The Persistence of the Belief that Madness is Hereditary

Gerald Schoenewolf PhD

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By Gerald Schoenewolf Ph.D.

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About the Author

Gerald Schoenewolf, Ph.D. is a New York State licensed psychologist who has practiced psychotherapy for over 33 years. He has authored 25 professional articles and 13 books on psychoanalysis and psychotherapy. His books include *101 Common Therapeutic Blunders, The Art of Hating,Counterresistance* and *Psychotherapy with People in the Arts*. He has also written and directed two feature films— *Therapy* and *Brooklyn Nights*. He lives in the Pennsylvania Poconos with his wife, Julia.

The Persistence of the Belief that Madness is Hereditary

For more than a century geneticists and social scientists have been trying to prove that mental illness is hereditary. Today most professionals and lay people believe that the hereditary nature of schizophrenia has been proven by accumulated evidence. However, a review of that evidence shows that such proof is far from conclusive. It is suggested, therefore, that the stubborn insistence on the genetic theory is itself the problem. A universal tendency to sweep under the rug the dark side of humanity leads to biased research.

Introduction

Recently news of a research project was leaked to all the major news services and television networks. The media quickly jumped to conclusions, blaring out headlines like, “New Genetic Link to Homosexuality Found.” Actually, a researcher in California, David Hamer, had discovered a possible link, but had not yet published his results; nor, of course, had anybody else replicated it. Later one of his assistants confessed to fraudulent methods and he was investigated by a government agency (Socarides, 1995). Yet, the popular media had announced this research as though it were a fact. This is an example of a human tendency with a long history—that of always wanting to attribute its problems to agencies outside of its control—the stars, inner demons, bad luck, the genes, etc. It is perhaps most prominent in our attitude

toward mental illness.

Schizophrenia is the most salient case in point. Today most mental health professionals, as well as most lay people, believe that madness is inherited, transmitted through genes. It is more than a belief; it is a strong contention that is held up almost as if it were a law, one that seems to defy any attempt to deal logically with it. Advocates of the genetic theory of schizophrenia most often cite twin studies as ultimate proof that schizophrenia is a thing of nature rather than nurture, and once having cited their evidence they invariably turn a deaf ear to any further discourse, as though one would have to be crazy (schizophrenic?) to dispute such anobvious fact. Their behavior is reminiscent of some religious fundamentalists. Try to argue logically with a Christian that Jesus could not really have been the son of God, and he will just shake his head and smile sympathetically at you. Try to argue logically with a schizophrenia-is-hereditary devotee and you will get a similar reaction.

Yet, a review of the research that purports to prove that schizophrenia is inherited or biochemical shows that such research is far from conclusive. Has the research been refuted? Has other research indicated other possible causes? If so, how can we explain this persistence in the genetic theory of schizophrenia and mental illness?

Genetic Transmission

Theories asserting that schizophrenia is inherited can be divided into two groups; those which focus on genetic transmission, and those based on the assumption of a neurophysiological dysfunction or biochemical imbalance. Sometimes the two overlap.

Researchers have been trying to find a genetic basis for schizophrenia since Charles Darwin (1859) emphasized the importance of heredity in the production of personality differences. The most often cited evidence for the genetic transmission of schizophrenia centers on twin studies. Early researchers would often come up with high concordance rates for monozygotic twins (up to 86 percent), while totally disavowing the influence of the environment. Recent researchers began speaking of an inherited predisposition to schizophrenia which interacted with environmental influence. Kolb (1977), summarizing concordance studies completed through the mid 1970s, found that concordance rates for monozygotic twins varied greatly from study to study, ranging anywhere from 14 to 86 percent, while those from dizygotic twins ranged from 0 to 22 percent. Geneticists claim that these concordance rates, found in many different societies at many different periods of time, demonstrating that twins separated from their mother at birth and raised in different environments can frequently both develop schizophrenia, show a definite genetic link. This genetic assumption is still maintained (Atkinson and Coia, 1995), and, in fact, recently a whole issue of *Nature* was devoted to it (Editor, 2010).

Lidz (1965) and Shean (1978), among others, have pointed out that monozygotic twins may have constitutional factors that predispose them to schizophrenia—they come from the same ovum and develop a unique identificational bond which results in a similar pattern of disturbed behavior throughout their lives. When one gets a cold, the other does, even if they are in different locales. In effect, twin studies may be interpreted as proving only that twins, due to their unique constitutions, are more susceptible to developing schizophrenia than nontwins. Hence, the data about monozygotic twins does not necessarily apply to the general population, nor does it even conclusively prove that twins have a genetic predisposition to schizophrenia. Other factors may be involved. For example, how traumatic is it for any infant to be separated from its mother at birth? And what about congenital factors? Does a schizophregenic mother transmit a schizophrenic tension state or biochemistry to her unborn child? In addition, studies have been conducted that failed to replicate this concordance.

One of the most stark studies ever done involved the Genain quadruplets. Several standard American textbooks on abnormal psychology carry, in their chapters on schizophrenia, a photo of the four identical smiling Genain women. They became famous in psychiatric circles because all four of them were diagnosed as schizophrenic. The name “Genain” is a pseudonym chosen by researchers, and comes from the Greek “dreadful gene.” Rosenthal (1963) provides a summary of the various researches that were done over the years.

Genetic researchers calculated the probability of all four quadruplets become schizophrenic as one and a half million to one; hence they concluded that there was “compelling evidence” for a genetic base for schizophrenia. However, such researchers ignored the abusive and bizarre behavior of both parents. From the case history we learn that both parents came from dysfunctional families. Mrs. Genain only married Mr. Genain when he threatened to kill her if she did not. After they were married they became virtual recluses.

Upon giving birth to the quadruplets, the Genain parents charged visitors 25 cents to see their daughters, but soon Mr. Genain became obsessively concerned about their safety, erected a fence, locked all the doors, and patrolled the yard with a gun. When the girls went to school, he did not allow them to mix with other children. The parents were so obsessed about masturbation that they had a surgeon remove the clitorises of two girls. Afterwards, the two were tied to their beds for a month, and they wet their beds twice a day and refused to eat. Only once were the girls ever allowed to go to a party, and then Mr. Genain stormed in halfway through and took them away from it. He allowed them to sing in a church, but made them quit when he found out the choir was taking a recreation break. The girls grew up extremely repressed and passive (see Johnstone, 1996). However, all these family factors have been discounted by geneticists.

In addition to twin studies, researchers have used family risk studies to

prove a genetic link to schizophrenia. Zerbin-Rudin (1972) studied the available literature and estimated risk figures of 9-l6 percent for children of schizophrenic parents, 8-14 percent for siblings, 1-4 percent for nieces and nephews, and 3-5 percent for parents. Meanwhile, the incidence of schizophrenia among the general population was estimated at 0.8 percent (Shields, 1967). Researchers assert that this evidence shows a clear blood relationship in the incidence of schizophrenia. Proponents of this theory also cite statistics showing that the incidence of schizophrenia is about the same in differing cultures. Critics argue that these researchers are biased, in that they are setting out to prove a genetic cause and are aware of the diagnosis of the index case from the outset. Second, they note that such statistics can just as easily be interpreted as evidence of a transmission of schizophrenic styles of child-rearing.

Adoption statistics are another area of study. These compare the incidence of schizophrenia in adopted children and their biological families. Kety and colleagues (1974) surveyed over 5,000 adults who had been adopted in early life. They found a 13.9 percent concordance rate between schizophrenic children and their biological families, while only 2.7 percent of adoptive relatives were schizophrenic. Such studies are flawed, Shean asserts, for several reasons. First, prenatal and early childhood environmental factors have not been accounted for. Not all adoptive children are taken from their mothers at birth; some remain with their mothers for weeks, months, even years. Second, most adoptive families know about the schizophrenic mother,

hence, they have negative expectations toward the child that may influence their rearing attitude. This research is therefore far from conclusive.

Genetic research in general has been plagued by problems of diagnostic unreliability. Schizophrenia is not an easily or absolutely identifiable characteristic, hence diagnostic ambiguities result in considerable variability among populations of schizophrenic patients. Tienari (1968) observed that different concordance rates (6-36 percent) could be obtained form the same subject population by applying different diagnostic criteria. Shean also notes that genetics is a very complex science, observing that "the number of genetically different sperm or ova that a single human can produce is…eight million" (1978, p. 103). It is difficult, if not impossible, to track down a recessive gene that might transmit a vaguely defined abnormal pattern of behavior such as schizophrenia. Coles (1982) points out three critical distinctions that must be made when considering genetic theories of etiology: between single gene and polygenic inheritance; between a genetically determined disorder and a genetically determined predisposition; and between inheritance and mutation. All of these factors are sources of endless debate and lead to the inconclusiveness of any research. In addition, after years of looking for the gene that causes schizophrenia, no gene has been found (Harrison and Owen, 2003).

Biochemical Theories

Biochemical theories assume that schizophrenia is caused by aberrant enzymatic or metabolic processes related to the neurotransmitters. Genetic and biochemical theories often seem to overlap, but biochemical defects do not necessarily stem from genetic sources. According to Frohman and Gottlieb (1973) biochemical studies have indicated inappropriate levels of plasma protein, indole animes and catecholamines, abnormal antibodies, disturbed hemolytic plasma factors, deviant carbohydrate metabolism, aberrant hormonal levels, abnormal levels of inorganic ions, and vitamin deficiencies.

Many investigators have claimed to prove that the dopaminergic synapses are involved in schizophrenic disorders (Shean, 1978). This theory comes from two sources. First, it was observed that amphetamines work on the brain's dopamine system to produce toxic amphetamine psychosis (the symptoms of which are similar to those of some forms of schizophrenia). Second, it was also observed that major tranquilizing drugs had what seemed like a curative effect on the topaminergic synapses (the symptoms of psychoses went away). From these observations and others, researchers began looking for biochemical causes of schizophrenia.

Others, such as Linus Pauling (1968) claim schizophrenia involves a vitamin deficiency, and prescribe large doses of niacin and vitamin C to cure it. However, Frohman and Gottlieb (1973) maintain that there is not enough data to show vitamins play any role in schizophrenia. Still others suggest a

“transmethylation hypothesis” (Osmond and Smythies, 1952). Pointing out the similarities of mescaline-induced experiences and schizophrenia, and between the chemical structure of mescaline and epinephrine, they concluded that schizophrenics undergo a transmethylation which turns norepinephrine to epinephrine which in turn affects behavior.

Again, none of this biochemical research is conclusive. Kety (1969) asserts five reasons why: (1) there is no evidence that the heterogenous forms of schizophrenia have a common etiology, so findings from one sample may not be confirmed by another; (2) biochemical research is conducted on patients with a long history of hospitalization in overcrowded institutions of low hygienic standards; (3) the quality and variety of the diet of institutionalized schizophrenics is different to that of control groups; (4) prolonged emotional stress, indolence, and lack of stimulation or exercise may alter many metabolic and physiological functions; (5) Exposure to radical therapies such as convulsive therapies and antipsychotic drugs may effect metabolic functions, even after therapy ceases.

Because biochemical theories seem so simple and so easy to verify, many researchers have been quick to assume that biochemical research is therefore more valid than, say, psychoanalytic research. However, none has been substantiated. “The highly publicized claims for the discovery of bio hemi al etiological agents,” Shean notes, “have not been confirmed when subjected

to rigorous scientific testing by independent investigators" (1978, p. 126).

The genetic researchers continue undaunted. As soon as critics refuse one of their claims, they conduct new research whose intent is to answer the critics. It becomes evident they are not looking for the truth (which should be the goal of all research), but are looking for a way to finally answer all their critics and to prove, finally and definitely, the genetic basis of schizophrenia. Each new claim of a discovery or breakthrough in understanding a genetic of biochemical basis for schizophrenia is immediately publicized and accepted as validated before it has been subjected to rigorous testing. Often, when such claims are later refuted, the refutations are given no publicity whatsoever, and the general impression lingers that the original claim was correct.

Environmental Research

Meanwhile little attention is paid by genetic researchers to the many detailed studies of the family environments of persons with schizophrenia. While giving lip service to these studies, the psychiatric establishment now states, as though it were a proven fact, that there is a genetic basis for schizophrenia and other mental diseases. Kolb, in Modern Clinical Psychiatry, citing the twin studies, asserts, "Over a half century of research into the genetics of schizophrenia has brought forward sufficient replicable evidence to leave little doubt of the existence of an inherited predisposition to the condition"

(1977, p. 380). Kolb, like others, does not take note of Lidz's observation about the constitutional factor of twins, or the other variables, such as prenatal conditions; if he did, he would have to amend his assertion to read that there is sufficient replicable evidence to leave little doubt of the existence of a susceptibility to the condition among monozygotic twins. That, and only that, has been undoubtedly proved. It has yet to be proven whether that susceptibility is genetic or congenital.

Environmental studies are now seen as passé; even if they have not been refuted by the scientific establishment, they have been pushed aside by the steamroller of obsessive empiricism. Yet, in fact, they have never been disproved. Mahler (1968), Lidz and colleagues (1965), Bateson and colleagues (1981), Wynne and colleagues (1981), Laing and Esterson (1964) and Piontelli (1992), are among the most prominent examiners of environmental derivatives. Their findings remain as striking today as when they were first conducted, offering detailed observations of dysfunctional relationships.

Mahler concentrated primarily on observations of mothers and children at the Masters Children's Center in New York. While not denying the possibility of an organic basis for autism in some children (due to brain damage or some other birth defect) she demonstrated how important a mother's eye contact and other nonverbal behavior toward an infant can be. She noted many cases of childhood autism in which the mother's hostility toward the infant, due to a

range of factors such as teen-aged pregnancy and postpartum depression, drove the child into a state of autistic withdrawal.

Lidz and colleagues conducted an extensive 12-year study of 17 schizophrenic families. These studies revealed characteristic patterns in the families of schizophrenics including (1) failure to form a nuclear family boundary because one or both parents remained primarily attached to family of origin; (2) marital schisms and lack of role reciprocity, or marital skews in which one partner yielded submissively to the domination and irrationality of the other; (3) failure to form a parental alliance and the blurring of generational boundaries between parents and children; (4)cognitive and communicational confusion, paranoid ideas, incestuous, and sex-role uncertainty; (5) failure to prepare children for separation from the family; (6) isolation of the family from the community; and (7) parental narcissism in which a parent failed to differentiate his/her own needs from the child. Lidz expanded on Fromm-Reichman's concept of the schizophrenic mother and added the concept of the schizophrenic father.

Bateson and colleagues coined the term, "double-bind," emphasizing how the schizophrenic mother puts the preschizophrenic into a double-bind. An example of this double-bind is the mother who verbally encourages her son to take initiative in school, yet when he attempts to leave home to visit a library, she entreats him not to leave her lest she become ill. Therefore, he is damned

if he does and damned if he does not. He becomes confused, builds up anger that he cannot resolve, does not develop a mature ego, nor mature socialization skills. And, as the mother is unaware of putting the son in a double bind, the son can never bring this fact up to her without being shamed by her and made to feel stupid. He then further doubts his perception of things and withdraws from a direct relationship with her and from others.

Wynne and colleagues studied family transactions through the medium of conjoint family therapy and showed how “pseudo-mutuality”—that is, denial—in schizophrenic family systems tends to engender schizophrenic thought disorder. Laing and Esterson found much the same thing in their studies of eleven families of schizophrenics in England. For example, when Laing and Esterson interviewed a schizophrenic patient and her parents, they observed the parents making faces at one another when their daughter spoke, but when the interviewers pointed out that the parents were making facial expressions, they both completely denied it. When a child is treated mockingly by parents, and then encounters their denial when she mentions it, she can only become frustrated and enraged as well as doubting her perception of reality.

Piontelli’s pioneering use of ultrasound to observe fetal behavior has verified the connection between prenatal environmental conditions and later personality development. These investigations began when she was analyzing an unusually restless 18-month-old toddler who was unable to sleep. Each day

this toddler moved restlessly about Piontelli's office, as though looking for something, searching every corner, behind every curtain, around every chair. Now and then he would shake objects, as if trying to bring them to life. When she mentioned this to his parents, they burst into tears and recalled that the boy had been, in fact, a twin. His twin brother had died two weeks before birth. "Jacob, therefore, had spent almost two weeks *in utero* with his dead and consequently unresponsive co-twin" (1991, p. 18).

This author has noted elsewhere that studies of multiple personalities offer additional proof that schizophrenia (or schizophrenia-like symptoms) are produced by environmental stress (Schoenewolf, 1991). In treating "Jennifer," it was noted that one of her seven personalities suffered from hallucinations, skewed thinking, paranoid projection, and motor disturbances. Her six other personalities might have been diagnosed as manic depressive, obsessive-compulsive, paranoid, impulsive-addictive, hysterical and schizoid. If seven different personalities, including one that is schizophrenic, can be produced in a single individual over the course of a childhood replete with harsh traumatic shocks (including sexual abuse before the age of three), the question of genetic susceptibility seem to become a secondary, if not moot, point.

Genetic researchers dismiss such studies as unscientific, unverifiable, and hence not to be taken seriously. Psychiatrists ask for more proof, as when Kolb (1977) commenting on Bateson's "double-bind," states, "What has not

been done to verify its usefulness in this and other psychological states is to define and reconstruct the specific learning contexts of the various clinical expressions. This requires definition of a precise connection between the initial paradox and the resulting pathology" (p. 388).

If it is not empirical, it is not to be trusted—so the scientists seem to say.And yet, as psychoanalysts have pointed out, matters of human behavior do not always submit themselves to empirical testing. Scientists assume that social science survey and observational studies are biased, lack proper controls, and are plagued by too many variables. Yet empirical studies are just as often biased and unreliable, even though they are wrapped in the language and trappings of science.

Human Narcissistic Disorder

In the litany of a Church service certain phrases are repeated over and over, such as, "Jesus, the son of God," so that the accumulated effect is to make the believer come to accept that such a concept is beyond doubt. It is, in essence, a form of hypnosis. Much the same thing has happened with respect to genetic theories of schizophrenia and other diseases. They are repeated over and over, in textbooks, on television, on the internet, in newspapers and periodicals. Most people today, like Kolb, are convinced of an inherited predisposition to schizophrenia, despite the fact that there has not been any

conclusive proof of it. In fact, incredibly, almost the entire psychiatric establishment and much of the mental health field has come to accept the genetic theory as a proven fact, when the only thing that has actually been proven is that monozygotic twins have a susceptibility to schizophrenia.

On the other hand, there is much evidence that schizophrenia is produced by the environment. It seems apparent that if an environment is schizophrenic, it will produce schizophrenia whether or not an infant is predisposed to schizophrenia. Can you imagine an infant confronted with the denial, projective identification, hostility, and double-binding behavior of an undiagnosed borderline or schizophrenic mother or father, looking up at the caretaker and saying, “Oh, no, you’re not going to drive me crazy, because I’m not predisposed to schizophrenia!” Indeed, one can safely say, after considering all existing research, that while constitutional factors may play a part, as they do in all emotional disturbances, the environmental factor is by far the more crucial one. Having said that, do you suppose the media will immediately pick up on this assertion and lavish us with headlines like, “Psychoanalyst finds that disturbed environments cause schizophrenia”? Very unlikely. More likely the notion that families cause schizophrenia would be dismissed as dated, mother-bashing, parent-bashing, family bashing, and an insult to humanity.

To understand this stubborn refusal to look objectively at this question, I will introduce another concept. To get the deepest grasp of why there is this

persistence in the belief in a hereditary wellspring of madness (as well as of other mental disorders), one must put aside the evidence and arguments and look instead at the process. Namely, there seems to be a phenomenon that has been visible throughout humankind's brief history. It has been given various names over the years, but for the purposes of this paper I will call it the Human Narcissistic Disorder (HND). In fact, let's give it a code number, so as to make it more palpable to the scientific establishment and so it will fit into the disorders of the DSM classification of mental disorders: HND 1000.01. Like individual narcissism, this cultural narcissism has at its core a grandiose denial ("Families are good and incapable of driving children mad") and an underlying rage ("…and don't try to tell me anything different!"). Grandiosity asserts that we are irrevocably good and defends against the reality of our the darker human side, which causes disturbances in our children. Any challenge to this defense results in a enraged attack on the person or group that makes the challenge.

Human Narcissistic Disorder has surfaced throughout history, whenever some new finding has proved to be a blow to the narcissistic grandiosity of humanity and that finding has been met with disbelief and scorn. The discoveries that the world is round, not flat; that the earth revolves around the sun; that our solar system is but one of many in the universe; that humans are evolved from lower animals; and that we in fact do not have free will but are genetically programmed and environmentally conditioned to believe and think

the way we do—all these discoveries were met with rage and scorn (and still are by many people). Any discovery that bursts this bubble of humanity's grandiosity, of its sense of importance, righteousness, innocence, omnipotence, omniscience, or well-being, is resisted, sometimes mightily.

Humanity's resistance to the idea that the environment (parents, families) engenders schizophrenia is one of the latest aspect of HND. Basically, it is a desire to sweep unpleasantness under the rug, to deny "man's cruelty to man." From the beginning of recorded history children have been encouraged to hide their problems from others and often even from themselves, to keep their "family skeletons in the closet." Commandments of most religions exhort children to "honor your father and mother." Just as individuals are encouraged to repress painful thoughts and memories and remain unconscious of their links with present behavior, so also societies repress and remain unconscious of the cruelties of families.

Fromm (1990) was one of the first to consider whether an entire culture could be diagnosed as insane. Just as people can be schizophrenic, so can a society. Fromm talks about a society in which people escape into over-conformity and the danger of robotism in contemporary industrial society. In the present context, one might point to the conformity and robotism of people who persist in promoting the genetic view of schizophrenia and resist being aware of their psychological cruelty to others. A society comprised of families in which

members are taught to deny, project, and displace anger (scapegoat a member of the family and putting the runt into a double bind), is a society in which genetic theories of the etiology of schizophrenia will abound. Indeed, such a society might not just prefer a genetic theory, it might insist on it, and would use public opinion as a force to shame and ridicule anybody who would dare to think otherwise (just as a parent will often shame and ridicule a child for daring to doubt the parent's love or good will).

Schizophrenia is humanity's darkest secret, one that we want desperately to keep out of sight because it reminds us of our own complicity in the matter. The unconscious desire to drive other people crazy, of which Searles (1959) has written, is a desire that hardly any of us wish to acknowledge, particularly those who are successful in doing so. This tendency to drive others crazy, primarily found in parent-child relationship but also prevalent in marital relationships, as depicted in the film, *Gaslight,* wherein a husband tries to drive his young wife crazy, may well be an inherent tendency, a survival mechanism. If a human is under stress, he or she will either sink under or try to push somebody else under in order to save himself or herself. Madness might be seen as a state of "being under," a withdrawal form active life and direct and meaningful communication in order to avoid the threat of being pushed under.

Searles (1959) found that the "effort to drive the other person crazy" was one factor regularly found in the cases he treated. "My clinical experience has

indicated that the individual becomes schizophrenic partly by reason of a long-continued effort, a largely or wholly unconscious effort, on the part of some person or persons highly important in his upbringing, to drive him crazy" (p. 254). Searles saw this effort to drive another person crazy as a way of psychologically murdering the other without having to take responsibility for it, a way of externalizing one's own threatening craziness, and of regaining a sense of omnipotence and control over others. He believed this unconscious desire to drive another crazy extended to psychiatry, referring to "so many of us who show a persistent readiness to regard this or that kind of functional psychiatric illness, or this or that particular patient, as incurable—in the face of, by now, convincingly abundant clinical evidence to the contrary." According to Searles, this attitude may mask "an unconscious investment in keeping these particular patients fixed in their illnesses" (p.279). Searles' notion of people unconsciously wanting to drive other people crazy can be seen as yet another aspect of HND.

Summary

The persistence in the belief that madness is hereditary is a relentless, obsessive drive to accumulate and prove theories that have never been validated by research. The stubbornness and often vehemence of the persistence may be indicative of a disorder. It has led to the treatment of schizophrenia with medications geared to maintaining the illness rather than

curing it. It has led to centuries of biased research and skewed results. A tower of evidence proves nothing if that evidence has been misguided.

It would probably be much better for society if researchers spent their time looking into the causes of HND 1000.01. If we could find a cure for that, we could probably find a cure for everything else. Indeed, HND 1000.01 may be the "virus" that engenders schizophrenia. "The fault, dear Brutus, is not in our stars," Says Shakespeare's Julius Caesar, "But in ourselves, that we are underlings."

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