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Research Report

Autistic adults show preserved normalisation of sensory responses in gaze processing

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ABSTRACT

Progress in our understanding of autism spectrum disorder (ASD) has recently been sought by characterising how systematic differences in canonical neural computations employed across the sensory cortex might contribute to clinical symptoms in diverse sensory, cognitive, and social domains. A key proposal is that ASD is characterised by reduced *divisive normalisation* of sensory responses. This provides a bridge between genetic and molecular evidence for an increased ratio of cortical excitation to inhibition in ASD and the functional characteristics of sensory coding that are relevant for understanding perception and behaviour. Here we tested this hypothesis in the context of gaze processing (i.e., the perception of other people's direction of gaze), a domain with direct relevance to the core diagnostic features of ASD. We show that reduced divisive normalisation in gaze processing is associated with specific predictions regarding the psychophysical effects of sensory adaptation to gaze direction, and test these predictions in adults with ASD. We report compelling evidence that both divisive normalisation and sensory adaptation occur robustly in adults with ASD in the context of gaze processing. These results have important theoretical implications for defining the types of divisive computations that are likely to be intact or compromised in this condition (e.g., relating to local *vs* distal control of cortical gain). These results are also a strong testament to the typical sensory coding of gaze direction in ASD, despite the atypical responses to others' gaze that are a hallmark feature of this diagnosis.

1. Introduction

Autism spectrum disorder (ASD) is a heterogeneous developmental condition, characterised by differences in social interaction, a strong preference for routine, repetitive motor behaviours, and sensory sensitivities (American Psychiatric Association, 2013; Lai, Lombardo, & Baron-Cohen, 2014). ASD has a strong yet highly complex genetic basis (Geschwind &

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State, 2015), and there is currently no explanation of the condition that bridges biological, cognitive, and behavioural levels of description. Recently, progress has been sought by drawing on general *computational theories of brain function* to characterise how systematic differences in the processing of sensory information may contribute to the sensory and social symptoms of ASD (e.g., Lawson, Rees, & Friston, 2014; Palmer, Lawson, & Hohwy, 2017; Rosenberg, Patterson, & Angelaki, 2015; Van de Cruys et al., 2014). These theories highlight the *control of cortical gain* as a computationally-important neural mechanism that a variety of genetic and molecular differences might converge on.

There is genetic and molecular evidence for an increased ratio of cortical excitation to inhibition in ASD (e.g., Rubenstein & Merzenich, 2003; Yizhar et al., 2011), and computationally, this can be related to the *divisive normalization* of sensory responses (Rosenberg et al., 2015). Divisive normalization occurs when the responses of a sensory neuron are not only driven by stimuli that excite it, but also modulated by the responses of local, functionally-related cell populations (e.g., those with adjacent spatial receptive fields). This is a form of neural gain control that may be instantiated by lateral inhibitory connections in sensory areas of the cortex. It is now well-established that this computation is employed in a widespread manner across sensory systems (Carandini & Heeger, 2012), playing an essential role in maintaining a sensory code that is robust to extraneous, context-dependent variation in neural firing.

Correspondingly, a key proposal is that symptoms in ASD, across sensory, cognitive, and social domains, reflect a widespread *reduction* of divisive normalisation in neural processing (Rosenberg et al., 2015). This hypothesis is attractive in its potential to link our expanding knowledge of the complex biological underpinnings of this condition to functional characteristics of sensory coding, and thereby perception and behaviour. Initial support for this idea comes from simulation analyses that demonstrate that certain low-level visual characteristics in ASD (e.g., weak visual spatial suppression) can feasibly arise through reduced normalisation of sensory responses in primary visual cortex (Rosenberg et al., 2015). Rosenberg and colleagues also argue that the notion of reduced normalisation computations, if a systemic feature of neural processing in ASD, can help to make sense of experimental data across a variety of domains, including local versus global processing, multisensory integration, and decision-making. However, the proposal as a whole largely remains to be tested, including how the proposed differences in sensory processing contribute to the behaviours defining the diagnostic criteria.

In the social domain, recent research has examined the role of divisive normalisation in the sensory coding of others' direction of gaze (Palmer & Clifford, 2017a, 2017b). This has revealed a distinct psychophysical signature of normalisation in neurotypical (NT) individuals, reflected in the fine-grained effects of *sensory adaptation* on subsequent perception of gaze direction. Sensory adaptation occurs when prolonged viewing of a specific direction of gaze (e.g., far leftwards averted gaze) causes a repulsive aftereffect such that subsequently presented faces are seen as looking more rightwards than their veridical direction of gaze. This phenomenon is thought to reflect targeted habituation of stimulus-selective sensory channels, and can be used to probe the underlying sensory coding of perceptual properties like gaze direction (Suzuki, 2005). The adaptive sensory coding of gaze direction is linked to cortical function in higher visual areas, namely anterior superior temporal sulcus (Calder et al., 2007; Carlin & Calder, 2013).

It is appealing to examine the function of divisive normalisation in ASD in the context of gaze perception, because atypical gaze-based behaviours are a cardinal diagnostic feature of ASD. This includes, for instance, a reduced tendency to seek mutual gaze when interacting with others, in both childhood and adulthood. Experimental research in ASD has shown differences in how attention is cued on the basis of others' gaze direction (Frischen, Bayliss, & Tipper, 2007), reduced salience of direct gaze (Senju, 2013), and subtle differences in the sensory coding of others' gaze direction, namely a reduced influence of recent sensory history on current perception (Lawson, Aylward, Roiser, & Rees, 2017; Pellicano, Rhodes, & Calder, 2013). Prominent social-cognitive theories also emphasise the role of eye gaze processing in our ability to make inferences about other people's mental states, issues which are commonly thought to be a core driver of social difficulties in ASD (Baron-Cohen, 1997; Lai et al., 2014).

Here we present a computational simulation analysis demonstrating that reduced divisive normalisation in the context of gaze perception is associated with distinct predictions regarding the psychophysical effects of sensory adaptation to gaze direction. Correspondingly, we compare sensory adaptation to gaze direction between adults with ASD and NT adults. This allows us to (1) empirically test the proposal that ASD is characterised by reduced divisive normalisation of sensory responses (Rosenberg et al., 2015), in a domain pertinent to the social symptoms of this condition, and (2) probe for differences more generally in the functional mechanisms that underlie sensory processing in the cortex, namely the adaptive coding of others' gaze direction across gaze-selective sensory channels. We find compelling evidence that the adaptive coding of others' gaze direction occurs as robustly in adults with ASD as in NT controls, including in the divisive normalisation of sensory responses. These results further our understanding of how information about others' gaze is processed in ASD, and help to adjudicate between recent computational accounts of this condition that emphasise problems in local versus distal gain control in sensory function (Lawson, Friston, & Rees, 2015).

2. Material and methods

2.1. Simulation of reduced normalisation in the sensory coding of gaze direction

2.1.1. Computational model of perceived gaze direction

Electrophysiological studies in macaque monkeys have identified individual cells in temporal cortex sensitive to the gaze direction of a seen face (Perrett, Hietanen, Oram, & Benson, 1992; Perrett et al., 1985). Psychophysical and functional neuroimaging research in humans similarly indicate that perceived gaze direction is coded across distinct neuronal populations tuned to different directions of gaze (e.g., leftwards *vs* rightwards gaze) (Calder, Jenkins, Cassel, & Clifford,