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Evolutionary dynamics of human autoimmune disease genes and malfunctioned immunological genes

Soumita Podder
Bose Institute, India

Abstract

One of the main issues of molecular evolution is to divulge the principles in dictating the evolutionary rate differences among various gene classes. Immunological genes have received considerable attention in evolutionary biology as candidates for local adaptation and for studying functionally important polymorphisms. The normal structure and function of immunological genes will be distorted when they experience mutations leading to immunological dysfunctions.

Here, we examined the fundamental differences between the genes which on mutation give rise to autoimmune or other immune system related diseases and the immunological genes that do not cause any disease phenotypes. Although the disease genes examined are analogous to non-disease genes in product, expression, function, and pathway affiliation, a statistically significant decrease in evolutionary rate has been found in autoimmune disease genes relative to all other immune related diseases and non–disease genes**.** Possible ways of accumulation of mutation in the three steps of the central dogma (DNA-mRNA-Protein) have been studied to trace the mutational effects predisposed to disease consequence and acquiring higher selection pressure. Principal Component Analysis and Multivariate Regression Analysis have established the predominant role of phosphorylation residues in guiding the evolutionary rate of immunological disease and non-disease genes followed by the m-RNA abundance, paralogs number, SNPs, alternatively spliced exon, protein disorder and protein residue burial.

Our study provides an empirical insight into the etiology of autoimmune disease genes and other immunological diseases. The immediate utility of our study is to help in disease gene identification and may also help in medicinal improvement of immune related disease.