better understood.

The following three described models that have evolved from Ayres' initial work provide the foundation for the primary ways in which sensory processing, particularly SOR, is currently conceptualized and measured. First, Winnie Dunn's (1997) conceptual model of sensory processing combines neurological thresholds of responding (low or high) with self-regulation behavioral strategies (passive or active), and includes 4 hypothesized patterns: sensation seeking, sensation avoiding, poor registration, and sensitivity to stimuli (i.e., SOR). The model suggests that individuals with low neurological thresholds need less stimulation than those with high neurological thresholds to notice and respond to input. Indeed, high levels of stimulation may be aversive for individuals with low neurological thresholds. Individuals with active self-regulation strategies attempt to control sensory input through active behaviors, whereas those with passive self-regulation strategies respond to the input as it occurs. Variation of these four patterns are common in the broader population and do not always represent dysfunction.

Second, Baranek, Reinhartsen and Wannamaker (2001) developed a model of sensory processing, which postulates that hyper- and hypo-responsive sensory modulation behavioral patterns may result from shifts in two sensory processing thresholds: sensory aversion and

sensory orienting. According to their model, optimal engagement in play and other activities is determined by the width between the two thresholds. Moreover, Baranek and colleagues (2001) theorize that the width between the orientation and aversion thresholds is determined by both child characteristics (e.g., attention, affect) and external factors (e.g., environmental and contextual features, caregiver interactions). They further suggest that this optimal band of engagement may be narrower in individuals who have sensory dysfunction (e.g., children with SOR or autism), leading to variations and fluctuations in responses.

Third and more recently, Miller and colleagues (2007) proposed a diagnostic classification of sensory dysfunction called Sensory Processing Disorders (SPD), which includes three patterns: Sensory Discrimination Disorder (SDD) - difficulty processing and/or interpreting sensory information, Sensory-based Motor Disorder (SMBD) - a motor challenge with an underlying sensory basis, such as postural disorder and dyspraxia, and Sensory Modulation Disorder (SMD). The SMD disorders are classified into three types: (a) sensory over-responsivity (SOR), which describes exaggerated, rapid onset and/ or prolonged reactions to sensory stimulation (e.g. distress from loud noises), (b) sensory under-responsivity (SUR), which explains unawareness or slow response to sensory input (e.g. not noticing extreme changes in temperature), and (c) sensory seeking (SS), which describes craving of, and interest in sensory experiences that are prolonged or intense (e.g. engaging in repeated, rhythmical movements) (ICDL; Miller et al. 2007). These patterns are not mutually exclusive and may co-occur in individual children across sensory modalities (Baranek et al., 2006; Liss et al., 2006). For the purposes of this paper, while the previously stated terms are used when referencing specific studies, the term sensory dysfunction is used to describe abnormal sensory processing in a general sense.

Mechanisms Underlying Sensory Dysfunction

Although sensory dysfunction may involve hypersensitivity, hyposensitivity or an atypical interaction and/or integration of information between sensory systems, the majority of studies considering the neural basis of sensory dysfunction in developmental disorders have focused on a single sensory modality and on neural mechanisms underlying hypersensitivity, or SOR. In SOR, behavioral responses to sensory stimuli across one or more sensory domains are exaggerated or negative (Dunn, 1999; Interdisciplinary Council on Developmental and Learning Disorders [ICDL], 2005; Lane, 2002; Parham & Mailloux, 2005; Reynolds & Lane, 2008; Zero to Three, 2016). This may arise from the nervous system over-enhancing or failing to weaken neural responsiveness to sensory input.

Atypical Sensory Adaptation

Evidence suggests that neural mechanisms for weakening neural responses to sensory input, sensory adaptation, may be dysfunctional in several disorders where SOR is co-morbid, such as in children with ASD, individuals with social anxiety, and individuals with schizophrenia. Adaptation is a powerful mechanism allowing our nervous system to remain most sensitive and responsive to novel, non-redundant, information via a weakening or dampening of responsiveness to repeated, redundant stimuli. For example, adaptive mechanisms contribute to our ability to distinguish between many exemplars of similar faces (Clifford & Rhodes, 2005). Adapting to non-information stimuli also helps highlight the information in our environment of the highest behavioral relevance, where the allocation of limited attentional and processing resources should be optimized. Thus, a failure of adaptation maintains heightened responsiveness to stimuli, failing to adjust the gain or sensitivity to sensory inputs. A failure of adaptation also limits the reprioritization of sensory processing resources and the redirecting of attention to

allow recent spatial and temporal information and sensory context to shape perception. Finally, a failure of adaptation impairs short term sensory plasticity across sensory domains.

Early on, before sensory processing disorders were included in the DSM-5 diagnostic criterion for ASD, evidence suggested dysfunctional mechanisms of sensory adaptation in children with SOR, children who may have also suffered with ADHD, obsessive compulsive disorder, or depression. Unlike controls, 5-12 year olds with SOR showed a weaker reduction in the amplitude of evoked brain responses, the P50 component of the auditory evoked response potential (ERP), following presentation of auditory stimulus pairs (Davies & Gavin, 2007; Davies et al., 2009). Auditory paired stimuli allow the measurement of strength of adaptation or sensory gating, the weakening of responses to redundant or irrelevant information. Not only did children with SOR show a weakened ability to filter out repeated or irrelevant auditory information compared to controls, but unlike controls, the capacity for sensory gating did not improve with age (Davies & Gavin, 2007; Davies et al., 2009). In adults, greater SOR symptomology was associated with weaker or less efficient sensory gating, as seen by weaker suppression of brain responses (P50 and N100 ERP components) to auditory stimuli (Kisley & Cornwell, 2006).

Dysfunctional mechanisms of sensory adaptation also have been documented in several disorders which also exhibit SOR, most notably in ASD and schizophrenia, both of which are more prevalent in males, but also in anxiety, which is more prevalent in females. For example, male children with ASD show a reduction in adaptation to high level visual information, the social cues contained in facial identity, compared to age- and ability-matched (verbal and non-verbal) typically developing boys (Pellicano et al., 2007). In this study, 11-year-old children were asked to discriminate two facial identities when they had or had not been previously

adapted to an opposite identity face. Exposure to a given face identity biased perception, resulting in an aftereffect biased towards the opposite identity face. This design allowed investigators to determine the degree of adaptation to faces independent of the ability of children to discriminate between faces. While face identification was unimpaired in children with ASD, they could discriminate facial identities as well as controls, they exhibited weaker adaptation to faces. Interestingly, there was a significant correlation between the strength of adaptation and autistic symptomology, with weaker adaptation associated with stronger symptomology.

Webb and colleagues (2010) extended these basic findings of atypical, weakened, adaptation to faces to a younger age group, toddlers (18 – 30 months). To study this younger cohort, they used a standard developmental paradigm of habituation (reviewed in Colomb & Mitchell, 2008) and monitored changes in looking time with repeated stimulus presentation. Toddlers with more severe autism symptomology showed a more severe reduction, or slowing, of habituation rates, or adaptation to faces but not to houses. Slower rates of learning about faces, that is, slower habituation, correlated with poorer social skills and verbal ability.

Given the findings of atypical adaptation in ASD, and in particular the differential findings for social versus non-social stimuli, one must also consider the influence of atypical attention on such findings. Previous studies in adults suggest that attention and adaptation can interact. For example, attending to a low-level visual feature, such as visual motion, can enhance adaptation to visual motion for attended stimuli relative to weaker adaptation under conditions of passive viewing (Rezec et al., 2004). At the neuronal level, evidence suggests that attention to low level visual features, such as orientation, may increase the adaptability of neurons, with more pronounced effects of attention on adaptation as one ascends the visual hierarchy (see Boynton, 2004 for a review). Attention has also been shown to increase the gain of adaptation for

more complex visual stimuli, such as faces (for example, Rhodes et al., 2011). Importantly, Ewing and colleagues (2013) found that the reduced adaptation to faces seen in children with ASD (8 – 16 years) was not driven by an underlying difference in the quantity or quality of attending to facial stimuli.

Dysfunctional mechanisms of adaptation also manifest in adults with ASD where adaptation to low level sensory input such as auditory loudness is reduced (Lawson, et al., 2015) and the magnitude of reduced adaptation, as quantified by a reduced BOLD signal following fMRI-adaptation, can correlate with individual differences in autistic traits (Ewbank, et al., 2014). However, the dysfunctional adaptation seen in adults with ASD may not always be carried over, or maintained, from childhood as compensatory mechanisms may come into play over the course of development. For example, despite reduced adaptation to facial identity in children with ASD, adults with ASD show facial adaptation effects similar to those found in controls (Cook, et al., 2014). Importantly, although behavioral differences in face adaptation were not found in adults, there may be underlying differences in neuronal responses which fail to reach threshold to manifest as overt behavioral differences, or alternatively, adults may be solving the task differently yielding the same behavior but via utilization of different underlying mechanisms.

Finally, dysfunctional adaptation has been documented in other disorders which are frequently comorbid with SOR, such as anxiety disorders. Individuals with social anxiety disorder display a persistent fear and anxiety in social or performance situations (DSM-5; American Psychiatric Association, 2013). It has been suggested that the negative biases and hypervigilance towards social stimuli seen in social anxiety (see Heinricks & Hofmann, 2001, for a review) may be perpetuated by weakened adaptation to emotional information allowing the

maintenance of enhanced sensitivity and over-responsiveness to emotional information. In anxious children threatening emotional facial expressions enhance brain responses, as measured by event related potentials (Hum et al., 2013; Kujawa et al., 2015). Social anxiety status in adults moderates the processing of emotional facial expression such that adults high in social anxiety show enhanced brain responses (greater amplitude steady state visual evoked potentials - ssVEPs) to threatening compared to happy or neutral faces, an enhancement not seen in adults low in social anxiety (Weiser et al., 2011). The emergence of negative perceptual biases and mechanisms of dysfunctional adaptation to emotional information early in pediatric social anxiety disorder warrants further investigation.

Complementary studies in animals investigating the role of stress on sensory processing dysfunction are providing important insights. For example, prenatal stress can yield suboptimal sensory processing for tactile and vestibular stimuli in neonatel monkeys (Schneider et al., 2017) and weakened adaptation to repeated tactile stimuli in adult monkeys (Schneider et al., 2008). Tactile overreactivity and poor vestibular control in neonates were associated with dysfunctional dopamine and serotonin regulation. Greater sensory dysfuntion was linked to the presence of a genetic variant for serotonin, the s allele genotype, rh5-HTTLPR, and to greater upregulation of dopamine, D_2R, binding in the striatum, a possible compensatory mechanism for low levels of synaptic dopamine (Schneider et al., 2017). Interestingly, there was a significant relationship between D_2R binding in the striatum of the adult monkey and sensory functioning in the neonate but only for animals carrying the s allele for the serotonin transporter genotype. These results suggest that mechanisms contributing to sensory processing dysfunction may involve complex interactions between the dopamine and serotonin systems. Future work in animals and humans is

required to identify the factors which contribute to maintaining sensory dysfunction throughout development, from childhood into adulthood.

Likewise, adults with schizophrenia-spectrum disorder, exhibit atypical adaptation to auditory (e.g., Adler et al., 1985) as well as visual and somatosensory (e.g., Andrade et al., 2016) stimuli. It has been suggested that dysfunctional adaptation may be a useful endophenotype for schizophrenia (Gottesman & Erlenmeyer-Kimling, 2001). As highlighted by Foxe and colleagues: "As an endophenotype, adaptation would lie closer to the 'shared genetic risk' contributing to the clinical state while being genetically less complex than higher-order symptoms and easier to objectively measure. " (Andrade et al., 2016, page 11). The developmental origins of such dysfunction requires further exploration.

Atypical Multisensory Integration

The above evidence provides a compelling mechanism, weakened adaptation, which may underlie some aspects of sensory over responsivity occurring in individuals whether or not they also suffer from autism spectrum disorder, social anxiety, or schizophrenia. Weakened adaptation could account for SOR in any sensory domain, visual, auditory, tactile or olfactory. However, adaptation studies to date have mostly focused on a given sensory domain in isolation. Yet, our experience of the world is not one of disparate and unrelated sensory events, which must be effort-fully linked, but rather of integrated percepts. We don't need to piece together the red color, the sweet taste, and the crunch to figure out that we are eating an apple. We take for granted our ability to integrate information seamlessly across our senses, an integration which aids us in everyday activities. For example, at a noisy party we tend to attend to the lips of the person speaking, because the visual information we obtain is correlated with the speech sounds being produced, and this can help us understand the speech better. It is via the proper working of

mechanisms of multisensory integration that we improve our ability to perceive sensory information, such as our ability to comprehend speech in noisy environments.

Furthermore, it is important to note that information in one sensory domain can interact with and influence our experience of another sensory domain. For example, a concurrent sound can enhance visual responsivity, improving visual contrast detection and altering neurophysiological signals in the alpha range (8-12 Hz) over occipital, or visual, cortical areas, and decreasing beta-band (14-20 Hz) coupling between occipital and temporal cortical areas (Gleiss & Kayser, 2014). Thus, enhancing responsivity in one sensory domain, as is the case in SOR, could potentially yield atypical processing in a second sensory domain and atypical interactions and integration across sensory domains.

Interactions across sensory domains are governed by rules optimizing sensory integration to enhance perception. Stein and colleagues (Meredith & Stein, 1986; Stein et al., 1987; Stein & Meredith, 1993) first outlined these rules from neurophysiological evidence governing multisensory integration in the superior colliculus: (1) *spatial coincidence:* inputs from different senses are spatially aligned, (2) *temporal coincidence*: inputs from different senses occur synchronously in time and (3) *weak individual inputs*: inputs from different senses are suboptimal, below or near threshold. This last rule is an example of the principle of inverse effectiveness, which predicts maximal integration across the senses when individual stimuli are only minimally effective (at or below threshold) if presented individually. Most recently, studies have begun to consider how stimulus intensity, or the principle of inverse effectiveness, interacts with other factors influencing interactions across the senses, namely, the spatial (Nidiffer et al., 2016) and/or temporal (Fister et al., 2016) constraints governing multisensory integration.

A growing body of evidence suggests that mechanisms of multisensory integration may be dysfunctional in several disorders where SOR is co-morbid, such as in individuals with ASD and Schizophrenia-spectrum disorders. Thus, in addition to problems in adaptation, dysfunctional interactions across sensory domains may be an important factor to consider as an underlying mechanism contributing to SOR.

In the realm of temporal coincidence, a relatively small window in time, the temporal binding window, indicates the likelihood that two stimuli across sensory modalities will be perceived as synchronous. The temporal binding window reflects individual differences in multisensory integration and can be highly variable across individuals (Stevenson et al., 2012). The narrower the temporal binding window, the fewer paired stimulus events are perceived as synchronous, yielding a stronger ability to correctly distinguish asynchronous from synchronous inputs. Thus, individuals with less reliable temporal processing or a broader temporal binding window, exhibit weaker multisensory integration.

A recent study by Stevenson and colleagues (2015) compared performance on several multisensory temporal processing tasks to relate the efficiency of multisensory temporal processing with deficits in audio-visual speech integration. Children with ASD were compared to typically developing controls (6-18 year olds) across multiple multisensory tasks involving simple and more complex visual and auditory stimuli. In general, as stimulus complexity increased, children showed less precise temporal processing. Children with ASD showed a speech specific deficit in multisensory processing, with the strength of audiovisual speech binding correlating with low-level multisensory temporal processing.

Deficient multisensory integration has also been found in adults diagnosed with Schizophrenia who demonstrate reduced susceptibility to multisensory illusions, indicative of less efficient multisensory integration (Stekelenburg et al., 2013; Vanes et al., 2016).

Importantly, studies find large individual variability in the strength of multisensory integration, especially for the integration of high level, complex, social stimuli such as faces and voices. Thus, a classic paradigm used to study multisensory processing is the McGurk effect, in which there is a discrepancy between visual speech related cues (lip movement) and auditory speech-related cues (sounds) which yield a novel percept which combines information from the two sensory channels (McGurk & McDonald, 1976). Recent work in adult controls who do not evidence psychopathology or neurodevelopmental disorders suggests large individual differences in the strength of the McGurk effect at both a behavioral (Mallick et al., 2015) and neuronal level (Nath & Beauchamp, 2012). Children (6 - 16 years) with ASD also tend to show less efficient multisensory integration for high level stimuli, as seen by a decreased susceptibility to the McGurk effect (Gelder et al., 1991; Smith & Bennetto, 2007). Interestingly, studies tend to find enhanced or unimpaired multisensory processing for simple, or nonsocial, multisensory stimuli compared to impaired processing for more complex, social, multisensory stimuli, such as those used for the McGurk effect (for example, Bertone et al., 2003). However, some studies indicate that children with ASD (7-16 year olds) may be less efficient at multisensory integration even for simple, non-linguistic and non-facial, stimuli at a behavioral and neuronal level (Brandwein et al., 2013). Thus, the complexity of the information, not only stimulus intensity or the temporal and spatial relationships between stimuli, can provide a detriment or benefit to sensory processing, worsening or enhancing sensitivity.

Finally, one must take into account the dynamic nature of the developing system, a consideration often difficult to tease out given the large range in age of children included in most studies. In a comprehensive study, involving a large sample of 84 children with ASD and 142 typical age-matched controls, Foxe and colleagues (2015) used a cross-sectional design spanning 5-12 and 13-15 year olds, to consider how multisensory integration changes in development. Whereas the younger cohort of children with ASD showed severe deficits in the multisensory integration of seen and heard speech, such a deficit was absent in the older cohort of children with ASD who were entering adolescence. It remains to be seen if such an amelioration in behavior is accompanied by an amelioration in neuronal responsiveness to multisensory input. Furthermore, in children with ASD the deficits in speech recognition were more severe as background noise increased, a condition known to improve speech recognition in typical controls (for example, Ross et al., 2007). Thus, there is a sweet spot yielding the most effective and strongest multisensory integration within the temporal constraints of seen and heard speech which depends on the salience of the sensory input, altered in this experimental design by embedding stimuli in noise. The absence of such a sweet spot of enhanced integration in ASD may be one consequence of over responsivity to sensory input altering the dynamic between the spatial and temporal constraints involved in multisensory interactions and the influence of stimulus salience on such constraints. The behavioral and neuronal development of such influences warrant further investigation.

Measurement andAssessment of Sensory Processing

Various measures of sensory processing, including SOR, have been developed from these conceptual models. The most common measure of sensory processing in children is the parent

questionnaire, for example: Sensory Profile (Dunn, 1999); Short Sensory Profile (McIntosh, Miller, Shyu, & Dunn, 1999); and Sensory Experiences Questionnaire (Baranek et al., 2006). A meta-analysis (Ben-Sasson et al., 2009) revealed that most studies of sensory processing in ASD rely on the Sensory Profile or adapted versions of the Sensory Profile, specifically, the Infant-Toddler Sensory Profile (ITSP; Dunn, 2002) for children 7-36 months old. However, alternative parent-report measures of sensory responses have been used (e.g., Baranek et al., 2006; Talay-Ongan & Wood, 2000; see Ben-Sasson et al., 2009 for a review).

Based on Dunn's (1997) model, the Sensory Profile was developed and normed on 1,037 typically developing children ages 3–10 years, and includes three areas (sensory processing, modulation, and behavioral/emotional responses), nine factor scores (sensory seeking, emotionally reactive, low endurance/tone, oral sensitivity, inattention/distractibility, poor registration, sensory sensitivity, sedentary, fine motor/perceptual), and four quadrants (low registration, sensory seeking, sensory sensitivity, sensory avoiding). The four quadrants of the Sensory Profile are based on Dunn's model of sensory processing previously described. It also measures aspects of emotion, attention and motoric responses to sensation in the environment. Children with SOR would be rated as having elevated sensory sensitivity on this measure.

The Sensory Experiences Questionnaire (Baranek, 1999b; Baranek et al., 2006), another parent report questionnaire, measures hypo- and hyper-responsive sensory patterns in the contexts of daily activities of children between six months and six years of age. The measure yields summary scores of these two response patterns in social and nonsocial contexts. Unlike the Sensory Profile, the Sensory Experiences Questionnaire was validated on typically developing children, as well as children with autism and developmental delays (Baranek et al., 2006). Children with SOR would be rated as showing the hyper-responsive patterns.

The Sensory Processing Assessment (Baranek, 1999a) is an observational, play-based measure of sensory response patterns (hypo- and hyper-responsiveness) designed for children with autism and developmental delays between six months and six years of age. It is administered in a lab setting and tests children's behavioral responses to social and nonsocial sensory stimuli across three modalities: auditory, visual, and tactile. Children are rated on the degree to which they approach and/or avoid novel toys, orient to various sensory stimuli in social and nonsocial contexts across three sensory modalities, habituate to a repeated auditory stimulus, and display repetitive and stereotyped behaviors.

The Sensory Over-Responsivity Scales is an observational measure of SOR for individuals from age three years through adulthood that was initially detailed in a pilot study (Schoen et al., 2008) and recently updated and renamed as the Sensory Processing Scale Assessment (Schoen, Miller, & Sullivan, 2014). The Sensory Processing Scale Assessment and Inventory (Schoen, Miller, & Sullivan, 2017) measure phenotypic variation in sensory modulation dysfunction across sensory domains in children aged 4-18 years. Both the observational and questionnaire components of the Sensory Processing Scale measure sensory processing variations across an array of sensory experiences including SOR.

Rates of SOR reported across studies vary depending on factors such as chronological age, developmental level, modality assessed, response pattern subtype, and method of sensory measurement. For example, in a study of sensory symptoms of infant/toddler boys with Fragile X Syndrome at 9, 12, and 18 months of age, Baranek and colleagues (2007) found risk for or deficient levels of sensory processing in over 70% of the sample when using observational measures (Sensory Processing Assessment), whereas parent report (Sensory Experiences Questionnaire) yielded much lower percentages (0% at nine months versus over 40% at 54

months). Ben-Sasson and colleagues (2008; 2009) reported that rates of SOR ranged from 10-17% for typically developing children within an epidemiological study. Of the group with elevated SOR symptoms measured in tactile and auditory domains, 76.4% demonstrated difficulties only in the tactile domain. Similarly, Tomcheck and Dunn (2007) found that 16% of their 3-5-year-old typically developing sample had total sensory scores in the "probable to definite difference" range, while Leekman and colleagues (2007) reported that 33% of their typically developing sample between 3-11 years of age had sensory symptoms. Regardless of the variability in rates of SOR across studies, as noted by several researchers, a smaller percentage of individuals with typical development experience SOR when compared to individuals with ASD (Ben-Sasson et al., 2008; Dahlgren and Gillberg, 1989; Tomchek & Dunn, 2007), and those with no other co-occurring psychiatric or neurodevelopmental disorders are often only affected in one modality (e.g., Ben-Sasson et al., 2009).

Ben-Sasson, Carter, and Briggs-Gowan (2010) conducted the only known longitudinal, epidemiological study documenting the normal developmental course of SOR with 521 typically developing children. They studied sensory sensitivity, which is a comparable construct to SOR, as measured by the Infant Toddler Social Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 2006) at three time points (Year 1: 11-24 months old; Year 2: 23-42 months old; Year 3: 31-51 months old) and SOR using the Sensory Over Responsivity scales (Schoen et al., 2008) during the 4th assessment period, when children were 7-10 years old. Results indicated stability in ITSEA sensitivity (i.e., SOR) scores across the first three years, with initial infant sensitivity patterns and change in sensitivity through toddlerhood/preschool years predicting SOR status in elementary school-age children. Children with elevated over-responsivity in elementary school had higher levels of sensitivity in infancy and a unique early trajectory. Additionally, 30-33% of

children with elevated SOR in elementary school showed evidence of higher sensitivity levels in early childhood. Furthermore, one-third of children with elevated SOR at 1-2 years old, had SOR at school age, and more than 50% of children with elevated SOR at three years old, had SOR at the elementary school time point. These results suggest persistence but also desistance of sensory signs in early childhood. In addition, a large percentage of children in this sample who were identified as having SOR at the elementary school age were not identified early on. This may be evidence of a later onset of SOR, or limitations of the assessment tools. Ultimately, these results suggest that additional longitudinal research is needed to better understand the development of SOR across the lifespan. Moreover, additional work is especially needed on the multiple sensory modalities in which SOR may emerge across time in typically developing children. Indeed, the majority of work on sensory symptoms has been done with individuals who have been diagnosed with other disorders, and particularly among individuals with ASD.

As previously mentioned, sensory symptoms are not unique to ASD. Individuals with other clinical and developmental disorders (DD) such as Fragile X Syndrome, language impairment, attention deficit hyperactivity disorder (ADHD), anxiety disorders, developmental delays, and intellectual disabilities also have elevated rates of sensory symptoms compared to typically developing peers. However, the rates in other psychiatric and neurodevelopmental disability groups are not as high as those observed among individuals with ASD. Indeed, given the high prevalence of unusual sensory processing in ASD, this has become a criterion for the presence of restricted and repetitive behaviors in the DSM-5 (American Psychiatric Association, 2013), precluding diagnosing both ASD and SOR.

The most substantial knowledge of sensory symptoms in individuals with ASD comes from research involving samples of preschoolers through adults. Several studies note the high

prevalence of sensory differences in preschoolers with ASD compared to those with typical development, ranging from 58% to 100% (e.g., Dahlgren & Gillberg, 1989; Provost et al., 2009; Tomchek & Dunn, 2007; Watling, Dietz, & White, 2001). Two studies (Provost et al., 2009; Watling et al., 2001) found that preschoolers with ASD compared to age- and gender-matched preschoolers with typical development differed on eight out of 10 Sensory Profile factors, and 83% of the 3-6-year-olds with ASD had worse scores on at least one Sensory Profile factor. Moreover, Baranek et al. (2006) reported that 69% of children with ASD (23 to 80 months) exhibited significant sensory symptoms, whereas Tomchek and Dunn (2007) found that 95% of 3-5 year-olds with ASD had probable or definite differences on their total Short Sensory Profile score. Dahlgren and Gillberg (1989) reported that 100% of three-year-olds with autism in their sample had auditory processing difficulties, whereas Jasmin and colleagues (2009) found that 58% of 3-4 year olds with ASD had a Sensory Profile total score in the atypical range, and 94% had atypical responses on at least one item or factor. Ultimately, while rates can vary based on the sensory dimensions and scores reported, there is a distinction in rates of sensory dysfunction between children with ASD and those with developmental delay or typical development. Furthermore, there are specific differences in SOR presentation between children of typical development, children with developmental delay and those with ASD. For example, children with ASD demonstrate more extreme hypo-responsiveness than children with other developmental delays (Baranek et al., 2006; Miller Reisman, McIntosh, & Simon, 2001; Rogers & Ozonoff, 2005). Although children with ASD are not generally more hyper-responsive than those with other developmental delays (Baranek et al., 2006; Rogers & Ozonoff, 2005), they are more likely to demonstrate co-occurring hypo-responsiveness and hyper-responsiveness than children with other developmental delays (Baranek et al., 2006). Therefore, their SOR symptoms

may be more likely to come to the attention of professionals due to their co-occurrence with other sensory symptoms.

In studies assessing SOR across older and broader age ranges of individuals with ASD, prevalence tends to be higher than in younger and narrower age ranges of samples. Sensory difficulties were reported in 94% of a sample of 3-11 year olds with ASD (Leekam et al., 2007; Study 1), 92.5% in 200 children and adults with ASD between 32 months and 38 years of age (Leekam et al., 2007; Study 2), and 85% of 99 items on the Sensory Profile for 3-13-year-olds with ASD were endorsed for sensory difficulties compared to a typically developing control group (Kientz & Dunn, 1996). Less is known about the prevalence of sensory symptoms during the first two to three years of life for those who will receive an ASD diagnosis, although Ben-Sasson and Carter (2013) found that including sensory-regulatory markers in addition to social communication and repetitive behaviors enhanced screening for children with ASD at 12 months of age. Baranek et al. (2006) found that 69% of parents of children with ASD between 23 and 80 months reported sensory difficulties on the Sensory Experiences Questionnaire, but this age range spans from toddlerhood through school age. Other studies have reported that about 83% of toddlers with ASD under the age of three had sensory problems, whereas 65% of toddlers and preschoolers with developmental delay had typical Short Sensory Profile scores (Wiggins et al., 2009).

Through a retrospective video analysis of sensory-motor behaviors of toddlers with ASD, Baranek (1999c) found that sensory symptoms differentiate infants who will later be diagnosed with autism from infants with other developmental delays or with typical development. Specifically, Baranek found that infants later diagnosed with autism: (a) required more prompts before responding to a name call and were less likely to orient to visual stimuli introduced into

the environment than children with other developmental delays, possibly showing the early emergence of a hypo-responsive pattern of sensory processing; (b) were more likely to show aversive responses to social touch, perhaps symptomatic of an emerging hyper-responsive pattern; and (c) engaged in more mouthing of objects, perhaps an early symptom of a sensory-seeking pattern. Interestingly, these sensory symptoms were apparent prior to the time that most parents had any concerns about their infants who were later diagnosed with ASD. Watson and colleagues (2011) postulated that Baranek's findings suggest that abnormalities in early developing sensory processing symptoms in infants with ASD may lead to consequences in other developmental domains.

While Baranek's (1999c) study highlights the importance of early detection of SOR in children with ASD, there are several methodological weaknesses that should be considered when interpreting the results. For example, video samples may be a narrow representation of children's behaviors. That is, parents preselect the situations that may favor pleasant situations and special achievements and avoid videotaping children during uneventful, unpredictable, or adverse conditions—a process that may obscure certain symptoms. Moreover, sampling a range of situations in 10 minutes provided a cross-section of age and behavior, but did not allow for the detailed analyses of infrequent or context-specific situations. In addition, some of the behaviors listed as sensory, such as requiring multiple prompts to respond to a name call may reflect specific deficits in social motivation, such that additional non-social under-responsiveness is necessary to confirm the presence of a sensory, rather than social, symptom. Further research is needed to determine the effects of various contexts on the specific behaviors of interest in this study, using more rigorous methodological designs (e.g., longitudinal), carefully matched

comparison groups, and stimuli that can distinguish atypical sensory from atypical social patterns of responding.

Sensory Treatment

Similar to the measurement of sensory processing, interventions designed for SOR also stem from Ayres' work on sensory integration (Ayres, 1972, 1979; Koomar & Bundy, 1991; Parham & Mailloux, 1996). More specifically, the sensory integration theory is based on principles from neuroscience, developmental psychology, occupational therapy, and education: 1) sensorimotor development is foundational for learning; 2) the interaction of the individual with the environment shapes brain development; 3) the nervous system is capable of change (i.e., plasticity); and 4) meaningful sensory–motor activity can change circuitry. Although new findings and knowledge demonstrate that the nervous system is even more complex and integrated than Ayres and others believed at the time, many of the principles that Ayres built the theory of sensory integration upon are still central to the application of current sensory integration interventions. This knowledge has been strengthened by basic research demonstrating that structural and physiological, as well as molecular and cellular changes in neural functions are possible and that meaningful sensory motor activities can change circuitry due to plasticity (Merzenich et al., 1984; Greenough et al., 1987; Kandel & Jessell, 1995; Kempermann & Gage, 1999; McKenzie, et al., 2003).

Sensory Integration therapy aims to focus directly on the neurological processing of sensory information as a foundation for learning higher-level (motor or academic) skills. Through somatosensory and vestibular activities actively controlled or sought out by the child, the nervous system is thought to be able to better modulate, organize, and integrate information from the environment, which in turn provides a foundation for further adaptive responses and

higher-order learning. Other necessary components of the sensory integration therapy model include a child-centered approach, scaffolding, facilitating progressively more sophisticated adaptive motor responses, and engaging the child in affectively meaningful and developmentally appropriate play interactions. Treatment goals may center on improving sensory processing to either (a) develop better sensory modulation as related to attention and behavioral control, or (b) integrate sensory information to form better perceptual schemas and practical abilities as a precursor for academic skills, social interactions, or more independent functioning.

Sensory integration approaches have been applied specifically to children with ASD, particularly via services to remediate behaviors indicative of sensory defensiveness and intolerances (Ayres & Tickle, 1980; Baranek, 1998; Williamson & Anzalone, 1997). Williamson and Anzalone (1997) define three elements to a sensory integrative approach when working with children with ASD: (1) helping parents understand their child's behavior and foster nurturing relationships; (2) helping parents and teachers modify the environment so that it matches the child's sensory needs; and, (3) helping children organize responses to sensory input.

Although sensory integration therapy is prevalent in practice, very few studies of this approach or similar occupational therapy techniques with children with and without ASD have been shown to be efficacious in reducing sensory symptoms. Recent systematic reviews examining the efficacy of sensory integration therapy (clinic-based interventions) and sensory-based (classroom-based) interventions broadly indicate that clinic-based interventions for children with ASD following manualized protocols show more promise than school-based interventions that are often specifically tailored for each child (Lang et al., 2012 and Case-Smith, Weaver, & Fristad, 2015). Studies of sensory-based interventions indicate limited efficacy. Miller, Coll, and Schoen (2007) conducted a randomized controlled trial on children with

sensory modulation disorder (including SOR) and found positive effects for sensory integration therapy on child performance using Goal Attainment Scaling (Miller, Coll, & Schoen, 2007). Although small randomized controlled trials resulted in positive effects for sensory integration therapies, additional rigorous trials using manualized protocols for sensory integration therapy are needed to evaluate effects for children with and without autism spectrum disorder and sensory processing problems.

Furthermore, there is a noticeable lag in the consensus regarding the merits of the previously mentioned sensory interventions due to limited rigorous methodologically sound research. –Researchers of SOR have offered several potential reasons for such a lag. Schaaf and Miller (2005) purport that one reason for this lag is that the science of occupational therapy is relatively new compared to fields such as psychology and medicine with longer traditions of research and trained scientists. Traditionally, training in OT has focused on service delivery of practical interventions and have only recently begun incorporating research science into training. Given the treatment-focused lens in which most providers are trained, it is not surprising that most of the reviewed studies include individualized interventions with very small sample sizes. These research designs may be better suited to meet the individual sensory needs of the participants, however replicability and standardization of treatment is challenging within this research framework. Moreover, most of the studies provide limited follow-up after intervention, thus it is not known whether positive effects are sustained in the long-term (Baranek, 2002). Ultimately, these methodological shortcomings decrease the likelihood of standardization of and confidence in the efficacy of many sensory treatments for children with SOR.

Conclusions

Individuals differ in their way of processing information from the auditory, tactile,

vestibular, proprioceptive, gustatory, and olfactory senses (Huebner and Dunn, 2001), and sensory dysfunction occurs when individuals process and/or modulate sensory input abnormally. There is strong evidence that for some individuals, hypersensitivity to specific sensory inputs, as occurs when individuals suffer from SOR, can disrupt learning and interfere with participation in social relationships and developmentally expected activities. Individuals with a range of other psychiatric and neurodevelopmental disabilities, such as ASD, schizophrenia, obsessive compulsive disorder, and social anxiety disorder have increased risk for SOR, but SOR also occurs as a clinically impairing condition in isolation. Unfortunately, the empirical literature offers very little information about the clinical course of, or optimal interventions for, individuals with SOR. There is a greater evidence base for individuals with ASD whose symptomatology includes sensory hypersensitivities. As recognition of SOR as a clinical entity has advanced, as evidenced by inclusion in the DC: 0-5 system (Zero to Three, 2016), there have also been major advances in studying putative neural substrates of sensory dysfunction, including difficulties in adaptation, sensory gating, and multisensory integration that can aid in understanding SOR. Advances in both questionnaire and observational assessments of SOR, as well as eye-tracking and non-invasive brain imaging technologies offer a plethora of directions for future study of mechanisms that may underlie SOR as well mechanisms, mediators, and moderators of evidence-based interventions. In 2009, Miller and colleagues called for translational research across clinicians studying sensory processing disorders and neuroscientists studying multisensory processing. We believe that pursuing translational research in this area can advance both basic neuroscience and improve the lives of individuals with SOR, who are clinically impaired by their hypersensitivities to sensory phenomena.

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