THEORETICAL NEUROSCIENCE I

Lecture 6: Synaptic conductances

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Content

Metric system reminder

$$milli = 10^{-3} \qquad micro = 10^{-6} \qquad nano = 10^{-9}$$

$$pico = 10^{-12} \qquad femto = 10^{-15} \qquad atto = 10^{-18}$$

1 Synaptic events

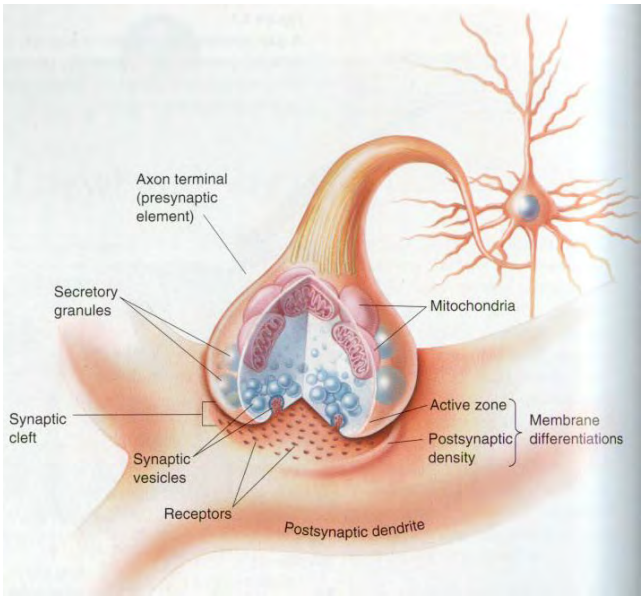

Figure 1: Synaptic components [1]

Presynaptic events

Spike depolarizes presynaptic terminal

$\downarrow$

voltage-dependent Ca^{2+}-channels open

$\downarrow$

$[Ca^{2+}]_{in}$ rises in presynaptic terminal

$\downarrow$

Vesicles fuse and transmitter is released

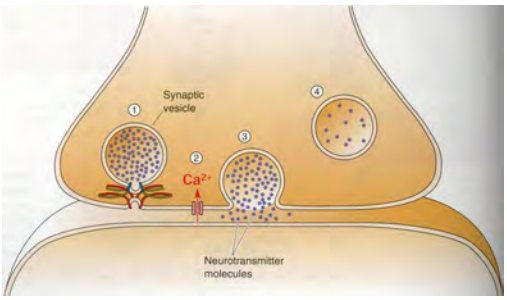

Figure 2: Synaptic interaction. [2]

Neurotransmitter molecules

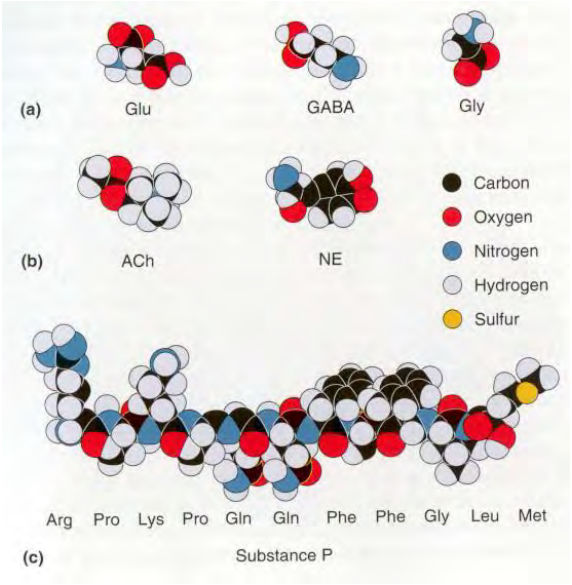

Figure 3: Molecules. [3]

Postsynaptic events

Transmitter binds to receptors in postsynaptic terminals

↓

Ion-channels open (directly or indirectly)

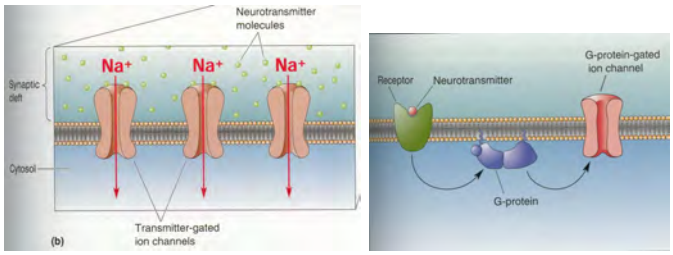

Figure 4: Ion channels [4] [5]

Ionotropic receptors control ion channels directly.
Metabotropic receptors control ion channels indirectly (G-proteins or other second messengers).
Synaptic efficacy

Changes in the strength (efficacy) of synapses are mediated by biochemical and structural changes in dendritic spines. Dendritic spines undergo genesis, elimination, and structural modification in response to stimuli.

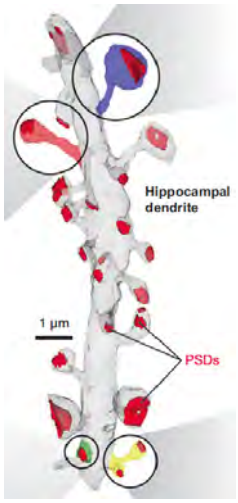

Figure 5: Hypocampal dendrite. [6]

2 Opening and closing of synaptic conductance

Both *amplitude* (efficacy) and *speed* (time-constants) of the opening and closing of postsynaptic conductances are functionally important. Biro

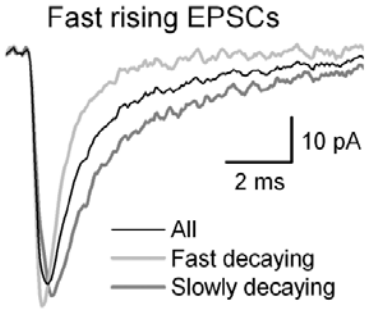

Figure 6: Fast rising EPSCs.

et al. (2005) J. Neurosci.

Kinetic model of receptor binding

In a simple model of a synapse with *ionotropic* receptors, the binding of transmitter molecules opens the channel and their unbinding closes the channel. Immediately following the release, transmitter concentration in the synaptic cleft is high. Thereafter, diffusion, degradation, and re-uptake rapidly reduce transmitter concentration.

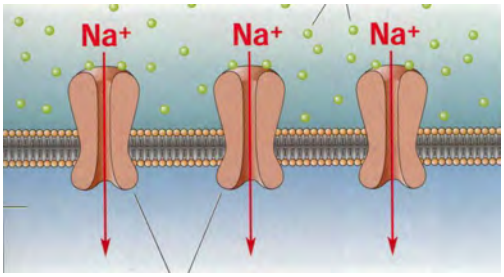

Figure 7: Sodium channels. [7]

Fractional and maximal conductance

The fractional synaptic conductance $P_s(t)$ reflects the time-course of the opening and closing of channels

$$0 \leq P_s(t) \leq 1$$

The maximal conductance $\bar{g}_s$ reflects the total number of channels

$$g_s(t) = \bar{g}_s \, P_s(t)$$

Rectangular transmitter pulse (discontinuous)

For the rising/opening phase, we assume $\alpha >> \beta$ and $P_s(0) = 0$:

$$\frac{dP_s}{dt} \approx \alpha_s \, (1 - P_s) \qquad \Rightarrow \qquad P_s(t) = 1 - e^{-\alpha \, t}$$

For the falling/closing phase, we assume $\beta >> \alpha$ and $P_s(0) = 1$:

$$\frac{dP_s}{dt} \approx -\beta_s \, P_s \qquad \Rightarrow \qquad P_s(t) = e^{-\beta \, t}$$

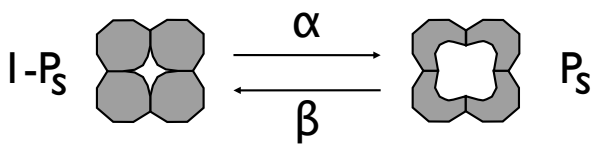

Figure 8: Rising/ opening phase.

Rectangular transmitter pulse

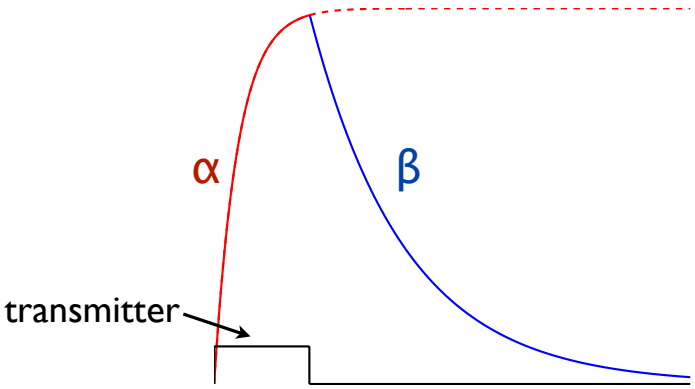

Figure 9: Alpha, Beta transmitter plots.

Difference of exponentials

A continuous formula is a difference between exponentials

$$P_s(t) = P_{max}\, B\, \left[\exp(-t/\tau_1) - \exp(-t/\tau_2)\right], \qquad t \geq 0$$

where $\tau_1 > \tau_2$ sets the *fall* time. The *rise* time is set by $\tau_1\tau_2/(\tau_1 - \tau_2)$.

Peak conductance is reached at

$$t_{peak} = \frac{\tau_1\, \tau_2}{\tau_1 - \tau_2}\, \ln \frac{\tau_1}{\tau_2}$$

B is a normalization factor which ensures $P(t_{peak}) = 1$

$$B = \left[\left(\frac{\tau_2}{\tau_1}\right)^{\frac{\tau_2}{\tau_1 - \tau_2}} - \left(\frac{\tau_2}{\tau_1}\right)^{\frac{\tau_1}{\tau_1 - \tau_2}}\right]^{-1}$$

Difference of exponentials

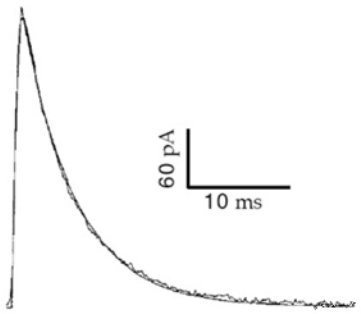

Figure 5.14: A fit of the model discussed in the text to the average EPSC (excitatory postsynaptic current) recorded from mossy fiber input to a CA3 pyramidal cell in a hippocampal slice preparation. The smooth line is the theoretical curve and the wiggly line is the result of averaging recordings from a number of trials. (Adapted from Destexhe *et al*, 1994.)

Figure 10: Difference of exponentials. [8]

Alpha function

Less cumbersome is an *alpha function*:

$$P_s(t) = P_{max}\, {}^{t}\!/\!_{\tau_s}\, \exp\left(1 - {}^{t}\!/\!_{\tau_s}\right), \qquad t \geq 0$$

where τ_s is both *rise* and *fall* time.

P_{max} is reached at $t = \tau_s$.

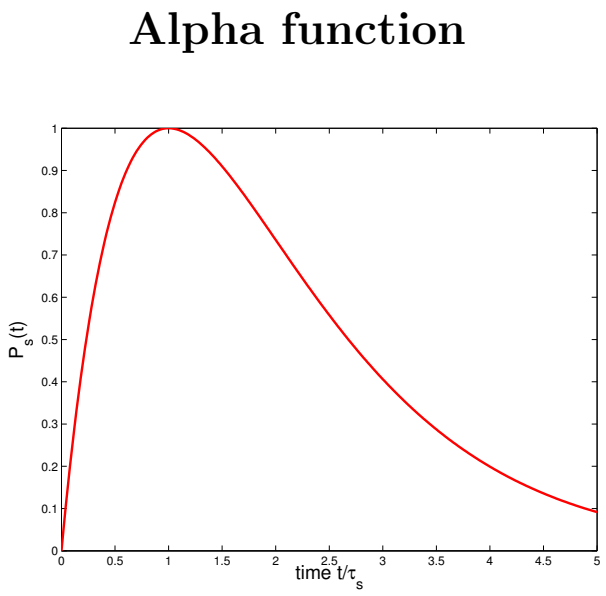

Figure 11: Alpha function.

Exponential decay

Even simpler is an *exponential decay*:

$$P_s(t) = P_{max} \exp\left(-t/\tau_s\right), \qquad t \geq 0$$

where τ_s is *fall* time.

This approximation is appropriate when the rise time can be neglected.

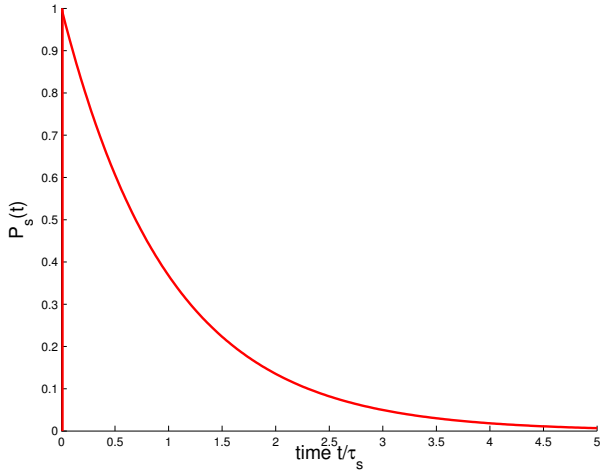

Figure 12: Exponential decay.

Synaptic conductance, current and charge

The synaptic conductance, current, and charge are related as follows:

$$g_s(t) = \bar{g}_s \, P_s(t) \qquad \text{conductance}$$

$$I_s(t) = \bar{g}_s \, P_s(t) \, (V_{rest} - E_s) \qquad \text{current}$$

$$Q_s = \int_0^\infty I_s(t) \, dt = \int_0^\infty \bar{g}_s \, P_s(t) \, (V_{rest} - E_s) \, dt \qquad \text{charge}$$

where $\bar{g}_s$ is the maximal conductance.

Approximate charge

For an exponentially decaying synapse with

$$\int_0^\infty P_s(t) \, dt = \int_0^\infty e^{-t/\tau_s} \, dt = \left[-\tau_s \, e^{-t/\tau_s} \right]_0^\infty = \tau_s$$

the total charge is approximately

$$\Delta Q = \int I_s \, dt \;=\; \int_0^\infty \left[\bar{g}_s \, P_s(t) \, (V_{ave} - E_{syn})\right] \, dt =$$

$$= \; \bar{g}_s \left[\int_0^\infty \, P_s(t) \, dt\right] \, (V_{ave} - E_{syn}) =$$

$$= \; \bar{g}_s \, \tau_s \, (V_{ave} - E_{syn})$$

conductance $\times$ *fall time* $\times$ *driving force*

Summary of opening and closing

- The effect of a synapse depends on both strength and speed.
- The speed of ionotropic synapses is governed by the kinetics of transmitter binding.
- Several functions are used to model the opening and closing of synaptic conductance.
- The total synaptic charge is approximately proportional to the maximum conductance and to the closing time.

3 GABA, AMPA, and NMDA synapses

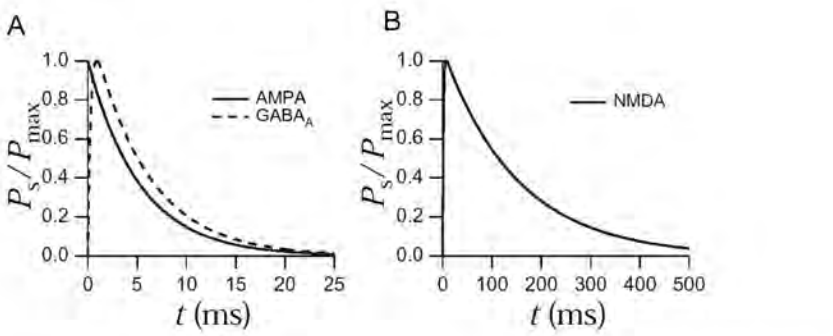

Figure 5.15: Time-dependent open probabilities fit to match AMPA, $GABA_A$, and NMDA synaptic conductances. A) The AMPA curve is a single exponential described by equation 5.31 with $\tau_s = 5.26$ ms. The $GABA_A$ curve is a difference of exponentials with $\tau_1 = 5.6$ ms and $\tau_{rise} = 0.3$ ms. B) The NMDA curve is the differences of two exponentials with $\tau_1 = 152$ ms and $\tau_{rise} = 1.5$ ms. (Parameters are from Destexhe *et al*, 1994.)

Figure 13: Synaptic conductances. [9]

Inhibitory synapses

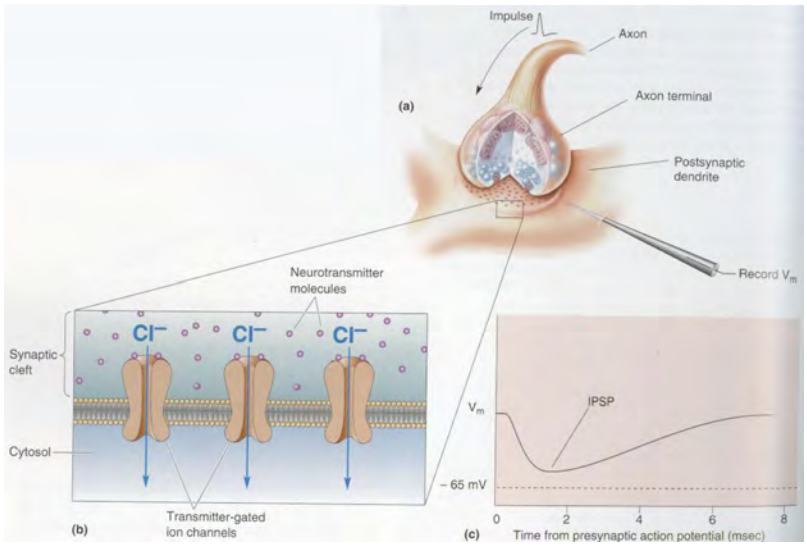

Figure 14: IPSP = inhibitory postsynaptic potential [10]

Many inhibitory synapses are activated by γ-aminobutyric acid (GABA).

GABA_A receptors are ionotropic and open a fast Cl^- channel.

GABA_B receptors are metabotropic and open a longer-lasting K^+ channel.

GABA synapses (deep cerebellar nuclei)

GABA synapses can be modeled as follows:

$$I_{GABA}(t) = g_{syn}(t)\,(V - E_{GABA}) \qquad E_{GABA} \approx -75\,mV$$

Purkinje cells ($GABA_A$)

$$g_{syn}(t) = \bar{g}_{syn}\, e^{-t/\tau}, \qquad \bar{g}_{syn} = 40\,pS, \quad \tau = 5\,ms$$

Cerebellar granule cells ($GABA_A$ and $GABA_B$)

$$g_{syn}(t) = \bar{g}_1\, e^{-t/\tau_1} + \bar{g}_2\, e^{-t/\tau_2} \qquad g_1 = 30\,pS, \quad \tau_1 = 5\,ms$$
$$g_2 = 10\,pS, \quad \tau_2 = 50\,ms$$

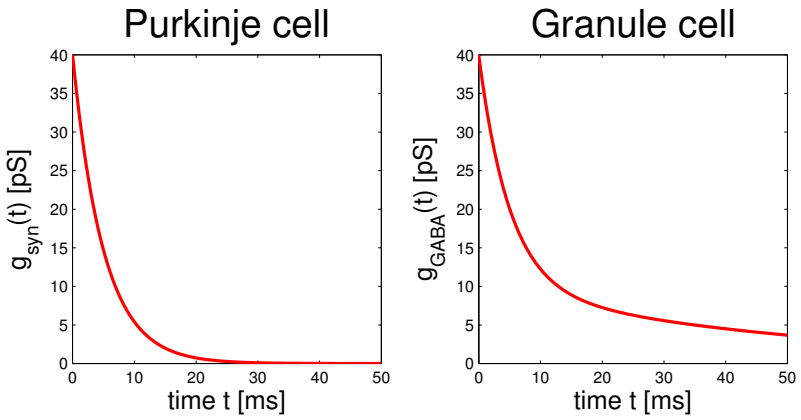

Figure 15: Purkinje vs. Granule cell.

$$\Delta Q = 40\,pS \cdot 5\,ms \cdot 10\,mV = 2\,aC \qquad 1.5\,aC + 5\,aC$$

Excitatory synapses

Many excitatory synapses use the transmitter glutamate and ionotropic receptors. These channels are typically permeable to Na^+, K^+, and Ca^{2+} and the effective reversal potential is $E_{syn}^{exc} \approx 0\,mV$.

Different types of ionotropic receptors are distinguished by the *agonists* ('substitute transmitters') to which they bind: AMPA and NMDA.

A given postsynaptic site may contain both AMPA and NMDA receptors.

AMPA synapse (deep cerebellar nuclei)

AMPA synapses may be modeled as follows:

$$I_{AMPA} = g_{AMPA}(t)\,(V - E_{AMPA}) \qquad E_{AMPA} \approx 0\,mV$$

$$g_{AMPA}(t) = \bar{g}_{AMPA}\,B\,\left[e^{-t/\tau_{decay}} - e^{-t/\tau_{rise}}\right]$$

$$\tau_{rise} = 0.09\,ms \qquad \tau_{decay} = 1.5\,ms \qquad \bar{g}_{AMPA} = 720\,pS \qquad B = 1.273$$

$$\Delta Q = 720\,pS \cdot 1.273 \cdot (1.5\,ms - 0.09\,ms) \cdot 65\,mV = 84\,aC$$

NMDA receptors

Permeable to Na^+, K^+, and Ca^{2+}, effective reversal potential $E_{syn}^{exc} \approx 0\,mV$.

Importantly, conductance also depends also on **postsynaptic** membrane potential. At resting potential, channel pore is blocked by extracellular $[Mg^{2+}]$. Depolarization clears this blockage.

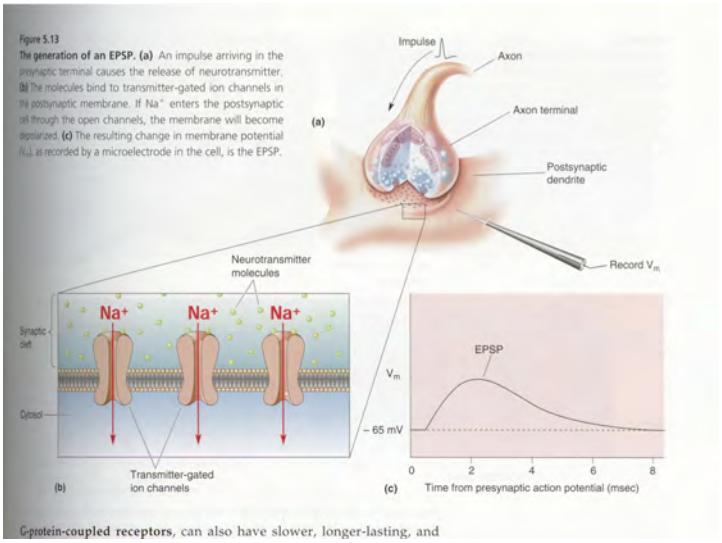

Figure 16: EPSP = excitatory postsynaptic potential. [11]

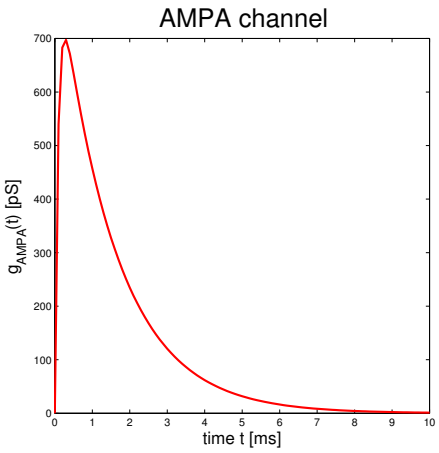

Figure 17: Ampa channel plot.

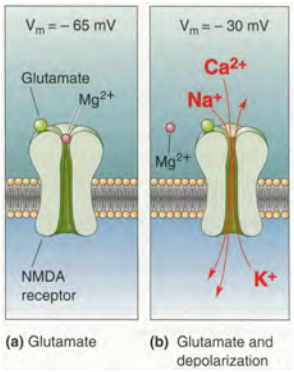

Figure 18: Glutamate channel interactions. [12]

NMDA synapse (deep cerebellar nuclei)

The combined dependence on time t, postsynaptic potential V_{post} and extracellular Mg^{2+} concentration can be modeled as follows:

$$I_{NMDA}(t) = g_{NMDA}(t)\,(V - E_{NMDA}) \qquad E_{NMDA} \approx 0\,mV$$

$$g_{NMDA}(t) = \bar{g}_{NMDA}\,B\,\left[e^{-t/\tau_{decay}} - e^{-t/\tau_{rise}}\right]\,G_{NMDA}(V_{post})$$

$$G_{NMDA}(V_{post}) = \frac{\beta}{\beta + e^{-\alpha\,V_{post}}\,\left[Mg^{2+}\right]_{out}}$$

$$\tau_{rise} = 3\,ms, \quad \tau_{decay} = 40\,ms, \quad \bar{g}_{NMDA} = 1200\,pS, \quad B = 1.358$$
$$\alpha = 0.062\,mV^{-1}, \qquad \beta = 3.57\,mM, \qquad \left[Mg^{2+}\right]_{out} = 1.2\,mM$$

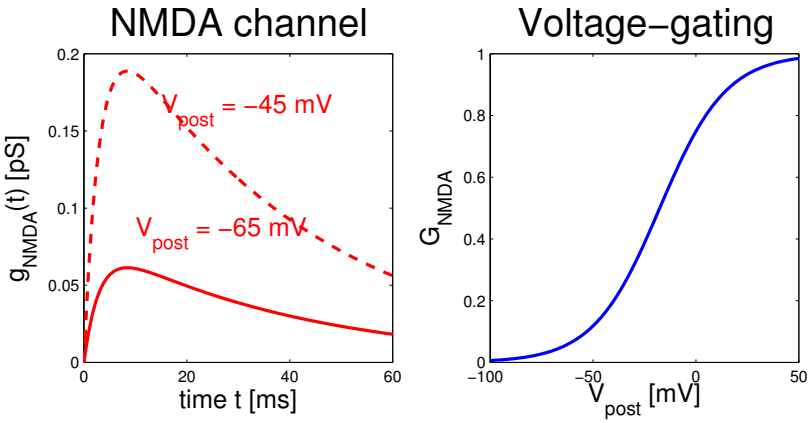

Figure 19: NMDA behaviors.

$$\Delta Q_{max} = 1200\,pS \cdot 1.358 \cdot (40\,ms - 3\,ms) \cdot 65\,mV = 3900\,aC$$

$$G_{NMDA}(-65\,mV) \approx 0.05, \qquad G_{NMDA}(-45\,mV) \approx 0.15$$

Importance of voltage gating

The fact that NMDA receptor channels require both pre- and postsynaptic activity (depolarization) is of supreme importance. It means that NMDA receptors can act as **coincidence detectors** for pre- and postsynaptic activity.

This functionality underlies *conditional* interactions, that is, the effect of one activity is conditional on another activity. The voltage-dependence of NMDA channels is also thought to play a role in synaptic modification and plasticity ("Hebbian learning").

Summary GABA, AMPA, NMDA

- We have introduced simple models for the most common synapse types.
- Inhibitory GABA_A synapses are medium fast: $\tau_{close} = 5\,ms$.
- Inhibitory GABA_B synapses also comprise a slow component: $\tau_{close} = 50\,ms$.
- Excitatory AMPA synapses are very fast: $\tau_{close} = 1.5\,ms$.
- Excitatory NMDA synapses are slow: $\tau_{close} = 50\,ms$.
- They are significantly permeable to Ca^{2+} ions.
- Their effectiveness grows with the postsynaptic potential, providing a mechanism for "coincidence detection".

4 Synapses and LIF neurons

The dynamic equation for the membrane potential $V(t)$ of a LIF neuron is

$$\tau_m \frac{dV(t)}{dt} = -[V(t) - E_L] + r_m\, I_e(t)$$

We assume a resting potential E_L, electrode current $I_e(t)$ (if any), membrane time constant τ_m, and threshold and reset potentials V_{th} and V_{reset}:

$$\tau_m = 20\, ms, \quad E_L = -70\, mV, \quad V_{th} = -54\, mV, \quad V_{reset} = -80\, mV$$

One synapse

Replacing (inward) electrode current $I_e(t)$ with an (outward) time-varying synaptic current $I_s(t)$ gives

$$\begin{aligned} \tau_m \frac{dV(t)}{dt} &= -[V(t) - E_L] - r_m\, I_s(t) = \\ &= -[V(t) - E_L] - r_m\, g_s\, P_k(t)\, [V(t) - E_s] \end{aligned}$$

with

$$-\underbrace{r_m\, g_s}_{strength} \qquad \underbrace{P(t)}_{time-course} \qquad \underbrace{[V(t) - E_s]}_{driving force}$$

Strength is given by *ratio* between membrane and synaptic conductance:

$$r_m\, g_s = \frac{g_s}{g_L}$$

Multiple synapses

With multiple synapses of the same kind, we simply add the individual conductances

$$\tau_m \frac{dV(t)}{dt} = -[V(t) - E_L] - r_m\, g_s \sum_k P_k(t)\, [V(t) - E_s]$$

with

$$-\underbrace{r_m\, g_s}_{strength} \quad \underbrace{\sum_k P(t)}_{time-course} \quad \underbrace{[V(t)-E_s]}_{driving force}$$

Post-synaptic currents and potentials

We define the synaptic (outward) currents in a post-synaptic neuron as

$$I_{PSC} = -g_s \sum_k P_k(t)\,[V(t)-E_s] \qquad \text{post-synaptic current}$$

and the synaptic contribution to the dynamic equation of a post-synaptic neuron as

$$V_{PSP} = -r_m\, g_s \sum_k P_k(t)\,[V(t)-E_s] \qquad \text{post-synaptic potential}$$

Synaptic effects

To assess synaptic effects, we rearrange the dynamic equation into the standard form:

$$\tau_m^{eff}\,\frac{dV}{dt} = -V + V_\infty^{eff}$$

$$V_\infty^{eff} = \frac{E_L + r_m\, g_s\ \Sigma_k\ P_k(t)\, E_s}{1 + r_m\, g_s\ \Sigma_k\ P_k(t)} \qquad r_m^{eff} = \frac{r_m}{1 + r_m\, g_s\ \Sigma_k\ P_k(t)}$$

$$\tau_m^{eff} = \frac{\tau_m}{1 + r_m\, g_s\ \Sigma_k\ P_k(t)}$$

Just like other conductances, synaptic conductances change effective equilibrium potential V_∞^{eff}, effective membrane resistance r_m^{eff} and effective membrane time constant τ_m^{eff}.

Presynaptic to postsynaptic spikes ...

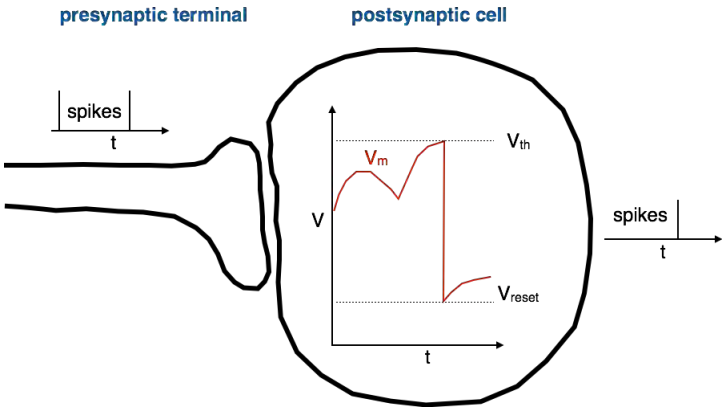

Figure 20: Pre-Postsynaptic interactions. [20]

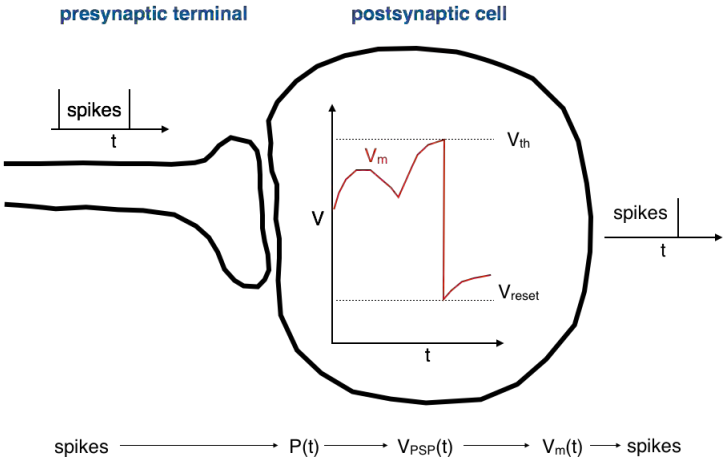

Figure 21: Pre-Postsynaptic interactions in 4 steps. [13]

Four steps

1. Presynaptic spike t_i releases transmitter and opens conductance $P_s(t)$ for $t \geq t_i$, say:

$$P_s(t) = {}^{t-t_i}/_{\tau_s} \exp\left(-{}^{t-t_i}/_{\tau_s}\right)$$

2. Conductance contributes $V_{PSP}(t)$ to dynamic equation of membrane potential:

$$V_{PSP}(t) = -r_m\, g_s\, P_s(t)\, [V_m(t) - E_s]$$

3. Equilibrium potential $V_\infty(t)$ and time-constant $\tau_m(t)$ change

$$V_\infty(t) = [E_L + r_m\, g_s\, P_s(t)\, E_s] \,/\, [1 + r_m\, g_s\, P_s(t)\, E_s]$$

4. Membrane potential $V_m(t)$ relaxes towards new equilibrium potential.

Example I: excitatory synapses

Consider excitatory synapses k, with a combined EPSP following presynaptic spikes i:

$$V_{EPSP}(t) = -r_m\, g_s \sum_k P_k(t)\, [V(t) - E_s]$$

$$P_k(t) = \begin{cases} \frac{t-t_i}{\tau_s} \exp\left(1 - \frac{t-t_i}{\tau_s}\right) & \text{if } t \geq t_i \\ 0 & t < t_i \end{cases}$$

$$E_s = 0\,mV \qquad r_m\, g_s = 0.25 \qquad \tau_s = 10\,ms$$

$$r_m\, g_s = 0.25 \qquad\qquad r_m\, I_e = 0\,mV$$

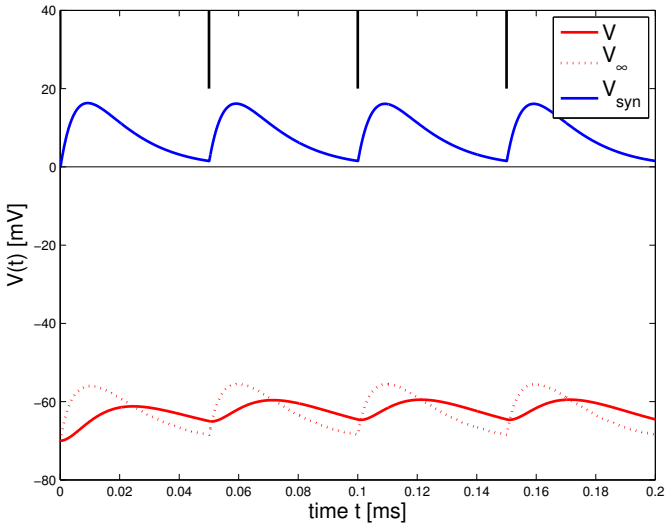

Figure 22: Excitatory synapses.

Without postsynaptic spikes

$$r_m\, g_s = 0.25 \qquad\qquad r_m\, I_e = 0\, mV$$

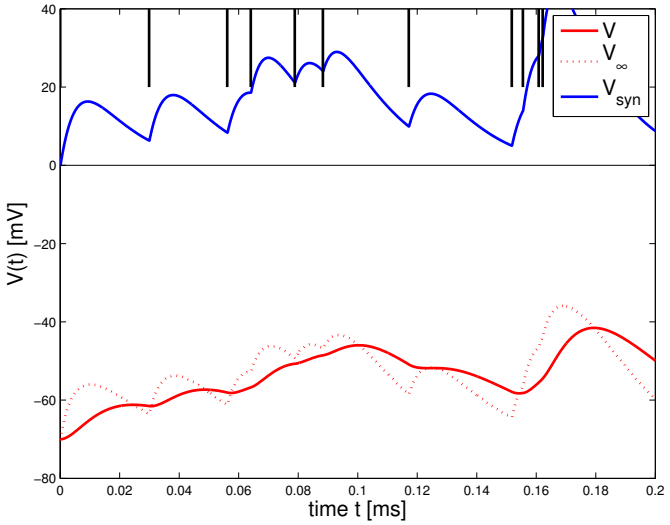

Figure 23: Excitatory without postsynaptic spikes.

With postsynaptic spikes

$$r_m\, g_s = 0.25 \qquad\qquad r_m\, I_e = 12\, mV$$

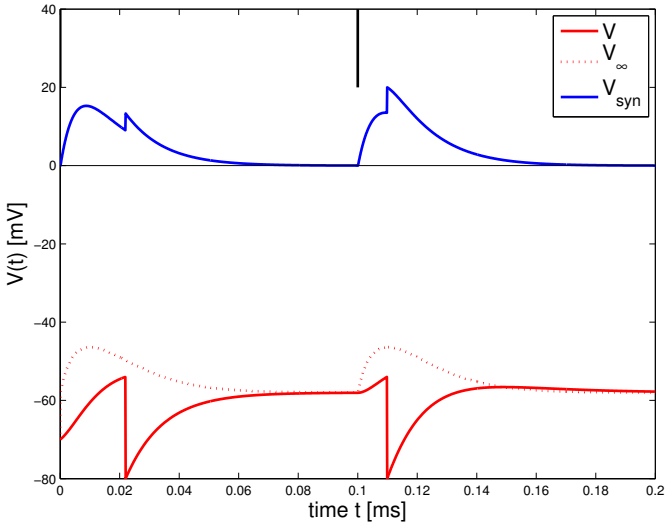

Figure 24: Excitatory with postsynaptic spikes.

$$r_m\, g_s = 0.25 \qquad\qquad r_m\, I_e = 12\, mV$$

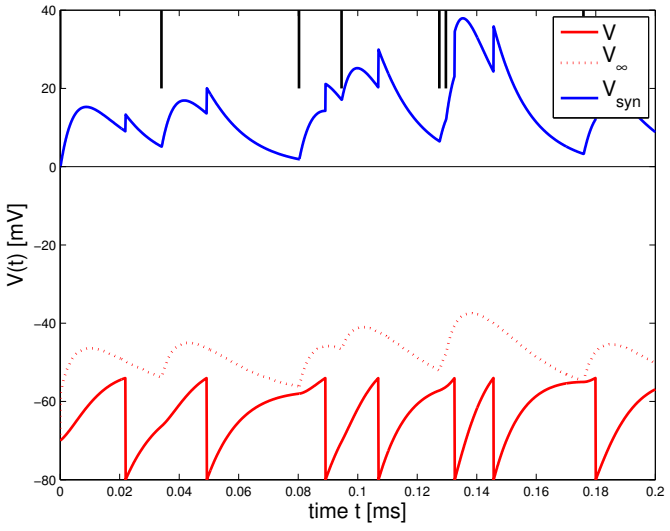

Figure 25: With postsynaptic spikes.

Example II: inhibitory synapses

Consider inhibitory synapses k, with a combined IPSP following presynaptic spikes i:

$$V_{IPSP}(t) = -r_m\, g_s \sum_k P_k(t)\,[V(t) - E_s]$$

$$P_k(t) = \begin{cases} \frac{t-t_i}{\tau_s}\, \exp\left(1 - \frac{t-t_i}{\tau_s}\right) & \text{if } t \geq t_i \\ 0 & t < t_i \end{cases}$$

$$E_s = -80\,mV \qquad r_m\, g_s = 1.0 \qquad \tau_s = 10\,ms$$

$$r_m\, g_s = 1.0 \qquad r_m\, I_e = 20\,mV$$

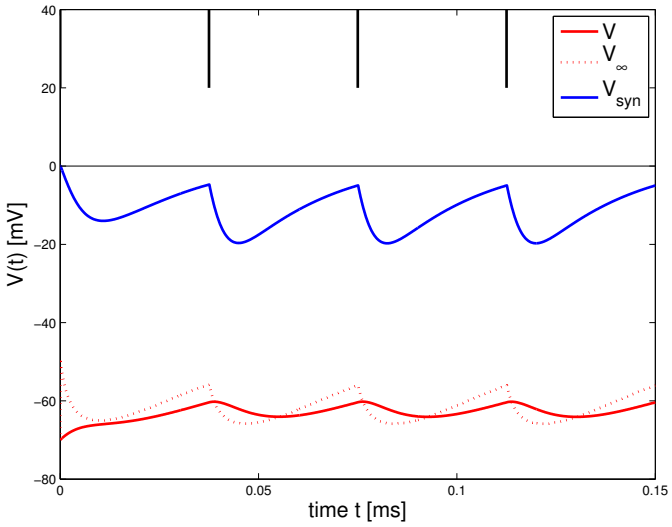

Figure 26: Inhibitory synapses.

Without postsynaptic spikes

$$r_m\, g_s = 1.0 \qquad r_m\, I_e = 20\,mV$$

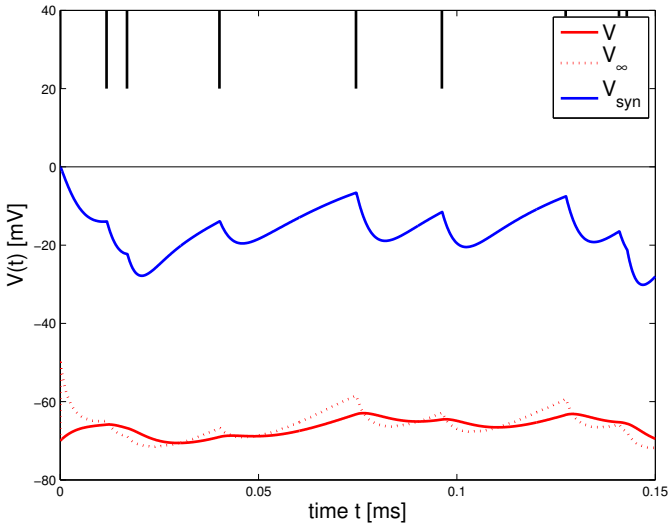

Figure 27: Inhibitory without postsynaptic spikes.

With postsynaptic spikes

$$r_m\, g_s = 1.0 \qquad\qquad r_m\, I_e = 30\, mV$$

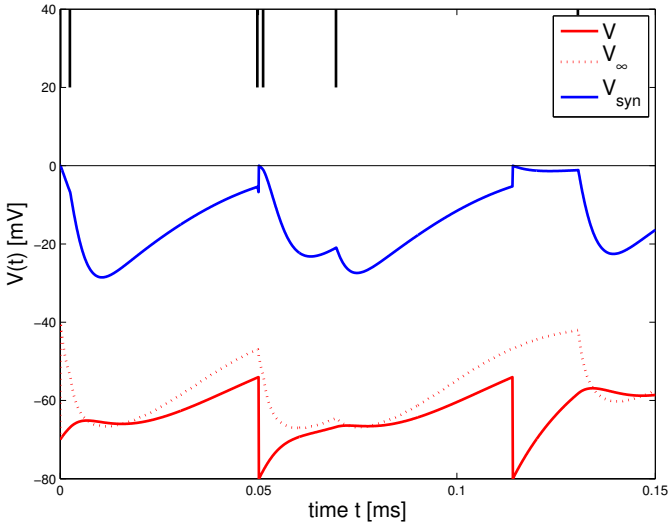

Figure 28: Inhibitory with postsynaptic spikes.

Summary synapses and LIF neurons

- Synaptic conductances directly affect:
  1. equilibrium potential
  2. membrane conductance/resistance
  3. membrane time-constant

- The postsynaptic current (PSC) is

  $$\textit{synaptic conductance} \times \textit{driving force}$$

- The postsynaptic potential (PSP) is the contribution to the dynamic equation

  $$\textit{membrane resistance} \times \textit{synaptic conductance} \times \textit{driving force}$$

- Post-synaptic spikes and reset change driving force!

5 Short-term plasticity

Synaptic efficacy $\bar{g}_{syn}$ can change when synapses are used (presynaptic activity). Transient changes that reverse with presynaptic inactivity: **short-term plasticity**. Permanent changes that depend on both pre- and postsynaptic activity: **long-term plasticity**.

In terms of the quantal model (Section 5), short-term plasticity changes the release probability P_r and is therefore a **presynaptic** mechanism.

Long-term plasticity changes both P_r and the postsynaptic quantal efficacy q and is therefore a **pre- and postsynaptic** mechanism.

Depression and facilitation

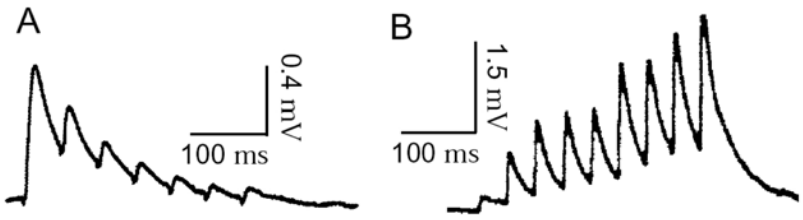

Figure 29: Depression and facilitation. [14]

Synapses show both short-term **depression** and short-term **faciliation**. In the first case the amplitude decreases during a rapid series post-synaptic potentials, in the second case in increases.

Use-dependent changes

Using a synapse changes P_{rel}, either increasing or decreasing it:

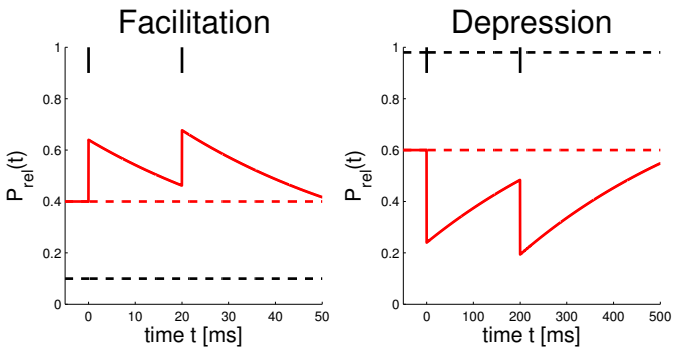

Figure 30: Facilitation and Depression.

When the synapse remains unused, P_{rel} relaxes back to its default value P_0, either low or high. **Note slow time-scale!**

Formalization

Variable release size $P_{rel}(t)$, default size P_0.

Between synaptic events:

$$\tau_P \frac{dP_{rel}(t)}{dt} = P_0 - P_{rel}(t), \qquad P_{rel}(t) = P_0 + [P_{ini} - P_0] \exp\left(-\frac{t}{\tau_P}\right)$$

After each synaptic event:

$$P_{rel} \to f_D \, P_{rel}(t) \qquad 0 \leq f_D \leq 1 \qquad \text{depression}$$

$$P_{rel} \to P_{rel} + f_F \, (1 - P_{rel}) \qquad 0 \leq f_F \leq 1 \qquad \text{faciliation}$$

Given an *average* initial value P_{ini}, the change between synaptic events is

$$P_{end} = P_0 + [f_D \, P_{ini} - P_0] \exp\left(-\frac{t_{isi}}{\tau_P}\right)$$

However, presynaptic events occur irregularly, so that t_{isi} and P_{end} takes a different value each time.

Figure 31: Poisson spikes.

Average release

The *average* release probability $\langle P_{rel} \rangle$ is the value at which the *average* change between events is zero:

$$\langle P_{end} \rangle = \langle P_{ini} \rangle \equiv \langle P_{rel} \rangle$$

$$P_{end} = P_0 + [f_D \langle P_{rel} \rangle - P_0] \exp\left(-\frac{t_{isi}}{\tau_P}\right)$$

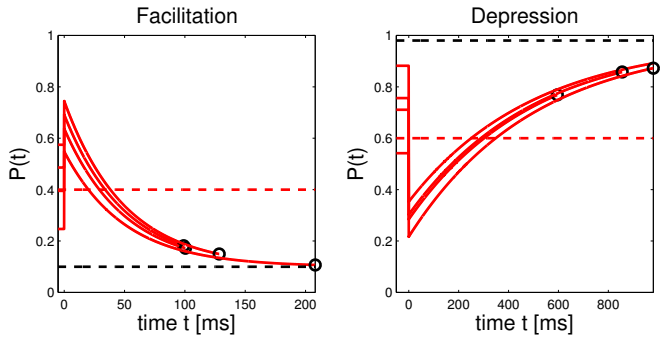

Figure 32: Average release.

f

Averaging over irregular spike trains (advanced)

To average over an irregular spike train, we must know the probability density function (PDF) of interspike intervals t_{isi}. Assuming a *Poisson* spike train of rate r, the PDF is

$$p(t_{isi})\, dt_{isi} = r\, e^{-t_{isi}\, r}\, dt_{isi}, \qquad \int_0^\infty p(t_{isi})\, dt_{isi} = 1$$

Average exponential decay:

$$\langle \exp\left(-{}^{t_{isi}}/_{\tau_P}\right)\rangle \;=\; \int_0^\infty \exp\left(-{}^{t_{isi}}/_{\tau_P}\right)\, p(t_{isi})\, dt_{isi} =$$

$$=\; r\int_0^\infty \exp\left(-t_{isi}\, r - {}^{t_{isi}}/_{\tau_P}\right)\, dt_{isi} = \frac{r\,\tau_P}{1+r\,\tau_P}$$

Solution of average release

$$P_{rel}(t_{isi}) = P_0 + \left[f_D\,\langle P_{rel}\rangle - P_0\right]\,\exp\left(-\frac{t_{isi}}{\tau_P}\right)$$

... taking the average ...

$$\langle P_{rel}\rangle = P_0 + \left[f_D\,\langle P_{rel}\rangle - P_0\right]\,\frac{r\,\tau_P}{1+r\,\tau_P}$$

... solving for $\langle P_{rel}\rangle$ ...

$$\langle P_{rel}\rangle = \frac{P_0}{1+r\,(1-f_D)\,\tau_P}$$

Average release and average transmission

Now we know how short-term depression affects the average release probability:

$$\langle P_{rel}\rangle = \frac{P_0}{1+r\,(1-f_D)\,\tau_P}$$

To obtain average transmission t, we simply multiply average release with the release rate r:

$$t \equiv \langle P_{rel}\rangle\, r = \frac{P_0\, r}{1+r\,(1-f_D)\,\tau_P}$$

Average effect of facilitation and depression

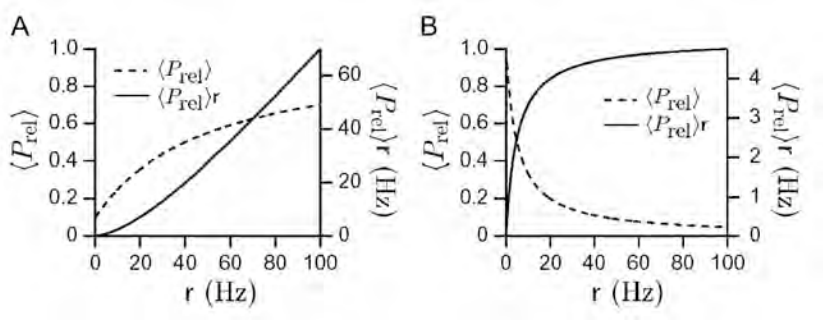

Figure 33: Left: STF enhances differences in r. Right: STD attentuates differences in r [15]

Transient effects of short-term depression (STD)

The transient effect is to *maintain* differences in firing rates. If the firing rate r doubles to $2r$, the *initial* transmission doubles, too:

$$t = \frac{P_0\, r}{1 + r\,(1 - f_D)\,\tau_P} \qquad \rightarrow \qquad t_{new} = \frac{2\,P_0\, r}{1 + r\,(1 - f_D)\,\tau_P}$$

$$t_{new}/t = 2$$

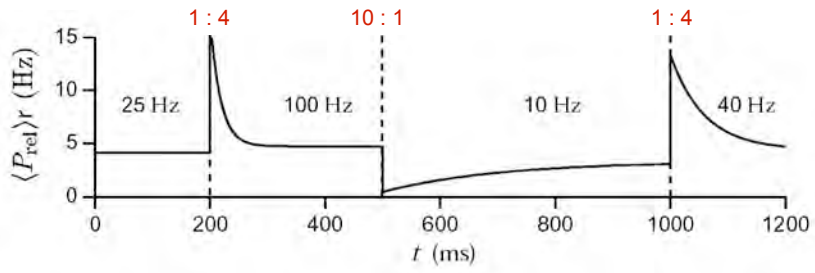

Figure 34: Transient effects. [16]

Sustained effects of short-term depression (STD)

The sustained effect is to *attenuate* differences in firing rates. If a sustained firing rate r doubles to $2r$, the *sustained* transmission

increases by a factor smaller than 2:

$$t = \frac{P_0\, r}{1 + r\,(1 - f_D)\,\tau_P} \qquad \rightarrow \qquad t_{new} = \frac{2\,P_0\, r}{1 + 2\,r\,(1 - f_D)\,\tau_P}$$

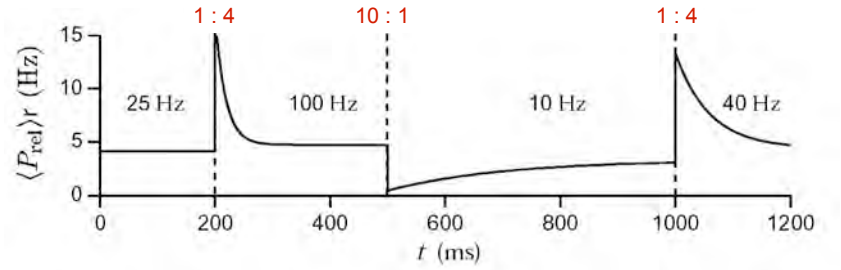

Figure 35: Short term depression. [16]

Summary of short-term plasticity

- Short-term plasticity temporarily changes the probability of presynaptic transmitter release.
- It is thought to be important for stable network activity.
- Its effect can be assessed by averaging over irregular spike trains.
- Short-term *depression* emphasizes transient changes over sustained changes in presynaptic firing.

General summary

1. Synaptic transmission is too complex for detailed physical modeling.

2. Descriptive formulas capture amplitude and time-course of post-synaptic conductance.
3. GABA, AMPA, and NMDA synapses differ in strength and speed.
4. NMDA synapses depend on presynaptic transmitter release and on postsynaptic potential ("coincidence detection").

5. Synaptic conductances affect equilibrium potential, membrane resistance, and membrane time-constant.

6. Short-term depression emphasizes transient changes in presynaptic firing.

6 Bibliography

1. PSY 32000: Meeting 05. Purdue University. Prof. Gerald S. Wasserman, 2009. Figure 9. Ref: $http://www1.psych.purdue.edu/Classes/psy32000/meeting05/02_023.jpg$

2. Sinapsi e Transmissione Sinaptica. Neuroscienze.net. Francesco albanese, 2010. Figure 2. Ref: https://www.neuroscienze.net/la-trasmissione-sinaptica/

3. Neurotransmitters. Neupsy key. Drzezo, 2016. Figure 4.1 Ref: https://neupsykey.com/neurotransmitters/

4. Folie 1. Exzitatorische (erregende) Synapsen. Goethe University Frankfurt. Bear. Slide 1, figure 1. Ref: $http://www.goethe-university-frankfurt.de/45915354/TPGrundstudium_05.pdf$

5. PSY 32000: Meeting 10. Purdue University. Prof. Gerald S. Wasserman, 2010. Figure 5. Ref: $http://www1.psych.purdue.edu/Classes/psy32000/meeting10/05_015.jpg$

6. Balancing structure and function at hippocampal dendritic spines. Pubmed.gov. Bourne JN, et al. 2008. Figure 1. Ref: $https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2561948/figure/F1/$

7. Folie 1. Exzitatorische (erregende) Synapsen. Goethe University Frankfurt. Bear. Slide 1, figure 1. Ref: $http://www.goethe-university-frankfurt.de/45915354/TPGrundstudium_05.pdf$

8. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.14 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$

9. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.15 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$

10. PSY 32000: Meeting 09. Purdue University. Prof. Gerald S. Wasserman, 2010. Figure 10. Ref: $http://www1.psych.purdue.edu/Classes/psy32000/meeting09/05_014.jpg$

11. PSY 32000: Meeting 09. Purdue University. Prof. Gerald S. Wasserman, 2010. Figure 4. Ref: $http://www1.psych.purdue.edu/Classes/psy32000/meeting09/05_013.jpg$

12. Kainate Receptor Antagonist. IHC2015. Anonymous, 2019. Figure 4. Ref: $https://ihc2015.info/skin/kainate-receptor-antagonist.akp$

13. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.17 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$

14. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.18 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$

15. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.19 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$

16. Model Neurons I: Neuroelectronics 5.1 Introduction 5.2 Electrical properties of Neurons. Anonymous. Figure 5.19 Ref: $https://www.semanticscholar.org/paper/Model-Neurons-I%3A-Neuroelectronics-5.1-Introduction/0e259937bb89cd3ede50063ac32b98e56086c1b6$