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Short-Term Synaptic Plasticity and Network Behavior

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We develop a minimal time-continuous model for use-dependent synaptic short-term plasticity that can account for both short-term depression and short-term facilitation. It is analyzed in the context of the spike response neuron model. Explicit expressions are derived for the synaptic strength as a function of previous spike arrival times. These results are then used to investigate the behavior of large networks of highly interconnected neurons in the presence of short-term synaptic plasticity. We extend previous results so as to elucidate the existence and stability of limit cycles with coherently firing neurons. After the onset of an external stimulus, we have found complex transient network behavior that manifests itself as a sequence of different modes of coherent firing until a stable limit cycle is reached.

1 Introduction

Short-term synaptic plasticity refers to a change in the synaptic efficacy on a timescale that is inverse to the mean firing rate and thus of the order of milliseconds. It is therefore natural to inquire whether and to what extent this has functional consequences and to elucidate the underlying mechanisms (Markram & Tsodyks, 1996; Abbott, Varela, Sen, & Nelson, 1997; Senn, Segev, & Tsodyks, 1997). The experimental observation underpinning short-term synaptic plasticity is the fact (Zucker, 1989) that the transmission of an action potential across a synapse can have a significant influence on the height of the postsynaptic potential (PSP) evoked by subsequently transmitted spikes. In some neurons, the height of the postsynaptic potential is increased by spikes that have arrived previously (short-term facilitation, STF). In others, the postsynaptic potential is depressed by previously arrived action potentials (short-term depression, STD).

Short-term synaptic plasticity, or simply short-term plasticity, is different from its well-known counterpart long-term plasticity, in at least two crucial points. First, *nomen est omen*, the timescale on which short-term plasticity operates is much shorter than that of long-term plasticity and may well be comparable to the timescale of the network dynamics. Second, short-term plasticity of a given synapse is driven by correlations in the incoming

spike train (presynaptic correlations), whereas classical long-term plasticity is driven by correlations of both pre- and postsynaptic activity; a prominent example of the latter is Hebb's learning rule (Hebb, 1949; Gerstner & van Hemmen, 1993).

The article is organized as follows. We start by analyzing a simple model of short-term plasticity that is an adaptation of the model of Tsodyks and Markram (1997) to the spike response model (Gerstner & van Hemmen, 1992). In section 3 we analytically discuss the implications of short-term plasticity for the behavior of a homogeneous, strongly connected network and show that the dynamics exhibits attractive limit cycles of coherent neuronal activity. This is illustrated in section 4, where we present computer simulations and discuss the transient behavior of the network that shows up before the dynamics has settled down in its limit cycle. Beforehand we define the difference between coherence and synchrony. "Coherently firing" means "periodically firing with constant phase difference," while firing synchronously" implies phase difference zero.

2 Short-Term Synaptic Plasticity

Modeling short-term plasticity is based on the idea that some kind of "resources" is required to transmit an action potential across the synaptic cleft (Liley & North, 1953; Magleby & Zengel 1975; Abbott et al., 1997; Tsodyks & Markram, 1997; Varela et al., 1997). The term *resource* can be interpreted as the available amount of neurotransmitter, some kind of ionic concentration gradient, or the postsynaptic receptor density or availability. We assume that every transmission of an action potential affects the amount of available synaptic resources and that the amount of available resources determines the efficiency of the transmission and therefore the maximum of the postsynaptic potential.

We intend to discuss short-term plasticity in the context of the spike response model, of which we give a short review; details can be found in Gerstner and van Hemmen (1992) and Kistler, Gerstner, and van Hemmen, (1997). It will turn out that this formalism is very convenient in deriving closed analytic expressions for the synaptic strengths as a function of spike arrivals and time.

2.1 Spike Response Neurons. The spike response model (Gerstner & van Hemmen, 1992) does not concentrate on the details of the synaptic transmission but focuses on the effect of an incoming action potential on the membrane potential at the soma. There it is described by an response function ϵ [with $\epsilon(t < 0) = 0$] that represents the time course of a postsynaptic potential. Several postsynaptic potentials are assumed to superpose linearly in space and time so that the membrane potential at the soma of

neuron i is given by

$$h_i(t) = \sum_{j,f} J_{ij} \epsilon \left(t - t_j^f - \Delta_{ij} \right) ,$$

where the t_j^f are the firing times of the presynaptic neuron j, J_{ij} is the strength of the synapse connecting neuron j to neuron i, and Δ_{ij} is the axonal delay from neuron j to neuron i.

A spike is triggered as soon as the membrane potential reaches the firing threshold ϑ from below. Refractory behavior is implemented by increasing the threshold for some time after the neuron has fired or, equivalently, by adding a negative afterpotential $\eta(t)$ to the membrane potential whenever the neuron has fired. Altogether we have

$$h_i(t) = \sum_{j,f} J_{ij} \epsilon \left(t - t_j^f - \Delta_{ij} \right) + \sum_f \eta \left(t - t_i^f \right) , \tag{2.1}$$

with

$$\lim_{t \nearrow t_i^f} h_i(t) = \vartheta \qquad \text{and} \qquad \lim_{t \nearrow t_i^f} \frac{dh_i(t)}{dt} > 0 . \tag{2.2}$$

The spike response model is a generalization of the standard integrate-and-fire model. This can easily be seen if the response functions ϵ and η are replaced by exponentials (for details, see Kistler et al., 1997).

If we want to include short-term plasticity, we have to replace the constants J_{ij} by functions of time, $J_{ij}(t)$, which give the strength of the synapse at time t. The relevant quantity for synaptic transmission is the synaptic strength at the time of the arrival of a presynaptic spike,

$$h_i(t) = \sum_{j,f} J_{ij} \left(t_j^f + \Delta_{ij} \right) \epsilon \left(t - t_j^f - \Delta_{ij} \right) + \sum_f \eta \left(t - t_i^f \right) . \tag{2.3}$$

The time-dependent synaptic strength $J_{ij}(t)$ is a function that depends on both time and the moments of arrival of the spikes from neuron j. This function will be computed in the next subsections.

2.2 Modeling Short-Term Depression. Simple models based on first-order reaction kinetics have repeatedly been shown to allow for a quantitative description of short-term plasticity at neuromuscular junctions (Liley & North, 1953; Magleby & Zengel, 1975) and cortical synapses (Tsodyks & Markram 1997; Varela et al., 1997). The model of Tsodyks and Markram (1997) assumes three possible states for the "resources" of a synaptic connection: effective, inactive, and recovered. Whenever an action potential arrives at a synapse, a fixed portion R of the recovered resources becomes

first effective, then inactive, and finally recovers. Transitions between these states are described by first-order kinetics using time constants τ_{inact} and τ_{rec}. The actual postsynaptic current is proportional to the amount of effective resources.

In the context of the spike response model, the three-state model can be simplified since the time course of the postsynaptic current, as it is described by the transition from the effective to the inactive state, is already taken care of by the form of the postsynaptic potential given by the response function ϵ. The only relevant quantity is the maximum (minimum)[1] of the PSP determined by the charge delivered by a single action potential. Since transitions from the effective and the inactive to the recovered state are described by linear differential equations, the maximum of the PSP depends on only the amount of resources that are actually activated by the incoming action potential. We may thus summarize the two-step recovery of effective resources by a single step and end up with a two-state model of active (Z) and inactive ($\bar{Z}$) resources. Each incoming action potential instantaneously switches a proportion R of active resources to the inactive state from where they recover to the active state with time constant τ; see Figure 1A. Formally,

$$\frac{dZ(t)}{dt} = -R\,Z(t)\,S(t) + \tau^{-1}\,\bar{Z}(t)\,, \qquad \bar{Z}(t) = 1 - Z(t)\,, \tag{2.4}$$

with $S(t) = \sum_f \delta(t - t_f)$ being the incoming spike train. This differential equation is well defined if we declare $Z(t)$ to be continuous from the left—$Z(t_f) := Z(t_f - 0)$.

The amount of charge that is released in a single transmission and therewith the maximum of the PSP depends on the amount of resources that are switched to the inactive state or, equivalently, on the amount of active resources immediately before the transmission. The strength of the synapse at time t is then a function of $Z(t)$, and we simply put $J(t) = J^0\,Z(t)$ where J^0 is the maximal synaptic strength with all resources in the active state.

Let us now suppose that the first spike arrives at a synapse at time t_0. Immediately before the spike arrives, all resources are in their active state, and $Z(t_0) = 1$. The action potential switches a fraction R of the resources to the inactive state so that $Z(t_0 + 0) = 1 - R$. After the arrival of the action potential, the inactive resources recover exponentially fast in t, and we have

$$Z(t > t_0) = 1 - R\,\exp\left[-(t - t_0)/\tau\right]\,.$$

At the arrival time t_1 of the subsequent spike, there are only $Z(t_1)$ resources in the active state, and the PSP is depressed accordingly (see Figures 2A and 2B).

[1] We henceforth drop the alternative minimum, which takes care of an inhibitory postsynaptic potential, and assume an excitatory one, the modifications for inhibition being evident.

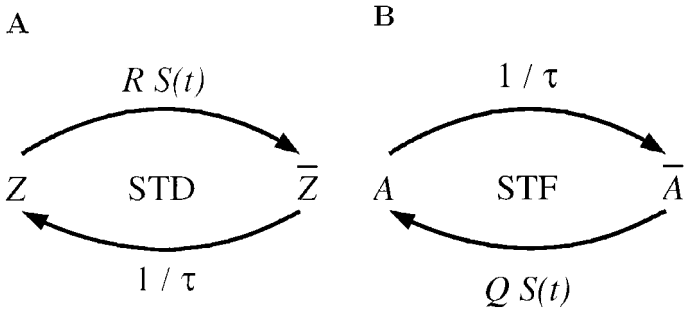

Figure 1: Schematic representation of the present model of short-term depression (A) and short-term facilitation (B). With short-term depression, every incoming action potential switches a proportion R of active resources Z to the inactive state $\bar{Z}$. This is symbolized as a first-order reaction kinetics with the time-dependent rate $R\,S(t)$; here S is the incoming spike train. From the inactive state, resources relax to the active state with time constant τ. The model for short-term facilitation emerges from the model for short-term depression simply by inverting the directions of the arrows. $\bar{A}$ represents the ineffective resources, which are decimated by incoming spikes with a rate $Q\,S(t)$. The active resources A relax back to the inactive state with a rate τ^{-1}.

From the first few examples we can easily read off a recurrence relation that relates the amount of active resources immediately before the nth spike to that of the previous spike,

$$\begin{aligned}
Z(t_0) &= 1 \\
Z(t_1) &= 1 - R\,\exp\left[-(t_1 - t_0)/\tau\right] \\
Z(t_2) &= 1 - \left[1 - (1 - R)\,Z(t_1)\right]\,\exp\left[-(t_2 - t_1)/\tau\right] \\
&\vdots \\
Z(t_n) &= 1 - \left[1 - (1 - R)\,Z(t_{n-1})\right]\,\exp\left[-(t_n - t_{n-1})/\tau\right]\,. \qquad (2.5)
\end{aligned}$$

In passing we note that instead of $Z(t_0) = 1$, we could have taken any desired initial condition $0 < Z_0 \leq 1$; the ensuing argument does not change.

The recurrence relation (see equation 2.5) is of the form $Z(t_n) = a_n + b_n\,Z(t_{n-1})$ with $a_n = 1 - \exp[-(t_n - t_{n-1})/\tau]$ and $b_n = (1 - R)\,\exp[-(t_n - t_{n-1})/\tau]$. Recursive substitution and a short calculation yield the following explicit expression for the amount of active resources,

$$\begin{aligned}
Z(t_n) &= a_n + b_n\,a_{n-1} + b_n\,b_{n-1}\,a_{n-2} + \cdots \\
&= \sum_{k=0}^{\infty} a_{n-k} \prod_{j=0}^{k-1} b_{n-j}
\end{aligned}$$

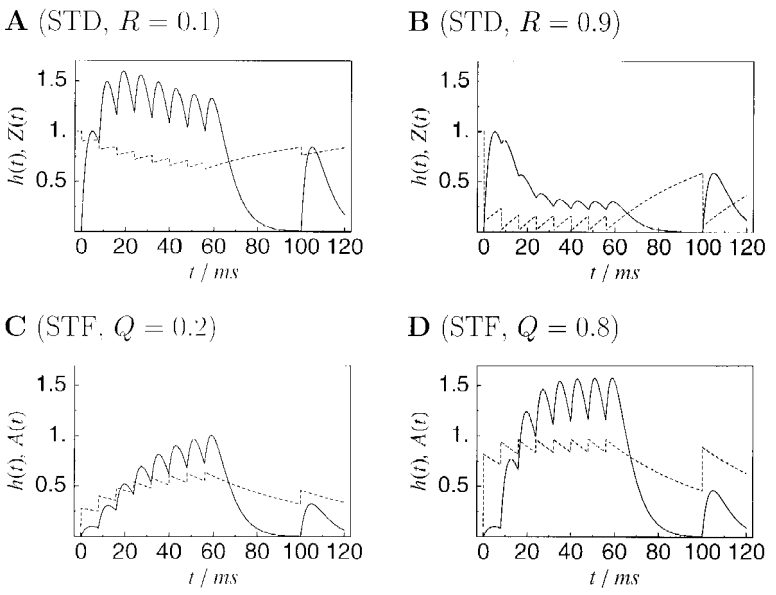

Figure 2: Membrane potential (solid line) and synaptic strength (dashed line) as a function of time in case of short-term depression (A, B) and facilitation (C, D). In (A), only a small portion $R = 0.1$ of all available resources is used during a single transmission, so that the synapse is affected only slightly by transmitter depletion. In (B), the parameter R is increased to $R = 0.9$. This results in a pronounced short-term depression of the synaptic strength. Short-term facilitation is illustrated in the lower two diagrams for $A_0 = 0.1$, $Q = 0.2$ (C) and $A_0 = 0.1$, $Q = 0.8$ (D). For all figures, the time constant of synaptic recovery is $\tau = 50$ ms, and the rise time of the EPSP equals 5 ms. The spikes arrive at $t = 0, 8, 16, \ldots, 56$ ms, and finally at $t = 100$ ms.

$$= \sum_{k=0}^{\infty} a_{n-k}(1-R)^k \exp[-(t_n - t_{n-k})/\tau]$$

$$= 1 - \frac{R}{1-R} \sum_{k=1}^{\infty} (1-R)^k \exp[-(t_n - t_{n-k})/\tau]. \qquad (2.6)$$

The synaptic strength at time t as a function of the spike arrival times $t > t_{n-1} > t_{n-2} > \cdots$ is given by

$$J(t; t_{n-1}, t_{n-2}, \ldots) = J^0 \left\{ 1 - \frac{R}{1-R} \sum_{k=1}^{\infty} (1-R)^k \exp[-(t-t_{n-k})/\tau] \right\}. \qquad (2.7)$$

This is a key result for what follows.

2.3 Periodic Input. The synaptic strength J is a nonlinear function of the spike arrival times t_f. We can give a simplified expression for J in the case of a sudden onset of periodic spike input. Let $t_n = n\,T$ for $n \geq 0$ and $t_n = -\infty$ for $n < 0$. We obtain from equation 2.6 for $n > 0$,

$$\begin{aligned} Z(t_n) &= 1 - \frac{R}{1-R} \sum_{k=1}^{n} (1-R)^k \exp[-k\,T/\tau] \\ &= 1 - \frac{R}{\mathrm{e}^{T/\tau} - (1-R)} \left\{ 1 - \left[(1-R)\,\mathrm{e}^{-T/\tau} \right]^n \right\}. \end{aligned} \tag{2.8}$$

The behavior of $Z(t_n)$ for large n can be read off easily from the above equation. Since $0 < \mathrm{e}^{-T/\tau}\,(1-R) < 1$, the braced expression converges to unity exponentially fast, and the rest, which is independent of n, gives the asymptotic value of $Z(t_n)$ as $n \to \infty$.

2.4 Modeling Short-Term Facilitation. In a similar fashion, we can devise a model that accounts for short-term facilitation instead of depression. To this end, we assume that in the absence of presynaptic spikes, the fraction of active synaptic resources $A(t)$ decays with time constant τ. Each incoming spike recruits a proportion Q from the reservoir $\bar{A}$ of ineffective resources; see Figure 1B. Then the dynamics of $A(t)$ is

$$\frac{dA(t)}{dt} = Q\,\bar{A}(t)\,S(t) - \tau^{-1}\,A(t), \qquad \bar{A}(t) = 1 - A(t), \tag{2.9}$$

with $S(t) = \sum_f \delta(t - t_f)$ as the incoming spike train and $A(t)$ being continuous from the left. Magleby and Zengel (1975) used a similar model to describe synaptic potentiation at the frog neuromuscular junction.

For a discrete set of spike arrival times $t_f = t_0, t_1, \ldots$ the amount of effective synaptic resources immediately before the nth spike as a function of that before the previous spike is

$$A(t_n) = a_n + b_n\,A(t_{n-1}), \tag{2.10}$$

where

$$\begin{aligned} a_n &= Q \exp\left(-\frac{t_n - t_{n-1}}{\tau} \right) \quad \text{and} \\ b_n &= (1-Q) \exp\left(-\frac{t_n - t_{n-1}}{\tau} \right). \end{aligned} \tag{2.11}$$

In a similar way to equation 2.6, we obtain an explicit expression for the amount of effective resources. We adopt a simple linear dependence of the synaptic strength J on the amount of effective resources A of the form

$J = J^0 [A_0 + (1 - A_0) A]$, $0 \leq A_0 \leq 1$, with J_0 being the maximal synaptic strength and $(J^0 A_0)$ its minimal strength (see Figures 2C and 2D). Altogether we have

$$J(t; t_{n-1}, t_{n-2}, \ldots) = J^0 \left\{ A_0 + (1-A_0) \frac{Q}{1-Q} \sum_{k=1}^{\infty} (1 - |!Q)^k \exp\left[-(t-t_{n-k})/\tau\right] \right\}. \quad (2.12)$$

In the case of periodic input with $t_n = n\,T$ for $n \geq 0$, and $t_n = -\infty$ for $n < 0$, the above equation reduces to

$$J(t_n)/J^0 = A_0 + (1 - A_0) \frac{Q}{e^{T/\tau} - (1-Q)} \left\{ 1 - \left[(1-Q) e^{-T/\tau} \right]^n \right\}. \quad (2.13)$$

This implies that as $n \to \infty$, the synaptic strength converges exponentially fast from below to the asymptotic value

$$J_{\infty}^{\mathrm{STD}} = J^0 \left[A_0 + \frac{(1 - A_0)\, Q}{e^{T/\tau} - (1-Q)} \right]. \quad (2.14)$$

3 Consequences for Network Dynamics

Short-term plasticity introduces a second timescale into the dynamics of a neural network. In this section we analyze the implications of short-term plasticity for a homogeneous network of excitatory neurons. We assume that each neuron is connected to all the other neurons, with all couplings and delays being identical. This setup can be thought of as an idealization of a large network of heavily interconnected neurons.

3.1 Locking and Short-Term Depression. A homogeneous network of N spike-response neurons can show coherent oscillations (Gerstner & van Hemmen, 1993). In the simplest case of constant synaptic strength, all neurons fire synchronously with period T provided

$$N \sum_{k=1}^{\infty} J \epsilon(k\,T - \Delta_{\mathrm{ax}}) + \sum_{k=1}^{\infty} \eta(k\,T) = \vartheta\,. \quad (3.1)$$

Here, ϵ and η are postsynaptic potential and refractory field of an SRM neuron, J is the synaptic efficacy, Δ_{ax} is the axonal delay, and ϑ the threshold.

There are other periodic solutions of the network dynamics that involve a partition of the neurons into n subpopulations of N/n neurons each (Gerstner & van Hemmen, 1993). The subpopulations fire their action potentials in an alternating way so that each neuron fires with period T, but the activity of the network has period T/n, which is given by

$$\frac{N}{n} \sum_{k=1}^{\infty} J \epsilon(k\,T/n - \Delta_{\mathrm{ax}}) + \sum_{k=1}^{\infty} \eta(k\,T) = \vartheta\,. \quad (3.2)$$

A coherent oscillation is stable if the spikes are triggered within the rising phase of the synaptic contribution of the local field (Gerstner, Ritz, & van Hemmen, 1996), that is, if

$$\frac{d}{dt}\left[\sum_{k=1}^{\infty}\epsilon(t+kT/n-\Delta_{\mathrm{ax}})\right]_{t=0} > 0\,. \tag{3.3}$$

The period T of the oscillation is a root of equation 3.2 and thus a function of the synaptic strength J. On the other hand, if we include short-term depression in our model, then the synaptic strength depends on the past spike arrival times. The fixed points of the dynamics are determined by the solutions of equation 3.2, if we replace J by its asymptotic value $J_{\infty}^{\mathrm{STD}}(T)$ as it follows from equation 2.8,

$$J_{\infty}^{\mathrm{STD}}(T) = J^0\left[1-\frac{R}{\mathrm{e}^{T/\tau}-(1-R)}\right], \tag{3.4}$$

or, alternatively, by the simultaneous solutions of equation 3.2 and

$$J = J_{\infty}^{\mathrm{STD}}(T)\,. \tag{3.5}$$

Figure 3 shows the solution to equation 3.2 as a function of J together with the graph of $J_{\infty}^{\mathrm{STD}}(T)$ or, more precisely, the graph of the inverse function $J \mapsto T(J) = (J_{\infty}^{\mathrm{STD}})^{-1}(J)$. The intersections of the graphs are the limit cycles of the network dynamics.

Stability deserves closer attention. We will first discuss the case of a slowly (adiabatically) changing synaptic strength, that is, $(1-R)\,\mathrm{e}^{-T/\tau}$ close to unity; see equation 2.8. With an adiabatically changing synaptic strength, the network will remain in the locked state unless this state becomes eventually unstable. This is the case if equation 3.3 is no longer fulfilled.

The period T of the locked state is a monotonically decreasing function of the synaptic strength J, and the asymptotic value $J_{\infty}^{\mathrm{STD}}$ is a monotonically increasing function of T. The simultaneous solutions of equations 3.2 and 3.5 that obey equation 3.3 are thus stable fixed points. To see why, imagine that the neurons lock with a period T smaller than the period T^* at the fixed point in Figure 3. Since locking is fast compared to the relaxation of the synaptic strength, this can be the case only if the actual value of the synaptic strength J is larger than that at the fixed point and, because of the monotony of $J_{\infty}^{\mathrm{STD}}$, larger than the corresponding equilibrium strength $J_{\infty}^{\mathrm{STD}}(T)$. Synaptic strength is thus declining, and the period T is increasing until the fixed point is reached. A similar argument holds for periods larger than that of the fixed point in Figure 3.

We now turn to the case where the synapses are substantially affected by the transmission of a single spike, that is, $(1-R)\,\mathrm{e}^{-T/\tau} \ll 1$. In this case

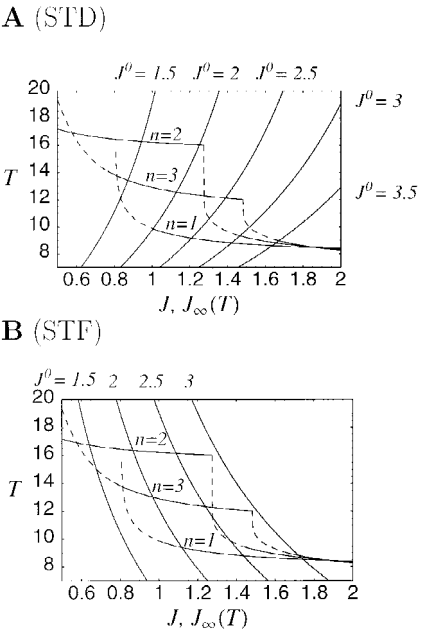

Figure 3: (A) This plot combines the graphs of $J_\infty^{\mathrm{STD}} = J_\infty^{\mathrm{STD}}(T)$ for various values of J^0 (see equation 3.4, solid lines) with graphs of the solutions T of equation 3.2 as a function of the synaptic strength J and $n = 1$ (lower trace), $n = 2$ (upper trace), and $n = 3$ (middle trace). A dashed line indicates those regions where the stability criterion, equation 3.3, is not fulfilled. The neurons are defined by an excitatory postsynaptic potential $\epsilon(t) = 5^{5/4}(\mathrm{e}^{-t/5} - \mathrm{e}^{-t})\,\Theta(t)/4$ and a refractory field $\eta(t) = -5\,\mathrm{e}^{(2-t)/5}\,\Theta(t-2)$, where Θ denotes the Heaviside function with $\Theta(t) = 1$ for $t > 0$ and $\Theta(t) = 0$ for $t < 0$. The threshold is $\vartheta = 0$, the axonal delay is $\Delta_{\mathrm{ax}} = 8$, and the parameters of short-term depression are $R = 0.05$ and $\tau_{\mathrm{syn}} = 200$. (B) As (A), but with short-term facilitation ($\tau_{\mathrm{syn}} = 200$, $Q = 0.05$, $A_0 = 0.1$) instead of short-term depression. Here too dashed lines indicate that stability is lost.

the synaptic strength can be taken as a function of the very last interspike interval only, and a calculation similar to that of Gerstner et al. (1996) shows that stability is solely determined by the criterion equation 3.3 independent of synaptic plasticity. Details can be found in the appendix.

3.2 Locking and Short-Term Facilitation. The arguments of the previous section go through almost unchanged if the synapses show short-term facilitation instead of short-term depression. We only have to replace the

asymptotic synaptic strength J_∞^{STD} by

$$J_\infty^{\mathrm{STF}}(T) = J^0 \left[A_0 + (1 - A_0) \frac{Q}{\mathrm{e}^{T/\tau} - (1 - Q)} \right]; \tag{3.6}$$

see equation 2.3.

The stability analyses for rapidly adapting synapses with short-term facilitation and short-term depression are equivalent. That is, stability of the limit cycle is given by the locking theorem represented by equation 3.3.

The argument for the adiabatic limit with short-term facilitation, however, is slightly more complicated than with short-term depression because both the period $T(J)$ and the asymptotic value $J_\infty^{\mathrm{STF}}(T)$ are monotonically decreasing functions. The stability of the fixed points depends on the slope with which the graphs of $(J_\infty^{\mathrm{STF}})^{-1}(J)$ and $T(J)$ intersect.

To see why the slope is the factor determining stability, let us assume that the neurons lock with a period T smaller than the period T^* of the fixed point or, equivalently, that the synaptic strength J is larger than J^*. Whether the corresponding equilibrium strength $J_\infty^{\mathrm{STF}}(T)$ is larger or smaller than J depends on the slope of $(J_\infty^{\mathrm{STD}})^{-1}(J)$ relative to the slope of $T(J)$. If $(J_\infty^{\mathrm{STD}})^{-1}(J)$ is steeper than $T(J)$, then the equilibrium strength is smaller than the actual strength, and the synapses will be weakened and the period will increase until the fixed point (J^*, T^*) is reached. Otherwise, if the equilibrium strength is larger than the actual strength, the synapses will be strengthened even more, and, thus, the fixed point is unstable.

4 Simulations

In order to illustrate the analytic considerations of the previous section, we have performed simulations on a network consisting of 100 spike response neurons. We have included noise in that we have replaced the sharp firing threshold ϑ by a firing probability that depends on the actual value of the membrane potential $h(t)$; that is, we have assumed an inhomogeneous Poisson process with

$$\mathrm{Prob}\{\text{spike in } [t, t + \mathrm{d}t)\} = \exp[\beta\,(h(t) - \vartheta)]\,\mathrm{d}t\,. \tag{4.1}$$

The parameter β controls the overall amount of noise in the system; for $\beta \to \infty$ the firing threshold is sharp.

The simulations confirm the predicted stability properties of sections 3.1 and 3.2. Furthermore, they show that the network can have a fairly complicated transient behavior before it settles down in its limit cycle. For example, in the case of slowly developing short-term facilitation, the synaptic weights grow as soon as the network starts firing because of the onset of some external stimulus. We have seen that there are several solutions to the locking equation 3.2, depending on the value of the coupling strength. The network

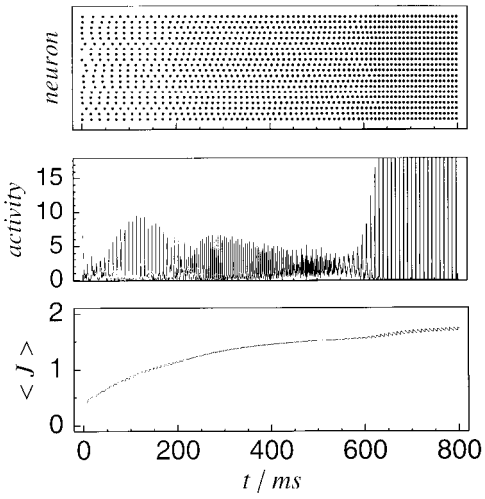

Figure 4: Transient behavior of a network of spiking neurons and short-term facilitation. (Top) A spike raster of 20 neurons randomly selected from the 100 neurons contained in the network. (Center) A plot of the network activity. (Bottom) The averaged synaptic strength as a function of time. Closer inspection of the firing patterns reveals intricate network behavior. From $t = 50$ ms to $t = 150$ ms, the neurons are organized in $n = 2$ subpopulations, which fire alternatingly. For $t = 250$ ms to $t = 400$ ms, there are $n = 3$ subsequently firing subpopulations. The stable state with $n = 1$, that is, with all neurons firing in phase, is not reached before $t = 550$ ms. The simulation has been performed with noise parameter $\beta = 20$; see equation 4.1. All the other parameters are identical to those of Figure 3B.

may thus pass through a series of different firing modes until a stable limit cycle is reached.

The network behavior is illustrated in Figure 4, which shows a spike raster, the mean network activity, and the averaged synaptic strength. As can be seen from the spike trains, the network passes through a coherent firing mode with two (50 ms $< t <$ 150 ms) and later with three (250 ms $< t <$ 400 ms) alternatively spiking subpopulations before it settles in a stable limit cycle where all neurons are firing synchronously. Figure 5 shows that the trajectory of the averaged synaptic strength and the averaged interspike interval is attracted by the stable solutions of equation 3.2 (locking) and follows these lines until the corresponding solution eventually becomes unstable and a transition to another firing mode occurs. As can be seen from Figure 5, limit cycles with different n-values may show up as asymptotic states of the network. We have $n = 1$ in Figure 5A and $n = 3$ in Figure 5B.

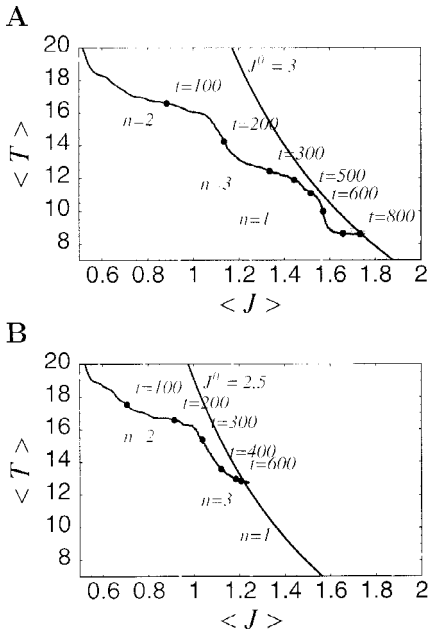

Figure 5: (A) Plot of a curve defined by the averaged coupling strength $\langle J \rangle$ and the averaged interspike interval $\langle T \rangle$ parameterized by the time t for the simulation data shown in Figure 4, with short-term facilitation. The curve has been smoothed by a moving average with a time window of 10 ms. The gray lines represent the asymptotic values of the synaptic strength and the solutions of equation 3.2; see Figure 3B. As can be seen from the graph, the network passes through a series of transient states before it finally reaches the stable limit cycle at the intersection of the lines $n = 1$ and $J^0 = 3$. (B) Similar plot as in (A) but for a simulation with slightly weaker synapses ($J^0 = 2.5$). Note that the system ends up in the fixed point with $n = 3$ and not in $n = 2$. This is due to the noise that partially destabilizes the ($n = 2$) mode and causes the network to leave this branch before the fixed point is reached.

5 Discussion

We have presented a simple model for short-term synaptic plasticity that, in conjunction with the spike response model, allows for an analytic treatment of the dynamics of a highly connected network in the presence of short-term depression or facilitation. Previous results on pulse-coupled oscillators (Mirollo & Strogatz, 1990; Kuramoto, 1991; Tsodyks, Mitkov, & Sompolinsky, 1993; Bottani, 1995; Gerstner, van Hemmen, & Cowan, 1996), are extended so as to include time-dependent synaptic weights and

arbitrary response functions. We have found that short-term depression does not affect the stability properties of a state with coherently firing neurons. Apart from transients, a network with short-term depression has the same long-term behavior as a network with static weights that are tuned to the corresponding equilibrium value of the dynamic synapses. With short-term facilitation, the stability properties depend on the slope of the $J_\infty^{\mathrm{STF}}(T)$-curve relative to the curve corresponding to the locking equation. We have performed an extensive parameter search but found no realistic parameter setting that would destabilize a solution of the locking equation that is stable in the absence of short-term facilitation. In any case the dynamics is dominated by attractive limit cycles with coherently firing neurons. This result is also confirmed by computer simulations.

In addition to the stability properties, the transient behavior of the network can be predicted as well. In the case of a slowly developing depression or facilitation, the dynamics evolves along the lines of the stable solutions of equation 3.2 in a diagram of the mean coupling strength and the mean firing period. A transition to another firing mode occurs as soon as the solution becomes unstable. Depending on the parameter values, a cascade of these mode transitions can produce a rich structure in the spike activity of the network.

Appendix

We show by means of a linear stability analysis that the stability of the coherent state in case of a rapidly evolving synaptic depression or facilitation does not depend on the details of the synaptic plasticity but is completely determined by the kernels ϵ and η, which represent postsynaptic potentials and refractory behavior. In the present case, the synaptic strength depends on only the very last interspike interval, and we define for short-term depression

$$J(t; t_{n-1}, t_{n-2}, \ldots) = J(t - t_{n-1}) = J^0 \left[1 - R \exp(-(t - t_{n-1})/\tau)\right], \quad \text{(A.1)}$$

or, for short-term facilitation,

$$\begin{aligned} J(t; t_{n-1}, t_{n-2}, \ldots) &= J(t - t_{n-1}) \\ &= J^0[A_0 + (1 - A_0)R \exp(-(t - t_{n-1})/\tau)]. \end{aligned} \quad \text{(A.2)}$$

We assume that the neurons fire in perfect synchrony up to time $t = 0$. At $t = 0$ we apply some external perturbation so that neuron i will not fire at $t = 0$, as it should, but at $t = \delta_i$, with $|\delta_i/T| \ll 1$. With this setup, we calculate the resulting jitter of the firing times in the next period at $t = T$. We define δ_i' to be the deviation of the firing time of neuron i from $t = T$. If $|\delta_i'| < |\delta_i|$ for all i, the coherent state is said to be stable.

In order to determine δ_i' we note that the local field of neuron i crosses the threshold ϑ at time $t = T + \delta_i'$,

$$h_i(T+\delta_i) = \sum_{j=1}^{N}\left[J(T+\delta_i)\epsilon(T+\delta_i'-\delta_j-\Delta) + \sum_{k=2}^{\infty} J(T)\epsilon(kT+\delta_i'-\Delta)\right] + \eta(T+\delta_i'-\delta_i) + \sum_{k=2}^{\infty}\eta(\delta_i'+kT) = \vartheta. \tag{A.3}$$

We linearize with respect to δ and δ', use $h_i(0) = \vartheta$, and obtain after short calculation

$$\delta_i' = \frac{\eta'(T)\,\delta_i + [J(T)\,\epsilon'(T-\Delta) - J'(T)\,\epsilon(T-\Delta)]\,\sum_{j=1}^{N}\delta_j/N}{\sum_{k=1}^{\infty}[J(T)\,\epsilon'(kT-\Delta) + \eta'(kT)]}, \tag{A.4}$$

where, except for δ_i', primes denote a derivative with respect to the argument.

The result, equation A.4, can be interpreted easily if we assume $\eta'(kT)$ and $\epsilon'(kT-\Delta)$ to vanish for $k > 1$. Furthermore, we assume $\sum_{j=1}^{N}\delta_j/N = 0$, which is a consequence of the strong law of large numbers if the network is sufficiently large and perturbations are random variables with zero mean. Then the deviation of the next firing time from $t = T$ equals

$$\delta_i' = \frac{\eta'(T)}{\epsilon'(T-\Delta)+\eta'(T)}\,\delta_i\,. \tag{A.5}$$

It is less than the initial perturbation δ_i, if $\epsilon'(T-\Delta) > 0$. This condition is the well-known result of the locking theorem proved by Gerstner et al. (1996).

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