Journal of Infectious Diseases & Therapy
ISSN: 2332-0877

Bocianowski, J Infect Dis Ther 2024, 12:4

Commentary Open Access

Using Association Mapping to Identify Candidate Genes Associated with Infection Diseases Resistance

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Received: 12-Jun-2024, Manuscript No. JIDT-24-138778; **Editor assigned:** 14-Jun-2024, Pre QC No. JIDT-24-138778 (PQ); **Reviewed:** 28-Jun-2024, QC No. JIDT-24-138778; **Revised:** 05-Jul-2024, Manuscript No. JIDT-24-138778 (R); **Published:** 12-Jul-2024, DOI: 10.4172/2332-0877.1000594

Citation: Bocianowski J (2024) Using Association Mapping to Identify Candidate Genes Associated with Infection Diseases Resistance. J Infect Dis Ther 12:594.

Abstract

Infectious diseases are responsible for a significant proportion of deaths in the world population. They are a major determinant of natural selection. Differential susceptibility to pathogens occurs among people from the same ethnic groups and belonging to different populations. The explanation for this phenomenon should be sought in the human genome. Ongoing research using commonly used genetic testing methods has made it possible to identify changes in certain genes that modulate an organism's susceptibility to a particular disease and the rate at which it develops. However, it is important to identify genes that determine resistance to infectious diseases. Genome-Wide Association Studies (GWAS) are a useful tool for identifying candidate genes, especially when combined with Quantitative Trait Loci (QTL) mapping to validate loci for observed traits.

Keywords: GWAS analysis; Association mapping; Infection diseases; Resistance

About the Study

Infectious diseases are one of the most common causes of death worldwide. Projections indicate that they will contribute to more than 15% of all deaths in 2030. Therefore, infectious diseases are among the 10 most common causes of premature death. The development and course of the disease depends on many factors: The dose of the pathogen, the time of exposure, age, the general condition of the body. In many patients, the development and course of the disease process appear not to be determined by the above aggravating factors [1].

Underlying immunity or modulation of an organism's response to infections is genetic variability, understood as gene-determined variation between individuals descended from the same population. Until recently, genome variability was most often characterized by single nucleotide polymorphisms. Current research is adding to our knowledge of Copy Number Variation (CNV). CNV is a common type of variation, affecting more than 1000 regions in the human genome, involving the acquisition or loss of DNA segments of $\geq$ 1000 bp. CNV-type changes affect gene expression variation and contribute to subsequent changes in chromosome structure. They are an important mechanism also responsible for evolutionary variation [2].

Mutations and polymorphic variants in genes can affect susceptibility to infection caused by a wide spectrum of microorganisms. Susceptibility to infection can also be more complex and multigene-dependent [3]. Such a diverse and multifactorial modulation of immunity and susceptibility to infection poses significant difficulties for research into their genetic background. The first step in this type of research is epidemiological analysis. It makes it possible to ascertain the existence of a genetic component affecting inter-population variation in the frequency and course of infection caused by the same microorganism [4].

Confirming a hypothesis that assumes the existence of genetically determined susceptibility to a specific disease requires the use of specialized statistical analysis. There are two main strategies for searching for resistance genes: Genome analysis using coupling analysis or association analysis and molecular analysis of the selected genes [4]. Coupling analysis involves studying the co-segregation of genetic markers with a disease phenotype. When a marker is located close to the gene responsible for the development or susceptibility to a particular disease, we assume that recombination between the marker and the gene will occur at a low frequency, and the marker will segregate with it. Thus, the presence of the marker will be significantly more frequent in individuals manifesting the disease phenotype, making it possible to narrow down the analysed region on the chromosome, and further to identify the gene. This type of strategy requires the study of multiple individuals-healthy and diseased-from the same family. In contrast, association analysis involves tracking the co-occurrence of known polymorphic variants that may be located in different parts of the genome with the disease phenotype, both in family members and in unrelated individuals. This approach makes it possible to identify gene variants more likely to occur in infected or diseased individuals [4]. GWAS are a useful tool for identifying candidate genes, especially when combined with QTL mapping to validate loci for susceptibility and resistance to disease [5].

Based on the results obtained from phenotyping and genotyping, association mapping is carried out using GWAS analysis. The latest techniques result in tens of thousands of genotyping data. This requires initial analysis and sorting of the data before further analysis. Most often, only sequences meeting the following criteria are selected for association analysis: Each sequence has at least 69 nt, minor allele frequency >0.25, missing observation fractions <10%. Association mapping is carried out using a method based on a mixed linear model with the population structure estimated by self-analysis and modeled

by random effects. The model used is based on the underlying genetic substructure in the population by retaining the most significant principal components from the molecular marker matrix.

The development of the immune response in response to infection of an organism with a specific pathogen depends on a number of factors, among which genetic predisposition is receiving increasing attention. The tremendous progress that has been made in recent years in genomics has enabled the development of studies on the interaction between the host genome and the genome of the pathogen that attacks it. Knowledge of the mechanism of infection and the immune response directed against a given pathogen at the molecular level seems to carry a number of tangible benefits: The development of new-generation drugs, the development of treatment strategies at different stages of the disease, the development of strategies for controlling epidemics and eliminating the pathogens that cause them. It will also be of tangible importance to know how individual polymorphisms affect the activity of genes and encoded proteins and their role in the regulation and course of pathogen-specific immune responses. This will enable individualized counselling on susceptibility to particular infectious diseases, estimating the prognosis for the course of the disease and monitoring the therapy used [6].

References

1. Bellamy R (1998) Genetic susceptibility to tuberculosis in human populations. Thorax 53:588-593.
2. Redon R, Ishikawa S, Fitch K, Feul L, Perry GH, et al. (2006) Global variation in copy number in the human genome. Nature 444:444-454.
3. Casanova JL, Abel L (2007) Human genetics of infectious diseases: A unified theory. EMBO J 26:915-922.
4. Clementi M, Di Gianantonio E (2006) Genetic susceptibility to infectious diseases. Reprod Toxicol 21:345-349.
5. Bocianowski J (2024) Using NGS technology and association mapping to identify candidate genes Associated with fusarium stalk rot resistance. Genes 15:106.
6. Kellam P, Weiss RA (2006) Infectogenomics: Insights from the host genome into infectious diseases. Cell 124:695-697.