Tactile sensitivity in Asperger syndrome

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Abstract

People with autism and Asperger syndrome are anecdotally said to be hypersensitive to touch. In two experiments, we measured tactile thresholds and suprathreshold tactile sensitivity in a group of adults with Asperger syndrome. In the first experiment, tactile perceptual thresholds were measured. Two frequencies of vibrotactile stimulation were used: 30 and 200 Hz. The results demonstrated significantly lower tactile perceptual thresholds in the Asperger group at 200 Hz but not at 30 Hz, thus confirming tactile hypersensitivity but only for one class of stimulus. A second experiment investigated whether self-produced movement affected the perception of touch in a group of adults with Asperger syndrome. A suprathreshold tactile stimulus was produced either by the participant (self-produced condition) or by the experimenter (externally produced condition) and participants were asked to rate the perception of the tactile stimulation. The results demonstrated that, while both Asperger and control groups rated self-produced touch as less tickly than external touch, the Asperger group rated both types of tactile stimulus as significantly more tickly and intense than did the control group. This experiment confirms the finding of tactile hypersensitivity, but shows that the perceptual consequences of self-produced touch are attenuated in the normal way in people with Asperger syndrome. An abnormality in this process cannot therefore account for their tactile hypersensitivity.

Keywords: Perception; Sensory; Somatosensory; Autism; Tickling; Self; Other; Methods of limits; Hypersensitivity; Weak central coherence

1. Introduction

“*To be just lightly touched appeared to make my nervous system whimper, as if the nerve ends were curling up. If anyone hit on the terrible idea of tickling me, I died. It was so way beyond unbearable un bearableness that I simply died—or that's what it felt like.” (Gerland, 1997, p. 38). “I pulled away when people try to hug me, because being touched sent an overwhelming wave of stimulation through my body... Small itches and scratches that most people ignored were torture... When my mother scrubbed my hair, my scalp hurt; I also had problems with adapting to new clothing on my body.” (Grandin, 1996). Despite these vivid autobiographical reports by individuals with autistic disorder, there is a surprising lack of empirical research on the sensitivity to touch in autism.

Hans Asperger, in his first description of autism, drew attention to the hypersensitivity of the senses, especially touch, smell and taste (Asperger, 1944; Talay-Ongan & Wood, 2000). Since then, hypersensitivity to touch has been reported extensively, mainly anecdotally, in people with autism. As shown in the examples above, people with autistic disorder and their carers report that they are intolerant of certain textures and find wearing certain materials aversive (see also Rogers, Hepburn, & Wehner, 2003; Willey, 1999). On questionnaires evaluating sensory perception, for example Dunn’s Sensory Profile questionnaire, parents report that their autistic children overreact to cold, heat, pain, tickle and itch and avoid being touched by other people (Dunn, 2001; Kientz & Dunn, 1997).

A related concept is tactile defensiveness, which is characterised by behaviours such as rubbing, scratching, negative expressions, withdrawal, or avoidance in response to...
tactile stimulation (Royeen, 1986). Tactile defensiveness is elevated in several developmental disorders including autism and is associated with enhanced response and slower habituation rates to a repeated tactile stimulus (Baranek & Berkson, 1994; Baranek, Foster, & Berkson, 1997). Thus, a lack of habituation in the neural pathways that normally occurs after being exposed repeatedly to a sensory stimulus is a possible explanation of tactile hypersensitivity. Some evidence for a failure to show response habituation to repeated stimulation in the visual and auditory domain has indeed been reported in autism (Barry & James, 1988).

Hypersensitivity may be the result of changes at one or more sensory processing stages, ranging from peripheral receptors in the skin, spinal synapses, the brain’s perceptual system, through to cognitive or emotional processes. However, it is unknown at what level the hypersensitivity reported in autism occurs. The theory of weak central coherence (Booth, Charlton, Hughes, & Happé, 2003; Frith, 1989; Happé, 1996, 1999; Jolliffe & Baron-Cohen, 2001), proposes that in autism information processing is biased such that individual stimuli are well analysed but not integrated sufficiently into a coherent meaningful Gestalt. Thus, hypersensitivity could be due to impaired top-down modulation of incoming stimuli (Frith, 2003; U. Frith, 2003). Top-down modulation in the brain normally acts as a filter so that expected stimuli do not have to be processed as thoroughly as new stimuli. Such filters normally function to prevent informational overload. If this aspect of information processing was impaired in autism then incoming stimuli would all be processed as unexpected, resulting in enhanced sensitivity. This account might explain why there is a lack of habituation.

Other accounts of perceptual abilities in autism (Mottron, Burack, 2001; Plaisted, 2001; Plaisted, Saksida, Alcantara, & Weisblatt, 2003) suggest that there is enhanced processing of detailed stimuli (Bonnel et al., 2003; Plaisted, O’Riordan, & Baron-Cohen, 1998), or an over-development of low-level perceptual operations which causes detection, discrimination, and other low-level tasks to be enhanced (Mottron, Burack, Iarocci, Belleville, & Enns, 2003), without implications for global processing. These accounts too suggest a mechanism for hypersensitivity.

The main problem of these theories is that they would predict hypersensitivity to all perceptual stimuli. However, several studies have shown that enhanced discrimination, which may be a type of hypersensitivity, does not apply wholesale to all stimuli even within the same modality. In the visual domain, studies have revealed a specific deficit in the processing of magnocellular properties of motion stimuli (Milne et al., 2002; Spencer et al., 2000), which is not accompanied by a deficit in processing of parvocellular properties of form (Spencer et al., 2000). Spencer et al. (2000) interpreted these results as demonstrating a specific deficit of dorsal (but not ventral) stream functioning in autism. An alternative explanation for these results pertains to the “complexity” of the visual stimuli. In a recent study on sensitivity to visual motion stimuli in autism, Bertone, Mottron, Jelenic, and Faubert (2003) suggest that first-order (simple) and second-order (complex) neural processes need to be distinguished. Second order, or complex, stimuli are those requiring additional integration of information (central coherence), while first order, or simple, stimuli do not. Bertone et al. (2003) showed a dissociation in motion direction identification thresholds in autism according to the complexity of the visual motion stimuli. While individuals with autism had similar identification thresholds as control subjects for simple motion, they were less sensitive than controls for complex motion, which requires integration. Although there are not known to be separate neural pathways in the processing of tactile stimulation, in the domain of touch, many of the anecdotal reports are suggestive of hypersensitivity to certain tactile stimuli and not others.

In this study, we examined the perception of touch in individuals with Asperger syndrome (AS) and normal control (NC) participants. In the first experiment, we examined sensitivity to vibrotactile stimuli at two different frequencies (30 and 200 Hz). These two frequencies were chosen because they are known to stimulate different mechanoreceptors in the skin. High-frequency vibration (200 Hz) stimulates Pacinian corpuscles and activates FAII fibres, whereas lower-frequency vibration (30 Hz) stimulates Meissner corpuscles and activates SAI fibres. We wished to explore whether hypersensitivity would be found in people with autism within one or both of these neural systems. The first experiment was therefore designed to investigate whether people with AS have lower tactile perception thresholds to vibratory tactile stimulation, and to investigate the generality of any effect across different submodalities of stimulation.

2. Experiment 1 method

2.1. Participants

A group of participants with a diagnosis of AS (N = 10; 3 females) and a group of NC participants (N = 9; 7 females) took part in Experiment 1. Each participant in the AS group had previously received a diagnosis of Asperger syndrome from an independent clinician according to standard criteria (DSM-IV, APA 1994). All participants were right handed. Participants were questioned about their general health and were excluded if they were on medication or had a history of psychiatric or neurological illness. The mean age of the participants was 32.2 (±12.9) years in the AS group and 26.9 (±9.5) years in the NC group. There was no significant difference between the ages of the two groups (t = 1.01; p = .33). Assessments of Verbal, Performance, and Full-Scale IQ were carried out on seven of the AS participants using the eleven IQ subtests of the Wechsler Adult Intelligence Scale (WAIS-IIIUK; Wechsler, 1999a). Due to time constraints, two AS participants were assessed using a shortened form of the WAIS, and one was tested using the Wechsler Abbreviated Scale of Intelligence.
Two frequencies of vibration were used: 200 and 30 Hz. The waveform was amplified using a Stereo Amplifier (Marantz PM520DC). Participants were seated throughout the experiment and effort was taken to ensure that they were sitting comfortably and that the position of their left hand was comfortable. The position of the participant’s index finger was maintained throughout the experiment.

A Method of Limits (MOL) procedure was used to determine the tactile perception threshold of each participant at each of the two frequencies. In MOL, stimulus intensity is gradually increased and decreased, and the participant is required to report the moment of stimulus detection and extinction, respectively. In this study, each vibration stimulus is reported in microns (μm) and refers to stimulus displacement amplitude. A descending block of trials was presented followed by an ascending block, with 5 presentations of each in total. In a descending block, a suprathreshold stimulus was applied to the participant’s fingertip, and the participant was asked to indicate whether he or she felt the stimulus or not. Threshold was taken as the amplitude at which the participant could no longer detect the stimulus. Threshold was taken as the amplitude at which the participant could not feel the stimulus on two consecutive ascents. In an ascending block, the amplitude of the first trial was determined as 3 steps below the threshold value found in the previous descending block. This value was increased using the same step sizes as in the descending block, and the participant was asked to indicate whether he or she felt the stimulus or not. Threshold was taken as the amplitude at which the participant could feel the stimulus on two consecutive trials. The same procedure was used for the 30 and the 200 Hz conditions, which were presented in a counterbalanced order in separate phases of the experiment. The tactile perception threshold of each participant was calculated as the mean value of the five ascending and five descending blocks for each frequency.

3. Results

All participants were able to perform the task. A between-subjects ANOVA was carried out with two factors (frequency and group). A main effect of frequency was found \[F(1,17) = 18.590, p < .01\] but there was no significant main effect of group \[F(1,17) = 1.209, p = .29\]. More interestingly, there was a significant interaction between frequency and group \[F(1,17) = 5.173, p < .05\]. Post hoc independent samples t tests were used to investigate the difference in thresholds between the two groups at each frequency. Tactile perception threshold at 200 Hz was significantly lower in the AS group than in the NC group \((t = -2.248, p < .05)\). Therefore, the AS group were hypersensitive to vibratory stimuli of 200 Hz, relative to the NC group (Fig. 2). No significant difference was found between the AS and NC groups for tactile thresholds at 30 Hz \((t = 1.703, p = .11)\), although there was a non-significant trend suggesting that the AS group were less sensitive than...
the NC group. Post hoc paired t tests were used to investigate the difference in thresholds between the two frequencies for each group. For the AS group, tactile perception thresholds were significantly lower for 200 Hz compared with 30 Hz \((t = -4.013, p < .01)\). For the NC group, tactile thresholds were also lower for 200 Hz compared to 30 Hz, but this did not reach significance on a two-tailed test \((t = -1.937, p = .09)\) (see Fig. 2).

Individual data are shown in Table 2. This demonstrates that for the 200 Hz stimuli, only two NC participants had thresholds that were lower than the mean threshold of the AS group, and only one AS participant had a threshold that was higher than the mean threshold of the NC group. The mean threshold for all subjects (from both groups) was calculated for each frequency (200 Hz mean threshold = 2.02 \(\mu\)m; 30 Hz mean threshold = 9.8 \(\mu\)m). The number of participants from each group with a lower threshold than the overall mean threshold was calculated for each group, and for each frequency. Comparison of the two groups revealed a significant difference between the number of participants with a lower threshold than the overall mean threshold at 200 Hz \((\chi^2(1) = 4.55, p < .05)\) but not at 30 Hz \((\chi^2(1) = 2.04, p = .2)\).

4. Discussion

The results of Experiment 1 demonstrate that AS individuals had significantly lower tactile perception thresholds (were hypersensitive) to vibrotactile stimuli at 200 Hz compared with the control group. This was not the case for vibrotactile stimuli at 30 Hz, where AS individuals showed a non-significant trend towards having higher tactile perception thresholds than the control group. Taken together these results suggest that AS individuals are hypersensitive to high frequency, but not to low frequency, vibrotactile stimulation. There are notable differences between high and low-frequency vibratory stimulation in terms of the receptor pathways activated by each type of frequency stimulus. Tactile stimuli vibrating at 200 Hz stimulate Pacinian corpuscles, whereas lower-frequency vibration (e.g., 30 Hz) stimulates Meissner’s corpuscles (Kandel, Schwartz, & Jessell, 2000). Meissner’s corpuscles are the most common mechanoreceptors of glabrous (hairless) skin (for example the fingertips), and their afferent fibres account for about 40% of the sensory innervation of the human hand. Meissner’s corpuscles are activated primarily by light touch. Pacinian corpuscles are large, encapsulated endings located in the subcutaneous tissue. These receptors differ from Meissner’s corpuscles in their morphology, distribution, and response threshold. Tactile thresholds are generally lower for high-frequency stimuli than for low-frequency stimuli, as was found in the current study. High-frequency vibration is often perceived as a “hum” or “buzz,” whereas low frequency feels more like a “flutter.” Pacinian corpuscles are involved in the discrimination of fine surface textures or other moving stimuli that produce high-frequency vibration of the skin. Stimulation of Pacinian corpuscle afferent fibres in humans induces a sensation of vibration or tickle (Selden, 2004). Thus, the specific hypersensitivity to higher-frequency vibrotactile stimuli in AS fits well with previous anecdotal reports relating to tickling sensations. To continue this focus on time-varying somatosensory coding, we investigated in a second experiment whether people with AS are hypersensitive to tickle, and whether self-produced movement affects the perception of tickle in AS.

Table 2

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<th>Mean threshold 30 Hz (in (\mu)m)</th>
<th>Mean threshold 200 Hz (in (\mu)m)</th>
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5. Experiment 2

Experiment 2 was designed to investigate whether (a) tickle to the palm of the hand is rated as more intense and tickly in AS because it is thought to involve Pacinian receptors, and (b) whether tickle sensation is modulated by self-generated movement in AS. For every intended action, the brain must issue a motor command to the muscles to execute the action. It is proposed that an ‘efference copy’ is generated in parallel with the motor command and used to make predictions about the sensory consequences of one’s own action (von Holst, 1954). It has been proposed that a forward model predicts the sensory consequences of the motor act and compares them with the actual effect of the movement (Wolpert, Ghahramani, & Jordan, 1995). This comparison can be used to cancel the sensory effect of the motor act, attenuating it perceptually compared with identical stimulation that is externally produced.

This predictive system can be used to filter incoming sensory signals, picking out sensory information caused externally, such as touch produced by an external object or agent, and distinguishing it from sensory stimulation that occurs as a necessary consequence of self-produced motion. It has been demonstrated that, in healthy people, self-generated sensory stimulation is perceptually attenuated relative to external tactile stimulation (Blakemore, Frith, & Wolpert, 1999; Shergill, Bays, Frith, & Wolpert, 2003; Weiskrantz, Elliot, & Darlington, 1971). One such demonstration of the attenuation of self-produced stimulation is that people cannot tickle themselves (Blakemore et al., 1999; Weiskrantz et al., 1971).

In Experiment 2, AS and NC participants were asked to rate the sensation of a tactile stimulus (a piece of soft foam) on the palm of their hand. The tactile stimulus was produced either by the participant himself or by the experimenter. It has been shown that schizophrenic patients with delusions of control and/or auditory hallucinations do not show the normal attenuation of self-produced relative to external tactile stimuli. Such patients perceive self-produced touch as equally tickly and intense as external touch (Blakemore, Smith, Steel, Johnstone, & Frith, 2000). Similarities have been drawn in terms of symptomatology and underlying cognitive deficits between schizophrenia and autism (Frith, 1992). One possibility is that people with AS, like schizophrenic patients with auditory hallucinations and/or delusions of control, would not show the normal attenuation of self-generated touch. In addition, because it is believed that stimulation of Pacinian corpuscle afferent fibres elicits a sensation of tickle, based on the results of Experiment 1, we predicted that people with AS would show hypersensitivity to tickle stimuli.

6. Experiment 2 method

6.1. Participants

A group of participants with a diagnosis of AS (N=16; 3 females) and a group of normal control participants (N=16; 9 females), all right-handed, took part in Experiment 2. This sample included all of the AS (N=10) and NC (N=9) participants from Experiment 1. The mean age of the participants was 27.3 (±12.0) years in the AS group and 33.9 (±12.5) years in the NC group. There was no significant difference between the ages of the two groups (t = −1.53, p = .14). Assessments of Verbal, Performance, and Full-Scale IQ were carried out on all additional participants in both groups using the eleven IQ subtests of the Wechsler Adult Intelligence Scale (WAIS-III UK; Wechsler, 1999a, 1999b). Age and IQ data from the new participants are shown in Table 3. Each participant gave his or her informed consent before taking part in this study, which was approved by the local ethics committee.

6.2. Procedure

6.2.1. Sandpaper control trials

Participants were asked to rate the roughness of four grades of sandpaper on a scale from 0 (not at all rough) to 10 (extremely rough), so that we could assess objectively their ability to rate tactile sensation. The pieces of sandpaper were of equal size and stuck to a piece of card. The participants were asked to move the index and middle fingertips of their left hand over each piece of sandpaper a maximum of six times, in a random order with their eyes closed. Participants were excluded from this study at the analysis stage if they failed to notice any difference in the roughness of the sandpaper.

6.2.2. Tickling procedure

The equipment that was used to produce the tactile stimulus consisted of a piece of foam attached to a plastic rod.
interesting to note that more AS participants than NCs rated the stimuli as irritating or painful. However, because of the low numbers of these ratings overall, we were unable to analyse these data statistically.

For tickly, intense and pleasant ratings, a two way repeated measures ANOVA was used to test the difference between perception ratings in the two conditions (self and external) and for the two groups of participants (AS and NC) and the interaction between condition and group.

7.3. Tickly ratings

There was a significant main effect of condition [F(1, 30) = 31.85, p < .005]. There was no significant main effect of group [F(1, 30) = 2.04, p = .16], nor was there a significant interaction between group and condition [F(1, 30) = 0.34, p = .56]. On the basis of the results of Experiment 1, we predicted that the AS group would rate the tactile stimuli as more tickly and intense than the NC group. To test this a priori prediction, independent sample t tests were used to test the significance of the difference between the ratings of the two groups in each condition. Tickly ratings were significantly higher for the group of AS individuals than for the NC group for self-produced touch (t = 1.26, p < .05 one-tailed) and for externally produced touch (t = 1.44, p < .05 one-tailed). Paired t tests were used to investigate the difference in ratings between the two conditions for each group. Tickly ratings were significantly lower for self-produced than for externally produced touch in both the AS group (t = -4.07, p < .005) and the NC group (t = -3.93, p < .05). These data are shown in Fig. 4.

7.4. Intensity ratings

There was a significant main effect of condition [F(1, 26) = 5.04, p < .05]. There was no significant main effect of group [F(1, 26) = 1.67, p = .21], nor was there a significant interaction between group and condition [F(1, 25) = 0.42, p = .52]. Post hoc independent samples t tests were performed to investigate the a priori prediction that there would be a difference in rating between the two groups in

7. Results

7.1. Sandpaper control trials

All participants were able to rate the roughness of the four pieces of sandpaper correctly except one AS participant (male, age 21), who rated 3 of the 4 grades of sandpaper as equally rough. His results were excluded from the analysis.

7.2. Self versus externally generated tactile stimulation

Six AS participants rated the stimuli as irritating and two rated them as painful. Two NC participants rated the stimuli as irritating and one rated them as painful. It is
each condition. These demonstrated that the intensity ratings were significantly higher in the group of AS individuals than in the NC group for self-produced touch ($t = 1.55, p < .05$ one-tailed) but not for externally produced touch ($t = 0.94, p = .12$). Paired $t$ tests were used to investigate the difference in ratings between the two conditions for each group. Intensity ratings were significantly lower for self-produced than for externally produced touch in the NC group ($t = -2.31, p < .05$) but not the AS group ($t = -1.10, p = .29$). Therefore, the AS group had similar levels of intensity ratings regardless of whether the stimulus was produced by the self or externally. This contrasts with the tickle data, which showed a significant self/other difference for both groups. These data are shown in Fig. 5.

7.5. Pleasant ratings

The ANOVA demonstrated that there was no significant main effect of condition [$F(1, 30) = 0.09, p = .77$], or group [$F(1, 30) = 2.27, p = .14$], nor was there a significant interaction between group and condition [$F(1, 30) = 1.18, p = .29$]. Since we had no a priori predictions with regards to the pleasant ratings, no post hoc tests were carried out.

8. Discussion

Experiment 2 was designed to investigate whether the difference in sensory perception between AS and NC participants found in Experiment 1 is found for tickle stimuli, which are believed to be produced by stimulation of Pacinian corpuscles. In addition, we sought to investigate whether the perception of tickle is modulated by self-generated movement. The results showed that AS and NC individuals show a similar attenuation of the tickleness of self-produced touch relative to external touch. Furthermore, AS individuals rated both self-produced and external touch as more tickly than did the control participants. These results, based on suprathreshold magnitude estimation, replicate the detection threshold results of Experiment 1, again suggesting hypersensitivity to certain types of tactile stimulation in AS, in particular tactile stimuli that are mediated by Pacinian corpuscles.

Previous studies have shown that healthy subjects perceive self-produced tactile stimuli as less tickly and intense than the same stimuli when it is externally produced (Blakemore et al., 1999). It has been proposed that this attenuation of self-produced stimulation occurs because it can be predicted based on efference copy produced in parallel with the motor command by a forward model (Wolpert et al., 1995). The forward model compares the predicted and actual sensory consequences of an action, and if the comparison shows a close match, the stimulation is classified as self-produced and is attenuated. External sensorn stimulation is not attenuated because it is not predicted by the forward model.

Psychiatric patients with auditory hallucinations and/or delusions of control, in which self-generated thoughts and actions are misclassified as external events, do not show the normal attenuation of self-generated touch (Blakemore et al., 2000). One possibility is that people with AS, like patients with auditory hallucinations and/or delusions of control, would not show the normal attenuation of self-generated touch. However, this was not supported by the results of the current study, in which both AS and NC groups showed a significant reduction in tickleness ratings for self-produced relative to externally produced tactile stimulation. This suggests that the forward model prediction and comparison process is functioning normally in people with AS, and highlights a difference between schizophrenia and autism.

Although the AS group in Experiment 2 showed the normal attenuation of self-generated touch, the results demonstrated hypersensitivity in AS for both external and self-generated stimulation. The AS group rated the tactile stimuli as significantly more tickly than did the control group in both conditions, and as more intense in the self-produced condition. These results support the results of Experiment 1, which demonstrate hypersensitivity (in terms of lowered perceptual thresholds) to high-frequency tactile stimulation, which activate Pacinian corpuscles, in AS.

9. General discussion

The current study, to our knowledge, is the first empirical investigation of tactile perception thresholds and tickle sensations in AS. While the present study has a number of limitations, such as a relatively small number of participants, and an unequal gender ratio, we believe that the finding is worthy of systematic exploration in future studies. These studies should include further clinical groups to establish the specificity of the phenomenon to autism. They should also include stimulation to different types of skin at different parts of the body to investigate possible peripheral contributions to hypersensitivity.

In expectation of these further studies, however, we make some preliminary comments on the frequency specificity of tactile hypersensitivity in AS. Our findings agree
well with anecdotal reports focussing on higher-frequency sensations, but it remains difficult to explain why the hypersensitivity is so specific. We think it unlikely that there is a specific abnormality in peripheral sensory receptors such as Pacinian corpuscles. We suggest instead that AS hypersensitivity occurs at some as yet unidentified neural level. Tactile stimuli mechanically stimulate the receptors in the skin and this information is then transmitted via the spinal cord to the thalamus, and on to cortical sensory areas. Tactile information is mapped onto the primary and secondary somatosensory cortex. The primary somatosensory cortex (SI; located in the postcentral gyrus) has a characteristic somatotopic organisation, with the most sensitive parts of the body occupying most cortical territory. Thus, the hand area in the primary somatosensory map is disproportionately large relative to other body parts. The secondary somatosensory cortex (SII), located in the parietal operculum, is responsible for higher level aspects of sensory processing such as tactile orientation, roughness, and stereognosis. Activity in SII is suppressed during self-produced relative to external touch (Blakemore, Wolpert, & Frith, 1998). The hypersensitivity to touch in AS shown in this study may be due to abnormal processing of touch in one or more of these sensory regions.

The somatosensory system has also been divided into a discriminative pathway subserving tactile perception, and an affective pathway subserving emotionally significant touch. Our measure of tactile threshold pertains to the discriminative rather than the affective pathway. Therefore, hypersensitivity to suprathreshold tactile stimuli in people with AS is not merely a matter of excessive dislike of a normal percept, but rather reflects a percept which is itself unusually intense.

Existing cognitive theories of autistic spectrum disorders predict hypersensitivity to all perceptual stimuli, whether this is due to enhanced processing of detailed stimuli (Mottron & Burack, 2001; Plaisted et al., 1998) or due to an impairment in top-down modulation of incoming stimuli (Frith, 2003; U. Frith, 2003). However, the present results together with several previous studies using visual stimuli (e.g., Bertone et al., 2003; Milne et al., 2002; Spencer et al., 2000) point to selective hypersensitivity even within the same modality. In visual motion perception, the complexity of the visual stimulus determines perceptual thresholds in autism (Bertone et al., 2003). Thus, individuals with autism had normal thresholds when detecting simple (first-order) motion, but elevated thresholds when detecting complex (second-order) motion. The parallelism between the idea of a magnocellular visual hypersensitivity in autism and the current results is striking. Magnocellular pathways are high frequency, with transient responses, as are the Pacinian pathways. Thus, the perceptual hypersensitivity may be linked to processing of rapidly changing, dynamic stimuli. Perhaps hypersensitivity in autism and AS can be characterised by increased sensitivity to second order, complex stimuli that require additional integration of information, independent of modality.

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References


