attention: Op18/stathmin; and the small GTPase Ran. Phosphostate gradients in Op18/stathmin, which destabilizes microtubules at least partly by tubulin sequestration in a phosphorylation-dependent manner [11–13], have been directly visualized in mitotic and interphase cells using phosphostate-specific fluorescence resonance energy transfer (FRET) probes [14]. The FRET analysis of Niethammer *et al*. [14] indicates that a region favoring microtubule assembly exists between the poles in mitotic cells. Phosphostate gradients in Ran, which indirectly controls microtubule assembly in mitosis via TPX2 in a GTP/GDP-state dependent manner [15,16], have been directly visualized via FRET in *Xenopus* egg extracts [17]. Consistent with these findings, Wollman *et al*. [1] show that perturbing Ran function extends the capture time 2–3 fold, suggesting that a high concentration of Ran-GTP around the chromosomes at least partially creates the favorable growth region for microtubule assembly.

Previous computer modeling of RanGTP gradients suggested that the gradients would be very small in somatic cell mitosis [18]. It may be, however, that even weak gradients could translate quantitatively into relatively large catastrophe and rescue frequency gradients in the cell. In addition to controlling catastrophe and rescue, Ran-GTP may also control microtubule nucleation around the chromosomes [16]. In general, it is not yet clear how any putative molecular gradient is actually 'read out' by microtubules to control their behavior spatially. Further, microtubules themselves may possess a history-dependent catastrophe that enables persistent assembly during the early part of a growth phase, with an increasing likelihood of catastrophe as elongation proceeds [6,19]. This has the effect of increasing the efficiency with which space is searched by narrowing the distribution of microtubule lengths around a length optimized for the search-and-capture process [20].

In summary, it has been appreciated for some time that chemical gradients shape the developing embryo. Evidence is accruing that they also shape the cytoplasm, which may facilitate formation of key intracellular connections, such as those between spindle poles and kinetochores during mitosis.

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Lightness Perception: Seeing One Color through Another

A newly described and dramatic visual illusion suggests that the retinal image is decomposed by the brain into overlapping layers, not into contiguous frameworks of illumination.

Alan L. Gilchrist

Incredible as it may seem, the upper chess pieces in Figure 1 are identical to the lower chess pieces. The upper pieces appear to be white pieces with black clouds in front of them. The lower pieces appear to be black with white clouds. This image, adapted from the recent paper by Anderson and Winawer [1], provides dramatic evidence of the ability of the human visual system to parse the retinal image into separate layers.

Ever since Johannes Kepler discovered that an image of whatever we look at is projected

onto the rear inner surface of the eye, it has been natural to assume that the rods and cones function much as modern day photocells, reporting the point-by-point intensity of light in the image.

But this simple notion immediately runs into trouble. There is absolutely no correlation between intensity in the image and perceived gray level — called lightness — of the surfaces we see [2]. For example, a black surface in the sunlight can easily reflect more light to the eye than a nearby white surface in shadow. And yet the white and black surfaces are perceived correctly.

Some have tried to salvage the simple pointwise encoding scheme by invoking neural inhibitory influences among neighboring receptor cells [3,4]. Stimulation of the retina by light not only produces neural activity at that location, but it also inhibits neural activity in the immediately surrounding region. Thus, although a black paper in sunlight and a white paper in shadow may evoke equal stimulation, the neural activity corresponding to the black paper in sunlight is strongly inhibited due to the bright surrounding context.

But there is more to the problem. We also perceive the level of illumination itself. Thus, as with the chess pieces seen through clouds, we perceive at least two separate values at each single point in the image: the lightness of the surface itself, and the brightness of its illumination.

Most lightness theorists have now accepted the concept, originally proposed by the Gestalt theorists of the early 20th century, that the retinal image is decomposed by the brain into separate components [5]. But there are two competing decomposition schemes: frameworks and layers. According to the frameworks approach, the image is divided into contiguous regions of illumination or shadow, like states on a map. Within each framework the highest intensity serves as the standard, or anchor, for white. The lightness of other surfaces within the framework is determined relative to this standard.

According to a different method of decomposition inspired by the computer revolution, the retinal image is decomposed into overlapping layers [6–8]. In effect the image is treated as a pattern of illumination projected onto a pattern of surface grays. This scheme is attractive. It comfortably accommodates the fact that we can report both the shade of surface gray and the level of illumination at each location in the visual field. It also accounts for the appearance of the chess pieces.

The layers concept exemplifies a computational strategy known as inverse optics [9]. The intensity at each point in the image is the product of a combination of factors: the proportion of light reflected by the surface at that location (called reflectance), the intensity of illumination incident on that surface, and certain properties of the intervening media, such as those of fog or filters. By the laws of optics these factors become entangled in the image. In principle they can be disentangled by hypothetical brain processes that are inversely related to the optics of entanglement.

For example, a red book on the dashboard of your car casts a red reflection in the windshield. Through the reflection you perceive distant objects, including green grass, in their normal colors. Light from the green grass and the red reflection physically mix to produce yellow. The yellow is observed when seen through a small hole punched in a piece of cardboard held up so it blocks out the surrounding context. Without the cardboard, however, no yellow is seen, only the red and green layers. The brain is thought to split the yellow light into the red and green layers using rules that invert the usual rules of color mixing. This is called scission.

Or consider the image of a white house reflected in the shiny surface of a black car. Neither the house nor the car appears gray where their images overlap. Rather the light at that location is perceptually split into a white and a black layer.

Strictly speaking, the illumination that falls on surfaces is not a separate layer. But the same scission algorithms that work for transparent layers can be effectively applied to the illumination. Mathematically a shadow and a sunglass lens have the same effect on the image [10].

When the processes of image formation are inverted in this way, surface reflectance is not merely computed, it is recovered. Sounds good, but it may be too good. We do not perceive gray shades with

Figure 1. The new illusion.

The upper and lower sets of chess pieces are identical. But mathematical relationships at their boundaries cause them to be differently segmented into objects and clouds.

complete accuracy. A gray object in shadow appears slightly darker than it would appear in sunlight. A gray paper looks lighter on a black background than on a white background. Many of these errors are captured in delightful illusions. These errors must come from the visual system itself. And these errors are systematic, not random. They constitute a sort of signature of the visual software employed by the brain [11]. Thus the overall pattern of lightness errors shown by humans provides a powerful constraint on theories of lightness.

Inverse optics models are great for computing gray shades correctly. But what about the errors? In principle, the errors could be accounted for by partial failures in the scission process. But such efforts to model the errors [12,13] have not proven very effective.

For this reason, several theorists have resurrected the older frameworks concept in a modified form that can explain the errors [14,15]. Combining the concept of frameworks with a process of crosstalk between frameworks, seems to provide an impressive account of lightness errors. Anderson and Winawer [1] acknowledge these claims of the frameworks approach. And yet, their chess-piece demonstration offers compelling evidence of perceptual scission.

Layer proponents, like Anderson and Winawer [1], argue that failures in the scission process could potentially account for the errors pattern. Likewise framework proponents suggest that framework-based models could potentially be expanded to include the perception of illumination. Both sides are open to an integration of the two approaches. Stay tuned.

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Peripheral Glia: Schwann Cells in Motion

Neuregulin signaling through ErbB receptors is known to play an essential role in Schwann cell proliferation, survival and myelination. Recent studies in zebrafish provide a peek at living Schwann cells migrating along axons *in vivo* and suggest that ErbB signaling, while not required for cell movement per se, is required to maintain the directed migration of these cells.

Cary Lai

Schwann cells —the glial cells that wrap peripheral axons— arise from trunk neural crest cells that find their way to the emerging axons of sensory and motor neurons. These progenitors are then thought to migrate along the outgrowing axons and to proliferate in order to produce a sufficient number of cells for the myelination of the axons. The number of these pre-myelinating Schwann cells is believed to be regulated by axon-derived survival signals. Each Schwann cell typically first envelops multiple axonal segments but ultimately surrounds a segment of a single axon [1]. Only large axons (diameter > 1 μm) are myelinated while smaller axons are not

Neuregulin as a Regulator of Schwann Cell Number and Myelin Thickness

The growth factor neuregulin-1 plays a pervasive role in the life of a Schwann cell [2]. Neuregulin-1 was identified over 25 years ago as two distinct biological activities. It was first described as glial growth factor [3], for its ability to serve as a potent Schwann cell mitogen; separately, it was shown to regulate acetylcholine receptors in muscle cells *in vitro* and described as the acetylcholine receptor-inducing activity (ARIA) [4]. It was also recognized that axonal membranes contained a substance, now known to be neuregulin-1, that promoted Schwann cell proliferation [5]. Subsequently, neuregulin-1 was recognized for its ability to support Schwann cell survival *in vitro* [6,7], a finding that suggested it may also function to regulate the number of pre-myelinating glia by serving as a limiting survival factor.

The primary receptor for neuregulin-1 in Schwann cells is a