

Relating Information Capacity to a Biophysical Model for Blowfly Retina

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Abstract

Our goal is to relate the structural and biophysical characteristics of blowfly visual neurons to their functional information processing aspects. Starting with the biophysics of information flow in the the early visual system of the blowfly, we construct a communication channel model that describes transmission and degradation of the visual signal in the photoreceptor and large monopolar cell. The channel model is a cascade of linear bandlimiting sections each followed by additive noise. Each section is modelled from first principles when possible, and parameters are determined from biophysical data available in the literature. The information capacity computed using our model compares favorably with empirical information rates derived from physiological experiments [5].

Sensory information processing in physical systems

We seek to gain better understanding of sensory information processing in physical systems both natural and engineered. To do so we must understand how to relate function to structure. We must also understand the tradeoffs between system performance and associated costs such as size, reliability and energy requirements. An information theoretic framework to quantify the tradeoffs in information processing in VLSI was presented in [2].

In this work we explore the relationship between structure and function in the blowfly retina. We construct a communication channel model that incorporates all physical transformations from photons at the photoreceptor to membrane voltage of the large monopolar cell in the lamina. This model allows us to investigate tradeoffs between cost and performance and provides a starting point for investigation into the efficiency of biological information processing.

The visual system of the fly

The visual system of the fly has been extensively

studied by physiologists. Vision in the blowfly (*Calliphora*) begins with two compound eyes which are each composed of a hexagonal array of ommatidia. Each ommatidium contains eight photoreceptors which receive light through a facet lens and respond in graded fashion to the incident light. Electrical signals from the photoreceptor cells project to cells in the lamina and the medulla. In this investigation we focus on the photoreceptors which project to large monopolar cells in the lamina.

The fly receives behaviorally relevant information as light reflected or emitted from objects in the environment. Photons are guided through optics distal to the photoreceptors. Absorption of photons activates photosensitive pigments in the photoreceptor cells. The activated pigments trigger a cascade of biochemical reactions which produce "messenger" molecules. These messengers cause ion channels in the photoreceptor membrane to open. The open channels provide a membrane conductance, which allows an ionic current to flow and changes the membrane voltage. This voltage change is propagated down a short axon to the lamina; there the membrane voltage modulates the release of neurotransmitter into the synaptic cleft, the space between the photoreceptor and the large monopolar cell (LMC). The neurotransmitter binds to postsynaptic receptors which are chloride channels. The open channels in the LMC membrane provide a conductance, which allows current to flow and changes the membrane voltage. This voltage change is propagated down the LMC axon to the synaptic terminal in the medulla. In the discussion that follows, we investigate the signals transduced through photoreceptors onto a single LMC, ignoring spatial aspects of information flow in the system.

A communication channel model

Information processing in the early visual system of the fly involves transformations between different physical degrees of freedom: photons, conformational state of proteins, concentrations of various chemical messengers, current, voltage. The goal of the above processes

is to communicate relevant information from one physical structure to another while preserving the message. We model these transformations as a cascade of communication channels that have bandwidth limitations.

Each of these transformations is associated with changes in the signal itself and with the inescapable introduction of noise. This begins even before transduction, as the arrival times of the photons are randomly distributed. Other sources of noise include the thermal activation of rhodopsin, the stochastic nature of channel transitions, and Johnson noise resulting from membrane impedance. We model each noise source as an independent, additive contribution to the channel.

In in our model the signal power $S_n(f)$ at any stage n is simply the result of a cascade of linear filters $H_i(f)$, and the noise power $N_n(f)$ is the summed power of m independent, additive noise sources $N_j(f)$ which are also transformed by the cascade of linear filters. Explicitly, the signal and noise at stage n are given by:

$$S_n(f) = \prod_{i=1}^n |H_i(f)|^2 S_p(f) \quad (1)$$

$$N_n(f) = \sum_{j=1}^m \prod_{i=k_j}^n |H_i(f)|^2 N_j(f) \quad (2)$$

where $S_p(f)$ is the power spectral density of the input signal and the noise from independent source j enters at stage k_j . The input to the system is the light reaching the photoreceptor as a function of time. We

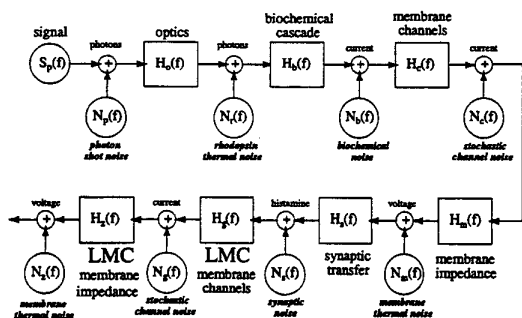


Figure 1: A communication channel model of the blowfly early vision.

explicitly model the filter characteristics and independent noise sources shown in Figure 1. We determine a linear filter characteristic for each stage, from first principles when possible and phenomenologically otherwise. These models are linear about an operating point, which is determined by the mean intensity of the incident light. Each noise source contributes independent, additive noise at the location in the system depicted.

For each noise source we determine the amplitude and power spectrum, also from first principles when possible and phenomenologically otherwise. While the cells under study exhibit nonlinearity [6], they have been studied as linear systems since at least 1965 [15], and their linear properties are well established in the literature [13]. Modelling the transfer functions as linear systems will be accurate when the variance of the signal is sufficiently small that the operating point remains fixed. This requirement is approximately satisfied for white noise stimulation protocols as in [5].

Channel capacity

The channel capacity of a system corrupted by Gaussian noise, in bits/sec, is given by [19]:

$$C = W \log_2 \left(1 + \frac{P}{N} \right) \quad (3)$$

when the noise variance is N , and the signal has bandwidth W and average power limitation P . This capacity is provided by a signal of Gaussian distribution. By extension for colored noise, the capacity becomes

$$C = \max_{S(f): \sigma_S^2 \leq P} \int_0^\infty \log_2 \left(1 + \frac{S(f)}{N(f)} \right) df. \quad (4)$$

Notice that the capacity is unaffected by an arbitrary filtering operation which affects signal and noise equally. It is, however, degraded by the addition of independent noise after such a filtering operation. It should be noted that Shannon capacity is an upper bound to the rate of information actually transmitted, and it assumes that the signal is limited only in average power and that the noise is normally distributed. Although these assumptions are not strictly true for this system, the fundamental Shannon limit provides a close approximation to the actual capacity.

Model details

With a communication channel model for the function and its relation to the structure established, we proceed to analyze the components of the model. This development is intended to elucidate the connection between the biophysics of transduction and the functional properties of signal filtering and degradation by noise. More details about the model components and parameters can be found elsewhere [1]. In the following, all spectra are considered to be single-sided, with frequencies ranging from 0 to ∞ .

Photons

Fundamentally light is a stream of randomly emitted photons, so the number of photons observed in any fixed time interval will vary about some average value. This variation can be thought of as "noise" superim-

posed on a signal which is the average number of photons.

We take the input signal to be the rate of photons reaching the eye as a function of time. This signal and its power spectral density $S_p(f)$ are determined by the environment. The distribution of observed samples about the mean value can be described by the Poisson distribution $P(k, \Delta t) = \frac{(I\Delta t)^k}{k!} \exp^{-I\Delta t}$ for $k = 0, 1, 2, \dots$, where I is the rate of the Poisson process. We take the photon noise to be the signal variance induced by photon shot noise. The power spectral density of the photon noise, in units of (photons/s)²/Hz, is given by

$$N_p(f) = 2I. \quad (5)$$

Optics

We consider the average intensity I to be an effective rate which accounts for optical spread and absorption, effective light intensity, and effective absorption rate of the photoreceptors. Thus the optical transfer is unity:

$$H_o^2(f) = 1. \quad (6)$$

Rhodopsin

The photosensitive pigment is rhodopsin, which consists of the chromophore retinal linked to the protein opsin. At low light intensities discrete bumps can be observed in the membrane voltage; each of these bumps results from current flow following the absorption of a single photon and resultant isomerization of rhodopsin [7].

Even in the absence of light, photoreceptors exhibit discrete electrical responses which are indistinguishable from single photon absorptions. Birge and Barlow [3] have suggested that an alternate reaction pathway is responsible for thermally generated events. We take the thermal isomerization rate to be $\lambda_r = 10^{-3}/s$, in accordance with empirical evidence [10, 17]. Thus for rhodopsin thermal noise, in units of (Rh*/s)²/Hz, we have

$$N_r(f) = 2 \cdot 10^{-3}. \quad (7)$$

Biochemical cascade

Each activation of a rhodopsin molecule triggers a cascade of biochemical reactions. This cascade ultimately produces molecules that modulate light-gated channels in the photoreceptor membrane. Changes in open probability translate into changes in conductance. The details of the phototransduction cascade in invertebrates remain unknown [25], thus our model for the biochemical cascade is phenomenological.

We model the transformation from activation of

rhodopsin molecules to membrane conductance as a linear filter $H_b(f)$. The biochemical cascade is considered to be a noiseless impulse response to each activated rhodopsin molecule. This treatment is essentially the same as the adapting bump model [23, 24]. The impulse response is modelled as a gamma function

$$h(t) = \frac{h_b}{n_b! \tau_b} \left(\frac{t}{\tau_b} \right)^{n_b} \exp\left(-\frac{t}{\tau_b}\right) u(t) \quad (8)$$

where h_b is an amplitude scaling factor, τ_b is a time scaling factor, n_b is a shape parameter, and $u(t)$ is the step function. The total current will be the convolution of this impulse response with the input, which is a sum of impulses each representing the activation of a rhodopsin molecule. The power spectral density of the visual signal is filtered by the transfer function $H_b^2(f)$, with units of (S/Rh*)², given by

$$H_b^2(f) = \frac{h_b^2}{[1 + (2\pi f \tau_b)^2]^{n_b+1}}. \quad (9)$$

The parameters of this transfer function have been estimated from physiological data of [12] and are summarized elsewhere [1]. We do not model noise contributed by the biochemical cascade, since the biophysical details of the cascade are unknown.

Stochastic Channels

In the dark adapted state, the membrane of the blowfly photoreceptor is at a resting transmembrane voltage of approximately $-60mV$. The transmembrane voltage V_m is defined as the potential inside the cell minus the external potential. When exposed to light it depolarises, quickly reaching a peak and eventually decaying to a steady state value. This steady state membrane voltage increases with light. The light-gated current is carried primarily by sodium and calcium ions. Potassium current opposes the voltage change induced by the light-gated flow.

Membrane channels are proteins which form pores through the cellular membrane. The pores can allow ions to flow in and out of the cell or prevent that flow, and this is described as being in an "open" or "closed" state. This physical mechanism transforms conductance, or the probability of channels being "open", into current across the membrane. This current flow is driven by the free energy difference across the membrane, and is proportional to that difference. The free energy is a sum of two terms, one due to the change in electrical potential across the membrane (V_m), and another due to the change in chemical potential, which results from the concentration gradient across the membrane ($-E_{ch}$). The transfer function from conductance to membrane current is given by $H_c(f)$, in units of Volts, and the power spectral density is multiplied by

the factor

$$H_c^2(f) = (V_m - E_{ch})^2. \quad (10)$$

Transitions between the states of a channel are stochastic. The transition probabilities can be modulated by the membrane voltage or by the presence of a ligand, as in the light-gated channels of blowfly photoreceptors. Fluctuations in the number of open channels result in noise in the membrane current. Channel kinetics lead to Lorentzian noise power spectral densities; for details consult [4, 11]. A simple channel with two states, open and closed, time constant τ_c , open probability n_∞ , single channel conductance γ_c and N independent channels, contributes current noise of the form

$$S_I(f) = \frac{S_I(0)}{1 + (f/f_c)^2} \quad (11)$$

$$S_I(0) = 4N\gamma_c^2(V_m - E_{ch})^2 n_\infty(1 - n_\infty)\tau_c \quad (12)$$

$$f_c = \frac{1}{2\pi\tau_c} \quad (13)$$

where $S_I(0)$ and f_c are parameters specifying amplitude and cutoff frequency, and the mean current is given by $\langle I \rangle = N\gamma_c(V_m - E_{ch})n_\infty$. The noise contributed by potassium channels is modelled according to Equation 11, using values reported in [22] and parameters estimated from the membrane impedance model described in the next section.

The opsin protein is very similar in *Calliphora* and *Drosophila*, exhibiting no difference in absorbance spectra and very similar amino acid sequences, especially in the cytoplasmic loops. The conserved structure of the cytoplasmic loops suggests similar conservation of the elements in the transduction cascade [9]. Hardie and Minke [8] found that the noise spectral density of the light-gated current in *Drosophila* was the sum of two Lorentzian components as in Equation 11. They do not estimate the maximum conductance, however, so an additional parameter is necessary to specify the magnitude of the channel noise. This magnitude is provided by the assumption that the open probability for the channels is low, so that the variance of the current is proportional to the mean [4]. This assumption seems to be satisfied in the experiments of Hardie and Minke. The resulting expression for the noise contributed by stochastic openings of the light-gated channels is

$$N_c(f) = \left[\frac{\frac{w_1}{w_2}}{1 + (2\pi f\tau_1)^2} + \frac{1}{1 + (2\pi f\tau_2)^2} \right] \frac{2\tau_1\tau_2}{\tau_1 + \frac{w_1}{w_2}\tau_2} \sigma_I^2 \quad (14)$$

where τ_1 and τ_2 are the time constants of the two components, $\frac{w_1}{w_2}$ gives their relative contributions, and σ_I^2 is the variance of the current.

Membrane impedance

The current which flows across the membrane is

transformed by the membrane impedance into the membrane voltage. Transfer of a signal within a cell is modelled using cable theory or compartmental modelling [18, 14]. Following van Hateren [20], we model the photoreceptor using three cable segments, two for the cell body and one for the axon. Each cable segment is considered as a two port, with axoplasm impedance z_a , membrane impedance z_m , and length l . The resulting transfer impedance Z_{tr} from input to synaptic terminal is given by

$$Z_{tr} = \frac{Z_{11}Z_{12}Z_{12}^a Z_t}{\{2Z_{11}Z_{11}^a Z_t + (2Z_{11}^2 - Z_{12}^2)(Z_{11}^a + Z_t) + \dots\}} \quad (15)$$

$$Z_{11} = \frac{z_a \cosh\left(\sqrt{\frac{z_a}{z_m}} l\right)}{\sqrt{\frac{z_a}{z_m}} \sinh\left(\sqrt{\frac{z_a}{z_m}} l\right)} \quad (16)$$

$$Z_{12} = \frac{z_a}{\sqrt{\frac{z_a}{z_m}} \sinh\left(\sqrt{\frac{z_a}{z_m}} l\right)} \quad (17)$$

where Z_{11} , Z_{12} , Z_{11}^a , and Z_{12}^a are the two-port impedances of the cell body and axon, and Z_t is the terminal impedance of the axon. The signal and noise power are filtered by the transfer function

$$|H_m(f)|^2 = |Z_{tr}|^2. \quad (18)$$

The membrane model consists of a capacitance C_m , light-gated conductance g_L with reversal potential E_L , leakage conductance g_{leak} with reversal potential E_{leak} , and potassium conductance g_K with dynamical parameters g_n and L_n and reversal potential E_K . The parameters g_n and L_n model the voltage dependence of the potassium channels. These membrane parameters contribute axoplasm impedance z_a and membrane impedance z_m of the form:

$$z_a = \frac{R_a}{\pi r^2} \quad (19)$$

$$z_m = \frac{\frac{1 + 2\pi j f L_n g_n}{SA}}{g_n + g_m - g_n C_m L_n (2\pi f)^2 + 2\pi j f (C_m + L_n g_n g_m)} \quad (20)$$

$$g_m = g_L + g_{leak} + g_K \quad (21)$$

where R_a is the axoplasmic resistivity, r is the radius, and SA is the surface area of the compartment, per unit length. For the axon this is just $2\pi r_a$, but for the cell body microvilli contribute much of the surface area, so the formula is $2\pi r_b + SA_m$. The parameters $g_L(V)$, $g_K(V)$, $g_n(V)$, g_{leak} , $L_n(V)$, r_b , l_b , and SA_m of the model have been estimated from physiological data and are summarized elsewhere [1].

Thermal equilibrium noise provides a fundamental lower limit to noise in any system. It is caused by thermal agitation of electrical charges. An arbitrary impedance $Z(f)$ contributes thermal voltage noise with

spectral density $N_V(f) = 4kTRe[Z(f)]$. Thus the thermal noise due to the photoreceptor membrane impedance is

$$N_m(f) = 4kTRe[Z_{tr}(f)]. \quad (22)$$

Synaptic Transfer

Synaptic transfer is modelled as a convergence of signals and noise. No filtering or additive noise is modelled. Six photoreceptors provide input to a single LMC. The signal, which is assumed to be correlated among the six photoreceptors, is increased by a factor of 6. The noise is independent among the six photoreceptors, and therefore upon convergence to the LMC the total noise is increased by a factor of $\sqrt{6}$. Power is increased by the factor

$$H_s^2(f) = \begin{cases} 36 & \text{for signal} \\ 6 & \text{for noise.} \end{cases} \quad (23)$$

LMC Membrane impedance

Following van Hateren and Laughlin [21], the LMC membrane impedance is modelled by an RC section followed by a two-port impedance. The transfer impedance Z_{tr} from input to synaptic terminal is

$$Z_{tr} = \frac{Z_t(Z_{cb} + Z_s + Z_{11} - Z_{12})}{Z_t + Z_{11} - Z_{12}} \quad (24)$$

where Z_{11} and Z_{12} are the two-port impedances of the axon, as defined previously, Z_{cb} is the impedance of the cell body, Z_s is the impedance of the synaptic zone, and Z_t is the terminal impedance of the axon. Equivalent circuits and parameters are as given in [21]. The signal and noise power are filtered by the transfer function

$$|H_z(f)|^2 = |Z_{tr}|^2. \quad (25)$$

The LMC membrane impedance contributes thermal noise:

$$N_z(f) = 4kTRe[Z_{tr}(f)]. \quad (26)$$

Results

Our model allows us to determine the signal, noise, and overall capacity at each stage of the system, for various operating points, thereby gaining a better understanding of the limiting processes as the signal and noise are transformed and various noise sources are added. We calculate capacity according to Equation 4. The capacity is plotted in Figure 2 as a function of incident light intensity. Empirical estimates from [5] are shown along with the results of the model for the photoreceptor and the LMC. Our model predicts information rates higher than the measured values, by $\approx 5 - 50\%$ for both the photoreceptor and the LMC. Undoubtedly some of the variability in the empirical

data results from differences among the individual neurons sampled, but it is also likely that our model does not yet account for all noise in the actual system.

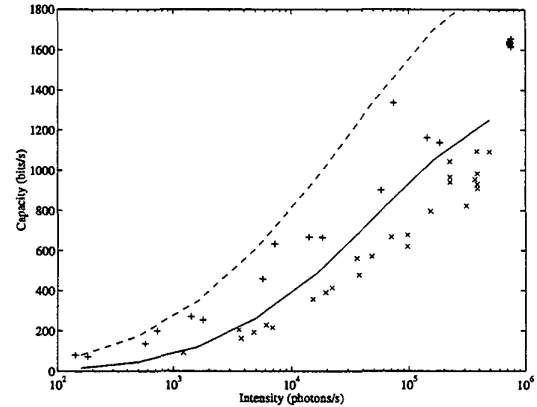


Figure 2: Information capacity computed from our model and estimated from experimental data [2]. '+'s (experimental estimates) and the dashed line (model) above them correspond to the LMC; 'x's (experimental estimates) and the solid line (model) above them correspond to the photoreceptor.

Our model also allows us to determine the dominant noise sources which limit the rates of information transmission. Figure 3 shows the output-referred noise, i.e. voltage noise at the LMC axon, for an incident intensity of 16000 effective photons/sec. Over the frequency range of physiological interest the dominant noise sources are photon shot noise and stochastic channel noise.

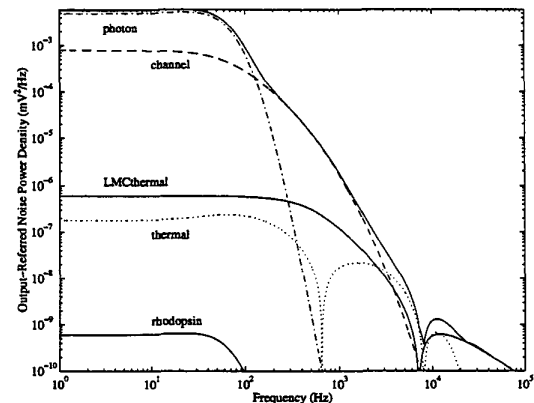


Figure 3: Contributions of independent noise sources to output noise.

Discussion

We analyze information processing in a communication system constrained by the physical components from which it is constructed, from photons to rhodopsin to biochemistry to membrane currents. The physical instantiation of the channel determines the noise, the signal constraints and the channel capacity. Such detailed analysis relates function to structure in a quantitative manner.

Information theoretic analyses typically consider communication between input and output for black box systems, but provide no insight into the mechanisms hidden within the box. We feel that it is important to understand neurobiology in terms of its fundamental and practical noise limitations. The models derived in the course of this work, furthermore, can be utilized to analyze tradeoffs between the various parameters of a biological system.

Once a quantitative measure of performance is established, i.e. capacity, its relation to costs such as power and constraints such as energy dissipation can be investigated. Biological systems are dissipative physical structures; signals are communicated by the flow of ions or other chemical substances, and some driving force must power this flow. Therefore communication and computation requires the dissipation of energy. The energetic cost of information processing in the blowfly retina has been reported to be as high as 10^7 ATP per bit[16], and our work predicts similar costs. We seek to understand how that energy expenditure is distributed across resources, and how different technologies and different ways of signal encoding are more or less energy-efficient.

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