- Reinforcement learning
  - Meso-cortical system
    - VTA -> primary/secondary association cortex, hippocampus, frontal cortex
  - Frontal cortex: working memory, reinforcement of current plans and strategies
  - What controls the VTA?
    - LH: hunger
    - Amygdala: emotional stimuli
    - PFC: planning, strategies
    - Lateral hypothalamus, amygdala, prefrontal cortex -> VTA

- Motor learning
  - Motor learning: learning within the motor system (little perceptual inputs required)
    ex. Ride a bike, play tennis, tie a shoe lace
  - Stimulus -> changes in neural circuit that detects a particular stimulus (perceptual system) -> changes in neural circuit that controls a particular behavior (motor learning) -> response

- Relational learning
  - Learning relationships between stimuli or previous memories; memory without meaning: ~ 30 mins (Ebbinghaus)
  - Use of contextual information (schema) to store or retrieve new memories
  - Schema: interconnected concepts/memories (continuously updated)
  - Memories can be manipulated by manipulating (consciously or not) their relationship to other memories; false memories: eyewitness testimony, implanted memories
- Memory: neural mechanisms
  - Neural substrate of memory: what is memory?
    - Neuronal activity alone cannot account for memories that last more than a few seconds
    - Synapses are the primary encoding mechanism
    - Memory items: a pattern of synapses (between excitatory cells)
    - Different kinds of memory means different kinds of learning -> different brain structures
  - The hippocampus: short-term memory
    - Basic anatomy: the tri-synaptic circuit
    - Long-term potentiation
    - Long-term depression
  - Rat hippocampus: tri-synaptic circuit
    - Hippocampus ->1 entorhinal cortex ->2 dentate gyrus ->3 CA3 -> CA1
  - Long-term potentiation
    - LTP induction = learning
    - Electrical test stimulus = recall (will look the same in long-term memory)
    - Memory = size of the EPSP
    - LTP induction = rapid simulations so that both presynaptic and postsynaptic sites are active
  - LTP: synapse specific
    - Before LTP: stimulate axon that forms synapse with neuron
    - After LTP: synapse is strengthened
      - Other synapses on that neuron are not affected
    - The Hebb learning rule: if presynaptic and postsynaptic sites are repeatedly active at the same time, the synapse is strengthened
- **Hippocampus**
  - Memory is associative…so is long-term potentiation
  - If 2 synapses, weak and strong, are stimulated at the same time (associated), the weak synapse becomes stronger
  - But in reality no stimulating electrodes

- **Associative LTP**
  - Many (almost simultaneous) inputs can be associated: dendritic spike
    (associating green hat with grandma: the neuron fires when thinking about grandma or hat)
  - Memories are represented by *patterns* of synapses

- **Memory: LTP and NMDA receptors**
  - If classical conditioning is implemented by neurons, there should be a neuronal mechanism that mimics it
  - **Fact 1:** synaptic modification occurs only if there is Ca2+ entry in a synapse (2nd messenger, including CaM-KII)
  - **Fact 2:** LTP occurs only when Ca2+ enters through NMDA receptors
  - NMDA receptors are permeable to Na+ and Ca+ but are normally blocked by Mg2+
  - NMDA receptors are unblocked only if there is postsynaptic depolarization:
    - Action potentials (dendritic spikes)
    - Depolarization through an electrode -> ie. NMDA unblocked only if there is postsynaptic activity
  - Unblocked NMDA receptors let Ca2+ through only if there is glutamate bound -> ie. Ca2+ enters only if there is presynaptic pathway
  - So Ca2+ enters through NMDA receptors only if there is pre and postsynaptic activity -> Hebb rule again
- LTD
  - Long-term depression: decrease in synaptic strength
  - Induction (i.e., learning)
    - High frequency stimulation (>10Hz) -> LTP
    - Low frequency stimulation (<10Hz) -> LTD
  - Result in a decrease in AMPA receptors
  - Also associative and selective
  - Goal of LTD: prevent overly strong synapses