Why the Inverted Human Retina Is a Superior Design

Jerry Bergman* **and Joseph Calkins****

Abstract

It is often claimed that the human retina is poorly designed because light must travel through the nerves and blood vessels to reach the photoreceptor cells, which are located behind the eye's wiring. Many specific reasons exist for this so-called backward placement of the photoreceptors. A major one is that it allows close association between the rods and cones and the pigment epithelium required to maintain the photoreceptors. It is also essential in both the development and the normal function of the retina. Both the rods and cones must physically interact with retinal pigment epithelial cells, which provide nutrients to the retina, recycle photopigments, and provide an opaque layer to absorb excess light.

Introduction

One of the most common examples of putative poor design in both the popular and scientific literature is the mammalian retina. The retina is the thin, light-sensitive organ located at the back of the eyeball. The claim is made that the vertebrate eye is functionally suboptimal because the retina photoreceptors are oriented *away* from incoming light (Ayoub, 1996, p. 19). Oxford professor Richard Dawkins considers this an example of poor design because he concludes that an

> engineer would naturally assume that the photocells would point towards the light, with their wires leading backwards towards the brain. He would laugh at any suggestion that the photocells might point away from the light, with their wires departing on the side *nearest* the light. Yet this is exactly what happens in all vertebrate retinas. Each photocell is, in effect, wired in backwards, with its wire sticking out on the side nearest the light. The wire has to travel over the surface of the retina, to a point where it dives through a hole in the retina (the so-called 'blind spot') to join the optic nerve. This means that the light, instead of being granted an unrestricted passage to the photocells, has to pass through a forest of connecting wires, presumably suffering at least some attenuation and distortion (actually probably not much but, still, it is the principle of the thing that would offend any tidy-minded engineer!) (Dawkins, 1986, p. 93).

Tuffs University Professor Daniel Dennett argued that, although the eye design is brilliant,

> it betrays its origin with a tell-tale flaw: the retina is inside out. The nerve fibers that carry the signals from the eye's rods and cones (which sense light and color) lie on top of them, and have to plunge through a large hole in the retina to get to the brain, creating the blind spot. No intelligent designer would put such a clumsy arrangement in a camcorder, and this is just one of hundreds of accidents frozen in evolutionary history that confirm the mindlessness

* Jerry Bergman, Ph.D., Biology Department, Northwest State College, 22-600 State Rt 34 Archbold, OH 43543, jbergman@northweststate.edu.

** Joseph Calkins, MD, ophthalmologist at Eye Group of Lancaster County, Lancaster, PA., jcalk@epix.net

Accepted for publication June 11, 2008

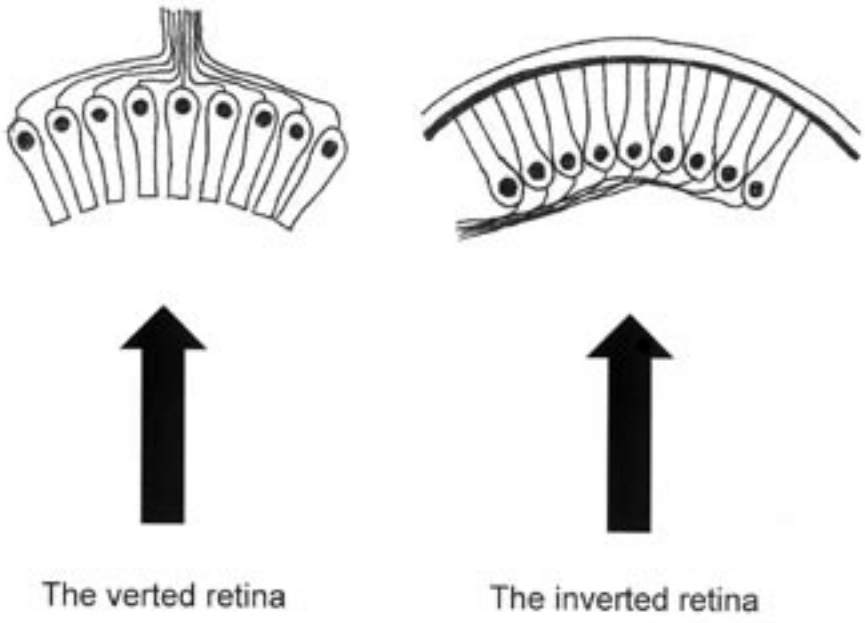

Figure 1. The basic design of the verted (left) and inverted (right) retinas, showing the light-sensitive cells. The arrow shows the direction the light travels into the retina. Note in the verted retina (left) the light-sensitive cells face toward the light, and in the inverted retina (right) design used in humans the light-sensitive cells face away from the light source. Drawing by B. L. Lindley-Anderson after Land and Nilsson (2005).

> of the historical process (Dennett, 2005, p. 4).

Williams claimed the retina is not just an example but one of the *best* examples of "poor design" in vertebrates that proves a "blind watchmaker" created life.

> Every organism shows features that are functionally arbitrary or even maladaptive…. *My chosen classic is the vertebrate eye*. It was used by Paley as a particularly forceful part of his theological argument from design. As he claimed, the eye is surely a superbly fashioned optical instrument. It is also something else, a superb example of maladaptive historical legacy.…Unfortunately for Paley's argument, the retina is upside down. The rods and cones are the bottom layer, and light reaches them only after passing through the nerves and blood vessels (Williams, 1992, p. 72, italics added).

Williams (1992) admitted that the vertebrate eye still functions extremely well in spite of the backward retina and argued that this does not negate the "fact of maladaptive design, however minimal in effect," which disproves "Paley's argument that the eye shows intelligent prior planning" (p. 73). Barash and Barash (2000) even claimed that the human

> eye, for all its effectiveness, has a major design flaw. The optic nerve, after accumulating information from our rods and cones, does not travel directly inward from the retina toward the brain as any minimally competent engineer would demand. Rather, for a variety of reasons related to the accidents of evolutionary history plus the vagaries of embryonic development, optic-nerve fibers first head away from the brain, into the eye cavity, before coalescing and finally turning 180 degrees, exiting at last through a hole in the retina and going to the brain's optic regions (p. 296).

After noting that the backward retina is a "classic" example of the "stupid features which support the idea that they are the result of evolution by natural selection" Frymire (2000) concluded that the inverted retina "results in an absurd situation in which the light has to travel through blood vessels and nerves before it reaches the rods and cones" (p. 36). Diamond (1985) added that, of all of our features,

> none is more often cited by creationists in their attempts to refute natural selection than the human eye. In their opinion, so complex and perfect an organ could only have been created by design. Yet while it's true that our eyes serve us well, we would see even better if they weren't flawed by some bad design. Like other cells in our bodies, the retina's photoreceptor cells are linked to a network of blood vessels and nerves. However, the vessels and nerves aren't located behind the photoreceptors, where any sensible engineer would have placed them, but out in front of them, where they screen some of the incoming light.…By contrast, the eyes of the lowly squid, with the nerves artfully hidden behind the photoreceptors, are an example of design perfection. If the Creator had indeed lavished his best design on the creature he shaped in his own image, creationists would surely have to conclude that God is really a squid (p. 91).

Kenneth Miller claimed that a prime example of "poor design" is the fact that in the human eye light has to travel through the neuron layers before it reaches the retina photoreceptors. He argued that this design provides clear evidence that the eye evolved by muta-

tions and natural selection and was not designed. An intelligent designer, he maintained, would not have placed "the neural wiring of the retina on the side facing incoming light. This arrangement scatters the light, making our vision less detailed than it might be" (Miller, 1999, p. 10). Thwaites (1982) argued that the inverted retina problem hits at the *core* of the design argument, historically a major basis of theism, because the "vertebrate eye shows poor design when compared to the eye evolved by the cephalopods" because vertebrates see everything

> through the nerves and blood vessels of the retina since the photosensitive elements of the retina are on the far side of the retina away from the light source. Clearly the cephalopod solution to retinal structure is more logical, for they have the photosensitive elements of the retina facing the light. Certainly the creationists need to explain why we got the inferior design. I had thought that people were supposed to be the Creator's chosen organism (p. 210).

Shermer claimed that anatomy of the human eye shows it is not intelligently designed because it is

> built upside down and backward, with photons of light having to travel through the cornea, lens, aqueous fluid, blood vessels, ganglion cells, amacrine cells, horizontal cells, and bipolar cells, before reaching the light-sensitive rods and cones that will transduce the light signal into neural impulses (Shermer, 2005, p. 186).

Williams (1997) added that "our eyes, and those of all other vertebrates, have the functionally stupid upside-down orientation of the retina" and that the "functionally sensible arrangement is in fact what is found in the eye of a squid and other mollusks" (pp. 9-10).

The so-called inversion of the retina is considered a suboptimal design primarily because of its simplistic comparison with a camera. Diamond argued that placing the rods and cones at the bottom layer and requiring light to pass through the nerves and blood vessels is the opposite of how an engineer would have designed the eye, and "a camera designer who committed such a blunder would be fired immediately" (Diamond, 1985, p. 91). And Edinger (1997) concluded that the "vertebrate eye is like a camera with the film loaded backward…if an engineer at Nikon designed a camera like that, he would be fired" (p. 761). This conclusion is based not only on the assumption that placing nerves and blood vessels in *front* of the retina reduces the retina's overall effectiveness, but also that another design would, as a whole, be superior. An evaluation of this argument reveals it is not only naive but also grossly erroneous.

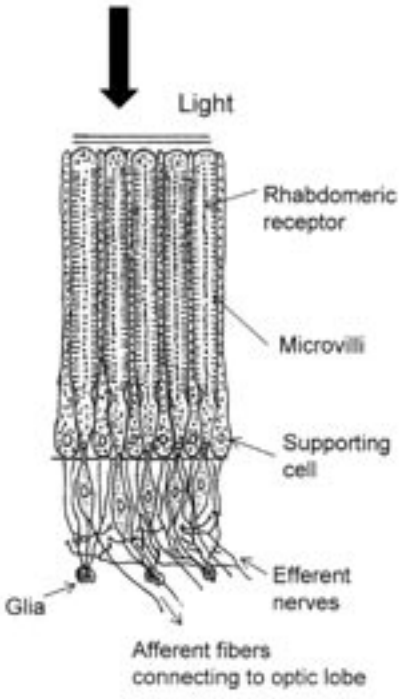

Figure 2. A cross section of the cephalopod eye receptors, called rhabdomeric receptors, which contain light-sensitive cells directly exposed to the incoming light. Note that the function of the pigment retina epithelium is served by the supporting cells located between the light-sensitive cells, reducing their number. Drawing by B. L. Lindley-Anderson after Land and Nilsson (2005).

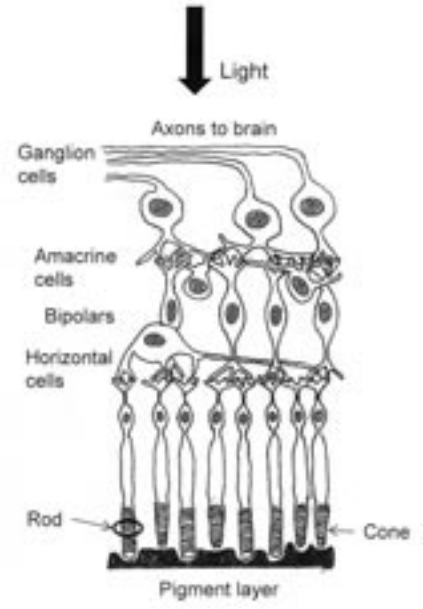

Figure 3. A cross section of the vertebrate retina, showing the retina pigment epithelium and other retina structures. Note the ganglion cells, the amacrine cells, the bipolar cells, and the horizontal cells. These are some of the structures that light must pass through before striking the photocells, the rods and cones. The retina pigment epithelium absorbs the light and supplies the photocells with nutrients among other functions. Drawing by B. L. Lindley-Anderson after Land and Nilsson (2005).

Verted and Inverted Eyes

Research has clearly shown why the human retina must have an "inverted" design, forcing the incoming light to travel through the front of the retina to

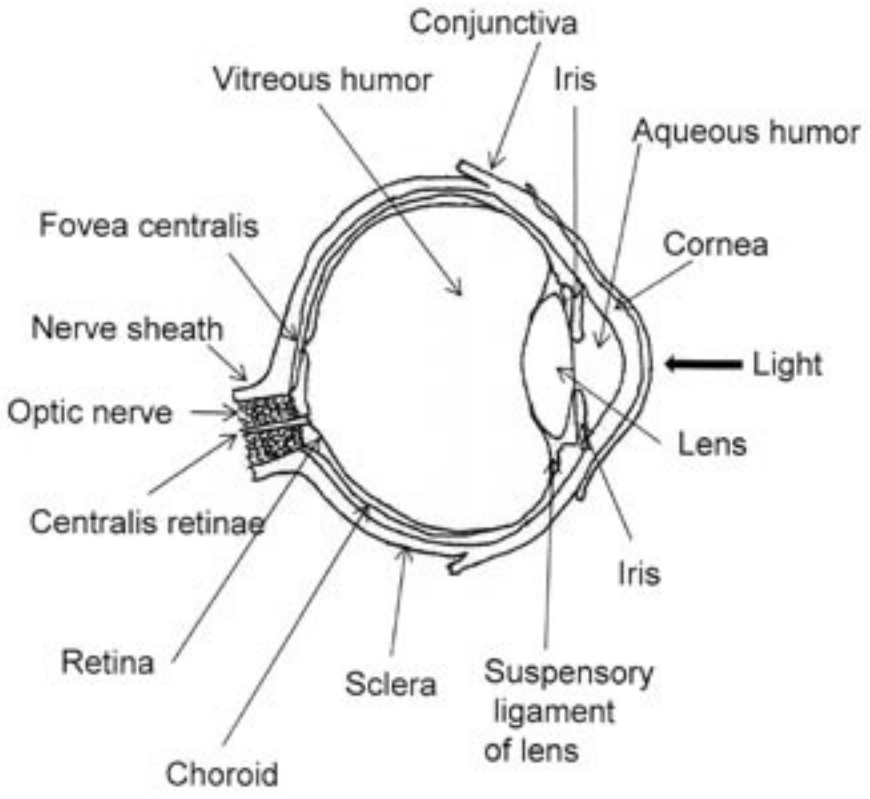

Figure 4. A cross section of the vertebrate eye illustrating some of its major structures. Drawing by B. L. Lindley-Anderson after Land and Nilsson (2005).

reach the photoreceptors. The opposite placement (where the photoreceptors face the front of the eye) is a "verted" design. Verted eyes are wired so that the photoreceptors face toward the light and the nerves are placed behind the photoreceptor layer (Miller, 1994, p. 30).

Most invertebrates and the pineal or dorsal eyes of lower vertebrates use the verted eye design, and most vertebrates (including mammals, birds, amphibians, and fish) use the inverted design. Most verted eye designs are very simple, although a few, such as the cephalopod eye (squids and octopi), are almost as complex as the vertebrate eye (Abbott et al., 1995). Even the better verted eyes are still "overall quite inferior to the vertebrate eye," a conclusion usually determined by measuring performance in response to visual stimuli (Hamilton, 1985, p. 60).

The Cephalopod Visual System

There are several reasons to conclude that evolutionists incorrectly understand the design of the inverted eye. The most advanced invertebrate eye known today is that used by certain cephalopods, but the most advanced eye may actually be the extinct trilobite (Bergman, 2007). The cephalopod visual system is poorly understood, both because it is so complex and because understanding its design is not a funding priority—as is research related to cancer or heart disease. It is known that the major anatomical difference between the human eye and the advanced cephalopod eye, such as the octopus, is the retina. The cephalopod retina is not only verted but also lacks the most sensitive part of the retina, the *fovea centralis* (Land and Nilsson, 2005, p. 64).

In contrast to the claims of Dawkins and others, no evidence exists that even the most advanced verted cephalopod eye is superior to the inverted eye. The sensitivity of the existing human inverted design is so great that a single photon is able to elicit an electrical response (Baylor et al., 1979). Consequently, functional sensitivity of the verted retina could not be significantly improved:

> Neurobiologists have yet to determine how such a negative system of operation might be adaptive, but they marvel over the acute sensitivity possible in rod cells. Apparently rod cells are excellent amplifiers. A single photon (unit of light) can produce a detectable electrical signal in the retina, and the human brain can actually "see" a cluster of five photons—a small point of light, indeed (Ferl and Wallace, 1996, p. 611).

Greater sensitivity than this single photon threshold, if this were possible, might actually result in poorer vision due to sensory overload. In a similar fashion, Williams syndrome patients have superior hearing compared to those with average hearing, allowing them to hear a faint whisper; however, this sensitivity causes them serious sensory overload problems such as in dealing with loud noises like thunder, which is actually physically painful.

Physiologically, the verted cephalopod retina is simpler compared to the inverted vertebrate retina. An example is there are "no equivalents of the amacrine, bipolar or ganglion cells in the cephalopod retina" (Wells, 1978, p. 150). The optic lobes, located behind the eyeball in cephalopods, must assume many of the image processing functions that occur in the inverted retina in ver-

tebrates. As an underwater animal that usually lives on the ocean bottom, its eye is designed to detect motion, not detail, as is true of human eyes. It must also maximize its utilization of light, since the ocean usually has little or no light at lower depths. The cephalopod eye

> undoubtedly forms an image, but the animal's visual perception is certainly quite different from that of man, which is greatly dependent upon interpretation by the brain. The cephalopod optic connections appear to be especially adapted for analyzing vertical and horizontal projections of objects in the visual field (Barnes, 1980, p. 454).

Pechenik (1991) indicated that although cephalopods can perceive shape, light intensity, and texture, they lack many of the advantages of an inverted retina, such as the ability to perceive small details. The cephalopod visual system is designed very differently from the inverted eye in other ways to enable them to function in their water world. Most cephalopods, including octopi, have only one visual pigment and are thus color-blind (Land and Nilsson, 2005).

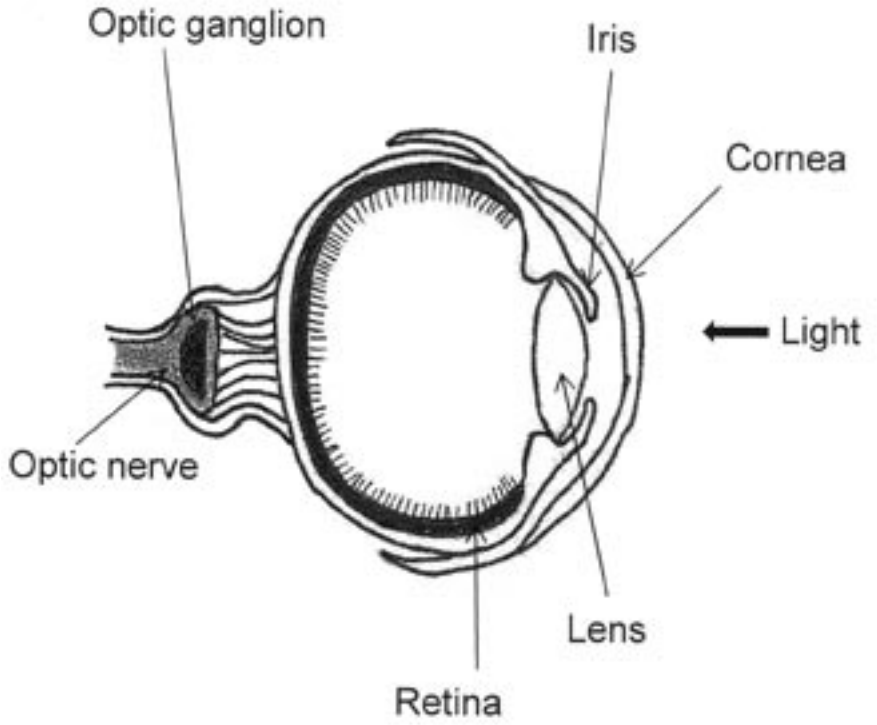

Figure 5. A cross section of the verted cephalopod rhabdomeric eye, illustrating some of its major structures. Also, note the large optic lobe located posterior to the eyeball, a structure that does much of the light processing completed by the vertebrate retina. Drawing by B. L. Lindley-Anderson after Land and Nilsson (2005).

Furthermore, the maximum resolvable spatial frequency in cycles per radian is 4,175 for humans and only 2,632 for octopi (Land and Nilsson, 2005). Their photoreceptor cell population is composed entirely of rods, which contain a "mere" 20 million retina receptor cells, compared to 126 million in humans (Young, 1971). Their rod outer segments contain rhodopsin pigment that has a maximum absorption in the blue-green part of the spectrum (475 nanometers [nm]), which is the predominant color in their environment. Photons change the rhodopsin to metarhodopsin, and no further breakdown or bleaching occurs (Wells, 1978). A second octopus retina pigment, retinochrome, has an absorption maximum of 490 nm, which is more sensitive to dim light (Wells, 1978). Humans have one rod type and three cone types. One cone type has a broad peak light frequency of 440 nm (blue), another type 540 nm (green), and the third type 570 nm (red) (Stoltzmann, 2006).

The squid's visual system must function in an aqueous medium. Water acts as a filter, and, as a result, the light is of a much lower intensity. Consequently, a squid's vision sensitivity is for shorter wavelengths (below around 400 nm) than a human's, which is from 400 to 700 nm (Peet, 1999). In bright light the cephalopod's pupils become thin and slit-shaped and are held in a horizontal position by a statocyst, an organ that uses gravity to determine the horizontal (Young, 1971). Their visual process is "quite similar to that of the batrachians, reptiles and insects. A 'photograph' of the recorded image is not traced on the retina as in man; instead cephalopods respond only to 'light and color variations of a moving object'" (Grzimek, 1972, p. 191).

Significantly, the octopus responds to certain motions of nonfood objects as if they were prey, but will not react to their normal food-objects if motionless (Spigel, 1965). The importance of motion supports the observation that the octopus eye is actually a simple "compound eye with a single lens" because each receptor cell is surrounded by photopigment containing microvilli, which form a rhabdomeric structure like a compound lens (Budelmann, 1994, p. 15). Each facet in a compound eye is either on or off, and object movement produces a change in the on-and-off pat-

tern—similar to the manner in which a series of light bulbs produces the illusion of movement by changing on-and-off patterns.

Our ignorance about the function of major parts of the cephalopod visual system, such as the optic lobe, prevents researchers from completing a more detailed analysis of cephalopod vision. How the basic eye types could have evolved from the putative primitive type is also unknown, in part because no transitional forms exist, nor do plausible hypothetical intermediate forms exist. An essential difference between vertebrate and invertebrate eyes is that the vertebrate eye photoreceptors face

> outwards towards the choroid, whereas in invertebrates they mostly point inwards towards the lens. But for that obstacle we should have been deluged with theories on the original evolution of the vertebrate eye from the invertebrate. As it is, vertebrate visual origins have to be approached with great caution, and…[t]here is nothing indisputable which can be used to explain the origins of the vertebrate eye from an invertebrate organ (Prince, 1956, pp. 334, 348).

All known animals have either verted or inverted retina eyes, and no evidence exists of transitional forms. Invertebrate eyes use either some type of a lens-based eye, such as cephalopods, or a compound eye, as used in trilobites and insects today. All known vertebrates have inverted eyes, and there are no known intermediates between the two.

As Ayoub asked, would "hundreds of thousands of vertebrate species—in a great variety of terrestrial, marine and aerial environments—really see better with a visual system used by a handful of exclusive marine vertebrates? In the absence of any rigorous comparative evidence, all claims that the cephalopod retina is functionally superior to the vertebrate retina remain entirely conjectural" (Ayoub, 1996, p. 20).

Rod and Cone Functions in Vertebrates

The *rods* and *cones* are photoreceptor cells located in the retina used to transduce light into electrical signals. Black-and-white transduction occurs in the rod shaped receptors, and color transduction occurs largely in the cone-shaped receptors (Ryan, 1994). The inverted retina vision system requires light to first pass through the cornea, then through the anterior chamber filled with aqueous fluid, and last, the lens, and the *vitreous humor*. Before reaching the retina, the light passes through the inner retina's cell layers (which contain a dense array of neural processing cells) and on past the rods and cones until it reaches the posterior (distal) end of these cells—wherein lie the so-called outer cell segments. The outer cell segments contain the photoreceptors, light-sensitive structures including the *photopigment*, where the transduction of light into receptor potentials occurs.

The photopigment family of proteins undergoes physical changes when they absorb light energy. The principal photopigment, opsin glycoprotein, is a derivative of *retinal* (a modified vitamin A molecule). Rods contain a single photopigment type called rhodopsin (rhodo meaning *rose* and opsis meaning *vision*). The cones contain one of three different kinds of photopigments called iodopsins, namely *erythrolabe* (most sensitive to red), *chlorolabe* (most sensitive to green) and *cyanolabe* (most sensitive to blue) (Shier et al., 1999, p. 482).

Vision functions by changes in the retina photopigments molecule caused by light. The molecule has a bent shape (*cis-retinal*) in darkness, and when it absorbs light, isomerization occurs, causing the molecule to form the "straight" form (*trans-retinal*). This causes several unstable intermediate chemicals to form, and, after about a minute, the *trans*-retinal form completely separates from opsin, causing the photopigment to appear colorless (for this reason the process is called bleaching). In order for the rods and cones to again function for vision, retinal must be converted from the *trans* back to the *cis* form. This resynthesis process, called regeneration, requires that the retina pigment epithelium (RPE) cells be located next to the rod and cone outer segments.

An average of five minutes is required for rhodopsin regeneration in rods, compared to 1.5 minutes for iodopsin regeneration in cones (Tortora and Grabowski, 1996). Excessive light causes blindness in the affected rods and cones until this regeneration process occurs, as shown by the temporary blindness that occurs after watching a very bright light flash from a camera strobe light (Snell and Lemp, 1989).

When rods and cones are stimulated by light, they release neurotransmitters that induce graded, local potentials in both bipolar and horizontal cells. By this means the rod and cone outer segments transduce light into electrical signals. The signals are then carried by the central nervous system neurons to bipolar cells that, in turn, synapse onto the ganglion cells, then to the lateral geniculate body of the thalamus, and, last, to the occipital region of the brain stem, where the information is organized into a useful image (Stoltzmann, 2006).

The Retinal Pigment Epithelium

One of the many reasons for the inverted design is that behind the photoreceptors lies a multifunctional and indispensable structure, the *retinal pigment epithelium* (RPE) (Martínez-Morales et al., 2004). RPE is a single-cell-thick tissue layer consisting of relatively uniform polygonal-shaped cells whose apical end is covered with dense microvilli and basal membrane infoldings. Posterior to the RPE is the vascular choroid layer, and posterior to it is the connective tissue known as the *sclera*. The RPE touches the extremities of both the rod and the

cone photoreceptors, and the microvilli interdigitate with their sides (Steinberg and Wood, 1994).

The photoreceptors (rods and cones) must face away from the front of the eye in order to be in close contact with the vascular choroid, which supplies the photoreceptors with nutrients and oxygen. This arrangement also allows a steady stream of the vital molecule retinal to flow to the rods and cones, without which vision would be impossible (Kolb, 2003). The verted design, on the other hand, would place the photoreceptors *away* from their source of nutrition, oxygen, and retinal. This design would fail because the rods and cones require an enormous amount of energy for their high metabolism required to function. In addition, due to phototoxicity damage from light, the rods and cones must completely replace themselves approximately every seven days or so. Seemingly simple in appearance, the RPE has "a complex structural and functional polarity that allows them to perform highly specialized roles" (Hewitt and Adler, 1994, p. 58). One of their major functions is to recycle the used retinal from the photoreceptors.

Vision depends on the isomerization of 11-*cis*-retinal to 11-*trans*-retinal in the rods and cones outer segments. Each light photon striking a photoreceptor isomerizes retinal, and billions of photons can strike the retina at any one second. The RPE constantly restores the chromophore to *cis*-retinal from its *trans* configuration caused by photostimulation, permitting visual pigment synthesis and regeneration (Dowling, 1987). The 11-*cis*-retinal must also be regularly replaced to maintain the cycle, a task for which the RPE is critical (Hewitt and Adler, 1994). The RPE manufactures retinal isomerase and other enzymes and stores large quantities of vitamin A to regenerate retinal.

Since RPE cells use enormous amounts of energy and nutrients, they must be in intimate contact with *both* the photoreceptors and the blood supply (in this case the choroid) to carry out this critical function (Marshall, 1996). Research on the eyes of different species has found that, although major differences among them exist, the RPE shows "little variation" (Kuwabara, 1994, p. 58). The small RPE variations are due to differences in the retina structure, indicating its critical role in the vision of all vertebrates. One study found retinol isomerase in all the major vertebrates tested and was lacking in all three cephalopods tested (Bridges, 1989). Bridges concluded that reciprocal flow of retinoids between the retina and the site of isomerase action in the RPE is a feature common to the visual cycle in all vertebrates (Bridges, 1989).

Phagocytic Role of the RPE

A major role of the RPE is to recycle the used rod and cone outer segment membranes, the cone portion closest to the RPE. The photoreceptors and RPE absorb an enormous amount of light on a continuous basis when the eyes are open. This light is converted largely into heat, requiring a very effective cooling system. The choroidal blood supply directly behind the RPE carries away, not only this heat, but also the relatively large amount of waste products produced by the high level of rod and cone metabolism. Which compounds are allowed to pass though this area is determined by basal membrane receptors. Cones usually contain from 1,000 to 1,200 disks, and rods from 700 to 1,000. The enormous amount of outer segment activity requires continual replacement of these disks (Bok, 1994). As the outer segment lengthens from its base, the oldest membrane, which is at the distal end, is shed in segments of one to three disks at a time. Those that are sloughed off are phagocytized by enzymes stored in RPE lysosomes and its components recycled (Tortora and Grabowski, 1996, p. 467).

The RPE phagocytosizes about ten percent of the outer segment disks of normal rod photoreceptors at its apex and renews the same amount daily (Benson, 1996). To replace those segments that are lost, new outer segment membranes are continually being produced at the outer photoreceptor segment base. Photoreceptor outer segments are renewed at "an astonishingly rapid pace" (Tortora and Grabowski, 1996, p. 467).

After RPE breaks down the ingested material, the free radicals and superoxides produced must be neutralized by superoxide dismutase, peroxidase, and other enzymes (Hewitt and Adler, 1994). This process is continuous, effectively maintaining the photoreceptor's high sensitivity (Benson, 1996). Bok and Young (1994) summarized this cycle, noting that the

> retinal pigment epithelium carries out several functions that are crucial for the normal operation of the visual system. One of these important roles, appreciated for about a decade, is the phagocytosis of rod outer segment debris. This scavenging activity goes on daily at an impressive rate in the normal retina. It can be accelerated to extraordinary levels when outer segments are damaged. Disruption of this phagocytic function may underlie a variety of clinical disorders, some of which result in blindness (p. 148).

RPE microvilli interdigitate and surround the photoreceptor outer segments so as to effectively carry out their phagocytic and recycling role (Bok and Young, 1994).

Nutrient Role of the RPE

The RPE selectively transports nutrients from choroidal circulation to both the photoreceptors and retinal cells. The RPE also helps maintain water and ion flow between the neural retina and the choroid, protects against free radical damage, and regulates retinoid metabolism (Martínez-Morales et al., 2004). The RPE functions similarly to a placenta to ensure that the outer retina

is protected from injurious compounds and yet allows the necessary nutrients to pass into the rod and cone area. RPE cell tight junctions are also part of the outer blood-retinal barrier, preventing diffusion of even small molecules into the vitreous humor and ensuring that the metabolites required by the outer retina can move to where they are needed when they are needed (Hewitt and Adler, 1994).

To ensure that enough of the needed nutrients pass the RPE barrier, the basal membrane is highly infolded to produce more surface area. This role is critical because the rods and cones require a greater blood supply than any other bodily tissue (Hewitt and Adler, 1994). This is important because of the high level of metabolism due to the complex chemistry required for vision, which necessitates a higher level of oxygen and nutrients. The RPE also synthesizes and secretes various extracellular matrix molecules that must be produced near the location where they are to be used.

If the photoreceptors were anterior to the neurons, as in the verted design, the blood supply would have to be either directly in the light path of the receptors or on their side, which would reduce enormously the number of photoreceptors used for sight. If the pigment epithelium tissue were placed in front of the retina, sight would be seriously compromised. The verted design would make vision impossible because the photoreceptors must be embedded in the retinal pigment epithelium to obtain the nutrients required to function.

Müller Cells Function as Optical Fibers

Placing the retina neural components in front of the photoreceptors does not produce an optical handicap for yet other reasons (Land and Nilsson, 2005). One is that the neural elements are separated by less than a wavelength of light. Consequently, very little or no scattering or diffraction occurs, and the light travels through this area as if it were at near-perfect transparency.

The Müller cells (which are radial glial cells) in front of the retina have both shape and optical properties that contribute to optimizing light transferral and reducing light scatter (Franze et al., 2007). Müller cells "have an extended funnel shape, a higher refractive index than their surrounding tissue and are oriented along the direction of light propagation" (Franze et al., 2007, p. 8287). The effect provides a "low-scattering passage for light from the retinal surface to the photoreceptor cells," functioning as fiber optic plates that are effective for low-distortion transfer of light images. Franze et. al. (2007) concluded that cells thought to interfere with light transmission are actually highly effective in reducing light scatter and distortion, helping to produce a sharp image.

The Macula

The importance of the RPE is indicated by the fact that one of the most common causes of blindness in the developed world, macular degeneration, is the result of RPE deterioration (Zhang, et al., 1995). In this disease the eye's macula loses its ability to function, causing major central vision loss. Without the nourishment and waste removal role of the pigment epithelium, retina cells will also die. Among the other diseases affecting the macula is *central serous retinopathy*, an ion pump malfunction and/or a result of choroidal vascular hyperpermeability.

Detached Retina and the Role of Pigment Epithelial Cells

The retina is connected to the RPE largely by the interphotoreceptor matrix. When the retina pulls away from the RPE at the interphotoreceptor matrix area, a *detached retina* results (Zamir, 1997). The RPE can then no longer effectively function to regenerate the rods and cones, causing vision to become distorted, and eventually the death of significant levels of retina tissue. Progressive detachment can often be halted by laser therapy, a procedure that is only minimally invasive because laser light is able to pass through the cornea and the lens without damaging them. Laser therapy stimulates the migration of the RPE cells, inducing the pigmentation line to form.

Functions of the Pigment

The many diverse functions of the retinal pigment epithelium cells that are "essential for the normal functioning of the outer retina" include producing a black pigment called *melanin* (Hewitt and Adler, 1994, p. 67). The melanin functions to absorb most of the light not captured by the retina, preventing the reflection and scattering of light within the eyeball. This inhibits light from being reflected off the back of the eye onto the retina, preventing degradation of the visual image and ensuring that the image cast on the retina by the cornea and lens remains sharp and clear.

Yet another function of the pigment is to form an opaque screen behind the optical path of the photoreceptors. This light absorptive property of the pigment is critical to maintaining high visual acuity. For this reason, normal retinal function requires that the RPE and photoreceptors be in close proximity. Lack of the pigment, as in albinism, can cause a variety of problems such as *fovea hypoplasia*, an abnormal routing of the optic nerve (Oetting and King, 1999; Lyle et al., 1997; Jeffery and Williams, 1994). As a result of this and other factors, albinism victims lack detailed central vision (Snell and Lemp, 1989; Williamson, 2005).

The Retina Pigment Epithelium's Role in Development

RPE is also critical for normal vertebrate eye development. A series of reciprocal cellular interactions during vertebrate eye development determine the fate of the eye components, and the

> presence of the RPE is required for the normal development of the eye *in vivo*. Its presence early in development is necessary for the correct morphogenesis of the neural retina (Raymond and Jackson, 1995, p. 1286).

The RPE actually plays a succession of roles during embryonic development, including trophic influence, transport functions, retinomotor response, and phagocytic and inductive interaction (Coulombre, 1994).

Does the Backward Design Block Light?

Nerve cell fibers and the small branches of the central retina artery and vein produce minimal hindrance to light reaching the photoreceptors because most cells are 60 to 70% water and, consequently, are largely transparent. When viewed under the microscope, most cells are largely transparent. It is for this reason stains, such as Eosin-Y and Hematoxylin 2, are required to better visualize the various cell parts. Myelin, an opaque whitish lipid that coats nerve axons, would block much light, but, in contrast to most peripheral nerves, nerve fibers in front of the retina are not mylinated. Furthermore, the larger blood vessels and nerve fibers skirt around the *area centralis*, where visual acuity is most important (Gregory, 1976). The vertebrate eye is highly effective in spite of the retina reversal because it is a precise visual instrument designed to function with the rods and cones facing away from the light.

> The tissues intervening between the transparent humors of the eye cavity and the optically sensitive layer are microscopically thin. The absorption and scatter of light is ordinarily minor, and functional impairment seldom serious.…Red blood cells are poor transmitters of light, but when moving single file through capillaries can cause only a negligible shading of the light sensors (Williams, 1992, p. 73).

These facts have forced Dawkins to note that many

> photocells point backwards, away from the light. This is not as silly as it sounds. Since they are very tiny and transparent, it doesn't much matter which way they point: most photons will go straight through and then run the gauntlet of pigment-laden baffles waiting to catch them (Dawkins, 1996, p. 170).

Moving shadows produced by the venules and arterioles are also highly functional because they produce momentary darkness to aid in the rod and cone regeneration. Constant bright light would excessively bleach the photopigment, and the lower light achieved by the existing design allows their regeneration.

Other Possible Designs

A major concern when critiquing the existing vertebrate retina design involves speculation on the quality of vision that would result from another design. No evidence exists that a verted human retina design, as in octopi, would result in better vision, and it would likely be worse. Comparisons of different eyes are difficult to make because, although the quality of the image projected on the retina can be evaluated by a study of the lens system's optical traits, direct knowledge about the actual image produced in the brain is lacking.

If the retina were reversed, the retinal pigment epithelium or its analog and its cellular support system would have to be placed either in front of the photoreceptors or on their side. Both of these approaches are clearly inferior to the existing vertebrate system, which produces superior sight for terrestrial animals. If located in front of the retina, depending on the transparency of these cells, this design could prevent most light from reaching the photoreceptors.

If the RPE functioning cells were located on each side of the rods and cones, as in the cephalopods, primarily only the sensory cell face would be able to respond to light. Octopi use support cells located next to the light-sensitive cells called rhabdomeric receptors that use photopigments containing microvilli (Land and Nilsson, 2005). The support cells also require increasing the space between the photoreceptors, further decreasing light able to strike the photoreceptors, and consequently lowering vision resolution. Prince (1956) even claims the cephalopod's side design "is protective and shields the receptors from excess light" (p. 343). Opaque wastes would accumulate in the light path, and the presence of required nutrients would further diminish the amount of light reaching the photoreceptors. Recycling the outer segments to allow rapid regeneration of the photoreceptors would also be a major problem if the photoreceptors faced the vision light path line. Verted designs produce the following concerns:

> Should the disk end of the rods and cones be reversed in direction so as to face the light…we would probably have a visual disaster. What would perform the essential function of absorbing the some 10,000 million disks produced each day in each of our eyes? They would probably accumulate in the vitreous humor region and soon interfere with light en route to the retina. If the pigment epithelium layer were placed on the inside of the retina so as to absorb the disks, it would also interfere with light trying to reach the rods and cones. Furthermore, the pigment epithelium, which is closely associated with the disk ends of the rods and cones, also provides them with nutrients for making new disks. The epithelium gets its nutrients from the rich blood supply in the choroid layer next to it. In order for the pigment epithelium to function

> properly, it needs this blood supply. To put both the pigment epithelium and its choroid blood supply on the inside of the eye, between the light source and the light-sensitive rods and cones, would severely disrupt the visual process (Roth, 1998, p. 109).

Although higher visual acuity may improve night vision, in humans it would result in difficultly seeing during daylight hours, which would not be functional for persons that must work in normal human-light environments (Sjostrand, 1989). Actually, a case can be made that *more* light blockage of the retina would be functional. Many persons must wear sunglasses because normal outdoor light is often too bright. In a review of the literature, Young (1992) found that excess solar radiation can be a serious health problem, and may

> explain the distinctive global pattern of age-related cataract among human populations—the risk of cataract depends on where one lives on the surface of the earth.…Current evidence provides the basis for the design of protective lenses that minimize the hazards of sunlight exposure without significantly interfering with vision. The prescription has two components—one to protect the lens, the other to protect the retina.…Use of sunglasses…should begin early in childhood and be continued throughout the life span whenever exposure to bright sunlight is desirable or necessary. Radiation damage to delicate ocular structures can occur at any age and tends to be cumulative (pp. 335-357).

Albinos lack iris pigment, requiring them to wear sunglasses in daylight because even moderately bright light may severely adversely affect their vision (Tortora and Grabowski, 1996, p. 461). Even blue-eyed persons are at a disadvantage because the blue pigment allows in more light than the darker iris pigments. Consequently, they suffer from more vision problems (Young, 1992). Being able to effectively read by very dim light may be an improvement in some situations, but since most human activities occur during daylight hours and darkness is functional to induce sleep due to pineal gland activity, the existing system appears to be the most effective.

Furthermore, although the light yellow tint of the eye lens filters out some ultraviolet light, the inverted eye design serves to filter out much of the remaining ultraviolet light. The incoming light must pass through the overlying neural components and blood vessels, and the penetrating power of ultraviolet light is markedly inferior to white light (Lumsden, 1994). The verted eye is used in animals such as the octopus, which live underwater, where most of the ultraviolet light is filtered out. Consequently, they have less need for this protection. Given the role of the pigmented epithelium, it is clear that the existing design is ideal.

Conclusions

A review of research on the vertebrate retina consistently concludes that each design is perfectly suited for the environment the organism normally lives in—even the system used by the most advanced cephalopods (Bergman, 2000; Bergman and Calkins, 2005; Wieland, 1996; Marshall, 1996). The design maximized for life in our environment would no doubt function poorly in another environment, such as that experienced by undersea bottom dwellers. The RPE metabolic machinery is "essential for the normal functioning of the outer retina [and] because of the nature of these interactions, it is essential that the RPE and photoreceptors be in close proximity" for normal retina function (Hewitt and Adler, 1994, p. 67). This review supports Hamilton's conclusion that

> instead of being a great disadvantage, or a "curse" or being incorrectly constructed, the inverted retina is a tremendous advance in function and design compared with the simple and less complicated verted arrangement. One problem amongst many, for evolutionists, is to explain how this abrupt major retinal transformation from the verted type in invertebrates to the inverted vertebrate model came about as nothing in paleontology offers any support (Hamilton, 1985, p. 63).

Rather than being fired, our camera designer would no doubt be promoted for utilizing a less obvious but far more functional design. It is clear that "eyesight is a compelling testimony to creative design" (DeYoung, 2002, p. 190). This short review covers only a few of the many reasons for the superiority of the existing mammalian retina design. Gratitude rather than impertinence seems the more appropriate response to its ingenious design.

Acknowledgments

I wish to thank Dr. Tara Richmond, O.D.; Clifford Lillo, MS; John Up-Church; David Stoltzmann, optical engineer; Don DeYoung, Ph.D., physicist; Jody Allen, RN; B. L. Lindley-Anderson for producing the illustrations; and George F. Howe for editorial assistance.

Joseph Calkins, MD, currently an ophthalmologist in private practice, was formally Assistant Professor of Ophthalmology at Johns Hopkins University in Baltimore, Maryland. His MD is from the University of Michigan Medical School in Ann Arbor, MI.

References

CRSQ: Creation Research Society Quarterly.

Abbott, J., R. Williamson, and L. Maddock. 1995. *Cephalopod Neurobiology*. Oxford University Press, Oxford, NY.

Ayoub, G. 1996. On the design of the vertebrate retina. *Origins and Design* 17:19–22.

Barash, D.P., and I.A. Barash. 2000. *The Mammal in the Mirror: Understanding Our Place in the Natural World*. W.H. Freeman, New York, NY.

Barnes, R.D. 1980. *Invertebrate Zoology*. Saunders, Philadelphia, PA.

Baylor, D. A., T.D. Lamb, and K.W. Yau. 1979. Response of retinal rods to single photons. *Journal of Physiology* 288:613–634.

Benson, E. 1996. Retinitis pigmentosa: unfolding its mystery. *Proceedings of the National Academy of Science USA* 93:4526–4528.

Bergman, J. 2000. Is the inverted human eye a poor design? *Journal of the American Scientific Affiliation* 52:18–30.

Bergman, J. 2007. Lack of fossil evidence for arthropod evolution is a major difficulty for neo-Darwinism. *CRSQ* 43:222–230.

Bergman, J., and J. Calkins. 2005. Is the backwards human retina evidence of poor design? *Impact* 388:1–4.

Bok, D. 1994. Retinal photoreceptor disc shedding and pigment epithelium phagocytosis. In Zinn, K.M., and M.F. Marmor (editors), *The Retinal Pigment Epithelium*, pp. 81–94. Harvard University Press, Cambridge, MA.

Bok, D., and R. Young. 1994. Phagocytic properties of the retinal pigment. In Zinn, K.M., and M.F. Marmor (editors), *The Retinal Pigment Epithelium*, pp. 148–174. Harvard University Press, Cambridge, MA.

Bridges, C.D.B. 1989. Distribution of retinol isomerase in vertebrate eyes and its emergence during retinal development. *Vision Research* 29:1711–1717.

Budelmann, B.V. 1994. Cephalopod sense organs, nerves and the brain: adaptations for high performance and life style. In Portner, Hans, et al. (editors), *Physiology of Cephalopod Mollusks*, pp. 13–33. Gordon and Breach, Basel, Switzerland.

Coulombre, A. 1994. Roles of the retinal pigment epithelium in the development of ocular tissue. In Zinn, K.M., and M.F. Marmor (editors), *The Retinal Pigment Epithelium*, pp. 53–57. Harvard University Press, Cambridge, MA.

Dalton, R. 2004. True colours. *Nature* 428:596–597.

Dawkins, R. 1986.*The Blind Watchmaker*. W. W. Norton, New York, NY.

Dawkins, R.. 1996. *Climbing Mount Improbable*. W.W. Norton, New York, NY.

Dennett, D. 2005. Show me the science. *New York Times*, August 28.

DeYoung, D.B. 2002. Vision. *CRSQ* 38:190–192.

Diamond J. 1985. Voyage of the overloaded ark. *Discover* 6:82–92.

Dowling, J.E. 1987. *The Retina: An Approachable Part of the Brain*. The Belknap Press of Harvard University Press, Cambridge, MA.

Edinger, S. 1997. Is there a scientific basis for creationism? *The Congressional Quarterly Researcher* 7:761.

Ferl, R., and R.A. Wallace. 1996. *Biology, the Realm of Life*. Harper Collins, New York, NY.

Franze, K., J. Grosche, S.N. Skatchkov, S. Schinkinger, C. Foja, D. Schild, O. Uckermann, K. Travis, A. Reichenbach, and J. Guck. 2007. Müller cells are living optical fibers in the vertebrate retina. *Proceedings of the National Academy of Science* 104:8287–8292.

Frymire, P. 2000. *Impeaching Mere Creationism*. Writers Club Press, San Jose, CA.

Gregory, R. 1976. *Eye and Brain*. World Universal Library, New York, NY.

Grzimek, B. 1972. *Grzimek's Animal Life Encyclopedia*. Van Nostrand Reinhold, New York, NY.

Hamilton, H.S. 1985. The retina of the eye—an evolutionary road block. *CRSQ* 22:59–64.

Hewitt, A.T., and R. Adler. 1994. The retinal pigment epithelium and interphotoreceptor matrix: structure and specialized functions. In Ryan, S.J. (editor), *The Retina*, Second Edition, pp. 58–71. Mosby, St. Louis, MO.

Jeffery, G., and A. Williams. 1994. Is abnormal retinal development in albinism only a mammalian problem? Normality of a hypopigmented avian retina. *Experimental Brain Research* 100:47–57.

Kolb, H. 2003. How the retina works. *American Scientist* 91:28–35.

Kuwabara, T. 1994. Species differences in the retinal pigment epithelium. In Zinn, K.M., and M.F. Marmor (editors), *The Retinal Pigment Epithelium*, pp. 58–82. Harvard University Press, Cambridge, MA.

Land, M.F., and D.E. Nilsson. 2005. *Animal Eyes*. Oxford University Press, Oxford, NY.

Lumsden, R. 1994. Not so blind a watchmaker. *CRSQ* 31:13–21.

Lyle, W.M., J.O.S. Sangster, and T.D. Williams.1997. Albinism: an update and review of the literature. *Journal of the American Optometric Association* 68:623–645.

Marshall, G. 1996. An eye for creation: an interview with eye-disease researcher Dr. George Marshall, University of Glasgow, Scotland. *Creation* 18:19–21.

Martínez-Morales, J.R., I. Rodrigo, and P. Bovolenta. 2004. Eye development: a view from the retina pigmented epithelium. *BioEssays* 26:766–777.

Meglitsch, P. 1972. *Invertebrate Zoology*. Oxford University Press, New York, NY.

Miller, K.R. 1994. Life's grand design. *Technology Review* 97:25–32.

Miller, K.R.. 1999. *Finding Darwin's God: A Scientist's Search for Common Ground between God and Evolution*. Cliff Street Books, New York, NY.

Oetting, W.S. and R.A. King. 1999. Molecular basis of albinism: mutations and polymorphisms of pigmentation genes associated with albinism. *Human Mutatagens* 13:99–115.

Pechenik, J. 1991. *Biology of the Invertebrates*. William C. Brown, Dubuque, IA.

Peet, J.H.J. 1999. Creation in the news: Dawkins' blind spot. *Origins* 26:2–4.

Prince, J. 1956. *Comparative Anatomy of the Eye*. Charles Thomas, Springfield, IL.

Raymond, S.M., and I.J. Jackson. 1995. The retinal pigment epithelium is required for development and maintenance of the mouse neural retina. *Current Biology* 5:1286–1295.

Roth, A. 1998. *Origins*. Review and Herald, Hagerstown, MD.

Ryan, S.J. (Editor). 1994. *The Retina*, 2nd Edition. Mosby, St. Louis, MO.

Sarfati, J. 1998. A review of *Climbing Mount Improbable* by Richard Dawkins. *Technical Journal*. 12:29–34.

Shermer, M. 2005. *Science Friction: Where the Known Meets the Unknown*. Holt/Times Books, New York, NY.

Shier, D., J. Butler, and R. Lewis. 1999. *Hole's Human Anatomy and Physiology*. William C. Brown, Dubuque, IA.

Sjostrand, F. 1989. An elementary information processing component in the circuitry of the retina generating the on-response. *Journal of Ultrastructure and Molecular Structure Research* 102:24–38.

Snell, R., and M. Lemp. 1989. *Clinical Anatomy of the Eye*. Blackwell Scientific Publications, Boston, MA.

Spalton, D.J., R.A. Hitchings, and P.A. Hunter (editors). 2005. *Atlas of Clinical Ophthalmology*, 2nd Edition. Mosby, St. Louis, MO.

Spigel, I.M. (Editor). 1965. *Readings in the Study of Visually Perceived Movement*. Harper and Row, New York, NY.

Steinberg, R.H., and I. Wood. 1994. The relationship of the retinal pigment epithelium to the photoreceptor outer segment in the human retina. In Zinn, K.M., and M.F. Marmor (editors), *The Retinal Pigment Epithelium*, pp. 32–44. Harvard University Press, Cambridge, MA.

Stoltzmann, D.E. 2006. The specified complexity of retinal imagery. *CRSQ* 43:4–12.

Thwaites, W. 1982. Design, can we see the hand of evolution in the things it has wrought? *Proceedings of the 63rd Annual Meeting of the Pacific Division; American Association of the Advancement of Science* 1:206–213.

Tortora, G., and S. Grabowski. 1996. *Principles of Anatomy and Physiology*. Harper and Collins, New York, NY.

Wells, M.J. 1978. *Octopus; Physiology and Behavior of an Advanced Invertebrate*. Chapman and Hall, London, UK.

Wieland, C. 1996. Seeing back to front: are evolutionists right when they say our eyes are wired the wrong way? *Creation* 18:38–40.

Williams, G.C. 1992. *Natural Selection: Domains, Levels, and Challenges*. Oxford University Press, New York, NY.

Williams, G.C. 1997. *The Pony Fish Glow and other Clues to Plan and Purpose in Nature*. BasicBooks, New York, NY.

Williamson, T. 2005. Vitreous and vitreo-Retinal disorders. In Spalton, D.J., R.A. Hitchings, and P.A. Hunter (editors), *Atlas of Clinical Ophthalmology*, 2nd Edition, pp. 263–297. Mosby, St. Louis, MO.

Young, J.Z. 1971. *The Anatomy of the Nervous System: Octopus Vulgaris*. Oxford University Press, New York, NY.

Young, R. 1992. Sunlight and age-related eye disease. *Journal of the National Medical Association* 84:353–358.

Zamir, E. 1997. Central serous retinopathy associated with adrenocorticotrophic hormone therapy. *Graefes Archives for Clinical Ophthalmology* 235:339–344.

Zhang, K., E. Nguyen, A. Crandall, and L. Donoso. 1995. Genetic and molecular studies of macular dystrophies: recent developments. *Survey of Ophthalmology* 40:51–61.

Zinn, K.M., and M.F. Marmor (editors). 1994. *The Retinal Pigment Epithelium*. Harvard University Press, Cambridge, MA.