Journal of Genital System & Disorders

Commentary A SCITECHNOL JOURNAL

Genetic Variability within the Human Population

Ágota Barabássy*

Department of Obstetrics and Gynecology, University Hospitals Case Medical Center, USA

***Corresponding author:** Barabassy A,Department of Obstetrics and Gynecology, University Hospitals Case Medical Center, USA, E-mail: agohbarab@gmail.com

Received date: 03 March, 2022, Manuscript No. JGSD-22-60016;
Editor assigned date: 04 March, 2022; Pre QC No. JGSD-22-60016(PQ);
Reviewed date: 21 March, 2022, QC No. JGSD-22-60016;
Revised date: 28 March, 2022, Manuscript No: JGSD-22-60016(R);
Published date: 31 March, 2022, DOI:10.4172/2325-9728.1000235.

Introduction

From 1900 to the present, Mendel's gene concept and Galton's biometric approach have coexisted; many current controversies, particularly in the field of behavioural genetics but also those concerning strategies in the genetic elucidation of common diseases, are readily understandable when the history of human genetics is viewed as a contest between these two paradigms. This is not to say that the two paradigms are mutually exclusive; in fact, Fisher in 1918 interpreted biometric analysis-based correlations between relatives in terms of gene activity. Some human geneticists have spent parts of their careers working within one paradigm and parts of their careers working within the other paradigm.

By and large, however, the two streams of research have few interconnections and may even become further polarized because of highly specialized training for each group, epitomized by the biochemical and molecular genetic laboratories for the one and the computer for the other group. In the first decades of the last century the biometric paradigm of Galton appeared to be very successful. Genetic variability within the human population was believed to be established for normal traits such as stature or intelligence as well as for a wide variety of pathologic conditions such as mental deficiency and psychosis, epilepsy, and common diseases such as diabetes, allergies, and even tuberculosis. Mendelian analysis, on the other hand, seemed to be confined to rare hereditary diseases; the ever repeated attempts at expanding Mendelian explanation into the fields of normal, physical characteristics and common diseases 4 Introduction were usually undertaken without critical assessment of the inescapable limitations of Mendelian analysis. The first major breakthrough of Mendelian genetics was the establishment of the three-allele hypothesis for the AB0 blood groups by Bernstein in the 1920s. However, the tendency toward specialization will inevitably continue, and it is possible that, in this process, important parts of human genetics will be resolved into fields mainly defined by research methods, such as biochemistry, chromosome research, immunology, molecular biology, or into certain clinical areas. For example, hereditary metabolic diseases or syndromes associated with dysmorphic features and developmental delay are often studied and treated by pediatricians with little genetic training. Several departments of neurology have established their own neuro genetics branches, which are often independent from the respective department of human genetics.

However, despite this tendency toward sub specialization, it is important to note that a laboratory performing genetic diagnostic procedures needs trained and experienced personnel, up-to-date equipment, and has to fulfill internationally defined quality standards, which are regulated by law in many countries. Therefore, it is probably not cost-effective to perform genetic diagnostics in small laboratories that offer only a few tests. Therefore, large laboratories performing all important human genetics diagnostic procedures may evolve to organizational structures in which human genetics remains united. Bacteria utilize a special energy saving system of genetic control called operons. The operon is a sequence of DNA that contains multiple genes used to produce multiple proteins for a single purpose. An example of an operon is the lac operon in E. coli. In order to break down lactose, E. coli must use a series of enzymes (beta-galactosidase, galactoside permease and transacetylase). The genes for these three enzymes are located in a row on the DNA and share a single promoter. Genes determining structure of a particular protein are called structural genes and the activity of structural genes are controlled by regulator genes, which lie adjacent to them. The genes lacZ, lacY and lacA which code for the three enzymes are the structural genes.

LacI gene codes for the repressor protein, hence is the regulator gene. Between the lacI gene and the structural genes lie promoter and operator genes. For transcription of the structural genes, the enzyme RNA polymerase first has to bind to promoter region. The operator region lies in between the promoter and structural genes and the RNA polymerase has to go through the operator region. Under normal circumstances, when the structural genes are not transcribed, the repressor protein is bound to the operator region thus preventing the passage of RNA polymerase from the operator region towards the operon. When lactose is available in the environment, the repressor protein leaves the operator region and binds to lactose because it has high affinity for lactose. This frees the operator region and the RNA polymerase enzyme moves towards the operon and transcribes the structural genes. The products of structural genes result in the metabolism of lactose. When lactose is no more available, the repressor protein goes back and binds to the operator region, thus stopping further transcription of structural genes. This way lactose acts both as inducer as well as a substrate for beta galactosidase.

Mutations

Mutations can alter the protein to which the antibiotic must bind resulting in a protein with little or no affinity for the drug. Mutations can be either step-wise, as seen with Penicillin, where high levels of resistance are achieved by a series of small-step mutations. Multiple-drug resistance in Mycobacteria is apparently the result of the step-wise accumulation of resistance to individual drugs. Mutations can also be one-step where single mutation is sufficient to bring about resistance in the bacteria as in Streptomycin resistance in Mycobacterium tuberculosis. In case of tuberculosis, Mycobacteria are known to mutate during the course of treatment. Initially the antitubercular drug kills the bacteria but soon resistant mutants develop which would eventually replace the sensitive ones resulting in treatment failure. It is for this reason that multiple drugs are included in the treatment of tuberculosis.

Citation: *Barabassy A (2022) Genetic Variability within the Human Population. J Genit Syst Disord 11:2.*