## CHAPTER ELEVEN

### *Evolution: Biology Says NO!*

We've dealt so far with three major questions and have seen that in every case the predictions of evolution are wrong, while those of creation are right.

(I) Can natural law explain the origin of matter and energy?

Visual #11-1

We saw that matter and energy could have come into existence only through the intervention of someone or something outside of known natural processes. Though one could still choose to believe in random chance, the most reasonable conclusion is that it was an intelligent being -- a creator.

(II) Can natural law explain how matter and energy reached their present condition?

a. If random chance brought matter and energy into existence in a disorganized condition, the laws of nature we know would prevent them from developing to their present state. Those who are committed to making up a purely naturalistic explanation have to invent new laws of nature that contradict many of the observations of science through the centuries.

b. If the God of theistic evolutionists is responsible, He (or it) would have had to intervene in nature an unimaginable number of times to bring matter and energy to their present state.

c. If a supernatural God brought matter and energy into existence in a mature condition, natural law would then be sufficient to bring them to where they are today.

(III) Can natural law explain the origin of life?

The origin of life also requires some influence that cannot be explained by known natural processes.

This leads to our fourth major question:

(IV) Once life began, can natural law explain how it reached its present condition?

We will see that presently observed processes do not create anything new; instead, they weed out defective specimens. Thus, if life began in a complex condition (**one** unexplainable event), natural law is sufficient to explain how it reached its present state. If it began in a disorganized condition, natural law is not sufficient. Evolutionists need to appeal to a great number of unexplainable events.

We should "give the devil his due," so to speak. Much of creationists' knowledge of the mechanisms of biology has developed in response to evolutionary errors and misunderstandings. For instance, until Darwin proposed natural selection as the mechanism to drive evolution, there was not much research into how new species develop within previously existing kinds. Likewise, until evolutionists proposed the existence of "pseudogenes" as evolutionary leftovers, few creationists were motivated to study the functions of some of the more obscure segments of DNA. Though evolutionary theory is consistently wrong, it has forced creationists not to be lazy and has led to many discoveries about how living things work.

## I. THE FOUR PILLARS OF EVOLUTION.

Visual #11-2

Evolution rests on four "pillars": *Embryology*, *Homology* (similar structures in creatures not considered to be close evolutionary relatives), *Biogeography* (geographic distribution), and *Paleontology* (the study of fossils) (Osborn, 1929, 31).

- We already saw that embryonic recapitulation is a fraud. The only time there are superficial similarities between different types of embryos is not at the beginning of development but partway through instead. The first pillar collapses.
- In this chapter we will see that evolution is unable to account for homology.
- We will also see that creation explains geographic distribution as well as evolution does.

Thus, the second and third pillars crumble.

- In the next four chapters we will see that the fossil record argues strongly against evolution.

The fourth pillar falls, and evolution is left standing upon faith alone.

In this chapter we will see that living organisms have built-in mechanisms that *prevent* evolution. If God used evolution, he had to override these mechanisms an incredible number of times. However, if he created life in a complex and fully functional state, the observed laws

of biology are well able to explain how the present array of living things came about. In this chapter we will see that biology shows that evolution *could* not occur; in the next four we will see that the fossil record shows that it *did* not occur.

## II. THE GENETIC CODE.

At its most basic level, the study of life depends on the study of cells. What gives a cell its identity? Why is it an amoeba instead of a human cell, a plant cell, or any other kind of cell?

Visual #11-3

The answer lies in *deoxyribonucleic acid*, or DNA, found in every living thing. DNA is an information storage system made up of four molecules called *nucleotides* (adenine, cytosine, guanine, and thymine, represented by the letters A, C, G, and T) used over and over in different sequences. The arrangement of the nucleotides is different for each type of organism. Even within a single type, the arrangement varies a certain amount from one individual to another.

Each nucleotide consists of a *base*, a *phosphate* ($PO_4$), and a 5-carbon-atom sugar known as *deoxyribose*. (In organic chemistry, anything ending with -ose is some sort of sugar.) The nucleotides are connected in a double helical strand -- the shape followed by the threads on a screw. DNA may contain only a few million nucleotides as in some bacteria, or it may contain billions as in humans and may other living things.

DNA is subdivided into smaller units called *chromosomes*, from the Greek words for "colored bodies." These become visible during cell reproduction. The number of chromosomes varies from one kind to another. Normal humans have 46, some animals have several hundred, and some simpler life forms have only a few. Chromosomes determine which characteristics are passed on from parents to offspring. The chromosomes are in turn divided into thousands of *genes*, which are made up of large groups of the four nucleotides A, C, G, and T. Many genes contain the information needed to produce a specific protein, while others serve as regulators for the cell's activity.

DNA has at least three major functions:

Visual #11-4

(1) It specifies the proper way to link thousands or millions of amino acids into proteins in order to construct and repair all the other parts of the cell. Every group of three nucleotides (a *triplet*) used to construct these 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.

(2) DNA furnishes the information to link those proteins together into a complete cell, as well as furnishing a template to duplicate itself so that future cells will also be able to reproduce.

(3) A strand of DNA has the potential to contain the genetic information to produce an entire kind of living things. There are numerous locations on the DNA where multiple variations of genes may occur. Though it is unlikely that any modern individual possesses every possible variation of every gene, the original ancestors of each kind could easily have carried the information to produce every variant within the kind.

DNA is a digital set of instructions. In much the same way we can convey instructions using the dots, dashes, and pauses of the Morse code (e.g. dot-dot-dot dash-dash-dash dot-dot-dot is Morse Code for "SOS"), DNA uses the nucleotides A, C, G, and T to convey instructions to the cell in which it resides. It tells the cell exactly what to do in order to grow and reproduce.

### A. WHAT EVOLUTIONISTS ARE OVERLOOKING: COMMUNICATION.

In claiming that the intricate information contained in DNA came together by random processes operating on chemicals that themselves were produced by random processes (e.g., the nucleotides in RNA), evolutionists are overlooking a fundamental principal of communication. When we want to convey a message we does not randomly make sounds which we hope will come together and make sense.

Visual #11-5

- We start with an idea.
- Then we decide how to convert that idea into syntax and grammar so that it can be expressed.

- Finally we convert those elements into letters and symbols that convey the idea to others.

Evolutionists, on the other hand, believe that:

- The letters of DNA and RNA (A. C. G. T, and U) came into existence randomly.
- Then they randomly assembled themselves into words and grammar (genes, chromosomes, etc.).
- Finally, they randomly turned into an intricate and fully functional information system.

This is not how communication works. They have the whole process backwards.

We humans have no problem looking at even a simple digital computer program and recognizing that it took intelligence to produce it. Yet some have a problem looking at the most complex digital program ever discovered - DNA - and recognizing that it took intelligence. It's hard to convince someone who has already decided that everything MUST be explained by purely natural processes that maybe some things require an explanation outside of nature.

Visual #11-6

**B. CELL REPRODUCTION AND ERROR-CHECKING.**

Visual #11-7

Each kind of cell has a set of basic instructions in its DNA. For example, human DNA contains some genes that are the same in all normal humans. These are known as *homeotic* genes. They contain the instructions for assembling a normal body with two eyes, two arms, a brain, a nose, etc., in specific places. The DNA also contains other genes that vary from one individual to another, such as those that determine hair color, eye color, shape of facial features, and the like. The variable genes (e.g., blue vs. brown eyes) are known as *alleles*. All the homeotic genes are expressed in each individual, that is, they produce visible results, but only a small percentage of the variable ones are. Nevertheless, they are available to be passed on to future generations, in which they may or may not be expressed.

Some organisms such as bacteria are composed of a single cell which has to perform all the functions of life. Others such as humans consist of billions or trillions of specialized cells (heart, liver, brain, skin, etc.) working together. Whether a cell is a complete organism or just a small part of one, it spends most of its life preparing to reproduce even while performing its other functions. It continually takes in raw materials and uses the information in its DNA as a template to assemble them into new proteins and new DNA. In the process, it gets larger and larger. Finally it splits.

The process of cell reproduction occurs in one of two ways:

Visual #11-8

(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."

DNA guides the reproduction of every part of the cell, including itself, in meticulous detail. A DNA copying error during reproduction produces a defective cell. Depending when and where a mutation occurs, its effects may be trivial or they may be fatal.

Visual #11-9

- *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, 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 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 variable 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.

***1. OPERATION OF ENZYMES.***

Cells require a great many chemical processes to happen continually, but it is not enough for these processes to occur at the rate they would if merely left in a dish of chemicals. In order for the cell to stay alive, the reactions need to take place far faster than they do by themselves. There must be some sort of a catalyst, a substance that speeds up chemical reactions with out being changed itself. (Sort of 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.

Visual #11-10

The types of catalyst used most often in cells are special protein molecules called *enzymes*. These 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, let's 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

Visual #11-11

halves from winding around each other. DNA Ligases reconnect the separate strands which are no longer coiled (Sarfati, 2018). 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.

Visual #11-12

***2. NUCLEOTIDE SELECTION.***

Visual #11-13

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, 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. An isolated hydrogen atom consists of a proton with an electron free to move around it. This arrangement may change 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.

***3. PRELIMINARY PROOFREADING.***

Visual #11-14

The next step uses an enzyme known as *proofreading exonuclease*, named by discoverers Douglas Brutlag and Arthur Kornberg of Stanford University. (Brutlag, 1972, 40-46) 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.

***4. ERROR DETECTION AND CORRECTION.***

Visual #11-15

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.

How do enzymes determine which half of the double strand is new and which is old? This seems to vary from one major type of organism to another, and is not well understood. If various groups evolved from common ancestors, they should all use the same system. Yet 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 al-

most totally eliminating copying mistakes. The whole system works because DNA 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! While one may choose to believe -- by faith -- that this system is accidental, it is an irreducibly complex system that functions as if it were carefully designed.

- Evolutionists believe that the enzymes and error correcting mechanisms are the result of nothing more than a long string of lucky accidents.

Visual #11-16

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. Atheists must believe that the very mechanisms that serve to prevent and correct errors must themselves have evolved by a series of thousands of perfectly coordinated errors that were *not* prevented and corrected. The error-prevention mechanisms evolved because of tremendous numbers of errors that were *not* prevented.

- Theistic evolutionists must believe that God added the error-correcting mechanisms later, then overrode those same mechanisms millions of times because He wanted so much to have evolution.

This is supposed to be state-of-the-art science.

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

Suppose that the first living cell somehow came into existence by the random chemical processes we discussed in Chapter Nine. Could its descendants gain new and advantageous features by accidental copying mistakes? Not likely.

### *1. CHEMICAL COMPOSITION OF DNA AND THE STRUCTURES IT SPECIFIES.*

Visual #11-17

Remember that the Oparin-Haldane hypothesis says the early atmosphere consisted mostly of hydrogen ($H_2$), methane ($CH_4$), ammonia ($NH_3$), and water vapor ($H_2O$). Thus, the only elements available to manufacture the components of cells would be *Hydrogen*, *Carbon*, *Nitrogen*, and *Oxygen*. However, such a mixture could not produce DNA, which requires the element *Phosphorus* as an essential ingredient in the phosphates which hold it together. But including phosphorus in the primordial soup complicates the chemical reactions even more, especially when it comes to getting necessary chemicals into the evolving not-yet-living first cell.

Evolutionists believe that a cell membrane composed of phospholipids spontaneously formed to enclose the proteins of the first living cell. However, such a membrane is almost completely impermeable to phosphates. Thus, the key substances needed to produce DNA would have been unable to penetrate the membrane. Even if they happened to be enclosed when the membrane formed around the proteins, no more phosphates could have gotten in to make more DNA. The cell could never have reproduced.

Even if we ignore this problem and assume that DNA could have formed spontaneously, it would have had a difficult time mutating to code for even "simple" organisms such as algae.

- Two of the amino acids used in cells, cysteine and methionine, require *Sulfur.*
- Even the "oldest" known organisms, stromatolites, are very similar to modern forms that rely on photosynthesis. This process centers around *Magnesium* atoms.
- Likewise, many animals use hemoglobin in their blood to circulate oxygen. Hemoglobin is centered around atoms of *Iron*.
- Other creatures such as the octopus have blood based on *Copper* atoms.

Visual #11-18

- Many animals use other elements such as *Calcium*, *Silicon*, *Selenium*, *Potassium*, *Molybdenum*, *Manganese*, and the like in very important functions. Some of these occur in very small amounts in the soil and are available in only a few types of plants.

According to evolution, mutations in the cells billions of years ago led to the development of the major groups such as animals, plants, bacteria, and fungi. Later, there would have had to be countless mutations within each of these groups to lead to the development of more and more modern types. But why should plants begin to extract from the soil exactly the minerals that animals needed? Unless there were millions of coordinated sets of parallel mutations, animals would never have survived.

- Mutations in the animal kingdom would have made various animals depend on specific elements available only in the soil. However, animals are *heterotrophs* -- that is, they cannot make their own food from the soil but must obtain it from other living things. Since animals cannot process the minerals in the soil they would have had no way to get these elements and would quickly have become extinct.
- Unlike animals, many plants are *autotrophs* -- that is, they **can** make their own food from scratch by extracting minerals directly from the soil. If the story of evolution is true, the correct mutations had to occur thousands of times within specific members of the plant kingdom to make them extract from the soil ***exactly*** the elements the animals needed at ***exactly*** the time the animals needed them.

At every step along the way, both the animals and plants would have to maintain at least minimum function. What an amazing series of mutations! Or could it be that they were designed that way?

### *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, let's consider a few mutations that convey an advantage to the affected individual.

Visual #11-19

(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 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 (Cross, 2017). Their underdeveloped wings are an advantage only in this one environment. Anywhere else in the world, they would be at an extreme *dis*advantage.

(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, undated)

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.

#### *a. Benefit vs. Harm of the Sickle-Cell Mutation.*

Visual #11-20

If an individual inherits the sickle-cell mutation from both parents, he will 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. But at what price? Sickle cell is incurable. Malaria, painful though it may be, can be cured. (Amazon explorers have reported that natives there use certain tree roots to cure malaria in as little as three days.) 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 ben-

efit, but the species suffers.

***b. How Cells Manufacture Protein.***

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

Visual #11-21

DNA resides in the interior of the cell. When 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. Scientists do not yet understand how the enzymes "know" which section of DNA contains the blueprint (some pattern of the nucleotides A, C, G, and T) for that protein, yet they do 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, *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 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, *deoxy*ribose. 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 messenger RNA moves out of the nucleus of the cell 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 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 latches onto a specific amino acid.

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:

Visual #11-22

| | |
|---|---|
| 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 - Lysine | GAT or GAC - Aspartic acid |
| GAA or GAG - Glutamic acid | ATT, ATC, or ATA - Isoleucine |
| 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 | |
| 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 (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 it starts. Anyone familiar with computer programming at the level of machine language would be deeply impressed with the level of sophistication in the coding of DNA.

Back to the protein manufacturing process: as the first tRNA molecule attaches to the triplets on the mRNA, it precisely positions the first amino acid. As the second 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 yet more enzymes and taken wherever the cell needs it. The mRNA and tRNA nucleotides can be reused indefinitely.

***c. Cause of Sickle Cell Hemoglobin.***

Out of the roughly three billion base pairs in human DNA, all it takes is a single copying mistake to produce sickle cell anemia.

Visual #11-23

Both normal and sickle cell hemoglobin consist of about 600 amino acids. The affected part of normal hemoglobin contains the following amino acid sequence:

-valine-histidine-leucine-threonine-proline-**glutamic acid**-glutamic acid-lysine-serine.

In contrast, sickle cell hemoglobin contains the sequence

-valine-histidine-leucine-threonine-proline- **valine** -glutamic acid-lysine-serine.

(Curtis, 1979, 70.) The substitution of valine for glutamic acid causes the problem. Let's see how this could happen.

Since a triplet of DNA nucleotides codes for each amino acid, this means about 600 triplets, or 1800 nucleotides, are used to align the 600 amino acids in hemoglobin. In particular, the crucial glutamic acid molecule above is specified by the DNA

Visual #11-24

triplet **GAG**. A single mistake on the DNA strand, the substitution of Thymine for Adenine, changes this to **GTG** and substitutes valine at the key spot in the hemoglobin molecule (Oak Ridge National Laboratories - http://www.ornl.gov/sci/techresources/Human_Genome/posters/chromosome/hbb.shtml). This slightly alters the shape of each hemoglobin molecule, causing the individual to have sickle cell anemia - all because of *one wrong nucleotide* among the billions on the DNA strand.

***d. Genetic Counseling.***

Mutations have at least one benefit: they create jobs for health care professionals.

The accumulation of mutations in the human gene pool has made it more and more risky to have children. Nowadays a couple considering whether to have a child may seek the advice of a genetic counselor who examines their DNA for mutations to see if children might be born deformed, ill, or otherwise handicapped. If mutations were beneficial to the species there would be no need for such a profession.

If evolutionists really believe that mutations are helpful to the species, we might wonder why they are not clamoring to build homes next to nuclear power plants so they can mutate and have better, more evolved children!

***e. Why Mutations are Harmful.***

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

Visual #11-25

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. This is how many evolutionists believe copying mistakes in DNA produce new information, one "point mutation" at a time. However, let's 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 c*h*eated" - the equivalent of a mutation in a nonessential allele - 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 the same 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.

Since we have never seen a single completely beneficial mutation -- remember that even if they help the individual they harm the species -- it seems unlikely that we could get very far before we reach this point. Remember from our discussion of entropy in Chapter Seven that (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. Evolution *could not occur* by random chance.

Of course, God could have used evolution if He wanted to. But since the Bible says He created everything to reproduce only "after its kind" and since the reproductive mechanisms of cells are geared to prevent change, it is obvious that He did not.

***3. NO MUTATIONS KNOWN TO INCREASE GENETIC INFORMATION.***

All a mutation can do is *change* preexisting information. It would take much more than that to cause evolution! New information would need to be *added* periodically to the DNA of an evolving line of organisms in order to produce new structures. Thus, if evolution 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.

***a. Unpredictability of Number of Base Pairs.***

Visual #11-26

The simplest known living cells have one or two million nucleotides in their DNA; many "simple" creatures 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. If we believe in evolution we might expect the numbers to stop there. They don't. 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. We thought we were the pinnacle of evolution, but the humble lily contains over 30 times as much information as we do! (Maybe that's why Jesus told us to consider the lilies of the field?)

Besides the fact that there is no evolutionary pattern to be found, all this additional genetic information could not 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.

***b. Viruses and Gene Therapy.***

Visual #11-27

The only candidate that might be able to add nucleotides to DNA is not mutation but the lowly virus. A virus is essentially a scrap of RNA or DNA wrapped inside a

protein coating. Some evolutionists point to the fact that viruses insert their genetic contents into a host's DNA, which opens the possibility of increasing the number of nucleotides. But are viruses a plausible source for all the extra DNA in the lily?

It is true that an experimental technique known as *gene therapy* uses specific viruses to treat patients with conditions caused by mutated genes. However, this is done by removing the *virus's* normal genetic material and inserting the desired *human* gene sequence into it. The altered virus is then introduced into the patient's cells in order to add the unmutated gene to his or her DNA. This technique has nothing to do with the evolutionary belief that viruses added DNA to evolving organisms.

- First, it is anything but random. A great deal of planning and intelligence is involved in selecting specific viruses that will not harm or kill the patient.
- Second, it's not the virus that helps the patient, but the gene it carries. The gene is not a normal part of the virus. It has to be deliberately added by careful genetic manipulation at the molecular level.
- Third, this is not a case of a mutation making an improvement. The mutation caused damage, which we try to correct by bringing in the original *un*mutated gene.

If evolutionists really believe that viruses are the source of new genetic information that improves the species, why don't they try to catch them instead of curing them?

***c. Viruses and Bacteria.***

Many people are afraid of bacteria. However, though some types are harmful to humans, the vast majority are not. Bacteria play a crucial role in supporting life on earth.

Visual #11-28

- If they did not help to decompose dead things, we would be surrounded by enormous piles of garbage.
- Many of the gases in the atmosphere (e.g., the fixated nitrogen needed by all known living things) are there as the result of bacteria doing their work of decomposition.
- Likewise, cattle are able to digest grass only because of the bacteria that live in their digestive systems.

Like bacteria, viruses also have a bad reputation because of terrifying images of Ebola and other deadly diseases. However, though some viruses such as AIDS and polio receive a great deal of negative attention, only a tiny percentage of the viruses in the world affect humans. In fact, only a few dozen affect any given species. We will deal later with the topic of whether God created harmful viruses, but our concern here is with the fact that the vast majority of viruses are *not* harmful. Viruses only become dangerous when they "jump species" and begin to afflict a different host than their natural one. Viruses do not harm their natural hosts! In fact, they serve a very useful purpose in bacteria.

Since bacteria reproduce asexually (mitosis only), we would expect each bacterium to have exactly the same DNA as its parent. If this were the case, each individual would be susceptible to exactly the same toxins, making it relatively easy to wipe out an entire kind. However, scientists have now learned that some viruses actually help ensure survival of various kinds of bacteria, by injecting genes that may make an individual bacterium immune to otherwise toxic conditions.

Bacteria contain a certain percent of unchangeable DNA called the *large replicon*, but sometimes viruses enable them to exchange a certain amount of genetic information in units called *small replicons*. A virus is not a living thing but instead consists of a scrap of RNA or DNA (such as a small replicon) inside a coating made of a few protein molecules. Each type of virus has a specific chemical makeup

which causes it to be able to attach to the cells of only certain kinds of hosts. When a virus attaches itself to the correct type of host -- usually only one species, or at most a very few -- it injects its genetic contents and slightly alters the host's DNA. If the host is a bacterium, its slightly modified DNA serves as insurance. If something happens to wipe out all its sister cells, it may still be able to survive and perpetuate the kind, including the new gene from the virus. As far as bacteria are concerned, then, viruses act somewhat like bees pollinating one flower after another, ensuring the survival of the kind.

The reason viruses are harmful to species other than their natural hosts is that the new segment of DNA they inject includes instructions for how to make copies of the virus. As the host cell reads its DNA so as to make the proteins it needs, it also reads the viral segment and makes whatever substance the coding calls for. Since the unsuspecting host has no use for the extra copy of the virus, it tries unsuccessfully again and again to make the desired protein. It accumulates more and more copies of the virus until it finally uses up all its resources and dies. It bursts, releasing many copies of the virus which can in turn affect other cells. Since the viral DNA was not part of the host's original gene sequence, the built-in error checking mechanisms are not equipped to deal with it. Thus, viruses are able to mutate rather quickly.

Remember, our concern is not whether or not viruses are harmful, but instead, whether they could gradually add tens of billions of nucleotides to the DNA of primitive life forms and cause them to evolve to higher and higher types. There are at least two reasons to believe this scenario is impossible.

*i. Limited number of genes inserted.*

No species is susceptible to more than a few dozen specific viruses. It wouldn't matter if there were millions of types of viruses floating around; the viruses could inject at most a few dozen genes into the host's DNA. This may be a self-limiting process anyway. Once a bacterial host has assimilated one virus, it is sometimes altered enough that it no longer accepts other types. Even if every type of bacterial cell on the way up the evolutionary ladder could absorb every type of virus that affected its species, this might add at most a few hundred thousand nucleotides, not the billions that evolution demands.

*ii. Effects limited to asexual reproduction.*

Even if bacteria were able to gain hundreds of genes and pass them on to their descendants, the process would stop as soon as the descendants evolved to the point of sexual reproduction. A multicelled organism reproducing sexually would not pass on any viruses to its offspring, unless the virus happened to have somehow attached itself to the one gamete that happened to unite with the one furnished by the other parent. The odds of this happening even once are infinitesimal, let alone the millions of times that would be needed to keep adding nucleotides to the DNA.

All in all, viruses are not a reasonable candidate to add billions of nucleotides to DNA. Evolutionists have no plausible source for all the extra genetic information that would have had to be added during the course of evolution.

***d. Duplication of Genes and Chromosomes.***

Visual #11-29

If viruses cannot significantly increase the length of the DNA strand, how could it have expanded to billions of base pairs? The other explanation put forth by evolutionists is that the extra base pairs come from duplication of already existing genes.

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. Evolutionists believe that new features evolved over billions of

years as such duplicate genes underwent copying mistakes leading to radically new structures. However, we've seen that mutations do not introduce new genetic information, but instead damage what was already there. Medical science also shows that duplicate DNA segments are very harmful -- 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)

It is quite a step of faith to believe that an extra copy of an already functioning gene could mutate to assume an entirely new function, e.g., producing blood where none ever existed before. Not only that, the process would have to repeat millions of times throughout the course of evolution to produce the rest of the circulatory system, the heart, lungs, brain, skin, eyes, ears, bones, fins, arms, legs, wings, claws, kidneys, glands, and so on.

Remember also that at minimum, living things require carbon, hydrogen, oxygen, nitrogen, sulfur, and phosphorous. While these mutations were producing all the 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. Who needs God when you have mutations?

One can believe by sheer faith that duplicate genes modified by mutation could accomplish all these things, but our observations through the centuries show that mutations do not produce new structures for the first time; they damage what was there already.

### *4. "MUTATIONS" IN BACTERIA AND INSECTS.*

Evolutionists frequently use bacteria and insects as examples of "evolution in action." They tell us that some bacteria have become "superbugs" by evolving a resistance to antibiotics, and that some insects have evolved an immunity to pesticides. This is not evolution at all.

Visual #11-30

Suppose you have a barnyard with machinery set up to automatically care for 1,000 chickens. You put in 950 white chickens and 50 brown ones. Now suppose that someone tampers with the feeding apparatus by putting in a special poison that kills only white chickens. Soon only 50 chickens are left, all of them brown. Since the farm is equipped to feed a thousand, they quickly multiply. Before long the barnyard is populated by 1,000 brown chickens. (The occasional white descendants continue to die out until all the white genes are gone.) The brown chickens did not evolve. They survived because they were resistant to the poison all along. Nothing has been added to the chickens' gene pool; instead, the ability to produce white chickens has been lost.

This is similar to what happens with bacteria and insects. We humans introduce antibiotics or poisons, to which most of them quickly succumb. Those that have a greater immunity survive and multiply until only the resistant strain is left. Nothing has been added; some of them were resistant from the start. If they had acquired this resistance by random mutations there would be many different strains in different geographic areas, each resistant to a different kind of poison. However, an Internet search shows that some bacteria thawed out after being frozen for over a hundred years have the same resistance as the supposedly new "superbugs" (e.g., Struzik, 1990). They've been here all along! Likewise, "mutated" insects or bacteria show immunity to the same poisons in widely different areas. This shows that their immunity is not the result of mutation but was present in some of them all along. It has nothing to do with evolution.

## D. PLEIOTROPY.

> Recommended resource: Michael Denton, *Evolution: A Theory in Crisis*, Adler & Adler, Bethesda, Maryland, 1986. Denton, an evolutionist specializing in molecular biology, presents some of the most devastating arguments against current evolutionary thought ever compiled in a single book. This book is a must for those seriously interested in biology. It can be obtained online or in local bookstores.

Most evolutionists believe new species appear as new genes develop by the replacement of nucleotides in their DNA. However, recent discoveries show that this is highly unlikely. Denton tells us that

Visual #11-31

> "Almost every gene that has been studied in higher organisms has been found to effect (sic) more than one organ system, a multiple effect which is known as pleiotropy" (Denton, 1986, 149).

For example, he notes that nearly every gene that affects the eye color of the house mouse also affects its body size. Likewise, fourteen of seventeen X-ray induced eye color mutations in the fruit fly *Drosophila melanogaster* also affected the shape of the female sex organs. Though the two systems have no obvious relation, changing one gene affects both.

These are not isolated cases. It seems that many segments of DNA are used two or more times during cell reproduction. DNA must contain far more information, encoded in a far more complex way, than we ever thought. Even one relatively minor change may have a drastic effect on an organism. The millions of changes required by evolution would have disastrous consequences.

As a self-replicating information storage system, DNA functions like a digital computer program. Any competent programmer uses *subroutines*, portions of the main program that can be called upon over and over from different places as needed. A change in one subroutine often changes the output of many places in the main program. DNA seems to work the same way. Pleiotropy shows us that it is a far more intricate program than we suspected. God invented subroutines long before we dreamed of computers.

## E. "PSEUDOGENES."

Visual #11-32

Cells use thousands of different types of proteins. (In humans, there are about 20,000 types.) The instructions needed to manufacture them take up only a tiny portion of the cell's DNA. Until the Human Genome project began to sequence human DNA, very little of the rest had a known function.

The portions not previously known to have a function often contain segments similar to each other and to other genes for which a function *is* known. In past years the unidentified segments were sometimes called "pseudogenes" or "junk DNA" and were claimed to be evolutionary leftovers from a common ancestor

The very name of pseudogenes ("false genes") is reminiscent of the concept of vestigial organs mentioned earlier. Because evolutionists in the late 1800s did not know the function of about 180 organs in the human body, they decided those organs had no function. Medical science has exposed their ignorance by discovering what almost all these so-called vestigial organs do. Now the argument has shifted from visible organs to microscopic segments of DNA. Evolutionists say that since they don't know the function of pseudogenes, there is no function.

This is quite an arrogant attitude. We continually learn more and more of the mysteries of DNA. For instance, we used to have no idea what the nucleotide sequence TTAGGG on a DNA strand meant. Now we recognize that it is a "stop" code indicating the end of a chromosome (Moyzis, 1991, 48-55). Creationists believe that a function will also be discovered some day for many of the supposed pseudogenes. Several factors already known may have something to do with the answer.

### *1. ASSEMBLY INSTRUCTIONS.*

Visual #11-33

Some say segments that do not code for proteins have no function. If we compare the construction of a cell to the construction of a house we see how ridiculous such a claim is. A house requires countless nails, screws, wires, pipes, paint, wood, sheetrock, roofing tiles, glass, light fixtures, switches, and so on. If we put in a work order to have these items delivered to our building site, the order could be seen as corresponding to the coding sections of DNA producing the correct proteins.

When the parts arrive, what do we have? A huge pile of unassembled parts that need to be put together. This is where the building plans and the skills of the construction workers come into play. Without them, we would never have a functional house.

To continue the analogy, scientists working with data from the Human Genome Project (Gates et al., 2021) have been learning more and more about the function of the non-coding sections. Hundreds of thousands of "long noncoding RNAs" (lncRNAs) that perform regulatory functions have now been identified (Marx, 2022). The coding genes allow the proteins to be manufactured, but the non-coding lncRNAs furnish the information to put them together into a living cell. They are not pseudogenes after all.

### *2. CELL DIFFERENTIATION.*

Visual #11-34

The DNA of every cell in an organism contains the same information, yet some of them become heart cells while others develop into skin cells, brain cells, or a myriad of others. Despite a great deal of speculation, no one is sure why a cell develops into one type and not the other. Perhaps pseudogenes have something to do with it.

### *3. REGULATORY FUNCTIONS.*

Researchers have discovered the function of a number of genes previously thought to be useless. For example, a gene known as p53 has no obvious function in normal human cells. However, biologists have recently discovered that it inhibits the runaway reproduction of defective cells. If p53 is missing or damaged, the defective cells can quickly develop into cancer tumors. Some pseudogenes may also have an as-yet-undiscovered regulatory function.

### *4. THREE DIMENSIONAL STRUCTURE OF DNA.*

So far we have considered DNA as if its full information content could be discovered by starting at one end and reading down its entire length - TTAGGGCATTGCA, etc. However, scientists are learning that DNA's 3-dimensional structure may have an important part to play in its replication (Wilder-Smith, 1993). During reproduction, it forms loops and makes contact with itself in many different places. While "pseudogenes" do not contain any known *transcriptional* information (one triplet specifying one amino acid), their contents may include *structural* information. They may be the contact points for the loops, or may be involved in specifying where the contact points are.

### *5. HIDDEN INFORMATION ENCODED IN DNA.*

At least a few segments of DNA contain far more information than is immediately obvious. We will see later in this chapter that the genes that code for human antibodies function something like a dictionary from which we can take selected words to form any sentence imaginable. Likewise, these fewer than 300 gene segments can code for up to 10 billion antibodies! This occurs because the cell is able to combine short pieces of them, not necessarily in sequence, in at least 10 billion ways (Behe, 1996, 127-134). There is far more here than meets the eye.

Perhaps "pseudogenes" are similar. The cell may use them in pieces rather than all at once.

### *6. DETERIORATION vs. EVOLUTION.*

Creationists believe that life was brought into existence in its most complex and organized condition. So-called pseudogenes could simply be damaged segments of the original DNA.

Even if none of these is the correct explanation, our ignorance of the function of "pseudogenes" doesn't mean they have none. There is no positive evidence that they have anything to do with evolution. We might wonder, too: If evolutionists believe "pseudogenes" don't do anything, then why do they spend so much time and money trying to sequence them?

Puzzling side note: some pseudogenes found in humans and gorillas are notably missing from our supposed closest relative, the chimpanzee (Woodmorappe, 2000). Why would transmission of the pseudogenes have skipped over such a crucial step?

## F. DIFFERENCES IN PROTEIN SEQUENCES OF DIFFERENT KINDS.

> See Denton, *Evolution: A Theory in Crisis*, Chapter 12, pp. 274-307. The following is a brief summary.

Each protein molecule is made up of hundreds of amino acids in a precise sequence. To illustrate, suppose a certain protein in humans contained the following sequence:

> -valine-histidine-leucine-threonine-proline-glutamic acid-glutamic acid-lysine-alanine- glycine-tyrosine-cysteine- and so on,

while in other kinds of organisms some of the acids were replaced with different ones. We could easily calculate the percentage of difference between the protein in humans versus other living things.

Visual #11-35

The amino acid sequences of some of the proteins found in different kinds of organisms have been studied in detail. One of the these is cytochrome C, which is involved in oxygen transfer within the body. The exact arrangement of amino acids in this protein differs from one kind to another by an amount that seems to correlate with how far apart they are on the evolutionary scale. This is often used to indicate an evolutionary sequence from lower life forms to man.

Cytochrome C is only one of thousands of proteins, though, and it is the *only* one that shows such a sequence. For instance, though cytochrome C in humans and in carp (fish) differ by thirteen percent, their hemoglobin differs by *fifty* percent! The hundreds of families of proteins each exhibit a different percent of variation. There is no evolutionary pattern (Denton, 1986, 296), though the differences in various proteins do show some interesting *non*-evolutionary patterns. Between any two major groups the differences are arranged in a hierarchical structure. For example, the carp's cytochrome differs by about 13% not just from humans, but from all non-fish vertebrates.

All the proteins studied so far show a similar pattern. Researchers have found that comparable proteins of any major group (class, order, etc.) show a fairly constant percentage of variation from those of any other major group. Suppose we compare an arbitrary protein, "Protein X," in vertebrates and insects. The amount of variation between any vertebrate and any insect is constant within a few percent. Any other arbitrary protein, "Protein Y," will probably show a different percentage than Protein X, but the percent difference between the Protein Y of any two major groups is still fairly constant. For example, within the vertebrates, all mammals differ from all birds by a different percentage; within the mammals, all primates differ from all rodents by yet another percentage, and so on. As Denton puts it (pp. 289-291),

> "Thousands of different sequences, protein and nucleic acid, have now been compared in hundreds of different species but never has any sequence been found to be in any sense the lineal descendant or ancestor of any other sequence... Each class at a molecular level is unique, isolated and unlinked by intermediates. Thus molecules, like fossils have failed to provide the elusive intermediates so long sought by evolutionary biology. Again, the only relationships identified by this new technique are sisterly. At a molecular level, no organism is 'ancestral' or 'primitive' or 'advanced' compared with its relatives."

The pattern of protein sequences agrees much better with the concept of initial complexity

(creation) than a gradual increase in information (evolution).

## II. HOMOLOGY - The Second Pillar of Evolution.

Visual #11-36

The first pillar of evolution was embryonic recapitulation - a fraud. The second pillar is *homology*, the presence of similar structures in otherwise dissimilar types of creatures. An example of homology is the basic mammalian body plan of four limbs with five appendages each (with a few exceptions such as marine mammals). Evolutionists believe the similarities indicate common ancestry; creationists believe they reflect common design.

Visual #11-37

Recent discoveries in molecular biology have caused a great deal of difficulty to this evolutionary scenario. If the similar features of different organisms are due to common ancestry, we would expect that these features should be produced by similar sites on the DNA of the different types. However, it turns out that structures thought to be homologous "are specified by quite different genes in different species" (Denton, 1986, 149).

Not only are the genes different, so is the method of development. Following are a few examples of seemingly homologous structures produced in non-homologous ways. Unless otherwise noted, quotes and page numbers refer to Denton's book.

- In **insects**, adult organs that are similar in different species are produced in "bewilderingly diverse" ways during metamorphosis (p. 148). For example, in the larvae of beetles (**Coeloptera**) the foregut and hindgut develop without any cell destruction. However, in butterflies (**Lepidoptera**) and flies (**Diptera**) new structures develop by the destruction of old ones. In ants and bees (**Hymenoptera**) likewise, new structures develop only as old ones are replaced (Chapman, 1969, 415-416). The final results are similar, yet they are arrived at by drastically different routes.
- Regarding **plant seeds**: conifer seeds (pine cones, etc.) are very similar to those of angiosperms (flowering plants) and are considered homologous by most botanists, yet key parts of the seed such as the ovule and endosperm form in a way that "profoundly differs in the two groups in a number of important respects." (pp. 148-149) Again, similar results come from dissimilar processes.
- In **vertebrate embryos**, "Homologous structures are often specified by non-homologous genetic systems and the concept of homology can seldom be extended back into embryology..."

Visual #11-38

Two key steps in the development of an embryo are the *gastrula* and *blastula* stages. The gastrula occurs when the fertilized egg has reproduced enough times to form a hollow sphere of cells one layer thick, then the blastula appears when an indentation forms in one end of the gastrula and develops into an inner layer or *endoderm*. The outer layer is now known as the *ectoderm*. The two are lined by a covering known as the *mesoderm*. From these early layers develop all the bodily systems.

These stages occur very early in embryonic development. Thus, evolution leads us to expect that they should be extremely similar in groups that are fairly close evolutionary relatives, such as vertebrates. However, Denton tells us that

> "In some ways the egg cell, blastula and gastrula stages in the different vertebrate classes are so dissimilar that, were it not for the close resemblance in the basic body plan of all adult vertebrates, it seems unlikely that they would have been classed as belonging to the **same phylum**... because of the great dissimilarity of the early stages of embryogenesis in the different vertebrate classes, organs and structures considered homologous in adult vertebrates cannot be traced back to homologous cells or regions in the earliest stages of embryogenesis. In other words, homologous structures are arrived at by different routes." (pp. 145-146)

So much for "embryonic recapitulation!"

- The **alimentary canal** (digestive tract) appears to be homologous in different vertebrate classes, yet it is "formed from quite different embryological sites..." (p. 146)

- **Vertebrate forelimbs** "... generally develop from different body segments in different vertebrate species. The forelimbs develop from the trunk segments 2,3,4, and 5 in the newt, segments 6,7,8, and 9 in the lizard and from segments 13, 14, 15, 16, 17 and 18 in man." (p. 146) Again, similar end results come from dissimilar mechanisms.
- **Development of the hand** in animals with five digits (fingers or toes). Ostrich embryos start with five digits in their "hands" (part of the wing). The three that eventually develop into something like fingers are digits two, three, and four. By comparison, in the "hands" of dinosaurs -- supposed to be the ancestor of birds -- the digits that developed into fingers were numbers one, two, and three. In human hands, the five digits develop from a thickening on the limb tip that splits into five parts; in frogs, the fingers grow from buds that are separated form the start. (Sarfati, 2002)
- Other examples: the kidney, ureter, amniotic & allantoic membranes (those which surround the embryo) develop differently in different species. (Denton, p. 146)

Denton is not alone in exposing the failure of homology to support evolution. British embryologist Sir Gavin de Beer, in an Oxford University Reader, tells us that genes which produce similar structures in unrelated species are quite different:

> "... homologous structures need not be controlled by identical genes, and homology of phenotypes [physical appearance] does not imply similarity of genotypes [genetic content]..."

Though he believes in evolution himself, he admits that homology does not furnish it any support.

> "...the inheritance of homologous structures from a common ancestor ... cannot be ascribed to identity of genes. The attempt to find 'homologous' genes, except in closely related species, has been given up as hopeless... It does not seem to matter where in the egg or the embryo the living substance out of which homologous organs are formed comes from. Therefore, correspondence between homologous structures cannot be pressed back to similarity of position of the cells of the embryo or the parts of the egg out of which these structures are ultimately differentiated." (de Beer, 1971, 15-16)

In summary, Sir Alister Hardy tells us that

> "The concept of homology is absolutely fundamental to what we are talking about when we speak of evolution - yet in truth we cannot explain it at all in terms of present day biological theory" (Hardy, 1965, 213).

The second pillar of evolution crumbles.

## III. GEOGRAPHIC DISTRIBUTION - the Third Pillar.

Visual #11-39

The third pillar of evolution is *biogeography*, or the geographic distribution of species. The same species or genera of animals and plants may be found in different places throughout the world, but with variations. In nature this may happen for several reasons.

(1). *Distance*. Roaches, for example, live almost everywhere. However, those in the western United States are somewhat different from those in the east. Distance has kept the two groups from interbreeding, allowing them to develop visible differences.

(2). *Natural barriers*. The squirrels on the north and south rims of the Grand Canyon belong to the same species, yet have a few visible differences. These have arisen because the two populations of squirrels cannot get across the canyon to breed with each other.

Many other species show similar types of variation. In extreme cases, group may specialize so much that they are no longer able to interbreed and are therefore classified as separate species. This process is known as *allopatric speciation.* Despite what evolutionists say, it has nothing to do with evolution.

### A. GEOGRAPHIC DISTRIBUTION AND NATURAL SELECTION.

Remember that a "species" is a reproductively isolated biological unit. Selective breeding experiments on the fruit fly *Drosophila* illustrate the concept well. The flies have been sep-

arated into groups according to desired characteristics such as eye color, number of hairs on the thorax, etc. As each new generation matures, only those individuals with the desired features are allowed to breed. The process is repeated for many generations until each group consists almost entirely of individuals that have the selected characteristics. If two groups that have been kept separated for a great many generations are finally allowed to mix together, they are sometimes unable to interbreed. In this case each of the groups is reproductively isolated from the other. Because of their inability to interbreed they are defined as separate species.

Such diversification has nothing to do with evolution. The breeders selected features that were already present. Not only have the groups failed to develop any new features, they don't even have all of the characteristics of the original parent flies. They have *lost* some of the original genetic information. (Their inability to interbreed may be related to the loss of other genes with no obvious relation to reproduction. As we saw earlier, many genes affect more than one body function.)

We can use a greatly simplified example to show how this phenomenon could happen in nature. Imagine a species of birds with two varieties, one with straight beaks and the other with curved beaks. Let's assume that there are just two alleles that control the beak shape, the "B" gene (straight beaks) and the "b" gene (curved beaks). Let's also assume that the B gene is dominant. If we start with two birds which each have both the dominant and recessive genes, they can pass on one of four combinations to their offspring, as follows:

Visual #11-40

| Gene from Father | Gene from Mother | Visible Result |
|---|---|---|
| B | B | BB - Straight beak |
| B | b | Bb - Straight beak |
| b | B | bB - Straight beak |
| b | b | bb - Curved beak |

On average, about one out of four babies will have a curved beak.

Visual #11-41

Suppose these birds live on an island fifty miles long and one mile wide with a dormant volcano in the center. On the island are two kinds of trees, one with seeds that require a straight beak to eat, the other with seeds requiring a curved beak. As long as things go along normally, the population contains a mixture of the two beak types.

Visual #11-42

One day the volcano erupts. Its poisonous gases kill all the animals on the eastern half, as well as all the trees whose seeds require straight beaks. (In nature, some individuals have a greater tolerance for harmful substances than others.)

Visual #11-43

Months later the volcano becomes dormant again. The surviving birds from the west begin to fly to the eastern half again. Those with straight beaks find nothing to eat and either fly back or starve. Those with curved beaks (bb genes only) stay and begin to reproduce. Soon there are only curved-beak birds on the eastern half of the island. Since they have no B genes, they will never again produce straight-beak offspring.

Visual #11-44

Another disaster strikes. A great windstorm blows many of the birds from both halves of the island to a different island beyond their normal flying range. (They are too far away to fly back.) All the trees on the new island have seeds that require straight beaks. The curved-beak birds starve, leaving a smaller and smaller percentage of b genes in the population. After many generations, almost all the b genes are eliminated. The few curved-beak birds that hatch on the island quickly die, leaving only straight-beak adults.

Visual #11-45

Visual #11-46

Is this evolution? Not at all. We started with a *founder population* which originally had two variants. After natural selection, there are still only two variants. However, some individuals have become specialized ("adapted") to fit into their environment. The curved-beaks have lost the ability to produce straight-beak offspring and the straight-beaks have

Visual #11-47

just about lost the ability to produce curved-beaks. The only thing that changed was the geographic distribution of the two varieties. Nothing new was added. Nothing evolved.

In the real world, there are seldom just two alleles controlling any given characteristic. However, the principle is the same. If a founder population starts to radiate outward from a starting point -- say, from the Atlantic Ocean toward the Pacific -- its members encounter varying food sources and environmental circumstances as they travel. The individuals whose characteristics are best suited for each area will tend to thrive and leave more offspring there. Those less suited will likely go back or continue to migrate. As they continue to spread out the process will repeat. Because of the gradual loss of genetic information in those that keep migrating, there will be a gradual shift in characteristics of the species from the starting to the ending points. The population will tend to become more and more specialized as it moves farther away from where it began. Some groups may even become reproductively isolated from the others. In this case, they are defined as new species even though they do not have any new features.

Visual #11-48

Evolutionists claim that such geographical variation explains the evolution of new types. It does nothing of the sort. The specialized groups each have *less* genetic information than their generalized ancestors in the founder population. They are degenerate, not more advanced. Evolution can't explain how the ancestors got all their genetic information in the first place. Geographic distribution has to do with the diversification of species, but it cannot explain their origin.

## B. UNUSUAL FEATURES OF ISOLATED GROUPS

### *1. ANIMALS OF THE GALAPAGOS ISLANDS.*

The Galapagos are a chain of 13 main islands and 48 lesser islets, located about 600 miles off the Pacific coast of Ecuador. The total land area is just under 4900 square miles, about 3/4 that of Hawaii. Because the chain is hundreds of miles from the mainland and the individual islands are separated from each other, this is an excellent place to observe exactly the kind of specialization described above. It was here that Charles Darwin put together some of his major ideas about evolution.

Despite being raised in a Unitarian household and attending divinity school, he abandoned his faith in God during his college years. As he looked for something to take God's place, he spent a great deal of time studying living things. While in his twenties, he obtained a position as naturalist on the British surveying ship *HMS Beagle* on its five year voyage around the world from 1831 to 1836. (He was always interested in living things and was recognized as an expert pigeon breeder.) When the ship landed at the Galapagos Islands in 1835, he observed many unusual animals such as specialized iguanas, tortoises, and birds, that fit with the unusual environment.

Visual #11-49

Remember that the idea of evolution had been around for thousands of years before Darwin, but no one had proposed a plausible mechanism. His grandfather Erasmus Darwin and others had already persuaded him that evolution was correct, so Charles was simply looking for a way to fill in the details. As he noted the unusual features of the Galapagos animals, he concluded that they must have evolved features as needed. He was unacquainted with the brand new science of genetics and was unaware of the function of genes. We now know that use and disuse of body parts -- Lamarckianism -- has nothing to do with inheritance. Genes, not need, determine the characteristics of offspring.

One of the main problems with Darwin's ideas about the Galapagos animals is that the islands are volcanic in origin and are believed to be no more than a few million years old. Evolutionists believe that the Galapagos animals' genes would take many millions of years to evolve, so they must have come from a founder population that already had the appropriate genes before they arrived. The unique environment led to

specialization, but it had nothing to do with the appearance of new genes. There had to be a founder population somewhere else for each of the Galapagos types.

Visual #11-50

The best-known animals of the Galapagos are the finches with their many different beak shapes. Some enthusiastic evolutionists have classified them into thirteen different species and up to six genera based on these differences. This is incorrect. Since at least some of the "species" are able to interbreed (White, 2002; Grant, 1992), they are not separate species but variants of a few or even one species.

Each beak shape matches well with certain types of food supplies such as the seeds of various types of trees. When the supply of any given food type increases due to weather and other environmental circumstances, the finches with the appropriate shape prosper. When the supply decreases, they tend to die off. This results in varying percentages of each beak shape within the finch population. If enough individuals with small beaks die off, the overall average beak size becomes larger; if enough large-beaks die off, the overall average size becomes smaller. However, when the circumstances change back, so do the percentages and the average beak size. This is a fine example of natural selection, but it is not evolution. No new genes are added to the finch population.

Visual #11-51

Let's consider an analogy that shows how the changing average beak size has nothing to do with evolution. Suppose a five foot tall dictator was sensitive about his height and decided to kill everybody taller than himself. The average height in his country would immediately decrease. However, as soon as he died or was overthrown, tall children would no longer be eliminated and the average height would return to its former level. Like the average beak size, this is not evolution. No new genes were added.

Natural selection can explain how the Galapagos animals might have become specialized after they arrived, but it cannot explain how their founding ancestors acquired all their genetic information in the first place.

***2. AUSTRALIAN MAMMALS.***

Visual #11-52

The only mammals known to be native to Australia are bats (whose ancestors presumably flew there long ago), marsupials (pouched animals), and monotremes (egg-laying mammals in which the females have one opening instead of two). The latter two groups include some exotic species. Where did they come from?

***a. Monotremes.***

No known transitional forms, either fossil or living, show how egg-laying mammals such as the platypus or echidna (spiny anteater) might have evolved. They simply appear fully formed in Australia.

***b. Marsupials.***

Marsupials differ from other (*placental*) mammals in their method of reproduction. Instead of giving birth to fully developed young, they produce immature offspring who immediately climb into the mother's pouch until they complete their development.

If natural selection pressure caused such a system to evolve, it would make sense to expect that the marsupials would all be weird creatures. Yet many animals in other parts of the world have marsupial equivalents - mice, cats, deer, wolves, badgers, moles, flying squirrels, and rabbits. We might guess that these marsupial versions evolved in response to natural selection pressures, until we consider that the pressures in many parts of Australia are different from the rest of the world. There is no known reason why Australian animals experiencing such different selection pressures would have evolved such similar features. Creationists believe the similarities are evidence of common design.

One species of marsupial, the opossum, lives in other areas besides Australia. Dozens of fossil species of marsupials are also found elsewhere. Marsupials must not

have evolved in Australia, but migrated there. Since their founder populations first appeared elsewhere, their presence in Australia has nothing to do with evolution.

One possible explanation for the lack of placental mammals in Australia may be the aftermath of Noah's Flood. As animals spread out from the Middle East, those who had to wait for their young to mature would spread more slowly. Those who could carry their young with them in pouches would have been able to travel farther in the same amount if time. If there used to be land bridges to Australia as many suspect, the marsupials may simply have gotten there before the bridges sank. The placental mammals may have gotten there too late.

## C. THE ORIGIN OF RACES.

Many creationists believe that "races" within the human species may have developed by a combination of natural selection and divine intervention.

The most obvious difference between people groups is the color of their skin. All of us except albinos have the same skin pigment, called *melanin*. Though a number of variables such as the amount of oil can modify skin shade somewhat, the amount of melanin is the main factor in how light or dark we are.

Our present understanding of biology indicates that the amount of melanin is controlled by three sites on the DNA strand, each of which has a place for two genes. These come from *gametes* containing three genes furnished by the father three furnished by the mother.

Each site has the potential to have two dominant alleles, two recessive, or one of each. As shown on the chart below, there are about twenty-seven possible combinations of genes. Since AaBBCC is equivalent to aABBCC, aabbcC is equivalent to aabbCc and so forth, these can combine so as to give anywhere from zero to six dominant genes. These combinations lead to seven main skin shades, or eight if we include albinos.

Visual #11-53

***Across: mother's contribution. Vertical: father's.***

**Number of dominant genes indicated by number above gene combination.**

| Gametes | ABC | ABc | AbC | Abc | aBC | aBc | abC | abc |
|---|---|---|---|---|---|---|---|---|
| ABC | 6 | 5 | 5 | 4 | 5 | 4 | 4 | 3 |
| | AABBCC | AABBCc | AABbCC | AABbCc | AaBBCC | AaBBCc | AaBbCC | AaBbCc |
| ABc | 5 | 4 | 4 | 3 | 4 | 3 | 3 | 2 |
| | AABBCc | AABBcc | AABbCc | AABbcc | AaBBCc | AaBBcc | AaBbCc | AaBbcc |
| AbC | 5 | 4 | 4 | 3 | 4 | 3 | 3 | 2 |
| | AABbCC | AABbCc | AAbbCC | AAbbCc | AaBbCC | AaBbCc | AabbCC | AabbCc |
| Abc | 4 | 3 | 3 | 2 | 3 | 2 | 2 | 1 |
| | AABbCc | AABbcc | AAbbCc | Aabbcc | AaBbCc | AaBbcc | AabbCc | Aabbcc |
| aBC | 5 | 4 | 4 | 3 | 4 | 3 | 3 | 2 |
| | AaBBCC | AaBBCc | AaBbCC | AaBbCc | aaBBCC | aaBBCc | aaBbCC | aaBbCc |
| aBc | 4 | 3 | 3 | 2 | 3 | 2 | 2 | 1 |
| | AaBBCc | AaBBcc | AaBbCc | AaBbcc | aaBBCc | aaBBcc | aaBbCc | aaBbcc |
| abC | 4 | 3 | 3 | 2 | 3 | 2 | 2 | 1 |
| | AaBbCC | AaBbCc | AabbCC | AabbCc | aaBbCC | aaBbCc | aabbCC | aabbCc |
| abc | 3 | 2 | 2 | 1 | 2 | 1 | 1 | 0 |
| | AaBbCc | AaBbcc | AabbCc | Aabbcc | aaBbCc | aaBbcc | aabbCc | aabbcc |

http://www.pbs.org/race/000_About/002_04-teachers-06.htm

In general, the greater the number of dominant genes the darker the skin and the smaller the number, the lighter. Thus, AABBCC is the darkest and aabbcc is the lightest. If two parents each had the full gene complement of AaBbCc, they could produce children with up to 27 combinations of genes. These would give seven different possible numbers of dominant genes and thus seven major skin shades (with variations due to skin oils, sun

exposure, etc.) ranging from extreme light to extreme dark. The children would not belong to different races; they would be brothers and sisters.

In order for Adam and Eve to produce all of us, they must have had a similar arrangement of genes. To the chagrin of white supremacists everywhere, they would likely have had medium brown skin. If their descendants were freely interbreeding, most would probably be a medium shade of skin color, with extremes of light and dark fairly rare. However, if people were separated so that those with dark skin genes associated only with each other and those with genes for light skin did the same, groups with extremes of light and dark could develop in just a few generations.

Visual #11-54

How could such a separation occur? The Bible says that God did it. Genesis 11:1-9, the account of the Tower of Babel, tells us that He divided the human race into different language groups – a very effective way to keep people from associating with each other. God determined which individuals would speak which languages. Though the Bible doesn't say how He decided, we can speculate that He knew which of them had which skin color genes and separated them accordingly.

Within a few generations after the initial separation, natural selection would have reinforced the skin color groupings. A high concentration of melanin produces two effects: (1) dark skinned people can tolerate sunlight much better than those with very light skin and are much less susceptible to skin cancer, and (2) dark skinned people need much more sunlight in order to produce substances such as Vitamin D which help them to stay healthy. Dark skinned people would tend to fare better in sunnier climates such as those near the equator, while those with light skin would do better where the sun is less intense, farther away from the equator. Until recent developments in rapid transportation, this was just about the way people of different skin darkness were distributed.

The differences between "races" such as facial features or hair texture are far less than those between different breeds of dogs or other animals. Nevertheless, skin color has been used as the basis for racism, slavery, and other atrocities. Surely God knew this would happen. Why did He separate us into different ethnic groups? It had nothing to do with any group being superior or inferior to any other. His purpose was twofold:

(1) To furnish a visual object lesson showing that He will not allow sinful mankind to unite in rebellion against Him, and

(2) To put a hunger in us to grope after Him and find Him (Acts 17:26-27).

He doesn't care if we are black, white, or any other color. When He looks at us He sees our sin, not our skin. Each of us needs to surrender to Jesus Christ as our Lord and Savior: then, when God looks at us, all He sees is the righteousness of His Son.

## IV. THE CASE FOR DESIGN IN NATURE.

Many evolutionists (e.g., Francis Crick, winner of the Nobel Prize for his co-discovery of the structure of DNA) admit that many structures in nature *look* like they were designed.

### A. HOW EVOLUTIONISTS DEAL WITH THE APPEARANCE OF DESIGN.

Visual #11-55

Remember that the most fundamental postulate for evolutionists is that everything must be explainable by purely natural processes. Any clear evidence of design in living things implies divine intervention. Even Darwin admitted that if this were the case, his theory would

Visual #11-56

"absolutely break down." So how can they reconcile the appearance of design with the axiom of randomness?

Darwin gives us a clue. Many creationists cite his admission in *The Origin of Species* that the eye was difficult to explain by chance:

Visual #11-57

"To suppose that the eye with all its inimitable contrivances for adjusting the focus to different distances, for admitting different amounts of light, and for the correction of Spherical and chromatic aberration, could have been formed by natural selection, seems, I freely confess, absurd in the highest degree..."

If we stop reading there, we would think that Darwin was admitting defeat. He was not! He went on to make up a story about how it could have happened:

> "Reason tells me, that if numerous gradations from a simple and imperfect eye to one complex and perfect can be shown to exist, each grade being useful to its possessor, as is certain the case; if further, the eye ever varies and the variations be inherited, as is likewise certainly the case; and if such variations should be useful to any animal under changing conditions of life, then the difficulty of believing that a perfect and complex eye could be formed by natural selection, should not be considered as subversive of the theory."

Visual #11-58

Atheistic evolutionists continue to follow Darwin's example. No matter what structures we point out as evolutionary difficulties, they can always make up a story. And since they are so clever, therefore their stories must be true and we creationists should just give up and start believing them!

Not all evolutionists are atheists, of course. Many believe in "Intelligent Design" (I.D.), a broad concept that could include either creation or theistic evolution. In the last few years there has been a nationwide movement to try to have I.D. included as an option in biology classes. However, every school district considering this possibility has immediately faced

Visual #11-59

legal challenges by such groups as the ACLU. Judges in almost every court case have accepted the atheistic position that since the presence of a designer cannot be tested scientifically, therefore intelligent design is not scientific.

It is true that we cannot scientifically prove the possibility of intelligent design. What the judges are overlooking, though, is that we also cannot scientifically **dis**prove the possibility of intelligent design. Either way this is not a disagreement over scientific facts, but instead, over an *a priori* assumption.

Visual #11-60

Recall from earlier chapters that there are two forms of logic relevant to the creation/evolution controversy: inductive and deductive. Inductive logic requires that we look at many phenomena and try to discover a pattern that points to a general principle, in an attempt to determine the most reasonable (most likely) conclusion. This is how the scientific method works. In deductive logic, on the other hand, we start with general principles (*a priori* assumptions) accepted as true without proof and apply them to specific cases. Assuming that the premises are true, then the conclusion MUST be true. To summarize the

Visual #11-61

contrast, the premises and conclusions of inductive logic result from examination of observable phenomena (*a posteriori*). They are testable. The premises of deductive logic, on the other hand, may come from inductive conclusions or may just be statements accepted as self-evident (*a priori*). They are not necessarily the result of testing.

Despite the deception that many judges have fallen into, whether you believe I.D. is possible or impossible, it's not scientific. It's a question of deductive logic based on opposite *a priori* assumptions:

- Either everything must be explainable by purely natural processes, or
- Some things may not be explainable by purely natural processes.

Neither one can be proven. It's a matter of deductive logic, not science.

## B. CREATIONIST ARGUMENTS FOR DESIGN.

Visual #11-62

In Chapter Ten we saw that there are three major types of arguments for design: (1) Opinion, (2) Extreme Improbability in a specific direction, and (3) Irreducible Complexity.

The first type of argument doesn't carry much weight with evolutionists. For example, though the presence of brightly colored fish around a coral reef might lead us to believe in a Creator with a sense of beauty, we can't prove that evolutionists are wrong when they attribute it all to random chance.

### *1. CONVERGENCE OF PROBABILITIES.*

In a completely random system, no one arrangement is any more or less improbable

than any other. (*Somebody* is probably going to eventually win the lottery.) If the same person wins the lottery week after week, though, we will probably suspect something other than randomness. Likewise, if we look at the carvings on Mount Rushmore, we can easily determine that the pattern of rocks is very *non*-random.

Visual #11-63

The search for design in nature is similar. We are not looking just for improbability, but improbability in a specific direction. Though an atheist could argue that any arrangement of atoms in nature is equally improbable, most arrangements produce meaningless junk. Only a few produce life. The question is, how improbable is it that those specific arrangements could arise by random processes?

Even the staunchest evolutionists must admit that many natural phenomena are difficult to explain as the result of random processes, yet they insist that it's all random anyway. Earlier we noted a principle known as Occam's Razor, paraphrased as *"the simplest explanation that fits all the facts is probably the best."* Closely akin to this is a concept that just about everyone (except evolutionists) intuitively recognizes: the *convergence of probabilities*.

Visual #11-64

Even if one event seems highly improbable, one could always explain it away by making up a story. Evolutionists have made up story after story about the universe outside the earth -- the big bang, the origin of the elements, the origin of stars and galaxies, the origin of the solar system, and so on. Likewise, when it comes to living things, they can make up stories about how any irreducibly complex structure could evolve by chance anyway. It is possible that any story could be right. There is no limit to the number of stories they can make up, any one of which could also be true. However, since evolution requires *everything* to be explainable by natural processes, if even ONE evolutionary story is wrong then all of evolution is falsified! The more stories evolutionists have to invent, the less likely it is that ALL of them are true and the more likely it is that the basic premise, random chance, is false.

Visual #11-65

Visual #11-66

Remember our hypothetical groundhog from the last chapter in Section I-B-2. Though it there is no theoretical barrier that would prevent him from making it across a busy thousand lane superhighway, his chances are so slim that if we find him on the other side we can be almost certain that somebody put him there. If we find a second groundhog on the far side of a second highway, then a third, fourth, and so on, we would be suspicious that the groundhogs had some help. Likewise, the greater the probability against a specific structure developing by random chance, the greater the probability it came from design. The more such structures we find, the greater the convergence of probabilities that they are the result of design, not chance.

Visual #11-67

***2. IRREDUCIBLE COMPLEXITY.***

Visual #11-68

This is the most compelling argument in dealing with specific structures that give the appearance of design. If a structure in any type of organism can be shown to be irreducibly complex, then all its parts would have had to be present and performing at least minimal function since the beginning of that type. The chance that it could have come together one piece at a time is virtually zero. To person who does not already have his mind made up that design is impossible, it is a virtual certainty.

## C. SPECIFIC EXAMPLES OF DESIGN.

Let's look at some examples from nature that point toward a high probability of design. As we do, challenge your students to identify which arguments for design apply to each: (1) Opinion, (2) Probability, or (3) Irreducible Complexity.

Recommended Resource: *Darwin's Black Box* by Michael Behe, referred to in Chapter Ten.

***1. DNA.***

We can't appeal to irreducible complexity to show design in DNA, because it is a fantastically complex information storage system that contains far more than the bare min-

imum needed to keep an organism alive. The principal argument for design in this case is the extreme improbability of assembling such complexity by chance.

DNA functions like a better designed computer program than any human author has ever written, making excellent use of space, chemical resources, and subroutines. (See the discussion of pleiotropy earlier in this chapter.) The probability that such a complex program could come together by random chemical action is comparable to the probability of our hypothetical groundhog making it across multiplied thousands of lanes on the superhighway.

No one has ever seen a self-replicating digital program come together by accident. It requires a programmer. (Even a self-improving computer program such as "artificial intelligence" *starts* with the human intelligence of the programmers.) The structure and operation of DNA are clear evidence of design.

***2. IRREDUCIBLE COMPLEXITY OF CELL REPRODUCTION.***

Visual #11-69

(1) In order to be alive, the first living cell would have needed enough parts such as proteins, enzymes, etc. to have at least minimal function.

(2) Since we are all supposed to have evolved from it, it would also need some sort of information storage system, a precursor to DNA, so that it could make reasonably functional copies of itself.

(3) It would not be sufficient just to have the stored information -- there would have to be a way to translate it into physical structures. Something that worked like messenger RNA would have to transport specific subsets of the information to a place where they could be used as a template for reproduction.

(4) In that location within the cell, there would need to be some structure that would allow the parts of the newly forming daughter cell to come together. Cells do this today by fastening messenger RNA to ribosomes at the endoplasmic reticulum, then using transfer RNA to put together each protein, one amino acid at a time. The proteins are transported wherever needed throughout the cell by tiny molecular machines, then assembled into functioning structures.

This is an amazingly complex and well coordinated system. Note that at the very least it requires messenger RNA, enzymes, ribosomes, endoplasmic reticulum, and transfer RNA. Each of these exists because DNA contains the information needed to produce them. But the very first living cell would not have had any DNA yet, so none of these parts essential to cell reproduction would have been present to continue the process. Only by a great exercise of faith can evolutionists avoid the obvious conclusion that DNA and cell reproduction are an irreducibly complex system.

***3. UNIVERSAL GENETIC CODE.***

Visual #11-70

(1) Remember that DNA is supposed to have evolved by accident some time after the first cell came alive. Once DNA finally evolved by some mysterious process, it would have contained very little information, only a small number of base pairs.

(2) Somewhere in the course of evolution, a tremendous amount of extra genetic information would have to be added so that instead of a few base pairs, many modern organisms have anywhere from millions to a hundred billion.

(3) Later, the mechanisms that serve to prevent and correct errors would have had to develop due to copying errors that were not prevented and corrected.

(4) However, one crucial thing "forgot" to evolve: the genetic code. Every type of organism, no matter how primitive or advanced it is supposed to be, uses the same genetic code and the same protein manufacturing mechanism of messenger RNA, transfer RNA, ribosomal RNA, ribosomes, and endoplasmic reticulum.

Evolution is supposed to have begun with primitive one-celled organisms and gradually progressed all the way to the highest mammals. However, the size, structure, and component design of the protein manufacturing apparatus (ribosomes, endoplasmic

reticulum, etc.) is virtually the same in every cell. Nothing can be shown to be ancestral to or descended from anything else (Denton, 1986, 250). Does it make sense that DNA could have evolved billions of steps from nothing to its present condition, while the genetic code and manufacturing apparatus it uses didn't change even the slightest bit?

***4. BLOOD COAGULATION.*** (Behe, 1996, 74-97)

Most of us take for granted that when we cut ourselves the bleeding will soon stop by itself. But why should it? If we puncture almost any other system filled with pressurized liquid it leaks until the pressure reaches equilibrium with its environment. Were it not for the blood's ability to coagulate, we would bleed to death the first time we got cut.

Behe likens blood coagulation to a Rube Goldberg machine. Goldberg was a cartoonist popular in the early to mid 1900s, known for his humorous drawings of elaborate contraptions designed to accomplish a simple purpose. For example, Behe shows (p. 75) Goldberg's plan for an automatic mosquito bite scratcher that requires 16 steps involving such components as a drunken bird and a somersaulting dog. (The children's game of "Mousetrap" is another example of a Rube Goldberg type machine.) Though humorous, many of Goldberg's contraptions were irreducibly complex: if any component failed to function properly, the whole thing wouldn't work.

Visual #11-71

Blood coagulation is much more involved than any of Goldberg's mechanisms, but it too is irreducibly complex. From the time you cut yourself until you stop bleeding, over twenty proteins and other factors are busily at work. These include multiple proenzymes and enzymes, at least one vitamin, and such things as "Christmas Factor" and Stuart Factor. Throughout the process these components cut, fasten, activate and deactivate each other at exactly the right times and rates. There are feedback and feed ahead control loops. The whole cascade involves dozens of steps. If even one of the components fails to work properly you either bleed to death or die of blood clots.

While an evolutionist might argue that our coagulation system could have evolved from a similar one in lower life forms, this doesn't answer the question of how the very first such system could have come into existence. Not every type of organism has such a system. In even the most "primitive" organisms that do, a single malfunctioning component kills the creature. It is not possible to put together such a mechanism one step at a time by modifying a previously existing mechanism of a different type in a lower life form. Blood coagulation is irreducibly complex. It had to be designed.

***5. ANTIBODY PRODUCTION.*** (Behe, 1996, 120-130)

At the microscopic level, it's a dangerous world. Our bodies are under constant attack from bacteria, viruses, chemicals, and who knows what else. Fortunately, our immune system protects us from such threats. When functioning properly, it can identify and destroy almost any invader imaginable.

Visual #11-72

In order to defend the body, the immune system needs first to identify invaders, then to mark them as targets, and finally to destroy them. While all these operations are fascinating, we will here consider only the identification and marking systems. Those who want to know more about the destruction mechanism should read Behe's book.

How does the immune system mark objects that constitute a threat? It uses *antibodies*, tiny Y-shaped molecules composed of two "heavy" and two "light" chains of amino acids, to identify foreign substances as targets. Because of the three-dimensional shapes of amino acids, the ends of the chains form finger-like protrusions with billions of possible shapes. When one of the combinations matches the shape of an invader, the antibody attaches to it. Whenever the immune system detects an object with an antibody attached, it "knows" that it must destroy it.

Visual #11-73

Since there are billions of possible types of invaders, how can the body produce antibodies to identify them all? After all, there are only about 3 billion nucleotides in our DNA. If every one of them were used to code for antibodies, they would constitute

about one billion triplets, coding for perhaps a few million types of antibodies. Yet our cells have the ability to produce over ten billion distinct types. How do they do it?

The key is in the programming. Only a small portion of DNA codes for antibodies, but it does so in an astonishingly efficient way. Researchers have discovered that a gene coding for an antibody does not need to be a continuous segment, but can be interrupted without harm. Thus, the antibody coding genes function like a biological dictionary. Just as we can form a complete sentence by taking a word out of the dictionary, skipping some, taking another, skipping more, taking another, and so on, likewise the cell takes a piece of a gene, skips some, takes another, skips more, takes another and so on, until it assembles the complete gene needed to produce a desired antibody. (We should note that some researchers are beginning to consider the possibility that at least a few so-called pseudogenes may also include noncontiguous coding segments.)

According to our present understanding, the "dictionary" is quite small, consisting of four gene clusters. The first cluster contains about 250 gene segments, the second has ten, the third has six, and the fourth has eight. If we take one from segment one, one from two, one from three, and one from four, there are about 120,000 possible combinations producing distinct types of heavy amino acid chains. Since the light chains need not come from the same segments, the number of possible combinations of heavy and light chains is tens of thousands of times greater. This enables the immune system to produce more than ten billion different types of antibodies.

Some might still insist that such an amazingly efficient system could have evolved by chance. Perhaps the groundhog might make it all the way across the highway, but it's not likely. Even if it were possible to assemble this system by accident, there's more.

When the body is invaded by a foreign substance such as a virus, it's not enough to have a bunch of antibodies floating in the bloodstream. The immune system needs to be able to rapidly manufacture billions more of the appropriate ones. This requires at least three steps:

(1) There needs to be a mechanism to attach each of the billions of types of antibodies to the outside of the cells that produced them so that those cells can serve as factories to produce many more.

(2) There needs to be a manufacturing apparatus inside the cell to duplicate only the desired antibody.

(3) It would be wasteful if the body manufactured billions of copies of billions of unneeded antibodies. There needs to be a "messenger" from the antibody on the outside of the cell to notify the manufacturing apparatus that it has captured a prisoner (the virus). Only when the messenger goes to the nucleus of the cell and notifies it to begin mass production does the cell begin its work in earnest.

So where did this amazing mechanism come from? Nobody knows. There is nothing in any known invertebrate from which it might have evolved.

It would be well worth your students' while to read Behe's book and learn more details of this elaborate system. The important point is that it is irreducibly complex. If the antibody is floating free it is useless; if there is no duplicating apparatus nearby it is useless; and if there is no messenger between the two it is useless. It is extremely unlikely that the antibody manufacturing system is the product of random chance. Anyone but the most ardent atheist would conclude that it seems to have been designed.

***6. CELL STRUCTURES.***

We could look at almost any part of a cell – its method of reproduction, the interaction of DNA and enzymes, DNA and membrane permeases, etc. – and see evidence of design. A few specific examples:

***a. Vacuoles.***

Visual #11-74

Water moves through membranes by a process called *osmosis*. If the concentration of chemicals in water is different on opposite sides of a membrane, the water flows through until the concentration is equalized. This can cause a problem in cells, which contain many substances besides water but often float in a watery environment. More and more water forces its way inside the cell to equalize the concentration. Eventually, the cell would burst because of the internal pressure - except for a built-in safety feature, hollow chambers called *vacuoles*. As water pressure increases to a dangerous level, the cell pumps some of the water into the vacuole, from which it is forced out of the cell. Where did the vacuole come from? It is present because it is programmed in the DNA. DNA is needed to make vacuoles, but vacuoles are needed to insure the survival of DNA. The same question faces us: could this irreducibly complex system have come together a piece at a time by chance?

***b. Lysosomes.***

Since cells are constantly dying in our bodies, why aren't we cluttered up with dead cells? Because most cells contain tiny "suicide sacs" called *lysosomes*. These are filled with enzymes capable of digesting proteins such as those that make up the cell. During the life of the cell they help break down nutrients into usable components. However, when it dies they rupture, releasing the enzymes and causing the dead cell to eat itself up. The waste material is then easily flushed out of the body.

Multicelled organisms could still live but would be much less healthy if the lysosomes did not rupture at the time of death. But why don't they rupture earlier? Because the cell environment prevents them from doing so. And why is that environment the way it is? Once again, we come back to DNA. Accidental or designed?

***7. SPECIALIZED ORGANS AND STRUCTURES.***

We already mentioned the giraffe's neck; later we will touch on photosynthesis, feathers, the mammalian ear, and other structures that point toward design. Here are a few other examples.

***a. Active Transport of Minerals in Plants.***

Visual #11-75

Plants get most of their hydrogen, carbon, and oxygen from air or water. Many of them also absorb minerals such as iron, potassium, zinc, calcium, molybdenum, magnesium, etc. from the soil. Since a plant is largely made of water and since it takes in these substances from wet soil, we would expect their concentration to be about the same inside and outside the plant. (According to the Second Law of Thermodynamics, everything tends toward equilibrium.) However, the concentration of minerals inside plants can range from 75 to 10,000 times greater than in the surrounding soil. There has to be a mechanism – *active transport* through enzyme action – to pull needed elements out of the soil and transport them into the plant. Where did the enzymes come from? They are programmed into plant DNA. One must ask himself: is it likely that such a highly ordered information system happened by accident?

***b. Sap Transport in Trees.***

Visual #11-76

Upright plants such as trees and shrubs need a constant flow of water and nutrients from the soil to their upper parts. This is a problem in tall trees.

Imagine you had a hundred foot high drinking straw in a glass of water. How hard would you have to suck on it to get a drink? Even if the straw didn't collapse, you couldn't do it. It's not the suction that makes the water move up the straw, it's the atmospheric pressure on the water that pushes it up. Normal atmospheric pressure can support a column of water about 32 feet high. Low pressure may support a bit less, high pressure a bit more. Even if we apply a perfect vacuum to the top of

the straw, the water will rise no higher than about 35 feet even under extremely high atmospheric pressure. Yet some trees grow hundreds of feet tall. How do they get their water-based sap up to the top?

Part of the force to lift the sap does come from reduced pressure as water evaporates from the top of the tree. However, most of the force comes from a built-in pumping mechanism that operates all the way up the tree. The roots exert a certain amount of pressure, but other little-understood processes maintain it all the way up to enable the tree to keep getting taller.

Whatever these processes are, they are programmed into the tree's DNA. Where did they come from? Natural selection should favor organisms that fit best with their environment, not those that have to develop elaborate mechanisms to overcome it. Evolution should produce trees no more than about thirty-two feet tall. Yet here they are. Could it be they were designed that way?

***c. Bacteria with Electric Motors.*** (Behe, 1996, 69-73)

Visual #11-77

We tend to think of bacteria as very simple organisms. However, some types contain at least one irreducibly complex structure that has no counterpart in more "advanced" cells - their swimming apparatus.

Any mechanism that moves an object through liquid must have at least three components: a paddle or propeller, some sort of motor, and a device to connect the two. Some bacteria have tiny paddles known as *cilia,* while others have the equivalent of propellers. Both types of propulsion have all the necessary components. The one we are concerned with is the latter, the rotary *flagellum*.

Certain bacteria swim by means of flagella, hairlike filaments with a corkscrew shape. Rather than waving back and forth like flippers, the flagella rotate like propellers. But what turns them? The power comes from microscopic acid-driven electric motors! The motors are so small that even our most advanced scanning techniques have difficulty revealing all the details, yet we know that they have a stator, a rotor, and electrical connections. There must also be some sort of extremely low friction protein bushings where the motor shaft penetrates the cell membrane. On top of everything else, the motors are individually reversible and connect to their respective flagella through biological gear boxes with a 30:1 gear reduction ratio! (Personal communication, Dr. Richard Lumsden.)

Though over 200 proteins are involved, the motor-connector-propeller system is irreducibly complex, as is the motor itself. There is no mechanism in any known living thing from which the bacterial propulsion system could have evolved. Though evolutionists point out that a few of the parts are similar to parts in other cell mechanisms and thus could have been "co-opted" to use in the motors, the majority are found nowhere else except in the motors. We can make a choice -- by faith -- to believe the motors are the result of accidental mutations, or we can reach the obvious conclusion that they were probably designed.

***d. Defense Mechanisms.***

Just two of the many elaborate defense mechanisms throughout nature:

i. *Corals*.

Some kinds of coral are supposed to date all the way back to the Ordovician Period, said to have ended 500 million years ago. One fossil deep-sea coral is virtually identical to its living counterpart, known as *Gorgonia*. These have an egg-shaped stinger called a *cnidocil* protruding slightly from a cap-covered hole filled with poison. When touched, the cnidocil springs out within *three to five ten-thousandths of a second* and injects its crippling venom (Fredericks, 1985, 87). Is it really reasonable to believe that such a lightning fast mechanism evolved by accident hundreds of millions of years ago in an otherwise almost

motionless deep-sea coral?

*ii. The Bombardier Beetle.* (Gish, 1977, 51-53; Behe, 1996, 31-36)

Visual #11-78

One of the most unusual defense mechanisms belongs to the "bombardier beetle," *Brachinus tschernikhi*. This insect has two internal storage chambers containing a concentrated mixture of hydrogen peroxide and hydroquinone, which have the potential to react violently when certain catalysts are added. Each of these chambers is connected to a combustion chamber through a narrow tube controlled by a sphincter muscle. The combustion chambers act as firing tubes.

When threatened, the beetle aims the tubes at the enemy and injects some of the hydroquinone/peroxide mixture into the combustion chambers along with the enzymes catalase and peroxidase. This produces a violent explosion of boiling hot, foul tasting liquid. A predator hit in the face with such a blast quickly loses interest in eating the beetle.

Could the chemistry have evolved one step at a time?

- If the mechanism to produce catalase and peroxidase did not evolve at the same time as that for concentrated peroxide and hydroquinone, the latter two substances would have been useless and would have taken up precious resources that could have been better used elsewhere.
- If the combustion control mechanism did not evolve at the same time as the chemical manufacturing apparatus, the beetles would have become extinct because they would have exploded.
- At the same time, they needed the storage chambers, connecting tubes, sphincter muscles, combustion chambers, and swivel tubes to deliver the blast.
- If the combustion chambers had not been strong enough to withstand the force of the blast they would have blown up. Even if the chambers were strong enough for normal circumstances, too great a concentration of chemicals at any one firing could generate too much heat and explosive force for the beetle's body to withstand. There has to be a regulating mechanism to precisely control the manufacture and mixing of the chemicals.

Though evolutionists might be able to invent a scenario in which such a mechanism could evolve one piece at a time by changes in previously existing components, they must once again ignore the details. Everything in the apparatus, from the mix of chemicals to the strength of the sphincter muscles to the shape of the storage and combustion chambers, exists in the beetle's body because it is coded for in the DNA. Once again we have to ask, what are the chances such a system could develop one mutation at a time? Very slim.

***e. Bioluminescence.***

Over a hundred species from at least three biological kingdoms are *bioluminescent*, that is, they have the ability to produce light by internal chemical reactions. At least one of these species is dated to the early Cambrian – the beginning of the Cambrian Explosion (DeLeo et al., 2024).

Visual #11-79

At minimum, any bioluminescent organism manufactures a light-producing chemical called a *luciferin* and an activating enzyme called a *luciferase*. (These are generic terms like protein or enzyme. Each species has a unique formula for the chemicals it produces.) These must be stored inside the creature, ready to be mixed when needed. In addition, many species such as cuttlefish and octopi have the ability to produce intricate moving patterns used for camouflage, hunting, etc. (See the 2024 *National Geographic* television series "*Secrets of the Octopus.")*

The luciferin, luciferase, storage chambers and control mechanisms are produced by information coded in the organism's DNA. So how could this system have

evolved? Some ancient line of non-bioluminescent creatures had to experience repeated mutations in DNA, building one upon another until all the correct parts had developed. Other bioluminescent organisms would eventually evolve from this common ancestor.

Visual #11-80

A major problem with this scenario: bioluminescence occurs not just in closely related species, but in varieties ranging from plankton to fungi to bacteria to soft-bodied invertebrates to insects to fish, and many more. A display at Chicago's Field Museum of Natural History says the phenomenon had to evolve over a hundred times independently, in organisms belonging not just to different species but to at least three different kingdoms.

Visual #11-81

Remember that Lamarckianism has been thoroughly falsified. Living things do not evolve the features they need. If anything is to evolve, it must be because of mutations in DNA.

Imagine how improbable it would be for bioluminescence to evolve in even a single species, then multiply that improbability by the improbability of every one of the other hundred or so species acquiring this ability. Evolutionists must totally ignore the laws of probability to maintain their faith in naturalism.

***f. Camouflage.***

Visual #11-82

Many animals have shapes, colors, or markings that enable them to blend in with their environment. These include the "walking stick" and other animals that look like leaves or twigs, fish that look like rocks, octopi and squid that can change colors and patterns, and many butterflies and moths. The latter have colors and designs that either make them hard to see or else fool predators into thinking they are poisonous.

The animals blend perfectly with their environment not because they *need* camouflage (Lamarckianism) but because it is programmed into their DNA. This is hard enough to explain when they look like inanimate objects such as rocks, but even harder when many of them look like surrounding plants whose features are also determined by *their* DNA. Either the two kinds of DNA evolved independently and just happen to fit together - *two* groundhogs crossing different superhighways - or else they were designed that way. When we remember the harmful effects of mutation and the cell mechanisms designed to prevent it, we see that it takes much more faith to believe in random chemical processes than in design.

***g. Symbiosis.***

The world is full of *symbiosis* or *mutualism*, in which members of unrelated species work together for their mutual advantage. A few examples:

*i. Insects and Plants.*

Many plants and insects depend on each other. For instance, bees feed on pollen from all sorts of plants. In the process of feeding they fly from one plant to another, spreading the pollen and enabling the plants to reproduce.

There are many examples of insect/plant interdependence, for instance, the desert yucca plant and the pronuba moth (Meldau, 1974, 114-116). The yucca blooms only at night, at certain times of the year. On the exact night it blooms, the moth breaks out of its cocoon, flies to a flower, gathers pollen, deposits it on a different flower, lays its eggs, then goes off and dies. When the eggs hatch the caterpillars eat their fill of seeds then lower themselves to the ground by a thread. They bury themselves in their cocoons and the process repeats. In those years when the plants don't bloom, the moths remain dormant in their cocoons. They only come out the very day the flowers bloom.

There are several species of the plant and several of the moth. Each species of plant reproduces with the aid of exactly one species of moth, and vice versa. Could this be the result of a fantastic series of parallel mutations?

*ii. Cleaning Symbiosis.*

Visual #11-83

- *Crocodiles and Plovers.*
  Crocodiles normally eat anything they can get in their mouths. However, they have a unique relationship with one species of bird, the Egyptian plover. When the plover approaches, the crocodile opens its jaws wide. The bird walks in, picks the leeches off the crocodile's gums, then walks safely back out. The crocodile gets a free cleaning and the bird gets a free lunch. (Barnett, 1960, 229)
- *Cleaning Stations in the Sea.*
  Marine biologists have discovered a number of "cleaning stations" in the sea. Fish of all kinds, including such voracious predators as sharks and barracudas, come to these areas and line up for cleaning. When one of the cleaners (usually small fish or shrimp) approaches, the predator opens its gills and mouth and allows the cleaner to swim in and remove fungus, parasites, and damaged tissue. When the cleaner is done it swims back out, the cleaned fish swims away looking for its next meal, and the next in line moves up for its turn. (Barnett, 1960, 240-241)

  Both of these are irreducibly complex systems requiring a cleaner willing to enter a predator's mouth and a predator willing to not eat it. If this mutual behavior is the result of mutations, we have to marvel at how fortunate the cleaners are. Just at the time they acquired a mutation that made them want to walk or swim into a crocodile, shark, or barracuda's mouth, the predator acquired a mutation that made him decide not to eat the cleaner. If the predator's mutation had come a little after the cleaner's, it would have been all over.

*iii. Hunting and Protection.*

Visual #11-84

The skunk clown fish (like "Nemo" in the Disney movie) is an ordinary little creature, except that it makes friends with the deadly sea anemone. Other fish stay away from the anemone because of its poison, yet it lets the skunk clown – as vulnerable to the poison as any other type of fish – swim around in its arms without attacking. The skunk clown receives protection from larger predators; in return, it brings back part of its prey to share with the anemone, which is unable to move in order to hunt food. (Barnett, 1960, 241)

***h. Migratory Birds*** (Gitt, 1986, 36-41)

Visual #11-85

Many kinds of birds migrate in the winter. Some, such as the ruby-throated hummingbird, travel thousands of miles over land. Others journey even farther over open water with no landmarks to guide them. These include the East Siberian Golden Plover, Alaska to Hawaii (4000 km); the North American Golden Plover, Labrador to north Brazil (about 5000 km); the Japanese snipe, Japan to Tasmania (5000 km); the needle-tailed swift of Eastern Siberia, Siberia to Tasmania (over 10,000 km); and the American sandpiper, Alaska to Tierra Del Fuego (16,000 km). If they were mistaken in their navigation by a fraction of a degree, they would miss their target and finally fall exhausted into the ocean. Yet they arrive at their destination year after year. How do they do it?

- Experiments have shown that they do not have to memorize the route. Different species have been shipped thousands of miles from home in crates that were periodically rotated to keep them disoriented. Yet when released, they find their way home.
- Perhaps they use the earth's magnetic field to help navigate, but so far we have not located a specific organ that would enable them to do so.
- We cannot explain their remarkable accuracy by saying they navigate only by

the sun and stars. These seem to make it easier, but the birds fly in all sorts of weather conditions. They don't get lost when it's cloudy.

Four factors are needed for successful migration: a point of origin, a destination, a means of locomotion, and a means of navigation. Though birds that fly over land might learn to navigate by landmarks, the navigation system of those that fly long distances over open ocean requires at least minimal function for survival.

The first birds are supposed to have evolved from reptiles hundreds of millions of years ago. Even if we were to accept these ages, fossils show us that they haven't changed much since. So when did they develop the ability to navigate by the stars or the earth's magnetic field? According to evolution, both the stars and the continents were arranged much differently millions of years ago than they are now. In addition, the magnetic field is supposed to have reversed several times. Whatever part of the birds' DNA gives them the homing instinct would have had to continually mutate since then to enable them to keep up with the changing arrangement of land, stars, and magnetism. Ridiculous, no? Such abilities could not evolve by trial and error. One error would take away the ability to make another trial. The birds would all fall into the ocean and drown. The alternative is obvious: they were designed that way.

Another amazing fact: such a lengthy nonstop journey requires more energy than an individual bird can store in its body. The birds overcome this problem by flying in V formation. The lead bird breaks the wind resistance for the others, enabling them to reduce energy consumption by 23%. When it gets tired it drops to the rear of the formation and the next bird moves up to take its place. The birds' cooperation with each other enables them to fly much farther than they could individually. Which better explains how they know what to do: purposeful design or random chance?

***i. The Human Eye.***

Visual #11-86

We saw that Darwin said that his theory would "absolutely break down" if any structure were found that could not be explained by the slow accumulation of minor changes. He admitted that the eye gave him a great deal of difficulty, but because of his presupposition that evolution must be true he made up a story about how it evolved anyway. Let's look at some features of the human eye Darwin didn't know about.

Visual #11-87

> The following is from "Design In the Human Eye" by Joseph Calkins, M.D., *Bible-Science News*, January 1992. The article may be obtained at https://creationmoments.com/article/design-in-the-human-eye/.

Visual #11-88

The retina lining the back of the eye is a thin, transparent membrane that contains millions of photoreceptors at a density of about 200,000 per square millimeter. This is many times greater than the concentration of circuitry on a computer chip. Some of these light receptors are rods, others are cones. They have a dynamic range of about ten billion to one: that is, they automatically adjust their "volume control" to enable you to see in light conditions ranging from dim starlight to bright sunshine. Compare this to the best photographic film, which has a dynamic range of about a thousand to one. The eye is ten million times better able to deal with changing conditions.

Each of these receptors is connected to a nerve that does a tremendous amount of preprocessing before sending the signal through the optic nerve to the brain. Dr. John Stevens, a professor of physiology and biomedical engineering, tells us (*Byte* Magazine, April 1985) that

"To simulate 10 milliseconds of the complete processing of even a single nerve

cell from the retina would require the solution of about 500 simultaneous non-linear differential equations one hundred times and would take at least several minutes of processing time on a Cray super-computer. Keeping in mind that there are 10 million or more such cells interacting with each other in complex ways, it would take a minimum of a hundred years of Cray time to simulate what takes place in your eye many times every second."

Though computers have become much faster since then, it would still take at least weeks or months for even the fastest ones to process the visual information your eyes take in each second.

Visual #11-89

Some skeptics claim that if the eye is designed at all, it is a poor design because the rods and cones are on the back of the retina instead of the front. This, they say, makes it harder for light to get to them. However, they ignore three key points:

1. The parts of the eye are so small that they are optically transparent. There is almost no loss as the light passes through the retina.
2. The eye has to continually keep itself in top condition. The "backwards" arrangement helps it to do so.

   Every time you look at a bright light some rods and cones are damaged. However, because the rods and cones are on the back instead of the front, they are in constant contact with nutrients and are able to repair themselves immediately. If the arrangement were reversed, it would take your eyes months to recover from a camera flashbulb instead of a few seconds.
3. The eye is far more complex than we knew just a few years ago. Scientists at Leipzig University reported in 2007 that there is no loss of light to the rods and cones because the eye uses microscopic fiber optic tubes to transmit the light to them (Franze, 2007). Humans only invented fiber optics a few decades ago. Little did we know that our eyes have used the technology since the beginning of humanity.

Consider this also: even if we follow Darwin's reasoning about a series of structures in nature that seemed more and more eye-like, any sort of eye would be useless without an optic nerve and a specialized area of the brain to interpret the signals it sends. This is an irreducibly complex system in which all the features work perfectly together. Could a series of random DNA mutations produce a coordinated group of structures that puts the fastest computers to shame, or is it perhaps more likely that somebody designed it all?

Many volumes are available for students wanting to learn more about design in nature. The point is: When we consider the complexity of DNA, the elaborate cell mechanisms geared to preventing mutations, and the fact that mutations damage preexisting genetic information rather than adding it, we are forced to the conclusion that evolution could not happen. When we look at the real world, we see that creation is a far more reasonable explanation of what did happen. Those who reject the possibility of creation (or any form of intelligent design) do so for religious reasons, not scientific.

Visual #11-90

## CHAPTER 11 REVIEW

Evolution rests on four "pillars": embryology, homology, biogeography, and paleontology. Embryonic recapitulation was a fraud. Homology and biogeography offer little support either.

I. DNA is the blueprint for the body structures of every living thing. Only three types of mutations (copying errors) would be available to pass on to future generations: those inherited from parents, those that occur during early embryonic development, or those that occur in the reproductive cells of an adult organism.

A. Every cell has at least three error checking and correcting mechanisms to prevent mutations.

B. Mutations are insufficient to account for the origin of new features in living organisms.

1. Different kinds of cells depend upon different kinds of rare elements. Random mutations in DNA are not sufficient to explain how the cells came to need these substances.
2. Mutations do not add genetic information; rather, they damage information that was already present.
3. While a few mutations benefit individuals, no mutation has ever been observed that gives the affected species a definite survival advantage over its unaltered relatives. For instance, birds with shrunken wings would die anywhere else but on their windy islands. Likewise, sickle cell anemia may give the individual a slight benefit, but it harms the species as a whole.
4. Since some plants and amphibians contain tens of thousands of times as many nucleotides in their DNA as the simplest cells do, evolution requires some mechanism to bring in all this extra genetic information. Mutations don't add anything, they just change what's already there. Viruses are not a good candidate either, since they harm or kill their host. Thus, the mechanism for adding information is unknown.
5. Bacteria and insects do not mutate when they develop strains resistant to antibiotics and pesticides. The genetic information enabling some individuals to resist these poisons is present all along. The rest die off, allowing the resistant ones to multiply and fill the ecological niche available.

C. Pleiotropy.

Almost every laboratory-induced mutation seen so far affects more than one body structure. A single mutation can have drastic effects on several body systems.

D. Pseudogenes.

The fact that some segments of DNA do not seem to code for amino acids is used as evidence for evolution. Instead, it is evidence for arrogance. Our ignorance of a function doesn't mean there is no function. It only means we are ignorant.

E. Protein Sequences.

Thousands of cell proteins have been studied. Each protein varies from one kind to another. Only one, cytochrome-C, can be used to show any kind of sequence that evolution could possibly have produced. No matter which protein we study, every member of any major group (kingdom, phylum, class, order, family) differs from every member of any other major group by a fairly constant percentage. Each protein varies by a different amount. There is no evolutionary explanation.

II. Homology - the second pillar.

Evolution says that similar structures in different types are due to common ancestry. This leads us to expect that homologous structures should be produced by homologous genes. They are not. Also, in many cases similar features arise from different areas in the embryo. Evolutionists have no way to explain these phenomena. Creationists interpret them as evidence of design.

III. Biogeography - the third pillar.

The members of a species may vary somewhat from one geographic location to another. There are two contradictory explanations:

(1) Evolution says they gained new features as they traveled.
(2) Creation says they had the genetic potential for specializing when they started.

The only way we could know for sure which is correct would be to follow the development of a species and analyze its DNA as it radiates outward from its starting point. No one has ever done this. However, since we have never seen any kind of creature evolve a totally new feature it seems much more likely that they started with more genes than they needed, then lost some as they became specialized to fit with various environments.

IV. Evidence of Design.

Throughout nature we see many phenomena that are extremely unlikely to have developed by random mutation.

A. The best arguments for design are
   1. Irreducibly complex machines in the cell that could not possibly have developed one step at a time from different types of machine.
   2. A convergence of probabilities against assembling multiple complex systems by random chemical action.

B. There are countless examples of design in nature. These include:
   1. DNA.
   2. Blood coagulation.
   3. Antibody production.
   4. Specialized mechanisms in the cell. Every cell, no matter how "primitive" or "advanced," uses the same genetic code, protein manufacturing mechanism, and method of DNA replication. There is no evidence that any kind of organism is the ancestor or descendant of any other.
   5. Specialized organs and structures that cannot be explained by a gradual accumulation of minor changes.
   6. Symbiosis, in which unrelated species depend on each other for their mutual benefit. They would have had to appear at the same time and in the same place. It takes more faith to believe they evolved independently and then became interdependent than it does to believe they were created to function together.

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