Appendix A:
Evidence For Sensory Processing Disorder

INTRODUCTION

In 1999, the KID Foundation formed the Sensory Processing Disorder (SPD) Scientific Work Group (SWG), a multidisciplinary collaboration of leading scientists from university-based research institutions, to stimulate cross-disciplinary research into sensory processing and sensory processing impairments, supported by the Wallace Research Foundation and the NIH. Areas included in past and ongoing research include: neuro-physiological reactions to sensory stimuli in children and adults; sensation processing at the neural level, sensory-related behavior, attention and emotion regulation, animal models of neuropathology, genetic studies, and studies related to clinical issues such as the utility, sensitivity/specificity, and discriminate validity of the diagnosis of SPD.

The SPD Scientific Work Group is submitting this proposal to include Sensory Processing Disorder in the Diagnostic and Statistical Manual V. This appendix to the application includes a summary of empirical data providing evidence about the existence of the SPD syndrome, based primarily on studies conducted by SWG members. The information herein includes: published, submitted, and in process work. Ongoing research is occurring in all areas cited in this Appendix. Members and affiliations of the SPD SWG are:

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**Pennington’s Model of Syndrome Validation**

This Appendix is organized by Pennington’s model of syndrome validation (Pennington, 1991; Pennington, 2002) which suggests that empirical data, when provided in five areas, increases the likelihood that a new syndrome is valid. The five domains are: neuropathology, signs and symptoms, developmental trajectory, etiology, and treatment effectiveness (see Figure 1 below). “If a syndrome is valid, it will satisfy tests of both convergent and discriminant validity across [these] levels of analysis” (Pennington, 1991, p. 24). Thus, if a condition is homogeneous across these five domains, and can discriminate the condition from other disorders across these five domains, likely it is a syndrome.

*Figure 1. Pennington’s Model of Syndrome Validation*

Pennington (2002) suggests that most disorders are defined first behaviorally with a set of signs and symptoms that comprise a phenotype of the disorder. He further suggests that while genetic and neuropathology studies can not progress without designated behavioral phenotypes, brain and genetic studies can force revision in phenotype descriptions, thus refining the syndrome definitions and subtypes. Also needed to verify a syndrome is evidence that the condition is *universal*, e.g., all individuals with the condition exhibit similar patterns, and evidence that the condition is *specific*, e.g., individuals without this disorder do not exhibit these signs.

**Scientific Workgroup Research on SPD**

Empirical data on sensory processing and SPD were gathered in each area of Pennington’s model, by the SPD scientific workgroup 1995 – 2007. The SPD studies reported here are incorporated within current, ongoing programs of research. Research on SPD is in its early...
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stages. Empirical data derived from multiple ongoing programs of research are summarized in the following pages. The following report highlights the consistent results that have emerged so far:

- First, individuals with SPD have a different and less organized pattern of responding to sensory stimulation in both the autonomic nervous system and central nervous system. Psychophysiologic data (e.g. electrodermal activity and EEG/event-related potential studies), suggest that individuals with SPD exhibit atypical and/or unique patterns of physiological functioning during and after sensory stimulation (e.g., differences from typical controls and individuals with other disorders on autonomic nervous system arousal and state, and on cortical function during the classic stages milliseconds after stimulation, e.g., P50, P100, N100, and P200). In addition, cortical multisensory integration is much less organized in those with SPD, than it is in controls.

- Second, data suggest that clinical groups such as ADHD, Autistic Spectrum Disorder and other DSM-IV-TR conditions can be discriminated from SPD using physiological outcomes, standardized performance assessments, and parent/teacher-report measures.

- Third, familial/twin studies suggest a genetic component to the etiology of SPD.

- Fourth, animal studies suggest that dopamine-related correlates of SPD exist and specific pharmacological agents can impact one suggested mechanism of the disorder, sensory gating, and also affect dopamine-related correlates of SPD exist.

- Fifth, human studies suggest that a specific sensory-based occupational therapy approach may be effective in ameliorating features of the disorder.

- Sixth, the clinical utility of adding SPD to the DSM-V-TR, and the ability diagnose SPD accurately were studied. A new performance assessment the Sensory Over and Under-Responsivity Scale has an 88% overall hit rate in identifying SPD, with a 1.3% false positive rate.
RESPONSE TO QUESTION 1: DESCRIPTION OF NEW DISORDER

Summary

100 Character Summary – Sensory Processing Disorder: persistent atypical over-responsivity or under-responsivity to neutral sensation

Sensory Processing Disorder is characterized by persistent atypical over- or under-responsivity to neutral sensations. The existence of a unique syndrome is supported by data in the five syndrome validation areas outlined by Pennington (1991, 2002): neuropathology, signs and symptoms, developmental trajectory, etiology, and treatment effectiveness (Pennington, 2002). Neuropathology data (from human and primate studies) have found that some autonomic and central nervous system functions of individuals with SPD are abnormal compared to typical controls. Signs and symptoms data demonstrate that individuals with SPD can be discriminated from typically developing individuals in areas of attention, emotion, and sensory processing on both behavioral and physiological measures resulting in problems in daily life functioning. Developmental trajectory studies in animal and human models have demonstrated that behavioral signs and symptoms, along with sensory gating (one likely underlying mechanism of SPD), improve with maturity. Etiology findings based on twin studies and animal models have linked SPD to genetic factors, prenatal and birth risk factors, environmental exposures, and developmental and health factors. Finally, treatment effectiveness data demonstrate that sensory-based interventions for SPD result in better outcomes compared to no-treatment and active-placebo controlled treatments.

Definition/Description of Sensory Processing Disorder

The essential behavioral feature of Sensory Processing Disorder (SPD) is the presence of persistent atypical response patterns to neutral, everyday, “non-noxious” sensory stimuli. When the responses to sensation of individuals with SPD are compared to the responses of typically developing individuals at a similar age and stage of development, significant impairments are observed in SPD that are associated with problems in social, academic, and/or occupational functioning. In SPD, a response to sensory stimulation occurs; however, behavioral responses are poorly regulated e.g., the duration of behavioral response, the intensity of the responses, and/or the type of responses made to neutral levels of sensory input, are abnormal, resulting in poorly graded responses relative to the sensory input, and hence, non-adaptive behaviors are observed.

The range of normal responses to sensory input is quite large. A response pattern is only labeled “disordered” when an individual’s ability to detect, regulate, interpret, or organize responses to sensory input is so impaired that abnormal attention, emotion, cognition or motor responses are observed. Abnormalities in an individual can occur in one or more of the following sensory domains: auditory, visual, tactile, olfactory, gustatory, vestibular (relation to gravity seen in response to changes in position or movement), or proprioception (sensation from muscles and joints). Because the disorder is related to sensory stimulation in the environment, patterns of responsivity may vary throughout the day and from day to day, resulting in behavioral or emotional symptoms that may appear inconsistent across contexts (ICDL, 2005; Zero To Three, 2005; Miller, 2007c).
Two atypical patterns (subtypes) are observed in SPD:

*Sensory Over-Responsivity*, where individuals exhibit severe and persistent aggression, withdrawal, or fear (“fight or flight” reactions) to specific sensory stimuli, typically perceived as neutral, non-noxious, and harmless by others.

*Sensory Under-Responsivity*, where individuals exhibit reactions that are slower and less intense and less precise than is typical (e.g., unaware of basic sensory input easily recognized by most people such as pain, hearing their name called).

**Empirical Evidence**

**Neuropathology**

*Measures of Peripheral Autonomic Nervous System Function in SPD*

In Children.

Investigation into the neuropathology of SPD began with studies of the sympathetic nervous system because individuals with SPD exhibit dramatic “fight or flight” reactions to levels of sensory stimuli perceived as non-aversive or “neutral” by typically developing individuals. Sympathetic nervous system function may be examined using electrodermal activity (EDA). EDA measures changes in the electrical conductance of the skin associated with sympathetic nervous system activation of eccrine sweat glands (Boucsein, 1992), and is considered a sensitive index of sensation, attention, emotion and cognition, related to sympathetic arousal (Critchley, 2002). Consequently, the Sensory Challenge Protocol was created using EDA outcome measures to establish a reliable laboratory paradigm for examining children’s reactions to sensory stimuli by recording EDA resulting from serial introduction of sensory stimuli in five sensory domains during a “space trip” (Miller, McIntosh et al., 1999). Figure 2 displays the EDA responses of a typically developing child during the Sensory Challenge Protocol. Figure 3 shows two examples of EDA from a Sensory Over-Responsive child, with larger amplitudes, more frequent responses, and no habituation. Figure 4 displays the EDA responses of a Sensory Under-Responsive child.
Figure 2. Example of Electrodermal Response in a Typically Developing Child

From Miller, et al. 1999

Figure 3. Examples of Electrodermal Responses in Two Children with Sensory Over-Responsivity

From Miller et al 1999

Figure 4. Example of Electrodermal Responses in a Child with Sensory Under-Responsivity

From Miller et al 1999

Using the Sensory Challenge Protocol, McIntosh and colleagues (McIntosh, Miller et al., 1999) demonstrated that children with SPD respond at higher amplitudes and habituate more slowly to stimuli than typically developing children (see Figure 5 below). They also found a significant association between children’s physiologic responses and their functional behavior scores based on parent report.
Because regulation of an individual’s reactivity involves a balance of activity occurring within both the sympathetic and parasympathetic divisions of the autonomic nervous system, Schaaf and colleagues studied the Sensory Challenge Protocol using parasympathetic nervous system function outcomes in children with SPD. Their pilot study, using the parasympathetic measure vagal tone (Porges, 1985), demonstrated that children with SPD have poorer parasympathetic regulation than controls ($p < .05$) (Schaaf, 2001).

In Adults.

Smith and colleagues, (Smith, 2004) have been using autonomic nervous system measures to study adults with sensory-over-responsivity. Sixteen adults (8 SPD) were studied. Sympathetic nervous system response was assessed by continuously monitoring electrodermal activity as well as heart rate and peripheral blood flow. After 15 minutes of acclimation, 25 sensory stimuli were administered (5 each in 5 sensory systems: olfactory, auditory, visual, tactile, and vestibular).

Smith et al. (2004), report that the SPD group demonstrated a significantly larger skin conductance response following tactile stimuli (68±28%) than the control group (20±5.6%; $p = 0.04$). There were no differences in heart rate between groups or in the initial peripheral blood flow response. Smith et al., suggest that because the sensory over-responsive group had a relatively large initial skin conductance response to tactile stimuli without a relatively large peripheral blood flow response, sensory over-responsive persons may have an attenuated adrenergic sympathetic activation of the peripheral vasculature and/or an elevated cholinergic sympathetic activation of eccrine glands. In another study, Sensory Over-Responsive adults who score below $-2$ two standard deviations on the sensory sensitivity portion of the Adult Sensory

*Figure 5A. Discriminating Children with SPD from Typically Developing Children on Electrodermal Reactivity*

![Diagram](image-url)
Profile when presented with 108 dB white noise via headphones demonstrated an elevated initial skin conductance (Brown, 2002).

**Measures of Central Nervous System Function in SPD**

Autonomic nervous system measures such as electrodermal activity, vagal tone, heart rate and peripheral blood flow have the advantage of measuring an individual’s interaction with the environment (Stern, Ray et al., 2001), however we can only infer from these measures what might be occurring in the central nervous system.

Thus, with evidence of peripheral nervous system dysfunction, further investigation into the neuropathology of SPD naturally evolved into studies of central nervous system function. One measure being utilized is electroencephalography (EEG), which complements peripheral nervous system measures by providing a direct measure of cortical activity. Often event-related potentials (ERPs) are extracted from EEG to examine responses specifically related to sensory stimulation.

**Sensory Gating**

In Children. Davies and Gavin (Davies, 2007) hypothesized that children with SPD would demonstrate different sensory gating and organization of sensory information than controls. Using a paradigm similar to that used previously for assessment of responsivity in ADHD (Olincy, Ross et al., 2000), autism (Kemner, 2002) and traumatic brain injury (Arciniegas, Adler et al., 1999), Davies and Gavin used real-time measures of brain activation during the processing of sensory stimuli (clicks) which were time-locked to the occurrence of each sensory stimuli (i.e., P50, N100 and P200). They measured both amplitude (in microvolts) and latency (in milliseconds) of responses to sensory stimulation to examine brain processing of auditory sensory stimuli in 28 children with SPD and 25 typically developing children, group matched on age, 5-12 years, and gender (Davies & Gavin, 2007).

First, the paradigm presented tones at different frequencies and intensities known to effect the threshold at which sensory information is detected e.g., N100 and P200. Second, the paradigm presented clicks at 500ms intervals. It has been established that typically developing individuals have a smaller positive ERP response to the second of the two clicks at about 50 ms post-stimulation (P50), representing suppression of the second response, frequently labeled sensory gating.

Results for the first paradigm illustrated that children with SPD, unlike controls, did not have an increased response to increased intensity of stimulation. In addition, results for the second paradigm indicated that children with SPD had less auditory sensory gating than controls ($p = .04$), suggesting that children with SPD have more difficulty filtering out or “gating” irrelevant sensory information. This may explain in part the behavioral signs and symptoms such as distractibility, impulsiveness, disorganization and emotional ability seen in SPD. In addition, sensory gating improved with age for typically developing children, but not for children with SPD.
Examining group difference scores revealed that some children in the SPD group demonstrated over-responsivity compared to controls, while others demonstrated under-responsivity relative to controls. Children with SPD could be divided into Sensory Over Responsive and Sensory Under Responsive based on their sensory gating scores relative to typical children.

In Adults. Concurrently, Kisley and colleagues have been examining the relation of behavioral over-responsivity after auditory stimulation to the underlying neural mechanisms of auditory processing in adults. Using an EEG/ERP paradigm (including sensory gating and mismatched negativity) a significant correlation was found between sensory gating and sensory over-responding in a healthy adult sample (Kisley, Noecker et al., 2004). Responses at P100 were correlated with modulation of stimuli, whereas response at N100 was correlated with filtering out background sounds. These results suggest that individuals with SPD over-process stimuli of low salience rather than over-responding to all stimuli in their environment. Typically, as stimulus “relevance” goes down, so too does the brain’s automatic response to that stimulus. But individuals that endorse higher rates of sensory over-responding, as quantified by the Adult Sensory Profile, exhibit similar amplitude brain responses to nearly all stimuli, regardless of their “relevance.” Kisley et al, suggest that this finding implies that the brain is automatically processing all stimuli to the same extent, rather than appropriately increasing or decreasing its response according to higher or lower stimulus relevance. Thus, the brain of adults with SPD is not over-responding to all stimuli, but only to those stimuli that should be effectively filtered, or “gated” out, indicating that adults on the SPD spectrum, as well as children with SPD, have atypical patterns of sensory gating.

Multi-Sensory Integration.

Multi-sensory integration refers to the ability of the brain to integrate information from multiple sensory systems. In the natural environment, a wide variety of sensory stimuli occur in various positions in time and space. While the input from the senses is initially separate, sensory input ultimately converges in the brain so that individual elements in the external world can be integrated. This integrated representation of the environment creates the foundation for determining the nature and significance of events, and creating a meaningful response.

One methodology commonly used to evaluate multisensory interactions is comparing neural responses to two unisensory stimuli, with a third neural response to a simultaneous presentation of the same two stimuli. Multisensory integration is suggested when responses to a simultaneous presentation of two stimuli is greater than the sum of the two unisensory responses (Laurienti, 2005). Using this approach, studies in animals (Stein and Meredith, 1993) and humans (Calvert, 2004), provide evidence of multisensory integration in various brain regions including the midbrain, thalamus and cortex. In addition, psychophysical studies have shown that the integration of multisensory input profoundly influences behavior (Stein and Meredith, 1993). Numerous studies focused on specifying the neural basis for the behavioral enhancement that has been observed during multi-sensory integration are currently underway, setting the stage for improved understanding of the neural mechanisms underlying multisensory integration.
In Children. Brett-Green and colleagues have adapted an animal model ERP paradigm (Stein and Meredith, 1993) to measure multisensory auditory-somatosensory integration in typically developing children. Brett-Green is testing children with SPD, examining the hypothesis that multisensory integration is atypical in children with SPD. The first phase of the research involved testing a multisensory event-related potential (ERP) paradigm in a typical cohort. Demonstrated was that the paradigm can measure multisensory integration in typically developing children (Brett-Green, submitted). ERPs recorded from electrode site Cz in one typically developing 10-year-old-boy (TYP) are shown in Figure 6 on the left side.

The figure on the left shows three types of sensory stimuli administered in the multisensory paradigm: 1) unisensory auditory stimuli, 2) unisensory somatosensory stimuli and 3) multisensory auditory and somatosensory stimuli administered concurrently. The waveforms reflect the sensory information processing stages that occur mainly in the cortex, which have a characteristic sequence of amplitude peaks (P100, N100, P200). Notable is that the ERPs have clear amplitude peaks. In addition, the multisensory ERP denoting simultaneous auditory and somatosensory stimulation (green line) exceeds the amplitude of either of the unisensory responses (red = auditory; black = somatosensory). This indicates the response to multisensory stimulation is stronger than the sum of the two unisensory responses.

The second phase involved using the paradigm to assess multisensory integration in children with SPD (n = 9 to date). Preliminary multisensory ERP data demonstrate individual differences between typically developing children (TYP) and children with SPD. For example, see the data from one 11-year-old boy with SPD in Figure 6 right side. In the SPD case, neither the unisensory nor the multisensory responses show clear amplitude peaks. In addition, the responses to multisensory stimulation (green line) are not larger than either of the unisensory responses.
Figure 6. Comparison of One Typically Developing Child to One Child with SPD on Multisensory Integration Paradigm

Group Differences between SPD and Typically Developing Children. Thirteen typically developing children (TYP) were compared to nine children with SPD (sensory over-responsivity) and several differences were found. In typical children, the classic P100, N100 and P200 ERP components are evident. (See left side of Figure 7 on page 15.) This figure shows the grand average multisensory ERP waveform recorded with simultaneous auditory- and somatosensory stimulation in typically developing children at electrode site Cz. The classic P100, N100 and P200 ERP components are evident. Topographical maps (at right side of Figure 7, on page 15) show the voltage distribution across the scalp during multisensory stimulation.

In contrast, data obtained from children with SPD (see left side of Figure 8, on page 15) shows similar data obtained from children with SPD. Comparison of the multisensory response between typically developing children and children with SPD reveals several notable differences:

1. The amplitudes the classic ERP components are small for SPD, especially the P100 and N100.
2. The SPD group shows less decrement in response amplitude over time.
3. Topographical maps show the range of potentials (measured in µV) across the scalp is smaller for the SPD group. (See Table 1 below for values in µV at P100 and N100 for SPD vs. TYP.)

Table 1. Values of P100 and N100 in µV for SPD and TYP

<table>
<thead>
<tr>
<th></th>
<th>P100 (100ms)</th>
<th>N100 (160ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TYP</td>
<td>4.1</td>
<td>3.7</td>
</tr>
<tr>
<td>SPD</td>
<td>3.4</td>
<td>3.2</td>
</tr>
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</table>

The extent of the areas activated during multisensory stimulation are smaller in children with SPD compared to typically developing children. In addition, the responses of children with SPD begin earlier, continue longer, and appear to be less in frontal areas than the responses of
typically developing children. The tendency for longer response time in SPD is consistent with the autonomic nervous system findings from McIntosh et al. (1999, described above) that children with SPD have sympathetic nervous system responses (electrodermal activity) that habituate more slowly after sensory stimulation compared to the responses of typically developing children.

In addition, the brain areas activated during multisensory stimulation are clearly larger in typically developing children.
**Figure 7.** Multisensory integration in a group of typically developing children

**Figure 8.** Multisensory integration in a group of children with SPD
Developmental Trajectory

Animal Model Research

Stein and colleagues (Stein, in process) are using a number of animal models to determine when multisensory integration begins in development. Current results indicate that multisensory integrative capabilities of the superior colliculus do not develop if the superior colliculus is anaesthetized and thus inactive during the phase of early life when multisensory capabilities are first being formed. Deficits have been demonstrated specifically in the association cortex in animals whose superior colliculus is kept inactive. Stein’s study suggests that anomalies in SPD may reflect problems with the development of the cortico-collicular axis, providing hypotheses into the etiology of the symptoms of SPD.

Physiological Evidence

Analyses of developmental trends showed that sensory gating improves with maturity in typically developing children ages 5-12, but does not improve with age in children with SPD (Davies, 2007). This suggests that gating abilities in children with SPD do not change as a function of either biologically driven maturity (e.g., physical growth) or the accumulation of experiences across time (e.g., learning).

Clinical Evidence

In addition, the signs and symptoms of SPD differ by age. Although individuals may be diagnosed at any age, symptoms of Sensory Processing Disorder are generally present (as reflected in developmental histories) early in development and include poor or irregular patterns in sleeping, eating, eliminating, and self-calming. Infants and toddler over-responsive symptoms are often attributed to “colic,” “fussiness,” or “difficult temperament,” and if oral-sensitivity is present the infant is at high risk for severe feeding difficulties. Infant and toddler under-responsive symptoms include significantly less curiosity and exploration of new objects, and places. Sensory under-responsivity is usually undiagnosed in infancy/toddlerhood but retrospectively these children may be described as “easy babies,” “good sleepers,” and “remarkably undemanding.”

Pre-school or kindergarten teachers are often among the first professionals to identify children who have sensory problems because school environments require adaptation and accommodation to sensory events that continually challenge children with SPD. These children display over- or under-responsive behaviors to classroom noises (e.g., fans, bells, instrumental music); tactile experiences (e.g., art projects, hands-on exploration); tastes (e.g., snack time); lights (e.g., fluorescent lights, blinking lights); unexpected touch (e.g., bumped by children while waiting in line or playing at recess); and motion (e.g., playground equipment).

Children with SPD also often encounter increased academic difficulties in increasing grades (e.g., 1st-3rd grade) due to more complex multi-sensory requirements associated with reading, writing, memorization, and timed tests.
Adolescents and adults tend to display fewer overt signs of SPD as they employ various compensatory strategies for managing and processing sensory information. However, inability to maintain consistent levels of energy, mood, and attention across sensory environments persist. Sensory over-responsivity in particular poses risk factors for intimate relationships and self-regulation, while sensory under-responsivity tends to be a risk factor for processing speed of academic information and occupational functioning.

**Etiology**

**Twin Studies**

Genetic influences on tactile and auditory over-responsiveness is being studied by Goldsmith and colleagues (Goldsmith, 2006) using twin methodology. By comparing monozygotic (MZ) to dizygotic (DZ) twins, variations due to underlying genetics versus environmental factors can be estimated. Higher concordance rates in MZ twins indicate that status is partly heritable.

Probandwise concordance rates were calculated separately for MZ and DZ twin groups. Using a population-based sample of 1394 toddler aged twins, the incidence of Sensory Over-Responsivity (SOR) was widely distributed, with an accumulation of cases in the extreme range. SOR was relatively distinct from other common dimensions of childhood behavioral dysfunction although children with SOR were at increased risk for developing internalizing problems, dysregulation and maladaptive problems. Goldsmith and colleagues (2006) found that in both auditory and tactile responsivity, MZ twins were more similar than DZ twins, leading to the inference that SOR has some genetic influence. Concordance rates were MZ (.72) and DZ (.53) for Auditory SOR and MZ (.82) and DZ (.27) for tactile SOR. From this initial study of familial aggregation it appears that SOR has moderate genetic influences with tactile overresponsivity demonstrating somewhat greater heritability.

**Retrospective Study**

A preliminary study addressing the prevalence of pre-natal, birth, and early childhood health and development problems in children with SPD was conducted using a retrospective record review on all children with SPD ages 3-14 years, who were evaluated and treated at a large private OT clinic near Boston 1996 to 2006 (n=1000), excluding adopted children and children with medical diagnoses (May-Benson, 2006). A variety of possible risk factors exceeding base rates in the population were identified in families and/or children treated for SPD including:

- **Pre-natal and/or birth factors.** 25% pregnancy complications; 42% labor/delivery complications; 34% assisted deliveries e.g., vacuum, suction and forceps; 13% pre-term < 37 weeks; 5 % umbilical cord insults;

- **Developmental and health factors.** 49% skipped the “terrible two’s;” 37 % brief / absent crawling; 32% sleep or feeding problems; 62% chronic ear infections; 27% serious injuries or illness; 25% jaundiced at birth, 20% colic as infants.

**Animal Model Studies**
Although controlled human studies exploring the relation of SPD to other known etiologic risk factors have not yet been completed, Schneider and colleagues (Schneider, 2006) are using animal model research paradigms to explore whether genetic factors might make the brain more vulnerable to SPD, with the advantage over human research of systematically controlling for environment and random assignment to groups. In Schneider’s studies, the sensory processing abilities of Rhesus monkey offspring exposed to risk factors of moderate levels of 1) alcohol in-utero, 2) prenatal stress, and 3) postnatal lead exposure were compared to the sensory processing abilities of control monkeys. Significant differences were found in all risk conditions, with the exposed monkeys showing more likelihood of producing SPD offspring.

Pre-alcohol exposed.

This group was primate mothers who voluntarily consumed .6g/kg of alcohol (~ two drinks) each day during pregnancy and control monkeys (e.g., mothers received sucrose as control for alcohol). The control monkeys demonstrated a relatively large initial withdrawal response to sensory stimuli, followed by a decrease in response across trials (i.e., a typical habituation pattern characterized by gradually decreasing responsiveness to tactile stimuli over repeated trials). Monkeys whose mothers had prenatal alcohol exposure (n = 38) were discriminated by their higher initial magnitudes of tactile withdrawal response that remained high over trials. Schneider cites literature that demonstrates concordance with humans neonates exposed to prenatal alcohol who have reduced habituation to auditory and visual stimuli (Barron, 1992), reduced orienting to olfactory stimuli (Hunt, 2004), and various central nervous system deficits (Livy, 2003).

Pre-stress.

This group was primate mothers who experienced a 10-minute removal from home cage and transport to dark room where three random loud noise blasts were administered 5 times a week over a 10-minute period, (90-145 days from gestation). Offspring demonstrated slightly lower initial reactions to the feather (tactile stimulus), followed by slightly increased magnitudes of withdrawal from repeated trials (e.g., this was the only group to show sensitization with amplitude of responsiveness increasing over trials without habituation).

Lead Exposed.

Monkeys whose mothers were exposed to lead had abnormally strong patterns of withdrawal to touch stimuli with increasing abnormal reactivity after the first few trials.

Conclusions.

Several conclusions can be drawn from these animal studies. First, the pattern of habituation/sensitization observed to repeated tactile stimulation differs as a function of prenatal experiences. Controls with no stress, alcohol, or lead exposure demonstrate increased magnitude of response to the initial tactile stimulation followed by habituation, like human controls. In
contrast, monkeys exposed to prenatal stress showed behavioral sensitization, and those exposed to prenatal alcohol showed a higher magnitude of Sensory Over-Responsivity. This suggests that both alcohol and stress affect Sensory Over-Responsivity and behavioral regulation. Schneider et al., notes that neuromodulation is likely sensitive to prenatal perturbations, which may cause cascading effects later in development (Schneider, 2007). Furthermore, results suggest that reduced regulation to sensory stimuli appears to result in delayed motor abilities, learning deficits and other adverse developmental outcomes in primates.

**Treatment Effectiveness**

The gold standard for outcome studies is randomized controlled trials (Bury and Mead, 1998) comparing the targeted intervention to either an active Alternate Placebo, and/or to a passive placebo, No Treatment (e.g., a wait-list condition). Criteria for rigorous randomized trials are well established (Boruch, 1997; Bury and Mead, 1998) and mandate inclusion of four primary criteria: 1) an objectively defined sample that is homogeneous with regard to the impairment studied (Bulpitt, 1983); 2) a “manualized intervention” where treatment is detailed in a manual that others can obtain to replicate the procedures (Boruch, 1997) with a method to monitor adherence to the specified delivery of treatment (Ottenbacher, 1991); 3) outcomes that are meaningful, appropriate and sensitive to hypothesized changes (Fuhrer, 1997); and 4) methodology that is rigorous, e.g., a) random allocation to experimental and control treatment groups, b) blinded outcome evaluators, and c) adequate power to evaluate the significance of effects (Jadad, 1998).

**Previous Studies of Sensory-Based Occupational Therapy**

A review of existing treatment effectiveness studies for SPD found that none of the previously published research studies evaluating treatment outcomes met all four rigorous criteria for a randomized trial (and often did not meet even one criterion) (Miller, 2003). Thus as of 2003, the only accurate conclusion that could be proffered was that no rigorous evidence exists supporting or denying the effectiveness of this treatment.

In spite of relatively universal agreement about the lack of well-controlled outcome studies, significant controversy exists regarding the interpretation of the findings of over 80 published articles from the field regarding the effectiveness of occupational therapy for SPD, based primarily on belief systems, rather than empirical data. Relevant publications include two meta-analyses (Ottenbacher, 1982; Vargas and Camilli, 1999) and four research syntheses (Schaffer, 1984; Arendt, MacLean et al., 1988; Polatajko, Kaplan et al., 1992; Hoehn and Baumeister, 1994). One meta-analysis suggests that the intervention approach does have a positive effect, but the article is 25 years old (Ottenbacher, 1982). The four review articles conclude that previous studies are not rigorous enough to make valid conclusions, while at the same time they conclude that OT is not effective with SPD. The other meta-analysis suggests that the treatment approach has no positive effect (Vargas and Camilli, 1999), but significant methodological flaws occurred in this paper including: 1) studies had extremely small sample sizes (median sample size = 4.5 for 13 studies), 2) samples were heterogeneous regarding diagnostic groups, 3) such general descriptions of treatment were provided that replication was impossible and, 4) studies had such poor power that an effect was unlikely to be detected if present (Type II error).
Pilot Treatment Study.

Miller and colleagues therefore conducted a pilot treatment study (Miller, 2007a) correcting previous research limitations by utilizing a homogeneous sample, manualized treatment, outcome measures sensitive to change from treatment, and rigorous methodology. Results of the pilot treatment study (without control groups) demonstrated significant pre-post changes from treatment (Miller, 2007a) (See Table 2). The findings provided needed support for implementing a randomized treatment trial.

Table 2. Results of Pilot Treatment Study evaluating the effect of Occupational Therapy with Children who have Sensory Processing Disorder

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-Treatment Mean Scores</th>
<th>Post-Treatment Mean Scores</th>
<th>Change in Mean Scores</th>
<th>Effect Size</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leiter-R</td>
<td></td>
<td></td>
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<tr>
<td>Attention</td>
<td>5.88</td>
<td>6.32</td>
<td>0.43</td>
<td>0.29 (0.20)</td>
<td>0.20</td>
</tr>
<tr>
<td>Cognitive/Social</td>
<td>76.83</td>
<td>80.32</td>
<td>3.57</td>
<td>0.50 (0.03)</td>
<td>0.03</td>
</tr>
<tr>
<td>SSP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Score</td>
<td>-3.39</td>
<td>-0.39</td>
<td>3.11</td>
<td>1.62</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Vineland Socialization</td>
<td>79.04</td>
<td>89.47</td>
<td>11.95</td>
<td>0.82</td>
<td>0.002</td>
</tr>
<tr>
<td>CBCL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Externalizing</td>
<td>60.93</td>
<td>56.95</td>
<td>-4.19</td>
<td>0.54</td>
<td>0.02</td>
</tr>
<tr>
<td>Internalizing</td>
<td>61.57</td>
<td>57.48</td>
<td>-3.67</td>
<td>0.43</td>
<td>0.07</td>
</tr>
<tr>
<td>GAS</td>
<td>30.37</td>
<td>55.68</td>
<td>25.31</td>
<td>2.16</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Key:

<table>
<thead>
<tr>
<th>SSP- Short Sensory Profile</th>
<th>Vine- Vineland Behavior Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>GAS- Goal Attainment Scale</td>
<td>Leiter- Leiter International Performance</td>
</tr>
<tr>
<td>CBCL- Child Behavior Checklist</td>
<td>Rating- Revised, Parent Rating</td>
</tr>
<tr>
<td></td>
<td>EDR- Electrodermal Reactivity</td>
</tr>
</tbody>
</table>

Pilot Randomized Controlled Trial.

Next, Miller and colleagues (Miller, 2007b) conducted a pilot randomized controlled trial of the effectiveness of occupational therapy with children who have SPD. They evaluated the effectiveness of three treatment groups: Occupational Therapy (OT), an active placebo called the Activity Protocol (a play protocol), and a passive placebo e.g., wait-list condition. Twenty-four children with SPD were randomly assigned to one of the three treatment conditions. Pre- and post- measures of behavior, sensory, adaptive function and physiology were administered. The manualized treatment was administered twice a week for 10 weeks in 50-minute sessions. Fidelity to the treatment protocol was analyzed using Fidelity Measure (Parham, 2007). The overall design is depicted in Table 3 below.

Table 3. Design of Randomized Controlled Pilot Study
Group differences at time-point two, after the first 10 weeks of treatment have been analyzed and results demonstrate that the group that received OT, compared to the other two groups, made significant gains on Goal Attainment Scaling ($p <0.001$) compared to No Treatment (the passive control) and Activity Protocol (the active control). The OT group also made significantly greater gains in Attention compared to No Treatment ($p = .03$); and compared to Activity Protocol (trend toward significance) ($p = .07$). The OT group also made significantly greater gains and on the Cognitive/Social Composite of Leiter-R ($p = .02$) compared to Activity Protocol. For both the Short Sensory Profile (SSP) total test score, and the CBCL Internalizing composite score, change scores were greater, but not significant, in the hypothesized direction for the OT group. These findings are displayed graphically in Figure 9. Effect sizes were: Leiter-P Attention and Cognitive/Social scores, (0.29), SSP total (.08), Vineland Socialization (.14), CBCL Externalizing (.10) and Internalizing (.07), and GAS (1.62).

*Figure 9.* Findings of Randomized Controlled Pilot Study
Physiologically, even with a very small sample, the OT group showed greater reduction in amplitudes of EDR compared to the AP and NT group. Though not significant, a trend was observed for the OT group to improve in the hypothesized direction (reduced hyper-reactivity), although the trend was not significant due likely to the small sample size. Inspite of the small sample, this randomized trial of treatment suggests that OT may be effective in ameliorating some signs and symptoms of children with SPD.

These preliminary findings suggest the need for completion of a larger randomized controlled trial of the effectiveness of Occupational Therapy with a sensory-based approach, so that a more definitive conclusion can be offered with reasonable assurance that results are not attributable to chance and that external and internal sources of invalidity have been fully controlled.

Pilot Multiple Case Studies.

Psychophysiological measures show promise as treatment outcome measures in a multiple case study of children with SPD before and after treatment (Miller, in process) using the multisensory event-related potential (ERP) paradigm. ERP data is collected on children with SPD before and after 20 occupational therapy treatment sessions to determine if there is a measurable change in multisensory integration. In multiple case studies (n = 5), meaningful changes in ERP data have been found, suggesting the multisensory event-related potential (ERP) paradigm may be useful as a measure of treatment effectiveness. Data from one subject pre-and post-treatment is provided below to illustrate preliminary results for this pilot study (see Figure 10 below).

*Figure 10.* Sample of changes in ERP data after OT intervention in one child
The child before treatment (on the left) has no clear peaks in response to auditory or somatosensory stimuli, however the post treatment data appear to indicate improvement. ERP amplitude peaks are clearer, and the amplitude of the multisensory ERP after treatment clearly exceeds the amplitude of either unisensory response. Although these results are preliminary, they suggest that: (1) the multisensory ERP paradigm may be a useful measure of treatment effectiveness, and (2) sensory-based occupational therapy may produce measurable changes in multisensory integration at the neurological level.

Deep Pressure Treatment.

Deep pressure and proprioceptive stimulation is one of the primary treatment methods used by occupational therapists to reduce heightened sensitivity to tactile stimulation in individuals who exhibit Sensory Over-Responsivity. Thus Smith and colleagues (Smith, submitted) studied the physiological outcomes of applying heavy touch and deep pressure. In their paradigm, first pressure was applied to the torso and thighs using a weighted blanket to two groups of adults, one with Sensory Over-Responsivity and the other matched controls. Next, a pressurized suit was used to measure the effect of pressure during the Sensory Challenge Protocol (Miller, McIntosh et al., 1999). The results, as measured by electrodermal activity, peripheral blood flow, and heart rate variability, are noted below.

Electrodermal Activity. Smith et al (submitted), found that the Sensory Over-responsive group had a significant difference in initial skin conductance response (electrodermal reactivity) to tactile stimuli, however the intervention reduced the response in both groups (p=0.01), eliminating the initial difference (p = .02). The application of deep tactile pressure to the torso and thighs appeared to differentially attenuate the sympathetic response to sensory stimuli in the Sensory Over-Responsive and control groups (p=0.01). There was no difference in skin conductance response between the groups in the pressure condition (p=0.2).

When tested during vestibular stimulation, (tip back in a chair), for both groups, the skin conductance response to vestibular stimuli was reduced overall in the pressure condition (p=0.02). Skin conductance responses to vestibular stimuli were not significantly different
between the Sensory Over-Responsive and control groups in either the no pressure or pressure conditions. The skin conductance response for the visual, auditory, and olfactory stimuli also demonstrated no significant differences between sensory over-responsive and control groups, but showed reduced responses in the pressure condition compared to no pressure (p<0.1).

**Peripheral blood flow.** To tactile stimuli, the overall peripheral blood flow response was reduced in the pressure condition compared to the no pressure condition for both groups (p=0.004). In addition, peripheral blood flow response was reduced in the pressure condition when compared to the no pressure condition for both groups during vestibular stimulation.

**Heart Rate.** Normal heart rate variability was observed; however, no discernible changes were noted in heart rate as a result of any of the sensory stimuli in the study.

**Summary.** Overall this study suggests that deep pressure may have a calming effect on the sympathetic nervous system. In addition, results suggest that Sensory Over-Responsive adults may have a differential response to deep pressure when compared to control adults. Skin conductance and peripheral blood flow responses to interventions may be useful in further differentiating effective treatment of SPD.

**Pharmacological Treatment**

Levin and colleagues (Levin, Petro et al., 2004) are studying neural mechanisms of normal and impaired sensory modulation, as well as potential pharmacological therapeutic approaches using pre-pulse inhibition (PPI) paradigms in a rat model. PPI, easily modeled in experimental animals, is useful for determining of the neural bases for receptor systems. Studying inhibition of the startle response (e.g., another measure of sensory gating) has implications for neurotransmitter interactions and potential future pharmacological therapeutics for SPD.

Normally a warning stimulus reduces startle reactions. Recently, both nicotine effects and the effect of nicotinic glutamate interactions on pre pulse inhibition (PPI) startle after auditory and tactile stimulation (after nicotine has been administered, and after dizocilpine is administered) has been studied (Levin, Petro et al., 2004). They found that nicotine facilitates PPI over various intensities and inter-stimulus intervals. Notably, low doses of nicotine enhance the sensory gating deficit. When nicotine and dizocilpine are both administered, there is a decreasing effect on PPI as the dose of dizocilpine increases (dizocilpine acting as a blockade on the nicotinic response). However, when clozapine is added to nicotine and dizocilpine, the % PPI increases significantly in relation to the dose of clozapine administered.

With tactile startle, a dose effect of clozapine on the intensity of response is also noted. As the amount of clozapine increases, the effect of the dizocilpine on reducing the %PPI decreases. In other words, clozapine increases sensory gating when gating has been pharmacologically reduced (using dizocilpine.). Levin et al, (2005), suggest that the added blockade of DA D2, D4 or 5-HT2 receptors with clozapine added may effectively reverse the impairment caused by the blockade of NMDA.
Clozapine is often used in the treatment of schizophrenia to reduce the risk of suicide. Off label, it is used to decrease mania, tardive dyskinesia, insomnia, obsessive-compulsive disorder, and may have other uses. Use in humans with SPD has not been investigated yet in a controlled study. Study of combination treatments affecting nicotinic receptors and receptor systems acted upon by clozapine may be a fruitful avenue for study. This is particularly interesting in light of the findings by Schneider et al (in press) for primates with SPD-like symptoms. Schneider found the sensory over-responsive primates had reduced habituation to repeated stimuli that was associated with increased D2 receptor binding of radiotracer suggesting up-regulation (super sensitivity) of striatal D2 receptors.

Conclusion

Sensory Processing Disorder (SPD) is hypothesized to explain the abnormal behavioral responses observed after sensory stimuli is experienced. SPD occurs along a spectrum from mild to severe and affects both children and adults, causing either over-responsivity or under-responsivity symptoms. In the proceeding section we have highlighted the significant differences between individuals with SPD and typically developing individuals in a variety of areas needed for syndrome validation based on Pennington’s model (1999, 2002). These areas include underlying brain processes, signs and symptoms, developmental trajectory, etiology, and treatment effectiveness. Collectively the empirical findings above suggest that the diagnostic category of SPD may be valid. Future research in all areas above is ongoing and will advance the knowledge base related to this disorder significantly over the next few years.

RESPONSE TO QUESTION 2:
THE NEW DISORDER DESCRIBES A CONDITION THAT IS NOT ADEQUATELY COVERED BY THE EXISTING DSM-IV-TR CATEGORIES.

Summary

The key difference between Sensory Processing Disorder (SPD) and other DSM-IV-TR diagnoses is that SPD is the only diagnosis that has sensory processing impairment as a primary and essential feature. In addition, SPD is the only diagnosis where behavioral disruptions can be directly tied to responses to sensory stimuli. No other disorder has sensory impairments as a core feature, although some do include references to sensory functioning (e.g., autism, schizophrenia). Discriminant validity studies of SPD have found significant differences between SPD and existing DSM-IV conditions such as ADHD, Autistic Spectrum Disorder, and Fragile X Syndrome. Both physiological and behavioral data highlight that SPD is the only condition that has sensory processing impairment as a universal and specific feature. In addition, cases of “pure SPD” have been identified in which subjects did not meet criteria for other DSM-IV conditions, but did meet criteria for SPD.

Empirical Data Comparing SPD to Other Conditions

Work by some members of the SPD Scientific Work Group has focused on discriminant validity of SPD compared to certain DSM-IV conditions, specifically Attention Deficit Disorder with and
without hyperactivity (ADHD) and Autistic Spectrum Disorder (ASD). Data related to these studies is summarized below.

In addition, Pauls and colleagues are currently conducting a longitudinal family study of Sensory Over-Responsiveness in individuals with Attention Deficit Hyperactivity Disorder (N=200), Obsessive Compulsive Disorder (N=200) or Gilles de la Tourette (N=200) Syndrome and their first-degree relatives. It will be the first systematic study designed to obtain discriminant validity data in a prospective sample of children with psychiatric disorders and their families. In addition, DNA is being collected from all families participating in this study. Since data is being collected from all family members, examining the familial patterns and testing specific genetic hypotheses regarding the transmission of Sensory Over-Responsivity within families is planned. Future studies can include candidate gene studies to provide more quantitative discriminant data comparing SPD to those disorders (Pauls, in process).

These studies will build on results obtained from other discriminant studies by members of the SPD Scientific Work Group outlined below.

**Attention Deficit Disorder (ADHD)**

The following summarizes physiological and behavioral differences that have been found between children with ADHD and SPD both physiologically and behaviorally. In general:
- the group with SPD does not habituate to repeated sensory stimulation, while those with ADHD do;
- Individuals with SPD have more sensory aversion and withdrawal behaviors compared to individuals with ADHD;
- Individuals with SPD can inhibit responses on a computerized rapid response task but those with ADHD can not;
- Behaviorally, more sensory aversion and withdrawal responses are noted in SPD. Individuals with SPD report problems with sustained attention, impulsivity and activity as being less salient than sensory aversiveness, while the opposite is reported for those with ADHD.

**Sensory Responsivity**

**Introduction**

Mangeot et al (Mangeot, Miller et al., 2001) compared 26 children with ADHD to 30 typically developing children and found behaviorally, that ADHD is characterized behaviorally by inappropriate impulsivity, inattention, and hyperactivity; however, these behaviors are not seen specifically in response to sensory stimuli, but rather appear fairly universal across settings.

> When the sensory functioning of children with ADHD is compared to typically developing children, the group with ADHD displayed significantly greater abnormalities in sensory responsivity on both physiological and parent report measures (Mangeot, Miller et al., 2001; Dunn and Bennett, 2002).
Physiologically, children with ADHD demonstrate significant differences from typically developing children in response to sensory stimuli, particularly on the first stimuli of a set of repeated stimuli.

However, of note is that children with ADHD display significant variation in response to sensory stimuli (Mangeot, Miller et al., 2001).

In order to begin examining if ADHD and SPD were the same syndrome (e.g., all children exhibiting symptoms of one disorder would be expected to exhibit symptoms of the other disorder), Miller and colleagues studied in a national sample of 2410 children (from national standardization of Leiter International Performance Scale – Revised; Roid and Miller, 1997), who were stratified by age, gender, ethnicity and socio-economic status. Ahn et al., (Ahn, Miller et al., 2004) found that 181 children (approximately 7.5 % of total sample) had significant symptoms of either atypical sensory responsivity or attention problems.

If ADHD and SPD were the same syndrome, all children exhibiting symptoms of one disorder would be expected to exhibit symptoms of the other disorder. In fact only 74 children exhibited symptoms of both disorders (~ 3% of total sample or 40% of impaired sample). Fifty-seven children exhibited only ADHD symptoms (2.4% of total sample or 31.5% of impaired sample) and 50 children exhibited only symptoms of disordered sensory processing (~ 2% of total sample and 27.5% of impaired sample) (see Table 4 below). The preliminary evidence from this study supports existence of two separate syndromes, ADHD and SPD, although a high co-morbidity rate (40%) appeared to exist.

### Table 4. Percent of General Population with Symptoms of Attentional vs. Sensory Disorders in a National Stratified Sample

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>50</th>
<th>/4</th>
<th>124</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of National Stratified Sample with Symptoms of Sensory and/or Attention Impairments</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ahn, Miller, et al., 2004

As a follow up, Miller and colleagues (Miller, Reisman et al., 2001) examined physiological reactivity in children with SPD (n = 32) compared to children with ADHD (n = 40) and controls (n = 46). The results demonstrated significant physiological differences between groups of
children with ADHD and SPD, particularly in the habituation variable. Children with ADHD did habituate to stimuli similarly to typically developing controls. In contrast, children with SPD did not habituate to sensory stimuli (Figure 11).

**Figure 11.** Discrimination of children with SPD and ADHD from Typically Developing Children during Sensory Challenge Protocol.

![Graph showing comparison of children with ADHD and SPD](image)

Behaviorally, the ADHD and SPD groups were compared on parent-rated behavior scales (i.e., Child Behavior checklist, Leiter International Performance Scale – Revised, and Short Sensory Profile). The characteristics (in Table 6) were found to have differences greater than .5 standard deviations between groups. The ADHD group had more difficulties with Attention, Impulsivity, Activity level, and Auditory Filtering, as well poor Social Abilities characterized by Aggression and poor Adaptation. By contrast, the SPD group had more difficulties with Tactile sensitivity, Taste/Smell sensitivity, Low Energy, and Withdrawal. The two groups were rated approximately the same on Anxiety, Social problems, Somatic complaints and Thought problems.

**Table 5.** Discriminating Behavioral Characteristics of ADHD vs. SPD

<table>
<thead>
<tr>
<th>Groups</th>
<th>Auditory Filtering</th>
<th>Attention Problems</th>
<th>Activity Level</th>
<th>Impulsivity</th>
<th>Adaptation</th>
<th>Aggression</th>
<th>Social Abilities</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ADHD</strong></td>
<td>-3.2</td>
<td>-2.3</td>
<td>-1.8</td>
<td>-1.9</td>
<td>-2.1</td>
<td>-1.8</td>
<td>-1.6</td>
</tr>
<tr>
<td>SPD</td>
<td>-2.6</td>
<td>-1.8</td>
<td>-1.1</td>
<td>-1.2</td>
<td>-1.7</td>
<td>-1.2</td>
<td>0.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Groups</th>
<th>Tactile Sensitivity</th>
<th>Taste &amp; Smell Sensitivity</th>
<th>Low Energy / Weak</th>
<th>Withdrawn</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SPD</strong></td>
<td>-2.6</td>
<td>-1.1</td>
<td>-3</td>
<td>-1</td>
</tr>
<tr>
<td><strong>ADHD</strong></td>
<td>-2</td>
<td>0.8</td>
<td>-2</td>
<td>0.5</td>
</tr>
</tbody>
</table>

From Miller et al., 2002
Finally, (Ognibene, McIntosh et al., 2004) conducted a pilot study comparing typically developing children (n = 25) to children with ADHD (n = 20), SPD (n = 11), or both ADHD and SPD (n = 12). Significant differences between groups was found, after controlling for problem behaviors, IQ, age, gender, and anxiety, on habituation and response inhibition. Although SPD and ADHD shared some behavioral features, children with SPD had a sensory habituation deficit (did not habituate to repeated sensory stimuli), and children with ADHD did not demonstrate that impairment (i.e., they did habituate to repeated stimuli). In contrast, children with ADHD demonstrated a significant response inhibition deficit on a computerized continuous performance task, whereas children with SPD did not have the same inhibition problem. These data suggest an apparent double dissociation between ADHD and SPD. Ognibene concluded based on this pilot data that habituation and inhibition may prove useful in differentiating these disorders (see Table 6).
Table 6. Comparison of ADHD and SPD on Sensory Habituation and Response Inhibition

<table>
<thead>
<tr>
<th>Groups</th>
<th>Habituates to repeated sensory stimuli</th>
<th>Inhibits on a computerized continuous performance task</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD Group</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>SPD Group</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>


Autistic Spectrum Disorder (ASD)

Although significant literature exists suggesting both Sensory Over-Responsivity and Sensory Under-Responsivity are present in children with autism by age 2.5 (Rogers, Hepburn, & Wehner, 2003), sensory dysfunction is not a core deficit in autism. Kientz and Dunn (1997) and VerMaas Lee (1999) found significant Sensory Over-Responsivity in children with autism compared to controls. Dunn and colleagues (2002) also report significant Sensory Over-Responsivity in children with Asperger’s Syndrome compared to controls.

In a physiologic study of the responses of children with Autism Spectrum Disorder to sensory stimulation, Miller and colleagues evaluated 30 children with ASD using the Sensory Challenge Protocol (Miller et al, 1999) with electrodermal activity as an outcome. Findings suggest that children with ASD (ages 5-12) fall into two groups. One group demonstrates higher levels of tonic electrodermal activity (e.g. general arousal state) maintained over sensory challenges (e.g. phasic reactions or “trait”). The other group demonstrates lowered tonic electrodermal activity (e.g. general arousal state is decreased) and they also display lowered reactions to sensory challenges (e.g. phasic reactions or “trait”) (Schoen et al, submitted). The data for these two ASD groups are displayed below in Figure 12.

Figure 12. Groups in ASD based on Electrodermal Activity during Sensory Stimulation

Grouping of Skin Conductance Level in Autistic Spectrum Disorder
Although most individuals with ASD do exhibit sensory symptoms, most individuals with SPD do not exhibit autistic signs and symptoms. The three core features of ASD, impaired interaction, impaired communication, and restricted repetitive, stereotyped patterns of behavior are not present in SPD unless the individual has co-morbid SPD and ASD. Studies evaluating theory of mind, repetitive movements and socialization/communication in SPD compared to ASD are initiated but have not yet been completed (Brout, in process).

**Fragile X Syndrome**

Fragile X syndrome is another clinical condition in which individuals of all ages exhibit quite significant Sensory Over-Responsivity (Miller, McIntosh et al., 1999). Physiologically, the group with the most severe sensory reactivity was observed in boys with Fragile X Syndrome who demonstrate significantly increased amplitude of electrodermal activity and poor habituation to sensory stimuli compared to controls (Miller, Reisman et al., 2001). These children differ from SPD, however, in cognitive level and genetic etiology.

**“Pure” SPD Case Studies**

A pilot study targeting 30 subjects is underway to ascertain the existence of individuals with SPD who do not meet criteria for other DSM-IV-TR conditions (Miller, in process). Subjects receive a battery of tests designed specifically to identify conditions such as ADHD, autism, OCD, and anxiety disorders, and are further evaluated by a developmental pediatrician and a psychiatrist to rule out psychiatric and medical conditions. To date, ten individuals have met the criteria for SPD only. This demonstrates that there are individuals who meet criteria of SPD and who do not meet criteria for other diagnoses listed in the DSM-IV-TR. The study will continue until 30 individuals have been identified to who meet the study criteria (Miller, in process).

**Conclusion**

The discriminant validity of SPD and several existing DSM conditions has been evaluated on both physiologic and behavioral levels. Discriminant studies using both physiological and behavioral measures have documented significant differences between SPD and ADHD. A possible double dissociation has been suggested with SPD having poor sensory habituation but adequate response inhibition, and ADHD exhibiting the inverse with poor response inhibition and adequate sensory habituation. Differences between Autistic Spectrum Disorder and SPD include socialization, communication and repetitive movements in the former but not the latter; and electrodermal activity higher and lower than typically developing individuals. Individuals with Fragile X syndrome have a genetic disorder in which severe Sensory Over-Responsivity is found; however, individuals with SPD, unlike those with Fragile X syndrome, have much higher intelligence (often above average) and no communication disabilities, and SPD is unlikely a single gene disorder. In addition, multiple case studies suggest that a group of individuals have significant sensory processing impairments in the absence of other diagnosed conditions. Further studies are needed to cross-validate findings above and to evaluate differences from other key disordered groups, including Generalized Anxiety Disorder, Obsessive Compulsive Disorder and Gilles de la Tourette Syndrome. These findings suggest strongly that the new disorder describes a condition that is not adequately covered by the existing DSM-IV-TR categories.
RESPONSE TO QUESTION 3: ADDING THE DISORDER WILL IMPROVE CLINICAL UTILITY

Summary

Studies of prevalence have found that 5% of children in the general population exhibit signs and symptoms of SPD severe enough to qualify for a diagnosis. Pilot consumer surveys of clinical populations of children suggest that parents support inclusion of a diagnosis of SPD in the DSM, primarily due to experiences of positive outcomes of sensory-based treatment for their children’s sensory symptoms, after having no responses to treatments for other diagnoses (e.g., ADHD, anxiety disorders, etc.). Surveys and focus groups of physicians have found that a majority of physicians are aware of SPD and support the inclusion of SPD in the revised DSM. Both consumers and physicians anticipate that the inclusion of SPD will lead to a better risk/benefit ratio for treatment of individuals with symptoms of SPD.

Prevalence Studies

For individuals with diagnosed developmental disabilities the rate of co-morbid SMD is estimated to be 40% - 80% (Baranek, Chin et al., 2002), depending on the specific developmental diagnosis. The prevalence of SMD in children in a general population is estimated in a recent survey study (Ahn, Miller et al., 2004). Parents of incoming kindergartners from a public school district were surveyed using the Short Sensory Profile, a parent-report screening tool assessing functional correlates of SMD (McIntosh, Miller et al., 1999). Of the 703 children surveyed, a conservative prevalence estimate suggested that 5.3% of the sample met criteria for SPD. In a subsequent study of 440 children who were gifted and talented (e.g. IQs +2 SD above mean), an even higher prevalence of SPD was demonstrated, 36%. See Table 8 below. (Miller, in process)

Table 8. Prevalence of SPD in Gifted and Talented Children

<table>
<thead>
<tr>
<th>SMD (No S/A)</th>
<th>Prevalence of SMD in Special Populations: Gifted and Talented</th>
<th>Cumulative Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frequency</td>
<td>Percent</td>
</tr>
<tr>
<td>Valid No SMD</td>
<td>317</td>
<td>75.1</td>
</tr>
<tr>
<td>Yes SMD</td>
<td>105</td>
<td>24.9</td>
</tr>
<tr>
<td>Total</td>
<td>422</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Carter and Briggs-Cowen are examining the prevalence of Sensory Over-Responsivity in a prospective study of a longitudinal birth cohort of children in the Greater New Haven area who have been followed from either 1- or 2-years of age and assessed with the Sensory Over-Responsivity Scale in Second Grade. Data about early emerging social-emotional problems and competencies, including a measure of sensory sensitivities at ages 1, 2, and 3 years of age and psychiatric diagnostic status in Kindergarten and Second Grade is available in the longitudinal study. By 2008, this project will provide data on the prevalence and correlates of Sensory Over-Responsivity in a representative community sample. It is also examining associations between infant/toddler and concurrent social-emotional and behavior problem correlates of Sensory Over-Responsivity in school-aged children.

Clinical Utility

Physician Responses

Two relevant studies have occurred examining clinical utility to date. The first was a series of focus groups led by Edward Goldson MD, Professor of Pediatrics at the University of CO Health Sciences Center. The second was a survey of 2400 physicians (pediatricians, neurologists, and pediatric psychiatrists) in Massachusetts (5% response rate). These studies show that most physicians are aware of the diagnosis of SPD and of those aware, a majority support inclusion in the revised DSM (64%). A small cadre of physicians are strongly opposed to inclusion (5%) and strongly advise parents not to seek treatment for SPD (10%). A group of physicians are either unaware of the diagnosis or are asking for addition information before deciding whether SPD should be included in the DSM (~30%). Details of these studies appear below.

Focus Groups with Pediatricians

Two pilot focus groups were conducted by Edward Goldson, MD, Professor of Pediatrics at the University of Colorado Health Sciences Center. Dr. Goldson and colleagues interviewed eight experienced Colorado pediatricians in two small groups to explore the perceptions of clinical providers about the clinical utility of adding SPD as a separate disorder to the DSM (Goldson, in process). These focus groups are being expanded to other medical and psychological disciplines in other cities nationwide in the next few years. The purpose of the focus groups was determine what pediatricians understand about SPD, whether they see children with these symptoms in their practices, and what advantages or /disadvantages they perceive by adding SPD to the DSM.

In general, the pediatricians were aware of SPD but requested clarification about the definition of subtypes. The pediatricians acknowledged that they were seeing children in their practices with the defined signs and symptoms of SPD, who tended to have diagnoses that included attention deficit/hyperactivity disorder, mood instability, and/or coordination disorder. However, most were unclear about how to provide services for these children. They reported that clinical care for these children is extremely time-consuming and taxing both for the families and as well as for physicians. Inaccurate diagnoses frequently result in children going from one specialist to another seeking better diagnoses and care. In some cases children are referred for developmental evaluations or are referred directly to occupational therapists where an evaluation for sensory functioning is provided and OT treatment instituted.
A further concern of the pediatricians was that the lack of an accepted diagnostic code makes it difficult for the physician to be reimbursed for his/her time, resulting in using a “pseudo-diagnosis” to access services for these children.

The focus groups provided preliminary evidence that physicians (pediatricians) are interested in learning more about SPD, how to manage children with this problem, and how to provide effective services for them. Both focus groups clearly articulated that inclusion of a new diagnosis would likely increase their own diagnostic accuracy, enhancing their ability to make appropriate referrals for intervention, improve outcomes for the children and their families (Goldson, in process).

**A Survey of Physicians**

A large survey study examining the beliefs and knowledge of physicians related to SPD was conducted in Massachusetts by mailing an 11-question survey to pediatricians, neurologists, and pediatric psychiatrists (May-Benson, T., Koomar, J. & Teasdale, A., 2006). The survey was mailed to 2400 physicians listed with the Massachusetts State Board of Registration in Medicine. Of the 122 surveys returned (5%), a majority (64%) supported the inclusion of SPD in the DSM, and a few were strongly opposed to inclusion (5%). Of the remaining respondents who did not express an opinion, many reported being unaware of the diagnosis. (See Table 8 below.)

*Table 8 Physician Survey Related to the Utility of SPD as a Diagnosis*

<table>
<thead>
<tr>
<th>Question</th>
<th>Results (n = 122)</th>
</tr>
</thead>
<tbody>
<tr>
<td>How familiar are you with SPD?</td>
<td>65% very familiar; 28% somewhat; 7% not familiar</td>
</tr>
<tr>
<td>Do you believe SPD is a valid diagnosis?</td>
<td>64% yes, valid; 26% unsure; 10% not valid</td>
</tr>
<tr>
<td>Do you support inclusion of SPD in the DSM?</td>
<td>66% support; 29% not definite, wanted more information; 5% strongly opposed</td>
</tr>
<tr>
<td>Do you encourage treatment for SPD?</td>
<td>64 % actively encourage; 26 neither encourage nor discourage; 10% actively discourage parents from seeking services</td>
</tr>
</tbody>
</table>

Particularly notable from this survey was that a large percentage (30%) of physician respondents wanted more information before deciding whether SPD should be included in the DSM revision. However, survey results suggest that a majority of physicians surveyed (64%) support SPD as a separate diagnosis from other conditions such as ADHD, Anxiety Disorders and Autistic Spectrum Disorder (May Benson, Submitted.).
**Consumer Responses**

To gather data from a consumer perspective on the clinical utility of SPD, a pilot survey was administered to 250 participants who attended educational SPD workshops (Ahn, in progress). More than half of the 124 respondents reported they were at the workshop as a parent (n = 67). First, families were queried about their experience of effectiveness and efficiency with regard to their children’s’ diagnosis. Respondents reported having spent an average of more than $3000, meeting with an average of 5 clinical professionals (e.g., physicians, psychologists), and receiving an average of seven different diagnoses (e.g., ADHD, anxiety, etc.), before obtaining a referral to an occupational therapist for evaluation of sensory processing abilities (Ahn, in progress). In addition, a qualitative review of descriptions of an open-ended question that asked about child diagnoses and symptoms found that behaviors that precipitated seeking clinical services, included tantrums, irritability, clinginess, inflexibility, and extreme emotional distress during daily routines (e.g., crying, aggressiveness, etc. associated with meals, traveling, bathing, etc.). When asked about effectiveness of previous treatments for these children, respondents reported mixed outcomes with medication management (e.g., Ritalin) or psychological treatments (e.g., family counseling), and reported positive outcomes with occupational therapy. From this pilot a more specific data are being collected through the KID Foundation website (see www.KIDFoundation.org/survey) and through other professional sources. Data on this survey will be collected through 2008 when results will be summarized and published. In addition, more than 6000 consumers have signed a petition posted on the KID Foundation website endorsing recognition of SPD in the DSM-V.

**RESPONSE TO QUESTION 4: A LOW RISK OF FALSE POSITIVES**

**Summary**

*Several valid and reliable parent and self-report screening measures exist to evaluate SPD. In addition, three performance measures exist. The newest performance measure, called the Sensory Over-responsivity and Under-responsivity Scale (SensOUR), demonstrates a low false positive rate using a cut-point of -2 standard deviations (False Positive rate: 1.3%; False Negative rate: 10.7%; Overall Hit rate 88%).*

**Instruments for Screening and Diagnosis**

Several parent and self-report screening measures exist to evaluate SPD, including the Sensory Processing Measure and three versions of the Sensory Profiles. Three performance assessments have also been developed: (1) The Sensory Integration and Praxis Scale (SIPT: Ayres, 1989), a diagnostic assessment widely used to evaluate related aspects of SPD (e.g. sensory discrimination, postural disorders, and praxis); (2) The Miller Assessment for Preschoolers (MAP: Miller, 1982, 1988) that evaluates related
aspects of SPD, but does not focus on Sensory Over- and Under-Responsivity; and (3) the Sensory Over-Responsive and Under-Responsive (SensOUR) Scale, which is under development (Schoen, Miller et al., 2005) These scales and available false positive data is provided below.

**Parent or Self Report Measures**

**The Sensory Profile(s)** (The Psychological Corporation: San Antonio)

The most widely used report measures are Dunn’s series of three scales: (1), the original Sensory Profile, and the School Companion, ages 3 to 10 (Dunn, 2001; 2006) for classroom and home; (2) the Adolescent and Adult Sensory Profile, ages 11 and older (Brown and Dunn, 2002); and (3) the Infant Toddler Sensory Profile ages birth to 3 years (2002). The Sensory Profiles examine sensory processing patterns in individuals who are at-risk or have specific disabilities related to sensory processing issues. Responses are based on self- or caretaker-reports. The scales have been standardized nation-wide (samples ranging from 500 to 1200). Resulting profiles from the measures highlight the effects of impaired sensory processing on functional performance in the daily life of an individual.

The SP measures have good reliability (coefficient Alpha’s ranging from .47-.91 on the Sensory Profile, from .64 -.78 on the Adolescent/Adult Profile and from .42 to .85 for the Infant/Toddler Profile). The adolescent/Adult profile correlates with the New York Longitudinal Scales Adult Temperament questionnaire (between .42 to .46), and the original Profile correlates with the School Function Assessment (.68 to .72) demonstrating some concurrent validity.

False positive rates using the Sensory Profile appear to be 19.9% for Factor 1 Sensory Seeking, 23% for Factor 2 Emotionally Reactive and 11.2% for Factor 5 Inattention/Distractibility.

**The Sensory Processing Measure** (Western Psychological Services, Los Angeles)

A new scale, the Sensory Processing Measure (SPM: 2006), is modeled on diagnostic subtypes in the Interdisciplinary Council on Developmental and Learning Disorders (ICDL, 2006) and the DC: 0-3 (Zero to Three, 2006). The SPM screens for Sensory Over-Responsivity and Sensory Under-Responsivity specifically with both home (caretaker) and school (teacher) scales. The SPM was standardized on 1051 children in grades K through 6 and has excellent reliability (scale internal consistencies range from .75 to .95; two-week test-retest correlations range from .94 to .98) and validity (evidence for validity include factor analytic studies, correlational results, and clinical discrimination studies). False positive rates are 15% using a cutoff T-score of 60 (lower bound of “Some Problems”) and 2% using a cutoff T-score of 70 (lower bound of “Definite Dysfunction”.)

**Performance Measures**
**Sensory Integration and Praxis Test (SIPT; Ayres, 1989)**

The Sensory Integration and Praxis (Ayres, 1989) scale has been widely used by occupational therapists to evaluate SPD based on Ayres’ (1972) constructs. Standardized nationwide on 1,997 children, the SIPT includes 17 subtests each having separate reported reliability and validity. SIPT Validity studies are numerous (Carrasco, 1991; Royeen, 1991; Walker and Burris, 1991; Lai, 1996; Mulligan, 1996; Rinner, 2002; Royeen and Mu, 2003). One study used 10,000 SIPT protocols to conduct a validity study using structural equation modeling (Mulligan, 1996). Mulligan found that the SIPT defines four separate “subtypes” including sensory over-responsivity. The SIPT does not provide a direct measure of Sensory Over-Responsivity and Sensory Under-responsivity, the two subtypes we are applying to include in DSM-V, but rather focuses on evaluation of other presumed aspects of SPD: sensory discrimination, postural disorder, and praxis, which are other presumed aspects of SPD.

**The Miller Assessment for Preschoolers (MAP; 1982, 1988)**

The MAP is an assessment for children ages 2 ½ to 5 1/2 that evaluates children’s neurological maturity and includes several subtests of sensory processing abilities. Standardized on 1200 children nationwide, the scale has good reliability (.98) and validity (e.g., discrimination between typically developing and pre-academic delay group: .75 specificity, .75 sensitivity). A longitudinal predictive validity study, evaluating the standardization sample four years after original testing, found an 80% correct hit rate, with false positives < 5% using a cutpoint 2 standard deviations from the mean.

**The Sensory Over-Responsive and Under-Responsive Scales (SensOUR)**

The Sensory Over-Responsive and Sensory Under-Responsive Scales (SensOUR; in development since 2004) measures sensory functions in all sensory domains (vision, audition, touch, olfaction, taste, proprioception and vestibular). The scale includes both a parent/self report inventory and a performance Assessment where specific standard items are administered by a trained examiner. Psychometrics of the instrument were established in three stages of study: 1) Instrument development; 2) Reliability and validity of the research edition and 3) Cross-validation of findings on the research edition with a second sample.

For the Over-responsive subtests, data were collected from typically developing individuals (N=104) and individuals with Sensory Over-Responsivity (N= 113), ages 3 to 55 in Phase 1. All items were reviewed for their internal reliability consistency, discriminant validity and construct validity and a revised Research Edition was constructed. Then a new unrelated sample was evaluated. Analyses of the Sensory Over-Responsivity portion of the SensOUR Assessment revealed moderate to high internal consistency reliability for the domains (.60 to .89) and the total test (.92). The reliability estimates for the Over-Responsive Inventory ranged from .65 to .88 for the domains, and .97 for the total test.
Construct validity of the scales included factor analysis of the Inventory and Assessment separately. A seven-component factor analytic solution for both the Assessment and the Inventory provided the most interpretable pattern of loadings with no singleton factors. Although groups with and without Sensory Over-Responsiveness were factored separately on the Inventory and Assessment, both had the same factor solution. Factor loading were .46 - .95 with the greatest number between .6 and .95.

With respect to discriminant validity the typically developing group was compared to the group with Sensory Over-Responsivity for the Assessment and the Inventory. For both the total test and domain scores discriminated groups (over-responsive vs. typically developing) at a meaningful and statistically significant level (significance by domain ranged from $p < .05$ to $p < .001$) (Schoen, in process). False positive rate was 1.3%, calculated using a cut-point of -2 standard deviations. To be considered “positive” for SPD both the Inventory and the examiner administered performance Assessment were rated “positive”. Data on the SensOUR included: (False Positive rate: 1.3%; False Negative rate: 10.7%; True Positive rate: 25.3%; True Negative Rate: 25.3%; and At Risk category: 37.3%; and an Overall Hit rate 88%) (Schoen et al., in process).
RESPONSE TO QUESTION 5:
DATA SETS AVAILABLE FOR REANALYSIS

Summary

As scientists have not historically been asking the set of questions needed to identify Sensory Processing Disorder, no current archived data sets contain sufficient items needed to meet SPD classification criteria—with the exception of the data sets used by members of the SPD Scientific Work Group (referred to in this proposal). While it is possible that other existing data sets (e.g. NIH, MTA study) may contain information related to sensory-based items, and item analyses might identify those that have relevance to SPD, it is unlikely that those existing data sets would include sufficient items needed to meet meaningful criteria for SPD.

No Existing Data Sets

All data sets from work of the members of the Sensory Processing Disorder (SPD) Scientific Work Group (SWG) used to derive data cited in the articles above are available for reanalysis. They include data from: Margaret Bauman MD, Harvard University Medical School; Margaret Briggs-Gowen PhD, Yale University; Alice Carter PhD, University of Massachusetts; Patricia Davies PhD, and William Gavin, CO State University; Hill Goldsmith PhD, University of Wisconsin Madison; Michael Kisley PhD, University of Colorado in Colorado Springs; Ed Levin PhD, Duke University, NC; David Pauls PhD, Harvard University Medical School; Mary Schneider PhD, University of Wisconsin – Madison; Roseann Schaaf PhD, Thomas Jefferson University; Sinclair Smith ScD, Drexel University and Temple University; Barry Stein PhD, Wake Forest College and research from The KID Foundation Research Institute: Lucy Jane Miller PhD, Roianne Ahn PhD, Barbara Brett-Green PhD, Jennifer Brout PsyD, and Sarah Schoen, PhD).

In addition, several national databases exist that might shed light on this issue, (e.g., the NIMH national ADHD Multi-site (MTA) study, and studies related to temperament, OCD and anxiety). However, scientists have not typically collected data related to Sensory Processing Disorder in research subjects. Therefore reanalysis would require item analyses of archived data sets, and are unlikely to contain the necessary raw data needed for meaningful SPD classifications. Currently no convenient databases exist from which a set of diagnostic screening questions could be pulled (except the data bases of the Scientific Work Group referred to in this proposal).


Stein, B. (in process). "Multisensory Integration from birth through early months in anaesthetized cats."


