Quantal Analysis

The experiment:

**epp** = end-plate potential (general term is epsp for excitatory post-synaptic potential)
**mepp** = miniature end-plate potential (general term is mepsp) = quantal unit

**Quantal hypothesis**: Single, spontaneous quantal events (mepps) represent the building blocks of for the synaptic potentials evoked by stimulation (epps).

\[ m = \text{“quantal content”} = \text{mean number of quanta (a.k.a. vesicles) that are released to make up the end-plate potential (epp)} \]

There are two ways of calculating \( m \).

**First method**: (this is essentially a restatement of the hypothesis!)

\[ m_1 = \frac{epp}{mepp} \]

- \( epp \) = mean amplitude of epp response (the post-synaptic response to one or usually more quanta being released)
- \( mepp \) = mean amplitude of miniature epp (in response to one quanta released)

**Second method**: (probabilistic)

Failures and variability in EPP amplitude implied probabilistic nature of transmission

Katz and colleagues considered models in which there were \( n \) quanta (vesicles) available for release with probability \( p \).

\[ m_2 = n \times p \]

One special case that accounts for situations where \( n \) is very large, and \( p \) is very small is Poisson statistics. After a little bit of math, you can arrive at:

\[ m_2 = \ln \left( \frac{\text{trials}}{\text{failures}} \right) \]
If \( m_1 = m_2 \), then transmitter release from vesicles obeys Poisson statistics.

Some definitions:

**Quantal content (m):** (see definition on first page) = quantal number: the number of quanta that are released, measured by the size of the epp in mV. It is modulated pre-synaptically by changing transmitter release.

**Quantal size:** the size of 1 quanta, measured as the smallest post-synaptic depolarization (mepp) in mV, i.e. the response to a single vesicle being released. It is modulated post-synaptically by changing the response to transmitter release.

<table>
<thead>
<tr>
<th>Perturbation</th>
<th>mepp</th>
<th>epp</th>
<th>quantal content (m)</th>
<th>quantal size</th>
<th>pre or post?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease ([Ca^{2+}]_{ext})</td>
<td>⇓</td>
<td>⇑</td>
<td>⇓</td>
<td>⇓</td>
<td>pre</td>
</tr>
<tr>
<td>Increase # receptors on post-synaptic terminal</td>
<td>↑</td>
<td>↑</td>
<td>⇓</td>
<td>↑</td>
<td>post</td>
</tr>
<tr>
<td>Add botulinum toxin</td>
<td>⇓</td>
<td>⇑</td>
<td>⇓</td>
<td>⇓</td>
<td>pre</td>
</tr>
<tr>
<td>Increase # voltage-gated (Ca^{2+}) channels on pre-synaptic terminal</td>
<td>⇓</td>
<td>↑</td>
<td>↑</td>
<td>⇓</td>
<td>pre</td>
</tr>
<tr>
<td>Add serotonin to the bath (provided the pre-synaptic cell responds to serotonin)*</td>
<td>⇓</td>
<td>↑</td>
<td>↑</td>
<td>⇓</td>
<td>pre, facilitation</td>
</tr>
</tbody>
</table>

* serotonin (5-HT) can have different roles. In class, we saw that a particular 5-HT receptor, a GPCR, leads to a signaling cascade where the formation of cAMP activates protein kinase A, which in this case leads to the closing of K+ channels. As a result, the post-synaptic cell’s response is longer (see slide 20 of lecture 8 for all the steps.)