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Commentary on Genetic Factors

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Commentary

Genetic factors seem to play a role in the development in anxiety disorders in humans. In familial studies, twin studies and adoption studies show genetic variances of 20% to 40%. The same was found for questionnaire variables such as neuroticism. Fear conditioning ability and predisposition to learn fear responses were extensively studied in animals. In rodents, fear processes or anxiety measured in the open field test or avoidance conditioning have revealed a strong genetic basis explaining most of the interindividual variance. However, the genes responsible for that trait have not yet been identified.

The situation is different for antisocial personality disorder and criminal behavior. Assuming that a deficit in anticipatory fear conditioning is the central symptom from which all consequent social problems follow, all evidence ranging from twin to adoption studies and gene sequencing suggests a moderate to strong genetic influence. If sensitivity to alcoholism and novelty-seeking are included as comorbidity factors the genetic variance increases further. Whether this is related to genetic fear conditioning remains to be seen.

The search for genetic factors is complicated by several factors. It is likely, for example, that disorders such as schizophrenia are etiologically heterogeneous. That is, it is probable that the syndrome of schizophrenia can be produced by a number of different combinations of genes and/or environmental factors. Thus, while each of a number of different genes may well increase risk for schizophrenia, it is likely that no single gene is *necessary* for the production of most cases. One approach to the problem of heterogeneity is to identify characteristics that distinguish specific, genetically more homogeneous, subtypes of schizophrenia or bipolar disorder. For example, found evidence for linkage at a locus on chromosome 11 with schizophrenia in one, but not the others, of several large pedigrees that they examined.

The schizophrenics in the extended family that *did* show linkage were distinguished from the families that did not by having a particularly severe and unremitting form of schizophrenia. If this finding can be confirmed in other pedigrees, it would provide a valuable example of the subtyping strategy.

In some cases, genetic diseases also are associated with *locus heterogeneity*, meaning that a deleterious mutation in any one of several genes can give rise to an increased risk of the disease. This is a finding common to many human diseases including Alzheimer's disease and polycystic kidney disease. Both allelic heterogeneity and locus heterogeneity are sources of variation in these disease phenotypes since they can have varying effects on the disease initiation, progression, and clinical severity.

Although there are many possible causes of human disease, family history is often one of the strongest risk factors for common disease complexes such as cancer, cardiovascular disease (CVD), diabetes, autoimmune disorders, and psychiatric illnesses. A person inherits a complete set of genes from each parent, as well as a vast array of cultural and socioeconomic experiences from his/her family. Family history is thought to be a good predictor of an individual's disease risk because family members most closely represent the unique genomic and environmental interactions that an individual experiences. Inherited genetic variation within families clearly contributes both directly and indirectly to the pathogenesis of disease. This chapter focuses on what is known or theorized about the direct link between genes and health and what still must be explored in order to understand the environmental interactions and relative roles among genes that contribute to health and illness.

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