Editorial Highlights on Medical Genetics of Infection (Special Issue)

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Editorial Note

Journal of Medical Microbiology and Diagnosis commemorates its decade long service to the scientific community by consistently publishing peer-reviewed articles and tracking the progress and significant advancements in the field of Microbiology. Ever since its inception in the year 2012, in addition to regular issue releases on a quarterly basis, this transdisciplinary journal is also releasing special issues and conference proceedings from time to time, thus comprehensively covering a wide range of topics and emerging challenges in Bacteriology, Clinical and Medical Diagnostics, Parasitology, Bacterial Infections. The journal focuses on application oriented research on Bacteriology, Clinical and Medical Diagnostics, Parasitology, Bacterial Infections. In this issue some of the recent and impactful research articles that were published by the journal will be discussed.

Since the early 1950s, the dominant paradigm in the human genetics of infectious diseases postulates that rare monogenic immunodeficiencies confer vulnerability to multiple infectious diseases (one gene, multiple infections), whereas common infections are associated with the polygenic inheritance of multiple susceptibility genes (one infection, multiple genes). Recent studies, since 1996 in particular, have challenged this view. A newly recognised group of primary immunodeficiencies predisposing the individual to a principal or single type of infection is emerging. In parallel, several common infections have been shown to reflect the inheritance of one major susceptibility gene, at least in some populations. This novel causal relationship (one gene, one infection) blurs the distinction between patient-based Mendelian genetics and population-based complex genetics, and provides a unified conceptual frame for exploring the molecular genetic basis of infectious diseases in humans.

For almost any given human-tropic virus, bacterium, fungus, or parasite, the clinical outcome of primary infection is enormously variable, ranging from asymptomatic to lethal infection. This variability has long been thought to be largely determined by the germline genetics of the human host, and this is increasingly being demonstrated to be the case. The number and diversity of known inborn errors of immunity is continually increasing, and we focus here on autosomal and X-linked recessive traits underlying complete deficiencies of the encoded protein. Schematically, four types of infectious phenotype have been observed in individuals with such deficiencies, each providing information about the redundancy of the corresponding human gene, in terms of host defense in natural conditions. The lack of a protein can confer vulnerability to a broad range of microbes in most, if not all patients, through the disruption of a key immunological component. In such cases, the gene concerned is of low redundancy. However, the lack of a protein may also confer vulnerability to a narrow range of microbes, sometimes a single pathogen, and not necessarily in all patients. In such cases, the gene concerned is highly redundant. Conversely, the deficiency may be apparently neutral, conferring no detectable predisposition to infection in any individual. In such cases, the gene concerned is completely redundant. Finally, the lack of a protein may, paradoxically, be advantageous to the host,

conferring resistance to one or more infections. In such cases, the gene is considered to display beneficial redundancy. These findings reflect the current state of evolution of humans and microbes, and should not be considered predictive of redundancy, or of a lack of redundancy, in the distant future. Nevertheless, these observations are of potential interest to present-day biologists testing immunological hypotheses experimentally and physicians managing patients with immunological or infectious conditions.

References

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