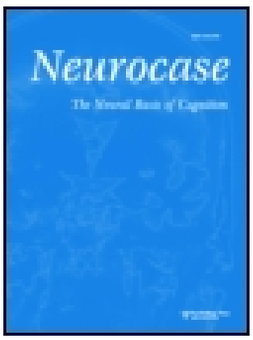

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REVIEW

Acquired and Inherited Forms of Cross-modal Correspondence

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Abstract

Coloured hearing synaesthesia (from the Greek *syn* [union] and *aisthesis* [sensation]), has been known to the scientific community for over 300 years and yet has gone relatively uninvestigated. In this review we chart recent research into the various forms of synaesthesia and contrast accounts of acquired and developmental forms of the condition. We also review the competing theories proposed to account for developmental forms, and conclude that preserved neonatal neural pathways between brain areas subserving audition and vision provide a likely explanation of the condition.

Cross-modal correspondences

In a recent issue of *Neurocase*, Halligan *et al.* (1996) report the case of an individual (DN) with left hemianaesthesia, who is seemingly only able to report tactile stimulation when allowed to observe the stimulus being applied. Halligan and colleagues suggest two possible explanations of this phenomenon, the first of which proposes that incoming visual information raises the stimulus to supra-threshold, whereas purely tactile stimulation fails to reach the threshold for detection. This may be analogous to the circumstances reported by Valler *et al.* (1993) in which the detection of tactile stimulation in patients with hemi-anaesthesia can be promoted by the introduction of water into the ear (caloric irrigation).

Their second hypothesis to explain the facilitation of tactile stimulus detection through vision is that their subject has acquired a form of synaesthesia in which visual information gives rise to a sensation of touch. This second explanation begs the question of what synaesthesia might be. In the following review we give an account of recent research with individuals with apparently inherited (developmental) forms of synaesthesia and contrast these findings with studies that have investigated acquired forms.

Definitions of synaesthesia

We, along with others (Marks, 1975; Cytowic, 1989a,b; Motluk, 1994) have chosen to define synaesthesia as occurring when stimulation of one sensory modality automatically triggers a perception in a second and in the absence of any direct stimulation to the latter. So, for example, a sound might automatically and instantly trigger the perception of vivid colour, or vice versa. Many combinations of synaesthesia are reported to occur naturally, including sound giving rise to visual percepts 'coloured-hearing' and smell giving rise to tactile sensation, as in Cytowic's (1993) subject MW. Our experience suggests that coloured-hearing synaesthesia is by far the most common form and that certain combinations of synaesthesia almost never occur (e.g. touch to hearing).

Acquired synaesthesia

A variety of neuropathological conditions have apparently given rise to acquired synaesthesias. In one of the earliest accounts Carnaz (1851) [cited in Krohn (1893) p. 33] speculated that synaesthesia of all forms was 'pathological and due to some optical lesion' and could therefore be seen as due to 'hyperaesthesia of the sense of colour'. Whilst

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being an unlikely explanation for all forms of synaesthesia, a number of more recent studies have reported instances of synaesthesia as a consequence of visual loss involving anterior visual pathways. For example, Lessell and Cohen (1979) reported three patients with unilateral eye or optic nerve disease who saw phosphenes (patches of light) only when they heard noises. Bender *et al.* (1982) also reported a patient with optical neuritis who saw a blue light whenever he heard a loud noise or experienced sudden pain. Additionally, Vike *et al.* (1984) report a man who when auditorily stimulated with 65 dB clicks saw kaleidoscopic and spiralling lights in his left eye. On removal of a large cystic mass extending from his left medial temporal region to the midbrain, his synaesthesia stopped. Rather more complex synaesthesia was reported by a subject studied by Rizzo and Eslinger (1989). This 17-year-old male, who had suffered perinatal visual deprivation as a consequence of retrolental fibroplasia, showed unusual associations between musical notes and coloured shapes. Perhaps one of the most extraordinary cases of acquired synaesthesia is that reported by Coriat (1913). The subject of report was said to experience coloured pain as the consequence of an attack of 'hysterical hemicrania' (possibly a description of migraine).

Thus it seems that most cases of acquired synaesthesia arise as a consequence of damage to anterior portions of the brain, often the optic nerve. In acquired forms the synaesthesia is usually little more than photisms or phosphenes, and typically lacks the complexities found in developmental synaesthesia. For example, subject EP, a developmental case studied by Baron-Cohen and colleagues (1987), reported seeing 'Darkish grey, with spinach green and pale blue' on hearing the word 'Moscow'. This description is markedly more complex than the descriptions given in the studies of subjects with acquired synaesthesia who tend to report simple, one-colour flashes of light. Since testing EP we have come across other developmental cases in which equally rich colour experiences are triggered by pain, touch and taste. In the following section we discuss some other characteristics of developmental synaesthesia which distinguish it from acquired forms.

Developmental synaesthesia

Developmental forms of synaesthesia often prompt scepticism in the minds of many neuroscientists. Given that the primary data are usually the phenomenological accounts given by synaesthetes, such doubt seems understandable. Establishing the existence of synaesthesia has been dependent upon the presence of 'symptoms'. However, the remarkable similarity in the accounts given by individuals with synaesthesia about their experience (see Baron-Cohen *et al.*, 1993; Harrison and Baron-Cohen, 1994, 1995) suggests that the condition is real and deserving of serious scientific attention. As Korb (1996) has recently observed:

"The probability that some large number of people, independently reporting that they have synaesthesia, would go on to report such a close match in their phenomenologies – and whose reported cross-modal linkages would be close to invariant after such long periods – without there being a common underlying physical structure (innate or acquired) to explain it is vanishingly small."

Korb's observation testifies to the existence of the condition, but relies upon the phenomenological accounts given by subjects. Recent investigations of the developmental form of the condition have sought to go beyond the phenomenological accounts and to obtain objective data. However, it is worth remarking at this point that reports of acquired forms do not seem to have been greeted with the same scepticism as developmental synaesthesia. This seems remarkable given that the data in both instances have been the subjective accounts given by the subjects studied. Admittedly, patients with acquired forms of synaesthesia have a demonstrable lesion, though, as we discuss later in this review, it is by no means apparent that the acquired forms can be 'explained' by the presence of brain damage.

Psychological investigations of developmental synaesthesia

Recent investigations of developmental synaesthesia have aimed to provide evidence more analogous to 'signs' than 'symptoms' of the condition. The study of subject EP (Baron-Cohen *et al.*, 1987) capitalized on the fact that for individuals with coloured-hearing synaesthesia, the colour–word or colour–sound correspondence is consistent and invariant across time. In order to provide objective evidence of the condition, Baron-Cohen and colleagues made detailed notes of subject EP's colour descriptions for over 100 words and letters. Ten weeks later, and without prior warning, EP was retested on the entire list and a comparison made between the descriptions given on each of the two occasions. Her performance was found to be totally consistent, a marked contrast to the performance of a control subject who was able to manage only 17% consistency one week later, in spite of having been prewarned of a retest.

A later group study of a further nine synaesthetes using a paradigm similar to that used with subject EP also found remarkably good performance in synaesthetes as compared with a group of controls. However, even such striking results as these could have been due to superior recall memories, though neuropsychological testing suggests that memory performance in these subjects is unremarkable, falling well within the normal range. These two studies provided the first objective evidence in support of the existence of developmental synaesthesia.

Functional neuroimaging studies of developmental synaesthesia

Recent developments in neuroimaging have allowed us to examine the human brain *in vivo* in a bid to uncover the functional anatomy of synaesthesia. Paulesu *et al.* (1995) have recently used Positron Emission Tomography (PET) to look for differences in regional Cerebral Blood Flow (rCBF) in synaesthesic subjects as compared with non-synaesthesic controls. This study showed that subjects with synaesthesia exhibited increased rCBF, as compared with control subjects, in two visual association areas (posterior infero-temporal cortex and the junction of the occipital and parietal cortices) when listening to words as compared to listening to tones (all subjects were blindfolded when being scanned). This study and a subsequent single case replication using functional magnetic resonance imaging (MRI) suggest that posterior cortical structures are involved in developmental synaesthesia. This provides a further difference between acquired and developmental forms of the condition, in that it is generally damage to anterior pathways that gives rise to the former.

Whilst these studies testify to the existence of the condition, they are mute regarding the causes. There has been no shortage of theories proposed to explain synaesthesia and in the following section we review the major accounts.

Learned association

One theory, originally proposed by Calkins (1893), argues that individuals who report 'seeing' colours upon hearing sounds have simply learned to associate particular colours with specific sounds. Note, however, that people with synaesthesia are typically unable to recall having learned these specific colour associations and often the associations are not particularly useful ones, with some subjects we have tested revealing that consecutive letters have been different shades of the same colour. Additionally, a comparison of the coloured alphabets of twins shows substantial variation in the colour–letter correspondences made by each of the pair. Variation exists between siblings, and between mothers and daughters in the same family. It is surprising that there is not greater similarity in the colour–letter correspondences of family members if coloured alphabets are acquired as learned associations.

Sensory leakage theory

Jacobs and colleagues (1981) proposed what we shall call the 'sensory leakage theory'. This is an account of how simple photisms arise in cases of acquired synaesthesia, though it could in principle be extended to account for developmental synaesthesia. The essence of this theory is that auditory information 'leaks' into pathways and areas in the brain that ordinarily deal with visual information. They suggest that the likely neural location for this leakage is the lateral geniculate nucleus, though they point out that there are 'numerous regions of the brain where visual and auditory pathways lie in close anatomic proximity' (p. 216). It is at these points that post-synaptic fibres might converge to cause the synaesthesia seen in other pathological states, such as congenital blindness and drug intoxication.

An important caveat to attach to the study by Jacobs and colleagues is that close examination of the patients reported reveals that four of these patients (cases 1, 2, 4 and 7) also experienced photisms in the *absence* of auditory stimulation, casting doubt on whether these instances should be described as cases of auditory visual synaesthesia at all. It is also worth observing that seven patients always experienced their photisms when they were 'relaxed, drowsy or dozing' (p. 214), circumstances in which hypnagogic hallucinations are well known to occur.

Bimodal neurons and disconnection

Evidence to support leakage between areas subserving different forms of sensory information is sparse, causing some difficulties for the theory of Jacobs *et al.* (1981). However, recent work suggests that rather than posit leakage, it is possible to find classes of neurons that are responsive to stimulation from more than one sensory modality. In the study carried out by Graziano *et al.* (1994), recordings were made from neurons ($n = 141$) in the ventral portion of the premotor cortex. Of these neurons, 27–31% were found to be bimodally responsive, firing as a result of either, or both, visual and somaesthetic stimulation. The existence of bimodally responsive neurons provides the potential for ambiguity with respect to how other brain areas interpret the firing of these neurons. Presumably, under normal circumstances, areas that deal with afferent sensory information provide details of the source of these data. However, as suggested by Halligan *et al.* (1996), when normal brain function is disrupted, these data might be lost. The patient reported by Halligan *et al.* (1996) might provide an example of this, in that as a result of brain damage causing hemianaesthesia, he is unable to feel tactile stimuli administered to his left side. However, when permitted to observe the application of the stimulus he reports tactile sensation. If the theory of Halligan and colleagues turns out to be an accurate account of DN's condition, then this provides an example of how the brain seeks to provide a meaningful interpretation of incoming sensory stimulation when deprived of a full complement of information. The disconnection theme also forms a major part of Cytowic's theory of the cause of synaesthesia, reviewed next.

Cytowic's theory of synaesthesia

Perhaps the most controversial theoretical account of synaesthesia is that most recently advanced by Cytowic

(1993). He proposes that synaesthesia occurs because 'parts of the brain get disconnected from one another ... causing the normal processes of the limbic system to be released, bared to consciousness, and experienced as synaesthesia' (p. 163). An analogy is drawn with migraine, based on the notion that in both conditions a stimulus causes a rebalancing of regional metabolism. Cytowic maps the analogy by pointing out that both synaesthesia and migraine (of some kinds) are evoked by a stimulus, and consequently, just as the migraine stimulus causes metabolic and circulatory changes, so too does the stimulus in synaesthesia.

Cytowic's principal evidence for the involvement of the limbic system is the 'stunning shut-down of the cortex' (p. 152) observed in the ^{133}xenon studies of rCBF in MW's brain. Direct evidence of the involvement of the limbic system would have been provided by evidence of blood flow changes in this brain region, though unfortunately neuroimaging using ^{133}xenon inhalation does not permit such deep structures to be imaged. Fortunately this is not a limitation shared by PET and so the importance of the limbic system in synaesthesia can be evaluated using this technique. Paulesu *et al.* (1995) report that in the comparison of control subjects with synaesthetes, as well as in the within-subjects comparison of words with tones in synaesthetes, considerable differences in rCBF were found. However, none of these differences were reported to be in the limbic system, casting doubt upon the involvement of this region in synaesthesia.

Preserved neural connectivity

We have couched our explanation of synaesthesia at three levels: functional, structural and genetic. Our functional account of the condition (Baron-Cohen *et al.*, 1993) sees synaesthesia as a breakdown of the normal *modular* structure of any sensory modality (Fodor, 1983). This implies that people with developmental synaesthesia possess abnormally structured brains in which for example 'auditory' data are passed via neural pathways to areas of the brain dealing with vision, thus linking the perceptual experience of hearing with a visual dimension, typically of colour (Baron-Cohen *et al.*, 1987, 1993).

Direct evidence for the existence of such pathways in cases of developmental synaesthesia is not currently available. However, research looking at neonatal development in other species suggests that transient pathways connecting visual and auditory areas are found in other species [e.g. the cat (Dehay *et al.*, 1984), and the macaque monkey (Kennedy *et al.*, 1989)]. Additionally, work reviewed by Maurer (1993) suggests that (1) human neonates engage in cross-modal matching (Lewkowicz and Turkewitz, 1980), and (2) auditory stimuli elicit a clear response from the occiput of human neonates (Neville, 1993). This implies that similar pathways may exist in humans during infancy.

The notion that developmental synaesthesia is due to preserved neural connections begs the question of why, in a relatively small group of individuals, such pathways should be preserved. We have recently found evidence that suggests synaesthesia may be inherited. This is not a new proposition: Galton (1883) suggested that 'the tendency is very hereditary' (p. 107). Our recent pedigree study of seven families is the first evidence that suggests a clear genetic link.

A neuropsychology of synaesthesia?

To return to the idea that synaesthesia might be due to a breakdown in modularity: This proposition fits well with what is, to the best of our knowledge, the only case of the loss of synaesthesia as a result of brain damage, patient JI reported by Sacks and Wasserman (1987). 'JI' suffered brain damage as the result of a car accident and as a consequence became cortically colour-blind ('achromatopsic'). However, when JI lost his ability to see colour, he also lost his coloured-hearing synaesthesia as well as his ability to dream in colour. Such a pattern of dysfunction opens up the possibility that these capacities may share a common neural substrate.

Conclusions

The combination of consistent phenomenological accounts of synaesthesia, together with the data collected using the traditional techniques of experimental psychology, suggests that developmental synaesthesia is a discrete condition. This work has furthered our understanding of the condition by lifting synaesthesia out of what Humphreys (1990) called the category of 'romantic neurology', and approaching it with serious scientific investigation. Whatever its basis, our contention is that the study of synaesthesia may prove a useful tool for understanding brain function.

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