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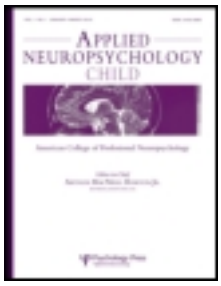

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ADHD and Sensory Processing Disorders: Placing the Diagnostic Issues in Context

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Attention-deficit hyperactivity disorder (ADHD) and sensory processing disorders are behaviorally defined conditions that often co-occur, while both diagnoses have been controversial in part due to the constraints of categorical behavioral diagnosis. However, neuroanatomic studies using neuropsychological tests as ''probes'' have clearly demonstrated that the various symptoms of ADHD are the result of abnormalities in large-scale brain networks. Sensory processing disorders have not yet been grounded within a neuroanatomical substrate. This article reviews sensory processing disorder as a categorically based diagnosis. It discusses certain possible neuroanatomical relationships between the symptoms of ADHD and sensory processing disorders, and suggests that the symptoms of sensory processing disorders be studied within the dimensional framework of research domain criteria.

Key words: ADHD, diagnostic comorbidity, sensory processing disorders

INTRODUCTION

Attention-deficit hyperactivity disorder (ADHD), as behaviorally defined within the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) system, is an early-onset developmental disorder characterized by impulsivity, inattention, and/or hyperactivity (American Psychiatric Association, 2000). The disorder is currently divided into three subtypes: Inattentive, Impulsive/Hyperactive, and Combined. It is well accepted that ADHD is heterogeneous in its presentation (Barkley, 2006). However, there is little, if any, neuroanatomic evidence validating these specific subtypes, and an alternative dual-pathway model supported by anatomical and behavioral evidence has been proposed and subsequently expanded into a possible triple-pathway model (Sonuga-Barke, 2002, 2003; Sonuga-Barke, Bitsakou, & Thompson, 2010; Sonuga-Barke & Fairchild, 2012). Regardless of unresolved subtyping questions, an enormous body of structural and functional neuroimaging research studies, many of them featuring neuropsychological tests as ''probes,'' have consistently demonstrated abnormalities in cortical, basal ganglia, and cerebellar brain regions in ADHD (Ashtari et al., 2005; Bush, 2010; Casey, Nigg, & Durston, 2007; Castellanos & Acosta, 2004; Castellanos & Proal, 2012; Depue et al., 2010; Konrad & Eickhoff, 2010; Pastura, Mattos, Gasparetto, & Araujo, 2011; Rubia, 2007; Vaidya, 2012; Voeller, 2004). These reviews present overwhelming evidence that ADHD symptoms are a manifestation of abnormally functioning brain circuitry. Some very recent studies have additionally more closely considered locus coeruleus-mediated dysfunction and associated alterations in circadian rhythms in modulating ADHD arousal deficits (Imeraj et al., in press).

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The term "sensory integration" was originally proposed by Ayres (2005). This term was introduced to identify a field of study focusing primarily on children who presumably demonstrated atypical behavioral responses to sensory stimulation. This clinical condition is currently referred to as "sensory processing disorder" (SPD). The term "sensory modulation disorder" (SMD) is considered a specific subtype of SPD, in which hyporesponsiveness and/or hyper-responsiveness to sensory stimuli are emphasized (Bar-Shalita, Vatine, Seltzer, & Parush, 2009). These conditions are primarily diagnosed by occupational and sometimes physical therapists. The diagnosis of regulation disorders of sensory processing is included in the Diagnostic Classification of Mental Health and Developmental Disorders in Infancy and Early Childhood (Zero to Three, 2005), and SPD is recognized in the *Diagnostic Manual for Infancy and Early Childhood* (Interdisciplinary Council on Developmental and Learning Disorders, 2005). However, neither SPD nor SMD have ever been included in the DSM and/or International Classification of Diseases (ICD) systems. Even though certain symptoms of SPD/SMD are unique, these symptoms have simply never been variables of diagnostic interest in psychiatry, neuropsychiatry, or neurology.

This does not mean these symptoms are unimportant. Nevertheless, the *Handbook of Pediatric Neuropsychology* makes no mention of these conditions, and the symptoms that comprise SPD/SMD receive only brief mention in a summary discussion of developmental dyspraxia (Hertza & Estes, 2011).

Neuropsychology's primary concern with SPD/SMD appears to have largely focused upon whether or not their associated symptoms really comprise independent "disorders." Significant overlap with ADHD symptoms has been noted, with estimates of comorbidity occurring 40% to 84% of the time, as reviewed by Hassan and Azzam (2012). This concern and confusion is understandable, as neither ADHD nor SPD is a homogeneous condition, and both diagnoses are commonly made according to purely observational and behavioral criteria. Additionally, the question of whether SPD/SMD ever occurs alone has not yet been resolved. Although Reynolds and Lane (2008) reported three cases in which the subjects were presumed to have presented with SPD without meeting criteria for any DSM diagnosis, it is not clear whether or not these subjects presented with other behavioral/cognitive symptoms, because, by definition, children diagnosed with SPD must experience some deficit in adjustment in daily life. Miller, Nielsen, and Schoen (2012) recently reported the results of a study that included 70 participants diagnosed with SMD alone, but all of these children were diagnosed by occupational therapists, apparently without input from other mental/behavioral health professionals. This same study comments on the notable difficulties involved in differentiating ADHD from SMD because the symptoms of inattention, impulsivity, and hyperactivity are often shared, while many/most non-SMD subjects in the study were diagnosed by other mental health professionals. The purpose of this article is to help clarify these diagnostic controversies while placing the symptoms of both ADHD and SPD within a functional neuroanatomic context.

SPD/SMD: MAKING THE DIAGNOSIS

The meaning of the term "sensory" in SPD/SMD needs to be clarified first. The Sensory Profile Questionnaire, which is perhaps the most commonly used observational rating scale to make this diagnosis, does not operationally define "sensory processing," nor does it provide a unifying underlying neuroanatomic construct to explain it (Dunn, 1997). At the same time, the instrument encompasses behaviors that go well beyond the scope of "sensory processing," and it refers to categories or behaviors and behavioral observations that are multifactorial or multiply determined. For example, some sections of the Sensory Profile refer to auditory, visual, tactile, oral, olfactory, and multisensory processing within these modalities, while others refer to sensory modulation (SMD) and relate it to "endurance and tone," body position and movement, and affect and emotional responsiveness. There is a section in which emotional and social responses are rated, and another that assesses presumed, predicted behavioral outcomes of sensory processing (Bar-Shalita et al, 2009). Many of the symptoms listed overlap with behaviors that are included in diagnostic categories of the DSM. In fact, according to the *Sensory Profile: User's Manual*, out of a total of 125 items, 43 items, or nearly 35% from the Sensory Profile item pool, represent "behaviors that were more common for children with ADHD" (Dunn, 1999, p. 44).

This complex combination of factors suggests that the Sensory Profile cannot be measuring a monolithic construct or "one thing." In fact, James and colleagues recently recognized the heterogeneity of these symptom presentations and identified two subtypes of SMD characterized by externalizing and internalizing behavioral presentations, respectively (James, Miller, Schaaf, Nielsen, & Schoen, 2011). The Interdisciplinary Council of Developmental and Learning Disorders (2005) has categorized SMD into three subtypes: sensory over-responsiveness (SOR), sensory under-responsivity (SUR), and sensory seeking/craving (SS/C). However, James and colleagues were unable to support or confirm the existence of these three particular behaviorally defined subtypes. Some of the symptoms

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listed in the Sensory Profile are vaguely defined and are observed in most children at least some of the time, which can lead to overdiagnosis of the condition. In an effort to limit false-positive diagnoses, the Sensory Profile requires identification of a group of behaviors—rather than the simple presence or absence of a single symptom—that interfere with an individual's ability to effectively participate in childhood activities to make the diagnosis.

In this regard, the Sensory Profile defines aspects of SPDs within four clusters or constructs. These constructs include "poor registration," "sensation seeking," "sensitivity to stimuli," and "sensation avoiding."

However, identifying clusters or groups of symptoms is not the same as identifying the neuroanatomic underpinnings that drive them, nor does identifying clusters or symptom groups clarify brain–behavior relationships. In this way, the Sensory Profile is akin to the DSM and ICD systems in that it represents a behaviorally defined nomenclature and not one that is neuroanatomically organized. Just as most conditions listed in the DSM are characterized by abnormalities in multiple brain regions (Koziol & Budding, 2009), it is likely that the varying presentations of SPD/SMD are characterized by anomalous functioning in multiple brain regions and mechanisms as well. There can be no "smoking gun" or single brain region that can generate this overarching range of symptoms. Our purpose is not to criticize the Sensory Profile or any other behaviorally defined diagnostic system. Our purpose is to clarify the neuroanatomic frameworks underlying symptom presentations to facilitate communication among disciplines, as well as to illustrate the inherent limitations of categorically based, observationally defined diagnostic systems. These systems invite diagnostic overlap, so that children can meet several diagnostic categories simultaneously, without providing a framework to explain such overlap. Even when considering the DSM only, it has been reported that children presenting for clinical treatment can meet full DSM-Fourth Edition (DSM-IV) criteria for one to five diagnoses simultaneously (Yaryura-Tobias, Rabinowitz, & Neziroglu, 2003). Therefore, it makes perfect sense that "shared" symptoms among and between different diagnostic categories will generate diagnostic confusion if a clinician is looking to define a "pure" form of a disorder. The fact of the matter is that there is no existing diagnostic nomenclature that meets current needs for diagnosing the problems of developing children.

FREQUENCY OF SYMPTOMS AND COMORBIDITY

Approximately 16% of the general population is estimated to have symptoms of SPD (Hassan & Azzam, 2012). It is common to find SPD/SMD "diagnosed" in children with other well-recognized DSM-based disorders. The tactile and other sensory perceptual hypersensitivities and hyposensitivities that are frequent features of SPD/SMD, for example, are observed in children with autism spectrum disorders (ASDs) and are often associated with increased stereotyped behaviors in that population (Wiggins, Robins, Bakeman, & Adamson, 2009). They are also observed in ADHD populations, and within this group, are associated with increased levels of hyperactivity (Reynolds & Lane, 2009). Sensory–perceptual hypersensitivities have been reported with comparable frequency in children with autism and with global developmental delays (Baranek, Boyd, Poe, David, & Watson, 2007; Boyd et al., 2010). They have additionally been reported with increased frequency in children who demonstrate sleep problems, behavioral problems, and other neurodevelopmental conditions, such as developmental coordination disorder—which itself is very frequently comorbid with cognitive and emotional regulation problems, including the so-called cerebellar cognitive affective syndrome (Cascio, 2010; Green, Baird, & Sugden, 2006; Levisohn, Cronin-Golomb, & Schmahmann, 2000; Marien, Wackenier, De Surgeloose, De Deyn, & Verhoeven, 2010; Tavano & Borgatti, 2007; Zwicker, Missiuna, & Boyd, 2009). Symptoms of SMD also occur frequently with cerebral palsy (Cascio).

When young, school-aged children present for clinical evaluation and are assigned a formal DSM-based diagnosis, a previous diagnosis of SPD/SMD is frequently observed in their histories. In addition, as reviewed by Barkley (2006) in his discussion of problems associated with ADHD, 47% of children with ADHD meet DSM-IV criteria for developmental coordination disorder. In a recent systematic review by Ghanizadeh (2011), at least two thirds of the clinical samples of children with ADHD were found to demonstrate at least one comorbid disorder. Additionally, children with ADHD and SMD (oversensitivity) usually experienced other problems such as anxiety—a relationship that has also been reported by Reynolds and Lane (2009).

These findings have at least four implications. First, certain neurobiologic mechanisms underlying SPD/SMD and other diagnosable neurodevelopmental disorders are shared. Second, as is true of the DSM system, the behaviorally and observationally defined Sensory Profile lacks a coherent neuroanatomic explanation. Third, as children presenting for SMD evaluation are often significantly younger compared with children diagnosed with ADHD and typically developing children (Miller et al., 2012), "sensory processing" anomalies might represent subtle indication of deficits in broadly defined "executive control" when such control is considered a self-regulatory function. SPD/SMD symptoms

might be evident earlier than more sophisticated executive control can be identified in a developing brain, which might explain why many of these children are later diagnosed with more conventionally diagnosable disorders such as ADHD. Finally, it is also theoretically possible that symptoms of SPD/SMD can be transient within the general population, depending upon the maturity level of various brain regions. Imaging studies using network analysis to study brain development have demonstrated that at each stage of normal neurodevelopment, age-specific skill sets correlate with age-specific, large-scale distributed brain networks that develop in a predictable way (Chu-Shore, Kramer, Bianchi, Caviness, & Cash, 2011; Gordon et al., 2011). It may be that even slightly delayed maturation within a specific network region could contribute to a presentation of early-onset symptoms that later spontaneously remit, perhaps simply reflecting a wider range of variation in neurodevelopmental trajectories.

SPD/SMD AND NEUROANATOMIC ORGANIZATION

Perhaps the most formidable roadblock to integrating SPD/SMD and the field of "sensory integration" with other diagnoses and developmental approaches lies in its lack of a coherent neuroanatomical underpinning. In her classic work, *Sensory Integration and the Child*, Jean Ayres concludes that the symptoms reflected in sensory integration disorder "are the end products of inefficient and irregular sensory processing in the brain" (2005, p. 54). She does not, however, address the significance of specific brain–behavior relationships, including the possible interactive roles of the neocortex, the basal ganglia, and cerebellum (Ayres). Koziol, Budding, and Chidekel (2011b), in a literal, concrete review of the term "sensory integration," discussed the cortico-cortical regions, integrative networks of the basal ganglia, and infrastructure of the cerebellum where "sensory integration" operations take place. However, Ayres did not give specific consideration to these brain regions and their interactions. Instead, "sensory integration" theory is based on a pyramid of sensory, cognitive, and behavioral systems that place tactile, vestibular, and proprioceptive systems at the base, above which are the distal senses of vision and audition, while the complex sensorimotor, cognitive, and behavioral systems are found at the highest levels (Cascio, 2010).

Although this model might make intuitive sense, the brain–behavior relationships inherent in this view of the brain's organization have not been established (Ayres, 2005). Accordingly, although the symptoms that comprise the criteria for SPD/SMD are real and common, the conditions lack clear operational definitions and they are poorly understood from an anatomic point of view. In another presentation, Koziol, Budding, and Chidekel (2011a) outlined how a child's ongoing sensorimotor interaction with the environment generates both procedural and declarative knowledge, emphasizing how the cortex, basal ganglia, and cerebellum interact in the development of cognition and executive function. Although this proposal does not validate the "pyramid model," it at least provides a neuroanatomic context within which to place it, while grounding all cognition within a sensorimotor framework.

Sensory hypersensitivities have been identified through behavioral observation and in some psychophysiological studies, but the results of studies that have focused on evaluating sensory thresholds have been inconsistent (Davies & Gavin, 2007; Reynolds & Lane, 2008). Some studies have focused on electrodermal reactivity (EDR/EDA) in an effort to make inferences about levels of activity within the sympathetic nervous system (Mangeot et al., 2001; Schaaf et al., 2010; Schoen, Miller, Brett-Green, & Nielsen, 2009; Schoen, Miller, Brett-Green, Reynolds, & Lane, 2008); however, this work does not speak to the myriad brain regions that can contribute to activity within the peripheral nervous system. In a recent study by Miller and colleagues (2012), it was concluded that EDA responsiveness differences could differentiate between those with SMD, ADHD, or dual diagnosis (SMD with ADHD), and normal controls. While they concluded that ADHD and SMD comprise separate dimensions and may represent different diagnostic categories, the investigators did not hypothesize about what underlying brain mechanisms would govern or generate these differences, despite the fact that the ADHD and SMD groups exhibited similar symptoms of inattention, impulsivity, and hyperactivity.

Investigations that focus on the role of the reticular activating system in regulating the peripheral nervous system appear to be in their infancy. Studies of ASD have attempted to explore the neural underpinnings of abnormal sensory processing within the auditory, tactile, and visual modalities with techniques such as electroencephalography, magnetoencephalography, and functional magnetic resonance imaging. However, the results of these studies of unimodal sensory processing and multisensory integration in ASD have been highly inconsistent and contradictory (Marco, Hinkley, Hill, & Nagarajan, 2011). Pasini and D'Agati (2009) have postulated that there is an alteration in neural networks and a possible dopaminergic role in sensory problems that are not linked to specific brain pathology in children with ADHD. Although a frequent finding in children and adolescents with ADHD is widespread

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(posterior) cortical thickness reductions, this has been associated with significantly more resting-state activity in sensory and sensory-related cortices in those with ADHD relative to normal control children (Klein, 2011; Narr et al., 2009; Tian et al., 2008).

Due to the lack of a coherent theory by which to understand SPDs, Koziol et al. (2011b) proposed the first theoretical, putative functional neuroanatomic conceptualization of SPD/SMD based on known neuroscientific principles believed to govern brain–behavior relationships. Generally accepted functions of the neocortex, basal ganglia, and cerebellum were described to illustrate how interactions between these brain regions generate both adaptive and pathological symptoms and behaviors. The symptoms of SPD/SMD were examined within this model and in relation to their impact upon the development of inhibitory control, working memory, academic skill development, and behavioral automation, all of which are problematic in children with SPD/SMD and ADHD. Similarly, the various abnormalities described in the different sensory domains of motor, emotional, and social behaviors were described within this model, as were possible etiologies.

To summarize this conceptualization, the neocortex operates according to principles of excitation and works as an elegant sensory processor and motor programmer constantly confronted with perceptions and decisions about behaviors. This ongoing confrontation generates an overwhelming ''selection problem'' in relation to choices for attention and action. While the basal ganglia serve multiple roles, their segregated cortical–striatal–pallidal–thalamic–cortical connectional profiles serve as a ''gating'' or ''selection mechanism,'' a massive inhibitory network working in concert with cortex and lower-level orienting systems to select appropriate perceptions and to select appropriate motor programs for action. Within this context, the cerebellum serves as a sensory and motor modulator, regulating the quality of experience and action, including the ''force'' of sensation and emotion, through segregated cerebro-cerebellar circuitry profiles. Symptoms of SMD were explained as influenced by levels of cerebellar activity. The symptoms of SOR, SUR, and SS/C were explained within the context of cortical-basal ganglia and cerebro-cerebellar modulatory control networks. (The terminology of SOR, SUR, and SS/C was abstracted from Miller et al. [2012] and essentially comprises the nomenclature of the two diagnostic manuals for young children listed earlier [Interdisciplinary Council on Developmental and Learning Disorders, 2005; Zero to Three, 2005]. This approach can just as readily be applied to the four clusters of the Sensory Profile; for a comprehensive review, see Koziol et al., 2011b.)

DISCUSSION: ADHD, SPD/SMD: PLACING THE DIAGNOSTIC ISSUES IN CONTEXT

ADHD and SPD/SMD both represent behaviorally defined ''disorders.'' Both are diagnosed according to behavioral observation. Additionally, both are heterogeneous in presentation and in outcome; the ''developmental course'' or trajectory of their presentations remains relatively unknown. This is further complicated in both disorders by using the same behavioral rating scales to evaluate children at different ages. These ''disorders'' are also subject to all the limitations inherent in diagnoses made according to DSM-rating scale-oriented criteria, which have been described in detail by Valo and Tannock (2010). In addition to the numerous instrument- and bias-related problems presented by rating scale-based diagnosis, the overlap of items between rating scales serves as a potent complicating factor. For example, nearly 35% of Sensory Profile items pertain to behaviors that are more like ADHD populations than children with SPD/SMD and/or ''normal control'' subjects. This type of diagnostic formulation invites ambiguity and comorbidity, making it impossible to identify the characteristics that might be unique to any given diagnosis. Even advocates of SPD/SMD diagnosis readily admit that ''the overlap of symptoms in children with SMD and ADHD makes it difficult to differentiate the two disorders'' (Miller et al., 2012).

Similarly, because ADHD and SPD/SMD are behaviorally defined, none of the observational rating scales used to make these diagnoses have any documented relationship to the neuroanatomic networks that underlie or ''drive'' the disorders in question. Nobody knows how the 18 criteria for ADHD ''map'' onto specific brain networks; similarly, nobody knows how the 125 behavior checklist items of the Sensory Profile ''map'' onto brain networks. As nearly 35% of the items on the Sensory Profile overlap with criteria for ADHD, how is it possible to conclude that different neurobiologic mechanisms are involved in these two presumably separate disorders? For example, it is well known that the ''disinhibition'' of prepotent responses (a certain specific type of impulsivity) activates a complex cortical–subcortical circuitry profile (see Koziol & Budding, 2009; Koziol & Stevens, 2012, for reviews). The right-hemisphere inferior frontal-basal ganglia–thalamic–cortical ''loop'' of interaction has consistently been implicated in ''response inhibition'' (Stevens, Kiehl, Pearlson, & Calhoun, 2007, 2009). What direct, objective neuroanatomic evidence supports the idea that the ''disinhibition'' of the ''SS/C'' (sensory-seeking) type should be mediated or governed by any other functional connectivity profile? From a neuropsychological perspective, it is impossible to propose differences in function and, in turn, evidence-based treatment

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programs in the absence of a neuroscientific knowledge base about what is generating the symptom picture.

Proponents of SPD/SMD at times emphasize highly inferential "compensatory" actions as a means of explaining behavioral observations that can be interpreted in multiple ways, without apparent concern for the underlying neurobiologic mechanisms that might possibly drive the symptoms. The attribution of a behavior as "sensory seeking" depends upon perspective and relies upon inferred compensatory mechanisms that have not been objectively demonstrated; the same "sensory-seeking" behavior observed by a neuropsychologist would more likely be interpreted as stimulus-bound behavior in which right-hemisphere frontal–striatal systems are known to play a primary role. So, what is the difference between "sensory-seeking" and "stimulus-bound" behavior? There is no difference. This type of utilization behavior is stimulus-bound, understood to be governed by a known frontal–striatal neuroanatomy until otherwise proven false. This cannot be proven to be false on the basis of peripheral EDA responses. In this regard, neuropsychology and neuropsychiatry seem to have the "edge" in explaining this behavior as a manifestation of a disturbance in frontal-basal ganglia mechanisms and functions, particularly because neuropsychological testing "probes" have been successful in identifying the neurodynamic mechanisms involved, while neuropsychiatry has demonstrated efficacy in treatment with psychostimulant medications (Cools et al., 2009; Frank, Santamaria, O'Reilly, & Willcutt, 2007; Stray, Ellertsen, & Stray, 2010). This functional neuroanatomic interpretation directly addresses the behavioral observations and inferences made in the *Sensory Profile: User's Manual* with respect to aspects of the "sensation seeking" and "sensitivity to stimuli" subtypes (Dunn, 1999, pp. 35–36).

Similar arguments can be made with respect to the factors of "poor registration" and "sensation avoiding." Children with poor registration are described as having low energy levels, appearing disinterested and apathetic, bring overly tired, and having flat or dull affect. It can be readily hypothesized that these behavioral descriptions comprise manifestations of dysfunction within reward circuitry profiles governed by the mesolimbic division of the dopaminergic reward system originating in the ventral tegmental area of the basal forebrain and including the anterior cingulate/medial circuitry network (Doll & Frank, 2009; Sonuga-Barke & Fairchild, 2012). A competing hypothesis could include a presentation characterized by mild central hypotonia, in which certain regions of the cerebellum and/or frontal motor cortices would be implicated. However, these types of brain–behavior relationships have not been systematically applied to the investigation of the so-called poor registration subtype, even though from a neuroanatomic perspective, these would be the first places to "look." "Sensation-avoiding" children are often described as presenting with ritualistic tendencies, having difficulties in making transitions from one task or activity to another, and as frequently experiencing emotional outbursts. They are sometimes termed stubborn and controlling. While these types of behaviors are interpreted in the *Sensory Profile Manual* as an attempt to limit sensory input, from a clinical perspective, these descriptions are also consistent with behavior related to various forms of anxiety disorder and with oppositional defiant disorder (ODD) diagnoses defined in DSM terminology. In this regard, it is also of interest that ODD has been interpreted as an obsessive-compulsive spectrum disorder (Koziol, 1994). In any event, frontal–striatal circuitry profiles, connections with the paleocortical division of the limbic system, and paralimbic–cerebellar connectional profiles have been implicated in mediating these types of behaviors (Mega, Cummings, Salloway, & Malloy, 1997; Schmahmann, Weilburg, & Sherman, 2007). However, once again, how these brain networks might relate to the "sensation-avoiding" subtype of SPD has not been investigated.

To address these issues, perhaps the field of "sensory integration"/"sensory processing" should consider a move away from the overarching, categorical model of diagnosis that has also hampered DSM nomenclature. Instead, the approach could shift toward a more dimensional perspective currently being encouraged in relation to DSM categorical diagnosis. The National Institutes of Health have emphasized a dimensional approach to understanding most mental disorders (Coghill & Sonuga-Barke, 2012; Cuthbert & Insel, 2010; Insel et al., 2010; Sanislow et al., 2010). This research domain criteria perspective emphasizes considering measurable behavior obtained from tests or activities as possible end-products of a line of genetic, physiological, and neural systems' function/dysfunction that converge from a variety of sources to manifest as a "disorder." This approach would greatly assist in identifying the underlying neurodynamic mechanisms of specific symptoms, and it would likely lead to the identification of large-scale, distributed brain networks underlying a "disorder" if the diagnosis truly represents a specific entity. Further, it is hoped that this approach will contribute to more effectively developing specific evidence-based treatment approaches to better serve the pediatric population that continues to present with comorbid diagnoses.

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