Commentary
Volume 13:3, 2022

Human Genetics and Embryology

ISSN: 2161-0436 Open Access

Genes have Evolved to Face the Constant Pressures Imposed by Pathogens

Luis Antony*

Department of Genetics, School of Science and Technology, USA

Description

Remarkable progress has been made in recent years in our comprehension of the genetic basis of immunity-related disorders, spanning from infectious diseases to autoimmune, inflammation, and allergies. This development has been facilitated by the advent of genome-wide technology. This issue of Current Opinion in Immunology brings together experts in the fields of evolutionary, clinical, and epidemiological genetics who review the most exciting areas of recent research from these different angles of human genetics of immunity to infection. The reviews in this issue clearly reflect the breadth of emerging knowledge. The application of evolution and genomics to human immunology, as illustrated here, complements experimental research in model organisms, uncovering previously unrecognised features of immunology and significantly advancing our understanding of the immune system and its diseases.

Our understanding of the levels of naturally occurring genetic variation in healthy individuals has significantly increased as a result of the accessibility of genome-wide surveys of genetic variation from hundreds of individuals from various ethnic backgrounds, such as those provided by the HapMap Consortium or the 1000 Genomes Project. This knowledge is crucial for comprehending the role of genetic variation in disease. These datasets might be analysed to learn more about the demographic development of human communities and to assess how natural selection has shaped the diversity of the human genome. Numerous genes that may have been targeted by selection in its various manifestations and intensities have been discovered as a result of genome-wide scans for selection. It's interesting to note that selection has prioritised genes associated to immunity, supporting the idea that diseases have played a significant role in the evolution of the human genome. The authors go over a number of noteworthy instances of these genes, particularly those involved in innate immunity, whose biological functions and patterns of genetic variation are in line with pathogen-driven selection. The identification of genetic conflict hotspots caused by the host-pathogen arms race provides an intriguing twist on traditional approaches to host-pathogen interactions in this review.

The host has two options when confronted by a pathogen: either try to directly target it by actively recognising a pathogen structure (the "offence" strategy), or utilise a broader reaction meant to impair the pathogen's life cycle (the "defence" strategy). Here, some fascinating instances of "offence" and "defensive" mechanisms are provided, with TRIM5alpha serving as one example of a "offence" tactic the host developed to combat viral infections. Yet, with the increasing capacity to sequence entire genomes at affordable prices, it is likely that similar approaches will soon be applied to elucidate new interaction factors between host immune cells and non-viral agents. The Plasmodium parasites, which are the primary cause of malaria in humans, are probably at the top of the list of the numerous pathogens that have put strain on our DNA. With regard to its effects on human fitness and genome evolution, malaria perhaps has the strongest clinical, epidemiological, and evolutionary evidence of any infectious disease. The authors talk about how known biological interactions can influence the evolution of these many protective genetic traits. Recent genome-wide association studies (GWAS) and multicenter efforts are uncovering more resistant loci, which appear to have an effect on the immune system's homeostasis. Red blood cell function genes have long been recognised as essential contributors in resistance to malaria. The intriguing but provocative theory put out by the authors is that certain loci that boost malaria protection may also improve the parasite's fitness, encouraging the spread of such protective alleles.

When researching natural selection, we frequently have a bias in favour of either purifying selection, which eliminates harmful mutations from the population, or positive selection, which encourages the spread of beneficial variants. Balancing selection is the process of maintaining two or more variants at a specific locus for variable amounts of time. The MHC locus, at which some genetic polymorphisms are shared across people and evolutionary distant species like fish or birds, is one of the most well-known examples of this selection regime. The authors highlight recent genome-wide data suggesting that extra immune activities may have been driven by long-term balancing selection. Interbreeding between contemporary humans and extinct people, like Neanderthals, is another factor that has probably contributed to the diversification of the human immune system. According to ancient DNA studies, Neanderthals may have contributed up to 4% of the present Eurasian genomes. It's interesting to note that this admixture entailed the introduction of alleles that provide humans an advantage, many of which, once more, reside in immune-related genes. Additionally, a large number of the alleles currently linked to type-II diabetes, systemic lupus erythematosus, IL-18 levels, and susceptibility to Crohn's disease in Europeans appear to have been introduced through interbreeding with Neanderthals. We hypothesise that some of the reported discrepancies between people of African ancestry and those of European ancestry in their immunological responses to infection and susceptibility to disease may be the result of asymmetrical interbreeding between current and archaic human groups. [1-5].

Acknowledgement

We thank the anonymous reviewers for their constructive criticisms of the manuscript. The support from ROMA (Research Optimization and recovery in the Manufacturing industry), of the Research Council of Norway is highly appreciated by the authors.

Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

References

1. Stehling, Oliver, Claudia Wilbrecht, and Roland Lill. "Mitochondrial iron–sulfur protein biogenesis and human disease." *Human Genet Embryol* 100 (2014): 61-77.

***Address for Correspondence:** Luis Antony, Department of Genetics, School of Science and Technology, USA, E-mail: blayeran567@edu.in*

Date of Submission: 04 May, 2022, Manuscript No. hgec-22-76425; **Editor assigned:** 05 May, 2022, Pre QC No. P-76425; **Reviewed:** 17 May, 2022, QC No. Q-76425; **Revised:** 21 May, 2022, Manuscript No. R-76425; **Published:** 27 May, 2022, DOI: 10.37421/2161-0436.2022.13.179

2. Yoshimi, Akihide, and Omar Abdel-Wahab. "Splicing factor mutations in MDS RARS and MDS/MPN-RS-T" *Human Genet Embryol* 105 (2017): 720–731.

3. Burdon, Jeremy J., Luke G. Barrett, Greg Rebetzke, and Peter H. Thrall. "Guiding deployment of resistance in cereals using evolutionary principles " *Human Genet Embryol* 7 (2014): 609-624.

4. Gillings, Michael R. "Lateral gene transfer, bacterial genome evolution, and the Anthropocene." *Human Genet Embryol* 1389 (2017) 20-36.

5. Denamur, Erick, and Ivan Matic "Prospects and challenges of multi-omics data integration in toxicology." *Human Genet Embryol* 60 (2006): 820-827.

How to cite this article: Antony, Luis "Genes have Evolved to Face the Constant Pressures Imposed by Pathogens" Human Genet Embryol 13 (2022): 179.