To the Chair of the Examiners for Part C of the FHS of Computer Science and Philosophy,

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HOW STRONG IS THE CASE FOR A MULTI-FACTOR ACCOUNT OF CAPGRAS DELUSION?

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How strong is the case for a multi-factor account of Capgras delusion?

A delusion is "a fixed belief that is not amenable to change in light of conflicting evidence."[1] A person who suffers from Capgras delusion has the belief that a close relative, usually a spouse, is an imposter in disguise. It is a type a monothematic delusion, as it is about one specific belief or theme, rather than being a polythematic delusion about a web of interconnected beliefs spanning various themes. While monothematic delusions are rare, they are useful to study, because all the cognitive faculties of the patients work exactly as normal. The only problem is their unshakeable delusion.

Two examples can be used to demonstrate how strong a Capgras patient's conviction is. The first is a woman who reported to the police that her husband had died and had been replaced by an identical looking man; she proceeded to wear mourning clothes, refused to sleep next to the 'double,' and tried to throw him out of their house.[2] The second is a man who believed that his family has been replaced by clones; he used a fake gun to threaten a reporter, convincing him to read out a statement about the substitution on television.[3] Moreover, in 46 of 260 reported cases of Capgras delusion, physical violence occurred.[4]

Considering how strong the case is for a multi-factor account of Capgras delusion requires finding the strongest specific formulation of a multi-factor account, and showing why that formulation is preferable to any other accounts. To do this, I start by examining Maher's anomalous experience hypothesis. Maher's account is a good starting place, not only because of its prominence, but also because it serves as a key component in multi-factor accounts, as it

[1] This is the clinical definition from DSM V, however, one of the central debates concerning delusions in philosophy is about what kind of mental states delusions they are, and whether they can even be called beliefs.
[2] Christodoulou, 1977
[3] Silva, Leong, Weinstock, and Boyer, 1989
[4] Förstl, Almeida, Owen, Burns, and Howard, 1991

is taken to be the 'first factor.' I critique it in two ways. The first is by using Campbell's top-down account, which criticises the bottom-up nature of Maher's and others' accounts. I show that Campbell's criticisms are invalid, and a bottom-up approach is superior. The second is by arguing that Maher's account is insufficient, and so more factors are needed for a bottom-up approach to work. Hence, I proceed by presenting Coltheart *et al.*'s two-factor framework and analysing it. I consider then give two competing specific accounts of what the second factor in this framework could be: a reasoning bias or a Bayesian account. I conclude by showing that the latter has advantages that the former does not, and therefore can be used to make the strongest multi-factor account of Capgras delusion.

1.

Maher's series of influential articles in the late 20th century outlines the anomalous experience hypothesis. This explains delusions as being normal, logical responses of an individual faced with an unusual experience. Maher writes that "the basic origin [of a delusion] lies in the anomalous experience."[5] The reasoning that is used by a delusional patient is no different from the reasoning used by people without delusions. However, non-delusional people, do not have that specific anomalous experience. The anomalous experience is usually identified as one of a broad range of neuropsychological anomalies.

Maher's account focusses on schizophrenia, which is a polythematic delusion, rather than on monothematic delusions. Building on his idea, Ellis and Young identify an anomalous experience when considering the Capgras delusion: a deficit in facial processing.[6] A Capgras patient is still able to recognise faces, but because of the neuropsychological deficit, they do not have the same affective or emotional response that people without the Capgras delusion

[5] Maher, 1988
[6] Ellis and Young, 1990

have when seeing familiar faces. The face of the patient's spouse would be highly familiar to them, so when the patient tries to make sense of seeing the familiar face, without having the normally associated affective response, they are led to conclude that their spouse is an imposter. Ellis and Young describe this as a "rationalization strategy" resulting in the thought that "the individual before them is … an imposter, a dummy, a robot, [etc.]"[7]

This rationalisation strategy can be detailed by using either an explanationist model, where the patient makes an inference that leads to their delusion, or an endorsement model, where the experience directly causes the delusion. The explanationist model argues that the patient's anomalous experience has a sparse content, e.g., the patient has a vague feeling of unease whenever they see their spouse. Their delusion then results from them trying to explain that vague feeling. The endorsement model argues that that the patient's anomalous experience has a rich content which directly warrants their delusional belief. For example, the content of the patient's perception is something like 'I am not looking at my wife.' On this model, the delusional belief comes from endorsing the content of a delusional perception.

Both models have disadvantages. The main problem for the explanationist model is that a delusion seems to be a terrible explanation for any kind of experience.[8] The main problem for the endorsement model is understanding how a person could have a delusional experience with rich content: for example, how does a Capgras patient experience their spouse as being an identical imposter?

When considering the aetiology of the delusion, the anomalous experience counts as a neuropsychological cause, as opposed to a psychological or psychiatric cause. Capgras delusion was first identified in 1923, and was originally thought to have psychoanalytic causes. The idea

[7] Ellis and Young, 1990: 244

[8] In Sections, I show how additional factors can make sense of why a delusional patient uses a delusion as an explanation.

of an imposter was thought to be due to a patient's subconscious way of handling mounting tensions between increasingly ambivalent feelings towards their spouse. More recently, it has been described as part of psychiatric syndromes.[9] However, examining causes in terms of cognitive neuropsychology is now the favoured approach. Ellis and Young explain how neural pathways are disrupted between the facial recognition system and the autonomic nervous system in the brain, often due to brain damage.[10] They use a two-component model of face recognition.[11] Seeing a familiar face evokes two things: (i) a conscious recognition of the identity associated with the face and (ii) an affective response to seeing that face. The former occurs via a ventral route, and is called a visuo-semantic pathway, as it constructs a visual image that encodes semantic information about a person's facial features. The latter happens via a dorsal route, and is called a visuo-affective pathway, as it produces an affective response (e.g., a feeling of familiarity) when seeing the face. Ellis and Young argue that Capgras delusion is caused by damage to the later route. This neurological deficit is unconscious, so the patient is not aware of the reduction in their affective response. The only way it would be consciously manifested would be as a vague feeling in the patient that something is different. This feeling that arises is why Ellis and Young reason that the delusion occurs: the patient relates that feeling of something being different when they encounter their spouse. The delusional belief is "the first delusion-relevant event of which the patient is aware."[12]

This theory was tested by measuring electrodermal responses (how conductive the skin is) to familiar and unfamiliar faces.[13] Electrodermal response is a marker of autonomic nervous

[9] Enoch and Trethowan, 1991

[10] It is not the case that damage to neural pathways has to occur as a result of brain damage. Corlett, D'Souza and Krystal (2010) report on a study where Capgras delusion was transiently induced in a healthy subject through the use of ketamine.

[11] This model was originally developed to account for prosopagnosia (face blindness). Prosopagnosia is thought to affect the visuo-semantic pathway, whereas Capgras delusion is thought to affect the visuo-affective pathway.

[12] Coltheart, Menzies and Sutton 2010: 264

[13] Ellis, Young, Quayle, and de Pauw (1997) test this using the faces of famous people, and Hirstein and

system arousal in response to a stimulus. Heathy patients had an increased response when seeing familiar faces, whereas Capgras patients had no measurable difference between seeing familiar and unfamiliar faces.

However, this way of thinking about a delusion may be problematic. Maher's hypothesis is a bottom-up approach, as it says that there must be something which causes the delusion; the delusion is best understood as an effect. Campbell calls this an empiricist approach, and criticises it for failing to account for the link between meaning and rationality. He instead proposes an account according to which delusional patients are non-rational.[14] I explore and analyse his criticisms of the bottom-up approach and the alternative account which he proposes next.

2.

According to Campbell, Maher's account can be distinguished into holding three separate claims: (i) one part of the cause of a delusional belief is an anomalous experience; (ii) the delusional belief can broadly be seen as a rational response to that experience; and (iii) the terms used by the delusional patient to explain the delusion retain their original meaning. Claim (i) is supported experimentally by Ellis and Young, as shown above. Claim (i) supports (ii) by the use of abductive reasoning, using either an explanationist model or an endorsement model, as outlined above. Claim (iii) is supposedly supported by (ii), as, insofar as the delusional belief is supported by the patient's experiences, it is possible to maintain a literal interpretation of their words. While the delusional patient is not fully rational, and does not always appreciate the tensions between how their delusional belief relates to other beliefs of theirs, they supposedly

Ramachandran (1997) replicate their findings. Bonifacci, Borlimi, and Ottaviani (2007) show similar results when using faces that are familiar to the Capgras patient as they are known by them personally, rather than by using the faces of famous people.

[14] Campbell, 2001

do accurately convey what they mean when describing their delusion.

Campbell rejects all three of these claims, and instead proposes an alternative top-down account. He rejects that (i) supports (ii), because the mere lack of affect when a patient encounters their spouse could not, by itself, constitute that perception having a particular content. He rejects (iii) because, following Quine, he argues that there is a constitutive link between how a person thinks about the meaning of a concept, and how they use that concept when reasoning. When reasoning, the concept must be used in a way that is systematically, causally dependent on the meaning with which we associate it. For example, a Capgras patient has an experience with the content 'this person before me is not the person I know, despite looking exactly like them.' Campbell argues we cannot ascribe the belief that 'this person before me is not the person I know' to them, as they are not using those words in the way a non-delusional person would use them when reasoning correctly.

Campbell proposes an alternative account, which instead holds that (a) monothematic delusions involve a "top down disturbance in some fundamental beliefs of the subject, which may consequently affect experiences and actions"[15] and (b) that delusions can be better understood as Wittgensteinian framework propositions.[16] Claim (a) argues that something fundamental changes within us, which causes us to experience the world in an altered way. In the case of Capgras delusion, Campbell argues that it is not the case that an anomalous experience causes the delusion; instead, it is the delusion that causes the anomalous experience. Claim (b) is about framework propositions, which are discussed by Wittgenstein in *On Certainty*. Wittgenstein argues that we accept some propositions which are exempt from doubt. Examples of such propositions are 'I have hands' or 'the world exists.' While they seem

[15] Campbell, 2001: 89

[16] These two claims are identified by Bayne and Pacherie (2004), who argue that they are independent and so can be assessed independently.

to say something factual about the world, meaning that they could be evaluated as true or false, Wittgenstein argues that these propositions cannot be doubted, as they serve the function in language of providing a framework within which other propositions can be evaluated. He compares them to a riverbed: they are required so that a river of language can flow meaningfully. Campbell writes that such propositions "are not … in any ordinary way subject to empirical scrutiny."[17] So when he describes delusions as framework propositions, he means that statements such as 'my spouse has been replaced by an identical imposter' act as a replacement for the linguistic framework that the patient would otherwise ordinarily use. The change in framework propositions of a Capgras patient's entails a change in the meaning of any and all terms used by the Capgras patient, which is why Campbell argues that they are not reasoning rationally. They have "lost their grip"[18] on the meaning of the words that they use, so a Capgras patient who states that 'my spouse is an imposter' means something genuinely different to what we mean when we state the exact same thing. A consequence of this is that there is no way of knowing precisely what the content of their delusion is.

However, Campbell's account is flawed.[19] Campbell writes that when a Capgras patient perceives their spouse, the lack of affect does not constitute the perception having a certain content. This is likely correct, as "one should not confuse the lack of experience with the experience of a lack."[20] However, the lack of affect when perceiving a spouse could easily generate the *experience* of a lack of affect. Bayne and Pacherie draw a parallel with patients who have lost their ability to see colour. Such patients' visual experience does not differ from normal visual experience because it lacks colour. Rather, it is different because the colour

[17] Campbell, 2001: 96
[18] Campbell, 2001: 94
[19] This follows Bayne and Pacherie (2004).
[20] Bayne and Pacherie, 2004: 3

shades have been replaced by various shades of grey. Thus, even conceding that when a Capgras patient perceives their spouse, the lack of affect is not enough to constitute there being a particular content of that perception, there *is* a particular content ascribed, as the feeling of familiarity when seeing a spouse is "replaced by a disturbing feeling of unfamiliarity and estrangement."[21] Therefore, Campbell's criticism of the 'rationalization strategy' which leads to a delusion is invalid.

Campbell's criticism of the claim (iii) above is tied to his idea about framework propositions. He rejects that Capgras patients' words are used with the correct meanings. However, it is ambiguous as to whether he wants to argue that the Capgras patient cannot use the terms 'spouse' or 'this person' in all contexts, or only in the context of framing the delusion. The latter position is more likely: Capgras patients do seem to know what these words mean when they talk about other people.[22] They even seem to use these words correctly when talking with their own spouse on the phone, as the delusion only manifests in-person. So why should the patient lose their grip on the meaning of words only when discussing delusions? It seems unlikely that words can be context-sensitive to that extent. Moreover, if Campbell is right, it is not clear whether there exists a better interpretation of what the patient *does* mean, given that they appear to use these terms correctly, and in a way which coheres with the other statements that they make. So, describing the beliefs of Capgras patients as framework propositions is unhelpful in advancing an account of their delusion.

Given these criticisms of Campbell's account, we move on to discussing a different criticism to Maher's anomalous experience hypothesis. This comes from Coltheart *et al*., who famously show that Maher's account is not sufficient to explain delusions.

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[21] Bayne and Pacherie, 2004: 4

[22] I.e., we judge them to be using the words correctly, and hence infer they have a correct understanding of the meanings of those words.

3.

Coltheart *et al.* argue that Maher's anomalous experience hypothesis is not a sufficient explanation of delusions. If it were sufficient, any person who had the anomalous experience that a given delusional patient has would have that same delusion. In the case of Capgras, this would mean that anyone with a deficit in their visuo-affective pathway should have the Capgras delusion. However, this does not happen. Tranel *et al.* describe patients with damage in their frontal lobes, who *do* exhibit a reduced affective response to familiar faces (measured using the electrodermal response), but do not then go on to have delusional beliefs about relatives being imposters.[23] Often in these cases, the experience is explained exactly how Capgras patients explain it, i.e., by saying that they had a vague feeling about the person they were seeing being different. These cases demonstrate that the neurophysiological explanation for Capgras is *not sufficient*: something more is needed to explain why a patient adopts the delusion. Hence, Coltheart *et al.* argue that the neuropsychological deficit is merely the first factor in the aetiology of a delusion. They propose adopting a two-factor framework.

The two factors can be framed as asking two questions: (1) Where does a delusion come from? and (2) Why does the patient not reject the delusional belief? The answer to the first question is the neuropsychological deficit which gives rise to an anomalous experience. The answer to the second question can be answered in various ways; momentarily I explain two such ways. Firstly, however, we must examine whether the two-factor framework is a good way of framing potential accounts of delusions in general. I outline two potential problems with the framework.

The first problem is that in searching for an answer to the second question, we need a

[23] Tranel, Damasio, and Damasio, 1995

coherent account of belief forming processes. However, as there is no consensus about belief formation and fixation, looking for an answer as to why a patient adopts a delusional belief is difficult. The second problem is that reducing accounts of delusion to just two factors may be an oversimplification. At least one other factor comes into play: the question of persistence. The two factors ask why a patient *hypothesizes* and subsequently *adopts* a delusional belief, but we also should ask why the delusion *persists* when the patient is presented with contradictory evidence.[24]

Nonetheless, the two-factor framework has its strengths: it helps to identify what is missing in a bottom-up explanation of delusions, given that the first factor alone is insufficient. This in turn helps with where to search for explanations. In the next two sections, I examine two accounts of what the second factor could be.

4.

One idea proposed for why delusional beliefs are adopted is a reasoning bias. Delusional patients show a 'jumping to conclusions' (JTC) bias. This means that they accept hypotheses as being correct on the basis of less evidence than a person without the delusion would require. A substantial amount of research supports the JTC bias: in a review of literature about JTC, Garety and Freeman conclude that there is "strong support for a reasoning bias in people with delusions, which is best described as a data-gathering bias: a tendency for people with delusions to gather less evidence than controls so that they jump to conclusions."[25] Experiments used to assess the JCT bias are based on probabilistic reasoning. One prominent

[24] For the sake of simplicity, I have written here that the original two-factor framework proposed the second question to be about belief adoption, and that it ignores the question of persistence. It is more accurate to describe the original framework as being ambiguous about whether the second factor refers to adoption or persistence. For more about this see Davies and Egan (2013).
[25] Garety and Freeman, 1999: 147

experiment, the beads task, is described as follows.[26]

In the beads task, participants are told that beads will be taken from one of two jars. Each jar contains two colours of beads in inverse ratios. For example, jar 1 would have red and blue beads in the ratio 85% red to 15% blue; jar 2 would have red and blue beads in the ratio 85% blue to 15% red. Participants are shown beads from one particular jar, and are required to guess which jar they are being drawn from. The participants request a bead as many times as they deem is necessary to come to a conclusion. After each draw, the participant is asked for their certainty level about which jar is being used. The numbers of draws required to reach a high and/or mean level of certainty is used as a measure, and cases where genuinely dis-confirmatory evidence is presented (e.g., all of the red beads from jar 2 being drawn at the start) are accounted for. The expectation is that delusional patients will come to a conclusion earlier, and indeed, a meta-analysis of studies shows that this is the case.[27]

This seems to suggest that the JTC bias can provide an answer to the second question, about why delusional beliefs are adopted. Garety and Freeman write that the bias likely plays a causal role, as "under certain conditions, [JTC bias could] contribute to erroneous inferences and, therefore, to delusion formation."[28] However, there is no evidence that the JTC bias can answer the persistence question, about why a belief is not rejected *after* it has been adopted. Moreover, it itself still needs further explanation: why do delusional individuals have that reasoning bias? A critic could argue that it simply moves the problem a step back. Some work has been done on this, however: reasoning is normally correlated with a person's long-term

[26] Another prominent experiment is the Wason four-card selection task, which also assesses probabilistic reasoning. Non-delusional participants are equally unsuccessful when this task is performed using abstract cards, and only show improvements when compared to delusional patients in scenarios where the cards have a themed context, e.g. a card with an alcoholic drink and a card with a drinking age limit.
[27] Fine, Gardner, Craigie, & Gold, 2007
[28] Garety and Freeman, 1999: 127

(semantic) memory, and that correlation is reduced in schizophrenic patients and individuals with high schizotypy scores. Nonetheless, much work is still needed to be done.

5.

The second approach for why delusional beliefs are adopted is a Bayesian one. It stems from an idea from Hemsley and Garety, that "[a] normative theory of how people *should* evaluate evidence relevant to their beliefs can provide a conceptual framework for a consideration of how they *do in fact* evaluate it."[29] When applied to the two-factor account, this idea proposes that we would be able to understand more about how delusions are adopted if we first understood, by the use of a probabilistic analysis, how delusional hypotheses were made. The aim of such an analysis would be to show that a normal deviation is to be expected, hence accounting for why some patients with an anomalous experience accept delusions, and why some do not.

In a Bayesian approach, subjective probabilities are calculated and continually updated based on available evidence. The probability of a given hypothesis H is equal to the old probability of that hypothesis H given new evidence E. I.e., if P' is the new probability distribution and P is the old probability distribution, then $P'(H) = P(H|E)$. It is important to note that we are not changing the hypothesis in light of new evidence, but just calculating the *new* probability of the *same* hypothesis.

Consider a Capgras patient. We label the true hypothesis, that their spouse genuinely is their spouse, H_T, and the delusional hypothesis, that the person who looks identical to their spouse is an imposter, as H_D. Let us also label the (incorrect) evidence as D. In this case, the evidence arises from the lack of affective response when the patient sees their spouse. The

[29] Hemsley and Garety, 1986: 52

probability $P(H_T)$ is higher than $P(H_D)$ initially: the patient sees someone who looks like their spouse and claims to be their spouse. However, when the patient sees their spouse, due to their neural deficit they do not have the normal affective response, thus supplying evidence. Over time, more evidence is gathered by the patient, and so the probabilities update, until eventually D is so big that $P'(H_T) = P(H_T|D)$ is lower than $P'(H_D) = P(H_D|D)$. As Coltheart *et al.* write, "It would be highly improbable for the subject to have the low autonomic response [D] if the person really was his wife, but very probable indeed if the person were a stranger."[30] Thus, by using abductive reasoning, Coltheart *et al.* argue that it is in fact reasonable for a patient to adopt the Capgras delusion.

Mckay criticises this account by raising the following two points. Firstly, given the content of the delusional hypotheses that we are considering, the delusional hypothesis must already have a high probability *before* any abnormal data is even gathered, in order for it to be considered in the first place. Given the absurdity of H_D, this is "unrealistically high,"[31] meaning that it would be unlikely to outweigh H_T. Secondly, the account from Coltheart *et al*. proposes that a delusion is hypothesized and adopted at the same time, thus combining the occurrence of the first and second factors. However, this cannot happen: factor two cannot precede or be acquired at the same time as factor one, as the delusion needs to be hypothesized before being adopted.

Mckay offers an alternative Bayesian approach that departs from the normative Bayesian model: he suggests discounting any prior probabilities before calculating updating probabilities. Doing so increases the relative weight of H_D to H_T. This creates a biased Bayesian model: instead of leaning towards the conservative or 'normal' belief that the patient's spouse

[30] Coltheart, Menzies, and Sutton, 2010: 277
[31] Mckay, 2012: 339

really is their spouse, it is biased towards the delusional belief. Although this seems odd, it provides explanatory adequacy: it can correctly predict and account for why a delusion is adopted, given abnormal data.

Moreover, by biasing a Bayesian model to discount any prior probabilities, there is no need to search for an additional factor that solves the persistence factor. Earlier, I explained that a problem with the two-factor account is that it only considers why delusional beliefs are hypothesized and adopted. It does not consider why they are maintained. However, on Mckay's biased model, because prior probabilities are discounted, the question of why a delusional belief persists is already answered: no weight is assigned to earlier hypotheses, so the strength of the delusional hypothesis can be maintained unchallenged. There is no need to identify a further persistence factor once the belief has been adopted. Although applying Bayesian models like this is clearly limited when considering real life belief systems, using statistical analyses can provide a strong account for why delusions occur.

6.

Assessing how strong the case is for a multi-factor account can only be done given the strongest example of such an account. I first argued that a multi-factor account is an example of a bottom-up approach to explaining delusions, and, by critiquing Campbell's top-down approach, argued that bottom-up approaches are superior to top-down approaches. I then argued that a single-factor account, namely Maher's anomalous experience hypothesis, is insufficient to explain delusions, and so, at the least, a second factor is needed. I presented the two-factor framework, and gave two options for what the second factor could be: the JTC bias, or Bayesian inferences. Of the latter, a biased Bayesian model is preferable to an unbiased Bayesian model, as it can answer the problem of why delusional beliefs persist. I argue that this is the strongest multi-factor account, and moreover, is the strongest overall account of Capgras delusion.

Sources and works cited

Bayne, T., & Pacherie, E. (2004). Bottom-Up or Top-Down: Campbell's Rationalist Account of Monothematic Delusions. *Philosophy, Psychiatry, & Psychology, 11*(1), 1-11.

Brighetti, G., Bonifacci, P., Borlimi, R., & Ottaviani, C. (2007). "Far from the heart far from the eye": Evidence from the Capgras delusion. *Cognitive Neuropsychiatry,12*(3), 189-97.

Capgras & Reboul-Lachaux, 1923

Campbell, J. (2001). Rationality, Meaning, and the Analysis of Delusion. *Philosophy, Psychiatry, & Psychology, 8*(2), 89-100.

Christodoulou, G. (1977). The syndrome of Capgras. *British Journal of Psychiatry, 130*(6), 556-564.

Coltheart, M., Menzies, P., & Sutton, J. (2010). Abductive inference and delusional belief. *Cognitive Neuropsychiatry, 15*(1-3), 261-287.

Coltheart, M. (2007). The 33rd Sir Frederick Bartlett Lecture Cognitive neuropsychiatry and delusional belief. *Quarterly Journal of Experimental Psychology, 60*(8), 1041-1062.

Corlett, P. R., D'Souza, D. C., & Krystal, J. H. (2010). Capgras syndrome induced by Ketamine in a Healthy Subject. *Biological Psychiatry*, *68*(1), e1-e2.

Davies, M., Coltheart, M., Langdon, R., & Breen, N. (2001). Monothematic Delusions: Towards a Two-Factor Account. *Philosophy, Psychiatry, & Psychology, 8*(2), 133-158.

Davies, M., & Egan, A. (2013). Delusion: Cognitive Approaches — Bayesian Inference and Compartmentalization. In *The Oxford Handbook of Philosophy and Psychiatry*, 689-728. Oxford: Oxford University Press.

Ellis, H., & Young, A. (1990). Accounting for delusional misidentifications. *The British Journal of Psychiatry : The Journal of Mental Science, 157*, 239-48.

Ellis, H., Young, A., Quayle, A., & De Pauw, K. (1997). Reduced autonomic responses to faces in Capgras delusion. *Proceedings of the Royal Society B: Biological Sciences, 264*(1384), 1085-1092.

Enoch, M., & Trethowan, W. (1991). *Uncommon psychiatric syndromes* (3rd ed.). Oxford: Butterworth-Heinemann.

Fine, C., Gardner, M., Craigie, J., & Gold, I. (2007). Hopping, skipping or jumping to conclusions? Clarifying the role of the JTC bias in delusions. *Cognitive Neuropsychiatry, 12*(1), 46-77.

Förstl, H., Almeida, O., Owen, A., Burns, A., & Howard, R. (1991). Psychiatric, neurological and medical aspects of misidentification syndromes: A review of 260 cases. *Psychological Medicine, 21*(4), 905-910.

Garety, P. A. and Freeman, D. (1999). Cognitive approaches to delusions: A critical review of theories and evidence. *British Journal of Clinical Psychology*, 38, 113-154.

Garety, P.A., Freeman. D. (2013) The past and future of delusions research: from the inexplicable to the treatable. *British Journal of Psychiatry, 203*(5), 327-333.

Hemsley, D., & Garety, P. (1986). The formation of maintenance of delusions: A Bayesian analysis. *British Journal of Psychiatry, 149*(JULY), 51-56.

Hirstein, W., & Ramachandran, V. (1997). Capgras syndrome: A novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings of the Royal Society B: Biological Sciences, 264*(1380), 437-444.

Maher, B.A. (1988). "Anomalous experience and delusional thinking: The logic of explanations". In T.F. Oltmann and B.A. Maher (Eds.), *Delusional Beliefs*, 15–33. New York: Wiley.

Mckay, R. (2012). Delusional Inference. *Mind & Language,27*(3), 330-355.

Silva, J.A., Leong, G.B., Weinstock, R. & Boyer, C.L. (1989) Capgras syndrome and dangerousness. *The Bulletin of the American Academy of Psychiatry and the Law*. *17*(1), 5-14.

Stone, T., & Young, A. (1997). Delusions and Brain Injury: The Philosophy and Psychology of Belief. *Mind & Language, 12*(3-4), 327-364.

Tranel, D., Damasio, H. & Damasio, A. R. (1995) Double dissociation between overt and covert face recognition. *Journal of Cognitive Neuroscience 7*(4):425–32.