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TRANSFER OF CONTRAST SENSITIVITY IN LINEAR VISUAL NETWORKS

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ABSTRACT

Contrast sensitivity is a useful measure of the ability of an observer to distinguish contrast signals from noise. While usually applied to human observers, contrast sensitivity can also be defined operationally for individual visual neurons. In a model linear neuron consisting of a filter and noise source, this operational measure is a function of filter gain, noise power spectrum, signal duration, and a performance criterion. This definition allows one to relate the sensitivities of linear neurons at different levels in the visual pathway. Mathematical formulae describing these relationships are derived, and the general model is applied to the specific problem of relating the sensitivities of parvocellular LGN neurons and cortical simple cells in the primate.

key words: contrast sensitivity, receptive fields, noise, neural networks, visual cortex, lateral geniculate nucleus

INTRODUCTION

Contrast sensitivity is the inverse of the luminance contrast required by an observer to detect a particular target. It is a useful measure of the performance of visual observers. In the form of a contrast sensitivity function, in which the targets are spatial or temporal sinusoids, it has been used to summarize the overall performance of the observer. Contrast sensitivity to these and other patterns have also been used to infer the structure of the visual machinery.

Visual neurons also may be characterized in terms of their contrast sensitivity. In a previous paper (Watson, 1990), I noted that existing methods of measuring contrast sensitivity of linear visual neurons could be described in a simple mathematical context, and this context led to a canonical expression for neural contrast sensitivity that involves the contrast gain of the neuron, the noise in the neuron, and the measurement duration. In this paper I show how this mathematical context may be extended to describe the relationship between sensitivities of neurons at various levels in the visual pathway. This in turn allows inferences regarding the role of various neurons in the contrast sensitivity of the human observer.

The plan of this paper is as follows. In Part 1 I develop a general mathematical framework in which to express the various components of the problem: the receptive field, the power spectrum of the neural noise, and the network of connections between neurons at various levels in the visual pathway. This framework leads to a general result relating the sensitivity of neurons at two adjacent levels. In Part 2, this result will be applied to the specific problem of relating the sensitivity of parvocellular neurons in the lateral geniculate nucleus (LGN) to the simple cortical cells of primary visual cortex (V1), and thence to the sensitivity of human observers. A list of notation is provided at the end of the text for reference.

Part 1

Linear visual networks

A neuron is linear, with respect to some response measure, if that measure obeys the principles of superposition and homogeneity. Superposition means that if two inputs are added, the response will be the sum of the individual responses. Homogeneity means that intensifying the input by some amount intensifies the response by the same amount. The response measure considered here is the momentary impulse rate, and by that measure many neurons in both retina and cortex are approximately linear, at least for inputs of moderate intensity. Even these neurons, however, are nonlinear when large changes in adapting luminance or contrast are considered. A linear analysis is nevertheless quite powerful in providing an understanding of the response of the neuron in a stable state of adaptation.

Many visual neurons, particularly in the earliest levels of the visual pathway, can be considered to lie in a serial cascade of layers, as shown in Fig. 1. The signal arrives at the left and moves to the right through the various boxes. Each box represents a linear filter, characterized by a *level transfer function* (LTF) L, which defines the spatiotemporal filtering imposed by that level and which is determined by the connections and transduction properties of that level. Following each filter is a summing point at which noise is added. Noise may also be added at the input (M_0).

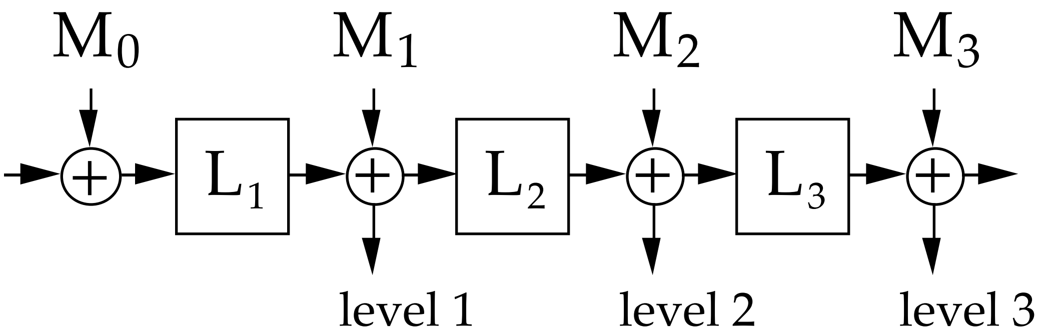

Figure 1. The early visual pathway depicted as a cascade of linear filters with additive noise at each level.

As a concrete example, we might consider the first level to be the photoreceptors, in which case M_0 represents noise in the image due to quantum fluctuations and possibly other sources, and M_1 represents noise generated within the photoreceptor. Level two might then represent the output of a retinal bipolar cell, with L_2 describing the linear combination of receptors, and M_2 representing the noise generated within the bipolar cell.

Since noise may be generated at various points within a neuron, the noise we associate with each layer is the sum of all these noises, referred to the output. In a linear system, the noise can be referred to either input or output. We choose the latter since it more clearly associates the noise with the corresponding neuron or level.

At each level the signal has two spatial dimensions, represented by a vector **x**, and a time dimension, *t*. Recording from a cell amounts to sampling the signal at one level at one spatial location. We shall elaborate on this point later on. The transfer functions $L_k(\mathbf{u},w)$ are written as functions of two spatial frequency dimensions, expressed as a vector **u**, and a temporal frequency dimension, *w*.

This formulation supposes that all neurons at one level are alike, except for their spatial location. It is therefore only appropriate for local regions of the visual field within which the spatial scale is roughly constant.

Receptive fields and transfer functions

The receptive field of a neuron, written $f(\mathbf{x},t)$, describes the contribution of contrast at location **x** and time *t* to the response at time 0. It is conventional to measure spatial coordinates relative to the center of the receptive field, so the neuron is located at [0,0]. In the context of linear systems theory, a more convenient representation is the impulse response, $h(\mathbf{x},t)$, which describes the contribution of contrast at time 0 and location [0,0] to the response of a neuron at time *t* and location **x**, and which is the reflection of the receptive field, $h(\mathbf{x},t) = f(-\mathbf{x},-t)$. The impulse response (or receptive field) is the result of all the filtering operations that have occurred at prior levels. The *spectral receptive field F* of the neuron is given by the Fourier transform of *f*. The *transfer function H* is the Fourier transform of *h*, and consequently $H = F^*$ (where the asterisk indicates a complex conjugate).The transfer function H_k of a neuron at level *k* is equal to the product of all the preceding level transfer functions,

$$H_k(\mathbf{u}, w) = \prod_{j=1}^{k} L_j(\mathbf{u}, w) \tag{1}$$

Contrast gain

In many physiological experiments, a neuron is characterized in terms of the magnitude of its response to each spatiotemporal frequency at unit contrast. Typically, this quantity is actually measured by noting the slope of the contrast response function (Kaplan & Shapley, 1986), or the inverse of the contrast required to yield a particular response, divided by that response (Enroth-Cugell, Robson, Schweitzer-Tong & Watson, 1983). This measure has been called both "responsivity" (Enroth-Cugell, et al., 1983) and "contrast gain" (Kaplan & Shapley, 1986), and we adopt the latter term here. In terms of the expressions introduced so far, contrast gain is given by the magnitude of the transfer function (or spectral receptive field),

$$G_k(\mathbf{u}, w) = |H_k(\mathbf{u}, w)| = |F_k(\mathbf{u}, w)| \ . \tag{2}$$

Contrast gain has units of imp/sec (we omit the dimensionless unit of contrast^{-1}). A possible source of confusion is that some authors have used units of "imp/sec / % contrast," (Kaplan & Shapley, 1986; Purpura, Kaplan & Shapley, 1988; Purpura, Tranchina, Kaplan & Shapley, 1990). This is equivalent to our measure divided by 100.

Noise

The noise added at each level is modeled as a stationary random process with dimensions of space **x** and time *t*. Each noise may be characterized by an autocorrelation function, $m_k(\mathbf{x}, t)$, or by its Fourier transform, the *power spectral density* $M_k(\mathbf{u}, w)$, which is a function of spatial and temporal frequency. The autocorrelation is a measure of the degree of correlation between samples of the noise process separated by a distance **x** and time *t*, while the power spectral density is a measure of the amount of noise at each spatiotemporal frequency. The power spectral density $M_k(\mathbf{u}, w)$ has units of $\mathrm{imp}^2\ \mathrm{sec}^{-2}\ \mathrm{Hz}^{-1}\ \mathrm{cycle}^{-2}\ \mathrm{deg}^2$.

The integral of the power spectrum, or equivalently the value of the autocorrelation at the origin, is the "average power" or variance of the noise process, which can be written σ_k^2. ($\mathrm{imp}^2\ \mathrm{sec}^{-2}$).

The total noise at a given level *k*, written $N_k(\mathbf{u}, w)$, is the result of all the noises introduced at prior levels, each shaped by the filters that it must pass through. Specifically, a noise $M(\mathbf{u}, w)$ passed through a filter $L(\mathbf{u}, w)$ becomes a noise $|L(\mathbf{u}, w)|^2 M(\mathbf{u}, w)$ (Papoulis, 1965). If the component noises at each level are independent and additive, then we can simply add their power spectra. Thus the total noise at level *k* may be written

$$N_k(\mathbf{u}, w) = M_k(\mathbf{u}, w) + |L_k(\mathbf{u}, w)|^2 N_{k-1}(\mathbf{u}, w) \tag{3}$$

The first term is the noise added at level k, the second is the total noise at the previous level, shaped by the squared magnitude of the level transfer function.

Together, eqn.s (1) and (3) allow us to collapse the complete network into an equivalent single stage, as shown in Fig. 2, with a single filter H_k and output noise N_k

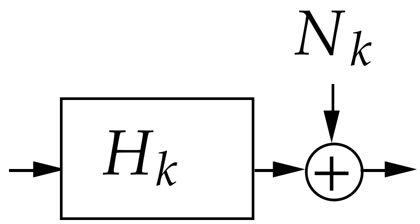

Figure 2. Equivalent single-stage representation of a linear visual network.

Sampling in space

The network described above has an output that is a function of both space and time, yet when we record a response from a neuron, it is solely a function of time. Mathematically, the response of a single neuron corresponds to a sample from the space-time output at a particular location, which we arbitrarily set to $\mathbf{x} = [0,0]$. The noise in the one-dimensional measurement is thus a stochastic process whose autocorrelation we write as $n_k(t)$, and whose power spectral density we write as $N_k(w)$.[1] The latter can be obtained from $N_k(\mathbf{u},w)$ by integrating over the two-dimensional spatial frequency variable $\mathbf{u}$,

$$N_k(w) = \int_{-\infty}^{\infty} N_k(\mathbf{u}, w)\, d\mathbf{u} \quad . \tag{4}$$

The resulting power spectral density has units of $\mathrm{imp}^2\ \mathrm{sec}^{-2}\ \mathrm{Hz}^{-1}$.

Contrast sensitivity

Contrast sensitivity for a neuron can be defined as the inverse of the contrast required to produce a neural response that is discriminable from noise with some specified reliability, as a function of the spatiotemporal frequency employed. A number of studies have examined contrast sensitivity of single

[1] To avoid a profusion of symbols, I use the same symbol N_k to identify three different functions: the three-dimensional power spectrum $N_k(\mathbf{u}, w)$, the purely spatial power spectrum $N_k(\mathbf{u})$, and the purely temporal power spectrum $N_k(w)$. The identity of the function is unambiguously indicated by its argument. The same convention is applied to functions M_k, L_k, and H_k, and to their lower case corresponding inverse Fourier transforms.

neurons in the LGN and cortex (Derrington & Lennie, 1982; Derrington & Lennie, 1984; Hawken & Parker, 1984; Troy, 1983a).[2] For a linear neuron, contrast sensitivity is given by

$$C_k(\mathbf{u}, w) = \sqrt{\frac{T}{2\tau}}\ \frac{G_k(\mathbf{u}, w)}{\sqrt{N_k(w)}} \tag{5}$$

where T is the measurement duration and τ is a performance parameter specifying the reliability of detection (Watson, 1990). For example, 75% correct in a two-alternative forced-choice task corresponds to $\tau = 2.78$. It is evident that contrast sensitivity is a ratio of contrast gain and the square root of the power spectrum, in other words, a dimensionless signal-to-noise ratio.

Contrast sensitivity transfer

The preceding provides a framework in which to relate the sensitivity of two adjacent levels in the visual pathway. We may think of this as the transfer of contrast sensitivity from one level to the next, and it will clearly depend on the transfer of both gain and noise, and upon the noise added at the higher level. For example, assume that we know the noise and contrast sensitivity at level *k-1*, and we know the level transfer function, and wish to determine the contrast sensitivity at level *k*. Taking the ratio of contrast sensitivities at levels *k* and *k-1*, we obtain

$$C_k(\mathbf{u}, w) = C_{k-1}(\mathbf{u}, w)\ \frac{G_k(\mathbf{u}, w)}{G_{k-1}(\mathbf{u}, w)} \sqrt{\frac{N_{k-1}(w)}{N_k(w)}} \tag{6}$$

The ratio of contrast gains is equal to the magnitude of the level transfer function L_k, so

$$C_k(\mathbf{u}, w) = C_{k-1}(\mathbf{u}, w)\ \left| L_k(\mathbf{u}, w) \right| \sqrt{\frac{N_{k-1}(w)}{N_k(w)}} \tag{7}$$

The next step is to expand the expression for the output noise at level *k*, which we do by combining eqn.s (3) and (4),

$$N_k(w) = \int_{-\infty}^{\infty} M_k(\mathbf{u}, w) + \left| L_k(\mathbf{u}, w) \right|^2 N_{k-1}(\mathbf{u}, w) \quad d\mathbf{u} \tag{8}$$

From eqn. (4),

$$N_k(w) = M_k(w) + \int_{-\infty}^{\infty} \left| L_k(\mathbf{u}, w) \right|^2 N_{k-1}(\mathbf{u}, w)\ d\mathbf{u} \tag{9}$$

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[2]Some studies have defined contrast sensitivity as the inverse of the contrast required to produce some arbitrary criterion response, eg 10 imp sec^{-1} (Enroth-Cugell & Robson, 1966; Linsenmeier, Frishman, Jakiela & Enroth-Cugell, 1982).

Combining eqn.s (7) and (9), we arrive at a final expression for the relation between contrast sensitivity at two levels

$$C_k(\mathbf{u}, w) = C_{k-1}(\mathbf{u}, w) \; |L_k(\mathbf{u}, w)| \sqrt{\frac{N_{k-1}(w)}{M_k(w) + \int_{-\infty}^{\infty} |L_k(\mathbf{u}, w)|^2 N_{k-1}(\mathbf{u}, w) \, d\mathbf{u}}} \tag{10}$$

Separable level transfer function.

Many of the following arguments are simplified if we assume that the LTF is separable in space and time, which also implies separability in spatial and temporal frequency. This assumption holds approximately (with some marked departures) for many visual neurons (Derrington & Lennie, 1982; Enroth-Cugell, et al., 1983; Frishman, Freeman, Troy, Schweitzer-Tong & Enroth-Cugell, 1987; Hamilton, Albrecht & Geisler, 1989; Tolhurst & Movshon, 1975; Troy, 1983b; Troy & Enroth-Cugell, 1989). It is not strictly true for direction selective simple cells, which are more nearly the sum of two separable functions (Hamilton, et al., 1989; Watson & Ahumada, 1983), though in this case similar assumptions would lead to almost the same result.

Separable noise power spectrum

It is also convenient to assume separability in space and time of the noise power spectral density at level *k-1*. This condition is unlikely to be precisely true. Even if all added component noises were uncorrelated (and hence all correlations in the output noise are due to filtering), and if all filters were separable, the resulting power spectrum would be a sum of separable functions, which is not necessarily separable. However, it seems likely that this assumption is not far from true (Mastronarde, 1983). In that event we write

$$N_{k-1}(\mathbf{u},w) = N_{k-1}(w) \; N_{k-1}(\mathbf{u}) \tag{11}$$

Recall that $N_{k-1}(w)$ is the integral over $\mathbf{u}$ of $N_{k-1}(\mathbf{u},w)$ (eqn. (4)), so that

$$\int_{-\infty}^{\infty} N_{k-1}(\mathbf{u}) \, d\mathbf{u} = 1 \tag{12}$$

Recall that the variance of the noise process is the integral of the power spectrum. For a separable process, the integral is the product of separate integrals, which may be thought of as the separate spatial and temporal variances. But since the two variances are reciprocally related, only their product has meaning. Hence eqn. (12) amounts to arbitrarily assigning unit variance to the spatial dimension, so that the total variance of the process is equal to the temporal variance. Eqn. (12) also implies that $n_{k-1}([0,0]) = 1$. Since $n_{k-1}(\mathbf{x})$ is normalized, it may be directly interpreted as the correlation between cells at the same level separated by vector $\mathbf{x}$.

Spatial Sampling of the Level Transfer Function

In the preceding analysis, we have treated the LTF as a continuous function. While this is mathematically convenient, it is at odds with our conventional picture of the discrete synaptic connections from one cell to the next. This apparent conflict is resolved in the following way. Each connection is made with a particular preceding neuron whose receptive field has a particular location. This situation may be represented by sampling the continuous level impulse response (the inverse Fourier transform of the LTF) at these locations. These samples represent the discrete weights associated with each neural connection.

This sampling in space will replicate the LTF in frequency, but this replicated LTF is multiplied by functions such as the contrast sensitivity and noise power spectral density of the previous level, both of which are likely to be low-pass functions. Provided that the replicas are outside the passband of these functions, sampling will have no effect on the shape of the predicted contrast sensitivity function, but will introduce a scalar factor D equal to the sample density in samples deg^{-2}. In that case sampling can be accounted for in the above eqns by replacing $L_k(\mathbf{u})$ everywhere with $D\ L_k^{'}(\mathbf{u})$, where the prime indicates the continuous version of the function. In particular, we write the complete separable LTF as,

$$L_k(\mathbf{u}, w) \;=\; \gamma\, D\ L_k^{'}(\mathbf{u})\ L_k(w) \tag{13}$$

Without loss of generality, we normalize the temporal and (continuous) spatial transfer functions, so that $\gamma\ D$ (a gain constant times the spatial sample density) describes the peak gain of the LTF.

The result of the preceding simplifications and assumptions is a new expression for contrast sensitivity,

$$C_k(\mathbf{u}, w) \;=\; C_{k-1}(\mathbf{u}, w)\left| L_k^{'}(\mathbf{u})\right| \left[\frac{M_k(w)}{\gamma^2 D^2 \left|L_k(w)\right|^2 N_{k-1}(w)} + \sigma^2_{s,k-1}\right]^{-1/2} \tag{14}$$

where

$$\sigma^2_{s,k-1} \;=\; \int_{-\infty}^{\infty} \left|L_k^{'}(\mathbf{u})\right|^2 N_{k-1}(\mathbf{u})\, \mathrm{d}\mathbf{u} \tag{15}$$

The integral of a power spectrum is the variance of a random process. We have assumed a separable power spectrum, so the variance can be regarded as the product of separate spatial and temporal variances. In this sense, $\sigma^2_{s,k-1}$ is the portion of the spatial variance at the output of level k contributed by level k-1.

Part 2

The development thus far has been abstract. Here I introduce a specific problem, and specific forms for the various functions, to show how the general principles can be applied. This will also allow a graphic presentation, which will help convey the ideas.

The specific problem I analyze is the relation between the contrast sensitivity of primate parvocellular lateral geniculate nucleus (LGN) cells, and of the simple cells of primary visual cortex (V1). This has been a subject of considerable debate. Parvocellular LGN cells have rather low peak contrast sensitivity (generally less than 10) (Derrington & Lennie, 1984; Kaplan & Shapley, 1986), while many V1 cells have peak contrast sensitivity as high as 100 (Hawken & Parker, 1984; Hawken, Parker & Lund, 1988). Furthermore, human and primate contrast sensitivity may attain values above 200 (De Valois, Morgan & Snodderly, 1974). Meanwhile, a second class of LGN cell, the magnocellular neurons, have peak sensitivities that are much nearer to cortical and psychophysical sensitivity. This has lead various authors to argue that the magnocellular system must be the substrate for psychophysical sensitivity (Hawken, et al., 1988; Kaplan & Shapley, 1986). As we shall show, the error here lies in assuming that the sensitivity at one level must be less than or equal to the sensitivity at prior levels. In fact, it may be much greater.

In the following sections, I have made use of so-called *unit Gaussians*, which are defined in the Appendix. Unit Gaussians allow a compact notation, and are easily integrated, multiplied, convolved, and Fourier transformed.

The levels *k-1* and *k* are now being associated specifically with LGN and cortical levels, respectively, and subsequent subscripts (*lgn* & *cortex*) will reflect this assignment.

Contrast sensitivity of parvocellular LGN neuron

The two-dimensional difference-of-Gaussians (DOG) function provides a reasonable model of the spatial contrast sensitivity function of the parvocellular LGN neuron (Derrington & Lennie, 1984). We therefore adopt the following expression for a the spatial distribution of contrast sensitivity,

$$c_{lgn}(\mathbf{x}) \;=\; v\left[\,{}_2\lambda_s(\mathbf{x}) \;-\; r_v\;\, {}_2\lambda_{s\,r_s}(\mathbf{x})\,\right] \tag{16}$$

where ${}_2\lambda_s(\mathbf{u})$ is a scaled unit gaussian (with unit volume) as defined in Appendix 1. The parameters are s: the spatial scale of the center Gaussian, r_s: the ratio of surround spatial scale to center spatial scale, v: the volume of the center Gaussian, and r_v: the ratio of volumes of surround and center unit Gaussians. When $r_v = 1$, the center and surround are in balance, and the neuron gives no response to uniform illumination. When $r_v = 0$, there is no surround. The LGN spatial contrast sensitivity function is the Fourier transform of eqn (16),

$$C_{lgn}(\mathbf{u}) \;=\; v\left[{}_2\Lambda_s(\mathbf{u}) \;-\; r_v\;\, {}_2\Lambda_{s\,r_s}(\mathbf{u})\right] \tag{17}$$

Because each unit Gaussian has unit volume, its transform ${}_2\Lambda_s(\mathbf{u})$ has unit peak gain (at $\mathbf{u}$=[0,0]). Thus the parameter v may also be regarded as the peak gain (in the frequency domain) of the center mechanism, and r_v as the ratio of peak gains of the surround and center Gaussians.

Derrington and Lennie (1984) provide DOG parameters for a set of six primate parvocellular LGN neurons, estimated at a temporal frequency of 5.2 Hz. We have derived a set of mean parameters, by averaging the six values of the parameters v, r_v, and r_s. The center spatial scale s was estimated by extrapolating to the fovea (by eye) their Fig. 6, which plots center radius versus eccentricity. The resulting values are $v = 13.66$, $s = 0.025$ deg, $r_s = 4.98$, $r_v = 0.65$, and the corresponding "average" parvocellular contrast sensitivity is shown in Fig. 3. Since all of our subsequent calculations are based on these averages, it should be acknowledged that there is considerable variability in these parameters. In particular, for the six cells of Derrington and Lennie, v ranged from 9.51 to 17.63.

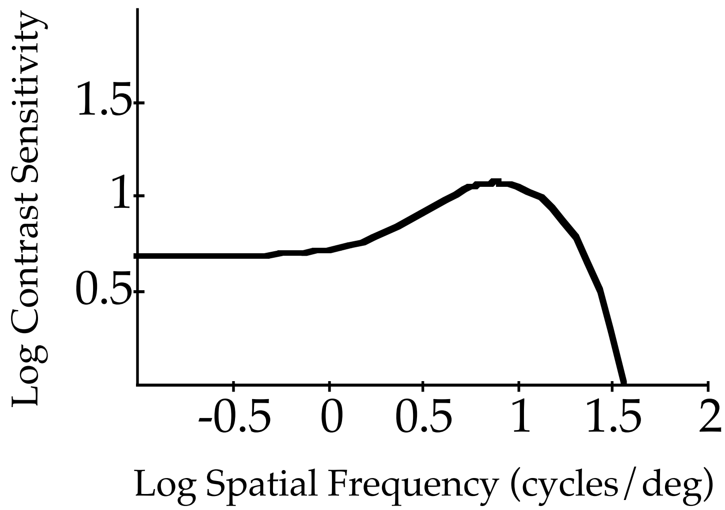

Figure 3. Average spatial contrast sensitivity function for primate foveal parvocellular LGN neurons, measured at 5.2 Hz. Curve is a difference of Gaussians (eqn. 17). Parameters derived from Derrington & Lennie (1984).

Geniculate temporal contrast sensitivity

An estimate of the parvocellular temporal contrast sensitivity function has also been taken from Derrington & Lennie (1984). Of their two estimates, we have taken the one with less low-frequency attenuation. This is given by

$$C_{lgn}(w) \quad = \quad (517 \exp[-0.128\ w] \ - \ 513 \exp[-0.135\ w]) \ / \ 11.5 \qquad (18)$$

The constant 11.5 serves to normalize the function at the frequency of 5.2 Hz at which the spatial contrast sensitivities were measured. The contrast sensitivity at any spatiotemporal frequency is then the product of $C_{lgn}(w)$ (eqn 17) and $C_{lgn}(\mathbf{u})$. The temporal contrast sensitivity function is pictured in Fig. 4.

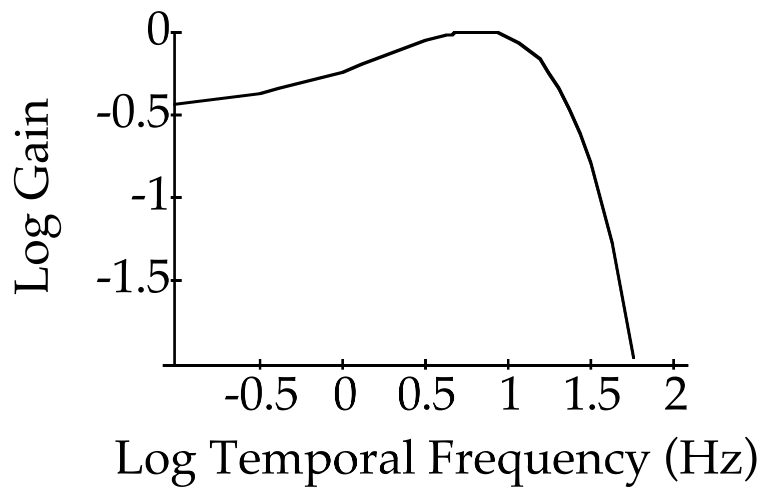

Figure 4. Temporal contrast sensitivity function for a primate parvocellular LGN neuron, estimated by Derrington & Lennie (1984). Curve is eqn. (18).

Geniculate temporal noise power spectrum

Temporal noise power spectra for primate LGN neurons are not available in the literature, but Troy has published data from a cat Y-type LGN cell (Troy, 1983b) from which a power spectrum can be estimated (Watson, 1990), as shown in Fig. 5.

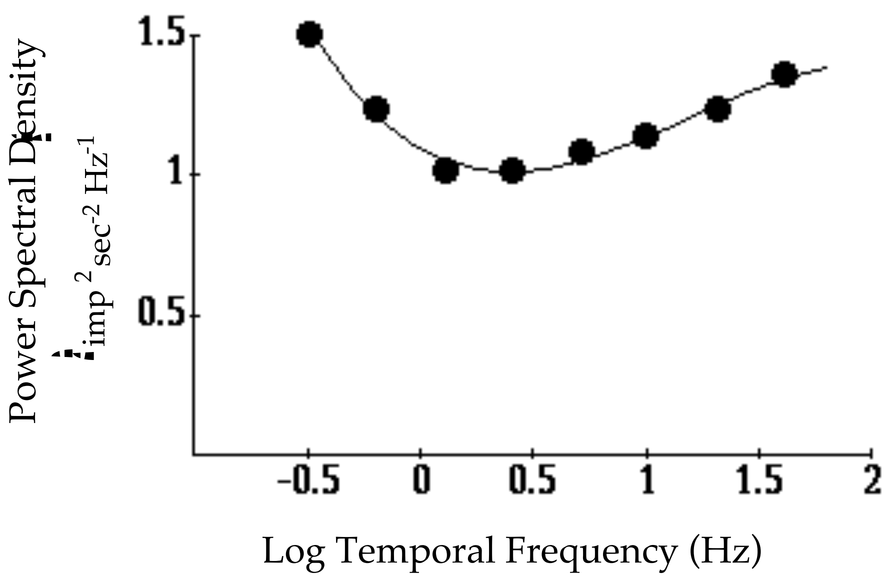

Figure 5 Noise power spectrum for a cat LGN neuron. Points estimated from data of Troy (1983b), by the method described in Watson (1990). Curve is eqn. (19).

The smooth curve is a third order polynomial (in log-log coordinates), fit by least squares, that we will use for interpolation,

$$N_{lgn}(w) \;=\; 10^{\left[1.1 - 0.46 \log(w) + 0.696 \log(w)^2 - 0.196 \log(w)^3\right]} \qquad (19)$$

Troy found almost identical power spectra for X-type cat LGN cells. Indirect evidence suggests that cat and primate have similar LGN noise power spectra, at least a medium temporal frequencies.[3] We therefore adopt eqn. (19) as the model primate LGN temporal noise power spectrum.

We may hope that in the near future, empirical power spectral densities for primate LGN cells will be available, as well as some theoretical understanding of its form. Robson and Troy (Robson & Troy, 1987) have noted that maintained discharges in cat retinal ganglion cells show interspike interval distributions that are Gamma distributed, with parameters of mean rate and Gamma order. This corresponds to a power spectrum which at low frequencies is equal to the mean rate divided by the order, rising to a peak at a frequency equal to the mean rate, subsiding at high frequencies to an asymptote equal to the mean rate. Gamma orders of around 8 and 4 were observed for X and Y cells, respectively.

Geniculate spatial noise power spectrum

Recall that the noise power spectrum is the Fourier transform of the autocorrelation. If all noise arose as white noise at the input (eg quantum fluctuations), then the LGN noise power spectrum would be the squared LGN contrast gain $G_{lgn}^2(\mathbf{u}, w)$, and the autocorrelation would be the inverse Fourier transform of this function.

For lack of better information, we assume that the spatial autocorrelation function of the LGN noise $n_{lgn}(\mathbf{x})$, is a two-dimensional unit Gaussian with spatial scale ρ , multiplied by ρ^2 to give it unit height. (see Appendix). There appear to be no published results on the spatial correlations amongst primate LGN cells. The only relevant data are estimates of spatial correlations between retinal ganglion cells in the cat obtained by Mastronarde (1983,1989). A brief summary of those results is that X-cells separated by one inter-cell spacing had correlations of up to 40%, while those separated by two spacings had correlations of around 6%. If we assume a foveal LGN spacing of about 0.01 deg, then these numbers lead to a value of about $\rho = 0.02$ deg, and we use this value in most subsequent calculations. Since $n_{lgn}(\mathbf{x})$ is radially symmetric, it could be expressed as a one-dimensional function of geniculate cell separation, but for consistency with notation elsewhere in this paper, we leave it as a

[3] Working in cat, Troy (1983b) reports a mean noise amplitude plus two standard deviations equal to 8.6 imp/sec at 5.2 Hz, while Derrington and Lennie (1984), working in primate LGN, report a corresponding figure of "about 10 imp/sec," suggesting that cat and primate are similar in the overall magnitude of their power spectra.

two-dimensional function. In that event, $N_{lgn}(\mathbf{u})$ (the Fourier transform of $n_{lgn}(\mathbf{x})$) is also a Gaussian, with spatial scale $1/\rho$, and height ρ^2,

$$N_{lgn}(\mathbf{u}) \; = \; \rho^2 \; {}_2\Lambda_\rho(\mathbf{u}) \tag{20}$$

Cortical temporal level transfer function.

The optimal and upper cut-off temporal frequencies of V1 cells are typically much lower than those of LGN cells. (Baker, 1990; Foster, Gaska, Nagler & Pollen, 1985; Movshon, Thompson & Tolhurst, 1978; Tolhurst & Movshon, 1975). This suggests a low-pass temporal LTF. We assume a simple exponential filter with time constant of 0.05 sec, to yield a cortical temporal gain that roughly matches the modal cut-off of 8 Hz shown by Foster *et al.*(1985). The magnitude of this transfer function is given by

$$L_{cortex}(w) \; = \; \left[\, 1 + (2\,\pi\, w\;\; 0.05)^2 \,\right]^{-1/2} \tag{21}$$

and is illustrated in Fig. 6.

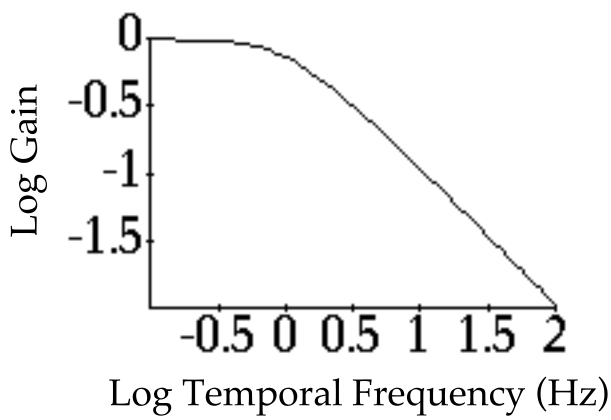

Figure 6. Magnitude of cortical temporal level transfer function with time constant 0.05 sec. Curve is eqn. (21).

In what follows we confine ourselves to predictions of spatial contrast sensitivity at the same temporal frequency at which the spatial LGN data were collected. The precise form of the cortical temporal LTF therefore has little effect on the predictions, but we include it for completeness and to emphasize that the formulae developed here predict the full spatio-temporal contrast sensitivity function.

Cortical spatial level transfer function

Unlike geniculate cells, which respond to a rather broad range of spatial frequencies at all orientations, most cortical neurons are selective for a modest band of spatial frequency and orientation. This selectivity is reasonably well modelled by a two-dimensional Gaussian in spatial frequency (Hawken & Parker, 1987; Jones & Palmer, 1987). In space this corresponds to a Gabor function (the product of a cosine and a two-dimensional Gaussian). Here I assume a Gabor function for the spatial level transfer function. Note that this will result in an overall cell transfer function that is the product of a DOG and a Gaussian. However, in the cases we consider, this will be very close to a simple Gabor. Note also that we invest no particular significance in the use of a Gabor function; it is merely a convenient and plausible device for limiting the frequency and orientation bandwidth of the cortical cell.

Therefore let the spatial level transfer function be

$$L'_{cortex}(\mathbf{u}) \;=\; \left[\,{}_2\Lambda_{\phi}(\mathbf{u}+\mathbf{u}_0\,) \;+\; {}_2\Lambda_{\phi}(\mathbf{u}\text{-}\mathbf{u}_0\,)\right] \tag{22}$$

where $\mathbf{u}_0$ is the Gabor spatial frequency and $u_0 = |\mathbf{u}_0|$ is its radial frequency. The spatial scale is $\phi = p / u_0$ deg, or p cycles. It can be interpreted as the half-width of the spatial Gaussian at an amplitude of 4.32% of maximum. Making the spatial scale a fixed number of cycles fixes the logarithmic bandwidth. Specifically, if the bandwidth in octaves is b then

$$p \;=\; \frac{2^b + 1}{2^b - 1}\sqrt{\frac{\ln 2}{\pi}} \tag{23}$$

For b= 1 octave, p has a value of 1.409 cycles, for b=1.4 octaves, p = 1.043 cycles. I will generally use a value of b=1.4 octaves, consistent with the data of De Valois, Albrecht & Thorell (1982) Finally, note that the function $L'_{cortex}(\mathbf{u})$ is normalized, as required by eqn. (13).

An approximation

The full expression for contrast sensitivity (eqn. (14)) contains a term $\sigma^2_{s,lgn}$ that in this context I will call the *geniculocortical spatial variance*, that is equal to the integral of the LGN spatial noise power spectrum, weighted by the square of the spatial cortical LTF (eqn. (15)). The power spectrum and the squared LTF are both Gaussian, so their product is a Gaussian, and the integral thus has an exact solution (see Appendix 1). We write the solution as a function of the spatial frequency u_0 of the cortical cell

$$\sigma^2_{s,lgn}(u_0) \;=\; \alpha\;\exp\!\left(-\,\pi\,\alpha\,p^2\right) \tag{24}$$

where

$$\alpha \;=\; \left[\,1/2 \;+\; \left(\frac{p}{\rho\,u_0}\right)^2\right]^{-1} \tag{25}$$

where p is the spatial scale of the cell in cycles and ρ is the LGN correlation distance (eqn. 20).

Since the power spectrum is essentially a constant ρ^2 at low frequencies, while the volume of the squared LTF is $\left(\frac{u_0}{p}\right)^2$,

$$\sigma_{s,lgn} \approx \frac{\rho\, u_0}{p} = \frac{\rho}{\phi} \tag{26}$$

For $\rho = 0.02$ deg and $b = 1.4$ octaves, the error in this approximation is less than 0.27 log units below 32 cycles/deg.

Case 1: No Cortical Noise

We have now specified all the components required to predict contrast sensitivity of cortical neurons, except for the cortical noise. We first consider the case of no cortical noise ($M_{cortex} = 0$). Examination of eqn. (14) shows that the resulting sensitivity does not depend on either sampling density D, the temporal LTF, or the gain factor γ,

$$C_{cortex}(\mathbf{u}, w) = C_{lgn}(\mathbf{u}, w) \left| L'_{cor}(\mathbf{u}) \right| / \sigma_{s,\, lgn} \tag{27}$$

Figure 7 shows predictions of contrast sensitivity for individual neurons in the case of no cortical noise.

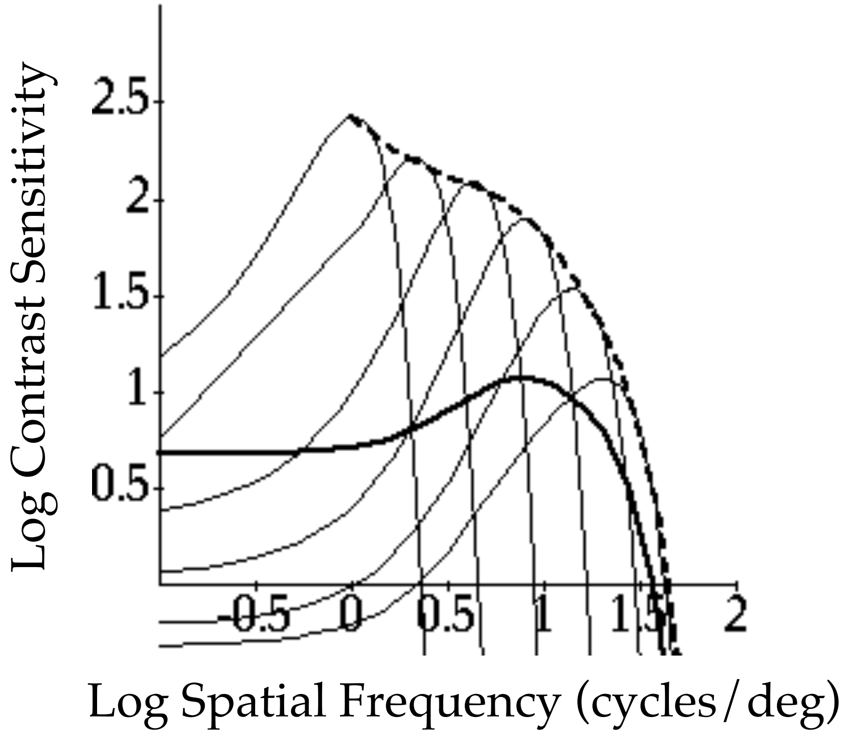

Figure 7. Predicted contrast sensitivity of cortical neurons when no noise is added at the cortical stage. The cortical neurons shown (thin solid lines, eqn. 27) have center frequencies of 1, 2, 4, 8, 16, and 32 cycles/degree, and each has a bandwidth of 1.4 octaves (p = 1.043 cycles). The LGN correlation distance is ρ = 0.02 deg The dashed line traces the peak sensitivity of the collection of neurons (eqn. 28). The heavy solid line is the sensitivity of the underlying LGN neurons (eqn. 17).

Peak function.

While eqn. (27) describes the contrast sensitivity of individual cortical neurons, it is edifying to consider a function that describes the peak sensitivity of each neuron as a function of its center spatial frequency u_0. This is the upper envelope of a family of sensitivity functions at different spatial frequencies, each described by eqn. (27). Recall that the spatial LTF is normalized, so this peak function is given by

$$\widehat{C}_{cortex}(\mathbf{u}, w) = C_{lgn}(\mathbf{u}, w) \; / \; \sigma_{s,lgn} \qquad (28)$$

This *peak function* is shown by the dashed line in Fig. 7.

Making use of our earlier approximation (eqn 26) for $\sigma_{s,lgn}$ we see that peak cortical contrast sensitivity is approximately equal to LGN contrast sensitivity, divided by spatial frequency, multiplied by the (constant) cell spatial scale in cycles, divided by the (constant) LGN correlation distance

$$\widehat{C}_{cortex}(\mathbf{u}, w) \approx C_{lgn}(\mathbf{u}, w) \, \frac{p}{\rho\,|\mathbf{u}|} \qquad (29)$$

Note that predicted cortical sensitivity rises as much as 1.4 log units above geniculate sensitivity. This illustrates one of the main points of this paper: *cells at one level may have a sensitivity that is much higher than that of cells at a prior level in a visual pathway.* In the present case, it says that V1 simple cells may have much higher contrast sensitivity than their parvocellular inputs. This result is not a mystery, it is due to the linear spatial pooling of signals over a wide area. This pooling will be discussed at greater length below.

It may be helpful to note that $\sigma_{s,lgn}$ is the square root of the portion of the LGN spatial variance that is "seen" by the spatial LTF of the cortical cell. As the spatial frequency of the cortical cell is reduced, the LTF narrows, and less of the variance is seen. Another way of thinking about it is that at lower frequencies, the spatial pooling area is larger, and this averaging reduces the spatial variance and thus enhances sensitivity.

Another observation is that predicted sensitivity grows without limit as the frequency of the cortical cell is lowered. However, the size of foveal simple cell receptive fields is presumably limited. For example, Hawken et al. (1988) found no cells within 1.5 deg of the fovea with peak spatial frequencies below 0.75 cycles/degree. Consequently, we are mainly concerned with the shape of the peak function above about 1 cycle/degree.

Effect of geniculate correlation distance ρ.

The previous figure was based on a value of ρ = 0.02 deg for the LGN correlation distance. Figure 8 shows the peak function for various other values of ρ.

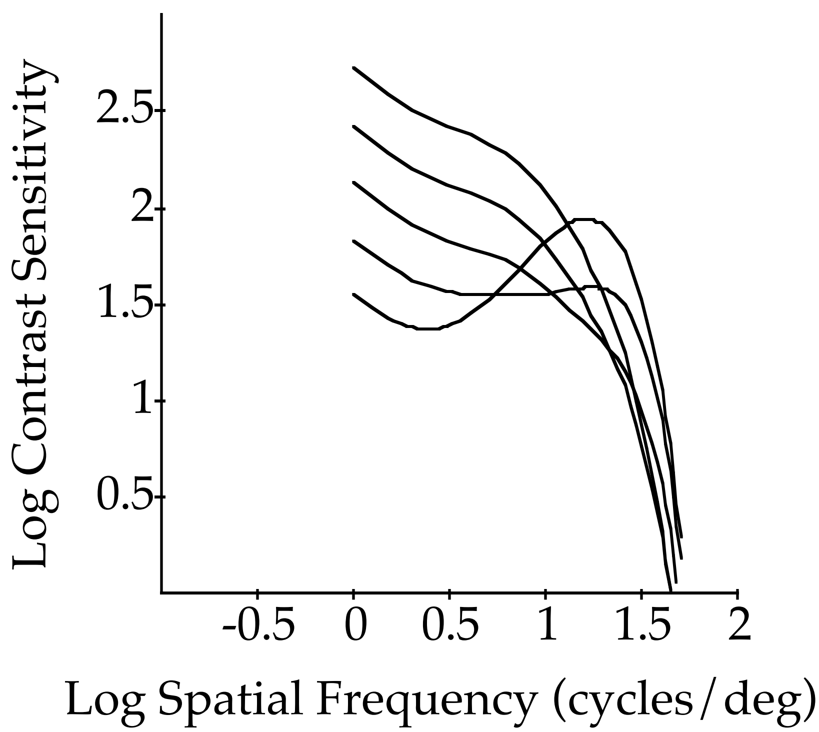

Figure 8. Peak contrast sensitivity with no cortical noise and geniculate correlation distances ρ of 0.01, 0.02, 0.04, 0.08, and 0.16 deg. Curves are from eqn. (28). Other details as in Fig. 7.

A rough characterization of the result is that increasing correlation reduces sensitivity at low spatial frequencies, but enhances sensitivity slightly at the highest spatial frequencies. The former effect is intuitive, since the greater the correlation, the fewer independent estimates of the signal there are to be pooled. The enhancement at high spatial frequencies is because no pooling is being done, and increased correlation corresponds to reduced noise in a local area.

These predictions are based on a Gaussian correlation (and power spectrum). Another shape for this power spectrum would of course alter the shape of the peak function.

Case 2: Cortical Noise

As we have seen, assuming an absence of cortical noise allows us to disregard several aspects of the model, such as the temporal LTF, the LGN sample density D, and the gain factor γ. The inclusion of cortical noise obliges us to consider these aspects, about which there are few data, and therefore adds degrees of freedom to the predictions. To avoid undue speculation, I will confine myself to a few general results and predictions.

Pelli (1990) has argued that, except at low spatio-temporal frequencies (below 4 Hz and 4 cycles/deg), psychophysical sensitivity is limited by quantum fluctuations. This would imply that cortical cells add little noise of their own over most of the frequency range. At low frequencies, he found an added neural noise component, which would tend to lower the peak function in this region. Although cortical noise may not be limiting over much of the spatiotemporal spectrum, we should nevertheless like to understand what effects it will have when it does intrude.

We have little information on the power spectrum of the noise added at the cortical level, $M_k(w)$. We therefore assume it has a constant density over the frequency range of interest, denoted by the constant M_k.

Cortical level gain.

The absence of cortical noise completely removes any effect of the level gain factor γ, because both signal and noise are amplified equally by the LTF. If cortical noise is present, some assumption must be made regarding γ. Note that this gain could be quite different for neurons of different spatial frequencies, following some function $\gamma(u_0)$, allowing an almost arbitrary shape for the resulting peak function (though it must always lie below the no-cortical noise curve, because additional noise can only reduce sensitivity). Empirically, some insight into this function might be offered by comparison of the LGN contrast gain at some visual field location and the contrast gain of cortical cells of various frequencies drawn from the same location. However such data appear not to be available.

In this section we consider the hypothesis that the gain is set adaptively at each spatial frequency to optimize the use of the available response range of each neuron. Neurons have a rather limited dynamic range, those in the LGN and cortex typically responding at less than 100 impulses/second (Sclar, Maunsell & Lennie, 1990). The maximum response produced by our linear model neuron is equal to the peak contrast gain times the maximum contrast. It seems essential that the gain of each cortical LTF be set in such a way that naturally occurring contrasts will generate responses within the dynamic range of the cell. The little available evidence (Field, 1987) suggests that the spatial contrast amplitude spectrum of natural imagery is proportional to $1/u$. This corresponds to equal energy within spectral regions of constant log bandwidth. Since our model cortical neurons are designed to have a constant log bandwidth, they will, if given equal peak gain, have equal expected energy in their outputs. This in turn means that each neuron, exposed to an ensemble of natural images, will produce a distribution of responses with equal standard deviation. The gain of the neuron should be set in such a way that the maximum response is proportional to this standard deviation. Therefore, to match the dynamic range of the neuron to the natural contrast distribution, the peak gains of neurons at different frequencies should be equated. We lack specific values for the absolute magnitude of the natural contrast power spectrum, so we will be content to adopt a constant cortical peak gain of γ_{cortex}. Then the gain factor of a neuron at spatial frequency u_0 will be

$$\gamma(u_0) = \frac{\gamma_{cortex}}{D \; \left| L_{cortex}(w_1) \right| G_{lgn}(u_o, w_1)} \tag{30}$$

This gain function yields a constant peak contrast gain for each cortical neuron (at some temporal frequency w_1) by compensating for the variations in gain introduced by the LGN neuron. It may be thought of as a "de-blurring" operation applied to the ensemble of cortical neurons. Note also that when inserted into eqn. (14), the density term D vanishes, so that for these predictions, as for the no-cortical-noise case, LGN spatial density plays no role.

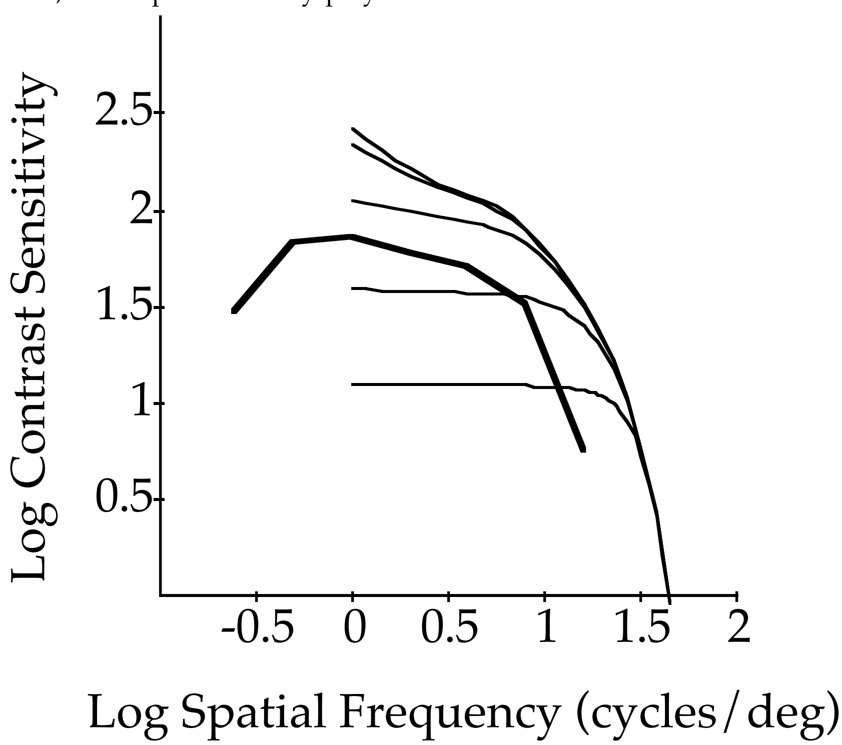

Figure 9. Predicted peak contrast sensitivity for cortical noise of 0, 0.1, 1, 10, 100 imp^2 sec^{-2} Hz^{-1}. Other parameters are ρ = 0.02 deg, τ=0.15 sec, b = 1.4 octaves., γ_{cor} = 100 imp sec^{-1}, w_1= 6.5 Hz. The heavy line shows psychophysical data from a human observer. Psychophysical details: 100 cd m^{-2}, 1 octave gabor, natural pupil, Gaussian time course with 1/e width of 0.5 sec (0 Hz). Physiological details: 200 cd m^{-2}, 1.4 octave Gabor, duration 1 sec, no eye movements, 5.2 Hz, 3 mm artificial pupil.

Figure 9 shows peak contrast sensitivities for five different amounts of cortical noise under the adaptive gain assumption. Increasing cortical noise has two effects: sensitivity is reduced at middle and low frequencies, and the curve becomes flatter in this frequency range. The flattening of the curve is explained as follows. Under the adaptive gain assumption, all neurons have the same peak gain regardless of their spatial frequency. Furthermore, we have assumed a flat cortical spatial noise spectrum. Thus as cortical noise comes to dominate the total noise, contrast sensitivity becomes independent of spatial frequency.

The adaptive gain hypothesis is but one possible conjecture regarding the relative gains of the neurons at different spatial frequencies. Other schemes may be entertained, but they cannot escape the constraint imposed by the limited dynamic range of the cortical neuron.

Relation to physiological contrast sensitivity

Contrast sensitivities of primate V1 cortical cells have been measured by Hawken and Parker (1984) and Hawken, Parker, & Lund (1988). In the earlier paper, the highest sensitivities were on the order of 100 (in lamina IVcα), while in laminae receiving input primarily from parvocellular neurons, highest sensitivities were around 40. In the later report, a large proportion of foveal cortical cells exhibited sensitivities of between 20 and 80 (laminae were not indicated). Despite the large range of peak sensitivities, and the sampling uncertainties inherent in this sort of experiment, it is clear that many V1 cells, even those presumably driven by parvocellular inputs, show sensitivities greater than that of typical parvocellular geniculate neurons.

Comparing these physiological results with the predictions, a moderate amount of cortical noise (1 imp^2 sec^{-2} Hz^{-1}) yields peak sensitivities of about 2 log units, comparable to the best sensitivities observed for V1 cells. Furthermore, human psychophysical sensitivities are quite close to V1 peak neural sensitivities when psychophysical targets are matched to the cells receptive field (Hawken & Parker, 1990), and as will be discussed below, psychophysical thresholds are roughly consistent with the predictions for moderate noise.

Relation to psychophysical contrast sensitivity

One reason we wish to understand the contrast sensitivity of visual neurons is the insight it may give us into the psychophysical sensitivity of human observers. Indeed, early measurements of neural contrast sensitivity, rather than contrast gain, were made to permit comparisons of neural and psychophysical sensitivity (Derrington & Lennie, 1982, 1984). We have adopted an operational definition of contrast sensitivity that allows us to make rather direct prediction of psychophysical sensitivity. Specifically, an observer relying on the output of a single cortical neuron, and making ideal use of the neurons response except for phase, would have the same contrast sensitivity as the neuron. Thus we may take peak functions like those in Fig.s 7-9 as predictions of psychophysical contrast sensitivity.

Figure 9 also shows one set of psychophysical contrast sensitivities collected from a human observer (Watson, 1987). Sensitivities were measured with one-octave Gabor functions with a mean luminance of 100 cd m^{-2} .These are similar to data collected by others (Banks, Geisler & Bennett, 1987; Banks, Sekuler & Anderson, 1991; Pointer & Hess, 1990). A detailed comparison of neural and psychophysical sensitivities is beyond the scope of this paper, but two general points may be made.

First, the peak sensitivities predicted at middle frequencies are on the same order as the psychophysical data. Thus the parvocellular pathway, despite its low contrast sensitivity at the LGN, is capable of providing the basis for human psychophysical contrast sensitivity.

The second point is that at high spatial frequencies the predicted sensitivity is much greater than the empirical sensitivity. The discrepancy appears larger than is likely to be accounted for by the various differences between the conditions of measurement for human and primate, although several of these are likely to account for some of the difference (see details in caption to Fig. 9). The predicted sensitivities at the highest frequencies are quite dependent upon the estimate of the LGN center size s, which we have assumed here to be 0.025 deg. However this must be enlarged by a factor of two to bring the curves into agreement.

Discussion

The first purpose of this paper was to derive some general principles and formulae to relate the contrast sensitivities of neurons at different levels in a visual pathway. In general the sensitivity at the higher level depends upon sensitivity at the lower level, upon the spatiotemporal noise power spectrum at the lower level, upon the level transfer function relating the gains at the two levels, and upon the noise added at the higher level. Some simple equations were derived to describe these relations in a formal and computable way.

A second goal was to illustrate these principles by applying them to the relation between contrast sensitivity of parvocellular LGN and cortex. The resulting predictions showed that while LGN cells individually have low contrast sensitivity, the resulting cortical sensitivities are as high as measured cortical and psychophysical sensitivities. This is consistent with other results suggesting that parvocellular neurons are the basis of contrast sensitivity, except at the lowest spatial and temporal frequencies (Merigan & Eskin, 1986).

One benefit of this analysis is that it indicates the quantities that govern sensitivity, and which might therefore prove valuable to measure. Among the unknowns that we encountered in applying this analysis to LGN and cortical cells were: the temporal noise power spectrum of the parvocellular LGN cell, the correlation amongst nearby LGN cells, and the noise power spectrum of the cortical cell.

Spatial pooling.

As we have noted, cortical cells may have sensitivities much greater than that of their LGN inputs, as a consequence of spatial pooling (Sclar et al. 1990). As a rule of thumb, the approximation in eqn. (29) shows that in the absence of cortical noise, plausible values of cortical cell bandwidth and essentially uncorrelated LGN cells ($p = 1$, b=1.47 octaves, ρ = 0.01 deg) lead to a relative sensitivity of cortex and LGN equal to 100 divided by the cell spatial frequency. Thus a cortical cell at 1 cycle/deg will be 100 times as sensitive as its LGN inputs.

It is interesting to consider the number of cells being pooled in this example. At 1 cycle/deg, p = 1 implies a circular pooling area of 1 deg radius, which when LGNs are spaced at 0.01 degree implies roughly $\pi\, 100^2$ = 31,415 LGN inputs! This number is undoubtedly an overestimate, since it does not take into account the decline in LGN cell density with eccentricity but it nonetheless gives a sense of the massive amount of spatial pooling that must be involved. Tanaka (1985) gives an estimate of 30 LGN cells driving one cortical cell, the number obtained by dividing the total cortical response by the contribution from one LGN cell. This assumes that all LGN inputs to the cortical cell have equal gain, which is unlikely, and for which Tanaka offers no evidence. It is also likely that his methods would select the LGN cells making the largest contribution to the cortical cell. Also, my estimate is only for the largest (lowest frequency) cortical cells, while the tuning of Tanaka's cells is unclear. Furthermore, Tanaka's recordings were made in cat, and their relation to primate cortex is not known.

Limitations of the analysis.

It is important to acknowledge some limitations of the analysis presented here. It is appropriate only for linear neurons, and cortical neurons are quite non-linear. However simple cells, which we model here, generally behave linearly up to their output, which undergoes a half-wave rectification and possible point non-linearity. Therefore the sensitivities we calculate must be interpreted as referring to quantities prior to these output nonlinearities. Some cortical cells also have no maintained discharge, in which case a single spike may be taken as the criterion. But then one has no direct means of estimating the internal noise of the cell.

Another complication is the possible non-linear adaptation of the cortical gain to the ambient contrast (Albrecht, Farrar & Hamilton, 1984; Heeger, 1991; Maddess, McCourt, Blakeslee & Cunningham, 1988; Ohzawa & Freeman, 1985). However, this process, while quite powerful in the cat, may be much less evident in primates . It would not, at any rate, have much effect for the near-threshold stimuli employed in these measurements.

A third limitation is the assumption of spatial homogeneity. This has two aspects: local disorder in spacing and size of receptive fields, and systematic increase in size and spacing with eccentricity. The former is unlikely to have large effects in foveal vision. As to the latter, human and monkey cone diameter and spacing increase by about 70-90% over an eccentricity of 1 degree (Packer, Hendrickson & Curcio, 1989; Samy & Hirsch, 1989). This inhomogeneity will have its greatest effect at the lower frequencies .Lower frequency cells require larger receptive fields (assuming constant log bandwidth), and thus must pool over a larger, more inhomogeneous region, in which the mean spacing is below that at the fovea. Relative to the homogeneous prediction, then, inhomogeneous predictions would be somewhat lower at the low frequency end. It should be noted however that the more fundamental relationships between gain, noise and contrast sensitivity discussed here are not dependent upon homogeneity, and inhomogeneous predictions could be derived from them.

A fourth limitation concerns the relation between neural and psychophysical contrast sensitivity. Under certain assumptions (one cell, phase-uncertain ideal observer) neural contrast sensitivity is a direct predictor of psychophysical sensitivity. Experiments can be designed that increase the reasonableness of these assumptions, for example, by matching the size of the stimulus and the target cell. However, various departures from these assumptions can be imagined, such as use of information from many cells, phase knowledge, or less than ideal detection. Nevertheless, the direct prediction is an important benchmark, from which these departures are relatively minor amendments.

Despite the cautions mentioned above, several rather strong conclusions emerge. The first is that the rather insensitive parvocellular neurons can feed very sensitive cortical cells, and can be the basis for very high psychophysical sensitivities.

A more general conclusion is that relationships between sensitivities at various levels in the visual pathway depend strongly upon the level transfer function, the noise at each level, and correlations among nearby cells. The formulae discussed here allow these factors to be combined to generate meaningful predictions.

A final observation is that the measurement of contrast gain, noise, and sensitivity of neurons at various levels may provide a powerful way of dissecting the functional anatomy of visual pathways, and of understanding of the relationship between neural and psychophysical contrast sensitivity.

Appendix: Unit Gaussians

The following is a scheme of notation for expressing Gaussians in space and frequency. The advantage of scaled unit Gaussians is that they convert manipulations of Gaussians, such as Fourier transformation, multiplication, convolution, and squaring, into simple algebraic manipulations of amplitudes and scales.

Unit Gaussian.

We define a *unit Gaussian* as

$$\lambda(x) = e^{-\pi x^2} \tag{A1}$$

This form has the virtue that it has unit area, and that it is its own Fourier transform:

$$\Lambda(u) = e^{-\pi u^2} \tag{A2}$$

Scaled Unit Gaussian.

We introduce a scale parameter, a, and a scaled unit Gaussian,

$$\lambda_a(x) = \frac{1}{|a|} \lambda(x/a) \tag{A3}$$

Note that the scaling is done in such a way as to preserve the area, rather than the peak value of the Gaussian. The scale parameter is a measure of the dispersion of the Gaussian, and is proportional to other measures such as standard deviation and width, as shown below. The Fourier transform of the scaled Gaussian is

$$\Lambda_a(u) = \Lambda(a\,u) \tag{A4}$$

Note that it is scaled in such a way as to preserve peak value, rather than area. This follows from the fact that the integral of a function is equal to the value of the transform at the origin, and we have fixed the area of the scaled Gaussian to be 1. Another virtue of this parameterization is that if we regard a as a measure of width, then the width of the Fourier transform is $1/a$.

Two Dimensions.

A two dimensional, radially symmetric scaled unit Gaussian can be written

$$_2\lambda_a(\mathbf{x}) = \lambda_a(\mathrm{x})\ \lambda_a(\mathrm{y}) \tag{A5}$$

and its Fourier transform,

$$_2\Lambda_a(\mathbf{u}) = \Lambda_a(\mathrm{u})\ \Lambda_a(\mathrm{v}) \tag{A6}$$

The following are some useful results:

Product.

$$\lambda_a(x)\ \lambda_b(x) \;=\; \frac{1}{\sqrt{a^2+b^2}}\ \lambda_{\frac{ab}{\sqrt{a^2+b^2}}}(x) \qquad \text{(A7)}$$

$$\Lambda_a(u)\ \Lambda_b(u) \;=\; \Lambda_{\sqrt{a^2+b^2}}(u) \qquad \text{(A8)}$$

Convolution.

$$\lambda_a(x)\ *\ \lambda_b(x) \;=\; \lambda_{\sqrt{a^2+b^2}}(x) \qquad \text{(A9)}$$

$$\Lambda_a(u)\ *\ \Lambda_b(u) \;=\; \frac{1}{\sqrt{a^2+b^2}}\ \Lambda_{\frac{ab}{\sqrt{a^2+b^2}}}(u) \qquad \text{(A10)}$$

Square.

$$\lambda_a^2(x) \;=\; \frac{1}{\sqrt{2}\ a}\ \lambda_{\frac{a}{\sqrt{2}}}(x) \qquad \text{(A11)}$$

$$\Lambda_a^2(u) \;=\; \Lambda_{\sqrt{2}a}(u) \qquad \text{(A12)}$$

Integral.

$$\int_{\infty}^{\infty} \lambda_a(x) \;=\; 1 \qquad \text{(A13)}$$

$$\int_{\infty}^{\infty} \Lambda_a(u) \;=\; \frac{1}{|a|} \qquad \text{(A14)}$$

Energy.

$$\int_{-\infty}^{\infty} \Lambda_a^2(u) \;\; du \;\; = \;\; \int_{-\infty}^{\infty} \lambda_a^2(x) \;\; dx \;\; = \;\; \frac{1}{\sqrt{2}\ \mathrm{a}} \tag{A15}$$

$$\int_{-\infty}^{\infty} {}_2\Lambda_a^2(\mathbf{u}) \;\; d\mathbf{u} \;\; = \;\; \int_{-\infty}^{\infty} {}_2\lambda_a^2(\mathbf{x}) \;\; d\mathbf{x} \;\; = \;\; \frac{1}{2\ \mathrm{a}^2} \tag{A16}$$

Width.

Let w be the half-width at half-amplitude of the Gaussian. Then

$$w \;=\; a\sqrt{\ln 2/\pi} \tag{A17}$$

Let w' be the half-width in the frequency domain. Then

$$w' \;=\; \frac{\ln 2}{\pi w} \tag{A18}$$

$$w' \;=\; \frac{\sqrt{\ln 2/\pi}}{a} \tag{A19}$$

NOTATION

This is a listing of the principal notation used in this paper, in approximate order of introduction. Where appropriate, units are indicated. Some general conventions adopted are: boldface symbols indicate vectors, upper case letter function names indicate Fourier transforms of corresponding lower-case functions.

k	level of a neuron in visual pathway
$\mathbf{x} = [x,y]$	spatial position [deg]
t	time [sec]
$\mathbf{u} = [u,v]$	spatial frequency [cycles/deg]
w	temporal frequency [Hz]
$f(\mathbf{x},t)$	cell receptive field [imp sec^{-1}]
$h(\mathbf{x},t)$	cell impulse response [imp sec^{-1}]
$F(\mathbf{u},w)$	cell spectral receptive field (complex) [imp sec^{-1},imp sec^{-1}]
$H(\mathbf{u},w)$	cell transfer function (complex) [imp sec^{-1},imp sec^{-1}]
$L(\mathbf{u},w)$	level transfer function (LTF)
$G(\mathbf{u},w)$	contrast gain [imp sec^{-1}]
$m(x,w)$	level noise autocorrelation
$M(u,w)$	level noise power spectral density [imp^2 sec^{-2} deg^2 Hz^{-1}]
$n(x,w)$	cell noise autocorrelation
$N(\mathbf{u},w)$	cell noise power spectral density [imp^2 sec^{-2} deg^2 Hz^{-1}]
$N(w)$	cell noise power temporal spectral density [imp^2 sec^{-2} Hz^{-1}]
$M(w)$	level noise power temporal spectral density [imp^2 sec^{-2} Hz^{-1}]
$C(\mathbf{u},w)$	contrast sensitivity
τ	performance parameter
γ	peak gain of LTF, divided by D
d	LGN sample distance [deg]
D	density of LGN cells [deg^{-2}]
s	spatial scale of LGN center Gaussian [deg]
r_s	ratio of spatial scales of LGN surround and center
v	volume of LGN center Gaussian [imp/sec]
r_v	ratio of volumes of LGN surround and center
$\lambda_a(x)$	unit scaled Gaussian with scale a
$\Lambda_a(u)$	Fourier transform of $\lambda_a(x)$
${}_2\lambda_a(\mathbf{x})$	two-dimensional unit scaled Gaussian with scale a
${}_2\Lambda_a(\mathbf{u})$	Fourier transform of ${}_2\lambda_a(\mathbf{x})$
ρ	LGN spatial correlation distance [deg]
u_0	cortical receptive field radial spatial frequency [cycles/deg]
$\mathbf{u}_0$	cortical receptive field spatial frequency [cycles/deg]
ϕ	cortical receptive field spatial scale [deg]
p	cortical receptive field spatial scale [cycles]
b	cortical cell spatial frequency bandwidth [octaves]

$\sigma^2_{s,k\text{-}1}$	spatial variance of noise at level *k* contributed by level *k-1*
$\widehat{C}\,(\mathbf{u},w)$	peak contrast sensitivity over an ensemble of cells of various **u**
γ_{cortex}	peak gain of cortical LTF, under adaptive gain assumption

Abbreviations:

LGN	lateral geniculate nucleus
LTF	level transfer function

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REFERENCES

Albrecht, D. G., Farrar, S., & Hamilton, D. B. (1984). Spatial contrast adaptation characteristics of neurons recorded in the cat's visual cortex. Journal of Physiology (London), 347, 713-739.

Baker, C. L., Jr. (1990). Spatial- and temporal-frequency selectivity as a basis for velocity preference in cat striate cortex neurons. Visual Neuroscience, 4, 101-113.

Banks, M. S., Geisler, W. S., & Bennett, P. J. (1987). The physical limits of grating visibility. Vision Research, 27(11), 1915-1924.

Banks, M. S., Sekuler, A. B., & Anderson, S. J. (1991). Peripheral spatial vision: Limits imposed by optics, photoreceptors, and receptor pooling. Journal of the Optical Society of America A, submitted.

De Valois, R. L., Albrecht, D. G., & Thorell, L. G. (1982). Spatial Frequency Selectivity of Cells in Macaque Visual Cortex. 22, 545-559.

De Valois, R. L., Morgan, H. C., & Snodderly, D. M. (1974). Psychophysical studies of monkey vision--III. Spatial luminance contrast sensitivity tests of macaque and human observers. Vision Research, 14, 75-82.

Derrington, A. M., & Lennie, P. (1982). The influence of temporal frequency and adaptation level on receptive field organization of retinal ganglion cells in cat. Journal of Physiology (London), 333, 343-366.

Derrington, A. M., & Lennie, P. (1984). Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. Journal of Physiology (London), 357, 219-240.

Dudgeon, D. A., & Mersereau, R. M. (1984). Multidimensional digital signal processing . Englewood Cliffs, NJ: Prentice-Hall.

Enroth-Cugell, C., & Robson, J. G. (1966). The contrast sensitivity of retinal ganglion cells of the cat. Journal of Physiology (London), 187, 517-552.

Enroth-Cugell, C., Robson, J. G., Schweitzer-Tong, D., & Watson, A. B. (1983). Spatio-temporal interactions in cat retinal ganglion cells showing linear spatial summation. Journal of Physiology (London), 341, 279-307.

Field, D. J. (1987). Relations between the statistics of natural images and the response properties of cortical cells. Journal of the Optical Society of America A, 4(12), 2379-2394.

Foster, K. H., Gaska, J. P., Nagler, M., & Pollen, D. A. (1985). Spatial and temporal frequency selectivity of neurones in visual cortical areas V1 and V2 of the macaque monkey. Journal of Physiology (London), 365, 331-363.

Frishman, L. J., Freeman, A. W., Troy, J. B., Schweitzer-Tong, D. E., & Enroth-Cugell, C. (1987).

Spatiotemporal frequency responses of cat retinal ganglion cells. Journal of General Physiology, 89, 599-628.

Hamilton, D. B., Albrecht, D. G., & Geisler, W. S. (1989). Visual cortical receptive fields in monkey and cat: spatial and temporal phase transfer function. Vision Research, 29(10), 1285-1308.

Hawken, M. J., & Parker, A. J. (1984). Contrast Sensitivity and orientation selectivity in lamina IV of the striate cortex of old world monkeys. Experimental Brain Research, 54, 367-372.

Hawken, M. J., & Parker, A. J. (1987). Spatial properties of neurons in the monkey striate cortex. Proceedings of the Royal Society of London, Series B, 231, 251-288.

Hawken, M. J., & Parker, A. J. (1990). Detection and discrimination mechanisms in the striate cortex of the Old-World monkey. In Vision: Coding and Efficiency, ed. C. B. Blakemore (pp. 103-116). Cambridge: Cambridge University Press.

Hawken, M. J., Parker, A. J., & Lund, J. S. (1988). Laminar organization and contrast sensitivity of direction-selective cells in the striate cortex of the old world monkey. Journal of Neuroscience, 8(10), 3541-3548.

Heeger, D. J. (1991). Computational model of cat striate physiology. In Computational models of visual perception, ed. J. A. Movshon, & M. Landy Cambridge: MIT Press, In press.

Jones, J. P., & Palmer, L. A. (1987). An evaluation of the two-dimensional gabor filter model of simple receptive fields in cat striate cortex. Journal of Neurophysiology, 58(6), 1233-1258.

Kaplan, E., & Shapley, R. M. (1986). The primate retina contains two types of ganglion cells, with high and low contrast sensitivity. Proceedings of the National Academy of Sciences, USA, 83, 2755-2757.

Linsenmeier, R. A., Frishman, L. J., Jakiela, H. G., & Enroth-Cugell, C. (1982). Receptive field properties of x and Y cells in the cat retina derived from contrast sensitivity measurements. Vision Research, 22, 1173-1183.

Maddess, T., McCourt, M. E., Blakeslee, B., & Cunningham, R. B. (1988). Factors governing the adaptation of cells in area-17 of the cat visual cortex. Biological Cybernetics, 59, 229-36.

Mastronarde, D. N. (1983). Correlated firing of cat retinal ganglion cells. I. Spontaneously active inputs to X- and Y-cells. Journal of Neurophysiology, 49(2), 303-324.

Mastronarde, D. N. (1989). Correlated firing of retinal ganglion cells. Trends in Neuroscience, 12(2), 75-80.

Merigan, W. H., & Eskin, T. A. (1986). Spatio-temporal vision of macaques with severe loss of P-beta retinal ganglion cells. Vision Research, 26, 1751-1761.

Movshon, J. A., Thompson, I. D., & Tolhurst, D. J. (1978). Spatial and temporal contrast sensitivity of neurones in areas 17 and 18 of the cat's visual cortex. Journal of Physiology (London), 283, 101-120.

Ohzawa, I., & Freeman, R. D. (1985). Contrast gain control in the cat visual system. Journal of Neurophysiology, 54, 651-665.

Packer, O., Hendrickson, A., & Curcio, C. A. (1989). Photoreceptor topography of the retina in the adult pigtail macaque (Macaca nemestrina). Journal of Comparative Neurology, 288, 165-183.

Papoulis, A. (1965). Probability, random variables, and stochastic processes. New York: McGraw-Hill.

Pelli, D. G. (1990). The quantum efficiency of vision. In Vision: Coding and Efficiency, ed. C. B. Blakemore Cambridge, UK: Cambridge University Press.

Pointer, J. S., & Hess, R. F. (1990). The contrast sensitivity gradient across the major oblique meridians of the human visual field. Vision Research, 30(3), 497-501.

Purpura, K., Kaplan, E., & Shapley, R. M. (1988). Background light and the contrast gain of primate P and M retinal ganglion cells. Proceedings of the National Academy of Sciences USA, 85, 4534-4537.

Purpura, K., Tranchina, D., Kaplan, E., & Shapley, R. M. (1990). Light adaptation in the primate retina: Analysis of changes in gain and dynamics of monkey retinal ganglion cells. Visual Neuroscience, 4, 75-93.

Robson, J. G., & Troy, J. B. (1987). Nature of the maintained discharge of Q, X, and Y retinal ganglion cells in the cat. Journal of the Optical Society of America A, 4, 2301-2307.

Samy, C. N., & Hirsch, J. (1989). Comparison of human and monkey retinal photoreceptor sampling mosaics. Visual Neuroscience, 3, 281-285.

Sclar, G., Maunsell, J. H. R., & Lennie, P. (1990). Coding of image contrast in central visual pathways of the macaque monkey. Vision Research, 30(1), 1-10.

Tanaka, K. (1985). Organization of geniculate inputs to visual cortical cells in the cat. Vision Research, 25(3), 357-364.

Tolhurst, D. J., & Movshon, J. A. (1975). Spatial and temporal contrast sensitivity of striate cortical neurons. Nature, 257, 674-675.

Troy, J. B. (1983a). Spatial contrast sensitives of X and Y type neurones in the cat's dorsal lateral geniculate nucleus. Journal of Physiology (London), 344, 399-417.

Troy, J. B. (1983b). Spatio-Temporal interaction in neurones of the cat's dorsal lateral geniculate nucleus. Journal of Physiology (London), 344, 419-432.

Troy, J. B., & Enroth-Cugell, C. (1989). Dependence of center radius on temporal frequency for the receptive fields of X retinal ganglion cells of cat. Journal of General Physiology, 94, 987-995.

Watson, A. B. (1987). Estimation of local spatial scale. Journal of the Optical Society of America A, 4, 1579-1582.

Watson, A. B. (1990). Gain, noise, and contrast sensitivity of linear visual neurons. Visual Neuroscience, 4, 147-157.

Watson, A. B., & Ahumada, A. J., Jr. (1983). A look at motion in the frequency domain. In Motion: Perception and representation, ed. J. K. Tsotsos (pp. 1-10). New York: Association for Computing Machinery.