Journal of Child Psychology and Psychiatry 46:12 (2005), pp 1255–1268 doi: 10.1111/j.1469-7610.2005.01431.x

Annotation: What do we know about sensory dysfunction in autism? A critical review of the empirical evidence

Sally J. Rogers and Sally Ozonoff

M.I.N.D. Institute & Department of Psychiatry and Behavioral Sciences, University of California – Davis, USA

Background: Unusual responses to sensory stimuli are seen in many children with autism. Their presence was highlighted both in early accounts of autism and in more recent first-person descriptions. There is a widespread belief that sensory symptoms characterize autism and differentiate it from other disorders. This paper examines the empirical evidence for this assumption. **Method:** All controlled experimental laboratory investigations published since 1960 were identified through systematic searches using Medline/PubMed and PsycInfo search engines. A total of 48 empirical papers and 27 theoretical or conceptual papers were reviewed. **Results:** Sensory symptoms are more frequent and prominent in children with autism than in typically developing children, but there is not good evidence that these symptoms differentiate autism from other developmental disorders. Certain groups, including children with fragile X syndrome and those who are deaf-blind, appear to demonstrate higher rates of sensory symptoms than children with autism. In reviewing the evidence relevant to two theories of sensory dysfunction in autism, over- and under-arousal theory, we find that there is very little support for hyper-arousal and failure of habituation in autism. There is more evidence that children with autism, as a group, are hypo-responsive to sensory stimuli, but there are also multiple failures to replicate findings and studies that demonstrate lack of group differences. **Conclusions:** The use of different methods, the study of different sensory modalities, and the changing scientific standards across decades complicate interpretation of this body of work. We close with suggestions for future research in this area. **Keywords:** Autism, sensory, arousal, psychophysiology.

As described by Bailey, Phillips, and Rutter (1996) in a comprehensive review of the state of the science in autism, current neuropsychological theory in autism is heavily weighted towards cognitive models of primary deficit. While models involving theory of mind, affective dysfunction, and executive deficits are widely discussed and drive much of the current neuropsychological research, a major weakness of these neurocognitive theories is their inability to give a convincing account of the third symptom set in autism – repetitive behaviors, sensory abnormalities, and behavioral rigidity (Bailey et al., 1996). Only executive function accounts of autism address any of the third cluster of symptoms. Turner (1999) has suggested that repetitive and ritualistic behaviors reflect difficulties in inhibiting ongoing behavior and generating novel behavior, but recent studies have not found strong relationships between repetitive behaviors and performance on executive function tasks (Lopez, Lincoln, Ozonoff, & Lai, in press; South, Ozonoff, & McMahon, 2005). No neuropsychological theory of autism attempts to explain the unusual sensory behaviors seen in many children (e.g., over- or under-responsiveness, preoccupations with sensory features of objects, unusual reactions to sensory stimuli). There is a widely held assumption that sensory and repetitive behaviors are closely related. As we discuss below, it has been suggested that repetitive behaviors have sensory origins or that both types of symptoms are driven by chronic hypo- or hyper-arousal. These hypotheses have not been adequately examined, with only a small corpus of empirical studies, many of which suffer from methodological limitations.

In contrast, the current clinical and treatment literature treats sensory dysfunction as an established core deficit in autism, with a theoretical focus on possible abnormalities in subcortical neural systems. Sensory integration theory is widely applied to autism by practitioners (Watling, Deitz, Kanny, & McLaughlin, 1999), even in the face of nonsignificant empirical findings and questionable rationales for many of the sensory therapies (Baranek, 2002). Strong statements have been made for the importance of sensory integration therapy in the treatment of autism (Greenspan et al., 1997). Most recently, renewed interest in auditory integration therapies illustrates the clinical conviction that sensory dysfunction is central to autism.

The purpose of the present paper is to review the empirical evidence, gathered from controlled studies, that examines the following hypotheses: 1) sensory dysfunction characterizes autism and differentiates it from other disorders (Ginn, Berry, & Andrews, 1981), and 2) abnormalities in general arousal levels and impairment in habituation cause sensory

Published by Blackwell Publishing, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA

symptoms. The paper will first review the historical development of sensory theories in autism. Then it will review the empirical findings on sensory functioning in autism, focusing on experimental laboratory investigations that have included a control group. We examine the empirical evidence supporting specific sensory theories of autism. Finally, the paper will conclude with suggestions for a theoretical model for considering sensory symptoms in autism and organizing future studies.

Literature review search strategies

Procedures for producing a systematic literature review (Cook, Mulrow, & Haynes, 1997) were carried out as follows. The search for evidence focused on identifying empirical studies comparing data from a group with autism to a comparison group on measures involving 1) behavioral and physiological responses to sensory stimuli, and 2) physiological measurement of general arousal levels. A computerized search was conducted using the keywords **sensory**, **arousal**, **orienting**, **perception**, **physiological**, and **habituation**, with **autism**, **autistic**, and **childhood schizophrenia** (the term used for autism up until the early 1970s). An Ovid search engine was used to search PsycInfo and Medline. Given the relatively small number of studies reported, data provided in chapters, as well as peer-reviewed journals, were included. A total of 75 published articles about sensory behavior in autism (48 empirical and 27 theoretical or conceptual papers) were examined for this review.

Sensory theories of autism

First descriptions of sensory symptoms

In the first paper on autism, Kanner (1943) described behaviors that would currently be considered sensory symptoms in seven of his eleven original cases (e.g., watching bowling pins fall and jumping excitedly; fears of machine noises, movement on tricycles, slides, and swings; mouthing inedible objects; pleasure or fear of spinning things; flicking lights and shaking objects while observing; panicking when something covers the face). Kanner considered, but rejected, a sensory hypothesis. 'Yet it is not the noise or motion itself that is dreaded. The disturbance comes from the noise or motion that intrudes itself, or threatens to intrude itself, upon the child's aloneness. The child himself can happily make as great a noise as any that he dreads and move objects about to his heart's content' (p. 245). Only a few years later, however, Bergman and Escalona (1949) focused on the sensory area and offered the first version of a sensory hypothesis of autism. 'We see … indications that these children start life with a high degree of sensitivity against which they eventually succeed in building some defenses' (p. 345). They hypothesized that the child's need to protect himself or herself from the sensory onslaught resulted in developmental distortions that eventually led to the symptoms that Kanner had described. More recently, first-hand accounts of the experience of living with autism have emphasized sensory symptoms and sometimes suggest that sensory overload significantly influenced the social difficulties of the disorder (Grandin, 1992; O'Neill & Jones, 1997; Williams, 1994).

In the 1960s, the main hypotheses concerning autism-specific sensory dysfunction and its effects on motoric, social, and cognitive functioning in autism were developed and empirical studies began. These main hypotheses are still present in current writings and can be characterized roughly as: **over-arousal** theories, **under-arousal** theories, **inconstancy** theories, and **impaired crossmodal processing** theories.

Over-arousal theories

Over-arousal theories are based on the hypotheses that children with autism 1) are more easily aroused by and reactive to sensory stimuli than other children, and 2) fail to or are much slower to habituate to stimuli in the environment than other children. Both of these contribute to general levels of over-arousal and heightened arousal in response to specific stimulation. The first discussion of physiological over-arousal in autism was provided by Hutt, Hutt, Lee, and Ounsted (1964). The findings from which the hypothesis was based included EEG recordings indicating unusually high levels of waking activation, increasing stereotypy in situations with increased environmental stimuli, and correlations between levels of stereotypy and EEG activation. These researchers suggested that the brainstem reticular formation was sustained at chronically high and inflexible levels in autism, and led to blocking of neural sensory pathways in order to prevent further over-arousal. This blocking dampened responses to sensations and led to avoidance of novelty; sameness provided a means of avoiding increased stimulation. Over-activation led to development of stereotypic behaviors and general withdrawal from the social world as means of controlling over-arousal. When such compensatory strategies were not available, the child reacted to excessive stimulation in a catastrophic manner.

The over-arousal theory has had many supporters. The Tinbergens (1972) suggested that withdrawal due to over-stimulation originated from overly intrusive parenting. Another version of this general over-arousal theory can be found in Dawson and Lewy (1989). These authors argued that general over-arousal, a low aversion threshold, and a very narrow range of optimal arousal in autism lead to specific withdrawal from the social world because social stimuli are less predictable, more complex,

more novel, and thus more arousing than nonsocial stimuli.

Under-arousal theories

Under-arousal hypotheses of autism have been put forth by Rimland (1964) and DesLauriers and Carlson (1969). Rimland suggested a deficit in the reticular activating system that impairs the child's ability to connect previous experiences with current ones, preventing learning and generalization, and contributing to lack of typical reaction or under-reaction to stimuli. DesLauriers and Carlson (1969) suggested an imbalance in the reticular activating system that causes a suppression of the limbic system. They felt that some children with autism (those with over-functioning reticular activating systems) would be over-aroused, while others (for whom the system was under-functioning) would be under-aroused. However, for both groups of children, the suppression of the limbic system resulted in a 'stimulus barrier' for normal environmental levels of affective input, resulting in a sensory deprivation situation in which the child with autism is unable to receive sensory and affective messages. Lack of activation of the limbic system's capacity to establish reward systems and affective learning results in behavior that is repetitive, maladaptive, and aimless. A deprivation model is being discussed again in autism (see Dawson et al., 2004; Loveland, 2001; Mundy & Markus, 1997; O'Connor et al., 2000) as part of social deficit theories. In these models, the mechanisms that underlie social orienting and social reward systems are hypothesized to be faulty, resulting in infants who spend less time than typical infants engaged in social contact, due to their lack of social initiative and interest. Their attention and learning mechanisms are less focused on the social world, and thus more focused on the physical world, than typically occurs, and a cumulative deficit in social learning results, along with a hypertrophied interest and involvement with physical objects and nonsocial activities.

Perceptual inconstancy theory

An influential explanation of the sensory symptoms in autism, the perceptual inconstancy theory, was suggested and developed by Ornitz and Ritvo (1968) over several decades, beginning in the 1960s. This work further elaborated the earlier over-arousal theories of the Hutts and colleagues (Hutt, Hutt, Lee, & Ounsted, 1964) and was built on a model of brainstem dysfunction influenced both by earlier theorizing about the reticular activating system and by their own research on vestibular abnormalities in autism. In a position far removed from the psychoanalytic views of the time, Ornitz and Ritvo (1968) suggested five main symptoms characterizing autism, of which the **primary** symptoms included abnormalities in perceptual integration and motility patterns, and the more **secondary** symptoms involved language, social, and developmental rate abnormalities. They conceptualized autism as stemming from abnormal states of arousal due to brainstem abnormalities, resulting in the presence of fluctuating states of both over-excitation and over-inhibition. These abnormal and unpredictable states of arousal interfered with the child's capacity to maintain perceptual constancy by varying the child's awareness or experience of the same stimulus. The presence of these states was thought to indicate dissociation between facilitatory and inhibitory systems that regulate sensory input for processing. A variant of this hypothesis posed by Kinsbourne (1987) also emphasized disruptions in state regulation leading to fluctuating periods of over- and under-arousal. He suggested that repetitive movements (1980) and over-focused attention on details (1991) could be compensatory strategies for states of both over- and under-stimulation, not just one or the other.

Tanguay and Edwards (1982) suggested that distortions in sensory input, particularly auditory, in early infancy could lead to a failure to develop more complex cognitive abilities. This is a cascade theory of development similar to the deprivation theories described above, wherein early deficits in simpler processes directly impair the development of more complex skills. These authors further suggested that findings of physiological differences in older children with autism, like abnormal evoked potentials, represent not the primary deficit, but a marker of the early abnormal processes that derailed development ('the whisper of the bang'). They also suggested that studies trying to identify primary abnormal processes in autism will have to focus on much younger children who are at the beginning of the 'autistic process,' since examining autism much later in development brings many more variables into play.

Crossmodal impairment theories

A more current effort to conceptualize the various symptoms seen in autism, including sensory symptoms, has been offered by Waterhouse, Fein, and Modahl (1996). In a broad-based paper that attempts to bring together the known pathoneurophysiology of autism with current theory, the authors focus on hippocampal abnormalities described in the autopsy studies of Kemper and Bauman (1998). Waterhouse et al. (1996) suggest that difficulties with crossmodal integration of sensory information lie at the heart of the sensory symptoms of autism. They suggest that this could be caused by abnormalities in the mossy fibers of the hippocampus, which synapse on abnormal 'islandized' CA3 pyramidal cells. This abnormality could result in a failure to bind all incoming sensory information from the same event or context with the spatiotemporal

information resulting from the event or context, resulting in impaired crossmodal integration of information (see also Brock, Brown, Boucher, & Rippon, 2002, for a similar argument regarding the neural basis of weak central coherence in autism).

A physiological basis for poor integration of sensory information across modalities has recently been suggested by Chugani and colleagues (1997, 1999), derived from their efforts to understand the well-replicated finding of increased peripheral serotonin levels in a large minority of persons with autism. The authors reported two PET studies examining serotonin synthesis capacity of children with autism compared to controls. Their 1997 findings involved distinct differences in serotonin synthesis in frontal cortex, thalamus, and cerebellum in all the boys but not the girl with autism, compared to sibling controls. In the 1999 study, they added a control group with epilepsy, and reported differing patterns of serotonin synthesis capacity for children with autism, involving lower than normal values early in life, and higher than normal levels in later childhood. Furthermore, they describe results from animal studies that manipulation of serotonin levels in developing animals can create the kinds of brain changes found in autism by Kemper and Bauman (Chugani et al., 1999). The authors observed that the neural pathways that would be most affected by these differences in serotonin synthesis were important for language production and integration of sensory information. They suggested that this abnormality in serotonin production could account for the sensory symptoms of autism, among others.

Summary. In reviewing the development of ideas concerning sensory symptoms in autism, one is struck by the amount of attention this set of symptoms received early on from mainstream theorists and researchers in autism and the contrasting paucity of new ideas and approaches since that time. Current sensory integration theories of autism are directly related to the very first hypotheses regarding sensory symptoms, offered by Bergman and Escalona in 1949. While the perceptual inconstancy theory of Ornitz and Ritvo is no longer widely discussed, the over-arousal and under-arousal theories of autism first suggested in the 1960s are still part of mainstream clinical conceptualizations of autism that focus on sensory dysregulation as a primary symptom of autism (Greenspan et al., 1997; Zero to Three, 1994). While the early theories were based on then current scientific knowledge of sensory processing, a question to be raised concerns how well current sensory theories have incorporated new findings from neuroscience about sensory processing.

As Bailey et al. state, 'the notion of low-level sensory-perceptual deficits, such as stimulus over-selectivity or sensory dominance, [has been] replaced by the concept of a general high-level cognitive dysfunction in deriving meaning…' (1996, p. 100). However, it is our contention that any satisfactory comprehensive theory of autism must address sensory symptoms. We next turn to the empirical data generated by studies of sensory functioning in autism, focusing on experimental laboratory investigations using control groups.

Empirical studies of sensory function in autism

Although the purpose of this annotation is to review experimental investigations of sensory dysfunction in autism, we begin with a brief overview of the work that has been done using parent questionnaires as an initial method of examining autism-specific abnormalities in sensory reactivity. In comparison to children with typical development, an early study reported significantly more abnormal responses to sounds, but no differences in responses to objects, during the first two years of life in young children with autism (Ornitz, Guthrie, & Farley, 1977). Using a more recently developed standardized parent questionnaire, the Sensory Profile, Kientz and Dunn (1997) reported that 84 of 99 questions differentiated the groups. Scores of children with autism were significantly higher on 8 of the 10 factors on the Sensory Profile, again in comparison to children with typical development, in a second study (Watling, Dietz, & White, 2001).

The earliest study comparing children with autism to clinical controls was reported by Wing (1969), who observed that many of the symptoms seen in children with autism were also present in other clinical groups of children. On her questionnaire, children with autism were reported to have significantly more sensory-perceptual abnormalities than both typically developing children and a group with Down syndrome, but few differences in sensory responses were found relative to children with receptive aphasia and children who were both blind and deaf. More recently, Lord and colleagues, using the Autism Diagnostic Interview-Revised, reported a significantly higher rate and intensity of parent-endorsed sensory dysfunction in children with autism than in a clinical control group with general developmental delays (Lord, 1995). This finding was replicated in two larger samples, relative to age- and IQ-matched groups with other developmental disabilities (Lord, Rutter, & LeCouteur, 1994; Lord, Storoschuk, Rutter, & Pickles, 1993). Rogers and colleagues, using the Short Sensory Profile, found that very young children with autism and those with fragile X syndrome were reported by their parents to have significantly more tactile, taste/smell, and auditory sensitivity than children with developmental delays and typically developing children, but the autism and fragile X groups did not differ from each other in any area of sensory reactivity (Rogers, Hepburn, & Wehner, 2003). This was replicated by Miller and colleagues (Miller, Reisman, McIntosh, & Simon,

2001), also using the Short Sensory Profile, who found that responses of parents of children with autism, as well as two other clinical groups (fragile X and sensory modulation dysfunction[1]), differed significantly from responses of parents of typically developing children, but that the responses of the three clinical groups were quite similar.

To summarize studies using parent questionnaires, more sensory symptoms are reported in children with autism than in those with typical development, as well as those with general developmental delays. There appear, however, to be a few specific clinical groups (fragile X syndrome, deaf-blind) whose parents report as much or more sensory dysfunction than parents of children with autism. The parent report method has some obvious limitations, including both memory and interpretation issues. In the next sections, we examine the patterns observed by objective raters in laboratory situations and evaluate support for the various theories of autism-specific sensory abnormalities that have been put forth.

Experimental studies relevant to arousal theories

Comparison to typical controls. In one of the earliest investigations, Metz (1967) examined 'autistic hypersensitivity' to sound by comparing four groups of children: 10 with autism, 10 with 'schizophrenia',[2] 30 typically developing children, and 10 'unusually successful' children. The experimental paradigm involved a sound output device playing music, the sounds of human breathing, unorganized sounds from percussion instruments, and voices reading. Participants were allowed to control the volume of each sound with a lever. Children with autism differed significantly from the 'successful' children and those with schizophrenia (but apparently not from the typical group) in their preference for higher volume levels. However, the children with autism had greater difficulty than the others learning how to operate the equipment, so intellectual differences may also have been at work. Contrary to expectations, the children with autism preferred louder sounds, supporting a hypo- rather than hypersensitivity theory of autism.

Bernal and Miller (1971) examined electrodermal (EDR) and heart rate responses of 20 children with autism (ages 3–13, mean age 6.5 years) and 20 typically developing children matched for chronological age to repeated trials of changes in light and auditory stimuli. Both sensory reactivity and habituation were examined. The only significant group difference was decreased responses to the first three visual stimuli by the children with autism. The participants responded less intensely than controls to auditory stimuli of various intensities, with similar habituation rates. Responses were similar to children with mental retardation, according to the authors, and could be interpreted as demonstrating hypo- rather than hyper-arousal. Motor stereotypies were also recorded and it was found that they occurred primarily during periods of inactivity and ceased upon initiation of the experimental stimuli. This did not confirm the hypothesized relationship between sensory stimulation and stereotypies put forth by the Hutts (Hutt et al., 1964, 1965).

Lelord, Laffont, Jusseaume, and Stephant (1973) reported a study involving electroencephalogram (EEG) responses of 11 lower functioning children with autism (mean age of 9) and 11 age-matched typical controls. The paradigm involved both response to tone and conditioned response to light–tone pairings. The children with autism demonstrated a reduced response to the tones alone (hypo-responsiveness). However, the conditioned EEG responses of the group with autism showed a different pattern than the typical group, which may indicate a difference in information processing rather than a difference in response to sensory input.

Rosenblum et al. (1980) examined auditory brainstem evoked responses (ABER) to tones in six children with autism and six typical controls, matched pairwise on sex and chronological age. Subjects with autism demonstrated a significantly longer total transmission time from wave 1 to wave 4, greater intra-subject variability of waveforms, and longer latencies in waves 3 and 4 in response to the presentation of tones. The findings are indicative of neurological immaturity, and the authors suggest that there is neurological abnormality in brainstem transmission of auditory input.

Dawson, Finley, Phillips, and Galpert (1986) examined brain hemisphere responses using cortical evoked responses from EEG recordings to three different kinds of sounds, a click used as the constant stimulus, a musical chord, and a spoken syllable 'da' for 17 verbal children with autism, mean age 13 years, and 17 age-matched typically developing controls. Children with autism demonstrated a different pattern of hemisphere specialization to the speech stimulus, with more right hemisphere dominance, but not reduced responses. No data on their response to the musical stimulus were reported. The authors do not discuss this finding as indicative of a general auditory sensory difference in the group with autism, but rather as an information processing difference of speech. They suggest that this difference may result from decreased language input due to the overall effects of autism on the child's social experience.

Pritchard, Raz, and August (1987) examined event-related potentials to visual stimuli in five

[1] A condition involving dysfunction in the regulation of responses to sensory input. Children with this disorder in the Miller et al. (2001) paper did not have any other clinical diagnosis. This group is controversial.

[2] It is very hard to interpret this group by today's diagnostic methods; they are probably best thought of as PDDNOS-type children or Wing's active but odd subgroup (1988).

males with autism compared to five typically developing controls. Subjects viewed flashes of light of three different intensities and were instructed to count the occurrence of the least frequent flash (this requirement to respond to the least frequent stimulus is called an oddball paradigm). The main difference involved the negative amplitude wave that occurs 100 milliseconds after the stimulus – the N100 wave – thought to index initial sensory processing of the stimuli. The N100 wave increased in amplitude as visual flash intensity increased for the group with autism only. The authors suggest that this pattern, called augmentation, indicates a failure of the sensory system to 'gate out' increases in stimulus intensity, which may lead to 'a degree of overload from stimulation in the visual modality.' The P300 wave (the positive amplitude wave that occurs 300 seconds post stimulus and is thought to index the cognitive component of the task) did not differ between groups.

Ceponiene et al. (2003) examined event-related brain potentials (ERPs) to auditory stimuli in nine high-functioning children with autism (mean age 9 years) and ten typically developing controls. Groups were not matched on intelligence or language level, but were of equivalent chronological age. Simple tones, complex tones, and vowels were presented in separate sequences, with an infrequent 'deviant' sound occasionally placed among the repetitive 'standard' sounds (i.e., an oddball paradigm). ERP recording allows the examination of both initial sensory processing of stimuli and later attentional orienting to stimuli. Sound processing was intact across all classes of stimuli in the children with autism, as measured by no differences from the control group in the P100, N200, and N400 components of the ERP and significant mismatch negativity, but there was significantly less orienting (a later, attentional process) to the deviant stimulus within the vowel paradigm. The authors interpreted this finding as indicative of normal auditory processing of stimuli, but abnormal attention to speech-like sounds at a later stage of information processing. Another recent study using an oddball paradigm (Tecchio et al., 2003) did not find intact auditory processing in lower-functioning children with autism, however. Using magnetoencephalography (MEG), this study did not measure a significant mismatch field (the MEG equivalent of mismatch negativity, which reflects the detection of a change in the physical characteristics of a repetitive sound). The authors suggest that this indicated an abnormality in the initial processing of auditory input in children with autism who also have a significant degree of mental retardation.

Finally, a recent study examined an even earlier aspect of sensory filtering, the gating of the auditory evoked potential (indexed by the P50 wave). No differences in P50 gating in response to auditory stimuli were found compared to healthy control children, suggesting normal activation of inhibitory pathways that act as initial filtering mechanisms for auditory stimuli (Kemner, Oranje, Verbaten, & van Engeland, 2002).

To summarize the studies that have compared children with autism to typically developing children, the empirical evidence of sensory hypo- or hyper-arousal is mixed. Only one study, which examined response to visual stimuli, found results suggestive of hyper-arousal and lack of habituation (Pritchard et al., 1987), but others have not (Bernal & Miller, 1971; Lelord et al., 1973). Most of the investigations of response to auditory stimuli found evidence of hypo-responsiveness (Bernal & Miller, 1971; Lelord et al., 1973; Metz, 1967; Tecchio et al., 2003) or no group differences (Ceponiene et al., 2003; Kemner et al., 2002). The range of experimental methods used across these studies (event-related potentials, EEG, EDR, etc.) likely contributes to the variability of findings.

Comparison to clinical controls. DiLavore, Lord, and Rutter (1995) used the Pre-Linguistic Autism Diagnostic Observation Schedule (PL-ADOS), a semi-standard play-based interaction assessment with strong psychometric properties, to study 21 non-verbal children with autism (mean age 49 months), as well as two groups of children with other developmental delays (21 three-year-olds and 21 two-year-olds), matched on IQ. On the PL-ADOS, the frequency and intensity of all unusual sensory behaviors across all modalities are captured by one rating. Children with autism were rated as having a significantly higher level of unusual responses to sensory stimuli than both clinical comparison groups.

James and Barry (1980) examined 20 subjects with autism matched pairwise on sex, age, and IQ with 20 subjects with mental retardation, and 20 subjects with typical development. The researchers measured pulse and respiration in response to visual stimuli consisting of white circles on a black background. At baseline (pre-stimulus), subjects with autism did not differ from either control group; thus, there was no general heightened arousal demonstrated by the subjects with autism. However, after repeated exposure to the stimuli, subjects with autism demonstrated higher pulse than controls and failed to show habituation of respiratory response. The authors conclude that there is an abnormality in sensory habituating mechanisms in autism that leads to a state of constant bombardment from environmental stimuli.

Frankel, Freeman, Ritvo, Chikami, and Carr (1976), trying to understand the role of visual stereotypies in autism, developed a task that simulated the visual effects of rapidly changing light levels created by hand and finger stereotypies carried out close to the eyes. The researchers studied behavioral responses to flickering light in 6 children with aut-

ism and 5 children with mental retardation, ages 3–10, roughly equivalent in IQ. The stimuli included light that flickered at various frequencies, which the children could maintain by pressing a lever. The independent variable was rate of bar pressing. The overall rate of bar presses did not vary by group, but the children with autism demonstrated a significantly higher rate of lever pressing than controls at the higher frequencies of flashes, which may be consistent with hypo-arousal and seeking of sensory stimulation. Contrary to prediction, the rate of motor stereotypies did not differ across groups in either low stimulation situations (sitting in a bare room for 20 minutes) or high stimulation situations (maximal flickering light), failing to support either hypothesized relationship between arousal and stereotypies.

Stevens and Gruzelier (1984) reported a study of electrodermal response (EDR) to auditory stimuli, comparing 20 subjects with autism to two groups: a group with mental retardation and a group with typical development, matched by age, sex, and handedness. IQ differences between the group with autism and the group with intellectual impairments were not significant. Two sets of tones were used, the less frequent tone being louder and longer than the other. In terms of habituation, there were no differences among groups in the number of orienting responses or in habituation patterns, thus demonstrating neither hypo- nor hyper-arousal in subjects with autism. There were no group differences in response amplitude, though both clinical groups showed a marginally significant tendency toward slower decrease in amplitudes than the typical group. The group with autism resembled the typical group in level of skin conductance responses, with the group with retardation showing lower responses. Finally, when subjects were matched by developmental level (MA), there were no differences in any of the temporal variables: response latency, rise, or recovery times.

Van Engeland (1984) also examined electrodermal response to auditory stimuli in 35 subjects with autism compared to 38 child psychiatric patients, 45 typical controls, and 20 adolescents with Down syndrome. Groups were not matched by age or IQ. The stimulus was a loud (85 dB) tone, repeated 24 times over random intervals ranging from 10 to 35 seconds. There were no group differences in spontaneous fluctuations in EDR, indicating no evidence of over-arousal in the subjects with autism. There were also no group differences in habituation rate. There were significantly more non-responders to the first trial in the group with autism than the other three groups. For that reason, they split each of the clinical groups into 'low' and 'high' arousal levels, based on initial level of pre-stimulus spontaneous fluctuations in EDR, and compared the low and high arousal groups across diagnoses. There were twice as many 'high' as 'low' arousal subjects in each group except Down syndrome, in which there were twice as many 'low' as 'high' responders. As might be expected, within each diagnosis, there were significant differences in EDR between the low and the high subgroups. Across diagnostic groups, there were still more non-responders (to the first stimulus) in the 'low' arousal group with autism than the other 'low' groups. There were no differences in responsiveness to the stimulus or habituation rate when the 'high' arousal subgroups were compared across diagnoses, though the 'high' group with autism showed faster EDR recovery from the stimulus than other groups. These data were interpreted as demonstrating no evidence of either general or specific hyper- or hypo-responsiveness in autism.

Niwa, Ohta, and Yamazaki (1983) examined event-related potentials to auditory stimuli, focusing on the P300 component, which is thought to reflect processing or evaluation of stimuli. They reported smaller P300 amplitude when four participants with autism passively listened to an auditory oddball paradigm, relative to four participants with Down syndrome and five with typical development. The magnitude of the difference in P300 amplitude relative to the participants with Down syndrome was quite large ($d = 2.13$), but failed to reach statistical significance due to the very small sample sizes. The authors interpreted the smaller P300 amplitude in autism as indicating difficulties in processing auditory stimuli.

Lincoln, Courchesne, Harms, and Allen (1995) examined earlier components of the ERP, the N100 and P200 components, which are thought to index initial reception of sensory input, in 10 high-functioning children with idiopathic autism, 10 children with language disorders, and 10 children with typical development. Stimuli were auditory tones that varied in pitch and volume. There were no group differences on EEG measures of the amplitude of the N100 and P200 responses or in response latencies. However, both clinical groups demonstrated significantly less increase in N100 amplitude as volume increased than the typical group. In an oddball paradigm in which the subjects had to discriminate between two different pitches by pressing a button, there were no group differences in reaction times or accuracy. Children with autism did not differ from the typical controls on N100 or P200 response amplitude or latency in this paradigm either, though the children with language disorder demonstrated an abnormal N100 response. Thus, only one autism-related difference emerged from this study, and it involved an under-response to increasing sound intensity. The group with receptive language disorder demonstrated much more abnormality than the group with autism. Lincoln et al. (1995) summarize their findings in the following way: '[There is] strong evidence that nonretarded individuals with autism … demonstrate normal auditory brainstem evoked responses … [that] suggest that the initial, primarily subcortical, neural generators of the ABER are intact' (p. 522).

The authors go on to say that midlatency auditory evoked responses (ERPs present between 10 and 80 msec) have also been found to be normal in autism, suggesting that the thalamic and auditory cortex areas subserving early and midlatency responses are functioning normally. Abnormalities associated with autism have occurred in studies examining later auditory ERPs, those in the P300 range (e.g., Niwa et al., 1983), which indicate difficulties with the **processing** of the auditory stimuli, rather than the **sensory reception** of those stimuli.

Miller and colleagues (2001) examined electrodermal responses to 10 repeated trials of carefully controlled sensory stimuli in five sensory modalities: auditory, tactile, olfactory, visual, and vestibular. Subjects included 8 higher-functioning children with autism (mean age of 8 years), 23 children with fragile X syndrome, 32 with sensory modulation disorder, 40 with attention deficit disorder, and 46 with typical development, all matched on chronological age. Subjects with autism demonstrated under-responsiveness to the initial stimuli and more rapid habituation than all other groups in all sensory domains. There was no evidence of general over-arousal or sensory hypersensitivity; in contrast, participants with autism were consistently hyposensitive in all sensory modalities, even relative to typical controls. This contrasted considerably with parental reports on the sensory questionnaire also administered in the study, in which elevations were demonstrated relative to the typical group in taste and tactile sensitivity (but not auditory, movement, visual, or general responsivity). Across all measures and modalities, the group with fragile X was more reactive than the group with autism.

Few studies have examined response to vestibular stimulation. One of the first was carried out by Ornitz and colleagues. Ornitz, Brown, Mason, and Putnam (1974) examined eye movements following rotation in 21 children with autism, ages 3 to 5 years, compared to a group of 25 age-matched typical controls. Duration of nystagmus and number of nystagmus beats were the dependent variables. In conditions in which the rotation (in the dark) was followed by environmental light, children with autism demonstrated shorter duration of nystagmus and fewer beats. This finding indicated a reduced motor response compared to normal controls, and it raises the question of impaired vestibular system functioning. These findings were replicated with a second set of subjects in a later study by the same research team (Ornitz, Atwell, Kaplan, & Westlake, 1985). More recently, Miller et al. (2001) also found evidence of hypo-responsivity to vestibular stimulation. They examined initial response and habituation to a vestibular stimulus (being tipped backwards in a chair repeatedly) using electrodermal and heart rate changes in children with autism compared to three different groups: children with fragile X syndrome, sensory modulation disorder, and typical development. Similar to their findings from the other sensory modalities, Miller et al. (2001) reported under-responsivity and rapid habituation of the group with autism to vestibular stimuli, as compared to all comparison groups. In contrast, Goldberg, Landa, Lasker, Cooper, and Zee (2000) failed to find group differences in vestibulo-ocular reflexes. Participants were 13 children with high-functioning autism and 10 typical controls, carefully diagnosed and matched. They were rotated in a chair in darkness and nystagmus during and after rotation was measured for 30 seconds with the head in two different positions. There were no group differences in response to the stimulus in either head position, failing to replicate earlier findings. There were also no relationships between parental reports of visual-vestibular symptoms in the children and their response to this task.

Summary. Unusual visual behaviors, such as close inspection of objects and preference for flickering lights, have been described in autism. However, the hypothesized physiological mechanisms underlying these visual behaviors – general over-arousal, over-reactivity to visual stimuli, difficulty with physiological habituation – are not strongly supported by experimental studies. Only three studies have examined responses to visual stimuli, with one finding heightened physiological responsiveness and reduced habituation (James & Barry, 1980), another finding essentially the opposite pattern, hypo-responsiveness and more rapid habituation (Miller et al., 2001), and the third also consistent with hypo-arousal (Frankel et al., 1976). Findings from the auditory studies are more consistently negative across a wide range of indices (EDR, pulse, respiration rate, ERP), with three investigations failing to find any group differences in response to auditory stimuli (Lincoln et al., 1995; Stevens & Gruzelier, 1984; van Engeland, 1984) and two finding hypo-responsiveness in the group with autism (Miller et al., 2001; Niwa et al., 1983). There is little evidence of general baseline over-arousal in subjects with autism, nor are there consistent abnormalities in habituation or with the initial orienting response. Similarly, studies of reaction to vestibular stimuli indicate no group differences (Goldberg et al., 2000) or hypo-responsivity (Miller et al., 2001; Ornitz et al., 1974, 1985). Across all sensory domains, these studies collectively provide far more evidence of physiological hypo- than hyper-responsiveness in children with autism.

Perceptual inconstancy theory

There have been no empirical studies examining support for this theory. Since this theory postulates fluctuating states of over- and under-arousal in autism, rather than simply one or the other, the general lack of empirical support for over-arousal is damaging to perceptual inconstancy models as well.

Crossmodal processing impairment theory

While crossmodal perception involves a different level of information processing than the simple responses to sensory stimuli reported above, it is included here because it represents one theory explaining sensory dysfunction in autism. A study of simple sensory synchronies across modalities was reported by Walker-Andrews, Haviland, Huffman, and Toci (1994). Borrowing a paradigm from infant perception studies, 23 children with autism were shown two videotapes of toys being operated, with the corresponding soundtrack from only one tape playing. Fifty-eight percent of the group with autism demonstrated preferential looking to the video image that was congruent with the sound, at a level seen in typically developing infants. The response showed no relationship with IQ or language development. Thus, the subjects demonstrated the ability to transfer sensory information from the visual to the auditory domain above chance level, but the lack of control subjects in this experiment makes the results difficult to interpret.

Hobson, Ouston, and Lee (1988) reported a study of crossmodal matching of visual and auditory stimuli involving 21 adolescents and young adults with autism individually matched by CA and verbal IQ to 21 persons with mental retardation. Stimuli involved listening to audiotapes of six sounds, after each of which an array of six photos was presented to the subject, who was to select the photo that matched the sounds. Stimuli included sounds and pictures of vehicles, birds, household appliances, gardening tools, water, and walking (a similar set of emotion-matching tasks was used, which will not be reported here). There were no significant differences in performance of the two groups on this task, with the group with autism achieving better scores than controls. This study was replicated by Ozonoff, Pennington, and Rogers (1990), using 14 children with autism (mean age of 6 years) and 14 typical children matched on language level. The children with autism demonstrated equivalent performance to the matched controls on these nonsocial crossmodal matching tasks involving sights and their accompanying sounds.

Thus, in the three studies that have examined crossmodal transfer, no differences were found between the performances of participants with autism and contrasting groups. However, given how little comparative, empirical work has been done in this area, there is certainly need for continued exploration.

Methodological issues

Before proceeding to a conclusion, it is important to review some of the methodological issues that these studies as a group present.

Subject selection. The first issue involves subject selection. Diagnosis of autism has evolved considerably over the past 30 years. While current research practices involve corroborated diagnoses, use of objective scales (Filipek et al., 1999), and exclusion of subjects with known medical conditions like Rett syndrome, fragile X syndrome, and seizures, these practices were not common in the research practices of 20 or more years ago. Early studies often drew heavily on institutionalized groups and did not always report IQ levels. The less well characterized the subject group, the less clearly generalizable are the results.

Choice of comparison groups. The second issue involves choice of comparison groups. It is currently widely accepted that autism is a developmental disorder, with symptoms and profiles changing with development. Asking whether a characteristic is specific to autism requires that the researchers demonstrate that the characteristic is not due either to general developmental delays or to general neurological impairment, accomplished by matching a group with autism to a group with the equivalent level of age and IQ scores. The necessity of this is underscored by the results of Stevens and Gruzelier (1984), who reported a significant developmental lag in physiological response to auditory stimuli. Relatively few studies carried out before 1985 or so followed these practices.

Changes in design over time. The third methodological issue in trying to pool information across 30 years of sensory studies in autism involves research design. These studies vary considerably in their demonstration of the reliability and validity of the measures used, consideration of effect sizes, and power estimates. The field has changed significantly, and it is difficult to know how heavily to weigh some of the earlier studies in light of today's experimental tools and practices, particularly those with very small sample sizes. Furthermore, in many studies the subject groups are too small to allow for conclusions to be made about the population or even the majority of children with autism.

Construct validity and reliability. A fourth and final methodological issue has to do with the nature of the constructs that we are considering. The constructs of sensory and repetitive behaviors have not been well defined, nor has their relationship to each other been examined carefully (Militerni, Bravaccio, Falco, Fico, & Palermo, 2002). Assumptions are made that stereotyped motor behaviors have sensory origins or serve a homeostatic function that compensates for either sensory overload (Hutt et al., 1965; Kinsbourne, 1980) or sensory under-arousal (hence the term 'self-stimulatory behavior'; Lovaas, Newsom, & Hickman, 1987). At present, however, few studies have examined such relationships, with conflicting

results. A significant decrease in heart rate with onset of motor stereotypies associated with distress has been reported (Willemsen-Swinkels, Buitelaar, Dekker, & van Engeland, 1998), supporting a homeostatic function of the repetitive behaviors. However, Berkson, Baranek, and colleagues found few significant correlations between sensory sensitivities/symptoms and motor stereotypies (Baranek, Foster, & Berkson, 1997a; Berkson, Gutermuth, & Baranek, 1995). A recent factor analysis of the Autism Diagnostic Interview – Revised found that sensory behaviors fell on one factor, while other repetitive behaviors, such as motor stereotypies and compulsive rituals, fell on different factors and intercorrelation among the constructs was low (Tadevosyan-Leyfer et al., 2003). Even within the sensory sensitivities construct, correlations between auditory and tactile sensitivities have been found to be low (Baranek, Foster, & Berkson, 1997b). As to the reliability of the constructs, one study found relatively poor inter-rater agreement on the presence of stereotypies and abnormal sensory responses, with kappa values ranging from .14 to .38 (Berkson et al., 1995). Two studies have found that data gathered via observation showed no agreement with data gathered via caretaker report on the same sensory modality (Goldberg et al., 2000; Miller et al., 2001). Thus, lumping together sensory-related behaviors across sensory modalities and repetitive behaviors as if they were a unitary construct has no support in the literature.

Summary: the state of the science concerning sensory dysfunction in autism

From the empirical work reviewed here, it appears that both general developmental effects and the presence of developmental disorders are related to differences in sensory responses, both behavioral and physiological. At the behavioral level, parents of children with autism, mental retardation, fragile X syndrome, sensory impairments, and sensory modulation disorder all report higher levels of abnormal sensory responses than parents of typically developing children. The differences are clearest when chronologically age-matched groups are compared, but some (fewer) differences are also present in the few studies that have used developmentally matched groups. Increased levels of sensory-related behaviors have been found in children with autism in some laboratory studies, but there are few differences from children with developmental delays. Thus, abnormal sensory response does not appear specific to autism. Similar levels of sensory-related behaviors are reported by parents of children with fragile X syndrome, abnormal sensory responses dysfunction, and deaf-blindness, and these three groups appear to have the most severe symptoms.

When we move to the question of why unusual responses to sensory stimuli are found in autism, we again have few answers. The major theories that seek to explain the behaviors suggest abnormalities in general arousal levels, in arousal levels in response to stimuli, and in habituation to stimuli. However, multiple studies have failed to document physiological indices of general over-arousal, specific over-arousal to stimuli, or problems with habituation to the stimuli. Several of the studies reviewed above specifically reported either no differences in resting physiological measures of arousal (EDR) or lower than normal responses. There was no support in the above controlled studies for a general heightened arousal in autism. In fact, studies have more commonly reported physiological under-responsiveness to stimuli. Examination of habituation patterns has most commonly revealed either no abnormality in habituation response or more rapid habituation to stimuli. Thus, theories of sensory defensiveness in autism, which suggest that the child is ignoring or rejecting incoming sensory stimuli as a way of avoiding over-arousal, are simply not supported by this body of work.

The findings reviewed here also have implications for our understanding of motor stereotypies in autism. One fairly common view of the function of stereotypic and repetitive behaviors suggests that they help to reduce abnormally high arousal levels (Hutt et al., 1964, 1965; Kinsbourne, 1980). However, two studies cited above demonstrated that stereotypies did not increase as level of sensory stimulation was increased (Bernal & Miller, 1971; Frankel et al., 1976). A competing view is that stereotypies increase arousal levels for children who are chronically under-aroused (DesLauriers & Carlson, 1969). The one study that examined stereotypies in a situation of under-stimulation (Frankel et al., 1976) found no support for this hypothesis either. Thus, the hypothesis that motor stereotypy specific to autism functions to regulate levels of stimulation and abnormalities in arousal levels appears to be without empirical support at this time.

How are the above findings to be interpreted? Given the methodological limitations in the majority of the investigations and the lack of replication across studies, it may well be too early to interpret the literature. One set of theories that has some support from our review of the empirical literature are the under-arousal theories provided early on by DesLauriers and Carlson (1969) and Rimland (1964). While general arousal levels have been found to be normal in children with autism, under-arousal in response to sensory stimuli was reported in a number of the studies reviewed above.

Other possible explanations for unusual sensory behaviors also come to mind. Lack of typical levels of responsiveness to sensory input could be part of a more general affective theory like Hobson's (and DesLauriers before him), in which there is a lack of response capacity to normal levels of stimuli, both from people and from environmental events – the

stimulus barrier theory. Another possibility is a kind of deprivation theory (Dawson et al., 2004; Loveland, 2001; Mundy & Markus, 1997; O'Connor et al., 2000), in which early input deficiencies have cascading effects on other domains of development over time. In developing children with unimpaired social capacity, the social world fills much of their time and provides much of their sensory input. For children with autism, relatively uninvolved with the social world and yet with considerable need for ongoing stimulation for their developing nervous systems, the world of objects and physical attributes becomes their main focus, and they may learn to use simple sensory and repetitive behaviors available to all young children to create additional sensory input for themselves. In this way of thinking, the sensory symptoms are secondary, rather than primary, to autism and not specific to it, since the same explanation could be (and has been) made for the presence of stereotypies and unusual response to sensory stimuli in blind children (Fraiberg, 1977), individuals with severe and profound retardation (Bodfish, Symons, Parker, & Lewis, 2000), and children with significant deprivation (O'Connor et al., 2000).

It is clear that a deeper understanding of the atypical sensory-related behaviors in autism awaits further empirical investigation. This is particularly important given the widespread recommendation of sensory integration services to treat 'sensory symptoms' for children with autism (Dawson & Watling, 2000; Goldstein, 2000; Watling et al., 1999). Sensory integration therapy is a frequently recommended intervention for children with autism, even though the empirical evidence of its efficacy is minimal (Baranek, 2002; Dawson & Watling, 2000). However, the theory underlying the empirical investigations is not the same theory that underlies the clinical sensory integration work. Answering questions about sensory-based treatments requires that this gap be closed (Stackhouse, personal communication). Auditory integration training (AIT) is another popular treatment based on a theory of sensory dysfunction. Given the evidence reviewed here, support for the theoretical rationales underlying these treatments is questionable. Finally, one study of sensory integration treatment for autism has reported that children who are hypo-responsive to sensory input respond less well to sensory integration therapy than children with hyper-arousal (Ayres & Tickle, 1980). Given the evidence of the pattern of sensory hypo-responsivity of children with autism, as a group, the efficacy of sensory integration therapy is in sore need of further study. Both AIT and sensory integration treatment have been reported by many families as helpful for their children, but it is not clear what aspects of the treatment are responsible. The quality of interaction with a skilled therapist, the organization of child attention around specific tasks, the consistency of certain activities and reinforcing consequences of certain behaviors may all have a beneficial effect on children's behavior, independent of any particular sensory experience. As with most treatments for autism, these treatments are in need of additional, well designed treatment trials in order to 1) test and validate their efficacy in autism and 2) determine what aspects of the treatment are responsible for positive outcomes. The sensory elements of these treatments may or may not be the primary 'active ingredients' responsible for their effects.

In closing, we echo DeMyer, writing in 1979, 'we need a research program in which all sensory modalities are studied as directly as possible, such as exploring cerebral evoked responses and connections between language [and] thought process and auditory perception…' Studies are needed that:

1. Provide careful diagnosis and detailed demographics of each of the subject groups.
2. Use clinical control groups matched on both age and IQ, as well as typically developing mental age-matched controls. As has been argued repeatedly since James and Barry (1981) first addressed the issue, use of control subjects who are matched on both age and IQ is crucial for controlling the effect of general maturational delays. Of similar importance is the use of clinical comparison groups, who to some extent control for the possible effects of compromised neurological functioning.
3. Explore developmental issues by keeping CA and MA age ranges narrow and replicating studies at various developmental points. It seems especially important to construct subject groups involving persons with lower IQ and higher IQ separately. Longitudinal studies of sensory responses are nonexistent and will be crucial for teaching us about the developmental course of sensory responses in autism and other developmental disabilities.
4. Examine both behavioral responses, using measures with strong psychometric properties, and physiological responses in the same study. The discrepancies between parent report of hyper-responsivity to stimuli and physiological findings of hypo-responsiveness are intriguing.
5. Move away from parent questionnaires and into controlled laboratory studies. The questions that are now being asked require a greater degree of experimental control than can be gained from respondent report.
6. Include at least two sensory modalities (and hopefully more) in studies, so we can have better data on the pervasiveness of abnormal responses.
7. Questions regarding overlap between sensory responses and repetitive motor behaviors were raised above, but in reality construct validity has not been established in any of the sensory domains. The Sensory Profile (Dunn, 1999) has

provided a tool with better psychometric properties. Such work needs to continue and broaden.

Sensory hypotheses of autism have been discussed almost since the condition was first described. However, careful empirical work in other areas, like cognition and communication, has provided far more definitive information about the nature of autism in those domains than in the sensory area. What empirical information is available is not particularly supportive of many current theories concerning the specificity of sensory dysfunction to autism. There is no consistent psychophysiological explanation (e.g., hypo- or hyper-arousal, deficits in habituation) for the sensory symptoms of autism. There is a great deal about the unusual sensory responses of autism that we do not know. The field needs to apply the same kind of rigor and programmatic research in this area as it has in so many other aspects of autism.

Acknowledgements

The authors wish to acknowledge the contributions of Tracy Stackhouse, OTR, to this paper, and the support of Debra Galik in manuscript preparation. Drs. Rogers and Ozonoff were supported by grant# U19 HD35468-7 from the National Institute of Child Health and Human Development, one of the NICHD/NIDCD Collaborative Programs of Excellence in Autism, as well as by NIMH R01 MH068398. In addition, Dr. Rogers was supported by NIDCD R21 DC 05574-3 and NIMH R21 MH 067363.

Correspondence to

Sally J. Rogers, M.I.N.D. Institute, 2825 50th St., University of California – Davis Medical Center, Sacramento CA 95817, USA; Email: sjrogers@ucdavis.edu

References

Ayres, A.J., & Tickle, L.S. (1980). Hyper-responsivity to touch and vestibular stimuli as a predictor of positive response to sensory integration procedures by autistic children. *American Journal of Occupational Therapy*, *34*, 375–381.

Bailey, A., Phillips, W., & Rutter, M. (1996). Autism: Towards an integration of clinical, genetic, neuropsychological, and neurobiological perspectives. *Journal of Child Psychology and Psychiatry*, *37*, 89–126.

Baranek, G.T. (2002). Efficacy of sensory and motor interventions for children with autism. *Journal of Autism and Developmental Disorders*, *32*, 397–422.

Baranek, G.T., Foster, L.G., & Berkson, G. (1997a). Sensory defensiveness in persons with developmental disabilities. *Occupational Therapy Journal of Research*, *17*, 1–14.

Baranek, G.T., Foster, L.G., & Berkson, G. (1997b). Tactile defensiveness and stereotyped behaviors. *American Journal of Occupational Therapy*, *51*, 91–95.

Bergman, P., & Escalona, S.K. (1949). Unusual sensitivities in very young children. *Psychoanalytic Study of the Child*, *3/4*, 333–352.

Berkson, G., Gutermuth, L., & Baranek, G.T. (1995). Relative prevalence and relations among stereotyped and similar behaviors. *American Journal on Mental Retardation*, *100*, 137–145.

Bernal, M.E., & Miller, W.H. (1971). Electrodermal and cardiac responses of schizophrenic children to sensory stimuli. *Psychophysiology*, *7*, 155–168.

Bodfish, J.W., Symons, F.J., Parker, D.E., & Lewis, M.H. (2000). Varieties of repetitive behavior in autism: Comparisons to mental retardation. *Journal of Autism and Developmental Disorders*, *30*, 237–243.

Brock, J., Brown, C.C., Boucher, J., & Rippon, G. (2002). The temporal binding deficit hypothesis of autism. *Development and Psychopathology*, *14*, 209–224.

Ceponiene, R., Lepisto, T., Shestakova, A., Vanhala, R., Alku, P., Naatanen, R., & Yaguchi, K. (2003). Speech-sound-selective auditory impairment in children with autism: They can perceive but do not attend. *Proceedings of the National Academy of Sciences*, *100*, 5567–5572.

Chugani, D.C., Muzik, O., Behen, M., Rothermal, R., Janisse, J.J., Lee, J., & Chugani, H.T. (1999). Developmental changes in brain serotonin synthesis capacity in autistic and nonautistic children. *Annals of Neurology*, *45*, 287–295.

Chugani, D.C., Muzik, O., Rothermal, R., Behen, M., Chabraborty, P., Mangner, T., daSilva, E.A., & Chugani, H.T. (1997). Altered serotonin synthesis in the dentato-thalamocortical pathway in autistic boys. *Journal of Neurology*, *42*, 666–668.

Cook, D.J., Mulrow, C.D., & Haynes, R.B. (1997). Systematic reviews: Synthesis of best evidence for clinical decisions. *Annals of Internal Medicine*, *126*, 376–380.

Dawson, G., Finley, C., Phillips, S., & Galpert, L. (1986). Hemispheric specialization and the language abilities of autistic children. *Child Development*, *57*, 1440–1453.

Dawson, G., & Lewy, A. (1989). Arousal, attention, and the socioemotional impairments of individuals with autism. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 49–74). New York: Guilford.

Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, *40*, 271–83.

Dawson, G., & Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism and Developmental Disorders*, *30*, 415–421.

DesLauriers, A.M., & Carlson, C.F. (1969). *Your child is asleep: Early infantile autism*. Homewood, IL: Dorsey Press.

DeMyer, M.K. (1979). *Parents and children in autism*. New York: John Wiley & Sons.

DiLavore, P.C., Lord, C., & Rutter, M. (1995). The Prelinguistic Autism Diagnostic Observation Schedule (PLADOS). *Journal of Autism and Developmental Disorders*, *25*, 355–379.

Dunn, W. (1999). *Manual for the sensory profile*. Austin TX: Psychological Corporation.

Filipek, P.A., Accardo, P.J., Baranek, G.T., Cook, E.H., Jr, Dawson, G., Gordon, B., Gravel, J.S., Johnson, C.P., Kallen, R.J., Levy, S.E., Minshew, N.J., Ozonoff, S., Prizant, B.M., Rapin, I., Rogers, S., Stone, W.L., Teplin, S., Tuchman, R.F., & Volkmar, F.R. (1999). Screening and diagnosis of autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *29*, 439–484.

Fraiberg, S. (1977). *Insights from the blind: Comparative studies of blind and sighted infants*. New York: New American Library.

Frankel, F., Freeman, B.J., Ritvo, E., Chikami, B., & Carr, E. (1976). Effects of frequency of photic stimulation upon autistic and retarded children. *American Journal of Mental Deficiency*, *81*, 32–40.

Ginn, P., Berry, P., & Andrews, R.J. (1981). The temperament of Down's Syndrome infants. *Journal of Child Psychology and Psychiatry*, *28*, 189–194.

Goldberg, M.C., Landa, R., Lasker, A., Cooper, L., & Zee, D.S. (2000). Evidence of normal cerebellar control of the vestibulo-ocular reflex in children with high-functioning autism. *Journal of Autism and Developmental Disorders*, *30*, 519–524.

Goldstein, H. (2000). Commentary: Interventions to facilitate auditory, visual, and motor integration in autism: 'Show me the data'. *Journal of Autism and Developmental Disorders*, *30*, 423–425.

Grandin, T. (1992). An inside view of autism. In E. Schopler & G.B. Mesibov (Eds.), *High-functioning individuals with autism* (pp. 105–126). New York: Plenum.

Greenspan, S.I., Kalmanson, B., Shahmoon-Shanok, R., Wieder, S., Gordon-Williamson, G., & Anzalone, M. (1997). *Assessing and treating infants and young children with severe difficulties in relating and communicating*. Baltimore: Zero to Three.

Hobson, R.P., Ouston, J., & Lee, A. (1988). Emotion recognition in autism: Coordinating faces and voices. *Psychological Medicine*, *18*, 911–923.

Hutt, C., Hutt, S.J., Lee, D., & Ounsted, C. (1964). Arousal and childhood autism. *Nature*, *204*, 908–909.

Hutt, C., Hutt, S.J., Lee, D., & Ounsted, C. (1965). A behavioural and electroencephalographic study of autistic children. *Journal of Psychiatric Research*, *3*, 181–197.

James, A.L., & Barry, R.J. (1980). Respiratory and vascular responses to simple visual stimuli in autistics, retardates, and normals. *Psychophysiology*, *17*, 541–547.

James, A.L., & Barry, R.J. (1981). General maturational lag as an essential correlate of early onset psychosis. *Journal of Autism and Developmental Disorder*, *11*, 271–283.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

Kemner, C., Oranje, B., Verbaten, M.N., & van Engeland, H. (2002). Normal P50 gating in children with autism. *Journal of Clinical Psychiatry*, *63*, 214–217.

Kemper, T.L., & Bauman, M.L. (1998). Neuropathology of infantile autism. *Journal of Neuropathology and Experimental Neurology*, *57*, 645–652.

Kientz, M.A., & Dunn, W. (1997). A comparison of the performance of children with and without autism on the sensory profile. *American Journal of Occupational Therapy*, *51*, 530–537.

Kinsbourne, M. (1980). Do repetitive movement patterns in children and animals serve a dearousing function? *Developmental and Behavioral Pediatrics*, *1*, 39–42.

Kinsbourne, M. (1987). Cerebral-brainstem relations in infantile autism. In E. Schopler & G.B. Mesibov (Eds.) *Neurobiological issues in autism* (pp. 107–125). New York: Plenum.

Kinsbourne, M. (1991). Overfocusing: An apparent subtype of attention deficit hyperactivity disorder. In N. Amir, I. Rapin, & D. Branski (Eds.), *Pediatric neurology: Behavior and cognition of the child with brain dysfunction* (pp. 18–35). Basel, Switzerland: Karger.

Lelord, G., Laffont, F., Jusseaume, P., & Stephant, J.L. (1973). Comparative study of conditioning of averaged evoked responses by comparing sound and light in normal and autistic children. *Psychophysiology*, *10*, 415–425.

Lincoln, A.J., Courchesne, E., Harms, L., & Allen, M. (1995). Sensory modulation of auditory stimuli in children with autism and receptive developmental language disorder: Event-related brain potential evidence. *Journal of Autism and Developmental Disorders*, *25*, 521–539.

Lopez, B.R., Lincoln, A.J., Ozonoff, S., & Lai, Z. (in press). Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders*.

Lord, C. (1995). Follow-up of two year olds referred for possible autism. *Journal of Child Psychology and Psychiatry*, *36*, 1365–1382.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *24*, 659–685.

Lord, C., Storoschuk, S., Rutter, M., & Pickles, A. (1993). Using the ADI-R to diagnose autism in preschool children. *Infant Mental Health Journal*, *14*, 234–252.

Lovaas, O.I., Newsom, C., & Hickman, C. (1987). Self-stimulatory behavior and perceptual reinforcement. *Journal of Applied Behavior Analysis*, *20*, 45–68.

Loveland, K.A. (2001). Toward an ecological theory of autism. In J.A. Burack, T. Charman, N. Yirmiya, & P.R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 17–37). Mahwah NJ: Erlbaum.

Metz, J.R. (1967). Stimulation level preferences of autistic children. *Journal of Abnormal Psychology*, *72*, 529–535.

Militerni, R., Bravaccio, C., Falco, C., Fico, C., & Palermo, M.T. (2002). Repetitive behaviors in autistic disorder. *European Child and Adolescent Psychiatry*, *11*, 210–218.

Miller, L.J., Reisman, J., McIntosh, D.N., & Simon, J. (2001). The ecological model of sensory modulation: Performance of children with Fragile X Syndrome, Autism, ADHD and SMD. In S. Roley, R. Schaaf, & E. Blanche (Eds.) *Sensory integration and developmental disabilities*. San Antonio, TX: Therapy Skill Builders.

Mundy, P., & Markus, J. (1997). On the nature of communication and language impairment in autism. *Mental Retardation and Developmental Disabilities Research Reviews*, *3*, 343–349.

Niwa, S, Ohta, M., & Yamazaki, K. (1983). P300 and stimulus evaluation process in autistic subjects. *Journal of Autism and Developmental Disorders*, *13*, 33–42.

O'Connor, T.G., Rutter, M., Beckett, C., Keaveney, L., & Kreppner, J.M., and the English and Romanian Adoptees Study Team. (2000). The effects of global severe privation on cognitive competence: Extension and longitudinal follow-up. *Child Development*, *71*, 376–390.

O'Neill, M., & Jones, R.S.P. (1997). Sensory-perceptual abnormalities in autism: A case for more research. *Journal of Autism and Developmental Disorders*, *27*, 283–293.

Ornitz, E.M., Atwell, C.W., Kaplan, A.R., & Westlake, J.R. (1985). Brain-stem dysfunction in autism; results of vestibular stimulation. *Archives of General Psychiatry*, *31*, 369–375.

Ornitz, E.M., Brown, M.B., Mason, A., & Putnam, N.H. (1974). Effect of visual input on vestibular nystagmus in autistic children. *Archives of General Psychiatry*, *31*, 369–375.

Ornitz, E.M., Guthrie, P., & Farley, A.H. (1977). The early development of autistic children. *Journal of Autism and Childhood Schizophrenia*, *7*, 207–229.

Ornitz, E.M., & Ritvo, E. (1968). Perceptual inconstancy in early infantile autism. *Archives of General Psychiatry*, *18*, 76–97.

Ozonoff, S., Pennington, B.F., & Rogers, S.J. (1990). Are there specific emotion perception deficits in young autistic children? *Journal of Consulting and Clinical Psychology, 31,* 343–361.

Pritchard, W.S., Raz, N., & August, G.J. (1987). Visual augmenting/reducing and P300 in autistic children. *Journal of Autism and Developmental Disorders*, *17*, 231–242.

Rimland, B. (1964). *Infantile autism*. New York: Appleton-Century-Crofts.

Rogers, S.J., Hepburn, S., & Wehner, E. (2003). Parent reports of sensory symptoms in toddlers with autism and those with other developmental disorders. *Journal of Autism and Developmental Disorders*, *33*, 631–642.

Rosenblum, S.M., Arick, J.R., Krug, D.A., Stubbs, E.G., Young, N.B., & Pelson, R.O. (1980). Auditory brainstem responses in autistic children. *Journal of Autism and Developmental Disorders*, *10*, 215–225.

South, M., Ozonoff, S., & McMahon, W.M. (2005). Repetitive behavior in the high-functioning autism spectrum: Phenotypic characterization and relationship to cognitive functioning. *Journal of Autism and Developmental Disorders*, *35*, 145–158.

Stevens, S., & Gruzelier, J. (1984). Electrodermal activity to auditory stimuli in autistic, retarded and normal children. *Journal of Autism and Developmental Disorders*, *14*, 245–260.

Tadevosyan-Leyfer, O., Dowd, M., Mankoski, R., Winklosky, B., Putnam, S., McGrath, L., Tager-Flusberg, H, & Folstein S.E. (2003). A principal components analysis of the Autism Diagnostic Interview-Revised. *Journal of American Child and Adolescent Psychiatry*, *42*, 864–872.

Tanguay, P.E., & Edwards, R.M. (1982). Electrophysiological studies of autism: The whisper of the bang. *Journal of Autism and Developmental Disorders*, *12*, 177–184.

Tecchio, F., Benassi, F., Zappasodi, F., Gialloreti, L.E., Palermo, M., Seri, S., & Rossini, P.M. (2003). Auditory sensory processing in autism: A magnetoencephalographic study. *Biological Psychiatry*, *54*, 647–654.

Tinbergen, E.A., & Tinbergen, U. (1972). *Early childhood autism: An ethological approach*. Berlin: Paul Parey Publishers.

Turner, M.A. (1999). Generating novel ideas: Fluency performance in high functioning and learning disabled individuals with autism. *Journal of Child Psychology and Psychiatry*, *40*, 189–201.

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*, 261–279.

Walker-Andrews, A.S., Haviland, J.M., Huffman, L., & Toci, L. (1994). Brief report: Preferential looking in intermodal perception by children with autism. *Journal of Autism and Developmental Disorders*, *24*, 99–107.

Waterhouse, L., Fein, D., & Modahl, C. (1996). Neurofunctional mechanisms in autism. *Psychological Review*, *103*, 457–489.

Watling, R., Deitz, J., Kanny, E.M., & McLaughlin, J.F. (1999). Current practice of occupational therapy for children with autism. *American Journal of Occupational Therapy*, *53*, 498–505.

Watling, R.L., Deitz, J., & White, O. (2001). Comparison of Sensory Profile scores in young children with and without autism spectrum disorders. *American Journal of Occupational Therapy*, *55*, 416–423.

Williams, D. (1994). *Somebody somewhere*. New York: Doubleday.

Willemsen-Swinkels, S.H.N., Buitelaar, J.K., Dekker, M., & van Engeland, H. (1998). Subtyping stereotypic behavior in children: The association between stereotypic behavior, mood, and heart rate. *Journal of Autism and Developmental Disorders*, *28*, 547–557.

Wing, L. (1969). The handicaps of autistic children: A comparison study. *Journal of Child Psychology and Child Psychiatry*, *10*, 1–40.

Wing, L. (1988). The continuum of autistic characteristics. In E. Schopler & G.B. Mesibov (Eds.), *Diagnosis and assessment in autism*. New York: Plenum Press.

Zero to Three. (1994). *Diagnostic classification of mental health and developmental disorders of infancy and early childhood*. Arlington VA: National Center for Clinical Infant Programs.

Manuscript accepted 19 October 2004