British Journal of Psychiatry (1991), **159** (suppl. 14), 65–69

The Delusional Misidentification Syndromes

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The basic concept characterising the delusional misidentification syndromes (DMS), particularly the Capgras variety, is the concept of double, or its equivalents. It seems that this concept is prominent in all cultures and this explains the amazing universality of these delusions. Cases of DMS have been reported in practically all parts of the world. In fact, most patients actually mention the word double (or σωσίας, sosie etc.) or refer to transformations (metamorphose, μεταμορφώσεις and the like). The preoccupation, and even enchantment, of man with the notion of doubles from time immemorial in all cultures, as reflected in the myths, traditions, literature, beliefs and metaphysical motions, has been dealt with extensively elsewhere (Christodoulou, 1986*a*).

Syndrome subtypes

Within the framework of the delusional misidentification syndromes four basic syndrome subtypes are included: the syndrome of Capgras (Capgras & Reboul-Lachaux, 1923), the syndrome of Frégoli (Courbon & Fail, 1927), the syndrome of intermetamorphosis (Courbon & Tusques, 1932) and the syndrome of doubles of the self or syndrome of subjective doubles (Christodoulou, 1978).

The syndrome of Capgras is the delusional negation of identity of a familiar person (or persons) in which the patient believes that the misidentified and the familiar persons are identical physically but different psychologically.

The syndrome of Frégoli is in fact a delusional systematic false recognition. Its essence is the delusional identification of a familiar person in strangers. The misidentified and the familiar persons are perceived by the patient as physically different but psychologically identical.

The syndrome of intermetamorphosis is like the syndrome of Frégoli with one difference: the patient believes that there are also physical similarities between the misidentified and the familiar persons. He also usually believes that people interchange as a result of some sort of *'charme'* (French word for bewitchment).

Lastly, the syndrome of subjective doubles is the delusional belief that a stranger has been transformed physically but not psychologically into the patient's own self.

It is perhaps necessary to make a distinction between misidentification syndromes in general and the *delusional* misidentification syndromes.

Misidentification syndromes can be delusional and non-delusional. For example, prosopagnosia and reduplicative paramnesia are surely misidentification syndromes but they are not delusional. The patient has insight into these phenomena and does not attribute, for example, his inability to recognise the face of his mother to transformations, plastic surgery or replacement by an identically looking double. We have suggested that in the presence of a marked paranoid component (e.g. in a setting of paranoid illness) these non-delusional misidentifications may become delusional. Without this paranoid component, non-delusional misidentifications are simply misidentifications – neurological rather than psychiatric. These views echo the position adopted by Sims (1986) when he underlines the need for differentiation of the DMS from autoscopy and prosopagnosia.

Delusional misidentification syndromes occur in various nosological settings but the majority of patients are schizophrenics. The clinical picture, irrespective of diagnosis, is dominated by a marked paranoid component.

I should perhaps draw attention to the fact that DMS occurring in a depressive setting are different in their clinical expression from those occurring in a schizophrenic setting. The former are more 'superficial' as they often appear to be secondary to the ideas of guilt and unworthiness inherent in severe depression (secondary rationalisation). Furthermore, these patients usually suffer from involutional depression, they have a good emotional relationship with the main misidentified person and usually respond to tricyclics (as does the basic illness).

The four syndrome subtypes often co-exist. For example, the symptoms of Madame de Rio Branco, the first patient described by Capgras & Reboul-Lachaux as suffering from *'illusion des sosies'*, included both the syndrome of Capgras and that of subjective doubles.

Vié (1930) considered the syndrome of Capgras as an 'illusion' of negative doubles, differentiating it from the 'illusion' of positive doubles, a term he reserved for the syndrome of Frégoli. Similarly, the present author distinguished the delusional 'hypo-identifications' of the patients with Capgras syndrome

https://doi.org/10.1192/S0007125000296517 Published online by Cambridge University Press

from the delusional 'hyper-identifications' of the patients with the other syndromes (Christodoulou, 1976, 1977). It must be clarified, however, that this author's view is that all four syndromes represent variants of the same basic condition; they often coincide or interchange and should more appropriately be classified together (Christodoulou, 1978).

The DMS should be differentiated from the transient misidentifications occurring in mania and from the banal false recognitions which occur in confusional states. Yet a confusional state or a slowly progressing dementing process provides a suitable ground for the development of these syndromes.

Role of depersonalisation/derealisation

Depersonalisation and derealisation experiences seem to play a major role in the development of the DMS (for review see Christodoulou, 1986*b*). They are frequently encountered in patients with DMS and this has been observed since the first case reports appeared in the literature. Capgras himself, in his original paper (1923), discussed the role of '*sentiment d'étrangeté*' for the production of the syndrome which bears his name and Courbon & Tusques (1932) discussed the role of loss of 'cenesthesia' (the feeling of functioning of the organism) for the production of the intermetamorphosis subtype. Clearly, both the feeling of strangeness and loss of cenesthesia are relevant (if not identical) to depersonalisation/derealisation. The same stands for other similar phenomena described by various authors like for example '*transitivisme*' of Wernicke, which has also, in later years, been associated with the DMS.

It seems possible that under certain conditions (paranoid element, cerebral dysfunction, charged emotional relationship to principal misidentified person) depersonalisation/derealisation symptoms may evolve into delusional misidentification symptoms. It is interesting to note that back in 1935 Mayer-Gross observed that depersonalisation occurs during the stage of minor intensity of an illness and disappears when the illness develops fully. Similar views were expressed by Ackner (1954) who believed that if there is a delusional development, depersonalisation experiences tend to be incorporated within the delusional system and this is accompanied by reduction of the feelings of strangeness and unreality.

Explanatory attempts

"This curious little syndrome", as Capgras called his '*illusion des sosies*', as well as the rest of the subtypes, has been explained on the basis of a variety of pathogenetic hypotheses (Table 1). Most of these

Table 1
Delusional Misidentification Syndromes: Explanatory attempts

Reference	Explanatory attempt
Capgras & Reboul-Lachaux (1923)	(a) Agnosia of identification produced by conflict between affective accompaniments of sensory and mnemonic images. (b) Feelings of strangeness stronger than feelings of familiarity.
Capgras, Lucchini & Schiff (1924)	(a) Marked suspiciousness and preoccupation with details (agnosia through hyperbolic attention). (b) Restitution delusion protecting from depersonalisation.
Capgras & Carrette (1924)	Mechanism protecting from unconscious prohibited desires.
Todd (1957)	Psychotic illness produces personality disintegration to primitive modes of thought with a revival of archaic conceptions concerning doubles and dualisms. Analogy to release of lower neurological mechanisms following destruction of higher and more recently acquired ones.
Enoch (1963)	Love-hate conflict resolved by directing hate feelings onto an imagined double thus sparing the original. Involved psychological mechanisms: projection and splitting.
Christodoulou (1975–1983)	Delusional evolution of experiences produced by cerebral dysfunction (depersonalisation/derealisation, false memories of familiarity, autoscopy, reduplicative paramnesia etc.). Psychological factors function as precipitants.
Joseph (1986)	Interhemispheric disconnection between the areas of the two hemispheres that decode afferent sensory information. Each hemisphere gives rise to a separate image, leading to an awareness at the conscious level of two separate persons, places, objects etc.
Ellis & Young (1990)	Intact ventral route to face recognition but damaged dorsal route. Patients receive a veridical image of the person but lack a confirming set of information which may carry an affective tone (Capgras syndrome). Dysfunction in person identity nodes and associated cognitive system (Frégoli syndrome). Inappropriate excitation of a face recognition unit (Intermetamorphosis syndrome). (*Bruce and Young model of face recognition, 1986*).

https://doi.org/10.1192/S0007125000296517 Published online by Cambridge University Press

hypotheses concern the best-known and most frequently occurring syndrome of Capgras. Some of the main explanatory attempts will be reviewed here.

Let us start with the interpretations proposed by Capgras himself. J.-P. Luauté (1986) studied the three basic publications of Capgras on the topic and interpreted the changing approaches of Capgras on the basis of biographical evidence. Luauté attributed Capgras' modifications of his original approach to influences by psychoanalytically minded students.

In his original publication (Capgras & Reboul-Lachaux, 1923) the author considers the syndrome as an agnosia of identification produced by a conflict between emotional accompaniments of sensory and mnemonic images. If the feelings of strangeness are stronger than the feelings of familiarity then a wrong emotional judgement is produced and a non-recognition follows. As hypothesised by Luauté, the model on which Capgras based his theory must have been the one expressed in Bergson's *Matière et Mémoire* (published, 1896) which proposes two kinds of recognition: (a) automatic and instantaneous recognition and (b) attentive recognition, where memory images are necessary.

In a later publication (Capgras *et al*, 1924) Capgras pointed out the element of hyperbolic attention of the patients and even defined the syndrome as "agnosia through too great attention". In this publication he also viewed the '*illusion des sosies*' as a restitution delusion defending against depersonalisation.

In his publication with Carrette (1924), Capgras considered the syndrome as a mechanism aiming at protection from unconscious prohibited desires (incestuous desires of a female patient towards her father).

Luauté (1986) considers the pathogenetic hypotheses published by Capgras *et al* in 1924 as reflections of the psychiatric orientation of the co-authors rather than of Capgras himself and objects to the fact that the authors failed to consider the strong organic element in their patients' condition, in spite of the fact that the patient Capgras described with Carrette was mentally defective and suffered from Basedow's disease and the patient he described with Lucchini & Schiff was suffering from neurosyphilis.

John Todd (1957) provided an explanation for the phenomena of doubles by hypothesising that psychotic illness causes regression to archaic modes of thought. He quoted evidence from various sources supporting the idea that the concepts of doubles and dualisms are very prominent in mythology and in the majority of primitive religions. Thus, he expressed the view that by causing disintegration of the personality, schizophrenic illness brings primitive mechanisms (like thinking in terms of dualisms and doubles) into the open.

Todd considers the above mechanism as analogous to the liberation of primitive neurological mechanisms which occur in some neurological illnesses as a result of CNS disintegration.

This is a fascinating and elegant hypothesis. Two reservations should perhaps be discussed. Firstly, if personality disintegration was the only cause accounting for DMS then DMS cases should occur much more frequently. Secondly, the patients with DMS usually suffer from the paranoid variety of schizophrenia which renders them less likely to develop personality disintegration. Furthermore, personality disintegration of schizophrenic patients with DMS has been observed to occur to a lesser degree in comparison to that occurring in schizophrenic patients without DMS (Christodoulou & Malliara-Loulakaki, 1981).

Enoch (1963, 1986) and Enoch *et al* (1967) consider the syndrome of Capgras as a psychological solution to the love–hate conflict. The positive feelings of the patient towards an important person in his life have changed. Love and hate exist simultaneously. As a consequence of this, the patient has to 'imagine' the existence of a double who becomes the 'bad' object while the original continues being the 'ideal'. Thus, hate is directed not to the original but to the 'bad' impostor. Projection and splitting are the basic psychological mechanisms involved in this phenomenon.

This important approach is of value and makes sense as a mechanism although it does not fully explain the DMS. It should be noted that the emotional attitude of the patients towards the principal misidentified person is not always negative or ambivalent. It may well be positive (Christodoulou, 1975). Besides, DMS cases have been reported not only with people but also with a range of inanimate objects (Christodoulou, 1977; Green, 1989; Rastogi, 1990). It has been argued that these objects are of emotional importance to the patients. Yet cases in which the affective bond to the inanimate object is totally absent have also been reported (e.g. Ball & Exworthy, 1990).

Clinical and laboratory investigation of a number of patients suffering from various DMS subtypes revealed evidence suggesting that a strong organic component contributes to the pathogenesis of DMS (Christodoulou, 1975, 1976, 1977, 1978, 1981). Cerebral dysfunction gives rise to a variety of experiences (depersonalisation/derealisation, false memories of familiarity, reduplicative paramnesia, autoscopy in the case of the syndrome of subjective doubles, etc.). These experiences, by themselves, do not lead to DMS, but in the presence of a strong paranoid component they may. A charged emotional relationship to a closely related person (who

https://doi.org/10.1192/S0007125000296517 Published online by Cambridge University Press

eventually becomes the double) acts as a precipitant but, in our opinion, this is neither necessary nor sufficient for the production of the DMS.

In addition to electroencephalography (EEG), brain scanning, echo-encephalography, pneumo-encephalography and psychometric evaluation (Christodoulou, 1975, 1976, 1977, 1981) modern techniques like computerised tomography scans, evoked responses, brain electrical activity mapping, positron emission tomography and single-photon emission computerised tomography of the brain (Joseph, 1986) have revealed consistent findings indicative of organic cerebral dysfunction. My own investigation of five patients with DMS with event-related potentials showed that waveform N100 had greater latency in comparison to normal controls. Since N100 waveform is associated with attention and information selection, these preliminary findings point to a cognitive dysfunction of the patients with DMS.

In favour of the organic approach is the observation that the appearance of DMS may predict the development of Alzheimer's disease (Molchan *et al*, 1990), herald the development of dementia (Lipkin, 1988) or occur in AIDS of the CNS (Crichton & Lewis, 1990).

Patients with DMS have many similarities to patients suffering from epileptic psychoses (or psychotic states occurring in an epileptic setting). These two groups of patients often share false memories of familiarity of the *déjà vu*, *déjà vécu*, *jamais vu* and *jamais vécu* type, religious delusions and depersonalisation/derealisation experiences. Moreover, both groups appear to have a rather benign prognosis in comparison to schizophrenics, as they do not seem to manifest the typical 'schizophrenic' deterioration in personality and mood. In view of these similarities and also in view of the fact that these groups of patients often share marked EEG abnormalities (particularly in the temporal lobe), consideration of patients with DMS as suffering from, broadly speaking, 'epileptic' psychoses has been proposed (Christodoulou, 1975; Christodoulou & Malliara-Loulakaki, 1981).

An interesting line of thought has been introduced by Joseph (1986) who considered the DMS as 'disconnection' syndromes. Joseph suggested that organic causes may produce disconnection between right and left cortical areas that decode afferent sensory information. The result of this would be lack of integrated representation of the outside world and formation of two separate, physically identical images. Thus the patient would be aware of two separate, physically identical persons, places, objects etc.

Ellis & Young (1990), in their carefully constructed hypothesis, have suggested that DMS may result from defects at different stages of an information-processing chain put forward by Bruce & Young (1986).

In explaining the Capgras variety, the authors make use of Bauer's view (1984, 1986) that there are two routes to face recognition, a ventral one which is responsible for conscious recognition and runs from the visual cortex to the temporal lobes via the inferior longitudinal fasciculus and a dorsal route which is responsible for covert unconscious recognition and runs between the visual cortex and the limbic system via the inferior parietal lobule. When the former route is damaged, prosopagnosia occurs, when the latter route is damaged, it is possible that the Capgras syndrome results. The patient receives the appropriate semantic information but lacks the affective confirmation of this information. As this refers to the syndrome of Frégoli and that of intermetamorphosis, according to the authors they represent dysfunctions at phase 3 and phase 2 of the face-recognition process advocated by Bruce & Young (1986).

In conclusion, these hypotheses, which can be reduced to the organic and the psychodynamic, have thrown much light on the enigmatic pathogenesis of the DMS but none of them fully explains these syndromes. Perhaps a holistic approach, combining all perspectives, might be able to provide a more satisfactory explanation. As pointed out in the relevant *Lancet* editorial (1986), "the muddled battleground of the organic vs psychodynamic debate . . . is a pathological split that is in itself a version of the double".

References

ACKNER, B. (1954) Depersonalization. II. Clinical syndromes. *Journal of Mental Science*, **100**, 854–872.

BALL, C. & EXWORTHY, T. (1990) Capgras' syndrome and town duplication. *British Journal of Psychiatry*, **154**, 889–890.

BAUER, R. M. (1984) Autonomic recognition of names and faces: a neuropsychological application of the Guilty Knowledge Test. *Neuropsychologia*, **22**, 457–469.

——— (1986) The cognitive psychophysiology of prosopagnosia. In *Aspects of Face Processing* (eds H. Ellis, M. Jeeves, F. Newcombe, *et al*). Dordrecht: Martinus Nijhoff.

BRUCE, V. & YOUNG, A. (1986) Understanding face recognition. *British Journal of Psychology*, **77**, 305–327.

CAPGRAS, J. & REBOUL-LACHAUX, J. (1923) Illusion des sosies dans un delire systematise chronique. *Bulletin de la Societé Clinique de Medecine Mentale*, **2**, 6–16.

——— & CARRETTE, P. (1924) Illusion des sosies et complexe d'Oedipe. *Annales Medico-Psychologiques*, **82**, 48–68.

———, LUCCHINI, P. & SCHIFF, P. (1924) Du sentiment d'etrangete a l'illusion des sosies. *Bulletin de la Societé Clinique de Medecine Mentale*, **121**, 210–217.

https://doi.org/10.1192/S0007125000296517 Published online by Cambridge University Press

CHRISTODOULOU, G. N. (1975) *The Syndrome of Doubles*. Athens: Associate Professorship Thesis, Athens University.
—— (1976) Delusional hyper-identifications of the Frégoli type. *Acta Psychiatrica Scandinavica*, **54**, 305–314.
—— (1977) The syndrome of Capgras. *British Journal of Psychiatry*, **130**, 556–564.
—— (1978) Syndrome of subjective doubles. *American Journal of Psychiatry*, **135**, 249–251.
—— & MALLIARA-LOULAKAKI, S. (1981) Delusional misidentification syndromes and cerebral "dysrhythmia". *Psychiatria Clinica*, **14**, 245–251.
—— (1986*a*) The origin of the syndrome of doubles. In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
—— (1986*b*) Role of depersonalization-derealization phenomena in the delusional misidentification syndromes. In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
COURBON, P. & FAIL, G. (1927) Illusion de Frégoli. *Bulletin de la Societe Clinique de Medecine Mentale*, **15**, 121–124.
—— & TUSQUES, J. (1932) Illusion d'intermetamorphose et de charme. *Annales Medico-psychologiques*, **90**, 401–405.
CRICHTON, P. & LEWIS, S. (1990) Delusional misidentification, AIDS and the right hemisphere. *British Journal of Psychiatry*, **157**, 608–610.
ELLIS, H. D. & YOUNG, A. W. (1990) Accounting for delusional misidentifications. *British Journal of Psychiatry*, **157**, 239–248.
ENOCH, D. M. (1963) The Capgras syndrome. *Acta Psychiatrica Scandinavica*, **39**, 437–462.
—— (1986) Whose double? In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
——, TRETHOWAN, W. & BARKER, J. (1967) The Capgras syndrome. In *Some Uncommon Psychiatric Syndromes*. Bristol: John Wright and Sons.
GREEN, B. H. (1989) Capgras' syndrome. *British Journal of Psychiatry*, **154**, 270–271.
JOSEPH, A. B. (1986) Focal central nervous system abnormalities in patients with misidentification syndromes. In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
LANCET (1986) Devils in disguise. *Lancet*, 5 July, 25–26.
LIPKIN, B. (1988) Capgras syndrome heralding the development of dementia. *British Journal of Psychiatry*, **153**, 117–118.
LUAUTÉ, J.-P. (1986) Joseph Capgras and his syndrome. In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
MAYER-GROSS, W. (1935) On depersonalization. *British Journal of Medical Psychology*, **15**, 103–126.
MOLCHAN, S., MARTINEZ, R., LAWLOR, B., *et al* (1990) Reflections of the self: atypical misidentification and delusional syndromes in two patients with Alzheimer's disease. *British Journal of Psychiatry*, **157**, 605–608.
RASTOGI, S. C. (1990) A variant of Capgras syndrome with substitution of inanimate objects. *British Journal of Psychiatry*, **156**, 883–884.
SIMS, A. (1986) Psychopathology of schizophrenia with special reference to delusional misidentification. In *The Delusional Misidentification Syndromes* (ed. G. N. Christodoulou). Basel: Karger.
TODD, J. (1957) The syndrome of Capgras. *Psychiatric Quarterly*, **31**, 250–265.

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