LESSON PLAN

Title: Zombie Autopsies Setting: In Classroom Subject: Biology - Neuroscience Grade Level: 9th-12th Grade Time Frame: 1 Hour Paired Dana Foundation Fact Sheets: 9th-12th Grade How Does the Brain Work? Next Generation Science Standards: Meets HS-LS1-2, HS-LS1-3

STUDENT OBJECTIVES

- Learn how abnormalities in the brain affect behavior.
- Explore hypothetical zombie neuroanatomy and how dysfunctional brain regions impact hypothetical zombie behavior.
- Synthesize knowledge of neurons, neurotransmitters, and neuroanatomy to create novel cures for the zombie brain.

BACKGROUND

This interactive lesson plan allows students to create cures to stop a Zombie Apocalypse. Teachers will learn about and share knowledge regarding neuroanatomy, neurotransmission, and neurotransmitters. These topics will be integrated to develop novel medications that target specific brain region abnormalities.

MATERIALS

- Printed copies of 9th-12th grade Dana Foundation fact sheet, "How Does the Brain Work?" **Downloadable at** www.dana.org/factsheets.
- Skittles, 3lb bag, found on Amazon.com at https://amzn.to/2rZJAfD.
- Clear plastic 5oz cups, found on Amazon.com at https://amzn.to/2EZo2ra.
- Brain diagram and neurotransmitter sheet printed out for each group, included in lesson plan.
- Zombie Autopsies 101 by PageTurnerFilms: https://www.youtube.com/watch?v=y42jrDVge9Q





WHAT TO KNOW BEFORE YOU TEACH

* Note: This content is primarily for the instructor's reference; the accompanying PowerPoint presentation will be for the students.

Zombie Neuroanatomy

Frontal Lobe:

- Functions: planning, reasoning, speech, movement, and problem-solving.
- The frontal lobe is critical for executive functions such as reasoning, planning, and problem-solving. Zombies can see and sense us, but they have a hard time using executive functions when interacting with humans. It is clear zombies lack a functional frontal lobe due to their impulsive nature and lack of consideration for the consequences of their actions. In addition, zombies cannot plan complex actions and therefore get stuck when it comes to opening a door or a window.

Amygdala:

- Functions: primitive emotion center fueling emotions such as fear and rage.
- Because zombies have a dysfunctional frontal lobe, the amygdala, the primitive emotion center of the brain responsible for our "fight or flight" response, has free reign. Usually, the frontal lobe sends signals to the amygdala to regulate its activity. Without those regulatory signals, the amygdala is hyperactive. A hyperactive amygdala impairs the zombie's ability to think rationally and make wise decisions, which can result in intense feelings of fear, rage, and aggression. This drives a zombie's need to attack!
- Dr. Schlozman, the author of *Zombie Autopsies* and host of the YouTube video provided above, notes that crocodiles are also mostly driven by their amygdala. Researchers have found if you take out the amygdala of the crocodile, the animal is significantly less likely to attack and retreat.



TEACHER BACKGROUND INFO

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Zombie Neuroanatomy

Anterior Cingulate Cortex:

- Functions: slows down communication from the amygdala to the frontal lobe.
- Communication from the frontal lobe to the amygdala dampens amygdala responses, causing you to be more rational. In contrast, communication from the amygdala to the frontal lobe dampens frontal lobe activity, causing you to behave impulsively. The cingulate gyrus governs the communication from the amygdala to the frontal lobe. The anterior portion of this gyrus slows the communication signals from the amygdala to the frontal lobe, and the posterior portion speeds these signals up. When your amygdala responds to a threat in your environment causing a fear response, the anterior cingulate cortex activates to slow communication to the frontal lobe, allowing you to think the situation through before acting on impulse.
- It is clear zombies have a dysfunctional anterior cingulate cortex because they act strictly on impulses and emotions such as rage. Dysfunction of the anterior cingulate cortex allows the posterior cingulate cortex to send strong communication signals from the amygdala to the frontal lobe. The combination of a hyperactive amygdala and an inhibited frontal lobe causes zombies to have traits such as hyper-aggression and to attack without thinking.

Cerebellum:

- Functions: balance and coordination.
- We know that zombies are capable of hunting humans. However, their movements are off-balance and unsteady. They have a stiff, wide-legged walk and constantly look like they are going to teeter over. This must be, in part, due to a dysfunctional cerebellum.





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Zombie Neuroanatomy

Basal Ganglia:

- Function: coordinating movements, fluidity of movement.
- The basal ganglia is implicated in motor control and fluidity of movement. Our basal ganglia allows us to do simple tasks, like picking up a glass, and more complicated tasks, like driving a car. Ultimately, the basal ganglia allows us to put movements together to achieve a goal. As we discussed above, zombies lack the ability to move properly. They are clumsy and unable to execute simple motor tasks. Therefore, they must have an abnormality of the basal ganglia.

Ventromedial Hypothalamus:

- Function: regulates eating behavior by sending hunger and satiety signals.
- The ventromedial hypothalamus is a specific nucleus of the hypothalamus. It is responsible for sending out hunger and satiety signals to regulate how much we eat. Zombies are constantly hungry, always looking for a human to prey on. Therefore, the ventromedial hypothalamus must not be functioning properly, making the zombie insatiable.





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PROCEDURE

[1] Each student reads 9th-12th grade Dana Foundation fact sheet, "How Does the Brain Work?"

[2] Introduce the exercise using the YouTube video, found in Materials.

- Notes on YouTube video
 - This lesson plan was inspired by the book *Zombie Autopsies*. The author, Dr. Steven Schlozman, is the host in the YouTube clip. He gives a brief introduction to zombies and the brain regions that are dysfunctional in them. You will discuss these brain regions in more detail with the accompanying PowerPoint.

[3] Give the necessary background information from the accompanying PowerPoint presentation.

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[4] Introduction to activity

• You have been chosen to help end the Zombie Apocalypse! It's your job to create a drug that will cure the dysfunctional parts of the zombie brain. You must use your expert knowledge of neurotransmission and neuroanatomy to create a cure using agonists and antagonists for the neurochemicals dopamine, serotonin, GABA, glutamate, and norepinephrine. When you are finished, you will present your cure to your classmates for feedback. Time is of the essence. Over a million people have already died, and more become infected every day!

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[5] Split students into groups.

- [6] Provide each group with skittles in a plastic cup and a brain diagram plus neurotransmitter sheet (see page 11 & 12). The skittles will serve as neurotransmitter agonists or antagonists. Students will place the skittles in the different brain regions, indicating they would like the drug to send that specific neurotransmitter agonist or antagonist to that particular brain region. The students do not have to use all the Skittles.
 - Dopamine: purple
 - Serotonin: green
 - GABA: red
 - Glutamate: yellow
 - Norepinephrine: orange





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Zombie Neuroanatomy

The Neuron & Neurotransmission:

- Cells within the nervous system are called neurons. Neurons are the basic working units in the brain designed to communicate with each other to carry out all brain functions. We have approximately 86 billion in our brains!
- A neuron is made up of three main parts: the dendrites, cell body, and axon. Dendrites receive information from other neurons while axons send information to other neurons via electrical signals. The cell body houses the nucleus and cytoplasm.
- The contact point between two neurons where communication occurs is called the synapse. When a neuron wants to send information, it conducts an electrical impulse down the axon. Something called the myelin sheath wraps around the axon, insulating the axon and allowing the electrical impulses to travel faster. Once the signal reaches the end of the axon—an area called the nerve terminal—chemical messengers called neurotransmitters are released. Neurotransmitters travel across the synapse and bind to receptors on the receiving neuron's dendrites.
- Specific neurotransmitters bind to specific receptors, similar to a lock and key. Binding of the neurotransmitter to its appropriate receptor acts like an on/off switch for the receiving neuron.



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Zombie Neuroanatomy

Neurotransmitters:

There are many types of neurotransmitters that can have a variety of effects in a variety of different brain regions. We will touch on a few that will be used in this lesson plan.

- Glutamate:
 - Glutamate is the main excitatory neurotransmitter in the brain. Upon binding to receptors, glutamate excites the receiving neuron and encourages neuronal activity. Glutamate is thought to enhance brain activity.
- GABA
 - GABA inhibits or silences neurons when binding to their receptors. An influx of GABA into a particular brain region can quiet the activity of that brain region.
- Dopamine
 - Dopamine is a critical neurotransmitter to signaling pathways in the brain that govern movement. A deficiency in dopamine can cause symptoms such as muscle rigidity, tremors, and difficulty making coordinated, fluid movements.
 For example, a deficiency of dopamine in the basal ganglia is a main cause of Parkinson's, a neurological disorder that affects movement.

- Dopamine also acts in the hypothalamus to help regulate hormone secretion.

- Serotonin
 - Serotonin levels increase in the amygdala in response to fear. Therefore, when you come into contact with a threat in your environment, an influx of serotonin occurs in the amygdala to promote an emotional response.
 - Serotonin signaling also has implications for satiety in the hypothalamus. It is thought that activation of certain serotonin receptors in the hypothalamus promotes satiety.
- Norepinephrine
 - Norepinephrine is known to be involved in behaviors such as arousal and sleep.





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Zombie Neuroanatomy

- Interestingly, norepinephrine is also released while under stress. High levels of norepinephrine in the prefrontal cortex can cause dysregulation of signaling systems and impair functioning of this brain region. Brains of patients with post-traumatic stress disorder show that the norepinephrine released during stress continues to remain at high levels in the prefrontal cortex for an extended amount of time.

Agonists & Antagonists:

- Many drugs act as agonists or antagonists at certain receptor sites.
- An agonist is a chemical that binds to a receptor, mimicking the action of the neurotransmitter that binds to that receptor site.
- In contrast, an antagonist binds to a receptor, blocking the receptor and preventing the appropriate neurotransmitter from binding at that site.
- Agonists are used to promote the intended action of the neurotransmitter at its receptor site, and antagonists are used to block the action.
- For example, a dopamine agonist will bind to dopamine receptors, mimicking the action of dopamine. In contrast, a dopamine antagonist will bind to dopamine receptors blocking endogenous dopamine neurotransmitters from binding.







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Zombie Neuroanatomy

Hypothetical Drug Example

- Drug Name: Zomban
- Drug actions in the brain and the effect on zombie behavior:
 - Glutamate agonist release in the frontal lobe. This will increase brain activity in the frontal lobe, bringing the frontal lobe online and allowing the frontal lobe to have an inhibitory effect on the amygdala. This will dampen the zombies' emotions of rage and fear and allow them to think more rationally and control their behavior.
 - Norepinephrine antagonist to the frontal lobe. This will dampen the stress response in this brain region and allow the frontal lobe to focus on executive functions such as problem-solving and reasoning. Again, this will help the zombies behave more rationally.
 - GABA agonist release in the amygdala. This will inhibit brain activity in the amygdala, dampening emotions of anger and rage.
 - Serotonin antagonist to the amygdala to further dampen feelings of fear that cause zombies to attack.
 - Glutamate agonist release in the anterior cingulate cortex. This will increase brain activity in this brain region helping to inhibit the strong communication signals from the amygdala to the frontal lobe. This inhibition will help the zombies regulate their impulsivity and emotional reactions.
 - Dopamine agonist to the cerebellum and basal ganglia to aid in movement and executing motor tasks. This will help improve the zombie gait.
 - Serotonin agonist to the ventromedial hypothalamus. This will promote satiety and help regulate the zombies' eating behavior.
- This is not an exhaustive list. Other possible drug actions exist. It is at the discretion of the teacher to create guidelines on how many brain regions and neurotransmitters must be targeted with the created drug.



PROCEDURE

[7] Explain the parameters for drug development.

- Neurotransmitter agonists or antagonists will be targeted to specific brain regions to promote or inhibit activity of these regions.
- Be sure students know to address: 1) whether the neurochemical is acting as an agonist or antagonist; and 2) for what neurotransmitter in their presentations.
- Neurotransmitter agonists or antagonists can go to multiple brain regions.

[8] Each group should come up with a creative name for their drug.

- [9] Have each group present their drug. The same brain diagram that was handed out to each group to aid in creating their drug will be in the accompanying PowerPoint. Each group can use this brain diagram as a visual tool to explain where their drug is acting in the brain.
 - Students should address which zombie behaviors their drug targets and the brain regions associated with those behaviors. How will your drug change or prevent these behaviors?
 - Students should explain the actions of their drug in the brain.
 - Which neurotransmitter agonists and antagonists were targeted to which brain regions and why?

ADDITIONAL RESOURCES

Please view the following YouTube video as a brief introduction to the lesson:

Zombie Autopsies 101 by PageTurnerFilms: https://www.youtube.com/watch?v=y42jrDVge9Q

• A collection of neuroscience puzzles and fact sheets for kids in grades K-12 that are available for download (PDF): www.dana.org/educators

This lesson plan was created in collaboration with Dr. Steven Schlozman, author of "The Zombie Autopsies," and has been adapted by Elizabeth Weaver, M.S., and Katie Partrick, M.S., for the Dana Foundation.



BRAIN DIAGRAM

FRONTAL LOBE

planning, reasoning, speech, movement, and problem solving ANTERIOR slows communication from the amygdala to CINGULATE the frontal lobe CORTEX

> BASAL GANGLIA motor control and

fluidity of movement

VENTROMEDIAL HYPOTHALAMUS

regulates eating behavior by sending hunger and satiety signals

AMYGDALA

primitive emotion center, fuels fear and rage signals coordination



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NEUROTRANSMITTER SHEET

Glutamate

Glutamate is the main excitatory neurotransmitter in the brain. Upon binding to receptors, glutamate excites the receiving neuron and encourages neuronal activity. Glutamate is thought to enhance brain activity.

GABA

GABA neurotransmitters inhibit or silence neurons when binding to their receptors. An influx of GABA into a particular brain region can quiet the activity of that brain region.

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Dopamine is a critical neurotransmitter to signaling pathways in the brain that govern movement. A deficiency in dopamine can cause symptoms such as muscle rigidity, tremors and difficulty making coordinated movements. For example, a deficiency of dopamine in the basal ganglia is a main cause of Parkinson's disease, a neurological disorder that affects movement. Dopamine also acts in the hypothalamus to help regulate hormone secretion.

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Norepinephrine

Norepinephrine is known to be involved in behaviors such as arousal and sleep. Interestingly, norepinephrine is also released while under stress. High levels of norepinephrine in the prefrontal cortex can cause dysregulation of signaling systems and impair functioning of this brain region. Brains of patients with PTSD show that the norepinephrine released during stress continues to remain at high levels in the prefrontal cortex for an extended amount of time.

