

## CHAPTER 4 - DEVELOPMENT OF LIFE AFTER ITS ORIGIN: How Did Living Things Get the Way They Are?

Science has to do with things we can test in the present. For instance, we can use microscopes to observe how cells maintain themselves and how they reproduce. However, the question of how they became able to do such things is not testable. The best we can do is come up with models based on initial complexity versus initial disorganization to see which seems more reasonable.

### I. REVIEW OF DNA AND GENETIC INFORMATION.

At its most basic level, the study of life depends on the study of cells. The reason a cell is human, plant, any other kind lies in DNA (deoxyribonucleic acid), found in every living thing.

DNA is the most efficient information storage system known. It is composed of millions or billions of chemical units that form strands that each have a double helix shape, like the threads on a screw. It is divided into smaller segments called *chromosomes* (Greek for “colored bodies”), which become visible during cell reproduction. These determine which characteristics are passed on from parents to offspring.

Each chromosome is divided into thousands of *genes*, which contain information needed to produce the structures of the cell. *Homeotic* genes (e.g., those that produce hearts, lungs, and so on) are crucial to life and are the same in every individual of a particular type. *Alleles* are genes that exist in multiple forms, such as the genes for hair and eye color. Many genes contain the information needed to produce a specific protein, while others serve as regulators for the cell’s activity.

Genes are made up of large groups of four molecules called *nucleotides*. (They occur in pairs, so the term *base pairs* is also used.) The nucleotides are adenine, cytosine, guanine, and thymine, represented by the letters A, C, G, and T. Each nucleotide consists of a base, a phosphate (PO<sub>4</sub>), and a 5-carbon-atom sugar known as *deoxyribose*.

The nucleotides are used over and over in different sequences, which are unique to each type of organism. The arrangement of A, C, G, and T conveys instructions to the cell, in a way similar to Morse code, which can spell out any message using dots, dashes, and pauses. (For example, dot-dot-dot dash-dash-dash dot-dot-dot is Morse Code for “SOS”).

A strand of DNA can contain dozens of chromosomes, thousands of genes, and hundreds of billions of base pairs.

### II. ORIGIN OF NEW FEATURES IN LIVING ORGANISMS.

#### A. INITIAL COMPLEXITY.

Initial complexity leads us to expect that the first representatives of every major type — not necessarily any modern species, but probably recognizable as the same genus — had their ordinal characters (those things that identify a dog as a dog, a cat as a cat, etc.) from the very beginning. They possessed all the genetic information the type has ever had. Since then, the genes have been spread around enough that any one individual may not possess all of them. Specialization may have occurred because of factors such as natural selection, but no new features have been added. The total information in the gene pool has not increased from the amount which was present in the first specimens.

Mutations (copying mistakes in DNA reproduction) should not add new features because they do not add to the information in the gene pool. They should be harmful or, at best, neutral to the survival of the type.

#### B. INITIAL DISORGANIZATION.

Initial disorganization leads us to believe that the first living things were very primitive single cells. They would have been composed of proteins but would not have had any sort

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of information storage system such as DNA. Unknown processes must have furnished some mechanism to allow them to reproduce. Perhaps this mechanism later changed into a single stranded information storage system like RNA by unknown processes, then still later changed to double stranded DNA.

Living things descended from the first cell must have periodically gained new features (lungs, eyes, bones, wings, feathers, brains, etc.) as their DNA experienced mutations. Though most mutations are harmful, a significant number of them would have had to be beneficial in order for these structures to appear for the first time. Thus, the first identifiable representatives of each major type (e.g., dogs and cats) would have had far less genetic information than the more highly evolved modern representatives.

### C. ACTUAL OBSERVATION - EFFECT OF MUTATIONS.

Since DNA is an information storage system, a simple analogy can illustrate the effect mutations have on it. Suppose we arrange some letters to say

In the beginning God created.

Could we replace one letter or space at a time to change this phrase to make it say

For God so loved the world ?

Of course we could. Many believe that copying mistakes in DNA produced new information in this way, one “point mutation” at a time. However, let us add a reasonable constraint. Starting with the first phrase we will replace one letter or space at a time, but the whole thing *has to make sense*, that is, it has to have at least minimal function, every step along the way. How far will we get now? Not very. We may get something that makes a little less sense such as “In the beginning God *cheated*” - the equivalent of a mutation in a nonessential part of the DNA - but before long, we will have a nonsense phrase corresponding to a mutation in a homeotic gene and will be unable to go any farther.

In a similar way, if we start with a functional organism and make changes in its DNA, we have to have something that makes biological sense every step of the way. Mutations in non-critical alleles may produce unusual characteristics (a new color of rose, for instance) or may not be visibly expressed, but a mutation in a homeotic gene will cause the DNA not to make sense. It will produce a corpse instead of an evolving organism.

#### 1. NO MUTATIONS KNOWN TO INCREASE GENETIC INFORMATION.

As seen in Chapter Two, (1) information in a closed system has never been seen to increase spontaneously, and (2) information in an open system increases only when more is inserted from outside. Random changes do not increase organization and information, they decrease it.

All a mutation can do is *change* preexisting information. However, in order to produce new structures in an evolving line of organisms, new information would need to be *added* periodically. If the idea of simple-to-complex is correct there should be a continually increasing amount of information in DNA from the simplest organisms all the way up to humans. This is not the case.

#### 2. NO MUTATIONS KNOWN TO BENEFIT A SPECIES.

Evolution is supposed to occur not at the level of individuals but of species. Despite occasional benefits to individuals or small groups, no one has documented a single mutation that gave a *species* an overall survival advantage. Nevertheless, a few mutations seem to convey an advantage to affected individuals.

(1) A certain mutation in the millimeter-long worm *Caenorhabditis elegans* (*C. elegans* for short) lengthens its life span from the normal 18 days to about 42. However, its rate of reproduction is less than its unmutated relatives, so it is questionable whether

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the change gives an overall benefit to the species (van Voorhies et al., 2006)

- (2) Some of the aquatic birds on the Galapagos Islands have small, poorly developed wings. This is believed to be the result of a mutation. The change allows the affected individuals to be better swimmers and divers, but they are unable to fly. Their underdeveloped wings are an advantage only in this one environment. Anywhere else in the world, they would be at an extreme *disadvantage* (Cross et al., 2017).
- (3) The best known seemingly beneficial mutation gives its human victims immunity to malaria. However, this is just a side effect. The mutation brings about a change in the shape of hemoglobin, the substance that enables blood to carry oxygen throughout the body. The result is sickle cell anemia (Mayo Clinic, n.d.).

Each red blood cell contains millions of hemoglobin molecules. Since the sickle cell mutation changes the shape of each one, the normally round blood cell assumes a sickle shape. The blood cells clump together in the blood vessels, cutting off oxygen to many parts of the body and killing millions of other cells.

An individual who inherits the sickle-cell mutation from both parents will probably die a slow, painful death. If he gets a sickle-cell gene from one parent and a normal hemoglobin gene from the other, he will have only a mild case of sickle-cell and need not worry about catching malaria. At what price? Sickle cell is incurable. As dangerous as malaria is, it can be cured. Sickle cell is passed on to the next generation. Malaria is not.

This is not raw material for evolution. The individual may receive a slight benefit, but the species suffers.

### 3. **UNPREDICTABILITY OF NUMBER OF BASE PAIRS.**

The simplest known living cells have one or two million nucleotides in their DNA; many creatures considered simple have many millions or a few billion; insects have hundreds of millions; birds have about a billion; reptiles have one or two billion; most plants have anywhere from hundreds of millions to several billion; humans have more than three billion. Since humans are believed to be the most highly evolved species, we might expect the numbers to stop there. They do not. Sharks and frogs have about four billion; newts and some varieties of beans have about thirty billion; and some lilies have almost a hundred billion. There is no pattern of simple to complex.

A question of interest in biology classes is whether all this additional genetic information could have come from mere substitution of one nucleotide for another during reproduction. Something would have had to repeatedly insert large numbers of additional nucleotides into the DNA of evolving organisms, resulting in tens of thousands of times as much genetic information in some creatures as in their hypothetical primitive ancestors.

### 4. **DUPLICATION OF GENES AND CHROMOSOMES.**

Every so often during cell reproduction, an extra copy of a functional gene may be inserted into the DNA. Since the original gene was working properly, the new one is not needed. Some believe that new features evolved over hundreds of millions of years as the duplicates underwent copying mistakes that produced radically new features such as. Of course, the process would have to repeat countless times to produce the rest of the circulatory system as well as the lungs, brain, skin, eyes, ears, bones, fins, arms, legs, wings, claws, kidneys, glands, and so on.

Such a belief requires that duplication must have introduced a great deal of new genetic information in the course of damaging what was already there. However,

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medical science has found that duplicate DNA segments are harmful rather than beneficial, as in Down syndrome (extra copy of chromosome 21), Warkany syndrome (chromosome 8), Patau syndrome (chromosome 13), and Edwards syndrome (chromosome 18) (Clancy & Shaw, 2008). Though a few individual mutations seem to convey an advantage to individuals as seen above, it is doubtful whether a single duplication of either a gene or an entire chromosome helps either an individual or a species.

Another factor to consider: “higher” living things require many more elements than just the carbon, hydrogen, oxygen, nitrogen, sulfur, and phosphorous found in some single celled organisms. While mutations and duplications in the animal kingdom were producing new structures, they would also have to modify the affected organisms to require other elements such as iron, calcium, sodium, copper, molybdenum, manganese, magnesium, silver, and many others. Since members of the animal kingdom are unable to extract these elements from the soil, there had to be parallel mutations or duplications in plants to make them go out and get the elements the animals would need before they evolved enough to depend on them for survival.

Scientific observation through the centuries shows that mutations and duplications do not produce new structures for the first time; they damage what was there already.

#### **D. SUMMARY OF EFFECTS OF MUTATION.**

The idea that living things could have gone from simple to complex by random mutations in DNA is not supported by scientific observation. Rather than increasing pre-existing genetic information, mutations cause damage to it.

### **III. ERROR-CORRECTING MECHANISMS IN DNA REPRODUCTION.**

Since the gases used to test the Oparin-Haldane Hypothesis for the origin of life do not include phosphorus, there is no provision for the origin of DNA. However, at least in the present, life is impossible without it. It has at least four major functions:

- DNA specifies the proper way to link thousands or millions of amino acids into proteins in order to construct all the other parts of the cell. Every group of three nucleotides (a triplet) used to construct the parts specifies the placement of one amino acid. Because each amino acid has a specific 3-dimensional shape, proteins likewise have very precise shapes determined by the structural information encoded in DNA.
- DNA furnishes the information to link those proteins together into a complete cell and repair any damage in the parts.
- Many segments of DNA do not code for proteins but contain information to perform regulatory functions instead.
- Not only does DNA contain the genetic information necessary to copy itself, but it also has so much extra storage capacity that it can produce an entire kind of living things.

Certain genes (*homeotic*) are necessary for an individual to simply remain alive. Other genes, though, occur in multiple variations called alleles. There are numerous locations on the DNA where these alleles are stored. Though it is unlikely that any modern individual possesses every possible allele of every gene, DNA has enough storage capacity that the original ancestors of each kind could easily have carried the information to produce every variant within the kind.

#### **A. INITIAL COMPLEXITY.**

Since life came into existence in a complex, fully functional condition, it is likely that cells will contain some sort of error-checking mechanism to minimize copying mistakes.

#### **B. INITIAL DISORGANIZATION.**

We saw previously that proposals for the origin of life such as the Oparin-Haldane

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Hypothesis deal with the formation of proteins only, with no explanation for the mechanism that would have allowed the cell to reproduce. Whatever this unknown mechanism was, it is believed to have eventually changed into something like RNA as a result of copying errors. Still later, the RNA is believed to have evolved into DNA by yet more errors.

Since a cell is the result of a long series of copying errors, they are a normal part of the simple-to-complex process of evolution. There would have had to be millions of such errors adding to the genetic information of evolving organisms, so preventing them would probably not be a high priority in the cell.

### C. ACTUAL OBSERVATION.

For a computer animated view of what continually goes on inside living things at the molecular level, go to [https://youtu.be/X\\_tYrnv\\_o6A](https://youtu.be/X_tYrnv_o6A) .

DNA guides the reproduction of every part of the cell, including itself, in meticulous detail. The process of cell reproduction occurs in one of two ways:

- (1) *Mitosis*, in which the cell divides into two “daughter” cells that each contain the same number of chromosomes as the parent cell. Most cell reproduction follows this pattern, in which the proteins of the parent cell are divided between the daughters. If one gets less, it manufactures what it needs using the information in its DNA. Unless a mutation (a copying mistake) occurs, the DNA of each daughter is identical to that of its parent.
- (2) *Meiosis*, in which there are two different parents. Each furnishes a *gamete*, a special reproductive cell containing only half the number of chromosomes found in the rest of the parent’s cells. When two gametes unite during sexual reproduction a new single-celled organism (a *zygote*) is formed. It has some DNA from each of its parents. This cell then reproduces by mitosis. Its daughters and their daughters repeat the process over and over until the organism develops into a recognizable specimen of the same kind as its parents. It exhibits some visible characteristics of each of them, and perhaps a few from grandparents and earlier ancestors.

During the process of sexual reproduction, the new organism receives all the homeotic genes from its parents. It also receives many of their alleles. Some of these may be dominant (for instance, the gene for brown eyes), while others may be recessive (blue eyes). If the organism receives at least one dominant allele in any of the variable areas, that trait is visibly expressed. Only if it receives a recessive gene from both its parents does it express the recessive characteristic. (For example, a child has blue eyes only if it gets the blue-eye allele from both parents.) Thus, many recessive genes may be present but not expressed in the individual. They may show up in future generations as “throwbacks.”

#### 1. EFFECTS OF MUTATIONS.

Depending when and where a mutation occurs, its effects may be trivial or they may be fatal. Even a single mutation in a key gene may cause extremely deleterious or lethal effects, e.g., sickle cell anemia, Tay-Sachs disease, muscular dystrophy, cystic fibrosis, congenital deafness, familial hypercholesterolemia, hemochromatosis, Huntington’s disease, and so on (Genetic Alliance, 2010).

- *Mutation in an adult*. Only those cells descended from a mutated cell inherit its altered DNA. If a mutation occurs in a non critical area in an adult, it may either not have much of an effect on the organism (neutral), or else it may lead to a disease such as cancer. However, if the mutation occurs in a reproductive cell it may be passed on to offspring.
- *Mutation in early stages*. A mutation in a homeotic gene during meiosis in the

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parent, or in the early stages of embryonic development, will almost certainly be harmful or fatal because it will be copied throughout the organism. A mutation in a nonessential gene may produce an odd characteristic or may have no visible effect, though it may be expressed in future generations.

Why do we not see a great many more mutations than we do? Because at least three mechanisms in the cell serve to prevent mistakes and correct them on the occasions when they do happen.

## 2. OPERATION OF ENZYMES.

Cells require a great many chemical processes to go on continually, but it is not enough for these processes to occur at the rate they would if left to themselves. In order for the cell to stay alive, the reactions need to take place far faster. There must be some sort of *catalyst*, a substance that speeds up chemical reactions without being changed itself. (Like a matchmaker who keeps getting her friends together yet never gets married herself.) Some catalysts such as platinum work passively by simply furnishing a surface for other chemicals to come together and react. Other catalysts actively capture the atoms or molecules they need and then force them to do a specific action.

The types of catalyst used most often in cells are special protein molecules called enzymes, some of which make reactions occur millions of times faster than they do on their own. Enzymes serve one of two functions, either putting things together or else taking them apart. Each enzyme is extremely specific in its operation, usually speeding up only a single chemical reaction.

Enzymes are so small - only a few millionths of an inch across - that they can manipulate individual atoms as needed. An enzyme is able to perform such a specialized function because, like all protein molecules, it has a precise 3-dimensional shape determined by the arrangement of the amino acids that compose it. It reacts only with substances that have shapes which complement its own at certain key contact points.

As an example of how enzymes work, consider the enzyme chymotrypsin. This molecule consists of about 4,000 atoms, all in specified position, arranged into 241 amino acid links. Dressler and Potter describe the way it operates:

“The fitting portion has a kind of springy atomic jaw that aligns the target link; the process is referred to as a lock-and-key mechanism. Chymotrypsin usually catches the charged end of the protein thread and severs the long thin chain. The parts move off, the spring resets and the unchanged enzyme molecule can act again.”  
(Morrison, 1992, 114)

This is only one of the thousands of enzymes in living organisms. Some help manufacture proteins and other parts of the cell, including copies of themselves. For instance, when DNA needs to reproduce the double helix is first “unzipped” by helicases. Then Type 1 topoisomerase cuts one of the DNA strands into shorter pieces to prevent tangling of the twin strands. It untwists, then Type 2 topoisomerase prevents the two halves from winding around each other. DNA Ligases reconnect the separate strands which are no longer coiled (Sarfati, 2018, 24-26). A number of other enzymes known collectively as DNA polymerases then use the two halves to make two complete new copies of the DNA. These enzymes maintain at least three levels of error-checking and ensure amazing accuracy in the copying process.

### a. Nucleotide Selection.

The double strands of DNA are complementary to each other, much like a film negative and the photograph made from it. They both contain the same information,

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but one is the opposite of the other. Wherever an “A” occurs on one half of the DNA, a “T” is found on the other, and vice versa; likewise, “C” matches with “G.”

These types are complementary because of matching numbers of *hydrogen bond* sites. A hydrogen atom consists of a proton with an electron free to move around it. This arrangement changes when the atom attaches to other elements to form compounds. In many cases the electron is drawn strongly to the other atoms, leaving hydrogen’s proton exposed and resulting in the compound having a positive end. The exposed proton can then be attracted to other atoms that have an excess of electrons, forming a hydrogen bond.

In DNA, “C” and “G” link up with each other because of hydrogen bonds at three matching positive and negative sites, while “A” and “T” link together at two sites. (In RNA, “U” takes the place of “T,” but still matches with “A”.) The hydrogen bonds between the paired nucleotides keep the two halves of the DNA strand together. However, during cell reproduction helicase enzymes (tiny motors) overcome these bonds and “unzip” the two halves of the DNA. Each half then acts as a template for one of the DNA polymerase enzymes to make two complete DNA strands out of the one that split in two. It selects the appropriate nucleotide from those available nearby and inserts it into the proper place on the newly forming strand. Because of the complementarity of A to T and C to G, the enzyme is usually able to select the appropriate nucleotide. The wrong one is inserted only about 1 in 100,000 times.

**b. Preliminary Proofreading.**

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The next step of DNA reproduction uses an enzyme known as *proofreading exonuclease*, named by discoverers Douglas Brutlag and Arthur Kornberg of Stanford University 1972, 241). Because of the physical structure of each nucleotide, the linked pair of “C” and “G” are the same distance across as the “A” and “T” pair. This ensures that a correctly formed DNA strand is a constant diameter. However, if a mismatched nucleotide is inserted into the newly forming strand (an “A” with an “A,” “C,” or “G,” a “C” with an “A,” “C,” or “T” and so on), it changes the diameter of the strand at that point and slows down the addition of the next nucleotide. When this occurs, the proofreading exonuclease removes the offending nucleotide and tries again to insert the one that fits. This reduces the frequency of errors to about one in 10 million.

**c. Final Error Detection and Correction.**

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Finally, other DNA polymerases reread the new double strand of DNA to make sure there are no mismatches. If they find one, they remove the defective segment on the new half, then repair it by rereading the segment on the original and remanufacturing and inserting a corrected, properly matched segment on the copy. This brings the rate of copying errors down to about one in 10 billion (Radman & Wagner, 1988, 40-46).

The method by which enzymes determine which half of the double strand is new and which is old is not well understood. If various groups evolved from common ancestors, they should all use similar systems, yet it seems to vary from one major type of organism to another. Different types “know” in different ways and make corrections only on the defective newly formed half.

These elaborate mechanisms in every cell do not *cause* evolution. They *prevent* it by almost totally eliminating copying mistakes. The whole system works because DNA

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produces the enzymes needed to produce correct DNA, but it requires the enzymes to be present from the beginning in order to make the DNA that makes them. This is an irreducibly complex system, all of whose parts have to be present at the same time for it to work at all.

- Those who believe life has gone from simple to complex believe that the enzymes and error correcting mechanisms are the result of nothing more than a long string of accidents.

Since the first living things are supposed to have used some unknown information storage system far less complicated than DNA, the DNA and all its features would have had to evolve later through copying mistakes. Those who believe that life evolved through purely natural processes must believe that the mechanisms that serve to prevent and correct errors are the result of a series of thousands or millions of errors that were *not* prevented and corrected.

- Those who believe that God directed a process whereby life gradually developed from simple to complex (evolution) may believe that (1) He created life then added the error-correcting mechanisms later, or else (2) He placed the mechanisms in living things from the beginning then overrode those same mechanisms millions of times to make them evolve from simple to complex.

#### **D. SUMMARY OF ERROR CORRECTING MECHANISMS.**

DNA reproduction and error correction work the same in every type of cell ever studied, from the simplest bacteria to the most advanced higher organisms. They all seem to have had this complex system from the beginning, rather than developing it gradually as they went from simple to complex.

#### **IV. SEXUAL VS. ASEXUAL REPRODUCTION.**

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Since bacteria are so numerous, it is safe to say that most living things reproduce asexually. However the number of types that reproduce sexually is greater than the number of types that do not. All mammals reproduce sexually, as do most vertebrates and many invertebrates.

In order for sexual reproduction to have evolved, there would have had to be a great many mutations slipping through the error correcting mechanisms so that some members of some ancient types of single celled organism would have gradually accumulated male characteristics such as the ability to produce sperm, while others would have had to accumulate female characteristics such as the ability to produce eggs. However, there are no candidates for the ancestral organisms.

#### **V. PROTEIN MANUFACTURING - UNIVERSAL GENETIC CODE.**

Cells are composed mainly of proteins, though there are thousands of different types of protein.

##### **A. INITIAL COMPLEXITY.**

Since even the most complex cells need to take in resources, they should be made of the same basic types of structures, e.g., proteins, as their food sources. The processes of manufacturing those structures should be similar.

##### **B. INITIAL DISORGANIZATION.**

Since DNA evolved long after the beginning of life, its functions might vary from one type of organism to another. In particular, the process of building up structures such as proteins might not be the same in different types.

##### **C. ACTUAL OBSERVATION.**

In order to understand how mutations affect cell structures such as proteins, we need to understand how the information in DNA is converted into these structures.



DNA resides in the interior of the cell. When some part of the cell needs more of a specific protein in order to grow, reproduce, or repair itself, it sends a message to the DNA by means of a chemical such as a hormone. This activates specific types of *ligase* and *helicase* enzymes that interact with DNA. Scientists do not yet understand how the enzymes “know” which section contains the blueprint for that protein (some pattern of the nucleotides A, C, G, and T), yet they find it. The ligases break the hydrogen bonds and the helicases temporarily unzip the DNA segment into its two halves. Various enzymes such as *RNA polymerases* use the exposed DNA segment to manufacture a special form of ribonucleic acid known as *messenger RNA*. mRNA consists of a chain of nucleotides that match with the exposed segment, except that Uracil substitutes for Thymine. (For example, if a section of DNA contains the pattern GAGCTA its corresponding mRNA contains CUCGAU.) When completed, the mRNA molecule is hundreds or thousands of nucleotides long.

The chemical difference between RNA and DNA is that RNA depends upon a sugar known as ribose, while DNA uses a sugar that has one less oxygen atom -- hence, *deoxyribose*. In DNA we can use “base pairs” and “nucleotides” synonymously. RNA, on the other hand, is a single-stranded molecule rather than a double helix. It does not use pairs.

The newly formed mRNA moves away from the area where the DNA is located to the *endoplasmic reticulum* area where the manufacturing is actually done. Once there, it attaches to special bodies called *ribosomes* to maintain its orientation while each triplet of its nucleotides matches up with one *transfer RNA* (tRNA) molecule, also made up of a triplet (or *codon*) of nucleotides. Each tRNA molecule serves a dual function: on one side it attaches to a specific triplet on the mRNA, and on the other it physically attaches to a specific amino acid.

As the first tRNA molecule attaches to the triplets on the mRNA, it precisely positions the first amino acid. As the second tRNA attaches it allows its amino acid to link up with the first. The first tRNA molecule is then released so it can go get another amino acid and get back in line. The third tRNA allows its acid to link with the second, releasing the second tRNA, and so forth. Once the sequence of amino acids is complete the protein is folded into a specific shape by other enzymes and taken wherever the cell needs it. The mRNA and tRNA nucleotides can be reused indefinitely.

Since four nucleotides are used in each of the three positions, there are sixty-four (four times four times four) possible triplet combinations. These code for the twenty left-handed amino acids used in living things. Scientists have discovered that DNA triplets code for the results as shown below:

TGG - Tryptophan	ATG - Methionine
TTT or TTC - Phenylalanine.	TAT or TAC - Tyrosine
TGT or TGC - Cysteine	CAT or CAC - Histidine
CAA or CAG - Glutamine	AAT or AAC - Asparagine
AAA or AAG - Lyseine	GAT or GAC - Aspartic acid
GAA or GAG - Glutamic acid	ATT, ATC, or ATA - Isoleucine
CCT, CCC, CCA, or CCG - Proline	ACT, ACC, ACA, or ACG - Threonine
GTT, GTC, GTA, or GTG - Valine	GCT, GCC, GCA, or GCG - Alanine
GGT, GGC, GGA, or GGG - Glycine	
CGT, CGC, CGA, CGG, AGA, or AGG - Arginine	
TAA, TAG, and TGA - “stop” codes indicating the end of a coding section	

AGT, AGC, TCT, TCC, TCA, or TCG - Serine TTA, TTG, CTT, CTC, CTA, or CTG - Leucine
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(Black, 1998)

Notice that more than one combination may code for the same amino acid. This allows the same segment of DNA to produce more than one structure, depending where the process of decoding starts. And since the two halves of the unzipped DNA strand are read in opposite directions, the redundancy also allows copying in opposite directions to produce the same results.

#### **D. SUMMARY OF PROTEIN MANUFACTURING.**

As nearly as scientists have been able to tell, every type of organism uses the same genetic code and the same manufacturing apparatus, whether the simplest types such as blue-green algae that are supposed to have survived unchanged for hundreds of millions of years, or the most advanced such as mammals. This is what the idea of complex-to-simple led us to expect, but it is extremely difficult to reconcile with the idea of simple-to-complex.

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#### **VI. CHAPTER SUMMARY.**

- There is no known natural explanation for the origin of DNA and the genetic information it contains. Whatever we believe, we have to depend on something outside of known natural processes.
- Mutations have never been seen to increase genetic information.
- Even when some mutations benefit individuals, they harm the affected species.
- The number of base pairs in living things does not follow any sort of pattern of increasing complexity.
- Duplication of genes and chromosomes has been observed to be a harmful rather than a beneficial process.
- There are intricate mechanisms that correct errors during DNA duplication. Those who believe everything must be explained by purely natural processes believe the error correcting mechanisms developed as a result of errors that were not corrected.
- Those who believe some sort of God directed the process of evolution believe He created the error correcting mechanisms then overrode them millions of times in order to make living things evolve.
- There is no known organism showing how sexual reproduction could have evolved.
- Every type of living thing known, from simplest to most complex, uses the same mechanism to manufacture the parts of its cells. They also use exactly the same genetic code.
- These mechanisms of the cell do not show any traces of evolution. Rather than showing development from simple to complex, they appear to have been complex from the time they began.