

Multiscaled randomness: A possible source of $1/f$ noise in biology

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We evaluate the possibility that the $1/f$ fluctuations observed in many biological time series result simply from the fact that biological processes have many inputs with differing time scales. We present a stochastic model whose output is the summation of multiple random inputs (i.e., different regulatory mechanisms). We derive the conditions under which the model reproduces the complex fluctuations and $1/f$ scaling observed in biological systems. Simulations demonstrate that if model parameters are unconstrained, the likelihood of generating $1/f$ noise is quite small. Thus, while the model can be used to generate $1/f^\beta$ noise with various scaling exponents, it is unlikely that the $1/f$ behavior observed in many biological systems is due *only* to the fact that these systems are regulated by many different inputs acting on different time scales. [S1063-651X(96)04307-3]

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The fractal nature of many biological systems has recently received much attention [1]. Self-similarity has been reported in the spatial structure [2] as well as in the temporal fluctuations of many biological processes including ion channel kinetics, auditory nerve firings, lung inflation, fetal breathing, human cognition, walking, blood pressure, and heart rate [3,4]. Many of these diverse biological processes fluctuate like $1/f$ noise [see Fig. 1(a)] observed in certain physical systems [5]. With $1/f$ noise, there exists no well-defined temporal scale for the correlation time and the autocorrelation function decays as a power law. Thus, the power spectrum, the Fourier transform of the correlation function, exhibits power law behavior: $S(f) \sim 1/f^\beta$. This implies that the current value of the biological signal (e.g., heart rate) co-varies not only with its most recent value but also with its long-term history in a scale-invariant, fractal manner. This unexplained fractal feature makes the ubiquity of $1/f$ behavior in biological time series especially intriguing.

A number of different mechanisms have been proposed for the emergence of $1/f$ noise in nature, e.g., intermittency [6] and self-organized criticality [7]. Some of these mechanisms clearly underlie the $1/f$ behavior of certain physical systems. However, the origin of $1/f$ noise in many biological systems remains unknown. Given the widespread nature of $1/f$ noise in complex and diverse biological systems, some have suggested that the finding of $1/f$ behavior is only a reflection of the fact that the final output is affected by many processes that act on different time scales. Indeed, it has been shown [8,9] that certain distributions of time scales can lead to $1/f$ behavior. This suggests the possibility that the complex fluctuations and $1/f$ scaling observed in many biological systems does not reflect anything “special” about the mechanism generating these dynamics. Could this behavior merely be a result of the multiple system inputs?

Here we examine in detail one of the most simple cases of a stochastic process with multiple time scales to study the conditions under which this type of system acts as a source for $1/f$ -like noise. Specifically, when can $1/f^\beta$ noise of the type observed in biological systems be generated through the

simple superposition of random components acting on multiple time scales: multiscaled randomness?

We study a generic but biologically motivated, simple model. In many biological systems, the output is affected by semiautonomous systems operating at disparate time scales. For example, heart rate is regulated on a beat-to-beat basis by the autonomic system. Heart rate oscillations with a period of approximately 4 s reflect vagally-mediated respiratory influences while those with a period of 10 s have been attributed to baroreflex modulation of sinus node activity. In

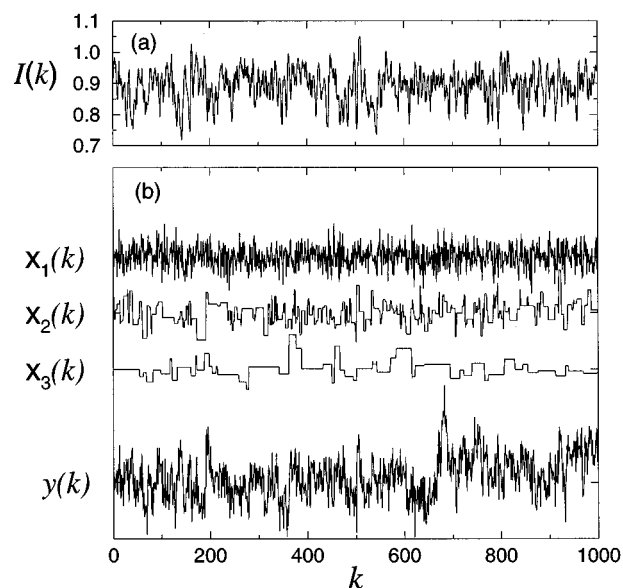


FIG. 1. (a) Heartbeat time series of a healthy adult. $I(k)$ is the time (s) between heartbeats and k is the beat number. (b) Model time series with 8 different noise components (only 3 are shown here). In this case, parameters of the inputs follow a simple scaling relation: $R_T=4$ and $R_A=1$ (see text). Heartbeat and model time series both contain fluctuations on different time scales that lead to a $1/f$ -type of noise (see Fig. 3).

addition, hormonal systems (e.g., renin-angiotensin system that regulates extracellular fluid volume) provide input with a much longer time constant and posture, activity level, meals, the sleep-wake cycle, and circadian rhythm all influence heart rate over still longer time scales. As a first approximation, one can view these inputs as distinct random processes. Each input will be associated with two parameters: 1) The first parameter describes the characteristic time scale, i.e., about how often this input changes. 2) The second parameter quantifies the magnitude of influence of each input to the whole system. For example, the effect of the sleep-wake input might take on one (approximately) constant value during the day and it will most likely change value during the night. Thus, the probability of a transition might be set to once every 1/2 day and its influence on heart rate may also have different magnitudes compared to other factors. The final output, e.g., heart rate, is the superposition of these random inputs.

The output of the model at any time step k , $y(k)$, is thus the sum of n random inputs, $x_i(k)$: $y(k) = \sum_{i=1}^n A_i x_i(k)$. Each input x_i takes on a Gaussian distributed random value (the current state of this input) and is amplified by a constant A_i that represents the relative effect of each input on the output. At each time step, the state of x_i changes with probability $1/\tau_i$, where τ_i is the time constant for input x_i [see Fig. 1(b)].

For each of the stochastic inputs, $A_i x_i$, the autocorrelation function is $C(t) = A_i^2 \exp(-t/\tau_i)$. It follows that its power spectrum is

$$S_i(f) = \text{Re} \int_0^\infty A_i^2 \exp(i2\pi ft - t/\tau_i) dt = \frac{A_i^2 \tau_i}{1 + (2\pi f \tau_i)^2}.$$

The power spectrum scaling exhibits a crossover from brown noise $S_i(f) = A_i^2 / (2\pi f)^2 \tau_i$ (for $f \gg f_i^*$), to white noise $S_i(f) = A_i^2 \tau_i$ (for $f \ll f_i^*$), where $f_i^* = (2\pi \tau_i)^{-1}$ is the crossover frequency.

For simplicity, first consider the case in which only two processes are superimposed: one with parameters τ_1, A_1 and the other with parameters τ_2, A_2 . Since there is no cross-correlation between these two processes, the overall power spectrum will be simply the sum of the individual power spectra, i.e., $S_{\text{tot}}(f) = S_1(f) + S_2(f)$. Note that $S_{\text{tot}}(f)$ can have three different regions of behavior delineated by $f_1^* = (2\pi \tau_1)^{-1}$ and $f_2^* = (2\pi \tau_2)^{-1}$ (Fig. 2). Under certain conditions (see below), in the region $f_2^* < f < f_1^*$ (region II of Fig. 2), the power spectrum can be approximated by $S_{\text{tot}}(f) \sim 1/f^\beta$, with β estimated from the two crossover points, $[f_1^*, S_1(f_1^*)]$ and $[f_2^*, S_2(f_2^*)]$. Let $\tau_2/\tau_1 = R_T > 1$ and $A_2/A_1 = R_A$. Thus

$$\beta = - \frac{\ln S_2(f_2^*) - \ln S_1(f_1^*)}{\ln f_2^* - \ln f_1^*} = \frac{\ln(R_A^2 R_T)}{\ln R_T} = 1 + 2 \log_{R_T} R_A. \quad (1)$$

A simple extension of the above discussion is a process with n time scales, with each input following the same scaling relation as defined by R_T and R_A , i.e., $\tau_2/\tau_1 = \tau_3/\tau_2 = \dots = \tau_n/\tau_{n-1} = R_T$ and $A_2/A_1 = A_3/A_2 = \dots = A_n/A_{n-1} = R_A$ [see Fig. 1(b)]. Therefore, $1/f^\beta$ scal-

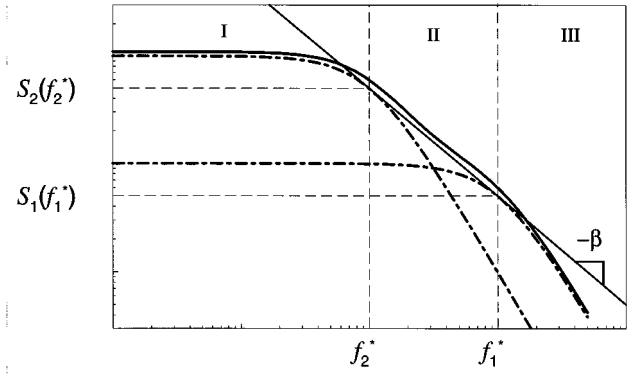


FIG. 2. Double-logarithmic plot of power spectrum density. Thick dot-dashed lines are for $S_1(f)$ and $S_2(f)$. Thick solid line represents $S_{\text{tot}}(f)$ and shows crossover from $1/f^2$ (brown) noise (region III) to $1/f^\beta$ -type of noise (region II) to white noise (region I). As shown in Eq. (1), the straight (solid) line going through the crossover points has a slope of $-\beta$.

ing can be observed over an extended region ($f_n^* < f < f_1^*$). Note, however, the model parameters do not necessarily obey the scaling relation defined by R_T and R_A . In general, model output will depend on how A_i and τ_i are chosen [10].

To make a quantitative comparison between the data shown in Fig. 1, we applied another scaling analysis to these data sets (in addition to power spectrum analysis). The method is termed detrended fluctuation analysis (DFA) [11,12]. This method is a modified random walk analysis [13] that makes use of the fact that a long-range (power-law) correlated time series can be mapped to a self-similar process by simple integration [9]. The integrated time series is self-similar if the fluctuation at different observation windows, $F(l)$, scales as a power law with the window size l . Typically, $F(l)$ will increase with window size l . A linear relationship on a double logarithmic graph indicates that $F(l) \sim l^\alpha$, where the scaling exponent is determined by calculating the slope of the line relating $\log F(l)$ to $\log l$ [14].

Results from DFA and power spectrum analyses are shown in Fig. 3. Both α and β indicate the presence of $1/f$ noise for the biological data as well as for the model simulation. Thus, a stochastic process with only 8 inputs can mimic $1/f$ behavior for about 4 decades if the amplitudes of all inputs, A_i , are equivalent and each τ_i is properly chosen.

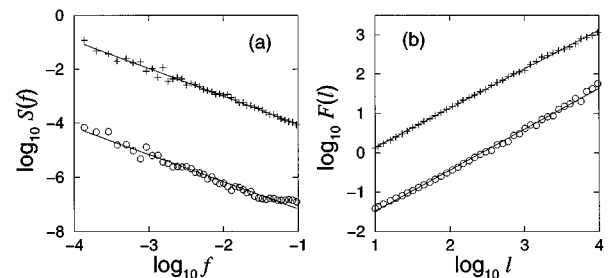


FIG. 3. Scaling analysis for heartbeat (\circ 's) and model ($+$'s) data (part of which are shown in Fig. 1) using (a) power spectrum (averaged over neighboring frequencies) and (b) DFA. Slopes of best fit lines of heartbeat and model data are -1.0 and 1.0 for power spectrum and DFA analysis, respectively.

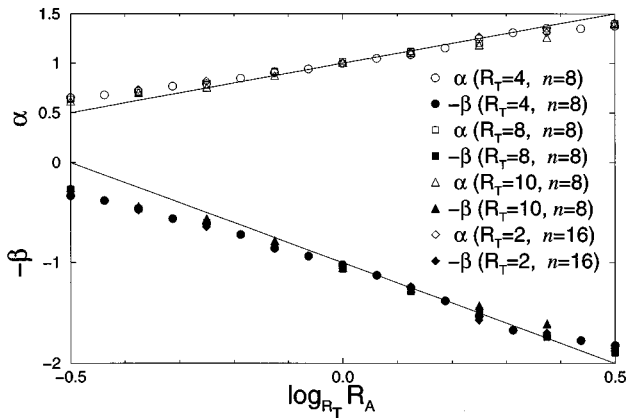


FIG. 4. Comparison of scaling exponents (α and β) measured from model simulations and theoretical values. Solid lines show theoretical values based on Eq. (1) and the relationship $\alpha = (\beta + 1)/2$ [11]. Open symbols represent estimated α by DFA and closed symbols represent estimated $-\beta$ by power spectrum analysis. Note the excellent agreement between simulation and theory at $\alpha \approx 1$ (or $\beta \approx 1$).

Figure 4 shows the theoretical and simulated values of α and β as a function of R_A , R_T , and the number of inputs n . Thus far we have demonstrated that under certain conditions the multiscaled randomness model can produce $1/f$ -type noise. Next, we study the sensitivity of the model to these constraints and the likelihood that the $1/f$ behavior of biological signals results from the summation of random processes acting on different time scales. To this end, we need a quantitative measure of how “good” the scaling is. Normally, the regression coefficient is a reasonable metric. However, we use a measure more sensitive to the stability of the scaling exponent, namely, the standard deviation (SD) of the local slopes, $\alpha_{\text{local}}(l)$, as a function of the scaling region l . If there is consistent scaling over an extended range, then $\text{SD}(\alpha) \approx 0$. In contrast, if α gradually crosses over from one value to another, then $\text{SD}(\alpha)$ will be significantly larger than 0. When we perform this calculation on the heartbeat interval time series (e.g., shown in Fig. 1), we find that for a group of 10 healthy subjects (reported in [4]), the $\text{SD}(\alpha)$ are all less than 0.15 and $0.8 \leq \alpha \leq 1.2$ in the scaling region $8 < l < 1024$ [15]. We use these quantitative measurements as a reference for evaluating the presence or absence of $1/f$ in model simulations. We first examine the case in which n time scales are chosen at random from a uniform distribution. In this situation, no assumptions are made about the time scales, i.e., every time scale is equally weighted. We check different configurations of n , amplitude (A) distribution, and time-scale distribution (all vary over a wide range). For each configuration of time scales and amplitudes, we generated 10^3 realizations with each data set containing 2^{15} points (same length as in the heart beat time series studied). The α exponent was then calculated together with the goodness of scaling behavior [$\text{SD}(\alpha)$]. We observe $1/f^\beta$ scaling in less than 2% of of these simulations [15]. As expected, increasing n decreased the likelihood of finding α different from 0.5. This indicates that random inclusion of additional time scales to the system is not sufficient to generate $1/f$. Next we study the model when we *a priori* impose struc-

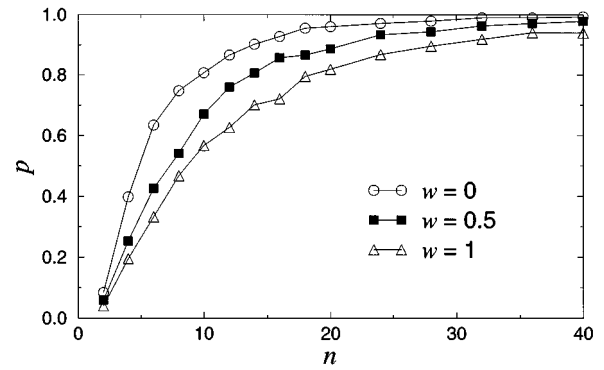


FIG. 5. Estimation of probability p of obtaining $1/f$ scaling similar to that in heartbeat data [$0.8 \leq \alpha \leq 1.2$ and $\text{SD}(\alpha) < 0.15$]. For each simulation, n time scales are selected from a uniform distribution on a logarithmic scale in the range of $1-10^4$. The parameters A_i associated with these inputs are chosen from a Gaussian distribution of mean 1 and standard deviation w . For fixed n , the upper bound of p comes from the case $w=0$. This indicates that if the amplitude of the noise inputs are allowed to vary, then the chance of obtaining $1/f$ scaling is smaller.

ture on the time scales. (This is in marked contrast to the previous situation.) Now, time scales are chosen from a uniform distribution with *logarithmic* time. At the limit $n \rightarrow \infty$, the distribution of time scales will converge to a type that can lead to $1/f$ time series as described in [8]. The question we ask here is as follows: if there are only a finite number of inputs (as is the case in many biological processes), what happens to the scaling behavior? As before, we study a variety of parameter combinations. Results are summarized in Fig. 5. Although the chance of obtaining $1/f$ scaling behavior is nonzero even for $n=2$, a large n value (approximately 4 time scales per decade [15]) is needed to ensure consistent (probability close to 1) $1/f$ scaling like that observed in the actual heartbeat data.

We have shown that there are conditions under which multiscaled randomness can produce behavior that is indistinguishable (at least in terms of self-similarity) from real world biological fluctuations. However, if all parameters of the model are free, then it is very unlikely that a system would by chance choose the “proper” parameters necessary to consistently generate $1/f$ -like noise. Nevertheless, model simulations suggest that perhaps some relatively simple processes are responsible for this puzzling behavior. Given this possibility, we believe that it is essential to perform similar types of analysis for biological data that exhibit fractal scaling behavior. In particular, for any biological time series with a $1/f^\beta$ power spectrum, one needs to examine how many reasonable time scales are involved in the specific process, the approximate values for those time scales, and the relative magnitudes of each influence.

Simulations similar to those described above should be done to examine the likelihood that observed scaling properties are due to known inputs and time scales. For example, to study the $1/f$ scaling of heartbeat time series, we assume, as a first approximation, that heart rate is predominantly regulated by 8 inputs (e.g., respiration, blood pressure) [15]. We find that the probability of obtaining $1/f$ scaling is very high if the amplitude of each noise input is identical. However,

when the amplitude of each noise input is allowed to vary, $1/f$ scaling is no longer obtained consistently. This suggests, at least for the model, that in addition to the multiple time scales in the system, the *balance* between different noise inputs is also crucial to $1/f$ scaling. These results are of interest in light of recent findings on the alteration of heart-beat scaling behavior in diseased subjects [12]. Perhaps this reflects the imbalance between different noise inputs due to the selective dropoff or domination of certain time scales under pathologic conditions. In general, Fig. 5 suggests a possible explanation for the ubiquity of $1/f$ scaling in diverse biological systems. If the time scales of the inputs affecting a biological system are “structured” and if there are a large number of inputs, then it is very likely that the output will be self-similar, even if individual input amplitudes and time scales are loosely “assigned.” If it turns out that many biological systems that exhibit $1/f$ scaling fulfill these criteria, then it would be interesting to examine the following: (a) if there is any plausible underlying mechanisms responsible

for the organization of these time scales, (b) what happens under pathologic conditions, and (c) how do biological systems with $1/f$ behavior differ from ones without this behavior? From a practical point of view, this model of multiscaled randomness provides a fast algorithm for the generation of time series that can mimic $1/f^\beta$ noise with $0 \leq \beta \leq 2$. Compared to other existing algorithms, this simple algorithm is relatively easy to understand and computationally efficient. From a biological perspective, the model can produce output strikingly reminiscent of the complex, $1/f$ fluctuations seen in biological data. Further study will determine the role of multiscaled randomness in the $1/f$ behavior of many biological processes.

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