Physics of Psychophysics: it is critical to sense

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Abstract. It has been known for about a century that psychophysical response curves (perception of a given physical stimulus vs. stimulus intensity) have a large dynamic range: many decades of stimulus intensity can be appropriately discriminated before saturation. This is in stark contrast with the response curves of sensory neurons, whose dynamic range is small, usually covering only about one decade. We claim that this paradox can be solved by means of a collective phenomenon. By coupling excitable elements with small dynamic range, the collective response function shows a much larger dynamic range, due to the amplification mediated by excitable waves. Moreover, the dynamic range is optimal at the phase transition where self-sustained activity becomes stable, providing a clear example of a biologically relevant quantity being optimized at criticality. We present a pedagogical account of these ideas, which are illustrated with a simple mean field model.

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THE DYNAMIC RANGE PROBLEM

Introduction

Physical stimuli impinge on our senses with a range of intensities that spans several orders of magnitude. How can animals cope with that scenario? In order for them to survive, their brains have to be able to distinguish among very weak input signals, as well as among very strong ones. This ability to distinguish among widely varying signals can be formalized in different ways, usually involving information theory as the main conceptual tool. Here we are going to employ an intuitive and much simpler concept to embody this ability: the dynamic range.

Consider for instance, the response curve labeled $m = 1$ in Fig. 1. The horizontal axis (in log scale) represents the stimulus intensity $r$ (for instance, the concentration of an odorant inside your nose, or the intensity of light reaching your retina), whereas the vertical axis is the response $F$ to that stimulus. Suppose $F$ represents the mean firing rate of some early sensory layers of the nervous system, which are responsible for the initial transduction from a physical stimulus to neural activity. This neural activity will presumably be “read” by other neurons in higher areas of the brain, which will further process it, and so on. What those higher areas “see” is therefore $F$, from which one could in principle infer $r$ by taking the inverse of the response function $F(r)$. Note, however, that for the $m = 1$ curve in Fig. 1 it would be difficult to perform such an inversion operation if the stimulus was very weak, say, $r \sim 10^{-5} - 10^{-4}$. The reason is that $F$ is very close to a plateau at its baseline activity $F_0 \equiv \lim_{r \to 0} F(r) (= 0$, in this example).
FIGURE 1. Linear saturating curve, corresponding to (normalized) Hill functions (Eq. 2) with exponents $m = 1$ (solid), $m = 1/2$ and $m = 1/3$ (dashed).

The same difficulty would arise for very strong stimulus (say, $r \sim 10^4 - 10^5$ in Fig. 1), in which case $F$ is very close to its saturation plateau $F_{\text{max}} = \lim_{r \to \infty} F(r)$.

To exclude these regions, the dynamic range $\Delta$ (measured in dB) is defined as $\Delta = 10 \log_{10} \left( \frac{r_{0.9}}{r_{0.1}} \right)$, where the range $[r_{0.1}, r_{0.9}]$ is obtained from the response interval $[F_{0.1}, F_{0.9}]$, as illustrated in Fig. 1. To estimate the range of stimuli that can be discriminated, one simply discards stimuli which are too faint to be detected ($r < r_{0.1}$) or too close to saturation ($r > r_{0.9}$). This is clearly an arbitrary choice, but it is usual in the biological literature and very useful as an operational definition. To account for systems which have a nonzero baseline activity $F_0$, the general definition of $F_x$ is simply

$$F_x = F_0 + x(F_{\text{max}} - F_0).$$

In the case of the $m = 1$ curve in Fig. 1, the dynamic range is about 19 dB, i.e. almost two decades. Therefore, if a system had such a response curve, it would have a hard time handling stimulus intensities varying by more than two decades (as natural stimuli do).

**Psychophysics: large dynamic range**

The fact that animals can operate with a wide dynamic range is most easily revealed in humans by classical results in Psychophysics [2]: the perception of the intensity of a given stimulus is experimentally shown to depend on the stimulus intensity $r$ as $\sim \log(r)$ (Weber-Fechner law) or $\sim r^m$ (Stevens law), where the Stevens exponent $m$ is usually $< 1$. Those empirical laws are known for about a century and have in common the fact that their dynamic range is large. You can convince yourself that small exponents lead to large dynamic ranges by looking again at Fig. 1, which shows Hill functions with different exponents $m$:

$$F(r) = \frac{F_{\text{max}}r^m}{r_0^m + r^m}. \tag{2}$$
FIGURE 2. A simplified picture of recruitment theory. Each dashed curve is a Hill function with $m = 1$ but different sensitivity $r_0$ (see Eq. [3]). The average of the five curves has a much larger dynamic range.

Notice that the Hill function can be thought of as a saturating Stevens law, both sharing the same exponent $m$ for low stimulus. It is a simple exercise to show that the dynamic range dependence on $m$ for the Hill function is 

$$\Delta = \frac{10}{m} \log_{10}(81).$$

Sensory neurons: small dynamic range

Let us focus on one particular sense, namely, olfaction. The dynamic range problem becomes evident when one looks at the experimental response curve of olfactory sensory neurons (OSNs), which are at the very first stage of signal processing and translate odorant concentration into firing rates: their dynamic range is small, typically about 1 or 2 decades only [3]! That is: on the one hand, sensory neuron responses look like the $m = 1$ curve in Fig. 1; on the other hand, psychophysical responses look like the $m < 1$ curves in Fig. 1. How can we reconcile those results? How could psychophysical laws with large dynamic range be physically implemented if individual sensory neurons have a small dynamic range? How do exponents $< 1$ arise from the dynamics of neurons?

One theory that tries to explain such a discrepancy has been proposed by Cleland and Linster [4]. Their idea lies on the presumed heterogeneity within the population of OSNs with the same type of odorant receptor. If some OSNs have more (less) receptors on their surface, they’ll be more (less) sensitive and their response curve will saturate earlier (later), like the dashed curves on the leftmost (rightmost) part of Fig. 2. As the odorant concentration gradually increases, more and more neurons would be “recruited”, so that the average response (solid curve) would have a large dynamic range even if each of the neurons had a small dynamic range. Appealing at first sight, the problem with such a “recruitment theory” is that for each order of magnitude in sensitivity, one needs an order of magnitude in receptor density. Experimentally, however, receptor over-expression is only about twofold [4], so it is plausible to assume that this is not the main mechanism responsible for the phenomenon.
A COLLECTIVE SOLUTION

In recent years we have been working on a different solution to the dynamic range problem [5, 6, 7, 8, 1]. The idea is that by coupling excitable elements with small dynamic range, one obtains an excitable medium whose response function will have a large dynamic range due to a collective phenomenon. In order to build a simple model of this mechanism, let us first study a toy model of a single excitable element.

Response function of a single excitable element

Olfactory sensory neurons behave as excitable elements. In the absence of an external stimulus, they essentially stay quiet. They will spike if odorant molecules with enough affinity bind to the receptors on their surface. After spiking, they undergo a refractory period before they can spike again. The stronger the odorant concentration, the more likely (on average) this process will repeat itself.

Consider a simple Greenberg-Hastings cellular automaton model, where each excitable element \( i = 1, \ldots, N \) has \( n \) states:

- \( s_i = 0 \) is a resting state (polarized neuron),
- \( s_i = 1 \) is an excited state (spiking neuron) and
- \( s_i = 2, \ldots, n-1 \) are refractory states (hyperpolarized neuron).

The rules are as follows:

\[
s_i(t+1) = \begin{cases} 
0 & \text{if } s_i(t) = 0 \\
1 & \text{if } s_i(t) \geq 1 \\
(\text{mod } n) & \text{otherwise}
\end{cases}
\]

After an excitation, the element goes through \( n-2 \) refractory states (blind to new stimuli) before returning to \( s_i = 0 \). We model the arrival of stimuli by a Poisson process: the probability for an element to jump from \( s_i = 0 \) to \( s_i = 1 \) is \( \lambda(r) = 1 - \exp(-r \delta t) \), where \( r \) is assumed to be proportional to the odorant concentration and the time step \( \delta t = 1 \text{ ms} \) sets the time scale of the model.

Notice that we have an ensemble of excitable elements which are not coupled to one another, so the problem can be solved exactly. If we denote by \( P_t(k) \) the probability that we find an element in state \( k \) at time \( t \), then the rules stated above immediately yield:

\[
\begin{align*}
P_{t+1}(1) &= \lambda P_t(0) \\
P_{t+1}(2) &= P_t(1) \\
& \vdots \\
P_{t+1}(n-1) &= P_t(n-2) .
\end{align*}
\]

To obtain \( P_t(0) \) we make use of the normalization condition \( \sum_{k=0}^{n-1} P_t(k) = 1 \). To obtain the response function in the stationary state, we take the limit \( t \to \infty \). Dropping the \( t \) index in the probabilities, Eqs. (3) lead to \( P(n-1) = P(n-2) = \ldots = P(1) = \lambda P(0) \). Normalization then leads to

\[
P(0) = 1 - (n-1)P(1) .
\]

Solving for \( P(1) \), we obtain the response function [5, 8]:

\[
P(1) = F(r) \delta t = \frac{\lambda(r)}{1 + (n-1)\lambda(r)} .
\]
We omit $\delta t = 1$ ms from now on. Since $\lambda (r)$ is approximately linear for small $r$, Eq. [5] is similar (but not identical) to a Hill function with $m = 1$. The reader is invited to show that the dynamic range is $\Delta = 10 \log_{10} \left\{ \ln \left[ 1 + \frac{9}{n} \right] / \ln \left[ 1 + \frac{1}{(9n)} \right] \right\}$, which is a smooth function of $n$ that quickly saturates at $10 \log_{10} (81) \simeq 19$ dB. This very simple model correctly reproduces the experimental fact that isolated OSNs have small dynamic range.

**Coupled excitable elements: mean field results**

All OSNs expressing the same receptor send their axons to the same glomerulus, where axon terminals meet the dendritic trees of about twenty mitral cells. Those dendrites are believed to be active, so each dendritic patch can be modeled as an excitable element. Moreover, it has recently been demonstrated that gap junctions (electrical synapses) exist among mitral cell dendrites [9]. What we would like to show is that the interaction among those excitable elements collectively lead to an enhancement of sensitivity and dynamic range.

Let us study a very simple model [1]. First, we assume that, owing to the gap junctions, a dendritic patch is randomly coupled to $K$ other patches, each one modeled by our simple cellular automaton and independently subjected to external stimuli with probability $\lambda (r)$. Furthermore, the coupling is such that an excitation at one site can propagate with probability $p$ to its quiescent neighbors. In a mean field description, the stationary probability that a site is in the excited state is $P(1) = F = P(0) [1 - (1 - \lambda) (1 - pF)^K]$, where the last parenthesis is the probability that no excitation comes from the $K$ neighbors (a fraction $F$ of which are likely to be active). Together with the normalization condition in Eq. [4] one arrives at the self-consistent equation for the response function

$$F(r) = (1 - (n - 1)F) \left[ 1 - \left( 1 - \frac{\lambda}{(n - 1)} \right) (1 - pF)^K \right] .$$

The solution of Eq. [6] for $K = 10$ and $n = 5$ is plotted in Fig. 3. Our control parameter is the branching ratio $\sigma \equiv pK$, which is approximately the average number of excita-
FIGURE 4. Optimal dynamic range is obtained at the critical value $\sigma_c = 1$.

tions transmitted by an excited site to its neighbors. Starting from $\sigma = 0$, we see that increasing $\sigma$ leads to amplified response of low stimuli due to propagation of excitable waves, so the dynamic range increases (Fig 4). Then, at $\sigma = \sigma_c \equiv 1$ a nonequilibrium phase transition occurs! For $\sigma > 1$, each site is transmitting more excitations than it is receiving, so it’s not surprising that even in the absence of external stimulus ($r \to 0$) the system is able to maintain a self-sustained activity (see inset of Fig. 3). If we keep increasing $\sigma$ above criticality, this self-sustained activity masks the response to weak stimuli, so the dynamic range decreases (recall the effect of $F_0$ in Eq. 1). Therefore, the maximum dynamic range is obtained precisely at criticality.

Another signature of criticality is the power law behavior of the response curve (Fig. 3, right). For $\sigma < 1$, the weak stimulus response is linear, $F \sim r$. But for $\sigma = \sigma_c$, the response is $F \sim r^{1/2}$, as can be easily verified by expanding Eq. 6 around $F = 0$. What is remarkable is that this exponent $1/2$ at criticality is very close to the measured Stevens exponents for light and odor intensity ($m = 0.5$ and 0.6, respectively [2]). We claim that Stevens law is a power law because our sensory systems should be critical. The motivation for being critical is clear: it allows the system to operate with high sensitivity and large dynamic range, both of which are desirable features for a brain living in a world “where extreme events exist, and where probabilities often have long tails” [10].

Experimentally, glomeruli have larger dynamic ranges than OSNs [11] (which was in fact what motivated the model). The hypothesis that the propagation of activity in the glomerulus is dominated by gap junctions could be tested by measuring the response curve in mice where Connexin-36 (the protein that accounts for the gap junctions) has been genetically knocked out (in fact, analogous experiments in the retina are consistent with this idea [3]). But clearly the mechanism we propose is not exclusive of electrically coupled systems, being a rather general property of excitable media.
CONCLUDING REMARKS

Those familiar with nonequilibrium phase transitions will recognize the response exponent $m$ at criticality as the critical exponent often named $1/\delta_h$ (see [12]), and $1/\delta_h = 1/2$ is just the well known mean field value. While the simple model we have presented seems well suited to describe an apparently disordered and highly interwoven structure like the olfactory glomerulus [1], one can go beyond mean field [8]. In excitable media with a different topology it is fair to expect that exponents will belong to the Directed Percolation (DP) universality class (even though this is not always the case [8]). If one looks at DP in hypercubic networks, for instance, $1/\delta_h$ is always $\leq 1/2$. In this sense, the mean field results for optimal dynamic range can be regarded as a lower bound. Networks with a different structure could easily surpass the peak at 26 dB of Fig. 4.

To summarize, we have presented a framework where psychophysical laws with large dynamic range emerge naturally from the interactions among excitable elements with small dynamic range. In particular, both the dynamic range and the sensitivity are optimized if the system is at the phase transition where self-sustained activity becomes stable. We point out that the dynamic range is an interesting observable, since it is dimensionless, easy to measure and of great biological relevance. The fact that it is maximized at a phase transition provides a clear example of optimal information processing at criticality, therefore building on a long history of efforts (both theoretical and experimental) along the same idea [13, 14, 15, 16].

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