IMMORTAL CELLS

AN INTRODUCTION TO THE CELL CYCLE, MITOSIS & CANCER

Lesson Overview

This is a lesson to teach students about the life cycle of a cell and cellular functions including growth, division (mitosis), repair, and cell death (apoptosis). It is intended for use in an introductory high school biology class. The cell processes are taught through a disease theme, in this case, exploring cancer.

Description of Activity

The lesson has 3 parts to it. It begins with a writing prompt to engage students in a class discussion about aging and death. From that the Hayflick limit idea is introduced – that cells have a limited number of times they can divide. This idea is compared to cancer cells which are essentially immortal. Students then explore the Inside Cancer website to learn about the Hallmarks of Cancer. Next, the students explore the events of mitosis and the cell cycle by constructing a simple mitosis puzzle and taking notes on the events happening at each stage. Finally, students revisit the Inside Cancer site to find where disruption of the normal life cycle occurs in cancer cells.

Background

Cell processes are the result of complex interactions between a cell's genetic code (DNA) and its environment. Cells grow, divide, and die in response to cellular and extracellular signals. Sometimes cells are damaged beyond repair by environmental factors and undergo a programmed cell death - *apoptosis*. Some cells take advantage of apoptosis to die for the greater good of the organism.

Before a cell divides, it must make copies of its DNA and all the cellular organelles needed for the daughter cells. Each time DNA replication occurs, mutations occur. The cell has mechanisms for repairing both mistakes in DNA replication and other non-replication mutations that occur, but they are not foolproof. Even these mechanisms for repair are controlled by proteins that are produced from genes that are susceptible to mutations. If mutations occur in any of the key molecules along the repair pathways, cancer susceptibility increases as mutation rates accelerate.

In addition, with each cell division the non-coding ends of chromosomes (telomeres) get shorter and shorter. Eventually, these telomeres (junk DNA) will get so short that