

# Neural Networks with Dynamic Synapses

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## Abstract

Transmission across neocortical synapses depends on the frequency of presynaptic activity (Thomson & Deuchars 1994). Inter-pyramidal synapses in layer V exhibit fast depression of synaptic transmission while other types of synapses exhibit facilitation of

transmission. To study the role of dynamic synapses in network computation, we propose a unified phenomenological model which allows computation of the post-synaptic current generated by both types of synapses when driven by an arbitrary pattern of action potential (AP) activity in a presynaptic population. Using this formalism we analyze different regimes of synaptic transmission and demonstrate that dynamic synapses transmit different aspects of the presynaptic activity depending on the average presynaptic frequency. The model also allows for derivation of mean-field equations which govern the activity of large interconnected networks. We show that dynamics of synaptic transmission results in complex sets of regular and irregular regimes of network activity.

## 1 Introduction

A marked feature of synaptic transmission between neocortical neurons is a pronounced frequency dependence of synaptic responses to trains of presynaptic spikes (Thomson & Deuchars 1994). The nature of this dynamic transmission varies between different classes of neurons. In our recent paper (Tsodyks & Markram 1996, see also Abbott *et al* 1997, Tsodyks & Markram 1997) we studied synaptic depression between neocortical pyramidal neurons with the aid of a phenomenological model. We found that the rate of depression is a primary factor in determining which features of the AP activity in the presynaptic population are most effective in driving the postsynaptic neuron.

The phenomenological formulation of (Tsodyks & Markram 1996, Abbott *et al* 1997) can be generalized to describe facilitating synapses between pyramidal cells and inhibitory

interneurons (Thomson & Deuchars 1994, Markram *et al*, 1998). This formulation has two major goals. First, it allows the quantification of the features of the AP activity of the presynaptic neurons and populations transmitted by these different types of synapses. Second, it can be used in deriving a novel mean-field dynamics of neocortical networks aimed at understanding the dynamic behavior of large neuronal populations without having to solve an equally large number of equations. Mean-field descriptions were extensively used in order to understand the possible computations of cortical neural networks (see e.g. Wilson & Cowan 1972, Amit & Tsodyks 1991, Ginsburg & Sompolinsky 1994, Tsodyks *et al* 1997). The novel formulation which uses the generalized phenomenological model of dynamic properties of synaptic connections between different types of neocortical neurons allows to study the effects of synaptic dynamics and synaptic plasticity on information processing in large neural networks.

## **2 Phenomenological model of neocortical synapses.**

In order to derive a coarse-grained description of neuronal dynamics we have to compute the post-synaptic current generated by a population of neurons with a particular firing rate. This can be done using the phenomenological model of neocortical synapses used in (Tsodyks & Markram 1997, Abbott *et al* 1997) which was shown to reproduce well the synaptic responses between pyramidal neurons. The model assumes that a synapse is characterized by

a finite amount of 'resources'. Each presynaptic spike (arriving at time  $t_{sp}$ ) activates a fraction ( $U_{SE}$ ; utilization of synaptic efficacy) of resources, which then quickly inactivate with a time constant of few milliseconds ( $\tau_{in}$ ) and recovers with a time constant of about 1 second ( $\tau_{rec}$ ). The corresponding kinetic equations read:

$$\begin{aligned}
 \frac{dx}{dt} &= \frac{z}{\tau_{rec}} - U_{SE}x\delta(t - t_{sp}) \\
 \frac{dy}{dt} &= -\frac{y}{\tau_{in}} + U_{SE}x\delta(t - t_{sp}) \\
 \frac{dz}{dt} &= \frac{y}{\tau_{in}} - \frac{z}{\tau_{rec}}
 \end{aligned} \tag{1}$$

where  $x$ ,  $y$  and  $z$  are the fractions of resources in the recovered, active and inactive states respectively. The post-synaptic current is taken to be proportional to the fraction of resources in the active state,  $I_s(t) = A_{SE}y(t)$ . The two major parameters of the model are  $A_{SE}$ , the absolute synaptic strength which can only be exhibited by activating all of the resources, and  $U_{SE}$ , which determines the dynamics of the synaptic response. For an individual synapse, the model reproduces the post-synaptic responses generated by any presynaptic spike train  $t_{sp}$  for inter-pyramidal synapses in layer V (Tsodyks & Markram 1997).

## 2.1 Modeling facilitating synapses

The formulation of Eqs. 1 does not include a facilitating mechanism, which is not evident between pyramidal neurons. It is however prominent in synapses between pyramidal neurons and inhibitory inter-neurons (Thomson & Deuchars 1994). A standard way of modeling short term facilitation is by introducing a 'facilitation factor' which is elevated by each spike by a certain amount and decays between spikes, possibly at several rates (see e.g. Mallart &

Martin 1967, Zengel & Magleby 1982). To add facilitation into our synaptic model, we therefore assume that the value of  $U_{SE}$  is not fixed but increased by a certain amount due to each presynaptic spike. The resulting model includes both facilitating and depressing mechanisms.

Increase in  $U_{SE}$  could reflect e.g. the accumulation of calcium ions caused by spikes arriving in the presynaptic terminal, which is responsible for the release of neurotransmitter (R. Bertram *et al* 1996). For a simple kinetic scheme, assume that an AP causes a fraction of  $U_{SE}$  calcium channels to open which subsequently close with a time constant of  $\tau_{facil}$ . The fraction of opened calcium channels determines the current value of  $U_{SE}$ , referred to as  $U_{SE}^1$ . The corresponding kinetic equation therefore reads:

$$\frac{dU_{SE}^1}{dt} = -\frac{U_{SE}^1}{\tau_{facil}} + U_{SE}(1 - U_{SE}^1)\delta(t - t_{sp}). \quad (2)$$

$U_{SE}$  determines the increase in the value of  $U_{SE}^1$  due to each spike and coincides with the value of  $U_{SE}^1$  reached upon the arrival of the first spike (in other words, at very low frequency of stimulation).

This equation can be transformed into an iterative expression for the value of  $U_{SE}^1$  reached upon the arrival of  $n$ -th spike in a train, which determines the post-synaptic response according to Eqs. 1:

$$U_{SE}^1(n + 1) = U_{SE}^1(n)(1 - U_{SE})exp(-\delta t/\tau_{facil}) + U_{SE} \quad (3)$$

where  $\delta t$  is the time interval between the  $n$ -th and  $(n + 1)$ -th spikes. If the presynaptic neuron emits a regular spike train at the frequency  $r$ ,  $U_{SE}^1$  reaches a steady value of

$$\frac{U_{SE}}{1 - (1 - U_{SE})\exp(-1/r\tau_{facil})}$$

Thus in this formulation,  $U_{SE}^1$  is a frequency dependent variable and  $U_{SE}$  is a kinetic parameter characterizing an activity dependent transmission in a given synapse <sup>1</sup>.

We should emphasize that facilitating and depressing mechanisms are intricately interconnected since stronger facilitation leads to higher  $U_{SE}^1$  values which in turn leads to stronger depression. The value of  $U_{SE}$  therefore determines the contribution of facilitation in generating subsequent synaptic responses. Facilitation is marked for small values of  $U_{SE}$  and is not observed for higher  $U_{SE}$ . We found that the main features of synaptic transmission between pyramidal neurons and inhibitory inter-neurons are well captured by this model with  $U_{SE} \sim 0.01 \rightarrow 0.05$  and  $\tau_{rec}$  is typically several times faster than  $\tau_{facil}$  (Markram *et al*, 1998; see also Fig. 1D). Fig. 1A,B shows responses from facilitating and depressing synapses with the same absolute strength to a regular spike train of 20Hz (but with input resistance of the facilitatory synapse's target 10 times higher). Fig. 1C illustrates the buildup of depression in facilitating synapses when they are stimulated at high frequencies. As a result, the stationary level of response exhibits a tuning curve dependence on the frequency, in agreement with experimental results (Fig. 1D).

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<sup>1</sup>One could introduce two independent parameters describing initial value and degree of facilitation of  $U_{SE}^1$ . More data is required to determine whether this is needed to accurately model facilitating synapses in neocortex

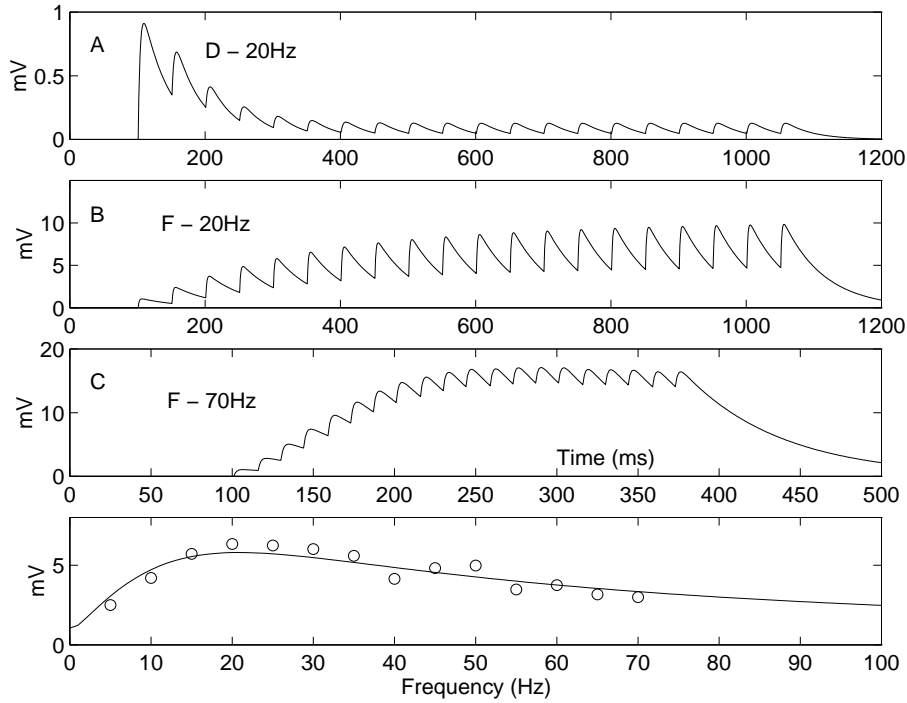


Figure 1: Phenomenological synaptic model. (A) Simulated post-synaptic potential generated by a regular spike train at a frequency of 20 Hz transmitted through a depressing synapse. (B) Same as in A for facilitating synapse. (C) Same as B but for a presynaptic frequency of 70 Hz. (D) Stationary level of EPSPs vs presynaptic frequency for facilitating synapses. Open circles - experimental results for one of the recorded synaptic connections between pyramidal neuron and inhibitory interneuron (details of experiments will be reported in Markram *et al*, 1998); solid line - model results. The post-synaptic potential is computed using a passive membrane mechanism ( $\tau_{mem} \frac{dV}{dt} = -V + R_{in} I_{syn}(t)$ ) with an input resistance of  $R_{in} = 100M\Omega$  for pyramidal target and  $1Giga\Omega$  for interneuron. Parameters: (A):  $\tau_{mem} = 40$  msec;  $\tau_{inact} = 3$  msec;  $A_{SE} = 250$  pA;  $\tau_{rec} = 800$  msec;  $U_{SE} = 0.5$ ; (BCD)  $\tau_{mem} = 60$  msec;  $\tau_{inact} = 1.5$  msec;  $A_{SE} = 1540$  pA;  $\tau_{rec} = 130$  msec;  $\tau_{facil} = 530$  msec;  $U_{SE} = 0.03$ ;

### 3 Population signal

We now go back to our original problem of signaling from a large population of presynaptic neurons. There is an infinite number of ways the neurons of a population can fire relative to each other. Analysis of neurophysiological data revealed that individual neurons *in-vivo* fire irregularly at all rates (Softky & Koch 1993), reminiscent of the so-called Poisson process. Mathematically, the Poisson assumption means that at each moment the probability for a neuron to fire is given by the value of the instantaneous firing rate and is independent of the timing of previous spikes. This assumption allows averaging the equations 1, 2 over different realizations of Poisson trains with a given rate, to obtain the new dynamics for the corresponding mean quantities (Amit & Tsodyks 1991):

$$\begin{aligned}
 \frac{d \langle x \rangle}{dt} &= \frac{1 - \langle x \rangle}{\tau_{rec}} - \langle U_{SE}^1 \rangle \langle x \rangle r(t) \\
 \frac{d \langle U_{SE}^- \rangle}{dt} &= -\frac{\langle U_{SE}^- \rangle}{\tau_{facil}} + U_{SE}(1 - \langle U_{SE}^- \rangle)r(t) \\
 \langle U_{SE}^1 \rangle &= \langle U_{SE}^- \rangle (1 - U_{SE}) + U_{SE}
 \end{aligned} \tag{4}$$

where  $r(t)$  denotes the rate of a Poisson train for the neuron at time  $t$ .  $\langle U_{SE}^- \rangle$  denotes the average value of  $U_{SE}^1$  immediately before the spike. Depressing synapses are described by the first of these equations with the fixed value of  $U_{SE}^1$  (see also Grossberg 1969 for the earlier analysis of these equations). In deriving Eqs. 4 we made a further simplification by assuming that the inactivation time constant  $\tau_{in}$  is much faster than the recovery one  $\tau_{rec}$ . This assumption is valid for inter-pyramidal synapses studied in (Markram & Tsodyks 1996) and for pyramidal - inter-neuron synapses (Markram *et al*, 1998). The evolution of

post-synaptic current can be obtained from the remaining equation for  $y$  and recalling that

$$I_s(t) = A_{SE}y(t):$$

$$\frac{d \langle y \rangle}{dt} = -\frac{\langle y \rangle}{\tau_{in}} + \langle U_{SE}^1 \rangle \langle x \rangle r(t). \quad (5)$$

which can be simplified to  $y = r\tau_{in}U_{SE}^1 \langle x \rangle$  if one is interested only in the time scale slower than  $\tau_{in}$ .

We should mention that while averaging equations 1 over different realizations of Poisson spike trains, we assumed that there is no statistical dependence between the variables  $x(t)$  and  $U_{SE}^1(t)$  and the probability of spike emission at time  $t$ . This is strictly valid only if there is no facilitation since in this case  $U_{SE}^1$  is a fixed parameter of the model and  $x(t)$ , which is a function of the spike arrival times prior to the current time, is independent of the probability of a spike at time  $t$  due to the Poisson assumption. However, if facilitation is included, both  $x(t)$  and  $U_{SE}^1(t)$  are a function of previous spikes and are thus not statistically independent. We thus performed simulations of equations 1, 2 for populations of presynaptic neurons firing Poisson spike trains with various modulations of their firing rate and compared the resulting post-synaptic current with the solution of the mean-field equations 4. We found that in all cases considered, mean-field solutions were good approximations (see e.g. Fig. 2). More detailed analysis, outlined in the Appendix, showed that mean-field approximation works because for all frequencies either  $U_{SE}^1$  or  $x$  have small coefficients of variation (CV) and thus the effect of the statistical correlations between them is small.

Equations 4, 5 can be solved analytically for an arbitrary modulation of the firing rates of the presynaptic population. In the case of depressing synapses, the solution takes a particular

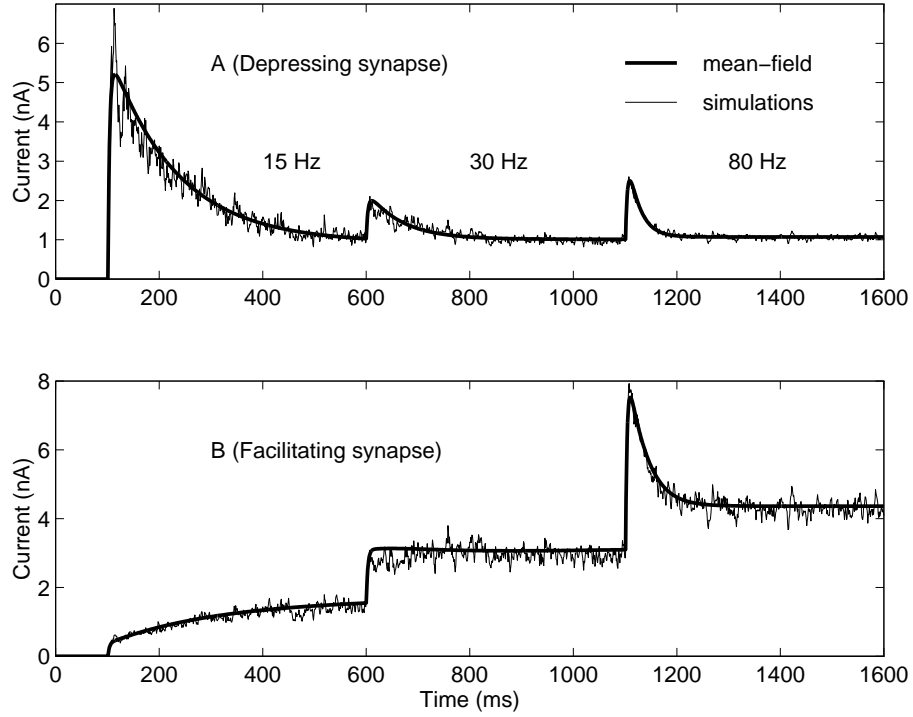


Figure 2: Post-synaptic current, generated by Poisson spike trains of a population of 1000 neurons with synchronous transitions from 0 Hz to 15 Hz to 30 Hz and then to 80 Hz, transmitted through facilitating (A) and depressing (B) synapses. Thick line - solution of mean-field equations 4, 5; thin line - simulations of 1000 spike trains with the use of the full model (Eqs. 1, 2). Parameters are the same as in Fig. 1 with  $A_{SE} = 250$  pA;

simple form:

$$\langle y(t) \rangle = U_{SE} r(t) \int_{-\infty}^t dt' \exp\left(-\frac{t-t'}{\tau_{rec}} - \int_{t'}^t dt'' U_{SE} r(t'')\right) \quad (6)$$

We use this equation to determine which features of the presynaptic AP train are transmitted by depressing synapses to their targets. Assuming that the presynaptic frequency changes gradually, one can write down the expansion over the derivatives of the frequency. The first two terms of this expansion are

$$\frac{r}{1 + rU_{SE}\tau_{rec}} + r' \frac{r}{(1 + rU_{SE}\tau_{rec})^3} + \dots \quad (7)$$

This expression describes the relative contribution of rate and temporal signaling in generating the postsynaptic response. The first term depends on the current rate which is dominant for frequencies which are small compared to the *limiting* frequency  $\lambda \sim 1/(U_{SE}\tau_{rec})$ . As the frequency increases this term saturates and thus progressively less rate signaling is possible. The main contribution at higher frequencies therefore comes from a transient term reflecting the changes in frequencies. In the context of population signaling, this means that only synchronous transitions in the population activity can be signaled to the postsynaptic neuron (Tsodyks & Markram 1997).

The solution of the full set of equations 4 for facilitating synapse has the same form as Eq. 7 with the single complication due to the fact that  $U_{SE}^1$  is now a functional of the frequency

$$U_{SE}^1 = U_{SE} \int_{-\infty}^t dt' r(t') \exp\left(-\frac{t-t'}{\tau_{facil}} - U_{SE} \int_{t'}^t dt'' r(t'')\right) \quad (8)$$

which has to be substituted in the Eq. 7. One could still analyze the qualitative features of this solution by noting that at very high frequencies,  $U_{SE}^1 \rightarrow 1$  and thus facilitating synapses behave in the same way as depressing ones, transmitting the information about the rate transitions. As the frequency decreases towards the 'peak frequency' (see Fig. 1D)

$$\theta = 1/\tau_{facil} + \sqrt{2/\tau_{facil}^2 + \frac{1 + U_{SE}}{U_{SE}\tau_{rec}\tau_{facil}}} \approx 1/\sqrt{U_{SE}\tau_{rec}\tau_{facil}}, \quad (9)$$

the presynaptic rate dominates in the postsynaptic response. The reason for this is that at this frequency facilitating and depressing effects compensate each other and the average amplitude of EPSP, which is  $\sim xU_{SE}^1$ , is approximately constant. At even smaller frequencies depressing effects become less relevant since  $x$  recovers to almost unity between the subsequent spikes. In this regime the postsynaptic signal mainly reflects the current value of rate amplified by the value of  $U_{SE}^1$ :

$$I_s \sim r(t) \int_{-\infty}^t dt' r(t') \exp(-(t - t')/\tau_{facil}) \quad (10)$$

The integral in this equation is roughly equal to the number of spikes emitted by the presynaptic neuron in the preceding time window of  $\tau_{facil}$ . In this regime, postsynaptic response is a delayed and amplified transformation of the presynaptic frequency.

As an example, we show in Fig. 2 the post-synaptic current resulting from a series of transitions in the firing rate for both depressing and facilitating synapses. All three regimes of transmission via facilitating synapses are illustrated in Fig. 2B.

## 4 Mean field network dynamics

The analysis of the previous section allows the formulation of a closed system of equations for the dynamics of a large network consisting of subpopulations of neurons with uniform connections. Each population could describe a cortical column which consists of neurons with similar receptive field properties. At this stage we assume that at each cortical location there are only two subpopulations of cortical neurons, comprised of pyramidal excitatory neurons and inhibitory inter-neurons. The coarse grained equations, describing the firing rates of these populations, have the same form as in (Wilson & Cowan 1972, Amit & Tsodyks 1991):

$$\begin{aligned}\tau_e \frac{dE_r}{dt} &= -E_r + g\left(\sum_{r'} J_{rr'}^{ee} y_{r'}^{ee} - J_{rr'}^{ei} y_{r'}^{ei} + I_r^e\right) \\ \tau_i \frac{dI_r}{dt} &= -I_r + g\left(\sum_{r'} J_{rr'}^{ie} y_{r'}^{ie} - J_{rr'}^{ii} y_{r'}^{ii} + I_r^i\right),\end{aligned}\tag{11}$$

where  $E_r$  ( $I_r$ ) is the firing rate of excitatory (inhibitory) populations located at the site  $r$ ;  $g(x)$  is a response function usually assumed to be monotonously increasing;  $J_{rr'}^{ee}$  denotes the absolute strength of the synaptic connection between excitatory neurons in the populations located at  $r$  and  $r'$  times the average number of such connections per one post-synaptic neuron, correspondingly for other interactions. Finally,  $I_r^e$  ( $I_r^i$ ) is the external input to the excitatory (inhibitory) population.  $y_{rr'}^{ee}$  (and corresponding values for all other synapses) has to be computed from the equations 4, 5 for each connection  $rr'$  with the corresponding set of kinetic parameters. Refractoriness of the neurons was ignored for simplicity.

These equations reduce to the ones of Wilson and Cowan (1972) if synaptic transmission is frequency independent, in which case  $x_r \equiv 1$  and hence  $y_r \sim E_r$ . In the presence of frequency

dependence they include effects of ever-changing synaptic efficacy due to depression and facilitation. This formulation allows for an analysis of the behavior of the network with any pattern of connections and external inputs. Since the goal of the paper is not to consider any particular computational model, we limit ourselves to two examples.

## 4.1 Network of one population

As the most simple example we consider a network which consists of only one population of excitatory neurons. Already in this case synaptic depression makes the network dynamics nontrivial. Equations 11 reduce to

$$\begin{aligned}\tau \frac{dE}{dt} &= -E(t) + g(JU_{SE}x(t)E(t)) \\ \frac{dx}{dt} &= -U_{SE}E(t)x(t) + \frac{1-x(t)}{\tau_{rec}}\end{aligned}\tag{12}$$

For convenience, the factor of  $\tau_{in}$  (see Eq. 5) was absorbed in the definition of  $J$ . One can solve these equations for the fixed point, where it simplifies into

$$E = g\left(JU_{SE} \frac{E}{1 + EU_{SE}\tau_{rec}}\right)\tag{13}$$

and can be illustrated using the graphical method (Fig. 3A). The rhs of Eq. 13 always saturates for arbitrary response functions due to synaptic depression. The system will therefore have a nontrivial fixed point with  $E > 0$  even in cases where without depression there is no stable solution.

The stability of the fixed point solution can be analyzed from Eqs. 12. The solution is stable if the following matrix has eigenvalues with negative real parts:

$$\begin{pmatrix} \frac{\beta J U_{SE} x^* - 1}{\tau} & \frac{\beta J U_{SE} E^*}{\tau} \\ -U_{SE} x^* & -U_{SE} E^* - \frac{1}{\tau_{rec}} \end{pmatrix} \quad (14)$$

where  $E^*$  and  $x^*$  are the values of  $E$  and  $x$  at the fixed point,  $\beta = g'(J U_{SE} x^* E^*)$ . For a linear-threshold gain function ( $\beta = const$ ) as in Fig. 3A, the phase diagram of the system is shown in Fig. 3B. For a given threshold, a fixed point solution appears when the synaptic strength exceeds the first critical value shown by the lower line on the diagram. Contrary to what one would expect from Fig. 3A, this solution remains unstable until the synaptic strength grows above a second critical value (upper line).

Even if the fixed point solution is stable, the system exhibits damped oscillations before reaching the steady state due to synaptic dynamics (Fig. 3C).

## 4.2 Network of two interconnected populations

This system was analyzed in (Wilson & Cowan 1972) for the case of linear synapses. They showed that if the external inputs are fixed, mean-field equations have two basic types of stable solutions - fixed points and limit cycles with the period on the order of  $\tau_e, \tau_i$ . In the present case, the mean-field equations have a much richer set of solutions because in addition to the pair of equations for  $E$  and  $I$  (Eq. 11) they also include dynamic equations for synaptic efficacies (Eq. 4). As a result, in addition to fixed points and simple limit cycles, the system exhibits a variety of rhythmic and irregular solutions which dominate the network behavior but are difficult to analyze in a completely general manner. Two particular novel solutions, one periodic and another irregular, are shown in Fig. 4A, B.

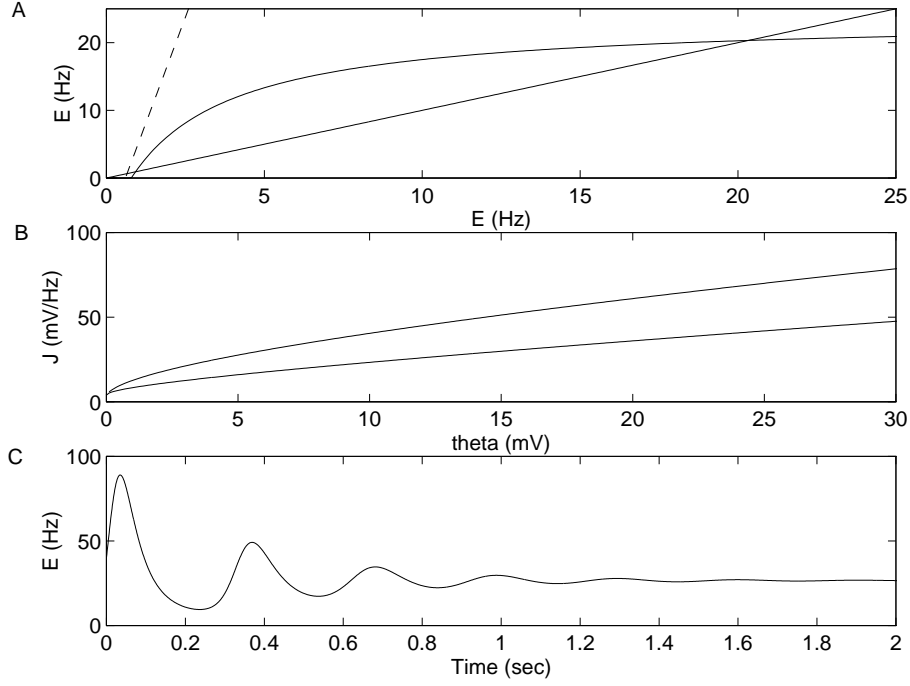


Figure 3: Solution of Eqs. 12 for the network of one excitatory population with homogeneous connections. (A): Graphical solution of the fixed point equation (13). The response function had a linear-threshold shape (dashed line) with the threshold  $\theta$  and slope  $\beta$ . The fixed point solution is given by the intersection of the two solid lines. (B) Phase diagram of the system in the space of  $\theta$  and  $J$ . (C) The solution of the dynamic equations (12). Parameters in (A) and (C):  $\theta = 15 \text{ mV}$ ;  $\beta = 0.5 \text{ mV}^{-1}\text{Hz}$   $J = 60 \text{ mV}\cdot\text{Hz}^{-1}$ ;  $U_{SE} = 0.5$ ;  $\tau_{rec} = 800\text{msec}$ ;  $\tau = 30\text{msec}$ .

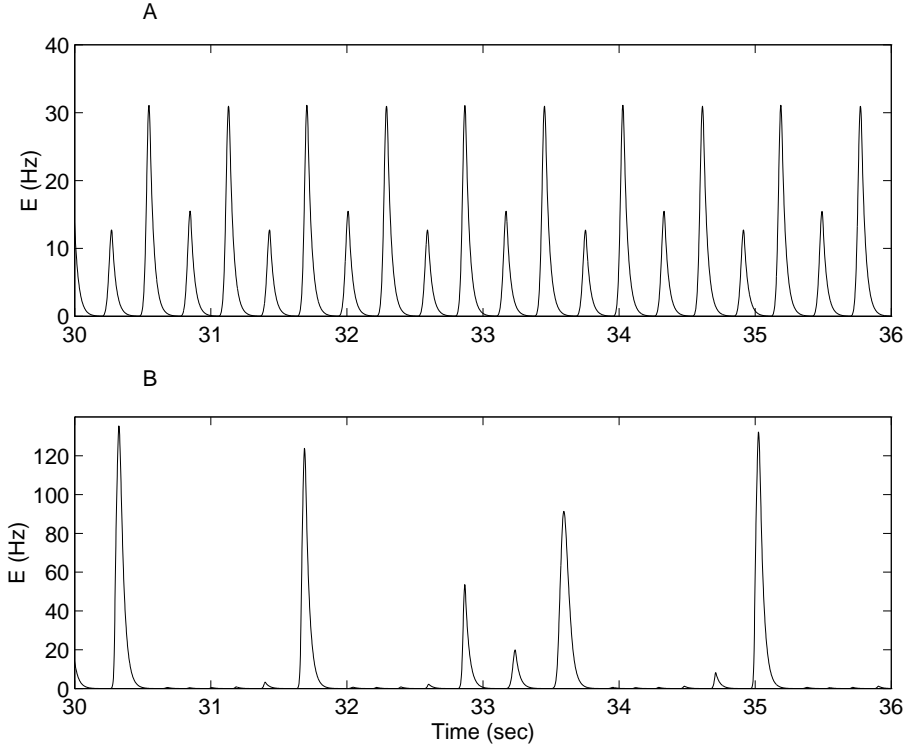


Figure 4: Solutions of Eqs. 11 for the network of two populations with homogeneous connections. (A): Population activity  $E(t)$  for the parameters  $I^e = 17$  mV;  $I^i = 15$  mV;  $J_{ee} = 50$  mV\*Hz $^{-1}$ ;  $J_{ei} = 40$ ;  $J_{ie} = 70$ ;  $J_{ii} = 19.5$ ;  $U_{SE} = 0.5$  (ee and ei);  $U_{SE} = 0.05$  (ie); 0.03 (ii);  $\tau_{rec} = 800$  msec (ee and ei); 600 msec (ie); 850 msec(ii);  $\tau_{facil} = 1000$  msec (ie); 400 msec (ii);  $\tau_e = 30$  msec;  $\tau_i = 40$  msec. (B): The same as in A but with  $J_{ii} = 0$ . The gain functions for both populations have the same form as in Fig. 3.

## 5 Conclusion

In this study we introduce a phenomenological model of synapse which allows computation of the post-synaptic responses generated by either facilitating or depressing synapses for an arbitrary train of presynaptic spikes. The model was used to define the signals that can be transmitted by these synapses and we show that signaling through these two types of synapses is fundamentally different at low firing rates but becomes more similar as the firing rate grows.

The model was also used to test the validity of the derivation of self-consistent mean-field equations for the dynamic behavior of large neural networks with arbitrary architecture of external inputs and internal interactions. The formalism was illustrated by considering two simple examples of networks consisting of one and two uniform populations of neurons. The purely excitatory network was shown to always possess a fixed point solution which can have arbitrary small firing rates. Adding an inhibitory population greatly increases the repertoire of behaviors, including the irregular sequence of population bursts of various amplitudes. Synaptic dynamics could therefore be an important factor in generating different states of cortical activity as reflected by EEG recordings.

An important challenge for the proposed formulation remains in analyzing the influence of the synaptic dynamics on the performance of other, computationally more instructive neural network models. Work in this direction is currently in progress.

## 6 Acknowledgments

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## 7 Appendix

The mean-field description of the section 3 was derived by adopting an approximation

$$\langle xU_{SE}^1 \rangle = \langle x \rangle \langle U_{SE}^1 \rangle . \quad (15)$$

The relative error of this approximation can be estimated using the Cauchy-Schwarz inequality of the probability theory:

$$\frac{|\langle xU^1 \rangle - \langle x \rangle \langle U^1 \rangle|}{\langle x \rangle \langle U^1 \rangle} \leq CV_x CV_U \quad (16)$$

where  $CV_x$  ( $CV_U$ ) stay for the coefficient of variation of the random variable  $x$  ( $U^1$ ); we use  $U^1$  instead of  $U_{SE}^1$  for brevity. Intuitively, this inequality states that if one of the random variables has a small CV, it's correlations with the other variables can be neglected. We can now use Eq. 2 and compute the CV of the  $U_{SE}^1$  for any presynaptic rate  $r$ . In the steady state, the result of computation is

$$CV_U^2 = \frac{r\tau_{facil}(1-U)^2}{2(1+r\tau_{facil})^2(1+Ur\tau_{facil}(1-U/2))} \quad (17)$$

$CV_x$  can be computed from Eqs. 1, again assuming the condition 15:

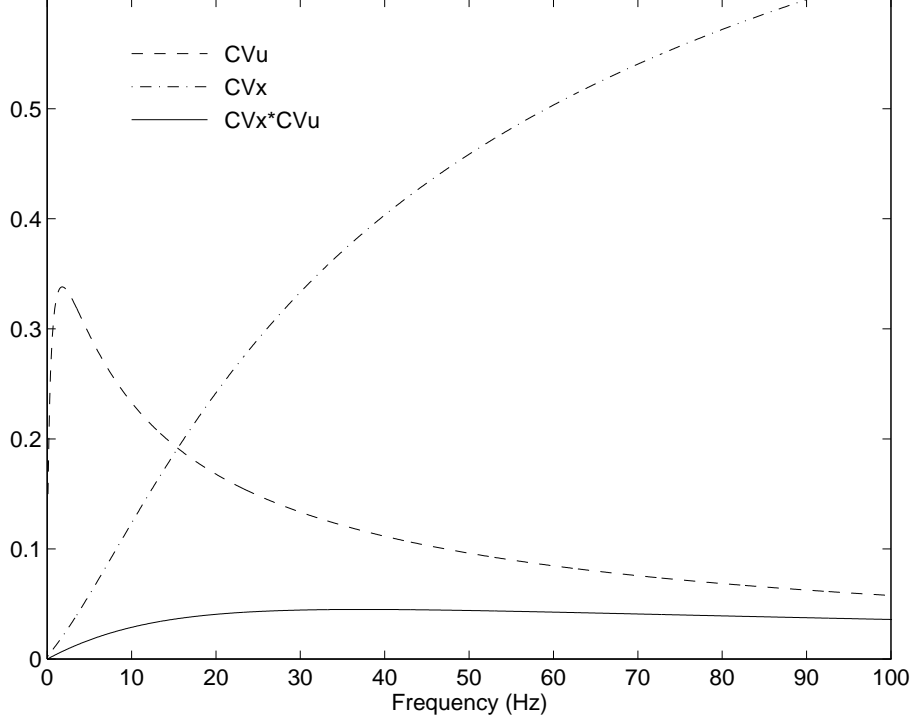


Figure 5: Coefficients of variation of  $U_{SE}^1$ ,  $x$ , as well as their product, as a function of presynaptic frequency. Parameters are the same as in Fig. 1B,C,D.

$$CV_x^2 = \frac{r\tau_{rec} \langle (U^1)^2/2 \rangle}{1 + r\tau_{rec} \langle U^1(1 - U^1/2) \rangle} \quad (18)$$

The self-consistency of the mean field theory can now be checked by plotting the product of  $CV_U$  and  $CV_x$  as a function of frequency (Fig. 5). The graph shows that for the set of parameters used in modeling facilitating synapses, derived from experimental traces, the relative error of Eq. 15 does not exceed 5 percent for any frequency. More detailed analysis of Eqs. 17,18 indicates that this error can only exceed a 10 percent level at the significantly shorter  $\tau_{facil}$  and higher values of  $U$  at which the model does not exhibit facilitating behavior any more.

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