Cone receptive field in cat retina computed from microcircuitry

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Abstract
The receptive-field profile of the cone in cat retina was computed. The computation was based on (1) the known anatomical circuit connecting cones via narrow-field bipolar cells to the on-beta ganglion cell; (2) the known physiological receptive-field profile of the on-beta (X) cell at the corresponding eccentricity; and (3) a model in which the beta receptive field arises by linear superposition of cone receptive fields. The computed cone receptive field has a center/surround organization with a center almost as broad as that of the beta cell center. The cone surround is comparably broad to that of the beta cell but somewhat lower in peak amplitude. The problems to which the center/surround receptive field are the solution, namely, signal compression and noise reduction, apparently must be solved before the first synapse of the visual pathway.

Keywords: Retina, Cone, Ganglion cell, Receptive field, Computation

Introduction
A cone at the level of the outer segment samples a small region of visual space. The aperture of a cone in the central area of cat retina is only about 0.01 deg, but since the image that it sampled is expanded by optical blur (Robson & Enroth-Cugell, 1978; Wässle, 1971), the sampled region ("receptive field") is more like 0.1 deg. Once the signal representing this tiny spatial sample reaches the cone axon terminal ("pedicle"), it is apparently integrated over a much wider area with the signals of many other cones. One mechanism for this is the lateral spread of photocurrents through electrical synapses between cone pedicles (Raviola & Gilula, 1975; Kolb, 1977; Smith et al., 1986); another is the negative feedback into the pedicle of synaptic currents generated by horizontal cells, which are GABAergic (Chun & Wässle, 1989; Sarthy & Fu, 1989). These mechanisms imply for the cone pedicle a receptive field with a rather broad center and a still broader, antagonistic surround. Physiological evidence for such an organization in cold-blooded vertebrates is ample (e.g., Baylor et al., 1971; Piccolino et al., 1981), but from cat cones in situ there are only a few recordings (Nelson, 1977).

One way to estimate the receptive field of the cone pedicle is to work backward from the receptive field of the beta (X) ganglion cell. This cell responds linearly (Rodieck, 1965; Enroth-Cugell & Robson, 1966), so one can model the beta cell receptive field as a linear superposition of cone pedicle receptive fields. A recent study has found the number, spacing, and synaptic weighting of cone pedicles that converge via bipolar cells onto a beta cell at 1-deg eccentricity (Cohen & Sterling, 1990a). Comparing this circuitry to measurements of the beta cell's photopic receptive field at this eccentricity (Cleland et al., 1979; Linsenmeier et al., 1982), we computed (by deconvolution) the receptive field of a single cone.

Methods
Anatomical circuit
The circuit illustrated in Fig. 1 summarizes an anatomical study (Cohen & Sterling, 1990a) of three on-beta ganglion cells that were adjacent to each other in a patch of retina at 1-deg eccentricity. The circuit includes 30 cones, spaced 6 μm apart, converging via chemical synapses onto seven b1 bipolar cells, which are narrow-field and closely spaced. The b2 axons mingle with the dendritic branches of the beta cell and form synapses. The field of cones so connected to the beta cell is about 45 μm diameter, broader than the 32 μm span of the beta dendritic tree. This is due to the lateral extension of bipolar processes.

It is established that the dendrites of adjacent b1 cells completely "tile" the cone pedicle array such that each pedicle contributes to at least one and often two b1 cells (Cohen & Sterling, 1990c). The number of synapses contributed by each cone converging on a given bipolar cell is unknown. However, for our present purpose, to determine the overall anatomical weighting function at the level of the ganglion cell, this number is of relatively little consequence. The reason is that in convolving two Gaussian weighting functions (see below) the width of the result is related to the sum of the squares of the compo-
nent functions (see Discussion). Since the effective span of the beta cell is 45 μm and that of a b2 cell is only 15 μm (Sterling et al., 1988; Cohen & Sterling, 1990c), it matters little to the final result whether the cone → bipolar weighting is flat (as we treat it here) or bumpy as it could conceivably be.

The b2 cell appears to provide the major excitatory input to on-beta cells (Cohen & Sterling, 1990a; McGuire et al., 1986). Typically, the b2 bipolar cells provide a beta cell with about half of its bipolar input and one-third of its total input (Cohen & Sterling, 1990a). Two other types of bipolar cell that innervate the ganglion cell, called b3 and b5, have dendritic fields co-spatial with the b1 array, and they receive input from approximately the same cones as the b1 array (Cohen & Sterling, 1990b, c). About 30% of the synapses on the beta cell are amacrine (Kolb, 1979; Sterling et al., 1988). The amacrine cells so far associated with the beta cell are narrow-field (Kolb & Nelson, 1984), and they probably collect from narrow-field bipolar cells. The chain of reasoning for this is that the only wide-field bipolar cell that innervates sublamina b apparently collects exclusively from blue cones (Cohen & Sterling, 1990c). The beta cell does not connect to blue cones (Cleland & Levick, 1974). Therefore, the amacrine cells that innervate the on beta must receive their input from bipolar cells other than the wide-field cell. Thus, it appears that all of the anatomical pathways from the outer plexiform layer to the on-beta cell descend from the immediately overlying cones.

**Weighting function**

Bipolar axons near the center of the ganglion cell dendritic field tend to provide many synapses, and the axons near the edge of the dendritic field tend to provide only a few (Cohen & Sterling, 1990a). This connection pattern, representing the relative strengths of convergent cone signals, creates a rather bumpy "weighting function" at the ganglion cell. To smooth this weighting function, we convolved it with a 22-μm diameter optical blur function (Robson & Enroth-Cugell, 1978; Wässle, 1971) and determined what Gaussian weighting function would produce the same result after optical blur.

Beta ganglion cells located at 1-deg eccentricity have short, stout dendrites (~30 μm dendritic tree diameter) (Boycott & Wässle, 1974; Kolb et al., 1981; McGuire et al., 1986; Cohen & Sterling, 1990a). Therefore, electronictic decay along the dendrite is essentially nil, and all synapses are electrotonically equivalent (Koch et al., 1982). However, beta cells located more peripherally have progressively longer, tapered dendrites, and thus probably have significant electronictic decay (Boycott & Wässle, 1974; Kolb et al., 1981; Rall, 1967; Creutzfeldt et al., 1970). Indeed, we found it necessary to include weighting functions for peripheral cells in order to produce good matches between the computed beta receptive fields and X cell receptive fields measured physiologically. Assuming Rm = 2500 Ω·cm² and Rl = 100 Ω·cm, a 0.5-μm diameter dendrite has a space constant of 176 μm. To include the effects of dendritic taper and soma loading, we used Gaussian weighting functions for beta cells at 10- and 20-deg eccentricity of, respectively, 90% and 83% of the dendritic tree diameters.

**Representation of ganglion cell receptive field**

The ganglion cell's fundamental response has been modeled as a "difference-of-Gaussians" (Fig. 1b; Rodieck, 1965; Enroth-Cugell & Robson, 1966; Linsenmeier et al., 1982; Derrington & Lennie, 1982; Frishman et al., 1987) This model is described by the equation:

\[
\text{Response} = \text{Center Gaussian} - \text{Surround Gaussian} = kc \exp\left(-\frac{(r/rc)^2}{2}\right) - ks \exp\left(-\frac{(r/rs)^2}{2}\right),
\]

(1)

where

\[
rc = \text{center radius},
\]

\[
ks = \text{surround amplitude},
\]

\[
rs = \text{surround radius}.
\]

To represent a beta cell's receptive field by this equation, we chose a value for the center radius rc and multiplied this by a constant value to derive the surround radius rs. The actual ratio rs/rc varies between 2–8 (Linsenmeier et al., 1982), so we chose 5 as an intermediate value. The center amplitude kc was always set at 1.0, and surround amplitude ks was determined by the value assumed (normally between 0.7 and 1.0) for the surround/center balance b:

\[
ks = b \cdot kc \cdot \frac{rc^2}{rs^2}.
\]

Therefore, for rs/rc = 5 and b = 1.0, the surround amplitude was 4% of kc. Final receptive-field amplitude was normalized to give a peak (center) amplitude of 1.0 after the subtraction of the surround from the center. This facilitated comparisons between receptive-field profiles.

**Computing the cone receptive field**

In the computations to follow, we derive the cone receptive field assuming linear summation (see Introduction). Linear summation implies that the surround/center balances b are equal in cones and ganglion cells. Therefore, we fixed b for cone and ganglion cell receptive fields and so reduced the number of free parameters used in calculating ks from 3 to 2 (rc, rs).

The computation was similar in effect to a deconvolution of the ganglion cell receptive-field profile by its anatomical connection profile. Instead of actually deconvolving, we determined the cone receptive field by a method of successive approximation based on convolution. The method, illustrated in Fig. 1 (right side) is as follows: (1) Estimate a cone receptive field using the difference-of-Gaussians eqn. (1) above. (2) Use the estimate as a template and copy it to make a two-dimensional array of cone receptive fields, spaced appropriately. (3) Multiply each cone receptive field by the cone's convergence weight (specified by the Gaussian weighting function given in Table 1) and add the result (displaced laterally according to the cone's location) to obtain a computed beta cell receptive field. (4) Compare the extents and amplitudes of the computed beta center and surround with the actual physiological data and then revise the estimate of the cone receptive field. (5) Repeat steps 1–4 until the computed and measured ganglion cell receptive fields match (Fig. 2). The final cone template arrived at in step 1 was the computed cone receptive field (Fig. 2, dashed line). The cone center diameter was matched to the nearest integer (in microns), and the surround was an integral multiple of the center.
Fig. 1. (a) Anatomic convergence of cones to ganglion cells. Thirty cones converge through seven b, bipolars to on-beta ganglion cell (from Cohen & Sterling, 1990a). (b) Method of deconvolution used to compute cone receptive field from beta receptive field. An initial cone receptive-field template was copied 30 times. Each copy was multiplied by its synaptic weight, spatially offset, and summed at the ganglion cell.

Results

Cone receptive field in area centralis

Since our computations relate the beta ganglion cell’s receptive field to its anatomical circuit, and since both vary markedly with eccentricity, it is obviously critical to compare them at the same retinal locus. The variation of the photopic on-beta receptive field with eccentricity has been well-studied (Peichl & Wässle, 1979; Cleland et al., 1979; Linsenmeier et al., 1982). Cleland et al. (1979) paid the most detailed attention to receptive fields in the area centralis. They located the center of this region as the intersection of the vertical and horizontal meridians and studied many cells (42) in the same animal, so variability at each eccentricity could be assessed. Typically, 90% of the data points fell within ±1/4 octave, which corresponds to variation of ±20% in the receptive-field center size at a given eccentricity.

The average receptive-field center diameter of eight cells reported by Cleland et al. (1979) at 1 ± 0.5 deg was 72 μm. These measurements were made with a bright bar extending across both center and surround, and the boundary between center and surround was defined to be the response that equalled the maintained firing rate. The “center” diameter measured this way approximates the zero-crossing width in a difference-of-Gaussians model. It was no surprise, then, turning to the measurements of Linsenmeier et al. (1982), to find that where the two studies overlap (8–10 deg), the Cleland et al. measurements of the “center” are larger by a factor of 1.3 (see Discussion). Therefore, to obtain rc at 1-deg eccentricity, we divided the Cleland et al. measurements at 1 deg by this factor. Thus corrected, rc for the on-beta ganglion cell at 1 deg was 28 μm. Assuming a rs/rc ratio of 5 (see Methods), the surround radius was 140 μm with peak amplitude at 4%.

The extent of the computed cone center rc for this set of beta cell parameters was 25 μm, about 90% that of the ganglion cell center (Fig. 2; Table 1). This seems at first counterintuitive, especially since one is accustomed to thinking of the cone receptive field as the width of the optical blurring function. However, the result is a direct consequence of the mismatch observed
Table 1. Comparisons of beta ganglion cell and derived cone receptive fields

<table>
<thead>
<tr>
<th></th>
<th>1 deg</th>
<th>(1-deg Ref)</th>
<th>10 deg</th>
<th>20 deg</th>
<th>(10-20 deg Ref)</th>
</tr>
</thead>
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<tr>
<td>X ganglion cell center diameter</td>
<td>56 μm</td>
<td>a,b</td>
<td>100 μm</td>
<td>150 μm</td>
<td>56 μm</td>
</tr>
<tr>
<td>Surround diameter</td>
<td>280 μm</td>
<td></td>
<td>500 μm</td>
<td>750 μm</td>
<td>280 μm</td>
</tr>
<tr>
<td>Surround amplitude</td>
<td>4 %</td>
<td></td>
<td>4 %</td>
<td>4 %</td>
<td>4 %</td>
</tr>
<tr>
<td>Beta dendritic arbor diameter</td>
<td>30 μm</td>
<td>d,e</td>
<td>100 μm</td>
<td>168 μm</td>
<td>100 μm</td>
</tr>
<tr>
<td>Weighting function diameter</td>
<td>30 μm</td>
<td></td>
<td>90 μm</td>
<td>140 μm</td>
<td>30 μm</td>
</tr>
<tr>
<td>cones converging</td>
<td>30</td>
<td></td>
<td>69</td>
<td>113</td>
<td>30</td>
</tr>
<tr>
<td>Cone center diameter</td>
<td>50 μm</td>
<td></td>
<td>66 μm</td>
<td>98 μm</td>
<td>50 μm</td>
</tr>
<tr>
<td>Surround diameter</td>
<td>280 μm</td>
<td></td>
<td>528 μm</td>
<td>784 μm</td>
<td>280 μm</td>
</tr>
<tr>
<td>Surround amplitude</td>
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<td></td>
<td>1.6 %</td>
<td>1.6 %</td>
<td>3.2 %</td>
</tr>
<tr>
<td>Spacing</td>
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<td>h</td>
<td>12 μm</td>
<td>14 μm</td>
<td>6 μm</td>
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<tr>
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<td></td>
<td>39 %</td>
<td>39 %</td>
<td>80 %</td>
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<tr>
<td>b1 bipolar center diameter</td>
<td>52 μm</td>
<td></td>
<td></td>
<td></td>
<td>52 μm</td>
</tr>
<tr>
<td>Surround diameter</td>
<td>280 μm</td>
<td></td>
<td></td>
<td></td>
<td>280 μm</td>
</tr>
<tr>
<td>Surround amplitude</td>
<td>3.4 %</td>
<td></td>
<td></td>
<td></td>
<td>3.4 %</td>
</tr>
</tbody>
</table>

All center and surround diameters measured at 1/e of peak amplitude. Center amplitudes normalized to 1.0 after center/surround summation.

a Linsenmeier et al., 1982.
b Cleland et al., 1979.
c Derrington & Lennie, 1982.
e McGuire et al., 1986.
f Boycott & Wässle, 1974.
g Kolb et al., 1981.
h Smith et al., 1986.
i Steinberg et al., 1973.

for the beta cell between the dimensions of anatomical circuit and the receptive field (Cohen & Sterling, 1990a).

The extent of the computed cone surround rs was 140 μm, and the surround amplitude ks was 3.2%. The amplitude of the cone surround was thus 80% that of the ganglion cell surround (Table 1). The reason why the amplitude of the beta surround is greater than that of the cone (when their centers are normalized to 1.0) is that the surrounds of the neighboring cones overlap more than their centers. Consequently, the cone surrounds sum more completely in the ganglion cell than do the centers (see Freed & Sterling, 1988).

Surround/center balance

The ganglion cell's total surround has been reported as weaker than the total center by a factor of 0.6 to 0.9 (Linsenmeier et al., 1982; Derrington & Lennie, 1982). We tried different beta cell surround/center balances b as low as 0.7 and found that the balance value b chosen for the ganglion cell receptive field did not affect the extent of the computed cone receptive field. Therefore, in the remainder of our computations, we set the surround/center balance to 1.0.

Sensitivity of cone center to variation in beta cell circuit

Dendritic field size of the beta cell varies somewhat at a given eccentricity (Boycott & Wässle, 1974; Kolb et al., 1981). To assess the sensitivity of our results to this type of variation, we computed the cone receptive field for beta cell circuits that differed by ±10% in (anatomical) extent from the standard circuit at 1 deg. The resulting cone center varied by only about ±4%.

Thus, in this case, variation in ganglion cell dendritic field size must have only a minor influence on the ganglion cell center (see Discussion).

Cone receptive field beyond area centralis

To compute cone receptive fields at 10 and 20 degs, we took the beta cell center radii from Linsenmeier et al., 1982, and, with corrections for shrinkage, cone spacings from Steinberg et al. (1973), and the beta cell dendritic fields from Boycott and Wässle (1974) and Kolb et al. (1981). The size of the cone array was taken as the number of cones encompassed by the ganglion cell dendritic arbor. Bipolar cells whose axons contact the edge of the ganglion cell dendritic arbor have dendrites that reach beyond it and so enlarge the convergent cone array (Sterling et al., 1988). However, in the present case this effect is apparently small. At 30-deg eccentricity the dendritic spreads of a b1 cell and a beta cell are, respectively, about 20 μm and 200 μm (Kolb et al., 1981; Boycott & Wässle, 1974) suggesting a potential expansion on the order of 10%. But this broadening of the synaptic weighting function would be counteracted by the steepening of the electrotonic weighting function (see Methods). Thus, bipolar dendritic arbors probably do not enlarge receptive fields of peripheral beta cells by more than 5% (see Discussion).

The computed cone receptive fields at 10 and 20 deg are compared in Fig. 3 to the measured beta cell receptive fields. As at 1 deg (Fig. 2), the cone centers at 10 and 20 deg are broad; in fact they are broader than the cone center at 1 deg, and this is consistent with lower cone density for peripheral cones. Yet, the peripheral cone center is narrower in proportion to the beta center to which it contributes. Figure 3 also shows that, as at
1 deg, the amplitude of the beta cell surround is greater than that of an individual cone surround. In fact, as larger numbers of cones converge to peripheral beta cells, the effect is even more marked (Table 1).

Bipolar cell receptive field

Based on bipolar circuits from Cohen and Sterling (1990a), we computed a $b_1$ bipolar receptive field at 1-deg eccentricity. The bipolar cell collects equal input from an array of five cones. Its receptive-field center radius was 26 $\mu m$, intermediate (not surprisingly) between that of the cone and beta ganglion cell, and its surround radius was also intermediate.

Discussion

Properties of the computed cone receptive field

The main results of our computations are (1) the center of the cone receptive field is broad (in the area centralis, 90% of the beta ganglion cell center); (2) the surround of the cone is nearly equal in extent to that of the beta cell but weaker; and (3) the extent of the cone receptive field increases with eccentricity but its fraction of the beta cell receptive field decreases.

The results depend on a match at each eccentricity between anatomical and physiological measurements. To variation in such matches the results are robust: a shift of $\pm 10\%$ in the width of the cone array and beta cell dendritic tree (at 1-deg eccentricity) for a given physiological measurement of its receptive field would change the cone receptive-field width by only $\pm 4\%$. This is because the beta cell receptive field is the convolution of two functions: the cone receptive field and the synaptic weighting function (Fig. 1b). As noted in Methods, when two Gaussian functions are convolved, their variances sum, so the width of the resulting Gaussian (after normalization) equals the root mean square of the original widths. Consequently, when Gaussians of unequal width are convolved, the smaller one contributes disproportionately little. At 1-deg eccentricity,
the beta synaptic weighting function (30 μm diameter) is narrower than the computed cone receptive field (50 μm diam), so its contribution to the beta cell receptive field (56 μm diameter) is relatively small. The actual cone receptive-field profile may not be truly Gaussian but it is probably close enough to justify this conclusion. At larger eccentricities, the diameters of the synaptic and dendritic weighting functions are stronger determinants of beta receptive-field size.

One concern is the effect of the stimulus on receptive-field measurements. Our results for beta cells at 1 deg depend on normalizing the measurements of two studies that used different stimuli and methods of analysis. Linsenmeier et al. (1982) used sine-wave gratings and fitted their data with the difference-of-Gaussians model. This method removed the influence of stimulus size on the measured center width. However, Cleland et al. (1979) used a stimulus bar (22 μm wide) and measured the center width at threshold (where the center response equaled the maintained firing rate). In this case, the bar dimensions would effect the measured center width. To explore this effect, we convolved the ganglion cell receptive fields from 1-deg and 10-deg eccentricity with a 22-μm bar (Fig. 4).

The main effect of this convolution is to strengthen the surround relative to the center. This in turn causes the width of the center at zero-crossing to be narrower than for a simple Gaussian. The widths at zero crossing for both convolutions are larger than the corresponding Gaussian widths at 37% peak amplitude by a factor of about 1.4. Thus, it seems plausible that the factor of 1.3 found empirically to relate the two sets of measurements (see Results) might have originated in the different methods of measurement.

Evidence for a center/surround cone receptive field

The idea that a cone receptive field is both broad and concentrically antagonistic emerges first from the results on cones of lower vertebrates (e.g. Baylor et al., 1971; Detwiler & Hodgkin, 1979; Piccolino et al., 1981; Murakami et al., 1982; Normann et al., 1984; Kraft & Burkhardt, 1986; Kaneko & Tachibana, 1986; Werblin, 1974). Similar behavior would be predicted on anatomical grounds for mammalian cones because, like the cones of lower vertebrates, they interconnect with each other through electrical synapses (Raviola & Gilula, 1975; Kolb, 1977; Smith et al., 1986; Tsukamoto et al., 1990b) and interconnect with horizontal cells through GABAergic chemical synapses (Chun & Wässle, 1989; Sarthy & Fu, 1989). Nelson’s (1977) recordings from cat cones do show quite broad centers, as computed here, but no indication of a surround. However, the cone’s computed surround is quite weak (~2-3% of the peak center response). The cone recordings reported by Nelson were rather small, only a few millivolts. To see a surround response on the order of 50 μV would probably have required temporal averaging. We have simulated the cone receptive field using a large-scale compartmental model based on outer plexiform layer circuitry (Smith & Sterling, unpublished). The results, to be presented elsewhere, agree with Nelson’s results for the cone center and support the suggestion offered here as to why a surround was not observed. Finally, hyperpolarizing current injected into rabbit horizontal cells antagonizes the ganglion cell center response (Mangel & Miller, 1987), and this is consistent with the idea that mammalian horizontal cells feed back negatively onto cones.

![Figure 4](image)

**Fig. 4.** Comparison of measured beta cell receptive field fitted to a difference-of-Gaussians (solid) with the same receptive field convolved with a stimulus bar 22 μm wide (dotted) at 1-deg (a) and at 10-deg (b) eccentricity. The center width measure of Linsenmeier et al. (1982) corresponds to 37% of peak amplitude on the solid line, and the center width measure of Cleland et al. (1979) corresponds to the zero crossing of the dotted line.
Origin of the beta cell receptive field

Our results rest on a specific hypothesis regarding the origin of the beta ganglion receptive field, namely, that it arises from linear superposition of cone receptive fields (Fig. 1). This simple model of the beta cell has not been stated explicitly before, but it emerges naturally from all that is known regarding both the beta cell's functional architecture and that of the outer plexiform layer:

1. The fundamental component of the beta cell's response is linear for stimuli within a certain limited dynamic range (Rodieck & Stone, 1965; Enroth–Cugell & Robson, 1966; Sakmann & Creutzfeldt, 1969; Cleland et al., 1975; Enroth–Cugell et al., 1983; Frishman et al., 1987).

2. The cone receptive-field center probably arises from electrical coupling and optical blur (see below).

3. The cone surround probably arises from horizontal cell negative feedback (see below).

4. Narrow-field bipolar cells convey most of the beta cell's input from cones, and the weighting of their synaptic connections at the inner plexiform layer contributes somewhat to the domed shape of the beta cell receptive-field center (Cohen & Sterling, 1990a).

Alternative model of beta cell receptive field

Although our model of the beta cell receptive field (linear superposition of center/surround cone receptive fields) fits much of what is known about the retina of cat and other species, it is not the only possibility. One alternative would route the center of the beta cell through narrow-field bipolar cells and the surround through wide-field cells, either bipolar or amacrine. This "separate pathway" model has been proposed to account for the fact that the beta cell center and surround adapt independently (Enroth–Cugell et al., 1975; Shapley & Enroth–Cugell, 1984).

This alternative does not fit the known anatomy. There exists a wide-field bipolar cell whose axon comingles with dendrites of the on-beta cell and that conceivably might convey the surround (Famiglietti, 1981; Kolb et al., 1981; Pourcho & Goebel, 1987; Cohen & Sterling, 1990b). However, this bipolar cell does not contact the beta cell directly (Cohen & Sterling, 1990b). Furthermore, it collects only from a small subset of cones (probably blue cones; see Famiglietti, 1981; Cohen & Sterling, 1990b,c). Since blue cone input to the beta cell is not observed (Cleland & Levick, 1974), this bipolar probably does not supply the amacrine cells that connect to the beta cell.

Most of the amacrine cells so far associated with the on-beta cell are rather narrow-field and, collecting as they apparently do from narrow-field bipolar cells, would be unsuited to convey a broad surround to the beta independently from its center. The wide-field amacrine cells that do exist have extremely fine processes (Kolb et al., 1981; Nelson & Kolb, 1985). If these processes are passive, they would tend to have rather short space constants unsuited to convey the surround. If instead these processes are regenerative, they would likewise be unsuited to the task. The obvious candidates from a morphological perspective to convey a smoothly decaying, wide (5 x center) surround would be the thick, broadly spread, electrically coupled dendrites of the type A horizontal cells (see Boycott et al., 1978). Quantitative support for this suggestion will be presented elsewhere (Smith & Sterling, unpublished). To summarize, anatomical evidence for a model in which center and surround feed forward independently to the beta cell is essentially nil, while for a model in which the center and surround merge at the outer plexiform layer, it is ample. A cable model for the spatiotemporal-frequency responses of the beta cell also favors the feedback origin of the surround (Chen & Freeman, 1989).

Implications of the beta cell model

Our model for the beta cell implies, since center and surround adapt separately (Enroth–Cugell et al., 1975), that adaptation must occur at the level of the cone. This might be accomplished by biochemical mechanisms in the outer segment (Baylor et al., 1979; Liebman et al., 1987; Pugh & Altman, 1988; Sneyd & Tranchina, 1989). If, as in turtle, there is pooling of adaptation between cones (Copenhagen & Green, 1987), it must be spatially restricted and must be accomplished before the center and surround mechanisms are summed in the cone pedicle. This is consistent with psychophysical evidence that adaptation is accomplished no deeper in the retina than the outer plexiform layer (Hayhoe & Smith, 1989).

Implications of the cone receptive field

The model implies that three critical integrative steps are accomplished before the visual signal crosses the first chemical synapse: adaptation (gain control), subtraction of low spatial frequencies to reduce redundancy (Barlow & Levick, 1976; Srinivasan et al., 1982), and pooling of higher spatial frequencies to reduce noise (Tsukamoto et al., 1990a). All subsequent elements in the retina then should show these properties. Furthermore, since the cone receptive fields are rather large compared to their spacing, subunits of these dimensions should appear at the level of the inner plexiform layer not only for linear mechanisms, but also for nonlinear mechanisms, since these too must derive from the cone receptive fields. Subunits of the appropriate dimensions contribute to the receptive field of alpha ganglion cells (Hochstein & Shapley, 1976).

If the cone receptive field has a center/surround organization, then so should the receptive fields of cone bipolar cells. In cat this has been observed for one bipolar type but not for others (Nelson & Kolb, 1983). However, since the surrounds of peripheral cones should be of low amplitude (Fig. 3, Table 1), so should surrounds of peripheral bipolar cells. So far, only a very few bipolar cells have been studied in cat; thus, the possibility that they have surrounds should not yet be excluded.

If the basic subunit summed at the ganglion cell has a small center and a large surround, it follows that as the ganglion cell dendritic field width (weighting function diameter) increases, not only would center width rc increase, but also the relative peak amplitude of the surround ks/kc. However, surround width for large dendritic fields should not increase proportionately. This is observed when comparing responses of peripheral versus central beta cells (Figs. 2 and 3) (Wiesel, 1960) and alpha versus beta cells at any given eccentricity (Freed & Sterling, 1988).

Establishing at an early stage a subunit whose functional spread is much wider than its spacing has important consequences to the design of later stages of retinal wiring. Most immediately it relaxes the constraints on how regular cell spacing
must be in the various independent mosaics. It also relaxes constraints on how regular the dendritic branching patterns of bipolar cells and ganglion cells must be. For example, bipolar dendritic fields commonly show marked radial asymmetries (Cohen & Sterling, 1990b, c). But this will hardly affect the symmetry in the bipolar receptive fields because the latter apparently arise by superposition of a broad, overlapping subunit already established at the cone pedicle. Similarly, the alpha ganglion cell dendritic tree sometimes fails to provide a branch where one is needed to "catch" contacts from a particular descending bipolar axon (Freed & Sterling, 1988), but this does not lead to a hole in the alpha receptive field because receptive-field centers of the neighboring bipolar cells are so broad.

**Function of the cone receptive field**

The receptive field of the ganglion cell serves, in effect, as a spatial band-pass filter. The surround removes low spatial frequencies to keep the signal within the cell's dynamic range (Barlow & Levick, 1976; Barlow, 1981; Srinivasan et al., 1982), and the center removes high spatial frequencies to reduce noise (Barlow, 1981; Tsukamoto et al., 1990). Both operations are involved in matching the signal to the information capacity of the channel. Our results suggest that such a band-pass mechanism is already established at the level of the cone pedicle. This is apparently necessary to match the cone signal to the information capacity of the circuit that connects cone to ganglion cell. (see Laughlin et al., 1987).

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**References**


