Please cite and quote from published version:
***Cognitive Neuropsychiatry*, 27 (2022), 430–446.**

Cotard delusion, emotional experience and depersonalisation

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Abstract

Introduction: Cotard delusion—the delusional belief "I am dead"—is named after the French psychiatrist who first described it: Jules Cotard (1880, 1882). Ramachandran and Blakeslee (1998) proposed that the idea "I am dead" comes to mind when a neuropathological condition has resulted in complete abolition of emotional responsivity to the world. The idea would arise as a putative explanation: if "I am dead" were true, there would be no emotional responsivity to the world.

Methods: We scrutinised the literature on people who expressed the delusional belief "I am dead", looking for data on whether such patients are reported as entirely lacking in emotional responsivity.

Results: In numerous cases, patients with Cotard delusion are described as experiencing emotions including anxiety, fear, guilt, distress, euphoria and worry.

Conclusions: We conclude that complete absence of emotional responsivity cannot be what prompts the delusional idea that one is dead. We propose that, in at least some cases, the idea "I am dead" comes to mind in response to symptoms of depersonalisation or derealisation, often present in cases of Cotard delusion, and give examples of Cotard patients with abnormalities in various neural areas that could be responsible for the presence of such symptoms.

1. Introduction: Cotard delusion

Cotard delusion owes its name to the Parisian neurologist and psychiatrist Jules Cotard (1840–1889) who published the seminal paper on this condition (Cotard, 1882; see also Cotard 1880/1999). He called the condition *Le délire des négations,* but it became widely known as "Cotard's syndrome" when Jules Séglas (1897, pp. 11, 13) adopted that term, following an earlier suggestion by Emil Régis (1893). Cotard's (1882) paper referred to eleven patients, of whom eight were regarded by Cotard as pure cases of the syndrome.

As noted by Berrios and Luque (1995a, p. 218) and Young and Leafhead (1996, p. 149), over the years since Cotard's original work the term "Cotard's syndrome" has become almost synonymous with the delusional belief that one is dead, even though only one of Cotard's eight pure cases had this delusion. The other seven patients exhibited a variety of symptoms, including other delusions, but not the delusion that they were dead. Since Cotard's original paper, however, there have been many published reports of people who expressed the belief that they were dead. It is this specific belief with which our paper is concerned.

These days Cotard delusion is frequently defined as the belief that one is dead (see e.g., Young et al., 1992), and we shall use the term "Cotard delusion" in that way, to refer only to the delusion of death. Thus we focus on this condition as a specific *symptom*, and not as a *syndrome* (a collection of symptoms that may not always co-occur), whilst noting that some have advocated the syndrome approach (e.g., Berrios and Luque, 1995a).

We reject the syndrome approach for the reason given by Young and Leafhead (1996): if there is no symptom common to all of those classified as exhibiting a particular syndrome, then there cannot be a single cause of that syndrome. For example, the term "Cotard delusion" is sometimes used to refer to various kinds of somatic delusion as well ("patients with Cotard delusions, who believe that they are dead or that their body organs have been lost"; Nishio and Mori, 2012, p. 216). However, because some people who express the belief that they are dead do not have any somatic delusions, and some people with somatic delusions do not have the belief that they are dead, the explanation of why patients come to have the belief that they are dead must be different from the explanation of why some patients come to have delusional somatic beliefs. This is so even though there are patients who exhibit both of these beliefs.

More generally, amongst patients who believe that they are dead, there is no other delusional belief that all such patients exhibit. So any proposal as to what causes someone to arrive at the belief that they are dead does not need to address any other delusion, even though patients who believe they are dead often have various other delusions too. That is why we will be concerned here solely with the delusional belief that one is dead.

2. What might cause Cotard delusion?

What could bring to someone's mind the idea that they might be dead? William James (1890) and Brendan Maher (1974) proposed the general theory that delusional beliefs arise as attempts to explain anomalous experiences, and Young and Leafhead (1996) suggested that this is in particular true of Cotard delusion: "Here, we have set out an approach to the Cotard

delusion which treats it as the patient's attempt to make sense of abnormal perceptual experiences in which things seem strange and unfamiliar" (1996, p. 168).[1] This kind of explanation of Cotard delusion was also put forward by Ramachandran and Blakeslee (1998, p. 167) who were explicit about what the particular abnormal experience prompting the delusion was: they proposed that patients with Cotard delusion have suffered a form of neuropathology which has led to "a complete lack of emotional contact with the world". We refer to this as the Ramachandran-Blakeslee Hypothesis (RBH). It has been frequently cited and widely discussed and we have subscribed to it in papers on the two-factor theory of delusional belief such as Coltheart and Davies (2021b).

Suppose that a person no longer experienced any kinds of emotional response. In such circumstances, the idea or hypothesis that one is dead, though bizarre, would be *explanatorily adequate*, since it is true that people who are dead do not experience such emotional reactivity. That is why one can readily imagine that abolished experience of emotional reactivity might prompt the thought "I am dead" as a putative explanation. So it is plausible that abolished experience of emotional reactivity might in some way be causally implicated in Cotard delusion: that is the RBH.

We have not interpreted the RBH as proposing merely that a patient with Cotard delusion continues to experience emotional responses, but at a reduced level by comparison with the level that past experience would lead them to expect. Rather, we have interpreted it as proposing that emotional responding is completely abolished. This is also how Gerrans (2002) interpreted the RBH:

> If Ramachandran is correct, then the Cotard subject has a lack of affective response to *all* perceptual inputs … . Consequently, *nothing* that occurs to her evokes the normal emotional response. (2002, p. 50; emphasis added)

Ramachandran and Blakeslee (1998) acknowledged that there was no extant evidence concerning whether or not there is a complete absence of emotional reactivity in people with Cotard delusion, and hence that the idea that such an impairment is causally implicated in this delusion was speculative. But they did point out that this hypothesis was readily testable:

> [T]hese ideas can be tested in the laboratory. [We] would predict that Cotard's syndrome patients will have a complete loss of [SCR[2]] for all external[3] stimuli … and this leaves them stranded on an island of emotional desolation, as close as anyone can come to experiencing death. (Ramachandran & Blakeslee, 1998, p. 167)

Unfortunately, to the best of our knowledge, there have still been no studies of SCRs to emotional stimuli (or any studies measuring autonomic responsivity with other indices such

[1] It is of some interest here that Cotard (1880/1999) described the delusions of his patient Mlle X as being related to "the experiencing of abnormal bodily sensations known to be common in patients with anxious melancholia: empty-headedness, oppression of the chest, absence of all sensations and of feelings of pleasure, …" (1880/1999, p. 277).

[2] Skin Conductance Response: an index of activity of the autonomic nervous system.

[3] The term "external" is not needed here because emotional responses occur to internal stimuli too, such as remembering a past event that was emotionally arousing. Recall of such events elicits SCRs even though no external stimulus is present.

as pupil size or heart rate) in patients with Cotard delusion, so this explicit prediction from the RBH has never been tested. Because of this, Corlett (2019, p. 167) commented: "in the absence of SCR data from Cotard patients, it remains premature to declare Cotard as having been explained by two-factor theory".[4]

However, SCR measurement is not the only way to investigate emotional reactivity in patients with Cotard delusion. An alternative is to investigate whether individual Cotard patients are ever reported as experiencing any forms of emotion.

3. Cotard delusion and emotional experience

Berrios and Luque (1995a) have argued that Cotard, himself, intended his term "le délire des négations" to refer to a kind of *depressive illness* and that, consequently, "to talk about the delusion of being dead as Cotard's delusion makes little sense, for *délire des négations* also entails the presence of anxiety, *severe depression*, and other attending delusions" (1995a, p. 219; emphasis added). In an analysis of 100 cases of what they refer to as "Cotard's syndrome", Berrios and Luque (1995b) found 89 cases associated with depression and 65 with anxiety. Young and Leafhead (1996) proposed that "the Cotard delusion represents a *depressed* person's attempt to account for abnormal perceptual experiences" (1996, p. 166; emphasis added). On the other hand, Nishio and Mori (2012) described a patient who, following a right-hemisphere stroke (that involved the fronto-temporoparietal region and thalamus), believed that he was dead but did not have "a depressed mood or feelings of fear" (2012, p. 218).

3.1 Emotional responding in patients with Cotard delusion

We are interested in the delusional belief, "I am dead" (which we refer to as "Cotard delusion"), and our question is whether the Cotard idea or hypothesis arises as a putative explanation of the total absence of any experience of emotional responding. Is it the case that, in each individual with Cotard delusion, experience of emotional responding is (or was, at the time of onset) totally abolished? We present seven cases (see also Grover et al., 2014; Ramírez-Bermúdez et al., 2010 (two cases); Freudenreich et al., 2012; Oberndorfer et al., 2017; and **Supplementary Table 1**). We have not assumed any essential connection between Cotard delusion and depression but depression is, indeed, reported in most of the cases that we present below (in cases 1–5).

3.1.1 Emotional responding in patients with Cotard delusion following damage to insular cortex

Case 1 Restrepo-Martínez et al. (2019) studied a 19-year-old patient Mr. A., with psychotic and catatonic symptoms, who had Cotard delusion. His ^{18}F-FDG-PET scan showed marked bilateral metabolic decrease in insular cortex (and in the occipital lobe) and a moderate decrease in anterior cingulate cortex, as well as bilateral hypermetabolism in frontal and medial temporal lobes. He was diagnosed with autoimmune limbic encephalitis. Mr. A expressed the belief that he was dead (and also expressed various delusional beliefs about his bodily organs). He said:

[4] Here Corlett was referring to the theory of delusional belief that we have offered in various papers over the past two decades (the most recent paper being Coltheart and Davies, 2021b).

> I want this to end; my mom got me dead. She sent me some evil spirits ... I do not want that to happen anymore; everything is wrong. I died before coming here.

Patient Mr. A showed no response to tactile and painful stimuli and was indifferent to pain. He had symptoms of depression and his emotional responding was clearly diminished, but it was not abolished. Mr. A was anxious, complained about his body, and would cry, "expressing guilt, anguish, and despair" (2019, p. 472).

Following treatment with electroconvulsive therapy (ECT), Mr. A's frontal and medial temporal lobe metabolism improved, as did his Montreal Cognitive Assessment score (from 15/30 before, to 26/30 after, treatment) and most of his symptoms—including his delusions. His insular cortex hypometabolism was substantially unchanged, however, and two symptoms remained. Mr. A. still had difficulty describing his own emotions (alexithymia) and he had "overvalued somatic symptoms arising from deficient visceral monitoring" (Restrepo-Martínez et al., 2019, p. 478).

Case 2 McKay and Cipolotti (2007) reported a 24-year-old patient, LU, with epilepsy following Herpes simplex encephalitis. She had bilateral insula damage, more severe on the right, and repeatedly stated that she was dead, being adamant that she had died two weeks prior to her clinical assessment. She was assessed as having a moderate level of depression and, despite her insistence that she was dead, her emotional responding was not abolished. She evidently experienced the emotions of distress and anxiety:

> She was extremely distressed and tearful as she related these beliefs [that she was dead and had died two weeks previously], and was very anxious to learn whether or not the hospital she was in was 'heaven'. (2007, p. 353)

Case 3 A 65-year-old patient with dementia and severe depression reported by Chatterjee and Mitra (2015) showed frontotemporal atrophy, particularly involving bilateral insular cortex, on MRI. She claimed that she was dead but her emotional responding was not abolished. She certainly experienced the emotion of fear. Having started to complain that she had cancer in her head,

> she elaborated that her brain was completely rotten now due to the cancer and expressed *extreme fears* for harming family members because of the infectious nature of her disease (2015, p. e52; emphasis added).

She also blamed herself for transmitting the disease to other family members.

3.1.2 Emotional responding in a patient with psychotic depression and Cotard delusion

Case 4 Young and Leafhead (1996) described the case of a 29-year-old patient, JK, who was admitted to hospital multiple times with episodes of psychotic depression (see also Young et al., 1994). When she was most unwell, she believed that she was dead. Patient JK acknowledged that she had experiences of bodily feelings (e.g., she could feel her heartbeat and could feel hot or cold). When it was put to her that these feelings amounted to evidence that she was not dead, but alive, she responded that "since she had such feelings even though she was dead, they clearly did not represent evidence that she was alive" (1996, p. 158).

Young and Leafhead (1996, p. 164) said that "feelings of lack of emotional responsiveness" are a prominent clinical sign in Cotard delusion and, specifically, in their cases. But they also stated that patient JK "felt frightened, confused, and guilty" (1996, p. 157).

> She was *worried* that she had done things for which her mother would get into trouble … . She felt particularly *guilty* about having claimed social security benefits … on the grounds that she was dead while she was claiming. JK was *extremely worried* that she would get into trouble about this. (Young & Leafhead, 1996, p. 157; emphasis added)

Thus, patient JK's emotional responsiveness was far from being abolished.

3.1.3 Emotional responding in patients with Cotard delusion following anti-NMDA-receptor encephalitis

Case 5 In a recent study of eighty-five patients with anti-NMDA-receptor (ANMDAR) encephalitis, Ramírez-Bermúdez et al. (2021) identified two patients with Cotard delusion.

A 37-year-old patient with ANMDAR encephalitis and catatonia received several sessions of plasma exchange therapy. A few days after the final session of plasma exchange, the catatonic symptoms were starting to improve. When asked how he felt, the patient replied, "I do not have feelings because I am dead … All human beings will be dead" and, a few days later, "I am dead, among other human beings. This is like a program where I am dead" (Ramírez-Bermúdez et al., 2021, p. 65).

In this patient, delusions of physical deterioration and paranoia began in the days following the Mexico City earthquake in 2017 and he felt guilty because "*he couldn't provide help when people most needed it*" (2021, p. 65). Given this background, it is of interest that he elaborated the delusional belief that he was dead, claiming to be "a dead person among the corpses under the earthquake debris" (p. 65).

The psychopathological features of this case (2021, p. 66, Table 1, Case 1) included alexithymia and insensitivity to pain, as well as depression, suggesting diminished experience of emotions.[5] They also included anxiety and delusions of guilt, indicating that experience of emotional responding was certainly not abolished. Indeed, depression and anxiety, fear and guilt, are recurrent themes in Cases 1 to 5.

Case 6 Ramírez-Bermúdez et al. (2021) also described a 44-year-old patient who firmly believed that she was dead, began to ask whether her family had attended her funeral, and told her husband to marry another woman. A few weeks later, after a period of recovery from her delirium and catatonia, she again instructed her husband to re-marry as she, herself, was dead, and she asked him whether her family had attended her funeral.

The psychopathological features of this case (2021, p. 66, Table 1, Case 2) did not include depression but did include alexithymia and insensitivity to pain—which would suggest

[5] In their case description, Ramírez-Bermúdez et al. (2021, Case 1) mentioned psychotic and catatonic symptoms (p. 65) and also stated that the patient was in a state of delirium on admission (p. 67). They did not mention depressive symptoms. Nevertheless, Table 1 (2021, p. 66) indicates that this patient had depression.

diminished experience of emotions. They did not include features such as anxiety or delusions of guilt that would indicate experience of emotion. Nevertheless, the authors' description of the patient's state in the days before Cotard delusion emerged is illuminating:

> a manic-like state of euphoria, hyperactivity, disinhibition, pressured speech, hypergraphia, decreased need for sleep, grandiose delusions, and aggressive behavior. (p. 65)

This clearly indicates that the patient's experience of emotional responding was far from being utterly abolished.

3.1.4 Emotional responding in a patient with a right temporo-parietal tumour and Cotard delusion

Case 7 Gonçalves and Tosoni (2016) described a 69-year-old patient who was admitted to hospital after brief episodes of numbness in the left hand and of dizziness and, during the night, developed Cotard delusion. In an interview the next day, she stated:

> P: I think I'm dead (…) it started during the night, like it was some kind of a dream, but this remain until now.
> (…)
> E: Do you rationally think you are dead?
> P: I do. It may not make much sense since I realize I have blood pressure when they measure it but I rationally think I am dead. (2016, p. 35)

The patient showed no signs of depression, nor of any affective disorder. In fact, there is nothing in the report to suggest that her emotional responsiveness was anything other than normal.

Brain MRI revealed a tumour in the right temporo-parietal lobe, consistent with the suggestion that abnormalities of right frontal, temporal or parietal lobes may play a role in some cases of Cotard delusion (Debruyne et al., 2009; Kudlur et al., 2007). The patient's delusion was treated with a corticosteroid to reduce the swelling associated with the tumour and it resolved within two days. She was discharged and referred to the neuro-oncology service.

3.1.5 Summary

We have presented seven cases of Cotard delusion in which patients experienced various emotions including anxiety, fear, guilt, distress, euphoria and worry. (Five additional cases are included in **Supplementary Table 1**.) The original case reports did not provide explicit assessments of the patients' experience of emotional responding but, in each case, there was an adequate basis for inference to the conclusion that experience of emotion was not totally abolished. In Case 7, there was no reason to suppose that the patient's emotional responding was anything other than normal. We do not know of any report of a patient with Cotard delusion in which—in contrast to the cases presented here—it is explicitly stated that the patient's experience of emotional responding was totally abolished.

4. Why does the idea "I am dead" come to mind? Toward an alternative hypothesis

Ramachandran and Blakeslee (1998) proposed that Cotard delusion arises because a form of neuropathology has led to "a complete lack of emotional contact with the world" (p. 167); it is this which leads patients to the idea that they are dead. This account of how the Cotard delusional idea or hypothesis arises has seemed plausible to us, so much so that we have subscribed to it in previous papers we have written on this subject. But detailed scrutiny of case reports reveals that Cotard patients are often described as experiencing various kinds of emotional responses. Thus, it cannot be the case that Cotard delusion is caused by abolition of emotional reactivity; the Ramachandran-Blakeslee Hypothesis (RBH) is not supported. How, then, are we to explain why the idea "I am dead" comes to mind in these patients?

4.1 The two-factor approach

We approach this question from the perspective of the two-factor theory of delusional belief (Coltheart, 2007; Coltheart et al., 2011; Coltheart & Davies, 2021b; Davies et al., 2001). According to that theory, any (monothematic) delusional condition is initially triggered by observation of a Surprising Event (SE), the occurrence of which is a consequence of the hypothesized First Factor that is a neuropsychological impairment or some other abnormality. The resulting delusional idea comes to mind as an attempt to explain this observation—that is, to render the event unsurprising. So, for example, when one observes that encountering one's wife surprisingly fails to generate an autonomic response to the sight of her face, entertaining the idea that one's wife has been replaced by a stranger—Capgras delusion—would explain the absence of autonomic response, since the faces of strangers do not evoke strong autonomic responses.

To explain any delusion in terms of the two-factor theory, one must first identify a SE which would be rendered unsurprising by the delusional belief. We must therefore seek to determine what SE is observed by Cotard patients. This must be an SE which would be unsurprising if the delusional person were dead; that is, if the delusional idea were true, the SE would be "a matter of course" (Peirce, 1903/1998, p. 231). Only then could we argue that this SE is responsible for the idea or hypothesis "I am dead" coming to mind.

According to the two-factor theory, even if the idea or hypothesis "I am dead" comes to mind, this is *not sufficient* for the Cotard delusion—the adoption and maintenance of this hypothesis as a belief, despite the evidence against it—to occur. The delusion requires a Second Factor, an impairment of hypothesis evaluation. (See e.g., Coltheart & Davies, 2021b, for the argument for a second factor, applied to a variety of delusions.)

4.2 An objection averted

An apparently devastating objection might be raised against the suggestion that the idea "I am dead" comes to mind in response to observation of a surprising event (SE). Those who are dead do not have experiences at all and they do not observe any events, whether surprising or routine. Consequently, having an experience or observing an SE—no matter what that experience or SE is—should be sufficient to discredit the idea "I am dead".

This objection is not, in fact, devastating as far as our two-factor theory of delusional belief is concerned. Suppose a patient observes an SE. This observation is Step 1 of our eight-step

Peircean-pathway model of how unexpected observations lead to new beliefs (Coltheart & Davies, 2021a,b; Davies & Coltheart, 2020). Step 2 is an associative process of generating a putatively explanatory hypothesis (Pinker, 2005; Rellihan, 2009). If a generated hypothesis is to be passed on to later steps along the pathway, it must meet the requirement—*sine qua non*—that *if it were true then the surprising event would follow as a matter of course* (Peirce, 1903/1998, p. 231).

There is, of course, evidence against the Cotard hypothesis—as there is against any delusional hypothesis. On many conceptions of what is involved in being dead, the fact that one is observing a surprising event counts strongly against the hypothesis that one is dead. But the steps involved in *testing* a generated hypothesis, and considering evidence against it, are Steps 5 to 8 along the Peircean pathway—the hypothesis evaluation stage of the model.

Hypothesis *generation* and hypothesis *evaluation* are distinct processes. Adoption and maintenance of a hypothesis as a belief despite the evidence against it indicates a failure of hypothesis evaluation. But having a hypothesis come to mind despite there being evidence against it does not indicate a failure of hypothesis generation. Indeed, because the observed event was surprising—not what prior beliefs would have suggested—a hypothesis that meets the Peircean requirement will inevitably depart in some way from prior beliefs.

4.3 Multiple surprising events whose observation might bring to mind the same idea

We do not require that it is the same SE that is involved in all cases of Cotard delusion. We have previously argued that the delusion of mirrored-self misidentification is sometimes generated by the SE resulting when a person with prosopagnosia looks in a mirror, and in other cases is generated by the different SE resulting when a person with mirror agnosia looks in a mirror (Breen et al., 2000, 2001). So we allow that different SEs—which might result from different first factors—may be associated with different cases of Cotard delusion. But all of these SEs must have the property that, if one were dead, the event in question would no longer be surprising: it would follow as a matter of course. Can such SEs be found in patients with Cotard delusion?

5. Cotard delusion and depersonalisation

Consider the following statements made by three (non-Cotard) patients:

(1) I see but I don't feel. I taste but it means nothing to me. … Music usually moves me, but now … it doesn't stir me. I saw Big Ben alight last night, normally a moving sight to me, but it might have been an alarm clock for all I felt.
(Bockner, 1949, p. 969)

(2) Flowers to me have lost their essence, I fail to see them as part of nature. They have become almost synthetic, artificial … . I fail to see the flower in all its authenticity.
(Sierra et al., 2002, p. 530)

(3) I do not feel I have a body. When I look down I see my legs and body but it feels as if it was not there. When I move I see the movements as I move, but I am not there with the movements.

(Sierra, 2009, p. 28)

5.1. Depersonalisation as a possible first factor in Cotard delusion

The three patients who made the statements above were describing symptoms of depersonalisation or derealisation, which *DSM-5* defines and distinguishes as follows:

> Depersonalization: Experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (e.g., perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing).
>
> *and*
>
> Derealization: Experiences of unreality or detachment with respect to surroundings (e.g., individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted).
> (American Psychiatric Association, 2013, p. 302)

We shall use the term *depersonalisation* in an inclusive way to encompass derealisation.

Each of the three patients described a surprising event (SE)—the unexpected absence of something that would normally be present, expected, and even routine. The three SEs are, respectively:

(1) I do not feel normal emotional responses to what I see, hear or taste.
(2) The things that I see do not seem familiar and indisputably real.
(3) I do not have the sense of my body and my actions being my own.

Each of these SEs has the crucial Peircean property that, if "I am dead" were true, the SE would no longer be surprising but would follow as a matter of course. If one were dead then

(1) absence of normal emotional responding;
(2) absence of the sense of familiarity and reality toward objects in the environment; and
(3) absence of the sense of embodiment and agency

would all follow as a matter of course. Thus, we can argue that SEs like these could be responsible for the Cotard delusional idea or hypothesis "I am dead" coming to mind.

Patients with Cotard delusion are often reported to have symptoms of depersonalisation (see e.g., Butler, 2000; Nejad et al., 2013; Wright et al., 1993; Young and Leafhead, 1996; and also the five patients discussed below in Sections 5.2.1–5.2.3). For such patients, it is plausible to suppose that the idea "I am dead" arose in response to observation of one or more SEs similar to those that we described above. Hence we suggest that depersonalisation acts as the first factor in at least some cases of Cotard delusion—that is, it is depersonalisation that brings to mind the idea "I am dead" in these cases. The suggestion that depersonalisation might be a factor in Cotard delusion was, in fact, made by Cotard (1891) himself (see Billon, 2016, p. 372)

and it has received recent attention from philosophers and interdisciplinary researchers (e.g., Billon, 2016; Gerrans, 2015, 2019; Radovic, 2017).

The patients with depersonalisation who made the statements (1) through (3) above did not have Cotard delusion. Thus, even if depersonalisation is a possible first factor in Cotard delusion, its presence is not sufficient for the delusion to occur. As we have said (see above, Section 4.1), according to the two-factor theory, a second factor—specifically, an impairment of hypothesis evaluation—must also be present if the first factor—here, depersonalisation—is to result in the occurrence of the delusion—here, Cotard delusion.

5.2 Symptoms of depersonalisation and their neural bases (in relationship to Cotard delusion)

The three SEs that we described correspond to three (of four) factors revealed by a factor analysis (Sierra et al., 2005) of responses by depersonalisation patients on the *Cambridge Depersonalization Scale* (Sierra & Berrios, 2000), as follows:

> *De-emotivity* (DE)—also known as emotional numbing or de-affectualisation;
> *Derealisation* (DR)—alienation from surroundings; and
> *De-somatisation* (DS)–also known as anomalous body experience, encompassing both loss of the sense of body ownership and loss of the sense of agency

(The fourth factor was anomalous subjective recall—absence of the sense of oneself being a participant in remembered events.)

We now provide examples of these three symptoms of depersonalisation and their neural bases in patients with Cotard delusion.

5.2.1 Symptoms of depersonalisation following right temporo-parietal damage in patients with Cotard delusion

Case A Following brain injury involving right temporo-parietal areas and some bilateral frontal lobe damage, patient WI "complained of a lack of familiarity of things he saw, especially buildings and people's faces" (Young et al., 1992, p. 800). He said that his vision was "like listening to a foreign language" and he had "feelings of unreality and difficulties in deciding whether events around him were real or just imagined" (p. 800). Patient WI showed symptoms of de-emotivity and derealisation and observed SEs such as these:

$(DE)_{WI}$ I do not feel normal emotional responses.
$(DR)_{WI}$ People and buildings do not seem familiar and events in the environment do not seem real.

If "I am dead" were true, these SEs would follow as a matter of course.

Case B A month after a right fronto-temporo-parietal stroke, a patient described by Nishio and Mori (2012) complained about feelings of depersonalisation and derealisation and said, "My death certificate has been registered. You are walking with a dead man" (p. 218). The authors proposed that feelings of derealisation or depersonalisation—such as feelings of

unreality—combined with abnormalities in feelings of familiarity—evident in unusual misidentifications of people and places—to prompt the idea "I am dead".

5.2.2 Symptoms of depersonalisation following damage to insular cortex in patients with Cotard delusion

Case C McKay and Cipolotti (2007) described a patient, LU, with bilateral insula damage (Case 2 in Section 3) who reported feelings of 'strangeness' toward her boyfriend. She showed an internalising attributional style and the authors suggested SEs similar to those in Capgras delusion, with de-emotivity and derealisation restricted to the visual modality and, indeed, to faces:

$(DE)_{LU}$ I do not feel the normal emotional response to my boyfriend.
$(DR)_{LU}$ My boyfriend no longer seems comfortably familiar.

If "I am dead" were true, these events would follow as a matter of course.

Case D Patient Mr. A (Case 1 in Section 3), described by Restrepo-Martínez et al. (2019), showed hypometabolism of insular cortex bilaterally and the authors suggested:

> [A]n interruption between the normal processing of internal and external stimuli in the IC [insular cortex] and other limbic areas could result in abnormal subjective feeling states, that could represent a first factor for the development of nihilistic delusions described by patients with Cotard syndrome. (p. 476)

We conjecture that Mr. A observed SEs of de-emotivity (specifically, alexithymia) and de-somatisation:

$(DE)_{Mr.A}$ I am not able to distinguish and identify my feelings.
$(DS)_{Mr.A}$ My organs and tendons do not feel right and I can no longer feel the blood flowing through my veins (see 2019, pp. 471–472).

If "I am dead" were true, these events would follow as a matter of course.

5.2.3 Symptoms of depersonalisation following ANMDAR encephalitis in a patient with Cotard delusion

Case E Funayama et al. (2018) described a patient with ANMDAR encephalitis, who showed symptoms of depersonalisation—specifically, de-somatisation ("I feel myself detached from my own body", p. 455)—and developed Cotard delusion and extreme somatic hallucinations. The authors proposed that ANMDAR encephalitis may lead to Cotard delusion by causing symptoms of depersonalisation. The patient observed an SE:

(DS) I no longer feel connected to my own body.

If "I am dead" were true, this event would follow as a matter of course.

5.2.4 Summary

We have presented five cases of Cotard delusion in which patients showed symptoms of depersonalisation: de-emotivity, derealisation or de-somatisation. (Four additional cases are included in **Supplementary Table 2**.) In Section 5.1, we argued that patients with symptoms of depersonalisation observe SEs with the crucial Peircean property:

> If "I am dead" were true, SE would follow as a matter of course.

In the cases that we have presented here, it is plausible that the Cotard delusonal idea "I am dead" arose in response to observation of such SEs, so that depersonalisation is the first factor in at least some cases of Cotard delusion.

6. Scope of the proposal and future directions

We propose the following hypothesis:

> In at least some cases of Cotard delusion, it is depersonalisation that brings to mind the idea "I am dead".

This hypothesis differs from the RBH in at least two significant ways. First, it is not a general claim about all patients with Cotard delusion. Second, in cases that do fall within its scope, it does not generate the prediction that all emotional responding will be abolished. De-emotivity is an aspect of depersonalisation but emotional responding is not totally abolished. Motor expressions of emotion, and also some emotional feelings, are preserved. Indeed, paradoxical as it may sound, "the very absence of feelings is frequently identified by patients as their major source of distress" (Sierra, 2009, p. 33).

A second prediction of the RBH was that, in patients with Cotard delusion, "all the sensory areas are disconnected from the limbic system" (Ramachandran & Blakeslee, 1998, p. 167). Limbic disconnection, whether physical or functional, is an important theme in depersonalisation research (Sierra et al. 2002; Sierra, 2009, p. 154). Sierra and Berrios (1998) proposed that the symptoms of depersonalisation result from an inhibitory mechanism that evolved to suppress emotional responding in life-threatening situations. Contrary to the second prediction of the RBH, however, it seems that limbic disconnection, de-emotivity and derealisation may be restricted to visual perception and perhaps even to faces in some cases of Cotard delusion (e.g., McKay and Cipolotti, 2007; Young et al., 1992).

A third strand or prediction of the RBH is that the idea "I am dead" may come to mind when one feels *both* alienation from one's surroundings (as in derealisation) *and* alienation from oneself (that is, loss of the normal sense of self):

> [A patient with Cotard delusion] feels so emotionally remote from the world and from himself that he will actually make the absurd claim that he is dead.
> (Ramachandran & Blakeslee, 1998, p. 248)

Ramírez-Bermúdez et al. (2021) described two patients with ANMDAR encephalitis (Cases 5 and 6 in Section 3), whose ^{18}F-FDG-PET scans showed hypometabolism of areas of medial parietal cortex that make up the posterior part of the default mode network (DMN). The authors commented that damage or hypoactivation in these areas of posterior DMN "could

alter normal feelings of familiarity toward oneself and the environment" (p. 68). It is plausible that the idea "I am dead" sometimes comes to mind in response to a patient's observation of loss of (aspects of) the normal sense of self (e.g., Charland-Verville et al., 2013)—and, of course, such loss would follow as a matter of course if "I am dead" were true. The cases described in this paper do not suggest, however, that loss of the normal sense of self is, or is part of, what prompts the idea "I am dead" in every case of Cotard delusion.

A question of interest is whether there can be an integrated account of how the idea "I am dead" comes to mind that encompasses aspects of depersonalisation and also loss or disruption of one or more of the multiple aspects of the normal sense of self or self-awareness (Millière, 2020). There are some reasons to think that such an account might be given. Dugas (1898/1996, pp. 458, 459) described depersonalisation as a condition in which mental states come to feel *alien to the self*, Sierra and David (2011) described depersonalisation as an *impairment of self-awareness* and other recent commentators have focused their accounts of depersonalisation and Cotard delusion on the sense of self or self-awareness (e.g., Billon, 2016; Gerrans, 2015, 2019; Radovic, 2017).

Even if we could give such an integrated account, we should still not expect that its scope would extend to every case of Cotard delusion. After the second patient described by Ramírez-Bermúdez et al. (2021) had recovered (Case 6 in Section 3), she gave her own account of how, while 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. (p. 67)

The feeling that time is passing unusually slowly is a symptom of depersonalisation that loads on the 'anomalous subjective recall' factor (Sierra et al., 2005; see also Mayer-Gross, 1935, p. 105) but immobility is neither a symptom of depersonalisation nor the loss of an aspect of the normal sense of self. Not being able to talk or move is, nevertheless, a surprising event that would follow as a matter of course if "I am dead" were true. Thus, catatonia resulting in immobility may be an alternative first factor in cases of Cotard delusion not involving depersonalisation.

7. Conclusion

The RBH account of Cotard delusion does not succeed because Cotard patients do not exhibit the complete lack of emotional reactivity postulated by this hypothesis. We have proposed instead that depersonalisation serves as the first factor in at least some cases of Cotard delusion because this would plausibly result in observation of a surprising event (SE) with the property:

If "I am dead" were true, SE would follow as a matter of course

and hence evoke the idea "I am dead".

And we have, further, given examples of Cotard patients with damage or hypometabolism in various neural areas that could be responsible for the presence of symptoms of depersonalisation in these patients.

Acknowledgements
We are grateful to the editor and two anonymous reviewers for their constructive comments on an earlier version of this paper.

Disclosure statement
No potential conflict of interest was reported by the authors.

Funding
The authors reported there is no funding associated with the work featured in this article.

Data availability statement
There is no data set associated with this article.

References

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. Washington, DC: American Psychiatric Association.

Berrios, G. E., & Luque, R. (1995a). Cotard's delusion or syndrome?: A conceptual history. *Comprehensive Psychiatry*, *36*, 218–223.

Berrios, G. E., & Luque, R. (1995b). Cotard's syndrome: Analysis of 100 cases. *Acta Psychiatrica Scandinavica*, *91*,185–188.

Billon, A. (2016). Making sense of the Cotard syndrome: Insights from the study of depersonalisation. *Mind & Language*, *31*, 356–391.

Bockner, S. (1949). The depersonalization syndrome: Report of a case. *Journal of Mental Science*, *95*, 968–971.

Breen, N., Caine, D., Coltheart, M., Roberts, C., & Hendy, J. (2000). Towards an understanding of delusions of misidentification: Four case studies. *Mind & Language, 15*, 74–110.

Breen, N., Caine, D., & Coltheart, M. (2001). Mirrored-self misidentification: Two cases of focal onset dementia. *Neurocase*, *7*, 239–254.

Butler, P. V. (2000). Diurnal variation in Cotard's syndrome (copresent with Capgras delusion) following traumatic brain injury. *Australian and New Zealand Journal of Psychiatry*, *34*, 684–687.

Charland-Verville, V., Bruno, M-A., Bahri, M. A., Demertzi, A., Desseilles, M., Chatelle, C., Vanhaudenhuyse, A., Hustinx, R., Bernard, C., Tshibanda, L., Laureys, S., & Zeman, A. (2013). Brain dead yet mind alive: A positron emission tomography case study of brain metabolism in Cotard's syndrome. *Cortex*, *49*, 1997–1999.

Chatterjee, S. S., & Mitra, S. (2015). "I do not exist" – Cotard syndrome in insular cortex atrophy. *Biological Psychiatry*, *77*, e52–e53.

Coltheart, M. (2007). Cognitive neuropsychiatry and delusional belief. *Quarterly Journal of Experimental Psychology, 60,* 1041–1062.

Coltheart, M., and Davies, M. (2021a). How unexpected observations lead to new beliefs: A Peircean pathway. *Consciousness and Cognition*, *87*, 103037, 1–13.

Coltheart, M., & Davies, M. (2021b). Failure of hypothesis evaluation as a factor in delusional belief. *Cognitive Neuropsychiatry, 26*, 213–230*.*

Coltheart, M., Langdon, R., & McKay, R. (2011). Delusional belief. *Annual Review of Psychology*, *62*, 271–298.

Corlett, P.R. (2019). Factor one, familiarity and frontal cortex: A challenge to the two-factor theory of delusions. *Cognitive Neuropsychiatry, 24,* 165–177.

Cotard, J. (1880/1999). Du délire hypocondriaque dans une forme grave de mélancolie anxieuse. *Annales Médico-Psychologiques*, *4*, 168–74 (translated by G. E. Berrios (1999) as 'On hypochondriacal delusions in a severe form of anxious melancholia', *History of Psychiatry*, *10*, 274–278).

Cotard, J. (1882). Du délire des négations. *Archives de Neurologie, 4*, 152–170, 282–295.

Cotard, J. (1891). *Études sur les Maladies Cérébrales et Mentales*. Paris: J-B. Baillère.

Davies, M. & Coltheart, M. (2020). A Peircean pathway from surprising facts to new beliefs. *Transactions of the Charles S. Peirce Society*, *56*, 400–426.

Davies, M., Coltheart, M., Langdon, R. & Breen, N. (2001). Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry & Psychology, 8,* 133–158.

Debruyne, H., Portzky, M., Van den Eynde, F., & Audenaert, K. (2009). Cotard's syndrome: A review. *Current Psychiatry Reports*, *11*, 197–202.

Dugas, L. (1898/1996). Un cas de dépersonnalisation. *Revue Philosophique de la France et de l'Étranger*, *45*, 500–507 (translated by M. Sierra and G. E. Berrios (1996) as 'A case of depersonalization', *History of Psychiatry*, *7*, 455–461).

Freudenreich, O., Basgoz, N., Fernandez-Robles, C., Larvie, M., & Misdraji, J. (2012). Case 5-2012: A 39-year-old man with a recent diagnosis of HIV infection and acute psychosis. *New England Journal of Medicine*, *366*, 648–57.

Funayama, M., Takata, T., & Mimura, M. (2018). Cotard's syndrome in anti-N-methyl-D-aspartate receptor encephalitis. *Psychiatry and Clinical Neurosciences*, *72*, 455–456.

Gerrans, P. (2002). A one-stage explanation of the Cotard delusion. *Philosophy, Psychiatry, & Psychology*, *9*, 47–53.

Gerrans, P. (2015). All the self we need. In T. Metzinger & J. M. Windt (Eds.), *Open MIND* (15) (pp. 1–19). Frankfurt am Main: MIND Group.

Gerrans, P. (2019). Depersonalization disorder, affective processing and predictive coding. *Review of Philosophy and Psychology*, *10*, 401–418.

Gonçalves, L. M., & Tosoni, A. (2016). Sudden onset of Cotard's syndrome as a clinical sign of brain tumor. *Archives of Clinical Psychiatry*, *43*, 35–36.

Grover, S., Aneja, J., Mahajan, S., & Varma, S. (2014). Cotard's syndrome: Two case reports and a brief review of literature. *Journal of Neurosciences in Rural Practice*, *5*, S59–S62.

James, W. (1890). *The Principles of Psychology* (in two volumes). New York, NY: Henry Holt and Company.

Kudlur, S. N. C., George, S., & Jaimon, M. (2007). An overview of the neurological correlates of Cotard syndrome. *European Journal of Psychiatry*, *21*, 99–116.

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

Mayer-Gross, W. (1935). On depersonalization. *British Journal of Medical Psychology*, *15*, 103–122.

McKay, R., & Cipolotti, L. (2007). Attributional style in a case of Cotard delusion. *Consciousness and Cognition*, *16*, 349–359.

Millière, R. (2020). The varieties of selflessness. *Philosophy and the Mind Sciences*, *1*(I), 8, 1–41.

Nejad, A. G., Anari, A. M. Z., & Pouya, F. (2013). Effect of cultural themes on forming Cotard's syndrome: Reporting a case of Cotard's syndrome with depersonalization and out of body experience symptoms. *Iranian Journal of Psychiatry and Behavioral Sciences*, *7*, 91–93.

Nishio, Y., & Mori, E. (2012). Delusions of death in a patient with right hemisphere infarction. *Cognitive and Behavioral Neurology*, *25,* 216–223.

Oberndorfer, R., Schönauer, C., Eichbauer, H., Klaushofer, K., & Friedrich, F. (2017). Cotard syndrome in hypoactive delirium: A case report. *Psychiatrica Danubina*, *29*, 500–502.

Peirce, C. S. (1903/1998). Harvard Lectures on Pragmatism (1903), Lecture 7: Pragmatism as the logic of abduction. In Peirce Edition Project (Eds.), *The Essential Peirce, Volume 2 (1893–1913)* (pp. 226–241). Bloomington: Indiana University Press.

Pinker, S. (2005). So how *does* the mind work? *Mind & Language, 20,* 1–24.

Radovic, F. (2017). The sense of death and non-existence in nihilistic delusions. *Phenomenology and the Cognitive Sciences*, *16*, 679–699.

Ramachandran, V. S., & Blakeslee, S. (1998). *Phantoms in the Brain: Human Nature and the Architecture of the Mind*. London: Fourth Estate.

Ramírez-Bermúdez, J., Aguilar-Venegas, L. C., Crail-Melendez, D., Espinola-Nadurille, M., Nente, F., & Mendez, M. F. (2010). Cotard syndrome in neurological and psychiatric patients. *Journal of Neuropsychiatry and Clinical Neurosciences*, *22*, 409–416.

Ramírez-Bermúdez, J., Bustamante-Gomez, P., Espinola-Nadurille, M., Kerik, N. E., Dias Meneses, I. E., Restrepo-Martínez, M., & Mendez, M. F. (2021). Cotard syndrome in anti-NMDAR encephalitis: Two patients and insights from molecular imaging. *Neurocase*, *27*, 64–71.

Régis, E. (1893). Note historique et clinique sur le délire des négations. *Gazette Médicale de Paris*, *2*, 61–64.

Rellihan, M. J. (2009). Fodor's riddle of abduction. *Philosophical Studies: An International Journal for Philosophy in the Analytic Tradition, 144*, 313–338.

Restrepo-Martínez, M., Espinola-Nadurille, M., Bayliss, L., Díaz-Meneses, I., Kerik, N.E. Mendez, M. and Ramírez-Bermúdez, J. (2019). FDG-PET in Cotard syndrome before and after treatment: Can functional brain imaging support a two-factor hypothesis of nihilistic delusions? *Cognitive Neuropsychiatry, 24,* 470–480.

Séglas, J. (1897). *Le Délire des Négations: Séméiologie et Diagnostic*. Paris, France: Masson, Gauthier-Villars.

Sierra, M. (2009). *Depersonalization: A New Look at a Neglected Syndrome*. Cambridge: Cambridge University Press.

Sierra, M. Baker, D., Medford, N., & David, A. S. (2005). Unpacking the depersonalization syndrome: An exploratory factor analysis on the Cambridge Depersonalization Scale. *Psychological Medicine*, *35*, 1523–1532.

Sierra, M. & Berrios, G. E. (1998). Depersonalization: Neurobiological perspectives. *Biological Psychiatry*, *44*, 898–908.

Sierra, M. & Berrios, G. E. (2000). The Cambridge Depersonalisation Scale: A new instrument for the measurement of depersonalisation. *Psychiatry Research*, *93*, 153–164.

Sierra, M. & David, A. S. (2011). Depersonalization: A selective impairment of self-awareness. *Consciousness and Cognition*, *20*, 99–108.

Sierra, M., Lopera, F., Lambert, M. V., Phillips, M. L., & David, A. S. (2002). Separating depersonalisation and derealisation: The relevance of the 'lesion method'. *Journal of Neurology, Neurosurgery and Psychiatry*, *72*, 530–532.

Wright, S., Young, A. W., & Hellawell, D. J. (1993). Sequential Cotard and Capgras delusions. *British Journal of Clinical Psychology*, *32*, 345–349.

Young, A. W., & Leafhead, K. M. (1996). Betwixt life and death: Case studies of the Cotard delusion. In P. W. Halligan & J. C. Marshall (Eds.), *Method in Madness: Case Studies in Cognitive Neuropsychiatry* (pp. 147–171). Hove, E. Sussex: Lawrence Erlbaum Associates.

Young, A. W., Leafhead, K. M., & Szulecka, T. K. (1994). The Capgras and Cotard delusions. *Psychopathology*, *27*, 226–231.

Young, A. W., Robertson, I. H., Hellawell, D. J., de Pauw, K. W. and Pentland, B. (1992). Cotard delusion after brain injury. *Psychological Medicine, 22,* 799–804.