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REVIEW

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Synaesthesia and autism: Different developmental outcomes from overlapping mechanisms?

Tessa M. van Leeuwen [a], Janina Neufeld[b], James Hughes[c] and Jamie Ward[c]

[a]Donders Institute for Brain, Cognition and Behaviour, Radboud University, Nijmegen, the Netherlands; [b]Center of Neurodevelopmental Disorders (KIND), Centre for Psychiatry Research, Department of Women's and Children's Health, Karolinska Institutet & Stockholm Health Care Services, Region Stockholm, Stockholm, Sweden; [c]School of Psychology, University of Sussex, Brighton, UK

ABSTRACT

Synaesthesia, a mixing of the senses, is more common in individuals with autism. Here, we review the evidence for the association between synaesthesia and autism with regard to their genetic background, brain connectivity, perception, cognitive mechanisms and their contribution to exceptional talents. Currently, the overlap between synaesthesia and autism is established most convincingly at the level of alterations in sensory sensitivity and perception, with synaesthetes showing autism-like profiles of sensory sensitivity and a bias towards details in perception. Shared features may include a predominance of local over global connectivity in the brain. When autism and synaesthesia co-occur in the same individual, the chance of developing heightened cognitive and memory abilities is increased. We discuss how the same theoretical models could potentially explain both conditions. Given the evidence, we believe the phenotypical overlap between autism and synaesthesia has been established clearly enough to invite future research to confirm overlapping mechanisms.

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Introduction

Synaesthesia is a sensory condition where specific inputs such as sounds, letters, tastes, or smells automatically trigger additional sensations such as colour, texture, or shape (Figure 1). A G-minor piano chord might, for instance, trigger the sensation of a blue cube with a smooth surface, or the word "stranger" might taste like potato purée. While, theoretically, all combinations of inducing stimuli and concurrent sensations are possible, most inducers are linguistic rather than strictly sensory in nature and most concurrents are visual such as colours or shapes (Ward, 2013). Each synaesthete has a unique pattern of couplings between inducing inputs and concurrent sensations. These couplings are generally stable in adult synaesthetes, meaning that for instance a letter or digit leads to the same colour sensation after years or even decades (Simner & Logie, 2008). Since a high consistency of inducer-concurrent couplings is usually seen as a defining feature of synaesthesia (but see Simner, 2012), synaesthetes are usually "diagnosed" using a test of consistency (Baron-Cohen et al., 1987) where they repeatedly indicate their synaesthesia (e.g., colours) for a set of inducers (e.g., letters and numbers). Using this approach, the prevalence of synaesthesia has been estimated to be about 4% of the general population with about 1% having grapheme-colour synaesthesia (GCS) where letters or digits induce colour sensations (Simner et al., 2006). In individuals with a diagnosis of Autism Spectrum Condition (ASC), however, synaesthesia has been reported to be more common than in the general population (Baron-Cohen et al., 2013; Neufeld et al., 2013). In this article, we summarize this evidence and discuss the possible underlying mechanisms.

ASC is a neurodevelopmental condition, with a prevalence of 1–2% in the general population (Elsabbagh et al., 2012; Lundström et al., 2015). It is characterized by difficulties in social communication and interaction, and patterns of restricted/repetitive behaviours, interests, or activities (American Psychiatric Association, 2013). Within the latter domain, alterations in sensory processing are today included as a defining criterion of ASC (hyper- and/or hypo-

CONTACT Tessa M. van Leeuwen tesvlee@gmail.com Donders Institute for Brain, Cognition and Behaviour, Radboud University, Montessorilaan 3, 6525 HR, Nijmegen, the Netherlands

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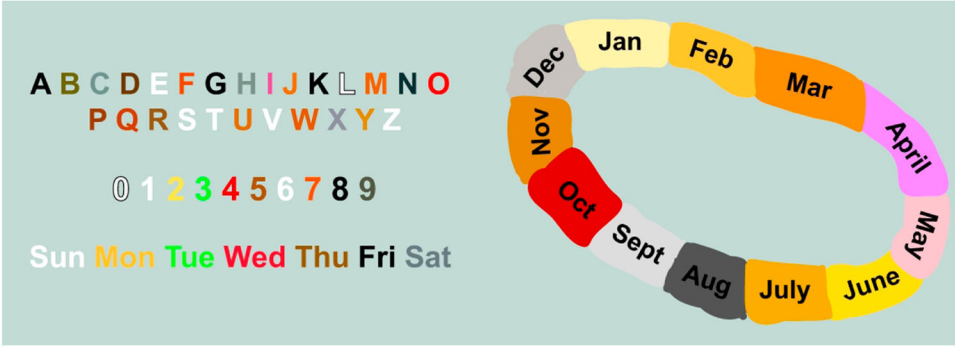

Figure 1. Illustrations of synaesthesia. Examples of grapheme-colour and week-day colour synaesthesia (left) and sequence-space synaesthesia (right). In sequence-space synaesthesia, sequences (in this case months) are experienced in a certain spatial configuration, sometimes situated around the body of the synaesthete. Sequence-space synaesthesia may involve colours but that is not always the case [To view this figure in colour, please see the online version of this journal].

sensitivity to sensory input or unusual seeking of sensory stimulation). Three studies have shown a higher prevalence of synaesthesia in people with a formal diagnosis of autism based on both self-report (Baron-Cohen et al., 2013) or consistency test results (Hughes et al., 2017; Neufeld et al., 2013). A fourth study screened a large population sample (N = 3,742) and reported an association between synaesthesia (tested for consistency) and a self-reported diagnosis of autism (Carmichael et al., 2019). As the study assessed many health conditions, the association did not survive correction for multiple comparisons but the effect size was moderate (odds ratio = 4.9) and relatively specific, i.e., most other health conditions, with the exception of anxiety, showed no reliable link with synaesthesia.

Rather than purely focusing on binary presence/absence of ASC diagnosis in synaesthetes, other studies have investigated the link between synaesthesia and continuous autistic traits, assessed using the Autism Spectrum Quotient, AQ (Baron-Cohen et al., 2001). We are aware of five studies that have applied this measure to verified synaesthetes with each showing a positive association between synaesthesia and either the total AQ or subscales of the AQ (Baron-Cohen et al., 2016; Van Leeuwen et al., 2019; Ward et al., 2017, 2018; Ward & Filiz, 2020), as discussed in detail below.

In summary, the association between synaesthesia and autism is replicable with results converging across different sampling methodologies (assessing autism amongst synaesthetes, assessing synaesthesia amongst autistic individuals, opportunistic sampling). Insofar as it has been assessed, the association has been found for different kinds of synaesthesia, with evidence that having more types of synaesthesia is linked to larger shifts along the autism spectrum (Van Leeuwen et al., 2019; Ward et al., 2018). The remainder of this article discusses how one can account for the co-occurrence of these two neurodevelopmental traits in terms of models of brain development, brain organisation and brain function (cognition). We stress that autism is intrinsically heterogeneous, with large individual differences in its manifestation including social, emotional and communication aspects, and that the overlap with synaesthesia may not be on all aspects. Also, there could be multiple ways to arrive at overlap in phenotypes: shared mechanisms, or non-shared mechanisms could cause the same observed outcome.

Shared biological mechanisms between synaesthesia and autism

In this section we consider whether synaesthesia and autism are likely to share overlapping genetic mechanisms or share similar features of brain architecture.

Genetic mechanisms

Both synaesthesia and autism are heritable (for synaesthesia see Bosley & Eagleman, 2015; for autism see Tick et al., 2016 for a meta-analysis). Both conditions are likely to be polygenic involving a mixture of rare genetic variants (with larger effect sizes) (Tilot et al., 2018) and more common genetic

polymorphisms (with small effect sizes) (Borenstein et al., 2009; Geschwind, 2011). One study on the genetic substrates of synaesthesia revealed linkage to a chromosome region (2q24.1; Asher et al., 2009) that has also been implicated in autism (International Molecular Genetic Study of Autism Consortium (IMGSAC), 2001), but given that several hundred genes are present in this region, this overlap is suggestive, at best.

People with synaesthesia regularly have multiple types (e.g., grapheme-colour together with music-colour synaesthesia): in a screening study, synaesthetes on average experienced 1.8 types of synaesthesia per person. The majority (64%) reported one synaesthesia type (Simner et al., 2006) and most multiple synaesthesias tend to occur within subgroups of synaesthesia types, e.g., multiple forms of coloured sequences (days, numbers, letters), with independence between the subgroups (Novich et al., 2011). Multiple types of synaesthesia can co-occur within the same family (e.g., a brother has grapheme-colour and a sister has lexical-gustatory, i.e., tasting words) (Barnett, Finucane, et al., 2008). This suggests that the genetic disposition towards synaesthesia is inherited, rather than one particular manifestation. It is currently debated whether this same genetic disposition could potentially give rise to a superficially very different phenotype such as autism. For instance, Bouvet et al. (2019) reported a large three-generational family in which multiple family members had either synaesthesia, (severe) autism, or both. In contrast, Tilot et al. (2019) did not find evidence for an increased polygenic risk for autism (identifying similarities to the known genetic profile of autism) in a sample of genotyped synaesthetes, but instead a small association with polygenic risk for schizophrenia. However, this needs to be extended with larger samples and include alternative measures of the synaesthetic phenotype (e.g., Ward & Filiz, 2020).

In summary, although the association between synaesthesia and autism and the family analyses of shared phenotypes suggests genetic bases, the evidence for an overlap between autism and synaesthesia from molecular genetics is presently lacking. By overlap, we mean e.g., mutations in similar types of genes (but not necessarily in identical genes) involved in relevant functions such as e.g., axonogenesis.

Brain architecture

Synaesthesia is typically explained in terms of hyper-connectivity among brain regions (Maurer & Mondloch, 2005; Ramachandran & Hubbard, 2001), whereas autism is more commonly associated with hypo-connectivity (Anderson, 2014). Hence, an association between the two in terms of neural architecture appears, at first sight, surprising. However, the situation is much more complex and nuanced than this. With regards to autism, whilst resting state functional connectivity reveals a pattern of widespread hypo-connectivity, certain networks show hyper-connectivity (e.g., sensorimotor-thalamic connections; Di Martino et al., 2014). It needs to be mentioned, however, that findings of altered brain connectivity in autism are highly heterogeneous and widespread, with little agreement between different studies and meta-analyses, limiting the generalizability of the findings (Pua et al., 2017).

One can also examine functional connectivity at different hierarchical levels. The neurotypical brain contains a network of high-level hubs that draw on information throughout the brain but tend mainly to talk to each other (termed rich-clubs; Van den Heuvel & Sporns, 2011). These higher-order integrative levels may be disrupted in autism, meaning that more localized, low-level forms of connectivity predominate (Hong et al., 2019): i.e., hypo-connectivity amongst globally-connected hubs with normal-to-hyper connectivity locally. This shift in pre-eminence from global to local information processing has frequently been used to explain the atypical sensory and cognitive profile of autism (e.g., weak central coherence, Happé & Frith, 2006; the enhanced perceptual functioning account, Mottron et al., 2006). These accounts are described in more detail later on.

For synaesthesia, the notion of hyper-connectivity stems in large part from the proposal that this phenomenon could arise from incomplete pruning of connections between sensory brain regions during early development (e.g., Maurer et al., 2013; Ramachandran & Hubbard, 2001). There is some empirical evidence for this in terms of increased white matter organization throughout the brain amongst synaesthetes, using diffusion tensor imaging (e.g., Rouw & Scholte, 2007; Zamm et al., 2013; but see Dojat et al., 2018 for null-results). In Rouw and Scholte (2007), white matter was more coherent (as measured with fractional anisotropy)

in ventral-occipital, parietal and frontal regions implicated in grapheme-colour synaesthesia. In Zamm et al. (2013), music-colour synaesthetes showed greater white matter coherence in a major white matter pathway that connects visual and auditory association areas to frontal regions. These differences in the brain tend to be more apparent in synaesthetes who project their colours externally ("projector" synaesthetes, Van Leeuwen, 2013). Other accounts emphasize localized changes in connectivity that might lead to atypical parcellation of adjacent cortical regions (Bargary & Mitchell, 2008) and atypical functional specializations ("a breakdown in modularity"; Baron-Cohen et al., 1993).

With regard to network analyses, Hänggi et al. (2011) performed a graph-theoretical analysis of the distribution of cortical gray matter thickness in synaesthetes and non-synaesthetes. They found hyperconnectivity throughout the brains of synaesthetes (reduced small-worldness) with increased clustering, which suggests enhanced local network efficiency. This is consistent with a pattern of shifted global-to-local connectivity as similarly reported for autism (Hong et al., 2019). On the other hand, in contrast to synaesthesia, decreased clustering (local efficiency) has been reported in autism in functional MRI studies (Itahashi et al., 2014; Rudie et al., 2013). Also, in synaesthesia, a functional connectivity study in EEG (Jäncke & Langer, 2011) report a similar small-world topology in synaesthetes and controls in their graph-theoretical analysis, but a stronger parietal hub in synaesthetes, among several other more interconnected areas (prefrontal, memory–related, visual areas). The mixed results for network analyses in both autism and synaesthesia stress that there is a clear need to contrast groups (autism and synaesthesia) directly using the same analytical measures (e.g., graph theory metrics).

In a whole brain resting state fMRI study by Dovern et al. (2012) stronger correlations between parietal and visual networks were found for synaesthetes. In Tomson et al. (2013) sequence-colour synaesthetes had more clustered connections within a local network that likely underlies their synaesthetic sensations than control participants, who tended to have more clustering in parietal-frontal areas. Both of these functional MRI studies suggest connectivity changes that are relatively localized.

It should be noted that changes in connectivity are not limited to autism and synaesthesia, as connectivity changes occur in many clinical conditions, e.g., schizophrenia. Hence, similarities in connectivity changes in autism and synaesthesia do not necessarily point at shared underlying mechanisms but that would then leave the question as to why they are co-occurring as unanswered. Also, it is currently unknown whether structural abnormalities in synaesthetes are causing the synaesthetic experiences, or whether structural changes are the result of the life-long experience of synaesthesia.

Another important aspect when studying brain connectivity is development. The developmental trajectories of synaesthesia and autism appear to differ, even though this is not readily understood. For instance, autism is per definition an early onset (developmental) disorder, with core symptoms manifesting as early as 2–3 years of age (DSM-5; APA, 2013). In contrast, in e.g., grapheme-colour synaesthesia, synaesthetic colour consistency develops gradually, with 34% of grapheme inducers being associated consistently with the same colours at 6/7 years of age evolving into 71% consistency at the age of 10/11 (Simner & Bain, 2013). However, it is unknown how early the first synaesthetic experiences emerge. Despite the heterogeneity of the brain imaging literature in autism, there is growing support for early brain over-growths and hyper-connectivity in childhood, followed by altered connectivity trajectories that lead to overall more hypo-connectivity in adulthood (for a review see Uddin et al., 2013). The hypothesis that synaesthesia arises from incomplete pruning during brain development is based on behavioural findings (Maurer & Mondloch, 2005), while the lack of brain imaging studies in child synaesthetes prevents us from comparing trajectories in brain development between synaesthesia and autism.

In conclusion, more studies using advanced whole-brain network analyses are needed for synaesthesia, and many studies in this area tend to be underpowered (for review and discussion, see Hupé & Dojat, 2015). Future research should directly compare autism and synaesthesia using the same measures, and larger samples can be obtained through multi-centre data-sharing initiatives.

Shared cognitive profiles between synaesthesia and autism

In discussing shared cognitive mechanisms between synaesthesia and autism, we do not wish to imply that this is an alternative to biological mechanisms

but rather acknowledge that many current cognitive accounts are not couched in terms of brain mechanisms. At present, there is more convincing evidence for a shared cognitive profile between autism and synaesthesia than shared biological mechanisms. This is important to keep in mind when discussing possibly shared vs non-shared mechanisms in reaching the same behavioural manifestation.

Shared sensory and attentional characteristics

Alterations in sensory processing (hyper- and/or hypo-sensitivities) are common in autism and therefore are part of the diagnostic criteria of ASC in the DSM-5 (American Psychiatric Association, 2013). As discussed below, evidence is accumulating that sensory processing is altered in synaesthesia as well (i.e., over and above having anomalous experiences).

Sensory sensitivity

In autism, hyper- and hypo-sensitivities to the environment are diverse, ranging from hypersensitivity for (e.g.,) touch, textures of food, bright lights (etc.) to the opposite profile of sensory seeking (Robertson & Baron-Cohen, 2017). Sensory hyper-sensitivity is experienced as one of the most intruding issues in daily life (Pellicano, 2013; Toeps, 2018) and up to 90% of individuals with autism are estimated to be affected by sensory atypicalities (Tomchek & Dunn, 2007). On questionnaires of sensory sensitivity individuals with autism typically score higher than neurotypical individuals, and among neurotypical individuals those with higher autistic traits (higher AQ) display stronger sensory atypicalities (Robertson & Simmons, 2013; Tavassoli et al., 2014, 2013; Tomchek & Dunn, 2007). Basic objective measures of sensory thresholds, for instance visual acuity or contrast sensitivity, tend not to differ in autism, or results are mixed (Robertson & Baron-Cohen, 2017; Simmons et al., 2009). However, the temporal integration of sensory signals may be altered (tactile, auditory and multisensory information), changing the quality of sensory processing (Robertson & Baron-Cohen, 2017), an issue to which we return below.

Interestingly, a very similar pattern emerges for sensory sensitivity in synaesthesia. Synaesthetes score higher than control participants (Van Leeuwen et al., 2019; Ward et al., 2017, 2018) on the Glasgow Sensory Questionnaire (GSQ) which indexes hyper- and hypo-sensitivities to seven sensory modalities. Among synaesthetes, those with more types of synaesthesia further scored higher on this questionnaire (Van Leeuwen et al., 2019; Ward et al., 2018). Synaesthetes present with both hyper- as well as hypo-sensitivities, similar to individuals with autism; however, in one study, synaesthetes only scored higher than control participants on the hyper-sensitivity subscales of the GSQ (Van Leeuwen et al., 2019). Several experimental studies suggest synaesthetes may be more sensitive to sensory stimulation, especially when it comes to the modality of the concurrent synaesthetic experience, e.g., colour or tactile experiences (Banissy et al., 2009, 2013; Barnett et al., 2008; van Leeuwen et al., 2013; Yaro & Ward, 2007). For some of these sensitivities, it is not clear whether they are the cause or consequence of synaesthesia (Ward, 2019).

Perception and attention

Besides subjective sensory sensitivity, perception and attention are also altered in autism. One characteristic of perception in autism is a shift in attention towards details, or, to put it the other way around, a reduced tendency to group items into a coherent whole (Bölte et al., 2007; Dakin & Frith, 2005). Attention-to-detail features centrally in several autism theories, such as the enhanced perceptual functioning model (Mottron et al., 2006) and the weak central coherence theory (Happé & Frith, 2006). This attentional bias in autism can manifest, for instance, as enhanced performance on the Embedded Figures Task, in which a small target shape hidden in a larger context figure has to be identified (e.g., Jolliffe & Baron-Cohen, 1997; Shah & Frith, 1983) or enhanced ability to detect small changes in a scene (reduced change blindness, e.g., Fletcher-Watson et al., 2012; Smith & Milne, 2009). Global visual processing in autism is thereby not truly impaired but may require relatively more time and effort (Robertson & Baron-Cohen, 2017; Van der Hallen et al., 2015).

In order to investigate parallels between synaesthesia and autism, several studies used the Autism Spectrum Quotient (AQ) questionnaire (Baron-Cohen et al., 2001). The AQ has five subscales indexing different autistic traits: Attention to detail, Social skills, Communication, Attention Switching, and Imagination. Synaesthetes were consistently found to score like individuals with autism on the AQ-Attention to detail

subscale, while scores on the other subscales were elevated to a lesser degree (Ward et al., 2017, 2018; Van Leeuwen et al., 2019). Building on this evidence, a number of studies investigated attention to detail in synaesthetes with more objective tests, using the Embedded Figures task, change blindness or motion coherence paradigms. On the Embedded Figures task, synaesthetes outperform controls on accuracy but not reaction time (Van Leeuwen et al., 2019; Ward et al., 2018). Similarly, on a change blindness task, synaesthetes detected more changes (Ward et al., 2018), similar to autistic individuals (Ames & Fletcher-Watson, 2010; Smith & Milne, 2009). On a motion coherence task, synaesthetes needed more coherently moving dots than controls before they were able to see the global direction of motion (Banissy et al., 2013; Van Leeuwen et al., 2019) consistent with reports for autism (Robertson et al., 2012; Simmons et al., 2009). The perception of detailed facial features is also enhanced in synaesthetes (Janik McErlean et al., 2016) as are responses to detailed non-synaesthesia inducing stimuli (Barnett et al., 2008). These results are all consistent with more attention for local elements in synaesthetes, resembling the findings in autism. It should be mentioned that the majority of studies into perceptual traits of synaesthetes included predominantly grapheme-colour or sequence-space synaesthetes. The effect of the type of synaesthesia on perception is not yet clear, but there is a dose effect of synaesthesia: the more types of synaesthesia an individual has (suggesting more modalities are included), the higher are self-reported sensory sensitivity and AQ-Attention to detail as well as motion coherence thresholds (Ward et al., 2018; Van Leeuwen et al., 2019). This implies that the association also holds for types of synaesthesia beyond grapheme-colour or sequence-space synaesthesia. The discussion above is limited to vision studies; studies on other modalities are limited in synaesthesia. There is evidence that synaesthesia is linked to absolute pitch (Gregersen et al., 2013) which is also reported for autism (Mottron et al., 1999). Also, synaesthetes experiencing touch are more sensitive in the tactile domain than non-synaesthetes (Banissy et al., 2009). This fits with tactile hypersensitivity reports for autism as assessed with the Glasgow Sensory Questionnaire (Robertson & Simmons, 2013).

Another feature of perceptual processing in autism is that it may be slower or less temporally precise. This has been shown to result in altered temporal integration of global motion patterns (Milne et al., 2002; Robertson et al., 2012) and reduced multisensory integration (Feldman et al., 2018; Stevenson et al., 2014) in autistic individuals. According to the meta-analysis by Feldman et al. (2018), individuals with autism demonstrated reduced audio-visual integration across studies compared to neurotypical controls, and the severity of autism symptoms was reduced in individuals with increased audio-visual integration. Reduced low-level multisensory integration in children with autism (e.g., beeps and flashes) is also correlated with difficulties in higher-level tasks (e.g., speech perception) (Stevenson et al., 2017). For multisensory perception in synaesthetes, findings are mixed. In Neufeld et al. (2012) synaesthetes were less sensitive to the double flash illusion, showing less strong automatic merging of the senses. In Brang et al. (2012), however enhanced susceptibility to this illusion was found. The age of the participants (around 35 in Neufeld et al., and around 20 in Brang et al.) might be a factor as multisensory integration in synaesthetes appeared to decrease faster than in controls with increasing age in Whittingham et al. (2014). In another study, also using slightly older participants, synaesthetes were less sensitive to the McGurk illusion, where auditory-visual integration is crucial (Sinke et al., 2012). These results are an indication that like in individuals with autism, multisensory integration is also altered in synaesthetes, although more studies would be needed to more thoroughly explore parallels with autism.

Shared social and communication characteristics

Although there is currently more consistent evidence for overlap in sensory and perceptual characteristics between autism and synaesthesia, overlap in social and communication aspects has been reported. As defined in the DSM-5 (APA, 2013), impairments in social interaction, language, and communication are important diagnostic criteria of autism. In synaesthesia, results of the Autism Spectrum Quotient for subscales other than AQ-Attention to detail (Social skills, Communication, Attention Switching, and Imagination), sometimes grouped together as AQ-Other (Hoekstra et al., 2008) have been mixed. Ward et al. (2017) reported elevated scores for synaesthetes on all separate AQ subscales, but only the scores on AQ-Attention to detail differed from those of control

participants. In Ward et al. (2018), scores on AQ-Other (grouped together) were significantly higher than those of controls, but scores on AQ-Attention to detail differed from controls even more (group x condition interaction). In Van Leeuwen et al. (2019), synaesthetes scored higher than controls on AQ-Attention to detail and AQ-Social skills, but not on the other subscales. Also in this paper, these was a dose effect of the number of synaesthesia types on AQ-Social skills: the more types of synaesthesia a person had, the higher the AQ-Social skills score. These results, although mixed and not very numerous, highlight that commonalities between autism and synaesthesia may stretch beyond the perceptual domain. Emotional or romantic feelings towards inanimate objects is another social trait linked to autism and synaesthesia (Simner et al., 2019).

Enhanced cognition and savant talent

The development of enhanced behavioural and cognitive skills is a trait that is sometimes observed in autism (Baron-Cohen et al., 2009; Happé & Vital, 2009; Happé, 2018; Pring, 2005; Treffert, 1988, 2009). These abilities are often referred to as savant talent or savant skills, particularly when they exist beyond the abilities of the general population (Treffert, 2009). Individuals diagnosed with autism can express extreme talents in one or multiple behavioural domains, for example in playing a musical instrument (Treffert, 2009) or drawing (Furniss, 2008). Enhanced cognitive skills are also found in autism in the form of highly superior memory and mental calculation skills (Boddaert et al., 2005; Itzchak et al., 2013; Tammet, 2006). Hughes et al. (2017) found that people with a diagnosis of autism who self-reported to have savant skills on a questionnaire were more likely to have synaesthesia (10.0%) than other people with autism (2.9%). This raises the possibility that it is the combination of autism and synaesthesia within the same individual that may give rise to the emergence of prodigious savant abilities.

There have been several accounts of the emergence of savant skills within autism. O'Connor and Hermelin (1991) concluded that autistic savant skills were strongly characterized by the tendency towards repetitive behaviours and a preoccupation with specific topics, with similar conclusions drawn elsewhere (Heaton & Wallace, 2004; Young, 1995). In addition, Baron-Cohen et al. (2009) proposed the hyper-systemizing theory, suggesting that savant skills could arise from autistic individuals' interests in rule-based systems. In a similar vein, the veridical mapping theory describes the emergence of savant skills from the enhanced ability of autistic individuals to detect regularities within and between systems, driven by a tendency towards a local perceptual style (Bouvet et al., 2014; Mottron et al., 2013). As noted above, some of these cognitive traits are also found in synaesthesia even in the absence of autism.

Whilst there are shared cognitive traits between synaesthesia and autism that are relevant to savantism (e.g., sensory sensitivity, attention to details), other accounts emphasize that it is the non-shared, i.e., complementary, traits that combine to lead to savant abilities. Baron-Cohen et al. (2007) suggested that when autism and synaesthesia co-occur the chances of developing savant skills is increased. This suggestion was based on their case study of a man, DT, who had co-occurring autism and multiple types of synaesthesia. DT held the European record for memorising pi to over 20,000 digits, could mentally multiply six-digit numbers at rapid speed, and showed unusual proficiency in language learning. Importantly, DT reports that his synaesthesia helps him to perform these extraordinary skills (Tammet, 2006), possibly due to the richer encoding and retrieval opportunities that his synaesthesia provides. For example, DT reports that his memory and calculation skills are facilitated by his ability to recall numbers in multiple forms—as numerals, colours, shapes, and textures.

In a similar vein, Simner et al. (2009) later suggested that the cognitive advantages associated with synaesthesia (such as enhanced memory; e.g., Rothen et al., 2012; Ward et al., 2019) might be elevated to prodigious savant levels when combined with the autistic tendency to obsess and over-rehearse. That is, each condition contributes different cognitive elements that, when combined, may lead to the emergence of savant skills. Hughes et al. (2019) showed that sequence-space synaesthetes were better able to acquire the autistic savant-like skill of calendar calculation involving calculating the day of the week for any given date. They suggested that synaesthetes' spatial experiences of time-based information may facilitate the learning of relevant time-based skills in a similar way that DT

uses his synaesthetic experiences when performing mathematical calculations.

In summary, there is convincing evidence of a shared profile of atypical perception and attention that is common between autism and synaesthesia. In both cases, these traits fall outside of the historic "core" of these developmental conditions: social problems in autism, and "extra" sensations in synaesthesia. However, they are central to many contemporary accounts, considered in the section below. These traits have been linked to the emergence of savant abilities in autism, which may have parallels in some of the cognitive abilities of synaesthetes. Alternatively, or in addition, synaesthesia and autism may bring together complementary abilities (e.g., aptitude for memory together with repetitive behaviour) that facilitate the emergence of savant skills.

Convergence in theoretical models of synaesthesia and autism

In this section we discuss possible theoretical models of perception in autism and synaesthesia that could provide a common explanation, but for which biological evidence is yet scarce. Specifically, we address accounts of altered cortical excitability and predictive processing models of perception.

Excitation/Inhibition imbalance, hyperexcitability, and stochastic resonance accounts

In autism, an imbalance between excitation and inhibition in the brain has been reported (e.g., Orekhova et al., 2007; Rubenstein & Merzenich, 2003). The excitation/inhibition imbalance model proposes that in the brains of individuals with autism, excitation is not met with sufficient inhibition. Reports include e.g., excess high frequency (gamma) oscillations during sustained visual attention (Orekhova et al., 2007), and specific (social) behavioural impairments and increased high-frequency power after optogenetic elevation of the excitation/inhibition balance in mouse medial prefrontal cortex (Yizhar et al., 2011). The hypothesis of excess excitation has also been explored with regard to differences in the neurotransmitters GABA (inhibitory) and glutamate (excitatory) in the brain, but this research so far has produced rather mixed results (Coghlan et al., 2012; Rojas et al., 2015). Magnetic resonance spectroscopy studies, for instance, report reduced glutamate concentration in the striatum and medial prefrontal cortex, and no change in GABA concentration (Horder et al., 2018), or a reduced influence of GABA on the dynamics of binocular rivalry in autism, while the concentrations of GABA and glutamate in visual cortex were not altered (Robertson et al., 2016). A recent review summarizing the magnetic resonance spectroscopy findings concludes that reports on glutamate concentration are mixed, while GABA is mostly decreased (Rojas et al., 2015).

In synaesthesia, the balance between excitation and inhibition has not been assessed so specifically, but there is evidence for hyperexcitable visual cortex in grapheme-colour synaesthesia (Terhune et al., 2011, 2015). In Terhune et al. (2011), synaesthetes required less stimulation than controls to perceive phosphenes that were induced via transcranial magnetic stimulation over visual cortex, without group differences in motor thresholds (a control location). In a follow-up paper, Terhune et al. (2015) showed that phosphene thresholds were negatively correlated with the concentration of glutamate in primary visual cortex as measured with magnetic resonance spectroscopy, with more of the excitatory neurotransmitter glutamate leading to lower phosphene thresholds. Interestingly, phosphene thresholds also varied with the subjective phenomenology of the synaesthetic experiences (projecting versus associating the synaesthetic colours, van Leeuwen, 2013), but all synaesthetes had lower thresholds than controls. Altogether these results imply hyperexcitability in the visual cortex of grapheme-colour synaesthetes.

The wider phenotype of synaesthesia in terms of sensory sensitivity and hyperexcitability led Lalwani and Brang (2019) to propose a stochastic resonance model of synaesthesia. They propose that synaesthesia can develop because of excessive neuronal noise in synaesthetes' brains, specifically in brain regions processing the modality of the synaesthetic concurrent sensation. This excess noise would allow signals coming through pre-existing multisensory pathways to reach strong enough levels to lead to co-activation of concurrent (e.g., colour) with inducer (e.g., letter) representations, which would develop into stable synaesthetic experiences over time. A similar proposal of enhanced noise in sensory processing has been used to explain altered

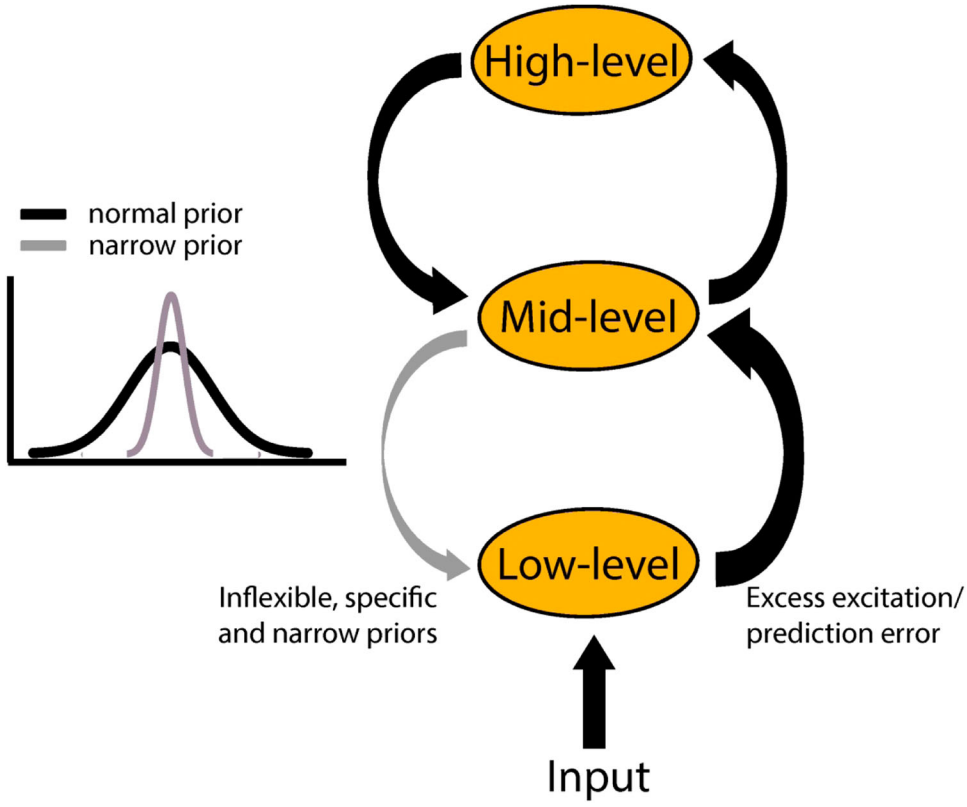

Figure 2. Predictive coding account of autism and synaesthesia. When input arrives at low-level brain areas, excitatory activity travels up the brain's hierarchy through feedforward connectivity (black upward arrows). Signals from higher order areas (downward arrows) based on higher or mid-level predictions (priors) are integrated with the feedforward excitatory signal from low-level areas, leaving some prediction error. In the case of autism and synaesthesia, however, inhibitory feedback from mid-level areas may be limited due to overly specific priors (narrow gray downward arrow), leaving excess excitatory activity (thick black upward arrow) to linger in the brain (excess prediction error). In synaesthesia, high-level priors are not proposed to be affected while overly specific mid-level priors could be the mechanism underlying synesthetic perception; in schizophrenia, high-level priors may be modified to fit with the altered sensory evidence, leading to hallucinations. For autism, the specific role of low vs high-level priors remains unclear to date [To view this figure in colour, please see the online version of this journal].

perception in autism (Simmons et al., 2009). An alternative account proposed reduced neural noise in autism, but does not seem to cover all aspects of altered perception (Davis & Plaisted-Grant, 2015), for instance, how low noise would lead to heightened subjective sensory sensitivity.

Predictive processing models of perception

Predictive processing accounts of perception involve the comparison of top-down knowledge (termed "priors") against incoming sensory signals. Priors are assumed to be probabilistic, and can consist of representations of statistical regularities of the sensory world (e.g., 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.

One version of this model suggests that autism reflects weaker priors such that people with autism are more likely to see the world as it is (Pellicano & Burr, 2012). The anti-thesis of this would be an over-reliance on prior knowledge to such an extent that the priors override the sensory evidence and generate illusory sensations. This has been put forward to explain hallucinations in schizophrenia (Sterzer et al., 2018). A dichotomy between perceiving the world as hyper-real (autism-like) versus surreal (schizophrenia-like) places synaesthesia in a grey zone. The atypical perceptual experiences of synaesthesia resemble an over-reliance on top-down priors as in the standard account of hallucinations in schizophrenia, but yet synaesthesia is known to co-occur with autism. (Evidence for increased co-occurrence between synaesthesia and schizophrenia is presently lacking, although synaesthetes report increased trait

schizotypy on some subscales, Banissy et al., 2012, and there is suggestive genetic evidence, Tilot et al., 2019).

There is an alternative account of altered perception in autism such that priors are overly specific, narrow, and inflexible (Van de Cruys et al., 2014). In this view, incoming sensory evidence tends to get treated as surprising and unpredictable. This explanation of autism is potentially more compatible with synaesthesia. Seth's (2014) predictive processing account of synaesthesia makes a distinction between mid-level and high-level priors. Mid-level priors are assumed to be strong and inflexible such that they can over-ride the sensory signals giving rise to the "false" perceptions of synaesthesia. High-level priors, on the other hand, concern things such as beliefs and knowledge. A synaesthete knows their synaesthesia is objectively false, i.e., that it reflects an internally generated sensation rather than being a property of the world like the inducer (even though synaesthetes are not always aware that others do not share their experience). But this knowledge is not sufficient to over-rule the strong mid-level prior so the experience persists (see Figure 2). In schizophrenia, a similar account is used to explain the presence of hallucinations by mid-level priors but, unlike synaesthesia, the high-level priors are modified to be consistent with their experiences. That is, they believe in the veracity of their unusual experiences and develop narratives consistent with that (i.e., delusions that explain them away). On this view, schizophrenia versus autism and synaesthesia would not be polar opposites but, instead, all three would have something in common: they would all have strong and inflexible priors (relative to neurotypicals), see Figure 2. In some cases this would generate overt false perceptions (synaesthesia, schizophrenia) but only in the case of schizophrenia would these be taken as real.

Although many researchers regard the predictive processing framework as being useful for explaining altered perception, in practice it can be hard to tease apart the underlying mechanisms. This is because the same symptom manifestation can arise from different mechanisms. For instance, a reliance on sensory evidence can occur either because the sensory evidence itself is more reliable or because the priors are less specific. Similarly, lower susceptibility to visual illusions could arise either from less reliance on priors or from lower-level deficits in perceptual grouping (Uhlhaas & Silverstein, 2005; van Leeuwen et al., 2018).

In sum, autism and synaesthesia may converge on exhibiting excessively strong priors, leading to altered perception. Overly specific, inflexible priors are fitting with an imbalance in excitation and inhibition in the brain: if priors are too specific to properly predict incoming sensory signals, i.e., they only predict a very specific type of input, this results in increased prediction error in the brain. In that sense, the different theoretical accounts of synaesthesia and autism all converge on an interpretation with excess low level activity or neuronal noise, and overly specific, but poorly functional top-down, inhibitory predictions (Figure 2). It is a question for future research whether the imbalance in the system originates from excess noise to begin with, or whether the tendency to create overly specific priors comes first (van Leeuwen, 2014).

Summary and future directions

Summarizing the parallels between autism and synaesthesia so far, we can state that the link between the two conditions has been firmly demonstrated with different methods. For instance, showing that synaesthesia is more common among people with autism and that synaesthetes possess more autistic traits. Clear evidence for genetic overlap is currently lacking although a large family study reports both synaesthesia and autism running in one family (Bouvet et al., 2019). In brain connectivity, a predominance of local over global connectivity is a possible pattern in both autism and synaesthesia, but more studies involving synaesthetes are needed to confirm this with direct comparisons between autism and synaesthesia. With regard to sensory processing, both groups show altered sensory sensitivity and in perceptual tasks, evidence for an autistic-like, detail-oriented style in synaesthesia is accumulating. Synaesthetes also show enhanced autistic traits in social domains. When synaesthesia and autism are experienced together, exceptional talents may emerge. Theoretical accounts of synaesthesia and autism seem to converge on a model in which priors are too specific, resulting in excess excitation in the brain because sensory input cannot be predicted well.

We believe the emerging evidence for overlaps between autism and synaesthesia have been

established firmly enough to warrant further confirmation of these links in future studies (e.g., in genetics, neural connectivity patterns, sensory processing, etc.). However, the mechanisms by which overlap is mediated remain more elusive. For sensory sensitivity in synaesthetes, objective measures of sensory thresholds and additional questionnaire methods could confirm the so far reported alterations in sensory sensitivity across different sensory modalities. Importantly, in adult synaesthetes we cannot be sure whether such sensitivities are the cause or consequence of experiencing synaesthesia for a long time (Ward, 2019). Therefore, exploring these issues in child synaesthetes would be a promising approach. When it comes to perception, it would be good to include different types of synaesthetes in perceptual studies and multisensory tasks. Does it matter what type of synaesthesia someone has, or is the amount of different types of synaesthesia crucial? Is multisensory integration in synaesthetes really altered compared to non-synaesthetes? And do any multisensory alterations resemble the patterns observed in autism? Given that the world around us is essentially multisensory in nature, and most natural learning happens in multisensory environments, it is essential to further our understanding of alterations in multisensory perception in the two groups.

One aspect to consider when studying autism and synaesthesia is that both conditions are relatively rare in the general population. Recruiting sufficient participants for meaningful analyses can be a challenge, especially when investigating individuals with autism *and* synaesthesia. In autism, it has been established that there is a continuum in autistic traits in the general population, ranging from a low to a high degree of autistic traits for each individual (e.g., De Groot & Van Strien, 2017). These sub-clinical autistic tendencies are widely recognized as being relevant for understanding clinically relevant autism, e.g., by studying elevated autistic traits in family members (Wheelwright et al., 2010). This provides the opportunity to also recruit individuals with a high degree of autistic traits into experimental studies, for instance assessed with the AQ (Baron-Cohen et al., 2001). For synaesthesia, there is evidence that synaesthetic consistency lies on a continuum in the general population (Bien et al., 2012; Burghoorn et al., 2020; Cuskley et al., 2019), which could be regarded as a "degree of synaesthesia", or, put differently, the strength of crossmodal associations in different individuals. It is disputed, however, whether one can really be a synaesthete to a certain degree, or whether the term should be reserved for those who have the phenomenological experience of synaesthesia (Ward, 2019). Nonetheless, given that the tendency to associate stimuli from different modalities with each other varies in the general population, one could attempt to recruit those with synaesthesia-like consistency scores into studies, even if overt synaesthetic experiences are not present.

We would like to stress that our review does not only pertain to milder forms of autism, given that the initial screening studies (Baron-Cohen et al., 2013; Neufeld et al., 2013) included non-biased populations recruited from general autism databases and/or the clinic. In Baron-Cohen et al. (2013) many participants were not able to complete a self-test for synaesthesia because they experienced it as quite overwhelming. Also, a case-study (Bouvet et al., 2014) concerned an individual with limited verbal abilities and synaesthesia.

With regard to the theoretical models of synaesthesia and autism, convergence on strong, inflexible priors and excess sensory excitation inspires the question of how a system develops into this final state. What comes first—a tendency to form inflexible, highly specific predictions, or noisy cortical processing with a lot of excitatory activity in primary sensory areas (van Leeuwen, 2014)? According to the stochastic resonance model of synaesthesia, excess neuronal noise in the system comes first, which is a hypothesis that could be tested explicitly. We feel that in this domain, longitudinal studies in children would be a promising method to pursue—in adults cause and consequence are no longer separable. Investigating the balance between excitatory and inhibitory neurotransmitters in both groups in more detail across the cortex would also provide more information about excitation and inhibition in primary vs associative areas of the brain.

Finally, although the focus of this manuscript has been on the association between synaesthesia and autism we do not dismiss the possibility of associations of synaesthesia to other mental health conditions. There is already evidence of links between synaesthesia and anxiety (Carmichael et al., 2019) and between synaesthesia and PTSD, post-traumatic stress disorder (Hoffman et al., 2019). A possible link

with schizophrenia has already been discussed (Tilot et al., 2019). Whatever the mechanisms behind these associations are, they are likely to require a reconceptualization of synaesthesia not just in terms of anomalous experiences (e.g., numbers evoking colour) but as a broader neurocognitive phenotype (Ward & Filiz, 2020).

To conclude, we stress the importance of studying synaesthesia in relation to autism: informing individuals with autism about synaesthesia can assist in formulating better tuned treatment plans, help to identify subtypes of autism (Happé et al., 2006), and further our understanding of mechanisms of autism. If multiple types of synaesthesia are associated with an increase in autistic traits, it would be interesting to investigate whether undiagnosed autism exists more commonly in synaesthetes compared to non-synaesthetes, and similarly, we could study why some people develop synaesthesia while others develop autism (with or without additional synaesthesia).

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ORCID

Tessa M. van Leeuwen http://orcid.org/0000-0001-7810-6348

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