Coding of color and form in the geniculostriate visual pathway (invited review)

Peter Lennie and J. Anthony Movshon

Center for Neural Science, New York University, New York, New York 10003

Received June 2, 2005; accepted June 2, 2005

We review how neurons in the principal pathway connecting the retina to the visual cortex represent information about the chromatic and spatial characteristics of the retinal image. Our examination focuses particularly on individual neurons: what are their visual properties, how might these properties arise, what do these properties tell us about visual signal transformations, and how might these properties be expressed in perception? Our discussion is inclined toward studies on old-world monkeys and where possible emphasizes quantitative work that has led to or illuminates models of visual signal processing. © 2005 Optical Society of America

OCIS codes: 330.1720, 330.1800, 330.4060, 330.5380, 330.6110, 330.7310.

1. INTRODUCTION

This issue of the Journal of the Optical Society of America A honors the contributions to visual science of Russell De Valois (1926–2003), and this paper reviews our current knowledge in the arena with which Russ was most involved, the neural coding of color and form in the geniculostriate pathways of the primate visual system. Our goal is not to focus on Russ's work alone, though we will of course consider many of his contributions, but instead to place his work in the broader context of what we do and do not now know about the neural foundations of visual perception. Our review is not exhaustive. It concentrates on the function and organization of the primate geniculostriate visual pathway and emphasizes two themes that were of particular interest to Russ: quantitative approaches to the study of neuronal function (with emphasis on the activity of individual neurons) and the relation of neuronal function to perception.

When Russ began his neurophysiological studies of primate vision in the 1950s, little of consequence was known about the central neural representation of the information in the visual image. The most important technique for studying this representation—single-cell recording in the central nervous system—was in its infancy, and Russ embraced it and became one of its pioneers. Trained as a psychologist, Russ drew his inspiration directly from visual psychophysics. In this respect he was out of the mainstream of the American tradition of visual neurophysiology, which tended to view psychophysical studies with a curious combination of skepticism and disdain (see, for example, Hubel[1]). The theme of linking physiology and psychophysics, however, had a powerful impact in other senses (for example, Mountcastle[2] and Kiang[3]) and formed a key element of the British tradition in visual science (e.g., Brindley[4]).

In a research career spanning half a century, Russ made enduring contributions in two distinct areas of neurophysiology: color vision and spatial vision. Two quite different histories shape these fields and Russ's engagement with them. By the 1950s psychophysics had constructed a powerful framework for steering physiological investigations (see, for example, Brindley[5]), most sharply at the level of fundamental photoreceptor mechanisms (which in primates were to remain largely inaccessible until the 1980s) but also at the level of postreceptoral mechanisms, on which Russ concentrated during the first years of his career. In spatial vision, and particularly the representation of form, psychophysics in the 1950s offered far less guidance. Mechanistic accounts of spatial vision were few and inadequate, and it took the demonstration of form-selective responses in visual neurons by Hubel and Wiesel[6–8] to inspire psychologists to develop mechanistic theories of pattern vision. It was only in the 1960s that the concept of parallel visual spatial channels pioneered by Robson and Campbell (see Graham[9]) began to gain wide acceptance and not until the 1970s that neurophysiological work, much by Russ and his colleagues, could be founded on a quantitatively-defined and psychophysically-based theory of spatial vision.

The scope of our review is confined to the pathway from retina through lateral geniculate nucleus (LGN) to striate cortex. In primates this is the route by which almost all visual information reaches the cerebral cortex. Other pathways convey signals to cortex without involving the LGN (for example, the superior colliculus[10]) or project from LGN to extrastriate cortex,[11–14] but these are numerically insignificant and in most cases probably incapable of activating cortex in the absence of the geniculostriate pathway.[15–18] Although a great deal is now known about extrastriate visual pathways, it is dwarfed by what we know about striate cortex, where the behavior of neurons amply illustrates many fundamental principles of visual coding. Striate cortex is also, not coincidentally, the part of the visual system on which Russ spent the most substantial part of his career.

Respecting both the chronology of the fields and the chronology and scope of Russ's contributions, we deal first with the question of how color is coded and represented up to striate cortex, then with form. Because we are ultimately interested in human vision, we confine our discus-

1084-7529/05/102013-21/$15.00

sion as much as possible to work in primates. This is especially so for color vision, where only the monkey provides an adequate model. But when we come to consider the details of cortical circuits, especially measured intracellularly, much data will be from cat, since there are few intracellular data available from primates.

2. COLOR CODING

Color vision has an undeserved reputation—both for being well understood and for being difficult to understand. The fundamentals of trichromacy and their expression in color matching have been understood since the nineteenth century and have been progressively exploited in color rendering since.[19] Very important advances were made before we knew anything about the physiological machinery of color vision, and the fundamental mechanisms have historically been characterized in abstract (though mathematically tractable) forms that are hard to connect with real visual machinery.[20] When Russ began his work there was universal agreement that color vision depended on three fundamental mechanisms, but we had little idea of their spectral sensitivities, and it was unclear whether they existed as three classes of cone photoreceptors.[5] Moreover, the existence of color-opponent mechanisms was still uncertain.[21] The characterization of color-opponent neurons in the monkey's LGN[22–24] altered the landscape profoundly, not just by establishing the nature of a post receptoral stage of analysis but also by making physiology relevant to a domain that had been *the* exemplary testimonial to the power of psychophysics. In the years since then, physiological work has become increasingly important, partly because it provides a vehicle for testing ideas about mechanism suggested by psychophysics, but more because it reveals the workings of machinery that is often inaccessible to psychophysics.

Some of the questions that preoccupied color scientists in the late 1950s are now substantially answered; others remain and have been joined by new ones. In what follows we review the substantial progress that has been made in understanding the physiology of color vision, highlighting particularly the organization that would have been concealed from psychophysical exploration.

A. Cone Signals and Spectral Sensitivity

In 1960 we knew neither the embodiment of the three fundamental mechanisms of color vision nor their spectral sensitivities. It was considered likely that the three fundamental mechanisms were photopigments, but it was unclear whether these were uniquely associated with distinct classes of photoreceptors.[5] Even ten years later Brindley[4] was unconvinced that physiological work would be illuminating: "The observed properties of single cells do not yet help us distinguish between … forms of the three-channel hypothesis … If more fully investigated they might so help, but I suspect that such an investigation would be very laborious and only slightly rewarding." Work on photoreceptors has in fact been spectacularly rewarding. Microspectrophotometry on primate cones established firmly that there were three types containing three different pigments (e.g., Ref. 25), and recordings of the light-evoked responses in individual cones not only demonstrated their univariance but also characterized their spectral sensitivities with remarkable precision,[26] leading to a satisfying agreement with estimates arrived at from psychophysics.[27] We have also attained, in a surprisingly short time, a substantial understanding of the variation among cone photopigments and its genetic control.[28]

Physiological work has also substantially answered another long-standing question about the organization of the early stages of color vision: Where is the site of the light adaptation that psychophysical work[29] has shown occurs substantially independently in the three fundamental mechanisms? We now know that relatively little of this occurs in the cones themselves,[30] but much of it (though not all) is expressed in recordings from horizontal cells, implicating the synaptic connections that cones make with horizontal cells.[31]

B. Second-Stage Mechanisms in Retina and LGN

Early physiological work on opponent mechanisms[22–24] focused on the LGN, which is in many respects easier to record from than retina, though it was soon clear[32] that the properties of neurons in LGN simply mirrored those of retinal ganglion cells. (The properties of ganglion cells in turn probably reflect those already present in midget bipolar cells, making bipolar cells the likely site of origin of color opponency.[33]) This work established the existence of two broad classes of color-opponent neurons, tuned to red–green and blue–yellow variations, respectively, and a third class of neuron that had spectrally broadband tuning, corresponding approximately to the luminosity function, V_λ. It offered powerful confirmation of an idea that had been hard to establish psychophysically,[21] and it revealed[23] some striking parallels between the behavior of neurons and the behavior of psychophysical observers. Although color-opponent neurons fell into two dominant clusters that had distinctively different chromatic signatures, these were not sharply segregated in the early work. The later introduction of methods that characterized the responses to small signals—modulations of the visual stimulus about a constant mean luminance and chromaticity[34]—coupled with our modern knowledge of the cone fundamentals[27] made it easy to demonstrate that the chromatically opponent neurons fell into discrete groups, one that receives inputs from only L and M cones and another that receives strong inputs from S cones.[34] Until recently, both types of chromatically opponent neurons were associated with the pathway that originates in midget ganglion cells and projects to cortex through the parvocellular (P) layers of the LGN; the broadband neurons were associated with the pathway that originates in parasol ganglion cells and projects to cortex via the magnocellular (M) layers of the LGN. It is now clear that neurons that receive strong inputs from S cones are not P cells but are neurochemically[35] and anatomically[36] distinct and form pathways that project distinctively to LGN[37] and cortex.[38]

The spectral characteristics of the three postreceptoral mechanisms are close to what would be required for an optimally efficient representation of the chromatic statistics of natural scenes.[39–41] The apparently neat alignment

of these mechanisms with three postulated on psychophysical grounds belies a physiological organization that is surprisingly complex and largely invisible to psychophysics because much of it involves linear mechanisms.

1. Achromatic Pathway

One puzzle is the substrate of the "achromatic" visual channel that is responsible for high spatial resolution. This is presumed to have the V_λ spectral sensitivity and therefore driven by inputs from L and M cones only. Although individual M cells can resolve spatial position with high precision,[42] the low density with which the mosaic of them samples the image (perhaps one ninth the density of the P cells[43]) makes it unlikely that they constitute the mechanism for resolving spatial detail. Sampling aside, the broadband spectral sensitivity of the M pathway has implicated it as the substrate of V_λ.[44] There is a variety of reasons to suppose that this is unlikely,[45] and doubts are reinforced by the recent observation that M cells often receive signals from S cones.[46,47] The more likely substrate of V_λ is the P pathway, which we know receives inputs from only L and M cones. Receptive fields of P cells have a center–surround organization, with different spectral sensitivities in center and surround. As a result, a neuron's chromatic signature depends on the spatial configuration of the visual stimulus used to drive it: When excited by stimuli containing high spatial frequencies, P cells respond well to achromatic patterns, but when excited by stimuli containing low spatial frequencies they respond best to chromatic patterns.[48] Information about spatial detail is thus encoded jointly with information about the red–green dimension of color variation. The signal conveyed by any individual neuron is ambiguous but can be disambiguated by analyzing the signals from several,[49,50] because P cells behave linearly and occur in four variant forms whose receptive fields have complementary distributions and signs of L- and M-cone inputs to center and surround.

2. S-cone Pathways

Like P cells, neurons that receive strong S-cone input exist in complementary forms, with S cones providing either excitatory or inhibitory drive. However, these are plainly not symmetrical. "Blue-on" (S+) cells, which are relatively frequently encountered, occur in at least two morphologically distinct forms[36,51]; "blue-off" (S−) cells, which are seldom encountered, have been identified with at least one other morphological type.[36,52] The on and off types also differ in their sensitivities and chromatic signatures: Blue-on cells are sensitive and receive S-cone signals opposed to inputs from L+M cones; blue-off cells are appreciably less sensitive, and many receive S+M cone inputs opposed to inputs from L cones.[47] Finally, the on and off types project to different layers in V1.[38] The chromatic signature of the blue-on type matches that inferred from some (e.g., Krauskopf *et al.*[53]) but not most (e.g., Refs. 21,54,55) psychophysical work; the chromatic signature of the blue-off type is unlike that generally inferred from psychophysical work, though it was anticipated by De Valois and De Valois.[50]

3. Receptive Field Organization in P Cells

Another complexity has been thrown into relief by the increasingly detailed information now becoming available about the numbers and arrangement of the cones of different types. S cones are detectable histochemically, and their number (about 8% of all cones) and arrangement in the retina (absent from the central fovea and random or nearly so elsewhere) have been known for some time.[56] No similar methods have yet distinguished L and M cones, although long-established psychophysical evidence, more recently corroborated by genetic analyses, points to an L:M cone ratio of near 2:1 on average. Recent work based on very-high-resolution imaging of the mosaic of cones in the living human eye[57] has shown that not only is the arrangement of L and M cones irregular (apparently random) but also that the ratio of L:M cones varies by at least a factor of 4 among individuals, without effect on their color vision.[58] These findings raise interesting questions about the organization of postreceptoral mechanisms: in a random or nearly random mosaic, clusters of cones of a single type will be common, and in a mosaic in which one cone type predominates there will be substantial regions in which no cones of the other type are present. This makes it hard to construct small receptive fields in which both the spatial and the chromatic properties are controlled: fine control of spatial sampling requires the cell to weight cone inputs by their positions in the receptive field; fine control of chromatic sampling requires the cell to weight cone inputs by their types. Control of one property is purchased at the expense of the other (Fig. 1). This problem—compounded by the general imbalance in the numbers of L and M cones—might be circumvented by constructing two pathways, one specialized for spatial sampling and indifferent to cone type, the other specialized for chromatic sampling and indifferent to cone position. Rodieck[59] (see also Calkins and Sterling[60]) suggested that only a subset of P cells might be relevant to color vision. Wiesel and Hubel[24] had distinguished two kinds of receptive field among parvocellular neurons: In type I, color-opponent mechanisms were segregated in center and surround; in type II, they overlapped fully. Rodieck suggested that color vision depended on type II cells, but there is no evidence that they constitute a distinct group among L–M opponent neurons (Wiesel and Hubel's type II cells were overwhelmingly those receiving strong S-cone input, which we now know are not P cells).

A good deal of evidence suggests that the organization of the P pathway favors spatial vision over color vision. First, the number of P cells far exceeds what would be required to support the modest spatiotemporal requirements of color vision. Second, the L- and M-cone types, reliably distinct only in old-world primates, are probably a recent evolutionary development,[61] so color opponency is likely to have been layered on top of the machinery of spatial vision. It is clear that color opponency can arise without distinguishing L and M cones. In and near the fovea a P cell's receptive field receives its dominant center input from a single cone, so if the surround draws indiscriminately on both cone types the receptive field will be color opponent. Lack of cone specificity in the surround need have little effect on the strength of opponency.[62] As recep-

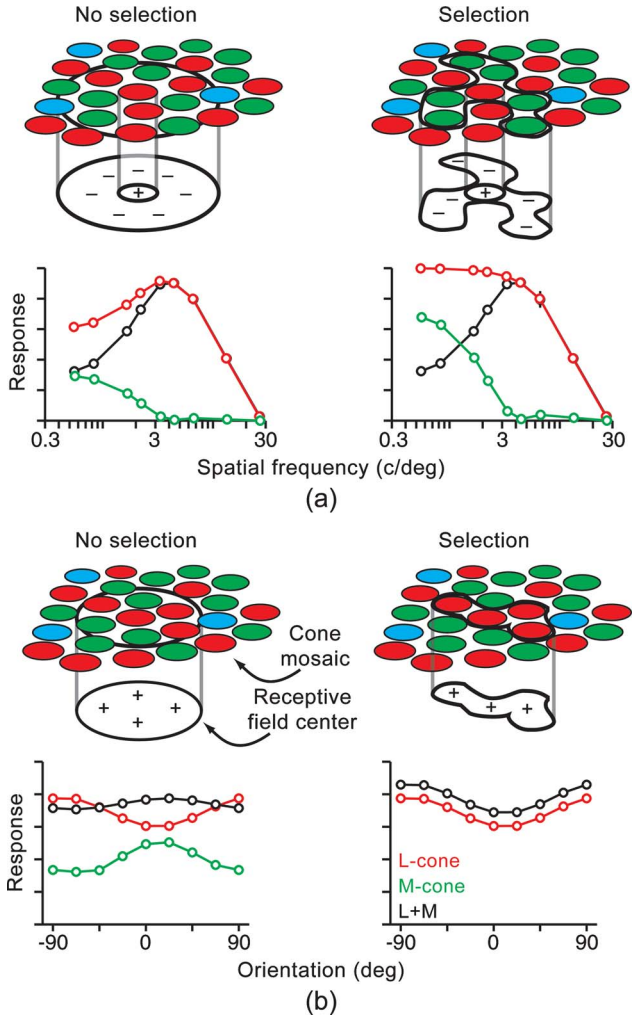

Fig. 1. Hypothetical receptive fields of P cells, illustrating the potential consequences of drawing indiscriminately or selectively on inputs from different classes of cones. (a) Receptive fields near the fovea, where the center input arises predominantly from a single cone. Without selection of cone inputs to the surround (left) the spatial frequency selectivity of the neuron will be band pass when measured with achromatic stimuli or stimuli that isolate the center cone type (in this case L) but low pass when measured with stimuli that isolate the other type (M). With selection of cone inputs to the surround (right), spatial frequency tuning will be low pass when measured with a cone-isolating grating of either type. (b) Receptive field in near periphery (ca. 10°), where the center receives input from a small number of cones. With or without selection of cone type (left), orientation selectivity measured with a grating of preferred spatial frequency will vary with the cone type that is isolated. With selection of cone type in the center (right), orientation selectivity will be independent of grating chromaticity. (Courtesy of S. Solomon.)

tive fields become more eccentric, with larger centers that draw on more cones, one would expect the color-opponent organization to become more variable and weaker on average but certainly not to disappear, given the occurrence of clusters of cones of a single type.[57,63] This is what happens,[64,65] but to tell whether it occurs by chance or by design will require a quantitative model of the consequences of clustering.

In central retina, where receptive fields are small, it has been especially hard to establish how L and M cones are distributed within them. Reid and Shapley[66] characterized receptive fields with binary modulation of pixels in a two-dimensional array. For P cells, maps made with L- and M-isolating stimuli were almost mirror images—surprising for a receptive field with center–surround organization. The cone specificity of the surround is also surprising, in view of evidence that its precursor is driven by both L and M cones: The principal drive to a P cell is the midget bipolar cell, whose receptive field has a surround formed by the H1 horizontal cell, which receives mixed L- and M-cone input[67] in proportions that locally match those found in ganglion cells.[64] Moreover, measurements of the spatial frequency tuning of P cells made with cone-isolating gratings[68] show that sensitivity to cone-isolating stimuli falls at low spatial frequencies in many neurons (notably those in which the center of the receptive field is dominated by L cones), implying that the same cone type exists in both center and surround. A firm answer to the question of whether P cells draw selectively on L and M cones might be obtained by exploiting the irregularity in their mosaics: in near periphery, where just a few cones drive the center of the receptive field, the particulars of their positions should make a cell orientation selective for gratings of near-optimal spatial frequency. The preferred orientation should vary with stimulus chromaticity if the center receives inputs from more than one kind of cone (Fig. 1).

C. Cortical Color Coding

Because so much psychophysical work on color vision has been focused on mechanisms that sit early in the visual pathway—the nature and spectral sensitivities of the three receptors and the character of opponent processes—and because only linear transformations are needed to convert the signals presented by ganglion cells (and LGN neurons as their surrogates) into a form compatible with psychophysics, one might suppose that cortex adds only modest refinements to the earlier analysis. This is clearly not the case.

1. Chromatic Preferences of Cells

Hubel and Wiesel,[8] and many others since, found that V1 contains few neurons that are obviously color opponent. This is surprising in view of the preponderance of such cells in LGN. Most simple and complex cells have spatially well-tuned receptive fields and respond best to achromatic patterns. The cells most sensitive to chromatic modulation (perhaps 10% of those encountered) generally respond poorly to achromatic stimulation, lack orientation selectivity, and have low-pass spatial frequency tuning, implying that the chromatically opponent mechanisms are coextensive.[8,69,70] These appear equipped to provide a signal about chromaticity that is indifferent to the spatial properties of the visual stimulus and thus a substrate for the chromatic mechanisms inferred from psychophysics. The trouble is that the neurons do not fall neatly into two ("red–green" and "blue–yellow") clusters.[69,71]

An influential modern psychophysical account[53] of the "cardinal" chromatic mechanisms (one most sensitive to pure L–M cone modulation; the other most sensitive to

pure S-cone modulation) exposed them through adaptation to chromatic modulation, to which P cells in LGN are not susceptible. These mechanisms evidently must lie in cortex, where many neurons adapt, yet the chromatic preferences of cells studied in V1 favor the cardinal directions less well than they do in LGN.[69,71–73] Lennie *et al.*[69] thought that the canonical mechanisms might emerge in extrastriate cortex, but subsequent work[74–76] found no indication of this. Moreover, recent work using MR imaging[77] makes clear that chromatically selective adaptation occurs somewhere in V1. The enigma is perhaps resolved by the discovery[78] that adaptation to chromatic modulation deforms tuning curves to expose in almost all V1 neurons two underlying mechanisms tuned to the cardinal directions. These otherwise hidden red–green and blue–yellow mechanisms might lie at the geniculocortical synapse or in the input layers of cortex, among neurons that are seldom caught with an electrode. Such an early locus, close to LGN inputs, probably explains why strong chromatic responses are prominent in work using evoked potentials[79] and MR imaging[80] despite the paucity of color-preferring cells. Because the red–green and blue–yellow mechanisms are susceptible to adaptation and are therefore readily accessible to psychophysical investigation, they enjoy a prominence that almost certainly exceeds their overall importance in representing color information. To the extent that physiological work captures representative samples of neurons, it is clear that red–green and blue–yellow mechanisms are most sharply articulated at the input to cortex and thereafter become increasingly obscured. This diffusion of the machinery of color vision is not easily captured in psychophysical work, although some clearly points to the existence of adaptable mechanisms at a level beyond the red–green and blue–yellow ones.[81,82]

The dispersion of chromatic preferences in cortex requires no more than linear combination of signals from underlying red–green and blue–yellow mechanisms. Signal combination might well be linear, but important nonlinearities are evident in the responses of cortical cells, and these have interesting effects on chromatic selectivity/tuning. We have already noted that contrast adaptation can deform a neuron's tuning curve. This occurs at multiple levels in V1, some early, in the underlying red–green and blue–yellow mechanisms, but some almost certainly at later stages after signals from these have been combined (this is also true of spatial adaptation, as we later discuss under Subsection 3.B.3, Contrast Adaptation). Other, fast-acting, nonlinearities alter tuning in interesting ways. In most neurons, an accelerating output nonlinearity that probably reflects the interplay of a threshold and noise (see Subsection 3.B.6, Threshold Nonlinearity) selectively enhances responses to the most effective stimuli, making chromatic selectivity contrast dependent and often sharper than is found in LGN.[71,83] A second, pervasive, nonlinearity (see later discussion in Subsection 3.B.5, Contrast Gain Control) also has substantial and sometimes complex effects on chromatic tuning.[47] One expression of it among neurons that respond to both achromatic and chromatic stimuli is that responses to achromatic stimuli saturate sooner than do responses to chromatic stimuli (notably those modulating S-cone input). The upshot is that chromatic opponency (especially when it involves S cones) is more evident at high contrast.

Relatively little work has been done on the dynamics of chromatic tuning, although what there is points to relatively sluggish[84] and nonlinear[85] behavior of neurons that receive substantial input from S cones.

2. Encoding the Spatial Structure of Color Signals

Most of the neurons in V1 that are unchallengeably important for color vision (those with nonoriented, spatially low-pass receptive fields) are almost as unchallengeably ill-equipped to convey information about the local spatial structure of images. Although the limited spatial bandwidth of color vision[86] does not warrant machinery that samples spatiochromatic variations with the high density needed to capture achromatic variations, we do need some machinery to represent spatiochromatic contrast. There is considerable uncertainty about what constitutes this. Hubel and Wiesel[8] first described neurons that responded well to chromatic change in a small stimulus patch but did not respond when the patch was enlarged. The commonest interpretation is that the neurons have "double-opponent" receptive fields: a central region organized with one form of opponency (e.g., L–M) enclosed by a surrounding region organized with the opposite form (M–L). An elaborate receptive field organization like this is in fact completely unnecessary to explain the basic observation that enlarging a spot diminishes the chromatic response (see later discussion in Subsection 3.B.9, Influence of Context on Responses to Form). Other work that has characterized receptive fields with gratings[69,70,87,88] makes clear that many orientation-selective neurons (particularly simple cells) can be driven by isoluminant gratings, and a lot of these show bandpass spatial selectivity. However, even this behavior need not mean that a neuron is chromatically opponent: A nonopponent neuron will be excited by isoluminant patterns when the ratio of L:M cones in its receptive field departs from the ratio of their weights (1:9:1) in the luminosity function, V_λ. The average ratio of L to M cones estimated for monkey is 1:6:1.[89]

Theoretical analyses of optimal methods for representing the spatiochromatic structure of natural scenes[41,90] have not yet provided consistent guidance on the potential significance of filters with the properties of double-opponent receptive fields. In any event, a strongly color-opponent receptive field selective for orientation and spatial frequency implies a carefully orchestrated *complementary* arrangement of inputs from cones of different types. The random arrangement of cones in the mosaic must make it hard to achieve the requisite control of both the spatial and the chromatic properties of a receptive field. The spatial arrangement of cone inputs to cortical receptive fields has been little studied. Conway and colleagues[91,92] characterized a sample of red–green opponent cells and found most to have the complementary arrangement of cone inputs expected of true double-opponent cells. This observation is surprising in view of the overall rarity of bandpass spatial selectivity among strongly opponent neurons, and it has been vigorously challenged on methodological grounds by Johnson *et al.*[93] They found[70] that the complementary arrangement of

cone inputs expected of a true double-opponent cell is in fact very rare.

Analysis of the cone inputs to simple and complex cells shows that many are *weakly* color opponent.[69,88,93] A useful way to conceive of this is that the different classes of cones are not identically distributed within the receptive field. Weak opponency might be important for conveying information about the spatiochromatic structure of images, but it might equally be an inconsequential side effect of the way in which cones of all types are tapped indiscriminately during the assembly of spatially-tuned receptive fields (Fig. 2). A useful approach to resolving this issue is to consider how reliably cells can convey spatiochromatic structure in images. In particular, is a neuron's tuning for color stable to variations in other attributes of the stimulus (e.g., its orientation)? We should expect stable chromatic tuning of neurons that have a role in signaling color but not of neurons whose chromatic properties are accidental. Neurons with weak opponency do indeed seem to carry the least stable color signals. Stimulus size has a substantial effect on the chromatic tuning of weakly opponent neurons, less effect on the tuning of those that are nonopponent, and no effect on the tuning of strongly opponent neurons.[88] Stimulus contrast has a substantial effect on the chromatic tuning of weakly opponent neurons, less effect on the tuning of nonopponent ones, and no effect on the tuning of strongly opponent ones.[47] In many weakly opponent cells the spatial frequency tuning depends on the chromatic properties of the stimulus.[69,70,87] These unstable characteristics are hard to reconcile with a role in color vision but are understandable as an epiphenomenon: Given the random arrangement of L and M cones in the retinal mosaic, a V1 cell that draws inputs from all available cones will often have different proportions of L and M cones in the antagonistic parts of its receptive field, particularly if the receptive field is small. This will result in spatial-frequency-dependent color opponency that is strongest at low spatial frequencies, and most pronounced in the neurons with small receptive fields. Weakly opponent neurons have among the smallest receptive fields in V1.[88]

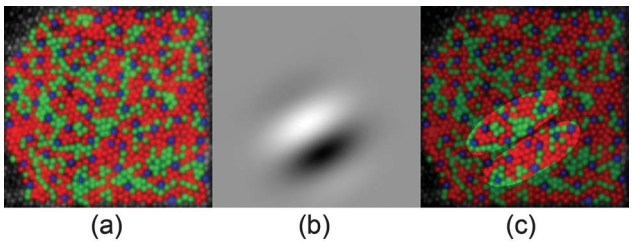

Fig. 2. How the random arrangement of L and M cones in the retinal mosaic can give rise to color opponency in cortical receptive fields. (a) The mosaic of identified cones in foveal retina of the macaque (from Ref. 63). (b) The spatial distribution of sensitivity within a notional simple cell receptive field tuned to $\sim 4\ \mathrm{c.deg^{-1}}$. (c) A cartoon of the two principal subregions of the receptive field superimposed as a window on the mosaic. Clustering of L and M cones results in the different subregions of the receptive fields (assuming they draw on all available cones) receiving L- and M-cone inputs in different proportions.

3. Influence of Spatial Context on Color Processing

It has been known for a long time that spatial as well as temporal context can have a powerful influence on color appearance. Among a range of sometimes dramatic effects (e.g., Refs. 94,95), chromatic induction—the change in the color of a patch brought about by enclosing it within a surrounding annulus of different color—has particularly interested physiologists because it might be simply explained by the behavior of neurons with double-opponent receptive fields.[96] Although the existence of such neurons remains controversial (see above), abundant evidence points to the powerful influence of spatial context on the behavior of other kinds of neurons in cortex. Early work on chromatic context focused on extrastriate cortex (e.g., Zeki[97]), but recent attention has been drawn to V1 and to the influence of the region around a neuron's classical receptive field (CRF) (the region within which stimulation directly alters discharge). For most neurons the CRF is embedded in a larger surrounding region where stimulation suppresses the response to concurrent stimulation of the CRF (see Subsection 3.B.9, Influence of Context on Responses to Form). Among neurons that responded well to patches of uniform chromaticity, Wachtler *et al.*[73] found that stimulation of regions well outside the CRF could alter the chromatic signature of the CRF. Solomon *et al.*[88] found something a bit different: although the color of the stimulus falling on the surround influenced the strength of suppression, it did not alter the chromatic signature of the CRF. Moreover, among those neurons that were strongly color opponent (those with nonoriented receptive fields and low-pass spatial tuning), surround suppression of any kind was rare; when it occurred it was weak and generally insensitive to isoluminant stimuli. As Ts'o and Gilbert[98] point out, cells with surrounds like this can behave as though they have double-opponent receptive fields (they do not).

3. SPATIAL CODING

A. Lateral Geniculate Nucleus

When driven by achromatic stimuli, the spatial receptive fields of primate LGN cells (and of the retinal ganglion cells that provide their input) for the most part resemble the classic center–surround fields first described in cat by Kuffler.[99] These cells can usefully be characterized in the spatial frequency domain, using the methods developed by Enroth-Cugell and Robson.[100] Because their receptive fields are circularly symmetric, LGN cells are for all practical purposes indifferent to grating orientation, so it is sufficient to consider their behavior in only one space or spatial frequency dimension. When studied with achromatic gratings, most macaque LGN cells have spatial tuning functions that are well described by the difference of two Gaussians, which correspond to the center and surround mechanisms of the receptive field.[101,102] Spatial frequency domain measurements can be used to estimate the spatial structure of the center and surround, and this analysis yields estimates of the sizes of these mechanisms that are in reasonable correspondence with the anatomy of their presumed retinal substrates.[103] The spatial frequency tuning curves of LGN cells are all very broad and

do not appear to correspond in any clear way with the orientation and spatial frequency selective mechanisms defined psychophysically (as we discuss below, cortical cells form a much better match). All but a few M and P cells show linear spatial summation by the criteria established by Hochstein and Shapley[104]; the few nonlinear cells are almost all M cells, but the suggestion that these might form a separate class[101] has not been borne out by subsequent work.[102,105]

In addition to their distinctive patterns of cone input noted earlier, there are three salient differences between M and P cells that are conveniently revealed with achromatic gratings. First, M cells are more sensitive to contrast, with a contrast gain that is about seven times greater than that of P cells at low contrasts.[101] At high contrasts, the difference in contrast response between the two types is attenuated because M-cell responses saturate while P-cell responses do not.[101] It is important to realize that neither cell type possesses a true contrast threshold—the contrast-response function for cells of all types rises smoothly and regularly for all contrasts[102,105]; there is no accelerating nonlinearity of the kind associated with cortical thresholds. Second, M cells respond better to high temporal frequencies than P cells, respond more transiently to contrast steps, and have somewhat shorter integration times and visual latencies.[105–108] Finally, M cells show evidence for a nonlinear contrast gain control mechanism like that characterized in cat retina by Shapley and Victor.[109,110] This gain control enhances responses to rapid stimulus fluctuations at high contrasts and is probably responsible for the contrast-response saturation and transient temporal response of M cells.

The spatiotemporal properties of the third (probably heterogeneous) cell group—the so-called K (koniocellular) cells—have been less well characterized than those of P and M cells,[111] although as we have discussed, some have distinctive chromatic properties. K cells are most prevalent at interlaminar boundaries but also occur within the main layers of the macaque LGN, so studies of M and P cells probably include some. Some might elude electrodes, but others probably have properties much like those of their neighboring P and M cells, since there is little sign that neurons recorded near interlaminar borders differ in spatiotemporal organization from those recorded in the core of the laminae.[105,112] This is also consistent with the spatiotemporal properties of K cells studied in primate species where the cells are more favorably located for electrophysiology.[113]

Virtually all the output from LGN is delivered to striate cortex, principally to layer 4c, which is divided into sublayers 4cα and 4cβ that receive input from the M and P layers, respectively.[114] Cells in layer 4c are quite small and extraordinarily densely packed[115] and so are difficult to isolate with microelectrodes and are therefore underrepresented in most cortical recordings. Many seem to have properties similar to those of LGN afferents, including limited binocular interaction, lack of orientation selectivity, and the distinctive response signatures of separate M- and P-cell inputs in the α and β sublayers.[8,116] Many studies and models of cortex discount these cells, in effect pretending that they are just cortical replicas of LGN inputs. There are indications that they have interesting physiological properties (see the earlier discussion of chromatic tuning in cortical neurons), but we do not discuss them further here.

B. Cortex

In 1968, Hubel and Wiesel[8] provided the first comprehensive description of the visual responses of cells in monkey V1. As noted earlier, they found few cells that responded vigorously or exclusively to purely chromatic stimuli: The striate cortex devotes the bulk of its resources to representing the achromatic spatial structure of images—an allocation that is reflected in the much greater spatiotemporal bandwidth of achromatic vision.

The three distinctive features of most cortical cells that differentiate them from cells in the LGN are binocular combination, orientation selectivity, and directional movement selectivity.[8] It is the latter two of these properties that will concern us here, along with the related property of selectivity for stimulus size or spatial frequency.[117,118] As proposed by Hubel and Wiesel, selectivity for orientation, size or spatial frequency, and direction seem to be established primarily through suitable convergent connections from LGN inputs to cells in the input layers of V1. The most direct evidence comes from experiments in cat,[119] but there is no reason to believe that cats and primates differ importantly in this respect. It has usually been supposed that this selectivity results from carefully sculpted patterns of input, but it has recently been shown by Ringach[120] (see also Soodak[121]) that known features of retinal ganglion cell mosaics combined with a haphazard pattern of feedforward connections can create a surprisingly accurate replica of the first stage of cortical stimulus selectivity; this idea is conceptually similar to one we considered earlier, that the chromatic properties of LGN cells can also arise from indiscriminate wiring.

The simplicity of the feedforward model makes it attractive to apply linear-systems approaches to the analysis of the spatial and temporal properties of cortical cells; the methods of frequency analysis are among the most appropriate and have gained many adherents (see Shapley and Lennie[122]). An important stimulus to this approach was the development of a rich body of psychophysical data and theory on the existence of channels selective for orientation, spatial frequency, and direction (see De Valois and De Valois[123] and Graham[9]). The clear expectation from this work was that individual cortical neurons would have properties corresponding to those expected of the elemental constituents of these channels, and this expectation was largely fulfilled by the demonstration of quantitatively appropriate selectivities in monkey cortical neurons,[117,118] along with behavioral demonstrations of the essential identity of monkey and human spatial visual performance.[124,125]

1. Space and Spatial Frequency Representations

The spatial receptive fields of cortical cells are conveniently thought of in four complementary dimensions—the two usual dimensions of space and the two dimensions of spatial frequency. In polar coordinates, two-dimensional spatial frequency is expressed as an orientation and a spatial frequency component, allowing

convenient comparisons with neuronal tuning properties and the simple computation of predicted linear receptive field profiles by Fourier analysis [Fig. 3(a)]. The essential feature exposed by this representation is that visual cortical neurons are localized in *both* space and spatial frequency and that populations of cells therefore jointly represent information about where stimuli are and what spatial structure they possess [Fig. 3(b)]. This joint representation of space and spatial frequency is equivalent to neither a pure spatial representation (pixels) nor the pure frequency representation (gratings) but retains some of the desirable features of each. The formerly energetic debate about whether cells represent "features" or "frequencies"[126,127] is thus easily answered: both and neither.

The mere existence of this four-dimensional selectivity in cortical cells does not tell us how the cortex represents images—there are many possible families of tuning functions that could be used to represent images, and there is no general agreement about which of these representations might be optimal, either for brains or for other image analysis machines. Families of neuronlike filters can be derived from statistical analysis of natural images,[128–130] but, since the shapes of the filters depend on the details of the analysis performed, this approach is only suggestive and does not generate a specific encoding model without additional (rather strong) assumptions.

The topographic representation of the visual field in cortex is well known (see Schwartz *et al.*[131] for an elegant representation and formal account). As is evident from Fig. 3(c), the two-dimensional spatial frequency tuning curves of cortical cells also effectively tile spatial frequency space, jointly representing all orientations and spatial frequencies. The specifics of the tiling tell how space and frequency are represented by these cells. Spatial frequency and orientation selectivity are related—when viewed in linear coordinates [Fig. 3(c)] the two-dimensional tuning curves are mostly moderately elongated along a radial axis, and extreme or amorphous shapes (e.g., sausages, amoebas) are rare. Across cells, orientation and spatial frequency bandwidths are roughly proportional, with the orientation bandwidth being somewhat smaller than the spatial frequency bandwidth when both are expressed in the linear frequency units used in Fig. 3(c) (hence the radial elongation of these fields). This corresponds to spatial receptive fields that are somewhat elongated parallel to the preferred orientation, as noted by Hubel and Wiesel [Fig. 3(b)]. For individual cells, tuning curves for orientation and spatial frequency are roughly independent, so that the preferred orientation does not depend much on the spatial frequency tested.[132]

To understand cortical encoding, it is also important to know how neuronal bandwidths vary across frequency space, in particular how they depend on preferred spatial frequency. If all receptive fields were scaled replicas of one another, then, as preferred spatial frequency increased, the bandwidth of tuning would remain constant when expressed in *ratio* units (octave bandwidth for spatial frequency and orientation tuning width in degrees). If, on the other hand, all receptive fields were the same size regardless of preferred spatial frequency, then the bandwidths would remain constant when expressed in *fre-*

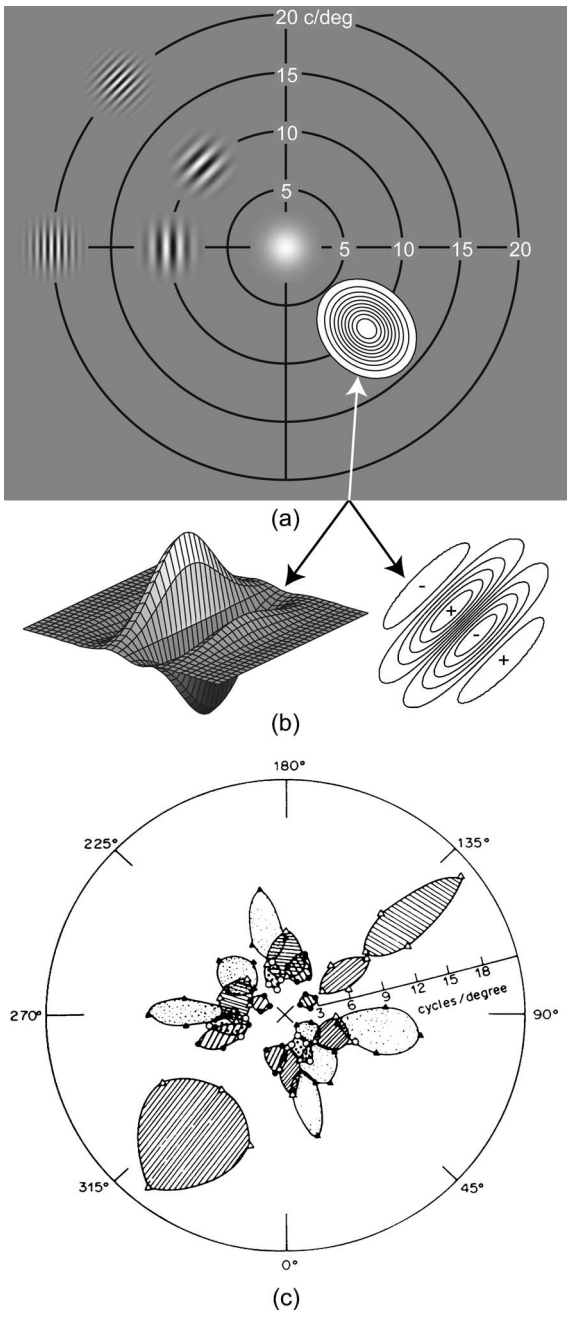

Fig. 3. How space and spatial frequency are jointly represented by cortical receptive fields. (a) Two-dimensional spatial frequency space, namely, the Fourier plane. Each point represents a grating of a particular orientation and spatial frequency, as indicated by the grating images placed on the appropriate locations of the space to the left and above the origin; the blob at the origin is a "grating" of zero spatial frequency. A tuning curve in orientation and spatial frequency forms a more or less compact zone in this space, as indicated by the contour map below and to the right of the origin. The Fourier transform of this tuning curve, as indicated by the arrows, gives (b) the receptive field profile of the matched linear neuron (shown in perspective and contour-map views), which is clearly similar to that of many simple cortical receptive fields. (c) The two-dimensional tuning curves of a population of cortical cells in macaque V1 are plotted in the Fourier plane. They are dispersed in orientation and spatial frequency to tile the space (from De Valois *et al.*[118]). Each tuning curve corresponds to an underlying spatial filter that can be computed by the arithmetic cartooned in (a) and (b).

quency units and would decrease in proportion to preferred frequency when expressed in ratio units. The truth lies in between these two extremes: Octave bandwidths decrease with preferred spatial frequency but not in proportion to it.[118] This behavior could reflect a continuous variation in bandwidth of a single population of filters, which would mean that the cortex treated information from low- and high-spatial-frequency mechanisms in a fundamentally different way. Alternatively, it could reflect the existence of multiple cell populations—for example, a group of narrowly tuned units preferring all frequencies and a second group of broadly tuned units preferring only low frequencies.

There are a number of image transforms in the family of "wavelet transforms" that are consistent with these properties,[133] but the available data do not crisply discriminate the many available alternatives. One striking feature of cortical spatial representation that is not easily captured by transform theories is the heterogeneity of stimulus selectivities exhibited by cells in V1—some cells are tuned for orientation and spatial frequency ranges as narrow as 10° and a fraction of an octave, while others respond to all orientations and a very wide range of spatial frequencies.[117,118] This suggests that V1 may best be thought of as containing multiple parallel representations of the image, each specialized to highlight some particular kind of information. The diverse pattern of output pathways from V1 to the extrastriate visual cortex[134,135] is certainly consistent with this view, as is the idea of multiple parallel processing streams within V1[136] that we consider below.

2. Representing Time and Motion

In the time domain, cortical response patterns differ distinctively from those in the LGN in that most cells have quite limited temporal resolution. Most LGN cells respond to gratings drifting at temporal frequencies in excess of 50 Hz, whereas few cortical cells resolve frequencies more than half as high[108,137] (this has always been something of a puzzle to physiologists—in most domains, cortical sensitivity is similar to our own, but at high drift rates cells stubbornly refuse to respond to stimuli that are easily seen). Some V1 cells have low-pass temporal characteristics and others are more bandpass, but there is little evidence for multiple distinct temporal channels. An exception might be the direction-selective cells, presumably with predominantly M-cell input from LGN, that project to extrastriate area MT; these seem to have unusually good temporal resolution.[138]

Directional movement selectivity, which can be visualized as a correlated sensitivity to spatial and temporal frequency, is a clear feature of a distinctive subpopulation of cortical cells, many of which are likely to derive their main inputs from the M cells of the LGN. The origins of direction selectivity are conceptually similar to the origins of orientation selectivity. Adelson and Bergen[139] offered the key insight that the detection of direction is equivalent to the detection of orientation in space–time and proposed a model of motion detection based on oriented linear space–time filters [Fig. 4(a)]. Subsequent work in both cat[140–142] and monkey[143–145] has uncovered the kind of linear spatiotemporal receptive field structures proposed by Adelson and Bergen in directionally-selective cells [Fig. 4(b)]. Just as the analogous maps in space predict orientation selectivity, these space–time maps provide at least a qualitative prediction of the cells' directional selectivity. As one would expect, cells that are not direction selective do not have filters that are tilted in space–time.

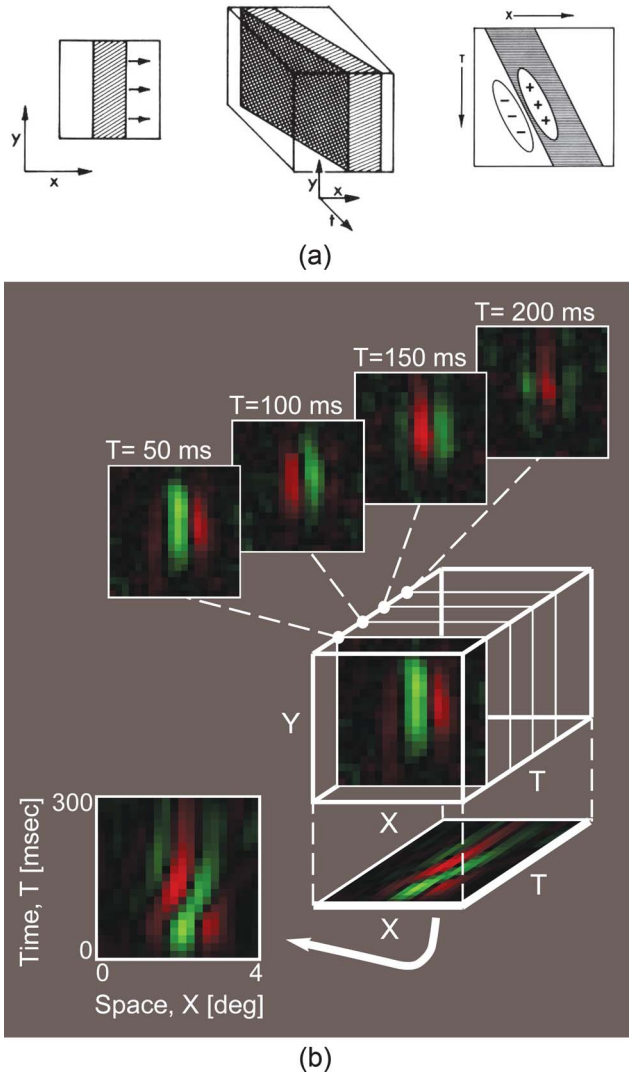

Fig. 4. Motion is orientation in space–time and is detected by receptive fields that are oriented in space–time. (a) A schematic diagram of a vertical bar in rightward motion (left), the volume it traces out in space–time (middle), and a view from "above" of the $x-t$ plane within which one can imagine a tilted receptive field that would be selective for direction of motion (right, from Adelson and Bergen[139]). (b) A three-dimensional space–time map of the receptive field of a simple cell from cat V1, derived with a reverse-correlation technique. At the top are four spatial receptive field maps computed by correlating each spike with a preceding random stimulus at four different indicated delays. The evolution of the receptive field with time is visualized by collapsing the resulting three-dimensional volume onto the $x-t$ plane indicated in perspective at the bottom right and brought upright at the bottom left. The receptive field is oriented in space–time, and the orientation predicts the neuron's direction preference (courtesy of G. C. DeAngelis after Ref. 277).

The creation of direction selectivity by linear filtering gives rise to an ambiguity in motion signaling—V1 cells are insensitive to the motion of stimulus components out-

side their filter passband, which means that they cannot, in general, signal the motion of complex patterns with multiple components dispersed in orientation.[138,146] The problem arises when orientation information is limited, which is sometimes referred to—not quite accurately—as the "aperture problem," because images viewed through small apertures often contain only a small range of orientations. This problem is solved by areas downstream of V1 such as MT[146]; the recent observation that some V1 cells can signal the motion of line endings[147] does not solve the problem because line endings contain all orientations, the aperture problem does not exist, and V1's linear directional filters therefore provide suitable motion signals.

In summary, as far as the initial establishment of cortical selectivity is concerned, it is productive to consider cells as linear filters operating on the three-dimensional pattern of spatiotemporal input; these filters correspond at least broadly with the spatial channels inferred from psychophysical experiments.[9,123]

3. Contrast Adaptation

An important link between the characteristics of the spatially-selective channels established psychophysically and the spatial selectivity of individual cortical neurons has been provided by contrast adaptation—a phenomenon whereby prolonged viewing of a contrast pattern either by a person[148] or a neuron[149] often results in loss of sensitivity or response from which recovery is slow.

Psychophysical work shows this loss of sensitivity to be substantially selective for the same stimulus attributes as cortical cells—orientation, spatial frequency, direction of movement, etc.—and as a result adaptation is often proposed as a key point of entry to the behavioral exploration of visual cortical machinery—the "psychophysicist's microelectrode." Physiological work has on the whole confirmed this inference but also has revealed some interesting complexities.

Contrast adaptation profoundly depresses response in many cortical neurons in cat[150,151] and monkey.[152] Although it is now known that M cells in LGN adapt[153] (reflecting changes that occur in retina[154]), that adaptation is not substantially expressed in cortex under the conditions typically used to study neurons. Nevertheless, studies of adaptation have not established particularly tight connections between the psychophysical channels and the properties of individual cortical neurons. First, adaptation clearly occurs at multiple stages—perhaps at every stage—of visual analysis, including pathways beyond striate cortex,[155] and the cascaded effect is expressed in perception. Second, within V1, it is clear that different neurons express adaptation differently. In some, sensitivity is proportionally depressed for all stimuli to which the cells respond, but in others the sensitivity is depressed locally, and the neuron's tuning on the relevant stimulus dimension is deformed, revealing drive from multiple separately-adaptable mechanisms. We noted earlier that this happens in chromatic mechanisms; it happens too in the spatial domain.[156,157] Third, there might be different forms of adaptation, working on different time courses. Although most work has characterized mechanisms that appear to lose and recover sensitivity on a time scale of seconds, some recent observations[158–160] point to the existence of a fast-acting form that also reveals multiple, tuned mechanisms driving many cells.

The multiple expressions of contrast adaptation partly reflect the action of multiple underlying mechanisms; two have been examined experimentally and theoretically. Adaptation causes hyperpolarization of the neuron's resting membrane potential that raises its threshold for firing.[161] This results from the activation of a particular kind of potassium channel and is nonsynaptic—it occurs even when cells are activated by direct current injection.[162] Adaptation can also result from synaptic depression, the progressive loss of efficacy of excitatory synapses as they exhaust themselves during activity.[163] Hyperpolarization induced by adaptation causes an unselective loss of sensitivity to all stimuli, whereas (homo)synaptic depression affects only signals carried by the depressed input connections. Given that adaptation can occur in at least two ways in a given cell, and at each successive level in the cortical network, it is not surprising that a complex picture results. This complexity is only faintly reflected back to the world of psychophysics, where many different patterns of neuronal adaptation might produce indistinguishable perceptual effects.

4. Linear Analysis of Cortical Receptive Fields

Hubel and Wiesel divided V1 receptive fields into two classes, on the basis of qualitative criteria that amount to a test of linear spatial summation. Simple cells respond to patterns that approximately match the separately-measured distributions of excitation and inhibition in their receptive fields; complex cells do not. The natural inference that simple cells are approximately linear in spatial summation, while complex cells are nonlinear, was demonstrated in cat by Movshon *et al.*[164,165] and in monkey by De Valois *et al.*[118] For simple cells, then, one expects the measured receptive field structure in space and spatial frequency to match through Fourier analysis. This was shown in the single dimension of spatial frequency by Movshon *et al.* and De Valois *et al.*, in two spatial dimensions in cat by Palmer and his colleagues,[166,167] and in space–time and spatiotemporal frequency by DeAngelis *et al.*[168] No similar demonstration exists for monkey, but there is every reason to assume the same outcome would be obtained. Another consequence of their linearity is that simple cells are sensitive to spatial phase and respond to drifting sinusoidal gratings with activity strongly synchronized to the passage of each grating bar across the receptive field.[118,164]

For complex cells as described by Hubel and Wiesel, one would not expect to see agreement between receptive field structure in space and spatial frequency, and this is generally evident even from casual exploration—complex cells often respond well to grating patterns in which several stimulus cycles fall within an apparently homogeneous region of the receptive field. For most complex cells, the question is complicated by the nonlinear nature of local responses, which are roughly equal for pattern increments and decrements. Complex cells are also relatively insensitive to spatial phase and respond to drifting gratings by elevating their firing rate without synchronization to the passage of the individual bars. Despite these strong signs of nonlinearity, it turns out that the most useful

models of complex cell responses begin with sets of linear filters, whose outputs are nonlinearly transformed and combined (in cats[165,169,170]; in monkeys[171,172]). A key feature of these models is that the underlying linear filters—not the later nonlinearities—determine the set of stimuli to which the neuron will respond. So, for example, these models predict that "second-order" stimuli defined by texture rather than luminance differences,[173] to which linear filters are blind, should not elicit responses from V1 complex cells. This is generally the case.[174] This also means that the four-dimensional space/spatial frequency representation of Fig. 4 remains useful even for complex cells—the spatial receptive fields in this representation correspond to the underlying linear filters; each complex cell combines several such filters.[139,165,172] This is of course merely a formal restatement of the hierarchical feedforward theory of cortical receptive field construction proposed by Hubel and Wiesel.[7]

All spiking neurons have one inescapable nonlinearity: They fire action potentials only above a certain voltage threshold and are silent otherwise. So even the simplest simple cell model is not linear but is an "LN" or linear–nonlinear model. Complex cell models based on linear filters are also LN models, and this formal similarity suggests that the two cell types might not be entirely distinct; a number of recent theoretical studies have made a similar suggestion.[175,176] Quantitative measurements of the degree to which each cell type's firing is modulated in synchrony with the bars of moving gratings show a strongly bimodal distribution but one that is continuous; cells with intermediate properties certainly exist.[177] Recent analyses show that the bimodal distribution of modulation evident in spiking activity is not associated, at least in cats, with a similar bimodality of the underlying voltage responses[178]; the bimodality in the spiking responses arises because simple and complex cells differ in the relationship between voltage and spiking activity. The possibility that simple and complex cells have similar underlying computational architectures is an attractive motivation for analyses that characterize all cortical cells using the same framework.[172]

5. Contrast Gain Control

Linear-systems analyses of cortical cells, especially simple cells, have proved to be very powerful. As we have seen, they provide reasonably accurate accounts of orientation, spatial frequency, and direction selectivity and also account well for V1 responses to such compound targets as checkerboards and plaids.[146,179] It is also clear, however, that linear models have a number of important limitations and inaccuracies, and much effort in recent years has gone into modifying and adapting these models to improve their performance and to provide an account of the way cortical receptive field properties emerge from the action of known cortical circuits.

It turns out that three important failures of the linear model can be understood in terms of a single mechanism. The first of these is that cortical cell responses to increasing contrast are not linear—responses saturate at high contrasts[180]; it is important to note that the saturation is not simply an output nonlinearity because saturation occurs at a fixed stimulus contrast and not at a fixed response level for different stimuli; this means that, in general, cortical tuning curves remain invariant in shape at different contrasts (in cat: Ref. 181; in monkey: Ref. 182). The second is that cortical cells are often suppressed by stimuli outside their orientation and spatial frequency tuning passbands, even though those stimuli do not evoke a response when presented alone.[182–187] The third is that the temporal characteristics of cortical response vary with contrast–as contrast increases, responses become faster and extend to higher temporal frequencies.[182,188,189] These three effects can be explained by a divisive normalization mechanism (or contrast gain control) that adjusts neuronal gain and dynamics in relation to the pooled activity of nearby neurons.[186,190,191] The contrast gain control model is a remarkably successful "fix" for the three main failures of the linear model, providing an accurate quantitative description of all three nonlinear effects.[182]

What remains unclear is the mechanism (or mechanisms) by which gain control works. As originally conceived, the gain control model assumed that shunting inhibition would be driven by the collected activity of nearby cortical cells. More recently, it has been suggested that the properties of the gain control mechanism, at least as it manifests itself in cross-orientation suppression, are different from what one would expect of signals arising in V1—in particular, suppression is evident from stimuli drifting at rates higher than those that are usually effective in V1 cells.[192,193] Some have suggested that the gain control arises as the result of depression of the synapses of LGN cells,[193,194] while others implicate feedback signals originating in cortical areas outside V1.[192] There are also other models of cortical circuitry based on quite different architectures that seem capable of accounting for the main observations on cortical gain control.[195] And of course it is quite possible that different aspects of gain control result from different underlying machinery—cross-orientation suppression, for example, might have a different basis than contrast saturation. However, the descriptive success of the gain control model is independent of the way it is implemented, and as such it provides a conceptual and quantitative advance over the basic linear model without obscuring any of its important virtues.

6. Threshold Nonlinearity

The contrast gain control model accounts for the saturation of cortical response at high contrast, but there is also an important nonlinearity in response at low contrast, attributable to the neuronal threshold for spiking. Most cortical cells have low or zero maintained firing rates, which means that, in the absence of stimulation, their mean membrane potential must be more negative than their spiking threshold. This means in turn that their responses to weak stimuli should, and do, have a relatively distinct threshold above which responses rise roughly linearly with the strength of the input.[164,196] In reality, a true "sharp corner" is not usually observed, and this can be understood in terms of the variability of the underlying voltage response: As stimulus intensity increases, this variability leads to a gradual increase in spiking activity as noise excursions become increasingly likely to bring the neuron to its firing threshold. This smoothes the sharp corner and creates an accelerating nonlinear rela-

tionship between the mean voltage response and the spiking response,[197] creating a contrast response that is approximately proportional to the square of contrast.[180,198–200]

This accelerating nonlinearity has an important impact on the selectivity of cortical cells, amplifying larger responses more than smaller ones and effectively increasing the selectivity of spike responses compared with the underlying voltage response.[197] This nonlinear enhancement of selectivity is evident in responses to a variety of stimuli and probably accounts for much of the deviation that is observed between the predictions of linear models and the actual spike responses.[201] As one would expect from this idea, the *voltage* response of cortical cells often adheres closely to the predictions of linear models, while the *spike* responses are clearly more nonlinear.[202–204]

7. Intracortical Inhibition

Contrast gain control models predict that stimuli ineffective in driving cortical cells may still suppress their responses to other stimuli, by modifying their contrast gain. But as we have seen, contrast gain control might not involve synaptic inhibition at all,[194] and inhibitory circuits are key elements of cortical function. About 20% of neurons in all layers of cortex are GABAergic local-circuit neurons, and a similar fraction of cortical synaptic contacts are inhibitory.[205,206] Blockers of GABA increase the excitability of cortical cells, suggesting that cells are tonically suppressed under normal conditions. What then might be the roles of cortical inhibition in determining response properties, and how does cortical inhibition act in the context of linear and nonlinear models of cortex?

There is an essential role for synaptic inhibition in linear models of cortical circuits. LGN cells, like any cells, rectify when their firing is suppressed to zero. A simple feedforward additive combination of LGN inputs would therefore not display the linear summation behavior evident in many cortical neurons; the rectification would create strong nonlinearities. Moreover, the nonlinearities would be contrast dependent, since LGN cells rectify only when their firing rates are modulated strongly enough to suppress their relatively high maintained activity. In fact, cortical cells that show roughly linear behavior do so at all contrasts.[207] The resolution of this problem is provided by "push–pull" circuits in cortex.[202,208–211] In its simplest form, the push–pull idea holds that for each excitatory input from the LGN, there is a complementary inhibitory input of opposite receptive field structure. The combination of the excitatory push and the inhibitory pull overcomes the rectification and restores linear behavior. Since all direct connections from LGN to cortex appear to be excitatory, the creation of the push–pull requires synaptic inhibition and has been successfully modeled in this way by Troyer *et al.*[195] among others.

Cortical inhibition also sharpens neuronal selectivity for orientation (and, by extension, presumably for other stimulus features) by suppressing responses to nonoptimal stimuli. This might be due to a general regulation of cortical excitability as predicted by the contrast gain control model, or it could be due to selective inhibition configured to suppress the flanks of tuning curves. Distinguishing these two kinds of inhibition is not straightforward. For example, intracortical infusions of GABA antagonists disrupt orientation selectivity,[184] but blocking GABA-mediated inhibition intracellularly does not have the same effect[212]; this suggests that the main effect of GABAergic input may be to regulate overall excitability and not to sculpt tuning curves. Evidence for tuned inhibition comes from experiments showing that cortical cells are often suppressed below their baseline rates by stimuli of whose orientations and spatial frequencies are close to the preferred (for example, Refs. 185,213). Orientation selectivity in the period immediately following stimulus onset sometimes shows delayed suppressive influences at nonpreferred stimulus orientations that create "Mexican-hat"-shaped tuning curves,[214,215] though the strength and prevalence of this effect has been questioned.[216] It turns out that "tuned" inhibitory effect is weak or absent when stimuli are confined to the central core of the receptive field,[217] suggesting that this effect is primarily a contextual modulation from the receptive field surround, where selective suppression is well documented (see below). In direction-selective cells, there is clear evidence for selective "opponent" inhibition by the nonpreferred direction of motion, but the inhibition in nondirectional cells is not obviously tuned.[172]

So there may be three more-or-less distinct forms of intracortical inhibition: a linearizing push-pull, an untuned regulation of gain or excitability, and a selective "sharpening" that specifically suppresses responses to nonoptimal stimuli.

8. Recurrent Cortical Amplification

In the classical scheme, orientation selectivity is determined by the spatial arrangement of feedforward inputs to the receptive field, enhanced by an accelerating nonlinearity. An argument against this idea is that it does not correspond well with known cortical anatomy: Even in the layers receiving direct LGN input, the great majority of excitatory synaptic contacts onto cortical cells are of intracortical origin.[218] Anatomical analysis suggests that recurrent excitatory connections function as a cortical amplifier that enhances relatively weak signals arriving from thalamus.[175,219,220] Suitably configured, this circuit could be coordinated with recurrent inhibition not only to amplify but also to enhance the selectivity of a weakly-tuned input from the LGN; this general idea forms the basis for a number of current models of visual cortex (e.g., Refs. 221–224). These generally postulate a weak and weakly-selective pattern of LGN input, amplified by recurrent cortical excitation from cells of similar preference and narrowed by recurrent inhibition from cells of differing preference. These models are certainly more realistically related to cortical circuits than the simpler feedforward models we have been considering and generally account well for most of the available data. They are, for example, consistent with evidence that the feedforward inputs to cortical cells may form only a compact central zone of the larger receptive field.[119,225]

The most challenging data for recurrent models of cortical selectivity are those of Ferster and his colleagues,[226,227] which attempt to isolate cortical cells from their intracortical inputs, leaving them driven only

by thalamic afferents. These are difficult experiments, but within their limits of precision they show that the selectivity of feedforward inputs matches those of the cells' responses measured under normal conditions; this finding leaves no role for cortical circuits in sharpening tuning. The data also reveal, however, that the feedforward input is, as expected, quite weak, and there is evidence that many simple cells may receive excitatory drive from complex cells.[172] Thus the role of recurrent cortical amplification in enhancing the gain of cortical responses seems indisputable, but its contribution to establishing or enhancing stimulus selectivity is less clear.

9. Influence of Context on Responses to Form

Earlier, we considered the influence of color context on color signaling by cortical cells, but, until now, our discussion of form processing has considered only the influence of stimuli that fall within the "classical" excitatory receptive field (CRF). In natural visual environments, stimuli are rarely isolated, and the spatiotemporal pattern falling on a particular receptive field is surrounded by other patterns that form a context. In recent years, much effort has been devoted to studying the influence of stimuli placed outside the CRF, to determine the influence of this context on V1 responses.

In their early studies, Hubel and Wiesel[8] noticed cells whose responses were suppressed when simple stimuli extended beyond the CRF. Originally they thought that these were a distinct class of "hypercomplex" cells, but it turned out that the property of end inhibition or "end stopping" was distributed variably across cells of both main types, with some examples of simple and complex cells being fully end stopped (hypercomplex), others being moderately suppressed, and others unaffected by contextual stimuli.[228–232] It also became clear that the phenomenon of end inhibition was in fact an inhibitory influence that could be detected throughout a roughly circular region surrounding the CRF on all sides and was not confined just to the end zones.[230–241]

The suppressive influence of the surround is selective—the most effective suppression is usually produced by stimuli whose orientation, spatial frequency, and direction of motion match the preferences of the CRF[231,232,236,240,241]; other, higher-order differences between center and surround stimuli may also influence surround suppression.[242] There is also some evidence that the suppression is strongest when the *stimulus* in the CRF matches the *stimulus* in the surround, even if neither is optimal for the cell.[237,240] The overall effect is that V1 cells respond best when their preferred stimulus is embedded in a nonpreferred context and are most suppressed when the preferred stimulus is surrounded by other preferred stimuli. Under some conditions, responses are not only not suppressed by mismatched surrounds but may actually be enhanced.[231,236,241] In other words, just as the center–surround organization of retinal ganglion cells enhances responses to spatial variations in luminance,[243] the analogous organization in cortical cells enhances responses to spatial variations in form and motion. This system may do more than just enhance "form contrast": Schwartz and Simoncelli[244] showed that the properties of V1 surrounds are well suited to remove the influence of long-range spatial correlations present in natural scenes and could therefore act to make V1's population across extended stimuli more efficient.

The mechanism of cortical surround suppression is complex and probably involves signals from several different sources. First, it might not be wholly distinct from the gain control by divisive normalization that we described within the receptive field. Divisive normalization models seem to provide the best quantitative account of surround suppression, just as they do for suppression within the CRF,[232,241] and it is therefore quite possible that some surround suppression—especially close to the CRF—is just a spatial extension of local suppressive mechanisms. Second, more remote surround effects may engage a different circuit. Although some components of suppression are fast,[241] others are clearly slow, being delayed 20–80 ms after the onset of response to stimuli in the CRF.[236,242,245,246] Angelucci *et al.*[246] and Cavanaugh *et al.*[232] showed that surround signals extend much further from the CRF than signals would be expected to propagate through horizontal interconnections within V1, and Bair *et al.*[245] showed that surround signals do not usually show the distance-dependent delay that would be expected from such horizontal propagation. Angelucci *et al.*[246] analyzed the feedforward and feedback connections between V1 and nearby extrastriate areas and concluded that the spatial extent of the surround was well matched to the spatial distribution of feedback input from the "near" extrastriate cortex, including such areas as V2, V3, and MT. Finally, some cells in the LGN have suppressive surrounds that attenuate responses to stimuli extending beyond their receptive field borders[247]; this suppression would of course be relayed forward to their V1 target cells, but there is no reason to believe it is stimulus selective.

4. SEGREGATION OF FUNCTION

One of the most vexing questions about the organization of striate cortex is the extent to which different attributes of the image are analyzed independently. We noted earlier the striking heterogeneity of tuning among cells—for example, the differences between those that respond well to chromatic modulation versus those that do not and the differences between those that are directionally selective and those that are not. Physiological evidence alone provides no sharp guidance as to whether the heterogeneity reflects multiple functional classes of neurons or simply the dispersion of the stimulus preferences needed to tile a multidimensional space with relatively narrowly tuned filters. A popular view is that functionally specialized groups of neurons are organized into coherent anatomical units. More might therefore be learned by coupling physiology to anatomy, from which we can establish whether neurons with particular visual characteristics are clustered.

The distinctive layered structure of striate cortex has been exhaustively characterized (reviewed by Colonnier[248]), as has the distribution of outputs and different types of inputs among the layers (reviewed in Lund[249] and Callaway[250]). A natural first step is to ask whether the parallel systems represented by the M, P,

and K pathways through the LGN remain segregated in V1. These three pathways distribute their terminals in different cortical layers: the M cells principally to layer 4cα, P cells principally to layers 4cβ and 4a, and K cells principally to layers 4a and 3 (reviewed by Lund[249]). Recordings made in layer 4 show laminar variations in response pattern that are characteristic of the appropriate LGN inputs,[116] but outside these the known correlation of layer with function is surprisingly slight. The most prominent association involves the M pathway from LGN to layer 4cα, and from there to layer 4b, where neurons have high contrast sensitivity and are often directionally selective[251]; many of these project to extrastriate area MT.[138] Yabuta *et al.*[252] showed that only the spiny stellate cells of layer 4b have selective M-pathway input; neighboring pyramidal cells (which likely project to areas V2 and V3) receive both M and P input from layer 4c.

There is now evidence[38] that the inputs from blue-on (S+) pathways from LGN terminate in layers 2/3 and upper 4a, while blue-off (S−) pathways terminate in layer 4a—both are some distance from the termination of the P pathway in layer 4cβ. But the special character of the pathways conveying cone-opponent signals to cortex is not expressed in any sharp stratification of the chromatic preferences of neurons, with the possible exception of their absence from layers 4cα, and 4b.[70,253]

A functional approach was pioneered by Malpeli *et al.*,[254] who recorded from V1 while reversibly inactivating individual LGN laminae. Most cortical cells showed evidence of mixed M and P input (K cells were then unknown, and the technique would have confounded K inputs with the others). Using a related method in bushbaby, a primate with separate K-cell layers, Allison *et al.*[255] showed a similar mixing of M and P inputs on most cortical cells outside layer 4, without the K-cell confound. Thus the physiological evidence provides little support for the idea of functional segregation by LGN input type and cortical layer once signals have entered the cortical circuit.

More attention has been paid to the expression of functional specialization within the two-dimensional map of the visual field represented on the cortical surface. The impetus to this was the discovery[256,257] that V1 contains regularly spaced regions (puffs, blobs) that are particularly reactive for the metabolic enzyme cytochrome oxidase. Early work to explore the physiology of neurons in blobs[258] found a concentration of color-opponent cells within them. Ts'o and Gilbert found this too[98] and also that blobs occurred in two types containing red–green and yellow–blue opponent cells (see also Landisman and Ts'o[259]). Other single-unit studies[69,260] have found no association between color opponency and blobs, and the distribution of LGN terminals of different types, and the routes of their subsequent pathways within V1, does not generally suggest much specialization. The P-dominant layer 4cβ projects substantially to the interblob regions[261,262] and perhaps also into blobs—different studies are not consistent here. M-dominant layer 4cα projects both directly and indirectly (via layer 4b) to blobs and interblob regions.[261,263]

The one clear anatomical result in this domain is that blobs are preferred targets of direct LGN projections from K cells.[35,264] Although blobs probably receive disproportionately strong input from the K pathway, this implies little about *functional* segregation because there is scant evidence that K cells carry distinctive visual signals—and the one exceptional group, the cells carrying S-cone signals, does not have a conspicuously periodic distribution of terminals in layer 4a.[38]

Larger-scale mapping to explore functional specialization using either 2–deoxyglucose[253] or optical imaging of evoked activity[265] also provides mixed evidence on specialization. Tootell *et al.* found that the greatest 2DG uptake in blobs resulted from chromatic *or* achromatic modulation of low-spatial-frequency patterns; Landisman and Ts'o found that the greatest differential activity evoked by isoluminant versus achromatic stimuli occurred in regions that were broadly isomorphic with blobs, although (as in the work of Tootell *et al.*) they found that chromatically modulated stimuli also evoked activity in interblob regions. Silverman *et al.*[266] noted that the apparent association between blobs and color opponency might well reflect a confound with a cortical mapping of spatial frequency tuning: Neurons tuned to low frequencies, whether color preferring or not, tend to occur in blobs. Moreover, they also have high contrast sensitivity,[267] implicating input from the M pathway. We noted earlier (in Subsection 3.B.1, Space and Spatial Frequency Representation) that cells tuned to low spatial frequencies tend to be more broadly tuned than ones preferring higher frequencies. Since the contrast distribution in natural images follows a $1/f$ relationship,[268] this broad tuning means that, on average, low-frequency-preferring cells in blobs would be more active than high-frequency-preferring cells outside them, leading to a greater local metabolic demand. Since cytochrome oxidase is a metabolic enzyme, one would expect it to be up-regulated in zones of higher metabolic activity.

Although the evidence for a special association between color opponency and blobs seems generally weak, we cannot rule out the possibility that chromatically opponent neurons occur in clusters within V1 (see, for example Dow and Vautin[269]). But clustering by itself is not strong evidence for anatomical segregation or a functional pathway because rich recurrent connectivity results in *all* receptive field properties being clustered in cortex. Clustering of functional properties is expected as a solution to the problem of representing multiple attributes of the image on a two-dimensional map[270] and seems often to be only loosely connected with anatomy. The basic visuotopic organization and columnar architecture provide a highly regular segregation of function at the level of a cortical hypercolumn.[271] Within a hypercolumn cortical inputs and outputs are very precisely segregated by layer, and intrinsic pathways seem to be precisely organized, yet variations in the visual properties of single neurons within a hypercolumn are usually modest and haphazard. Two nearby cortical neurons, chosen at random, will tend to respond to the same stimuli and to fire in a correlated way, suggesting that free mixing of signals in cortex is the rule rather than the exception.[272] Subthreshold spontaneous activity is strongly correlated over very wide areas of cortex and surprisingly long times,[273,274] again suggesting that neuronal activity patterns are not tightly confined to

small domains. This makes it difficult to distinguish functionally important cortical populations on structural grounds alone.

5. VISUAL ROLES OF NEURONS

To identify a particular neuronal population on functional grounds is in some ways easy. One can test responses to variations along a set of reasonably-chosen dimensions and use the result to declare a neuron selective for some dimensions and not others. By examining other neurons from the same part of the brain, one can determine what range of selectivities exist in the population of interest and ask whether the population represents the dimension in a comprehensive way. By this criterion, V1 cells represent virtually all relevant perceptual dimensions, as is fitting given V1's role as vision's gateway to the cortex. But suppose we refine the question and ask what dimensions V1 represents in a way that is *useful* for making perceptual decisions?

A convenient way to frame this is to ask: For what dimensions does a population of V1 cells give a response that is *invariant*—that is, for what dimensions is neuronal selectivity independent of the value on other dimensions? Consider the problem of reading out activity from a population of cells. Suppose that for V1 neurons the optimal orientation is independent of spatial frequency (it is). To interrogate a population of such neurons about orientation, one could add up the activity of cells preferring a single orientation but all spatial frequencies, because the peak of the population activity will remain at the optimum (the strong form of this occurs when the two dimensions are *separable*, meaning that the joint tuning function is the product of the separate tunings). But now suppose that the optimal color depends on spatial frequency (as it does for weakly opponent V1 cells). Now it is not simple to determine color from the population activity because a decoder must "look up" the spatial frequency to use the tuning information for each element, and simply adding activity will usually give an incorrect (or at best imprecise) answer. In psychological experiments, many pairs of dimensions are called *separable*, in the sense that variations in one dimension do not affect discrimination performance on the other; other pairs are *integral* and interfere with one another.[275]

So to return to our original quest for perceptually relevant representations in V1, we can ask: For which perceptually-separable pairs of dimensions does V1 provide an invariant code? We cannot provide a comprehensive answer, but there are informative examples. For combinations of dimensions, such as position, orientation, and spatial frequency, dimensions seem separable for all V1 cells, and the representation in V1 is the right one to consider for perceptual decision making.[276] For combinations involving color, only strongly opponent cells provide the right kind of representation—for this to be useful the output of this subpopulation should probably be segregated in some way. For yet other dimensions, such as the motion of compound patterns like plaids, V1 does not contain an invariant representation at all.[146] In such cases, further analysis of the output of V1 is required to compute a separable representation on which to base perceptual decisions. For this, we must look downstream, to the visual areas of the extrastriate cortex.[134]

ACKNOWLEDGMENTS

Work on this review was supported in part by research grants from the National Institutes of Health (EY 2017 and EY 4440). In preparing it we have benefited greatly from comments on earlier versions of the manuscript from Sam Solomon, Nicole Rust, and Lynne Kiorpes and from discussions with many colleagues and students.

Address correspondence to P. Lennie at pl@cns.nyu.edu, 1-212-998-3530, or J. A. Movshon at movshon@nyu.edu, 1-212-998-7880.

REFERENCES AND NOTES

1. D. H. Hubel, "Cortical neurobiology: a slanted historical perspective," Annu. Rev. Neurosci. **5**, 363–370 (1982).
2. V. Mountcastle, "The evolution of ideas concerning the function of the neocortex," Cereb. Cortex **5**, 289–295 (1995).
3. N. Y. Kiang, "Processing of speech by the auditory nervous system," J. Acoust. Soc. Am. **68**, 830–835 (1980).
4. G. S. Brindley, *Physiology of the Retina and the Visual Pathway*, 2nd ed. (Arnold, 1970), p. 311.
5. G. S. Brindley, *Physiology of the Retina and the Visual Pathway* (Arnold, London, 1960).
6. D. H. Hubel and T. N. Wiesel, "Receptive fields of single neurones in the cat's striate cortex," J. Physiol. (London) **148**, 574–591 (1959).
7. D. H. Hubel and T. N. Wiesel, "Receptive fields, binocular interactions, and functional architecture in the cat's visual cortex," J. Physiol. (London) **160**, 106–154 (1962).
8. D. H. Hubel and T. N. Wiesel, "Receptive fields and functional architecture of monkey striate cortex," J. Physiol. (London) **195**, 215–243 (1968).
9. N. Graham, *Visual Pattern Analyzers* (Oxford U. Press, New York, 1989).
10. M. M. Adams, P. R. Hof, R. Gattass, M. J. Webster, and L. G. Ungerleider, "Visual cortical projections and chemoarchitecture of macaque monkey pulvinar," J. Comp. Neurol. **419**, 377–393 (2000).
11. W. Fries, "The projection from the lateral geniculate nucleus to the prestriate cortex of the macaque monkey," Proc. R. Soc. London, Ser. B **213**, 73–86 (1981).
12. J. Bullier and H. Kennedy, "Projection of the lateral geniculate nucleus onto cortical area V2 in the macaque monkey," Exp. Brain Res. **53**, 168–172 (1983).
13. A. Lysakowski, G. P. Standage, and L. A. Benevento, "An investigation of collateral projections of the dorsal lateral geniculate nucleus and other subcortical structures to cortical areas V1 and V4 in the macaque monkey: a double label retrograde tracer study," Exp. Brain Res. **69**, 651–661 (1988).
14. L. C. Sincich, K. F. Park, M. J. Wohlgemuth, and J. C. Horton, "Bypassing V1: a direct geniculate input to area MT," Nat. Neurosci. **7**, 1123–1118 (2004).
15. P. H. Schiller and J. G. Malpeli, "The effect of striate cortex cooling on area 18 cells in the monkey," Brain Res. **126**, 366–369 (1977).
16. H. R. Rodman, C. G. Gross, and T. D. Albright, "Afferent basis of visual response properties in area MT of the macaque. I. Effects of striate cortex removal," J. Neurosci. **9**, 2033–2050 (1989).
17. H. R. Rodman, C. G. Gross, and T. D. Albright, "Afferent basis of visual response properties in area MT of the macaque. II. Effects of superior colliculus removal," J. Neurosci. **10**, 1154–1164 (1990).
18. C. E. Collins, D. C. Lyon, and J. H. Kaas, "Responses of neurons in the middle temporal visual area after long-standing lesions of the primary visual cortex in adult new world monkeys," J. Neurosci. **23**, 2251–2264 (2003).

19. J. D. Mollon, "The origins of modern color science," in *The Science of Color*, S. K. Shevell, ed. (Elsevier, 2003), pp. 1–39.
20. "The CIE triangle is brilliantly ingenious as an aid to the calculations of chromaticities which can be upheld in a court of law where colour specification is in dispute. But that triangle is monstrous as an indication of what is going on in the mechanism of vision. It displays all colours as a mixture of three primary lights, none of which have an existence that can be easily imagined. One of the three primaries is bright; it is a pure green from which is subtracted a lot of red which it does not contain. The other two primaries are quite dark; they have strong colour but zero luminance. These do not seem to me ingredients that lead to clarity in our conception of colour mechanisms and I am astonished that some physiologists and many psychologists employ them to instruct the young and bewilder the old." W. A. H. Rushton, "Pigments and signals in colour vision," J. Physiol. (London) **220**, 1–31P (1970).
21. L. M. Hurvich and D. Jameson, "An opponent-process theory of color vision," Psychol. Rev. **64**, 384–404 (1957).
22. R. L. De Valois, C. J. Smith, A. J. Karoly, and S. T. Kitai, "Electrical responses of primate visual system. I. Different layers of macaque lateral geniculate nucleus," J. Comp. Physiol. Psychol. **51**, 662–668 (1958).
23. R. L. De Valois, I. Abramov, and G. H. Jacobs, "Analysis of response patterns of LGN cells," J. Opt. Soc. Am. **56**, 966–977 (1966).
24. T. N. Wiesel and D. H. Hubel, "Spatial and chromatic interactions in the lateral geniculate body of the rhesus monkey," J. Neurophysiol. **29**, 1115–1156 (1966).
25. J. K. Bowmaker, H. J. A. Dartnall, J. N. Lythgoe, and J. D. Mollon, "The visual pigments of rods and cones in the rhesus monkey, *Macaca mulatta*," J. Physiol. (London) **274**, 329–348 (1978).
26. D. A. Baylor, B. J. Nunn, and J. L. Schnapf, "Spectral sensitivity of cones of the monkey *Macaca fascicularis*," J. Physiol. (London) **390**, 145–160 (1987).
27. V. Smith and J. Pokorny, "Spectral sensitivity of the foveal cone photopigments between 400 and 500 nm," Vision Res. **15**, 161–171 (1975).
28. G. H. Jacobs, "Photopigments and seeing—lessons from natural experiments," Invest. Ophthalmol. Visual Sci. **39**, 2205–2216 (1998).
29. W. S. Stiles, "The directional sensitivity of the retina and the spectral sensitivities of the rods and cones," Proc. R. Soc. London, Ser. B **127**, 64–105 (1939).
30. D. M. Schneeweis and J. L. Schnapf, "The photovoltage of macaque cone photoreceptors: adaptation, noise, and kinetics," J. Neurosci. **19**, 1203–1216 (1999).
31. V. C. Smith, J. Pokorny, B. B. Lee, and D. M. Dacey, "Primate horizontal cell dynamics: an analysis of sensitivity regulation in the outer retina," J. Neurophysiol. **85**, 545–558 (2001).
32. P. Gouras, "Identification of cone mechanisms in monkey ganglion cells," J. Physiol. (London) **199**, 533–547 (1968).
33. D. Dacey, O. S. Packer, L. Diller, D. Brainard, B. Peterson, and B. Lee, "Center surround receptive field structure of cone bipolar cells in primate retina," Vision Res. **40**, 1801–1811 (2000).
34. A. M. Derrington, J. Krauskopf, and P. Lennie, "Chromatic mechanisms in lateral geniculate nucleus of macaque," J. Physiol. (London) **357**, 241–265 (1984).
35. S. H. C. Hendry and T. Yoshioka, "A neurochemically distinct third channel in the macaque dorsal lateral geniculate nucleus," Science **264**, 575–577 (1994).
36. D. M. Dacey and O. S. Packer, "Colour coding in the primate retina: diverse cell types and cone-specific circuitry," Curr. Opin. Neurobiol. **13**, 421–427 (2003).
37. P. R. Martin, A. J. White, A. K. Goodchild, H. D. Wilder, and A. E. Sefton, "Evidence that blue-on cells are part of the third geniculocortical pathway in primates," Eur. J. Neurosci. **9**, 1536–1541 (1997).
38. S. Chatterjee and E. M. Callaway, "Parallel colour-opponent pathways to primary visual cortex," Nature **426**, 668–671 (2003).
39. G. Buchsbaum and A. Gottschalk, "Trichromacy, opponent colours coding and optimum colour information transmission in the retina," Proc. R. Soc. London, Ser. B **220**, 89–113 (1983).
40. D. L. Ruderman, T. W. Cronin, and C. C. Chiao, "Statistics of cone responses to natural images: implications for visual coding," J. Opt. Soc. Am. A **15**, 2036–2045 (1998).
41. T. Lee, T. Wachtler, and T. Sejnowski, "Color opponency is an efficient representation of spectral properties in natural scenes," Vision Res. **42**, 2095–2103 (2002).
42. B. B. Lee, C. Wehrhahn, G. Westheimer, and J. Kremers, "The spatial precision of macaque ganglion cell responses in relation to vernier acuity of human observers," Vision Res. **35**, 2743–2758 (1995).
43. V. H. Perry, R. Öhler, and A. Cowey, "Retinal ganglion cells that project to the dorsal lateral geniculate nucleus in the macaque monkey," Neuroscience (Oxford) **12**, 1101–1123 (1984).
44. B. B. Lee, P. R. Martin, and A. Valberg, "The physiological basis of heterochromatic flicker photometry demonstrated in the ganglion cells of the macaque retina," J. Physiol. (London) **404**, 323–347 (1988).
45. P. Lennie, J. Pokorny, and V. C. Smith, "Luminance," J. Opt. Soc. Am. A **10**, 1283–1293 (1993).
46. S. Chatterjee and E. Callaway, "S cone contributions to the magnocellular visual pathway in macaque monkey," Neuron **35**, 1135–1146 (2002).
47. S. G. Solomon and P. Lennie, "Chromatic gain controls in visual cortical neurons," J. Neurosci. **25**, 4779–4792 (2005).
48. R. L. De Valois, D. M. Snodderly, E. W. Yund, and N. K. Hepler, "Responses of macaque lateral geniculate cells to luminance and color figures," Sens Processes **1**, 244–259 (1977).
49. P. Lennie and M. D'Zmura, "Mechanisms of color vision," Crit. Rev. Neurobiol. **3**, 333–400 (1988).
50. R. L. De Valois and K. K. De Valois, "A multi-stage color model," Vision Res. **33**, 1053–1065 (1993).
51. D. M. Dacey and B. B. Lee, "The blue-on opponent pathway in the primate retina originates from a distinct bistratified ganglion cell," Nature **367**, 731–735 (1994).
52. D. M. Dacey, H. W. Liao, B. B. Peterson, F. R. Robinson, V. C. Smith, J. Pokorny, K. W. Yau, and P. D. Gamlin, "Melanopsin-expressing ganglion cells in primate retina signal colour and irradiance and project to the LGN," Nature **433**, 749–754 (2005).
53. J. Krauskopf, D. R. Williams, and D. W. Heeley, "Cardinal directions of color space," Vision Res. **22**, 1123–1131 (1982).
54. C. R. Ingling, Jr, "The spectral sensitivity of the opponent-colors channels," Vision Res. **17**, 1083–1090 (1977).
55. B. R. Wooten and J. S. Werner, "Short-wave cone input to the red–green opponent channel," Vision Res. **19**, 1053–1054 (1979).
56. F. M. de Monasterio, S. J. Schein, and E. P. McCrane, "Staining of blue-sensitive cones of the macaque retina by a fluorescent dye," Science **213**, 1278–1281 (1981).
57. A. Roorda and D. R. Williams, "The arrangement of the three cone classes in the living human eye," Nature **397**, 520–522 (1999).
58. D. H. Brainard, A. Roorda, Y. Yamauchi, J. B. Calderone, A. B. Metha, M. Neitz, J. Neitz, D. R. Williams, and G. H. Jacobs, "Functional consequences of the relative numbers of L and M cones," J. Opt. Soc. Am. A **17**, 607–614 (2000).
59. R. W. Rodieck, "Which cells code for color?" in *From Pigments to Perception*, A. Valberg and B. B. Lee, eds. (Plenum, 1991), pp. 83–94.
60. D. J. Calkins and P. Sterling, "Evidence that circuits for spatial and color vision segregate at the first retinal synapse," Neuron **24**, 313–321 (1999).
61. J. D. Mollon, "Uses and evolutionary origins of primate colour vision," in *Evolution of the Eye and Visual System*, J. Cronly-Dillon and R. L. Gregory, eds. (Macmillan, 1991), pp. 306–319.

62. P. Lennie, P. W. Haake, and D. R. Williams, "The design of chromatically opponent receptive fields," in *Computational Models of Visual Processing*, M. S. Landy and J. A. Movshon, eds. (MIT Press, 1991), pp. 71–82.
63. A. Roorda, A. B. Metha, P. Lennie, and D. R. Williams, "Packing arrangement of the three cone classes in primate retina," Vision Res. **41**, 1291–1306 (2001).
64. L. Diller, O. S. Packer, J. Verweij, M. J. McMahon, D. R. Williams, and D. M. Dacey, "L and M cone contributions to the midget and parasol ganglion cell receptive fields of macaque monkey retina," J. Neurosci. **24**, 1079–1088 (2004).
65. S. G. Solomon, B. B. Lee, A. J. White, L. Ruttiger, and P. R. Martin, "Chromatic organization of ganglion cell receptive fields in the peripheral retina," J. Neurosci. **25**, 4527–4539 (2005).
66. R. C. Reid and R. M. Shapley, "Space and time maps of cone photoreceptor signals in macaque lateral geniculate nucleus," J. Neurosci. **22**, 6158–6175 (2002).
67. D. M. Dacey, L. C. Diller, J. Verweij, and D. R. Williams, "Physiology of L- and M-cone inputs to H1 horizontal cells in the primate retina," J. Opt. Soc. Am. A **17**, 589–596 (2000).
68. S. G. Solomon and P. Lennie, "Spatial organization of L- and M-cone inputs to neurons in the macaque LGN," presented at the Vision Sciences Society Annual Meeting, Sarasota, Fla., May 6–11, 2005.
69. P. Lennie, J. Krauskopf, and G. Sclar, "Chromatic mechanisms in striate cortex of macaque," J. Neurosci. **10**, 649–669 (1990).
70. E. N. Johnson, M. J. Hawken, and R. Shapley, "The spatial transformation of color in the primary visual cortex of the macaque monkey," Nat. Neurosci. **4**, 409–416 (2001).
71. R. L. De Valois, N. P. Cottaris, S. D. Elfar, L. E. Mahon, and J. A. Wilson, "Some transformations of color information from lateral geniculate nucleus to striate cortex," Proc. Natl. Acad. Sci. U.S.A. **97**, 4997–5002 (2000).
72. R. G. Vautin and B. M. Dow, "Color cell groups in foveal striate cortex of the behaving macaque," J. Neurophysiol. **54**, 273–292 (1985).
73. T. Wachtler, T. J. Sejnowski, and T. D. Albright, "Representation of color stimuli in awake macaque primary visual cortex," Neuron **37**, 681–691 (2003).
74. D. C. Kiper, S. B. Fenstemaker, and K. R. Gegenfurtner, "Chromatic properties of neurons in macaque area V2," Visual Neurosci. **14**, 1061–1072 (1997).
75. K. R. Gegenfurtner, D. C. Kiper, and J. B. Levitt, "Functional properties of neurons in macaque area V3," J. Neurophysiol. **77**, 1906–1923 (1997).
76. H. Komatsu and Y. Ideura, "Relationships between color, shape, and pattern selectivities of neurons in the inferior temporal cortex of the monkey," J. Neurophysiol. **70**, 677–694 (1993).
77. S. A. Engel and C. S. Furmanski, "Selective adaptation to color contrast in human primary visual cortex," J. Neurosci. **21**, 3949–3954 (2001).
78. C. T. Tailby, S. G. Solomon, N. T. Dhruv, N. Majaj, and P. Lennie, "Habituation reveals cardinal chromatic mechanisms in striate cortex of macaque," presented at the Vision Sciences Society, Annual Meeting, Sarasotsa, Fla., May 6–11, 2005.
79. J. Rabin, E. Switkes, M. Crognale, M. E. Schneck, and A. J. Adams, "Visual evoked potentials in three-dimensional color space: correlates of spatio-chromatic processing," Vision Res. **34**, 2657–2671 (1994).
80. S. Engel, X. Zhang, and B. Wandell, "Color tuning in human visual cortex measured with functional magnetic resonance imaging," Nature **388**, 68–71 (1997).
81. J. Krauskopf, D. R. Williams, M. B. Mandler, and A. M. Brown, "Higher order color mechanisms," Vision Res. **26**, 23–32 (1986).
82. M. A. Webster and J. D. Mollon, "The influence of contrast adaptation on color appearance," Vision Res. **34**, 1993–2020 (1994).
83. A. Hanazawa, H. Komatsu, and I. Murakami, "Neural selectivity for hue and saturation of colour in the primary visual cortex of the monkey," Eur. J. Neurosci. **12**, 1753–1763 (2000).
84. N. P. Cottaris and R. L. De Valois, "Temporal dynamics of chromatic tuning in macaque primary visual cortex," Nature **395**, 896–900 (1998).
85. G. D. Horwitz, E. J. Chichilnisky, and T. D. Albright, "Blue–yellow signals are enhanced by spatiotemporal luminance contrast in macaque V1," J. Neurophysiol. **93**, 2263–2278 (2005).
86. G. J. C. van der Horst and M. A. Bouman, "Spatiotemporal chromaticity discrimination," J. Opt. Soc. Am. **59**, 1482–1488 (1969).
87. L. G. Thorell, R. L. De Valois, and D. G. Albrecht, "Spatial mapping of monkey V1 cells with pure color and luminance stimuli," Vision Res. **24**, 751–769 (1984).
88. S. G. Solomon, J. W. Peirce, and P. Lennie, "The impact of suppressive surrounds on chromatic properties of cortical neurons," J. Neurosci. **24**, 148–160 (2004).
89. S. S. Deeb, L. C. Diller, D. R. Williams, and D. M. Dacey, "Interindividual and topographical variation of L:M cone ratios in monkey retinas," J. Opt. Soc. Am. A **17**, 538–544 (2000).
90. M. S. Caywood, B. Willmore, and D. J. Tolhurst, "Independent components of color natural scenes resemble V1 neurons in their spatial and color tuning," J. Neurophysiol. **91**, 2859–2873 (2004).
91. B. R. Conway, "Spatial structure of cone inputs to color cells in alert macaque primary visual cortex (V-1)," J. Neurosci. **21**, 2768–2783 (2001).
92. B. R. Conway, D. H. Hubel, and M. S. Livingstone, "Color contrast in macaque V1," Cereb. Cortex **12**, 915–925 (2002).
93. E. N. Johnson, M. J. Hawken, and R. Shapley, "Cone inputs in macaque primary visual cortex," J. Neurophysiol. **91**, 2501–2514 (2004).
94. P. Monnier and S. K. Shevell, "Large shifts in color appearance from patterned chromatic backgrounds," Nat. Neurosci. **6**, 801–802 (2003).
95. S. X. Xian and S. K. Shevell, "Changes in color appearance caused by perceptual grouping," Visual Neurosci. **21**, 383–388 (2004).
96. N. W. Daw, "The psychology and physiology of colour vision," Trends Neurosci. **7**, 330–335 (1984).
97. S. M. Zeki, "Colour coding in the cerebral cortex: the responses of wavelength-selective and colour-coded cells in monkey visual cortex to changes in wavelength composition," Neuroscience (Oxford) **9**, 767–781 (1983).
98. D. Y. Ts'o and C. D. Gilbert, "The organization of chromatic and spatial interactions in the primate striate cortex," J. Neurosci. **8**, 1712–1727 (1988).
99. S. W. Kuffler, "Discharge patterns and functional organization of mammalian retina," J. Neurophysiol. **16**, 37–68 (1953).
100. C. Enroth-Cugell and J. G. Robson, "The contrast sensitivity of retinal ganglion cells of the cat," J. Physiol. (London) **187**, 517–552 (1966).
101. E. Kaplan and R. M. Shapley, "*X* and *Y* cells in the lateral geniculate nucleus of the macaque monkey," J. Physiol. (London) **330**, 125–144 (1982).
102. A. M. Derrington and P. Lennie, "Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque," J. Physiol. (London) **357**, 219–240 (1984).
103. L. J. Croner and E. Kaplan, "Receptive fields of P and M ganglion cells across the primate retina," Vision Res. **35**, 7–24 (1995).
104. S. Hochstein and R. M. Shapley, "Quantitative analysis of retinal ganglion cell classification," J. Physiol. (London) **262**, 237–264 (1976).
105. J. B. Levitt, R. A. Schumer, S. M. Sherman, P. D. Spear, and J. A. Movshon, "Visual response properties of neurons in the LGN of normally reared and visually deprived macaque monkeys," J. Neurophysiol. **85**, 2111–2129 (2001).

106. P. H. Schiller and J. G. Malpeli, "Functional specificity of lateral geniculate nucleus laminae of the rhesus monkey," J. Neurophysiol. **41**, 788–797 (1978).
107. J. R. H. Maunsell and J. R. Gibson, "Visual response latencies in striate cortex of the macaque monkey," J. Neurophysiol. **68**, 1332–1344 (1992).
108. M. J. Hawken, R. M. Shapley, and D. H. Grosof, "Temporal-frequency selectivity in monkey visual cortex," Visual Neurosci. **13**, 477–492 (1996).
109. R. M. Shapley and J. D. Victor, "The effect of contrast on the transfer properties of cat retinal ganglion cells," J. Physiol. (London) **285**, 275–298 (1978).
110. R. Shapley and J. D. Victor, "The contrast gain control of the cat retina," Vision Res. **19**, 431–434 (1979).
111. S. H. Hendry and R. C. Reid, "The koniocellular pathway in primate vision," Annu. Rev. Neurosci. **23**, 127–153 (2000).
112. J. A. Movshon, L. Kiorpes, M. J. Hawken, and J. R. Cavanaugh, "Functional maturation of the macaque's lateral geniculate nucleus," J. Neurosci. **25**, 2712–2722 (2005).
113. A. J. White, S. G. Solomon, and P. R. Martin, "Spatial properties of koniocellular cells in the lateral geniculate nucleus of the marmoset *Callithrix jacchus*," J. Physiol. (London) **533**, 519–535 (2001).
114. D. H. Hubel and T. N. Wiesel, "Laminar and columnar distribution of geniculo-cortical fibers in the macaque monkey," J. Comp. Neurol. **146**, 421–450 (1972).
115. J. O'Kusky and M. Colonnier, "A laminar analysis of the number of neurons, glia, and synapses in the visual cortex (area 17) of adult macaque monkeys," J. Comp. Neurol. **210**, 278–290 (1982).
116. G. G. Blasdel and D. Fitzpatrick, "Physiological organization of layer 4 in macque striate cortex," J. Neurosci. **4**, 880–895 (1984).
117. P. H. Schiller, B. L. Finlay, and S. F. Volman, "Quantitative studies of single cell properties in monkey striate cortex. III. Spatial frequency," J. Neurophysiol. **39**, 1334–1351 (1976).
118. R. L. De Valois, D. G. Albrecht, and L. G. Thorell, "Spatial frequency selectivity of cells in macaque visual cortex," Vision Res. **22**, 545–560 (1982).
119. J. M. Alonso, W. M. Usrey, and R. C. Reid, "Rules of connectivity between geniculate cells and simple cells in cat primary visual cortex," J. Neurosci. **21**, 4002–4015 (2001).
120. D. L. Ringach, "Haphazard wiring of simple receptive fields and orientation columns in visual cortex," J. Neurophysiol. **92**, 468–476 (2004).
121. R. E. Soodak, "The retinal ganglion cell mosaic defines orientation columns in striate cortex," Proc. Natl. Acad. Sci. U.S.A. **84**, 3936–3940 (1987).
122. R. M. Shapley and P. Lennie, "Spatial frequency analysis in the visual system," Annu. Rev. Neurosci. **8**, 547–583 (1985).
123. R. L. De Valois and K. K. De Valois, *Spatial Vision*, Oxford Psychology Series (Oxford U. Press, Oxford, 1988), p. 381.
124. R. L. De Valois, H. Morgan, and D. M. Snodderly, "Psychophysical studies of monkey vision. 3. Spatial luminance contrast sensitivity tests of macaque and human observers," Vision Res. **14**, 75–81 (1974).
125. D. C. Kiper, K. R. Gegenfurtner, and L. Kiorpes, "Spatial frequency channels in experimentally strabismic monkeys revealed by oblique masking," Vision Res. **35**, 2737–2742 (1995).
126. J. G. Robson, "Neural images: the physiological basis of spatial vision," in *Visual Coding and Adaptability*, C. S. Harris, ed. (Erlbaum, 1980), pp. 177–214.
127. D. G. Albrecht, R. L. De Valois, and L. G. Thorell, "Visual cortical neurons: Are bars or gratings the optimal stimuli?" Science **207**, 88–90 (1980).
128. B. A. Olshausen and D. J. Field, "Emergence of simple-cell receptive field properties by learning a sparse code for natural images," Nature **381**, 607–609 (1996).
129. A. J. Bell and T. J. Sejnowski, "The 'independent components' of natural scenes are edge filters," Vision Res. **37**, 3327–3339 (1997).
130. J. H. van Hateren and D. L. Ruderman, "Independent component analysis of natural image sequences yields spatio-temporal filters similar to simple cells in primary visual cortex," Proc. R. Soc. London, Ser. B **265**, 2315–2320 (1998).
131. E. Schwartz, R. B. Tootell, M. S. Silverman, E. Switkes, and R. L. De Valois, "On the mathematical structure of the visuotopic mapping of macaque striate cortex," Science **227**, 1065–1066 (1985).
132. M. A. Webster and R. L. De Valois, "Relationship between spatial-frequency and orientation tuning of striate-cortex cells," J. Opt. Soc. Am. A **2**, 1124–1132 (1985).
133. S. Mallat, *A Wavelet Tour of Signal Processing* (Academic, 1999).
134. D. J. Felleman and D. C. Van Essen, "Distributed hierarchical processing in the primate cerebral cortex," Cereb. Cortex **1**, 1–47 (1991).
135. K. S. Rockland and D. N. Pandya, "Laminar origins and terminations of cortical connections of the occipital lobe in the rhesus monkey," Brain Res. **179**, 3–20 (1979).
136. M. S. Livingstone and D. H. Hubel, "Segregation of form, color, movement, and depth: anatomy, physiology and perception," Science **240**, 740–749 (1988).
137. K. H. Foster, J. P. Gaska, M. Nagler, and D. A. Pollen, "Spatial and temporal frequency selectivity of neurones in visual cortical areas V1 and V2 of the macaque monkey," J. Physiol. (London) **365**, 331–363 (1985).
138. J. A. Movshon and W. T. Newsome, "Visual response properties of striate cortical neurons projecting to area MT in macaque monkeys," J. Neurosci. **16**, 7733–7741 (1996).
139. E. H. Adelson and J. R. Bergen, "Spatiotemporal energy models for the perception of motion," J. Opt. Soc. Am. A **2**, 284–299 (1985).
140. J. McLean and L. A. Palmer, "Contribution of linear spatiotemporal receptive field structure to velocity selectivity of simple cells in area 17 of cat," Vision Res. **29**, 675–680 (1989).
141. R. C. Reid, R. E. Soodak, and R. M. Shapley, "Directional selectivity and spatiotemporal structure of receptive fields of simple cells in cat striate cortex," J. Neurophysiol. **66**, 505–529 (1991).
142. D. J. Tolhurst and A. F. Dean, "Evaluation of a linear model of directional selectivity in simple cells of the cat's striate cortex," Visual Neurosci. **6**, 421–428 (1991).
143. M. S. Livingstone, "Mechanisms of direction selectivity in macaque V1," Neuron **20**, 509–526 (1998).
144. R. L. De Valois, N. P. Cottaris, L. E. Mahon, S. D. Elfar, and J. A. Wilson, "Spatial and temporal receptive fields of geniculate and cortical cells and directional selectivity," Vision Res. **40**, 3685–3702 (2000).
145. B. R. Conway and M. S. Livingstone, "Space–time maps and two-bar interactions of different classes of direction-selective cells in macaque V1," J. Neurophysiol. **89**, 2726–2742 (2003).
146. J. A. Movshon, E. H. Adelson, M. S. Gizzi, and W. H. Newsome, "The analysis of moving visual patterns," in *Pattern Recognition Mechanisms*, C. Chagas, R. Gatass, and C. Gross, eds. (Springer-Verlag, 1985), pp. 117–151.
147. C. C. Pack, M. S. Livingstone, K. R. Duffy, and R. T. Born, "End-stopping and the aperture problem: two-dimensional motion signals in macaque V1," Neuron **39**, 671–680 (2003).
148. C. Blakemore and F. W. Campbell, "On the existence of neurones in the human visual system selectively sensitive to the orientation and size of retinal images," J. Physiol. (London) **203**, 237–260 (1969).
149. L. Maffei, A. Fiorentini, and S. Bisti, "Neural correlate of perceptual adaptation to gratings," Science **182**, 1036–1038 (1973).
150. D. G. Albrecht, S. B. Farrar, and D. B. Hamilton, "Spatial contrast adaptation characteristics of neurones recorded

in the cat's visual cortex," J. Physiol. (London) **347**, 713–739 (1984).
151. I. Ohzawa, G. Sclar, and R. D. Freeman, "Contrast gain control in the cat's visual system," J. Neurophysiol. **54**, 651–667 (1985).
152. G. Sclar, P. Lennie, and D. D. DePriest, "Contrast adaptation in striate cortex of macaque," Vision Res. **29**, 747–755 (1989).
153. S. G. Solomon, J. W. Peirce, N. T. Dhruv, and P. Lennie, "Profound contrast adaptation early in the visual pathway," Neuron **42**, 155–162 (2004).
154. D. Chander and E. J. Chichilnisky, "Adaptation to temporal contrast in primate and salamander retina," J. Neurosci. **21**, 9904–9916 (2001).
155. A. Kohn and J. A. Movshon, "Adaptation changes the direction tuning of macaque MT neurons," Nat. Neurosci. **7**, 764–772 (2004).
156. J. A. Movshon and P. Lennie, "Pattern-selective adaptation in visual cortical neurones," Nature **278**, 850–852 (1979).
157. M. Carandini, H. B. Barlow, L. P. O'Keefe, A. B. Poirson, and J. A. Movshon, "Adaptation to contingencies in macaque primary visual cortex," Philos. Trans. R. Soc. London, Ser. B **352**, 1149–1154 (1997).
158. J. R. Müller, A. B. Metha, J. Krauskopf, and P. Lennie, "Rapid adaptation in visual cortex to the structure of images," Science **285**, 1405–1408 (1999).
159. V. Dragoi, J. Sharma, and M. Sur, "Adaptation-induced plasticity of orientation tuning in adult visual cortex," Neuron **28**, 287–298 (2000).
160. V. Dragoi, J. Sharma, E. K. Miller, and M. Sur, "Dynamics of neuronal sensitivity in visual cortex and local feature discrimination," Nat. Neurosci. **5**, 883–891 (2002).
161. M. Carandini and D. Ferster, "A tonic hyperpolarization underlying contrast adaptation in cat visual cortex," Science **276**, 949–952 (1997).
162. M. V. Sanchez-Vives, L. G. Nowak, and D. A. McCormick, "Membrane mechanisms underlying contrast adaptation in cat area 17 *in vivo*," J. Neurosci. **20**, 4267–4285 (2000).
163. F. S. Chance, S. B. Nelson, and L. F. Abbott, "Synaptic depression and the temporal response characteristics of V1 cells," J. Neurosci. **18**, 4785–4799 (1998).
164. J. A. Movshon, I. D. Thompson, and D. J. Tolhurst, "Spatial summation in the receptive fields of simple cells in the cat's striate cortex," J. Physiol. (London) **283**, 53–77 (1978).
165. J. A. Movshon, I. D. Thompson, and D. J. Tolhurst, "Receptive field organization of complex cells in the cat's striate cortex," J. Physiol. (London) **283**, 79–99 (1978).
166. J. P. Jones and L. A. Palmer, "The two-dimensional spatial structure of simple receptive fields in cat striate cortex," J. Neurophysiol. **58**, 1187–1211 (1987).
167. J. P. Jones, A. Stepnoski, and L. A. Palmer, "The two-dimensional spectral structure of simple receptive fields in cat striate cortex," J. Neurophysiol. **58**, 1212–1232 (1987).
168. G. C. DeAngelis, I. Ohzawa, and R. D. Freeman, "Spatiotemporal organization of simple-cell receptive fields in the cat's striate cortex. II. Linearity of temporal and spatial summation," J. Neurophysiol. **69**, 1118–1135 (1993).
169. H. Spitzer and S. Hochstein, "A complex-cell receptive field model," J. Neurophysiol. **53**, 1266–1286 (1985).
170. J. Touryan, B. Lau, and Y. Dan, "Isolation of relevant visual features from random stimuli for cortical complex cells," J. Neurosci. **22**, 10811–10818 (2002).
171. M. S. Livingstone and B. R. Conway, "Substructure of direction-selective receptive fields in macaque V1," J. Neurophysiol. **89**, 2743–2759 (2003).
172. N. C. Rust, O. Schwartz, J. A. Movshon, and E. P. Simoncelli, "Spatiotemporal elements of macaque V1 receptive fields," Neuron **46**, 945–956 (2005).
173. C. Chubb and G. Sperling, "Drift-balanced random stimuli: a general basis for studying non-Fourier motion perception," J. Opt. Soc. Am. A **5**, 1986–2007 (1988).
174. L. P. O'Keefe and J. A. Movshon, "Processing of first and second-order motion signals by neurons in area MT of the macaque monkey," Visual Neurosci. **15**, 305–317 (1998).
175. F. S. Chance, S. B. Nelson, and L. F. Abbott, "Complex cells as cortically amplified simple cells," Nat. Neurosci. **2**, 277–282 (1999).
176. L. Tao, M. Shelley, D. McLaughlin, and R. Shapley, "An egalitarian network model for the emergence of simple and complex cells in visual cortex," Proc. Natl. Acad. Sci. U.S.A., **101**, 366–371 (2004).
177. B. C. Skottun, R. S. De Valois, D. H. Grosof, J. A. Movshon, D. G. Albrecht, and A. B. Bonds, "Classifying simple and complex cells on the basis of response modulation," Vision Res. **31**, 1079–1086 (1991).
178. N. J. Priebe, F. Mechler, M. Carandini, and D. Ferster, "The contribution of spike threshold to the dichotomy of cortical simple and complex cells," Nat. Neurosci. **7**, 1113–1122 (2004).
179. K. K. De Valois, R. L. De Valois, and E. W. Yund, "Responses of striate cortex cells to grating and checkerboard patterns," J. Physiol. (London) **291**, 483–505 (1979).
180. D. G. Albrecht and D. B. Hamilton, "Striate cortex of monkey and cat: contrast response function," J. Neurophysiol. **48**, 217–237 (1982).
181. G. Sclar and R. D. Freeman, "Orientation selectivity in the cat's striate cortex is invariant with stimulus contrast," Exp. Brain Res. **46**, 457–461 (1982).
182. M. Carandini, D. J. Heeger, and J. A. Movshon, "Linearity and normalization in simple cells of the macaque primary visual cortex," J. Neurosci. **17**, 8621–8644 (1997).
183. O. D. Creutzfeldt, U. Kuhnt, and L. A. Benevento, "An intracellular analysis of visual cortical neurones to moving stimuli: response in a co-operative neuronal network," Exp. Brain Res. **21**, 251–274 (1974).
184. A. M. Sillito, "The contribution of inhibitory mechanisms to the receptive field properties of neurones in the striate cortex of the cat," J. Physiol. (London) **250**, 305–329 (1975).
185. K. K. De Valois and R. B. H. Tootell, "Spatial-frequency-specific inhibition in cat striate cortex cells," J. Physiol. (London) **336**, 359–376 (1983).
186. A. B. Bonds, "Role of inhibition in the specification of orientation-selectivity of cells in the cat striate cortex," Visual Neurosci. **2**, 41–55 (1989).
187. G. C. DeAngelis, J. G. Robson, I. Ohzawa, and R. D. Freeman, "Organization of suppression in receptive fields of neurons in cat visual cortex," J. Neurophysiol. **68**, 144–163 (1992).
188. D. G. Albrecht, "Visual cortex neurons in monkey and cat: effect of contrast on the spatial and temporal phase transfer functions," Visual Neurosci. **12**, 1191–1210 (1995).
189. D. G. Albrecht, W. S. Geisler, R. A. Frazor, and A. M. Crane, "Visual cortex neurons of monkeys and cats: temporal dynamics of the contrast response function," J. Neurophysiol. **88**, 888–913 (2002).
190. W. S. Geisler and D. G. Albrecht, "Cortical neurons: isolation of contrast gain control," Vision Res. **32**, 1409–1410 (1992).
191. D. J. Heeger, "Normalization of cell responses in cat striate cortex," Visual Neurosci. **9**, 181–197 (1992).
192. J. D. Allison, K. R. Smith, and A. B. Bonds, "Temporal-frequency tuning of cross-orientation suppression in the cat striate cortex," Visual Neurosci. **18**, 941–948 (2001).
193. T. C. Freeman, S. Durand, D. C. Kiper, and M. Carandini, "Suppression without inhibition in visual cortex," Neuron **35**, 759–771 (2002).
194. M. Carandini, D. J. Heeger, and W. Senn, "A synaptic explanation of suppression in visual cortex," J. Neurosci. **22**, 10053–10065 (2002).
195. T. W. Troyer, A. E. Krukowski, N. J. Priebe, and K. D. Miller, "Contrast-invariant orientation tuning in cat visual cortex: thalamocortical input tuning and correlation-based intracortical connectivity," J. Neurosci. **18**, 5908–5927 (1998).

196. M. Carandini and D. Ferster, "Membrane potential and firing rate in cat primary visual cortex," J. Neurosci. **20**, 470–484 (2000).
197. J. S. Anderson, I. Lampl, D. C. Gillespie, and D. Ferster, "The contribution of noise to contrast invariance of orientation tuning in cat visual cortex," Science **290**, 1968–1972 (2000).
198. D. J. Heeger, "Half-squaring in responses of cat striate cells," Visual Neurosci. **9**, 427–443 (1992).
199. D. J. Tolhurst and D. J. Heeger, "Comparison of contrast-normalization and threshold models of the responses of simple cells in cat striate cortex," Visual Neurosci. **14**, 293–309 (1997).
200. G. Sclar, J. H. R. Maunsell, and P. Lennie, "Coding of image contrast in central visual pathways of the macaque monkey," Vision Res. **30**, 1–10 (1990).
201. D. J. Tolhurst and D. J. Heeger, "Contrast normalization and a linear model for the directional selectivity in simple cells in cat striate cortex," Visual Neurosci. **14**, 19–25 (1997).
202. J. A. Hirsch, J. M. Alonso, R. C. Reid, and L. M. Martinez, "Synaptic integration in striate cortical simple cells," J. Neurosci. **18**, 9517–9528 (1998).
203. I. Lampl, J. S. Anderson, D. C. Gillespie, and D. Ferster, "Prediction of orientation selectivity from receptive field architecture in simple cells of cat visual cortex," Neuron **30**, 263–274 (2001).
204. N. J. Priebe and D. Ferster, "Direction selectivity of excitation and inhibition in simple cells of the cat primary visual cortex," Neuron **45**, 133–145 (2005).
205. C. Beaulieu and M. Colonnier, "A comparison of the number of neurons in individual laminae of cortical areas 17, 18 and posteromedial suprasylvian (PMLS) area in the cat," Brain Res. **339**, 166–170 (1985).
206. S. Hendry and R. K. Carder, "Organization and plasticity of GABA neurons and receptors in monkey visual cortex," Prog. Brain Res. **90**, 477–502 (1992).
207. D. J. Tolhurst and A. F. Dean, "The effects of contrast on the linearity of spatial summation of simple cells in the cat's striate cortex," Exp. Brain Res. **79**, 582–588 (1990).
208. P. Heggelund, "Receptive field organization of complex cells in cat striate cortex," Exp. Brain Res. **42**, 90–107 (1981).
209. L. A. Palmer and T. L. Davis, "Receptive-field structure in cat striate cortex," J. Neurophysiol. **46**, 260–276 (1981).
210. D. Ferster, "Spatially opponent excitation and inhibition in simple cells of the cat visual cortex," J. Neurosci. **8**, 1172–1180 (1988).
211. L. J. Borg-Graham, C. Monier, and Y. Frégnac, "Visual input evokes transient and strong shunting inhibition in visual cortical neurons," Nature **393**, 369–372 (1998).
212. S. Nelson, L. Toth, B. Sheth, and M. Sur, "Orientation selectivity of cortical neurons during intracellular blockade of inhibition," Science **265**, 774–777 (1994).
213. R. L. De Valois, E. W. Yund, and N. Helper, "The orientation and direction selectivity of cells in macaque visual cortex," Vision Res. **22**, 531–544 (1982).
214. D. L. Ringach, M. J. Hawken, and R. Shapley, "Dynamics of orientation tuning in macaque primary visual cortex," Nature **387**, 281–284 (1997).
215. D. L. Ringach, M. J. Hawken, and R. Shapley, "Dynamics of orientation tuning in macaque V1: the role of global and tuned suppression," J. Neurophysiol. **90**, 342–352 (2003).
216. J. A. Mazer, W. E. Vinje, J. McDermott, P. H. Schiller, and J. L. Gallant, "Spatial frequency and orientation tuning dynamics in area V1," Proc. Natl. Acad. Sci. U.S.A. **99**, 1645–1650 (2002).
217. D. Xing, R. M. Shapley, M. J. Hawken, and D. L. Ringach, "The effect of stimulus size on the dynamics of orientation selectivity in Macaque V1," J. Neurophysiol. **94**, 799–812 (2005).
218. V. Braitenberg and A. Schütz, *Cortex: Statistics and Geometry of Neuronal Connectivity*, 2nd ed. (Springer, 1998).
219. R. J. Douglas and K. A. Martin, "A functional microcircuit for cat visual cortex," J. Physiol. (London) **440**, 735–769 (1991).
220. R. J. Douglas, C. Koch, M. Mahowald, K. A. C. Martin, and H. H. Suarez, "Recurrent excitation in neocortical circuits," Science **269**, 981–985 (1995).
221. R. Ben-Yishai, R. L. Bar-Or, and H. Sompolinsky, "Theory of orientation tuning in visual cortex," Proc. Natl. Acad. Sci. U.S.A. **92**, 3844–3848 (1995).
222. D. C. Somers, S. B. Nelson, and M. Sur, "An emergent model of orientation selectivity in cat visual cortical simple cells," J. Neurosci. **15**, 5448–5465 (1995).
223. P. Adorjan, J. B. Levitt, J. S. Lund, and K. Obermayer, "A model for the intracortical origin of orientation preference and tuning in macaque striate cortex," Visual Neurosci. **16**, 303–318 (1999).
224. D. McLaughlin, R. Shapley, M. Shelley, and D. J. Wielaard, "A neuronal network model of macaque primary visual cortex (V1): orientation selectivity and dynamics in the input layer 4Calpha," Proc. Natl. Acad. Sci. U.S.A. **97**, 8087–8092 (2000).
225. X. Pei, T. R. Vidyasagar, M. Volgushev, and O. D. Creutzfeldt, "Receptive field analysis and orientation selectivity of postsynaptic potentials of simple cells in cat visual cortex," J. Neurosci. **14**, 7130–7140 (1994).
226. D. Ferster, S. Chung, and H. Wheat, "Orientation selectivity of thalamic input to simple cells of cat visual cortex," Nature **380**, 249–252 (1996).
227. S. Chung and D. Ferster, "Strength and orientation tuning of the thalamic input to simple cells revealed by electrically evoked cortical suppression," Neuron **20**, 1177–1189 (1998).
228. B. Dreher, "Hypercomplex cells in the cat's striate cortex," Invest. Ophthalmol. **11**, 355–356 (1972).
229. C. D. Gilbert, "Laminar differences in receptive field properties of cells in cat primary visual cortex," J. Physiol. (London) **268**, 391–421 (1977).
230. M. K. Kapadia, M. Ito, C. D. Gilbert, and G. Westheimer, "Improvement in visual sensitivity by changes in local context: parallel studies in human observers and in V1 of alert monkeys," Neuron **15**, 843–856 (1995).
231. J. B. Levitt and J. S. Lund, "Contrast dependence of contextual effects in primate visual cortex," Nature **387**, 73–76 (1997).
232. J. R. Cavanaugh, W. Bair, and J. A. Movshon, "Nature and interaction of signals from the receptive field center and surround in macaque V1 neurons," J. Neurophysiol. **88**, 2530–2546 (2002).
233. J. I. Nelson and B. J. Frost, "Intracortical facilitation among co-oriented, co-axially aligned simple cells in cat striate cortex," Exp. Brain Res. **61**, 54–61 (1985).
234. G. C. DeAngelis, R. D. Freeman, and I. Ohzawa, "Length and width tuning of neurons in the cat's primary visual cortex," J. Neurophysiol. **71**, 347–374 (1994).
235. R. L. De Valois, L. G. Thorell, and D. G. Albrecht, "Periodicity of striate-cortex-cell receptive fields," J. Opt. Soc. Am. A **2**, 1115–1123 (1985).
236. J. J. Knierim and D. C. Van Essen, "Neuronal responses to static texture patterns in area V1 of the alert macaque monkey," J. Neurophysiol. **67**, 961–980 (1992).
237. A. M. Sillito, K. L. Grieve, H. E. Jones, J. Cudeiro, and J. Davis, "Visual cortical mechanisms detecting focal orientation discontinuities," Nature **378**, 492–496 (1995).
238. M. P. Sceniak, D. L. Ringach, M. J. Hawken, and R. Shapley, "Contrast's effect on spatial summation by macaque V1 neurons," Nat. Neurosci. **2**, 733–739 (1999).
239. M. P. Sceniak, M. J. Hawken, and R. Shapley, "Contrast-dependent changes in spatial frequency tuning of macaque V1 neurons: effects of a changing receptive field size," J. Neurophysiol. **88**, 1363–1373 (2002).
240. J. R. Cavanaugh, W. Bair, and J. A. Movshon, "Selectivity and spatial distribution of signals from the receptive field surround in macaque V1 neurons," J. Neurophysiol. **88**, 2547–2556 (2002).
241. J. R. Müller, A. B. Metha, J. Krauskopf, and P. Lennie,

"Local signals from beyond the receptive fields of striate cortical neurons," J. Neurophysiol. **90**, 822–831 (2003).
242. K. Zipser, V. A. F. Lamme, and P. H. Schiller, "Contextual modulation in primary visual cortex," J. Neurosci. **16**, 7376–7389 (1996).
243. F. Ratliff, "Contour and contrast," Sci. Am. **226**, 91–101 (1972).
244. O. Schwartz and E. P. Simoncelli, "Natural signal statistics and sensory gain control," Nat. Neurosci. **4**, 819–825 (2001).
245. W. Bair, J. R. Cavanaugh, and J. A. Movshon, "Time course and time-distance relationships for surround suppression in macaque V1 neurons," J. Neurosci. **23**, 7690–7701 (2003).
246. A. Angelucci, J. B. Levitt, E. J. Walton, J. M. Hupe, J. Bullier, and J. S. Lund, "Circuits for local and global signal integration in primary visual cortex," J. Neurosci. **22**, 8633–8646 (2002).
247. S. G. Solomon, A. J. White, and P. R. Martin, "Extraclassical receptive field properties of parvocellular, magnocellular, and koniocellular cells in the primate lateral geniculate nucleus," J. Neurosci. **22**, 338–349 (2002).
248. M. Colonnier, "The organizing principles of the primary visual cortex in monkey," in *Cerebral Cortex*, A. Peters and E. G. Jones, eds. (Plenum, 1985).
249. J. S. Lund, M. J. Hawken, and A. J. Parker, "Local circuit neurons of macaque monkey striate cortex: II. Neurons of laminae 5B and 6," J. Comp. Neurol. **276**, 1–29 (1988).
250. E. M. Callaway, "Local circuits in primary visual cortex of the macaque monkey," Annu. Rev. Neurosci. **21**, 47–74 (1998).
251. M. J. Hawken, A. J. Parker, and J. S. Lund, "Laminar organization and contrast sensitivity of direction-selective cells in the striate cortex of the old world monkey," J. Neurosci. **8**, 3541–3548 (1988).
252. N. H. Yabuta, A. Sawatari, and E. M. Callaway, "Two functional channels from primary visual cortex to dorsal visual cortical areas," Science **292**, 297–300 (2001).
253. R. B. H. Tootell, M. S. Silverman, S. L. Hamilton, R. L. De Valois, and E. Switkes, "Functional anatomy of macaque striate cortex III. Color," J. Neurosci. **8**, 1569–1593 (1988).
254. J. J. Malpeli, P. Schiller, and C. L. Colby, "Response properties of single cells in monkey striate cortex during reversible inactivation of individual lateral geniculate laminae," J. Neurophysiol. **46**, 1102–1119 (1981).
255. J. D. Allison, P. Melzer, Y. Ding, A. B. Bonds, and V. A. Casagrande, "Differential contributions of magnocellular and parvocellular pathways to the contrast response of neurons in bush baby primary visual cortex (V1)," Visual Neurosci. **17**, 71–76 (2000).
256. J. C. Horton, "Cytochrome oxidase patches: a new cytoarchitectonic feature of monkey visual cortex," Philos. Trans. R. Soc. London, Ser. B **304**, 199–253 (1984).
257. E. W. Carroll and M. T. Wong-Riley, "Quantitative light and electron microscopic analysis of cytochrome oxidase-rich zones in the striate cortex of the squirrel monkey," J. Comp. Neurol. **222**, 1–17 (1984).
258. M. S. Livingstone and D. H. Hubel, "Anatomy and physiology of a color system in the primate visual cortex," J. Neurosci. **4**, 309–356 (1984).
259. C. E. Landisman and D. Y. Ts'o, "Color processing in macaque striate cortex: electrophysiological properties," J. Neurophysiol. **87**, 3138–3151 (2002).
260. A. G. Leventhal, K. G. Thompson, D. Liu, Y. Zhou, and S. J. Ault, "Concomitant sensitivity to orientation, direction, and color of cells in layers 2, 3, and 4 of monkey striate cortex," J. Neurosci. **15**, 1808–1818 (1995).
261. T. Yoshioka, J. B. Levitt, and J. S. Lund, "Independence and merger of thalamocortical channels within macaque monkey primary visual cortex: anatomy of interlaminar projections," Visual Neurosci. **11**, 467–489 (1994).
262. N. H. Yabuta and E. M. Callaway, "Functional streams and local connections of layer 4C neurons in primary visual cortex of the macaque monkey," J. Neurosci. **18**, 9489–9499 (1998).
263. E. M. Callaway and A. K. Wiser, "Contributions of individual layer 2-5 spiny neurons to local circuits in macaque primary visual cortex," Visual Neurosci. **13**, 907–922 (1996).
264. E. A. Lachica and V. A. Casagrande, "Direct W-like geniculate projections to the cytochrome oxidase (CO) blobs in primate visual cortex: axon morphology," J. Comp. Neurol. **319**, 141–158 (1992).
265. C. E. Landisman and D. Y. Ts'o, "Color processing in macaque striate cortex: relationships to ocular dominance, cytochrome oxidase, and orientation," J. Neurophysiol. **87**, 3126–3137 (2002).
266. M. S. Silverman, D. H. Grosof, R. L. De Valois, and S. D. Elfar, "Spatial-frequency organization in primate striate cortex," Proc. Natl. Acad. Sci. U.S.A. **86**, 711–715 (1989).
267. D. P. Edwards, K. P. Purpura, and E. Kaplan, "Contrast sensitivity and spatial frequency response of primate cortical neurons in and around the cytochrome oxidase blobs," Vision Res. **35**, 1501–1523 (1995).
268. D. J. Field, "Relations between the statistics of natural images and the response properties of cortical cells," J. Opt. Soc. Am. A **4**, 2379–2394 (1987).
269. B. M. Dow and R. G. Vautin, "Horizontal segregation of color information in the middle layers of foveal striate cortex," J. Neurophysiol. **57**, 712–739 (1987).
270. R. Durbin and G. Mitchison, "A dimension reduction framework for understanding cortical maps," Nature **343**, 644–647 (1990).
271. D. H. Hubel and T. N. Wiesel, "The Ferrier lecture. Functional architecture of macaque monkey visual cortex," Proc. R. Soc. London, Ser. B **198**, 1–59 (1977).
272. M. Shadlen and W. T. Newsome, "The variable discharge rate of cortical neurons: implications for connectivity, computation, and information coding," J. Neurosci. **18**, 3870–3896 (1998).
273. I. Lampl, I. Reichova, and D. Ferster, "Synchronous membrane potential fluctuations in neurons of the cat visual cortex," Neuron **22**, 361–374 (1999).
274. D. A. Leopold and N. K. Logothetis, "Spatial patterns of spontaneous local field activity in the monkey visual cortex," Rev. Neurosci. **14**, 195–205 (2003).
275. W. R. Garner, *The Processing of Information and Structure* (Erlbaum, 1974).
276. P. Lennie, "Single units and visual cortical organization," Perception **27**, 889–935 (1998).
277. G. C. DeAngelis, I. Ohzawa, and R. D. Freeman, "Receptive-field dynamics in the central visual pathways," Trends Neurosci. **18**, 451–458 (1995).