Week 9
Introduction to Neuroscience: NSC-1306

The concepts this resource covers are the topics typically covered during this week of the semester. If you do not see the topics your particular section of class is learning this week, please take a look at the weekly resources listed on our website for additional topics throughout the semester.

We also invite you to look at the group tutoring chart on our website to see if this course has a group tutoring session offered this semester.

If you have any questions about these study guides, group tutoring sessions, private thirty minute tutoring appointments, the Baylor Tutoring YouTube channel or any tutoring services we offer, please visit our website www.baylor.edu/tutoring or call our front desk during open business hours (M-Th 9 AM-8PM on class days) at 254-710-4135.

Keywords: Brain Damage, Neurological Diseases, Neuroplasticity

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Topic of the Week: ***Brain Damage & Neuroplasticity***

RETRIEVAL PRACTICE: ***(Reference Week 8 Resource if Needed/Answers at the End!)***

1. What is the major difference between Broca's and Wernicke's aphasia?
2. What makes the Wernicke-Geschwind model different from theories pertaining to the lateralization of language?

While there are many structures in place designed to protect the brain, it can still become damaged as a result of tumors, injuries, or other neurological diseases.

- **Brain Tumor:** mass of cells that grows independently of the rest of the body
  - **Encapsulated Tumors:** grow within their own membrane
    - **Acoustic Neuromas:** tumors that grow on nerves or tracts
    - **Meningiomas:** tumors that grow between the **meninges**
  - **Infiltrating Tumors:** grow diffusely through surrounding tissue
  - **Benign Tumors:** surgically removable with little risk for further growth
  - **Malignant Tumors:** difficult to remove or destroy, and continue to grow after attempts to remove or destroy them
  - **Metastatic Tumors:** originate in one organ and spread to another

- **Gliomas:** develop on glial cells, are infiltrating, rapidly growing, and are the most common form of malignant brain tumors
- **Cerebrovascular disorders:** major cause of death and neurological dysfunction
  - **Stroke:** sudden onset **cerebrovascular disorders** that cause brain damage; symptoms - amnesia, **aphasia** (language difficulties), paralysis, and coma
    - **Infarct:** area of dead/dying tissue produced by a **stroke**
    - **Penumbra:** vulnerable area surrounding the **infarct** targeted for treatment to potentially recover this damaged tissue
  - **Cerebral Hemorrhage:** bleeding in the brain
    - **Aneurysm:** pathological balloon-like dilation that forms in the wall of an artery at a point where the elasticity of the wall is defective
  - **Cerebral Ischemia:** disruption of the blood supply to an area of the brain
    - **NOTE:** Commonly caused by a **thrombus** (blockage of blood flow by a plug at formation site), an **embolus** (blockage of blood flow in a smaller blood vessel by a plug formed in a larger blood vessel), and **arteriosclerosis** (blood vessels are narrowed/blocked by an accumulation of fat deposits).
- **Close-Headed Injuries:** produced by blows that do not penetrate the skull
  - **Contusions:** involve damage to the cerebral vascular system that produces internal **hemorrhaging** resulting in a **hematoma**
    - **Hematoma:** localized collection of clotted blood in an organ or tissue (a "bruise"); in this case, a bruise on the surface of the brain
    - **Contrecoup Injuries:** contusions that occur on the opposite side of the brain as the blow
  - **Mild TBI (mTBI):** a disturbance of consciousness following a blow to the head and there is no evidence of **contusion** or other structural damage
  - **Chronic Traumatic Encephalopathy (CTE):** **dementia** and cerebral scarring often observed typically in athletes in high-impact sports that have experienced repeated **concussive**, or **subconcussive** blows to the head

Brain damage can also be caused by **infections** in the brain, **neurotoxins**, and even some **genetic factors**.

Highlight #1: Neurological Diseases

There are three major **neurological diseases** that damage the brain in different ways that are important to discuss along with their specific **animal models**.

- **Parkinson's Disease:** a movement disorder that is associated with the degeneration of **dopaminergic neurons** in the **substantia nigra**

Symptoms: pronounced tremors during inactivity, muscular rigidity, difficulty initiating movement, slowness of movement, and a mask like face
Damage: Damage mainly occurs in the **substantia nigra** particularly along the **nigrostriatal pathway** to the **basal ganglia** which is involved with motor control. As the disease progresses, there is a substantial loss of **dopamine** in this region and the surviving clumps of these neurons are called **Lewy Bodies**.
Treatments: **L-Dopa**, a precursor to **dopamine**, can slow down the development of the disease, but not stop it. In addition, **deep brain stimulation**, primarily to the **subthalamic nucleus** helps in some cases, but has some concerning side effects.
MPTP Model of Parkinson's Disease: nonhuman primates respond to **MPTP**, a synthetic opiate, in the same manner of humans, displaying the motor symptoms of this disease, cell loss in the **substantia nigra**, and a major reduction in **dopamine**, and can be helpful in studying the potential causes of **PD**.

Huntington's Disease: a progressive, terminal disorder of motor and intellectual function that is produced in adulthood by a **dominant gene**

Huntingtin: dominant gene that is mutated in cases of **HD**

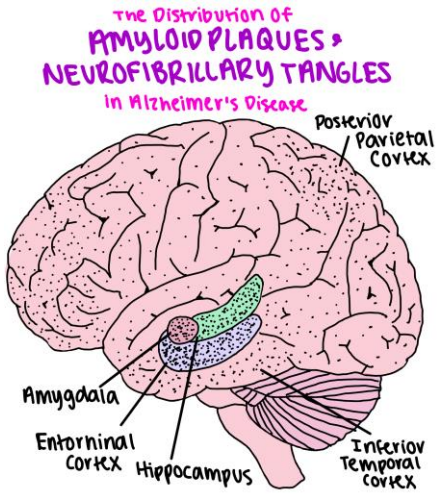
Huntingtin Protein: protein whose synthesis is controlled by the **huntingtin gene** and is abnormal in individuals with this disease as it causes these proteins to more easily clump together

Symptoms: complex, unwilled movements of entire limbs with psychiatric and cognitive deficits that greatly diminish their quality of life
Damage: characterized by severe cell death in the **caudate nucleus** of the **basal ganglia** by a mutation in the **huntingtin gene** from an excess of **triplet repeats** with these mutations being passed to one-half of their offspring, leading this offspring to develop **HD**

Alzheimer's Disease: a progressive neurodegenerative disorder that is the most common form of **dementia** in the elderly with three defining causes including **neurofibrillary tangles**, **amyloid plaques**, and **neuron loss**

Preclinical Stage: pathological changes in the brain without any behavioral or cognitive symptoms
Prodromal Stage (Mild Cognitive Impairment): not as severe, but is an indicator that symptoms of **AD** are progressing
Dementia Stage: initially a progressive decline in memory, deficits in attention, and personally changes that come more severe as time goes on

Symptoms: signs for **AD** appear as a decline in cognitive function (attention deficits, confusion, memory decline) and personality changes (irritability, paranoia) which eventually becomes **dementia** and an inability to perform simple, everyday behaviors (swallowing)



Damage: a general loss of **cholinergic neurons** specifically in the **basal forebrain** along with a buildup of **amyloid plaques** and **neurofibrillary tangles** within neurons in important memory structures like the **amygdala**, **hippocampus**, and **entorhinal cortex**

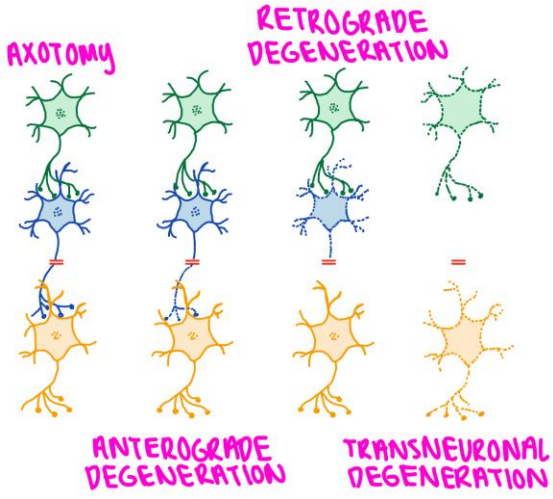
Transgenic Mouse Models of Alzheimer's Disease: **transgenic** mice, or mice that have genes from another species in their genome, accumulate human **amyloid protein** and have **amyloid plaques** distributed throughout the brain, specifically in memory structures, with behavioral and cognitive deficits that mimic that of humans with **AD**, and can be helpful in studying the potential causes of **AD**.

NOTE: These mice do not have **neurofibrillary tangles** and overexpress the important **tau protein** that comprise them.

Highlight #2: Responses to Nervous System Damage

An important function of the **nervous system** is its ability to respond to the previously discussed **damage** that can arise in a variety ways. Therefore, it is important to discuss the mechanisms by which the **nervous system** responds to these forms of damage.

Neural Degeneration: a component of both brain development and disease that is greatly influenced by a variety of factors including nearby **glial cells**, the activity of **degenerating neurons**, and the particular cause of **degeneration**; to induce this, **axons** are precisely cut



Anterograde Degeneration: the degeneration of the **distal segment** (to the **synaptic terminals**) which occurs

rapidly as it is cut off from the metabolic center of the **neuron**, or the **cell body**

Retrograde Degeneration: the degeneration of the **proximal segment** (to the **cell body**) which progresses gradually and can either induce:

Degenerative Changes: neuron will ultimately die of **apoptosis**

Regenerative Changes: neuron undergoes a massive synthesis of proteins that will be used to replace the degenerated axon

Transneuronal Degeneration: degeneration of a neuron caused by damage to another neuron it is linked to by the **synapse**

Anterograde Transneuronal Degeneration: spreads from damaged neurons to neurons on which they synapse

Retrograde Transneuronal Degeneration: spreads from damaged neurons to the neurons that synapse on them

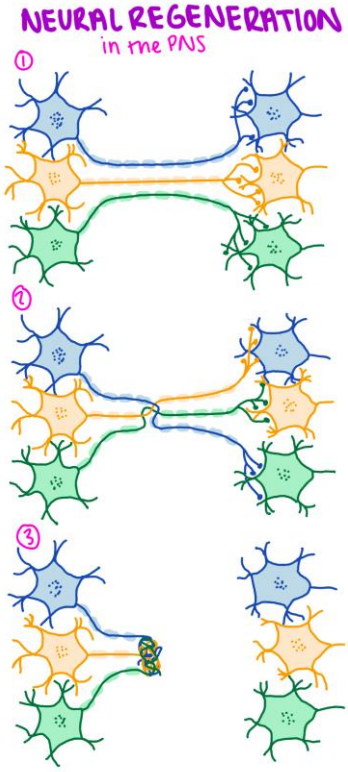
Neural Regeneration: the regrowth of damaged neurons

NOTE: The capacity for accurate axonal growth in adult mammals is greatly diminished in the **PNS** and virtually nonexistent in the **CNS**.

Schwann Cells: clear debris and scar tissue resulting from neural degeneration and promote regeneration in the **PNS** by producing **neurotrophic factors** and **cell-adhesion molecules**

Oligodendroglia: do not clear debris or stimulate/guide regeneration in the **CNS** and actively release factors that block regeneration

NOTE: Astrocytes in the **CNS** form a **glial scar** after injury and is a barrier that prevents growth.



Regeneration in the PNS: occurs in three primary ways including:

1) If the original **Schwann cell** sheaths remain intact, the regenerating axons grow through them to their original targets
2) If the peripheral nerve is severed and the cut ends become separated by a small distance, axons grow into incorrect sheaths and targets
3) If the cut ends of a nerve become widely separated or if a large portion is damaged, there can be no meaningful regeneration

Collateral Sprouting: the growth of axon branches from mature neurons, usually to **postsynaptic** sites

abandoned by adjacent degenerated axons

Neural Reorganization: occurs in the **cortex** as the brain has the ability to reorganize itself in response to **experience** or commonly after peripheral damage and can occur via:

1) Strengthening existing connections between neurons
2) Growth of new connections by **collateral sprouting**

NOTE: Surviving axons in the **CNS** can make synaptic contact with other brain areas vacated by damaged axons, commonly in the **sensory** and **motor cortex**.

Recovery of Function: can possibly occur due to **cognitive reserve** or **adult neurogenesis**

Cognitive Reserve: allows cognitive tasks to be accomplished in new ways, explaining how function can be recovered

Neurogenesis: **stem cells** migrate to the site of damage, but there is no direct evidence that it recovers the functions of that region

Beyond this, the brain has the ability to change with everything one learns, experiences, and remembers, critically occurring in youth. This ability is called **neuroplasticity**, and researchers have been interested in how this ability could potentially assist recovering the functions of the brain after damage occurs.

Neurotransplantation: fetal **pluripotent stem cells** are placed in regions of the brain to stimulate the growth of new cells to recover the functions of the damaged brain regions they specifically target

NOTE: There have been some successful studies in this, but further research is ongoing along with how **rehabilitative training** can further assist in restoring the functions of damaged areas in the **nervous system**!

CHECK YOUR UNDERSTANDING

Concept Check: *(answers found on the last page)*

1. What is the difference between encapsulating and infiltrating tumors?
2. What is important about the infarct and penumbra in the context of a stroke?
3. What is the MPTP model of Parkinson's Disease and why is it significant?
4. What is the difference between anterograde and retrograde degeneration?
5. How can regeneration in the PNS occur?

THINGS YOU MAY STRUGGLE WITH

1. This chapter has a lot of vocabulary terms that are important to know! It can be challenging to differentiate between them, so it can be helpful to make flashcards.
    a. Once you have these terms solidified, it can be helpful to test yourself with a friend on these terms so you both practice active recall!
2. The mechanisms by which the nervous system responds to brain damage can be tricky to understand, so try to draw out the different forms of degeneration along with how the brain recovers from this.
    a. Neurotransplantation is a tricky concept that goes along with these mechanisms. Try to spend some time drawing out how this research is performed to restore function in these damaged areas!
3. The various neurological diseases in this chapter are important to understand as well as to know the key symptoms, damages, and animal models for each of them. It can be helpful to make charts to organize your thinking and to compare between these diseases to understand them fully!

CONGRATS: You made it to the end of the resource! Thanks for checking out these weekly resources! Don't forget to check out our website for group tutoring times, video tutorials, and lots of other resources at www.baylor.edu/tutoring!

Answers to Retrieval Practice and Check Your Understanding questions are below!

Retrieval Practice:

1. **Broca's aphasia patients have difficulties with speech production, but can comprehend written/spoken language while Wernicke's aphasia patients have no difficulties with speech production, but cannot comprehend what they or others are saying (they make no sense when they speak - word salad)**
2. **WG model has to do with the cerebral LOCALIZATION of language, meaning it delves into the locations of the brain that work together in circuits to produce or comprehend language**

Concept Check:

1. **Encapsulating (grow within their own membrane); Infiltrating (grow diffusely in surrounding tissue)**
2. **Infarct (dead/dying tissue around area of the stroke); Penumbra (vulnerable area around the infarct that is targeted for treatment after a stroke to restore potentially function)**
3. **MPTP mirrors symptoms of PD → animals display motor symptoms, cell loss in substantia nigra, and a reduction of dopamine - treating this can help to bring about findings on how PD develops and potential treatments for it**
4. **Anterograde (damage from the axon to the synaptic terminals, rapid); Retrograde (damage from the axon to the cell body)**
5. **Schwann cells intact (grow through sheath to original targets); Slight disconnection (grow to incorrect targets); Large distance/damage (grows in a tangled clump)**