Lecture 13 Neurodevelopment, Brain damage and Regeneration

1. During development of the central nervous system, several different stages (7 of them) lead to maturation of a functional and healthy brain. What are the stages?
2. What in general neural plate/tube eventually lead to becoming?
3. Proliferation is what? What is a stem cell, a progenitor cell, blast cell and a specialized cell? How do these cells match up with differentiation of a stem cell to a Specialized cell
4. In general we have 4 classes of cells; epithelial cells (ex skin cells), muscle cells (ex heart), bone cells and neural type cells. I only really care about neural cells. What is a definition of neural cell? What do they do? What kinds are there (keep in mind Neural cells are further categorized into Neurons (many kinds) and glial cells (many kins too))?
5. What is migration refer to?
6. What function does radial glial provide to migrating cells? Where is the cell portion of the radial glial found? This is also the location were Stems cells grow and differentiate start to migrate from.
7. Radial glial cells create a scaffolding complex which becomes what in a developed brain? The layers in the cortex!
8. What 2 chemicals help promote migration of neural cells? Crudely what attracts or repels cells? Is it the chemical or concentration?
9. The general class of chemicals that helps keep neurons alive is known as?
10. What is the sequence of events in the developing CNS, i.e. What is the order of events neural migration, genesis and maturation?
11. When does the bulk of neurogenesis, neural-migration and neural maturation take place? I.e. Before birth or after birth?
12. Neural maturation involves what 3 developmental aspects of the neuron?
13. Differentiation and specialization of cells into neurons or glial cells also depends on surrounding cells that they interact with!
14. What is neural Darwinism?
15. Although, neurogenesis and neural-migration are complete before birth, why does the brain increase in size and weight after birth????????
16. What and when does specialization, myelination and synaptogenesis occur? What happens during these events?
17. When does synaptogenesis primarily occur and when does synaptic pruning occur? How does this relate to the increase in brain weight and also behavior?

18. True (a) or False (b)? In humans, the phenomenon of axon myelination is usually complete by birth.


20. What is 2 examples given in class that demonstrate that environmental input or experience continue to modify the CNS (modify neural maturation process all 3!) after birth? What changes in the CNS are produced from these experiments?

21. What is fetal alcohol syndrome?

22. Do specialized skills learned early in life promote modifications in the brain? If so how?

23. Very crudely what is Epigenetic?

24. The brain can be damaged via multiple events or things what are some? Traumatic Brain Injury (TBI), Cerebrovascular Injury (CVI), Tumors, Anoxia (e.g., near drowning, drug overdose), Toxins (e.g., lead), Diseases (e.g., Herpes, encephalitis), Neurodegeneration, Seizures, Infections, Degenerative diseases, Closed head injuries: Acute blow drives brain tissue against inside wall of skull. Know in general what is characteristic of these different brain injuries.

25. What is tPA used to treat, what does it do?

26. What are additional treatment to reduce the severity of strokes (hemorrhages and aneurysms)? Note strokes lead to glutamate excitotoxicity, so there is a line of offensive drug treatments that all help reduce the severity of damage.

27. Plasticity after brain damage happens through either regeneration or reorganization, what is difference between these?

28. What makes regeneration successful?

29. regeneration or reorganization involves the axon, what about generation of new neurons in adult brains? What 2 or 3 brain regions can make en neurons?

30. What 2 environmental influences can promote an increase in Dentate gyrus production of new neurons?
31. What are some treatments that can help axonal growth and synaptogenesis? What are the cell types that help or inhibit axonal growth? And where are they found?

32. What is one reason stem cells are not good treatment options in regrowing severed neurons?

33. What type of immature cell has recently been proven to be a good treatment for rebuilding severed neuronal connections? Why and what is unique about these cells, i.e. What do they promote and what do they help suppress?

**Lecture 14 Learning and Memory**

1. What is the definition of Learning and Memory giving in class? What is accusation-consolidation-retrieval refer to?
2. How do we infer that learning has taken place and memory too.
3. I described 2 categories of learning or acquisition of information (Non-associative and Associative), what are sub categories of these learning processes? And know the examples I give to help understand the differences.
4. The differences between Habituation/sensitization to - classical conditioning to - instrumental conditions is what?
   a. Habituation/sensitization require repeated exposure to the eliciting stimulus and no association is needed!
   b. classical conditioning requires an association between a neutral stimulus and an unconditional stimulus (US = is a stimulus that always produces the same behavioral response, ex shock = fear/ freezing behavior).
   c. instrumental conditioning requires an incentive (reinforcer positive [food, sex, money] or negative [no food, no sex, owe money for bad behavior, grounded, curfew etc]) Note, punishment is not the same thing as negative reinforcement, rather punishment is a horrible learning tool it produces associated other acquired unwanted behaviors like fear, hate, neurosis, anger and aggression etc. Negative reinforcement is not punishment, rather negative reinforcement is removal of a positive incentive. For example get paid for good grades (positive incentive) or owe money for bad
grades, late credit card payment or speeding ticket. Punishment is physical, verbal emotional harm inflicted on a subject!

5. Why do I call instrumental condition trial and error learning?
6. What brain area do I think is important for mental trial and error learning? What is unique about this brain area that allows it process and perform specific mental functions no other brain area can?
7. What is a sensory register memory, short-term memory and long term memory? What are their capacities and durations?
8. Why is it useful (almost a complete requirement really) that sensory registers have High capacity but low duration, what about short- and long-term memory?
9. 2 main categories of memory are Explicit and implicit, what are the subcategories? What are simple examples of each?
10. What are the 2 types of amnesia? What are events that can produce Amnesia?
11. Who is patient H.M.? And why do we care about him?
12. What 5 things did we learn from H.M? What did H.M not have deficits in after his surgery?
13. What Brain areas were removed from H.M.? Why is this important to know?
14. What type of information could H.M. Still acquire?
15. What type of memory did H.M. Have loss of?
16. What other brain areas are important for memory and what type of memory do they facilitate? SO do we have multiple memory systems?
17. I talked about and animal model that showed the importance of the rhinal cortex, what is the rhinal cortex important for? And WHAT is the Hippocampus important for? Think in terms of types of Recognition!
18. We know that Amnesia has a gradient of loss How so?
19. We know that both consolidation and retrieval of memories is subject to disruption how so?
20. What Brain areas facilitate memory?
21. What is systems consolidation? How is this related to how we experience, acquire and store information into memories? How does this relate to Hebb’s theory of fire together wire together and how does this relate to neural patterns and neural indexes?
22. What is the indexing theory? How does this theory work? What is meant that one neural system expresses a neural representation of the experience and another neural pattern indexes this neural pattern.
23. Why does indexing happen in the Hippocampus and Amygdala and cerebellum, but not the rest of the cortex (primary cortexes and association cortexes)?
24. What is unique about the hippocampus that makes it ideal for indexing?
25. Why does it make sense that no neuron stores experiences or content? And why does it possibly make sense that neurons index the neural systems that are responsible for the experience?
26. What and How does indexing provide for pattern separation? This is another reason indexing of the hippocampus is important?
27. What is the one example that displays the ability of the hippocampus to index episodic and associative memories?
28. So you know the Hippocampus, amygdala and cerebellum (and to a smaller capacity/extent the basal ganglia) are all brain areas that have immense Plasticity capabilities! And these are all brain areas important for the storage of specific types of memories? What are some general types of memories associated with these brain areas?
29. Multiple brain regions have been closely associated with a number of different implicit memory systems. Which brain region do you think would be especially important for learning how to play the piano, which can take years to master?
30. If you were exposed to multiple repeated loud noises that later reduced your startle responses to that noises this would be an example of what type of learning?
31. Watson and collaborators made Albert (a child) cry when a loud noise was repeatedly presented together with little white furry creatures (rats and bunnies), this is an example of what type of learning? In this example, what is there an unconditioned stimulus used to scare little Albert? Or is there an incentive?
32. Training of a puppy to "roll over" by offering a treat to the dog only after the correct behavior (roll over) is performed is an example of what type of learning?
Lecture 15 LTP

1. What is the unique structure that neurons communicate by?
2. All Synapses can be modified through multiple ways what are they
   However, not all synapses can undergo a specific type of modification, what is this modification? It is called synaptic plasticity or synaptic genesis and requires LTP!
3. What is cellular consolidation?
4. What is LTP and how discovered this phenomena?
5. What are the 2 stages of LTP?
6. How do researchers test for LTP? What is the setup and needed tools?
7. What is the tri-synaptic neural circuit in the hippocampus?
8. Where in this tri-synaptic neural circuit can we place an stimulating electrode and a recording electrode to demonstrate LTP?
9. What is a Weak stimulus (WS or testing stimulus)? What is a Strong stimulus (SS or inducing stimulus)?
10. What is a fEPSP? What is it recording and where?
11. What are the 2 ligand receptors are need to induce-express-maintain LTP? What type of receptor are they in terms of functional properties and NTs that they bind?
12. What is unique or what role does the AMPA receptor play in LTP induction-expression-maintains?
13. What is unique or what role does the NMDA receptor play in LTP induction-expression-maintains?
14. What is the sequence of receptor activation for the induction-expression-maintains of LTP?
15. How do we know that NMDA is need for induction but not expression and maintenance of LTP and the AMPA is required for all of these steps?
16. NMDA is considered the molecular protein responsible for associative learning why?
17. What role does AMPA play in S-LTP? What ion and receptor leads to this action? S-LTP is in short the immediate addition of more AMPA receptor into the post-synaptic membrane. This happens because NMDA-R let in Ca++ which cause AMPA to be trafficked into the membrane. This is why after a strong stimulus and subsequent week stimulus will result in a larger depolarization of the neuron!
18. What role does NMDA-R play in S-LTP?
19. How S-LTP related to short-term memory?
20. What is L-LTP and how is it related to long term memory?
21. In L-LTP what molecule (ion) let into the cell leads to signal transduction processes that lead to an increase in protein production (increase gene transcription and translation)? What other NT leads to similar effects (it is BDNF)?
22. What proteins are increased in L-LTP? And what purpose to these proteins serve?
23. How do we know that maintenance of LTP (S-LTP to L-LTP) requires new proteins to be made?
24. Give a summery and steps of How High frequency Simulation leads to LTP?
25. S-LTP leads to post-translational modifications, which supports induction and expression of LTP. And L-LTP leads to new protein synthesis, which supports maintenance and expression. Regardless both Enhance AMPA receptor activity and functioning by increasing the number of AMPA receptors in the membrane. L-LTP, additionally increases the actual size or remodeling of the dendritic spine which supports consolidation.