Capgras delusion: a window on face recognition

Hadyn D. Ellis and Michael B. Lewis

Capgras delusion is the belief that significant others have been replaced by impostors, robots or aliens. Although it usually occurs within a psychiatric illness, it can also be the result of brain injury or other obviously organic disorder. In contrast to patients with prosopagnosia, who cannot consciously recognize previously familiar faces but display autonomic or covert recognition (measured by skin conductance responses), people with Capgras delusion do not show differential autonomic activity to familiar compared with unknown faces. This challenges traditional models of the way faces are identified and presents some epistemological questions concerning identity. New data also indicate that, contrary to previous evidence, covert recognition can be fractionated into autonomic and behavioural/cognitive types, which is consistent with a recently proposed modification of the modal face recognition model.

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Delusions, by their very nature, are bizarre – none more so than the Capgras delusion, one of the most interesting disorders of misidentification. Capgras delusion is characterized by the firm and sometimes dangerous belief that some people are no longer who they were: instead they have been replaced by doubles, impostors, robots, aliens and so forth[1–3] (see Box 1). This particular delusion provides a good opportunity to explore the efficacy of the relatively new discipline of cognitive neuropsychiatry, which attempts not only to explain various psychiatric signs and symptoms within models of normal cognitive functioning but which also uses data that do not fit these models to encourage their modification[4].

A recent well-publicized UK court case of Capgras delusion involved a teacher named Alan Davies who, following a car crash, developed the belief that his wife, Christine, had died in the incident and that the woman living with him was an impostor, someone with whom he is now uncomfortable. He still insists that his real wife died in the accident and he successfully sued the driver of the other vehicle for the distress caused. In court a consultant psychiatrist explained that Mr Davies was suffering from Capgras delusion[5].

Capgras delusion most usually occurs within a psychiatric setting, accompanying a diagnosis of paranoid schizophrenia; but it can also result from neurological, toxic or other organic conditions[3,6], which makes traditional, psychodynamic explanations for the delusion difficult to sustain[7,8]. Moreover, the beliefs of duplication normally described in relation to other people have also been observed for objects[6,9]. In this case, the sufferer insists that tools, ornaments and other household objects have been replaced by near exact doubles.

It is not usual for face and object Capgras delusion to co-exist – suggesting the domain specificity of the disorder. In this review we shall confine our discussion to the face-related form of Capgras delusion. In particular, we intend to review recent work exploring its implications for our understanding of normal face-recognition processes. The essential paradox is that patients with Capgras delusion simultaneously recognize a face and, at the same time, deny its authenticity. We will try to show that this phenomenon is not simply some bizarre and inexplicable belief but that it can provide us with a fascinating clue as to the very nature of normal face recognition and thus, if not radically challenge, then at least require modification to some of the received models of face processing (Box 2). Before getting to that point, however, it is necessary first to discuss an indirectly-related neurological disorder, prosopagnosia, which is the profound loss of face recognition following, usually, a right inferotemporal lesion[10,11].

Prosopagnosia

Although prosopagnosia means the complete inability to recognize previously familiar faces (voice recognition remains, however) work by Bauer and, subsequently, many others has revealed that some patients with the disorder might reveal covert signs of recognition. Both autonomic (SCR, skin conductance response, see Box 3)[12–14] and CNS (event-related potentials)[15] measures have been used to demonstrate the fact that, despite the absence of overt, conscious face recognition, at some level patients with prosopagnosia can discriminate the faces of people they know (i.e. they show the normal elevated SCRs to previously familiar faces compared with faces never encountered before the brain damage that caused the prosopagnosia). Covert face recognition has also been revealed using behavioural measures, such as priming and interference (Box 4).

Prosopagnosia and Capgras delusion

Ellis and Young suggested that, despite their obvious differences, there might be a link between prosopagnosia and Capgras delusion[16]. They posited the idea that the two conditions might be mirror images of one-another as depicted in Fig. 1. According to this hypothesis, if prosopagnosia is the result of damage to the system responsible for generating conscious face recognition, sometimes leaving an unconscious or

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Box 1. The Capgras delusion

Following 19th century reports in Germany by Kahlbaum[a], Capgras and Reboul-Lachaux[b,c] described 'Mme M.', who displayed a florid set of psychotic symptoms, including the belief that her husband, children, neighbours and others had been replaced by doubles. The doubles themselves were replaced by other doubles (80 times in the case of her husband). Usually, however, patients with the delusion report that people emotionally close to them have been substituted. Often they hold this belief with frightening conviction, even being prepared to kill the 'impostor'[d].

Capgras delusion occurs in a variety of settings[e] as a symptom of idiopathic psychiatric illness (e.g. schizophrenia or mood disorders) or of disorders characterized by cerebral dysfunction secondary to structural brain damage or toxic-metabolic conditions. It seems parsimonious to seek a common explanation for the delusion, regardless of its aetiology.

Cases have been described where the delusion that pets[f,g] or even inanimate objects[h,i] have been replaced by replicas. Rarely, some patients have been described with both Capgras delusion for faces and objects/places[j]. These cases illustrate the suggested link between Capgras delusion and reduplicative paramnesia[k].

The anomalous experiences that underlie Capgras delusion, however, might not be a sufficient condition to produce the firmly-held beliefs of substitution. It may be necessary to invoke damage to a second-stage at which attributions are made[l].

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covert mechanism intact, then Capgras delusion might arise when the reverse occurs, that is, an intact overt system, coupled with a malfunctioning covert system.

If, as suggested by Ellis and Young[16], Capgras delusion results from an intact overt face recognition system coupled with the absence of confirmatory input from the system or systems that underlie covert face recognition, then, as they argued,

> 'When patients find themselves in such a conflict (that is, receiving some information which indicates the face in front of them belongs to X, but not receiving confirmation of this), they may adopt some sort of rationalization strategy in which the individual before them is deemed to be an impostor, a dummy, a robot, or whatever extant technology may suggest.' (Ref. 16, p. 244.)

Ellis and Young pointed out that, from their hypothesis, one clear prediction can be made:

> '…Capgras patients will not show the normally appropriate skin conductance responses to familiar faces, despite the fact that these will be overtly recognized.' (p. 244.)

Tests of the autonomic hypothesis

Two studies have been published aimed at testing the prediction that patients with Capgras delusion will not show differential SCRs to familiar and unfamiliar faces[17,18]. The study by Ellis *et al.* was made on five psychiatric patients with Capgras delusion, and their data compared with five matched psychiatric controls and five normal controls. Figure 2a summarizes the results: as predicted, unlike the other two groups, the Capgras patients showed no differential SCRs to familiar faces. It should be noted that the absolute level of SCRs for Capgras patients was low and Fig. 2a shows range-corrected scores. Figure 2b reveals that the SCRs to a repeated loud tone was the same for those with the Capgras delusion and the normal controls, suggesting that the face result cannot be attributed simply to a more general abnormality in autonomic responses to external stimuli.

In the second study, Hirstein and Ramachandran[18] examined a man who, following a car accident, developed the belief that his parents had been replaced by impostors. Again, this patient's SCRs to familiar faces (some personally known to him) were no different from those elicited by unfamiliar faces. Interestingly, the patient seemed to have no delusions about his parents when listening to their voices over the telephone: thus, his Capgras delusion would appear to be a strictly face-related phenomenon.

Theoretical implications

Bauer[12] suggested that the occurrence of autonomic face discrimination in the absence of overt recognition implies that there are two distinct and independent routes to recognition: one (the identification detector),

Box 2. The modal model of face processing

Cognitive models of face processing have undergone many stages of evolution since the basic sequential, modal model was first described[a]. Although earlier box-and-arrow diagrams[b] (see Fig. I) were useful in describing many findings concerning face processing, more recent, neural-network implementations of such models have allowed more explicit predictions to be generated[c–e].

Regardless of how these models are described, they all have in common a single route from face recognition units (FRUs), through person identity nodes (PINs) to the semantic information units (SIUs). The FRUs are activated when a familiar face is recognized. The PINs act as a multimodal gateway linking information from different domains about the same person. The SIUs are a store of attributes that are potentially known about the famous person.

Many of these models predict that lesions to the connections between layers will lead to a graded degradation of information processing[d–f]. A certain degree of lesion, for example, might mean that the model is incapable of retrieving semantic information associated with an activated FRU. If this lesioning is limited, however, some degraded signal may find its way through and have an implicit effect on the network. Such a signal, for example, might lead to easier relearning of semantic information or it might make recognition of information from other domains (e.g. a name) easier. In this way, neural-network models of a modal face processing system predict that a lesioned system will still show evidence of limited face processing in spite of an inability explicitly to recognize a face. Such models, therefore, have been useful in the understanding of prosopagnosia.

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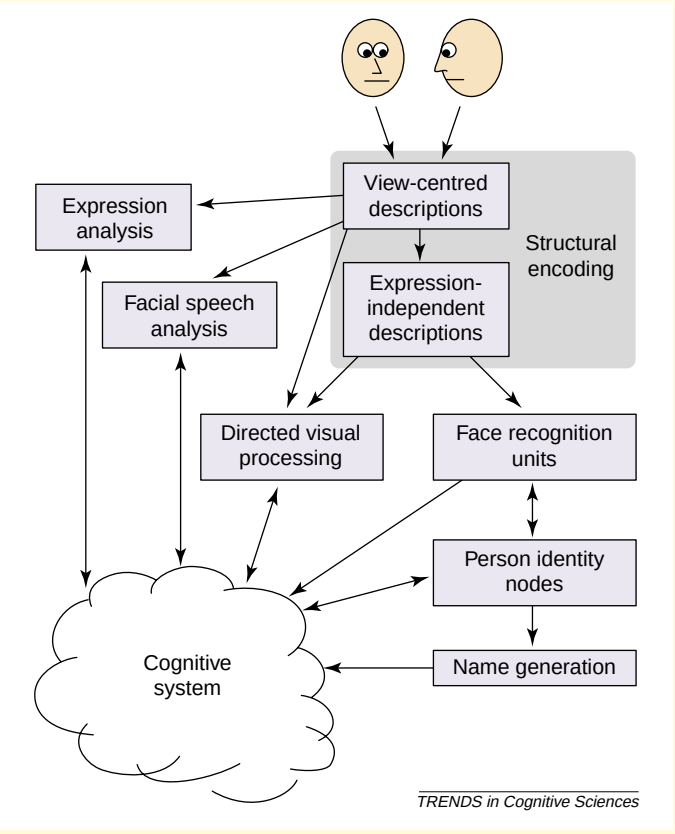

Fig. I. A box-and-arrow version of the modal model of face processing as derived from Bruce and Young[b]. Information regarding the identity of the face is extracted by mechanisms situated along a single route (shown in blue), which is parallel to those for dealing with the expression of the face and picture codes of the image.

termed the ventral route, involving structures along the longitudinal fasciculus between visual cortex and limbic system; and the other, the dorsal route (capable of detecting the 'significance' of a face) passing from visual cortex through the superior temporal sulcus, inferior parietal lobe and cingulate gyrus to the limbic system (primarily the amygdala).

This model, which is illustrated in Fig. 3, is inconsistent with the modal, single route model of face recognition shown in Box 2. Moreover, the discovery that overt face recognition can occur with the apparent lack of covert recognition (i.e. Capgras delusion) fuels the need to challenge the modal model. At the very least, some modification of the modal model is required. The question is do we need to posit two distinct face recognition systems, one processing identity information and the other extracting the emotional significance or, perhaps, familiarity of faces?

Modified dual-route model

Breen *et al.* have recently attempted to answer this question[19]. First, they criticized Bauer's neuroanatomical argument for the dorsal route possessing any structures capable of either face recognition or of producing an affective response to familiar stimuli (though this ignores the possible role in identification of the superior temporal sulcus[20]). As they point out the dorsal pathway is well known to subserve visually guided actions rather than object recognition[21,22]. Breen *et al.* argue instead that face recognition is only conducted by structures situated along the ventral route and that, subsequent to recognition, affective responses to faces are provided by ventral limbic structures, especially the amygdala. Thus, they can explain both the prosopagnosia results and the Capgras delusion data by their model, which, essentially, is a modified dual-route account of face processing that involves core recognition stages followed

Box 3. Autonomic covert recognition: skin conductance responses

The use of skin conductance responses (SCRs) has been a valuable tool in identifying autonomic responses to stimuli. The technique, as pioneered by Venables and colleagues[a,b], was standardized by a committee report[c] allowing for its unambiguous use in many domains including face recognition[d]. Recently, increased SCRs have been found to correlate not only with activity in the amygdala but also in a range of neuroanatomical structures including the right fusiform gyrus[e], an area that has be implicated in face processing.

The SCR method involves recording the changes in the electrical conductivity of a person's skin on his or her hand as the amount of sweat within the eccrine sweat ducts varies[f] (Fig. I). The person, therefore, does not have to be sweating overtly in order for there to be changes in his or her skin conductance. Heightened arousal will lead to more sweat in the sweat ducts even without sweating taking place. As sweat is a good electrical conductor then the higher up the sweat ducts (and, therefore, closer to the skin surface), the greater the person's skin conductance will be.

The SCRs are recorded by placing a small voltage (typically 1.22 V) across the subject using two Ag/AgCl electrodes placed on the underside of two fingers of the same hand. It is possible, therefore, by measuring the electric current to calculate the resistance of the skin (skin conductivity is the inverse of the skin's resistance). The person's SCRs can be plotted as a continuous trace throughout an experiment (Fig. II).

SCRs have been investigated following presentation of faces[d] and it has been established that a peak SCR occurs between 1 and 5 seconds after presentation of a face. Further, the amplitude of the peak following a familiar face is considerably larger than that following an unfamiliar face. This SCR differential occurs even when the faces cannot be overtly recognized[g]. The differential effect has also been repeated for prosopagnosic patients[h]. The conclusion is, therefore, that the SCR differential is a method of determining autonomic covert recognition of faces.

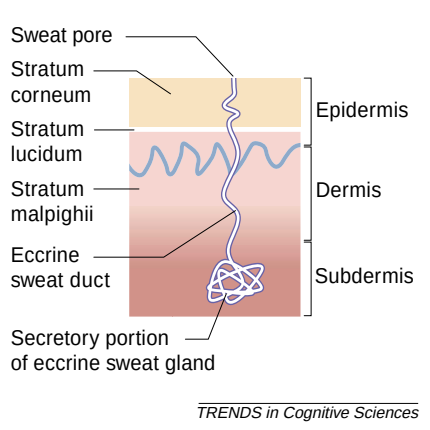

Fig. I. An eccrine sweat gland and duct. Sweat rising up the duct acts to raise the conductivity of the person's skin. Adapted from Ref. f.

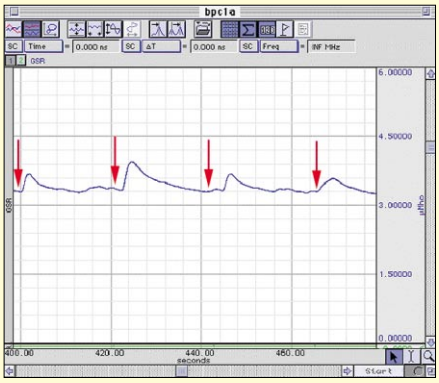

Fig. II. A typical skin conductance trace. The red arrows indicate the presentation of a face to the subject. The skin conductance is plotted as the blue line. The skin conductance response for each face is the size of the peak that occurs between 1 and 5 seconds after presentation.

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by parallel identification and affective-response stages. The proposed identification of the latter with centres within the amygdala echoes that made by others[21].

Although the Breen *et al.* model provides an interpretation for lack of SCR differentiation observed with Capgras patients, it, like earlier accounts, does not describe how the failure to receive a confirmatory feeling of familiarity to a face becomes a delusion[23]. As described earlier, it has been proposed that a second facility must be impaired. That facility being one that compares the conclusions of the two routes of processing. The location of this facility is presented in an adapted version of the Breen *et al.* model in Fig. 4.

How many kinds of covert face recognition?

Covert face recognition can be shown using a variety of techniques (see Boxes 3 and 4). But do these manifestations have the same locus? The model shown in Fig. 3 implies that there are at least two loci: one (probably the person identity node stage) producing behavioural/cognitive forms of covert recognition; and the other, in the affective-response system, causing autonomic covert recognition.

The patient, LF, in Bauer's original demonstration of autonomic covert face recognition was later tested for signs of behavioural/cognitive covert recognition[24]. He showed this category of covert recognition, too; which led to the inference that the two kinds of covert responses might be governed by the same mechanism. This does not accord with the model of Breen *et al.*[19] Figure 4 demonstrates that autonomic covert recognition can be dissociated from overt recognition (because of the dual routes), but it is not necessarily the case that behavioural/cognitive covert

Box 4. Cognitive/behavioural covert recognition

Covert recognition of faces can be shown either by measures of accuracy or response latency. Such covert recognition may be referred to as cognitive or behavioural covert recognition and is possibly separate from the autonomic covert recognition described in Box 3[a].

Evidence of cognitive/behavioural covert recognition in prosopagnosic patients has been identified in a variety of tasks[b,c]. Some of these tasks are described below:

Name re-learning

A famous face that cannot be overtly recognized together with two names (one correct, one incorrect) are presented to a prosopagnosic patient who is required to learn to associate one of the names with the face. For half the faces, the correct name must be associated, and for the other half an incorrect name is provided. For example, a prosopagnosic patient, PH, remembered more of the correct name-face pairings than the incorrect name–face pairings. This demonstrates that there is some saving in his face processing that makes relearning of a name easier than learning a new name.

Face interference

In this task a face and a name are presented at the same time and the subject is required to make a rapid category decision about the named person (e.g. politician or not). The accompanying face might be from the same category, a different category or an unfamiliar person. Normal subjects are fastest when the name and face are from the same category and slowest when they are from different categories. This shows how the face can either facilitate or interfere with a task that only requires attention to the name. It has been found that PH (and other prosopagnosic patients) also shows the same pattern of results with facilitation and inhibition in spite of not being able to recognize the presented faces. This interference, it is assumed, must come from covert recognition.

Face-name priming

This task is similar to the face interference task but the name follows the presentation of a face. The face might be of the same famous person, a different famous person or an unfamiliar person. The subject's task is to make a speeded response as to whether or not the name is of a famous person. It has been found with normal subjects and some prosopagnosics that reaction times to the name following the face of the same person are faster (priming takes place) than when the name follows an unfamiliar face. For the prosopagnosics, of course, this priming is further evidence of covert recognition.

Of the three tasks described here, the first one requires a failure to recognize a face overtly and so does not offer a useful method of investigating covert recognition where overt recognition can be established. The face interference task and the face-name priming task can be employed where overt recognition is also taking place. These two tasks, therefore, have been used to establish the presence of cognitive/behavioural covert recognition for a Capgras patient who can recognize faces overtly[a].

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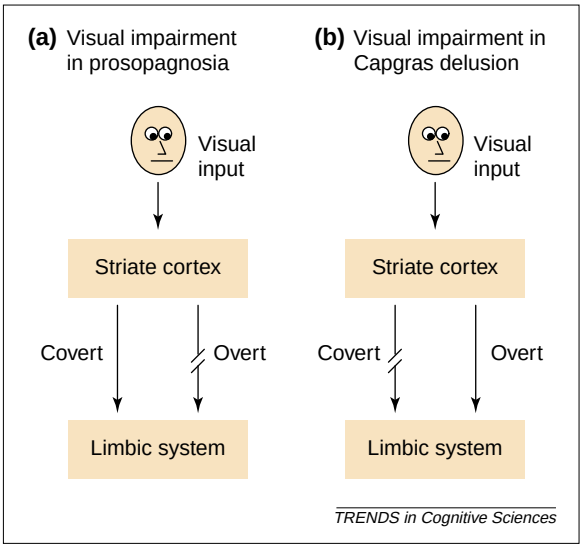

Fig. 1. Capgras delusion as a mirror of prosopagnosia. On the model that there are two routes to visual recognition, one covert and one overt, prosopagnosia represents an interruption in the overt route to recognition (a), whereas Capgras delusion represents an interruption to the covert route (b). Adapted from Ref. 16.

recognition is dissociated from overt recognition. There is evidence that a lesioned single-route neural network might be unable to demonstrate overt recognition yet still display covert recognition that is analogous to behavioural/cognitive covert recognition[25,26]. Such evidence means that it is possible that behavioural/cognitive covert recognition is a result of processing via the overt recognition route and so is separable from autonomic covert recognition.

This model, with two loci of covert recognition, can accommodate the data from LF. He could possess enough processing at the face-recognition unit (FRU) level to initiate autonomic activity signalling recognition. Partial interruption between the FRU and personal identity node (PIN, see Box 2) would explain why he is unable to recognize faces but still shows priming and interference from faces. But how can we tackle the question as to whether one or more loci are involved in producing different types of covert face recognition?

Fractionating covert face recognition

If there is a single locus for covert face recognition phenomena then one might expect that those with Capgras delusion will not only fail to show SCR discrimination to familiar and unfamiliar faces but will also fail to evince behavioural/cognitive forms of covert recognition, such as priming and interference effects. A single locus of covert recognition would mean that interruption of autonomic covert recognition would also imply interruption of behavioural/cognitive covert recognition.

A recent attempt to test this was made with a psychiatric patient, BP, with Capgras delusion[27]. She was administered a standard SCR test and also two traditional tests of behavioural/cognitive covert

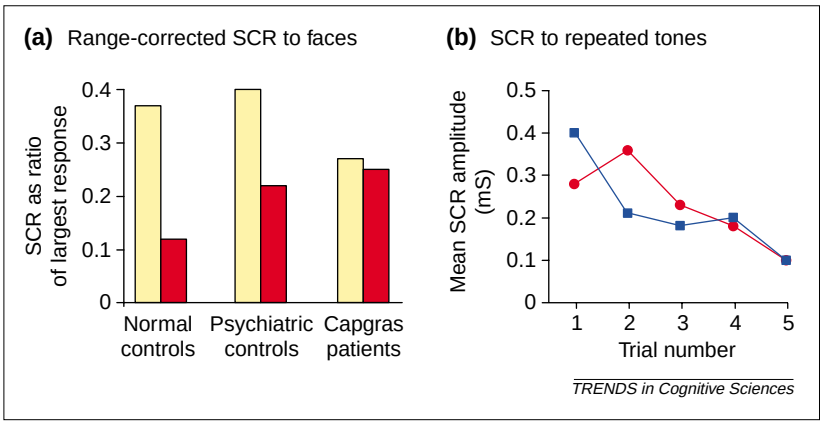

Fig. 2. (a) Mean skin conductance responses (SCRs) to familiar faces (yellow bars) and unfamiliar faces (red bars) for three groups of subjects. The normal subjects and non-delusional psychiatric controls show larger responses to familiar compared with unfamiliar faces. Capgras patients do not show this differential response. (b) Comparison of SCRs to a repeated tone. This demonstrates that the lack of a familiar face differential for Capgras patients cannot be attributed to a general lack of SCR responsivity. Both Capgras patients (blue symbols) and normal controls (red) show a pattern of habituation as the tone is repeated. Data redrawn from Ellis *et al.*[17]

recognition. As expected, she showed no autonomic covert recognition; but her performance on the behavioural/cognitive tests was perfectly normal – indicating a dissociation between the two types of covert face recognition.

This result is entirely consistent with the model of Breen *et al.*, and is at variance with the idea of a single locus for different kinds of covert face recognition. BP has an intact overt face recognition system, which presumably, also allows her to reveal behavioural/cognitive forms of covert recognition; but the link to the affective responses systems is compromised. Thus, covert face recognition can be fractionated, which has obvious ramifications for modelling normal face recognition – not least because, in addition to the two mechanisms described here, there could be others, each governing a different type of covert recognition[25,26].

The double dissociation between autonomic recognition and overt recognition of faces seen between prosopagnosics and Capgras patients means that it is no longer possible to interpret face recognition as proceeding in a strictly sequential fashion along a single route. The modal model of face recognition, and, indeed all models, other than that of Breen *et al.*, need to be adapted to accommodate findings from patients with Capgras delusion. How this can be done, and what further predictions can be generated from such models remain to be seen.

Figure 4 includes what we believe to be an important modification to the Breen *et al.* model. Outputs from the face recognition system and the consequent affective response to which it gives rise must at some point become re-integrated so that they can provide the necessary data for the person to be identified by comparing the joint information representing recognition and affective response against a stored (and therefore expected) representation[26]. Whether this is carried out in a separate centre or is integral to the face processing system is debatable. We, however, take the view expressed in Fig. 4 that the affective response system and the personal information must each feed into an integrative device. Such a device would then compare the expected affective response with the actual affective response and some kind of attribution

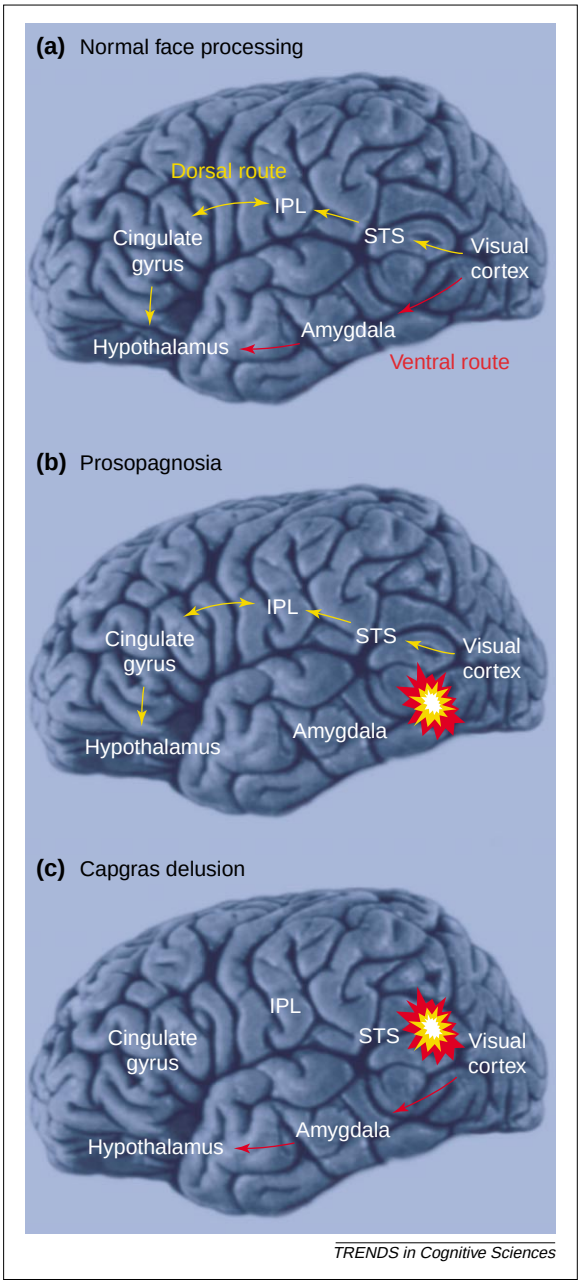

Fig. 3. Neuroanatomical account of face processing. (a) Normal face processing. The yellow route shows the covert dorsal route via the IPL (inferior parietal lobule) and the STS (superior temporal sulcus). The red route is the overt ventral route to recognition. (b) In prosopagnosia the overt ventral route is damaged, hence face recognition is compromised. (c) This account can also be applied to explain Capgras delusion, where the damage is postulated to be in the covert dorsal route. Adapted from Ref. 11.

process would take place. How such an integrative device would compare the two forms of information and the workings of the attributional process remains to be understood but it is obviously important for the complete understanding of Capgras syndrome.

Delusional experiences

Capgras delusion raises many interesting issues concerning the interaction between cognitive processes underlying person recognition and accompanying

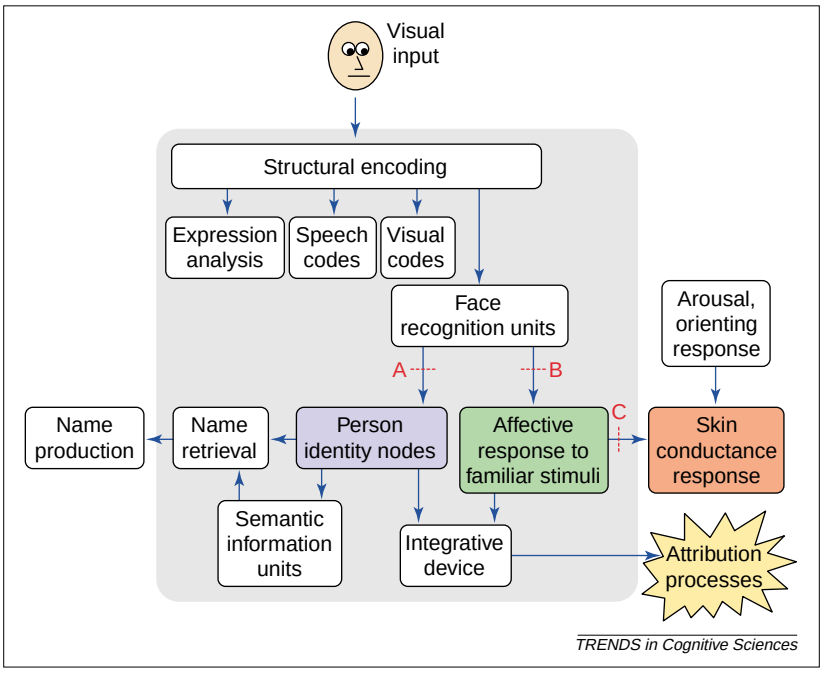

Fig. 4. An adaptation of the Breen *et al.* model of face recognition and misidentification syndromes[19]. This model also suggests how the two routes of recognition must be brought together in order for a delusion to occur. An abnormality at location marked 'A' will lead to a loss of overt face recognition and, therefore, is an account for prosopagnosia. An abnormality at location marked 'B' will lead to a loss of the affective response and the autonomic reaction to a face, and will therefore also lead to a conflict in the integrative device and is an account for Capgras delusion. An abnormality at location C will lead to a loss of differential skin conductance responses for familiar and unfamiliar faces but will not lead to delusions; hence, this is an account for the performance of fronto-ventromedial lesioned subjects. One difference between this model and the Breen *et al.* model is that the person identity nodes and the affective response module are not directly connected. Connection between these modules would imply that damage at A or B could be circumvented.

Questions for future research

- How are the identity processes and affective response processes integrated and how does the system 'know' something is missing when comparing the stored representations of known individuals?
- What mechanisms are involved in producing the delusions (of impostors, robots, aliens, etc.) after the anomalous perceptual experiences described here?
- Why do some patients who fail to elicit larger SCRs to familiar faces not develop Capgras delusion?
- Which brain areas are associated with imbuing facial precepts with affective tone?
- What attributes of a familiar face lead to increased skin conductance? Is mere familiarity sufficient or is it the associated memories that produce the autonomic response?
- The Capgras delusion is normally found in the visual domain, but there are reported Capgras delusions in the auditory and haptic domains. How can these other forms be reconciled with a theoretical description that is currently confined to face processing?

emotional responses, not least the basis for our sense of familiarity when encountering someone we know well, which can, as both William James[28] and Bertrand Russell[29] suggested, involve an automatic concurrent 'glow'. The same may well be true in the parallel domain of object recognition: those items with which we are particularly familiar or to which we are in someway attached may enjoy special cognitive status – which, of course, is entirely consistent with evidence for Capgras delusion for objects. It might also play a role in the aetiology of reduplicative paramnesia, a condition arising from brain lesion or dementia, in which the patients assert that places and people exist in more than one context[30].

In all of these cases it is worth stressing the fact that anomalous perceptual experiences, leading to delusions that are usually monothematic and fairly circumscribed, have to be explained by the individual to him or herself[31,32]. In other delusional states there may well be similar underlying perceptual anomalies that require an internally-generated explanation[33]. What is interesting is how powerful the individual and cultural biases can be. To those unaffected by such delusions, such bizarre beliefs appear to be an odd way to interpret sensory data, however unusual they may be[4]. These patients show what Stone and Young refer to as a bias in favour of observational adequacy, rather than accepting a more conservative explanation for their experiences[34]. It is this sort of analysis that allows a growing confidence to those who subscribe to the burgeoning field of cognitive neuropsychiatry[35].

It has been shown that patients with fronto-ventromedial lesions who also show no differential SCRs to famous and unfamiliar faces do not display the Capgras delusion[12]. It would appear, therefore, that simply a lack of autonomic response is not itself sufficient to produce the Capgras delusion. There are two possible ways of explaining why some patients who do not display differential SCR show the Capgras delusion whereas others do not. The first explanation is that some patients choose to believe the confabulations necessary for the full-blown delusion to become manifest because of a second-stage abnormality, perhaps within an attribution stage following face processing[18,36,37]. The second explanation is that the fronto-ventromedial lesioned patients still experience the affective response to familiar faces but there is some interruption between this stage of processing and the processes causing the changes in SCRs. This account, therefore, places the abnormality at different locations for the two different groups of patients (either prior to or after the affective response to familiar stimuli, see Fig. 4).

Capgras delusion, then, serves both to test our ideas as to how faces and other objects are recognized and to provide unique insights into these processes. In doing so, however, this seemingly bizarre phenomenon itself raises a number of new questions yet to be addressed.

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Eye movements during reading: some current controversies

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For many researchers, eye-movement measures have become instrumental in revealing the moment-to-moment activity of the mind during reading. In general, there has been a great deal of consistency across studies within the eye-movement literature, and researchers have discovered and examined many variables involved in the reading process that affect the nature of readers' eye movements. Despite remarkable progress, however, there are still a number of issues to be resolved. In this article, we discuss three controversial issues: (1) the extent to which eye-movement behavior is affected by low-level oculomotor factors versus higher-level cognitive processes; (2) how much information is extracted from the right of fixation; and (3) whether readers process information from more than one word at a time.

Although researchers have measured eye movements since 1879, recent technological innovations have allowed scientists a much more accurate view of the relationship between eye movements and reading[1]. Prior to about 1975, researchers tended to focus primarily on the observable surface aspects of eye movements in reading and there were few attempts to use eye-movement data to infer underlying cognitive processes in reading[2]. However, recent research on eye movements during reading has undergone both a paradigm shift and a resurgence – instead of being viewed as a simple observable behavior that is unrelated to reading, many researchers now use eye-movement data as a vital tool for understanding the on-line operations involved in the reading process. For the most part, eye-movement data have proved to be highly reliable and useful in inferring the moment-to-moment processing of individual words and larger segments of text. However, a

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