Information Approach to Image Coding in the Neurobiological Retina

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Abstract. In this article, we explore several parallels between neurobiological and engineering approaches to the structure of the retinal neural system, drawing on information theory representations. We demonstrate that the information approach developed in this article allows for a much closer connection between these specialized concepts of neural networks.

Keywords: Source-Channel Separation Theorem, NLN model, Image Coding.

1 Introduction

Brain-inspired information technologies are a rapidly growing area of research, development, and innovation today [1]. The explosive growth of activity in this field is driven by the advantage and potential of biological neural principles over the traditional von Neumann paradigm in computing. It is enough to note such potential as: computing embedded in RAM; massive parallelism; extreme energy efficiency; event-driven / spiking computing; sparse coding [2]. Popular examples of brain-inspired systems being implemented include areas such as Edge AI and robotics, enabling autonomous vehicles and smart devices to process data locally, without the use of energy-intensive data centers; Sensory processing systems, such as silicon retinas and cochleae, artificial olfactory (AO) systems (e-noses), etc.; Brain-computer interfaces (BCI) for directly connecting biological brain signals to digital computers, for example, to control prosthetics; Medical modeling for matching brain scan data of individual subjects with neuromorphic patterns in neurological diagnostics, etc..

By its very nature, developing systems that mimic the functioning of the brain is an interdisciplinary field, combining neurobiology, computer science, and electronics, and focused on adapting the principles of biological intelligence to algorithmic and hardware solutions, including AI applications. However, the vastness of this field, its multifaceted nature, and a certain diversification significantly complicate cooperation between specialists from different fields. For example, the central question of how neural network functions arise from the activity of multiple brain cells is often treated in such a specific way in neurobiology and electronics that some specialists have virtually no understanding of others. We believe that the main reason for this misunderstanding is the lack of simple models that would allow us to, firstly, express in a clear

and compact form (for example, in the form of information concepts) the relationship between the input and output of neural circuits in the presence of natural input signals; secondly, to define an unambiguous computational structure that allows for the analysis of computations; and, thirdly, to establish a connection between the internal functional structure of the model and the functions of real biological neurons. [3].

It should be noted that the situation described above is somewhat exaggerated: today, several generally recognized successful models exist. For example, deep neural network models [4] have demonstrated high results in solving complex problems, including simulating object recognition by the human central visual system (HVS) or sound discrimination by the auditory cortex. However, due to the rather specialized formal description, it is unclear how to interpret the role of deep networks in terms of their contribution to general neural computations or how to relate them to individual biological neurons [3]. Existing Retina models – simple linear–nonlinear (LN) model, generalized linear model (GLM), or two-layer LN–LN model with nonlinear subunits demonstrate higher computational interpretability – the mathematical components of the models are even named using neurobiological terms [5]. However, the formal description of the input data – a light stimulus in the form of some integrable function and its processing in the outer layer of the retina using a spatiotemporal linear filter is not easy to interpret in precise neurobiological terms.

The LN model [6] of the retina, which is considered as a neural structure, is based on the well-established neurobiological fact that many neurons (in the retina, visual cortex, and other parts of the nervous system) have so called receptive fields (RF) structure. A neuron's RF is a region in a neural network that can influence a given neuron. For retinal neurons RFs are certain receptor regions in the outer layer of the retina. In its most well-known version [6], the LN involves (L) linear filtering with RF window of the recorded stimulus, followed by (N) nonlinear transformation of the filtered signal. The purpose of the latter is to prevent negative discharge frequencies, as well as to achieve a threshold value (action potential) and saturation of responses. The LN model has become the de facto standard model for the response of retinal ganglion (output) cells and underlies many approaches to neural computing.

Formally, the components of the LN model are defined mathematically as follows [5]. First, the gradient potential $B(\vec{x},t)$ of retinal bipolar cells, formed in response to a light stimulus, is formalized as a linear functional of the radiation intensity on the retina $S(\vec{x}',t')$:

$$B(\vec{x},t) = \int_{\vec{x}',t'} K(\vec{x}'-\vec{x},t'-t)S(\vec{x}',t')d\vec{x}'dt' \,, \qquad (1)$$

where the response kernel $K(\vec{x}'-\vec{x},t'-t)$ describes the RF of a bipolar cell located at position $\vec{x}$ on the retina. The bipolar cell potential $B(\vec{x},t)$ is then "rectified" by a nonlinear transformation at the input of the retinal ganglion cells (retina outputs) and, using a formula similar to (1), is recalculated to output as signal spikes.

The main achievement of the described LN model is its remarkable simplicity and interpretability of functions (1). Unfortunately, as experimental neurophysiological studies have shown, the LN model often poorly predicts the responses of retinal ganglion cells to realistic stimuli such as natural images. This led to the development of

the so-called LN-LN or "subunit" model [7]. In this model, each subunit is represented by an LN model, which includes its own type of independent spatial filter (1), the signal of which is nonlinearly transformed before summation into integrated ganglion cell excitation. The subunits are considered to correspond to bipolar cells that provide excitatory input to ganglion cells, so excitation in this model already exhibits nonlinear spatial integration.

Some time ago, in order to overcome the shortcomings of the LN model, we also proposed a new approach to modeling retinal functions [8]. It differs from the above-mentioned LN-LN model mainly in terms of the representation of the input data. Namely, instead of considering the values of the registered radiation intensity at the input of the retinal receptors as a continuous function $S(\vec{x}', t')$, we proposed to accept as input data the actual registration result, which is a set $\{\vec{x}_i\}$ of discrete (photo)counts in the outer segment of the receptors. From a physical point of view, this approach appears to be more adequate and universal, since it takes into account a uniform representation of the input data from the very beginning, regardless of, for example, whether the detected radiation is weak, medium, or bright. A number of arguments in favor of the proposed approach from the point of view of the physics of the phenomenon and the resulting quality of images formed from the counts can be found in [9]. In general, our proposed model occupies an intermediate position between the LN and LN-LN models, so we found it appropriate to denote it as the NLN model [8].

The input data representation in the proposed NLN model – the input image representation – is based on the ideal image concept we developed earlier [I0]. The latter considers the classical representation of an image in the form of radiation intensity distributed over the retina as the generation intensity function of discrete random events – counts [10]. Thus, an ideal image is a discrete set of a finite number of random counts $X = \{\vec{x}_i\}$, where $i = 1, \dots, N$ are the coordinates of the registered events within the light-sensitive two-dimensional surface. Note that the number of counts N is also a random variable. Starting from this concept, we were able to model the main processes in the retina from receptors to ganglion cells and show that, based on local optimization, it is possible to formulate a procedure for encoding the optic nerve, considered as a data transmission channel. Thus, in the work, relying primarily on information-theoretical concepts, we attempt to link, on the one hand, numerous neurobiological data, and, on the other, the algorithmic principles of neural networks. In this regard, the work begins with a review of a number of neurobiological facts about the structure of the retina and their information interpretation. Then, based on this material, in several subsequent sections, algorithms for processing information propagating along the visual pathways are sequentially constructed in accordance with the layered structure of the retina. The work concludes with a formulation of the procedure for coding ON- and OFF-visual channels and a discussion of the numerical organization of this procedure.

2 The Retina as Encoder of the Visual Information System

The modern concept of information systems and their basic principles were laid down by Claude Shannon and his colleagues [11]. Since then, the standard scheme of information (communication) systems is assumed to be a sequence of fairly general pipeline elements, also known as a data pipeline, connected in series: information Source → Encoder → data transmission Channel → Decoder → information Receiver.

One of the major achievements of K. Shannon is the discovery (1948) of the fact that source and channel coding can be relatively independent, connected only through output–input messages in the form of a sequence of some internal variables (digital in computer technology) [11]. This statement is known as the Shannon Source-Channel Separation Theorem and is a fundamental principle of information theory. In its precise formulation, the theorem states that the message transmission problem can be divided into two independent stages without loss of optimality: data compression (Source encoding) and data transmission (Channel encoding) [12]. Let us note here that the same applies to the decoder, although it is not considered in this work.

The visual system (in mammals) is a network of neurons that communicate with each other through specialized connections called synapses, and their purpose is to transmit visual information from the outside world (Source) to the brain (Receiver) for processing and analysis. The system includes the retina, the optic nerve, and then, via the lateral geniculate nucleus, regions of the occipital cortex. It is easy to see that the optic nerve in the standard diagram of an information system exactly corresponds to the data transmission channel, the retina to the encoder, and the cerebral cortex to the decoder (and information Receiver).

In this regard, the retina, as an encoder, converts messages from the Source – the intensity of light falling on its surface – into a neural impulse (spike) signal for the optic nerve. Incoming light is not only detected but also undergoes significant processing before the channel is encoded. For these purposes, the retina is equipped with a layered structure: the light-sensitive cells of the outer layer of the retina – photoreceptors (rods, cones) are followed by synaptically connected bipolar, horizontal and amacrine interneurons of the inner layer, which, in turn, form synapses with the cells of the ganglion layer — output cells of the retina (see Fig. 1) [13].

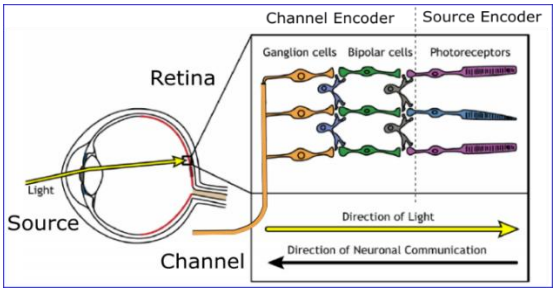

Fig. 1. Scheme of propagation of visual information along the retina (in the direction opposite to the propagation of light). Adapted from [13].

Light absorption occurs via photopigments in the outer segment of photoreceptors, which initiates a cascade of biochemical reactions that alter the membrane potential of the inner segment and, consequently, the amount of neurotransmitter released by the terminals of the photoreceptor (the process of phototransduction). Therefore, in accordance with Shannon's concept of information systems, it is natural to identify the outer (receptor) layer of the retina with the Source encoder (see Fig. 1). In this case, it is natural to consider the membrane potential of the receptors as encoder internal variables. It should be noted that, unlike most neurons in the central nervous system, retinal neurons, including photoreceptors (excluding ganglion cells), do not generate action potential. Instead, activation results in a continuous change in their membrane potential, which (to distinguish it from the action potential) is called a graded potential [14]. Changes in graded potential proportionally change the rate of release of the neurotransmitter acting on postsynaptic neurons. Interestingly, the type of neurotransmitter is the same for all retinal neurons—glutamate [15].

Synapses between photoreceptor terminals and bipolar cells (as well as horizontal cells) are formed in the outer plexiform layer, which separates the outer layer of photoreceptors and the inner layer of bipolar cells. Short axonal processes of bipolar cells, in turn, form synaptic contacts with the dendritic processes of ganglion cells in the inner plexiform layer. Much larger ganglion cell axons, gathered in a bundle, form the optic nerve and transmit information about retinal stimulation to the visual cortex (see Fig. 1) [14]. Therefore, in accordance with Shannon's concept of information systems, it is natural to identify the remaining part of the retina—the inner and ganglion layers—with the channel encoder (see Fig. 1).

Note in connection with the above review that the terms "inner" and "outer" in the retina denote relative locations from the center of the eyeball in the direction of light propagation (inner—closer to the center; outer—further away—closer to the pigment epithelium). The fact is that in the retina, the direction of information propagation is opposite to the propagation of light (see Fig. 1). Light, before being registered by the receptors, is forced to pass through all the retina layers.

3 The Source Encoding in Retina – the Photocounts

As outlined above, the outer layer of retina — the receptors layer is natural to identify with the Source encoder (see Fig. 1). The retinal receptors are composed of two types: rods and cones (see Fig. 2). Both types have two segments: the outer (adjacent to the pigment epithelium), consisting of membranous discs containing light-sensitive photopigment, and the inner, containing the cell nucleus and giving rise to synaptic endings (terminals) that contact bipolar or horizontal cells [14].

Rods and cones differ in shape (from which they derive their names), the type of photopigment they contain, their distribution within the retina, and the nature of their synaptic connections. These properties reflect the fact that the rod and cone systems (i.e., the receptor cells and their connections within the retina) are specialized for different aspects of vision. The rod system has very low spatial resolution but is extremely sensitive to light; therefore, it specializes in sensitivity at the expense of reso-

lution. In contrast, the cone system has very high spatial resolution but is relatively insensitive to light—it specializes in visual acuity at the expense of sensitivity. The diversity of photopigments in the cone system also allows humans and many other animals to distinguish colors [14].

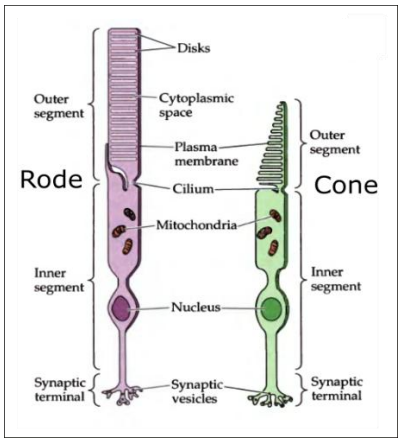

Fig. 2. Structural similarities and differences between rods and cones. The main differences are in size and shape, as well as in light sensitivity. Adapted from [14].

The ability of rods and cones to respond to different ranges of light intensities is explained by differences in their photopigments (rhodopsin in rods, and one of three photopsins in cones), which leads to differences in their transduction mechanisms. For example, rods respond reliably to even a single photon of light, whereas a comparable response in cones requires over 100 photons. However, this does not mean that cones are incapable of efficiently capturing photons. Rather, the change in current caused by the capture of a single photon in cones is relatively small and difficult to distinguish from background noise [14].

As for the topographic distribution of rods and cones on the retinal surface, they differ significantly. In the area of the retina near the optical axis, in the area of the fovea (in the area covering ~ 1^0 of the visual field, but corresponding to the most acute vision), there are no rods at all, and out of a total of 6.4 million cones, it contains ~ 17,500 cells. Thus, all 125 million rods are distributed in the retina outside the fovea (their density there significantly exceeds the density of cones) [16]. This emphasizes the specialization of the receptors: rods are responsible for peripheral vision (the entire visual field) and smooth changes in illumination on it in shades of gray (scotopic — night vision); cones specialize in color vision and are responsible for the acuity of central vision, discrimination of lines, contrasts and other small details (photopic — daytime vision) [14].

The significant difference in the topographic arrangement of the rod and cone systems determines the degree of their convergence. Each rod bipolar cell contacts a number of rods, and many rod bipolar cells contact a given amacrine cell, forming significant convergence. In contrast, the cone system is much less convergent. Thus,

each retinal ganglion cell that dominates central vision (called midget ganglion cells) receives input from only one cone bipolar cell, which, in turn, contacts a single cone. Convergence makes the rod system a superior light detector, since small signals from multiple rods combine to generate a large response in the bipolar cell. At the same time, convergence reduces the spatial resolution of the rod system. Accordingly, a one-to-one ratio between cones and bipolar and ganglion cells is precisely what is required for maximum visual acuity [14].

The outer and inner segments of rods in the mammalian retina are typically thinner than those of cones. For example, the inner segments of rods in the human peripheral retina are 2 microns in diameter, while those of cones are approximately 6 microns. However, in the fovea, where only cone photoreceptors are located, cones are thinner than the average rod, with a diameter of approximately 1.5 microns [17]. Thus, to improve visual acuity, the diameter of cones in the fovea is compressed to almost four times their average size. Assuming an eye diameter of 24 mm, we find that the angle of cone resolution at the sharpest vision is ~0.2'. Considering that the diffraction resolution of a pupil with a diameter of 3 mm is ~0.5', we find that the size of the cones determines the resolution of images by the retinal receptors to be practically equal to the theoretical (diffraction) value.

The registration process in receptors begins with the absorption of light by the visual pigment in the outer segment and its subsequent conversion into an electrical signal which is transmitted to the terminals of the inner segment of the receptors. As a result of this process, a series of spontaneous conformational changes occur, and eventually the pigment passes into a “bleached” (active, meta II) state, in which it poorly absorbs light [14]. Later in the bleached state, it recombines with opsin, thereby restoring the "dark" visual pigment. Complete regeneration and adaptation of the pigment to darkness takes 30 to 45 minutes in rods, while regeneration in cones occurs much more quickly, in approximately 1.5–3 minutes [18].

Based on the provided data, we will construct a simple statistical model of photon registration by a pigment molecule: the ideal point director model. Assuming that the observation time T_d is much shorter than the pigment regeneration time, we will assume that each pigment molecule (detector) can exist in only two states: passive, awaiting photon registration and active as a result of photon registration. Let us associate a binary random variable $s \in \{0, 1\}$ with these states ($s = 0$ corresponds to the passive state, and $s = 1$ to the active state). Clearly, this defines the simplest probabilistic scheme – a Bernoulli trial scheme. This scheme is defined by a single parameter – the probability of success $0 \leq p \leq 1$ — the probability of the event of photon detection and transition to the active state $s = 1$. The corresponding probability distribution P_s on {0,1} is as follows:

$$P_s(0) = 1 - p; \;\; P_s(1) = p \; . \tag{2}$$

From (2), it follows that the mean $\langle s \rangle = 0 * (1 - p) + 1 * p = p$, i.e., the average detector activity is equal to the probability of photon detection, and, conversely, the probability of photon detection is equal to the average of the states over an ensemble of identical detectors. The variance s also has the simple form $\langle (s - p)^2 \rangle = p(1 - p)$ and, for small p, tends to the mean $\langle s \rangle$, as in the Poisson distribution.

The above interpretation of (2) allows us to relate the parameter p to the intensity of the radiation incident on the detector. Specifically, assuming that the detector area is equal to σ_d, we consider an ensemble of N identical detectors. Let the radiation energy incident on one detector over time T_d be equal to $E_d = I\sigma_d T_d$, where I is the radiation intensity. Then, the total energy per ensemble is equal to $E = NE_d = NI\sigma_d T_d$. Dividing E by the quantum energy $h\nu$ (h is Planck's constant, ν is the characteristic frequency of the radiation), we obtain the average number of photons incident on the detector ensemble: $\langle m\rangle = NI\sigma_d T_d/h\nu$. On the other hand, the average number of events over the ensemble is equal to the sum of the averages over all detectors, i.e. $\langle n\rangle = N\langle s\rangle = Np$. Introducing the quantum efficiency $\eta = \langle n\rangle/\langle m\rangle$ as the ratio of the average number of photon registration events to the average number incident on the ensemble, we obtain $\eta = (h\nu/I\sigma_d T_d)p$ — a value independent of the ensemble size N. Considering the parameter η to be a fixed characteristic of the pigment substance, and $h\nu$ to be a characteristic of the registered radiation, we finally obtain $p = \gamma I\sigma_d T_d$, where $\gamma = \eta/h\nu$ is a coefficient determined solely by the physics of the interaction of radiation with the pigment substance. In other words, the probability p of an event registering a photon by the detector is proportional to the intensity of the incident radiation I. Assuming that the pigment regeneration time is shorter than the radiation stationarity time, and the measurement time T_d is shorter than the regeneration time, we choose the scale of time such as $T_d = 1$. With this choice, the probability p, which is also the average intensity of events registration, can be expressed as a multiple of the radiation intensity I : $p = \gamma I\sigma_d$.

Returning to the outer layer of the retina, we note that the receptors comprising it are densely packed along the retinal pigment epithelium and attached to it by their outer segments. In other words, the outer segments of the photoreceptors are aligned along the surface of the retina and taken together, form a uniform layer of pigment molecules approximately one-fortieth of a millimeter (25 μm) thick. Far from the fovea, the inner segments of the cones become thicker than the outer segments, which somewhat undermines the idea of a uniform layer of pigment molecules. However, since the inner segments scatter light into the outer segments, the efficiency of light detection is approximately the same as if a uniform layer of pigment molecules existed [19]. Furthermore, given that the packing density of pigment molecules on receptor disks is remarkably uniform (even across different vertebrate species) and is ~ 25,000 μm^{-2} (corresponding to a concentration of ~3.5 mM) [20], one can roughly estimate the total number of pigment molecules in an area, for example, the fovea (diameter ~ 0.5 mm) as $N \sim 10^{10}$.

Based on the presented facts about the distribution of pigment molecules – ideal point detectors along the retina – we can use the concept of an ideal image [I0] that we developed earlier to model the registration by detectors of the radiation falling on some sensitive surface. Thus, if we denote by $I(\vec{x})$, $\vec{x} \in \Omega$ the radiation intensity on retina surface Ω and by $X = \{\vec{x}_i\}$, $i = 1, \dots, n$ the random coordinates of the registered photons – counts, then their joint distribution according ideal image concept will be as follows [10]:

$$P(\{\vec{x}_k\}|\, I(\vec{x})) = \prod_{k=1}^{n} \rho(\vec{x}_k)\sigma_k\, P_{\langle n\rangle}(n)$$
$$P_{\langle n\rangle}(n) = \frac{\langle n\rangle^n}{n!}\exp(-\langle n\rangle)\,,\ \ \rho(\vec{x}_k) = \frac{I(\vec{x}_k)}{\int_\Omega I(\vec{x})d\vec{x}}\ , \tag{3}$$

where $\langle n\rangle = \gamma \int_\Omega I(\vec{x})d\vec{x}$ is the average number of counts across the entire retina. Note that the number of counts n in (3) is also a random variable.

From a statistical point of view, the distribution of coordinates of the registered photons of an ideal image (3) is a two-dimensional inhomogeneous point Poisson process (PPP). As was shown in [I0], this is a fairly general model for registering a certain stimulus $I(\vec{x})$, by a system of N point detectors under the local condition $\gamma I(\vec{x}_i)\sigma_i \ll 1$ or under the corresponding necessary global condition $\gamma \int I(\vec{x})d\vec{x} \ll N$. We note that the dimensionality of the space of detectors (the dimension of $\vec{x}$) was not important in the derivation of (3), just as the nature of the stimulus in the form of a two-dimensional radiation intensity $I(\vec{x})$ was not important either. The latter should be noted in connection with the fact that it is sometimes claimed that the Poisson statistic of the registered counts $X = \{\vec{x}_i\}$ is due to the Poisson statistics of the photons from the light source [21], citing the conclusions of quantum electrodynamics. This is incorrect, since Poisson statistics only hold in the special case of a coherent state of radiation, while in the case of, for example, squeezed states of radiation, this is not the case [22]. The model proposed above is based on the semiclassical theory of registration of deterministic radiation, considered as a classical electromagnetic field, but which is absorbed by pigment molecules (detectors) in the form of discrete quanta.

The model proposed above for recording incident radiation by retinal pigment molecules (3) is quite simple and, as shown in [10], convenient for theoretical analysis. However, in practice, the model parameters prove to be too large for processing and analyzing actual data in real time. Indeed, it is known that on a sunny day, the photon flux per 1 second through a surface of 1 mm^2 is ~ 10^{15} (or, correspondingly, through 1 μm^2 ~ 10^9) [19]. Considering that the packing of pigment molecules is ~ 2.5 10^4 μm^{-2} (see above), it can be concluded that a significant portion of these molecules will record incident photons. Consequently, given that the retinal fovea contains N ~10^{10} pigment molecules, the characteristic numbers of counts $\langle n\rangle$ (3) on the retina will be values of approximately the same order of magnitude.

Evolutionarily, to solve the problem of practical processing of large volumes of registered data, pigment molecules were distributed across the outer segments of the retinal receptors. Considering that the number of cones in the fovea is approximately 2*10^4, such a reduction in the size of the registration system is quite noticeable. Considering that each cone registers on average ~ 5*10^3 photons [23], we conclude that the foveal receptor system is quite capable of monitoring ~ 10^8 photons.

To statistically describe (represent) the data recorded by the receptors, there is no need to re-derive the joint distribution of the numbers of counts per receptor – we can use the results already obtained in [10]. Specifically, we will choose as a model of the receptor system a set of non-overlapping retinal regions $\{S_j\}$, $j = 1, \dots, L$, not necessarily densely covering its surface Ω. Let the values σ_j define the areas of these regions. If radiation of intensity $I(\vec{x})$ falls on the surface of the retina, we determine the

average count intensity on the receptors by $\lambda_j = \gamma \int_{S_j} I(\vec{x})d\vec{x}$. As follows from the above considerations, that values λ_j being the average value of the number of counts l_j for the j-th receptor, $\langle l_j \rangle = \lambda_j$, are simultaneously a parameters of the Poisson distributions for l_j. Since all $\{l_j\}$ are defined in disjoint regions $\{S_j\}$, they are statistically independent and have a joint probability distribution of the form:

$$P(\{l_j\} \mid I(\vec{x})) = \prod_{j=1}^{L} \frac{\lambda_j^{l_j}}{l_j!} \exp(-\lambda_j)\,. \tag{4}$$

The Poisson nature of (4) also allows us to write the variances of the number of counts l_j as $\Delta_j^2 = \langle (l_j - \lambda_j)^2 \rangle = \lambda_j$, which in our case coincides with the average $\langle l_j \rangle$.

The representation by occupation numbers $\{l_j\}$ – the representation by the numbers of counts registered by receptors, specified by the statistical description (4) – will be further treated as an internal receptor representation encoding the source $I(\vec{x})$.

We note an important property of the receptor representation. As is known for a joint Poisson distributions of the form (4), the distribution of the sum of the numbers $l = l_i + \cdots + l_j$ on any Ω subregion, that is the union of receptors $S = S_i \cup \ldots \cup S_j$, will be automatically a Poisson random variable with the parameter $\lambda = \lambda_i + \cdots + \lambda_j$ (the additivity property of the representation).

4 The Source Encoding in Retina – the Phototransduction

A photon captured by a pigment in the outer segment of a photoreceptor (activation) triggers a cascade of biochemical events that ultimately alter the graded potential of the inner segment. Change in graded potential proportionally changes the rate of release of the neurotransmitter acting on postsynaptic neurons. Photoreceptors differ from most neurons of the nervous system in that their membranes are hyperpolarized rather than depolarized when activated by light. In the dark, the receptor is in a depolarized state with a membrane (dark) potential of approximately –40 mV (somewhat lower absolute value than other neurons of central nervous system at equilibrium, approximately –60 / –80 mV). A gradual increase in light intensity leads to the potential becoming more negative, up to saturation at a level of ~ –65 mV. Thus, in the dark, when the photoreceptors are relatively depolarized, there is a certain rate of neurotransmitter release, while in the light, when the membrane is hyperpolarized, it decreases. Although this may seem illogical, it turns out that a direct proportionality between the brightness of illumination and the rate of neurotransmitter release is not necessary for the visual information formation; only an unambiguous relation between their changes is required [14]. Perhaps the purpose of maintaining the dark potential in a depolarized state is to generate higher postsynaptic responses under low light than under highlight. This may be due to the nonlinear input-output relationships of photoreceptors, which have maximum gain near the dark potential [24].

Based on the above facts and following the traditions of retinal modeling, outlined, for example, in the Standard Model of the Retina [5], we will formulate a model of

the dependence of the receptor output w – the graded membrane potential on the illuminance intensity at the receptor input $I(\vec{x})$. However, here it is necessary to clarify the problem statement.

In fact, the intensity $I(\vec{x})$ as the continuous function of $\vec{x}$ on the pigment surface $S \subset \Omega$ of the receptor is unknown. Moreover, its average value over the receptor area $I_S = \int_S I(\vec{x})d\vec{x}/\sigma$ is unknown, where σ is the area of S. Receptor registered only I_S random estimate $\hat{I}_S = l/\gamma\sigma$, where l is the number of registered counts. However, since $\langle l \rangle = \lambda = \gamma I_S \sigma$, this estimate is unbiased. Further, following the absorption of photons, a complex biochemical cascade of reactions occurs, providing a multiple amplification of the overall photon energy $lh\nu$ to the value of the dark potential shift by $|\Delta w| = Clh\nu$, where C is the gain coefficient. Thus, it has been calculated that the absorption of one photon by a rhodopsin molecule (in rods) results in the closure of approximately 2% of all receptor ion channels open in the dark [14]. Without attempting to estimate the gain C, we will take into account only the fact that the (hyperpolarized) shift is a multiple of l – the number of registered photons, with some real coefficient $A > 0$, i.e. $\Delta w = -Al$. As a result, the dependence of the receptor output w on the number of registered counts l can be represented in the following simplified (linear) form:

$$w = w_d + \Delta w = w_d - Al\,, \tag{5}$$

where $w_d \approx -40$ mV is the dark potential of the receptor. Note that, due to the randomness of l, the graded membrane potential w is also random (but ceases to be purely Poisson). However, due to the linearity of (5), the mean and variance of w can be easily found:

$$\langle w \rangle = w_d - A\gamma I_S \sigma\,,\ \langle (w - \langle w \rangle)^2 \rangle = A^2 \gamma I_S \sigma = A\langle w \rangle\,. \tag{6}$$

Relationships (6) can also be considered from a slightly different point of view, noting that the Poisson distribution of l (4) for large $\lambda \geq 10$ (in our case $\lambda \sim 10^4$, see above) is well approximated by a Gaussian continuous random variable with mean λ and variance λ [26]. Moreover, from (5) it follows that w is then also approximated by a Gaussian random variable z with parameters $\langle z \rangle = w_d - A\lambda$ and $\langle (z - \langle z \rangle)^2 \rangle = A^2\lambda$, which for $\lambda = \gamma I_S \sigma$ coincide with (6).

5 The Channel Encoding in Retina – Emphasizing the Contrast

Graduated potentials generated by receptors in the outer retinal layer are transmitted via terminal synapses of receptors in the outer plexiform layer to bipolar and horizontal cells in the inner nuclear layer. The roles of these cells vary. Bipolar cells are the connecting element in three-neuron circuits: receptor – bipolar cell – ganglion cell, representing the most direct pathways for transmitting information recorded at the retinal input to the optic nerve at its output. Horizontal cells, through their processes, mediate lateral interactions (in the outer plexiform layer) between photoreceptors and

bipolar cells, including for the purpose of ensuring the visual system's sensitivity to light intensity contrast (to fine details) over a wide range of brightness changes [14].

Let's begin with the horizontal cell model. Horizontal cells, while postsynaptic to photoreceptors, are also presynaptic to some of them, forming a feedback loop. Receiving signals from broad areas of receptors, they exert a (feedback) influence on cones located in narrower areas, thereby participating in the formation of the receptive fields of bipolar cells [25]. In short, signals from horizontal cells are not transmitted directly to the retinal output but are distributed in the outer plexiform layer, forming one of the indirect (lateral) pathways for information propagation.

The effects of presynaptic receptors on horizontal cells preserve the sign of membrane potential change (sign-preserving synapse): when receptors hyperpolarize in response to light, this also causes hyperpolarization in horizontal cells. A key aspect of horizontal cell function is that, being presynaptic to certain receptors in a feedback loop, they influence them in a synaptically inverting manner (sign inverting synapse): when horizontal cells hyperpolarize in response to light, this causes a decrease in hyperpolarization in their postsynaptic receptors. [13].

Since each horizontal cell has multiple processes connecting it to the receptor presynapses, small contributions from individual receptors can collectively cause a significant change in its membrane potential. Moreover, since adjacent horizontal cells have gap coupling with a given cell, this further expands the multitude of receptors influencing the transmembrane potential of the network of interconnected cells. Let the receptors influencing a given horizontal cell occupy retinal regions $\{S_j\}$. Then the combined hyperpolarization H of horizontal cell, consisting of graded membrane potentials of receptors (5), will have the form of the following sum (here the concept of linearity of the Standard Model of the Retina [5] is also used):

$$H = H_d + \sum_j h\Delta w_j = H_d - D\sum_j l_j\,, \tag{7}$$

where $H_d \approx$ –30 /–40 mV is an analogue of the constant dark potential, h>0 is the coefficient for recalculating changes in presynaptic membrane potentials Δw_j (5) with preservation of the sign in the contributions to the hyperpolarization of the horizontal cell, $D = hA > 0$. Note that, according to the above remark about the receptive representation (4), the sum $l_f = \sum_j l_j$ in (7) is a Poisson random variable with the parameter λ_f:

$$\begin{gathered}\lambda_f = \langle l_f\rangle = \langle\textstyle\sum_j l_j\rangle = \gamma\sum_j\int_{S_j} I(\vec{x})d\vec{x} = \gamma\int_{S_f} I(\vec{x})d\vec{x} = \gamma I_f\sigma_f\,,\\ I_f = \int_{S_f} I(\vec{x})d\vec{x}/\sigma_f\,,\end{gathered} \tag{8}$$

where $S_f = \bigcup_j S_j$ is the total retinal area controlled by the horizontal cell, σ_f is its area, I_f is the average intensity of the registered radiation at S_f, $\lambda_f = \gamma I_f\sigma_f$ is the average of the total number of counts l_f of the registered radiation at S_f.

Returning to the expression for the receptor outputs without feedback (5), we will further assume that the graded receptor potential v corrected by the horizontal cell, sent to the presynapses of bipolar cells, has, according to (7), the following form:

$$v = w + Bl_f = w_d - Al + Bl_f \tag{9}$$

where $B = gD > 0,\ g > 0$ is the conversion factor for potential changes with the opposite sign of the membrane potential $-D\sum_j l_j$ of a horizontal cell.

Let us consider the statistical characteristics of the graded potential v (9), assuming that the current receptor does not directly affect the horizontal cell: $S \not\subset S_f$. This means that l is not included in the sum $l_f = \sum_j l_j$, thus l is independent on l_f (because it is independent of all l_j). Considering the independence and Poisson property of all terms, we can write in accordance with (9):

$$\begin{gathered}\langle v\rangle = w_d - A\gamma I_S\sigma + B\gamma I_f\sigma_f\ , \\ \langle (v - \langle v\rangle)^2\rangle = A^2\gamma I_S\sigma + B^2 I_f\sigma_f\end{gathered} \tag{10}$$

Relations (10) are useful for analysis in the important special case of uniform illumination $I(\vec{x})= I$ on the surface of the receptor and in the region of the horizontal cell (when they together form the receptive field of a bipolar and then ganglion cells). In this case $I_S = I_f$ and, as follows from (10), the average shift of the potential v from w_d is $\langle \Delta v\rangle = \langle v - w_d\rangle = \gamma\big(B\sigma_f - A\sigma\big)I_f$ and is equal to zero if $B = A\,\sigma/\sigma_f$. In other words, with such a choice of the feedback coefficient, regardless of the magnitude of the intensity I_f, the operating point of the receptor will remain near the optimal value of the dark potential w_d (see above discussion).

We will assume that the receptor is optimized specifically for this case. In this case, the corrected graded potential of the receptor Δv has the form:

$$\Delta v = A(\frac{\sigma}{\sigma_f} l_f - l) \tag{11}$$

and statistical characteristics (10) of corrected graded potential Δv are simplified to:

$$\begin{gathered}\langle \Delta v\rangle = A\gamma\sigma\big(I_f - I_S\big) \\ \langle (\Delta v - \langle \Delta v\rangle)^2\rangle = \gamma A^2\sigma\left[\frac{\sigma}{\sigma_h} I_f + I_S\right] \approx A^2\gamma\sigma I_S\text{'}\end{gathered} \tag{12}$$

where the approximate expression for the dispersion corresponds to the case $\sigma \ll \sigma_f$ and coincides with the dispersion in the absence of feedback (6).

From (12) it follows that already at the early stages of visual information processing, neural signals are determined not by the absolute number of photons l captured by the receptor, but by the relative stimulation intensity – a value corrected by $l_f\,\sigma/\sigma_f$. Moreover, the correction obviously depends not only on the local intensity characteristics I_S (through $\langle l\rangle = \lambda = \gamma I_S\sigma$, but also on the intensity level of the radiation surrounding the receptor I_f (through $\langle l_f\rangle = \lambda_f = \gamma I_f\sigma_f$).

Although it may seem that the action of horizontal cells reduces the sensitivity of retinal receptors (12), this is not the case. As follows from (12), when changing the intensities I_f and I_S over a wide range, but maintaining the contrast $\Delta I = I_f - I_S$, the variability Δv (12) of v remains the same in the region close to w_d (especially in the case $I_S{\sim}I_f$). This allows us to adapt the working range of the photoreceptor (approximately 30 mV) to a wide range of illumination changes [14].

The model (11, 12) can be easily extended to bipolar cells and further to output ganglion cells. The simplest extension of the model is to the midget bipolar cells that have compact dendritic terminals connecting them to only one cone, as well as axonal terminals that always have contact to only one output midget ganglion cell. Moreover, it is the midget cells that control 80% of the retina fovea providing high visual acuity, which is the main focus of this paper. The only difference that distinguishes midget bipolar cells from the retinal neurons considered above is the nature of their response to the presynaptic potential Δv (11). Namely, midget bipolar cells are divided into two groups: ON-center cells (activated by a light spot in the center of their RF on a dark background, corresponding to $l_f\ \sigma/\sigma_f < l$) and OFF-center cells (activated by a dark spot in the center of the RF on a light background, corresponding to $l_f\ \sigma/\sigma_f > l$).

If we recall all the proposed model properties of the neurons, in particular, the fact that they allow a Gaussian approximation (see the commentary to (6)), then we immediately come to the conclusion that the presynaptic potentials that they determine for ON and OFF ganglion cells exactly correspond to the coding algorithm synthesized by us in the work [27]. Thus, it can be said that the results of the optimization of the receptor parameters (feedback coefficient B in (9)) provide an alternative way, based on the available neurobiological data, confirming the coding of data recorded by the retina, discovered by us earlier.

6 Conclusions

In this study, using generally accepted information-theoretical concepts, we were able to combine on a single platform, on the one hand, extensive neurobiological data on human brain function, and on the other, a number of algorithmic solutions from the cloud of artificial neural networks. The work is based on a number of neurobiological facts about the structure of the retina and its informational interpretation. Based on the analysis results, a consistent synthesis of algorithms for processing information propagating along the visual pathways was conducted, in accordance with the layered structure of the retina. The study concludes with confirmation of the encoding procedure for ON- and OFF-channels of the retina, thereby demonstrating the universal nature of approach proposed.

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