Cancer Journeys: From the Microscopic to a Call to Action

Chris Sikich

Philadelphia High School for Girls

ABSTRACT

Cancer is not a topic taught with much depth in high school biology. This unit opens up what students know about cancer from personal experience to examine cancer from many fronts. They first do this by examining cancer through a model organism: the fruit fly. With authentic research on genetically enhanced flies with cancer, students will determine if diet choices impact cancer survival. The second part of the unit goes into the cellular mechanism of signaling to see how two different proteins, when mutated, can short circuit a cell into becoming cancerous. The third part of the unit looks at evolution and cancer by examining how organisms besides humans get cancer. While showing our relatedness, there is the question of how extensive the reach of natural selection is on cancer and how animal size potentially plays a role in cancer occurrence. The final part of the curriculum takes the well-informed cancer scientist into the reality of healthcare disparities and how inequitable cancer diagnosis, treatment and survival is rampant. This ends with students using their voices to make a call to action to their communities about the necessity for cancer healthcare equity.

KEYWORDS

biology, cancer, science, inquiry, genetics, mutation, cell, cell signaling, evolution, call to action, CER, project

CONTENT OBJECTIVES

In my 15th year of teaching biology I have found the least amount of time to teach specific concepts or more specialized topics in biology that stray from curricular standards like cancer. We are to touch on it, but with the constraints of so many other topics and standardized tests (district-mandated Benchmarks, 3-4 times a year, and a course-culminating state-mandated Keystone exam that students must get a proficient on to graduate) we only get to gloss over it. This is a disservice to everyone involved, as students are most engaged when encountering topics that are connected to personal experience like cancer and I myself can speak to its personal toll as both of my parents have died because of cancers (lung and prostate).

As a magnet all-girls public school, Philadelphia High School for Girls attracts students who are future leaders and problem solvers and exposure to biological principles underlying cancer may allow them to participate in summer cancer-related internships in Philadelphia. Sixty-six percent of our students are African American and 57% are from low-income households. According to "Cancer and Cancer Health Disparities in Philadelphia," 472 of every 100,000 Philadelphians were newly diagnosed with cancer in 2016, while 190 of every 100,000 Philadelphians died from cancer in 2016. There is an increased incidence of cancer in African Americans and low-income households. For example, African Americans have a greater than 20% higher incidence of colorectal cancer compared to Cahcasians thus understanding the

biology of cancer will have direct relevance to my students (Augustus and Ellis, 2018). The bottom line is that our students have encountered cancer in a direct or indirect way.

My curriculum is going to take students on cancer journeys, from the microscopic cell signaling to the macroscopic level of evolution and social justice. The first curricular concept is using the model organism fruit flies that have a genetic mutation predisposing them to cancer. We will use fruit flies to collect real time data on how nutrients impact tumor growth. The second concept will be cell signaling, a concept almost never mentioned in regular high school biology while receiving a whole unit in AP biology. Cell signaling pathways in normal cells and how cancer cells hijack them to promote uncontrolled growth will connect to genetics and how mutations in specific genes cause aberrant signaling. The third curricular concept is connecting humans to other organisms through cancer occurrence. With an understanding of cancers being found throughout multicellular life, how natural selection connects to aging and Peto's Paradox, evolution will trigger some intriguing questions about the future of cancer treatment. The final curricular concept is how social disparities relate to cancer incidence, treatment and mortality. Cancers are often found in higher incidences among non-white and socioeconomically disadvantaged populations that are related to lifestyle. Such revelations will lead students to make a call to action about cancer treatment equity. This will be a unit about the biology of cancer that reveals the quantitative and qualitative data to humanize cancer and understand the biological concepts underlying cancer initiation and progression.

The following are the individual objectives for each piece of content for the whole cancer biology unit.

1. Students will understand how to use a model organism to test for dietary effects on cancer.
2. Students will understand how mutations in cell signaling proteins can lead to cancer.
3. Students will understand how evolution and natural selection acts on or does not act on cancer and what the implications of this is for the future of cancer treatment.
4. Students will understand what healthcare disparities exist in cancer diagnosis, treatment and mortality, leading them to call for action in the community on these issues.

Fruit Fly Cancer Research

This curriculum must begin with an understanding of cancer. In the simplest of terms, cancer is uncontrolled cell growth. This unregulated growth begins with an ancestral cell that has a mutation to cause cell reproduction when it should not happen. This reproduction occurs usually because of mutations in either proto-oncogenes or tumor suppressor genes. The former switches on growth while the latter turns off such growth. Mutations alter these genes so mutated proto-oncogenes stimulate too much growth while mutated tumor suppressor genes are inactivated, preventing inhibition. For a tumor, or a mass of cells, to form, at least six of the first cell's controlling genes must be mutated to cause the accelerated or inappropriate growth (Weinberg, 1996). There are benign tumors that pose little risk, but when the cells have these multiple mutations and invade surrounding tissue, malignancy occurs. And when a malignant tumor enters the bloodstream and travels around the body, metastasis has occurred (Lodish et al., 2000). The four parts of the curriculum unit will explore pieces of all of these parts of the cancer story.

As cancer is highly personal, this curriculum is going to build up concepts surrounding cancer development and research so the highly personal subject of having cancer can have many contexts. Using a model organism for study will be an important first step in this journey. A model organism is a highly researched non-human species used to aid in understanding biological processes (Your Genome, 2020). The fruit fly (*Drosophila melangaster*) is a common model organism, with 60% of its genome being identical to humans and around 65% of genes responsible for diseases in humans being found in fruit flies (Ugur et al., 2016). Fruit flies are small (3 mm long) and require a minimal diet of cornmeal and yeast that needs to be changed regularly. Their reproductive cycle is quick, so in 8-14 days new flies emerge (Your Genome, 2020). All of these factors and more (like their large polytene chromosomes that are easily seen under a light microscope) make fruit flies a perfect model organism (Puppels, 1999).

With a life span of up to 50 days, fruit flies never live long enough to develop cancer (Linford et al., 2013). But with the genetic similarities comes the ability to model cancer, especially since much of the cell growth signal pathways in mammals are conserved or essentially the same in fruit flies (Millburn et al., 2016). The modeling is typically in fly tissues similar to mammalian epithelial tissue, which is widespread throughout the body, forming the covering of all body surfaces. Around 90% of human cancers originate in the epithelium, making the research in flies vital to understanding cancer (Hanahan and Weinberg, 2000). In fruit flies, the oncogene, a gene that can transform a cell into a tumor cell, Ras is switched on so it can become a tumor cell. Experiments can be done to see if things such as diet inhibit or promote the growth of tumors, such as shown in a Ras-involved exocrine pancreatic cancer where diet may influence Ras mutations to cause the cancer (Morales et al., 2007). Food ingredients can be added to the standard fly food and the amount of eggs laid and adult longevity can be determined, showing whether or not an altered diet can impact the cancer genes experimentally switched on in the flies.

Fruit Fly Cancer Research Objectives:

1. Students will understand what cancer is and what can cause it.
2. Students will understand what a model organism is and the value of model organisms to scientific research.
3. Students will understand fruit fly anatomy, physiology and their value as a model organism.
4. Students will understand how to design and run an experiment using fruit flies.

Cancer Cell Signaling

Outside the cell are molecules that can trigger activity inside the cell. This activity is a type of cell communication caused by ligands. Ligands can come in many shapes and sizes, from hormones to amino acids, fatty acids to nucleotides. If they are hydrophilic they bind to receptors on the cell membrane. If they are hydrophobic they can bind to intracellular receptors (Lodish et al., 2000). These molecules are part of signal transduction pathways, translating an extracellular stimulus into an intracellular response. This response usually ends with a change in cell function, like turning on transcription factors so certain proteins can be made. And there are often multiple steps within the pathway, with the original signal being amplified to cause a large response (Campbell and Reece, 2005).

Since in the Fruit Fly Cancer Research part of the curriculum epithelial tissue was focused on, here there is a continuance with epithelial tissue-connected cancer. When

considering this type of cancer, one receptor that can be mutated, leading to cancer proliferation, is a trans-membrane glycoprotein called epidermal growth factor receptor (EGFR). Outside the cell is a binding domain and inside the cell is a tyrosine kinase domain. The intracellular portion regulates epithelial cell proliferation, which is key to having proper cell cycle and cell division activities. When a ligand binds extracellularly, signal transduction cascades occur internally. When there are mutations that cause the extracellular domain to trigger internal signal transduction cascades in the absence of an external ligand, cancerous cells can proliferate (Bethune et al., 2010). In all known cancers, EGFR is mutated almost 7% of the time, with lung, glioblastoma, breast and colon cancers among those resulting from mutations (My Cancer Genome, 2017). There are EGFR homologs in fruit flies named "faint little ball" and "torpedo," connecting the curriculum units together (Lusk et al., 2017).

Another vital cell signaling protein class is RAS. Protooncogenes that are small GTPases (guanosine triphosphate enzyme), they are involved in signal transduction. Found inside cells, RAS is activated after a cell-surface receptor, like EGFR, has a proper ligand that bound to it. This extracellular ligand-binding causes an intracellular change to the binding protein that releases a phosphate group. When this phosphate group is added to the GDP (guanosine diphosphate) connected to RAS, the GDP becomes GTP, switching RAS from inactive to active ("RAS Pathway"). From this point, RAS binds to and activates effectors that regulate activity downstream from RAS. RAS activation can lead to the progression of the cell cycle, transcription of DNA into mRNA, transporting of molecules and apoptosis, or cell death, among other cellular occurrences. RAS mutations can lead to the excess of RAS-bound GTP, causing overactive signal transduction that can lead to unwanted results like activating the making of more cells or not leading to apoptosis when that should be the result. EGFR is directly connected in some cases, with overexpression of tyrosine kinase leading to the activation of RAS when it should not be activated (Gurung and Bhattacharjee, 2015). Mutations in the RAS family of genes accounts for more than 30% of all human cancers (Cancer.gov). Among cancers connected to RAS are pancreatic, colorectal, lung and colon, among others (My Cancer Genome, 2017). And, importantly for the curriculum, RAS is found in fruit flies (Mirzoyan et al., 2019).

Cancer Cell Signaling Objectives:

1. Students will understand what the purpose of cell signaling and signal transduction is.
2. Students will understand the importance of proteins EGFR and RAS to cellular communication.
3. Students will understand how cancer can be linked to mutations in EGFR and RAS.

Cancer and Evolution

Since we are studying a model organism (fruit fly) and how diet potentially impacts cancer expression in a cancer pathway found in humans as well (a version of EGFR found in flies as well as RAS), the links between such diverse, distantly related organisms as insects and mammals clearly shows the sharing of a common ancestor and evolution overall. The next step in this journey is to look at the prevalence of cancer outside of humans. Cancer begins with the advent of multicellularity, as an organism would need to have the ability to make new cells to make cells in excess. So only eukaryotes, the only organisms that are multicellular, can have

cancer. Within the realm of eukaryotes, cancers are found in almost all mammals as well as most non-mammal vertebrates, invertebrates and plants as well (Albuquerque et al., 2018).

The information on the similarity of cancer occurrence shows how common cancer is and how we humans are much more like other organisms than we might think. Though lung, prostate and testicular cancers are uniquely common in humans, other animals have other common cancers not as common to us. For example, in captive non-human primates uterine and gastrointestinal cancers are common, while not in humans. On the other hand, lymphomas are found throughout vertebrates. All mammal orders except that of the monotremes have been found to have cancer occurrence (Albuquerque et al., 2018). When entering the invertebrates, there is evidence in hydra, which are small, fresh-water organisms, that tumors have formed in them (Domazet-Lošo et al., 2014). Even extinct organisms have been found to have cancer, including dinosaurs (Rothschild et al., 2003). Plants can develop tumors as well, though rarely spontaneously. Usually, plant tumors are caused by bacteria, fungi or viruses (Doonan and Sablowski, 2010).

Though certainly not all cancers are connected to aging, a vast amount of them can be connected to getting older. Natural selection is a key theory of evolution stating that organisms best adapted to their environment will survive and produce more than enough offspring for some of them to survive so they ultimately pass their genes on at least to their grandchildren (Campbell and Reece, 2005). When organisms like humans pass the age of reproduction, evolutionary theory suggests that natural selection is not acting on them as much. So, as we age, diseases connected to aging become common because natural selection is not selecting for or against the ability to fight off these diseases and live any longer (Kirkwood and Austad, 2000). Though the lifetime risk of dying from cancer in the United States was 21% from ages 0-60 in 2007-2009, the highest incidence of cancer in 2009 occurred in the age range of 65-69 with over 200,000 reported cases (White et al., 2015). So, it is not possible to state unequivocally that natural selection rarely impacts cancer occurrence, it is another evolutionary connection of pedagogical and scientific value.

Then there is the issue of size. There is a great diversity of median organism size within the animal kingdom. You have mice and then you have whales. The larger an organism, the more cells you have in an organism since cell size stays relatively constant. So, with more cells you should have more of a chance for cancer. This is not the case, which is Peto's Paradox (Peto et al., 1975). Humans have a cancer risk of 11-25%, which is not vastly different from mice. In elephants, on the other hand, it is only 5%, showing cancer is not found at consistent rates among organisms (Tollis et al., 2017). Mathematically humans have a certain chance of getting colon cancer based on the number of stem cells in the colon. By the age of 90, humans mathematically should have a 2.5% chance of contracting colon cancer. Actual data from the American Cancer Society shows it is 5%. Based on the fact that a blue whale is 1,000 times the size of a human, the same equation reveals that 100% of whales have colon cancer by the age of 80. Based on what is known about whales, which can live over 100 years, this is not the case. This is Peto's Paradox in a nutshell. So what is preventing elephants and whales from contracting vast amounts of cancer? There are many suggestions including lower mutation rates in non-sex cells, more copies of tumor suppressor genes and fewer proto-oncogenes, among others (Caulin and Maley et al., 2011). This paradox shows that we do not yet understand all the evolutionary pressures at play, so scientific research still has a long way to go. If we can understand what is helping larger organisms to survive we might be able to translate that knowledge into a way to stop the spreading of cancer.

Cancer and Evolution Objectives:

1. Students will understand how many different types of organisms show evolutionary relationships based on how they can get cancer.
2. Students will understand how natural selection acts on aging and how this impacts the occurrence of cancer.
3. Students will understand what Peto's Paradox is and how it relates to potential cancer occurrence.

Cancer Treatment Disparities

This curriculum looks at cancer at the microscopic level with the fruit fly cancer research and cell signaling and then zooms out to see evolutionary connections between different organisms with regards to cancer. All three of these curriculum parts do touch on two important themes: what causes cancer and how can we treat cancer. So for the final portion of the curriculum the realities of cancer treatment and the disparities found within treatment will be exposed.

How can people's health be determined? There are multiple components to the answer to this question, as found in Social Determinants of Health (SDOH). The five domains of SDOH are economic stability, education access and quality, health care access and quality, neighborhood and built environment and social and community context. The SDOH are key to how healthy people are, with some examples including safety in housing, racism, employment opportunities, food access and nutrition, pollution and literacy, among others (Office of Disease Prevention and Health Promotion, 2020). When diagnosed with cancer, the SDOH impacts every step of the cancer journey. This journey goes from prevention to screening to diagnosis to treatment. Whether someone dies or survives is also connected to the SDOH (Cancer Care Ontario, 2018).

The racial differences in cancer outcomes can be stark. Age-adjusted breast cancer mortality rates are higher among African American women than Caucasian women. Caucasian women breast cancer survival rates have improved, while survival rates among African American women have improved much less. Data for lung cancer among African American men and colorectal cancer for both African American men and women show the same type of disparities when compared to their Caucasian counterparts. Then there is diagnosis of cancer, with African Americans being diagnosed at more advanced stages than their Caucasian counterparts in female breast cancer and both male and female lung and colorectal cancers (Esnaola and Ford, 2012).

The disparities are broad. The American Association for Cancer Research Cancer Disparities Progress Report 2020 identifies many populations facing disparities. These populations are: racial and ethnic minorities, individuals with differing ancestries, individuals with a low socioeconomic status, disabled individuals, individuals without or with limited insurance, people living in certain communities, members of the LGBT+ community, immigrants, asylum seekers or refugees and those who are adolescents and those who are elderly.

Only a national culture of health equity can lead to the changes needed in the United States. Such a culture would have an understanding of the community a patient comes from, understand the SDOH, actively acknowledge and work towards ending implicit bias, have empowered health care professionals available to all and bring health literacy to all in the community (Daniel et al., 2018).

Cancer Treatment Disparities Objectives:

1. Students will understand what Social Determinants of Health are and their importance.
2. Students will understand what health disparities occur among multiple demographic categories of the population.
3. Students will understand how to connect with their community to communicate health care disparities in cancer.

TEACHING STRATEGIES

Fruit Fly Cancer Research

This part of the curriculum will take 1-2 weeks. This curriculum should be done after teaching cell parts, cell cycle and mitosis so students have mastery of cell structure and division. On the first day of the unit cancer will be introduced as a concept. Students will be asked to fill out the first two parts of a KWL chart to show what they know, want to know and what they learned about cancer. After classroom discussion about what is written they turn these in. These charts will be returned later in the unit so they can fill in what they learned. There will be lecture and note-taking about what cancer is and how it is connected to genes. In groups, students will be asked to brainstorm ways to prevent or reduce risk of cancer. Students will share ideas and ultimately diet will be selected.

The unit's second day will bring a lesson on what a model organism is. Students will start with the question of why humans need to use other organisms to test for disease causes, like cancer, preventions and cures. After exploring this as a class, the concept of model organisms will be introduced. Students will investigate model organisms in a jigsaw activity in groups of four and then share out with the class. On the third day of the unit fruit flies will then be introduced as the model organism for the unit. There will be lecture and note-taking on fruit fly anatomy, metabolism, reproduction, life cycle, genes and chromosomes. Students will do a comparison on how fruit flies are similar to mammals and humans and how they are different. On the fourth day of the unit students would use microscopes to photograph and identify structures on the fruit flies as well as use the internet to search for other images to compile a dossier on the anatomy of a fruit fly throughout its development.

The unit's fifth day will synthesize the concepts of cancer, model organisms and fruit flies into an experiment with fruit flies. In groups of two to four, students will determine how fruit fly diet might impact cancer occurrence. Students will either pick a food source that they think is healthy or unhealthy and hypothesize how it might impact fruit flies that are genetically modified to have cancer. After determining their experimental question, hypothesis, independent and dependent variables, student groups will read and plan out the experiment. On the sixth day of the unit students will begin the experiment, having flies in vials with normal fruit fly food and in vials with the new fruit fly diet. Since students will not interact with the fruit flies again for 7 days, the rest of the experiment will occur during other parts of the unit that are not focused on fruit fly cancer research.

After 7 days, students will remove adult flies from the vials. Then, 8 days later, further on in the overall unit of study, students will record fruit fly pupa count and the number new adults. With all of this data, students can then determine how flies in normal food faired against the healthy or unhealthy food they used. With this data, students will produce a CER (Claim

Evidence Reasoning) paper. In this, they will make a claim based on the data and reason out whether or not the food they used impacted how cancer affected the fruit flies.

Cancer Cell Signaling

With EGFR and RAS being involved in so many different cancers, there is a great opportunity to have students research human cancers connected to these. On the first day of the Cancer Cell Signaling portion of the curriculum (which would come as the fruit fly experiment is occurring), there should be an introduction to cell signaling in general. Terms like ligand, receptor protein, kinase, extracellular, intracellular, signal transduction and proliferation should be introduced through lecture with note-taking. Since there is going to be a concentration on EGFR and RAS, students in groups of two to four (these can be the same groups as in fruit fly experiment or different groups) should be divided up so half of the group researches EGFR and the other half of the group researches RAS in the following ways: what each protein can do in cell signaling, what binds to them, some cell types they are found in and some organisms other than humans they are found in. Students will begin a Google Slideshow first with the definitions provided in the beginning of this part of the unit and then with this information on EGFR and RAS. Students will then share in their larger groups their findings and there would be a class discussion of answers.

The next part of this assignment, which will carry into the second and third day of this part of the unit, will involve students from the larger groups of two to four searching the internet for instances of EGFR and RAS being connected in cell signaling. As a group, students should find one unique image per student. They then should put the image on a Google Slide and fill translate into words what is going on. Students will have to do a fair amount of research for this, and will certainly get stumped with some concepts, but will overall develop a picture of the connections between these two proteins, their value and the complexity of cell signaling. Each group will then add images and explanations from this part of the unit to the Google Slideshow for Cancer Cell Signaling and share out with the class.

On the fourth day of the Cancer Cell Signaling part of the unit students will delve into mutations of these proteins and their cancerous results. Despite not covering genetics yet in class at this point (unless this is taught in AP or IB Biology, where students most likely would have already had a genetics backing from a previous biology class), students will select either EGFR or RAS (with half the students in each group picking one and the other half picking the other). Students will research what cancers are connected to the proteins (naming at least four), what types of mutations occur to the proteins (naming and describing at least two) and what is changed in the normal cell's activities to result in a cancerous cell. For the last part of this, students will go back to what they did in the previous part of the unit to talk about normal activity versus the cancerous activity. Students will add information from this research to the growing Google Slideshow from this unit. The class will discuss and by the end of the fifth day of the Cancer Cell Signaling each student will have a Google Slideshow to turn in as the assessed piece of work.

Cancer and Evolution

At this point in the school year, evolution generally has not been taught. Students should have a general understanding from previous life science units in lower grades about what

evolution is and what natural selection is. The first day of this part of the curriculum should begin with a review of what the two concepts are. Students will then begin a multi-day exploration into how similar and different humans are from other organisms when it comes to cancer, how natural selection acts towards cancer with regards to age and what Peto's Paradox says about evolution. Students will be in groups of three to six. In these groups, they will be divided up to read one original scientific paper on one of the three aforementioned topics. The papers and topics are:

1. What are similarities in cancers among organisms? -- Albuquerque, Thales A., et al. "From Humans to Hydra: Patterns of Cancer across the Tree of Life." *Biological Reviews*, vol. 93, no. 3, 2018, pp. 1715–1734., doi:10.1111/brv.12415.

2. How does selection act on aging and cancer risk? -- White, Mary C., et al. "Age and Cancer Risk." *American Journal of Preventive Medicine*, vol. 46, no. 3, 2014, doi:10.1016/j.amepre.2013.10.029.

3. What does Peto's Paradox say about caner evolution? -- Caulin, Aleah F., and Carlo C. Maley. "Peto's Paradox: Evolution's Prescription for Cancer Prevention." *Trends in Ecology & Evolution*, vol. 26, no. 4, 2011, pp. 175–182., doi:10.1016/j.tree.2011.01.002.

On what would be the third day of this part of the curriculum, students will shift back to Fruit Fly Cancer Research in order to remove adult flies from the vials. The next day, when students return to Cancer and Evolution, they will then analyze the title, abstract, introduction, figures, tables and conclusion to answer the question for each paper. Students will reconvene into larger groups to share out. Then, using all the information provided, which will be put into a Google Document examining each paper, each student will come up with a scientific problem they would like to research connecting evolution, cancer and finding ways to fight and possibly cure certain cancers. Each student would propose a question or problem statement, why they came up with the question or problem statement and what they would need to research to set up their experiment. Students would share these with the class. All in all, the Cancer and Evolution part of this unit will take five to seven days.

Cancer Treatment Disparities

For the final portion of this curriculum unit, students will work independently and produce a call to action about disparities in cancer health care in some form: a pamphlet, poster, public service announcement video, informational website, or a series of letters to the editor of a newspaper or politician, among others. This call to action would serve the greater good of each student's community, whether it be their school community, home community, part of Philadelphia they live in, Philadelphia itself or whatever the student defines as "community." Students will help this community understand what is not working in healthcare with relation to cancer and what can be done to change this.

The first day of the Cancer Treatment Disparities part of the curriculum students will learn about the Social Determinants of Health (SDOH) through lecture and note-taking. With this backing, students will do research for the rest of day one and throughout day two. They will look at one or more of the following articles:

1. Cancer Down Nationwide, but 'Hot Spots' Persist: https://www.cnn.com/2017/01/24/health/cancer-cluster-disparities-county-study/index.html

2. Racism in Cancer Care is Failing Black Patients. Can We Change the System?: https://www.prevention.com/health/health-conditions/a35473538/racism-cancer-black-patients/

3. Did Disparities Kill the King of Wakanda? Chadwick Boseman and Changing Landscape of Colon Cancer Demographics: https://www.statnews.com/2020/08/31/disparities-kill-king-of-wakanda-chadwick-boseman-changing-landscape-colon-cancer-demographics/

4. Tackling a Racial Gap in Breast Cancer Survival: https://www.nytimes.com/2013/12/20/health/tackling-a-racial-gap-in-breast-cancer-survival.html

From the article or articles each student reads, they will generate a list of at least five interesting facts and at least five further questions they have about the topic. Then they will use this to push them to research the questions as well as data online connected to cancer occurrence in Philadelphia and/or elsewhere by demographics – age, race, socioeconomic status, where they live and education. With the answers to these questions and the data, students will then do the call to action project. They will get three to four days to do this. Students will then present what they have produced with the class.

By the time this is part of the curriculum is done, the final part of the first part of the curriculum can be done, with data collection of fruit flies followed by CER construction. At the end of this entire curriculum, students will be getting back their KWL chart they filled in the beginning, with L left blank of course. Students now fill in at least 10 items in the L part and share in pairs or larger groups.

Recap of Whole Curriculum Timeline

*Days 1-6: Fruit Fly Cancer Research
*Days 7-11: Cancer Cell Signaling
*Days 12-13: Cancer and Evolution
*Day 14: Fruit Fly Cancer Research Continued
*Days 15-17: Cancer and Evolution Concludes(if more days adjust the schedule)
*Days 18-21: Cancer Treatment Disparities (if more days adjust the schedule)
*Days 22+: Fruit Fly Cancer Research Concludes

CLASSROOM ACTIVITIES

++Below are three classroom activity lesson plans that go along with this curriculum unit.

**Classroom Activity 1: Fruit Fly Cancer Research

Claim Evidence Reasoning Paper

In this activity, which is the culmination of the Fruit Fly Cancer Research part of the curriculum, students will take their data from their experiment and make a claim based on the results of whether or not the diet of the fruit flies impacted the how the fruit fly cancer affected the fruit fly population overall.

ESSENTIAL IDEA:

*Cancer is uncontrolled cell growth that can be modeled in fruit flies.

STANDARDS:

*BIO.B.1.1.2: Compare the processes and outcomes of mitotic and meiotic nuclear divisions.

*BIO.B.2.3.1: Describe how genetic mutations alter the DNA sequence and may or may not affect phenotype.

*BIO.B.3.1.3: Explain how genetic mutations may result in genotypic and phenotypic variations within a population.

*HS-LS1-4: Use a model to illustrate the role of cellular division and differentiation in producing and maintaining complex organisms.

CER (Note for this assignment, please look in the MATERIALS FOR CLASSROOM USE part of the RESOURCES at the end of this curriculum plan to find a standard rubric for CERs.):

1. Students will analyze data collected from fruit fly experiment on how many fruit fly pupa there are as well as how many new adults remained in vials after 15 days of the experiment. Based on this data students will make a claim about how diet affected the survival of fruit flies. Students will share claim each other in groups of 2-4. There will be a classroom discussion about proper claims. Claims will be revised as needed.
2. Students will take data and put it into table and graph form for the evidence part of the paper. They will share the evidence in groups of 2-4 (they could be the same groups as in #1 or different groups). There will be a classroom discussion about proper evidence. Evidence will be revised as needed.
3. Students will work with information learned in class about cancer and model organisms as well as the claim and evidence to come up with the overall reasoning part of the paper. They will make sure to include a guiding scientific principle to connect the reasoning to the claim and evidence. They will share the evidence in groups of 2-4 (they could be the same groups as in #1 and/or #2 or different groups). There will be a classroom discussion about proper reasoning. Reasoning will be revised as needed.
4. Students will then share entire revised CER, revised because of feedback on all three parts, in groups of 2-4 (they could be new groups or the same as and previous groups in parts #1, #2 and/or #3).
5. Students will then turn in entire revised CER.

EVALUATION:

*Students will be evaluated on their completion of each step of the writing and revision process and on the final draft of the CER.

**Classroom Activity 2: Cancer and Evolution

Reading a Scientific Paper on Cancer and Evolution

In this activity, each student is in a group of three to six and reading one of the three scientific journal articles. Students will analyze the title, abstract, introduction, figures, tables and conclusion to answer the question for each posed for whatever paper they are reading. They then share with the group and create a Google Document with information on all three journals as collected from all members in the group.

ESSENTIAL IDEA:

*Scientific paper literacy is key to understanding past and present science research.

STANDARDS:

*BIO.B.3.1.1: Explain how natural selection can impact allele frequencies of a population.

*BIO.B.3.2.1: Interpret evidence supporting the theory of evolution.

*HS-LS4-3: Apply concepts of statistics and probability to support that organisms with an advantageous heritable trait tend to increase in proportion to organisms lacking this trait.

SCIENTIFIC PAPER LITERACY:

1. Students will be in groups of three to six. If three, one of the following questions and accompanying articles will be assigned to each student. If six, each pair of students analyzes the same question and paper. The papers are:
    A. What are similarities in cancers among organisms? -- Albuquerque, Thales A., et al. “From Humans to Hydra: Patterns of Cancer across the Tree of Life.” *Biological Reviews*, vol. 93, no. 3, 2018, pp. 1715–1734., doi:10.1111/brv.12415.
    B. How does selection act on aging and cancer risk? -- White, Mary C., et al. “Age and Cancer Risk.” *American Journal of Preventive Medicine*, vol. 46, no. 3, 2014, doi:10.1016/j.amepre.2013.10.029.
    C. What does Peto’s Paradox say about caner evolution? -- Caulin, Aleah F., and Carlo C. Maley. “Peto's Paradox: Evolution's Prescription for Cancer Prevention.” *Trends in Ecology & Evolution*, vol. 26, no. 4, 2011, pp. 175–182., doi:10.1016/j.tree.2011.01.002.

2. Students will read the paper in order to answer the question of the paper.
3. Students will analyze the title, abstract, introduction, figures, tables and conclusion to answer the question for each posed for whatever paper they are reading. They will use the

guide called “Dissecting a scientific paper about evolutionary biology III” found at UC Berkeley’s Understanding Evolution site (https://evolution.berkeley.edu/evolibrary/teach/journal/dissectingapaper3.php) to guide them through this journey of reading and comprehending a scientific paper.

4. Students will type of their findings and share with the group out loud and by adding to a group Google Document they will turn in.
5. The group will revise the group Google Document based on discussion so that all the questions posed for the papers are answered and all the analysis information is correct.

EVALUATION:

*Students will be evaluated on how they individually answered their individual science paper question and individually analyzed the paper overall. They will also be evaluated on the group’s overall work on all questions and analysis.

**Classroom Activity 3: Cancer Treatment Disparities

Cancer Treatment Disparities Research, Inquiry and Call to Action

In this part of the Cancer Treatment Disparities section of the curriculum, students will read one or more articles provided, record information from each article and ask questions generated from the readings. With this starting point, students will research each of the questions generated as well as cancer demographics. This will be the background necessary for the call to action project that completes this activity.

ESSENTIAL IDEA:

*There are disparities in individual cancer diagnosis, treatment and survival.

STANDARDS:

*BIO.B.1.1.2: Compare the processes and outcomes of mitotic and meiotic nuclear divisions.

CANCER TREATMENT DISPARITIES RESEARCH, INQUIRY AND CALL TO ACTION:

1. Students will work individually. They will choose 1-2 of the following articles to read:

    A. Cancer Down Nationwide, but ‘Hot Spots’ Persist: https://www.cnn.com/2017/01/24/health/cancer-cluster-disparities-county-study/index.html
    B. Racism in Cancer Care is Failing Black Patients. Can We Change the System?: https://www.prevention.com/health/health-conditions/a35473538/racism-cancer-black-patients/
    C. Did Disparities Kill the King of Wakanda? Chadwick Boseman and Changing Landscape of Colon Cancer Demographics: https://www.statnews.com/2020/08/31/di

sparities-kill-king-of-wakanda-chadwick-boseman-changing-landscape-colon-cancer-demographics/

D. Tackling a Racial Gap in Breast Cancer Survival: https://www.nytimes.com/2013/12/20/health/tackling-a-racial-gap-in-breast-cancer-survival.html

2. Students will write down a list of at list fiver interesting acts from each article as well as five questions generated from each article.
3. Students will use the questions to then do independent research. They will research each question using the internet, recording website information, data and other valuable information to answer each question.
4. Students will research cancer demographics for Philadelphia and elsewhere. The demographics they will research include age, race, socioeconomic status, where they live and education.
5. All of this generated information will then be the background information for a call to action. Students will figure out how to use the information acquired to serve the greater good of their community. This community could be the school, home, neighborhood, Philadelphia overall or some other space the student defines as "community."
6. Students will help inform this community of the cancer treatment disparities through one of the following formats (or another format possibly): pamphlet, poster, public service announcement video, informational website, letters to the editor of a newspaper or letters to a politician.
7. Students will then present the call to action to the whole class.

RESOURCES

+Bibliography for Teachers

*American Cancer Society. *Cancer Facts & Figures for African Americans 2019-2021*. American Cancer Society, 2019.

This resource is connected to Cancer Treatment Disparities and provides cancer facts and figures on African Americans.

*Abegglen, Lisa M., et al. "Potential Mechanisms for Cancer Resistance in Elephants and Comparative Cellular Response to DNA Damage in Humans." *JAMA*, vol. 314, no. 17, 2015, p. 1850., doi:10.1001/jama.2015.13134.

This resource is connected to Cancer and Evolution and provides information on types of cancer resistance in elephants.

*Albuquerque, Thales A., et al. "From Humans to Hydra: Patterns of Cancer across the Tree of Life." *Biological Reviews*, vol. 93, no. 3, 2018, pp. 1715–1734., doi:10.1111/brv.12415.

This resource is connected to Cancer and Evolution and provides information on types of cancers differing organisms have.

*Augustus, Gaius J, and Nathan A Ellis. “Colorectal Cancer Disparity in African Americans: Risk Factors and Carcinogenic Mechanisms.” *The American journal of pathology* vol. 188,2 (2018): 291-303. doi:10.1016/j.ajpath.2017.07.023

This resource is connected to the curriculum introduction and provides information on colorectal cancer disparities.

*Barberán, Sara et al. “Evolution of the EGFR pathway in Metazoa and its diversification in the planarian Schmidtea mediterranea.” *Scientific reports* vol. 6 28071. 21 Jun. 2016, doi:10.1038/srep28071

This resource is connected to Cancer and Evolution and provides information on EGFR evolution.

*Bethune, Gillian et al. “Epidermal growth factor receptor (EGFR) in lung cancer: an overview and update.” *Journal of thoracic disease* vol. 2,1 (2010): 48-51.

This resource is connected to Cancer Cell Signaling and provides information on EGFR in lung cancer.

*Campbell, Neil A., and Jane B. Reece. *Biology*. Pearson, Benjamin Cummings, 2005.

This resource is connected to Cancer Cell Signaling and provides information on signal transduction.

*Caulin, Aleah F., and Carlo C. Maley. “Peto's Paradox: Evolution's Prescription for Cancer Prevention.” *Trends in Ecology & Evolution*, vol. 26, no. 4, 2011, pp. 175–182., doi:10.1016/j.tree.2011.01.002.

This resource is connected to Cancer and Evolution and provides information on Peto’s Paradox.

*Chien, S. “Homophila: Human Disease Gene Cognates in Drosophila.” *Nucleic Acids Research*, vol. 30, no. 1, 2002, pp. 149–151., doi:10.1093/nar/30.1.149.

This resource is connected to Fruit Fly Cancer Research and is a resource for connections in researching human genetic diseases and studying fruit flies.

*Daniel, Hilary, et al. “Addressing Social Determinants to Improve Patient Care and Promote Health Equity: An American College of Physicians Position Paper.” *Annals of Internal Medicine*, vol. 168, no. 8, 2018, p. 577., doi:10.7326/m17-2441.

This resource is connected to Cancer Treatment Disparities and provides information on how to promote health equity.

*Domazet-Lošo, Tomislav, et al. “Naturally Occurring Tumours in the Basal Metazoan Hydra.” *Nature Communications*, vol. 5, no. 1, 2014, doi:10.1038/ncomms5222.

This resource is connected to Cancer and Evolution and provides information on cancer in hydra.

*Doonan, John H., and Robert Sablowski. “Walls around Tumours — Why Plants Do Not Develop Cancer.” *Nature Reviews Cancer*, vol. 10, no. 11, 2010, pp. 794–802., doi:10.1038/nrc2942.

This resource is connected to Cancer and Evolution and provides information on cancer in plants.

*Esnaola, Nestor F, and Marvella E Ford. “Racial differences and disparities in cancer care and outcomes: where's the rub?.” *Surgical oncology clinics of North America* vol. 21,3 (2012): 417-37, viii. doi:10.1016/j.soc.2012.03.012

This resource is connected to Cancer Treatment Disparities and provides information on racial differences and disparities in cancer care and outcomes.

*Feitelson, Mark A., et al. “Sustained Proliferation in Cancer: Mechanisms and Novel Therapeutic Targets.” *Seminars in Cancer Biology*, vol. 35, 2015, doi:10.1016/j.semcancer.2015.02.006.

This resource is connected to Fruit Fly Cancer Research AND Cancer Cell Signaling and provides information on sustained proliferation of cancer development and progression.

*Gewin, Virginia. “Massive Animals May Hold Secrets of Cancer Suppression.” *Nature*, 2013, doi:10.1038/nature.2013.12258.

This resource is connected to Cancer and Evolution and provides information on animal size and cancer suppression.

*Gou, Hong-Feng et al. “Epidermal growth factor receptor (EGFR)-RAS signaling pathway in penile squamous cell carcinoma.” *PloS one* vol. 8,4 e62175. 24 Apr. 2013, doi:10.1371/journal.pone.0062175

This resource is connected to Cancer Cell Signaling and provides information on the EGFR-RAS signaling pathway in a penile cancer.

*Gurung, Arun Bahadur, and Atanu Bhattacharjee. “Significance of Ras Signaling in Cancer and Strategies for Its Control.” *Oncology & Hematology Review (US)*, vol. 11, no. 02, 2015, p. 147., doi:10.17925/ohr.2015.11.02.147.

This resource is connected to Cancer Cell Signaling and provides information on RAS and cancer.

*Hanahan, Douglas, and Robert A Weinberg. “The Hallmarks of Cancer.” *Cell*, vol. 100, no. 1, 2000, pp. 57–70., doi:10.1016/s0092-8674(00)81683-9.

This resource is connected to Fruit Fly Cancer Research and provides information about epithelial cancers.

*Huang, Huan. “Signal Transduction in Trypanosoma Cruzi.” *Advances in Parasitology*, 2011, pp. 325–344., doi:10.1016/b978-0-12-385863-4.00015-0.

This resource is connected to Cancer Cell Signaling and provides information on signal transduction.

*Kirkwood, Thomas B., and Steven N. Austad. “Why Do We Age?” *Nature*, vol. 408, no. 6809, 2000, pp. 233–238., doi:10.1038/35041682.

This resource is connected to Cancer and Evolution and provides information on aging.

*Leroi, Armand M., et al. “Cancer Selection.” *Nature Reviews Cancer*, vol. 3, no. 3, 2003, pp. 226–231., doi:10.1038/nrc1016.

This resource is connected to Cancer and Evolution and provides information on cancer selection.

*Linford, Nancy J., et al. “Measurement of Lifespan in *Drosophila Melanogaster*.” *Journal of Visualized Experiments*, no. 71, 2013, doi:10.3791/50068.

This resource is connected to Fruit Fly Cancer Research and provides information about fruit fly lifespan.

*****Livengood K, Diez Roux A, Mullin G, Acharya B, Bettigole C, Moore K, Rollins H, Washington R. *Community Brief: Cancer in Philadelphia*. Drexel University Urban Health Collaborative, March 2020.

This resource is connected to the curriculum introduction and provides information on cancer health disparities in Philadelphia.

*****Lodish, A, et al. *Molecular Cell Biology 4th Edition*. W.H. Freeman, 2000.

This resource is connected to Fruit Fly Cancer Research and Cancer Cell Signaling. It provides background knowledge of cancer and cell signaling terminology.

*Lusk, Jay B et al. “Epidermal Growth Factor Pathway Signaling in Drosophila Embryogenesis: Tools for Understanding Cancer.” *Cancers* vol. 9,2 16. 7 Feb. 2017, doi:10.3390/cancers9020016

This resource is connected to Cancer Cell Signaling and provides information on EGF pathways in fruit flies.

*Millburn, Gillian H., et al. “FlyBase Portals to Human Disease Research Using Drosophila Models.” *Disease Models & Mechanisms*, vol. 9, no. 3, 2016, pp. 245–252., doi:10.1242/dmm.023317.

This resource is connected to Fruit Fly Cancer Research and provides information about similarities in cell growth signaling pathways.

*Mirzoyan, Zhasmine, et al. “Drosophila Melanogaster: A Model Organism to Study Cancer.” *Frontiers in Genetics*, vol. 10, 2019, doi:10.3389/fgene.2019.00051.

This resource is connected to Fruit Fly Cancer Research and provides information about how valuable fruit flies are as model organisms.

*Morales, Eva et al. “Food and nutrient intakes and K-ras mutations in exocrine pancreatic cancer.” *Journal of epidemiology and community health* vol. 61,7 (2007): 641-9. doi:10.1136/jech.2007.060632

This resource is connected to Fruit Fly Cancer Research and provides information about the connection between food and Ras mutations.

*Oda, Kanae et al. “A comprehensive pathway map of epidermal growth factor receptor signaling.” *Molecular systems biology* vol. 1 (2005): 2005.0010. doi:10.1038/msb4100014

This resource is connected to Cancer Cell Signaling and provides information on EGFR cell signaling.

*Peto, R, et al. “Cancer and Ageing in Mice and Men.” *British Journal of Cancer*, vol. 32, no. 4, 1975, pp. 411–426., doi:10.1038/bjc.1975.242.

This resource is connected to Cancer and Evolution and provides information on what becomes Peto’s Paradox.

*Philadelphia, City of. “City of Philadelphia Community Health Explorer.” *Community Health Explorer*, Phila.gov, healthexplorer.phila.gov/racial-disparity/.

This resource is connected to Cancer Treatment Disparities and is a database of Philadelphia health statistics.

*Puppels, G.J. “Confocal Raman Microspectroscopy.” *Fluorescent and Luminescent Probes for Biological Activity*, 1999, pp. 377–406., doi:10.1016/b978-012447836-7/50031-2.

This resource is connected to Fruit Fly Cancer Research and provides information about value of fruit fly chromosome size.

*Pylayeva-Gupta, Yuliya, et al. “RAS Oncogenes: Weaving a Tumorigenic Web.” *Nature Reviews Cancer*, vol. 11, no. 11, 2011, pp. 761–774., doi:10.1038/nrc3106.

This resource is connected to Cancer Cell Signaling and provides information on RAS and cancer.

* Richardson, Lisa C., et al. “Patterns and Trends in Age-Specific Black-White Differences in Breast Cancer Incidence and Mortality – United States, 1999–2014.” *MMWR. Morbidity and Mortality Weekly Report*, vol. 65, no. 40, 2016, pp. 1093–1098., doi:10.15585/mmwr.mm6540a1.

This resource is connected to Cancer Treatment Disparities and focuses on differences in breast cancer incidence and mortality between African Americans and Caucasians.

*Rojas, Ana Maria et al. “The Ras protein superfamily: evolutionary tree and role of conserved amino acids.” *The Journal of cell biology* vol. 196,2 (2012): 189-201. doi:10.1083/jcb.201103008

This resource is connected to Cancer and Evolution and provides information on the evolution of RAS.

*Rothschild, B. M., et al. “Epidemiologic Study of Tumors in Dinosaurs.” *Naturwissenschaften*, vol. 90, no. 11, 2003, pp. 495–500., doi:10.1007/s00114-003-0473-9.

This resource is connected to Cancer and Evolution and provides information on cancer in dinosaurs.

*Saad, Ismail If et al. “The RAS subfamily Evolution - tracing evolution for its utmost exploitation.” *Bioinformation* vol. 10,5 293-8. 20 May. 2014, doi:10.6026/97320630010293

This resource is connected to Cancer and Evolution and provides information on the evolution of RAS.

*Simanshu, Dhirendra K et al. “RAS Proteins and Their Regulators in Human Disease.” *Cell* vol. 170,1 (2017): 17-33. doi:10.1016/j.cell.2017.06.009

This resource is connected to Cancer Cell Signaling and provides information on RAS proteins and their regulators in human disease.

*Sigismund, Sara et al. “Emerging functions of the EGFR in cancer.” *Molecular oncology* vol. 12,1 (2018): 3-20. doi:10.1002/1878-0261.12155

This resource is connected to Cancer Cell Signaling and provides information on EGFR and cancer.

*Tollis, Marc, et al. “Peto’s Paradox: How Has Evolution Solved the Problem of Cancer Prevention?” *BMC Biology*, vol. 15, no. 1, 2017, doi:10.1186/s12915-017-0401-7.

This resource is connected to Cancer and Evolution and provides information on Peto’s Paradox.

*Ugur, Berrak, et al. “Drosophila Tools and Assays for the Study of Human Diseases.” *Disease Models & Mechanisms*, vol. 9, no. 3, 2016, pp. 235–244., doi:10.1242/dmm.023762.

This resource is connected to Fruit Fly Cancer Research and provides information about the amount of genes shared between flies and humans.

*Weinberg, R A. “How cancer arises.” *Scientific American* vol. 275,3 (1996): 62-70.

This resource is connected to Fruit Fly Cancer Research and provides background knowledge of how cancer arises.

*Wennerberg, Krister, et al. “The Ras Superfamily at a Glance.” *Journal of Cell Science*, vol. 118, no. 5, 2005, pp. 843–846., doi:10.1242/jcs.01660.

This resource is connected to Cancer Cell Signaling and provides information on RAS.

*White, Mary C., et al. “Age and Cancer Risk.” *American Journal of Preventive Medicine*, vol. 46, no. 3, 2014, doi:10.1016/j.amepre.2013.10.029.

This resource is connected to Cancer and Evolution and provides information on how natural selection is connected aging and cancer.

*Wieduwilt, M J, and M M Moasser. “The epidermal growth factor receptor family: biology driving targeted therapeutics.” *Cellular and molecular life sciences : CMLS* vol. 65,10 (2008): 1566-84. doi:10.1007/s00018-008-7440-8

This resource is connected to Cancer Cell Signaling and provides information on EGFR and its essential roles in cells.

*Yamamoto, Shinya, et al. “A Drosophila Genetic Resource of Mutants to Study Mechanisms Underlying Human Genetic Diseases.” *Cell*, vol. 159, no. 1, 2014, pp. 200–214., doi:10.1016/j.cell.2014.09.002.

This resource is connected to Fruit Fly Cancer Research and is a resource for fruit fly mutation usage for studying human genetic diseases.

*“Cancer Facts & Figures 2021.” *American Cancer Society*, American Cancer Society, www.cancer.org/research/cancer-facts-statistics/all-cancer-facts-figures/cancer-facts-figures-2021.html.

This resource is connected to Cancer Treatment Disparities and has current data on cancer.

***** "Colon Cancer Treatment Pathway Map." *Colon Cancer Treatment Pathway*, Cancer Care Ontario, 2018, www.cancercareontario.ca/sites/ccocancercare/files/assets/DPMColonTreatment.pdf.

This resource is connected to Cancer Treatment Disparities and shows what the cancer treatment pathway is.

*****"Ras Pathway." *Addgene*, Addgene, www.addgene.org/cancer/ras-pathway/.

This resource is connected to Cancer Cell Signaling and provides a resource for visualizing the RAS pathway.

*****"The RAS Initiative." *National Cancer Institute*, National Institutes of Health, www.cancer.gov/research/key-initiatives/ras.

This resource is connected to Cancer Cell Signaling and provides a resource for understanding the link between RAS and cancer.

*****"Social Determinants of Health." *Social Determinants of Health - Healthy People 2030*, Office of Disease Prevention and Health Promotion, 2020, health.gov/healthypeople/objectives-and-data/social-determinants-health.

This resource is connected to Cancer Treatment Disparities and explains what the Social Determinants of Health are.

*****"What are Model Organisms?" *Your Genome*, The Public Engagement Team at the Wellcome Genome Campus, 19 June 2015, www.yourgenome.org/facts/what-are-model-organisms.

This resource is connected to Fruit Fly Cancer Research and provides information about the value of model organisms to research.

*****"Why Use The Fly in Research?" *Your Genome*, The Public Engagement Team at the Wellcome Genome Campus, 19 June 2015, www.yourgenome.org/facts/why-use-the-fly-in-research.

This resource is connected to Fruit Fly Cancer Research and provides information about the value of fruit flies to research.

**Cancer Disparities Progress Report*, American Association for Cancer Research, 2020, CancerDisparitiesProgressReport.org.

This resource is connected to Cancer Treatment Disparities and is a progress report that reveals what the current cancer disparities are in the U.S.

**My Cancer Genome*, Vanderbilt-Ingram Cancer Center, 2017, www.mycancergenome.org/.

This resource is connected to Cancer Cell Signaling and provides a resource for finding out about how certain proteins and their mutations are connected to certain cancers.

+Reading List for Students

Fruit Fly Cancer Research

*****"What are Model Organisms?" *Your Genome*, The Public Engagement Team at the Wellcome Genome Campus, 19 June 2015, www.yourgenome.org/facts/what-are-model-organisms.

This resource provides information about the value of model organisms to research.

Cancer Cell Signaling

*Campbell, Neil A., and Jane B. Reece. *Biology*. Pearson, Benjamin Cummings, 2005.

This resource provides information vital to understanding cell signaling.

Cancer and Evolution

*Albuquerque, Thales A., et al. "From Humans to Hydra: Patterns of Cancer across the Tree of Life." *Biological Reviews*, vol. 93, no. 3, 2018, pp. 1715–1734., doi:10.1111/brv.12415.
This is one of the scientific papers assigned for this part of the unit.

*White, Mary C., et al. "Age and Cancer Risk." *American Journal of Preventive Medicine*, vol. 46, no. 3, 2014, doi:10.1016/j.amepre.2013.10.029.

This is one of the scientific papers assigned for this part of the unit.

*Caulin, Aleah F., and Carlo C. Maley. "Peto's Paradox: Evolution's Prescription for Cancer Prevention." *Trends in Ecology & Evolution*, vol. 26, no. 4, 2011, pp. 175–182., doi:10.1016/j.tree.2011.01.002.

This is one of the scientific papers assigned for this part of the unit.

*Dissecting a scientific paper about evolutionary biology III, https://evolution.berkeley.edu/evolibrary/teach/journal/dissectingapaper3.php

This resource is necessary for help with evaluating scientific papers.

Cancer Treatment Disparities

*Cancer Down Nationwide, but 'Hot Spots' Persist, https://www.cnn.com/2017/01/24/health/cancer-cluster-disparities-county-study/index.html

This is one of the articles assigned for this part of the unit.

*Racism in Cancer Care is Failing Black Patients. Can We Change the System?, https://www.prevention.com/health/health-conditions/a35473538/racism-cancer-black-patients/

This is one of the articles assigned for this part of the unit.

*Did Disparities Kill the King of Wakanda? Chadwick Boseman and Changing Landscape of Colon Cancer Demographics, https://www.statnews.com/2020/08/31/disparities-kill-king-of-wakanda-chadwick-boseman-changing-landscape-colon-cancer-demographics/

This is one of the articles assigned for this part of the unit.

*Tackling a Racial Gap in Breast Cancer Survival, https://www.nytimes.com/2013/12/20/health/tackling-a-racial-gap-in-breast-cancer-survival.html

This is one of the articles assigned for this part of the unit.

+Materials for Classroom Use

Fruit Fly Cancer Research

*"An Introduction to Fruit Flies." *The Berg Lab*, University of Washington, 11 July 2017, depts.washington.edu/cberglab/wordpress/outreach/an-introduction-to-fruit-flies/.

This resource discusses the fruit flies and their value for research.

**EClose Institute*, EClose Institute, 10 May 2021, ecloseinstitute.org/.

This resource is connected to the suggested fruit fly experiment involving genetically altered fruit flies and diet changes.

*Cancer Resources on HHMI's BioInteractive, https://www.hhmi.org/sites/default/files/Biointeractive/Outreach/NABT%20Cancer2.pdf

This resource collects many valuable items on HHMI's BioInteractive site.

* "Scientific Explanations: Claim Evidence Reasoning." https://msleablog.files.wordpress.com/2017/03/37ee4-1447362717821.jpg

This resource provides an outline of the requirements for a Claim Evidence Response paper.

Cancer Cell Signaling

**My Cancer Genome*, Vanderbilt-Ingram Cancer Center, 2017, www.mycancergenome.org/.

This resource provides a resource for finding out about how certain proteins and their mutations are connected to certain cancers.

Cancer and Evolution

*Evo in the news: Another perspective on cancer, https://evolution.berkeley.edu/evolibrary/search/lessonsummary.php?&thisaudience=13-16&resource_id=184

This resource provides a lesson on analyzing information on the evolutionary underpinnings of cancer.

Cancer Treatment Disparities

*Cancer Disparities, https://www.cancer.gov/about-cancer/understanding/disparities

This resource provides a wealth of information on cancer disparities with links to other vital information about what cancer is and cancer statistics.

Appendix

When thinking about how the unit plan implements the Pennsylvania science standards and Next Generation Science Standards, each of the following unit sections use the corresponding standard(s):

Fruit Fly Cancer Research

*BIO.A.1.2.2: Describe and interpret relationships between structure and function at various levels of biological organization.

Since model organisms are examined, relationships between organisms are revealed.

*BIO.B.1.1.2: Compare the processes and outcomes of mitotic and meiotic nuclear divisions.

This part of the unit deals with cancer, which is a possible outcome of cellular reproduction.

*HS-LS1-4: Use a model to illustrate the role of cellular division and differentiation in producing and maintaining complex organisms.

A model organism is used to illustrate how cellular division can go wrong to produce cancer cells.

Cancer Cell Signaling

*BIO.B.1.1.2: Compare the processes and outcomes of mitotic and meiotic nuclear divisions.

This part of the unit deals with cancer, which is a connected to cell signaling errors.

*BIO.B.2.3.1: Describe how genetic mutations alter the DNA sequence and may or may not affect phenotype.

This part of the unit deals with how cell signaling proteins can be mutated, leading to cancer cell proliferation.

*HS-LS1-4: Use a model to illustrate the role of cellular division and differentiation in producing and maintaining complex organisms.

A model organism is used to illustrate how cellular division can go wrong in connection with cell signaling errors.

Cancer and Evolution

*BIO.B.3.1.1: Explain how natural selection can impact allele frequencies of a population.

This part of the unit directly discusses how natural selection is connected to cancer.

*BIO.B.3.2.1: Interpret evidence supporting the theory of evolution.

This part of the unit shows how many different types of organisms can get cancer, showing common ancestry.

*HS-LS4-3: Apply concepts of statistics and probability to support that organisms with an advantageous heritable trait tend to increase in proportion to organisms lacking this trait.

This part of the unit is connected to Peto's Paradox and how large animals have lower cancer occurrences because of organisms having traits allowing for them to avoid cancer.

Cancer Treatment Disparities

*BIO.B.1.1.2: Compare the processes and outcomes of mitotic and meiotic nuclear divisions.

This part of the unit directly deals with the results of mitosis that are cancerous.