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The One-Factor Theory

This chapter will consider the one-factor approach to delusion formation. I begin by examining what is meant by *factor,* since this is obviously crucial to a proper discussion of the approach's merits, particularly as part of a wider debate with a rival theory which argues for two factors. I then consider delusional beliefs as *explanations* of anomalous experiences (a claim shared by one- and two-factor theorists), before identifying the point of disagreement: the one-factor theory's claim that such explanations are *normal* ones. The idea of delusional beliefs as normal explanations naturally gives rise to concerns regarding hypothesis selection, that is, selection of the delusional hypothesis might indicate the need for a second factor. I discuss this in the service of properly understanding the commitments and resources of the one-factor theory. Finally, I turn to the most common objection to the view: the objection from dissociation. I offer two responses before concluding that the one-factor approach should be the default approach to understanding the genesis and maintenance of monothematic delusions.

1. Preliminaries

One-factor theories of delusion formation are a branch of a wider view, *empiricism,* according to which anomalous experiences are part of the causal story for the formation of a delusion (see Bongiorno & Parrott, Ch. 26). One-factor versions of empiricism have it that the anomalous experience is the only factor to which we need to appeal to explain why someone forms or maintains a delusional belief. Two-factor theories also fall under the empiricist umbrella, accepting the story so far, but adding a second factor to explain delusion in the form of a reasoning bias, deficit, or performance error (see Davies & Coltheart, Ch. 29). This chapter will focus on the one-factor approach, with particular emphasis on how its resources have been underestimated by its opponents. I will focus on *monothematic* delusions (hereafter simply 'delusions'), because the recent debate between one- and two-factor theorists has taken place in this context (two-factor theorists also restrict their remits in this way, see e.g. Davies et al. 2001: 137, Coltheart et al. 2011: 282; Coltheart 2013: 103, Coltheart & Davies 2021: 225–6).[1] I will also take for granted doxasticism about delusion, that is, I take it that delusions are *beliefs*. Here again, I am following the convention set by the debate within empiricism (for defences of doxasticism see Bayne & Pacherie 2005, Bortolotti 2009, and Noordhof, Ch. 19).

2. Factors and abnormality

It is, of course, crucial to be precise regarding what is meant by *factor* in a piece discussing factors in delusion. In turn, it will be equally crucial to be precise about what is meant by *abnormality.*

One- and two-factor accounts take themselves to be identifying features of the context that are explanatorily relevant to *delusional* belief formation and maintenance in particular. The task isn't to identify all the background features and cognitive contributions of everyday belief formation, *and then* add in whatever other versions of those things are also required to explain belief which is properly characterized as *delusional.* Rather, one- and two-factor theorists alike begin with a shared background of ordinary belief formation, and then take their task to be one of identifying what *else* we need to explain the genesis and maintenance of belief of a particular kind. All sides can agree that in the ordinary case of garden variety beliefs about cats on mats and grass being green, we are zero-factor

theorists. When we turn to delusion, those extra ingredients required in our explanation are the *factors,* and the disagreement lies in how many of those we need.

Factors then are not merely causal contributions. The task is not to create an inventory of everything to which we need to appeal in order to explain delusional belief. Even in everyday cases, if that were the task, the inventory would be substantial. We would need to appeal to the subject having oxygen in her environment, having suitably developed cognitive capacities to form beliefs, being in such-a-such a place *p* at such-and-such a time *t* to receive evidence *e,* and so on. Background conditions are not factors. Nor, importantly, are various quirks of cognition that might go into an explanation of why folk have particular beliefs. My Uncle Mark's belief that *the earth is flat* can be in part explained by appeal to the epistemic bubbles and chambers in which he is immersed (Nyguen 2020), as well as the exercise of, say, intentionality bias (Brotherton 2015: 188–9) or need for uniqueness (Imhoff & Lamberty 2017). My Auntie Eileen's belief that *the positions of celestial bodies influence the trajectory of human relationships* can be in part explained by her preference for non-naturalistic explanations of various phenomena, and her engagement with numerous astrological media. My Grandmother's belief that *poltergeists inhabit her home* can be in part explained by high intuitive and low analytical thinking (Lindeman & Aarnio 2006). These idiosyncrasies are also not factors. They are simply part of the wide catalogue of quirks and tendencies in human psychology. If our task were a causal inventory, they would make an appearance. But that is not our task.

How then should a factor be understood if mere causal contribution is not what we mean? In addition to causally contributing to the formation or maintenance of a delusional belief, the term *factor* picks out a contribution which is *abnormal.* Two-factor theorists have often recognised this. Tony Stone and Andrew Young talk of delusional reasoning being 'abnormal' and 'differences between people with and without delusions' (Stone & Young 1997: 342). Martin Davies and colleagues characterise the second factor as 'a departure from what is normally the case' (Davies et al. 2005: 228). Ryan McKay and colleagues characterise the deficit two-factor approach as one which 'conceptualises delusions as involving dysfunction or disruption in ordinary cognitive processes' (McKay et al. 2010: 316–17). Finally, in discussing how to defend the one-factor approach Philip Gerrans suggests showing the second factor to in fact be describing 'a rationalization process which is within the normal range' (Gerrans 2002: 48). This wouldn't be a defence of the one-factor approach if the second factor were not proposed to constitute an abnormality.[2]

Of course, understanding *factor* as picking out a causal contribution to delusion which is *abnormal* raises a new question: how should we understand *normality*? There are broadly two ways: functionally or statistically. Take *functional normality* to be picking out the property of being within the range of reasoning styles between which evolutionary selection has not distinguished, and *functional abnormality* the opposite. Take *statistical normality* to be picking out the property of occurring in non-delusional populations, and *statistical abnormality* the opposite. Often these notions of normality will not characterise different sets of belief, that is, where we find functional normality we also find statistical normality, and vice versa. But they are separable, and it's important to be clear on which is in play when we're seeking to adjudicate between one- and two-factor theories. For example, suppose that a particular style of reasoning *R* were functionally normal, but occurred in all and only people with delusions. Were we working with a statistical notion of abnormality, *R* would be a factor. Were we working with a functional notion of abnormality, it would not. In what follows I will understand two-factor theorists as seeking to identify a functional abnormality against a statistical assumption (that is, functional

abnormalities purported to be involved in delusion are also taken to be statistical abnormalities).[3]

Now that we know how to understand what is meant by *factor,* we can be more precise in stating the commitments of the one- and two-factor approaches. One-factor theories of delusion have it that to explain the formation or maintenance of a delusional belief, along with a range of background conditions, we can[4] appeal to an abnormal anomalous experience. Two-factor theories add the requirement of an abnormality in belief formation or evaluation in light of such an experience.

3. Delusions as explanations of experience

As already noted, subjects with delusions often undergo some profoundly anomalous experiences, and it is the recognition that these experiences are explanatorily relevant to the project of understanding the formation and maintenance of these beliefs that is the backbone of the empiricist approach. Let us see some examples to get a sense of what subjects may be labouring under.

A subject with perceptual delusional bicephaly (the belief that *one has a second head*) may hallucinate a second head on her shoulder (Ames 1984). Not all anomalous experiences are hallucinatory in this way, that is, they do not all present objects and properties in the world that are not really there. In Capgras delusion (the belief that *someone familiar*, often a loved one, *has been replaced by an imposter*), the experience has been understood as the *absence* of something expected. In particular, the subject has reduced affective response to familiar faces traceable to ventromedial prefrontal cortex damage (Tranel, Damasio, & Damasio 1995, Coltheart 2007), or right lateral temporal lesions and dorsolateral prefrontal cortex damage (Wilkinson 2015, Corlett 2019). In the case of Cotard delusion (the belief that *one is dead* or *has ceased existing*), similar damage has been found and it has been suggested that these subjects have no emotional feelings regarding their environment (Young et al. 1992: 800).

Coltheart and colleagues identify the key point of the one-factor theory to be the claim that 'delusional beliefs are normal attempts to explain abnormal perceptual or affective phenomena' (Coltheart et al. 2011: 284). They trace this idea back to William James, their evidence for doing so is given in the following quotation:

> 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. (James 1890: chapter XIX, 114, fn. 122)

Coltheart and colleagues take Brendan Maher to be the inheritor of the view, capturing it as the claim that '[a] delusion is a hypothesis designed to explain unusual perceptual phenomena' (Maher 1974: 103). However, these quotations from James and Maher in fact only get us to the idea that delusional beliefs are explanations of anomalous experience. This is consistent with the two-factor theory. Two-factor theorists can accept the insight from James and Maher that delusions arise from attempts to explain abnormal data, but they build on that insight by insisting that we need another factor to explain why the explanation is taken up in belief, or why the belief is maintained.

What distinguishes the one-factor theory is the claim that the ways in which these explanations of experience are formed and maintained is *normal.* That is, whatever cognitive influences are involved in the formation or maintenance of a delusion, they are

within the normal range for human psychology. All sides can accept the idea of delusional hypotheses as explanations of experience, the point of divergence is on whether we need to appeal to any abnormality to explain the adoption and maintenance of those hypotheses (or even their generation, see discussion in next section). The two-factor account says that we do. The one-factor account that we do not. Let us turn to Maher's view to further elucidate this key idea.

4. Delusions as *normal* explanations for experience

Maher defended the idea that delusions are adopted as explanations for experience and are 'developed in much the same way that normal beliefs are' (Maher 1988: 22). As Coltheart notes, although Maher's view was primarily concerned with explaining the adoption of delusions of reference (and usually in the context of schizophrenia), his approach can nevertheless be explored in the context of monothematic delusions (Coltheart 2011: 284).

Maher draws an analogy with science, suggesting that delusional hypotheses are best thought of as like scientific theories – both 'serve the purpose of providing order and meaning for empirical data obtained by observation' (Maher 1988: 20). The individual with a delusion is presented with a puzzle in their anomalous experience, which they come up with a theory to explain. Delusional hypotheses can become tenacious, and seemingly insensitive to counter-evidence (for more on delusion and evidence see Flores, Ch. 12).[5] Maher claims that this is analogous to scientific theory change:

> As in science, a coherent theory is only overthrown by a better theory and the chances that this can be done successfully by a clinician are reduced when the patient has found a generally satisfactory theory of his own. (Maher 1974: 107)

Of course, better theories are sometimes resisted because they conflict with a scientist's commitment to her own theory.

Let us see whether Maher's account can answer the two questions which structure the opposing account. The two-factor theory seeks to answer two questions regarding the genesis and maintenance of delusional belief:

> The first question is, what brought the delusional idea to mind in the first place? The second question is, why is this idea accepted as true and adopted as a belief when the belief is typically bizarre and when so much evidence against its truth is available to the patient?
>
> (Coltheart et al. 2011: 271)

One- and two-factor theorists alike have a fairly straightforward answer to the first question – they can appeal to the anomalous experience that the delusion is taken to explain. We have seen above what this looks like for Maher – the delusional hypothesis is prompted by the anomalous experience, and, via abductive inference, is taken to explain that experience. The second question is taken by two-factor theorists to be unanswerable without appeal to a second factor (Davies 2009: 72). Can we explain why a bizarre belief is adopted in the face of evidence against it which is available to the subject? This is where a bit of pressure is often thought to apply to the one-factor theory: why does the subject opt for such a poor explanation of the data generated by the anomalous experience? How can the one-factor theory explain why the explanation opted for is so flawed?

Some theorists have gone further, suggesting that it is not that delusional explanations are merely *poor*, they are 'unintelligible' (Nie 2023, cf. Sullivan-Bissett & Noordhof 2024: 3–5), or 'nonstarters', and 'the explanations of the delusional patients are nothing like explanations as we understand them' (Fine et al. 2005b: 160). Matthew Parrott, in the context of assessing the predictive processing account of delusion (see Corlett, Ch. 30) argues that the implausibility of delusional explanations is so extreme that we should posit impaired or disrupted mechanisms of hypothesis generation (Parrott 2021: 342). This kind of concern can of course be levelled against the one-factor approach which understands delusions as normal explanations of experience.

However, if, as Parrott puts it, 'simply *considering* an implausible delusional hypothesis as a candidate explanation manifests a clear departure from ordinary cognition' (Parrott 2021: 342, my emphasis), we might well be in the realms of a more general objection to the empiricist research programme, rather than something which could help us adjudicate between the one- and two-factor versions thereof. After all, some versions of the two-factor theory relate the second factor to belief *evaluation* rather than formation, and so if there is a problem concerning hypothesis generation, it is one which is equally pressing for two-factor theorists. Parrott speculates that some proposed second factors (cognitive biases) might influence hypothesis generation in such a way that a two-factor account could explain why nonstarter hypotheses are even entertained by subjects. But he notes that without a more developed model of hypothesis generation, we're not yet in a position to determine the prospects for such an approach (Parrott 2021: 344, fn. 24).

Let us move from the non-starter problem to the less extreme nearby problem: delusional explanations are *really poor* explanations. This problem has been taken to be applicable to the one-factor approach in particular. Indeed, Davies and colleagues have it that the idea that delusions arise from the subject's 'normal construction and adoption of an explanation' for an anomalous experience is problematic because 'delusional patients construct explanations that are not plausible and adopt them even when better explanations are available' (Davies et al. 2001: 147). More recently, Coltheart and Davies have argued that given that delusions are 'often bizarre or outlandish', the subject's conflicting knowledge or other beliefs ought to function as disconfirmatory evidence leading to the rejection of the delusional hypothesis (Coltheart & Davies 2021: 222).

I make two points in reply. First, constructing implausible explanations instead of better explanations is hardly unique to delusions. Beliefs in conspiracy theories and the paranormal will often share these features, and yet it is rare to find the suggestion that we have clinically abnormal belief formation or evaluation in these cases. Everyday irrationalities are seen as perfectly well-suited to carry the explanatory burden (Noordhof & Sullivan-Bissett 2021: 10302–3, 2023: 92–4). This needn't be to deny that there are important differences between delusional beliefs and beliefs of these other kinds (as Chenwie Nie interprets us (2024: 16, see Sullivan-Bissett & Noordhof 2024: 7 in reply)), but the latter do demonstrate that the charge of poor hypothesis selection is very far from a charge uniquely applicable to delusional explanations. Far more needs to be said before that feature of delusion gives us grounds for a second factor. Furthermore, in these other cases of poor hypothesis selection, it can be more difficult to make excuses on behalf of the subject's seemingly poor judgement. Although (some) conspiracy beliefs or paranormal beliefs may enjoy limited social support, they are not responses to highly anomalous and repeated experiences which are often the basis of delusions.[6] As I have noted elsewhere in a discussion of paranormal beliefs:

> [W]hen psychologists studying subjects with delusions become convinced that there needs to be a second factor involving clinical irrationality, they don't keep in firm view just how bizarre the beliefs of subjects in the normal range are, which are formed on the basis of less profound anomalous experiences with socially supported paranormal interpretation. If clinical irrationality is not required here, then it is not required for delusional beliefs. (Noordhof and Sullivan-Bissett 2023: 96)

Second, we can also question the implicit assumption that alternative explanations are *available* in the relevant respect. Let us distinguish three kinds of unavailability of alternatives: *strict* (where the alternative is inaccessible to the subject, perhaps because it is based on information opaque to introspection or otherwise irretrievable); *motivational* (where the alternative is inhibited or inaccessible due to motivational factors); and *explanatory* (where the alternative strikes the subject as implausible enough to not be regarded as a genuine contender) (for more on these notions of unavailability see Sullivan-Bissett 2018: 925–6). Speculatively, strict unavailability of alternatives might be bound up with anomalous experience, although this would need to be reconciled with case reports of people with delusions recognising the bizarre nature of their belief (see e.g. Alexander et al. 1979: 335).[7]

With respect to motivational unavailability, it is commonplace to recognise that beliefs can be formed with some assistance from motivational influences, and indeed, that alternative beliefs can be kept at bay by influences of this kind.[8] Delusions too. Most obviously perhaps in the case of motivational delusions, where the content believed is also the content desired (for example, erotomania or Reverse Othello delusion). But even in delusions which have unwelcome contents, significant relief from the distress caused by anomalous experience may be had for a subject upon forming the delusional beliefs and *figuring things out*. In addition, the alternative hypothesis that one's experience arises from a problem with oneself is hardly motivationally neutral – in the case of Capgras, accepting that one's imposter experience is neurobiological is 'not a particularly uplifting prospect' (Bortolotti 2023: 59, see also 107). Furthermore, as Maher puts it, 'the social costs and consequences of major decisions made under the influence of the delusion may create a situation in which it is very difficult for the patient to re-examine the belief and publicly reject it' (Maher 2006: 182). (See also Lisa Bortolotti's discussion of delusional persistence and identity, 2023: 83).

Explanatory unavailability may also have something to offer. Maher takes the psychological and epistemic weight of anomalous experience very seriously, describing delusions as developed 'via evidence powerful enough to support [them]' and anomalous experiences as ones which cannot be 'reasoned away' (Maher 1974: 99). Maher's point here is not the obvious one that argument cannot make experience cease, but rather that the experience has epistemic import not easily undercut by claims about its veridicality. Such epistemic import may go some way towards explaining why delusional hypotheses are preferred to non-delusional ones: the experiences may strike one as better explained by the former. As Maher puts it, 'asking patients to prefer a naturalistic theory to their own' would be 'tantamount to asking them to trust the evidence of other people's senses in preference to their own', which, although 'not impossible', is also 'not readily done by most people' (Maher 1988: 25).

In addition, alternative explanations might simply strike the subject as less good than the delusional explanation, insofar as they are being offered by those in poorer epistemic positions:

> Surely, we know our mother or spouse better than anybody else and can tell the subtle differences between the original and the imposter while the people around us are more likely to be fooled by substitution. (Bortolotti 2023: 42, see also Reimer 2009: 679–80)

In discussing the nature of delusional explanations, we have seen the range of resources available to the one-factor theorist. I have been brief but the take-home point is that we can approach poor hypothesis selection in much the same way as we do elsewhere. That is not to deny that it is perfectly interesting to investigate the range of cognitive influences which might contribute to the selection of the less good explanation, indeed, huge swathes of research in psychology and cognitive science seek to do this for other kinds of belief (religious, conspiratorial, paranormal). But since these influences are decidedly not *factors*, that project ought to be kept separate from the one engaged in by one- and two-factor theorists. At this point, we have been given no reason to suppose that the explanatory toolbox usually employed in explanations of other strange beliefs would be inadequate when turning to delusion.[9]

I turn now to discuss an extremely common objection to the one-factor approach, which will reveal the range of explanatory resources available to the one-factor theory.

5. The objection from dissociation

The objection from dissociation has been repeatedly levelled against Maher's one-factor approach. A very clear statement of it is found in Davies and colleagues (2001). In reflecting on experiences that could lead to eight types of delusion they say:

> On Maher's view, simply suffering from any one of these experiences would be sufficient to produce a delusion, because a delusion is the normal response to such unusual experiences. It follows that anyone who has suffered neuropsychological damage that reduces the affective response to faces should exhibit the Capgras delusion; anyone with a right hemisphere lesion that paralyzes the left limbs and leaves the subject with a sense that the limbs are alien should deny ownership of the limbs; anyone with a loss of the ability to interact fluently with mirrors should exhibit mirrored-self misidentification, and so on. However, these predictions from Maher's theory are clearly falsified by examples from the neuropsychological literature. (2001: 144)

Davies and colleagues proceed to survey several cases of dissociation. This objection has been raised again and again. Indeed, to my knowledge, all two-factor theorists argue that if a one-factor theory were true, then every subject who had the relevant anomalous experience would have the delusional belief. But, since this is not the case, there must be a second factor (see e.g. Chapman and Chapman 1988: 174; Garety 1991: 15; Garety et al. 1991: 194–5; Davies and Coltheart 2000: 11–12; Young and De Pauw 2002: 56; Davies et al. 2005: 224–5; Fine et al. 2005a: 145; Coltheart et al. 2011: 284–5; Coltheart 2015: 23; Miyazono 2018: 39; Coltheart and Davies 2021: 213ff; Nie 2023: 9–10).

There are broadly two ways to respond to the objection. The first is to deny that there are any such cases, and the second is to accept that there are, but show that this is consistent with the one-factor approach. I consider these in turn.

5.1 Denying dissociation

There are two ways of denying dissociation. The first is to contest the validity of the supposed empirical observation (Davies and colleagues themselves note this with respect to some of their cases (2001: 145)). Consider the case of Capgras delusion, its associated anomalous experience, and purported cases of dissociation. Coltheart has argued that the absence of autonomic response taken to be indicative of anomalous experiences in Capgras subjects is also present in subjects with ventromedial lesions, but the latter subjects do not have the Capgras delusion (Tranel et al. 1995, Coltheart 2007: 1048–9). However, Sam Wilkinson points out that the lesions are in different areas. Whilst Capgras subjects tend to have right lateral temporal lesions and dorsolateral prefrontal damage, those subjects taken to constitute examples of dissociation have ventromedial prefrontal damage (Wilkinson 2015: 18, see also Corlett 2019 for discussion of the Tranel and colleagues study and its implications for two-factor theories).

The second way to deny dissociation is to distinguish between experiences leading to delusions and those, apparently identical, experiences, which do not. In considering the potential problem of dissociation, Maher suggests that, compared to experience in the healthy population, 'the kinds of anomalous experience that deluded patients have appeared to be much more intense and prolonged' (Maher 1999: 566), and are 'repeated or continue over an extended period' (Maher 2006: 182). If that were right, then anomalous experience (at the requisite intensity and length) would be sufficient for delusion – apparent cases of the same experience would in fact be cases of experience of a more modest nature. To support this idea, Maher draws on Torsten Ingemann Nielsen's (1963) study in which subjects displayed signs of delusion-like thinking having undergone artificially induced anomalous experiences in a laboratory setting (for discussion see Maher 2006: 182, Noordhof and Sullivan-Bissett 2021: 10281–2). The lesson drawn is that if delusion-like explanations can be prompted by even brief and unrepeated anomalous experiences, then when such experiences are 'more intense and prolonged', they are sufficient for delusion formation. Dissociation is not explained by appeal to a second factor, but rather debunked by appeal to differences in experience.[10]

Davies and colleagues reply to both the claims of duration and intensity. On *duration*, they note that in general, delusions do not arise only after a prolonged period of the subject labouring under an anomalous experience. On *intensity* they say that it is unclear how it is that we could quantify the intensity of an experience (2001: 146). However, they draw on experimental data from Connie Cahill and colleagues (1996) showing that normal subjects and subjects with schizophrenia responded differently to an anomalous experience of hearing their own voice pitch-distorted. Normal subjects were able to identify the voice as their own, despite the distortion, whilst those subjects with schizophrenia often identified the voice as coming from another agent. The frequency of attributing a voice to another agent was correlated both with severity of delusion and the degree of pitch distortion (Cahill et al. 1996: 207). Cahill and colleagues take their findings to suggest that

> the "hallucination-like" reports elicited by our paradigm resulted from an interaction between an unusual perceptual experience (distorted auditory feedback) and an abnormal mechanism for belief formation present in deluded patients. (Cahill et al. 1996: 201)

I make two points here. First, the study participants had schizophrenia. Davies and colleagues open their paper by contrasting monothematic delusions with 'the

polythematic and elaborated delusions or delusional systems that are characteristic of some schizophrenia patients' (Davies et al. 2001: 135). Their two-factor theory is tagged explicitly to monothematic delusions arising from brain injury in particular (although they note an ambition for it to extend to monothematic delusions in psychiatric patients (Davies et al. 2001: 137)). Given this, we must be wary of appealing to experimental data on subjects with delusions in the context of schizophrenia to inform our account of monothematic delusion.[11]

Second, the experimental data are consistent with Maher's claim that cases of dissociation are cases of different experience with respect to duration and/or intensity. Even granting that Cahill and colleagues achieved sameness of experience with respect to intensity across their participants, dissociation of anomalous experience in the laboratory need not suggest the possibility of dissociation of *delusional* experience outside of it. It is consistent with the claim that a particular kind of experience (meeting the relevant thresholds of length and intensity) is sufficient for delusion formation, that we find dissociation in interpretation of experiences which do not have these features to the relevant threshold. The participants underwent thirteen trials (each with difference pitch distortion) which were presented in a random order. The experience then, understood as *hearing voice x at pitch y* was not repeated (Cahill et al. 1996: 206). Were the experiences as *intense* as those associated with delusions? It is hard to deny that the intensity of an experience could be affected by the broader context (taking place in a laboratory, knowing the genesis even if not the source), and the possibility of cognitive penetration resulting from this background knowledge could well make one's experience less intense than the experiences associated with delusion. Cahill and colleagues may have demonstrated a difference in response to experience without a difference in intensity, but there are reasons to doubt that such experience is a good model for the anomalous experience to which deluded subjects are responding outside of the laboratory. (For further critique of the relevance of Cahill and colleagues' study for understanding the nature of anomalous experience, see Reimer 2009: 679.)

5.2 Embrace dissociation

The second response to the objection from dissociation is to show that the observation of dissociation is consistent with the one-factor approach. That is, let us proceed without questioning any further the claim that there are cases of *same experience,* but only some of those experiences lead to delusion. Let us instead turn to the *sufficiency claim,* that is, the claim that anomalous experience is *sufficient* for delusion formation. The force of the objection from dissociation comes from taking Maher to endorse the sufficiency claim, without which, dissociation would be unproblematic. I suggest that (1) it is *probably* a mistake to understand Maher as a proponent of the sufficiency claim, but in any case (2) it is *definitely* a mistake to understand the one-factor approach as requiring it.

Davies and colleagues characterise Maher's view as one which holds that 'delusions are false beliefs that arise as normal responses to experiences' (Davies et al. 2001: 133), and in outlining Maher's account, they quote the four hypotheses constituting his model:

- Delusional beliefs, like normal beliefs, arise from an attempt to explain experience.
- The processes by which deluded persons reason from experience to belief are not significantly different from the processes by which non-deluded persons do.
- Defective reasoning about actual personal normal experience is not the primary contributor to the formation of delusional beliefs.

- The origins of anomalous experience may lie in a broad band of neuropsychological anomalies.

(Maher 1999: 550–1, cited in Davies et al. 2001: 138)

Nothing in the characterization of Maher's position here suggests an endorsement of the sufficiency claim. Maher is rather interested in *normality*. In earlier work he had it that the cognitive activity of people with delusions is 'essentially indistinguishable' from that employed by non-delusional people, and talks of delusions being developed 'through the operation of *normal cognitive processes*' (Maher 1974: 103, my emphasis). Later he argued that '[t]he cognitive processes by which delusions are formed are in no important respect different from those by which normal beliefs are formed' (Maher 1992: 262). And in the above outline of his view, the first two points concern normal processes of belief formation directed at strange experience. Not only does nothing in these claims suggest that anomalous experience is sufficient for delusion formation, they suggest quite the opposite! Normal cognitive processes, in addition to anomalous experiences, are also clearly implicated in a delusion's genesis. *Normal* cognition will tolerate a range of responses to particular experiences. It could thus even be a prediction of the one-factor approach, and indeed utterly unremarkable, that some people will have a given anomalous experience but not go on to develop a delusion.

Davies and colleagues move from something's being *normal,* to something's being *sufficient*. This move is evident when they say '[o]n Maher's view, simply suffering from any one of these experiences would be sufficient to produce a delusion, *because* a delusion is the normal response to such unusual experiences' (Davies et al. 2001: 145, my emphasis). But the key point for Maher was that whatever cognitive quirks or intellectual styles we find to be involved in the move from experience to belief formation and maintenance, they do not constitute clinical abnormalities.

All of that said, though, there are two places that Maher hints at the sufficiency claim, which is why I say that he was only *probably* not committed to it. The first is when he cites Graham Reed:

> Given the necessary information, the observer can empathise with the subject; If he himself were to have such an unusual experience he would express beliefs about it which would be just as unusual as those of the subject. (Reed 1974: 154, cited in Maher 1999: 551)

The second hint at the sufficiency claim comes later in the same paper where Maher turns to pre-empt the objection from dissociation (Maher 1999: 566). He responds by denying dissociation, and appealing to duration and intensity as the relevant experiential difference makers (as discussed in the previous sub-section).

So did Maher endorse the sufficiency claim? If he did, it was certainly not central to his approach. When we look at the various outlines of his model, the sufficiency claim or anything equivalent is missing (see for example the commitments of the model given in 1992: 262–4; 1999: 550–1; 2006: 181–2). Where there is some evidence that he endorsed sufficiency (in quoting Reed and responding to the problem of dissociation, 1999), he concludes that same paper in a way unfriendly to the sufficiency claim:

> It is entirely possible that delusions, like normal beliefs, arise from heterogenous sources. […] the study of delusions […] highlights the cognitive processes that typically emerge in the attempt to find meaning in the presence of uncertainty. (Maher 1999: 567)

It is hard to reconcile the idea that delusions arise from heterogenous sources and a range of cognitive processes with the idea that anomalous experiences are *sufficient* for delusion formation. Maher's quoted conclusion here does not suggest that it is his view that an experiential anomaly would – whatever else might be going on with the subject cognitively – produce or sustain a delusion. There is, then, limited evidence that Maher endorsed the sufficiency claim. And yet, such a perceived endorsement has been the main grounds on which his view has been rejected.

However, even if Maher were a one-factor theorist of the sufficiency kind, this is not the only way of being a one-factor theorist, and the overall prospects of the approach have been vastly underestimated when this is not recognized. The key point of the one-factor approach is that there is one clinical abnormality involved in the genesis and maintenance of a delusional belief. As Gerrans put it, for the one-factor theorist delusions are 'rationalizations of anomalous experiences via reasoning strategies that are not, in themselves abnormal' (Gerrans 2002: 47). As I note elsewhere, '[n]othing in the statement of this approach suggests that anybody who has the definitive anomalous experience must have the delusional belief as well' (Noordhof and Sullivan-Bissett 2021: 10297). The two-factor theorist is mistaken in expecting the one-factor theorist to identify an anomaly which would, whatever the psychology, give rise to delusion. As Maher puts it:

> Normal beliefs appear to be acquired in many different ways. […] We do not seek to find a single cause of normal beliefs. Nor should we assume that manifestly similar clinical phenomena necessarily arise from a pathway that began with a single specific pathology. (Maher 1992: 267)

The empirical observation of different beliefs arising from the same experience is simply not relevant in assessing the merits of the one-factor view, since the key point is not that delusional beliefs have a single cause, only that of the range of causes of delusional beliefs, only one of them has the feature of being abnormal.

Now, it might be accepted that, strictly speaking, dissociation is not inconsistent with the one-factor theory – the observation that some non-delusional people have the experience implicated in delusion is, technically, no mark against the account. However, it might nevertheless be thought that cases of dissociation lend support to two-factor theories, who take such cases as their starting point for identifying a second factor. A natural question to ask in the face of dissociation cases is *what explains the difference* between those who have the experience and become delusional, and those who have the experience and do not? Two-factor theorists have a ready-made answer in their pockets: what explains the difference is the second factor, present in the person who becomes delusional, and absent in the person who does not. Does the one-factor account have anything to offer here?

The one-factor theorist can help herself to the many resources of cognitive science, social psychology, epistemology, philosophy of mind, and so on in explaining cases of dissociation. Everyday irrationalities, or idiosyncrasies, can bear the weight of the explanatory burden. This kind of project, though, need not be taken to be within the remit of a one-factor theorist's task. Her task is to identify the number of factors needed to explain delusional belief, *not* to explain the various other causal contributions in this context.

6. Concluding remarks

In this chapter I have overviewed Maher's one factor approach. I explicated its virtues and explanatory resources by considering hypothesis selection, and a key objection from dissociation. We have seen that the one-factor account is not put under pressure by the idea that delusions are poor explanations, or the (apparent) observation of cases of dissociation.

The two-factor theory has been advertised as able to fill the explanatory gaps charged to be left by the one-factor theory. This is based on an underestimation of the resources available to the one-factor theory. I haven't spoken to the positive case for the two-factor theory (although I have done some of this work elsewhere, see Noordhof and Sullivan-Bissett 2021). I have not, then, shown that there is no case to be made for a two-factor theory, only that it ought not be motivated by the misperceived inadequacies of a one-factor approach. For my money, the one-factor approach should be the default hypothesis.

I finish with a couple of methodological remarks. I have argued elsewhere that the research trajectory of researchers seeking a second factor is different from that of researchers investigating alien abduction beliefs (Sullivan-Bissett 2020), paranormal beliefs (Noordhof and Sullivan-Bissett 2023), and conspiracy beliefs (Ichino and Sullivan-Bissett *under review*). Now that we can see the shape of the one-factor theory, it is my view that these methodological differences in our approaches to understanding delusions as compared to other bizarre, evidence-resistant beliefs, is not justified.

Finally, a temptation is to identify some cases where it does look proper to appeal to two factors. Particular cases may well involve abnormalities of the kind two-factor theorists appeal to. But that does not justify generalizing from particular cases to the nature of delusions simpliciter. And the claim of the two-factor theorist is strong, as Coltheart and Davies have recently put it: 'a delusion will *only* result when a second factor is also present' (2021: 215, my emphasis), and in several places Coltheart, Davies, and their collaborators have suggested that their approach is intended to apply to *all* monothematic delusions (see e.g. Coltheart et al. 2011: 285). But it is a mistake to generalise from particular cases to a claim about the nature of monothematic delusion simpliciter. If we're in the market for a theory of monothematic delusions as a kind, the one-factor account strikes me as a good place to start.

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[1] Paul Franceschi (2008) has argued for a one-factor approach to polythematic delusions, understanding them as rising from *apophenia,* helped along by common errors of reasoning.

[2] Sometimes this characterisation of a factor is not kept firmly in view. For example, Max Coltheart and Davies have recently defended a two-factor theory of the Koro delusion (the belief that *one's penis is shrinking into one's abdomen in a way that might be fatal*) (2024)). The 'factors' they appeal to are, by their own lights, perfectly ordinary experiences, together with culturally normal background beliefs, limited formal education, and/or sociocultural factors. However, the explanatory power of factor-talk is lost when we divorce it from picking out abnormalities, since the explanatory role of e.g. limited formal education in an explanation of a belief is not a role relevant to an explanation of *delusional* belief in particular. Theories identifying the various normal range contributions to delusions are of course interesting and important, but talk of factors in this context obscures the special explanatory role played by particular kinds of contribution (those which are abnormal).

[3] Although factors are often associated with neuropsychological damage, two-factor theorists have been clear that they need not be (see e.g. Coltheart et al. 2011: 291). Functional abnormalities need not always be realised by neuropsychological damage, but where functional abnormality remains hard to make out, theorists could fall back on statistical abnormality to capture the relevant anomalies. Since this is a taxonomical difficulty for all involved, I put this complication aside.

[4] I say *can* because I do not want to rule out delusions arising as explanations of experiences which need not be abnormal (erotomania might be such a case, see also discussion in Bell et al. 2008). Understanding the one-factor approach as an *at most* claim may well be idiosyncratic, but the key point is that we certainly do not need a second factor. This flexibility may allow us to tell a story about alien abduction beliefs which could be properly characterized as delusions, even though the experience which prompts them may be shared among healthy subjects and so is not a statistical abnormality (Sullivan-Bissett 2020). We might also characterise the Koro belief as a delusion, even though the experiences which might prompt the hypothesis

are everyday (illness, urination, ejaculation) and so do not constitute a functional abnormality (see Coltheart and Davies 2024 for discussion).

[5] For reasons of space, I do not consider the relationship between delusion and evidence, and how that bears on the plausibility of the one-factor account. I have done so elsewhere (see Noordhof and Sullivan-Bissett 2021, 2023, Sullivan-Bissett *forthcoming*). For arguments that delusions are evidence-responsive see Flores (2021).

[6] Garry Young has argued that, at least in the case of Capgras delusion, in light of the anomalous experience, 'the subject feels justified in broadening the scope of what he feels is epistemically possible, as he looks to explain what is happening' (Young 2023: 161).

[7] In their discussion of Parrott's objection that predictive processing theories cannot explain non-starter hypothesis generation, Federico Bongiorno and Philip Corlett draw on Jakob Hohwy who has it that 'those more probable alternatives are not selected because they are unable to explain away aberrant prediction errors at the right spatiotemporal fineness of grain' (2013: 161, cited in Bongiorno and Corlett *forthcoming*).

[8] One way of motivational influences having this effect is through evidence avoidance, something taken to be key to the development and maintenance of self-deceptive beliefs, and even more everyday cognitive failings like the application of confirmation bias (Flores 2021: 6309).

[9] So often the one-factor theory earns itself a bad reputation because it is mistaken to be overly generous. Indeed, some folk have even understood Maher to claim that forming a delusional belief on the basis of an anomalous experience is *rational response* to that experience (Davies and Coltheart 2000: 8, Bentall et al. 2001: 1149, Bortolotti 2009: 57). It has long been recognised that this is not the claim of the one-factor approach (Gerrans 2002: 48).

[10] Marga Reimer takes forward this idea suggesting that, in the case of Capgras at least, the neurological damage 'causes *both* an affective deficit in face processing *and* some other experiential abnormality', and suggests that the resulting experience could be sufficient to generate Capgras (Reimer 2009: 678). The second experiential abnormality would explain the special intensity or vividness of the anomalous experience. B. S. Lana Frankle has recently made a similar argument. She notes that two subjects (one delusional, one not) can have perfect overlap in the information they share, by which she means that someone with delusional experience could, in theory, articulate that experience in a way that it is fully captured and understood by a non-delusional subject. However, to explain dissociation she speculates that we can posit a 'fundamental qualia about certain altered perceptual experiences that is beyond the realm of explicit knowledge' (Frankle 2021: 7). And so, even though experiences that prompt delusions may resemble experiences which do not prompt delusions the addition of a particular kind of qualia in the case of the former can explain why delusions are prompted. This hypothesis is destined to remain in the realms of speculation; Frankle gives no theoretical or empirical justification for it, and although it is friendly to the one-factor account, no one not already committed to such an approach would have any new grounds to accept it.

[11] Although Davies and colleagues note that the relationships between monothematicity and circumscription on the one hand, and polythematicity and elaboration on the other are not exceptionless (Davies et al. 2001: 135), their focus is nevertheless on monothematic delusions, and *not* delusions as they occur in the broader context of schizophrenia.