

Sensory perception in autism

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Abstract | Autism is a complex neurodevelopmental condition, and little is known about its neurobiology. Much of autism research has focused on the social, communication and cognitive difficulties associated with the condition. However, the recent revision of the diagnostic criteria for autism has brought another key domain of autistic experience into focus: sensory processing. Here, we review the properties of sensory processing in autism and discuss recent computational and neurobiological insights arising from attention to these behaviours. We argue that sensory traits have important implications for the development of animal and computational models of the condition. Finally, we consider how difficulties in sensory processing may relate to the other domains of behaviour that characterize autism.

Cognitive empathy

The ability to understand and respond appropriately to others' mental states and emotions (unlike affective empathy, the ability to respond with an appropriate emotion to others' mental states or feelings).

The ability to reflect on our own and others' thoughts and emotions (that is, theory of mind) is a defining characteristic of human cognition. Children with autism spectrum conditions (ASCs; henceforth 'autism') show delays in the development of this capacity¹, with knock-on consequences for cognitive empathy² across the lifespan. Interestingly, these alterations in social cognition are accompanied by a very different perceptual experience of the world. Atypical sensory experience is estimated to occur in as many as 90% of autistic individuals^{3,4} and to affect every sensory modality: taste⁵, touch^{6,7}, audition⁸, smell^{9,10} and vision¹¹. A central challenge of autism research is to identify the common thread that unites these various aspects of cognition and sensation. What neurobiological alterations might affect processes as disparate as social cognition and sensory perception?

This challenge is highlighted by the latest international diagnostic criteria for autism, which now include sensory sensitivities as a core diagnostic feature¹². Although sensory symptoms were noted in early reports of the condition¹³, they have historically been construed as secondary aspects of autistic cognition rather than as primary phenotypic markers (see [Supplementary information S1](#) (box)). As well as having clinical implications for creating autism-friendly environments, understanding the importance of sensory differences in autism is crucial for neurobiological accounts of the condition. Because the neural computations underlying sensory processing are relatively well understood in typically developing individuals and are conserved between humans and other animals, studies of sensory behaviour have considerable potential for shedding light on autistic neurobiology¹⁴. Further, as precursors to developmental milestones in social cognition, sensory symptoms could potentially serve as early diagnostic markers.

However, the issue of primacy is key. Is autism, as often posited, a disorder of the 'social brain' (REF. 15), with sensory differences representing either secondary consequences after a lifetime of reduced social interaction or alterations in domain-general mechanisms (such as attention) that affect both social processing and sensory processing? Or are the sensory differences primary in terms of both development and neurobiology?

Here, we explore whether sensory traits are, in fact, core phenotypic markers of autism. To do this, we apply four tests of core phenotypic status, by asking whether autistic sensory traits are present in early development, substantially improve diagnostic accuracy when included in diagnostic assessments, reflect alterations to neural circuitry in sensory-dedicated regions of the brain, and are evident in genetic animal models of the condition.

The evidence we review suggests that the autistic cortex is affected by distinct, low-level changes in neural circuitry that is dedicated to perceptual processing (including primary sensory areas). Further, perceptual symptoms in individuals with autism are evident early in development, account for independent variance in diagnostic criteria of the condition, and show a persistent relationship to clinical measures of higher-order social cognition and behaviour. We suggest that an understanding of the perceptual symptoms in autism may provide insight into signature differences in canonical neural circuitry that might underpin multiple levels of autistic features, and may thus help to elucidate autistic neurobiology. We also discuss how primary sensory changes might relate to higher-order aspects of cognition in autism.

Sensory processing in autism

Sensory symptoms have been clinically documented as early as 6 months of age in infants later diagnosed with autism^{16,17} — considerably earlier than children reach key

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developmental milestones in social cognition, such as joint attention (14–18 months)¹⁸. Sensory symptoms not only precede¹⁷ but also are predictive of social-communication deficits¹⁹ and repetitive behaviours in childhood²⁰, as well as eventual diagnostic status¹⁹. Assessments of sensory traits in the broader autism phenotype suggest a genetic component to these symptoms: the parents and siblings of individuals with autism show higher levels of self-reported sensory traits relative to the general population^{21,22}. Importantly, greater atypicalities in sensory processing are observed in families that are thought to have higher genetic liability for autism (multiplex families) than in families with a single individual diagnosed with autism (simplex families), in which the genetic basis of autism is likely to be *de novo*²¹.

Taken together, these findings suggest that such traits represent early markers of autism. Yet, are these traits primary, or do they simply reflect secondary outcomes of alterations in domain-general neural mechanisms, such as attention? In this section, we briefly review laboratory-based characterizations of autistic sensory behaviour, drawing particular attention to replicated findings in the literature (for in-depth reviews, see REFS 11,23–27), before approaching this question.

Visual detection

Individuals with autism have been characterized as ‘seeing the trees, but not the forest’: attuned to details of the perceptual world at the expense of the global percept they compose²⁸. This framework for understanding autistic sensory experience emphasizes that perceptual processing cannot simply be characterized as a talent or a deficit²⁹ or as reflecting hypersensitivity or hyposensitivity. Rather, perceptual representation in autism exhibits a relative bias towards local over global features of a sensory scene, which can be more or less advantageous depending on task demands³⁰.

This detail-focused perceptual style is well captured by two studies of autistic visual behaviour (FIG. 1). First, individuals with autism often show faster detection of single details (targets) embedded in cluttered visual displays (that is, among distractors) and a relative insensitivity to the number of distractors in the display³¹. This visual search superiority in autism has been widely replicated^{31–37} and extended as a promising early marker in toddlers through eye-tracking^{38,39}. Second, machine-learning approaches have shown that gaze patterns from individuals with autism during passive viewing of naturalistic, complex scenes favour scene regions that rank high in pixel-level saliency (for example, regions that are salient in terms of contrast, colour or orientation) compared with object-level saliency (for example, relating to the size, density or contour complexity of objects) or semantic-level saliency (for example, of text, tools or faces), which drive gaze biases in neurotypical individuals⁴⁰. This data-driven approach provides a compelling demonstration of detail-focused visual preferences in autism, even in the context of naturalistic viewing.

One prediction from these demonstrations would be that individuals with autism might have superior detection or discrimination thresholds for static stimuli⁴¹.

However, perplexingly, basic measures of visual sensitivity such as visual acuity^{37,42}, contrast discrimination^{43,44}, orientation processing, crowding^{45,46} and flicker detection^{47,48} have all been shown to be typical in autism, leaving unresolved the question of how the autistic brain gives rise to rapid and accurate perception of detail. There are some replicated atypicalities in low-level visual processing in autism, particularly in the domain of high-spatial-frequency stimuli^{49,50}, but these are unlikely to account for the full magnitude of autistic superiority in visual search, where stimuli are not necessarily of high spatial frequency.

Temporal synthesis of sensory signals

If basic visual detection thresholds for static, local stimuli are typical in autism, why do individuals with autism display altered local–global processing? One possibility is that perceptual processing in autism may be marked not by an overall bias towards enhanced local perception but rather by a shift in the temporal pattern of local–global processing towards slower global processing⁵¹. This may particularly affect dynamic visual representations, which are by their nature built up over time. This hypothesis rests on evidence from research suggesting that temporal processing of local sensory signals is slower and/or noisier in individuals with autism in the domains of visual, tactile, auditory and multisensory processing.

Visual motion processing. Unlike with static stimuli, individuals with autism often exhibit atypical processing of dynamic (social or non-social) visual stimuli^{52–54} (FIG. 1). Although detection thresholds for local motion are typical⁵⁵ or even superior in autism^{56,57}, individuals with autism often struggle with global motion perception: that is, the ability to discern the global direction (for example, rightward or leftward motion) of a ‘cloud’ of local visual motion signals (for instance, moving dots)^{58,59}. These deficits are predictive of the severity of higher-order autistic symptoms^{58,59} and are particularly pronounced when the motion signal is weak or the time to integrate is short^{58,59}, suggesting that global motion processing in autism is not disrupted *per se* but evolves more slowly over time.

Tactile perception. As in the visual domain, evidence for alterations in basic tactile detection thresholds in autism is mixed — with some studies finding typical^{60,61} and others reporting superior⁶² or reduced sensitivity compared with controls⁷ — although the tactile paradigms used in these studies vary. One difference in autistic tactile perception is well replicated: whereas control individuals present worse detection thresholds for stimuli that gradually increase in amplitude over time into a detectable range (reflective of dynamic thresholds) relative to acute stimuli (which require static thresholds), dynamic presentation does not impair tactile sensitivity in individuals with autism^{7,63}. This difference is proposed to stem from reduced feedforward inhibition in the autistic sensory cortex^{7,64}, consistent with magnetoencephalography findings⁶⁵, and again suggests alterations in the temporal features of sensory processing in autism.

Joint attention

An early-developing cornerstone of social cognition: the child’s ability to use another person’s gestures and gaze to direct his or her attention to objects or events in the environment.

Broader autism phenotype

Mild autistic traits (in both social and sensory processing domains) often observed in relatives of individuals with autism in multiplex families.

Multiplex families

Families in which multiple individuals have an autism diagnosis; family members may carry shared genetic risk factors.

Crowding

The breakdown of visual recognition of peripheral stimuli in cluttered visual environments.

a Individuals with autism show higher pixel-level saliency



Where do you naturally look in a scene?

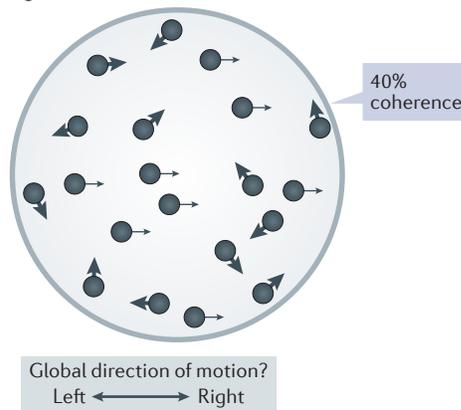
Pixel level
(e.g. colour, intensity, orientations)

Object level
(e.g. size, solidity, convexity, eccentricity of objects)

Semantic level
(e.g. tactile contact between people and objects, actions, text, faces)



b Individuals with autism show atypical perception of global motion



c Individuals with autism show weaker binocular rivalry

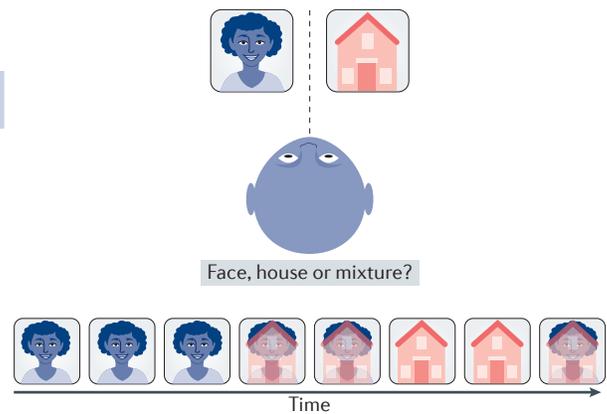


Figure 1 | Trade-off in visual perception in autism. **a** | In naturalistic viewing, gaze patterns of individuals with autism reveal greater preferences for scene regions with high pixel-level saliency (for example, regions of high contrast, colour or orientation) at the expense of regions rich in semantic-level saliency (for example, regions including tactile contact between people and objects, actions, text and faces)⁴⁰. The photograph has been modified as an example to highlight these various levels of image features. **b** | In dynamic visual displays, individuals with autism require longer presentation times and higher signal-to-noise ratios to determine the general direction of dynamic stimuli (in the example, a set of dots moving generally to the right with 40% coherence)^{53,59}. **c** | Individuals with autism show weaker binocular rivalry. Here, two images, one presented to each of an individual's eyes, alternate back and forth in perception as each is suppressed in turn by competitive interactions in visual cortex. In autism, individuals report (via button press) fewer perceptual switches between the inputs to their left and right eyes, as well as a reduced strength of perceptual suppression (when one image is fully suppressed from visual awareness). This replicated behavioural signature of autism in vision is predictive of the severity of social cognition symptoms measured using the Autism Diagnostic Observation Schedule (ADOS)^{127,132,133}.

Auditory perception. Similar disruptions in the temporal envelope of sensory processing have been observed in the domain of auditory processing in autism. Children with autism often show difficulty discerning the relative presentation order of two closely occurring tones⁶⁶ and show delayed evoked neural responses to auditory tones compared with typically developing children^{67,68}. This latency in auditory responses predicts autism symptom severity⁶⁹ and is observed in response to pure tones as well as to complex, social stimuli (such as speech

sounds)⁷⁰, raising the hypothesis that this difference might precipitate higher-order autistic difficulties in communication^{71,72}.

Multisensory binding. Converging evidence suggests a deficit in multisensory integration in autism, both in humans^{69,70,73–78} and in animal models^{76,79}. Specifically, individuals with autism demonstrate an elongated window of audio–visual temporal binding: relative to control individuals, they are less able to discern the presentation

order of a tone and flash at close temporal offsets and are more likely to perceive asynchronous events as synchronous^{74,80}. Further, whereas control individuals are faster to detect a visual stimulus when presented with an auditory tone as opposed to when presented alone, this behavioural benefit is reduced in autism, paralleled by a reduction in multisensory facilitation measured using electroencephalography⁸¹. Deficits in multisensory binding are particularly observed with audiovisual speech paradigms^{74,80,82,83} and may be developmental cornerstones of deficits in language and communication⁸⁴ (see below).

A temporal processing problem? In sum, altered temporal processing of sensory stimuli is seen in several sensory modalities in autism. Specifically, in autism, local stimuli often elicit delayed evoked responses in the auditory domain, and integration of multiple local stimuli into a global percept often requires a wider window of temporal binding. These differences may particularly tax multisensory processing, in which stimuli must be integrated from two sensory modalities⁸⁵, and dynamic perception, in which signals are built up over time.

Yet are these processing differences actually differences in sensation, or could they result from atypical modulation of sensory processing by higher-order cognitive mechanisms? For example, superiority during conjunctive search could arise from differences in parallel processing⁸⁶, deficits in judging global motion in two-alternative forced-choice tasks could arise from altered decision criteria⁸⁷, and reductions in multisensory binding could arise from differences in the cognitive mechanisms involved in drawing causal inferences⁸⁸. In the next section, we discuss neuroimaging findings that demonstrate differences in the low-level primary sensory areas of the autistic brain.

Neuroimaging evidence

Consistent with the psychophysical evidence indicating a low-processing-level origin of the local–global perceptual style in autism, neuroimaging evidence strongly suggests that autistic sensory traits are indeed low-level in origin (FIG. 2). Atypical responses in primary sensory cortices have been observed in autism, across sensory modalities and during multimodal perception.

Global-motion perception tasks (FIG. 1b) involve both sensory and decision-making processes and have therefore been particularly useful in determining whether autistic perceptual differences are truly sensory in origin^{89–91}. The slower integration of local motion signals into a global percept observed in autism^{58,59,92,93} (discussed above) could be caused either by an atypical representation of local motion signals in early visual cortex (in the primary visual area (V1) and the primary motion area (MT)) or by alterations in the decision criteria by which these signals are integrated (in the intraparietal sulcus (IPS)) over time into a global percept⁵⁸. Functional MRI (fMRI) studies have revealed that whereas the IPS response is typical in autism in these tasks, V1 and MT show reduced responses to low-strength motion signals (that is, with short durations and/or low coherence) in autism — presumably limiting the rate at which

motion signals can be integrated into a global percept at higher-order processing stages⁵⁹. Atypical V1 and MT responses in autism have been observed in several motion-processing studies^{94–97}, although whether they can account for deficits in perceiving biological motion in the condition, or simply contribute to differences in processing non-social global motion stimuli, is debated⁹⁸.

In further support of a low-level origin of autistic sensory differences, a robust signature of autistic sensory cortices is an increase in the inter-trial (within-individual) variability of evoked responses^{99–101} (FIG. 2b). This replicated difference affects the visual, somatosensory and auditory cortices of individuals with autism (with some exceptions¹⁰²) and differentiates people with autism from individuals with schizophrenia¹⁰³ (BOX 1). This finding may reflect a disruption of the excitatory–inhibitory balance (E–I balance), which typically modulates the trial-by-trial reliability of evoked sensory responses, in the autistic cortex¹⁰⁴. Alterations in the functional architecture of sensory cortex have been observed as well: larger population receptive fields have been measured in extrastriate regions of the autistic visual cortex, including MT, and these co-vary with autistic traits¹⁰⁵ (FIG. 2b). Another persistent finding in neuroimaging studies of autism is unexpected cross-activation of visual cortex during auditory tasks⁷⁷ — potentially reflecting auditory–visual synaesthesia, which is more common in people with autism than in the general population¹⁰⁶.

Together, these findings indicate that neural signatures of autism are evident in early sensory processing — as early as in primary sensory regions of the autistic brain. Granted, attention modulates neural responses in these early sensory regions^{107,108}; thus, it is difficult to attribute group differences in primary sensory areas to local changes in sensory signalling rather than to top-down attentional modulation, especially given that direct manipulations of attentional load are lacking in the fMRI studies described above. However, neuroanatomical changes in low-level primary sensory regions of the autistic brain suggest local alterations in the circuitry of sensory cortex. For example, cortical minicolumns are reported to be wider in both the primary auditory cortex and higher-order association areas in autism¹⁰⁹ (but see REF. 110). Moreover, behavioural studies suggest that atypical attentional deployment is unlikely to explain the detail-focused visual perception in autism: although people with autism show sharper enhancement of visual performance around a cued location than do control individuals^{111,112} and have difficulties tracking multiple moving objects regardless of object speed¹¹³, these individuals show typical measures of visual performance at the peak of a cued location^{46,114}.

Overall, this pattern of findings is compatible with the hypothesis that sensory differences are core phenotypic markers of autism. Higher-order neural processes that govern how sensory representations are modified by attention, integrated towards decision criteria, or influenced by task demands and expectation may also be altered in autism. However, given the evidence for alterations in primary sensory cortex during perceptual processing in autism, higher-order differences alone are unlikely to account for the perceptual experience of

Population receptive fields

A model-driven quantitative measurement of the average size and shape of receptive fields contained within a single functional MRI voxel.

Cross-activation

Activation of one sensory-dedicated cortical region by sensory stimulation of another modality.

Synaesthesia

The cross-activation of one sensory modality by stimulation of another.

Cortical minicolumns

Basic anatomical units of the neocortex, in which neurons are arranged in vertical columns across cortical layers of the brain.

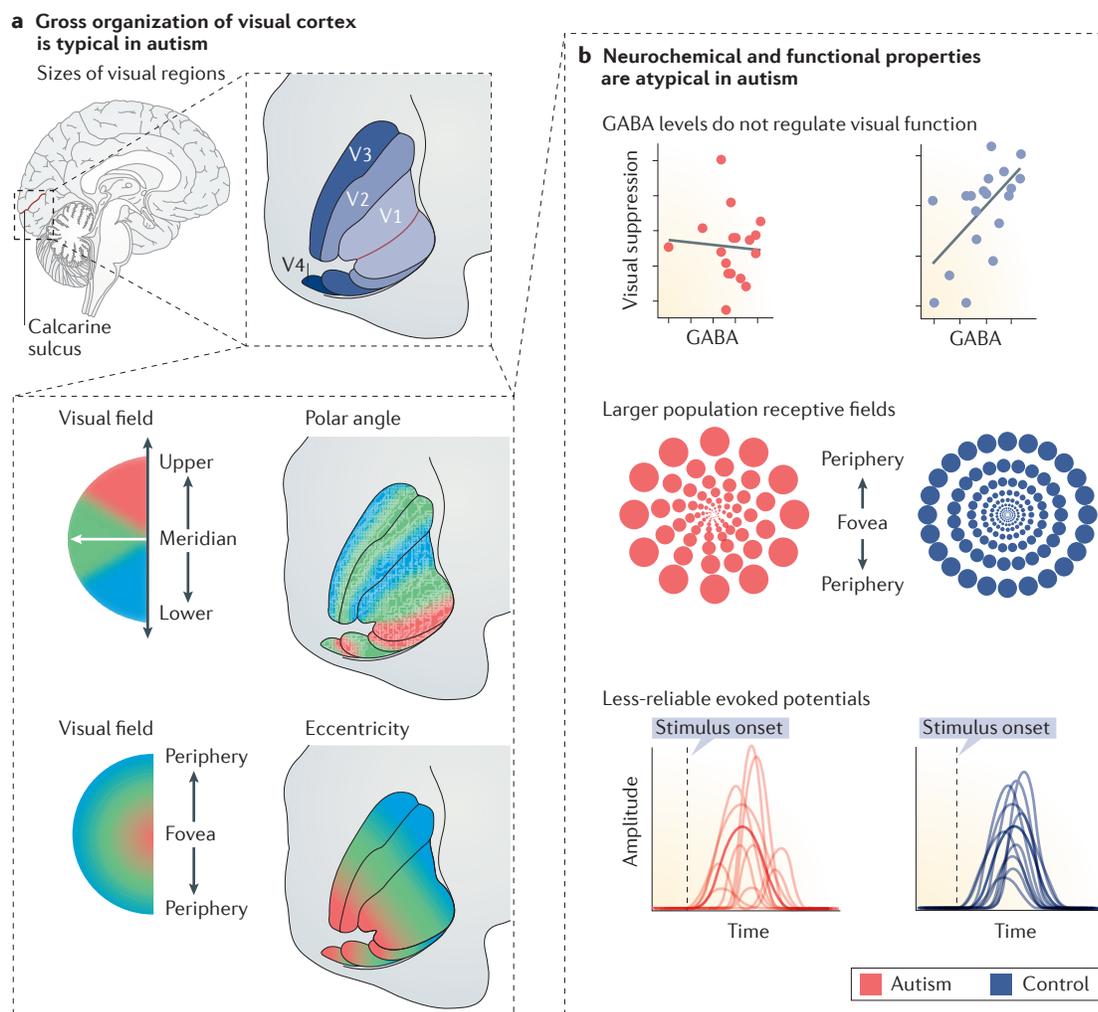


Figure 2 | **Neuroimaging evidence for low-level origin of visual symptoms in autism.** Atypical representations in primary sensory areas have been observed in autism in different sensory modalities. **a** | In the visual cortex, the gross organizational properties of visual areas are typical in terms of the surface area devoted to each early visual cortical region (V1, V2, V3 and V4); the cortical magnification function (that is, the ratio in the cortical area dedicated to foveal versus peripheral representations; not shown); and retinotopic maps, the cortical area dedicated to each part of the visual field, assessed in terms of polar angle (upper and lower visual fields) or eccentricity (distance from the fovea)¹⁰⁵. **b** | However, distinct changes in the neurochemical composition, functional architecture and signalling fidelity of early visual cortex are observed in autism. Specifically, magnetic resonance spectroscopy (MRS) measurements implicate GABA in visual suppression deficits in autism¹²⁷. Control individuals evidence a tight linkage between the strength of visual suppression and GABA levels in visual cortex, but this link is absent in autism (upper graphs). Measurements of the size of population receptive fields in the visual cortex find larger population receptive fields in autism¹⁰⁵ (shown schematically in the middle graphs). Cortical responses evoked by sensory stimuli (including moving dots, auditory tones and tactile stimuli), as measured using functional MRI, are less reliable in individuals with autism^{100,103} (schematic responses shown here in the lower two graphs). The upper two graphs in part **b** are adapted with permission from REF. 127, Cell Press/Elsevier.

individuals with autism. With this in mind, we consider some putative alterations in low-level neural circuitry that may characterize sensory regions of the autistic brain.

Circuit-level insights

As the neural mechanisms of sensory perception have been well characterized using electrophysiology and psychophysical approaches, sensory symptoms may offer concrete insights into circuit-level differences in the autistic brain¹⁴. Indeed, our understanding of the neurobiology of autism has undergone many advances owing

to tests of neural circuitry theories of the condition in the domain of sensory perception. Here, we focus in particular on the hypothesis that the autistic sensory cortex might be marked by differences in GABAergic signalling, as this hypothesis has been tested using neuroimaging approaches as well as computational approaches.

Reduced GABAergic signalling

E–I imbalance is posited to be a central characteristic of the neurobiology of autism, inspired in part by the high prevalence of seizures (perhaps as high as 1 in 3 by adolescence)

Binocular rivalry

A visual phenomenon in which two images, presented simultaneously to the two eyes, alternate in perception as neuronal pools coding for each eye's percept compete for perceptual dominance.

Spatial suppression

A visual phenomenon in which motion discrimination is counter-intuitively attenuated at larger, instead of smaller, stimulus sizes, probably owing to suppressive interactions (centre-surround antagonism or inhibitory feedback).

Critical period

A developmental period during which a neural system (such as vision) is particularly plastic and sensitive to environmental influence.

among people with autism¹¹⁵. GABA receptor perturbations have been associated with autism through genetic^{116–121} and histological studies¹²², and GABAergic signalling is disrupted in several different mouse models of autism^{123,124}. The pivotal roles of GABA in canonical cortical computations¹²⁵ and neurodevelopment¹²⁶ indicate that the GABAergic signalling pathway is key to the neurobiology of autism¹².

Magnetic resonance spectroscopy (MRS) studies have linked disruptions in autistic visual processing^{126,127} to GABA concentrations in early visual cortex (including V1). Specifically, binocular rivalry — a basic visual function that depends on the strength of inhibitory interactions in visual cortex^{128–131} — is weaker in autism^{127,132,133}, and this deficit is associated with reduced GABAergic action in early visual cortex¹²⁷ (FIGS 1c,2b). This replicated behavioural signature of autism is also predictive of the severity of social cognition symptoms measured using the Autism Diagnostic Observation Schedule (ADOS)^{127,132}. Two further MRS studies have reported reduced GABA levels in auditory and somatosensory cortex of autistic individuals^{127,134}, suggesting that reduced inhibition may characterize several cortical regions and perhaps underpin several sensory traits in autism.

Several behavioural and neuroimaging findings regarding autistic visual perception have been theoretically linked to altered inhibitory neurotransmission in the brain. Findings of decreased spatial suppression^{134,135}, atypical representations of motion signals^{58,59}, more within-individual variability in evoked responses^{100,136} and expanded population receptive fields^{105,137} each recapitulate the effects of blocking GABAergic sensory signalling in animal studies^{104,138,139}. Yet, which part of the GABAergic pathway might be atypical in autism remains unclear. Mixed evidence implicates the availability of GABA itself¹⁴⁰, the prevalence or integrity of GABA receptors^{141–144}, the polarity of GABAergic action (which shifts from excitatory to inhibitory during the critical period of development)¹⁴⁵, and the density of cortical inhibitory interneurons¹²³.

Moreover, various other neurotransmitters and neuromodulators of GABAergic signalling may have a role in autistic sensory symptoms. For example, given that excitatory and inhibitory signalling typically exhibit homeostatic coupling during sensory development¹⁴⁶ and learning¹⁴⁷, alterations in GABAergic signalling in autism might be expected to be accompanied by alterations in excitatory signalling. Indeed, higher levels of glutamate in blood plasma¹⁴⁸ and higher glutamate

Box 1 | Comparison with other psychiatric conditions

Although much progress has been made in characterizing differences in sensory processing in autism, less is known about which of these differences are unique to autism or are seen in other neurodevelopmental conditions. This point is crucial for the early identification and translational potential of sensory behavioural assays. Survey-based studies have detected higher rates of sensory abnormalities in autism compared with other developmental disabilities, such as Down syndrome²¹⁹. However, these questionnaire-based observations can only measure the magnitude of sensory sensitivities in a condition, rather than the characteristics of sensory processing. Below, we review key empirical findings that highlight similarities and differences in sensory function between autism and other psychiatric conditions. This evidence of patterns of sensory-processing differences in Rett syndrome, schizophrenia and dyslexia that are distinct from those in autism lends support to the notion that specific deficits in autistic sensory behaviour may indeed be able to serve as selective, objective markers of autism.

Rett syndrome

Until the recent revision of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-V)¹², Rett syndrome (RTT) was included under the diagnostic umbrella of the autism spectrum, as individuals with RTT have many phenotypic similarities to individuals on the autism spectrum. However, the sensory profile of individuals with RTT is distinct from that of individuals with autism. Notably, individuals with RTT exhibit differences to control individuals even in basic visual acuity paradigms^{171,220}, whereas similarly basic measures of low-level visual function (including visual acuity, contrast sensitivity and flicker detection) are typical in individuals with autism⁴².

Schizophrenia

Given the evidence for genetic overlap between schizophrenia and autism²²¹, common sensory paradigms have been used to investigate these conditions. Importantly, such paradigms have revealed distinct patterns of sensory behaviour differences in autism and schizophrenia. For example, whereas neural responses evoked by sensory stimuli are more variable in autism¹⁰⁰, individuals with schizophrenia show typical response variance and lower-amplitude evoked responses¹⁰³. Second, although reduced surround suppression is consistently observed in schizophrenia in many perceptual tasks^{222–224}, similar deficits are only seen in autism at low stimulus contrasts¹³⁴. Last, whereas a robust reduction in perceptual suppression during binocular rivalry has been observed in autism^{127,132,133}, the opposite finding — increased perceptual suppression — is reported in schizophrenia²²⁵. Together, these findings illustrate distinct profiles of alterations in sensory processing in people with autism and individuals with schizophrenia.

Dyslexia

Individuals with dyslexia, similar to individuals with autism, often demonstrate deficits in global-motion perception compared with controls²²⁶ and reduced activity in the primary motion area in neuroimaging studies²²⁷. However, evidence suggests that global-motion-processing deficits in dyslexia are secondary to reduced time spent reading, rather than being primary to the condition²²⁸: deficits are not observed when individuals with dyslexia are compared with reading-matched typical controls and are ameliorated by reading training²²⁹.

Divisive normalization

A canonical neural computation in which the activity of a neuron is divided by the total activity of neighbouring neurons to reflect context-dependent responses.

Pre-pulse inhibition

A sensory phenomenon in which the behavioural response to a strong sensory stimulus is dampened by a weak preceding stimulus, probably through feedforward inhibition.

receptor expression¹⁴⁹ have been observed in individuals with autism, although empirical links with autistic symptoms have not yet been reported. Other neuromodulators, such as testosterone and oxytocin, modulate GABAergic signalling^{150–152} and are associated with autistic traits^{153–155}. Future research is needed to establish the role of these, and other, molecules in modulating inhibitory signalling in regions of the autistic brain.

Computational accounts

To date, most circuit-level computational accounts of autism build on a seminal theory that proposed an excitation-dominant imbalance of neurotransmission in the autistic brain¹⁵⁶. On the basis of this theory, two computational models that attempted to recapitulate specific aspects of autistic sensory behaviour support the hypothesis of reduced inhibition relative to excitation in autistic visual circuitry^{157,158}.

In the first account, Vattikuti and Chow¹⁵⁷ demonstrate that an excitation-dominant circuit could simulate reports of less-precise saccadic targeting (dysmetria) and reduced saccadic velocity (hypometria)^{159–162} in autism. The model predicts an increase in recurrent excitatory activity in the autistic cortex. In turn, this increase is predicted to reduce the spatial specificity of the neural population code for a saccadic target (leading to dysmetria)

and to dampen sensitivity to activity from outside the self-excitatory system, therefore decreasing the rate at which saccadic switching between targets can occur (leading to hypometria). A second model¹⁵⁸ implicates reduced inhibition in a specific neural computation in autism. The authors propose that reducing the spatial spread of inhibition during divisive normalization may recapitulate two behavioural results in the autism literature: reduced spatial suppression¹³⁴ and sharper spatial processing¹¹² (but see REF. 14).

Computational approaches draw together disparate findings under a unifying framework that, when informed by circuitry-level models of neural function, may reveal generalizable principles of neural differences in autism. However, a key limitation of such approaches is that they are developed *post hoc* to recapitulate select behavioural deficits and thus risk losing explanatory and predictive power. We recommend that future computational studies of autistic behaviour be coupled with empirical tests of their predictions in novel experimental paradigms.

Overall, converging evidence from neuroimaging, psychophysics and computational modelling supports the long-held hypothesis that altered GABAergic inhibition may underpin visual symptoms in autism. Given the pivotal roles of GABA in canonical cortical computations¹²⁵ and neurodevelopment¹²⁶, future work will need to interrogate whether the neural changes to the circuitry in the visual system also characterize other regions of the autistic cortex.

Box 2 | Genetic animal models of autism

Genetically modified animals represent a powerful tool for discovering circuit-level alterations in autistic neurobiology. The contribution of genetics to autism is well established: autism heritability is as high as 54–88% for monozygotic twins, compared with 10–33% for dizygotic twins^{230,231}, and many genetic risk factors for autism have been identified through copy number variant, genetic-linkage and genome-wide association studies^{232,233}. Notably, gene variants that confer high penetrance for autism occur in a small subset of the autism population — fewer than 2% of individuals with the condition^{234,235} — suggesting that the genetic aetiology of autism is complex and polygenic. Nevertheless, diverse genetic mutations may have converging downstream effects on specific biological pathways²³⁶. Thus, studying neural development in single-gene mutant animal models of autism may shed light on the aetiology of the condition by identifying common neurobiological pathways affected by different autism-associated mutations, along with their contributions to autistic-like traits in animals.

Animal models of human psychiatric conditions are typically held to three standards of validity: construct validity (whereby the condition is caused by the same biological alteration as in humans), face validity (the behaviour of the animal bears a strong resemblance to human behaviour), and predictive validity (the responses to therapy are likely to translate into humans)²³⁷. Genetic models of autism are exemplars of the first of these standards, construct validity, as they model a specific genetic mutation that is found in people with autism.

However, a major challenge for animal research is the lack of face and predictive validity. Behavioural symptoms in animals rarely present a compelling analogue to human experience, in part because most core features associated with autism in humans manifest in social cognitive functions, such as theory of mind or language comprehension, which are arguably human-specific. Behavioural assays in animal models of autism have traditionally focused on analogues for repetitive behaviours and social anxiety, such as marble burying and sociability²³⁷ — traits that are not specifically related to autism but that also manifest in models of obsessive-compulsive disorder and social anxiety.

Translatable behavioural assays in autism research would facilitate the discovery of generalizable principles about neural circuitry that can move from animals to humans. Measures of sensory behaviour represent promising avenues for such translational assays, given the conserved nature of neural computations involved in sensory processing between animals and humans¹⁴.

Translational research

Two lines of research support the notion that investigations into sensory behaviours might provide promising translational tools for autism research (BOX 2; FIG. 3). First, *de novo* mutations associated with autism converge on pathways that influence synaptic connectivity, signalling and plasticity¹⁶³ and therefore would be predicted to affect wide-ranging neural processes such as sensory perception that are not necessarily confined to the ‘social brain’. Second, increasing evidence suggests that sensory traits are present in common genetic models of autism. For example, mice with mutations in *Mecp2*, *Gabrb3*, *Shank3* or *Fmr1* (which encode methyl-CpG-binding protein 2, GABA type A receptor (GABA_AR) subunit-β3, SH3 and multiple ankyrin repeat domains protein 3, and fragile X mental retardation protein 1, respectively) all demonstrate tactile hypersensitivity, as measured in pre-pulse inhibition paradigms^{124,164}. These sensory traits are specifically linked to the loss of GABA_AR-mediated inhibition in both *Mecp2*-mutant mice and *Gabrb3*-mutant mice¹²⁴, suggesting that disrupted GABAergic neurotransmission is a common feature in multiple genetic models of autism^{123,124,165,166} (but see also REF. 167).

Genetic animal models of autism have also been shown to exhibit deficits in multisensory perception. Mice harbouring genetic mutations in *Gad2* (also known as *Gad65*; encoding glutamate decarboxylase 2), *Shank3* or *Mecp2* show reduced electrophysiological signatures of multisensory integration, which are again specifically linked to reduced GABAergic signalling in neural regions implicated in integrating cross-modal input

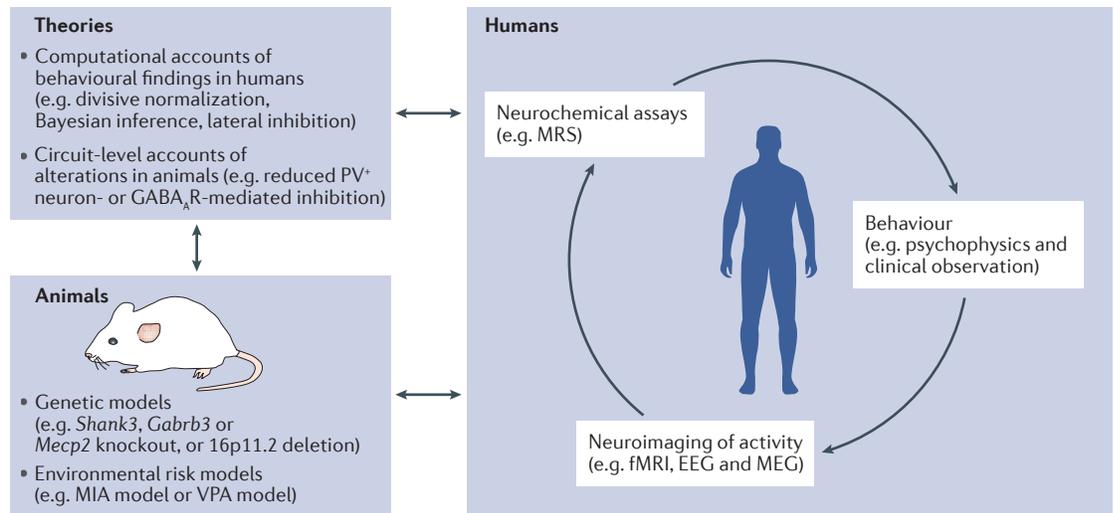


Figure 3 | Sensory symptoms as translational behavioural markers of autism. Given the well-characterized and evolutionarily conserved nature of sensory circuitry in the brain, perceptual symptoms represent a clear tool for translational research. Animal-level research can motivate the design of sensory paradigms in humans and, conversely, aid in delineating alterations in neural circuitry that produce behavioural traits in humans as well as downstream neural targets that merit investigation in humans. When animal-level research implicates a specific neurotransmitter pathway, human neuroimaging methods (magnetic resonance spectroscopy (MRS) or positron emission tomography (PET)) can probe the integrity of this pathway in humans and test whether it underpins behavioural differences. Computational and circuit-level theories of human and animal-level findings can help to uncover principles of neural function that might generalize across sensory paradigms, across sensory modalities and even to other domains of autistic traits. A crucial next step is to test whether genetic models of autism recapitulate the observed human-level behavioural differences using identical sensory paradigms. EEG, electroencephalography; fMRI, functional MRI; GABA_AR, GABA type A receptor; MIA, maternal immune activation; MEG, magnetoencephalography; PV⁺, parvalbumin-expressing; VPA, valproic acid.

(including the insular cortex), and which can be rescued by pharmacologically enhancing GABAergic signalling in early development⁷⁶. These atypical electrophysiological and behavioural markers of sensory processing across multiple monogenic models of autism independently implicate reduced GABAergic inhibition in the aetiology of these features.

Interestingly, one animal model study argues for a feedforward developmental role for sensory deficits in the broader autism phenotype. Specifically, targeted prenatal (but not adult) silencing of GABAergic inhibition in peripheral somatosensory neurons, sparing the CNS, produced tactile sensitivities and social interaction deficits in mice¹²⁴. Importantly, this finding suggests that sensory symptoms in development alone may be sufficient to produce social deficits. Further work is needed to isolate the subcircuits by which this GABAergic deficit in somatosensory pathways led to these social-processing deficits, as well as the relevance of these findings to humans.

Despite these parallel findings in human and animal studies, a lack of directly comparable behavioural paradigms currently limits the potential of studies of sensory behaviour to serve translational research (BOX 2). One naive assumption in animal research is that sensory perception in autism can be ubiquitously characterized as ‘hypersensitive’ — a generalization that, as we have seen, does not capture the nuances of sensory behaviour in people with autism. Standard tests of ‘hypersensitivity’

used in the animal literature, such as startle response or pre-pulse inhibition, show mixed results in people with autism^{168–170}. Thus, we suggest that a standardized set of sensory paradigms that demonstrate clear psychophysical differences in humans and that are suitable for translation into animals would enable validation of animal models of autism and further research into the neurobiology of the condition.

An elegant example of work that paves such a clear translational path can be found in the Rett syndrome (RTT) literature. Here, neural and behavioural sensory phenotypes established in animal and human models strongly correspond: *Mecp2*-knockout mice and individuals with RTT both exhibit a reduction in the amplitude of visually evoked responses in V1 and reduced spatial visual acuity^{165,171}. Interestingly, in the case of the *Mecp2*-knockout mice, these differences stem from a reduction in the activity of GABAergic parvalbumin-expressing interneurons, which affects both excitatory and inhibitory responses¹⁶⁵. Current candidate paradigms and measures for translational autism research may include binocular rivalry^{127,132,133}, multisensory perception^{69,70,73–78} and latency of auditory responses^{67,68} (FIG. 3).

Linking with higher-order traits

The co-occurrence of autistic differences in both low-level perceptual behaviour and high-level social-cognitive processing is a central puzzle of autism research. One clue

McGurk effect

A perceptual illusion in which a sound (for example, of the syllable /ba/) paired with a visual signal (a mouth pronouncing /ga/) produces a third percept (voice and mouth /da/).

Pragmatics

The ability to use the social context of an utterance to inform and communicate meaning.

is that perceptual differences in individuals with autism often predict the severity of higher-order autistic traits^{36,38,52,69,105,112,127,172–177} in the laboratory setting. Large-scale studies also demonstrate covariance between questionnaire-based measures of sensory sensitivities and autistic traits in the general population^{11,177,178}, in both Western and Japanese cultures¹⁷⁹. This correlation presents a strong argument for a relationship between sensory and social-cognitive processing in autism (BOX 3).

Neurobiological accounts of how and why these lower- and higher-order symptoms might be related in autism are largely divided into two camps. ‘Sensory-first’ accounts posit that social-cognitive symptoms may be downstream effects of atypical sensory processing in early development, whereas ‘top-down’ accounts posit that symptoms in sensation and social cognition might co-arise from alterations in domain-general mechanisms (such as attention, decision-making or causal inference) that affect both levels of information processing in the brain.

In this section, we discuss evidence for and against these accounts. There are, of course, many theories of autism that offer elegant accounts of one domain — but not multiple domains — of autistic features^{11,41,180,181}; however, here we focus only on theories that offer a unifying account of diverse domains of autistic behaviour. Finally, we highlight a third approach, which we call the ‘canonical micro-circuitry view’, that posits that disparate levels of autistic features share common neural mechanisms.

Box 3 | Time to give up on a unified account of autism?

Some have argued that it is time to give up on a centralized account of autistic traits and that perhaps the disparate categories of autistic symptoms each have independent genetic causes and neural origins²³⁸. This argument largely rests on studies of autistic personality traits in large normative twin samples (including more than 3,000 twin pairs), which suggest that the degree of parent-reported autistic-like trait severity in social, communicative and repetitive behaviours are only modestly genetically related in typically developing children^{239–241}. Further, autistic-like trait severity in typically developing children sometimes ‘peaks’ in single trait areas: it is estimated that 10% of children show autistic traits in only one symptom domain²³⁸.

By contrast, studies of individuals with autism suggest stronger genetic overlap between autistic symptom domains. One small twin sample of autistic individuals found that common genetic factors represent the primary drivers of both social-communication symptoms and repetitive behaviours, with high heritability²⁴². Furthermore, if autistic symptom domains are indeed fractionable, it remains perplexing that autistic perceptual symptoms often strongly co-vary with social-cognitive symptoms both at the population level^{175–178,214} and in the laboratory^{36,38,52,105,112,127,172–174}, as well as with clinical assessments of repetitive behaviours^{20,243,244}.

Resolving the question of whether autistic behavioural domains are indeed fractionable will require overcoming three limitations of past studies. First, strong phenotypic measures of autistic behaviour across symptom domains that can be adapted for large-scale studies are needed to directly probe the relationship between symptom domains, rather than relying on measures of parental report, which show only modest test–retest reliability²⁴⁰. Second, genotyping individuals with autism may provide more clarity regarding genetic factors shared by different autistic symptom domains than twin studies afford²⁴⁵. Last, sensory behaviours should be assessed in genetic studies of the autistic phenotype, as they are now included in the diagnostic criteria of autism and show clear experimental links to social and communicative traits¹².

In the meantime, we suggest it may be premature to give up on the hypothesis that symptoms of autism in different domains spring from common neurobiological and genetic origins.

Sensory-first accounts

Sensory-first accounts, which are motivated in part by studies of sensory deprivation during child institutionalization^{182,183}, hold that atypical sensory processing during early development causally stunts typical development of social cognition, in a feedforward manner. After all, dynamic sensory information is the medium of social communication: subtle fluctuations in the pitch of spoken language cue prosody, coordinated motions of the face communicate emotions and cues relevant to empathy¹⁸⁴, and the preparatory motions of a person’s body relative to other objects in the world communicate intentions and requests¹⁸⁵. Thus, a child who struggles to integrate dynamic sensory information may also struggle to build social information into meaningful representations or, alternatively, may find social information confusing and therefore self-select away from exposure or engagement with social information^{186,187}. As discussed above, this hypothesis seems consistent with recent findings in animals¹²⁴.

Elegant research on the relationship between multisensory binding deficits and language processing in autism supports such a feedforward causal link. The ability to perceptually bind sensory signals across auditory and visual senses is fundamental to language perception, as it facilitates the integration of vocal and facial cues¹⁸⁸. As discussed above, individuals with autism often show reduced multisensory binding⁶⁶, particularly for social stimuli (such as faces and voices)^{72,83}. Furthermore, altered audiovisual binding thresholds in autism predict less-robust integration of visual and auditory signals of spoken language in tests of the McGurk effect¹⁸⁹ as well as a lower ability to accurately perceive speech in a noisy auditory environment⁸⁴. These studies clearly illustrate how differences in basic sensory processing might affect the development of higher-order functions such as language perception.

Sensory-first accounts have strong merits but also shortcomings. First, among verbal individuals with autism, differences in language processing compared with that in neurotypical controls particularly peak in the domain of pragmatics¹⁹⁰, but why sensory differences would particularly affect this feature is not clear. Second, the neural basis of theory of mind is comparable in blind and sighted individuals, suggesting its development does not depend on typical sensory experience¹⁹¹. Thus, although sensory-first views may account for difficulties in certain aspects of language development, it is difficult to explain top-level autistic deficits in theory of mind from cascading difficulties in building stable sensory representations.

Top-down accounts

Top-down accounts posit that a centralized deficit in domain-general cognitive processes (such as attention, decision-making or causal inference) underpins deficits in both sensory and social-cognitive processing in autism. One instantiation of this theory is the ‘weak central coherence’ hypothesis, which posits that autistic neurobiology is characterized by a centralized perturbation of neural processes that aggregate information

(sensory or cognitive) into coherent percepts or cognitions¹⁹². In this account, sensory signalling is presumed to be unaffected in the autistic brain; rather, a higher-order mechanism that integrates these representations is altered. A neurobiological realization of such a theory would be, for example, deficits in association areas of the brain, where multimodal sensory representations are integrated with task demands.

However, top-down accounts such as the weak central coherence hypothesis are not immediately compatible with the empirical findings of alterations in low-level sensory cortex discussed above. It is particularly challenging to use top-down accounts to explain the neuroanatomical observations of altered minicolumn architecture in not only the association cortices but also the primary auditory cortex of post-mortem autistic brains¹⁰⁹. It is therefore unlikely that centralized cognitive accounts will be able to provide a unifying factor for autistic symptoms in sensory and higher-order cognition.

Canonical micro-circuitry view

We turn now to a third hypothesis, which we call the canonical micro-circuitry view. This view is largely inspired by genetic studies in autism that implicate changes in synaptic connectivity, signalling and plasticity in the condition¹⁶³. Such low-level changes would not necessarily be confined to particular cortical regions (such as the ‘social brain’) but would be predicted to affect basic components of neural circuits throughout the brain¹⁶³. Given that many cortical regions share neural motifs^{14,193} that participate in common canonical computations, genetic disruption of neural motifs might affect many regions of the brain and produce structurally similar behavioural traits in various perceptual and cognitive domains¹⁹⁴.

In addition to divisive normalization (mentioned above), another candidate neural motif-mediated computation that has recently been implicated in autism is Bayesian perceptual inference. Bayesian inference has been shown to be implemented in every neural domain, including sensory perception¹⁹⁵, motor planning¹⁹⁶, language¹⁹⁷, social cognition¹⁹⁸ and proprioception¹⁹⁹. Individuals with autism have been posited to have perceptual representations in which bottom-up sensory input is weighted more than top-down predictions¹⁸¹. Other theories challenge this hypothesis, holding that autistic perception could instead be characterized by imprecise sensory representations²⁰⁰, aberrant weighting of sensory prediction errors²⁰¹ or aberrant updating of priors²⁰². Compellingly, one recent study shows a reduced reliance on implicitly learned priors when discriminating sensory representations in a volatile environment, an effect that is reflected in reduced measures of surprise, as derived from pupil dilation, in individuals with autism²⁰³. Further empirical studies of autistic behaviour are needed to disentangle these hypotheses and to specify the levels of cortical processing at which Bayesian inference might be altered in autism. However, the rubric of Bayesian inference presents the opportunity to test whether systematic failures of a common

computational principle might account for different domains of autistic symptomatology. For example, can weaker priors aptly describe autistic performance on sensory, pragmatic-language and social-cognitive tasks?

How might we go about identifying altered neural motifs in autism? We suggest that this is a two-part endeavour that involves both human and animal model research. In human research, we might start by identifying behavioural paradigms in which similar differences in autism can be observed across different domains of processing (for example, in perception, language and cognition) and that might therefore engage a common neural motif. One example of such a task might be ambiguity resolution. In visual perception, individuals with autism are slower than controls to resolve low-level perceptual ambiguity of two conflicting inputs presented to the two eyes (binocular rivalry)^{127,132,133}. Similarly, in pragmatic language, when presented with sentences containing words that could have two meanings (for example, homographs such as ‘bow’ or ‘bass’), children with autism struggle to resolve this ambiguity, failing to use the sentence context to inform their pronunciation and often defaulting to the more common pronunciation^{204,205}. Ambiguity resolution in both domains of representation — in visual perception and in language — may plausibly rely on neural motifs consisting of reciprocal inhibitory competitive interactions between neural populations that vie for perceptual representation^{206,207}.

Such a motif may even be a neural substrate of theory-of-mind challenges in autism¹: during theory-of-mind judgements, the child must co-activate and flexibly alternate between two, sometimes conflicting, representations of the world — their own understanding and another person’s — to navigate social interactions. Interestingly, the ability to resolve ambiguity in perception (during perceptual bistability), in language (homograph understanding) and in social cognition (theory of mind) tends to develop at approximately the fourth year of life^{208,209}. Furthermore, individual differences in the onset of these abilities correlate across domains^{209,210}, suggesting that ambiguity resolution across these processing domains may be linked. Notably, in children with autism, individual differences in perceptual bistability predict theory-of-mind performance and ADOS scores^{127,132,209}.

Once a potential neural motif has been identified in humans, animal model research may help to identify specific disruptions in neural circuitry that underpin this motif (FIG. 3). When animal model research implicates a specific neurotransmitter pathway, human neuroimaging studies using MRS or positron emission tomography can probe the integrity of this pathway in humans and test whether it underpins behavioural differences. Finally, a crucial step will be to test whether genetic animal models of autism recapitulate the observed human-behavioural differences. Given the relative ease of translating sensory behavioural findings between humans and animal models, further research into symptoms of autism in the sensory domain may lead to promising translational opportunities.

Neural motifs

Stereotyped, local neural circuits that are found in multiple regions of the brain and participate in common canonical computations (such as habituation, response normalization or biased competition).

Bayesian perceptual inference

A model of perception in which prior knowledge about a stimulus is combined with noisy, stimulus-evoked sensory signals to infer the percept and generate prediction errors.

Ambiguity resolution

The ability to impose meaning on ambiguous sensory information. Two or more interpretations may be equally viable (as in bistable visual phenomena, such as in binocular rivalry) or can be disambiguated using contextual information.

Looking forward

This Review posits that sensory symptoms are core, primary characteristics of the neurobiology of autism. Specifically, sensory processing differences in autism are visible early in development^{211–213}, as early as infancy¹⁶, and are predictive of diagnostic status later in childhood^{19,38,39}. They predict higher-order deficits in social and cognitive function in adults^{178,214} and explain independent variance in social and communication symptoms in diagnostic assessments²¹⁵. Moreover, autism-associated sensory symptoms reflect alterations in sensory-dedicated neural circuitry^{59,100}, including neuromolecular and anatomical changes in primary sensory regions of the brain^{64,109,127}, rather than secondary consequences of alterations in higher-order cognitive processes. These differences manifest in both humans and genetic animal models of autism, in which GABAergic signalling is often commonly affected^{64,76,123,124,127}, holding promise for translational biomarkers of the condition.

This conclusion marks a revolutionary shift in our conception of autism from its early diagnostic characterizations¹³ and calls into question modern ‘social brain’ theories¹⁵, in which sensory deficits are hypothesized to be epiphenomenal to core deficits in social processing. Moving forward, neurobiological theories of autism must account for atypical processing in both social and sensory domains.

One of the biggest challenges to formulating neurobiological theories of autism has been the persistent difficulty of documenting robust, replicable differences between individuals with autism and controls,

even with simple tests of sensory processing. Given the genetic heterogeneity of the autistic population, one promising contribution of sensory paradigms may be the ability to stratify the autistic population into more homogeneous subgroups of individuals who share common underlying neurobiological alterations, such as on the basis of sensory differences that are associated with certain genetic polymorphisms^{216,217}. Indeed, sensory subtypes are often reported in children with autism in clinical surveys²¹⁸. Identifying and characterizing such subgroups in the laboratory setting will require the analysis of larger samples than are typically used. In the meantime, replications in independent samples of participants and a number of statistical practices must be used to ensure meaningful between-group comparisons, including using nonparametric statistics when data violate assumptions of normality, bootstrapping statistical comparisons to minimize the effects of outliers, matching groups on relevant psychophysical factors, and eye tracking when retinal position is a relevant variable for task performance.

Autism affects every domain of human experience: from sensation and perception to motor behaviour, emotion, communication and cognition. A central challenge of autism research is to understand how these disparate domains might be related. We suggest that research on sensory symptoms may be able to help untangle this complexity, shedding light on circuit-level alterations in the brain that might affect various domains of cortical processing in autism and offering avenues for translational research.

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SUPPLEMENTARY INFORMATION

See online article: S1 (box)

ALL LINKS ARE ACTIVE IN THE ONLINE PDF

Box S1 | How does sensation fit into the history of autism?

1943: First descriptions of autism include sensory features.

Kanner¹ describes atypical sensory behaviours (including heightened sensitivity to noise and touch, attraction to visual patterns and spinning objects, finger-stimming in front of the eyes) in seven of the eleven children with autism whom he observed, but he considered these symptoms to be epiphenomenal to core deficits in interacting with the external world: “intrusion comes from loud noises and moving objects [...] 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” (p. 245).

1947 and 1960–1970: Origins of ‘sensory-first’ theories of autism.

Bergman and Escalona (1947)² hypothesize that sensory sensitivities have an early developmental onset in autism and cause the child to withdraw from the social world, proposing that: “these children start life with a high degree of sensitivity against which they eventually succeed in building some defenses” (p. 345).

Eveloff (1960)³ hypothesizes that altered sensory processing interferes with the development of self-representations in autism: “Briefly, a sense of self apparently depends a good deal on the integration of the body image with properly cathected, well-functioning distance receptors of sound, sight, and smell” (p. 102).

Hermelin and O’Connor (1970)⁴ posit that individuals with autism show an atypical preference for specific sensory modalities over others (e.g. visual and tactile over auditory), perhaps leading to difficulties with language development.

1967: Sensory processing abnormalities are posited as underlying impairments in autism.

Psychologist Lorna Wing notes the “detail-oriented” perceptual style in autism: “attending to minor or trivial aspects of people or objects instead of attending to the whole (p. 50)”. She proposes this feature along with “abnormal responses to sensory experiences (p. 49)” as diagnostic criteria in the first scheme for the clinical description of autism⁵. These features, however, are not included among the first DSM diagnostic criteria for autism in 1980⁶.

1978: Robustness of sensory features in autism is questioned.

Richer⁷ firmly argues that disruptions of sensory processing in autism are red-herrings: sensory processing is not affected in autism and any theory based on sensory processing in autism: “is as incoherent as it is untestable”. Similarly, Rutter⁸ argues that “visuo-spatial perceptual defects do not play any essential role in the development of infantile autism” (p. 91), forwarding the hypothesis language impairment is at the heart of the condition.

1982: Experimental demonstrations of visuospatial talent in autism.

Using the Embedded Figures Test, Shah and Frith⁹ demonstrate measurable visuospatial processing talents in autism, which they posit are the result of a cognitive reduction in the drive for meaning: “Perhaps they [the children with autism] were able to locate the target figure so easily because the

overall meaning of the complex figure (or the embedding context) was not relevant or dominant for them” (p. 618).

1983: Origins of ‘social-perception’ theories of autism.

Rutter¹⁰ concludes that sensory symptoms are byproducts of primary deficits in social cognition: “The data suggest that the deficit does not lie in the processing of stimuli of any particular sensory modality or, indeed, of stimuli that are defined in terms of any particular sensory qualities. Rather, it appears that the stimuli that pose difficulties for autistic children are those that carry emotional or social ‘meaning’” (p. 528).

1987: First cognitive-based theories of sensory symptoms in autism.

Frith and Baron-Cohen¹¹ later posit that: “low-level perceptual processes are intact in autistic children. Instead, [talents in visuospatial processing] indicate a central cognitive deficit as the primary problem area” (p. 99).

2013: Sensory processing are first included among international diagnostic criteria of autism.

The 2013 revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)¹² includes sensory symptoms, specifically “hyper-or hypo-reactivity to sensory input or unusual interest in sensory aspects of environment”, among the diagnostic criteria of autism.

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