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The two-factor theory of delusion

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Abstract

In the two-factor theory of delusion, the first factor explains why a delusional idea or hypothesis came to mind in the first place and the second factor explains why the hypothesis was adopted and maintained as a belief rather than being rejected—as it should have been—on the basis of available evidence and background knowledge that counted against it. The theory has been applied to cases of monothematic delusions—mirrored-self misidentification, Capgras, Cotard and Fregoli delusions, somatoparaphrenia and the delusion of alien control—in which the first and second factors have been neuropsychological impairments. It is not, however, part of the two-factor theory that the two factors must be neuropsychological. The theory has been applied to provide explanations—without appeal to neuropsychology—of cases of folie à deux, somatic delusion, and alien abduction delusion, for example. It has proved illuminating to consider the two-factor theory against the background of an eight-step model of the normal pathway from surprising facts to new beliefs, based on the work of the American pragmatist philosopher Charles Sanders Peirce—the Peircean pathway model. This has allowed a more substantive account of the associative processes by which a delusional hypothesis is generated in response to observation of a surprising fact. One consequence of the account is that hypothesis generation is a locus of individual differences. The model also provides a structure in which to investigate the cognitive nature of the failure of hypothesis evaluation. One proposal is that this is a bias against disconfirmatory evidence.

1. Introduction

The starting point for the two-factor theory of delusion is that an explanation of any case of monothematic delusional belief requires answers to two questions. The first question is:

> What initially prompted the delusional idea or hypothesis?

William James proposed that delusional ideas arise as putatively explanatory hypotheses prompted by bodily sensations: "The delusions of the insane are apt to affect certain typical forms, often very hard to explain. But in many cases they are certainly theories which the patients invent to account for their abnormal bodily sensations" (1890, Volume 2, p. 114). Brendan Maher made a similar proposal: "Strange events, felt to be significant, demand explanation. [A] delusion is a hypothesis designed to explain unusual perceptual phenomena" (1974, p. 103).

We think that James and Maher were broadly correct about our first question: delusional ideas arise as possible explanations of unpredicted phenomena. The trigger for the generation of a delusional idea or hypothesis is a prediction error (failed prediction) and the two-factor theory has been a prediction-error theory since its inception (Langdon & Coltheart, 2000; also see Coltheart 2005a, p. 73, 2005b, p. 155; for more on prediction error accounts, see Corlett, Ch. 31, this volume). It is clear, however, that an answer to the first question—an account of what prompted a delusional idea or hypothesis—would not yet provide an explanation of a case of delusion. The reason is that a delusional idea or hypothesis is not yet a delusion. It is not a belief—let alone a "fixed belief [that is] not amenable to change in light of conflicting evidence" (*DSM-5*, 2013, p. 87). Thus, an explanation of a case of delusion also requires an answer to a second question:

> Why was the delusional idea or hypothesis adopted and maintained as a belief rather than being rejected—as it should have been—on the basis of available evidence and background knowledge that counted against it?

An answer to the first question indicates a first factor in the explanation of a case of delusion—a factor that prompts hypothesis generation—whereas an answer to the second question indicates a second factor—resulting in a failure of hypothesis evaluation.

Plausible answers to our first question have been identified for cases of several monothematic delusions, including mirrored-self misidentification, Capgras delusion, Cotard delusion, Fregoli delusion, somatoparaphrenia and the delusion of alien control (see Table 1).[1] In these cases, the first factors have been neuropsychological in nature and (as Table 1 illustrates) first factors vary from delusion to delusion and may also vary between cases of the same delusion (e.g., mirrored-self misidentification). As a result of the first factor neuropsychological impairment, the person observes or encounters an unpredicted phenomenon—a surprising fact or event—and this, in turn, prompts the delusional idea or hypothesis (see Table 1, columns 1–3).

[1] For reviews, see Coltheart, 2007, 2010; Coltheart, Langdon & McKay, 2011; Coltheart, Menzies & Sutton, 2010; Davies, Coltheart, Langdon & Breen, 2001; Langdon & Coltheart, 2000.

Table 1

Six types of delusional condition, the specific unpredicted phenomenon associated with the delusion, the delusional hypothesis which would explain this phenomenon, and cases where the specific unexpected phenomenon associated with each delusion is present in people who are nevertheless not delusional.

Delusional condition	Unpredicted phenomenon	Delusional hypothesis	Non-delusional cases in which the unexpected phenomenon is present
Mirrored-self misidentification (e.g., Breen, Caine, Coltheart, Roberts & Hendy, 2000: case FE)	Failure to recognise the face one sees when looking into a mirror as one's own face.	The person I see when I look in the mirror is a stranger, not me.	Many people with prosopagnosia are not delusional.
Mirrored-self misidentification (e.g., Breen, Caine, Coltheart, Roberts & Hendy, 2000: case TH)	Mirror agnosia present, so mirrors treated as windows. The seen person appears to be in the space behind the glass.	The person I see when I look in the mirror is a stranger, not me.	Binkofski, Buccino, Dohle, Seitz & Freund (1999): mirror agnosia without delusion.
Capgras delusion (e.g., Edelstyn & Oyebode, 1999)	Failure of autonomic response to familiar faces (e.g., face of spouse).	This person I am looking at is a stranger, not my spouse.	Following neurosurgery to treat intractable epilepsy, a patient reported that there was something different about her mother—"it didn't feel like her"; but the patient had no delusion. (Turner & Coltheart, 2010).
Cotard delusion (e.g., Young, Robertson, Hellawell, de Pauw & Pentland, 1992)	Depersonalisation (e.g., de-emotivity, derealisation, de-somatisation).	I am dead.	Many people with depersonalisation symptoms do not have Cotard delusion. [a]
Fregoli delusion (e.g., Langdon, Connaughton & Coltheart, 2014)	Presence of autonomic response even to unfamiliar faces. [b]	People with whom I am familiar are present in my environment, disguised.	Vuilleumier, Mohr, Valenza, Wetzel & Landis (2003): strong autonomic responses to unfamiliar faces (we presume) but no delusion.
Somatoparaphrenia (e.g., Vallar & Ronchi, 2009)	Paralysis and loss of kinaesthetic and proprioceptive feedback from the arm.	This limb (the paralysed limb) is not mine, it is someone else's.	Many people with a paralysed limb and without kinaesthetic and proprioceptive feedback are not delusional.
Passivity delusion ("alien control") (e.g., Stirling, Hellewell & Quraishi, 1998)	Failure of cancellation of feedback from motor response by efference copy.	Other people can cause my limbs to move without my volition.	In "haptic deafferentation", the patient gets no sensory feedback from actions performed (Fourneret, Paillard, Lamarre, Cole & Jeannerod, 2002). But no delusion present.

Notes

[a] A diagnosis of depersonalisation disorder requires that reality testing is intact. Patients describe their experiences in 'as if' terms.

[b] Here, we adopt a suggestion by Ramachandran and Blakeslee (1998, p. 171). But we also note that Langdon et al. (2014) argue, against this suggestion, that "the Fregoli delusional content is generated when hyperexcitation from the cognitive system to the PINs [person identity nodes] causes a known person to be identified as present, even when no matching face is also present" (2014, p. 628).

Here are two examples. First, as a result of significantly impaired face processing (Breen, Caine & Coltheart, 2001), patient FE encountered the unpredicted phenomenon of seeing in the mirror a face that he did not recognise as his own face, and this prompted the idea that the person that he saw in the mirror was not himself. Second, as a result of disconnection of the face processing system from the autonomic nervous system, a person with Capgras delusion encounters an unpredicted phenomenon—the absence of the predicted autonomic response to a familiar face (e.g., the spouse's face). The person (consciously) observes only that there is something odd about the viewed individual but, by unconscious processes that draw on the information that the faces of strangers do not evoke autonomic responses, the unpredicted phenomenon prompts the idea that the individual is a stranger.

Delusional ideas or hypotheses should be rejected on the basis of available evidence and background knowledge that counts against them but, in every case of delusion, a delusional hypothesis is adopted and maintained as a belief. This failure to reject the hypothesis is not explained by the first factor (indicated by the answer to the first question) but by a second factor (indicated by an answer to the second question). Earlier expositions of the two-factor theory have typically argued the need for a second factor by presenting cases in which the first factor is present but the corresponding delusion is absent. This is not a matter of presenting, for each delusion, a single case of dissociation: first factor without delusion. Rather, there are large numbers of people who have the first factor impairment, and observe or encounter the unpredicted phenomenon that prompts the delusional idea, but who do not have the delusion. This shows that the first factor (and the resulting unpredicted phenomenon) does not, by itself, explain the delusion; there must be at least one explanatory factor in addition to the first factor. (For arguments against the need for a second factor, see Noordhof and Sullivan-Bissett, 2021; Sullivan-Bissett, 2022; and Sullivan-Bissett, Ch. 29, this volume. Note that arguments for or against a second factor in delusions may depend on how the notion of a 'factor' is understood.)

The question might be raised, however, why this shows that there must be, not just some additional factor or other but, specifically, a second factor that results in a failure of hypothesis evaluation. In earlier expositions of the argument for a second factor there has been an implicit assumption that the delusional idea comes to the minds of all people who have the first factor impairment and observe or encounter the resulting unpredicted phenomenon. Given this assumption, all people with the first factor but not the corresponding delusion have entertained the delusional hypothesis and have rejected it as false—or, at least, have declined the opportunity to adopt and maintain it as a belief. These non-delusional people demonstrate that having the first factor and generating the delusional hypothesis does not inevitably lead to the delusion. By comparison with the non-delusional people, the people who adopt and maintain the delusional hypothesis as a belief show a failure of hypothesis evaluation. (We shall return to this point in Section 4, below.)

There is some reason to propose that, in the cases under discussion (in which the first factor is neuropsychological in nature), the second factor—resulting in failure of hypothesis evaluation—is also neuropsychological, with a neural basis in damage to, or hypoactivation of, right dorsolateral prefrontal cortex (rDLPFC). Coltheart (2007, 2010) reviewed evidence supporting this proposal and Coltheart and colleagues (2018) found that repetitive transcranial magnetic stimulation (rTMS) to rDLPFC (but not rTMS to left DLPFC) resulted in

healthy subjects being less likely to reject the false hypotheses embodied in hypnotic suggestions.

In the early development of the two-factor theory of delusion, the methodology of the (then) emerging discipline of cognitive neuropsychiatry (David 1993; Halligan and David 2001) was adopted. That is, the methods of cognitive neuropsychology were applied to psychiatric disorders—specifically, to delusions (for discussion, see Young, 2000). Questions about the scope of the theory were explicitly addressed (e.g., Coltheart, 2005a). One option would have been to limit the scope of the theory to neuropsychological cases of delusion and to leave it to some other theory to explain other cases. The option that was actually adopted was:

> to explore the idea that, in any delusion, there is a factor that explains where the idea came from in the first place and a second factor that explains why the idea becomes an enduring belief rather than being rejected; but these factors are not always neuropsychological deficits. (2005a, p. 75)

2. The Two-Factor Theory and the Peircean Pathway Model

According to the two-factor theory of delusion, the first factor results in the person observing or encountering an unpredicted phenomenon—a surprising fact or event—and this prompts the delusional hypothesis. In early expositions of the theory, rather little was said about the process of hypothesis generation that brings the delusional hypothesis to mind. The second factor results in a failure of hypothesis evaluation but, again, rather little was said about the processes that are involved in hypothesis evaluation and how they might fail. It has proved illuminating to consider the two-factor theory against the background of an eight-step model of the normal pathway from surprising facts to new beliefs, based on the work of the American pragmatist philosopher Charles Sanders Peirce (1839–1914)—the Peircean pathway model (see Figure 1).[2] As Marshall and Halligan remarked:

> One would … hope that theories of normal belief-formation will eventually cast light on both the content of delusions and on the processes by which the beliefs came to be held. (1996, p. 8)

Peirce used the term "abduction" for the process by which explanatory hypotheses are generated from surprising observations:

> Long before I first classed abduction as an inference it was recognized by logicians that the operation of adopting an explanatory hypothesis,—which is just what abduction is,—was subject to certain conditions. Namely, the hypothesis cannot be admitted, even as a hypothesis, unless it be supposed that it would account for the facts or some of them. The form of inference therefore is this:
>
> The surprising fact, *C*, is observed;
> But if *A* were true, *C* would be a matter of course.
> Hence, there is reason to suspect that *A* is true.
>
> (Peirce, 1903/1998, p. 231)

[2] For discussion of the Peircean pathway model, see Coltheart & Davies, 2021a, 2021b, 2022; Davies & Coltheart, 2020.

This three-line inference, set out in Peirce's 1903 *Harvard Lectures on Pragmatism*, is commonly considered to be his canonical rendering of the logical form of abductive inference.

We can see how Peirce's work on abduction—and, particularly, his abductive inference—might shed light on the processes implicated in the formation of delusional beliefs if, for example (see Table 1, case FE), we take Peirce's surprising fact *C* to be:

> I do not recognise the face that I see when looking into the mirror as my own face.

and his hypothesis *A* to be:

> The person I see when I look in the mirror is a stranger, not me.

The three-line abductive inference would then be:

> The surprising fact *C*, "I do not recognise the face that I see when looking into the mirror as my own face", is observed;
> But if *A*, "The person I see when I look in the mirror is a stranger, not me", were true, *C* would be a matter of course.
> Hence, there is reason to suspect that "The person I see when I look in the mirror is a stranger, not me" is true.

Step 1 along the Peircean pathway is observation of a surprising fact and Step 2 is generation of a hypothesis that putatively meets the critical criterion that if the hypothesis were true, the surprising fact would follow as a matter of course. If hypothesis generation is understood as an empirical—not necessarily ideal—process then it would be as well to confirm (i) that the generated hypothesis actually met the critical criterion. In some of his work (e.g., 1901/1998, pp. 106—110), Peirce proposed additional desiderata: the hypothesis should be (ii) testable and (iii) reasonably economical. Here, the notion of economy encompassed both efficient deployment of limited resources—time, energy and money—and explanatory virtues such as breadth, depth, simplicity and naturalness—the features of a hypothesis that contribute to its 'loveliness' in the terminology of Lipton (2004).[3] Thus, Step 3 along the Peircean pathway is a threefold assessment of the generated hypothesis. If the hypothesis does not satisfy the critical criterion and the additional desiderata then the system must return to Step 2 and generate a new hypothesis that might satisfy the three requirements. If, on the other hand, the hypothesis generated at Step 2 does satisfy the critical criterion and the additional desiderata at Step 3 then it is passed on to Step 4—the step of abductive inference.

[3] In Lipton's (2004) terminology, the *loveliest* hypothesis or candidate explanation is the one that would "provide the most understanding", whereas the *likeliest* is the one that is "best supported by the evidence" (p. 57).

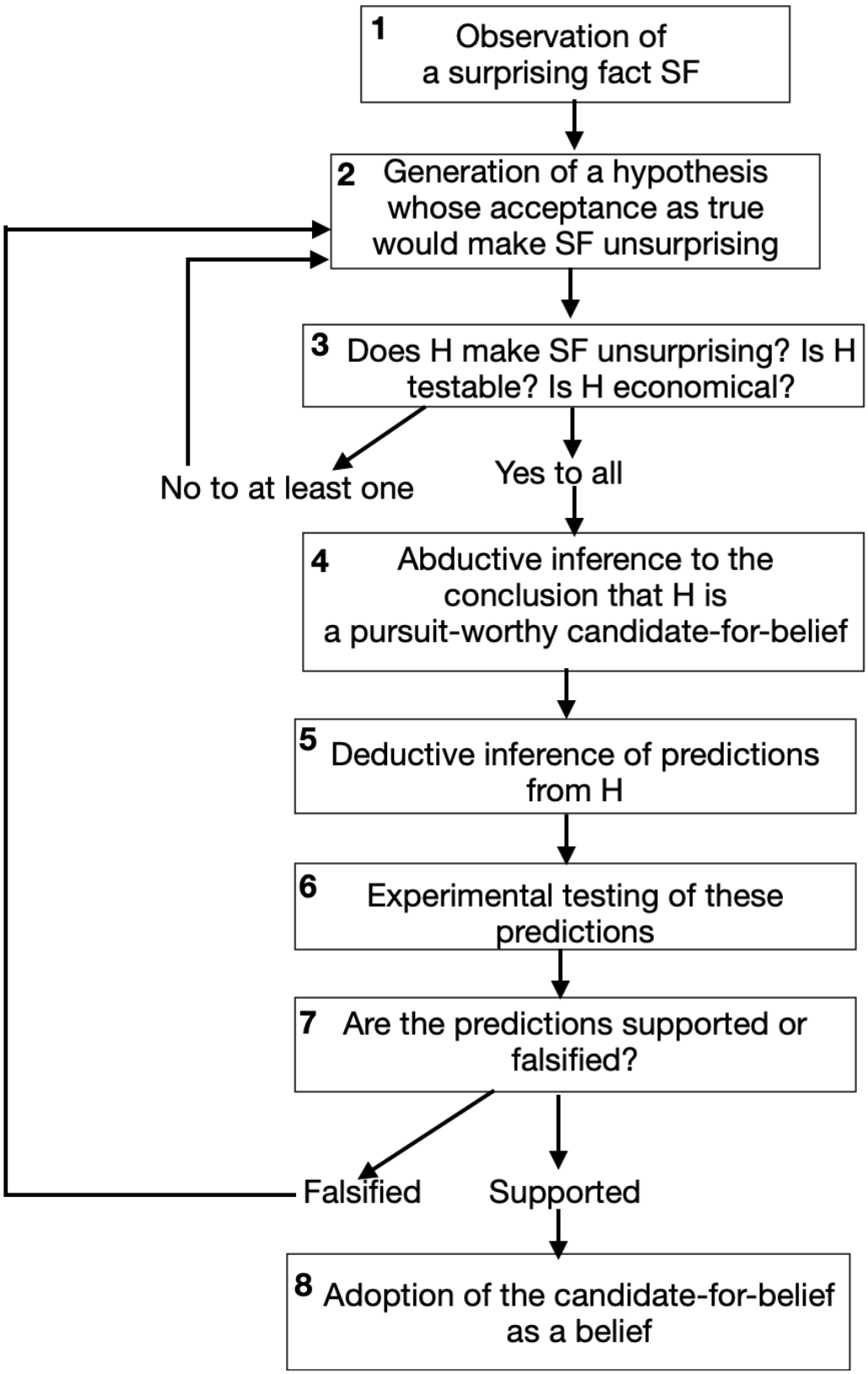

Figure 1
Peircean pathway model of the adoption of a new belief in response to the observation of a surprising fact (Coltheart & Davies, 2021a, 2021b, 2022; Davies & Coltheart, 2020).

How are we to understand the conclusion of the abductive inference: "There is reason to suspect that *A* is true"? It is important that the conclusion is not that hypothesis *A* provides the best explanation of surprising fact *C*—Peirce's abduction is *not* inference to the best explanation. Nor is the conclusion that hypothesis *A* is true, nor even that *A* is probably true. Peirce, himself, said: "Abduction is the process of forming an explanatory hypothesis. … Abduction merely suggests that something *may be*. … [I]t merely offers suggestions" (1903/1998, pp. 216–217). We interpret the conclusion of the abductive inference along the lines that hypothesis *A* is a candidate or 'suspect' for being the true explanation of the surprising fact *C*.

The abductive inference at Step 4 has three premises, taking account of Peirce's additional desiderata:

> The surprising fact, *C*, is observed;
> But if the hypothesis *A* were true, *C* would be a matter of course.
> The hypothesis *A* is testable and reasonably economical.

The conclusion is:

> The hypothesis *A* can be considered a pursuit-worthy candidate for being the true explanation of the surprising fact *C***.**

Following the step of abductive inference, the hypothesis—or candidate-for-belief—must be tested. Step 5 along the Peircean pathway is deductive inference of predictions from the candidate-for-belief and Step 6 is the testing of these predictions. Peirce usually focused on experimental testing of predictions but predictions can also be tested by observations or by drawing on available background knowledge. Step 7 is then assessment of the candidate-for-belief in the light of the results of the Step 6 testing. If the predictions are falsified then the candidate-for-belief is rejected and the system must return to Step 2 and generate a new hypothesis. If the predictions are supported then the candidate-for-belief may be adopted as a belief at Step 8. (Peirce referred to this process of testing and confirmation as "induction", so that the pathway comprised abduction, deduction and induction.)

When we consider a case of delusion, the first factor results in observation of a surprising fact at Step 1 of the Peircean pathway and this triggers a process of hypothesis generation at Step 2. Pre-testing assessment of the generated delusional hypothesis at Step 3 is then followed, at Step 4, by adoption of the hypothesis, *not* as the *true* explanation of the surprising fact or as a *belief*, but as a *possible* explanation of the surprising fact and as a *candidate-for-belief*. The person commits no error in considering the delusional hypothesis as a candidate-for-belief, but the candidate should be rejected and an alternative hypothesis should be generated. The person's adoption and maintenance of the delusional hypothesis as a belief demonstrates a failure of hypothesis evaluation at one or more of Steps 5–7—resulting from the second factor.

In the following four sections, we shall consider in more detail the processes of hypothesis generation (Step 2) and of hypothesis evaluation (Steps 5–7).

3. Individual Differences in Hypothesis Generation

Concerning Step 2, Peirce said:

> The inquiry begins with pondering these [surprising] phenomena in all their aspects, in the search of some point of view whence the wonder shall be resolved. At length a conjecture arises that furnishes a possible Explanation. (1908/1998, p. 441)

Peirce also referred to "[t]he whole series of mental performances between the notice of the wonderful phenomenon [*C*] and the acceptance of the hypothesis [*A*]" (ibid.). He wrote about psychological—particularly, associative—processes and he conducted psychological experiments. But he did not provide a fully satisfactory account of the psychological processes by which a hypothesis is generated.

Drawing on hints from Peirce and work by Pinker (2005) and Rellihan (2009), Coltheart and Davies (2021a) proposed that hypothesis generation is an associative heuristic procedure that takes advantage of two features of spreading-activation networks: direct access to content-addressable memory (rather than potentially exhaustive serial search) and pattern completion. There is, however, a residual concern arising from the fact that, for a given surprising fact, there will be many hypotheses with the property that, if the hypothesis were true, the surprising fact would follow as a matter of course. In fact, as Peirce remarked, "the possible explanations of our facts may be strictly innumerable" (1901/1998, p. 107). Having Step 2 pass innumerably many hypotheses to Step 3 for assessment cannot be an aspect of a realistic account of human cognitive processes. In response to this concern, we can note that, on any associative account, the time taken to generate a hypothesis will vary as a function of the strength of its association with the observed surprising fact. So hypothesis generation will be asynchronous and, for any given surprising fact, one hypothesis will be generated first—at which point the operation of Step 2 could be paused. In fact, Coltheart and Davies (2021a) proposed that, from Step 2 onwards along the Peircean pathway, "only one hypothesis is being entertained at any one time" (p. 7).

Research using the Alternative Uses paradigm (Guilford, 1971; Guilford, Christensen, Merrifield & Wilson, 1978) casts further light on the nature of associative evocation of hypotheses. In this paradigm, participants are asked to generate as many alternative uses for a familiar object as they can; for example, "What can you do with a brick?". Gilhooly, Fioratou, Anthony and Wynn (2007) reported: "First responses tend to be based on contextualized personal experience stored as episodic or more generalized autobiographical memories" (p. 623) and they provided an example: "I remember my father used a brick to stop a car rolling away" (p. 615). Some later responses were based on "retrieval of one or more properties of the target object and a search of semantic memory for uses or functions which have as a requirement the retrieved property or properties" (p. 618). An example of an alternative use for a pencil was: "A pencil is sharp so can be used to poke holes in paper" (p. 615).

Associative evocation of explanatory hypotheses draws on autobiographical memory and semantic memory (available knowledge of facts) no less than does performance of the Alternative Uses task. Furthermore, it is beyond dispute that there are individual differences in autobiographical memory (not everyone remembers their father using a brick to stop a car

rolling away) and in semantic memory (different people know different facts). It follows that hypothesis generation at Step 2 of the Peircean pathway is a locus of individual differences. For example, a patient with Cotard delusion gave an account—after her recovery—of how, when she was in the acute phase of catatonia (of which immobility is a frequent symptom), the idea that she was dead came to mind: "she thought that she was dead because of the feeling that time was passing extremely slow, and because she could not talk or move despite her will" (Ramírez-Bermúdez et al., 2021, p. 67). A doctor specialising in the diagnosis, treatment, and neurological nature of catatonia—with quite different background knowledge from the patient—might one day experience the same unpredicted phenomena as the patient but not generate the Cotard delusional hypothesis, "I am dead". The doctor's feeling that time was passing slowly and his inability to talk or move might, instead, evoke the hypothesis, "I am suffering from catatonia".

4. Individual Differences and the Argument for a Second Factor

If a large number of individuals observe or encounter the same surprising fact or event at Step 1 then we should predict that there will be some variation in the explanatory hypotheses that are generated at Step 2. This certainly calls into question the implicit assumption (mentioned toward the end of Section 1, above) that the same delusional idea comes to the minds of all people who have the same first factor and observe or encounter the resulting unpredicted phenomenon.

It might seem that if the implicit assumption is called into question then the argument for a second factor (specifically, a second factor that results in a failure of hypothesis evaluation) is also called into question—but that would not be correct. The argument for a second factor depends on there being people of two kinds. There are people with the first factor who, having generated the delusional hypothesis, evaluate it and reject it and are not delusional; and there are other people with the first factor who, having generated the same delusional hypothesis, adopt and maintain it as a delusional belief. The implicit assumption had the consequence that *all* people with the first factor who are not delusional are of the first kind: they generated the delusional hypothesis and then evaluated and rejected it. Without the assumption, we must allow that there might be people with the first factor who are not delusional simply because they did not generate the delusional hypothesis. Nevertheless, provided there were still people of the first kind, the argument would proceed in just the same way.

People of the first kind demonstrate that having the first factor and generating the delusional hypothesis does not inevitably lead to the delusion. So, concerning any person of the second kind, we can ask (our second question) why the delusional hypothesis was not rejected. What explains this person's failure of hypothesis evaluation? The second factor is whatever is indicated by the answer to that question. In order to resist the argument for a second factor, a critic of the two-factor theory must argue that there are no people of the first kind; the only way to avoid delusion is not to generate a delusional hypothesis in the first place. Thus, the critic must claim that the possibility that a person with the first factor might generate the delusional hypothesis and then evaluate and reject it can be excluded. The onus would be on the critic to justify that claim.

Might the needed justification be provided by a Spinozan account of belief formation? As James summarised this account, "all propositions are believed through the very fact of being conceived" (1890, Volume 2, p. 290) and, as Gilbert (1991) explained, "Spinoza argued that to comprehend a proposition, a person had implicitly to accept that proposition" (p. 108). (For an extended defence of the Spinozan account, see Mandelbaum, 2014.) On such an account it would, indeed, be impossible for a person to evaluate a generated hypothesis before believing it. Consequently, there would be no people of the first kind—no people who, having generated a delusional hypothesis, evaluated and rejected it rather than believing it.

Adoption of a Spinozan account would not, however, allow a critic to resist the argument for a second factor. A Spinozan account excludes evaluation of a hypothesis before it is initially adopted as a belief—evaluation that would lead either to acceptance or else to rejection of the hypothesis. But evaluation is re-located to a point after initial acceptance of a hypothesis as a belief. This post-acceptance evaluation leads either to certification or else to unacceptance of the belief (see Gilbert, 1991, p. 109, Figure 1; also see Mandelbaum, 2014, p. 62, Figure 2). If a Spinozan account is assumed then the second factor in the two-factor theory does not, strictly speaking, result in a failure of hypothesis evaluation but rather in a failure of belief evaluation. In this new setting, the argument for a second factor depends, once again, on there being people of two kinds. There are people with the first factor who, having generated the delusional hypothesis and initially accepted it as a belief, evaluate it and unaccept it and are not delusional; and there are other people with the first factor who, having generated the same delusional hypothesis and initially accepted it as a belief, certify and maintain the delusional belief.

People of the first kind demonstrate that having the first factor, generating the delusional hypothesis and initially accepting it as a belief does not inevitably lead to the delusion. By comparison with these non-delusional people, any person of the second kind—who certifies and maintains the delusional belief—shows a failure of belief evaluation, and we can ask what explains that failure. Again, the second factor is whatever is indicated by the answer to that question. To resist the argument for a second factor given a Spinozan account, the critic must claim that the possibility that a person with the first factor might generate the delusional hypothesis, initially accept it as a belief, and then evaluate and unaccept the delusional belief can be excluded. Once again, the onus would be on the critic to justify that claim.

The Peircean pathway model (see Figure 1) assumes that a generated hypothesis is adopted at Step 4 as a research question to be investigated and that the hypothesis is adopted as a belief at Step 8 only if "prediction after prediction … is verified by experiment" (1901/1998, p. 97). Peirce did allow, however, that sometimes—perhaps if the hypothesis was considered to be especially pursuit-worthy at Step 4—a subject might not just express the hypothesis "in the interrogative mood", but might have an "uncontrollable inclination to believe" it (1908/1998, p. 441). We interpret this to mean that a subject might proceed directly from Step 4 to Step 8, altogether omitting Steps 5–7. A belief that is adopted as the result of uncontrollable inclination still stands in need of evaluation and, as the Peircean pathway model is configured, this post-adoption evaluation would require a return from Step 8 to Step 5. If a Spinozan account of belief were adopted then the model could be reconfigured to have the generated hypothesis initially accepted as a belief at Step 4 (or, even

earlier, at Step 3), with post-acceptance testing at Steps 5–7. The belief would be unaccepted if was refuted, or certified at Step 8 if it was supported.

5. Failure of Hypothesis Evaluation and the BADE Paradigm

Concerning the steps to be taken after Step 4, the step of abductive inference when the generated hypothesis is adopted as a candidate-for-belief, Peirce said:

> That which is to be done with the hypothesis is to trace out its consequences by deduction, to compare them with results of experiment …, and to discard the hypothesis, and try another, as soon as the first has been refuted.
> (Peirce, 1901/1998, p. 107)

The Peircean pathway model's identification of three steps between adoption of the generated hypothesis as a candidate-for-belief (Step 4) and adoption of the hypothesis as a belief (Step 8) provides a structure in which to investigate the cognitive nature of the failure of hypothesis evaluation that results from the second factor. There could be a failure at Step 5 to derive predictions from the delusional hypothesis; or a failure at Step 6 to test derived predictions by experiment or observation or by drawing on available background knowledge; or a failure at Step 7 to recognise that the results of testing have falsified the predictions, to reject the refuted hypothesis and to generate an alternative. Whereas the first factor—and the resulting unpredicted phenomenon and the associatively evoked delusional hypothesis—inevitably varies from delusion to delusion, the second factor always results in a failure of hypothesis evaluation. In principle, however, the two-factor theory allows that the precise nature of this failure of hypothesis evaluation—and its location at Step 5, 6 or 7—might vary from case to case.

Coltheart and Davies (2021b) examined several published cases of delusion to find out whether Steps 5 and 6 were executed properly. In some cases, predictions were clearly derived (Step 5) and tested (Step 6). For example, patient YY (Brighetti, Bonifacci, Borlimi & Ottaviani, 2007) had Capgras delusion and believed, "This person is not my father, but a stranger". From the Capgras delusional hypothesis, patient YY derived predictions, such as "This person will not be able to answer questions about my childhood", and tested these predictions (e.g., by asking the person questions). Given the results of this testing (e.g., patient YY's father answered the questions correctly), patient YY should have recognised that the predictions were falsified, rejected the refuted hypothesis and generated an alternative; but that is not what happened. There was a failure of hypothesis evaluation at Step 7. In other cases, people derived from their delusional hypothesis predictions that were falsified by background knowledge that was already available—even without experiment or observation. But, again, there was a failure of hypothesis evaluation at Step 7. They did not reject the refuted hypothesis.

There may be other cases of delusion in which one or other (or both) of Steps 5 and 6 are not properly executed but it seems that, in at least some cases, there is a failure of hypothesis evaluation at Step 7. One possibility to consider is that this failure is not specific to the delusional hypothesis but results from a more general failure to reject—or, at least, to reduce credence in—hypotheses in the face of counter-evidence. There is a substantial body of research using the BADE paradigm to investigate a bias against disconfirmatory evidence in

individuals with schizophrenia (e.g., Sanford, Veckenstedt, Moritz, Balzan & Woodward, 2014).

In the BADE paradigm, subjects rate the plausibility of four possible interpretations of a scenario that is presented in three consecutively-presented statements (see Table 2). After the first statement is presented (and then after each successive statement), the subject rates each interpretation on a scale from 0 (implausible) to 10 (very plausible). Two 'lure' interpretations seem plausible after the first statement but are disconfirmed by later statements. An absurd interpretation seems implausible after the first statement and remains implausible. A true interpretation does not seem to be the most plausible after the first statement but is confirmed by the third statement. A bias against disconfirmatory evidence is demonstrated by the subject's failure to reduce the ratings of the initially plausible 'lure' interpretations as they are shown to be implausible. (A bias against confirmatory evidence would be demonstrated by failure to increase the rating of the true interpretation.)

Table 2
Two examples from the BADE paradigm

	Scenario	
	STATEMENT 1 **Amy doesn't like high waves** **STATEMENT 2** **Amy has already been under water for a few minutes** **STATEMENT 3** **It is Amy's job to be at the beach** [a]	**STATEMENT 1** **Jenny can't fall asleep** **STATEMENT 2** **Jenny can't wait until it is finally morning** **STATEMENT 3** **Jenny wonders how many presents she will find under the tree** [b]
	Interpretation	
1	Amy is learning wind-surfing	Jenny is nervous about her exam the next day
2	Amy witnessed the tsunami catastrophe in Thailand	Jenny is worried about her ill mother
3	Amy is afraid that fish might bite her on the nose	Jenny loves her bed
4	Amy is a lifeguard	Jenny is excited about Christmas morning

Notes
[a] See Veckenstedt, Randjbar, Vitzthum, Hottenrott, Woodward and Moritz (2011, p. 178).
[b] See Sanford, Veckenstedt, Moritz, Balzan and Woodward (2014, p. 2730).

In their study of 214 subjects, of whom 164 had a diagnosis of schizophrenia (or schizo-affective disorder), Sanford and colleagues (2014) found that 'evidence integration' scores (a composite score for bias against disconfirmatory evidence and bias against confirmatory evidence) were significantly higher in highly delusional individuals with schizophrenia than in low-delusional individuals with schizophrenia, people with obsessive-compulsive disorder, and healthy control subjects. In a meta-analysis of eight studies using the BADE task, McLean, Mattiske and Balzan (2017) found a stronger bias against disconfirmatory evidence in schizophrenia patients with delusions than in schizophrenia patients without delusions and healthy control subjects. These findings are consistent with the proposal that a general cognitive bias against disconfirmatory evidence "may underlie maintenance of delusions in the face of counter-evidence" (Sanford et al., 2014, p. 2729), but we know of no research

using the BADE paradigm to investigate people with delusions but without a psychiatric diagnosis.

6. Bias Against Disconfirmatory Evidence in Patients with Anosognosia

There is some evidence of a bias against disconfirmatory evidence in patients with anosognosia for their left-side motor impairments following right-hemisphere stroke. Anosognosia is a delusion but the delusional hypothesis ("I can move my left arm and leg") is not newly generated, and so the early steps of the Peircean pathway model are not relevant. The answer to our first question, "What initially prompted the delusional idea or hypothesis?", is that it has been true throughout the patient's life—until the brain injury that resulted in the patient's motor impairments. First factor neuropsychological impairments in anosognosia do not result in observation of a surprising fact or event. Instead, they prevent the patient from observing the surprising fact of their motoric failure when they try to move their arm or leg and, in some cases, they result in an illusion of motoric success—illusory limb movements (Davies, Aimola Davies & Coltheart, 2005; Davies, McGill & Aimola Davies, in press). There is still a mass of available evidence against the delusional hypothesis: patients cannot stand up from their chair or walk upstairs unaided and cannot perform bimanual tasks such as tying a knot or picking up a tray of glasses. The answer to our second question, "Why was the delusional idea or hypothesis maintained as a belief rather than being rejected—as it should have been—on the basis of available evidence against it?", is the same as before: a second factor results in a failure of hypothesis evaluation at one or more of Steps 5–7.

Is there a failure, specifically, at Step 7 in patients with anosognosia? Vocat, Saj and Vuilleumier (2013) used a riddle task in a study of right hemisphere stroke patients—with or without anosognosia—and healthy control subjects. Subjects were given five successively more informative clues and, after each clue, they were asked to guess the target word (see Table 3). The first clue was sufficiently uninformative to create doubt in healthy subjects, while the final clue was intended to leave no doubt about the correct answer. The measure of bias against disconfirmatory evidence was the number of times (across ten riddles) the subject proposed the same incorrect guess following two consecutive clues in the same riddle.

Patients with anosognosia were no less likely than the other two groups to respond with the correct target word after the final clue, but were more than twice as likely as the other two groups to repeat the same incorrect guess.

> This suggests that anosognosics had no general problem in reasoning but required a repeated signal of errors, or a larger incongruence between a new clue and the previous guess, in order to prompt a re-appraisal of their preceding responses and to trigger a new solution. (Vocat et al., 2013, p. 1778)

In other words, the patients with anosognosia showed a bias against disconfirmatory evidence.

Table 3
Two examples from the riddle task

Clue	Target Word	
	CARROT	HEART
1	I am a food	My weight is approximately 300 g
2	I am very cultivated	I produce a regular sound
3	I am a vegetable	Sport makes me feel excited
4	I am usually orange	I am usually on the left rather than on the right side
5	The rabbits adore me	Lovers often draw me

The proposal that, in at least some cases of delusion, the failure of hypothesis evaluation resulting from the second factor may take the form of a bias against disconfirmatory evidence is broadly consistent with the earlier proposal (at the end of Section 1) that the second factor has its neural basis in damage to, or hypoactivation of, rDLPFC). In fact, Vocat and colleagues (2013) suggested (citing Coltheart, 2010) that the anosognosia patients' failure to update current beliefs despite the presence of incongruent information "might reflect damage to prefrontal cortical areas" (p. 1778)—and, of course, these patients had only right hemisphere damage. Also, in a study of patients with focal lesions who performed the Wisconsin Card Sorting Task, Gläscher and colleagues (2019) found that lower rates of hypothesis updating in response to disconfirmatory evidence were associated with lesions "located primarily in the right PFC [prefrontal cortex] reaching from dorsolateral PFC to the frontal pole and mostly focused in the underlying white matter" (p. 5). Finally, in an fMRI study of schizophrenia patients—with or without delusions—and healthy control subjects, Lavigne, Menon and Woodward (2020) found that reduced activity in a cognitive evaluation network including rDLPFC, during integration of disconfirmatory evidence in a novel task, was associated with poorer performance on the BADE task (outside the scanner) and with delusions in individuals with schizophrenia.

7. Delusion without Neuropsychological Impairment

The two-factor theory of delusion has mainly been applied to cases of delusion in which the first factor is neuropsychological in nature; and we have seen that there is some support for the proposal that, in such cases, the second factor (that results in failure of hypothesis evaluation) is also neuropsychological. It is, nevertheless, not part of the two-factor theory that one or both of the two factors must be a neuropsychological impairment. The theory allows that observing or encountering an unpredicted phenomenon that does not result from a neuropsychological impairment might prompt a delusional hypothesis, and also that the second factor might not be neuropsychological—for example, it might be motivational. To put this point another way: answers to the two questions which are at the heart of the two-factor theory of delusion (What initially prompted the delusional idea? Why was the delusional idea adopted and maintained as a belief rather than being rejected, as it should have been?) do not inevitably require an appeal to neuropsychology.

There are three examples where the two-factor theory has been applied to the explanation of delusion without appeal to neuropsychology: folie à deux delusion, a particular form of somatic delusion, and alien abduction delusion. We discuss each in turn.

Folie à deux

In folie à deux, a delusional belief of one individual (the "primary") is subsequently adopted by another (the "secondary") via social contact between the two (for a review, see Arnone, Patel & Tan, 2006). Sometimes there is more than one secondary and, typically, the primary and secondary or secondaries are members of the same family. Langdon (2013) and Nielssen, Langdon and Large (2013) have offered an account of folie à deux—specifically, of the secondary's delusion—in terms of the two-factor theory.

What initially prompted the secondary's delusional idea? It did not arise in the secondary's mind endogenously, as a hypothesis generated to explain an unpredicted phenomenon. Rather, it arose exogenously, as an idea communicated to the secondary by the primary. Thus, folie à deux is an example of socially-transmitted delusion. (Koro is another example—see Li, 2010.)

Why did the secondary adopt and maintain this idea as a belief, rather than rejecting it? Langdon (2013) distinguished two kinds of answer to this question. The second factor might be endogenous and relatively permanent—perhaps "a neuropathological vulnerability" (p. 76)—or exogenously imposed and temporary—contingent on the relationship between the secondary and the primary.

Nielssen and colleagues (2013) retrospectively analysed five cases of folie à deux and concluded that all six secondaries were delusional and had a significant impairment of hypothesis evaluation (see 2013, p. 397, Table 2). In three secondaries, this impairment was judged to be explained by neuropathy associated with schizophrenia or schizoaffective disorder. In the other three secondaries, it was at least partly explained in terms of the relationship with a dominant primary (sister, wife or brother), whose beliefs were accepted uncritically. For two of these three secondaries, subsequent separation from the primary resulted in remission of the delusion. For one of these two secondaries, there was "no obvious neuropathological basis" (p. 404) for the impairment of hypothesis evaluation—a particularly clear case of a non-neuropsychological second factor.

A non-neuropsychological somatic delusion

A person described by Coltheart and Langdon (2019) believed that his gums were rotting, even though he had visited oral pathologists on numerous occasions and all had informed him that his gums were normal. What initially prompted his delusional idea?

Coltheart and Langdon (2019) reported that he had not been accepted into University—though other members of his ambitious family had succeeded—and that this had triggered depression and despondency. Perhaps the first possible explanation of his not going to University that came to his mind was that he had been rejected because he lacked certain abilities. If that triggered depression and despondency, he might have set himself to generate an *alternative* explanation. His having an unpleasant physical condition that he did not want other people to see would explain why he was not going to University, but why was it the

rotting gums explanation, specifically, that came to mind? Coltheart and Langdon could only speculate, but the person told them that his gum disease started in his late teens after he had cut his gum while eating. Recollection of such events might have prompted the rotting gums idea.

There was no evidence of any neuropsychological abnormality in this young man. So, why did he adopt and maintain the rotting gums delusional idea as a belief rather than rejecting it? Coltheart and Langdon (2019) suggested a motivational explanation: "his adoption of the belief about his gums allowed him to interpret his not going to University as something that did not imply a personal rejection" (p. 84). So here the failure of hypothesis evaluation occurred because it was motivated by a personal benefit.

We have argued that the failure of hypothesis evaluation resulting from the second factor may take the form of a bias against disconfirmatory evidence (Coltheart & Davies, 2021b; also see Sections 5 and 6, above). Individuals who are strongly motivated to adopt and maintain a particular hypothesis as a belief may establish an exceptionally high threshold for accepting that disconfirmatory evidence falsifies the hypothesis—or even for accepting that the evidence warrants reducing their credence in the hypothesis.

Alien abduction delusion

This is the belief that one has been abducted by extraterrestrial beings and subsequently returned to Earth. What might bring such an idea to mind? A plausible proposal (Holden & French, 2002) is that the idea comes to mind as a result of an experience of sleep paralysis—inability to perform voluntary movements of the trunk and limbs at sleep onset or on waking. Sleep paralysis may be accompanied by hallucinations of three main kinds: (i) a sensed presence and visual, auditory and tactile sensations as if there were an intruder in the room; (ii) breathing difficulties, feelings of pressure on the chest and of suffocation; and (iii) feelings of floating, flying or falling, and out-of-body experiences.

Many people sometimes have these experiences as they wake. For example, Sharpless and Barber (2011) found that 7.6% of the general population, 28.3% of students and 31.9% of psychiatric patients experienced at least one episode of sleep paralysis. But most of these people do not believe that they have been abducted by aliens. Why is it that in a subset of people who have these experiences, the idea "I am being abducted by aliens" comes to mind and is adopted as a belief?

McNally and Clancy (2005) reported that 'abductees' entertained "a wide range of 'New Age' beliefs" (p. 120) and McNally (2012) provided some detail: "belief in foretelling the future/tarot cards (70% [*versus* 8% in the control group]), astrology (60% [*vs* 25%]), ghosts (70% [*vs* 42%]), bioenergetic healing therapies (70% [*vs* 17%]), and alternative/herbal remedies (80% [*vs* 58%])" (p. 7). French and colleagues (2008) reported that people who claimed to have had extraterrestrial contact scored significantly higher than control subjects on two scales measuring beliefs about, and experiences of, paranormal phenomena.

The presence of these unorthodox beliefs may help explain, not only why the alien abduction idea came to mind, but also why it was adopted and maintained as a belief. Acceptance of unorthodox beliefs may indicate "a susceptibility to uncritically accept beliefs"

(Nielssen et al., 2013, p. 399). The literature on alien abduction delusion provides some evidence of reduced cognitive ability to carry out hypothesis evaluation (Cheyne & Pennycook, 2013; French et al., 2008; McNally et al., 2004) and also of strong motivation to maintain the alien abduction belief (Clancy, 2005; McNally, 2012). There is, however, no suggestion of neuropsychological impairment. (For a one-factor account of alien abduction delusion, see Sullivan-Bissett, 2020. We note again that arguments for or against a second factor may depend on how the notion of a 'factor' is understood.)

8. Conclusion

In the two-factor theory of delusion, the first factor explains why a delusional idea or hypothesis came to mind in the first place and the second factor explains why the hypothesis was adopted and maintained as a belief rather than being rejected—as it should have been—on the basis of available evidence and background knowledge that counted against it.

It has proved illuminating to consider the two-factor theory against the background of an eight-step model of the normal pathway from surprising facts to new beliefs—the Peircean pathway model. This has allowed a more substantive account of the associative processes by which a delusional hypothesis is generated. One consequence of the account is that hypothesis generation is a locus of individual differences. The model also provides a structure in which to investigate the cognitive nature of the failure of hypothesis evaluation. One proposal is that this is a bias against disconfirmatory evidence.

The two-factor theory has been applied to cases of monothematic delusion in which the first factor has been a neuropsychological impairment. There is some reason to propose that, in these cases, the second factor is also neuropsychological, with a neural basis in damage to, or hypoactivation of, rDLPFC. It is not, however, part of the two-factor theory that the two factors must be neuropsychological. The delusional hypothesis might be socially transmitted, for example, and the failure of hypothesis evaluation might result from a motivated bias against disconfirmatory evidence. In recent work, the two-factor theory has been applied to provide explanations of folie à deux, a somatic delusion, and alien abduction delusion—without appeal to neuropsychology.

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