

## A New Variant of "Subjective" Delusional Misidentification Associated with Aggression

**REFERENCE:** Silva JA, Leong GB, Rhodes LJ, Weinstock R. A new variant of "Subjective" delusional misidentification associated with aggression. *J Forensic Sci* 1997;42(3):406-410.

**ABSTRACT:** Delusional misidentification syndromes are psychotic conditions in which the affected individual experiences delusions of radical change concerning the identity of others and/or of the self. These syndromes may lead to aggression, including serious violence toward others. In this article, we describe and analyze in detail an aggressive individual who suffered from a delusion that physical and psychological replicas of himself existed. We specifically analyze the link between the patient's subjective misidentification delusion and his resulting aggression. Both the roles of phenomenology and biology of delusional misidentification are evaluated as potential contributors of aggression.

**KEYWORDS:** forensic science, forensic psychiatry, delusional misidentification, aggression, violence, psychosis, visual perception, chromosomal abnormalities

In 1923, Capgras and Reboul-Lachaux described a case of a woman who believed that physical replicas of her relatives, neighbors, and others existed in her environment (1). This presentation of delusional misidentification has come to be known as the syndrome of doubles or Capgras syndrome (2-4). Although not as well appreciated, Capgras and Reboul-Lachaux also noted that their patient also believed in the existence of physical replicas of herself (1,4). In 1978, Christodoulou proposed that the delusion of doubles of one's self be considered a specific delusional misidentification syndrome (DMS) and called it the syndrome of subjective doubles (5). The syndrome of subjective doubles also appears to have another important characteristic, namely that the alleged physical double of the delusional patient, appears to have a psychological identity different from that of the patient (5-7).

The causes of DMS's are complex and poorly understood. Nonetheless, it is known that many cases of DMS's, including subjective delusional misidentification, are associated with mild to severe biological abnormalities (8-12), especially those associated with non-dominant hemispheric dysfunction (8,10,13).

<sup>1</sup> Associate professor of psychiatry, University of Texas Health Science Center at San Antonio; and staff psychiatrist, South Texas Veterans Health Care System, San Antonio, Texas.

<sup>2</sup> Associate professor of clinical psychiatry, University of Missouri-Columbia School of Medicine; and chief of psychiatry, Harry S. Truman Memorial Veterans Hospital, Columbia, Missouri.

<sup>3</sup> Assistant professor of psychiatry, University of Texas Health Science Center at San Antonio, San Antonio, Texas.

<sup>4</sup> Clinical professor of psychiatry, UCLA School of Medicine; and staff psychiatrist, West Los Angeles Veterans Affairs Medical Center, Los Angeles, California.

Received 6 June 1996; accepted 9 Sept. 1996.

In this article, we report a case of the syndrome of subjective misidentification associated with violent behaviors that was also unusual in that the affected patient conceptualized his replica as not only physically but also psychologically identical to the original person. Phenomenological and biological factors that may have some relevance for understanding the present case will be discussed.

### Case History

Mr. A, a 43 year-old divorced right-handed male, was psychiatrically hospitalized for worsening paranoid delusions and visual and auditory hallucinations. Mr. A believed that extraterrestrial aliens had constructed people via surgical techniques, who looked exactly like him and with minds identical to his own. At times, he stated that he was not sure if he himself was a replica of the original. During such times, he would think that perhaps the original Mr. A had been killed by the extraterrestrials. Mr. A believed that his mind, which was simultaneously located in his brain as well as in his replicas, enabled him to experience the "other worlds" in which his physical replicas were located. He claimed that his robotic nature enabled him to be resistant to pain. He also believed that he and his replicas were partially biologic and partially robotic entities that were designed as fighting machines utilized in wars. Intermittently he endorsed having feelings of depersonalization and derealization. Mr. A also harbored the belief that physically identical replicas of his father, brother, sister, and several cousins had been constructed by extraterrestrial aliens. He added that these replicas had different minds than the originals. Mr. A claimed that he could hear and see the extraterrestrials responsible for the construction of the replicas.

Mr. A reports a history of participating in over 100 physical fights. One of these led to a charge of aggravated assault for which he was found not guilty by reason of insanity. As a result he spent one year committed to a forensic psychiatric hospital. On another occasion, he spent several months in jail after assaulting a police officer. He had previously been in jail several times for his violent attacks on others.

Mr. A's family history included a mother who suffered from paranoid schizophrenia. Mr. A currently smoked marijuana regularly. His past history revealed abuse of cocaine and LSD. He had a history of one head injury that resulted in a three-day coma. This head injury occurred at age 29. The onset of his psychotic illness occurred when he was 18 years old. He denied having serious medical illnesses including seizures. His physical, including neurological, examination revealed no abnormalities. An EKG was consistent with an old myocardial infarction, although an extensive medical record review found no history of cardiac symptoms or disease. His serum chemistries, urinalysis, and complete

blood count were within normal limits. His head MRI scan revealed small vessel ischemic changes along the left frontal lobe. His EEG was normal. Approximately 11 years prior to his last psychiatric hospitalization, due to Mr. A's history of significant violence he had been referred for genetic analysis to rule out an XYY karyotype or any other cytogenetic abnormality. Mr. A had no XYY karyotype constitution. However, his karyotype revealed 45 chromosomes instead of the usual 46. This was explained by a fusion of chromosomes 13 and 14. His karyotype was the result of a Robertsonian translocation (14) involving chromosomes 13 and 14.

Psychological testing revealed that the Benton Facial Recognition (BFRT) test score of 41 was in the normal range (15). In the Warrington Recognition Memory Test (WRMT) for faces, he scored 29, corresponding to below the first percentile. In the WRMT for word memory recognition, he scored 50, corresponding to above the 99th percentile, i.e., in the normal range (16). One year later, he was retested with the BFRT again scoring 41. On that occasion, he was also retested with the WRMT and scored again 50 on the word memory recognition subtest. On that testing the face memory recognition subtest he had scored 36, at the 1.9th percentile of a normal sample. During his first testing, he was administered the Wisconsin Card Sorting Test (WCST; 17) and had eight perseverative errors, consistent with normal executive abilities. On the Rey-Osterreith Complex Figure (18), he performed well on immediate and delayed recall of the figure, suggestive of normal constructional abilities. On the Riley Questionnaire of Experiences of Dissociation (RQED; 19), he scored 16. In a normal sample tested with the RQED, the mean was 9.92 with a standard deviation of 4.28. A task involving 24 facial expression photographs from the Ekman and Friesen (20) series corresponding to four slides each of the emotions of anger, disgust, fear, happiness, sadness, and surprise was administered. The test involved identifying the correct emotional label from the six above-mentioned emotions. He accurately labeled the emotion on a face in 79% of the slides. These 24 slides are on the average accurately labeled 90% of the time in a normal sample, amounting to an average of 21.7 pictures. On the Buss-Durkee Hostility-Guilt Inventory (21), he received the maximum score of 10 on the assaultiveness scale. He also scored high on suspicion and verbal hostility negativism components of that inventory.

On mental status examination, Mr. A was oriented in all spheres. His abstractive abilities were within normal limits. His short- and long-term memory was also within normal limits. His mood was mildly labile and associated with changes in hostility and anxiety. He met DSM-IV criteria for schizophrenia, paranoid type, chronic, and cannabis abuse (22). He was treated with antipsychotic medication and after approximately one month of treatment, his mood lability and auditory hallucinations decreased considerably. However, his visual hallucinations, depersonalization, and derealization as well as his paranoid delusional thinking including his misidentification delusions remained unchanged.

## Discussion

### *Phenomenology and Psychiatric Diagnostics*

The case of Mr. A presents with delusions of replication of both psychological and physical identities of the patient projected into the patient's environment. This presentation is consistent with the traditional case of subjective delusional misidentification insofar as the physical identity of the patient is replicated (5–7). However, in contradistinction to the typical case of subjective delusional

misidentification, the psychological identity was also thought to be replicated.

The present case is similar to classical cases of Capgras syndrome in so far as both syndromes involve replications of physical identity associated with delusional thinking (1,5,7,23) but here again the syndromes are different from each other because in Capgras syndrome the psychological identity of the misidentified object is different compared with the original (1,3,23), whereas in Mr. A's case, no changes in psychological identity were postulated when comparing the original with resulting identities. Mr. A also experienced at times the belief that he himself was a replica. This belief is suggestive of the reverse Capgras syndrome which can also be termed subjective Capgras within the self. In this syndrome, the affected individual believes in radical personality changes but does not postulate changes in physical appearance from the original identity. The affected person then concludes that he or she is a different person than the objective self (24–27). However, in contrast to the latter syndrome, Mr. A also believed that he himself had not experienced fundamental changes in psychological identity although he believed he was a different personal "unit" than his original body and mind.

Mr. A suffered from paranoid schizophrenia. This finding is consistent with the psychiatric literature which reveals that paranoid schizophrenia is the most common psychiatric disorder associated with DMS's (4,28).

### *Neurobiologic Factors*

Many cases of DMS's appear to involve non-dominant cerebral abnormalities, many of which can only be inferred via neuropsychological testing (8,10). In the case of Mr. A the WRMT for faces placed his memory recognition for faces below the 2nd percentile on two occasions separated by one year. Given that facial recognition processing appears to involve many non-dominant cerebral areas (29,30), it can be inferred that the present case of subjective delusional misidentification involving physical and psychological replication of the self which presents with face recognition memory abnormalities may also involve non-dominant brain abnormalities. Other studies of DMS's that have also utilized the WRMT also have resulted in similar findings (13,31,32). In Mr. A's case his BFRT scores were at the low end of the normal range (15). These results are also consistent with previously published cases that show immediate face matching processing to fall in the low normal to impaired range among those with DMS's in general. These studies indicate that immediate unfamiliar face recognition may be mildly impaired in DMS's (13,31–34).

Mr. A's performance on the task involving labeling of facial affect (20) suggests little impairment in this area. In previously reported cases of individuals suffering from DMS's, however, they have scored normal to significantly below average responses on similar tasks (13). It should also be stressed that although performances of different types of face processing tests may result in performances in the normal range, it is still possible that intense affective states such as anger or fear in the setting of acute psychotic states may increase the likelihood of deficits in face processing during the state of emotional intensification. Therefore, underlying face processing abnormalities may not be evident at a time when a DMS individual is more calm and is concurrently being tested (35).

Mr. A performed well on the Rey-Osterreith complex figure, a test that assesses constructional abilities (18), suggesting that in the index case, face processing abnormalities may not be explained as a function of generalized constructional abnormalities but that

in fact his “constructional” abnormality may be more specific to social objects.

We must emphasize, however, that many cases of DMS's appear to involve bilateral brain abnormalities (12,36–38), suggesting that not only nondominant but dominant cerebral structures may also be implicated in delusional misidentification. In the case of Mr. A, the WRMT revealed normal recognition memory for words on two testings suggesting that the neural structures serving mnemonic mechanisms that are necessary for word recognition are not abnormally affected. However, Mr. A's head MRI revealed ischemic vessel changes in the frontal lobe suggesting the possibility that other neural structures within the frontal lobe may be implicated in the case of subjective delusional misidentification. It has also been previously postulated that delusional misidentification may be caused by frontal lobe dysfunction because frontal lobe function is thought to be necessary for normal interpretation of symbolic knowledge important for assessments of personal identity (39). Moreover, this may require normal bilateral frontal lobe function. Furthermore, both right and left prefrontal area functioning may also be necessary for face processing (30,40) suggesting that in DMS's both dominant and nondominant cerebral dysfunction associated with face processing may be implicated in the genesis of delusional misidentification. The results of the WCST suggest that Mr. A's executive function abilities are not impaired. However, structural prefrontal abnormalities may be present that may be associated with other areas of dysfunction such as difficulties with impulse control and aggression (41) or with topographical processing of social objects as previously mentioned.

#### *Subjective Delusional Misidentification and Aggression*

Most types of DMS's may be associated with aggressive thinking as well as violence (42–45). Subjective delusional misidentification may also be associated with various degrees of aggression. For example, Christodoulou reported the case of an 18-year-old woman who believed that two women had assumed her physical identity. She attacked one of these women and pulled her hair (5). Another patient believed that a physical clone of his own body had been made as a part of a plot to kill him. He believed that an entertainment industry star and a politician were involved in the plot. He threatened the life of the star and the politician and also threatened to bomb their premises (7).

The history of Mr. A reveals that he had a long-standing history of repeated serious assaults on others, associated with his chronic psychosis. This has led to repeated confinement in psychiatric hospitals or jails. The Buss-Durkee Hostility-Guilt Inventory, a frequently used test in the assessment of hostility also revealed that Mr. A had a high propensity for attack and that his verbal and suspicious hostility were high, consistent with Mr. A's pattern of serious aggression associated with his paranoid psychotic disorder.

As previously mentioned, Mr. A believed that his clones were essentially machines designed to engage in combat. Furthermore, he believed at times that he was a participant in armed conflicts and at times he became deluded that he was a clone. His delusion of subjective misidentification was chronic and partially as a result of his delusion, he had initiated many serious physical fights. Other factors such as hallucinations and impulsivity may have contributed to his physical fights, independently of his delusional cognition.

From a phenomenologic standpoint, most cases of aggressive DMS's are dangerous because they misidentify people in the environment that they conceptualize as malicious and worthy of their

hostility (42–45). As previously stated, Mr. A's subjective delusional misidentification is unusual from a phenomenological perspective because he believed that his replicas were also mentally identical to him. This characteristic may have important forensic implications because the previously reported cases of aggressive subjective delusional misidentification probably have an increased likelihood of aggression because the patient believed in the existence of entities with bodies identical to his but minds different to his and who are intent on harming the patient verbally and/or physically. Because Mr. A believed that he had the same mind as his physical clones, and the patient was not suicidal, he did not conceptualize his clones as intending harm to him. Therefore, from a phenomenologic point of view, his aggression must originate in a different manner than in most cases of aggressive subjective delusional misidentification.

The dangerousness of Mr. A that derived from his subjective delusional misidentification appeared to be generated by his over-identification with his clones. This is especially highlighted by his intermittent beliefs that he himself was a clone and not the original identity. This over-identification was likely fueled by his belief not only that he was physically identical to his clones but that his mind was not only identical but also the same as the minds of his other physical replicas. Indeed he claimed that his mind was simultaneously present in his other replicas. The fact that he also believed that his mind was involved in coordinating physical combat in wars taking place in other worlds where many of his clones allegedly lived and frequently destroyed other beings, probably exacerbated his hostility and his readiness to attack others.

The level of derealization and depersonalization experienced by Mr. A was intense. His marked depersonalization is partially evident by his belief that he was at times someone else and his mind was essentially located in different bodies. His derealization was evident by his visions and beliefs of being in other worlds. Although no quantitative measures specific to depersonalization or derealization were available, we utilized the questionnaire of experience of dissociation as a rough measure of these experiences because both derealization and depersonalization are considered dissociative experiences and are represented in the scale. The results of the scale also suggest that Mr. A experienced substantial depersonalization, derealization, and other dissociative experiences in comparison to normals (19). Mr. A's tendency to experience intense depersonalization and derealization may be relevant to the genesis of his aggression because these phenomena may have reinforced his beliefs that he could find himself elsewhere in a war zone as a clone invulnerable and resistant to harm and pain. It should also be emphasized that depersonalization and derealization phenomena are very frequently associated with the delusional misidentification process in general (23,46).

#### *Biological Factors in Dangerous Subjective Delusional Misidentification*

At the biological level, it should be stressed that frontal lobe abnormalities as well as deficits in related areas may not be only associated with DMS's but also with aggression (47). This situation may apply to the present case because frontal cerebral abnormalities were noted via neuroimaging. Furthermore, visual-perceptual difficulties may be associated with dangerous DMS's patient and have also been hypothesized to increase the patient's aggression (32).

In this situation, aggression may increase because elements of DMS may be experienced at conscious or subconscious levels of visual processing, and this situation may increase asymmetrical

facial distortions while attempting to recognize others, with potential increases in fear and anger directed toward others, especially delusionally misidentified objects. Furthermore, temporal lobe and deep limbic structures encompass areas that are known to be involved in normal face processing (48), when defective, may not only lead to problems with face recognition (49) or delusional misidentification (50–52), but may also be implicated in aggression (53,54).

Mr. A's karyotype was consistent with a Robertsonian translocation. Although previous studies have associated cytogenetic abnormalities as well as other genetic factors with aggressive behavior (55) and DMS's may on occasion be associated with a chromosomal abnormality (56), or may co-occur in family members (57), there is little evidence to support a connection between Mr. A's dangerous delusional misidentification and his chromosomal abnormalities. Such translocations, however, may be associated with congenital abnormalities including mental retardation (58). Warburton, for example, reported a 3.7% risk of serious congenital anomalies for Robertsonian translocations in her series of cases involving prenatal diagnoses (59). It is important, however, to emphasize that these genetic abnormalities have not been causally linked with psychosis or serious aggression. We also strongly caution that determining causality of psychosis to specific genetic abnormalities remains controversial as is well illustrated by the debate that was generated by premature reports attempting to link the XYY karyotype to violent behavior (60). Furthermore, neither chromosomes 13 or 14 have been candidates for genetic sites associated with the development of schizophrenia (61–63). Therefore, Mr. A's chromosomal abnormalities are not likely to explain his aggression or his psychosis.

In summary, subjective delusional misidentification may be associated with violent behavior. The specific phenomenology of their DMS may help explain subjective reasons that motivate the affected individual to act aggressively toward others. Underlying biological factors may also help explain both subjective aspects of delusional misidentification as well as aggression associated with DMS's. There is therefore a need for future studies of aggressive individuals with subjective delusional misidentification in which not only aggression is carefully measured but it is also studied as a function of phenomenological and biological factors.

## References

1. Capgras J, Reboul-Lachaux J. L'illusion des "sosies" dans un délire systématisé chronique. *Bull Soc Clin Méd Mentale* 1923; 11:6–16.
2. Enoch MD. The Capgras syndrome. *Acta Psychiatr Scand* 1963;39:437–62.
3. Todd J. The syndrome of Capgras. *Psychiatr Q* 1957;31:250–65.
4. Berson RJ. Capgras' syndrome. *Am J Psychiatry* 1983;140:969–78.
5. Christodoulou GN. Syndrome of subjective doubles. *Am J Psychiatry* 1978;135:249–51.
6. Kamanitz JR, El-Mallakh RS, Tasman A. Delusional misidentification involving the self. *J Nerv Ment Dis* 1989;177:695–8.
7. Silva JA, Leong GB. Delusional misidentification syndromes and prominent figures. *Am J Forensic Psychiatry* 1993;14:39–44.
8. Feinberg TE, Shapiro RM. Misidentification-reduplication and the right hemisphere. *Neuropsychiatry Neuropsychol Behav Neurol* 1989;2:39–48.
9. Malloy P, Cimino C, Westlake R. Differential diagnosis of primary and secondary Capgras delusions. *Neuropsychiatry Neuropsychol Behav Neurology* 1992;5:83–96.
10. Fleminger S, Burns A. The delusional misidentification syndromes in patients with and without evidence of organic cerebral disorder: A structured review of case reports. *Biol Psychiatry* 1993;33:22–32.
11. Paillère-Martinot ML, Dao-Catellana MH, Masure MC, Pillon B, Martinot JL. Delusional misidentification: A clinical, neuropsychological, and brain imaging case study. *Psychopathology* 1994; 27:200–10.
12. Mentis MJ, Weinstein EA, Horwitz B, McIntosh AR, Pietrini P, Alexander GE, et al. Abnormal brain glucose metabolism in the delusional misidentification syndromes: A positron emission tomography study in Alzheimer's disease. *Biol Psychiatry* 1995; 38:438–49.
13. Young AW, Ellis HD, Szulecka K, de Pauw KW. Face processing impairments and delusional misidentification. *Behav Neurology* 1990;3:153–68.
14. de Grouchy J, Turleau C. Autosomal disorders. In: Emery AEH, Rimoin DL, editors. *Principles and practice of medical genetics*. Edinburgh, Churchill Livingstone, 1990;(1):247–71.
15. Benton AL, Des Hamsher K, Varney NR, Spreen O. Contributions to neuropsychological assessment. New York, Oxford University Press, 1983:30–43.
16. Warrington EK: Recognition memory test manual. Berkshire, England, Nfer-Nelson Publishing Company, 1984.
17. Heaton RK. Wisconsin card sorting manual. Odessa, Florida, Psychological Assessment Services, 1983.
18. Spreen O, Strauss E. A compendium of neuropsychological tests: Administration, Norms, and Commentary. New York, Oxford University Press, 1991.
19. Riley KC. Measurement of dissociation. *J Nerv Ment Dis* 1988;176:449–50.
20. Ekman P, Friesen W. Pictures of facial affect. Palo Alto, California, Consulting Psychologists Press, 1976.
21. Buss AH, Durkee A. An inventory for assessing different kinds of hostility. *Journal Consulting Psychol* 1957;21:343–9.
22. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th Edition. Washington, DC: American Psychiatric Association, 1994.
23. Silva JA, Leong GB. The Capgras syndrome in paranoid schizophrenia. *Psychopathology* 1992;25:147–53.
24. Siomopoulos V, Goldsmith J. Two reports of the Capgras syndrome. *Am J Psychiatry* 1975;132:756–7.
25. Signer SF. Capgras' syndrome: The delusion of substitution. *J Clin Psychiatry* 1987;48:147–50.
26. Silva JA, Leong GB, Shaner AL. A classification system of misidentification syndromes. *Psychopathology* 1990;23:27–32.
27. Silva JA, Leong GB. Delusions of psychological change of the self. *Psychopathology* 1994;27:285–90.
28. Kimura S. Review of 106 cases with the syndrome of Capgras. In: Christodoulou GN, editor. *The delusional misidentification syndromes*. Basel, Switzerland: Karger, 1986;121–30.
29. Sergeant J, Ohta S, MacDonald B. Functional neuroanatomy of face and object processing—A positron emission tomography study. *Brain* 1992;115:15–36.
30. Haxby JV, Ungerleider LG, Horwitz B, Maisog JM, Rapoport SI, Grady CL. Face encoding and recognition in the brain. *Proc Nat Acad Sci USA* 1996;93:922–7.
31. Young AW, Reid I, Wright S, Hellawell DJ. Face-processing impairments and the Capgras delusion. *Br J Psychiatry* 1993; 162:695–8.
32. Silva JA, Leong GB, Garza-Treviño, LeGrand J, Oliva D Jr, Weinstock R, et al. A cognitive model of dangerous delusional misidentification syndromes. *J Forensic Sci* 1994;39:1455–67.
33. Silva JA, Leong GB, Weinstock R, Wine DB. Delusional misidentification and dangerousness: A neurobiological hypothesis. *J Forensic Sci* 1993;38:904–13.
34. Silva JA, Leong GB. Visual-perceptual abnormalities in delusional misidentification. *Can J Psychiatry* 1995;40:6–8.
35. Herrington A, Oepen G, Spitzer M. Disordered recognition and perception of human faces in acute schizophrenia and experimental psychosis. *Compr Psychiatry* 1989;30:376–84.
36. Joseph AB, O'Leary DH. Anterior cortical atrophy in Fregoli syndrome. *J Clin Psychiatry* 1987;48:409–11.

37. Joseph AB, O'Leary DH, Wheeler HG. Bilateral atrophy of the frontal and temporal lobes in schizophrenic patients with Capgras syndrome: A case control study using computed tomography. *J Clin Psychiatry* 1990;51:322-25.
38. Silva JA, Leong GB, Lesser IM, Boone KB. Bilateral cerebral pathology and the genesis of delusional misidentification. *Can J Psychiatry* 1995;40:498-9.
39. Benson DF, Stuss DT. Frontal lobe influences on delusions: A clinical perspective. *Schizophr Bull* 1990;16:403-11.
40. Stone VE, Nisenson L, Eliassen JC, Gazzaniga MS. Left hemisphere representations of emotional facial expressions. *Neuropsychologia* 1996;34:23-9.
41. Giancola PR. Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. *Aggressive Behavior* 1995;21:431-50.
42. Fishbain DA. The frequency of Capgras delusions in a psychiatric emergency service. *Psychopathology* 1987;20:42-7.
43. de Pauw KW, Szulecka TK. Dangerous delusions: Violence and misidentification syndromes. *Br J Psychiatry* 1988;152:91-6.
44. Silva JA, Leong GB, Weinstock R. The dangerousness of persons with misidentification syndromes. *Bull Am Acad Psychiatry Law* 1992;20:78-86.
45. Silva JA, Leong GB, Weinstock R, Klein RL. Psychiatric factors associated with dangerous misidentification delusions. *Bull Am Acad Psychiatry Law* 1995;23:53-61.
46. Christodoulou GN. Role of depersonalization-derealization phenomena. In: Christodoulou GN, editor. *The delusional misidentification syndromes*. Basel, Switzerland, Karger, 1986: 99-104.
47. Raine A, Buchsbaum MS, Stanley J, Lottenberg S, Abel L, Stoddard J. Selective reduction in prefrontal glucose metabolism in murderers. *Biol Psychiatry* 1994;36:365-73.
48. Seek M, Mainwaring N, Ives J, Blume H, Dubuisson D, Cosgrove R, et al. Differential neural activity in the human temporal lobe evoked by faces of family members and friends. *Ann Neurol* 1993;34:369-72.
49. Young AW, Aggleton JP, Hellawell DJ, Johnson M, Brooks P, Hanley JR. Face processing impairments after amygdalotomy. *Brain* 1995;118:15-24.
50. Jovic Z, Staton RD. Reduplication after right middle cerebral artery infarction. *Brain Cognition* 1993;22:22-30.
51. Signer SF. Localization and lateralization in the delusion of substitution. *Psychopathology* 1994;27:168-76.
52. Rapcsak SZ, Polster MR, Comer JF, Rubens AR. False recognition and misidentification of faces following right hemisphere damage. *Cortex* 1994;30:565-83.
53. Convit A, Czobor P, Volavka J. Lateralized abnormality in the EEG of persistently violent psychiatric inpatients. *Biol Psychiatry* 1991;30:363-70.
54. Volkow NR, Tancredi LR, Grant C, Gillespie H, Valentine A, Mullani N et al. Brain glucose metabolism in violent psychiatric patients: A preliminary study. *Psychiatry Research: Neuroimaging* 1995;61:243-53.
55. Volavka J. *Neurobiology of violence*. Washington, DC, American Psychiatric Press, 1995:123-31.
56. Faber R, Abrams R. Schizophrenia in a 47, XYY male. *Br J Psychiatry* 1975;127:401-3.
57. Brown ES, Thompson R, Suppes T. Capgras' and Fregoli's syndromes in one family. *J Clin Psychiatry* 1996;57:137-8.
58. Fryns JP, Kleczkowska A, Kubián E, van den Berghe H. Excess of mental retardation and/or congenital malformation in reciprocal translocations in man. *Hum Genet* 1986;72:1-8.
59. Warburton D. De novo balanced chromosome rearrangements and extra marker chromosomes identified at prenatal diagnosis: Clinical significance and distribution of breakpoints. *Am J Hum Genet* 1991;49:995-1013.
60. Susuki D, Knudtson P. *Genethics: The clash between the new genetics and human values*. Cambridge, Harvard University Press, 1990:123-41.
61. Kendler KS, Diehl SR. The genetics of schizophrenia: A current, genetic-epidemiologic perspective. *Schizophr Bull* 1993; 19:261-85.
62. Cloninger CR. Turning point in the design of linkage studies of schizophrenia. *Am J Med Genetics* 1994;54:83-92.
63. Wang S, Sun C, Walczak CA, Ziegler JS, Kipps BR, Goldin LR, et al. Evidence for a susceptibility locus for schizophrenia on chromosome 6pter-p22. *Nature Genetics* 1995;10:41-6.

Additional information and reprint requests:  
 J. Arturo Silva, M.D.  
 Psychiatry Service (116A)  
 South Texas Veterans Health Care System  
 (Audie L. Murphy Division)  
 7400 Merton Minter Blvd.  
 San Antonio, Texas 78284