7

SIGHT

From the Land of the Blind

Sight is quite a rarity. Eyes are absent, at least in a conventional sense, from the plant kingdom, as well as from the fungi, algae and bacteria. Even in the animal kingdom eyes are not at all common property. There are said to be thirty-eight fundamentally different models of body plan – phyla – in the animal kingdom, yet only six of them ever invented true eyes. The rest have endured for hundreds of millions of years without the benefit of seeing anything at all. Natural selection did not scourge them for lacking sight.

Set against this spartan background, the evolutionary benefits of eyes loom large. All phyla are not equal, and some are far more equal than others. The Chordata, for example, the phylum that includes ourselves and all other vertebrates, comprises more than 40,000 species; the Mollusca, including slugs, snails and octopuses has 100,000; and the Arthropoda, including crustaceans, spiders and insects, numbers more than a million, making up 80 per cent of all described species. In contrast, most of the lesser known phyla, including such oddities as the glass sponges, rotifers, priapulid worms and comb jellies, mostly known only to classically trained zoologists, have relatively few species, tens or hundreds; the Placozoa, just one. If we add them all up, we find that 95 per cent of all animal species have eyes: the handful of phyla that *did* invent eyes utterly dominates animal life today.

Of course, that might be no more than chance. Perhaps there are other subtle advantages to the body plans of these particular phyla that we have

missed, quite unrelated to eyes, but that seems unlikely. The evolution of proper eyes, capable of spatial vision rather than simply detecting the presence or absence of light, gives every appearance of having transformed evolution. The first true eyes appeared somewhat abruptly in the fossil record around 540 million years ago, close to the beginning of that 'big bang' of evolution, the so-called Cambrian explosion, when animals burst into the fossil record with breathtaking diversity. In rocks that had been virtually silent for aeons, almost all the modern phyla of animals sprang into existence practically without warning.

The close correspondence in time between the explosion of animal life in the fossil record and the invention of eyes was almost certainly no coincidence, for spatial vision must have placed predators and prey on an entirely different footing; this alone could, and perhaps did, account for the predilection for heavy armour among Cambrian animals, and the much greater likelihood of fossilisation. The biologist Andrew Parker, at the Natural History Museum in London, has made a plausible case that the evolution of eyes drove the Cambrian explosion, in an entertaining, if at times infuriatingly partisan, book. Whether eyes really could have evolved so abruptly (or whether the fossil record is misleading in this regard) is a question we'll consider later. For now let's just note that sight gives far more information about the world than smell, hearing, or touch possibly can, for the earth is drenched in light, and we can hardly avoid being seen. Many of the most marvellous adaptations of life are a response to being seen, whether strutting for sex in the case of a peacock or a flower, parading the great armoured plates of a stegosaurus, or careful concealment in the world of a stick insect. Our own societies are so image-conscious that I scarcely need to labour the point.

Beyond utility, the evolution of sight is culturally iconic, because eyes appear so perfect. From Darwin onwards, eyes have been perceived as an apotheosis, a challenge to the very notion of natural selection. Could something so complex, so perfect, really evolve by unguided means? What possible use, say sceptics, is half an eye? Natural selection calls for a million gradations, each of which must be better than the last, or the half-built structure will be ruthlessly purged from the world. But the eye, say these sceptics, is perfect in the same way as a clock – it is irreducible. Remove a few of the bits and it

won't work any more. A clock without hands is worth little, and an eye without a lens or a retina is worthless, or so we're told. And if half an eye is no use then the eye cannot have evolved by natural selection or any other means known to modern biology, and so must be evidence of celestial design instead.

The many vitriolic arguments over perfection in biology rarely do more than entrench already hardened positions. Defenders of Darwin counter that the eye is actually far from perfect, as anyone who wears glasses or contact lenses, or who is losing their sight, knows only too well. This is certainly true, but there is a danger in this kind of theoretical argument, which is to gloss over the many subtleties that undoubtedly exist. Take the human eye, for example. A common argument has it that the design flaws run very deep and are in fact good evidence of the way in which evolution has cobbled together inept unplanned structures, crippled by its own lack of foresight. A human engineer, we're told, would do a much better job; indeed an octopus does. This glib assertion overlooks the mischievous rule known as the second law of Leslie Orgel: Evolution is cleverer than you are.

Let's consider this case briefly. The octopus has an eye much like our own, a 'camera' eye, with a single lens at the front and a light-sensitive sheet, the retina, at the back (equivalent to the film in a camera). Because the last ancestor we shared with the octopus was probably some sort of worm, lacking a proper eye, the octopus eye and our own eye must have evolved independently and converged upon essentially the same solution. This inference is supported by a detailed comparison of the two types of eye. Each develops from different tissues in the embryo and ends up with distinct microscopic organisation. The octopus eye seems to be far more sensibly arranged. The light-sensitive cells of the retina point out towards the light, while the neuronal wires pass back directly to the brain. In comparison, our own retina is often said to be plugged in backwards, an apparently idiotic arrangement. Rather than jutting out, the light-sensitive cells sit at the very back, covered by neuronal wires that pass forwards on a roundabout route to the brain. Light must pass through this forest of wires before it can reach the light-sensitive cells; and worse still, the wires form a bundle that plunges back through the retina as the optic nerve, leaving a blind spot at that point.[1]

But we should not be too quick to dismiss our own arrangement. As so often in biology, the situation is more complex. The wires are colourless, and so don't hinder the passage of light much; and insofar as they do, they may even act as a 'waveguide', directing light vertically on to the light-sensitive cells, making the best use of available photons. And probably more importantly, we have the advantage that our own light-sensitive cells are embedded directly in their support cells (the retinal pigment epithelium) with an excellent blood supply immediately underneath. Such an arrangement supports the continuous turnover of photosensitive pigments. The human retina consumes even more oxygen than the brain, per gram, making it the most energetic organ in the body, so this arrangement is extremely valuable. In all probability the octopus eye could not sustain such a high metabolic rate. But perhaps it doesn't need to. Living underwater, with lower light intensity, the octopus may not need to re-cycle its photopigments so quickly.

My point is that there are advantages and disadvantages to every arrangement in biology, and the outcome is a balance of selective forces that we don't always appreciate. This is the trouble with 'just-so' stories: all too often we see only half the picture. Arguments too conceptual in nature are always vulnerable to counterblasts. Like any scientist, I prefer to follow the train of data. And here the rise of molecular genetics in the last decades furnishes us with a wealth of detail, giving very particular answers to very particular questions. When these answers are all threaded together, a compelling view emerges of how the eye evolved, and from where – a surprisingly remote and green ancestor. In this chapter, we'll follow this thread to see exactly what use is half an eye, how lenses evolved, and where the light-sensitive cells of the retina came from. And in piecing together this story, we'll see that the invention of eyes really did alter the pace and flow of evolution.

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It's easy to treat the question 'what use is half an eye?' with derision: which half, the left or the right? I can sympathise with Richard Dawkins's truculent riposte: half an eye is 1 per cent better than 49 per cent of an eye; but for those of us who struggle to conjure up a clear image of half an eye, 49 per cent of

an eye is even more stupefying. Actually, though, a literal 'half-an-eye' is a very good way of approaching the problem. The eye does divide neatly into two halves, the front and the back. Anyone who's been to a conference of ophthalmologists will appreciate that they fall into two great tribes: those who work at the front of the eye (cataract and refractive surgeons, dealing with the lens and the cornea), and those who work at the back (the retina), treating such major causes of blindness as macular degeneration. The two tribes interact reluctantly, and at times barely seem to speak the same language. Yet their distinction is a valid one. Stripped of all its optical accoutrements, the eye is reduced to a naked retina: a light-sensitive sheet with nothing on top. And exactly such a naked retina is a fulcrum of evolution.

The idea of a naked retina may sound bizarre, but it fits quite happily into an equally bizarre environment, the deep ocean black-smoker vents that we visited in Chapter 1. Such vents are home to an astonishing array of life, all of which depend, in one way or another, on the bacteria that live directly on the hydrogen sulphide gas emanating from the vents. Perhaps the strangest, and certainly the most celebrated, are the giant tubeworms, which reach eight feet long. Though distantly related to normal earthworms, the tubeworms are literally gutless wonders, possessing neither mouth nor intestine, instead depending for their sustenance on sulphur bacteria nurtured within their own tissues. Other giants found at the vents include huge clams and mussels.

All these giants are found only in the Pacific Ocean, but the Atlantic vents harbour their own wonders, notably the swarming shrimp *Rimicaris exoculata*, which throng in multitudes beneath the smoking chimneys. The name literally means 'eyeless rift shrimp', an unfortunate misnomer that must have returned to haunt its discoverers. Certainly, as might be expected from their name and habitat in the black depths of the ocean, the shrimp don't have conventional eyes. They completely lack the eye-stalks of their surface-dwelling cousins, but they do possess two large flaps on their backs. And although rather nondescript in appearance, these strips reflect light like cats' eyes in the glare of the deep-sea submersibles.

The flaps were originally noticed by Cindy Van Dover, her discovery marking the beginning of one of the more remarkable scientific careers of our times. She is the kind of scientific explorer that Jules Verne used to write

about, as endangered a species today as any that she studies. Van Dover now heads the Marine Laboratory at Duke University, and has visited virtually all of the known vents, and more than a few unexplored ones, as the first female pilot of the naval deep-sea submersible *Alvin*. She later discovered that exactly the same giant clams and tubeworms inhabit cold sites on the seafloor, where methane seeps up from the bowels of the earth; clearly the chemical conditions, rather than the heat, are the driving force behind life's exuberance at the bottom of the sea. Back in the late 1980s, though, all this lay ahead, and she must have felt pretty tremulous in sending off samples of the blind shrimp's tissue flaps to a specialist in invertebrate eyes, with the rather lame question, might this be an eye? If you were to mangle a retina, came back the laconic answer, it might look a bit like this. While lacking the usual paraphernalia of eyes – lens, iris, and so on – the blind shrimp possessed what seemed to be naked retinas, running partway down their back, despite living in the black depths of the ocean (see Fig. 7.1).

As more studies were carried out, the findings were better than Van Dover had dared hope. The naked retinas turned out to possess a pigment with properties very similar to that responsible for detecting light in our own retina, called *rhodopsin*. What's more, this pigment was packed into light-sensitive cells characteristic of normal shrimp eyes, even though the overall appearance of the retina was very different. So perhaps the blind shrimp really could see light at the bottom of the ocean. Might the vents themselves produce a faint glow, Van Dover wondered? After all, hot filaments glow and the vents were certainly both hot and full of dissolved metals.

Nobody had ever switched off the lights on *Alvin* before. In pitch blackness, such a manoeuvre was worse than pointless, as there was a good chance that the craft would drift into a vent and fry those aboard, or at least its own instruments. Van Dover had not yet descended to the vents herself, but succeeded in persuading geologist John Delaney, who was about to venture down, to switch off the lights and point a digital camera at a vent. While the blackness was unbroken to the naked eye, Delaney captured on camera a sharply defined halo around the vent, 'hovering in the darkness like the grin of a Cheshire cat'. Even so, these first pictures gave no inkling of what kind of light was emitted – what colour, or how bright. Would it really be possible

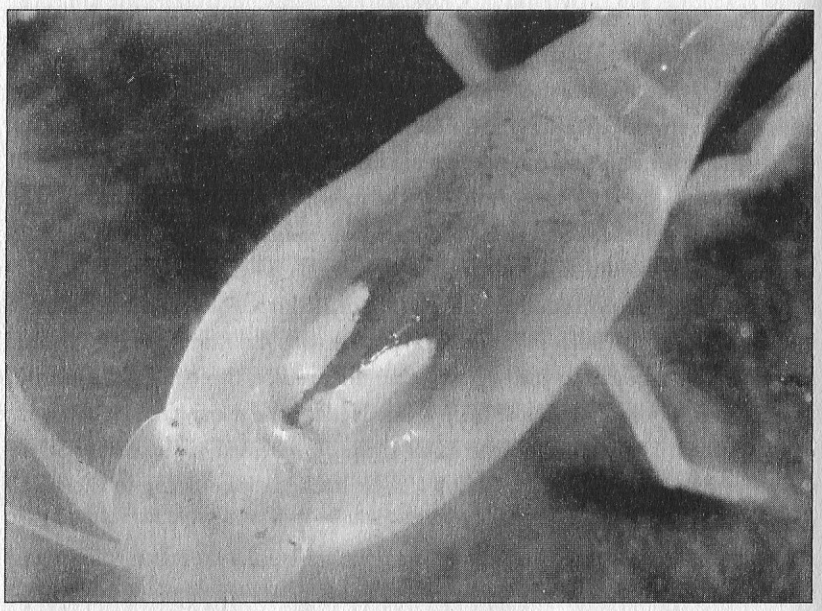

Figure 7.1 Eyeless shrimp *Rimicaris exoculata*, showing the two pale naked retinas running down the back.

for the shrimp to 'see' the vents' glow, when we ourselves could see nothing at all?

Like hot filaments, black smokers were predicted to glow red, with wavelengths reaching into the thermal (near infrared) range. In theory, shorter wavelengths, in the yellow, green and blue parts of the spectrum, should not be emitted at all. This prediction was confirmed by some early, albeit crude, measurements, in which coloured filters were placed over the lens. Presumably, if the shrimp could see the vents' glow, then their eyes would need to be 'tuned' to see red or near infrared rays. Yet the first studies of the shrimp's eyes suggested otherwise. On the contrary, the rhodopsin pigment was stimulated most by green light at a wavelength of around 500 nanometres. While this might have been passed off as an aberration, electrical readings of the shrimp retina, though very difficult to carry out, also implied that the shrimp could see only green light. That was odd. If the vents glowed red, and the

shrimp could see only green, they were as good as blind. Were these strange naked retinas functionless, then, perhaps degenerate organs like the blind eyes of cave-fish? The fact that they were found on the backs of the shrimp, rather than on their heads, suggested that they were not degenerate but that conjecture hardly amounted to proof.

The proof came with the discovery of larvae. The vent world is not as eternal as it seems, and individual vents often die, choking on their own effluent, in the span of human lives. New vents erupt elsewhere on the ocean floor, often many miles away. For vent species to survive they must cross the void from dying to nascent vents. While the mobility of most adults is hampered by their close adaptations to vent conditions – just think of the giant tubeworms lacking a mouth and gut – their larvae can be disseminated in vast numbers throughout the oceans. Whether the larvae hit on new vents by chance (dispersal by deep ocean currents) or some unknown homing device (following chemical gradients, for example) is a moot point, but the larval forms are not at all adapted to the vent world. For the most part, they are found far closer to the surface, albeit still deep in the sea, at a level where the dying rays of sunlight percolate. In other words, the larvae live in a world where eyes are useful.

Among the first larvae to be identified were those of a crab known as *Bythograea thermydron*. Intriguingly, like the adult vent shrimp, the adult crab flaunts a pair of naked retinas instead of proper eyes; but unlike the shrimp these retinas are found on its head, in the place where one might expect to find eyes. However, the most striking finding was that the larvae of this crab *did* have eyes, perfectly normal eyes, at least for a crab. So when eyes were useful, the crabs had eyes.

There followed a procession of larvae. Several species of vent shrimp live alongside *Rimicaris exoculata*, but are easily overlooked, as they are solitary beasts that don't swarm in such hordes. They too turn out to possess naked retinas, on their heads rather than their backs, and like the crab their larvae have perfectly normal eyes. Indeed, the last larvae to be identified were those of *Rimicaris* itself, partly because the larvae are confoundingly similar to those of other shrimp, and partly because they, too, have quite normal eyes upon their heads.

The discovery of normal eyes on larvae was richly significant. It meant that the naked retina was not merely a degenerate eye – the end-point of generations of loss, any residual function congruent with life in virtual pitch-blackness. The larval forms had perfectly good eyes: if they preferred to lose them during maturation, that had nothing to do with generations of irreversible evolutionary loss: it was something more deliberate, whatever the costs and benefits. By the same token, the naked retina had not evolved 'up' from scratch, attaining a nominal degree of performance that could never rival a true image-forming eye in this benighted environment. Rather, as the larvae mature to the adult form, sinking down to the vents, their eyes degenerate and all but disappear, their fancy optics reabsorbed, step by careful step, leaving but a naked retina. In the case of *R. exoculata* alone, the eyes disappear altogether, and the naked retinas apparently develop from scratch on its back. All in all, a naked retina seems to be more use than a complete eye in a series of different animals: it's not a one-off, not a coincidence. Why?

The value of a naked retina lies in the balance between resolution and sensitivity. Resolution refers to the ability to see (resolve) the details of an image. It improves with a lens, cornea, and so on, as these all help focus light on to the retina, forming an image. Sensitivity is an opposing process, referring to the ability to detect photons. If we have a low sensitivity, we make poor use of available light. In our own case, we can enhance our sensitivity to light by enlarging the aperture (the pupil) and switching to a population of more light-sensitive cells (rod cells). Even so, such measures can only go so far; the mechanical contrivances needed to resolve images at all ultimately restrict our sensitivity. The only way to improve sensitivity any further is to lose the lens and enlarge the aperture indefinitely, increasing the angle through which light can enter the eye. The largest aperture of all is no aperture at all – a naked retina. Taking these factors into consideration, a fairly simple calculation shows that the naked retina of adult vent shrimp is at least 7 million times more sensitive than the fully formed eyes of their own larvae.

So by sacrificing resolution, the shrimp gain the ability to detect extremely low levels of light and, up to a point, where it comes from, at least to the nearest hemisphere: above or below, back or front. Being able to detect light at all could make the difference between life and death in a world poised

between temperatures hot enough to cook the shrimp in seconds or too cold and remote to survive. I picture a shrimp drifting off into outer space like an astronaut losing touch with his spaceship. This might explain why *R. exoculata* has eyes on its back, living as it does, in its hordes, on the ledges directly beneath the vents. It is no doubt most comfortable when detecting just the right amount of light filtering down from above on its back, its head buried beneath the thronging multitudes. Its more solitary cousins have apparently forged a slightly different deal, with naked retinas on their heads.

We'll leave the question of why the shrimp see green in a red world for later (they're not colour-blind). For now, the bottom line is that half an eye – a naked retina – is better than a whole eye, at least under certain circumstances. We hardly need to consider how much better half an eye is than no eye at all.

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A simple naked retina, a large light-sensitive spot, is in fact the departure point for most discussions of the evolution of the eye. Darwin himself imagined the process beginning with a light-sensitive spot. He's often quoted distressingly out of context on the subject of eyes, not only by those who refuse to accept the reality of natural selection, but even on occasion by scientists eager to 'solve' a problem that supposedly eluded the great man. So he's quoted, correctly, as writing:

> 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 or chromatic aberration, could have formed by natural selection, seems, I freely confess, absurd in the highest possible degree.

What is too often omitted is the very next sentence, which makes it plain that Darwin did not consider the eye an obstacle at all:

> Yet reason tells me, that if numerous gradations from a perfect and complex

> eye to one very imperfect and simple, each grade being useful to its possessor, can be shown to exist; if, further, the eye does vary ever so slightly, and the variations be inherited, which is certainly the case; and if any variation or modification in the organ be ever useful to an animal under changing conditions of life, then the difficulty of believing that a perfect and complex eye could be formed by natural selection, though insuperable by our imagination, can hardly be considered real.

In plainer terms, if some eyes are more complex than others, if differences in eyesight can be inherited, and if poor eyesight is ever a liability, then, says Darwin, eyes can evolve. All these conditions are fulfilled in plenty. The world is full of simple and imperfect eyes, from eyespots and pits, lacking a lens, to rather more sophisticated eyes that parade some or all of Darwin's 'inimitable contrivances'. Certainly eyesight varies, as anyone wearing glasses, or agonisingly losing their sight, knows all too well. Obviously we're more likely to be eaten by a tiger or hit by a bus if we don't see it coming. And of course 'perfection' is relative. An eagle's eyes have four times the resolution of our own, with the ability to detect details a mile away, while we see some eighty times better than many insects, whose vision is so pixelated that it could qualify as art.

While I imagine that most people would accept Darwin's conditions without hesitation, it is still difficult to conceive all the intermediate stages: imagining the whole continuum, if not actually insuperable, is far from being superable, to twist P. G.Wodehouse.[2] Unless each step is beneficial in itself, a complex eye can't evolve, as we've seen. In fact, though, the progression turns out to be easily superable. It has been modelled as a sequence of simple steps, shown in Fig. 7.2, by the Swedish scientists Dan-Eric Nilsson and Susanne Pelger. Each succeeding step is an improvement, beginning with a naked retina and ending with an eye similar to that of a fish, and not unlike our own. It could of course (and did) go much further. We could add an iris, capable of expanding and contracting the pupil to vary the amount of light entering the eye, from bright sunlight to evening gloom. And we could attach muscles to the lens to change its shape, pulling and squashing, enabling the eye to shift its focus from near to distant points (accommodation). But these

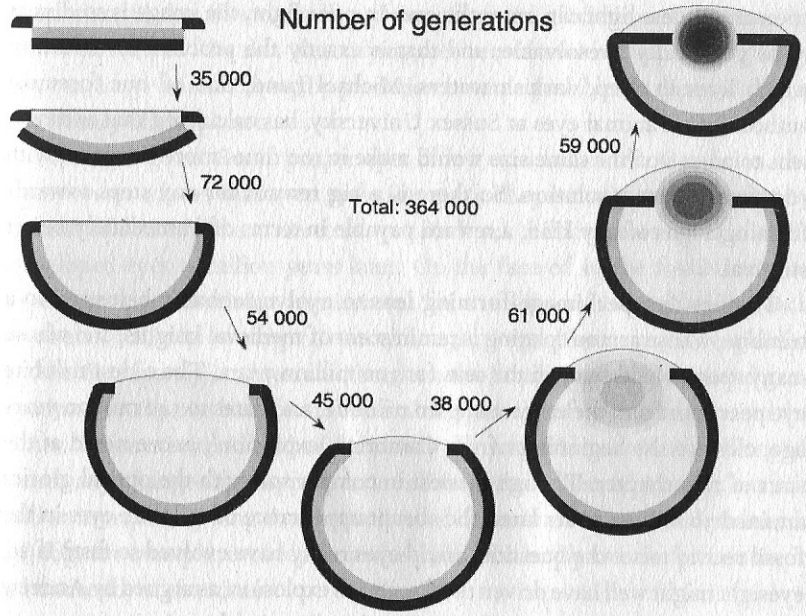

Figure 7.2 The succession of steps needed to evolve an eye, according to Dan-Eric Nilsson and Susanne Pelger, with an approximate number of generations for each change. Assuming each generation is one year, the full progression requires somewhat less than half a million years.

are finessing touches that many eyes lack; and they can only be added to an eye that already exists. So we'll content ourselves with a similar succession in this chapter – to evolve a functional image-forming eye, if still a bit clunky in the optional extras.[3]

The crucial point in this succession is that even the most rudimentary lens is better than no lens at all (anywhere other than a black smoker, of course); a blurred image is better than no image. Again, there is a trade-off between resolution and sensitivity. A perfectly good image can be formed using a pinhole camera, for instance, without a lens at all. Indeed, pinhole eyes exist in some species, notably the nautilus, a living relative of the ammonite.[4] The problem for the nautilus is sensitivity – a sharp image requires a small

aperture, so less light can enter the eye. In poor light, the image is so dim as to be practically irresolvable; and that is exactly the problem for nautilus, which lives in deep, darkish waters. Michael Land, one of our foremost authorities on animal eyes at Sussex University, has calculated that adding a lens to an eye of the same size would make it 400 times more sensitive, with 100 times better resolution. So there is a big reward for any steps towards forming a lens of any kind, a reward payable in terms of immediately better survival.

The first 'proper' image-forming lens to evolve probably belonged to a trilobite, whose armour plating is reminiscent of medieval knights, and whose many species held sway in the seas for 300 million years. The oldest trilobite eye peers out from the earliest known trilobite, and dates to 540 million years ago, close to the beginning of the Cambrian 'explosion', as we noted at the start of this chapter. Though modest in comparison with the optical glories attained 30 million years later, the abrupt appearance of trilobite eyes in the fossil record raises the question, could eyes really have evolved so fast? If so, eyesight might well have driven the Cambrian explosion, as argued by Andrew Parker. If not, then eyes must have existed earlier, and for some reason never fossilised; and if that's the case then eyes could hardly have driven the big bang of biology.

Most evidence suggests that the Cambrian explosion happened *when* it did because a change in environmental conditions permitted an escape from the straitjacket of size. The ancestors of the Cambrian animals were almost certainly tiny and lacking in hard parts, explaining the lack of fossils. This would also have prevented the evolution of useful eyes. Spatial vision requires a large lens, an extensive retina, and a brain capable of interpreting the input, and so can only evolve in animals large enough to meet these demands. Much of the groundwork, such as the naked retina and a rudimentary nervous system, was probably in place in small animals living before the Cambrian, but further developments were almost certainly stifled by small size. The immediate impetus for the evolution of large animals was most likely rising levels of oxygen in the air and sea. Large size and predation are only possible in high oxygen levels (nothing else can provide the energy necessary; see Chapter 3) and oxygen rose swiftly to modern levels shortly before the

Cambrian, in the aftermath of a series of global glaciations known as the 'Snowball Earth'. In this electrifying new environment, supercharged by oxygen, large animals living by predation became possible for the first time in the history of the planet.

So far, so good. But if proper eyes didn't exist before the Cambrian, the question resurfaces with even more force, could eyes really evolve so fast by natural selection? There were no eyes at all 544 million years ago and well-developed eyes 4 million years later. On the face of it, the fossils seem to contradict the Darwinian requirement for a million subtle gradations, each one beneficial in its own right. In fact, though, the problem is largely explained by the discrepancy in timescales, between the familiar span of lives and generations, on the one hand, and the numbing passage of geological eras on the other. When measured against the steady rhythm of hundreds of millions of years, any change that happens in a million seems indecently hasty; but it is still an inordinately long time in the lives of organisms. All our modern breeds of dog, for example, evolved from the wolf, admittedly with help from us, in a hundredth of that time.

In geological terms, the Cambrian explosion happened in the blink of an eye – no more than a few million years. In evolutionary terms, though, this is time aplenty: even half a million years ought to have been more than enough time to evolve an eye. In proposing their sequence of steps (see Fig. 7.2), Nilsson and Pelger also computed the time needed. They assumed, conservatively, that each step would be no more than a 1 per cent change to that particular structure, a slightly deeper eyeball, a touch more lens, and so on. When totting up all the steps, they were startled to find that only 400,000 individual changes (not so far from the million I've been glibly throwing around) were needed to progress from a naked retina to a fully formed eye. Then, they assumed that just one change took place per generation (although there could easily have been several at once, making this another conservative estimate). Finally, they assumed that an 'average' marine animal, in which the changes were taking place, would breed once a year. On this basis, they concluded that it would take less than half a million years to evolve a whole eye.[5]

If all these considerations are correct, then the appearance of eyes really could have ignited the Cambrian explosion. And if that's the case, then the

evolution of the eye must certainly number among the most dramatic and important events in the whole history of life on earth.

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There is one troubling step in Nilsson and Pelger's progression: the first stage in building a lens. Once a primitive lens exists, it's easy to see how natural selection could modify and improve it; but how are the requisite components assembled in the first place? If the various bits and pieces needed to build a lens have no use on their own, should they not be unceremoniously ditched by natural selection before building works can begin? Might this difficulty perhaps explain why the nautilus never developed a lens, even though it would have benefited from doing so?

Actually the question is a non-question, and for now at least the nautilus must remain an oddity for unknown reasons, as most species found a way (including the closest living relatives of the nautilus, octopus and squid); and some found stunningly inventive ways. Although the lens is manifestly a specialised tissue, its construction has been strikingly opportunistic, time and again, its basic building blocks pilfered from any handy nearby source, from minerals and crystals to enzymes, even just bits of the cell.[6]

The trilobite is an excellent example of such opportunism. It really could have transfixed you with a stony eye, for, uniquely, trilobite lenses were made of crystal, the mineral calcite. Calcite is another name for calcium carbonate. Limestone is an impure form; chalk a much purer form. The white cliffs of Dover are almost pure calcite, formed from tiny disorganised crystals that scatter light randomly, giving chalk its white colour. In contrast, if the crystals grow slowly (often in mineral veins) calcite can form into fine, clear structures with a slightly wonky cube shape, known as rhombs. Rhombs have a curious optical property, which arises naturally from the geometry of the constituent atoms: they deflect light from all angles, except one privileged axis straight across the middle. If light enters a rhomb along this axis, dubbed the *c*-axis, it passes straight through, unhindered, as if ushered along a red carpet. This curious property the trilobites turned to their advantage. Each one of the many eye facets wields its own tiny calcite lens along this privileged *c*-axis

(see Fig. 7.3). Light can pass through each lens from only one direction to the underlying retina.

Quite how the trilobites grew crystal lenses, aligning whole arrays with the correct orientation, is unknown and is likely to remain so, as the last one expired in the Permian extinction, 250 million years ago. But the fact that trilobites are silenced by this vast tract of time doesn't mean that there is no way of knowing how it might have come about. One good clue cropped up from an unexpected source in 2001. It seems that the trilobite lens is not as unique as once thought: one living animal, a brittlestar, also makes use of calcite lenses to see.

There are around 2,000 species of brittlestar, each sprouting five arms like their starfish cousins. Unlike starfish, the brittlestars have long, slender arms hanging down, which snap off if pulled upwards (hence their name). All brittlestars have skeletons made from interlocking calcite plates, which also form spikes on their arms used for grasping prey. Most of them are insensitive to light, but one species, *Ophiocoma wendtii*, confounds observers by scuttling for dark crevices a metre away at the approach of a predator. The trouble is it doesn't have eyes or, at least, so everyone thought until a research team from Bell Labs noticed arrays of calcite knobs on its arms, resembling the lenses of trilobites (see Fig. 7.4). They went on to show that these knobs do indeed function as lenses, focusing light on to photosensitive cells below the lenses.[7] Though lacking anything we'd recognise as a brain, the brittlestar has functional eyes. As *National Geographic* had it at the time: 'In a twist of nature, the sea has eyes in its stars.'

How do the brittlestar lenses form? Although many details are still missing, in broad terms they form in the same way as other mineralised biological structures, like the spines of sea urchins (also made of calcite). The process begins within cells, where high concentrations of calcium ions interact with proteins, which bind them into fixed positions, 'seeding' crystal formation in the same way that an optimist, standing outside an empty grocery store, used to seed a queue in the Soviet Union. One person, or one atom, is immobilised, and the rest stick to that.

In a celebration of reductionism, if the proteins responsible for seeding the calcite crystals are purified and smeared on a sheet of paper, then placed

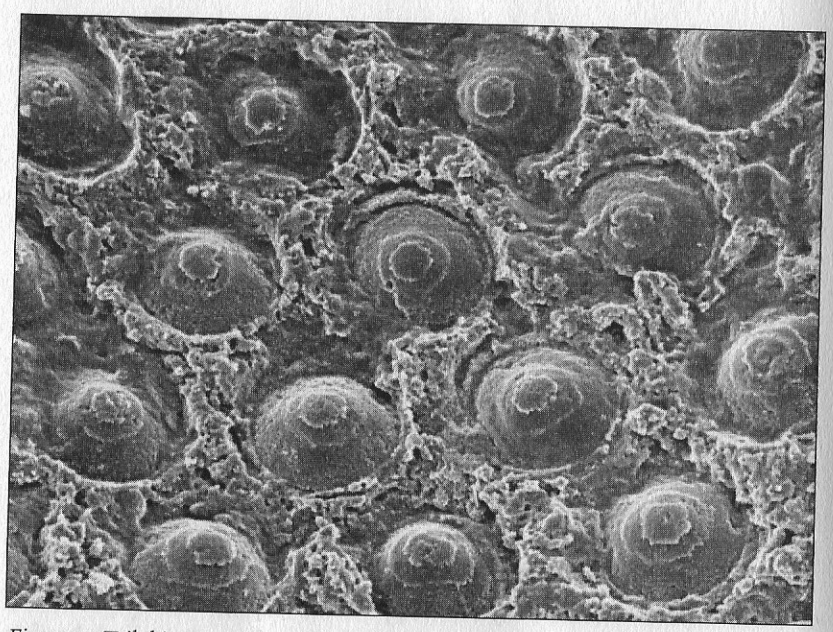

Figure 7.3 Trilobite crystal lenses from *Dalmanitina socialis*, found in Ordovician rocks in Bohemia, Czech Republic; showing details of the inner surface of the lenses; approximately half a millimetre across.

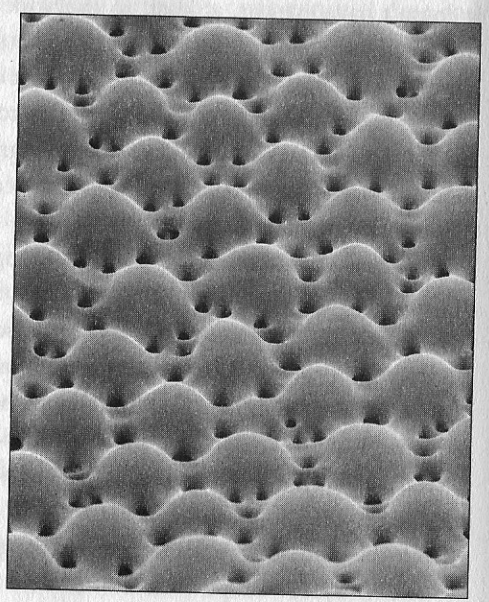

Figure 7.4 The crystal lenses of the brittlestar *Ophiocoma wendtii*, which are found on the skeletal plates at the top of each arm, protecting the joints.

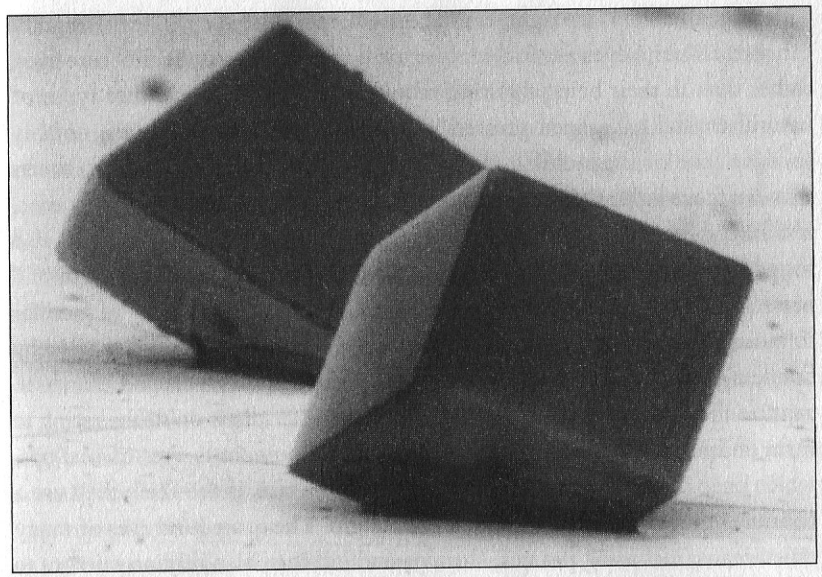

Figure 7.5 Rhombic crystals of calcite, growing on paper smeared with acidic proteins from mollusc shells, placed in a strong calcium carbonate solution. The optical *c*-axis, the only direction in which light passes through the crystal without scattering, is pointing straight up.

in a strong solution of calcium carbonate, perfect crystals grow right there on the paper, forming into rhombs with their optical *c*-axis pointing straight up, just as in a trilobite lens (see Fig. 7.5). There's even a hint of how it came about in the first place: the exact choice of protein doesn't matter much. It matters only that the protein should bristle with acid side-chains. Back in 1992, a decade before anyone knew about the brittlestar lenses, the biomineralologists Lia Addadi and Stephen Weiner grew lovely calcite lenses on a sheet of paper, using acid proteins isolated from mollusc shells, which certainly can't see. In other words, marvellous as it all is, the entire process takes place spontaneously when common proteins mix with common minerals. It is marvellous, certainly, but no more miraculous than the fantastic bristling arrays of crystals found in natural caverns like the Cave of the Swords in Mexico.

Yet for all their sharp sight, crystal eyes were a blind alley. The real significance of trilobite eyes lies in their historical importance, as the first true eyes, rather than in their being a lasting monument of evolution. Other types of natural crystal have been pressed into service by other creatures, notably guanine (one of the building blocks of DNA), which crystallises into sheets that can focus light. Guanine crystals give fish scales their silvery iridescence, and have been added to many cosmetics for the same reason; they're also found in (and take their name from) guano, the dried excrement of birds and bats. Similar organic crystals function as biological mirrors; they're familiar to most people as the 'reflectors' in cats' eyes. They improve night vision by bouncing light back into the retina, giving the receptors a second chance to capture the few scarce photons. Other mirrors focus light on to the retina to form an image. These include the beautiful and numerous eyes of scallops, which peep out from between the tentacles at the rim of the shell; they use a concave mirror beneath the retina to focus light. The compound eyes of many crustaceans, including prawns, shrimps and lobsters, also rely on mirrors to focus light, again using natural crystals along the lines of guanine.

In general, though, the central thrust, and the greatest glories, of evolution have been lenses composed of specialised proteins, like our own. Are these, too, opportunistic constructions, cobbled together from existing components that have other uses about the body? Although it's sometimes claimed that evolution is a historical science, and so cannot be proved one way or another, it does in fact make very specific predictions that can be tested. In this case, the theory predicts that the lens proteins should be recruited from among existing proteins, with other uses about the body, on the grounds that a specialised lens protein could not possibly evolve before the existence of the lens itself.

The human lens is obviously an extremely specialised tissue: it is transparent; blood vessels are excluded; and the cells have lost almost all their normal features, instead concentrating proteins into a liquid crystal array, capable of bending light to form a clear image on the retina. And, of course, the lens is capable of changing shape to alter the depth of field. What's more, the extent to which light is bent varies across the lens, evading faults like spherical aberration (where light passing through the centre or the edge of the lens is focused at different points). Given all this we might conjecture that the proteins needed

to fabricate such a refined array would be unique, with optical properties simply not found in mundane everyday proteins. But if we did, we'd be totally wrong.

The proteins found in the human lens are called crystallins, named in the expectation that they would indeed possess unique properties. They make up some 90 per cent of all proteins in the lens. Because lenses are so similar in different species, in both their appearance and function, it seemed reasonable to assume that all were composed of a similar protein. Yet when techniques for comparing the sequence of building blocks in different proteins became widely available, from the early 1980s, the reality came as a surprise. The crystallins are *not* structural proteins and most of them are not even specific to the lens; all do other jobs elsewhere about the body. Even more unexpectedly, many crystallins turned out to be enzymes (biological catalysts) with normal 'housekeeping' functions somewhere else in the body. The most abundant crystallin in the human eye is called α-crystallin, for instance; it is related to a stress protein first found in the fruit fly *Drosophila*, and now known to be widespread in animals. In humans, it functions as a 'chaperone', which is to say, it shields other proteins from damage. As such, it's not only found in the eye, but also in the brain, liver, lung, spleen, skin and small intestine.

To date, eleven types of crystallin have been catalogued, only three of which are found in the eyes of all vertebrates; the rest vary from one group to another, implying they have been 'recruited' to work in the lens quite independently, again exactly as predicted for the ad hoc approach of natural selection. We won't dwell on the names or functions of these enzymes, but it's shocking that this group of metabolic proteins, each with its own task about the cell, has been plucked out and pressed into an utterly different service. It's as if the army conscripted only tradesmen, and members of guilds at that, to form the standing army. But whatever the reasons, there is nothing about this eccentric policy to suggest that it's particularly hard to recruit lens proteins.

All in all, there is nothing special about lens proteins: they are plucked from elsewhere in the body and pressed into service. Virtually all proteins are transparent, so colour is not an issue (only proteins coupled to pigments, like haemoglobin, have much colour of their own). Changes in optical properties like the degree of light-bending (refraction) across the lens are achieved simply

by varying protein concentration, which certainly requires finesse, yet is hardly a big conceptual stumbling block. Why so many lens proteins are enzymes, if there is any reason at all, is unknown; but whatever the reason, perfectly formed lens proteins plainly did not leap from the head of Zeus.

A window into how all this comes about is the lowly invertebrate known by the undignified name *sea squirt* (specifically *Ciona intestinalis*, or literally 'pillar of intestines'; Linnaeus was hardly any more kindly). The adult form gives away little of its heritage, being essentially a translucent bag attached to a rock, bearing two yellowish swaying siphons, through which water enters and exits. They are so common in coastal waters around the UK that they're considered a pest. But the larvae divulge the squirt's deep secret, and show them to be much more than just a pest. The larvae look a little like tadpoles and can swim around, making use of a rudimentary nervous system and a pair of primitive eyes, which lack lenses. Once the little squirt has found a suitable home, it attaches itself soundly to the spot and then, needing it no longer, reabsorbs its own brain (a feat that arouses much admiration among university professors, Steve Jones quips).

Though the adult sea squirt is unrecognisable as anything related to ourselves, the tadpole larvae give the game away: the sea squirt is a primitive chordate, which is to say it has a notochord, a forerunner of the spinal chord. This places it among the earliest branches of the chordates, and so all vertebrates. In fact it split from the vertebrates before the evolution of the lens. And this means that the sea squirt, with its simple eye, might give an insight into how the vertebrate lens formed in the first place.

And so it does. In 2005, Sebastian Shimeld and his colleagues in Oxford found that, despite lacking a lens, *C. intestinalis* does have a perfectly good crystallin protein, not in its eye, but tucked away in its brain. Who knows what it's doing there, but that's not relevant to us here. What is relevant is that the same genes that direct lens formation in vertebrates also control the activity of this protein; in the squirt they function in the brain as well as in the eye. So the entire apparatus for building a lens was present in the common ancestor of the vertebrates and sea squirts, before each went its own way. A small switch in regulation in vertebrates transferred the protein from brain to eye. Presumably, similar poaching forays account for the repeated conscription of

other crystallins from elsewhere in the body, some in the common ancestor of vertebrates, and others more recently in specific groups. Why the sea squirt line failed to make a simple switch in resource use is a mystery; perhaps a rock is not a hard place to find, even without a lens. Even so, the sea squirt is the odd one out. Most vertebrates did succeed – it happened at least eleven times. And so there are no particularly difficult steps in the sequence to make an eye.

From this riot of appropriated proteins, crystals and minerals that make up the lens of diverse species, the proteins of the retina stand in stark contrast. One protein in particular stands out: the molecule responsible for sensing light, rhodopsin. Recall the vent shrimp *Rimicaris exoculata*, with its naked retinas. Despite the utter peculiarity of the deep ocean vent world, despite the strangeness of the naked retinas down its back, despite the shrimp's ability to detect a faint glow where we cannot, despite living on sulphur bacteria, having blue blood and lacking a backbone, despite last sharing a common ancestor with us around 600 million years ago, long before the Cambrian explosion, despite all this, the vent shrimp use the same protein that we do to see. Is this deep link across time and space no more than an uncanny coincidence, or is it something more significant?

The shrimp's protein and ours are not exactly the same, but they're so similar that if you turned up in court and tried to convince a judge that your version was not a badly concealed plagiarism, you'd be very unlikely to win. In fact, you'd be a laughing stock, for rhodopsin is not restricted to vent shrimp and humans but is omnipresent throughout the animal kingdom. We know little about the inner workings of the trilobite eye, for example, which were not preserved along with its crystal lenses; but we know enough about its relatives to say with some certainty that its eyes would have contained rhodopsin. Every animal, with remarkably few exceptions, relies on exactly the same protein. Trying to persuade a judge that your rhodopsin is not plagiarised would be like trying to claim that your television set is fundamentally different from everyone else's, just because it's bigger or has a flat screen.

This remarkable conformity could conceivably have come about in several ways. It could mean that everyone inherited the same protein from a common ancestor. There's been a lot of little changes in the last 600 million years, of course, but it's still obviously the same protein. Or it could mean that there are such serious design constraints on molecules able to detect light at all that everyone has been forced to come up with basically the same thing. That would be like watching television on a computer screen, a case of different technologies converging on a similar solution. Or, finally, it could mean that the molecule has been passed around freely from one species to another, in a case of rampant theft rather than inheritance.

It's easy enough to discard the third option. Gene theft does exist between species (genes are moved around by viral infections, for example) but it's not common outside bacteria; and when it happens it sticks out like a sore thumb. The catalogue of trifling differences between proteins across species can be superimposed over the known relationships between the species. If the human protein happened to be stolen and inserted into a vent shrimp, it would stare back at us like an illegal alien, clearly related to humans rather than shrimp. On the other hand, if the differences had gradually accumulated over time in the ancestors of the shrimp, then the shrimp protein would be most similar to its close relatives, the prawns and lobsters, and would be most different from its most distant relatives, like us; and this is indeed the case.

If it wasn't stolen, was rhodopsin reinvented from engineering necessity? This is harder to say for sure as there is indeed a sense of reinvention, if only once. The vent shrimp's rhodopsin is about as distant from our own as it's possible to get, for two very similar molecules. In between the two is a spectrum of intermediates, but this spectrum is not quite continuous. Instead, it falls into two groups, roughly corresponding to the vertebrates on the one hand and the invertebrates on the other (including the shrimp). This difference is magnified by a whole context of opposites. In both cases, the light-sensitive cells are modified nerve cells, but there the resemblance ends. In shrimp and other invertebrates, the rhodopsin is plugged into membranes that sprout from the top of the cell like spiky hair (microvilli); in vertebrates, a single projection (a cilium) protrudes from the top of the cell like a radio mast. This mast is convoluted into a succession of deep horizontal folds,

making it look more like a stack of discs sitting on top of the cell.

Inside the photoreceptor cells, these differences have their counterparts in biochemistry. In the vertebrates, when light is absorbed, a cascade of signals strengthens the electric charge across the membrane of the cell. The invertebrates do exactly the opposite: when light is absorbed, a completely different cascade causes the membrane to lose its electric charge altogether; and it is this that triggers the nerve to fire off its message *light!* to the brain. All in all, two rather similar rhodopsins are found in utterly contrasting cell types. Does all this mean that the photoreceptor cells evolved twice, once in the invertebrates, and again in vertebrates?

That certainly sounds like a plausible answer and was exactly what most of the field believed until the mid-1990s, when suddenly everything changed. None of the facts is wrong; it's just that they turned out to be only half the story. Now it looks as if everyone uses rhodopsin because everyone inherited it from a common ancestor. It looks as if the earliest precursor of the eye only evolved once.

The iconoclastic Swiss developmental biologist Walter Gehring, at the University of Basel, has promulgated this revision most forcefully. One of the discoverers of the *hox* genes (responsible for laying out body plan), Gehring went on to make a second monumental discovery in 1995, in one of the most startling experiments in biology. Gehring's team took a gene from a mouse and inserted it into the fruit fly *Drosophila*. This was no ordinary gene, with a minor ensemble role: under its malign guidance the fruit fly suddenly started sprouting whole eyes on its legs, wings and even antennae (see Fig. 7.6). These strange diminutive eyes peeping out from peculiar places were not the familiar camera eyes of mice and men, but compound eyes, displaying all the arrays of facets characteristic of insects and crustaceans. What this gruesome experiment proved with visceral force was that the genes needed for growing an eye in a mouse and in a fly were the same: they had been preserved, with amazing fidelity, down 600 million years of evolution, ever since the last common ancestor of vertebrates and invertebrates, to the point that they were still interchangeable. Put the mouse gene in a fly and it took over the fly systems, wherever it was placed, commanding the subordinate hierarchy of fly genes to build an eye right there on the spot.

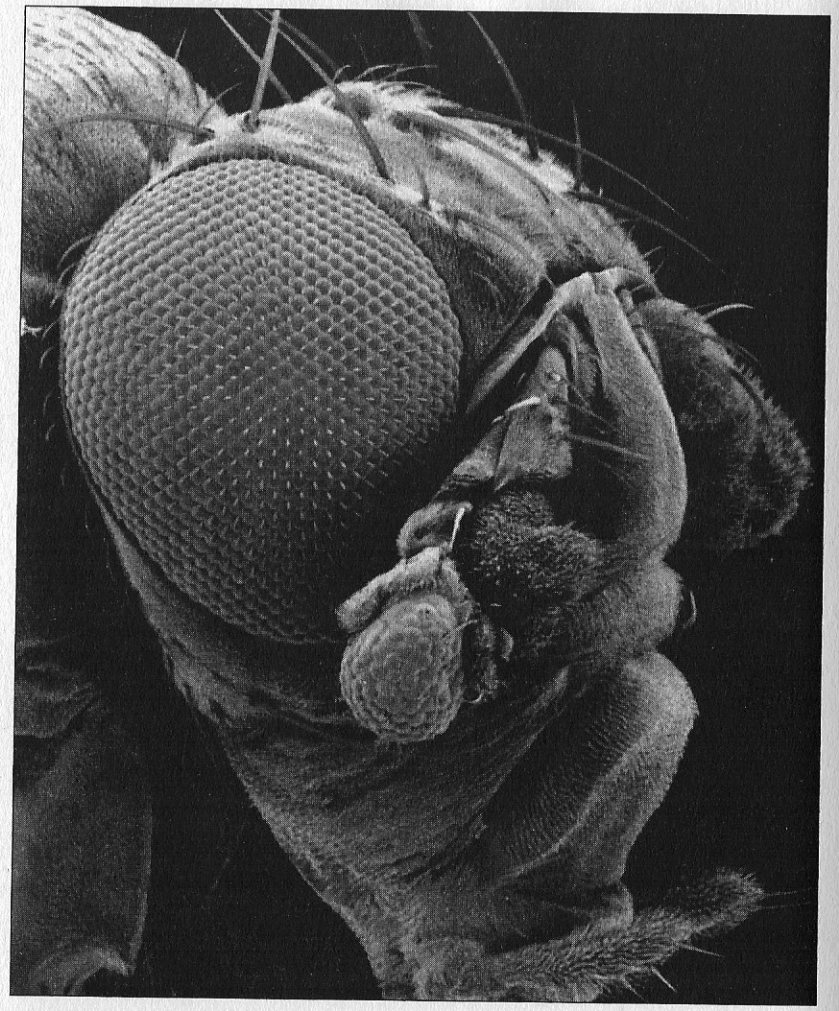

Figure 7.6 Scanning electron micrograph of the head of a fruit fly (*Drosophila*), showing a diminutive extra eye on the antenna, induced by genetically engineering a mouse *Pax6* gene. The same gene controls eye development in both vertebrates and invertebrates, and must have done so in their common ancestor, perhaps 600 million years ago.

Nietzsche had once taught in Basel, and perhaps in homage, Gehring referred to the mouse gene as a 'master gene'. I wonder whether 'maestro gene' might have been more appropriate; certainly less bombastic and perhaps more plural. Like an orchestral conductor conjuring up the most beautiful music without sounding a note himself, the gene calls forth the structures of the eye by ushering in individual players, each with their own part to play. Different versions of the same gene were already known through their mutations in flies, mice and men. In mice and flies, the gene was called *Small eye* and *Eyeless*, respectively, referring, in the inverted terms to which geneticists are horribly prone, to the degree of deficit in its absence. In our own case, mutations in the same gene cause the disease aniridia, in which the iris fails to develop; though an unpleasant and frequently blinding condition, a curiously limited outcome for a master gene supposed to supervise the whole construction of the eye. But that's if only one copy of the gene is damaged. If both copies are damaged or lost, the entire head fails to develop.

The picture has grown more complex since Gehring's seminal experiment. His 'master gene' is now known as *Pax6*, and is both more powerful and less lonely in its elevation than had seemed. *Pax6* has since turned up in practically all vertebrates and invertebrates, including the shrimp; a closely related gene is even found in jellyfish. And it turns out that *Pax6* is not only behind the formation of eyes but also large parts of the brain; hence the lack of head development when both copies are missing. At the same time, *Pax6* is not alone. Other genes, too, can summon up whole eyes in *Drosophila*; in fact it seems to be a peculiarly easy thing to do. These genes are all plainly related to each other and are very ancient. Most of them are found in both invertebrates and vertebrates, albeit with slightly differing roles and contexts. Sadly, the beautiful music of life is called forth not by a conductor but by a small committee.

The bottom line is that the same committee of genes controls eye formation in both vertebrates and invertebrates. Unlike rhodopsin, there is no practical 'engineering' reason for the process to be controlled by the same genes; they are all faceless bureaucrats and might just as easily have been a different bunch of faceless bureaucrats. The fact that it's always the same bunch (unlike the lens proteins, for example) betrays the hand of history, the quirkiness of

happenstance rather than the force of necessity. And this history suggests that the photoreceptor cell evolved just once, in a common ancestor of the vertebrates and invertebrates, under the control of a small committee of genes.

There's another reason to believe that the photoreceptor evolved just once – the direct testimony of a living fossil. The survivor is a tiny marine ragworm, *Platynereis*, a few millimetres long and covered in bristles. A denizen of muddy estuaries and favoured bait of anglers, one wonders how many know that its overall shape and morphology has barely changed since Cambrian times. A worm like this was the common ancestor of both the vertebrates and invertebrates. Like all vertebrates and many invertebrates, the ragworm is bilaterally symmetrical: it's the same on both sides, unlike a starfish. This symmetry makes all of us *bilaterians*, an insect as much as you or me. Crucially, the ragworm evolved before this design, pregnant with potential, exploded into all the marvellous incarnations we see about us today. It is a living fossil of the primordial bilaterian, the ur-bilaterian, and that's why Detlev Arendt and his colleagues at the European Molecular Biology Lab in Heidelberg were interested in its photoreceptor cells.

They knew that the ragworm's eyes are similar in their design to the invertebrates, rather than the vertebrates, right down to the type of rhodopsin they use. But in 2004 the Heidelberg team discovered another clutch of light receptors, buried away in its brain. These were not used for seeing at all, but rather for the circadian clock, those internal rhythms that govern sleep and wakefulness and distinguish night from day, even in bacteria. Not only did the circadian clock cells use rhodopsin, but they were instantly recognisable (to experts like Arendt, at least) as *vertebrate* photocells, a recognition later confirmed by more detailed biochemical and genetic tests. The ur-bilaterian, Arendt concluded, possessed both types of photocell. And that meant that the two types hadn't evolved independently in totally different lines, but rather were 'sister' cells that had evolved together in the same organism – an ancestor of the ur-bilaterian.

Of course, if this common ancestor of vertebrates and invertebrates possessed both types of photocell, then we might have inherited both, if only we knew where to look. And so, it seems, we did. The year after the living fossil offered up its secrets, Satchin Panda and his colleagues at the Salk Institute,

San Diego, followed up a hunch about some cells in our own eye – the retinal ganglion cells – which influence human circadian rhythms. Although not specialised for light detection, they too possess rhodopsin. It's an unusual form known as melanopsin; and it turns out to be characteristic of *invertebrate* photocells. Remarkably, this circadian rhodopsin in our own eyes is closer in its structure to the rhodopsin in the naked retinas of the vent shrimp than it is to the other type of rhodopsin that shares the human retina.

All this implies that the vertebrate and invertebrate photocells sprang from the same source. They are not separate inventions, but sister cells with a mother in common. And that mother cell, the primordial photocell, the ancestor of all animal eyes, evolved just once.

The bigger picture that emerges, then, is this. A single type of light-sensitive cell, containing the visual pigment rhodopsin, evolved in a common ancestor of the vertebrates and invertebrates, under the control of a small committee of genes. Later, this light-sensitive cell was duplicated, and the two daughter cells became specialised to function either in eyes or in a circadian clock. For reasons that may have been no more than chance, the vertebrates and the invertebrates each selected opposing cell types for these tasks, so that eyes developed from different tissues in the two lines, giving rise to major embryonic differences between similar eyes in, for example, the octopus and mankind. The first station en route to the complete eye was a naked retina: a sheet of light-sensitive cells, composed of one or the other type of light-sensitive cell, according to the lineage. Some organisms still retain simple, flat, naked retinas, while in others the sheet became recessed into pits, able to cast a shadow and give a sense of where the light came from. As these pits deepened, the trade-off between sensitivity and resolution meant that any form of lens was better than none at all; and all kinds of unexpected materials, from minerals to enzymes, were recruited to the task. A similar process took place in different lines, giving rise to a cacophony of lens types; but the optical constraints on building a functional eye restricted this variety at the molecular level to a small range of large-scale structures, from camera eyes like our own to the compound eyes of insects.

There are, of course, innumerable details to fill in; but this, in broad brush stroke, is how the eye evolved. No wonder that we share the same rhodopsin

with vent shrimp: we all inherited it from the same ancient ancestor. But that still leaves us with one big question to conclude this chapter – who was this ancestor? The answer, once more, lies in the genes.

✦

Down in the deep-sea vents, Cindy Van Dover was worried about light. Her vent shrimp could apparently detect green light with extraordinary sensitivity, using a rhodopsin similar to that in our own eye; and yet the early measurements showed that the vents did not glow green. What was going on?

In a wry bit of advice to young scientists, offered during his retirement speech, an eminent researcher remarked that on no account should one ever repeat a successful experiment: it will certainly turn out a bitter disappointment.[8] The converse – never hesitate to repeat a failure – is less obviously true, yet Van Dover had good reason to try. Like a dead man, rhodopsin doesn't lie. If it absorbs green light, she reasoned, then there must be some green light there to absorb. Presumably, the rudimentary equipment used in the early studies was just not as sensitive as a shrimp's naked retina.

A new and altogether more sophisticated photometer was commissioned from the space scientists at NASA, who knew all about detecting radiation in the inky blackness of outer space. Named ALISS (Ambient Light Imaging and Spectral System), the device duly did detect light at other wavelengths. Down in the wonderland of the vent world, ALISS charted a small peak in the green part of the spectrum with an intensity orders of magnitude greater than predicted on the basis of theory. The new measurements were soon corroborated at other vents. Although the source of this eerie green glow is still a mystery, there is no shortage of exotic hypotheses. Small bubbles of gas emerging from the vents and crushed in the high pressure of the ocean can give rise to visible light, for instance, as can the formation and shock fracture of crystals under heat and pressure.

If Van Dover's faith in rhodopsin was well placed, she was only playing the odds. Rhodopsin has an impressive ability to track the conditions. The deep blue sea is so called because blue light penetrates further through water than do other wavelengths. Red light is soon absorbed by water, and can't

penetrate far; yellow light gets a little further; orange further still. But from about twenty metres down, most light is green and blue, becoming bluer with depth. The blue light scatters around, making everything in the ocean deep a shade of blue. The rhodopsin pigments of fish track this blue shift with finesse, a trick known as spectral tuning. So around 80 metres down, we find fish that have rhodopsins which absorb green light best (at around 520 nanometres), but by 200 metres down, in the last fading embers of light, the fish possess rhodopsins that absorb blue light (at about 450 nanometres). Interestingly, the vent crab *B. thermydron*, which we met earlier, reverses this shift as it moves to the vent. The larvae of the crab live in deep blue waters and have a rhodopsin that absorbs blue light best, at a wavelength of 450 nanometres. In contrast, the naked retina of the adult has a rhodopsin that absorbs light at a wavelength of 490 nanometres, closer to the green. The shift is small but deliberate. Given that the rhodopsin of vent shrimp, too, absorbs green light at 500 nanometres, Van Dover's antennae had every reason to twitch.

Our own colour vision depends on the ability of rhodopsin to shift wavelengths. We have two types of photoreceptor in our retina, the rods and cones. Strictly speaking, only the rod cells contain rhodopsin; the cones contain one of three 'cone opsins'. But in reality this distinction is unhelpful, as all of these visual pigments are basically the same in structure: all are composed of a particular type of protein – an 'opsin', plugged across the membrane with a sevenfold zigzag – bound to a derivative of vitamin A called retinal. Retinal is a pigment, and as such is the only bit responsible for absorbing light. When it absorbs a photon it changes shape from a kinked to a straight form, and this is enough for it to set in motion the whole biochemical cascade that in the end signals *light!* to the brain.

Although it is the retinal that absorbs light, by far the most important factor for 'spectral tuning' is the structure of the opsin protein. Small changes in structure can shift its absorption from the ultraviolet (about 350 nanometres) in insects and birds to the red (about 625 nanometres) in chameleons. So by combining several slightly dissimilar opsins, each one with a different absorption, colour vision is possible. Our own cone opsins absorb light maximally in the blue (433 nanometres) green (535 nanometres) and red (564 nanometres) parts of the spectrum, together giving our familiar visible range.[9]

While the opsins are broadly similar in their overall structure, the differences between them divulge a fascinating history of life. All were formed by duplication followed by divergence, and can ultimately be traced back to an ancestral opsin gene. Plainly, some of these duplications happened more recently than others. Our 'red' and 'green' opsins are closely related, for example: the gene was duplicated in a common ancestor of the primates. This duplication gave the primates three types of cone opsin (or it did after they'd diverged a bit, anyway) rather than two, giving most of us three-colour (trichromatic) vision. A few unfortunates who are red–green colour-blind have lost one of these genes again, making them dichromatic, like almost all other mammals, their poor vision reflecting, perhaps, a relatively recent nocturnal past spent hiding from dinosaurs. Why the primates regained three-colour vision is disputed. The most popular theory suggests that it helped to spot red fruit against green leaves; an alternative, more socially oriented idea, argues it helped discriminate emotions, threats and sexual signals, from blushing to barefaced lies (and it's interesting that all trichromatic primates are bare-faced).

I said the primates 'regained' three-colour vision, but in fact we're still the poor relations among other vertebrates. Reptiles, birds, amphibians and sharks all have four-colour vision, and it seems likely that the common ancestor of the vertebrates was tetrachromatic, with an ability to see into the ultraviolet.[10] A lovely experiment confirmed the possibility: by comparing the gene sequences of living vertebrates, Yongsheng Shi and Shozo Yokoyama, at Syracuse University, New York, predicted the sequence of the ancestral vertebrate gene. As yet we have no way of guessing, from first principles alone, exactly what wavelength this ancestral rhodopsin would have absorbed. Nothing daunted, Shi and Yokoyama used genetic engineering techniques to build the protein, and then measured its light absorption directly. It duly turned out to be in the ultraviolet (360 nanometres).

The deepest branch in the tree of opsins lies between the vertebrates and the invertebrates, as we've seen. But even that living fossil, the ur-bilaterian ragworm *Platynereis*, still has two types of opsin, corresponding to those of the vertebrates and the invertebrates. So what did the grand ancestor of all animal opsins look like, and where did it come from? The answer is not known for sure, and several hypotheses jostle for prominence. But our guide has been

the gene itself, and in using it we have traced our way back through 600 million years. How much further back can we go? According to Peter Hegemann and his colleagues at Regensburg University in Germany, the gene does indeed give an answer, and one that is utterly unexpected: the earliest progenitor of the eye, they say, evolved in *algae*.

Algae, like plants, are masters of photosynthesis, and can call upon all kinds of sophisticated light-sensitive pigments. Many algae use these pigments in simple eyespots to register the light intensity and, if necessary, to do something about it. So, for example, the luminously beautiful algae *Volvox* forms into hollow spheres composed of hundreds of cells, up to one millimetre across. Each cell wields two flagella that poke out like oars; these beat in the dark, but stop in bright light, steering the whole sphere towards the sun, tracking the best conditions for photosynthesis. The command to stop is controlled by the eyespots. The surprise is that the light-sensitive pigment in the eyespots of *Volvox* is rhodopsin.

Even more unexpectedly, the *Volvox* rhodopsin looks as if it is ancestral to all animal opsins. The site where retinal binds to the protein contains sections that are exactly the same as both the vertebrate and the invertebrate opsins, practically a mixture of each, in fact. And the overall structure of the gene, with its eclectic mixture of coding and non-coding sequences (technically known as introns and exons), also betrays an ancient link to both vertebrate and invertebrate opsins. It's not proof, but it is exactly what one would predict for an ancestor of both families. And that means there's a good chance that the mother of all animal eyes was, of all things, a photosynthetic alga.

That, of course, raises the question: how on earth did algal rhodopsin get into animals? Certainly the lovely *Volvox* is not on a direct line to the animals. But a quick look at the eyespot structure immediately suggests a clue: the rhodopsin is embedded in the membranes of the *chloroplasts*, those tiny structures in algae and plant cells responsible for photosynthesis. A billion years ago, the ancestors of the chloroplasts were free-living photosynthetic bacteria, namely cyanobacteria, which were engulfed by a larger cell (see Chapter 3). That means that eyespots are not unique to *Volvox*, necessarily, but to the chloroplasts, or perhaps even their forebears, the cyanobacteria.[11] And chloroplasts are found in many other types of cell too, including a few protozoa,

some of which *are* among the direct ancestors of the animals.

The protozoa are single-celled organisms, the best known of which is the amoeba. The seventeenth-century Dutch pioneer of the microscope Antony van Leeuwenhoek first saw them, along with his own sperm, and memorably referred to them as 'animalcules', distinguishing them from the microscopic algae, which he classified with plants as being basically vegetable. But this simple division hid a multitude of sins, for if we magnified some of these little animalcules up to our own size, we'd be terrorised by monsters that are half beast, half vegetable, staring back at us like the paintings of Arcimboldo. In more sober terms, some motile protozoa that swim around in pursuit of prey also contain chloroplasts, giving them an algal dimension, and indeed they acquired them in exactly the same way as the algae, by engulfing other cells. Sometimes these chloroplasts remain functional, backing up the dietary needs of their host; but in other cases they degenerate, leaving behind their characteristic membranes and genes as a fading memory of a once-glorious past, or, like the miscellaneous bits and pieces in the workshop of a tinker, the basis of a new invention, an invention, perhaps, like an eye. And exactly such a microscopic chimera, rather than *Volvox* itself, is the kind of creature that some researchers (notably Walter Gehring again) speculate may conceal the mother of all animal eyes.

Which tiny chimera? No one knows, but there are beguiling clues, and much to learn. Some protozoa (dinoflagellates) have astonishingly complex mini-eyes, with a retina, lens and cornea all packed into the same cell. These eyes seem to have developed from degenerate chloroplasts, and they too use rhodopsin. Whether animal eyes developed from them directly or indirectly (via a symbiosis) in this teeming and little known microcosm, is an open question. Whether it happened as a predictable step or as an outrageous freak of fortune, we can't say. Yet this kind of question, at once specific and universal, is the very stuff of science, and I hope that it will inspire a rising generation with eyes in their stars.

8

HOT BLOOD

Breaking the Energy Barrier

Time flies by when you're the driver of a train, runs a children's lyric. And who can't remember the reverse as a child – the endless minutes of mind-numbing tedium in the back of a car, asking repeatedly, 'Are we there yet, Daddy?' I imagine most readers will also remember the distress of watching their ageing grandparents, or parents, slow down to a snail's pace, in the end sitting inscrutably as hours pass by like minutes. Both extremes are far removed from the tempo of our own world, the andante of an adult human being.

We don't need Einstein to tell us that time is relative. But what Einstein established rigorously for time and space is, as ever, more impressionistic in biology. As the celebrated wag Clement Freud had it: 'If you resolve to give up smoking, drinking and loving, you don't actually live longer, it just seems longer.'[1] Yet there is a real sense in which time rushes through childhood, and crawls through old age. It lies in our internal settings, our metabolic rate, the rate at which our hearts beat and our cells burn up food in oxygen. And even among adults there are striking differences between the active and the slovenly. Most of us shift slowly from one to the other. The rate at which we slow down, or indeed gain weight, depends much on our metabolic rate, which varies innately between individuals. Two people who eat the same and exercise equally will often differ in their tendency to burn off calories while at rest.