Abstract: Delusional behavior in humans presents a rich opportunity for philosophical and psychological communities to unite in an effort to understand the nature of our own minds, especially the relationship between our seemingly immaterial beliefs and material brains. This paper explores recent work on delusional behavior, and highlights conceptual problems in recent empirical and conceptual investigations. This paper also investigates the natures of two particularly peculiar delusions – the belief that your loved ones are identical imposters (called Capgras delusions) and the belief that you are dead (called Cotard delusions) – are investigated. Young (1999) has argued that these delusions are really two sides of the same coin and that individual difference in emotional style/personality account for the different content of the delusions. In response, I argue that the content of Cotard delusions indicates an etiological difference to Capgras delusions. In addition, Young has not verified the “sequence” of factors in his explanation. If it is the case that the symptoms occur in a different order, Young’s explanation would be demonstrated false.

The interaction between the human brain and the human mind occupies a place of irony for the scientific and philosophical community. It is this relationship between our minds and our bodies that represents a significant part of who we are – it is the connection between a shared material world and our private consciousnesses life. Yet, theorists are hit with significant limitation. Ethical restraints disallow the intentional creation of neurologically impaired humans that would be ideal candidates for the study of neuro-psychology.

It is because of this irony that we should be especially attentive at the prospect of accidental or natural deviations from normal human behavior and their bodily correlates. This paper will explore recent psychological and philosophical explanations of delusions, explain
Young’s (1999) proposed commonality between two specific types of delusion, Capgras and Cotard delusions, and argue that his proposed unity can be differentiated on etiological grounds.

I. Background

Recent work in psychology has argued that delusions are not simply failures of logic or reality checking. Rather, many delusions are logically created explanations to abnormal sensory stimuli (Maher, 1974). For example, someone who is losing their hearing may develop the belief that they are being spoken about behind their back. It seems everyone is speaking more quietly than they used to, and one possible explanation the impaired person could generate is that those people are keeping secrets from them.

Abstract reasoning tests have demonstrated a significant difference between clinical and general populations in the amount of data each group requires before reaching a conclusion. Huq, Garety and Hemsley (1988) filled two jars with different colored beads in different ratios. The participant was asked to assess whether or not a given series of beads was more likely to be drawn from the first or second jar. Participants with delusions requested significantly less information before coming to a conclusion than their non-clinical counterparts - even in a task that bears no relationship to the content of their delusions. For the rest of this discussion I will refer to this behavior – the use of significantly less information in coming to a conclusion that is exhibited by those with delusions – as reasoning bias.

Much of this work is bogged down by two main research problems. Though the nature of some specific delusions has been studied, psychology is far from being able to explain all
delusions it has encountered. Further, there is a belief that research should be focused on individual symptoms (like delusions of grandeur) rather than the larger system of disorder (schizophrenia) that generates those symptoms (Costello, 1992). This has two effects.

Our scope is limited. Because we are dealing with information about only a small number of delusions the, claims that we can make about delusions as a whole are limited. We have no way, at present, of verifying that the conclusions we come to are true of all delusions.

It is possible to miss underlying structures which could unify or differentiate delusional behavior and effect treatment strategy. What we are missing is a “germ theory” of delusions. Consider, strep-throat and the common cold. For a period of time, they both have fatigue and congestion. If we focus on their symptoms alone, the two diseases are likely to be confused for the same thing during their beginning stages – even though one is caused by bacteria and the other by a virus. A research focus on symptoms is likely to miss the kind of etiological theory we need to both classify delusional behavior.

What is perhaps most difficult is defining what a delusion actually is. Many argue that delusions are strongly held false beliefs, but this is put in direct conflict with how beliefs seem to operate. Dennet (1981) has argued that beliefs don’t exist in isolation, rather they function as parts of a whole – a kind of web of belief.

To think about this, we could consider the idea of inserting false beliefs into someone’s head. If I wanted to convince someone that they were married, this would require more than simply inserting the belief that they are married. It would require modification of all other beliefs relevant to marriage. I would need memories of the wedding and insert an explanation
about where the spouse is likely to be at any given time, etc. Thus, if a new belief is created it requires the modification of other existing beliefs.

However, when a given person has a delusion, they devote large amounts of focus to the content of that delusion, but not the other relevant beliefs that delusion should modify. Generally, a person with a delusion of grandeur who believes he is Napoleon does not usually march in a field in France, obsess over strategy, or attempt to find his troops.

Two hypotheses emerge. It may be that delusions are beliefs, even though they do not affect some of the other beliefs in the web. Perhaps those with delusions fail to consider sufficient evidence for their delusional beliefs (reasoning bias) in addition to failing to explore the implications of their new belief. Or, perhaps some of the utterances given by those with delusions are actually metaphors. Halligan and others (1995) did a case study of a patient with somatoparaphrenia who had an obsession with his foot. He believed commented that it looked like a cows foot and felt heavy. What is interesting here is that this patient, when we consider his symptoms, is speaking completely reasonably. If his foot is feels too heavy to move without significant effort, then it is reasonable to say so. That cannot be regarded as a false belief, it is a reasonable explanation of an anomalous perceptual experience (heaviness of the foot). Further it is important to note that the patient uses simile. He says his foot is like a cows foot, which is reasonable if he cannot move his toes – it is a comparison, not a belief.

Some authors are willing to go even further than calling delusions metaphors. They are willing to say that a patient’s delusions are merely “empty speech acts that disguise themselves as beliefs” (Berrios, 1991, p8). This is a bit extreme because there is evidence to suggest some people with delusions (18%) do show violent behavior, and are willing to act on their delusions
(Forstl et al., 1991). Thus, delusions seem to occupy some sort of space between direct claims about the properties of the material world and metaphoric explanations of odd experiences.

A solution to this problem is important for both medicine and philosophy. In order to treat a patient, it is important to understand the nature of the claims they are using to describe their symptoms.

Given this, I will continue with my analysis of two specific delusions, Capgras and Cotard delusions, under the assumption that the claims made by the subjects of inquiry are: (a) ambivalently held beliefs (they are not always treated as “true” by their believers) and (b) the claims made by those with delusions are, at least, held strongly enough as to motivate violence based on their content.

III. Cotard and Capgras Delusions and Young’s Connection

Capgras and Cotard delusions involve especially peculiar claims, even by psychiatric standards. People with Capgras delusions claim that one or more of their close family members has been replaced by an identical imposter. Those with Capgras delusions report widespread feelings that things have changed and act with a mood of suspiciousness. People with Cotard delusions believe that they are dead and are usually in a depressed mood.

Young (1999) explains that after many years of research what has stood out to him about Capgras delusions is their seeming inversion of the neurological disorder prosopagnosia (difficulty recognizing faces). Indeed, one of the most striking features of the delusion is the patient’s inability to describe exactly what is different about the imposter. After all, how does the person with a delusion know that their loved one has been replaced, if the imposter is the
same in appearance and behavior? Or rather, why do they believe that to be the case? To find out, it is helpful to compare the delusion to prosopagnosia.

In prosopagnosia, people have a difficult time with facial recognition, even for faces that should be the most significant for them (faces of their spouse, their own face, etc). However, even absent being able to consciously recognize a face, some non-conscious emotional responses remain. Bauer (1984) measured the autonomic responses of participants with prosopagnosia and found that although they cannot recognize faces, the autonomic responses are more significant with faces that should be familiar. These autonomic factors (such as skin conductance response) are argued to be measures of emotional response. In other words, people with prosopagnosia cant recognized their loved ones, but still get an emotional response when they see them.

This is the opposite for those with Capgras delusions, where patients can recognize the face of their spouses (even if they don't believe it is the real deal), yet their affective responses seem to have dissipated. Young (1999) proposed that Capgras delusions arise as an attempt, on the part of the patient, to explain loss of affect response when exposed to a loved one. This explains the feelings that things are different (because they actually do feel different) and also explains why those with Capgras delusions usually believe a close relative (who should give them the largest emotional response) have been replaced. It is easy to shrug off no emotional response to an acquaintance, more difficult to ignore lack of emotional response to your mother.

Young’s (1999) theory has been tested by Ellis et al (1997). They found a loss of differential skin conductance response. Additionally, Ramachandran (1997) also noted that
patients who believe their parents to be imposters behave differently when with their parents in the room versus on the phone. When patients spoke with parents on the phone they no longer believed their parents to be imposters, which suggests an emotional disruption that has to do with vision (or perhaps even simply faces) only. Thus, it is reasonable to conclude, consistent with the above argument that delusions are rational responses to perceptual abnormality, that Capgras delusions arise as an attempt to explain the loss of emotional response to a loved one.

Young (1999) argues that the explanation just provided is also what accounts for the behavior in Cotard delusions, in which people believe that they are dead. And, in this case, there is a similar loss of affective component in facial recognition. Young has explained that the difference in content of these two delusions is likely to be determined by what he calls “attribution style” – the attribution of negative events to personal or external causes based on mood. For purposes of this discussion, attributional style will be limited to descriptions of negative life events given by those with delusions. Additionally, I will focus only the two styles that Young (1999) mentions: suspicious (associated with Capgras delusions) and depressed (associated with Cotard delusions).

Earlier work on delusion has demonstrated that people with persecutory delusions are likely to attribute a given problem to external causes. For example, when given the prompt “you have a job interview and it goes badly” and imagine a possible reason why, those with persecutory delusions are likely to blame an external factor like their interviewer being in a bad mood (Candido and Romney 1990). Those whose mood is typically suspicious are likely to respond to their newfound emotional impairment with a suspicious scenario involving an
external source. Those with a depressive history (which is everyone who has a Cotard delusion) is likely to respond to the same lack of affect with a self oriented explanation – that they are dead.

IV. Objections

While this theory seems able to draw an interesting connection between two different delusions, and even explain their interaction with neurological events, it does have two major etiological shortcomings. First, the more pervasive nature of depression and delusion for the Cotard patient is unaccounted for. Neurological testing of a Cotard patient demonstrated feelings of depersonalization and derealization (Gerrans, 1999). These feelings reflect a large and systemic change for the Cotard patient’s experience of the world. It makes sense that this is reflective of a neurological change which is broader in scope than in Capgras delusions. That is, for Cotard patients, multiple neurological sub-systems seem to have been affected. However, in the case of Capgras delusions, only affective responses to familiar faces changed.

Thus, although loss of affect may be involved for both cases, it seems likely that more neurological subsystems must be affected for the Cotard patient than for Capgras. This would differentiate them etiologically and would mean that the lack of facial-affective response that Young (1999) describes is not likely the primary factor in causing delusions, and it would also mean that delusions have do not different content solely because of attributional style. Capgras and Cotard patients are not reacting (attributing) to the same stimulus in different ways. They are responding to two qualitatively different stimuli: loss of the reality of others or loss of reality of the self and the world.
Additionally, Gerrans (1999) has argued that there is a second etiological component that could differentiate the two delusions. Because a depressive component is not present in Capgras delusions, the question of when depression is involved in the sequence of the Cotard delusion could indicate that emotional response to faces is not a unifying factor, only a coincidental one. Garrens admits that he does not know which explanation is actually the case, but there are three possible progressions of the Cotard delusion that have to do with its depressive component. Two of these scenarios represent a major etiological difference between the two delusions and would refute the idea that it is attribution style that causes the difference in content of the delusion.

Because these scenarios involve the hypothetical rearrangement of several factors, it is important to clarify the variables we are working with. First, the following three sequences are about Cotard patients only. So when “attribution style” is mentioned, it is only the type used by Cotard patients (internalization of blame based on depression) that I am referring to. “Loss of affect” will also only be used for the Cotard patient. Thus, it will be applied for the whole spectrum of emotional loss (loss of reality, self, and facial response), not just loss of emotional responses to faces. Keeping with the trend, the delusions mentioned in the following sections will be ones that include the belief that one is dead, unless otherwise specified. Finally, reasoning bias will be used with definition given earlier in the discussion – use of little data in the formation of a conclusion. All of the following scenarios will involve these four variables arranges in different combinations and sequences.

The first scenario is as as Young has argued, the Cotard patient was already depressed at the time of neurological malfunction and, upon noticing the change, attributed it to themselves
personally. The explanation they came up with, based on an internalized view of the problem, was that they have died. Thus Young’s progression is:

**Reasoning Bias + Loss of affect + Depressive Attributional Style => Delusion**

The second scenario, against Young, is that it could be that the Cotard patient’s depression is caused by the affective malfunction. Consider that the conclusion that you are dead is likely to be associated with strong negative emotions. In this case, attributional style could not play (a very important) role in the formulation of the delusion because the delusion would be *prior* to the attributional style (depression). The new formula for the creation of Cotard delusions would be:

**Loss of Affect + Reasoning Bias => Cotard Delusion => Depression (the souce of attributional style)**

Finally, we could imagine a scenario where major depression brings on the loss of affect and the delusion. In these instances, we would expect that the delusion would dissipate along with depressive symptoms, as it already has in a case study done by Young (Gerrans, 1999). That depression brings on the loss of affect and the delusion makes even more sense when we consider the range of disruption the Cotard’s patient experiences. They have lack of affective response to facial recognition, but also to the rest of the world. Additionally, the severity of their conclusion is much greater and is a belief in a fundamental change in the system, not just
one person. This makes sense when we consider the global effect depression has on neurological function. Depression affects not only a person’s affect-response (as we see in the Cotard patient) but also, general happiness, and cognition (Gerrans, 1999). This is given even further support when we consider that Cotard delusions can emerge without the kind of organic tissue damage described by Young. All of these factors contribute to the position that the loss of affect that Young believed to be anterior to delusion, is actually a result of the same factor that also caused the delusion. Depression has brought on the delusion and loss of affect.

So, the third, most likely formula is:

Depression + Reasoning Bias => Loss of Affect + Delusion

V. Conclusion

Finally, to limit my claims, I do believe that the basic structure of delusion mentioned earlier is correct. That delusions form as a response to perceptual changes seems to be born out in the data. Thus, the basic framework Young is using is still correct. However, his claim that Cotard and Capgras delusions are both formed because of different attributional styles used during the same perceptual change seems flawed. The more pervasive nature of non-delusional and delusional symptoms in Cotard patients seems to indicate broader neurological changes than the relatively limited changes in Capgras patients. Additionally, sequencing and consideration of the factors involved in depression hint that there are at least two other likely scenarios that can explain Cotard delusions in ways that are etiologically different from the formation of Capgras elusions.
Works Cited


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