

What is the relationship between Aphantasia, Synaesthesia and Autism?

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Abstract

For people with aphantasia, visual imagery is absent or markedly impaired. Here, we investigated the relationship between aphantasia and two other neurodevelopmental conditions also linked to imagery differences: synaesthesia, and autism. In Experiment 1a and 1b, we asked whether aphantasia and synaesthesia can co-occur, an important question given that synaesthesia is linked to strong imagery. Taking grapheme-colour synaesthesia as a test case, we found that synaesthesia can be objectively diagnosed in aphantasics, suggesting visual imagery is not necessary for synaesthesia to occur. However, aphantasia influenced the type of synaesthesia experienced (favouring ‘associator’ over ‘projector’ synaesthesia - a distinction tied to the phenomenology of the synaesthetic experience). In Experiment 2, we asked whether aphantasics have traits associated with autism, an important question given that autism – like aphantasia – is linked to weak imagery. We found that aphantasics reported more autistic traits than controls, with weaknesses in imagination and social skills.

Keywords: aphantasia, synaesthesia, autism, mental imagery, Generation Scotland

1. Introduction

When asked to imagine a sunset, most people can create a picture of the scene within their mind's eye, as a visual mental image. People with *aphantasia* lack this ability (no clear 'mental picture' in the mind) even though they can describe what a sunset looks like (Zeman, Dewar, & Della Sala, 2015). Scientists have known for at least 100 years that some individuals do not experience visual imagery (Galton, 1880) but aphantasia has only recently entered mainstream research (e.g., Jacobs, Schwarzkopf, & Silvanto, 2018; Keogh & Pearson, 2018; Zeman et al., 2015, 2020). The present study adds to this body of literature by investigating the relationship between aphantasia and two other neurodevelopmental conditions, *synaesthesia* and *autism spectrum conditions* (henceforth, autism). As we shall see below, synaesthesia has been linked with higher-than-average mental imagery (e.g., Barnett & Newell, 2008; Price, 2009; Spiller, Jonas, Simner, & Jansari, 2015), raising the question of whether people with aphantasia can experience synaesthesia at all. In contrast, autism has been linked with *poor* imagery (or at the very least, with poor *imagination* - a distinction we will elaborate on below) raising the question of whether aphantasia and autism might overlap in some way. We present two studies testing these ideas, but begin with a brief overview of key concepts.

Aphantasia is most often a congenital or life-long condition, in which individuals experience an absence of visual imagery, or imagery that is only vague or dim (Zeman et al., 2015). Previous prevalence estimates for aphantasia range from 0.7% (Zeman et al., 2020) to 2.1% (Faw, 2009) and 6.7% (Betts, 1909) for individuals with no mental imagery at all, but are as high as 10-11% or 15.3% (Faw and Betts respectively) for imagery that is either absent or dim/vague. Since visual imagery has been linked to a number of aspects important in everyday life (e.g., autobiographical memory recall; short term memory recall; task-oriented motivation; Keogh & Pearson, 2014; Schacter & Addis, 2007; Vasquez & Buehler, 2007), it may seem surprising that many aphantasics live their lives without knowing they are different (Watkins,

2018; Zeman, Dewar, & Della Sala, 2016). However, some aphantasics describe problems with autobiographical memory and face recognition (Zeman et al., 2020), and the condition may have implications for visual processing strategies and even career choices (e.g., aphantasics are less likely to enter the arts, and more likely to work in science and maths; Zeman et al., 2020). In the current study we will further investigate the implications of aphantasia, asking whether it is related to two other conditions: autism and synaesthesia. We explore these conditions in particular because they too have potentially atypical imagery phenomenology, and this raises questions about how they might intersect with aphantasia.

Synaesthesia is a neurodevelopmental trait in which the senses intermingle (e.g., Simner, 2019; Simner & Hubbard, 2013). For example, listening to music can trigger the experience of colours for *sound-colour synaesthetes* (Ward, Huckstep, & Tsakanikos, 2006) or tastes in the mouth for *sound-taste synaesthetes* (Beeli, Esslen, & Jäncke, 2005), while *sequence-space synaesthetes* think about time units and other sequences (letters, numbers) in spatial patterns (e.g., the synaesthete might feel that days unfold in a zigzag line across the visual field, or that calendar months wrap around the body; e.g., Havlik, Carmichael, & Simner, 2015). Here, we focus on a variant of synaesthesia known as *grapheme-colour synaesthesia*, in which colours are triggered by numbers or letters (Meier & Rothen, 2013; Simner, Glover, & Mowat, 2006; Simner, Mulvenna, et al., 2006; Ward, Simner, & Auyeung, 2005). This variant is relatively common (affecting 1.1-1.5% of people; Carmichael, Down, Shillcock, Eagleman, & Simner, 2015; Simner & Carmichael, 2015; Simner, Mulvenna, et al., 2006), easily diagnosed (e.g., Eagleman, Kagan, Nelson, Sagaram, & Sarma, 2007), and the best understood synaesthesia to date (Simner, 2019). Important to our purposes here, researchers have suggested that heightened visual imagery may be required for experiencing synaesthesia generally (Barnett & Newell, 2008; Price, 2009). This means that aphantasics, due to their absence of visual imagery, may be less likely to experience synaesthesia – or indeed, unable to

experience it at all. In our study we therefore ask whether synaesthesia (associated with high imagery) is precluded in those with aphantasia (associated with low imagery), or if not, whether it influences the type of synaesthesia experienced (e.g., does a less vivid synaesthesia result?). We briefly review the literature linking synaesthesia with imagery below.

Links between synaesthesia and imagery have been found in multiple domains. Grapheme-colour synaesthetes, for example, score significantly higher than controls on visual imagery measures such as the *Vividness of Visual Imagery Questionnaire (VVIQ; Marks, 1973)* (Barnett & Newell, 2008; Chiou, Rich, Rogers, & Pearson, 2018), *Verbalizer-Visualizer Questionnaire (Richardson, 1978)* (Kirby, Moore, & Schofield, 1988; see Meier & Rothen, 2013), *Sussex Cognitive Styles Questionnaire (Imagery sub-scale; Meador, Simner, Rothen, Carmichael, & Ward, 2016)*, and the *French Questionnaire on Mental Imagery-51 (FQMI-51; Chun & Hupé, 2016)*. Indeed, synaesthetes report higher imagery than controls across multiple sensory modalities, and especially for modalities involved in their synaesthesia (e.g., higher taste imagery for people with synaesthetic tastes; Spiller et al., 2015). Chiou et al. (2018) have recently backed up these self-reports with a binocular rivalry test. Grapheme-colour synaesthetes showed stronger priming than controls when asked to imagine a colour that subsequently appeared in the display. The degree to which the imaged colour becomes dominant in the rivalry reflects strength of visual imagery (Pearson, 2014; Pearson, Clifford, & Tong, 2008; Pearson, Rademaker, & Tong, 2011) and indeed, aphantasic individuals do not show this priming effect (Keogh & Pearson, 2018). Grapheme-colour synaesthetes are also faster than controls when making decisions about letters held in mind as a visual image (Spiller & Jansari, 2008). Some have even gone as far as to suggest that synaesthesia may be nothing more than imagery itself: Price (2009) proposed that heightened imagery may simply allow certain individuals to become aware of naturally occurring cross-modal associations held by all people (e.g., associations between space and time, triggering ‘sequence-space synaesthesia’;

Price, 2009). Either way, this body of research claims that vivid visual imagery plays a pivotal role in the development of synaesthesia (see Ward, 2019 for a discussion). If true, this would suggest aphantasic individuals may be less likely to experience synaesthesia – or may not be able to experience synaesthesia at all.

An alternative view is that heightened imagery in synaesthesia may be simply a referral bias. Simner (2013) pointed out that most studies have tended to test self-referred synaesthetes (who have made some effort to reach out to researchers), and it may be precisely those synaesthetes with the strongest imagery (i.e., most intense synaesthesia) who self-refer. Support comes from studies that have *failed* to show superior self-reported visual imagery in some cohorts of synaesthetes (Seron, Pesenti, Noël, Deloche, & Cornet, 1992; Spiller & Jansari, 2008; Ward et al., 2018) and that heightened imagery seems to emerge only in synaesthetes who are most *aware* of their synaesthesia (Ward et al., 2018). Simner (2013) therefore suggests that enhanced visual imagery may be a characteristic of certain synaesthetes (e.g., those who self-refer) but not others. A recent study by Brang and Ahn (2019) sought to eliminate the bias of self-referred synaesthetes, by screening a general population sample for synaesthesia, and using a double-blind recruitment in which neither participant nor researcher knew who were the target group. As a consequence, their grapheme-colour synaesthetes were no different to controls in their imagery vividness (measured by the *VVIQ*) although they did still differ in how often they used imagery (measured by the *Subjective Use of Imagery Scale, SUIS*; Reisberg, Pearson, & Kosslyn, 2003). Another recent study also sought to eliminate the self-referral bias, again by screening for synaesthesia in the general population (rather than asking synaesthetes to come forward). Spiller, Harkry, McCullagh, Thoma, and Jonas (2019) found that scores in a test assumed to identify grapheme-colour synaesthetes (or synaesthesia-like behaviour) did correlate with scores in a test assumed to tap mental imagery (*Animal Tails Test*; Farah, Levine, & Calvanio, 1988). However, we suggest here that neither test may have

met its intended goal: the test for grapheme-colour synaesthesia was successfully passed by 24% of their sample (instead of the known synaesthesia prevalence of 1-2%), and their test for mental imagery – although widely accepted as such – can be performed just as well by aphantasics (who have no imagery) as by controls (Milton et al., 2020; Zeman et al., 2010)¹. From this brief literature review it seems evident that the relationship between visual imagery and synaesthesia is somewhat complex and remains unclear. We note here that if synaesthesia were *not* causally linked to heightened imagery, we would anticipate finding cases of grapheme-colour synaesthesia in aphantasic individuals (and vice versa). Alternatively, if high imagery is necessary for synaesthetes, we would expect synaesthesia in aphantasic individuals to be absent.

A related hypothesis is that aphantasia might influence the *type* of synaesthesia experienced. This hypothesis is built on the distinction between ‘projector synaesthetes’ and ‘associator synaesthetes’ (Dixon, Smilek, and Merikle, 2004). For projectors, synaesthetic colours feel like part of the outside world (e.g., projected onto the written typeface in grapheme-colour synaesthesia). For associator synaesthetes, colours are less ‘veridical’, often feeling internal to the body (e.g., appearing in the ‘mind’s eye’) or are simply “known” in some intrinsic way (Ward, Li, Salih, & Sagiv, 2006). The projector-associator distinction has been supported by neuroscientific measures (projectors have greater white matter coherence in inferior temporal cortex than associators; Rouw & Scholte, 2007), and behavioural measures (i.e., projectors are faster to name synaesthetic colours when viewing coloured graphemes, while associators are faster to name text-colour; Dixon et al., 2004; Ward, Li, et al., 2006).

¹ In this task (*Animal Tails Test*; Farah, Levine, & Calvanio, 1988), participants imagine an animal and state whether its tail is ‘short’ or ‘long’. This was devised as a test of mental imagery although we now know it is entirely possible to perform this task without any imagery at all (aphantasics perform well, and use instead their intact visual memory and visual semantic knowledge). We direct the reader to debates on how scientists use their own imagery abilities to direct their science (Reisberg et al., 2003), which may have played a role in devising this test.

Simner (2013) has suggested that this projector-associator distinction may rely on imagery differences, in that projectors may simply be synaesthetes who – aside from synaesthesia – happen to have high mental imagery. Simner (2013) suggests their high imagery may allow synaesthesia to become ‘scene-like’ to an extreme extent. Supporting this hypothesis, Amsel, Kutas and Coulson (2017) showed that self-reported imagery is higher in projectors than associators (using the *Object-Spatial Imagery and Verbal Questionnaire*; Blazhenkova & Kozhevnikov, 2009) and that projectors show neurological markers of heightened imagery (i.e., larger lateral occipital N170 responses and smaller P1 event-related potentials to visual stimuli compared to associators; see Ganis & Schendan, 2008; Hirschfeld, Feldker, & Zwitserlood, 2012). In summary, this body of literature proposes two ideas in parallel: that synaesthesia requires high mental imagery, and that projector synaesthetes have higher imagery than associators. Here we directly test these ideas using people with aphantasia. We hypothesise that having aphantasia may *preclude* having synaesthesia, or if it does not, it may make synaesthetes more likely to be associators than projectors.

Our final hypothesis relates to the relationship between aphantasia and autism, another neurodevelopmental condition with links to imagery. People with autism show a range of developmental differences, for example, in social processing, communication, sensory sensitivity, and – importantly for us – deficits in imagination (American Psychiatric Association, 2013). For example, children with autism engage less in imaginative behaviour such as pretend play (Baron-Cohen, 1987; Davis, Simon, Meins, & Robins, 2018; Jarrold, Boucher, & Smith, 1996) and have deficits in imaginative drawing (Low, Goddard, & Melsner, 2009; Scott & Baron-Cohen, 1996; Ten Eycke & Müller, 2014). Although visual imagery and imagination are in many ways distinct concepts, they are often confused or seen as interchangeable (Arcangeli, 2020; Faw, 2009; Thomas, 1999) perhaps especially by people with high visual imagery themselves. Nonetheless, people with aphantasia can use their imagination

without having any visual imagery at all², as evidenced by writers and artists with aphantasia (Zeman et al., 2019). Nonetheless, there may yet be a relationship between visual imagery and imagination more broadly. For example, González, Campos, and Pérez (1997) found a positive correlation between scores on *The Torrance Tests of Creative Thinking* (in verbal and figural creative thinking; Torrance & Ball, 1992) and conventional mental imagery tasks (*The Spatial Test of Primary Mental Abilities* and *The Gordon Test of Visual Imagery Control*; Richardson, 1969; Thurstone & Thurstone, 1989). Similarly, when participants are asked to imagine future events, their visual imagery (in the *VVIQ*; Marks, 1973) predicts the amount of sensory, spatial, and emotional information described, as well as the personal importance and significance of the event (D'Argembeau & Van der Linden, 2006). Important for our purposes, the highly influential questionnaire for traits associated with autism (the *Autism Spectrum Quotient*; *AQ*; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001), contains a question asking about the strength of visual imagery within its 'imagination' subscale. This shows that deficits in visual imagery are directly captured by assessments of autism (albeit under the ambiguous heading of 'imagination'). Here, we therefore investigate whether aphantasia and autism may be linked, through some weak visual imagery ability.

In contrast to the above research, other studies have questioned whether there is any link between autism and low visual imagery at all. Hughes et al. (2018) found no difference between autistic participants and controls in the '*Imagery Ability*' subscale of the *Sussex Cognitive Styles Questionnaire* (*SCSQ*; Mealor, Simner, Rothen, Carmichael, & Ward, 2016), and indeed other research shows that people with higher AQ scores find it easier (not harder)

² The final author of this paper has aphantasia, but reports no trouble whatsoever imagining any event, hypothetical or real. (For her, imagination is a sense of 'knowing' rather than 'seeing'). She scores highly on traits related to imagination (e.g., she is high on Openness to Experience; McCrae & Costa, 1987) and scored highly throughout schooling on imaginative activities such as creative writing. However, when imagination is conflated with visual imagery (e.g., in questionnaires asking about the vividness of mental images) she scores poorly. In this sense, imagery and imagination are unrelated. However, there may yet be 'hidden' links between the two (e.g., it is theoretically possible that some elements of a scene might be more difficult to imagine in the absence of mental imagery). We explore these hidden links here.

to judge the ‘imageability’ of words (e.g., does ‘blush’ lend itself to a visual picture more easily than ‘hypothesis’?; Esposito, Dellantonio, Mulatti, & Job, 2016). Other research too shows the cognitive style in autism of ‘thinking in pictures’, which may point to *elevated* imagery in autistic individuals (e.g., Kana, Keller, Cherkassky, Minshew, & Just, 2006; Kunda & Goel, 2008, 2011; Soulières, Zeffiro, Girard, & Mottron, 2011). Overall, the relationship between visual imagery and autism therefore remains unclear. But if autism is driven, even in part, by a deficit in visual imagery – or if visual imagery deficits are a consequence of autism – we might predict higher levels of autistic traits in people with aphantasia. This might be especially evident in the imagination subscale of the AQ (even when its question about visual imagery is removed).

We therefore present two studies investigating the relationship between aphantasia, synaesthesia and autism. Experiments 1a and 1b explore the interplay between aphantasia and synaesthesia within two separate large-scale samples: (a) members of the general population recruited from the testing cohort *Generation Scotland* (www.ed.ac.uk/generation-scotland) (Smith et al., 2013), and (b) a group of synaesthetes tested via the online platform known as the *Synaesthesia Battery* (www.synesthete.org; Eagleman et al., 2007). These cohorts are described further below, but we hypothesise that if high visual imagery is related to (or necessary for) synaesthesia, aphantasia may not co-occur with synaesthesia at all. However, if synaesthesia can exist in the absence of imagery, we may find people with synaesthesia among those with aphantasia (and vice versa), although synaesthesia might manifest differently (more associator-traits than projector-traits within aphantasia). In a final Experiment, we investigate the relationship between aphantasia and traits associated with autism given the possibility of low visual imagery across both aphantasia and autism. We hypothesise that autism traits may be higher in aphantasics than controls from the general population, and perhaps particularly in the AQ subscale of imagination.

2. Experiment 1a: Can synaesthesia and aphantasia co-occur?

In this study we screened over a thousand members of the general population for both synaesthesia and aphantasia. Both conditions are relatively rare, so we designed our recruitment materials to attract as many cases as possible; i.e., we explicitly described both conditions within our invitation to participate. This was designed to maximise the number of cases for us to examine, but also means that our prevalences of synaesthesia or aphantasia cannot be taken as population-wide estimates (and indeed our study was not designed to be a prevalence count). Importantly, however, our samples can be compared to each other, to ask whether we can find cases of synaesthesia in a cohort of aphantasics (and vice versa).

2.1. Methods

2.1.1. Participants

Our participants were 1285 people from the *Generation Scotland Scottish Family Health Study cohort* (770 female, 515 male) (Smith et al., 2013). Generation Scotland is a large-scale resource of data available for research purposes (www.ed.ac.uk/generation-scotland), which includes adult volunteers recruited through primary care services across Scotland (e.g., via General Practices, for more information on the recruitment process see Smith et al., 2006). As part of the Generation Scotland project, the *Scottish Family Health Study* (GS:SFHS; Smith et al., 2013) collected genetic and health data for a cohort of over 20,000 of these volunteers. Some of the participants consented to re-contact in the event of further studies, and approximately 6,000 of these also provided email addresses. This allowed us to recruit a sample of participants from these volunteers via email to take part in the present study. Participants were asked to complete a series of questions and tasks online to investigate ‘health, minds and bodies of the people of Scotland’. Both imagery and synaesthesia were mentioned explicitly in recruitment.

Four additional participants (2 female, 1 male, 1 other) were excluded because they did not complete the imagery component of our test. In our final sample of 1285 participants, age was not specified but all were 18 years or older. As compensation for taking part, participants were entered into a prize draw for £100.

2.1.2. Materials and procedure.

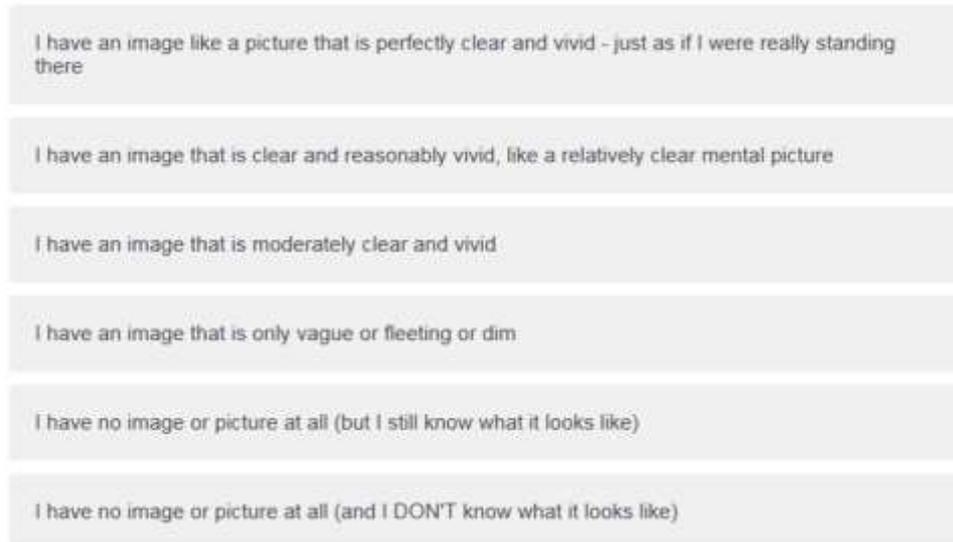
Participants were contacted via email and provided with a URL link to our online study. The study contained the following two measures in the order shown below. These were embedded among other measures to be reported elsewhere. Individuals provided informed consent prior to testing, and ethical approval was provided by the *University of Sussex Cross-Schools Science and Technology Ethics Board*, and ethical approval for the GS:SFHS study was obtained from the *Tayside Committee on Medical Research Ethics (on behalf of the National Health Service)*.

Visual Imagery Questionnaire. We measured visual imagery using an in-house task (see Figure 1), in which we asked participants to think about the building where they lived. Participants were asked to rate ‘how much your memory of it is like a picture’. Participants responded with one of five responses (ranging from 0 – ‘I have no image or picture at all and I don’t know what it looks like’, to 5 – ‘I have an image like a picture that is perfectly clear and vivid – just as if I were really standing there’; see Figure 1 for all response options). In line with previous research showing that aphantasic individuals rate their visual imagery as absent or vague/dim (Keogh & Pearson, 2018; Zeman et al., 2015), participants were classified as aphantasic if they responded in the range of 0-2 (i.e., rating their imagery as ‘no image at all’, or ‘vague or fleeting or dim’)³.

³ We did not use longer imagery questionnaires (e.g., VVIQ) because our participants were time-restricted, given other measures in the same battery. We return to this issue in Experiment 1b.

Some people can form clear pictures in their mind. Others can't form any pictures in their mind at all (even though they can still remember what things looked like). And some people are in between. Let's find out where you are on this scale.

Close your eyes and imagine the building where you live. Try to form a picture of it in your mind, as if you were standing in front of it. Click one option below to describe how much your memory of it is like a picture.



I have an image like a picture that is perfectly clear and vivid - just as if I were really standing there

I have an image that is clear and reasonably vivid, like a relatively clear mental picture

I have an image that is moderately clear and vivid

I have an image that is only vague or fleeting or dim

I have no image or picture at all (but I still know what it looks like)

I have no image or picture at all (and I DON'T know what it looks like)

Figure 1. The visual imagery question used to classify aphantasia within the *Generation Scotland* sample.

Grapheme-colour synaesthesia diagnostic. We tested only one variant of synaesthesia to limit the burden on our participants (and we selected grapheme-colour synaesthesia in particular for the reasons given in the Introduction). We diagnosed grapheme-colour synaesthesia by using the ‘gold standard’ diagnostic (Eagleman et al., 2007), which we replicated to host on our local server. This diagnostic is the most widely used scientific assessment for grapheme-colour synaesthesia worldwide. In the first part of the test, participants are asked to indicate whether they believe they experience grapheme-colour synaesthesia (“Do numbers or letters cause you to have a colour experience?”). Participants *not* reporting grapheme-colour synaesthesia are shown to an exit page (and can then complete the other parts of the test). Participants reporting grapheme-colour synaesthesia are subsequently given an objective diagnostic test to verify their self-report. In this test,

participants are identified as synaesthetes if they demonstrate the known characteristic of *consistency-over-time* (i.e., for genuine synaesthetes, associations tend to stay the same over time; e.g., if A is red for any given synaesthete, it is always red when the synaesthete is repeatedly asked). Hence, in the objective test, participants were presented with the letters A-Z and the numbers 0-9, each displayed three times in a randomised order. Participants selected their associated colour for each grapheme using an on-screen colour palette. The test computes the variation in the colours provided for the same grapheme (e.g., summing the distances between the colours chosen for the three 'A' trials). The mean colour variation is then calculated across all graphemes (for information about the testing interface and other measurement details, see Eagleman et al., 2007). If this mean colour distance is small, the individual's synaesthetic colours are consistent (i.e., close in RGB colour space; Eagleman et al., 2007), with scores lower than 1.43 indicating a diagnosis of synaesthesia (Rothen, Seth, Witzel, & Ward, 2013).

3. Results

3.1. *Can aphantasia and synaesthesia co-occur?*

We first divided our sample into those with and without aphantasia. Within our total cohort ($N = 1285$), there were 212 participants (115 female, 97 male) on the visual imagery question who scored within the aphantasic range (i.e., they scored within the range of 0-2; M score = 1.33, $SD = .48$). This gives an overall aphantasia prevalence of 16.5%. Here our classification follows prior literature in defining aphantasics with imagery either vague/dim/fleeting (n71), or completely absent (n141).

We then classified all our participants according to the synaesthesia diagnostic, and found 14 people with grapheme-colour synaesthesia (6 female, 8 male), giving an overall synaesthesia prevalence of 1.1%. There were 2 people (both male) with grapheme-colour synaesthesia within our 212 aphantasic individuals, and 12 people (6 female, 6 male) with

grapheme-colour synaesthesia within our 1073 non-aphantasics. This provides a prevalence of grapheme-colour synaesthesia of 0.9% in aphantasics, and 1.1% in non-aphantasics (see table 1), which was a non-significant difference ($p = 1.00$, Fishers Exact Test). Bayes factors (BF) were calculated (using R version 3.5.1, R Core Team, 2018; BayesFactor version 4.2, Morey & Rouder, 2018) to better understand this null effect. A BF of $<.33$ is taken as evidence for the null hypothesis (Dienes, 2014), while >3 is taken as evidence for the alternative hypothesis. We calculated a BF of .022 which provides good evidence in favour of the null hypothesis, suggesting no difference in rates of synaesthesia between aphantasics and non-aphantasics.

We next looked at the prevalence of aphantasia in our synaesthete and non-synaesthete groups. There were 2 aphantasics among our 14 synaesthetes (2 male), and 210 aphantasics among our 1271 non-synaesthetes (115 female, 95 male). This gives an aphantasia prevalence of 14.3% in the synaesthete group, and 16.5% in the non-synaesthete group – again a non-significant difference ($p = 1.00$, Fishers Exact Test) with a BF of .022, again supporting the null hypothesis. Overall, our results suggest that high imagery is therefore not a pre-requisite of synaesthesia, and that is possible to have synaesthesia with little or no imagery at all. Finally we found that mean imagery scores for synaesthetes ($M = 3.86$; $SD = 1.46$) and non-synaesthetes ($M = 3.84$; $SD = 1.30$) were statistically equivalent, ($t(13.23) = -.047$, $p = .963$, $d = .01$, 95% CI [-.86, .83], BF = .27), following others (e.g., Seron et al., 1992; Spiller & Jansari, 2008; but see also Barnett & Newell, 2008; Chiou et al., 2018; Ward et al., 2018).

Table 1

Frequency of grapheme-colour synaesthetes in aphantasic and non-aphantasic groups (displayed as a percentage of each group in brackets) within the Generation Scotland sample.

	Aphantasic (n=212)	Non-aphantasic (n=1073)	Total (n=1285)
Synaesthete	2 (.9%)	12 (1.1%)	14 (1.1%)
Non-synaesthete	210 (99.1%)	1061 (98.9%)	1271 (98.9%)

4. Discussion

Our two key results were that grapheme-colour synaesthesia can indeed exist within people with aphantasia, and it is no less prevalent in aphantasics versus non-aphantasics. Rates of grapheme-colour synaesthesia in aphantasics (0.9%) and non-aphantasics (1.1%) were statistically equivalent, and reflective of the prevalence of grapheme-colour synaesthesia in the general population (1.1-1.5%; Carmichael, Down, Shillcock, Eagleman, & Simner, 2015; Simner & Carmichael, 2015; Simner, Mulvenna, et al., 2006). Rates of aphantasia were also statistically equivalent across synaesthetes (14.3%) and non-synaesthetes (16.5%), and again largely reflective of other estimates (e.g., Betts, 1909). Importantly, our overall group-sizes were small. In other words, despite screening over a thousand participants, our groups of synaesthetes with and without aphantasia were 2 and 12 respectively. We therefore sought to replicate our finding in Experiment 1b, with a larger sample of synaesthetes. We also sought to improve on our methodology: in Experiment 1a we evaluated aphantasia using a single question, while in Experiment 1b we will use the full *Vividness of Visual Imagery Questionnaire 2* (VVIQ-2; Marks, 1995). This change can allow us to have more confidence in our assessments of aphantasia. In testing larger samples in Experiment 1b, we also asked an additional question: does aphantasia influence the *type* of synaesthesia experienced (projector or associator synaesthesia; see below)?

5. Experiment 1b: Can synaesthesia and aphantasia co-occur (a replication)? Does aphantasia influence the type of synaesthesia experienced?

In this study we recruited far larger numbers of synaesthetes by examining data from the largest international online destination for synaesthetes: *The Synesthesia Battery* is a standardised collection of tests and questionnaires for assessing synaesthesia and related phenomenology (Eagleman et al., 2007). The battery includes the gold-standard consistency

test for objectively diagnosing grapheme-colour synaesthesia cloned in Experiment 1a, as well as questionnaires to assess the type of synaesthesia experienced (e.g., the *Projector-Associator (PA) Questionnaire*; Rouw & Scholte, 2007). The battery also includes a questionnaire to measure visual imagery (*VVIQ-2*; Marks, 1995). Using these three elements of the *Synaesthesia Battery*, we aim to replicate our results from Experiment 1a (synaesthesia occurring equally in aphantasics, and non-aphantasics, and vice versa), but also predict that people with aphantasia may tend to have a different variant of synaesthesia (i.e., more associator-like than people without aphantasia).

5.1. Methods

5.1.1. Participants

Our participants were 16,246 individuals who had taken the Synaesthesia Battery between 2007 to 2018. Participants had a mean age of 29.11 years ($SD = 11.76$), and were predominantly female (77.2%; i.e., 12,539 female; 3482 male; 225 ‘other’). This female bias does not reflect an underlying epidemiological fact (see Simner & Carmichael, 2015; Simner, Mulvenna, et al., 2006) but likely the well-known finding that women participate in online surveys more often than men (Smith, 2008). Participants navigated to the website (www.synesthete.org) via various means: some located it by internet searches about synaesthesia, and others were directed by a range of synaesthesia researchers, since this testing portal is the most widely used diagnostic tool in the field of synaesthesia.

5.1.2. Materials and procedure

Participants completed three measures: (a) the objective assessment for grapheme-colour synaesthesia, (b) the *Projector-Associator (PA) Questionnaire* (Rouw & Scholte, 2007), designating any synaesthete as either an associator or projector, and (c) a visual imagery questionnaire (*VVIQ-2*; Marks, 1995), which allows us to assess participants for aphantasia. Details of these tests are given below. All participants provided informed consent prior to

taking part, and ethical approval for this study came from the *University of Sussex Cross-Schools Science and Technology Ethics Committee*, and the ethics board at *Baylor College of Medicine*.

Grapheme-colour synaesthesia diagnostic. Details of the diagnostic for grapheme-colour synaesthesia are given in Experiment 1a. Unlike Experiment 1a however, which replicated the diagnostic tool and hosted it on a server at the University of Sussex, in the current study the site was hosted at the online international meeting place for synaesthetes: www.synesthete.org.

PA questionnaire (Rouw & Scholte, 2007). This questionnaire distinguishes projector synaesthetes from associator synaesthetes. All participants were given this test, irrespective of how they performed in the diagnostic above (but our results will focus on scores from those ultimately diagnosed as synaesthetic). Participants indicated the degree to which they agreed with 10 statements about their synesthetic experiences (on a scale of 1 – ‘Strongly disagree’, to 5 – ‘Strongly agree’). Half the statements captured the experiences of projectors (e.g., “When I look at a certain letter/number, the synesthetic color appears somewhere outside my head (such as on the paper)”), and half captured the experiences of associators (e.g., “When I look at a certain letter/number, the accompanying color appears only in my thoughts and not somewhere outside my head (such as on the paper)”). In line with standard procedure (Eagleman et al., 2007; Rouw & Scholte, 2007), an overall PA score was generated by subtracting the mean score for the associator questions from the mean score for the projector questions. Associators were classified by scoring below 0, and projectors by scoring above 0. The original questionnaire also contained two additional items which we removed because they did not appear to adequately distinguish projectors from associators (“When I look at a certain letter or number, I see a particular color”, and “The color has the same shape as the letter/number”).

VVIQ-2 (Marks, 1995). Within this questionnaire, participants were asked to think of a series of eight scenarios (e.g., “A country scene ...”). Participants had to rate “the picture that comes before your mind’s eye” for four aspects per scenario (e.g., “The contours of the landscape”). Ratings were given on a scale of 1-5 as follows: 1 – ‘No image at all, you only “know” that you are thinking of the object’; 2 – ‘Vague and dim’; 3 – ‘Moderately clear and vivid’; 4 – ‘Clear and reasonably vivid’; and 5 – ‘Perfectly clear and as vivid as normal vision’. The questionnaire was scored by summing responses to all 32 questions, giving possible scores in the range of 32-160. In line with previous research showing that aphantasic individuals rate their visual imagery as absent or vague/dim (Keogh & Pearson, 2018; Zeman et al., 2015), aphantasics were classified by scoring between 32-64 on the VVIQ-2 (where a score of 32 indicates no imagery at all, and a score of 64 represents rating all items as ‘vague and dim’).

6. Results

6.1. *Can aphantasia and synaesthesia co-occur?*

We first divided our sample into those with and without aphantasia so we could compare rates of synaesthesia across each group. Here we classified aphantasics as those scoring ≤ 64 on the VVIQ-2, again in line with aphantasics rating their imagery as absent, vague or dim within prior literature. Within the total sample (16,246), there were 196 people (M age = 28.03, SD = 11.49; 145 female, 49 male, 2 other) with a VVIQ-2 ≤ 64 (M score = 52.76, SD = 10.36), giving an overall aphantasia prevalence of 1.2%. We next considered the synaesthesia diagnostic, and found 12,589 people with grapheme-colour synaesthesia overall (M age = 28.62, SD = 11.36; 9844 female, 2572 male, 173 other). There were 144 synaesthetes among our 196 aphantasics (M age = 27.24, SD = 11.42; 104 females, 38 males, 2 other), and there were 12,445 synaesthetes among our 16,050 non-aphantasics (M age = 28.63, SD = 11.36; 9740 females, 2534 males, 171 other). This provides a prevalence for grapheme-colour synaesthesia of 73.5% in aphantasics, and 77.5% in non-aphantasics (see table 2), a difference that was non-

significant [$\chi^2(1, N = 16246) = 1.61, p = .204$; chi-square test with Yates continuity correction] (NB. the percentage of synaesthetes is high because synaesthetes are a priori attracted to this website, but our values can be meaningfully compared to each other⁴). We calculated a BF of .018 which provides good support for the null hypothesis, suggesting there is no difference in rates of synaesthesia in aphantasics and non-aphantasics.

We next looked at the reverse case: i.e., the prevalence of aphantasia in our synaesthete and non-synaesthete groups. There were 144 aphantasic individuals among our 12,589 synaesthetes (M age = 27.24, $SD = 11.42$; 104 females, 38 males, 2 other), and 52 aphantasics among our 3657 non-synaesthetes (M age = 30.21, $SD = 11.52$; 41 females, 11 males). This gives an aphantasia prevalence of 1.1% in the synaesthete group, and 1.4% in the non-synaesthete group, with no significant difference [$\chi^2(1, N = 16246) = 1.61, p = .204$; chi-square test with Yates continuity correction]. Again, a BF of .018 provides good support for the null hypotheses, providing evidence for there being no difference in rates of aphantasia in synaesthetes and non-synaesthetes.

Our results replicate our finding from Experiment 1a, showing that high imagery is not a pre-requisite of synaesthesia, and that it is perfectly possible to have synaesthesia with little or no imagery at all. As an added measure of conservativeness, we can also look for synaesthesia by splitting aphantasics into those with a small amount of imagery (dim and

⁴ Both groups also had high imagery, which may be unsurprising since both were self-motivated to navigate to a website to explore the possibility they had synaesthesia (and awareness of synaesthesia grows with imagery; Ward et al., 2018). Control means were yet higher ($M = 124.44$; $SD = 23.19$) than synaesthetes ($M = 121.83$; $SD = 22.49$; $t(5799.91) = 6.03, p < .001$), which may be the reason they (falsely) believed they were synaesthetic. We can confirm both are high against a more typical control group, who were given the VVIQ-2 at the online workplace Amazon Turk ($n=502$; M age = 36.55, $SD = 11.53$; $n=208$ female, $n=292$ male, $n=2$ other). A one-way ANOVA comparing the three groups revealed a significant effect of group ($F(2,16745) = 38.98, p < .001$), with Amazon Turk participants ($M = 115.84$; $SD = 25.80$) reporting significantly lower imagery than both synaesthetes ($t(531.78) = -5.12, p < .001$) and our original controls ($t(617.21) = -7.08, p < .001$). Hence, while the recruitment methods in our study are appropriate for our own aims (i.e., finding synaesthetes with aphantasia), they are likely unsuitable for the type of groupwise comparisons which have been the focus of other papers (e.g., Barnett & Newell, 2008; Brang & Ahn, 2019; Chiou et al., 2018; Seron et al., 1992; Spiller & Jansari, 2008; Spiller et al., 2015).

vague; scoring 64 on the VVIQ-2) versus those with no imagery whatsoever (scoring 32 on the VVIQ-2). Out of the 21 individuals with ‘dim and vague’ imagery (scoring 64 on the VVIQ-2), 15 were classified as synaesthetes, and out of the 20 individuals with no imagery at all (scoring 32 on the VVIQ-2), 12 were classified as synaesthetes. There was no difference between these proportions [$\chi^2(1, N = 41) = .195, p = .659$; chi-square test with Yates continuity correction] showing that synaesthesia occurs to the same degree whether aphantasics have little imagery or none at all. We calculated a BF of .90 which provides anecdotal support for there being no difference in the rates of synaesthesia in aphantasics with weak imagery, compared to absent visual imagery. Importantly, our results show that it is indeed possible to have synaesthesia with no visual imagery whatsoever.

Table 2

Frequency of grapheme-colour synaesthetes in aphantasic and non-aphantasic groups (displayed as a percentage of each group in brackets) within the Synesthesia Battery sample.

	Aphantasic (n=196)	Non-aphantasic (n=16050)	Total (n=16246)
Synaesthete	144 (73.5%)	12445 (77.5%)	12589 (77.5%)
Non-synaesthete	52 (26.5%)	3,605 (22.5%)	3,657 (22.5%)

6.2. Does aphantasia influence the type of synaesthesia experienced? (i.e., are aphantasics more likely to be associators than projectors)?

Next, we examined the PA questionnaire data to categorise synaesthetes as associators or projectors. From the original dataset of 12,589 confirmed synaesthetes, we excluded 476 individuals with incomplete PA data (i.e., 346 individuals who had not attempted the questionnaire, and 130 who had not finished it), and a further 244 individuals with an overall PA score of 0 (i.e., whose status as projector vs. associator was unclear). Out of the remaining 11,869 ($n = 130$ aphantasics, $n = 11,739$ non-aphantasics), 1073 were projectors ($n = 14$ aphantasics, $n = 1059$ non-aphantasics) and 10,796 were associators ($n = 116$ aphantasics, $n =$

10,680 non-aphantasics). As such, 89.2% of aphantasic synaesthetes were associators, and 10.8% were projectors. A similar result was found in non-aphantasics: 91.0% of synaesthetes were associators, and 9.0% were projectors. Indeed, there was no significant difference in the occurrence of associator or projector synaesthesia across groups [$\chi^2(1, N = 11869) = .289, p = .591$; chi-square test with Yates continuity correction]. We then calculated a BF to quantify evidence for this null effect. Our calculated BF of .007 provides strong evidence for there being no difference in the occurrence of projector or associator synaesthesia across aphantasic and non-aphantasic synaesthetes.

This analysis initially suggests that people with aphantasia have the same proportions of associator/projector synaesthesia as non-aphantasics. However, this finding required us to make categorical divisions of the PA data to form two groups (associators, projectors). We therefore ask whether a link between aphantasia and associator synaesthesia might emerge by treating scores continuously. This is likely to be a more appropriate treatment of the data since Skelton, Ludwig, and Mohr (2009) have suggested that projector/associator status resides on a continuum, rather than as discrete categorisations. Hence, we investigated whether aphantasic synaesthetes have lower/more negative PA scores (indicating ‘stronger’ associator traits) than non-aphantasics. As hypothesised, we found that overall PA scores were indeed significantly lower in aphantasic synaesthetes (i.e., more associator-like; $M = -2.58, SD = 1.57$) than in non-aphantasic synaesthetes ($M = -2.20, SD = 1.54$; see figure 2). This difference was significant in an independent samples t-test with Welch correction ($t(131.79) = 2.78, p = .006, d = 0.25, 95\% CI [.11, .66]$).

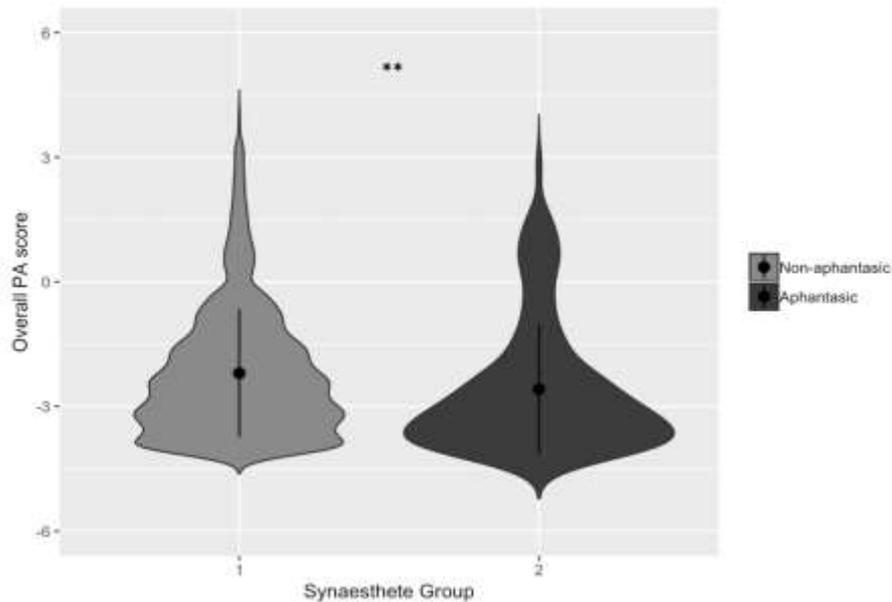


Figure 2. Overall PA questionnaire score distribution and means (with standard deviations) as a function of group (aphantasic synaesthetes vs non-aphantasic synaesthetes) within the *Synesthesia Battery* sample. Lower scores represent more associator-like traits. *Note.* * $p < .05$, ** $p < .01$, *** $p < .001$.

7. Discussion

In Experiment 1b we have investigated visual imagery using the largest sample of synaesthetes to date. We have replicated our finding from Experiment 1a that synaesthesia can co-occur with aphantasia, thereby showing that visual imagery is not a pre-requisite for synaesthesia to arise. The prevalence of synaesthesia was again equivalent for both aphantasics and non-aphantasics (and equally, rates of aphantasia were equivalent in synaesthetes and non-synaesthetes). As expected, rates of synaesthesia were very high in this sample (73.5% in aphantasics, and 77.5% in non-aphantasics) and far higher than in the general population (i.e., around 1.1-1.5%; Carmichael et al., 2015; Simner & Carmichael, 2015; Simner, Mulvenna, et al., 2006). This is simply because our testing took place at an online destination for synaesthetes (i.e., people who visit the Synesthete Battery are already likely to be synaesthetes, by virtue of their visit). Importantly, however, we can compare the prevalences within our aphantasic and non-aphantasic groups, and in doing so, we replicate our finding from Experiment 1a. Our

results also show that even individuals who report the most extreme experience of aphantasia (i.e., no visual imagery whatsoever) can still have synaesthesia, indicating that high imagery (or indeed any imagery) is not a pre-requisite. Nonetheless, we found that low imagery influences the *type* of synaesthesia experienced: aphantasic individuals reported a synaesthesia that was more associator-like (i.e., more negative scores on the PA questionnaire) compared to non-aphantasics. We return to these issues in our General Discussion.

8. Experiment 2: What is the relationship between aphantasia and autism?

In Experiment 1 a and b, we looked at two conditions traditionally thought to differ in imagery (synaesthesia as high imagery; aphantasia as low/absent imagery). We now turn our attention to a third neurodevelopmental condition – autism – which has been linked to low visual imagery (related to weak imagination symptomatology). We investigate here whether aphantasia and autism may be linked in some way, given their possible shared deficits in visual imagery.

8.1. Method

8.1.1. Participants

We recruited 118 aphantasics (M age = 38.47, SD = 14.14) and 118 matched controls (M = 37.87, SD = 15.22). Groups were matched for age ($t(234) = -.310, p = .757$) and gender (aphantasic group: 69 females, 49 males; controls: 66 females, 51 males, 1 other). Aphantasic participants were recruited from two sources: 102 were recruited from the University of Sussex's *Imagery Lab - Aphantasia Cohort* (M age = 39.94, SD = 14.27; 57 females, 45 males), and an additional 16 aphantasics were recruited from the University of Exeter *Eye's mind* database (M age = 29.06, SD = 8.88; 12 females, 4 males). Aphantasics were classified by their scores on the 16-item *VVIQ* (Marks, 1973), a shorter version of the *VVIQ-2* (Marks, 1995). In line with previous research showing that aphantasic individuals rate their visual imagery as absent or vague/dim (Keogh & Pearson, 2018; Zeman et al., 2015), aphantasics (M = 18.50, SD

= 3.65) were classified by scoring between 16-32 on the VVIQ. The score range for classifying aphantasia is lower in the VVIQ (16-32) compared to the VVIQ-2 (32-64) simply because the former contains fewer items.

Control participants were recruited from social media, word-of-mouth, Amazon's MTurk, and the participant recruitment system at the University of Sussex. Controls were confirmed as non-aphantastic again using the VVIQ (scores >32, $M = 58.49$; $SD = 13.07$). As a compensation for taking part, MTurk participants were compensated \$2 for our 15 minute test, undergraduate students received course credits and non-students were entered for a prize draw of £25.

8.1.2. Materials and procedure

Our aphantastic participants had already completed the VVIQ prior to participating in the present study, as part of their entering our participant databases. We presented the AQ (and VVIQ for controls) using our online in-house testing platform (www.syntoolkit.org), and participants completed the study from their own homes. All participants provided informed consent prior to taking part, and ethical approval was provided by the University of Sussex *Cross-Schools Science and Technology Ethics Board*.

Autism Quotient (AQ; Baron-Cohen et al., 2001). The AQ measures traits associated with autism. Participants rate how much they agree with a series of 50 statements on a scale of 1 ('Definitely agree') to 4 ('Definitely disagree'). The questions are divided equally into five subscales measuring different aspects of autism symptomology: communication (e.g. "I frequently find that I don't know how to keep a conversation going"), imagination (e.g. "When I'm reading a story, I can easily imagine what the characters might look like"; reversed scored), social skills (e.g. "I find it hard to make new friends"), attention switching (e.g. "I prefer to do things the same way over and over again") and attention-to-detail (e.g. "I tend to notice details that others do not"). Responses are scored as 0 or 1, where 1 is allocated to responses of

“definitely” or “slightly” for behaviours associated with autism (good attention-to-detail, but poor communication, imagination, social skills, and attention switching). Approximately half of the items are reversed scored. In line with standardised scoring (Baron-Cohen et al., 2001), scores equal or greater than 32 indicate the possible presence of autism.

9. Results

Firstly, we removed an item from the imagination subscale of the AQ which asks directly about visual imagery ability (question 3: ‘If I try to imagine something, I find it very easy to create a picture in my mind’). Since aphantasics would, by definition, score low on this question due to their lack of visual imagery, it was necessary to remove this question to avoid circularity⁵. We analysed overall AQ scores for aphantasics and controls using an independent samples t-test with Welch correction. Aphantasics had higher overall AQ scores ($M = 23.84$; $SD = 8.57$) compared to controls ($M = 20.51$, $SD = 5.98$), and this difference was significant ($t(209.07) = -3.46$, $p = .001$, $d = .45$, 95% CI [-5.23, -1.44]). Hence, aphantasic individuals reported significantly more traits associated with autism than controls (see Figure 3).

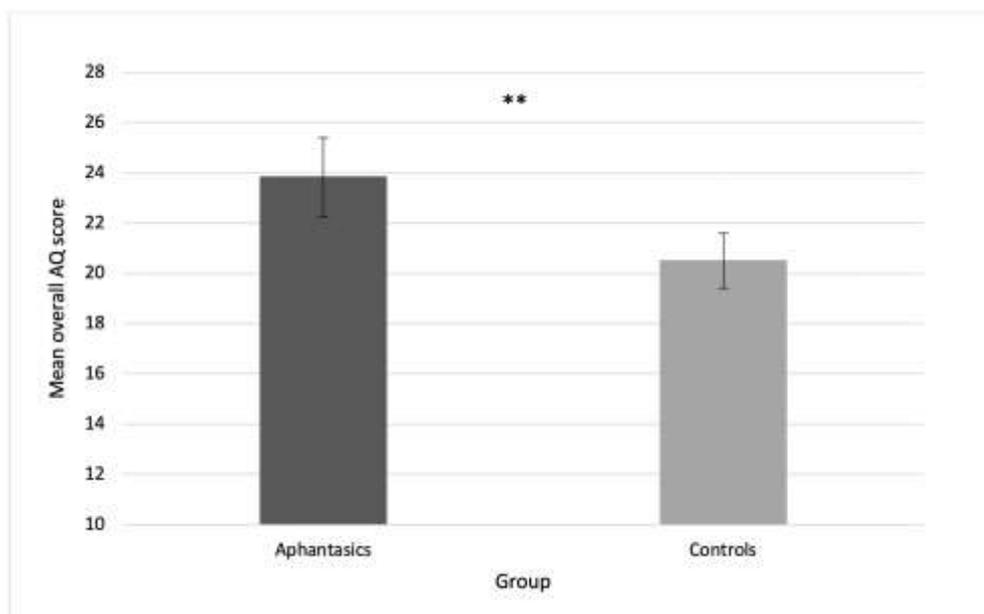


Figure 3. Mean overall AQ scores (with 95% confidence intervals) as a function of group (aphantasics, controls). *Note.* * $p < .05$, ** $p < .01$, *** $p < .001$.

⁵ We note that the overall pattern of results remained the same overall when including question 3 of the AQ.

We then conducted a 5 x 2 ANOVA broken down by subscale (imagination, social skills, communication, attention-to-detail, attention switching) and group (aphantasic, control). There was a main effect of group ($F(1, 234) = 11.95, p = .001, \eta_p^2 = .049$), and subscale ($F(3.34, 782.54) = 37.15, p < .001, \eta_p^2 = .137$; with Greenhouse-Geisser correction), and an interaction between them ($F(3.34, 782.54) = 8.56, p < .001, \eta_p^2 = .035$; with Greenhouse-Geisser correction). To investigate our results further, we conducted a series of planned independent samples t-tests with Welch correction for the AQ sub-scales, and adjusted our p values for (n5) multiple comparisons using the Bonferroni method. After correction, aphantasics scored significantly higher on the imagination subscale (indicating ‘less imaginative’; $M = 4.55, SD = 2.05$) compared to controls ($M = 2.87, SD = 1.71; t(226.63) = -6.83, p_{corrected} < .001, d = .89, 95\% \text{ CI } [-2.16, -1.19]$). Aphantasics also scored significantly higher on the social skill subscale (indicating poorer social skills; $M = 4.96, SD = 2.97$) compared to controls ($M = 3.85, SD = 2.56$), ($t(228.93) = -3.08, p_{corrected} = .01, d = .40, 95\% \text{ CI } [-1.82, -.40]$). There were no significant differences on the remaining three subscales: for communication (aphantasics: $M = 3.90, SD = 2.53$; controls: $M = 3.31, SD = 1.98; t(221.43) = -1.98, p_{corrected} = .245, d = .26, 95\% \text{ CI } [-1.17, -.002]$), attention-to-detail (aphantasics: $M = 4.85, SD = 2.34$; controls: $M = 5.14, SD = 2.32$), ($t(233.98) = .98, p_{corrected} = 1.65, d = .12, 95\% \text{ CI } [-.30, .89]$), and attention switching (aphantasics: $M = 5.56, SD = 2.47$; controls: $M = 5.32, SD = 2.05$), ($t(226.47) = -.80, p_{corrected} = 2.11, d = .11, 95\% \text{ CI } [-.82, .34]$). Our data are illustrated in Figure 4.

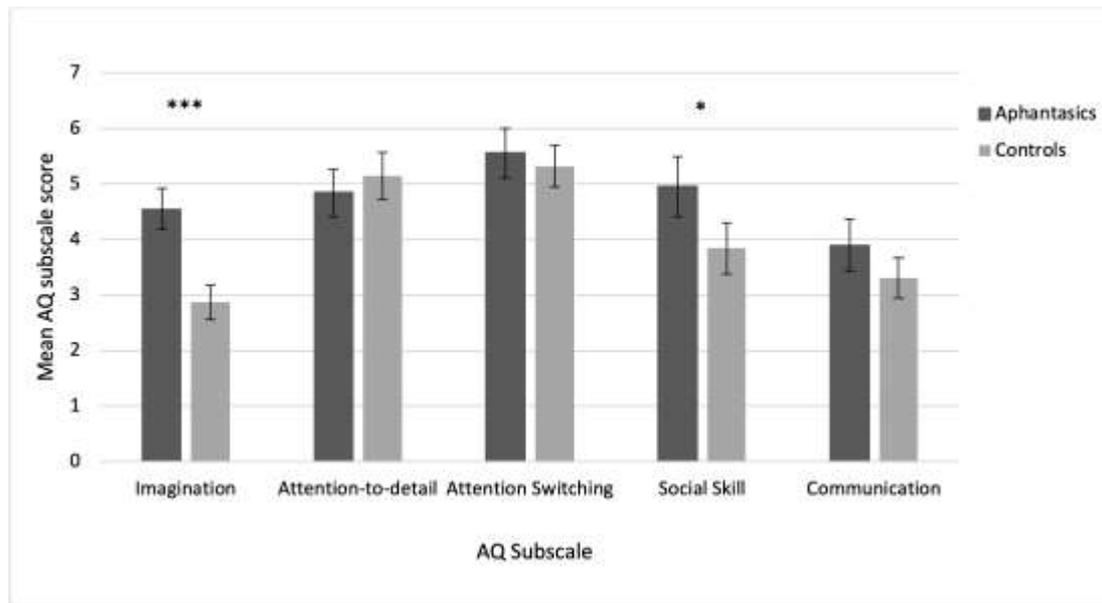


Figure 4. Mean AQ subscale scores (with 95% confidence intervals) as a function of group (aphantasics, controls). Higher scores indicate more autistic-like responses (e.g., poorer imagination and social skills). *Note.* Corrected *p* values shown at **p*<.05, ***p* < .01, ****p*<.001.

Finally, since AQ scores ≥ 32 indicate the possible presence of autism (Baron-Cohen et al., 2001), we also investigated whether there were more people with scores in this range in the aphantasic group, compared to our control group. There were 24 people in our aphantasic group with an overall AQ score ≥ 32 , and 4 people in our control group (again, overall AQ scores excluded question 3 for reasons stated above). This difference was highly significant [$\chi^2(1, N = 236) = 14.63, p < .001$; chi-square test with Yates continuity correction]. A bayes factor of 802.43 provides very strong evidence for the alternative hypotheses, i.e., a difference between groups in the number categorised as ≥ 32 AQ. Although we cannot draw conclusions about clinical diagnoses of autism in our sample, our results show that aphantasic people were far more likely to report levels of autism traits suggestive of an autism spectrum condition compared to controls.

10. Discussion

In Experiment 2 we have shown that people with aphantasia report higher AQ scores (more traits associated with autism than controls), and fall more often within the range

suggestive of autism (≥ 32). When examining subscales, we found AQ differences within the social skills subscale, and also within the imagination subscale (even when removing a confounding question that asks directly about aphantasia phenomenology). This shows that weak imagination symptomatology associated with autism may also be characteristic of aphantasia. Overall then, our results from Experiment 2 demonstrate a link between aphantasia and autism traits, not only in the expected imagination subscale, but also more broadly.

11. General Discussion

We investigated the relationship between aphantasia, and two other neurodevelopmental conditions: synaesthesia, and autism. In Experiment 1a we asked whether aphantasia and synaesthesia can co-occur, an important question given the conflicting visual imagery phenomenology assumed in aphantasia (low imagery; Zeman et al., 2015) and synaesthesia (high imagery; e.g., Spiller et al., 2015; but see Seron et al., 1992; Ward et al., 2018). We found that rates of synaesthesia were equal across aphantasics and non-aphantasics, showing that synaesthesia can indeed occur within aphantasic individuals. In Experiment 1b we replicated this with a larger sample, and a more robust measure of aphantasia. Here we also found that synaesthesia can arise even in the most extreme aphantasia cases where there is a total absence of visual imagery (at least in terms of self-reported imagery on the VVIQ). These results reinforce the fact that visual imagery is not necessary for the trait of synaesthesia to arise.

Our findings conflict with previous research suggesting that synaesthesia involves heightened visual imagery compared to controls (e.g., Barnett & Newell, 2008; Chiou et al., 2018; Chun & Hupé, 2016; Meier & Rothen, 2013; Price, 2009; Spiller et al., 2015) and we point to a hypothesis offered by Simner (2013). Simner has suggested that synaesthesia studies may sometimes exaggerate imagery differences in synaesthetes because they often test self-referred synaesthetes (and perhaps those with the most vivid phenomenological experience are

more likely to self-refer). To our knowledge, our sample size is the largest ever used to examine imagery in synaesthetes and this has allowed us to capture (and count) instances of low imagery synaesthetes who might otherwise be less obvious within smaller samples. It is interesting to note that we found aphantasic synaesthetes not only when screening a general population sample (Experiment 1a), but even when examining rather self-aware synaesthetes, who had made a self-motivated effort to navigate to an online testing site for synaesthesia (Experiment 1b). Hence, while higher imagery might make synaesthetes more self-aware of their synaesthesia (Ward et al., 2018), a complete absence of imagery does not preclude this.

In the present study we did, however, find that aphantasia influenced the way synaesthesia was experienced: aphantasic synaesthetes had stronger associator traits than synaesthetes without aphantasia. Importantly, associator synaesthesia not only encompasses colours in the ‘mind’s eye’ (potentially linked to imagery, but a phraseology used by aphantasics too) but also includes simply *knowing* what colours must be. This latter seems particularly compatible with the notion of aphantasia and may explain how aphantasics are just as likely as the general population to experience synaesthesia, albeit with associator-like traits. Our behavioural results mirror neuropsychological evidence from synaesthetes and aphantasics, who both appear to rely less on sensory cortices: aphantasics show lower activation in visual cortex during a mental imagery task (compared to high imagers; Logie, Pernet, Buonocore, & Sala, 2011), while associator synaesthetes have lower grey matter volume (compared to projector synaesthetes; Rouw & Scholte, 2010). It therefore appears that aphantasic individuals experience a synaesthesia characterised less by ‘perceiving’ synaesthetic colours, and more by an intrinsic awareness or ‘knowing’ of colours.

Although we found more associator-traits in aphantasic synaesthetes (vs. non-aphantasic synaesthetes) when treating scores on a continuum, we found no categorical effects: in both groups there were approximately 90% associators and 10% projectors. The projectors

with aphantasia are initially puzzling, because projectors experience synaesthetic colours like real-world percepts outside their body, and this has often been interpreted as very high imagery. One reason may simply be measurement error, but an alternative is that aphantasics might report projected synaesthesia in same way they report other visual knowledge – as a metaphor. For example, aphantasics could easily imagine someone standing opposite them, including the colour of their hair. Aphantasics would be likely to report that the hair–colour is ‘out there in space’ even though they have no visual imagery to form an iconic representation. Likewise, aphantasics may interpret projector items in the *PA Questionnaire* in a similar way. Put differently, a core feature of projector synaesthesia is the spatial location of the colour, and spatial relations are intact in aphantasics (Bainbridge, Pounder, Eardley, & Baker, 2020), and may even be stronger than usual (Keogh & Pearson, 2018). Intact spatial imagery could give aphantasics the ability to describe synaesthetic associations ‘in space’, despite being unable to image colours iconically. This opens a new and interesting debate about whether projectors should be defined by their percept-like experiences (possibly linked to strong imagery) or their external reference frames (possibly without imagery at all). We leave this debate open to explore in future work (see also Ward, Li, et al., 2006).

In Experiment 2 we used a standardised measure of autism traits (*Autism Quotient; AQ*; Baron-Cohen et al., 2001) and found that aphantasics scored higher than controls from the general population. We also found significantly more aphantasics (vs controls) over the threshold of 32 – suggestive of clinical levels of autism (Baron-Cohen et al., 2001). Previous research had conflicted on whether autism speaks to visual imagery. Whereas some have suggested low imagery in some autistics (i.e. ‘word-fact’ thinking styles; Grandin, 2009), others found no difference (e.g., Hughes et al., 2018; using the ‘*imagery ability*’ subscale of the *SCSQ*), while others still suggested imagery may be stronger (Esposito et al., 2016; Kana et al., 2006; Kunda & Goel, 2008, 2011; Soulières et al., 2011). Our own study shows clearly

that there is a link between poor imagery (aphantasia) and higher levels of autism traits. When looking within the subscales, we found a difference firstly for ‘imagination’. Poor imagination in people with low imagery suggests these concepts may be related – but we stress that they are *not identical* (something high imagers may find counter-intuitive). Our results show that even people with an *entire absence* of imagery can still report imagination, and some aphantasics reported very strong imagination indeed. To be clear, our finding shows that – as a group – aphantasics have a slight deficit in imagination, but this is not true for all, and imagination can nonetheless operate at a functional level even when imagery is absent. However, our group difference suggests that mental imagery may aid in the process of imagination, perhaps in the construction and maintenance of imagined scenarios. This suggestion has neurological parallels since areas involved in visual imagery (e.g., precuneus; Fletcher et al., 1995; Fulford et al., 2018) are activated when members of the general population are asked to imagine past and future events (Addis, Wong, & Schacter, 2007; Hassabis, Kumaran, & Maguire, 2007; Okuda et al., 2003; Schacter & Addis, 2007), and especially future events which arguably require more imagination (Szpunar, Watson, & McDermott, 2007). But similar studies have yet to be conducted on those without imagery, leaving it unclear whether imagining and imagery necessarily overlap by definition, or whether they simply overlap in most people. Nonetheless, our data show that low imagery and poor imagination are linked, and suggest, perhaps, that lacking visual imagery may drive – at least in part – some of the impairments in imagination that are widely seen in autism (e.g., Baron-Cohen, 1987; Davis et al., 2018; Jarrold et al., 1996; Ten Eycke & Müller, 2014).

We also found that aphantasics scored higher in the autistic subscale of social skills (i.e., had poorer social skills). This may rest on notions of theory of mind and perspective taking. Problems in ‘theory of mind’ (i.e., the ability to understand another’s mental state) have long been a defining feature of autism (Baron-Cohen, 2000; Baron-Cohen, Leslie, & Frith,

1985; Frith, 2001) and correlate with autistic deficits in social skills (Dawson & Fernald, 1987; Mazza et al., 2017; Perner, Frith, Leslie, & Leekam, 1989). According to a dominant view, theory of mind relies on perspective taking, which involves being able to mentally transform a representation of one's body from a first-person (ego-centric) point of view, to a third-person (hetero-centric) perspective (Decety & Grèzes, 2006; Vogeley & Fink, 2003). People with autism or those high in autistic traits often have impairments in this type of mental body transformation as well as in "embodiment" more broadly (simulating the mental and physical state of another within one's own body) (Conson et al., 2015; Gauthier et al., 2018; Kessler & Wang, 2012; Pearson, Marsh, Hamilton, & Ropar, 2014). Here, we suggest that visual imagery may be involved in this process of mental body transformation, helping individuals to take on the perspective of others. Low visual imagery may therefore give rise to difficulties in social skills by influencing how well an individual can perspective-take, and thereby understand the mental state of others.

One interesting consideration is how people high in autism traits are more likely to have aphantasia (as suggested here), but also synaesthesia (Baron-Cohen et al., 2013; Neufeld et al., 2013). Prior to our study this may have been a confusing finding, since autism and synaesthesia were assumed to have polar imagery requirements (low and high respectively). We have now shown, however, that having aphantasia does not preclude synaesthesia at all – and the slightly elevated rates of synaesthesia in autism may themselves be further evidence for the fact that high imagery is not a pre-requisite for synaesthesia to arise. Importantly, however, we note that the traits linking autism and aphantasia on the one hand (subscales of imagination and social skills), and autism and synaesthesia on the other (the subscale of attention to detail; Ward et al., 2017) seem to be very different.

One possible limitation of our study is that control participants were recruited differently to aphantasics in Experiment 2. Our recruitment of controls involved, at times, what

might be considered a more ‘social’ approach (i.e., 5% of controls were recruited via word of mouth), which may have lowered the mean AQ score for social skills in our control group (indicating ‘better’ social skills). However, most controls (81%) were recruited via online methods, which were either similar to methods for recruiting aphantasics (e.g., online groups/forums), or targeted a forum where rates of autism are known to be higher than the general population, not lower (i.e., 49% came from MTurk, which has been shown to have twice the rates of autism compared to real-world community samples; Chandler & Shapiro, 2016). The remaining controls (14%) were recruited via the participant recruitment system at the University of Sussex. As such, we suggest that our recruitment likely did not differentially influence AQ scores across aphantasics and controls. A second limitation is that we did not investigate clinical cases of autism, but looked at AQ trait profiles. This was an important step, given limited previous research investigating the relationship between autism and aphantasia, and the novelty of our research question. Our results provide – for the first time – evidence for aphantasia and autism being linked in some way, and future research may continue to investigate this association further.

A third limitation is that we diagnosed aphantasia using self-report measures. Although we used just a single question to identify aphantasia in Experiment 1a, we replicated our results using a multi-item questionnaire in Experiment 1b (*VVIQ-2*; Marks, 1995). The *VVIQ* (Marks, 1973, 1995) is the current gold standard measure of aphantasia, used widely in contemporary aphantasia research (e.g., Bainbridge et al., 2020; Dawes, Keogh, Andriillon, & Pearson, 2020; Jacobs et al., 2018; Keogh & Pearson, 2018; Zeman et al., 2015, 2020). Indeed, *VVIQ* scores map consistently onto behavioural measures of visual imagery (Pearson, 2014). In their imagery binocular rivalry task (see Introduction) scores on the *VVIQ* correlate positively with the degree of dominance the general population (Pearson et al., 2008, 2011; Rademaker & Pearson, 2012), suggesting that people generally have good metacognition about their visual

imagery abilities. Taken together, this shows that the VVIQ is a robust measure for classifying aphantasia.

One final limitation is our focus on one variant of synaesthesia only (grapheme-colour synaesthesia). However, we imagine a similar pattern of results for other visual synaesthetics (e.g., *taste-to-colour*, *sound-to-colour*; Downey, 1911; Ward, Huckstep, et al., 2006). And since people with aphantasia often experience imagery deficits in other sense modalities (i.e., imagery deficits in sound, taste, smell imagery etc.; Dance, Ward, & Simner, 2020; Dawes et al., 2020), we may yet find that people without taste imagery, for example, can nonetheless experience taste synaesthetics (i.e., lexical-gustatory synaesthesia, where words trigger tastes; Ipsier, Ward, & Simner, 2020; Ward & Simner, 2003). Future research asking questions such as these will further our understanding of the relationship between aphantasia and synaesthesia.

In conclusion, our results from the present study begin to characterise the relationship between aphantasia, synaesthesia and autism. We have shown that synaesthesia occurs at equal rates in aphantasia as in the general population, but that aphantasic synaesthetes show stronger associator (than projector) traits compared to ‘phantasic’ synaesthetes. This suggests that aphantasics experience synaesthesia phenomenologically differently, but that visual imagery is not necessary for synaesthesia to develop. We also found that it was possible for aphantasics to experience projector synaesthesia – a variant usually thought to require strong visual imagery. We have assumed this means that aphantasic projectors have an external spatial reference frame for their experiences, even if those experiences are not percept-like. Finally, we showed that people with aphantasia are higher in traits associated with autism, especially within the imagination and social skills subscales. This suggests that absent or weak visual imagery may play a part in driving weaknesses in these domains within autism spectrum conditions. In sum, our study serves to enhance our understanding of aphantasia, in showing its relationship to synaesthesia, and to autism traits.

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