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Review

Mirror-sensory synaesthesia: Exploring 'shared' sensory experiences as synaesthesia

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ABSTRACT

Recent research suggests the observation or imagination of somatosensory stimulation in another (e.g., touch or pain) can induce a similar somatosensory experience in oneself. Some researchers have presented this experience as a type of synaesthesia, whereas others consider it an extreme experience of an otherwise normal perception. Here, we present an argument that these descriptions are not mutually exclusive. They may describe the extreme version of the normal process of understanding somatosensation in others. It becomes synaesthesia, however, when this process results in a conscious experience comparable to the observed person's state. We describe these experiences as 'mirror-sensory synaesthesia'; a type of synaesthesia identified by its distinct social component where the induced synaesthetic experience is a *similar* sensory experience to that perceived in another person. Through the operationalisation of this intriguing experience as synaesthesia, existing neurobiological models of synaesthesia can be used as a framework to explore how mechanisms may act upon social cognitive processes to produce conscious experiences similar to another person's observed state.

Contents

1. Introduction

Synaesthesia is a phenomenon in which an unusual perceptual experience occurs in response to ordinary stimulation (Rich and Mattingley, 2002). Synaesthesia has been estimated to occur in up to 4% of the population (Simner et al., 2006), a number much higher than earlier estimates would suggest (Baron-Cohen et al., 1996; Cytowic, 1989). Some of the most well-known forms of synaesthesia include grapheme-colour and phoneme-colour synaesthesia: where words, letters or digits elicit an experience of colour when seen or heard (Rich and Mattingley, 2002). For example, grapheme-colour synaesthesia has been estimated to have a prevalence rate

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of 1.4% (Simner et al., 2006). However, synaesthesia has been used to describe many other variants, with up to 63 currently reported (Day, 2010). With such a high number of variants, it is not surprising that there may be disagreement between researchers in what criteria are required to describe an experience as a synaesthesia (see Simner, 2010).

In the last decade, there have been cases in the literature of what we call 'mirror-sensory synaesthesia,' describing the experiences of mirror-touch and mirror-pain synaesthesia (also known as synaesthetic touch and synaesthetic pain, respectively). Mirror-touch synaesthesia occurs when the observation of tactile stimulation to another induces the experience of being touched (e.g., Blakemore et al., 2005). Similarly, mirror-pain synaesthesia is when the observation of noxious stimulation to another induces an actual experience of pain (e.g., see Giummarra and Bradshaw, 2008). These variants of mirror-sensory synaesthesia are unlike typical touch or pain, where sensory receptors respond to contact to the self (e.g., pain: see Melzack and Wall, 1965). Rather, mirror-sensory synaesthesia is triggered by the visual processing of touch or pain experienced by another person. Mirror-sensory synaesthesia can occur seemingly developmentally (e.g., Banissy et al., 2009; Osborn and Derbyshire, 2010), or be acquired following pain-related trauma (Fitzgibbon et al., 2010b) or transiently induced through perceptual manipulations (Ramachandran and Brang, 2009). This type of synaesthesia may be just as, if not more, common than other types such as grapheme-colour synaesthesia. Research on mirror-touch synaesthesia, for example, reports an incidence rate of 1.6% in a general population sample (Banissy et al., 2009). No objective measure of mirror-pain synaesthesia has yet been established. However, approximately 30% of the general population may experience what we have described as mirror-pain synaesthesia (Osborn and Derbyshire, 2010), and reports of acquired mirror-pain synaesthesia suggest up to 16% of amputees experience this phenomenon (Fitzgibbon et al., 2010a).

In a recent review, it was proposed that mirror-sensory synaesthesia may come about through activation within the somatosensory cortices that is similarly seen during actual somatosensation (Keysers et al., 2010). In a related review, we proposed that mirror-pain synaesthesia in amputees may come about through disinhibition of systems involved in 'empathy for pain': understanding another's pain experience (Fitzgibbon et al., 2010b). Both these reviews address the potential mechanisms for mirror-sensory synaesthesia but do not operationalise the phenomena, with the former not addressing acquired variants, and the latter only addressing acquired mirror-pain synaesthesia. As a large body of experimental work has begun to emerge in this area (e.g., Banissy et al., 2009, 2011; Banissy and Ward, 2007; Fitzgibbon et al., in press, 2010a; Giummarra et al., 2010; Goller et al., in press; Osborn and Derbyshire, 2010), it is important to operationalise this phenomena.

In this review, we will propose these mirror-sensory experiences are best classified as a type of synaesthesia. Although they may represent the extreme version of otherwise normal processes of understanding others, when another person's sensation becomes consciously "shared" it then becomes synaesthesia. We speculate that the unique feature of mirror-sensory synaesthesia, the transference of a similar sensation another person is experiencing onto oneself, may implicate social cognitive mechanisms. Through the operationalisation of mirror-sensory synaesthesia as a type of synaesthesia we are able to combine a neurobiological model of social cognition with existing neurobiological models already proposed for synaesthesia. By doing so, we can for the first time offer an explanation for both congenital and acquired variants of mirror-sensory synaesthesia. Although these neural systems may be related to empathy, evidence of increased empathy in mirror-sensory synaesthetes may be a by-product and not the cause of mirror-sensory synaesthesia.

2. Synaesthesia

Synaesthesia occurs when stimulation in one modality ("inducer") results in an unusual experience in another or within the same modality ("concurrent") (Grossenbacher and Lovelace, 2001) (extensive reviews on synaesthesia can be found elsewhere, e.g., Hubbard and Ramachandran, 2005; Rich and Mattingley, 2002). Examples include the experience of taste from words (Ward and Simner, 2003), the experience of shapes from tastes (Cytowic, 1993), to, perhaps most well-known, the experience of colours from letters and numbers. However, even within categories of synaesthesia (e.g., grapheme-colour), there is variation in individual experiences. For example, the colours perceived in grapheme-colour synaesthesia can appear as a mist, in the shape of the letter, out in space or in the mind's eye (Dixon et al., 2004; Rich et al., 2005). Such variation in experience has led some to argue that current definitional features of synaesthesia may not be present across all variants (Simner, 2010). Moreover, although most often synaesthesia is used to describe experiences present since early childhood (developmental), it has also been used to describe experiences acquired later in life following brain injury (e.g., Ro et al., 2007; Vike et al., 1984), sensory deafferentation (e.g., Armel and Ramachandran, 1999), amputation (e.g., Fitzgibbon et al., 2010a) or other pain-related conditions (e.g., Villemure et al., 2006). Even transient experiences induced during the use of hallucinogenic drugs have been described as synaesthesia (e.g., Aghajanian and Marek, 1999).

Although there are different forms of synaesthesia, they all differ from other types of 'normal' and 'abnormal' sensory processing in multiple ways. First, they are set apart from normal sensory experiences as they are not common in the general population, are involuntary, and tend to be constant over time (Rich and Mattingley, 2002) (although, for a discussion against certain definitional criteria including consistency, see Simner, 2010). Moreover, although normal perception requires us to integrate incoming sensory information from multiple sources to understand our environment and events, only one sensory input is required to produce a synaesthetic experience (Sagiv et al., 2011). Second, they are different to abnormal sensory experiences seen in pathological conditions in that rather than a lack of function, synaesthesia involves the elicitation of an event; a positive symptom. Further, with the exception of particular brain injuries, synaesthesia typically does not occur within a psychiatric or neurological context (Ward and Mattingley, 2006). For example, an auditory hallucination in schizophrenia would not be considered a synaesthetic experience. Hallucinations generally occur spontaneously, whereas in synaesthesia, sensory input is required to trigger the experience and is often predictable. In addition, synaesthetes rarely get confused between synaesthetic experiences and 'reality'. In contrast, a hallmark of hallucinations is the lack of distinction between the internally and externally triggered experiences. Somatosensory flashbacks in response to triggers related to a traumatic event, as has been reported in post-traumatic stress-disorder (Whalley et al., 2007), are also not consistent with a definition for synaesthesia. In this case, a pain memory is induced within the context of a psychiatric illness.

Multiple mechanisms have been proposed to explain synaesthesia (for a review, see Hubbard and Ramachandran, 2005). A possible genetic influence is supported by the finding that people who experience synaesthesia are likely to have biological relatives who are also synaesthetes (Baron-Cohen et al., 1996). Moreover, females are more likely to report synaesthesia than males (Barnett et al.,

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2008; Baron-Cohen et al., 1996; Rich et al., 2005). However, there are also large numbers of synaesthetes without a biological relative with synaesthesia (e.g., Rich et al., 2005), and even a case where only one of a pair of monozygotic twins had synaesthesia (Smilek et al., 2001). Further, other studies have shown no sex bias in the prevalence of synaesthesia (Simner et al., 2006). This discrepancy may reflect the self-referral sampling methods in previous studies (e.g., Ward and Simner, 2005), which may result in an inflated female–male ratio as women generally seem more likely than men to contact a research group (Rich et al., 2005). Even if synaesthesia does occur in families, it is not always the specific type of synaesthesia that is inherited, rather that one's chance of manifesting a type of synaesthesia is increased (Barnett et al., 2008). Thus, although there may be a genetic component, it is not clear how strong an influence it has on the development of synaesthesia in an individual.

At a neural level, there are two primary accounts proposed for synaesthesia and its variability. These accounts argue either for a primary structural or functional difference in the brains of synaesthetes that results in cross activation between brain regions (Bargary and Mitchell, 2008). Structural explanations suggest that synaesthesia is the outcome of atypical connections between brain areas involved in sensory processing (Baron-Cohen et al., 1993). In particular, some synapses connecting one sensory cortical area with another may not be pruned during early development (Maurer, 1997; Ramachandran and Hubbard, 2001), perhaps due to genetic factors (Ward and Simner, 2005) resulting in altered cortical connectivity compared with non-synaesthetes (Bargary and Mitchell, 2008; Rouw and Scholte, 2007). In a recent diffusion tensor imaging (DTI) study, Rouw and Scholte (2007) found an increase in fractional anisotropy in a group of grapheme-colour synaesthetes compared to non-synaesthetes, which they interpret as indicating atypical structural connectivity in synaesthetes. Note however, both structural and functional differences between synaesthetes and non-synaesthetes have not always been well replicated (e.g., Hupe et al., in press), and that such results do not indicate what caused this difference in signal: If there are differences in connectivity in synaesthetes and non-synaesthetes, it is not clear whether structural differences, mediated by genetic or other factors, caused synaesthesia, or whether the increased connectivity is instead a consequence of synaesthesia (Cohen Kadosh and Walsh, 2008).

Another possibility is that cross activation occurs through altered function in brain areas of synaesthetes (Cohen Kadosh et al., 2009; Grossenbacher and Lovelace, 2001). One such proposal is that synaesthesia is the result of disinhibited feedback in otherwise normal neural pathways. For instance, sensory signals are generally discharged to multi-sensory areas through forward connections, which are then met by feedback connections whereby top–down signalling prevents multi-sensory experiences. For synaesthetes, however, disinhibition in the feedback connections might bring about another sensory experience (Grossenbacher and Lovelace, 2001). Alternatively, disinhibited feedback may come about *within* a brain area through the disinhibition of irrelevant unimodal neurons (Cohen Kadosh and Henik, 2007), for example, activation of unimodal neurons in the auditory cortex as a result of visual and somatosensory stimulation (e.g., Brosch et al., 2005). In support of such functional explanations, a recent study showed that under post-hypnotic suggestion, synaesthetic experiences can arise in non-synaesthetes (Cohen Kadosh et al., 2009). This suggests that hyperconnectivity is not a prerequisite for synaesthesia. In addition, these experimental findings imply that previous findings of apparent 'structural' differences in synaesthetes (see Rouw and Scholte, 2007) may not be causal but rather a by-product of changes in inhibitory processing (Cohen Kadosh and Henik, 2007; Cohen Kadosh and Walsh, 2008).

Due to the wide range in type and interpersonal variability in synaesthetic experience (e.g., experiencing one type vs. experiencing multiple types; acquired vs. developmental etc.), it seems possible that there may not be a one-size fits-all explanation for all types of synaesthesia (Cohen Kadosh and Terhune, 2011). This heterogeneity of synaesthetic experience is reminiscent of phantom limb phenomena, in which there are also multiple mechanisms proposed (both structural and functional) (for a discussion, see Hubbard and Ramachandran, 2005). For some amputees, phantom sensations begin almost immediately following the loss of a limb supporting an immediate functional change perhaps through the unmasking of existing synapses (e.g., Borsook et al., 1998). In fact, phantom limb sensations can even be experienced in non-amputees during regional anaesthesia (e.g., Paqueron et al., 2004). However, structural changes are also implicated in phantom limb phenomena with the specific remapping of the somatosensory and even thalamic cortices following amputation (Flor et al., 2006; Reilly and Sirigu, 2008).

To explore the possibility of multiple mechanisms involved in mirror-sensory synaesthesia, it is important to differentiate between main types of synaesthesia, considering their heterogeneity, and identify differentiating characteristics. In the case of mirror-sensory synaesthesia, this is critical, as these experiences, although not necessarily a disorder, are potentially maladaptive to the individual. This is particularly true in the case of mirror-pain synaesthesia, which is not only unpleasant, but may interfere with everyday life. Although different types of synaesthesia may differ in the specific mechanisms, a better understanding of each type individually will enhance our knowledge of how they fit together and the potential for shared features and mechanisms.

3. Mirror-sensory synaesthesia

Typically, a synaesthetic experience is induced in a visual, auditory, olfactory or gustatory domain resulting in an unpredictable (although not completely idiosyncratic; see Rich et al., 2005; Simner et al., 2005) experience in another such domain. The sensory modalities of touch (tactition) and pain (nociception) are part of the somatosensory system, and can be induced synaesthetically in response to viewing that same sensation in another (e.g. Banissy et al., 2009). Unlike other synaesthesia, it has been suggested that 'mirror systems' are implicated in the experience of mirror-touch and mirror-pain synaesthesia (e.g. Blakemore et al., 2005). Mirror systems refer to cortical areas that respond both to observing another person's state and being in that same state oneself (Decety and Jackson, 2004; Gallese, 2003; Preston and de Waal, 2002). Mirror systems are thought to be made up of mirror neurons that were first reported as responding to both observation of an action and performing that action in the ventral premotor cortex (F5) and the parietal area (PF) of monkeys (di Pellegrino et al., 1992). These areas have become known as the classical mirror neuron areas (for a review, see Rizzolatti and Craighero, 2004).

Human mirror systems may extend beyond the classical regions observed in monkeys (for a review, see Keysers and Gazzola, 2009, 2010). Mirror systems have been reported in humans for not only actions (Rizzolatti et al., 1996), but also for emotions (for a review, see Bastiaansen et al., 2009) such as disgust (Wicker et al., 2003), and for facial expressions (Carr et al., 2003). Similar to mirror systems for action and emotion (for a review, see Gallese, 2007; Iacoboni and Dapretto, 2006; Rizzolatti and Craighero, 2004), mirror systems for sensation have also been reported, specifically for pain (e.g. Avenanti et al., 2005; Jackson et al., 2005) and touch (e.g. Blakemore et al., 2005; Keysers et al., 2004). This suggests that when observing touch and pain in another, we activate similar cortical areas as when we are touched or experience pain ourselves. This

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'mirror effect' has been interpreted as reflecting processes involved in allowing us to understand others (for a review, see Iacoboni, 2009). However, others argue that mirror systems are not an adaptation for social understanding, only playing a minor role in these processes (Hickok, 2009) (we explore this issue further in Section 5.3), and others even suggest that there is little to no relationship between the mirror neurons first identified in monkeys and the mirror activity reported in humans (Dinstein et al., 2008; Turella et al., 2008). Here we summarise the literature describing mirror-sensory synaesthetic experiences and investigations of this unique experience that may be mediated through hyperactivity of mirror system activity (discussed in Section 5).

3.1. Mirror-touch synaesthesia

The observation of touch has long been considered a purely visual event. However, new evidence suggests that a somatosensory component is also activated in the observer; observed touch is processed in both visual parts of the brain, and in somatosensory areas (Blakemore et al., 2005; Ebisch et al., 2008). Further, for some people seeing another being touched can result in a first-hand tactile sensation to the self: mirror-touch synaesthesia (Banissy et al., 2009; Blakemore et al., 2005). Like other forms of synaesthesia, mirror-touch synaesthesia has been reported following brain damage (Halligan et al., 1996, 1997), amputation (Ramachandran and Brang, 2009) and in otherwise normal individuals (Blakemore et al., 2005).

The first functional magnetic resonance imaging (fMRI) study of mirror-touch synaesthesia showed that similar areas of the brain involved in physical touch are activated in the experience of mirror-touch synaesthesia (Blakemore et al., 2005). In fact, both non-mirror-touch synaesthetes and a mirror-touch synaesthete activate overlapping areas of the brain when observing and experiencing touch (the 'tactile mirror system'), including premotor and parietal regions, and the primary and secondary somatosensory cortices. However, when comparing the neural activation of a mirror-touch synaesthete to non-synaesthetes when observing touch, Blakemore et al. (2005) observed greater activation within somatosensory cortices as well as bilateral activation of the anterior insula, in the synaesthete.

Based on these data, Banissy and Ward (2007) hypothesised that mirror-touch synaesthetes should be unable to discriminate real from synaesthetic touch. They tested mirror-touch synaesthetes with a visual-tactile spatial congruity paradigm, where they asked participants to report where they were being touched (cheeks vs. hands) while, at the same time, observing touch delivered to another person. On congruent trials, the observed touch was delivered to the same location as the touch delivered to the participant. On incongruent trials, the observed touch was on a different location to where it was on the participant. The authors found that mirror-touch synaesthetes performed slower on incongruent trials compared to congruent trials, and made more errors overall than controls. These results provide the first objective behavioural evidence of the authenticity of mirror-touch synaesthesia as they indicate that mirror-touch synaesthetes have greater difficulty in discriminating between real and synaesthetic touch. They also found that mirror-touch synaesthetes scored higher than controls on the emotional reactivity subscale of the empathy quotient (EQ), suggesting that the enhanced mirror system activity reported by Blakemore et al. (2005) may be modulated by empathy.

The prevalence of mirror-touch synaesthesia may be as high as 1.6% in the general population (Banissy et al., 2009). This estimate of 1.6% prevalence makes mirror-touch synaesthesia one of the most common forms of synaesthesia, alongside, for example, grapheme-colour synaesthesia with a prevalence of 1.4% (Simner et al., 2006). This estimate was obtained by first asking an undergraduate student population the question 'do you experience touch sensations on your own body when you see them on another person's body?' to which 10.8% responded positively. To test the validity of this response, those who responded positively went on to view a series of videos depicting touch; of these, only 2.5% reported an induced somatic sensation. This group of potential mirror-touch synaesthetes then completed the visual-tactile spatial congruity task described above (see Banissy and Ward, 2007), which identified 1.6% of all participants as true mirror-touch synaesthetes, based on reaction time differences between congruent and incongruent trials, and/or more errors than controls. The authors ruled out the possibility that these findings are due to somatotopic cueing, where a visual event prompts tactile processing, as they found no significant difference between groups when participants observed the projection of light upon a face rather than the face being touched.

To determine characteristics of mirror-touch synaesthesia, Banissy et al. (2009) merged the results of the 1.6% of true mirror-touch synaesthetes with 12 mirror-touch synaesthetes (total $N = 21$) previously identified by the group. No difference was found between these groups in reaction time or error type on the paradigm described above. When reporting these mirror-touch synaesthetes' responses to videos depicting touch, the authors found the experience was: (1) relatively specific to touch on observed to corporeal bodies, it did not occur with inanimate objects; (2) induced in a location often spatially corresponding to the touch on the other's body; and (3) most commonly specular, like looking in a mirror. Only a few subjects reported an anatomical frame of reference, where, for instance, observed touch on someone's right thigh caused the sensation to occur on their own right thigh, implicating potential use of mental rotation tactics (see Parsons, 1987). This is in concordance with evidence of parietal visuo-tactile bimodal neurons in the monkey brain active for the self, responding to stimuli applied to the body part of an experimenter, and most commonly with a specular rather than anatomical frame of reference (Ishida et al., 2010). Finally, Banissy et al. (2009) also found that a large proportion of this group also reported other synaesthesia-like experiences, which is consistent with the finding that types of synaesthesia tend to co-occur (Simner et al., 2006).

Further exploration of the characteristics of mirror-touch synaesthesia suggest that top-down processes may modulate the intensity of the mirror-touch synaesthetic experience (Holle et al., 2011). In a recent study, mirror-touch synaesthetes observed videos of touch, and reported whether a synaesthetic touch experience was elicited, and its intensity. The authors found that the reported intensity of the synaesthetic response was strongest when observed touch was to real bodies. However, when touch was observed to dummy bodies, pictures of bodies, or disconnected bodies, the reported intensity of the induced sensation was significantly weaker. Although this is based purely on subjective report, it suggests that an entirely automatic account of mirror-sensory synaesthesia may be inappropriate.

As discussed earlier in this section, findings by Blakemore et al. (2005) suggest that the experience of mirror-touch synaesthesia activates similar areas of the brain as those involved in perceiving touch. If mirror-touch synaesthesia is induced through the same neural networks as experiencing touch in non-synaesthetes, it should be possible to induce synaesthetic touch-like experiences in non-synaesthetes through perceptual manipulation. Serino et al. (2008) investigated how perception of sub-threshold stimulation to the face was affected by simultaneously observing touch to one's own face, to another person's face, or to an inanimate object. The authors found that participants were better able to detect sub-threshold tactile stimuli on their own faces while observing another person's face being touched. This effect increased when

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observing one's own face compared to another's being touched, and was non-existent when observing an inanimate object. These results demonstrate that there is interaction between systems for perceiving and observing touch, and therefore may be seen as support for the touch mirror system. It seems reasonable to propose that these non-synaesthetic mechanisms also underpin the similar effects seen in mirror-touch synaesthesia.

A recent study by Banissy et al. (2011) provides further support for heightened sensorimotor mirror system activity in mirror-touch synaesthetes. In this study, the authors compared mirror-touch synaesthetes to non-synaesthetic control participants on measures of facial expression and identity recognition. Mirror-touch synaesthetes scored higher than controls on measures of facial expression recognition but not on identity recognition. Current understanding of these two processes suggest that to understand another's facial expression we simulate that same expression (e.g., Goldman and Sripada, 2005); in contrast a simulationist account for identity recognition is unlikely (Calder and Young, 2005). Accordingly, the findings of Banissy et al. (2011) suggest that mirror-touch synaesthetes have superior simulation mechanisms relative to people without mirror-touch synaesthesia. This finding suggests mirror-touch synaesthesia provides the benefit of increased perception of emotions in others. Moreover, such effects are consistent with the proposal of these individuals having an overactive touch mirror system.

In the first study to investigate mirror-touch synaesthesia in a clinical group, Ramachandran and Brang (2009) reported a mirror-touch-like response in four upper-limb amputees. In this study, the participants reported that the observation of another's hand being touched elicited the experience of being touched on the phantom hand. The authors suggest that this experience was induced by the loss of sensory input from the missing hand to indicate that the observed touch was not their own. This in turn resulted in the prevention of normal inhibitory processes of mirror system activity indicating that the observed touch was not their own. This is consistent with other reports of acquired synaesthesia following sensory loss (e.g., Armel and Ramachandran, 1999; Ro et al., 2007). However, this hypothesis cannot be extended to mirror-sensory synaesthetes who are not amputees (e.g. Bradshaw and Mattingley, 2001; Osborn and Derbyshire, 2010), nor does it account for mirror-sensory synaesthetic experiences not triggered by observing touch in the same limb as that amputated (Giummarra and Bradshaw, 2008), or when mirror- pain is not felt in the phantom (e.g., Fitzgibbon et al., 2010a).

Most recently, about a third of a sample of amputees reported experiencing a tactile sensation in their phantom limb when observing another being touched (Goller et al., in press). These experiences had a common pattern: they were most often reported in the phantom limb or stump, and increased in intensity when the observed touch was more painful than non-painful and seen on a real person, as opposed to a dummy or object. It was also found that these touch 'responders' scored higher than non-responders on an empathy measure of emotional reactivity. Perhaps suggesting why some amputees may be more susceptible in developing mirror-touch synaesthesia following amputation than others.

3.2. Mirror-pain synaesthesia

Like touch, seeing pain activates more than just the visual parts of the brain. Observing pain in another activates similar areas of the brain as if the observer is experiencing pain, comprising what is known as the mirror system for pain (Jackson et al., 2006). Pain describes the perception of nociception: actual tissue damage (Van Damme et al., 2010). Like touch, there have been reports that people have experienced pain even in the absence of noxious stimulation. For example, a builder presented to an emergency department in severe pain after having a 15 cm nail pass through his shoe. However, on removal of the shoe, it was found that his foot was not injured and that the nail had passed between his toes (Fisher et al., 1995). Further, some patients experience painful flashbacks where pain is re-experienced; for example, following the 2005 London bombings (Whalley et al., 2007), or incomplete anaesthesia during surgery (Salomons et al., 2004). Another example is in a normal population where pain has been hypnotically induced (Derbyshire et al., 2004). There have even been reports where events not normally considered noxious, for example; social exclusion, have elicited a functional response similar to physical pain (Eisenberger et al., 2003). It is not surprising, therefore, that for some people the observation of pain in another can result in an actual experience of pain. This has been reported in mirror-pain synaesthesia following pain-related trauma (Fitzgibbon et al., 2010a), and seemingly from birth (Osborn and Derbyshire, 2010).

The first clear report of a relationship between observed and 'real' pain was anecdotally described in a man with hyperalgesia, a heightened sensitivity to pain, who felt pain when he observed his wife in pain (Bradshaw and Mattingley, 2001). For example, his wife reported that even a slight knock of her finger would cause her husband distress, as he would feel actual pain in his own finger. In another report following a painful labour, a woman reported experiencing shooting pain radiating from her groin down her legs whenever told of another person's painful experience (Giummarra and Bradshaw, 2008). Following the birth of her second child through caesarean section delivery, this experience has worsened with an increase in frequency and lower-level stimulation required to induce the experience (Giummarra and Bradshaw, personal communication).

The most common reports of mirror-pain synaesthesia have, however, been in amputee populations. Indeed, even before the identification of mirror-pain synaesthesia, there had been reports of phantom limb pain being triggered by seeing others in pain in horror films or when witnessing an accident (e.g. Katz, 1992; Wilkins et al., 2004). Giummarra et al. (2008) reported eight cases of lower-limb phantom-limb patients reported that their phantom pain is triggered by observing, thinking about, or inferring that another person is in pain. Examples of these accounts include an amputee who felt strong, painful 'electric' impulses in his phantom foot when he observed sutured wounds, another who felt pain when he observed someone cut or hurt themselves, and another who reported their phantom foot 'going crazy' when he heard a gruesome story. Finally, one amputee experienced mirror-pain synaesthesia when he observed experiences he personally associated with pain, such as walking barefoot. Mirror-pain synaesthesia has also been reported in one upper limb amputee where phantom limb pain was experienced when watching footage of amputations on television, others being injured on their arms, or when stimuli associated with potential pain/amputation (e.g., axe, chainsaw, sharp knife) were near her own arm, or near another's limbs (Giummarra et al., 2008).

In the first preliminary investigation to determine the incidence and frequency of mirror-pain synaesthesia in an amputee population, we found that 16.2% of all participants reported that observing or imagining pain in another triggers their phantom pain (Fitzgibbon et al., 2010a). We also found that mirror-pain synaesthesia was more likely to be experienced in the phantom limb, and to be triggered (a) by observation of another in pain rather than imagining another in pain; (b) by any type of pain rather than specific types of pain; and (c) in response to any person in pain rather than, for example, just loved ones. Our findings suggest that the experience of mirror-pain synaesthesia is not uncommon among amputees. Similar to mirror-touch synaesthesia (see Banissy et al., 2009), this number may be reduced when controlling for false positives, perhaps through systematic investigation of behavioural

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response to pain images (see future research for a discussion). Alternatively, this high percentage may reflect the specific group of interest (amputees), as mirror-pain synaesthesia may be more common in trauma/chronic pain groups, or in cases of sensory loss, than in the general population.

Our group has also found that mirror-pain synaesthesia in amputees is not just sensory in nature but has an associated motor response (Giummarra et al., 2010). In this study we used a modification of the rubber limb illusion where manipulation of vision and touch can cause healthy populations to embody a rubber limb (see Botvinick and Cohen, 1998). We found that lower-limb amputees who experience mirror-pain synaesthesia reported an induced pain and/or a motor response (e.g., avoidance, contraction and withdrawal) in the phantom leg when the embodied hand was threatened (e.g., with a syringe, mousetrap, retractable knife) (Giummarra et al., 2010). These induced experiences may come about through hypervigilance for harm, which results in a 'flight' response – alternatively (or in combination), the motor system is activated during the illusion, and may prime synaesthetic reactions. Finally, the experience may have been painful as there was no feedback from the missing limb to indicate that the participant's motor response was carried out. We will discuss potential mechanisms for this sensitivity in Section 5.

The first documentation of what we describe as mirror-pain synaesthesia in the general population was recently reported by Osborn and Derbyshire (2010). In this study, the authors presented a series of still and video images depicting pain to a group of undergraduate students. Approximately one third of this group, 'pain responders', reported feeling pain in response to these images. Ten of these pain responders and 10 matched non-responders (people who did not experience a pain sensation in response to the images) then underwent fMRI while observing still pain images, and emotional images that score comparably to the pain images for unpleasantness, fear, disgust, and sadness. When viewing pain images compared to emotional images, the pain responders activated both emotional and sensory areas involved in pain processing, whereas there was little difference between the image sets in the non-responder group.

Osborn and Derbyshire (2010) did not find evidence of a motor component, as proposed to be involved in amputees (see Giummarra et al., 2010). They did not find any pain-related motor responses in the participants (e.g., grimacing), nor was there much evidence for motor involvement in their fMRI results. The authors also did not find any significant correlation between pain intensity ratings of perceived pain and an empathy measure. They did, however, find that pain responders scored higher than controls on a measure of empathy. This is consistent with the possibility that individuals who have an induced somatic sensation as a result of observing touch or pain in another may have elevated empathic traits (see Banissy and Ward, 2007). Limitations of this study include the lack of an objective measure of the induced pain, and the variability of the stimuli; sometimes there was just one person or set of limbs per image, and others included more than one; sometimes facial expression was visible; and finally differences in ethnicity of the individual(s) in the images which is known to affect pain processing. For example, in one study, no sensorimotor resonance was seen when participants observed the pain of racial out-group members compared to a stranger of the same race (Avenanti et al., 2010). Despite these limitations, this study suggests that mirror-pain synaesthesia can be identified in an undergraduate population, and that people who report mirror-pain synaesthesia have higher activation in widespread areas of pain-related neural regions when observing pain compared to those who do not.

In our most recent study, we used EEG to investigate the neurophysiological response to pain-related images of amputees who experience mirror-pain synaesthesia compared with controls (Fitzgibbon et al., in press). Neural activity was recorded as participants observed still images of hands and feet in potentially painful (e.g., a knife placed over a hand) and non-painful (e.g., a hand and a knife side by side) situations. Participants were asked to either rate the intensity of the pain observed if it was real or to state if there was a hand or a foot in the image. We found that the pain synaesthete group had significantly reduced ERP response relative to controls in response to both pain and non-pain images, regardless of task demands. We interpret these results as indicating an inhibition of the processing of observed pain (e.g., avoidance/guarding as a protective strategy). As this was observed in response to both conditions and tasks, mirror-pain synaesthetes may be anticipating pain due to hypervigilance to pain cues. For instance, it is possible that the non-painful images elicited neural changes associated with anticipating pain as both a limb in additional to a potentially threatening tool are present. This is supported by evidence that suggests the anticipation of somatosensation can increase activation in the primary somatosensory cortex without actual stimulation (see Carlsson et al., 2000).

We also analysed band power (ongoing neural activity). We found that the pain synaesthete group had reduced theta and alpha band power at a central electrode compared with controls (Fitzgibbon et al., in press). As theta is known to increase during cognitive demand (Klimesch, 1999), we interpret this reduction in theta as a reflection of similar inhibitory mechanisms to those seen in the ERP results. Alpha activity, comparatively, is thought to decrease as cognitive load increases (Klimesch, 1999), suggesting that our observed reduction in alpha may indicate a disinhibition in control processes that results in mirror-pain synaesthesia. Finally, we found no differences between pain intensity ratings of images, or empathy measures between groups. Together with the findings of Osborn and Derbyshire (2010), this suggests that it is unlikely that personal dispositions such as empathy are the sole cause of mirror-pain synaesthesia. Alternatively, if increased empathy is a benefit of mirror-sensory synaesthesia, it is not consistent across all individuals, or developmental versus acquired mirror-sensory synaesthetes.

4. Mirror-sensory synaesthesia as a unique type of synaesthesia

There are similarities between mirror-sensory synaesthesia described here and other forms of synaesthesia. We argue that the experiences induced in mirror-sensory synaesthesia are, by definition, truly synaesthetic as they are an unusual perceptual experience occurring in response to observing sensory stimulation in another. However, there are also differences that make mirror-sensory synaesthesia distinct to other forms of synaesthesia and link mirror-sensory synaesthesia to normal social perception. Here we provide an overview on the shared and unique features of mirror-sensory synaesthesia compared with more typical forms of synaesthesia and everyday perception.

A defining feature of synaesthesia is that the induced perceptual experience is anomalous, or uncommon within the general population, and occurs outside of a psychiatric or neurological disorder (Ward and Mattingley, 2006). Mirror-touch synaesthesia is not common and is experienced by only 1.6% of the general population (Banissy et al., 2009). Mirror-pain synaesthesia, however, may be experienced by up to 30% of the general population (Osborn and Derbyshire, 2010), and 16.2% of amputees (Fitzgibbon et al., 2010a). However, unlike mirror-touch synaesthesia, studies on mirror-pain synaesthesia are yet to test for false-positives. Mirror-sensory synaesthesias have also not been reported in relation to pathology or psychiatric/neurological disorder, although amputees reporting mirror-pain synaesthesia have a significant medical

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history. Mirror-pain synaesthesia following amputation is similar to other forms of acquired synaesthesia following, for example, brain injury (e.g., Ro et al., 2007; Vike et al., 1984).

Synaesthesia is involuntary in response to certain sensory inputs (Ward and Mattingley, 2006), for example, certain letters may elicit specific shades of green. Similarly, in mirror-sensory synaesthetes, observing touch or pain in another person involuntarily triggers the perceptual experience of touch or pain in the self. This experience is also specific to the individual, where it appears that only certain touch or levels of pain in another triggers the synaesthetic experience. Grapheme-colour synaesthetic experiences can be experienced even by simply thinking of the inducers (see Dixon et al., 2000; Grossenbacher and Lovelace, 2001). This also occurs in mirror-sensory synaesthesia where it has been reported that mirror-pain synaesthesia can occur when imagining another person in pain (Fitzgibbon et al., 2010a). Finally, synaesthetic experiences are unidirectional, in that the inducer causes the concurrent experience and not vice versa (Ward and Mattingley, 2006). Analogously, mirror-sensory synaesthesia is also unidirectional; experiencing touch or pain to the self does not induce visions of touch or pain in another or even synaesthetic referral of pain to the phantom in amputees.

The induced experience of mirror-touch synaesthesia is also comparable to another perceptual experience (i.e., actual touch or pain). This means that, like other synaesthetic experiences, the perceptual experience itself is not intrinsically abnormal or unlikely. For example, colours or tastes are otherwise normally encountered, but their synaesthetic expression is out of context. As synaesthetic experiences are everyday perceptual experiences, it is not surprising that they recruit the same areas of the brain active as in normal perception (for a discussion, see Spector and Maurer, 2009). For example, the experience of synaesthetic colour activates areas responsive to colour, such as V4 in some synaesthetes (e.g. Hubbard et al., 2005; Nunn et al., 2002) or other visual areas (Rich et al., 2006); although other studies have not found colour areas active in synaesthesia (e.g., Hupe et al., in press; Paulesu et al., 1995; Rouw and Scholte, 2010). Similarly, mirror-sensory synaesthesia appears to activate the same areas of the brain as in normal touch or pain perception (Osborn and Derbyshire, 2010).

The fact that mirror-sensory synaesthesia is so similar to normal perception has led some to imply that a somatic experience in response to the perception of another's experience may be a relatively normal experience (Keysers et al., 2010). This raises the possibility that mirror-sensory synaesthesia may be a spectral phenomena ranging from understanding the somatic sensation of another, perhaps involving an aspect of emotional distress, to the actual experience of a similar sensation (Keysers et al., 2010). This would suggest that the reports of mirror-sensory synaesthesia represent only the extreme cases of normal perception where conscious experiences occur. This is not the first report of an extreme version of a normal process; music-colour synaesthetes and non-synaesthetes alike pair high-pitched sounds with light/bright colours. Despite this likeness, non-synaesthetes do not experience the colour, but are rather making colour associations based on implicit mappings (Ward et al., 2006). Indeed, systematic patterns in auditory-visual synaesthetes match those of non-synaesthetes for colour, shape, and location (Chiou and Rich, submitted). Perhaps other synaesthetic experiences exist on a spectrum and the field of synaesthesia research is biased towards the study of extreme cases defined by their consistency thereby excluding other cases and preventing full understanding of the phenomenon (Simner, 2010). Nevertheless, conscious somatic experience triggered by observed somatic experience in another is yet to be reported universally or even as a common occurrence.

The primary unique feature of mirror-sensory synaesthesia, setting it apart from other synaesthesias, is that the relationship between the inducer and concurrent sensation involves a similar sensory experience to that observed in another person. For example, in mirror-pain synaesthesia the inducer is the observation of pain in another, which results in the experience of mirror-pain synaesthesia (concurrent). In more well-studied synaesthesias, such as grapheme-colour, at first glance the inducer and concurrent appear unrelated (although some non-random trends have been observed, see Rich et al., 2005; Simner et al., 2005), as indeed is also the case with pain-related synaesthesias that are not mirror-sensory in nature (e.g., where an individual's experience of pain elicits a synaesthetic response of colour, taste, or smell (Dudycha and Dudycha, 1935; Ward, 2008)). It should be noted, however, that there are other reports of non-random and seemingly obvious synaesthetic experiences in other forms of synaesthesias. For example, the word 'rhubarb' may elicit the taste of rhubarb (Ward and Simner, 2003), words may elicit a taste that actually sounds similar, e.g. 'cinema' eliciting the taste of cinnamon rolls (Ward et al., 2005), and the word 'red' producing the colour red (Rich et al., 2005). Nevertheless, this differs from mirror-sensory, in which another person's sensory experience induces a similar sensory experience in the synaesthete. We do not see cases where people report that seeing another person listening to music elicits the feeling of listening to music (although theoretically it is possible!).

A further key difference from other types of synaesthesia is the potential for mirror-sensory synaesthesia to be maladaptive and hugely distressing, especially for mirror-pain synaesthetes. This is different to the majority of synaesthesias where detrimental effects to the individual are not observed (see Baron-Cohen, 1996) and the individual does not realise it is unusual. In most types of synaesthesia, the experience can go unnoticed as abnormal, as it is often developmental and therefore very rarely experienced as disruptive. Comparatively, some of our mirror-pain synaesthetes describe social withdrawal, including fear of watching television and/or movies, particularly the news, or anything horror- or action-related. This is similar to the disabling and not dissimilar disorder of imitation behaviour, where individuals automatically imitate actions and/or gestures they observe in another person (De Renzi et al., 1996; Lhermitte et al., 1986). Mirror-sensory synaesthesia however, may also be accompanied by certain beneficial abilities, such as mirror-touch synaesthetes having better facial expression recognition than non-synaesthetes (Banissy et al., 2011). This is similar to a few reported cases where synaesthesia has been related to improved cognitive ability and memory (e.g., Luria, 1968; Smilek et al., 2002).

Here, we have presented how mirror-sensory synaesthesia is similar to other synaesthetic variants, including in that it may be based on normal mechanisms. We also identified the obvious relationship between inducer and concurrent experience in mirror-sensory synaesthesia, where a person experiences a similar sensory experience to that observed in another. We proposed that mirror-sensory synaesthesia be considered as a synaesthesia, and that its unique qualities are used in the development of appropriate neurobiological models for this intriguing phenomenon.

5. A neurobiological basis of mirror-sensory synaesthesia

Thus far in this paper, we have described mirror-sensory synaesthesia and given a justification for it to be considered synaesthesia. Nevertheless, we also identified its distinct quality separating it from other synaesthesia: the inducer is the perception of a sensory experience in another person, and the concurrent is a similar experience to that observed. We now turn to potential neurobiological mechanisms of mirror-sensory synaesthesia by exploring

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how a neurobiological model of social cognition combined with neurobiological mechanisms proposed for synaesthesia may produce mirror-sensory synaesthesia.

As discussed earlier, mirror-like activity has been documented in domains other than action and in areas outside the classical mirror neuron regions (for a review, see Keysers and Gazzola, 2009). It is unknown whether these "systems" make up a specific network or reflect independent brain areas with mirror properties that are linked via numerous neural circuits. Mirror-sensory synaesthesia may involve somatosensory mirror systems. Somatosensory systems describe the brain regions, including the somatosensory cortices, the insula and the rostral congulate cortex that process somatosensation: the processing of sensory events to the body including tactile and nociceptive information (for a review of somatosensation in social perception, see Keysers et al., 2010). If mirror-sensory synaesthesia is mediated by an increase of functioning within the same regions of the brain that allow us to experience touch or pain, it may be best to consider mirror-sensory synaesthesia as a dimensional and not binary phenomenon. This does not discount actual somatic experience when observing touch or pain in another as abnormal, but rather characterises it as a conscious experience similar to another's state which is at the extreme end of the spectrum. In the case of mirror-sensory synaesthesia, these mirror systems are active above the threshold for conscious perception, such that observing touch or pain results in the experience of touch or pain. In the following section, the possibility that hyperactivity in somatosensory mirror systems is the primary mechanism involved in mirror-sensory synaesthesia, perhaps resulting from similar processes implicated in other forms of synaesthesia, will be addressed. Enhanced empathy will also be identified as an alternate mechanism for mirror-sensory synaesthesia, as well as the possibility that mirror systems are a by-product of learned association.

5.1. Hyperactivity in somatosensory mirror systems

The experience of mirror-sensory synaesthesia may be related to heightened excitability within somatosensory mirror systems, active both when observing another person's sensory state and experiencing that same state oneself. Although mirror systems have not previously been implicated in other forms of synaesthesia, they may play an important role for mirror-sensory synaesthesia. Two potential mechanisms that may affect mirror system activity are: atypical *structural* connectivity (e.g., increased neural connections) or *functional* disinhibition of otherwise normal connections (e.g., changes in neurotransmitter activity) between areas of the brain involved in vision and somatosensation (see Fig. 1). Both of these accounts have already been proposed for other forms of synaesthesia (e.g. Bargary and Mitchell, 2008).

Atypical neural connectivity is one mechanism that may cause hyper-connectivity between areas involved in vision and somatosensation and consequently the mirror-sensory synaesthesia. Such atypical neural connectivity, resulting from reduced pruning of synapses in early development, has been proposed as a potential mechanism of synaesthesia in general (Maurer, 1997). It is possible that atypical connectivity may be involved in developmental mirror-sensory synaesthesia, in which there is no identifiable cause or onset of the phenomenon. Atypical connectivity may also be involved in acquired mirror-sensory synaesthesia in amputees as a result of cortical reorganisation following amputation. Indeed, acquired synaesthesia is often reported following sensory loss (e.g., Armel and Ramachandran, 1999; Jacobs and Donoghue, 1991; Ro et al., 2007), suggesting that the unmasking or development of synaptic connections between visual and somatosensory may be implicated in acquired mirror-sensory synaesthesia (Goller et al., in press). However, cortical reorganisation cannot explain mirror-sensory synaesthesia where cortical reorganisation is not expected, or when tactile experiences in non-synaesthetes are modulated through manipulation of perceptual thresholds in non-synaesthetes (see Serino et al., 2008). This suggests that hyper-connectivity is not a pre-requisite for mirror-sensory synaesthesia.

An alternate mechanism is that mirror-sensory synaesthesia may result from hyperactivity of otherwise normal brain areas for touch or pain. Typically, activation in mirror areas is greater when one experiences a sensation, or an emotion, or carries out an action compared to when the same experience is observed in another. This is thought to reflect inhibitory processes involved in the mirror system that prevent one from experiencing or imitating the observed sensation/emotion or action (e.g. Kraskov et al., 2009). In fact, reduced inhibitory control in relevant mirror systems has been implicated in imitation behaviour (Archibald et al., 2001). An absence of, or reduction in, normal inhibitory mechanisms within these mirror systems could reasonably lead to the *experience* of that touch or pain–mirror-sensory synaesthesia.

Disinhibition of otherwise normal connections is supported by research investigating mirror-touch and mirror-pain synaesthesia reviewed earlier that implicate greater vicarious activation in somatosensory brain regions compared to controls (Blakemore et al., 2005; Osborn and Derbyshire, 2010). To recap, in the first study of mirror-touch synaesthesia, Blakemore et al. (2005) used fMRI to map brain activity underlying both non-synaesthetic and synaesthetic perception of touch. Observing touch activated the tactile mirror system in both synaesthetes and non-synaesthetes; notably, however, activation was greater in the case of synaesthetes. In the first study of mirror-pain synaesthesia, SI and SII activation was only observed in participants who reported feeling pain, 'responders', as well as greater activation in other areas such as the insula when observing injury to another compared to those who did not report pain (Osborn and Derbyshire, 2010). In another study, non-mirror-touch synaethetes showed subtle effects on tactile perceptual thresholds from observing touch at the same time as experiencing touch (Serino et al., 2008). These data, and the other studies reviewed above, suggest that although normally we do not experience touch or pain when these experiences are seen in another, the mechanisms are present that connect these two instances. This idea is consistent with proposals for other forms of synaesthesia (Ward et al., 2006). It therefore seems plausible that, in mirror-sensory synaesthesia, disinhibition or overactivity of the mirror systems could result in the conscious perception of touch or pain.

Functional changes in mirror-sensory synaesthesia may be explained in two ways (see Fig. 1). First, if the mirror-sensory synaesthesia is developmental, then the altered function may be the result of atypical development, as suggested in individuals with autistic spectrum disorders (Williams et al., 2001), or as occurring naturally, perhaps through a genetic predisposition, as is implicated in other forms of synaesthesia (Baron-Cohen et al., 1996). Second, in the case of acquired mirror-sensory synaesthesia, normal mirror system processing has somehow become disinhibited. As the onset is later in life, we propose such a change requires some event or circumstance. One commonality in people who report acquired mirror-sensory synaesthesias is that it usually occurs following intense, traumatic, or chronic pain. One mechanism by which this might occur is that such prior pain causes hyperactivity of the somatosensation mirror system, possibly as a by-product of hypervigilance to pain cues (Fitzgibbon et al., 2010b). This increase in activity then results in the failure to prevent evocation of a similar sensory sensation on perceiving touch or pain. The idea that mirror systems can be affected by experience is consistent with evidence that mirror systems can be modulated by sensorimotor learning (Catmur et al., 2007).

The involvement of somatosensory mirror areas in mirror-sensory synaesthesia is supported by TMS studies that have

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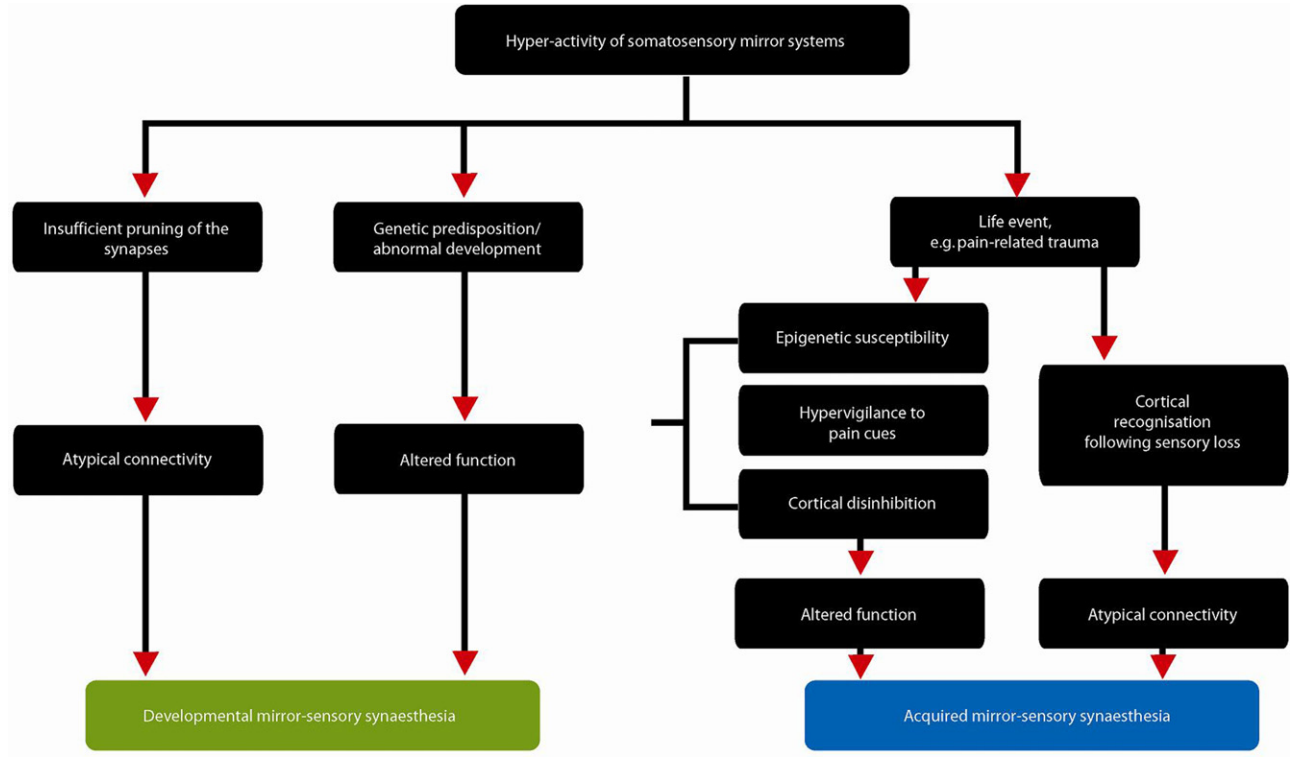

Fig. 1. Schematic depicting possible ways atypical connectivity or altered function may produce hyperactivity of the somatosensory mirror systems that may result in developmental and acquired forms of mirror-sensory synaesthesia.

demonstrated modulation of sensorimotor empathy in response to the observation of pain and touch in others in healthy controls (Avenanti et al., 2005; Bufalari et al., 2007), individuals with higher scores of psychopathic traits (Fecteau et al., 2008), and individuals with Asperger's disorder (Minio-Paluello et al., 2009). In healthy controls, somatosensory evoked potentials thought to reflect activity in the somatosensory cortex were shown to be modulated by observing pain and touch in another (Bufalari et al., 2007). Observation of pain-related stimuli has also been shown to cause a reduction in motor-evoked potentials, which was negatively correlated with sensory ratings of another's pain (Avenanti et al., 2005). This inhibitory effect, and its relationship with sensory ratings, was not observed in a study of participants with Asperger's disorder, who have impairments in empathy (Minio-Paluello et al., 2009). Finally, in another study using healthy controls, participants who scored highest on a cold-heartedness subscale of a psychopathic trait measure showed the greatest inhibition (Fecteau et al., 2008). Together, these findings indicate that the somatosensory system is likely to be involved in providing information about another's sensory state.

There may be a genetic component involved in the proposed hyperactivity of somatosensory mirror systems in mirror-sensory synaesthesia. A recent fMRI study documented reduced neural activation in higher-order pain centres in $\alpha 2\alpha 3$ mutant mice when experiencing thermal pain (Neely et al., 2010). In contrast, in areas involved in vision, hearing and olfaction, the mutant mice displayed increased activation compared to control mice in response to touch or pain stimulation. These findings provide the first evidence of a specific gene involved in sensory cross-activation. Predisposition to mirror-sensory synaesthesia may also correspond to epigenetic factors (for a discussion, see Zhang and Meaney, 2010) that predispose one to have heightened sensitivity to stress/pain/threat, potentially even from birth. For example, offspring of holocaust survivors (Yehuda et al., 2002) or women pregnant during the USA 9/11 World Trade Centre attacks (Yehuda et al., 2005) have been found to have lower cortisol levels that may cause an increased propensity to post-traumatic stress disorder. Such epigenetic mechanisms may also be why some people experience mirror-sensory synaesthesia following trauma and others do not, even after similar traumatic experiences. Both the use of animal models and investigation of pain history in first-degree relatives in mirror-sensory synaesthetes may be valuable in determining the involvement of genetic mechanisms in mirror-sensory synaesthesia.

5.2. *Mirror-sensory synaesthesia: Result of enhanced empathic capacity?*

It is currently unknown why some people experience mirror-sensory synaesthesia developmentally, and why some (but not all) people acquire the experience following trauma. In the previous section, we discussed potential neurobiological mechanisms for mirror-sensory synaesthesia; however, certain psychological aspects, or personal traits such as empathy may also be involved. Moreover, mirror system activity may be dependent on and modulated by cognition (Keysers and Gazzola, 2009). Empathy, for example, is implicated in how we understand and perceive sensation in others (Bufalari et al., 2007; Jackson et al., 2005) and is modulated by multiple factors including cognitive appraisal and perspective taking (for a review, see Hein and Singer, 2008). Empathy refers to the capacity to understand another person's state in the context of the self (de Vignemont and Singer, 2006; Decety and Jackson, 2004). Empathy does not seem relevant for most forms of synaesthesia. For example, experiencing specific tastes in response to words (e.g. Ward and Simner, 2003) has nothing to do with understanding another person's state. In the case of mirror-sensory synaesthesia however, there is a direct relationship between the experience of one person and a similar experience in the mirror-sensory synaesthete. This relationship has significant implications for synaesthesia in general, as it links synaesthesia with universal

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cross modal associations, and not processes unique to synaesthetes (e.g., atypical connectivity).

There is some evidence that empathy may be involved in mirror-sensory synaesthesia. Mirror-touch synaesthetes, both developmental and acquired, score significantly higher on the emotional reactivity index of the empathy quotient measure than non-synaesthetes (Banissy and Ward, 2007; Goller et al., in press). In another study, participants who reported experiencing pain in response to pain-related images scored higher on a measure of state empathy than non-responders (Osborn and Derbyshire, 2010). Most recently, mirror-touch synaesthetes scored higher than controls on measures of facial expression recognition (Banissy et al., 2011). In contrast, in two recent studies from our group, we found no difference between amputees with mirror-pain synaesthesia compared with amputees without mirror-pain synaesthesia, and non-amputee controls in measures of empathy (Fitzgibbon et al., in press; Giummarra et al., 2010).

Although there is currently limited evidence, should generalised empathic ability (measured by behavioural empathy measures) underlie mirror-sensory synaesthesia then we would expect that mirror-sensory synaesthetes would experience a wide range of observed phenomena in addition to touch and pain. We argue that the absence of reports that mirror-sensory synaesthetes experience any other sensory or emotional state observed in other people indicates that increased empathy is not the driving mechanism of mirror-sensory synaesthesia. Instead, perhaps, the experience of mirror-sensory synaesthesia may cause greater empathy in some people. It is possible, however, that mirror-sensory synaesthetes experience other mirrored states, and this has not been reported due to lack of enquiry. Future research will need to address the potential relationship between empathy and mirror-sensory synaesthesia, as well as synaesthesia more broadly. A key consideration, however, will be the inconsistency of findings relating behavioural empathy scores with neural activity; for example, although some studies of healthy populations have demonstrated a relationship between empathy scores and cerebral response in healthy controls (Avenanti et al., 2009; Cheng et al., 2008; Loggia et al., 2008; Singer et al., 2004), others have failed to replicate such findings (Avenanti et al., 2005; Jackson et al., 2005; Lamm et al., 2007).

5.3. Mirror-sensory synaesthesia: Learned association?

It is thought that mirror systems may have evolved as an adaptation for interpersonal understanding (Gallese and Goldman, 1998; Iacoboni et al., 1999; Rizzolatti et al., 2001). Although mirror systems may contribute to social cognitive function, they may be secondary to associated processes that facilitate understanding. In fact, it has been proposed that the mirror neuron system is a by-product of Hebbian association (Heyes, 2010; Keysers and Gazzola, 2009). According to this view, the pairing of the visual of somatic experience and the somatic sensation, such as observing being touched and the associated tactile sensation of touch, may be enough to activate somatosensory mirror areas when seeing another experience a somatic sensation. The theory of learned association is supported by studies demonstrating the effects of sensorimotor modulation of mirror areas. For example, mirror associations occur between the sound of a piano and premotor areas within just a few hours (Lahav et al., 2007). Similarly, ballet dancers demonstrate increased mirror activity when observing ballet movements compared to dancers of another speciality (Calvo-Merino et al., 2005). Finally, expected effects of the mirror system activation can be reversed through training, as demonstrated by a TMS investigation where muscle response congruent to observed movements can be modulated following training of an incongruent muscle response (Catmur et al., 2007). Accordingly, it has been suggested that mirror neurons did not evolve as an adaptation for a specific function but may just be a by-product of association that, in some cases, has favourable benefits (Heyes, 2010). If Hebbian learning is involved in the development of mirror systems, then mirror-sensory synaesthesia would be the result of a learned association through sensorimotor experience mediated by mirror systems. This is clearly not incompatible with our suggestion that mirror systems are hyperactive in mirror-sensory synaesthesia resulting in an actual somatic sensation. Therefore, regardless of how these mirror systems arise, there is reason to investigate their potential role in mirror-sensory synaesthesia.

6. Future research

Research into mirror-sensory synaesthesia is relatively new and many questions remain unanswered. Of primary importance is: (1) determining why some individuals acquire mirror-sensory synaesthesia following pain-related trauma, but others do not even when clinically identical; and (2) the development of a tool to validate individual experience of mirror-sensory synaesthesia to reduce inclusion of any false positives. Objective tests of mirror-sensory synaesthetic experience will need to distinguish between people empathising with others, and people actually experiencing touch or pain. This may be a difficult task for mirror-pain syneasthetes: the method to test for false positives in mirror-touch synaesthetes involves a vision-touch congruency experiment, where mirror-touch synaesthetes make more errors than non-synaesthetes when touch applied to their own face is incongruent with touch they simultaneously observe to another face (see Banissy and Ward, 2007). The development of an analogous vision-pain congruency experiment would involve inducing pain, which is ethically fraught.

It is also important that mirror systems (and Hebbian learning) and their potential role in producing mirror-sensory experiences are explored further. Currently, it is not entirely clear which brain areas constitute the mirror system for touch and/or pain. For example, secondary somatosensory cortex activation in response to observed touch has been noted when the observed touch is to the legs (Keysers et al., 2004), yet primary somatosensory cortex activation has been reported when the observed touch is to the arm (McCabe et al., 2008), or face (Blakemore et al., 2005). It has also been found that neural activation can be modulated by manipulating the description of the observed touch, for example, 'rich moisturising cream' or 'basic cream' (McCabe et al., 2008), or the intentionality of the observed touch (Ebisch et al., 2008). Similar inconsistencies are also seen in the mirror system for pain. For example, some studies suggest that affective areas involved in pain processing are primarily involved in processing observed pain (e.g. Singer et al., 2004), whereas other studies suggest that sensory areas are also active (e.g. Avenanti et al., 2005). Further, such activation may also be influenced by cognitive manipulations (e.g. Lamm et al., 2007). Taken together, these studies highlight that somatosensory mirror systems are highly malleable, and susceptible to factors such as passive versus active observation, description of the stimuli, intensity of the stimuli, intentionality, experience, perspective taking, and attention (Avenanti et al., 2006; Minio-Paluello et al., 2006). For a full understanding of the mechanisms underlying mirror-sensory synaesthesia, more research is needed in the involvement of mirror systems for touch and pain.

7. Conclusions

In this review, we have operationalised mirror-sensory synaesthesia; an intriguing experience where the observation or imagination of somatosensory stimulation in another (e.g., touch or pain) induces a similar somatosensory experience in oneself.

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Mirror-sensory synaesthesia may be an extreme version of a normal process, classified as a synaesthesia when conscious experiences occur. In mirror-sensory synaesthesia, there is a non-arbitrary relationship between the inducer and concurrent: the inducer is the perception of a sensory experience in another person, and the concurrent experience is similar to that observed. It is important that unique features of mirror-sensory synaesthesia be identified, and that sub-groups with similar experiences are documented and recognised. This will lead to a better understanding of the neurobiological mechanisms that produce these experiences. Finally, in this review it is proposed that mirror-sensory synaesthesia may emerge through hyperactivity in somatosensory mirror systems mediated by similar mechanisms proposed in synaesthesia. We hope that by operationalising mirror-sensory synaesthesia the scientific community may be better placed to uncover the underlying mechanism(s) of this experience.

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