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Phenomena of Life and Death Based on Nonphysical Gene and Computer Model of Organism

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Abstract

Genetic program (biological information), the driving force of life, is currently believed to be the information encoded by a chemical structure, DNA. The molecular gene (genome) concept is founded on this view. It now faces considerable conceptual problems and is examined here vis-à-vis nonphysical gene originally proposed by Wilhelm Johannsen. Molecular gene also fails miserably to explain the phenomena of life and death. Nonphysical gene is conceived in the light of computer model of organism. Organism is natural biocomputer or more precisely biorobot. It has both hardware and software. The computer model treats biological information (biosoftware) as information stored on the chromosome like the software of a computer stored on its hard disk. The chemical structures in the cell including DNA constitute the hardware. Life can be defined as the manifestation of the execution of the instructions carried in the biosoftware, and death as the result of deletion of the biosoftware from the biosystem. A dead body is like a computer without software. This will explain why a dead cell cannot be cultured and life cannot be created through chemical means. Computers, robots, etc. that run on man-made software can be considered as forms of artificial life.

Keywords: computer model of organism; molecular gene; genome; nonphysical biosoftware, phenomenon of life, phenomenon of death

1. Introduction

In 1865, the Austrian monk Johann Gregor Mendel proposed three laws governing heredity, which however did not see light of the day until after 30 years when in 1900 three botanists independently and almost simultaneously rediscovered them.

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Bateson coined the term "genetics" for the emerging science of heredity in 1906. Subsequently in 1909, Wilhelm Johannsen introduced the notions of "genotype" and "phenotype". In addition, for the elements of the genotype, he proposed the term "gene". Johannsen had reservations with respect to gene's particulate nature and had also warned against conceiving genes for a particular character (Johannsen, 1911). Thus the gene remained a hypothetical entity as Mendelian genetics did not permit supposition of material genetic elements.

"[I]t became the conviction of many geneticians in the 1920s, among them Morgan's student Herman J. Muller, that genes had to be material particles. Muller saw genes as fundamentally endowed with two properties: that of *autocatalysis* and that of *heterocatalysis*…. With his own experimental work, Muller added a significant argument for the materiality of the gene, pertaining to the third aspect of the gene as a unit of mutation. . In 1927, he reported on the induction of Mendelian mutations in *Drosophila* by using X-rays. The conclusion was at hand that the X-rays must have altered some molecular structure, like spontaneous mutations, in a permanent fashion. But the experimental practice of X-raying, which eventually gave rise to a whole 'industry' of radiation genetics in the 1930s and 1940s, did by itself not open the path to the material characterization of genes as units of heredity. On the occasion of the fiftieth anniversary of the rediscovery of Mendel's work in 1950, Muller thus had to confess: "[T]he real core of gene theory still appears to lie in the deep unknown. That is, we have as yet no actual knowledge of the mechanism underlying that unique property which makes a gene a gene – its ability to cause the synthesis of another structure like itself, [in] which even the mutations of the original gene are copied. [We] do not know of such things yet in chemistry" (Rheinberger et al., 2004).

It has been known since about 1913 that the individual active units of heredity - the genes - are strung together in one-dimensional array along the chromosomes, the threadlike bodies in the nucleus of the cell. George Beadle and Edward Tatum during the late 1930s and early 1940s established the connection between genes and metabolism.

They proposed the "one gene, one enzyme hypothesis". Since chemical reactions occurring in the body are mediated by enzymes, and since enzymes are proteins and thus heritable traits, it is supposed that the gene and proteins are related. These views of gene function strengthened the idea of genetic specificity leading to molecularization of the gene.

In the early 1940s, Oswald Avery and his colleagues purified the deoxyribonuleic acid (DNA) of one strain of bacteria, and demonstrated that it was able to transmit the infectious characteristics of that strain to another, harmless one (Rheinberger et al., 2004). Elucidation of the structure of DNA as macromolecular double helix by Francis Crick and James D. Watson in 1953 and *in vitro* characterization of the process of protein biosynthesis led to the idea that it was the linear sequence of ribonucleic acid derived from one of the DNA strands that directed the synthesis of a linear sequence of amino acids, or a polypeptide, and that this process was mediated by an adaptor molecule (RNA template). In 1958 Francis Crick formulated the "sequence hypothesis" (triplet code or codon, i.e., three bases at a time specified one amino acid) and the "central dogma" of molecular biology. All these considerations ultimately led to defining the molecular gene. According to the classical molecular concept, a gene is a stretch of DNA that encodes a functional product, a single polypeptide chain or RNA molecule. The entire collection of genes encoded by a particular organism is the "genome" that is supposed to constitute the genetic program. Johannsen's non-particulate gene thus metamorphosed into particulate gene. The molecular gene was born!

The material gene is not strictly a product prompted by research findings; rather it is more a product of the conviction of geneticists that *the gene has to be material entity*. The assumption of "one gene, one protein" makes the genes generally synonymous with proteins. Thus the term "gene" refers to the gene that codes for protein. Molecular biology opened the floodgates of boundless optimism about the ability of the super molecule DNA to decipher the mechanism of life as well as the potential of gene for genetic manipulation. Today with the recognition of the molecular genome as the biological program, an organism is reduced to mere bundle of physical particles or molecules. In his classic and influential textbook, *The Molecular Biology of the Gene*, James Watson stated: "We have complete confidence that further research of the intensity given to genetics will eventually provide man with the ability to describe with completeness the essential features that constitute life." (Watson, 1973). But he was grossly wrong.

Peter Cook reflects: "Watson and Crick must have thought that the sequence was everything. But life is much more complicated than that." (Pearson, 2003).

2. Problems with the Material Gene

Over the past six decades following elucidation of the chemical structure of DNA, the genetic research has been centred round the molecular gene and genome concepts. Genome, a chemical structure, is believed to constitute the genetic program or the 'blue print of life' that is responsible for the phenotypic characters and biological functions of an organism. A discussion of the anomalies and inadequacies of the molecular gene (genome) is presented here to show why the material gene concept cannot explain the phenomena of life and death.

2.1 Molecular Gene is Indefinable

The perception that DNA molecule encodes the biological program has run into serious problems. Although molecular biologists hoped that it would be possible to identify the genes for different attributes of an organism, the gene remained elusive. According to geneticist Peter Portin, "The gene is no longer a fixed point on the chromosome, producing a single messenger RNA. Rather, most eukaryotic genes consist of split DNA sequences, often producing more than one mRNA by means of complex promoters and/or alternative splicing. Furthermore, DNA sequences are movable in certain respects, and proteins produced by a single gene are processed into their constituent parts.

Moreover, in certain cases the primary transcript is edited before translation, using information from different genetic units and thereby demolishing the one-to-one correspondence between gene and messenger RNA. Finally, the occurrence of nested genes invalidates the simpler and earlier idea of the linear arrangement of genes in the linkage group, and gene assembly similarly confutes the idea of a simple one-to-one correspondence between the gene as the unit of transmission and of genetic function...." (Portin, 1993). Other leading scientists like Thomas Fogle and Michel Morange also concede that there is no longer a precise definition of what could count as a gene (Fogle, 2000; Morange, 2000). An important objective of genome projects is the identification of genes. Current estimates of human genes generated from genome sequencing range between 30,000 and 40,000 with occasional excursions to 100,000 or more.

One reason for the continuing ambiguity is that genes are neither well defined nor easily recognizable (Eddy, 2001). Horace Freeland Judson notes: "The phrases current in genetics that most plainly do violence to understanding begin "*the gene for"*: the gene for breast cancer, the gene for hypercholesterolaemia, the gene for schizophrenia, the gene for homosexuality, and so on. We know of course that there are no single genes for such things." (Judson, 2001).

The objective of genomic research is to ultimately understand the relationships between heritable units and their phenotypes. But it appears that genome concept would not deliver this information. The genome organization is extremely complex. Genes reside within one another, share some of their DNA sequences, are transcribed and spliced in complex patterns, and can overlap in function with other genes of the same sequence families. "Today, in the era of genomic sequencing and intense effort to identify sites of expression, the declared goal is to search for genes, entities assumed to have physical integrity. Ironically, the sharper resolving power of modern investigative tools make less clear what, exactly, is meant by a molecular gene, and therefore, how this goal will be realized and what it will mean", observes Fogle (Fogle, 2000).

Instead of generating more evidence in support of the particulate nature of the gene, research in molecular biology is generating evidence to the contrary. Craig Holdrege observes: "The complexity at the molecular level reveals that the simple mechanisms one imagined in the 1960s simply do not exist in that form. It has become less and less clear what a gene actually is and does. And although the deterministic gene is still the gene that lives in the minds of many students, lay people, and - at least as a desire - in the minds of many biologists, the findings of late twentieth century genetics show one thing clearly: the simple deterministic gene, the foundational "atom" of biology is dead. There is no clear-cut hereditary mechanism - no definite sequence of nitrogenous bases in a segment of a DNA molecule that determines the make-up and structure of proteins, which in turn determine a definite feature of an organism." (Holdrege, 2005). Evelyn Fox Keller makes the case for a radically new thinking about the nature of heredity in her book *The Century of the Gene*. In her articulate and insightful history of genetics and molecular biology, she suggests that most of our common assumptions about genes are either too simplistic or simply incorrect.

It turns out, for example, that a single functioning gene may be split and found in several locations on a chromosome, and it is rare that a gene can be determined to have caused any particular trait, characteristic or behavior (Keller, 2000).

2.2 Lack of Genome-Phenome Correspondence

Studies at the molecular level fail to demonstrate the expected correspondence between genome and phenotype. The most spectacular example of this is the morphological dissimilarity between human being and chimpanzee despite a 98.7% similarity in their DNA (Wells, 2001). Although evolutionary biologists speak of genomes of chimp and man as being almost identical in support of their argument of human evolution from an animal, and for establishing chimpanzee as the closest animal ancestor of human being, they have not enumerated so far the identical phenotypic characters in human and chimp in terms of anatomy, physiology, development and other biological features. In fact there is none! A chimp is not even 0.1% human being nor a human being 0.1% chimp. A human being differs from chimp in every detail and at every point of the body. The only similarity between chimp and man is in the DNA. The differences in traits, characteristic behaviour, instincts and capabilities between human (*Homo sapiens*) and chimpanzee (*Pan* sp.) are far greater than the small degree of sequence divergence (1.3%) could account for.

The chimp-human comparison is a case of similar genomes but dissimilar phenotypes. The reverse case is also known. *Caenorhabditis elegans* and *C. briggsae* are physically very similar organisms. It takes an expert to distinguish them. The two have near-identical biology, even down to the minutiae of developmental processes. Surprisingly, however, their genomes are not so similar. *C. elegans* has more than 700 chemoreceptor genes when *C. briggsae* gets on by just 430. There are also many genes unique to each of them (Blaxter, 2003). “Typically when people say that the human genome contains 27,000 genes or so, they are referring to genes that code for proteins,” points out Michel Georges, a geneticist at the University of Liège in Belgium. But even though that number is still tentative – estimates range from 20,000 to 40,000 – it seems to confirm that there is no clear correspondence between the complexity of a species and the number of genes in its genome. Fruit flies have fewer coding genes than roundworms, and rice plants have more than humans (Gibbs, 2003).

Many insects exhibit alternative morphologies (polyphenisms) based on differential gene expression rather than genetic polymorphism (differences in genes themselves). One of the best understood insect polyphenisms is the queen-worker dimorphism in honey bees. Both the queens and the workers are females but morphologically distinct forms. Besides, the queen is fertile whereas the worker is sterile. Studies conducted with the bee species *Apis mellifera* revealed that numerous genes appeared to be differentially expressed between the two castes (Evans and Wheeler, 1999). The seven differentially expressed loci observed in the study belonged to at least five distinctly different functional groups. The queen and the worker castes in honey bee provide an unfailing proof of natural existence of similar genomes exhibiting dissimilar phenotypes. The absence of genome-phenome relationship is very much evident from these studies. It implies that molecular genome does not constitute the biological program. All these cases indicate the independent existence of biosoftware as non-molecular information stored on the chromosome.

2.3 Molecular Gene Concept is Chemically Untenable

Several non-chemical features have been attributed to the material genome. There are several obvious departures from the chemical fundamentals. It has been observed that an overwhelming 95% of DNA consists of non-coding DNA in eukaryotes and about 5% is constituted by the coding-DNA (or the genes). The non-coding DNA (ncDNA) is referred to as "junk DNA". Though structurally comparable to coding DNA, surprisingly, the so-called junk DNA does not encode similar biological information (or vice versa).

Another surprising feature of the genome is that DNA is the only molecule in nature that can undergo self-alteration. How is it possible for a chemical structure to encode information for its own change? For example, in human being with the formation of the zygote, the biological program comes into operation for the development of the individual. The zygote undergoes ontogenetic development; then the individual passes through adult stage and old age, and ultimately dies. It is a continuous process like the operation of an integrated computer program. During ontogenetic development, the genome produces not only tissues with diverse functions but also undergoes itself changes in its structure as is evident from the recent discovery of variations in the genomes of different tissues (Gottlieb et al., 2009).

This discovery sprang from an investigation into the underlying genetic causes of abdominal aortic aneurysms (AAA) by a team of researchers led by Morris Schweitzer at McGill University. They found major genetic differences between blood cells and tissue cells of the same individuals. The finding calls into question one of the most basic assumptions of human genetics that DNA in every cell in the body is essentially identical to every other cell. Apart from that, the discovery undercuts the rationale behind numerous large-scale genetic studies conducted over the last 15 years, studies which were supposed to isolate the causes of scores of human diseases. Except for cancer, the vast majority of genetic samples used in large-scale studies come in the form of blood. However, if blood and tissue cells do not match genetically, these ambitious and expensive genome-wide association studies are to be treated as flawed. There are two angles to this issue. One is, if genome remains identical in diverse tissues, how can they differ cytologically and functionally from one another; and the other is, if genome is different in different tissues, how such variation can occur. Either way, the molecular gene concept cannot explain.

A fundamental nature of chemical molecule is that it cannot lose the properties assigned by its structure. The genome is an exception to this rule also! It appears that it can lose its property as is evident from its behaviour in a dead body. Although the genome is intact at the time of death, the dead body does not show signs of life. What happened to the biological information encoded by its genome? If biological program is encoded by the structure of DNA, how can it lose that information as evidenced by the lack of life in the dead body? There is no scientific explanation for this observed anomaly.

There are also other odd features. Issues like overlap, alternative splicing, and pseudogenes are chemically inexplicable. "Pseudogenes are similar in sequence to normal genes, but they usually contain obvious disablements such as frameshifts or stop codons in the middle of coding domains. This prevents them from producing a functional product or having a detectable effect on the organism's phenotype…. The boundary between living and dead genes is often not sharp. A pseudogene in one individual can be functional in a different isolate of the same species… and so technically is a gene only in one strain…. there are other pseudogenes that have entire coding regions without obvious disablements but do not appear to be expressed…. Ultimately, we believe that identification of genes based solely on the human genome sequence, while possible in principle, will not be practical in the foreseeable future." (Snyder and Gerstein, 2003).

The variation observed in the use of triplet codes among organisms is another issue. Like the pseudogene this aspect is against chemical fundamentals. The degenerate nature of the biological code implies several triplets coding per amino acid. Further, two amino acids have only one mRNA codon each; AUG for methionine and UGG for tryptophan. Hence 59 degenerate triplets code 18 amino acids; these 18 have two to six synonymous codons each. Choices between synonymous codons are not random; some codons in the set specific to an amino acid are used more than the others (Grantham, 2001). The 'genome hypothesis' which tries to explain the variation in codon use states that codon use is species-specific, i.e., each genome or type of genome shows a particular pattern of choices between synonymous codons. Thus overall codon usage differs between taxa; but codon bias is also influenced by other factors like gene length, gene expressivity (the amount of protein made per gene), environment and lifestyle of the organism (Grantham, et al., 1981). The codon bias gives rise to the paradox whether protein evolution determined DNA sequence or DNA commanded protein evolution. Many such dilemmas remain in molecular evolution. The origin of bias in codon and anticodon frequencies continues to elude researchers (Grantham, 2001).

All these anomalies in one way or the other question the scientific validity of the material gene (genome) concept. The observations, on the other hand, justify the non-physical nature of the genetic program originally proposed by Wilhelm Johannsen (Johannsen, 1911).

What we know in chemistry is that the chemical information is encoded by the molecular structure and it is this information that is deciphered in terms of the physical and chemical properties of the substance. The specificity and stability of the properties of a molecule under a given set of environmental conditions are determined by the chemical structure. There is no material in nature that shows departure from these chemical fundamentals. On the other hand, the genetic program (biological information) is dynamic in nature as evidenced from the variable phenotype it produces with time. It is this dynamic information that is superimposed over the chemical information encoded by the structure of DNA in the molecular gene concept. There is no chemical structure in nature that shows dynamic properties with time or under similar environmental conditions. The superimposition of dynamic biological information on the DNA structure cannot be scientifically justified as it violates the structure-property constancy. Take the case of a butterfly egg.

It has a genome. Even if the genetic information encoded by the genome is taken for granted, the entire structure can have specific biological information commensurate with its structure. But what is observed is that it produces constantly changing phenotypes with time and even develops totally different biosystems. The larva and butterfly developed from the same genome in the egg are two different biosystems in their own right. How is it possible to explain evolution of two different phenotypes from the same genome without sacrificing chemical fundamentals? On the other hand, recognition of independent existence of the genetic program as stored information like the software of a computer would enable us to explain every biological phenomenon as manifestation of the execution of the instructions in the sequence specified in the biosoftware.

3. Computer Model of Organism

Living organism can be best described as natural biocomputer or more precisely natural biorobot (particularly animals as they have well-developed brain, sensors, etc.). The computer analogy permits us to vividly comprehend the most complex and mysterious phenomenon called life. An organism has visible biohardware and invisible (non-physical or intangible) biosoftware. The cell, the basic unit of a living system, is a biochip. The structures in the cell (organelles and nuclear structures including DNA), tissues and organs at the level of the organism constitute the biohardware. Appropriate biohardware (cell structures) including DNA is produced in the cell in accordance with the biosoftware for its execution.

In computer parlance the biosoftware is an integrated program or sets of instructions in the right sequence for the development of the organism, execution of various bioprocesses, its behaviour, instincts, habits and every other task performed by the organism. The bioprogram is the biological information, the driving force of life. The nonphysical gene concept proposed by Wilhelm Johannsen implies that biological information is not coded in the form of a molecule. It has no chemical structure or visible features and is comparable to computer program. Today in the era of computer technology we know the information stored on a computer hard disk exists in nonphysical form. The nature of genetic information can also be understood similarly. The software or information stored on a computer disk does not form part of the chemical structure of the memory device. But it requires a physical medium (e.g., disk) for its storage. Similarly, the biosoftware is nonmaterial but requires a physical medium for its storage.

The storage device (biomemory) of the cell is the chromosome. It is however not possible to pinpoint which constituent of the chromosome acts as the storage medium. Chromosome may be described as the hard disk of the organism. Information the human brain stores in the memory can also be viewed similarly.

Based on the computer model of the organism, the phenomenon of life can be defined as the manifestation of the execution of the biosoftware stored on the chromosome. Death can be viewed as the state of a computer system without software. In other words, death of an organism occurs when the biosoftware is deleted (irreversibly lost) from its body cells. A dead body is therefore like a computer without software. The non-particulate nature of biosoftware and its deletion from the body cells will explain why life cannot be restored to a dead body and why dead cells cannot be cultured. The non-molecular biosoftware also explains why a dead cell does not show any sign of life although genome is intact in it. Computer, robot and other systems that run on man-made software represent forms of artificial life.

3.1 Bioinformation Storage in the Cell

Assumption of the chromosome as the storage device of the cell implies that biosoftware as well any other biological information is stored on the chromosome in discrete packets, which may be referred to as biomemes. The term 'meme' was introduced by Richard Dawkins to mean 'replicator' (Dawkins, 1976). However the term is used here not with the connotation of a "replicator" or with the other characteristics originally assigned to it. 'Meme' is used here to denote piece of information in the abiotic and biotic segments of the universe. The meme relating to biological information may be referred to as 'biomeme'. A biomeme is a set of instructions in the right sequence to perform a task, which may be any biological function including development of cell structures (hardware). The totality of biomemes in the cell constitutes the biosoftware of the organism. Similarly the totality of hardware components constitutes the so-called phenotype. The biomemes may be assumed to have been stored on the chromosomes in specific sectors (Fig. 1). A sector on the chromosome may be storing one or more biomemes. Therefore change in the position, deletion, addition or swapping of chromosome sectors can lead to change in semantic content and hence the biosoftware of the organism.

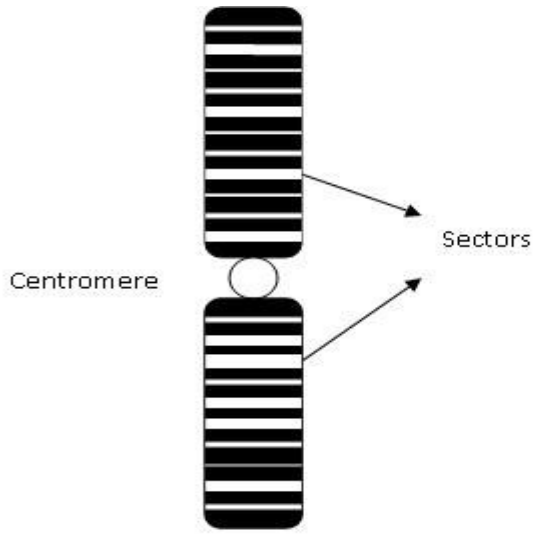

Fig. 1. Organization of biomemory as sectors on the chromosome.
Note: A sector may be storing one or more biomemes

Chromosomal aberrations, swapping of chromosome sectors (crossing over) during meiosis, etc., considered by biologists as errors or mistakes are in fact natural biosoftware engineering mechanisms.

Viewed in the light of computer model of the organism, the DNA technology is mere manipulation at the level of hardware. It is precisely hardware technology and not biosoftware technology. Biologists are erroneously pursuing a chemical trail to find the source of biological information. In the process they superimpose biological information over chemical information – the chemical structure (e.g., DNA). In computer parlance, the genetic modification (alteration of DNA) biologists carry out is like changing the typeface of an electronic typewriter connected to a computer. When a typeface is changed it will print most of the time a wrong word with the changed typeface although occasionally it may print a meaningful word. In other words, we are presently trying to find hardware solution for software problem. This will explain why in spite of concerted global effort in genetic engineering, nothing substantial could be achieved so far.

3.2 Evidence for Non-Molecular Nature of Biological Program

In 1970 Miroslav Radman discovered that the phenomenon of mutation is cell-directed. He found that bacteria harboured information to make mutations (Chicurel, 2001). In 1988 Cairns *et al*. confirmed that genetic mutations are induced from within the cell. They found cell-induced changes of various elements of the lac operon in *Escherichia coli* bacteria (Cairns et al., 1998). According to Chicural, "…depending on their environmental conditions, bacteria might be able to direct mutations to particular genes….Outraged, a number of evolutionary biologists quickly embarked on their own studies to test the notion" (Chicurel, 2001). Goodman described the studies conducted by Joshua Lederberg at the University of Wisconsin which showed that mutations for resistance to some antibiotics occurred spontaneously in cells that had never been exposed before to the antibiotics (Goodman, 1992). Resistance of *Plasmodium* parasites that cause malaria to chloroquine drugs is a typical example (Gelb and Hol, 2002). A more recent report of resistance of bacteria to antibiotics also provides evidence of cell-induced mutation (Kohanski et al., 2010). Commenting on the work, Martin Enserink writes: "Traditionally, the development of antibiotic resistance – a big and growing problem in medicine – has been seen as a passive phenomenon. Haphazard mutations occur in bacterial genomes, and bacteria randomly swap genetic elements.

Every now and then, a mutation or a bit of newly acquired DNA enables the microbes to detoxify antibiotics, pump them out of the cells, or render them harmless in another way. When these microbes are exposed to antibiotics, natural selection will allow them to outcompete the ones that aren't resistant. But in the past 6 years, a different view has emerged…. Researchers have discovered that mutation rates in bacteria sometimes go up in response to stress, in some cases promoting resistance." (Enserink, 2010).

Reviewing the works in this area, Pennisi remarked: "Genetic change, and hence the evolution of new species, is commonly thought to result from small, random mutations in individual genes, but a growing wealth of data emphasizes that the perception is wrong. Indeed the mutations leading to evolutionary change can involve the wholesale shuffling or duplication of the genetic material, changes that can affect the expression of genes or free up duplicated genes to evolve new functions.

What's more, these changes may not be totally random….mainstream biologists need to consider genomes, and the kinds of evolutionary changes they undergo, in a much different light." (Pennisi, 1998).

These cases support the non-molecular biosoftware concept. The results of these studies, if viewed in the light of non-molecular program existing within the cell, would indicate that mutation of DNA occurred as per the directive originating from within the cell and is an intracellular event and not caused by a mutagen external to the organism. Secondly the changes that occur in DNA should be seen not as change in genetic program but a change in biohardware to execute the programs necessitated by the new situation faced by the organism concerned.

Results obtained in several other studies can also be explained the same way. For instance, the observations made by Grant and Grant of the changes in beak size of Darwin's finches (bird species) (Grant and Grant, 2002) can be explained as environment-induced biomemetic change and not as evolution as the authors explained. They studied two predominant species namely, *Geospiza fortis* (medium ground finch) and *G. scandens* (cactus finch). The main food items of the birds were seeds, flowers, etc. The former had a bigger beak and could crack larger and harder seeds whereas the latter had a smaller beak and hence was more efficient in handling smaller seeds. Their results indicated that mean body size and beak shape were significantly different in both species at the end of a thirty-year experimental period.

The changes in beak size occurred depending on the kind of seeds available to them in a changing environment influenced by drought etc. The environmental changes acted as switches to bring specific biomemes into operation and as a result beak size altered to suit the new environment. The changes in morphological characters observed cannot be considered as random phenomena but are biosoftware-directed changes to counter specific environmental stress.

Besides the environment-induced responses, there are many kinds of DNA repairs and natural biosoftware engineering mechanisms. Rosenfeld gives a detailed account of the self-healing strategies of the master molecule, DNA. If a base is put in wrong place during replication, there are enzymes to correct the mistake. Purines, without any errors and without any damages drop out by the thousands every day presumably due to wear and tear of existence in the chromosomes only to be promptly replaced by insertases. A base can spontaneously undergo change.

A cytosine, for example, will lose an amino group and become uracil. Uracil is perfectly at home in RNA but not in DNA. The enzymes called uracil glycosylases recognize the uracil, remove it and replace it with a new cytosine. Suppose that an error has occurred in one of the DNA strands say, a T has been put across from a G, where a C really belongs. This would give rise to two strands one with a G and the other with a T. The enzymatic apparatus 'knows' that cannot be correct, but how does it know whether to replace the C with a T on one strand, or the C with an A on the other? If the replacement takes place not on the right strand, the result would be either death of the cell or a mutation. How does it know which is the authentic original? (Rosenfeld, 1981). But still the question of how a chemical structure (DNA) is *aware of* the change in its composition or how the wrong one is corrected cannot be explained by the molecular gene concept. DNA repair is a true reflection of the existence of the biological program independent of the DNA structure.

From the foregoing, it is clear that biological program exists in the cell itself independently of any structure (e.g., DNA) to bring about necessary changes in hardware at times of need. However, these reports are treated by biologists as heretical as they go against the current molecular genome concept. In all these cases depending on the stimuli or signals (abiomeme) received from the environment specific biomemes, if available, are triggered into operation concurrently modifying or producing appropriate hardware (which includes DNA also). These cases confirm the availability of biomemes in organisms to counter environmental stresses including resistance development.

Failure of synthetic genome to come to life was also reported recently (Gibson et al., 2008; Gibson et al., 2010). This constitutes the experimental evidence against the belief that genome represents the biosoftware of organism and proves instead the nonmaterial existence of biosoftware.

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