

# Lecture 15

Wednesday, July 14, 2021

23:39

## Molecular Mechanisms of Nociception

- Nociception - the sensory nervous system's process of encoding noxious stimuli; intense chemical, mechanical, or thermal stimulation of sensory nerve cells called *nociceptors* produces a signal that travels along a chain of nerve fibers via the spinal cord to the brain
- Lots of stuff can induce nociception (such as ATP)
- When enough stimulus causes the nerve to depolarize, the different info is sent to the CNS; this transmission isn't immune to error
- Proinflammatory mediators can directly activate the nociceptors, generate action potentials, or facilitate the firing of the nociceptors, resulting in peripheral sensitization and hyperalgesia
- Calcitonin gene-related peptide (CGRP) contributes to central sensitization in animal models of inflammation or nerve injury
- Pain perception disrupts muscle fiber recruitment
- Substances that are released during central sensitization can modify motor neuron excitability
  - o This can alter muscle recruitment patterns
- NGF (nerve growth factor) facilitates axon growth and regeneration, protects against death and damage of the cell, promotes angiogenesis, regulates Schwann cell proliferation and differentiation, etc.
- SAD DAVE:
  - o Sensory = Afferent = Dorsal
  - o Dorsal = Afferent Ventral = Efferent
- Consistent firing turns on genes that create more receptors
  - o Thus, consistent firing begets *easier* firing
- Prostaglandins come from arachidonic acid which lives in sarcolemma in phospholipid bilayer
- Have pain fibers and motor fibers [nerves]
- Myasthenia gravis - autoimmune disease where antibodies are destroying nicotinic acetylcholine receptors
  - o Muscle weakness --> can lead to paralysis

- We can deactivate and desensitize nerves (with Botox, Atropine) as well as activate and hyper-sensitize them