Dynamic Stochastic Synapses as Computational Units

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Abstract

In most neural network models, synapses are treated as static weights that change only on the slow time scales of learning. It is well known, however, that synapses are highly dynamic, and show use-dependent plasticity over a wide range of time scales. Moreover, synaptic transmission is an inherently stochastic process: a spike arriving at a presynaptic terminal triggers release of a vesicle of neurotransmitter from a release site with a probability that can be much less than one.

We consider a simple model for dynamic stochastic synapses that can easily be integrated into common models for networks of integrate-and-fire neurons ("spiking neurons"). The parameters of this model have direct interpretations in terms of synaptic physiology. We investigate the consequences of the model for computing with individual spikes, and demonstrate through rigorous theoretical results that the computational power of the network is increased through the use of dynamic synapses.

1 Introduction

In most neural network models, neurons are viewed as the only computational units, while the synapses are treated as passive scalar parameters ("weights"). It has, however, long been recognized (see for example

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[Katz, 1966, Magleby, 1987, Zucker, 1989, Zador and Dobrunz, 1997]) that biological synapses can exhibit rich temporal dynamics. These dynamics may have important consequences for computing and learning in biological neural systems.

There have been several previous studies of the computational consequences of dynamic synapses. [Little and Shaw, 1975] investigate a synapse model described in [Katz, 1966] for the neuromuscular junction, and describe possible applications for memory tasks. [Abbott et al., 1997] show that use-dependent depression of synapses can implement a form of dynamic gain control. [Tsodyks and Markram, 1997] and [Markram and Tsodyks, 1997] propose that dynamic synapses may support a transition from rate coding to temporal coding. [Liaw and Berger, 1996] investigate a network model that involves dynamic synapses from an excitatory neuron to an inhibitory neuron, which sends feedback directly to the presynaptic terminals. They show through computer simulations tuning the relative contributions of excitatory and inhibitory mechanisms can selectively increase the network output cross-correlation for certain pairs of temporal input patterns (speech waveforms). On a more abstract level [Back and Tsoi, 1991] and [Principe, 1994] investigate possible uses of filter-like synapses for processing time series in artificial neural networks .

These previous models were based on data obtained from studies of populations of peripheral or central release sites[1]. Experimental data on the temporal dynamics of individual release sites in the central nervous system have only recently become available [Dobrunz and Stevens, 1997, Murthy et al., 1997]. In this article we investigate a model for the temporal dynamics of single release sites motivated by these findings. In this model, synapses either succeed or fail in releasing a neurotransmitter-filled vesicle, and it is this probability of release that is under dynamic control. The parameters of the resulting stochastic synapse model have an immediate interpretation in terms of synaptic physiology, and hence provide a suitable framework for investigating possible computational consequences of changes in specific parameters of a biological synapse. After the presentation of this model in section 2, we will analyze computational consequences of this model in section 3. We focus here on computations on short spike trains, which have not been addressed in the previous literature.

[1] At the neuromuscular junction, each synapse contains thousands of release sites. In the cortex, pairs of neurons are typically connected by multiple release sites, although the multiplicity is lower [Markram, 1997]. By contrast, synapses from hippocampal region CA3 to region CA1 pyramidal neurons are often mediated by a single release site [Harris and Stevens, 1989].

2 A Model for the Temporal Dynamics of a Single Synapse

Single excitatory synapses in the mammalian cortex to exhibit a *binary* responses: at each release site, either zero or one neurotransmitter-filled vesicles is released in response to spike from the presynaptic neuron. When a vesicle is released, its contents cross the synaptic cleft and open ion channels in the postsynaptic membrane, thereby creating an electrical pulse in the postsynaptic neuron. The *probability* $p_S(t_i)$ that a vesicle is released by a synapse S varies systematically with the precise timing of the spikes t_i in spike train; the mean size of the postsynaptic response, by contrast, does not vary in a systematic manner for different spikes in a spike train from the presynaptic neuron [Dobrunz and Stevens, 1997]. Moreover, the release probability varies among different release sites; that is, release probability is heterogenous ([Hessler et al., 1993, Rosenmund et. al, 1993, Allen and Stevens, 1994, Manabe and Nicoll, 1994, Bolshakov and Siegelbaum, 1995, Markram and Tsodyks, 1996, Ryan et al., 1996, Stratford et al., 1996, Castro-Alamancos and Connors, 1997, Dobrunz and Stevens, 1997, Murthy et al., 1997]).

We represent a spike train as a sequence $\underline{t}$ of firing times, i.e. as increasing sequences of numbers $t_1 < t_2 < \ldots$ from $\mathbf{R}^+ := \{z \in \mathbf{R} : z \geq 0\}$. For each spike train $\underline{t}$ the output of a synapse S consists of the sequence $S(\underline{t})$ of those $t_i \in \underline{t}$ on which vesicles are "released" by S , i.e. of those $t_i \in \underline{t}$ which cause an excitatory or inhibitory postsynaptic potential (EPSP or IPSP, respectively). The map $\underline{t} \rightarrow S(\underline{t})$ may be viewed as a stochastic function that is *computed* by synapse S . Alternatively one can characterize the output $S(\underline{t})$ of a synapse S through its *release pattern* $\underline{q} = q_1 q_2 \ldots \in \{R, F\}^*$, where R stands for release and F for failure of release. For each $t_i \in \underline{t}$ one sets $q_i = R$ if $t_i \in S(\underline{t})$, and $q_i = F$ if $t_i \notin S(\underline{t})$.

The central equation in our dynamic synapse model gives the probability $p_S(t_i)$ that the i^{th} spike in a presynaptic spike train $\underline{t} = (t_1, \ldots, t_k)$ triggers the release of a vesicle at time t_i at synapse S,

$$p_S(t_i) = 1 - e^{-C(t_i) \cdot V(t_i)} \ . \tag{1}$$

The release probability is assumed to be nonzero only for $t \in \underline{t}$, so that releases occur only when a spike invades the presynaptic terminal (*i.e.* the spontaneous release probability is assumed to be zero). The functions $C(t) \geq 0$ and $V(t) \geq 0$ describe, respectively, the states of facilitation and depletion at the synapse at time t .

The dynamics of facilitation are given by

$$C(t) \;=\; C_0 + \sum_{t_i<t} c(t-t_i) \;, \tag{2}$$

where C_0 is some parameter ≥ 0 that can for example be related to the resting concentration of calcium in the synapse. The exponential response function $c(s)$ models the response of $C(t)$ to a presynaptic spike that had reached the synapse at time $t-s$: $c(s) = \alpha \cdot e^{-s/\tau_C}$, where the positive parameters τ_C and α give the decay constant and magnitude, respectively, of the response. The function C models in an abstract way internal synaptic processes underlying presynaptic facilitation, such as the concentration of calcium in the presynaptic terminal. The particular exponential form used for $c(s)$ could arise for example if presynaptic calcium dynamics were governed by a simple first order process.

The dynamics of depletion are given by

$$V(t) \;=\; \max(\,0\,,\; V_0 - \sum_{t_i:\, t_i<t \text{ and } t_i\in S(\underline{t})} v(t-t_i)) \;, \tag{3}$$

for some parameter $V_0 > 0$. $V(t)$ depends on the subset of those $t_i \in \underline{t}$ with $t_i < t$ on which vesicles were actually released by the synapse, i.e. $t_i \in S(\underline{t})$. The function $v(s)$ models the response of $V(t)$ to a preceding release of the same synapse at time $t-s \leq t$. Analogously as for $c(s)$ one may choose for $v(s)$ a function with exponential decay $v(s) = e^{-s/\tau_V}$, where $\tau_V > 0$ is the decay constant. The function V models in an abstract way internal synaptic processes that support presynaptic depression, such as depletion of the pool of readily releasable vesicles. In a more specific synapse model one could interpret V_0 as the maximal number of vesicles that can be stored in the readily releasable pool, and $V(t)$ as the expected number of vesicles in the readily releasable pool at time t.

In summary, the model of synaptic dynamics presented here is described by five parameters: C_0, V_0, τ_C, τ_V and α. The dynamics of a synaptic computation and its internal variables $C(t)$ and $V(t)$ are indicated in Fig. 1.

For low release probabilities, Eq. 1 can be expanded to first order around $r(t) := C(t) \cdot V(t) = 0$ to give

$$p_S(t_i) = C(t_i) \cdot V(t_i) + O([C(t_i) \cdot V(t_i)]^2). \tag{4}$$

Similar expressions have been widely used to describe synaptic dynamics for *multiple* synapses [Magleby, 1987, Markram and Tsodyks, 1996, Varela et al., 1997].

In our synapse model, we have assumed a standard exponential form for the decay of facilitation and depression (see e.g.

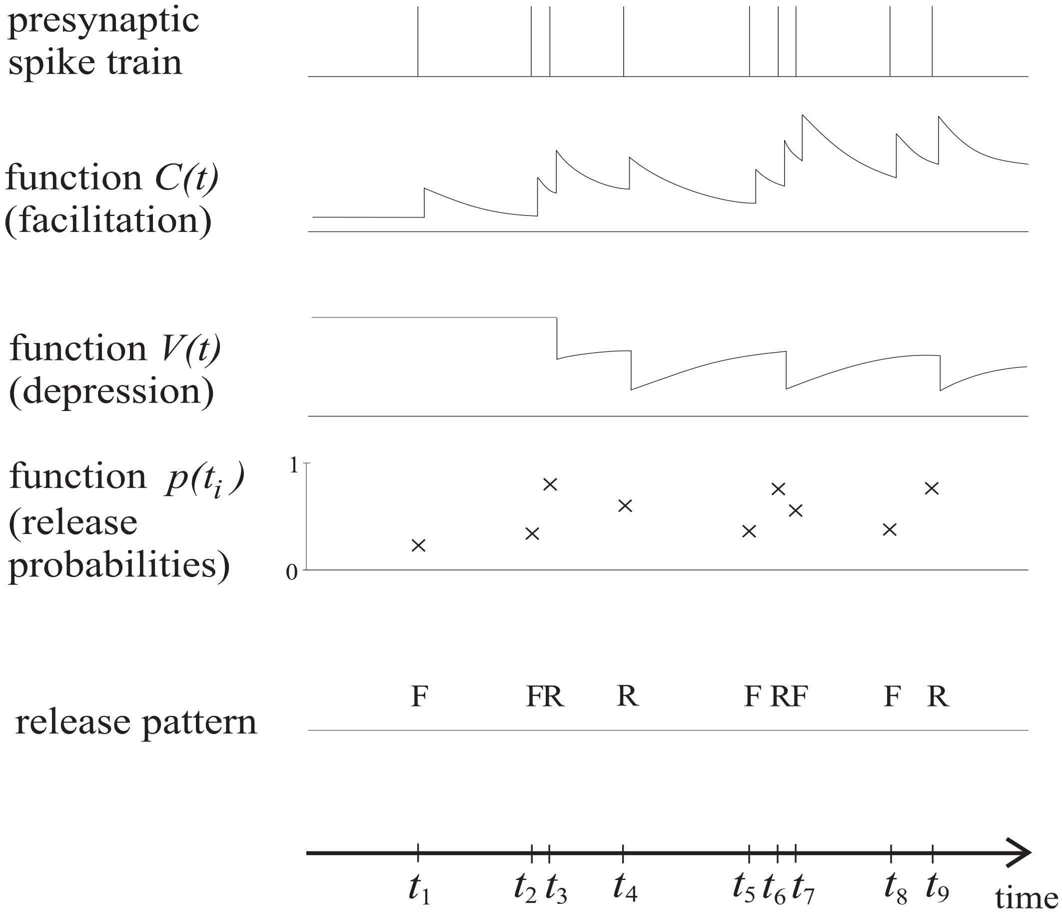

Figure 1: *Synaptic computation on a spike train* $\underline{t}$*, together with the temporal dynamics of the internal variables* C *and* V *of our model. Note that* $V(t)$ *changes its value only when a presynaptic spike causes release.*

[Magleby, 1987, Markram and Tsodyks, 1996, Dobrunz and Stevens, 1997, Varela et al., 1997]). We have further assumed a multiplicative interaction between facilitation and depletion. While this form has not been validated at single synapses, in the limit of low release probability (see Eq. 4), it agrees with the multiplicative term employed in [Varela et al., 1997] to describe the dynamics of multiple synapses.

The assumption that release at individual release sites of a synapse is binary, *i.e.* that each release site releases 0 or 1—but not more than 1—vesicle when invaded by a spike, leads to the exponential form of Eq. 1 [Dobrunz and Stevens, 1997]. We emphasize the formal distinction between *release site* and *synapse*. A synapse might in principle consist of several independent release sites in parallel, each of which has a dynamics similar to that of the stochastic "synapse model" we consider.

It is known that synaptic facilitation and depression occur on multiple time scales, from a few hundred milliseconds to hours or longer. Hence in a more complex version of our model one should replace $C(t)$ and $V(t)$ by sums of several such functions $C_j(t), V_j(t)$ with heterogenous parameters (in particular different time constants τ_{C_j}, τ_{V_j}). We refer to [Maass and Zador, 1998] for details.

3 Results

3.1 Different "Weights" for the First and Second Spike in a Train

We start by investigating the range of different release probabilities $p_S(t_1), p_S(t_2)$ that a synapse S can assume for the first two spikes in a given spike train. These release probabilities depend on $t_2 - t_1$ as well as on the values of the internal parameters $C_0, V_0, \tau_C, \tau_V, \alpha$ of the synapse S. Here we analyze the potential freedom of a synapse to choose values for $p_S(t_1)$ and $p_S(t_2)$. We show in Theorem 3.1 that the range of values for the release probabilities for the first two spikes is quite large. Furthermore Theorem 3.1 shows that a synapse loses remarkably little with regard to the dynamic range of its release probabilities for the first two spikes if it tunes only the two parameters C_0 and V_0. To prove this, we consider a "worst case" scenario, where $t_2 - t_1, \alpha, \tau_C, \tau_V$ are arbitrary given positive numbers. We prove that in spite of these "worst case assumptions" any synapse S can assume almost all possible pairs $\langle p(t_1), p(t_2)\rangle$ of release probabilities by choosing suitable values for its remaining two parameters C_0 and V_0.

The computation of the exact release probabilities $p_S(t_j)$ for the spikes t_j in a spike train $\underline{t}$ is rather complex, because the value of $V(t_j)$ (and hence the value of $p_S(t_j)$) depends on which preceding spikes $t_i < t_j$ in $\underline{t}$ were released by this synapse S. More precisely, the value of $p_S(t_j)$ depends on the release pattern $\underline{q} \in \{\mathrm{R,F}\}^{j-1}$ that the synapse had produced for the preceding spikes. For any such pattern $\underline{q} \in \{\mathrm{R,F}\}^{j-1}$ we write $p_S(t_j|\underline{q})$ for the conditional probability that synapses S releases spike t_j in $\underline{t}$, provided that the release pattern $\underline{q}$ was produced by synapse S for the preceding spike train $\underline{t}$. Thus the release probability $p_S(t_2)$ for the second spike in a spike train can be written in the form

$$p_S(t_2) = p_S(t_2|q_1 = \mathrm{R}) \cdot p_S(t_1) + p_S(t_2|q_1 = \mathrm{F}) \cdot (1 - p_S(t_1)) \; . \tag{5}$$

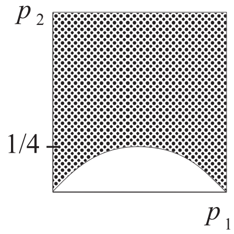

Figure 2: *The dotted area indicates the range of pairs $\langle p_1, p_2 \rangle$ of release probabilities for the first and second spike through which a synapse can move (for any given interspike interval) by varying its parameters C_0 and V_0.*

Theorem 3.1 *Let $\langle t_1, t_2 \rangle$ be some arbitrary spike train consisting of two spikes, and let $p_1, p_2 \in (0, 1)$ be some arbitrary given numbers with $p_2 > p_1 \cdot (1 - p_1)$. Furthermore assume that arbitrary positive values are given for the parameters α, τ_C, τ_V of a synapse S. Then one can always find values for the two parameters C_0 and V_0 of the synapse S so that $p_S(t_1) = p_1$ and $p_S(t_2) = p_2$.*

Furthermore the condition $p_2 > p_1 \cdot (1 - p_1)$ is necessary in a strong sense. If $p_2 \leq p_1 \cdot (1 - p_1)$ then no synapse S can achieve $p_S(t_1) = p_1$ and $p_S(t_2) = p_2$ for any spike train $\langle t_1, t_2 \rangle$ and for any values of its parameters $C_0, V_0, \tau_C, \tau_V, \alpha$.

The proof of Theorem 3.1 is given in Appendix A.1.

If one associates the current sum of release probabilities of multiple synapses or release sites between two neurons u and v with the current value of the "connection strength" $w_{u,v}$ between two neurons in a formal neural network model, then the preceding result points to a significant difference between the dynamics of computations in biological circuits and formal neural network models. Whereas in formal neural network models it is commonly assumed that the value of a synaptic weight stays fixed during a computation, the release probabilities of synapses in biological neural circuits may change on a fast time scale within a single computation.

One might use this observation as inspiration for studying a variation of formal neural network models where the values of synaptic weights may change during a computation according to some simple rule. The following fact will demonstrate that even in the case of a single McCulloch-Pitts neuron (i.e., threshold gate), this suggests an interesting new computational model. Consider a threshold gate with n inputs, that receives an input $\vec{x}\vec{y}$ of $2n$ bits in two subsequent batches $\vec{x}$ and $\vec{y}$ of n bits each. We assume that the n weights $w_1, \ldots, w_n$ of this gate are initially set to 1, and that the threshold of the gate is set to 1. We adopt the following very simple rule for changing these weights between the presentations of the two parts $\vec{x}$ and $\vec{y}$ of the input: the value of

w_i is changed to 0 during the presentation of the second part $\vec{y}$ of the input if the i-th component x_i of the first input part $\vec{x}$ was non-zero. If we consider the output bit of this threshold gate after the presentation of the second part $\vec{y}$ of the input as the output of the whole computation, this threshold gate with "dynamic synapses" computes the boolean function $F_n : \{0,1\}^{2n} \to \{0,1\}$ defined by $F_n(\vec{x}, \vec{y}) = 1 \Longleftrightarrow \exists i \in \{1, \ldots, n\}(y_i = 1 \text{ and } x_i = 0)$. One might associate this function F_n with some novelty detection task since it detects whether an input bit has changed from 0 to 1 in the two input batches $\vec{x}$ and $\vec{y}$.

It turns out that this function cannot be computed by a small circuit, consisting of just 2 or 3 "static" threshold gates of the usual type, that receives all $2n$ input bits $\vec{x}\vec{y}$ as one batch. In fact, one can prove that any feedforward circuit consisting of the usual type of "static" threshold gates, which may have arbitrary weights, thresholds and connectivity, needs to consist of at least $\frac{n}{\log(n+1)}$ gates in order to compute F_n. This lower bound can easily be derived from the lower bound from [Maass, 1997b] for another boolean function $CD_n(\vec{x}, \vec{y})$ from $\{0,1\}^{2n}$ into $\{0,1\}$ which gives output 1 if and only if $x_i + y_i \geq 2$ for some $i \in \{1, \ldots, n\}$, since $CD_n(\vec{x}, \vec{y}) = F_n(\vec{1} - \vec{x}, \vec{y})$.

3.2 Release Patterns for the First Three Spikes

In this section we examine the variety of release patterns that a synapse can produce for spike trains $t_1, t_2, t_3, \ldots$ with at least three spikes. We show not only that a synapse can make use of different parameter settings to produce different release patterns, but also that a synapse with a *fixed* parameter setting can respond quite differently to spike trains with different interspike intervals. Hence a synapse can serve as *pattern detector* for temporal patterns in spike trains.

It turns out that the structure of the triples of release probabilities $\langle p_S(t_1), p_S(t_2), p_S(t_3)\rangle$ that a synapse can assume is substantially more complicated than for the first two spikes considered in the previous section. Therefore we focus here on the dependence of the *most likely release pattern* $\underline{q} \in \{R, F\}^3$ on the internal synaptic parameters and on the interspike intervals $I_1 := t_2 - t_1$ and $I_2 := t_3 - t_2$. This dependence is in fact quite complex, as indicated in Fig. 3.

Fig. 3A shows the most likely release pattern for each given pair of interspike intervals $\langle I_1, I_2\rangle$, given a particular *fixed* set of synaptic parameters. One can see that a synapse with fixed parameter values is likely to respond quite differently to spike trains with different interspike intervals. For example even

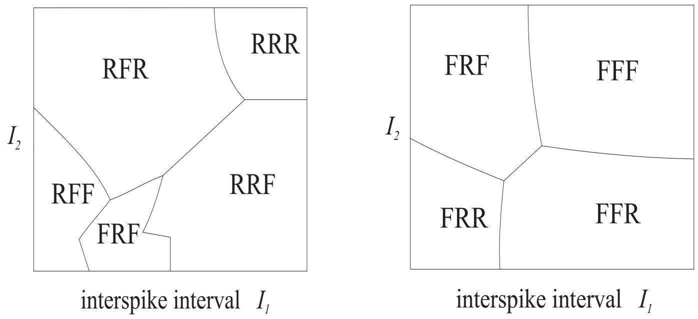

Figure 3: **(A, left)** *Most likely release pattern of a synapse in dependence of the interspike intervals I_1 and I_2. The synaptic parameters are $C_0 = 1.5$, $V_0 = 0.5$, $\tau_C = 5$, $\tau_V = 9$, $\alpha = 0.7$.* **(B, right)** *Release patterns for a synapse with other values of its parameters ($C_0 = 0.1$, $V_0 = 1.8$, $\tau_C = 15$, $\tau_V = 30$, $\alpha = 1$).*

if one just considers spike trains with $I_1 = I_2$ one moves in Fig. 3A through 3 different release patterns that take their turn in becoming the most likely release pattern when I_1 varies. Similarly, if one only considers spike trains with a fixed time interval $t_3 - t_1 = I_1 + I_2 = \Delta$, but with different positions of the second spike within this time interval of length Δ, one sees that the most likely release pattern is quite sensitive to the position of the second spike within this time interval Δ. Fig. 3B shows that a different set of synaptic parameters gives rise to a completely different assignment of release patterns.

We show in the next Theorem that the boundaries between the zones in these figures are "plastic": by changing the values of C_0, V_0, α the synapse can move the zone for most of the release patterns $\underline{q}$ to any given point $\langle I_1, I_2 \rangle$. This result provides another example for a new type of synaptic plasticity that can no longer be described in terms of a decrease or increase of the synaptic "weight".

Theorem 3.2 *Assume that an arbitrary number $p \in (0,1)$ and an arbitrary pattern $\langle I_1, I_2 \rangle$ of interspike intervals is given. Furthermore assume that arbitrary fixed positive values are given for the parameters τ_C and τ_V of a synapse S. Then for any pattern $\underline{q} \in \{R, F\}^3$ except RRF, FFR one can assign values to the other parameters α, C_0, V_0 of this synapse S so that the probability of release pattern $\underline{q}$ for a spike train with interspike intervals I_1, I_2 becomes larger than p.*

The proof of Theorem 3.2 is rather straightforward (see

[Maass and Zador, 1998]).

It was *not* claimed in Theorem 3.2 that the occurrence of the release patterns RRF and FFR can be made arbitrarily likely for any given spike train with interspike intervals $\langle I_1, I_2 \rangle$. The following Theorems show that this is in fact false.

Theorem 3.3 *The release pattern RRF can be made arbitrarily likely for a spike train with interspike intervals I_1, I_2 through suitable choices of C_0 and V_0 if and only if $e^{-I_1/\tau_V} < e^{-(I_1+I_2)/\tau_V} + e^{-I_2/\tau_V}$. In particular, the pattern RRF can be made arbitrarily likely for* any *given interspike intervals I_1, I_2 and any given value of α and τ_C if one can vary τ_V in addition to C_0 and V_0 .*

On the other hand if the values of τ_C and τ_V are fixed so that $e^{-I_1/\tau_C} \leq e^{-(I_1+I_2)/\tau_C} + e^{-I_2/\tau_C}$ and $e^{-I_1/\tau_V} \geq e^{-(I_1+I_2)/\tau_V} + e^{-I_2/\tau_V}$, then the probability of the release pattern RRF is at most 0.25 for any assignment of values to α, C_0 and V_0 .

The proof of Theorem 3.3 is given in Appendix A.2.

Theorem 3.4 *Consider some arbitrarily fixed positive value for the synapse parameter τ_C . There does not exist any pattern $\langle I_1, I_2 \rangle$ of interspike intervals for which it is possible to find values for the other synapse parameters α, C_0, V_0 and τ_V so that the release pattern FFR becomes arbitrarily likely for a spike train with interspike intervals I_1, I_2 .*

Proof: It is not possible to find for any fixed $I_1, I_2 > 0$ values for α and V_0 so that simultaneously $\alpha \cdot e^{-I_1/\tau_C} \cdot V_0$ becomes arbitrarily small and $(\alpha \cdot e^{-(I_1+I_2)/\tau_C} + \alpha \cdot e^{-I_2/\tau_C}) \cdot V_0$ becomes arbitrarily large. ∎

3.3 Burst Detection

Here we show that the computational power of a spiking (e.g. integrate-and-fire) neuron with stochastic dynamic synapses is strictly larger than that of a spiking neuron with traditional "static" synapses (cf Lisman, 1997). Let T be some given time window, and consider the computational task of detecting whether at least one of n presynaptic neurons $a_1, \ldots, a_n$ fire at least twice during T ("burst detection"). To make this task computationally feasible we assume that none of the neurons $a_1, \ldots, a_n$ fires outside of this time window.

A method for burst detection by a single neuron with dynamic synapses has been proposed [Lisman, 1997]. The new feature of Theorem 3.5 is a rigorous proof (given in Appendix A.3) that no spiking neuron with static synapses can solve this task, thereby providing a separation result for the computational power of spiking neurons with and without dynamic synapses.

Theorem 3.5 *A spiking neuron v with dynamic stochastic synapses can solve this burst detection task (with arbitrarily high reliability). On the other hand no spiking neuron with static synapses can solve this task (for any assignment of "weights" to its synapses).* [2]

3.4 Translating Interval Coding into Population Coding

Assume that information is encoded in the length I of the interspike interval between the times t_1 and t_2 when a certain neuron v fires, and that different motor responses need to be initiated depending on whether $I < a$ or $I > a$, where a is some given parameter. For that purpose it would be useful to translate the information encoded in the interspike interval I into the firing activity of populations of neurons ("population coding"). Fig. 4 illustrates a simple mechanism for that task based on dynamic synapses.

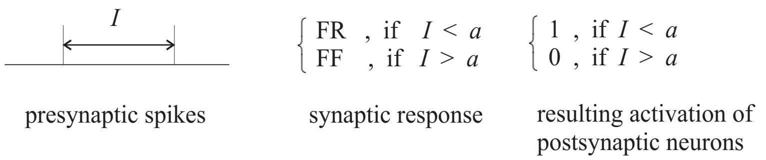

Figure 4: *A mechanism for translating temporal coding into population coding.*

The synaptic parameters are chosen so that facilitation dominates (i.e., C_0 should be small and α large) at synapses between neuron v and the postsynaptic population of neurons. The release probability for the first spike is then close to 0, whereas the release probability for the second spike is fairly large if $I < a$ and significantly smaller if I is substantially larger than a. If the resulting firing activity of the postsynaptic neurons is positively correlated with the total number of releases of these synapses, then their population response depends on the length of the interspike interval I.

[2] We assume here that neuronal transmission delays differ by less than $(n-1) \cdot T$, where by *transmission delay* we refer to the temporal delay between the firing of the presynaptic neuron and its effect on the postsynaptic target.

A somewhat related task for neural circuits is discussed in [Bugmann, 1998]. Suppose a population of neurons is to be activated Δ time steps after a preceding cue, which is given in the form of transient high firing activity of some other pool of neurons. It is not obvious how a circuit of spiking neurons can carry out this task for values of Δ that lie in a behaviourally relevant range of a few hundred msec's or longer. One possible solution is described in [Bugmann, 1998]. An alternative solution is provided with the help of depressing synapses by a variation of the previously sketched mechanism. Assume that these synapses are moved through very high firing activity of the presynaptic neurons (the "cue") to a state where their release probability is fairly low for a time period in the range of Δ. Continued moderate activity of the presynaptic neurons can then activate a population of neurons at a time difference of about Δ to the "cue".

4 Discussion

We have proposed and analyzed a general model for the temporal dynamics of single synapses that is on the one hand sufficiently complex to reflect recent experimental data, on the other hand sufficiently simple to be theoretically tractable — at least for short spike trains. The internal parameters $C_0, V_0, \tau_C, \tau_V, \alpha$ of our model have a direct interpretation in terms of the physiology of single synapses. This model thereby provides a tool for analyzing possible functional consequences of hypotheses and experimental results from synapse physiology. For example, intrasynaptic calcium dynamics and the size of the readily releasable vesicle pool are plausible candidate targets for long term plasticity. In Theorem 3.1 we show that by just changing two parameters (C_0 and V_0), a synapse can already attain the full dynamic range of release probabilities for two spikes (with arbitrary interspike interval), that could theoretically be attained by changing all 5 parameters in the synapse model. In Theorem 3.2 we show further that by tuning an additional third parameter α, corresponding to the amount of calcium that enters the presynaptic terminal upon arrival of an action potential, a synapse can be adjusted to respond to any given pattern of interspike intervals in a train of 3 spikes with a specific release pattern. On the other hand our Theorems 3.3 and 3.4 also make concrete predictions regarding the limitations of synaptic dynamics for short spike trains. Finally we have given in Theorem 3.5 a rigorous proof that dynamic synapses increase the computational power of a spiking neuron, and we have shown at the end of section 3.1 a related separation result on a more abstract level.

For longer spike trains the dynamics of the model considered in this paper becomes too complex for a rigorous theoretical analysis, but it is easy to simulate in a computer. Results of computer simulations for longer spike trains can be found in [Maass and Zador, 1998] and [Zador et al., 1998].

5 Acknowledgement

We would like to thank Harald M. Fuchs for the software used in the generation of Figure 3.

A Appendix

A.1 Proof of Theorem 3.1

We first show that the condition $p_2 > p_1 \cdot (1 - p_1)$ is a necessary condition. More precisely, we show that $p_s(t_2) > p_S(t_1) \cdot (1 - p_S(t_1))$ holds for any spike train $\langle t_1, t_2 \rangle$ and any synapse S, independently of $t_2 - t_1$, the values of its internal parameters and the precise synapse model.

This argument is very simple. One always has $C(t_2) > C(t_1)$, and in addition $V(t_2) = V(t_1)$ if the synapse does not release for the first spike. This implies that $p_S(t_2|q_1 = F) > p_S(t_1)$. Hence equation (5) implies that $p_S(t_2) \geq p_S(t_2|q_1 = F) \cdot (1 - p_S(t_1)) > p_S(t_1) \cdot (1 - p_S(t_1))$.

The proof of the positive part of Theorem 3.1 is more complex. We want to show that for *any* given pair of numbers $p_1, p_2 \in (0, 1)$ with $p_2 > p_1 \cdot (1 - p_1)$, for *any* given spike train $\langle t_1, t_2 \rangle$, and any given values of the parameters α, τ_C, τ_V of our basic synapse model one can find values for the parameters C_0 and V_0 so that

$$p_S(t_1) = p_1 \text{ and } p_S(t_2) = p_2 \ .$$

We first observe that according to equations (1) and (5) we have

$$p_S(t_1) = 1 - e^{-C_0 \cdot V_0} \tag{6}$$

and

$$\begin{aligned} p_S(t_2) = (1 - e^{-(C_0 + \alpha \cdot e^{-(t_2 - t_1)/\tau_C}) \cdot \max(0\ ,\ V_0 - e^{-(t_2 - t_1)/\tau_V})}) \cdot p_S(t_1) \\ + (1 - e^{-(C_0 + \alpha \cdot e^{-(t_2 - t_1)/\tau_C}) \cdot V_0}) \cdot (1 - p_S(t_1)) \ . \end{aligned} \tag{7}$$

Fix $\rho \in (0, \infty)$ so that $1 - e^{-\rho} = p_1$. Hence in order to achieve $p_S(t_1) = p_1$ it suffices according to equation (6) to choose values for C_0 and V_0 so that $C_0 \cdot V_0 = \rho$. If we define C_0 by

$$C_0 := \frac{\rho}{V_0}, \tag{8}$$

then the equation $C_0 \cdot V_0 = \rho$ is satisfied by any positive value of V_0. With the substitution (8) the right hand side of (7) becomes a continuous function $f(V_0)$ of the single variable V_0. We will show that this function $f(V_0)$ assumes arbitrary values in the interval $(p_1 \cdot (1 - p_1)\ ,\ 1)$ when V_0 ranges over $(0, \infty)$.

We first show that $f(V_0)$ converges to $p_1 \cdot (1 - p_1)$ when V_0 approaches 0 (and C_0 varies simultaneously according to (8). In this case the exponent in the first term on the right hand side of (7) converges to 0, and the exponent $-(C_0 + \alpha \cdot e^{-(t_2 - t_1)/\tau_C}) \cdot V_0$ in the second term converges to $-\rho$. We then exploit that $1 - e^{-\rho} = p_1$ (by definition of ρ). In the other extreme case when V_0 becomes arbitrarily large, both of these exponents converge to $-\infty$. Therefore the right hand side of (7) converges to $p_1(t_1) + (1 - p_S(t_1)) = 1$.

Finally we observe that $f(V_0)$ is a continuous function of V_0, and hence assumes for positive V_0 any value between $p_1 \cdot (1 - p_1)$ and 1. In particular $f(V_0)$ assumes the value p_2 for some positive value of V_0. ∎

A.2 Proof of Theorem 3.3

Proof: Let $\langle t_1, t_2, t_3 \rangle$ be a spike train with interspike intervals I_1, I_2 . Assume first that $e^{-I_1/\tau_V} < e^{-(I_1+I_2)/\tau_V} + e^{-I_2/\tau_V}$. We note that this condition can be satisfied for any given I_1, I_2 if τ_V is made sufficiently large relative to I_1, I_2 . Set $V_0 := e^{-(I_1+I_2)/\tau_V} + e^{-I_2/\tau_V}$. Then the probability of release for the first two spikes can be made arbitrarily large by choosing a sufficiently large value for C_0 , while the probability of release for the third spike becomes simultaneously arbitrarily small.

We now consider the consequences of the assumption that $e^{-I_1/\tau_V} \geq e^{-(I_1+I_2)/\tau_V} + e^{-I_2/\tau_V}$. If in addition $e^{-I_1/\tau_C} \leq e^{-(I_1+I_2)/\tau_C} + e^{-I_2/\tau_C}$, which can always be achieved by making τ_C sufficiently large, then this assumption implies that $p_S(t_3|q_1 = q_2 = R) \geq p_S(t_2|q_1 = R)$. Hence

$$\begin{aligned} Pr[RRF] \ &= \ p_S(t_1) \cdot p_S(t_2|q_1 = R) \cdot (1 - p_S(t_3|q_1 = q_2 = R)) \\ &\leq \ p_S(t_1) \cdot p_S(t_2|q_1 = R) \cdot (1 - p_S(t_2|q_1 = R)) \ \leq \ 0.25 \ . \end{aligned}$$

∎

A.3 Proof of Theorem 3.5

Proof: One can choose the parameters C_0 and V_0 of n excitatory synapses from $a_1, \ldots, a_n$ to v in such a way that $\alpha \cdot e^{-T/\tau_C} \cdot V_0$ is sufficiently large and $C_0 \cdot V_0$ is sufficiently small, for any given values of the other parameters of these n synapses. In this way the release pattern FR gets arbitrarily high probability for these synapses for any spike train $\langle t_1, t_2 \rangle$ with $t_2 - t_1 \leq T$.

If one sets the firing threshold of neuron v so low that it fires upon receiving at least one EPSP, then the neuron v with n dynamic synapses solves the burst detection task with arbitrarily high reliability.

In order to prove the second part of Theorem 3.5 we have to show that it is impossible to set the parameters of a spiking neuron v with n *static* synapses (and transmission delays that differ by less than $(n-1) \cdot T$) so that this neuron v can solve the same burst detection task. In order to detect whether any of the preceding neurons $a_1, \ldots, a_n$ fires at least twice during the time window of length T one has to choose the weights $w_1, \ldots, w_n$ of the synapses between $a_1, \ldots, a_n$ and v positive and so large that even two EPSP's in distance up to T with amplitude $min\{w_i : i = 1, \ldots, n\}$ reach the firing threshold of v. Since by assumption the differences in transmission delays to v are less than $(n-1) \cdot T$, there are two preceding neurons a_i and a_j with $i \neq j$ whose transmission delay differ by less than T. Hence for some single firing times of a_i and a_j during the time window of length T that we consider the resulting EPSP's arrive simultaneously at the trigger zone of v. By our preceding observation these two EPSP's together will necessarily reach the firing threshold of v, and hence cause a "false alarm". ∎

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