

Evolution & **Intelligent Design**

Session 9

Part III – Intelligent Design

Clarification: Intelligent Design is not a creationist organization that promotes a particular design agent as in the biblical account in Genesis. They separate their purpose from creationist groups like Answers in Genesis and ICR, even though virtually all researchers are men and women of Christian faith.

The reason for this caveat is that Intelligent Design desires to “introduce” the world to the Designer concept without alienating individuals who believe biblical creationism is unscientific.

1. History to Present

Key speakers, writers and lecturers like Phillip E. Johnson began late in the 20th century to explore the problem of increasing information in Darwinian evolution based on knowledge of DNA’s role in mutations; and the problems Darwin himself identified – complexities that couldn’t be explained by gradualism.

In the 1980’s, Michael Behe of LeHigh University began exploring irreducible complexities – less to destroy Darwinism as to bolster support for divine creation. Using the “*mouse trap*” example, Behe extended the concept to the eye, the bacterial flagellum, and the complex process for clotting of the blood.

Later, these observations were picked up by Stephen Meyer in the interest of formally declaring Intelligent Design as a discipline of science. Meyer understood and appreciated the implications of the complexities of Behe and others, but was convinced even more signs of design could be evidenced in the nature and functions of the cell. DNA, genes, RNA transcription, gene expression and the making of proteins have irreducible complexities by the truckload.

Meyer has focused on the cell (as in his book *Signature in the Cell*) but has also begun to capitalize on the problems of traditional Darwinism and the fossil record – hardly pursued anymore – specifically how to account for the information and development needs of the Cambrian Explosion.

Meyer helped to found Discovery Institute (www.discovery.org)

Immediate Reaction of the Science Community

Immediate reaction came in the form of name calling – the first line of attack for the progressive left. Few dealt with the research straight on. A review of *Signature in the Cell* by Francisco Ayala for the BioLogos Foundation ironically demonstrated that Ayala hadn't even read the book and was commenting on what he had heard others say about it. Such is the bias toward anything critical of evolution.

Rather than deal with specific claims, authors like Richard Dawkins chose to call Behe worthy of sympathy rather than disdain for having disengaged from real science at the behest of his religious beliefs. Typical British snobbery accompanied Dawkins' recommendation that Behe be pitied for having fallen too willingly into the ditch – a clever way to dismiss the need to address Behe at all.

In subsequent years, researchers began to counter Behe's points with observations and explanations they believed effectively contradicted Behe (discussed later). Many of these rebuttals undermined some aspect or other of the main corpus of Behe's thesis, but failed to answer significant remaining points.

Respecting the centerpiece of Behe's work – irreducible complexities – the feasibility of some parts being already in use in other functions failed to explain how 75% of the remaining parts not yet in existence came about through natural selection. It is a recurrent case of answering a few items of minutia and letting them stand for a comprehensive explanation.

David DeRossier of Brandeis University succeeded in explaining where some parts might have come from in certain complexities, but declined to mention how that explanation couldn't account for the majority of other parts nowhere available. No matter – Behe was shown to be wrong.

Kenneth Miller of Brown University attempted to explain that supposed irreducible complexities could easily have had intermediate use as other functions – offering the partial mousetrap as a crude but functional tie clip.

This is really where the rub comes in for all “demonstrations” of evolution. Every supposed evidence that evolution has occurred at the macro level involves an appeal to minute observations that *microevolution* has occurred; but then moves to assumptions, in many cases circular assumptions that these lead to macroevolution.

For example, we know the genetic pathway for domestic cats in relation to larger more diverse cats like bobcats, mountain lions, tigers, leopards, panthers. But one can't name the dinosaurs prior to mammals who had to be their supposed direct ancestors without employing assumptions or suppositions.

Ergo – macroevolution – the key conclusion of the theory that explains the origin of all life forms and species - must be *believed* to have been accomplished by microevolution working over long periods of time in order to finish out the theory.

Creationism in Disguise

Evolutionists are quick to leap to the conclusion that ID is unscientific because it's just creationism with a new hat, disguised surreptitiously to get an inroad back into good standing with science.

Conversely, Intelligent Design begins with observations in science – like molecular biology and biochemistry – and poses a new hypothesis that better explains the observations than evolution.

ID Can't Discover Its Chief Conclusion – the Designer

Evolutionists also reject ID as pseudo-science because the Intelligent Designer in this hypothesis can't be discovered by making or repeating the observations and can't otherwise be discovered as the cause of the observations.

So if scientists will see the need in the end to believe an Intelligent Designer exists, why bother proposing ID?

Ironically, neither can evolution be observed as the origin (in the macro sense) of all life forms. It is at best a set of assumptions that joins disconnected facts. If an assumption (devoid of proof) about the actual process of evolution is allowed, why can't an assumption (devoid of proof) be allowed for ID?

Non-repeatable or Non-observable Processes

Darwinism asks the scientific method to examine what evolution has supposedly left behind that might describe what happened in the past. As such, evolution has observations that can't be repeated. So Darwinism can't be charged with failing the method just because its past action can't be observed.

Furthermore, extrapolating past processes as those working today has the expectation that we should be able to observe the current day's processes in action. Again, the pace of evolution is such that it rules this out because it operates too slowly for one observer or even many observers to capture or test.

In contrast, half-lives of radiometric materials are also too slow to observe them happening. Yet science never balks at using an extrapolation method to establish half-life.

2. Problem of Increasing Information

Can Mutations Account for the Origin of All Life Forms?

The New Darwinist assumption is that, given enough time, mutations in the DNA beginning in the first living organism could produce all the life forms both extinct and living today.

Two Questions:

- 1) The sheer capability of DNA to add all the new information needed?
- 2) Has there been enough time?

Capability of adding all the new information needed?

Humans have 3 billion information units (base pairs) in their DNA

First life is not assumed to have had only one unit. Let's assume 160,000 to 12 million units in the DNA of first life

Primitive DNA must have been capable of adding 2.98 – 2.99 billion additional units to its DNA to arrive at human DNA.

$$3 \text{ billion} - 160,000 = 2.99 \text{ billion} \quad 3 \text{ billion} = 12 \text{ million} = 2.98 \text{ billion}$$

Creationists Phillip Johnson and Stephen Meyer claim primitive DNA cannot add that much new information, especially new body plans yet to exist.

Evolutionists remind us that new information simply means new combinations of the four basic units (nucleotides) ATGC and they only come in matched pairs. Thus, all the building blocks of the language are already in the genes of the DNA. So we're talking about new insertions/additions/duplications of a finite number of already existing possibilities.

But having all the letter combinations available in an unguided process does not ensure an intelligent instruction will arise. Having our 26 English letters merely present with a process that might accidentally recombine them does not assure us that words we can understand will result given enough time.

Enter the famous Monkeys and Typewriters parody of Bob Newhart.

One of Bob Newhart's skits - *An Infinite Number of Monkeys* - featured an experiment that tested the hypothesis that if you corralled an infinite number of monkeys in a room with typewriters, given enough time, they would eventually type all the great books. The skit is about a man who is monitoring the monkeys and has found something notable.

"Uh Harry hold on, post 15 has something. I think this is famous . . . Ahh, 'to be or not to be . . . that is the gzornenplat!'"¹

¹ © Warner Bros. Records Inc, 1960

The lesson here for evolution is how long the observer will have to wait until another monkey comes up with that phrase and completes it correctly. Also, the parody requires an observer in order to recognize something useful. The monkeys themselves have no capability to know when something useful has been produced or how to produce it again. This is what one gets with random chance.

In the cell, when a successful protein has been made, no independent observer signals the front end of the process (DNA and genes) that something useful has been produced and let's have another.

But in reality there's no need for a signal to the front end since the gene will automatically continue to produce the instructions for the protein whenever it is needed. The question is: how did the *need* for the protein arise simultaneously? The protein must be sent to the site needing its effect. Initially this process only works in one individual organism but it can be passed on to offspring whether beneficial or not - *unless it is undone by the next mutation*.

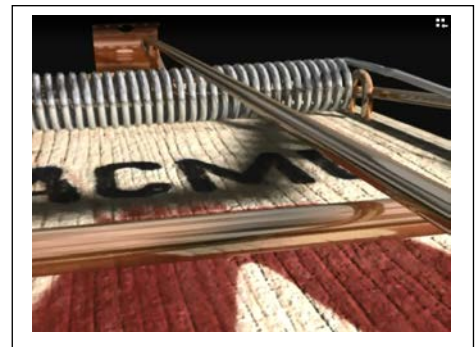
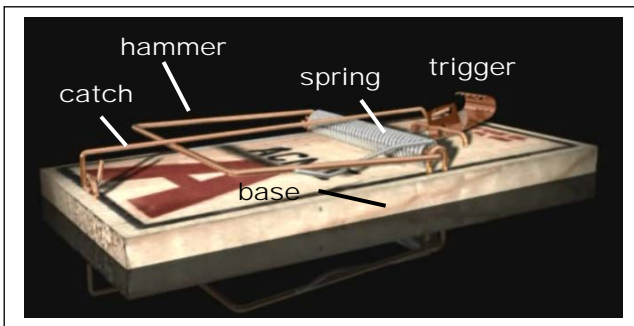
3. Irreducible Complexities

Definition: a collection of components or features that will not perform their functions unless all components are present at the same time. i.e an irreducible complexity cannot arise gradually in parts because the parts will not bestow an advantage until all the parts are present and properly inter-related.

The Mouse Trap Example

Michael Behe was first to gain notoriety for this example as a simple illustration of irreducible complexity.

A mouse trap has a base, a frame, a spring, a catch for the spring, and a moveable bar (*hammer*).



The explanation is that if any single part is lacking (or yet to be provided) the mousetrap is of no use (bestows no advantage).

This is what gradualism proposes – that states will exist along the path with parts “yet to be provided” and these intermediate states are useful and bestow an advantage.

But if the base was created first by itself, it bestows no advantage. Any other part coming first by itself gets the same result. Even if we pretend that some advantage is gained by the first component existing by itself, the addition of another single component does not improve the situation.

In essence, all the components must be present and properly inter-related before the mouse trap can perform the function that bestows its advantage.

Richard Dawkins attempted to debunk this example by claiming that biological components had another function (in another application) before being copied and reused in the present complexity. They were re-assigned by a mutation that brought them together.

An example is that given by Kenneth Miller of Brown University. A beginning mousetrap that had only the base, spring, and hammer wouldn't make a mousetrap, but would make a crude tie clasp:



Three parts of a would-be mousetrap could be used intermediately as a tie clasp. While true, it isn't applicable since the tie clasp would simply be further developed by adding what was needed for the trap. The tie clasp disappears (is absorbed) in the arrival of the trap.

But in DNA and protein manufacture, the other applications Dawkins talks about that use the partial features of the future mousetrap continue to be made by DNA. The DNA must borrow these successful features for use elsewhere in the DNA in order to evolve the mousetrap.

This borrowing of gene sequences in DNA is really not a known operation of DNA. Instead, it's simply a matter of the same gene sequences mutating by chance in another part of the DNA –

were the new application will arise. Nor is there a learned awareness in DNA that volunteers certain sequences to be available when genes mutate.

So it's purely a matter of chance that the same combination of base pairs will arise again independently in another area of the DNA, bestowing the additional components for use there.

Blood Clotting

Clotting involves a cascade of protein effects and conditions which have to be present in the blood but not active until clotting is needed.

Clotting is effected when a material called *fibrin* forms a web-like assembly of proteins that capture blood cells when needed.

But fibrin is always present, so it needs an inactive form while it exists in the blood until needed, else all blood everywhere would be clotted from the presence of fibrin. Fibrin's soluble inactive form is called *fibrinogen*. However a mechanism is needed to turn fibrinogen into fibrin.

A molecular protein called *thrombin* cleaves the ends of fibrinogen, converting it to fibrin and making it sticky and able to attach to other fibrin molecules undergoing the same process.

Similarly, when the clot needs to be removed, another mechanism must act on the fibrin, else the clot would remain at the site indefinitely. As it happens, fibrin is actually at the end of a cascade of actions used to trigger fibrin and involve timing and location so as to prevent clotting from occurring at the wrong time at the wrong place.

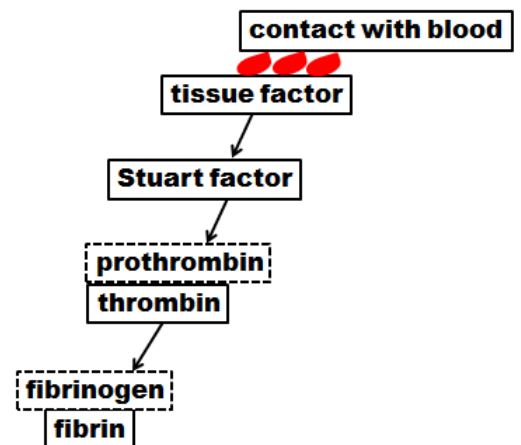
But thrombin cannot exist in its active form all the time either else it too will always be cleaving fibrinogen which will always be converting to fibrin and the blood will constantly clot. The inactive form of thrombin is *prothrombin*.

But here the cascade of dependencies continues upward. Prothrombin must in turn be regulated as to when to convert to thrombin. Its control molecule is something called the *Stuart factor*. And Stuart factor must in turn be regulated by another component called *tissue factor*.

Finally, the tissue factor is triggered by contact with blood in the case of a cut or injury of cells which brings them into contact with the circulatory cells.

So the cascade is as follows:

- tissue factor cells not normally in contact with blood
- a cut or injury causing bleeding
- tissue factor come in contact with blood
- tissue factor triggers Stuart factor to begin controlling prothrombin



- triggered by Stuart factor, prothrombin begins to create thrombin
- thrombin cleaves fibrinogen and converts it to fibrin
- finally, fibrin forms the clot at the time and place needed.

So why not have the presence of blood simply trigger fibrin to be made from fibrinogen? Each factor or functional molecule lacks the information or control feature to regulate location, timing and duration. Each of the molecules in this cascade contribute some form of control the component below it lacks.

Evolution, therefore, is not able to demonstrate how this cascade could come into existence step-by-step. Fibrin could not have come into existence first and let the other controls come later. The organism would die while waiting. Tissue factor could not come into existence and affect fibrin because it wouldn't have the intervening controls needed to regulate or even make fibrin from fibrinogen. Again, the organism would die waiting.

Human Eye

As earlier, the first primitive photo-sensitive spot without lens or visual focus would be an advantage only if it was accompanied by a nerve and the intelligence at the brain to interpret the signals received. This makes it an irreducible complexity even at this primitive stage.



In very general terms, a photosensitive spot would logically seem an improvement over no spot at all. But when we probe deeper into how the spot would be beneficial, problems arise for evolution.

Each of the white lines represents a separate mutation. The first appearance of a photosensitive spot at far right would be of no use to the organism if its signals go nowhere. Another mutation is needed to provide a nerve.

Still not of any use if not connected to the brain, another mutation must provide a

connection. Still of no use until the brain can process the new signal types, another mutation is needed to add processing to the brain.

If each of these mutations appears separately, the question is how the organism is improved in stages while waiting for the complete arrangement to finish development?

Toward a Complex Eye

Darwinists love to present a plausible path from the photosensitive spot to the complex eye in selected steps on a chart like the one at right.

But where did such a chart originate? The first mistaken assumption is that each of the phases is supported by some fossil giving evidence of the eye at this step. Of course, fossilization would not preserve the soft organ tissue of the eye.

Another possibility is that scientists have found living organisms that utilize one phase or another of this picture and find the function helpful and useful.

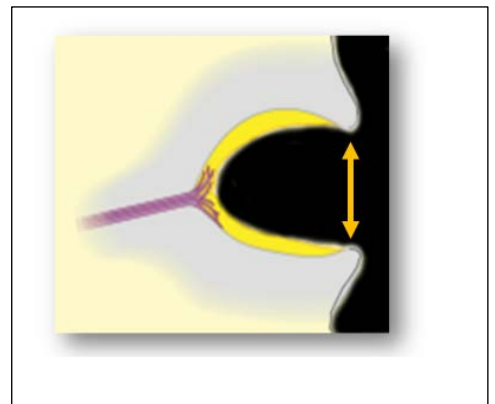
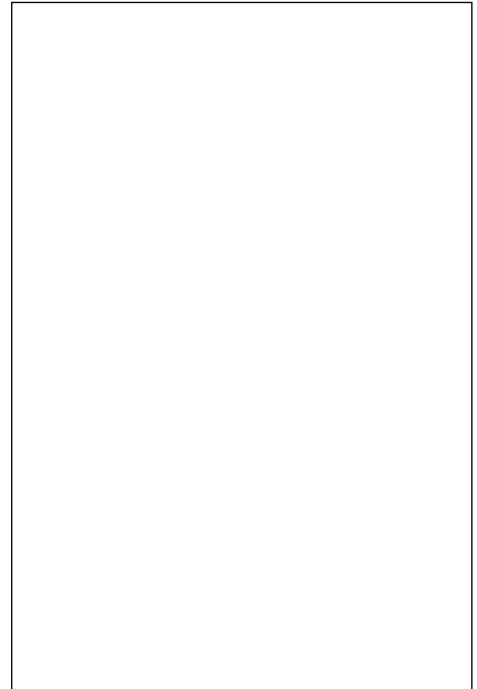
Unfortunately, this gets us no closer to how the eye developed in reality in ancient organisms if referring to modern species. We wouldn't offer a conclusion that T-Rex lived 65 million years ago on the basis of examining a lizard today.

In terms of usefulness and improvement, the second step in frame 2, upper right, forms a cavity or chamber and moves the light spot to the posterior of the chamber.

While this looks visually encouraging to researchers because it is the first move to an eye socket, it isn't clear how this would benefit the organism over having the spot out on the surface. Formerly light from anywhere could strike the spot, now only light coming in through the opening is sensed.

The question is whether the organism is better having its light limited to the opening?

(see the illustration below)

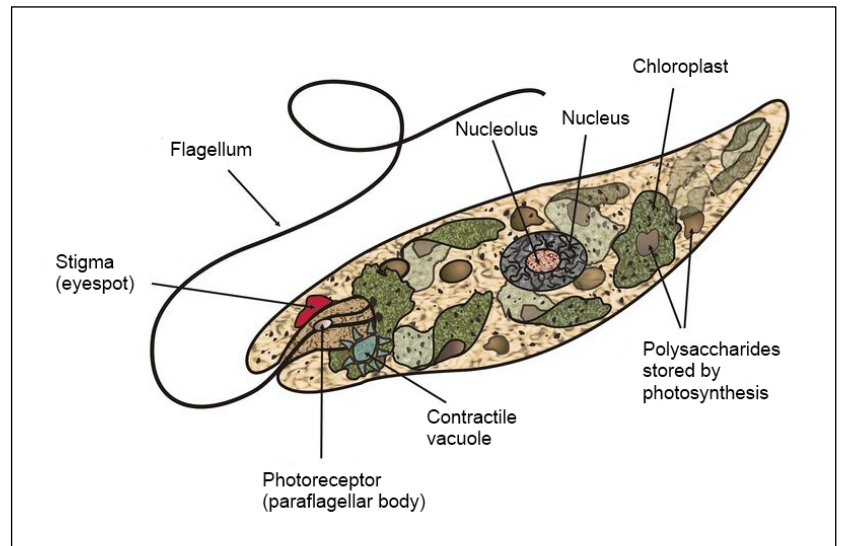




Light sensitivity for a spot out on the surface is similar to the view on the left – open and unrestricted. Moving the spot into a chamber with an opening is similar to viewing the same scene as shown at right.

Also Darwinistically, it isn't clear whether the path in such an organism actually leads to a species with a retina, lens, iris etc. This is because such a lineage or pedigree of life forms can't be established except through supposition.

Eugelena



While modern life forms exist with a sensitive spot, no prehistoric organism with a primitive spot has ever been found, since the flesh of ancient marine life was never preserved.

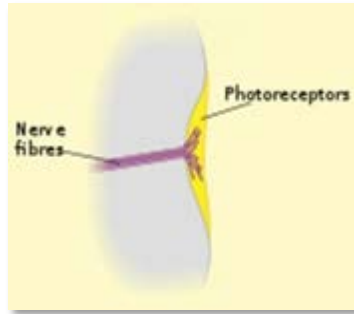
In the case of modern counterparts, the organism has the critical addition – to also sense and interpret light in the nervous system and brain. Phytoplanktons use photosynthesis for energy conversion. But even with these examples, how does one establish that such an organism in prehistoric times leads to an elaborate eye in other organisms?

Moving back from the fully developed eye, how do we suppose a form missing any component present today could have been at an advantage?

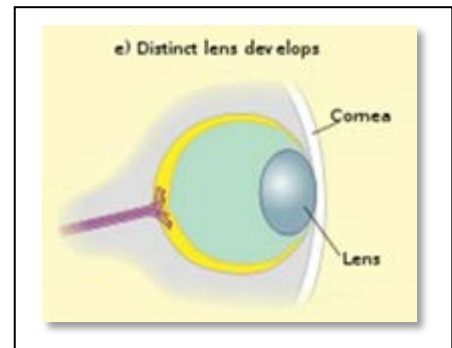
The Components

One must begin with the retina even if one excludes color vision with rods and cones. That would be the one counterpart to the most primitive component - the photo-sensitive spot. At some point we have to move from spots on the surface to spots behind a lens.

The photoreceptor must move back into a chamber and spread out along the posterior which then needs the intermediacy of a lens initially missing.



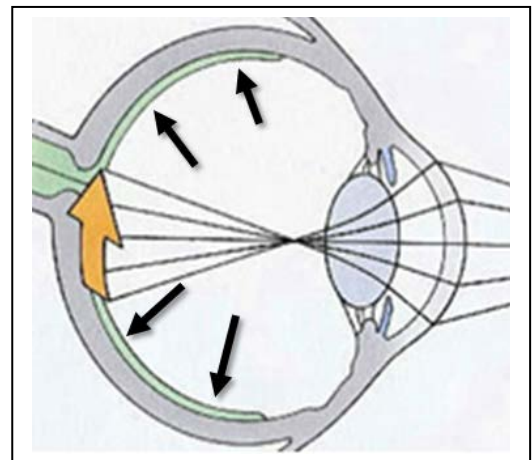
If a lens does develop by random mutations, how can it be of the right refraction to focus light on the retina? Failed attempts that put an inadequate lens in place or off center would render that individual less adapted and eliminate him.



While organisms with the recessed retina await the arrival of the correct lens, how are they benefited by this arrangement in the meantime?

The lens also focuses on a portion of the retina. How would evolution account for the areas of the retina not targeted by the lens?

Also, the modern lens changes shape to accommodate different focus conditions. How would this feature have evolved by trial and error and been useful in the interim?



The Iris

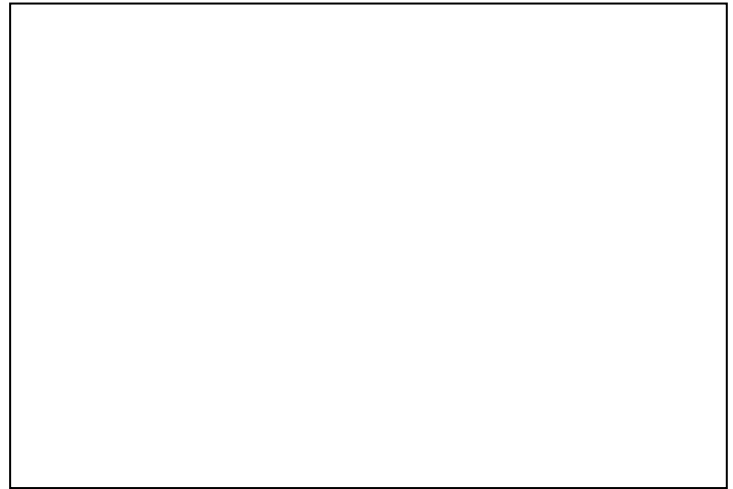
The modern iris is comprised of two sets of muscles that produce the sizing of the pupil as needed:

Both circular and radial muscles are employed to contract or dilate the iris.

The circular muscles actually cause the constriction or dilation. The radial muscles (far right) control how much or how little.

What if evolution produced the radials first? What would they do, since they have no circular muscles yet to control?

How would the intermediate effect be beneficial in the interim?



Complexity in the Retina

The eye has arteries that nourish the retina and the photoreceptor elements comprising them. These vessels exist directly on the surface of the retina that is supposed to receive all focused imagery from the lens. In retina exams at an ophthalmologist's office, the patient can sometimes see these vessels when light is first shone directly onto the retina. *So why don't we see these arteries all the time as in figure B below?*

A curious property of our rods and cones within the retina layer allow them to "compute" the image that is blocked by the shadow of the retinal vessels.

Motion picture production uses computer graphics integration (CGI) to allow images generated in a computer to be merged with regular filmed images. CGI is also used to remove unwanted items like wires, replacing them with computed pixels for the blocked areas.

Similarly, our rods and cones replace imagery that ought to be contacting our retina but is blocked.

Figure A



Figure B



The question is how a mutation could arise whose feature is to compute imagery from adjacent imagery? Nor is it a matter of making up pixels, but creating impulses the photoreceptors can pass on to the optic nerve.

