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**Individual Differences in Sensory Sensitivity: A Synthesising Framework and Evidence
from Normal Variation and Developmental Conditions**

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Abstract

For some people, simple sensory stimuli (e.g. noises, patterns) may reliably evoke intense and aversive reactions. This is common in certain clinical groups (e.g. autism) and varies greatly in the neurotypical population. This paper critically evaluates the concept of individual differences in sensory sensitivity, explores its possible underlying neurobiological basis, and presents a roadmap for future research in this area. A distinction is made between subjective sensory sensitivity (self-reported symptoms); neural sensory sensitivity (the degree of neural activity induced by sensory stimuli); and behavioural sensory sensitivity (detection and discrimination of sensory stimuli). Whereas increased subjective and neural sensory sensitivity are assumed to increase together, the status of behavioural sensory sensitivity depends on the extent to which the increased neural activity is linked to signal or noise. A signal detection framework is presented that offers a unifying framework for exploring sensory sensitivity across different conditions. The framework is discussed, in more concrete terms, by linking it to four existing theoretical accounts of atypical sensory sensitivity (not necessarily mutually exclusive): increased excitation-to-inhibition ratio; predictive coding; increased neural noise; and atypical brain connectivity.

Keywords:

Sensory sensitivity; autism; synaesthesia/synaesthesia; migraine; predictive coding; connectivity; neural noise.

The same simple sensory stimulus may reliably evoke different responses in different individuals. For some people, certain sounds (e.g. of chewing) may be reported as extremely unpleasant or the feel of a clothes label may cause endless irritation. For others, repetitive high contrast visual patterns (e.g. ‘op art’) can appear to shimmer or induce feelings of nausea. These kinds of atypical sensory sensitivities have been linked to a wide variety of conditions including migraine (Schwedt, 2013), autism spectrum disorder (Tavassoli, Hoekstra, & Baron-Cohen, 2014), ADHD (Bijlenga, Tjon-Ka-Jie, Schuijers, & Kooij, 2017), Tourette syndrome (Belluscio, Jin, Watters, Lee, & Hallett, 2011), and synaesthesia (Ward et al., 2017). One lady with autism describes her experiences thus: “the sensory overload caused by bright lights, fluorescent lights, colours, and patterns makes the body react as if being attacked or bombarded, resulting in such physical symptoms as headaches, anxiety, panic attacks or aggression” (p. 43 Williams, 1994). These differences in subjective sensory sensitivity also vary greatly within the neurotypical population, to the extent that they can overlap in severity with individuals with a clinical diagnosis (A. E. Robertson & Simmons, 2013).

The aim of this paper is to critically evaluate the concept of individual differences in sensory sensitivity, explore its possible underlying neurobiological basis, and present a roadmap for future research in this area. The focus of the paper is at the symptom level, rather than seeking to offer more general explanations of any one particular condition (e.g. autism). It integrates evidence across multiple conditions and from neurotypical variation, as well as from multiple senses. The aim is not to argue that a single explanation will apply to all conditions linked to atypical sensory sensitivity (although it might). Rather, the aim is to facilitate dialogue between researchers working in different areas, and to develop a common

conceptual framework and common methodologies that enable the similarities and differences across conditions to be better understood.

The starting point is the symptomatology itself, which can be regarded as hyper-sensitivity (i.e. over-responsiveness to sensory stimuli) and/or hypo-sensitivity (i.e. under-responsiveness) and typically manifests itself across different senses. For example, the DSM-V diagnostic criteria for autism states the following: “Hyper- or hypo-reactivity to sensory input or unusual interests in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).” Various questionnaires have been developed to quantify and understand the range of symptoms (Crane, Goddard, & Pring, 2009; Robertson & Simmons, 2013; Tavassoli et al., 2014) and some are translated into other languages (Sapey-Triomphe, Moulin, Sonie, & Schmitz, 2018; Takayama et al., 2014). These are suitable for high functioning adults, and comparable measures exist for children (Dunn, 1999). For instance the Glasgow Sensory Questionnaire (GSQ) asks questions relating to hyper- and hypo-sensitivity from different sensory modalities (Robertson & Simmons, 2013). When administered to a neurotypical population, it was found that all questions tended to load on to a single, common factor. That is, the same people tend to report both hyper- and hypo-sensitivities and they do so across multiple senses. The same pattern, albeit with considerably higher scores, has been found when these measures have been given to special groups including autism and synaesthesia (e.g. Ward et al., 2017). The co-occurrence of hyper- and hypo-sensitivities is a puzzle. This may be because these concepts, particularly hypo-sensitivities, are ill-defined. For instance, repetitive or unusual sensory behaviours are sometimes assumed to reflect hypo-sensitivity but could be explained in other ways (e.g. obsessive motor acts, rather than low sensory reception). Example items including “Do you eat the same foods most of the time?” and “Do

you flick your fingers in front of your eyes?’. Most models focus primarily on atypical hyper-sensitivity and, arguably, this is the symptom that most impacts life quality. This paper will also consider how the various models could account for hypo-sensitivity in addition to hyper-sensitivity.

The fact that multiple sensory modalities are reported to be affected on questionnaire measures of atypical sensory sensitivity is suggestive of a central origin (i.e. brain-level) rather than peripheral origin (e.g. at the level of receptors or ascending nerve fibers). Whether multiple senses are always affected is less clear. A likely exception is photosensitive epilepsy which is related specifically to vision (Porciatti, Bonanni, Fiorentini, & Guerrini, 2000). Other possible exceptions are the newly reported conditions of misophonia (extreme aversion to certain sounds, see Brout et al., 2018) and ‘visual snow’ (tiny dots in visual field often accompanied by light aversion, Schankin, Maniyar, Digre, & Goadsby, 2014). However, there is some evidence that non-auditory modalities are hyper-sensitive in misophonia (Wu, Lewin, Murphy, & Storch, 2014) and tinnitus is prevalent in people with visual snow (Schankin, et al., 2014). Migraine, although frequently studied in terms of visually induced symptoms, has also been noted to be linked to hyper-sensitivity across multiple sensory modalities (Schwedt, 2013). Sensory sensitivity questionnaires developed for other conditions, such as autism, have yet to be run on this group. Modality-specific hyper-sensitivities can, of course, emerge as a result of compensatory neural plasticity in response to acquired sensory loss in one modality. Examples of this include increased subjective sensitivity to sounds as a result of partial hearing loss and tinnitus (termed hyperacusis, Norena, 2011), and enhanced subjective sensitivity to touch following tissue damage (termed allodynia, Woolf, 2011). The present review focusses on stable non-acquired individual differences that likely reflect neurodevelopmental differences.

In the following section, I outline a framework for linking the clinically relevant subjective symptoms to objective neurophysiological and behavioural findings. In the remaining sections, I discuss several specific theoretical models for explaining atypical sensory sensitivity (primarily hyper-sensitivity): namely, increased excitation-to-inhibition; enhanced sensory processing (including predictive coding models); increased neural noise; and atypical brain connectivity.

Individual Differences in Sensory Sensitivity and a Simple Framework in Terms of Neural Signal and Noise

Firstly, it is important to make a distinction between individual differences in subjective sensory sensitivity, behavioural sensory sensitivity, and neural sensory sensitivity. Different parts of the literature tend to focus on only one of these, leading to a confused and inconsistent picture. By definition, clinical symptoms focus on subjective sensory sensitivity: they are first person reports of aversion to bright lights, feelings of ‘sensory overload’, etc. Behavioural sensory sensitivity, on the other hand, refers to the ability to discriminate or detect sensory stimuli. Finally, individual differences in neural sensory sensitivity refer to differences in the magnitude of a neurophysiological response (e.g. BOLD in fMRI, EEG, or spiking rate) to a given sensory stimulus across individuals. These have tended to be the focus for animal models of sensory sensitivity.

How are these three different concepts related to each other? Although there are a wide variety of mechanisms that could contribute towards individual differences in sensory sensitivity, I will suggest that they map on to a relatively small number of basic possibilities that arise from considering perceptual processing in terms of neural signal and neural noise. With regards to the relationship between subjective sensory sensitivity and neural sensory

sensitivity, one pervasive assumption in the literature (reviewed below) is that groups/individuals who are subjectively hyper-sensitive have a greater neural response to those sensory stimuli. Importantly, this would occur irrespective of whether the neural response was signal (i.e. stimulus-relevant) or noise (i.e. stimulus-irrelevant) or both. There is surprisingly little direct evidence for this assumption. Groups known to report subjectively high sensory sensitivity, such as migraine (Coutts, Cooper, Elwell, & Wilkins, 2012) and autism (Schwarzkopf, Anderson, de Haas, White, & Rees, 2014), have a greater BOLD response than neurotypical people to the same sensory stimuli. These studies did not measure subjective sensory sensitivity directly (e.g. using a questionnaire) but other fMRI studies that have done so have shown correlations between self-report and stimulus-induced activity in sensory cortices (e.g. Green et al., 2015; Green et al., 2013). However, little is known about the specific contribution of different brain regions (e.g. striate or extrastriate) or the kind of information (e.g. colour, motion, luminance) that is coded by the neural populations that track individual differences in subjective sensory sensitivity. Moreover, it is possible, given that much neural processing is unconscious, for neural activity in certain regions to go up or down but without these changes contributing to subjective intensity or aversion to sensory stimuli (for discussion of how levels of neural activity don't necessarily track visual awareness see Leopold, 2012). The same concerns can be applied to all current animal models of individual differences in sensory sensitivity, which sidestep subjective sensory sensitivity altogether. However, the assumption that increased neural activity (in at least some populations of neurons) is linked to increased subjective sensory sensitivity remains the working hypothesis of all major current theories. A null hypothesis, although not typically stated in such terms, is that individual differences in subjective sensory sensitivity are not related to either neural or behavioural sensory sensitivity but instead solely reflect post-sensory processing (e.g. idiosyncratic affective responses).

The situation with regards to behavioural sensory sensitivity varies from theory to theory and, here, it does matter crucially whether the increased neural response primarily reflects increased signal (in which case increased behavioural sensitivity goes with increased neural sensory sensitivity and increased subjective sensory sensitivity) or reflects increased noise (in which case decreased behavioural sensitivity tends to go with increased neural and increased subjective sensory sensitivity).

These basic ideas can be formalised in simple equations (from O'Hare & Hibbard, 2016; Zhaoping, 2006) ...

$$O = K(S) + N_a$$

O refers to the output, i.e. the population response of neurons. In the terminology introduced earlier, it is effectively a measure of neural sensory sensitivity (and a likely correlate of subjective sensory sensitivity).

S refers to the signal-based neural response, and K() refers to the encoding function of the signal (i.e. what is done with it). For instance, K() may amplify the signal (e.g. a mechanism referred to as gain control, e.g. Schwartz & Simoncelli, 2001), or determine whether the sensory signal is fed-forward to other neurons or not. To pre-empt a later discussion, according to the predictive coding framework a sensory signal that is unexpected engenders more neural activity (i.e. K(S) is large) than one that is predicted (i.e. K(S) is small). Individuals who have problems in predicting their sensory world would tend to have a larger K(S) and, hence, a larger output response (O) than in individuals who are good at predicting their sensory world.

N_a refers to the level of background noise (spontaneous neural activity). A system that has higher noise levels would generate a higher output response (O). Although noise would tend to reduce behavioural sensory sensitivity there are certain scenarios in which it can enhance it; for instance, if the sensory signal is just below detection/discrimination

threshold then noise can raise it above the threshold. This phenomenon is termed stochastic resonance (McDonnell & Abbott, 2009) and has been proposed as a candidate mechanism for atypical sensory sensitivity in autism (Simmons et al., 2009), as discussed in more detail later.

There is another potential source of noise that has been termed multiplicative noise (e.g. O'Hare & Hibbard, 2016) and can be represented by the addition of N_m to the equation below:

$$O = K(S).(1+N_m) + N_a$$

N_m refers to noise that is only present when a signal is present (and proportional in size to the signal). It could be conceptualised as aberrant propagation of activity from neurons carrying the signal to other neurons that connect to it but are not optimally tuned to that signal. These could be neighbouring neurons within sensory cortex, for instance due to individual difference in excitation-to-inhibition characteristics (e.g. Rubenstein and Merzenich, 2003). It could also lead to propagation of neural activity to non-sensory regions (e.g. amygdala) that reflects, for instance, individual differences in functional/structural connectivity to and from sensory regions. The resulting profile is that sensory processing appears more diffusely distributed. Increases in N_m due to individual differences would increase neural sensory sensitivity (and, by implication, subjective sensory sensitivity) but would not increase behavioural sensory sensitivity. In such cases, it is not clear whether one can isolate the signal component from the noise component (e.g. O'Hare & Hibbard, 2016) but the net effect is a sensory-evoked neural response that is larger and more variable (for methods of measuring the latter see Vilidaite & Baker, 2017).

One of guiding design principles of neural computation is to maximise efficiency in order to reduce the metabolic costs of processing information that is redundant, irrelevant, or predictable (e.g. Atick, 1992). For instance, in natural visual scenes there is a tendency for

any point in space to have similar properties to its neighbours (e.g. colour, luminance), except at edges, and for these properties to extend over time, except when the object moves. Thus a pixel-like representation is metabolically/computationally inefficient because it is typically the case that one part of a spatial and temporal signal can be predicted by others. A sparser system that represents only surfaces, edges, and other changes would be more efficient. Within this computational framework, the basic problem being solved is that of maximising behavioural sensory sensitivity whilst minimizing neural sensory sensitivity (because a high amount of neural responsiveness is metabolically costly). However, different individuals/groups may adopt somewhat different solutions to this problem owing to intrinsic differences in their brain function and/or structure (e.g. connectivity, plasticity, neurotransmitters), with individual differences in subjective sensory sensitivity emerging as a consequence of this balancing act. These are considered in more detail below.

To summarise, although there is no shortage of different mechanisms that could affect the different terms in this equation, this signal processing approach (extended from research on migraine) offers a unifying framework for thinking about individual differences in sensory sensitivity across these mechanisms and across groups. In the sections below, specific models and evidence are summarised in relation to this framework. An overview summary is given in Table 1 of specific models and how they relate to this framework.

INSERT TABLE 1 ABOUT HERE

Theory (examples/s)	Neural Mechanism	Neural sensory sensitivity	Subjective hyper- sensitivity	Behavioural sensory sensitivity	Possible explanations of increased hypo-sensitivity	Link to signal detection
Increased Sensory Sensitivity due to an Increased Excitation-to-Inhibition Ratio (e.g. Rubenstein and Merzenich, 2003)	More glutamate/less GABA leading to propagation of neural activity and less sparse neural representations	Increased	Increased	Decreased	Behaviourally hypo-sensitive despite sensory stimuli being subjectively intense	N _m
Sensory Sensitivity due to Different Balance between Priors and Sensory	Unpredicted sensory stimuli engender more neural activity	Increased	Increased	Increased (mainly)	Failure to adapt to repeated stimuli may render some stimuli hypo-sensitive	K(S)

Inputs (e.g. van der Cruys et al., 2014)						
Increased Sensory Sensitivity due to Differences in Endogenous Noise (e.g. Simmons et al., 2009)	Increased spontaneous neural activity (i.e. in both presence and absence of a sensory stimulus)	Increased	Increased	Decreased (mainly)	Fluctuations in noise can both enhance (hyper-) and reduce (hypo-) sensitivity	N_a
Increased Sensory Sensitivity due to Altered Network Connectivity (e.g. Markram & Markram, 2010)	Increased propagation of neural activity	Increased	Increased	Uncertain (depends on the type of connections)	Uncertain	$N_m?$

Increased Sensory Sensitivity due to an Increased Excitation-to-Inhibition Ratio

Rubenstein and Merzenich (2003) put forward a model of autism based on the notion of an imbalance between excitation and inhibition, such that autism is assumed to reflect either increased excitation and/or reduced inhibition. This may reflect differences in receptors or neural circuits that involve the neurotransmitters glutamate (the main excitatory neurotransmitter in the brain) or GABA (gamma-aminobutyric acid, the main inhibitory neurotransmitter in the brain). Evidence in general support of this account comes from: the high prevalence of epilepsy and seizures in people with autism (Tuchman & Rapin, 2002), genetic variations within the GABA system producing autistic symptoms (e.g. see Coghlan et al., 2012), and animal models of autism that manipulate GABA or glutamate (e.g. Han et al., 2012). Although developed with autism in mind, the general assumptions of the model are relevant for thinking about individual differences in sensory sensitivity more generally.

With regards to linking this brain-based mechanism to cognitive functions, Rubenstein and Merzenich (2003) argued that an increased ratio of excitation to inhibition disrupts functional differentiation during development. Specifically, more positive coupling of neuronal responses leads to less specific tuning to properties in the environment, i.e. a less sparse neural representation. The latter essentially corresponds to increased multiplicative noise (N_m) in the equation above. In terms of neural architecture, neocortical function is organised into mini-columns which consist of both excitatory and inhibitory neurons that respond within the mini-column to related stimuli, but with GABA-mediated lateral inhibition between mini-columns that serve to ‘sharpen’ the neural response (e.g. Isaacson & Scanziani, 2011). Rubenstein and Merzenich (2003) argue that increasing this ratio (i.e. more

glutamate and/or less GABA) would result in a sensory stimulus eliciting a stronger, but more noisy/variable, neural response (hyper-sensitive at the neural level) because it is less sharp and less sparse. This increased ratio gives rise to cognitive dysfunction, including sensory hyper-sensitivity (at neural level and presumably in terms of subjective sensory sensitivity). However, neural responses that are more noisy would lead to less behavioural sensory sensitivity (i.e. worse discrimination of sensory signals). This also offers one example how hyper-sensitivity (increased subjective intensity) and hypo-sensitivity (worse sensory detection/discrimination) can co-occur without contradiction. Figure 1 shows an example of different patterns of neural responsiveness corresponding to sparse and diffuse sensory signals (and also the possibility, discussed later, of increased background noise).

INSERT FIGURE 1 ABOUT HERE

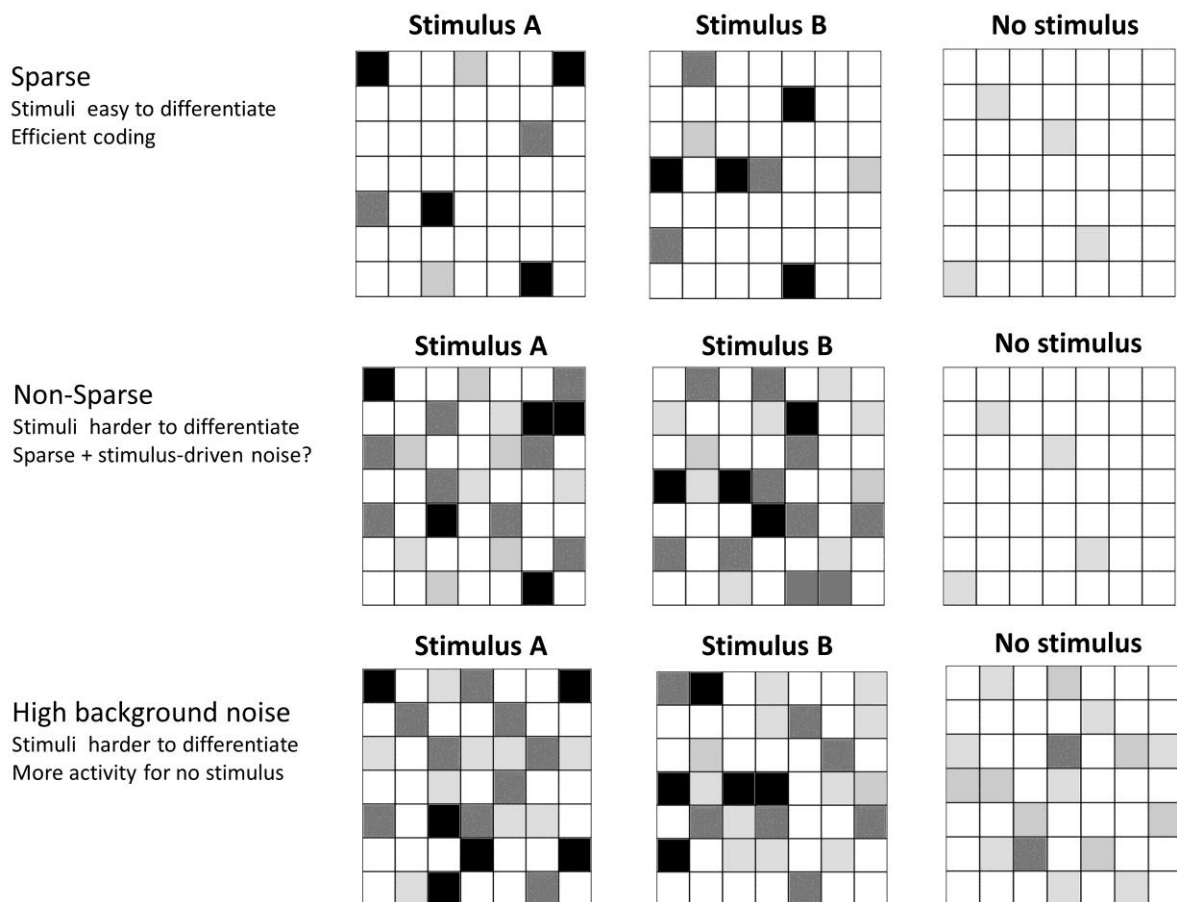


Figure 1: Neural responsiveness to different sensory stimuli (A and B) and to the absence of a stimulus in three different scenarios. The squares on a grid could represent individual neurons, cortical mini-columns, or voxels in fMRI (the basic principle is the same). Non-sparse responses (middle row) and high background noise (bottom row) have a larger overall activity which may correspond to individual differences in neural and subjective sensory sensitivity. The extent to which the patterns of activity to stimulus A and B are uncorrelated would relate to behavioural sensory sensitivity, but will also manifest in differences in the degree to which these patterns can be ‘decoded’ externally (e.g. from techniques such as multi-voxel-pattern analysis in fMRI).

Neurotypical individual differences

There are several lines of direct evidence from neurotypical individual differences (i.e. unrelated to autism) that support this model of sensory sensitivity. In humans, the MRI-based technique of Magnetic Resonance Spectroscopy (MRS) can estimate local concentrations of GABA and glutamate (Schirmer & Auer, 2000). Higher levels of GABA in sensorimotor cortex are correlated with better vibrotactile frequency discrimination (Puts, Edden, Evans, McGlone, & McGonigle, 2011) and, within occipital cortex, higher levels of GABA are associated with better visual orientation discrimination (Edden, Muthukumaraswamy, Freeman, & Singh, 2009). Continuous theta-burst stimulation (cTBS) of the occipital cortex using the technique of TMS (Transcranial Magnetic Stimulation) increases the local GABA concentration and enhances the visibility of weak visual stimuli (Allen et al., 2014). These studies all support the assumption that a higher level of GABA results in a more differentiated (sparser) neural response (less neural sensory sensitivity) and, hence, enhanced behavioural sensory sensitivity. This research remains largely silent on subjective sensory sensitivity. One study that did examine subjective sensory sensitivity used

individual differences in subjective pain thresholds and found correlations with glutamate, but not GABA, concentrations in various regions involved in pain perception (Zunhammer et al., 2016). It may be the case that pain sensitivity is different from other senses (as it is an inherently affective stimulus), or that GABA and glutamate are linked to different aspects of sensitivity.

Genetic individual differences related to GABA and glutamate functioning have been linked to differences in sensory sensitivity. There are two kinds of GABA receptor (A and B), and mutations in the α -subunit of the GABA-A receptor (GABRB3) have been linked to autism (Buxbaum et al., 2002). Polymorphisms in this gene in the non-clinical child population are linked to differences in tactile sensitivity assessed both behaviourally and via parental report, with some polymorphisms enhancing and others decreasing sensitivity (Tavassoli, Auyeung, Murphy, Baron-Cohen, & Chakrabarti, 2012). Ugarte et al. (2000) found that genetically modified mice lacking this subunit have increased sensitivity (in terms of their behavioural responsiveness/aversion) to pain and touch (see also DeLorey et al., 2011). By contrast, Ko et al. (2005) found that mice lacking glutamate receptor 5 (GluR5) had significantly reduced sensitivity to pain. However, it is to be noted that in studies employing genetic variations it cannot be ascertained whether the effects are specifically sensory as opposed to, say, changing the affective nature of a sensory stimulus – given that the genetic differences are pervasive across all brain systems.

Clinical groups

What about autism? In support of the model, individual voxels in extrastriate cortex show larger visual receptive fields, measured by fMRI, in people with autism and greater responsiveness to simple visual stimuli, i.e. less sharp responses at least in the spatial domain (Schwarzkopf et al., 2014). However, the model of Rubenstein and Merzenich (2003) makes

the clear prediction that behavioural sensory sensitivity should be impaired in autism (whilst being subjectively hyper-sensitive), due to increased neural noise (i.e. propagation of activity from stimulus-relevant neurons to stimulus-irrelevant neurons - increased N_m). However, in many domains behavioural sensory sensitivity appears to be normal or superior in autism (Laurent Mottron et al., 2013; Simmons et al., 2009). Some have even taken this as possible evidence of enhanced lateral inhibition in perception (Laurent Mottron et al., 2013; Plaisted, 2001), which runs completely counter to the increased excitation: inhibition ratio model. Others have argued that the hypothesized deficit from the increased excitation:inhibition ratio model may be more apparent on some sensory tasks than others. Puts et al. (2014) found that people with ASC had no difficulty in vibrotactile frequency discrimination or vibrotactile temporal-order judgments, but they did have significant impairments related to the modulation of perception, such as through adaptation mechanisms. They argue that the latter, more than the former, may depend on lateral inhibitory functioning (via GABA) for ‘sharpening’ of neural representations. But, unfortunately, it is very hard to predict a priori which tasks require more/less lateral inhibition, and previous research has shown that vibrotactile frequency sensitivity is related to GABA concentrations (Puts et al., 2011) so group differences in this task could reasonably have been expected.

Robertson, Ratai, and Kanwisher (2016) found that overall levels of GABA and glutamate in visual cortex did not differ between an autistic and control group, but the correlations between GABA and behaviour did differ between groups. In the technique of binocular rivalry, a different image is projected to each eye but only one image at a time tends to be subjectively perceived, and the perceived image alternates over time. The rate of alternation is decreased in autism (i.e. more perceptual stability C. E. Robertson, Kravitz, Freyberg, Baron-Cohen, & Baker, 2013). In controls, there was a significant positive relationship between both GABA and glutamate concentrations and increased perceptual

stability (although whether GABA and glutamate concentrations are themselves correlated was not assessed). In people with autism, whilst glutamate showed this relationship GABA concentrations did not. Given that there were no group differences in GABA concentrations the result suggests that the deficit lies specifically in coupling the neurotransmitter with the relevant behaviour.

In summary, the model of increased excitation: inhibition ratio makes clear predictions about some aspects of sensory sensitivity: specifically worse behavioural discrimination, and a larger neural response to sensory stimuli as a result of increased glutamate and/or decreased GABA. There is some evidence for this in terms of neurotypical individual differences. There is far less evidence concerning subjective sensory sensitivity (aversion to sounds, touch, light, etc.). The evidence from autism is also mixed. There is evidence for GABA dysfunction in autism, but the nature of that mechanism is unclear (it is unlikely to be the concentration of GABA), and its relationship to sensory sensitivity lacks evidence. More importantly, the theory does not readily predict why people with autism have some sensory strengths but it instead predicts a more pervasive pattern of dysfunction.

Sensory Sensitivity due to Different Balance between Priors and Sensory Inputs

The increased ratio of excitation: inhibition model argues that sensory hypersensitivity arises because the activated sensory representation is less sparse due to unchecked propagation of excitatory activity across neuronal populations. Other theoretical accounts make related assumptions but assume it reflects differences in information processing within the stimulus-relevant neural code (the $K(S)$ part of the previous equation, rather than the N_m or N_a parts). Specifically, it has been argued that the activated neural representation may be sparser when it reflects previous perceptual history (e.g. statistical properties of the natural environment, Hibbard & O'Hare, 2015) or current predictions (e.g.

Rao & Ballard, 1999), relative to unfamiliar or unpredicted sensory inputs. To translate this in to individual differences, people who are unable to predict their sensory experiences or use prior perceptual knowledge of the world will be hyper-sensitive, at least in terms of having an increased neural response to sensory stimuli. I will discuss below how this might translate into differences in behavioural and subjective sensory sensitivity.

Predictable sensory stimuli have a sparser neural response

Hibbard and O'Hare (2015) developed a neural model of primary visual cortex responses to uncomfortable images which are known to be implicated in some forms of sensory hypersensitivity including migraine and epilepsy. The model was based on the principle of behaviourally optimal coding to reduce the metabolic costs of neural activity. Visual images can be decomposed into different spatial frequencies (f) with higher spatial frequencies corresponding to finer detail and lower spatial frequencies corresponding to broader surface information. However, in real-world scenes the relative amount of each spatial frequency is statistically regular and favours lower frequency components (given by the power law $1/f^\beta$ where $\beta=0.8$ to 1.2 Tolhurst, Tadmour, & Chao, 1992). Cortical neurons in V1 are, by contrast, preferentially tuned to a somewhat higher 2-6 cycle per degree range (Devalois, Albrecht, & Thorell, 1982), which may maximise sensitivity to under-represented spatial frequencies in the environment (Sharpee et al., 2006) and also lead to a sparser response than if the neural code exactly mirrored the properties of the environment. By modelling neural responses to different images, based on physiological data about how they are tuned, it was shown that natural images do indeed activate a sparser code (in terms of population activity of neurons) whereas images that contain only mid- and high spatial frequency components produce much greater activity. Images with only mid- and high-spatial frequency components, which are rare in the natural environment, are known to be

rated as uncomfortable to view by most people (Juricevic, Land, Wilkins, & Webster, 2010), and are more prone to trigger epileptic seizures (Radhakrishnan et al., 2005) and migraines (Debne, 1984). High spatial frequency stimuli also evoke larger visual-evoked potentials in people with autism (Vlamings, Jonkman, van Daalen, van der Gaag, & Kemner, 2010) and synaesthesia (Barnett et al., 2008), whereas lower spatial frequencies do not. This offers an account as to why some kinds of stimuli may be linked to sensory hypersensitivity more than others (i.e. differences in neural activity relating to $K(S)$ in the initial equation) but it is not, in itself, an account of individual differences.

The assumptions relating efficiency to sparse coding derive from Information Theory (Shannon, 1948), but are also conceptually related to similar notions derived from the Predictive Coding framework or Bayesian brain (e.g. Friston, 2005; Rao & Ballard, 1999). In this framework, incoming sensory signals are compared against top-down knowledge (termed ‘priors’). Priors are assumed to be probabilistic, and can consist of representations of statistical regularities of the sensory world (e.g. typical distribution of spatial frequencies in natural scenes; the fact that light tends to come from above) but also more context-specific information such as an expectation that the letter X will be presented next. A set of neurons, assumed to be within sensory cortex itself, compares the sensory signal with these priors and computes a ‘prediction error’ (essentially the difference between what is expected and the sensory input). Only if there is a mismatch between these (i.e. the prediction error is large) is the sensory signal fed-forward and processed further. This theory generates the specific hypothesis that there will be less sensory neural activity (i.e. sparser codes) when processing expected relative to unexpected information. There are several lines of research consistent with this. The expectation of an upcoming visual stimulus decreases the overall (fMRI) activity in early visual cortex, V1, but increases its information content as shown by multi-voxel pattern analysis (Kok, Jehee, & de Lange, 2012; see also Summerfield, Trittschuh,

Monti, Mesulam, & Egner, 2008). This is consistent with a sharpened (i.e. sparser) and more differentiated neural code.

Individual Differences in Sensory Prediction

The predictive coding framework has been extended to account for individual differences, such as those linked with autism, and to the symptom of sensory hypersensitivity more specifically. Pellicano and Burr (2012) argue that people with autism see the world more accurately – as it really is – as a consequence of being less biased by prior experiences. For instance, they can be better able to copy impossible figures (L. Mottron, Belleville, & Menard, 1999). Pellicano and Burr (2012) claim that people with autism still possess priors but they may be less precise, and more uncertain. Moreover, they claim that less certain expectations about the sensory world may lead to feelings of being overwhelmed: i.e. increased subjective sensory sensitivity. Thus, in this account both behavioural and subjective sensory sensitivity go together (both are enhanced), because the individual difference lies in greater processing of the sensory signal, $K(S)$, rather than greater noise. There is some suggestive evidence to support this. Autistic people with savant skills (e.g. in music, art, memory) have higher subjective sensory sensitivity than autistic controls matched in other respects including education and autism symptom severity (Hughes, Ward, Gruffydd, Baron-Cohen, & Simner, in press). There are, however, somewhat different accounts of autism within the predictive processing framework (Palmer, Lawson, & Hohwy, 2017; Van de Cruys et al., 2014). Van de Cruys et al. (2014) argues that the difference lies in having too precise bottom-up signals thus “Overweighting of irrelevant prediction errors causes sensory overload” (p. 661). This sits in contrast with Pellicano and Burr’s (2012) position that people with autism have too imprecise priors, but the net effect is similar: sensory signals are taken at face value (and both accounts posit changes in $K(S)$ rather than noise). Some research has

attempted to pull these mechanisms apart. Lawson, Mathys, and Rees (2017) measured how people with autism and controls responded to unexpected events (including using pupil size measures) during trial-by-trial variability in the presentation of sensory stimuli. By fitting the results to a formal Bayesian model, they showed that people with autism tend to learn more about the volatility (the random fluctuations) which led to non-generalizable predictions and, hence, showing less surprise. This kind of process is often called ‘over-fitting’ or treating noise as if it is signal (although the sensory system itself may not necessarily be noisier), consistent with the model of Van de Cruys et al. (2014–2017). Hybrid accounts, that depend on the ratio of precision of priors to precision of sensory input, may also help to reconcile these different perspectives.

These predictive coding accounts make specific claims about when perception will be impaired in autism and when it is likely to be enhanced. Two scenarios are considered in more detail: multisensory integration, and sensory adaptation. A tendency to weight bottom-up signals too precisely (as in Van de Cruys et al., (2014) may result in less integration of signals from different sensory modalities and it is to be noted that people with autism show less multisensory integration (Stevenson et al., 2014). If two sensory signals are uncertain (less precise) then they will influence each other more, leading to multisensory integration. If, however, two sensory signals are more certain (more precise) – as proposed for autism – then they are less likely to be integrated. These unintegrated signals will be treated as two (unimodal) sensory events rather than a single (multisensory) event. This duplication of events may be another possible source of ‘sensory overload’ in autism and in other groups in which multisensory integration appears to be reduced including synaesthesia (Sinke et al., 2014) and migraine (Brighina et al., 2015). However, few studies have contrasted subjective sensory sensitivity (e.g. assessed via questionnaire) and multisensory processing directly. One study that did find that subjective sensory hyper-sensitivity was related to reduced

visuo-tactile temporal acuity and increased visual bias when presented with multisensory stimuli in both autistic and neurotypical individuals (Poole, Gowen, Warren, & Poliakoff, 2017).

There is also evidence that conditions linked to subjective sensory hyper-sensitivity show less effects of perceptual adaptation (e.g. in autism, Pellicano, Jeffery, Burr, & Rhodes, 2007; Puts et al., 2014). This is consistent with the notion that the predictability of the stimulus is not being coded in the normal way. Adaptation refers to a weaker neural response to a repeated, or continuous, sensory stimulus, such that the stimulus becomes less likely to elicit a behavioural response (termed habituation). Everyday examples of this might be the smell of someone's perfume or the noise of a digger outside. For most people, these stimuli may be intense and aversive to begin with, but will become subjectively less intense over time, perhaps to the extent that they are no longer consciously perceived. Thus, high sensory sensitivity could be an outcome of weaker adaptation mechanisms that prevent ongoing, normal sensory experiences from being attenuated (e.g. the feel of clothes labels, the hum of a computer). Takarae and Sweeney (2017) also speculate on how reduced adaptation could also lead to clinical symptoms of hypo-sensitivity: a problem in adapting to old stimuli may reduce the capacity to prioritise (and hence, notice) new sensory stimuli. Less adaptation could also explain greater perceptual stability during binocular rivalry in autism (C. E. Robertson et al., 2013), because the dominant percept does not spontaneously weaken. There is also evidence for weaker adaptation in migraine, assessed using EEG evoked potentials, in several modalities and not just vision (Brighina, Palermo, & Fierro, 2009). Synaesthetes show longer duration (i.e. less adaptation) to the McCollough effect, but normal duration of retinal after-images (Ramachandran & Marcus, 2017). Again, changes in adaptation will most likely reflect how the sensory signal is updated over time (i.e. $K(S)$), but less neural noise could also contribute to weaker adaptation. In all cases, the evidence is indirect and

future research needs to link individual differences in adaptation to measures of subjective sensory sensitivity.

Finally, the predictive coding framework has a particular way of framing the relationship between attention (commonly thought of as the selection of sensory stimuli for further processing) and perception. Within the predictive coding account, attention is defined as optimisation of precision; that is, paying attention to some sensory signal means that the precision weighting on this signal is increased so that it has more influence (Hohwy, 2012). In the case of endogenous attention, subjects increase precision (assign attention) of a signal to particular spatial locations based on top-down expectations (in this case about expectations of precision, rather than expectations of the stimulus itself). Exogenous attention, however, is thought to be a process of assigning greater precision-weighting to stimuli that are very salient based on low-level features, for example, objects with sudden onsets or high contrast. So to extrapolate this to van der Cruys et al.'s (2014) model, the suggestion that people with autism assign too great a precision to sensory signals is the same as saying that they are hyper-attentive.

Increased Sensory Sensitivity due to Differences in Endogenous Noise

Endogenous noise (N_a in the equations above) occurs both in the presence and absence of sensory signals. An increase in the level of endogenous noise would have several effects. It would increase the overall level of neural activity (i.e. increased neural sensory sensitivity and, by implication, increased subjective sensory sensitivity). It would also increase variability from time to time: perhaps leading to both reports of hyper- and hypo-sensitivity. Although behavioural sensory sensitivity would tend to be reduced (because the signal:noise ratio is reduced) in some circumstances it could be enhanced through the process

termed stochastic resonance (McDonnell & Abbott, 2009). In stochastic resonance, weak sensory signals that are just below the threshold for detection/discrimination can be boosted by the presence of noise as shown in Figure 2. In their review of visual abilities in autism, Simmons et al. (2009) concluded that increased endogenous noise might explain many of the perceptual difficulties but also some of the enhancements. For example, some studies show both perceptual advantages and disadvantages in the same autistic participants (Bertone, Mottron, Jelenic, & Faubert, 2005) which can be characterised as single versus multiple sources of noise; where a single source could be advantageous (due to stochastic resonance) but multiple sources are disruptive when combined. This review also noted the large variability in inter-individual perceptual thresholds in autism, consistent with a noise explanation. Other evidence that could potentially be consistent with increased endogenous noise is the fact that both autistic traits and increased sensory sensitivity, varying in the neurotypical population, are linked to anomalous perceptual experiences in everyday life (Horder, Wilson, Mendez, & Murphy, 2014).

INSERT FIGURE 2 ABOUT HERE

Stochastic Resonance

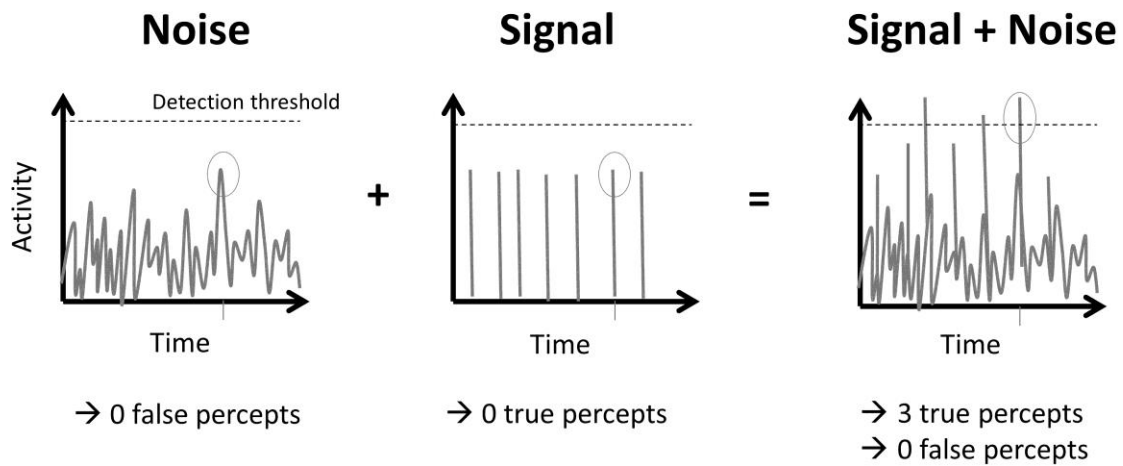


Figure 2: *The summation of sub-threshold noise and sub-threshold signal can boost behavioural sensory sensitivity, termed stochastic resonance. Too little noise will result in no benefit. Too much noise will also be deleterious, and result in ‘false perceptions’.*

At this juncture it is worthwhile contrasting this account with others discussed so far given that all emphasise a role for neural noise (but in different ways). Firstly, the predictive coding account of van de Cruys et al. (2014) does not assume a higher level of endogenous noise but, rather, assumes that normal sensory noise is over-interpreted in autism (i.e. treated as signal). The increased excitation:inhibition account does predict increased neural noise but, in this case, it is primarily stimulus-evoked noise (N_m) that leads to a less differentiated response. These different interpretations could be teased apart empirically. For instance, the method of tRNS (transcranial random noise stimulation) can be used to measure the level of stochastic resonance over visual cortex (van der Groen & Wenderoth, 2016). This study

found that adding a medium amount of neural noise enhanced detectability of visual stimuli but too little noise or too much noise did not. To translate to individual differences: if an individual already had an optimal amount of neural noise then adding more would worsen performance, but if an individual had too little noise then adding tRNS would boost performance. An alternative method uses TMS. High levels of endogenous noise over visual regions should be associated with lower TMS phosphene thresholds: if the brain requires less external stimulation to elicit some behaviour/experience (e.g. a phosphene) then we can conclude that it has higher levels of intrinsic excitability. In terms of individual differences, it has been shown that lower phosphene thresholds (Terhune et al., 2015) and lower motor thresholds (Stagg et al., 2011) are linked to increased glutamate (not GABA) in visual and motor cortices respectively. It remains to be shown how this relates to subjective sensory sensitivity measures, if at all.

In contrast to Simmons et al., (2009), Davis and Plaisted-Grant (2015) argue that autism is linked to less endogenous neural noise. They cite a variety of evidence including that less noisy (and more precise) sensory signals are less likely to be combined (similar to predictive coding), and greater perceptual stability in autism (e.g. in tasks such as binocular rivalry). However, this theory doesn't obviously speak to atypical subjective sensory sensitivity such as the DSM-V criteria. For instance, it is not clear how less endogenous neural noise explains the 'sensory overload' descriptions of hyper-sensitivity. However, the low versus high noise accounts can be empirically disentangled using methods such as tRNS and TMS. Moreover, these approaches can be extended beyond autism. For instance, increased neural noise accounts have been applied to migraine (O'Hare & Hibbard, 2016).

Increased Sensory Sensitivity due to Altered Network Connectivity

One suggestion is that hyper-connectivity to/from sensory regions leads to both increased subjective sensory sensitivity and increased neural activity (although that neural activity may be propagated away from sensory cortices). Within the framework outlined above, this most closely fits with an increased signal-induced noise (N_m). Therefore, this most closely resembles the Increased Excitation-Inhibition Ratio model. In the increased excitation:inhibition account, the noise effectively spreads within the stimulated sensory system and will necessarily affect behavioural sensory sensitivity. However, if changes of connectivity occur at other spatial scales (e.g. longer range connections between sensory systems and the amygdala) then the impact on behavioural sensory sensitivity is uncertain (and potentially non-existent).

With regards to autism, there is strong evidence of atypical structural and functional brain connectivity although the pattern is complex. Functionally, resting state fMRI reveals large-scale hypo-connectivity between many regions although some regions display hyper-connectivity, including connectivity between subcortical (thalamic, basal ganglia) regions and primary sensory and motor regions (Di Martino et al., 2014). In terms of structural connectivity, assessed with DTI, the organisation of the white matter tends to be less coherent in autism (Anagnostou & Taylor, 2011). Grey matter thickness can be increased in autism, and this may be a marker of increased local connectivity (Raznahan et al., 2010). Grey-matter differences are age-dependent with visual and auditory regions showing an atypical age-related increase in autism, but other regions (e.g. prefrontal) showing the opposite pattern (Watanabe & Rees, 2016). What is far less clear is how this connectivity profile is related to the profile of atypical sensory sensitivity documented for autism. It is possible that increased thalamic to cortical sensory signals leads to atypical development of these target areas. It is also unclear whether similar individual differences in connectivity are implicated in other conditions linked to atypical sensory sensitivity. Synaesthesia has been linked to a profile of

hyper-connectivity across multiple brain regions (Hänggi, Wotruba, & Jäncke, 2011; Rouw, Scholte, & Colizoli, 2011). Neurotypical variation in hyper-sensitivity to noise has been shown to be linked to increased grey matter in auditory cortex but also a number of non-sensory limbic regions including amygdala and hippocampus (Kliuchko et al., 2018). Whilst migraine has been linked to reduced grey matter volumes in certain regions (Dai et al., 2015), other research that specifically examined visual cortical regions (MT+ and V3A) found increased grey matter as well as sub-cortical white matter differences in visual pathways (Granziera, DaSilva, Snyder, Tuch, & Hadjikhani, 2006).

A number of brain imaging studies using fMRI have suggested a link between sensory hyper-sensitivity and increased activity in limbic regions (amygdala, hippocampus). Green et al. (2013) presented mildly aversive auditory and visual stimuli to children with autism and neurotypical controls. The autism group showed increased BOLD responses in primary sensory cortices and also amygdala, hippocampus, and orbitofrontal cortex. In both groups, the degree of this activity was positively correlated with individual differences in reports of sensory sensitivity made by a parent. A subsequent study report similar findings using tactile stimuli (Green et al., 2015). Using functional connectivity analysis, Green et al. (2017) found that increased connectivity between the thalamus and amygdala was linked to the sensory hyper-sensitivity in autism, and they speculated that the amygdala may then lead to a heightened responsiveness in sensory cortex (i.e. an amygdala-driven attentional alerting mechanism).

A connectivity-based account of sensory sensitivity in autism has been developed by Markram and Markram (2010), termed Intense World Theory. The Intense World Theory was initially motivated by the clinical observation that pregnant mothers treated with the anti-epileptic drug, Valproic Acid (VPA), had a high incidence of autistic-like symptoms in their

offspring (Moore et al., 2000). The vast majority of subsequent evidence comes from animal models of offspring treated with VPA. The cardinal behavioural and neurophysiological symptom in these animals is a hyper-reactivity to stimuli (notably sensory and affective stimuli), including a lack of habituation to repeated sensory stimuli. Superficially, this appears to resemble the increased excitation:inhibition model discussed earlier. However, the evidence suggests that the mechanism of action of VPA does not disrupt overall excitation and inhibition (at least not to sensory stimuli) but, instead, leads to a 50% increase in connectivity affecting both excitatory and inhibitory neurons (Rinaldi, Silberberg, & Markram, 2008). It is this increased connectivity that drives hyper-reactivity (increased neural sensory sensitivity) and is assumed to drive subjective sensory sensitivity (an ‘intense world’). With regards to behavioural sensitivity, the claim is that the increased local connectivity enhances perceptual ability but makes sensory processing more autonomous (i.e. less biased by context or global cues). Further evidence is clearly needed to link the Intense World Theory (driven primarily by animal models) to human neuroscience and behaviour.

Summary and Outstanding Questions

This manuscript has attempted to sketch out a broad framework for considering individual differences in sensory sensitivity linked to both neurotypical variation, and neurodevelopmental group-based differences. It has been suggested that future research needs to more clearly articulate different ways in which sensory sensitivity can be described. Specifically, three concepts of sensory sensitivity are described: subjective sensory sensitivity (strong feelings of aversion/intensity that define clinical symptoms); neural sensory sensitivity (the degree of neural activity induced by sensory stimuli); and behavioural sensory

sensitivity (detection and discrimination of sensory stimuli). The extent to which these different facets of sensory sensitivity are related can be understood in terms of a simple consideration of signal detection from neural signal and noise. This is extended from previous accounts (e.g. relating to migraine; O'Hare and Hibbard, 2016) to offer a unifying framework for exploring sensory sensitivity across different conditions (e.g. autism, migraine, synaesthesia). The framework is discussed, in more concrete terms, by linking it to four existing theoretical accounts of atypical sensory sensitivity: increased excitation-to-inhibition ratio; predictive coding; increased neural noise; and atypical brain connectivity. These four accounts all have certain strengths and weaknesses in explaining the available evidence that make hard to adjudicate between them. These are summarised in Table 2 and reflect a largely personal perspective on the current state of the field. An over-arching criticism of the field is that no existing account convincingly links together the three different aspects of sensory sensitivity delineated at the outset of this paper (subjective, behavioural and neural).

To some extent, these different accounts of atypical sensory sensitivity are not mutually exclusive. This is partly because they are pitched at different levels of analysis, to use Marr's term. Marr (1982) described how explanations can occur at three different levels: computational (what problem is being solved and what is the approach for solving it); algorithmic (how the problem is solved); and implementational (how the solution is implemented physically). Accounts such as predictive coding, are couched primarily at the algorithmic level whereas other accounts such as increased excitation:inhibition or Intense World Theory are couched primarily at the implementational level. It is, of course, conceivable that differences in neurotransmitter functionality or neural connectivity drive algorithmic differences, resulting in differences in the way that sensory stimuli are predicted. At the computational level (in Marr's terms), it is suggested that the basic problem being

solved is that of maximising behavioural sensory sensitivity (because this is functionally adaptive) whilst minimizing neural sensory sensitivity (because a high amount of neural responsiveness is metabolically costly). However, different individuals may adopt somewhat different solutions to this problem owing to intrinsic differences present in their ‘software’ (cognition) or ‘hardware’ (neural architecture). It is these different solutions to the same problem that give rise to individual differences in sensory sensitivity.

To give one example, Shriki, Saleh and Ward (2017) developed a computational model of synaesthesia in which the learning algorithm was set to maximise behavioural sensory sensitivity of two ‘modalities’ but under a variety of different starting conditions (e.g. differences in plasticity, differences in the relative sensitivities of the modalities). The extent to which the model exhibited a synaesthetic response (i.e. a sensory stimulus to one modality eliciting activity in both modalities; a less sparse neural response) depended on these atypical starting conditions (most solutions did not exhibit this synaesthetic behaviour). However, it is important to note that these atypical solutions can, for that individual, still be statistically optimal.

Table 2: An evaluation of the four different theoretical accounts of individual differences in sensory sensitivity

Theoretical Accounts	Strengths	Weaknesses	Outstanding Questions
Increased Sensory Sensitivity due to an Increased Excitation-to-Inhibition Ratio	<ul style="list-style-type: none"> • Good evidence of a link between GABA/glutamate concentrations and neurotypical individual differences in behavioural sensory sensitivity • Evidence of GABA dysfunction in ASD but less clear how this is linked to behavioural and subjective sensory sensitivity 	<ul style="list-style-type: none"> • Few studies have explored GABA/glutamate in terms of subjective sensory sensitivity (clinical symptoms) • Does not readily explain the pattern of perceptual strengths (as well as weaknesses) in ASD 	<ul style="list-style-type: none"> • Is an increased excitation:inhibition ratio linked both to increased subjective sensory sensitivity and reduced behavioural sensory sensitivity? • If so, which conditions does this apply to and is it specific to particular sensory modalities?
Sensory Sensitivity due to Different Balance between Priors and Sensory Inputs	<ul style="list-style-type: none"> • Makes testable predictions about the kinds of scenarios in which perception may be enhanced or impaired • Less adaptation (less attenuation of predictable stimuli?) has been found in a variety of conditions linked to atypical sensory sensitivity 	<ul style="list-style-type: none"> • Theory is underspecified at the implementational level • Lack of evidence linking this account to subjective sensory sensitivity 	<ul style="list-style-type: none"> • What neurobiological differences lead to differences in predictive processing and why? • How is it possible to distinguish between less reliance on priors versus overly precise predictions?
Increased Sensory Sensitivity due to Differences in Endogenous Noise	<ul style="list-style-type: none"> • May account for unusual perceptual experiences (e.g. migraine aura) 	<ul style="list-style-type: none"> • Existing accounts tend not to distinguish different sources of noise (termed here as additive and multiplicative) 	<ul style="list-style-type: none"> • Can less endogenous noise (as opposed to more) explain high subjective sensory sensitivity?
Increased Sensory Sensitivity due to Altered Network Connectivity	<ul style="list-style-type: none"> • Clear evidence of differences in structural/functional connectivity in conditions linked to atypical sensory sensitivity 	<ul style="list-style-type: none"> • Less clear predictions about whether behavioural sensory sensitivity correlates positively or negatively with subjective sensory sensitivity 	<ul style="list-style-type: none"> • Which specific aspects of connectivity (e.g. small world features) are relevant to atypical sensory sensitivity?

	<ul style="list-style-type: none">• Some evidence that these differences correlate with subjective sensory sensitivity		<ul style="list-style-type: none">• What is the role of atypical connectivity to regions such as amygdala in subjective sensory sensitivity?
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Outstanding Issues

The remaining sections consider specific research questions, and identify possible methods for addressing them.

What is the neural basis of atypical sensory sensitivity?

As noted at the outset, there is a pervasive assumption that increased subjective sensory sensitivity and increased neural sensory sensitivity go hand-in-hand. However, a more detailed consideration of the theories and evidence suggests that this question should be broken down into more specific questions and hypotheses.

One of the key questions to be resolved is whether increased neural activity is linked to the amount of activity per se or the number of active ‘units’ (neurons, voxels, etc.). That is, the extent to which activated neural representations differ in activity per se or in terms of sparseness, and the extent to which these patterns of activity are stable or variable over time (the latter being indicative of noise). Different theories make different predictions concerning this. It also needs to be determined whether these differences only manifest themselves in the presence of a sensory stimulus but also in its absence (if level of background noise differ). It is also unclear which regions in sensory cortex are linked to individual differences in sensory sensitivity, and whether concomitant activity in non-sensory regions (e.g. amygdala) is a better predictor. Finally, studies involving TMS (individual differences in phosphene threshold) and tRNS are important for assessing individual differences in neural noise.

What is the relationship between different conditions with atypical sensory sensitivity?

Although it has been argued that there is no necessary reason why different conditions linked to atypical sensory sensitivity should have the same cause, this is essentially an empirical question. This is best addressed by applying common methods across multiple conditions; an approach that is, hitherto, lacking. We have attempted to explore this comparing synaesthesia and autism (Ward et al., 2017), two conditions which occur together more than chance (e.g. Baron-Cohen et al., 2013), but the approach can be extended more widely.

One feature that has been reported across multiple conditions linked to sensory hyper-sensitivity is less adaptation and habituation; i.e. such that repeated or prolonged sensory stimulation does not lead to a large drop-off in the neural or behavioural response. However, different measures of adaptation have been used across different studies and the specific link with subjective sensory sensitivity remains untested. It is also to be noted that even if reduced adaptation were a common feature across conditions, there could be differences in the way this is implemented (e.g. failure to suppress from top-down priors, or less build-up of noise).

What is the relationship between sensory sensitivity and other clinical symptoms?

The extent to which atypical sensory sensitivity is linked to other clinical symptoms or other cognitive differences remains to be tested. Historically, sensory sensitivity has been linked to a sensitive personality type that includes introversion and increased emotionality (Aron & Aron, 1997). This idea has parallels going back to the Eighteenth century notions of ‘sensibility’. Some of these personality characteristics (e.g. preference for solitude) would perhaps now fall within autism spectrum tendencies, but some accounts continue to make a link between sensory and emotional sensitivity. For instance, the Intense World Theory makes the claim that sensory hyper-sensitivity and emotional hyper-sensitivity are two co-

occurring outcomes of the atypical brain development underpinning autism (Markram & Markram, 2010). Anxiety is a common clinical feature of autism and is correlated with subjective sensory hyper-sensitivity (Mazurek et al., 2013). Some theories suggest that anxiety is linked to heightened awareness of one's bodily responsiveness or interoception (Domschke, Stevens, Pfleiderer, & Gerlach, 2010). Does subjective hyper-sensitivity to internal bodily signals co-occur with hyper-sensitivity to external senses such as sounds and vision? This would be an important area to explore.

What is hypo-sensitivity?

Most theories focus on sensory hyper-sensitivity perhaps because it is conceptually easier to explain and because it is a more important clinical symptom than hypo-sensitivity. Nevertheless, the apparent association between hyper- and hypo-sensitivities (Horder et al., 2014; Robertson & Simmons, 2013) remains an important area to investigate. These ideas also need more clarity in terms of how they are defined and measured. For instance, low responsiveness to sensory stimulation could be interpreted as 'hypo-sensitivity' but it need not be: sensitivity and responsiveness are separate constructs (Pluess, 2015). Several accounts have been discussed in this paper, and these make testable predictions. For instance, it is possible for someone to have subjective sensory hyper-sensitivity but, at the same time, be behaviourally hypo-sensitive to stimuli (i.e. less able to notice or discriminate them). It may also be the case that poor adaptation means that sensory stimuli are not prioritised in a typical way (i.e. such that old stimuli are less perceptually salient, so that new stimuli can be more easily detected). The relationship, if any, between hypo-sensitivity and repetitive sensory-motor acts (e.g. flicking ones fingers in front of the eyes) also needs to be understood as these ideas have sometimes been conflated. It may, for instance, be the case that repetitive acts are

repeated simply because their effects do not diminish in the same way over time (less adaptation) for some people.

What stimulus properties elicit hyper-sensitive (or hypo-sensitive) responses?

Little is known about the particular characteristics of sensory stimuli that are known to elicit strong responses in people who self-report high subjective sensory sensitivity, although the general properties of stimuli known to be rated as uncomfortable have been studied (auditory: e.g. Kumar, Forster, Bailey, & Griffiths, 2008; visual: e.g. Juricevic, et al., 2010). The suggestion that sensory stimuli that have a natural tendency to elicit a large, non-sparse neural response may be linked to hyper-sensitivity is an idea that can be explored across different sensory systems. It is sometimes reported that some individuals (e.g. in misophonia) have their own idiosyncratic triggers, but whether there are commonalities across such cases remains to be determined. If there are individuals for whom a very small set of triggers can be identified then a role of learning would need to be ruled out. However, this explanation would be less convincing for those who report a more pervasive pattern of sensitivities. Preliminary evidence suggests a heritable component to increased sensory sensitivity (Donaldson, Stauder, & Donkers, 2017). A possible middle-ground is that people with high sensory sensitivity have an increased aversion/intensity to a wide-range of sensory stimuli but, over and above that, some of these triggers (e.g. the sound of chewing) acquire a special status (akin to phobic responses).

Summary

This paper has critically evaluated the concept of individual differences in sensory sensitivity, explored its possible underlying neurobiological basis, and presented a roadmap for future research in this area. I hope that this will facilitate dialogue between researchers

working in different areas through the consideration of a common conceptual framework and common methodologies that enable the similarities and differences across conditions to be better understood.

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