



Capgras Syndrome: a disorder of kin recognition

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Capgras Syndrome is a psychiatric condition characterized by the delusional conviction that someone well known to the patient, usually a close relative, has been replaced by an imposter. The prevailing hypothesis, advanced in recent decades by V. S. Ramachandran and Max Coltheart amongst others, is that two causative factors combine to give rise to this peculiar belief: the first factor is an impaired grasp of reality associated with right frontal lobe damage; the second factor, more specific to Capgras Syndrome, is the absence of the normal autonomic (affective/emotional) response one would expect to have when encountering familiar others. It has been claimed that the high incidence of close relatives amongst the misidentified is because we normally have our strongest emotional reactions to family members, and consequently the discrepancy presented by the absence of these responses in Capgras Syndrome is powerful enough to trigger the delusion. In this paper, I set forward an alternative hypothesis: that the tendency to target relatives is evidence of a cognitive module facilitating recognition of kin, and that damage or disruption to the underlying neural network is the second causal factor in Capgras Syndrome. I will start by briefly summarizing existing views and opinions of Capgras Syndrome as being (a) one of several archetypal monothematic delusions; (b) a right hemisphere disorder; (c) an impairment of face recognition; (d) a failure of identity perception and (e) a multimodal “neuropathology of the self”. I subsequently advance a novel hypothesis that Capgras Syndrome is indicative of damage to a specialized kin recognition module.

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INTRODUCTION

Capgras Syndrome is a psychotic condition characterized by the delusion that someone has been replaced by an imposter. It is named after an early twentieth century French psychiatrist, Joseph Capgras, who himself referred to the condition as “*l’illusion de sosies*”, i.e. the illusion of doubles (Capgras and Reboul-Lachaux 1923). Although cases involving ‘doppelgänger’ doctors, household staff, neighbors, pets and inanimate belongings have all been reported, typically the targets of the delusion are close relatives of the subject (Ellis, Luauté and Retterstøl 1994, Wallis 1986). Some of the earliest attempts to explain the condition sought psychodynamic causes in the social and emotional tensions experienced by the subject in their formative years; by contrast, since the 1970s, there has been greater interest in identifying the physical basis of the disorder (Berson 1983, de Pauw 1994, Edelstyn and Oyeboode 1999). A third of reported cases are accompanied by some form of cerebral damage (Ellis and de Pauw 1994, Signer 1987, 1994).

The dominant theory of the condition today, expounded by Ramachandran and Coltheart (amongst others), is that Capgras Syndrome is caused by disruption to the neural systems responsible for attaching the ‘correct’ emotive/affective valence to a familiar person or face. When encountering someone fails to provoke the anticipated feeling of closeness, the patient subconsciously resolves the disparity by concluding that they must actually be someone else - an imposter (Breen, Caine and Coltheart 2001, Coltheart 2007, Davies, Coltheart and Langdon, et al. 2001, Devinsky 2009, Ellis and Young 1990). The tendency of the delusion to target close relatives has been explained on the grounds that “only with one’s parents or spouse does one expect a glow of arousal” (Hirstein and Ramachandran 1997) and “it is only when this discrepancy is very large that the stranger/impostor hypothesis is generated” (Coltheart, Langdon and McKay 2011). The misrecognition of kin is

commonly presented as being an incidental and secondary outcome of a more fundamental causal factor, namely the lack of affective response to familiar people in general. As you will have read in the abstract, I am proposing that the targeting of relatives seen in Capgras Syndrome is not a secondary symptom but rather a primary sign, and *is indicative of damage to a neural module responsible the recognition of kin.*

As background, I will briefly introduce five aspects of Capgras Syndrome that have been highlighted in the scientific literature, before turning to the kin recognition hypothesis. The five themes are that Capgras Syndrome is (a) a two-factor monothematic delusion; (b) symptomatic of right hemisphere dysfunction; (c) a disorder of face recognition; (d) a disorder of identity perception; and (e) a fundamental “neuropathology of the self”. I will then present a new hypothesis which draws together elements from these, and connects them to the extensive body of research on human kin recognition and its cultural and evolutionary importance.

CURRENT THEORIES

CAPGRAS SYNDROME AS A TWO-FACTOR MONOTHEMATIC DELUSION

Capgras Syndrome is one of the more famous examples of a “monothematic delusion” - a fixed specific false belief held despite all evidence to the contrary. Other examples include Fregoli Syndrome, in which the patient believes that strangers are known persons in disguise, and Cotard syndrome, in which the patient believes him or herself to be dead. It has long been noted that such obsessions, or *idées fixes*, can occur in individuals who appear otherwise rational. The English seventeenth century philosopher John Locke, proposed that “madmen” had not “lost the faculty of reasoning”, but rather “err as men do that argue right from wrong principles” (Locke 1690). Both William James (1842-1910) and Brendan Maher (1924-2009) followed Locke in interpreting delusions as rational conclusions built on defective experiential foundations (James 1890, Maher 1974, 1988, Pacherie 2009) More recently, Max Coltheart has adopted a similar approach, with the subtle clarification that the delusional belief is inferred from anomalous “data”, rather than deduced from phenomenal experiences (Coltheart 2007, 2010, Coltheart, Menzies and Sutton 2010). On the premise that each recognized type of monothematic delusion corresponds to a particular physiological abnormality, Coltheart and the philosopher Martin Davies identifying various different types of anomalous data with specific monothematic delusions: they associate lowered affective responses with Capgras Syndrome, and, at the other end of the spectrum, they identify elevated affective responses with hyperfamiliarity to persons (Fregoli syndrome) and/or to places (reduplicative paramnesia) - see summary in Table 1 (Davies, Coltheart and Langdon, et al. 2001, Devinsky 2009).

Several researchers, including Maher, have speculated that underlying abnormalities might be sufficient in themselves to trigger delusions (Fine, Craigie and Gold 2005, Gerrans 2002, Maher 1974, 1988). However, similar physiological abnormalities can occur without inducing delusional beliefs (Davies and Coltheart 2000, Langdon and Coltheart 2000, Lipson, Sacks and Devinsky 2003, Vuilleumier, et al. 2003). This suggests that another factor is responsible for rendering some subjects particularly susceptible to delusions as Coltheart and others have argued (Coltheart, Langdon and McKay 2011, Davies, Coltheart and Langdon, et al. 2001, Hirstein 2010). More specifically, damage to the right lateral prefrontal cortex has been associated with delusional states (Coltheart 2010, Hirstein and Ramachandran 1997, Stone and Young 1997).

Table 1: Eight monothematic delusions adapted from Davies, Coltheart and Langdon, et al. (2001).

| Syndrome | Delusion | Cause |
|----------|---|-------------------------------|
| Capgras | Close relatives have been replaced with impostors | Flattened affective responses |
| | | |

| | | |
|---|---|--|
| Cotard | Subject believes him/herself to be dead | Global affective flattening |
| Fregoli | People encountered by S are known others in disguise | Heightened affective responses |
| Reduplicative paramnesia | Multiple instances of places or people | Heightened affective responses |
| Alien control syndrome | S believes their body is acting under a volition other than their own | Loss of experience of self-initiation of action or thought. |
| Thought insertion | S believes thoughts are being implanted in their mind from elsewhere | Loss of experience of self-initiation of action or thought |
| Unilateral neglect (hemineglect) | Denial of ownership of one side of one's body | Loss of kinesthetic and proprioceptive feedback |
| Mirrored self-misidentification | Failure to recognize self in a mirror | Disorder of face processing or inaccessibility of visuo-motor transformations for mirrored space |

MONOTHEMATIC DELUSIONS AND LATERALIZATION: CAPGRAS SYNDROME AS A RIGHT HEMISPHERE DELUSION

For some types of the classical monothematic delusion, both factors are associated with right hemisphere damage and dysfunction. As such, monothematic delusions can be the result of unilateral brain damage. This is the case for *reduplicative paramnesia*—in which the patient believes in multiple instances of individual persons, places or things (Weinstein, Kahn and Sugarman 1952), and *unilateral neglect* in which the subject's awareness of one side (usually the left) of their body or of space is impaired (M. Critchley 1953, Mesulam 1981, Stein 1989).

A few studies have suggested Capgras Syndrome is similarly lateralized to the right hemisphere. In 1977, Hayman and Abrams asserted that two cases of Capgras Syndrome displayed evidence of “right-sided cerebral dysfunction” (Hayman and Abrams 1977). They cited a left-sided palmomental reflex as evidence in one case, although it has since been established that lateralization cannot be reliably inferred from this reflex (Gotkine, et al. 2005, Owen and Mulley 2002). Two years later a further case associated with right-sided dysfunction was reported, prompting speculation that Capgras Syndrome could be a variant form of reduplicative paramnesia (Alexander, et al. 1979). A diagnosis of reduplicative paramnesia might have been more appropriate in this case, in that the subject believed family members had been reduplicated rather than replaced. These early reports fueled consideration that Capgras Syndrome could be considered a right hemisphere disorder (Cutting 1990, Feinberg and Shapiro 1989).

Between 1989 and 2001, four meta-analyses explored patterns of lateralization of physical and functional abnormalities in cases of Capgras Syndrome and associated disorders. In the first of these studies, Todd Feinberg and R. M. Shapiro (1989) collated details of 86 subjects with misidentification or reduplication conditions for whom some form of “neurological dysfunction was reasonably certain”; only 26 of these specifically involved misidentification of *persons*, whereas the remaining 60 cases had other targets including places (i.e. reduplicative paramnesia), time (*déjà vu*), and of body parts.

Although there were numerically more cases of right hemisphere dysfunction than left sided in both groups, this was only statistically significant in the larger, latter non-person group.

A second study (Förstl, et al. 1991) surveyed 260 cases of misidentification disorders, dividing these into three groups depending on whether the target of the delusion was (a) other people, (b) self, or (c) place. The first group was by far the largest, numbering 203 cases, including 174 cases described as “Capgras Syndrome”. Out of the 203 cases in this group, unilateral lesions were reported for only 10 (5%): 9 right sided cases and one left sided. The authors did not identify the location of lesions more precisely than the left or right hemisphere, nor did they report on the number of subjects with *bilateral* lesions or abnormalities.

In the third study, Stephen Signer amassed 570 cases of reduplication and/or misidentification disorders (Signer 1994). 79 of the 570 cases (c. 14%) were identified as having localized or lateralized abnormalities, of which 63 were patients with delusions that “persons or objects have been replicated by essentially identical doubles” (a description that encompasses both Capgras Syndrome and reduplicative paramnesia). Signer charted (where available) the cerebral lobe that was the “predominant site of lesion” in the 79 cases, finding similar incidence of left sided (31) and right sided (35) lesions, with the most commonly affected regions being the right frontal lobe (21 cases) and left temporal lobe (13 cases).

A fourth meta-analysis (Breen, Caine and Coltheart 2001) collected the findings from neuroimaging studies of 69 patients with Capgras Syndrome. 40 of these had evident organic damage, including nine with lateralized lesions as follows: six cases with unilateral right hemisphere damage; two cases involving “global atrophy; and a right focal lesion”, and a single case of unilateral left hemisphere damage.

The main findings from these four studies are collated in **table 2**. The relatively high incidence of right hemisphere damage (whether unilateral or bilateral) is compatible with the proposed link between right frontal lobe damage and susceptibility to delusional beliefs in general. Although cases identified with unilateral left sided damage or dysfunction are rare, it is notable that a few have been reported. Signer’s observation that the *left temporal* lobe was the second most commonly affected region aligns well with the hypothesis that Capgras Syndrome is due to affective flattening, in that temporal lobes are thought to play a key role in processing familiarity (E. Critchley 1989, Gainotti 2007, Montaldi and Mayes 2010, Penfield and Perot 1963, Wong and Gallate 2012).

Table 2: Location and Lateralization of Cerebral Damage

| Study | Condition | Description | Left | Right | Bilateral |
|--|--|------------------------------|------|-------|-------------|
| Feinberg and Shapiro (1989) | Reduplication of persons | “hemispheric dysfunction” | 2 | 8 | 16 |
| Förstl et al. (1991) | Misidentification of other people | “focal lesions” | 1 | 9 | Unspecified |
| Signer (1994) | Reduplication/ misidentification disorders | “predominant side of lesion” | 31 | 35 | 13 |
| Breen, Caine and Coltheart (2001) | Capgras Syndrome | lesion | 1 | 8* | 31 |

* includes 2 cases with “global atrophy and a right focal lesion”

CAPGRAS SYNDROME AS A DISORDER OF FACE RECOGNITION

Capgras Syndrome is often considered to be a disorder of face recognition, and more specifically as due to disruption to the neural pathways that facilitate pairing the appropriate emotional responses with individual faces (Breen, Caine and Coltheart 2000, Ellis and Lewis 2001, Gerrans 2002, Hayman and Abrams 1977, Hirstein and Ramachandran 1997, Young, Ellis, et al. 1990, Young, Reid, et al. 1993). As early as the 1970s there was speculation that Capgras Syndrome might be caused by an impaired ability to recognize faces (prosopagnosia) (Hayman and Abrams 1977, Shraberg and Weitzel 1979). However, Bauer's subsequent discovery that prosopagnosics retain a *covert* (nonconscious) ability to recognize faces, helped to establish a dual pathway model of face recognition, in which one visual stream facilitates explicit/conscious face recognition, and a second stream mediates affective responses (Bauer 1984, 1986, Tranel and Damasio 1985). Bauer concluded that the persistence of covert recognition in cases of prosopagnosia showed that the latter stream remained intact. Ellis and Young later suggested that this two pathway model could also be used to explain Capgras delusion, as a mirror image of prosopagnosia, i.e. that Capgras Syndrome might result from damage to the latter stream, impairing emotional recognition, whilst the *former* stream remains intact (Ellis and Young 1990). Their theory was subsequently corroborated by findings showing that covert / autonomic recognition of familiar faces (as measured by skin conductance response) is impaired in Capgras Syndrome (Brighetti, et al. 2007, Ellis, Lewis and Moselhy, et al. 2000, Ellis, Young and Quayle, et al. 1997, Hirstein and Ramachandran 1997). Ellis and Young's hypothesis was instrumental in popularizing the notion of a "double dissociation" between prosopagnosia and Capgras Syndrome, in which each is the result of a damage to one of two distinct pathways, each of which is responsible for different components of face recognition.

CAPGRAS SYNDROME AS A DISORDER OF IDENTITY PERCEPTION

Capgras Syndrome is one of a range of conditions collectively referred to as 'delusional misidentification disorders' (DMDs). The distinction between *appearance* and *identity* is conceptually complex and at times ambiguous. In ancient Greece, *prosopon* (the etymological root of 'prosopagnosia') referred to both person and to face. This tension between these two component parts of our selves is a mainstay of culture from ancient mythologies through to Shakespeare's plays (which a famously are peppered with cases of mistaken identity) and Hollywood blockbusters. Whilst the adoption and interpretation of Ellis and Young's two pathway model has commonly fostered a view of Capgras Syndrome as a disorder of face recognition, the double dissociation between prosopagnosia and Capgras Syndrome can, conversely, support an alternative interpretation that in Capgras Syndrome, it is not face recognition but rather identity perception that is impaired.

In a variation of Ellis and Young's two pathway model, Hirstein has proposed the existence of two distinct cerebral networks, one responsible for representation and recognition of external appearances, and a second responsible for the "internal representation" of persons and things (Hirstein 2005, 118-128). This model lends itself to a view of DMDs being due to disruption of an ability to successfully recognize perceived and projected individual *identities* rather than merely superficial appearance (Berson 1983, Cutting 1990, Enoch 1986, Hirstein 2005, Margariti and Kontaxakis 2006)

Identity recognition is not the same as, and need not entail, the recognition of faces. Prosopagnosics can identify people from non-facial cues such as gait, clothing, hairstyle, and voice (Damasio, Damasio and van Hoesen 1982, Damasio, Tranel and Damasio 1990). Conversely, misidentification in Capgras Syndrome can, and does, occur in blind subjects (Dalgalarondo, Fujisawa and Banzato 2002, Hermanowicz 2002, Reid, Young and Hellawell 1993, Rojo, et al. 1991). Recognizing and misrecognizing identity cannot be reduced to the processes responsible for recognizing *faces*. In 1986 Bruce and Young proposed a role for "person identity nodes" in the recognition process (Bruce and Young 1986) and a distinction between the recognition of faces and of identities has been acknowledged in some subsequent models of Capgras Syndrome (Breen, Caine and Coltheart 2000). Neural network theories allow the possibility that "person identity nodes" may be represented by networks in the brain, assimilating inputs from a variety of sensory modalities.

Further evidence against the view of Capgras as a face recognition disorder can be found in cases of

Capgras were the misidentification is triggered in other modalities, such as the blind woman who claimed she could tell that her husband was an imposter by the taste of his skin, his smell, and his build (Dalgalarondo, Fujisawa and Banzato 2002); and another case where one (sighted) patient's delusion was primarily triggered in response to hearing her son's *voice* (Lewis, et al. 2001). Such cases act as counterpoints to Hirstein and Ramachandran's classic case in which the delusion was triggered by sight, but not when talking on the phone (Hirstein and Ramachandran 1997). However, modality specific cases are few and far between. The scarcity of single or specific modality accounts in the case reports implies that Capgras Syndrome is, much more commonly, a multimodal condition, although few reports have specifically focused on this.

CAPGRAS SYNDROME AS A "NEUROPATHOLOGY OF THE SELF"

In a series of recent publications, Todd Feinberg (author of the 1989 meta-analysis discussed earlier) has argued that DMDs are thoroughly multimodal "neuropathologies of the self", and cannot be tied to a single cognitive defect (Feinberg 2001, 2009, 2010, 2011). In accord with Hirstein & Ramachandran, and Coltheart & Davies, Feinberg postulates that one contributory cause of Capgras delusion is impaired capacity for reality checking, although Feinberg's interpretation of the influence of right frontal lobe damage on this process is much more overtly "psychoanalytical". More specifically, Feinberg supposes that right hemisphere damage contributes to the delusion by fostering "ego disequilibrium" and a return to infantile immature fantasies. Moreover, he identifies the second factor as "an *under-relatedness* to personally significant aspects of the self". According to Feinberg, these two negative factors give rise to "productive" positive symptoms, most notably the delusion itself, which acts as a form of "psychological defense" (Feinberg 2013).

Feinberg's ready acceptance of psychoanalytic terminology sets him apart from the other contemporary accounts discussed above and the approach adopted in the current paper. There is nonetheless scope for dialogue and discussion between his view and the more mainstream cognitive psychological approaches. In a commentary on Feinberg's position, William Hirstein, concedes that "the great majority of cases" do appear to be multimodal, albeit with the caveat that "this has not been systematically studied" (Hirstein 2010). More critically, Hirstein has accused Feinberg of a lack of clarity in his concept of "personal significance", and questions the capacity of this theory to explain why Capgras patients would resort to the conclusion/delusion that their relative is an imposter.

CAPGRAS SYNDROME AS A DISORDER OF KIN RECOGNITION

TWO DEFINITIONS OF KINSHIP

Since the emergence of "social neuroscience" in the late twentieth century, neuroscientists have turned their attentions to topics that had previously, traditionally, fallen under the purview of social anthropologists and evolutionary biologists, and in particular our interactions with other members of our species (Hamilton 1964, Lévi-Strauss 1949). This has, to some extent, included scientific explorations of our interactions with kin. Whilst interdisciplinarity has potential for building new connections and hypotheses, the resulting discussions are particularly prone to terminological pitfalls in that words can (and often do) hold different nuances and meanings for researchers working in different fields. Hence, for more biologically minded scientists, the term "kin" is frequently interpreted as designating close *genetic* relatives; by contrast, for researchers more interested in beliefs and behavior, such as sociologists and social anthropologists, the term is commonly used to refer to those who are *perceived and treated as part of the family group*.

To date, there has been little, if any, engagement or cross-referencing between the scholarly literature on Capgras Syndrome and studies of kin recognition. As such, it is not overly surprising there has not been any real consideration or discussion as to whether the "close relatives" targeted are relatives in a *biological* or in a *social* sense. Of course, the two categories overlap (with the proviso that spouses are a very special case). However, they can also lead to quite distinct predictions and conclusions depending on which sense is used: for instance, the reports of doppelgänger family pets (Darby and Caplan 2016, Rösler, Holder and Seifritz 2001, Somerfield 1999) take on quite a different significance if

one adopts a social definition of kin as part of the family unit.

Across the animal kingdom a wide array of specialized mechanisms facilitate and mediate interactions between kin (in both of the above senses of the word). Kin recognition is thought to have two clear survival benefits: firstly, it provides a ready-made framework for 'nepotistic' alliances, i.e. preferential mutual treatment between members of the same kinship group; secondly, the ability to distinguish close kin allows the avoidance of in-breeding (i.e. incest), which could otherwise lead to the accumulation and expression of recessive genes and negatively impact survival chances of the resultant offspring (Antfolk, Lieberman and Santtila 2012, Hepper 1991, Penn and Frommen 2010).

Compared to species such as the extensively-studied paper wasps, for which phenotypic cues are sufficient to distinguish kin (Jackson 2007), humankind's cognitive toolkit allows us to represent, recognize, interact with, and relate to kin in ways that greatly transcend the processing of superficial sensory cues. Our ability to think in terms of *entities* and *identities* and to maintain complex conceptual relationships between these (in other words, our astounding capacity for conceptual symbolic thought) is not restricted to the niche area of identifying family members. Nonetheless, the specific failure of the identification process in Capgras Syndrome may be revealing, not least because it might provide us with clues to the evolutionary origins of this distinctively human ability.

Olfactory-based mechanisms play a leading role in social interactions between many non-primate mammals, with roles in territory marking, kin recognition and mate selection (Brown and Eklund 1994). Whilst scents and pheromones also play some part in mediating human behavior (Havlicek and Roberts 2009, Lundström, et al. 2009), primate evolution was accompanied by an growing reliance on visual rather than olfactory acuity (Allman and McGuiness 1988, Jacobs 2008, 2009, Rouquier, Blancher and Giorgi 2000, Souza, Gomes and Silveira 2011). Moreover, primates, including humans, undergo a comparatively long period of dependency in infancy: the resultant extended period of cohabitation provides an enhanced opportunity for the development of kinship bonds and recognition of family social structure (Chapais 2008). Over 100 years ago, the Finnish sociologist, Edvard Westermarck, proposed that cohabitation during childhood suppressed sexual attraction, i.e. that people who grow up together do not see each other as prospective mates (Westermarck 1891). Westermarck's hypothesis has since been corroborated by studies of (genetically unrelated) people raised together, for instance, on Israeli Kibbutzim (Lieberman and Lobel 2012, Shepher 1971, Wolf 1995).

In a paper published in *Nature*, Debra Lieberman, John Tooby and Leda Cosmides, have proposed that humans have a computational biological "central kinship mechanism" which allows us to estimate the degree of kinship from two main cues: the intimate early relationship between mother and offspring, and the duration of co-residence. They note that other factors, such as the discrimination of scent and "resemblance" may also contribute to this process. This "kinship estimator", they argue, uses algorithms "to compute the magnitude of a regulatory variable—a kinship index—for each individual" which in turn informs two "programs" one for facilitating nepotistic behavior ("sibling altruism") and another for incest aversion (Lieberman, Tooby and Cosmides 2007).

Kin recognition and Capgras Syndrome have traditionally been explored via quite distinct and disparate research programs. Discussion of 'kin recognition' is entirely absent from the literature on the Capgras delusion, and vice versa. Yet, if a "kin recognition module" does exist, this might provide an explanation for the specificity of the delusion to close relatives. Functional or physical disruption to this module might result in anomalous reactions to viewing relatives, contributing to the delusion that the relative is an imposter. Lieberman et al leave open the question of how and where this "central kinship mechanism" might be physically manifest in the "wetware" of the brain, although several other studies have reported specific patterns of endocrinal (oxytocin and vasopressin) and neural (especially limbic region) activity accompany mother-child bonding and between romantic partners (Bartels and Zeki 2000, 2004, Carter 1998, Insel and Young 2001, Leibenluft, et al. 2004).

Temporal lobe function has been associated with both kin recognition and Capgras Syndrome. Signer's

observation that subjects in his meta-analysis had a notable incidence of *left* temporal lobe damage raises the intriguing possibility that parts of the neural networks responsible for selective autonomic responses to kin are lateralized in the left hemisphere. Neuroimaging studies of healthy subjects viewing photos of their romantic partners and offspring have reported significant levels of activation in the left temporal lobe, and specifically a well-connected subcortical structure called the insula (Bartels and Zeki 2000, 2004, Carter 1998, Leibenluft, et al. 2004). The insula is a functionally diverse and integral part of the limbic system, which receives and integrates sensory inputs from multiple modalities, and plays a role in autonomic responses to personal identities (Cauda, et al. 2012, Craig 2011, Haxby and Gobbini 2011). Scent-based kin recognition in women has also been associated with activity in the insula (Lundström, et al. 2009). In short, it is not implausible to suppose the insula, and specifically the left insula, plays some part in human kin recognition.

One of the more puzzling features of Capgras Syndrome is that the delusion only occurs in the process of recognizing a few individuals (usually close family members) yet skin conductance responses appear to be suppressed on viewing *all* familiar persons, whether family or friends. There is, however, an overlooked element that may benefit from further research, namely the lateralization (i.e. side of the body) on which the skin conductance responses are measured to assess limbic activity. Previous studies have followed convention by basing their findings on readings taken from the non-dominant (i.e. left) hand (Brighetti, et al. 2007, Ellis, Young and Quayle, et al. 1997, Hirstein and Ramachandran 1997). Limbic system connections to the peripheral nervous system are ipsilateral: whilst skin conductance responses on the left hand and limbs may give a valid measure of activity in the left hemispheric limbic system, there may be differing patterns of activity levels in the *right* hemisphere (Banks, Bellerose, et al. 2012, Banks, Bellerose, et al. 2014, Picard, Fedor and Ayzenberg 2016).

This leaves open the possibility – as of yet unexplored – that damage to the left temporal lobe could result in the flattening of left sided autonomic skin conductance responses, yet the right temporal lobe may still retain some capacity for emotional recognition of known persons. *If* (and this is a big if) kin recognition functions were to some extent localized to the left hemisphere, then left temporal lobe damage could conceivably effectively extinguish the “special glow” evoked by kin. This is of course, largely, conjecture – but it is a hypothesis that can be easily tested in future studies by routinely measuring skin conductance bilaterally (or where more resource is available, by using more sophisticated imaging methods.)

To sum up: according to the established dominant model, Capgras Syndrome is caused by abnormally disrupted and flattened affective responses to known people. This primarily manifests in the misidentification of *family* members because only with family is the disparity between anticipated and actual response sufficient to trigger the delusion. In other words, the focus on family is incidental and tangential. By contrast, in this paper the targeting of family members takes on a different and additional significance, being indicative of damage to (and the existence of) a human cognitive kin recognition module. These two hypotheses are not entirely mutually incompatible. In *both* scenarios, family members have a special association with Capgras delusion. Both accounts suppose that the emotional response evoked on viewing family differs from that of viewing friends. The open question probed by this article is whether this difference between our responses to family and friends is merely one of degree, or whether it is also one of kind. Is the apparent targeting of kin merely the tip of the iceberg, the point at which the more general loss of affective responses to others becomes most ‘visible’? Or does Capgras Syndrome reveal hidden processes of kin recognition, making kin recognition itself more central and fundamental to the etiology of Capgras than previous theories have supposed? Irrespective of which hypothesis holds true, further research into the neural basis of kin recognition – how we represent, react and interact with family members - has potential for interdisciplinary collaboration that connects traditionally disparate fields of neuroscience, anthropology and evolutionary psychology.

IDENTIFICATION AS A MECHANISM OF HUMAN KIN RECOGNITION

If humans do indeed possess cognitive abilities allowing us to estimate the degree of relatedness of

others, as Lieberman and colleagues have proposed, how might these abilities compare to, and differ from, mechanisms for kin recognition in other species? Given the elevated status of vision in the hierarchy of the senses in both primates and man, one might expect to see evidence that we are able to discriminate kin from non-kin visually. And indeed, several studies have demonstrated that we are predisposed to discriminate favorably towards people who look like us, which may be a valid indicator of genetic kinship (Bressan and Zucci 2009, DeBruine 2002, DeBruine, Jones and Little, et al. 2008, DeBruine, Jones and Watkins, et al. 2011, Platek and Kemp 2009). Yet even a moment's introspection is enough to reveal that the way we commonly identify kin is a cognitive leap ahead of this. We don't rely exclusively or even primarily on superficial sensory cues or phenotypic resemblances to estimate kinship. Rather, we learn who our relatives *are*: we develop and retain multifaceted conceptions of individual identities, including the relationships between individuals, and ourselves. We are so familiar with the human capacity to maintain and recognize identities that it is easy to overlook just how profoundly powerful and pervasive this cognitive tool is.

The ability to tether appearances and identities, persons and faces, appears to be impaired in Capgras Syndrome and - curiously - this impairment predominantly presents as the misrecognition of *family members*. This raises the prospect (though by no means proves) that the ability *to identify* has some kind of association with kin recognition. One possibility that merits consideration is that the ability to tether appearances and identities *may itself have emerged as, or in tandem with, a new and improved means to identify kin, by a multimodal means that allows the recognition of persistent identities and relationships over time* – in other words, that identification allowed the development of a structured kinship recognition system, in a milieu in which the previous heavy reliance on scent-based recognition was increasingly inadequate for the task.

CONCLUSION

Capgras Syndrome is, by reputation at the very least, a 'kin recognition disorder', in that it is characterized by the delusion that that someone, *typically a close relative*, has been replaced by an imposter. Whereas early psychodynamic theories considered the familial dimension of the condition important, more recent cognitive neuropsychological approaches have relegated this peculiar yet distinctive aspect of the condition to the periphery. Over the last forty years, a greater emphasis has been placed on identifying an organic basis for the disorder. Like other monothematic delusions and delusional misidentification disorders, Capgras Syndrome is associated with an impaired grasp on reality associated with right frontal lobe dysfunction. It has been proposed that a second factor determines why delusions take the particular form they do. In the case of Capgras Syndrome, impaired emotional responses on encountering other people has widely been put forward as the second factor. I have suggested an alternative second factor, namely damage or disruption to a neural network that plays a role in recognizing and representing *identities of family members*.

Whilst kin recognition has been of significant interest to sociologists and evolutionary psychologists for decades, the possibility of a relationship between Capgras Syndrome and the mechanisms for kin recognition has yet to be given any serious consideration within the cognitive neuropsychological literature. Further exploration of relevant factors, such as modality, laterality, and cerebral regions and processes involved, could potentially prove fruitful in further improving our understandings of not only Capgras Syndrome but also of human kin recognition and identity perception.

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