10 Retina[1]

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10.1 Introduction

Teeters and Arbib (1991) (Teeters and Arbib 1991) presented a model of the anuran retina which qualitatively accounts for the characteristic response properties used to distinguish ganglion cell types in anurans. Teeters et al. (1993) tested the model's ability to reproduce quantitatively tabulated data on the dependency on stimulus shape and size, with a new implementation of the model in the neural simulation language NSL. Data of Ewert & Hock (1972) relating toad R2, R3, and R4 ganglion cell responses to moving worm, antiworm, and square-shaped stimuli of various edge lengths are used to test stimulus shape and size dependency. Gaillard et al. (1998) submitted the model to the whole battery of physiological experiments to validate the performance under different stimulation conditions. We stress here the importance of a populational approach to the models. We place more emphasis on the variation of response properties in a population of neurons of the *same* class, rather than questing for *the* neuron of a given type.

10.2 Model Description

The anuran retina model of Teeters et al. (1993) accounts for the qualitative characteristic response properties used to classify anuran ganglion cell types as well as for the quantitatively determined ganglion cell responses dependent on stimulus size and shape. The structure of the model is shown in figure 10.1.

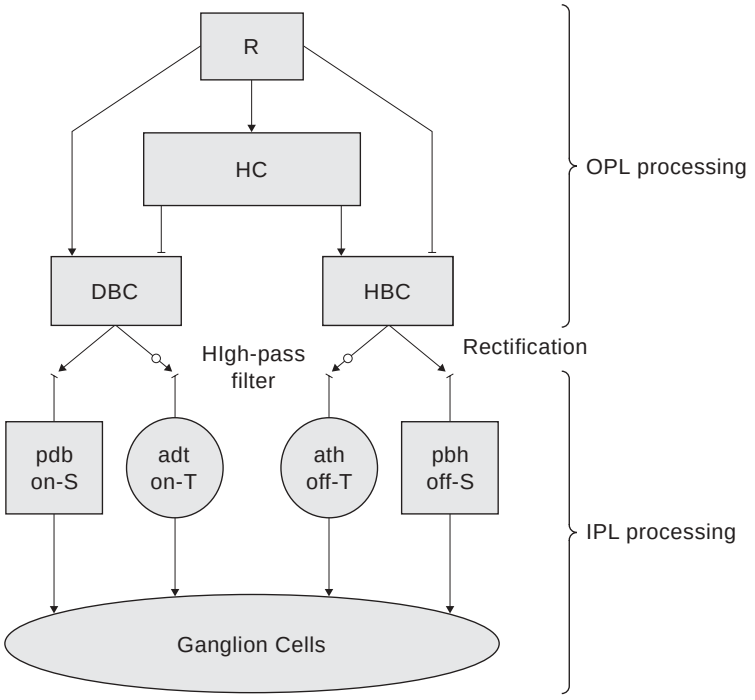


Figure 10.1
Overview of model structure. Cell types are: R - Receptors; HC - Horizontal cells, DBC and HBC - Depolarizing and Hyperpolarizing bipolar cells; PBD and PBH - positive part of bipolar cell potentials; ATD and ATH - transient amacrine cells from DBC and HBC channel; OPL - outer plexiform layer. IPL - inner plexiform layer.

The top part shows the layers of cells that feed all the ganglion cells, while the bottom part shows the specific inputs for each ganglion cell type. Each single cell in these diagrams represents a layer of cells in the formal model. We summarize the different

layers of the model in table 10.1 and the equations for the model in table 10.2. We will present possible improvements as the exposition progresses.

Abbreviation	Description
R	Receptor cell
H (HC)	Horizontal Cell
R0 - R4	Retinal ganglion cell types: classes 0 to 4
HBC	Hyperpolarizing Bipolar Cell
DBC	Depolarizing Bipolar Cell
PBH	Positive component of the Hyperpolarizing Bipolar Cell
PBD	Positive component of the Depolarizing Bipolar Cell
ATH	Hyperpolarizing Transient Amacrine
ATD	Depolarizing Transient Amacrine
ERF	Excitatory Receptive Field
IRF	Inhibitory Receptive Field

Table 10.1
Neural layers.

Neuron Layer	Equations	
Receptor	$R = 1 - I$ (10.1)	
Horizontal	$\tau_H\, dH/dt = H_0 - H$, $H_0 = 0$ ambient light, 1 ambient dark; $\tau_H = 0.1$ (10.2)	
	Off channel	On channel
Bipolars	HBC = R - H (10.3) $PBH = \max(HBC, 0)$ (10.5)	DBC = H - R (10.4) $PBD = \max(DBC, 0)$ (10.6)
Amacrines	$\tau_a\, dHBX/dt = HBC - HBX$, $\tau_a = 0.3$ (10.7) $ATH_t = \max$ (HBC-HBX, $ATH_{t-1}\, e^{-t/\tau_a}$) (10.8)	τ_a dDBX/dt = DBC - DBX (10.9) $ATD_t = \max$ (DBC-DBX, $ATD_{t-1}\, e^{-t/\tau_a}$) (10.10)
R0 Cells	$R0 = k0 * ATD - k1 * ((3 \cdot ATH) + ATD)$ (10.11) with k0 = mask(4, 1.8, 1), k1 = mask(15.5, 3.7, 0.8)	
R1 Cells	$R1 = k0 * (PBD+PBH+ATD+ATH) - k1 * (ATD+ATH)$ (10.12) with k0 = mask(3, 2.3, 1), k1 = mask(19.5, 4.6, 3)	
R2 Cells	$R2 = g \cdot ((k0 * PBH) + tc)$ (10.13) where tc = k0∗ATH - k1 ∗ (ATH+ATD), and g = pos(tc) where pos(x) = 1 if x > 0, 0 otherwise with k0 = mask(4, 2.4, 1), k1 = mask(19.5, 4.6, 3)	
R3 Cells	$R3 = k0 * a - (k1 * a)_{delayed}$ (10.14) where a = p · ATD + ATH with p = 0.4, k0 = mask(8, 2.4, 1), k1 = mask(19.5, 4.6, 1.4) while $(s)_{delayed}$ = signal s delayed by 40 milliseconds.	
R4 Cells	$R4 = k0 * (ATH - x \cdot ATD)$ (10.15) with x = 1, k0 = mask(15.5, 3.5, 1).	

Table 10.2
Algorithms for receptors through ganglion cells in the model.

Receptors (R) convert light energy into neural potentials. The hyperpolarizing response to light is modeled by setting the receptor potential to the inverse of light intensity (*I*) that ranges from 0 (dark) to 1 (light). Adaptation and other complexities are not included in the model. Note that in the case of R2, the model uses two temporary

variables *tc* and *g* where *tc* is the total transient input to the cell and *g* is a gate which is set to 1 if the net transient excitation is larger than the inhibition.

Horizontal cells (H) form the surround receptive field of both bipolar cell types. They are modeled so that they are only sensitive to the background illumination of the surround (H_0 in table 10.2) and are spatially invariant (uniform potential model) through the infinite spread of the activation within the cells. This simple interpretation of horizontal cell function ignores the effect of presentation of a local stimulus and suggests that their main function is to bias the bipolar cells so they operate in their region of maximal sensitivity.

Bipolar cells (HBC, DBC) are computed as a difference between receptor and horizontal cell activity. Hyperpolarizing bipolar cells (HBC) hyperpolarize in response to light, depolarizing bipolar cells (DBC) depolarize in response to light. **PBH** and **PBD** are the positive components of the HBC and DBC responses.

Transient **Amacrine Cells (ATH, ATD)** convert the sustained bipolar outputs into transient signals. The transient amacrines are modeled as pseudo differentiators which operate by subtracting the leaky-integrated bipolar potential from the sustained bipolar potential, and then amplifying the difference if it is above threshold. We modeled the Bipolars and Amacrines to have one-to-one connections from the preceding layers based on the following assumptions: (i) horizontal cells in this model have a uniform potential which in effect makes the spatial connection properties mostly irrelevant, and (ii) dendritic tree diameters of the Bipolars and Amacrines are smaller than those of the ganglion cells.

The model input to the ganglion cells (receptors through bipolar and amacrine) ignores optics, different receptor types, light adaptation, and distinctions between other subtypes of horizontal, bipolar, and amacrine cells. It is not our claim that this simplification exhausts the functionality of these cells. Rather, we seek to emphasize that only those properties analyzed in this paper are essential for understanding the range of ganglion cell properties described here. In fact, the Teeters and Arbib (1991) implementation of the horizontal and bipolar cells does not really affect the outcome of the stimulus shape and size discrimination tasks. Nevertheless we need the horizontal and bipolar cells to account for other phenomena caused by changes in whole field illumination.

Ganglion cells (R0 – R4) receive input from bipolar and amacrine cells. Unlike the bipolar and amacrine cells which have one-to-one connections to their preceding layers, each ganglion cell input is composed of a central ERF (Excitatory Receptive Field), and a wider IRF (Inhibitory Receptive Field). The spatial properties of the ERF and IRF are specified as two-dimensional Gaussians. The notation "*mask*(*dia, sig, wgt*)" in table 10.2 denotes a 2-dimensional Gaussian with standard deviation *sig* (in visual degrees) which is truncated with diameter *dia* (so that the Gaussian values are replaced by 0 for points more distant than *dia*/2, also in visual degrees, from the center), and which is normalized so that the sum of all elements is equal to *wgt* (for a more detailed description see Appendix I). The ERF extent is modeled as arising from ganglion cell dendritic tree topology that is narrowly spread, whereas the IRF arises from a more widely spread topology. The corresponding diagrams are shown in figure 10.2.

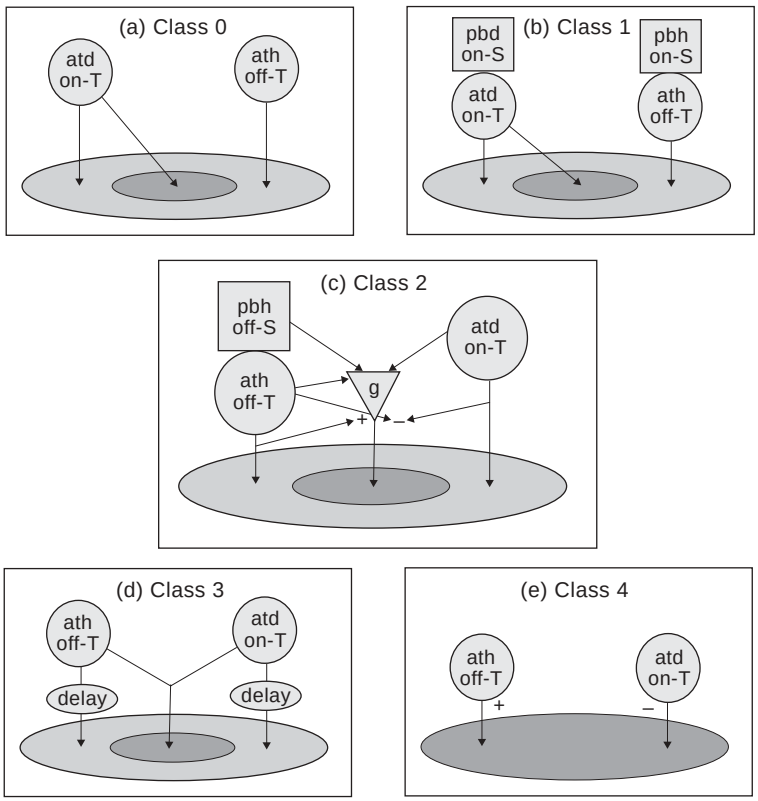


Figure 10.2
Ganglion cells R0 through R4. The receptive field for ganglion cells type R0 through R3 is composed of a small excitatory receptive field (ERF) and an overlapping larger inhibitory receptive field (IRF). The ERF and IRF in the R4 model are the same size. Input to both ERF and IRF are from bipolar and amacrine cells (pbd, phb, ath, atd). Spatial connections and other details of the algorithms are not shown here but are given in the text. (From Teeters and Arbib 1991.)

Stimulus Shape and Size dependency

In general, the average response of anuran ganglion cells to a moving stimulus depends on stimulus configuration, size, and velocity—a long thin bar moving in the direction of its long axis (a "worm" stimulus) will normally give a different response than the same sized stimulus moving perpendicular to its long axis ("antiworm"). Likewise, a square shaped stimulus will often generate a different response than do worm or antiworm stimuli. The response dependence on the edge length of moving worm, antiworm, and square-shaped stimuli has been determined in the toad (Ewert and Hock, 1972; Ewert, 1976) and in the frog (Schürg-Pfeiffer and Ewert, 1981). The data sets are quite different even though the same anuran cell types are recorded. In the frog data, only the R3 cell shows a distinct difference in response to worm, antiworm, and square stimuli. Although Teeters and Arbib (1991) mainly tuned the ganglion cell models to frog data, this paper will use the toad data because toad ganglion cells show a much better ability to differentiate between stimulus types.

Ewert (personal communication) only used a cell's response to the *leading edge* of the stimulus to calculate the average response (nevertheless, our temporal graphs show both the leading and the trailing edge). In accordance to this methodology, we relied on the leading edge response to calculate the average response—in all the cases the leading edge responses are clearly discernible from the residual responses. Our ability to match these data (and those analyzed by Teeters and Arbib (1991)) suggests that the model is indeed robust enough to serve as a valid "front end" for *Rana computatrix* (Arbib, 1987).

A brief qualitative analysis of the model responses to various stimulus shapes and sizes could offer some useful guidelines for further tuning of the base model. An instantaneous response of a ganglion cell is the result of summation of ERF induced excitatory response and the IRF induced inhibitory response. The inputs to ERF and IRF could be of different combinations of channels (PBH, PBD, ATH, ATD) depending on

the ganglion cell types. For instance, R2 receives PBH and ATH channels for its ERF, ATH and ATD channels for its IRF. However, sustained bipolar channel (PBH, PBD) responses and transient amacrine channel (ATH,ATD) responses present different spatial characteristics. For example, the PBH bipolar channel layer forms an activation profile identical to the size and shape of the dark stimulus. The Teeters and Arbib (1991) model uses a high pass filter to represent the amacrine cells as they convert sustained bipolar signals into transients. The resulting amacrine cell layer forms an exponentially decaying surface starting from the edges of the moving stimulus: the ATH layer forms such a surface starting from the leading edge of a dark moving surface, and the ATD layer from the trailing edge.

If the shapes of the stimulus classes are restricted to rectangles and if each bipolar and amacrine cell has maximum instantaneous firing rate of 1, overall activities of PBH and ATH on their layers are:

$$PBH_{sum} = lh \tag{10.15}$$

$$ATH_{sum} = h \int_{x=0}^{1} e^{-x/v\tau} dx \tag{10.16}$$

where x is the distance between the amacrines corresponding to the leading edge and the position of amacrines the stimulus has passed over. Obviously, PBH_{sum} is a function of both stimulus length *(l)* and height *(h)* while ATH_{sum} is only dependent on height of the stimulus for given velocity *(v)* and time constant *(τ)*. Thus, while the activation pattern on the PBH layer directly reflects stimulus shape and size, ATH layer activation pattern produces identical firing patterns for worm, antiworm and square so long as they have the same height. These different spatial firing patterns of bipolars and amacrines will form the basis of the shape dependence of ganglion cells.

The average response of anuran ganglion cells usually increases with stimulus size smaller than the ERF. Assuming that response durations are about equal for a given velocity, the increase in ganglion cell response in our model stems from the fact that as stimulus size increases, it excites a larger area of receptors and thus bipolars and amacrines. This increases the instantaneous ganglion cell response that is proportional to the sum of activation of amacrines and/or bipolars within the ERF. However, bipolar and amacrine contributions to the response growth will be different in that bipolar channel contributions will increase proportional to the stimulus area but the amacrine channel contribution will increase proportional to the height. As the stimulus size increases beyond the ERF and into the IRF region, the IRF-contributed inhibition takes effect and reduces the total response.

Due to size limitations, in this chapter we will focus on R3 cells for a detailed analysis. A similar analysis is provided in Teeters et al. (1993) for the rest of the ganglion cells.

10.3 Model Implementation

The model implementation consists at the top level of a **RetinaModel** and a **Retina** module. The **Retina** module contains a Visin module for generating synthetic visual input, the Receptors, Horizontal Cells, Bipolar Cells, Amacrine Cells and Ganglion Cells, R2, R3 and R4, each organized into its own module, as shown in figure 10.3.

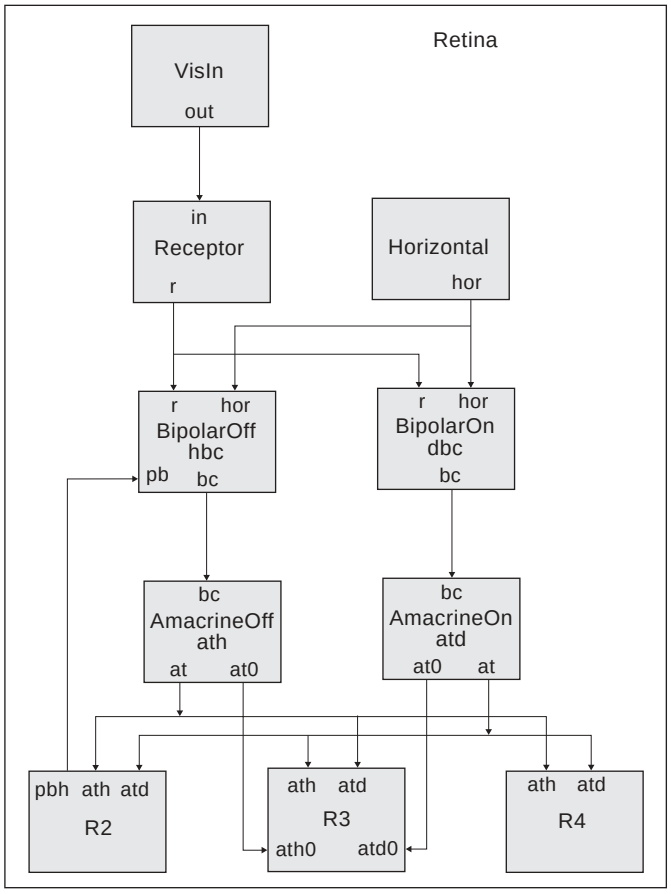


Figure 10.3
Retina module consisting of the following submodules interconnected between them: Stimulus, Receptors, Horizontal Cells, Bipolar Cells (on and off channel), Amacrine Cells (on and off channels) and ganglion cells R2, R3 and R4

Note that we use the same module definition for both on and off channel bipolars and Amacrines.

Visin

The Visin module uses a special input structure to simulate visual input (see Appendix III for details). Different kinds of moving (or static) stimuli may be defined interactively. In the **simRun** function the model simply needs to specify the `in.run()` function call for the actual computation to take place. Note that we initialize *in* to 0 previously to reset the visual input.

```
public void simRun()
{
   in = 0;   // need to reset all values first
   in.run(); // compute stimulus position according to pars
      set in file
   out = in; // export
}
```

Receptor

The Receptor module contains an input port *in* receiving visual input, while an output port *r* sends output to the following modules in the data path. The **initRun** method initializes *r* to 0. The **simRun** method sets the receptor *r* value to the input stimulus while time is less than 7.0 in order to stop it in the ERF. After that its value is sustained from its last value staying constant.

```
public void simRun()
{
if (system.getSimTime() <= 7.0)
       r = in;
}
```

Horizontal Cells

The horizontal cells get their input from the ambient light through *hLevel*. The **Horizontal** module has only a single output port *hor*. The **initRun** method initializes *hor* to 0. The **simRun** method computes a differential equation corresponding to a leaky integrator model for the horizontal cells

```
public void simRun()
{
   nslDiff(hor,tm,hLevel - hor);
}
```

Bipolars

Bipolar cells receive input from both the receptors and the horizontal cells. We describe a single Bipolar module for both the on and off channel cells. The distinction is made in terms of an *on_off* instantiation parameter. If "1" the cells are considered *Hyperpolarizing Bipolars* and if "-1" they are considered Depolarizing Bipolars. The **initRun** method initializes *bc* and *pb* to 0. The **simRun** method computes the bipolar cell activity *bc* as a difference between the values of the receptors and horizontal cells, with its sign depending on whether they are on or off channel cells. An additional output to the module is *pb* corresponding rectified value from the cell activity.

```
public void simRun()
{
   bc = on_off*(r - hor);
   pb = nslMax(bc,0);
}
```

Amacrines

Amacrine cells receive input from the bipolar cells. We describe a single Amacrine module for both the on and off channel cells. The distinction is made this time in terms of whether it receives input from an on or off channel bipolar cell. The **initRun** method initializes all variables to 0 and gets the value of the system delta to be used for numerical approximation (dt = system.getRunDelta()). The **simRun** method computes the amacrine cell activity *at* through an average exact method instead of the leaky integrator method.

```
public void simRun()
{
   // nslDiff(ax,tm, bc - ax);       // Euler method.
   ax = diff_ae(ax,bc,old_bc,tm);   // Average Exact Method.
   at1 = 5*(bc - ax);
   at2 = nslExp(-dt/tm.getData())*at;      // compute from
      previous at value
   at = nslMax(at1,at2);
   old_bc = bc;                     // keep old bc
}
```

The following average exact approximation method is used,

```
private NslFloat2 diff_ae(NslFloat2 v,NslFloat2 s,NslFloat2
      prev,NslFloat0 tm)
{
   float dt,tc,temp;
   int vmax = v.getRows();  // Size of the matrices.

   NslFloat2 term1(vmax,vmax);
   NslFloat2 term2(vmax,vmax);

   dt = system.getSimDelta();
   tc = tm.getData();

   if (dt != 0 && tc != 0){
      temp = exp(-dt/tc);
      term1 = (NslFloat2) ((1 - temp) * s + temp * v);
      term2 = (NslFloat2) ((s - prev) * (temp * (tc + dt) –
         tc) / dt);
   }
   return (term1 + term2);
}
```

Ganglion Cell R2

We model only ganglion cells R2, R3 and R4. All of them receive input from both the on and off channel amacrine cells. R2 in particular also receives input from the rectification of the on channel bipolar cell. The R2 module thus includes three input ports without any output port. It includes both an excitatory receptive field (ERF) and an inhibitory receptive field (IRF). The **initRun** method initializes the cell activity to 0. It also calculates the excitatory and inhibitory receptive fields through a Gaussian function. R2 in particular calculates the difference of gaussians (DOG) between *erf* and *irf* in *rf*.

```
public void initRun()
{
   r2 = 0; r2f = 0;
   nslGaussian(erf,erf_dia,erf_sig,erf_wgt); // Gaussian ERF
kernel
   nslGaussian(irf,irf_dia,irf_sig,irf_wgt); // Gaussian IRF
      kernel
   rf = erf - irf;           // DOG for the r2 ganglion cells
}
```

The **simRun** method computes a sustained *erf* (*sust_erf*) and *irf* (*sust_irf*) values from the *erf* and *irf* rectified bipolar cell input convolution, respectively. The cell activity *r2* is computed from a convolution of *r2* with the amacrine cell input. The output *r2f* is computed by a ramp function.

```
public void simRun()
{
    sust_erf = newconv(erf, pbh_erf * pbh); // sustained erf
       input
    sust_irf = newconv(irf, pbh_irf * pbh); // sustained irf
       input
    sust = sust_erf - sust_irf;                    // sustained
       input

    temp = ath + trailing * atd; // trailing is the effect of
       the trailing
                        // edge set to 0 to get Ewert's data
    r2 = newconv(rf, temp) + sust; // New convolution and No
       Leaky Integ.
    r2f = k*nslRamp(r2);
}
```

The following convolution method returning a 2d matrix of different size (in this example 1x1) is used:

```
private NslFloat2 newconv(NslFloat2& a, NslFloat2& b)
// a is the Mask and b is the input layer.
{
  int saimax = a.getRows();
  int sajmax = a.getCols();
  int sbimax = b.getRows();
  int sbjmax = b.getCols();

  int leftbound = 1;    // 32; for the 72x72
  NslFloat2 c(1,1); // Make this variable size // c(8,8) for
   72x72

  for (int i = 0; i < leftbound; i = i+4) {
    for (int j = 0; j < leftbound; j = j+4){
     float val = 0.0;
     for (int m = 0; m < saimax; m++)
       for (int n = 0; n < sajmax; n++)
        val = val + a[m][n] * b[i+m][j+n];
     c[i/4][j/4] = val;
    }
   }
  return c;
}
```

Ganglion Cell R3

The ganglion cells R3 are similar to R2 in that they receive input from both the on and off channel amacrine cells. The R3 module includes two input ports without any output port. It includes both an excitatory receptive field (ERF) and an inhibitory receptive field (IRF). The **initRun** method initializes the cell activity to 0. It also calculates the excitatory and inhibitory receptive fields through a Gaussian function.

```
public void initRun()
{
    r3 = 0; r3f = 0;
    nslGaussian(erf,erf_dia,erf_sig,erf_wgt); // Gaussian ERF
        kernel
    nslGaussian(irf,irf_dia,irf_sig,irf_wgt); // Gaussian IRF
        kernel
}
```

The **simRun** method computes an *all* input value from both amacrine cells for its *erf* while storing old values for its *irf*. The cell activity *r3* is computed from a convolution of *r3* with the amacrine cell input from *all* by its *erf* and *old* for its *irf*. The output *r3f* is computed by a ramp function.

```
public void simRun()
{
    all = p * atd + ath;
    old = p * old_atd + old_ath;

    r3 = newconv(erf, all) - newconv(irf, old);
    r3f = k*nslRamp(r3);
}
```

Ganglion Cell R4

The ganglion cells R4 are similar to R2 and R3 in that they receive input from both the on and off channel amacrine cells. The R4 module includes two input ports without any output port. It includes only an excitatory receptive field (ERF). The **initRun** method reinitializes the cell activity to 0. It also calculates the excitatory receptive field through a Gaussian function.

```
public void initRun()
{
    r3 = 0; r3t = 0; r3f = 0;
    nslGaussian(erf,erf_dia,erf_sig,erf_wgt); // Gaussian ERF
        kernel
}
```

The **simRun** method computes the cell activity *r4* by a convolution with the amacrine cell input difference by its *erf*. The output *r4f* is computed by a squashing function on *r4t* computed as a ramp function on *r4*.

```
public void simRun()
{
r4 = newconv(erf, (ath - atd));
    r4t = nslRamp(r4);
    r4f = k*r4t/(r4t+0.2); // Squashing function
}
```

10.4 Simulation and Results[3]

As previously mentioned, we do quantitative modeling of Anuran retina responses for stimulus shape and size dependency. In this simulations we test the model's ability to reproduce quantitatively tabulated data on the dependency on stimulus shape and size

(Ewert 1976). The goal has been to match Ewert's quantitative data on the Toad's retinal ganglion cells. Input to the model is Light on the receptors (40X40 to simulate the receptive field of a single ganglion cell of each type). The model also simulates "simple" Horizontal and Bipolar cells. The output of the model represents the temporal firing rate of a Ganglion cell of each type (R2, R3, R4). Note that there is no trailing-edge effect since Ewert computed his data with only the response to the leading edge. Furthermore, no Leaky Integrators we used for the Ganglion Cells.

Simulation Parameters

The simulation parameters include the *delta* and *endTime*

```
nsl set system.runDelta 0.066 ;# Simulation Time Step = 66
   msec.
nsl set system.runEndTime 7.0 ;# Total simulation time = 7 sec
```

Model Parameters

Model parameters are set for the different modules.

Horizontal Cell parameters:

```
nsl set retinaModel.retina.hor.tm 0.1
nsl set retinaModel.retina.hor.hlevel 0  ;# Uniform horizontal
   cell potential
         ;# 0 if the background is bright, 1 if dark.
```

Amacrine Cell parameters:

```
nsl set retinaModel.retina.ath.tm 0.3
nsl set retinaModel.retina.atd.tm 0.3
```

Ganglion Cell R2 parameters:

```
nsl set retinaModel.retina.r2.pbh_erf 0.3
nsl set retinaModel.retina.r2.pbh_irf 0

nsl set retinaModel.retina.r2.trailing 0 ;# Effect of trailing
   edge on R2.
nsl set retinaModel.retina.r2.k 43.8 ;# Scaling Factors for
   ganglion cells.

nsl set retinaModel.retina.r2.erf_dia 4.0 ;# R2 ERF diameter.
nsl set retinaModel.retina.r2.irf_dia 19.5
nsl set retinaModel.retina.r2.erf_sig 2.4 ;# R2 ERF sigmoid
   (for the Gaussian)
nsl set retinaModel.retina.r2.irf_sig 4.0
nsl set retinaModel.retina.r2.erf_wgt 1.0 ;# R2 ERF weight
nsl set retinaModel.retina.r2.irf_wgt 2.3     .
```

Ganglion Cell R3 parameters:

```
nsl set retinaModel.retina.r3.p 0.5  ;# Effect of trailing
   edge on R3
nsl set retinaModel.retina.r3.k 44.0
nsl set retinaModel.retina.r3.erf_dia 8.0
nsl set retinaModel.retina.r3.irf_dia 19.5
nsl set retinaModel.retina.r3.erf_sig 2.0
nsl set retinaModel.retina.r3.irf_sig 10.0
nsl set retinaModel.retina.r3.erf_wgt 1.15
nsl set retinaModel.retina.r3.irf_wgt 2.38
```

Ganglion Cell R4 parameters:

```
nsl set retinaModel.retina.r4.k 37.5
nsl set retinaModel.retina.r4.erf_dia 15.5
nsl set retinaModel.retina.r4.erf_sig 3.5
nsl set retinaModel.retina.r4.erf_wgt 1.0
```

Input Stimulus

Visual input stimulus plays an important role in the Retina model. To simulate this input the model uses the NSL input library which generates arbitrary sized 2D rectangles moving on the visual field as explained in Appendix III. To be able to incorporate this input the modeler needs to specify mapping parameters between the input and the receptor layer.

In the Retina model, the user has to choose among different types of stimuli. There are 15 options among Worms, Antiworms and Squares of 2, 4, 8, 16 and 32 visual degrees. These sizes are used in order to reproduce Ewert's data on presentation of Squares, Worms and Antiworms from 2 to 32 visual degrees. A particular concern on stimulus presentation relates to single cone receptors in the retina. While they have a density of about 5 to 30 cells per visual degree depending on their location (Carey, 1975) simulation tests have shown that a density of only 2 cells per visual degree allows sufficient accuracy for modeling responses to the stimuli considered here. When the stimulus edge partially covers a receptor, we set the receptor inputs to values proportional to the area covered by the actual (analytical/continuous) stimulus. The error from the edge effect is then about 4% relative to the analytical solution (Teeters, 1989).

We allow an arbitrary size and shape bitmap to represent our stimulus. In the simulations for the size and shape dependence of the ganglion cells, the velocity of the stimulus was set to 7.6°/sec so that the stimulus moves approximately 15 pixels in the grid each simulated second.

The following code in the "retina.nsl" file contains specification parameters for the visual input. The first values to be set are the distance between adjacent array elements in the receptor in mapping to the visual input coordinates (*dx* and *dy*) together with the origin of the coordinate system (*xz* and *yz*). Instead of 0.5°/cell we specify here 2°/cell for visualization purposes. This will affect the sizes of stimuli chosen in that we will make them 4 times as big to compensate for the enlargement.

```
nsl set retinaModel.retina.visin.input.dx 2
nsl set retinaModel.retina.visin.input.dy 2
nsl set retinaModel.retina.visin.input.xz 0
nsl set retinaModel.retina.visin.input.yz 20
```

For each stimulus we want to simulate on the visual field we choose its size and initial position. In the retina model, the user will choose among three types of stimuli with varying sizes, consisting of 5 sizes starting with 2 degrees. In table 10.3 we show three different experiments for the Retina model.

Stimulus	Initial Center Location (*xc,yc*)	Size (*dx,dy*)	Speed (*vx,vy*)
Antiworm	(-1,0)	(2,16)	(7.6,0)
Worm	(-8,0)	(16,2)	(7.6,0)
Square	(-8,0)	(16,16)	(7.6,0)

Table 10.3
Algorithms for the model of ganglion cells

Since we will be scaling values, as previously explained, by 4, we provide a corresponding *scale* variable as follows,

```
set scale 4
```

For a WORM stimulus we load the "worm.nsl" file:

```
set dx [expr 16*$scale]  ;# Size, 2 4 8 16 32
set dy [expr 2*$scale]  ;# CTE
set vx [expr 7.6*$scale] ;# Speed, number of squares per
   second, 7.6 deg/sec.
nsl create BlockStim stim -layer retinaModel.retina.visin.in \
   -spec_type center -xc [expr -$dx/2] -yc 0 -dx $dx -dy $dy –
      vx $vx
```

Note how we create a rectangle or "BlockStim" whose size is given by "dx" and "dy", its speed by "vx", all scaled by the *scale* factor, and whose initial center position is given by "-xc" and "-yc". The resulting temporal output for the three ganglion cells for a 16x2 worm is shown in figure 10.4.

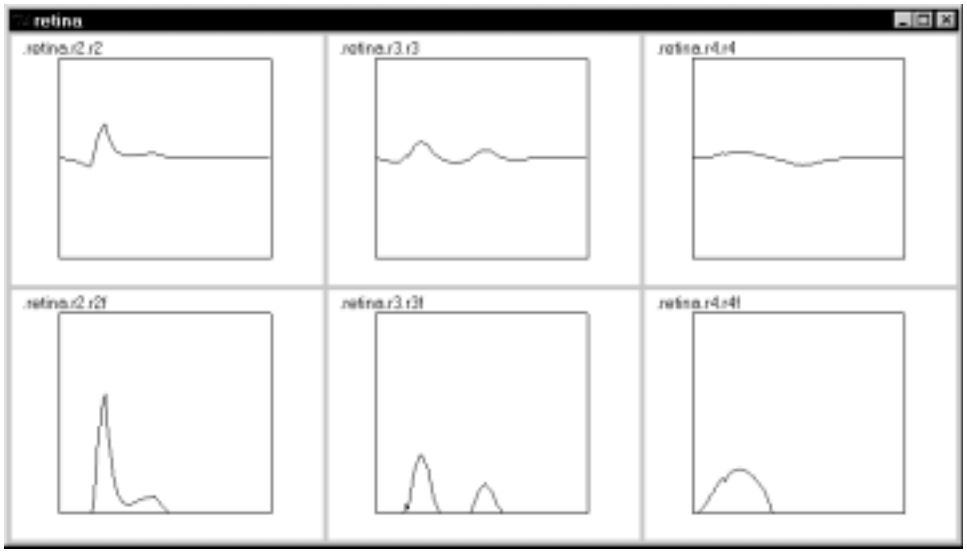
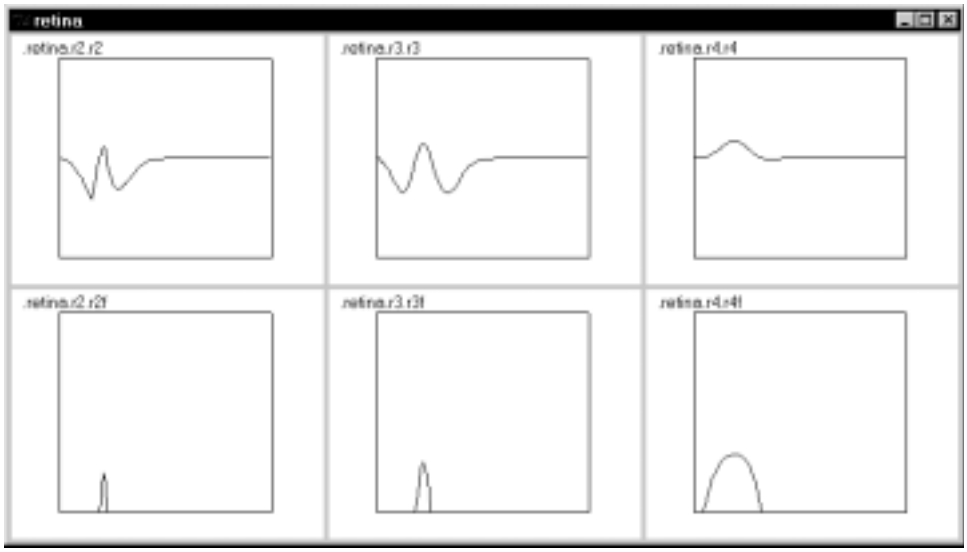


Figure 10.4
These graphs display cell activity (top level) and firing rate (bottom level) versus time in seconds for a 16x2 moving worm. Columns specify different types of ganglion cells, R2, R3 and R4.

For ANTIWORM as the stimulus we need to load the “antiworm.nsl” file:

```
   set dx [expr 2*$scale]
   set dy [expr 16*$scale]  ;# Size, 2 4 8 16 32
   set vx [expr 7.6*$scale] ;# Speed, number of squares per
second, 7.6 deg/sec.
   nsl create BlockStim stim -layer retinaModel.retina.visin.in \
      -spec_type center -xc [expr -$dx/2] -yc 0 -dx $dx -dy $dy -
vx $vx
```

Again we create a rectangle or “BlockStim” whose size is given by “dx” and “dy”, its speed by “vx”, all scaled by the *scale* factor, and whose initial center position is given by “-xc” and “-yc”. The resulting temporal output for the three ganglion cells for a 2x16 antiworm is shown in figure 10.5.



Antiworm: 2x16

Figure 10.5
These graphs display cell activity (top level) and firing rate (bottom level) versus time in seconds for a 2x16 moving antiworm. Columns specify different types of ganglion cells, R2, R3 and R4.

For a SQUARE as the stimulus we need to load the “square.nsl” file:

```
   set dx [expr 16*$scale]  ;# Size, 2 4 8 16 32
   set dy [expr 16*$scale]  ;# Size, 2 4 8 16 32
   set vx [expr 7.6*$scale] ;# Speed, number of squares per
      second, 7.6 deg/sec.
   nsl create BlockStim stim -layer retinaModel.retina.visin.in \
      -spec_type center -xc [expr -$dx/2] -yc 0 -dx $dx -dy $dy –
         vx $vx
```

The resulting temporal output for the three ganglion cells for a 16x16 square is shown in figure 10.6.

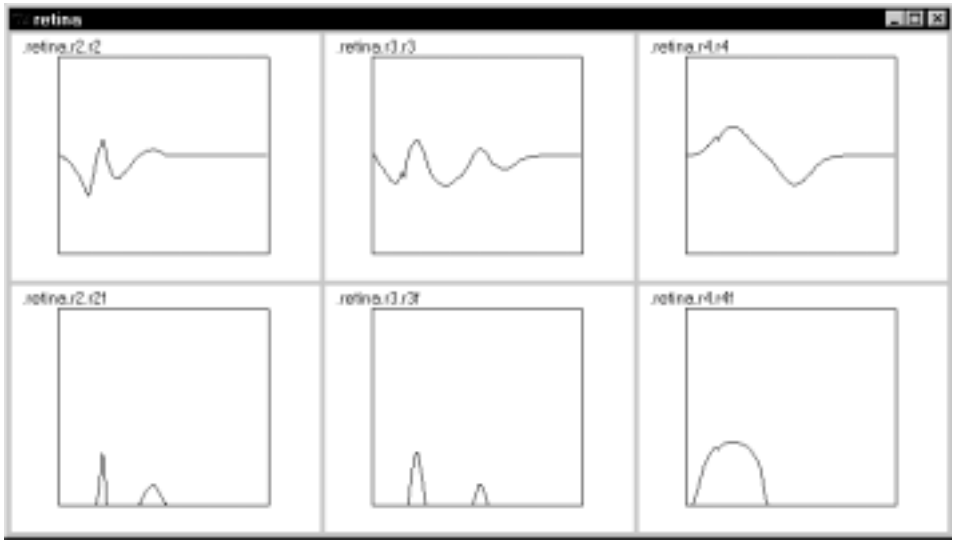


Figure 10.6
These graphs display activity (top level) and firing rate (bottom level) versus time in seconds for a 16x16 moving square. Columns specify different types of ganglion cells, R2, R3 and R4.

To allow comparison between the model behavior and tabulated data, the temporal responses of the ganglion cells generated by the model are converted to an average response that is then scaled. The average response is calculated as the area under the above threshold curve divided by the time from first to last above threshold response to the leading edge of the stimulus (or, in other cases, from the beginning of the leading edge response to the end of the trailing edge response):

$$\sum_{t=T_0}^{T_0} \frac{R(t)}{T_n - T_0} \tag{10.17}$$

where T_0 is the time for the first such response and T_n is the time for the last. If the response decays in an exponential manner and is not actively abolished, the response duration will be infinitely long. For that reason the threshold used is not zero but a small positive number (0.001).

The analogous experimental average is equal to the total number of spikes divided by the time from first to last spike during this period. Scaling is achieved by multiplying all calculated average responses by a "scaling constant" so that the scaled average response to a 2x2 square moving at 7.6°/second matches that found experimentally by Ewert (1976).

10.5 Summary

The essential features of the models presented in this paper enabling a close match to the stimulus shape and size dependency data were also used by several earlier models that attempted to explain those properties in anurans.

For example, the DOG center-surround structure was used to account for response in the R2 and R3 cells by an der Heiden & Roth (1987, 1989), Ewert & von Seelen (1974, also reported in Ewert 1976), and by Grüsser & Grüsser-Cornehls (1973). Variations in the temporal filter characteristics of retinal elements have been used by Eckmiller (1975), Grüsser (1967) and Grüsser et al. (1968) to account for variations in the velocity exponent. However, where the previous models were specialized to account for only particular phenomena, the models in this paper are not only able to account for the dependence on stimulus shape and size, but also able to account for the generation of characteristic ganglion cell response properties despite additional constraints applied to the ganglion cell models developed by Teeters and Arbib (1991). For the R2, R3 and R4

cell models given in Teeters and Arbib (1991), the response dependence on stimulus shape and size was tested in two parts. First, the original unmodified model was tested. Second, parameter adjustments and in some cases algorithm modifications, were made in an attempt to "tune" the model to attain a closer match to the experimental data on stimulus shape and size dependence. While the original untuned models did not quantitatively match the data, they were qualitatively correct.

Stimulus Size Dependence of R3 Cells

The Teeters and Arbib (1991) model does not match the data very well because the response to worms decreases with increasing edge length and there is a separation of the square and antiworm responses for large stimuli. Both of these effects are due to the inclusion of the slight trailing edge response generated by the model to long square and worm stimuli when calculating the average response.

Only minor changes are needed to tune the model. Excluding the trailing edge (and relying only on the leading edge) response for the calculation of the average responses (Ewert, personal communication) allows a good match to the data, although the response to the 32° square and antiworm is too large. Further, increasing the weight and the standard deviation of the IRF Gaussian mask and a little decrease in standard deviation of ERF mask allows a better match. This suggests that the IRF receptive field is essentially like a plateau, with a very small decay with distance, while the ERF receptive field is like a sharp peak. Comparison with the R2 temporal responses (Teeters et al., 1993) reveals that the main differences lie in (i) R2 responses show a sustained component for the long worm and a sustained rebound for the large square stimuli, while (ii) R3 responses show transient responses for both the leading and the trailing edges of the long worm and large square stimuli. The simulated temporal responses of R2 and R3 for the different stimuli approximate observed experimental data (Gaillard, personal communication) fairly well.

Predictions Based on the Modified Model Behavior

We now consider two important questions in detail: how do the changes made to the models here affect their ability to account for characteristics addressed by Teeters and Arbib (1991), and what predictions result from the changes?

- **R2 cell**: Characteristic R2 responses as identified by Maturana, Lettvin, McCulloch & Pitts (1960), and Grüsser & Grüsser-Cornehls (1976) are a lack of response to a diffuse light change, a lack of response to a moving antiworm longer than 10°, a prolonged response to a moving stimulus that stops in the ERF, a sensitivity to movement, a cessation of sustained response following a transient off of the general illumination, and a stronger response to small objects than to large objects. All of these characteristic responses but one are found in the toad R2 cells studied by Ewert & Hock—toad R2's respond to antiworm stimuli up to 16° in length. The tuning performed in this paper does not destroy the ability of the R2 model to account for these properties. Specifically, reduction in the IRF weighting to the R2 model will not allow response to full field flashes because the total IRF weighting is still larger than that of the ERF. Sensitivity to movement is preserved because the modified R2 cell receives input from transient amacrine cells that respond only to change. However, inclusion of PBH input to IRF leads to:
- **Prediction 1**: For R2 cells tested by Ewert & Hock (1972), while the average response to squares and antiworms of the same height may be almost identical, the temporal responses (spike trains) will be different as shown in figure 10.4.

Future refinements of the Retina Model

The shape/size tuned retinal model could be tested against other qualitative and quantitative data such as the average response as a function of velocity, adaptive state, etc. (Grüsser & Grüsser-Cornehls, 1976). In order to account for these data, we should probably incorporate more detailed physiological and morphological facts. Some of the most obvious ingredients could be:

1. A more detailed Horizontal cell model that is sensitive to the presentation of local stimuli.
2. Feedback loops among some layers (e.g., feedback from the amacrines to the bipolars).
3. Multi-compartmental dendritic processing and axonal transmission properties.

Also we have to note that the modeling of transient amacrine cells was based on phenomenological observations rather than on detailed physiological data on these cells. It might be possible to express the comparatively more responsive synaptic transfer process of R3s by, for instance, decreasing the amacrine time constant.

Teeters (1989) comments that the high-pass filter transient amacrine is unsuitable for the R4 cell model. This points out the need for an improved transient generating mechanism in the R4 cell. In retrospect, this is not surprising, because other properties of R4 cells, such as rhythmical bursting and delayed response to illumination decreases also cannot be accounted for easily by the high pass filter mechanism used in the model. Rhythmical bursting also occurs in some R3 cells (Maturana et al., 1960), suggesting that a high pass filter may also be inadequate to explain all of their response properties. Some type of negative feedback, with time delays or voltage dependent activation, is an obvious candidate mechanism that could generate oscillations in the neural potentials leading to bursting type response patterns. Further simulations will be needed to determine if such a mechanism can be made to simultaneously account for the characteristic R4 properties, the velocity exponent, rhythmic bursting, and the long response duration to a decrease in illumination. However, some of the characteristic R4 properties such as prolonged response to a stationary dark object and to the general illumination decrease can be achieved by incorporating the off channel bipolar inputs. In fact, Lee (1986) uses the sustained amacrine channel, PBH in our model, as the sole input to his R4 cells. It may also be possible to model the R4's large time constant for a moving object with the proper formulation of spatially sensitive horizontal cells.

Providing a flexible framework for modeling anuran retina

In summary, our current retina model cannot match all the experimental data, but does show how a relatively simple model can explain a wide range of ganglion cell properties. It also makes clear how, by changing parameter values of different inputs to the ganglion cells, the response properties of the ganglion cells will in turn change. For instance, when the weight of the input from the PBH to the IRF of R2 cells is increased, the previously described average response of the cell will diminish as well as the strength of the sustained response.

We should also note that retinal ganglion cells of the "same" type show a population of responses, as is elegantly shown in Gaillard's (personal communication, 1991) experiment on R3 type cells. Gaillard's result shows surprisingly large variances in ERF size, temporal activation patterns, etc., among the R3 cells. Similarly, we can expect that bipolars and amacrines will also form statistical distributions of responses. It may be that during embryogenesis a connection pattern from amacrines to a ganglion cell will be basically homogeneous, but that during postnatal development certain connections are strengthened while some are weakened thus giving the diversity among ganglion cells of

the same type. The fact that reciprocal connections exist between the bipolar cells and amacrines gives some hope that a similar connectivity may exist between amacrines and ganglion cells, which could provide information paths for selective strengthening and weakening required for diversity.

In our current model the amacrine population is represented by a layer of cells that share exactly the same properties. This has proven enough to match the experimental data described in this paper. But it is certain that the real retina contains several kinds of amacrine cells showing different properties, and this could promote higher variability in the response profiles of the ganglion population whose response depend on amacrine input. For instance, in our preliminary studies on the velocity dependence of ganglion cells we found it beneficial to decrease the high pass filter (amacrine) time constant from 300ms to 50ms for the R3 and R4 ganglion cells to yield a better fit to the quantitative data. This suggests that the amacrine time constant may be better represented as forming a statistical distribution such as a normal distribution centered at a "typical" value and that the amacrines feeding into the R3 consists mostly of the values in the lower spectrum. The populational approach could also be applied to the ganglion cells. Thus, we are led to place more emphasis on the variation of response properties in a population of neurons of the "same" class, rather than questing for "the" neuron of a given type.

One question that could arise when considering the populational approach is whether there exists an ill-defined boundary or just a "continuum" between different classes of ganglion cells. Should we construct a model so that it is possible for one category of cells to jump to another simply by, for instance, adjusting the "power" of a sustained input or the transient input? Gaillard (personal communication) has found "R3-like" units whose characteristic responses are similar to R3 units but whose velocity dependence is closer to that found in R2 ganglion cells. Their response profiles are stronger in intensity and temporally more extended than those of typical R3 units. R3s differ from R2s in that (i) their ERFs are larger, (ii) their ERFs receive no sustained input channel, and (iii) they have delayed IRF-inhibition. We believe the significance of these differences increases in the order listed above. We also think the more important a characteristic is, the less flexible are the parameters that make the characteristic. Notice that the "discrimination curves" of R2 and R3 cells to different stimuli are surprisingly similar. The main difference lies in a shift of the optimal length of the square (S) and the antiworm (A) from 4° (R2 units) to 8° (R3 units) and consequently in a shift of the crossing point between Worm (W), S, and A curves. This difference can be accounted for by a simple difference in the R3's ERF size and therefore we may predict that some R3 cells may have smaller ERFs so that their responses to dynamic visual stimuli are similar to R2 responses.

Notes

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2. A. Weitzenfeld developed the NSL3.0 version from the original NSL2.1 model implementation written by F. Corbacho as well as contributed Section 10.3 and part of section 10.4 to this chapter.
3. The Retina model was implemented and tested under NSLC.