The Masks of Identities: Who's Who? Delusional Misidentification Syndromes

Carolina A. Klein, MD, and Soniya Hirachan, MD

Delusional misidentification syndromes (DMSs) are complex psychotic phenomena that may be present in a variety of ways within the context of several neurological and psychiatric disorders. Since the first case of Capgras syndrome was described in 1923, various other syndromes have been identified, including Fregoli syndrome, intermetamorphosis, subjective doubles, reduplicative paramnesia, mirrored self, delusional companions, and clonal pluralization of the self. In this article, we review each of the different syndromes in definition and presentation, as well as the field's attempts at classifying them. We then describe their role in forensic psychiatry, particularly in regard to their potential as a marker of a particular subpopulation or of illness severity and their consideration in risk assessments of violence. A review of the literature was conducted for this purpose, and, although it was extended to include publications from over four decades, it revealed a paucity of research on DMSs.

J Am Acad Psychiatry Law 42:369–78, 2014

> Without wearing any mask we are conscious of, we have a special face for each friend.—Oliver Wendell Holmes

Few concepts in psychiatry can be as confusing as the delusional misidentification syndromes (DMSs). One goal in psychiatry is to achieve a better understanding of the self: who it is, how it is organized, and how it develops and reacts to others. DMSs introduce a multiplicity of aspects into this understanding of identities and relationships, adding to its inherent multifaceted complexity. They are fascinating because they are disruptions in what we consider the normal integrity of the self, and for forensic experts, interesting for how they help us understand entanglements between mental health and unlawful behavior.

Delusional misidentification syndromes are rare psychopathologic phenomena that may occur within the context of schizophrenia or affective or organic illnesses. They include Capgras syndrome, Fregoli syndrome, intermetamorphosis syndrome, syndrome of subjective doubles, mirrored self, delusional companions, and clonal pluralization of the self. Misidentification syndromes show a great degree of overlap and do not represent distinctive syndromes, nor can they be regarded as an expression of a particular disorder. Evidence suggests that one type of misidentification delusion may evolve into another type.[1] However, these syndromes merit distinct identification and therapeutic approaches because of their possible underlying disorders and their potential for dangerous behavior.[2] Furthermore, for forensic experts, they may be instrumental in assessments of risk and criminal responsibility.

In this article, we review the available literature regarding these syndromes. We also analyze DMSs and the forensic population, seeking any correlations between diagnosis of DMSs and other key concepts such as dangerousness or legal underpinnings. Finally, we attempt to describe guidelines for the clinical management of these patients, or for the incorporation of this psychopathology into forensic assessments.

Definitions and Classifications

DMSs all carry a common classic theme of one person being an exact likeness of another: the *sosie* or double. They can be distinguished as hypoidentifica-

Dr. Klein is Associate Program Director, Forensic Psychiatry Fellowship, Georgetown University Hospital, Washington D.C., and Chief Medical Officer, The Maia Institute, Alexandria, VA. Dr. Hirachan is Forensic Psychiatry Fellow, Western Psychiatric Institute and Clinic, University of Pittsburgh Medical Center, Pittsburgh, PA. Address correspondence to: Carolina A. Klein, MD, 2050 Ballenger Avenue, Suite 200, Alexandria, VA 22314. E-mail: cklein@maiainstitute.org.

Disclosures of financial or other potential conflicts of interest: None.

tions (Capgras syndrome) and hyperidentifications (the other syndromes).[3] Different terminologies and classifications have led to confusion in the past. Roessner[4] presented a classification that included two categories: one in which the object of the delusion is physically altered (or replaced) in the patient's mind, and a second in which the object is doubled, rather than replaced or transformed. Another proposed classification system using developmental and regressive understanding has also been proposed,[5] in which DMSs are divided into two main subgroups: relational misidentifications (misidentification of human relationships) and identical misidentifications (misidentifications of identity itself, which includes Capgras and Fregoli). Identical misidentifications are further subdivided into divided-identity type, unionized-identity type, and transformed-identity type. This typologic approach allows for developmental understanding and the application of regressive theories. Signer[6] proposed an extension of reverse types of misidentification syndromes, distinguished by alteration of the self rather than of others. Beyond definitions, the phenomena have sparked human interest throughout time, as reflected in stories and myths about doppelgangers, imposters, and clones. Some of these artistic examples emphasize the importance to human psychology of the identification of self and others and the potential ramifications of wrongful identification. We briefly review the concepts and psychopathology and offer a few illustrative examples.

Capgras syndrome was first described in 1923 by psychiatrists Joseph Capgras and Jean Reboul-Lachaux. It is the most prevalent of the delusional misidentification syndromes and is described as a disorder in which a person holds a delusion that an identical-looking impostor has replaced a friend, spouse, parent, or other close family member. One can imagine this syndrome in the performance by Donald Sutherland in the movie, *Invasion of the Body Snatchers*,[7] a remake of the original 1956 science fiction film in which humans are replaced by emotionless alien clones. Reverse Capgras syndrome refers to the psychological change of the self as opposed to others. A study[8] found that, of 30 subjects with this syndrome, most of the new identities were famous figures or others who were admired by the affected person. Most of the individuals also experienced a sudden belief or realization of having a new identity or of having rediscovered a pre-existing one.

Leopoldo Fregoli, an entertainer from the late 19th century, perfected a style of performance known as protean or quick-change. He could switch costumes and characters during his stage shows so rapidly that it was thought that several other Fregolis must have existed for his act to be possible. Fregoli syndrome is the delusional belief that one or more familiar persons, usually persecutors following the patient, repeatedly change their appearances (i.e., the same person assumes numerous different disguises).

Intermetamorphosis is a misidentification syndrome in which an individual has the erroneous belief that familiar persons have exchanged identities. In the syndrome of subjective doubles, patients believe that there are other persons who look like them, but that they have different traits and live different lives. This situation has been commonly depicted in movies, such as *The Sixth Day*,[9] where Arnold Schwarzenegger's character is cloned without his knowledge or consent, and in TV shows, such as *Battlestar Galactica*[10] and *Star Trek*,[11] where clones represent the main rivals to the shows' heroes. Mirrored-self misidentification involves the misperception that one's reflection in the mirror is a stranger. Individuals affected with the syndrome of delusional companions believe nonliving objects possess consciousness, can think independently, and feel emotion. The movie *Night at the Museum*[12] features objects exhibited at a museum that appear alive to the protagonist. Clonal pluralization of the self differs from the syndrome of subjective doubles, in that the patient believes that there are multiple copies of himself who are physically and psychologically similar to themselves. As an example, Ranjan and colleagues[13] reported a patient with schizophrenia who thought that there were triplicate copies of herself and others.

Further extensions of these core DMSs have also been described. Somatoparaphrenia is a subtype of asomatosognosia, in which patients also display delusional misidentification and confabulation. It also involves orbitofrontal dysfunction, which distinguishes it from asomatosognosia.[14] Reduplicative paramnesia is the belief that a place or location has been duplicated or relocated. This is the scenario in the movie *The Truman Show*,[15] where the protagonist finds that his world is actually a reality TV show set. Similarly, the concept of the physical world as an illusion has been depicted in *Vanilla Sky*,[16] *The Thirteenth Floor*,[17] and *The Matrix*.[18] Other extensions of DMS have been postulated to include lycanthropy,[19]

Ekbom syndrome, delusional hermaphroditism,[20] delusion of sexual transformation,[21] and the antichrist delusion.[22] DMSs have even been documented in the context of a *folie à deux* shared delusion of doubles.[23]

Etiology

Feinberg[24] recently described a comprehensive, multimodal, hierarchical model for understanding the neuropathologies of the self, which includes negative factors (defects or absence of neurological functions) and positive factors (productive, defensive, and motivational brain features). Neurobiological findings strongly support a structural basis for DMSs. Facial processing involves right ventromedial occipitotemporal regions and areas of prefrontal cortex via the uncinate fasciculus and limbothalamic pathways.[25] Other researchers[26] have suggested that impairment of facial recognition plays a role in the pathogenesis of DMSs. Lesions found in DMSs are usually bifrontal, right hemispheric, or both. A disconnection is observed between the frontal lobes and the right temporolimbic regions (hippocampus), which are necessary for reconciling information about self-identification of the person and his associated emotions.

Neuropsychological testing further suggests that misidentification delusions are associated with subtle abnormalities in facial recognition abilities and with nondominant cerebral compromise.[1] Underactivity in the perirhinal cortex seems to be responsible for loss of familiarity in Capgras syndrome, whereas overactivity seems to account for hyperfamiliarity, seen in the Fregoli, intermetamorphosis, and subjective doubles syndromes. Impaired connectivity between the right fusiform and right parahippocampal areas has also been implicated in deficits in visual memory recall, face recognition, and identification processes in these patients.

According to cognitive models, the dysfunction extends beyond facial recognition, whereby the person cannot be globally considered. The feeling of familiarity is absent because of the inability to integrate successive memories about a person along with episodic experiences, thus generating delusional doubles in accordance with the patient's needs or drives.[27]

Devinsky[28] postulated a dual mechanism: on the one hand, negative effects from the right hemisphere and frontal lobe dysfunction impair self-monitoring, ego boundaries, and attached emotional valence to familiar stimuli; on the other, the preserved left hemisphere areas exert a positive effect from release or overactivity, providing a narrator from the monitoring of self, memory, and reality. This effect leads to excessive and false explanations, or, because of the dual-category style of cognitive categorization, it leads to invention of a duplicate or impostor to resolve conflicting information. Politis and Loane[29] echoed this theory and highlighted a consensus that right and bifrontal lesions, as well as the cognitive dissonance associated with impairments in memory, visuospatial abilities and conceptual integration, are common factors in DMSs (reduplicative paramnesia in particular).

Psychodynamic models may also be used to understand the phenomenology and subjective experience of DMSs. The central theme revolves around defining identity and its multifaceted dimensions. Consideration must be given to the introjected sense of self and the capacity to hold a reflective and observing ego. Conversely, the pathological expression that would give way to a DMS is the experience of depersonalization. Other ego functions may be compromised, including the loss of motivation and self-initiated drives. DMSs may also be conceptualized as defensive structures, whereby negative aspects of the self are split off and projected into an external (and targetable) other.

From a developmental perspective, an arrested early development or regression process may be identified. Misidentification phenomena are manifestations of defense mechanisms of splitting and projection.[30] An aspect of the internalized self or object representation to whom negative emotions are attached are split off from the self and projected externally, onto a different identity. These mechanisms, being primitive in nature, can also be explained by regression theory.[31] When higher cerebral functioning is affected, its compromise results in reactivation of primitive modes of thinking characterized by the theme of doubles and dualisms, also found in myths, primitive religion, and literature.

Other variants of this theory propose that deep regression reactivates a developmental stage before the establishment of object constancy, where there is a splitting of objects into all good or all bad and an absence of self–object differentiation. This effect is consistent with other elements of relations theory, in which a primitive self is unable to establish a trusting relationship with a cohesive other, described as the

Kleinian paranoid–schizoid position. Along these lines, others highlight the inability to attribute uniqueness to the self and surrounding people,[32] despite positive and negative attributes.

Altered affective response toward others may also be at play. Intolerable affective ambivalence toward others may be neutralized through the imagined existence of doubles.[33] For example, individuals with Capgras syndrome may harbor anger or envy toward a close relative. Denial is then used to make this emotion tolerable and free of guilt for the delusional person. If this mechanism becomes insufficient, the person may then split the object, attributing only positive feelings to the original object and only negative feelings to the delusionally altered identity. Projection is involved in directing the negative emotions at the object without experiencing significant internal conflicts. Projective identification becomes possible when the patient in turn experiences positive feelings coming from the delusionally altered identity. The affected individual fears others as hostile and may even strike preemptively in response to it.[34]

Others have placed emphasis on the role of language and narrative in the manifestation of DMSs. They propose difficulties in the self-reflexive property of the human mental functioning and the first-person linguistic expression of human experience,[35] with an aberrant semantic processing of identity.

Diagnosis, Treatment, and Prognosis

Ongoing discussion persists within the field pertaining to whether DMSs are neurologic or psychiatric syndromes, whether they are a feature of schizophrenia or delusional disorder, or whether they constitute a distinct phenomenon described in the Diagnostic and Statistical Manual of Mental Disorders.

Several treatment approaches have been described. Treatment of co-occurring psychiatric, substance use, or medical disorder is required.[36–38] Antipsychotics[39] are often used. According to case reports, DMSs have responded favorably to olanzapine,[40] sulpiride and trifluoperazine,[41] clorazepate,[42] and pimozide.[43] Antidepressants may be useful regardless of whether the primary diagnosis is a mood disorder.[44] If the DMS is associated with mania, lithium is a mainstay.[45,46] Group therapy has also been proposed.[47]

Prevalence and Relevance in the Psychiatric and Forensic Population

DMSs have been associated with both focal and diffuse neurologic conditions, such as hypothyroidism,[48] right hemispheric stroke,[49] multiple sclerosis,[50] and dementia. They have also been associated or identified with psychiatric conditions, especially schizophrenia, Alzheimer's disease (AD),[51] and PTSD.[52] Research has shown that within psychotic illnesses, paranoid schizophrenia seems to be the most common diagnosis in patients with DMSs.[53,54]

Prevalence in all psychiatric inpatients ranges from 1.3 to 4.1 percent.[55] A study in Turkey at a university hospital inpatient setting showed the 5-year prevalence rate of Capgras syndrome to be 1.3 percent (1.8% for females and 0.9% for males).[56] However, DMSs occur more frequently than previously thought.[57] According to Dohn and Crews,[58] the prevalence of DMSs among patients identified as schizophrenic is 15 percent. They postulated an estimated prevalence of 0.12 percent in the general population for Capgras syndrome. A study in patients with AD demonstrated a prevalence between 2 and 30 percent.[59] In a different study, DMS was identified in 15.8 percent of cases of AD, 16.6 percent of patients with Lewy body dementia, and 8.3 percent of individuals with semantic dementia.[60] With regard to inpatient prevalence, a survey was conducted of all admissions to a locked psychiatric inpatient unit in the Boston metropolitan area from April 1983 to June 1984. Twenty-six (3.1%) of 835 patients admitted to the unit met the criteria for DMSs.[61]

Prevalence specifically within the forensic population is unknown. Some case reports have been published and will be discussed below.

Is There an Association Between DMS Phenomenology and Violence?

No studies have looked at the association between DMSs and legal history or type of offense. Data correlating criminal behavior and DMSs are limited mostly to case reports or, at best, to descriptive retrospective studies with a low number of study subjects. To assess this correlation accurately, one would need a reliable diagnosis of the phenomena and the legal history, which are often unavailable. To our knowledge, research gathering such data has not

been conducted to date. However, some literature addressing this association is currently available.

Research has shown that DMS patients view the misidentified person with suspicion and hostility,[62] which may contribute to a mounting paranoia and physical aggression in the form of pre-emptive self-defense. Silva *et al.*[63] postulated that delusional cognition drives the affected individual to construct a narrative in which the misidentified object is conceived as a person for whom biographical history radically departs from a stable, good construct (the original identity), only to be replaced by a bad object, which is then considered authentic. In their research, they found that these patients become aggressive toward the misidentified objects, because they perceive the newly constructed object as threatening to their own welfare. In their book, Silva *et al.*[47] explain how such disturbed thinking can result in dangerous/aggressive acting out toward individuals who are at risk of being harmed by such patients. Of their sample of six patients, all exhibited verbal aggression, and five became physically aggressive toward delusionally misidentified objects.

Another study of 82 subjects with DMSs defined violence as verbal threats or physical violence directly associated with a misidentification delusion. Fifty of the 82 patients had attacked someone else, the most common victims being parents.[64] In another study by Silva *et al.*,[65] of 29 patients with DMSs, 16 had threatened others without acting on the threats, whereas 13 became physically assaultive in connection with their misidentification syndromes. In yet another study, Silva *et al.*[66] also found that dangerous patients with DMSs were less likely to use weapons than were their non-DMS counterparts.

Aggression or physical assault may escalate to the level of murder, as described in the cases above. Silva *et al.* also reported the case of a homicide associated with delusional misidentification.[67] Capgras syndrome has been described in incidents of parricide.[68] In one review of the literature, delusional misidentification cases leading to homicide usually involved more than one delusionally misidentified object; in four of nine cases of DMS-associated homicide, there was a prior history of serious physical violence directed at other delusionally misidentified objects.[67] In a French study, the highest percentage of homicide occurred in patients with paranoid schizophrenia, and their delusions of misidentification had usually been present for an extended period before the homicidal act.[69]

Special consideration may be given to cases where a DMS involves a child, especially as the object of a delusion. Silva *et al.*[70] explored these cases more than 20 years ago, finding that they may involve mechanisms of aggression that differ from paranoid self-defense. They described how DMSs have been found in some folklore and regional legends that suggest that aggression might rid the impostor and return the authentic personality. In one Swedish tale, a woman who believed that her child was an impostor was instructed to put the child in an oven to recover her original baby. Although these actions may not literally translate into reality, the concept of physically extricating the imposter to regain the authentic persona remains. Furthermore, DMS has been described in the context of puerperal depression with psychotic features, where cases of Capgras[71] and Fregoli[72] syndromes have been reported (a phenomenon historically called changeling). When the child is the entity that is believed to have been replaced, the risk of aggression toward the child increases, as the mother perceives that violence may be the only means of freeing the original child from the impostor.

Rather than simply investigating the relationship between two entities (DMS and violence), we must ask a more difficult question: across (or within) diagnostic groups, are patients with misidentification delusions more likely to behave violently than comparable patients without such delusions?[73] De Pauw and Szulecka[41] postulated that morbid suspiciousness, hostility, and discord, as well as previous aggressive behavior and delusions that focus on interpersonal relationships, rather than on locations or inanimate objects, render DMS patients particularly prone to attacking the subjects of their suspicions. Nestor and coworkers[74] found a higher incidence of Capgras syndrome in forensic psychiatric patients with psychotic disorders who had committed homicide or other severely violent acts, than in less violent patients.

Risk Factors for Violence in DMS

Violence in DMSs has been determined to be multifactorial, and several of these factors have to be taken into consideration to gauge adequately the risk of violent enactment of DMSs. These patients seem to have a significant history of physical aggression,

independent of their delusional misidentification.[66] Increased risk of violence in DMSs may be derived from their high-risk triad of highly valued beliefs, negative affects, and identified targets (often a close relative or attachment figure). Anger due to delusion is a key factor that explains the relationship between violence and acute psychosis,[75] and close relatives or attachment figures have been established as significantly higher risk targets.

Based on four case reports, De Pauw and Szulecka[41] identified certain factors that may have contributed to aggression in each of their reported patients. These include structural or metabolic brain injury, subjectively experienced salient historical events (associated with negative emotions), low intelligence, poor social and occupational skills, primary psychiatric pathology, and pre-existing personality constructs. It is important to note that these factors were significant for each of these cases and are not offered as generalized risk factors for violence.

Specifically regarding Capgras syndrome, a review study[76] looked at demographic and clinical features that may have contributed to an increased risk for violence in cases of Capgras syndrome involving assault. Common factors noted included men with long-standing delusions, a history of aggressive behavior, diagnosis of schizophrenia, and comorbid substance use. Persecutory delusions and anger toward the misidentified person were often present, and command hallucinations and sexual preoccupation were at times also present. Negative syndrome features, such as social withdrawal and blunted affect, were noted. The victim was usually a cohabiting family member, and the violence was usually well planned.

Silva *et al.*[65] also found that males represent a higher proportion (70%) of patients with DMSs exhibiting aggressive behavior. Other factors that increase the likelihood of aggression include specific delusions, such as erotomania[77,78] or delusional jealousy.[79] In a three-patient case report,[80] the authors argued that the degree of threat perceived by the patient from the delusionally misidentified object is the most important factor in determining the patient's response to the delusions. The authors of another case report of three patients agreed with that principal factor and added that impulsivity and dissociation may also play a significant role.[81] Case reports suggest that alcohol and substance intoxication facilitate patients' acting on the delusions.[82,83] Thompson and Swan[84] described 2 subjects with Capgras syndrome who, after heavy drinking, committed serious acts of violence toward family members, who had become subjects of delusional misidentification. Although substances such as alcohol may facilitate the enactment, they do not account for the genesis of the delusional beliefs.

DMS as a Marker of Illness Severity

Given the potential for severe violence, presumed to result as the endpoint of the development of the illness, we attempted to examine whether the existence of DMS denotes a particular subgroup of mentally ill patients. To our knowledge, no studies have been conducted that analyze the relationship between DMS and markers of illness severity, such as duration of illness, age at onset, Global Assessment of Functioning (GAF) or other functionality markers, total number of hospitalizations, or length of stay. With regard to illness severity, a study by Silva and colleagues[47] of 25 subjects noted that the Brief Psychiatric Rating Scale (BPRS) total score and grandiosity score were higher in DMS subjects than non-DMS subjects. A study comparing neuropsychological functions found no statistically significant differences in neuropsychological functions in schizophrenic patients with and without DMS.[64] However, the study involved only 22 patients and did not include indicators of psychiatric illness severity. To our knowledge at the time of this writing, no valid rating scales or other structured clinical or forensic assessments are available for specific use in patients with DMSs.

There are no formal guidelines for delineating the standards of assessment of these syndromes. Furthermore, there are no systematic studies on treatment requirements of these patients when compared with the more general or forensic population, including the use of intensive precautions and monitoring; the number of failed medication trials; or the use of complex therapies, such as polypharmaceutical regimens, clozapine, or electroconvulsive treatments (ECT). The literature does offer some retrospective or descriptive observations regarding treatment. Zanker[85] states that symptoms of DMS are very refractory to treatment despite various neuroleptic therapies.

In a meta-analysis, Silva *et al.*[86] examined 104 misidentification syndrome cases published between 1957 and 1994, where information regarding treatment with antipsychotic medications was available:

70 patients showed improvement, whereas 34 did not. Christodoulou[87] reported in his research on 20 patients with DMSs that the syndromes failed to remit in 7 of the 20 cases, and in the others, remission occurred either synchronously with or later than the remission of the underlying psychosis. Other isolated case reports suggest that patients with DMSs who have an underlying organic etiology show remission on most occasions.[88,89]

More accurate data gathering is rendered difficult, given that these syndromes are often underdiagnosed as distinct manifestations of psychoses. There are no guidelines currently for their assessment and treatment. Some authors have proposed manners of furthering exploration. Hillers and others[26] propose that there is a common underlying neuropsychiatric mechanism to these disorders; they therefore propose a neuropsychological battery to assess Capgras syndrome. Some researchers[90] have used hypnosis to recreate mirrored-self misidentification, thereby gaining insight into delusional beliefs.

More recently, event-related potentials (ERPs), especially the auditory P300 component, have constituted a useful tool for exploring brain–behavior relations. DMSs are thought to be related to dissociation between recognition and identification processes. Working memory is considered responsible for the integration and online manipulation of information, and ERPs and subsequent measured P300 wave forms are considered an index of the online updating of working memory. A study[91] found P300 amplitude in prefrontal areas to be significantly reduced in patients with schizophrenia with DMS compared with those with schizophrenia without DMS and controls. They also found P300 latency in the central midline brain region to be significantly prolonged in the DMS subgroup. The methods discussed herein may offer future ways of identifying and studying these patients.

Specific Areas of Concern for Forensic Assessments

Most of the research cited above focused on the role of identifying DMS for the purposes of risk assessment of violent behavior. However, added consideration must be given to other areas in forensic assessments where DMSs may play a significant role given their impact on rationality, understanding of wrongfulness, ability to control voluntary acts, and culpable mental state. In the criminal arena, a DMS may represent the essence of incompetency, whether the DMS applies to the self or others involved in the crime or in the legal proceeding. The presence of a DMS may also raise a particular argument in criminal-responsibility disputes, in that the cognitive prong would be inherently impaired. The question of criminal responsibility could also involve the volitional prong in some jurisdictions, and the assessment of whether the patient was able to conform his conduct, even if he believed the victim (or himself) to be a different identity altogether. In states without an insanity defense, the presence of DMS perhaps should be considered for the purposes of establishing *mens rea* and willingness to commit a criminal act in a knowing and voluntary manner. Finally, a DMS as an element of severe illness could be considered a mitigating factor, or a relevant factor in determining sentencing needs (especially for therapeutic considerations).

In the civil arena, identification of DMS toward a child could be a center point of establishing parental capacities and custodial rights. Matuszak and Parra[92] described a case in which child custody was disputed because the mother believed that her daughter had been replaced by an impostor. DMS might also be attached to a caregiver or guardian of an adult patient, directly affecting the patient's welfare. The presence of DMS could have a direct association with medical decision-making capacity should the patient believe, for example, that his doctor has been replaced by an impostor. Moreover, it becomes particularly relevant in matters of testamentary capacity, especially given the strong correlation between DMS and structural or neurological pathologies common in the elderly.

DMSs have, in fact, been a key element in several legal cases. In *People v. Singer*[93], defendant Jerry Singer was found guilty but mentally ill in the murder of three people by stabbing. Mr. Singer perceived of three people who ran the apartment building where he lived as having been replaced by clones and operating under the influence of psychics who were intent on chasing him out of his own apartment. Both psychiatrists who provided expert witness testimony found the defendant to have schizophrenia and Capgras syndrome, and opined that he was insane at the time of the killings.

Joshua Hoge was found not guilty by reason of insanity in the stabbing and murder of his mother and stepbrother. Mr. Hoge had paranoid schizophre-

nia, and Capgras syndrome led him to believe that imposters had replaced them. In *Re: the Estate of Pamela Kissinger v. Joshua Hoge*[94] the appellate court determined that his insanity acquittal did not make the state's slayer statute inapplicable to him, as was argued in civil court.

Conclusions

DMSs are complex psychotic phenomena that may arise in the context of neurological or psychiatric pathology and may be more prevalent in such populations than originally thought. Despite this possible underestimation, there is a paucity of research surrounding DMSs, a dearth of recent scientific literature on the subject, and very limited analysis of their impact on forensic psychiatry practice. DMSs may be a hallmark of a particular subgroup within the forensic population correlated with particular criminal behaviors or of illness severity in the general psychiatric population.

As we have explored in this article, DMSs are relevant to forensic psychiatry for several reasons. They may represent an independent risk variable and should be incorporated into dangerousness assessments. There might be a significant correlation with criminal behavior. They may also be a nuclear element in criminal insanity, civil and criminal competencies, and relationships involving the welfare of others or of the patient.

There are no formal guidelines currently delineating the standards for assessment or treatment of these syndromes. This lack of guidance becomes particularly relevant in establishing standard of care and appropriate practice. Following our findings in this review, we propose that DMSs should be directly and specifically screened for identification in the psychiatric assessment, given their correlation with neurological injury and morbidity as well as the increased risk of violence. Once identified, organic, structural, and metabolic conditions must be ruled out and adequately treated. Comorbid substance use must also be identified. Routine blood work should address metabolic parameters of hepatic and renal function (to identify causes or indicators of delirium), thyroid function, and, when pertinent, levels of drugs such as lithium. Brain imaging may help identify some neurological pathologies, such as stroke and multiple sclerosis. Neuropsychological testing can be conducted, as deemed necessary.

Finally, we emphasize the need for more rigorous research on these syndromes, to advance the understanding of their psychopathology, establish accurate incidence and prevalence rates, and correlate them with clinical indicators of severity or dangerousness. In the forensic arena, it would be interesting to examine the importance of the relationship with the persons who are the focus of abnormal beliefs or perceptions, especially in situations of odd or bizarre attachments, and the relationship with stalking. Systematic study of the variables involved in DMSs is crucial, perhaps best achieved through a case-control study that compares patients with DMS to similar patients who do not present such syndrome. In a large state hospital setting, for example, patients identified as having a DMS could be compared against those with no DMS in terms of violence, length of hospital stay, number of failed medication trials, and severity of criminal activity (if pertinent), among other areas.

References

1. Silva JA, Leong GB, Wine DB, *et al:* Evolving misidentification syndromes and facial recognition deficits. Can J Psychiatry 37: 574–6, 1992
2. Förstl H, Almeida OP, Owen AM, *et al:* Psychiatric, neurological and medical aspects of misidentification syndromes: a review of 260 cases. Psychol Med. 21:905–10, 1991
3. Christodoulou GN, Margariti M, Kontaxakis VP, *et al*: The delusional misidentification syndromes: strange, fascinating, and instructive. Curr Psychiatry Rep 11:185–9, 2009
4. Roessner V: A new classification of the delusional misidentification syndromes. Psychopathology 35:3–7, 2002
5. Nishida H, Shinbo Y, Kuramitsu M, *et al:* One possible classification of the delusional misidentification syndromes and its developmental, regressive understandings (in Japanese). Seishin Shinkeigaky Zasshi 98:533–54, 1996
6. Signer SF: Capgras' syndrome: the delusion of substitution. J Clin Psychiatry 48:147–50, 1987
7. Invasion of the Body Snatchers. Directed by P. Kaufman. Screenplay by W. D. Richter. United Artists, 1978
8. Silva J, Leong G: Delusions of psychological change of the self. Psychopathology 27:285–90, 1994
9. The Sixth Day. Directed by R. Spottiswoode. Written by C. and M. Wibberly. Columbia Pictures, 2000
10. Battlestar Galactica. TV series based on the book by G. A. Larsen. Developed by R. Moore. Skye Television, 2004–2009
11. Star Trek. TV Series. Created by G. Rodenberry. Desilu Productions, 1966–1967 Paramount Pictures, 1968–1969
12. Night at the Museum. Directed by S. Levy. Screenplay by T. Lennon and R. B. Garant. 20th Century Fox, 2006
13. Ranjan S, Chandra PS, Gupta AK, *et al*: Clonal pluralization of self, relatives, and others. Psychopathology 40:465–7, 2007
14. Feinberg TE, Venneri A, Simone AM, *et al:* The neuroanatomy of asomatosognosia and somatoparaphrenia. J Neurol Neurosurg Psychiatry 81:276–81, 2010
15. The Truman Show. Directed by P. Weir. Written by A. Niccol. Paramount Pictures, 1998

16. Vanilla Sky. Directed and Screenplay by C. Crowe. Paramount Pictures, 2001
17. The Thirteenth Floor. Directed by J. Rusnak. Screenplay by J. Rusnak and R. Centeno-Rodriguez. Columbia Pictures, 1999
18. The Matrix. Directed by J. Silver. The Wachowski Brothers. Warner Bros. Pictures, 1999
19. Silva JA, Leong GB: Lycanthropy and delusional misidentification. Acta Psychiatr Scand 111:162, 2005
20. Jagadheesan K, Sandil R, Nizamie SH: Delusional hermaphroditism: a rare variant of delusional misidentification syndrome. Psychopathology 35:52–4, 2002
21. Silva JA, Leong GB, Penny G: Delusion of sexual transformation as delusional misidentification syndrome. Can J Psychiatry 43: 956–7, 1998
22. Silva JA, Dassori A, Leong GB: The antichrist delusion as a delusional misidentification syndrome of the self. Can J Psychiatry 42:90, 1997
23. Christodoulou GN, Margariti MM, Malliaras DE, *et al:* Shared delusions of doubles. J Neurol Neurosurg Psychiatry 58:499–501, 1995
24. Todd E. Feinberg: Neuropathologies of the self and the right hemisphere: a window into productive personal pathologies. Front Hum Neurosci 7:472, 2013
25. Scherf KS, Thomas C, Doyle J, *et al*: Emerging structure-function relations in the developing face processing system. Cereb Cortex 2013 June 13 (Epub ahead of print); doi:10.1093/cercor/bht152
26. Hillers RR, Madoz-Gurpide A, Tirapu UJ: Propuesta de una batería neuropsicológica para la exploración del síndrome de Capgras [Capgras syndrome: a proposal of neuropsychological battery for assessment]. Rev Esp Geriatr Gerontol 46:275–80, 2011
27. Madoz-Gurpide A, Hillers-Rodríguez R: Capgras delusion: a review of aetiological theories. Rev Neurol 50:420–30, 2010
28. Devinsky O: Delusional misidentifications and duplications: right brain lesions, left brain delusions. Neurology 72:80–87, 2009
29. Politis M, Loane C: Reduplicative paramnesia: a review. Psychopathology 45:337–43, 2012
30. Warchala A, Krupka-Matuszczyk I: Zespół misidentification urojenia [Delusional misidentification syndrome]. Wiad Lek 59: 702–6, 2006
31. Sinkman AM: The Capgras delusion: a critique of its psychodynamic theories. Am J Psychother 37:428–38, 1983
32. Margariti MM, Kontaxakis VP: Approaching delusional misidentification syndromes as a disorder of the sense of uniqueness. Psychopathology 39:261–8, 2006
33. O'Reilly R, Malhotra L: Capgras syndrome: an unusual case and discussion of psychodynamic factors. Br J Psychiatry 151:263–5, 1987
34. Kernberg, O.F: Neurosis, Psychosis and the borderline states, in Comprehensive Textbook of Psychiatry Vol. IV (vol 4, ed 4). Edited by Kaplan H and Sadock BJ. Baltimore: Williams and Wilkins, 1985
35. Rodrigues AC, Banzato CE: Delusional misidentification syndrome: why such nosologic challenge remains intractable. Psychopathology 39:296–302, 2006
36. Schlesinger LB (ed): Explorations in Criminal Psychopathology. Springfield, IL: Charles C. Thomas Publisher, 2007
37. Khanna R, Khanna N: Delusions of Substitution and diabetes mellitus. Int J Psychiatry Med 21;105, 1991
38. Madakasira S, Hall TB: Capgras syndrome in a patient with myxedema. Am J Psychiatry 138:1506, 1981
39. Christodoulou GN: Treatment of the "syndrome of doubles." Acta Psychiatr Belg 77:254–9, 1977
40. Lykouras L, Typaldou M, Gournelis R *et al:* Coexistence of Capgras and Fregoli syndromes in a single patient: clinical, neuroimaging and neuropsychological findings. Eur Psychiatry 17: 234–5, 2002
41. De Pauw KW, Szulecka TK: Dangerous delusions: violence and the misidentification syndromes. Br J Psychiatry 152:91–6, 1988
42. Joseph AB: Delusional misidentification of the Capgras and intermetamorphosis types responding to clorazepate: a case report. Acta Psychiatr Scand 75:330–2, 1987
43. Tueth MJ, Cheong JA: Successful treatment with pimozide of Capgras syndrome in an elderly male. J Geriatr Psychiatry Neurol 5:217–9, 1992
44. Christodoulou GN: Delusional hyper-identifications of the Fregoli type: organic pathogenic contributors. Acta Psychiatr Scand 54:305, 1976
45. Wilcox J, Wazir, R: The Capgras symptom and nondominant cerebral dysfunction. J Clin Psychiatry 44:70, 1983
46. DriscollR, Chithiramohan R., Brockman B: Capgras syndrome, mania, and delusionally motivated assaults. J Forensic Psychiatry 2:49, 1991
47. Silva J, Leong, G, Weinstock, R. Misidentification syndromes, aggression and forensic issues, in Explorations in Criminal Psychopathology: Clinical Syndromes with Forensic Implications. Springfield, IL: Charles C. Thomas Publisher, 1996, pp 33–49
48. Nishihara K, Kinoshita H, Kurotaki N, *et al:* Could subclinical hypothyroidism cause periodic catatonia with delusional misidentification syndrome? Psychiatry Clin Neurosci 64:338, 2010
49. Hoffmann M: Isolated right temporal lobe stroke patients present with Geshwind Gestaut syndrome, frontal network syndrome and delusional misidentification syndromes. Behav Neurol 20:83–9, 2008
50. Sidoti V, Lorusso L: Multiple sclerosis and Capgras' syndrome. Clin Neurol Neurosurg 109:786–7, 2007
51. Silva JA, Leong GB, Weinstock R, *et al:* Delusional misidentification and aggression in Alzheimer's disease. J Forensic Sci 46: 581–5, 2001
52. Silva JA, Leong GB, Harry BE, *et al:* Dangerous misidentification of people due to flashback phenomena in posttraumatic stress disorder. J Forensic Sci 43:1107–11, 1998
53. Berson RJ: Capgras' syndrome. Am J Psychiatry 140:969–78, 1983
54. Kimura S: Review of 106 cases with syndrome of Capgras. Bibl Psychiatr 164:121–30, 1986
55. Silva JA, Leong GB, Weinstock R, *et al*: Delusional misidentification syndromes and dangerousness. Psychopathology 27: 215–9, 1994
56. Karatas G, Zeren T, Ozpoyraz N: The prevalence of Capgras syndrome in a university hospital setting. Acta Neuropsychiatr 15:290–5, 2003
57. Kirov G, Jones P, Lewis SW: Prevalence of delusional misidentificaion syndromes. Psychopathology 27, 2148, 1994
58. Dohn HH, Crews EL: Capgras syndrome: a literature review and cases series. Hillside J Clin Psychiatry 8:56–74, 1986
59. Harwood DG, Barker WW, Ownby RL, *et al*: Prevalence and correlates of Capgras syndrome in Alzheimer's disease. Int J Geriatr Psychiatry 14:415–20, 1999
60. Harciarek M, Kertesz A: The prevalence of misidentification syndromes in neurodegenerative syndromes in neurodegenerative diseases. Alzheimer Dis Assoc Disord 22:163–9, 2008
61. Joseph AB: Observations on the epidemiology of the delusional misidentification syndromes in the Boston metropolitan area: April 1983–June 1984. Psychopathology 27:150–3, 1994
62. Enoch M. Trethowan WH, Barker JC: Some Uncommon Psychiatric Syndromes. Bristol, UK: Wright, 1967

63. Silva JA, Leong GB, Garza-Trevino ES, *et al:* A cognitive model of dangerous delusional misidentification syndromes. J Forensic Sci 39:1455–67, 1994
64. Lykouras L, Typaldou M, Mourtzouchou P, *et al:* Neuropsychological relationships in paranoid schizophrenia with and without delusional misidentification syndromes: a comparative study. Prog Neuropsychopharmacol Biol Psychiatry 32:1445–8, 2008
65. Silva JA, Gregory B, Leong MD, *et al:* The dangerousness of persons with misidentification syndromes. Bull Am Acad Psychiatry Law 20:77–86 1992
66. Silva JA, Leong GB, Weinstock R, *et al*: Psychiatric factors associated with dangerous misidentification delusions. Bull Am Acad Psychiatry Law 23:53–61, 1995
67. Silva J, Harry B, Leong G, *et al:* Dangerous delusional misidentification and homicide. J Forensic Sci 41:641–4, 1996
68. Bourget D, Gagné P, Labelle ME: Parricide: A comparative study of matricide versus patricide. J Am Acad Psychiatry Law 35:306–12, 2007
69. Benezech M, Bourgeois M, Yesavage J: Violence in the mentally ill: a study of 547 patients at a French hospital for the criminally insane. J Nerv Ment Dis 168:698–700, 1980
70. Silva J, Sharma K, Leong G, *et al:* Dangerousness of the delusional misidentification of children. J Forensic Sci 37:830–8, 1992
71. De Leo D, Galligioni S, Magni G: A case of Capgras delusion presenting as a postpartum psychosis. J Clin Psychiatry 46:242–3, 1985
72. O'Sullivan D, Dean C: The Fregoli syndrome and puerperal psychosis. Br J Psychiatry 159:274–7, 1991
73. Dinwiddie S, Yutzie S: Dangerous delusions? Misidentification syndromes and professional negligence. Bull Am Acad Psychiatry Law 21:513–21, 1993
74. Nestor PG, Haycock J, Doiron S, *et al:* Lethal violence and psychosis: a clinical profile. Bull Am Acad Psychiatry Law 23:331–41, 1995
75. Coid JW, Ullrich S, Kallis C, *et al:* The relationship between delusions and violence: findings from the East London first episode psychosis study. JAMA Psychiatry 70:465–71, 2013
76. Bourget D. Whitehurst L: Capgras syndrome: a review of the neurophysiological correlates and presenting clinical features in cases involving physical violence. Can J Psychiatry 49:719–25, 2004
77. Hintzen AK, Wilhelm-Gobling C, Garlipp P: Combined delusional syndrome in a patient with schizophrenia: erotomania, delusional misidentification syndrome, folie a deux and nihilistic delusion. Ger J Psychiatry 13:96–9, 2010
78. Goldstein RL, Laskin AM: De Clerambault's syndrome and claims of psychiatric malpractice. J Forensic Sci 47:852–5, 2002
79. Silva JA, Ferrari MM, Leong GB, *et al*: The dangerousness of persons with delusional jealousy. J Am Acad Psychiatry Law. 26: 607–23, 1998
80. Aziz MA, Razik GN, Donn JE: Dangerousness and management of delusional misidentification syndrome. Psychopathology 38: 97–102, 2005
81. Carabellese F, Rocca G, Candelli C, *et al*: Mental illness, violence, and delusional misidentification: the role of Capgras' syndrome in matricide. J Forensic & Legal Med 21:9–13, 2014
82. Aziz MA, Razi KGN, Donn JE: Dangerousness and management of delusional misidentification syndromes. J Forensic Sci 42: 670–4, 1997
83. Atta K, Forlenza N, Gujski M, *et al:* Delusional misidentification syndromes separate disorders or unusual presentations of existing DSM-IV categories? Psychiatry (Edgmont) 3:56–61, 2006
84. Thompson AE, Swan M: Capgras' syndrome presenting with violence following heavy drinking. Br J of Psychiatry 162:692–4, 1993
85. Zanker S: Chronische und feuerfesten Fregoli Syndrom [Chronic and refractory Fregoli syndrome]. Psychiatr Prax 27:40–1, 2000
86. Silva JA, Leong GB, Miller AL: Delusional misidentification syndromes: drug treatment options. CNS Drugs 5:89–102, 1996
87. Christodoulou GN: Course and prognosis of the syndrome of doubles. J Nerv Ment Dis 166:68–72, 1978
88. Edson JA, Reolon AP, Sanches A: Report of seven neurological patients with misidentification syndrome. Available at: http://www.einstein.br/biblioteca/artigos/Vol2Num4/Report%20of%20seven%20neurological_02.pdf. Last accessed January 15, 2014
89. Collins MN, Hawthorne ME, Gribbin N, *et al*: Capgras' syndrome with organic disorders. Postgrad Med J. 66:1064–7, 1990
90. Barnier AJ, Cox RE, Connors M, *et al:* A stranger in the looking glass: developing and challenging a hypnotic mirrored-self misidentification delusion. Int J Clin Exp Hypn 59:1–26, 2011
91. Papageourgiou C, Lykouras L, Alevizos B, *et al:* Psychophysiological differences in schizophrenics with and without delusional misidentification syndromes: a P300 study. Prog Neuropsychopharmacol Biol Psychiatry 29:593–601, 2005
92. Matuszak J, Parra M. That's not my child: a case of Capgras syndrome. Psychiatric Times, 2011. Available at: http://www.psychiatrictimes.com/articles/that%E2%80%99s-not-my-child-case-capgras-syndrome. Accessed January 14, 2014
93. *People v. Singer*. 628 N.E.2d 592 (Ill. App. Ct. 1993)
94. In re Estate of Kissinger, 206 P.3d 665 (Wash. 2009)