conformity with the results on Deccan traps already reported by Radhakrishnamurty (1963)[8] and Bhimasankaram (1964, 1965)[2,3] and very similar to the magnetization of the Coal field dykes (Singh and Rao, 1971)[10].

It is interesting to note that whereas the Mirzapur quartzites (Mishra, 1965; Sahasrabudhe *et al.*, 1966)[6,9] have a magnetization corresponding to the northern hemisphere, the results on the site 1(Billi, Mirzapur) indicate the position of India in southern hemisphere and is similar to the position of India during Deccan trap eruption. Hasnain (1970[4] in a similar case suggests the intrusion of lavas in the tensional cracks freshly produced during the northward drift of India at the time of Deccan trap activity. Alternatively and most probably, it may represent the magnetization direction of Precambrian rocks during the southward movement of India. It is quite similar to the magnetization of Mundwara complex (Athavale *et al.*, 1963)[1] of Precambrian age. This indicates that the igneous activity represented by the dykes near Billi junction in Mirzapur District could be of the same age as that represented by the Mundwara complex of Rajasthan. Singh and Rao (1970)[11] have reported similar cases for the magnetization of metabasic rocks of Dhanbad, which correspond to the magnetizations of both the hemispheres.

In some recent studies, Anjanappa (1971)[15] and Anjanappa and Suryanarayana (1971)[16] have reported Palaeomagnetism of Precambrian dolerite dykes of Andhra Pradesh. The magnetization corresponds to a position of the Indian site in the southern hemisphere very close to the equator. The three sets of dykes studied give rise to the three types of magnetization corresponding to the normal, reverse and intermediate directions of southern hemisphere. The normal and reverse magnetizations combined give a pole position (VGP) of about 65° N and 65° W, whereas the intermediate magnetization gives rise to a pole position of about 7° N and 51° W. The dyke giving rise to the intermediate magnetization being, probably, the youngest, seems to have been magnetized during the southward drift of India. Same thing seems to be true for the Mysore dykes reported by Hasnain and Qureshy (1971)[4] as indicated earlier in this paper by the present authors.

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BIOLOGY AND GENETICS AT THE MOLECULAR LEVEL

A SYMPOSIUM on "Biology and Genetics at the Molecular Level" was held on 9th November, 1971 during the 37th Annual Meeting of the Indian Academy of Sciences at the Raman Research Institute, Bangalore, 7–9 November, 1971. Professor T. S. Sadasivan, Director, Centre for Advanced Studies in Mycology and Plant Pathology, University Botany Laboratory, Madras, was the Chairman of the Symposium. The following are the authors' summaries of the papers presented and discussed at the Symposium.

Concept of Gene Action

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During the last two decades the depth of knowledge attained on the concept of parti-

culate inheritance of Mendel has been so impressive that one feels quite comfortable with words like molecular genetics and genetic manipulation, genetic engineering, etc. "Genes" were identified as the material that control phenotypic expression of characters after the rediscovery of the important laws of heredity of Mendel. The curiosity and deepsighted enquiry on inborn errors of metabolism raised the question of relationship between "genes" and chemical functions of living cells. The "one gene-one enzyme" concept put this relationship on much firmer foundation and it was shown possible by detailed experiments with the mold *Neurospora*.

The entry of bacteria as experimental material to test in more detail this relationship was so timely that questions raised after the experiments with *Neurospora* got answered with bacteria. Among such answers were that (1) Deoxyribonucleic acid is the chemical molecule that carries the properties of genes. (2) Alteration in the chemical nature of deoxyribonucleic acid can lead to gene mutation. (3) Such mutations are directly expressed in the form of change in the structure and function of proteins. It is also well established that changes in proteins lead to alteration in the rate of chemical reactions whose product by itself or through other chemicals lead to morphological or physiological traits of the individual organism. During the identification of these relationships, sophisticated experimental techniques involving several biophysical and biochemical principles and characters of cellular chemical molecules (with bacteria and bacteriophages) have revealed the genetic code, colinearity, regulation of gene function and finally *in vitro* synthesis of a piece of DNA having specific gene function.

However, the efforts, that have been put to universify the concept of gene function seen in micro-organisms to eukaryotes and other higher organisms, have met with difficulties, because of the complexity of the cellular component, the chromosomes, which carry the DNA, along with proteins. Nevertheless large number of important steps in the process of gene expression are common between the eukaryotes and procaryotes. But how the genes are allowed to express at times, but other times kept inactive? This process, perhaps, forms the basis of cellular differentiation leading to tissue differentiation. This is still a mystery. The prognosis is not bleak, because a large amount of data tend to support the concept that interaction of proteins with DNA may have a significant role in the process of gene expression. The roles of histones and acidic proteins are getting more and more delineated. The role of repressor, a protein molecule, in the expression of bacteria has been well established. The expression of certain bacterial and bacteriophage genes is controlled by a repressor protein synthesized by regulatory genes. Only when the repressor protein is inactivated are the genes expressed. The regulation of the regulatory genes themselves is an interesting problem. In the case of the *lac* operon of *E. coli* the repressor gene, *Lac I* appears to be expressed at the same level whether or not the operon is induced. In the phage λ, the product of the gene CI, a repressor, is controlled by the product of the gene *tof*; and such an action leads to lysis of the host cell. During lysogeny the repressor prevents expression of the *tof* gene. In *Salmonella thyphimurium* a regulatory gene product, a repressor, represses its own synthesis besides regulating the synthesis of the enzymes under its control—such examples of gene regulation are likely to be shown in higher organisms and perhaps will lead to greater knowledge on the process of gene action in relation to differentiation and morphogenesis.

Molecular Basis of Sporulation

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Sporulation in bacteria is the simplest form of cellular differentiation one can envisage. The complex problem of animal cell differentiation has hitherto defied the systematic study of the biologists. Hence study of the events leading to or following bacterial differentiation is of immense interest to all concerned.

The recent impact of the interests of the biochemists and molecular biologists in this field has opened up a large-scale understanding of the phenomenon of bacterial sporulation. Sporulation involves the metamorphosis of a fragile vegetative cell into a dormant resistant spore much like our own ageing phenomenon! Several macromolecular happenings characterise this event.

The question is how does this happen? Are the cells predetermined to sporulate? If their are genes for differentiation, what makes them "silent" throughout the vegetative phase and

what "turns them on at the right time? It has been shown recently that RNA synthesis is the target of regulation and the controlled expression of the various genes at various times seems to be better understood now. It is the alteration of this control at the time of sporulation that results in the inactivation of the genes for the synthesis of ribosomal RNA. This results in sporulation since ribosomal RNA constitutes the major machinery of protein synthesis, so vital to the young cells.

But then there are still more fascinating questions to be asked and answers to be learned. What triggers this differentiation process or switch-over? One possible approach to this problem stems from the fact that the sporulating bacteria are also endowed with secondary cellular processes like antibiotic production. Some of our studies tend to assign these compounds to a regulatory role in controlling morphogenesis. Is this chemodifferentiation the cause or effect of sporulation, is the question that is currently engaging our attention.

Molecular Genetics and Human Diseases

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Diseases afflicting humans can be divided into three groups: 1. Those which are caused by external agents and which persist in the body only so long as the agent remains in the body. Typical examples of this group are bacterial diseases like typhoid and pneumonia, digestive disorders like gastritis, etc. Normally these diseases, infectious by nature, do not cause or leave any permanent change in the person. 2. Secondly, there are those diseases which are due to defects or deficiencies in the composition of the person, in his genetic make-up. These diseases, arising out of "inborn errors of metabolism", are essentially hereditary by nature, the germ cells being affected. The persons afflicted, thus, remain 'genetic cripples' throughout their lifetime, though they may not actually show-off or 'suffer' from the disease, which may remain dormant and recessive for a long time. 3. Lastly, there is that unique disease, cancer, which can arise due to the action of a variety of external agents, but all leading to the uncontrolled proliferation of some somatic cells. Once the cells are altered, the disease becomes self-perpetuating and the extrinsic agent is no longer needed. The disease is neither infectious, nor hereditary as only the somatic cells are involved.

Ever since Garrod's classic treatise on "Inborn Errors of Metabolism" appeared, more and more knowledge has piled up on this subject and an increasing number of diseases has been traced to anomalies at the genetic level. A defective gene gives rise to a defective protein, which does not fulfil its obligations, resulting in metabolic defects and consequent outward manifestations in the individual concerned. A genetic disease, which has been thoroughly investigated and completely understood, is sickle-cell anemia. Humans afflicted with this disease possess a haemoglobin that is structurally different, though only very slightly, from that of the normal individuals. The abnormal haemoglobin contains one valine residue instead of one glutamic acid moiety; and this makes all the difference between life and death. For the red blood cells containing this abnormal haemoglobin tend to 'sickle' in the venous blood due to poor oxygenation and are summarily destroyed by the spleen very rapidly, resulting in anemia, and death.

Wilson's disease is another instance of 'genetic crippling'. Here, the free copper is found to an enormous extent in circulation, which is subsequently deposited in liver, brain and eyes. This causes the hepatolenticular degeneration. Two reasons may be adduced for this: either the copper-binding protein, ceruloplasmin, is not produced at all or structurally altered in such a way to prevent the binding to the metal. The distorted information flowing out of a defective gene has thus resulted in insufficient or structurally-altered protein, which gives rise to this genetic disease.

Thus any anomaly at the molecular level, either a gene block or gene alteration results in erroneous expressions at the enzyme translational level, which is inherited. A genetic basis for many developmental abnormalities has been adduced in many human diseases, some of which are mentioned below: albinism and phenylketonuria (both occurring due to defects in the amino acid metabolism—of tyrosine), galactosemia (where the enzyme galactose-1-P uridyl transferase is absent, resulting in accumulation of galactose in the infant), maple-sugar urine disease (a fatal disease due to defective metabolisms of the branched chain amino acids, leucine, isoleucine and valine), and xanthosis (where the absence of the enzyme xanthine-oxidase results in elevated concentrations of xanthine in plasma and urine). Many other human diseases like muscular distrophy and diabetes are now

thought to be resulting from aberrations at the molecular level.

In cancer, malignant cells could have arisen only from normal cells. How exactly the malignant transformation takes place is not certain. It could be due to alteration of bases in the DNA, resulting in 'somatic mutations', or it may be due to removal of genetic repressors. But whatever may be the genetic alteration, it is perpetuated in the subsequent daughter cells. The discovery of the Philadelphia chromosome in chronic myeloid leukemia and the frequent chromosomal abnormalities seen in the nuclei of the cancer cells afford further evidence for genetic alterations in cancer.

Molecular Biology of Plant Disease

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The principles of molecular biology are just beginning to be applied to the study of plant diseases. It is well recognised that genes and gene products of both the parasite and the host participate in host/parasite interactions. It, therefore, implies that gene activity of the parasite may be controlled by host genes and *vice versa*. Although the gene for gene concept is firmly entrenched in plant pathology, the underlying molecular mechanisms have not been elucidated. However, available evidence indicates that genetic activity of host and parasite may be controlled at the transcriptional, translational or enzyme level. Recent work suggests that the transcription pattern of host DNA may be changed during pathogenesis and inhibition of transcription of host DNA in infected cells may be mediated by histones.

The infection process by fungi is frequently accompanied by morphogenetic changes and the formation of infection structures would appear to depend on the synthesis of messenger RNA. Cell wall degrading enzymes seem to be under the genetic control of both the pathogen and the host. Resistance genes of the host are assigned functions in determining the type of glycosidic linkages in cell walls. Virulence may be reflected in the absence of a specific gene product. Mono- and disaccharides released from host cell walls may act as highly specific effectors for the induction and repression of polysaccharide-degrading enzyme synthesis by pathogens. Newer evidence also suggests that glycoproteins of plant cell walls may be analogous to animal antibodies. These proteins inhibit pathogen-secreted endopolygalacturonases but not several other enzymes, and distinguish between polygalacturonases secreted by different species of pathogenic fungi.

Synthesis of proteins and increased enzyme levels are ubiquitous phenomena in diseased tissues of plants. It is suspected that a *de novo* synthesis is involved owing to the triggering of silent host genes into activity by gene products of the pathogen. In incompatible host-parasite relationships this may lead to the synthesis of abnormal metabolites (phytoalexins) implied in disease resistance. It has been proposed that genes controlling the phytoalexin responses may be derepressed by compounds having the potential to change the conformation of double stranded DNA. Interaction between specific products of avirulent genes of the pathogen and specific receptors, controlled by resistance genes, on host cell membrane has also been invoked in explaining the gene for gene relationship in plant diseases.

Biogenesis of Membranes

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The biomembranes carry out a wide array of biological functions—selective permeability, electrical insulation and energy transduction are randomly selected examples. Almost all membranes contain protein, lipid and carbohydrate in varying proportion. To fit in all these components into an organised, functional structure is engaging the attention of chemists and biologists around the world. The proposal of Danielli and Davson in the early thirties about the bilayer structure of membrane is still dominating our conceptualisation about membrane structure.

The biogeneticists are interested in knowing how these various compounds are put together. The main question is—is there a universal model of membrane building, whether it be a template-and-mold model or an assembly-line model ? The situation has become more complicated by the finding that some membrane systems like mitochondria and chloroplasts have their own DNA and are semi-autonomous. Are these membranes then self-duplicating ?

Survey of literature to date establishes (albeit temporarily) two things : (*a*) The assembly-line model seems to be the favoured system in many of the cases studied.

(*b*) The membrane structure is flexible enough to permit independent turnover of individual components.

The field is young and the problem is not insoluble (although the membranes are !).