Abstract

Background: (DSM-5) has been the predominant diagnostic instrument in psychiatry. Aims: To show that several transparent DSM-5 flaws may contribute to “not easily observable” adverse effects. Method: We studied the Diagnostic Statistical Manual of Mental Disorders 5th edition (DSM -5) and International classification of diseases 10th edition (ICD-10). Also six major DSM five psychiatric disorders were assessed for diagnostic accuracy. Results: In comparison with ICD-10 with no apparent flaws, DSM -5 have major flaws including inaccurate diagnosis , inability to differentiate a complication from a disease, insensitivity to input from biochemical essays and imaging. Average DSM-5 accuracy was 62.5%. DSM -5 may contribute to inaccurate diagnoses and treatment for some people with substance abuse disorders.

Introduction

Since its first introduction in 1952 The Diagnostic and Statistical Manual of Mental Disorders (DSM) and the subsequent revised editions have become the predominant psychiatric diagnostic instrument [1].

The evolution and rising popularity of DSM has coincided with increasing awareness of crucial biological influences - regional brain function, brain plasticity, genetics and thermodynamic factors – in the pathogenesis of psychiatric disorders [2-5].

Diverse studies have revealed specific neuroimaging and genetic abnormalities in schizophrenia [6-8], chronic pain [9] depression [10-13], obsessive-compulsive disorder [14,15], bipolar disorder [16], antisocial behavior [17], autistic spectrum disorder [18] etc. In essence, modern tools of science have redefined our current knowledge of psychiatric disorders in the past half-century.

Method

DSM 5 was carefully studied and compared with ICD-10. Also six DSM 5 psychiatric disorders (schizophrenia, bipolar disorder, depression, social anxiety disorder, attention deficit disorder and substance use disorder) were reviewed for diagnostic accuracy in 4 categories (completeness and objectivity of data, inclusion of common etiology or pathophysiology, ability to differentiate a complication from a primary psychiatric disorder).

Results

1. Transparent flaws of DSM -5.
2. Diagnostic accuracy rating.
3. Not easily observable Harmful Effects of DSM-5.

1. Transparent flaws of DSM -5

Some of the major diagnostic flaws of DSM -5 are transparent and shown (Table 1) in comparison to ICD- 10 (19). They are:

A. Lack of scientific accuracy
B. Insensitivity to biology, pathophysiology, biochemical essays and neuroimaging.
C. Inability to differentiate a complication or progression of a disorder from a primary disorder.
D. DSM-5 may also discourage clinical judgments rooted in pathophysiology.

A. Lack of scientific accuracy: A medical diagnosis demands inclusion of clinical findings supported by objective data. Also, manifestations (signs and symptoms) without shared etiology or pathophysiology do not constitute disease or disorder. Thus, a DSM-5 deficit - absence of objective evidence or common etiology of a cluster of symptoms - renders some DSM-5 disorders false or "possible disorders in need of objective data to qualify as disorders .

A hypothetical example: A man diagnosed with alcohol use disorder consistent with DSM -5: Once, he drank more than he had intended to and he and his spouse argued often for she opposed drinking on religious or moral grounds. Not only the inclusion of subjective data but also the absence of objective data (blood alcohol or breathalyzer measurements) render his condition a manifestation and not a disorder. Furthermore, subjective input compromises the integrity of the diagnosis of alcohol use disorder.

B. DSM -5 is insensitive to etiology pathophysiology biochemical essays and imaging: DSM -5 does not utilize vital data of dynamic brain function (regional brain function, neuro- transmission, plasticity). Of major significance a DSM -5 trained clinician may be conditioned to

Correspondence to: Alen J Salerian MD, Neuroscience Center, 5325 Westbard Avenue Bethesda MD 20816, USA; Tel: 301-204-9004; E-mail: alensalerian@gmail.com

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apply deductive logic of linear thinking to complex dynamic brain functions to diagnose a scientifically invalid DSM-5 disorder.

A hypothetical example: A person diagnosed with attention deficit disorder without neuroimaging or laboratory studies may actually be a victim of early Alzheimer’s or some other condition that may present with symptoms resembling attention deficit disorder.

C. DSM-5 cannot differentiate a disease from a disease complication or progression and classifies them as comorbid conditions: In medicine, conditions with a shared etiology or pathophysiology (i.e., hypertension and hypertensive retinopathy, diabetes and diabetic neuropathy) are viewed as a disease and a complication of a disease. A distinction is made by specifying conditions as “due to”. Various brain dysfunctions identified as psychiatric disorders may lead to diverse conditions that may represent progression or complications of primary psychiatric disorders. DSM-5 classifies them as comorbid conditions. According to DSM there is no such thing as progression or complication of psychiatric disorders.

Hypothetical cases: Someone with attention deficit disorder developing substance use disorder. Or for a man with social anxiety disorder developing alcohol use disorder as a complication of his unrecognized and untreated social anxiety disorder. According to DSM-5 both conditions would be classified as two independent psychiatric disorders.

D. DSM-5 discourages clinical decisions rooted in pathophysiology: In medicine, presenting symptoms may facilitate clinical decisions consistent with presumed pathophysiology or etiology. Heuristics- medical short cuts prompted by signs and symptoms are built upon pathophysiological associations. Bulging eyes and weight loss may help consider hyperthyroidism. DSM-5 makes pathophysiology irrelevant. For, it simply requires a collection of symptoms to represent a disorder.

A hypothetical case: A woman with symptoms of anhedonia, excessive worry, insomnia, diminished motivation, initiative and energy, passive suicidal thoughts and recent history of drinking. After a two month long alcohol overdose she stopped drinking a week before visiting a psychiatrist.

DSM-5 diagnosis: Major depression and Alcohol use disorder.

From a psychobiological perspective her symptoms may be consistent with “prefrontal cortex hypo function with low dopamine” often associated with depressive symptoms and lack of mastery of impulses. Alcohol induced euphoria-with elevated dopamine may counteract her underlying hypo dopaminergic state contributory to her poor judgment, impulse control and alcohol overdose. Unlike International classification of diseases, DSM-5 discourages a hypothetical yet physiologically sound discussion.

2. DSM-5 accuracy scale results

Overall accuracy based upon six major DSM-5 psychiatric disorders was 62.5%. Completeness was 132 vs maximum possible score 240, objectivity 230 vs maximum possible score 240, inclusion of etiology and pathophysiology 6 vs maximum possible score of 60, ability to differentiate a diagnosis a complication from a primary psychiatric disorder 6 vs maximum possible score 60.

3. Not easily observable DSM-5 harmful effects

A. DSM-5 Classifies Complications As Comorbid Conditions.

B. DSM-5 may contribute to unscientific and wasteful research.

C. DSM-5 may promote moral, political and religious views to contaminate public health policy.

A. DSM-5 classifies complications as comorbid conditions: A person with a primary psychiatric disorder (i.e., social anxiety disorder or attention deficit disorder) may develop a substance use disorder as a complication of his or her primary disorder. Due to DSM-5’s flawed architecture, this highly realistic phenomenon is classified as an independent coexisting illness. Paradoxically, someone with substance use disorder may receive less than optimal treatment partly because, some therapeutic strategies may discourage utilization of habit forming pharmacological agents (i.e., benzodiazepines or amphetamine salts for patients with past or present diagnoses of substance abuse.

A Hypothetical Case: A young adult with comorbid attention deficit disorder and cocaine use disorder. This person may not receive optimal psychopharmacological treatment due to concerns about addiction possibly discouraging the use of psycho stimulants. Of psychobiological significance ADD associated prefrontal cortex hypofunction may predispose some people to substance abuse and psycho stimulants have been shown to be effective to treat ADD.

B. DSM-5 may contribute to unscientific wasteful research: A diagnostic instrument that may produce inaccurate results may mislead research, waste human and financial resources and delay scientific progress.

C. DSM-5 may promote prejudicial influences to contaminate diagnosis and treatment and public health policy: The DSM’s heavy reliance on subjective input and exclusion of medical and biological data may contribute to morality, politics or religion to influence both diagnoses and treatment of psychiatric disorders and indirectly influence public health policy.

Of significant DSM-5 may artificially increase the recognition and diagnoses of psychiatric disorders that present with visual and easily observable manifestations and may decrease the recognition and diagnosis of those disorders that require complex conceptualization and sophisticated diagnostic methods.

The exacerbation of DSM-5’s heavy reliance on subjective and easily observable symptoms by it’s exclusion of objective evidence in diagnosing alcohol and substance use disorders might have led to possibly heightened and selective fear substance use disorders and contributing to a hostile environment for people with those disorders. Collectively, the misrepresentation of complications as disorders and the relative ease in diagnosing substance use disorders might have subjectively influenced therapeutic and policy decisions-extrordinarily restrictive and counter therapeutic regulations limiting optimal care for addiction in America- void of scientific objectivity. A case in example is the medically questionable restrictions to easy access to methadone and buprenorphine.

Discussion

This study suggests DSM-5 consistently excludes critical data about brain function (Table 1), has several serious flaws (Table 2) and its validity and benefits are compromised by several not easily observable harmful effects (Table 3).
It may be possible to argue that despite the absence of crucial pathophysiological data, clinical observations may be sufficient to diagnose a psychiatric disorder. That may be true for some of the conditions yet the overall validity of DSM-5 is compromised because of numerous misrepresentations of psychiatric manifestations as psychiatric disorders.

It can also be argued that various flaws are insignificant or inconsequential.

This is a point of emphasis consistent with modern physics and complexity theory [19]: Individually, by itself, each flaw may be dismissed as inconsequential or insignificant. Yet, collectively, they may represent an extraordinary risk of profound adverse consequences (Table 4). Of significance, possible harm from DSM’s delayed and not easily observable effects seem to be consistent with the butterfly effect of minor initial errors in complex systems [19]. Lorenz theorized “sensitive dependence of complex systems on initial events” and proposed that the delayed emergence of catastrophic consequences of minor events, such as a butterfly flapping its wings off the Amazon might trigger a hurricane in Kansas sometime later [19]. Although a possible DSM induced butterfly effect remains hypothetical, sadly DSM induced harmful effects seem to be real.

For public safety a possible correlation between the DSM flaws and lack of progress in psychiatry should deserve scientific scrutiny. For, we may observe that some of the benchmarks of progress in psychiatry (annual deaths from suicide and illicit drugs) in contrast to the measurable gains against cancer and cardiovascular mortality indicate that the ravaging effects of disease have not been as vigorously combated substance use disorders and illicit drugs.

| Table 2. DSM-5 accuracy* |
|---------------------------|
| Based upon Schizophrenia, Bipolar disorder, Depression, Attention deficit disorder, Substance use disorder and Social anxiety Disorder |
| Completeness of input (4items) | (Max Score=240) |
| 1. History | 10.5=50 |
| (10.5=50) + (1.1=1) = 51 |
| 2. PE | 10.6=60 |
| 3. Biochemical data | 10. 6=60 |
| 4. Imaging data | 10.6=60 |
| 231 |
| Objective of input (4items) | (Max Score=240) |
| 1. History | 10.6=60 |
| 2. PE | 10.6=60 |
| 3. Biochemical data | 1.6=6 |
| 4. Imaging data | 1.6=6 |
| 132 |
| Shared etiology or pathophysiology (1item) | (Max Score= 60) |
| 1.6=6 |
| Ability to differentiate (1 item) | (Max Score=60) |
| primary disorder vs complication | 6 |
| Accuracy score: | 375 |
| Best possible score: | 600 |
| Accuracy %42.5 |

Scoring: On a scale of 1 to 10 with 1 representing the worst and 10 representing the best, rate six DSM 5 disorders (schizophrenia, bipolar disorder, attention deficit disorder, depression, social anxiety disorder and substance use disorder) for diagnostic accuracy. For each psychiatric disorder rate 4 categories (completeness of input, objectivity of input, evidence for shared etiology and pathophysiology, and inability to differentiate a complication from a primary psychiatric disorder). Both completeness and objectivity of input have 4 subcategories each (history, physical exam, biochemical essays and imaging) to be rated independently.

* This scale has not been validated yet.

May Lead To

- Underrepresentation of population with primary psychiatric disorders and overestimating the number of people with primary substance use disorders.
- Less than optimal treatment for people with dual diagnosis of a primary psychiatric disorder and an alcohol or substance use disorder.
- Overestimation of morbidity and mortality from substance use disorders and underestimation of morbidity and mortality from psychiatric disorders.
- Draconian measures - criminalization of psychiatry and pain medicine - to combat substance use disorders and illicit drugs.
- Reduced access to controlled substances, epidemics of deaths by suicide and heroin overdose.

Table 4. A Hypothetical butterfly effect of a minor DSM flaw

Wrongful classification of primary psychiatric disorders and their complications as independent co-existing disorders

Table 4. Diagnostic accuracy for 6 major DSM 5 disorders in 4 categories

| CD | S | BD | D | SA | ADD | SU | Total |
|----|---|----|---|----|-----|----|-------|
| H  | 10| 10| 10| 10| 10  | 1  | 51    |
| P  | 10| 10| 10| 10| 10  | 10 | 60    |
| BE | 1 | 1 | 1 | 1 | 1   | 1  | 6     |
| I  | 1 | 1 | 1 | 1 | 1   | 1  | 6     |

| OD | S | BD | D | SA | ADD | SU | Total |
|----|---|----|---|----|-----|----|-------|
| H  | 10| 10| 10| 10| 10  | 1  | 51    |
| P  | 10| 10| 10| 10| 10  | 10 | 60    |
| BE | 1 | 1 | 1 | 1 | 1   | 1  | 6     |
| I  | 1 | 1 | 1 | 1 | 1   | 1  | 6     |

Accuracy Score: 375
Best Possible Score: 600
Accuracy %62.5

Coding: S=Schizophrenia; BD=Bipolar disorder; D=Depression; ADD=Attention deficit disorder; SA=Social anxiety disorder; SU=Substance use disorder; CD= completeness of data; OD= objective data of; H=History; P=Physical; BE=Biochemical assays; I= imaging; E+P= etiology and pathophysiology; D= Ability to differentiate complication from the primary psychiatric disorder

Our knowhow and psychiatric knowledge make the development of a new diagnostic instrument similar to ICDM 10 feasible. Yet, this noble task is beyond the narrow scope of this article.

Conflict of interest statement

This is to confirm that I have no conflict of interest in the publication of this manuscript. I also confirm that I have not received any monies or funds for the preparation of this manuscript.
References

1. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. 2013. American Psychiatric Association. Washington DC.

2. Meyer JS, Quenzer LF (2005) Psychopharmacology. Sinauer Ass Sunderland Mass.

3. Nolte J (2008) The Human Brain. An introduction to its functional anatomy; Mosby. Elsevier Publications.

4. Ramón de la Fuente J, Nicodimi H (1993) [Biological markers in psychiatry]. Acta Psiquiatr Psicol Am Lat 39: 117-122. [Crossref]

5. Gottron MI, Gould TD (2003) The endophenotype concept in psychiatry: etymology and strategic intentions, Am J Psychiatry 160: 636-645. [Crossref]

6. Weinberger DR, Berman KF, Zee RF (1986) Physiological dysfunction of dorsolateral PF in schizophrenia regional cerebral blood flow (rCBF) evidence. Arch Gen Psychiatry 43: 114 – 124. [Crossref]

7. Patel NH, Vyas NS, Puri BK, Nijran KS, Al-Nahhas A (2010) Positron emission tomography in schizophrenia: a new perspective. J Nucl Med 51: 511-520. [Crossref]

8. Andreasen NC, Nastallah HA, Dunn V, Olson S, Grove W, et al. (1986) Structural abnormalities in the frontal system in schizophrenia. A magnetic resonance imaging study. Arch Gen Psychiatry 43: 136 – 144. [Crossref]

9. Apkarian AV, Sosu Y, Sonty S, Levy RM, Harden RN, et al. (2004) Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. J Neurosci 24: 10416-10415. [Crossref]

10. Mayberg H (1997) Limbic frontocortical dysregulation: A proposed model of depression. J Neuropsychiatry Clin Neurosci 9: 471 – 481. [Crossref]

11. Sible E, Wang Y, Joeven-Waldorf J, Gaiteri C, Surget A, et al. (2009) A molecular signature of depression in the amygdala. Am J Psychiatry 166: 1011-1024. [Crossref]

12. Salerian AJ, Altar CA (2012) The prefrontal cortex influence over subcortical and limbic regions governs antidepressant response by $N=H/(M+R)$. Psychiatry Res 204: 1-12. [Crossref]

13. Pizzagelli DA (2011) Frontal cortex cingulate dysfunction in depression: toward biomarkers of treatment response. Psychopharmacology 36: 183-186. [Crossref]

14. Buchsbaum MS, Someya T, Wu JC, Tang CY, Bunney WE (1997) Imaging bipolar illness with positron emission tomography and magnetic resonance imaging. Psychiatric Annals 27: 489-495.

15. Pena-Gaario J, Ruiperez-Rodrigue MA, Barro-Loscertales A (2010) The neurobiology of obsessive-compulsive disorder: new findings from functional magnetic resonance imaging. Rev Neurol 50: 541–510. [Crossref]

16. Menzies L, Chambalain SR, Laird AR, Thelen SM, Sahakian BJ, et al. (2008) Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: the orbito-fronto-striatal model revisited. Neurosci Biobehav Rev 32: 525–549. [Crossref]

17. Stalenheim EG (2004) Long-term validity of biological markers of psychopathy and criminal recidivism: follow-up 6-8 years after forensic psychiatric investigation. Psychiatry Res 121: 281–291. [Crossref]

18. Konstantareas MM, Homatidis S (1999) Chromosomal Abnormalities in a Series of Children with Autistic Disorder. J Autism Dev Disord 29: 275–285. [Crossref]

19. Amaral Cda S, da Silva-Boghossian CM, Lelo AT, Colombo AP (2011) Evaluation of the subgingival microbiota of alcoholic and non-alcoholic individuals. J Dent 39: 728-738. [Crossref]

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