Interactive computer simulations for teaching tumor immunology

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Funded through the American Association of Immunologists

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Teacher Section

I. Overview

The concepts that surround cancer are often times oversimplified and misunderstood. Students' beliefs run the spectrum from it being something you can catch from one person, such as the flu, to something that is inherited. Other conceptual pitfalls include the idea that cancer is a single mutation event and that your body can do little to defend itself once a cancerous cell arises. Compounded by current treatments of cancer, surgery, chemotherapy, and radiation, the idea that there is an immune response similar to that elicited by foreign pathogens is one lost on most students.

This computer simulation exercise provides students the chance to develop an understanding of key concepts as it relates to cancer and immune surveillance. The first concept being that cancer arises from the accumulation of mutations, which results in uncontrolled proliferation and spread. The second concept is immune surveillance, which posits that in healthy individuals tumors are recognized and eliminated by the immune system before progressing to clinical disease.

Critical to these two concepts are the ideas that the human body can be compromised by both external pathogens and by continually arising transformed cells. Secondly, as a result of cell surface proteins, the body differentiates between "self" versus "non-self" cell types. Thirdly, cells can elicit an immune response if they are regarded as "dangerous", conversely going unchecked until deemed a problem by the body. Finally, the body is always balancing levels of good and bad cells via elimination, equilibrium, and escape.

Working either by their self or in small groups, students will monitor and manipulate levels of cellular proteins, which will determine whether a cell becomes a cancer cell. In addition, students will be able to quantify the effects of changing the levels of proteins and whether the body responds in kind with an immune response.

These lessons are geared for a general biology course, but they can be adapted to other levels depending on models used. These exercises are targeted at addressing the standards outlined in the *Benchmarks for Scientific Literacy* as well as the *National Science Education Standards.*

National Benchmarks and Standards:
Benchmarks for Scientific Literacy (American Association for the Advancement of Science – Project 2061)

Benchmark 5C-Cells
- Grades 6-8
  - "All living things are composed of cells…" pg. 112
  - "Cells repeatedly divide…" pg.112
- Grades 9-12
  - "Every cell is covered by a membrane that controls what can enter and leave the cell. In all but quite primitive cells, a complex network of proteins provides organization and shape and, for animal cells, movement." pg.113
  - "Within every cell are specialized parts…most cells in multicellular organisms perform some special functions that others do not." pg. 113
  - "**Gene mutation in a cell can result in uncontrolled cell division, called cancer.** Exposure of cells to certain chemicals or radiation increases mutations and thus increases the chance of cancer" pg.114

Benchmark 6C – Basic Functions
- Grades 6-8
  - "Specialized cells and the molecules they produce identify and destroy microbes that get inside the body." pg.137
  - "The immune system is designed to protect…and against some cancer cells that arise within." pg. 138
  - "Communication between cells is required to coordinate their diverse activities…" pg.138

National Science Education Standards (National Research Council)

- Life Science Content Standard C Grades 5-8
  - Structure and Function in Living Systems
    - **"Disease is a breakdown in structures or functions of an organism. Some diseases are the result of intrinsic failures of the system. Others are the result of damage by infection by other organisms." pg. 157**
- Life Science Content Standard C Grades 9-12
  - The Cell
    - Cells have particular structures that underlie their functions.
    - Inside are thousands of molecules and specialized structures.
    - Cells store and use information to guide their function
    - **"Cell functions are regulated. Regulation occurs both through changes in the activity of the functions performed by proteins and through the selective expression of individual genes. This regulation allows cells to respond to their environment and to control and coordinate cell growth and division." pg.184**

Science Background:

Concept 1:

Cancer is not the product of one mutation, but rather it is a product of an accumulation of mutations. Similar to a see-saw, the influences which drive cells to make copies of themselves have to be countered by the non-proliferative influences. It is when this biochemical balance becomes disrupted that tumor formation results. The following are the four mutagenic influences which result in cancer when they work with each other.

- Mutation 1: Loss of Cell Cycle Inhibitor - If a cell's gene making it sensitive to anti-growth signals the cell will proliferate. The effect is similar to a car losing its brakes.
    - This cell will continue to divide but will be limited by the amount of growth factor receptors on the cells surfaces. When the # of receptors decay, the effect of this first mutation is muted.

- Mutation 2: Self-sufficient Growth Oncogene – Oncogene is derived from the word *onkos*, which is greek for tumor or mass. An oncogene whose product when expressed at high levels cause normal cells to become cancerous. When this gene is mutated, a cell can produce a protein that drives a cell defying the directive of cellular death. Oncogenes are normal and stimulate growth at normal rates. When mutated, it is similar to having a weight on the accelerator of a car.
    - This mutation's effect is limited by shortening telomeres upon each cell division. If the telomeres become too short, self-sufficient growth is muted.

- Mutation 3: Activation of Telomerase Gene – Normal cells have a limited number of divisions it can undergo. With each division, the chromosomal cap, which is called a telomere, shortens. This provides a limit to division. However, if a mutation induces telomerase production, telomeres will not shorten. An example of this is the HeLa cell line. This line of immortal cells has been used in laboratories since 1951.
    - This mutation is limited by apoptosis pathway (cell death pathway). Activating the telomerase gene will only be effective if the programming controlling cellular death can be disrupted or mutated.

- Mutation 4: Inactivation of Apoptosis Pathway – Critical genes that control when a cell will die can be interfered with (ex. Papillomavirus gene interfering with the p53 protein critical to cell apoptosis) or mutated. This final mutation could promote the Bcl-2 oncoprotein which has anti-apoptotic effects. A recent study entitled *bcl-2 oncoprotein in colorectal hyperplastic polyps, adenomas, and adenocarcinomas* suggested that the bcl-2 oncoprotein may play a role in colorectal tumorigenesis.

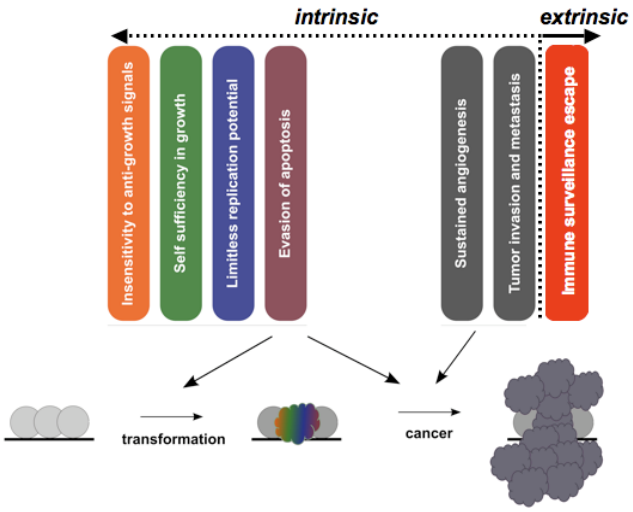


Figure 1. Each mutation by itself is not enough to generate a cancerous cell, however these mutations, when enacted at the same time, will provide enough of an impetus for uncontrolled cell growth.

A cluster of tumor cells may have the same mutations since they are the derivative of a progenitor cell. However, not all cells experience the same conditions. Some at the center of the cluster may face shortages of oxygen and nutrients while others may fragment away to begin tumor proliferation in another body tissue. So even at the cellular level, the homeostatic balance of cellular survival is a product of both nature and nurture.

The balance of cellular creation and apoptosis (death) in normal cells is expressed by the ratio 1 born : 1 dies. A tumor however is a result of this ratio being altered. Too much cell birth or too little cell death results in a tumor. It does not take a large difference in cell birth or death to generate a large tumor over time. When speaking of this homeostatic relationship, refer to the three E's, Equlibrium, Elimination, and Escape.

- Equilibrium – More cancer cells dying than arising
- Elimination – Equal number of cancer cells dying as arising
- Escape – More cancer cells arising than dying

Concept 2:

Immune surveillance relates to the interaction of the immune system and those cells involved in tumor formation. The immune system must recognize and eliminate those cells before they can progress to clinical disease. It is well known that the body's immune system is responsible for dealing with external pathogens such as bacteria (ex. Staphylococcus), viruses (ex. Herpes), as well as parasites (ex. Schistosoma). It is less known that pre-cancerous and cancer cells are also under surveillance by the immune system. The mammalian immune system has evolved two measurable defense mechanisms known as the innate and adaptive immunity response systems. The innate system responds to pathogens without specificity and will not "remember" further subsequent infections. Examples of these cell types are macrophages, which are dispersed throughout the body and respond to infections by recruiting other immune cells via protein production of cytokines. These cells can also take part in clearing the infection by engulfing (phagocytosis) pathogenic organisms. The adaptive immune

system has specificity and memory. Examples of these cell types are B and T cells. B cells are produced in the bone marrow and generate anti-bodies who bind to the future pathogens. T cells are made in the thymus and will either signal other immune cells or take part in killing damaged and/or infected cells.

Just as the color of a soldier's uniform is an indicator to friends and foe as to whose side they are on, the immune system uses surface proteins on cells as indicators as to their nature. The problem of "self vs. non-self" is exacerbated by the fact that many of the same marker proteins that exist on the surface of a normal cell, also exist on the surface of a cancer cell. It is the amount of marker proteins that can evoke a differential immune response.

One theory proposed as to the action of the immune system is the stranger and danger theory. Once a cell has been determined to be either like the tissue surrounding it or different, the immune system must decide the next action. Some cells may be noted by the body as being different (stranger), however not worthy of T cell execution (danger). This may explain why resident bacteria are not eliminated as they do not present danger signals. If a foreign or native cell causes other cells to die or undergo lysis, antigens (danger signals) are released and are ultimately recognized and learned by T cells. The presence of tumor antigens and their recognition by the immune system are the key to the elimination of cancerous cells.

EduClient Download:

In order to run the software tool, you'll need to download the EduClient application. This application can be found at the following address:

http://www.endogenics.com/education/index.php?page=client

You'll have the option of downloading an Apple, Windows, or Linux version of the software.

Once downloaded, open the EduClient software and choose the ***Multi-hit mutagenesis model of cancer.***

All exercises the students are asked to complete pertain to the above model.

Student Outcomes

Students will be able to combine facts and ideas they've learned through direct instructional medium (lecture, worksheets) and the use of computer aided simulations to create novel interpretations based on the data returned. Because the models provide some freedom of choice as well as conceptual scaffolding, students will be able to carry out trial and error in silico experiments eventually synthesizing predictions based on those models. The goal is not to show the students what they need to derive, but rather allow them to arrive at their own conclusions.

Learning Objectives:

Cancer

- Learn that cancer is a multi-step mutation driven process that accumulates over time.
- Identify the four mutation driven influences.
- Perform the cause and effect nature of each mutation .
- Derive what multiple mutations elicit when combined together .
- Explain the homeostatic relationship between that of normal cells as well as that of cancer cells when speaking of equilibrium, elimination, and escape.

Immune Surveillance

- Learn the roles of B and T cells when responding to both internal and external pathogens.
- Perform cause and effect trials as it relates to antigen presenting proteins. Manipulate protein levels to generate novel data.
- Synthesize questions and generate answers as a result of running computer models.

Time Requirements:

Pre-Cell Simulation

- 50 minute periods, approximately 5 days
    - Day 1, 2 – Introduce Prokaryotes and Eukaryotes
        - Lab: Prokaryotes
    - Day 3, 4 – Introduce Eukaryotes
        - Lab: Eukaryotes – Onion, Cheek Cell, Other.
    - Day 5: Cell Cycle, Pre-Quiz

Cell Simulations

- 50 minute periods, approximately 5 Days
    - Day 5 – Introduction to Cancer and Immunology
        - Class Discussion: "What Do You Know)
    - Day 6 - Cell Simulation Activity 1
    - Day 7 – Cell Simulation Activity 2
    - Day 8 – Cell Simulation Activity 3
    - Day 9 – Cell Simulation Activity 4
    - Day 10 – Wrap-Up, Post-Quiz

Advanced Preparation / Materials and Equipment

- Computer Lab: 15-30 computers (based on 30 students per class)
- Internet connectivity, preferably high speed
- Microscopes / Lens Paper
- Prepared Slides
- Methylene Blue
- Onion
- Toothpicks
- Eyedropper
- Glass Slides / Cover Slips

Student Prior Knowledge and Skills

Students should have a prior knowledge of the cell theory, cell types, and basic cellular structures. They will need to know how to operate microscopes as well as prepare wet mount slides. They need to have familiarity with computers, specifically opening a web browser as well as manipulating editable fields.

Common Misconceptions regarding Cancer and the Immune System

- While most diseases have a genetic component, many students believe that most cancer is an inherited disease, examples provided being BRCA 1 and BRCA 2.
- Cancer rates are perceived to be on the rise however since 1950 (excluding lung cancer), rates have fallen 19%.
- Cancer causes are incorrectly attributed to sugar substitutes, bruises from being hit, microwave ovens, eating certain foods (pork / spicy foods), breast feeding, and antibiotics.
- Students believe that the immune system only plays a role when it is infected by external pathogens.
- Students believe there is a magic bullet to curing cancer, not being aware that there are 220 different cell types all with the potential for developing unique cancer cells.
- There also is the belief that if you keep yourself healthy, you won't get cancer. Discomfort arises when students are presented with the idea that cancer can happen by chance.
- Areas of the body that show higher cellular division do not necessarily show higher cancer rates. Example, brain tumors are more common than small intestine cancer, yet intestinal cells divide at a higher rate.

Pre/Post Lab Questions and Answers.

1. How do cancer cells arise? Cancer cells are the product of normal cells who have accumulated a series of mutations (in our example 4). Factors that can influence such mutation developments include environmental exposure to carcinogens to normal transcription errors.

2. How many times does a normal cell divide? ~50

3. What is the relationship between the immune system and cancer?
The immune system is responsible for monitoring cancerous cells. When a cell is deemed cancerous, CTL's (cytotoxic lymphocyte) seek and destroy them.

4. What is an example of a cytotoxic lymphocyte? T-Cells

5. What is a telomere? Segments of DNA at the end of the chromosome. These shorten with each cellular division, to the point where unless elongated, cellular apoptosis is the result.

6. What is the difference between benign and malignant? Benign tumors are non-invasive which means they do not infiltrate surrounding tissues. They may push surrounding tissues aside but do not invade. Malignant tumors damage surround tissues through invasion and spread to other parts of the body.

7. What does it mean if a cell has metastasized? The spread of cancer to another organ/tissue in the body that is non-adjacent. Usually the result of a cancer cell that has used the lymph or blood vessels as a means by which to travel.

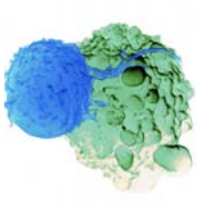
8. How are cancer cells recognized by the immune system? Cancer cells will present and abnormal number of antigens (little protein flags) to immune system cells, CTL's, for recognition.

9. What are the three E's? Elimination – more cancer cells are being killed by the immune system than arise. Equilibrium – equal number of cancer cells are being killed as arise. Escape – more cancer cells arise than can the immune system eliminate.

Interactive computer simulations for teaching tumor immunology

Student Section

Name:_____Date:_____Class:_____



Cancer: One Step At A Time

Background:

The body is made of roughly 100 million million cells. That's a lot of cells! If we had to build you from scratch making one thousand cells every second, it would take you 3171 years to make you. The job is not done at that point. You have to make new and replacement cells every day just to keep up maintaining your body. How many cells? You need to replace about 300 billion every day. That's 12.5 billion cells every hour for the rest of your life.

Imagine keeping all of these cells organized and doing their intended jobs. In order to do that, every new cell must receive a good copy of DNA from its parent cell which tells it what to be and how to do its job. These instructions are written in the language of DNA and packed into every cell. Six feet of instruction compressed into 1/5000th of an inch.

How much DNA do you make in your lifetime? If you lived 80 years you make enough DNA in your life to stretch to the Sun and back 56,560 times. In the process of making that much DNA, mistakes are inevitably made. Imagine having to copy a textbook with 3 billion letters without making a mistake. Surprisingly, your body does a good job of this. However, the copying process isn't perfect. In addition to making copying mistakes, most of us expose ourselves to cancer causing agents (carcinogens) that also change the DNA.

Cancer is a multi-step process that involves the changing of DNA in critically important locations. Each mutation by itself isn't enough to create a cancerous cell, however if you compound mutations, a cancerous cell can result. One sequence we'll study in detail is provided.

If the body is compromised of cancerous cells, it will respond by invoking a host of white blood cells like CTL's – T cells, NK cells. These CTL's look for antigen presenting cells. They locate both those cells that produce high amounts of antigen and also those cells that produce low amounts of antigen. If the immune system works, the cells will be eliminated. If the rate of cancer cell production is zero, the immune system is in a state of equilibrium. If the cells proliferate despite the immune system response, it is called escape. The conditions described above are known by immunologists as the **Three E's: Elimination, Equilibrium, and Escape**.

Name:_____Date:_____Class:_____

Mutation #1. **Loss of Cell Cycle Inhibitor** – A mutation in this gene causes a cell to ignore its instructions and stop growing. It takes away a cell's sensitivity to anti-growth factors. A good way to think of this is to picture a car with no brakes.

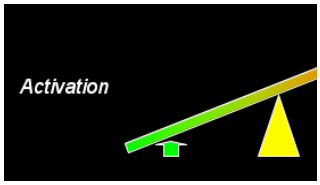
Mutation #2. **Self-sufficient Growth Oncogene** – Cells have genes that control normal growth rates. If this gene is mutated the cell will grow at an accelerated rate. A good way to think of this mutation is if someone puts a weight on the accelerator of a car allowing the car to speed out of control.

Mutation #3. **Activation of Telomerase Gene** – Normal cells will undergo approximately 50 divisions (+/-10) before dying. This is known as the Hayflick limit. One of the contributing reasons for the limit is that segments of DNA at the end of the chromosome, known as a telomere, begin to shorten every time you make a copy of a cell. It is almost like telling the age of a cell. The more copies that have been made of one cell, the 'older' the cell becomes. With this mutation, the telomeres (caps) are added back onto the chromosomes' end delaying further cellular death. Just like a gas tank in a car, every gallon of gas you take out of the tank, the closer it is to not running. This mutation is the equivalent to always keeping the tank full even though the car is burning gas.

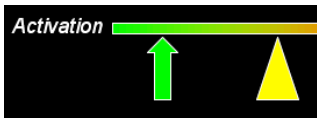
Mutation #4. **Inactivation of Apoptosis Pathway** – There are genes that control when a cell will die (cell death - apoptosis). The final mutation can promote the resistance to apoptosis by "knocking out" a gene that promotes cellular death. (ex. Papillomavirus gene interfering with the p53 protein critical to cell apoptosis)

The Immune Response: The Three E's.

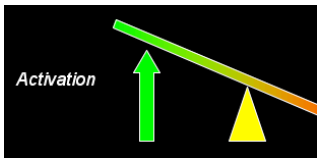
The immune system will "inhibit" the "activation" of cancer cells in the ELIMINATION phase.



When the immune system kills an old cancer cell for every new one, this phase is called EQUILIBRIUM.



When the immune system cannot eliminate cancer cells fast enough, the cancer is said to have ESCAPED immune surveillance.



Name:_____Date:_____Class:_____

Vocabulary: **(Resources to use – textbook, dictionary.com, google)**

Angiogenesis: _____

Antigen: _____

Apoptosis: _____

B-Cell: _____

Benign: _____

Cell Cycle: _____

Chemotherapy: _____

Elimination: _____

Equilibrium: _____

Escape: _____

Macrophage: _____

Malignant: _____

Metastatize: _____

Nascent: _____

Oncogene: _____

Pathogen: _____

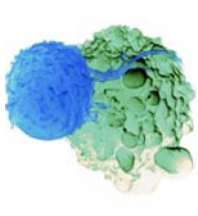
Surface Proteins: _____

T-Cell: _____

Telomerase: _____

Tumor: _____

Name:_____Date:_____Class:_____



Cancer Questions

"Class Discussion: What do you know?"

Introduction:

Want your "humors" restored, like the ability to sneeze? Then go see a barber. Up until the 19th century physicians based much of their views on the "humoral theory" which states that everything in the universe is based on the four elements: earth, water, fire, and air. Feeling crazy? Go see a barber, let that blood out. Want to get a musical voice? You know who to see…snip,snip. You may even have a hole drilled in your head to let your evil thoughts out.

There are many ideas around the topic of cancer that are true and false. How do you get it? Who can get it? Where does it come from and why? How do you treat it?

☛In the chart below, classify your ideas regarding cancer.

Fill in the following:

What You Know: Facts, irrefutable, you would fight to the death to defend!
Not Sure?: These are ideas that you've heard, but wouldn't put money on it.
Want To Know: Ideas you'd like to explore further.

Known	Not Sure?	Want to Know

1. Do you believe there will ever be a cure for cancer? Explain.

_____
_____
_____

2. Do you have any experience with cancer in your family/friends? Explain.

_____
_____
_____

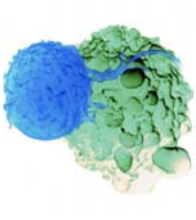
3. 100 years from now, how do you believe physicians will view current treatments for cancer? (Surgery, Radiation, Chemotherapy…) Explain.

_____
_____
_____

4. Based on your discussion with the class, write a one paragraph description of what cancer is. Your description should be written as if you are explaining it to a 1st grader.

_____
_____
_____
_____
_____

Name:_____Date:_____Class:_____



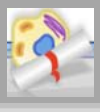
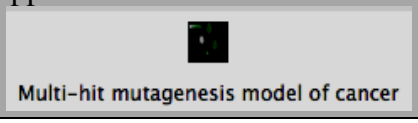
Cancer: Lab Exercise 1

"Get To Know the Model"

Introduction:

In this exercise you'll become familiar with EduClient as well as with some of the terminology used in the exercises. You'll be asked to focus on five groups of cells, each of which have acquired mutations. As you work with EduClient, pay close attention to the differences and similarities amongst the cells.

To begin this exercise…

- Open the EduClient Application 
- Select the recording: 

☛ *Before you press play on the recording*

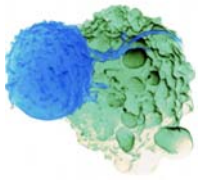
1. *Click on* --- Default Resource --- *in the upper left hand corner*
2. *Click* Cancel *from the resource menu after you've answered question set 1*

Question Set #1	
1.	How many resource options (Ways to track cells) do you see? _____
2.	How many mutants are listed? _____
3.	What does wild type mean? _____
4.	What does apoptosis mean? _____

In the lower corners of the screen you'll see the controls to run the simulation. Use your mouse to scroll over each and write their function.

Question Set #2	Controls	Function
	1.	
	2.	
	3.	
	4.	
	5.	
	6.	

Name:_____Date:_____Class:_____



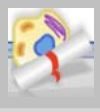
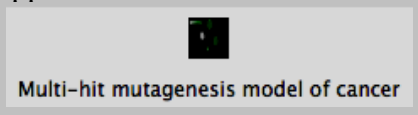
Cancer: Lab Exercise 2

"Wild Type and Mutants"

Introduction:

Cells will develop mutations over time depending on a number of influences both internal and external. Some of these influences include cancer-causing agents like UV light, cigarette smoke, radon gas…etc. These mutations prove most dangerous when they're partnered with other mutations. It is the accumulation of these mutations over time in cells that generate cancerous tumors. This exercise is designed for you to explore five clusters of cells and discover their differences and similarities.

To begin this exercise…

- Open the EduClient Application 
- Select the recording: 

This computer exercise starts at the very beginning with five cells at Step 0.

Click on  *and advance the simulation to Step 5. Each step is like a moment forward in time.*

Name	*Mutation(s)*
Mutant 1	*Mutation 1*
Mutant 2	*Mutations 1 and 2*
Mutant 3	*Mutations 1, 2, 3*
Mutant 4	*Mutations 1, 2, 3, 4*

Question Set #3

1. Describe the location of Mutant 1:
   _____
2. Describe the location of Mutant 2:
   _____
3. Describe the location of Mutant 3:
   _____
4. Describe the location of Mutant 4:
   _____
5. Make a prediction as to which mutant cluster will produce the greatest number of cells if allowed, Explain:
   _____
   _____
6. Was your hypothesis confirmed? _____

Name:_____Date:_____Class:_____

Cancer: Lab Exercise 2

"Wild Type and Mutants contd."

☛ *Under the progress bar is a description of the cellular conditions/progress that the cells are experiencing.*

Click on ▶ *and advance the simulation. Record observations in the table below.*

Click the ❚❚ button to properly record your descriptions/observations when the simulation is running.

Table #1

Cellular Conditions (Description)	**Simulation Steps (Ex: Step 5 to step 53)**	*Observations of Mutants 1,2,3,4, Wild-Type* **(Record what you observed)**
_____ _____ _____ _____	Start: _____ Stop: _____	WT. _____ 1. _____ 2. _____ 3. _____ 4. _____
_____ _____ _____ _____	Start: _____ Stop: _____	WT. _____ 1. _____ 2. _____ 3. _____ 4. _____
_____ _____ _____ _____	Start: _____ Stop: _____	WT. _____ 1. _____ 2. _____ 3. _____ 4. _____

Name:_____Date:_____Class:_____

Cancer: Lab Exercise 3

"Observing the Resources"

☛ *In the resources menu, choose the topic from the Question set under the ---Default Resource---*

--- Default Resource ---

Click on ▶ *and advance the simulation. Record observations in the table below.*

Click the ⏸ button to properly record your descriptions/observations when the simulation is running.

Question Set #4 – Cell Cycle Progression

1. What did the 5 clusters of cells look like when you started the simulation? (Color / Size / Activity Level?)

   _____
   _____
   _____

2. What did the cells changing color indicate?

   _____

3. When did each cluster stop changing color and what does that mean if it does stop changing color?

   _____
   _____

4. Do you believe these clusters are in competition with each other? If so, what would they be competing over?

   _____
   _____

Question Set #5 – Apoptosis

1. Which cell cluster had the highest rate of apoptosis? How could you tell?

   _____
   _____

2. Provide reasons why one cluster may have lived longer than the other clusters? (Why did some have advantages over others?)

   _____
   _____

3. Why might it be beneficial for the overall organism to have cells that are 'pre-coded' to die?

   _____
   _____

Name:_____Date:_____Class:_____

Cancer: Lab Exercise 4

"Cancer Has Natural Enemies"

Introduction:

All cells including cancer cells have proteins on their surface (cell membrane). The surface proteins indicate to neighboring cells whether they are friend / foe. Both MHC 1 and pSrc class molecules serve as the "peptide flags" on a cell's surface. If there are too many peptides on the surface of a cell, the antigen receptors on the CTL (cytotoxic lymphocyte / T cells – type of white blood cell) will bind with the cell and promote cellular death. If there are no flags, a different kind of CTL called the NK (natural killer) cell will kill the cancer cell. Either way, the body is in a constant predator prey relationship where bad cells arise and need to be controlled.

☛ *In the resources menu, choose the topic from the Question set under the ---Default Resource---*

Click on  *and advance the simulation. Record observations in the table below.*

Click the  button to properly record your descriptions/observations when the simulation is running.

Question Set #6 – pSrc*/MHC1	1. How can you tell whether some cells are showing more peptides than the others? _____ 2. If a cell is presenting a high number of antigens (MHC1, pSrc), which type of cell responds to eliminate it? _____ 3. Run the simulation and describe what happens to the cell numbers as the simulation runs? Be descriptive. (Color, Size, Activity Level) _____ _____ 4. How many cells existed at the beginning of the simulation? _____ 5. How many cells existed in the middle of the simulation? _____ 6. What happened to the cell numbers towards the end of simulation? _____ 7. Does this model represent Elimination, Escape, or Equilibrium? Explain. _____ _____ _____

Name:_____Date:_____Class:_____

Cancer: Lab Exercise 4

"Cancer Has Natural Enemies"

☛ *In the resources menu, choose the topic from the Question set under the ---Default Resource---*

--- Default Resource ---

Click on ▶ *and advance the simulation. Record observations in the table below.*

Click the ❚❚ button to properly record your descriptions/observations when the simulation is running.

Question Set #7 – pSrc*/MHC1

1. Describe what you saw as you ran the simulation.
   _____
2. At what stage(s) did you see a major number change?
   _____
3. Propose a reason (mechanism) as to why cells who all reside in the same cluster show differences in their surface protein levels and survival rates?
   _____
   _____
   _____
4. Why within a cluster of cells can you get such different results of cellular survival?
   _____
   _____
   _____
5. What are the ultimate consequences on those who study immunological therapies if cancer cells present antigens differently?
   _____
   _____
   _____
6. Does this model represent Elimination, Escape, or Equilibrium? Explain.
   _____
   _____
   _____

Comparing Plant and Animal Cells **Name:** _____

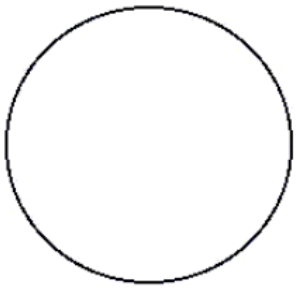
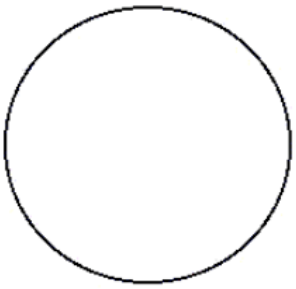
Problem: How are plant and animal cell alike? How are they different from one another?

Introduction: In this lab you will survey various cells from both animals and plants: cheek cells (animal), elodea (plant) – an aquatic plant.

Exercise 1. Cheek Cells

Procedure:

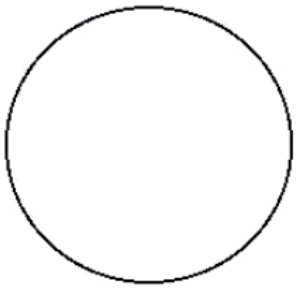
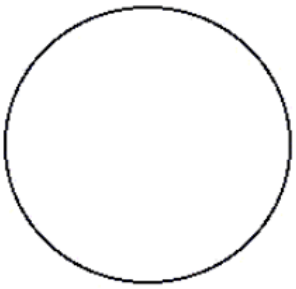
Using the flat end of a toothpick, gently rub the inside of your cheek being careful not to scrape too hard. Now rub the flat side of the toothpick, that has your cells on it, onto a glass slide. Place a drop of methylene blue on these cells for approximately on minute allowing the stain to affix to your cheek cells. Apply a cover slip then rinse off any excess stain and press your slide between multiple layers of bibulous paper.

Low Power  High Power 	1. Sketch your cheek cells under high and low power. 2. Using an arrows, identify the nucleus, cell (plasma) membrane, and cytoplasm.

Exercise 2. Elodea

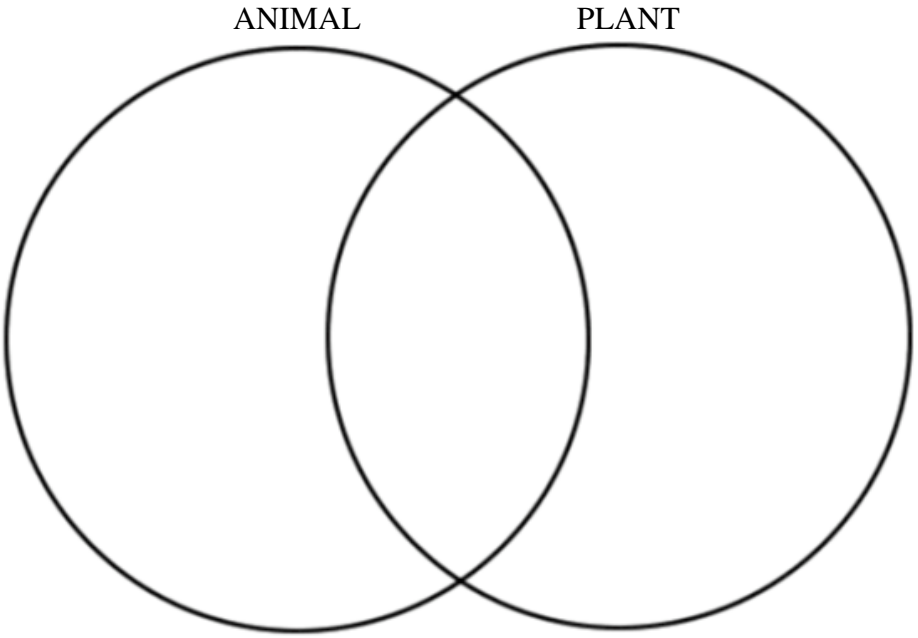
Procedure:

Extract a small leaflet from the Elodea plant and place it on a clean slide. Use an eye dropper and place a drop of water on the leaf. Cover the leaf with a cover slip and observe on both low and high powers.

Low Power  High Power 	1. Sketch the elodea specimen under low and high power. 2. Using arrows, identify the nucleus, cell wall, chloroplasts, central vacuole, and cytoplasm.

Exercise 3. Venn Diagram

Procedure: In the circle on the left labeled "ANIMAL", make a list of only those structures that are unique to animal cells. In the circle on the right labeled "PLANT", make a list of only those structures that are unique to plant cells.. Lastly, in the middle where the circles meet, make a list of those structures that both animals and plants share in common.



Name:_____Date:_____Class:_____

Cancer: Pre-Quiz

1. How do cancer cells arise?

_____
_____
_____

2. How many times does a normal cell divide? _____

3. What is the relationship between the immune system and cancer?

_____
_____
_____

4. What is an example of a cytotoxic lymphocyte? _____

5. What is a telomere? _____

6. What is the difference between benign and malignant?

_____
_____
_____

7. What does it mean if a cell has metastasized?

_____
_____
_____

8. How are cancer cells recognized by the immune system?

_____
_____
_____

9. What are the three E's?

_____
_____
_____

Name:_____Date:_____Class:_____

Cancer: Post-Quiz

1. How do cancer cells arise?

_____
_____
_____

2. How many times does a normal cell divide? _____

3. What is the relationship between the immune system and cancer?

_____
_____
_____

4. What is an example of a cytotoxic lymphocyte? _____

5. What is a telomere? _____

6. What is the difference between benign and malignant?

_____
_____
_____

7. What does it mean if a cell has metastasized?

_____
_____
_____

8. How are cancer cells recognized by the immune system?

_____
_____
_____

9. What are the three E's?

_____
_____
_____