1. Dr K’s office hours:
   In office   Tuesdays  2:30 - 3:30 PM   (In office: 3122A Pacific Hall)
   Group      Fridays  11:00 AM - noon   (Meeting room: 3501 Pacific Hall)

2. Dr K’s problem-solving session: Wed 5-6 in 3500 Pacific Hall.

3. Dr. K’s e-mail: wkristan@ucsd.edu (put BIPN 100 in the subject line)

4. TA sections, office hours:

<table>
<thead>
<tr>
<th>Day</th>
<th>Time</th>
<th>Section</th>
<th>IA</th>
<th>Office Hour</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fri</td>
<td>11:00-11:50am</td>
<td>SEQUO 148</td>
<td><strong>Winjet Chou</strong></td>
<td>Wed 3-3:50 pm</td>
<td>HSS 1145B</td>
</tr>
<tr>
<td>Fri</td>
<td>12:00-12:50pm</td>
<td>SEQUO 147</td>
<td><strong>Saatchi Patell</strong></td>
<td>Tues 8:40-9:40 am</td>
<td>Muir Woods</td>
</tr>
<tr>
<td>Fri</td>
<td>1:00-1:50pm</td>
<td>WLH 2115</td>
<td><strong>Justine Liang</strong></td>
<td>Tues 10-10:50 am</td>
<td>Hi Thai</td>
</tr>
<tr>
<td>Fri</td>
<td>2:00-2:50pm</td>
<td>WLH 2206</td>
<td><strong>Hao Shi</strong></td>
<td>Fri 3-3:50 pm</td>
<td>HSS 1128a</td>
</tr>
<tr>
<td>Mon</td>
<td>9:00-9:50am</td>
<td>WLH 2115</td>
<td><strong>Tim Macaulay</strong></td>
<td>Tues 12:30-1:20 pm</td>
<td>Mandeville Coffee Cart</td>
</tr>
<tr>
<td>Mon</td>
<td>4:00-4:50pm</td>
<td>WLH 2208</td>
<td><strong>Mallorie Nguyen</strong></td>
<td>Thurs 2-2:50 pm</td>
<td>Goody's Market</td>
</tr>
<tr>
<td>Mon</td>
<td>5:00-5:50pm</td>
<td>WLH 2208</td>
<td><strong>Donel Purcella</strong></td>
<td>Mon 3:30-4:20 pm</td>
<td>Price Center East, SR 2</td>
</tr>
<tr>
<td>Mon</td>
<td>6:00-6:50pm</td>
<td>WLH 2115</td>
<td><strong>Kyra Rashid</strong></td>
<td>Thurs 10-10:40 am</td>
<td>Revelle Commuter Lounge</td>
</tr>
</tbody>
</table>
In notes for Lecture #1, this is Fig 1.7:

![Diagram of a control system with set point, comparing unit, controller, and controlled variable.]

Fig. 1.7

.....not this:

![Diagram with incorrect connections and labels.]

Fig. 1.7
In Lecture 2 notes, top of p. 6:

h. Note well: This is NOT an equilibrium potential for any of the ion channels! There is a sizable current across every one of the channels. In fact, can calculate how much current flows through each of them:

\[ I_K = (V_m - E_K) G_K = (-66.5 - (-90)) \times 80 = +1.88 \text{ nA} \]
\[ I_{Na} = (V_m - E_{Na}) G_{Na} = (-66.5 - 60) \times 15 = -1.90 \text{ nA} \]
\[ I_{Cl} = (V_m - E_{Cl}) G_{Cl} = (-66.5 - (-70)) \times 5 = +0.02 \text{ nA} \]

So: a large amount of K\(^+\) flows out of the cell (because the \(G_K\) is so large), and a nearly equal amount of Na\(^+\) flows into the cell (because \(V_m\) is so far from \(E_{Na}\)), and very little Cl\(^-\) flows into the cell (because \(E_{Cl}\) is close to \(V_m\) and because \(G_{Cl}\) is so small).
very little Cl⁻ flows into the cell (because $E_{\text{Cl}}$ is close to $V_m$ and because $G_{\text{Cl}}$ is so small).
Lecture 2,3 presented the ionic basis of resting potentials and action potentials in neurons

Resting potential

Results from ongoing leakage of ions (mostly Na\(^+\), K\(^+\), and Cl\(^-\)) across the membrane.

- through specific membrane-spanning proteins called channels.
- each channel type generates a voltage (equilibrium potential) based upon its concentration gradient across the membrane (*Nernst Equation*).

The amplitude of the resting potential is a sum of the amplitudes of the equilibrium potentials of the ions weighted by their relative conductances (*Chord Conductance Equation*).

Action potential

Results from the activation of specialized Na\(^+\) and K\(^+\) channels by a depolarizing voltage.

- the voltage-gated Na\(^+\) channels are faster and shut off automatically ("inactivation")
- the voltage-gated K\(^+\) channels are slower and shut off only by loss of depolarization.

The inactivation of Na\(^+\) channels and prolonged time-course of the K\(^+\) channels causes a decreased excitability ("refractoriness") of the membrane.

- the refractoriness insures that the action potentials conduct in only one direction.

Action potentials move along an axon at a rate determined by (1) axon diameter, and (2) myelination.
Function of myelin: Saltatory conduction (for speed)

---

Silverthorn, 5th Ed., p. 271
Neurons generate four kinds of potentials we need to consider:

1. Resting potential (all cells)
2. Action potential (neurons, muscle fibers, some hormonal cells)
3. Synaptic potential (neurons and muscle fibers)
4. Receptor potential (only sensory neurons)
The frog neuromuscular junction

Neuromuscular transmission: leopard frog (*Rana pipiens*)

Bernard Katz
1911-2003
The neuromuscular junction (NMJ), a model synapse
Chemical synaptic transmission
Basis of excitatory synaptic reversal potentials

EPSP (NMJ) reversal potential

Membrane potential

$E_{Na} \quad +60 \text{ mV}$
$+30 \text{ mV}$

$E_{rev} \quad 0 \text{ mV}$
$-30 \text{ mV}$

$E_K \quad -100 \text{ mV}$
$-90 \text{ mV}$

$-70 \text{ mV}$

$-30 \text{ mV}$

$h_a + k = \text{Net current}$
Postsynaptic potentials (PSPs) are of two types:

- Some PSPs cause hyperpolarization:
  - IPSP
  - Notice the amplitude of PSPs compared with action potentials.

- Others cause depolarization:
  - EPSP
  - Notice how long PSPs last compared with action potentials.

PSPs are typically smaller and slower than action potentials.
IPSP reversal potential in a neuron.

-52 mV
-68 mV
-88 mV
-108 mV
-126 mV

$V_{\text{threshold}}$
$V_{\text{rest}}$
$V_{\text{reversal}}$

10 mV
10 msec
Whether the PSP is excitatory (EPSP) or inhibitory (IPSP) depends upon the location of the reversal potential \( V_{\text{EPSP}} \) or \( V_{\text{IPSP}} \) relative to the threshold potential \( V_{\text{threshold}} \).
The reversal potentials for most IPSPs are hyperpolarizing from the resting potential:

....but some IPSP reversal potentials depolarize from the resting potential:
### SUMMARY: Action Potentials vs. Synaptic Potentials

<table>
<thead>
<tr>
<th></th>
<th>Action potentials</th>
<th>EPSPs (at NMJ)</th>
<th>IPSPs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effect of depolarization:</td>
<td>Initiates APs</td>
<td>Decreases $V_{\text{EPSP}}$</td>
<td>It's complicated...</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No effect on $G_{\text{EPSP}}$</td>
<td>No effect on $G_{\text{IPSP}}$</td>
</tr>
<tr>
<td>Neurotransmitter effect:</td>
<td>No effect</td>
<td>$ACh$ increases $G_{\text{ions}}$</td>
<td>Transmitter increases $G_{\text{ions}}$</td>
</tr>
<tr>
<td>Response regenerative or graded?</td>
<td>Regenerative</td>
<td>Graded with amount of ACh</td>
<td>Graded with amount of transmitter</td>
</tr>
<tr>
<td>Ions involved:</td>
<td>$Na^+$, $K^+$</td>
<td>$Na^+$, $K^+$</td>
<td>$K^+$ and/or $Cl^-$</td>
</tr>
<tr>
<td>and sometimes $Ca^{++}$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ions move through same or separate channels?</td>
<td>Separate</td>
<td>Same</td>
<td>Separate</td>
</tr>
<tr>
<td>Effect of TTX:</td>
<td>Blocked</td>
<td>Ne effect</td>
<td>Ne effect</td>
</tr>
<tr>
<td>Effect of TEA:</td>
<td>Prolonged</td>
<td>No effect</td>
<td>No effect</td>
</tr>
<tr>
<td>Effect of curare:</td>
<td>No effect</td>
<td>Blocked</td>
<td>No effect</td>
</tr>
</tbody>
</table>
Mechanisms of fast and slow postsynaptic responses.

Fig. 8-23 in Silverthorn, 6th edition
Mechanisms for inactivating neurotransmitters

Fig. 8-24 in Silverthorn, 6th edition
**Post-synaptic neuron:**

**Spatial summation**
- Action potential in Input 1
- Action potential in Input 2
- Simultaneous action potentials in both Input 1 and Input 2

**Temporal summation**
- Action potential in Input 1
- 2 action potentials in rapid succession in Input 1
<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Typical effects¹</th>
<th>Structure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine (Ach)</td>
<td>Fast excitation; slow inhibition</td>
<td><img src="image" alt="Acetylcholine Structure" /></td>
</tr>
<tr>
<td>Glycine (Gly)</td>
<td>Fast inhibition</td>
<td><img src="image" alt="Glycine Structure" /></td>
</tr>
<tr>
<td>γ-aminobutyric acid (GABA)</td>
<td>Fast inhibition; slow inhibition</td>
<td><img src="image" alt="GABA Structure" /></td>
</tr>
<tr>
<td>Glutamate (Glu)</td>
<td>Fast excitation; slow change in postsynaptic metabolism</td>
<td><img src="image" alt="Glutamate Structure" /></td>
</tr>
<tr>
<td>Norepinephrine (Nor-epi)</td>
<td>Slow excitation; slow inhibition</td>
<td><img src="image" alt="Norepinephrine Structure" /></td>
</tr>
<tr>
<td>Dopamine</td>
<td>Differs with location but causes slow postsynaptic effects</td>
<td><img src="image" alt="Dopamine Structure" /></td>
</tr>
<tr>
<td>Serotonin (5-HT = 5-hydroxytryptamine)</td>
<td>Slow excitation or slow inhibition</td>
<td><img src="image" alt="Serotonin Structure" /></td>
</tr>
<tr>
<td>Nitrogen oxide (NO)</td>
<td>Synaptic modulation</td>
<td><img src="image" alt="Nitrogen Oxide Structure" /></td>
</tr>
<tr>
<td>Adenosine triphosphate (ATP)</td>
<td>Both fast and slow synaptic transmission</td>
<td><img src="image" alt="ATP Structure" /></td>
</tr>
<tr>
<td>Histamine</td>
<td>Slow modulation</td>
<td><img src="image" alt="Histamine Structure" /></td>
</tr>
</tbody>
</table>

¹ Notice that the effect of a neurotransmitter depends on the properties of the post-synaptic cell. However, for most neurotransmitters it is possible to identify their most probable effect.
Channels and potentials (voltages)

Membrane potential is the voltage measured across the cell membrane; it is represented as $V_m$.

In these neurons, there are three kinds of membrane potentials:

<table>
<thead>
<tr>
<th>Channel type</th>
<th>Ions responsible</th>
<th>Type of membrane potential</th>
</tr>
</thead>
<tbody>
<tr>
<td>“leak”</td>
<td>$K^+$, $Na^+$, $Cl^-$</td>
<td>resting potential</td>
</tr>
<tr>
<td>voltage-gated</td>
<td>$Na^+$, $Ca^{++}$, $K^+$</td>
<td>action potential</td>
</tr>
<tr>
<td>ligand-gated</td>
<td>$Na^+$ + $K^+$, $K^+$ + $Cl^-$</td>
<td>synaptic potential</td>
</tr>
</tbody>
</table>