March 10, 2014
Synaptic Plasticity - What is it?
Due today - RP Lab

Intrinsic Synaptic Plasticity (short term)
Extrinsic Synaptic Plasticity
Synaptic changes due to disease


Simulation - Postsynaptic Inhibition

Post-inhibitory rebound
2 other mechanisms?

Conductance decrease synaptic potential??

Conductance decrease synaptic potential
Close resting leak K^+ channels.
Cell depolarizes because of resting Na^+ conductance no longer balanced

Conductance decrease potentials move away from reversal potential
Intrinsic Synaptic Plasticity (short term)
Change in synaptic strength due to activity of synapses
Important for:
- coincidence detection
- gain control
- oscillatory networks (phase onset and offset, cycle period
- Habituation, sound localization
- Learning and Memory

Extrinsic Synaptic Plasticity
Change in synaptic strength due to neuromodulatory substances
Important for:
- Network reconfiguration (active network members, their excitability and synaptic connections)

Intrinsic Synaptic Plasticity transforms time interval into what?

Extrinsic Synaptic Plasticity transforms time interval into voltage amplitude

Facilitation, Post-tetanic Potentiation, Depression

This week in Lab:
Intrinsic synaptic modulation
Facilitation
Intrinsic synaptic modulation:
Post-tetanic potentiation

Intrinsic synaptic modulation:
Depression

Intrinsic synaptic modulation
highly variable in different brain networks:

Depression

Facilitation

Mixed

Synaptic Modulation
Sites of Possible Synaptic Change?
Sites of synaptic change in short term plasticity?

Facilitation:
- MEPP sizes do not change.
- MEPP frequency increases.
- Quanta content (m = PSP/mini) is increased pre or post?

Residual Ca hypothesis for facilitation:
Presynaptic Ca builds up with each AP

Sequestration of calcium after an action potential
- Takes 100-500 msec to bring calcium levels to normal after an AP

Non-linear dependence of transmitter release on $[Ca]_i$

AP brings in 5 units of Ca
$5^1 = 525$
80% uptake before Next AP
1 unit left $= 1$
Next AP = $1 + 5$ units
$6^0 = 1296$
(twice as much NT release!)
Facilitation

1) Simulations of expected peak and residual Ca\(^{2+}\) levels not able to account for facilitation.

2) Using Ca\(^{2+}\) sensitive dyes, pre-synaptic [Ca\(^{2+}\)] was not raised enough to account for enhanced synaptic transmission.

3) The time course of I\(_{\text{K(Ca)}}\) is too fast. The decay of this current should reflect the decay of residual Ca\(^{2+}\).

Ca may be acting at multiple sites of the synaptic machinery

BUT:

PTP:

1) Correlates with decay of Ca\(^{2+}\) image in whole terminal, not just at release sites, and reduced by Ca\(^{2+}\) chelators.

2) In crustacean motor neurons, Na\(^+\) has a role, perhaps through the Ca/Na pump. Entry of Na\(^+\) during AP firing may reduce the efficiency of the Na/Ca exchanger to remove Ca\(^{2+}\).

3) Ca\(^{2+}\) unloading from mitochondria or other internal stores.

4) In Aplysia- MEPP frequency up with PTP, but not amplitude. Both pre- and post- synaptic Ca\(^{2+}\) chelators reduce PTP, as well as postsynaptic hyperpolarization.[

Mechanisms of Synaptic Depression?

Mechanisms of Synaptic Depression?

A CF B

Shibor, a temperature sensitive fly mutant, vesicle recycling is blocked

Depression correlates with depleted vesicles