Spring Brain Conference

Neuroscience Teaching Team

The Synapse

SYNAPSE DEMO - Students will learn about how neurons communicate with each other through synaptic activity, and how synaptic activity can be altered through drug use and in disease.

I. Brain is made up of nerve cells
   - A. Show brain, neuron, parts of the neuron
   - B. How many neurons are in the brain? (~100 billion)
   - C. How do neurons communicate with each other?
     - i. What is the synapse?
     - ii. Neurotransmitters ("signaling molecules")?
     - iii. Receptors?
   - D. Can you think of examples in which the communication between brain cells is altered or abnormal?
     - i. Drugs
     - ii. Disease/neurological disorders

II. Synapse Demo
   - A. There are three **main concepts** that we want the students to learn about synaptic activity:
     - i. Baseline synaptic activity (show synapse):
       1. How neurons communicate with each other.
       2. There are different types of neurotransmitters (NT) in the brain that do different things (some excitatory--increase excitation in post-synaptic cell; some inhibitory—reduce excitation in post-synaptic cell).
     - ii. How drugs affect synaptic activity:
       1. Initial drug exposure/acclimation: increase NT release, increase activation of post-synaptic cell—depending on age group and time, can explain excitatory and inhibitory neurotransmitters in the context of stimulants/depressants.
       2. Tolerance: post-synaptic cell wants to bring activity back to normal, so it removes some receptors. It then takes more drugs to elicit the same response as occurred after the initial use. ("homeostasis": back to normal)
       3. Withdrawal: once the drug is removed, there are fewer receptors and fewer NT being released, so the post-synaptic cell has much less activity. Symptoms of withdrawal are usually opposite of the drug-induced ones (e.g. amphetamines cause hyper-activity/alertness, so amphetamine withdrawal would cause sleepiness)
     - iii. How different brain disorders/diseases could effect synaptic activity:
       1. Schizophrenia: increased activity of dopamine.
          a. Treatment: blocking dopamine receptors
       2. Epilepsy: increased glutamate activity
          a. Treatment: blocking glutamate receptors, or increasing GABA.
       3. Parkinson's disease: loss of dopamine
          a. Treatment: increase dopamine (in this case, there can also be tolerance/withdrawal.)

Each state will be given about 4 minutes for explanation (see above) and 15 sec of game time. The main instructor will announce the time schedule as the class proceeds.

   - B. Rules of the game:
     - i. Students must use fingers to flick a ping-pong ball (neurotransmitter/signaling molecule) across the game board (synapse) and have it attach to a receptor on the

other side. The game board should be propped up so that balls roll back down if they do not attach to a receptor. Once a ball is attached to a receptor, it stays there until the minute is over. If a ball gets stuck but is not physically Velcro-ed to a receptor, push it back down to the bottom. After the 15 sec is over, the score (# of balls attached to receptors) should be reported by a student.

C. Synapse Conditions:

i. Normal synapse: 4 balls, 4 receptors. (homeostasis)

1. Explain that the ping-pong balls are neurotransmitters, what the receptors are, the big picture of what the game-board represents (see above, show picture of synapse).
2. It's very important that the students make the connection that the more balls they get stuck to the receptor, the more neuron #2 responds (i.e. giving you pleasure, pain, thought, etc.)

ii. Drugs: 8 balls, 4 receptors

1. Adding more balls (neurotransmitters) mimics the brain's response to drugs. Therefore, nerve #2 gets more excited.
2. For example, use of amphetamines (stimulants) causes increased synaptic activity of the neurochemicals dopamine and neuroepinephrine.
    a. Some examples of amphetamines are diet pills, Ritalin, and cocaine.
3. The use of some depressants causes increased synaptic activity of inhibitory neurotransmitters (which has an end effect of reduced excitation, or inhibition) *This might be confusing, since the end result of increased neurotransmission is reduced excitation, or inhibition.*
    a. Some examples of depressants include alchohol and tranquilizers.

iii. Tolerance: 8 balls, ~~4~~ 2 receptors

1. The brain adapts to the high level of drug-induced signals by reducing the number of receptors (to try to bring it back to 'normal'). It then takes more drug (hence more signal) to get the same response from neuron #2.

iv. Withdrawal: 2 balls, 2 receptors.

1. When a person stops taking the drug, the synapse is starved of activity (less balls/neurotransmitters/signal);
2. The result is the opposite of when you were taking the drug (e.g. amphetamine withdrawal symptons include sleepiness, depression, sick).

v. Disease/disorders:

1. Some brain disorders are due to too much signal/activity/neurotransmitter release.
    a. Epilepsy (glutamate/GABA): It occurs in about 1 in every 100-200 people.
        i. Over-activation of neurons (by increased glutamate (increased excitation) or decreased GABA (decreased inhibition)) causes seizures.
    b. Schizophrenia (dopamine): About 1 of every 100 people (1% of the population) is affected by schizophrenia.
        i. Increased dopamine transmission can cause symptoms like hallucinations, disorganized speech, delusions.

c. Parkinson's disease (loss of dopamine neurons): In the United States, at least 500,000 people are believed to suffer from Parkinson's disease, and about 50,000 new cases are reported annually.
    i. Reduced dopamine can cause symptoms like resting tremor, difficulty/slow moving, inability to move/speak.

2. These disorders can be treated using drugs that change synaptic activity, either by blocking the receptors (to reduce reduce synaptic activity) or by increasing neurotransmitter levels (to increase synaptic activity).
    a. Epilepsy drugs: Antiepileptic drugs work by reducing the abnormal firing of cortical neurons. These drugs may change the activity of neurotransmitters responsible for seizures (e.g. block glutamate receptors (to reduce excitation) or increase GABA activity (to inhibit excitation).
    b. Schizophrenia drugs: block dopamine and some serotonin receptors
    c. Parkinson's drugs: stimulate dopamine production to increase dopamine levels at the synapse.
    d. *Depending on time and age group, it might be interesting to explain how increased usage of anti-schizophrenia drugs produce parkinsonian symptoms (b/c of decreased dopamine), and how anti-parkinsonian drugs can produce schizophrenia-like symptoms (b/c of increased dopamine).*