

Brain Research on Mental Disorders: A Criticism

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Abstract

Objective: To expose the problems and inherent limitations of neuroscience-based brain research on mental disorders. **Method:** Discussion of the theory underlying brain research on mental disorders, followed by a systematic evaluation of typical studies. **Results:** The fundamental problem is that brain researchers fail to differentiate between biological mental disorders in which brain processes cause the disorder (notably schizophrenia, bipolar disorder, and melancholic depression) and learned mental disorders in which brain processes mediate but do not cause the disorder (which is the case with reactive depression, reactive anxiety, OCD, and PTSD). Researchers have been unsuccessful in identifying mechanisms in the brain that cause biological mental disorders, and will never be able to locate the innumerable specific neural connections that mediate learned mental disorders. Moreover, the author's review of typical studies in this field shows that they have serious problems with theory, measurement, and data analysis, and that their findings cannot be trusted. **Conclusions:** Neuroscience-based brain research on mental disorders, unlike other neurological research, has been an expensive failure and it is not worth continuing.

Keywords

Neuroscience-Based Brain Research, Biological Mental Disorders, Learned Mental Disorders, Research Problems

1. Introduction

The prevalence of serious mental disorders has shown no decline over the years despite the emergence of new drugs and new forms of psychotherapy [1] [2] [3]. Accordingly, the hope is that neuroscience research on brain components and

their functions, which has been generally successful in diagnosing and treating neurological disorders, will provide a breakthrough for mental disorders. But will it be a breakthrough? What has been the value of neuroscience-based brain research on mental disorders so far?

In this article, the present author reviews typical studies in this field, exposing the problems that make them untrustworthy and of no value in detecting and treating mental disorders. The article begins in section 2 with theory, looking at the two possible roles for brain processes in mental disorders, namely a causal role in the emergence of biological mental disorders and a mediating role in the acquisition of learned mental disorders. Section 3 evaluates a selection of typical neuroscience studies that cover both disorder types. Section 4 summarizes the problems. The last part, section 5, draws some general conclusions.

2. Theoretical Roles of Brain Processes in Mental Disorders

The theorized roles of brain processes differ critically depending on whether we are dealing with biological, internally caused mental disorders, or learned, externally caused mental disorders. In biological mental disorders, brain processes play a direct causal role (see **Figure 1**, panel A). They do this either electrically through impaired nerve circuitry in the brain, chemically through over-production or under-production of gene messenger proteins, or physically by damage or disease [4]. Biologically caused mental disorders include schizophrenia, bipolar disorder in the severe form known as bipolar I, and “endogenous” or what is more plainly called melancholic depression. There is always severe dysfunction with biological mental disorders. The dysfunction with schizophrenia is often chronic and worsening, whereas with bipolar disorder and melancholic depression, the dysfunction occurs during unpredictable sudden-onset and sudden-recovery episodes and can be largely absent for long periods between episodes.

Learned mental disorders, on the other hand, are different in that they do not originate from a biological cause but rather are involuntarily learned in response to life events (**Figure 1**, panel B). The main examples are “exogenous” or reactive depression [3] [5] and all anxiety disorders [3] [6] including various forms of situationally conditioned reactive anxiety as well as fear-induced obsessive-compulsive disorder and shock-induced post-traumatic stress disorder. Readers may note that I have omitted an important class of learned mental disorders—eating disorders. This is because eating disorders are almost always the result of externally caused reactive depression or reactive anxiety, and what must be treated are the various causes of *these* disorders rather than the eating behavior itself. Brain processes play a very different role in learned, externally caused mental disorders. They do not directly cause the disorder. Instead, these brain processes operate, or what in psychology is called mediate, between the external stimulus cause and the observed symptom response. As shown in panel B, the primary mediating processes are innate and learned stimulus-response, S-R, connections located in the brain cell synapses where the stimulus sensation

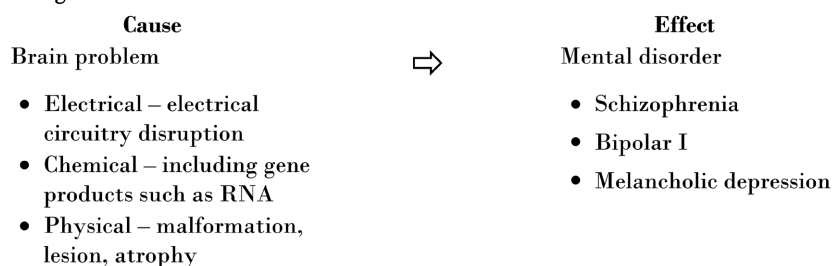
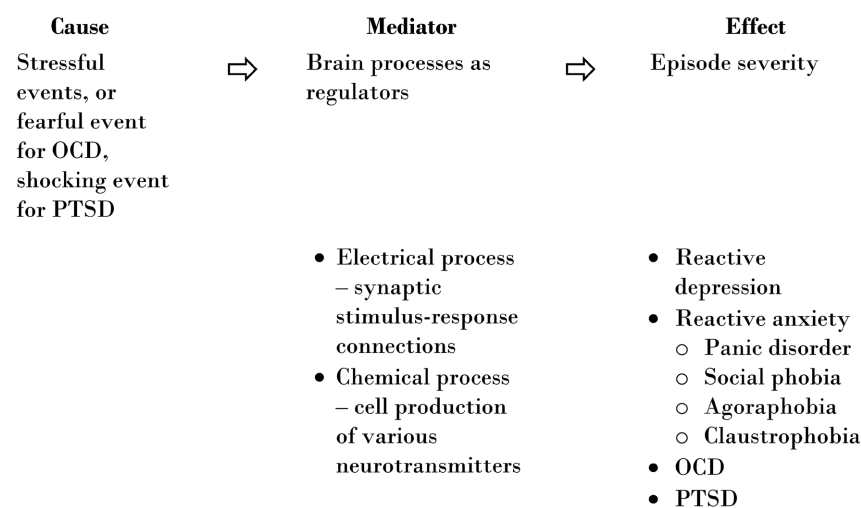
A. Biological Mental Disorders**B. Learned Mental Disorders**

Figure 1. Hypothesized role of brain processes in biological vs. learned mental disorders.

producing neuron meets the learned response-carrying neuron (for more detail on the actual learning processes, see [3]). Impairment of the brain cell itself can also play a role by reducing chemical neurotransmitter production.

It should be noted that the DSM-5 and the ICD-11, the two most influential mental disorder diagnostic systems worldwide, make no attempt to identify the causes of mental disorders. This is a major failing of both diagnostic systems and it places serious limitations on our understanding of mental disorders and of effective treatment.

3. Evaluation of Typical Studies

Research evaluators and, equally importantly, general readers of research need to understand that one major uncorrectable flaw in the study—a so-called fatal flaw—means that the whole study is compromised. As a sometime editor and long-time research reviewer, I have devised a hierarchical evaluation procedure in which I look for 1) the theoretical argument made to justify doing the study in the first place, and if that is adequate I then look at 2) the validity of the measures of the dependent variable and 3) of the independent variable or variables, and if the validity is acceptable, I then check 4) the appropriateness of the data analysis and its interpretation, and if that is sensible, I look finally at 5) what the authors say are the study's implications for practice. I do confess that as a mea-

surement expert, I have found it most efficient to look firstly at the measures, where very often I find that the researchers are not measuring what they claim to be measuring or are measuring it so badly that the findings are not to be trusted.

In the present article, I have adapted this step-by-step procedure for evaluating brain research on mental disorders (see **Figure 2**). Note that only two of the steps, the first on theory and the last on practical implications, are potentially correctable because if you are a reviewer, you can ask the author or authors to supply the missing information. If, however, the problem is with one or more of the middle steps—those concerning the measures and the data—then the paper should be rejected outright or, if published, you should ignore it. This is the case with the four studies reviewed next.

3.1. Brain Research and Biological Mental Disorders

I am going to review two studies on biologically caused mental disorders that

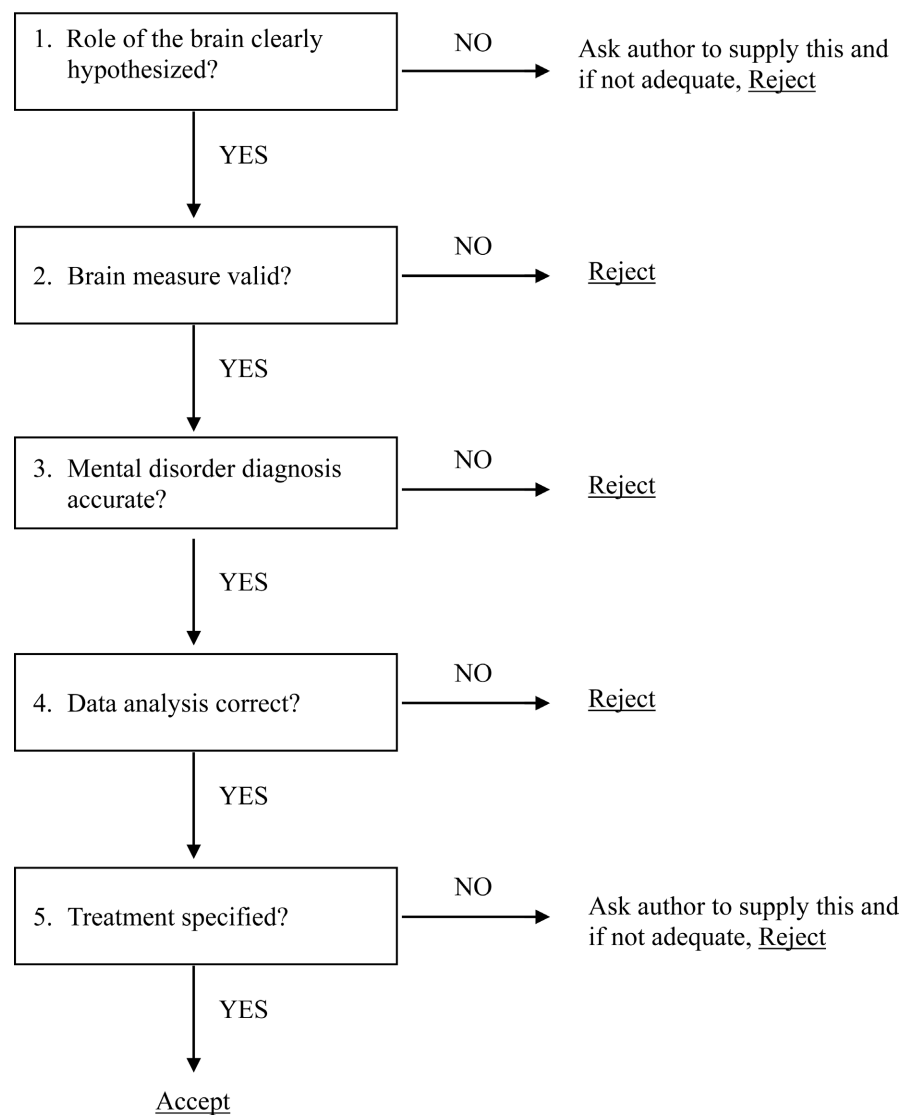


Figure 2. Hierarchical evaluation procedure for brain research on mental disorders.

illustrate two different methods of neuroscience-based brain research, namely, MRI brain scanning, and post-mortem brain tissue analysis.

Brain Scanning and Schizophrenia

Kochunov and his team of 22 co-authors [7] in a study published in the prestigious, broad-readership journal *JAMA Psychiatry*, set out to test the hypothesis that “altered connectivity” in the white matter of the brain in schizophrenics would reduce their working memory capacity via reduced information-processing speed.

1) *Theory problems.* Kochunov *et al.*'s theory is a problem firstly because of one of the many disagreements in brain biology. According to the definitive psychiatry text by Sadock and Sadock [4] it is deficient *grey* matter, the brownish-grey nerve cells and their dendrites, rather than white matter, which is the whitish-tissue nerve fibers and their myelin covering, that is thought to limit working memory. Secondly, Kochunov *et al.*'s theory is a problem because the implied main hypothesis is surely that deficiencies in the structure of the white brain matter would be present in the brains of schizophrenics and absent in the brains of healthy controls. As it happened, Kochunov *et al.* found no difference in white matter structure between schizophrenics and healthy controls—a null finding that was hidden away in the online appendix in Table e2.

Kochunov *et al.*'s finding of no difference in white matter structure between schizophrenics and controls, of course, means that their hypothesis of white matter damage in schizophrenics causing reduced working memory should have been abandoned.

2) *Brain measure not valid.* Kochunov *et al.* used MRI scanning to measure something called “fractional anisotropy,” and I had to go to a dictionary [8] to see what this term meant. The first word, I suspect, should be “fractionated,” which means broken or separated, rather than “fractional” which can mean separated but in its much more usual sense means very small or insignificant. And the second word, “anisotropy,” according to the dictionary, means dissimilar (aniso) turning (tropy), which I suggest is not an adequate description of structural deficiency. As I will note again later, specialized terminology does not help outside readers to understand neuroscience research.

There is also a question regarding the sample of schizophrenia outpatients on which the MRI measures were taken. Schizophrenia sufferers, one would suspect, are unlikely to be able to endure the confinement and loud banging and clanging that goes on when you are placed in an MRI machine [9]. Also, an unknown number of patients were admitted to the study with a diagnosis of schizoaffective disorder rather than schizophrenia, as discussed next.

3) *Mental disorder diagnosis inaccurate.* Kochunov *et al.*'s study was on patients DSM-IV diagnosed as having schizophrenia *or* schizoaffective disorder. But according to the DSM, schizoaffective disorder is classified as a mood disorder, not a schizophrenia disorder. In schizophrenia, or “nonaffective psychosis,” the delusions and hallucinations occur independently of the patient's mood state, whereas in schizoaffective disorder the delusions, and less often hallucina-

tions, occur only during a depressive or manic episode. The brain-based causes of the two disorders are therefore likely to be different and the researchers should have excluded the schizoaffective disorder patients or at least analyzed them separately.

4) *Data analysis and interpretation incorrect.* The data analysis in Kochunov *et al.*'s study appeared to be correct technically, but the main finding on brain matter differences was null, and therefore their working memory hypothesis could not be tested.

5) *Treatment implications not clear.* The only schizophrenia treatment implication offered by Kochunov *et al.* was a vague reference to white matter-directed pharmacological interventions ([7], p. 965). It is hard to see how Kochunov *et al.* could recommend any treatment given their completely null findings.

Brain Tissue Analysis and Schizophrenia, Bipolar Disorder, and Major Depression

The most ambitious brain research study that I came across is the study by Australian neuroscience researcher Natalie Matosin, lead author on a study with 35 other international researchers [10] who submitted post-mortem brain tissue data from six different laboratories across the world. She and her colleagues analyzed the brain tissue to try to find a particular brain chemical difference that might be the cause, quite remarkably, of three mental disorders—schizophrenia, bipolar disorder, and major depressive disorder. This study was published in one of the leading neuroscience journals, *Acta Neuropathologica*, a journal to which I wrote a rejoinder to Matosin *et al.*'s study that was not unexpectedly rejected since I was an outsider questioning the editor's acceptance decision. I now take this opportunity to summarize the problems that I raised.

1) *Theory problems.* Matosin *et al.* set out to demonstrate that excess gene expression of FKBP5/FKBP51 mRNA, which are so-called messenger proteins created by one's genetic DNA, is the cause not only of major depression but also of bipolar disorder and schizophrenia—but only if the now-deceased patient had experienced *early life stress*. This hypothesis is dubious because all three disorders are known to emerge regardless of early stress [4] and while it is true that stressful events in adolescence or adulthood can precipitate *episodes* of these disorders, stress is not the cause of the disorder in the first place [11]. In any case, Matosin *et al.* did not have a record of whether the deceased person had been exposed to early life stress, so right from the start they were unable to test their theory. Nevertheless, let us consider what else they did wrongly.

2) *Brain measure not valid.* Matosin *et al.* analyzed brain tissue taken from deceased persons, using tissue samples obtained after getting permission from the deceased's family or guardian. This is probably a biased sample in that permission is rare and seems more likely to be given if the person died of a non-apparent or medically ambiguous cause [12]. Further complicating measurement of brain tissue is that it is the most complex type of tissue in the human body. It is ultra-soft and becomes deformed even under its own weight,

with the degree of deformation affected by storage temperature and thawing temperature [13]. Furthermore, the post-mortem interval, which is the length of time between death and the brain tissue analysis, greatly affects the amount and quality of gene material expressed [14].

Matosin *et al.*'s hypothesis was that the three mental disorders would be detectable as *excess* mRNA protein in the brain (again, only if the person had experienced early life stress). The problem here is that the researchers had no way of telling whether the protein amounts that they measured were excessive. All the researchers could do was compare the average level of protein expressed in each of the three mental disorder groups compared with the average level expressed in the control group. Not only is this group comparison very imprecise as shown by the wide scatter of protein expression levels in all four groups, but the average levels did not differ—see Figure 1 on p. 446 for FKBP5 protein and Figure 4 on p. 452 for FKBP51 protein. Thus, even though Matosin *et al.* failed to test their hypothesis properly, their published data make it most unlikely that it would have been supported.

3) *Mental disorder diagnosis inaccurate.* The mental disorder diagnoses of the deceased persons in Matosin *et al.*'s study were not obtained directly from a psychiatrist. Instead, the diagnoses were made indirectly by interviewing someone, most likely a family member or friend, who had known the deceased person well (online supplement, p. 5). Whereas the symptoms of schizophrenia are reasonably evident to close family and friends, those of melancholic depression and bipolar disorder, with the distinction between the two disorders often requiring multiple assessments and up to 10 years to decide [5], are not. The diagnoses in Matosin *et al.*'s study therefore cannot be assumed to be accurate.

4) *Data analysis incorrect.* Matosin *et al.* relied on conventional null hypothesis significance testing, NHST, in their analysis but their hypothesis was not null but directional, namely that the mean, or rather mean excess, mRNA expression levels would be *higher* in the three mental disorder groups than in the control group. However, according to Figures 1 and 4, the disorder groups' means were often below or basically equal to the control group mean, thus rejecting the directional hypothesis. Even if Matosin *et al.* had used the now preferable method of reporting means together with their confidence intervals (e.g., [15]), the same conclusion would follow because the directional hypothesis is rejected by the overlapping interquartile box-plot data in the figures.

5) *Treatment implications not clear.* The Abstract for Matosin *et al.*'s article (p. 439) began with the rationale that we need “novel targets for treatment” and concluded with the statement that the FKBP5 type of gene has been proven in this study to be one such target. However, senior author Elizabeth Binder's conflict-of-interest disclosure at the end of the article (p. 456) says that the FKBP5 is an antidepressant target, so it is apparent that this would not be helpful for developing the antipsychotic type of drugs needed to control schizophrenia and bipolar disorder. Also, as I noted above, the mRNA protein-release levels dif-

ferred far too much across individuals to make the targeting of the FKBP5 or FKBP51 feasible.

3.2. Brain Research and Learned Mental Disorders

With learned mental disorders, it is not so much the genes or neurons themselves that are of interest but rather the almost innumerable synaptic stimulus-response connections that can become established between them [4]. They include *innate* or unconditioned S-R connections involved in inborn phobias, inborn aversive reactions to pain, and natural approach reactions to sugar in foods and beverages. They also include the almost innumerable *learned* or conditioned S-R connections: the persistent ruminative thoughts in reactive (as well as melancholic) depression and the hard-to-stop feelings of fear or dread in anxiety disorders; the compulsive handwashing and other operantly reinforced repetitive behaviors in OCD; and the Pavlovian classically conditioned visual and auditory memories triggered in attacks of PTSD.

The almost infinite number of these learned connections essentially puts paid to the idea that neuroscientists can locate specific ones in the brain, let alone do anything to modify them. Accordingly, brain research investigations of the learned mental disorders have turned out to be rather tangential, as exemplified by the two neuroscience studies reviewed next. They deal with two of the most difficult-to-treat learned mental disorders, PTSD and cannabis addiction, and they illustrate two other methods of neuroscience research, so-called functional magnetic resonance imaging, and gene association searching.

Functional MRI and PTSD

Neuroscience researchers Perl *et al.* [16] conducted a thoughtfully designed experiment to test whether memories of the trauma-inducing event could be differentiated from memories of sad but non-traumatic events, and from memories of calming, positive events. To do this, they used a version of MRI called *functional* MRI (fMRI) that measures brain-regional blood flow in response to external stimuli, with blood flow assumed to correspond with brain cell activation in that region [9]. Perl *et al.*'s study, conducted with seven other researchers listed as co-authors, was published in one of the leading neuroscience journals, *Nature Neuroscience*.

1) *Theory reasonable.* Perl *et al.* devised what at first looked like a clever test of their memory difference hypothesis. They simulated the stimulus cause of the trauma by first asking PTSD-diagnosed patients to elaborate on what they recalled as their main traumatic experience, which may have been an event that occurred in combat, a fire or accident or some other disaster, or a sexual assault or incident of family violence. They then asked them to recall and elaborate on a sad but non-traumatic event, such as the death of a friend or relative, a family dispute, a miscarriage, or a job loss. And lastly, they asked them to recall and elaborate on a positive but calm event, such as going for a walk, playing golf, or meeting a friend for coffee. These elaborations were recorded and then rewritten

by the researchers as short audio scripts which were played back to the patient while he or she was undergoing the fMRI brain scan. My argument is not with the theory but with the way Perl *et al.* tested it.

2) *Brain measure not valid.* The fMRI brain measures were rendered invalid by the stimuli Perl *et al.* used to elicit them. This is because Perl *et al.* performed a computer-aided “semantic similarity” content analysis of the patients’ elaborations of the experiences as a way of post-classifying the stimulus event as either traumatic, sad, or calming. This was unnecessary and could only confuse matters given that the distinctions were already built in by the three separate event descriptions asked for beforehand. Further reducing the validity of the stimuli was Perl *et al.*’s use of scripts written and read by a researcher rather than using the patient’s own voice and words to recreate the event.

There are also problems with fMRI brain measurement. One problem is that it is rarely tolerable for mental patients. The 28 participants in Perl *et al.*’s study were PTSD outpatients and it is doubtful that traumatized individuals, particularly anyone traumatized in combat, would be comfortable in the noisy and claustrophobic MRI chamber. A second problem was that fMRI’s continuous brain signals are liable to be distorted by involuntary in-scanner head movements [17], resulting in brain readings that are very unstable [18]. This was evident in Perl *et al.*’s study by the very different fMRI readings across individuals, even when they were reacting to what was supposed to be the same type of memory.

3) *Mental disorder diagnosis not accurate.* Perl *et al.* relied on the Clinician-Administered PTSD Scale for the DSM-5, the CAPS-5, to make the post-traumatic stress disorder diagnosis [19]. However, the CAPS-5 is a hopelessly inaccurate measure (see [20]). The 20 items in the CAPS-5 scale include only three items that correspond to the required symptoms of PTSD—namely, involuntary recurrence of memories, dreams, or flashbacks of the traumatic event—and only *one* of these symptoms, at a level of 3 or 4 on the 0-4 answer scale of clinician-related severity, is required for a positive diagnosis. The remaining 17 items in the CAPS-5 address other symptoms that could be side effects of the required symptoms or the results of other disorders such as depression. Most critically from a research standpoint, it is possible to obtain a high total score on the CAPS-5 measure without having *any* of the necessary symptoms of PTSD.

4) *Data analysis incorrect.* As it happened, Perl *et al.* did not find any support for their main hypothesis that the brain readings would differ between PTSD memories and merely sad memories (see the vertical axis of their [Figure 3e](#) on p. 2230, where for some reason they did not include the calm memory results). The data, nevertheless, are meaningless because, as I have demonstrated above, the experimental stimuli, the fMRI brain measure, and the PTSD measure were not valid.

5) *Treatment implications not clear.* There are no treatment implications that

follow from Perl *et al.*'s study. Even if they *had* been able to differentiate PTSD memories from sad memories, clinicians would be concerned with treating only the PTSD memories and would not even ask about the other.

Gene Searching and Cannabis Addiction

The brain research method I will turn to now is a method known as a genome-wide association study, or GWAS, which is essentially a blood sample search for gene malformations that are present in the brain of individuals known to have the disorder, and absent in the brain of individuals who do not [21]. The scope of the search needed is massive because to date there are approximately 22,000 known genes in the human brain, with the likelihood of many more being discovered. A further complication is Segal *et al.*'s finding [22] that, for mental disorders, the regional areas in the brain where affected genes seem to be located are extremely varied, even among individuals with purportedly the same diagnosis.

The mental disorder studied in the GWAS study that I am going to review is cannabis addiction, which has become a growing problem with the relaxation of laws in most western countries governing cannabis use. Adding to the problem is that the percentage of THC, the main psychedelic chemical in cannabis, has increased over the years from about 2% - 4% in the hippie and Woodstock era of the 1960s and 1970s to around 15% - 24% today [23], and it is also found, albeit at a lower concentration, in so-called medicinal cannabis. The study is by Pasman *et al.* [24], written with more than 50 co-authors and published in a leading neuroscience journal, *Nature Genetics*.

1) *Theory inadequate.* Pasman *et al.* set out to find so-called SNPs, single nucleotide polymorphisms, which as far as I can tell are alternative forms of the same gene, that they expected to differ in cannabis users but not in non-users. Because these differences are apparent at birth, Pasman *et al.* seem to have implied that they are a *cause* of cannabis addiction. This is doubtful, from what I have read. Cannabis addiction is a learned, not an inherited disorder, and a more plausible theory of addiction to cannabis is that, with regular intake, the dopamine receptor gene D2 becomes insensitive or damaged such that a higher dose or more frequent administration of the drug is needed [4]. Whether or not the cannabis user becomes addicted, however, seems to depend more on how the user responds to dopamine depletion between doses, and how far the user will go to quell the withdrawal symptoms. If the compulsion to re-dose leads to compulsive seeking and use of the drug even in the face of serious health and social consequences, then the person is considered to be addicted [25]. Survey figures from 20 years ago [4] suggest that about one in 10 regular cannabis users were becoming addicted but this could possibly be as high as one in five with the higher-strength cannabis prevalent now.

2) *Brain measure not valid.* Pasman *et al.* measured differences in gene formation found in blood samples. Harvard Medical School cannabis expert Bertha Madras, however, has found that brain levels of cannabis are two to three times

higher than blood levels of the drug, and that cannabis persists in the brain, in the fatty myelin white matter that insulates the nerve fibers, long after the blood level has gone down [26]. This persistence of the cannabis in the brain is unlike the relatively fast dissipation of drugs such as alcohol, she notes, and is what can cause long-term cognitive functioning damage and possible schizophrenia even after usage has stopped.

3) *Cannabis addiction diagnostic accuracy.* Pasman *et al.*'s dependent variable was entirely unsatisfactory. They studied what they called "lifetime cannabis usage" which, to the casual reader, would mean regular cannabis usage over one's lifetime. But Pasman *et al.* allowed it to mean *any* usage of cannabis *ever*, including merely trying cannabis out of curiosity, which almost half of Americans over the age of 12 say they have done [4]. Pasman *et al.* used far too loose a definition of cannabis use, seriously reducing the value of their study.

4) *Data analysis incorrect.* As is typical of GWAS studies, Pasman *et al.* analyzed only the correlations between differences in gene formation and cannabis use, and the gigantic sample size means that even a very small correlation is likely to be statistically significant.

5) *Treatment implications.* Anything short of a perfect relationship between gene formations and a given mental disorder means that any attempt at treatment would be far too risky.

4. Summary of the Problems with Brain Research on Mental Disorders

The problems—fatal flaws—in these neuroscience-based brain studies are summarized in **Table 1**. None of the studies can survive a proper evaluative review. Most of them fall at the first hurdle by not explaining adequately the theory underlying the research, failing especially to explain whether the genes play a causal role, as in biological mental disorders, or whether they play a mediating role by regulating the internal response to the external stimulus, as in learned mental disorders. It may be argued that brain research studies of mental disorders could still be valuable if they were to be regarded, as in other fields of science, as "blue sky" research devoted only to finding relationships for others to explain. However, brain research studies are of no use if they have any of the middle three problems—invalid brain measures, inaccurate mental disorder diagnoses, or incorrect or misinterpreted data. Any one of these problems, like a broken link in a chain, negates the whole study. Also, all brain research studies fall at the last hurdle in failing to specify mental disorder treatments. The paradox here is that treatment would be prevented by the fact that there is far too much individual variability around the average results to allow for precise targeting, and to treat individuals without certainty that they have the disorder would be out of the question.

Whereas there may be some studies somewhere that escape the above criticisms, I could not find them, and their existence would not change the fact that most if not all the published brain research on mental disorders is not to be trusted.

Table 1. Summary of brain research study evaluations.

Study	Theory clear	Brain measure valid	Mental disorder diagnosis accurate	Data analysis and interpretation correct	Treatment implications clear
Biological Mental Disorders					
Kuchanov <i>et al.</i> (2017): MRI of gene structure—Schizophrenia	NO	NO	NO	NO	NO
Matosin <i>et al.</i> (2023): Brain-tissue analysis of gene mRNA expression—Major Depressive Disorder, Bipolar Disorder, Schizophrenia	NO	NO	NO	NO	NO
Learned Mental Disorders					
Perl <i>et al.</i> (2018): fMRI of neural patterns—PTSD	YES ^a	NO	NO	NO	NO
Pasman <i>et al.</i> (2018): Gene search from blood sample—Cannabis addiction	NO	NO	NO	NO	NO

^aPerl *et al.*'s theory was correct for what they studied, namely PTSD symptom types. However, their measures were not valid.

5. General Conclusions about Brain Research on Mental Disorders

Neuroscience-based brain research on mental disorders is riddled with problems, yet it is allowed to continue unabated. Neuroscience researchers have been able to largely hide these problems by filling their articles with obscure technical jargon that probably is not even equally understood by their peers, let alone being decipherable by the general reader or by mental health professionals. They preserve the mystique by including impressive-looking color illustrations that do nothing to make the findings clearer and, if anything, serve only to remind us of how little we know about the human brain.

Another undesirable outcome has been the intense competition for neuroscience research funding in the form of government research grants. This competition, of course, puts great pressure on neuroscience researchers to be selective in data analysis to produce statistically significant results. Continued funding, in turn, depends on getting papers published, the chances of which have increased dramatically with the proliferation of journals and the willingness of editors to accept articles with an implausibly large number of co-authors and badly flawed methodology.

I began this paper by pointing out that the prevalence of serious mental disorders has shown no change for years, that mental health treatments have not improved either, and that this is why there has been so much hope placed in the neuroscience-based approach. However, if neuroscientists were confident about curing mental disorders, especially biologically caused mental disorders, they would surely be recommending the same brain-invasive treatments used for

neurological disorders, such as gene therapy surgery to replace or supplement affected gene cells with genetically corrected cells. But to my knowledge no neuroscientists working on mental disorders have gone this far, proposing instead only drug treatment that at best can have only a temporary effect. Drug treatment, though, is a problem because of the repeated finding of a very strong placebo effect that is causing pharmaceutical companies to basically give up on investing in the development of new psychiatric drugs [2] [27] [28]. The placebo effect is also evident in psychotherapy, the main hope for curing learned mental disorders. This is seen in the well-established finding that the main determinant of psychotherapy effectiveness is placebo-like trust in the psychotherapist, regardless of the type of psychotherapy that the therapist employs [3] [29]. Only sophisticatedly designed and costly behavior therapy [3], practiced in a small minority of cases, avoids the placebo effect.

All this suggests that the outlook for the treatment of mental disorders is grim and I see no hope of brain research making the outlook any better. We should stop funding it now.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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