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Anxiety and Depression in Inherited

Sajya M*

Department of Anxiety and Depression in Inherited, Johns Hopkins University School of Medicine., Baltimore

Introduction

Genetic linkage describes the way during which two genes that are located on the brink of one another on a chromosome are often inherited together. In 1905, William Bateson, Edith Rebecca Saunders, and Reginald C. Punnett noted that the traits for flower color and pollen shape in sweet pea plants appeared to be linked together. A few years later, in 1911, Thomas Hunt Morgan, who was studying heredity in fruit flies, noticed that the attention color of a fly was related to the fly's sex and hypothesized that the two traits were linked together. These observations led to the concept of genetic linkage, which describes how two genes that are closely associated on an equivalent chromosome are frequently inherited together. In fact, the closer two genes are to at least one another on a chromosome, the greater their likelihood is that of being inherited together or linked. In contrast, genes located farther faraway from one another on an equivalent chromosome are more likely to be separated during recombination, the method that recombines DNA during meiosis. The strength of linkage between two genes, therefore, depends upon the space between the genes on the chromosome. During their analysis, the researchers realized that there was an excess in the number of parental phenotypes (purple-long and red-round) in the F2 results. In particular, of the 2,132 F2 plants, 1,199 were expected to have purple flowers and long pollen grains, but instead, there were a whopping 1,528 plants with this phenotype. Similarly, only 133 plants were expected to possess red flowers and round pollen grains, but the researchers observed nearly 3 times that a lot of (381). It is now understood that the differences between the expected and observed results were statistically significant ($P < 0.005$), which means that the data could not be explained solely by chance. Genetic linkage analysis may be a powerful tool to detect the chromosomal location of disease genes. It is supported the observation that genes that reside physically close on a chromosome remain linked during meiosis. For most neurologic diseases that the underlying biochemical defect wasn't known, the identification of the chromosomal location of the disease gene was the primary step in its eventual isolation. By now, genes that are isolated during this way include examples from all kinds of neurologic diseases, from neurodegenerative diseases like Alzheimer, Parkinson, or ataxias, to diseases of ion channels leading to periodic paralysis or hemiplegic migraine, to tumor syndromes such as neurofibromatosis. With the advent of new genetic markers and automated genotyping, genetic mapping can be conducted extremely rapidly. Genetic linkage maps are generated for the human genome and for model organisms and have provided the idea for the development of physical maps that let the rapid mapping of disease traits. As soon as a chromosomal location for a disease phenotype has been established, genetic linkage analysis helps determine whether the disease phenotype is only caused by mutation in a single gene or mutations in other genes can give rise to an identical or similar phenotype. Often it is found that similar phenotypes can be caused by mutations in very different genes. Good examples are the autosomal dominant spinocerebellar ataxias, which are caused by mutations in several genes but have very similar phenotypes. In addition to providing novel, genotype-based classifications of neurologic diseases, genetic linkage analysis can aid in diagnosis. However, in contrast to direct mutational analysis like detection of an expanded CAG repeat within the Huntingtin gene, diagnosis using flanking markers requires the analysis of several relations .

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***Corresponding author:** Sajya, Department of Anxiety and Depression in Inherited, Johns Hopkins University School of Medicine., Baltimore, E-mail: ssing112@jh.edu*

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