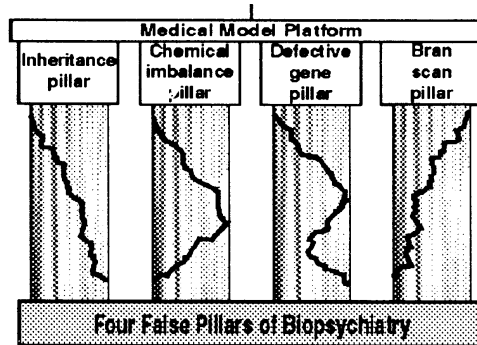


The Four False Pillars of Biopsychiatry

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In order for individuals to fully affirm a *non*-biological model of mental illness, it will be necessary to have a solid understanding of the main fallacies behind the biological model. Even though biopsychiatric researchers are willing to admit that, to this date, no pathophysiological or cause-and-effect evidence exists, they still consistently claim that they are getting closer and that they are making great strides with the new technology now available to them.

For example, a major textbook on psychiatry, concerning possible biological evidence for schizophrenia, states,

*The past five decades have borne witness to an impressive period of discovery in the neurobiological basis of schizophrenia. Modern psychiatric research has produced an abundance of evidence supporting the notion that schizophrenia is a disorder primarily related to brain dysfunction.*¹

Yet, hidden within this quote is the admission that, as yet, nothing conclusive has been found. In fact, to better affirm that absolutely no pathophysiological evidence has been found, refer to the National Institute of Mental Health's website on schizophrenia.² Since they fund much of the research on mental illness, this website will give an instant update if any true physiological conditions have been found. [I will quote directly from this website at the end of this paper.]

In reference to the quote from the textbook, the author used the term "supporting the notion." The main question then becomes: If I and so many others in my field claim that no pathophysiological evidence exists, how did this *notion* or possible myth come about?

In order to answer this question in a brief manner, as well as present the material in an easy-to-digest form, I will need to condense the research considerably. To do so I will address the main biological

evidence that is most often presented to the public as well as found in the major textbooks on psychopathology. Thus, after reading this material, a student or a professional should be able to go to his or her textbook and identify the major problems with the biological model.

A note before we start. Even though I have attempted to present the research in as easy-to-understand a manner as possible, such research may be new to many readers. Because of the importance of this research in terms of making critical and lifelong decisions for those suffering emotionally, it is well worth the extra time and effort to properly understand this material.

The Basic Fallacy

If there is supposedly an “abundance of evidence” to support a biological model—although I and many other professions still do not believe that such evidence will ever produce any cause-and-effect results—where then is the basic problem, and how does such a difference of opinion exist? Or, if I and others are right, how can so many highly intelligent, well-meaning, and educated researchers be wrong? In fact, *how can the vast majority of professionals be wrong?*

The problem is not so much in the research methodology, but in the basic assumption that precedes the research. When a person examines the research data, the data can often point towards either a biological or environmental conclusion. Thus the conclusions drawn from the research data are not based primarily on the data, but on the *personal assumptions* of the researchers. Figure 1 diagrams this point for further clarity.

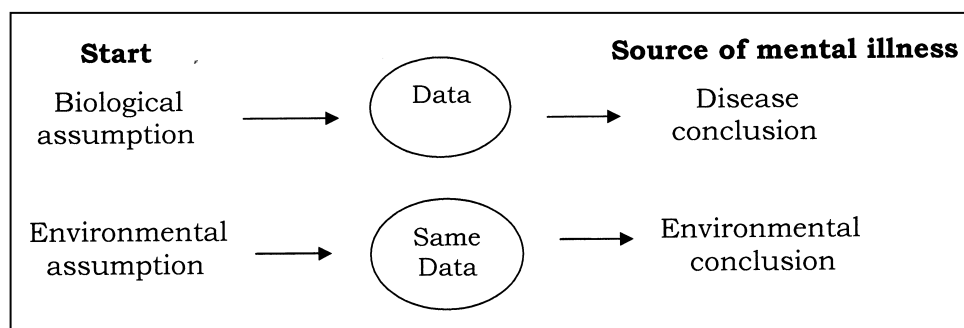


Figure 1. Conclusions based on personal assumptions.

Obviously, the above point regarding researchers’ personal assumptions is true because, once again, no pathophysiological evidence has been found and both sides admit to this. So the big question then becomes, “What assumptions, biological or environmental, are

incorrect?” One of the main goals of this report is to answer this question.

The Four False Pillars of Biopsychiatry

To expose the basic fallacies behind the research used to attempt to establish a biological basis for mental illness, I have divided the research into what I refer to as *The Four False Pillars of Biopsychiatry*. These four pillars are as follows:

False Pillar #1: *The Inheritance Pillar.* Mental illness runs in families; therefore, mental illness must be inherited.

False Pillar #2: *The Chemical Imbalance Pillar.* Medication works by correcting a chemical imbalance; therefore, mental illness must be a disease.

False Pillar #3: *The Defective Gene Pillar.* Defective genes have been found for some disorders; therefore, mental illness must be a genetic disorder.

False Pillar #4: *The Brain-Imaging Pillar.* Evidence of a “diseased” brain can be detected using modern brain-imaging instruments.

I will now briefly address the main tenets and fallacies of each pillar. Even though I will not present an exhaustive analysis of the research, once you understand the basic fallacies to each pillar, you will be able to understand the fallacies behind the new research or theories that will most likely be proposed in the future.

False Pillar # 1: The Inheritance Pillar *Mental illness runs in families; therefore,* *mental illness must be inherited.*

How many times have you either heard or read that “since mental illness runs in families, it must be inherited.” The truth is that an almost unlimited number of behavioral traits can run in families that are not genetic, such as language, food preferences, accents, religious and cultural beliefs, and the way people walk, talk, and so forth.

So how do researchers attempt to prove that mental illness is inherited? Before I answer this question, let me tell you that this pillar is perhaps the most important pillar of the four. In fact, as this pillar falls, so do the rest of the pillars.

This pillar is extremely important because, if researchers can illustrate or come to believe in some way that mental illness is inherited, they can then assume that defective genes are involved. Thus, if mental

illness is inherited and defective genes are involved, then biological psychiatry can literally (and it has) run into a thousand dead ends but still continue to believe in a biological model. In other words, if through studies researchers can conclude that mental illness is inherited, even though no defective genes, chemical imbalances, or anything else has been found, researchers can continue to believe in their basic model. But, if this one pillar is false, then for the most part, the rest of the pillars collapse.

The inheritance pillar is basically supported by three sources of data: (a) family statistics, (b) twin studies, and (c) adoptive studies. Once again for simplicity, I will focus on the research for schizophrenia. But remember, the argument presented for schizophrenia can be applied to the other mental health conditions, especially mania, depression, and ADHD. In fact, researchers Leo and Joseph state,

The genetic theory of schizophrenia is frequently cited as evidence in favor of a genetic predisposition to other conditions; the thinking being that if schizophrenia is genetic, then depression, obsessive compulsive disorder, attention deficit disorder and a host of other DSM-IV categories must also have their roots in problematic genes.³

Let's now start our analysis of this pillar with family statistics. But before we start, I need to state that much of the following information has come from the excellent investigative work done by Jay Joseph, PhD.

Family Statistics

Figure 2 is a typical graph of the rate of schizophrenia among relatives that is found in most basic psychology textbooks. The data for this graph originally came from Irving I. Gottesman's book, *Schizophrenia Genesis*.⁴

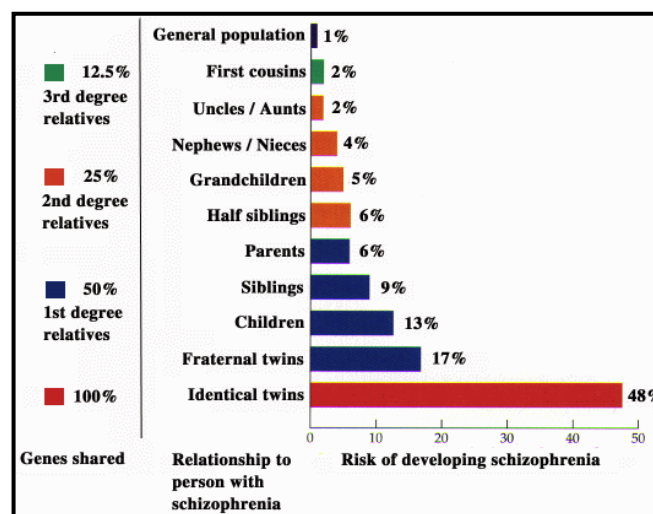


Figure 2. Rates of schizophrenia among relatives of schizophrenia.

As you can see by the graph, the closer the genetic relationship, the greater the concordance rate. According to geneticists, this is substantial proof that genetics plays a major role in the etiology of schizophrenia and other mental health disorders.

Even though, according to researchers Joseph and Leo, the data and methods used to develop this graph are loaded with major methodological errors,⁵ the data can still be interpreted one of two ways. First, the results can be seen as evidence of a genetic component. On the other hand, such rates could also indicate the importance of the family environment in the development of schizophrenia. Thus, the *only* correct and logical conclusion that can be drawn from this graph is that the family can play an important role in the development of the symptoms of schizophrenia as well as other mental health disorders.

Studies of Twins

As shown in the Figure 2 graph on family statistics, the identical-twin concordance is close to 50% (48%). If you look at any of the textbooks on psychopathology, the rate quoted will be from 40% to 50% and even higher. In fact, the National Institute of Mental Health (NIMH) states that “The identical twin of a person with schizophrenia is most at risk, with a 40 to 65 percent chance of developing the disorder.”⁶

Of all the data pertaining to the different pillars, this figure is the most important and most widely used figure to support a genetic model and thus the biological model in general. For example, a research article may start off with something like, “Since the concordance rate for identical twins is about 50%, we can assume that schizophrenia is a biological disorder.”

As another example, I was attending a seminar where the presenter was discussing the efficacy of a new antipsychotic drug. The very first slide in his presentation was a big “50%” that covered practically the whole screen. The presenter, a psychiatrist from a major research university, then said, “Since the identical twin concordance rate for schizophrenia is 50%, we can conclude that psychiatric medication works by correcting a chemical imbalance due to genetic defects.” With this assumption, he went on to attempt to sell the benefits of that particular drug to an audience consisting of fellow psychiatrists.

A twin-studies research project is basically set up in the following way. To determine the concordance rate between identical twins, first the researchers locate a group of individuals who have been diagnosed with schizophrenia and who also have an identical twin. Then the researchers attempt to locate the other identical twin to determine whether or not that twin has been diagnosed with schizophrenia. Then a concordance rate is calculated.

For example, let’s say the researchers located an original group of 100 identical twins diagnosed with schizophrenia (called probands).

Then, when the twin siblings have been located, let's assume 47 out of the 100 have also been diagnosed with schizophrenia. The concordance rate for this study would then be 47/100 or 47%.

Let's now take a look at the actual data that is used to support this 40% to 50% and higher figure. Figure 3 lists the published studies between 1920 and 1987.

MZ Twin Concordance rates	
Luxenberger (1928)	59%
Rosanoff et al. (1934)	61%
Essen-Moller (1941/70)	29%
Kallmann (1946)	69%
Slater (1953)	68%
Inouye (1961)	36%
Tienari (1963/75)	15%
Gottesman & Shields (1966)	42%
Kringlen (1967)	27%
NAS-NRC (1969/83)	18%
Fischer (1973)	36%
Koskenvuo et al. (1984)	11%
Average of studies	40%

Figure 3. Schizophrenic twin studies with corresponding pairwise MZ concordance rates.⁷

These are pairwise rates, but some studies also reported proband-wise rates. Notice that the monozygotic (MZ) rate using the pairwise statistical method of all the studies is 40%, not 50%. A proband-wise statistical method was developed specifically for studies conducted regarding schizophrenia. It, however, inflates the average rate closer to the 50% often quoted.

Whereas in the pairwise method the probands are counted only once, they are counted twice in the proband-wise method if each of a pair of twins concordant for schizophrenia is determined independently.

For example, assume Joan and Joanne are identical twins, both diagnosed with schizophrenia. If in the process of finding 100 probands of identical twins to compare with their twin siblings, Joan and Joanne were both in the original 100 proband group, they would be counted as only *one* pair in the pairwise method. They would be counted as *two* probands (counted twice) in the proband-wise method. It is this double counting that inflates the concordance rate in the proband method.

Again, it is important to note at this point that we are not examining actual biological evidence, but numbers that can be easily manipulated through the use of different statistical methods. Simply using the proband-wise method rather than the more conservative pairwise method can often inflate the concordance rate by 25%.⁸

You may recall it was Mark Twain who stated that “There are three kinds of lies, lies, damn lies, and statistics.”

Second, and more importantly, notice that the studies range all the way from a high of 69% to a low of 11%. How can such a wide variation exist with the simple process of figuring concordance rates?

If you notice, most of the higher rates were also from the earlier or older studies. There were two major interrelated problems with the earlier studies: (a) poor research methodology, and (b) strong sociological biases on the part of the researchers.

The research or methodology inadequacies were the result of the fact that the studies were often not blind (in which researchers were personally aware of enough of the particulars to insert a bias), the diagnostic criteria were inconsistent, and at that time, the zygosity of the individuals could not be determined by medical means.

For example, if one identical twin had been diagnosed with schizophrenia and placed in a hospital for several years, then, due to his emotional condition and the extremely poor conditions of the hospital, several years later this twin may look quite different from the other twin. If the twins were identical, but not concordant for schizophrenia, the researchers could eliminate this pair by claiming that the twins were not identical. This would benefit their results in favor of a higher concordance rate by boosting the concordant twins found versus the non-concordant twins.

In one particular recorded example, researchers were investigating a set of twins, one of them hospitalized and diagnosed with schizophrenia and the other one still living at home. By interviewing the parents and neighbors, to the best of their knowledge, this pair was identical.

When the researchers actually attempted to interview the at-home twin to determine if the twin male was suffering from schizophrenia, the parents told the researchers that their son stayed upstairs most of the time and did not want to come down to be interviewed.

So, was this twin also suffering from schizophrenia (paranoid of people), or was he simply afraid that if he came down he may be hospitalized and labeled like his brother? In other words, his fears may have been legitimate.

But here is the dilemma that the researchers faced in this situation, as well as in others. If they did not diagnose him with schizophrenia, this case would swing the data in favor of a non-biological explanation. If they diagnosed him with schizophrenia, that would swing the data in favor of a genetic or biological explanation.

In addition, a major sociological bias was involved. Today researchers use this grossly exaggerated figure (50%) to continue the myth of a biological model. But the researchers of the earlier studies used the data for a terrible and deceptive reason.

The earlier, more inaccurate, and inflated rates were used to support the growing eugenics and Social Darwinism movements,* which claimed that certain groups of individuals (the physically disabled and especially the “feeble-minded”) should be prevented from procreating. Evidence from twin studies purporting genetic inferiority lent credibility to the passage of sterilization laws applicable to these groups first in the United States and then in Germany. This “evidence” also fueled Hitler’s racism and provided additional rationalization for the Holocaust.

In order to illustrate the inherent bias of the twin-study researchers, it is necessary to identify those prominently engaged in the research. Francis Galton (1822–1911), a British statistician and cousin of Charles Darwin, became the father of the eugenics movement. He originated the idea that twin studies would provide a way to measure differences associated with either inheritance or environment. Yet it was Ernest Rüdin (1874–1952) who became the recognized founder of psychiatric genetics.

Rüdin was an internationally known psychiatrist and eugenicist who firmly believed that schizophrenia was an inherited disorder. His Munich research department became the pioneering center for the study of psychiatric genetics. Rüdin conducted the first family-prevalence study regarding schizophrenia, the results of which were published in 1916.⁹

While in high school, Rüdin formed a students’ temperance association and attracted attention for his uncompromising position on racial hygiene. In 1901, he received his doctorate and went to work for Emil Kraepelin. In 1903, at the age of 29, Rüdin formulated his program for a future “racially pure utopia,” and in 1909 he became the senior physician at Kraepelin’s Munich hospital. The first twin study regarding schizophrenia was performed by one of Rüdin’s top associates, Hans Luxenberger. In fact, the authors of four of the first five twin studies regarding schizophrenia (Luxenberger, Essen-Moller, Kallmann, and Slater; see Figure 3) were students at Rüdin’s Institute for Psychiatric Research in Munich, Germany. The above four researchers trained several of the other researchers, including Shields, Gottesman, and Fischer.

When Hitler came to power, Rüdin served on a panel with Heinrich Himmler. Himmler was chairman of the *Task Force of Hereditary Experts*. This task force drew up the German sterilization law of 1933 for “congenital mental defects,” which included schizophrenia and manic-

* Eugenics is the study of, or belief in, the possibility of improving the qualities of the human species or a human population by such means as discouraging reproduction by persons having genetic defects or presumed to have inheritable undesirable traits (negative eugenics), or encouraging reproduction by persons presumed to have inheritable desirable traits (positive eugenics).

Social Darwinism is a 19th century theory inspired by Darwinism, by which the social order is accounted as the product of natural selection of those persons best suited to existing living conditions and in accord with which a position of laissez-faire is advocated.

depressive psychosis. Seven years later, approximately 70,000 mental patients had been put to death under the Nazi “euthanasia” program. In 1991, psychiatrist Peter Breggin wrote, “Rüdin embodies the direct link between psychiatry and Hitler.”¹⁰ To say the least, twin studies became the cornerstone of psychiatric genetics and the growing eugenics and Social Darwinism movements because they purportedly provided the evidence necessary to label mental illness as a disease and to “purify the race” by precluding the mentally ill from procreating.

Most Accurate Studies

Some of the later studies, such as Kringlen, Fischer, and Onstad, used only hospital records that can also create a sampling bias in favor of higher concordance rates.¹¹ Only two studies use the more accurate, register-based data and do not use hospital records.¹² Figure 4 shows the results of these two studies (taken from Figure 3). This 17% figure is a long ways off from the 40% to 65%.

Schizophrenic Twin Studies Pairwise Concordance rates	
Tienari (1963/75)	15%
NAS-NRC (1969/83)	18%
Average of studies	17%

Figure 4. Schizophrenic twin studies using register-based data.

How Rates Can Be Manipulated

Let me now offer you an example of how rates can be inflated over time. I admit this is an extreme example, but it illustrates what researchers can do with data when there is no pathophysiological evidence to support the data.

Hoffer and Pollin’s 1970 study had the second largest sample of twins and came close to having the twins diagnosed blindly. The study resulted in an age-corrected concordance rate of 15.5%.¹³

In 1972, Allen, Cohen, and Pollin re-analyzed the data and came up with a new concordance figure of 27%.¹⁴ When Mary Boyle looked more closely at their analysis, she noted that the diagnostic criteria had been widened and that the authors had evaluated each twin pair knowing their zygosity status.¹⁵

The authors admitted that while reviewing the data, “the reviewer may have been influenced toward seeing more similarities in MZ pairs than DZ [dizygotic] pairs.”¹⁶ This factor would tend to increase the MZ concordance rate.

Gottesman and Shields, who are strongly biased towards the genetic model, took the 27% pairwise figure and recalculated it using the proband-wise method, coming up with a figure of 43%.¹⁷ Expanding the diagnostic criteria even more, in 1991 Gottesman came up with a rate of

53%.¹⁸ So by manipulating the data, researchers increased the concordance rate from an insignificant 15.5% to an overwhelming 53%.

In summary, it is downright discouraging and frightening to understand that the same highly distorted data that was used to support the holocaust is now used to support the medical model.

Monozygotic vs. Dizygotic Twin Studies

Another way of using twin studies is to compare the concordance rates between identical or monozygotic (MZ) twins with same-sex non-identical dizygotic (DZ) twins.* Since DZ twins only share 50% of their genes and MZ twins share 100% of their genes, any major differences can be assumed to be the possible result of a genetic influence.

For example, if you go back to the chart on family statistics (Figure 2), you will see that the identical twin concordance rate is 48%, whereas the non-identical (fraternal) rate is only 17%. So is this evidence of a genetic influence? Not necessarily.

In order for the MZ-DZ difference to show evidence of a possible genetic disorder, the *Equal Environmental Assumption* (EEA) must be validated. The EEA claims that the environmental influences on fraternal same-sex twins are basically the same as with identical twins. If fraternal twins experience the possibility of a greater environmental difference than identical twins, the differences between the MZ and DZ rates could be accounted for by environmental factors.

Let's use an example to make sure the EEA is clearly understood. Compare one set of identical twins where both of them are attractive and athletic with a set of fraternal twins where one is attractive and athletic and the other is not as attractive or athletic. The two identical twins will likely be treated much more alike than the two fraternal twins. The good-looking athletic fraternal twin will most likely receive more attention than the other fraternal twin. On the other hand, the good-looking and athletic fraternal twin may be subjected to much more pressure to succeed. Regardless, there is a strong possibility that one twin will be treated much differently than the other and that the twins will also want to be seen as different.

This is a simple example, but investigators have often reached the same conclusion. For example, Harold Carter, a twin researcher, writes,

The assumption that the nurture influences are approximately equal for fraternal and identical twins... seems untenable to anyone who has had much contact with twins in their own social environment... Identical twins obviously like each other better, they obviously have the same friends more often; they obviously spend more time

* Identical or monozygotic (MZ) twins originate from the fertilization of the same ovum, whereas fraternal or dizygotic (DZ) twins originate from the fertilization of two ova.

*together; and they are obviously treated by their friends, parents, teachers, and acquaintances as if they were more alike than fraternal twins are.*¹⁹

In summary, not only do the modern day textbooks, as well as the researchers, fudge on the twin concordance rates, they also fail to address the key issue, which is the *Equal Environment Assumption*.

Adoption Studies

First of all, it is worth noting that adoptive studies are much more difficult and expensive to conduct. Thus, if conclusive results could have been deduced from family and twin statistic studies, adoptive studies would not have been necessary.

Adoptive studies attempt to eliminate the “environmental” factor by assuming that, since the children in the study have been removed from their biological families at an early age, the biological family environmental element can be excluded from the equation. According to researchers, adoption studies show an elevated risk for schizophrenia among the offspring of mothers with schizophrenia.²⁰

This simply means that adoptive children of biological mothers diagnosed with schizophrenia have a greater chance to be diagnosed with schizophrenia than adoptive children whose biological mothers were not diagnosed with schizophrenia. Since the environmental element is theoretically eliminated by the children’s not living with or being raised by their biological parents, a genetic component is then assumed to exist.

Even though many methodological problems are involved with adoption studies, the main factor that undermines a genetic conclusion is the issue of “selective placement.” It has been shown that adoptees who were born into highly dysfunctional families have a much greater chance of being adopted into dysfunctional families. Thus, many of the adoptive children who eventually were diagnosed with schizophrenia literally started out in one toxic situation and then were placed in another toxic situation. Lewontin, Rose and Kamin stated that the selective placement factor alone “undermines the theoretical separation of genetic and environment variables claimed for adoptive studies.”²¹

For example, when the data from the most important adoption study for schizophrenia (Kety et al., 1968)²² was analyzed, it was determined that selective placement was a major factor, since 24% of the index adoptive parents had a history of hospitalized mental illness, but not one single control parent did.

In other words, selective placement had occurred because the index children (those diagnosed with schizophrenia), who came from families with a much higher incidence of mental illness, were then placed in adoptive homes where 24% of the parents had been hospitalized for a psychiatric condition. Thus, this data would definitely point towards an

environmental model—not a genetic one. Lewontin, Rose, and Kamin had some strong remarks in reference to this study in particular:

*The schizophrenic adoptees, who indeed had been born into shattered and disreputable families, acquired their schizophrenia as a result of the poor adoptive environments into which they were placed. The fact that one's adoptive parent goes into a mental hospital clearly does not bode well for the psychological health of the environment in which one is reared.*²³

As with the twin studies, the propaganda for the adoptive method has been relentless in favor of a genetic model. For the most part, neither the public nor the professional community is offered a clear and honest picture of these studies. Yet, in reference to the above schizophrenic adoption studies, Gottesman and Shields referred to these studies as “the straw that broke the environmentalists’ backs.”²⁴

Summary of False Pillar #1

Once again, the only conclusion that can be drawn from this pillar is that the family plays an important role in the emotional development of the child. But it is *improper* to conclude by these studies alone that such disorders as schizophrenia are genetically based.

As a final note, Loren Mosher, who oversaw much of the earlier research on schizophrenia at the NIMH when some of the major inheritance studies were funded, stated that Nazi Germany developed the best genetic research project possible. They either killed or sterilized a whole population of individuals diagnosed with schizophrenia. Yet within one generation, the same incidence of those diagnosed with schizophrenia returned. If the disorder had been truly genetic, such a quick return would not have been possible.

With this pillar behind us, I will now attempt to progress through the other three pillars more rapidly.

*False Pillar #2: The Chemical Imbalance Pillar
Medication works by correcting a chemical imbalance;
therefore, mental illness must be a disease.*

As with the first pillar, I am sure you have heard the saying that psychiatric medications work by correcting an imbalance. Again, part of this false belief is based on the validation of the first pillar; that a true defect of some kind exists. In fact, psychiatrist and researcher Nancy Andreasen presents the hope of this model in the following quote:

*Scientists will create medications that will correct the abnormal expression of disease-producing genes. Molecular biology will someday permit us to perform psychosurgery at the level of the gene.*²⁵

The chemical imbalance model has been used by psychiatry and the drug companies to promote the selling of these drugs, claiming in essence that these drugs are necessary to correct a defect. Such a model may have made some sense at the beginning.

Soon after these drugs were introduced, it was discovered that they work by affecting the flow of certain neurotransmitting chemicals. In order for a neuron to fire, neurotransmitters are released from the end of one neuron, flow across a small gap to the next neuron, and then attach themselves to that neuron. If enough of these neurotransmitting molecules are able to attach themselves, the next neuron fires and the particular electrical message continues.

At first, science did not have the technology to test or determine whether chemical imbalances existed. So the two sides, biological versus environmental, began to battle it out on this issue. The biological side claimed a chemical imbalance was involved, whereas the environmental side claimed just the opposite, that the drugs were similar to alcohol or street drugs, in that they worked by disabling the brain.

Today, science has the necessary technology to determine whether or not chemical imbalances exist, but none have been found. It is this fact that has prompted the present (2014) director of the NIMH, Thomas Insel, to state that “earlier notions of mental disorders as chemical imbalances or social constructs are beginning to look antiquated.”²⁶ Also, William Whirling, M.D., professor of psychiatry at UCLA, stated to a room full of psychiatrists that “We have been misleading the public about the chemical imbalance model for 40 years.”²⁷ Finally, ex-director of the NIMH, Steve Hyman, upon summarizing over 40 years of research pertaining to the effects of antipsychotic drugs, reported that the use of the drugs actually creates, rather than corrects, a biochemical imbalance within the brain and in fact works similar to drugs of choice or street drugs.²⁸

If you think about it, the disabling model makes much more sense. How many individuals use alcohol and/or drugs to block out painful emotions, to live with intense loneliness, or simply to feel relaxed at an important social gathering?

No chemical imbalances exist, and that is a fact, not a theory. Drugs may be useful and needed at times, but they do not support a disease or defect model of mental illness.

False Pillar #3: The Defective Gene Pillar
Defective genes have been found for some disorders;
therefore, mental illness must be a genetic disorder.

Simply put, researchers have searched the entire human genome, as well as combed the entire world looking for appropriate multiple-generational families to study. *To date, not one single gene for any so-*

called mental illness has been identified. They find markers or “possibilities,” but when other researchers attempt to duplicate the results, they cannot, and the original claims have had to be retracted.

Because no single gene has been found for any mental disorder, to overcome this problem researchers now believe that they are unable to find a single gene because several genes are involved, each playing a small role. As “silly” as this conclusion may seem, researchers still plug along because of—you guessed it—the first pillar. Quoting from an article titled “Genetic basis of complex human behavior,” the authors stated, “it is possible that there are no genes of major effect to be found despite clear twin and adoptive evidence for genetic influence.”²⁹ “Clear evidence”! Again, without the first pillar, biopsychiatric research would not have a leg (pillar) to stand on.

At this point I want to move on to the last pillar, the brain-scan pillar. Researchers are now using brain-scanning equipment to attempt to find defective genes, so I will finish up Pillar #3 after explaining the fallacies of Pillar #4.

*False Pillar #4: The Brain-Imaging Pillar
Evidence of a “diseased brain” can be detected
using modern brain-imaging instruments.*

Because no chemical imbalance, defective gene, or any other pathophysiological evidence has been identified, this pillar has become biopsychiatry’s last great hope. With the brain-scanning equipment now available, researchers can measure or take a picture of actual differences in the brain of a person diagnosed with mental illness and then compare it to a so-called normal person.

Structural Studies

Figure 5 shows a set of magnetic resonance imaging (MRI) pictures that can be found in almost any basic textbook on psychopathology or general psychology. The image on the right is that of a person diagnosed with schizophrenia. It reflects an enlargement in the lateral ventricular area (notice the arrows in the diagram) of the brain. The brain scan of a non-schizophrenic or normal person on the left shows no enlargement. Do these images then represent actual biological evidence of a diseased brain?

A few years ago, researchers Chua and McKenna decided to take an extensive look at all the brain-scan research concerning schizophrenia. They published a massive 20-page journal article in the *British Journal of Psychiatry* reporting that the most “consistently replicated brain abnormality is structural, and takes the form of lateral ventricular enlargement.”³⁰ Even with this finding, they called it a “risk

factor,” not necessarily connected to the cause of mental illness. Before we go any further, let’s make sure we understand the primary role of the ventricles in the brain.



Figure 5. Lateral ventricular enlargement differences.

The brain is very soft and jelly-like. It must be protected from shock because of its considerable weight (approximately 1400 g) and its delicate construction. Outside the skull, a human brain cannot even support its own weight.

The brain contains a series of interconnecting chambers called ventricles. The brain is well protected and supported by the cerebrospinal fluid contained in the ventricles. The brain’s net weight immersed in this liquid is only 80 g. Shock from a sudden blow or even mild jarring is cushioned by the cerebrospinal fluid. Without the protection of this fluid, the brain would bounce back and forth within the skull, causing considerable damage.

Figure 6 shows a picture of the ventricular system of the brain.

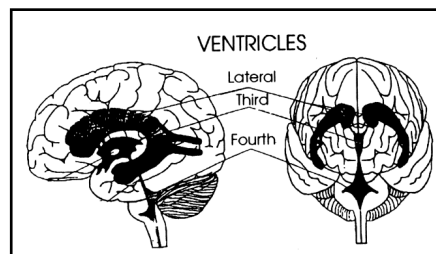


Figure 6. Ventricular system of the human brain.

The largest chambers of the ventricular system are the lateral ventricles. Because the ventricles show up vividly on imaging instruments, they serve as important structural landmarks. When parts of the brain have atrophied (shrunk), the ventricles enlarge to compensate; therefore, they are important indices of possible brain diseases.

With evidence of a ventricular enlargement in the brain of a person diagnosed with schizophrenia, and with the assumption that schizophrenia is a disease, it would only be natural to assume that the brain had atrophied due to the disease of schizophrenia, whatever that is. But because no pathophysiological evidence of a disease has been found, perhaps there are other explanations for ventricular enlargement (brain atrophy or shrinkage). In fact, stress may be the major factor.

When someone is under stress, more cortisol is secreted into the brain tissue area than is secreted under normal circumstances. As a result, the brain loses some of its ability to absorb water. The brain then shrinks slightly because it is holding less water, not because it is affected by some disease.

As the brain shrinks, the ventricular areas *must* become larger, with more cerebrospinal fluid used to make up for the difference in volume.

In other words, just as our body perspires or begins to shiver to compensate for body temperature changes, the ventricular area increases and decreases as an adjusting mechanism. Thus, the enlargement is not a pathophysiological state but a *natural* protective process.

In addition, such factors as depression, early childhood trauma, and alcoholism can result in ventricular enlargement.³¹

Loss of Gray Brain Matter

As brain-imaging equipment and technology have advanced, the claim that schizophrenia and bipolar disorders in particular are true brain diseases has also increased. The following is an actual set of MRI brain scans showing how the loss of gray matter (brain cells) has spread over time.³²

To obtain these images, measurements of a group of diagnosed individuals is compared to a group of non-diagnosed individuals to see if there is a loss of gray matter over time. As the images are taken, every two years for example, without a doubt the images do show a loss of gray matter in the diagnosed individuals versus the non-diagnosed individuals. In Figure 7, the red (versus the blue) areas illustrate where the loss has occurred.*

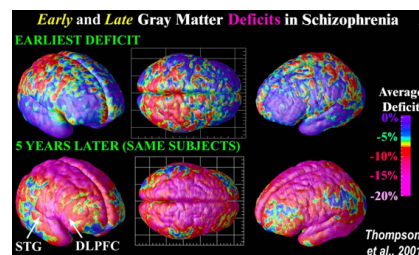


Figure 7. Loss of gray matter over time.

* Visit (www.schizophrenia.com/family/disease) for better colored pictures.

From the perspective of the biopsychiatrists, these results are quite spectacular and convincing, leading to headlines such as the following:³³

Studies of never-treated patients confirm Schizophrenia is a brain disease.

People with bipolar disorder-or manic depression suffer from an accelerated shrinkage of their brain.

UCLA brain researchers using a powerful new technique have created the first images showing the devastating impact of schizophrenia on the brain.... The findings show how a dynamic wave of tissue loss engulfs the brain of schizophrenic patients in their teen-age years.

Lack of gray matter in brain is linked to Schizophrenia and Bipolar disorder.

It [schizophrenia] moved across the brain like a forest fire, destroying more tissue as the disease progresses.... scientists detected gray matter loss of more than 10 percent....

The above headlines represent statements by some of the best credentialed researchers in the world, using the most advanced equipment possible. These statements and images can be quite startling to those diagnosed with a mental illness, as well as to their loved ones. So, do researchers now have conclusive, non-debatable evidence that the disorders of schizophrenia and bipolar disorder are true brain diseases? Let's first make sure a couple of points are understood before we attempt to answer this present challenge.

What Is Gray Brain Matter?

Gray matter is made up of neuronal cell bodies. The gray matter includes regions of the brain involved in muscle control, sensory perception such as seeing and hearing, memory, emotions, and speech. In living tissue, gray matter actually has a gray-brown color, which comes from capillary blood vessels and neuronal cell bodies.

What Can Cause the Loss of Gray Matter?

Actually, a whole list of factors can possibly cause the loss of gray matter. Some of these include smoking, lack of sleep, lack of exercise, such medical conditions as fibromyalgia, and, finally, stress. Since there is considerable evidence and agreement among professionals that stress can cause the loss of gray matter, let's look at this element more closely.

Stress and the Loss of Gray Brain Matter

Bruce McEwen, head of neuroendocrinology [should there be an e] at the Rockefeller University in New York, has reported that rats subjected to repeated exposure to stress experience shrinkage of the

neurons in the hippocampus and the prefrontal cortex.³⁴ In addition, an examination of the brains of soldiers suffering from PTST showed a 5 to 10 percent loss of gray matter volume, indicating neuron damage.³⁵

In fact, as far back as 1996, researcher and professor Sapolsky used MRI brain scanning equipment to show that stress can shrink the brain. Quoting Sapolsky from a news release, “The work of several research groups shows links between long-term stressful life experiences, long-term exposure to hormones produced during the stress, and shrinking of the part of the brain involved in some types of memory and learning.”³⁶

So, let me ask the obvious question: Are mentally ill persons, especially the chronically ill, exposed to any of the above factors? Are excessive smoking, lack of sleep, lack of exercise, and especially stress often a part of their daily lives?

I believe so. If there is a loss of gray matter associated with mental illness, then it seems foolish to assume that some gene or group of genes can be responsible. Why is it so hard for researchers to put two and two—or actually one and one—together? Stress sets off a whole bunch of chemicals in the brain, including cortisol, which has been called the “death hormone” by Dr. Nicholas Perricone. In his words,

*Large amounts of cortisol are toxic when they circulate in our system for prolonged periods of time. Our brain cells, or neurons, are extremely sensitive to the effects of cortisol. When cortisol is circulating at a high level, it causes the brain cells to die.*³⁷

Over time, these chemicals damage the brain cells. This is a proven fact, not a theory. But this does not mean that the symptoms of schizophrenia (or other major mental disorders) are the result of a physically defective brain. This is correlational, not causal evidence.

Psychiatric Medications Causing Brain Atrophy

There has also been an ongoing and quite heated argument regarding whether the medications given to mentally ill persons result in brain damage. Because both sides can back up their position with supposedly “valid” research, I will stay away from this debate at this time, except to say that it makes common sense to me that these drugs, which are foreign substances to the brain, would eventually have a destructive effect on the brain. Again, Steven Hyman, the ex-director of the NIMH, states that these drugs work in a fashion similar to that of drugs of choice or street drugs. In addition, psychiatrist Grace Jackson recently reported a tenfold increase in Alzheimer’s and a 25-year shortening of a person’s life span due to the use of antipsychotic drugs.³⁸

But here is the main point: Whatever causes differences in the structure of the brain, it is incorrect to conclude that such differences are related to or cause mental illness. There is substantial evidence that

such differences are related to emotional and/or environmental stress. Actually, neither side disputes this “fact”.

Functional Studies

The aforementioned studies are considered structural studies because they examine different structural parts of the brain to see if there are actual physical differences between a so-called normal brain and the brain of a diagnosed mentally-ill person. The other kind of study or measurement used today is referred to as a *functional study*. Functional studies measure differences in the rate of glucose metabolism to different parts of the brain. Glucose is a form of sugar and the primary source of energy for brain cells. Like structural studies, functional studies also attempt to compare differences between someone diagnosed with a certain mental illness to someone without such a diagnosis.

But, again, this is correlated evidence, not causal evidence. In other words, just because two things occur at the same time, it is improper to conclude that one causes the other or is evidence of a causal event.

Here is the example I like to use to simplify the major error involved. At the age of seven, my son was attacked by a large dog and severely bitten. In fact, if the owner had not retrieved the dog, the results could have been life-threatening.

Several months later I was walking with my son and his friend across a field when my son spotted a large dog running loose. He instantly became quite nervous and moved closer to me for protection, while his friend did not seem frightened at all. If somehow I could have measured how each of their brains were metabolizing glucose (energy consumption), I am sure I would have seen some differences. Because of my son’s traumatized, near-death experience, obviously he would have been using parts of his brain differently with the threat of another dog running loose.

So, does that mean that my son’s anxious or fearful reaction was the result of some brain defect or that his brain was simply operating in an understandable survival mode? Yet this is exactly how researchers attempt to draw their conclusions using functional brain-scanning equipment.

When I was first trying to make sense of this research, I was able to obtain an appointment with a top brain-scan researcher at a local research university. After a short time in his office, I finally asked him how he could determine whether or not the results pointed towards a biological or environmental conclusion. He told me that no one could, but since the concordance rate for schizophrenia is 50%, researchers can conclude that the results indicate a biological origin. When I began to

discuss the problems with the 50% figure, he suddenly told me he was quite busy and that he needed me to leave.

It was obvious to me that he knew something about the major fallacies behind his research conclusions but did not want to admit to it.

New Model and Attempt to Find Defective Genes

Some of the technological advancements now available to researchers have resulted in a new approach to locate possible defective genes. Instead of combing the human genome directly, researchers now start with evidence acquired from functional brain scans (Pillar #4), then attempt to locate defective genes from this data (Pillar #3). If genes could be found, then this would automatically validate Pillar #1 and subsequently give hope to developing more precise medications to correct the chemical imbalances (Pillar #2).

The model for this approach is actually quite easy to understand. The researchers start by first using functional brain scanning equipment to locate possible differences in how a diagnosed person, versus a non-diagnosed person, metabolizes glucose. After locating an area in the brain where there is a *difference*, researchers then attempt to locate the genes that are responsible for the activation of that area of the brain. Once these genes are located, they then attempt to determine if any of these genes are defective.

Let me return to the dog-biting incident with my son for clarity. Let's assume that my son was showing signs of paranoia over going outside to play without the protection of a parent. Let's also assume that the researchers were unaware or did not take the time to become aware of the dog-biting incident because they were primarily interested in brain structural and functional differences.

The researchers then subjected my son to a PET scan while they had him anticipate going outside. Sure enough, a particular part of his brain was metabolizing glucose at a much higher rate than a normal person. How then do researchers attempt to find a possible defective gene from this data?

Several years ago researchers at the Allen Institute for Brain Science developed an online interactive atlas of the human brain showing the activity of the more than 20,000 human genes. With this atlas, scientists can now determine where, in the brain, genes that encode specific proteins are active. Using the atlas, researchers can zoom in on brain structures thought to be altered in mental disorders such as schizophrenia to find the molecular footprint of these diseases.

Such a map could possibly enable scientists interested in the role of a particular gene or set of genes in, for example, depression, to bypass the tedious and expensive laboratory work needed to examine possible

molecular culprits of the disease, one at a time. Instead researchers could “theoretically” search the atlas to see where in the brain genes are active as well as what other genes, active in the same regions, may be involved.

In other words, since researchers now know what genes affect what areas of the brain, if the researchers simply *assume* that the differences that they see is evidence of a defective brain, then defective genes must be involved. But again, they must go all the way back to trust the data in Pillar 1 to claim any validation to their conclusions.

So what are the present results as of 2011 using this new technique of starting from Pillar #4 or brain scans and then attempting to identify the specific so-called defective genes? Again, when researchers first began their hunt for defective genes, they were hoping to find one main dominant gene responsible for schizophrenia. But when no genes were found, researchers began to claim that part of the problem may be that as many as 10 genes were involved, each playing a role. Then the number grew to 20, still with no actual gene found. Soon after that, the number of possible genes grew again, to 100.

With this new research approach, scientists are now claiming that perhaps as many as 900 genes are involved.³⁹ Yet not a single one has been identified as causing any psychiatric condition. Read Colin Ross’s response to the possible involvement of so many genes:

Imagine if the protein products of each of these hundreds of genes were identified. Then imagine that the levels and functions of each of these proteins could be identified in an individual with a mental disorder. This would require testing for the intracellular concentration of hundreds of different proteins. Targeting any one of these gene products would have to take 25–50 medications to reduce his or her symptoms by 50%. It is hard to imagine how any person could tolerate the side effects and drug–drug interactions of 25–50 medications.⁴⁰

In addition, if several hundred or more genes are involved in schizophrenia, how many others might result in depression and mania? And what about schizoaffective disorder, a cross between schizophrenia and manic-depressive disorder? Are there different genes for each separate disorder? What about schizophreniform, a milder form of schizophrenia?

Summary of the Four False Pillars

The following summarizes the different pillars.

False Pillar #1, The Inheritance Pillar: *Mental illness runs in families; therefore, mental illness must be inherited.*

Inheritance studies make a strong case that families can at times play a key role. But the studies cannot determine if this role is due to biological or environmental reasons. On the other hand, both biopsychiatric and non-biopsychiatric followers admit that, environmentally, the family does play a key role.

False Pillar #2, The Chemical Imbalance Pillar: *Medication works by correcting a chemical imbalance; therefore, mental illness must be a disease.*

With the latest technology, it is possible to determine if a chemical imbalance exists; none has been found. On the other hand, it is also an accepted fact that when psychotropic drugs are used, major changes in the brain occur. For example, with the use of both the older and newer antipsychotic medications, upwards of 87% of the dopamine receptors are blocked.⁴¹ Quoting Peter Breggin in reference to the antidepressant Prozac, “the receptors actually die off or disappear”; and with rats, “Up to 60% of some subtypes of serotonin receptors can disappear.”⁴² Continuing, Breggin stated,

*Prozac once again illustrates the principle that the differences between therapeutic effects and adverse effects are merely steps along a continuum from mild to extreme toxicity. Drug-induced feelings of wellbeing can be understood as an early stage along the continuum from mild euphoria to mania, sometimes combined with a generalized dulling or flattening of emotional responsiveness and social sensitivity.*⁴³

In other words, the drug does not correct some imbalance but may make people feel better (there is a powerful placebo effect with this drug) because (a) it has a stimulant effect, and (b) it helps to suppress the person’s emotional pain.

Studies have also shown that the drug Ritalin, used for ADHD, affects the same areas of the brain as cocaine.⁴⁴ Thus, the only honest conclusion is that *these drugs produce, rather than correct, an imbalance.*

False Pillar #3, The Defective Gene Pillar: *Defective genes have been found for some disorders; therefore, mental illness must be a genetic disorder.*

Again, with the new technology, researches have now scanned the entire human genome and have not been able to find one defective gene for any mental disorder. By believing that as many as 900 different genes may be involved with schizophrenia alone, biopsychiatry is unable to admit defeat and continues to request additional research funds to keep their model alive.

False Pillar #4, The Brain-Imaging Pillar: *Evidence of a “diseased” brain can be detected using modern brain-imaging instruments.*

Because no pathophysiological evidence such as lesions exists, brain scans can only measure differences. There is substantial evidence that these differences are due to stress, drug therapy, and normal adjustments to difficult situations (i.e., fear of dogs). There is no evidence that these differences are due to some biological diseased condition.

Test Time

Let's now see if you can pass a test on the *Four False Pillars of Biopsychiatry*. The following is an explanation of the possible causes of schizophrenia taken directly from the website of the National Institute of Mental Health on January 25, 2015.⁴⁵ As you will see, I have identified the four false pillars in their attempts to give evidence to a biological model for schizophrenia. My notes are in brackets. I have deleted some of the irrelevant material. Refer to their website for a full description and update. The following is a reprint of the pertinent information from their website, with my added comments shown in square brackets and italics.

NIMH—What Causes Schizophrenia?

Experts think schizophrenia is caused by several factors. *[Obviously they don't know yet.]*

Genes and environment. Scientists have long known that schizophrenia runs in families... *[Pillar #1]* The risk is highest for an identical twin of a person with schizophrenia. He or she has a 40 to 65 percent chance of developing the disorder. *[A 40 to 65 percent chance? Remember the twin studies? The higher figures were developed by Nazi supporters of racial hygiene.]*

We inherit our genes from both parents *[Pillar #2]*. Scientists believe *[Believe = nothing found]* several genes are associated with an increased risk of schizophrenia, but that no gene causes the disease by itself... These genetic differences involve hundreds of different genes and probably disrupt brain development. *[Hundreds and probably?]*

Other recent studies suggest that schizophrenia may result in part when a certain gene that is key to making important brain chemicals malfunctions. *[Pillars #2 & #3—no chemical imbalance has been found or they would report it.]*

Scientists think interactions between genes and the environment are necessary for schizophrenia to develop. *[They are admitting to an environmental factor but also admitting to no biological factor at this time.]* Many environmental factors may be involved, such as exposure to viruses or malnutrition before birth, problems during birth, and other not yet known psychosocial factors. *[Obviously scraping the bottom of the barrel for different theories at this point.]*

Different brain chemistry and structure. Scientists think that an imbalance in the complex, interrelated chemical reactions of the brain involving the neurotransmitters dopamine and glutamate, and possibly others, play a role in schizophrenia *[Pillar #2 again]. [Can't find an imbalance in dopamine, for example, so now think an imbalance between neurotransmitters. But since hundreds of different neurotransmitters, this ought to keep this basic theoretical model and the research funds coming and going for a long time.]*

Also, in small ways *[small ways?]* the brains of people with schizophrenia look different than those of healthy people. *[Pillar #4]* For example, fluid-filled cavities at the center of the brain, called ventricles, are larger in some *[some?]* people with schizophrenia. The brains of people with the illness also tend to have less gray matter, and some areas of the brain may have less or more activity. *[You know about these concocted results.]*

You can bring up the same website to uncover the Four False Pillars for depression and ADHD if you wish. Again, I took this information from the NIMH website on January 25, 2015, and they will have the most up-to-date information. Even if you are still leaning towards a biological model, take into consideration the 200-year history of recovery through the proper use of psychotherapy and the fact that about one-third of all those diagnosed with schizophrenia recover with little or no help. In addition, in third-world countries where psychiatric care is at a minimum, the recovery rate for those diagnosed with schizophrenia is almost twice as great as in developing countries where individuals have access to present-day psychiatric care, including psychotropic medications.⁴⁶

Keys to Defeating the Four False Pillars of Psychiatry: A Quick Reference

Pillar #1 *Family statistics*—can't determine if environmental or genetic.

Twin studies—do not prove the equal environment assumption (EEA).

Adoptive studies—selective placement invalidates the studies.

Therefore, can conclude only that “somehow” families may play an important role.

Pillar #2 *No chemical imbalances* have been found after extensive research.

Pillar #3 *No defective gene found* that “causes” any form of mental illness after searching the entire human genome for years.

Pillar #4 *Brain scan studies:*

Structural—stress and psychiatric medications can and do result in brain atrophy.

Functional—differences found are the person's individual adjustment to stressful situations.

Some Final Notes

In doing additional research on the four false pillars, I ran across an article on the Internet titled, “Another blank on schizophrenia gene.”⁴⁸ The first line of the article reads, “Scientists have drawn another blank in their search for a universally applicable genetic explanation for schizophrenia, *strengthening* [my emphasis] the case for new approaches.”

The big question then becomes, if they have been searching for years and have not found a single gene, how does a multitude of dead ends justify strengthening additional research and, of course, funds for that research? A few lines further down they give us the answer: “Schizophrenia is known to involve genetic factors, since people who have relatives with the condition are more likely to have it, with 40 to 60% concordance between identical twins.”

Just as these twin studies were fundamentally important to Nazi Germany, so are they to today's researchers as the researchers continue to justify their position against absolutely no pathological evidence.

With the lack of any true cause and effect, pathophysiological evidence, biologically oriented psychiatry may actually be on the verge of bankruptcy. A May 6, 2013, article in *The New York Times* quoted leading experts pertaining to the present state of their “scientific” approach to the understanding and treatment of those suffering emotionally. Quoting from the article,

Decades of spending on neuroscience have taught scientists what they do not know, undermining some of their most elemental assumptions... The mechanisms of the field’s most commonly used drugs—antidepressants like Prozac, and antipsychosis medications like Zyprexa—have revealed nothing about the causes of those disorders.

*Dr. Insel [past director of the NIMH] is one of a growing number of scientists who think that the field needs an entirely new paradigm of understanding mental disorders.*⁴⁸

Just recently (October 2014), *Scientific American* contained an article, “Why We Need to Abandon the Disease-Model of Mental Health Care,” reading, in part, “We should not look to medication to ‘cure’ or even ‘manage’ non-existent underlying ‘illnesses.’”⁴⁹

Elsewhere in the article, the author stated, “We must move away from the disease model, which assumes that emotional distress is merely symptomatic of biological illness, and instead embrace a model of mental health and well-being that recognizes our essential shared humanity.”⁵⁰

Just the other day I received an email from a friend and colleague who believes much the same as I do concerning the origin of what we refer to as mental illness. He wrote:

I went to a lecture by one of these leading neuro-researchers from U.W. of Madison—neuroimaging, whereby, they have their own MRI, go to our prisons and scan “known psychopaths.” It is true their brain looks different, and less neurofibers between 2 regions of the brain; however, out of a room of... oh, I’d say 200 MD/PhDs/etc... I was the only one that stood up when he said it was a brain disorder caused by the shortage of these fibers, and said “I’m sorry, but could the difference be caused by these guys having horrible experiences growing up in non-perceived nurturing homes, and thus, what we are seeing is simply the aftermath of a person not having learned empathy, compassion, and emotional regulation? Isn’t that data you are showing just correlational data, and thus, you really can’t say it causes someone to be a psychopath?” The room went quiet, but to the lecturer’s credit, he agreed completely, apologized, then said he sometimes gets ahead of himself and does believe it to be causal, but has not been able to show that yet. The lecture went on.

Unfortunately, the whole biopsychiatric field of research has gotten ahead of itself but will *not* admit to it.

As a result of the lack of true pathophysiological evidence, many prominent psychiatrists have expressed doubts concerning a biological position for mental illness. A few years ago, Susan Kemker, M.D., wrote:

*The fact that I believed this dogma (that biology is the science of psychiatry) made Pam's (1990) critique of biological psychiatry especially unsettling. When I read his work, I felt that my entire education as a psychiatrist was subject to question. Some of the studies being scrutinized were known to me as major contributions to the field. I was shocked to find not a single "landmark" study emerging as methodologically sound.*⁵¹

Colin Ross, M.D., Clinical Associate Professor of Psychiatry at Southwest Medical Center in Dallas, Texas, from whom I earlier quoted, also wrote:

*When I entered my psychiatric residency, I believed that research had demonstrated the genetic foundation of schizophrenia and had shown that schizophrenia is primarily a biomedical brain disease. This view was almost universally accepted at my medical school, and I never heard serious criticism of it while in training. It was by a gradual process that I began to become more and more aware of the cognitive errors pervading clinical psychiatry—unwittingly demonstrated to me by my residency supervisors.*⁵²

In closing, my main hope in writing this paper is that each reader, regardless of his or her relationship to the field of mental health, will search for the truth as these two individuals have done. It is the only responsible path to take.

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