

# Delusional Disorders: An Overview

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## Abstract

*In recent years, delusional disorders have emerged as a focus of clinical research and treatment innovation. Despite these advances, clinicians are still relatively ill informed about this group of disorders. The present review tries to update on historical aspects, current nosology, epidemiology, validity of the concept and treatment strategies for the delusional disorders. Over the years the concept of delusional disorders has been changing and has still not crystallized fully. The variation in diagnostic criteria over the years has hampered research in this field. The available research is mostly retrospective, with control groups of schizophrenia, and focusing more on demographic variables than the illness, treatment and outcome variables. There is a need for prospective studies with a variety of control groups and focus on a wide array of parameters that can help to validate delusional disorders as an independent diagnostic entity (German J Psychiatry 2006;9:62-73).*

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## Introduction

Once viewed as rare so as not to warrant a separate classification, in recent years paranoid disorder has emerged as a focus of clinical research and treatment innovation. Growing literature has revitalized the efforts to understand, define, characterize and treat this disorder. However despite these advances, clinicians are relatively ill-informed about this disorder and many may have only seen an occasional case. This is so as persons with this disorder don't regard themselves as mentally ill and actively oppose psychiatric referral, experience little impairment and in the infrequent psychiatric encounter may get labeled with schizophrenia or mood disorder (Manschreck, 2000).

The present review tries to update on historical aspects, current nosology and validity of the concept, and treatment strategies for the delusional disorder within the constraints of the search strategies employed. The search strategies for

this review included electronic databases as well as hand-search of relevant publications or cross-references. Electronic search included PUBMED and other search engines. Cross-searches of electronic and hand-search key references often yielded other relevant material. The search terms used, in various combinations, were: paranoia, paranoid disorders, delusional disorder, validity, treatment, management, history, genetic studies, family studies and nosology.

## History of the Paranoid Concept

Schifferdecker & Peters (1995) provided a comprehensive history of the delusional disorder, which can be broadly divided into Pre-Kraepelinian, Kraepelinian and Post-Kraepelinian eras.

*Pre-Kraepelinian era.* Coined by the ancient Greek the word paranoia meant 'besides' and 'self' (Mind). Heinroth (1818) used 'Verrücktheit' and paranoia synonymously, to describe

a condition characterized by ‘unfreedom (meaning mental disorder) of spirit with exaltation of the faculty of thoughts, perversion of concepts, but undisturbed perception (thus primarily cognition, and not perception, was affected)’. He believed certain persons to be predisposed to develop paranoia: vain, eccentric, brooding people who aspired to the highest and lowest; with or without external cause, when their perverse conduct and vain efforts lead them to a point of great strain and in an utter perplexity, whose outbreaks of restlessness and perversity affect their entire behaviour, which carried the unmistakable traces of its origin. He also divided paranoia into ‘simplex’ and ‘catholoca’. In modern psychiatry the term ‘paranoia’ was first used by Kahlbaum (1863) to designate those in whom the symptom manifested primarily in intelligence. He divided paranoia into 3 content types: *ascensa* (subject claims to be something other than what he is), *descensa* (the subject imagines, for example, to be possessed or be a devil) and *Immota* (with oversensitivity to stimuli and hallucinations). Krafft-Ebing (1869) considered paranoia mainly as delusions based on an underlying cognitive disorder and being independent of any affective foundation.

*Kraepelinian era.* Kraepelin’s concept and definition of paranoia changed with each edition of his manual. In his last manual he wrote that paranoia should be confined to thematically and logically consistent delusions, prophetic delusions, delusions of grandeur, and so on, but most of all persecutory delusions, which he traced back primarily to a psychopathic disposition. He differentiated paranoid disorders from dementia praecox in that patients with paranoia had no disturbance of the form of thought as opposed to the delusional content, and the main defect was in the judgement. The personality was well preserved, even though the illness might last several decades and the only behavioural changes were those related to the delusional beliefs. He described subcategories: paranoia, paraphrenia, and dementia paranoides. Paraphrenia developed later than dementia praecox, was milder than dementia praecox, was similar to paranoid schizophrenia - with fantastic delusions and hallucinations, but with less thought disorder, better preservation of affect, less personality deterioration and little loss of volition. Dementia paranoides had earlier onset, initially resembled paranoia but showed a deteriorating course, because of which it was considered as a form of dementia praecox that arose from disorder of thought, cognition and emotion. Mayer (1921) followed up Kraepelin’s cases of paraphrenia and challenged the validity of this category because a majority of the patients had an outcome similar to that of dementia praecox. Karl Kolle (1931) followed up Kraepelin’s cases of paranoia and suggested it overlapped with dementia praecox. In his revision of Paranoia, Kruger (1917) described paranoia as ‘erection of a system of delusions of persecution and grandeur, which is constructed and developed logically, does not go outside the realms of possibility, does not alter the subjects personality apart from a narrowing of his sphere of interest which may diminish his psychological adaptability, and finally does not affect the subjects perception in areas which are not important to the delusion system and the illness is notably chronic’.

Bleuler (1920) took up Kruger’s description of paranoia and emphasized purely psychogenic basis for paranoia in contrast

to schizophrenia. Describing paranoia as a ‘psychopathic reaction’ or ‘situational psychosis’ he broadened the definition to include cases with hallucinations, a paranoid form of dementia praecox (he renamed it schizophrenia) and an intermediate group; he believed paranoia described by Kraepelin so rare as not to warrant a separate classification and advocated careful exploration of schizophrenic symptoms in these cases. He also emphasized the occurrence of paranoid symptoms in other conditions. Kretschmer (1921) talked about paranoid personalities characterized by depressive, pessimistic and narcissistic traits, which develop paranoid features acutely when key or precipitating experiences occurred. He also emphasized that paranoia had a favorable prognosis. Schneider (1949) described paranoia as a peripheral type of schizophrenic psychosis.

Henderson and Gillespie (1944) described the ‘paranoid spectrum’ concept that included paranoia and paranoid schizophrenia linked by an intermediate paraphrenia. In this spectrum ‘Cluster A’ personality disorders of DSM-IV (APA, 1994) are linked but lie outside it, because some studies suggest that if an individual with one of these becomes psychotic the resulting illness will be one of those on the spectrum. Paranoid schizophrenia is included within the spectrum because it has features in common with paranoia, but other forms of schizophrenia remain outside. Possibly 10% of patients with paranoia or paraphrenia will deteriorate to schizophrenia, some older patients to dementia, but the remainder remain diagnostically stable. Thereafter, the ‘paranoid psychoses’ did not figure in active research due to the supposed rarity of its occurrence and the lack of clarity of its concept.

George Winokur (1977) and Kendler (1980, 1981a, 1981b) provided the impetus for the resurgence of interest in the paranoid psychoses. Winokur renamed this illness as delusional disorder (DD). Kendler (1980) elaborated Winokur’s criteria and suggested a division into simple delusional disorder (without hallucinations) and hallucinatory delusional disorder – a distinction currently regarded as redundant.

## Paranoid Concept

The term persecutory delusions may be used to label ordinary suspiciousness or ‘normal’ or ‘abnormal’ grandiose, litigious, hostile and jealous behaviours. To reduce the confusion Manschreck (1996, 2000) took the 5-point approach. 1. ‘Paranoid’ is a clinical construct used to interpret observations, and to apply this construct effectively the clinician must know its meaning and be able to make accurate observations of paranoid behaviour. 2. ‘Paranoid’ means the clinician has judged that the person’s behaviour is psychopathological; the judgment based on the person either being disturbed or disturbing others. 3. Even if clinically ‘central’, the paranoid features are not necessarily associated with schizophrenia and can appear in other psychiatric and medical conditions and thus may indicate psychopathology, but with no specific cause or outcome. 4. The ‘paranoid’ labeling is

based on features that are either subjective (private mental experience of the patient) or objective (observation of manifest behaviour). The subjective features include delusions of reference, persecution, grandeur, infidelity, supernatural, love, jealousy and imposture, and ideas of reference or strongly held ideas. The objective features include suspiciousness, sensitivity, sullenness, hate, irritability, quick annoyance, critical or accusatory behaviour, self-righteousness, litigiousness, grandiosity or excessive self-importance, violence, aggressiveness and obstinacy. As a group these subjective and objective features are difficult to define operationally, limiting the precision of the paranoid concept. Also, some of these can be manifestations of entirely normal behaviour. The judgment that such behaviours are paranoid rests on their extremeness and inappropriateness, their presence in combination or association with other behaviours in the list, or the presence of delusions. Finally, paranoid delusions traditionally have referred to a wide variety of delusions, not simply those of grandeur, persecution, or jealousy.

Munro provided many clinical descriptions of DD. He pointed out the unique and striking features of DD in the way in which the patient could move between delusional and normal modes of thought and behaviour. In the former (delusional mode), the individual was over alerted, preoccupied with the delusional ideas, and gave a sense of being remorselessly driven, while the normal mode was associated with relatively calm mood, reasonable range of affect, neutral conversation with an ability to be engaged in everyday topics, and some capacity for pursuit of normal activities (Munro, 1992).

## Paranoid psychosis and classificatory system

Despite advances made in the nosology of DD, the plethora of current definitions reflects a lack of consensus. The reasons could be that DD occur infrequently, the patient continues to function and live in the community without ever seeking treatment, and minimal overt identifying characteristics leading to misdiagnosis. The concept that DD is distinct from schizophrenia and mood disorder has recently been recognized by many psychiatrists. Manschrek (2000) has given a lucid description of developments in the classification of DD, the summary of which is provided below.

*DSM System.* The DSM-I & II (APA, 1952 & 1968) defined paranoid reactions as conditions with persecutory or grandiose delusions but generally lacking hallucinations. The subtypes were- Paranoia (a chronic disorder with systematized delusions) and Paranoid state (a more acute, less persistent condition with less systematized delusions). The DSM-III (APA, 1980) established new definitions, but earlier concepts were still evident in the essential features of paranoid disorder as persistent persecutory delusions or delusional jealousy not attributable to any other mental disorder; included were paranoia, shared paranoid disorder, acute paranoid disorder, and a residual category – atypical paranoid disorder. A subdivision was provided by duration of symptoms: a duration of more or less than 6 month separated paranoia from acute paranoid disorder. The drawbacks of DSM-III were: the

boundaries between these conditions and other disorders such as paranoid personality disorder or paranoid schizophrenia were vague; different types of paranoid disorders were classified on the basis of chronicity; the criteria narrowed the bounds of previous classifications by excluding cases with marked hallucinations or hypochondriacal, erotomanic and similar delusions. The DSM-III-R (APA, 1987) tried to minimize the confusion by re-labeling paranoid disorder as DD and highlighted as an essential feature the formation of delusions in the absence of schizophrenia, mood disorder or organic disorder. In addition, the diagnosis required the criterion of 1-month duration and subtyping was based on the predominant content of delusion (jealous, erotomanic, somatic, etc.), broadening the category to include unusual delusions as well as the more common persecutory type. In many respects the criteria were identical to Kraepelin's definition of paranoia except that Kraepelin was reluctant to include cases with hypochondriacal or somatic delusions or hallucinations. The DSM-III-R also introduced the term non-bizarre delusions and renamed the shared paranoid disorder as induced psychotic disorders not elsewhere classified, along with schizophreniform, schizoaffective disorder and brief reactive psychosis. This approach represented a fundamental departure from DSM-III, which placed DD among the paranoid disorders. The limitation of DSM-III-R was that the distinction between schizophrenia and delusional disorder was made unclear and controversial. The DSM-III-R defined this boundary by the non-bizarre qualities of delusions in delusional disorder and absence of other odd or bizarre behaviours apart from the delusions. However, bizarre/non-bizarre are difficult to define and apply reliably. DSM-IV (APA, 1994) tried corrective measures by suggesting terms like systematized and prominent, which again have limitations. This promoted the case for modifying the criteria by using the level of impairment to characterize the distinction between schizophrenia and DD. Thus, the poor functioning in DD was the result of the delusions. In contrast, poor functioning in schizophrenia usually results from cognitive compromise, and the positive and negative symptoms, especially avolition. DSM-IV also attempted to resolve the issue of the classification of delusional variants of somatoform disorder, specifically body dysmorphic disorder. It permitted dual diagnosis of body dysmorphic disorder and DD when a delusional belief was present in the former. This approach of giving two diagnoses to the same symptom reflected the available research on the relationship between the two disorders and underlined the need for further research to clarify these distinctions. DSM-IV applied similar solution to delusional variant of hypochondriasis and obsessive compulsive disorder (OCD); an OCD patient may also be diagnosed as with DD. Lastly, DSM-III-R category induced psychotic disorder was renamed in DSM-IV as shared psychotic disorder; this change reflected the attempt to avoid the term 'paranoid' and to identify the condition without reference to any presumed cause or mechanism.

*ICD System.* The ICD-9 (WHO, 1978) contained more categories for paranoid disorders than the DSM-II. Most paranoid disorders in ICD-9 fell under the rubric paranoid state and included simple paranoid state, paranoia, paraphrenia and induced psychosis; additional subcategories included

**Table 1. Comparative Nosology of Delusional Disorders in Classificatory Systems (Manschrek, 2000)**

ICD-9 (1978)	DSM-III (1980)	DSM-III-R (1987)	ICD-10 (1992)	DSM-IV (1994)
Paranoid state, simple			Delusional disorder	
Paranoia	Paranoia	Delusional (Paranoid) disorder		Delusional disorder
Paraphrenia (involitional para-noid state, late paraphrenia)			Delusional disorder	
Induced psychosis (Folie à deux, induced paranoid disorder)	Shared paranoid disorder	Induced psychotic disorder	Induced delusional disorder	Shared psychotic disorder
Other specified states (paranoia querulans, Sensitiver Beziehungswahn)			Delusional disorder	
Unspecified paranoid	Atypical paranoid disorder		Persistent delusional disorder, unspecified	
Acute paranoid reaction (bouffée délirante)	Acute paranoid disorder		Paranoid reaction	
Psychogenic paranoid psychosis (protracted reactive paranoid psychosis)				

other and unspecified paranoid state. Acute paranoid condition and psychogenic paranoid psychosis were classified separately. ICD-10 (WHO, 1992) created a classification similar to DSM-III-R and DSM-IV with subtypes of the disorder overlapping with the DSM-IV subtypes. It included paraphrenia under Persistent DD but delusions were to be present for about 3 months for diagnosing DD. For disorders of lesser duration, a diagnosis of acute and transient psychotic disorder was made. Induced psychotic disorder was considered a separate designation with a phenomenology similar to the persistent delusional disorder.

The comparison shows that paranoid state of ICD-9 was similar to DD of ICD-10. Paranoia of ICD-9 & DSM-III was same as DD of ICD-10, DSM-III-R, and DSM-IV. Paraphrenia of ICD-9 was classified as delusional disorder in ICD-10 but did not find any place in DSM classification of DD.

## Epidemiology

The incidence rates reported for DD substantiate the impression that DD is uncommon but not rare. The rate for inpatient admissions was reported as 0.3-0.5% (Winokur, 1977) and as 0.5-9% in a subsequent literature review (Kendler, 1982); the latter also reported that DD constituted 2.7% of functional psychosis. The reported outpatient rates for DD have varied between 0.83-1.2 % (Yamada et al, 1998; Hsiao et al, 1999). For DD in general population the reported annual first admission rates were 0.7-3 and prevalence rates were 24-30 per 100,000 population (Manschreck, 2000).

In India the rates have varied: 5% cases of paranoia out of all patients with delusions (Kala and Wig, 1978), 0.5% of the psychiatric outpatients having delusional parasitosis (Srinivasan et al, 1993), and 1% of the total outpatients having DD including half with delusional parasitosis (Hebbar et. al, 1999).

## Validity of Delusional Disorder

In terms of relevance to the question of validity of DD the available data can be divided into three classes of potential validators (this schema represents an adaptation and enlargement of the validating criteria for psychiatric illness as outlined by Robin and Gaze, 1970): antecedent, concurrent and predictive validators (Table 2).

**Table 2: Validators for diagnosis of delusional disorder**

Antecedent	Concurrent	Predictive
Demographic factors	Physiological	Course and outcome
Premorbid personality	Neuropsychological	Response to treatment
Precipitating factors	Neurophysiological	
Family studies	Neuroimaging	
	Biological	
	Genetics	

**Table 3: Demography of Delusional Disorder**

Study	Year of Publication	Age at onset in years	At first admission		
			Age in years	Sex ratio	Marital status (% married)
Rimon	1965	45.7		F>M	
Retterstol	1966	57		M>F	
Winokur	1977	21-50	40-49	M>F	
Kendler	1982	34-45		F>M	
Hsiao	1999	42.4		M>F	
Dayton	1917-1933		40-49		60
Mental Health Statistics, Canada	1932-1976		40-49		66-72
Michigan State Hospital Statistical Report	1933-1938		40-49		77
Kendler	1984		35-55		
Yamuda	1998			F>M	
Jorgensen	1985			F>M	
Stephen	2000			F>M	
Peilock	1913				69
Malzberg	1940				67

M= Male; F= Female

## Antecedent Validators

### Demographic factors

*Age at onset:* At the onset of illness the DD cases are older than the schizophrenics: the commonest age at onset being 34-45 years (Table 3). Yamada et al (1998) reported the oldest age at onset for the persecutory type and the youngest for the somatic type.

*Age at admission:* The peak age for first admission for the DD is between 40-49 years followed by age at first presentation being 30-39 years (Table 3); in contrast first admissions for schizophrenia peak at 20-29 years or 25-34 years. The difference in age at first admission between DD and affective disorders is much less.

*Sex ratio:* Recent studies have reported that for DD first admissions females outnumber males (Table 3); some studies reported that percentage-wise cohorts of DD had less males compared to schizophrenia, and more males compared to affective illness.

*Marital Status:* At admission 32% DD cases were found to be never married (Table 3) compared to 50-69% patients with schizophrenia; the comparable data for affective disorder subjects was similar.

*Educational History:* The DD cases were reported to be more poorly educated than cases with affective illness (Kendler, 1982).

*Occupational Status:* Retterstol (1966) reported 79% and 74% of the DD cases respectively to be self supporting and with no major period without work compared to 31% and 30% respectively of the cases with schizophrenia. Winokur (1977) also reported the DD cases to have a satisfactory

work history compared to the cases suffering with schizophrenia.

*Social Factors:* The DD cases have been consistently reported to have a poor financial condition, similar to cases with schizophrenia but worse than that of cases with affective disorder (Kendler, 1982; Dayton, 1940; Annual Health Reports of Mental Institutions, 1932-1945 & Mental Health Statistics, 1946 -1954, Canada, 1932-1952). Kendler (1984) also reported that first admission cases of DD were more likely to be foreign born than patients with either schizophrenia or affective illness.

*Premorbid Personality:* The cases with DD were more likely to be extrovert, dominant and hypersensitive compared to schizophrenics who were more likely to be schizoid, introvert and submissive (Bonner, 1951; Johanson, 1964; Retterstol, 1966).

*Precipitating Factors:* Compared to schizophrenia, the most common precipitating factors in DD group were social isolation and conflict with conscience (Retterstol, 1966).

*Family Studies:* Kendler and associates have carried out the bulk of work in this area. Between 1981 and 1995, using self-generated or DSM-III or DSM-III-R criteria of DD they found no strong familial relationship between DD and schizophrenia; however, some relationship with alcoholism was reported in their most recent study (Kendler and Walsh, 1995).

Munro and Mok (1995) reported the following findings in only meta-analysis of data available on DD, mainly in relation to treatment response: female to male ratio of 3:2; the mean age of females being higher than that of males at the time of case identification; and high rates of widowhood in females and celibacy, especially in males. Only 18.7 % of DD patients had a positive family history of psychiatric disorder; but incomplete reporting suggested this was a gross underestimation. A combination of organic brain disorder and/or alcohol or substance abuse was relatively more common among males than females.

**Table 4: Family studies of delusional disorder**

Author, Year	Diagnostic criteria	Control group	Method used	Results and conclusions
Winokur, 1977	Self generated criteria	No control	Retrospective chart review	Little familial association between delusional disorder and affective disorder. Prevalence of Schizophrenia in families of probands of paranoia was approximately equal to that found in families of schizophrenic probands.
Kendler et al, 1981	Self generated criteria	Schizophrenia	Interview with relatives	Delusional disorder is not a part of schizophrenic spectrum.
Kendler & Hay, 1981	Self generated criteria	Schizophrenia	Interview with probands & relatives	Prevalence of schizophrenia in the relatives of delusional disorder proband was less than that in the relatives of the schizophrenics.
Kendler et al, 1985a	DSM-III	Patients with surgical illness	Interview with probands & chart review	Increased risk of paranoid disorder in relatives of schizophrenics.
Kendler et al, 1985b	Self generated criteria	Patients with medical illness, schizophrenia	Family history, RDC	The morbid risk of schizophrenia in relatives of delusional disorder and medical controls was similar and lower than that in the relatives of the schizophrenics.
Watt, 1985	DSM-III	Schizophrenia, recurrent unipolar depression	Interview with probands & relatives using family history, RDC	Rate of occurrence of schizophrenia in the families of paranoid proband was significantly lower than the rate in the families of proband with late onset schizophrenia.
Kendler & Walsh, 1995	DSM-III-R	Schizophrenia, affective illness and general population	SCID	No strong relationship between delusional disorder and schizophrenia, however has a relationship with alcoholism.
Howard, 1997	Self generated criteria	Healthy elderly	Family history, RDC	Relatives of late-life onset non-affective psychosis were not at increased risk of developing early or late onset schizophrenia.

## Concurrent Validators

### Physiological

Biological data on DD is scarce. Deafness, conventionally regarded as an etiological factor in paranoid disorders, has been less well documented for DD than for late paraphrenia (Kay & Roth, 1961) and paranoid schizophrenia (Cooper et al., 1974). Thomas (1981) found little increase in paranoid symptoms with deafness.

Hayman (1913) found amenorrhea to be markedly lower in DD than in cases with schizophrenia or affective illness. Bonner (1950) noted that hypertension was more frequent in DD than in cases with paranoid schizophrenia.

### Neuropsychological

Fould and Owen (1963) used the Runwell symptom and sign inventory, hysteroid-obsessoid questionnaire, and punitive scale to measure psychological traits and symptoms. They found their DD group to differ from both paranoid and non-paranoid schizophrenics whereas the two groups of schizophrenia were similar. Tarter and Perley (1975) studied DD and paranoid schizophrenia cases with rod and frame test, size estimation test, and Minnesota Multiphasic Personality Inventory (MMPI); the two groups differed on MMPI but not on the other two perceptual tests. Herlitz and Forsell (1996) examined memory functions in two groups of elderly adults with and without suspected DD using a variety of episodic recall and recognition tasks, and found that those with suspected DD had a mild episodic memory deficit in the absence of other cognitive functions compared to normal subjects. Comparing schizophrenia and DD cases for neurocognitive functioning; Evans et al (1996) found non-

**Table 5: Course and Outcome studies of delusional disorder**

Author (Year)	Sample size	Diagnostic criteria	Follow-up Duration	Results and Conclusions
Faergemen (1963)	9	Not Mentioned	15 Years	22% developed schizophrenia
Johanson (1964)	52	Not Mentioned	Up to 4-1/2 years	12% developed schizophrenia
Retterstol (1966,1970)	163	Not Mentioned	5-15 years	13% developed schizophrenia, 6% manic-depressive illness. 79% were self-supporting and 74% had no major period without work. (compared to 31% and 30% of schizophrenics respectively). 39% were psychotic on long term follow up (compared to 87% schizophrenics).
Winokur (1977)	29	Self-Generated	Up to 20 years	Rediagnosed: 4% as schizophrenia, 3% as affective disorder. 30% recovered socially.
Berner et al (1984)	84	ICD-9	6-9 years	Course: chronic in 63%, episodic in 30% cases, and 7% recovered.
Jorgensen & Munk-Jorgensen (1985)	50	ICD-8	5-15 years	8% had full remission, 70% partial remission, and 22% were continuously psychotic.
Opjordsmoen & Rettersol (1991)	72	DSM-III-R	20 years	Shorter duration of illness had better outcome based on clinical and GAS scores.
Stephens et al (2000)	60	DSM-IV	>5 years	27% recovered, 52% unimproved; 28% rediagnosed with schizophrenia and 8% with BAPD.

significantly lower impairment, while Jeste et al (1991) found greater impairment on the Halstead-Reitan test. Studies on erotomania- a specific subtype of DD, have reported deficits in cognitive flexibility and associative learning that are mediated by frontal-sub cortical systems, as also deficits in verbal and visuospatial skills (Faujii et al, 1999).

## Neurophysiological

Gambini et al (1993) studied smooth pursuit and voluntary saccadic eye movements in cases of DD and schizophrenia, and normal subjects and found that schizophrenic cases differed from normal subjects in smooth pursuit eye movements, whereas both patient groups showed more saccades than normal subjects during the smooth pursuit test, and the DD patients and normal subjects differed in some voluntary saccadic eye movements. They concluded that there was dysfunction in eye tracking in DD. The same group (Campana et al, 1998) reported another study demonstrating abnormal smooth pursuit eye movements in DD, indicating a cerebral dysfunction similar to that detected in patients with schizophrenia.

## Neuroimaging

Naguib and Levy (1987) used CT scan and found ventricular brain ratio (VBR) to be 13% in late paraphrenia compared to

9.75% in controls. Flint et al (1991) used CT scan to find clinically unsuspected cerebral infarction in all cases of late onset paranoia compared to only 8% cases with paraphrenia. Howard et al. (1994) used MRI and found lateral ventricle volume in DD cases to be much greater than that in schizophrenia and almost twice than that in healthy controls.

Miller et al (1991) reported areas of hyper intense MRI signals in deep white matter in temporal and frontal lobes as an anatomically non-specific finding in late paraphrenia and later related these findings to localized disturbances of cerebral blood flow (Miller et al, 1992). Reduced cerebral blood flow in left parietal and temporal regions was also reported in a SPECT study (Ota et al, 2003).

## Organic/Biological

Cummings (1985) prospectively studied 20 consecutive patients with organic delusions. He reported that simple delusions occurred in patients with cortical and hippocampus lesions (e.g., Alzheimer's disease and multi-infarct dementia) and usually improved after treatment with low doses of neuroleptic drugs. In contrast, complex delusions usually occurred in patients with sub cortical lesions (e.g., Parkinson's disease, idiopathic calcification of based ganglia) and were more resistant to treatment. Lo et al. (1997) found that compared to functional cases of DD, cases with organic DD had less family psychiatric history, an older age of onset of psychiatric disorder, lower treatment doses of antipsychotic drugs and longer hospital stay.

**Table 6: Differential Diagnoses for delusional disorder (Adapted from Manschreck, 1996)**

Disorder	Delusions	Hallucinations	Awareness	Other features
Delusional disorder	+	Occasional	Alert	Relatively free of psychopathology
Psychotic disorder due to a general medical condition, with delusion	+	+	May be impaired	Cognitive changes; perceptual changes; substance abuse history; impairment of functioning frequent
Substance-induced psychotic disorder	+ (can be bizarre)	+	Acute: impaired, Chronic: may be alert	History of substance abuse; impaired functioning likely
Schizophrenia	+ (bizarre)	+	Alert	Emotional changes, pervasive thought disorder; role impairment
Major depressive episode	+ (usually mood congruent)	±	Alert	Concerted changes in mood and neurovegetative features
Manic Episode	+ (usually mood congruent)	±	Alert	Concerted changes in mood, decreased need for sleep, energy, lack of inhibition
Obsessive–Compulsive disorder	-	-	Alert	Not psychotic; impaired functioning likely
Personality disorder	-	-	Alert	Not psychotic
Somatoform disorder	-	-	Alert	Not psychotic
Shared psychotic disorder	+	-	Alert	Close associate has same delusions

## Genetics

Catalano et al (1993) studying genotype of schizophrenia, normal and DD subjects found that involvement of genetic variation in the Dopamine D<sub>4</sub> receptor gene confirmed susceptibility to DD. Zenner et al. (1998) also found multiple genetic polymorphisms of the human dopamine D<sub>4</sub> receptor (hD<sub>4</sub>R) and reported 12 bp repeat in axon 1 to be associated with DD.

In a molecular genetic study of DD, Morimoto et al (2002) found genotype frequency of the DRD2 gene Ser311Cys to be higher in cases with persecutory type DD (21%), compared to schizophrenia cases and controls (6% each). There was a significant positive correlation between the polymorphic (TCAT)(n) repeat in the first intron of the TH gene and pretreatment levels of pHVA in DD. They suggested that DD, especially the persecutory type, included a dopamine psychosis and that polymorphism of the DRD2, DRD3 and/or TH gene was part of the genetic basis underlying the hyperdopaminergic state that produced paranoid symptoms.

## Predictive Validators

### Course and outcome

The available studies on outcome/diagnostic stability in DD using sample sizes of 9-163, different diagnostic criteria, and up to 20 years follow-up have shown the re-diagnoses of 3-28% as schizophrenia and 3-8% as affective illness; in others- the diagnosis was stable. Also the global outcome of DD is shown to be better than that for schizophrenia. Kendler and Walsh (1995) reported the duration of illness for DD (38 ± 26) months, to be shorter than that for schizophrenia (159 ± 134) months. Stephen et al. (2000) found that poor follow up was related to reclusive personality, poor premorbid history, onset 6 months or more before admission, gradual onset, lack of insight, single marital status and lack of precipitating factor. Using the first four of the above-mentioned variables, which were predictive of long term outcome, they developed a prognostic scale for the DD.



# Treatment of Delusional Disorder

## Pharmacotherapy

Much of the literature on treatment of DD is in the form of reports on individual or very small series of cases. The introduction of oral neuroleptic pimozide and its successful use in the treatment of DD has prompted some researchers to claim its therapeutic specificity for DD, which is not shared by other antipsychotics. Further on, this is claimed to justify the retention of the concept of paranoia as a separate diagnostic entity. Munro & Mok (1995), in their meta-analysis, reviewed approximately 1000 articles on paranoia/delusional disorder from 1961 onwards and selected 257 cases as per DSM-IV criteria. Much of the information obtained was of poor quality. They divided the 'response to treatment' data into categories of 'recovery', 'partial recovery', and 'no improvement' and where applicable, 'noncompliance'. They found that earlier a variety of neuroleptics were used but since 1980 pimozide was the drug of choice. Adequate treatment details were available for 209 cases, out of which 110 (52.6 %) showed recovery, 59 (28.2 %) showed partial recovery, and 40 (19.2 %) showed no response. For pimozide they found 68.5% cases as fully recovered and 22.4 % as partially recovered, making a total of any recovery of 91%; the contrasting data for other antipsychotics was 22.6% full recovery, 45.3% partial recovery and a total of any recovery of 68% - the difference was significantly in favour of pimozide ( $p < 0.001$ ). Beneficial effects in DD have also been found with risperidone (De Leon et al, 1999; Elmer et al, 2000).

Srinivasan et al, (1994) from India found good response to antipsychotic treatment using trifluoperazine, haloperidol, chlorpromazine, and electroconvulsive therapy; 11 out of 19 cases of delusional parasitosis showed complete remission, and five maintained the recovery for more than 3 years.

Besides conventional antipsychotics beneficial responses have been reported with SSRIs (Lane, 1990; Hollander et al, 1989; Gross, 1991), MAOIs (Jenike, 1984), Clomipramine (Wada et al, 1999) and other TCAs (Pylko & Sicignan, 1985), and ECT (Jordan & Howe, 1980).

## Non-Pharmacological Treatment

Compassion, reassurance and treatment of the underlying disorder may eliminate the delusional symptoms. Insight oriented psychotherapy is usually contraindicated but a combination of supportive psychotherapeutic approaches and possibly cognitive behavioural intervention is considered sensible. The goals of supportive therapy are to allay anxiety and initiate discussion of troubling experiences and consequences of the delusions, thereby gradually developing collaboration with the patient (Manschrek, 2000). Cognitive approaches have attempted to reduce delusional thinking through modification of the belief itself; focusing on the associated reasoning on the reality testing of the deluded

patient. Simon et al (1999) reported that a third of their DD patients with chronic delusions, when treated with cognitive therapy for delusional modification, responded with a reduction in the degree of belief. The outcome was predicted by the change within therapy session and variation in the conviction at baseline.

All the above validators argue in favour of the distinctiveness of DD but it is likely that some cases of DD will develop schizophrenia or mood disorder. Hence, the current clinical criteria have limitations and need improvement, which may be possible with more rigorous research in relation to certain validation parameters i.e. biological markers, treatment response and outcome.

## Differential Diagnosis of Delusional Disorder

DD being uncommon and possessing some characteristics of the full range of paranoid illness, it is clearly a diagnosis of exclusion (Table 6 gives the differential diagnoses for DD). The clinical assessment of paranoid features requires three steps: Initially the clinician must recognize, characterize, and judge as pathological the presenting paranoid features. Next, the clinician must determine whether the paranoid features form a part of a syndrome or are isolated. Finally, the differential diagnosis should be developed

## Conclusions

Since the introduction of the term paranoia by Kahlbaum in modern psychiatry, the concept of paranoia/DD has kept on changing over the years and it has not crystallized fully as yet. The variation of the diagnostic criteria over the years has hampered the research in this field. The other limitations to the research in this area are: low incidence, small sample sizes, lack of insight and low impairment in these patients thereby hindering treatment seeking. The available research is mostly retrospective, with control groups of schizophrenia and focusing on demographic variables more than the illness, treatment and outcome variables. Thus we need prospective studies with a variety of control groups and focus on a wide array of parameters like biological marker, treatment response and outcome to help validate DD as an independent diagnostic entity.

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