Brain Disease Hypothesis for Schizophrenia Disconfirmed by All Evidence

Al Siebert, PhD
Professional Development Center, Portland State University, Portland, OR

Prominent psychiatrists are stating that schizophrenia is a brain disease like Alzheimer’s, Parkinson’s, or multiple sclerosis. These statements are disconfirmed by scientific facts: no neurologist can independently confirm the presence or absence of schizophrenia with laboratory tests because the large majority of people diagnosed with schizophrenia show no neuropathological or biochemical abnormalities and a few people without any symptoms of schizophrenia have the same biophysiological abnormalities. People with schizophrenia do not usually progressively deteriorate: most improve over time. Psychotherapy and milieu therapy, without medications, have led even the most severely disturbed individuals with schizophrenia to full recovery and beyond. Many people diagnosed with schizophrenia have recovered on their own without any treatment, something never accomplished by a person with Parkinson’s, Alzheimer’s, or multiple sclerosis.

Los psiquiatras prominentes están declarando que la esquizofrenia es una enfermedad cerebral como Alzheimer, Parkinson o esclerosis múltiple. Estas declaraciones no son confirmadas por las pruebas científicas; ningún neurólogo puede confirmar independientemente la presencia o ausencia de un esquizofrénico por las pruebas de laboratorio, porque la gran mayoría de la gente diagnosticada con una esquizofrenia no demuestra ninguna anormalidad neuropsicológica o bioquímica, y algunas personas sin síntomas de esquizofrenia tienen las mismas anormalidades biofisiológicas. Las personas que tienen una esquizofrenia tienen las mismas anormalidades biofisiológicas. Las personas que tienen una esquizofrenia usualmente no se deterioran progresivamente; la mayoría se mejoran con el tiempo. La psicoterapia y terapia milieú, sin medicamentos, han llevado aun a los individuos más severamente disturbados por una esquizofrenia hasta la recuperación completa y más allá. Muchas personas diagnosticadas con una esquizofrenia se han recuperado solas sin ningún tratamiento, algo nunca logrado por una persona con Alzheimer, Parkinson o esclerosis múltiple.

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Il est fréquemment affirmé que la schizophrénie est une maladie du cerveau comme la maladie d'Alzheimer, la maladie de Parkinson, ou la sclérose en plaques. Ces affirmations sont néanmoins contredites par les faits scientifiques: aucun neurologue ne peut confirmer indépendamment la présence ou l'absence d'une schizophrénie au moyen d'un test de laboratoire car la grande majorité des gens avec le diagnostic de schizophrénie ne montrent aucune anormalité neuropathologique ou biochimique et quelques personnes sans symptômes cliniques ont le même profil biophysique. Habituellement, les individus avec une schizophrénie ne se détériorent pas progressivement: la plupart s'améliorent avec le temps. La psychothérapie et la thérapie de milieu, sans médicaments, ont conduit les patients les plus gravement perturbés à un rétablissement complet et parfois même à un bien-être psychologique supérieur que l'état pré-morbide. Finalement, plusieurs personnes avec le diagnostic de schizophrénie se sont rétablies sans aucun traitement — un résultat jamais observé chez des personnes atteintes de la maladie de Parkinson, d'Alzheimer, ou de la sclérose en plaques.

The shooting of two United States Capitol building guards in July 1998, by a man said to be suffering from “paranoid schizophrenia,” led to a flurry of media attention on “schizophrenia.” Unfortunately, prominent psychiatric experts used these media opportunities to state that schizophrenia is a brain disease like Alzheimer's disease, Parkinson's disease, or multiple sclerosis. These statements are inconsistent with published scientific facts and mislead the public.

For example, on the television show NewsHour (Farnsworth, 1998), broadcast on the Public Broadcasting System (and later on National Public Radio) in the United States on July 27, 1998, journalist Elizabeth Farnsworth interviewed Dr. Nancy Andreasen, director of the Mental Health Clinical Research Center at the University of Iowa and Editor-in-Chief of the American Journal of Psychiatry, and David Pickar, Chief of the Experimental Therapeutics Branch of the National Institute of Mental Health. Andreasen stated: “[Various] things add up to produce a brain injury that we then recognize as schizophrenia. . . . it's just about universally recognized that it's a brain disease like every, like all other brain diseases, Alzheimer's, Parkinson's disease and so on.” Pickar stated: “There's no question. This is a brain disorder.” E. Fuller Torrey (1983, 1997; Yolken & Torrey, 1995), another nationally prominent psychiatrist, has repeatedly stated that schizophrenia is in the same category as other brain disorders such as Parkinson’s disease, Alzheimer’s disease, and multiple sclerosis.

These statements distort and misrepresent research findings published in the scientific literature in eight significant ways.

(1) Schizophrenia is not “a single disease.” Starting with the original descriptions by Emil Kraepelin (1902) and Eugen Bleuler (1950) nearly 100 years ago, informed professional references describe “the schizophrenias” as a group of conditions. According to O'Donnell and Grace (1998): “a major problem in the study of schizophrenia is the diversity of its symptoms, leading to the suggestion that schizophrenia is actually a cluster of diseases” (p. 267). Statements to the effect that schizophrenia is “a brain disease” (singular) are misleading.
(2) Evidence for the brain disease hypothesis is weak. A few individuals diagnosed with schizophrenia show certain brain abnormalities, but the brain scans of most people diagnosed with schizophrenia fall within normal ranges (Weikert & Weinberger, 1998). Some psychiatrists, however, attribute undue significance to weak evidence (Boyle, 1990). For example, in a research study of childhood schizophrenia (Nopoulos, Giedd, Andreasen, & Rapoport, 1998), the authors concluded that "patients with extremely early onset (childhood) forms of schizophrenia may have more severe developmental anomalies than those with adult onset" (p. 1074). The data actually revealed that of the 24 patients studied, only 3 (12.5%) showed abnormal enlargement of certain brain structures, while 21 (87.5%) did not. In the healthy control group, one subject had a similar brain anomaly.

Ismail, Cantor-Grace, & McNeil (1998) found that when schizophrenic patients had certain neurological abnormalities, their siblings had very similar abnormalities, without clinical signs of schizophrenia. Andreasen (1995) found that a few people without symptoms of schizophrenia have brain abnormalities similar to those of some schizophrenic subjects. According to Lewine (1998), "there is no brain abnormality in schizophrenia that characterizes more than 20-33% of any given sample. The brains of the majority of individuals with schizophrenia are normal as far as researchers can tell at present" (p. 499). In addition, rarely do studies with positive findings control for the effects of prolonged use of neuroleptics and other drugs.

(3) The "brain disease" hypothesis cannot accommodate solid evidence that many people completely recover from schizophrenia. Unlike Parkinson's, Alzheimer's, and multiple sclerosis, schizophrenia does not necessarily progress toward lifelong debilitation, and the outcome for any one person is unpredictable (Marengo, 1994; Mendel, 1989; Möller & von Zerssen, 1988; Ponyat, 1992). Manfred Bleuler (1979a), author of the authoritative textbook on schizophrenia, wrote:

... the belief that schizophrenic psychoses are essentially a progression toward dementia and death ... is ... a tragic error. ... Nearly a third of schizophrenics recover for good. In general the psychosis does not progress more after five years from its outbreak but, rather, improves. ... These and other facts concerning the course and outcome of schizophrenic psychoses are certainly not characteristic of organic cerebral and metabolic disease. (p. 1407)

Indeed, longitudinal studies of thousands of ex-patients in many countries show that one-half to two-thirds of the individuals diagnosed as schizophrenic have achieved full recovery or significant improvement many years later. The percentages are:

(a) Bleuler (1968), Zurich study: 23% fully recovered, 43% significantly improved, 66% total.
(b) Huber, Gross, Schuttler, and Linz (1980), Bonn study: 26% fully recovered, 51% significantly improved, 57% total.
(c) World Health Organization (1979), worldwide 2-year follow-up: 26% very favorable, 25% favorable, 51% total.
(d) Ciompi (1980), Lausanne study: 29% fully recovered, 24% significantly improved, 53% total.
(e) Harding, Brooks, Ashikaga, Strauss, & Breier (1987), Vermont study: 34% fully recovered, 34% significantly improved, 32% total.
(f) Tsuang, Woolson, and Fleming (1979), Iowa study: 20% fully recovered, 26% significantly improved, 46% total.
(g) Hegarty, Baldessarini, Tohen, Waternanz, & Oopen (1994), meta-analysis of 320 outcome studies covering all countries, all decades, with 51,800 subjects 5-6 years after being diagnosed schizophrenic with broad criteria: 46.5% improved.
(h) Warner (1994), review of 85 outcome studies during 1956-1985: 20%-25% complete recovery, 40%-45% social recovery, 60%-70% total.
(i) Wiersma, Nienhuis, Sloff, and Giel (1998), 15 year follow-up of a Dutch cohort: 27% complete remission, 50% partial remission.

Many of the expatients in the studies listed above were evaluated 20 to 35 years after discharge. Those who recovered include expatients once viewed as the most profoundly disturbed. Courtenay Harding and her colleagues (1987) tracked down and evaluated 82 individuals who, 20 to 25 years before, had been the most hopeless, chronically disturbed, backward patients when discharged from a state hospital into a rehabilitation program. Harding emphasizes that “for one-half to two-thirds the long-term outcome was neither downward or marginal, but an evolution to various degrees of productivity, social involvement, wellness, and competent functioning” (p. 730). Many were found to be completely symptom free.

(4) No brain disease has ever been cured with psychotherapy or the passage of time. Many therapists have reported observing full recovery from schizophrenia with psychotherapy and/or milieu therapy (Artiss, 1962; Colbert, 1996; Fromm-Reichman, 1950; Harding, 1995; Jung, 1961; Karon, 1998; Laing, 1967; Mosher, 1999; Perry, 1974; Secheyeve, 1951; Siebert, in press; Sullivan, 1962). In the Soteria studies, young adults diagnosed as acutely schizophrenic were stabilized with no medication and nonprofessional helpers just as well and quickly as a similar group sent to a psychiatric hospital (Mosher & Menn, 1978). Many individuals diagnosed with schizophrenia have recovered on their own without medications or psychotherapy (Brody, 1952; French & Kasonin, 1941; Hoffman, 1985; Rubins, 1969).

In recent times the best known case of spontaneous recovery from schizophrenia is that of John Forbes Nash. In 1949, at the age of 21 Nash wrote a Ph.D. thesis that established him as a mathematical genius. Nine years later, Nash suffered a mental breakdown and was diagnosed with paranoid schizophrenia. His life and career were devastated. During the next 20 years he functioned marginally in the United States and Europe and was hospitalized many times for brief periods. Old friends, former colleagues, and admirers stayed in touch with him and continued to be kind to him. Then, for unknown reasons, Nash had a sudden remission. According to his ex-wife and sister, the two people who knew him best, his recovery was not due to any medications or psychological treatments (Nasar, 1998).

(5) Some people diagnosed with schizophrenia progress beyond recovery. A schizophrenic experience may in some cases have a beneficial effect on those
diagnosed, leading to favorable changes in personality and psychological growth (Arieti, 1979; Bernheim & Lewine, 1979; Bleuler, 1950; Bowers, 1979; Cancro, 1974; French & Kasonin, 1941; Jung, 1961; Menninger, 1963; Perry, 1974; Pickering, 1976; Rubins, 1969; Silverman, 1970; Sullivan, 1962; Warner, 1994). John Weir Perry (1999) reported that 85% of the clients (all met DSM criteria for schizophrenia and were "severely psychotic") treated at Diabasis, "not only improved, without medication, but most went on growing after leaving" (p. 147).

For some, a schizophrenic episode appears to function as a breakthrough to a higher level of mental and emotional functioning (Pickering, 1976; Sannella, 1981; Siebert, 1996). Silvano Arieti (1978) stated that "with many patients who receive intensive and prolonged psychotherapy, we reach levels of integration and self-fulfillment that are far superior to those prevailing before the patient was psychotic" (p. 20). Earlier, Arieti (1974) wrote: "some of my patients whom I consider cured have achieved important positions in life, in the academic world as well as in other activities" (pp. 616-617).

No one with Parkinson's, Alzheimer's, or multiple sclerosis is known to have fully recovered and developed a level of health and functioning superior to their pre-illness condition.

(6) The "cause" of schizophrenia is unknown (American Psychiatric Association, 1994; Gottesman, 1991). Andreasen stated during her interview (Farnsworth, 1998) that schizophrenia results from "multiple things—perhaps a genetic predisposition, nutritional factors early in life, viral infections, head injuries, exposure to toxins, exposure to drugs of various kinds, illicit drugs. All these things add up to produce a brain injury that we then recognize as schizophrenia." This statement is a guess, not a scientific fact. Given the encompassing list of risk factors, why do not more people exposed to them develop schizophrenia? Why do not more siblings of individuals diagnosed with schizophrenia—who have identical genetic predispositions and are exposed to the same neurological trauma—develop schizophrenia (Ismail, Cantor-Grace, & McNeil, 1998)?

If Andreasen's speculations were true, many people with similar "genetic predispositions" would eventually develop schizophrenia from the multiple neurological trauma caused by chronic smoking and drinking, cumulative environmental toxins, viral infections, poor nutrition, an aging brain, decreased immune system efficiency, and early stages of such brain diseases as Alzheimer's and Parkinson's. Proponents of the "brain disease" hypothesis cannot explain why schizophrenias occur so consistently in physically healthy young adults, aged 16 to 25, but rarely in anyone over 40, regardless of any physiological stressor (Arieti, 1979; M. Bleuler, 1979b; Hoffer & Osmond, 1966; Lewine, 1998; Poynter, 1992; Smith, 1982).

(7) There is no "universal" recognition that schizophrenia is a brain disease "like all other brain diseases." Medical textbooks and pathology journals do not include schizophrenia as a pathophysiological condition (Schaler, 1998). Medical specialties that deal with neuropathology and neurological diseases such as Parkinson's and multiple sclerosis, have nothing to say about the schizophrenias. None of the following neurology journals published articles on

Andreasen’s claim of almost “universal” recognition of schizophrenia as brain disease is also belied by statements from many psychiatrists and psychologists, clinically experienced with the schizophrenias, who see no convincing evidence for the theory (M. Bleuler, 1979a; Breggin, 1997; Karon, 1998; Lewine, 1998; Menninger, 1970).

Even the *Diagnostic and Statistical Manual* (fourth edition) of the American Psychiatric Association (1994), states plainly: “No laboratory findings have been identified that are diagnostic of schizophrenia” (p. 280). This statement highlights that the “brain disease” hypothesis stands or falls on simple criteria. A true brain disease must be identified and confirmed by laboratory tests. No blood chemistry, neurological, or brain scan test (or any other test) independently evaluated by a neurologist, biochemist, or pathologist who knows nothing about the patient’s clinical symptoms is able to reliably discriminate between a person experiencing a first episode of schizophrenia and someone who is not (Andreasen, 1987). However, such a test might well identify someone who has been taking neuroleptic medications for many years.

(8) “Treatments for schizophrenia are often worse than the “disease.” In the Farnsworth (1998) interview, Pickar stated that when people stop taking their medications “the consequences can be very severe.” What Pickar did not report, however, is that withdrawal symptoms can be disabling and mimic psychosis (Cohen, 1997) and that long-term drug use may be quite harmful. Neuroleptic medications may cause profound brain dysfunction and frequently lead to irreversible tardive dyskinesia, in up to 50% or more of long-term patients (Breggin 1997; Buckley, 1982; Cohen, 1997; Mosher, 1999; Rappaport, Hopkins, & Hall, 1978). This is solidly established fact in psychiatry.

Many people diagnosed as schizophrenic say they are helped by neuroleptic drugs. Pickar and other biological psychiatrists should inform the public that many people are seriously harmed by neuroleptic medications and that many people can recover and maintain a full recovery from a schizophrenia without medications.

**DISCUSSION**

As we have seen, some psychiatrists significantly distort and misrepresent research findings published in the psychiatric literature. They consistently downplay evidence that most people with schizophrenia do not have brain or biochemical abnormalities and that some people with similar abnormalities have no signs of schizophrenia. People with neuropathological diseases have
never been cured by psychotherapy and are rarely found decades later to be fully recovered.

Although some weak positive correlations have been found between the presence of schizophrenic symptoms and certain brain abnormalities, a basic scientific principle remains: correlation does not mean causation. In some instances, an underlying cause may lead to both brain abnormalities and schizophrenic symptoms. Some psychotherapists reporting cases of successful recovery from schizophrenia say that the symptoms often trace back to extremely traumatic childhood incidents that created powerful, conflicting feelings of loneliness and terror (Karon, 1998). What if some “schizophrenic” conditions turn out to stem from some form of chronic childhood traumatic stress disorder (Ford, 1998) that has persistent effects on brain structure or function?

The real “tragedy of schizophrenia” may be that thousands of people diagnosed with “schizophrenia” are led to believe that they have a chronic, debilitating, progressive brain disease like the incurable diseases of Alzheimer’s, Parkinson’s, and multiple sclerosis. For many, this amounts to hearing themselves sentenced to a slow, painful, early death. Yet no one ever dies of schizophrenia, even when it is untreated (Mendel, 1989). Is this erroneous and misleading information contributing to the high suicide rates of people diagnosed as schizophrenic?

Responsible, scientifically accurate statements to the media about schizophrenia might be expressed as follows:

A person diagnosed as having schizophrenia is expressing thoughts, feelings, and behaviors very disturbing to others and usually, but not necessarily, disturbing to the person expressing them. Research suggests that a few people diagnosed with schizophrenia have neurological complications, but many people with the same neurological profile do not develop schizophrenia. There is no known physical cause or medical cure for schizophrenia. Some people believe that they benefit from medications that control their undesirable symptoms, many people are harmed by the medications, and other people do better without medications. About 1 person in 10 never recovers from the original disturbed or disturbing experience and the effects of psychiatric treatment, but 5 or 6 out of 10 can be expected to fully recover or significantly improve. At present we cannot predict who will develop schizophrenia or why, who will recover or who will not. Further, we cannot explain why some people recover within weeks or months while others take from 5 to 20 years to recover.

REFERENCES


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Offprints. Requests for offprints should be directed to Al Siebert, PhD, P. O. Box 505, Portland, OR 97207.