



CASE TEACHING NOTES **for** **"Not An Old Person's Disease"**

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OVERVIEW / BACKGROUND

Case Synopsis

This case focuses on melanoma, which is a particularly deadly type of skin cancer. In this fictitious story, students are introduced to a 20-year-old female who sunburns easily and has several other melanoma risk factors. One day, she discovers that a mole on her leg has started to itch. When she looks at it, it looks different from what she remembers. On the advice of a friend, she decides to get it checked out. Her doctor explains the role of genes in cancer formation and intends to do a biopsy to determine whether her mole is malignant or benign.

The overall goal of this case is to introduce students to the genetic basis of cancer development. Although it helps if students have a basic background in genetics, the terminology needed to understand the material presented here is minor and can be integrated into the case presentation itself. Concepts such as gene, mutation, recessive, and dominant can simply be discussed or defined at appropriate times throughout the case.

This case was written specifically for a general education health and wellness class that is entirely case based. Students who enroll in this course have a wide range of ages and backgrounds, from first year students to seniors, and from non-science majors to biology majors. After having taught the case several times over the past two years, the authors have concluded that the material is presented in a way that is comprehensible for this type of diverse audience.

Objectives

Students will learn:

1. the risk factors of melanoma
2. that cancer is not necessarily an "old person's disease"
3. that cancer is caused by mutations in normal genes found in cells
4. that there is a difference between benign and malignant tumors
5. the ABCDE's of self-examination for melanoma
6. that predisposition to melanoma (and other cancers) can be inherited

BLOCKS OF ANALYSIS

One of the broader concepts that we hope students will grasp from this case is that cancer, for the most part, is not something that you "catch" like a cold. For certain, melanoma is not contagious. As described in the case, it is usually the result of mutation(s) in genes that every healthy person has and needs in order to make more cells when necessary. These are the cell cycle genes.

One class of cell cycle genes is called proto-oncogenes. There are normal situations that arise when cells need to grow and divide, and these proto-oncogenes are responsible for initiating this growth and division. However, these normal genes can be transformed into cancer-causing genes when mutations occur in them that cause them to allow cells to divide all the time. If cells are dividing all the time, an abnormal mass of cells, or cancer, can result. These mutated forms of the normal proto-oncogenes are called oncogenes. The prefix onco- in oncogene refers to genes that can lead to cancer, just like an oncologist is someone who studies cancer. "Proto-" refers to the fact that even though the normal role of these genes is not to cause cancer, they *can* be mutated to cause cancer. It is possible to have one mutated copy and one normal copy of a proto-oncogene and still get cancer. In this case, the mutation is dominant and it doesn't matter that there is still one good (unmutated) copy of the gene.

Another class of genes that can lead to cancer when mutated are the tumor suppressor genes. These are the genes that the doctor refers to when he is discussing Judy's possible predisposition to cancer. Just as their name sounds, these genes make proteins that normally inhibit cell division and prevent tumors from forming. Just as proto-oncogenes normally tell cells to *start* growing and dividing, these normal tumor suppressor genes tell cells to *stop* growing and dividing. If one copy of a tumor suppressor gene has a mutation in it, your second copy is still present in your cells to do its normal job. However, if both copies of a tumor suppressor gene have a mutation, then your cells will not have that normal control to stop them from dividing at an inappropriate time. Again, if cells are growing all the time, an abnormal tumorous growth of cells, or cancer, can result.

In summary, there is a careful balance in normal cells between the "cell division-promoting" proto-oncogenes and the "cell division-inhibiting" tumor suppressor genes. If this balance is tipped one way or another by mutation, cancer can develop.

One possible point of confusion with the idea that you don't "catch cancer" is if students ask about viral-induced cancers such as the human papillomaviruses that can lead to cervical cancer or human immunodeficiency virus (HIV) that can lead to Kaposi's sarcomas. Viral infections can lead to cancer in two ways. First, viruses can insert their DNA into the host's genome. Insertion into a cell cycle gene could lead to its inappropriate activation. (Likewise, insertion into a tumor suppressor gene could lead to its inappropriate inactivation.) In this scenario, it is still the host's own genes that are contributing to the cancer. A second possibility is that viruses can bring along their own genes that promote cell division in the human host. Therefore, if the virus DNA is inserted somewhere in the host DNA that results in these viral genes' being constantly activated, a cancer could develop. In this situation, the cancer *is* the result of foreign genes and, therefore, the cancer was "caught." Because this might be confusing to students, we purposely do not discuss viruses in the case. However, if the discussion moves in this direction, the instructor may want to cover this. Any current introduction to biology, cell biology, or genetics text should have additional information on the cell cycle and cancer.

Answer Key

Answers to the questions posed in the case study are provided in a separate answer key to the case. Those answers are password-protected. To access the answers for this case, go to [the key](#). You will be

prompted for a username and password. If you have not yet registered with us, you can see whether you are eligible for an account by reviewing our [password policy](#) and then [apply online](#).

CLASSROOM MANAGEMENT

The class that we have taught typically consists of 30 students divided into six groups. However, this case would also work with a larger class. The groups are assigned by the instructor at the beginning of the semester and do not change during the semester. This case can be presented within a 60-minute class period, although it may be rushed. The authors have presented this case in one of two ways successfully:

- We handed out Parts I and II consecutively during one 60-minute class period and then finished the case during a second class period.
- We presented all three parts consecutively during a 2-hour class period, leaving ample time at the end for questions.

Alternatively, the case could be taught over two class periods as follows:

After completing Part I, each group is asked to generate a list of questions to which Judy may want to know the answers. After sharing these questions as a class, the most important four or five questions are chosen by class vote. Each member of each group is then responsible for finding the answer to one of the questions. The second class period begins with a discussion of their findings, after which the rest of the case is presented. This scenario allows the students to participate in the case as "patients" rather than simply as "readers."

In some cases, depending on the science background of the audience, the concepts may be difficult for students and the instructor should be prepared to provide a mini-lecture when necessary. Students should also be made aware of the websites and should be encouraged to find more information if they would like to learn more about melanoma and how to self-examine.

REFERENCES

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* **Note:** Both authors contributed equally to this case and its teaching notes.

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