Main Question: How can the environment and genetic change impact the chances of getting cancer?

Problem: People from all over the world get cancer and suffer from it. Cancer is caused through various ways starting from different parts of your body and the organs in it. Can the environment and genetics affect your chance of getting cancer if so, then how?

Date: October 18 2024 Time: 9:30-9:52

1. How is cancer usually caused, how does it develop?

2. How/Can cancer get passed down through genetics?

3. How do others think cancer is caused?

4. What are Tumors? Are they cancerous?

Date: October 18 2024 Time: 3:30-3:41

Background Research

How is cancer usually caused?

Cancer can be caused in multiple ways, however the most common way cancer is caused is by genetic alteration. This already partially answers one of my questions but how does it go through generations? Another way cancer can simply be caused is by mutations towards DNA, within cells. Lastly, one other way I found out cancer is caused by not taking management over yourself and doing things like smoking which is how most people think cancer is caused including myself.

Date: October 20 2024 Time: 5:30- 5:57

Nucleus

Inside nearly every single cell in your body there is a structure called the nucleus. Inside the nucleus there are 23 pairs of chromosomes that make up genes. Genes tell cells what to do and how to behave. We each have about 20,000 genes or chromosomes. Largest to smallest: cells come first, then nucleus, chromosomes are in there, genes are inside chromosomes and lastly DNA is in genes.

Date: October 23 2024 Time: 7:00- 7:27

How does cancer develop?

Cancers develop because something has gone wrong with one or more genes in a cell. It could be that the cell has grown old or became damaged. Those changes kills the cell which makes the cell stop functioning completely and new cells usually take their place. Sometimes that process breaks down and damages the cell's growth, they begin to multiply when they shouldn't. Those cells can form tumors. Tumors are abnormal growth of body tissue. Tumors can be cancerous but also not cancerous. So IF it does become cancerous, it will start to spread and grow uncontrollably.

Date: October 27 2024 Time: 3:03- 3:35

How is cancer passed down through genetics?

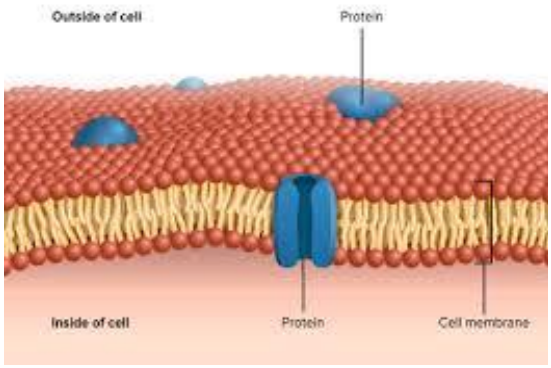
Just because a couple of your ancestors have been diagnosed with cancer, whether it is the past or present. It doesn't mean there will be gene changes to everyone in your family. It depends on multiple different aspects.

1. Who was the person in your family that had cancer
2. The types of cancer they had
3. How old they were when they got cancer
4. How closely you are related to that specific person

Date: October 31 2024 Time: 5:32- 5:43

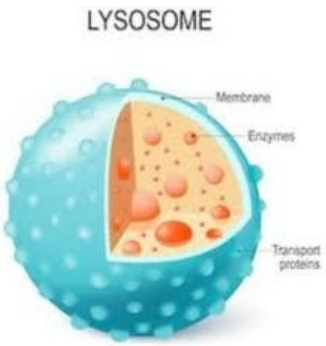
What Cells Are Made Up Of

Mitochondria: The Mitochondria is an organelle that generates energy. It is sometimes known as the “Powerhouse of the cell.”

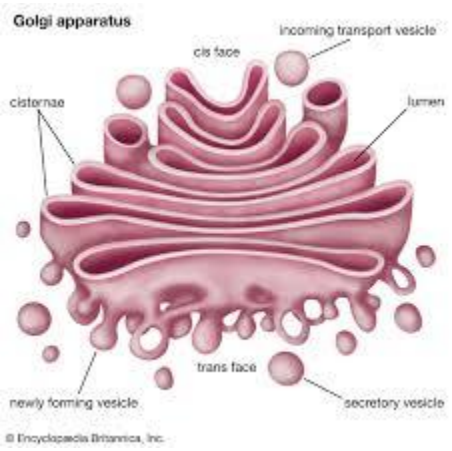


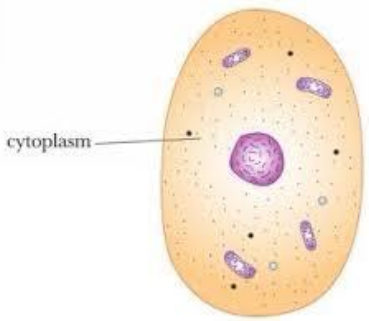
Cell membrane: It surrounds the cell providing protection from bad bacterias, fungi’s and more from going into the cell. It is a semi-permeable cell which means it lets some things out and some in. An example of the cell membrane allowing a substance to enter is glucose.

Lysosomes: This organelle acts like a garbage disposal. It breaks down waste products in cells, like old dead cell particles that are no longer needed.

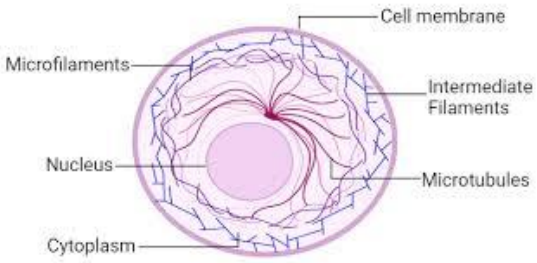


Golgi Apparatus: This organelle is like a post office. Post offices take mail, modify it, and then ship it off. The Golgi apparatus does this but with proteins that it receives instead.





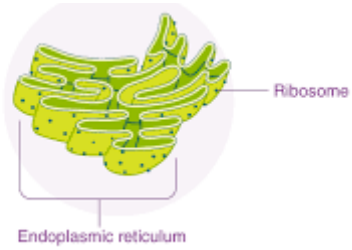
Cytoplasm: Gel-like substance that is inside of the cell. It supports and protects the cell organelles that float around/slide past each other. It is basically like jello and the organelles could be fruit floating in the jello.



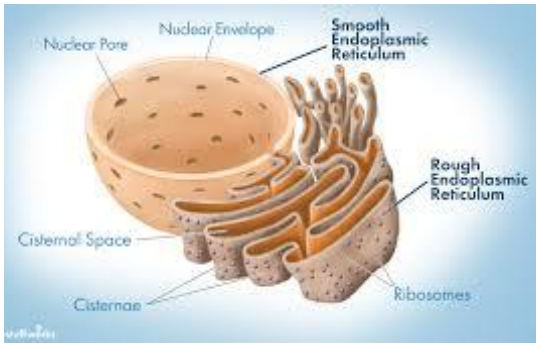
Cytoskeleton: This organelle is kind of like our skeleton. Our bones allow us to have specific shapes. The cytoskeleton helps the cell to maintain its shape.

Date: November, 4 2024 Time: 8:03- 9:30

Ribosomes: Build proteins and proteins are essential to help living organisms grow and repair tissue.

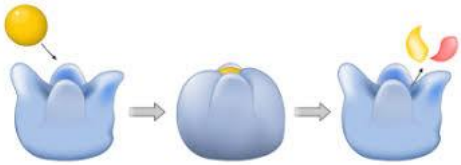


Endoplasmic Reticulum: Then endoplasmic reticulum is made up of two parts which are the Rough ER (endoplasmic reticulum) and the Smooth RE(endoplasmic reticulum). The Rough ER is involved with protein synthesis. At the Ribosomes the proteins are created and then enter the ER. While the proteins are at the Rough ER they are chemically modified and then transported to their next destination, which

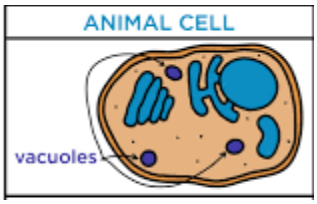


is usually the golgi apparatus. The Smooth ER isn't like the Rough ER, this has no ribosomes, so it's called SMOOTH ER. Smooth ER has enzymes that synthesize membrane lipids and detoxifies drugs.

- Enzymes: Proteins that help speed up metabolism, or the chemical reactions in our bodies. They build some substances and break others down.

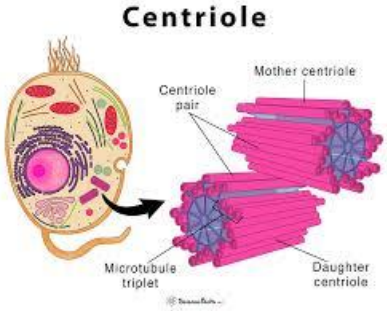


- Lipids: Are fatty compounds that perform a variety of functions in your body. They are a part of the cell membrane and help control what goes in and comes out. They also help with moving and storing energy, absorbing vitamins and making hormones.



Small Vacuole: This is like a garbage man. It helps store waste products so the rest of the cells are protected from it.

Centrioles: Organelles that help cells divide during Mitosis and Meiosis.



- Mitosis: A process of cell division where one cell divides into two genetically identical daughter cells
- Meiosis: A type of cell division in sexually reproducing organisms that reduces the amount of chromosomes in gametes.(a reproductive cell of an animal or plant)

Date: November, 12 2024 Time: 7:00- 7:31

Is Cancer a Genetic Disease?

Yes, cancer is a genetic disease. It is caused by changes in genes that control the way cells multiply. Each cell has a copy of your genes, which act like an instruction manual. Genes are sections of DNA that carry instructions to produce proteins. Scientists found hundreds of DNA and genetic mutations that help cancer grow and occur because: random mistakes in our DNA result as our cells multiply. Our DNA is altered by carcinogens in our environment, EX. tobacco, UV rays, and human papillomavirus (a viral infection that causes skin or mucous membranes growth) which was inherited from one of our parents. DNA changes, whether caused by a random mistake or the carcinogen (a carcinogen is an organism capable of causing cancer) can take place throughout our lives. Even in the womb which means you can be born with cancer. While genetic mutations aren't harmful on their own, over the years those healthy cells can become cancerous cells.

Three types of Cancer Genes:

Cancer is caused by changes in genes that control cell growth, division, and repair. The main types of genes involved are:

<u>Oncogenes:</u>

These genes help cells grow, but when they mutate or become overactive, they cause uncontrolled cell growth. These mutations usually happen during life, but can sometimes be genetic.

Tumor Suppressor Genes:

These genes help slow down cell growth and trigger cell death when needed. When damaged, cells can grow uncontrollably. Mutations in these genes usually occur over time.

DNA Repair Genes:

These genes fix mistakes in DNA during cell division. If they fail, mistakes build up, leading to cancer. Mutations in these genes can be inherited or happen during life.

Cancer usually results from a buildup of mutations in these genes, often from environmental factors or aging, though some mutations are genetic.

Are Genetic Mutations Bad?

Genetic mutations aren't necessarily a bad thing, in fact sometimes they can actually be beneficial. It really all depends on the location or context. There is something also known as genetic mutation. In general, the more pairs that are affected by a mutation, the larger the effect will be, so the mutation will be more harmful.

Date: November, 24 2024 Time: 5:00- 6:45

Addition to How is cancer passed down through genetics?

How cancer can be passed down through genetics when genetic mutations increase the risk of developing cancer. However, it's important to note that not all cancers are genetic. Here's how genetic inheritance can play a role:

1. **Inherited Gene Mutations**: Some families have genetic mutations that are passed from parents to children. These mutations are typically found in specific genes that control cell growth and division. When these genes are altered, it can increase the likelihood of cells becoming cancerous. Notable examples include mutations in the **BRCA1** and **BRCA2** genes, which increase the risk of breast or ovarian cancers.
2. **Dominant vs. Recessive Inheritance**: Most inherited cancer-related gene mutations follow a dominant inheritance pattern. This means that inheriting just one copy of the altered gene from either parent can increase the risk of cancer. In some cases, mutations are recessive, requiring two copies of the altered gene (one from each parent) to increase cancer risk.
3. **Genetic Testing**: People with a family history of certain cancers may undergo genetic testing to look for genetic mutations. Identifying a mutation can help the risk of cancer, guide early screening, or inform preventive measures.
4. **Environmental and Lifestyle Factors**: Even when a genetic tendency to cancer exists, environmental and lifestyle factors

such as diet, smoking, and exposure to carcinogens(substance or organism that is capable of causing cancer) can still play a significant role in the development of cancer. Therefore, genetic mutation does not guarantee that cancer will develop, but it increases the likelihood.

In summary, cancer can be passed down through genetics when gene mutations significantly raise the risk, but the development of cancer is usually influenced by a combination of genetic, environmental, and lifestyle factors.

Date: November, 30 2024 Time: 3:00- 4:50

Does Cancer Skip Generations?

Cancer doesn't "skip" generations, but genetic cancer risks can appear in different generations. Here's how:

1. **Genetic Inheritance**:

   Some cancer-related gene mutations can be passed from parent to child. If a parent has a mutation (like **BRCA1** or **BRCA2**), the child has a 50% chance of inheriting it.

   Even if the mutation is genetic, cancer may not develop because of lifestyle or environmental factors.

2. **"Skipping" Generations**:

   Cancer might appear in one generation but not the next, but this doesn't mean it's skipped. Other factors may affect

whether cancer develops in the second generation.

Date: December, 9 2024 Time: 10:00- 10:30

How Do You Get Cancer From The Environment?

Environmental factors can significantly influence the development of cancer. Below are several key topics related to cancer and the environment:

1. **Carcinogens in the Environment**:

   Air Pollution: Exposure to pollutants like benzene, formaldehyde, and diesel exhaust increases the risk of lung cancer and other cancers.

   Tobacco Smoke: Smoking and exposure to secondhand smoke are major environmental factors that contribute to various cancers, especially lung cancer.

   Pesticides and Herbicides: Exposure to certain chemicals in agricultural places has been linked to an increased risk of non-Hodgkin lymphoma and other cancers.

   Industrial Chemicals: Substances like asbestos, arsenic, and certain solvents (Ex: benzene, vinyl chloride) have been found to increase cancer risks, particularly lung, liver, and bladder cancers.

2. **Diet and Nutrition**:

   Processed Foods and Red Meat: A diet high in processed

meats and red meats has been associated with an increased risk of colorectal cancer.

Obesity and Cancer Risk: Excess body weight is a known risk factor for various cancers, including breast, colorectal, liver, and kidney cancers.

Chemicals in Food: Certain food additives, preservatives, and contaminants (Ex: acrylamide in fried foods, aflatoxins in improperly stored crops) may increase cancer risk.

3. **Radiation Exposure**:

   Ultraviolet (UV) Radiation: Overexposure to UV radiation from the sun or tanning beds can cause skin cancers, including melanoma, basal cell carcinoma, and squamous cell carcinoma.

   Ionizing Radiation: Exposure to ionizing radiation, such as from nuclear accidents (Ex: Chernobyl, Fukushima) or medical imaging, can increase the risk of leukemia, thyroid cancer, and other cancers.

4. **Occupational Exposure**:

   Workplace Hazards: Certain occupations expose workers to carcinogens, such as those working in mining, construction, and chemical industries.

   Shift Work and Cancer: Some research suggests that working night shifts may disrupt the body's circadian rhythm, potentially increasing the risk of breast cancer and other cancers.

5. **Environmental Toxins and Cancer**:

Heavy Metals: Exposure to heavy metals such as lead, cadmium, and mercury has been linked to various cancers, including kidney, lung, and liver cancers.

Endocrine Disruptors: Chemicals that interfere with hormone systems, such as bisphenol A, phthalates, and pesticides, may increase the risk of hormone-related cancers like breast and prostate cancer.

6. **Climate Change and Cancer**:

   Changing Patterns of Exposure: Climate change can lead to changes in environmental factors that influence cancer risks, such as increased levels of air pollution, higher temperatures, and more intense UV radiation.

   Impact on Vulnerable Populations: Rising temperatures and extreme weather events may disproportionately affect populations, leading to higher rates of heat-related illness and cancers linked to environmental stressors.

7. **Environmental Justice and Cancer**:

   Disparities in Exposure: Low-income and minority communities often face higher levels of environmental pollution and limited access to healthcare, increasing their cancer risks.

   Advocacy and Policy: Efforts to address environmental justice involve recommending stronger environmental regulations, community awareness, and health screenings in affected communities.

8. **Chemicals in Consumer Products**:

Cosmetics and Personal Care Products: Certain chemicals in cosmetics, such as parabens and formaldehyde-releasing agents, may increase cancer risk.

Cleaning Products and Household Chemicals: Some cleaning agents and air fresheners contain substances that have been linked to respiratory cancers or endocrine disruption.

9. **Water Contaminants**:

   Drinking Water Contaminants: Contaminants like radon, arsenic, and chlorinated compounds in drinking water can cause the development of cancers, including bladder, kidney, and lung cancers.

10. **Environmental Impact on Cancer Prevention**:

    Public Health Measures: Efforts to reduce cancer risk through public health campaigns, such as promoting smoking, reducing exposure to harmful chemicals, and encouraging healthier lifestyles, can have a significant impact on cancer prevention.

    Regulations and Policies: The role of government regulations in reducing environmental carcinogens (Ex: restrictions on toxic chemicals, emissions, and pollution control) plays an important part in reducing cancer rates.

These topics highlight the relationship between the environment and cancer. They also explain the importance of environmental and public health policies in reducing exposure to carcinogens and promoting cancer prevention. Date: December, 12 2024 Time: 2:00- 3:30

Hypothesis

If mutations happen in the cell cycle, then genes, and cancer cells will grow faster because these mutations disturb normal control of cell division. For example, mutations can make cells divide uncontrollably, leading to quicker tumor growth which means the cancer will be more violent. Your model can predict how these mutations affect cancer growth and how fast it continues.

Materials

- Computer with internet access
- Log Book
- The IDE (integrated development environment) called Scratch

Procedure

1. To create the project from the bottom up, open Scratch.
2. Select "Create" at the top of the screen to create a new project.
3. Select the variables tab to create new variables.
    a. Select the "Make a Variable" box.
    b. Type the new variable names in the pop-up window that says "New Variable." The variable names are:
        i. *Mutation Rate*
        ii. *Cancer Mutation Threshold*

iii. *Total Number of Mutations:* How many mutations are in the DNA of the cells in your model.

iv. *Number of Cell Cycles*: How many cell cycles have passed before a cancer cell appears.

v. *Cell death*

c. Select ok to add the variable to the checklist, like in Figure 3.

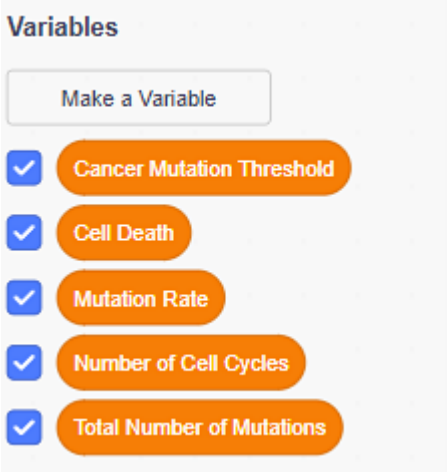


Figure 3. Scratch blocks for the five variables needed to build the model.

4. Select the "Events" tab.
    a. Drag and drop the "when the green flag is clicked" icon to the main screen.
5. Select the "Variables" tab.
    a. Select your variable from the drop-down menu in the set variable function.

b. Drag and drop each pre-set variable to the program. Place and attach them under the "when the green flag is clicked" icon, like Figure 4.

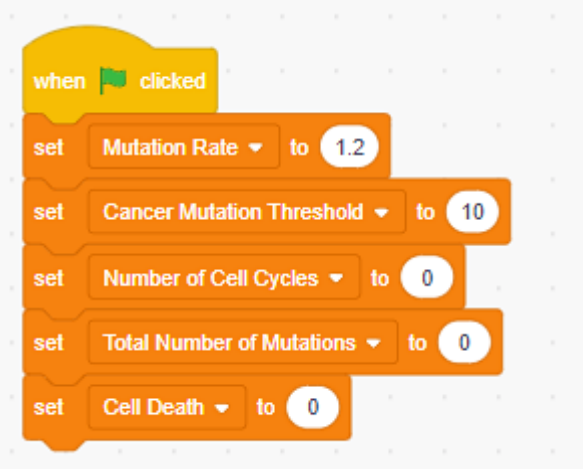
c. Set the starting values for each of the 5 variables to their threshold rates.

Links from the table: Mutation Rate- Lee-Six, H., et al. (2018, September 5).*Population dynamics of normal human blood inferred from somatic mutations.* Retrieved August 27, 2024. (Look at paragraph 2 under the title “Somatic mutations acquired during embryonic development”

Cancer Mutation Threshold- Martincorena I., et al.(2017, November 6). *Universal Patterns of Selection in Cancer and Somatic Tissues.* Retrieved August 27, 2024. (See paragraph 5 under the title “Number of Driver Mutations per Tumor”

Death Cells- Zhang, W., et al. (2024, March 7) *Pan-cancer evaluation of regulated cell death to predict overall survival and immune checkpoint inhibitor response.* Retrieved August 27, 2024. (Look at the first paragraph of title “Result”

d. The remaining thresholds of the variables not shown in Table 1 should be set to zero.



e. Figure 4. Scratch code showing the starting values of the variables based on research findings links provided above.

6. Create a separate program for what happens during a single-cell division. Figure 5 shows one possible solution.

   a. Select the change function to increase the *Number of Cell Cycles* by one after each cell division.

      i. Select the *Number of Cell Cycles* variable in the drop-down menu with the change text out front.

      ii. Double-check to make sure the change number is by increments of 1.

   b. Select the change function to increase the *Total Number of Mutations* in the daughter cell by the *Mutation Rate*.

      i. Select the *Total Number of Mutations* variable in the drop-down menu with the change text out front.

Drag and drop it under the previously changed variable.

ii. Then, drag and drop the *Mutation Rate* variable over the number box to insert the variable.

c. Add a statement to indicate whether the cell survives or if there is *Cell Death*.

i. Select and set the *Cell Death* variable under the previous variables. Drag and drop it under the previously changed variable.

ii. Go to the operator tab, select the pick random command with two number boxes, and drag and drop it over the number box to create a range.

iii. Change the cell death range to reflect the chance of escaping cell death (0.1%) to the range from 0.000 to 1.000 for the *Cell Death* variable.

d. Add an if-then statement to indicate what happens when a daughter cell escapes *Cell Death*.

i. Go to the control tab to select an if, then statement. Drag, drop, and attach it under the previous variable.

ii. Go to the operator's tab and select a greater than statement

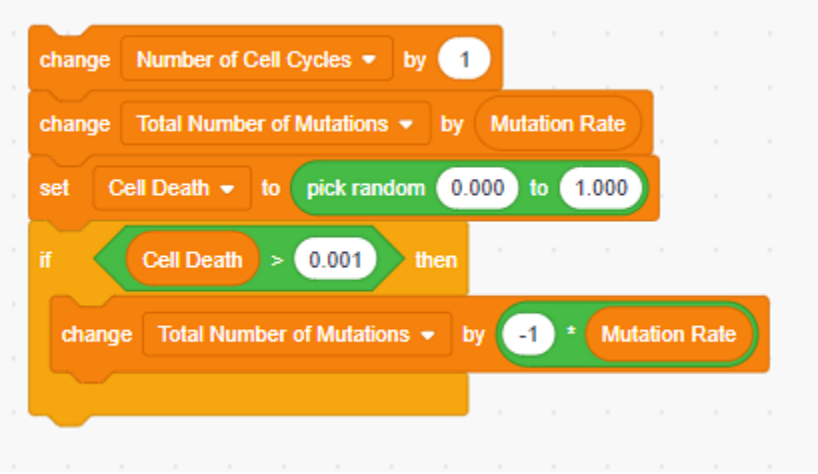
iii. Add the *Cell Death* variable on the left of the greater than symbol.

iv. Update the threshold to 0.001. The daughter cells' chance of escaping *Cell Death* is low (>0.001). Therefore, if the value is greater than 0.001, the daughter cell will die.

v. Add a change statement with the *Total Number of Mutations* variable to the insert of the then statement. If the daughter cell dies, this will decrease the *Total Number of Mutations* variable.

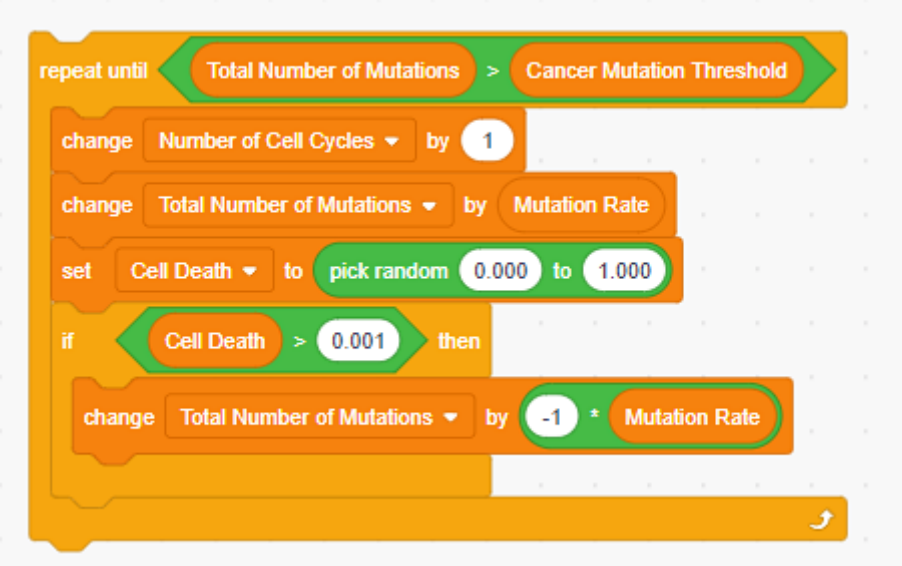
vi. Go to the operator tab and add a multiplication operator to the number box.

vii. Update the number box in the change statement to -1 multiplied by the *Mutation Rate* since a decrease in this variable indicates that there are fewer of the *Total Number of Mutations* since the daughter cell died, but the mutated parental cell remains.

7. 

8. Figure 5. Scratch code for what should happen during a single cell cycle.
9. Now that we have the code for a single cell division, we need to add code to make the cancer model continuously repeat cell cycles until the conditions for cancer are met. One possible solution is shown in Figure 6.
    a. To create a repeating loop to model what happens to mutated cancer cells over time:
        i. Select the control tab. Drag and drop the repeat until control under your wait 1-second function.
        ii. Go to the operator tab. Drag and drop the greater than operator into the hexagon of the repeat until function.
            1. Add the *Total Number of Mutations* variable to the left number box.
            2. Drag and drop the *Cancer Mutation Threshold* variable to the right number box.
        iii. Move the second set of programs representing a single cell cycle from Figure 5 into the repeat until function to represent multiple cell cycles.
            1. *Optional: You can also add graphics to make the program visually appealing and clear to users when the model is finished running. To do this, go to the Looks tab and insert the switch*

costume to function before and after the program, like in the example model.



2. Figure 6. The scratch code for a single cell cycle will be repeated until the cancer mutation threshold is met.

10. Add a wait 1-second control function between the single cancer cell variable thresholds (Figure 4) and the repeating cell cycles step (Figure 6).

Run your model *at least* ten times. Each time, record in your lab notebook how many cell cycles (*Number of Cell Cycles*) it takes for a cancer to appear.

Modifying The Cancer Disease Model

1. Once you have your starting cancer disease model built, you are ready to modify it to see what happens to the chances of

developing cancer due to aging with additional specific environmental and genetic elements. Keep in mind that your original cancer model still contributes to the risk of cancer because all humans age. However, additional environmental and genetic factors can increase/decrease your risk.

2. **Environment Element:**

Smoking cigarettes is an environmental factor that changes a person's chances of getting lung cancer. Use this link: Klein, A. (2016, November 3). *Every 50 cigarettes smoked cause one DNA mutation per lung cell*. Retrieved August 27, 2024. (See the second paragraph of the article. It's about how smoking affects the mutation rate of cells. Use this information to modify your cancer disease model to include the impact of smoking a pack of cigarettes a day.

An example is 'If the mutation rate for smoking a certain amount of cigarettes is 2 per cell for every 50 packs smoked. Then per day would be 1 pack per day. So divide the number of mutations by the number of packs per day, like this: 2/2.5=0.8 this will give the average number of mutations per cigarette pack per day. Then. add that to the previous mutation rate like this: 1.2 + 0.8 = 2

Based on your own calculations, update the *Mutation Rate* variable.

Run your model at least ten times for people who smoke 1 pack a day.

Record each trial in your lab notebook, to keep track of the *Number of Cell Cycles* it takes for a cancer cell to appear.

3. **Genetic Factor:**

Some families with a history of breast cancer have mutations that change the function of the *BRCA*1 (Breast cancer gene 1) or *BRCA2* (Breast cancer gene 2) genes. Use this link: Zámborszky, J. et al. (2016, July 25). *Loss of BRCA1 or BRCA2 markedly increases the rate of base substitution mutagenesis and has distinct effects on genomic deletions.* Retrieved August 27, 2024. (See paragraph 4 of the results section. This is to help modify your starting cancer disease model to see what happens to the chances of getting breast cancer with a *BRCA1* or *BRCA2* mutation.

An example is if the mutation rate is five times higher than the normal rate, then you will need to multiply the previous mutation rate from the original model by five, so 1.2 x 5 = 6.

Based on your own calculations, update the *Mutation Rate* variable.

Run your model at *least* ten times for people with *BRCA1* or *BRCA2* mutations.

Record each trial in your lab notebook, to keep track of the number of cell cycles it takes to create a cancerous cell.

4.Compare your results for the three cancer disease models you built (cancer model, cancer model + smoking, and cancer model + genetic mutation in *BRCA1* or *BRCA2*).

> What is the range of the *Number of Cell Cycles* for each model? The range is the minimum and maximum number. What is the average *Number of Cell Cycles* (of the 10 trials) before cancer appears in each model?

Calculate the years it will take for the cell to become cancerous. To do this, use the primary literature to inform your calculation that, on average, the cell cycle takes 1 full day to complete. An example is if the *Number of Cell Cycles* = 9426 cycles / 365 cell cycles per year = 26 years

> According to the NCI, the average age of cancer diagnosis is 66. However, the multiplication rate of mutated daughter cells that escape apoptosis and the immune system takes place approximately 25 years before this! Add the years until the cells become cancerous to 40 to get the predicted age of diagnosis for each model. An example is if the number of years until the cell becomes cancerous is 9.8, add 9.8 years + 40 years to get a predicted age of diagnosis of 49.8 years.

Validating Your Model

Use these links: - NCI. (2021, March 5) *Age and Cancer Risk.* Retrieved August 28, 2024. (Read paragraph 2) For age impact on cancer risk

-LoPiccolo, J., et al. (2024, January 9). *Lung cancer in patients who have never smoked - an emerging disease.* Retrieved August 28, 2024. (Review paragraph two under the Epidemiology heading) For Smoking's impact on lung cancer

-NCI Staff. (July 12, 2017). *Large Study Verifies Cancer Risk for Women Carrying BRCA1 or BRCA2 Mutations.* Retrieved August 27, 2024. (Review paragraph 2 under the Necessary Confirmation heading) For *BRCA1* or *BRCA2* impact on breast cancer

and any additional resources to find the average age of cancer diagnosis due to aging, smoking, and BRAC1 or BRAC2 status from real-world data. Fill in your research findings in Table 3.

- Then, compare your model's results to see if they make sense compared to the real-world data.
    a. Compare your original model to the average age of diagnosis of cancer.
    b. Compare your models to the average age at which smokers versus non-smokers are diagnosed with lung cancer.

c. Compare your models to the average age frequency of people with breast cancer with and without mutations that change the function of *BRCA1* or *BRCA2*.

- Do your models match the real-world data? Explain why or why not.

Data

Table 1: Cancer Model					
No. of Test	Mutation Rate	Cancer Mutation Threshold	No. of Cell cycle	Total no. of Mutation	Cell Death
1	1.2	10	12683	10.8	0.000679
2	1.2	10	9844	10.8	0.000951
3	1.2	10	6758	10.8	0.0008
4	1.2	10	5820	10.8	0.000214
5	1.2	10	8548	10.8	0.000007
6	1.2	10	20510	10.8	0.000943
7	1.2	10	14673	10.8	0.00041
8	1.2	0	12244	10.8	0.000687
9	1.2	10	7876	10.8	0.000634
10	1.2	10	5493	10.8	0.000776

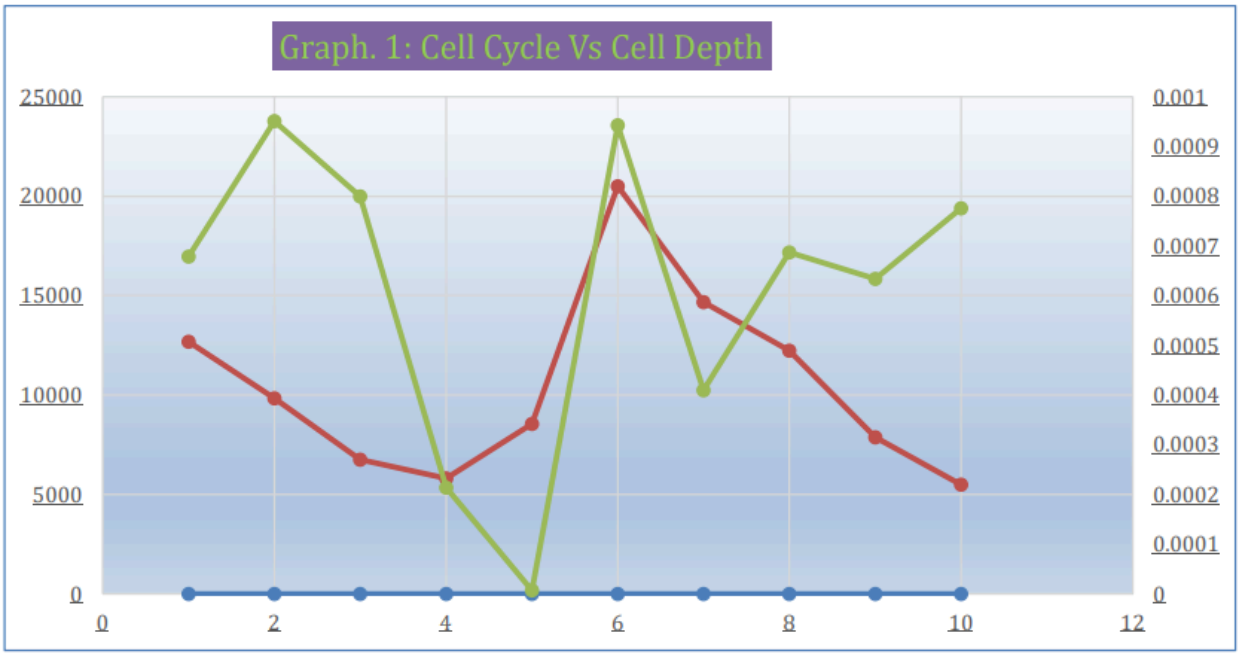


Table 2: Smoking plus Cancer Model					
No. of Test	Mutation Rate	Cancer Mutation Threshold	No. of Cell cycle	Total no. of Mutation	Cell Death
1	2	10	6771	12	0.000865
2	2	10	9735	12	0.000752
3	2	10	5452	12	0.000822
4	2	10	7165	12	0.000644
5	2	10	4722	12	0.00935
6	2	10	7340	12	0.000649
7	2	10	9721	12	0.000992
8	2	0	6126	12	0.000014
9	2	10	3807	12	0.000876
10	2	10	5007	12	0.000043

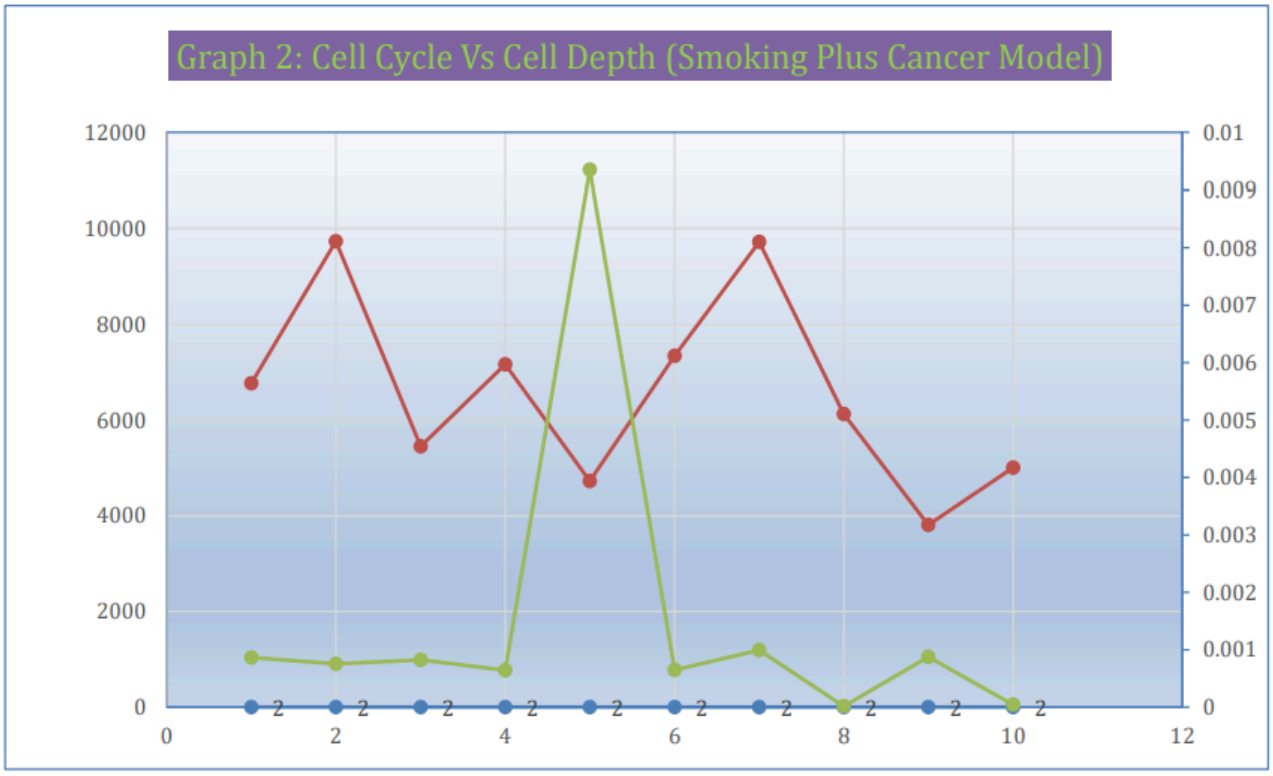


Table 3: Cancer Plus genetic Model					
No. of Test	Mutation Rate	Cancer Mutation Threshold	No. of Cell cycle	Total no. of Mutation	Cell Death
1	6	10	1418	12	0.000461
2	6	10	1927	12	0.000345
3	6	10	512	12	0.000136
4	6	10	1618	12	0.000864
5	6	10	3029	12	0.000949
6	6	10	1153	12	0.000792
7	6	10	2439	12	0.000462
8	6	0	1305	12	0.000449
9	6	10	4456	12	0.000306
10	6	10	477	12	0.000706

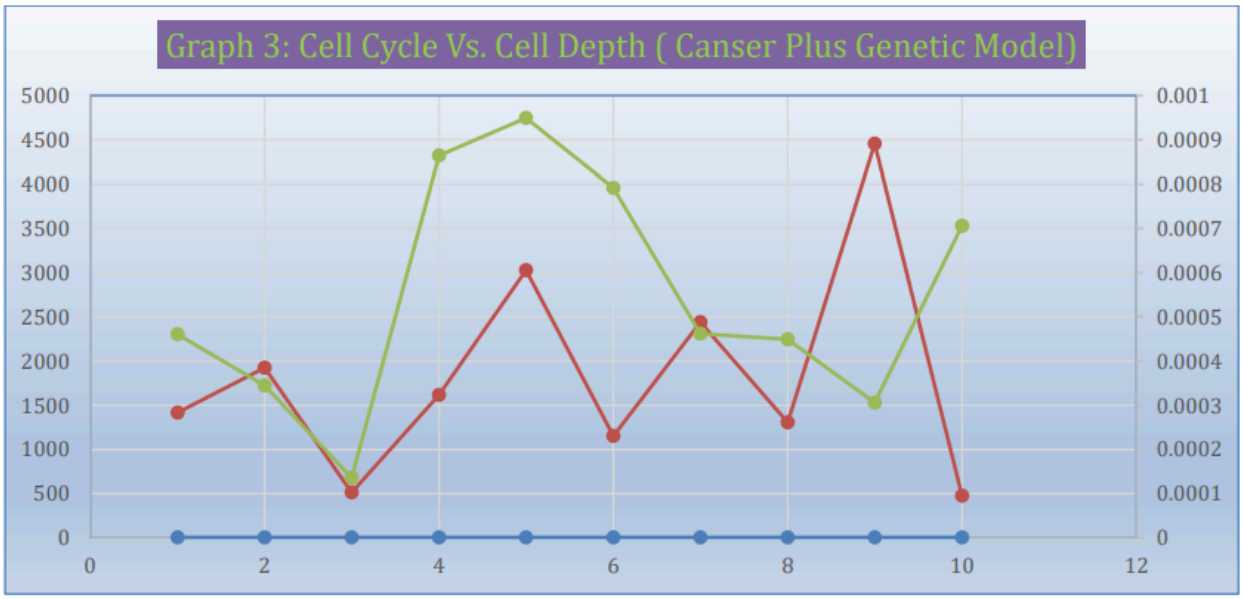
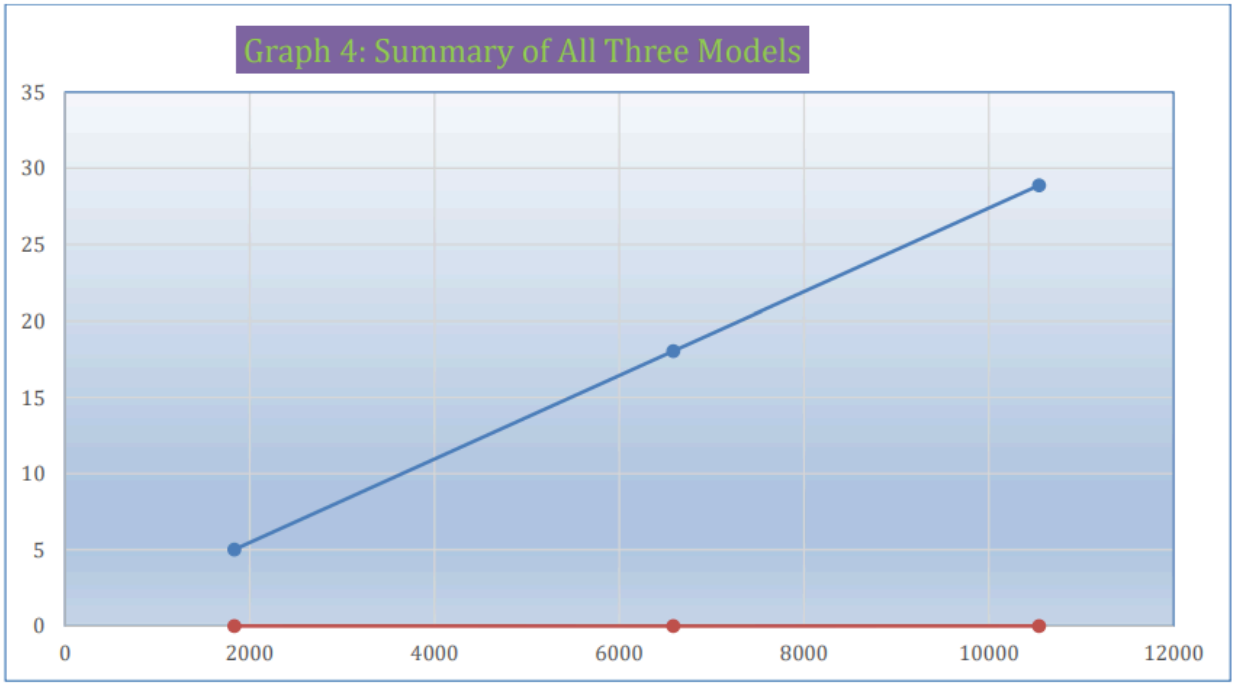


Table 4: Summary of all three Models					
Name of Model	No. of Cell Cycle range	Range No. of Cell Cycle (Min.- Max)	Average No. of Cell Cycle	Years until the cell becomes Cancer	Predicted Age of Diagnosis (Years)
Cancer Model	1-10	5493-20510	10544.9	28.89	69 years
Cancer Plus Smoking Model	1-10	3847-9735	6584.6	18.04	58 years
Cancer Plus Genetic Model (BRCA 1 or BRCA 2)	1-10	477-4456	1833.4	5.02	45 years



Observation

When I was constructing my cancer model I noticed is, if the starting values or 1 little variable is out of place then the model won't even start. Something else I noticed is the Mutation Rate, Cancer Mutation Threshold, and Total number of Mutations rate didn't change throughout the 10 rounds I ran the model. This was similar with my other two models (Smoking + Cancer model
and Cancer model Cancer model + Genetic mutation) but the numbers weren't always the same. Ex 1.2 and 2 or 10.8 and only 10.

The cell death value is a really small number and continuously started with 0.000. If Cell Death is below 0.001, then the total number of mutations decreases by -1 * Mutation Rate, which leads to slower mutation growth. This introduces a survival effect where cells with higher cell death rates might take longer to reach the cancer threshold. Values with higher cell cycles have relatively higher cell death values. Some trials with lower cell cycles have lower cell death values, suggesting that faster mutation growth can happen when cell death is minimal. Some cells take significantly longer due to frequent mutation loss caused by high cell death rates. The most important thing I noticed is that Genetic mutations (BRCA1/BRCA2) lead to cancer the fastest (5.02 years), with the youngest predicted age of diagnosis (45 years). Smoking advances cancer formation compared to the baseline cancer model, leading to an earlier diagnosis at 58 years. The standard cancer model has the longest time until cancerous transformation (28.89 years) and a predicted diagnosis at 69 years.

Date: December, 30 2024 Time: 11:00- 3:50

Analysis

This experiments mutation growth in cells while factoring in cell death, using a mutation rate of 1.2 and a cancer mutation threshold of 10. The number of cell cycles required to reach the threshold varied significantly, ranging from 5,493 to 20,510 cycles, highlighting the impact of mutation loss due to cell death. Randomized cell death values influenced mutation progression, with higher cell death

slowing growth and lower cell death allowing faster threshold achievement. Despite variability, all trials eventually reached the cancer threshold, showing that while cell death can delay progression, it does not entirely prevent it. This study underscores the balance between mutation growth and loss, mirroring real biological processes. Future research could explore different mutation rates and cell death thresholds to further analyze their effects on cancer progression. Date: January 30 2024 Time: 1:00- 1:50

Variables

Manipulated: carcinogen exposure or gene mutations
Responding: cancer cell growth or mutation rate
Controlled: cell type, experimental conditions

Date: February 1, 2024 Time: 9:20- 9:40

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   Date: October 23 2024

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Conclusion

In conclusion, my hypothesis was correct because I stated that the mutation growth during the cell cycle can lead to uncontrolled cell division. My model demonstrates that a higher mutation rate results in faster cancer progression, but it also introduces complication by showing how cell death slows this process. Mutation loss due to cell

death causes change in how quickly cells reach the cancer threshold, meaning cancer growth isn't always rapid or aggressive. So, cancer progression depends on the balance between mutation growth and mutation loss, making it an unpredictable but measurable process.

The development of cancer is influenced by both genetic and environmental factors, as they affect the way cells grow and divide. Genes play a role in regulating cell division, making sure that new daughter cells are healthy copies of their parent cells. However, mutations in specific genes, such as oncogenes and tumor suppressor genes, can dearange this process, leading to uncontrolled cell growth and tumor formation. These mutations can be inherited or caused by environmental exposures, examples being radiation, chemicals, and lifestyle.

During normal cell division, checkpoints in the cell cycle help prevent errors and repair damaged DNA. When these systems fail, due to genetic mutations and other factors, abnormal cells continue to divide unchecked, leading to the development of cancerous tissues. Also, mutations in DNA repair genes that can prevent the body from correcting mistakes, increasing the chance of developing cancer.

Understanding the interaction between genetics and environmental factors allows scientists to develop better cancer prevention and treatment strategies. Regular screenings, genetic testing, and targeted therapies play a key role in detecting individuals at higher risk and offering more effective treatment options. These help identify signs of cancer, assess genetic tendencies, and make sure that treatments are targeted to help specific genetic mutations, improving

outcomes for patients.

So we should try to maintain a healthy lifestyle and supporting ongoing research in genetics and cancer biology, we can reduce cancer risk and improve overall health outcomes.

Date: February 12, 2024 Time: 8:30- 9:15