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Post-Genomic Strategies for Elucidating the Etiology of Schizophrenia

Abstract

Human genetics studies have been remarkably successful over the past two decades. Using Genome-Wide Association Studies (GWAS) of common (>1-5% frequency) single nucleotide polymorphisms (SNPs) initially and more recently of rarer genetic variation discovered by DNA sequencing of exomes and whole genomes, hundreds of thousands of statistically significant associations have been found to traits and diseases. Unfortunately, our increase in understanding the biology of these traits and diseases has not increased at the same rate. The current task for the field of genetic and molecular biology is to turn our knowledge of locations in the genome that increase risk into the where, when and how molecular pathways in the body are altered and how these alterations change tissues and higher order functions. For instance, we want to know how changes in the genome alter the structure and function in the brain, and how each of these alterations affect behavior. I will describe two model systems that attempt to probe these connections from genome to higher-order properties of the brain and organism.