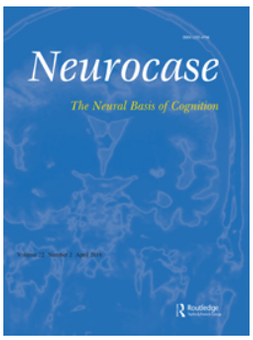

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"Cat-gras" delusion: a unique misidentification syndrome and a novel explanation

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ABSRACT

Capgras syndrome is a distressing delusion found in a variety of neurological and psychiatric diseases where a patient believes that a family member, friend, or loved one has been replaced by an imposter. Patients recognize the physical resemblance of a familiar acquaintance but feel that the identity of that person is no longer the same. Here we describe a 73-year-old male with right posterior frontal and bilateral anterior-medial frontal damage from prior brain trauma with a similar delusion of an imposter replacing his pet cat. Misidentification syndromes for animals, as opposed to humans, have been rarely reported. Neuropsychological testing showed deficits in executive processing and memory retrieval with prominent intrusions and false positive responses. The delusional belief content in Capgras syndrome has been hypothesized to result from loss of an emotional or autonomic response to familiar stimuli, from theory of mind deficits, or from loss of self-environment distinctions. We instead propose that Capgras delusions result from a dysfunction in linking external stimuli with retrieved internal autobiographical memories pertaining to that object. This leads to an erroneously learned identity that persists as a specific delusional belief.

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KEYWORDS
Delusional misidentification syndrome; Capgras; confabulation; episodic memory; identity

Introduction

Capgras syndrome is a misidentification syndrome characterized by delusions that a familiar person has been duplicated and replaced by an imposter. Capgras syndrome is part of a larger group of delusional misidentification syndromes (DMS) that include Fregoli syndrome, intermetamorphosis, the syndrome of subjective doubles, and reduplicative paramnesia. First described by Capgras and Reboul–Lachaux in 1923, Capgras delusions have been found in patients with both primary neurological and psychiatric diseases. Here we describe a patient who presented with a delusion of an imposter replacing his pet cat.

Case

A 71-year-old right-handed male was presented to the neurobehavioral clinic for evaluation of memory impairment and functional decline. He had a history of former heavy alcohol use, obstructive sleep apnea, atrial fibrillation, and a pacemaker placement for sick sinus syndrome. He reported repeated head traumas from playing professional hockey, as well as a traumatic right subdural hematoma (SDH) requiring surgical evacuation.

His neurobehavioral symptoms started 15 years prior to presentation when he was forced to retire from his job as an auditor due to an aggressive outburst against his co-workers. This led to a psychiatric hospitalization at which point he was diagnosed with bipolar disorder. For the next 15 years, he had episodes of manic behavior, which included excessive spending ($40,000 in 1 month on golf-related paraphernalia and a car), hoarding behavior (filling the spare bedroom with old magazines and electronics), and sexual promiscuity (pursuing young women who worked at the rehabilitation facility he attended). These were interspersed with episodes of apathy, social withdrawal, and lack of personal hygiene. His wife additionally noted a 7–8 year history of memory impairment. He would misplace things, such as his glasses, car keys, and wallet, and occasionally forget details of conversations.

Six years prior to presentation, he became acutely paranoid in the setting of stopping his psychiatric medications. He passed his wife the written notes stating that their house was being monitored, and often mistook persons in parking lots for Federal Bureau of Investigation agents. He then became obsessed with the idea that his pet cat had been replaced by an imposter cat that was involved in the conspiracy against him. He knew that the current cat resembled his pet cat physically, but that the personality or psychic core of his cat had been replaced. His symptoms improved with medications and he has had no further delusions of imposters replacing his cat.

His neurological exam was notable for poor eye contact and frequently relying on his wife to answer questions, but otherwise normal affect and linear thought process. He was fully oriented. Executive function was impaired; he could not perform serial 7's after 93, and after stalling, started repeating the days of the week backwards (a previous task). He skipped April when naming the months of the year backwards. Speech was fluent, with normal naming, repetition, and comprehension. Short-term recall for verbal words was 2/5 at 5 minutes and improved to 4/5 with semantic cues, and 5/5 with multiple choices, although he initially had three false answers (often words from other parts of the cognitive testing session).

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Table 1. Neuropsychological test results.

NEUROPSYCHOLOGICAL EXAMINATION		Raw	%	Comments	Classification
Estimated Premorbid Intelligence					
WTAR		27	30		Average
	Verbal IQ	94			
	Performance IQ	95			
	Full Scale IQ	94			
Visuospatial Function					
WAIS-IV	Block Design	20	16		Low Average
ACE-R	Visuospatial	12/16	2		***Below Cutoff***
Attention & Executive Functions					
WAIS-IV	Digit Span Forward	8	25		Average
	Digit Span Backward	7	37		Average
	Digit Span Sequencing	5	16		Low Average
Controlled Oral Word Association	FAS	18	2		Borderline
	Animals	7	<1		***Impaired***
	Vegetables	6	2		Borderline
	Total Set-Loss Errors	4			
	Total Repetition Errors	0			
Trails	Part A	65	4		Borderline
	Part B			D/C at "4' with 4 errors after 180'"	***Abnormal***
Graphomotor series				Perseverative; kept patterns going in a second line without stopping. Had to be asked to d/c task.	***Abnormal***
Language					
Boston Naming Test		50	32		Average
	# Correct with Phonemic Cue	7			
WAIS-IV	Verbal Comprehension (prorated)	27	37		Average
	Similarities	22	37		Average
	Information	12	37		Average
Memory					
WMS-IV	Auditory Memory (LM)	12	10		Low Average
	Logical Memory I	24	16		Low Average
	Logical Memory II	7	5		Borderline
	Recognition	10/23			
Free and Cued Selective Reminding Test	Free Recall	16			***Below Cutoff***
	Free and Cued Recall	44			At Cutoff
Rey 15-Item	Immediate Recall	9/15			
	Recognition Hits	13/15			
	False Positives	14			***Abnormal***

ACER: Addenbrooke's Cognitive Exam- Revised; WAIS-IV: Wechsler Adult Intelligence Scale—4th ed.; WTAR: Weschler Test of Adult Reading; WMS-IV: Wechsler Memory Scale—4th ed.

He had difficulty copying a cube and on his clock, drawing circles on the numbers rather than drawing the hands.

Formal neuropsychological testing confirmed deficits in visuospatial processing, executive function, and memory retrieval, with frequent perseveration, intrusions, and false responses on memory tasks (see Table 1).

A CT scan showed diffuse generalized atrophy as well as focal right middle frontal gyrus encephalomalacia in the area of his prior SDH and craniotomy (Figure 1). A positron emission tracer (PET) scan showed evidence of hypometabolism in the anterior-medial frontal lobes bilaterally, as well as in the right posterior frontal lobe (Figure 1). He could not have an MRI due to his pacemaker.

Discussion

"Cat-gras" syndrome

Capgras delusions have rarely been reported with animals. Review of the literature reveals two cases reported in pet cats (Ehrt, 1999; Reid, Young, & Hellawell, 1993), two cases in pet birds (RöSLER, Holder, & Seifritz, 2001; Somerfield, 1999), and one in a pet dog (Wright, Mindham, & Burn, 1994). The majority of these cases occurred during a psychotic episode with other paranoid and persecutory delusions, as in our patient. However, our case is unique in three respects. First, reported cases in the literature have been in socially isolated patients without close friends or family members, which was not the case in our patient. Second, Capgras delusions for animals have been reported exclusively in psychiatric patients, while our patient's evidence of bilateral frontal and right frontoparietal injuries from prior brain trauma is consistent with those found in other DMS cases attributed to neurological disease (Devinsky, 2009). Finally, this is the first case to report neuropsychological testing in a patient with Capgras for pets, showing deficits in fronto-executive, visuospatial, and memory-retrieval with prominent intrusions and false answers. Taken together, our patient's delusion semiology, neuroimaging, and neuropsychological profile suggest a novel neuropsychological mechanism of DMS.

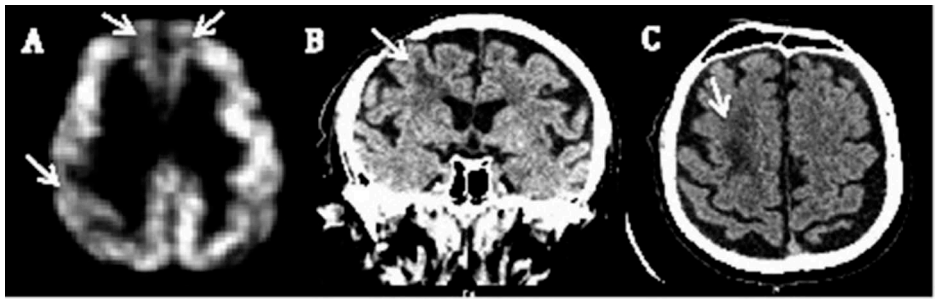

Figure 1. PET and CT findings.

(A) FDG-PET image showing hypometabolism in the bilateral anterior-medial frontal lobes and right posterior frontal lobe (arrows). (B) Coronal and (C) axial CT head images showing right middle frontal gyrus encephalomalacia from prior SDH.

Theoretical accounts of DMS

Most explanations of misidentification syndromes use Capgras delusions as a model. Ellis and Young proposed two parallel pathways for facial recognition, including an overt pathway involved in conscious recognition and a covert pathway involved in the feeling of recognizing familiar faces (Ellis & Young, 1990). The authors predicted that damage to the ventral visual pathway leads to absence of overt identification and prosopagnosia, and damage to a more dorsal visual pathway leads to absence of covert identification and Capgras delusion. This theory has been supported by experiments using Galvanic skin responses (GSR), a marker of autonomic arousal that is present in patients with prosopagnosia, despite lack of conscious facial identification (Tranel & Damasio, 1985), and absent in patients with Capgras, despite overt recognition (Ellis, Young, Quayle, & De Pauw, 1997; Hirstein & Ramachandran, 1997). Hirstein and Ramachandran argue for a conceptually similar explanation, although they hypothesize that covert recognition arises sequentially after overt object recognition rather than in parallel, due to a disconnection between object recognition areas in the ventral visual pathway and limbic areas involved in emotional representations or memories of an object (Hirstein & Ramachandran, 1997). However, supporting anatomical evidence for this theory is lacking, as patients with DMS are not typically found to have lesions in these areas (Devinsky, 2009; Feinberg, 2005). Such a hypothesis would also predict global hypoemotionality toward visual stimuli, which is not typically found in these patients, as well as purely visual misidentifications, which is not supported by patients developing the syndrome who are blind (Reid et al., 1993).

More importantly, the validity of using GSR as a marker of the type of familiarity important in DMS has been challenged, given that GSR responses in prosopagnosia patients are not associated with a conscious percept of familiarity (Young, 2009). Indeed, more recent evidence suggests that changes in GSR are heterogeneous and can be differentiated into short-latency and long-latency responses, with short-latency responses seen in prosopagnosia patients corresponding to a repeated exposure effect in primary visual areas, while long-latency responses, corresponding to conscious familiarity determinations due to cognitive resources devoted to object recognition, remain absent(Morris, Cleary, & Still, 2008). The short-latency GSR may lead to a preference for familiar faces in prosopagnosia patients (Greve & Bauer, 1990) and to a general, positive affective mood in response to familiar objects (Stone & Valentine, 2003), but not to the conscious feeling of familiarity.

Based in part on this evidence, Young proposes that in Capgras delusion, there is a conflict that arises between the currently perceived person and the remembered person. According to this model, because the "remembered person" has a short GSR response while the perceived person does not, a delusional patient therefore "prefers" the remembered person and forms the belief that the perceived person is an imposter (Young, 2008, 2009). There are several conceptual problems with this formulation. First, many familiar objects have a negative affective association (e.g., to those who have repeatedly caused harm), and in such cases, it is unclear how an autonomic response would lead to a "preference" for a perceived versus. remembered stimuli. Furthermore, normal individuals who experience an emotional discrepancy between a perceived and remembered object, such as during an argument with an otherwise close friend, do not therefore believe that their friend is an imposter or different person. Finally, this theory is still based on the proposition that abnormal autonomic responses are primarily responsible for the delusional belief content in DMS. However, patients with damage to the ventromedial prefrontal cortex (vmPFC) do not have an autonomic response to familiar persons, but nevertheless do not develop DMS(Tranel, Damasio, & Damasio, 1995), while some patients with DMS have normal GSR when viewing pictures of the persons and places pertaining to their delusional misidentifications (Moser, Cohen, Malloy, Stone, & Rogg, 1998; Thiel, Studte, Hildebrandt, Huster, & Weerda, 2014), suggesting that altered GSR is not a universal finding in these disorders.

Hirstein proposed that Capgras delusion is instead the result of a disconnection between areas involved in theory of mind and emotional and memory processes in the limbic system, leading to impaired attributions of thoughts and beliefs to familiar persons (Hirstein, 2010). Such an explanation could easily be extended toward pet animals, which are perceived as having thoughts, beliefs, desires, and personalities by their owners. Extrapolating this to delusions involving familiar places is less intuitively plausible. While one might use this theory as a novel explanation to metaphors attributing personalities and psychic qualities to specific places or inanimate objects, such an explanation would remain highly

speculative, and likely unnecessary. Even so, several additional concerns would remain. This theory offers no explanation for why DMS involve personally significant persons, animals, and places. It is not clear how disorders of over relatedness, such as Fregoli syndrome, would fit into this framework. Finally, while some patients with Capgras delusions have been reported to have deficits in theory of mind tasks (Thiel et al., 2014), other patient groups with theory of mind deficits (autism spectrum disorder, frontotemporal dementia) do not develop DMS.

Finally, the egocentric disequilibrium theory (Feinberg, 2011) proposes that DMS result from abnormalities in determining the relatedness of objects in the environment (other persons, animals, and places) to the self. This framework is used to describe a range of disorders, including Capgras syndrome, Fregoli syndrome, other reduplicative paramnesias, and asomatagnosia, as disorders of "ego boundaries", where people, places, or objects with personal significance are erroneously judged to be overrelated or underrelated. However, it is not clear what an ego boundary or self-relational attributes are, or how they can expand or narrow to result in misattributions of over relatedness and under-relatedness. Ego-disequilibrium theory also does not explain the persistence of specific misidentifications over time, or the phenomena of reduplication, where multiple identities are believed to exist simultaneously.

Dysfunction in retrieved autobiographical memories: a novel hypothesis

A theoretical account of DMS should propose a common mechanism for all DMS syndromes given that: (1) DMS delusions can involve delusions in multiple objects domains simultaneously; (2) DMS delusions can involve feelings of hypo- and hyper-familiarity in the same patient; and (3) lesions in the same neuroanatomical locations can cause each of these different syndromes. It should also attempt to explain why delusions involve personally meaningful stimuli, why such delusions persist for specific objects over time, and the phenomena of reduplication found in many of these disorders. One such explanation is that DMS result from dissynchronization between stimulus-induced object recognition and retrieval of appropriate autobiographical memories, leading to a conflict between externally and internally generated beliefs about the identity of a particular object in the environment. This was the original intuition of Pick when he classified reduplicative paramnesia as a disordered memory state (Pick, 1903), as well as more recent hypotheses about abnormal memory retrieval in DMS (Staton, Brumback, & Wilson, 1982). Findings in our patient similarly suggest that memory impairment is an important contributing factor to DMS. We, therefore, propose a model where the delusional belief content in DMS results from dysfunctional linking between externally perceived objects and appropriately retrieved internal autobiographical memories associated with an object, leading to an erroneous learned belief that a familiar external object is a new, distinct entity (explaining the reduplication of physical bodies by the creation of duplicate, imposter identities), or that an internally generated memory is erroneously linked to an unfamiliar external object (leading to the reduplication of identities by the creation of new, disguised persons; Figure 2).

The first important point in our proposed model is that representations of identity involve autobiographical memories, and that dysfunction in linking these memories with external stimuli results in the delusional belief content in DMS. Autobiographical memory retrieval allows the brain to link the currently perceived stimuli with previous experiences, allowing an object to be perceived as the same, unique entity existing over time. In this sense, linking an external object with the appropriate internally remembered autobiographical

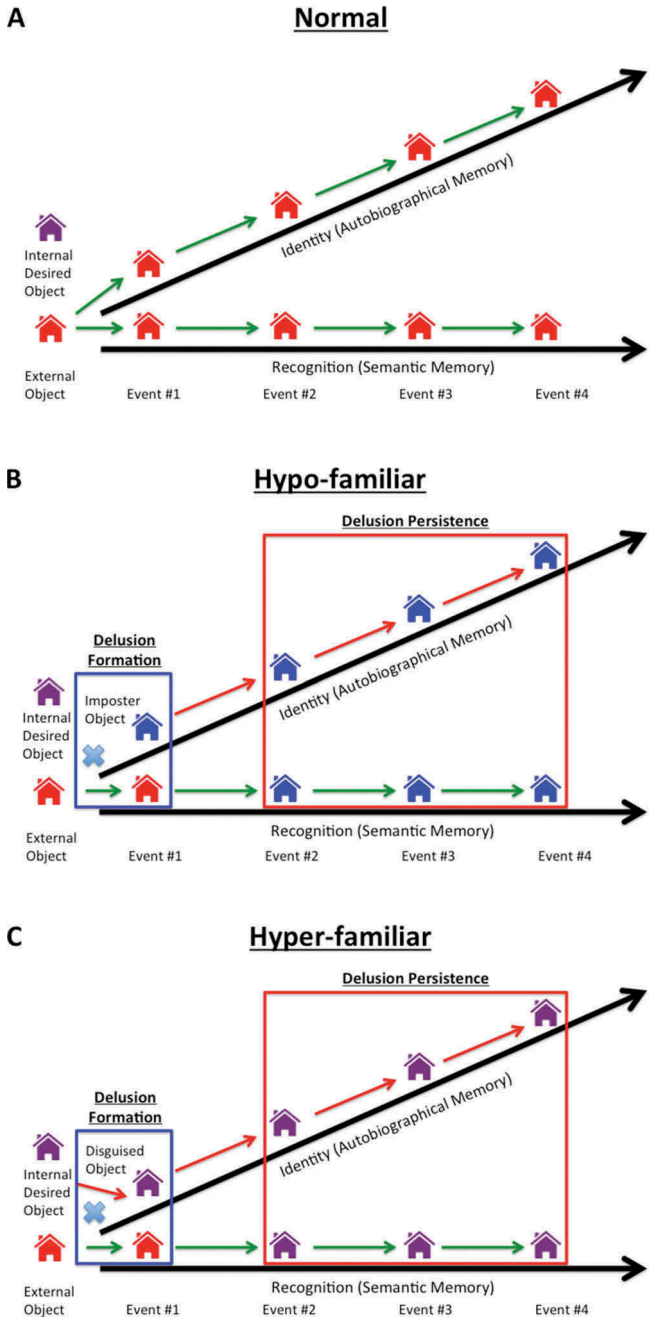

Figure 2. Proposed mechanism for DMS.

Proposed mechanism for (A) Normal perception of a personally familiar object; (B) DMS involving delusion of a personally familiar object being an imposter or replica; (C) DMS involving delusion of a familiar object in disguise. Note that reduplication occurs because of the creation of a new representation (impostor or disguised object).

memory establishes that object's identity. Identity, in this sense, would be similar to covert recognition in the Ellis and Young model, while overt object recognition would instead be part of the semantic memory system, hypothesized to involve person identity nodes (PIN's) located in the anterior temporal lobes (Collins & Olson, 2014). Our definition of identity would include persistent beliefs and personality traits of familiar persons (Hirstein, 2010), and because autobiographical memory is first-person, explain why DMS are self-relational (Feinberg, 2011). This definition also explains why personally salient persons, places, and animals, which have rich representations within autobiographical memory, are specifically misidentified in DMS.

The second specific point in our model is that delusional belief persistence and specificity result because the misidentification results in a new autobiographical memory based on this erroneously learned association. On subsequent encounters, it is the misperceived identity that is retrieved. While this belief can be rationally challenged (patients can often acknowledge their belief is irrational and sometimes momentarily admit it is therefore likely to be wrong), they are equally able to "reason" why the delusion is likely to be true (finding incidental differences, such as a table being in a slightly different orientation in a misidentified room or hair being differently parted in a misidentified person), leading to reinforcement of a delusional belief. This is similar to the mechanism of Capgras delusion proposed by Young, where there is an anomalous event of belief generation involving the "loss" of familiarity when this feeling should be suspected, but that the delusional belief persists due to the erroneously learned "lack" of expected familiarity to an imposter. It is also similar to a neurobiological model of delusional belief generation in psychiatric patients where dysfunction within the mesolimbic dopaminergic reward pathway leads to inappropriately associative learning resulting in the persistence of delusional beliefs (Corlett, Taylor, Wang, Fletcher, & Krystal, 2010). Others have proposed that a "second-hit" involving dysfunction in belief evaluation is necessary (Coltheart, 2007, 2010), which could contribute to both the erroneous belief generation as well as to the persistence of a delusional belief in our model.

In the case of hypo-familiar delusions such as Capgras, object recognition via the semantic memory system occurs, but this does not trigger retrieval of relevant autobiographical representations. This leads to the belief that the perceived person, physically similar to a loved one, is somehow a "new" or "different" person. The patient from Hirstein and Ramachandran, for example, perceived pictures of the same model with different head and eye orientations as "different" persons in each picture, though physically identical, consistent with our theory (W Hirstein & Ramachandran, 1997). According to the second step in our model, the brain would actually encode this misidentified person as a "new" identity within autobiographical memory, therefore leading to the persistence of this delusion on subsequent encounters where a loved one is identified as the imposter. This would also explain the reduplicative phenomena, where both the imposter and familiar person exist as distinct autobiographical memories.

Hyper-familiar delusions would result when external stimuli are unable to appropriately constrain autobiographical representations triggered by internal stimuli, such as a want or desire. This leads to an inappropriate linkage of the currently perceived stimulus with an identity triggered by these internal wishes. Again, delusions would persist for this specific linkage due to this encoding this as a newly learned association. Moreover, this model would be consistent with psychiatric and psychological observations that content of hyper-familiar delusions resemble primitive defense mechanisms such as "wish fulfillment," where a patient desires to be at home when they are in the hospital or to see a deceased loved one when they are ill and afraid (Feinberg, 2011). According to our model, such wishes or desires trigger internally generated retrievals from autobiographical memory and are then erroneously associated with inappropriate external objects in the environment, leading to the misidentification.

Conclusions

In summary, we present the case of a patient with a Capgras syndrome involving the delusion that his cat had been replaced by an imposter. This case demonstrates that DMS can extend beyond persons and places to include non-human animals. Neuropsychological testing showing executive dysfunction, memory-retrieval deficits, and confabulations suggest that these processes are important in DMS. Our theory proposes that the abnormal belief content in DMS occurs due to dysfunction in linking externally perceived stimuli and internally generated identity representations from autobiographical memory. An inability of the external object to trigger the appropriate retrieval of autobiographical memory would lead to the erroneous belief that the external object is an imposter or replica (leading to hypo-familiar delusions such as Capgras), while an inability of the external object to constrain inappropriately generated identities from autobiographical memory would lead to the erroneous belief that the external object is the familiar identity in disguise (leading to hyper-familiar delusions such as Fregoli). The personal nature of these delusions is explained by the fact that autobiographical memories extend to personally salient objects, including familiar persons, pet animals, and places. Importantly, these abnormal belief experiences are encoded as a new autobiographical memory, leading to the creation of a new imposter (e.g., Capgras) or disguised (e.g., Fregoli) identity. This "new" identity explains the phenomena of reduplication, the persistence of the delusional belief, and the specificity of the delusion to a particular misidentified person. While our theory will likely require refinement as new challenges from additional clinical observations emerge, it offers a comprehensive explanation for these fascinating disorders.

Disclosure statement

No potential conflict of interest was reported by the authors.

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