A spiking neuron model for synchronous flashing of fireflies

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Abstract

Certain species of fireflies show a group behavior of synchronous flashing. Their synchronized and rhythmic flashing has received much attention among many researchers, and there has been a study of biological models for their entrainment of flashing. The synchronous behavior of fireflies resembles the firing synchrony of integrate-and-fire neurons with excitatory or inhibitory connections. This paper shows an analysis of spiking neurons specialized for a firefly flashing model, and provides simulation results of multiple neurons with various transmission delays and coupling strengths. It also explains flashing patterns of some firefly species and examines the synchrony conditions depending on transmission delays and coupling strengths.

Keywords: Fireflies; Synchronization; Spiking neurons; Integrate-and-fire neurons

1. Introduction

The synchronization phenomena have been observed in many biological animals. Birds in a flock flap their wings in phase, some crickets chirp in unison and a herd of horses keep a similar timing of gaits. Certain species of fireflies flash rhythmically in perfect unison (Buck and Buck, 1976). It was reported that thousands of fireflies in southeast Asia and the Pacific congregate in trees and produce a synchronous flashing.

Especially fireflies show a group behavior of flash communication while a school of fish are responsive to the leader’s movement. There has been a study of biological models for flashing behavior of fireflies. A pioneering work for synchronization of fireflies has been reported by Buck (1988). He investigated a variety of firefly species including Pteroptyx malaccae, Pteroptyx cribellata and Photinus pyralis. He suggested two kinds of flash models, phase-advance entrainment model and phase-delay entrainment model.

Some species, such as P. pyralis try to flash immediately after stimulation with light. Their behavior is called phase-advance entrainment, because the stimulation with light triggers flashes earlier than normal. In contrast, P. cribellata shows flashes a delay after a light stimulation instead of a prompt response, which is described as a phase delay model.

Buck’s firefly model for flashing behavior resembles a spiking neuron model in several respects.

- A flash light from a firefly corresponds to one neuron spike. The membrane potential increases to a threshold level and then the neuron emits a spike or light message.
- The flashing rhythm or frequency is controlled by interactions with neighbor fireflies, as a spiking neuron can adjust its spike time depending on inhibitory or excitatory connections with neighbor neurons.
- A firefly has a neural delay time from the brain to the light organ, as a spiking neuron has its own transmission delay to communicate with other neurons.
- The membrane potential for flashing is decreased to the basal level and then it increases again, as a
spiking neuron has a decay (refractory) period and an integrating period.

- A firefly flash triggers flashes of other fireflies and the entrainment model of a group of fireflies can be seen as a neuronal model of all excitatory or all inhibitory connections among multiple neurons.
- The flash synchrony model of fireflies is similar to the synchrony model of spiking neurons.

The last aspect is related to the synchrony of neuron spikes often studied in the neural network field, which will be the main point of this paper. We propose a biological model with the form of a spiking neuron to explain the synchronous flashing behavior of fireflies.

In the phase advance model displayed in Fig. 1, an excitation level of pace-maker in the firefly brain is enhanced by an external light stimulus. Each time excitation reaches a threshold level, a neural signal is transmitted into the light organ in the abdomen. An example of this model is *P. pyralis*, the whole group of the species rarely synchronizes flashing (Buck, 1988). Instead, localized synchrony or wave sweeping synchrony has been reported in some species with this phase advance model. A cluster of male fireflies flash and then the flash of light is dispersed into neighbor males. One or more local clusters replace the male flashes sequentially. In the phase delay model shown in Fig. 2, an excitation potential of pace-maker is reset to the basal level by a light stimulation and the potential increase is restarted to reach the threshold level for flashing. The flashing style of *P. cribellata* is known as an example of the phase delay model and it shows a regular synchronization. An interesting property of its flashes is that it can shorten the driving period of flashes or lengthen it, depending on the frequency of stimulating signals. This can be explained by Buck’s model which has a resetting operation of pace-maker potential (Buck and Buck, 1976, Buck, 1988) as shown in Fig. 2. The above two entrainment models can be analyzed by the spiking neuron model with all excitatory or all inhibitory connections.

The flashing interaction among fireflies following the phase advance model is similar to all excitatory connections among neurons, because the membrane potential is excited by a light stimulation of neighbor fireflies. The excitatory connections without transmission delays and without refractory periods have been investigated (Mirollo and Strogatz, 1990; Campbell and Wang, 1996; Gerstner and van Hemmen, 1992), and they can induce synchronization. Mirollo and Strogatz (1990) argued that Buck’s model is not appropriate for synchronization because he assumed a linearly increasing potential. They showed that excitatory connections among neurons can lead to synchronization if a convex monotonic function with concave-down curve is taken for membrane po-
They also presented the formal analysis for a dynamic system of phase differences with the convex potential function. Transmission delays, however, was not considered for their spiking function. In fact, the desynchronizing effect of excitatory connections, when they are involved with transmission delays, has been studied (Nischwitz and Glünder, 1995; Ernst et al., 1995; Knoblauch and Palm, 2002). In real fireflies, neural delays from the brain to the light organ are unavoidable, and so the delays greatly influence synchronous flashing.

The phase delay model corresponds to neurons with inhibitory connections. In the field of integrate-and-fire neurons it has been shown that inhibitory connections among neurons can generate synchronized firing, if transmission delays are introduced (Nischwitz and Glünder, 1995; Ernst et al., 1998). Transmission delays influence the performance of synchronization for both excitatory and inhibitory connections (Choe, 2001; Nischwitz and Glünder, 1995; Horn and Opher, 1999; van Vreeswijk and Abbott, 1994). Nischwitz and Glünder (1995) showed that some level of transmission delays can cause desynchronization of neuron spikes and also the synchrony performance is periodic with respect to transmission delays in simulation.

Buck’s pace-maker model shown in Fig. 1 and 2 has an excitation potential of saw-tooth shape. According to the argument of Mirollo and Strogatz (1990), the convex shape of the potential function will be taken for a spiking neuron model in this paper. The decay component from threshold level to basal level can be modeled as a refractory period in a neuron. Normally the transmission delays from sensors to motor actions of flashing are around 200 ms and the regular endogenous period of _P. crubeellata_ is almost 1000 ms (Buck, 1988). Also it can be inferred from his firefly model that the decay period is around 200 ms or larger, since the species can shorten the flashing period down to 800 ms and lengthen it up to 1600 ms.

Some fireflies have various flash pulse patterns depending on what time of the night the fireflies were observed and whether females were encountered (Case, 1984; Mosseiff and Copeland, 2000). In this paper, we do not consider the pace-maker neurons to induce various frequencies or patterns. We will focus on how to form flash-like synchrony phenomena with a simple spiking neuron model and investigate the similarity between a spiking neuron model and the phase entrainment model of fireflies.

Our spiking neuron model is extended from Buck (1988)’s pace-maker model; a transmission delay, a decay period and a resetting operation of membrane potential are added to an integrate-and-fire neuron model, because Buck’s model assumed linearly increasing potential and it is not an accurate model in demonstrating synchronization (Mirollo and Strogatz, 1990). The suggested model resembles spiking neuron models which have been studied previously (Gerstner et al., 1996; Gerstner, 2001; Ernst et al., 1995, 1998; van Vreeswijk, 1996). Gerstner et al. (1996) have a postsynaptic potential model with a slow decay term, where firing occurs when the postsynaptic response due to the presynaptic input from other neurons exceeds its threshold level. Also Gerstner (2001) assumed that the synaptic input has an exponential pulse rather than a simple impulse pulse and their spiking neuron model is more realistic than the neuron model of this paper. Our spiking neuron model follows a pace-maker model to simulate firefly flashings and the neuron fires with a regular period, even without any presynaptic input from neighbor neurons, as a real firefly can flash regularly without any light stimulation.

Ernst et al. (1998) analyzed the dynamics of pulse coupled oscillators with coupling strengths and delays, and simulated the interaction of multiple neuron oscillators to show phase clustering of neuron spikes. However, refractory period and decay term were not considered for their model. Nischwitz and Glünder (1995) also simulated pulse-coupled oscillators with coupling strengths and various delays, and their model has a refractory period in which there is no change of membrane potential over input signals, regardless of inhibitory or excitatory connections.

The suggested spiking neuron model in this paper has a transmission delay, a decay period and a resetting operation by strong inhibitions. The main contribution of this paper is to show the synchrony conditions of the given spiking neuron model specialized for the firefly flashing model as well as simulate multiple neurons to understand the flashing patterns of fireflies.

We will first start with an integrate-and-fire neuron model for the dynamic flashing behavior of fireflies in Section 2. A refractory period with a decay component is added to a conductance neuron model, which will be consistent with Buck’s firefly model (Buck,
In Section 3, two pulse-coupled neurons with inhibitory and excitatory connections are analyzed to find the synchrony conditions depending on coupling strengths and transmission delays. Then in Section 4 we simulate multiple-pulse-coupled neurons to model flashing patterns of fireflies. We assumed that during the refractory period inhibitory connections can be active but excitatory connections are inactive\(^1\) (the membrane potential is not changed by excitation in that period). The phase advance or the phase delay model for flashing of fireflies is investigated analytically in terms of excitatory or inhibitory connections and, we can determine the effect of transmission delays and coupling strengths.

2. Model

In an integrate-and-fire neuron model, an oscillator neuron integrates its membrane potential and fires a spike, once it reaches its threshold, and then decays its potential to zero. In this paper a simple model of temporal integration for membrane potentials will be considered. It is equivalent to a leaky integrate and fire neuron with constant input.

\[
\frac{1}{\gamma} x_i = -x_i + c I + \sum_{k=1}^{N} e_{ik} s(x_k, -\delta)
\]

where \(s(x_k, -\delta)\) is the spike function to represent an impulse state of the \(k\)-th neuron before a delay, \(\delta\), \(N\) the number of neighbouring neurons, \(x\), the membrane potential, \(e_{ik}\) the connection weight from neuron \(k\) to \(i\), and \(I\) is the input. There are two intervals in a period, the integrating period and the decay period. For the integrating period, \(c\) is positive and for the decay period, \(c\) is negative and \(\gamma\) is high. When the potential of an oscillator \(i\) reaches its threshold, it will generate a spike and then its value will decay to zero. The above integrate-and-fire neuron model is similar to the spike response model (Gerstner et al., 1996; Gerstner, 2001) in that it has the process of voltage charge and discharge as in the resistance–capacitance (RC) circuit. The constant input in the model will lead to a regular firing of a neuron and the potential function is periodic. Thus its phase function can be written as follows:

\[
f(\phi) = \begin{cases} 
 f_1(\phi) = c_1 e^{-\gamma(\phi - \rho)} - 1 & \text{if } 0 \leq \phi \leq \rho \\
 f_2(\phi) = c_2 (1 - e^{-\gamma(\phi - \rho)}) & \text{if } \rho < \phi \leq T 
\end{cases}
\]

where \(\rho\) is the decay (or refractory) period and \(T\) is the period of the oscillator (we assume \(\rho < T/2\)). The interval for \(T - \rho\) is an integrating period. The spike function is shown in Fig. 3(a). We define an inverse\(^2\) function \(g\) for \(f\) on two intervals, such that \(g(f(\phi)) = \phi\).

3. Analysis of two neurons

3.1. Inhibitory connections

We first start an analysis of two neurons and this analysis will provide an idea of the synchrony conditions of neuron spikes depending on transmission delay, coupling strength, decay period and integrating period. In the spiking neuron model, transmission delays are added to the inhibitory connections. Let \(\epsilon\) be the coupling strength between two neuron oscillators. We define a phase lag function \(\omega\) as \(\omega(\phi, \epsilon) = g(f(\phi) - \epsilon)\). It forms a new phase by dropping the membrane potential of a neuron. It will lag a phase \(\phi\) by inhibitory action. The synchrony performance depends on the relationship between transmission delays and initial phases, and also on the coupling strength between neurons (Kim, 2003). The relationship is subdivided into four cases as in Table 1. An example of the phase diagram is displayed in Fig. 3(b).

The functions \(u_1, u_2\) in Table 1 are phase differences after each oscillator fires, respectively. A cycle of two inhibitory actions produce \(u_1, u_2\) phases for each action. The function \(u_2\) will be iterated for the next series of firings. In this phase analysis, a refractory period is involved and thus the relation between refractory period and transmission delay or between initial phase and refractory period should be considered. The refractory period has a concave-up-decay of the membrane potential and thus the derivative of the potential is monotonously increasing.

\(^1\) This is not confirmed for real biological neurons, but this model will be useful to explain the resetting entrainment model suggested by Buck (1988).

\(^2\) Strictly speaking, an inverse function \(g\) needs two different functions \(g_1, g_2\) for \(f_1, f_2\), respectively.
For $\delta < \rho$, the refractory period and for $\delta \geq \rho$, at least one oscillator experiences a phase shift in the integrating period.

For $\delta < \rho$, the phase lag function by iteration map largely depends on coupling strength. Three possible cases will be investigated: $\epsilon > f(b)$, $\epsilon \leq f(2\delta)$, and $f(2\delta) < \epsilon < f(b)$.

For $\epsilon > f(b)$, two oscillators can in principle achieve synchronization. If there is a phase difference $\phi$ satisfying the condition $u(\delta - \phi, e) + \phi = \rho$, the two oscillators will be synchronized after a cycle. It requires $\epsilon = f(\delta - \phi) - f(\phi) > f(b)$. However, it is not stable to reach the moment that $u(\delta - \phi, e) + \phi = \rho$, starting with an arbitrary phase lag $\phi$. Normally, the limit cycle results in $u(\delta - \phi, e) = \rho$. For this occasion, the phase difference between two oscillators is constant for every cycle of inhibitory actions, but a phase-advanced oscillator will become lagged to the other oscillator after one cycle. Thus, it will have a fixed point $\phi^* \leq \delta - g(e)$. The phase difference will have the maximal fixed point $\delta - g(e)$ and if $\phi < \delta - g(e)$ with $u(\delta - \phi, e) = \rho$, $\phi$ is itself a fixed point.

For $f(2\delta) < \epsilon < f(b)$ or for $\epsilon < f(2\delta)$ and $2\delta \geq \rho$, there exists a fixed point $\phi^* > 0$ such that $u(\delta - \phi^*, e) + 2\phi^* = \rho$ and $u(\delta + \phi^*, e) = \rho$. If $u(\delta - \phi, e) + 2\phi > \rho$, the phase lag increases, and if $u(\delta - \phi, e) + 2\phi < \rho$, the phase lag decreases.

Thus, the two oscillators will reach an equilibrium state of phase difference $\phi^* > 0$ to satisfy the condition $u(\delta - \phi, e) + 2\phi = \rho$. It occurs only if $u(\delta + \phi, e) = \rho$. When $u(\delta + \phi, e) < \rho$, the phase lag becomes larger.

The fixed point with the above condition $u(\delta - \phi, e) + 2\phi = \rho$ can be approximated by $g(e) - \delta$ (actually $\phi^*$ will be smaller than $g(e)$). The greatest lower bound for coupling strength can be obtained from $u(\delta + \phi, e) < w(2\delta, e) = \rho$, which produces $\epsilon > f(2\delta)$.

If $\epsilon < f(2\delta)$ and $2\delta < \rho$, then the phase difference between the two oscillators increases as time passes and then it reaches the moment that the phase lag is larger than the transmission delay. Later one oscillator experiences inhibition during the integrating period and the other oscillator has the inhibition in the refractory time. It accelerates increasing the phase...
difference, and when the phase gap becomes around half the period, the phase lag is stabilized. Thus, the condition $\epsilon < f(2\delta)$ also implies desynchronization of two oscillators.

For $\epsilon = f(\delta)$, two oscillators can have a perfect synchronization. This coupling strength will have $w(\delta - \phi, \epsilon) < \rho$ and $w(\delta + \phi, \epsilon) = \rho$. Given an arbitrary initial phase $\phi$, the phase difference after one cycle is $\rho - w(\delta - \phi, \epsilon) - \phi$, which is smaller than $\phi$. As the cycle of inhibitory actions repeats, the phase difference will decrease. Thus, it will asymptotically reach the fixed point $\phi' = 0$.

From the above analysis for $\delta < \rho$, we have three conditions, $\epsilon \in (0, f(\delta))$ will guarantee the synchronization. Therefore, $\delta > \rho$ will ensure the condition without refractory period. According to the analysis of phase lock conditions in Table 1, $\epsilon \in (0, f(\delta))$ will guarantee the synchronization. Thus, it will asymptotically reach the fixed point $\phi' = 0$.

In case 3 ($\phi \leq T - 2\delta > T/2$), three possibilities, $\phi < -\delta - \rho$, $\phi > -\delta - \rho$ and $\phi > \delta$ are possible. For $\phi > -\delta - \rho$, a phase-lagged oscillator drops its potential in refractory time, and the other oscillator can lag its phase either in integrating period or in refractory period. In the former case the phase-lagged oscillator drops its potential in integrating period, and the other oscillator experiences inhibition during refractory time. It increases its phase difference, and the two oscillators will reach the moment that $\delta + u_1(\phi) < \delta$ and $\delta + u_1(\phi) > \delta$. Thus, it will reach the phase difference of around half the period. In case 4 ($\phi > T - \delta, \delta > T/2$), three possibilities, $\phi < -\delta - \rho$, $\phi > -\delta - \rho$ and $\phi > \delta$ exist. For $\phi > -\delta - \rho$, the phase-lagged oscillator drops its potential in refractory time, and the other oscillator can lag its phase either in integrating period or in refractory period. In the latter case the phase-lagged oscillator drops its potential in integrating period, and the other oscillator can lag its phase either in integrating period or in refractory period.

In the former case the phase-lagged oscillator drops its potential in integrating period, and the other oscillator experiences inhibition during refractory time. It increases its phase difference, and the two oscillators will reach the moment that $\delta + u_1(\phi) > \delta$. Thus, it will reach the phase difference of around half the period.
inhibitions will lead to a fixed point of half the period. However, if \(|\omega_1| \leq T - \delta\), then the problem reduces to case 3.

In summary, synchronization can appear in cases 1 and 3 within a range of coupling strength. If \(\delta < \rho\), the phase difference will be more advanced forward in phase. If \(\delta \geq \rho\) to case 3. An oscillator will be more advanced forward in phase.

Phase-lagged oscillator keeps its membrane potential at a fixed point of half the period. If \(\omega < \delta\) is a synchronization zone with \((0 \leq \omega < \delta, T - \delta < \phi < T)\) for \(\delta < T/2\), or \((0 \leq \omega < T - \delta, \delta < \phi < T)\) for \(\delta > T/2\).

3.2. Excitatory connections

Excitatory connections with transmission delays are now considered. We define a new phase lag function \(\nu\) as \(\nu(\phi, \epsilon) = \omega f(\phi) + \epsilon\). It forms a new phase by increasing its membrane potential by \(\epsilon\). It will advance a phase by excitatory action \(\epsilon\).

In a similar way to inhibitory connections, we can subdivide possible relations between transmission delays and initial phases into four cases as shown in Table 1. For each case, the synchrony condition with a transmission delay and a refractory period will be analyzed as below. During the refractory period, the membrane potential of a neuron will not be increased, because the neuron is in a fatigue state.

In case 1 \((\phi \leq \delta, \delta \leq T/2)\), two occasions \(\delta < \rho\), \(\delta \geq \rho\) are available. For \(\delta \geq \rho\), \(\nu(\delta - \phi, \epsilon) = \delta - \phi\) and also \(\nu(\delta + \phi, \epsilon) = \delta + \phi\). It will have a constant phase difference. If \(\delta + \phi > \rho\), the phase difference after a cycle of two excitations will increase. Repeating excitatory actions will change \(\phi\) to a phase gap larger than the transmission delay. The problem reduces to case 2. For \(\delta \geq \rho\), two cases \(\phi < \delta - \rho\) and \(\phi \geq \delta - \rho\) are available. For the former case, the phase gap increases by different phase shifts of the two oscillators, unless the coupling strength is too large. It will soon reach the moment that \(\phi \geq \delta - \rho\). If \(\nu(\delta - \phi, \epsilon) = T - \phi\) with \(\phi < \delta - \rho\), then the two oscillators will be synchronized. However, this phase difference is not stable and it rarely happens. If \(\nu(\delta - \phi, \epsilon) + 2\epsilon = T\) and \(\nu(\delta + \phi, \epsilon) = T\), they will have a fixed point of phase difference \(\phi\). The fixed point \(\phi\) can be approximated by \(g(1 - \epsilon)\) or smaller. In the situation \(\phi \geq \delta - \rho\), the phase difference is increased, because a phase-lagged oscillator keeps its membrane potential constant in the refractory time, and the phase-advanced oscillator will be more advanced forward in phase.

The phase difference after several cycles will become larger than the transmission delay, which belongs to case 2.

In case 2 \((\phi > \delta, \delta \leq T/2)\), a phase-lagged oscillator experiences a phase shift in the integrating period. If \(|\omega_1(\phi)| \leq \delta\), the phase difference between two oscillators is smaller than the transmission delay, which is reducible to case 1 \((\omega_1, \phi)\) is the phase difference changed after the first firing of an oscillator as shown in Table 1). Otherwise, two positions, \(\delta + u_1 \leq \rho\) and \(\delta + u_2 > \rho\), are possible. For \(\delta + u_1 \leq \rho\), a phase-advanced oscillator has its excitation input in the refractory time and the other oscillator takes a phase shift during the integrating period. Thus, the phase gap decreases. The fixed point \(\phi^*\) becomes \(\delta\), since the two oscillators will be in the refractory period. If \(\delta + u_1 > \rho\), two oscillators take their phase shift during integrating period. When \(\nu(\delta - \phi, \epsilon) = T\), \(\nu(\delta + u_1, \epsilon) = \nu(2\delta, \epsilon)\). For \(2\delta \leq \rho\), the fixed point of \(\phi\) is \(\delta\). If \(2\delta > \rho\), oscillators can have a fixed point \(\phi^*\) such that \(\nu(\delta - \phi^*, \epsilon) = T\) and \(\nu(2\delta, \epsilon) - \delta = \rho^*\), and thus \(\phi^* \leq \delta + g(1 - \epsilon)\). It may happen that for \(\delta + u_1 > \rho\), the phase difference decreases and then \(\delta + u_2 \leq \rho\). For \(\delta + u_2 > \rho\), the phase difference depends on the relative position of \(\delta - \rho\) and \(\delta + u_2\).

The phase gap will have a fixed point around half the normal period.

In case 3 \((\phi \leq T - \delta, \delta > T/2)\), there are two possible situations, \(\phi \leq \delta - \rho\) and \(\phi > \delta - \rho\). For \(\phi \leq \delta - \rho\), a phase-lagged oscillator receives an excitation signal in the refractory time and thus the phase difference increases. After several cycles of excitations, the phase difference becomes so large that \(\delta + \phi > T\). Then two oscillators receive an excitation during the refractory time and \(\phi\) itself becomes a fixed point. For \(\phi \leq \delta - \rho\), the phase gap will increase if the coupling strength is not large \((1 - f(\delta + \phi))\), and then it may reach the phase difference \(\phi > \delta - \rho\). If \(\nu(\delta + \phi, \epsilon) = T\) and \(\phi < \delta - \rho\), it can obtain a fixed point of phase difference \(\phi^*\) such that \(T - \nu(\delta - \phi^*, \epsilon) = 2\rho^*\) and thus \(\phi^* \leq g(1 - \epsilon)\). Especially if \(T - \nu(\delta - \phi, \epsilon) = \phi\), the two oscillators will be in a perfect synchrony, but this will scarcely occur.

In case 4 \((\phi > T - \delta, \delta > T/2)\), the excitation time for each oscillator should be checked. If \(\phi > \delta\), a phase-lagged oscillator experiences a phase shift in the integrating period and the problem reduces to case
3 after the first firing. For $\phi \leq \delta$, a phase function will depend on each excitation time. The conditions can be classified with whether or not $\delta - \phi$ and $\delta + \phi$ are in the refractory period. If $\delta - \phi < \rho$ and $\delta < \delta + \phi < T + \rho$, both oscillators receive stimulation during the refractory time and their phase difference will be constant. If $\delta - \phi < \rho$ and $\delta + \phi \geq T + \rho$, only a phase-lagged oscillator has an excitation in the refractory period and thus the phase difference increases until it is larger than the transmission delay ($\phi > \delta$), which is reducible to case 3. If $\delta - \phi \geq \rho$ and $\delta + \phi < T + \rho$, a phase-advanced oscillator has no phase shift and the phase gap decreases, which also reduces to case 3. If $\delta - \phi \geq \rho$ and $\delta + \phi \geq T + \rho$, both oscillators experience a phase shift in the integrating period, but soon the fixed point of phase difference will be around $\phi = T/2$ or $\phi = \rho$.

In summary, two neuron oscillators with excitatory connections hardly achieve synchronization. If the coupling strength $\epsilon$ is close to $1 - f(\delta)$ in case 1 and case 3, oscillators will have better synchrony performance, or when transmission delays are very small, there will be more chance of synchronization. The fixed point of phase difference is near $\delta$ in several situations.

4. Experiments

A group of fireflies should be modeled with multiple neurons instead of two oscillators. However, the analytic solution of phase difference on multiple neurons is difficult. We thus provide several experiments of multiple neurons with various delays and coupling strength on inhibitory connections or excitatory connections.

First, inhibitory connections were tested with various delays. Fig. 4 shows the synchrony performance for initially evenly spaced phases and it is dependent on transmission delays: the vertical bar represents firing of each neuron and the phase-lagged oscillator will fire at later time. Generally they show localized synchrony groups on small transmission delays. Phase difference can be seen as a distance concept and neurons with small phase differences will be called neighbor neurons. In simulation neighbor neurons are gradually combined into a cluster of neurons. Fig. 4(a)-(d) shows eight groups, five groups, three groups and one group, respectively. The number of groups is inversely proportional to the transmission delay. From the analysis of two oscillators, the synchronization can occur when $\phi < \delta$ or $\phi > T - \delta$ for $\delta < T/2$. It implies that 2 asks band area can have synchrony performance, and thus the number of synchrony groups can be approximated asymptotically by $T/2\phi$. This synchrony clustering result is similar to the result of spiking neurons without refractory period (Ernst et al., 1998).

To see synchronization performance, we define a spike density function within a window size as follows:

$$s(t) = \frac{1}{nW} \sum_{i=1}^{n} \sum_{k=0}^{W-1} h(i, t + k\eta)$$

where $n$ is the number of neurons, $W$ the window size, $\eta$ the time step in the simulation, and $h$ the spike detection function as below:

$$h(i, t) = \begin{cases} 1 & \text{if there is a spike of the } i\text{th neuron within } [t, t + W) \\ 0 & \text{otherwise} \end{cases}$$

A spike density shows how many oscillators are synchronized within a given time slot, and its maximal value 1 is a complete synchronization within the time slot. The window size for spike density indicates the time slot. The synchrony performance can be measured with the maximal spike density during a given time span, since a spike density fluctuates as time passes. When several window sizes $W = 3, 5, 7, 10$ were tested for the maximal spike density, there was no big difference of the curve shape among them, while a larger window size created a more smooth curve of the maximal spike density.

For evenly spaced phases with 30 oscillators, we have the synchrony performance with inhibitory connections in Fig. 5. It is greatly influenced by coupling strength and transmission delays. The performance is globally periodic with respect to transmission delays for small coupling strength. With transmission delays greater than the period of oscillators, the synchrony performance can be improved, since the phase difference between oscillators reduces by the effect of more inhibitory actions. When the coupling strength is increased, it gives more chance to synchronize neighbor oscillators and thus the synchrony performance increases.
Fig. 4. Synchrony performance of 30 neurons with various delays on inhibitory connections ($n = 30$, $\epsilon = 0.01$, vertical bar: spike) (a) $\delta = 80$ ms (b) $\delta = 100$ ms (c) $\delta = 200$ ms (d) $\delta = 400$ ms.

Fig. 5. Maximal spike density with different coupling strength on inhibitory connections ($W = 3$, $n = 30$) (a) $\epsilon = 0.01$ (b) $\epsilon = 0.02$. 
Fig. 6. Synchrony performance of 30 neurons with various delays on excitatory connections ($n = 30, \epsilon = 0.01$, vertical bar: spike) (a) $\delta = 80$ ms (b) $\delta = 100$ ms (c) $\delta = 200$ ms (d) $\delta = 300$ ms.

When similar experiments were taken with all excitatory connections of spiking neurons, we can also see a synchrony clustering in Fig. 6. Unlike inhibitory connections, the number of synchrony groups is increasing in proportion to the transmission delay. Excitatory connections have relatively more difficulty to synchronize. To check the synchrony performance, the maximal spike density was measured for a wide range of transmission delays. Fig. 7 shows that high spike density can be obtained when the transmission delay is close to zero or close to the period of oscillators, as expected from the analysis of two oscillators. When the transmission delay is close to half the period, oscillators are divided into two groups for synchronization and the performance becomes around 0.5. If we increase transmission delays with larger coupling strength, the synchrony performance is increased because neighbor oscillators have more chance of being excited and absorbed together with the same spiking time.

When we use a large coupling strength between each pair of neurons, their performance can be enhanced as shown in Fig. 8(a)-(b). Excitatory connections produce more frequent synchrony of firings than inhibitory connections. If we test the synchrony performance using transmission delays less than refractory period, the synchrony performance of excitatory actions becomes much worse—see Fig. 8(c)-(d). Strong excitatory connections require transmission delays larger than the refractory period for high synchrony performance. *P. cribellata* in the phase delay model has a regular synchronization pe-
Fig. 7. Maximal spike density with different coupling strength on excitatory connections ($W=3, n=30$) (a) $\epsilon=0.01$ (b) $\epsilon=0.02$.

Fig. 8. Spike density vs. time with large coupling strength ($W=3, n=30, \epsilon=0.17$) (a) inhibitory action ($\delta=300\text{ ms}$) (b) excitatory action ($\delta=300\text{ ms}$) (c) inhibitory action ($\delta=200\text{ ms}$) (d) excitatory action ($\delta=200\text{ ms}$).
Fig. 9. Membrane potential for large coupling strength ($\epsilon = 0.5$, $\delta = 0.2T$) (a) inhibitory action (b) excitatory action.

iod of 1000 ms, and the spiking neurons shown in Fig. 8(c) show almost 1000 ms synchrony period using a 200 ms transmission delay and a 200 ms decay period.

Normally, excitatory activations generate frequent spikes in a short period of time, because it can shorten the period of oscillation by increasing the potential level. We believe that this kind of strong excitations will not be observed in the firefly model. Frequent flashing of light will exhaust out the energy of the insect, which may be a disadvantage rather than profit to survive the environment in an evolutionary concept. Thus, a relatively small coupling strength for excitatory action will be used in many species of fireflies, which follows the phase advance model. From the experiments, small coupling strength on excitatory connections with about 200 ms neural delay shows very low synchrony performance. This result agrees with to the fact that Buck (1988) observed rare or local synchrony in many fireflies with the phase advance entrainment model.

Fig. 9 shows how oscillators are synchronized together. We assume a 200 ms transmission delay for spiking neurons as there is a neural delay of 200 ms from the brain to the light organ in P. cribellata or P. malaccae. Spiking neurons with inhibitory connections are synchronized only after a few cycles. Those with excitatory connections form synchrony clusters but without perfect synchrony.

5. Discussion

The simulation experiments showed that there are synchrony clustering phenomena for both excitatory and inhibitory connections. Buck (1988) assumed a resetting operation of inhibitory action for his entrainment model of P. cribellata. The resetting operation corresponds to a large coupling strength for inhibitory action in our model. Fig. 9(a) shows the process of synchronization with large inhibitions. One or two cycles later neurons can synchronize together as fireflies synchronize flashes in a few cycles. If the coupling strength is very large, neighbor synchrony groups will be combined easily into one large synchrony group.

If transmission delays are added to excitatory connections with small coupling strength, all neurons rarely synchronize their spikes. Instead localized synchrony can occur. However, the simulation experiments with a refractory period show a potential of synchronization for a large coupling strength. It seems that P. pyralis uses a small coupling strength on excitatory connections, and its low synchrony performance indirectly supports the idea.

Local synchronization can be predicted with small coupling strength regardless of whether the connections among neurons are excitatory or inhibitory. The formation of synchrony clustering observed in Fig. 4 and 6 may be a possible explanation of local synchrony in firefly flashing. Buck (1988) mentioned that there was an observation of synchrony waves among local groups in flashing of some firefly species. If
fireflies are remotely distant one another, the light intensity is relatively low to trigger flashing, and thus a collection of flashing, not one flash, may trigger flashing of another group of fireflies by increasing the intensity. In another respect, the flashing patterns of fireflies may result from an intrinsic property of synchrony clustering in the spiking neurons, which was shown in the experiments on both inhibitory and excitatory connections. The local synchrony is automatically developed by the relationship between phase difference and transmission delays with small coupling strength. It is not clear whether the flashing waves of fireflies is due to the intensity difference by the distance between each group or due to the phase difference in relation with delays.

Another synchronous firefly is *Pteroptyx malaccae*. It has a flashing cycle with an interval of around 560 ms. Wave synchrony occurs among the species even though it follows the phase delay model; local groups drift and a flashing wave spreads among groups. It was observed that waves sweep from tree to tree (Buck and Buck, 1968). This species shows the ability of adapting its frequency to a given light signal as well as achieving zero-phase synchrony for a single light, while *P. cribellata* only adapts its frequency to a light signal. The flashing mechanism of *P. malaccae* seems more complex than *P. cribellata* and possibly its synchrony clustering may be due to its small coupling strength.

6. Conclusion

It was assumed in this paper that a spiking neuron interacts with neighbor neurons by all excitatory or inhibitory connections. The spiking neuron model suggested in this paper has a decay period, an integrating period and a transmission delay for each spike. The model resembles a biological model of synchronous flashing of fireflies. The phase advance model and the phase delay model correspond to excitatory and inhibitory actions, respectively.

We first started with an analysis of two neuron oscillators and then showed the simulation results of multiple neurons, which produced the similarity between the spiking neuron model and the flashing model of fireflies. The spiking neuron model suggested in this paper explains the whole mechanism of flashing in a simple manner. It has shown possible synchronization states and a local synchrony model depending on coupling strength. However, it is still arguable that the whole flashing mechanism even for one specific species can be explained by only a few spiking neurons. In real fireflies there are many kinds of flashing patterns which are not described here but are much more complex. Ermentrout (1991) showed an adaptive model using many neuron oscillators for synchrony of the *P. malaccae*. It would be a more plausible model for real fireflies than our spiking neuron model.

We assumed identical spiking neurons in which the same transmission delay is taken for each neuron without noise. To be more realistic we need to allow a variation of transmission delays and noise in a group of neurons or fireflies. Also the light intensity for the visual input should be considered together. It is not clear yet whether the suggested neuron model will also fit other synchronized phenomena or whether the model is suitable for the synchronization of biological spiking neurons. We need further study for this argument.

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References


