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Information maximization as a principle for contrast gain control

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ABSTRACT

Contrast gain control has been found to be an important and common mechanism underlying the visual system's adaptation to the statistics of the visual scenes. Yet, the biophysical factors and computational rules governing its operation remain elusive. In this paper, we first studied the basic factors underlying contrast gain tuning in a neuronal model. We found that the nonlinearities (threshold and saturation), which are common to all spiking neurons, determines the preferred contrast sensitivity as well as the maximum information coding capacity of the neuronal model. We then investigated the design principles underlying adaptive gain control in various stimulus conditions, and found that an adaptive rescaling mechanism predicted by information transmission maximization can explain a variety of observed contrast gain control phenomena in neurophysiological experiments, including the divisive adaptation of the input-output function to mean contrast, and the inverse power law relation between response gain and input contrast. Our results indicated that the contrast gain control mechanisms in the visual systems may have a purpose of maximizing information encoding of input signals in varying environmental conditions.

INTRODUCTION

The visual systems exhibit great flexibility in adapting their input-output functions to the mean (Creutzfeldt, 1972; Werblin and Copenhagen, 1974; Walraven et al., 1990) and the variance (Shapley and Victor, 1978; Ohzawa et al., 1982) of luminance intensity in the visual environment. The amplitude gains of the transfer functions of visual neurons were found to decrease with input contrast (Shapley and Enroth-Cugell 1984). The relationship between the amplitude gain and the input contrast has been found to follow an inverse power law relationship (Truchard et al., 2000). In addition, the contrast response functions of visual cortex neurons were found to adapt to the mean contrast by shifting along the contrast axis to match the range of the prevailing input signals (Ohzawa et al., 1982; Geisler and Albrecht 1992). These phenomena are called contrast gain control and have been observed in many different types of neurons in sensory systems of many species, such as neurons in the retina (see reviews by Shapley and Enroth-Cugell 1984; Benardete and Kaplan, 1999), striate (Ohzawa et al., 1982), extrastriate visual cortex (Kohn and Movshon 2003) of mammals, and fly H1 neurons (Brenner, et al., 2000; Fairhall et al., 2001).

Recently, a number of biophysical and neural models have been advanced to account for contrast gain control, including the normalization model (Heeger 1992, Carandini et al., 1997), the synaptic depression model (Abbott et al., 1997) and a more recent model based on background excitatory and inhibitory synaptic modulation (Chance et al., 2002; Prescott and Koninck 2003). There are also intensive recent experimental efforts to isolate the various biophysical factors and mechanisms underlying contrast gain control. Biophysical factors that have been implicated in gain control include threshold (Heeger 1992; Sakai and Naka 1995), synaptic depression (Abbott et al., 1997),

synaptic noise (Chance et al., 2002), dendritic saturation (Prescott and Koninck 2003), long-term slow adaptation (Ohzawa et al., 1985; Smirnakis et al., 1997), and active ionic channels in the spike generation (Sanchez-Vives et al., 2000; Kim and Rieke, 2001, 2003). While it is possible that these multitudes of modulating biological and cellular mechanisms can co-exist to affect various aspects of contrast gain adaptation (Demb 2002; Piebe and Ferster 2002), the rules by which the various factors are adjusted to mediate gain control and the principles governing the determination of these factors remain unclear.

In this paper, we investigated the basic biophysical causes and the computational principles underlying contrast gain control by studying a cascade model of an adaptive linear kernel followed by a static nonlinearity. We first isolated the effect of gain tuning due solely to the static nonlinearity and then investigated the principles underlying the adaptive rescaling mechanism. We found that the amount of rescaling predicted by the principle of maximizing information transmission enables the model to reproduce many important aspects of the contrast gain control phenomena that cannot be explained by the static LN model alone, including the divisive adaptation of the contrast-response function to mean contrast (Ohzawa et al., 1985; Geisler and Albrecht 1992), and the power-law relationship in the gain-contrast curve (Truchard et al., 2000). This work therefore makes clear the relationships between static nonlinearity and gain tuning, and the relationships among adaptive linearity, contrast gain control and information maximization. Our results suggest that contrast gain control observed in neurophysiological experiments reflect an underlying adaptive mechanism that serves to maximize information encoding of the input signals in a dynamic environment.

MATERIALS AND METHODS

The static LN cascade model

It has been recognized that the static nonlinearity alone can potentially produce some change in effective gain as measured in neurophysiological experiments (Chander and Chichilnisky 2001, Schwartz et al., 2002; Pillow and Simoncelli, 2003; Paninski 2003). Understanding the scope of the influence of static nonlinearity in contrast gain control is important for dissecting the different aspects of the phenomena. To dissect factors necessary for the common properties of all spiking neurons and factors specific to neurons with contrast gain control properties, we begin with the basic linear-nonlinear (LN) model which captures two essential properties of sensory neurons: linear kernel and static nonlinearity.

[Figure 1 about here.]

The static LN cascade model (see Fig.1a) is given by a linear kernel function $h(t)$, followed by a static nonlinearity $g(.)$, which has been widely used to model the experimental phenomena (see a review by Meister and Berry 1999). Here, $h(t) = \sin(\pi t/\tau_a)\exp(-t/\tau_b)$ with τ_a = 80 ms and τ_b = 100 ms. Linear response $x(t)$ is given by

$$x(t) = \int_0^{+\infty} h(\tau)s(t-\tau)d\tau. \qquad (1)$$

The nonlinearity is specified by

$$y(t) = g(x) = \begin{cases} 0, & if \quad x < \theta, \\ x - \theta, & if \quad \theta < x < \eta, \\ \eta - \theta, & if \quad x \geq \eta. \end{cases} \qquad (2)$$

where θ is threshold and η is saturation level. *y(t)* is the response of the neuron. What are the exact effects of the static nonlinearity on the recovered transfer function *h'(t)* measured in neurophysiological experiments? What is the relationship between the real linear function *h(t)* and the recovered linear kernel *h'(t)* ? Attempts have been made by experimentalists to isolate the 'adaptive' effect due to static nonlinearity in the gain adaptation phenomena. In these studies, the transfer function of the neuron was usually obtained using the spike-triggered averaging methods (Chander and Chichilnisky 2001; Schwartz et al., 2002; Pillow and Simoncelli, 2003; Paninski 2003) or Wiener Kernel techniques and their extensions (Lee and Schetzen 1965; Korenberg 1988; Marmarelis 1993). In our derivation, we use *h'(t)* to indicate the recovered linear kernel, and *g'(t)* the recovered nonlinearity, so as to investigate the relationship between *h(t)* and *h'(t)*, *g(t)* and *g'(t)*.

Signals with Gaussian distributions are widely observed in nature and have been widely used as input signal to study neurons' response properties in experimental studies (e.g. Sakai et al., 1995; Smirnakis et al., 1997; Benardete and Kaplan, 1999; Truchard et al., 2000; Kim and Rieke, 2001; Rieke, 2001; Chander and Chichilnisky, 2001). Here, Gaussian white noise stimulus *s(t)* with mean μ and SD σ is used as input signal. Its probability density function (PDF) is given by $p_s = \frac{1}{\sqrt{2\pi\sigma^2}} e^{-\frac{s^2}{2\sigma^2}}$. The linear response *x(t)* also has a Gaussian distribution with PDF $p_x = \frac{1}{\sqrt{2\pi\sigma_x^2}} e^{-\frac{x^2}{2\sigma_x^2}}$, where σ_x is given by $\sigma_x^2 = < x^2(t) > = \sigma^2 \int_0^{+\infty} h^2(\tau) d\tau$, where <...> denotes time average.

The adaptive LN cascade model

In the second part of the paper, we will investigate the underlying factors and design principles for contrast gain control phenomena by the adaptive LN model. We introduce an adaptive rescaling mechanism to the LN model to investigate how the information transmission capacity of the neurons, as determined by their thresholds and saturation levels, can be fully utilized by an adaptive rescaling mechanism. This mechanism rescales the amplitude of the linear function by a factor β depend on input σ (Fig. 1b). With this addition, the linear kernel is given by $h(t) = \beta(\sigma)\sin(\pi t/\tau_a)\exp(-t/\tau_b)$. We call $\beta(\sigma)$ the adaptive rescaling factor, and the model the adaptive LN cascade model (Fig.1b).

Information theory

The amount of information transmitted by either the static or the adaptive LN cascade can be quantified using Shannon's information theory (Shannon and Weaver 1949). For a communication channel with input $s(t)$ and output $y(t)$, the total output entropy

$$H(y) = -\sum_{y} p(y)\log_2 p(y) \qquad (3)$$

quantifies the channel's theoretical limit on information transfer capacity, while the mutual information in discrete form (Dayan and Abbott 2001),

$$I_m = H(y) - H(y \mid s) = -\sum_{y} p(y)\log_2 p(y) + \sum_{s,r} p(s)\,p(y \mid s)\log_2 p(y \mid s), \qquad (4)$$

measures how much of that capacity is utilized to transmit and encode the input signal. H(y\mid s)

can be defined as noise entropy, accounting for the variability in the response that is not due to variations in the stimulus, but comes from noise sources. For simplicity, we consider the noiseless case, where $H(y \mid s) = 0$. In this case, the mutual information is set to be equal to the output entropy $I_m = H(y)$. The probability distribution of the output response $y(t)$ can be derived from Eq.[1] and [2]. We can compute the entropy of $y(t)$ directly from this distribution using Eq.[4].

RESULTS

Wiener kernel methods (Lee and Schetzen 1965; Korenberg 1988; Marmarelis 1993) were frequently used in neurophysiological experiments to identify or recover the transfer function of the neurons using Gaussian white noise input. It was found that the transfer function recovered from the sensory neurons can change in three ways, though not necessarily simultaneously: (1) a decrease in the gain of the kernel with an increase in signal variance (Shapley and Victor, 1978,1979,1980; Smirnakis et al., 1997; Truchard et al., 2000; Kim and Rieke 2001,2003; Rieke 2001; Chander and Chichilnisky 2001). (2) a dilation of the kernel size in space and time at lower contrast stimuli (Shapley and Victor, 1978,1979,1980; Smirnakis et al., 1997); and (3) a divisive adjustment of the contrast-response curve as a function of input mean contrast (Ohazawa et al., 1982, 1985; Geisler and Albrecht 1992; Kohn and Movshon 2003). Here, we will first examine the effect of the static nonlinearity on gain tuning and then discover the adaptive mechanism underlying the observed contrast gain control phenomena.

Effective gain tuning due to the static nonlinearity

To understand how static nonlinearity can modulate the effective transfer function of the cascade model, we derive an effective transfer function *h'(t)*, a function of input variance σ^2, the response threshold θ and the saturation level η. First, the Fourier transform of the output *x* of the linear kernel in the cascade model is given by

$$X(f) = H(f)S(f) \quad (5)$$

where *H(f)* and *S(f)* are the Fourier transforms of the linear kernel *h(t)* and the input signal *s(t)* respectively, and *f* is temporal frequency. According to Bussgang's theorem (Bendat 1990), for any memoryless nonlinear system *y = g(x)* with an input signal drawn from a Gaussian distribution, *K(f)*, the Fourier transform of the optimal linear transfer function, specifying the input-output relationship of the static nonlinearity *g(x)* is given by

$$K(f) = \frac{Y(f)X(f)^*}{X(f)X(f)^*} = \frac{< xg(x) >}{\sigma_x^2}, \quad (6)$$

where *X(f)* and *Y(f)* are the Fourier transforms of signal *x(t)* and output *y(t)* respectively, and * stands for conjugate.

For the entire static LN model, the optimal linear transfer function *T(f)*, the Fourier transform of the resultant linear kernel *h'(t)*, is given by

$$T(f) = \frac{Y(f)S(f)^*}{S(f)S(f)^*}, \quad (7)$$

where *S(f)* is the Fourier transform of input signal *s(t).* Combining Eqs.[3] and [4], and noting that *X(f) = H(f)S(f),* we have

$$T(f) = H(f)K(f) = H(f)\frac{< xg(x) >}{\sigma_x^2}, \quad (8)$$

where *H(f)* and *T(f)* are the Fourier transforms of the original linear kernel *h(t)* and the resultant first order Wiener Kernel *h'(t),* respectively. This indicates the entire effect of the static nonlinearity on the recovered kernel is simply introducing a gain scaling factor to the original linear kernel in the LN model. $\alpha = \frac{< xg(x) >}{\sigma_x^2}$ is called the gain factor.

Although we restrict ourselves here to the case of a Gaussian input signal, Eq.[6] can be easily generalized to handle the non-Gaussian case using Scarano's generalization of Bussgang's theorem (Scarano, 1991). Although this generalization requires that the nonlinearity *g(.)* be smooth, the threshold and saturation function used here can be approximated by nonlinearities that are smooth, such as a sigmoidal function.

Therefore, the resultant linear kernel *h'(t)* is given by $h'(t) = \alpha \cdot h(t)$ where gain factor α quantifies how the amplitude of the recovered linear kernel *h'(t)* is affected by the static nonlinearity (threshold θ, saturation η) and the standard deviation σ of the stimulus variation.

The gain factor α is estimated by

$$\alpha(\sigma) = \frac{< xg(x) >}{\sigma_x^2} = \frac{\int_{\theta}^{\eta} x(x-\theta)\, p_x dx + (\eta-\theta)\int_{\eta}^{+\infty} x p_x dx}{\sigma^2 \int_0^{+\infty} h^2(\tau) d\tau}. \qquad (9)$$

Performing the integrations and simplifying yields

$$\alpha(\sigma) = \frac{1}{2}[erf(\frac{\eta}{\sigma_x\sqrt{2}}) - erf(\frac{\theta}{\sigma_x\sqrt{2}})] = P[x(t) \in [\theta, \eta]]. \qquad (10)$$

[Figure 2 about here.]

The basic conclusion of this analysis is that the gain of the measured effective transfer function will change with input variance due to the effect of the static nonlinearity, even though the parameters of the LN model θ and η are fixed. To illustrate this phenomenon, we fix θ = 5 and η = 40, and plot the gain α for signals with different σ's according to the analytical equation. Fig.2a shows the resultant linear kernel *h'(t)* is heavily dependent on the value of σ. Interestingly, we found that α is not monotonic, it increases with σ in the small σ range, reaches a maximum, and decreases with a further increase in σ (circles in Fig.2b). To confirm these analytical results, we applied the standard Wiener kernel technique (Lee and Schetzen 1965; Korenberg 1988; Marmarelis 1993) to recover the linear Wiener Kernel for the whole static LN model based on the Gaussian white noise input *s(t)* and the output *y(t)* of the model. The amplitude gain of the recovered kernel (triangles in

Fig.2b) in this computational study is shown to match well with the prediction of our theoretical analysis. The recovered kernel $\hat{h}'(t)$ only exhibited gain scaling relative to the linear kernel $h(t)$. There is no temporal dilation or contraction of the linear kernel. The computational study therefore confirms the correctness of our theoretical results.

The gain tuning curve arises from the static nonlinearity and thus is a function of the threshold θ and the saturation level η. This gain tuning curve determines the neuron's preference to the distributions of different σ. The optimal σ_{opt} in which gain is maximum can be obtained by differentiating Eq.[10], which gives

$$\alpha_{opt}^2 = \frac{\theta^2 - \eta^2}{2(\ln\eta - \ln\theta)\int_0^{+\infty} h^2(\tau)d\tau} \quad . \qquad (11)$$

The obtained σ_{opt} increases with saturation η (see Fig.2c). It also increases slightly with an increase in threshold θ (Fig 2d). This might provide a mechanism and rules for a neuron to adjust its transfer function and gain tuning curve according to the statistical context of the input signals. However, the range of adjustment of the optimal σ by changing η and θ is rather limited. Based on this analysis, we conclude that in the discovering contrast gain control phenomena conventional kernel method (Lee and Schetzen 1965; Korenberg 1988; Marmarelis 1993) cannot avoid the nonlinear effect. Nonlinearity introduces a contrast gain tuning phenomenon, which is an intrinsic property of the nonlinear systems. This tuning property determines the preferred contrast sensitivity of the system to the input signals with various statistical distributions. However, note that the relationship between effective kernel gain and input variance in this LN model is non-monotonic in the general

case (Fig.2b). This is inconsistent with recent experimental findings, which showed that the recovered gain of the linear kernel decreases with the input contrast (Shapley and Victor 1978, 1979, 1980; Benardete and Kaplan 1999), and even displays an inverse power law relationship (Truchard et al., 2000; Chander and Chichilnisky 2001; Smirnakis et al., 1997). Thus, static LN model cannot explain the contrast gain control phenomena. To isolate gain change due to adaptive change in the system, one must factor out the effect due to static nonlinearity. Additional adaptive mechanisms may exist accounting for the interesting power law relationship. (Under extreme circumstances, if very strong input is presented to the static LN model that causes the neuron to saturate and fire at its maximal rate, it is possible for this model to follow the inverse power law relationship. However, this is not general case examined in experiments.)

Effective nonlinearity

Effective nonlinearity as defined in experimental studies (Chander and Chichilnisky 2001) is the relationship between the neuron's actual output *y(t)*, and the linear response *x'(t)* of the estimated (recovered) linear kernel *h'(t)*. Here we will demonstrate the relationship between the real nonlinearity *g(x)* and the recovered effective nonlinearity *g'(x')*.

Recall that $h'(t) = \alpha \cdot h(t)$, the generator signal $x'(t)$ is given by the convolution of the effective kernel with the stimulus, i.e.

$$x'(t) = \int_0^{+\infty} h'(\tau) s(t-\tau) d\tau. \qquad (12)$$

[Figure 3 about here.]

In Fig.3a, we plot the recovered effective nonlinearity for the static LN model for various values of σ. The curves shift to the left with an increase in σ. However, this shift in the effective nonlinearity $g'(x')$ curve is entirely due to the scaling of $h(t)$ by the gain factor α in a static LN model. When $x'(t, \sigma)$ is divided by gain factor $\alpha(\sigma)$, all the effective nonlinearity curves become superimpose together (Fig.3b). Thus, given that the static nonlinearity is invariant, one can estimate the gain factor α' that makes the rescaled effective nonlinearity $g'(x'/\alpha')$ overlap with $g(x)$. This is basically the intuition behind the neurophysiological experimental studies by Kim and Rieke (2001), Rieke (2001), and Chander and Chichilnsky (2001). They modeled the sensory neuron by cascade linear kernel and static nonlinearity for various input contrasts. Assuming the static nonlinearity is invariant with contrast, they identified the α' that scales the $g'_H(x)$ for high contrast to be equal to the static nonlinearity of the low contrast $g'_L(x)$. Then they computed linear filter $h'_L(t) = h'_H(t)/\alpha'$ and found that this adjusted $h'_L(t)$ is still different from the original $h_L(t)$ (see Fig.4 in the paper by Chander and Chichilnsky (2001)). Then they concluded that the gain change observed in the recovered linear kernel might be derived from an underlying contrast gain control mechanism. Our theoretical analysis makes clear the underlying assumptions of their approach and the implication of their results. Furthermore, our analytical results provide a means for estimating the underlying static nonlinearity $g(x)$ and the effective gain α in such neurophysiological experiments. These results clearly indicate that contrast gain control goes beyond effective gain change due to the static nonlinearity.

Invariant input-output relationship

Contrast response curve or the input-output relationship is useful for probing the contrast gain

control phenomena (Ohzawa et al., 1985; Geisler and Albrecht 1992). Gaussian white noise signal, while useful for deriving the effective linear wiener kernel, is inappropriate for estimating the input-output relationship of the system. This is because the temporal variation of the signal is faster than the convolution window of the cell's kernel, resulting in many-to-one input-output (I/O) mapping, and thus a fuzzy I/O curve. To recover the input-output relationship, experimenters typically used a stimulus that keeps an input attribute constant for a period of time Δt, and obtain the output by averaging the response of the neuron during that period (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992). For example, a spatial sinewave grating of a particular luminance contrast (the input attribute) will be drifted across the receptive field of the measured neuron. The temporal frequency of the sinewave grating is typically about 2 cycles per second. Gratings of several contrast values are presented for $\Delta t = 4$ seconds each, for a total of 10 times. These contrast values are chosen to be within one octave of a given mean value. This experiment is then repeated several times, with different mean contrast values (see Fig.1 in the paper by Ohzawa et al., 1985). For each mean value, a contrast sensitivity curve is plotted which gives the neural response for each contrast value. The response of the neurons was found to adapt to the mean contrast of the signals, causing the contrast sensitivity curve to shift to the right as the mean contrast is increased (see Fig.3 in the paper by Ohzawa et al., 1985). This adaptation of the contrast response function to mean contrast is a hallmark of contrast gain control.

[Figure 4 about here.]

Can static LN model produce the shifting contrast response relationship? To answer this question, we simulated this experiment with the static cascade LN model, using one-dimensional temporal sinewave gratings of different contrasts (see the red line in Fig.4a) as input to the neuron. Here, a

sinewave grating with a temporal frequency of 10 Hz can be considered the carrier signal (see Fig.4a), amplitude modulated by the input contrast signal *c(t)*. Signals modulated by each contrast *c(t)* is presented for Δt = 4 seconds. The contrast values are drawn from a Gaussian white distribution with standard deviation σ_c. This standard deviation determines the mean contrast level of the signal in each sequence, which lasts for 1000 seconds. The input-output curves are obtained from sequences of four different mean contrast levels, with σ_c = 1, 5, 10, and 20 respectively. To plot the input-output curve, the model's response for each time bin Δt is averaged to get a mean output value for each contrast value.

Fig.4b shows the input-output curves for the various mean contrast levels. The perfect overlap of the input-output curves indicate that the contrast sensitivity function for this static model does not shift with mean contrast level as observed in neurophysiological experiments. The slope of the input-output relation is independent of mean contrast level, and is not affected by the nonlinearity. Hence, the shifting of the contrast sensitivity function as observed in the similarly constructed neurophysiological experiments (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003) cannot be attributed to the static nonlinearity. There should exist adaptive mechanisms underlying contrast gain control phenomena and shifting contrast response curves. It should be noted that when the contrast variation is slow (i.e. Δt_c, the time unit of constant contrast σ is large) relative to the characteristic time scales of the linear kernel *h(t)*, then the contrast response function is invariant as in this simulation experiment. When the contrast variation is fast (Δt_c < 0.1 second) or when the luminance variation is too fast (i.e. temporal frequency of the sinewave > 20 Hz in each Δt_c) relative to the time scales of the linear kernel, then the input-output relationship of the system will be distorted to be fuzzy due to the convolution

effect discussed at the beginning of this subsection.

Information maximization principle

In the last section, we have showed that static nonlinearity can modulate the gain of the effective kernel as a function of σ, resulting in contrast gain tuning and that it determines the preference of the system to input distributions. However, we found it failed to account for two important neurophysiological observations concerning contrast gain control, i.e. (1) the shift of the contrast response function as a function of the mean contrast (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003); (2) the inverse power law relations between response gain and contrast (Smirnakis et al., 1997; Truchard et al., 2000; Chander and Chichilnisky 2001). What could be the mechanisms and principles that can account for these adaptation phenomena? Contrast adaptation phenomena have been interpreted in terms of the principle of information transmission maximization (Atick and Redlich 1992, Adorjan et al., 1999; Wainwright 1999; Brenner and Bialek 2000, Schwartz and Simoncelli 2001) from several perspectives. However, the analytical relationship between information maximization and contrast gain control is not clear. What is the relationship between gain tuning and information transmission in the static LN model and what is the relationship between the adaptation and maximal information transmission? In what sense can contrast gain control be understood in terms of information maximization? To answer these questions, we first analyzed information transmission quantitatively in the static LN model. Then we investigated how information transmission can be maximized for each signal distribution by adaptively rescaling the linear kernel in the LN cascade model. We will show in the following that the results of this adaptive rescaling can account for several important aspects of contrast gain control that static nonlinearity fails to explain. This result

therefore establishes an additional conceptual link between information maximization and various phenomena in investigation of contrast gain control.

Information transmission in the static LN model

The gain tuning effect due to the static nonlinearity can affect the information encoding process of the system. Now we use Shannon's information theory (see Eq.[4]) to quantify the information transmission of the LN model. We computed the mutual information I_m for various threshold, saturation and stimulus σ by Eq.[4]. Fig.5a shows that mutual information, in a way similar to effective gain, varies nonlinearly with input σ, exhibiting a tuning curve, with maximum at an intermediate σ. This optimal σ is corresponding to the maximum information transmission of the system, and is denoted by σ_{opt}'. For a given σ, the maximum I_m increases with an increase in saturation value or with a decrease in the threshold value. The tuning curve indicated that any nonlinear system with threshold and saturation properties can only best encode or transmit signals of a particular range of σ, and will not encode adequately signals outside this range without adaptation of its parameters. The variance-dependent mutual information tuning curve can be adjusted by modifying its threshold and saturation levels, in a manner similar to its adjustment of its variance-dependent gain tuning curve (see Fig.2c and 2d). In fact, mutual information I_m is roughly proportional to the gain factor α (see Fig.5b), suggesting that efficient information encoding and gain maximization are tightly correlated.

The above findings indicate that for any system like such a static model, only signals with preferred variance can be processed efficiently by the system. For the input with other variances, the information transmission capacity of the system cannot be fully utilized. Considering the visual

systems exhibit great flexibility to adapt themselves to various environment conditions, in the next section, we will explore an adaptive mechanism that will allow the system to fully utilize its transmission capacity regardless of the variance of the signals.

[Figure 5 about here.]

Information maximization in the adaptive LN model

Fig.5a shows that for a static LN model with given threshold and saturation, there exists an optimal input distribution with σ_{opt}' which can induce maximal information transmission in the LN system (recall that σ_{opt} maximizes gain). To maintain maximal information rate for any given input σ, we propose an adaptive mechanism that rescales the amplitude of the linear kernel in the LN cascade so that the output of the linear kernel x' is effectively adjusted to operate at the optimal regime of the given static nonlinearity. Let the rescaling factor be $\beta_{adapt}(\sigma)$, then the linear kernel for the adaptive LN model is given by,

$$h_A(t) = \beta_{adapt}(\sigma) * \sin(\pi t / \tau_a) \exp(-t / \tau_b) \qquad (13)$$

where $\beta_{adapt}(\sigma)$ is determined as the appropriate scaling factor necessary for maximizing information transmission for each input variance. The precise biophysical mechanism for mediating this effect is not known at presence, and presumably can be mediated by a variety of biophysical or network feedback mechanisms. Our primary task here is to elucidate the rules underlying the choice of the scaling factor, and the ramification of such a choice on the contrast gain control phenomena.

We propose that the adaptive rescaling mechanism essentially chooses $\beta_{adapt}(\sigma) = \beta_{opt}$ for each σ so that the maximum of the information transmission capacity I_{max} of the system can be reached. Note that, for the adaptive LN model,

$$x(t) = h_A(t) * s(t,\sigma) \quad (14)$$
$$= \beta_{adapt} h(t) * s(t,\sigma) \quad (15)$$
$$= h(t) * (\beta_{adapt}\, s(t,\sigma)) \quad (16)$$
$$= h(t) * s(t, \beta_{adapt}\sigma) \quad (17)$$

Therefore, choosing $\beta_{adapt} = \beta_{opt} = \sigma_{opt}' / \sigma$ produces an output $x(t)$ that is statistically equivalent to the output produced by the static linear model when exposed to stimulus with $\sigma = \sigma_{opt}'$. Thus, this definition of β_{opt} maximizes the information transmission for the adaptive LN model. Fig.6a shows that the information transmission for such an adaptive LN model is maintained at the highest level I_{max} independent of the variance of the signal input. Note that for the static LN model, I_m varies with σ, with only one global maximum I_{max} at a particular σ_{opt}' (see Fig.6a). The adaptive model thus ensures that the capacity of the system be fully utilized for any statistical inputs. The maximum of information transmission is constrained only by the threshold and the saturation level (Fig.6b). The lower (or higher) is the threshold (saturation), the higher is the maximum information rate.

[Figure 6 about here.]

Recent experimental works on H1 neurons of blowfly by Brenner, et al. (2000) and Fairhall et al., (2001) provided direct evidence that the scaling of the input-output relation is set to maximize information transmission for each distribution of signals. Fairhall et al., (2001) found that when the

variance of the input σ^2 was abruptly changed from one level to another, the information transmission rate dipped transiently, but then rapidly bounced back to the same constant level. While they have not established that this constant level was the highest possible level of information transmission rate, the fact that it is maintained at a constant level is consistent with our proposal that an adaptive mechanism is used to maintain information transmission at the highest capacity of the model for different signal distributions (Fig.6b).

The total response gain of the adaptive LN model here is defined as the amplitude of the recovered linear kernel from input *s(t)* and output *y(t)*. From Eq.[13], we know that total gain comes from two effects: gain due to the nonlinearity effect (see Eq.[9]), and gain due to the true adjustment $\beta_{adap}=\beta_{opt}$. Gain due to the nonlinearity, i.e., α, can be derived from Eq.[16] and Eq.[9], where we see that α is a constant for various input σ and equal to $\alpha(\sigma_{\text{opt}}')$. Thus, the total gain factor for the adaptive LN model (the gain between $s(t, \sigma)$ and $y(t)$) is

$$\gamma(\sigma) = \beta_{\text{opt}}\alpha(\sigma_{opt}^{'}) = \alpha(\sigma_{opt}^{'})\sigma_{opt}^{'} / \sigma \qquad (18)$$

Fig.6c demonstrates the inverse power-law relationship between input σ and the total gain γ of the adaptive LN model. This inverse power law relationship in the gain-variance curve has been observed in several recent experimental studies (Truchard et al., 2000, Smirnakis et al. 1997; Chander and Chichilnisky 2001). In particular, Truchard et al. (2000) decomposed the measured behaviors of the neurons into an adaptive linear kernel and a static nonlinearity. They found the gain of the recovered linear function of a cat simple cell and the contrast of the monocular input stimulus follows an inverse power-law relationship (Fig.6d). The prediction of the optimal γ -- σ

relationship (with slope -1) is superimposed on the Fig.6d. A slope of -1 indicates the adaptive system is completely effective in information maximization, while a slope of 0 indicates complete ineffectiveness. The measured simple cells exhibited a slope around -0.75, suggesting that the neurons, to a large extent, are re-scaling the gain adaptively to optimize information transmission. It is important to note that without the information maximizing adaptive rescaling, the relationship between the response gain and σ is a bell-shape curve (as shown in Fig.2) rather than an inverse power-law. Our analytical results therefore provide insights to the empirical findings of Truchard et al. (2000), illuminating the connection between the empirical inverse power-law observed and the principle of adaptive rescaling for information maximization.

Adaptation of the contrast response functions

We have shown earlier (see Fig.4) that for the static model, the contrast response functions (I/O curve) for various mean contrast levels σ_c are invariant. We now proceed to investigate the contrast response function in the adaptive LN model for the same setting. Can adaptive rescaling by β_{opt} in the adaptive LN model explain the observed adaptive shift in the contrast response curve as a function of the mean contrast? We repeat the computational experiment described in Fig.4a on the adaptive LN model. β_{opt} in the model is determined by information maximization based on each input mean contrast level. Fig.7a shows that the contrast response functions, i.e., $c(t) \sim y(t)$ curves, change their slopes for four different mean contrast levels (σ_c = 1, 5, 10, and 20 respectively). In a log-log plot, this change in slope is manifested as a horizontal shift in the contrast response curve (Fig.7b). This behavior is qualitatively similar to the neurophysiological observations (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003). When the input contrast is divided by the mean contrast σ_c, the contrast response functions become

superimposed on top of each other, demonstrating that the adaptation is a divisive effect. Thus, the predicted rescaling of the linear kernel based on information maximization can explain the divisive contrast response curves observed in a lot of neurophysiological experiments (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003). A similar rescaling of input-output relations has also been observed by Brenner et al., (2000) in their experiments on H1 neurons of blowfly. Our theoretical results thus demonstrate the underlying connection of these experimental findings on contrast gain control from an information theoretical prospective.

[Figure 7 about here.]

DISCUSSION

In this paper, we investigated the basic biophysical causes and the computational principles underlying contrast gain control phenomena. We first studied a model that is composed of a linear kernel followed by a static nonlinearity, called a linear-nonlinear (LN) model. The LN cascade model is an abstraction that captures the essential properties of sensory neurons. The linear kernel captures the receptive field, and the static nonlinearity captures the two basic features common to all spiking neurons -- response threshold and saturation level. Thus, it is widely used in both computational studies and the analysis of neurophysiological data (Meister and Berry, 1999). Recent theoretical studies (Chander and Chichilnisky 2001; Kim and Rieke 2001; Rieke 2001; Schwartz et al., 2002; Pillow and Simoncelli, 2003; Paninski 2003; Yu and Lee 2003) revealed that many of the observations from earlier neurophysiological experiments on gain adaptation cannot cleanly decompose the effect due to the static nonlinearity and the effect due to adaptive adjustment

of the underlying parameters of the system. Here, we isolated the gain tuning effect of static nonlinearity through theoretical analysis and simulation to understand which aspects of the gain control phenomena can be explained by static nonlinearity and which aspects have to be addressed by additional mechanisms. Our results show that static nonlinearity alone can produce gain tuning and information tuning as a function of input variance. This tuning effect is indeed belongs to the coherence resonance (CR) phenomena observed in various physical, chemical and biological systems (Gammaitoni et al., 1998). A common feature of CR is that the coherence measure or response property in the output of a complex system can be maximized by the fluctuating signal with optimal variance. Our results provide a basic framework for the CR phenomenon in a static nonlinear system. That is, for any system with threshold and saturation, their information coding properties can potentially display a CR phenomenon. In the discovering the adaptive behaviors of the sensory systems, we should factor out components of gain modification due to the CR from the real adaptive mechanisms.

Recent studies (Truchard et al., 2000; Chander and Chichilnisky 2001) suggested that the adaptive behaviors in contrast gain control experiments can be simply modeled by an adaptive linear kernel cascaded with a static nonlinearity. The effective gain of the linear kernel and the contrast of the input signals exhibit an interesting power law relationship. However, the underlying reasons for this behavior of the linear kernel, and the relationship between the kernel adaptation and information transmission are not clear. Here, we investigated the hypothesis that the adaptive linear rescaling is governed by the principle of information maximization. For any signal with a given variance, the linear kernel amplitude can be adjusted to an optimal level by an adaptive mechanism so that the information transmission is maximized. This model is successful in reproducing three

important phenomena observed in previous experiments related to contrast gain control: 1) the logarithmic decay of the linear kernel gain with the input contrast (Smirnakis et al., 1997; Truchard et al., 2000; Chander and Chichilnisky 2001); 2) the divisive adjustment of the contrast response functions in adaptation to different mean contrast levels (Ohzawa et al., 1982,1985; Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003); 3) the rescaling input/output relationship (Brenner, et al., 2000; Fairhall et al., 2001) for maximal information transmission, as well as the invariant information transmission for various input contrasts (Fairhall et al., 2001). Our theoretical work therefore provides a coherent framework for understanding why the various experimental observations listed above are in fact evidences in support of the proposal that contrast gain control is a mechanism for information maximization.

The principle of information maximization has been proposed in various theoretical frameworks for reasoning about sensory neuronal receptive field development (Linsker 1991; Atick and Redlich 1992; Olshausen and Field 1996; Bell and Sejnowski 1997; Adorjan et al., 1999; Wainwright 1999), contrast adaptation in retinal ganglian cells (Atick and Redlich 1992), contrast gain control by normalization mechanisms in the primary visual cortex (Schwartz and Simocelli, 2001), and been hypothesized to play a role in the adaptive rescaling of the input-output functions in fly H1 neurons (Brenner, et al., 2000; Fairhall et al., 2001). Our contribution lies in providing a theoretical framework, with a simple model, to illuminate how effective gain control due to static nonlinearity can be factored out, and how the variety of observed phenomena related to contrast gain control can be reduced to an adaptive rescaling mechanism dictated by the principle of information maximization. Therefore, our work unifies the understanding of all these phenomena by a common design purpose and principle, and provides a theoretical framework for future experimental design

and data analysis in contrast gain control study.

It is important, however, to note that there are elements of gain control phenomena that are not explained by the information-maximizing adaptive LN model discussed here. For example, the linear kernel has been found to dilate or contract in time during dark or light adaptation, resulting in a change in the temporal frequency tuning of the neurons (Shapley and Victor, 1978,1979,1980). These effects can arise from dynamic change in the parameters of the system, such as the time constants (Pillow et al., 2003), active ion-channels (Sanchez-Vives et al., 2000; Rieke, 2001; Kim and Rieke, 2001,2003), or the stochastic bifurcation dynamics of a more complex neuronal model neuronal model (Yu and Lee 2003) and cannot be accounted for by the current adaptive LN model. Another widely observed phenomenon that cannot be explained by the simple adaptive LN model is the decrease of response gain or response saturation level, which might be related to long-term adaptation process (tens of seconds) (Geisler and Albrecht 1992; Sanchez-Vives et al., 2000; Kohn and Movshon 2003). Further model investigation may need to involve a firing rate adaptation process (Wilson and Humanski 1993; Smirnakis et al., 1997; Sanchez-Vives et al., 2000).

What biophysical processes are responsible for mediating the adaptive rescaling proposed in our model remains an open question. Recent works on the biophysical mechanisms of contrast gain control have revealed many possible mechanisms (Heeger, 1992; Carandini et al., 1997; Holt and Koch 1997; Abbott et al., 1997; Chance et al., 2002; Prescott and Koninck 2003) for mediating some basic contrast gain control phenomena such as the divisive shift in input-output relations. Biophysically, a single neuron itself is a circuit, whose transfer function is controlled by the ionic conductances and capacitances. A recent study by Stemmler and Koch (1999) has demonstrated

that how the voltage dependence conductances of neurons can be adaptively adjusted to maximize the information encoding for various inputs. The changing in conductances indeed reflects changes in transfer function. In fact, numerous biophysical factors have been implicated in neurophysiological experiments or conjectured in computational models, which may relate to adapting transfer functions of the sensory neurons. They include the activation of the Na^+-activated and Ca^{2+}-activated K^+ currents inside single neurons (Sanchez-Vives et al., 2000; Rieke 2001; Kim and Rieke 2003; Bacccus and Meister 2003), normalization process (Heeger, 1992; Schwartz and Simoncelli 2001), synaptic depression (Abbott et al., 1997; Adorjan et al., 1999), background excitation and inhibitory synaptic modulation (Chance et al., 2002; Prescott and Koninck 2003), dendritic adjusting (Prescott and Koninck 2003) and long-term slow adaptation (Ohzawa et al., 1985; Wilson and Humanski 1993; Smirnakis et al., 1997; Sanchez-Vives et al., 2000). These proposals, are concerned primarily with the biophysical and cellular implementation of gain control. While the exact mechanisms and implementations of contrast gain control depend highly on the specific systems, organisms and cellular networks, they might all be subjected to the same set of computational principles and purpose. Our work provides insight and understanding on contrast gain control at the computational theoretic and algorithmic levels. It reveals that information maximization might be a central design principle governing the operation of the adaptive nervous systems.

REFERENCES

Abbott LF, Varela JA, Sen K, Nelson SB (1997) Synaptic Depression and Cortical Gain Control. Science 275:220-223.

Adorjan P, Piepenbrock C, Obermayer K (1999) Contrast adaptation and infomax in visual cortical neurons. Rev Neurosci 10:181–200.

Atick JJ, Redlich AN (1992) What does the retina know about natural scenes? Neural comput 4:196-210.

Baccus SA, Meister M (2002) Fast and slow contrast adaptation in retinal circuitry. Neuron 36:909-919.

Bell AJ, Sejnowski TJ (1997) The 'independent components' of natural scenes are edge filters. Vision Res 37:3327-3338.

Benardete EA, and Kaplan E (1999) The dynamics of primate M retinal ganglion cells. Visual Neuroscience 16:355-368.

Bendat JS (1990) Nonlinear system analysis and identification from random data. (John Wiley and Sons, New York).

Brown SP, and Masland RH (2001) Spatial scale and cellular substrate of contrast adaptation by retinal ganglion cells. Nature Neuroscience 4:44-51.

Brenner N, Bialek W, and de Ruyter van Steveninck R (2000) Adaptive rescaling maximizes information transmission. Neuron 26:695-702.

Carandini M, Heeger DJ, Movshon JA (1997) Linearity and normalization in simple cells of the macaque primary visual cortex. J Neurosci 17:8621-8644.

Chance FS, Abbott LF, Reyes AD (2002) Gain modulation from background synaptic input. Neuron 35:773-782.

Chander D, Chichilnisky, EJ (2001) Adaptation to temporal contrast in primate and salamander retina. J Neurosci 21:9904-9916.

Creutzfeldt OD, (1972) Transfer function of the retina. Electroencephalogr. clin. Neurophysiol.

Suppl. 31:159-169.

Dayan P, Abbott LF (2001) Theoretical Neuroscience (MIT Press, Cambridge) Chap.4, pp.129.

Demb JB (2002) Multiple mechanisms for contrast adaptation in the retina. Neuron 36:781-783.

Fairhall AL, Lewen GD, Bialek W, de Ruyter van Steveninck R (2001) Efficiency and ambiguity in an adaptive neural code. Nature 412:787-792.

Gammaitoni L, Hanggi P, Jung P, and Marchesoni F (1998) Stochastic reaonance. Review of Modern Physics 70:223-287.

Geisler WS, Albrecht DG (1992) Cortical neurons: isolation of contrast gain control. Vision Res 8:1409-1410.

Heeger DJ (1992) Normalization of cell responses in cat striate cortex. Vis Neurosci 9:181-197.

Holt GR, Koch C (1997) Shunting inhibition does not have a divisive effect on firing rates. Neural Comput 9:1001-1013.

Kim KJ, Rieke F (2001) Temporal contrast adaptation in the input and output signals of salamander retinal ganglion cells. J Neurosci 21:287-299.

Kim KJ, Rieke F (2003) Slow Na+ inactivation and variance adaptation in salamander retinal ganglion cells. J. Neurosci 23:1506-1516.

Kohn A, Movshon JA (2003) Neuronal adaptation to visual motion in area MT of the macaque. Neuron 39:681-691.

Korenberg MJ (1988) Identifying nonlinear difference equation and functional expansion representations: The fast orthogonal algorithm. Ann Biomed Eng 16:123–42.

Koutalos Y, Yau KW (1996) Regulation of sensitivity in vertebrate rod photoreceptors by calcium. Trends Neurosci 19:73–81.

Lee YW, Schetzen M (1965) Measurement of the Wiener kernels of a non-linear system by cross-

correlation. Int J Control 2:237–254.

Linsker R (1997) A local learning rule that enables information maximization for arbitrary input distributions. Neural Comput 9: 1661-1665.

Marmarelis VZ (1993) Identification of nonlinear biological systems using Laguerre expansions of kernels. Annals of Biomedical Engineering 21:573-589.

Meister M, Berry MJ (1999) The neural code of the retina. Neuron 22:435-450.

Ohzawa I, Sclar G, Freeman RD (1982) Contrast gain control in the cat visual cortex. Nature 298:266-268.

Ohzawa I, Sclar G, Freeman RD (1985) Contrast gain control in the cat's visual system. J Neurophysiol 54:651-667.

Olshausen BA, Field DJ (1996) Emergence of simple-cell receptive field properties by learning a sparse code for natural images. Nature 381:607-609.

Paninski L (2003) Convergance properties of three spike-triggered analysis techniques. Network-Comp Neural 14: 437-464.

Pillow JW, Simoncelli EP (2003) Biases in white noise analysis due to non-Poisson spike generation. Neurocomp 52-4:109-115.

Priebe NJ, Ferster D (2002) A new mechanism for neuronal gain control. Neuron 35:602-604.

Prescott SA, De Koninck Y (2003) Gain control of firing rate by shunting inhibition: Roles of synaptic noise and dendritic saturation. P Natl Acad Sci USA 100:2076-2081.

Rieke, F. (2001) Temporal contrast adaptation in salamander bipolar cells. J. Neurosci, 21, 9445-9454.

Sakai HM, Naka KI (1995) Response dynamics and receptive-field organization of catfish ganglin cells. J Gen Physiol 105:815-835.

Sanchez-Vives MV, Nowak LG, McCormick DA (2000) Membrane mechanisms underlying contrast adaptation in cat area 17 in vivo. J Neurosci 20:4267–4285.

Scarano G (1991) Cumulant series expansion of hybrid nonlinear moments of complex random-variables. IEEE Transactions on Signal Processing 39:1001-1004.

Shannon CE, Weaver W (1949) The Mathematical Theory of Communication, (Ur-bana, IL: Univ. of Illinois Press).

Shapley RM, and Victor JD (1978) The effect of contrast on the transfer properties of cat retinal ganglion cells. J Physiol 285:275-298.

Shapley RM, and Victor JD (1979) The contrast gain control of the cat retina. Vision Res 19:431–434.

Shapley RM, and Victor JD (1980) The effect of contrast on the response of the Y cells. J. Physiol. 302:535–547.

Shapley RM and Enroth-Cugell C. (1984). Visual adaptation and retinal gain control. Progress in Retinal Research, 3:263–346.

Schwartz O and Simoncelli EP (2001) Natural signal statistics and sensory gain control. Nature Neurosci 4:819-825.

Schwartz O, Chichilnisky EJ, Simoncelli EP (2002). Characterizing neural gain control using spike-triggered covariance. Adv. Neural Information Processing Systems 14:269-276.

Smirnakis SM, Berry MJ, Warland DK, Bialek W, and Meister M (1997) Adaptation of retinal processing to image contrast and spatial scale. Nature 386:69-73.

Stemmler M, Koch C (1999) How voltage-dependent conductances can adapt to maximize the information encoded by neuronal firing rate. Nature Neurosci 2:521-527.

Truchard AM, Ohzawa I, and Freeman RD (2000) Contrast Gain Control in the Visual Cortex:

Monocular Versus Binocular Mechanisms. J Neurosci 20:3017 - 3032.

Walraven J, Enroth-Cugell C, Hood DC, MacLeod DIA, Schnapf JL (1990) The control of visual sensitivity. In: Visual perception: the neurophysiological foundations. (Spillmann L, Werner SJ, eds), pp53–101. San Diego: Academic.

Wainwright MJ (1999) Visual adaptation as optimal information transmission. Vision Res 39:3960-3974.

Walraven J, Enroth-Cugell C, Hood DC, MacLeod DIA, Schnapf JL (1990) The control of visual sensitivity: receptoral and postreceptoral processes. In: The neurophysiological foundations of visual perception (Spillman L, Werner J, eds), pp 53-101. New York: Academic.

Werblin FS, Copenhagen DR (1974) Control of retinal sensitivity. III. Lateral interactions at the inner plexiform layer. J Gen Physiol 63:88-110.

Wilson HR, Humanski R (1993) Spatial frequency adaptation and contrast gain control. Vision Res 33:1133–1149.

Yu YG, Lee TS (2003) Dynamical mechanisms underlying contrast gain control in single neurons. Phys Rev E 68: 1901-1907.

FIGURE CAPTIONS

Figure 1

(a) The static LN model is consisted of a linear filter *h(t)* followed by a static nonlinearity *g(.)*. *x(t)* is the response the linear filter, produced by convolving input *s(t)* with the filter transfer function *h(t)*. The nonlinearity *g(.)* acts on *x(t)* to generate the system response *y(t)*. (b) The adaptive LN

model is consisted of a linear filter *h(t)* followed by a nonlinearity *g(.)*; the amplitude of the linear filter *h(t)* is controlled by a rescaling factor β, which acts as an adaptive mechanism.

Figure 2

Effective gain of the static LN model. (a) Effective kernels for a LN model with threshold $\theta = 3$ and saturation $\eta = 50$ recovered from input stimuli of different σ. (b) Effective gain α is a function of σ, exhibiting a gain tuning curve. This tuning curve is predicted by the theoretical analysis, and is confirmed by the simulation results. (c) The σ where α reaches maximum is called $\sigma_{opt.}$ σ_{opt} increases linearly with saturation level η for different threshold values (θ = 3, 6 and 10). (d) For fixed saturation levels (η = 50, 100 and 200), σ_{opt} increases monotonically, but not linearly, with threshold θ.

Figure 3

Effective nonlinearity of the static LN model (with $\theta = 3$ and $\eta = 50$). (a) Effective nonlinearity is expressed as the relationship between the response of the linear kernel *x'(t)*, and the system output *y(t)*. The recovered nonlinearities change with σ. (b) However, when *x'(t)* is divided by the effective gain α, the effective nonlinearity becomes invariant, indicating that the static nonlinearity is not changed, and that the only change in the system is in the adjustment in the effective gain of the linear kernel brought about by the nonlinearity.

Figure 4

Contrast response function for the static LN model with $\theta = 5$ and $\eta = 50$. (a) An example of an input contrast signal with sinewave modulation (temporal sine frequency is 10 Hz). The input

contrast signal *c(t)* (magnitude of the the sinewave) changes every 4 seconds. The standard deviation of the contrast levels for this sequence is $\sigma_c = 1$. (b) Contrast response functions obtained from input contrast signals of mean contrast $\sigma_c = 1$, 5, 10 and 20 respectively. They are apparently independent of the mean contrasts.

Figure 5

(a) Mutual information I_m as a function of input σ for four different sets of thresholds and saturation levels. (b) $I_m(\sigma, \theta)$ as a function of $\alpha(\sigma, \theta)$ for various values of η. Each point corresponds to a value of $\alpha(\sigma, \theta)$ and a value of $I_m(\sigma, \theta)$.

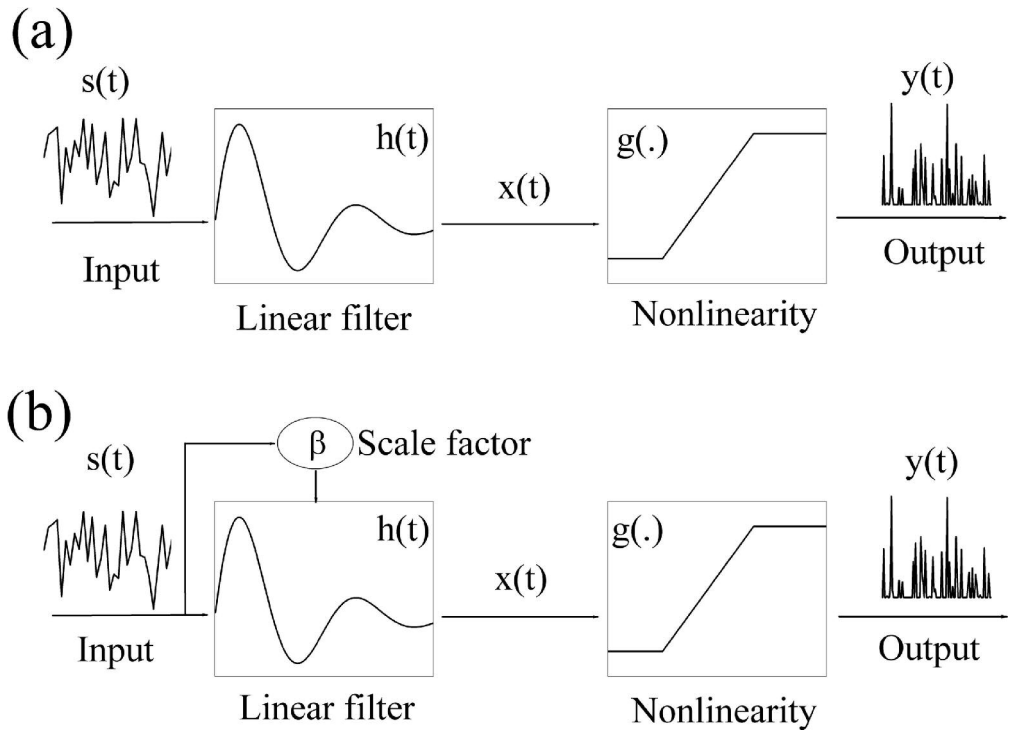
Figure 6

(a) Mutual information I_m varies with input standard deviation σ in the static LN model (threshold $\theta = 0$ and saturation $\eta = 50$) but is kept at maximum rate by choosing the appropriate β_{opt} for each σ in the adaptive LN model. (b) I_m is maintained at the maximum level for various θ and η. (c) The amplitude of the recovered kernel γ follows an inverse power-law relationship with input σ. (d) Experimental data by Truchard et al. (2000) shows that monocular gain decreases with stimulus contrast (from 2.5% to 50%) for two recorded cells. They are close to the inverse power-law relationship (shown for comparison as the dash line with slope of -1).

Figure 7

Contrast response functions or input and output curves for the adaptive LN model ($\theta = 5$ and $\eta = 50$). (a) The input and output curves, i.e., *c(t) ~ y(t),* recovered from input contrast signals *c(t)* and output *y(t)* of the whole system. Four classes of contrast signals are used, which noted by their

standard deviations σ_c = 1, 3, 5, and 10 respectively. Here, different standard deviations represent different mean contrast levels. The stimuli are same as described in Figure 4. The contrast response function is no longer invariant to σ_c, but becomes flatter (divisive effect) with increase in σ_c. (b) The adaptation effect is manifested as the shifting of the contrast response function as a function of the mean contrast along the log contrast axis. (c) This adaptive shift is a divisive effect, as the contrast response functions collapse together when the contrast axis is scaled (divided) by the mean contrast.



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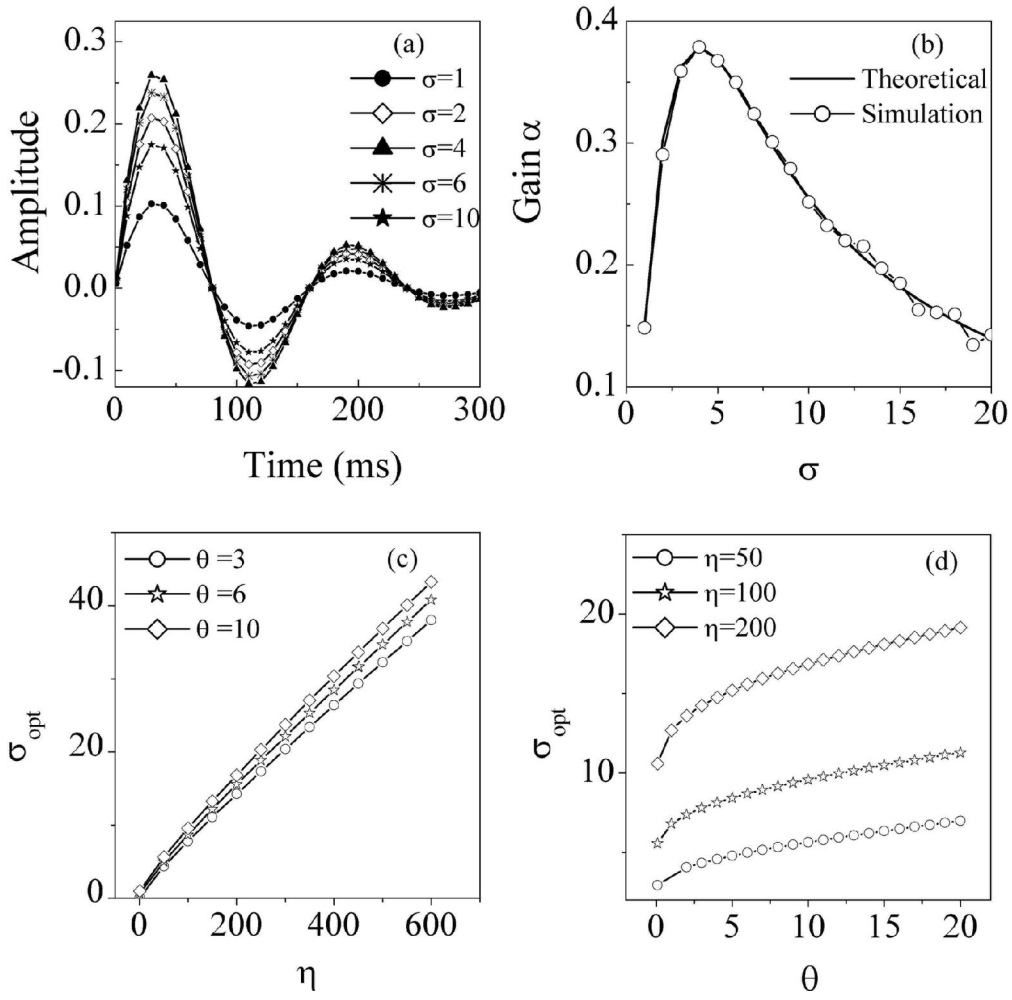

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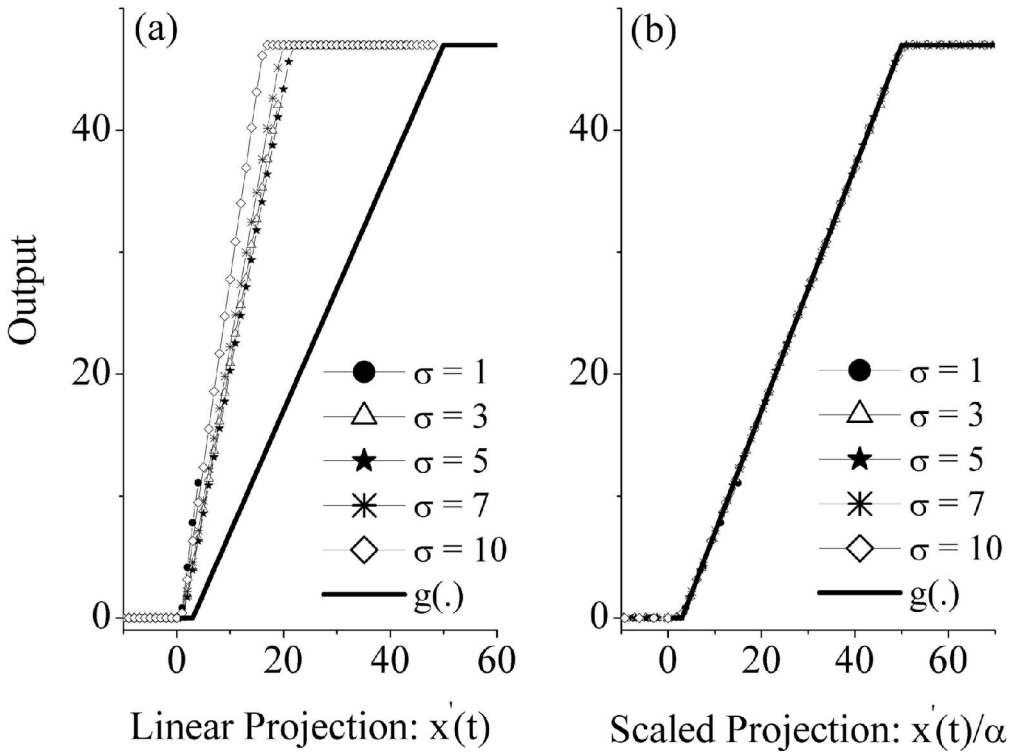

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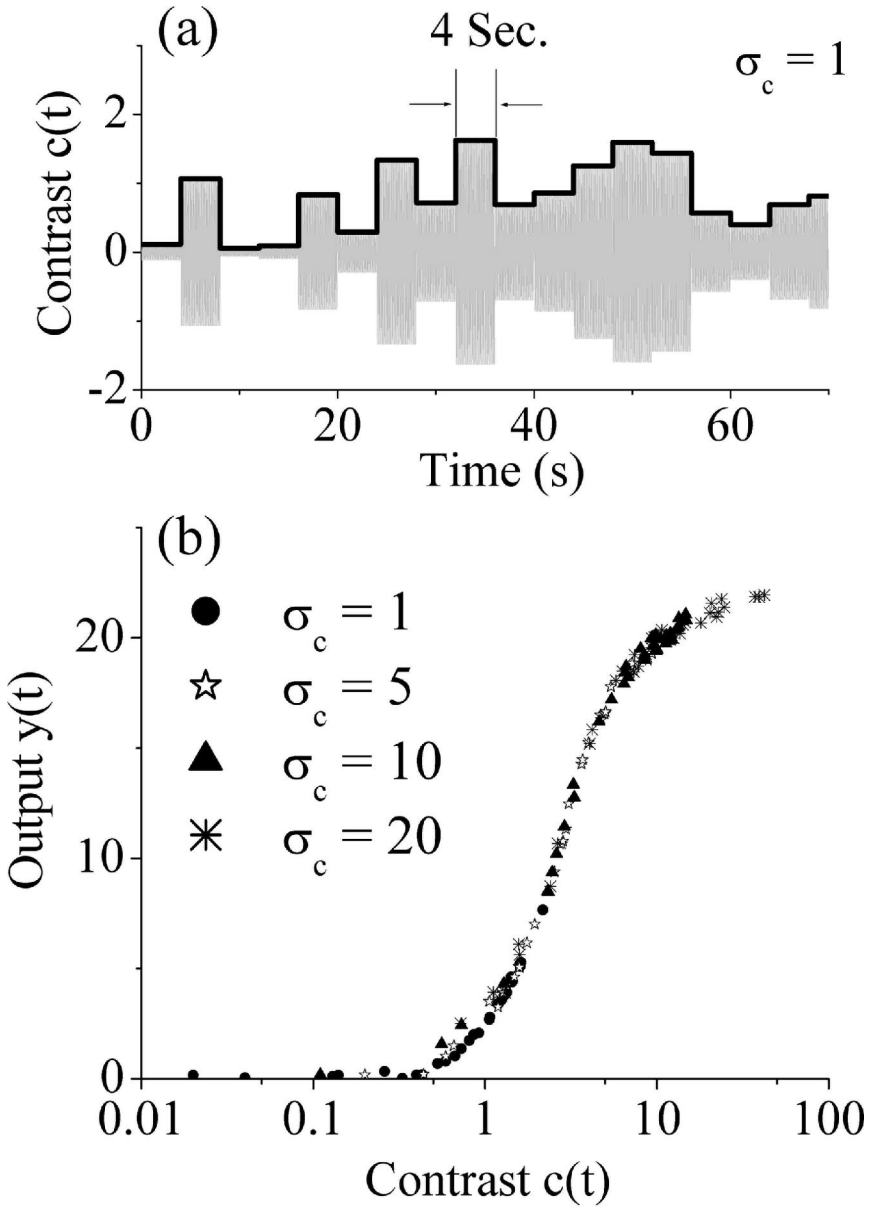

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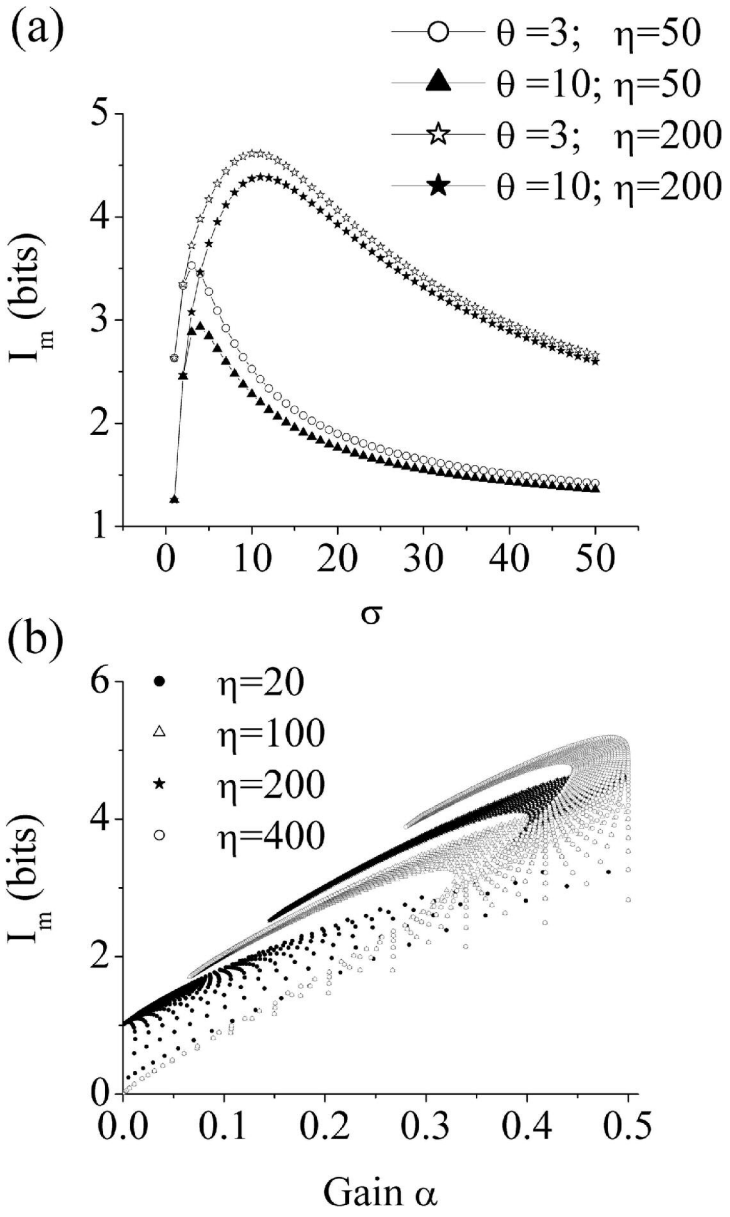

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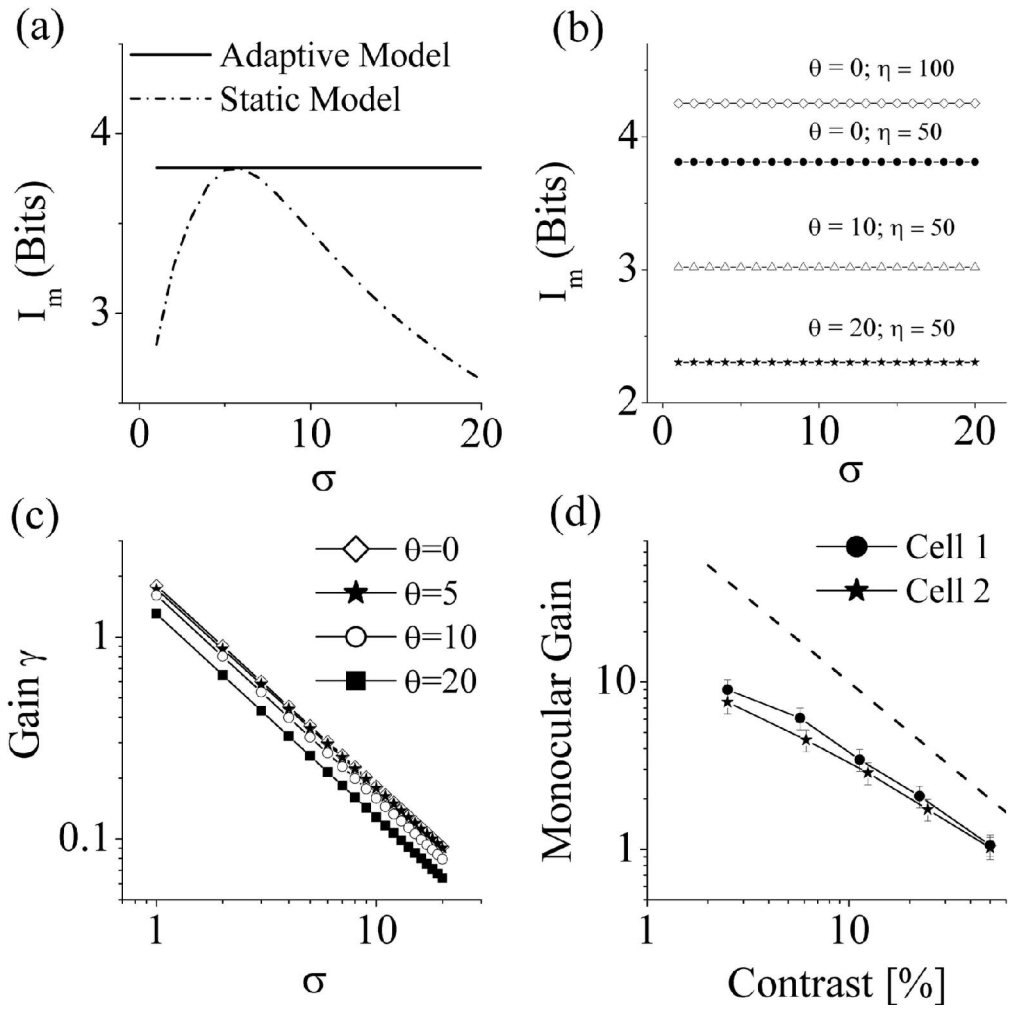

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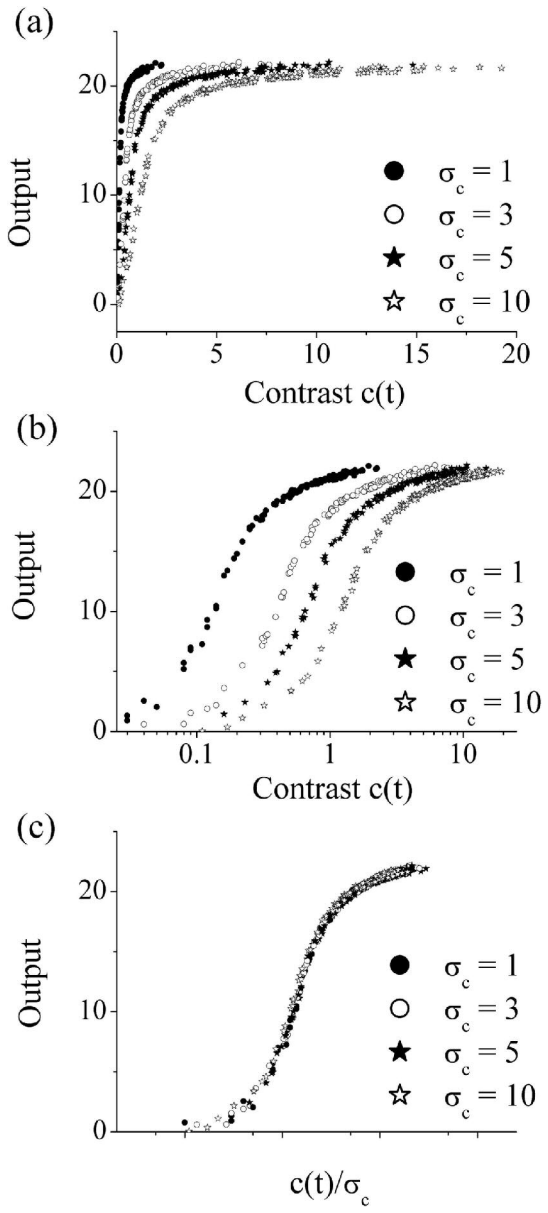

Figure 7
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