A quantitative comparison between spike-train responses of Hodgkin-Huxley and integrate-and-fire neurons

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(October 1, 1999)

Abstract

Spike-train responses of single Hodgkin-Huxley (HH) and integrate-and-fire (IF) neurons with and without the refractory period, are calculated and compared. The HH and IF neurons are assumed to receive spike-train inputs with the constant interspike intervals (ISIs) and stochastic ISIs given by the Gamma distribution, through excitatory and inhibitory synaptic couplings: for both the couplings the HH neuron can fire while the IF neuron can only for the excitatory one. It is shown that the response to the constant-ISI inputs of the IF neuron strongly depends on the refractory period and the synaptic strength and that its response is rather different from that of the HH neuron. The variability of HH and IF neurons depends not only on the jitter of the stochastic inputs but also on their mean and the synaptic strength. Even for the excitatory inputs, the type-I IF neuron may be a good substitute of the type-II HH neuron only in the limited parameter range

PACS No. 87.19.La, 87.10.+e

Keywords Hodgkin-Huxley neuron, integrate-and-fire neuron, spike train, inhibitory rebound

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I. Introduction

A neuron generates the action potential, which propagates along the axon of a cell toward synapses exciting neurons in the next stage. There have been much theoretical studies on neurons which are responsible for encoding and decoding information carried by action potentials [1]. In the most popular neuron models like the Hopfield model [2], the output is described as a continuous variable which is slowly varying in time. The output is usually interpreted as short-time average of the rate of action potentials. This is based on the experimental observation that the mean firing rate depends on the applied stimulus to motor and sensory neurons.

On the contrary, experimental evidences showing that the detailed timing and organization of action potentials matter, have been reported in many biological systems; sonar processing of bats [3], sound localization of owls [4], electrosensation in electric fish [5], visual processing of cats [6] [7], monkeys [8] and human [9]. These suggest the importance of studying how neurons make computations based on the action potential timing with a resolution of the sub-millisecond range, receiving and emitting spike trains.

Since Hodgkin and Huxley (HH) [10] first proposed the reliable model for squid giant axon, its property has been intensively investigated [11]-[17]. The HH model has been widely adopted for an investigation of biological systems with proper modifications of conductance channels [18]. Because the HH model is described by the non-linear differential equations for four variables, its treatment is not easy, and then various types of simplified dynamical models have been proposed [19]. Among them, the simplest one is the integrate-and-fire (IF) model, which has been employed for a study on many kinds of subjects relevant to a single neuron as well as neural networks [20].

It has frequently claimed that the IF model captures the essentials of real neurons [21]-[25]. On the other hand, some studies have shown that the IF model is not realistic [26]-[28]. Actually the IF and HH models show important differences in their responses to applied, excitatory and inhibitory currents. For an excitatory (depolarizing) input dc current, $I_e$, the IF neuron, which is classified as the type I, shows the self-excited oscillation with an arbitrary low firing frequency, $f_o$ [29]. On the other hand, the HH neuron belonging to the type II has the discontinuous $f_o - I_e$ relation at the critical current, $I_c$, above which it shows the oscillation with a narrow range of $f_o$ (see Fig.1(a)). For inhibitory (hyperpolarizing) input currents, the HH neuron may bring about a firing by the so-called rebound process against our intuition [30], while the IF neuron cannot. This inhibitory rebound is realized in some non-linear dynamical neuron models like Fitzgh-Nagumo model.

Quite recently, the present author [31] has investigated the responses of the HH model to various types of spike-train inputs with constant, chaotic and stochastic interspike intervals (ISI): Ref.[31] is referred to as I in this paper. Our calculation in I shows that behavior of the variability to stochastic ISI inputs of the HH model is rather different from that obtained based on the IF model [27][32]. This is consistent with the recent calculation of Brown, Feng and Feerick, [28], who show a stronger dependence of the variability on the level of inhibitory inputs in the IF neurons than in the HH neurons.

It is the purpose of the present paper to elucidate the origin of the difference of the responses of IF and HH models to spike-train inputs, investigating whether the IF model may be an adequate substitute of the HH model. In order to clarify the point, we first
employ the spike-train inputs with simple constant ISIs, and then the stochastic ISI inputs to get some insight to the controversial variability problem initiated by Softky and Koch [33].

Our paper is organized as follows: In the next sec.II, we describe a simple neuron model adopted for our numerical calculation. In sec.III, we investigate the response of our system to inputs with the constant ISI. Stochastic inputs with the Gamma distribution are treated in sec.IV. The final section VI is devoted to conclusion and discussion. Although some of the calculated results for the HH neuron have been published in I, we include them for the completeness of the present paper.

2. Adopted model

We adopt a simple system consisting of a neuron and a synapse; the former is described by the HH or IF model and the latter by the alpha function. We will investigate the response of our neuron when spike-train inputs are applied through the synapse.

2.1 Hodgkin-Huxley model

The HH model is described by the non-linear coupled differential equations for the four variables, V for the membrane potential, and m, h and n for the gating variables of Na and K channels, and it is given by [10]

\[ \frac{\bar{C}dV}{dt} = -g_{Na}m^3 h(V - V_{Na}) - g_K n^4 (V - V_K) - g_L (V - V_L) + I, \] (1)

\[ \frac{dm}{dt} = -(a_m + b_m) m + a_m, \] (2)

\[ \frac{dh}{dt} = -(a_h + b_h) h + a_h, \] (3)

\[ \frac{dn}{dt} = -(a_n + b_n) n + a_n. \] (4)

Here the reversal potentials of Na, K channels and leakage are \( V_{Na} = 50 \) mV, \( V_K = -77 \) mV and \( V_L = -54.5 \) mV; the maximum values of corresponding conductivities are \( g_{Na} = 120 \) mS/cm\(^2\), \( g_K = 36 \) mS/cm\(^2\) and \( g_L = 0.3 \) mS/cm\(^2\); the capacity of the membrane is \( \bar{C} = 1 \mu F/cm^2 \); detailed expressions for \( a_m, b_m \) et al. are presented in Refs. [10] [31] [34].

2.2 Integrate-and-Fire model

We adopt the IF model which may include the absolute refractory period. The dynamics of the membrane potential, V, and the phenomenologically introduced variable, p, is described by

\[ C \frac{dV}{dt} = -g (1 + a)(V - V_r - pV_d) + (1 - p)I, \] (5)

\[ \frac{dp}{dt} = (-1/\tau_p) [p - \Theta(p - w)], \] (6)

with \( w = (V_t - V)/(V_t - V_r) \), \( a = \tau_t/\tau_m - 1 \) and \( \Theta(x) \) is the Heaviside step function. Here C is the capacitance and \( g (= C/\tau_m) \) the conductance of the membrane; \( \tau_m \) is the life time of membrane potential and \( \tau_t \) refractory period; \( V_t \) and \( V_r \) are the reset and threshold potentials, respectively. In the limit of \( \tau_t \to 0 \) and \( \tau_p \to 0 \), our IF model given by Eqs.(5) and (6) is equivalent to the conventional IF model [20], in which the reset condition is given by \( V(t_{om}) = V_r \) and \( V(t_{om}^+) = V_t \) at \( t_{om} \), the firing time of \( V \) \((m = 0, 1, ..)\).
The variable $p$ is zero in the active period and it is one in the refractory period. The rapid transient between the two states of $p$ is given by Eq.(6) with a small $\tau_p$. In the active period ($p = 0$), Eq.(5) becomes

$$\frac{dV}{dt} = -(1/\tau_m)(V - V_t) + I/C,$$  \hspace{1cm} (7)

which is just the same as that given in the conventional leaky IF model [20]. On the other hand, in the refractory period ($p = 1$) Eq.(5) becomes

$$\frac{dV}{dt} = -(1/\tau_r)(V - V_t - V_d),$$ \hspace{1cm} (8)

where $\tau_r = \tau_m/(1 + \alpha)$. Equation (8) shows that the reset of $V$ is accomplished by the deriving potential, $V_t + V_d$, with the time constant $\tau_r$ which is smaller than $\tau_m$.

The advantages of the present model given by Eqs.(5) and (6) are that it includes the refractory period and that it automatically resets the membrane potential. The model similar to ours was previously proposed by Horn and Opher[35]. The physical meaning of their model is, however, not transparent and their $\alpha$ term, which corresponds to our $V_d$ term in Eq.(6), improperly persists in the active period. It is crucial to reject this term in the active period (Eq.(7)) for a discussion of the input-output response of the IF neuron.

The bold curve in Fig.1 expresses the $f_o - I_i$ relation of the HH neuron, showing the discontinuous transition at $I_c = 6.3\mu A/cm^2$. It is rather difficult to choose the parameters of the IF model so as to reproduce the $f_o - I_i$ relation of the HH neuron because the IF and HH neurons belong to the different type neurons, although the exact choice of parameter values is not crucial. After several tries [36], we have determined to employ the following parameters for our numerical calculations: $V_t = -75 \text{ mV}$, $V_i = -55 \text{ mV}$, $V_d = -10 \text{ mV}$, $\tau_m = 20 \text{ msec}$ [28], $\tau_p = 0.02 \text{ msec}$ and $C = 4\mu F/cm^2$. As for the refractory period, $\tau_r$, we adopt the two choices; neurons with $\tau_r = 0.1$ and 2.0 msec are referred to as the IF0 and IF1 neurons, respectively.

After a simple calculation, we get the $f_o$ dependence on $I_i$ of IF neurons, given by

$$1/f_o = T_o = \tau_m \ln\left(\frac{I_i}{I_i - I_c}\right) + \tau_r \ln\left[\frac{V_d - (V_t - V_i)}{V_d}\right] + O(\tau_p),$$ \hspace{1cm} (9)

where the critical current, $I_c$, is given by

$$I_c = \frac{C}{\tau_m} (V_i - V_t),$$  \hspace{1cm} (10)

leading to $I_c = 4\mu A/cm^2$. Dashed and solid curves in Fig.1 express $f_o$ for IF0 and IF1 neurons, respectively.

Figure 1(b) shows the examples of the self-excited oscillations of the membrane potentials of the HH, IF0 and IF1 neurons. For IF neurons we plot $V + c p(1-p)$ instead of $V$: the $c$ term ($c = 350$) yields the spiky contribution.

2.3 Synaptic inputs

We consider the delta-function-type spike-train input expressed by

$$U_i(t) = V_a \sum_n \delta(t - t_{in}),$$ \hspace{1cm} (11)
where \( V_a \) is the magnitude of the action potential, and the firing time \( t_{in} \) for arbitrary \( n \) is assumed to be recurrently defined by

\[
t_{in+1} = t_{in} + T_{in}(t_{in}),
\]

\[
t_{i1} = 0,
\]

(12)

ISI of input spike, \( T_{in} \), being generally a function of a given time \( t_{in} \).

It has been reported that biological synapses exhibit temporal dynamics during neuronal computations [23] [37]. We, however, treat the synapse as a static unit for a simplicity of our calculation. The spike train given by Eq.(11) is injected through the synapse, yielding the postsynaptic current \( I_i \) given by

\[
I_i(t) = A_{syn} \sum_n \alpha(t - t_{in}),
\]

(14)

where \( A_{syn} = g_{syn} (V_a - V_{syn}) \), \( g_{syn} \) and \( V_{syn} \) are the synaptic conductance and reversal potential, respectively, and the alpha function, \( \alpha(t) \), is defined by [31] [34]

\[
\alpha(t) = (t/\tau_s) e^{-t/\tau_s} \Theta(t),
\]

(15)

\( \tau_s \) being the time constant relevant to the synapse conduction. The positive and negative \( A_{syn} \) stand for the excitatory and inhibitory couplings, respectively. When \( T_{in} \gg \tau_s \), Eqs.(14) and (15) yield pulse currents with the maximum value of \( I_{i \text{max}} \) at \( t = T_{in} + \tau_s \) and with the half-width of 2.45 \( \tau_s \). On the contrary, when \( T_{in} \lesssim \tau_s \) the temporal summation of input currents is realized because an input pulse comes before the current induced by its preceding pulse is not attenuated. We assume \( \tau_s = 2 \) msec and treat \( A_{syn} \) as an adjustable parameter.

When the membrane potential \( V \) oscillates, it yields the spike-train output, which may be expressed by

\[
U_o(t) = V_a \sum_m \delta(t - t_{om}),
\]

(16)

in a way similar to Eq.(11), where \( t_{om} \) is defined as the time when the membrane potential \( V(t) \) crosses \( V_z = 0 \) mV from below. The output ISI is defined by

\[
T_{om} = t_{om+1} - t_{om}.
\]

(17)

Differential equations given by Eqs.(1)-(4) for the HH neuron (or Eqs.(5) and (6) for the IF neuron) including the external current given by Eqs.(11)-(15) are solved by the forth-order Runge-Kutta method with the integration time step of 0.01 msec. The calculations are performed for 2 sec in the constant-ISI case and for 20 sec in the stochastic ISI case. If ISI of spike-train input or output is about 10 msec in the latter case, the size of its sample is about 2000. Although this figure is not sufficiently large for statistics of ISI data, we hope an essential ingredient will be clarified in our numerical investigation.

### 3. Constant-ISI inputs
### 3.1 HH neurons

Let us first consider the HH neuron which receives spike-train inputs given by Eqs. (11)-(15) with the positive $A_{syn}$ and constant $T_{in}$. The solid curve in Fig. 2(c) expresses the time course of the membrane potential, $V$, for the excitatory postsynaptic current, $I_i$, with $A_{syn} = 40$ shown by the solid curve in Fig. 2(b), which is induced by an applied spike-train input, $U_i$, with $T_{in} = 20$ msec as depicted in Fig. 2(a) (for time courses of potentials for inputs with $T_{in} = 10$ msec, see Fig. 5 of I). For this spike-train input of $T_{in} = 20$ msec, we get a regular spike-train output with $T_{om} = 20$ msec. This is in contrast with the case of spike-train input of $T_{in} = 10$ msec, in which output spike-train is phase locked with the ratio of $4:3$, oscillating with a long cycle of 40.00 msec ($=11.25 + 12.36 + 16.39 = 4T_{in}$), where 11.25, 12.36 and 16.39 are the values of output ISIs.

Solid and dashed curves in Fig. 3(a) show the average ($\mu_o$) and root-mean-square (RMS, $\sigma_o$) values of $\{T_{om}\}$ for input ISI of $T_{in} = 10$ msec when $A_{syn}$ is changed. Filled circles denote the distribution of $\{T_{om}\}$ for a given $A_{syn}$. For example, in the case of $A_{syn} = 40$, we get $T_{om} = 11.25, 12.36$ and 16.39 msec as mentioned above. For $A_{syn} > 56$, ISI of the output is the same as that of input, then the coefficient defined by $k = \mu_o/\mu_i$ is unity. When $A_{syn}$ is decreased, we get $k$ larger than unity, and $k = 2$ for $8 < A_{syn} < 28$. Below $A_{syn} = 8$, we have no output spikes.

Solid and dashed curves in Fig. 3(b) express $\mu_o$ and $\sigma_o$ when the average of input ISI ($\mu_i$) is changed with $A_{syn} = 40$. We obtain $k = 1$ for $\mu_i \approx 12$ msec, $k = 2$ for $6 \approx \mu_i \approx 8$ msec, and $k = 3$ for $\mu_i = 4$ msec: otherwise $k$ is non-integer. We obtain no output ISIs of $T_{om} < 10$ msec for $T_{in} < 12$ msec, which is due to the low-pass filter character of the HH neuron [31].

Next we discuss the inhibitory input case. The dash curve in Fig. 2(c) expresses the time course of membrane potential with $T_{om} = 20$ msec when we apply the inhibitory postsynaptic current with $A_{syn} = -40$ and $T_{in} = 20$ msec, which works to hyperpolarize the membrane potential, $V$. When the postsynaptic current given by the alpha function (Eq.15) decreases and vanishes, $V$ changes to restore to the rest level and it crosses the threshold to yield an action potential with a delay of about 15 msec. Thus the HH neuron can fire even for the inhibitory input by the rebound process [30]. This process, however, requires an appreciable periods: when the input ISI is less than about 15 msec, the HH neuron cannot fire because the next inhibitory spike is applied before the hyperpolarized membrane potential crosses the threshold level.

By changing $A_{syn}$ and $\mu_i$, we perform similar calculations, whose result is summarized in Fig. 4(a). It expresses the phase diagram showing the region where the integer $k$ is obtained in the $\mu_i - A_{syn}$ space. Note that between the integer $k$’s, we have non-integer solutions; for example, in the case of $\mu_i = 10$ msec and $A_{syn} = 40$, we get $k = 1.33$ between $k = 1$ and 2. We notice for positive $A_{syn}$ that there is the wide $k = 1$ region and that the regions of larger $k$ appear at the left side of the $k = 1$ region. For negative $A_{syn}$, the HH neuron fires for input with $\mu > 15$ msec, for which we get the states of $k = 1$ and $k = 2$.

### 3.2 IF neurons

Now we consider the IF neuron to which the constant-ISI input is applied. The solid (dashed) curve in Fig. 2(d) shows the time course of the membrane potentials of the IF1 neuron for excitatory (inhibitory) inputs. When the excitatory input with $T_{in} = 20$ msec
is applied, its membrane potential is depolarized to cross the threshold, and it emits the spike-train output with $T_{om} = 20$ msec. When the inhibitory input is applied, its membrane potential is hyperpolarized. After postsynaptic current vanishes, $V$ changes to restore to the rest level but cannot cross the threshold level. The behavior of $V$ of the IF1 neuron is in strong contrast with that of the HH neuron shown by the dashed curve in Fig.2(c). Thus the IF1 (and IF0) neuron is not excitable for inhibitory spike-train inputs.

Figure 5 shows the time courses of $U_i(t)$ and $V(t)$ of the IF1 neuron for $T_{in} = 10$ msec with $A_{syn} = 40, 64$ and $120$. For $A_{syn} = 64$, IF1 neurons regularly emit spike trains of $T_{om} = 10$ msec with a delay of about $3$ msec. For $A_{syn} = 40$, the output ISI becomes larger than $10$ msec, because it can emit output pulse after integrating small input signals by virtue of the type-I neuron although a single input pulse is insufficient to trigger the output pulse. On the contrary, for a large $A_{syn} = 120$, a single input pulse make the IF1 to emit irregularly multiple pulses with $T_{om}$ smaller than $T_{in}$. The time course of the membrane potential of the IF0 neuron is ostensibly similar to that of the IF1 neuron (not shown).

The $A_{syn}$ dependence of the responses of the IF0 and IF1 neurons to inputs with $\mu_i = 10$ msec is shown in Fig.6(a) and 7(a), respectively. Solid (dashed) curves express $\mu_o$ ($\sigma_o$) and filled circles denote $\{T_{om}\}$ for a given $A_{syn}$. For the IF0 neuron, we get the integer values of $k = 1$ for $51 \lesssim A_{syn} \lesssim 58$, $k = 2$ for $A_{syn} = 32$, $k = 3$ for $A_{syn} = 26$ and $k = 4$ for $A_{syn} = 22$. On the contrary, for the IF1 neuron we get the integer values of $k = 1$ for $54 \lesssim A_{syn} \lesssim 83$, $k = 2$ for $32 \lesssim A_{syn} \lesssim 37$, $k = 3$ for $A_{syn} = 26$ and $k = 4$ for $A_{syn} = 22$. When we compare these results with that of HH neurons (Figs. 3(a) and (b)), we notice that $k \lesssim 3$ is realized for small $A_{syn}$ and that $k < 1$ exists at large $A_{syn}$.

Figures 6(b) and 7(b) show the $\mu_i$ dependence of $\mu_o$ and $\sigma_o$ of the IF0 and IF1 neurons, respectively, with $A_{syn} = 64$. We get $\mu_o = \mu_i$ ($k = 1$) for $\mu_i > 15$ ($\mu_i > 6$) msec in the IF0 (IF1) neuron. When $A_{syn}$ is reduced, we obtain the states with larger $k$. For example, for $A_{syn} = 40$, we get $k = 2$ with $16 < \mu_i < 22$ ($12 < \mu_i < 22$) msec in the IF0 (IF1) neuron; the $k = 1$ state is not available (not shown).

From calculations by changing $A_{syn}$ and $\mu_i$, we obtain the phase diagrams of the integer $k$ values in the $\mu_i-A_{syn}$ space for the IF0 and IF1 neuron, which are shown in Figs.4(b) and (c), respectively. Because IF0 and IF1 neurons cannot fire for inhibitory inputs, results only for the positive $A_{syn}$ are shown. A comparison of them with the corresponding phase diagram of the HH neuron (Fig.4(a)) shows

(1) the $k = 1$ region, particularly of the IF0 neuron, is greatly reduced,
(2) although the $k \geq 2$ region in the HH neuron appears at the left side of the $k = 1$ phase, such phases in IF0 and IF1 neurons appear below the $k = 1$ phase,
(3) the phase with $k < 1$ appears for IF0 and IF1 neurons although it does not exist for the HH neuron in the parameter range shown in the Fig.4(a), and
(4) an inclusion of the refractory period in the IF1 neuron widens the $k = 1$ region.

4. **Stochastic-ISI inputs**

The ISIs of spike-train input, $T_{in}$, in Eq.(12) are assumed to be independent random
variables with the Gamma probability density function given by

\[ P(T) = s^r T^{r-1} e^{-sT} / \Gamma(r) \]  

(18)

for which we get \( \mu_i = r/s \), and \( \sigma_i = \sqrt{r}/s \), \( \Gamma(r) \) being the gamma function. For a later purpose, we define the dimensionless variability given by

\[ c_{v\lambda} = \sigma_{\lambda}/\mu_{\lambda} \quad (\lambda = i \text{ and } o) \]  

(19)

for input (\( \lambda = i \)) and output ISIs (\( \lambda = o \)), from which we get \( c_{vi} = 1/\sqrt{r} \) for the Gamma-distribution inputs. For \( r = 1 \) in Eq.(18) we recover the exponential distribution \( (c_{vi} = 1) \) and a Poisson distribution for the number of spikes in a given time interval. In the limits of \( r \rightarrow \infty \) and \( s \rightarrow \infty \) with keeping \( \mu_i = r/s \) fixed, Eq.(18) reduces to \( P(T) = \delta(T - \mu_i) \), the constant ISI with \( \mu_i = T \) and \( c_{vi} = 0 \).

The spike-train input created by the Gamma-distribution generator is applied to our neural system. Calculations are performed by changing \( A_{syn} \) or \( \mu_i \) by keeping the value of \( c_{vi} \) fixed. Note that because the number of our sample of input ISI is not sufficiently large, the obtained \( c_{vi} \) fluctuates around the intended values.

4.1 HH neurons

The histogram in Fig.8(a) shows the distribution of excitatory input ISIs \( (A_{syn} = 40) \) with \( c_{vi} = 0.40 \) and \( \mu_i = 10 \) msec, which leads to output ISIs with the distribution shown in Fig.8(b) \( (c_{vo} = 0.25 \) and \( \mu_o = 14.84 \) msec). Output histogram in Fig.8(b) has no distributions at \( T_{om} < 10 \) msec, which arises from the low-pass filter character of the HH neuron as shown in Fig.3(b).

Figures 9(a) and (b) show the results of \( \mu_o, \sigma_o \) and \( c_{vo} \) as a function of \( A_{syn} \) for inputs with \( c_{vi} = 0.4 \) and \( 1.0 \), respectively \( (\mu_i = 10 \) msec). Comparing them with the results for the constant ISI \( (c_{vi} = 0) \) shown in Fig.3(a), we note that for larger \( c_{vi} \), values of \( \mu_o > 20 \) msec are much accumulated in the region of smaller \( A_{syn} \) region. This is due to the integration character of neurons for weak inputs and it is also realized in the IF neuron (Figs.10(a) and 11(a)).

The dependence of output ISIs on \( \mu_i \) for \( c_{vi} = 0.4 \) and \( 1.0 \) are shown in Fig.9(c) and (d), respectively. We note that the structure at \( \mu_i < 10 \) seen in the case of \( c_{vo} = 0 \) (Fig.3(b)), disappears because of the randomness in inputs. Although \( \mu_o \sim \mu_i \) for large \( \mu_i \), we get \( \mu_o > 10 \) msec for small \( \mu_i \), since the HH neuron plays as the low-pass filter. For \( c_{vi} = 1.0 \), we get \( \mu_o \sim (\mu_i + 10) \) whereas \( \sigma_o \sim \mu_i \), which yield an increase in \( c_{vo} \) as increasing \( \mu_i \).

Figures 9(e) and (f) express the \( \mu_i \) dependence of output ISIs for inhibitory inputs with \( c_{vi} = 0.4 \) and \( 1.0 \), respectively. Their comparisons with the result for \( c_{vi} = 0.0 \) (Fig.3(c)) show that the HH neuron may fire for stochastic inputs with \( \mu_i < 15 \) msec. It is interesting to note from a comparison of Fig.9(c) (Fig.9(d)) with Fig.9(e) (Fig.9(f)) that the \( \mu_i \) dependence for inhibitory inputs is quantitatively similar to that for excitatory inputs.

4.2 IF neurons

Histograms in Figs.8(c) and (d) show the distributions of output ISIs of the IF0 neuron and those of the IF1 neuron, respectively, for excitatory inputs of \( c_{vi} = 0.4 \) and \( \mu_i = 10 \)
msec whose distribution is plotted in Fig.8(a). Solid and dashed histograms in Fig.8(c) and (d) express the results of $A_{\text{syn}} = 40$ and 64, respectively. The input histogram has a peak at about $T_{\text{in}} = 10$ msec, as expected. On the contrary, output ISIs of IF0 and IF1 neurons for $A_{\text{syn}} = 64$ have peaks at lower values of ISI. This is understood from Figs.6(a) and 7(a) which show that $k < 1$ for $\mu_i < 16$ (12) msec in the IF0 (IF1) neuron. Furthermore, for the IF1 neuron, the refractory period makes the neuron to emit no outputs for $T_{\text{m}} < 4$ msec.

Figures 10(a) and (b) (11(a) and (b)) show output results for excitatory inputs with $c_{\text{vi}} = 0.4$ and 1.0, respectively, of the IF0 (IF1) neuron as a function of $A_{\text{syn}}$. As $c_{\text{vi}}$ is increased, large $\mu_o$ values are accumulated in the region with a small $A_{\text{syn}}$. Although this phenomenon is realized also in the HH neuron (Fig.9(a) and (b)), the $\mu_o$ value is 100 msec at most in the HH neuron while the maximum $\mu_o$ value exceeds 1000 msec in the IF neurons. The difference in $\mu_o$ and $\sigma_o$ between the IF0 and IF1 neurons become smaller for larger $c_{\text{vi}}$, although $c_{\text{vo}}$ of the IF0 neuron is always larger than that of the IF1 neuron.

The $\mu_i$ dependence of the output ISIs for excitatory inputs with $c_{\text{vi}} = 0.4$ and 1.0 of the IF0 (IF1) neuron are shown in Fig.10(b) and (d) (11(b) and (d)), respectively. With increasing $\mu_i$, both $\mu_i$ and $\sigma_i$ increase, but $c_{\text{vo}}$ tends to saturate. For larger $c_i$, we get larger $c_o$, as expected. It is interesting to compare the results of the IF neurons with those of the HH neuron. For inputs with $c_i = 0.4$ and $\mu_i = 10 - 30$ msec, we obtain $c_{\text{vo}} = 0.25 - 0.40, 0.47-0.77$ and 0.40-0.45 for the HH, IF0 and IF1 neurons, respectively. Similarly, for inputs with $c_i = 1.0$ and $\mu_i = 10 - 30$ msec, we get $c_{\text{vo}} = 0.56 - 0.86, 1.01-1.13$ and 0.85-0.96 for the HH, IF0 and IF1 neurons, respectively. We note that the relation:

$$c_{\text{vo}}^{HH} < c_{\text{vo}}^{IH1} < c_{\text{vo}}^{IF0} \sim c_{\text{vi}},$$

holds in our calculation, related discussion being given in the next section.

5. Conclusion and discussion

Since Softky and Koch \[33\] reported a large $c_{\text{vo}} (\sim 0.5 - 1.0)$ in cortical neurons in visual V1 and MT of monkeys, it has been controversial how to understand the large variability of neurons to stochastic inputs \[33\]-\[38\]-\[43\]. There have been much discussions on this subject using the IF model. Some theoretical studies show that IF models lead to small $c_{\text{vo}}$ because an integration of a large number of random inputs works to reduce the variability \[23\]-\[28\]. On the other hand, other studies have shown that IF neurons may yield an appreciable value of $c_{\text{vo}}$ \[24\]-\[28\].

Figure 12 shows the $c_{\text{vi}} - c_{\text{vo}}$ plot of ISI data having reported for HH (Figs.3(b), 9(c) and 9(d)), IF0 (Figs.6(b), 10(c) and 10(d)) and IF1 neurons (Figs.7(b), 11(c) and 11(d)) and of new results calculated for $c_{\text{vi}} = 0.7$. Open squares, filled triangles and circles denote the results of the HH, IF0 and IF1 neurons, respectively. We note the variability of output ISIs may be large and nearly the same as that of input ISIs in HH and IF neurons. Scattered values of $c_{\text{vo}}$ for a given $c_{\text{vi}}$ mean that $c_{\text{vo}}$ depends not only on $c_{\text{vi}}$ but also strongly on $\mu_i$ (and $A_{\text{syn}}$). For example, when the $\mu_i$ is varied from 2 to 30 msec in the IF0 neuron, we get $c_{\text{vo}} = 0.20 - 0.48$ for $c_{\text{vi}} = 0.4, c_{\text{vo}} = 0.40 - 0.84$ for $c_{\text{vi}} = 0.7$ and $c_{\text{vo}} = 0.64 - 1.18$ for $c_{\text{vi}} = 1.0$. Our calculation reconciles the dispute among the earlier
calculations yielding small \([23, 28]\) and large \(c_{vo}\) \([27, 28, 41]\) based on the IF model; the difference in the calculated \(c_{vo}\) may be due to the difference in the parameters adopted in their calculations.

It should be noted in Figs.9, 10 and 11 that the variability of output ISIs generally increases with increasing input ISIs, which is in agreement with the biological data (Fig.3 of Ref.\[42\]). When the input ISI is small compared to the characteristic integration time, the neuron acts as an integrator yielding a small \(c_{vo}\), while when the reserve is true, the neuron play a role of the coincidence detector with a fairly large \(c_{vo}\).

To summarize, we have performed numerical calculations of the responses of the HH and IF neurons to inputs with the constant and stochastic ISIs, to make a comparison between them, after we had chosen the parameters such as for the IF model to mimic the time dependence of the membrane potentials of the HH neuron (Fig.1(a)). Our calculations have shown the followings:

(i) the HH neuron can fire for both excitatory and inhibitory inputs whereas the IF neuron only for excitatory inputs,

(ii) responses of the HH and IF neurons show the complicated behavior to spike-train inputs even with the simple constant ISIs, for which \(\mu_o\) (or \(k = \mu_o/\mu_i\)) is generally functions of \(\mu_i\), \(c_{vi}\) and \(A_{syn}\),

(iii) the response to constant ISIs of the IF neurons is rather different from that of the HH neuron: the \(k = 1\) region of the IF neuron, in which neurons properly respond to inputs, is much narrower than that of the HH neuron (Fig.4),

(iv) the variability of IF and HH neurons to stochastic ISIs shown in Fig.12, may be large and follow the relation given by Eq.(20),

(v) the \(A_{syn}\) dependence of the variability of the IF neuron is stronger than that of the HH neuron, and

(vi) an inclusion of the refractory period in IF1 depresses the \(c_{vo}\) values and improves to some extent its response, widening the \(k = 1\) region.

The difference in the item (i) arises from the fact that the HH model has the rebound process against the hyperpolarized membrane potential while the IF model does not. In order to make the item (ii) more concrete, we show in Figs.13(a) and (b), the \(f_i - f_o\) plots of the HH and IF0 neurons, respectively, where \(f_\lambda = (1/\mu_\lambda)\) is the mean frequency of input \((\lambda = i)\) and output ISIs \((\lambda = o)\). Saturating functions with the maximum frequency of \(f_{o\max} \sim 100\) Hz given by the \(f_i - f_o\) plots of the HH neuron (Fig.13(a)), are similar to the sigmoidal function of \(g(x) = (1 + \tanh x)/2\) adopted in the formal rate-coding models \[2\]. However, monotone increasing functions given by those of the IF0 neuron (Fig.13(b)) are quite different from \(g(x)\). The \(f_i - f_o\) plots of the IF1 neuron express also increasing functions but saturate with the large maximum frequency of \(f_{o\max} \sim 500\) Hz (not shown). The main origins yielding the differences cited in the items (iii)-(vi) are (1) the difference of the \(f_o - I_i\) relation of the type-I IF neuron from that of the type-II HH neuron and (2) the difference in the refractory period. The continuous \(f_o - I_i\) relation in the IF neuron yields a large \(k\) \((\geq 3)\) after integrating small inputs for a small \(A_{syn}\). The IF models with the vanishing or small refractory periods emits the output ISI with \(k < 1\). Then the \(k = 1\) region in the phase diagram of the IF neuron is much reduced compared with that of the HH neuron (Fig.4). The item (iv) is consistent with earlier calculations using the IF \[27, 28, 41\] and HH models \[28\]. Brown, Feng and Feedick \[28\] show that the variability
of the IF neuron has a stronger dependence on the number of synaptic inputs, \( N_s \), than that of the HH neuron. Since their \( N_s \) is expected to correspond to our \( A_{\text{syn}} \) in a crude sense, the item (v) is consistent with their result. They also claim that an inclusion of the absolute refractory period in the IF neuron decreases the variability and that it increases the disparity between the results for the IF and HH neurons. The former agrees with our item (vi) but the latter does not. Gutkin and Ermentrout predict based on the Morris-Lecar (ML) model that the variability of type-I ML neuron is larger than that of type-II ML one, which is supported by our calculations (Eq.20)). However, their claim that the type-II neuron yield only a small \( c_{\text{vo}} \), cannot be applied to the HH model.

The HH neuron responds to static and spike inputs differently from the IF neuron. The HH neuron shows the complex behavior not shared with an IF neuron. The chaotic oscillation induced by an applied sinusoidal currents and the firing by the inhibitory rebound are never realized in the single IF neuron. Our calculations have shown that even for the excitatory inputs the IF model may be a good substitute of the HH model only within the limited parameters. There are many experimental and theoretical evidences showing that reciprocally inhibitory neurons play important roles in real systems such as Hippocampus and thalamus, for which the IF model cannot be used. We should mind advantage and disadvantage of the IF neuron in modeling biological neural systems.

Acknowledgment

This work is partly supported by a Grant-in-Aid for Scientific Research from the Japanese Ministry of Education, Science and Culture.
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Figure Captions

Fig.1 (a) The $f_o - I_i$ plot of the HH (bold solid curve), IF0 (dashed curve) and IF1 neurons (solid curve), and (b) the self-excited oscillations of the HH, IF0 and IF1 neurons, scales for IF0 and IF1 neurons being shifted by 200 and 400 mV, respectively.

Fig.2 The time courses of (a) the input spike train, $U_i$, with $T_{in} = 20$ msec, (b) the postsynaptic currents, $I_i$, and (c) the membrane potential, $V$, of the HH ($A_{syn} = \pm 40 \mu A/cm^2$) and (d) $V$ of IF1 neurons ($A_{syn} = \pm 64 \mu A/cm^2$); solid and dashed curves denote excitatory and inhibitory cases, respectively, and scales for $U_i$ and $I_i$ are arbitrary. (see text).

Fig.3 Responses of the HH neuron to constant ISIs. (a) mean ($\mu_o$, solid curve), RMS ($\sigma_o$, dashed curve) and the distribution (filled circles) of output ISIs as a function of $A_{syn}$ for $\mu_i = 10$ msec (the arrow denotes the input ISI); (b) those as a function of $\mu_i$ with $A_{syn} = 40 \mu A/cm^2$ and (c) with $A_{syn} = -40 \mu A/cm^2$ (dotted lines express $k = 1, 2$ and 3).

Fig.4 The phase diagrams of the (a) HH, (b) IF0 and (c) IF1 neurons in the $\mu_i - A_{syn}$ space, showing the states with integer $k (= \mu_o/\mu_i)$ for constant-ISI inputs given by Eqs.(14) and (15), crosses denoting no outputs. The results of IF0 and IF1 are shown only for positive $A_{syn}$ because they cannot fire for inhibitory inputs.

Fig.5 The time courses of (a) constant-ISI input and (b) the membrane potential of IF1 neuron with $A_{syn} = 40$, (c) $A_{syn} = 64$ and (d) $A_{syn} = 120 \mu A/cm^2$. The scale of (a) is arbitrary and those of (c) and (d) are shifted by 200 and 400 mV, respectively.

Fig.6 Responses of the IF0 neuron to constant ISIs. (a) mean ($\mu_o$, solid curve), RMS ($\sigma_o$, dashed curve) and the distribution (filled circles) of output ISIs as a function of $A_{syn}$ for $\mu_i=10$ msec (the arrow denotes the input ISI); (b) those as a function of $\mu_i$ with $A_{syn} = 40 \mu A/cm^2$ and (c) with $A_{syn} = -40 \mu A/cm^2$ (dotted lines express $k = \mu_o/\mu_i = 1$).

Fig.7 Responses of the IF1 neuron to constant ISIs, same as in Fig.6.

Fig.8 Histograms of (a) the stochastic input ( $\mu_i = 10$ msec, $c_{vi} = 0.4$), (b) output ISIs of the HH neuron, (c) of the IF0 neuron and (d) of the IF1 neuron, solid (dashed) histograms being for $A_{syn} = 40$ (64) $\mu A/cm^2$.

Fig.9 Responses of the HH neuron to stochastic ISIs; (a) $\mu_o$ (solid curve), $\sigma_o$ (dashed curve) and $c_{vo}$ (thin solid curve) as a function of $A_{syn}$ for inputs of $\mu_i = 10$ msec with $c_{vi} = 0.4$ and (b) with $c_{vi} = 1.0$ (the arrow denotes $\mu_i$); (c) those as a function of $\mu_i$ with $A_{syn} = 40 \mu A/cm^2$ for inputs of $c_{vi} = 0.4$ and (d) of $c_{vi} = 1.0$; (e) those as a function of $\mu_i$ with $A_{syn} = -40 \mu A/cm^2$ for inputs of $c_{vi} = 0.4$ and (f) of $c_{vi} = 1.0$.

Fig.10 Responses of the IF0 neuron to stochastic ISIs; (a) $\mu_o$ (solid curve), $\sigma_o$ (dashed curve) and $c_{vo}$ (thin solid curve) as a function of $A_{syn}$ for inputs of $\mu_i = 10$ msec with $c_{vi} = 0.4$ and (b) with $c_{vi} = 1.0$ (the arrow denotes $\mu_i$); (c) those as a function of $\mu_i$ with $A_{syn} = 40 \mu A/cm^2$ for inputs of $c_{vi} = 0.4$ and (d) of $c_{vi} = 1.0$; (e) those as a function of $\mu_i$ with $A_{syn} = -40 \mu A/cm^2$ for inputs of $c_{vi} = 0.4$ and (f) of $c_{vi} = 1.0$. (see text).
curve) and $c_{vo}$ (thin solid curve) as a function of $A_{syn}$ for inputs of $\mu_i = 10$ msec with $c_{vi} = 0.4$ and (b) with $c_{vi} = 1.0$ (the arrow denoting $\mu_i$); (c) those as a function of $\mu_i$ with $A_{syn} = 64 \mu A/cm^2$ for inputs of $c_{vi} = 0.4$ and of $c_{vi} = 1.0$.

Fig.11 Responses of the IF0 neuron to stochastic ISIs, same as Fig.10.

Fig.12 $c_{vo}$ against $c_{vi}$ of HH (open squares), IF0 (triangles) and IF1 neurons (circles) (see text).

Fig.13 The $f_i - f_o$ plot of (a) the HH and (b) IF0 neurons, dashed curves denoting the extrapolation.
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