

Opinion piece



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Synaesthesia: a distinct entity that is an emergent feature of adaptive neurocognitive differences

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In this article, I argue that synaesthesia is not on a continuum with neurotypical cognition. Synaesthesia is special: its phenomenology is different; it has distinct causal mechanisms; and is likely to be associated with a distinct neurocognitive profile. However, not all synaesthetes are the same, and there are quantifiable differences between them. In particular, the number of types of synaesthesia that a person possesses is a hitherto underappreciated variable that predicts cognitive differences along a number of dimensions (mental imagery, sensory sensitivity, attention to detail). Together with enhanced memory, this may constitute a common core of abilities that may go some way to explaining why synaesthesia might have evolved. I argue that the direct benefits of synaesthesia are generally limited (i.e. the synaesthetic associations do not convey novel information about the world) but, nevertheless, synaesthesia may develop due to other adaptive functions (e.g. perceptual ability, memory) that necessitate changes to design features of the brain. The article concludes by suggesting that synaesthesia forces us to reconsider what we mean by a 'normal' mind/brain. There may be multiple 'normal' neurodevelopmental trajectories that can sculpt very different ways of experiencing the world, of which synaesthesia is but one.

This article is part of a discussion meeting issue 'Bridging senses: novel insights from synaesthesia'.

1. Introduction

The aim of this paper is to consider the phenomenology of synaesthesia, its defining characteristics, and relation to similar entities. In particular, the paper makes the claim that synaesthesia is not on a continuum with neurotypical cognition, by which it is meant that one cannot rank non-synaesthetes along some hypothetical synaesthesia spectrum. However, there may be continuity in the underlying disposition to develop synaesthesia which, among synaesthetes, results in some synaesthetes having more extreme profiles (more types of synaesthesia, more atypical cognition, and—presumably—more atypical brains). Finally, the paper will argue that the benefits of having synaesthesia lie primarily in the accompanying cognitive profile than in the (defining) unusual experiences themselves.

2. Synaesthesia and its fringes

In this section, three defining characteristics of synaesthesia are proposed and discussed in relation to certain other phenomena that share some of these characteristics (the list is not exhaustive and readers are referred to chapters in Deroy [1]). It has been suggested that the definition of synaesthesia might be overhauled as we learn more about the causes of synaesthesia [2]. The view articulated in this paper is that the definition of synaesthesia will remain relatively fixed because it is based on the subjective experiences of synaesthetes. But a better scientific understanding of synaesthesia will give us a much better idea as to where synaesthesia sits in relation to other phenomena.

The three characteristics are as follows:

- (1) Synaesthesia consists of an inducer (the triggering stimulus) and a concurrent (the elicited synaesthetic experience) [3]. Both the inducer and concurrent coexist so, for instance, a sound-vision synaesthete would both hear a sound (the inducer) as well as see it (as a synaesthetic concurrent).
- (2) Synaesthesia is involuntary. It is not under volitional control. This is typically demonstrated through Stroop-like paradigms in which task-irrelevant synaesthetic colours interfere in other tasks [4]. I, and others, have previously referred to this as 'automaticity' but this term has been criticized for implying inevitability [5]. Synaesthesia is not always inevitable (e.g. if an inducer is subliminal or unattended), as discussed later.
- (3) Synaesthetic experiences (concurrents) are percept-like. The inducer need not be percept-like and could be conceptual.

There are other phenomena that share some, but not necessarily all, of these characteristics. This has led to some uncertainty and disagreement as to where synaesthesia begins and ends. To give a flavour of this debate, I shall consider three examples: that of mental imagery, learned associations and grapheme personifications.

Synaesthetic concurrents are like mental images. Mental images are percept-like experiences that can extend to different modalities (not just vision) and could be projected externally. Indeed synaesthetes tend to report more vivid mental imagery in everyday life (i.e. beyond their synaesthesia itself), such as when imagining a fictitious scene (e.g. [6]) or recalling a previous memory [7]. This suggests that the connection between synaesthesia and mental imagery is more than a superficial resemblance. However, most mental imagery does not have the characteristics of being involuntary or induced [8]. Another example of involuntary mental imagery is flashbacks in PTSD (post-traumatic stress disorder), and grapheme-colour synaesthesia is a significant risk factor for this following trauma [9].

With regards to learned associations, these fulfil the first and second criteria (induced and involuntary), with the third criteria (percept-like) open for debate. For many years, the consensus view was that synaesthetic associations were not learned. However, some role of learning cannot be denied based on, for instance, some degree of correspondence between synaesthetic associations and childhood coloured letters [10]. Perhaps the more interesting question is why a synaesthete might continue to re-experience, in a percept-like form, certain learned associations for the entire duration of their life. It may be that synaesthesia is influenced by the environment, including idiosyncratic pairings (e.g. from books) or other regularities (e.g. cross-modal correspondences such as high pitch being bright or pointy) [11]. But this is not the same thing as arguing that it is caused by the environment alone, as might be the case for other forms of association.

With regards to whether synaesthetic experiences must be percept-like, some synaesthesia researchers (perhaps the majority of them) have been willing to downplay this characteristic in order to allow grapheme personification (e.g. thinking of '5' as male or bossy) to be called a type of synaesthesia. The evidence suggests that personification and more canonical types of synaesthesia (e.g. grapheme-colour) tend to co-occur [12], and it is binary in nature (either present or absent). So there is indeed an important connection that

needs to be explained. However, we would not want to call vivid mental imagery a type of synaesthesia just because it co-occurs with it, and the same logic can be extended to personifications. If we were to lose the third characteristic (percept-like) then synaesthesia would have the very broad definition of being any kind of involuntary association. Ultimately what is needed is a clearer model that explains why some kinds of associations are linked to synaesthesia and others are not (at which point disagreements about terminology become irrelevant).

It is important to make a distinction between the characteristics of synaesthesia and the causes of synaesthesia (cf. [2]). This is because the same set of characteristics could emerge from multiple causes. A broad distinction exists between developmental (or idiopathic) synaesthesia and acquired synaesthesia. Acquired synaesthesia can emerge from compensatory plasticity following sensory loss [13], or transiently from certain psychoactive substances [14], although it is largely unknown how these brain mechanisms relate to developmental synaesthesia. Moreover, attempts to train adults to have grapheme-colour synaesthesia clearly tick all the characteristics for synaesthesia listed above [15]. But trained synaesthesia has a different causal mechanism (unless one argues that child synaesthetes trained themselves deliberately) and has other characteristics (transient rather than lifelong associations). Trained synaesthetic associations could be construed as a phenocopy: an environmentally created phenotype that resembles one that is determined by genetic factors.

In short, synaesthesia can be defined in terms of a core set of characteristics (phenomenology-based) that may emerge from several different, but potentially related, causes. The current focus is on developmental synaesthesia and, in the next section, I argue that this is not on a continuum with neurotypical cognition.

3. Synaesthesia is special: it is not on a continuum with neurotypical cognition

By 'special' I mean that synaesthesia is phenotypically distinct. We are not all synaesthetes, not even weakly so (e.g. [16]). The contrary view is that synaesthesia lies on a continuum with neurotypical cognition such that all people can be ranked according to the degree with which they have synaesthesia (e.g. [17]). One of the challenges for defending the position of synaesthesia being special is that synaesthesia clearly emerges from variations in genetics and brain architecture within the general population. Synaesthetes are not from the planet Mars. The claim made here is that while these starting neurodevelopmental differences may be subtle (and possibly continuously distributed), they nevertheless lead to a different developmental trajectory with a phenotypically different outcome (synaesthete versus non-synaesthete). In the sections below, I defend the position that synaesthesia is special with both a conceptual argument (the impossibility of a half-synaesthete) and with reference to existing evidence.

(a) The impossibility of a half-synaesthete

If all people can be ranked according to the degree with which they have synaesthesia, then what are the characteristics of a hypothetical 'half-synaesthete'? That is, someone midway between a canonical synaesthete and a canonical

non-synaesthete. A number of possibilities can be considered for such a continuum: the number of inducers and concurrents; the vividness of concurrents; the consistency of associations; and strength of traits linked to synaesthesia.

Firstly, people may differ in terms of how prolific their synaesthesia is (the size of the set of inducers and/or concurrents). This variability undoubtedly occurs and, below, I will argue that it is important. But could we take two non-synaesthetes (i.e. for whom the set size of inducers and concurrents is zero) and rank them on some sort of synaesthesia spectrum? I would argue not. Of course, these individuals could be ranked as equal to each other, but this has the effect of placing all non-synaesthetes into a single rank, thus creating a distinct category. There are some borderline cases here. If a person has only one single association (e.g. Tuesday is yellow) then is this person a synaesthete? According to the definitional criteria above, they would be a synaesthete (and not a half-synaesthete) because there is nothing in the criteria about how many associations a person needs. However, we might wonder whether this has a different cause (a learned association) from more canonical cases.

Secondly, the vividness of the synaesthetic concurrent(s) can vary continuously as can its degree of involuntariness. But this generates the same response as before: how do we rank the non-synaesthetes for whom these experiences do not exist? They would have to be ranked equally thus forming a distinct category of non-synaesthetes.

Thirdly, the consistency of the associations appears to vary on a continuum (e.g. when measured using conventional diagnostic tests of synaesthesia; [18]). Here, we have to be careful to distinguish our measurement tool (clearly continuous in nature) from the thing we are trying to measure (which may or may not be continuous). Synaesthetes vary in their ability to perform these tests (e.g. because they cannot choose a metallic gold to represent their experience) as do non-synaesthetes (e.g. because they employ effective or ineffective strategies). However, there is no good reason to rank a non-synaesthete who uses an effective strategy on this test as being 'more synaesthetic' than a non-synaesthete who does not. Consistency tests are a useful diagnostic heuristic, but it is uncertain what they reveal beyond that.

Finally, there are traits linked to synaesthesia that vary continuously in the population. These include mental imagery, memory and certain perceptual abilities (all considered in more detail below). Here, it is possible to rank non-synaesthetes along a continuum rather than grouping them all together, although it becomes a leap of the imagination to argue that someone high in these traits (but lacking any inducer-concurrent pairings) is a 'half-synaesthete'. A better way of describing this scenario might be in terms of endophenotypes: heritable characteristics that cluster with a condition. Indeed, there is some preliminary evidence that non-synaesthetic relatives of synaesthetes show some of these associated traits [6,19], and this is an important avenue for future research. To be clear, the claim that synaesthesia is phenotypically special does not preclude the possibility that other individuals share certain other aspects of behaviour, brain configuration, or genetic predisposition with synaesthetes (they almost certainly will). An analogy could perhaps be made between having the 'ingredients' for generating synaesthesia and the finished product itself: it may be possible to have the former without the latter (e.g. stochastic processes in development; [20]).

So where does this preamble lead us? Figure 1 outlines a summary of my claims. Synaesthesia is binary: people either have it or they do not. However, not all synaesthetes are alike: for instance, they differ in terms of their phenomenology (vividness, automaticity) and extensiveness (the number of types of synaesthesia they have). In this respect, you could argue that synaesthetes themselves lie on a continuum (they are more or less extreme in nature), but attempting to put non-synaesthetes on that continuum would simply recreate a categorical difference (all non-synaesthetes ranked the same). In the sections below, I put forward a case for the notion of a 'synaesthetic disposition'. This consists of three things: a predisposing genotype, a characteristic neural architecture and a distinct cognitive profile (or endophenotype as introduced above). People with a high synaesthetic disposition have a high probability of developing synaesthesia and, I will propose, have a high probability of developing multiple kinds of synaesthesia to such an extent that the number of types of synaesthesia that a person possesses acts as a proxy marker for their synaesthetic disposition. In theory, everybody (synaesthete and non-synaesthete) could be ranked according to their synaesthetic disposition but that does not make all people synaesthetic (hence the analogy between ingredients and finished product). The synaesthetic disposition will almost certainly have multiple components and it is the combination of components (rather than a single 'magic' ingredient) that makes it distinctive. The probabilistic notion of a disposition also means that someone could have a high synaesthetic disposition and not be a synaesthete. But these people would also have distinctive cognitive profiles, be more likely to produce children who develop synaesthesia, and perhaps be more susceptible to synaesthesia-like experimental manipulations [21]. In later sections, I shall also argue that it is this synaesthetic disposition (rather than synaesthesia itself) that has an adaptive value in evolutionary terms.

(b) Evidence for a distinct cognitive profile of synaesthesia

The previous section introduced the idea of different 'ingredients' (genetics, brain architecture, cognition) that might ultimately lead to the emergence of synaesthesia. This idea is picked up in detail in this section and the following one. One of the fundamental issues is that of unpicking causes and effects. For instance, grapheme-colour synaesthetes have a more excitable visual cortex (as shown by phosphene thresholds with transcranial magnetic stimulation; [22]) but is a hyperexcitable visual cortex a cause of synaesthesia, or a consequence of it? These kinds of question are far from straightforward to address but there are a number of methods for tackling them. One can explore the time course of development of synaesthesia in childhood (e.g. [23]), although the evidence here is presently sparse. One can also explore, in adults, whether these kinds of differences are specific to one kind of synaesthesia (i.e. related to the specific pattern of inducers and concurrent) or are a more general feature of having synaesthesia (in which case it would be a stronger candidate for being part of a start-up kit for the emergence of synaesthesia). Creating 'phenocopies' of synaesthesia (through training) is also an approach that speaks to this issue by providing a proof of principle. Finally, neural network models of synaesthesia may also offer a proof of principle as to how

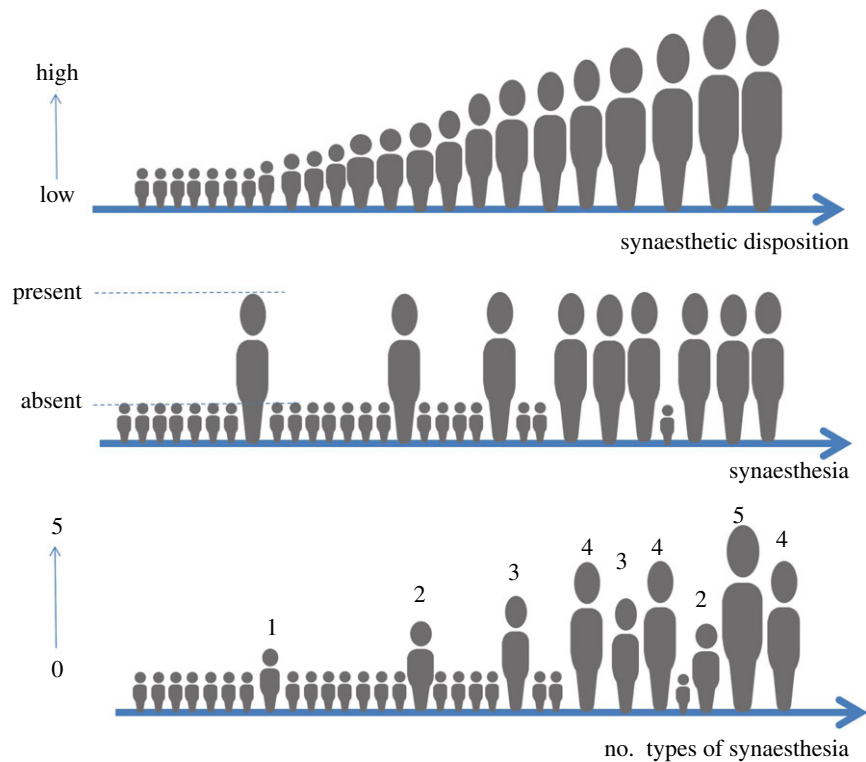


Figure 1. A synaesthetic disposition (consisting of a characteristic genotype and neurocognitive profile) increases the likelihood of synaesthesia emerging (people have it or not) and increases the likelihood of different kinds of synaesthesia emerging (some synaesthetes are more extreme than others).

different parameters of a model (or its inputs) can lead both to the emergence of synaesthesia and other associated traits [24].

Certain changes in perception are candidates for being linked to synaesthesia more broadly (i.e. irrespective of the type of inducer or concurrent). The Glasgow Sensory Questionnaire measures changes in subjective sensory sensitivity to different sensory modalities with items such as ‘Do bright lights ever hurt your eyes/cause a headache?’ and ‘Do you hate the feel or texture of certain foods in your mouth?’ [25]. Within the neurotypical population, items tend to load on a single factor suggesting that individual differences in sensory sensitivity are multi-modal and are not strongly tied to different senses [25]. Conditions such as autism and synaesthesia show the same trend (multiple senses affected) albeit with much higher scores than the neurotypical population [26]. Thus, a grapheme-colour synaesthete will report heightened sensory sensitivity but not just to vision, to all other senses. Ward *et al.* [27] explored whether this is because they have additional kinds of synaesthesia. Having additional types of synaesthesia (e.g. tickertape, sequence-space, lexical-gustatory, music-colour) does matter: but it is the number of kinds of synaesthesia that matters rather than the specific types. Having more types of synaesthesia is linked to greater sensory sensitivity across all modalities, and this was referred to as a dose-like effect. So the visual sensory sensitivity of a grapheme-colour synaesthete depends on whether he/she also has additional types of synaesthesia such as lexical-gustatory or tickertape synaesthesia. Moreover, having more types of synaesthesia is linked to increased sensory sensitivity for modalities that have nothing to do with their synaesthesia (e.g. vestibular sensitivity such as dizziness). This set of results might seem very counterintuitive: but only if one holds to the belief that it is the types of synaesthesia that are generating the changes in sensory sensitivity. If, instead, increased subjective sensitivity is a predisposing influence for (all kinds of)

synaesthesia then the results are logical: the greater the sensory sensitivity then the greater the probability of developing synaesthesia—and the greater the probability of developing multiple kinds of synaesthesia.

One of the original motivations for exploring atypical sensory sensitivity is that this is a feature of autism spectrum disorder, and synaesthesia and autism are known to co-occur more than chance [28,29]. Autism is defined by a cluster of symptoms that have traditionally focused on socio-communicative impairments and limited repertoire of interests, but now includes atypical sensory sensitivity. It is also linked to certain perceptual abilities such as the ability to detect local details in complex arrays of stimuli (e.g. [30]). Although a formal diagnosis of autism is made via a clinician, there are various informal instruments that are sensitive to autism and have the advantage of being able to measure sub-clinical differences in autistic tendencies. The AQ (autism spectrum quotient) is a 50-item scale that includes five subscales: (poor) social, (poor) attention-switching, (good) attention-to-detail, (poor) communication and (lack of) imagination [31]. Synaesthetes have significantly elevated AQ scores, but with a disproportionately elevated score on attention-to-detail [26,32]. This is the only AQ subscale that measures an ability (rather than impairment) and, indeed, the average synaesthete can perform difficult objective attention-to-detail tasks (change blindness) far better than most people, and as well as people with very high AQ scores (greater than 2 s.d. from the average) [32]. As such, synaesthetes possess many of the abilities of autism without necessarily incurring the impairments (and it is hard to imagine how good ability on a task such as change blindness has anything directly to do with having coloured graphemes). However, there is a genuine risk of synaesthetes having the full range of autistic symptoms too and this risk, together with attention-to-detail, shows a dose-like function: more types of synaesthesia are

linked to higher levels of autistic traits [32]. One might wonder whether any shift up the autism spectrum disproportionately increases sensory symptoms before other non-sensory symptoms emerge, but this is not the case. Anorexia nervosa is associated with elevated AQ scores but the affected subscales centre on socio-communicative problems and not attention-to-detail [33]. As such, there may be at least two vulnerabilities to autism: one that is centred on sensory symptoms (synaesthesia-like) and one that is centred on social dysfunction (anorexia-like). It is unclear how the genetics and brain wiring of synaesthesia and autism will turn out to be related; however, such a relationship is predicted by the behavioural data. Autism is often characterized as exuberant local connectivity often at the expense of global connectivity [34]. It is interesting to note how the ‘adjacency theory’ of Ramachandran *et al.* and Hubbard *et al.* [35,36] also explains the particular pattern of inducer-concurrent pairings in terms of increased local connectivity (e.g. graphemes and colour pair together because they are adjacent).

There are some instances in which the cognitive profile of synaesthetes differs from that reported in autism: notably, in the domains of imagery, creativity and memory. Although some high-functioning people with autism, such as Temple Grandin [37], describe thinking in rich visual images this is not generally the case when it is measured on standard questionnaires [38]. Indeed, the AQ contains the ‘imagination’ subscale in which low mental imagery is considered an indicative feature of autism [31]. By contrast, synaesthetes report vivid mental imagery and this can extend across multiple senses [39]. Is it the type of synaesthesia that matters or the amount of synaesthesia (i.e. number of types)? Spiller *et al.* [39] reported that both of these factors are important, including a similar dose-like effect to that noted earlier for sensory sensitivity. So the vividness of visual imagery of a grapheme-colour synaesthete will be higher if he/she reports other, seemingly unrelated, kinds of synaesthesia such as lexical-gustatory (and similarly for imagery involving other senses). This, again, suggests that increased mental imagery (across multiple senses) is a predisposing influence on synaesthesia although specific modalities might get further enhanced as a consequence of particular kinds of synaesthesia developing.

Creativity has been reported to be increased in synaesthesia in terms of both psychometric tests [40,41] and artistic inclinations (e.g. [42,43]). On a psychometric test of creativity (the Remote Associates Test), Ward *et al.* [41] noted that having more types of synaesthesia was linked to higher scores on this measure. These differences in creativity and imagery-based thinking are also tied to a commonly found difference in personality traits among synaesthetes. Namely, synaesthetes report significantly higher ‘openness to experience’ and this itself is correlated with the number of types of synaesthesia that a person has [44].

With regards to memory, interest in the relationship between synaesthesia and memory has been piqued by fascinating case studies such as that of Shereshevskii [45] with seemingly unlimited memory capacity, and that of Daniel Tammet [46] who, through his combination of synaesthesia and autism, can learn Icelandic in a week and memorize pi to over 20 000 decimal places. A recent meta-analysis of episodic memory ability by grapheme-colour synaesthetes on standard laboratory tests (e.g. word lists, recognition memory of scenes) shows significantly better memory ability

with a Cohen’s *d* of around 0.6 [47]. This was found for both stimuli that induce synaesthesia (e.g. words, digits for these synaesthetes) and those that do not (e.g. visual scenes). This suggests that memory ability is not closely tied to the nature of the synaesthesia, although further research is needed on more diverse types to back this up. The meta-analysis found significantly weaker benefits in tasks of working/short-term memory (Cohen’s *d* = 0.36) even when the material involved synaesthetic inducers (e.g. digit span) [47]. The differences in memory ability of synaesthetes may, in some way, be related to other cognitive differences such as in (non-synaesthetic) imagery and perception. Adult synaesthetes report unusually vivid sensory details of childhood memories, and from earlier in life [7]. Functional imaging studies show differences in visual cortex when synaesthetes (relative to controls) are recognizing and retrieving visual memory associations to fractals, i.e. a stimulus that is not directly relevant to their synaesthesia [48].

It is worthwhile elaborating on whether these differences in long-term memory ability are likely to be important given that other synaesthesia researchers have been underwhelmed by the findings: referring to them as ‘small’ [49] and ‘ordinary’ [50]. Figure 2 shows what two populations would look like if they differed by an effect size of 0.6. This shift in ability would place the average synaesthete at the 73rd percentile relative to non-synaesthetes—a difference that will almost certainly have importance in the real-world. Nevertheless, there is considerable overlap between the distributions. Some synaesthetes, in fact, have worse memory than the neurotypical average. It would be tempting, but wrong, to claim that these synaesthetes do not have enhanced memory. The important question is: enhanced relative to what? One cannot take the neurotypical mean ($z = 0$) as the benchmark. Instead, one needs to consider the level of memory functioning that these same people might have had if they did not have synaesthesia. This is not an abstract, philosophical argument. It can be looked for in the data. If synaesthesia enhances memory for everybody then the mean will shift upwards but the shape of the distribution will be the same, and this appears to be the case when real data is plotted (also figure 2). By contrast, if only half of the synaesthetes have a memory advantage and half are like non-synaesthetes then the distribution would be squashed (shorter and broader) as well as shifted.

Considering the upper tail of the distribution, most synaesthetes do not have exceptional memory. From this fact, it would be tempting, but erroneous, to claim that there is no relationship at all between synaesthesia and exceptional memory. However, the nonlinearity of the normal distribution means that the further along the upper tail one samples, then the relative proportion of synaesthetes goes up and up. Thus, a high proportion of synaesthetes in the exceptional tail-end is predicted by even a moderate (0.6) shift in ability: we would expect synaesthetes to outnumber non-synaesthetes by nearly 7-to-1 in the region above three standard deviations of the neurotypical mean (all things being equal). If one ranks 1000 people by memory ability then the person in that distribution who is statistically most likely to have synaesthesia is the 1000th (i.e. most extreme) person. Although I have couched this argument with respect to memory, it applies to any of the potential cognitive enhancements linked to synaesthesia discussed above. Thus, even moderate effect sizes are theoretically compatible

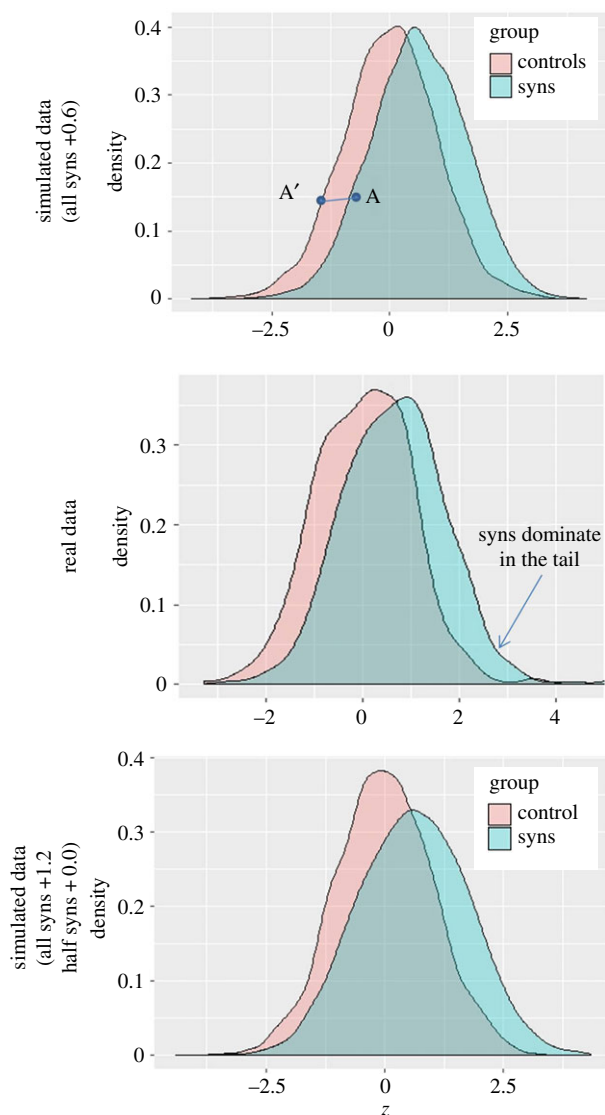


Figure 2. The top panel shows simulated data (3000 points in each group) based on a standard normal distribution with ‘synaesthetes’ shifted by $z = +0.6$. The bottom panel shows simulated data (3000 points in each group) based on a standard normal distribution with ‘synaesthetes’ drawn evenly from two distributions ($z = +1.2$ or $z = 0.0$). The middle panel shows actual memory data from published studies from our research group. It is possible to have ‘enhanced’ memory without memory performance being above average ($z > 0$) or exceptional ($z > 2$) with respect to neurotypical memory ability. Synaesthete A has below average memory but is nevertheless better than what might otherwise be expected (shown by A’). (Online version in colour.)

with the idea that all synaesthetes show unexpected differences in ability (i.e. a distinctive neurocognitive profile), and population-based moderate effect sizes can be a driving force for exceptional talents linked to synaesthesia.

If synaesthetes have a distinct cognitive profile then could we separate synaesthetes from non-synaesthetes on cognitive scores alone? This question is important primarily as a scientific question rather than a diagnostic one (given that we already have good diagnostic measures). Although the cognitive findings are reliable (i.e. have been replicated), they tend to be medium effect sizes (Cohen’s d between 0.5 and 0.8). This is inadequate for categorizing any individual based on any given test score. However, there is a reason to believe that a collective pattern across multiple tests could classify

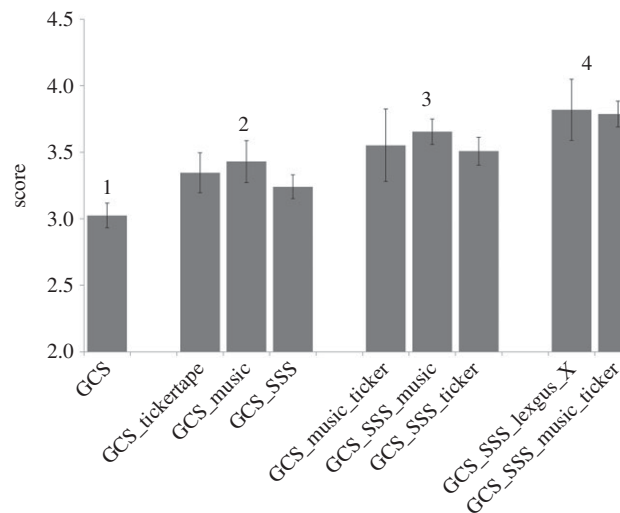


Figure 3. The CLaN questionnaire asks questions about the phenomenology of grapheme-colour synaesthesia (the extent to which it is externally localized, automatic/involuntary and deliberately used). Having additional types of synaesthesia results in more extreme scores on this measure (see electronic supplementary material for further information). Questions are answered on a 1–5 scale (with a higher score indicative of more extreme synaesthesia, after reverse coding). GCS, grapheme-colour synaesthesia; music, musically induced colours; SSS, sequence-space synaesthesia; lexgus, lexical-gustatory synaesthesia. (Online version in colour.)

participants using machine-learning approaches. This would constitute evidence of distinctiveness and this is an area for future research.

People with multiple kinds of synaesthesia not only have more extreme cognition, they also tend to have more extreme phenomenology. The CLaN (Coloured Letters and Numbers) questionnaire asks about various aspects of grapheme-colour synaesthesia including the extent to which colours are localized externally (being a projector), the extent to which the colours appear automatically, and how much they use it in everyday life [51]. Figure 3 shows that grapheme-colour synaesthetes with additional kinds of synaesthesia have more extreme scores on this measure (see electronic supplementary material for further details). Again, it seems strange to think that having ‘extra’ kinds of synaesthesia directly drives more extreme grapheme-colour phenomenology (or vice versa). Instead, the suggestion here is that a synaesthetic disposition acts as a latent variable that drives multiple differences. The synaesthetic disposition manifests itself in several ways: as a more distinctive cognitive profile; as driving the emergence of multiple kinds of synaesthesia; and creating a more extreme phenomenology of synaesthesia (including being a projector). It may also drive the emergence of phenomena such as grapheme-personification suggesting that, whether one chooses to label it as synaesthesia or not, it is a by-product of the causal mechanisms that create synaesthesia. The notion of a synaesthetic disposition might also explain why some people acquire synaesthesia under certain conditions (e.g. after blindness) whereas others do not (i.e. these people may have had a high synaesthetic disposition prior to blindness). Thinking of synaesthesia in this way has the potential to unite the field. It suggests that the profile of what it is to be a synaesthete is coherent and not completely at the whim of the particular pattern of inducers and concurrents (also [44]).

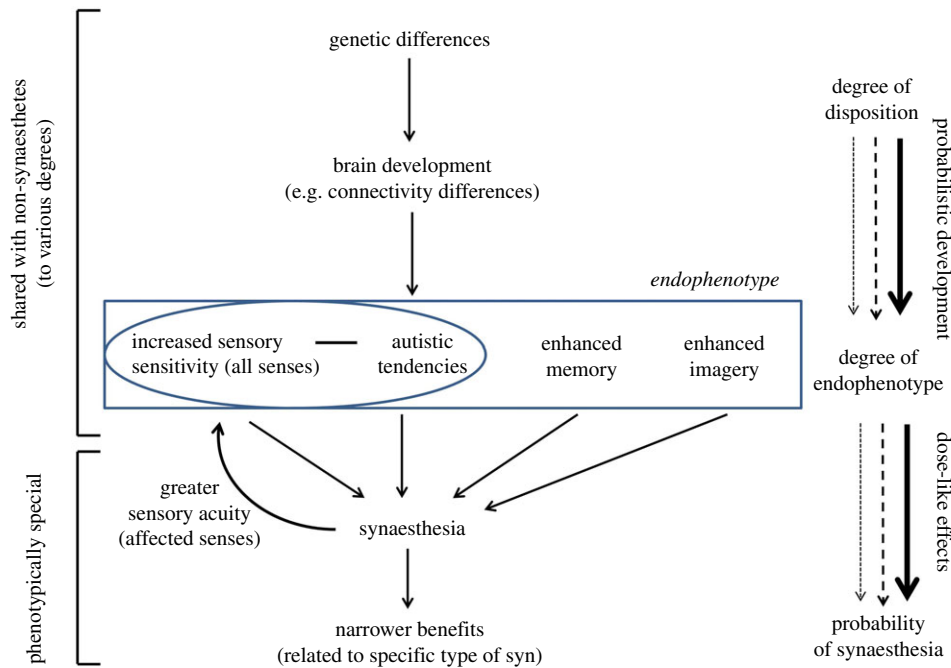


Figure 4. A simple framework for the development of synaesthesia in which the disposition towards having synaesthesia varies within the population but the presence of synaesthesia emerges in some people but not others. Note that many of the cognitive traits linked to synaesthesia are considered to be a predisposing influence to synaesthesia (rather than a consequence of developing synaesthesia), although synaesthesia may have some narrower direct benefits too (e.g. a synaesthete could use their coloured letters to remember spelling patterns). (Online version in colour.)

(c) Towards a unified model for the development of synaesthesia

This section extends the previous discussion to incorporate a (non-exhaustive) summary of evidence from genetics and neuroscience. A simple overview is given in figure 4. The top half of the model, shared with non-synaesthetes to various degrees, constitutes the synaesthetic disposition discussed previously whereas the presence of synaesthesia is regarded as binary.

Synaesthesia has been known, since its earliest study, to be heritable [52] and this view is being endorsed by modern genetic methods [53]. However, what appears to be inherited is a disposition to synaesthesia because neither the type of synaesthesia (grapheme-colour, lexical-gustatory, etc.) nor the particular associations (A = red, etc.) aggregate in families [54] and, as noted before, it is quite common for an individual to possess multiple kinds of synaesthesia. The view taken here is that the type of synaesthesia that develops is essentially a random event (see also [55]) given that we know of no biological or environmental influences on this. However, the particular associations are shaped by the environment and constrained by the architecture of the brain (e.g. the adjacency principle). This includes first-order associations (e.g. from childhood letter sets; [10]), and second-order associations (e.g. high-frequency graphemes being more saturated colours) including cross-model correspondences (for similar views and discussion see [11,56]). The disposition to develop synaesthesia may itself be graded (rather than all or nothing) and have cascading graded effects on brain development and cognition. However, the emergence of synaesthesia is assumed to be all-or-nothing, like the outcome of a flip of a coin, but playing out across multiple brain regions (flipping a handful of coins, in this analogy). Although there may be two outcomes (synaesthesia or not), the probability of those outcomes will be weighted by the person's disposition (flipping multiple

weighted coins). Someone with a small disposition to synaesthesia is unlikely to develop synaesthesia and, if they do, it is even more unlikely to be prolific (because the chances of synaesthesia emerging independently several times in their brain is low). But someone with a high disposition towards synaesthesia is likely to develop at least one kind of synaesthesia and may well develop multiple kinds. This analogy explains the dose-like effects but, importantly, in this model, it is the degree to which a person has a synaesthesia-like neurocognitive disposition that biases the number of kinds of synaesthesia that are likely to emerge (and not vice versa).

The question of how genetic differences alter brain development in synaesthetes is largely unknown (for discussion see [57]). However, the notion of hyper-connectivity, structural and/or functional, remains central to all current theories. The Neonatal Synaesthesia Hypothesis argues that synaesthesia reflects a disruption of the normal process of pruning synapses and pruning certain pathways during development (e.g. [58]): i.e. resulting in hyper-connectivity in synaesthetes. The number of synapses in the human brain peaks soon after birth and declines thereafter [59], and there is evidence of certain tracts connecting auditory-visual cortices, potentially important for synaesthesia, that diminish in early life [60]. As such, there is a biological plausibility to this account. A more controversial claim is that synaesthesia might be present from birth in everyone, but maintained by only some people [58]. An alternative viewpoint, more consistent with the model here, is that individual differences in the disposition towards synaesthesia (cognitively, neurobiologically) develop early in life but these differences may predate the onset of synaesthesia itself (during childhood) and, indeed, lead to the possibility of having a synaesthetic-brain-type without synaesthesia. From a more historical perspective, grapheme-colour synaesthesia could not have existed before the cultural invention of literacy (which has only become near-universal within

the last few hundred years), so the synaesthetic disposition must predate this phylogenetically (species development) if not ontogenetically (individual development). This idea is returned to in more detail in the next section.

Although there are a significant number of neuroimaging studies on synaesthesia, they are dominated by research on grapheme-colour synaesthesia and the bigger picture of the neural basis of synaesthesia is lacking. It has even been questioned whether a distinctive neural marker for synaesthesia exists at all [61]. Global measures of brain connectivity (e.g. derived from graph theory) would provide one way of exploring this over-and-above the idiosyncratic differences that pertain to individual variants of synaesthesia. The present framework makes testable predictions that more extreme synaesthetes (more types of synaesthesia; more extreme phenomenology; more extreme cognitive profiles) will be more extreme on these kinds of measures. There is some evidence that the effects of synaesthesia are linked to a variety of changes in the brain beyond those that might be expected from the particular inducers and concurrents [62,63]. These include increases in the organization of white matter connectivity in multiple regions, together with multiple regional differences in grey matter density. Although white matter has become synonymous with measures of connectivity, this is somewhat misleading: grey matter density is influenced by connectivity in the form of synaptic exuberance (as opposed to axonal connectivity). Along these lines, Hänggi *et al.* [64] cross-correlated local measures of cortical thickness to argue that there was 'globally altered' network topography in the brains of grapheme-colour synaesthetes showing higher clustering coefficients (a marker of local hyper-connectivity) among other network differences. These are potential markers of synaesthesia (i.e. neural predispositions) that transcend the particular kind of synaesthesia experienced (e.g. in this study, there was no special status for the fusiform region that processes graphemes and colours).

The question of how and why the particular cognitive symptoms (endophenotype) of synaesthesia are relevant remains to be fully fleshed out, although some speculations are warranted. As mentioned earlier, synaesthetic experiences are like mental images (i.e. simulations of percepts, separate from reality) so it is hardly surprising that this propensity is important for its development [65]. Synaesthetic experiences are also like memory associations. Although I have couched the memory differences in terms of ability, there may also be differences in learning style. Bankieris *et al.* [66] found that synaesthetes showed more perseveration of learned knowledge (i.e. across contexts), which may perhaps explain why synaesthesia is not unlearned in the face of considerable evidence that most letters are not coloured. Autistic traits (including sensory ones) may reflect similar principles of brain organization across these two conditions. However, some of the cognitive differences found in synaesthetes do seem to be tied to type of synaesthesia that emerges and would, therefore, appear to be best explained as a consequence of the emergence of synaesthesia (rather than a cause). One example of this is that people with visual concurrent experiences have better colour discrimination but not tactile discrimination, whereas people with mirror-touch have the reverse pattern [67].

A recent computational model of synaesthesia attempted to formally explore some of these issues. Shriki *et al.* [24] examined the properties of a neural network comprised of two sets

of neurons that can be conceptualized as representing the inducer and concurrent (e.g. sound and colour). These neurons have the potential to communicate within and across modalities, although the cross-talk (synaesthetic) connections are initially non-functional (weighted near zero). The feed-forward inputs to these neurons were statistically independent from each other so there were no regularities in the input signals (i.e. it does not learn from repeated association). The network was trained to optimally discriminate between sensory inputs (maximizing the mutual information between input to the network and neuronal output), and Shriki *et al.* [24] were interested in scenarios in which non-zero (i.e. functional) cross-talk connections stabilized between modalities. Most simulations did not result in cross-talk but some did, resulting in a simulation of synaesthesia (i.e. input to modality A resulted in output activity in modalities A + B). These included conditions of sensory deprivation (in which one modality was far less sensitive than another), which resembles acquired synaesthesia. Conditions of high plasticity (learning rate) also lead to synaesthesia emerging, which may resemble developmental synaesthesia (e.g. enhanced memory). There were also other emergent properties such as increased sensory sensitivity of the concurrent, and monotonic mappings between inducer and concurrent (e.g. akin to a pitch-lightness correspondence) even though such mapping was not present in the input. Although the specifics of this model require further formal testing, the general approach may be a useful one. The model was not trained to develop synaesthesia in some trivial way (e.g. Hebbian learning) but, instead, the model was trained to optimize sensory information processing and synaesthesia emerged under certain special starting conditions. In this model, synaesthesia is an emergent property.

4. Why does synaesthesia exist?

In this section, I consider why synaesthesia exists in terms of possible adaptive (evolutionary) advantages of having synaesthesia (see also [68]). As an extension of the arguments in the preceding sections, two broad possibilities will be considered. Firstly, that synaesthesia has some direct functional benefits. Secondly, that synaesthesia goes hand-in-hand with other adaptive benefits. This is consistent with the view that synaesthesia is an emergent property, and that a synaesthetic disposition has (to some extent) an existence of its own. Of course, there is also a third (null) possibility that, while synaesthesia may be cognitively beneficial to that individual, it is irrelevant for survival or reproduction and, hence, is neither positively nor negatively selected.

(a) Synaesthesia as information-bearing

Synaesthesia could be said to be functional if it provided the synaesthete with new information about the state of the world. If this were found, then synaesthesia could even be regarded as a true form of perception [69]. In most cases, synaesthesia cannot be said to fulfil that role. For instance, if a person is presented with a letter 'A' on a computer screen then the synaesthetic associations to that stimulus (e.g. its redness or sourness) does not provide any new information about the identity of that letter. A non-synaesthete is perfectly capable of recognizing it as an 'A' and, so it follows, the synaesthetic associations are redundant and epiphenomenal. Given an ambiguous grapheme such as MUSIC/34S67

the synaesthetic colour depends on the interpretation of the inducer, as opposed to the colour determining the interpretation [70]. Indeed, given the standard definition of synaesthesia as a concurrent triggered by an inducer, it might seem hard to imagine any scenarios in which a concurrent could be more informative than the inducer itself. However, there are some examples in the literature that appear to contradict this and are worthy of consideration.

One potential example is hearing-motion synaesthesia in which visual movement triggers auditory synaesthetic experiences. Saenz and Koch [71] carried out a rhythm discrimination task in which either two sequences of flashes or two sequences of beeps (like Morse code) are judged as same or different. It was reported that hearing-motion synaesthetes outperform non-synaesthetes on the visual version of the task. The explanation for this is that hearing is more sensitive than vision in the temporal domain (crucial for rhythm), so being able to hear the flashes in the visual version of the task gives the synaesthetes a performance advantage relative to controls. But there is a problem in this logic. It is not clear how a lower resolution inducer (flashes) gives rise to a higher resolution, more informative, concurrent (sounds). Low-resolution flashes should induce low-resolution sounds. An alternative explanation for their results is that it involves cognitive processes beyond the sensory signals themselves. This may include differences in auditory versus visual short-term memory, such that the former is more durable. In this example, the information in the concurrent is not strictly carrying novel information but may be more readily accessible to post-perceptual maintenance and inspection.

Another example along these lines, anecdotally reported by some grapheme-colour synaesthetes, is that it can help to identify and remember spelling patterns. For instance, consider the spelling of 'separate' which is notoriously difficult to learn because of the unstressed second syllable (consistent with 'seperate'). Grapheme-colour synaesthetes may be able to use the perceptual grouping of colours linked to the two 'a' letters to note and remember the pattern. In a similar vein, Ward *et al.* [72] discuss whether sound-colour synaesthetes might be able to use their visual concurrent to memorize the pitch of, say, middle C (similar to perfect pitch). In both of these instances, the concurrent can act as a tool for the synaesthete. This is a repurposing of the synaesthesia rather than a direct (information-bearing) benefit of it, although some have argued that this explains why synaesthesia exists [56].

A more extreme example of synaesthesia being information-bearing would be scenarios in which the concurrent is experienced in the absence of an inducer. This issue has been widely studied in the case of grapheme-colour synaesthesia given the ease with which one can present letters and words subliminally. However, the evidence that the concurrent can be perceived in the absence of an inducer is weak [73]. This suggests that, in these studies, the synaesthetic concurrent is no more informative than the inducer itself. A rather different approach to this issue was taken by Simner & Ward [74] who examined tip-of-the-tongue states (elicited by pictures of uncommon words, e.g. castanets) in lexical-gustatory synaesthetes. They found that the synaesthetes could experience the concurrent (e.g. a taste of tuna fish) in the absence of word retrieval (e.g. 'castanets'). This could be taken as an example of a full concurrent elicited by an inaccessible inducer. However, the claim made in that study is that the

inducer is the word-meaning (which was fully accessible) rather than the word-form (which was not).

Of course, normal perception typically precedes from low-resolution inputs to higher resolution outputs by using knowledge of the world to fill-in the gaps in the input signal. Whether synaesthesia ever operates like this is uncertain (the evidence reviewed above suggests not). One potential example is the enhanced recognition of facial expressions found in people with mirror-touch synaesthesia [75,76]. Unlike the previous examples, the relationship between inducer (observed expression) and concurrent (somatosensory activity) is non-arbitrary such that the somatosensory system contains independent knowledge about possible facial configurations that can boost performance (i.e. the concurrent is independently informative). However, this example might be stretching the argument too far because it is unlikely that mirror-touch synaesthetes actually experience synaesthesia when looking at facial expressions. It may reflect, instead, a greater propensity for visuo-tactile interactions in these people outside of their overt synaesthetic experiences themselves (certainly these mechanisms are unlikely to be unique to synaesthetes [77]).

In conclusion, synaesthesia is not typically information-bearing (over and above the information in the inducer) but, instead, it can afford different strategies for cognitive processing that can sometimes be advantageous.

(b) Synaesthesia as a spandrel

In evolutionary biology, a spandrel is a phenotypic characteristic that is a by-product of the evolution of some other characteristic, rather than a direct product of adaptive selection (e.g. [78]). It takes its name from an architectural feature (a triangular space between arches) that is itself functionless but is a consequence of other functional design choices in a building. Extending this argument to synaesthesia, we would state that synaesthesia is largely functionless but is a by-product of some other beneficial design feature(s) of the brain. There are actually two slightly different versions of the spandrel account. One is that synaesthesia is truly functionless, and the other is that while being a by-product of some primary function it may, nevertheless, be co-opted for some secondary functions (perhaps along the lines of those scenarios described in the last section).

In these spandrel views, synaesthesia is little more than a 'colourful sideshow' [79]. If so, then what is on the main stage? Three possibilities, already discussed above are: creativity, memory and perception. A fourth possibility, namely heightened mental imagery, is not considered viable because the evidence largely suggests that mental imagery itself is epiphenomenal (another possible spandrel). Of course, these possibilities are not necessarily mutually exclusive if, as suggested previously, synaesthesia emerges from some interaction between them.

According to Ramachandran & Hubbard [80, p. 58], 'Synesthesia causes excess communication among brain maps... it could lead to both synesthesia and to a propensity towards linking seemingly unrelated concepts and ideas—in short creativity. This would explain why the apparently useless synaesthesia gene has survived in the population.' This is a clear spandrel account in which synaesthesia is a by-product of some other adaptive function (creativity) linked to design changes in the brain. Similar claims can be made

regarding memory and some aspects of perception (e.g. attention-to-detail), that appear to extend across multiple forms of synaesthesia. The model of Shriki *et al.* [24], described earlier, is effectively a spandrel account. It states: 'Our model repositions synaesthesia not as some quirk of aberrant connectivity but rather as a functional brain state that emerges, under certain conditions, as a consequence of optimizing sensory information processing' (p. 12).

While the evidence that synaesthesia is linked to certain cognitive benefits is compelling, our current attempts to link it to evolution are more of a just-so story. For instance, is synaesthesia beneficial for survival/reproduction as well as cognitively beneficial? Of course, the answer to that question will have more to do with benefits in prehistoric (hunter-gatherer) lifestyles than current ones but it is easy to imagine how these abilities (memory, perception) would have been beneficial to their survival. What is harder to imagine is what kinds of synaesthesia would have existed for prehistoric people (e.g. without literacy, or calendars) given that most contemporary types of synaesthesia involve these inducers [81]. But, of course, under a spandrel account the nature of the synaesthesia (e.g. what acts as an inducer) is a complete irrelevancy: it is all about the synaesthetic brain-type (disposition) rather than the synaesthesia itself. The types of synaesthesia that emerge are likely to reflect a combination of cultural exposures (e.g. literacy) and brain organization (e.g. the adjacency of grapheme and colour regions), but there is no deep meaning (or evolutionary benefit) in, for instance, 'Monday' being green.

If we assume that synaesthesia (or, more properly, the synaesthetic brain-type) is being positively selected for, then this should mean that the prevalence of synaesthesia itself would rise over a suitably long timescale. This is possible, but it is also possible that there are other pressures that keep the prevalence balanced. For instance, there is a link between synaesthesia and autism, and people with autism (at least in the modern world) are less likely to reproduce [82]. PTSD (post-traumatic stress disorder) is more common in people with grapheme-colour synaesthesia exposed to trauma [9]. Again, this is a good example of where the synaesthetic disposition (e.g. to involuntary mental imagery, good memory) is likely to be more important than synaesthesia itself (e.g. 5 = yellow). Whether there are other costs to having synaesthesia remains to be seen (e.g. [83]).

In summary, in evolutionary terms, synaesthesia is a spandrel: a colourful sideshow. But this is not to say that research on synaesthesia is unimportant, it just forces us to think more carefully about why it is important (it is one feature of a larger pattern of neurocognitive differences). Nor does this idea deny that individual synaesthetes find beauty or personal meaning in their experiences (they do), but evolution is blind to such considerations.

5. What does synaesthesia reveal about the normal mind and brain?

If synaesthesia is not on a continuum with normality then how can one defend the claim that studying synaesthesia can inform models of normal functioning (i.e. within a standard neuropsychological framework)? The question itself is ill-posed as it assumes that there is such a thing as 'normal functioning' as opposed to, say, a multiplicity of different

kinds of normal functioning of which synaesthesia is one example. Perhaps the bell-curve notion in which normality is a continuous distribution needs to be replaced by a 'lumpy' model of what it is to be neurotypical, in which qualitatively different kinds of minds and brains coexist. This represents an interesting middle ground between the traditional monolithic notion of 'normal' on the one hand and, on the other hand, the platitude that 'everyone is unique'. Synaesthetes possess a distinctive cognitive profile that is tied together by a distinctive set of unusual experiences (i.e. synaesthetes have something in common despite being unique). Synaesthesia is special, rare and normal (i.e. not a pathology). So to directly answer the initial question: synaesthesia can inform models of normal functioning because it is one particular mode of normal functioning. The question of how science and society characterizes these differences in neurotypical function is ripe for further debate and exploration. If we rebrand synaesthesia as an alternative exemplar of normality then does it become less interesting as an object of study? I would argue not, including for biomedical science. For instance, we have shown that synaesthetes occupy an important cognitive niche in that they have many of the talents linked to autism while sitting on a knife-edge of increased vulnerability to other symptoms of autism [32]. As such, the study of synaesthesia can be important for biomedical science but without the need to pathologize synaesthesia itself. Studying synaesthesia, in parallel with other conditions, could provide a novel window into clinical vulnerabilities. Research along these lines would constitute a neuropsychology of individual differences. This resembles the traditional neuropsychological approach of using brain lesions to 'carve cognition at its seams' [84] but, instead, we are using naturally occurring variations in brain structure/function driven, at least in part, by genetic differences (and identified through differences in subjective experience). Although synaesthesia is phenotypically special, the underlying causes of synaesthesia (what I have termed the synaesthetic disposition) are made up of 'ingredients' that are, to some extent, shared by others. It is the combination of these ingredients that may be distinctive (i.e. relatively rare). However, it may nevertheless be possible for some people to have this profile and to not have synaesthesia (assuming the synaesthetic disposition is probabilistic rather than deterministic).

While the defining phenotype of synaesthesia might be distinct and special, there are many ways (probably most ways) in which synaesthetes do not differ from other people in the population. In this instance too, studies of synaesthesia can inform models of normal functioning because these aspects of cognition are typical (i.e. shared with non-synaesthetes). In this approach, synaesthesia is a tool for understanding the mind and brain rather than the object of study in its own right. To give some concrete examples of this approach, different models of language make different claims about whether compound words (e.g. 'download') consist of one or two lexical entries. One approach to answering this question is to ask whether synaesthetes are more likely to have one or two colours for these words, relative to non-compound words matched for length and other psycholinguistic properties [85]. Behind this approach is a tacit assumption that synaesthetes and non-synaesthetes represent compound words in similar ways (i.e. synaesthetes are not special in this regard), such that the study of synaesthetes

provides a legitimate source of evidence to this research question. Another example is the study of cross-modal correspondences (e.g. pointy shapes associated with sourness or high pitch). It has been argued that these are shared between both synaesthetes and non-synaesthetes (e.g. [86]). Using this approach, it could be possible, through studying synaesthesia, to uncover previously unknown cross-modal correspondences that turn out to be universal (i.e. shared with non-synaesthetes). Again, the unusual experiences of synaesthesia are being used as a research tool and, in these situations, synaesthesia provides converging evidence but not privileged evidence.

To summarize, synaesthesia can be both phenotypically special and draw upon some cognitive processes that are shared with non-synaesthetes. It is a variation of the normal mind and brain; a different exemplar of what it is to be 'normal'.

6. Summary

In this article, I argue that there is a divide between synaesthetes and non-synaesthetes (synaesthetes are phenotypically special),

but that there are important differences among synaesthetes. The amount of types of synaesthesia that a person possesses is linked to a more extreme cognitive profile and, one would predict, more extreme differences in genotype and brain organization. These cognitive differences are generally advantageous and (in the main) do not appear to be linked to the particular profile of inducers and concurrents. For this reason, and others, I suggest that synaesthesia owes its existence, in evolutionary terms, to these associated abilities. In terms of future directions, modern synaesthesia research has focused almost exclusively on grapheme-colour synaesthesia to the virtual exclusion of all others. This paper is, in part, a call to researchers to investigate synaesthesia in a more holistic way.

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