Critiquing the Psychiatric Model

Edited by
Eric Maisel and Chuck Ruby

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This book first published 2022
Ethics International Press Ltd, UK
British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Print Book ISBN: 978-1-871891-69-0
eBook ISBN: 978-1-871891-70-6

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EDITOR'S INTRODUCTION

Eric Maisel

For those of you familiar with the concerns of those who have their doubts about the ethics, tactics, logic and legitimacy of what we might call the mental health establishment, there may prove to be precious little new in the volumes of the Ethics International Press Critical Psychology and Critical Psychiatry series.

You may already know all you need to know about psychiatry as a pseudoscience and a pseudo-medical specialty, about psychotherapy as expert talk or as something else again, about the validity or invalidity of psychological testing, about the differences between "diagnosing and treating mental disorders" and the mere collecting of "symptoms" into "symptom pictures" which then get affixed a convenient label, and so on.

But even if you know all this, you may find it convenient to hear from many voices in one place. At the very least, you can now point your friends and colleagues who haven't had the chance to deconstruct all of this to a series where they can familiarize themselves with the basics and get their feet wet, so to speak, in these troubling seas.

And, even if you do know this territory pretty well, there may be some new things for you to encounter, learn, and experience. No one can keep up on all the world's mischief. And we have invited world voices into this series, so some of the perspectives presented in these volumes may actually prove new to you.

For the series premier, two volumes are appearing simultaneously as companion volumes, one called *Critiquing the Psychiatric Model* and a second called *Humane Alternatives to the Psychiatric Model*. We thought that trying to put all this material into a single volume would prove too unwieldy (and rather expensive for the reader to purchase) and that two companion volumes would make for a nice alternative. We anticipate many more volumes appearing in this ongoing series; if you like to know more about those proposed volumes, and more about possibly contributing to one of them, please visit here: *https://ethicspress.com/pages/the-ethics-international-press-critical-psychology-and-critical-psychiatry-series*

Trying to explain why the concerns explored in these volumes should be located in the territory of "ethics" would take us down paths we do not need to travel, into the definitional morasses of how to get from "what

is" to "what ought to be," whose values are being promoted, and, most basically, what do we mean by "ethical"? Let me just present a few basic points as to why the concerns presented in these volumes are ethical in nature:

- If you claim that you are doing medicine, or suggest that you are doing medicine without actually making that claim, and you aren't doing medicine, that amounts to an ethical matter, wouldn't you say?

- If, as an answer to a question on a psychological test, I tell you that I prefer something, say that I like solitude, and then you repeat back to me that I prefer that something, just changing the wording and claiming, say, that I am an introvert, that is a linguistic transaction of a certain sort and not a test. What is being "tested" in that transaction? To the extent to which psychological tests are not genuine tests, or really nothing like tests at all, that is an ethical matter, wouldn't you say?

- If you claim that certain chemicals are "treating" a "disorder" but in fact they are just chemicals with powerful effects, some of those effects sometimes desirable and many of those effects regularly undesirable, that is an ethical matter, wouldn't you say? It is not just a linguistic matter or a language game to call a chemical a "medication" when it isn't—it is also an ethical issue, yes?

- If I claim that I am "practicing psychotherapy" and that at the core of that activity is the "diagnosing and treating of mental disorders," and the whole construct is fishy, that is an ethical matter, wouldn't you say? If you are putting your psychological and emotional life in my hands, it would be nice if you knew something about the psychological and emotional life of human beings and had more in your arsenal than a symptom checklist, an ability to listen, and some rote questions, yes? To put the matter another way, if someone calls himself or herself an expert at something and isn't, that is an ethical matter, yes?

Certainly, society giving us the right to electroshock you is an ethical matter. So is society giving us the right to incarcerate you for your unusual but not illegal behaviors. So is society giving us the right to label you with some psychiatric label because of your political views, as part of tactics of oppression, because you are a child and can't defend yourself from labeling (and the chemicals that will follow), and for other social and political motives. So is society denying the relationship between poverty and "poor mental health," denying the relationship between oppression and "poor mental health," denying that circumstances matter when it comes to your mental health, and in countless other ways denying that the realities of your life matter to your emotional wellbeing.

There is much that is wrong and there is much that ought to change. "Ought" is a value word squarely in the domain of ethics. If you agree that there is a lot that ought to change with respect to our mental health paradigms and practices, then you are agreeing that we are properly in the domain of ethics here. In Volume 1, we look (relatively briefly) at some of what is wrong. In Volume 2, we look (not at all comprehensively) at where changes might be made. We hope that you find these two volumes, and the series of which they are a part, both provocative and helpful.

We welcome feedback, we hope that you will promote these books in your networks, and we look forward to hearing from you if you think that you might like to contribute to a future volume, if you might perhaps like to take on the role of editor for a future volume, or if you might like to propose a future volume. We are happy to train a lens on any aspect of psychology and psychiatry that deserves some scrutiny. Come join us in this worthy endeavor.

EDITOR'S INTRODUCTION

Chuck Ruby

Shortly before the attacks on September 11, 2001, I started a second career as a psychologist after having served 20 years in the U.S. military. I remember being shocked and perplexed at how that apparently simple terrorist operation, carried out by just a few disaffected people with box cutters, brought a country to its knees.

I didn't know at the time, but I was about to be shocked and perplexed even more about another kind of attack, although this one had been underway for many years, and it has brought generations to their knees. As I slowly worked my way into the field of clinical psychology and private practice, I began to discover how much of the mental health industry had been using a similarly simple operation on people who ask for help, but who many times receive harm.

With initially good intentions, an ostensibly benevolent system of mental healthcare has unfolded into a system of control with the main purpose of judging the appropriateness of emotional distress and personal choices and then ensuring compliance with "appropriate" ways of being. But in a leap of logic, those emotions and choices are branded as illnesses. Once designated an illness, they are then seen as obvious targets of a paternalistic system of medicine. However, it is a "system of medicine" that is based on morality, not science.

We have been given the opportunity to talk in depth about this attack in The Ethics International Press Critical Psychology and Critical Psychiatry Series. The first two volumes are our attempt to set the stage and bring you to the same starting point. They are foundational in scope. In the first volume, we reveal the problem as it affects many aspects of society and in the second, we suggest alternative ways to understand and deal with the many serious difficulties that we all encounter in our lives' journeys. Other volumes will follow, focusing on narrower, finetuned aspects of the mental health industry, and solutions that are largely intended to restore a humane way of assisting our fellow travelers.

Much of what is contained in this series has been written elsewhere over the last several decades. But this is an attempt to bring it all together in one place. The goal is to focus and enhance our message to the multitude

of professionals and consumers who have yet to hear about the problem. There are many, many professionals within the mental health industry, including myself, my co-editor, and all the authors who contribute chapters to this series, who are trying to right this wrong. We are trying to reform the mental health industry away from control and coercion under the guise of medicine, and toward a truly voluntary and helpful endeavor that values the human right to self-determination. We want a system that is available for people who seek out help and respects those who do not want our help.

We encourage you to contact us if you are a kindred spirit. We are open to ideas for upcoming volumes in this series and chapters that would complete the volumes. To find out more about this series please visit: https://ethicspress.com/pages/the-ethics-international-press-critical-psychology-and-critical-psychiatry-series. Now, buckle up and enjoy!

Chuck Ruby, Ph.D., Lt Col (ret)
Psychologist
Executive Director
International Society for Ethical Psychology and Psychiatry

ANATOMY OF A FAILED PARADIGM OF CARE

Robert Whitaker

In 1980, when the American Psychiatric Association (APA) published the third edition of its *Diagnostic and Statistical Manual of Mental Disorders (DSM III)*, it adopted a disease model for diagnosing and treating mental disorders. The APA quickly set out to market its new model to the American public, and gradually this paradigm of care took root in developed countries around the world.

Forty years have now passed, and thus it is possible to assess what this revolution has wrought. What a review of the evidence reveals is this: research has failed to validate the disease model, and in terms of clinical outcomes, the model has proven to be a public health disaster.

Here are the markers of this scientific and clinical failure:

- The biology of major mental disorders remains unknown.
- The chemical imbalance theory of mental disorders didn't pan out.
- The search for genes hasn't turned up any finding of clinical use.
- The diagnoses in the *DSM* are understood to lack validity.
- Disability due to psychiatry disorders has jumped dramatically in country after country that has adopted this model of care.
- Long-term outcomes for major disorders have worsened in the past forty years.

The disease model: a commercial success

In 1984, Nancy Andreasen, who would soon become editor-in-chief of the *American Journal of Psychiatry, published a best-selling book titled The Broken Brain: The Biological Revolution in Psychiatry*. In it, she set forth the disease model that the APA had adopted when it published DSM III. "The major psychiatric illnesses are diseases," she wrote. "They should be considered medical illnesses just as diabetes, heart disease and cancer are.

The emphasis in this model is on carefully diagnosing each specific illness from which the patient suffers, just as an internist or neurologist would."[1]

At this same time, leaders in American psychiatry began telling of how major psychiatric disorders were due to chemical imbalances in the brain. Researchers, it seems, were discovering the very molecules that cause madness, depression, and other major disorders. "Our field is exploding with new information, optimism, and enthusiasm," said APA president Carol Nadelson in 1985. "Psychiatry has moved from a backwater to the forefront as a medical specialty, largely because of the research explosion, particularly in the neurosciences."[2]

When Eli Lilly brought Prozac to market in 1988, the public was told that depression was due to too little serotonin in the brain and that this new drug upped serotonergic activity, and thus could be said to fix a chemical imbalance in the brain. The drug was "like insulin for diabetes." Prozac was touted as a wonder drug, with the pill gracing the covers of *Newsweek* and *New York* magazines.[3,4]

In the mid 1990s, a second generation of "atypical antipsychotics" came to market, with these new drugs – Risperdal, Zyprexa, and Seroquel—said to balance dopamine and other neurotransmitters in the brain, and they too were presented to the public as breakthrough medications.

As these medications were marketed, the APA, with its publication of *DSM IV* in 1994, further expanded the pool of potential users of these drugs. Criteria for disorders were loosened, new diagnoses were added. Based on *DSM* criteria, researchers subsequently determined that 26% of American adults suffered "from a diagnosable mental illness in a given year."[5]

The publication of *DSM III* in 1980 also kicked off a boom in the medicating of American children and adolescents. This was when the diagnosis of attention deficit disorder was born, and soon parents were hearing that Ritalin helped balance brain chemistry in youth so diagnosed. *DSM IV* needed 86 pages to describe the many disorders that could afflict children and teenagers, and based on the diagnostic criteria in that manual, the Centers for Disease Control and Prevention concluded that 13 percent of youth 8 to 15 years old experienced a bout of mental illness each year.[6]

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[1] N. Andreasen, *The Broken Brain* (New York: Harper & Row, 1984), 29-30, 138.

[2] C. Adelson. "Response to the presidential address." *Am J Psychiatry* 142 (1985): 1009-14.

[3] G. Cowley, "Prozac: A Breakthrough Drug for Depression," *Newsweek*, March 26, 1990.

[4] F. Schumer "Bye-Bye, Blues," *New York*, December 18, 1989.

[5] R. Kessler. "Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the Naitonal Comorbidity Survey Replication." *Arch Gen Psychiatry* 62 (2005): 617-27.

[6] Centers for Disease Control and Prevention, National Health and Nutrition Examination Survey, 2012.

The disease model proved to be a resounding commercial success. In the United States, spending on psychiatric drugs increased from around $800 million in 1985 to $40 billion in 2011. This was a 50-fold increase in spending on psychiatric drugs.[7]

The scientific failure

At the heart of the disease model was the chemical imbalance story. This told of psychiatric disorders with known pathologies. Depression was due to too little serotonin. Schizophrenia was due to an overactive dopamine system. ADHD was due to too little dopamine. And so forth. Just as Andreasen had said in her book, psychiatric disorders were discrete illnesses of the brain.

However, the chemical imbalance theory had not arisen from the discovery of such abnormalities in patients diagnosed with these different disorders. Instead, it had arisen in the 1960s from an understanding of how antipsychotics and antidepressants acted on the brain. Antipsychotics blocked dopamine receptors in the brain, and thus researchers hypothesized that schizophrenia was due to too much dopamine activity. Antidepressants upped serotonergic activity in brain, and thus researchers hypothesized depression was due to too little serotonin. The thought was that the pathology for each disorder would be the opposite of the drug's mechanism of action, and once this hypothesis was born, it was applied to other psychiatric disorders, such as ADHD.

In the 1970s, researchers began efforts to see if patients diagnosed with schizophrenia or depression indeed suffered from a chemical imbalance in the brain. The low serotonin theory began to fall apart in 1984, when investigators at the National Institute of Mental Health (NIMH) concluded after an investigation of this type that "elevations or decrements in the functioning of serotonergic systems per se are not likely to be associated with depression."[8]

Further research into the low-serotonin theory of mental disorders failed to find any such abnormality in depressed patients (prior to their exposure to antidepressants), and in 1999, the editors of the APA's *Textbook of Psychiatry* declared the monoamine hypothesis dead (serotonin is a monoamine.) They noted that it had been a shaky hypothesis from the start. "Inferring neurotransmitter pathophysiology from an observed action of a class of medications is similar to concluding that because

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[7] IMS Health, annual reports, "Top Therapeutic Classes by U.S Spending."

[8] J. Maas. "Pretreatment neurotransmitter metabolite levels and response to tricyclic antidepressant drugs," *Am J Psychiatry* 141 (1984): 1159-71.

aspirin causes gastrointestinal bleeding, headaches are caused by too much blood and the therapeutic action of aspirin in headaches involves blood loss," they wrote. "Additional experience has not confirmed the monoamine depletion hypothesis."[9]

While investigations into dopamine function in schizophrenia patients produced a more nuanced story, research still failed to find that they regularly suffered from an overactive dopamine system prior to their being medicated. As Eric Nestler and former NIMH director Stephen Hyman wrote in their 2002 book *Molecular Psychiatry*, "there is no compelling evidence that a lesion in the dopamine system is a primary cause of schizophrenia.[10]

A few years later, Kenneth Kendler, coeditor-in-chief of *Psychological Medicine*, neatly summed up the bottom line: "We have hunted for big simple neurochemical explanations for psychiatric disorders and not found them."[11]

The search for genes has also proven futile. Although the public has regularly been told that there is a genetic component to mental disorders, large population studies determined that genetics accounted for less than 2.3% of the risk related to developing a psychiatric disorder.[12] UK researchers who conducted large gene-sequencing studies to assess the genetic risk of developing schizophrenia or an affective disorder reported that, in each case, their findings were "completely negative."[13]

Just as there are no biological markers useful for diagnosing psychiatric disorders, there are no genetic tests that can be used to assess increased risk for developing a psychiatric condition.

The demise of the chemical imbalance theory of mental disorders and the failure to find biological or genetic markers has led leaders in American psychiatry to acknowledge that *DSM* diagnoses "lack validity." In 2012, the chair of the *DSM IV* task force, Allen Frances, put it this way: DSM diagnoses "no longer seem at all reducible to simple diseases, but rather

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[9] S. Dubovsky, "Mood disorders," in *Textbook of Psychiatry*, edited by R. Hales, third edition (Washington, DC: American Psychiatric Press, 1999): 516.

[10] E. Nestler, *Molecular Neuropharmacology* (New York: McGraw Hill, 2001): 392.

[11] As cited by J. Lacasse. "Serotonin and depression: a disconnect between the advertisements and the scientific literature." *PLoS Med* 2 (2205):1211-16.

[12] A. Rammos. "The role of polygenic risk score gene-set analysis in the context of the omnigenic model of schizophrenia." *Neuropsychopharmacology* 44 (2019): 1562-59.

[13] T. Balakrishna. "Assessment of potential clinical role for exome sequencing in schizophrenia." *Schizophrenia Bulletin* 46 (2020): 328-335. D. Curtis. "Analysis of 50,000 exome-sequenced UK Biobank subjects fails to identify genes influencing the probability of developing a mood disorder resulting in psychiatric referral." *Journal of Affective Disorders*, 281 (2021): 216-219.

are better understood as no more than current convenient constructs or heuristics that allow us to communicate with one another as we conduct our clinical, research, educational, forensic, and administrative work."[14]

In short, the scientific literature tells of decades of research that failed to validate the disease model that the APA adopted when it published *DSM III*. The biological causes of major disorders remain unknown, the chemical imbalance theory never panned out, no significant genetic associations to mental illness have been found, and the diagnoses in the *DSM* are understood to be constructs, as opposed to validated disorders.

The rise in disability

The disease model that has been promoted to the American public-and in developed countries around the world—tells of great medical progress. If that is so, the burden of mental disorders could be expected to have declined in the past forty years, both in the United States and in other developed countries.

The opposite has occurred. The number of adults receiving disability payments for psychiatric disorders in the United States has soared during the "disease model" era, and similar increases have occurred in other countries that adopted this model of care.

In the United States, Social Security payments to support the "disabled" mentally ill come through two programs: Supplemental Security Income (SSI) and Social Security Disability Insurance (SSDI). In 1987, there were 1.25 million adults that received disability payments through these two programs because of a mental illness (a disability rate of one in every 194 Americans.) In 2019, the number of disabled mentally ill totaled 4.67 million, a disability rate of one in every 70 Americans. The disability rate nearly tripled over this 30-year period.[15]

The rise in the number of children and adolescents "disabled" by mental illness is even more striking. In 1987, there were 16,600 children under 18 years old that received a federal disability payment due to mental illness. This group comprised 5% of the total number of children receiving an SSI payment that year. In 2020, there were 717,907 children on the SSI disability rolls due to a mental disorder, accounting for 65% of the total number of disabled children.[16]

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[14] See R. Whitaker and L. Cosgrove, *Psychiatry Under the Influence* (New York: Palgrave MacMillan, 2015): 60-61.

[15] Social Security Administration, annual statistical reports on the SSDI and SSI programs, 1987-2019. As one in every seven SSDI recipients also receives an SSI payment, the total disability numbers are calculated as follows: SSI recipients + (.857 x SSDI recipients).

[16] SSI annual statistical reports, 1987 – 2020.

The story is much the same in other developed countries. Iceland, the U.K., Norway, Sweden, Denmark, Germany, Australia, and New Zealand have all reported marked increases in disability due to psychiatric disorders during the past 30 years.[17]

Worsening long-term outcomes

Schizophrenia

In the mid 1990s, a second-generation of "atypical" antipsychotics were brought to market. These drugs – Risperdal, Zyprexa, Seroquel and others—were touted as "breakthrough" medications that could help people diagnosed with schizophrenia recover and function fairly well in society. However, a meta-analysis of recovery rates for schizophrenia patients in the United States and other developed countries showed that schizophrenia outcomes have worsened since the introduction of the atypicals, and are now worse than at any time since the schizophrenia diagnosis was introduced more than a century ago.

In the first half of the twentieth century, slightly less than 20% of patients diagnosed with schizophrenia "recovered" and were able to function fairly well in society. Antipsychotics were introduced into asylum medicine in 1955, and recovery rates remained stable for the next 20 years. However, the recovery rate declined to 10% from 1976 to 1995, and since the introduction of the atypicals, the recovery rate has dropped even further to 6%.[18]

The best longitudinal study conducted in the modern era provides evidence why that is so. In the late 1970s and early 1980s, Martin Harrow and Thomas Jobe began following 200 psychotic patients that were admitted to two Chicago hospitals (one private and one public.) Theirs was a young cohort, with a median age of 23, and two-thirds were experiencing either a first or second hospitalization.

All patients were treated conventionally in the hospital with antipsychotics, and then, following their discharge, Harrow and Jobe periodically assessed how they were doing. Were they asymptomatic? Working? What were their social lives like? How was their cognitive function? And were they taking antipsychotic medication? At the end of 15 years, Harrow and Jobe

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[17] As cited by R. Whitaker, Anatomy of an Epidemic, Broadway Books: New York, 20152nd edition), 364. See also, R. Whitaker, presentation to the UK Parliamentary Working Group in May 2016: "Causation, Not Just Correlation, Increased Disability in the Age of Prozac." Available at: *https://www.madinamerica.com/wp-content/uploads/2017/01/Causation-not-just-correlation-.pdf*

[18] E. Jaaskelainen. "A systematic review and meta-analysis of recovery in schizophrenia." *Schizophrenia Bulletin* 39 (2013): 1296-1306.

still had 145 participants in their study, 64 diagnosed with schizophrenia and 81 with milder psychotic disorders.

At the end of two years, those in the schizophrenia group who had stopped taking their medication were doing slightly better than those on medication. Then, over the next 30 months, the collective fates of the two groups diverged. The off-med group continued to improve, and by the end of 4.5 years, 39% were in recovery and more than 60% were working. In contrast, the outcomes for the medicated group worsened during this period, and at the 4.5-year mark, only 6 percent were in recovery and few were working.

The recovery rate was more than six times higher for the unmedicated group, and it stayed that way for the rest of the study. At the end of 15 years, 40% of those off antipsychotics were in recovery, and more than half were working. Only 5% of those who were taking antipsychotics were in recovery—the very outcome that had become customary for medicated patients during the atypicals era.[19]

"I conclude that patients with schizophrenia not on antipsychotic medication for a long period of time have significantly better global functioning than those on antipsychotics," Harrow reported at the 2008 annual conference of the American Psychiatric Association.

In subsequent publications, Harrow and Jobe reported that those off medication were much better in every domain: they were less anxious, had better cognitive functioning, and more likely to be working. Most of those who were stable off medication at the 4.5-year assessment remained stable throughout the study, whereas those who were medication compliant throughout the study relapsed much more frequently and were much more likely to be actively psychotic at follow-up assessments.[20]

Other long-term studies of psychotic patients in the modern era have similarly reported higher recovery rates for those off antipsychotic medication.

- In a study of first-episode psychotic patients in the Netherlands, the recovery rate at seven years was 53% for those who tapered down to

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[19] M. Harrow. "Factors involved in outcome and recovery in schizophrenia patients not on antipsychotic medications." *Journal of Nervous and Mental Disease* 195 (2007):406-14.

[20] M. Harrow. "Do all schizophrenia patients need antipsychotic treatment continuously throughout their lifetime? A 20-year longitudinal study." *Psychological Medicine*, (2012):1-11. M. Harrow. "Does long-term treatment of schizophrenia with antipsychotic medications facilitate recovery?" *Schizophrenia Bulletin* 39 (2013): 436-38. M. Harrow. "Pharmacological Treatment for Psychosis: Emerging Perspectives." Presentation in Syracuse, NY, October 2, 2014. M. Harrow. "Does treatment of schizophrenia with antipsychotic medications eliminate or reduce psychosis?" *Psychological Medicine* 44 (2014):3007-16.

a low dose of an antipsychotic or got off the medication versus 17% for those who remained on a standard dose.[21]

- In a study of first-episode patients in Denmark, 74% of those off medication at the end of 10 years were asymptomatic and 37% were working, compared to a remission rate of 37% and an employment rate of 16% for those on antipsychotic medication.[22]

- In a study of Finnish schizophrenia patients born in 1966, 63% of those off medication in the year 2000 were asymptomatic and only 16% were on disability, whereas only 20% 0f those on medication were asymptomatic and 50% were on disability.[23]

Global mortality rates for schizophrenia patients have also worsened since 1980. In the 1970s, the standardized mortality rate (SMR) for schizophrenia patients stood at 1.84 (meaning that schizophrenia patients were nearly twice as likely to die in any one year compared to a cohort in the general population matched for age and gender.) This mortality gap grew to 2.98 during the 1980s and to 3.20 during the 1990s. Australian investigators, in an analysis of mortality rates in 25 nations, also found that the mortality gap was significantly higher in "developed" countries—the very societies that had adopted the disease model of care—than in lesser-developed countries.[24]

The mortality gap appears to have worsened since then. Finnish investigators reported that from 1995 to 2000, schizophrenia patients died at 4.5 times the rate of the general population.[25] A study of Swedish schizophrenia patients from 2006 to 2010 reported a SMR of 4.8.[26] Meanwhile, UK investigators reported that the SMR for schizophrenia patients increased by .11 points per year from 2000 to 2010, and then more rapidly from 2010 to 2014 (.34 points per year.)[27]

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[21] L Wunderink,."Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation of maintenance treatment strategy." *JAMA Psychiatry* 70 (2013):962-5.

[22] R. Wils, R. "Antipsychotic medication and remission of psychotic symptoms after a first-episode psychosis." *Schizophrenia Research* 182 (2017): 42-8.

[23] J. Moilanen. "Characteristics of subjects with schizophrenia spectrum disorder with and without antipsychotic medication – A 10-year follow-up of the Northern Finland 1966 Birth Cohort study ." *European Psychiatry* 28 (2013): 53-58.

[24] S. Saha. "A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time?" *Arch Gen Psychiatry* 64 (2007):1123-31

[25] M. Kiviniemi. "Mortality, disability, psychiatric treatment and medication in first-onset schizophrenia in Finland: the register linkage study. " Dissertation. Available at: *https://www.madinamerica.com/wp-content/uploads/2020/05/Finnish-National-Study.pdf*

[26] M. Torniainen. "Antipsychotic treatment and mortality in schizophrenia." *Schizophrenia Bulletin* 41 (2015):656-663.

[27] J. Hayes. "Mortality gap for people with bipolar disorder and schizophrenia: UK-based cohort study 2000–2014." *Br J Psychiatry* 211 (2017): 175-81.

Depression

In the era before the introduction of antidepressants, depression was understood to be an episodic disorder. Leaders at the NIMH emphasized this again and again in their writings in the 1960s and early 1970s. "Depression is, on the whole, one of the psychiatric conditions with the best prognosis for eventual recovery with or without treatment," wrote Jonathan Cole in 1964. Most depressive episodes, noted Dean Schuyler, head of the depression section at the NIMH, "will run their course and terminate with virtually complete recovery without specific intervention."[28]

However, after antidepressants began to be widely prescribed, at least a few clinicians noted that while their patients were getting better more quickly now, they were also relapsing more frequently. The drugs seemed to be causing a "chronification" of the disease. A Dutch physician, J.D. Van Scheyen, conducted a five-year study of depressed patients treated with and without antidepressants, and found that "systematic long-term antidepressant medication . . . exerts a paradoxical effect on the recurrent nature of the vital depression. In other words, this therapeutic approach was associated with an increase in recurrent rate and a decrease in cycle duration."[29]

By the mid 1980s, with outcomes for depressed patients worsening, many experts in mood disorders reconceptualized depression as a chronic disease. But rather than seeing this change as due to the use of antidepressants, they argued that depression had run a chronic course all along, and it was only now that researchers were discovering this fact.

Then, starting in 1994, Italian psychiatrist Giovanni Fava published a string of papers that put the focus back on antidepressants as a likely cause of this change in the long-term course of depression. Antidepressants upped serotonergic activity, and in compensatory response, the brain dialed down its own serotonergic activity, and this could "sensitize" the brain to depression, Fava wrote. "Antidepressant drugs in depression might be beneficial in the short term, but worsen the progression of the disease in the long term, by increasing the biochemical vulnerability to depression . . . Use of antidepressant drugs may propel the illness to a more malignant and treatment unresponsive course."[30]

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[28] See R. Whitaker, *Anatomy of an Epidemic*, Broadway Books: New York, 20152nd edition), 152-3.

[29] J. Van Scheyen. ""Recurrent vital depressions." *Psychiatria, Neurologia, Neurochirurgia* 76 (1973): 93-112.

[30] G. Fava. "Do antidepressant and antianxiety drugs increase chronicity in affective disorders?" *Psychotherapy and Psychosomatics* 61 (1994): 125-31. G. Fava. "Holding on: depression, sensitization by antidepressant drugs, and the prodigal experts." *Psychotherapy and Psychosomatics* 64 (1995): 57-61.G. Fava. "Potential sensitizing effects of antidepressant drugs on depression." CNS Drugs 12 (1999):247-56. G. Fava. "Can long-term treatment with antidepressant drugs worsen the course of depression?" *J Clin Psychiatry* 64 (2003): 123-33.

The NIMH then conducted what it hailed as the "largest" antidepressant trial ever, and the results confirmed that medicated depression indeed runs a chronic course. There were 4,041 "real world" patients enrolled into the STAR*D trial, most of whom were only moderately ill, and yet only 38% ever remitted, even for a short period of time, and at the end of one year, only 108 had remitted and stayed well and in the trial. This was a documented "stay-well" rate of 3%.[31]

Numerous longer-term studies have reported that depressed patients treated with antidepressants are more likely to remain symptomatic and functionally impaired than similar patients treated with non-drug methods (or no treatment at all.) Researchers in the UK, the Netherlands, and Canada have all reported such findings. A World Health Organization study conducted in 15 cities around the world did so as well.[32] Meanwhile, a NIMH study on the course of unmedicated depression found that 85% were recovered at the end of one year.[33]

That outcome, when reviewed alongside the STAR*D results, tells of drug treatment gone horribly wrong.

Bipolar disorder

There are two notable aspects to the bipolar story that has unfolded since the publication of DSM III. The first is that bipolar disorder, which used to be called manic-depressive illness, now runs a much more chronic course than it did prior to the psychopharmacology era. The second is that the prevalence of bipolar illness has soared.

In a 2007 paper, Harvard psychiatrist Ross Baldessarini and colleagues laid out the worsening of bipolar outcomes in the modern era. Before lithium was introduced, there was "recovery to euthymia [no symptoms] and a favorable functional adaptation between episodes." Now, with antidepressants, mood stabilizers, and antipsychotics regularly prescribed to patients so diagnosed, there is "slow or incomplete recovery from acute episodes, continued risk of recurrences, and sustained morbidity over time." In the pre-pharmacotherapy era, 85 percent of bipolar patients would regain complete "premorbid" functioning and return to work. Now only a third achieved "full social and occupational functional recovery to their own premorbid levels."[34]

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[31] H. Pigott. "Efficacy and effectiveness of antidepressants." *Psychotherapy and Psychosomatics* 79 (2010):267-79.

[32] See Whitaker, ibid, 164-168.

[33] M. Posternak, "The naturalistic course of unipolar major depression in the absence of somatic therapy." *J Nerv and Ment Disease* 194 (2006): 324-9.

[34] N. Huxley. "Disability and its treatment in bipolar disorder patients." *Bipolar Disorders* 9 (2007): 183-96.

As for the jump in prevalence, prior to the pharmacotherapy era, manic-depressive illness was a rare disorder, affecting perhaps one in a thousand people.[35] In 2003, researchers reported that 6.4% of American adults suffer from bipolar symptoms, or one in every 16 people.[36]

This dramatic increase is due in part to an expansion of the criteria for making this diagnosis, starting with the criteria set forth in *DSM III*, and in part because antidepressants increase the risk that a person with unipolar depression will have a manic episode and convert to bipolar.[37] Stimulants prescribed for ADHD can also stir manic and psychotic episodes that lead to a bipolar diagnosis.

With depression now running a chronic course, and bipolar diagnoses soaring and outcomes worsening, disability due to mood disorders has notably increased in the United States during the past 40 years, and similar rises in disability have been reported in country after country that have adopted widespread use of antidepressants.[38] UK researchers have also found that the mortality gap for bipolar patients worsened after the atypicals were introduced.[39]

ADHD

Attention deficit disorder was born as a diagnosis in 1980, when *DSM III* was published. Subsequent iterations of the DSM then made it easier to diagnosis the condition. National surveys found that the percentage of American youth diagnosed with ADHD grew steadily during this period, topping out at 10% in 2011, with about two-thirds treated with stimulants.[40]

And once again, just as is the case with antipsychotics and antidepressants, this drug treatment worsens long-term outcomes. In the early 1990s, the NIMH mounted the Multisite Multimodal Treatment Study to assess the long-term impact of stimulant treatment, and at first there was good news.

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[35] D. Healy. "The latest mania: selling bipolar disorder." *PLOS Medicine* 3 (2006): e236.

[36] L. Judd. "The prevalence and disability of bipolar spectrum disorders in the US population. " *J Affective Disorders* 73 (2003): 133-46.

[37] A. Martin. "Age effects on antidepressant-induced manic conversion." *Arch of Pediatrics & Adolescent Medicine* 158 (2004): 773-80.

[38] See R. Whitaker, presentation to the UK Parliamentary Working Group in May 2016: "Causation, Not Just Correlation, Increased Disability in the Age of Prozac." Available at: *https://www.madinamerica.com/wp-content/uploads/2017/01/Causation-not-just-correlation-.pdf*

[39] J. Hayes. "Mortality gap for people with bipolar disorder and schizophrenia: UK-based cohort study 2000–2014." *Br J Psychiatry* 211 (2017): 175-81.

[40] Centers for Disease Control and Prevention. "Data and Statistics about ADHD." *https://www.cdc.gov/ncbddd/adhd/data.html*

At the end of 14 months, those prescribed stimulants by ADHD experts were doing slightly better than those treated with behavioral therapy. However, at the end of three years, "medication use was a significant marker not of beneficial outcome, but of deterioration," the MTA investigators reported. At the end of six years, medication use was "associated with worse hyperactivity-impulsivity and oppositional defiant symptoms," and with greater "overall functional impairment.[41] The medicated group were also shorter in height than the off-medication group.

Studies of youth diagnosed with ADHD in Australia and in Quebec have similarly found that stimulants are associated with worse long-term outcomes.[42,43]

A failed paradigm of care

The "revolution" that was launched in 1980 with the publication of *DSM III* has failed in all aspects: the biology of mental disorders remains unknown, DSM diagnoses are understood to lack validity, and drug treatments for psychotic disorders, mood disorders and ADHD have been found to worsen long-term outcomes, lowering recovery rates and impairing functional capabilities. The scientific literature tells of how psychiatry's disease model has proven to be a public health disaster, one that has caused extraordinary harm in developed countries around the world.

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[41] The MTA Cooperative Group. "A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactive disorder." *Arch Gen Psychiatry* 56 (1999): 1073-86. P. Jensen. "3-year follow-up of the NIMH MTA Study." *J American Academy of Child & Adolescent Psychiatry* 46 (2007): 989-1002. B. Molina. "Delinquent behavior and emerging substance abuse in the MTA at 36 months." *J American Academy of Child & Adolescent Psychiatry* 46 (2007): 1028-39. B. Molina. "MTA at 8 years." *J American Academy of Child & Adolescent Psychiatry* 48 (2009):484-500.

[42] Western Australian Department of Health. "Raine ADHD study: Long-term outcomes associated with stimulant medication in the treatment of ADHD children, 2009."

[43] J. Currie. "Do stimulant medications improve educational and behavioral outcomes for children with ADHD?" NBER working paper 19105, June 2013.

DECONSTRUCTING PSYCHIATRIC DIAGNOSIS

Sami Timimi

Let me explain why technically speaking there is no such thing as a psychiatric diagnosis. The creation of a mythology of mental illness that lacks scientific credibility has led to dominant beliefs and practices facilitating the rapid growth of psychiatric diagnoses and the tendency to deal with what is conceptualised as aberrant behaviour or emotions through technical – often pharmaceutical – interventions, a phenomenon I refer to as the 'McDonaldization' of mental health. I recommend that for progress in mental health theory, research, and practice, we must remove the concept of a 'psychiatric diagnosis.'

Despite over a century of research to establish possible causes using psychiatric diagnosis as the framing, the cupboard of positive findings remains astonishingly bare. There are no markers, no genes (apart from a significant portion of those with a learning disability), and no identifiable characteristic brain abnormalities. Studies looking at outcome from treatment with either pharmaceutical or psychotherapeutic models matched to diagnosis have not shown outcomes improving over time. What has increased instead are the numbers who get a psychiatric diagnosis, the amount of psychiatric medication prescribed, the numbers who become long-term patients, and the numbers who claim disability allowances for a psychiatric problem.

If the concepts we used in mental health practice had a scientific basis and/or were clinically meaningful, then we should be seeing something very different in both the science and the outcomes. Why do we have such an impasse? To start with, we literally don't know what we're talking about when we refer to mental disorders and illnesses.

What sort of 'thing' is a mental health problem?

What do people mean when they talk about mental disorder, mental health, or mental illness? What sort of 'thing' is a mental disorder? Where are its boundaries? When does an experience or behaviour become abnormal, disordered, or pathological and who decides based on what?

While the issue of where to place boundaries between the ordinary and the not ordinary is something medicine often grapples with, when it comes to

what we label as 'mental health' we have a whole new level of potential confusion, uncertainty, and meanings to get through before we can assert something to be out of the ordinary, abnormal, or disordered. In psychiatry, the entire phenomena, and not just the boundaries, require interpretation.

The territory for what we call psychiatric 'symptoms' (or psychopathology) of a mental disorder are experiences and behaviours that have meanings and that may be interpreted differently by different cultures, different times, and in different settings. This means that psychiatry is an area of practice where there is not only disagreements and debates about where the boundaries of a condition are, but we also have to take into account the significance and relevance of the diverse meanings that can be attached to these symptoms, for example that they are interpreted as symptoms in one interpretive framework but not in another.

Is that patient in front of me who reports intense sadness, difficulty getting to sleep, waking up before five am every morning, and experiencing a poor appetite, suffering from a 'depressive disorder' or experiencing understandable heartbreak and grief after the breakup of a long-term relationship a few months back? If you argue that both can be true, then culturally-speaking both depression and grief may be said about the patient as what they 'have.' One however cannot be a diagnosis (depression) as it explains nothing, it just describes some aspects of the patient's experiences, while the other (grief) could indeed be a 'diagnosis', as it suggests an explanation.

Even though grief in the above scenario is being used as an explanation, in truth I have no access to the patient's inner mental workings. None of us do. With grief, depression, or both, I still do not know what sort of a 'thing' I am dealing with. Is it a medical disease in her brain, is it the psychological process of grief, is it the loss of a social network that she had with that partner, is it her concern about how this is impacting her son, is it the fear of returning to work after a long absence, is it the impending change in her financial security, is it that she has come to suspect that she has 'depression' which is depressing her even further? Is it all of these things? In truth, I don't know anything definitive about what has caused her presentation; and likely neither does she. I can't escape my subjectivity; and I can't escape the patient's subjectivity, either. I can only guess at the 'diagnosis' (that is, the proximal explanation).

When it comes to our emotional experiences, we just have embodied experience. We then use words connected with cultural meaning-making systems to attach to that experience. The meaning scaffolding we then use can itself transform our experience of the experience. "*You are broken hearted*" creates a different scaffold to "*you are depressed*," or to "*you are*

surviving and recovering from a painful experience" or even to, "*I can see how your suffering has helped you see your life in a transformed way.*"

Labelling the experience as a diagnosis of 'clinical depression' thus creates a particular scaffold, rather than discovering any 'truth' about that experience. Our choice of scaffold has a potentially profound impact on how individuals then interpret their experiences, which in turn impacts on their subsequent feelings and behaviours.

There is no such thing as a psychiatric diagnosis

In medicine, diagnosis is the process of determining which disease or condition explains a person's symptoms and/or signs. ***Diagnosis is a system of classification based on cause.*** Making an accurate diagnosis is a technical skill that enables effective matching of treatment to address specific pathological processes. Pseudo-diagnoses, like for example 'Attention Deficit Hyperactivity Disorder' (ADHD) or 'Autistic Spectrum Disorder' (ASD), cannot explain behaviours or experiences, as there are only descriptions and not explanations.

Even using the word 'symptom' in relation to experiences and behaviours is problematic, as in medicine symptoms usually refer to patients' suffering/experience as a result of an underlying disease process and is therefore associated in our minds with a medical procedure leading to an explanation for the symptom.

We are meaning-seeking creatures and so have used classification systems extensively to classify all manner of things. A diagnostic classification is a classification by explanation, in other words by cause. That's why we say "*My doctor said that the cause of my chest pain was acid reflux, not a heart attack.*" This way of classifying works well when we can measure and empirically test, in a reliable way, bodily functioning. Diagnosis then provides a framework for research into treatments that address causes. Scientific methodology can be used and will lead to the development of a technical framework for classifying and treating conditions that affect the human body. In this medical universe, we generally know what 'thing' we are dealing with.

Take for example the fairly straightforward situation where there is minimal confusion about what sort of 'thing' we are dealing with. Somebody has an accident and experiences extreme pain and some swelling in their leg and they can't walk on it. At the hospital, an X-ray reveals there is a fracture in the tibia (shin bone). In this scenario, the medical model is working at its best. The fracture of the tibia is what is known as a 'natural kind,' so in terms of classification the diagnosis explains an abnormality in the person's physical body which can be empirically verified and measured.

As a natural kind that can be seen, it exists out there in the world beyond our subjective hypothesis. It is a verifiable fact of nature and we can develop knowledge bases about fractures of the tibia by comparing many people who have the same condition, trying out different treatment approaches and combinations, grading different types of severity, and looking at the various factors (in the fracture, the body of the person, the type of accident, and so on) that might affect responses to different treatments. Medicine is particularly good at these emergency scenarios where there is an identified abnormality and where the treatment period is relatively short.

Not all presentations to doctors follow this easy-to-understand idea of what sort of thing we are dealing with. Let's take diabetes as an example. The connection between symptoms and the underlying cause may not be as immediately apparent. A diagnosis of diabetes refers to an abnormally high level of sugar in the blood and this can be measured (for example through a test of blood sugar levels after a period of fasting). Type II diabetes could present just as a susceptibility to infections, or generalised tiredness and so could go unnoticed for months or even years. Nonetheless, there is a physical parameter that can be measured and there is a physiological process present in the physical body and that exists in the world external to the doctor who carries out the diagnosis and is verifiable with independent data (blood sugar levels).

So, in this example, whilst the connections between symptoms and disease are not as clear, may involve other factors than just the sugar metabolism, and may be missed in the early stages or by a poorly trained doctor, the diagnosis again is explanatory. It is pointing to an abnormality that can cause symptoms in the patient and will cause more if not treated. But there are many disagreements in diabetes diagnosis and treatments; for example, when to consider the blood sugar has crossed a threshold justifying a diagnosis, whether to just use dietary approaches and for how long, when to use medication, how to deal with complications, the psychological impact of having a chronic disease, the social dimension of long-term care, and so on. But still, we know what sort of 'thing' diabetes is.

Now we start to get into medical conditions which can have recognisable symptoms and sometimes physical signs and some objective tests, but in which there are mysteries as to the initial cause or explanation. Many types of headaches, such as migraines, are good examples of this category. Diagnoses such as migraine are mainly based on a description of symptoms. We are now moving toward a descriptive rather than explanatory system. However, given that there are characteristic physical symptoms (such as, in migraine, that you may get blurring of vision, pain behind the eyes on one side of the face, etc.), it is likely that there is physical pathology. The

presentation tends to be characteristic and there are physical symptoms, and so it is reasonable to assume that it involves physiological processes. So, we kind of know what sort of a 'thing' migraine is, though we are now getting into a rather fuzzier territory.

Can you sense how we are slipping away from explanation toward description and what problems this will cause?

With pain and with the nervous system involved, psychological aspects are becoming more prominent. But the idea of diagnosis still stands, even if it's to conclude that while the migraine is a diagnosis (in that it explains the physical symptoms), it can be brought on or sometimes even mimicked by psychological factors. But psychiatric diagnoses do not do even this much.

Consider the following example. If we were to ask the question "*What is ADHD?*" it's not possible to answer that question by reference to a particular known pathological abnormality, as none have been found. Therefore, there are no medical tests for ADHD, nor are there recognisable physical phenomena (symptoms). Instead, to answer the question we will have to provide a description, and a highly socialized one at that, such as "*ADHD is the presence of 'abnormal' levels of poor concentration, hyperactivity and impulsivity*".

Contrast this with asking the question, "*What is diabetes?*" If a doctor were to answer this question in the same manner by just describing symptoms, such as needing to urinate excessively, thirst, and fatigue, he or she could be in deep trouble as a medical practitioner, as there are plenty of other conditions that may initially present with these symptoms; and diabetes itself may not present with these symptoms in a recognisable way.

In order to adequately and accurately answer the question, "*What is diabetes?*" you would have to refer to its pathology involving abnormalities of sugar metabolism, as in, "*Diabetes is a disease that occurs when blood glucose (sugar) is too high.*" In most of the rest of medicine, a diagnosis explains and has some causal connection with the patient's experiences and/or symptoms. Diagnosis thus sits in a 'technical' explanatory classification framework.

The problem of using a classification like 'ADHD' to explain an experience (i.e., as a diagnosis) can be illustrated by asking another set of questions. If a doctor were asked by someone why his or her child is hyperactive and the doctor answered that this is *because* they have ADHD, then a legitimate follow-up question to ask is, "*How do you know that this hyperactivity is caused by ADHD?*" The only answer the doctor can then give to that question is that "*I know this is ADHD because they are hyperactive.*"

In other words, if we try to use a classification that can only describe in order to explain, we end up with what philosophically is known as a 'tautology.' A tautology is a circular thinking trap. A description cannot explain itself. Using ADHD to explain hyperactivity is like saying the pain in my head is caused by a headache or my cough is caused by a 'coughing disorder'. In psychiatry, what we are calling diagnosis will only describe but is unable to explain and therefore it isn't a diagnosis.

If the rest of medicine were practiced like psychiatry, then when you go to your General Practitioner (GP – this is the UK title for a primary care doctor) because you have a recurrent cough, the GP wouldn't examine you at all; he or she would just ask you questions about your cough and relevant history and perhaps get you to fill in a questionnaire. He or she would then pronounce that you have a 'Recurrent Cough Disorder – RCD' and give you a steroid inhaler to take once a day. The inhaler has non-specific effects and will open the airways, so at least in the short-term there would be some improvement in symptoms for many patients with a cough.

However, if you had a chest infection, your condition would likely ultimately get worse. Furthermore, long-term steroids can have all sorts of unpleasant and dangerous side effects if taken in sufficient quantities. Thus, this sort of negligent 'treatment' will have every chance of making things worse, perhaps even fatally worse, in the longer term, without ever understanding the potential causes of that cough.

But you wouldn't really expect your doctor to behave like that. At the very least, you would expect him or her to listen to your chest with a stethoscope, to seek out physical signs, and perhaps arrange further tests (like a chest X-ray) if he or she remained uncertain as to the cause of the cough. In the rest of medicine, diagnosis really matters. It will guide the doctor towards a treatment that addresses the initial cause of the cough.

The failure of decades of basic scientific research to reveal any specific biological or psychological marker that identifies a psychiatric diagnosis is well recognised. Unlike the rest of medicine, which has developed diagnostic systems that build on a causal and physiological framework, psychiatric diagnostic manuals have failed to connect diagnostic categories with any causes or physical markers. Thus, there are no physical tests referred to in any mental health diagnostic manual that can be used to help establish a real diagnosis.

Despite the belief that psychiatric disorders have a significant genetic loading, molecular genetic research is failing to uncover any specific genetic profile for any psychiatric disorder. Possible genetic abnormalities appear to account for an insignificant percentage of possible associated