lacking *BUD6*, a gene with well-studied functions in cell polarity, are reported to allow ERC segregation into the daughter, reducing the retention of this aging factor in the mother and resulting in her enhanced longevity [17].

Another important question is whether the impacts of cellular asymmetry identified in yeast also play a role in mammalian cell aging. Adult stem cells can divide asymmetrically to produce another stem cell and a cell committed to differentiation toward a specific lineage. A recent study found that damaged proteins in *Drosophila* are distributed asymmetrically, becoming enriched in either the differentiating progeny or the stem cell, depending on the type of stem cell interrogated [18]. Given that the ability of adult stem cells to re-populate damaged tissue declines with age [19], it will be critical to understand how asymmetric segregation of aging factors during cell division influences stem cell fitness and regenerative potential.

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Neuroscience: Who Needs a Parasol at Night?

New measurements of nerve cells in the eye show how very dim lights are processed by night-vision pathways.

Paul R. Martin

The eye feeds the brain with information about the visual world, the information all being transmitted via the optic nerve. Optic nerve fibres — in humans there are about one million for each eye — are the extended processes of nerve cells called retinal ganglion cells. As their name suggests, these cells are housed in the retina, which lines the back of the eye and contains the nerve circuits that process signals from the light-sensing rod and cone photoreceptor cells. Because all visual sensations rely on ganglion cell activity, we need to understand how, why, and when ganglion cells respond to light. In a new study reported in this issue of *Current Biology*, Ala-Laurila and Rieke [1] measured responses of ganglion cells at the sensitivity limit of night-time (scotopic) vision. Their results may help us understand how we can find our way around in starlight. More generally, they suggest a clever strategy of nerve signal processing: the eye sends high-gain and low-gain signals in parallel to the brain. But first, a little history.

Anatomy Is Destiny

In 1935, Stephen Polyak, a Croatian-born Professor of Anatomy at the University of Chicago, was deeply engaged in his now-classic anatomical studies of the retina of humans and other primates [2]. He discovered a conspicuous cell type with signal-collecting processes (dendrites) that looked like "an open Chinese umbrella or parasol". He described two subtypes of parasol cells that send their dendrites to distinct sublayers of the retina. Polyak also showed that the dendrites of parasol cells are small in extent near the centre of the retina and are larger toward the retinal edges. These observations turned out to have key functional correlates for vision [3,4]: the parasol cell's sublevel of dendrite stratification sets its response to light onset (On parasol) or light offset (Off parasol), and the dendritic field size determines the spatial resolving power of the cell.

The intervening years have taught us that parasol cells are a crucial part of the visual system. They are essential for motion perception, and they make big contributions to form vision under

low image contrast, for example on a foggy day, and in low light conditions, as on a starlit night [5]. But whether and how parasol cells transmit the tiny intensity changes that humans can detect using their night-time vision has been unclear. Now, nearly eighty years after the first description of parasol cells, Ala-Laurila and Rieke [1] have made the requisite measurements: they deliver a fascinating "yes and no" answer that opens a new way of thinking about how the eye sends messages to the brain.

Dark Side of Vision Research
To a non-scientist, studying vision when it so dark that you can hardly see might seem strange. But a lot can be learned about the visual system from experiments carried out in near-darkness. Here's how to do it. First, you plunge your experiment into utter darkness for about an hour so that the retina can fully charge its biochemical batteries for photon absorption. Next, you briefly ignite a very dim light source and spray a tiny dose of photons toward the retina. Then you observe the response of your human or animal subject — or, like Ala-Laurila and Rieke [1], observe activity of a nerve cell in an excised retina preparation. The final, hair-raising part of the act requires you to steep the results in an exotic brew of equations, corrections, estimates and probabilities, linking them back to the quantum distribution and probability of absorption of the photons that you think you should have sprayed in the first place. Phew!

It seems surprising that such complex experiments work at all, but they have produced broadly consistent results, across many vertebrate species and diverse experimental conditions. It turns out that a dark-adapted retina is a most efficient photon-catching device, where signals derived from absorption of just a few photons can percolate across a chain of nerve cell connections in the retina (Figure 1), cause changes in activity of retinal ganglion cells, and create the perception of a just-detectable flash for human observers [6–8].

In an impressive technical achievement, Ala-Laurila and Rieke [1] adapted an *in vitro* preparation of macaque retina [9] to preserve normal rod photoreceptor connections and function, and measured ganglion cell activity — action potential or 'spike'
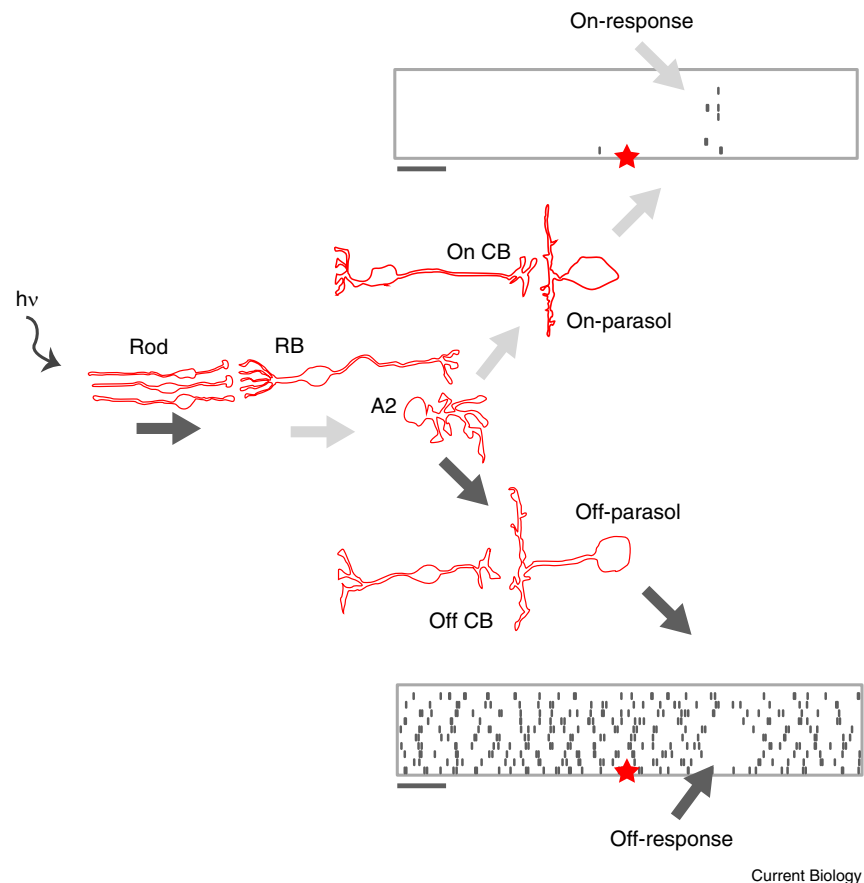
Figure 1. Pathway from photon absorption to parasol cell response.
Photon absorption by rod photoreceptors (Rod) causes a brief pause in neurotransmitter release (dark arrow) at the connection to rod bipolar interneurons (RB). Low transmitter levels activate the RB (light arrow), which in turn activates a second interneurone (amacrine cell type A2). The A2 suppresses Off cone bipolar cells (CB) but activates On cone bipolar cells, thus generating complementary patterns of spike activity in On parasol and Off parasol ganglion cells. Each row of tick marks in the box inserts shows spike responses to a single presentation of a weak flash delivered at the time indicated by the star symbol. Each tick mark shows time of a single spike. The On parasol response is weak, comprising two or fewer spikes for each flash. The Off parasol response comprises a more robust and long-lived pause in the ongoing spike activity. Box plots adapted from Figure 1 in [1]. Time scale bar = 100 ms.

discharge — right at the sensitivity limit of scotopic vision. Parasol cells are the likely messengers of weak night-vision signals because they have large and dense signal-collecting area (remember Polyak!), receive strong functional rod input [10], and provide input to brain pathways that detect low-contrast stimuli [5].

Eye Gives Brain a Choice of Gain
Ala-Laurila and Rieke [1] measured responses of On parasol cells and Off parasol cells to flashes of light delivered in darkness or on top of weak background lights. The responses of Off parasol cells conformed to expectation from studies of cat retina [11]: the cell spits out a random stream of spikes which is interrupted by even

the weakest flashes. Most of the gaps in the spike stream can be attributed to single-photon absorptions (Figure 1). But the responses of On parasol cells gave a big surprise. Spikes were only emitted by On parasol cells following flashes that caused simultaneous absorption of multiple photons across the (several thousand) rods providing inputs to the cell [1,12]. The On parasol cell seems prepared to miss out on many single-photon absorptions, in favour of reliably detecting a stronger signal that there is a light out there in the world.

By varying flash and background strength, and subjecting the cells' responses to signal-detection analysis, Ala-Laurila and Rieke [1] gained more evidence that Off parasol and On

parasol cells treat very weak signals quite differently. In signal-detection parlance, the criterion point of On parasol cells is set high, to avoid false positives — that is, to avoid spurious reporting of noise as true photon absorption — whereas Off parasol cells provide a noisy, but high-sensitivity record of activity in their inputs. Intriguingly, the noise-rejecting step appears to operate at the final stage of the nerve pathway from rods to On parasol cells. It is manifest only close to the detection limit, with On parasol cells delivering a more faithful readout of the number of absorbed photons under brighter conditions.

Previous studies of mouse retina showed that otherwise symmetric temporal properties of On and Off cells breaks down at scotopic levels [13], but the relation of this phenomenon to the gain asymmetry across On parasol and Off parasol cells is not yet clear. There also appears to be a difference between monkey On parasol cells and the 'On beta' cells in cat retina, which show strong maintained activity in the dark [11]. Details of the physiological links between these observations will be important for future studies.

A key message from Ala-Laurila and Rieke's [1] paper is that the eye at night may not be working uniformly as a high-gain photon detector. It delivers a conservative estimate of light increments in the visual field, in parallel with a high-gain but noisy estimate of light decrements. When allowed to report or implicitly vary their confidence level, human test subjects will happily trade-off sensitivity for reliability in visual detection tasks, [6,7,14]. How the newly-discovered asymmetry at low light levels could influence night-time visual performance is not yet known and is a fascinating question for night-active as well as day-active researchers to ponder.

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Climate Change: Many Ways to Beat the Heat for Reef Corals

Reef-building corals are vulnerable to heat stress and are facing widespread losses due to climate change. A new study shows that coral heat tolerance can result from selection on a suite of genes to maintain genetic flexibility.

Andrew C. Baker

Coral reef ecosystems are vanishing. Many reefs worldwide have lost over 50% of their coral cover over the last 30–40 years, and some have lost more than 90% [1–3]. The principal factor behind coral mortality is climate change-induced heat stress, as well as disease, which often acts as the *de facto* executioner of corals weakened by thermal stress [4]. These impacts are often compounded by nutrient pollution and the loss of herbivorous fish and urchins, which promote the growth of competing macroalgae, as well as ocean acidification, which slows coral regeneration [1,5]. In light of these alarming declines, the question of whether corals can adapt to warming oceans has sparked considerable interest, but the consensus opinion has been that corals have relatively long generation times and show low genetic diversity, which hampers their ability to evolve quickly enough to respond to the warming rate of 2°C or more that is forecast for this century [5]. However, in a paper in this issue of *Current Biology*, Bay and Palumbi [6] demonstrate that the genetic basis for heat tolerance in corals may lie in maintaining a diverse set of alternative alleles across multiple loci, suggesting that coral populations with these characteristics may be better able to persist into the future. In combination with other recent studies from the same lagoon in American Samoa [7,8], these findings reveal how multiple genetic threads are woven together to build a more heat-tolerant coral, and suggest that some corals may be able to respond to warmer conditions much more quickly than was previously assumed.

Like trees in a tropical rainforest, corals are critical ecosystem engineers that build the reef habitat on which many other marine species depend. This is achieved by a solar-powered mutualism between the coral animal and a diverse set of unicellular dinoflagellate algal partners in the genus *Symbiodinium* (as well as a variety of other microbial mutualists and viral associates whose metagenome constitutes the coral 'holobiont' [9,10]). However, this very successful partnership also represents an Achilles' heel: exposure to unusually high temperatures results in coral 'bleaching' — eviction of algal