What theoretical interpretations of Capgras Delusion exist and to what extent are they supported by neurological evidence?

Abstract

Capgras Delusion (CD) is a rare Delusional Misidentification Syndrome (DMS) characterized by the fixed pathologic belief that an impostor has replaced a loved-one. Although originally regarded as a purely functional psychiatric condition, neurological research indicates CD has an organic etiology. Three theoretical explanations of the causes of CD are outlined and critically considered in light of neurological evidence. These theories include the possibility that CD results from: a disruption to the neural mechanisms underlying face processing (Ellis & Young, 1997), right frontal lobe damage and an inferotemporal-limbic disconnection (Hirstein & Ramachandran, 1997) or a hemispheric imbalance (Devinsky, 2009). Ultimately, whilst neurological research is partially supportive of the two latter theories (Hirstein & Ramachandran, 1997; Devinsky, 2009), this field of research requires use of more sophisticated and sensitive neuroimaging techniques to better elucidate the underlying cause/s of CD.

Key words: Capgras Delusion; misidentification disorder; neuropsychiatry
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Capgras Delusion (CD) is classified as a reduplicative Delusional Misidentification Syndrome (DMS) in the DSM-IV (American Psychiatric Association, 2000) and is the fixed pathologic belief that a physically identical ‘double’ or ‘impostor’ has replaced a loved-one (Christodoulou, Margariti, Kontaxakis, & Christodoulou, 2009; Devinsky, 2009). Sufferers recognise the loved-one’s physical similarities but misidentify them, claiming for example: “She looks exactly like Wilma… [but] she is not the Wilma I know” (Lucchelli & Spinnler, 2007, p. 194). Although initially regarded as a purely functional psychiatric condition (Enoch, 1963) numerous cases studies linking CD with brain damage and neurodegenerative disorders indicate an organic etiology (Ellis & Young, 1990). Elucidating the underlying neural mechanisms of CD is key to developing effective and appropriate treatment for sufferers (Ellis & Young, 1990; Papageorgiou, Ventouras, Lykouras, Uzunoglu, & Christodoulou, 2003). Theories that attempt to explain how and why CD is caused vary in the degree to which neurological evidence (e.g., case/imaging studies) supports them. Ultimately more sensitive imaging techniques are needed to further elucidate the neurological underpinnings of CD.

Ellis & Young (1990) proposed CD is caused by disrupted neural mechanisms underlying face processing. Patients recognise a familiar person because their ventral-stream, responsible for overt face recognition, is intact. They misidentify them because of a disconnection along the dorsal-stream, which Ellis and Young argue is usually responsible for retrieving affective information. The resulting delusion reflects the patient’s post-hoc rationalization for the discrepancy between
conscious recognition and absent feelings of familiarity. This theory relies on double-dissociations between patients with prosopagnosia and capgras for empirical support. Patients with prosopagnosia have ventral damage and cannot recognise familiar faces but elicit appropriate affective responses (Bauer & Verfaellie, 1988). Ellis and Young (1997) argued since patients with CD show the reverse symptoms they must have dorsal damage.

Though compelling, Ellis and Young’s dorsal disconnection theory lacks neurological support. If CD is caused by a dorsal-stream disruption, patients with organic CD should have parietal and/or occipital lesions (Hirstein & Ramachandran, 1997). While true for some (e.g., Edelstyn, Oyebode, & Barrett, 2001) most capgras patients have frontal and/or temporal lesions (Signer, 1994) suggesting the dorsal-stream need not be implicated. If anything, the high rate of temporal involvement indicates a ventral-stream disconnection (Hirstein & Ramachandran, 1997). Additionally, the dorsal disconnection theory is predicated upon a dated model of face recognition (Bauer, 1984) that is no longer supported by neurological evidence (Breen, Caine, & Coltheart, 2000). Studies on patients with optic-ataxia (dorsal-stream damage) suggest the dorsal-stream is involved in planning and guiding actions toward objects (Milner & Goodale, 2008) not covert face recognition. Also, patients with optic-ataxia are not known to experience CDs, suggesting the dorsal-stream is not causally implicated.

Like capgras patients, those with ventromedial prefrontal cortex (VMPC) damage recognise familiar people but do not elicit appropriate skin conductance responses (Tranel, Damasio, & Damasio, 1995). If CDs are post-hoc rationalizations
of the discrepancy between conscious recognition and absent feelings of familiarity, patients with VMPC damage should, but do not, experience CDs (Coltheart, Langdon, & McKay, 2011). Thus, even if a dorsal disconnection is somehow responsible for absent feelings of familiarity in capgras patients, Ellis and Young’s theoretical interpretation is incomplete as other regions are likely implicated in the manifestation of the delusion itself (Coltheart et al., 2011; Ramachandran, 1995). Overall, the dorsal disconnection theory is not supported by neurological evidence. Hirstein and Ramachandran (1997) offer an alternate theory that accounts for the problems met by Ellis and Young’s. They proposed a ventral, not dorsal disconnection causes CD.

Hirstein and Ramachandran (1997) interpret CD as a “general memory management problem” (p. 441) caused by an inferotemporal-limbic disconnection and damaged right-frontal lobe. Face processing areas in the temporal lobe (i.e., fusiform gyrus) operate normally but are disconnected from limbic regions. This prevents facial information from being evaluated for emotional significance by the amygdala. In the absence of limbic activation, patients are unable to form associations between a particular person and episodic memories of them (after onset of capgras syndrome). This prompts the brain to create a new memory store or ‘file’ for this person every time the patient sees them, as if they had never met. Right-frontal damage causes misidentifications to become delusions because it prevents the right-hemisphere from counterbalancing the left-hemisphere’s tendency to explain discrepancies (Ramachandran, 1995).

Numerous case studies provide neurological support for the involvement of the right-frontal and temporal lobes in CD. Right-frontal damage is often reported in
organic capgras cases (e.g., Signer, 1994) and often associated with delusions in non-capgras cases (e.g., Levine & Grek, 1984; Sultzer et al., 2003). Though such findings support a necessary role for the right-frontal lobe in delusion onset, it may not be sufficient. It is also not clear whether the right-frontal lobe is associated with delusions for the reason Hirstein and Ramachandran suggest.

Although no direct evidence exists for an inferotemporal-limbic disconnection in CD, involvement of the temporal lobe (Signer, 1994) specifically limbic regions is well supported. Combined Single-Photon Emission Computerized Tomography (SPECT) scans of patients with Lewy Body Dementia (LBD) and DMSs indicated significant hypoperfusion (decreased blood flow) in limbic and paralimbic regions (Nagahama, Okina, Suzuki, & Matsuda, 2010). This included the hippocampus, which is argued to contribute to familiarity assessments by retrieving relevant episodic information (Bowles et al., 2007). Thus supporting Hirstein and Ramachandran’s theory that CD is partly caused by an inability to retrieve recent episodic memories associated with someone they know. However, CD was largely underrepresented in the sample; twenty-two DMS patients were studied but only five experienced CDs. Thus unless all cases had hypoperfusion in the exact same areas, the extent these findings are representative of CD is questionable.

Another, more unusual case study, further supports the hypothesis that the limbic system is affected in CD (Antérion, Convers, Desmales, Borg, & Laurent, 2008). Patient ‘RP’ had Parkinson’s disease and right-temporal atrophy specific to the amygdala and hippocampus. ‘RP’ believed his wife was a ‘double’ during sexual intercourse with her. Because the delusion occurred when feelings of familiarity
should have been at their peak, Antérion et al. (2008) argued the limbic system might be more heavily involved in CD than Hirstein and Ramachandran gave credit and that the limbic disconnection may not be specific to face processing areas. Thus far, the above neurological evidence suggests the right-frontal lobe and limbic system is affected in CD. Although useful, such studies cannot tell us whether a more specific disconnection between limbic regions and face processing areas causes it.

Though not specific to CD, one study indicates a localized limbic disconnection is possible in reduplicative DMSs. Reduplicative Paramnesia (RP) is another DMS involving the delusional belief that a well-known place has been duplicated (Budson, Roth, Rentz, & Ronthal, 2000). Hirstein and Ramachandran (1997) argued in a similar vein to CD, a disconnection between the Parahippocampal Place Area (PPA) and limbic system causes RP. In support, Magnetic Resonance Imaging (MRI) of a patient with RP revealed white matter damage specific to Broadman’s areas nineteen and thirty-seven, disrupting parahippocampal-limbic regions (Budson et al., 2000). Thus supporting Hirstein and Ramachandran’s theory that the exact location of the limbic disruption could depend on whether reduplicative delusions are restricted to places (parahippocampal-limbic) or faces (fusiformgyrus-limbic). In summary, neurological evidence supports the hypothesis that temporal and frontal regions are affected in CD but does not directly support the existence of a specific inferotemporal-limbic disconnection. Devinsky (2009) offers a more comprehensive theoretical account of CD that incorporates some of Hirstein and Ramachandran’s theory.
Devinsky (2009) argues CD is caused by “right brain lesions [and] left brain delusions” (p.80). Usually, the left-hemisphere seeks order and reason, interprets situations, generates explanations and makes categorisations. The right-hemisphere monitors reality, makes familiarity decisions via the temporal and frontal lobes and keeps the left-hemisphere ‘in-check’. Right-hemisphere damage causes the left to become overactive, unleashing “a creative narrator” (p. 80). Without the right-hemisphere to monitor it and signal feelings of familiarity, the left’s bias to explain discrepancies and form categorisations causes the belief that a recognizable person must be a ‘double’. Despite clear evidence this belief is wrong, frontal-lobe damage disrupts the ability to flexibly disregard it, causing the belief to become fixed.

As Devinsky’s theory is based on existing neurological evidence, the hypothesised roles of each region and their involvement in CD are well supported. The role of the right-temporal lobe in forming feelings of familiarity is supported by a study that found patients with epilepsy, experienced déjà vecu after seizures or stimulation in this region (Vignal, Maillard, McGonigal, & Chauvel, 2007). The right-temporal lobe is often affected in patients with capgras (Feinberg, DeLuca, Giacino, Roane, & Solms, 2005; Signer, 1994), particularly limbic regions (see above). The left-hemisphere’s tendency to explain discrepancies is evident in corpus callosotomy patients who confabulate when asked to explain their reaction to stimuli presented in the left visual field (Gazzaniga, 2000; Sperry, Zaidel, & Zaidel, 1979). Though the left-hemisphere is usually spared in organic capgras cases (Feinberg et al., 2005), it is not clear whether it becomes ‘over active’ as Devinsky suggested. To elucidate this, future research could use functional MRI (fMRI) to measure activity levels in the left-hemisphere while patients view photos of people they believe are
‘doubles’. Although “right-brain lesions” likely cause CD, whether this causes “left-brain delusions” is not clear at present.

Frontal-lobe damage has previously been associated with the onset of delusions in non-capgras cases (e.g., Malloy & Richardson, 1994) and is highly prevalent in CD and other DMSs (e.g., Malloy, Cimino, & Westlake, 1992). Non-delusional patients with frontal damage find it difficult to flexibly alternate between rules on a card-sort task (Anderson, Damasio, Jones, & Tranel, 1991). Suggesting frontal-lobe damage does not cause delusions but could prevent patients from flexibly alternating between possible explanations, as Devinsky suggested. Ultimately, neurological evidence supports Devinsky’s theory but to a limited extent. Although the right-hemisphere and frontal lobes are affected in CD, whether CD is due to hemispheric imbalance is not yet clear.

Overall, the extent theories considered are supported by neurological evidence is restricted. Existing research on CD is mainly relational, drawing associations between regions affected by brain damage and CD. Although such studies are useful for identifying which regions contribute to the delusion, they cannot tell us how or why they do this (Christodoulou et al., 2009). Also, it is difficult to elucidate whether the entire affected region (e.g., temporal lobe) is causing CD or whether it is masking a crucial disconnection (e.g., inferotemporal-limbic). In many cases the imaging techniques used to examine patients, such as Computerized Tomography (CT) are not sensitive enough to identify such subtle disconnections (Hirstein & Ramachandran, 1997).
The above problems could be overcome by adopting more sophisticated imaging techniques in the study of CD. FMRI could offer insight into how delusions develop (Ellis, 2007) and whether the left-hemisphere is over active as Devinsky (2009) suggested. Diffusion Tensor Imaging (DTI) produces images of neural tracts in the brain, so could help elucidate whether an inferotemporal-limbic (Hirstein & Ramachandran, 1997) or dorsal (Ellis & Young, 1990) disconnection exists in capgras patients. Alternatively, Quantitative Magnetic Resonance Imaging (QMRI) would enable detection of subtle differences between areas of the brain that are not otherwise visually detectable (Chang & Jara, 2005) but could be influencing behaviour.

To conclude, different theoretical interpretations of CD exist, which vary in the extent neurological evidence supports them. Ellis and Young (1990) suggest a dorsal disconnection causes CD but this is unsupported. Hirstein and Ramachandran (1997) suggest right-frontal lobe damage in addition to an inferotemporal-limbic disconnection causes CD. Case studies indicate the frontal lobes and limbic system are affected but no study has explicitly reported a disconnection. Devinsky (2009) suggests CD is caused by right-hemisphere damage specific to the frontal and temporal lobes resulting in left-hemisphere delusions. Evidence supports the involvement of right frontal and temporal lobes but whether they cause the left-hemisphere to become over-active is not clear. Ultimately, this field of research would benefit from adopting more sensitive imaging techniques to be able to elucidate further, which theory, if any, is better accounted for by neurological evidence.
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