

Genetics of Infectious Diseases

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Description

Disease syndromes caused by infectious agents have occurred throughout the history of recent humans. As a result of our continued interactions with pathogens, our genomes are shaped through processes of co-evolution, with pathogen-imposed selection pressures resulting in selection signatures in ancient and modern human genomes. As one of several illustrative examples, genetic diversity involving human red blood corpuscle structure and performance is being impacted by an evolutionary race with malaria that's reciprocally seen in the parasite genome.

Genome-Wide Association Studies (GWASs) are performed within the field of human genetics to spot disease- or phenotype-related genetic variants. Infectious diseases are caused by bacteria, viruses, parasites, or fungi and these pathogens are considered together of the environmental factors of disease onset. The first GWAS in communicable disease was reported in 2007 for Acquired Immuno Deficiency Syndrome (AIDS).

Infectious diseases represent a serious ill health worldwide, both in terms of morbidity and mortality. A complex combination of environmental, pathogen and host genetic factors plays a task in determining both susceptibility to particular microbes and therefore the course of infection. Numerous studies have now mapped and identified relevant genes employing a sort of both family-based and population-based approaches. Much interest has been focused on susceptibility to malaria, HIV/AIDS and mycobacterial infection, but other bacterial, viral and parasitic diseases are receiving increasing attention. Some major genes are identified by genome scans of multi-case families, and mouse genetics has contributed to mapping and identification of a couple of genes.

Novel Approaches

A key emerging area is that the extent of population-specific

associations genome-wide. This successively emphasizes the relative paucity of GWAS undertaken in diverse worldwide populations, many of which have the very best burden of communicable disease. Greater efforts are going to be needed to focus GWAS and other genetic studies on such populations, especially where ethnic background could also be related to differential risk. It is thus imperative that genotyping arrays are designed for worldwide populations which the supply of huge imputation reference panels from diverse populations enables effective statistical inference of additional non-genotyped SNPs (and hence substantially increasing the informativeness of GWAS at no additional cost). New technologies like those supported low-coverage whole-genome sequencing may provide an alternate cost-effective approach for genotyping common and rarer genetic variants.

As is clear from the studies reported thus far herein, infections can afflict populations limited by geography and native ecology, and it's therefore crucial that genetic associations are tested for in non-European cohorts. However, such analyses accompany technical challenges due to the necessity to regulate the info for potential confounding by differences in population structure or relatedness which will cause an excess of type I errors. These aspects are appreciated for a few times, and lots of methods are developed to facilitate such adjustments. One method is principal component analysis (a mathematical method to summarize the most sources of variance in data) to model ancestry differences and account for differing allele frequencies in several populations. A further development is in statistical modelling using linear mixed model algorithms which will account for population structure and cryptic relatedness simultaneously.

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