



Failing to self-ascribe thought and motion: Towards a three-factor account of passivity symptoms in schizophrenia

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ABSTRACT

There has recently been emphasis put on providing two-factor accounts of monothematic delusions. Such accounts would explain (1) whether a delusional hypothesis (e.g. someone else is inserting thoughts into my mind) can be understood as a *prima facie* reasonable response to an experience and (2) why such a delusional hypothesis is believed and maintained given its implausibility and evidence against it. I argue that if we are to avoid obfuscating the cognitive mechanisms involved in monothematic delusion formation we should split the first factor (1 above) into two factors: how abnormal experience can give rise to a delusional 'proto-hypothesis' and how a 'proto-hypothesis' in consort with normal experiences and background information, can be developed into a delusional hypothesis. In particular I will argue that a schizophrenic is faced with the unusual requirement of having to identify an introspectively accessible thought as one's own, and that this requirement of identification is the central experiential abnormality of thought insertion, auditory verbal hallucination, and alien control (i.e. passivity symptoms). Additionally, I will consider non-experiential factors which are required for the formation of a delusional hypothesis.

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1. Background/introduction

Schizophrenia has attracted attention from philosophers of mind and philosophers of cognitive psychology largely because the passivity symptoms of thought insertion and alien control challenge some of the most intuitive beliefs we have about what is an adequate source of justification for our self-knowledge. (I will take 'self-knowledge' to mean introspection-based knowledge of one's thoughts and actions.) The intuitive view is that if I receive information about my own thoughts and movements on the basis of introspective information, this is sufficient to know (1) what sort of thinking or action I am engaged in and (2) that it is *me* who is doing the thinking and acting. However, the schizophrenic symptoms of thought insertion and alien control appear to bring the second claim into question: that introspective information is sufficient for knowing it is *me* who is doing the thinking and acting, and not someone else (e.g. see Campbell, 1999).

My aim in this article is to provide an explanation of abnormal experience for the passivity symptoms of schizophrenia. I will argue that what is essential to understanding these symptoms is that they result from an unusual requirement placed on the schizophrenic: a need to identify who is the agent of one's own thoughts and actions. I will

argue that this requirement of identification is the central feature of the abnormal experience associated with passivity symptoms. As I take the fundamental aspects of the abnormal experience underlying passivity symptoms to be the same, I will focus my attention on thought insertion—mentioning alien control and auditory verbal hallucination when discussion of such cases is helpful. I will offer this explanation by expanding on a 'two-factor' account used to explain delusions by Davies and Coltheart (2000).

2. Monothematic delusions, two-, and three-factor accounts

As noted by Davies and Coltheart (2000) several delusions can be categorized as monothematic in that they focus on a central theme (Davies et al., 2001). Examples of monothematic delusions include Capgras Delusion (the belief that individuals close to the person have been replaced by imposters) reduplicative paramnesia (an arm [attached to me] is someone else's and that person has three arms), the Cotard Delusion (the belief that I am dead), thought insertion, and alien control. Monothematic delusions are contrasted with polythematic delusions which focus on several themes and interact significantly with one's other beliefs. Schizophrenia often involves several kinds of delusions, including polythematic paranoid delusions. However, Davies and Coltheart are not attempting to characterize mental disorders such as schizophrenia, but rather the feasibility

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of a general framework for explaining delusions which focus on a central theme.

Davies' and Coltheart's project is thus significantly different than projects concerning etiological explanations of particular syndromes. For instance, Capgras Delusion has been witnessed in subjects with several disorders, including AIDS, Alzheimer's disease, epilepsy, Lewy-body dementia, multiple sclerosis, pituitary tumor, schizophrenia, and viral encephalitis (Coltheart et al., 2007). Regardless of the different neuropathologies that give rise to similar delusional claims, we can still provide a uniform *cognitive* explanation as to why delusions focusing on a central theme are made.

The two-factor account of monothematic delusions requires of *cognitive* accounts of monothematic delusions that they address the following two claims. In regards to any delusional hypothesis we must explain:

1. why a delusional hypothesis is a "prima facie reasonable response to the subject's experience", and
2. how one can adopt and maintain a delusional hypothesis given its "utter implausibility and the uniform skepticism with which other people greet it?" (Davies and Coltheart, 2000).

The two-factor account assumes that monothematic delusions have their origins in abnormal experience. Thus, there is the common presumption that monothematic delusions cannot result solely from abnormal reasoning. Among those who hold that monothematic delusions result from abnormal experience, researchers are divided into those that think delusional beliefs are rational responses to abnormal experience (e.g. Maher, 1974, 1988, 1992, 1999) and those who think that delusional beliefs are irrational responses to abnormal experience (e.g. Stone and Young, 1997; Davies and Coltheart, 2000; Coltheart et al., 2007; Coltheart, 2013). While both camps are capable of giving two-factor accounts, the first camp will explain the second factor in terms of normal reasoning and continuing abnormal experience¹ whereas the second camp will explain the second factor in terms of abnormal reasoning alone. (As my concern is just with abnormal experience and how it can lead to the formation of a delusional hypothesis I will elaborate primarily on the first factor.)

Granting this central role given to abnormal experience in accounting for delusion formation, we might ask how a delusional hypothesis can be a prima facie reasonable response to an abnormal experience. I take the demands on what makes a response to an abnormal experience prima facie reasonable to be quite minimal. For instance, having a hallucination of a miniature pink elephant provides one with prima facie reason for believing that there is a pink elephant. Of course, this belief is easily defeated by other considerations – that's why it is just a prima facie reasonable response. However, having a hallucination of a pink elephant does not provide me with prima facie reason for believing that good golfers make horrible mathematicians. We might say that in order for a response to an experience to be prima facie reasonable, the response (e.g. a belief or hypothesis), has to adequately represent the experience the individual is presented with. This account of prima facie reasonableness places the following explanatory burden on us: whatever account of abnormal experience is given, we must be able to give an account of it such that some delusional hypotheses can be

taken to adequately represent the experience whereas other hypotheses cannot.

A problem with the first factor of the two-factor account is that it has an ambiguity that can result in an explanatory inadequacy: the first factor does not disambiguate whether a proper explanation should be one which explains how the delusional hypothesis is a *prima facie* reasonable response to either (1) *just* an abnormal experience or (2) an abnormal experience in addition to normal experiences and background information. Problems arise if we adopt the first approach as it will demand a highly intricate abnormal experience to bring about the delusional hypothesis. We can see this demand in the following characterization of the abnormal experience involved in thought insertion:

One central phenomenon of the "psychosis of thinking"...is thought insertion ... in which the patient feels that alien thoughts are being inserted into his mind

[Campbell, 1999, 615.]

In this account, the abnormal experience is supposed to be captured by the description of the feeling 'that alien thoughts are being inserted into his mind'. If one could have a feeling which is properly described as 'alien thoughts are being inserted into my mind', then the delusional hypothesis 'alien thoughts are being inserted into my mind' would adequately represent that experience and thus be a *prima facie* reasonable response to this abnormal experience. But without a previous experience of (real) alien thoughts and (real) thought insertion, how would we be able to describe such a feeling?²

To reduce the demand for a highly intricate abnormal experience, as required by the former interpretation mentioned above, we can disambiguate the first factor in the following way:

0. What delusional proto-hypothesis can be understood as a *prima facie* reasonable response to the subject's abnormal experience?
- 1'. How can we explain the development of a delusional hypothesis in light of the subject's delusional proto-hypothesis, inferences, normal experiences, and background information?

By 'proto-hypothesis' I mean a hypothesis which is a *prima facie* reasonable response to *just* the subject's abnormal experience. As such, a delusional proto-hypothesis serves as an explanatory intermediary between abnormal experience and the adopted delusional hypothesis. As such, there is no expectation that the delusional proto-hypothesis be explicitly entertained by the subject in question. The proto-hypothesis will aid in an explanation of what can be a reasonable response to an abnormal experience (captured by the 0 factor) and what inferences, normal experiences, and background assumptions can lead to the development of a delusional hypothesis (captured by the 1' factor).^{3,4} Whereas this account allows us to posit a simpler (i.e. more plausible) abnormal experience than Campbell's account above, we must then do extra work to explain how a delusional hypothesis is formed.

3. Passivity symptoms, explanatory challenges, and corollary discharge

As I have now characterized the framework within which I will discuss monothematic delusions in schizophrenia, I will now address

¹ Maher has theorized that it is repeated abnormal experiences that result in the formation and maintenance of delusional beliefs. As such, he accounts for the first and second factor of monothematic delusions by appealing to abnormal experience (Maher, 1999, 566). In what follows, I agree with Maher in terms of accounting for the first factor: schizophrenics exhibit sufficient rationality, that the simplest explanation for the formulation of a delusional hypothesis need not invoke failures of rationality in addition to abnormal experience. For reasons beyond the scope of this paper, I do not think that the maintenance of delusional hypothesis can be given a similar 'rational' account given the tendency of delusional schizophrenics to adopt beliefs on the basis of minimal positive evidence (see e.g. Langdon et al., 2010; Moritz and Woodward, 2005; Colbert and Peters, 2002; Young and Bentall, 1997; Garety et al., 1991; Huq et al., 1988).

² Contrast this with the task of describing an auditory verbal hallucination. While such hallucinatory experiences are often not experientially identical with hearing normal speech, such experiences can at least be approximately described by appeal to the ordinary experience of hearing speech.

³ If there are cases where a delusional hypothesis can be formed solely on the basis of abnormal experience, the proto-hypothesis will just be the delusional hypothesis.

⁴ The factors on the three-factor account are labeled '0', '1', and '2'. I start with '0' so that the factor labeled '2', which is the same on both accounts, need not be relabeled. The second factor on the three-factor account is labeled '1' so as not to be confused with the first factor of the two-factor account labeled '1'.

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