Invited Paper

One more reason why neurons need to be noisy

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ABSTRACT

This note discusses how information contained in a neural message is transmitted depending on two schemes of encoding: stochastic or deterministic. For the first case, it is shown that the rate of information loss is minimized for a range of signal to noise ratios entering the channel with noise and signal amplitude of the same order of magnitude. In contrast, at the deterministic limit, (i.e., signal amplitude very large compared with the noise) the rate of information loss increases; approximately by a power law of the distance traveled by the message. The exponent depends linearly on the time constant of the function relating speed of propagation vs period of reexcitation of the axon.

1- INTRODUCTION

Recent work has advocated an active role for noise in neural function\(^1\text{-}^4\). It has been shown that increasing the amplitude of the noise at a given sensory input results in an improvement of the signal to noise ratio (SNR) at some measurable output (e.g., "stochastic resonance"\(^1\text{-}^{11}\)). In that, there is an optimum non-zero noise amplitude at which sensory neurons seem to transfer more information. It is also known that, in some sensory modalities, the amplitude of the intrinsic noise (of thermal, ionic, etc. origin) is on the same order of magnitude as the fluctuations induced by typical signals acting on sensory receptors. In other words the SNR at the sensor is usually not far from one. This note addresses three aspects of the celebrated role of stochastic resonance in neural function. The first (obvious) question is why nature has not maximized, in the first place, the signal to noise ratio at the receptor level by increasing (the efficacy of the transducer and) the signal amplitude. The second aspect is concerned with how a given code (which may or may not use noise) is preserved as its propagates from the periphery to the brain. The third question is how the usual measures of SNR in the context of stochastic resonance relate with what seems to be important in neural function: information measures. Overall these questions relates with the essence of the title: it is not clear what theoretical reasons the brain (the most efficient machine) has to be as noisy as it is.

2- NEURAL ENCODING (... in one paragraph)

Signals from the outside world are transduced by a variety of sensory receptors into neuronal spikes (also called "action potentials", see cartoon in Figure 1). The interval of time between two consecutive action potentials supposedly encode the energy impinging on the receptor at a given moment (but also there are spikes originated by internal-external noise). Usually larger signals result in shorter intervals and vice versa. A given "message" encoded in this way then can be transmitted by axons (transmission lines in Fig. 1) far away for diverse purposes.
3. WHAT ABOUT TRANSMISSION OF INFORMATION?

Once a given message is written as a sequence of inter-spike intervals how is that code preserved down the transmission line? From an information theory viewpoint, axons will be "perfect channels" if the timing between spikes is maintained as spikes propagate from the receptor to the brain. That requires a constant (or any message) speed of propagation. However, it is well known that the speed at which a given action potential propagates through axons depends on the inter-spike interval. Specifically, if preceding a spike the axon was inactive for a relatively long period of time, "p", that spike will travel at full speed (as fast as 120 meters per second, for instance in "fast axons"). On the other hand, if the axon is re-excited too quickly the resulting spike will travel at a much slower speed (typically the slower speed can be 30-50% of the full speed). Figure 2 represents a (normalized to one) speed vs period of reexcitation for a typical axon. The data plotted in Fig. 2 (speed= 1-exp(p/tau) with tau=10 ms; p expressed in ms, speed normalized to the maximum velocity) is only an approximation of real biological behavior; absolute values for speeds and time constants vary among neurons by up to three orders of magnitude. For instance, realistic ranges for speeds are from 0.1 to 120 meters per second and for time constants from 5 to 300 msec.
From the relation plotted in Fig. 2 it is not too difficult to derive the sequence of intervals at discrete points down an axon from any initial (i.e., at one end) p value. It is clear that if the initial p is, let’s say, 50 msec, interval measured at any point down the axon will also be 50 msec, since the curve predict constant speed. However, for initial p’s falling in the nonlinear part of the curve, where slower speed is predicted, the interval will prolong as it propagates down the axon. In Figure 3 the graphical solution of the predicted sequence of intervals is plotted for the case of an initial p=5 msec. Point a is the initial value of p, points b to d are the first three values (at three points down the axon) predicted.
constant speed). In this case the long interval will become shorter because of the previous delay in propagating the short interval. This double effect on the intervals which can be summarized as "shorter becoming longer and longer becoming shorter" makes the density distribution of instance intervals narrower as it propagate through the axon.

The global effect of the speed vs period function can be quantified by measuring the information at discrete points of a simulated axon. The numerical representation of a axon obeying the curve in Fig. 2 is simple. Basically it implies that the hypothetical axon is divided into nodes of a given length. Using the equation of speed vs period, the delay to travel such a segment is then calculated for a given input p value. In that way an output p value is obtained which is used as input p for the subsequent node in the chain. This calculation is repeated for each interval to complete the number of nodes of the axon. Transferred information is estimated by imputing a uniform distribution of intervals at the first node (i.e., the receptor) and calculating the probability distribution at each node down the numerical axon. For the results presented here, the partition in the density distribution equals 1 msec, which may or may not be a realistic assumption for all neurons.

![Graph](image)

**Figure 4**

Figure 4 shows information (in log of bits) loss as the message progresses in the simulated axon (distance is expressed in log of number of nodes) for three time constant values of the speed vs period curve. It is seen that for all three time constants represented in the figure information is lost following a power law. The exponent ε of the power law depends almost linearly on the time

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constant tau (see Figure 5). Is nature aware of this? If so axons with smaller time constant should be connected with "hungry bits sensors", and for short axons should connect these sensors with the brain!

![Graph with labeled axes](image)

**Figure 5**

4. INFORMATION COMING OUT OF A REALISTIC (NOISY) SPIKE GENERATOR

At this point it seems obvious that information will be lost under propagation if the sensory receptor were to originate short intervals. From first principles of sensory physiology is known that one way to prevent that condition is to keep the signal amplitude small. (In short, the reason is related to the fact that neurons need more energy to "fire" a spike when re-excited to quickly). Therefore if signals are kept small, by modulating the input-output efficiency of the receptor, no short intervals will start travelling down the axon and no information will be lost. However, it is also necessary to insure that very small signals have a chance to induce a spike (i.e., there is a minimum energy to induce a spike). Here is where noise enters into the picture. In this case no single receptor will encode the signal but a parallel array of noisy receptors generating similar (but not correlated) spike trains. None of these trains will contain short intervals, since the signal is kept small.

Figure 6 shows the results of a simulation in which the "receptor" (i.e., the "spike generator") was modeled using the Hodgkin and Huxley (HH) ionic model. Noise of increasing variance (white, with zero mean) and signal (sinusoidal, 20 msec period, amplitude 3 μa) were added to the HH model. The spike intervals generated were feed to the axon modeled as in the previous section (τ = 5 msec) and information calculated at the first (H(x)) as well as in the 250th node (H(x,y)) as a function of noise variance. It can be seen that increasing noise (i.e., decreasing SNR at the input of the HH model) results in an initial increase in the transferred information through the axon. The non-monotonic behavior of this information measure is similar to the more classical SNR.
estimates in stochastic resonance\textsuperscript{1-4}. The optimum of transmitted information occurs for noise variance of about 15 $\mu$A, which corresponds to an input SNR of 0.2 (i.e., 3/15).

![Figure 6: Information (bits) vs. Noise Variance (Ua)](image)

The case of a deterministic signal is trivial. By increasing the signal amplitude a sequence of phase lockings between the signal and the neuron is obtained\textsuperscript{12-14}. The periodicity of the phase locking changes from 1:0 to 1:1 as a function of signal amplitude on a devil staircase (with fractions predicted in the Farey tree). For any amplitude (in a wide range of frequencies) the interspike intervals repeat periodically. Input information, will always be restricted to 1 bit, since intervals repeat exactly. Transferred information will depend on the frequency and amplitude of the signal. Signals of relatively (to the curve in Fig. 2) high frequency will be degraded upon transmission as was discussed in the previous section.

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6. REFERENCES
