

# Is Schizophrenia Disappearing? The Rise and Fall of the Diagnosis of Functional Psychoses: an Essay

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## Article Info

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## Abstract

The category diagnosis of functional psychoses builds on views of influential professionals. Until the second half of the 1800s, the conceptions of mania and melancholia from the Greek antiquity included largely all functional psychoses. Disturbed mood and energy were central symptoms, and the idea of unitary psychosis prevailed. From the 1900s this was followed by a dichotomy between schizophrenia and affective psychoses and broadening of the schizophrenia concept. Affective symptoms were strongly downgraded. Many psychoses with mixed features were described, and there have now long been four main categories of functional psychoses – affective, schizophrenic, schizoaffective/cycloid/reactive/polymorphic, and delusional/paranoid psychoses. The last three are included in “psychotic disorders”. The boundaries between categories have varied with time, place and professionals’ views. DSM-5 is updated with separate chapters for catatonia and psychotic symptoms, both unspecific, and removal of the subtypes of schizophrenia. However, time may be running out for categorical psychosis diagnoses, which may be replaced by continuum, spectrum, dimensional and research domain criteria. Affective symptoms are often difficult to acknowledge, diagnosis is often done on the basis of preconceptions, and patients’ affect characterized accordingly. Chronic mood disorders may appear as schizophrenic or paranoid psychosis, end-stages like heart failure in heart diseases. This underscores the importance of early and optimal treatment of mood disorders, which may be the most important cause of schizophrenia and other functional psychoses.

Psychosis is a mental state with grossly impaired reality testing, manifesting as different mixtures of delusions, hallucinations, deviant thinking and abnormal motor behavior, so-called positive symptoms. Negative symptoms - reduced emotions, interests, will and social participation - are also common, as are disturbed mood/affect and energy. When no detectable organic cause is present, psychoses have been denoted functional from the 1800s<sup>1-9</sup>.

The category diagnosis of functional psychoses builds on the views of influential professionals and includes four main groups<sup>1,2,10</sup>: 1) psychotic mood/affective disorders, 2) schizophrenia with the subgroups paranoid, hebephrenic/disorganized and catatonic schizophrenia, 3) schizoaffective/cycloid/reactive/polymorphic psychoses, and 4) delusional/paranoid psychoses. In the diagnostic manuals, the last three groups are included in “psychotic disorders.”

The occurrence of the groups varies for unknown reasons and with professionals’ views. For example, under the heading “Is schizophrenia disappearing?” Der, Gupta and Murray<sup>11</sup> reported a 50 percent fall in the incidence of psychotic disorders in England and Wales from the mid-1960s to the 80s. Later, Lake and Hurwitz<sup>12</sup>

depicted a gradual shift in diagnoses from schizophrenia via schizoaffective disorder to psychotic mood disorders from the 1960s to the 2000s. Fink and others<sup>13,14</sup> have argued that catatonia should be separated from schizophrenia because it is mostly seen in major mood and organic brain disorders. Lake<sup>15</sup> have hypothesized that grandiosity and guilt cause paranoia, so that paranoid schizophrenia is a psychotic mood disorder. Only hebephrenic/disorganized schizophrenia would be left, however, Lake<sup>6</sup> has reasoned extensively for schizophrenia being a “misdiagnosis”.

In Japan and South Korea schizophrenia has been renamed, reducing the stigma and improving the communication with the patients. Others, too, think the time is in for replacing the term<sup>16</sup>, for one thing because it leads the thought away from other aspects than treatment with antipsychotics. Especially, this concerns the treatment of mood disturbance, which may be masked by psychotic symptoms. Mood and thinking are always associated. This has been acknowledged from the antiquity. However, from the end of the 1800s, this was overshadowed by a dominating emphasis on thought disturbance. History has shown this to be unfavorable, leading to underestimation of emotional life and mood<sup>6,17</sup>.

Until the second half of the 1800s, the conceptions of mania and melancholia from the Greek antiquity included largely all functional psychoses, with disturbed mood and energy as central symptoms<sup>3,4</sup>. A common pathology was assumed, and the idea of “unitary psychosis” prevailed<sup>6,18</sup>. However, in the second half of the 1800s some influential European psychiatrists argued for several other psychoses, and the idea of “unitary psychosis” receded into the background. In 1860-70 Ewald Hecker and Karl Ludwig Kahlbaum described a psychosis variant, hebephrenia, in young people whose development most often had been somewhat slow. However, the starting point was still a mood disorder - the illness often progressed “from melancholia, to mania, to confusion, and then to dementia”<sup>19</sup>. Kahlbaum also described catatonia<sup>13</sup>, i. e. severe motor disturbance in psychoses, however, only “to put order in the confused field of melancholia attonita”<sup>3</sup>. Other types of psychosis were also described, especially periodic and circular insanity, amentia/confusion psychosis, and dementia paranoides<sup>4,20</sup>.

At the turn of the century Emil Kraepelin grouped hebephrenia, catatonia and dementia paranoides - when leading to psychic invalidity - in a narrow concept of dementia praecox. Melancholia, mania, manic-melancholic mixed states, periodic and circular insanity, amentia/confusion psychosis and paranoia were included in a broad concept of manic-depressive insanity<sup>2,20</sup>. In the 1910s Paul Eugen Bleuler replaced the term dementia praecox with schizophrenia, markedly broadened the concept, and described “basic or fundamental” disturbances to be pathognomonic to schizophrenia<sup>2,21</sup>. However, these

disturbances overlap with negative symptoms, depression, and the rich associative thinking in bipolar disorder<sup>6,8,9,22</sup>. Kraepelin later became in doubt of his dichotomy and wrote in 1920: “It is becoming increasingly clear that we cannot distinguish satisfactorily between these two illnesses and this brings home the suspicion that our formulation of the problem may be incorrect”<sup>6,23</sup>. However, this was little noticed - there was now in the minds of most professionals established a mental “firewall” between schizophrenia and manic-depressive insanity<sup>13</sup>.

In the 1940-50s the “firewall” was further strengthened by Kurt Schneider. He listed “first rank” symptoms - types of hallucinations and delusions he meant were specific to schizophrenia<sup>2,24</sup>. Bleuler and Schneider based the differential diagnosis on the hierarchical principle of Karl Jaspers: After organic symptoms came schizophrenic/psychotic symptoms, which came before mood/affective and neurotic/personality symptoms<sup>2</sup>. The importance of mood/affective symptoms was strongly downgraded.

Sigmund Freud’s hypotheses offered explanations and hope for treatment. According to Max Fink “an image of dementia praecox as a brain disease was replaced by an image of disorganization induced by childhood experience and memories, best relieved by individual psychoanalysis, a philosophy enthusiastically adopted by Paul Eugene Bleuler”<sup>13</sup>. Therefore, early diagnosis was important - “even a trace of schizophrenia is schizophrenia”, was emphasized in a 1954 textbook<sup>25</sup>. All this made the concept of schizophrenia extensive and unclear - as is still the case<sup>6</sup>.

Several influential psychiatrists tried to compensate for the “firewall” by defining disorders with a mixture of schizophrenia-like and affective symptoms, and several terms appeared for overlapping syndromes.<sup>2</sup> The American psychiatrist Jacob Kasanin introduced in 1933 the term schizoaffective psychosis with reference to nine patients who had previously been diagnosed with dementia praecox/schizophrenia<sup>26</sup>. The Norwegian psychiatrist Gabriel Langfeldt followed in the 1920-30s a similar patient group, which he labelled schizophreniform psychosis. However, a later follow-up indicated that most of his patients had suffered from affective disorders<sup>27</sup>.

Based on works of Carl Wernicke, the German psychiatrists Karl Kleist and Karl Leonhard described similar syndromes in the first and midst of the twentieth century under the term cycloid psychosis, with three overlapping forms - motility psychosis, confusion psychosis and anxiety-elation-psychosis<sup>28-30</sup>. Postpartum psychosis became the “flagship” for this diagnosis, and the Italian-Swedish psychiatrist Carlo Perris underscored the importance of electroconvulsive therapy (ECT) and lithium<sup>31</sup>. Corresponding syndromes were in Denmark and Norway often named psychogenic or reactive psychosis<sup>32,33</sup>.

The most severe forms equalled a syndrome with many names: Bells mania, delirium grave, delirium acutum, delirious mania, acute deadly psychosis and lethal/malignant catatonia. The Norwegian psychiatrist Ottar Lingjærde and the Danish psychiatrist Erik Strömngren emphasized the life-saving effect of ECT<sup>33,34</sup>.

In ICD-10 the new term acute polymorphic psychosis was chosen for syndromes as above<sup>10</sup>. It is assigned to chapter F2 psychotic disorders. However, it has much in common with affective psychosis, and can be considered a subgroup of bipolar disorder as well. Thus, the Swedish psychiatrist Jan-Otto Ottosson, in his textbook from 1983 to 2015, uses cycloid syndrome as a synonym for polymorphic psychosis, and classifies it as a variant of bipolar disorder<sup>35</sup>.

The terms paranoid psychosis and paranoid schizophrenia are often used interchangeably, the latter preferentially later in an illness course or if hallucinations are present. However, one should always suspect hidden mood disorder behind persecutory and bodily delusions<sup>15</sup>.

The extensive concept of schizophrenia was increasingly criticized, particularly in the USA, because most so-called schizophrenic symptoms, taken alone and in cross-section, had little validity for diagnosis, prognosis, or treatment response. Moreover, it resulted in underdiagnosis of affective illnesses and compromised clinical treatment and research<sup>17,25</sup>. In 1980 this critic was taken account of in DSM-III<sup>36</sup>. The hierarchy of Jaspers<sup>2</sup> was reversed by making depressive and manic/hypomanic symptoms exclusion criteria for schizophrenia, and it was emphasized that so-called mood incongruent psychotic symptoms, as delusions and hallucinations about persecution and influence, could occur in affective disorders<sup>36</sup>. In 1993, affective syndromes as exclusion criteria for schizophrenia were also included in ICD-10<sup>10</sup>.

Mood symptoms are often difficult to acknowledge, and the clinician's education, experience and preconceptions are crucial<sup>37</sup>. Thus, referring to studies on the specificity of Bleulerian symptoms, Andreasen and Akiskal wrote: "Typically, clinicians decided that the patient had schizophrenia or depression and then characterized his affect accordingly"<sup>22</sup>. When psychologist Arnhild Lauveng<sup>38</sup> is memorizing to "... the big greyness ... how the world lost its colours and I was afraid of being dead", she thinks this must be "affective flattening" and "prodromal syndrome", phrases she has learned to be related to schizophrenia. However, such symptoms indicate severe depression as well<sup>8,9</sup>, and "prodromal syndrome" may develop in different directions, most often to a non-psychotic mood disorder<sup>39</sup>. Lauveng<sup>38</sup> also describes a phase compatible with hypomania prior to her psychosis.

Pathologic depression is not common sadness, but the mental pain that cannot be fully defined. Consequently,

"the clinical disorders of affect struggled for recognition", and those afflicted are often "unable to behave as a rational observer"<sup>3</sup>. The patient may deny depression and the clinician may end up characterizing the patient's mood as "neutral". The patient doesn't necessarily look like being depressed and might even appear lively if he/she has a bipolar disorder or temperament. Depression may become more obvious when psychotic symptoms diminish, however, may then be explained as "post-psychotic depression"<sup>40</sup>. Thoughts and emotions are always intermingled and being psychotic with "neutral" mood seems anti-intuitive.

Many words and phrases have been used in diagnostic criteria for pathological depression – sad, dysphoric, depressed, blue, low, down in the dumps, despondent, hopeless, irritable, fearful, worried, anxious, discouraged, don't care, loss of pleasure/enjoyment, loss of interest<sup>2</sup>. However, none is fully adequate. The "Vienna research criteria" are based on objective signs – changes in affectivity, emotional resonance, drive, and biorhythm – resembling "negative symptoms"<sup>2</sup>. Anhedonia is central to depression<sup>3</sup>, as are impaired self-respect, self-esteem, self-love, and self-preservation. Suicide is often the only way out of frantic hopelessness, emotional pain, ruminative flooding and (near) psychotic somatization<sup>41</sup>.

DSM is improved in the 5<sup>th</sup> edition - the subgroups of schizophrenia have been removed, catatonia has got its own codes, and psychotic symptoms are described in a separate chapter, indicating that there are no psychotic symptoms specific of schizophrenia or other disorders<sup>1</sup>. The category diagnosis, particularly of psychotic and mood disorder, may have contributed to lack of progress, because these disorders overlap, are not static and develop in stages<sup>5,39</sup>. Thus, Timothy Crow has for more than 30 years argued for a continuum extending from unipolar, through bipolar affective illness and schizoaffective psychosis, to typical schizophrenia, with increasing degrees of defect<sup>42</sup>. Tesli et al. include affective psychoses in "one common broad psychosis spectrum", supported by genetic and brain studies<sup>43,44</sup>.

Van Os and Kapur<sup>7</sup> suggest the rating of the five dimensions of positive symptoms, negative symptoms, neurocognitive alterations, mania and depression, in addition to category diagnosis. In my opinion, motor and vegetative symptoms should be included as separate dimensions. The Research Domain Criteria (RDoC), implies relating neurobiological findings (genes, molecules, cells, circuits, physiology) to behavior, self-report and treatment effects independent of predefined categories, to find new constellations or dimensions with better validity<sup>43</sup>.

Various treatment options may be indicated across diagnostic categories. Differences in response rate between groups cannot be relied upon when treating an individual

patient. Although antipsychotics generally are the first choice of drug for psychotic symptoms, antidepressants may reduce negative symptoms and suicide in schizophrenia<sup>45</sup> and improve cognitive function in psychotic disorders<sup>46</sup>. In one study antidepressants reduced transition to psychosis in high-risk subjects more than antipsychotics<sup>47</sup>, consistent with psychotic experiences being a marker of affective dysregulation<sup>48</sup>. Response to lithium may appear unexpectedly across diagnostic borders<sup>49</sup>. Electroconvulsive therapy may be indicated in all severe psychotic states. Chronicity and suicide justify the classical question of quality control: "Was the right thing done, and was it done right?"<sup>50</sup>. All methods must be considered as soon as possible in (near) psychotic states, because these states are more serious than the risks of treatment trials.

The concept of schizophrenia may disappear and the differential diagnosis of psychoses be characterized as a failure. However, there are signs of rebuilding. The "firewall" between psychotic and affective disorders should be replaced by descriptions of dimensions and illness spectra. Phrases like "neutral" and "flat" affect/mood should be avoided due to the difficulty in acknowledging pathological mood. Chronic, often sub-optimally treated, severe/psychotic mood disorder may appear as schizophrenia or paranoid psychosis<sup>6,17,37</sup>, representing end-stages like heart failure after different heart diseases. This strengthens the importance of optimal treatment of mood disorders, which may be the most important cause of schizophrenia and other functional psychoses.

## References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5 ed. Arlington: American Psychiatric Association; 2013.
- Berner P, Gabriel E, Katschnig H, et al. Diagnostic criteria for functional psychoses. 2 ed. Cambridge: Cambridge University Press; 1992.
- Berrios GE. The history of mental symptoms. Cambridge: Cambridge University Press; 1996.
- Goodwin F, Redfield Jamison K. Manic-depressive illness: bipolar disorder and recurrent depression. 2 ed. Oxford: Oxford University Press; 2007.
- Heckers S, Barch DM, Bustillo J, et al. Structure of the psychotic disorders classification in DSM-5. *Schizophr Res*. 2013;150(1):11-4.
- Lake CR. Schizophrenia is a misdiagnosis: implications for the DSM-5 and the ICD-11. New York: Springer; 2012.
- van Os J, Kapur S. Schizophrenia. *Lancet*. 2009;374(9690):635-45.
- Cohen AS, Najolia GM, Kim Y, Dinzeo TJ. On the boundaries of blunt affect/allogia across severe mental illness: implications for Research Domain Criteria. *Schizophr Res*. 2012;140(1-3):41-5.
- Messinger JW, Treméau F, Antonius D, et al. Avolition and expressive deficits capture negative symptom phenomenology: implications for DSM-5 and schizophrenia research. *Clin Psychol Rev*. 2011;31(1):161-8.
- World Health Organization. The ICD-10 classification of mental and behavioural disorders: diagnostic criteria for research. Geneva: WHO; 1993.
- Der G, Gupta S, Murray RM. Is schizophrenia disappearing? *Lancet*. 1990;335(8688):513-6.
- Lake CR, Hurwitz N. Schizoaffective disorder merges schizophrenia and bipolar disorders as one disease--there is no schizoaffective disorder. *Curr Opin Psychiatry*. 2007;20(4):365-79.
- Fink M. Rediscovering catatonia: the biography of a treatable syndrome. *Acta Psychiatr Scand Suppl*. 2013;127(441):1-47.
- Fink M, Shorter E, Taylor MA. Catatonia is not schizophrenia: Kraepelin's error and the need to recognize catatonia as an independent syndrome in medical nomenclature. *Schizophr Bull*. 2010;36(2):314-20.
- Lake CR. Hypothesis: grandiosity and guilt cause paranoia; paranoid schizophrenia is a psychotic mood disorder; a review. *Schizophr Bull*. 2008;34(6):1151-62.
- Lasalvia A, Penta E, Sartorius N, Henderson S. Should the label "schizophrenia" be abandoned? *Schizophr Res*. 2015;162(1-3):276-84.
- Lipton AA, Simon FS. Psychiatric diagnosis in a state hospital: Manhattan state revisited. *Hosp Comm Psychiatry*. 1985;36(4):368-73.
- Kumbier E, Herpertz SC. Helmut Rennert's universal genesis of endogenous psychoses: the historical concept and its significance for today's discussion on unitary psychosis. *Psychopathology*. 2010;43(6):335-44.
- Sedler MJ, ed. Schoelly M-L, trans. The legacy of Ewald Hecker: a new translation of "Die Hebeephrenie". *Am J Psychiatry*. 1985;142(11):1265-71.
- Kraepelin E, Barclay RM, Robertson GM, trans. Manic-depressive insanity and paranoia. Bristol: Thoemmes Press; 1921/2002.
- Bleuler E. Brill AA, trans. Textbook of psychiatry. New York: Macmillan & Co; 1924.
- Andreasen NC, Akiskal HS. The specificity of Bleulerian and Schneiderian symptoms: a critical reevaluation. *Psychiatr Clin North Am*. 1983;6(1):41-54.
- Kraepelin E. Patterns of mental disorder. In: Hirsch SR, Shepard M, eds. Marshall H, trans. Themes and variations in European psychiatry an anthology. Bristol: John Wright & Sons Ltd; 1920/1974:7-30.
- Schneider K. Clinical Psychopathology. New York: Grune & Stratton; 1959.
- Pope Jr HG, Lipinski Jr JF. Diagnosis in Schizophrenia and Manic-Depressive Illness. *Arch Gen Psychiatry*. 1978;35(7):811-28.
- Kasanin J. The acute schizoaffective psychoses. *Am J Psychiatry*. 1933;90(1):97-126.
- Bergem AL, Dahl AA, Guldberg C, Hansen H. Langfeldt's schizophreniform psychoses fifty years later. *Br J Psychiatry*. 1990;157:351-4.
- Kleist K. Cycloid, paranoid, and epileptoid psychoses and the problem of degenerative psychoses. In: Hirsch SR, Shepard M, eds. Marshall H, trans. Themes and variations in European psychiatry: an anthology. Bristol: John Wright & Sons Ltd; 1928/1974:297-331.
- Leonhard K. Cycloid psychoses - endogenous psychoses which are neither schizophrenic nor manic-depressive. *J Ment Sci*. 1961;107(449):633-48.
- Pillmann F, Arndt T, Ehrh U, et al. An analysis of Wernicke's original case records: his contribution to the concept of cycloid psychoses. *Hist Psychiatry*. 2016;11(44):355-69.
- Healy D. Mania. Baltimore: John Hopkins University Press; 2008.
- Retterstøl N. The Scandinavian concept of reactive psychosis,

- schizophreniform psychosis and schizophrenia. *Psychiatr Clin (Basel)*. 1978;11(3):180-7.
33. Strömberg E. Psychogenic psychoses. In: Hirsch SR, Shepard M, eds. *Themes and variations in European psychiatry: an anthology*. Bristol: John Wright & Sons Ltd; 1968/1974:97-117.
  34. Lingjærde O. Delirium acutum: Beitrag zum Studium der Pathogenese und der Therapie. *Arch Psychiatr Nervenkr Z Gesamte Neurol Psychiatr / Arch Psychiatr Zeitschr Neurol*. 1954;192(6):599-612.
  35. Ottosson J-O. *Psykiatri*. Stockholm: Lieber AB; 2015.
  36. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 3 ed. Washington, D.C.: American Psychiatric Association; 1980.
  37. Meyer F, Meyer TD. The misdiagnosis of bipolar disorder as a psychotic disorder: some of its causes and their influence on therapy. *J Affect Disord*. 2009;112(1-3):174-83.
  38. Lauveng A. *I morgen var jeg alltid en løve [Tomorrow I was always a lion]*. Oslo: Cappelen; 2006.
  39. Johannessen JO, McGorry P. DSM-5 and the 'Psychosis Risk Syndrome': The need for a broader perspective. *Psychosis*. 2010;2(2):93-6.
  40. Jeczmierni P, Levkovitz Y, Weizman A, Carmel Z. Post-psychotic depression in schizophrenia. *Isr Med Assoc J*. 2001;3(8):589-92.
  41. Yaseen ZS, Kopeykina I, Gutkovich Z, et al. Predictive validity of the Suicide Trigger Scale (STS-3) for post-discharge suicide attempt in high-risk psychiatric inpatients. *PLoS One*. 2014;9(1):e86768.
  42. Crow TJ. The continuum of psychosis and its implication for the structure of the gene. *Br J Psychiatry*. 1986;149:419-29.
  43. Morris SE, Rumsey JM, Cuthbert BN. Rethinking mental disorders: the role of learning and brain plasticity. *Restor Neurol Neurosci*. 2014;32(1):5-23.
  44. Tesli M, Espeseth T, Bettella F, et al. Polygenic risk score and the psychosis continuum model. *Acta Psychiatr Scand*. 2014;130(4):311-7.
  45. Tiihonen J, Mittendorfer-Rutz E, Torniainen M, et al. Mortality and cumulative exposure to antipsychotics, antidepressants, and benzodiazepines in patients with schizophrenia: an observational follow-up study. *Am J Psychiatry*. 2016;173(6):600-6.
  46. Steen NE, Aas M, Simonsen C, et al. Serum level of venlafaxine is associated with better memory in psychotic disorders. *Schizophr Res*. 2015;169(1-3):386-92.
  47. Fusar-Poli P, Frascarelli M, Valmaggia L, et al. Antidepressant, antipsychotic and psychological interventions in subjects at high clinical risk for psychosis: OASIS 6-year naturalistic study. *Psychol Med*. 2015;45(6):1327-39.
  48. van Os J. The many continua of psychosis. *JAMA Psychiatry*. 2014;71(9):985-6.
  49. Gualtieri CT. *Brain injury and mental retardation: psychopharmacology and neuropsychiatry*. Philadelphia: Lippincott Williams & Wilkins; 2002.
  50. Wyszewianski L. Quality of care: past achievements and future challenges. *Inquiry*. 1988;25(1):13-22.