Degree project

# The Origin of the Doppelgangers: A Review of the Neurological Explanations of Capgras Syndrome

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## Abstract

At its core, Capgras syndrome is the delusional belief that someone close to you has been replaced by an identical imposter. Capgras has historically been explained with Freudian psychodynamic theories concerning latent hostility and a sense of personal or professional inadequacy. There is now widespread consensus in the scientific community that Capgras syndrome can arise from brain lesions. Various hypotheses have been proposed to explain its mechanism. The mirror-image-of-prosopagnosia hypothesis posits a disruption of the dorsal visual pathway. Another hypothesis emphasizes the role of a single lesion affecting the functional connectivity of the retrosplenial cortex. Our analysis of 10 case studies reveals inconsistencies with these hypotheses, particularly in accounting for the diverse lesion locations observed in Capgras patients. Our findings suggest that Capgras syndrome likely stems from a multifactorial aetiology involving neurological and neuropsychiatric factors. Lesions may impact multiple areas associated with facial processing and belief evaluation, challenging the notion of a single lesion explanation.



*Keywords*: Capgras, delusion, neuroimaging, lesion.

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Beliefs are an integral part of human cognition, serving as an interpretive framework explaining the world around us, and are generated when an event occurs for which the individual needs an explanation. Candidate beliefs are formed and evaluated based on biases and verisimilitude, then at least provisionally accepted as true (Connors & Halligan, 2015). When the individual is presented with a better explanation, their belief system should be updated; but this isn't always the case, and some cling to their convictions in opposition to all observable evidence. Delusions are defined in the *Diagnostic and statistical manual of mental disorders* (5th ed.; American Psychological Association, 2013, p. 87) as

> ...fixed beliefs that are not amenable to change in light of conflicting evidence. Their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, grandiose)... The distinction between a delusion and a strongly held idea is sometimes difficult to make and depends in part on the degree of conviction with which the belief is held despite clear or reasonable contradictory evidence regarding its veracity.

Delusional disorders afflict approximately 0.2% of the global population (American Psychiatric Association, 2013, p. 92). The most common theme is persecution, where the individual believes themself oppressed or harassed by some group or individual, when observers would say that they're self-evidently not. More bizarre delusions include beliefs that aren't logically or physically possible, like organs having been removed from one's body without leaving any marks (p. 87).

Delusional misidentification syndromes are characterized by the belief that some place, object, or person, typically affectionately close to the individual, has been altered or replaced. The number of delusional misidentification syndromes varies from four to eleven depending on the categorization scheme (four for Christodoulou, 1986; eleven for Joseph, 1986). Some researchers (Lykouras et al., 2008; Klein & Hirachan, 2014) argue that some syndromes overlap and are too transient to be regarded as distinct syndromes. The most well-known and researched delusional misidentification syndrome is Capgras syndrome (originally called *illusion de sosie* ["illusion of doubles"]), first described by French psychiatrist Joseph Capgras (Capgras & Reboul-Lachaux, 1923). His patient, Madam M., was a 53-year-old French woman who believed that her husband and children had been replaced by doppelgangers. As time progressed, so did the delusion. Ultimately her family, neighbours, and the police had all been replaced by doubles. Madame M. claimed that she herself had several doubles, who received visits and packages that the real Madame M. did not.

Capgras syndrome stands out among delusional misidentification syndromes in that the patient cannot properly identify the subject of the delusion, whereas in other such syndromes, the patient tends to ascribe an identity incorrectly (Barrelle & Luatué, 2017). In the Frégoli delusion, the patient believes that someone is following them around dressed up as someone else (Roane et al., 2019). With intermetamorphosis, the patient believes that someone has changed their physical appearance to look like someone they are not (Ventriglio et al., 2020).

Capgras syndrome afflicts 0.14-1.3% of psychiatric patients (Salvatore et al., 2014; Tamam et al., 2003), 1.1% of stroke patients (Kakegawa et al., 2020), and 14.1% of first acute psychotic episode patients (Salvatore et al., 2014), affecting women more often than men with a ratio between 1.4:1 (Pandis et al., 2019) and 2:1 (Förstl et al., 1991; Bell et al., 2017). Its diverse aetiologies shape the theories concerning its neural basis and patients' experience of their condition. Pandis et al. (2019) found in their systematic review that both functional and organic aetiologies ("functional" refers to cases stemming from psychological issues with no clear structural damage, "organic" to cases with clear physiological damage[1]) result in delusions centered on persons close to the patient; but patients with a functional aetiology (144 of 255 cases; 56%), stemming from such conditions as schizophrenia (83 of 255 or 32%), tend to have delusions encompassing a wider variety of imposters; whereas those with an organic aetiology (111 of 255 or 43%), stemming mainly from dementia (39 of 255 or 15%) or

[1] These terms are used in their historical way and can be considered less than suitable today.

organic delusional disorder (50 of 255 or 19%), tend to have delusions centered on inanimate objects.

The perceived intentions of the doubles can be subtly pleasant but confusing, as in the case of a man who believed that his entire family had been doubled but expressed positive feelings toward both of his wives and was thankful that his original wife had managed to find such a suitable substitute (Alexander et al., 1979). They can also be malicious, as in the case of Madam M., who thought that everyone was colluding against her (Capgras & Reboul-Lachaux, 1923). When the patient feels threatened, they may act out in a misguided attempt to defend themselves. That said, cases of violence mostly occur when patients also suffer from schizophrenia or schizoaffective disorder (Bourget & Whitehurst, 2004), as in the case of a 45-year-old man diagnosed with schizoaffective disorder who killed his father with the justification that the victim was not his father but an imposter who wanted to hurt the family (Trotta et al., 2021).

**Previous explanations of Capgras syndrome**

In the original Capgras paper, the authors note that Madam M. no longer felt the sense of familiarity she once had when viewing faces; they interpret the delusion as a product of clouded emotional judgment (Capgras & Reboul-Lachaux, 1923). In subsequent papers, Capgras leaned more towards Freudian explanations, attributing the condition to Oedipal feelings: by denying the identity of a loved one such as a father (Capgras, 1924), the daughter is free to express her amorous feelings towards him (Ellis, 1994).

Other psychodynamic theories (like the just-mentioned Freudian one used by Capgras) claim that the syndrome is caused by the patient's inability to reconcile with their shortcomings. One possibility is that the patient creates an unflattering double of their spouse who inhabits the patient’s flaws, and whom they can use as a scapegoat for unfulfilled aspirations (Davidson, 1941). Some theories suggest that a paranoid personality type, combined with feelings of depersonalization and derealization (detachment from oneself and surroundings) contributes (Todd et al., 1981). Theories like these reigned supreme until neuropsychological theories based on cerebral dysfunction took hold at the end of the 20th Century (for a brief historical overview see e.g., Barrelle & Luauté, 2017).

Ellis and Young (1990) suggest that Capgras mirrors prosopagnosia, where recognizing faces is made difficult due to ventral visual pathway damage. While prosopagnosia patients exhibit expected skin conductance responses (a common method for investigating emotional arousal; Dawson et al., 2011)[2], Capgras patients recognize faces but lack emotional connection, which according to Ellis and Young could indicate dorsal visual pathway damage. They suggest that damage to this pathway disconnects facial visual stimuli from emotional responses typically originating from the limbic system via the inferior parietal lobule. (Note that the idea of a limbic system is controversial; see, e.g., Rolls, 2015). Hirstein and Ramachandran (1997) report on a 30-year-old man, post-coma, who believed his family members were replaced by imposters, but only during face-to-face encounters. During phone-conversations the delusion was dispelled, suggesting a connection to visual stimuli. However, Hirstein and Ramachandran critique Ellis and Young's theory, arguing that temporal or frontal lobe lesions, which are common in Capgras syndrome, do not align with the proposed dorsal visual system disconnection.

Cases of Capgras syndrome have been observed in blind people, casting further doubt on Ellis and Young. Rojo et al. (1991) report on a 32-year-old man who has been blind for three years due to diabetic retinopathy. While recovering from a hypoglycaemic coma, he began to experience haptic and visual hallucinations. A week after the hallucinations disappeared, he started to believe that his mother had been replaced by a double because her hand felt softer. Cases of Capgras have also been found in congenitally blind people (Reid et al., 1993).

Coltheart (2010) proposes that two deficits are necessary to create a lasting delusional belief: first, a neuropsychological deficit giving rise to the delusional belief (in Capgras syndrome, this likely involves the inability to appropriately connect emotional responses with

[2] Tranel et al. (1995) show that bilateral damage to the ventromedial frontal cortex induces the same skin conductance response as Capgras patients, but without the delusional belief.

familiar identities); and second, the inability to properly evaluate the belief. The second factor distinguishes Capgras syndrome as a delusional disorder from other conditions that hinder identification like prosopagnosia, where individuals are often aware that there's a logical explanation for their difficulty recognizing faces. While the deficit required for the first factor likely varies among delusional disorders, the second – belief evaluation – is thought to be linked to disruptions in the right lateral prefrontal cortex (Coltheart, 2010), an area heavily implicated in executive functions like decision making and general intelligence (Friedman & Robbins, 2021).

A study by Darby and Prasad (2016) on the lesion locations of delusional misidentification syndromes seems to back this up. They collected imaging data from 61 patients believed to suffer from some kind of delusional misidentification. Among the imaging methods used were magnetic resonance imaging (MRI), which uses powerful magnets to generate detailed images of body structures, and computed tomography (CT), which uses x-rays to produce detailed cross-sectional images of the body. The results show that right hemisphere frontal regions were significantly ($p < .001$) correlated with delusional misidentification disorders. The researchers note that, since 37% of the lesions were located elsewhere, the delusion is probably due to network disruptions, rather than having a focal point.

A meta-analysis on the functional connectivity of lesion locations in patients with delusional misidentification syndrome (Darby et al., 2017) focused on networks related to familiarity and belief evaluation. The researchers found that all 17 lesions in their study were functionally connected to the retrosplenial cortex: an area on the medial surface of the temporal lobe. The retrosplenial cortex connects to the hippocampus, parahippocampal regions, limbic system, and parietal cortex, serving a variety of uses, such as the retrieval of autobiographical memory and theory of mind (the ability to understand that other people have their own intentions and beliefs).

Autobiographical memory and theory of mind are of high interest as they play a valuable role in understanding the relationship between the emotions typically associated with one’s environment (Bahk & Choi, 2018). This could explain the occurrence of Capgras delusions in both sighted and blind patients, as these processes handle identification and emotional connection without being tied to a specific sensory modality.

Darby et al.’s (2017) meta-analysis revealed that tasks that involved updating one's representation of the world and reorienting attention increased activity in the ventral frontal cortex. By comparing their Capgras patient data with data from other neurological conditions, the researchers found that damage to the right ventral and dorsal frontal cortex are more related to delusions than any other neurological disorder, but only Capgras patients consistently show lesion locations functionally connected to the retrosplenial cortex.

**Previously implicated neural correlates of Capgras**

The most prominent cortical area related to delusions is the frontal lobe, which Coltheart (2010) suggests is the seat of the necessary secondary factor for a delusion to take hold: i.e., damaged belief evaluation, which the frontal lobe (typically in the right hemisphere) is heavily involved with. Lesions in Capgras patients have been found in other areas, such as the parietal, parieto-occipital, and occipital lobes through which the dorsal visual pathway travels, damage to which could be a factor in Capgras syndrome (Ellis & Young, 1990). The temporal and insular lobes are heavily involved with memory and emotional processing (see, e.g., Höistad & Barbas, 2008); as previously stated, some theories (Capgras & Reboul-Lachaux, 1923; Hirstein & Ramachandran, 1997; Darby et al., 2017) argue that the syndrome could be explained by a disconnection between memory of the object and either its visual representation or the emotional response it once brought.

Another relevant structure is the thalamus, a subcortical structure through which all sensory modalities except smell pass. If damaged, it can play a part in psychotic states: e.g., by inducing delusions or delirium (Onofrj et al., 2023).

**Aim of this systematic review**

Research on this topic can help medical professionals prepare themselves and their patients for what their patients might experience after suffering brain damage, as Capgras syndrome at times does not reveal itself until years after damage has occurred (Darby & Prasad, 2016). Our review seeks to analyse the existing neuroscientific literature to identify specific lesion locations in patients associated with Capgras syndrome, aiming to enhance understanding of its neural mechanisms. Previous attempts at investigation have encompassed the entire spectrum of delusional misidentification disorders and not focused solely on Capgras syndrome.

Although Capgras syndrome is relatively rare, individuals with Capgras syndrome experience tremendous levels of psychological distress, which extends to their families and caregivers. Investigating this and similar exotic conditions holds the potential to understanding more about the brain's ability to tie memories and affective responses to familiar stimuli along with the cognitive processes involved in belief formation (Connors & Halligan, 2015). So, what is the consensus of the literature on the lesion locations in Capgras syndrome and what can we learn from it?

## Methods

### Search strategy

A search was conducted 16 May 2024 in the databases Web of Science, Scopus, and Medline EBSCO. The search string was Capgras AND (MRI OR "magnetic resonance imaging" OR CT OR "computed tomography").

Duplicates were removed manually. The search yielded 233 results: 49 from Web of Science, 130 from Scopus, and 54 from Medline. With all duplicates removed, 127 hits remained (see Figure 1). Of the 127 hits, 116 were excluded. The remaining 11 were sought for retrieval and, after the second screening, seven of the hits were included in the review. Three more studies were found in the reference lists of previous studies and were evaluated for suitability and included, which left the systematic review with 10 studies.

### Inclusion and exclusion criteria

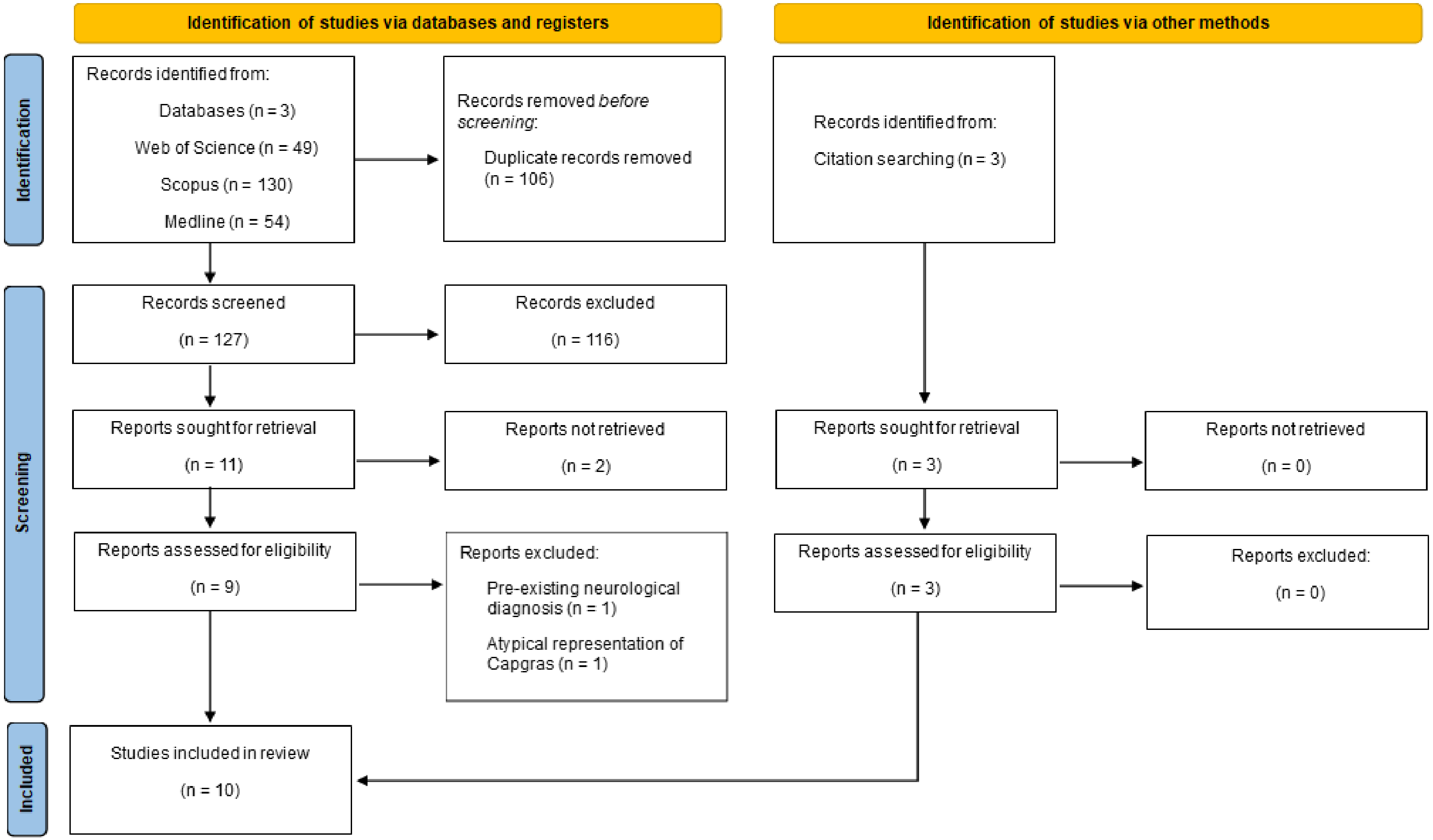
Publications had to be written in English, appearing in peer-reviewed journals, with no restrictions on publication date. Studies needed to be (1) empirical case studies (2) addressing Capgras delusion with (3) no explicit diagnosis of other psychiatric or neurological disorder while (4) making use of either MRI or CT scanning, as they are the most common structural brain imaging techniques used. The extraction of relevant data was made by both authors (S.O. and P.T.), and the studies included in the systematic review were divided into two parts. For the first part, S.O. was the first reviewer and P.T. the second. For the second part, P.T. was the first reviewer and S.O. the second.

### Data extraction

The data extracted included the authors' names; publication year; patient's age, gender, and lesion location; neuroimaging method used; neuropsychiatric tests and their results; any other tests applied (such as SCR); subject(s) of the delusion; duration of the delusion; and perceived hostility of the double.

Figure 1

*PRISMA diagram*




*From*: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71.

## Results

The 10 articles included in the review were case studies, some of which included experimental sections. Six of the patients were female and four male. Ages were 16-87 ($M$ = 46.7; $SD$ = 25.2).

Eight patients had lesions indicated by scanning. Three of the eight had more than a single lesion, one having as many as seven (see more in the summary of Garcha et al. 2018 below and Table 1). Two showed no lesions, though one (Walsh et al., 2017) had enlarged bilateral ventricles, which is known to cause neurological and neuropsychiatric abnormalities in some cases.

Several patients had some level of global cerebral atrophy. The onset mechanism varied from totally unknown to seizures and complications from disease. All patients suffered delusions concerning a loved one, save one whose delusion centred on two hospital staff members who cared for her during her hospital stay. The majority of delusions encompassed more than a single person or object, most common being spouses and close family members. Three patients had delusions concerning a physical location: a hospital, their home, and two countries. The subject of the delusion is typically thought to be perceived as hostile and dangerous (see, e.g., Barrelle & Luauté, 2018, p. 35 and Salvatore et al., 2014, p. 262). While that holds true for some of the included studies, it is far from the rule. Often this point is not addressed, or the interactions are described as simply confusing or strange. Luca et al. (2013) report on a case where there had been tension building between the patient and the subject of the delusion prior to the onset of Capgras symptoms, but otherwise no reports of a pre-existing adverse relationships were found.

The duration of delusions varied from a couple of days to several years. None of the cases that spanned more than a year noted if the delusion was ever resolved.

Patients' neuropsychiatric evaluations varied greatly. Some articles did not note if any evaluation was done or simply stated that an evaluation was done but did not specify which tests were used, which the patients passed with varying degrees of success. Others received full batteries (see Thiel et al., 2014). While we excluded cases where there was an explicit diagnosis of a psychiatric condition, some studies included patients who showed signs of possible diagnoses (see, e.g., Luca et al., 2013).

Most lesions were in the right hemisphere, which was expected, as was the presence of frontal lobe lesions. Frontal, parietal, parieto-occipital, occipital and temporal lesions were found, consistent with prior systematic reviews on delusional misidentification syndromes. Subcortical lesions aren't common in Capgras but have been reported (Darby & Prasad, 2016), so the lesions to the thalamus and semioval centers (the main regions of white matter located beneath the cortex projecting to and from most brain areas) were unexpected.

## Summary of articles

### *Brighetti et al., 2007*

Brighetti et al. (2007) report on a 22-year-old woman with no previous psychiatric diagnosis who, following a brief delusional episode where she could not recognize her own handwriting and was left shocked and catatonic, developed Capgras delusion mostly concerning her father, though for a two-month period it involved her mother, uncle, maternal grandfather, and paternal grandparents as well, all of which was resolved with antipsychotics except for the delusion concerning her father, who she believed to be an imposter who had conspired with her mother to kill her real father.

Computed tomography (CT) showed no lesions or abnormalities. Skin conductance recording and visual path scanning tests were performed while viewing photographs of familiar and unfamiliar faces, showing no differentiating results compared to eight age-matched, healthy controls subjected to the same kind of stimuli.

The authors believe that, based on their findings, trouble identifying faces leads one to explore the face more fully, which might help explain Capgras symptoms. They note that facial recognition leads to more focus on the eye region and that recognition of familiar faces coupled with low autonomic response leads to gaze avoidance. The authors do not note if the delusional belief concerning the father was eventually resolved.

**Table 1.**
*Summary of extracted data*

| Author | Age | Gender | Lesion Location(s) | Neuroimaging method | Other tests | Neuropsychiatric test | Neuropsychiatric evaluation | Imposter | Duration | Perceived hostility |
|---|---|---|---|---|---|---|---|---|---|---|
| Brighetti et al. 2007 | 22 | female | none | CT | SCR, Visual scanning path eye gaze test | WAIS-R (verbal and performance), structured interview according with DSM-IV, Iowa Gambling Task | normal; minor deficit in decision making | father (persistent); mother, uncle, maternal grandfather, and paternal grandparents (brief) | two years+ | hostile to neutral |
| Crichton & Lewis, 1990 | 30 | male | bilateral parietal | CT | EEG | WAIS-R (verbal and performance), mental state exam | normal; anxious, perplexed, distractible, paranoid | two colleagues, mother, hospital | seven days | the two colleagues: hostile, mother not noted |
| Dejode et al. 2001 | 24 | female | right parieto-occipital | CT | EEG, unspecified neurological exam | not noted | normal | the intensive care unit physicians | a few days | neutral |
| Ferguson et al. 2017 | 16 | male | left frontal | MRI | focal neurological and laboratory evaluation | not noted | one year history of agitation, social isolation, declining function, delusional beliefs and lack of self-care | parents | not noted | hostile |
| Garcha et al. 2018 | 65 | female | right frontal, right insular, right occipital, left parieto-occipital, right frontal, right parietal, right temporal | CT, MRI | not noted | unspecified mental state examination | mild confusion, right-sided visual field cut, left-sided neglect | husband | four weeks | friendly |
| Hirstein & Ramachandran, 1997 | 30 | male | none | CT | gaze direction, accuracy of face discrimination, perception of emotions in others, SCR | mental status exam (three objects, orientation in time and space, serial sevens, writing) | normal | father, mother, grandfather, self, countries | two years+ | friendly |
| Luca et al. 2013 | 53 | female | bilateral frontal, | MRI | EEG | MMSE, | test may indicate an unspecified personality disorder, | eldest daughter | one year+ | hostile, but not dangerous |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | bilateral semioval centers | | | DSM-IV Axis II Personality Disorder | paranoia, OCD traits, no diagnosis made | | | |
| Nuara et al. 2020 | 87 | male | right temporal | MRI, FDG-PET | Thermal IR imaging, SCR (visual and auditory modality) | MMSE, CDT, 3O3P, recognition of other's emotions, modified BFR, identification of famous faces | normal | son | one year+ | neutral, but suspicious |
| Peña-Salazar et al. 2014 | 70 | female | left frontal, left thalamus | CT, SPECT | diagnostic tests (biochemistry, viral serology, ultrasonography, urine toxicology) | unspecified neuropsychology examination | normal (with anxious and depressive symptoms) minor verbal and executive amnesia compatible with slight cognitive deterioration | husband, children | 10 days | not noted |
| Thiel et al. 2014 | 70 | female | right frontal | MRI, fMRI | SCR | MWT-B, BFR, EKF-S, CANTAB, CERAD, TEKT | mostly normal, diminished planning ability, word recognition, trouble recognizing famous faces | husband, home | three years+ | not noted |

*Note. The plus-sign (+) in the "duration" column refers to the authors not noting if the syndrome ever went away.*

*3O3P: Three-Objects-Three-Places test, BFR: Benton facial recognition test, CDT: Clock drawing method, CERAD: Consortium to Establish a Registry of Alzheimer's Disease, DSM-IV: Diagnostic and Statistical Manual of Mental Disorders fourth edition, EEG: Electroencephalogram, EKF-S: Emotionale-Kompetenz-Fragebogen [Emotional competency questionnaire], MMSE: Mini-mental state exam, MWT-B: Multiple choice word test, TEKT: Testbatterie zur Erfassung von Konfabulationstendenzen [Test battery for assessment of confabulation tendencies], WAIS-R: Wechsler Adult Intelligence Scale – Revised.*

**Table 2**
*Lesion locations across all studies*

| Lesion Locations | N = 16[1] |
|---|---|
| Hemisphere | |
| Right | 9 (56%) |
| Left | 4 (25%) |
| Bilateral | 3 (19%) |
| Region | |
| Frontal | 6 (38%) |
| Parietal | 2 (13%) |
| Parieto-occipital | 2 (13%) |
| Temporal | 2 (13%) |
| Insular | 1 (6.3%) |
| Occipital | 1 (6.3%) |
| Semioval centers | 1 (6.3%) |
| Thalamus | 1 (6.3%) |

[1] n (%)

### *Crichton & Lewis, 1990*

Crichton and Lewis (1990) report on a 30-year-old man with no previous psychiatric diagnosis who displayed Capgras syndrome after being hospitalized due to AIDS complications. The Capgras symptoms developed six days into the stay, following a flu-like illness. He also developed visual and auditory hallucinations during this time. The Capgras delusion encompassed his mother (whom he noted looked like his mother, but was convinced wasn't), two colleagues who came to visit (replaced by alien doppelgangers), and the physical hospital building (a garage masquerading as a hospital). The patient became deeply paranoid thinking the "aliens" were there to abduct him. Besides his mother, no other family members or close friends were included in the delusion.

CT scan two weeks after the psychotic symptoms began showed a slight increase in width of the sulci over the vertex and a small area of decreased density in the right upper medial parietal region. Nine days later, he was scanned again. The new scan showed that the widening was still present, but there was no evidence of decreased density in the parietal region.

The authors note that right-hemisphere damage is typical in cases of Capgras; it could be that the symptoms were caused by a combination of the transient right parietal lesion disturbing visual and memory processing with neuropsychiatric complications due to

AIDS. The Capgras syndrome started to diminish seven days post delusional onset.

### *Dejode et al., 2001*

Dejode et al. (2001) report on a 24-year-old woman who, due to severe adult respiratory distress syndrome complicating existing pneumococcal pneumonia, was admitted to the emergency room. Because of hypoxemia (low blood-oxygen level), the woman was intubated and ventilated. On the $12^{th}$ day, doctors performed reconstruction of the carotid artery with an internal saphenous vein graft. On the $22^{nd}$ day, the patient progressively regained normal consciousness as doctors began reducing sedation.

Neurological examinations showed normal results at this time. The woman had no history of psychiatric disorder. On day 29 following admission, her right lower limb had epileptic fits with myoclonic movements, and she experienced optical illusions with permanent distortion of objects. She was diagnosed with Capgras syndrome because she stated that, while some of the intensive care unit physicians were real, others were fakes: i.e., they had been replaced by identical looking imposter. She otherwise understood that what she was experiencing was abnormal and gave no indications of experiencing hallucinations.

CT scanning revealed a watershed cerebral infarct (a type of stroke) between the right posterior and right middle cerebral arterial areas (parieto-occipital area). The delusion disappeared after a few days, around the same time that the epileptic fits improved. The woman received anticonvulsant therapy, which continued after she was discharged. The authors conclude that Capgras may have arisen due to lesions in the right cerebral hemisphere.

### *Ferguson et al., 2017*

Ferguson et al. (2017) report on a 16-year-old man with a twelve-month history of social isolation, declining cognitive function, delusional beliefs, and lack of self-care. The patient was admitted to the hospital after making a police report and claiming that his parents were not his real parents.

Examination resulted in no focal neurological findings. MRI revealed a lesion in the teen's left frontal periventricular white matter along the frontal horn of the left ventricle projecting to the middle and inferior frontal gyri. The authors hypothesize that left retrosplenial and right frontal connectivity are affected by silent lesions and that the syndrome can be initiated via a separate left-hemisphere route. The authors do not note whether the delusion disappeared or remained.

### *Garcha et al., 2018*

Garcha et al. (2018) report on a 65-year-old woman who was admitted to hospital following a generalized tonic-clonic seizure. Two days after admission, the patient showed typical Capgras symptoms, believing her husband had been replaced by an identical double. She claimed that he only looked like her husband but instead was some close friend, but she could not identify who this friend was, referring to him only as the "other [husbands name]" (p. 96). The delusion vanished when her husband was not present, and she was listening only to his voice. It did not encompass any other family members or friends. CT scanning showed a thrombus (blood clot) in the proximal M2 segment of the right middle cerebral artery and subacute infarction within the watershed territory of the left middle and posterior cerebral arteries, indicating a recent stroke.

The next day, MRI showed acute infarctions in the right frontal, insular, occipital and occipitotemporal regions. A repeat of the earlier CT scan showed a new infarction in the right temporal lobe. A repeat MRI scan three days after the first showed new small infarctions in the right frontal, parietal and superior temporal lobe. The authors note that, since Capgras syndrome can take a while to manifest, it is hard to say whether the initial frontal lesion or the newer temporal lesions were mainly responsible. The authors do not explain how these lesions could cause the condition except by way of mentioning both the Darby et al. (2017) retrosplenial cortex connectome theory and Ellis and Young's (1990) mirror image of prosopagnosia theory. The delusion gradually started to diminish after three days and appeared to be gone after 10 days. A follow-up with the patient reported that she could recognize her husband four weeks post dismissal. She explained that she was suspicious of her husband during this time, but her family helped convince her of her husband's identity.

### *Hirstein & Ramachandran, 1997*

Hirstein and Ramachandran (1997) report on a 30-year-old man who, due to a traffic accident, had a right parietal fracture and was in coma for three weeks. The patient recovered his cognitive skills quickly upon waking but began to regard his grandfather and his parents – mostly his father – as imposters. His delusion was only present when interacting with these people directly and not when talking to them over the phone. The delusion also involved old pictures of himself, where he would claim that it was someone identical to him but not him, as the man in the picture had a moustache that he did not have. It did not, however, involve his reflection in the mirror. He would make strange claims such as that a check that had been sent to him was sent "to the other [patient]" (p. 438) or that his hair was a wig (presumably because his scalp did not feel familiar given the scars from his surgery). The delusion encompassed countries but to a much smaller extent: he claimed there were two Panama's and two USAs. A mental-status exam revealed no evidence of dementia or deficit in higher cognitive function.

CT scanning revealed bilateral enlargement of the ventricles, especially the right. The scan also revealed frontal bilateral areas of encephalomalacia (softening or degeneration of brain tissue). When the accuracy of the patient's judgment of gaze direction was tested, he received a lower result than controls in every condition. The patient claimed that more than one model took part in the pictures used for the eye-gaze experiment, which was not the case. Skin conductance was recorded while the patient viewed familiar vs. unfamiliar faces. The patient showed decreased autonomic response to familiar faces compared to controls.

The authors propose that, when the limbic system and the areas of ventral stream processing in the temporal lobe processing fail to communicate, Capgras syndrome results.

### *Luca et al., 2013*

Luca et al. (2013) report on a 53-year-old woman with no previous psychiatric diagnosis. She began suffering from Capgras syndrome twelve months prior to admission, with no clear triggering event. The delusion centered on her eldest daughter, with whom she always had had a fraught relationship. The delusion was persistent over the phone as well as face to face. She believed that the fake daughter was disingenuous and outright deceitful, and she felt disgusted by the daughter. Magnetic resonance imaging showed moderate-sized lesions in frontal subcortical white matter and bilaterally in the semioval centers.

The patient's disposition and behaviour were suggestive of obsessive-compulsive disorder. The authors suggest that this could indicate a link between obsessive compulsive disorder and Capgras syndrome stemming from a known association between obsessive fears and pre-existing cognitive deficits, likely caused in this case by the lesions in the semioval centers. They offer a psychodynamic explanation as well: the patient and her daughter had always had a troubled relationship; by creating a double of the daughter, the patient could hate her openly. The authors do not note if or when the delusion was resolved.

### *Nuara et al., 2020*

Nuara et al. (2020) report on an 87-year-old man with no previous psychiatric diagnosis. The patient developed a delusion regarding his son, whom he believed to have been replaced by an imposter. This only occurred when looking at his son face to face and not when hearing his voice or viewing photos of him.

A six cm diameter arachnoid cyst in his right temporal lobe was discovered by chance following a minor head trauma four years prior. In the right temporal lobe, the regions next to the cyst and the bilateral frontal lobes showed hypometabolism one month after the observation, when a fluorodeoxyglucose positron emission tomography (FDG-PET) scan was performed. Due to global brain atrophy, the patient had mild bilateral ventricular enlargement. Results from his neurological examination were normal, though mild hearing loss was noted. A few days after the delusion's onset, the patient's mood and behaviour calmed, but the patient was still suspicious towards his son.

The patient could recognize others' emotions, discriminate faces, and identify famous faces. Skin conductance recordings were made simultaneously as thermal infrared imaging. The patient had higher skin conductance results when viewing pictures of his daughter in comparison to his son and strangers. When the test was made but with voices instead of pictures, he received higher skin conductance on both his daughter and his son compared to strangers. The authors conclude that the delusion occurred because of an impairment of visuo-limbic interplay and reduced metabolism of the frontal areas.

### *Peña-Salazar et al., 2014*

Peña-Salazar et al. (2014) report on a 70-year-old woman with no history of psychiatric care. She had high cholesterol and made her first visit to a primary physician due to anxious and depressive symptoms. She was unsuccessfully treated with escitalopram. After two months, she had high anxiety, selective mutism, and strange on a visit to the emergency room, and she made strange responses to questions. According to her, her children had been replaced by imposters and she was a widow, even though her family was present in the emergency room. Diagnostic tests were made and revealed no pathological findings.

A small abnormal lacunar image was revealed by cranial CT in her left thalamus as well as discrete cortical atrophy and left frontal subcortical hypodensity, which all could have resulted from a cerebrovascular accident three to six months prior. Hypofunction in the left thalamic region was indicated by single-photon emission computed tomography (SPECT).

The woman was given antipsychotic, antidepressant, and anxiolytic therapy, which she responded to favourably. After five days, the woman's hallucinations and insomnia disappeared, and her anxiety sharply decreased. Ten days later, she began to accept her family, an improvement that continued after she was discharged. Neuropsychological examination made in outpatient care revealed slight cognitive deterioration and minor verbal and executive amnesia. The authors explain the case as caused in part by a disconnection between the frontal lobe on the one hand and the temporal and limbic regions on the other.

### *Thiel et al., 2014*

Thiel et al. (2014) report on a 70-year-old woman with unknown psychiatric history. The patient suffered a right frontal intracerebral haemorrhage three years prior to admission. During recovery, she developed spatial disorientation and Capgras syndrome encompassing her husband and her home. The patient did not appear to feel threatened by the husband but was persistent in the belief that it wasn't the man she married. She frequently mentioned to the replacement husband that she wanted "to go home" despite being home.

MRI scan showed a large right prefrontal lesion leaving only the ventromedial and medial orbitofrontal cortex unharmed. Functional magnetic resonance imaging (fMRI) was collected during facial recognition tasks of familiar and unfamiliar faces and compared to 13 older controls. The results show that her facial recognition was not diminished but, while looking at a picture of her husband, there was differentiating activity in the left posterior cingulate cortex and left posterior superior temporal sulcus: two regions associated with the extended facial processing system, which infers others' thoughts and feelings. Skin conductance recording was performed, but the data were inconclusive due to high variance in the control group.

The authors believe that two factors are necessary for Capgras. They suggest their findings to indicate that the first factor is diminished activity in the extended facial processing system in the left hemisphere. The second factor, in line with Langdon & Coltheart (2000), is diminished belief evaluation due to disruptions in right frontal areas. The authors do not note if or when the delusion was resolved.

## Discussion

Our review set out to further enhance understanding of the neural mechanisms of Capgras syndrome by investigating the different kinds of brain lesions that can lead to the condition. From our review, it is evident that Capgras syndrome is a complicated condition that cannot be explained by a single lesion location. We did find a bias towards the right hemisphere and frontal regions, consistent with prior research on delusional misidentification syndromes (Darby & Prasad, 2016) and delusional disorders more generally (Coltheart, 2010). This bias indicates that the condition requires damage to belief-evaluation areas within the right frontal lobe. In most cases, it is unclear exactly which subsections within the lobes were damaged, though Thiel et al. (2014) mention that the ventromedial and medial orbitofrontal cortex survived the stroke; the accompanying fMRI analysis revealed that the left superior temporal lobe showed lower than expected activation when the patient viewed her husband's face.

Nuara et al. (2020) report damage to the left superior temporal lobe, and Garcha et al. (2018) report damage to the right-hemisphere equivalent, indicating that damage to the extended facial processing system could play a part in the condition, which is expected since several studies note that the delusion is only present while viewing the imposter's face (see, e.g., Hirstein & Ramachandran, 1997; Nuara et al., 2020). Most of the studies do not note this detail though, limiting the ability to draw a conclusion. Others note that the delusion was still present over the phone (Luca et al., 2013), but this could be due to a strongly held belief that the identity had been duplicated, or a strong mental representation of the imposter's face activating the facial processing systems. Previous research has shown that visualising faces activates facial recognition areas, although it is unclear if this activation extends to the extended facial processing system (see, e.g., Burton et al., 2011). Activation of facial recognition areas by means other than viewing the face might explain how Capgras has been found in blind people, as the primary facial processing area still shows face selectivity during tactile stimulation (Murty et al., 2020). An interesting way to test this would be by using modern functional neuroimaging techniques while patients suffering from Capgras listen to familiar voices or by testing patients' abilities to produce vivid mental imagery.

As said, Darby et al. (2017) offer damage to belief-evaluation systems and the functional connectivity of the retrosplenial cortex as the most likely explanation for delusional misidentification syndromes, with a focus not on facial processing but on autobiographical memory. As their study is not focused specifically on Capgras, it is not directly comparable but could prove insightful. Comparison of the available scans and the figures highlighting lesion locations show that three of the included studies (Dejode et al., 2001; Garcha et al., 2018; Nuara et al., 2020) found lesions in comparable areas, while Thiel et al. (2014) found a general overlap, as it the lesion spans most of the right frontal lobe. Garcha et al. (2018) did not include scans of all lesions they identified; but, of the ones included (four of seven), two showed location overlaps: the right insular and right temporal lesions. Note that this comparison was done by hand. It is possible that some areas overlap in terms of functional connectivity, limiting the ability to draw conclusions. The Ferguson et al. (2017) lesion is in white matter that projects to the middle and inferior frontal gyri, which do not have a significant connection to the retrosplenial cortex.

Either the available information on lesion locations in the studies is not clear or specific enough to support the Darby et al. (2017) hypothesis – i.e., location is only recorded by lobe or general brain region (see, e.g., Crichton & Lewis, 1990, for simply "bilateral parietal") – or the hypothesis might need to be modified to handle the increasing variety of lesions found in Capgras patients – for example, the white matter lesion found by Ferguson et al. (2017) – by removing the claim that a single lesion interrupting the functional connectivity of the retrosplenial cortex is enough to cause Capgras. Ferguson et al. (2017) make the interesting point that there might be lesions beneath the resolution of current neuroimaging techniques that play a key role in conditions like Capgras – such as in the case of Bobes et al. (2016), where a Capgras patient had reduced density in the left inferior fronto-occipital fasciculus tract (a white matter tract connected to goal-oriented behaviour) without any visible lesions.

The patients with only a single lesion (Crichton & Lewis, 1990; Dejode et al., 2001; Ferguson et al., 2017; Nuara et al., 2020; Thiel et al., 2014) do not reveal overlap in lesion location (bilateral parietal, left frontal, right temporal, and right frontal respectively), indicating something more at play. The field is predominantly in agreement that Capgras syndrome requires damage to belief-evaluation systems in the right frontal lobe (see, e.g., Darby & Prasad, 2016; Kakegawa et al., 2020). Hard-to-find lesions in the frontal lobe in conjunction with damaged autobiographical or facial-processing areas could go a long way toward explaining the varied lesion locations in Capgras patients; but this is an unsatisfying conclusion.

We agree that facial processing plays a key role in Capgras syndrome, but the presence of duplicated locations and inanimate objects in some cases makes this difficult to accept unconditionally. The three cases that had delusions about something other than only people (countries, the hospital, their own home) had varying lesion locations (none, bilateral parietal, and right frontal, respectively). In the case of countries, bilateral ventricle enlargement was present with the right ventricle especially large, which could indicate some right frontal hypofunction; but this is speculative.

Examination of the two cases with no identified lesions reveals no phenomenological similarities, save that they both had brief episodes where their delusions encompassed multiple individuals before stabilizing onto a single subject (Hirstein & Ramachandran, 1997; Brighetti et al., 2007). In all other cases, the subjects of the delusions were consistent.

Capgras has occasionally been linked to violence. We found no report of violence in our review, where we deliberately excluded individuals with prior psychiatric diagnosis. This seems to support the possibility that, while Capgras can exacerbate violent urges, violent tendencies more likely stem from comorbidities such as schizoaffective disorder (Bourget & Whitehurst, 2004).

**Limitations**

A major limitation is that there is no official diagnosis for Capgras (see, e.g., Klein & Hirachan, 2014), which means that it's up to the individual doctors/researchers to evaluate if the patient's symptoms align with whatever the professional's definition of Capgras is. In clinical settings, Capgras can overlap with other delusional misidentification syndromes in its delusional contents, such as duplicated places (Thiel et al., 2014), which typically is referred to as reduplicative paramnesia, though some treat reduplicative paramnesia as its own misidentification syndrome, others as a feature of misidentification syndromes in more general terms: see, e.g., Ventriglio et al. (2020) where reduplicative paramnesia is first brought up as a distinct syndrome, then again as a feature of the Frégoli delusion, though not by name. The variety of delusional contents surrounding Capgras could have to do with a lack of awareness of the different delusional misidentification syndromes. Capgras has a diverse aetiology, associated with both neuropsychiatric and neurological disorders, which complicates its identification in clinical settings even more, highlighting the need for strict diagnostic criteria.

When reading Capgras case studies, it quickly becomes evident that there is no standardized testing methodology. Six out of 10 cases used CT and five used MRI (see Table 1). MRI is considered to have clear advantages over CT when evaluating closed-head trauma (see, e.g., Orrison et al., 1994) and would therefore be the better method to use when studying patients with Capgras. SCR was used in four out of 10 cases. However, the data recorded in one study (Thiel et al., 2014) was so variable in the control group, due to the age of the participants, that it was impossible for the experimenters to compare the control group's results with the patient, which may suggest that the method is not ideal when studying elderly patients with Capgras. In four cases, a neuropsychiatric evaluation was mentioned but unspecified (Garcha et al., 2018; Peña-Salazar et al., 2014) or not even noted (Dejode et al., 2001; Ferguson et al., 2017).

A limitation of our own making is that we did not decide what definition of Capgras we would use in our inclusion criteria, instead relying on the doctor's/researcher's own diagnosis. Since Capgras and Reboul-Lachaux (1923) reported the first case of Capgras syndrome, the classic definition has been a conviction that typically but not exclusively, a family member or close friend has been replaced by an identical imposter. Some extend the definition to include pets, inanimate objects, locations (Barrelle & Luatué, 2017, p. 37), and countries (Hirstein & Ramachandran, 1997). We included two cases in our review that had a delusion about both a close family member or friend and a location (Crichton & Lewis, 1990; Thiel et al., 2014), one case where the patient had a delusion concerning her intensive care unit physicians (Dejode et al., 2001), and one where the patient believed that he, himself, had been duplicated, but only when viewing a photo (Hirstein & Ramachandran, 1997). The places might fit better as a separate reduplicative paramnesia diagnosis. By opting for narrower inclusion criteria, we could have eliminated some confusion surrounding the subject.

Even though our study found a bias towards right frontal regions, it was limited by the variable quality of the case reports and their occasional lack of information, such as which neuropsychiatric tests were conducted, along with the fact that some lesions could have been missed due to the relative weakness of CT scanning.

## Ethical and societal aspects

Three of the case studies had experimental elements (Hirstein & Ramachandran, 1997; Brighetti et al., 2007; Thiel et al., 2014), but only one made reference to any kind of informed consent (Thiel et al., 2014), with the disclaimer that the trials were conducted according to the Declaration of Helsinki, regarding the ethical principles of conducting experiments on humans (World Medical Association, 2022). It is worth noting that all of the neuroimaging used in the studies was non-invasive.

Prior research has mostly focused on delusional misidentification syndromes in general, not solely on Capgras syndrome. Results from this review could provide meaningful information by showing the complexity of the delusion. Awareness of Capgras patients' underlying condition would support psychiatrists and aid in understanding patients with other disorders where beliefs play an important role, such as schizophrenia.

## Future research

Future studies of Capgras syndrome would benefit greatly from an official DSM-V set of diagnostic criteria. Standardized testing and data-collection methodologies would aid in comparison of cases and allow more credible analysis. A clear standardized testing methodology, with MRI as the primary brain-imaging technique, should be followed when one suspects Capgras. More detailed information about the patient's delusions could provide insights into the nature of Capgras. For instance, if the delusion's duration was consistently noted in case reports, it could help with finding correlations with other factors, such as lesion location or neuropsychiatric evaluation. As there is no standardized method for evaluating delusional severity, Darby and Prasad (2016) have proposed using delusion duration as a proxy.

## Conclusion

Investigating the location of brain lesions in Capgras patients reveals that the condition cannot be explained by a single lesion location. Consistent with prior research, a bias towards frontal regions, especially the right hemisphere, was found, but lesions were ultimately found in a variety of cortical and subcortical regions. While Capgras has been observed in blind people, it still seems likely that the condition is tied to diminished facial processing coupled with a deficit in belief evaluation.

Understanding the nature of this complex syndrome could help shed light on how beliefs and delusions are formed, which would aid a variety of fields, providing a basis for psychiatric treatments and deepening understanding of the brain. It could give insights into the emotional connections required by facial processing. Studying relatively exotic conditions like Capgras allows a continued advance in understanding the human condition.

Final word count: ***7359***

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