Research Article

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Are some DSM5 diagnosis complications of other psychiatric disorders?

Abstract

This article offers evidence supported by clinical observations to propose that many DSM 5 psychiatric disorders may represent complications of other psychiatric disorders. Evidence is presented to suggest that DSM 5's linear architecture is inadequate to measure the dynamic complexity of brain function. Of significance is the butterfly effect of sensitive dependence on initial errors to trigger major delayed complications that may render DSM 5 a potentially harmful diagnostic tool. It seems that DSM's inherent deficit not to distinguish a disease from a disease complication is a major handicap for psychiatry.

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Introduction

Brain function and human behavior are dynamic, complex governed by physical laws and have a sensitive dependence upon initial conditions.[1,2] They are also governed by influences – eigenvalues – that are not easily observable (air, stress, electricity magnetic fields).[1,2] Consistent with theories proposed by Poincart and Lorenz complex systems are vulnerable to initial errors with exponentially magnified adverse outcome, a phenomenon recognized as the butterfly effect.[3] This suggests an early diagnostic error may lead to delayed adverse outcome.

DSM 5 (the diagnostic statistical manual of psychiatric disorders) is a diagnostic tool with profound influence on psychiatric research and treatment.[4] Is it possible that DSM five architecture is insensitive to physical laws? Furthermore is it possible that DSM 5 fails to differentiate causation from association, is dismissive of unobservable influences and cannot distinguish a disease from a disease complication? And does this suggest that because of its inherent flaws DSM five may delay progress or contribute to inappropriate diagnosis and treatment?

Methods

This paper will review diverse examples of DSM 5 diagnostic categories that may represent a complication or progression of a disease. Examples of erroneous DSM five disorders will be discussed under the following headings:

1. Addictive disorders develop after prefrontal cortex dysfunction

2. Chronic schizophrenia develops long after an initial untreated psychotic episode.

3. Amygdala dysfunction precedes some depressions.

The following examples are consistent with the observation that some psychiatric disorders may represent symptom clusters of disease progression:

Addictive disorders develop after prefrontal cortex dysfunction

1. 99% of people with addictive disorders suffer from a comorbid psychiatric condition.[5]

2. Logical behavior is a domain of prefrontal cortex function and is incompatible with recurrent self-destructive behavior. This suggests that prefrontal cortex dysfunction preceeds addictive behavior.

3. Neuro imaging evidence suggests distinct brain abnormalities are associated with diverse addictive disorders.

Chronic schizophrenia develops long after an initial untreated psychotic episode

Chronic schizophrenia may represent the end-stage of progressive degeneration similar to neurosyphilitic psychosis caused by treponemapallidum infections.[6,7] Is it possible that two major deficits of DSM architecture i.e. exclusion of pathophysiology and insensitivity to regional brain dysfunction have contributed to labeling different stages of disease progression as distinct disorders unrelated to each other?

Clinical observations are consistent with the hypotheses that psychosis of unknown origin may lead to provide schizophrenia.[8]

a. In general negative symptoms of schizophrenia develop long after initial psychotic episode and often associated with prefrontal cortex atrophy.

b. In general successful treatment of an acute episode prevents or diminishes severity of illness.

c. Predominant hereditary influences are absent.

Amygdala dysfunction precedes some depressions

Amygdala dysfunction preceeds some depressions.[9] Animal studies also suggest the emergence of symptoms and signs of heightened stress consistent with progressive degeneration. The unpredictable chronic mild stress (UCMS) model mimics the influence of environmental stressors contributory to depressive pathology and a timeframe or antidepressant response.[10] It has been shown that UCMS induces a depressive like syndrome in mice consistent with progressive deterioration in coat state, reduced weight gain and increased agonistic and emotion related behaviors. Those symptoms were reversed by chronic administration of an antidepressant.[10]

Conclusion

4 major deficits handicap DSM5 with possible adverse consequences for research, social policy and clinical practice.

i. Failure to distinguish a disease from a disease complication.

ii. Inconsistent use of biological evidence to form a diagnosis.

iii. Dismissal of eigenvalues.

iv. Dismissal of pathophysiology

DSM 5 is flawed as a scientifically reliable instrument to diagnose neuropsychiatric disorders. The potential of adverse outcomes and their impact on people with neuropsychiatric disorders necessitate urgency to validate the observations of this study and develop scientifically valid diagnostic tools in psychiatry (Table 1).

Table 1 Major DSM 5 Deficits

S.no	Major DSM 5 Deficits
1	Does not recognize eigenvalues i.e. unobservable influences.
2	Does not distinguish a disease from a disease complication.
3	Does not differentiate causation from association.
4	Is dismissive of regional brain function and pathophysiology.

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Conflicts of interest

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