Sensory-modulation disruption, electrodermal responses, and functional behaviors

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It was hypothesized that children clinically identified with sensory-modulation disruptions (SMD) would have atypical physiological responses to sensation, and that such responses would predict parent-reported behavioral responses to sensation. Nineteen children with clinically identified disruptions, aged 3 to 9 years, mean 6.0 years, and 19 ageand sex-matched healthy (control) children, aged 3 to 9 years, mean 6.6 years, were examined. The subjects were presented with five stimuli. Ten trials were conducted for each stimulus and the electrodermal activity of the child was recorded. Four children with SMD did not show electrodermal responses (EDR) to stimulation; all control children responded. Excluding non-responders, children with SMD showed more and larger EDR than control children. Participants with disruptions habituated more slowly to repeated stimulation, as measured by the number of responses to stimuli and proportion of stimuli that evoked responses. Children with atypical EDR had more parent-reported abnormal behavioral responses to sensation. Children with clinically identified SMD respond physiologically differently to sensory stimuli than typically developing children; these differences have ramifications for functional behavior.

Sensory modulation is the ability to regulate and organize reactions to sensations in a graded and adaptive manner (Avres 1972, Royeen and Lane 1991, Parham and Mailloux 1996). The term may refer to either physiological or behavioral adjustments in response to sensory stimulation. Sensory-modulation disruption (SMD) is hypothesized to result from disrupted nervous-system processing of sensory stimuli (Fisher and Murray 1991). At the physiological level, SMD refers to disruptions in the mechanisms of habituation and sensitization of the central nervous system (Kandel 1991). It is thought that the disruptions result from alterations in the structure and function of nerve cells, affecting synaptic transmission.

At the behavioral level, sensory modulation describes the complex process of perceiving sensory information and generating responses that are appropriately graded to or congruent with the situation. Individuals with SMD have hyperresponsive behaviors and/or hyporesponsive behaviors in response to sensory stimuli (Royeen and Lane 1991, Dunn 1997). Behaviorally, SMD presents as unusual patterns of sensation seeking or sensation avoiding (Parham and Mailloux 1996) frequently evoking 'fight or flight' reactions to harmless or non-noxious sensory input (Ayres 1979). Manifestations include distraction, impulsiveness, abnormal activity levels, disorganization, anxiety, and emotional lability that produces deficient social participation, insufficient self-regulation and inadequate perceived competence (Cohn and Miller 1999).

The prevalence of SMD symptoms in the general population is approximately 10 to 12% (estimates based on Ayres 1989, Ermer and Dunn 1998). Up to 30% of individuals with developmental disabilities have SMD (Baranek et al. 1997). Despite this, empirical research on the phenomenon is scarce. No one has determined whether individuals clinically identified as having SMD show physiological responses to stimuli that differ consistently from responses of those without SMD. Do individuals with clinically identified SMD show physiological abnormalities in response to sensory stimuli? Do these physiological abnormalities relate to specific functional behaviors? This paper presents the first laboratory evidence that children identified with behavioral SMD show abnormal physiological reactions to sensory stimuli, and that these atypical physiological responses are associated with higher frequencies of abnormal functional performance responses to sensation.

One way to determine the extent to which individuals respond to stimuli is to assess their electrodermal activity after stimulation. Electrodermal activity refers to the changes in the electrical conductance of the skin associated with eccrine sweat-gland activity. It includes two variables: firstly, the skin conductance level which is the slow, tonic change measured across many discrete stimuli; secondly, the electrodermal responses (EDR) related to specific stimuli which are the quick, phasic changes imposed on shifts in tonic level in conductivity (Fowles 1986). EDR (previously termed 'galvanic skin response') occur in the presence of startling or threatening stimuli, aggressive or defensive feelings (Fowles 1986), and during positive and negative emotional events (Andreassi 1989).

Individuals with conditions causing atypical responses to stimuli often exhibit abnormal EDR. In individuals with Down syndrome (Wallace and Fehr 1970, Clausen et al.

1976, Martinez-Selva et al. 1995), schizophrenia (Kim et al. 1993), and attention-deficit-hyperactive disorder (ADHD) (Satterfield and Dawson 1971, Fowles and Furuseth 1994) hyporesponsiveness is shown. Individuals with ADHD (Rosenthal and Allen 1978) and conduct disorder (Zahn and Kruesi 1993) demonstrate faster-than-normal habituation to repeated sensation, suggesting less sensitivity to the stimuli. Two groups demonstrate hyperresponsiveness: children with autism show greater reactivity (Bernal and Miller 1970) and arousal (Stevens and Gruzelier 1984) than typically developing children, although they do not always react to stimuli (van Engeland 1984); and people with fragile X syndrome show greater magnitude of response, more responses to each stimulation, and responses to a greater proportion of stimuli (Miller et al. 1999; see also Belser and Sudhalter 1995). They also appear to habituate less to repeated stimulation (Miller et al. 1999); this may be related to defensive reactions to stimuli (Boucsein 1992).

To evaluate whether individuals clinically identified with behavioral manifestations of SMD show atypical physiological responses to sensory stimuli, we recorded electrodermal activity during controlled sensory stimulation. Four hypotheses were developed.

The first hypothesis was that more children with SMD would fail to respond to sensory stimulation than children without SMD. This hypothesis was developed because some children identified as having SMD are reported to be extremely underresponsive to sensory stimuli (Kinnealey 1973, Knickerbocker 1980, Royeen and Lane 1991, Dunn 1997).

The second hypothesis was that children with SMD would show greater EDR after sensation than children without SMD (after excluding children with no EDR greater than 0.05 micrombos, i.e. non-responders). This was hypothesized because some children with SMD are described as being overreactive to stimulation (Royeen and Lane 1991, Parham and Mailloux 1996, Dunn 1997).

The third hypothesis was that children with SMD would show slower rates of habituation than children without SMD. This was hypothesized because most children with SMD appear hypersensitive and defensive to common stimuli.

The fourth hypothesis was that individuals with atypical EDR patterns would show more abnormal functional behavioral responses to sensation than those who showed midrange EDR patterns. This was hypothesized because clinicians originally identified SMD through observing children's difficulty in functional behaviors. It was thus explored whether such atypical functional behaviours were associated with abnormal responses at the psychological level (i.e. EDR).

Method

SUBJECTS

Nineteen children clinically identified as having SMD and 19 healthy control children, matched on age (mean SMD age 6.0 years; mean control age 6.6 years; t[36]=1.09, P=0.283, two-tailed) and sex, participated in the study. There were 14 boys and five girls in each group.

Children with SMD were recruited from the occupational therapy (OT) department at The Children's Hospital in Denver, CO, USA. Referrals to OT for outpatient evaluation are usually made because the child is experiencing difficulties adjusting to the requirements of home, school, or community life. The child may present with aggressive or withdrawn behavior, sensory or motor problems, inattention and impulsiveness, which disrupt the quality of life for the child and family. Referrals are generally made by physicians and teachers, although occasionally by parents. Inclusion was based on an examiner's rating of behavior during intake testing, a telephone interview with parents, and a detailed open-ended parent interview by the second author. Children with diagnosed medical conditions such as cerebral palsy, fetal alcohol syndrome, and autism were excluded, as were participants who had motor or behavior problems but did not have specific abnormal reactions to sensory stimuli.

Children in the SMD group demonstrated difficulties in behavior regulation during intake test administration, had reports by parents of significant symptoms in two or more sensory domains, and had confirmation of modulation difficulties during the parent interview. Specifically, children were described with either over- or underreactivity to sensation or both. Behaviorally, the children present with unusual patterns of sensation seeking or sensation avoiding. On the hyporeactive-to-sensation spectrum, behaviors include the seeking of all kinds of movement, compulsion to touch people and objects, turning the volume up high, and making excessive noise. On the hyperreactive-to-sensation spectrum, behaviors include negative response to unexpected touch, sounds, or bright lights; avoidance of certain tastes and smells; aggressive or emotional reaction to touch; anxiety or distress when the child's feet are lifted off the ground. These sensory disabilities resulted in problems with social participation, self-regulation, and perceptions of competence. Children with fragile X syndrome, Tourette syndrome (using ICD-9 codes, US Department of Health and Human Services, 1991), autism, or mental retardation (using DSM-IV codes, American Psychiatric Association 1994) were excluded. One member of the SMD group had ADHD. All had intelligence scores within normal limits (>85) on the Wechsler Intelligence Scale for Children-III (Wechsler 1991).

The control sample was also recruited from Denver, CO, -- USA. None had traumatic birth history, unusual medical conditions, atypical educational development, or traumatic life events. All had normal intelligence, and demonstrated ageappropriate behavior and learning ability as reported by their parents.

LABORATORY PROCEDURE

Figure 1 presents a diagram of the Sensory Challenge Protocol, a procedure which gauges the responses of individuals to repeated sensory stimulation (Miller et al. 1991, see Appendix for a full description). The protocol comprises 50 sensory stimuli, with 10 contiguous trials in each of five sensory modalities. Experimenters blind to the child's group administered each stimulus in a standard schedule 15 or 19 seconds apart with 20 seconds between each sensory modality. Each stimulus trial lasted 3 seconds. Stimuli were given in the following order: olfactory (wintergreen oil in vial), auditory (siren at 90 decibels), visual (20-watt strobe light at 10 Hz), tactile (feather lightly moved along the face), and vestibular (chair tilted back to a 30° angle).

Electrodermal activity was recorded throughout the session. Steps were taken to minimize participant anxiety and movement. The laboratory was fashioned to resemble a spaceship. Before and during the application of electrodes, children viewed a segment of a video in which technicians attach electrodes to astronauts. During the protocol, the participants' arms rested on the armrests of the chair. If the child moved excessively or if there were problems with the electrodes, the computer operator observing the session added a comment to the data file. The data analyst used this information when reducing and scoring the physiological data (see below). The extent to which participant movement may have affected data collected using our procedures was evaluated; movement does not appear to be a significant influence on our results (see Appendix I and Miller et al. 1999).

MEASURES

The sensory profile

A parent of each participant completed the sensory profile (Dunn 1994), a measure of functional behaviors associated with abnormal responses to sensory stimuli. Parents indicate the frequency with which their children exhibit abnormal behaviors in response to sensory stimulation. We used a 51item condensed research version of the profile (see McIntosh et al. 1999, for information on a short sensory profile). Determination of which items to use in the reduced version was based on several factors, including content of item, its association with other related items, and its ability to discriminate between EDR patterns. This scale assesses sensitivity of touch, vision, hearing, taste and smell, movement, auditory filtering, as well as low energy, and seeking sensation. Higher scores reflect more behaviors which are within normal limits. The possible range of total raw scores on this scale is 51 to 255.

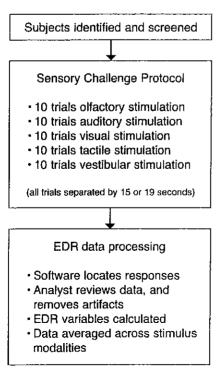


Figure 1: Steps in experimental procedure.

Electrodermal responses

We assessed EDR using changes in skin conductance associated with the presentation of stimuli (for basic procedures see Fowles et al. 1981; for additional information see Miller et al. 1999). We applied Autogenics 5-mm diameter electrodes (Stoelting Co., Wood Dale, Illinois, USA) to the fingertips of the second and third fingers of the right hand (Scerbo et al. 1992) and secured them with a velcro band. A Coulbourn isolated skin conductance coupler (S71-23) (Coulbourn Instruments, Allentown, Pennsylvania, USA) applied a constant 0.5 volt potential across the electrode pair, and conditioned the signal. Because the study was interested in responses to each stimulus (EDR) rather than changes in the slower fluctuating tonic skin-conductance level, AC coupling was used. This corrects for drifts in baseline conductance level over the extended time of the presentation of stimuli (see Boucsein 1992). A low-cut filter set to 0.2 Hz was used; signals above 0.2 Hz are passed without distortion in amplitude. A computer sampled the signals at 50 Hz, then digitized and stored the data.

The data were reduced and scored using a custom-written computer program (KIDCal, version 1.2, Denver, CO, USA, see Miller et al. 1999). The program established baseline skin conductance by examining the electrodermal readings before the stimulus presentation portion of the protocol began. The data analyst reviewed the entire tracing to evaluate the position of the baseline. The baseline was adjusted for six children with SMD and four control children because artifact or variability within the prestimulus period appeared to affect the baseline the program set. Baselines set by the analyst were fixed to the level at which most responses ended over the entire data collection period.

Peaks in electrodermal activity that were at least 0.05 micromhos in amplitude above the previously set baseline, occurred more than 0.8 seconds after each stimulus, at least 0.6 seconds after a previous peak, and at least 0.6 seconds before the next stimulus were marked as EDR. Responses of less than 0.05 micromhos were not considered valid (Dawson et al. 1990, Boucsein 1992).

After all peaks meeting these criteria were marked, the analyst reviewed the electrodermal tracing for the entire stimulation period. The data analyst compared the tracing with the comments of the computer operator and removed all artifactual peaks. Two SMD participants and two control children showed artifactual responses that required removal. Three variables were used to describe electrodermal responses. Because each was highly intercorrelated across the five sensory modalities (r=0.58 to 0.94; see also Miller et al. 1999), modalities were averaged to create a single score for each trial. Trials 9 and 10 were excluded due to missing data.

The first variable was the mean magnitude of response to each stimulus (Boucsein 1992). When there were multiple responses to a single stimulus, only the amplitude of the largest peak was used. As usually found in skin-conductance responses, magnitude data were positively skewed and therefore required logarithmic transformation before analysis (Dawson et al. 1990, Boucsein 1992). Due to the log of 0 (a non-response) being undefined, a score of 1 was added to all magnitudes before the transformation was performed (Kirk 1982).

The second variable was the number of responses to each

stimulus. This is the sum of peaks occurring between 1 second poststimulus and 0.6 seconds before the presentation of the next stimulus. We chose this window to avoid counting responses related to anticipation of stimuli.

The third variable was the proportion of sensory domains to which the person responded at each trial. For example, if a participant responded to the first olfactory, auditory, and visual stimuli, but not to the first tactile and vestibular stimuli, that person's proportion at trial 1 would be $0.60\ (3/5)$. To evaluate reliability of the electrodermal variables, we retested 26 children 1 week after their original tests (13 with SMD and 13 matched controls). All measures were strongly positively correlated across time (r=0.79 to 0.82).

Results

GROUP DIFFERENCES IN ELECTRODERMAL RESPONSES

There were four non-responders in the SMD group, and none in the control group. (Non-responders were defined as those who had no responses greater than 0.05 micromhos on any of the trials). The statistically significant difference in distribution supports the first hypothesis ($\chi^2(1)=4.47$, P=0.03). An absence of electrodermal responses to sensory stimuli is more common among children with SMD than control children.

These non-responders decrease the average EDR levels of the SMD group. Therefore, we excluded them and their matched controls when evaluating whether children with SMD are more reactive to stimuli than children without SMD and whether the SMD children habituate less than do typically developing control children. Ten boys and five girls remained in each group. The groups did not differ in age (mean age of child with SMD, 6.1 years; mean age of control child, 6.7 years; t[28]=1.19, P=0.24, two-tailed). Within the SMD sample, the EDR tracings showed a hyperresponsive pattern. Figure 2 is an example of this pattern from the SMD group. Compared with the normal tracing in Figure 3, the line in Figure 2 displays larger amplitude responses and more responses after each stimulus.

Three EDR variables (magnitude, number, and proportion) were used to test our second and third hypotheses. For each variable, group (SMD versus control) by trial (order of stimulus, with responses averaged across sensory modalities) ANOVAS were computed.

The hypothesis that children with SMD would have highermagnitude responses to sensory stimuli and less evidence of habituation over repeated exposure to the same stimulus than control children was confirmed by the ANOVA. As displayed in Figure 4, children with SMD showed larger responses to stimuli (mean 0.063 log micromhos, SD 0.052) than the control group (mean 0.026, SD=0.020; F[1,28]=6.50, P=0.017). Both groups showed decreases in magnitude with repeated exposure (F[7,22]=8.25, P<0.001). Contrasts showed significant linear (F[1,28]=39.5, P<0.001), and quadratic (F[1,28]=20.4, P<0.001) trends in habituation. (As with subsequent contrasts involving habituation, only linear and quadratic results are reported unless noted.) Contrary to the third hypothesis, the group by trials interaction was not significant (F[7,22]=0.53); groups did not differ in change in response magnitude with repeated stimulation.

The second and third hypotheses were also tested using the number of responses to each stimulus. Figure 5 shows the mean number of responses across trials for each group. In accordance with the second hypothesis, children with SMD showed more responses to each stimulus (mean 1.17, SD 0.66) than children without SMD (mean 0.64, SD 0.54; F[1,28]=5.11, P=0.032). The whole sample showed fewer responses with repeated exposure (F[7,22]=6.81, P<0.001); contrasts show significant linear (F[1,28]=35.9. P<0.001) and quadratic (F[1,28]=23.3, P<0.001) patterns. The third hypothesis was supported by a significant group by trials interaction (F[7,22]=2.42, P=0.053). The two groups did not respond to repeated stimulation in the same way (i.e. they habituated at different rates). The only significant pattern of these differences was a third-order polynomial trend (F[1,28]=4.24, P=0.049),

The proportion of stimuli to which the child responded

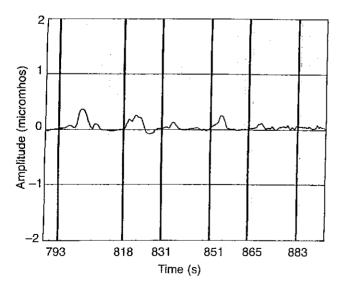


Figure 2: Hyperresponsive EDR pattern taken from the SMD group. Vertical lines show presentation of stimulus.

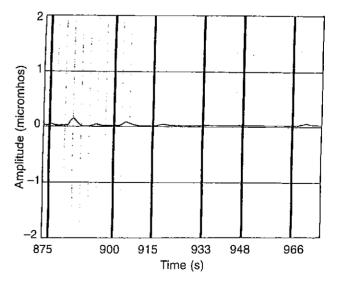


Figure 3: Normal EDR pattern taken from the comparison group. Vertical lines show presentation of stimulus.

at each trial was also used to test the second and third hypotheses. Figure 6 shows the proportion of responses by group and trial; children with SMD did not respond to stimuli significantly more times (mean 0.63, SD 0.30) than the control children (mean 0.44, SD 0.30; F[1, 28] = 2.07, P=0.107). For the combined sample, there was a significant effect of repeated exposure (F[7,22]=8.57, P<0.001); follow-up tests revealed significant linear (F[1,28]=34.9)P<0.001) and quadratic (F[1,28]=37.6, P<0.001) trends. The third hypothesis received borderline support. There was a marginally significant interaction (F[1,7]=2.31,P=0.063), with the proportions of the control group dropping more quickly across trials than those of the SMD group. Although the significance of the interaction was marginal, the presence of group differences is supported by

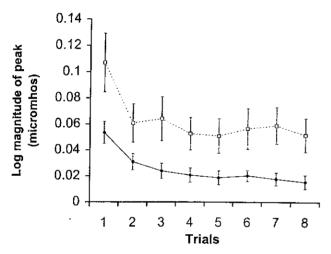


Figure 4: Magnitude (log) of primary EDR responses across trials, displayed for □SMD and ■control groups. Lines represent standard errors.

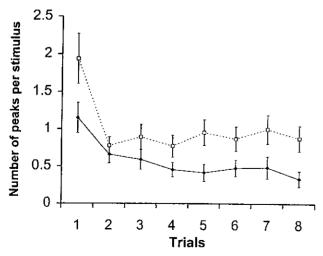


Figure 5: Number of peaks after each stimulus across trials. displayed for □SMD and acontrol groups. Lines represent standard errors.

the significant linear pattern in group differences across trials (F[1,28]=4.87, P=0.036).

PREDICTION OF FUNCTIONAL BEHAVIORAL REPORTS BY EDR PATTERN The validity of the hypothesis that individuals with abnormal EDR patterns would have lower scores on the reduced sensory profile than those with normal patterns was tested by dividing participants into three groups on each EDR variable (low, midrange, and high). Cut-off points for the groups were determined by looking for naturally occurring breaks in the distributions that would differentiate extreme low and high responses. The non-responsive group (N=4) for all variables) is defined above. The midrange group (for magnitude, N=24; number N=26; and proportion N=25) had an average response magnitude of 0.02 micromhos (log), 0.58 peaks after each stimulus, and responded to an average of 39% of stimuli. The hyperresponsive group (for magnitude N=10; number N=8; and proportion N=9) had a minimum mean response magnitude of 0.06 log micromhos (group mean 0.09), a minimum of 1.35 peaks after each stimulus (mean 1.78), and at least an average proportion of responses greater than 79% (mean 87%). All children in the nonresponsive groups had SMD. Approximately 30% of the children with midrange EDR patterns and 80 to 87% of the children in the high groups had SMD.

One-way ANOVAs were used to evaluate whether there were EDR group differences on the sensory profile. Followup t tests (two-tailed) were then conducted to determine if each extreme group differed from those with midrange EDR, and whether the hyper- and hyporesponsive groups differed from each other. We computed one ANOVA for each EDR variable. As hypothesized, there were significant differences in the sensory profile among EDR groups divided by magnitude (F[2,35]=8.74, P=0.001); number (F[2,35]=8.71, P=0.001); and proportion (F[2,35]=11.0, P<0.001). Tables I to III display t tests for the group differences. As predicted, the hyporesponsive group and hyperresponsive group each had significantly lower scores less typical on the sensory profile than did those with a midrange EDR pattern. The

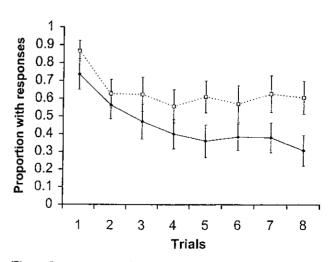


Figure 6: Proportion of stimuli to which participants responded at each trial, displayed for ⊇SMD and ■control groups. Lines represent standard errors.

non-responsive group displayed a marginally significant tendency to have lower sensory profile scores than the hyperresponsive group.

Discussion

These data provide the first evidence that children with clinically identified SMD show consistent physiological differences from children without SMD in response to sensory stimuli. Four conclusions can be drawn from the data. Firstly, more children in the SMD group than in the control group fail to respond to sensation on this physiological measure. Secondly, among participants who respond, the children with SMD show more electrodermal responses and responses of greater magnitude than the control children. Thirdly, the SMD group habituates more slowly than the group of control children, as measured by number of responses to stimuli, and proportion of stimuli to which they responded. Fourthly, those who have abnormal EDR patterns show more behaviors associated with abnormal responses to sensation.

When combined with other research, the data suggest that SMD is not merely an alternative label for children with learning or behavioral difficulties. For example, although children with SMD, ADHD, and conduct disorder display behavioral difficulties, the physiological responses found among those with SMD differ from findings for other clinical groups. The largest group of children with SMD in our study is hyperresponsive to stimuli. The opposite is true for individuals with ADHD (Satterfield and Dawson 1971, Fowles and Furuseth 1994). In addition, the slower habituation of those with SMD distinguishes them from people with ADHD (Rosenthal and Allen 1978) and conduct disorder (Zahn and Kruesi 1993), who show faster-than-normal habituation. Given that SMD is not reducible to these other disorders, it is important to expand research on the relatively understudied phenomenon of sensory modulation difficulties.

Some clues to understanding SMD may be in its relation to two other disorders. Both hyporesponsiveness and hyperresponsiveness in the SMD group is potentially similar to the findings among children with autism. Autistic children do not always respond, but when they do, they respond more strongly than typically developing children (Bernal and Miller 1970, van Engeland 1984). Further, the hyperresponsiveness among those with SMD resembles an attenuated form of the pattern in people with fragile X syndrome (Miller et al. 1999). Future work should establish the degree of similarity in responses across clinical groups, and determine if there are corresponding underlying disorders among these syndromes.

The findings of this study provide bases for additional research into the nature and causes of SMD. Firstly, what are the differences between non-responding SMD children and hyperresponding children? The non-responders could be hyperreactive children who have stopped reacting to external stimuli all together in response to overly intense stimuli (Royeen and Lane 1991, Dunn 1997). Alternatively, there may be different underlying disorders, one causing physiological hyperresponsiveness, and one causing physiological hyporesponsiveness. Researchers should examine the influence of variation in the intensity and duration of stimuli, examine the consistency of the patterns within individuals over time, and search for non-EDR differences between the hyper- and hyporesponsive groups.

Second, it is necessary to determine the extent of these physiological effects. In the current study, we established that children with SMD show atypical electrodermal activity after sensory stimuli. We do not know their reactions to other types of stimuli, or their electrodermal activity in the absence of stimuli. In addition, despite the strong association between our three EDR variables, the group differences were not found equally on each variable. Subsequent work should explore the meaning of any consistent differences among these variables. Further, differences were found in EDRs which indirectly assess sympathetic-nervous-system activity (Fowles 1986, Andreassi 1989, Dawson et al. 1990). The parasympathetic nervous system may be affected also, as it is heavily involved in regulation (see DeGangi et al. 1991).

The last finding in this study indicates that the physiological differences in children with SMD have behavioral ramifications. The ability of EDR to predict scores on a functional-behavior measure suggests that the physiological differences may cause difficulties in adjusting to typical life situations. This analysis needs to be replicated in another sample due to the selection of items for the short research version of the sensory profile being based, in part, on the

Table I: Comparison of short sensory profile scores between hyporesponders and midrange responders

A	mplitude	Number	Proportion
Hyporesponders (N)	÷	÷	i
mean (SD)	140 (24)	140 (24)	140 (24)
Midrange responders (N)	24	26	25
mean (SD)	209 (35)	208 (35)	210 (34)
t	3.78	3.78	3.96
P	< 0.001	< 0.001	< 0.001

Table II: Comparison of short sensory profile scores between hyperresponders and midrange responders

A	mplitude	Number	Proportion
Hyperresponders (/V)	10	8	9
mean (SD)	179 (32)	175 (33)	172 (29)
Midrange responders (N)	24	26	25
mean (SD)	209 (35)	208 (35)	210 (34)
t	2.35	2.34	3.00
P	0.025	0.026	0.005

Table III: Comparison of short sensory profile scores between hyporesponders and hyperresponders

	Amplitude	Number	Proportion
Hyporesponders (N)	4	4	4
mean (SD)	140 (24)	140 (24)	140 (24)
Hyperresponders (N)	10	8	9
mean (SD)	179 (32)	175 (33)	172 (29)
t	2.20	1.91	1.98
P	0.048	0.086	0.0~3

items' capacity to discriminate between our groups. Nonetheless, the finding of an association suggests a connection between physiological and behavioral responses to sensory stimuli. Both measures (EDR and the sensory profile) may prove valuable in assessing effects of intervention on SMD.

The clinical implications of this research are significant, though preliminary. These findings suggest that SMD may be a valid syndrome, and further research is needed to confirm the discriminant and convergent validity of the condition. If it is found to be a valid syndrome, then additional research should explore the efficacy of diagnosis with EDR and clinical tools such as the sensory profile. It will be critical to look further at similarities and differences between SMD and attention disorders, anxiety disorders, and specific learning disorders. Children with SMD show physiological responses to sensory stimuli that differ from responses of typically developing children and children with other disorders. These abnormal responses predict higher frequencies of parent-reported functional-behavior difficulties. Demonstrating these associations are the first steps in understanding the nature and causes of SMD. There is promise for quickly expanding knowledge of this phenomenon.

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Appendix Sensory Challenge Protocol

The Sensory Challenge Protocol was created to gauge individuals' responses to sensory stimulation, during which experimenters present sensory stimulation while electrodermal activity is recorded continuously. Except what might be surmised from observation, experimenters are blind to the condition of the participants. The description below explains how a child would be tested. (The instructions are similar for adults).

INTRODUCTION

The child is taken to a laboratory that is set up to resemble a spaceship (the child has been prepared for this). On one wall is a one-way mirror through which the computer operator can observe the session and make appropriate adjustments in marking events or annotations to the record, if needed.

A small wooden console painted to resemble a control panel for a spaceship is centered approximately 60 cm in front of the child's eyes. A hole in the console enables the child to see the screen of a 13 inch video monitor and a strobe light.

The child is asked to sit in a sturdy armchair placed on a 71 cm² tilt board anchored firmly on four 10 cm wooden cubes. The ambient light in the room is set at a low level throughout the protocol. The experimenter attaches electrodes to the child, as s/he watches a sec-

tion of a video depicting astronauts with electrodes attached to them. The video helps the participants become involved in and comfortable with the application of the electrodes. The computer operator and experimenter communicate through headsets. If either needs to halt the proceedings or make adjustments, it can be done with a minimum of disruption to the laboratory session. When the equipment has been tested and computer operator has set the child's baseline, the experimenter is signalled to begin the protocol.

There are ten contiguous trials in each of five sensory systems administered in one of the following orders: olfactory, auditory, visual, tactile, and vestibular; or tactile, visual, auditory, olfactory to control for possible order-effects; (vestibular remains last in both orders, as it is the one most likely to disrupt the proceedings). The stimuli are presented for 3 seconds each, and are administered on a standard, pseudo-random schedule 15 or 19 seconds apart, with 20 seconds between each sensory modality. The presentation of all stimuli, controlled by a recorded set of instructions given to the experimenter and the computer operator simultaneously through earphones, is as follows:

OLFACTORY

The olfactory stimulus is wintergreen oil (Walgreen's wintergreen oil, synthetic methyl salicylate nf), kept about 1.25 cm deep in a small vial with a cotton ball. The experimenter places the vial about 2.5 cm from the participant's nose, centered between nose and lips and then moves it in a 2.5cm path from the left to right to left, taking 1 second to make each excursion from side to side. The child is then asked to inhale the odor. The top of the vial is then covered to try to trap any lingering odors in the bottle and taken away from the child's nose.

AUDITORY

The auditory stimulus is a series of identical presentations on a tape recorder. A professionally recorded fire-engine siren plays at 90 decibels. As with the olfactory stimuli, there are 10 stimulation events each 15 or 19 seconds from the preceding stimulation event.

VISUAI

A commercially available 20-watt strobe light was set at 10 flashes per second, and positioned slightly below eye level. The strobe was attached to an Able-Net Incorporated power link, to enable the experimenter to turn the strobe on and off as directed by the audiotape using a foot pedal. The strobe is activated for 3 seconds then remains off until the next trial.

TACTILE

A cloth finger puppet with a 5 cm feather attached to his hat, from the Miller Assessment for Preschoolers (Miller 1988) was used as the tactile stimuli. The experimenter gently places the feather on the participant's right ear canal, then gently draws it along chin line to bottom of chin, and then raises the feather to the child's left ear. Each movement is timed to correspond with the seconds counted on the audiotape.

VESTIBULAR

The participant's chair rests on the top surface of a 'tilt board' supported by a 10 cm cube at each corner. The platform is 71 cm² of plywood attached to a rotation platform (62.5 cm) available from Achievement Products Inc. (Canton, OH, USA). Before administering the movement stimuli, the experimenter removes the two blocks located behind the participant's seat while holding the platform steady. The child is then smoothly and slowly tipped backward to a 30° angle.

If at any point the child experiences severe discomfort or expresses a wish to stop, the session is terminated. Every reasonable effort is made to encourage the child to complete the session.

References

- American Psychiatric Association. (1994) Diagnostic and Statistical Manual of Mental Disorders. 4th edn. Washington, DC: American Psychiatric Association.
- Andreassi JL. (1989) Psychophysiology: Human Behavior and Physiological Response. Hillsdale, NJ: Lawrence Erlbaum.
- Ayres AJ. (1972) Sensory Integration and Learning Disorders. Los Angeles: Western Psychological Services.
- (1979) Sensory Integration and the Child. Los Angeles: Western Psychological Services.
- (1989) Sensory Integration and Praxis Tests. Los Angeles: Western Psychological Services.
- Baranek GT, Foster LG, Berkson G. (1997) Sensory defensiveness in persons with developmental disabilities. Occupational Therapy Journal of Research 17: 173-85.
- Belser RC, Sudhalter V. (1995) Arousal difficulties in males with fragile X syndrome: a preliminary report. Developmental Brain Dysfunction 8: 270-9.
- Bernal ME, Miller WH. (1970) Electrodermal and cardiac responses of schizophrenic children to sensory stimuli. Society for Psychophysiological Research 7: 155–68
- Boucsein W. (1992) Electrodermal Activity. New York: Plenum Press. Clausen J, Lidsky A, Sersen EA. (1976) Measurement of autonomic functions in mental deficiency. In: Karrer R, editor. Developmental Psychophysiology of Mental Retardation. Springfield, IL: Thomas. p 39-91.
- Cohn E, Miller LJ. (1999) Parental homes for therapy outcomes: children with sensory modulation disorders. American Journal of Occupational Therapy 56. (Forthcoming.)
- Dawson ME, Schell AM, Filion DL. (1990) The electrodermal system. In: Cacioppo JT, Tassinary LG, editors. Principles of Psychophysiology: Physical, Social, and Inferential Elements. New York: Cambridge University Press. p 295-324.
- DeGangi GA, DiPietro JA, Greenspan SI, Porges SW (1991) Psychophysiological characteristics of the regulatory disordered infant. Infant Behavior and Development 14: 37-50.
- Dunn W. (1994) Performance of typical children on the sensory profile: an item analysis. Journal of Occupational Therapy 48: 967-74.
- (1997) The impact of sensory processing abilities on the daily lives of young children and their families: a conceptual model. Infants and Young Children 9: 23-35.
- Ermer J, Dunn W. (1998) The Sensory Profile: a discriminate analysis of children with and without disabilities. American Journal of Occupational Therapy 52: 283-90.
- Fisher AG, Murray EA. (1991) Introduction to sensory integration theory. In: Fisher AG, Murray EA, Bundy AC, editors. Sensory Integration: Theory and Practice. Philadelphia: FA Davis. p 3-26.
- Fowles DC. (1986) The eccrine system and electrodermal activity. In: Coles MGH, Donchin E, Porges SW, editors. Psychophysiology: Systems, Processes, and Applications. New York: Guilford Press. p 51-96.
- Christie MJ, Edelberg R, Grings WW, Lykken DT, Venables PH. (1981) Publication recommendations for electrodermal measurements. Psychophysiology Research 18: 232-9.
- Furuseth AM. (1994) Electrodermal hyporeactivity and antisocial behavior. In: Routh DK, editor. Disruptive Behavior Disorders in Childhood. New York: Plenum Press. p 181-205.
- Kandel ER. (1991) Cellular mechanisms of learning and the biological basis of individuality. In: Kandel ER, Schwartz JH, Jessell TM, editors. Principles of Neural Science. 3rd edn. Norwalk, CT: Appleton and Lange. p 1009-31.
- Kim DK, Shin YM, Kim CE, Cho HS, Kim YS. (1993) Electrodermal responsiveness, clinical variables, and brain imaging in male chronic schizophrenics. Biological Psychiatry 33: 786-93.
- Kinnealey M. (1973) Aversive and nonaversive responses to sensory stimulation in mentally retarded children. American Journal of Occupational Therapy 27: 33-40.
- Kirk RE. (1982) Experimental Design: Procedures for the Behavioral Sciences. 2nd edn. Pacific Grove, CA: Brooks/Cole.
- Knickerbocker BM. (1980) A Holistic Approach to the Treatment of Learning Disorders, Thorofare, NJ: Slack.
- Martinez-Selva JM, Garcia-Sanchez FA, Florit R. (1995) Electrodermal orienting activity in children with down syndrome. American Journal on Mental Retardation 100: 51-8.

- McIntosh DN, Miller LJ, Shyu V, Dunn W. (1999) Development and validation of the short sensory profile. In: Dunn W. editor. The Sensory Profile: Examiner's Manual. San Antonio, TX: Psychological Corporation, p 59-73.
- Miller LJ. (1988) Miller Assessment for Preschoolers (MAP). San Antonio, TX: Psychological Corporation.
- McIntosh DN, McGrath J, Shyu V, Lampe M, Taylor AK, Tassone F. Neitzel K, Stackhouse T, Hagerman R. (1999) Electrodermal responses to sensory stimuli in individuals with fragile X syndrome: a preliminary report. American Journal of Medical Genetics 83: 268-79.
- Parham LD, Mailloux Z. (1996) Sensory integration. In: Case-Smith J. Allen AS, Pratt PN, editors. Occupational Therapy for Children. 3rd edn. St Louis, MO: Mosby, p 307-55.
- Rosenthal RH, Allen TW. (1978) An examination of attention, arousal, and learning dysfunctions of hyperkinetic children. Psychological Bulletin 75: 689-715.
- Royeen CB, Lane SJ. (1991) Tactile processing and sensory defensiveness. In: Fisher AG, Murray EA, Bundy AC, editors. Sensory Integration: Theory and Practice. Philadelphia: FA Davis, p 108-36.
- Satterfield JH, Dawson ME. (1971) Electrodermal correlates of hyperactivity in children. Psychophysiology 8: 191-7.
- Scerbo AS, Freedman LW, Raine A, Dawson ME, Venables PH. (1992) A major effect of recording site on measurement of electrodermal activity. Psychophysiology 29: 241-6.
- Stevens S, Gruzelier J. (1984) Electrodermal activity to auditory stimuli in autistic, retarded, and normal children. Journal of Autism and Developmental Disorders 14: 245-60.
- US Department of Health and Human Services. (1991) The International Classification of Diseases. 9th Revision, Clinical Modification. Washington, DC: US Department of Health and Human Services.
- van Engeland H. (1984) The electrodermal orienting response to auditive stimuli in autistic children, normal children, mentally retarded children, and child psychiatric patients. Journal of Autism and Developmental Disorders 14: 261-79.
- Wallace RM, Febr FS. (1970) Heart rate, skin resistance, and reaction time of mongoloid and normal children under baseline and distraction conditions. Psychophysiology 6: 722-31
- Wechsler D. (1991) Wechsler Intelligence Scale for Children: Manual. 3rd edn. San Antonio, TX: Psychological Corporation.
- Zahn TP, Kruesi MJP. (1993) Autonomic activity in boys with disruptive behavior disorders. Psychophysiology 30: 605-14.