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Accounting for Delusional Misidentifications

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Accounts of the major DMSs are given using theoretical models of the functional components underlying recognition of familiar people. Thus, Capgras' syndrome is suggested to involve impairment of processes that can support 'covert' recognition of familiar faces in prosopagnosia. It therefore forms a potential 'mirror image' of the impairments underlying prosopagnosia, and earlier attempts to link the two conditions directly are questioned. Frégoli syndrome and intermetamorphosis are explained as defects at different stages of an information-processing chain. Not only are these accounts consistent with the association of different DMSs with different brain injuries, but they also offer both suggestions for new inquiries and predictions about possible preserved and impaired abilities.

The delusional misidentification syndromes (DMSs) are psychiatric disorders distinguished by the fact that they all involve some deviation from normal processes of recognising people. A recent collection of papers on DMS (Christodoulou, 1986) gives a good coverage of the history, findings and issues, and Young & Ellis (1989*a*) discuss theoretical explanations for the way people process physiognomic information. As yet, however, the theoretical insights gained from psychological and neurological studies have had little impact on the traditionally psychiatric field of DMS.

Before discussing three of the most prominent DMS disorders (Capgras', Frégoli and intermetamorphosis), we first describe the model of face recognition by Bruce & Young (1986), which extends that by Hay & Young (1982) and is the best summary of available data. More extensive discussions of such models are available elsewhere (e.g. Bruce, 1988; Young & Ellis, 1989*a*; Ellis, 1989). Here, we simply focus on those

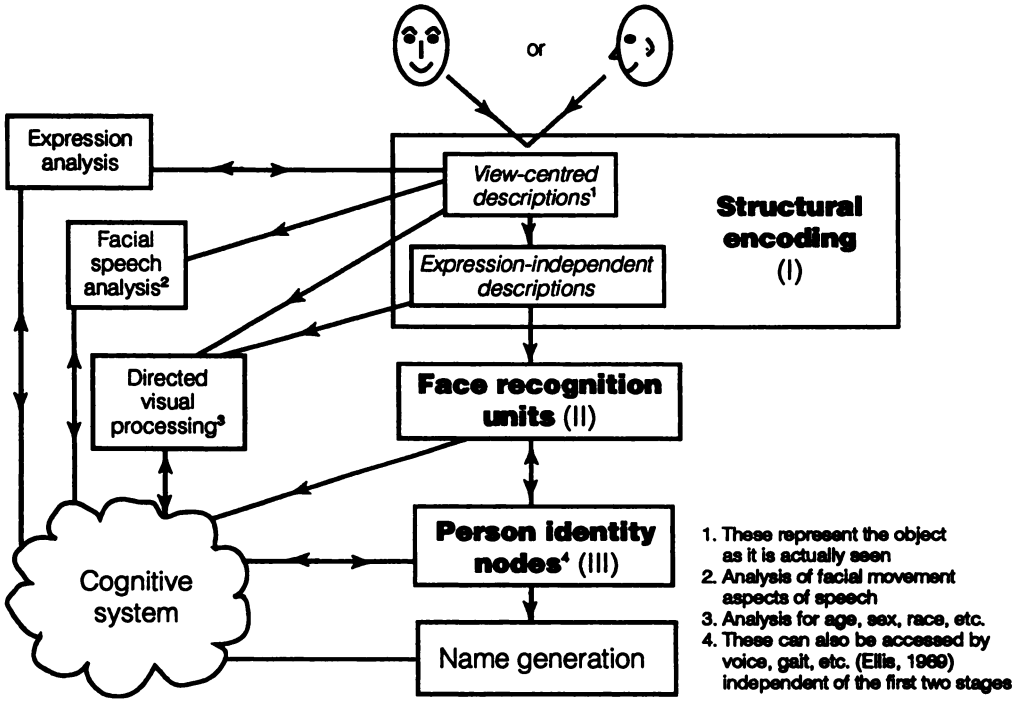


FIG. 1. The information-processing model of familiar face recognition (Bruce & Young, 1986).

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parts of the model that are relevant for our attempts to provide theoretical explanations for DMS. Following this, we consider the neurological condition of prosopagnosia in order both to underline the conclusions of the face-recognition model and to provide the specific hypothesis for our analysis of Capgras' syndrome.

A model of face recognition

It is possible to attempt a theoretical explanation of DMSs within the kind of information-processing model of face recognition put forward by Bruce & Young (1986*a*). According to this model (Fig. 1) the recognition of a familiar face involves sequential stages. Firstly, there is a stage (I) in which the information concerning the structure of the seen face is encoded using 'descriptions' which are view-centred (i.e. represent the object as it is actually seen) and/or independent of expression. These descriptions provide raw data which are then analysed for expression, facial speech and information about age, sex, race, etc. Marr's (1982) model of vision was adapted for this stage. The second stage (II) is where the familiarity or otherwise of a face is signalled by the Face Recognition Units (FRUs). Familiar faces then stimulate information held at the Person Identity Node (PIN) level (III), where semantic or biographical information is stored. This third stage may also be accessed by voice or gait or other means of signalling identity. According to Hay & Young (1982), Bruce & Young (1986*a*), and Ellis (1986*a*), other aspects of face processing, such as the perception of facial expression, are accomplished independently from and in parallel with the determination of individual identity. Furthermore, information about the person's name is stored and retrieved independent of his or her biographical details.

Support for the Bruce & Young (1986*a*) model of face recognition comes from a diary study of everyday errors of identification (Young *et al*, 1985). This revealed problems including diarists recognising a face as being familiar but being unable to access biographical information concerning the person, and, more often, them knowing not only that the face is familiar but also all sorts of biographical details, yet not being able to locate the individual's name. Such experiences are consistent with the idea that the recognition process can become 'blocked' at each of the successive stages shown in Fig. 1.

Other experiments using reaction-time techniques have also produced data consistent with a sequential series of decision stages each of which takes a finite time to complete in a prescribed order (see Bruce, 1988; Sergent, 1986). The existence of independent modules for analysing other aspects of faces than identity (categorical definitions of age, sex, race, etc. and facial expression) are also supported by latency data (see Bruce, 1986; Young *et al*, 1986; Bruce *et al*, 1988).

Much of the strongest evidence for this model however, comes from neurological case histories of patients suffering from prosopagnosia, the inability to recognise previously familiar faces (Bodamer, 1947) which usually follows bilateral occipital-inferotemporal damage (Meadows, 1974). This condition produces a more or less complete breakdown of overt face recognition ability in which even the most familiar faces – friends, relatives, family, and the patient's own face when seen in a mirror – are not consciously recognised. (For a history of the disorder, see Ellis, 1989.) Typically, prosopagnosic patients fall into one of two or more groups depending on whether their symptoms are primarily perceptual, whereby they see faces as blobs, caricatures, all alike, etc., or more memory based, whereby faces look normal enough but evoke no sense of familiarity (Hécaen, 1981; De Renzi, 1986).

The majority of prosopagnosics have suffered strokes and are middle-aged to elderly men (Mazzuchi & Biber, 1985). Usually, bilateral areas of occipito-temporal cortex are involved (Meadows, 1974; Damasio *et al*, 1982), but in some cases neuroimaging techniques have revealed occipito-temporal lesions involving only the right cerebral hemisphere (De Renzi, 1986; Landis *et al*, 1986). Most prosopagnosic patients manage to identify familiar people on the basis of voice, gait and clothing which suggests that the damage occurs before the PIN stage of processing. Some display problems in identifying facial expression (Shuttleworth *et al*, 1982) but most prosopagnosics can interpret the moods of others based upon physiognomic cues (Bruyer *et al*, 1983). Face recognition and expression interpretation, however, are doubly dissociable, implying separate mechanisms (Kurucz & Feldmar, 1979). By the same token, face recognition and the ability to make use of lip-reading to aid normal communication also show double dissociation (Campbell *et al*, 1986). As Ellis (1986*b*) has argued, these different cases lend support to the various stages of the model shown in Fig. 1.

Theoretical approach to understanding DMS

Our aim in this paper is to advance explanations for the major DMSs based on the current understanding of normal face-recognition processes and the ways

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in which they can be impaired. In doing this, our explicit intention is to extend the cognitive neuropsychological approach to the study of conditions that have been traditionally defined as being psychiatric in nature.

Recent developments in cognitive neuropsychology have leant heavily on the view that complex mental functions involve the interaction of a number of modular subcomponents (Fodor, 1983; Ellis & Young, 1988; Shallice, 1988). The neurological and neurophysiological literature now provides abundant evidence for such modular organisation, which would make sense in terms of the important 'design principles' of keeping essential neural interconnections as short as possible and allowing part of a complex system to be modified by experience or evolution without adversely affecting the performance of the rest of the system (Marr, 1982; Cowey, 1985).

Studies of the different types of breakdown that can affect performance of any complex cognitive capacity can thus provide insight into the underlying organisation of the responsible functional components. More importantly, an adequate theoretical model should be able to account for the types of impairment that can occur and in turn, should itself be modified if inconsistent forms of impairment are actually observed (Ellis & Young, 1988).

Here we try to demonstrate that this approach which has been successful in understanding the breakdown of normal performance, both in terms of everyday slips and errors and the deleterious effects of brain injury, can also be of benefit in understanding delusional misidentifications. In doing so, we do not entirely take a leap into the dark, since much recent evidence has begun to suggest some organic involvement in DMS, and parallels to neurological conditions have already been drawn. Furthermore, such an approach, carried out systematically, has advantages in allowing a more theoretical understanding of the conditions, in suggesting aspects worthy of further investigation and in predicting certain patterns of co-occurring symptoms.

The last point leads us to an important caveat. It is traditional to refer to delusional misidentification *syndromes*, but in fact, unlike when they were first introduced, each of these is now actually defined by a single particular symptom, rather than a symptom constellation. We would thus prefer to speak of Capgras *symptom*, and so on, but have retained the conventional 'syndrome' terminology here because it has become so thoroughly entrenched in the literature.

The major DMSs

Capgras' syndrome

In 1923 Capgras & Reboul-Lachaux reported the case of a 53-year-old woman who displayed what they called "l'illusion des sosies", a delusional belief that people who she knew had been replaced by identical doubles (see Berson, 1983, for an explanation of 'sosies' and Christodoulou, 1986, for the historical/mythological background). In addition to believing that her husband and children had been replaced by doubles, the woman later claimed that police and neighbours had been similarly duplicated. She also believed that there existed doubles of herself.

Berson (1983) reports a computer search which located 133 similar case reports in English, as well as reports in French, Italian, Russian, Dutch, German, Spanish and Japanese; and Signer (1987) found reports of 315 patients in the English and French literature. In most cases the diagnosis was of schizophrenia. Many cases also appeared to involve organic disorders, but, regardless of the cause of Capgras' syndrome, the symptoms of most patients are dominated by a marked paranoid component (Berson, 1983). It is also worth noting that the people believed to have been duplicated are generally those close to the patients. These are the kind of people for whom the patient has strongly positive or negative affective responses. Sometimes patients believe that there is more than a single impersonator (Todd *et al*, 1981).

Capgras' patients tend to be paranoid, or, at least, rather suspicious by nature, and the doubles are invariably thought of as evil or dangerous in some way. Capgras' patients also tend to suffer from depersonalisation–derealisation. Patients sometimes report that everything looks strange, for example, things may look painted or not natural and faces may look like masks or wax models or seem to have been changed by plastic surgery. Interestingly, the instance of Capgras' among females is generally thought to be rather higher than it is among males (Sims & White, 1973).

Organic v. functional aetiology of Capgras' syndrome

Although labelling Capgras' syndrome as a manifestation of paranoid schizophrenia has constituted an explanation of the disorder for some commentators, it is a rather unsatisfactory and incomplete one. Merrin & Silberfarb (1976), whose ideas were more impressive, saw a connection between depersonalisation–derealisation and Capgras' syndrome

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symptoms when they wrote "a number of cases began with reduced feelings of unreality or depersonalisation, (were) followed by indiscriminate misidentification and finally by the establishment of the Capgras' delusion". However, as Todd *et al* (1981) point out, this in itself does not fully explain the development of Capgras' syndrome, because patients afflicted by feelings of depersonalisation or derealisation have insight and appreciate the illusory nature of the phenomenon, whereas those with Capgras' delusion generally don't.

In the main, Capgras' syndrome has been treated as a functional disorder. A number of investigators have noted, however, that Capgras' patients often have associated brain lesions, or that their symptoms may have resulted from some form of poisoning or other exogenous factor. In his review of focal abnormalities of the central nervous system in patients with a DMS, Joseph (1986) discovered that the majority of patients have clear computerised tomography (CT) scan evidence of brain abnormalities. Of the 23 patients studied by him, approximately two-thirds had signs of cortical atrophy. In all cases the signs were bilateral: in some they were largely frontal; in others parietal or temporal. Unfortunately, Joseph did not break down his group by specific DMS so there is no way of attributing specific sites of brain damage to particular types of DMS. It may be noted that Kiriakos & Aranth (1980) found obvious evidence of organic damage in only four out of 13 Capgras' patients, but they employed X-ray and electroencephalography (EEG) measures – rather gross forms of imaging – whereas many lesions require CT scan or magnetic resonance imaging (MRI) to become evident. Joseph (personal communication, 1989) claims that all DMS patients given full evaluation have revealed some CNS abnormality.

Fishbain *et al* (1986) reported the case of someone displaying Capgras' syndrome following metrizanide myelography. The patient, a 67-year-old male, had been admitted to hospital following pain in his left calf. Twenty hours after myelography, the patient developed the following symptoms: severe vomiting, severe headache, confusion, disorientation to time and place (he thought he was in his son's home) and a delusional belief that his doctor was a duplicate of the original. After treatment with prochlorperazine, symptoms gradually remitted.

MacCallum (1973) also reported Capgras' syndrome following medical intervention; in this case it appeared to be due to the patient inhaling an overdose of a bronchial dilator containing adrenaline and adropinemethonitrate. In another of MacCallum's cases, Capgras' syndrome appeared to be related to the patient's diabetes. A patient described by Hay *et al* (1974) showed Capgras-like symptoms following electroconvulsive therapy (ECT). All of these instances cast doubt on Enoch's (1963) suggestion that the title 'Capgras' syndrome' should be reserved for the delusional misidentification and replacement for an identical double in a clear sensorium, and this *without organic basis* (i.e. in a setting of functional illness with a psychodynamic interpretation).

Explaining Capgras' syndrome

As we have indicated earlier, a number of researchers now take the view that Capgras' syndrome may be the result of underlying organic brain damage. Although the evidence for this is not yet conclusive, modern brain-imaging techniques have shown that in a large number of cases of Capgras' syndrome there is clear evidence of brain lesions. Nonetheless, some commentators, such as Bersen (1983), argue that "organic factors in themselves, however, seem neither necessary nor sufficient to explain the particular and peculiar content of the delusion". This view is perhaps overstated.

One of the most interesting suggestions put forward by those who favour an organic basis for Capgras' syndrome is that the condition is related to one long recognised in the neurological literature, known as reduplicative paramnesia (Alexander *et al*, 1979). Such paramnesias often involve reduplication of places, with the patient maintaining that he/she is in a place which is an almost exact copy of the actual location. There is a clear parallel with the Capgras' delusion, which involves reduplication of people, and Alexander *et al* (1979) demonstrated that a combination of bilateral frontal and right hemisphere damage seemed to be present for patients of both types. The link between the two conditions is supported by Anderson (1988), who drew attention to a number of cases in which Capgras' patients were observed to reduplicate more than just people. Interestingly, Kapur *et al* (1988) reported the case of a 71-year-old man who developed reduplicative paramnesia for places but not faces following right frontal vascular lesion, indicating the dissociable nature of reduplicative paramnesia.

A link between prosopagnosia and Capgras' syndrome has also been suggested, but never successfully forged. Lewis (1987) in his study of a single case of transient Capgras' syndrome, for example, provided MRI data demonstrating the presence of bilateral occipito-temporal lesions as well as smaller bilateral frontal lesions. The frontal lesions are consistent with the findings of Alexander *et al* (1979) but occipito-temporal lesions are the usual anatomical correlate of prosopagnosia (Meadows,

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1974; Damasio *et al*, 1982). As we have said, the occipito-temporal lesions underlying prosopagnosia are often bilateral, but in some cases (based only on evidence from brain imaging) are apparently located exclusively in the right cerebral hemisphere (De Renzi, 1986; Landis *et al*, 1986).

The patient studied by Lewis (1987) had actually shown signs of mild prosopagnosia during childhood, and this prompted him to suggest some sort of connection between the two syndromes. According to Lewis there seems to be a parallel between prosopagnosia and Capgras' syndrome. In the latter case there would appear to be a disconnection between the percept of a face and its evocation of affective memories.

Joseph (1986) also posited a disconnection explanation. "It may be that in misidentification syndromes in particular and reduplicative phenomena in general, brain disease causes a disconnection between the right and left hemisphere cortical areas, that decode afferent sensory information and maintain the normal functions of orientation to person, place, time and object relationship that we describe as 'orientation'." In Joseph's view each hemisphere forms a representation of a face separately. Normally, these images are 'fused' by interhemispheric transfer, and thus are presented on a conscious level as a fully integrated representation of the external world. "In patients with misidentification syndromes . . . each hemispheric 'image' is presented separately leading to an awareness of the conscious level of two simultaneously separate but physically identifiable or similar persons, places, objects, times or object relationships. Depending upon which hemispheric connections are most impaired, the clinical syndromes of misidentification, reduplication, or disorientation will result". Although there is as yet no very compelling empirical evidence to support Joseph's view, it merits further investigation and is not necessarily inconsistent with the theoretical ideas which we present shortly.

Face-processing impairments in Capgras' patients

There seems little doubt that face-processing impairments can be found in Capgras' patients. Tzavaras *et al* (1986) found that on face-matching tests (Tzavaras *et al*, 1970) DMS patients were significantly poorer than pathological and normal controls. They deemed this "an infra-clinical prosopagnosic symptom" which may imply some organic factor in DMS. Shraberg & Weitzel (1979) studied two Capgras' patients by giving them the Benton face-matching test (Benton *et al*, 1983) which they claimed was particularly sensitive to prosopagnosia. Finding that their Capgras' patients also performed poorly on this test they then argued that a parallel existed between the two syndromes. However, as Benton (1980) has emphasised, the face-matching test is not sensitive to prosopagnosia: many, if not most, prosopagnosics can adequately perform the test (Bauer, 1984; Young & Ellis, 1989*b*), although they may rely on unusual strategies (Newcombe, 1979). Instead, it is patients with right parietal lesions who perform such tasks especially poorly; yet these are not prosopagnosic (i.e. they remain able to recognise familiar faces). In the same way, it should be emphasised that patients who experience the Capgras' delusion are not prosopagnosic: they continue to recognise without apparent difficulty the dummies and imposters that have replaced their relatives. So if there is any connection between DMS and prosopagnosia it is unlikely to be a direct one.

Other misconceptions of the nature of prosopagnosia have also been introduced into the literature on Capgras' syndrome. For example, Todd *et al* (1981) maintain that in order for an organic aetiology for Capgras' syndrome to be established, it is necessary to show a clear cause and effect relationship between prosopagnosia and Capgras' syndrome. They even add that the association is particularly doubtful in view of the fact that there are usually no signs of any visual field defects in Capgras' patients. Here they appear to believe, erroneously, that visual field defects are a necessary concomitant of prosopagnosia. This is not the case. Prosopagnosia can exist in the absence of visual field defects, although they frequently accompany the condition but not, it would seem, in any causally connected way (Meadows, 1974).

If there is a connection between prosopagnosia and Capgras' syndrome, then it operates in a rather more subtle way. In order to develop this argument, it is necessary to consider evidence for different recognition routes, derived from the phenomena of 'covert recognition' of faces by prosopagnosics. Bauer (1984) studied a prosopagnosic patient, LF, who had suffered bilateral occipito-temporal brain damage following a motorcycle accident. He was profoundly prosopagnosic, and yet when shown pictures of previously familiar faces (famous or personally known), along with five names drawn from the same semantic category, which were read out to him, LF displayed significantly greater autonomic responses to the correct name compared with the four incorrect names. This technique, known as the Guilty Knowledge Test, is widely used by American police forces as a method for detecting lying by criminal suspects. Bauer's data mainly concern the skin

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conductance response (SCR). LF showed a discriminating SCR to some 62.5% of the known faces – well above any chance level of performance (i.e. 20%). Tranel & Damasio (1985) confirmed Bauer's observations using a modified technique in which a series of randomly arranged known and unknown faces were presented to two prosopagnosic patients. They also showed increased autonomic reactions to the previously known faces.

Bauer (1984, 1986) advanced the view that there are two routes to facial recognition. The main route runs from visual cortex to temporal lobes via the inferior longitudinal fasciculus. Following Bear (1983), he termed this the 'ventral route': it corresponds to the system responsible for overt or conscious recognition, and it is the route which typically is damaged in cases of prosopagnosia. The other, described as the 'dorsal route', runs between the visual cortex and the limbic system, via the inferior parietal lobule, and is sometimes intact in prosopagnosic patients. It is this latter route which, Bauer claims, gives the face its emotional significance and hence, when the ventral route is selectively damaged, can give rise to covert recognition (i.e. recognition at an unconscious level).

If we accept Bauer's dual recognition route (and from the work of De Haan *et al*, 1987, it may be that we should be looking for more than two routes), we can try to apply the notion towards an understanding of Capgras' syndrome. Young & Ellis (1989*a*) have begun to explore the phenomenon of Capgras' syndrome by suggesting that it is a mirror image of prosopagnosia. In other words, patients with Capgras' syndrome seem to have an intact primary or ventral route to face recognition, but may have a disconnection along or damage within the secondary or dorsal route. This would mean that they receive a veridical image of the person they are looking at, which stimulates all the appropriate overt semantic data held about that person, but they lack another, possibly confirming, set of information which, as Lewis (1987) and Bauer (1986) have independently suggested, may carry some sort of affective tone. 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 rationalisation strategy in which the individual before them is deemed to be an imposter, a dummy, a robot, or whatever extant technology may suggest.

Derombies (1935) also attached great importance to the affective state of Capgras' patients. He suggested that Capgras' syndrome results from a simultaneous intellectual recognition and affectively engendered non-recognition of faces: at one level the patient recognises the face but at another level he or she does not recognise it. More recently (and also independently) Anderson (1988) made a similar suggestion when he argued "that the Capgras' delusion results from lesions of the pathway for visual recognition at a stage where visual images are imbued with affective familiarity". Anderson's ideas (and those of Derombies) are similar to the ones presented above although he (1988) is not able to be more specific about the mechanism by which "affective familiarity" may operate. Indeed, one difference is that our hypothesis rests on the view that more than one recognition pathway is involved, and makes the clear prediction that Capgras' patients will *not* show the normally appropriate skin conductance responses to familiar faces, despite the fact that these will be overtly recognised.

The explanation, however, is still incomplete. Bersen (1983) pointed out that in Capgras' syndrome, not all faces seem to be duplicated, only those of people close to the patient. A possible answer to this is that it is only in these instances that one expects more than the basic information about a face. Only certain faces normally have associated with them a particularly strong affective component, and therefore only these are vulnerable in the case of any disconnection between the visual areas and the cerebral structures involved in Bauer's dorsal recognition pathway.

One may extend this argument to other reduplicative paramnesias. Places, objects, etc. are not affectively neutral and so the absence of an emotionally charged input could produce the feeling of recognition, but it not being quite right. Again this would be particularly true for those places and objects with which the patient is most familiar.

Frégoli syndrome

In 1927 Courbon & Fail reported another form of DMS. Their patient was a 27-year-old labourer's daughter who had worked variously as a domestic servant, in a factory, in a restaurant, etc. She was paid by the day and slept in Salvation Army type refuges. Her abiding passion was the theatre; she preferred to go without food in order to attend a performance. Admitted to hospital following an attack on an employer, she reported to Courbon & Fail that she was the victim of enemies: in particular of the actresses Robine and Sarah Bernhardt whom she had often seen in the theatre. She insisted that these actresses had followed her about for many

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years, taking the form of people she knew or met, overpowering her thoughts, making her do this instead of that and stroking her or forcing her to masturbate. The patient reported often being interfered with or attacked by people who particularly took the form of Robine.

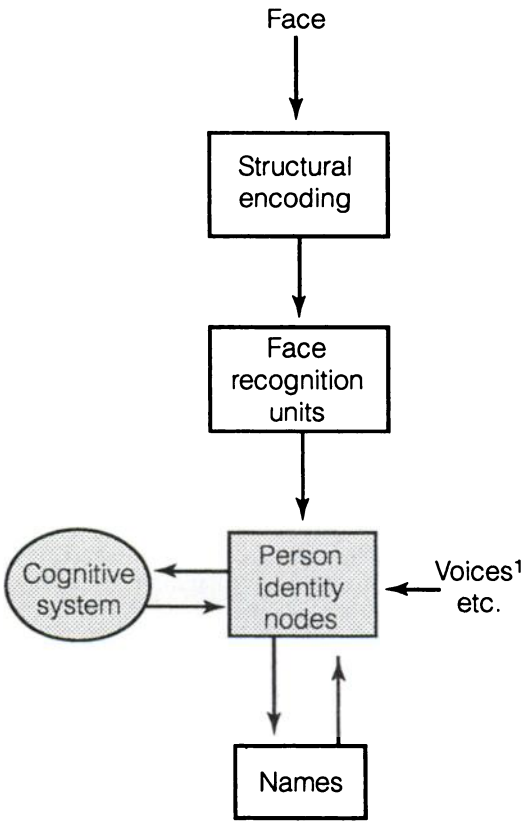
Courbon & Fail adopted the term 'Frégoli syndrome' after the Italian actor and mimic, Leopoldo Frégoli, famous for his ability to impersonate people on the stage. The hallmark of the Frégoli syndrome is the delusional misidentification of familiar persons disguised as others. De Pauw *et al* (1987) classified the illusion of Frégoli as hyper-identification. They contrast this with Capgras' syndrome, which was classified as hypo-identification. They reported the case of Mrs C, a 66-year-old widow, who believed that she was being persecuted by her cousin and his female friend. She described in detail how the couple disguised themselves with make-up, wigs, dark glasses, false beards and different clothes, and repeatedly accosted her when she was out. She confronted strangers in public demanding that they reveal their true identity; she took complicated routes on her way home to shake off her persecutors; and she often reported their activities to the local police. "They keep changing their clothes and their hairstyles, but I know it's them. He can look like an old man. They want a medal for doing that. It's like an actor and actress preparing for different scenes," she said.

Mrs C had a history of brain damage: she suffered haematoma over the left frontal area after a fall in 1982. A CT scan also showed right-sided posterior-temporo-parietal infarct together with general cortical atrophy. She had earlier been treated for hypertension and she had also been diagnosed as having right-sided temporal arteritis. Although she recovered from her initial symptoms of speech slurring and memory problems as well as her orientation difficulties, she then developed the delusional belief just described. Her Frégoli syndrome seemed to be controllable with trifluoperazine.

As in cases of Capgras' syndrome, Frégoli may be associated with some degree of objective face-processing impairment. De Pauw *et al* (1987) found that Mrs C scored poorly on the Warrington face-recognition test. More detailed investigation of such impairments would be worthwhile.

Frégoli syndrome bears some relation to a type of error that all of us make in everyday life (Young *et al*, 1985). When we expect to meet a particular person, occasionally and transiently we will misidentify a stranger as that person. However, we usually quickly correct this mistake when inconsistent evidence is picked up. In the Frégoli syndrome, on the other hand, such inconsistencies are also noted, but attributed to the effects of disguise. The syndrome can thus be characterised as involving impaired decision mechanisms. In Bruce & Young's terms, the impairment is in the cognitive system itself, which places an inappropriate evaluation on the evidence it receives. Such decision mechanisms can also malfunction for normal people, as other everyday errors collected by Young *et al* (1985) showed.

The PIN stage and associated cognitive system would therefore be the likely site for malfunction leading to Frégoli syndrome (see Fig. 2). Specifically, either the PIN system becomes driven by a deranged cognitive system or hyperexcitable nodes representing particular people appear to become engaged, sometimes almost regardless of whatever output from the previous FRU stage occurs, and impaired decision mechanisms accept this evidence. Since strangers' faces



1. Not shown in original model.

FIG. 2. Simplified face-recognition model indicating the level at which it is proposed the illusion of Frégoli occurs.

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are often the source of the patients' delusions it is clear that it would be of interest to study whether any degree of resemblance to the misidentified person is necessary.

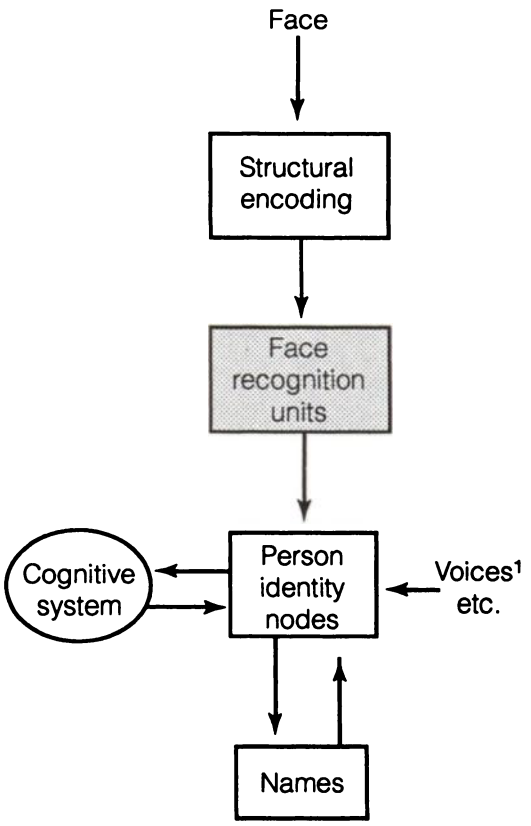
Intermetamorphosis

First described by Courbon & Tusques (1932), intermetamorphosis refers to cases where, for the patient, the physical appearance of some people may change radically to correspond with the appearance of someone else. Courbon & Tusques described a 49-year-old depressive patient who reported that animals and objects she owned took the form of another animal or object. She also experienced changes in her husband's appearance, which could become exactly like that of a neighbour or could rapidly transform to look larger or smaller or younger. Various people in rapid succession also took on the guise of her son, but she used the particularly large size of his feet to discount these delusions.

Courbon & Tusques (1932) pointed out that intermetamorphosis involves a false recognition of both appearance and associated identity, whereas in the illusion of Frégoli the patient is not confused about physical appearance. Intermetamorphosis is relatively rare: Bick (1986) reviewed two cases reported after Courbon & Tusques' (Christodoulou, 1975; Malliaros *et al*, 1978) and presented another, a 42-year-old woman who had had a long history of epilepsy and paranoid schizophrenia. During a period in hospital, she became suddenly agitated and declared that her doctor was her dead uncle.

According to Bick (1986), three of the four cases reviewed by him had temporal lobe epilepsy. Joseph (1987) has subsequently described a 49-year-old patient who displayed both Capgras' syndrome (believing her husband had been replaced by an identical imposter) and intermetamorphosis (stating that several other patients looked like her father and that her son's physical appearance had altered). In this case a CT scan was given and it revealed mild atrophy of the frontal lobe, severe bilateral atrophy of the temporal lobe, moderate atrophy of the occipital lobe and a mildly enlarged ventricular system. The *prima facie* evidence for a possible organic component in intermetamorphosis, as for other DMS disorders, is thus quite strong.

In order to establish an explanation for intermetamorphosis within the Bruce & Young (1986) model of normal face recognition processes, it is necessary to focus on the second stage, the FRU stage, highlighted in Fig. 3. Representations for known faces are posited to exist, each of which is somehow excited or triggered by the appearance of



1. Not shown in original model.

FIG. 3. Simplified face recognition model indicating the level at which it is proposed intermetamorphosis occurs.

the particular person. The FRUs then signal familiarity and, in turn, communicate with appropriate PINs in the semantic memory system where the person's biographical details are stored.

Intermetamorphosis may be construed as an inappropriate excitation of an FRU which could occur, for example, if the particular FRU had a considerably lowered triggering threshold. A corollary to this suggestion is that it is more likely that faces similar to the misidentified person will cause the unit to fire inappropriately, and, as with the Frégoli symptom, more systematic investigation of the resemblance between the appearance of the actual and delusionally substituted people would be useful. A clear prediction from our account of these conditions is that physical resemblance will be important in triggering episodes of intermetamorphosis but much less important to the Frégoli delusion.

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At this stage it is not possible to establish why and how such threshold shifts occur, but notice that once an FRU has fired it will automatically then cause the corresponding and equally inappropriate PIN to become excited. Unfortunately, none of the case histories of patients' behaviour during such an instance of misidentification has reported how the patients responded to voices. According to the model shown in Fig. 3, voices input from a different recognition system but employ the same PINs. Joseph & O'Leary (1987) indeed reported a Frégoli case who claimed that a Mr B had been replaced by another acquaintance. While the physical appearance of Mr B was unchanged he appeared to have the other person's voice. Thus, whereas Frégoli patients may misperceive voices, those with intermetamorphosis syndrome could make an incorrect facial identification but should not produce the same error on the voice. This should produce discrepancies at times which would, perhaps, be resolved because of the primacy of visual over aural perception but could, nonetheless, be elicited during clinical interview.

Conclusion

The advantages in attempting to explain various DMS states within the theoretical constructs derived from studies of normal and pathological face recognition are numerous. Chief among them, of course, is the hope that it may lead both to a complete understanding of DMSs and, in turn, assist clinicians in diagnosis and treatment.

Secondly, the opportunity to bring together a seemingly disparate set of phenomena, for some time effectively dealt with as being largely unrelated, carries with it many advantages. For example, the ideas that have been posited regarding Capgras' syndrome allow us to perceive a very obvious deficit in the model outlined in Fig. 1, that is, it only depicts the 'overt' route to face recognition. Experiments on prosopagnosic patients, together with the theoretical ideas we have presented here, strongly suggest that the model is incomplete in that it only displays a single route to recognition and fails to accommodate what, perhaps, may turn out to be a sort of emotional recognition of faces (and indeed other routes, as well).

The third aspect of our endeavours to which we wish to draw attention is its potential value in organising clinical observations of DMS patients. We have suggested various types of evidence that should be collected routinely in such cases. It is, for instance, clearly important to note patients' responses to voices as well as faces, because by doing so we may refine our ideas concerning the level of impairment. Some assessment of the similarity between people mistakenly perceived in intermetamorphosis and wrongly categorised in Frégoli syndrome would also be of theoretical benefit.

The final point we wish to stress is that discussing DMS in relation to prosopagnosia reinforces the suggestions made by Joseph (1986) and others that DMS may originate primarily from organic bases. The next step must surely be to derive routinely in cases of DMS high-quality brain images that should assist in classification. In time it should be possible to correlate particular DMSs with damage to specific brain areas.

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