

# **BIOGENETIC AND ENVIORNMENTAL ORIGINS OF MENTAL ILLNESSES**

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### Abstract

**Problems:** Many researchers believe that Post Traumatic Stress Disorders (PTSD), Conduct Disorders (CD), and Attention Deficit-Hyperactivity Disorder (ADHD) are biological in origin.

a) What is your view on this matter?

b) With a knowledge of nature/nurture controversy, what are the theories associated with the biological and genetic causes of schizophrenia?

The challenge of applied or clinical psychology is to inclusively extend the horizon of psychopathology in which psychobiological factors are explored as a reliable step toward understanding the etiological factors behind mental disorders. The variation observed in individual differences in normal and psychopathological behaviors have raised an array of controversies among researchers. The disease model theorists hold genetic factors as a major contributor at the most distal end of a complex genetic-to-behavior pathway. The fundamental behaviorists, on the other hand, believe that if separated from environmental factors, the whole search for the origin of mental disorder is stunted and meaningless. Therefore, the interface between biogenetic and environmental factors in the etiologies of mental disorder remains controversial. The middle way or the prototype for a complex systems approach to psychopathology is the integrative approach that investigates the sequential and concurrent roles of biogenetics, environment, chance, and time factors in the causation of such mental disorders like, Post Traumatic Stress Disorders (PTSD), Conduct Disorders (CD), Attention Deficit-Hyperactivity (ADHD), and Schizophrenia.

## **Introduction**

### **The opinions of researchers as to the causes of Post Traumatic Stress Disorders, Conduct Disorders, Attention Deficit-Hyperactivity Disorder and schizophrenia?**

Attempts to discover a generalized criterion for defining and identifying mental pathologies has been the major problem in the field of clinical psychology. In the medical profession, definition and identification are achieved by the knowledge of what constitutes normal anatomy and physiological functioning (Adams, Luscher, & Bernat, 2001). Invariably, a successful diagnosis of mental pathology must depend on a classified scheme that examines the normative data of human behavior in order to empirically isolate those behaviors that militate against proper and meaningful functioning. Such procedure is otherwise referred to as psyche (mental) pathology (disorder, illness).

Psychopathology is a branch of clinical psychology that studies abnormal behaviors with a focus on their classification, categories, nomenclatures and scientific understanding of their causal factors (Adams, Luscher, & Bernat, 2001). Mental disorders studied under these entities embrace a multidimensional behavior component that directly or indirectly influences an individual's mental and physical functions. The pathways through which behavior disorders have been studied are reactivity (the property of being responsive or precipitated by an external cause), temperament, body dysregulation, personality, plasticity, learning and memory, and sensitization.

As the field of psychology conspicuously shifts toward a medical model, biopsychology modalities have been used to explain the etiology of many behavior disorders (Fowles, 2001; Rhee, et al., 2001; Sutker & Allain, 2001). Today, subjects like neuropsychology (effects of the brain and nervous system on behavior and mental experience), psychoendocrinology (effects of hormones on human behavior and mental experience), psychopharmacology (effect of drugs on behavior), and psychophysiology (correlation between the mind and the body) have become household names in the field of clinical psychology (Garraghty, Churchill, & Banks, 1998).

Generally, biogenetic and environmental models represent useful and complementary alternatives to investigating etiological factors in mental disorders, because they fundamentally aim at elucidating human brain structure and its effects on an individual's daily functions in a given situation (Garraghty, et al, 1998; Kandel, 1998). Any approach that compromises the natural characteristics visible in a family-offspring relationship is bound to confuse the genetic and environmental influences that are necessary in understanding the causes and origin of a mental disorder (Gottesman, 2001). On the other hand, comparative studies of human biology and psychology will illuminate the hazes present in psychopathology (Flowes, 2001; Gottesman, 2001).

The popular method used in studying the etiology of mental disorders is aimed at discovering biogenetic and environmental influences in mental disorders and was developed from clinically oriented studies of adopted children and twins (Rhee, Feigon, Bar, Hadeishi, & Waldman, 2001). These studies revealed that at the root of most

psychopathological cases, there is an eminent probability of biogenetic and environmental influences (Fowles, 2001; Rhee, et al., 2001). Furthermore, the finding predicted significant continuum from Axis II (e.g., schizoaffective) to Axis I disorders (e.g., schizophrenia) (Fowles, 2001; Kendell, 1982; Rhee, et al., 2001; Sutker & Allain, 2001). The continuum has been attributed to a specific genetic liability rather than environmental factors. Although biogenetic continuum from Post Traumatic Stress Disorders (PTSD), Conduct Disorders (CD) or Oppositional Defiant Disorder (ODD), and Attention Deficit-Hyperactivity Disorder (ADHD) to schizophrenia has not been scientifically verified (Fairbank, Ebert, Caddell, 2001; Sutker & Allain, 2001), it does not undermine specific clinical evidences that support genetic- environment factors (Sutker & Allain, 2001). Actually, reliable scientific evidences suggest that genetic-environmental components significantly constitute the basic features of most major mental disorders (Thapar & McGuffin, 1996; Silberg, Rutter, Meyer, Mace, Hewitt, Simonoff, Pickles, Loeber & Eaves, 1996; Rhee, et. al, 2001). In the past, researchers have used methodological procedures that included the studies of different family data, parent-offspring concordance or correlations, adoption records, and twin studies to infer genetic and environmental influence etiological symptoms.

In contemporary behavior studies, twin data analyses have become the design of choice for drawing inferences regarding genetic and environmental influence on individual mental health history (Rhee, et. al., 2001). Investigation on the part that gene and environment plays in severe behavior disorders has attracted sumptuous attention among scientists, leading to attempts to verify whether certain disorders usually diagnosed in

infants, children, and adolescents continue or progress into adult disorders (example, schizophrenia or mood or anxiety disorders). The effects of environment on the brain structure, family genetic vulnerability, and related mental problems among adopted children are today integrated and effectively studied under psychology and biology. . Such infusion has broadened the etiological knowledge of mental disorders and aid in the quest to understand the origin and the hideous patterns in mental disorders (Fowles, 2001).

### **Posttraumatic Stress Disorder (PTSD)**

Posttraumatic Stress Disorder (PTSD) is an anxiety disorder arising as a delayed and protracted response after experiencing or witnessing a traumatic event involving actual or threatened death or serious injury to self or others (Colman 2001; Diagnostic and Statistical Manual of Mental Disorders-third edition, 1980 (DSM-III); DSM-IV; Fairbank, Ebert, & Caddell, 2001). The kind of event that seems particularly likely to produce subsequent stress and anxiety disorders is one that is usually associated with intensely stressful experiences that involve exposure to levels of danger and fear that exceed normal coping capacity of the individual (Brett, 1993; Fairbank, et al., 2001; Rosenzweig, Leiman, Breedlove, 1999). For example, in the DSM-III (1980) and DSM-III-R (American Psychological Association (APA), 1980/1987) traumatic event is defined as an event outside the coping range of usual human experience, which invokes significant symptoms of serious psychological distress and “may even involve direct damage to central nervous system” (DSM-III-R, p.248). Memories of intense and horrible events intrude into consciousness and produce intense visceral actions (arousal in the

somatic nerves or viscera produced by epinephrine following fear and trembling) that the original event caused (Rosenzweig, et al., 1999). These traumatic memories are easily awakened by harmless stressful circumstances akin to the previous experience. Even though PTSD is prevalent among those who served and lived in an intense combat areas, studies show that it can readily be associated with persons who either witnessed or were themselves involved in abusive and violent relationships, brutal murder, actual or threatened death or serious injury to self, sudden loss of a close friend, torture, kidnapping, or forced immigration that created intense fear, helplessness, and horror (Fairband et al., 2001; Bremner, Southwick, & Charney, 1999). Recent studies support the claim that there is interaction of biogenetic and environmental factors in the cause of PTSD. In twin studies, researchers maintained that the specific contribution of inheritance to PTSD accounts for one-third of the variance of PTSD (Fairband et al., 2001; Rosenzweig, et al., 1999).

The psychobiological origins of PTSD can be linked to neural networks that control fear, loss (extinction), and behavioral sensitization, which further suggests that the amygdala and some brain pathway that controls fight/ flight response behavior are directly connected to PTSD. Exposure to intense stress leads to high levels of circulating glucocorticoids, which might lead to cell loss in the hippocampus or might prevent normal cell gain in the hippocampus. This is why in many cases of PTSD, victims (patients) show sign of amnesia, flashback, and deficits in short-term memory (Rosenzweig, et al., 1999).

### **Conduct Disorders (CD)**

Conduct Disorder (CD) is a disruptive behavior associated with antisocial and aggressive behaviors mostly diagnosed among young children and adolescents, although it may be

diagnosed in individuals who are older than age 18 . In most part, Conduct Disorder (CD), Oppositional Defiant Disorder (ODD), and Antisocial Disorder (ASD) have significant correlated communality in their features and characteristics, however, the DSM-IV made a sharp distinction between Oppositional Defiant Disorder (ODD) and Conduct Disorder (DSM-IV). Many investigators believe that CD is basically caused by shared and nonshared environmental factors which include high rates of parental psychopathology, especially substance abuse and antisocial disorder, divorce and marital conflicts, and dysfunctional parenting practice (Frick & Silverthorn, 2001; Rhee et al., 2001; van den Oord, Verhulst, & Boomsma, 1996; Waldman et al., 1998). In their reports, Lyons and colleagues observed a low magnitude of genetic influences in ODD and DC, whereas, Slutske, Heath, Dinwiddie, Madden, Bucholz, Dunne, Statham, & Martin found no evidence for shared environmental influences on retrospective reports of CD symptoms.

Additionally, some researchers believe that a number of neurological vulnerabilities play major roles in the etiology of CD because it has been discovered that very aggressive and antisocial children have lower levels of serotonin, epinephrine, and high levels of testosterone (Frick & Silverthorn, 2001). The unique and irregular temperamental features and impulse control found in children with CD can be linked to abnormalities in the sympathetic arm of the automatic nervous system (Frick & Silverthorn, 2001).

### **Attention Deficit-Hyperactivity Disorder**

In the DSM-IV (2000), it is maintained that Attention Deficit-Hyperactivity Disorder (ADHD) is essentially and predominantly manifested in children with significant and persistent features of inattention and/or hyperactivity-impulsivity symptoms capable of

causing remarkable impairment in two or more social settings. The principal characteristics of ADHD include persistent inattention, hyperactivity, and impulsivity, with some of the signs and symptoms appearing before age 7. Significant and serious social, academic, and occupational problems found in most children and adolescents can be attributed to this disorder. Researchers estimate that about ten per cent of young school age children are affected by this disorder, of which boys are astronomically over-represented (Colman, 2001).

Many researchers have identified significantly more biogenetic influences in the etiology of ADHD than have been recorded in CD, and ODD (Frick & Silverthorn, 2001; Rhee et al., 2001). Developmental failure in the area of the brain is responsible for inhibition and self-control. The parts of the brain that may be affected include the caudate nucleus, the globus pallidus, the prefrontal cortex, and the vermis of the cerebellum areas that use dopamine to communicate with others. Problems in the *executive* region of the brain have been suggested to be at the core of the origin of most serious mental disorders (Colman, 2001; Frick & Silverthorn, 2001). Furthermore, neurochemical agents, like catecholamines, have also been linked to the root of ADHD (Frick & Silverthorn, 2001). In most part, children of parents with Mood and Anxiety Disorders, Learning Disorders, Substance-Related Disorders, and Antisocial Personality Disorders are vulnerable to ADHD (DSM-IV, 2000).

### **Theories Associated with Biogenetics of Schizophrenia**

Comparatively, schizophrenia may not be the most severe of all disorders with psychotic features, but it is the most complicated because of its distinct constellation of signs and symptoms (APA, 2001). The DSM-IV's presentation of the symptoms of schizophrenia as involving "a range of cognitive and emotional dysfunctions which include perception, inferential thinking, language and communication, behavioral monitoring, affect, fluency and productivity of thought and speech, hedonic capacity, volition and drive, and attention" (p. 299), has been variously explored by researchers to buttress their theories of psychobiogenetic origin of schizophrenia (Frick & Silverthorn, 2001; Kolb & Wishaw, 1996; Rosenzweig, et al., 1999; Schneider & Tarshis, 1986). The various categories that characterize the symptoms and signs used in the diagnostic evaluation of schizophrenia include both positive and negative symptoms (Frick & Silverthorn, 2001).

As a matter of fact, many scientists believe that the biogenetic origin of schizophrenia can be well explained from its negative symptoms (delusion, hallucination, and disorganized thinking), while ecological vulnerability or ecovulnerability can be accounted for by psychology and environment (APA, 2000; Frick & Silverthorn, 2001; Rosenzweig, et al., 1999). Even though a complete remission (or a return to full premorbid functioning) for schizophrenia is presently unattainable, its positive symptoms can successfully respond to treatment whereas "in many individuals, negative symptoms persist between episodes of positive symptoms" (APA, 2000, p. 309).

### **Genetic Impression in Schizophrenia**

In order to buttress their biogenetic conceptualization of schizophrenia, many researchers believe that the difference between schizophrenia and diabetes is that schizophrenia center in the very part of the body that controls behavior-the brain. Hedaya (1996) believes that just as diabetes is genetically transferable, schizophrenia also can be genetically transmitted from generation to generation (Hedaya, 1996, see p.123). The behavioral geneticists basically supported a statistical proposition that schizophrenia is fundamentally linked to a trait that is attributable to genetic variation within a group (Gottesman, 2001). This is because researchers found over representation of schizophrenia among children of schizophrenic parents, even adopted children or those in foster homes have shown high rates of the disorder. Secondly, the studies of schizophrenia among twins suggested a significant correlation between identical twins rather than fraternal twins.

The most explicit studies made toward the genetic theory are studies of monozygotic (MZ) twins and the dizygotic (DZ) twins. Kendler vehemently believed that genes account for approximately 60-70% of the variance in transmission of schizophrenia. His claims, however, are based on his variety of studies on more than 800 MZ and 1000 DZ twins. In these studies, it was discovered that paranoid and schizotypal personality disorders were higher in relatives of schizophrenics. Apparently, it is hard to accept the validity of this claim if it is not scientifically linked with the presupposition that a gene that directs the production of the dopamine receptor (D2) is associated with schizophrenic episodes (Nathan & Gorman, 1998). If a child has a schizophrenic genetic trait, prenatal

complications, nutritional problem, and lags maturational development (Gottesman, Aston, and Moldin, 1999), he or she stands a greater chance of becoming schizophrenic than the sibling who had normal development processes. Since monozygotic twins share equally in these areas of human development, their affectability should be counted as developmental factors rather than inheritability (Thapar, Poulton, & Harrington, 1999).

### **Genetic analysis of dopamine receptor gene**

While family, twin, and adoption studies suggest that genetic factors play a major role in the etiology of schizophrenia, the mode of genetic transmission remains clouded and uncertain. More than ever, research has shifted to the metabolic actions of the synaptic mechanisms which recognize the presence and the possibilities of chemical imbalances in metabolic processes, hormonal distributions, drugs, and their affects in the psychological function they mediate. Kety and his colleagues believe that the psychological processes, mediated through biochemical reactions of the synapses, control the individual's power of perception, cognition, attention, motivation, mood, and other emotional and mental states. Impairment synaptic metabolism may affect emotional ability and accounts for variation and irregularity in general human cognitive ability.

Undoubtedly, the complex traits of schizophrenia are represented by more than one locus of mal-developed human genome. Rather, investigators that focus on DNA, gene-mapping technique, and the dopaminergic system may have better etiological explanations for schizophrenia (Gottesman, 2001). All genetic variations originate from a change in the DNA sequence called mutation. A large number of agents in our environment are known to cause mutations, which include ionizing radiation and many

other different chemical reactions. However, mutations can occur spontaneously during the process of DNA replication. The differing DNA sequences of a gene are called “alleles”. If an individual has the same alleles on both members of a chromosome pair (from both parents), he or she is said to be a homozygote. Difference in alleles of DNA sequence, however, means that the individual is heterozygote. It is known that many of the different physical characteristics, such as height, color of skin and eyes, are determined by genetic variations. Some of the genetic variations are inconsequential while others cause diseases (Gottesman, Aston, and Moldin, 1999).

A number of laboratory techniques have been employed to determine gene variation that causes schizophrenia. For example, protein electrophoresis, which recognizes differences in proteins on the basis of their electrical charges, has been used to determine amino acid variation in human beings. This method has been rejected because it cannot detect all the variations in the amino acid sequences. A new deoxyribonucleic acid (DNA) based research method, which detects the amino acid variations at the DNA level has been recognized by many geneticists. This technique, known as Restriction Fragment Length Polymorphism (RFLP), is developed from the availability of large numbers of bacterial enzymes which cleave or cut DNA at the specific recognized sites known as restriction or recognized sites (Asherson, Curran, McGuffin, 1999)

Using this technique, many studies have been made of the schizophrenic-gene replication. Bruce Bower reported investigation of more than 100 families that suggest a gene located on the chromosome 13, which contributes to at least some cases of

schizophrenia that usually appears in younger ages. A specific sequence on chromosome 8 also shows signs of boosting susceptibility to schizophrenia for some people who possess the signature sequence on chromosome 13. The genetic epidemiologist, Ann Pulver, maintains that what's exciting is that we have the first evidence to support the theory that different sets of genes can create a susceptibility to schizophrenia. Earlier efforts to locate susceptibility of schizophrenia have had mixed results. Some evidence indicates that an unidentified gene on chromosome 6 contributes to the disorganized thinking, delusions, and hallucination typical of this mental ailment.

In order to support their views, researchers have used the DNA blood samples of those diagnosed with schizophrenia and the members of their extended families. The studies maintained that a genetic link to chromosome 13, in 8 areas was found in both individuals diagnosed with schizophrenia and in their families. A genetic link only to chromosome 13 characterized the families in which several members had mood disorder, as well as hallucinations and delusions. In a similar research, Sevilla Detera-Wadleigh of the National Institute of Mental Health in Bethesda, Maryland are believed to have often found the same chromosome 13 sequences in family members of individuals diagnosed with bipolar disorder, or manic depression. This finding argues that if heritability is composed of DNA traits, the gene on chromosome 13 can increase the likelihood of schizophrenia in offspring of those suffering the ailment. This finding has been challenged because of its lack of precision and limited access to other areas of great importance in determining heritability.

Molecular biologists have resorted to the technique referred to as “gene mapping” to study genes-chromosome interplay and heritability. These techniques are used widely in the study of central nervous system (CNS) structure and function. Methods of research permit investigation of different proteins, step by step, from gene transcription to the post-transcriptional processes. These techniques are also being applied in the investigation of enzymes responsible for the synthesis of neurotransmitters and receptors. Furthermore, Seaman et al (1994) studied the dopamine D4 receptor gene in a group of schizophrenics and control subjects. These studies are carried out in order to determine the frequency of the dopamine receptor gene variants in which serine or glycine substitutions have occurred. When administered with glycine substitutions, 23 out 183 controls, representing 12.6% of the control population, and three out of 24 schizophrenics, representing 12.3% of those tested, revealed a replacement of thiamine by guanine. The identical prevalence of this variant between two groups indicates that the variant is not associated with schizophrenia. This variant, however, was found only in black subjects, but none of the 147 Caucasians (113 control and 34 schizophrenics) revealed this variant.

Concentrations of serine and glycine in the cerebrospinal fluid (CSF) and plasma have shown some evidence of the genetic control of these amino acid variations. Cerebrospinal fluid levels of several amino acids showed a similar pattern in a twin sample. Devor and Waziri reported significant genetic control of plasma concentrations of serine and glycine in 28 nuclear families with 108 members. The authors suggested that the control of the plasma concentration was carried out via a single major gene locus and the glycine metabolizing enzymes, SHMT, was a likely candidate for this single gene locus.

**The Biochemical Activities of Dopamine.**

Dopamine is one of the neurotransmitters. Neurotransmitters are electrochemical substances released from pre-synaptic axonal terminals into the post-synaptic receptor. The neurotransmitters that will occupy us here are the catecholamines that are released in form of epinephrine, norepinephrine, and dopamine. The Calcium ion plays an important role in the release and responses of the transmitters. Irregularities in the calcium ion may delay the arrival of action potential and the opening of the  $Ca^{++}$  gateway to allow the entry of the  $Ca^{++}$  into the pre-synaptic terminal. Neurotransmitters are also receptors or post-synaptic agents, and their cells have the ability to recognize neurotransmitter, neuropeptide, hormones, and other chemical substances.

Investigators have identified several subtypes of DA receptors that are categorized as D<sub>1</sub>, D<sub>2</sub>, D<sub>3</sub>, D<sub>4</sub>, and D<sub>5</sub>. This categorization follows the order of their discovery (Rosenzweig et al., 1999). With the exception of D<sub>1</sub> and D<sub>5</sub>, all other DA receptors, D<sub>2</sub>, D<sub>3</sub>, and D<sub>4</sub> share the same properties and are similar to each other. However, the stimulation of D<sub>1</sub> receptors causes an increase in cAMP levels, whereas stimulation of D<sub>2</sub> receptors decreases the cAMP. There are also autoreceptors on the presynaptic terminal, which are thought to exert an inhibitory feedback effect on the DA neuron.

Dopaminergic synapses have been of great interest because of the evidence that antipsychotic drugs that are effective in alleviating the symptoms of schizophrenia have the common effect of interfering with transmission at the dopaminergic synapse in the brain (Rosenzweig et al., 1999).

Jung and Schmauss (1999) found that, in mice targeted, disruption of dopamine D2, D3, and D2/3 receptors substantially impairs agonist-promoted dopamine D-1 receptor activity in brain region where dopamine is known to influence behavior that are often disrupted in schizophrenia. Dopamine acts on two types of receptors, both of which are linked with cyclic adenosine monophosphate (cAMP) and regulates reactions generated by hormones and neurotransmitters (Crowe, 1995).

Recent research studies indicate that the primary mode of operation of antipsychotic drugs is to bind to, and to block, the D2 receptors. In contrast to these blocking actions of antipsychotic drugs, it is known that drugs that are DA agonists such as amphetamine, that enhance or mimic the actions of DA, can induce schizophrenia-like behavior. It has been tempting therefore to believe that schizophrenia is due to hyperactivity of the DA neuron in the brain. But how this hyperactivity of the DA can be transmitted to a generation of siblings is yet to be cleared (Schneider & Tarshis, 1986).

Molecular mechanisms, activities, and metabolism of the dopaminergic synapses can be explained as follows: The synthesis of the enzymatic pathway starts with the action of tyrosine (Tyr). Tyr is catalyzed and converted to dihydroxyphenylalanine (DOPA). The DOPA is transported and stored within the neural network as dopamine (DA). Storage of DA is blocked and inhibited by the actions of reserpine (Res). DA is released by the actions of exocytosis. During the onward transmission from pre-synapses to receptors or post-synapses, the DA binds itself to the D<sub>1</sub> receptor, acting through stimulatory G protein (G<sub>2</sub>) to increase the level of cAMP, or binds with the D<sub>2</sub> receptor, acting through

inhibitory G protein to lower levels of cAMP. Most of the antipsychotic medications/drugs, such as butyrophenones, block the action of the D<sub>2</sub> receptor.

### **Conclusion**

Among clinical psychologists, the awareness and acceptance of biological modality theory in psychopathology have drastically increased over the years. Nonetheless, many tend to cut themselves off from various psychobiological disciplines that have obvious relevance to understanding the origin of human behavior and psychopathology. Apparently, for the field of clinical psychology to advance its search for the cause and origin of mental disorder, it must adopt a more versatile perspective against the background of the earliest rudimentary concepts and classifications of mental disorder that are clouded in mysticism, divinity, and speculation (Fowles, 2001; Kandel, 1998). Therefore, the tasks of investigating the underlying etiology of mental disorders, which constitute one of the cornerstones of psychopathological research, should also permeate the field of clinical psychology. These tasks should earnestly consider a broad examination of a class of variables (psychophysiological, neurotransmitter, hormonal and environmental activities) thought to have direct or indirect influence on the etiology of mental disorders.

The reality of biogenetic vulnerability in mental disorders is becoming more obvious than ever, thanks to the variety of research programs that is now experienced in the field of psychology. For this reason, it has been scientifically demonstrated that some precarious mental disorders once attributed to the wrath of gods emanate from biopsychological

phenomenon. For example, it has been known that schizophrenic patients have a higher risk of giving birth to offspring with major mental or personality disorders, and the risk increases if identical twins are involved. Nevertheless, the actual genetic activation or dominance involved in causation of mental disorders is not fully known since no single gene has been identified as a risk factor in many of the serious mental disorders. That the gene or genes involved interact with some environmental factor is clear since identical twins share the disorder only about 50% of the time. According to Schwartz and Africa (1988), each individual has an 8% risk of schizophrenia if his or her sibling is schizophrenic, a 12% risk if one parent is affected, a 14% risk of sharing the disorder with a fraternal twin, a 38% risk if both parents are affected, and 47% risk when two individuals share the same exact genes.

In real life experience, these figures can vary extensively, giving the individual the course to think that the vulnerability theory rather than strict biological inheritability is more attainable than the notion that schizophrenia is inherited.

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