ACTIVITY 2: EXAMINING CANCER PATIENT DATA

INTRODUCTION

In this activity, we'll examine genes that are mutated in the tumors of actual patients to identify patterns and trends.

Cancer consists of a group of diseases caused by mutations in the DNA of cells. Some mutations are inherited, but most occur during a person's lifetime as a result of random errors in replication. Environmental factors that damage DNA, such as smoking and sunlight, can also cause mutations to occur.

As a single cell in the body accumulates mutations, one of those mutations could provide a survival or a growth advantage to the cell, causing the cell to divide at a faster-than-normal pace or to not die. The resulting daughter cells with that mutation, which are dividing quickly, are more likely to accumulate additional mutations. Additional mutations that affect cell division may cause those cells to divide even faster. Eventually a cell may acquire enough mutations that it starts to grow and divide uncontrollably (**Figure 1**).

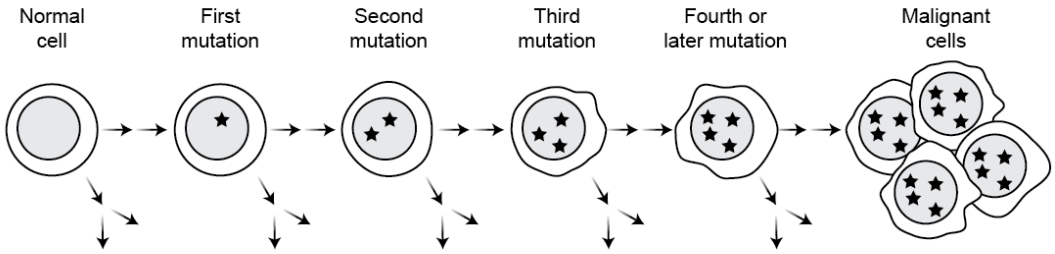

Figure 1. Schematic of cancer development. Cells accumulate mutations as they divide. Mutations that are most advantageous for cell growth and survival are passed on to daughter cells, which, in turn, acquire further mutations and may eventually become malignant cancer cells. (The arrows represent multiple cell divisions.)

MATERIALS

- *Cancer as a Genetic Disease* video clip (http://www.hhmi.org/biointeractive/cancer-genetic-disease-video-highlights)
- *Activity 2: Examining Cancer Patient Data* student document, which includes the *Video Worksheet*,
- a *Cancer Patient Card*

PROCEDURE

Part 1: To begin, watch the *Cancer as a Genetic Disease* video clip (8:30 minutes) featuring cancer researcher Dr. Charles L. Sawyers. Use the *Video Worksheet* to review the important concepts.

Part 2: Your instructor will give you a cancer card (**Figure 2**). Each card describes the important genetic mutations found in a cancer from one person. The DNA from your patient's tumor was sequenced to identify all the mutations present. Only mutations known to drive the development of cancer are listed on the cards.

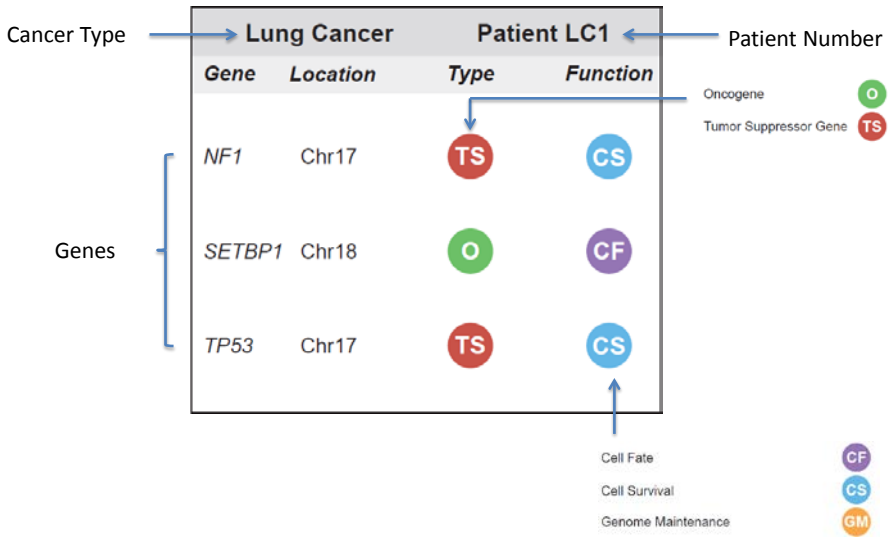

Figure 2. Example of a *Cancer Patient Card* and legend.

Find the other students in your class who have the same type of cancer on their cards. For example, if you have a pancreatic cancer card, look for other students with pancreatic cancer cards. Form small groups based on cancer type, compare your cards, and record your observations.

Some questions to consider:

- Are all the genes located on the same chromosomes?
- Are the same genes involved in the same types of cancer?
- Is the same number of genes affected in all the cancers?
- Are some functions more common than others?

Part 3: Now look for students who share your group number. For example, all the students with Group 1 cards will gather. Form groups based on group numbers, compare your cards, and record your observations. Remember that each card represents a different cancer patient. Are there any new patterns or trends that you did not identify in your first group?

Part 4: After your class discussion, complete a 3-2-1 analysis. In this analysis, you will share

- three things you learned from the activity,
- two things that surprised or interested you, and
- one question you still have.

NAME _____ DATE _____

VIDEO WORKSHEET

1. What was the main purpose of the large-scale cancer study that Dr. Sawyers describes in the video?

2. Why was it important to sequence both cancer DNA and normal DNA from people with cancer?

3. How did the investigators share data as they worked? And why was it important to share?

4. As of spring 2013, about _____ genes associated with cancer have been identified. What is the approximate breakdown of oncogenes versus tumor suppressor genes?

5. Using Dr. Sawyers's analogy (the gas pedal and brake), a mutated oncogene is like _____ and a mutated tumor suppressor gene is like _____. What does this mean in terms of how the cell grows and divides?

6. Distinguish between a proto-oncogene and an oncogene.

7. The mutated allele (oncogene) is **dominant/recessive** compared to the normal, nonmutated allele (proto-oncogene) on the other chromosome. (Circle a choice.)

8. The mutated allele of a tumor suppressor gene is **dominant/recessive** compared to the normal, nonmutated allele on the other chromosome. (Circle a choice.)

9. Does Dr. Sawyers think many more cancer genes will be identified? Will the number grow exponentially?

10. List the three "buckets" in which scientists categorize cancer genes. Approximately how many genes are in each bucket?

11. How do *p53* and *cyclin D1* differ in how they affect the cell cycle?

12. Is *p53* a **tumor suppressor gene/oncogene**? Is *cyclin D1* a **tumor suppressor gene/oncogene**? (Circle a choice.)

13. Consider genome maintenance genes:

a. Does DNA polymerase make mistakes during DNA replication?

b. How often?

c. Explain the proofreading system.

d. Explain what happens if a mutation occurs in the genes that encode proofreading enzymes.

14. Why is it that the longer we live, the more likely we are to develop cancer?