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Revisiting Maher's one-factor theory of delusion

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Abstract

How many factors, i.e. departures from normality, are necessary to explain a delusion? Maher's classic one-factor theory argues that the only factor is the patient's anomalous experience, and a delusion arises as a normal explanation of this experience. The more recent two-factor theory, on the other hand, contends that a second factor is also needed, with reasoning abnormality being a potential candidate, and a delusion arises as an abnormal explanation of the anomalous experience. In the past few years, although there has been an increasing number of scholars offering a variety of arguments in defence of Maher's one-factor theory, these arguments have not been adequately addressed by two-factor theorists. This paper aims to address this gap by critically examining the arguments on three crucial issues: the intelligibility of delusions, the dissociation between anomalous experiences and delusions, and the empirical evidence of a second factor. I will argue that the Maherian notion of anomalous experience is not sufficient for explaining delusions and the two-factor theory is on the right track in its search for the missing factor in the aetiology of delusions.

Keywords: delusion; one-factor theory; two-factory theory; anomalous experience; reasoning abnormality

1. Introduction

Delusions are the paradigmatic symptoms of madness (Jaspers, 1913/1997, p. 93). A patient with the Capgras delusion may believe that "This woman [the patient's wife] is an imposter" (Capgras & Reboul-Lachaux, 1923; translated in Ellis et al., 1994; Coltheart & Davies, 2022); a patient with the Cotard delusion may believe that "I'm dead" (Cotard, 1882); a patient with the delusion of thought insertion may believe that "Alien thoughts, such as 'Kill God', are put into my mind" (Frith, 1992; Roessler, forthcoming). Delusions may seem so baffling that it seems to be a formidable task for one to get a grasp on what is going on in them. The bafflement is famously reflected in Jaspers' (1913/1997) remark that certain delusions are ununderstandable (Eilan, 2000; Hoerl, 2019).

In the current literature, there is a wide range of competing theories of delusions (for review, see e.g. Connors & Halligan, 2020; Sass & Pienkos, 2013). Among them, the two-factor theory (Coltheart & Davies, 2021; Davies et al., 2001) is arguably "the most influential neurocognitive account of delusion in the scientific literature" (Braun & Suffren, 2011, p. 2).

The two-factor theory is often presented as a development based on Maher's classic one-factor theory (1974, 1988, 1992, 1999). According to Maher, the only factor, i.e. the only departure from normality, in the aetiology of a patient's delusion is the patient's anomalous experience, and delusions arise as normal explanations of anomalous experiences. The two-factor theory accepts that anomalous experiences play important roles in the aetiology of delusions, but argues that a second factor is also involved, a candidate for which is reasoning abnormality. Despite the popularity of the two-factor theory, in the past few years there has been an increasing number of scholars offering a variety of intriguing arguments in defence of Maher's one-factor theory (Franceschi, 2010; Noordhof & Sullivan-Bissett, 2021; Reimer, 2009; Sakakibara, 2019; Sullivan-Bissett, 2020, 2022).[1] So far, these arguments have not been properly addressed by two-factor theorists, however.

[1] Maher's one-factor theory was initially developed with a focus on delusions in schizophrenia, while the two-factor theory focused on monothematic delusions of neuropsychological origin. As a reviewer for this journal points out, if these two types of delusion require substantially distinct explanations, then some of the apparent disagreements between proponents of the one-factor theory and proponents of the two-factor theory may be a result of them not fully recognizing the target of each other's theories. Indeed, in his later work, Maher (2006) also seemed open to the possibility "that the origins of delusions are heterogeneous" (p. 183). However, it is equally worth noting that both Maher and two-factor theorists take their framework as applicable to delusions in general. Maher (1999), for example, suggested that his one-factor theory of delusion is "not confined to schizophrenia." (p. 556) In addition, recent defenders of Maher also take the one-factor theory as applicable to monothematic delusions (Noordhof & Sullivan-Bissett, 2021, p. 10281; see also Reimer, 2009; Sullivan-Bissett, 2020, 2022). Likewise, two-factor theorists argue that "the 2-factor account of particular delusions like Capgras and Cotard still applies even when these delusions occur in the context of schizophrenia" (Coltheart et al., 2007,

In this paper, I will fill in this blank by critically examining the arguments from both two-factor theorists and one-factor theorists. The central question I will focus on is whether Maher and his defenders' view is correct that anomalous experiences are the only factor in the aetiology of delusions. I will set aside other questions such as whether a specific version of the two-factor theory offers a satisfactory explanation of delusions (for critique, see e.g. Corlett, 2019; Parrott, 2016; for defence, see e.g. McKay, 2019; Nie, 2016, 2019), and what the contributions made by other theories of delusions are.

Here is the plan. Section 2 will introduce the gist of Maher's one-factor theory: the anomalous experience hypothesis and the normal reasoning hypothesis. As an illustration, Section 3 will discuss several candidates for the anomalous experience in the Capgras delusion. Section 4 will discuss the details of the arguments from both proponents and opponents of Maher's one-factor theory, which revolve around three central issues: the intelligibility of delusions, the dissociation between anomalous experiences and delusions, and the empirical evidence of a second factor. Overall, I will argue that the Maherian notion of anomalous experience is not sufficient for explaining a delusion and the two-factor theory is on the right track in its search for the missing factor in the aetiology of delusion.

2. Maher's one-factor theory

Maher's (1974, 1988, 1992, 1999) one-factor theory is widely accepted as a starting point for the modern frameworks for explaining delusions (see e.g. Davies & Egan, 2013, p. 690). According to Maher, delusions arise as *normal* explanations of *anomalous* experiences. This view is not without precursors. Coltheart et al. (2010) have traced it back to James' (1890/1952) *The Principles of Psychology,* in which James wrote: "The delusions of the insane are apt to affect certain typical forms, very difficult to explain. But in many cases they are certainly *theories* which the patients invent to account for their bodily sensations" (chap. XIX, emphasis added).

Maher's one-factor theory, however, does not merely reinstate the view that delusions are the subjects' theories to explain their anomalous experiences. According to Maher (1988),

p. 642). In this paper, I will view the one-factor theory as a framework aiming to explain delusions in general. I will set aside issues concerning whether the one-factor theory might be the best explanation of a particular type of delusion.

delusions are a particular kind of theory: "normal theories" or normal explanations (p. 20). What does Maher mean by normal theories or normal explanations? He (1974) writes:

> It is the core of the explanations (i.e. the delusions) of the patient are derived by cognitive activity that is essentially indistinguishable from that employed by non-patients, by scientists, and by people generally. . . . [A] delusion is a hypothesis designed to explain unusual perceptual phenomena and developed through the operation of normal cognitive processes. (p. 103)

A variety of cognitive processes may be employed when we explain something: such as attention, remembering, reasoning, and metacognition (Horne et al., 2019). Among them, Maher emphasises that the reasoning processes in the aetiology of delusions are normal. To highlight this point, we can call it the normal reasoning hypothesis (instead of the normal-cognition hypothesis).

Distant kin of the normal reasoning hypothesis were arguably prominent in the 17th and 18th centuries, during which period many believed that "delusions resulted from failures (caused by physical reasons) in the apparatus that served to acquire experiences. The system designed to process information was in order." (Berrios, 1991, p. 7)

For Maher, the normal reasoning hypothesis is not merely a hypothesis but was *supported* by the repeated failure of empirical studies to find *logical reasoning abnormalities* in schizophrenia. In a review published in 2000, Mujica-Parodi et al. argue that "research to date has been inconclusive on the fundamental question of whether patients with delusional ideation show abnormalities in logical reasoning." (p. 73; see also Maher, 2001)

The key to understanding Maher's one-factor theory is his conception of anomalous experience. Four important claims constitute Maher's theory. First, Maher's anomalous experience is a *conscious* experience: patients with delusions are consciously aware of the anomaly associated with their experience. Second, the anomalous experience is *not* anomalous in the sense that it provides the patients with distorted shapes, colours, sounds, and the like. This is in line with Jaspers' (1913/1997) observation that "perception itself remains normal and unchanged" in patients with delusions (p. 100). Third, the anomaly is an anomalous feeling of significance. According to Maher (1999), feelings of significance are a distinctive kind of primary experiences, in the sense that "they have the same quality of irreducible directness as do such experiences as sensory experience of color, the feeling of physical pain, the experience of sound, and other sensations." (p. 552) At the subconscious level, feelings of significance are generated by a monitoring mechanism when it detects "discrepancies between expected and

perceived input experience" (p. 556). Fourth, the feeling of significance is imprecise. It is "a *vague general feeling* that prompts us to look for [an explanation]" (p. 554, emphasis in original).

To illustrate how feelings of significance give rise to beliefs in non-delusional cases, Maher offers the following example:

> A common experience that we encounter occurs when we meet somebody familiar to us, but have the conscious experience that the person "looks different somehow." We scan the person's appearance and may fail to detect any concrete differen[ce]. We ask, and find out that our friend has changed hair-style, shaved off a beard, grown a beard, or so forth. Sometimes our scan has already identified the difference and we do not need to ask. This writer once shaved off his beard and the next day was greeted with comments ranging from direct recognition of what had changed, to the case of one colleague who commented "There's something different about you. Oh yes. You are wearing a different kind of tie." (p. 553)

This is a helpful example to compare with the Capgras delusion: the belief that this woman looks like his wife but is not his wife. According to Maher, both the ordinary belief in the quoted case and the Capgras delusion arise as *normal* explanations of the experiences with feelings of significance. The difference between them is that, while the feeling of significance in the quoted case is a normal feeling, the feeling of significance in the Capgras delusion is something that normally would (and should) not have occurred, and hence is anomalous.

Despite the fact that the comparison between the quoted non-delusional cases and the Capgras delusion helps us get a grasp on what Maher thinks is going on in delusions, it does not suffice to tell us, however, what exactly the anomalous experience is such that the Capgras delusion arises as a normal explanation of it. Let us call the feeling of significance in the Capgras delusion *significance*$_d$, and call the feeling of significance in the quoted non-delusional case *significance*$_n$. Significance$_d$ is unlikely to be the same as significance$_n$. By definition, significance$_n$ is prevalent in our daily lives. If significance$_d$ were the same as significance$_n$ and the Capgras delusion were a normal explanation of significance$_n$, then the belief that someone is an imposter would have been a familiar belief in our daily lives. The latter is, of course, not true. Therefore, the anomalous experience in the Capgras delusion must be a distinctive kind. Now the question is: how can we know what exactly this distinctive kind of anomalous experience in the Capgras delusion is?

3. Searching for the anomalous experience in the Capgras delusion

To find out what a person's experience is, one natural way is to ask the person. But people with delusions are often reluctant or find it difficult to give detailed and accurate reports of their experiences: the reason why they are reluctant to report may include the worry about the social stigma of mental illness; and the reason why they find it difficult to report may include the fact that their experiences are too anomalous to be described by ordinary language, which, one might argue, has not evolved to describe something so alien to people without delusions. Sometimes when patients are relatively engaging in an interview, their reports, such as the claim of the patients with the Capgras delusion that they noticed a little mark on the imposter's ear, sound more like confabulations rather than the actual anomalous experiences that give rise to their delusions.

Because of these obstacles and many others, in the past psychiatrists and philosophers had to heavily rely on intuitions and conjectures, which unfortunately often conflicted with each other. With little consensus on how they can be empirically verified or disconfirmed, these theories often ended up in chaos. Similar chaos is infamously evident in the literature on psychodynamic theories of mental disorders. In their review of psychodynamic theories of the Capgras delusion, Ellis and de Pauw (1994) complained: "Over the last seven decades, a plethora of mutually-incompatible psychodynamic explanations, reflecting the imagination and conceptual frameworks of individual authors, have been invoked to account for the CD [Capgras delusion]." (p. 318)

The major breakthrough is made by Ellis and Young (1990), with the help of which we now have a better understanding of what the anomalous experience in the Capgras delusion may be. Ellis and Young propose that the Capgras delusion is the "mirror image of prosopagnosia" with covert recognition (p. 244). People with prosopagnosia are unable to overtly recognise familiar faces, such as the faces of famous people, their friends, family members, or even their own (Ellis, 1989). However, some people with prosopagnosia may exhibit forms of covert recognition of familiar faces: for example, when they see familiar (versus unfamiliar) faces, some retain a higher level of activity in the autonomic nervous system, which is measured by skin conductance (e.g. Bauer, 1984).

According to Ellis and Young's (1990) "mirror image" proposal, in comparison to the individuals with prosopagnosia who are unable to overtly recognise familiar faces but retain normal covert autonomic responses to familiar faces, people with the Capgras delusion can

overtly recognise familiar faces but have abnormal covert autonomic responses to familiar faces (p. 244). While it is true that many people with the Capgras delusion have no difficulty in overtly recognising familiar faces given that they claim that the imposters look like the persons they impersonate, it is an empirically testable prediction that people with the Capgras delusion have abnormal autonomic responses to familiar faces. Since it was proposed, this prediction has been empirically confirmed by a number of studies (Bobes et al., 2016; Brighetti et al., 2007; Ellis et al., 1997; Ellis et al., 2000; Hirstein & Ramachandran, 1997; Nuara et al., 2020).

Now it seems clear that people with the Capgras delusion have reduced autonomic responses to familiar faces. And, compared with pure conjectures about what the anomalous experience in the Capgras delusion is, Ellis and Young (1990) and others' work on the reduced autonomic responses to familiar faces gives us a solid ground to find out what the anomalous experience in the Capgras delusion is.

The reduced autonomic responses measured by skin conductance themselves, however, do not suffice to tell us what the anomalous experience is. This is because the activity in the autonomic nervous system is both an unconscious phenomenon (Coltheart, 2005) and "a nonspecific physiological response": the autonomic nervous system can be aroused in many different ways, such as by a loud tone (Breen et al., 2000, p. 57). There are different views about what information is encoded in the reduced autonomic responses, and what kind of anomalous experience is underpinned by the reduced autonomic responses. Regarding the anomalous experience, there are at least three different views in the literature.

First, according to what we can call the Affective View, which is evident in Ellis and Young (1990), the autonomic activity "carr[ies] some sort of affective tone." (p. 244) That is to say, people with the Capgras delusion experience reduced affective responses at the conscious level. When the person with the Capgras delusion sees his wife, he has "an experience of seeing a face that looks just like … [his wife], but without experiencing the affective response that would normally be part and parcel of that experience." (Stone & Young, 1997, p. 337)

Our affective states include a wide range of emotions and moods. Among them, the feeling of familiarity is assumed by many scholars to be at the centre of the aetiology of the Capgras delusion. It is also mentioned by Ellis and Young (1990), in which they sometimes suggest that the autonomic activity carries the tone of the "affective familiarity" in particular. That is to say, people with the Capgras delusion experience a feeling of reduced familiarity when they see familiar faces.

Second, according to what we can call the Alert View, advocated by Coltheart (2005), we have an "automatic and unconscious" prediction and comparison system whose job is to make predictions and compare the predictions with inputs. If the predictions and inputs do not match, then an alert, which is not yet a conscious experience, will be reported "to consciousness to instigate some intelligent conscious problem-solving behaviour". In the Capgras delusion, when the person sees his wife, his prediction and comparison system predicts high-level autonomic responses, but this does not match with the received low-level autonomic responses, the mismatch results in the prediction and comparison system's "reporting to consciousness 'There's something odd about this woman.'" (p. 155) That is, the person's anomalous experience is an experience with the content that there's something odd about this woman.

While Coltheart's proposal about the anomalous experience in the Capgras delusion is based on a detailed analysis of Ellis and Young's (1990) work on the abnormal autonomic activity, the more general ideas about the prediction and comparison system and the feeling of significance echo Maher's view. Maher (1999) writes:

> Survival requires the existence of a detector of changes in the normally regular patterns of environmental stimuli, namely those that are typically dealt with automatically. The detector functions as a general non-specific alarm, a "significance generator," which then alerts the individual to scan the environment to find out what has changed. (p. 558)

Third, according to what we can call the Endorsement View, the content of the Capgras delusion is already encoded in the anomalous experience (Aimola Davies & Davies, 2009; Davies & Egan, 2013; Langdon & Bayne, 2010). When the person with the Capgras delusion sees his wife, he has the anomalous experience that "This woman looks like my wife but she is not my wife."[2]

Let us compare these three views with Maher's conception of anomalous experience. The Endorsement View is obviously at odds with Maher's view that delusions are normal explanations, because according to the Endorsement View the content of the delusion is already encoded in the anomalous experience and the delusion is hence not an *explanation*, in any ordinary sense, of the anomalous experience. It is worth noting that there are versions of the two-factor theory which accept the Endorsement View (Aimola Davies & Davies, 2009; Davies

[2] It is of some interest to notice the possibilities that a person might have a combination of any of these three conceptions of anomalous experience. These possibilities are, however, under-explored in the literature. This paper will set aside issues related to them.

& Egan, 2013; Langdon & Bayne, 2010). However, since our present concern is the question of whether Maher's conception of anomalous experience is sufficient to explain delusions, I will set aside these versions of the two-factor theory rejecting Maher's conception of anomalous experience.

The Alert View can be taken as being broadly in line with Maher's conception: both can agree that the subject has an experience that "There's something odd about this woman." As for the Affective View, it is not as straightforward as it may appear whether it should be taken as similar to Maher's conception of anomalous experience. On the one hand, Maher himself seems to take it as a candidate for his conception of anomalous experience when he explicitly mentions Ellis and Young's (1990) work. Maher (1999) writes:

> The misidentification syndromes, for example, provide instances in which a focal disturbance of the experience of recognition leads to delusions such as the Capgras, Cotard, and Fregoli syndromes (e.g., Ellis & Young, 1990). Indeed, the model of delusion formation provided in this paper posits that the basic origin lies in the anomalous experience, regardless of how that anomaly arose. (p. 566)

On the other hand, however, there is an important discrepancy between Ellis and Young's conception of anomalous experience and Maher's conception: while according to Maher (1999) the anomalous experience is "a *vague general feeling* [of significance] that prompts us to look for [an explanation]" (p. 554, emphasis in original), according to Ellis and Young (1990) the anomalous experience has a relatively precise content: that is, the feeling of reduced affective responses or the feeling of reduced familiarity in particular. Of course, the feeling of reduced affective responses may make the subject experience a feeling of significance. But this feeling of significance is not vague; rather it is relatively definite in that it is caused by the feeling of reduced affective responses.

Admittedly, the feeling of reduced affective responses may also make the subject wonder what the cause of the reduced affective responses is, and it may appear unclear to the subject what the cause is. But there is no evidence that this kind of wondering and unclarity is normally not associated with the "*vague general feeling"* that, according to Maher, would lead to delusions.

To be clear, I am not saying that it is impossible for people with delusions to have the kind of anomalous experience proposed by Maher; the point is that the feeling of reduced affective responses does not seem to be the same as the anomalous experience proposed by

Maher; it is obviously not caused by the anomalous experience proposed by Maher (rather, it is underpinned by the reduced autonomic activity); nor does it seem to be the cause of the anomalous experience proposed by Maher.

Now we have to make a choice: we may take it that the vague general feeling of significance is the only conception of anomalous experience proposed by Maher, and accordingly take it that in the above quotation Maher simply gives an inaccurate reading of Ellis and Young's conception of anomalous experience; or we may take it that Maher actually proposes that both kinds of anomalous experience, i.e. the vague general feeling of significance and the feeling of reduced affective responses, can be the only factor that causes delusions. Neither option seems ideal. Nonetheless, the second option seems to be a more charitable reading of Maher's one-factor theory. In what follows, I will take the second option, and discuss whether either of these conceptions of anomalous experience can be the only factor that causes delusions.

4. A critique of Maher's one-factor theory

In my view, the arguments concerning whether Maher's conception of anomalous experience is sufficient to explain delusions, by and large, revolve around three issues: the intelligibility of delusions, the dissociation between anomalous experiences and delusions, and the empirical evidence of a second factor. In this section, I will discuss a series of arguments from both proponents and opponents of Maher's one-factor theory, and develop a few new arguments. I will argue that it is unlikely that the anomalous experience, as it is currently understood in the literature, is the only factor in the aetiology of a delusion.

4.1. The intelligibility argument

Jaspers (1913/1997) famously argued that we are unable to "sink ourselves into the psychic situation and *understand … by empathy*" how a delusion emerges from another mental event (p. 301). One important aspect of Jaspers' point is that there is an absence of an intelligible link between delusions and the mental events that are proposed to be their cause. The absence of an intelligible link can be manifested in many respects (Eilan, 2000; Hoerl, 2019). At the centre are the flagrant ways delusions flout the norms concerning the relationship between beliefs and evidence, or the flagrant ways delusions flout evidence for short. This

bafflement clinically defines delusions: according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), "[d]elusions are fixed beliefs that are not amenable to change in light of conflicting evidence." (American Psychiatric Association, 2013, p. 87).

Consider the Capgras delusion: the belief that the patient's wife is an imposter. First, the hypothesis that an ordinary person's wife is replaced by an imposter is implausible in the light of common knowledge (Parrott, 2016), even though it is a familiar topic in movies, science fiction, and mythologies (Christodoulou, 1986). Second, it is at odds with the fact that the "imposter" looks like his wife and knows things that only his wife knows. Third, it is also at odds with the testimonies of his friends and relatives. Fourth, many patients with delusions may have partial insight into the force of the counterevidence and the implausibility of the delusions. Regarding patients with the Capgras delusion, Young (1998) reported: "If you ask 'What would you think if I told you that my wife has been replaced by an impostor?,' you will *often* get answers to the effect that it would be unbelievable, absurd, an indication that you had gone mad." (p. 37, emphasis added)

Can an anomalous experience underpinned by the reduced autonomic activity explain why the patient obstinately believes that his wife is an imposter? In the last section, we have discussed that plausible candidates for the anomalous experience include the experience of reduced affective responses or the experience of reduced familiarity (The Affective View), and the experience that "There's something odd about this woman" (The Alert View). In the light of these anomalous experiences, the imposter hypothesis might be *an* explanation of the anomalous experience in the sense that it can help explain why the patient does not have a normal experience when he sees his wife. But being an explanation itself is not a good reason for the patient to adopt the imposter hypothesis in the light of the significant counterevidence. Moreover, there are lots of other hypotheses which not only are better explanations of the anomalous experience than the imposter hypothesis, but can better deal with the counterevidence. Coltheart (2007), for example, suggests the following two alternative hypotheses:

- The brain-damage hypothesis: "I have suffered a stroke that has disconnected my face recognition system from my autonomic nervous system."
- The fading-love hypothesis: "I no longer love my wife." (p. 1059)

Of course, it is a fact that in the Capgras delusion the patient adopts the imposter hypothesis. But the anomalous experience falls short of explaining why the imposter hypothesis

is adopted in the light of the significant counterevidence and other more plausible hypotheses. One plausible suggestion is that although the anomalous experience is an important factor, it is not sufficient, and some additional factor or factors must be involved in the aetiology of delusions.

4.1.1. Two distinct lines of defences of the one-factor theory and their problems

Defenders of Maher's one-factor theory have offered two distinct lines of responses. One is to argue that the imposter hypothesis is not an implausible explanation; the other is to argue that the anomalous experience is much more anomalous than the proposed anomalous experiences: i.e. the experience of reduced affective responses or the experience of reduced familiarity (The Affective View), and the experience that "There's something odd about this woman" (The Alert View). Let us consider them in turn.

The implausibility of the imposter hypothesis

Regarding the (im)plausibility of the imposter hypothesis, Reimer (2009) writes:

> Although scientists and philosophers (and educated persons more generally) typically regard all such phenomena as equally incredible, that does not mean that they are so regarded by persons generally. Many people believe in the possibility—if not the actuality—of such spectacular things. A few Google searches of (inter alia) aliens, U.F.O.s, Bigfoot, the Loch Ness Monster, and Elvis confirm as much. It is doubtful that only psychiatric patients are visiting these websites. Thus, the patient's general metaphysical beliefs, the rejection of which is entailed by the Impostor Hypothesis, might not be that deeply entrenched after all. (p. 676)

There are two main points in Reimer's argument. One is that educated persons' general metaphysical beliefs are different from less educated persons' general metaphysical beliefs: the former is incompatible with the imposter hypothesis while the latter is compatible with the imposter hypothesis. The other is that patients with the Capgras delusion are less educated persons. If both points are true, then the imposter hypothesis is compatible with patients' general metaphysical beliefs, and hence is not implausible for patients.

It might strike someone as an intuitive view that if a person is less educated or has some abnormal metaphysical beliefs, then they are somewhat more likely to be diagnosed with delusions. But this does not entail that people with delusions, in general, are less educated or have abnormal metaphysical beliefs. Crucially, there is no statistical evidence in support of the view that people with the Capgras delusion are less educated. Nor is there any statistical evidence that the general metaphysical beliefs or knowledge possessed by people with delusions are significantly different from that possessed by people without delusions. In fact, when common knowledge is explicitly examined, there is evidence that people with delusions do not have abnormal knowledge. For example, Young and Leafhead (1996) tested a patient WI who had the Cotard delusion that he was dead. They found that WI's knowledge about being alive or dead did not differ from that of non-delusional control subjects:

> To explore WI's knowledge of whether other people were alive or dead, he was given a simplified version of Kapur's Dead or Alive Test (Kapur et al., 1989). The names of 30 famous people were presented, 10 of whom were alive at the time of testing, with the remaining 20 having died between 1960 and 1989. In each case, WI was asked whether the person was alive or dead. For those he thought had died, he was also asked to give the year in which they died (scored as correct if it belonged to the appropriate decade) and whether they were killed or died of natural causes. [It turned out that] …the performance of WI and eight control subjects matched for age and education (mean age 28 years, SD 2.92 years). For all parts of the test, WI scored as well as the controls. Hence, it is clear that his delusional belief in his own death had not affected WI's general knowledge of whether other people were alive or dead, and was not accompanied by any retrograde amnesia. (p. 156)

Moreover, the fact that many patients have partial insight into the force of counterevidence and the implausibility of their delusions suggests that their general knowledge is not significantly different from non-delusional people (Young, 1998). This point seems also true for patients with schizophrenia. For example, Startup (1997) reported that patients with schizophrenia had "little difficulty" in identifying other patients' delusions as symptoms of mental illness, even though they were unable to apply the same standards to their own delusions (p. 203). Similarly, Feyaerts et al. (2021) reported that, in their interview, patients with schizophrenia "were often well aware that delusional experience would be judged as bizarre or unlikely when set against normal evidential standards." (pp. 794-795) In short, the evidence

suggests that delusions are unlikely to be compatible with patients' general knowledge or general metaphysical beliefs.

The anomaly of the anomalous experience

Let us turn to the arguments appealing to the anomaly of the anomalous experience. Maher (1999) argues that "the kinds of anomalous experience that deluded patients have appeared to be much more *intense* and *prolonged* than those that occur to the population in general." (p. 566, emphasis added) Defenders of Maher's one-factor theory have repeatedly emphasised this point. Reimer (2009), for example, writes: "[T]he experience is widely thought to involve a *profound* feeling of unfamiliarity, of estrangement. This is a feeling that goes well beyond a mere absence of familiarity." (p. 674) However, Maher and Reimer do not tell us about what exactly the intense, prolonged, profound anomalous experience is. Merely insisting that there is such an anomalous experience that is the only factor in the aetiology of delusions offers limited help in explaining delusions.

Sakakibara (2019) offers an illustration of the intense anomalous experience by comparing it to intense emotions:

> [The intense anomalous experiences] are functionally comparable to intense emotions that we sometimes have, in that both work as "irruptive motivation." Strong emotions irrupt into deliberative means-end reasoning and take over one's judgements and actions (Prinz, 2004). For instance, intense fear may cause one to "run away from situations that could be rewarding" (Prinz, 2004, p. 84). Similarly, an intense experience irrupts through deliberative reasoning and rushes the subject into holding delusional thoughts, even though this is not warranted given the totality of evidence. (p. 177)

Suppose it is true that an intense anomalous experience would rush the subject's belief forming processes. But if the subject is in a rush, wouldn't it be more likely that they would rush into some more plausible and available hypotheses, as opposed to the far-fetched delusional hypotheses? In the Capgras delusion, wouldn't it be more likely that the patient rushes to believe the brain-damage hypothesis or the fading-love hypothesis,[3] as opposed to

[3] There might be cases in which these two specific hypotheses are not very plausible candidates. For example, a reviewer for this journal suggests that the fading-love hypothesis might not be a plausible candidate in cases in which the patient experiences that his wife looks and behaves differently. I agree that what would count as a more plausible hypothesis is dependent on the details of the patient's personal experience and the social/cultural context;

the imposter hypothesis? That is, even if we accept that an intense anomalous experience might "irrupt" through the subject's reasoning, appealing to the irruptive nature of anomalous experience still does not tell us why a delusional hypothesis, as opposed to some more plausible and available hypotheses, is adopted by the subject.

It is worth noting that I think Sakakibara's conception of the irruptive nature of anomalous experience could shed extra light on cases in which the content of the delusion is already encoded in the anomalous experience (the Endorsement View). It could help explain why the subject hastily endorses the content of their anomalous experience. Furthermore, it may also help explain why the subject fails to reject the delusion in the light of counterevidence. This might be because it is very difficult for the subject to "maintain a distance from" the irruptive intense anomalous experience (Sakakibara, 2019, p. 178). Nonetheless, it is equally important to remember that the Endorsement View is at odds with Maher's view that delusions are normal explanations (see §3). This is because according to the Endorsement View the content of the delusion is similar to the content of the anomalous experience and the delusion is hence not an *explanation*, in any ordinary sense, of the anomalous experience. That is to say, despite the explanatory power of Sakakibara's conception in the endorsement case, it does not help Maher's one-factor theory explain why a delusional hypothesis, as opposed to other more plausible and available hypotheses, is adopted by the subject to *explain* the anomalous experience.

To the best of my knowledge, in the literature on one-factor and two-factor theories, there has been no satisfactory account of the anomalous experience such that the anomalous experience itself suffices to explain why delusions are formed and maintained in the face of counterevidence. Of course, the lack of such an account, so far, does not prove that it is impossible that there exists anomalous experience of this kind in the light of which delusions are inevitable.[4] But for the one-factor theory to avoid the critique that it is a mere conjecture rather than a highly plausible account, we need to know more about what the anomalous experience is.

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and it might vary from case to case. Nonetheless, I think the general point is still correct that no matter what the more plausible hypothesis is in a particular case, the imposter hypothesis is unlikely to be a very plausible hypothesis that the patient should adopt or rush to believe.

[4] Independently of the tradition following Maher, phenomenologists have offered various accounts of how a distinctive kind of experience itself can be sufficient for the development of a delusion (e.g. Parnas & Henriksen, 2016). The question of how these phenomenological theories can help develop Maher's one-factor theory remains to be seen.

4.2. The dissociation between anomalous experiences and delusions

The standard argument offered by two-factor theorists against Maher's one-factor theory is the dissociation argument (see e.g. Coltheart & Davies, 2021, Table 1). Regarding Maher's one-factor theory, we can discern two variants of the dissociation argument.

The first variant argues that there are cases in which the individuals have an anomalous experience similar to the proposed anomalous experience that gives rise to a delusion, but they do not have the delusion. A second factor is, therefore, hypothesized to explain the delusion. Regarding the Capgras delusion, Turner and Coltheart (2010, pp. 371-372) reported a case (studied by Nora Breen and Mike Salzberg) in which the person had an anomalous experience that was arguably similar to the anomalous experience of people with the Capgras delusion, but the person did not have the Capgras delusion.

The second variant argues that there are cases in which the individuals have a neuropsychological deficit similar to the neuropsychological deficit underpinning the anomalous experience that gives rise to a delusion, but they do not have the delusion. A second factor is, therefore, hypothesized to explain the delusion (see e.g. Davies & Egan, 2013, p. 691). Regarding the Capgras delusion, there are individuals with ventromedial frontal lesions who have reduced autonomic responses to familiar faces and probably have an anomalous experience similar to that in the Capgras delusion, but they do not have the Capgras delusion (Tranel et al., 1995).

Some defenders of Maher's one-factor theory have attempted to reject the premise that the anomalous experience and/or the underpinning neuropsychological deficit in delusional cases are similar to those in non-delusional cases. For example, they may emphasise the possibility that the "anomalous" experiences in delusional cases are *somehow* significantly different from the anomalous experiences in non-delusional cases (Franceschi, 2010; Reimer, 2009; Sakakibara, 2019; Sullivan-Bissett, 2020). If this is the case, then there might be no need to appeal to a second factor to explain delusions. The problem with this line of defence is, however, that such a possibility has not been supported by empirical evidence, particularly when monothematic delusions are concerned.[5] By contrast, as we have seen, two-factor

[5] This is not to say that there is no empirical evidence that is compatible with the one-factor theory. For example, as a reviewer for this journal points out, the effectiveness of pharmacotherapy in alleviating schizophrenic delusions can be well explained by the one-factor theory. It might be that anti-dopaminergic drugs alleviate schizophrenic delusions by alleviating the intensity of the anomalous experience. However, this sort of evidence is equally compatible with the two-factor theory. This is because the two-factor theory accepts that the anomalous experience is one of the two necessary factors and hence by changing the anomalous experience anti-dopaminergic

theorists have offered empirical evidence for the premise of the dissociation argument (Turner & Coltheart, 2010; Davies & Egan, 2013). Overall, it seems the empirical evidence we have so far is in favour of the dissociation argument.

These two variants of the dissociation argument suggest that the proposed first factor, i.e. the anomalous experience, may not be sufficient, and a second factor may help explain delusions. But, importantly, the dissociation arguments do not entail that the second factor is necessarily a reasoning abnormality. Nor do they preclude the possibility that there is more than one missing factor. As far as the dissociation arguments are concerned, it is also possible that the missing factor is not a reasoning abnormality but some other kind of neuropsychological abnormality.

It is worth emphasising that the dissociation argument only shows that the proposed first factor is not sufficient because of the *single* dissociation cases in which the proposed first factor is present, but the corresponding delusion is not. The second factor is proposed to help explain the single dissociation cases. The dissociation argument does not, and does not need, to make a further claim about the relationship between the nature of the first factor and that of the second factor: for example, it does not make a claim about whether there is some neuroanatomical overlap between them, whether there is some overlap between the cognitive underpinnings of the first factor and those of the second factor, or whether there is some degree of interaction between the first and second factors, let alone a double-dissociation relationship between them.

4.3. The empirical evidence of reasoning abnormalities

Maher's one-factor theory was supported by the repeated failure of empirical studies to find logical reasoning abnormalities in schizophrenia (Mujica-Parodi et al., 2000; Maher, 2001). Since then, however, more studies have been developed and a variety of reasoning abnormalities have been found to be associated with delusions. In a recent review of empirical studies, McLean et al. (2017) argue that delusions are associated with the jumping to conclusions bias, the bias against disconfirmatory evidence, the bias against confirmatory evidence, and liberal acceptance. Based on their own empirical studies of anosognosia (with which the subject may have the delusion that their paralysed arm is not paralysed but normal)

drugs can affect the delusion. What defenders of the one-factor theory really need is a different sort of evidence that can only be explained by the one-factor theory but cannot be explained by the two-factor theory.

and the review of the literature, Aimola Davies and colleagues argue that people with delusions suffer from an impairment of working memory and/or executive function, which are essential for reasoning processes (Aimola Davies et al., 2009; Aimola Davies & Davies, 2009). These new empirical findings suggest that some reasoning abnormalities may be an important factor in the aetiology of delusions.[6]

Many defenders of the one-factor theory do not deny the validity of these empirical studies. Instead, they argue that the reasoning abnormalities in delusions are nonetheless within the normal range. To see their points, we need to take a closer look at their arguments. In the following, I will discuss what I will call the meta-theory argument and the analogical argument in defence of the one-factor theory.

4.3.1. The meta-theory argument in defence of the one-factor theory and its problems

In her defence of Maher's one-factor, Sullivan-Bissett (2020) argues that people with delusions "have normal-range reasoning applied to abnormal experiences." (p. 683) What is a "normal-range" reasoning process? How should we distinguish a normal-range reasoning process from an abnormal-range reasoning process? Sullivan-Bissett does not offer an answer. Instead, she argues that the burden is equally on two-factor theorists "since [they argue] that there is some cognitive feature of subjects who have delusions which is abnormal and differentiates them from the non-delusional population." (p. 684)

From the perspective of two-factor theorists, there is a clear difference between how people with delusions reason and how people without delusions reason, and the reasoning in delusions falls outside of the normal range.[7] However, Sullivan-Bissett is correct that without a meta-theory of what counts as a normal-range reasoning process, one-factor theorists could insist that the reasoning in delusions still "fall[s] into the normal range" (p. 683). Call this the meta-theory argument.

[6] The mere association between reasoning abnormalities and delusions does not by itself establish that reasoning abnormalities are necessary in the aetiology of delusions. Two-factor theorists also need to theoretically explain why reasoning abnormalities are necessary (see e.g. Davies & Egan, 2013). However, given this paper focuses on critically analysing the arguments developed by defenders of Maher's one-factor theory and does not aim to offer a comprehensive defence of a specific version of the two-factor theory, I will set aside the latter discussion.

[7] This is certainly not to deny that the formation and maintenance of delusions may also involve certain normal-range reasoning, such as motivated reasoning (Davies, 2009; Rigoli et al., 2021). Two-factor theorists' key hypothesis is that certain abnormal reasoning must play an important role.

How to break the impasse? One way, of course, is to develop a meta-theory of what counts as a normal-range reasoning process. I shall not take this approach. Instead, I argue that appealing to the meta-theory argument would do more harm than benefit to the one-factor theory.

To the best of my knowledge, there is no meta-theory of what counts as an anomalous experience. So, following a similar meta-theory argument, a zero-factor theorist can argue that the anomalous experience falls within the normal range and hence we should reject the one-factor theory as well.

There is no widely accepted meta-theory of what counts as a delusional belief either (Bortolotti et al., 2016, p. 48). So, following another similar meta-theory argument, a delusion-denialist (compare Szasz, 1974) could argue that delusional beliefs fall within the normal range, and hence there is no need to develop any theory of delusions in particular: the existing theories of normal beliefs are sufficient for explaining delusions.

Here is a dilemma for one-factor theorists. On the one hand, if they retain the meta-theory argument, then they can defend the one-factor theory against the two-factor theory, but they have to explain why the zero-factor theorist and the delusion-denialist are wrong. It is far from clear how this can be done because the zero-factor theorist and the delusion-denialist are using similar meta-theory arguments. On the other hand, if they give up the meta-theory argument, then they do not have to deal with the zero-factor theorist and the delusion-denialist, but their defence of the one-factor theory will be lost as well. For now, it seems that appealing to the notion of *abnormal-range* factors is not an attractive defence of the one-factor theory.[8]

4.3.2. The analogical argument in defence of the one-factor theory and its problems

By appealing to Cassam and others' work on conspiracy theories, Noordhof and Sullivan-Bissett (2021) offer an analogical argument that the irrationality in delusions is within the normal range. They write:

> Consider also conspiracy theories, which we will understand as explanations of events that appeal to the intentional states of conspirators, who intended the event and kept their intentions and actions secret (Mandik, 2007, p. 206). Those who believe in such theories—so-called conspiracy theorists—are prime

[8] This critique of Sullivan-Bissett's notion of *abnormal-range* factors applies to Noordhof and Sullivan-Bissett's (2021) notion of *clinically abnormal* factors.

> examples of epistemically irresponsible subjects whose beliefs seem utterly impervious to counterevidence. As Quassim Cassam points out, 'there aren't too many examples of committed conspiracy theorists changing their minds' (2019, p. 93). Conspiracy theorists are especially relevant to discussion here since, perhaps similarly to some monothematic delusions, '[t]here is almost no explanation that isn't too bizarre for the conspiracy theorist's taste' (Cassam, 2019, p. 22). … many researchers interested in conspiracy theorists make no claims about clinically abnormal cognition, rather, they appeal to individual differences in personality to explain being conspiracy-minded (see Cassam, 2019, pp. 40–43 for discussion) or as involving a particular worldview (Keeley, 1999, p. 123, Cassam, 2019, p. 100). Such normal range irrationality is the kind of thing which can contribute to such thinkers displaying epistemic irresponsibility. … Similar things can be said for folk with monothematic delusions. … the epistemic irresponsibility displayed by such subjects is representative of normal range irrationality… (pp. 10302-10303)

Their argument seems to go as follows: The kind of epistemic irresponsibility in delusions is the same as the kind of epistemic irresponsibility in conspiracy theories; normal-range irrationality, by which Noordhof and Sullivan-Bissett (2021) mean, e.g. the differences in personality, can explain the kind of epistemic irresponsibility in conspiracy theories; therefore, normal-range irrationality can explain the kind of epistemic irresponsibility in delusions.

While it is true that both delusions and conspiracy theories are notorious types of beliefs that are impervious to counterevidence, and in this very general sense the subjects in both cases are epistemically irresponsible, it does not follow that the kind of epistemic irresponsibility in delusions is the same as the kind of epistemic irresponsibility in conspiracy theories. There is a wide range of types of beliefs that are impervious to counterevidence, which, besides delusions and conspiracy theories, may also include akratic beliefs (Heil, 1984), superstitious beliefs (Scheibe & Sarbin, 1965), and some of the religious beliefs (McKay & Ross, 2020). In spite of the fact that these beliefs are all impervious to counterevidence, and in this very general sense the subjects with any of these beliefs may be taken as being epistemically irresponsible, it is, however, unlikely that the specific kinds of epistemic irresponsibility in all these beliefs are the same.

We might have some reason for thinking that the same normal-range irrationality could explain delusions if it turned out that the specific way delusions are impervious to

counterevidence is the same as the specific way conspiracy theories are impervious to counterevidence. A closer comparison between Cassam and others' account of conspiracy theories and what we already know about delusions, however, shows that the latter is not true.

There are many significant differences between the epistemic irresponsibility in conspiracy theories and the epistemic irresponsibility in delusions. For example, conspiracy theories are often politically, financially, or ideologically motivated, whereas delusions are not; Conspiracy theories are based on the premodern view that "complex events are capable of being controlled by a small number of people acting in secret, and that this is what gives these events a deeper meaning" (Cassam, 2019, p. 26), whereas delusions lack such a base. Conspiracy theories are often shared by a group of individuals or within a certain community, whereas there is no "community united in common delusions" (Jaspers, 1913/1997, p. 284). These differences already suggest that the irrational factors in delusions are much more severe than the so-called normal-range irrationality in conspiracy theories.

Furthermore, when evidence is concerned, Cassam (2019, Chapter 4; 2020) argues that conspiracy theories are impervious to counterevidence in a *self-sealing* way. Cassam (2020) writes:

> [T]he arguments that give rise to them [i.e. conspiracy theories] are designed to be immune to refutation. Contrary evidence is attributed to the conspiracy and the absence of evidence of conspiracy is taken as evidence of the skill of the conspirators. (p. 3)

For example, for conspiracy theorists who believe that the Bush administration is behind the 9/11 attacks, the official report of the 9/11 Commission is part of the conspiracy, and the lack of evidence that the Bush administration is behind the 9/11 attacks is the evidence that the Bush administration is very good at hiding their conspiracy.

Are delusions impervious to counterevidence in such a self-sealing way? It does not seem so. It is not a characteristic of delusions that the subjects take the absence of evidence as evidence that their delusions are true. Nor is it a characteristic of delusions that the subjects attribute contrary evidence to their delusions. Rather, they can often recognise contrary evidence as contrary evidence, even though they do not change their delusions in the face of contrary evidence. As Corlett and Fletcher (2021) write:

> [P]atients with monothematic delusions appear to understand what sort of evidence might undermine their beliefs but do not modify them in the face of

> such evidence. They do not appear to take on and use such disconfirmatory evidence. This, of course, is one of the key criteria for establishing that a delusion is present so presumably, *if the patients did not show this effect, they would not be considered to be deluded*. (p. 233, emphasis added)

The self-sealing feature of conspiracy theories indicates that the evidence that is at odds with conspiracy theories is, in a sense, explained away in a logically coherent, albeit unsound, way. By contrast, the evidence that is at odds with delusions is not explained away: instead, delusions are held in the face of counterevidence. This significant difference between the way conspiracy theories are impervious to counterevidence and the way delusions are impervious to counterevidence suggests that, even though the kind of irrational factors in conspiracy theories is within the normal range, it does not follow that the irrational factors in delusions are within the normal range. On the contrary, the flagrant ways delusions flout evidence suggest that if some irrational factors, such as the reasoning abnormalities that are empirically demonstrated to be associated with delusions, play important roles in the aetiology of delusions, then they are likely to be beyond the normal range.

5. Conclusion

This paper conducted a critical analysis of the arguments regarding whether Maher's conception of anomalous experience as the only departure from normality is sufficient to explain delusions. If the analysis is along the right lines, then it is likely that some additional factor or factors, i.e. departures from normality, may be involved in the aetiology of delusions; and the two-factor theory is on the right track in its search for the missing factor in the aetiology of delusions.

This, of course, does not entail that the two-factor theory is without its own challenges (for critique, see e.g. Corlett, 2019; Parrott, 2016; for defence, see e.g. McKay, 2019; Nie, 2016, 2019). Nevertheless, the challenges faced by the two-factor theory should not be taken as reasons that we have to return to Maher's one-factor theory. The arguments and evidence presented in this paper suggest that reverting to Maher's one-factor theory is not a promising option. Instead, I suggest that a better way to make some progress is to focus on searching for the missing factor, for which reasoning abnormality is a candidate proposed by the two-factor theory, and at the same time keep an open mind to the possibility that there could be some missing factor not yet captured by existing theories of delusions.

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References:

Aimola Davies, A. M., & Davies, M. (2009). Explaining pathologies of belief. In M. R. Broome & L. Bortolotti (Eds.), *Psychiatry as cognitive neuroscience* (pp. 284-324). Oxford University Press

Aimola Davies, A. M., Davies, M., Ogden, J. A., Smithson, M., & White, R. C. (2009). Cognitive and motivational factors in anosognosia. In T. Bayne & J. Fernandez (Eds.), *Delusion and self-deception: Affective and motivational influences on belief formation* (pp. 187-225). Psychology Press

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (dsm-5)*. American Psychiatric Association

Bauer, R. M. (1984). Autonomic recognition of names and faces in prosopagnosia: A neuropsychological application of the guilty knowledge test. *Neuropsychologia*, *22*(4), 457-469.

Berrios, G. E. (1991). Delusions as "wrong beliefs": A conceptual history. *British Journal of Psychiatry*, *159*(S14), 6-13.

Bobes, M. A., Gongora, D., Valdes, A., Santos, Y., Acosta, Y., Fernandez Garcia, Y., Lage, A., & Valdes-Sosa, M. (2016). Testing the connections within face processing circuitry in capgras delusion with diffusion imaging tractography. *Neuroimage Clin*, *11*, 30-40.

Bortolotti, L., Sullivan-Bissett, E., & Gunn, R. (2016). What makes a belief delusional? In I. McCarthy, K. Sellevold, & O. Smith (Eds.), *Cognitive confusions: Dreams, delusions and illusions in early modern culture* (pp. 37-51). Modern Humanities Research Association

Braun, C. M. J., & Suffren, S. (2011). A general neuropsychological model of delusion. *Cognitive Neuropsychiatry*, *16*(1), 1-39.

Breen, N., Caine, D., & Coltheart, M. (2000). Models of face recognition and delusional misidentification: A critical review. *Cognitive Neuropsychology*, *17*(1), 55-71.

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), 189-197.

Capgras, J., & Reboul-Lachaux, J. (1923). L'illusion des "sosies" dans un délire systématisé chronique. *Bulletin de la Société Clinique de Médicine Mentale*, *2*, 6–16.

Cassam, Q. (2019). *Conspiracy theories*. Polity Press

Cassam, Q. (2020). *Philosophy for the real world: Knowledge, ignorance and bullshit [lecture 3, class handout]*. University of Warwick, Coventry, UK.

Christodoulou, G. N. (1986). The origin of the concept of 'doubles'. In G. N. Christodoulou (Ed.), *The delusional misidentification syndromes* (pp. 1-8). Karger

Coltheart, M. (2005). Conscious experience and delusional belief. *Philosophy, Psychiatry, & Psychology*, *12*(2), 153-157.
Coltheart, M. (2007). The 33rd bartlett lecture: Cognitive neuropsychiatry and delusional belief. *Quarterly Journal of Experimental Psychology*, *60*(8), 1041-1062.
Coltheart, M., & Davies, M. (2021). Failure of hypothesis evaluation as a factor in delusional belief. *Cognitive Neuropsychiatry*, *26*(4), 213-230.
Coltheart, M., & Davies, M. (2022). What is capgras delusion? *Cognitive Neuropsychiatry*, *27*(1), 69-82.
Coltheart, M., Langdon, R., & McKay, R. (2007). Schizophrenia and monothematic delusions. *Schizophrenia Bulletin*, *33*(3), 642-647.
Coltheart, M., Menzies, P., & Sutton, J. (2010). Abductive inference and delusional belief. *Cognitive Neuropsychiatry*, *15*(1), 261-287.
Connors, M. H., & Halligan, P. W. (2020). Delusions and theories of belief. *Consciousness and Cognition*, *81*, Article 102935.
Corlett, P. R. (2019). Factor one, familiarity and frontal cortex: A challenge to the two-factor theory of delusions. *Cognitive Neuropsychiatry*, *24*(3), 165-177.
Corlett, P. R., & Fletcher, P. (2021). Modelling delusions as temporally-evolving beliefs. *Cognitive Neuropsychiatry*, *26*(4), 231-241.
Cotard, J. (1882). Du délire des négations. *Archives de Neurologie*, *4*, 152–170, 282–195.
Davies, M. (2009). Delusion and motivationally biased belief: Self-deception in the two-factor framework. In T. Bayne & J. Fernandez (Eds.), *Delusion and self-deception: Affective and motivational influences on belief formation* (pp. 71-86). Psychology Press
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 compartmentalisation. In K. W. M. Fulford, M. Davies, R. G. T. Gipps, G. Graham, J. Z. Sadler, G. Stanghellini, & T. Thornton (Eds.), *The oxford handbook of philosophy and psychiatry* (pp. 689-727). Oxford University Press
Eilan, N. (2000). On understanding schizophrenia. In D. Zahavi (Ed.), *Exploring the self: Philosophical and psychopathological perspectives on self-experience* (pp. 97-113). John Benjamins Publishing
Ellis, H. D. (1989). Past and recent studies of prosopagnosia. In J. R. Crawford & D. M. Parker (Eds.), *Developments in clinical and experimental neuropsychology* (pp. 151-166). Springer
Ellis, H. D., & de Pauw, K. W. (1994). The cognitive neuropsychiatric origins of the capgras delusion. In A. S. David & J. C. Cutting (Eds.), *The neuropsychology of schizophrenia* (pp. 317-335). Psychology Press
Ellis, H. D., Lewis, M. B., Moselhy, H. F., & Young, A. W. (2000). Automatic without autonomic responses to familiar faces: Differential components of covert face recognition in a case of capgras delusion. *Cognitive Neuropsychiatry*, *5*(4), 255-269.
Ellis, H. D., Whitley, J., & Luaute, J.-P. (1994). Delusional misidentification: The three original papers on the capgras, fregoli and intermetamorphosis delusions. *History of Psychiatry*, *5*(17 Pt 1), 117-146.
Ellis, H. D., & Young, A. W. (1990). Accounting for delusional misidentifications. *British Journal of Psychiatry*, *157*(2), 239-248.
Ellis, H. D., Young, A. W., Quayle, A. H., & De Pauw, K. W. (1997). Reduced autonomic responses to faces in capgras delusion. *Proceedings: Biological Sciences*, *264*(1384), 1085-1092.

Feyaerts, J., Kusters, W., Van Duppen, Z., Vanheule, S., Myin-Germeys, I., & Sass, L. (2021). Uncovering the realities of delusional experience in schizophrenia: A qualitative phenomenological study in belgium. *Lancet Psychiatry*, *8*(9), 784-796.

Franceschi, P. (2010). A logical defence of maher's model of polythematic delusions. *Journal of Philosophical Research*, *35*(2).(English translation and postprint of a paper originally published in French in *Philosophiques*, 2008, vol. 35-2, pp. 451-475)

Frith, C. D. (1992). *The cognitive neuropsychology of schizophrenia*. Psychology Press

Heil, J. (1984). Doxastic incontinence. *Mind*, *XCIII*(369), 56-70.

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

Hoerl, C. (2019). Understanding and explaining in psychiatry. In G. Stanghellini, M. Broome, A. V. Fernandez, P. Fusar-Poli, A. Raballo, & R. Rosfort (Eds.), *The oxford handbook of phenomenological psychopathology* (pp. 407-413). Oxford University Press

Horne, Z., Muradoglu, M., & Cimpian, A. (2019). Explanation as a cognitive process. *Trends in Cognitive Sciences*, *23*(3), 187-199.

James, W. (1890/1952). *The principles of psychology*. Encyclopædia Britannica, Inc.

Jaspers, K. (1913/1997). *General psychopathology* (J. Hoenig & M. W. Hamilton, Trans.). Johns Hopkins University Press

Kapur, N., Young, A., Bateman, D., & Kennedy, P. (1989). Focal retrograde amnesia: A long term clinical and neuropsychological follow-up. *Cortex*, *25*(3), 387-402.

Langdon, R., & Bayne, T. (2010). Delusion and confabulation: Mistakes of perceiving, remembering and believing. *Cognitive Neuropsychiatry*, *15*(1), 319-345.

Maher, B. A. (1974). Delusional thinking and perceptual disorder. *Journal of Individual Psychology*, *30*(1), 98-113.

Maher, B. A. (1988). Anomalous experience and delusional thinking: The logic of explanations. In T. F. Oltmanns & B. A. Maher (Eds.), *Delusional beliefs* (pp. 15-33). John Wiley and Sons

Maher, B. A. (1992). Delusions: Contemporary etiological hypotheses. *Psychiatric Annals*, *22*(5), 260-268.

Maher, B. A. (1999). Anomalous experience in everyday life: Its significance for psychopathology. *Monist*, *82*(4), 547-570.

Maher, B. A. (2001). Delusions. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (pp. 309-339). Springer

Maher, B. A. (2006). The relationship between delusions and hallucinations. *Current Psychiatry Reports*, *8*(3), 179-183.

Mandik, P. (2007). Shit happens. *Episteme*, *4*(2), 205-218.

McKay, R. (2019). Measles, magic and misidentifications: A defence of the two-factor theory of delusions. *Cognitive Neuropsychiatry*, *24*(3), 183-190.

McKay, R. T., & Ross, R. M. (2020). Religion and delusion. *Current Opinion in Psychology*, *40*, 160-166.

McLean, B. F., Mattiske, J. K., & Balzan, R. P. (2017). Association of the jumping to conclusions and evidence integration biases with delusions in psychosis: A detailed meta-analysis. *Schizophrenia Bulletin*, *43*(2), 344-354.

Mujica-Parodi, L. R., Malaspina, D., & Sackeim, H. A. (2000). Logical processing, affect, and delusional thought in schizophrenia. *Harvard Review of Psychiatry*, *8*(2), 73-83.

Nie, C. (2016). Delusional beliefs, two-factor theories, and bizarreness. *Frontiers of Philosophy in China*, *11*(2), 263-278.

Nie, C. (2019). Continuing commentary: Challenges or misunderstandings? A defence of the two-factor theory against the challenges to its logic. *Cognitive Neuropsychiatry*, *24*(4), 300-307.
Noordhof, P., & Sullivan-Bissett, E. (2021). The clinical significance of anomalous experience in the explanation of monothematic delusions. *Synthese*, *199*(3-4), 10277-10309.
Nuara, A., Nicolini, Y., D'Orio, P., Cardinale, F., Rizzolatti, G., Avanzini, P., Fabbri-Destro, M., & De Marco, D. (2020). Catching the imposter in the brain: The case of capgras delusion. *Cortex*, *131*, 295-304.
Parnas, J., & Henriksen, M. G. (2016). Epistemological error and the illusion of phenomenological continuity. *World Psychiatry*, *15*(2), 126-127.
Parrott, M. (2016). Bayesian models, delusional beliefs, and epistemic possibilities. *The British Journal for the Philosophy of Science*, *67*(1), 271-296.
Prinz, J. J. (2004). *Gut reactions: A perceptual theory of emotion*. Oxford University Press
Reimer, M. (2009). Is the impostor hypothesis really so preposterous? Understanding the capgras experience. *Philosophical Psychology*, *22*(6), 669-686.
Rigoli, F., Martinelli, C., & Pezzulo, G. (2021). I want to believe: Delusion, motivated reasoning, and bayesian decision theory. *Cognitive Neuropsychiatry*, *26*(6), 408-420.
Roessler, J. (forthcoming). Thought insertion and the ontology of thinking In P. López-Silva & T. McClelland (Eds.), *Intruders in the mind: Interdisciplinary perspectives on thought insertion*. Oxford University Press
Sakakibara, E. (2019). Intensity of experience: Maher's theory of schizophrenic delusion revisited. *Neuroethics*, *12*(2), 171-182.
Sass, L. A., & Pienkos, E. (2013). Delusion: The phenomenological approach. In K. W. M. Fulford, M. Davies, R. G. T. Gipps, G. Graham, J. Z. Sadler, G. Stanghellini, & T. Thornton (Eds.), *Oxford handbook of philosophy and psychiatry* (pp. 632-657). Oxford University Press
Scheibe, K. E., & Sarbin, T. R. (1965). Towards a theoretical conceptualisation of superstition. *The British journal for the philosophy of science*, *16*(62), 143-158.
Startup, M. (1997). Awareness of own and others' schizophrenic illness. *Schizophrenia Research*, *26*(2), 203-211.
Stone, T., & Young, A. W. (1997). Delusions and brain injury: The philosophy and psychology of belief. *Mind & Language*, *12*(3-4), 327-364.
Sullivan-Bissett, E. (2020). Unimpaired abduction to alien abduction: Lessons on delusion formation. *Philosophical Psychology*, *33*(5), 679-704.
Sullivan-Bissett, E. (2022). Against a second factor. *Asian Journal of Philosophy*, *1*(1).
Szasz, T. S. (1974). *The myth of mental illness: Foundations of a theory of personal conduct* (Revised ed.). Perennial Library
Tranel, D., Damasio, H., & Damasio, A. R. (1995). Double dissociation between overt and covert face recognition. *Journal of Cognitive Neuroscience*, *7*(4), 425-432.
Turner, M., & Coltheart, M. (2010). Confabulation and delusion: A common monitoring framework. *Cognitive Neuropsychiatry*, *15*(1), 346-376.
Young, A. W. (1998). *Face and mind*. Oxford University Press
Young, A. W., & Leafhead, K. M. (1996). Betwixt life and death: Case studies of the cotard delusion. In P. W. Halligan & J. C. Marshall (Eds.), *Method in madness: Case studies in cognitive neuropsychiatry* (pp. 147-171). Lawrence Erlbaum Associates