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Attributional style in a case of Cotard delusion

Ryan McKay [a,b,c,*], Lisa Cipolotti [a]

[a] Department of Neuropsychology, The National Hospital for Neurology and Neurosurgery, London, UK
[b] The Center for Cognitive Studies, 111 Miner Hall, Tufts University, Medford, MA 02155, USA
[c] Department of Psychology, Charles Sturt University, Bathurst, Australia

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Abstract

Young and colleagues (e.g. 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*. Mahway, NJ: Lawrence Erlbaum Associates.) have suggested that cases of the Cotard delusion (the belief that one is *dead*) result when a particular perceptual anomaly (caused by a disruption to the affective component of visual recognition) occurs in the context of an internalising attributional style. This hypothesis has not previously been tested directly. We report here an investigation of attributional style in a 24-year-old woman with Cotard delusion (''LU''). LU's attributional style (and that of ten healthy control participants) was assessed using the Internal, Personal and Situational Attributions Questionnaire (Kinderman, P., & Bentall, R. P. (1996). A new measure of causal locus: the internal, personal and situational attributions questionnaire. *Personality and Individual Differences, 20*(2), 261–264.). LU showed a significantly greater proportion of internalising attributions than the control group, both overall and for negative events specifically. The results obtained thus support an association of Cotard delusion with an internalising attributional style, and are therefore consistent with the account of Young and colleagues. The potential brain basis of Cotard delusion is discussed.

Keywords: Attributional style; Cotard delusion; Case study

1. Introduction

Few pathologies of the self are as profound and striking as those reported in cases of Cotard syndrome, which can involve the belief that one is *dead*. The assertions of some patients with this delusion come close to violating the famous Cartesian dictum *cogito ergo sum*. Descartes explored the limits of radical scepticism and concluded that whereas one could certainly doubt the evidence of one's senses, it was not possible to doubt one's existence. Yet some Cotard patients maintain that they are dead or that they do not exist (Young & Leafhead, 1996).

* Corresponding author. Present address: Department of Psychology, Charles Sturt University, Panorama Avenue, Bathurst, NSW 2795, Australia. Fax: +1 617 627 3952.
E-mail address: ryantmckay@mac.com (R. McKay).

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The classic reports of this condition were published by the psychiatrist Jules Cotard (e.g. Cotard, 1882), who described a clinical state that he termed *délire des négations*. The French eponym *délire de Cotard* was later adopted, and translated into English as Cotard's syndrome (Berrios & Luque, 1995a). Although this latter designation is often identified with the belief that one is dead, Cotard himself did not regard that belief as an essential defining feature of the condition he described (Berrios & Luque, 1995b; Young & Leafhead, 1996). Young and Leafhead's analysis of Cotard's (1882) cases revealed a series of commonly occurring features and symptoms, including self-deprecatory delusions, suicidal ideation,[1] feelings of guilt, and denial of body parts. Young and Leafhead's subsequent comparison of three patients with the belief that they had in fact died revealed a consistent combination of additional symptoms including depressed mood, abnormal feelings, depersonalisation and derealisation, and evidence of face-processing impairments. More exotic concurrent symptoms have been reported elsewhere, including hydrophobia (Nejad, 2002) and lycanthropy (Nejad & Toofani, 2005). The issue of whether the Cotard phenomenon is best conceptualised as a psychiatric symptom or a discrete syndrome is yet to be resolved (Silva, Leong, Weinstock, & Gonzales, 2000; Young & Leafhead, 1996). For present purposes we shall equate the term "Cotard delusion" with the belief that one is dead.

According to Gardner-Thorpe and Pearn (2004), the Cotard delusion usually presents in the context of schizophrenia or bipolar disorder, although it may also occur subsequent to organic insult. Temporo-parietal lesions of the non-dominant hemisphere are particularly implicated in cases of Cotard delusion associated with cerebral trauma. A patient described by Young, Robertson, Hellawell, de Pauw, and Pentland (1992) provides an example of this presentation. For months following a motorcycle accident, the patient was convinced that he was dead. Computerised tomography (CT) scans showed contusions affecting temporo-parietal areas of the right hemisphere as well as some bilateral damage to the frontal lobe.

A variety of authors have suggested that the most comprehensive explanation of monothematic delusions such as the Cotard delusion will implicate contributing factors at two levels—the experiential and the inferential (see for e.g. Davies, Coltheart, Langdon, & Breen, 2001; Ellis & Young, 1990; McKay, Langdon, & Coltheart, 2005b; Wright, Young, & Hellawell, 1993; Young & de Pauw, 2002; Young, Leafhead, & Szulecka, 1994; c.f. Gerrans, 2000, 2002). Such two-factor explanations incorporate an *empiricist* perspective on delusion formation (Campbell, 2001), in that they implicate unusual perceptual experiences (caused by a spectrum of neuropsychological abnormalities) as a first factor in delusion formation (see also Maher, 1992, 1999; Maher & Ross, 1984). Anomalous perceptual experiences are not thought to be sufficient for the development of delusions, however, because there exist individuals with such experiences who do not develop delusory beliefs about them (Langdon & Coltheart, 2000). Two-factor models thus invoke an additional explanatory factor or set of factors, to account for how perceptual anomalies lead to the adoption of delusional beliefs.

Young and colleagues (e.g. Wright et al., 1993; Young, 2000; Young & Leafhead, 1996; Young et al., 1994) have proposed that the Cotard delusion results from a similar anomalous perceptual experience to that putatively involved in the Capgras delusion (see Ellis, Whitley, & Luaute, 1994). Patients with the Capgras delusion believe that a loved one has been replaced by a physically identical impostor. Two independent studies have demonstrated that Capgras patients fail to show the normal pattern of autonomic discrimination (as indexed by skin-conductance response) between familiar and unfamiliar faces (Ellis, Young, Quayle, & de Pauw, 1997; Hirstein & Ramachandran, 1997). Whereas control participants showed significantly greater skin-conductance responses to familiar faces, for Capgras patients familiar and unfamiliar faces engendered skin-conductance responses of equivalent magnitude.

On the basis of such results, Capgras patients are thought to have damage to neural pathways underpinning the emotional component of face recognition (Ellis & Young, 1990; Langdon & Coltheart, 2000; Stone & Young, 1997). The ensuing discordance between experiences of the way someone "looks" and the way they

[1] Readers may rightly wonder at the paradox inherent in a person entertaining thoughts of suicide while simultaneously believing that they are dead. Such paradoxes are not uncommon where delusions are concerned (see, for example, Breen, Caine, Coltheart, Hendy, & Roberts, 2000; Brett-Jones, Garety, & Hemsley, 1987), and in fact their existence constitutes a key objection to what is known as the "doxastic conception" of delusions, i.e. the idea that delusions are beliefs. The fact that deluded individuals often seem unconcerned by manifest contradictions between their delusions and their other beliefs (Bayne & Pacherie, 2005) seems at variance with the idea that our beliefs are integrated in a "web of belief" (Quine & Ullian, 1970). Some commentators (e.g. Currie & Jureidini, 2001) have therefore argued that delusions are not actually beliefs at all.

"feel" is thought to underpin the impostor delusions of these patients. The spirit of this formulation dates back to the original paper by Capgras and Reboul-Lachaux (see Ellis et al., 1994), and later to Derombies, who "suggested that the syndrome results from simultaneous intellectual recognition and affectively engendered non-recognition of faces". (1935, cited in Enoch & Trethowan, 1991; p. 13).

Following Cotard himself (see Young & Leafhead, 1996), Young and colleagues have suggested that disruptions to the affective component of visual recognition may occur in Cotard cases as well as in Capgras cases. However, whereas Capgras patients interpret the resultant experiences in accordance with a paranoid, projective attributional style (see Kinderman & Bentall, 1997; Lyon, Kaney, & Bentall, 1994), Cotard patients interpret them in accordance with a depressive, introjective attributional style (see Peterson et al., 1982; Seligman, Abramson, Semmel, & von Baeyer, 1979). In other words, whereas Capgras patients make an external attribution ("that woman is not my wife but rather a physically identical impostor"), Cotard patients attribute the cause of the anomalous experiences to themselves ("that woman looks like my wife but doesn't 'feel' like her—it must be because I'm dead"). Young and colleagues thus hypothesise that cases of Cotard delusion result when a particular perceptual anomaly (caused by a disruption to the affective component of visual recognition) occurs in the context of an internalising attributional style. This hypothesis has yet to be tested directly (Fig. 1).

An alternative, *single factor* proposal is that whereas Capgras patients have a circumscribed disconnection syndrome, involving disruption to pathways underpinning the emotional component of *face* recognition, Cotard patients have a more global disconnection of *all* sensory areas from the limbic system, "leading to a complete lack of emotional contact with the world" (Ramachandran & Blakeslee, 1998; p. 167; see also Gerrans, 2000, 2002). Under this latter proposal one would *not* expect Cotard patients to display a marked internalising attributional style (Fig. 2).

We report here an investigation of attributional style in a 24-year-old woman with Cotard delusion. The results obtained support an association of Cotard delusion with an internalising attributional style, and are thus consistent with the account of Young and colleagues.

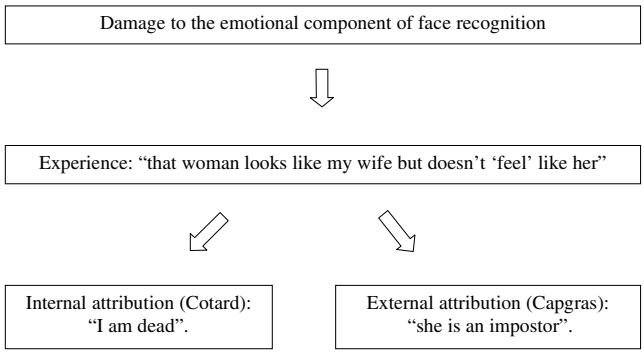

Fig. 1. The Young et al. explanation of the Cotard and Capgras delusions.

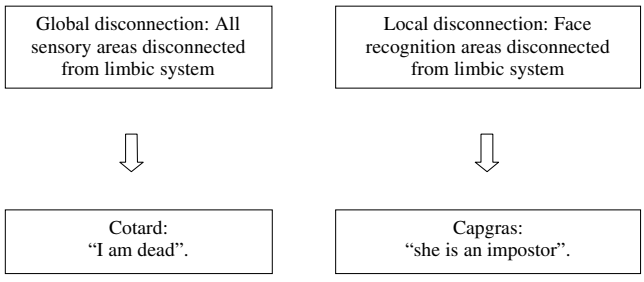

Fig. 2. The Ramachandran and Blakeslee explanation of the Cotard and Capgras delusions.

2. Methods

2.1. Case description

2.1.1. Background

LU, a 24-year-old secretary, was admitted on 19/11/2004 to the Acute Brain Injury Unit of the National Hospital for Neurology and Neurosurgery in Queen Square, London. She was admitted in status epilepticus with recurrent tonic-clonic and partial epileptic seizures. Her diagnosis was ultimately confirmed as Epilepsy following Herpes simplex encephalitis and she was discharged a month after admission, although her seizures continued intermittently in the subsequent months. At the time of admission, LU was living in London with her boyfriend. Her parents were in the United States where they had been living for three years.

2.1.2. Imaging results

A Magnetic Resonance Imaging (MRI) scan revealed an abnormal T2 high signal in the insula, claustrum and adjacent white matter which extended down to the temporal stem on the right side. Similar but less severe changes were seen in the left insular cortex. These features were noted as consistent with a viral encephalitis.

2.1.3. Neuropsychological assessment

Two weeks post-admission, the first author assessed LU in the Neuropsychology Department of the National Hospital for Neurology and Neurosurgery (see Table 1 for test results). LU's neuropsychological profile was characterised by impaired attention and concentration functions and impaired judgement (documented in the context of significant distress). In addition, there was evidence of impaired recognition memory for faces in the context of intact recognition memory for words and for topographical visual information. This latter finding is entirely consistent with three cases of Cotard delusion reviewed by Young and Leafhead (1996): WI (Young et al., 1992), JK (Young et al., 1994) and KH (Wright et al., 1993). Like LU, all three of these patients were selectively impaired on the Warrington (1984) Recognition Memory Test for faces (see Table 2 for comparison of scores).

Table 1
LU's performance on neuropsychological tests

	Raw score	Percentile
Advanced progressive matrices	6/12	18th%ile
WAIS-R digit span	4	Scaled Score—2
Cognitive estimates	11.5	<1st %ile
Weigl sorting test	Pass	
Object decision	18/20	>5% cut-off.
Recognition memory test–Words	46/50	25–50th%ile
Topographical memory test	24/30	25th%ile
Recognition memory test–Faces	32/50	<5th%ile

Table 2
Comparison of LU's performance on Warrington's (1984) Recognition Memory Test with the performance of three patients reviewed by Young and Leafhead (1996)

	Faces		Words	
	Accuracy	z	Accuracy	z
LU	*32*	*3.29****	46	0.02
WI	28	4.41***		
JK	35	2.45**	45	0.21
KH	33	2.99**	44	0.40

Accuracy score out of maximum possible of 50 correct, z = number of SDs below control mean; asterisked scores are significantly below the control mean: $^{**}z > 2.33$, $p < 0.01$; $^{***}z > 3.10$, $p < 0.001$.

2.1.4. Delusional presentation

At neuropsychological assessment LU presented with the Cotard delusion. She repeatedly stated that she was dead and was adamant that she had died two weeks prior to the assessment (i.e. around the time of her admission on 19/11/2004). She was extremely distressed and tearful as she related these beliefs, and was very anxious to learn whether or not the hospital she was in, was "heaven". When asked how she thought she had died, LU replied "I don't know how. Now I know that I had a flu and came here on 19th November. Maybe I died of the flu." Interestingly, LU also reported that she felt "a bit strange towards my boyfriend. I cannot kiss him, it feels strange—although I know that he loves me." Other presenting symptoms included reported sensations of dizziness, as well as musical hallucinosis (hallucinations of disco music), tactile hallucinations (a feeling of running water on her left forearm) and visual hallucinations (moving walls).

LU's conviction that she had died appeared to diminish over the next few days. During this time her beliefs showed some degree of susceptibility to cognitive restructuring. In particular, two days after neuropsychological assessment LU responded to a number of general questions about death, after which she revised her degree of conviction that she had died from an initial 100% down to 40%. In this conversation LU was asked whether she had ever seen a dead person before, and if so how she had known that the person was dead. LU responded that after her grandmother's death she had viewed her grandmother, and that she knew her grandmother was dead because her eyes were closed and she was motionless. LU acknowledged that the fact that she herself was moving and talking was inconsistent with the typical characteristics of dead people, and she subsequently expressed some uncertainty about her beliefs. Within a week of the initial neuropsychological assessment, her delusion appeared to have completely resolved.

2.2. Experimental investigation

The following questionnaires were administered to LU the day after the initial neuropsychological assessment was conducted. LU continued to maintain that she was dead at this time.

2.2.1. Attributional style

The Internal, Personal and Situational Attributions Questionnaire (IPSAQ: Kinderman & Bentall, 1996) comprises 32 statements, each of which describes a hypothetical social event of either positive or negative valence (e.g., "A neighbour invited you in for a drink"; "A friend ignored you"). Respondents are instructed to vividly imagine each event and to write down the one most likely cause of each situation. Respondents are then required to categorise each cause as to whether it is primarily something about themselves (internal attribution), something about another person or group of people (external–personal attribution), or something about the situation (external–situational attribution). The questionnaire is scored by summing the number of internal, external–personal, and external–situational attributions for positive and negative events separately. Kinderman and Bentall (1996) report satisfactory internal reliability for the IPSAQ.

In view of the aims of the present study, two attributional bias indices (hereafter "IPSAQ indices") were computed for each participant. First, an *internalising bias (IB) index* was calculated by dividing the number of internal attributions (across both positive and negative events) by the total number of attributions made. This index thus represents the proportion of a participant's overall attributions that are internal. Second, an *internalising bias for negative events (IBN) index* was calculated by dividing the number of internal attributions for negative events by the total number of attributions made for negative events. This index thus represents the proportion of a participant's attributions for negative events that are internal.

2.2.2. Depression

Levels of depression were assessed using the depression subscale of the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995). The DASS is a 42-item self-report instrument designed to measure the emotional states of depression, anxiety and stress. Each of the three DASS scales contains 14 items. Participants rate the extent to which they have experienced each state *over the past week* using 4-point severity/frequency scales. Scores for Depression, Anxiety and Stress are calculated by summing the scores for relevant items, and fall into one of five severity-rating ranges (Normal, Mild, Moderate, Severe and Extremely Severe).

The DASS has demonstrated internal consistency and concurrent validity in the acceptable-to-excellent ranges (Antony, Bieling, Cox, Enns, & Swinson, 1998).

2.2.3. Control participants

LU's scores on the IPSAQ and the DASS were compared with those of a sample of control participants, comprising ten psychiatrically healthy female participants with a mean age of 26.7 years ($SD = 6.0$). Control participants were drawn from a larger mixed-sex sample ($n = 19$), recruited from the general population for a different study (see McKay, Langdon, & Coltheart, 2005a). As this larger sample was significantly older than LU (mean age = 35.89 years, $SD = 11.71$, $t[18] = 4.43$, $p < .001$), the 10 youngest female participants were selected to comprise an age- and gender-matched control sample for the current investigation. There was no significant difference in age between patient LU and this control sample, $t(9) = 1.42$, $p = 0.189$.

Control participants had been screened using the Affective and Psychotic Screening Modules of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Williams, & Gibbon, 1997). Exclusion criteria for the control sample had included any history of serious head injury and/or central nervous system disease, current substance abuse (as per DSM-IV criteria: all participants had been administered the substance use disorders screening module of the SCID-I), previous persistent substance abuse (having met DSM-IV criteria for more than two of the past five years) and fewer than eight years of formal education.

3. Results

The two IPSAQ indices and the DASS depression score are shown in Table 3 for LU and for the control group. In each case LU's score was compared to the control group using Crawford and Howell's (1998) modified *t*-test (one-tailed). LU was found to have a greater internalising bias than the control participants, in that she showed a significantly greater proportion of internalising attributions, both overall and for negative events specifically, than the control group. She also reported a higher level of depression than the control group, although the difference was only borderline-significant.

In view of the established connection between depression and internalising attributions on the one hand (Peterson et al., 1982; Seligman et al., 1979; Sweeney, Anderson, & Bailey, 1986), and depression and Cotard's syndrome on the other (Berrios & Luque, 1995a; Enoch & Trethowan, 1991; Young & Leafhead, 1996), it is possible that both LU's nihilistic delusions and her evident internalising bias are consequences of her depression. However, it should be noted here that her DASS depression score places her in only the Moderate range for depression. Moreover, the self-reported depression of one of the ten control participants was greater than LU's, yet this participant was clearly not delusional and her internalising indices were comparable to the control group mean (in fact numerically lower; IB = .38, IBN = .31).

4. Discussion

This is the first report to document an internalising attributional bias in a case of Cotard delusion. Our Cotard patient, LU, showed a significantly greater internalising bias, both overall and for negative events specifically, than a group of age- and gender-matched control participants. LU also had greater self-reported depression than the control group, although she scored in only the Moderate range for self-reported depression and was in fact less depressed than one of the (non-delusional) control participants. The difference between her depression score and that of the control group was only borderline-significant.

Table 3
IPSAQ indices and DASS depression scores for patient LU and the healthy control participants (ranges in parentheses)

	Patient LU	Control means (ranges)	Statistics
IPSAQ indices			
IB	0.69	0.42 (0.28–0.59)	$t[9] = 2.67$, $p = .013$
IBN	0.88	0.39 (0.19–0.75)	$t[9] = 2.92$, $p = .009$
DASS depression	20	7.90 (0–21)	$t[9] = 1.76$, $p = .056$

The results obtained are consistent with the account of Young and colleagues, who have suggested that the Cotard delusion may arise when anomalous perceptual experiences, resulting from neurological disruption to the emotional component of visual recognition, interact with an internalising attributional style. Although we were unable to formally investigate evidence of anomalous perceptual experience in the present study, LU's self-reported feelings of "strangeness" regarding her boyfriend are strikingly consistent with the anomalous experience thought to underpin cases of the Cotard delusion (namely, a disjunction between experience of the way a loved one "looks" and the way they "feel"). Moreover, neuropsychological testing revealed selective impairments in LU's processing of faces, which Young and Leafhead documented in all three Cotard cases they reviewed and which they suggested leads to "a consequent lack of familiarity of seen things" (Young & Leafhead, 1996, p. 164).

Young and colleagues have suggested that cases of the Cotard and Capgras delusions may stem from fundamentally similar perceptual anomalies. The two groups of patients are said to explain these anomalies in distinct ways because of contrasting attributional tendencies (internalising for Cotard and externalising for Capgras). A competing proposal (Ramachandran & Blakeslee, 1998; see also Gerrans, 2000, 2002) disregards the influence of attributional style, and holds that the Cotard and Capgras delusions differ only in the circumscription of their perceptual anomalies. Thus whereas Capgras delusion involves disruption to pathways underpinning the emotional component of *face* recognition, Cotard patients have a more global disconnection of *all sensory areas* from the limbic system. Under this competing proposal one would not expect the Capgras and Cotard delusions to be associated respectively with externalising and internalising attributional biases.[2]

The present results support the hypothesis that an internalising attributional bias is a factor in the aetiology of Cotard delusion. Future research, utilising a formal measure of attributional style, is now needed to directly test the hypothesis that *Capgras* delusion is associated with an *externalising* attributional bias. Future studies employing measures of autonomic arousal (e.g. skin-conductance) might also profitably investigate whether Cotard patients do in fact show autonomic arousal deficits, and if so whether these deficits are restricted to facial stimuli or whether they involve a more global loss of autonomic responsiveness to *all* stimuli (as predicted by Ramachandran & Blakeslee, 1998).

4.1. The brain basis of Cotard delusion

We shall conclude our discussion by considering the brain areas likely to be involved in cases of the Cotard delusion. Let us first examine this issue with respect to the two interacting contributory factors posited by Young and colleagues.

4.1.1. Factor One—Anomalous perceptual experiences stemming from neurological disruption to the emotional component of visual face recognition

Young and colleagues have suggested that in cases of the Cotard and Capgras delusions, overt recognition of familiar faces is relatively preserved, whereas autonomic responses to such faces are compromised. This idea incorporates the earlier proposal of Bauer (1984, 1986) that overt and autonomic indices of face recognition are underpinned by separate neural systems. Bauer posited two pathways between the visual system and the limbic system, a 'ventral' pathway involving ventromedial occipitotemporal cortex (subserving overt facial recognition) and a 'dorsal' pathway through the superior temporal sulcus and the inferior parietal lobule (underpinning covert recognition). The implication of Young et al's ideas is that in cases of the Cotard delusion, the dorsal route is damaged or disconnected, while the ventral route remains intact. Young (2000) notes, however, that the neurological specifics of Bauer's proposal have been questioned by others in the field (see e.g. Hirstein

[2] Note, however, that the association we report between Cotard delusion and an internalising attributional bias is not, strictly speaking, *in*consistent with the competing proposal. It's simply that this association is not a *prediction* of that proposal. It may be that aspects of *both* proposals are correct, in which case an internalising bias may be part of the aetiological picture of Cotard delusion insofar as it nuances the causal explanation adopted for the anomalous experience; the nature of that experience itself, however, may extend well beyond the emotional component of face recognition. In this connection, we note that our own Cotard patient was beset by a bewildering array of sensory symptoms, and thus an anomalous experience of familiar faces may not have been the only significant experiential component as far as her delusion was concerned.

& Ramachandran, 1997; Tranel, Damasio, & Damasio, 1995). Breen, Caine, and Coltheart (2000) provide an authoritative review of these issues, and criticise Bauer's formulation on the grounds that there is actually no evidence that the dorsal visual-limbic pathway is capable of visual recognition of faces. Breen et al. propose instead a single neuroanatomical face processing route, involving only the ventral visual pathway. These authors argue that the affective component of face recognition is contributed by ventral limbic structures, in particular the amygdala. Cases of Capgras (and, by Young et al's extension, *Cotard*) delusion would then involve a neuroanatomical disruption either in the connection between the inferotemporal lobes (subserving face matching and recognition) and the amygdala, or in the amygdala itself.

4.1.2. Factor Two—Internalising attributional tendencies

Using fMRI, Blackwood et al. (2000) found that internal attributions for events involve activation of the left precentral gyrus and the left middle temporal gyrus. Insofar as such attributions are involved in the aetiology of the Cotard delusion, therefore, activation of these brain regions is implicated.

In considering the brain basis of Cotard delusion, it may be instructive to locate this condition in the context of other, related disorders of belief. Feinberg and Keenan (2004, 2005) review a host of what they call "Clinical disorders of the self... conditions that alter the relationship between the individual and their body as seen directly or in a mirror, or their personal relationship to significant persons, places or objects in their environment" (2005, p. 665). Interestingly, these authors do not touch on Cotard delusion, although they distinguish several variants of Capgras syndrome, including—in addition to the classic syndrome for persons—Capgras for places, for parts of one's body, and for one's mirror image.[3]

As an aside, it is not clear that the mirror condition described by Feinberg and Keenan (2004)[4] is correctly characterised as a species of Capgras delusion. After all, their patient, 'Susan', referred to her reflection as "the 'other' Susan" (Feinberg & Keenan, 2004; p. 53), and evidently did not believe that she had been *replaced* by that 'other' (see Breen et al., 2000). It may be, therefore, that this is more properly a case of delusional *reduplication*. An interesting comparison can be made with two cases reported by Breen, Caine, and Coltheart (2001), both of whom misidentified their reflection as a stranger.[5] As with Feinberg and Keenan's (2004) case, the fact that the reflections in these cases were not thought of as replacements for the deluded individuals suggests that they are not properly described as instances of the Capgras phenomenon.

More generally, we feel that Feinberg and Keenan's (2005) conception of what constitutes a "disorder of the self" may be overly broad. After all, the Capgras and Frégoli syndromes (at least as classically conceived) involve beliefs that are explicitly about *other* individuals.[6] Such conditions certainly affect the self insofar as they alter one's relationship to other people, but that seems to us to be too inclusive a criterion. There are other conditions that concern the self much more directly, such as reverse intermetamorphosis (the belief that one has transformed, physically and/or psychologically, into another person; Breen et al., 2000; Hanin, Perlow, Ben-Daniel, & Itzhaki, 1994; Silva & Leong, 1996), or depersonalization disorder (the persistent feeling that one is detached from one's body; see Simeon & Hollander, 1993). In any case, it seems clear that if the Capgras and Frégoli delusions count as perturbations of the self, then Cotard delusion must as well. Let us turn, then, to a brief consideration of the brain regions implicated in Feinberg and Keenan's (2005) "disorders of the self", for the light that this may shed on the brain regions likely to be involved in Cotard delusion.

Feinberg, DeLuca, Giacino, Roane, and Solms (2005) have recently reviewed a series of 27 previously published cases of such disorders. Consistent with earlier reports (e.g. Signer, 1994; Stuss, Picton, & Alexander, 2001), they found a strong association between such conditions and damage to right-frontal regions of the brain. This association was manifested in both a statistically significant right-hemispheric bias across the frontal, temporal and parietal lobes, and in a significant bias for frontal lobe incidents across both hemispheres.

[3] Note that this is not an exhaustive taxonomy of Capgras cases. For instance, cases of Capgras delusion have also been reported for non-human animals (Reid, Young, & Hellawell, 1993; Rosler, Holder, & Seifritz, 2001; Somerfield, 1999) as well as for inanimate objects (Abed & Fewtrell, 1990; Anderson, 1988; Anderson & Williams, 1994; Castillo & Berman, 1994; Ellis et al., 1996; Feinberg, 2001; Nejad & Toofani, 2006).

[4] The original report of the case they describe can be found in Feinberg and Shapiro (1989).

[5] As did a further case reported by Feinberg (2001).

[6] Frégoli syndrome involves the belief that a stranger is really a familiar person in disguise (Courbon & Fail, 1927).

These findings are complemented by the results of some recent neuroimaging studies. Keenan and colleagues (Keenan, Gallup, & Falk, 2003; Keenan, McCutcheon, & Pascual-Leone, 2001) found activation of right-frontal regions in response to presentation of a photograph of the participant's face (see also Platek, Keenan, Gallup, & Mohamed, 2004; Sugiura et al., 2000), whereas Nakamura et al. (2001) found that self-*voices* also activate right-frontal regions. Craik et al. (1999) found specific right-frontal activations when participants rated trait adjectives for self-relevance (compared with other judgements about trait adjectives).

On the basis of these clinical and neuroimaging findings, Feinberg and Keenan (2005) posit a crucial role of the right hemisphere, and in particular the right-frontal region, "in establishing the appropriate relationship between the self and the world" (p. 675). Given the severity of the self-disturbance involved in Cotard delusion, one would expect disruption to such regions to also figure in the aetiology of this condition. Certainly this is consistent with the presentation of Young et al.'s (1992) patient (described above). With regard to our own patient LU, although the nature of her disease prevented a good localization of her anatomical lesions, it was notable that her performance was impaired on a task thought to be sensitive to frontal lobe dysfunction (Cognitive Estimates; Shallice & Evans, 1978; see Blair & Cipolotti, 2000; Leng & Parkin, 1988; Smith & Milner, 1988). Future reports of Cotard delusion occurring subsequent to organic insult will serve to clarify how well Feinberg and Keenan's neurological picture fits this condition more generally.

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