Capgras Delusion: How can we investigate and understand a delusion?

Richard Shillcock
To understand how to carry out research on something as odd as the Capgras Delusion
“… theories which the patients invent to account for their bodily sensations.” William James (1890)

“… a hypothesis designed to explain unusual perceptual phenomena.” Maher (1974)

Monothematic versus polythematic delusions.

Such delusions can occur in the absence of schizophrenia or any other psychosis.
What is the relevant *level of description*, at which to understand a delusion?

Originally, *psychodynamic approaches* were used exclusively.

These have been replaced by *neuro-level approaches*.

A *combination* of neuro- and psychological level approaches is necessary.
Capgras delusion

“That’s not my wife, it is an impostor who looks just like her”
Capgras delusion
Fregoli delusion

“I am constantly being followed by people I know, but I can’t recognize them because they are always in disguise.”
Cotard delusion

“I am dead.”
Mirrored-self misidentification

“The person I see when I look in the mirror isn’t me, it is some stranger who looks like me”
Somatoparaphrenia

“This limb isn’t mine, it is yours.”
De Clerambault’s delusion (erotomania)

“Person X is secretly in love with me”
(Person X being some important or famous person who has never encouraged this idea)
Othello syndrome

‘My wife is having an affair’
Skin conductance response (SCR)

Arousal of the sympathetic nervous system ("flight or fight") causes sweating and reduces conductance.
There is a reduced SCR in people with Capgras Delusion. Perhaps that causes it? ("This doesn’t feel like I’m looking at my spouse …")

Ellis et al. (1997)
Patients with ventro-medial lesions have good recognition but low SCRs, and no Capgras delusion. (Patients with occipito-temporal lesions have an SCR in the absence of overt recognition.)

Tranel, Damasio & Damasio (1995)
A provisional conclusion

Coltheart et al. (2007, 2010)

The dissociation between the visual processing and the warm feeling of recognition seems to be a necessary condition for Capgras Delusion, but not a sufficient one.

We seem to need a two-factor theory.

Perhaps there is right hemisphere involvement?

“updat(ing) a cognitive hypothesis”, “belief evaluation”? 
A provisional conclusion

Staff et al. (1999)

RH anterior hypoperfusion in deluded vs non-deluded Alzheimer's patients.

Perhaps something in the RH is responsible for the “second factor”.

sagittal

coronal

transverse

Z value
Can we theorise about “updat(ing) a cognitive hypothesis”, “belief evaluation”?

Over-reliance on endogenous information relative to exogenous information. (They have a Bayesian description of this imbalance.)

It only affects one belief because the “evidence” (i.e. no affect for face of spouse) continually re presents itself to the individual.
“cognitive-behavioral therapy for delusions involv[es] engagement, the building of trust, discussing a range of explanations for the delusional beliefs, and reality testing (eliciting examination of evidence, logical inquiry, and reasoning)”
Cold caloric left vestibular stimulation increases activation of the right hemisphere, including regions of the right frontal lobe, and can cause temporary remission of a somatoparaphrenic delusion.

Activating the lateral, right frontal lobe can thus perhaps facilitate belief revision.
A predictive coding approach

The “Bayesian Brain” hypothesis: the brain is continually updating its “beliefs” on the basis of new evidence. (Such realistic probabilities are often counterintuitive.)

“Active inference”: the individual perceives/cognizes by making predictions and checking them against the real-world data of its own actions, the actions of others, or events in the world.

This is a version of “top-down vs. bottom-up.”

It’s a one-factor theory.
Your engine light is flashing (because it is too sensitive). The garage can’t find anything wrong. You suspect the garage is hopeless. But to them you seem paranoid. You have a (metacognitive) belief: that the engine light reports precise information about a belief (the engine is overheating). It’s all about how predictions or prediction errors are used to inform inference or hypotheses.
Predictive coding

Adams et al. (2013)

Prior belief is existing probability – knowing spouse is spouse.

Sensory evidence is new data – no affect (SCR), therefore a stranger.

Posterior belief is the updated belief after seeing the new data.

Posterior expectation is the likeliest (mean) value of the belief/delusion.
Predictive coding

Adams et al. (2013)

The width (variance) of the distributions is the inverse of precision.

Posterior belief is biased toward the prior or sensory evidence in proportion to their relative precision.

Posterior expectation can be biased toward sensory evidence by increasing sensory precision – or failing to attenuate it – or by decreasing prior precision.
There are abnormalities in schizophrenic smooth-pursuit eye-movements. These indicate abnormalities in the predictive use of sensory data.

Holzman (2001); Hutton & Kennard (1998)
Predictive coding theorists have *schematic suggestions* about the location and operation of delusion-sustaining abnormalities at the level of neurobiology: NMDA receptor function and dopaminergic neuromodulation, influencing post-synaptic gain.

The overarching principle is a reduction in the precision of prior beliefs relative to sensory evidence.

Adams et al. (2013)
Challenges

To make some new testable predictions to flesh out these neuropsychological and neurophysiological models.

To be able to say something about how the hypotheses get into the brain in the first place (... through the activity of the individual – it’s not all about the inside of the cranium).

To extend the theorizing to different delusions and hallucinations, in specific ways.

To make things better for those with these conditions.


Film of Capgras patient: https://www.youtube.com/watch?v=JQsQgoPQ24s