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Sensory Processing in Autism: A Review of Neurophysiologic Findings

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ABSTRACT: Atypical sensory-based behaviors are a ubiquitous feature of autism spectrum disorders (ASDs). In this article, we review the neural underpinnings of sensory processing in autism by reviewing the literature on neurophysiological responses to auditory, tactile, and visual stimuli in autistic individuals. We review studies of unimodal sensory processing and multisensory integration that use a variety of neuroimaging techniques, including electroencephalography (EEG), magnetoencephalography (MEG), and functional MRI. We then explore the impact of covert and overt attention on sensory processing. With additional characterization, neurophysiologic profiles of sensory processing in ASD may serve as valuable biomarkers for diagnosis and monitoring of therapeutic interventions for autism and reveal potential strategies and target brain regions for therapeutic interventions. **(*Pediatr Res* 69: 48R–54R, 2011)**

Autism spectrum disorders (ASDs) are defined clinically by impairment in communication, social interaction, and behavioral flexibility (1). In this review, ASD is used to include individuals with the full range of symptoms from the most severe form of the condition, autistic disorder or autism, to the milder forms, Asperger syndrome (AS) and pervasive developmental disorder not otherwise specified (PDD, NOS). Even within a diagnosis of autism, there can be a wide range of intellectual ability. ASD is clearly not a one size fits all diagnosis. There are many known etiologies that contribute to an ASD phenotype, including genetic variations (*e.g.* fragile X and tuberous sclerosis), environmental exposures (*e.g. in utero* valproic acid exposure), and prematurity. There also exists considerable phenotypic variation involving the pace of language development, the presence of epilepsy, and the range of cognitive ability. What does appear to be common to individuals across the spectrum are atypical behavioral responses to sensory information. More than 96% of children with ASD report hyper- and hyposensitivities in multiple domains. Similar to the wide range of spectrum severity found for communication and social deficits, sensory behavioral differences also range from mild to severe, and these behavioral differences can endure through adulthood (2–6).

Sensory processing concerns have been a key feature of ASD clinical descriptions from the original independent seminal reports by Asperger (7) and Kanner (7a) to first person accounts (7b). The distress caused by particular sensory stimuli can cause self-injurious and aggressive behavior in those who are unable to communicate their duress. Although sensory hyper- and hyporesponsiveness are not unique to ASD, they appear to be more prevalent in this population than in other developmental disabilities (4,8,9). There is limited consensus regarding the pattern of these sensory deficits in ASD. However, historically, proximal senses such as touch, smell, and taste were thought to be particularly at risk and to indicate developmental immaturity (10,11). Interestingly, these tend to be the least well studied of the sensory modalities, whereas there is mounting evidence for disruption of the auditory and visual processing pathways and a surging interest in multisensory integration (MSI). We will review the current literature on the neurophysiology in individuals on the autism spectrum with a focus on the processing of simple sensory input in the auditory, visual, and tactile modalities. We begin by considering the processing of unimodal stimuli, then we will address MSI, and finally, we will examine the role of attention on the sensory processing stream.

Auditory Sensory Processing

As language deficits are a core feature of ASD, the study of auditory processing is essential to considering the roots of ASD and to conceptualizing rational interventions. One way of measuring the flow of auditory information processing is through the traditional auditory brainstem response in which the electrical activity evoked from a series of clicks or tones is recorded in milliseconds using surface electrodes. Incoming auditory stimuli from the vestibulocochlear nerve (cranial nerve VIII) travel to processing structures in the brainstem (cochlear nuclei and the superior olivary complex) and midbrain (inferior colliculus). The auditory brainstem response literature reports varied and contradictory findings. Some studies show no differences in central transmission latency nor amplitude (12,13). In contrast, other studies have shown prolonged latencies in child and adolescent ASD cohorts (14,15). Both of these studies found significantly longer III–V interpeak latency (thought to represent neural conduction time

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Abbreviations: AS, Asperger syndrome; **ASDs,** autism spectrum disorders; **ERP,** event-related potentials; **MEG,** magnetoencephalography; **MMN,** mismatch negativity; **MSI,** multisensory integration

between cranial nerve VIII and the lateral leminiscus). A recent study increased the complexity of the auditory processing demand by using a "forward masking paradigm" in adults with AS and found an attenuation of wave III amplitude that separated AS individuals from control, schizophrenic, and attention deficit hyperactivity disorder individuals (16). In addition, work by Russo *et al*. (17,18) with ASD children demonstrates typical brainstem responses to clicks; however, they find differences in response to varied pitch and speech sounds with noise. Consequently, although brainstem abnormalities do not appear sufficient to explain the deficits for all individuals on the autism spectrum, there is literature suggesting measurable differences in early auditory pathways, especially with increasingly complex stimuli. Understanding the nature of this fundamental step in the auditory sensory stream is crucial because the ability to acquire and parse a variety of incoming sounds forms the foundation for language and communication.

Beyond the brainstem, cortical auditory sensory processing has traditionally been examined using event-related potentials (ERPs) with EEG and magnetoencephalography (MEG). Simple auditory stimuli are presented, and the brain responses are collected over multiple trials and averaged to generate information about the temporal and spatial resolution of responses. Both EEG and MEG studies show atypical latencies in the early peaks (<150 ms) that are thought to reflect activity from the primary and association auditory cortices. Unfortunately, these studies have shown differences in latency in both directions. Two studies using 1000 Hz tones showed faster cortical latencies with varying tone durations. Ferri *et al*. (19) used a 100-ms tone, and Martineau *et al*. (20) used a 4-ms tone. Others have shown delayed latencies relative to controls. Bruneau *et al*. showed late auditory evoked potentials with a 750-Hz tone and 200-ms duration (21); Oram Cardy *et al*. (22) found the same delay with a 1-kHz tone and 300-ms duration; and Roberts *et al*. (23), using MEG, report a delay in right hemisphere M100 response to 300-ms tones over a range of frequencies (200, 300, 500, and 1000 Hz). When assessing vowel sounds, complex nonspeech sounds, and complex tones, Whitehouse and Bishop (24) found differences in the early peak latencies of the standard repeated tones. Studies from two groups, Bruneau *et al.* (21) and Oram Cardy *et al.* (22), suggest that in this realm of low-level processing, earlier and higher right hemisphere cortical peaks might predict better language function (21,22). The discrepancy in findings may reflect the considerable variation in ages, diagnosis, and paradigms used. The addition of behavioral phenotyping and correlations is likely to help clarify this work going forward. Furthermore, the investigation of more complex auditory tasks (such as pitch discrimination and speech *versus* nonspeech paradigms) coupled with variation of attention will aid in understanding altered cortical processing in ASD and will be addressed further in the attention section below.

In general, the neurophysiologic study of auditory processing in autism does suggest atypical neural activity as early in the processing stream as the primary auditory cortex. However, as Whitehouse and Bishop (24) suggest, these differences may be a result of top-down inhibitory processes mediating encoding and early sound processing. As we learn more, we may be able to predict the nature of the atypical cortical activity by defining the etiology of the individual's ASD, such that children with Fragile X may show one form of cortical differences whereas children with 16p11 duplications may show another form. It is probable that the atypical processing is related to the unusual behavioral responses so commonly observed in children on the autism spectrum such as covering of the ears to seemingly benign sounds such as the vacuum cleaner and the blender. Furthermore, one might conjecture that if the auditory input is perceived as unpleasant or noxious, affected individuals will learn to avoid auditory input, and thus curtail the learning that comes from listening to the people and world around them. Comprehension of the potentially atypical auditory processing in children with autism may be key to parsing different etiologies of autism, targeting treatments to children with auditory hyper/hypo-sensitivities, and ameliorating overwhelming auditory sensory input to facilitate learning.

Tactile Sensory Processing

Although tactile sensitivity is commonly reported in ASD, it has received far less attention in the neuroscience literature than auditory sensitivity (25). Common clinical complaints are avoiding light touch to the head and body as occur with grooming and particular clothing. The psychophysical tactile studies look at thresholds and sensitivity using vibrotactile stimuli. Adults with AS showed lower tactile perceptual thresholds for 200 Hz but not 30 Hz vibrotactile stimuli, implying a specific hypersensitivity in the Pacinian corpuscles receptor pathway (3). Tactile hypersensitivity was again shown to vibrotactile stimuli as well as thermal stimuli but not to light touch in adults with autism (26). In contrast, in a small sample of children with autism, there were no tactile perceptual threshold differences for vibrotactile (40 and 250 Hz) detection (27). However, this study did suggest a correlation between a measure of behavioral tactile sensitivity phenotype and emotional/social reaction. (This trend is considerably underpowered with a sample size of only six boys.) Beyond threshold investigation, Miyazaki *et al*. (28) demonstrate an enhanced early (low-level) somatosensory evoked potential peak in young autistic children using median nerve stimulation that was most prevalent in the right hemisphere response. Coskun *et al*. (29) most recently investigated somatosensory mapping in high functioning adults with autism using MEG. High functioning adults with autism appear to have a disrupted cortical representation of their face and hand. Again, because of the heterogeneity of ASD, the electrophysiology and functional imaging work in this domain should include behavioral measures so that within group differences do not obscure real between group differences. There is a tremendous need for further exploration in this domain as atypical tactile sensitivity appears with particularly high frequency in the autism population.

Visual Sensory Processing

Individuals with ASD also exhibit atypical visual behavior that can be construed as attempting to avoid visual input (*e.g.*

covering eyes at bright lights) or to seek additional visual stimuli (*e.g.* twisting fingers in front of eyes) (4). Similar to the auditory and tactile domains, there is considerable discrepancy in neurophysiological findings. There are suggestive reports in the visual domain of enhanced detail perception, particularly for simple stimuli with impairment in more complex tasks (30). Some threshold studies show no difference between ASD individuals and controls in contrast sensitivity for low *versus* high spatial frequencies or motion/form processing (31,32). Other visual-evoked potential studies indicate that individuals with ASD possess atypical early peaks with impairments in object boundary detection (33), decreased contrast detection ability in both still and moving stimuli at a range of signal/noise ratios (34), and undifferentiated responses for mid- and high spatial frequency gratings (35). Local motion processing studies show differences in second order (texture defined) motion processing but intact first-order (luminance defined) processing, suggesting difficulties with effective integration of incoming stimuli that is magnified with more nuanced tasks (36).

One of the most well-studied aspects of visual perception in autism is that of face processing given the pertinence of this skill for human social interaction (37). As Klin (38) suggests, the literature is heavily confounded by differences in the familiarity of the face, attention, gaze direction and fixation, and the type/complexity of the stimulus. A functional MRI study with eye tracking shows that activation of the fusiform gyrus and the amygdala is reduced in an ASD cohort, as well as their unaffected siblings, but correlates positively with fixation time on the eye region of the face (39,40). An ERP study again highlights group differences that are dependent on directed attention such that ASD individuals do not show the expected increase in the N170 (face processing) wave with directed attention (41). An EEG study assessing γ-band activity, thought to represent the binding of visual information, gives convergent evidence for a neurophysiologic difference in AS face processing (42). Furthermore, the type of visual information matters; children with autism may respond more robustly than controls to neutral and detailed, high spatial frequency information and less robustly to the rapid low-frequency processing that is so critical to our fast-paced social world (43). The emotional valence of face processing has been investigated with a recent study suggesting hyperactivity in the right amygdala with altered connectivity between the frontal and temporal lobes (44). It is a challenge to interpret whether these differences represent primary cortical abnormalities, result from decreased visual exploration in early infancy, or are secondary to a primary social cognitive deficit.

Deficits in simple stimuli and faces extend to studies of biological motion, such that children with autism show impairments in the processing of dynamic noise, motion coherence, and form-from-motion detection (45). There are suggestions that this observed deficit may result in part from atypical processing of emotional information as children with autism were found to differ from control children only in their ability to name emotional point-light displays and not point-light displays of everyday objects (46). This finding suggests a potential disconnection from the limbic or "emotion" neural networks that inform primary sensory processing. Speaking to a genetic underpinning for these differences, inefficient motion processing has been found in siblings of individuals with ASD as well (47). In accordance with theories of increased local cortical activity (48) with impaired long-range connectivity (49), individuals with autism appear to be over-recruiting their left primary cortex compared with typicals during a motion coherence functional MRI study (50). Taken as a whole, these studies further support a disruption in the processing of basic unimodal sensory information that forms the backbone of higher order cortical abilities such as socialization.

Low-Level Multisensory Integration

Similar to the aforementioned deficits in unimodal sensory processing in children with ASD, these individuals may also perform poorly during conditions that require collapsing information across multiple modalities (or MSI). Many of the atypical perceptual experiences reported in those with ASD are believed to be due to an inability to properly filter or process simultaneous channels of visual, auditory, and tactile inputs (51). There is evidence that sensory illusions that require the proper concatenation of inputs across multiple domains operate at a different level in ASD, compared with typically developing individuals. In the "flash-beep" illusion, multiple auditory tones paired with a single transient visual stimuli can induce the perception that multiple flashes are present. At a cursory level, it appears that the integration necessary to produce this illusion is preserved in ASD, as demonstrated through a lack of difference between patients and Intelligence Quotient (IQ)-matched typical individuals (52). However, when the timing between stimulus sets is perturbed during presentation, deficits in processing begin to emerge in subjects with autism. Typically, disparity between the auditory and visual stimulus onset times will impact the effect of the illusion, until they appear uncoupled at a certain threshold. Foss-Feig *et al*. (53) were able to demonstrate that, in subjects with autism, the time duration between stimuli that continue to produce the illusion are broader than in typically developing individuals. The observation that broader temporal gaps continue to produce a "flash-beep" illusion in individuals with ASD suggests a level of inefficiency in the MSI in this population.

Electrophysiological studies probe the neural mechanisms of ASD that can manifest as behavioral multisensory deficits. EEG studies of multisensory processing have reported abnormal timing and level of activity within electrophysiological signatures of brain processing. Courchesne *et al*. (54,55) report that in individuals with ASD, a reduction in response amplitude (compared with typically developing children) is evident when concurrent auditory and visual stimuli streams are presented. The sequence of activity in the brain during MSI seems to deviate in children with autism, particularly within the later stages of processing when sensory information is collapsed. When auditory and somatosensory stimuli are presented in parallel, early ($<$100 ms) electrical potentials in primary sensory cortices are relatively spared in ASD; however, responses that follow this initial stage of activity in the cortex (at around 175 ms) are limited and delayed in ASD (56). These investigations indicate that both the magnitude

and the latency of activity in the brain may contribute to multisensory processing deficits in ASD.

Higher-Order Multisensory Integration

Although both behavioral and neurophysiological processing impairments in simple MSI have been reported in ASD, salient differences in sensory integration are also evident at a complex level, particularly during speech comprehension and production. When audio and visual speech stimuli are staggered and presented to individuals with autism, performance drops to a chance level and indicates deficits in speech comprehension (57). Multimodal illusions of linguistic processing in ASD, such as the McGurk effect, suggest that improper timing of sensory integration contributes to observable deficits in communication in ASD. In the McGurk effect, visual processing (*e.g.* lip reading) is combined with auditory processing (phoneme perception) to produce the comprehension of spoken language. Although both typically developing and ASD individuals perform well during this task, typical individuals show a greater dependence on visual feedback (lip reading) compared with ASD (58,59). When both groups are trained on the visual feedback component of the McGurk effect, ASD participants fail to show improvements in performance (60,61). Furthermore, a reliance on visual feedback in noisy auditory environments is unattainable for ASD participants (61). An inability to "fall back" on certain sets of sensory stimuli in the presence of challenging environmental stimuli may contribute to the communication deficits that are well characterized in this disorder.

MSI investigations exploring the specific neurophysiological mechanisms that are compromised in ASD is just beginning (62). Many of the regions known to integrate multiple sensory inputs have been implicated, including prefrontal cortex and association regions of the temporal lobe. At the cellular level, postmortem studies of ASD have illustrated that the columnar density in the neocortex is dense in autism, potentially facilitating local processing (63). It has also been hypothesized that the cerebellum, a structure that shows significant changes in neuronal density in autism (64), may play a role in impaired sensory integration in the disorder. This mediation could occur through atypical filtering of afferent inputs, although these exact mechanisms are unclear (65). Many of the neocortical fields that play a role in MSI are also part of a putative "mirror neuron" network, first identified in homologues of these regions in nonhuman primates (66). Given the observable deficits in imitation and empathy known to be a core feature of the autism spectrum, it has been proposed that communication deficits arise from an inability of multisensory "mirror neurons" to concatenate information to facilitate higher order cognitive function (67). However, others propose that as sensory integration is dependent on the rapid exchange of information between distinct cortical and subcortical regions, disruptions in connectivity likely play the causative role (68). The ASD literature suggests both direct axonal disconnection such as has been implied by the abnormalities of the corpus callosum (69) and indirect disruption of long-range firing synchrony (70,71).

Attention Impacts Every Stage of Sensory Processing

The discussion of sensory processing in ASD would be incomplete without the consideration of the role of attention on cognitive processing. In their review, Allen and Courchesne (72) suggest that that the clinical observation of heightened reactivity to seemingly meaningless stimuli (*e.g.* intense tantrums in response to the hum of a blender) may be related to a neurobehavioral driven distractibility. Furthermore, narrowed interest and repetitive behaviors may represent deficits in attentional shifting. However, even defining attention is a challenging matter. According to Talsma *et al.* (73), "attention is a relatively broad cognitive concept that includes a set of mechanisms that determine how particular sensory input, perceptual objects, trains of thought, or courses of action are selected for further processing from an array of concurrent possible stimuli, objects, thoughts and actions." Functionally, an individual must be able to select certain sensory inputs for enhanced processing while either filtering out or suppressing others. This selective attention can be further subdivided in operations such as attentional switching and sustained attention over time (74–76). Many brain regions are involved in processing, modulating, and integrating sensory information. There has been a particular focus on the superior colliculus, the cerebellum, and the frontal lobes in understanding this rapid and multidirectional flow of information, which is mediated by attentional demands and resources (77,78). We suggest that this multidirectional flow of information is impaired for individuals with ASD and that this disruption in cortical communication underlies the individual's inability to attend to their environment in a flexible, productive, and meaningful way. In the following sections, we will focus on two aspects of attending: first, the ability to shift focus from stimuli of one type to another (attentional switching); and second, the effect of increasing the array of information presented to measure the subject's ability to select what information needs to be attended to and what needs to be ignored (selective attention).

Attentional Shift or Switch

In this section, we will focus on studies in which the subject shifts their attention to changes in the stimuli. In ASD neurophysiologic research, the most common form of attentional switch is between a repeated stimulus and an unfamiliar or novel stimulus within the same sensory modality (exogenous attention). However, shifting paradigms can also require the subject to move from one modality to another or to shift visual or auditory focus in space (endogenous attention). In the auditory domain, researchers have primarily used the oddball paradigm to investigate attentional shift. In the oddball paradigm, a stimulus that varies on a single parameter (deviant) such as duration, frequency or intensity, is randomly inserted into a train of repeated (standard) stimuli. This deviance leads to the generation of a negative deflection on an evoked potential recording at 150–200 ms, which is best recorded from the fronto-central sites (79). This paradigm can be extended from covert (preattentive) to overt attention with a

task requiring a response to the deviant (target), and other variations of this paradigm include a third rare stimuli as a nontarget (novel) comparison. In the oddball paradigm, the difference between the neural response to the standard stimuli and the deviant stimuli is called the mismatch negativity (MMN) when using an EEG recording technique or the mismatch field when using MEG. MMN/mismatch field wave forms have generated widely disparate results from normal in an ERP study of high functioning children with autism (80) to completely absent in an MEG study of low-functioning individuals with autism (81). Although there are conflicting data from other studies (82–85), Gomot *et al*. (86,87) report faster MMN latencies for pitch variation and atypical activation of the left anterior cingulate. This location has been implicated in attentional switching and correlated with a behavioral measure of intolerance to change. This reduced mismatch latency to pitch variation in conjunction with superior pitch recognition has been interpreted to support the theory of perceptual enhancement, whereby local processing networks are over connected at the expense of long-range connections with integration and attention networks (88–90).

Conflicting findings have also been reported for auditory MMN amplitudes. Several groups have found increased MMN amplitude in samples of adults and children with AS and ASD (19,91,92), whereas Dunn *et al*. (12) found reduced MMN amplitudes using a passive paradigm. Attention shifting for individuals with autism has received less focus in the visual and somatosensory domains, perhaps related to the intense interest in the auditory domain as the gateway for understanding the language and communication deficits that are central to ASDs. When Kemner *et al*. (93) assessed the role of visual attention using an oddball paradigm with both a passive condition and an active counting task, they found that children with autism did not differ from controls in the passive condition, but they did show a larger response to the deviant stimuli during the active task condition.

The importance of directed or overt attention on the effects of cortical processing of novelty is further highlighted by the work of Whitehouse and Bishop (24). To clarify previous findings, suggesting that orienting deficits in autism might be speech-sound specific (80), Whitehouse and Bishop performed a layered study of boys with high functioning autism examining whether processing deficits were due to a perceptual impairment (in acoustic encoding or discrimination of different speech sounds) or a function of cognitive factors (such as reduced attention). They found that, during a passive condition, children with autism showed attenuated early cortical responses to speech sounds but not complex tones. However, when the children were instructed to attend to and respond to the deviant condition, these amplitude differences were no longer evident. Similarly, Dunn *et al*. (12) found that the decreased MMN to simple stimuli, apparent during a passive condition, normalized with directed attention. These studies suggest that a "top down" process mediated by directed attention influences basic sensory processing for individuals on the autism spectrum.

Selective Attention

Beyond the effects of attentional shifting, there is interest in how individuals with ASD select what information to attend to, what to ignore, and how this guides their ability to make sense of the changing world around them. In EEG/MEG studies of attentional shift, one response property of interest is the P300. The P3a is a positive deflection culminating around 300 ms that is thought to reflect orienting to changes in the environment that may underlie attentional switching; the P3b is a component of the late attention peak that reflects task-related cortical activity and may underlie working memory. The P3b is thought to emanate from temporal and parietal neural sources (94). The earliest autism study reporting a P300 attention wave targeted attention by presenting a train of stroboscopic flashes with an occasional missing flashes (95). In the three individuals investigated, the study investigators found good accuracy in the behavioral task but small or absent late positive waves. This suggests, as has been seen in the auditory literature, that in simple tasks, behavioral performance can be similar between groups while the cortical activity differs. In a series of visual oddball studies, Courchesne *et al*. (54) first used a letter mismatch and found normal P3b amplitudes; in a later study, they used blue and red squares (color mismatch) and again found typical P3b responses with targeted attention (54,55). In a subsequent study, they added an additional level of spatial complexity to the task—there were five empty squares, one of which was designated to be attended to; when the circle appeared in the attended box (target), the participant responded with a button press; when the circle appeared in an "un"attended box, the condition was ignored. In this visual-spatial selective attention task, they found a delay in the frontal P3a (attention orienting) and a diminution in the parietal P3b (96). With this degree of spatial challenge, this cohort of high functioning ASD males had difficulty in both speed and accuracy relative to matched controls. This series suggests that increasing the attention and capacity demands of this visual task leads to both behavioral and physiologic differences in individuals with autism versus controls, whereas simple visual attention tasks may fail distinguish them. Other visual oddball studies support this finding of diminished P3 amplitudes and have correlated a shorter visual fixation period with the P3 diminution (93,97). These investigations suggest that the density and complexity of the incoming stimuli may affect the degree to which the attention neural networks are recruited for processing of incoming sensory information.

Our ability to attend appears to have a limited capacity (*i.e.* there is a finite quantity of information that can be considered simultaneously), and we therefore need to selectively concentrate on one aspect of the environment while ignoring other features to effectively and efficiency process sensory input (75). This capacity may be even more limited in certain subgroups of individuals with ASD. An ERP auditory task with selected spatial attention demonstrates this capacity effect: high functioning adults with autism showed both behaviorally diminished ability to selectively tune into a specified sound source as well as an ERP signature of this deficit with

relatively broader N1 and shallower P3 peaks when compared with a typical control group (98). This finding was only evident with increased task complexity (*i.e.* more speakers and a continuous, rapid stream of complex tone distractors). In a task of divided attention between visual and auditory stimuli, the failure of the autism group to modulate the slow negative wave in response to focused/divided/ignored conditions is thought to indicate a potential deficit in selective inhibition and attention (99). This finding echoes the anecdotal reports of parents that children with autism can function typically in a well-controlled environment but decompensate in the face of challenging sensory environments such as a grocery store or an animated birthday party. Children with autism may have more difficulty with automatic processing of information and may already rely more heavily on already overloaded attention and working-memory based networks, such that when the stimuli reach and exceed capacity, the processing system fails (12,90).

Conclusion

Given the ubiquitous nature of sensory behavioral differences for individuals with autism, understanding the neural underpinnings of basic sensory processing in ASDs is an important task. Furthermore, as the neurophysiologic data mount, we suggest that differences in sensory processing may actually cause core features of autism such as language delay (auditory processing) and difficulty with reading emotion from faces (visual processing). Interpreting the neuroscience has been complicated by the heterogeneity of the disorder as well as the difficulty in designing tasks that can precisely probe our finely tuned and intricately connected sensory neural networks. Despite these challenges, tremendous gains have been made over the past 30 years and will guide both our understanding of the disorder as well as provide insights into how to strengthen basic processing and attention for affected individuals.

Going forward, studies of infant siblings of individuals affected with ASD can provide an understanding of whether sensory processing differences are a primary feature of the disorder or a result of learned behaviors. Behavioral intervention trials, such as computerized training modules and self-regulation programs, need to be studied both for efficacy and to determine whether there is normalization of neural activity in affected individuals. Psychopharmacology studies targeting attention and arousal paired with functional imaging assessments hold great promise in providing valuable treatment models. Finally, careful sensory behavioral phenotyping is essential for both understanding our neurophysiologic research as well as tailoring appropriate and effective treatments.

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