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Quantum and Non-Quantum Formulation of Eye's Adaptation to Light's Intensity Increments

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Abstract

Context and background: A quantum formulation of vision in vertebrates was proposed in the early 1940s. The number of quanta useful for enabling vision was found. The time interval required for their absorption, however, was never specified. In the early 1950s, experimental data on the effects of light's intensity increment on vision indicated that the quantum formulation is true only at low light's intensities. In this case, a vaguely described signaling adaptation mechanism was invoked to explain the separation between vision at low and high intensities, accompanied by the switch from rod to cones as photoreceptors. **Motivation:** In this article, we want to prove the validity of the non-totally-quantum formulation and unveil the nature of the signaling adaptation mechanism. **Hypothesis:** To accomplish our proof, we hypothesize that the amount of energy transferred and conserved in light's interaction with the eyes is given by the product of light's intensity (or power) times its period. **Method:** We construct and use the plots of the trends of light's intensity increments and the corresponding changes in the axon's membrane capacitance versus adapting intensity. **Results:** We find that 1) the average solar light's intensity is the critical value that separates low from high light's intensity regimes in vision, and 2) changes in the capacitance of the axon's membrane enable the signaling adaptation of vision when light's intensity changes. **Conclusions:** We prove the validity of the non-totally-quantum formulation and unveil the nature of the signaling adaptation mechanism. Our proof is supported by the model based on light's intensity times period as being the energy conserved in light-matter interaction This model suggests that 1) all the waves in the electromagnetic spectrum, at the correct intensity for each frequency, could be used to produce the effects of optogenetics in diagnostics and therapy, and 2) it takes seconds to minutes to see details in the dark when light is switched off.

Keywords

Biosignaling, Visible Light, Vision, Intensity Increment

1. Introduction

Recent experimental results in infrared (IR) spectroscopy [1] and in the description of the interaction between IR light and capacitors [2] show that light's intensity (power/area) plays a critical role in establishing the amount of energy transferred to capacitors and conserved in the interaction with them. Specifically, through the law of conservation of energy it was found that the magnitude of the product of light's power P times light's period τ, *i.e.* $P\tau$ (where $\tau = \frac{1}{\nu}$, and ν is frequency), equals the magnitude of the electrical, thermal, and mechanical energy, or combination thereof, transferred from the electromagnetic (EM) wave to the capacitors. In this article, we show that $P\tau$ is the energy conserved also in the mechanism of vision in vertebrates, which involves the nervous system and its complex network of components acting as capacitors. In proving this conclusion we solve the dispute between the quantum and non-quantum formulation of eye's adaptation to light's intensity increments.

In order to explore the consequences of the hypothesis that $P\tau$ is the energy conserved in the mechanism of vision in vertebrates, we briefly review the process of vision [3]. The nerve cells, or axons, can be depicted as capacitors in which the axon's membrane acts as the dielectric layer, whereas the intra- and extra-cellular fluids, inside and outside the axons, respectively, act as electrodes. These fluids consist of Na^+, K^+, Ca^{2+}, and Cl^- ions in solution. When light hits the eye, a decrease of the cyclic guanosine monophosphate (cGMP) concentration takes place, which closes the Na^+ and K^+ ion channels [3]. This phenomenon triggers the hyperpolarization from −40 mV to −70 mV of the axon's membrane. Afterwards, the closure of the Ca^{2+} gate causes the Ca^{2+} concentration to drop, generate a cascade of biochemical processes [3] [4], and depolarize the axon from its resting potential of −70 mV. If sufficient energy is supplied to depolarize the axon to the threshold potential of ~−55 mV (a voltage jump of ~15 mV), an action potential (V_{ap}) is fired. In turn, the action potential polarizes the axon up to +40 mV at the synaptic terminal. The total voltage jump from −70 mV to +40 mV has a magnitude of ~110 mV. A wave of action potentials thus generated can travel along the axons to the axon terminal, and then to the optic nerve [3]. The production rate of the action potentials is related to light's intensity.

We hypothesize that, during the formation of the action potentials, light's energy is transformed into electrical energy $\frac{1}{2}CV^2$, where C is the capacitance of the axon's membrane [5], and V is the depolarization voltage from the cell's resting potential of −70 mV to the threshold potential of ~−55 mV. The depolarization voltage V has a magnitude of ~15 mV, and is the voltage needed to fire

the action potential V_{ap}. Based on this hypothesis, we contribute in the dispute arisen about a quantum or a non-quantum formulation of vision in vertebrates involving signaling through the axon's membrane embedded between the intra- and extracellular fluids. The dispute evolved between the early 1940s and 1950s with Hecht *et al.* [6] [7], and Müller [8] [9] as major protagonists. Arguing with photons, or quanta of light, in a low light's intensity regime, Hecht and coworkers found that a minimum threshold of about 8 photons is required to trigger vision [6] [7]. On the other hand, Müller found, through experimental data, that different levels of light's intensity require different photochemical adaptation [3] processes. This finding challenges Hecht's quantum formulation because, in Müller's words, "For different adapting intensities different proportionality terms would relate the number of quanta absorbed and the stimulus intensity" [9]. The stimulus intensity thus changes the number of quanta absorbed by the eye in a defined time interval. Specifically, at low light's intensity, for which transduction is carried out by rod cells, the process of adaption is different than at large light's intensity, where the transduction process is carried out by cone cells. At low light's intensity, the predictions of Hecht and coworkers are confirmed in Figure 3 in [9]. At large light's intensity, however, the absorption constant changes, and the response of the eye departs from the trends at low light's intensity, and thus from the prediction by Hecht *et al.* [6] [7]. However, neither does Müller provide an explanation of his findings, nor does he address other questions generated by his results, such as: 1) What is the adaptation process that adjusts the behavior of the eye to increasing light's intensity? 2) What factor decides the magnitude of the light's intensity that separates its low and high regimes, shifts the transduction process from rod to cone cells, and establishes the regime in which the formulation by Hecht *et al.* [6] [7] is not valid?

In this article, we reproduce the results in Figure 3 in [9], and attempt to respond to the questions listed above that were neglected by Müller. Our strategy consists of using the law of conservation of energy, along with the assumption that $P\tau$ is the energy conserved in the mechanism of vision in vertebrates. We propose that the adaptation process employed by the eye to adjust its behavior to increasing light's intensity consists of changing the magnitude of the axon's membrane capacitance C: small values of C are pursued at small light's intensities, whereas larger values are pursued as light's intensity increases and approaches a critical value I_0. Thus, larger C values, associated to larger light's intensities, are also associated to larger rates of production of action potentials. The ability of the axon's membrane to change its capacitance underlines that, unlike the capacitors used in electronics, which have a fixed capacitance, the "capacitors" in the nervous system have a variable capacitance. We identify the critical intensity I_0 with the average intensity of solar light: 136 mW/cm^2, according to data provided by the Laboratory for Atmospheric and Space Physics—University of Colorado at Boulder. With the information on light's intensity and axon's capacitance, we reproduce Müller's plots of $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ [9],

and define $\Delta I = I_0 - I$ as the intensity increment. We then show that the critical intensity I_0 generates a singularity in the behavior of the eye's response to an intensity increment $\Delta I = I_0 - I$ as a function of the adapting intensity *I*. Furthermore, we find that the above-mentioned singularity at $I = I_0$ separates the low and high regimes of the adapting intensity *I*. Finally, we picture the adaptation mechanism to light's intensity increments as consisting of changes in the axon's membrane capacitance *C*. In low light's intensities, these changes in *C* are slow, as they are related to the slow synthesis of rhodopsin in rods, while in intense light the changes in *C* are fast, as they are related to the fast synthesis of the opsins in cones. This picture explains, for instance, why it takes seconds to minutes to the eye to define the details of objects in the dark, when light is suddenly switched off. The need of a certain time interval to implement the adaptation mechanisms is evident also in other adaptation processes, e.g. chemotaxis [3], *i.e.* the directional motion of cells towards a source of a chemical gradient. Other adaptation processes requiring time are described in [10].

Our findings related to $P\tau$ as being the energy conserved in the mechanism of vision in vertebrates, suggest a possible application to optogenetics [10] [11]. This technique exploits the ability of light to activate viruses and generate a potential across cellular membranes [12] [13]. Assuming $P\tau$ to be the amount of energy conserved in light-matter interaction, we can estimate the energy effective in activating the viruses. For example, with typical optogenetics parameters, such as blue light at wavelength $\lambda = 450$ nm, and power $P = 3.5$ mW [11] [14], it is possible to activate specific axon terminals in the parabrachial nucleus [11]. The energy $P\tau$ in this case is 5.25 aJ, and the depolarization voltage of 15 mV, needed to fire the action potential, is achieved if the capacitance in the axon's membrane is ~46 fF. On the other hand, with green light at $\lambda = 561$ nm and $P = 12$ mW, it is possible to silence, or deactivate, the axon terminals in the parabrachial nucleus [11]. The energy supplied by this green light to the virus is $P\tau = 22.44$ aJ. Achieving a voltage smaller than the depolarization voltage $V = 15$ mV, necessary to avoid firing the action potential, requires a capacitance of the axon's membrane as large as ~200 fF, in agreement with the arguments on adaptation and related time intervals discussed in the previous paragraph [10] [12]. From these two examples we infer that, if energy is the major issue explaining the effectiveness of optogenetic tools, than activation and silencing of the axon terminals in the parabrachial nucleus, as described in the examples above, could be successfully achieved by radio waves at, e.g. $\tau = 20$ ns, $P_{\text{activation}} = 0.26$ nW, and $P_{\text{silencing}} = 1.12$ nW, respectively. This hypothesis would be the basis for "radio-genetics" using radio waves (not just low-frequency magnetic fields as in [15]).

2. Methods

We use the law of conservation of energy to describe the transfer of energy from light to axons as follows:

$$P\tau = \frac{1}{2}CV^2, \tag{1}$$

where P and τ are light's power and period, respectively, C is the capacitance of the axon's membrane, and V is the depolarization voltage needed to fire the action potential. For most axons, $V = 15\text{ mV}$. The transfer of energy from light to the axon occurs indirectly through a complex biochemical process known as the visual cascade [3] [4]. In order to start the visual cascade, the energy $P\tau$ in Equation (1) needs to be at least as large as the activation energy of the biochemical reactions involved in the visual cascade. To maximize the probability of such a match, the vertebrate's eye evolved such as to adapt the capacitance in the axon's membrane to the critical intensity $I_0 = 136\text{ mW/cm}^2$ of the solar light, where $I_0 = \frac{P_0}{\text{area}}$. More specifically, this adaptation means that, in the presence of solar light, the axon's membrane is required to have an adequate value of the capacitance C. Thus, using Equation (1) we can estimate that, to produce the depolarization voltage $V = 15\text{ mV}$ necessary to trigger an action potential with green solar light at wavelength 532 nm (or $\tau = 1.77\text{ fs}$), requires $C = 2.14\text{ pF}$. This value is in good agreement with the baseline capacitance of few pF found in retinal axon membranes [5]. This baseline capacitance changes when light induces an axon's depolarization process, thus an action potential [5]. Typical magnitudes of capacitance changes are of the order of $\Delta C_0 = 200\text{ fF}$ (or 0.2 pF) [5]. We designate ΔC_0 as the critical capacitance change.

With this information we begin the investigation of the effects of low light's intensity on the eyes, searching in particular the conditions needed to supply the depolarization voltage V of 15 mV required to drive the axon from the resting potential of −70 mV to the threshold potential of −55 mV, and so fire action potentials. As previously mentioned, to investigate these effects we follow a procedure similar to that adopted in [9], which consists of plotting $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ (where $\Delta I = I_0 - I$). Before illustrating our results we note that, while we have a specific value for I_0 ($I_0 = 136\text{ mW/cm}^2$, the critical intensity and average solar intensity), [9] does not provide any magnitude for any critical intensity, and discusses only the magnitude of the ratio $\frac{I}{I_0}$, without reference to the possible specific values of I and I_0. In our computation, we start off considering light at $I = 1\text{ mW/cm}^2$, then slowly allow I to increase up to $I_0 = 136\text{ mW/cm}^2$, and beyond. The values we compute in this procedure for $\frac{I}{I_0}$, $\log_{10}\left(\frac{I}{I_0}\right)$, $\frac{\Delta I}{I}$, and $\log_{10}\left(\frac{\Delta I}{I}\right)$ are all reported in **Table 1**. The values in **Table 1** enable us to plot $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ in **Figure 1(a)**. This plot is similar to the one in Figure 3 of [9], reproduced in **Figure 1(b)**,

which Müller obtained from experimental data of intensity discrimination. In addition, in Table 1 we report the values of the capacitance C obtained from Equation (1) solved for intensity $I=\frac{P}{\text{area}}$ assuming $\tau=1.77$ fs (green light at 532 nm) and $V=15$ mV (the depolarization voltage required to drive the axon from its resting potential of −70 mV to the threshold potential of −55 mV). Thus, we compute C as $C=\frac{2I\tau}{V^2}$, from Equation (1), then $\frac{\Delta C_0}{C}$, and $\log_{10}\left(\frac{\Delta C_0}{C}\right)$. Figure 1(c) reports $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$. In [9] there is no graph corresponding to Figure 1(c).

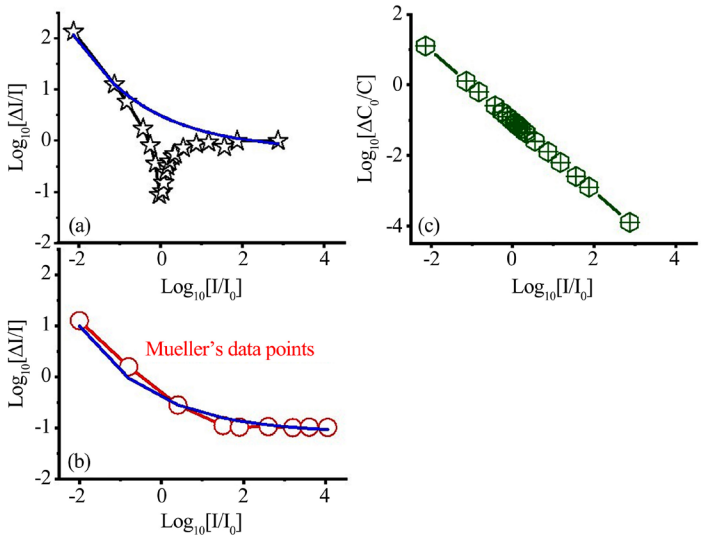

Figure 1. (a) Plot of $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$, where I is light's intensity, I_0 the critical intensity, which coincides with the average intensity of solar light: 136 mW/cm^2, and $\Delta I=I_0-I$. The intensity I ranges from 1 mW/cm^2 to 10^5 mW/cm^2. The numerical value of each point is taken from Table 1. The continuous fitting line consists of the exponential function $\left\{0.69\exp\left[-\frac{\log_{10}(I/I_0)}{1.8}\right]-1.1\right\}+0.9$. (b) Plot of $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ from the 9 experimental data points reported in Figure 3 of [9], from experimental intensity discrimination studies. The continuous fitting line consists of the exponential function $0.69\exp\left[-\frac{\log_{10}(I/I_0)}{1.8}\right]-1.1$, which is the same as the fitting line in Figure 1(a) without the +0.9 offset. (c) Plot of $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$, where $\Delta C_0=200$ fF (or 0.2 pF) is the critical capacitance change [5]. The values in the abscissa are the same as in Figure 1(a). The values of C, computed from Equation (1), $\frac{\Delta C_0}{C}$ and $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ are reported in Table 1.

Table 1. For various light's intensities I we compute and report the values of $\frac{I}{I_0}$, $\log_{10}\left(\frac{I}{I_0}\right)$, $\frac{\Delta I}{I}$, and $\log_{10}\left(\frac{\Delta I}{I}\right)$. We note that I_0 is the critical intensity, identified with the average intensity of solar light: 136 mW/cm^2, and $\Delta I = I_0 - I$. The computations start from low light's intensity at 1 mW/cm^2, and move to the critical intensity I_0, and beyond. In column 5, the numbers that are the real part of complex numbers are highlighted with an asterisk (*). Complex numbers arise because of the singularity at I_0 reported in **Figure 1(a)**. The presence of the singularity at I_0 signifies that for $I > I_0$ the ratio $\frac{\Delta I}{I}$ is negative ($\frac{\Delta I}{I} < 0$). Thus, $\log_{10}\left(\frac{\Delta I}{I}\right)$ is a complex number. For the same values of light's intensity I, and using Equation (1) with $I = \frac{P}{\text{area}}$, where P is light's power, we compute and report the values of the axon's membrane capacitance C. In our computation, $C = \frac{2I\tau}{V^2}$ from Equation (1), where we assume $V = 15$ mV, the depolarization voltage needed to trigger an action potential, and $\tau = 1.77$ fs, the period of green light with wavelength 532 nm. We then compute $\frac{\Delta C_0}{C}$ and $\log_{10}\left(\frac{\Delta C_0}{C}\right)$, where $\Delta C_0 = 200$ fF (or 0.2 pF) [5] is the critical capacitance change in the axon during the visual cascade [3] [4]. **Figure 1(c)** reports $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$.

I [mW/cm^2]	$\frac{I}{I_0}$	$\log_{10}\left(\frac{I}{I_0}\right)$	$\frac{\Delta I}{I} = \frac{I_0 - I}{I}$	$\log_{10}\left(\frac{\Delta I}{I}\right)$	C [fF]	$\frac{\Delta C_0}{C}$	$\log_{10}\left(\frac{\Delta C_0}{C}\right)$
1	0.0073	−2.13668	135.2	2.13	15.73	12.71	1.10415
10	0.073	−1.13668	12.6	1.1	157.3	1.2715	0.104303
20	0.147	−0.8327	5.8	0.763	314.6	0.6357	−0.19673
50	0.3676	−0.43462	1.72	0.235	786.5	0.2543	−0.59466
75	0.55	−0.2596	0.813	−0.0899	1179.7	0.1695	−0.7708
100	0.7353	−0.1335	0.36	−0.444	1573.0	0.127	−0.8957
125	0.919	−0.003668	0.088	−1.055	1966.2	0.1017	−0.9926
150	1.103	0.04257	−0.103	−0.987(*)	2359.5	0.0848	−1.0718
160	1.176	0.0704	−0.15	−0.824(*)	2516.8	0.0795	−1.0998
180	1.323	0.12156	−0.244	−0.6126(*)	2831.4	0.0706	−1.15095
200	1.47	0.1673	−0.32	−0.4948(*)	3146.0	0.0636	−1.19673
225	1.645	0.2185	−0.395	−0.403(*)	3539.2	0.0565	−1.24787
270	1.985	0.29776	−0.4963	−0.3042(*)	4247.1	0.0471	−1.32706
300	2.206	0.3436	−0.547	−0.262(*)	4719.1	0.0424	−1.37284
500	3.676	0.5654	−0.728	−0.1379(*)	7865.0	0.0254	−1.594654
1000	7.353	0.8665	−0.864	−0.0635(*)	15730	0.0127	−1.89585
2000	14.706	1.1675	−0.932	−0.0306(*)	31,460	0.0063	−2.19675
5000	36.76	1.5534	−0.9728	−0.012(*)	78,650	0.0025	−2.59465
10,000	73.53	1.86646	−0.9864	−0.0059(*)	157,300	0.0013	−2.89585
100,000	735.3	2.86646	−0.99864	−0.0006(*)	1,573,000	0.00012	−3.89585

3. Results

We compare **Figure 1(a)** with Figure 3 in [9], reproduced in **Figure 1(b)**. Müller's results in both Figure 3 of [9], and in **Figure 1(b)**, consist of 9 data points obtained after averaging over many trials in order to minimize the errors. The points in Figure 3 of [9], (reproduced in our **Figure 1(b)**) do not exhibit any singularity as the one appearing at $I = I_0$ in **Figure 1(a)**. Instead, the points in Figure 3 of [9], follow the continuous fitting line shown in both **Figure 1(a)** and **Figure 1(b)**. With an offset of +0.9, this fitting line overlaps the data points we provide from our computations in **Figure 1(a)**, both on the left and on the right side of the singularity at $\log_{10}\left(\frac{I}{I_0}\right) = 0$ (*i.e.* for $\frac{I}{I_0} = 1$). However, there is no overlap between the data points and the fitting line in the neighborhood of the singularity. Despite this mismatch, we must acknowledge that the singularity around $\log_{10}\left(\frac{I}{I_0}\right) = 0$ is required to justify the trends of the experimental data points reported by Müller [9]. Indeed, with logharitmic functions but without the singularity, we would expect the points in **Figure 1(a)** and **Figure 1(b)** to follow a straight line, which is not what is experimentally observed.

The analytical expression of the continuous fitting line consists of the exponential function $0.69\exp\left[-\frac{\log_{10}(I/I_0)}{1.8}\right]-1.1$ in Figure 3 of [9], and **Figure 1(b)**, and $\left\{0.69\exp\left[-\frac{\log_{10}(I/I_0)}{1.8}\right]-1.1\right\}+0.9$ in **Figure 1(a)**. The two functions are the same, except that an offset of +0.9 is applied to the fitting line in **Figure 1(a)**. The presence of the singularity at I_0 signifies that for $I > I_0$ the ratio $\frac{\Delta I}{I}$ is negative ($\frac{\Delta I}{I} < 0$). Thus, $\log_{10}\left(\frac{\Delta I}{I}\right)$ is a complex number, whose real part is reported in **Figure 1(a)** and **Table 1**. In **Table 1** the numbers that are the real parts of complex numbers are highlighted by an asterisk (*). The fact that $\log_{10}\left(\frac{\Delta I}{I}\right)$ is a complex number for $I > I_0$ might indicate that exposure to very large light's intensities promotes anomalous phenomena in vertebrate's eyes. An example of an anomalous phenomenon is the uneasiness we experience under exposure to extremely intense light, which we naturally and immediately avoid. The anomaly might be justified considering that vertebrate's eyes evolved in time constrained by the exposure to a maximum sun light's intensity of on-average 136 mW/cm^2, the value of the critical intensity I_0.

In Figure 3 of [9], and in our **Figure 1(b)**, we observe a continuous exponential line without singularity for $\log_{10}\left(\frac{I}{I_0}\right) > 0$. As previously discussed, the analytical expression of this line is $\left\{0.69\exp\left[-\frac{\log_{10}(I/I_0)}{1.8}\right]-1.1\right\}$. The absence of a

singularity can be explained by assuming that in Müller's experiment the critical intensity $I_{0\text{-Mueller}}$ was much less than 136 mW/cm^2 so that for the 7 points on the right of the graph in Figure 3 of [9], and in our **Figure 1(b)**, the condition $\frac{\Delta I}{I} < 0$ is verified. We thus conclude that $I_{0\text{-Mueller}} < I \ll 136\ \text{mW/cm}^2$. The fact that the data points in Müller's experiment require an exponential fitting function and those in our numerical experiment a logharitmic function, defined in an interval containing a singular point at $I = I_0$, might be adopted to explain the offset of +0.9 existing between the data in **Figure 1(b)** and those in **Figure 1(a)**.

Figure 1(c) illustrates some of the consequences of the change in the capacitance C of the axon's membrane occurring when the vertebrate's eye interacts with light of intensity I. Specifically, in **Figure 1(c)** we report $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$, where $\Delta C_0 = 200$ fF (or 0.2 pF) [5] is the critical capacitance change, and the abscissa is the same as in **Figure 1(a)**. Thus, we are describing the effects of the changes in C in the light intensity conditions examined in **Figure 1(a)**. To offer a quantitative description of these changes, in **Table 1** we report the values of C, $\frac{\Delta C_0}{C}$, and $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ for the same intensities I considered in **Figure 1(a)**. No singularity is observed with the capacitance changes because ΔC_0 has a fixed non-zero value. As a consequence, the trend in $\log_{10}\left(\frac{\Delta C_0}{C}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ is linear, as illustrated in **Figure 1(c)**. **Table 1** also shows that, in the light intensity range between 1 mW/cm^2 to 136 mW/cm^2, the values of the capacitance vary between 15 fF to ~2 pF. The observed trends suggest that the change of the photoreceptors from rods, at low light's intensity, to cones, in bright light, does not introduce singularities in the changes of magnitude of the capacitance.

4. Discussion

Our work contributes in solving the dispute between the quantum [6] [7] and the non-totally quantum formulation [8] [9] of the phenomenon of vision. Originally, the dispute evolved between the early 1940s and 1950s with Hecht *et al.* [6] [7], and Müller [8] [9], as major protagonists. The quantum formulation by Hecht *et al.* [6] [7], supporting the single-photon response-based explanation of vision [3], is to-date largely accepted. However, the work by Müller [8] [9], supporting the non-quantum formulation of vision, singled out some limitations of the single-photon response. Indeed, by collecting and analyzing experimental data, Müller showed a strong dependence of the process of vision on the adaptation mechanism required by the eyes to adjust to light's intensity changes. This dependence of the adaptation mechanism to light's intensity infers that the existence of the minimum threshold of about 8 photons is not sufficient to justify

the start of the vision process, as established in the quantum formulation [6] [7], as light's intensity changes. Müller drew his conclusions from the trends he detected in graphs representing $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ shown in Figure 3 of [9], and in our **Figure 1(b)**. Müller, however, was unable to explain the trend of his experimental data, which appear to follow an exponential rather than a logarithmic function. Probably due to this mathematical mismatch, Müller's conclusions were not granted further consideration in the scientific community. Our results in **Figure 1(a)**, on the other hand, reproduce Müller's experimental data and, in addition, are obtained using logarithmic functions. To construct the data reported in **Figure 1(a)**, we identify a critical intensity I_0 in the process of vision, which corresponds to the average intensity of solar light: 136 mW/cm^2. With this choice of I_0, we establish that the intensity increment is $\Delta I = I_0 - I$ and, with this assumption, we construct the graph representing $\log_{10}\left(\frac{\Delta I}{I}\right)$ versus $\log_{10}\left(\frac{I}{I_0}\right)$ shown in **Figure 1(a)**. In our computation, the intensity I_0 is extremely relevant because it originates the singularity illustrated in **Figure 1(a)**. We choose I_0 as the critical intensity under the hypothesis that the average intensity of solar light guided the evolution of the vertebrate's eyes. On the other hand, to derive the relationship between intensity (or $\frac{P}{\text{area}}$), voltage and capacitance in **Figure 1(c)** we use Equation (1). This equation implies that $P\tau$, where P is the power and τ the period of the light, is the energy of the visible light transferred and conserved in the interaction with the axons through the visual cascade [3] [4] to enable vision. Once transferred to the axons through the visual cascade, light's energy $P\tau$ is transformed into electric energy of magnitude $\frac{1}{2}CV^2$ stored in capacitors, *i.e.* the axons, as inferred in Equation (1), where C is the capacitance of the membrane in the axon, and V is the depolarization voltage of ~15 mV needed to trigger the action potential.

Equation (1) sheds light to the adaptation mechanism implied in Müller's experimental data and in our **Figure 1(a)**, suggesting that the capacitance C of the axon's membrane is the only parameter that can change within the axon to enable the eyes to adjust to the energy $P\tau$ transferred from light. The adjustment needs to occur such as to generate the depolarization voltage $V \sim 15\,\text{mV}$ necessary to trigger the action potentials: this is the criterion that enabled us to derive the values of C in **Table 1**. Changing the magnitude of C in the axon requires sufficient time (few seconds to minutes) to enable the appropriate type and amount of ions to actually penetrate the axon's membrane. This fact elucidates why our eyes require some time to adjust while passing from vision in high to low light's intensity. More specifically, low light's intensity needs very small capacitances, large $\frac{\Delta C_0}{C}$ ratios (see **Table 1**), and possibly large time intervals to allow the membrane to reach such small capacitances. Other phenomena tes-

tify that light intensity plays a significant role in adaptation mechanisms. For example, the unicellular green alga *Chlamydomonas* can swim toward or away from light. In this process, called phototaxis, this alga produces a photoreceptor current when illuminated by light. This photoreceptor current was found to increase with the light's intensity and with intensity's rate of increase [16].

The validity of Equation (1) in explaining vision's adaptation to different light's intensities has further implications, including the fact that it is a proof that the magnitude of the energy $P\tau$ is the amount of energy transferred and conserved during the interaction between light and matter. The quantity $P\tau$ should therefore be considered in optogenetics. This research- and therapy-technique is usually performed using blue or other visible light [11]. Adopting $P\tau$ in optogenetics seems to suggest that the same amount of energy transferred by, e.g. blue light with $\tau = 1.49$ fs (corresponding to $\nu = 0.67\times10^{15}$ Hz) at $P = 3.5$ mW, that is $P\tau = 5.2$ aJ or 32.6 eV, could be supplied by radio waves at $P = 0.26$ nW with $\tau = 20$ ns (corresponding to $\nu = 50$ MHz, which is within the frequency range of amateur radio transmitters). It is noticeable that the power with radio waves is 6 orders of magnitude lower than that with blue light. Thus, it is reasonable to ask whether the same results of optogenetics with blue or other visible light could be achieved with radio waves with the proper power. In other words, we might ask 1) whether optogenetics is related to the specific frequency of the EM waves used, or to the energy that these EM waves transfer, and 2) what is the impact of the wavelength of the EM waves on the size of the optogenetics samples, or on the ability of focusing the EM wave on the target. Pursuing the answer to these questions is the objective of research in progress.

5. Conclusion

The hypothesis that $P\tau$ is the energy conserved in the mechanism of vision in vertebrates enables us to support the non-totally quantum formulation of the mechanism of vision, and thus, that the existence of a minimum threshold of about 8 photons is not sufficient to justify the start of the vision process in vertebrate's eyes. In addition, our results unveil the adaptation mechanism required by the vertebrate's eyes to adjust to different levels of light's intensity. This mechanism consists of the change of the capacitance of the axon's membrane. Furthermore, we clearly mark the low and high-intensity regimes in vertebrate's vision by identifying the critical intensity I_0, corresponding to the average intensity of solar light: 136 mW/cm^2. Well below the critical intensity I_0, some features of the quantum formulation of the mechanism of vision are still effective. However, for $I > I_0$, this is not the case because of the changes occurring in the axon's membrane capacitance. This separation between low and high-intensity regimes was unidentified in previous experimental results supporting the non-totally quantum formulation of the mechanism of vision. Finally, our future plans include the application of our findings based on $P\tau$ and the law of conservation of energy to extend the spectrum of the electromagnetic waves suitable

as optogenetic tools.

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Conflicts of Interest

The author declares no competing financial interests.

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Nomenclature

C = capacitance

ΔC_0 = critical capacitance change

$I = \dfrac{\text{power}}{\text{area}}$ = intensity

I_0 = critical intensity

$\Delta I = I_0 - I$ = intensity increment

P = power

V = depolarization voltage

V_{ap} = action potential

ν = frequency

λ = wavelength

τ = period