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The prevalence of synaesthesia depends on early language learning

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ABSTRACT

According to one theory, synaesthesia develops, or is preserved, because it helps children learn. If so, it should be more common among adults who faced greater childhood learning challenges. In the largest survey of synaesthesia to date, the incidence of synaesthesia was compared among native speakers of languages with transparent (easier) and opaque (more difficult) orthographies. Contrary to our prediction, native speakers of Czech (transparent) were more likely to be synaesthetes than native speakers of English (opaque). However, exploratory analyses suggested that this was because more Czechs learned non-native second languages, which was strongly associated with synaesthesia, consistent with the learning hypothesis. Furthermore, the incidence of synaesthesia among speakers of opaque languages was double that among speakers of transparent languages other than Czech, also consistent with the learning hypothesis. These findings contribute to an emerging understanding of synaesthetic development as a complex and lengthy process with multiple causal influences.

1. Introduction

1.1. Learning and synaesthesia in the early years of research

During the first wave of scientific interest in synaesthesia, many researchers argued for a strong relationship between learning and synaesthesia (cf. Calkins, 1893, 1895; Flournoy, 1893; Galton, 1883; Jewanski, Simner, Day, & Ward, 2011; Marks, 1975). Mary Calkins, for example, argued that the unusual conscious experiences of adult synaesthetes develop in childhood "largely due to attention and to cultivation" of associations that the child has found "useful and pleasant" (Calkins, 1893). For example, when discussing number-form synaesthesia, in which numbers are experienced in a complex spatial arrangement in peripersonal space, Calkins argued that "...it is to the highest degree probable that most of these forms originate in the self-helping, topographical imagination of children introduced to the intricacies of number and word series" (Calkins, 1895). Calkins allowed that childhood synaesthesia might develop for other reasons (such as unusual neu-

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rophysiology), but suggested that even if this were the case, its preservation into adulthood depended on its being cultivated for learning (Calkins, 1893). Here we present a large-scale study that seeks to test the claim that childhood learning plays a causal role in the development or preservation of synaesthesia, by correlating the presence of synaesthesia in adults with their childhood language learning experiences.

1.2. A modern turn to genetics and rejection of learning

After more than fifty years of neglect (with a few notable exceptions such as Marks, 1975), synaesthesia re-emerged as a subject of serious scientific inquiry towards the end of the 20th Century (beginning with Cytowic, 1989a). At this time straightforward genetic accounts of its development gained prominence for a number of compelling reasons. The very strong tendency of synaesthesia to run in families, which had been reported for well over a century (Galton, 1883), was re-confirmed (Barnett et al., 2008; Baron-Cohen, Burt, Smith-Laittan, Harrison, & Bolton, 1996; Baron-Cohen, Harrison, Goldstein, & Wyke, 1993), and female:male ratios of 3:1 or higher were reported (Barnett et al., 2008; Baron-Cohen et al., 1993, 1996; Cytowic, 1989a). Furthermore, researchers at the time estimated the prevalence of synaesthesia to be 0.05% or lower (Baron-Cohen et al., 1996; Cytowic, 1989a, 1997; Ramachandran & Hubbard, 2001a). This overall pattern was consistent with synaesthesia being caused by a relatively rare x-chromosome-linked dominant mutation that is lethal to a substantial proportion of male embryos, similar to genetic diseases such as Rett syndrome (Bailey & Johnson, 1997).

While support for a genetic account was mounting, support for learned synaesthesia was dwindling. Many of the researchers responsible for the modern resurgence of interest in synaesthesia found it implausible that synaesthetic associations could be learned (cf. Cytowic, 1989b; Maurer, Gibson, & Spector, 2012; Ramachandran & Hubbard, 2003). Experiments using associative learning paradigms failed to produce any evidence of synaesthetic experiences (Elias, Saucier, Hardie, & Sarty, 2003; Meier & Rothen, 2009). Furthermore, synaesthetes themselves often describe their synaesthesia as being part of their earliest memories, and as being automatic, involuntary, and stable throughout their lives, characteristics that do not seem entirely compatible with a strong role for learning. Finally, researchers were at pains to establish that synaesthetic experiences (known as *concurrents*) were *genuinely perceptual*, and argued against critics who said that synaesthetic associations were learned (e.g. Ramachandran & Hubbard, 2001a, 2001b). These researchers and critics appeared united in assuming that people could not *learn* to have non-veridical perceptual experiences such as seeing the letter "A" as red. Researchers acknowledged that most stimuli that trigger synaesthesia (known as *inducers*), such as letters, were learned cultural artefacts. But aside from this prerequisite, synaesthesia was understood as fundamentally unrelated to learning.

1.3. The return of learning?

Recent research suggests a more nuanced view. While inheritance certainly plays a role in the development of synaesthesia, there is no single "synaesthesia gene": two genetic studies have implicated different loci (Asher et al., 2009; Tomson et al., 2011), and there is incomplete concordance in monozygotic twins (Bosley & Eagleman, 2015). Larger-scale surveys found a weak sex bias or none at all (Rouw & Scholte, 2016; Sagiv, Simner, Collins, Butterworth, & Ward, 2006; Simner & Carmichael, 2015; Simner et al., 2006; Ward & Simner, 2005), suggesting that previous studies had not detected a genuine sex difference, but merely found that women were more likely to respond positively to advertisements for synaesthesia studies. Furthermore, they found an overall incidence for synaesthesia of 4.4% (Simner et al., 2006), and an incidence of grapheme-colour synaesthesia of 1–1.4% (Carmichael, Down, Shillcock, Eagleman, & Simner, 2015; Simner & Carmichael, 2015; Simner et al., 2006). This is far less than very early reports (e.g. Calkins, 1895), but is orders of magnitude larger than hereditary illnesses such as Rett syndrome.

The evidence in favour of a learned aspect of synaesthetic development has also strengthened. Every known behavioral marker of synaesthesia can be produced in previously non-synaesthetic adults given associative training (Bor, Rothen, Schwartzman, Clayton, & Seth, 2014), trained participants report experiences similar to those reported by "natural" synaesthetes (Bor et al., 2014; Howells, 1944; MacLeod & Dunbar, 1988), and it is now established that many grapheme-colour synaesthetes learned their letter colours from childhood toys (Witthoft & Winawer, 2006; Witthoft, Winawer, & Eagleman, 2015). Furthermore, whatever adults may remember of their childhood synaesthesia, grapheme-colour synaesthetic associations are not stable in early life, and indeed their development continues for many years, at least into late childhood (Simner & Bain, 2013; Simner, Harrold, Creed, Monro, & Foulkes, 2009).

Thus it is not surprising that the idea that learning of some kind plays an important role in synaesthetic development has returned to prominence (cf. Asano & Yokosawa, 2013; Blazej & Cohen-Goldberg, 2015; Bor et al., 2014; Jürgens & Nikolic, 2012; Mroczko-Wasowicz & Nikolic, 2014; Price & Pearson, 2013; Simner, 2007; Simner & Bain, 2013; Simner et al., 2009; Ward & Simner, 2003; Ward, Simner, & Auyeung, 2005; Watson, Akins, & Enns, 2012; Watson, Akins, Spiker, Crawford, & Enns, 2014; Watson, Blair, Kozik, Akins, & Enns, 2012; Witthoft & Winawer, 2006, 2013; Witthoft et al., 2015; Yon & Press, 2014). The appropriate question is not "does learning influence synaesthetic development?", but rather "to what extent and in what manner does this influence occur?".