

Lab 7: UV light, Melanoma, Mitosis

Name _____

Date _____

Learning Objectives:

1. Understand the relationship between wavelength and energy of light.
2. State the three types of ultraviolet light, their wavelengths, and their effects on skin cells if any.
3. Describe the two types of ingredients found in sunscreens.
4. Define SPF and use it to calculate how long someone can theoretically stay in the sun without getting a sunburn.
5. Define melanin, melanocytes, and melanoma.
6. Describe interphase and the stages of the mitotic phase.
7. Recognize the stages of mitosis and cytokinesis in both plants and animal cell using a microscope and cell models.

Pre-Lab Activity:

Prior to this lab, read the case study and answer the questions appropriately.

Case Study: Not An Old Person's Disease – Soaking up the Sun

PART I

Judy sat up, took a swig from her water bottle, and wiped the sweat off of her face. She glanced at her watch and frowned. Had they only been out here for half an hour? Man, trying to get a good tan was way too much work. She glanced over at Mariah who was sleeping peacefully in her lounge chair. No wonder she looked like a Greek goddess every summer—she enjoyed lying in the sun. Judy decided that her normally pasty-white skin was tan enough, getting a little red again in fact, and she started to gather her things. Anyway, she reasoned, a dark tan might clash with her red hair.

Judy stopped for a second to scratch her calf. It had been itching for several days now, and she figured that the mosquitoes had really gotten her at the barbecue the other night. Since she was on the ground, she bent her neck to look at it and was surprised to see that it wasn't a mosquito bite but a mole. She'd had this mole ever since she could remember, but it had never itched before. She looked a little more closely and noticed that the mole looked kind of different than she remembered. It was a little bigger and the edges were sort of jagged. One edge was a bit darker than the other and in the middle was a raised purplish-black dot that she tried to wipe away but couldn't. As she was staring at her leg, Mariah woke up.

"What are you day dreaming about, your hot date with Tim tomorrow night?" Mariah teased as she rolled on her side, her black hair falling into her eyes.

Judy shook her head, "Ha, ha, I do have more things in my life to ponder. Anyway, I just noticed that a mole on my leg looks kind of weird and it keeps itching."

Mariah sat up with a concerned look on her face. "Let me see it." As she looked at Judy's leg, she said quietly, "Maybe you should go to the clinic." As Judy stared at her, she continued. "My mom sent me a pamphlet on skin cancer last year. You know, trying to convince me to stop lying in the sun so much. I glanced through it before I tossed it into the garbage. I remember some of the pictures of skin cancer and they kind of looked like your mole."

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"What are you talking about?" Judy said defensively. "How could I have skin cancer? I'm barely in the sun compared to you."

"Alright, alright. It's not like I'm a doctor or something. I just remember those pictures. Come on, let's just call the clinic so they can check you out. I'm sure they'll tell you I'm crazy."

Questions

1. What are some differences between Judy and Mariah that might make Judy more "at risk" for skin cancer than Mariah?
2. What observations did Judy make concerning her mole?

PART II

Judy did a little research about skin cancer on the Internet before her doctor's appointment the next morning. She learned that most cases occurred in people who were significantly older than she was. It didn't make sense that it would happen to her—she was only 20 years old!

The articles talked about how UV light from the sun causes mutations in your DNA. Accumulation of DNA mutations over many years can cause certain cell cycle genes called "proto-oncogenes" to become super-active. Judy actually felt thankful for the college biology course she had been forced to take. She knew that DNA was the hereditary material that acted as a "blueprint" for everything our cells make and that a gene is a piece of DNA that contains the instructions for making a single protein. However, she would have to ask the doctor about these proto-oncogenes.

In the examining room, Dr. O'Brien was silent as he looked at the mole on her leg. Finally, he said kindly, "I want to do a biopsy. All that means is we'll remove your mole and look at the cells under a microscope and see if they look abnormal."

Judy could feel the tears welling up in her eyes. "You mean you can tell if I have a tumor by just looking at some cells?"

"Whoa, slow down a minute," Dr. O'Brien replied calmly. "It's very possible that your cells will look completely normal. And to clarify something, a tumor is not necessarily the same thing as cancer." She looked confused, so he continued. "A tumor means that cells have divided and piled up on one another in a single mass. But not all tumors are automatically cancerous and life threatening. A benign tumor is a mass of normal-looking cells. These tumors are not considered cancer and they're usually relatively easy to treat—we just remove them. On the other hand, a malignant tumor is a mass of abnormal cells whose growth cannot be controlled by the regular mechanisms. In addition, malignant tumors often spread to other parts of the body instead of just staying in one spot. We call this process metastasis. Malignant tumors are cancerous and, therefore, a very serious condition. To treat them, we have to remove the cancerous cells that we find and then do chemotherapy treatments to be sure to kill all the cancer cells that we may have missed surgically."

"So, if I have cancer, is it from these proto-oncogenes that I read about on the Internet?"

The doctor smiled and seemed pleased that Judy had been reading about this on her own. "Let's back up for a minute. You see, we have tens of thousands of genes in our cells, but it's not like mutations in just any of them will lead to cancer. The genes that get mutated and can cause cancer are of a specific type called cell cycle genes. Everyone has a set of cell cycle genes in each of their cells that code for cell cycle proteins. Cell cycle proteins control if and when cells divide. Sometimes we need to make more cells in our bodies, and some of these cell cycle proteins allow that normal process of cell division to occur. At other times we don't want the cells to divide, so different cell cycle proteins inhibit cell division then. In normal, healthy cells, the formation of the cell cycle proteins is tightly controlled so the activating proteins are only made when we really need more cells and the inhibitory proteins are only made when we don't need more cells. However, mutations (or alterations) in these genes can eliminate this tight regulation and lead to uncontrolled cell division. This is what happens in many types of cancer: a normal cellular process, cell division, is no longer properly controlled."

Judy thought for a second. "Okay, but you still didn't say what these proto-oncogenes are."

"Oh yes, sorry. The activating class of these cell cycle genes consists of the proto-oncogenes. The normal job of these genes is to code for proteins that promote cell division. Certain mutations in proto-oncogenes result in proteins that are active all the time, causing cells to divide continuously. But let's not get ahead of ourselves—we won't know if your mole is even cancerous until we do the biopsy."

When Judy didn't say anything in response right away, Dr. O'Brien continued, "I know this is all a bit confusing, but it's good that you're asking these questions. We'll make sure that you understand this a little better before you leave today."

Just then a nurse knocked and entered the room. He needed Dr. O'Brien for a minute. At least Judy would have a minute to gather her thoughts.

Questions

1. Considering the differences between a benign tumor and a malignant tumor, why might a benign tumor be easier to treat?
2. Judy learned that every single person has these cell cycle genes so cells in our body can divide when necessary. What are some normal circumstances where our bodies might need to make more cells?
3. Every person has these cell cycle proto-oncogenes, but not every person has cancer. Why might this be the case?

PART III

Dr. O'Brien had handed Judy a pamphlet on melanoma before he left the room. As she started reading it, Judy realized that this is what they were going to test her for. Melanoma is a type of skin cancer that starts in your melanocyte cells, or the pigment cells of your skin. Actually, a mole is just a clump of melanocytes.

There were pictures of cancerous moles in the pamphlet and one of them looked a little like hers, only bigger. She was starting to get worried.

Dr. O'Brien returned just then and apologized for the disruption. "Do you have any more questions?"

"Well, I just don't understand how it's possible for a 20-year-old to get cancer. Isn't cancer an old person's disease?"

"You're right that most cancer patients are older. That's because you accumulate mutations in your genes over time. UV light, cigarette smoke, other chemicals, and even aging itself can cause mutations in genes. The kind of cancer that we're checking your mole for is melanoma. This is the most common cancer in people aged 25 to 29 and is the fastest-growing category of cancer, increasing four percent each year. You are a bit young, but you may be genetically predisposed to melanoma."

"What does that mean?" Judy asked.

"Well, remember the cell cycle genes that we were talking about earlier? I described the proto-oncogenes as being needed for activating cell division. Well, remember that I also mentioned a class of cell cycle genes that prevent cell division. These genes are called tumor suppressors. You usually have two good copies of each of these tumor suppressor genes, but sometimes you inherit one good copy and one mutated copy. Basically it means that, along with inheriting genes for your red hair or blue eyes, you may have inherited a mutated cell cycle gene. You had it when you were born, so it's not the result of lying in the sun too much or anything like that. But for these types of genes to lead to cancer, you need to have both copies of the gene mutated. One good copy is sufficient to prevent your cells from dividing aberrantly. So you can be born with one mutated copy, but not actually get cancer unless the other copy accidentally gets mutated sometime during your life. Mutations in the second copy could be the result of UV light from the sun, like in the case of melanoma. Since you may have been born with one mutated copy already, you are "predisposed" to getting cancer. You are one step closer to getting cancer compared to someone who doesn't have a mutation. We see a genetic, or hereditary, component in about five to 10 percent of melanoma cases. Do you know if anyone in your family has had melanoma?"

Judy shook her head, "I'm pretty sure that my dad hasn't, but my parents split up when I was young and I haven't really talked to my mom in the last 10 years. I thought I heard that she had some mole thing removed a long time ago, but I didn't think that it was cancer."

The doctor said, "You might want to call her. Now, let's get this biopsy done and hopefully prove that all this talk about cancer is unnecessary."

Questions

1. Now that you know a little more, what are the risk factors that increase a person's chances of having melanoma?
2. How does sunlight contribute to the development of melanoma?
3. What does it mean to be predisposed to getting cancer? If you inherit a mutated cell cycle gene, does that automatically mean that you will get cancer some day? If you inherit a mutated cell cycle gene and participate in risky behaviors such as sunbathing, does that mean that you will automatically get cancer some day?

Introduction:

To see any object we need light and whenever we think about light the most powerful natural source we think about is sunlight. Sunlight is a form of **electromagnetic energy**. All electromagnetic energy, including light is made up of particles called **photons**, and these particles travel in **waves**. These waves of light vary in their wavelength and have different levels of energy. The energy associated with these waves is measured as the distance between two adjacent peaks or crests of a wave, and called the **wavelength**. The most commonly used unit for measuring wavelength is nanometers (nm) (recall the base unit what you have studied in the metric lab, $1 \text{ nm} = 10^{-9} \text{ m}$), but to be more accurate in quantum physics the measurement unit is angstrom which is 10^{-10} m . Among all the different ranges of wavelength only a narrow range could be detected by the human eyes and we call that spectrum **visible light**. Visible light is in the range between 400 – 700 nm. Other forms of light also exist on the electromagnetic spectrum but are not visible to humans. Light waves with wavelengths below 380 nm are called **ultraviolet rays** or (**UV**) rays and those with wavelengths more than 700 nm are called **infrared rays**. The lower the wavelength, the shorter the waves and the more powerful they are or have higher amounts of energy. Although humans are only able to see a very narrow band of the electromagnetic spectrum, over time scientists have discovered ways to use other parts of the energy spectrum to enhance our daily lives and augment the medical field. Figure 1 below shows the ranges of wavelengths and their relationship with the amount of energy they contain.

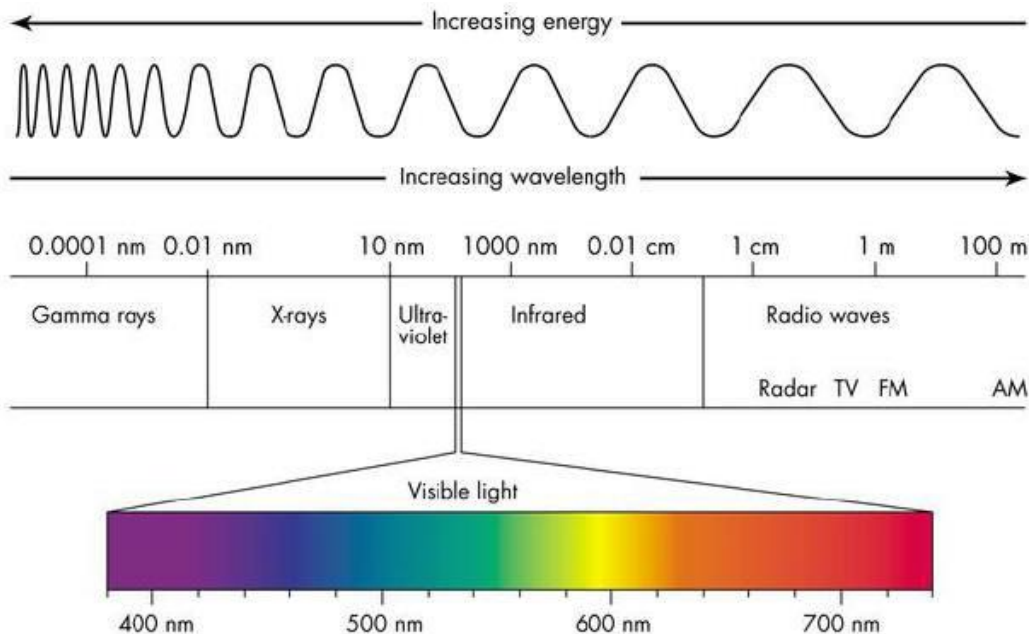


Figure 1. Light wavelengths and energy (lumenistics.com)

Among the waves that reach Earth's atmosphere about 95-96% are infra-red and visible light and only 3-5% are UV rays. UV rays are required by our body for vitamin D production and calcium homeostasis, but in excess they are harmful mutagens and could cause severe damage to skin which can cause cancer. There are three types of UV rays emitted by the Sun – UVA, UVB and UVC, and these are classified based on their wavelengths. UVA rays range between 320 – 400 nm and are the least harmful, UVB rays range between 290 – 320 nm, and the shortest UV rays are UVC, between 100 – 290 nm. UVC is the strongest (lowest wavelength) among the three types and therefore the most harmful. But the UVC rays are completely

absorbed by the ozone layer and the upper atmosphere and therefore these rays never reach ground level. UVB rays pose the next level of harm to humans, but 95% of UVB is also absorbed by the ozone layer. The small fraction of UVB rays that do penetrate the ozone and reach the Earth's surface are those which are mostly responsible for sunburn. UVB exposure of extended period of time is also known to cause wrinkles and pre-mature aging and now it is known that UVB can also cause skin cancer. UVA rays theoretically have the longest wavelengths and therefore the lowest energy, but since these rays are NOT blocked by the ozone, our skin is constantly exposed to these UV rays. Despite having longer wavelengths, UVA has a strong capacity of penetrating into our skin and can cause severe DNA damage leading to skin cancer or melanoma. Looking at our evolutionary history it has been discovered that over time humans produce increased amounts of a pigment, **melanin**, which acts as a natural sunscreen. Melanin is produced by a particular type of skin cells called **melanocyte** which shield the DNA in nucleus from the damaging effect of UV rays. This is why when we are exposed to sun we can get a tan after long exposure as our body will produce more melanin to shield our DNA from UV exposure. It is also established now that there is a direct link between skin damage caused by overexposure to UVB and UVA rays and the development of skin cancer or melanoma.

We need UV light for our normal physiology, but we cannot allow it to cause potential damage to our skin and body. Therefore, it is highly encouraged to utilize protective measures when outside for prolonged periods of time. Covering of all skin surfaces is not always feasible, so instead alternate forms of protective coverings known as sunscreens are used. Sunscreens are categorized as either **physical** or **chemical** based on the ingredients contained to provide protection against the radiation from UV light. Physical ingredients tend to be **inorganic** in nature, meaning they do not contain carbon molecules and block and reflect the radiation coming from UV light. Chemical sunscreens, in comparison are often **organic** components (carbon-containing) that work to absorb UV radiation, resulting in dissipation of the energy and therefore less damage. Most sunscreens are a combination of these two to provide efficient protection against both UVA and UVB rays.

When you are outside, along with sunscreens, remember to wear sunglasses for eye protection and also wear light colored loose clothing covering your Body. You also need to understand how effective the sunscreen you will be using. The efficiency of any sunscreen is expressed as **SPF** or **Sun Protection Factor**. SPF is a measure of how long a person can remain in the sun without burning based on the particular sunscreen that has been applied. For example:

Dr. Davis normally starts to burn, without sunscreen, after 15 minutes of continuous sun exposure. Therefore if she applied a sunscreen of SPF 30, this would allow her to stay outdoors for 30 times longer. In order to figure out just how long she can stay in the sun, wearing SPF 30 without burning, we can do a simple calculation:

$$\begin{aligned}\text{Length of time until getting sunburn} &= (\text{minutes before burning}) \times (\text{sun protection factor}) \\ &= 15 \times 30 \\ &= 450 \text{ minutes then divide by 60 (minutes in hour)} = 7.5 \text{ hours}\end{aligned}$$

Note: Due to pollution and other factors the ozone layer is damaged by which we were getting exposed to more damaging UV rays.

Critical thinking:

Is it wise to use tanning salon to get a tan? Why might UVC be used as a disinfectant to kill microorganisms in food, air, etc. by means of UV lamps? Have you ever seen this in use?

Activity 1: UV Bead and Sunscreen Protocol

There are many ways to protect yourself from the sun. Today you can buy sunscreen as a stick, a spray, or a lotion. SPF of sunscreen can be found ranging from an SPF of 4 to an SPF of 100. Always check the expiration of your sunscreen as the chemicals can lose their efficiency over time. Hats, swimsuit cover-ups, and sunglasses are also recommended.

UV beads are beads that contain a unique pigment that changes color when exposed to UV light from the sun or other UV sources. This allows us to use the beads as UV indicators to test various forms of sun protection.



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Figure 2. UV Beads

Materials Needed

For a team of four students, you need:

- Sunscreens of varying SPF, brand, and/or mode of applying (e.g., stick, spray, lotion)
- Sunglasses
- 12 UV beads of the same color
- UV Lamp
- Twine
- Cotton swabs
- Paper, Tape, and Marker

Exercise

1. Create a hypothesis to test the ability of various sunscreen products to block UV light using UV indicators (UV colored beads on twine). Remember that the best hypotheses test only one variable.

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Variables can include the SPF of the sunscreen, spray versus lotion, different brands, or expired versus non-expired sunscreen.

2. List the variables that you will be testing.

- Independent variable:
- Dependent variable:
- Controlled variables:

3. Look at the available products provided on the instructor bench. You can select up to three products for your experiment. Record the products you selected in the table provided below. A positive control (no product) will be used for comparison allowing for full color change. Record your predicted results in the provided table 1.

Table 1. Predicted Effects of Sunscreen

Experimental Group	Product (Include brand, SPF, and whether it is lotion or spray)	Prediction
1	None (control)	
2		
3		
4		

Procedure

1. Use the UV light at the instructor bench to select 3 beads per experimental group of the same color (e.g., if you have 4 groups, you will need 12 beads of the same color). Cut a short piece of twine for each experimental group and 1 sheet of paper.
2. Thread 3 beads onto each piece of twine. Each piece of twine with its beads is now an experimental group. See instructor's demo.
3. Treat each experimental group with its appropriate product. For lotions, use a clean swab to coat all three beads evenly on the twine. For sprays, spray the three beads evenly with sunscreen **over the sink and try not to directly breathe in the chemicals as you spray.**
4. Tape each prepared experimental group including your control onto the sheet of paper and use a marker to label appropriately.
5. You instructor may request that you complete the experiment inside or outside.
 - a. For outside experiment: Place your paper with experimental groups in direct sunlight for 10 minutes.

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- b. For inside: Direct the UV lamp provided at your bench to direct UV light on your paper with your experimental groups for 5 minutes. You may have to do it in groups to ensure all groups get maximum amount of light.
6. Take a picture of your beads directly after removing them from the UV light as they change quickly back to the original color. Note any changes in color, paying special attention to changes in the intensity of the color and whether the beads become more or less opaque (i.e. how difficult it is to see the twine through the beads). Rate the intensity of the color change seen with each type of sun protection using a scale of 1 (no change) to 5 (intense color). Record your observations in the data table 2 below.

Table 2. Actual Results of Sunscreen

Experimental Group	Product (Include brand, SPF, and whether it is lotion or spray)	Intensity (Scale 1-5)
1	None (control)	5
2		
3		
4		

Questions

1. How do the results compare to your predictions? Do your results accept or reject your hypothesis? What factors may have influenced your results?
2. Which products seemed most effective?
3. If, without sunscreen, a person can stay in the sun 10 minutes without burning, how long can they stay in the sun with...
 - a. SPF 15 lotion protection?
 - b. SPF 40 lotion protection?
 - c. SPF 100 lotion protection?
4. For an SPF 100, would you trust it to protect for the amount of time you calculated?

Activity 2: Mitosis

We previously stated the UVB light can disrupt cell division (**mitosis**). Mitosis is the part of the cell cycle where a single cell divides its chromosomes and its cytosol to produce two identical daughter cells. It is

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used for growth, maintenance and repair of tissues in multicellular organisms. You will be observing interphase, and the stages of the mitotic phase: prophase, metaphase, anaphase, telophase and cytokinesis in both plant and animal cells. **Interphase** and the **Mitotic phase** make up one **cell cycle (Fig 2)**.

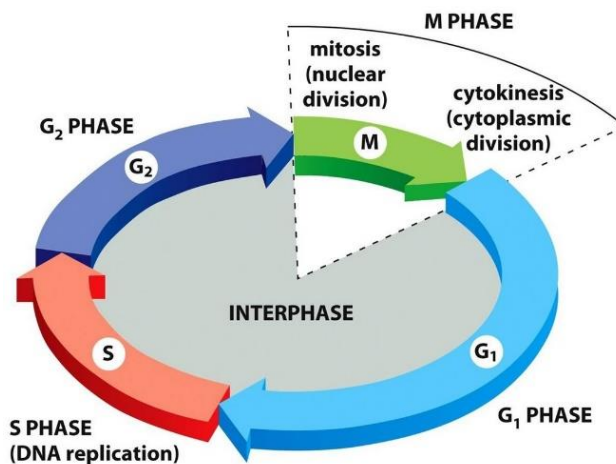


Figure 3. Cell cycle

Materials Needed

For a team of four students, you need:

- 2-4 microscopes
- Onion root tip slide
- White fish blastula slide
- Animal cell mitosis model
- Plant cell mitosis model

Exercise A - Viewing Plant Mitosis under the Microscope

For plant mitosis use the slide in your slide box labeled **Onion** or **Allium root tip** that contains 3 cross sections.

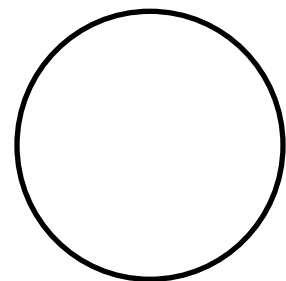
Procedure

1. Using the low power of your microscope examine one of the root tips noting the number of cells that have darkly stained filamentous chromosomes.
2. Now increase the magnification up to 40X and using the mechanical stage move the slide to find each of the mitotic stages described below. You may need to look at all 3 root tips to find all of the stages.
3. Draw an example of what you see under the microscope for each phase in the provided circles.

Phases and Important Structures to Identify:

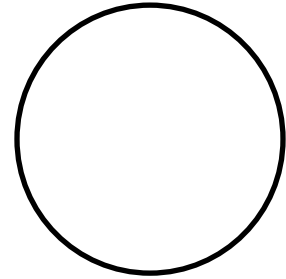
- **Interphase**

The non-dividing stage of cell cycle. During this time the **nucleus** is visible. The individual chromosomes are not visible during this stage but are replicated in S phase. This replication produces a pair of identical chromatids held together by a centromere.



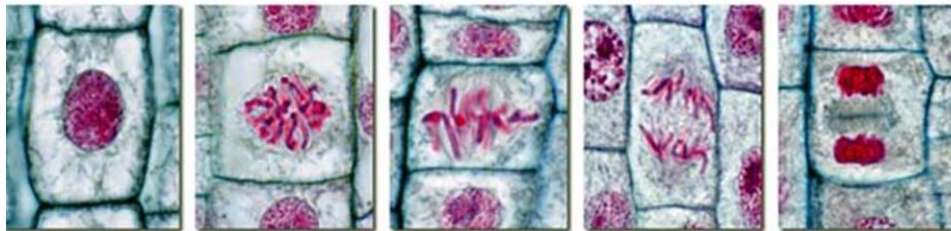
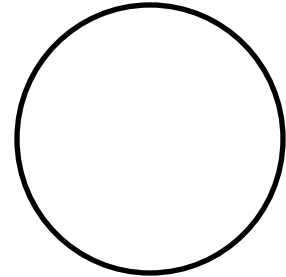
- **Prophase**

During this stage the nuclear envelope will be disassembled. The chromosomes, now containing two chromatids joined by a centromere, become visible and appear as dark linear structures. The spindle begins to form. You should be able to see the **chromosomes** but neither the chromatids nor the centromere will be visible at this magnification.



- **Metaphase**

The spindle apparatus is completely formed and spindle fibers have attached to each chromosome at the centromere. This causes the chromosomes to become aligned on the equator. You should be able to see the spindle fibers. Make sure you identify the **chromosomes** and the **equator**.

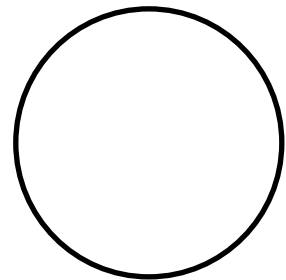


Interphase Prophase Metaphase Anaphase Telophase and Cytokinesis

Figure 3. Cell division in plant cells.

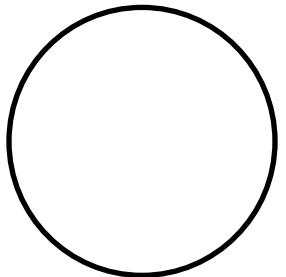
- **Anaphase**

The centromere divides allowing the sister chromatids to begin to move apart. At this point since the chromatids have separated and each has its own centromere they are each called a chromosome. The chromosomes have begun to move to their respective poles. Be sure to identify the chromosomes and the **spindle**.



- **Telophase**

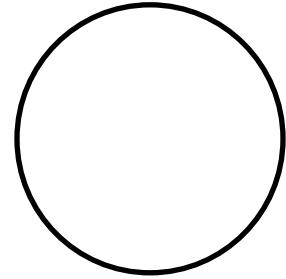
Once the chromosomes have reached their respective poles they will uncoil and gradually become less visible. The nuclear envelope will be reformed around each complete set of uncoiling chromosomes. The nucleolus will once again become visible. Look for a cell in which the chromosomes have arrived at their respective poles.



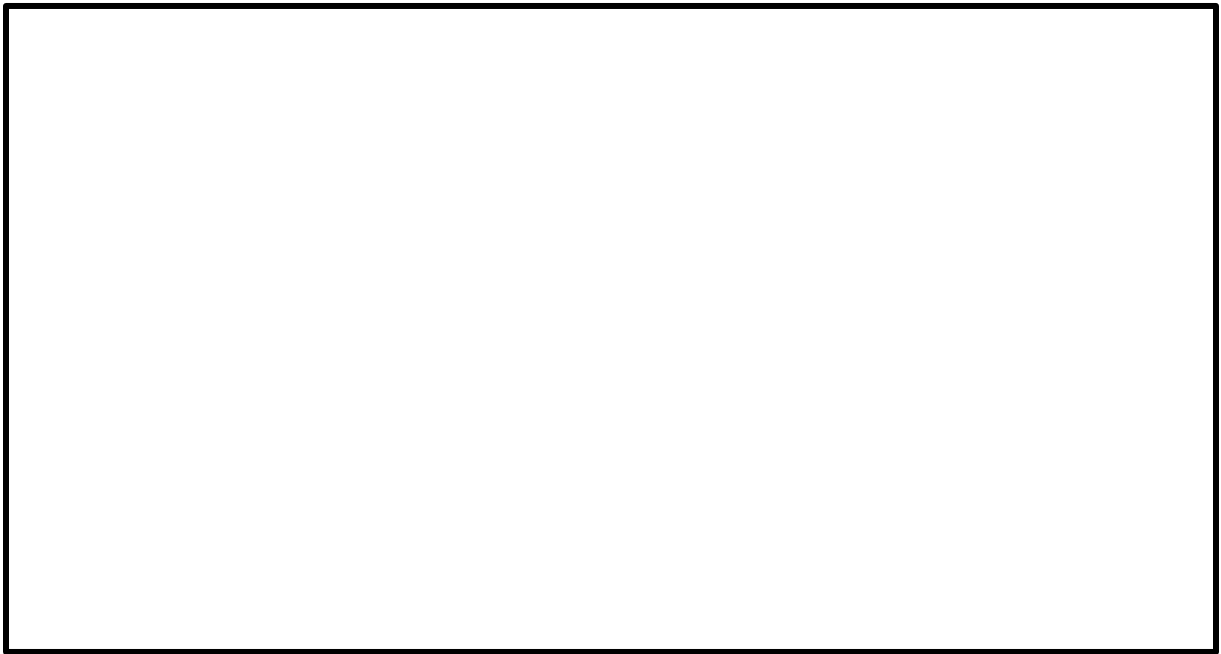
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- **Cytokinesis**

During telophase a **cell plate** will form from the center of the equator outward separating the cytoplasm of the two new daughter cells. When the cell plate has completely divided the two daughter cells they are considered to be in interphase. Look for cells in late telophase where the cell plate should be visible and the chromosomes are being reorganized into a nucleus.



4. Identify the parts and phases of mitosis using the plant cell models displayed in the front of the classroom. Draw or paste a photo of the models below.



Exercise B - Viewing Animal Mitosis under the Microscope

For animal cell mitosis use the slide in your slide box labeled **white fish blastula** that contains several cross sections.

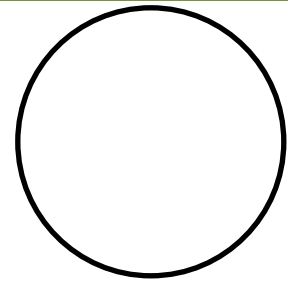
Procedure

1. Place the *whitefish blastula* slide under low power and locate a group of dividing cells.
2. Focus it on high power and notice that the cells are less ordered than you saw in the onion root tip. This may allow you to view mitosis from several angles causing some stages to look atypical. Look for cells that are more typical in appearance.
3. Draw an example of what you see under the microscope for each phase in the provided circles.

Phases and Important Structures:

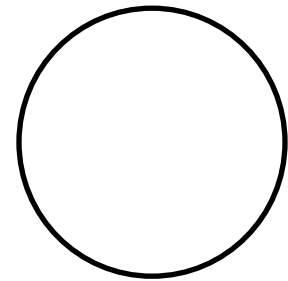
- **Interphase**

This stage will be similar to what you saw in the plant cell but there is no cell wall present.

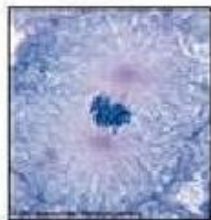


- **Prophase**

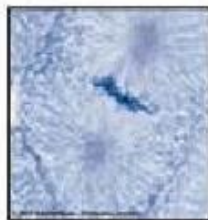
The nuclear membrane disassembles and the chromosomes become visible as thread like structures. You will notice that the chromosomes are smaller and more numerous than in the plant cell. Two cytoplasmic structures not found in the plant cell called centrioles have migrated to opposite poles and are beginning to align the microtubules of the spindle. While each centriole is not visible the microtubules migrating out from it will be visible and have a star like appearance. Make sure you locate the **chromosomes** and the **spindle fibers**.



Interphase



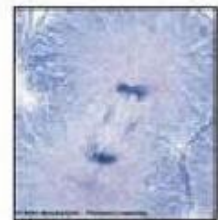
Prophase



Metaphase



Anaphase

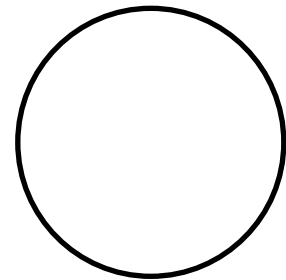


Telophase and Cytokinesis

Figure 4. Cell division in animal cells.

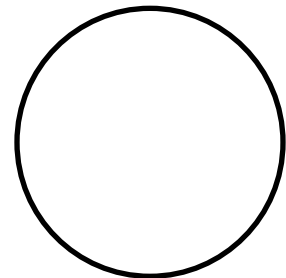
- **Metaphase**

Events in this stage are very similar to the onion root tip. One difference you might note is that the spindle and its individual fibers are easier to see. Be sure to identify the **aster**, **spindle fibers**, **chromosomes** and **equator**.



- **Anaphase**

Events here are like in the onion root tip.



- **Telophase and cytokinesis**

Unlike the onion root tip no cell plate forms during cytokinesis. Instead the plasma membrane appears to pinch in on either side of the equator forming a V like structure called the **cleavage furrow** (Figure 5). This will continue to deepen until it divides the cell into half to form two identical daughter cells. Each daughter cell is about half the size of the parent cell.

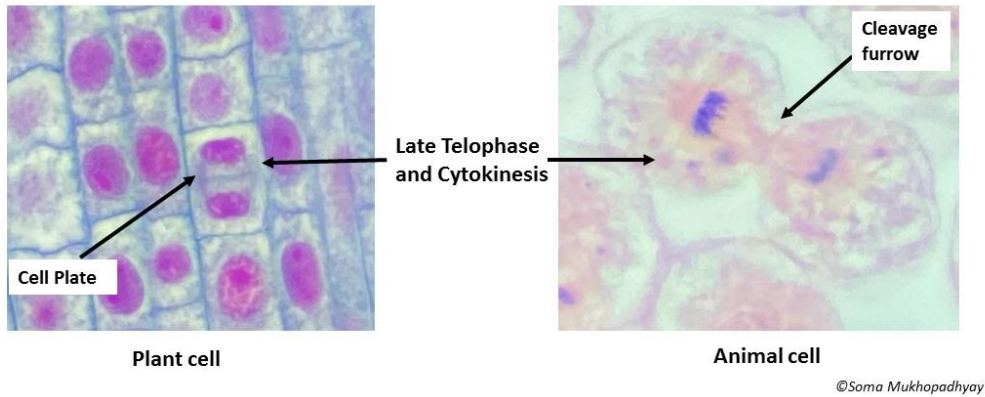
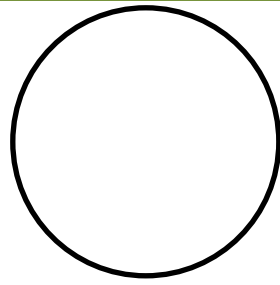
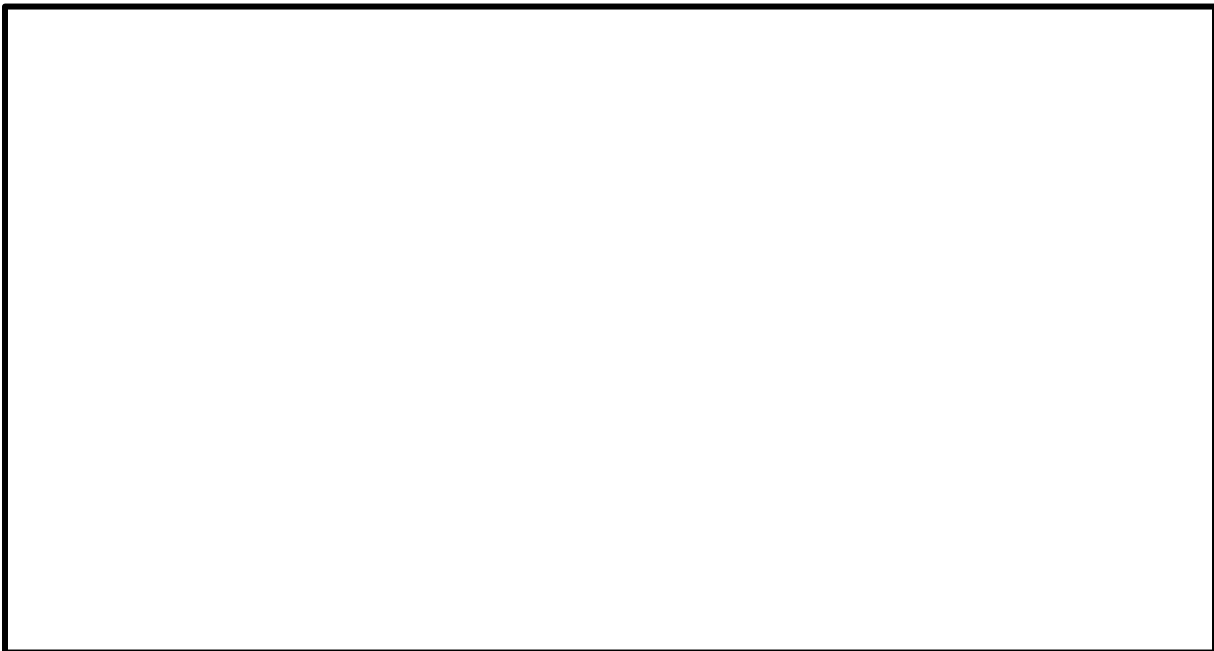


Figure 5. Cytokinesis in plant and animal cells.

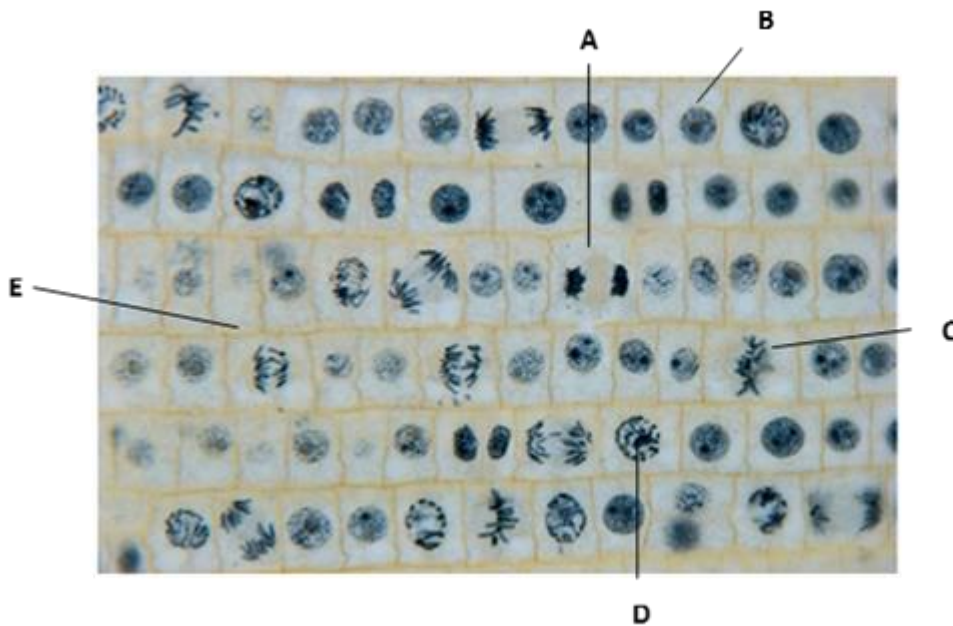
4. Identify the parts and phases of mitosis using the animal cell models displayed in the front of the classroom. Draw or paste a picture of the models below.



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Questions

1. Which stage(s) of cell division vary between plants and animals? Explain the difference(s).
2. What happens to cells when division “runs amok”?
3. Identify the phase of the cell cycle and mitosis for each cell identified.



Reference:

Kim Spaccarotella, UV light Lab, Kean University

Jennifer Miskowski and Anne Galbraith, Case study. University of Wisconsin—La Crosse

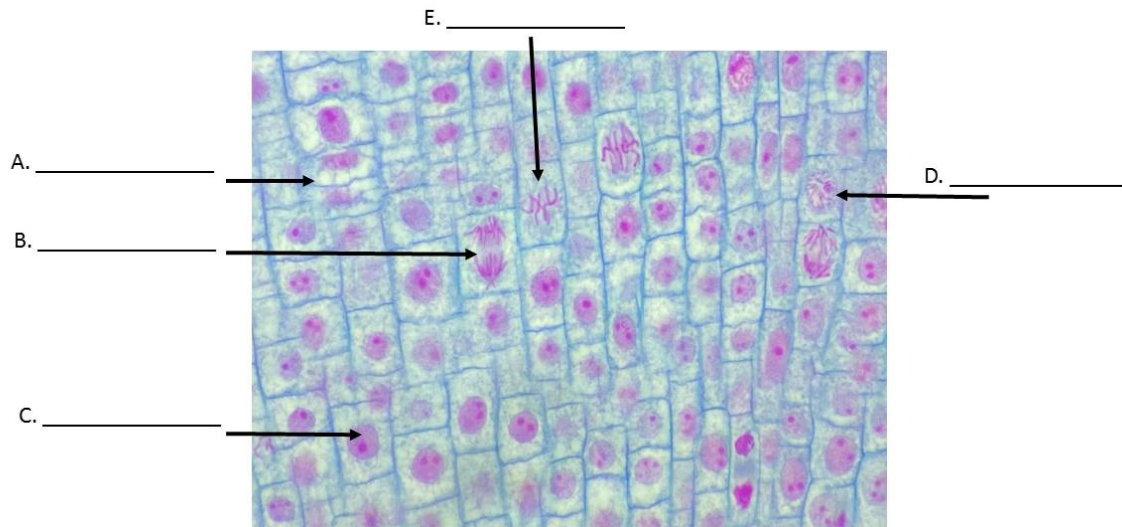
Post-Lab Activity:

Name _____

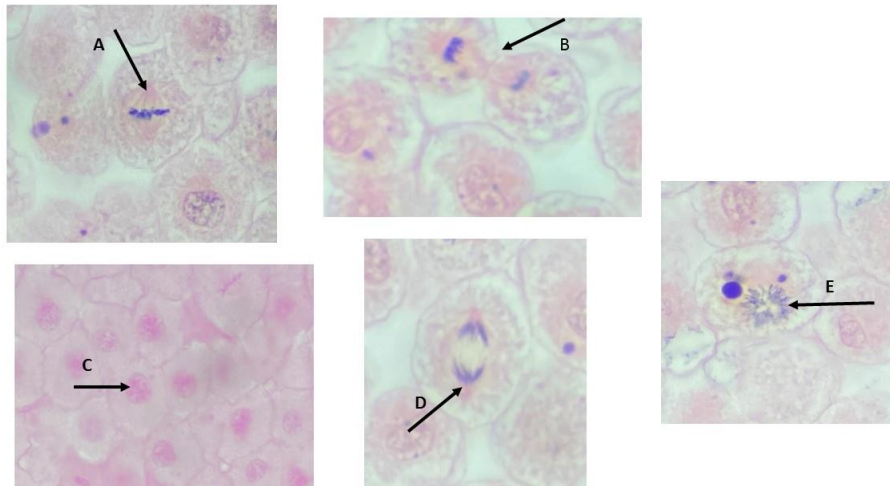
Complete the following.

1. Identify the stages of cell division at the tip of the pointer.

a. Is this a plant cell or animal? _____

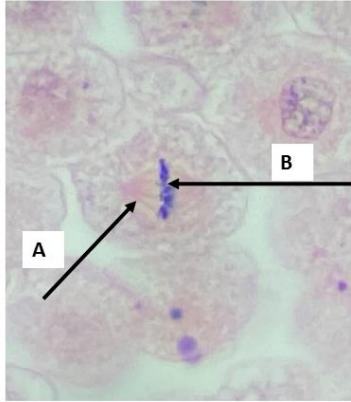


2. Arrange the stages of cell division in right order of sequence. _____.



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3. Identify the following structures: A _____, B _____
a. Is this a animal cell or plant cell _____



4. a) Identify the following structure at the tip of the pointer.
b) Is this a animal cell or plant cell _____

