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INVARIANT CONTRAST ADAPTATION IN THE PRIMATE OUTER PLEXIFORM LAYER (OPL)

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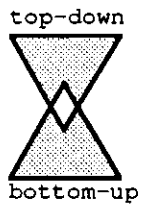

Invariant Contrast Adaptation in the Primate Outer Plexiform Layer(OPL)

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1 Introduction

Primate photopic vision operates over 7 logarithmic units(lu) of retinal illuminance without significant degradation of luminance contrast sensitivity[10] even though the light intensity domain in individual cone photoreceptor is less than 4 lu[24]. The question of how exactly is it achieved remains open.

The invariance of luminance contrast processing is critically important for lightness and color constancy [11,19,8] as well as for aerial contrast phenomena[11,9]. The invariance process depends on proper luminance contrast processing at all stages of the visual system starting with the retina. Although these phenomena have been extensively investigated in the past, the neural structure underlying mechanism of interpreting surface reflectance as white, grey or black remains a matter of controversy. Despite the paucity of accurate anatomical and physiological data about primate visual system, progress in modeling computational aspects of perceptual theories underlying color and lightness constancy has helped to elucidate the role of the retina in these visual functions. Furthermore, the most recent anatomical and physiological experimental results suggest that the site of luminance contrast processing in the primate retina is the outer plexiform layer(OPL)[4,5]. This makes it possible to examine in depth the compatibility between computational theories based on psychophysics and physiological data.

We present quantitative investigation of the luminance contrast processing properties of the primate OPL using a computer model based on known anatomical and physiological data. Our simulation results demonstrate that simple network interactions can transform absolute retinal illuminance into relative luminance contrast; center-surround antagonism in cone receptive field(RF), verified physiolgically in vertebrates, can explain contrast processing properties in primates observed psychophysically[27,2,6].

2 Organization of the Primate OPL

The OPL of the primate retina plays a major role in luminance contrast processing. Light hyperpolarizes photoreceptors which drive horizontal cells(HC's) via non-inverting chemical synapses. Both types of horizontal cells, narrow receptive fields (HC1) and broad receptive fields (HC2), contact all cone pedicles in their dendritic fields. In the fovea, each HC1 contacts 6-7 cones and each HC2 contacts 13-14 pedicles regardless of cone chromatic type[4,3,5]. Both horizontal cell types have axon terminal(AT) that makes synaptic contacts with spherules(for HC2) and pedicles(for HC1) exclusively[4]. HC cell bodies and their AT's are thought to be electrically isolated and thus functionally independent[5]. Although HC1 and HC2 both appear to be equally sensitive to all light wavelengths, we hypothesize HC1's are more important than HC2's for foveal photopic vision because of HC2's lower density in the fovea[3,16]. In the present investigation only HC1's are modelled.

Fidelity of the model is critically dependent on the accuracy of the anatomical and physiological parameters such as the cone to HC1 density ratio(i.e. coverage factor). The cone to HC1 coverage factor has been estimated to be 3-4[4,26]. Pedicle size is critical since there is a receptoral areal magnification factor(more than 10 in the fovea[17]) going from the outer segment to the pedicle of a cone. Knowing the HC1 to pedicle coverage factor and the pedicle to HC1 RF size allows for the calculation of the HC1 RF overlap which is approximately 50%. To determine the extent of cone-cone and HC1-HC1 coupling one needs to estimate the gap junction conductance for cones and HC1's. Unfortunately, there is no known physiological data on primate gap junction conductance. Estimate for cone-cone and HC1-HC1 RF sizes is obtained by interpreting psychophysical data. From [27], the cone-cone RF diameter is estimated to be 7 cones and HC1-HC1 RF diameter is 27 HC1's. Although no functional feedback synapses have been verified in primates the necessary synaptic structures do exist in ample quantities[15]. Anatomical and physiological correlates of feedback synapse have been reported in turtles[1] and in Tiger salamanders[12,13,20,18,21].

The involvement of feedback in relative contrast enhancement and color coding have been reported in turtles[1,7], Tiger salamanders[18,13], and fish[22]. Although it is generally believed that feedback plays similar role in primate OPL, there are no results to confirm this notion. The role of feedback in invariant contrast adaptation has been more difficult to verfiy. Nevertheless, in Tiger salamanders it has been shown that feeback from HC can instantaneously shift the cone operating point to be in register with ambient light intensity of the surround; this preserves relative contrast invariance[19].

3 A Primate OPL Model

Our primate OPL model consists of two layers representing cones and the HC1's(Fig. 1). Functional view of the model is shown in Fig. 2. The sign of the various signals are actually opposite that of their respective physiological correlates(e.g. hyperpolarization is represented by signal increment in the model). The mathematical formalism of the model is presented in Eqns. 1 through 4.

$$
\begin{aligned}
O_c(t+1) &= ORF(\\
&\quad ICF(G_{l-c1}, G_{l-c2}, S_{l-c}) + \\
&\quad ICF(G_{c-c1}, G_{c-c2}, S_{c-c}) - \\
&\quad ICF(G_{hc-c1}, G_{hc-c2}, S_{hc-c}) - \\
&\quad L_c O_c(t) - \\
&\quad K_c) \qquad (1) \\
S_{l-c} &\equiv \textit{incident light intensity.} \\
S_{c-c} &\equiv \textit{Gaussian weighted sum of cone} - \textit{cone inputs.} \\
S_{hc-c} &\equiv \textit{Gaussian weighted sum of } HC1 - \textit{cone inputs.} \\
G_{l-c1}, G_{l-c2} &\equiv \textit{incident light gain parameters.} \\
G_{c-c1}, G_{c-c2} &\equiv \textit{cone} - \textit{cone gain parameters.} \\
G_{hc-c1}, G_{hc-c2} &\equiv HC1 - \textit{cone gain parameters.} \\
L_c &\equiv \textit{cone voltage dependent leakage modulation parameter.} \\
K_c &\equiv \textit{constant cone leakage.} \\
O_{hc}(t+1) &= ORF(\\
&\quad ICF(G_{c-hc1}, G_{c-hc2}, S_{c-hc}) + \\
&\quad ICF(G_{hc-hc1}, G_{hc-hc2}, S_{hc-hc}) - \\
&\quad L_{hc} O_{hc}(t) -
\end{aligned}
$$

$$K_{hc}) \tag{2}$$

$$\begin{aligned}
S_{c-hc} &\equiv \textit{Gaussian weighted sum of cone} - HC1 \textit{ inputs.} \\
S_{hc-hc} &\equiv \textit{Gaussian weighted sum of } HC1 - HC1 \textit{ inputs.} \\
G_{c-hc1}, G_{c-hc2} &\equiv \textit{cone} - HC1 \textit{ gain parameters.} \\
G_{hc-hc1}, G_{hc-hc2} &\equiv HC1 - HC1 \textit{ gain parameters.} \\
L_{hc} &\equiv HC1 \textit{ voltage dependent leakage modulation parameter.} \\
K_{hc} &\equiv \textit{constant } HC1 \textit{ leakage.}
\end{aligned}$$

$$ICF(G_1, G_2, I) = G_2 \tanh(G_1 I) \tag{3}$$

$$\begin{aligned}
G_1 &\equiv \textit{lumped pre} - \textit{nonlinearity gain.} \\
G_2 &\equiv \textit{lumped post} - \textit{nonlinearity gain.}
\end{aligned}$$

$$ORF(I) = \begin{cases} I & \text{if } I \geq 0 \text{ and } I \leq 1 \\ 1 & \text{if } I > 1 \\ 0 & \text{if } I < 0 \end{cases} \tag{4}$$

Cone input connections are modelled using three transfer functions representing the two excitatory (hyperpolarizing) input types due to incident light and neighboring cones and the inhibitory (depolarizing) input type due to feedback from neighboring HC1's. The cone output is the sum of the three input transfer functions(**Input Conductance Functions**(ICF's)) which differ only in parameters(see Eqn. 3). On the other hand HC1's receive only excitatory inputs from neighboring cones and HC1's. Each of the inputs to HC1 is also characterized by unique ICF. Each ICF operates on a Gaussian weighted sum of inputs over a two dimensional RF(see Table 1 for the actual RF sizes).

Parameters	Value
cone-cone RF diameter	7 cones
HC1-HC1 RF diameter	27 HC1's
cone-HC1 RF diameter	3 cones
HC1-cone RF diameter	3 cones
G_{l-c1}	0.662
G_{l-c2}	1.750
G_{c-c1}	0.221
G_{c-c2}	2.000
G_{hc-c1}	0.883
G_{hc-c2}	1.000
G_{c-hc1}	0.132
G_{c-hc2}	1.000
G_{hc-hc1}	0.530
G_{hc-hc2}	1.000
L_c	0.000
L_{hc}	0.000
K_c	0.300
K_{hc}	0.000

Table 1: Model Parameters

The cone output is generated by rectifying(via the **Output Rectification Function**(ORF) shown in Eqn. 4) the result of the sum of three ICF outputs minus two leakage terms(fixed and voltage dependent). The output of a HC1 is determined in a similar manner as the cone output.

It is clear now that an ICF is really a lumped model of the actual synaptic conductance characteristics that might vary from synapse to synapse. This lumped model is adequate since the goal is to identify the *robust* physiological characteristics of the OPL. A more serious limitation is the fact that all signal delays in the model are equal and constant(i.e. all capcitances have the same value). This limitation was introduced intentionaly to simplify the model and facilitate the analysis of the steady-state behavior.

4 Simulation Results

All simulations were conducted using *RetSim*, a retinal network simulator developed within the UCLA-SFINX simulation environment[14]. Separate experiments were conducted to demonstrate two aspects of the contrast processing properties of the primate OPL model, namely invariant contrast adaptation and relative contrast enhancement. A model with a 128x128 cones and 42x42 HC1's was used in all simulations. Identical setup was used for all experiments(Fig. 3) except for the stimulus configuration which is unique to each experiment. In all cases the center spot diameter and the annulus inner diameter was equal to 7 cone diameters and the annulus outer diameter was equal to 27 cone diameters.

Figure 4 shows the center cone response to a stimulus with a 1.5 lu center-surround reflectance difference maintained under various illumination settings. The response curve can be divided into 4 sub-regions, region I(-3 to -2 lu), region II(-2 to 0 lu), region III(0 to 3 lu),and region IV(3 to 7 lu). Region III is of interest since invariant contrast adaptation occurs here. The invariance is seen as the near constant response over the region. However, region III does not really show the entire extent of the invariant contrast adaptation response since the region bounds are responses to a 1.5 lu difference stimulus. At a given operating point the linear cone response domain is about 4 lu(see Figure 5) thus the correct extent of the invariant contrast adaptation region is -1.5(=0-1.5) to 5.5(=3+2.5) lu; the model is capable of invariant contrast adaptation over 7 lu(=5.5-(-1.5)). Region I characterize the sub-threshold reponse of cones. Region II is the transition region from sub-threshold to super-threshold response regions. Region IV basically illustrates the gradual failure of feedback control.

To show the high contrast sensitivity of the model a series of experiments were done where the surround is fixed at a given illuminance level while the center illuminance is varied from -3 to 7 lu in 0.5 lu increments. The center cone response for 7 different surround settings were recorded and are shown in Figure 5. It is clear that feedback from HC1 shifts the cone response curve to achieve invariant contrast adaptation while preserving high contrast sensitivity. These results quantitatively demonstrates that high contrast sensitivity and invariant contrast adaptation can coexist.

5 Discussion

The cone response curves shown in Figure 5 correspond surprisingly well to actual primate extracellular recordings[24]. Traditionally, three basic mechanisms have been hypothesized to explain cone light adaptation, namely response compression, pigment bleaching, and cellular adaptation involving network interactions between cones and HC's. The model presented here is essentially a quantitative model of cellular adaptation and the results support the cellular adaptation hypothesis.

Results shown in Figure 4 correlate well with psychophysically determined foveal threshold-versus-intensity(tvi) curves[2,6] up to 4 td. Clearly, cone response over the invariant contrast adaptation region is not perfectly constant thus it does not agree Weber-Fechner law completely. This fact is actually consistent with the observed phenomenon of *more light means better sight*[25]. It is possible that invariant contrast adaptation underlies the observed Weber-Fechner-like behavior but further study is necessary.

Invariant contrast adaptation evidently fails at higher luminance levels however the failure need not be as dramatic as shown in Figure 4. Cone pigment bleaching is known to be a signficant sensitiviy reduction mechanism at high illuminance levels[23,10] which ameliorates the effect of invariant contrast adaptation failure. Data[23,24] shows cone pigment bleaching becomes significant at illuminance levels above 4 td which is roughly the onset point of invariant contrast adaptation failure. The net effect of bleaching is more than 1 lu of operating curve shift at high illuminance levels[24]. Indeed, even wider invariant contrast adaptation region can be achieved if pigment bleaching mechanism is utilized.

In summary, the simulation results from a rather simple model of the primate OPL appear to be quantitatively consistent with available physiological and psychophysical data on primate foveal photopic vision. We tentatively conclude the present model forms a bridge among the anatomical, physiological and psychophysical data. The results also lends further support to the belief that luminance contrast

processing is accomplished no later than the first synaptic stage(i.e. in OPL).

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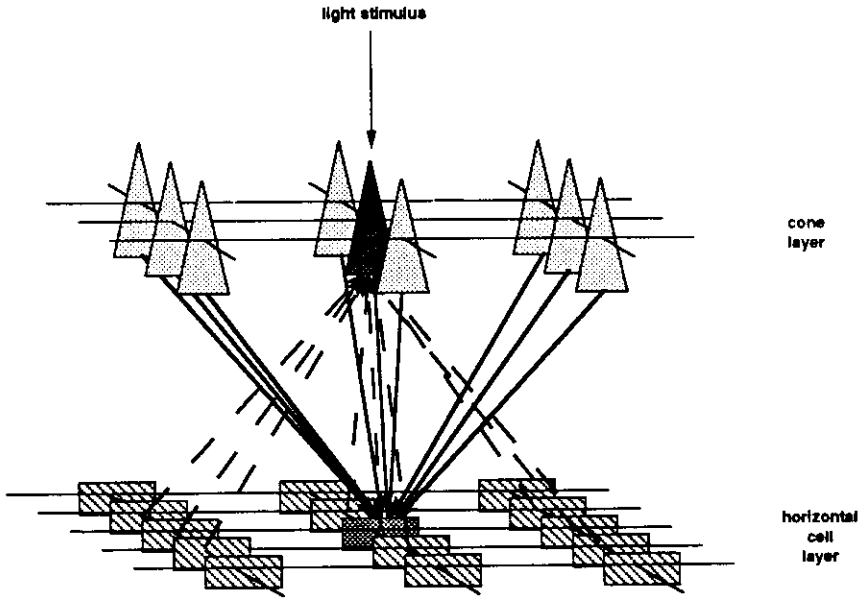

Figure 1: Model Structural Schematic

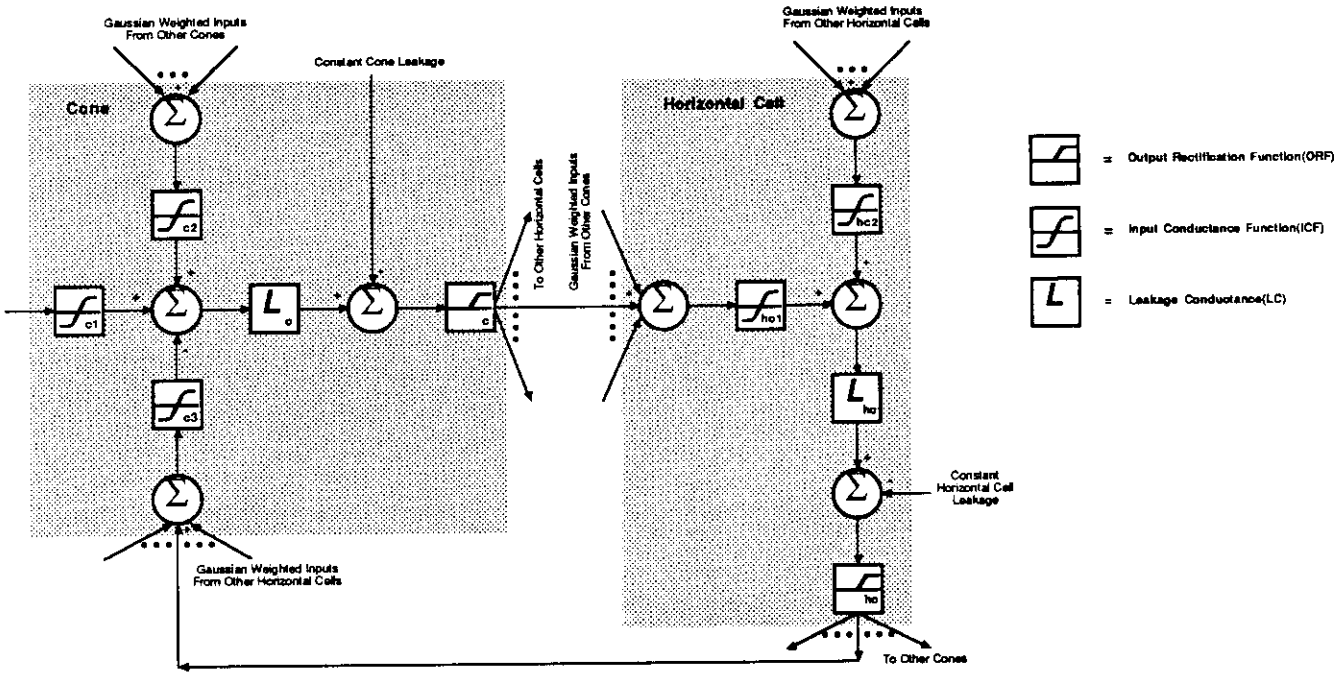

Figure 2: Model Functional Diagram

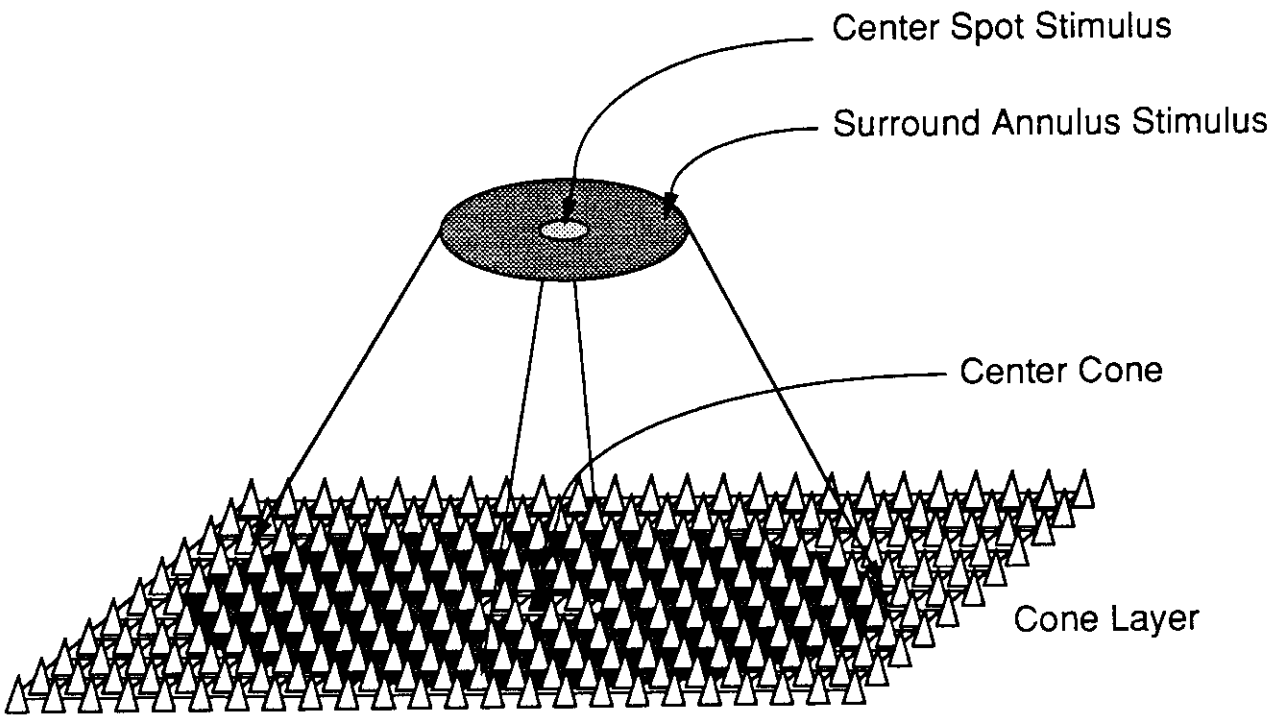

Figure 3: General Experimental Setup

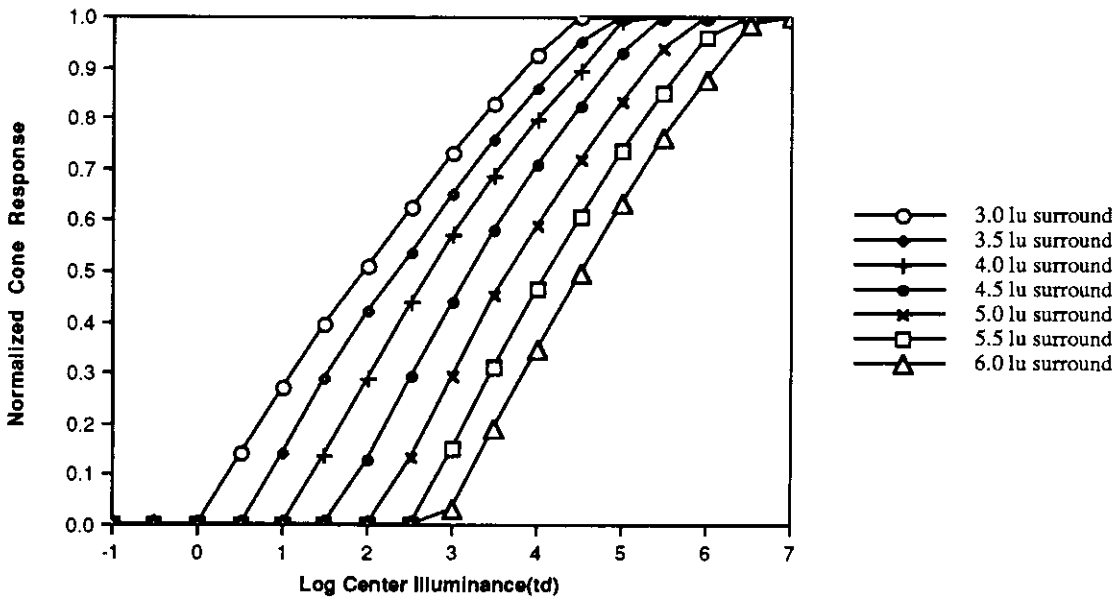

Figure 4: Center cone response to stimulus with a constant 1.5 lu center-surround reflectance difference under various uniform illumination settings. 1.5 lu was chosen as it roughly represents the the reflectance difference between white and black under uniform illumination.

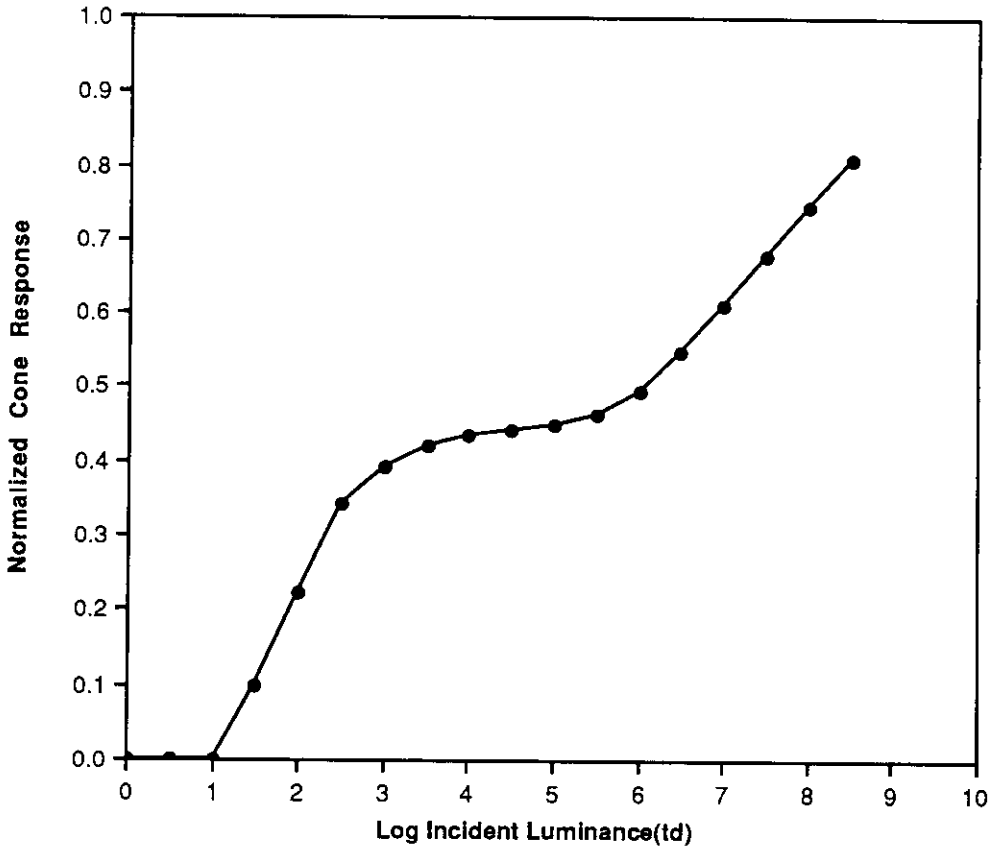

Figure 5: Center cone response to stimulus center-surround luminance difference under various fixed surround settings.