Vision and the Single Photon

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ABSTRACT

The human visual system has an amazing sensitivity – even a single photon catch can trigger the release of a signal in a rod photoreceptor cell under certain circumstances. However, behaviorally it requires on an average 5-8 photons for a human to 'see" a flash of light. This discrepancy is due to the intrinsic "dark noise" in the visual system. Various aspects of human visual sensitivity to single photons are reviewed and discussed.

Keywords: Photons, rods, visual sensitivity, retina,noise, psychophysics,photoreceptors, visual system

The visual process is complicated. However, a bottom-up view of the process that leads to our "seeing" can be broken up into essentially six major steps. These are: (1) imaging by which a spatial light distribution is created on the retina by the dioptrics of the eye, (2) detection, which refers to the transduction process by which the energy of the light quantum is absorbed by the photoreceptors (rods and cones) and converted into electrical potentials (transduction) and the subsequent neural activity, (3) encoding wherein the signal/information from the photoreceptors is decomposed, organized and encoded by subsequent neural layers, (4) transmission, to the brain of the encoded signal. (Here parallel visual pathways are used), (5) processing in which different components of the image are analyzed separately and (6) representation, identification, categorization, recognition and other higher level cognitive processes.

. The first step, imaging, is satisfactorily explained by basic optics [1]. In order to understand the other steps we need to have knowledge of the underlying molecular biophysics and neural science. The ultimate step (6) in the visual process is in the realm of cognitive science, massively parallel neural networks, and even in what is called neurophilosophy (e.g., see reference 2). It is also important to realize the dominance of vision in our sensory processes. Approximately 60% of all nerve fibers from a sensory organ to the brain come from the eyes. From the ear about 30,000 nerve fibers transmit acoustic information to the brain, while from each eye it is between 1 and 3 million. The brain cortex contains about 800000 nerve cells in the auditory cortex, while the visual cortex contains about 500 million nerve cells. Additionally, the eye operates over an amazing range of light levels, covering an intensity range of approximately 12 log units. A survey of various aspects of visual perception and neuroscience can be found, for example, in the books by Palmer[3], Norton et al[4], Devalois[5] or Wandell[6].

In this article dealing with the photon and the visual process, I shall review briefly the second step, namely detection and how discuss how it relates to the visual process. Like other sensory systems, the visual system has exquisite sensitivity (a fascinating review is given in reference 7). In particular, I will discuss the nature of vision at (or near) absolute threshold. Absolute threshold implies that rod photoreceptors signal the absorption of single photons and the resulting signal is transmitted across the neural layers of the retina and then to the brain. Since 1905, when Einstein refined Planck's quantum hypothesis scientists have investigated the fundamental question of how many photons are needed to "see". This question of the sensitivity of the eye is not simply one of physics, but also that of the criterion used by the observer, thus bringing in the behavioral response of human into the picture. This leads to the conclusion that questions such as this can only be answered in a statistical manner, in terms of a response probability. In fact, it was thought that the number of quanta might be very small (of the range of 1-100) and in fact Lorentz hypothesized that a just detectable flash of light delivered about 100 photons to the cornea[8]. Identifying the minimum number of photons

required for seeing from this number is difficult, because of uncertainties in determining the number of photons absorbed by the retinal photoreceptors. This quantum efficiency value has been estimated to be (based on scatter and absorption by the ocular media), to be in the range of 0.1 to 0.3., indicating anywhere from 10 to 30 photons for the observer to detect a flash of light. It is also known that the best values of threshold are obtained after total dark adaptation of nearly 30-45 minutes. It is found that the completely dark adapted rod photoreceptors approach the sensitivity limit set by the quantization of light as well as Poisson fluctuation in photon absorption. To get an idea of the sensitivity, it is found that on a moonless night only one rod in 10,000 receives a photon during the integration time of the rod signals. In fact isolated rod photoreceptors signal the absorption of single photons [8, 9] and (as noted below) psychophysical (behavioral) response requires the absorption of only a few photons (e.g., reference 10). This indeed is a far cry from the 10-30 absorbed photons. The classic work to answer this question how many photons are necessary to detect a flash was done by Hecht, Schlaer and Pirrenne[10] and by van der Velden [11]. In their classic paper, Hecht et al pointed out that the answer to the question of how many photons are required need to be stated statistically. They pointed out that only 5-8 quanta absorbed in the rods was required for a human observer to detect 65% of such flashes of light. The experiment is one of the classic "beautiful" experiments of all time and I require all my graduate students to study the original paper as an example of how a good experiment should be planned, conducted and analyzed!

Hecht et al (as well as other studies) [e.g., 10, 12] measured the fraction of trials in which a flash was seen as a function of the number of photons at the cornea. This curve, called a psychometric function showed a broad transition from flashes that were rarely seen to those frequently seen, and from this curve, the threshold and quantum efficiency were determined based on the assumption that the variability in a subject's response was due to Poisson statistics of photon absorption. Implicit in the analysis are two other basic ideas: only those instances wherein the number of photons which exceed a threshold number T were seen and that the average number of photons contributing to 'seeing' was directly proportional to the number of photons incident at the cornea, **N,** the constant of proportionality being the quantum efficiency **Q**. With these assumptions, we can write a simple Poisson process that relates the probability of seeing a flash delivering on average **N** photons to the cornea as:

$$P = \sum_{n \geq T}^{\infty} \frac{\exp(-QN)(QN)^n}{n!}$$

The above expression can be used to calculate the threshold from the slope of the psychometric curve. If the threshold is small, the Poisson fluctuations in the number of absorbed photons from one trial to the next will make the transition broad; the transition will become steep with increasing threshold. The quantum efficiency can be calculated by the shift required by the curve plotted from the above equation to match the experimentally obtained frequency of seeing data. Hecht et al determined a quantum efficiency of about 0.06, a number considerably lower than the estimate based upon the ocular media properties. It should be emphasized that even with higher quantum efficiencies, the probability likelihood that a single rod absorbed more than one photon on any trial is very small, since the test spot used in the experiments covered an area on the retina containing about 500 rods. In fact, they state "This small number of quanta in comparison with the large number of rods involved (500), precludes any significant two quantum absorptions per rod and means that in order to produce a visual effect, one quantum must be absorbed by each of 5 to 14 rods in the retina".

Why this discrepancy between the result that individual rod receptors detect single photons and the 5-8 photons necessary for 'seeing"? The frequency of seeing analysis made a fundamental assumption that all noise in the visual system is the result only of Poisson fluctuations in photon absorption. If this is strictly true, visual sensitivity will reach the quantum limit. In reality, visual sensitivity is hampered by background noise occurring along the visual retina-cortex pathway. There have been many studies to describe and show physiologically and mathematically the events occurring in the retina in response to stimuli and when no stimuli are present. In the following sections, I will attempt to answer the following three questions: What is happening in the visual pathway that creates this spontaneous activation of visual signals, what effects does this have on some of our visual abilities, and how does the brain interpret and differentiate between noise (false positive) and light (true positive) signals? Biologically induced variations in thresholds for stimuli processing has been described in other senses as well as for vision. The amount of random or biological noise in our systems is a difficult area of study, because our amazingly sensitive visual system is not fully understood; it is an interesting topic for study nonetheless.

What is retinal background noise? Spectral absorptions that occur in the photoreceptors cause a neural cascade which is encoded as sight in the occipital cortex, however, there are ever present visual stimulations occurring randomly without photonic absorption by the photoreceptors. Vision begins when the protein molecule rhodopsin in the photoreceptor outer segment absorbs a photon from the visual spectrum of light. Here in the retina, the photoreceptors align themselves according to the exit pupil of the eye, where photon absorption is maximized. This photoreceptor alignment and orientation and consequent waveguide properties of photoreceptors are collectively known as the Stiles-Crawford effects. In other words, photoreceptors behave as classic fiber optics elements, guiding the incident electromagnetic wave to sites of absorption, the rhodopsin molecules. What is interesting in this process is the fact, that in this aspect of light transduction, we can consider light propagation in terms of the wave nature of light. In fact, the familiar waveguide modal patterns have been observed in human photoreceptors. A good review of various aspects of photoreceptor orientation, and waveguiding can be found elsewhere.[13-15] The rhodopsin molecule is an intrinsic membrane protein. Retinal (vitamin A) is attached to the rhodopsin molecule in the 11-cis configuration. Light energy conducts an isomerization of the retinal from this 11-cis form to all-trans retinal. After procession through several intermediate forms, opsin detaches from the rest of the molecule. A G protein functions to initiate a cascade that lowers cyclic GMP levels in the outer segments, the loss of cGMp will cause a disruption in the ion flow occurring at the plasma membrane and a hyperpolarization ensues that will project from photoreceptors through the retina, to ganglion cells through the optic nerve and continue to the occipital lobe to be perceived as "sight".

A series of molecular changes in the photoreceptors initiate the birth of a visual event. Trans-cellular ion flow is maximal in the dark (dark flow), and photonic absorption interrupts dark flow. Cyclic GMP usually functions to hold open cation channels in the plasma membrane, creating a transcellular depolarization. Under normal dark conditions, calcium and sodium move intracellularlly, and potassium diffuses extracellularlly in the inner segment of the photoreceptor. In the absence of light, this ion flow allows an inner potential of around -40mV. At this membrane polarization level gammaminobutryic acid (GABA) is released as the neurotransmitter from the rod pedicles and cone spherules terminals to the bipolar cells. The closure of these protein gated channels creates a potential of about -65mV. At this new negative potential, glutamate is used as the neurotransmitter across the synaptic cleft and continuing through the neural circuit of the visual system. A wonderful account of the basic steps in the visual process (as well as a good introduction to the retinal circuitry is given in references 16 and 17).

Given this background on basic events in the transduction process it is now possible to discuss some aspects of visual noise. Horace Barlow in a classic article[18] attacked this intriguing discrepancy between single photon excitation and the number necessary for seeing and advocated the then-revolutionary concept of "dark light", being responsible for the difference. Dark light was a name given to internal events, such as spontaneous decomposition of photopigment (Larry Thibos of Indiana University has suggested that these events be renamed as *scotons* or *photoffs*). Barlow hypothesized this based on the fact that observers occasionally report seeing a flash even when no light was delivered and that the detection threshold depended on these false-positive responses. Barlow did experiments in which observes use two criteria –"yes" or "maybe" in a frequency of seeing experiment. He found that the "maybe" responses had a lower threshold and higher false positive rate than "yes" responses. Barbara Sakitt [19], modified Barlow's experiment by having the subjects rate the strength of a series of dim flashes (magnitude estimation procedure) on a scale from 0-6, 0 being no light at all to 6 being very bright. From a series of experiments she constructed a series of frequency of seeing curves and estimated thresholds for ratings of 1 or more, 2 or more, etc. As expected, as criterion rating increased, the number of false positives and sensitivity decreased. These papers show that false positives can trade for detection threshold across a range of criteria. This implies that different criteria correspond to different signal to noise ratios and observers choose where to operate depending on how many "mistakes" they are allowed to make. These experiments show that only a very small number of photons, possibly only one, contribute to detection. Barlow and Sakitt have assumed that the noise is an additive component. In this case the probability of seeing becomes:

$$P = \sum_{n \geq T}^{\infty} \frac{\exp(-Q(N+D))(Q(N+D))^n}{n!}$$

Where D is the additive Poisson noise expressed as an equivalent number of photons at the corneal plane. If this is assumed, then it is possible to show that even in darkness (N=0), the probability of seeing is non-zero. In fact, Barlow fit his "yes" and "maybe" results with the above equation and showed that they had a common amount of 'dark light" (but of course, different thresholds). Sakitt did similar analysis and showed that only the threshold changed between different criteria.

A major problem with the psychophysical experiments is that fits to the frequency of seeing curves are not unique. This is because, the behavioral quantum efficiency can trade for additive Poisson noise when fitting frequency of seeing data . This uncertainty in quantum efficiency produces a nearly 10 fold range in estimates of threshold and dark light. In fact, it has been estimated, based on spatial and temporal summation characteristics of the rod array, rod density, assumed quantum efficiency, etc., that the equivalent rate of photon like noise events in rod photoreceptors that the dark light ranges from 0.002 to 0.03/sec[20]. Lillywhite[21] has suggested that multiplicative noise could help resolve the discrepancy between psychophysically obtained quantum efficiencies and absorptive quantum efficiencies. This model assumes that several Poisson noise sources operate sequentially, and the product of their probability distributions determines the response statistics. Of course, additive noise is still needed to account for the false positives [22]. An important caveat in psychophysical experiments is that they combine into one all parameters that could lower sensitivity, including central factors [23,24]. Researchers have done electrophysiological studies on the fidelity of signals in the retinal ganglion cells[25-28]. These results show that the retina can detect and process single photon responses. Their results show that there is a source of discrete independent noise events originating in the rods or in the retinal circuitry.

Spontaneous background noise in the retina is usually described as having two components. Baylor et al[29,9] described discrete thermal activations in the photoreceptors which accounts partially for background noise. Light absorption raises an electron to a higher energy level, allowing normal isomerization from cis to trans retinal. Thermal activation can presumably produce nuclear vibrations that produce this same cis-trans isomerization that occurs in response to true photon absorption[30]. Thus a component of retinal noise is thermally induced. Another portion of retinal noise has its origins in fluctuations in the concentration of cGMP caused by cGMP phosphodiesterase (PDE), which is responsible for dark noise release of GABA[31]. PDE concentrations vary in tiny increments, enough to throw our intricate visual system off and cause some spontaneous false positives. Background retinal noise is generated among both the rod and the cone photoreceptors. Cones have been shown to be noisier than rods, possibly hampering our visual ability when we fixate on important stimuli in our environment, but presumably having less of an effect on our sensitivity at absolute visual threshold levels. This would make sense because we know rods function as low light detectors, and would therefore necessitate quieter biological conditions. In addition, it is also found that the power spectrum of dark noise had the same shape as the spectrum of a dim flash of light, evidence that that retinal noise consists of random events with an average shape of the single photon response. As far as their research could describe, the false positive noise isomerizations looked identical to true light reactions. Therefore the neural circuit must have some way of weeding out the false hits, or setting a strict criterion to disallow weaker signals from the retina.

Physiological noise is indistinguishable from signals generated by light stimuli, and it is hypothesized to be the main neural limit to our visual acuity. Continuous noise in mammalian rods can generate fluctuations that look very much like true photon responses[9]. Researchers have attempted to determine how much retinal background noise contaminates our processing of true retinal light signals. Using salamanders, Chichilinisky and Rieke[32] were able to show that the sensitivity of rod signals was limited by continuous noise in the rod photoreceptors. When visual stimuli were strong and in quick succession, noise in the rod photocurrent limits the ability the salamander retina to compute true responses. Their work also exhibits variation between different species; toads and primate studies have shown different results. Thus it cannot be generalized that retinal noise is temperature dependent across different species, especially to humans, although it has been shown to be true in toads. So if rods are capable of detecting single photon absorptions, why is our visual system incapable of the same? Biological noise in our retina limits our sensitivity at the absolute threshold of light detection; does this affect our everyday visual competence?

The equating of noise limited behavior with noise due to spontaneous activation of rhodopsin neglects other sources of noise in the rods, such as continuous noise [29,31,33]. It has been hypothesized that single photon responses can be separated from continuous noise by a threshold nonlinearity at the synapse between rods and rod bipolar cells in the retinal circuity[34-36] which would also reject the rod's ingle photon response. It is also possible that rods don't just detect the presence or absence of light but are useful in other visual tasks which set forth further limits on sensitivity. These could include estimating motion by analyzing temporal information from the rod array. This can be achieved by encoding photon arrival times precisely and band pass filtering at the rod synapse to extract the information [37,38] .

The understanding of how the retina encodes and transmits the signal from photon absorption is not completely understood. Could retinal noise be the dominant limit to our ability to "see" photon levels below our threshold? It is unknown where exactly the blame lies. There is significant noise throughout our visual system not only in the outersegments, but also horizontally and vertically through the retina and along the entire neural circuit. Background noise does exist from spontaneous events in the photoreceptors outer segments. There is also a level of noise in the post-photoreceptors visual circuitry of the retina. Portions of our visual systems noise must be attributed to retinal neural-circuit intricacies and further neural circuit difficulties from optic nerve to cortex. There are many unknowns in our knowledge, for example, we do not know the statistical properties of vesicle release at the synaptic junctions. Our understanding of how single photon responses are processed at the synapses and encoded in subsequent neural layers is incomplete. Recent anatomical and physiological work has identified other pathways that rod signals can take through the mammalian retina (see reference 39 for a review). It has been hypothesized that these other pathways process rod signals over a wide range of light levels. Is it possible that our eyes "sees light" when our brain does not at absolute visual thresholds? It is also important to point out another important facet of vision, namely, the so-called *Principle of Univariance*. This principle basically states that once a photon is absorbed, all information about it (i.e.,wavelength) is lost for subsequent processing. This Principle of Univariance was already implied in Thomas Young's formulation of the trichromatic process of color vision. It implies that the response of a visual photoreceptor depends upon the number of quanta it catches but not upon the wavelength of the quanta. Rhodopsin, for example, absorbs green light more readily than red, and thus green light will excite rods more than will an equal energy of incident red light. If instead of measuring the energy of the incident light, we measure the energy absorbed, then it is found that red and green lights that are equally absorbed will have identical rod-exciting effects and will appear identical by rod vision. From photon incidence, through transduction, along integration, and until visual encoding, our sense of vision it is a beautifully complex mechanism. The retinal pathway also serves us when light levels are extremely high, so our visual sensitivity in low light levels is insignificant compared to variety of conditions in which we need our visual systems to perform. The human visual system is extremely sensitive and capable of performances that trump our ability to understand it completely (yet!).

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