Capgras' Syndrome in First-Episode Psychotic Disorders

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Key Words

Capgras' syndrome, diagnosis · First-episode psychotic disorders · Imposters · Misidentification

Abstract

Background: Misidentification phenomena, including the delusion of 'imposters' named after Joseph Capgras, occur in various major psychiatric and neurological disorders but have rarely been studied systematically in broad samples of modern patients. This study investigated the prevalence and correlated clinical factors of Capgras' phenomenon in a broad sample of patient-subjects with first-lifetime episodes of psychotic affective and nonaffective disorders. **Methods:** We evaluated 517 initially hospitalized, first-episode psychotic-disorder patients for the prevalence of Capgras' phenomenon and its association with DSM-IV-TR diagnoses including schizophreniform, brief psychotic, unspecified psychotic, delusional, and schizoaffective disorders, schizophrenia, bipolar-I disorder and major depression with psychotic features, and with characteristics of interest including antecedent psychiatric and neurological morbidity, onset type and presenting psychopathological phenomena, using standard bivariate and multivariate statistical methods. **Results:** Capgras' syndrome was identified in

73/517 (14.1%) patients (8.2–50% across diagnoses). Risk was greatest with acute or brief psychotic disorders (schizophreniform psychoses 50%, brief psychoses 34.8%, or unspecified psychoses 23.9%), intermediate in major depression (15%), schizophrenia (11.4%) and delusional disorder (11.1%), and lowest in bipolar-I (10.3%) and schizoaffective disorders (8.2%). Associated were somatosensory, olfactory and tactile hallucinations, Schneiderian (especially delusional perception), and cycloid features including polymorphous psychotic phenomena, rapidly shifting psychomotor and affective symptoms, pananxiety, ecstasy, overconcern with death, and perplexity or confusion, as well as rapid onset, but not sex, age, abuse history, dissociative features, or indications of neurological disorders. **Conclusions:** Capgras' syndrome was prevalent across a broad spectrum of first-episode psychotic disorders, most often in acute psychoses of rapid onset.

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Introduction

Capgras' syndrome involves delusional misidentification in diverse primary psychotic or major affective disorders, as well as in neurological disorders, including

traumatic brain injury, epilepsy, stroke, and dementia. This report summarizes the concept, epidemiology, and clinical aspects of the syndrome, and reports new data on its occurrence in patients evaluated systematically and prospectively from first episodes of a broad range of psychotic disorders.

Overview of the Capgras Misidentification Concept

The psychopathological phenomenon of delusional misidentification involving ‘doubles’ or ‘imposters’ is named after Jean Marie Joseph Capgras (1873–1950), a French psychiatrist. In 1923, with Jean Reboul-Lachaux, he reported a case of *l’illusion des sosies* (the illusion of doubles), involving ‘replacement’ by imposters of family members (husband, children), later also police and neighbors as well as herself, as reported by a woman patient [1]. Capgras and Reboul-Lachaux [1] considered the phenomenon to involve an *interpretative illusion*, rather than misperception of external stimuli. The essential manifestation was a false belief that real and familiar persons or oneself are replaced by mysterious, sometimes malevolent, or morally reprehensible imposters. Similar cases were reported in the early 20th century French psychiatric literature [2], and several case reports of Capgras’ syndrome or related phenomena have appeared over the past 60 years [2–8]. Conceptually related phenomena [3] include the 19th century phenomenon of *Personenverwechslung* (mistaken identity) with a pervasive sense that another person is not whom he appears to be [4, 9, 10]. In the early 20th century, the *Fregoli syndrome* was described as a delusional belief that a persecutor whose identity is well known to the patient undergoes repeated changes of appearance and is believed to have ability to take over the bodies of other persons [11], usually with sinister or menacing intent; delusional influence on thoughts and actions was also highlighted in the original case report [11, 12]. In addition, *intermetamorphosis* is characterized by the firm belief that multiple individuals and objects from the patient’s environment may reciprocally exchange their psychological as well as physical identities and transform one into another [13, 14]. Despite distinctive differences these phenomena seem to share the core feature of a profound, sometimes subtle, change in perception of reality or attribution of meaning as regards authenticity of identity of the self or others [2, 3]. These phenomena may also share a common neurobiological substrate [15–17].

Several features of Capgras’ phenomenon, specifically, are characteristic [4–7]. Misidentification, in the sense of delusional denial of authenticity of identity of a clearly recognized person, is essential [2]. Those affected report

that one or more well-known persons (usually family members), selectively, or even the patient himself have been replaced by substitutes. Each ‘double’ is experienced as very similar to the ‘original’ in appearance and behavior, sometimes differing in minor details. The misidentification is experienced with certainty, sustained over time, and usually is not altered by efforts at clarification, and so is considered a delusional experience. The ‘original’ may be idealized, or anger and even aggressive behavior may be expressed towards the ‘double’. Affected persons usually maintain clear consciousness, with apparently intact cognitive functions, but typically are strongly paranoid, with hostility and mistrust, sometimes with feelings of depersonalization, derealization and emptiness [2, 3]. Additional features that may be related to the phenomenon of ‘subjective doubles’ (delusional belief of the existence of doubles of oneself) in the Capgras’ syndrome [2] can include *autoscopy* or experiencing oneself as separate from one’s own body [18] and *heautoscopy* or perceiving oneself as a double, usually viewed at a distance [19]. Capgras’ syndrome patients may also present with some features of nihilistic delusions that are characteristic of Cotard’s syndrome, including delusional perceptions of physical transformation, or even of not being alive – evidently as a corollary of the belief that one has been replaced [20–22]. Also, contemporary theoretical psychopathology hypothesizes that the two phenomena may be pathogenetically correlated [23]. Capgras’ phenomenon is sometimes associated with erotomanic delusions or concerns about infidelity [24–26].

Epidemiology

Systematic studies of the prevalence of misidentification syndromes in particular disorders are uncommon. Nevertheless, in addition to primary psychiatric disorders, Capgras’ and related phenomena have been associated with a range of neurological conditions (e.g., arteriovenous malformation, delirium, migraine, multiple sclerosis, brain trauma or tumor) as well as primary psychiatric disorders [27–32]. However, the prevalence of Capgras’ and other misidentification syndromes among specific psychiatric disorders diagnosed by modern criteria is not securely quantified. Estimates have been made from case series involving particular psychiatric disorders, often schizophrenia. Notably, in a review of all 4,200 patient presentations in 1983 to the University of Miami Psychiatric Emergency Service, 6 cases were determined to have a Capgras delusion (0.14% overall, and 0.17% of cases of psychotic disorders). Most of the patients identified had threatened or shown violent behavior towards a

familiar person, and many had neuromedical factors as suspected contributors [33].

In a review of 260 case reports of delusional misidentification syndromes, 174 patients had a Capgras phenomenon (66.9%); associated diagnoses ranked: schizophrenia, usually paranoid type (127/174, 73.0% of Capgras cases) > dementia or other organic mental disorders (46/174, 26.4%) > mood disorders (29/174, 16.7%) [34]. Joseph [35] identified Capgras' syndrome in 26/835 hospitalized psychiatric patients (3.11%); 92.3% (24/26) of the cases were associated with paranoid schizophrenia and 7.69% (2/26) with an affective disorder. A sample of 195 consecutive inpatients with functional psychosis yielded a prevalence of delusional misidentification syndromes of 4.10% (8/195) [36]. In another sample of 25 hospitalized schizophrenia patients treated with clozapine, the prevalence of Capgras' phenomenon was estimated at 28.0% (7/25) [37]. These findings indicate relatively high prevalence of Capgras' syndrome among psychotic-disorder patients (929/5,515; 16.8%), especially men diagnosed with schizophrenia, typically with paranoid or other delusional features, and sometimes with aggressive behavior [30, 34, 38, 39].

Among neurological disorder patients, approximately 10% of cases of advanced dementias have been diagnosed with Capgras' phenomenon [40]. In another sample of patients with dementia of the Alzheimer or Lewy-body types, 16% had some form of misidentification syndrome, whereas those with frontotemporal lobe dementia or Parkinson's disease had none [41]. In 55 consecutive patients with Lewy-body type dementia 20.0% (11/55) had Capgras' phenomenon; associated clinical findings included visual hallucinations and anxiety [32]. These observations suggest a prevalence of approximately 15.3%, or very similar to risks among persons with major psychiatric disorders as just summarized. The phenomenon has also been associated with epilepsy [42–45]. Proposed neurobiological mechanisms that might contribute to Capgras' phenomenon include right hemisphere hypo-function [46], altered connectivity of multimodal cortical association areas and paralimbic and limbic structures [47], deficits in facial processing [29, 48], dysfunction of working memory [17], dopamine deficiency [49], and bilateral dysfunction of frontotemporal connectivity [50, 51]. However, further research is required.

Given the paucity of studies on the prevalence of Capgras' phenomenon and on associated clinical features, we investigated the 517 patient-subjects in the McLean-Harvard International First-Episode Psychosis Project who had been reliably diagnosed by DSM-IV-TR criteria with a range of affective and nonaffective psychoses, with their

diagnoses verified by systematic, prospective follow-up and formal diagnostic assessments over several years following a first-lifetime hospitalization.

Methods

Subjects and Diagnostic Assessments

We studied a total of 517 initially hospitalized, first-episode patients with a broad range of DSM-IV-TR psychotic disorders in the McLean-Harvard-International First-Episode Psychosis Project based at McLean Hospital and the University of Parma. Subjects were recruited in 1989–2003 and evaluated by methods detailed previously [52, 53]. Briefly, experienced, specially trained master's level evaluators with more than 5 years of experience, recruited and assessed patient-subjects from psychiatric inpatient units at McLean Hospital and the University of Parma Medical Center within 72 h of first-lifetime psychiatric hospitalizations for a first episode of any major psychiatric illness with psychotic features. Project protocols were reviewed annually and approved by the McLean Hospital Institutional Review Board and the Ethical Committee of the University of Parma Medical Center through 2013, and are in full accord with the Helsinki Declaration of 1975 and Health Insurance Portability and Accountability Act requirements. All subjects gave written informed consent for participation and anonymous, aggregate reporting of findings. All subjects underwent prospective, structured, reassessments approximately weekly during index hospitalization and then at 6 and 12 months, and yearly thereafter for several years after initial hospitalization. Clinical, diagnostic, and research protocols were identical at both study sites.

Diagnostic assessments included the Structured Clinical Interview for DSM, Patient version (SCID-P) examinations at intake and at 2 years, followed by best-estimate diagnostic procedures based on the consensus of several clinically expert investigators (P.S., H.-M.K., C.M., M.T.), as well as considering all available information. Diagnoses were updated to meet DSM-IV-TR criteria since 2000, and compared with ICD-10 criteria. Long-term diagnostic stability was assessed with blinding to initial SCID-P-based and best-estimate, investigator-consensus diagnoses [52, 53].

Patient-subjects presented in a first-lifetime episode of nonaffective or affective psychotic illness. Exclusion criteria were: (a) acute alcohol or drug intoxication or withdrawal, or any delirium; (b) previous psychiatric hospitalization, unless for detoxification or a nonpsychotic illness; (c) intellectual disability documented with a Wechsler Adult Intelligence Scale intelligence quotient <70 or clinical evidence of other organic mental disorder or dementia; (d) index syndromal illness present >6 months; (e) any previous psychotic illness, or (f) prior treatment with an antipsychotic drug for a total of 4 or more weeks, or an antidepressant or mood-stabilizer for 3 months or longer [52, 53].

For all 517 first-episode psychotic subjects, comprehensive reviews of all available information describing and dating current and preceding clinical psychopathological phenomena, as well as clinical findings during regular and systematic follow-up assessments over at least 2 years, were conducted in random order by the same psychopathology expert (P.S.) while held blind to initial and later diagnoses. Assessments of antecedent, current, and follow-up clinical phenomena also involved best-estimate procedures based

on all available information, including initial and 2-year SCID-P assessments (with diagnoses removed), medical records, and clinical narratives from interviews of family members and primary treating clinicians. In addition, we applied the *AMDP System: Manual for the Assessment and Documentation of Psychopathology* [54] and the Bonn Scale for the Assessment of Basic Symptoms [55] to develop comprehensive, systematic and detailed inventories of psychopathology, including indications of the presence of Capgras' phenomenon at first-lifetime psychotic presentation. To assess the presence of cycloid features [56–58] we also considered the diagnostic criteria of Perris [57], Brockington et al. [59], Salvatore et al. [60] and Yadev [61]. We also rated the evolution of first episodes from their initial symptomatic presentations and prodromes as being *acute* (<1 month), *subacute* (1–6 months) or *gradual* (>6 months). The working definition of Capgras' phenomenon applied in this study was a delusional belief in the existence of virtually identical 'doubles' of persons significant to a patient or the patient him- or herself [1, 2].

Data Analyses

We compared subjects with versus without evidence of Capgras' phenomenon for potential covariates of interest. Preliminary testing employed ANOVA (t) methods for continuous variables, and contingency tables (χ^2 or Fisher's exact p) for categorical factors. Measures with at least suggestive differences ($p < 0.10$) in initial bivariate comparisons were then entered, stepwise, into logistic regression modeling to identify factors independently associated with Capgras' phenomenon, based on odds ratios and their 95% confidence intervals. Averages are means with standard deviations or medians with interquartile ranges unless stated otherwise. Analyses are based on commercial statistical programs (Stata®, Stata Corp., College Station, Tex., USA; Statview®, SAS Institute, Cary, N.C., USA).

Results

Subject Characteristics

A total of 517 adult patients were entered at hospitalization for a first-lifetime episode of a primary psychotic disorder or major affective disorder with psychotic features, and then followed systematically, at 6- to 12-month intervals, for a minimum of 2 (mean 5.8 ± 3.2) years. Of the 517 patient-subjects, 55.3% were men, and age at intake averaged 31.5 ± 13.6 (median 29; interquartile range: 21–38) years. The observed overall prevalence of Capgras' phenomenon was 14.1% (73/517; table 1). Risk of the phenomenon did not differ by sex, current age, or estimated age at onset of first antecedent morbidity (table 1).

Risk versus Diagnosis

The prevalence of Capgras' phenomenon varied among diagnostic groups ($p = 0.015$; table 1). Based on final DSM-IV-TR diagnostic criteria, apparent preva-

lence was highest among persons diagnosed with a schizophreniform disorder (50.0%), brief psychosis (34.8%), or unspecified psychosis (NOS, 23.9%), lowest among those with bipolar-I disorder (10.3%) or schizoaffective disorder (8.20%), with intermediate risks among persons diagnosed with major depression (15.0%), schizophrenia (11.4%), or delusional disorder (11.1%). Prevalence among subjects with acute or relatively brief psychotic illnesses (schizophreniform, brief, and unspecified) was 2.3 times greater than among other diagnostic groups (24.2%/10.6%; $p = 0.0001$).

Other Clinical Risk Factors

Several psychotic psychopathological features were strongly associated with Capgras' phenomenon. By their strength of association, they ranked: somatosensory, olfactory and tactile hallucinations, Schneiderian delusional perceptions and possibly feelings of alienation (thought passivity), as well as rapid onset (all $p \leq 0.01$; table 1). There also was a 2.1-fold greater prevalence of Capgras' phenomenon at the Parma site (24.5%) than the McLean site (11.8%; $p = 0.001$), as well as a 1.8-fold greater prevalence of features of cycloid psychosis among subjects with Capgras' phenomenon ($p = 0.02$). Factors not significantly associated with Capgras' phenomenon included indications of neurological disorders, current or past features of erotomania or delusional jealousy, a history of sexual or physical abuse, and the presence of dissociative or posttraumatic symptoms (table 1).

Multivariate Modeling

Factors at least suggestively associated with Capgras' phenomenon in the preceding preliminary bivariate analyses were then entered, stepwise by descending preliminary significance, into multivariate logistic regression modeling. This process yielded four factors that remained significantly and independently associated with Capgras' phenomenon: (a) acute or brief versus other types of psychotic illnesses, (b) Schneiderian delusional perceptions, (c) olfactory hallucinations, and (d) somatosensory hallucinations (table 2). No other factor, including study site, was significantly associated with Capgras' phenomenon in multivariate modeling.

Discussion

A main finding is evident lack of specificity for associations of the Capgras phenomenon across a broad range of first-episode psychotic disorder types assessed, since

Table 1. Characteristics associated with Capgras' phenomenon in 517 first-episode psychotic patients

Characteristic	Capgras' phenomenon, %		Risk ratio ^a	Statistic (χ^2 or t)	p value
	present	absent			
Cases (n = 517)	14.1	85.9	–	–	
Women	49.3	43.9	1.12	0.74	0.39
Ages, years					
At first antecedent morbidity	20.0±12.5	20.2±13.2	1/1.01	0.14	0.89
Current	29.6±12.8	31.8±13.7	1/1.07	0.94	0.33
DSM-IV-TR diagnosis, n ^{b, c}				17.4	0.015
Schizophreniform disorder (n = 2)	50.0	–	–		
Brief psychosis (n = 23)	34.8	–	–		
Unspecified psychosis (NOS; n = 46)	23.9	–	–		
Major depression (n = 60)	15.0	–	–		
Schizophrenia (n = 52)	11.4	–	–		
Delusional disorder (n = 18)	11.1	–	–		
Bipolar-I disorder (n = 255)	10.3	–	–		
Schizoaffective disorder (n = 61)	8.20	–	–		
Acute/brief psychoses ^d	24.2	10.6	2.28	15.0	0.0001
Onset type				45.8 ^e	<0.0001
Acute (<1 month)	54.8	–	–		
Subacute (1–6 months)	32.9	–	–		
Gradual (>6 months)	12.3	–	–		
Psychotic symptoms					
Somatosensory hallucinations	40.6	12.4	3.27	19.8	<0.0001
Olfactory hallucinations	44.4	13.0	3.42	14.1	0.0002
Tactile hallucinations	35.3	13.4	2.63	6.50	0.01
Visual hallucinations	17.7	13.1	1.35	1.57	0.22
Gustatory hallucinations	20.0	14.1	1.42	0.14	0.70
Auditory hallucinations	15.1	13.3	1.14	0.33	0.70
Schneiderian first-rank symptoms					
Delusional perceptions	17.5	9.55	1.83	6.61	0.01
Thought passivity	21.3	12.5	1.70	4.85	0.03
Thought interference	20.6	13.2	1.56	2.51	0.11
Any	15.8	7.55	2.09	5.38	0.02
Cycloid psychosis features	22.0	12.4	1.77	4.76	0.02
Neurological history					
Head trauma	27.8	13.6	2.04	2.87	0.09
Epileptic seizures	13.7	14.1	1/1.03	0.003	0.96
'Soft' neurological signs	23.1	13.9	1.66	0.88	0.35
Any neurological findings	17.4	13.8	1.26	0.45	0.50
Dissociative or posttraumatic symptoms	23.5	13.8	1.70	1.28	0.26
Erotomania/delusional jealousy	14.5	7.41	1.96	1.06	0.30
History of physical or sexual abuse	20.8	13.8	1.51	0.94	0.33
Study sites			2.07	10.1	0.001
McLean (n = 423)	11.8	88.2			
Parma (n = 94)	24.5	75.5			

Proportions are of specified features among patients with vs. without Capgras' syndrome unless stated otherwise.

^a Risk ratio: present/absent.

^b Rates are prevalence of Capgras' syndrome in final, consensus DSM-IV-TR diagnoses.

^c There was little difference between bipolar-I disorder patients presenting in DSM-IV-TR mixed states (10.7%) vs. mania (10.0%; $\chi^2 < 0.01$, $p = 0.95$). There was also no significant difference among subtypes of patients diagnosed with schizophrenia, although risks tended to be higher among hebephrenic and paranoid types (13.8%) than all others (catatonic, simple, or undifferentiated: 7.14%; $\chi^2 = 0.67$, $p = 0.41$).

^d 'Acute/brief' psychoses = DSM-IV schizophreniform + brief + NOS vs. all other disorders at intake.

^e Based on χ^2 for (acute + subacute) vs. gradual.

Table 2. Multivariate logistic model: factors associated with Capgras' phenomenon

Factors	Odds ratio (95% CI)	χ^2	p value
Olfactory hallucinations	3.22 (1.05–9.90)	4.19	0.041
Somatosensory hallucinations	2.43 (1.01–5.83)	3.97	0.046
Acute psychosis	2.25 (1.31–3.88)	8.54	0.0035
Delusional perceptions	1.96 (1.07–3.42)	4.80	0.028

Based on factors considered in preliminary bivariate analyses for table 1. CI = Confidence intervals.

their early course, systematically and repeatedly under the same conditions of evaluation, and with efforts to provide blindness to initial and final research diagnoses. This lack of clear diagnostic selectivity, particularly for affective versus nonaffective psychotic disorders, is consistent with some reports appearing over the past century. However, many of these were based on anecdotal clinical observations, or samples of limited diagnostic range, particularly involving schizophrenia that sometimes was diagnosed by criteria no longer considered standard [2, 38]. Other case series found large excesses of the phenomenon in patients diagnosed with schizophrenia, particularly of the paranoid type, even based on modern diagnostic criteria [33–36]. In addition, the Capgras phenomenon and other delusional misidentification syndromes have been surprisingly prevalent among patients with various neurological disorders [29, 32, 34, 38, 40, 41, 49, 62, 63]. Although lifetime neurological comorbidities were taken into account in the present analysis of factors associated with Capgras' phenomenon, neurological patients were not included.

The present study is rare in investigating the prevalence of Capgras' phenomenon and associated factors in a large sample of patient-subjects hospitalized for first-lifetime episodes of either nonaffective or affective, primary psychotic disorders, and prospectively followed up for several years. The observed prevalence of Capgras' phenomenon was 14.1%, or very similar to the estimate of 16.8% in the literature reviewed above, with an overall pooled prevalence of 16.6% (1,002/6,032; 95% confidence interval: 15.7–17.6%) among patients with severe psychiatric disorders. Risks for manifesting Capgras' phenomenon at first-lifetime psychotic presentation were especially high in patients with disorders of relatively rapid onset, diagnoses of acute and brief psychotic disorders or the presence of cycloid features [56–61], and

were associated with particular types of psychopathology, including Schneiderian delusional perceptions and passivity feelings, as well as olfactory and somatosensory hallucinations. Except for greater risk in acute psychotic disorders, we found little diagnostic selectivity for the phenomenon.

In the original case description by Capgras and Reboul-Lachaux [1], the 'illusion of doubles' was considered to represent misidentification based not on perceptual error, but on affective response resulting in an interpretative illusion. Some patients report that the 'impostor' resembles the original virtually perfectly, but notably lacking are feelings of familiarity, closeness, and intimacy with the misidentified person [64]. That is, the disturbance may be understood in psychopathological terms as an abnormal attribution of meaning, as in Schneider's [65] delusional perception (a significantly associated phenomenon; table 2), rather than as arising from a defective perceptual process, at least in nonneurological cases [3]. Affected persons make great efforts to resolve the contradiction between correct perceptions of physical appearance and erroneous attribution of their meaning, usually with a new significance of an abstract, impersonal or arbitrary quality while realizing a loss of familiar meaning in the here and now [64]. With patients being detached and alienated from their own perceptions, the significance of perceptions no longer retains meaning [64].

Abnormal self-awareness or coenesthesia and associated derealization and depersonalization may be key factors in the pathogenesis of Capgras' phenomenon [3–8, 23, 66–68]. At the onset of psychosis, coenesthetic disturbances often are experienced as subjective malaise and uneasiness. In time, however, subjective experiences of ineffable and mysterious changes of reality are projected onto an outer world that becomes eerie, puzzling, and stage-like, where persons or things give the impression of being artificial and inauthentic imitations. The purpose of such changes remains indeterminate, but selected features or persons may seem to gain special or overwhelming physiognomic expression and either lose their distance or fuse with the patient [64, 69]. Feelings of perplexed estrangement, along with perceptual fragments that stand out from their experiential context [69], infuse both the self and its surroundings with metaphysical and pervasive anguish, derealization and depersonalization. In this setting, the illusion of misidentification emerges with increasing delusional certainty. The present finding of a significant association of the Capgras phenomenon with Schneiderian delusional perceptions, passivity phenomena, as well as somatosensory and olfactory halluci-

nations accords with this psychopathological perspective of the syndrome as originating from an incorrect attribution of meaning in the context of initially altered bodily sensations and probably crumbling integrity of self-external boundaries [23, 66–68].

The relatively high prevalence of Capgras' phenomenon found in acute, transient, and unspecified psychotic illnesses with rapid onset and including cycloid features (table 1) accords with the observations reported by Leonhard [56], Brockington et al. [59] and Salvatore et al. [60] that both in confusion-motility and anxious-beatific forms of cycloid psychoses, misidentification phenomena often emerge along with paranoid ideas of reference, perceiving ominous meaning in indifferent events, as well as derealization, depersonalization and perplexity. In general, the present findings, based on standardized methods of evaluation of a large number of patients at two sites early in the course of a broad range of psychotic disorders, associated Capgras' misidentification phenomenon with relatively acute psychotic disorders or with major depression with psychotic features, more than with schizophrenia, chronic-delusional, schizoaffective, or bipolar disorders. This proposed association of Capgras' phenomenon with acute versus chronic mental illness may also be congruent with the appearance of the misidentification phenomenon in various medical and neurological disorders, particularly in acute conditions including delirium and stroke. The link between the preferential emergence of Capgras' phenomenon in acute, transient, or unspecified psychoses with cycloid features, and in acute neurological disorders may represent dissolution of the structure of consciousness, marked by confusion, twilight or dreamlike states, and strong affective arousal, resulting in growing ambiguity and inability to distinguish the familiar from the unknown or imaginary [70].

Notable limitations of this study include relatively small numbers of patients with some disorders, lack of

repeated assessments of Capgras' phenomenon during follow-up that verified diagnoses, as well as lack of patients with neurological disorders for comparison with the large cohort of persons diagnosed with a range of primary first-episode psychotic disorders. Also, the relatively low prevalence of Capgras' phenomenon in schizophrenia patients may reflect the first-episode nature of our sample, leaving open the possibility that chronically psychotic patients might show a higher prevalence due to cognitive impairments and formal thought disorders. Despite these limitations, the findings support the impression that Capgras' phenomenon occurs at substantial frequency and across a wide range of psychotic disorders, perhaps with some preference for relatively acute disorders.

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Disclosure Statement

Dr. Bhuvaneswar was employed as medical director at Covance Inc. and formerly at Shire Inc. and by EnVivo Pharmaceuticals; she holds EnVivo stock options; she has also consulted to Sepracor Pharmaceuticals on anxiety disorders. Dr. Tohen was formerly employed by Eli Lilly (to 2008), is a consultant to the company, and has recently received honoraria or consulted for Abbott, AstraZeneca, Bristol-MyersSquibb, Eli Lilly, Forest, Glaxo-SmithKline, Johnson & Johnson, Lundbeck, Merck, Otsuka, Sepracor, Sunovion, Roche, and Wyeth Corporations; his spouse was an employee and minor stockholder at Eli Lilly (to 2013). No other author or immediate family member has current or recent (≥ 5 years) financial relationships that might represent potential conflicts of interest.

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