A GENERAL REVIEW ON QUANTITATIVE AND MOLECULAR GENETICS ALONG WITH THE STUDY OF HUMEN BEHAVIOUR

Pranjali Ajit Dixit
Principle
Vasantrao Purke Arts and Science Junior College,
Borgadi Pusad, MH.
bawaneshruti002@gmal.com

ABSTRACT

There are different ways in which researchers can study the contribution that genetic factors make to human behaviour. First, there are observational studies, which involve assessing and comparing relatives such as twins or siblings, families and adopted children. This type of research is called quantitative genetics because it aims to examine the extent to which variation in a trait is influenced by genetic factors in a population. It uses statistical methods to examine and compare groups of people, without focusing on particular genes. Secondly, researchers can try to identify differences in genes that contribute to trait variation in characteristics or traits between individuals. This type of research is called molecular genetics and its application to behavioural research is explained in the current paper. it also includes study of molecular genetics researchers .in some extent we try to study about behavioural genetics.

KEYWORDS *- Molecular genetics, Behavioural genetics, Quantitative genetics, Research*

INTRODUCTION

Human behaviour is influenced both by the genes that we inherit and the environment in which we live. With the significant advances in our knowledge of genetics and publication of the draft sequence of the human genome, the focus of research has moved once again towards understanding the biological contribution to behaviour. Some researchers are attempting to locate specific genes, or groups of genes, associated with behavioural traits and to understand the complex relationship between genes and the environment. This is called research in behavioural genetics. In contrast to research into the genetic basis of diseases and disorders, researchers in behavioural genetics investigate aspects of our personalities such as intelligence, sexual orientation, susceptibility to aggression and other antisocial conduct, and tendencies towards extraversion and novelty-seeking. If genes that influence particular behavioural traits are identified, it could become possible to test for the presence of variations in these genes in individual people. No such tests currently exist. Moreover, there is disagreement about whether tests that predict human behaviour accurately could ever be developed. But even if genetic tests could not yield predictions of a definite outcome, it may nonetheless be possible that tests that suggest an individual will have an increased chance of possessing a particular trait to a greater or lesser degree might be developed. Such hypothetical tests might be undertaken for a variety of purposes. One purpose would be simply to gain more knowledge about the influence of genes on behaviour. Another purpose might be that of intervention or treatment, for example to prevent aggressive behaviour by using medicines, or by attempts to change relevant aspects of the environment. A further purpose might be that of selection. This encompasses, for instance, prenatal testing, the streaming of children in

schools on the basis of intelligence and aptitude, the screening of employees and jobseekers to exclude those with traits that employers consider undesirable, and the use by insurers of genetic information about behaviour and personality traits in order to estimate risk. Yet another purpose of this study is to analyse the molecular genetics and the quantitative genetic

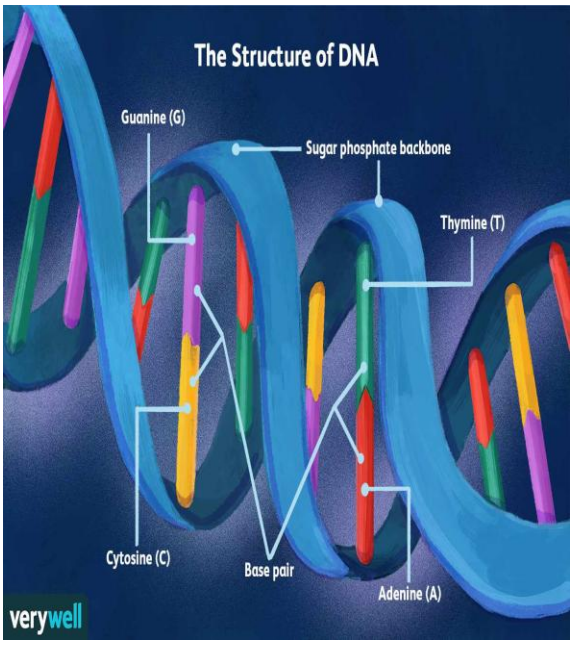
Each species of living organism has a unique set of inherited characteristics that makes it different from other species. Each species has its own developmental plan—often described as a sort of "blueprint" for building the organism— which is encoded in the DNA molecules present in its cells. This developmental plan determines the characteristics that are inherited. Because organisms in the same species share the same developmental plan, organisms that are members of the same species usually resemble one another, although some notable exceptions usually are differences between males and females. For example, it is easy to distinguish a human being from a chimpanzee or a gorilla. A human being habitually stands upright and has long legs, relatively little body hair, a large brain, and a flat face with a prominent nose, jutting chin, distinct lips, and small teeth. All of these traits are inherited—part of our developmental plan and help set us apart as Homo sapiens. But human beings are by no means identical. Many traits, or observable characteristics, differ from one person to another. There is a great deal of variation in hair colour, eye colour, skin colour, height, weight, personality traits, and other characteristics. Some human traits are transmitted biologically, others culturally. The colour of our eyes results from biological inheritance, but the native language we learned as a child result from cultural inheritance. Many traits are influenced jointly by biological inheritance and environmental factors. For example, weight is determined in part by inheritance but also in part by environment: how much food we eat, its nutritional content, our exercise regimen, and so forth. Genetics is the study of biologically inherited traits, including traits that are influenced in part by the environment. The fundamental concept of genetics is that: Inherited traits are determined by the elements of heredity that are transmitted from parents to offspring in reproduction; these elements of heredity are called genes. The existence of genes and the rules governing their transmission from generation to generation were first articulated by Gregor Mendel in 1866. Mendel's formulation of inheritance was in terms of the abstract rules by which hereditary elements (he called them "factors") are transmitted from parents to offspring. His objects of study were garden peas, with variable traits like pea colour and plant height. At one time genetics could be studied only through the progeny produced from mattings. Genetic differences between species were impossible to define, because organisms of different species usually do not mate, or they produce hybrid progeny that die or are sterile. This approach to the study of genetics is often referred to as classical genetics, or organismic or morphological genetics. Given the advances of molecular, or modern, genetics, it is possible to study differences between species through the comparison and analysis of the DNA itself. There is no fundamental distinction between classical and molecular genetics. They are different and complementary ways of studying the same thing: the function of the genetic material.

Molecular genetics

The foundation of genetics as a molecular science date back to 1869, just three years after Mendel reported his experiments. It was in 1869 that Friedrich Miescher discovered a new type of weak acid, abundant in the nuclei of white blood cells. Miescher's weak acid turned out to be the chemical substance we now call DNA (deoxyribonucleic acid). For many years the biological function of DNA was unknown, and no role in heredity was ascribed to it. This first section shows how DNA was eventually isolated and identified as the material that genes are made of.

DNA: The Genetic Material

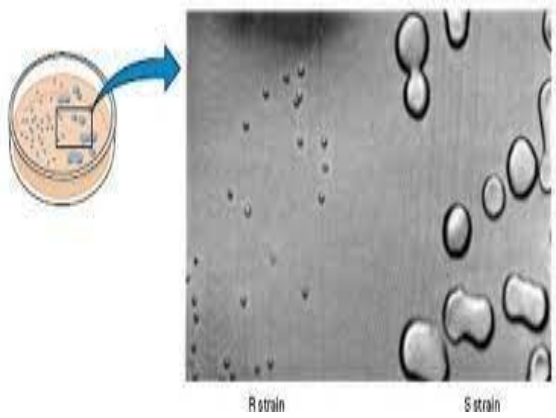
The cell nucleus plays a key role in inheritance was recognized in the 1870s by the observation that the nuclei of male and female reproductive cells undergo fusion in the process of fertilization. Soon thereafter, chromosomes were first observed inside the nucleus as thread-like objects that become visible in the light microscope when the cell is stained with certain dyes. Chromosomes were found to exhibit a characteristic "splitting" behaviour in which each daughter cell formed by cell division receives an identical complement of chromosomes Further evidence for the importance of chromosomes was provided by the observation that, whereas the number of chromosomes in each cell may differ among biological species, the number of chromosomes is nearly always constant within the cells of any particular species. These features of chromosomes were well understood by about 1900, and they made it seem likely that chromosomes were the carriers of the genes. By the 1920s, several lines of indirect evidence began to suggest a close relationship between chromosomes and DNA. Microscopic studies with special stains showed that DNA is present in chromosomes. Chromosomes also contain various types of proteins, but the amount and kinds of chromosomal proteins differ greatly from one cell type to another, whereas the amount of DNA per cell is constant. Furthermore, nearly all of the DNA present in cells of higher organisms is present in the chromosomes. These arguments for DNA as the genetic material were unconvincing, however, because crude chemical analyses had suggested (erroneously, as it turned out) that DNA lacks the chemical diversity needed in a genetic substance. The favoured candidate for the genetic material was protein, because proteins were known to be an exceedingly diverse collection of molecules. Proteins therefore became widely accepted.



Experimental Proof of the Genetic Function of DNA

An important first step was taken by Frederick Griffith in 1928 when he demonstrated that a physical trait can be passed from one cell to another. He was

working with two strains of the bacterium Streptococcus pneumoniae identified as S and R. When a bacterial cell is grown on solid medium, it undergoes repeated cell divisions to form a visible clump of cells called a colony. The S type of S. pneumoniae synthesizes a gelatinous capsule composed of complex carbohydrate (polysaccharide). The enveloping capsule makes each colony large and gives it a glistening or smooth (S) appearance. This capsule also enables the bacterium to cause pneumonia by protecting it from the defence mechanisms of an infected animal. The R strains of S. pneumoniae are unable to synthesize the capsular polysaccharide; they form small colonies that have a rough (R) surface (Figure 1.1). This strain of the bacterium does not cause pneumonia, because without the capsule the bacteria are e immune system of the host. Both types of bacteria



Quantitative genetics

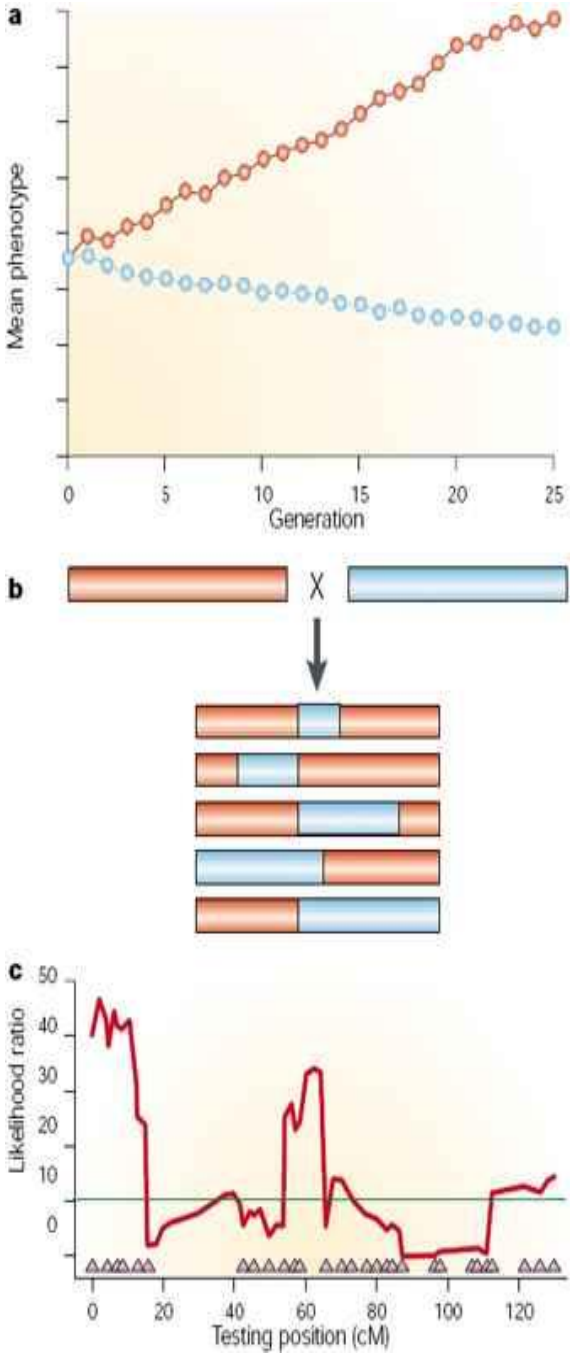
Single-predictor analysis

This section briefly covers two types of single predictor analysis strategies - QTL analysis and genome-wide association study (GWAS). Genome-wide association studies involve scanning markers across the genomes of many people to find genetic variations associated with a particular disease. We name them as "single-predictor" because the predictor can be a genetic marker (GWAS) or a single position between flanking markers (QTL analysis). This type of method focuses on one position on the genome at a time, fits a parametric model, and performs a hypothesis test on the genetic parameter. Both QTL analysis and GWAS require a genome-wide screening for significant loci. Single-predictor analysis is the simplest and the most direct way to detect potential causal genes for quantitative traits.

QTL analysis

A QTL (quantitative trait locus) is a chromosomal segment containing one or more genes that contributes to the variation observed for a quantitative trait. The genetic effects of a QTL are the combined effects of the genes located in the segment. GARDNER and LATTA (2007) have summarized that the average confidence interval around QTL is about 15.6 cm based on more than 200 mapped QTL. Generally speaking, QTL analysis is a statistical method that links two types of information - phenotypic data (trait measurements) and genotypic data (DNA variants) - in an attempt to explain the genetic basis of variation in complex traits. QTL analysis allows researchers in fields as diverse as agriculture, evolution, and medicine to link complex phenotypes to specific regions of chromosomes. The goal of a QTL analysis is to identify the action, interaction, number, and precise location of the regions (QTL) affecting a certain complex trait of interest. Two things are required in order to conduct a QTL analysis in an experimental population: 1. Two or more strains of organisms that differ genetically with regard to the trait of interest; 2. Genetic markers that

distinguish between these parental lines. By typing genetic markers as tags along the genome, DNA information of a 15 population can be obtained. The QTL interval mapping strategy was developed when the genetic markers were not as dense as the SNP array nowadays across the genome. When people started using micro-satellite markers (SSRs), every marker, regardless the information content, could be very precious. Techniques such as interval mapping were invented for mining as much information as possible from the limited number of sparse markers, trying to detect QTL harbored within flanking markers. An example of a powerful experimental design is the F2 inter cross. To carry out the QTL analysis, the parental strains are inter crossed, resulting in heterozygous (F1) individuals. These individuals are then mated to produce F2 individuals. The phenotypes and genotypes of the derived (F2) population are scored. Such an experimental design makes it possible to do QTL interval mapping, which is a linkage analysis technique that infers genotypes between flanking markers in order to identify QTL not lying on the marker positions. The possibility of doing such inference comes from recombination/crossing-over between homologous chromosomes. During a meiosis, chromosome segments are shuffled, so that the pieces, including the genetic markers therein, that are genetically linked to a QTL influencing the trait of interest will segregate more frequently with trait values, whereas unlinked markers will not show significant association with the phenotype Statistical modelling is of central importance for identifying QTL.



Genetics and human behaviour:

while it might be correct to say that a particular genetic variant is part of the cause of a particular trait, or that it is one causal factor, it will seldom be the only cause, nor is it likely to be either a necessary or sufficient condition for the trait to be manifested. Furthermore, even if particular genes that contribute to a trait can be identified, this is only a small part of the story. There is still a need to

understand the very indirect pathway between a gene, a particular protein and an individual scoring highly on an IQ test or having an aggressive personality. Our understanding of these causal pathways is at an even earlier stage than our understanding of which genes influence behavioural traits, which is itself extremely limited the complexity of human behaviour and the difficulties in understanding how genes are involved may seem overwhelming. There is wide agreement that genes do have an indirect effect on behaviour. However, some suggested that any attempt to understand the processes by which genes influence behaviour will certainly fail. We disagree. We consider that it is neither a theoretical nor a practical impossibility to identify genes that contribute to behavioural traits and to understand some of the mechanisms by which they do so. However, we note that terminology such as 'a gene for X' or 'a set of genes for X' is very misleading because it fails to convey the complexity of the role of genetic factors in causal explanations of human behaviour. Genes determine which proteins are made. They do not determine which behavioural or personality traits an individual possesses. Furthermore, the product of an individual gene will only very rarely be directly related to a complex behavioural characteristic. It will normally interact with many other genes and with many non-genetic factors, which means that the predictive capability of tests for any single or small number of genes will in general probably be quite limited. Nonetheless, the proteins that genes make and the way these affect our bodies and brains will be one part of an explanation of human behaviour. Reporting research in behavioural genetics Research which claims to show an association between particular genetic variants and particular traits tends to receive considerable attention in the scientific and lay media. The various methods of research in this field are not infallible, and the reviews of the evidence show that few findings have been replicated successfully to date. Thus, reports of such things as 'gay genes' or 'smart mice' convey a highly inaccurate impression of the state of the research. The lack of reporting of negative or contradictory findings exacerbates this problem. These difficulties are not unique to research in behavioural genetics. However, it does seem that such research is, at present, particularly susceptible to reporting which, whether strictly accurate or not, is misleading in the impression it gives to the reader. The potential for the abuse of findings in this area means that the reporting of this research ought to be conducted with particular care.

CONCLUSION

Quantitative genetics involves statistical methods that attempt to distinguish the effects of genetic and environmental factors on variation in certain behavioural traits, which can be quantitatively measured, between groups of individuals. Estimates of heritability and other statistical techniques are useful in understanding the relative contribution of different types of influence and their relation to each other In case of Molecular genetics to identify variation in particular genes that influences behaviour, by examining the DNA of individuals. This is difficult because there are usually many genes involved, each of which may only have a small effect. Many associations between a genetic variant and a behavioural trait have been reported but have not been successfully repeated by

other researcher. Predicting human behaviour from genetic information Even if it is not known precisely how a genetic variant contributes to a behavioural trait, it might be possible to predict how likely it is that individuals with that genetic variant will display the trait in question. Here, it is important to differentiate between predicting the future development of a phenotypic trait or specific behaviour, and measuring a phenotypic trait that is already established in an individual and can be observed. For example, if there were a genetic variant, or group of genetic variants, known to be associated with lower or higher intelligence, it would be possible to measure the genotype of a baby and to make some prediction of the IQ that the baby will have as an adult. Alternatively, measuring that genotype in an adult might enable the current IQ of the adult to be estimated. A third scenario for the predictive use of genetic information would be to predict the likelihood of the future occurrence of a specific act linked to a behavioural trait, for example an act of aggression.

REFRENCES -

1. *Ambrogelly A, Palioura S, Soll D (2007) Natural expansion of the genetic code.Nat Chem Biol 3:29–35*

2. *Bock A, Forchhammer K, Heider J, Baron C (1991) Selenoportein synthesis: an expansion of the genetic code. Trends Biochem Sci 16:463–467*

3. Breslauer KJ, Frank R, Bl¨ocker H, Marky LA (1986) Proc Natl Acad Sci 83:3746–375098 3 Molecular Biology

4. Brett D, Pospisil H, Valc´arcel J, Reich J, Bork P (2001) Alternative splicing and genome complexity. Nature Gen 30: 29–30

5. Bundschuh R, Gerland U (2006) Dynamics of intramolecular recognition: base-pairing is DNA/RNA near and far from equilibrium. Eur Phys J 10:319–329

6. Coffin JM (1996). Retroviridae. In: Fields BN, Knipte DM, Howley PM (eds.) Fields Virology Raven Publication

7. Collier J, Shapiro L (2007) Spatial complexity and control of a bacterial cell cyclecar Opin Biotechnol 18:333-340

8. Gibson M, Bruck J (2000) Efficient exact stochastic simulation of chemical system with many species and many channels. J Phys Chem A 104:1876–1889

9. Gillespie D (1976) A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. J Phys Chem 22:403–434

10. Gillespie D (1977) Exact stochastic simulation of coupled chemical reactions.J Phys Chem 81:2340–2361

11. Hewitt L, Kasche V, Lummer K, Lewist RJ, Murshudov GN, Verma GN (2000) Structure of a slow processing precursor penicillin acylase from Escherichia colireveals the linker peptide blocking the active cleft. J Mol Biol 302:887–898

12. Hohsaka T, Sisido M (2002). Incorporation of non-natural amino acids intoproteins. Curr Opin Chem Biol 6:809–815

13. Jimenez-Sanchez A (1995) On the origin and evolution of the genetic code. J Mol Evol 41:712–716

14. Kelly E, Russell SJ (2007) History of oncolytic viruses: genesis to genetic engineering. Mol Ther 15:651–659

15. Kopelman R (1988) Fractal reaction kinetics. Science 241:1620–1626

16. Lee SW, Mao C, Flynn CE, Belcher AM (2002) Ordering of quantum dots using genetically engineered viruses. Science 296:892–895

17. Mattick JS, Makunin IV (2006) Non-coding RNA, Hum Mol Gen 15:17–29

18. McMahon BJ (2005) Epidemiology and natural history of hepatitis B. Semin Liver Dis 25:38

19. Nam KT, Kim DW, Yoo PJ, Chiang CY, Meethong N, Hammond PT, Chiang YM, Belcher AM (2006) Virus-enabled synthesis and assembly of nanowires for lithium-ion battery electrodes. Science 312:885–888

20. Noad R, Roy P (2003) Virus-like particles as immunogens. Trends Microbial 11:438–444

21. Ryu WS (2003) Molecular aspects of hepatitis B viral infection and the viral carcinogenesis. J Biochem Mol Biol 36:138–143

22. SantaLuica J Jr, Hicks D (2004) The thermodynamics of DNA structural motifs.Annu Rev Biomol Struct 33:415–440

23. Savageau MA (1995) Michaelis-Menten mechanism reconsidered: implications of fractal kinetics. J Theor Biol 176:115–24
24. Schnell S, Turner TE (2004) Reaction kinetics in intracellular environments with macro molecular crowding: simulations and rate laws. Prog Biophysics Mol Biol85:235–260
25. Tijssen K (1993) Laboratory techniques in biochemistry and molecular biology. Elsevier, Amsterdam