The schizophrenia theory

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The schizophrenia theory is reinforced by the biological standard of psychiatry and remains the leading model of mental illness within the mentalhealthservice. Genetic factors commonly have accounted for an appraised 81–85% of the variance in liability to developing schizophrenia. Twin studies provide a benchmark approximation of heritability by which molecular genetic studies can guesstimate the degree of the contributions from either a particular poly-morphism or a GCTA pattern to the global genetic variance in schizophrenia liability, also offering evidence of the degree to which schizophrenia and other psychotic disorders may be co-heritable.

In a preliminary way, last century's changes in the rate or scale of environmental risk factors for schizophrenia have decreased the relative importance of environmental relative to genetic factors in schizophrenia liability (Kläning, 2106). In a May 3rd, 2013 an official press release from the leader of the DSM-5 Task Force, conceded that " despite four decades of determined effort, researchers have failed to identify any genetic variants that cause severe psychopathology" (Fosse, Joseph, ; Richardson, 2015).

In 2014, the Schizophrenia Working Group of the Psychiatric Genomics Consortium issued outcomes from the biggest genetic study of schizophrenia to date, broadcasting that 108 statistically substantial single nucleotide polymorphisms (SNPs) together could report for only 3. 4% of variation on the liability scale.

Given the long history of disappointments to replicate early findings in molecular genetics searches, it is premature to resolve that even this low

level of explained variance does not represent false positives. Most essentially, the lack of direct genetic confirmation of anything close to the high heritability estimates from twin studies has led to the view that there is a missing heritability problem for schizophrenia as well as for other mental disorders overall.

Environment

The extreme concordance for schizophrenia in monozygotic (MZ) twins is usually understood as verification of genetic influence. Some who study twins through lineage or population strategies have concluded that MZ twins nurtured apart (MZA) share fundamentally no environmental influences, and the MZA connection is a direct approximation of heritability.

Few researchers, specifically those focused on schizophrenia, have taken advantage of the fact that MZ twins propose a special opportunity to study prenatal influences. The placenta is a diagnostically important element of prenatalenvironment: " It is a physical and physiological link between mother and child, and it exhibits variations with regard to membrane type, size, shape, and circulation which may be important in themselves or may affect the nutrition of the embryo or the transport of drugs, toxins, and other agents which can influence brain development" (Davis; Phelps, 1995).

Genetics

The statement that genetics plays a role in the etiology of schizophrenia is recognized by all researchers in the field. There is substantial debates, however, regarding both the degree of that role and its defined nature; recent attention in linkage analysis and molecular genetics has amplified

such debates. Theories regarding the genetics of schizophrenia range from monogenic to polygenic.

The genes may possibly be dominant or recessive, at a single locus or several loci, have adaptable levels of penetrance and produce variable levels of disease expressivity. The genes may theoretically code for neurochemical (e. g., dopamine) abnormalities which could produce the symptoms of schizophrenia directly. Or they may control specific events in neurodevelopment, such as the proliferation and migration of neurons, which could produce the symptoms more indirectly.

Discussion

The schizophrenia hypothesis and the criteria for diagnosis are built intodiagnosticmanuals and provide mental health professionals with a structure, consistency, understanding and a standard from which they can provide clinical interventions and study their role and interventions. Yet, acceptance of genetic causes for schizophrenia is abhorrence to a person-centered, evidenced-based and recovery-orientated mental health service.

The concern of accepting a genetic cause for schizophrenia is the conviction that the symptoms and behavior are genetically encoded. If schizophrenia has a genetic base, then the frequency of schizophrenia will be greater amongstfamilymembers and/or relatives than it would be in the general populace. Looking at the principles that monozygotic or identical twins share, 100% of their genes are First-degree.

Relatives such as parents, fraternal twins, child, siblings share 50% of genes, second-degree relatives such as grandparents, aunts/uncles, half-siblings,

niece/nephew, grandchildren share 25% of their genes and third-degree relatives such as great grandparents, great aunts/uncles, cousins, great-grandchildren share 12. 5% of genes.

These philosophies dictate that the closer the relationship to the relative with schizophrenia the higher the risk of developing schizophrenia. It doesn't appear to be an unvarying rate of frequency in the studies that are being conducted and the practical differences makes it's dif? cult to compare and appeal firm unequivocal conclusions about the nature of hereditary transmissions of schizophrenia. One of the purest conclusions that can be sketched from the findings of family studies, however, is that they uniformly support an environment argument for schizophrenia.