

## Psychosis, agnosia, and confabulation: an alternative two-factor account

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*Introduction.* Theories of delusions which rely on a combination of abnormal experience and defective belief evaluation and/ or cognitive bias are the subject of an emerging consensus. This paper challenges the validity of these theories and constructs a two factor alternative.

*Methods.* The paper starts by identifying the difficulty the current theories have explaining the complex delusions of schizophrenia and then, by considering, first, the aetiology of somatopsychotic symptoms, and second, the literature on the relationship between confabulation and allopsychotic symptoms, demonstrates that the natural solution is to retain the experiential factor whilst replacing the second factor with confabulation.

*Results.* The paper is then able to demonstrate that the resultant two-factor theory can clarify recent work on the aetiological role of auto-noetic agnosia and on the relationships between confabulation, delusion, and thought disorder.

*Conclusions.* The theory supersedes currently available theories in terms of its simplicity, fruitfulness, scope and conservatism and represents an advance in the search for unified theory of psychosis.

**Keywords:** delusion; confabulation; somatopsychosis; experience; auto-noetic; agnosia

### Introduction

Coltheart, Langdon, and McKay (2007) extended a two-factor explanation of Capgras' delusion to somatoparaphrenic delusions and from there to paranoid delusions in schizophrenia. The authors argued that abnormal experiences or "data" give rise to delusional hypotheses which are not rejected because of damage to a belief evaluation system located in the right frontal cortex. Coltheart et al.'s attempt to bring all delusions under a single theory represents an important advance on theories of psychosis that neglect somatoparaphrenic symptoms and/or focus on cognitive bias to underwrite the transition from abnormal experiences to delusions (see, for example, Broome et al., 2005).

Metcalf, Langdon, and Coltheart (2007) and Turner and Coltheart (2010) then applied the two-factor theory to inform the relationship between confabulation and delusion. Essentially, the authors argued, first, that mnemonic factors in confabulation and experiential factors in delusions operate at the same first-factor level, and second, that confabulations and delusions share common evaluative and monitoring second-factor processes. The validity of these claims rests on crucial assumptions

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about the first-factor aetiology of confabulation and the original justification for relying on second-factor defective belief evaluation in a theory of delusions.

The starting point for this paper is the observation that theories that rely on abnormal experience and defective belief evaluation and/or cognitive bias have difficulty explaining how complex delusional hypotheses are generated. It will be proposed that the solution can be found by reflecting on the aetiology of somatopsychotic symptoms and also on the work of Johnson (1988, 1991; Johnson, O'Connor, & Cantor, 1997; Johnson & Raye, 2000), and involves replacing the defective belief evaluation and/or cognitive bias factor with confabulation.

It will be argued that the resultant two-factor theory clarifies, first, the aetiological role of autozoetic agnosia (Frith & Done, 1989; Keefe, 1998), and, second, the relationship between confabulation and delusion. The latter will be achieved through a discussion of the aetiology of confabulation which allows defective "evaluation" to be reintroduced in its proper, more restricted context. It will then be suggested that the new theory provides a natural explanation of why individuals with schizophrenia confabulate, and can inform the understanding the relationship between confabulation and thought disorder (Lorente-Rovira, Pomerol-Clotet, McCarthy, Berrios, & McKenna, 2007; Nathaniel-James & Frith, 1996) in a way that gives an early indication of how the gap between research on delusions and research on language impairments in schizophrenia might be bridged.

### **Abnormal experience, cognitive bias, and belief evaluation**

The existence of pseudohallucinations or "subjective sensory experiences which are the consequence of functional psychiatric disorders and which are interpreted in a non-morbid way by the patient" (Hare, 1973, p. 474), coupled with the observation that delusions can arise from thematically related abnormal experiences, suggest that theories that explain delusional content primarily in terms of abnormal experience are likely to be correct that this should be the first factor in a two-factor explanation of psychotic symptoms. Strictly speaking, the nature of the first factor will need qualifying when somatopsychotic symptoms and autozoetic agnosia are discussed later in the paper.

The contentious part of "empiricist" theories, as Campbell (2001) refers to them, is the account they give of how abnormal experiences are converted into delusions. In an important paper, Coltheart et al. (2007) argue that there are two requirements on an adequate explanation of this transition: (1) to explain how (often complex) delusional content arises from a mere experience which has become known as the "abductive inference problem" (Bayne & Pacherie, 2004; Coltheart, Menzies, & Sutton, 2010); and (2) to explain why delusional content, once generated, is not simply rejected as false.

The most parsimonious approach is to address (1) and (2) simultaneously by suggesting that delusions are abnormal experiences that are somehow "endorsed" as accurate representations of reality (Bayne & Pacherie, 2004, p. 3). The difficulty with subsuming all delusions under this theory, however, is that it is *prima facie* unlikely that the content of complex delusions (even nonclassical Capgras' delusions such as Cutting's patient who thought her common-law husband was the son of Adam and Eve [1997, p. 141]) is exclusively experiential.

Most empiricist theories acknowledge this, and argue that delusional content arises, in part, out of an attempt to *explain* abnormal experiences. Cognitive bias has

previously been invoked in this context with Broome et al. (2005), for example, arguing that data-gathering and attribution biases result in the delusional interpretation of abnormal experiences. However, we will not discuss this proposal in detail since not only do not all psychotic individuals exhibit cognitive bias (Kemp, Chua, McKenna, & David, 1997), but delusions can be “‘immune’ to reasoning to some extent, even when reasoning abilities are intact” (Turner & Coltheart, 2010, p. 363). It is perhaps worth adding, that the analogous but more familiar role that cognitive bias plays in two-factor explanations of somatisation disorder (Turner, 2006, pp. 29–30) suggests that a two-factor theory which relies on it will in any case struggle to account for complex delusional abductive inferences.

Coltheart et al. (2007) share the view that delusional content results from an attempt to explain abnormal experiences, but the authors continue to rely on abnormal experience to do most of the content generating work. According to Coltheart et al. (2010), for example, “the delusional hypothesis provides a much more convincing explanation of the highly unusual data [i.e., abnormal experience] than the nondelusional hypothesis; and this fact swamps the general implausibility of the delusional belief . . .” (p. 278). If this approach is extended to complex delusions, it suggests that David’s (1990, p. 804) patient’s delusional hypothesis that he had an actual power station, complete with labourers, machinery, and cooling towers inside him is exhibiting a “perfectly rational response to very abnormal data” (Coltheart, Menzies, & Sutton, 2010, p. 281).

This places a considerable theoretical burden on experience. Furthermore, by not employing a second content generating mechanism, which could contribute to the explanation of why delusional hypotheses are not rejected, Coltheart et al. (2007) have no way of avoiding the introduction of a second aetiological factor the sole theoretical purpose of which is to fulfil this role. With this in mind, Coltheart et al. postulate that there is a region of the right frontal cortex which, when damaged, prevents the correct evaluation of delusional hypotheses.

Coltheart et al. (2010) articulate defective belief evaluation as “irrationally ignor[ing] or discount[ing] the evidence on the basis of its incompatibility with the hypothesis to which they have become committed; so the delusional belief persists” (p. 281). Now, we have already suggested that there are difficulties relying on cognitive bias to generate content, and the same considerations are likely to apply if the process is reintroduced to explain “defective belief evaluation”. On the other hand, if defective belief evaluation is understood as a neuropsychological deficit, as Coltheart et al. (2007) imply, then this, as we will see in the next section, is difficult to reconcile with a point made by the philosopher Gareth Evans that “when a subject wants to make absolutely sure that his judgment is correct, he gazes again *at the world* (thereby producing or reproducing, an informational state in himself)” (1982, p. 227, emphasis in original).

### **Somatopsychosis and confabulation**

We will return to defective belief evaluation when discussing the relationship between delusions and confabulations, but the natural solution to the problems caused by complex delusional hypotheses is to construct a theory that relies on a content generating mechanism which can both make a substantial contribution to content and simultaneously explain why delusional hypotheses are not rejected. This

mechanism will come into view if we examine Coltheart et al.'s (2007) extension of their theory of Capgras' Syndrome to somatoparaphrenic delusions.

Coltheart et al. (2007) consider the development of a delusion of nonownership of a paralysed limb in an individual with a left hemiplegia following a right-sided brain insult. The authors then ask, what could have happened to the hemiplegic individual for this delusion to arise? The answer, they suggest, is that the abnormal experience of paralysis requires an explanation and the delusion of nonownership of the limb provides this. We will refer to this as Assumption 1. Coltheart et al. then argue that because many hemiplegic individuals do not develop delusions, a second aetiological factor is required and, in view of the typically intact left hemisphere, this must have something to do with the right hemisphere. We will refer to this as Assumption 2.

In terms of Assumption 1, it is difficult to understand why not being able to move a paralysed limb would lead an individual to hypothesise that their limb belonged to someone else. Why, especially in the absence of significantly disturbed thinking, is the paralysed individual's first thought not "I am paralysed"? This concern is compounded by reports of cases that involve recognition of paralysis but not of ownership. Cutting (1997, p. 273), for example, mentions von Anygal and Frick's (1941) patient who claimed his paralysed side was his paralysed brother.

Coltheart et al. (2010, p. 283) later recognise that such cases represent an objection to their theory, but it is the reversal of the direction of causation between "limb ownership" and "paralysis" at the point of theory construction which gives rise to the difficulties. The most likely explanation for such cases is that the failure to recognise one's paralysis, i.e., anosognosia, is a manifestation of difficulties with bodily representation or "corporeal comprehension", as Devinsky and D'Esposito (2004, p. 77) refer to it, which in their most severe form amount to hemisomatagnosia, which involves a failure to recognise the limb(s) as one's own.

As in von Anygal and Frick's (1941) "paralysed brother" case, this does not preclude implicit awareness of the existence of a paralysis, and it is well recognised individuals use personification and other metaphors such as "a 'piece of rusty machinery' or 'dead wood' indicating knowledge that something is amiss" (Weinstein, 1991, p. 242). However, it is the underlying absence of experience, what Bisiach and Germiniani (1991) refer to as a "representational lacuna" (p. 31), and what we might refer to as the "experiential abnormality" rather than an "abnormal experience" of paralysis, that is the natural basis of delusions of nonownership.

Hemisomatagnosia interferes with the capacity to form a correct judgement and, in doing so, makes the introduction of a separate process of defective belief evaluation, at least in the sense intended in Coltheart et al. (2007), unnecessary. In other words, a delusion is not something that results from an experiential abnormality which is then incorrectly evaluated. It results (in part) from an experiential abnormality which itself involves an interference with the capacity to form correct judgements. This interference will also afflict the second order judgements involved in Coltheart et al.'s second-factor process of defective evaluation and this is where we see the importance of Evans's (1982) point. When a subject wants to make absolutely sure that his judgement is correct (i.e., evaluate it), he gazes not at his experiential abnormality, but rather through it, and this will only reproduce the original information state in himself.

The difficulty, however, is that we still require an explanation of how complex somatopsychotic delusions, such as those about “paralysed brothers”, result from hemisomatagnosia. In order to resolve this we need to postulate a further cognitive process which creates, as Weinstein puts it, a “fictional account . . . [that gives] order and predictability to a catastrophic event” (1991, p. 245). This process is recognised to be confabulation, and, unlike defective belief evaluation and cognitive bias, it is a familiar consequence of brain damage that has clear content-generating capabilities.

Furthermore, and turning to Coltheart et al.’s (2007) Assumption 2, although confabulation is likely to arise out of an attempt to make sense of the experiential abnormalities originating in the damaged right hemisphere, the evidence from “split brain” or corpus callosotomy patients supports the hypothesis that it requires left hemispheric “interpretative” activity in the presence of disrupted interhemispheric relations (Phelps & Gazzaniga, 1992). In other words, the generation of psychotic symptoms is likely to require the constructive activity of an intact left hemisphere as much as the lack of activity associated with a damaged right hemisphere.

By identifying the difficulties with Coltheart et al.’s (2007) account of somatopsychotic symptoms, we have arrived at a two-factor theory that relies on confabulation and a broader experiential factor, which includes agnosia, i.e., an “experiential abnormality” rather than an “abnormal experience”. The latter distinction may appear semantic, but empiricism’s overly narrow construal of the experiential factor at the theory construction stage obscured the theoretical availability of confabulation, notwithstanding that it is phenomenologically related to the experiential deficits that the theory maintains underlie delusions, and can perform the content generating work explaining the complex somatopsychotic delusional hypotheses in a way that obviates the need to rely on Coltheart et al.’s defective belief evaluation.

It is important to acknowledge that it is an advantage of Coltheart et al.’s (2007) proposal that it recognises the pivotal theoretical importance of somatopsychotic symptoms to formulating a general theory of delusions. However, the neglect of somatopsychotic symptoms by earlier empiricist theories (Bayne & Pacherie, 2004) drives an aetiological wedge through the reduplicative delusions (i.e., Capgras’ and somatopsychotic) and removes the means to explain even complex versions of the very Capgras’ delusions that empiricist theories were originally formulated to address.

### **Allopsychosis and confabulation**

There are a number of reasons why empiricist theories have failed to appreciate the relevance of confabulation to the aetiology of delusions. Maher’s (1974/1988) original emphasis on experientially generated content exerts a continuing influence, as does the continuing tendency to attribute confabulation to memory dysfunction. We will examine the relationship between confabulation and memory dysfunction later, but it is important to notice that even if one focuses on this, it has long been recognised that there are two cognitive processes that generate false memories; as Kopelman (1987) put it, “two types of confabulation”.

Bonhoeffer (1904) originally recognised the difference between provoked confabulation, involving minor distortions in recall, and spontaneous confabulation, involving “an alternative autobiographical or semantic narrative, which may or may

not accompany genuine lacunae in the ‘true’ account of matters” (Cutting, 1997, p. 355). Provoked confabulation is essentially a form of cognitive bias on which empiricist theories rely to act as their second factor, either in isolation or as part of the mechanism that leads to defective belief evaluation. However, recalling Weinstein’s (1991) description of the cognitive deficit in somatopsychosis, it is spontaneous confabulation that has the theoretical potential to explain complex content.

This realisation can be attributed to Johnson who, in a series of papers, developed a “reality monitoring” theory of confabulation and then extended this to explain delusions. According to Johnson, confabulations are false statements that are not made to deceive (1991, p. 187) and which occur when people “confuse the origin of information, misattributing something that was reflectively generated to perception” (p. 180). By “reflectively generated”, Johnson means “produced by the imagination” and this indicates that confabulations are essentially imaginations mistaken for memories.

Johnson refers to these errors as “reality monitoring failures” and suggests that they are more likely to occur when imaginations are rich in perceptual information and/or poor in cognitive operations information, as these make it more difficult to identify the source of information. However, Johnson’s difficulty is that reality-monitoring failure does not explain how often-complex imaginations arise in the first place. Johnson et al. (1997) solve this problem by hypothesising that confabulators have “a propensity towards detailed imaginations” (p. 203) and conclude that confabulation is due to an interaction between detailed imagination, defective source monitoring, and problems with the systematic retrieval of memories. We will argue later that confabulation does not require memory dysfunction and that the propensity to detailed imaginations and source monitoring problems form the basis of an adequate two-factor theory of confabulation.

In the remainder of this section we will concentrate on Johnson’s extension of her work on confabulation to explain delusions. One way of understanding Johnson’s strategy is to view her as, first, separating off the mistaking of *ongoing* imaginations for perceptions in order to explain hallucinations, and, second, arguing that delusions arise when specific imaginations are subject to reality monitoring failures. The difficulty is that most delusional content cannot be understood in terms of mistaking imaginations for memories and this suggests that reality-monitoring failures may account for only a subset of delusions, i.e., delusional memories. Interestingly, Kopelman (2009) recently emphasised the similarity between confabulation and delusional memories, whilst concluding that subsuming confabulation and delusion under a single theory has limited explanatory utility.

In fact, many imaginations seem to be accepted as they are imagined for the first time and this suggests that the reliance on detailed imaginations will have to be supplemented with an account of how ongoing imaginations can be mistaken for beliefs. Now, whilst Johnson herself does not articulate the issues this way, her (1988) treatment of hallucinations as “reality testing failures” (p. 35) contains crucial insights which can be generalised to produce the two-factor theory of delusions, which is consistent with the two-factor theory of somatopsychotic symptoms developed earlier.

In considering how hallucinations arise, Johnson’s first argument is that imaginations are so similar to perceptions that an individual could easily become

confused. However, since imaginations have insufficient perceptual information and too much cognitive operation information to be misclassified, Johnson recognises that this will not explain hallucinations. In order to address the perceptual information requirement, Johnson invokes abnormal experience through a discussion of Horowitz's suggestion that "hallucinations...may be elaborated from elementary sensations...in the retinal ganglionic and postretinal neural network and/or from anatomic bodies within the eyeball" (1988, p. 38).

Unfortunately, even when abnormal experience relieves the imagination of responsibility for some content generation, it takes a considerable amount of imaginative work to interpret *muscae volitantes* as rats, or the visual consequences of a partially detached retina as the blood of Christ (Johnson, 1988, pp. 38–39). Johnson's solution, which we can infer from the remark that "loss of control makes a self-generated event seem like a perceptual event" (p. 53), is to hypothesise that imaginations have little cognitive operations information attached to them. Johnson and Raye (2000) write, "if a confabulation is reasonably detailed and does not have strong cognitive-operations information associated with generating it, then it would be judged... 'real'" (p. 57).

If we now generalise this interpretation of Johnson's (1988) proposals to delusions, we find that a combination of abnormal experience and detailed imaginations, which have reduced cognitive operations information attached to them, explains how content arises and is not rejected. This proposal has the advantages of, first, embodying the main insight of empiricism by acknowledging the importance of abnormal experience, and, second, avoiding the need to introduce Coltheart et al.'s (2007) notion of defective evaluation, by ensuring that the two delusional hypothesis-generating factors contribute to the defective source monitoring, which explains why these hypotheses are not rejected: (1) experiential abnormalities, by furnishing thoughts with the perceptual information, and (2) confabulation, by reducing cognitive operations information attached to psychotic content.

It is worth emphasising the fundamental difference between Johnson's (1988) notion of defective monitoring, which focuses on the *process*, which gives rise to information, and Coltheart et al.'s (2007) notion of defective evaluation, which focuses on the *content* of information. Johnson's defective monitoring is therefore not at all what Coltheart et al. originally intended by defective evaluation, and in fact has more in common with the defective monitoring of actions which will be discussed in the next section. However, since Johnson's defective monitoring contributes to the explanation of how individuals make false statements without being aware that they are false, it is partly responsible for delivering what Coltheart et al. were trying to achieve by introducing defective evaluation, albeit without having to elevate this to the status of a factor in a theory of delusions.

Returning to the thread of the discussion, Johnson and Raye (2000) argue that perceptual information plays a significant role in heuristic (or nondeliberative) source monitoring and that cognitive operations information is important to systematic (or deliberative) source monitoring, which they suggest "may require interhemispheric cooperation" (p. 62). We will argue later that the defective monitoring that constitutes the second factor in a theory of confabulation is due to imaginations lacking cognitive operations information, and then relate this to problems with interhemispheric communication. However, for now the important point is that by reflecting on Johnson's (1988) work, we arrived at a two-factor theory

of visual hallucinations which is consistent with our two-factor theory of somatopsychotic symptoms and, in virtue of relying on a propensity to detailed imaginations, is capable of being seamlessly extended to explain even complex allopsychotic delusional hypotheses.<sup>1</sup>

### **Auto-noetic agnosia and confabulation**

Auto-noetic agnosia forms the basis of an entirely separate theory of psychosis (Frith, 1992; Frith & Done, 1989; Keefe, 1998; Larøi, Barr, & Keefe, 2004), a clarification of which will offer an opportunity to further underline the need for both confabulation and for a broader construal of the experiential factor than empiricist theories of delusions strictly allow. The approach is based on the idea that when, in ordinary circumstances, a motor instruction is sent, a corollary message or refference copy of this signal is also sent to an internal monitoring system. The purpose of the refference copy is to allow for an internal prediction of what will occur and, by comparing this prediction with perceived outcome, behaviour can be adjusted to meet the changing demands of complex tasks.

The system is a monitoring system that allows identification of self-generated stimuli (Cahill & Frith, 1996, p. 284), and, according to Cahill and Frith's (1996) version of the theory, psychotic symptoms occur when an individual executes an action without a "refference copy" of the motor instruction being available for internal monitoring, with the consequence that the individual is unable to recognise that the action was caused by himself. This approach to the monitoring of action is clearly well suited to explaining "certain auditory hallucinations (failure to monitor the initiation of inner speech and thought) and passivity experiences such as delusions of control (failure to monitor intentions to act)" (p. 285).

However, there are two sets of symptoms that are resistant. The first includes symptoms the content of which cannot be traced to the failure to identify a normal internal event. Cahill and Frith (1996) acknowledge these cases when they indicate that it is not clear the account can explain visual hallucinations. However, they do not mention equally problematic cases in which the content is in some sense *externally* generated, such as in psychotic illusions. The second set of symptoms that cannot be fully explained by auto-noetic agnosia are complex delusions. Indeed, even complex auditory hallucinations and passivity phenomena are problematic, in that it is one thing to believe that one's thought is a voice, but quite another to attribute it to evil spirits (to use one of Cahill and Frith's own case examples).

The only way of salvaging an aetiological contribution for auto-noetic agnosia is for Cahill and Frith (1996) to acknowledge the point made by Johnson and Raye (2000, p. 62) that more complex delusions are likely to involve additional factors, and to add confabulation to his theory. The modified version of the theory will then explain complex delusions and complex forms of symptoms which auto-noetic agnosia was originally directed at. However, notice that visual hallucinations remain recalcitrant, since these (as Johnson and Frith have effectively concluded from opposite directions) require the additional postulation of abnormal experience (Johnson & Raye, 2000; Cahill & Frith, 1996). This suggests that we will have to allow that abnormal experience in visual hallucinations plays an equivalent aetiological role to that played by auto-noetic agnosia in auditory hallucinations and passivity phenomena.



It is interesting that empiricist theories have effectively recognised this, although their attempts to bring auditory hallucinations and passivity phenomena under their own theory causes them to construe auto-noetic *agnosia* as abnormal experience. Coltheart and Davies suggest that “a cognitive abnormality, either in the generation of the efference copy and feedback information, might give rise to abnormal experiences of action . . . [which if accepted] as veridical would be a step on the way to the delusion of alien control” (2000, p. 37). This approach has an advantage over Frith’s theory because it brings out that auto-noetic *agnosia* is really just an experiential abnormality, thereby facilitating its assimilation into a two-factor theory. The difficulty, however, is that the implicit removal of the *agnosia* from auto-noetic *agnosia* is a further example of the experiential abnormality being construed too narrowly at the theory construction stage.

The neglect of confabulation is less obvious in this situation because it results from a distancing of auto-noetic *agnosia* from the *agnosias* underlying the somatoparaphrenic delusions that empiricist theories prior to Coltheart et al. (2007) did not attempt to accommodate. This is perhaps why Bayne and Pacherie (2005, p. 176, Note 7) detect Frith’s difficulties with complex delusions, whilst not perceiving that their own version of empiricism is susceptible to the same criticism. The result, in any case, is that we have again arrived at our original two-factor theory, only now, assisted by Frith’s contribution, the theory has been further refined by an understanding that the experiential abnormality underlying a further subset of psychotic symptoms, i.e., auditory hallucinations and passivity phenomena, is auto-noetic *agnosia*.

### **Memory dysfunction, frontal dysfunction, and confabulation**

Johnson and Raye (2000) argue that the distinction between the “delusion” and “confabulation” is governed by whether or not a particular false claim is made in the presence of brain damage or psychopathology and that “an aetiology-based distinction between delusions and confabulations [is] questionable” (p. 37). However, because Johnson’s work antedated the contributions that imposed the current structure on the debate about the aetiology of delusions (Bayne & Pacherie, 2004; Campbell, 2001; Currie, 2000; Langdon & Coltheart, 2000), a two-factor theory that included confabulation was not clearly articulated. On the other hand, theorists who have taken Maher’s proposals as a starting point have emphasised the experiential basis of monosymptomatic delusional content to an extent that the relevance of confabulation did not, at least until recently, come into view.

Metcalf et al. (2007) and Turner and Coltheart (2010) constitute an important attempt to progress beyond these obstacles by relating confabulation to a two-factor theory of delusions. However, before we try to show that these authors’ reliance on a common second factor “failure to reject unsubstantiated thought” (Turner & Coltheart, 2010, p. 357) inherits the original difficulties caused by focusing on evaluation at the expense of content generation, we need to lessen the resistance to the current proposals, by clarifying the relationship between confabulation and memory disorder. This is particularly important, because whilst Turner and Coltheart (2010) focus primarily on a common second factor shared by confabulations and delusions, and could, strictly speaking, remain neutral on the first-factor process that gives rise to confabulations, the current proposals rely on a component

of confabulation (detailed imaginations) to produce delusional content and as such confabulation cannot be due to memory dysfunction.

There is substantial evidence that memory impairment is not sufficient for confabulation. Hirstein (2005) refers to studies of patients with frontal lobe lesions (Shimamura, Janowsky, & Squire, 1990) and post anterior communicating aneurysm surgery (Vikki, 1985) who have memory impairment but do not confabulate; and second, studies of Korsakoff's Syndrome where memory impairment persists whilst confabulation improves (Stuss, Alexander, Lieberman, & Levine, 1978). The more difficult question is whether memory impairment is necessary for confabulation. Turner, Cipolotti, Yousry, and Shallice (2008) concluded that it does seem to be, although since some of their confabulating patients scored in the normal range on memory tests, and they could find no single measure of memory on which all of their sample were impaired, their findings do not exclude the possibility that memory impairment and confabulation involve damage to two different closely associated neural systems (Stuss et al., 1978). If one then considers that confabulation also occurs in hemisomatagnosia, corpus callosotomy, and schizophrenia in the absence of memory impairment (on schizophrenia see Lorente-Rovira et al., 2007; Nathaniel-James & Frith, 1996), it appears likely that memory impairment is not necessary for confabulation.

This does not exclude the possibility of coexisting memory dysfunction, and indeed cognitive impairment more generally, exacerbating confabulation (Dalla Barba, 1993), and it is perhaps worth noting the situation in which amnesia is associated with metamemory difficulties (i.e., a representational lacuna in the domain of memory). There is no reason why a representational lacuna relating to the memory could not function like a representational lacuna relating to the body and give rise to an experiential abnormality that would influence confabulatory content. In such cases, the resultant mnemonic confabulation would be what the theory proposed in the paper regards as a delusion, thereby giving substance to Kopelman's point about the relationship between confabulations and delusional memories. However, this is a special case that does not lend any more support than does hemisomatagnosia for a paralysed limb to the claim that confabulation in general is due to memory dysfunction.

The absence of a primary aetiological role for memory dysfunction must be accommodated by any attempt to relate confabulations and delusions and this brings us back to Metcalf et al.'s (2007) extension of their two-factor theory of delusions to confabulations. Essentially, Metcalf et al. suggest that the first factor in confabulation is operating at the same level as the first factor in delusion, although in the former it is mnemonic and in the latter perceptual or affective. This is somewhat difficult to reconcile with Turner and Coltheart's (2010) comment that a confabulation by a patient that he had met a woman who had a bee's head "did not involve distortion of true memory; instead it appeared to involve imagination" (p. 353). Furthermore, if, as this example suggests, memory dysfunction is not essential to confabulation, Metcalf et al. have identified a first factor that does not have a role in a theory of confabulations with a first factor that most authors agree does have a role in a theory of delusions.

The importance of Turner and Coltheart's (2010) comments about the role of the imagination in the "bee's head" confabulation now becomes apparent in that we require another first factor in a theory of confabulation and, according to the current

proposals, it is this factor that contributes to content generation in both confabulations and delusions. Notice, further, that Metcalf et al. (2007) cannot simply modify Coltheart et al.'s account of the aetiology of somatopsychotic symptoms and argue that since these involve confabulation, the first factor in confabulation must be experiential, as this places empiricism on the horns of a dilemma: Either deny that somatopsychotic and allopsychotic symptoms should be subsumed under the same theory of delusions, or accept that delusions, at least in complex cases, are not primarily experiential. The experiential basis of delusional content is closely associated with the claim that delusions are beliefs and together these represent the central tenets of the empiricism that Coltheart et al.'s original approximation of somatopsychotic and allopsychotic symptoms was intended to protect.

The possibility of filling the first factor gap left by the absence of memory dysfunction by extending the first factor experiential abnormality is almost equally unattractive to the current proposals as this would involve completely absorbing the concept of confabulation into the concept of delusion. The problem with this is that corpus callosotomy patients have neither memory nor experiential deficits in the sense required by the two-factor theory of delusions developed here, and yet they confabulate. So, providing we want to allow that this is essentially the same phenomenon as that which generates delusional content, which seems intuitively plausible, we must look to interhemispheric communication for the first factor in a theory of confabulation.

What corpus callosotomy cases indicate is that when the left hemisphere is deprived of information from the right hemisphere it generates interpretations and it is these that constitute confabulatory content. However, for this to occur, it may only be necessary that a portion of the corpus callosum or crucial adjacent structures are damaged. This could occur with a circumscribed lesion in the inferior medial prefrontal system (Turner et al., 2008), which would be consistent with evidence of dissociation between frontal executive dysfunction (the traditional alternative aetiology to memory dysfunction) and confabulation, even in schizophrenia (Lorente-Rovira et al., 2007). Let us suggest, then, that such a lesion is the cause of the first factor in a theory of confabulation, Johnson's propensity to detailed imaginations, which when combined with the defective monitoring to be discussed in the next section, constitutes the second factor in the proposed theory of delusions.

This suggestion is consistent with evidence of damage to the corpus callosum in schizophrenia (Downhill et al., 2000), but must be reconciled with delusions where there are first-factor experiential abnormalities due to a posterior lesion. Here one can envisage two possible routes to the propensity to detailed imaginations: first, through coexisting damage by a second lesion in the inferior medial prefrontal region, or, second, because the posterior lesion causing the experiential abnormalities is sufficiently extensive that it involves relevant structures. (See Feinberg, Venneri, Simone, Fan, & Northoff, 2010, for evidence that somatoparaphrenic patients have greater orbitofrontal damage than hemisomatagnosic patients.) Irrespective of which of these pertains, they entail that the first factor in delusions is not the first factor in confabulation per se, a proposal that firms up the distinction between hemisomatagnosic claims due to right-sided representational problems, and complex delusional content, such as in von Anygal and Frick's (1941) "paralysed brother case", due to inferior medial prefrontal damage.

### **Defective evaluation, defective monitoring, and confabulation**

We are now in a position to address the more complex problems associated with Turner and Coltheart's (2010) claim that confabulation and delusion share a common second factor. With this in mind, we have seen that empiricist theories in general share a common reliance on experience to generate delusional content and that they were originally formulated to explain monosymptomatic delusions, specifically Capgras' delusion, and it was suggested that this approach has difficulties explaining even complex Capgras' delusions. However, it was only when Coltheart et al. (2007) generalised their account to somatopsychotic symptoms (without attempting to address complex delusions) that the precise nature of the difficulties became apparent.

In order to bring out these difficulties, we need to revisit the key elements of Coltheart's et al.'s (2007) arguments:

suppose we construct a general theory . . . meant to apply to the explanation of all kinds of monothematic of delusion: to somatoparaphrenia, for example . . . What distinguishes left-hemiplegic people with somatoparaphrenia from these left-hemiplegic non deluded others. Whatever this is, it is something to do with the right hemisphere . . . because . . . the left hemisphere is typically intact . . . The function of this [damaged] region of the right hemisphere is, therefore, belief evaluation. (p. 644)

The important point to notice is that by not recognising the need to accommodate complex delusional contents, and then not building confabulations into a theory of delusions, Coltheart et al. had no alternative but to direct evaluation at the first factor in their theory of delusions. In other words, evaluation is directed at the representational problems that underlie psychotic symptoms, but which, according to the current proposals, are not susceptible to evaluation because experiential abnormalities themselves interfere with the ability to form correct judgements. Instead, it is the interpretations generated by the left hemisphere (which, as mentioned earlier, corpus callosotomy patients show us can be intact in confabulation) that provide the natural target for defective "evaluation".

Thus, when Turner and Coltheart (2010) apply Coltheart et al.'s (2007) theory of delusions to confabulations and postulate a "common set of evaluative and monitoring processes (the second factor)" (p. 357), this involves an "inter-level identification" between a factor that we have suggested has no role in a theory of delusions, i.e., defective belief evaluation, and a factor which Johnson holds has a central role in the aetiology of confabulation, i.e., defective monitoring. As a result the question of whether a theory of delusions can do without defective belief evaluation, which we have argued it can and should, is conflated with that of whether a theory of confabulation (and, by proxy, of delusions) can do without defective monitoring. We have already acknowledged that it cannot when discussing Johnson's reliance on cognitive operations information to explain why detailed imaginations are not rejected or, perhaps more accurately, correctly categorised (Johnson & Raye, 2000).

We will return to cognitive operation information shortly, but having concluded that experiential abnormalities interfere with the ability to make correct judgements in a way that obviates the need for defective evaluation of first-factor content in delusions, could we not simply redeploy Coltheart et al.'s (2007) defective evaluation against detailed imaginations? After all, it would seem that an individual's failure to

detect that his unsubstantiated hemisomatagnosic claims are false must at least in part be due to the right-sided representational lacuna itself. Hirstein (2005) has this possibility in mind, when he writes:

First, a false claim will be generated because the area responsible for that knowledge domain is damaged and some other much less competent area has generated the answer. Then the answer cannot be properly checked because the only area that can do this is damaged. (p. 147)

This would give partial substance to Coltheart et al.'s (2007) claim that the defective evaluation of evidence for an unsubstantiated claim plays a role in the failure to reject unsubstantiated delusional content. However, if we understand this as a higher level right-sided process defectively evaluating the content arising from a lower level right-sided process, then we will conflate hemisomatagnosic claims with complex delusional contents. Alternatively, if we understand the claim in terms of a higher level right-sided process defectively evaluating left hemispheric detailed imaginations, then since these would not arise without the presence of the first factor, there is no conceptual space for *nondefective* evaluation. In other words, the interference with the capacity to form correct judgements involves an interference with the capacity to evaluate the judgements thus formed, even if they are delivered by the left hemisphere.

The delusion that one's paralysed side is one's brother surely cannot in any meaningful sense be attributed to a failure to "evaluate" a false claim. In other words, it seems counterintuitive to claim that an individual is able to check, as it were, whether their arm is their brother or indeed, whether they have a power station inside them. Defective monitoring, however, is an important notion and this brings us back to the second factor in confabulation, and to Johnson and Raye's (2000) discussion of cognitive operations information and its relationship to interhemispheric relations. Now, we have previously attributed detailed imaginations to problems with the right to left transfer of information due to damage in the inferior medial prefrontal region. However, interhemispheric traffic is not one way, and this opens up the possibility that problems with the left to right transfer are responsible for detailed imaginations not having cognitive operations information attached to them.

Turner and Coltheart (2010) articulate defective evaluation/monitoring as "not tagging for doubt", but the introduction of the notion of a "tag" when viewed in light of the following comment suggests a move away from Coltheart et al.'s (2007) original construal of defective belief evaluation: "in the absence of a tag, fragments of ideas, or ideas purely derived from the imagination, would acquire the same status as fully formed beliefs" (p. 359). This formulation is consistent with the theory developed in this paper, providing that we understand "tag" in terms of cognitive operations information, and therefore defective monitoring as "not tagging for source". This avoids both the intuitive and theoretical difficulties associated with the notion of evaluating the content of detailed imaginations whilst successfully accounting for why they are not correctly categorised. Note also that the existence of *pseudologia fantastica* is evidence of conceptual room for the nondefective monitoring which is not possible if the process is directed at first-factor contents. In conclusion, then, the second factor in a theory of confabulations (and the second

part of the second factor in a theory of delusions) is defective source monitoring, caused by the very same damage in the inferior medial prefrontal system that causes the first factor.

Finally, it is worth adding that Currie (2000) proposes a theory of delusions according to which detailed imaginations are not correctly categorised because of defective source monitoring explicated in terms of a Frithian auto-noetic agnosia for the imagination. However, by neglecting experiential abnormalities in favour of “perception-like imaginings” (Currie & Ravenscroft, 2002, p. 11), Currie’s theory of delusions approximates to a theory of confabulation. In the course of examining Currie’s proposals, Bayne and Pacherie (2005) have noticed that “it may be more plausible to describe delusional patients as suffering from an impairment in the *control* of rather than the *monitoring* of imagination” (p. 177, emphases in original). This paper has tried to show that, in fact, an adequate theory of confabulation will include impairments in the control and not just the monitoring of the imagination, and that an adequate theory of delusions will include this and underlying experiential abnormalities.

### **Schizophrenia, thought disorder, and confabulation: Towards a unified theory of psychosis**

According to the current proposals, two separate aetiological processes come together to give rise to psychotic symptoms. If this is correct, then evidence of these processes coming apart and operating independently of one another would provide significant support for the theory. With this in mind, the presence of experiential abnormalities in schizophrenia is a relatively uncontroversial matter with Cutting (1997), for example, referring to Gross and Huber’s case of a female with schizophrenia who reported that the left side of her husband’s face suddenly seemed “so sad and serious . . . as if he were split into two parts” (p. 109). It is difficult to conceive of a reason for doubting that such symptoms tell us something important about the aetiology of psychotic symptoms.

However, it is the relationship between confabulation and schizophrenia, which has only recently started to attract empirical attention (Nathaniel-James & Frith, 1996), that is more interesting. It has long been recognised that patients with schizophrenia confabulate and, although these will naturally present as “provoked” in experimental settings, the historical literature contains unequivocal accounts of “spontaneous” confabulation. Lorente-Rival et al. (2007) refer to Kraepelin’s account of “extraordinary stories” (p. 1) and Fish (1974) points out that “some schizophrenics confabulate, producing detailed descriptions of fantastic events which have never happened” (p. 63).

The current proposals provide both a natural way of understanding the presence of confabulation in schizophrenia and a potential explanation of aspects of the phenomenology, such as the fact that ‘delusions tend [*sic*] to be fixed, compared with the shifting and changing nature of many confabulations’ (Kopelman, 1999, p. 202). In other words, perhaps psychotic individuals have a general tendency to confabulate and coexisting experiential abnormalities anchor a subset of confabulations to produce (two-factor) delusions. The more complicated question is whether the current proposal can advance the understanding of empirical correlates of confabulation in schizophrenia, and in particular Nathaniel-James and Frith’s

(1996) and Lorente-Rovira et al.'s (2007) finding that confabulation is associated with thought disorder.

The difficulty is, how do we understand this relationship and both authors question whether clinicians and researchers could be describing the same phenomenon in different ways. The main obstacle Lorente-Rovira et al. (2007) perceive to this possibility is that “the speech of neurological patients with confabulation . . . is understandable – it is only the factual content which strikes the listener as odd – whereas the defining characteristic of thought disorder is that speech become difficult to follow” (p. 8). The authors go on to make the point that “if it is accepted that confabulation in schizophrenia is different from the neurological form of the symptoms, then some phenomenological overlap with thought disorder might become a more viable option” (p. 8).

Lorente-Rovira et al. (2007) point out that empirical findings on confabulation in schizophrenia, namely the reorganising and restructuring of ideas in a story and the use of approximate and new words, resemble the manifestations of thought disorder. After referring to one of Chaika's (1974) patients who said his mother's name was Bill and that St. Valentine's day was the start of the official breeding season for birds, Lorente-Rovira et al. conclude that “while *episodic* confabulation, as seen in neurological disorders, and thought disorder, are not the same thing, conceptualising some aspects of thought disorder as *semantic* confabulation may be an idea with some heuristic value” (p. 9, emphasis in original).

Lorente-Rovira et al. (2007) do not, however, consider the possible role of confabulation in the aetiology of delusions and this both reintroduces the distinction between “two types of confabulation” (albeit in a different form to that proposed by Bonhoeffer, 1904, and Kopelman 1987) and drives an aetiological wedge between somatopsychotic and allopsychotic delusions. What is required is an interpretation of association between confabulation and thought disorder in schizophrenia that is informed by its proposed role in the aetiology of delusions, and there are two possibilities: First, confabulation and thought disorder are coexisting processes that have a joint impact on the use of language; second, confabulation and thought disorder are different manifestations of the same process.

The first interpretation is an extension of arguments about the nonessential relationship between memory/frontal executive dysfunction and confabulation. If confabulations are part of delusions, then the correlation between confabulation and thought disorder could be due to an underlying correlation between delusions and thought disorder. This interpretation would need to accommodate the phenomenological overlap between thought disorder and confabulation, but this could be explained by former being superimposed on the latter. Accordingly, Nathaniel-James and Firth (1996) suggest that “thought disorder may be a contributor to the severity of confabulation rather than its presence” (p. 397). One potential difficulty is that the current theory predicts a correlation between confabulation and delusions, and Nathaniel-James and Frith did not find this.

The second interpretation (favoured by Lorente-Rovira et al., 2007, but more difficult to reconcile with neuroanatomical considerations) is that confabulation and thought disorder are on a semantic–syntactic continuum between unsubstantiated claims and incoherent speech. The theory developed in this paper offers a way of reconciling this interpretation with Campbell's (2001) underexplored insight that “the really key question about the deluded subject is how the use that she makes of

the terms in which she frames her delusion relates to her knowledge of the meaning of those terms” (p. 95). Empiricist theories, on the other hand, are more or less committed to the “preservation of meaning” in delusions (Bayne & Pacherie, 2004, p. 2). The question is whether, given the requirement to explain both complex delusions and thought disorder in light of the association between confabulation and thought disorder, they can afford to be.

## Conclusion

This paper has argued that theories that rely on defective belief evaluation and/or cognitive bias cannot explain complex delusions, and that replacing these with confabulation solves this problem in a way that brings somatopsychotic and allopsychotic symptoms under one theory of delusions. The development of this theory yielded a two-factor theory of confabulation consisting of the propensity to detailed imagination and defective source monitoring. It was suggested that the two factors that lead to confabulation are due to reciprocal problems with interhemispheric communication consequent upon damage in inferior medial prefrontal region and that, as with delusions, memory and frontal executive dysfunction are not of primary aetiological relevance. It was argued that the relationship between confabulation and language impairments may also be nonessential, but that confabulation may be on a continuum with thought disorder and, if this were the case, the inclusion of confabulation in a theory of delusions provides a possible starting point for relating research on delusions to research on language impairments in schizophrenia.

## Note

1. The term “allopsychotic” is used here to refer to symptoms about the external world rather than the body.

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