The Eye: An Evolutionary Dilemma—Part 2

Staff Writer

If we were to look at a cross-section of a human eye, we would see that it is rather like a camera. The “film,” or retina, is towards the back where the optic nerve comes in. The retina, like film, is light sensitive. It doesn’t *store* the image, rather, it converts the photons of light that come into the eye to some sort of electrical signal. This signal is sent to the brain for processing which is then interpreted as vision.

The interior of the eye is actually black. If you think of what color the interior of a camera is, this is about what you would predict. You can’t have a bright interior in the camera because light starts bouncing around and creates flare. So the interior of the eyeball is black.

The eye is filled with a gelatinous material somewhat like Jello, which helps to hold its physical shape. You see, we just can’t take the eye and flatten it or lengthen it without affecting its focus. After all that’s the way a camera focuses. We move the lens away from the film plane to focus up close, and we move the lens toward the film plane to focus at a distance. The eye does not change its shape; it does not focus by running its lens back and forth in relationship to the retina. The eye uses a much more sophisticated system that I’m not aware that anyone has tried in a camera. It would be much to difficult to carry off.

The lens actually changes shape. That is, it dynamically changes its shape so that it has a different refraction depending on its shape. Out in front of that lens, we have the iris diaphragm that behaves exactly the same as a diaphragm does on a camera. Again you need to have a little shade that closes in around the lens and opens again, to change the f-stop, as it were, to adjust for varying amounts of light. And finally, out in front we have the cornea, which is a transparent sheet that’s related to skin. In the eye the lens is sitting just behind the iris diaphragm, and it’s held in position by little filaments that run out to the periphery of the eye. The lens is actually suspended by filaments called zonule filaments that hold it in sort of 3-dimensional space. These filaments are under tension that pull the lens, and because the lens is flexible and can change shape, it tends to be held in a flattened position.

This is the position the lens would be in when it’s focused at a distance. This tension is built into the architecture of the eye. When the focusing muscles of the eye are called into play to change the shape of that lens, let’s say to restore it to its round position for close focus, these muscles cause slack in the suspending filaments that hold the lens. The lower tension in these filaments allows the lens to become more round, which is its intrinsic shape. Unfortunately with age the flexibility of the lens changes, and that’s why it becomes necessary for us to take over some of this focusing function with glasses that are either bifocals or trifocals. The lens is not able to turn over its cells like most other tissues of the body. These cells are permanent. Once they’re formed they can never be replaced again. They never divide and the lens sometimes hardens.

Let’s take a brief look at several parts of the eye. The cornea is the window through which our eye peaks. Most of the cornea is made out of a tissue that we call collagen. That is the connective tissue of the body. This must be kept transparent and requires a certain state of hydration to be transparent.

The iris diaphragm sits in front of the lens. Notice that the diaphragm has to be opaque to light and indeed it does have a black pigment in it. The pigments in the iris diaphragm also impart the color that we associate with people's eyes. Now there is a layer of muscle in that diaphragm that's arranged like a purse string, so that when it contracts it causes that diaphragm to make a small hole. And then there are radial muscles that splay out in all directions from the aperture of the iris diaphragm when they contract, they open the pupil and make it larger. One of the beautiful complexities built into the eye is this: when we focus up close and our lens is allowed to become round, we induce a considerable amount of spherical aberration in the lens of our eye. Automatically the iris diaphragm closes down over the lens to use a smaller portion of the lens to limit spherical aberration. This is why when we read and look at something closely, we need more light than we normally need, because our iris diaphragm is automatically closing down to correct for spherical aberration as we focus closely and that lens rounds up.

We could go into thousands of complexities of the eye. As one example, think about what an ophthalmologist might see it when looking into your eye with an ophthalmoscope. The back of the eye is an area on our body where the microvascular or small blood vessels are very close to the surface. They're right on the surface of the retina and by looking into the eye we can see the status of these blood vessels quite easily. This can be an indicator of health, just general body health as well as health and condition of the eye. The remarkable thing is that we actually look through that plexus of blood vessels. They're in front of the retina and our brain filters out this information so that, although they are there, and although we are seeing a network of blood vessels in front of us, they do not enter into our vision.

There is a little interesting experiment you can do to demonstrate that in fact you do look through your own blood vessels. Shine a slide projector on a screen, but with no slide in it, so you have just a bright illuminated screen. Then take a card and punch a hole in it with a paper punch. Look at that screen and wiggle that card back and forth so that the hole quickly blocks your view, opens your view, blocks your view, opens your view. When you do this, what happens is that changing light causes a shadow to be cast on your retina from these vessels so that the vessels then appear to be in a little different position. And because the shadow is in a little different position and you have not learned to filter that particular image out, you will suddenly see that in your line of vision up on the white screen is a plexus of blood vessels. Imagine evolving an eye. You do have to keep it alive, which means it needs a blood supply. Does it make sense to "ruin" everything by putting the blood supply on top of, covering, the very tissue that needs to be our sensitive surface for vision?

Let's look at the retina now. There are ten million light sensitive cells in the retina. There are approximately 200,000 per square millimeter (a millimeter is about the thickness of the edge of a dime) in its most sensitive part. These light-sensitive cells are of two basic varieties, the rods and the cones. The rods are sensitive to very low levels of light. In fact, they're sensitive to as little light as a single photon. This photon of light is capable of converting a chemical in these rods called rodopsin into a different form that somehow gets translated into vision. The interesting thing that evolutionists get excited about is that visual pigments like rodopsin are even found in microorganisms. We're not quite sure what they're doing there, but they say here's a protein that's just been waiting around to be used in an eye. I don't know if I'm entirely convinced by that argument.

The other type of the cells in the eye are the cones, and these are the cells that are

sensitive to color vision. Rods are only sensitive to basic black and white. Now in a camera, we can only tolerate a certain range of light; a camera with a typical type of film can take a dynamic range of about 1000-to-1. In other words, the brightest thing you can perceive and the darkest thing you can perceive would be about a 1000-to-1 ratio. In the case of the human eye, the ratio or the dynamic range is ten billion-to-1. One of the tricks that apparently is employed is that rodopsin itself can bleach in the presence of light. These 10 million cells that are the photoreceptor cells of the eye are quite sensitive to damage—when we look at the sun or an arc-welder, we destroy many of them right away—but fortunately they grow back. In fact, the turnover time of these cells in the eye is about 7 days, so they have to be made over and over again.

Now the photoreceptor cells are incredibly complex. They behave like high-gain amplifiers and they do signal processing. That is, when light comes in they take this light and they process this light in a way. There are nerves that form an association with these cells and these nerves finally go to the visual cortex of the brain. There are actual mathematical calculations that are apparently involved in the signal processing. It's been estimated for example, that there are about 10 billion calculations that occur every second in the retina.

Not too long ago a biomedical engineer by the name of John Stevens tried to mimic some of this signal processing that occurs in the retina of the eye with the aid of a very powerful mainframe. He concluded, regarding trying to simulate the signal processing that goes on in the retina: "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, nonlinear, differential equations, 100 times and would take at least several minutes of processing on a Kray 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 100 years of Kray computer time to simulate what takes place in your eye many times every second."

A conservative conclusion from all of this is that chance mutation could not provide the richness of complex anatomical and biochemical structure necessary to give natural selection any chance whatever to produce an eye. Rather, too many mutations would destroy the very eyes that we have.