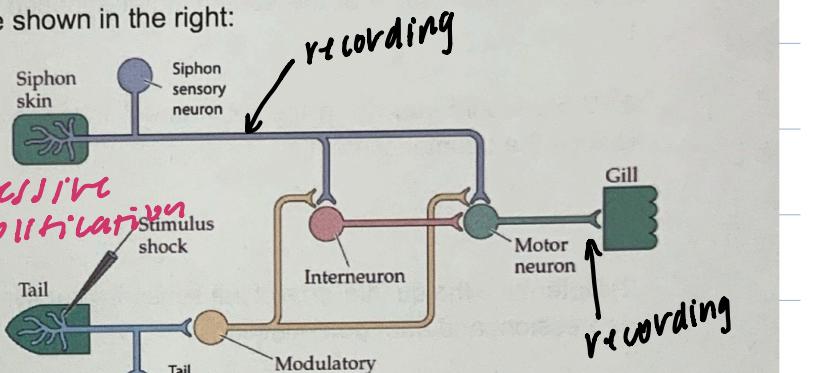


In the 1970's and 1980's, Erik Kandel and his colleagues used *Aplysia californica* to explore the mechanisms of short-term plasticity. Today we will go through some of their key findings.

### Activity 1: Habituation and Sensitization

When touched on its siphon, *Aplysia californica* contracts its gill. This behavior is called the gill-withdrawal reflex. The neural circuits underlying this reflex are shown in the right:

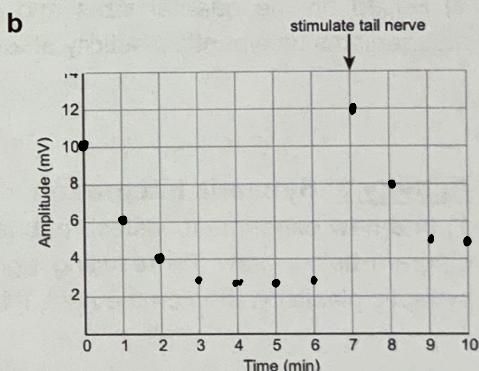
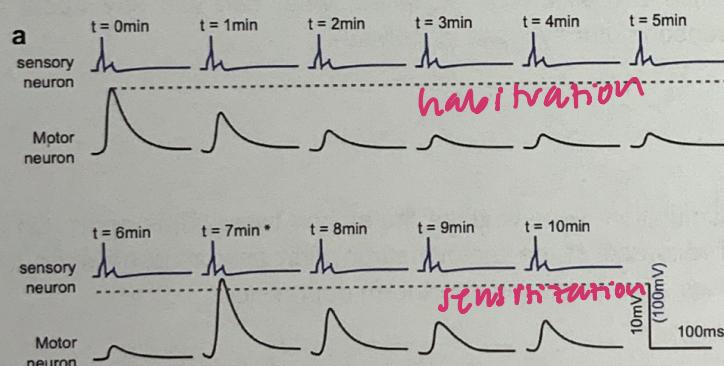
- 1) Define the terms habituation and sensitization. Describe/discuss how to cause habituation and sensitization of the gill-withdrawal reflex in *Aplysia*? *repeated stimulation smaller EPSP*



*repeatedly shock siphon: habituation*

*shock on tail (another area): large sensitization response*

To isolate the underlying circuit changes that cause the behavioral response, you record the activity of the siphon sensory neuron and motor neuron while electrically activating the sensory neuron once per minute. At t = 7min, you pair the stimulation with a strong electrical pulse on the tail (see \* symbol below). The results are shown below:



- 2) On the circuit diagram at top, draw the placement of the two recording electrodes used in this experiment.

- 3) Why are the electrical responses in the two neurons different?

The sensory neuron has an AP when stimulated. EPSP

The motor neuron response is reflective of the response to the stimulus

- 4) Use the recording data from panel a to plot the EPSP amplitude over time in b.  
 5) Describe and interpret panel b. When is depression / potentiation taking place?

↓  
 reflexes are being depressed → longer-lasting (sustainability) of synapses above baseline

### Activity 2: Quantal Analysis

You want to explore the synaptic mechanisms underlying habituation and sensitization. However, the EPSPs are too large to perform quantal analysis, so first you decrease the quantal content and then perform a quantal analysis of synaptic transmission at the sensorimotor synapse. The results are shown on the right:

- 1) What would you do in the experiment (change solutions, add drugs, etc.) reduce the quantal content?

- Drug that increases activity of neurotransmitter degradation enzyme.
- Decrease inhibitory ion ( $\text{Ca}^{2+}$ )

- 2) Determine the quantal size of the sensorimotor synapse before plasticity, after depression, and after potentiation.

before plasticity: 10 mV

after depression: 10 mV

after potentiation: 10 mV

- 3) Using the EPSP sizes from Activity 1, calculate the quantal content for the three stages.

before plasticity:  $10000/10 = 1000$

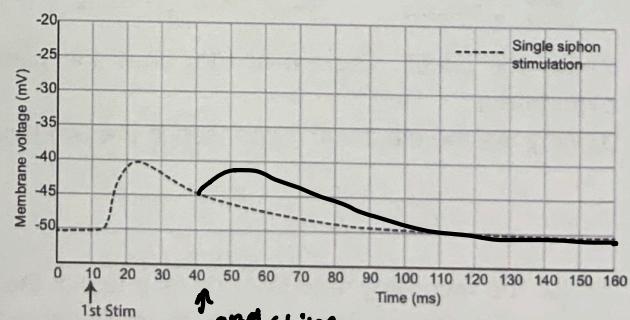
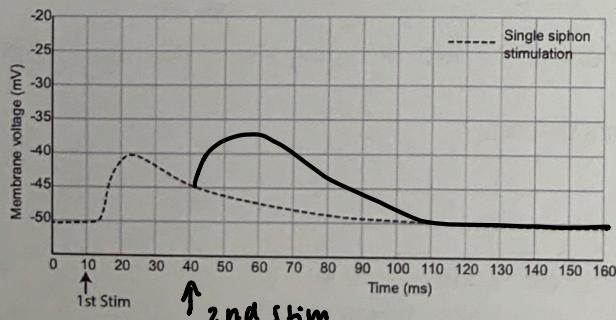
after depression:  $3000/10 = 300$  after potentiation:  $12000/10 = 1200$

- 4) Based on the quantal sizes and quantal contents you calculated, what can you say about the mechanisms of synaptic plasticity at the sensorimotor synapse in Aplysia?

Reduced quantal content but not size.

### Activity 3: Synaptic Integration

- 1) In a new experiment, instead of one stimulation, you stimulate the siphon twice 30ms apart. On the diagram below draw the resulting signal recorded at the motor neuron, first assuming no short-term synaptic plasticity, then on the right, if the synapse displayed short-term depression.



- 2) What mechanism underlies the difference between the two plots?

Depiction of vesicles in the presynaptic neuron

## Exam studying

- previous hours

- objectives

- redo problem sets

EPSP: on the scale of 0 - 30 ms