The Neuroscience of Learning

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Key Information

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Course Outline

Week One: How We Learn
 Topic 1. Repetition and Hebbian Learning
 Topic 2. Feedback and Types of Learning

Week Two; How We Learn
 Topic 1. Long Term Potentiation and Synaptic Plasticity
 Topic 2. Dopamine and the Basal Ganglia

Week Three: What We Learn
 Topic 1. Explicit Memory
 Topic 2. Implicit Memory

Week Four: What We Learn
 Topic 1. Neural Basis of Memory
 Topic 2. Internal Models

Week Five: How We Can Improve Learning
 Topic 1. Distributed Practice, Random Practice, Variable Practice
 Topic 2. Specificity of Practice, Part-Whole Practice, Mental Imagery

Week Six: How We Can Improve Learning
 Topic 1. Sleep, Diet, and Exercise
 Topic 2. Age, Learning Disorders

How do we learn?

REPETITION

Definition: Learning

Changes in internal processes that are reflected by relatively stable changes in performance.

Hebb (1949)

"When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased"

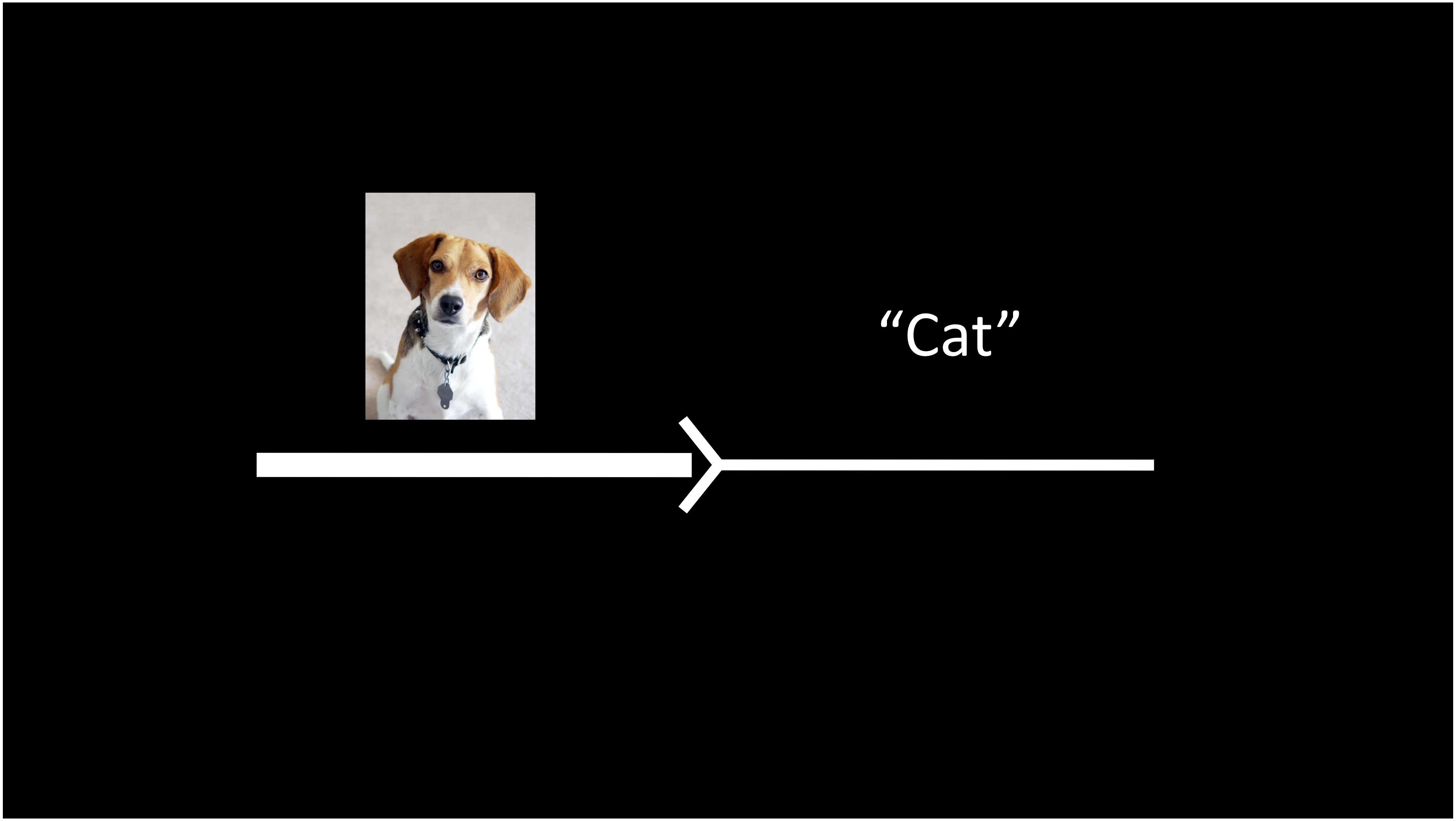
"The organization of behavior"

Hebb's Rule

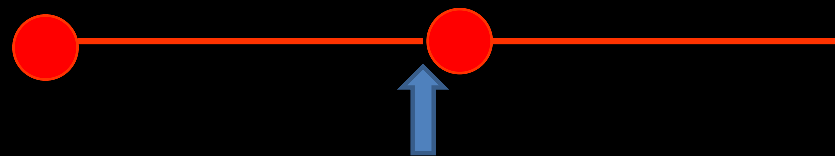
From a learning perspective, the goal of the system is to increase the strength of the neural connections that are effective.

Hebb's Rule:

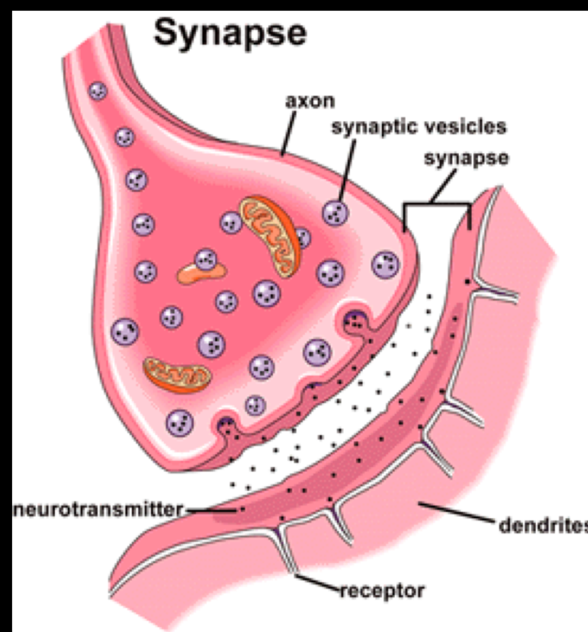
"neurons that fire together wire together"

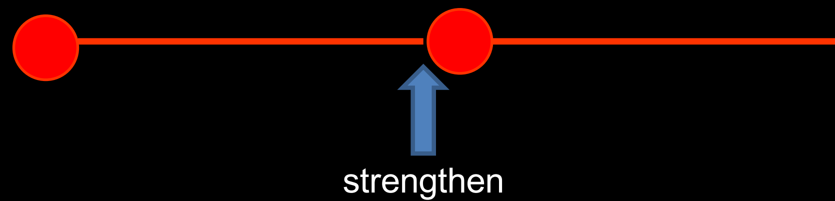

“Cat”

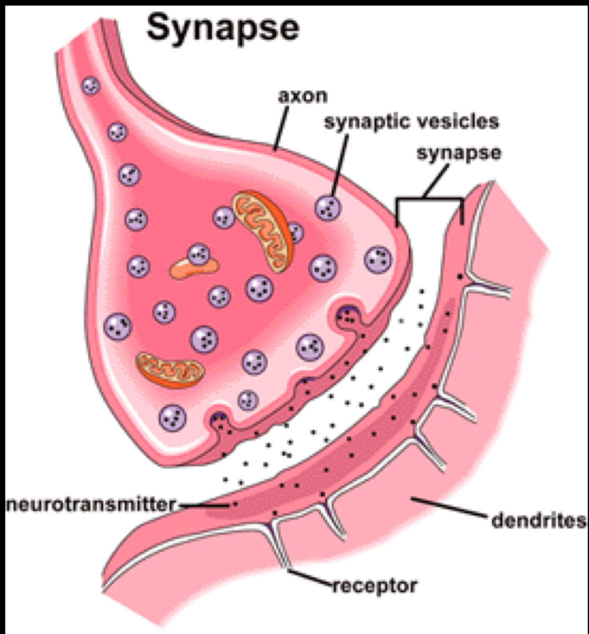
“Dog”



strengthen


Synapse
axon
synaptic vesicles
synapse
neurotransmitter
dendrites
receptor



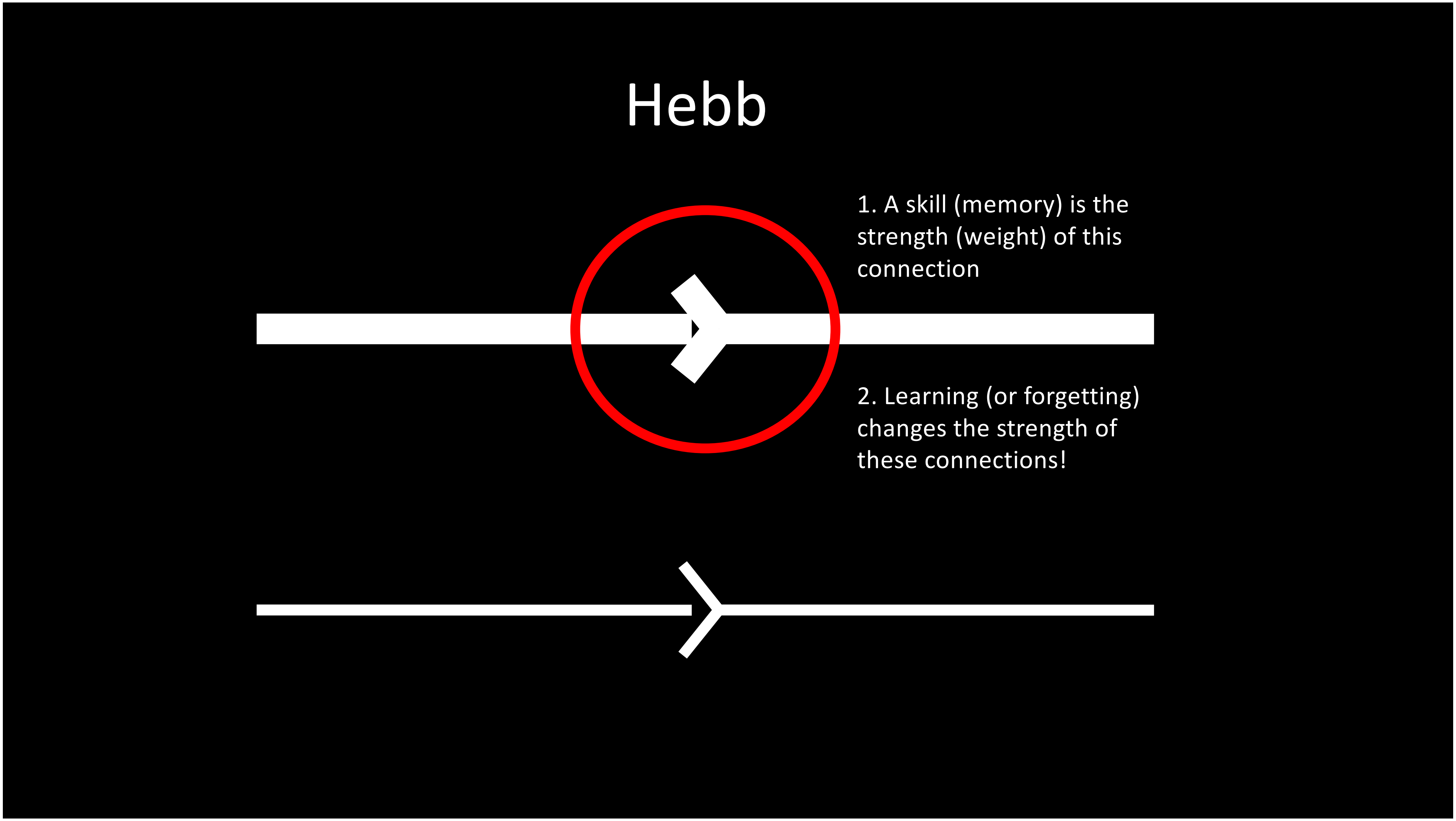


HOW?

"Synaptic Plasticity"

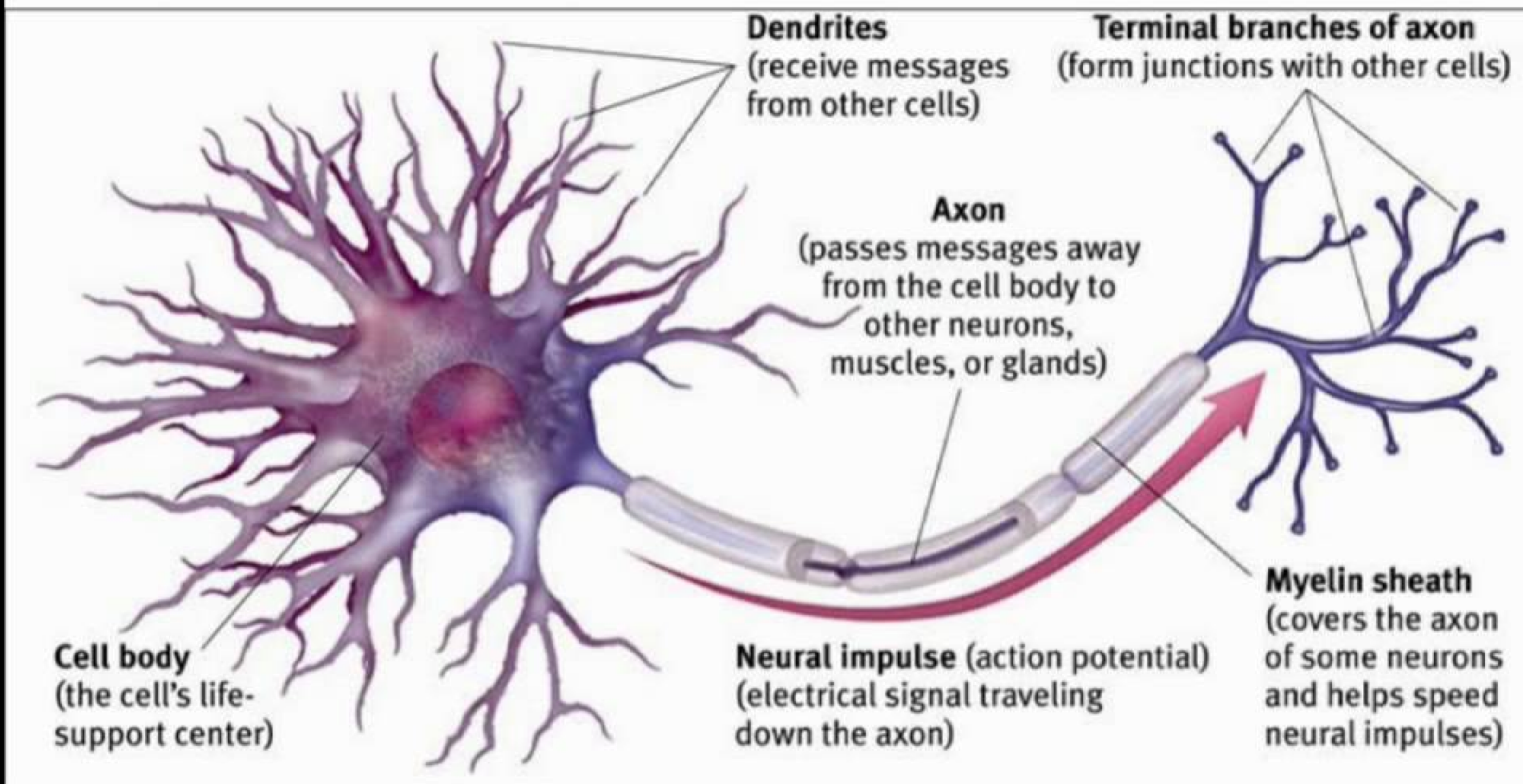
Definition: Learning

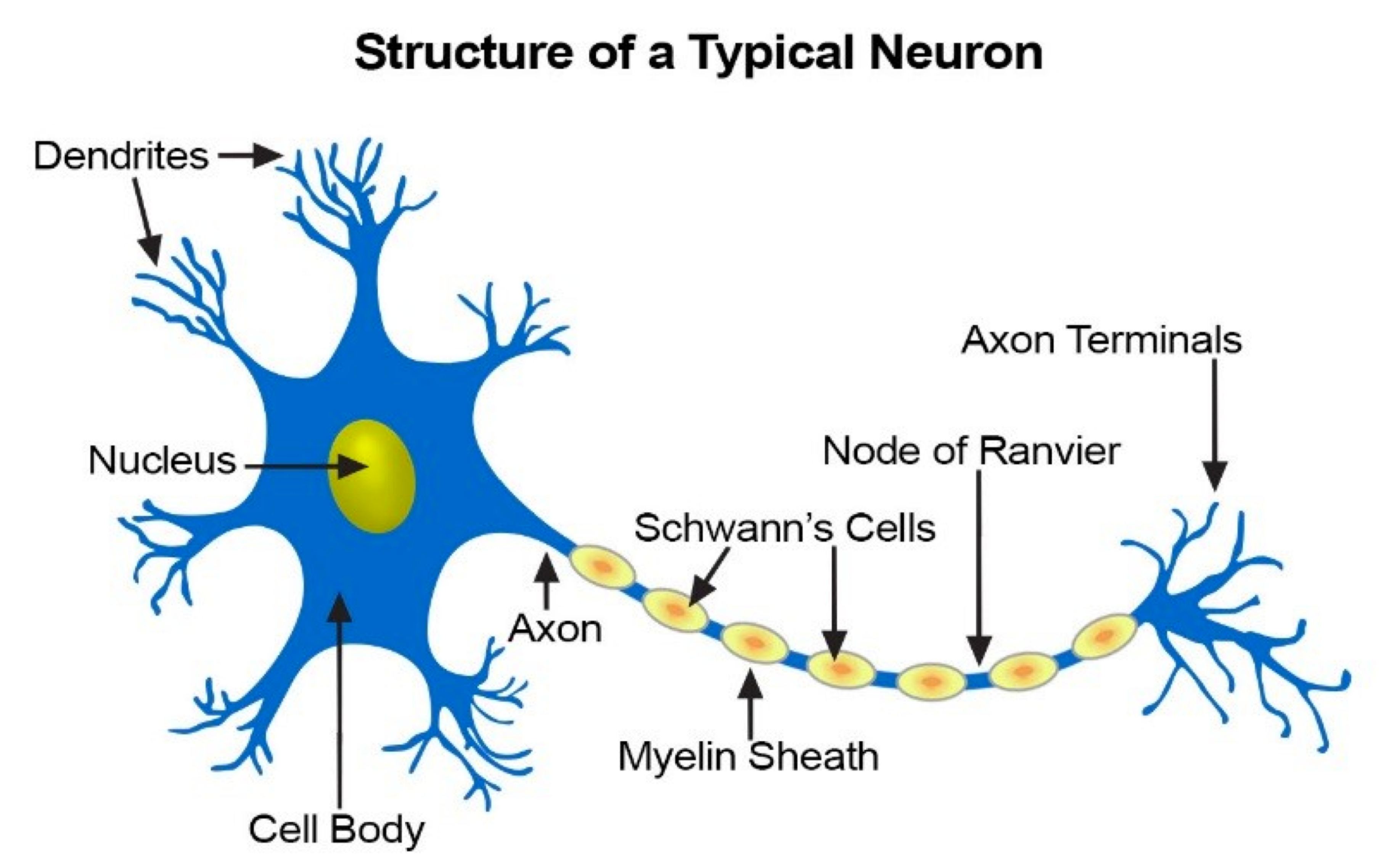
Changes in internal processes (neural connections are strengthened) that are reflected by relatively stable changes in performance (because these changes are relatively permanent).


Hebb
1. A skill (memory) is the strength (weight) of this connection
2. Learning (or forgetting) changes the strength of these connections!

How a Neuron Fires

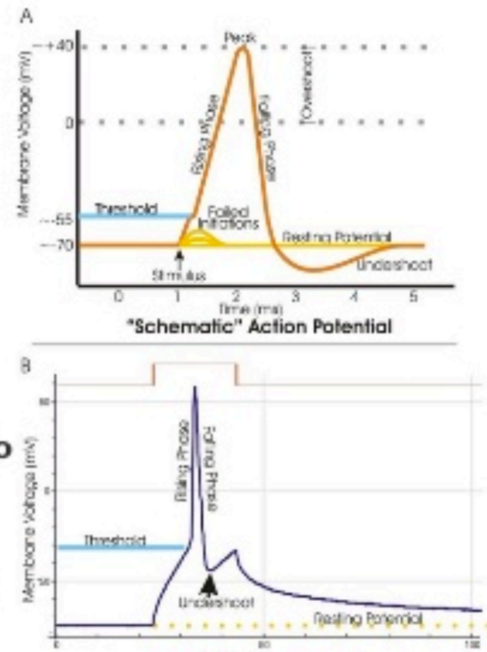
Basic Neural Structures




Structure of a Typical Neuron
Dendrites
Nucleus
Cell Body
Axon
Schwann's Cells
Myelin Sheath
Node of Ranvier
Axon Terminals

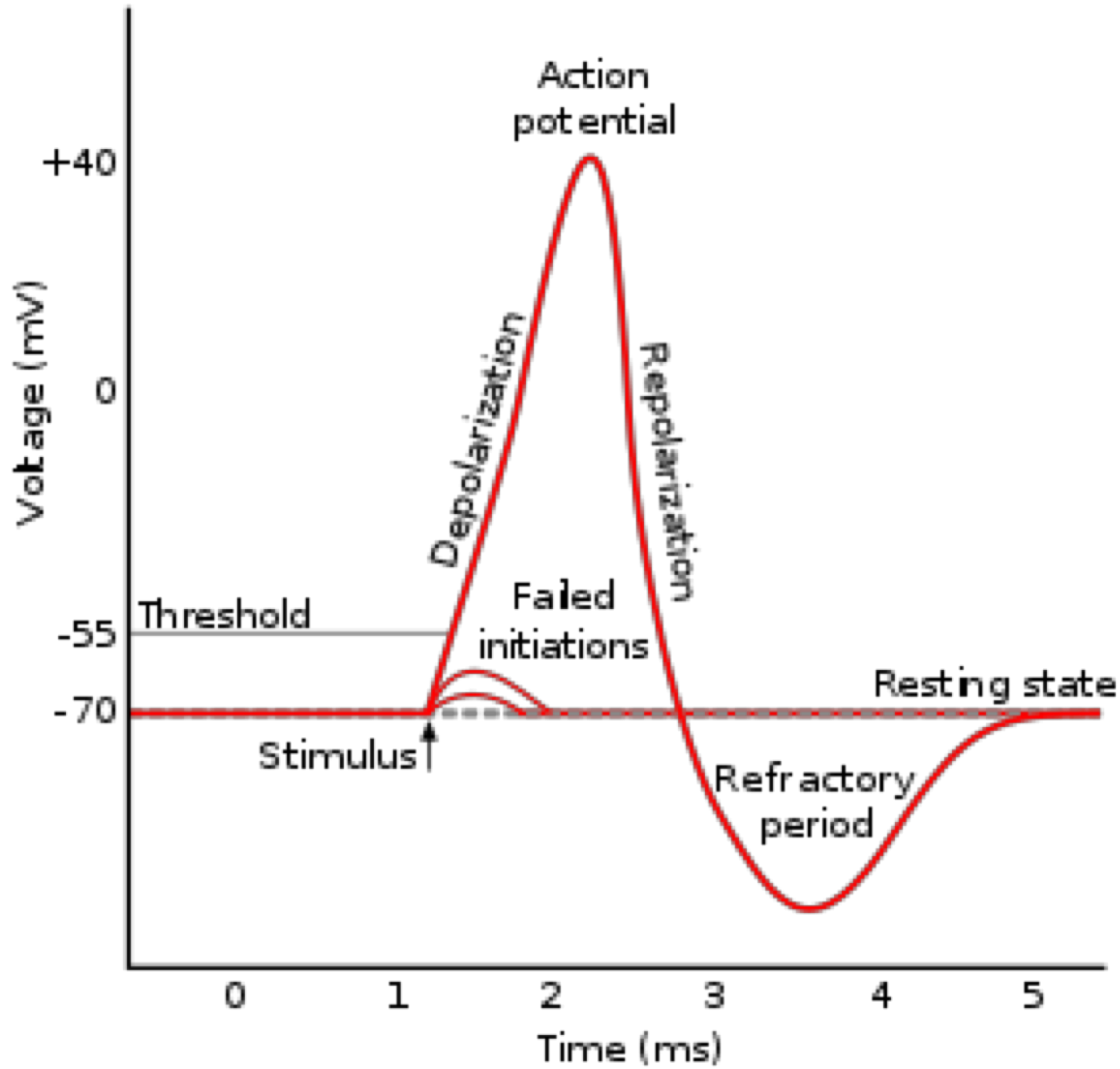
Neural Communication Terms

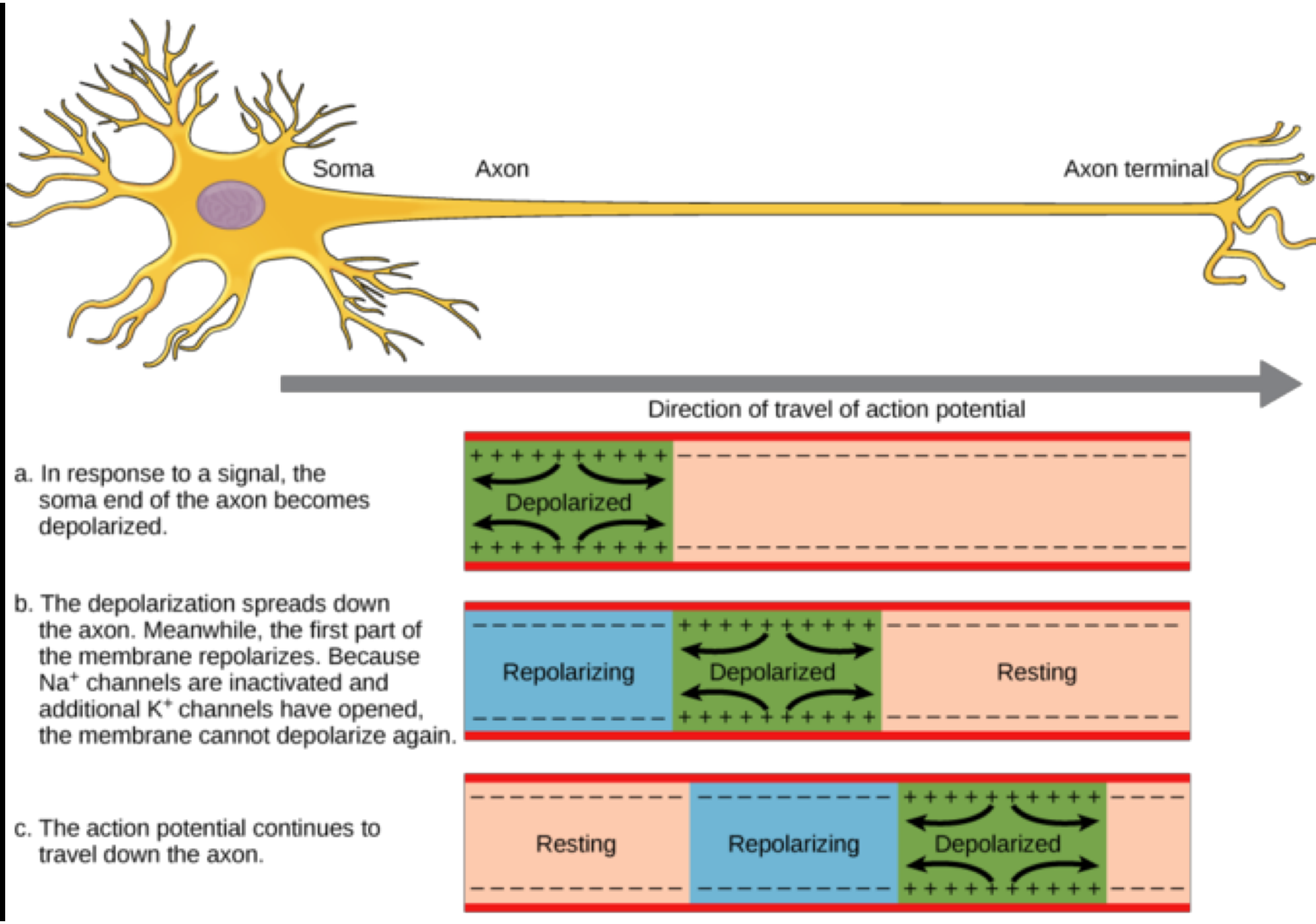
- **Resting Potential**
  - Negative Ions inside the axon
- **Action Potential**
  - Positive Ions move inside the axon
- **Refractory Period**
  - Neuron can't fire (resetting effect)
- **Sodium-Potassium Pump**
  - How the Neuron Fires
- **Absolute Threshold**
  - How much stimulation the neuron needs to fire
- **All-or-None Response**
  - A neuron fires or it doesn't fire (like a gun)

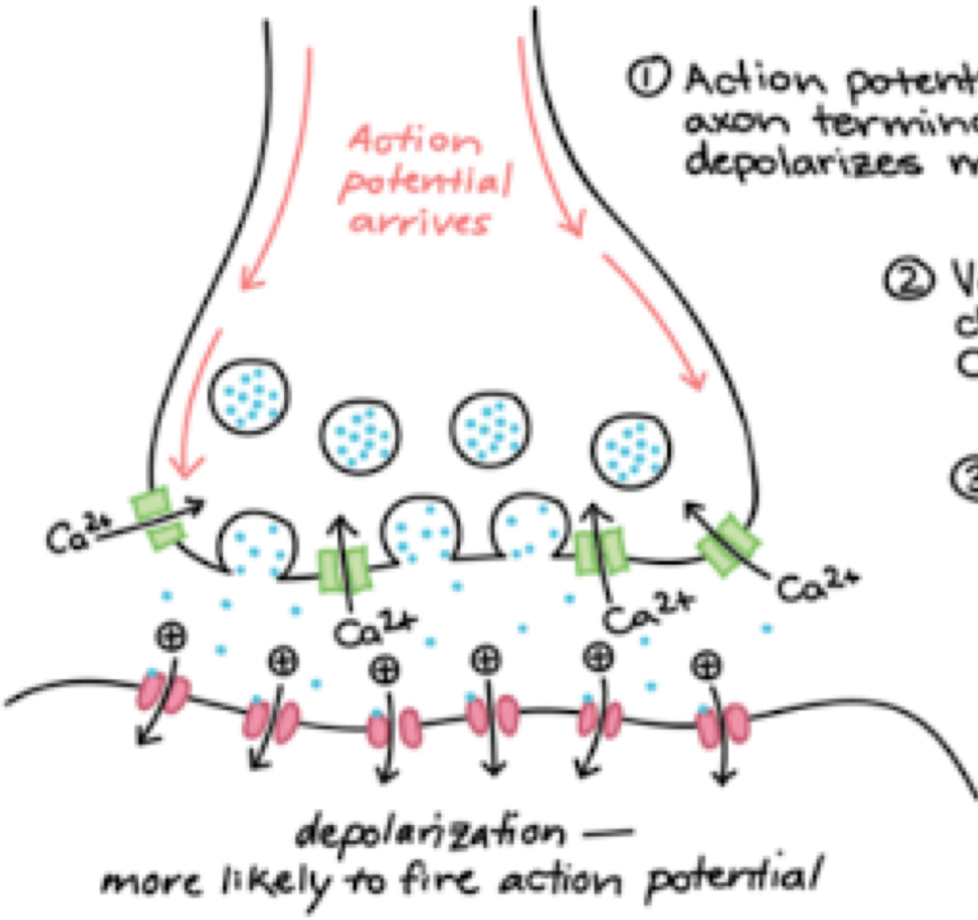


"Schematic" Action Potential

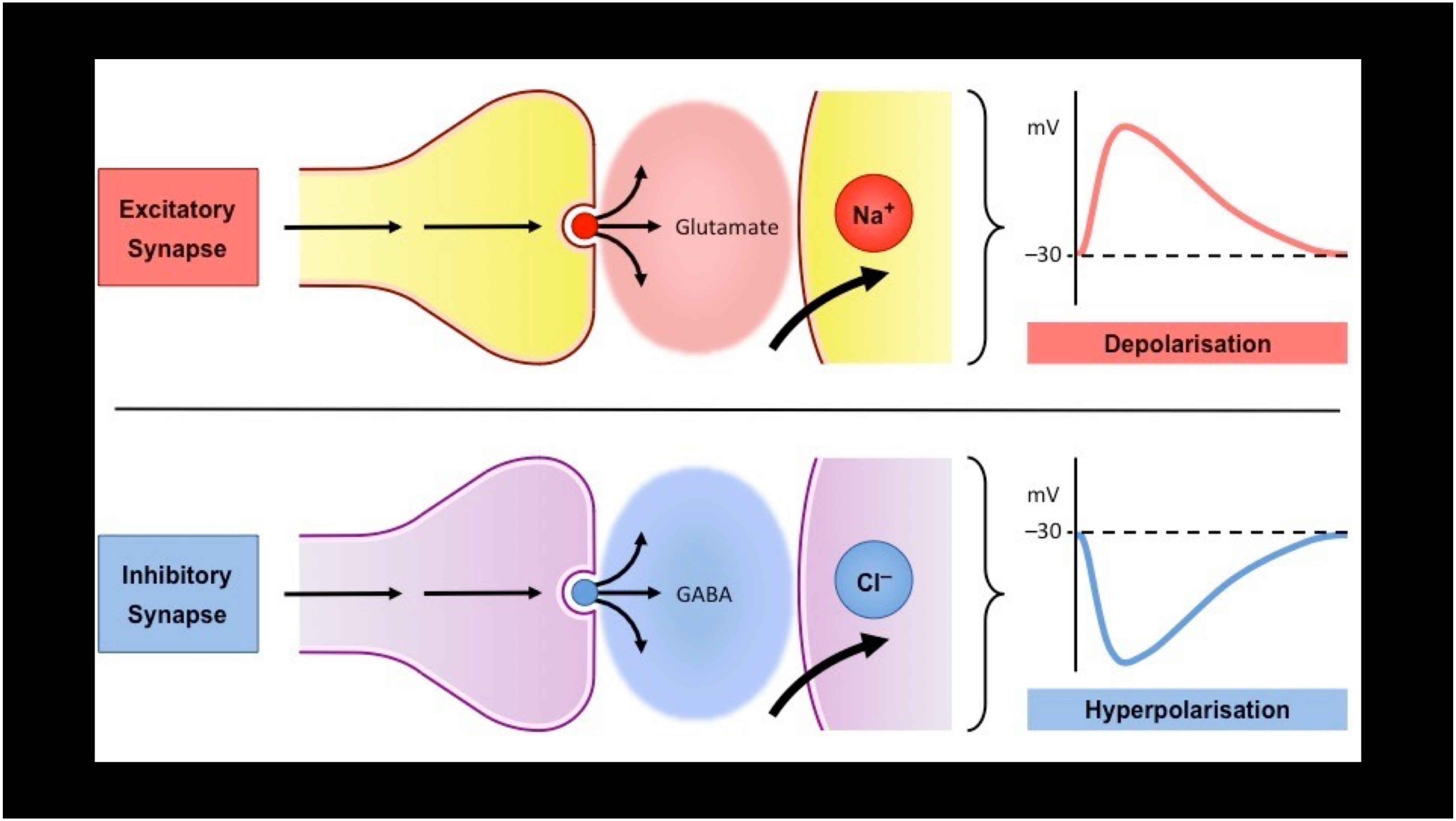
"Real" Action Potential

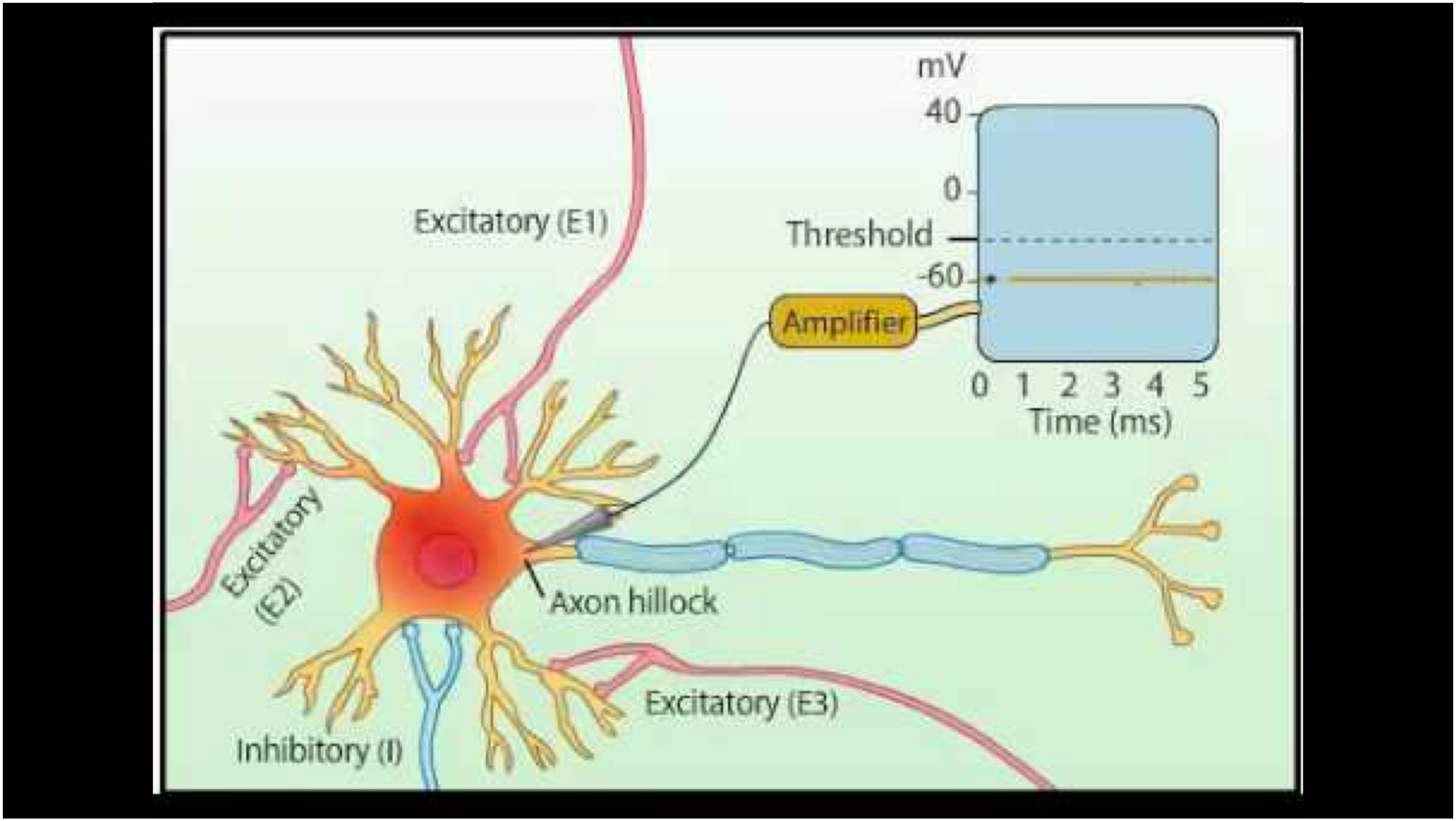

Action
potential
+40
0
-55
-70
Voltage (mV)
Depolarization
Repolarization
Threshold
Failed
initiations
Resting state
Stimulus
Refractory
period
0
1
2
3
4
5
Time (ms)


Soma
Axon
Axon terminal
Direction of travel of action potential
a. In response to a signal, the soma end of the axon becomes depolarized.
Depolarized
b. The depolarization spreads down the axon. Meanwhile, the first part of the membrane repolarizes. Because Na+ channels are inactivated and additional K+ channels have opened, the membrane cannot depolarize again.
Repolarizing
Depolarized
Resting
c. The action potential continues to travel down the axon.
Resting
Repolarizing
Depolarized



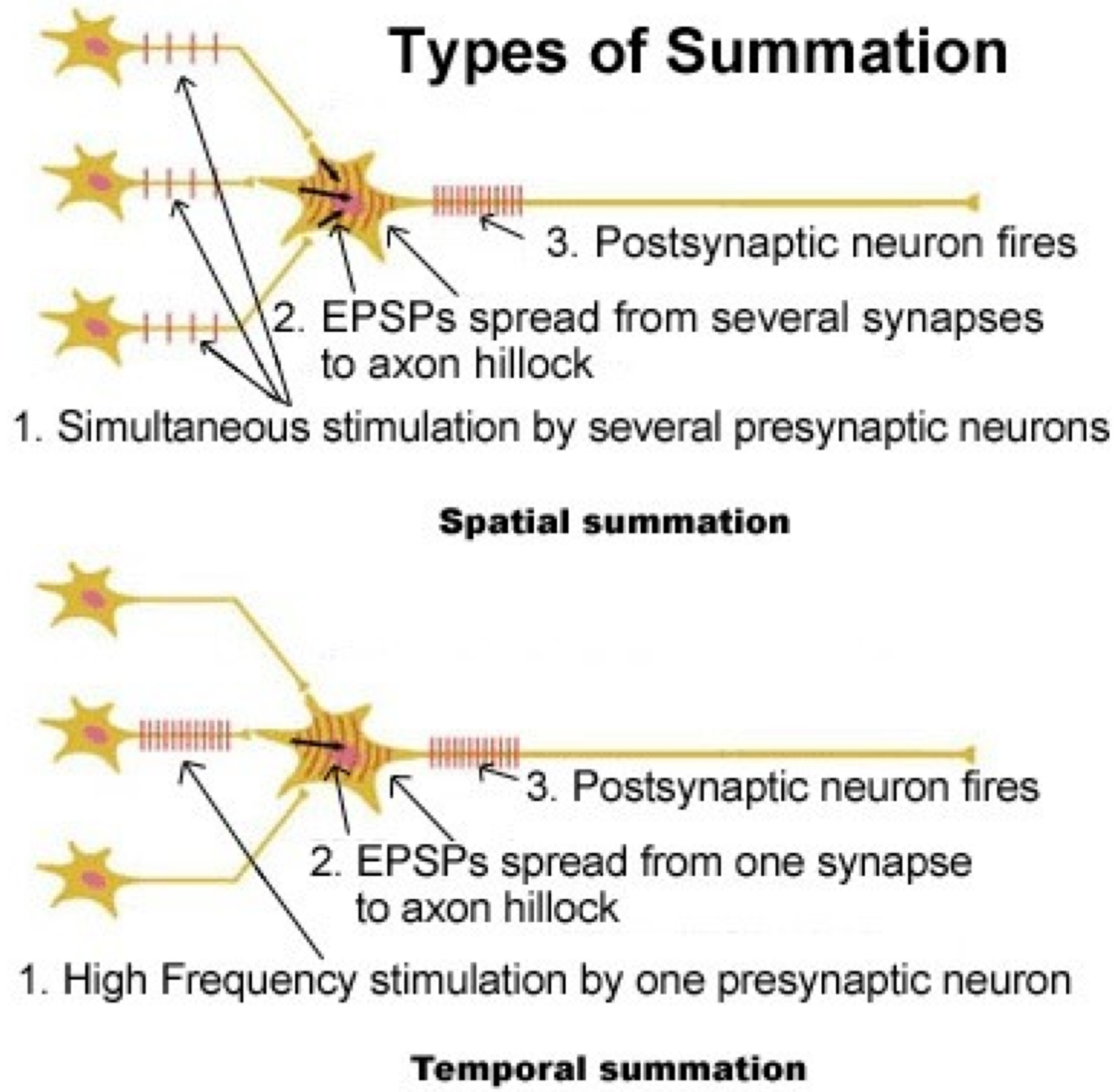
1. Action potential reaches axon terminal and depolarizes membrane.
2. Voltage-gated Ca^{2+} channels open and Ca^{2+} flows in.
3. Ca^{2+} influx triggers synaptic vesicles to release neurotransmitter.
4. Neurotransmitter binds to receptors on target cell (in this case, causing positive ions to flow in).

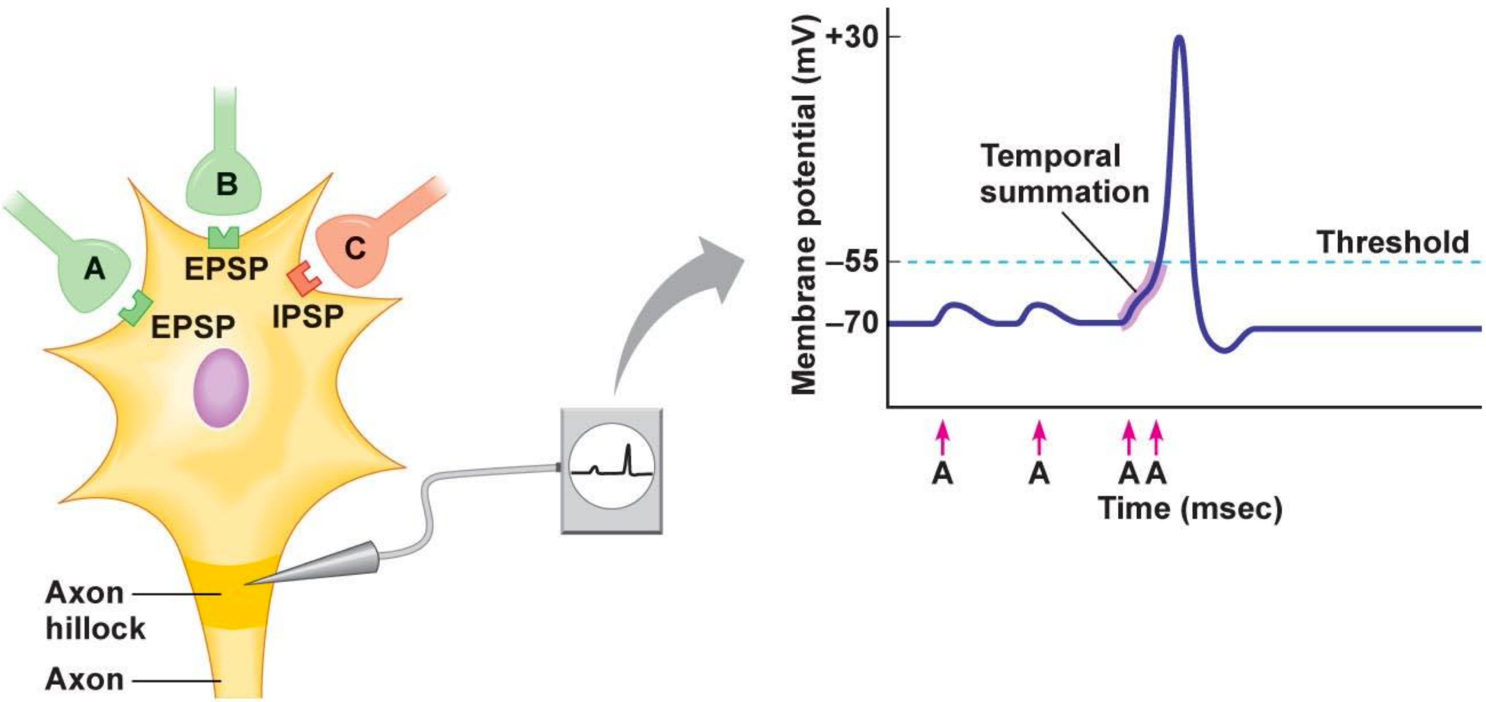

Excitatory Synapse
Glutamate
Na+
mV
−30
Depolarisation
Inhibitory Synapse
GABA
Cl−
mV
−30
Hyperpolarisation

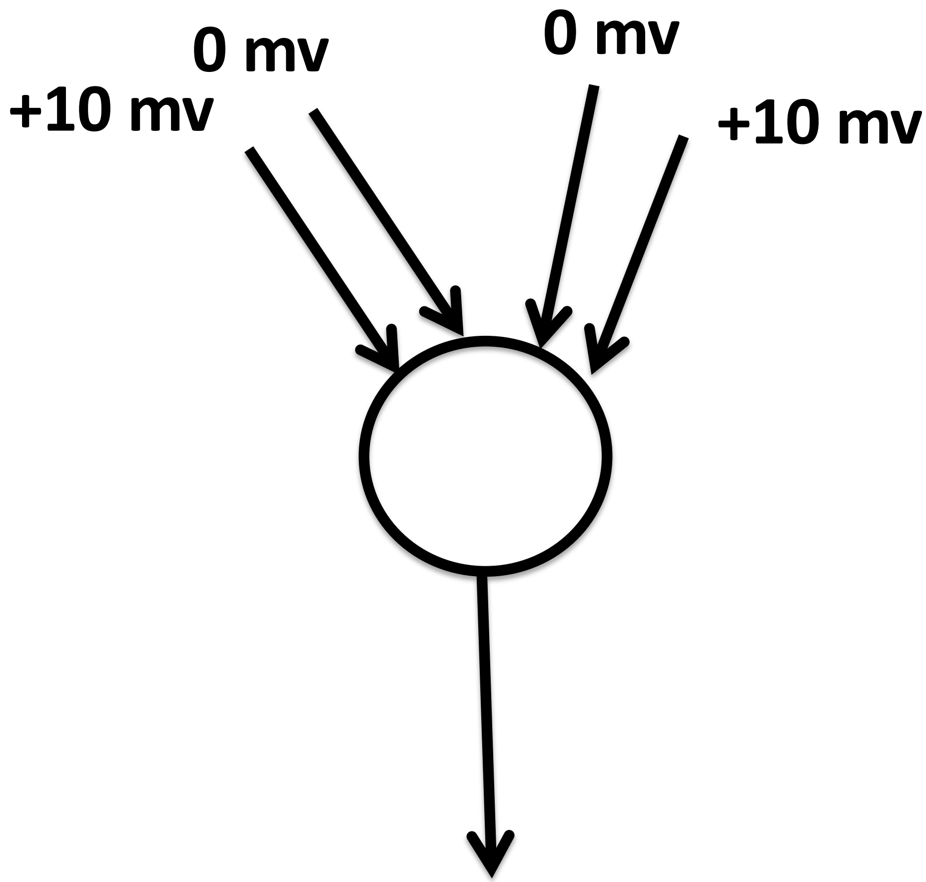


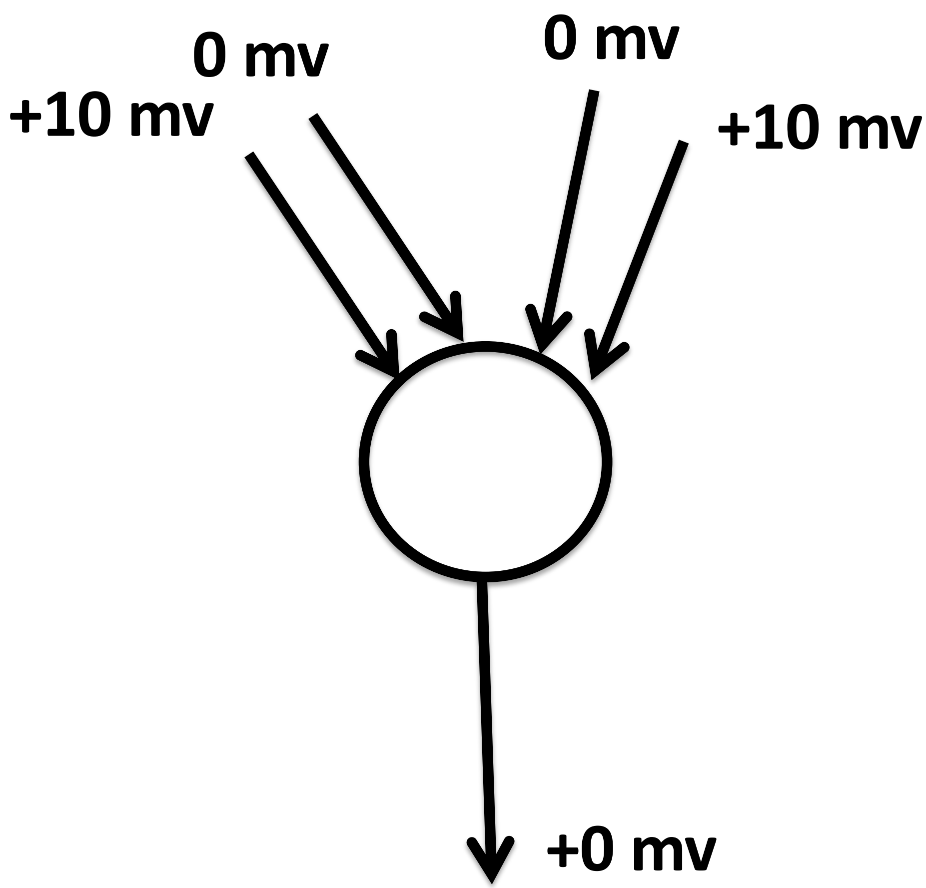
mV
40
0
Threshold
-60
Amplifier
0 1 2 3 4 5
Time (ms)
Excitatory (E1)
Excitatory (E2)
Axon hillock
Excitatory (E3)
Inhibitory (I)

Types of Summation

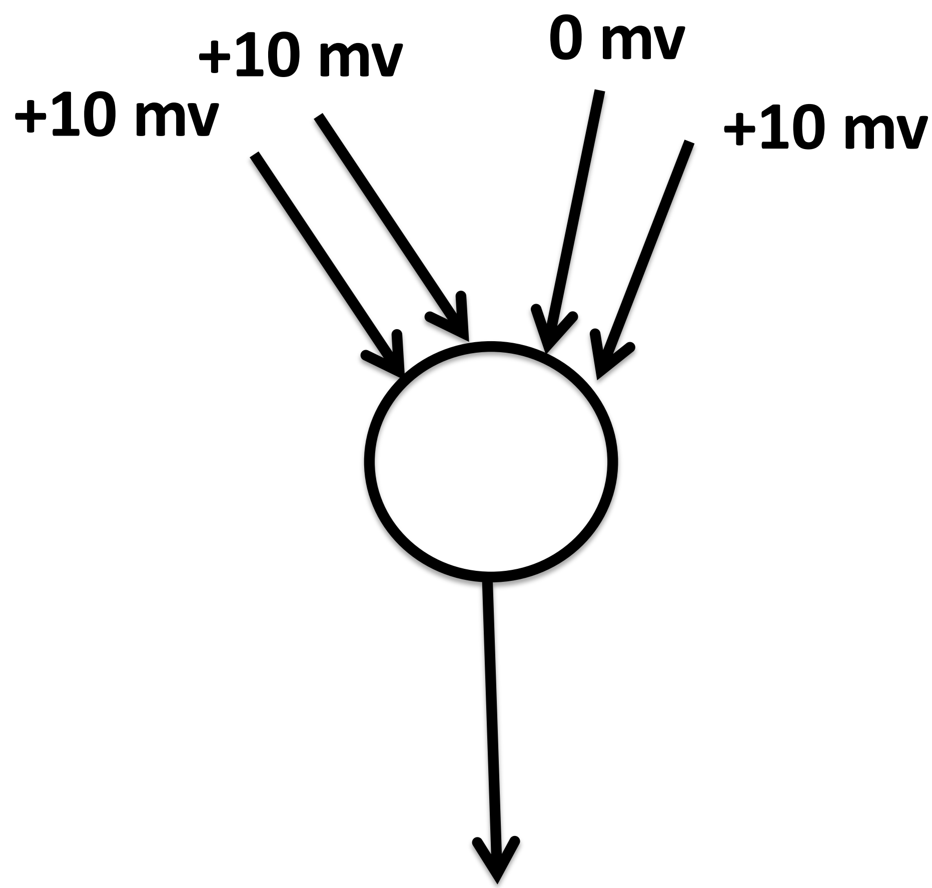


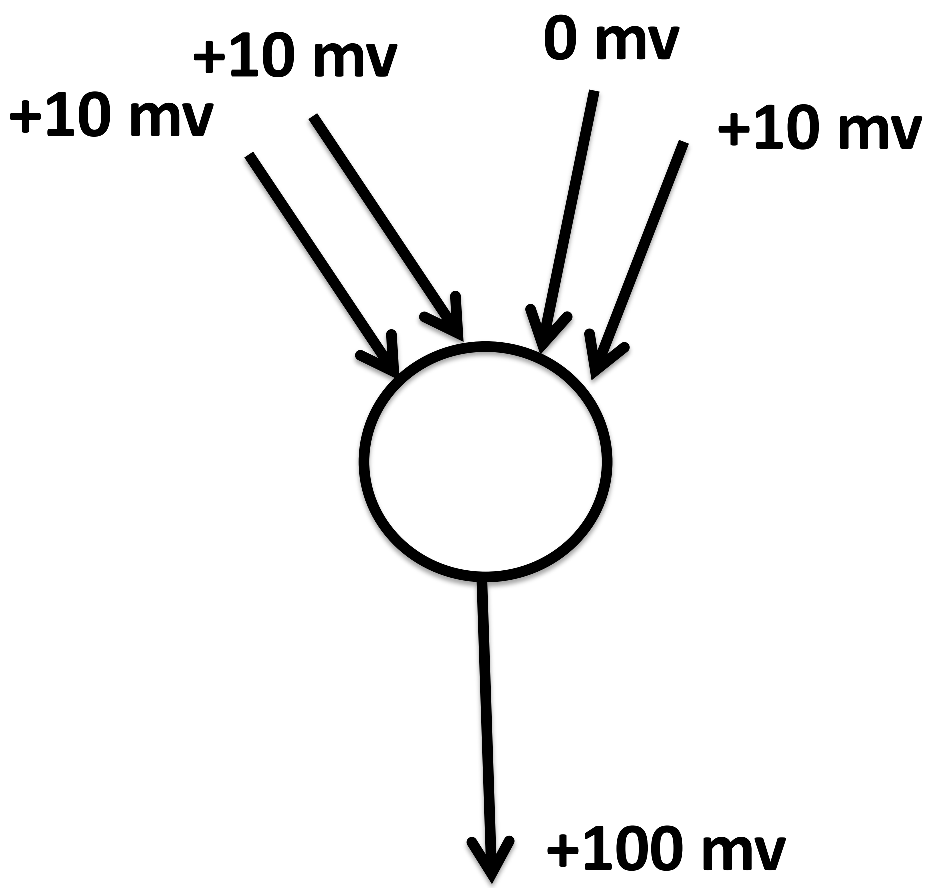

B
A
EPSP
C
EPSP
IPSP
Axon
hillock
Axon
Membrane potential (mV)
+30
–55
–70
Temporal
summation
Threshold
A
A
A A
Time (msec)

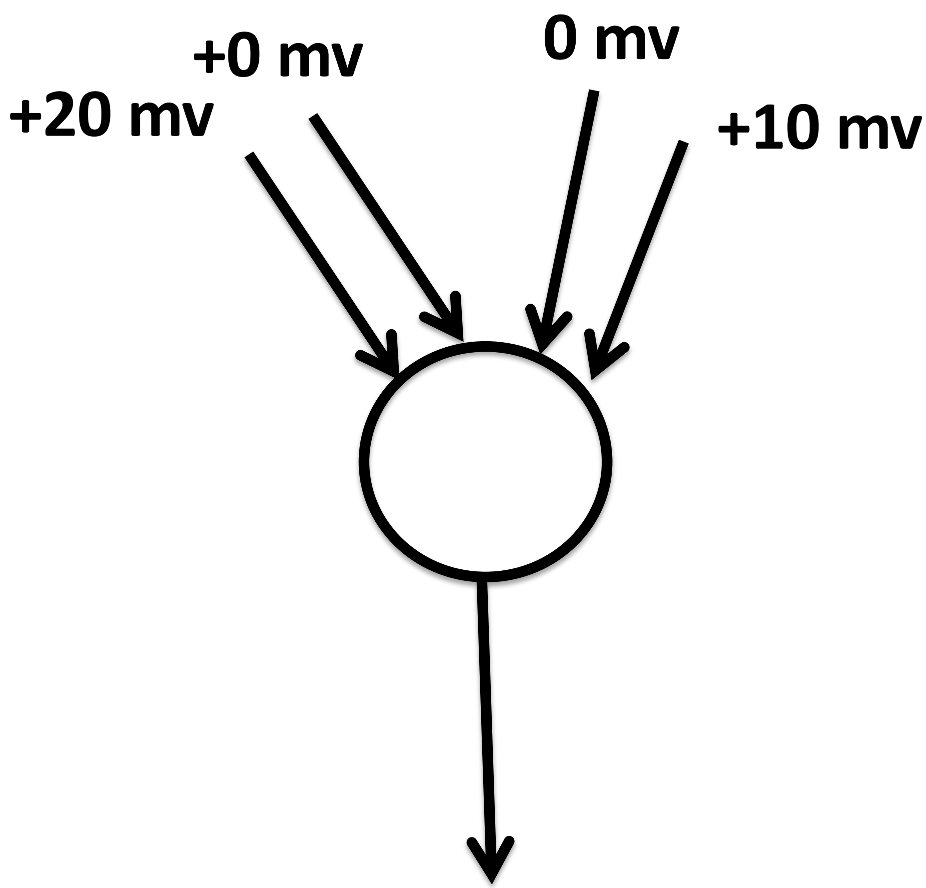

0 mv
0 mv
+10 mv
+10 mv

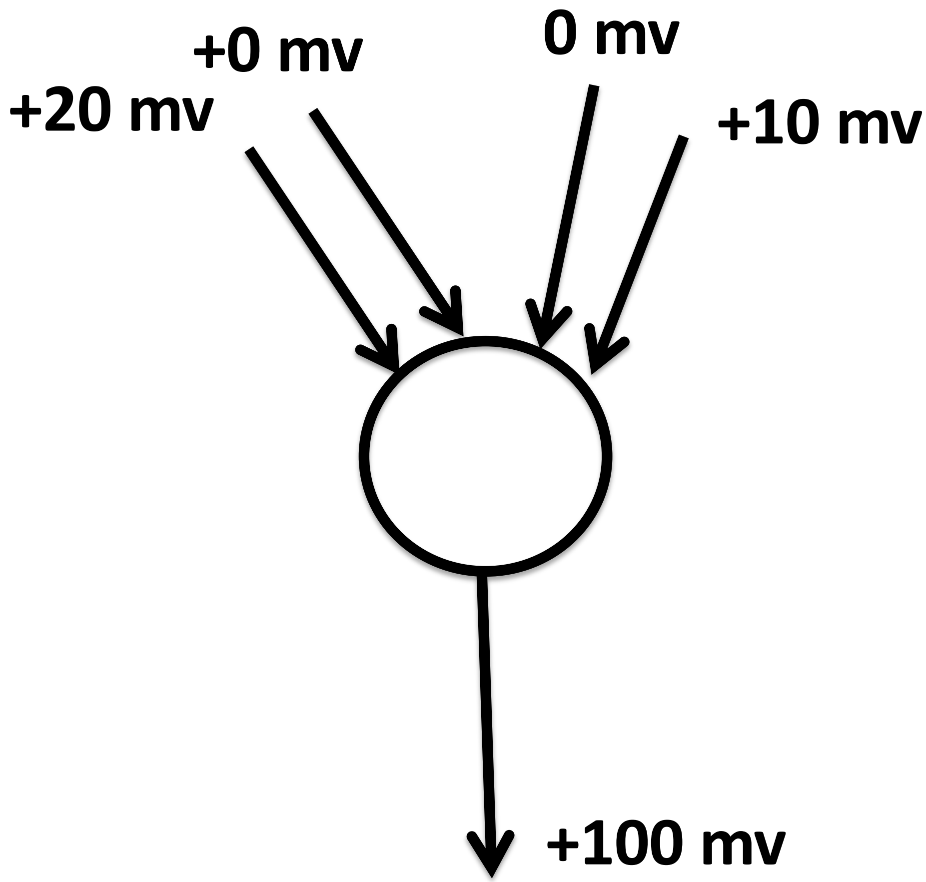


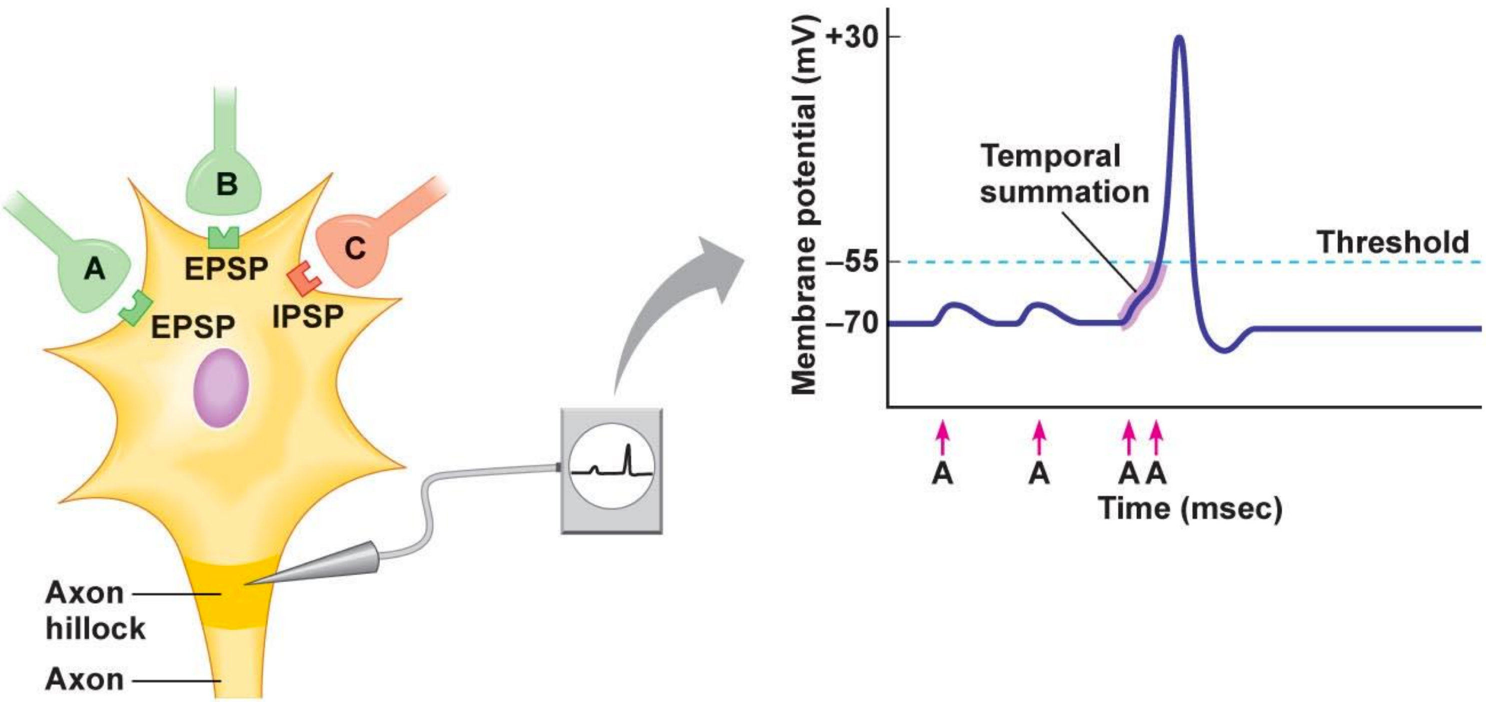
0 mv
0 mv
+10 mv
+10 mv
+0 mv


+10 mv
+10 mv
0 mv
+10 mv


+10 mv
+10 mv
0 mv
+10 mv
+100 mv

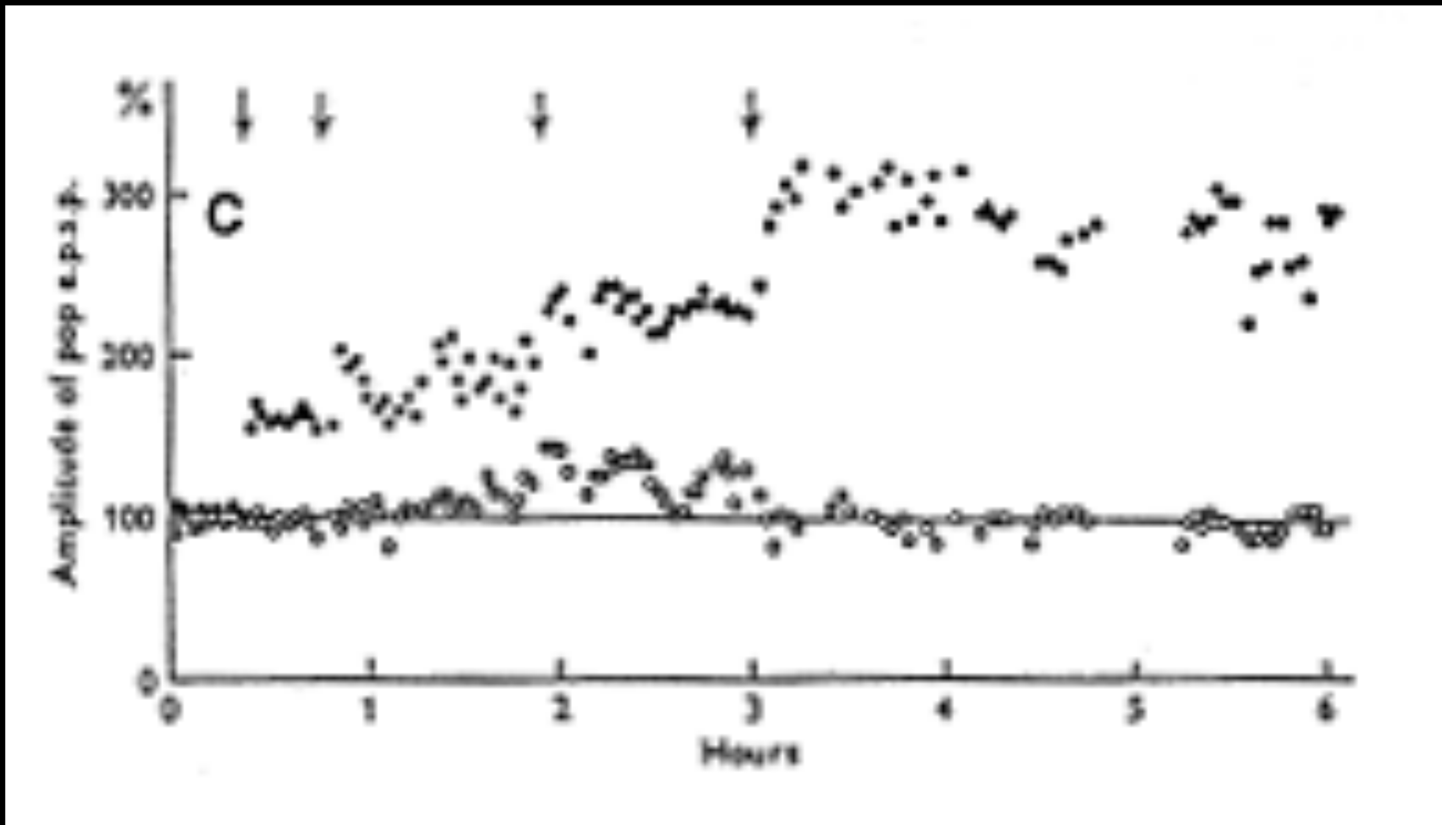

+20 mv
+0 mv
0 mv
+10 mv


+0 mv
0 mv
+20 mv
+10 mv
+100 mv


B
A
C
EPSP
EPSP
IPSP
Axon hillock
Axon
Membrane potential (mV)
+30
–55
–70
Temporal summation
Threshold
A
A
A A
Time (msec)

Short Term Changes: LTP

Bliss and Lomo’s First Published LTP Experiment



LTP

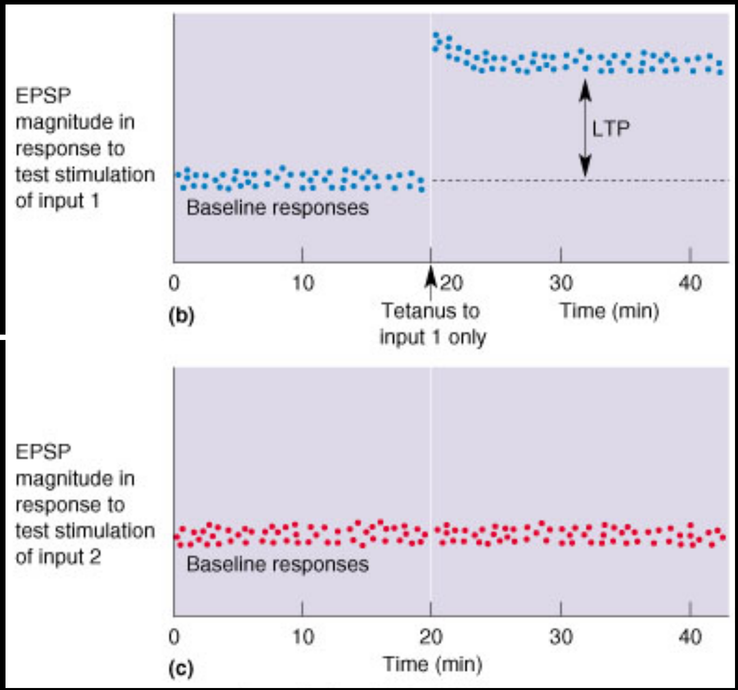
Typical LTP experiment: record EPSP's in CA1 cells (magnitude)

Step 1: weakly stimulate input 1 to establish baseline

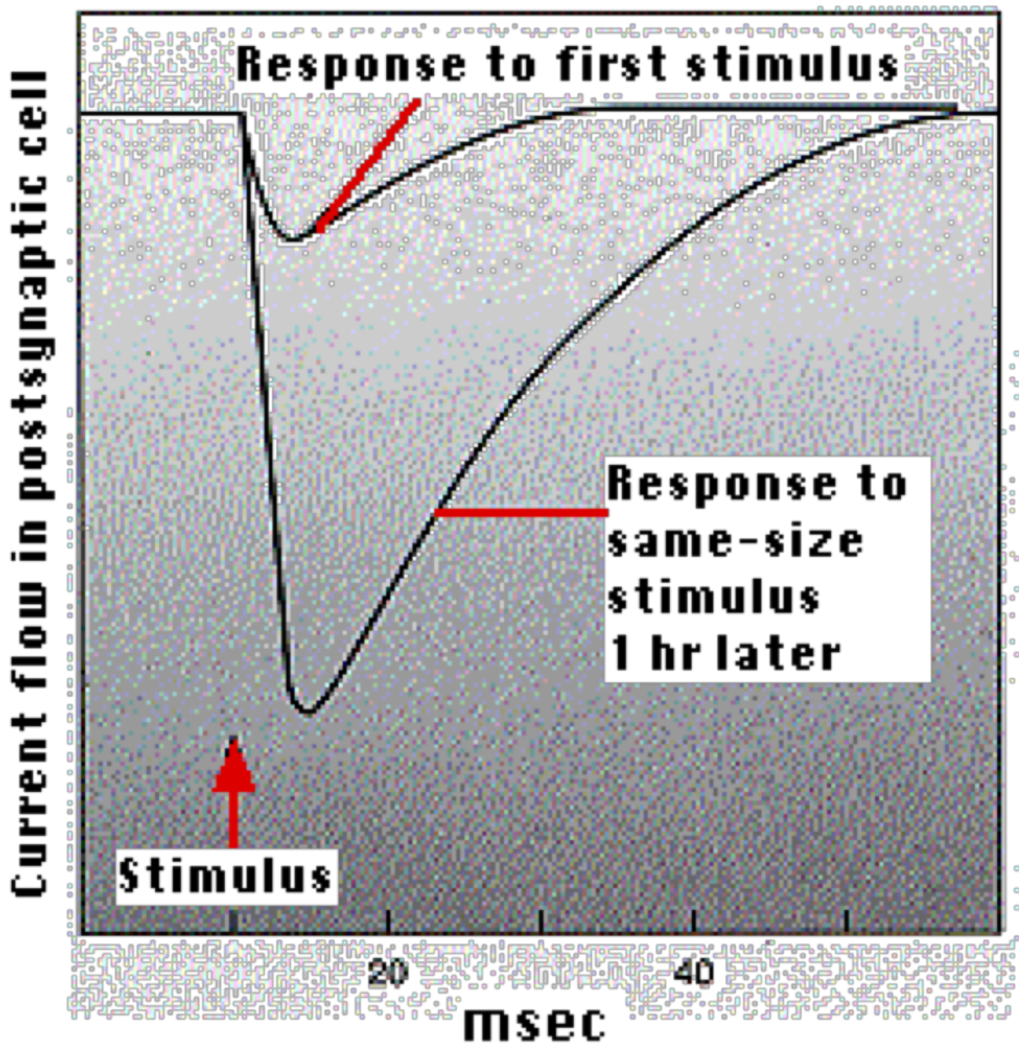
Step 2: give strong stimulus (tetanus) in same fibers (arrow)

Step 3: continue weak stimulation to record increased responses

Step 4: throughout, check for responses in control fibers (input 2)



Normal mice



Response to first stimulus
Response to
same-size
stimulus
1 hr later
Stimulus
Current flow in postsynaptic cell
20
40
msec

LTP

LTP is input specific.

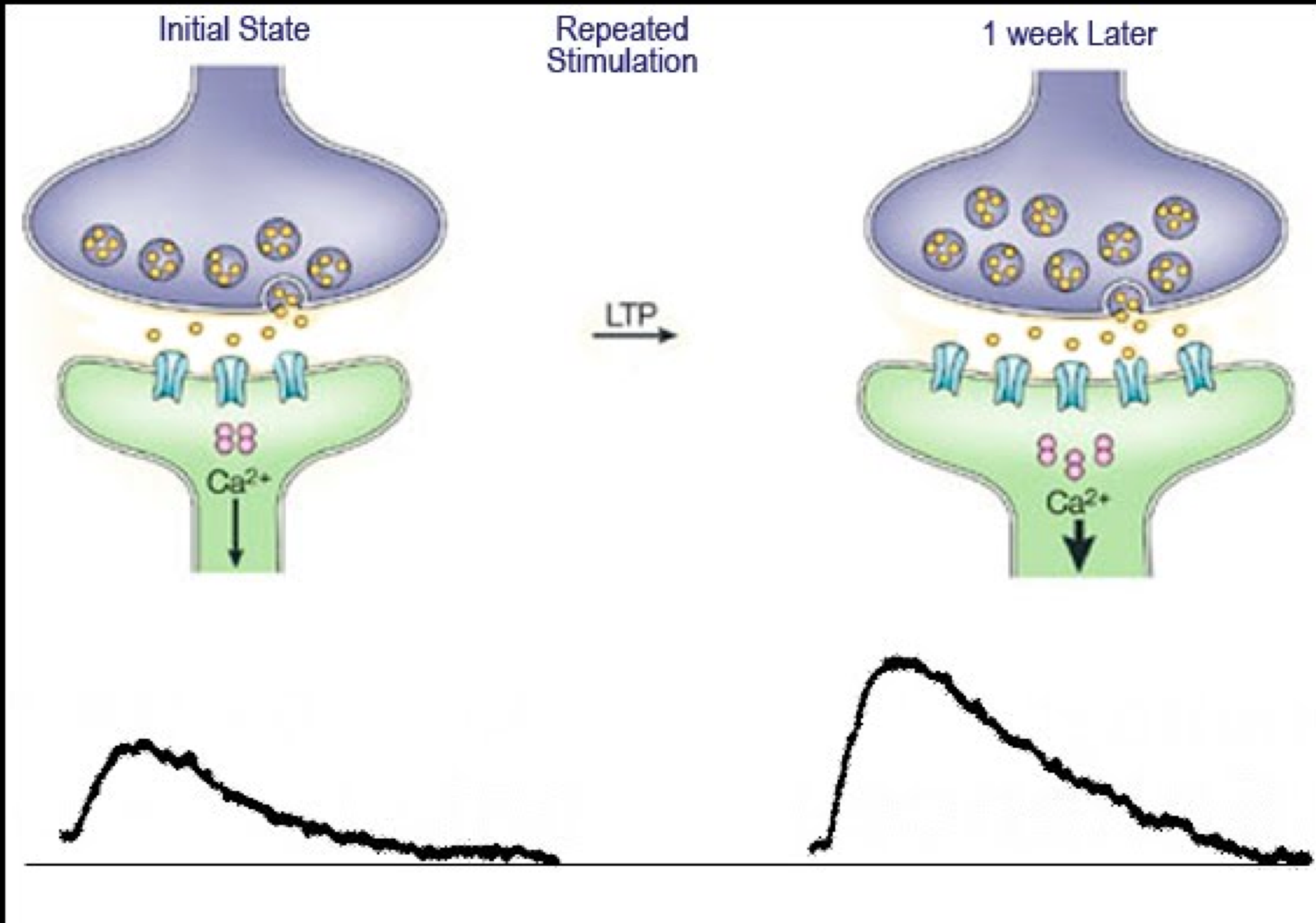
LTP is long-lasting (hours, days, weeks).

LTP results when synaptic stimulation coincides with postsynaptic depolarization (achieved by cooperativity of many coactive synapses during tetanus)(called cooperativity)

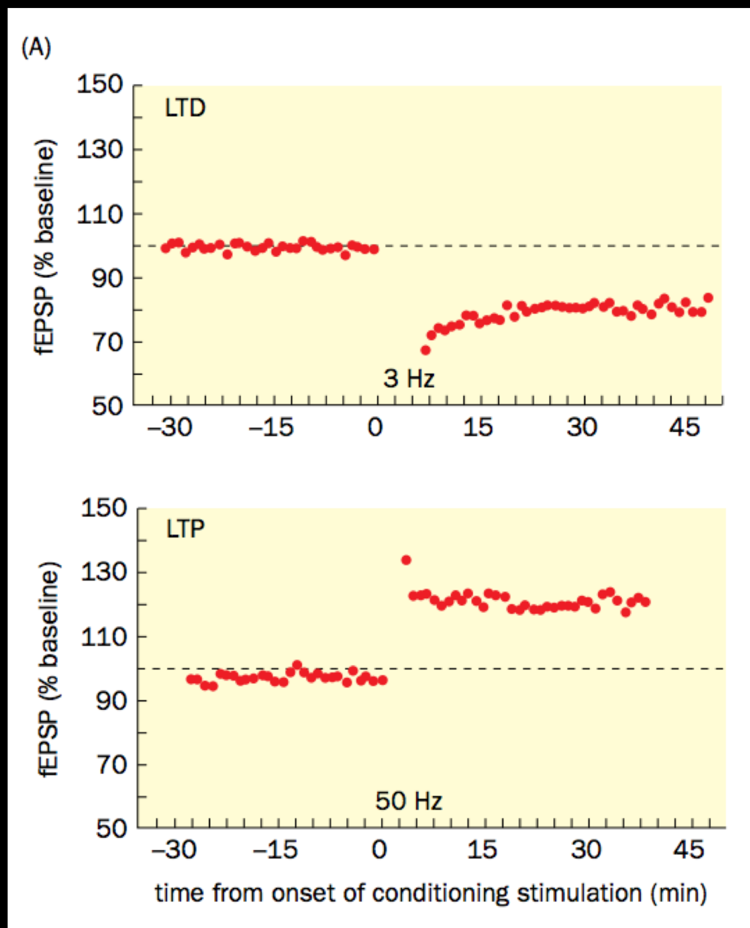
The timing of the postsynaptic response relative to the synaptic inputs is critical.

LTP has Hebbian characteristics ("what fires together wires together", or, in this case, connects together more strongly).

LTP may produce synaptic "sprouting".


Initial State
Repeated
Stimulation
1 week Later
LTP
Ca^{2+}
Ca^{2+}

LTD

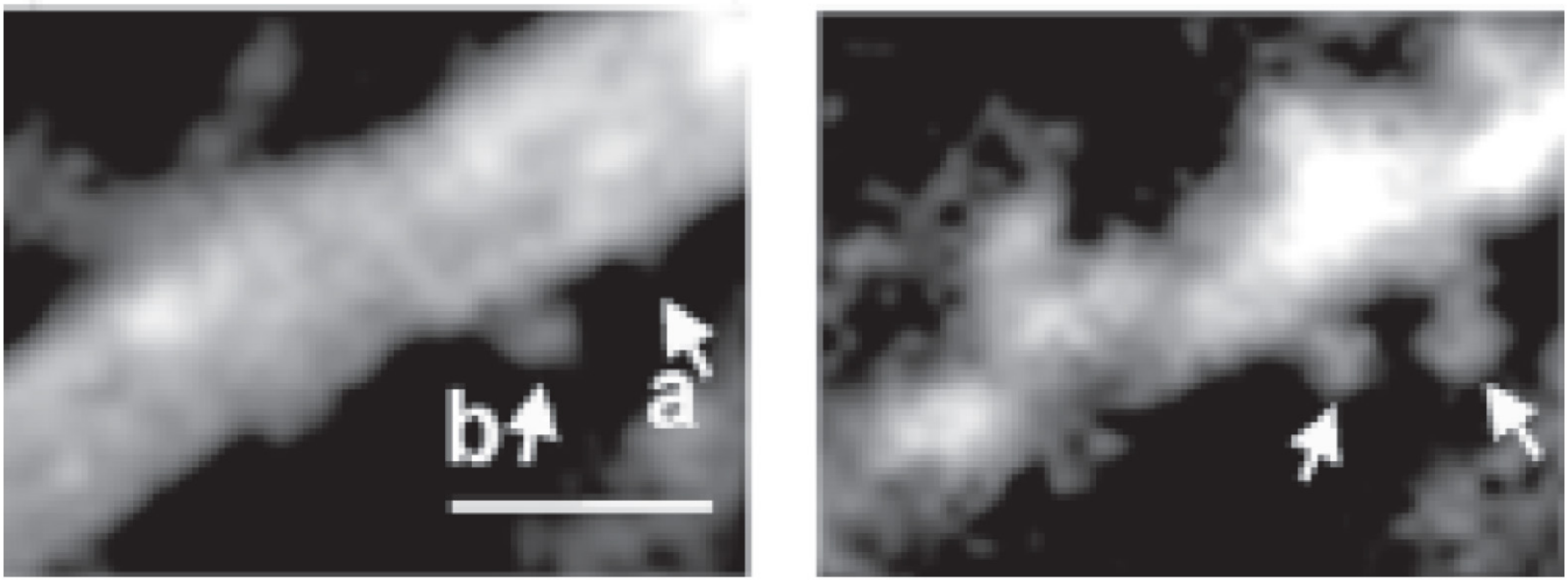


You cannot only have the strengthening of synapses!

You have to be able to weaken bad connections and LTP is the candidate mechanism for this

Synaptic Plasticity

C7B12F10.eps



Before LTP After LTP

[redacted]

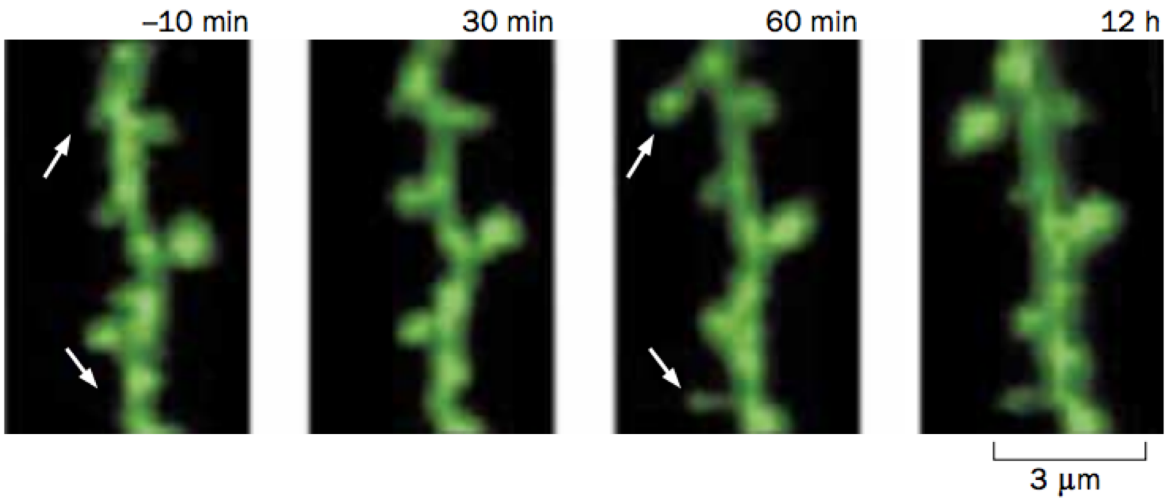
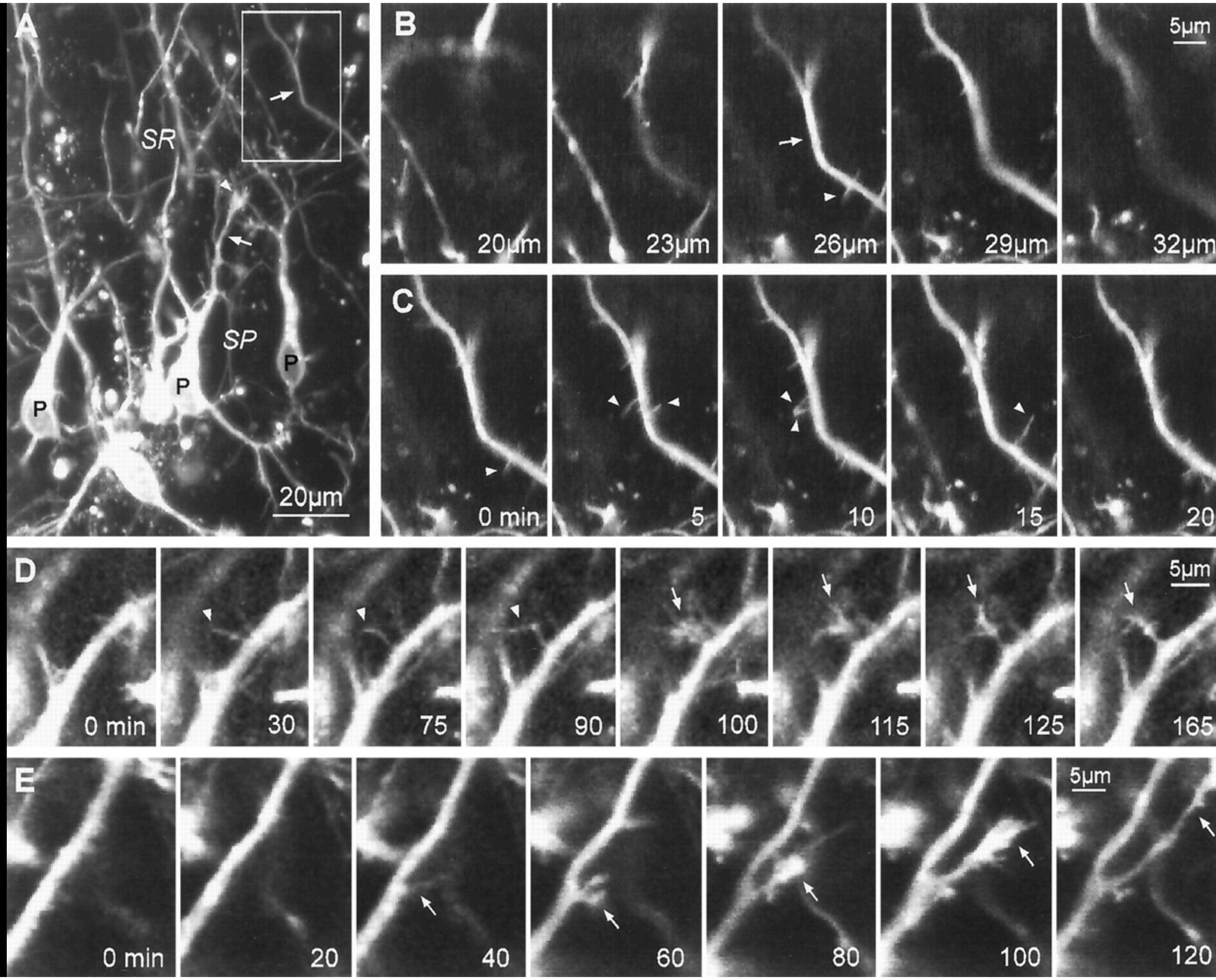
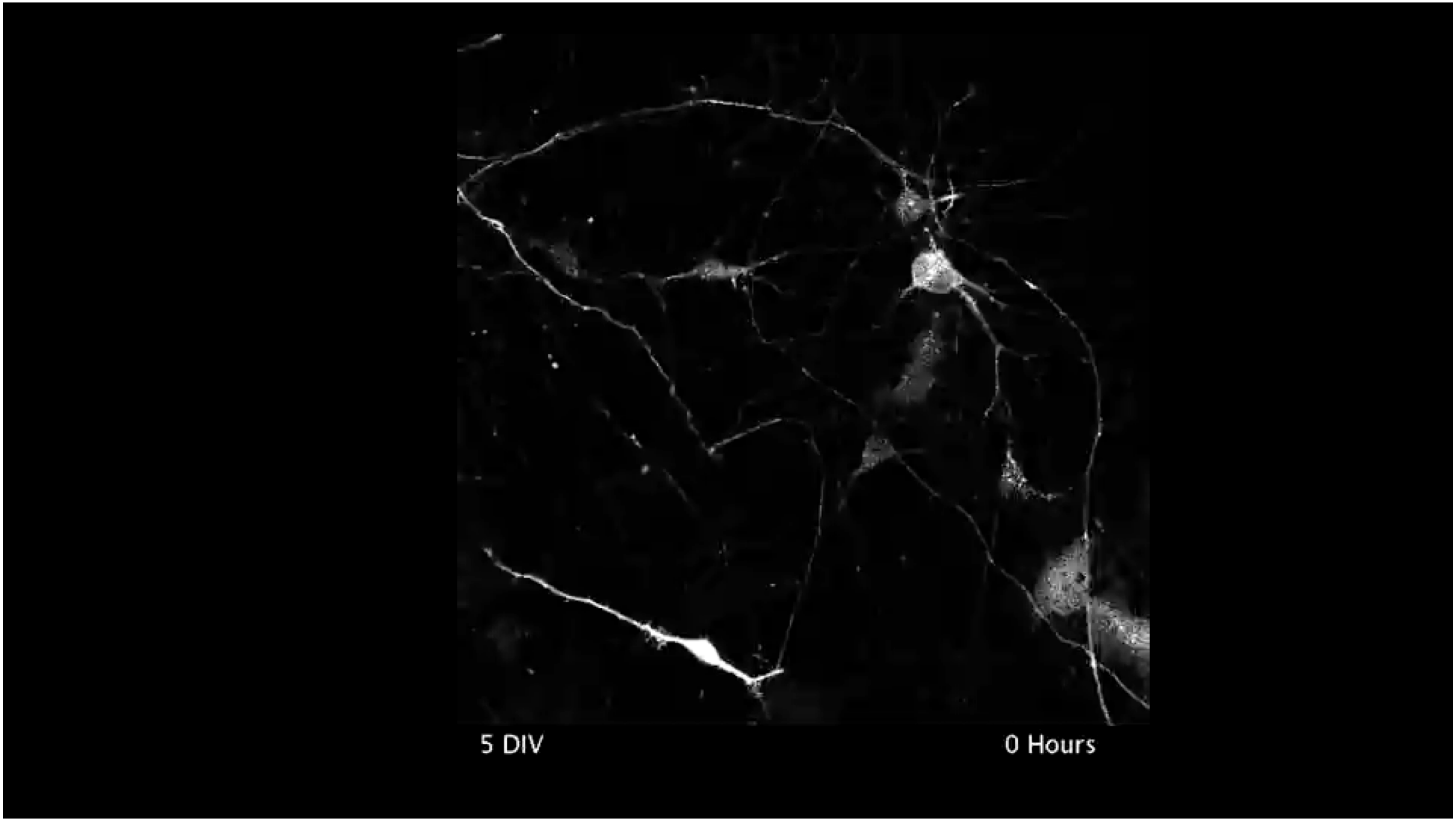


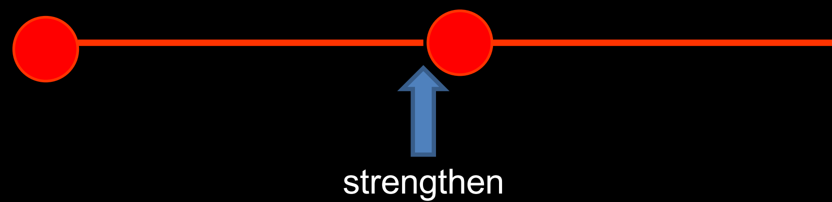
Figure 10–18 Growth of dendritic spines correlates with LTP. LTP is accompanied by the formation of two new spines (arrows) in CA1 pyramidal neurons from a cultured hippocampal slice that was imaged using two-photon microscopy. Time-lapse images were taken at −10, +30, +60 min, and +12 h relative to the onset of LTP induction (not shown). (From Engert F & Bonhoeffer T [1999] *Nature* 399:66–70. With permission from Macmillan Publishers Inc.)

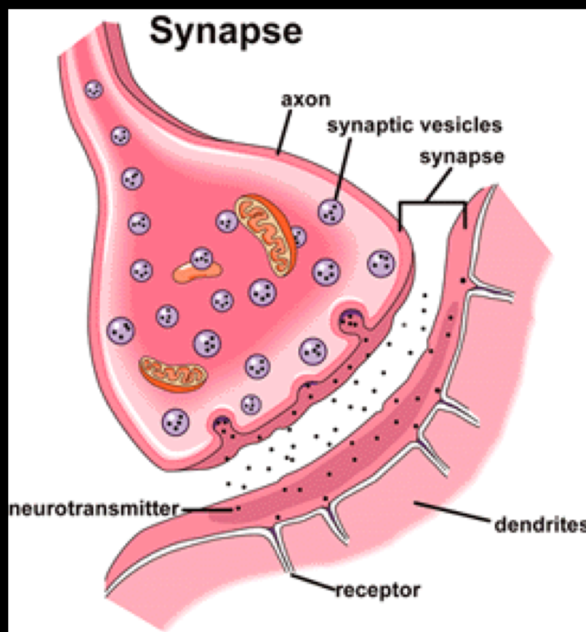
[redacted]



A
SR
SP
P
P
P
20µm
B
20µm
23µm
26µm
29µm
32µm
5µm
C
0 min
5
10
15
20
D
0 min
30
75
90
100
115
125
165
5µm
E
0 min
20
40
60
80
100
120
5µm


5 DIV
0 Hours

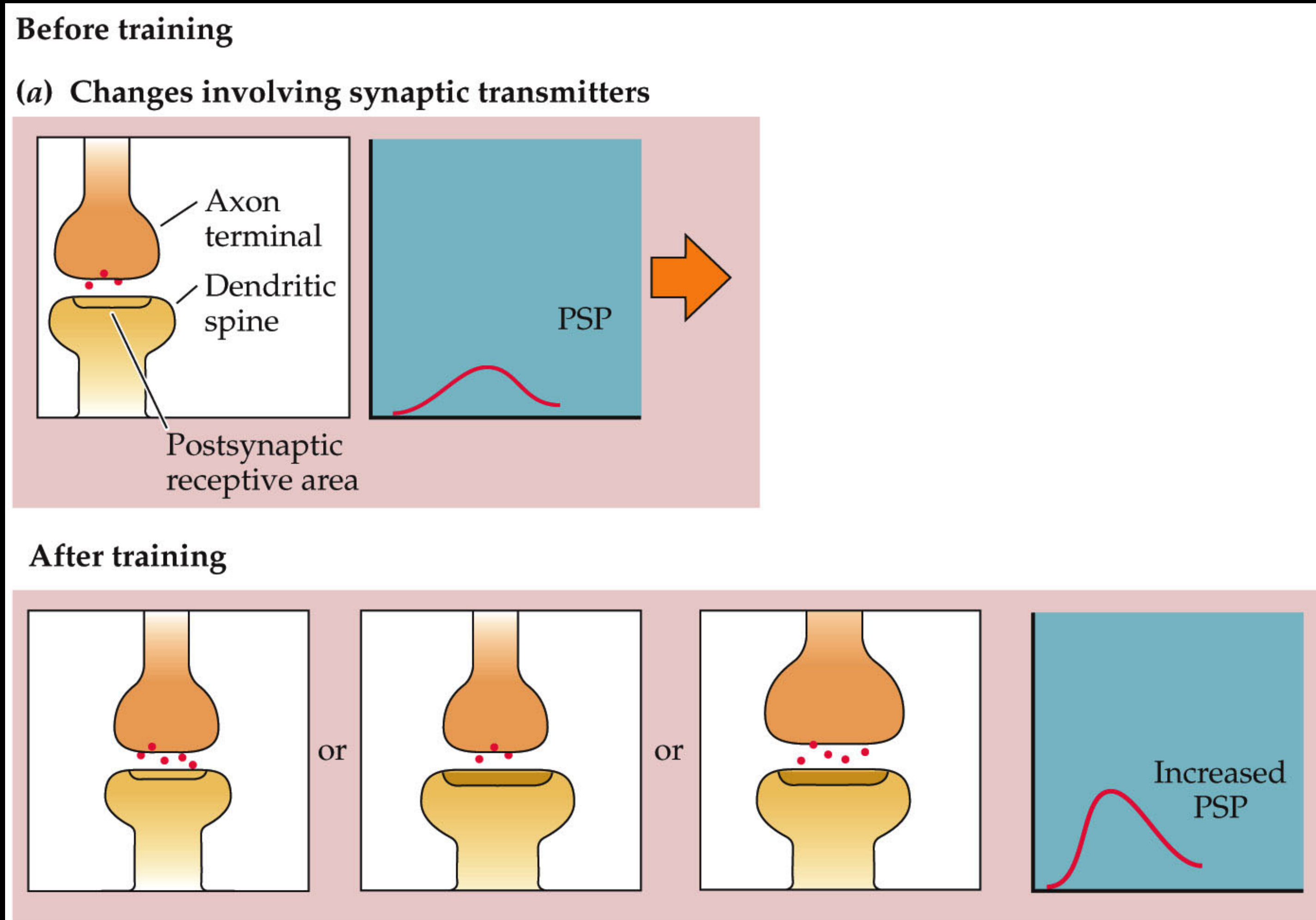




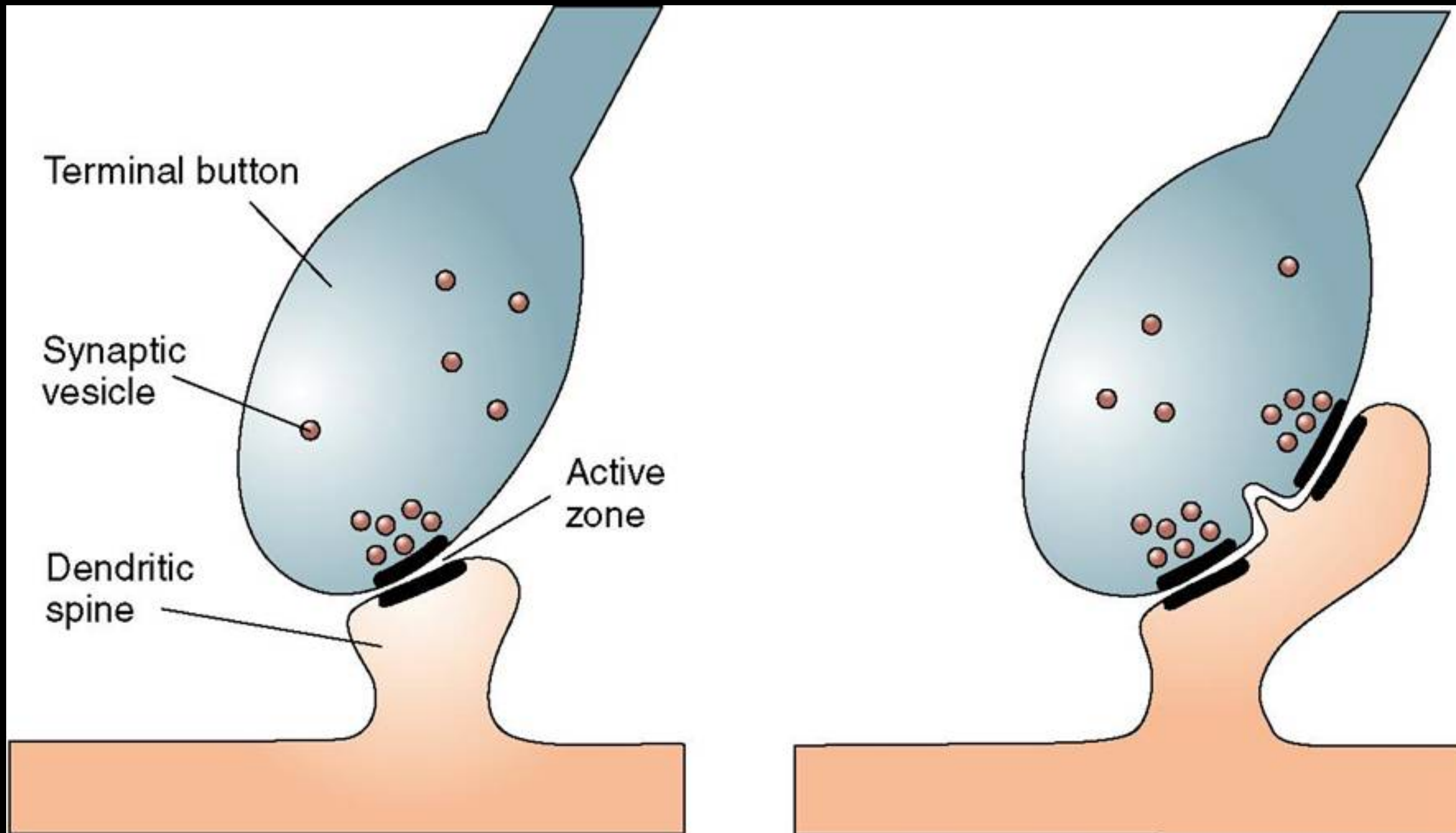
HOW?

Increased neurotransmitter release
Increase receptors
Structural changes

Figure 18.2 Synaptic Changes That May Store Memories (Part 1)



Biological Psychology 5e, **Figure 18.2 (Part 1)**



Before long-term potentiation

After long-term potentiation:
Generation of a perforated synapse

(a)

(b)

So what?

Stronger neural responses and stronger neural connections are more likely to be used again.

As such, a repeated sport skill is more likely to be executed the same way than one that is repeated less often.

Recall the lessons of expertise... 10000 hours!

Thus what does the LTP state do?

Long Term Memory

1. Change in post synaptic neuron

2. Change in pre-synaptic neuron

3. Structural changes (bridging)

** N.B. It is important to note that LTP is the precursor for these LONG TERM changes.

So what we know…