# The Role of Genetic Mutations in Cancer

<table>
<thead>
<tr>
<th>Primary Subject Area</th>
<th>Biology</th>
</tr>
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<tbody>
<tr>
<td><strong>Grade Level</strong></td>
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## Overview
This lesson plan will provide an introduction to cancer as a group of genetic diseases that arise due to mutations in somatic cells. We will discuss the control of the cell cycle, the loss of that control by mutation, the environmental factors that contribute to mutation, and the hereditary mutations that increase the risk of developing cancer.

## Approximate Duration
6 days

## Louisiana Framework

### Content Standards
- **Life Science**
  - The students will become aware of the characteristics and life cycles of organisms and understand their relationships to each other and to their environment.

### Science As Inquiry
- The students will do science by engaging in partial and full inquiries that are within their developmental capabilities.

### Benchmarks
- (LS-H-B1) explaining the relationship among chromosomes, DNA, genes, RNA, and proteins
- (LS-H-B2) comparing and contrasting mitosis and meiosis
- (LS-H-B3) describing the transmission of traits from parent to offspring and the influence of environmental factors on gene expression
- (SI-H-A4) formulating and revising scientific explanations and models using logic and evidence
- (LS-H-G4) exploring current research on the major diseases with regard to cause, symptoms, treatment, prevention, and cure

### Grade-Level Expectations (GLEs)
- 7. Identify the basic structure and function of nucleic acids (e.g., DNA, RNA) (LS-H-B1)
- 8. Describe the relationships among DNA, genes, chromosomes, and proteins. (LS-H-B1)
- 10. Analyze pedigrees to identify patterns of inheritance for common genetic disorders. (LS-H-B3)
- 7. Choose appropriate models to explain scientific knowledge or experimental results (e.g. computer simulations). (SI-H-A4)
- 41. Describe causes, symptoms, treatments, and preventions of major communicable and noncommunicable diseases (LS-H-G4)

## Students’ Misconceptions Addressed
- Everything causes cancer.
- You can’t control your risk for developing cancer.
- Cancer is only for old people.
- Cancers cannot be predicted.
- Cancer arises quickly and is due to a single event.
- If a family member has cancer, others in the family will also.
- Cancer in Southwest Louisiana is caused by industrial pollution and occupational exposures, so local industries must be held responsible.
Objectives

1. The student will be introduced to the nature of cancer, the relevance of cell cycle control, and the mutations which disrupt this control. Other concepts include the causes, prevalence, and heritability of cancer.

2. The student will learn that there are many types of cancer, that some people inherit predispositions to particular types of cancer, and that some people make choices that increase their risk for cancer. The student will recognize that the incidence of cancer increases with age and that a person’s chance of surviving cancer increases with early detection and treatment.

3. The student will learn that there are sophisticated molecular mechanisms for controlling cell division, including cell cycle checkpoints controlled by the products of tumor suppressor genes and growth factors controlled by the products of proto-oncogenes. The student will learn that cancer is the result of mutations in or loss of several of these genes, and therefore the loss of cell cycle control.

4. The student will learn that mutations may occur in somatic cells or germ cells, and that mutations may be spontaneous (endogenous in origin) or induced by mutagens (exogenous in origin). The student will discern the relevance of somatic and germline mutation to the development of cancer. The student will distinguish endogenous causes, which include replication errors and meiotic recombination from exogenous causes, which include chemical and physical mutagens.

5. The student will learn about the inheritance of cancer susceptibility, including those cancers caused by mutations in tumor suppressor genes, proto-oncogenes, and DNA repair genes. The student will learn that inheritance of a mutant allele is only the first event in a multistep process of cancer development. The student will learn that many of these mutations are of recent germline origin, but that they occur randomly as a result of normal biological processes.

Lesson Materials and Resources

Handouts derived from PowerPoint presentations
Activity 1 The Faces of Cancer Worksheets and Envelopes
Activity 2 Cancer and the Cell Cycle Worksheets
Guest Speaker: Dr. Joni B. Drost, Assistant Professor, McNeese State University

Technology Tools and Materials

1. Computer with LCD projector
3. PowerPoint slide shows 1. Drost-DesOrmeaux GENA Cancer Lesson Introduction.ppt
   2. Drost-DesOrmeaux GENA Cancer Lesson Cell Cycle.ppt
   3. Drost-DesOrmeaux GENA Cancer Lesson Mutation.ppt
   4. Drost-DesOrmeaux GENA Cancer Lesson Heredity.ppt

Background Information

- Students will have completed coursework on mitosis, meiosis, DNA structure and function, and protein synthesis. Students will have calculated probabilities of genotypes and phenotypes of offspring given parental genotypes in Mendelian monohybrid crosses.

Lesson Procedures

Day 1

ENGAGE - We will introduce Dr. Drost who will participate in the activities. Students will begin Activity 1 The Faces of Cancer Steps 1-3. In the activity, students participate in a role play about people who develop cancer, assemble data about the people’s experiences with cancer, and then discuss the generalizations that can be drawn from these data. This opening activity introduces cancer as a public health issue that can be systematically studied using the methods of science.

EXPLAIN - Following Activity I Steps 1-3, Dr. Drost will deliver a lecture (see the PowerPoint lecture entitled Drost-DesOrmeaux GENA Cancer Lesson Introduction.ppt). Students will be given PowerPoint handouts to accompany the lecture. A formative assessment, in the form of open-ended discussion, will be conducted.
Day 2
EXPLORE - Complete Activity 1 *The Faces of Cancer* Steps 4-18.

DAY 3
ENGAGE - Students will view the News Alerts videos (Activity 2 *Cancer and the Cell Cycle* CD-ROM, *Cell Biology* and *Cancer*) describing several historical observations suggesting causes of cancer.

EXPLORE AND EXPLAIN - Students will begin Activity 2 *Cancer and the Cell Cycle* using the CD-ROM, *Cell Biology* and *Cancer*. In the activity, students use five CD-ROM-based animations to help them construct an explanation for how cancer develops and then use their new understanding to explain the observations described in the News Alerts videos.

ELABORATE - Dr. Drost will lecture on cell cycle control (see *Drost-DesOrmeaux GENA Cancer Lesson Cell Cycle.ppt*). Students will be given PowerPoint handouts to accompany the lecture. A formative assessment, in the form of open-ended discussion, will be conducted.

Day 4
EXPLAIN – Dr. Drost will lecture on mutations (see *Drost-DesOrmeaux GENA Cancer Lesson Mutation.ppt*). Students will be given PowerPoint handouts to accompany the lecture. A formative assessment, in the form of open-ended discussion, will be conducted. Students will complete Activity 2 *Cancer and the Cell Cycle*.

Day 5
ELABORATE - Dr. Drost will lecture on the inheritance of cancer predispostions (see *Drost-DesOrmeaux GENA Cancer Lesson Heredity.ppt*). Students will be given PowerPoint handouts to accompany the lecture. A formative assessment, in the form of open-ended discussion, will be conducted. The remainder of the class period will be used for class discussion and review.

Day 6
EVALUATE - Cancer test

**Assessment Procedures**

**Formative Assessment**
Open-ended questioning and discussion throughout the lesson (see Notes Pages of PowerPoint presentations)

**Summative Assessment**
1. Activities - which contain a written assignment with short answer and discussion questions
2. Cancer test – a comprehensive test using essay and multiple choice questions

**Lesson Development Resources**

**Contact Information**
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GENA
The Role of Genetic Mutations in Cancer

Dr. Joni B. Drost
Department of Biology and Health Sciences
McNeese State University

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Introduction

What is Cancer?

• A group of genetic diseases that arise in somatic cells
  • Cancer cells share two fundamental properties:
    – Uncontrolled cell division
    – Metastasis, spread to other locations
  • Cancer cells show higher than normal rates of:
    – Mutation
    – Chromosomal abnormalities
    – Genomic instability

What Causes Cancer?

• The Cell Cycle
  • Genes Involved in Regulating the Cell Cycle
  • What Causes Cancer?
    – Mutation
      • Environmental Influences
      • Somatic Mutations
    – Genetic influences
      • Germline Mutations

Cell Cycle

Mitosis & Cytokinesis

Rates of developing invasive cancers among individuals by age group in the U.S.

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<thead>
<tr>
<th>Cancer Type</th>
<th>Gender</th>
<th>20</th>
<th>40</th>
<th>60</th>
<th>70 or over</th>
<th>80 and over</th>
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<td>1 in 12</td>
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<td>1 in 310</td>
<td>1 in 220</td>
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<td>1 in 50</td>
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</table>

Cell Cycle

Interphase

Rates of Cell Division

1. Some cells are in a resting stage and do not divide
2. Other cells are continually dividing
3. When they get worn out, they die (Apoptosis)

Environmental Agents Contribute to Human Cancers

- Includes anything that damages DNA.
- Carcinogens – chemicals, radiation, some viruses, and chronic infections.

Somatic Mutations

Environmental Influences

Predisposition to Some Cancers Can Be Inherited

- Most cancers result from somatic cell mutations, but 50 forms of hereditary cancer are known.

Genetic Influences

Germline Mutations

- Mutations in
  - Tumor Suppressor Genes
  - Proto-Oncogenes
  - DNA Repair Genes

Hereditary Cancers

<table>
<thead>
<tr>
<th>Inherited Predispositions to Cancer</th>
<th>Chromosome</th>
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</thead>
<tbody>
<tr>
<td>Early-onset familial breast cancer</td>
<td>17q</td>
</tr>
<tr>
<td>Familial adenomatous polyposis</td>
<td>5q</td>
</tr>
<tr>
<td>Familial melanoma</td>
<td>9p</td>
</tr>
<tr>
<td>Giartin syndrome</td>
<td>9q</td>
</tr>
<tr>
<td>Hidradenoma polyposis colon cancer</td>
<td>2p</td>
</tr>
<tr>
<td>Li-Fraumeni syndrome</td>
<td>17p</td>
</tr>
<tr>
<td>Multiple endocrine neoplasia, type 1</td>
<td>11q</td>
</tr>
<tr>
<td>Multiple endocrine neoplasia, type 2</td>
<td>22q</td>
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<tr>
<td>Neurofibromatosis, type 1</td>
<td>17q</td>
</tr>
<tr>
<td>Neurofibromatosis, type 2</td>
<td>22q</td>
</tr>
<tr>
<td>Retinoblastoma</td>
<td>13q</td>
</tr>
<tr>
<td>Von Hippel-Lindau syndrome</td>
<td>3p</td>
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<tr>
<td>Wilms tumor</td>
<td>11p</td>
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The Role of Genetic Mutations in Cancer

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McNeese State University

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The Cell Cycle

- Regulation of the Cell Cycle
- Cell Cycle Checkpoints
- Genes Involved in Regulating the Cell Cycle
- Apoptosis

Cell Cycle

Mitosis & Cytokinesis

- Nuclear Division in Four Phases
- Each new nucleus is genetically identical to the parent
- Each new cell is genetically identical to the parent

Rates of Cell Division

1. Some cells never divide.
   a. Permanent G_0: nerve cells, muscle cells, red blood cells
   b. Resting in G_0 and do not divide until signaled

2. Other cells are regularly dividing.

3. Apoptosis
   - When they get damaged or worn out, they die.

What Determines How Often Cells Divide?

Cells divide when
- Nutrients are plentiful
- The DNA is replicated
- Growth Factors are present
- Space is available
  - No contact with neighbors
  - Contact Inhibition

Cells stop dividing when
- Nutrients are limited
- The DNA is damaged
- Growth Factors are limited
- When they contact neighbors
  - Contact Inhibition
Characteristics of Cancer Cells

- Cancer cells are unable to enter G0 & divide continuously.
- Loss of genetic control of cell cycle
  - Mutations in Tumor Suppressor genes
  - Mutations in Proto-Oncogenes
- Loss of Contact Inhibition
- Loss of ability to undergo Apoptosis
- Abnormal migration from neighboring cells – Metastasis!

Cancer Cells Metastasize, Invading Other Tissues

- Tumors:
  - Benign
  - Malignant
- To Metastasize from the primary tumor, cancer cells separate from the extracellular matrix that holds the cells together.
- Metastasis is controlled by many genes.

Cell Cycle Control and Checkpoints

Cancer Cells Contain Genetic Defects in Cell-Cycle Regulation

- Cells halt progress through the cell cycle if there are mistakes in DNA replication, repair, or chromosome assembly.
- If DNA damage is so severe that repair is impossible, the cell may initiate Apoptosis.

Many Cancer-Causing Genes Disrupt Control of the Cell Cycle

- At the checkpoints, cells decide whether to proceed to the next stage of the cell cycle.
The products of tumor suppressor genes normally regulate cell cycle checkpoints and initiate Apoptosis. When tumor suppressor genes are mutated or inactivated, cells are unable to respond normally to cell cycle checkpoints or can't undergo apoptosis if DNA damage is extensive.

**p53**
- The p53 tumor suppressor gene encodes a nuclear protein that represses or stimulates transcription of more than 50 different genes.
  - p53 can stop the cell cycle at several phases or start apoptosis in response to DNA damage.

*Retinoblastoma Protein is a tumor suppressor protein*
- controls the G1/S cell cycle checkpoint.

**Proto-oncogenes**
- Genes whose products promote cell growth & division.
- They encode:
  - factors that stimulate expression of other genes
  - molecules that stimulate cell division
  - cell-cycle regulators that move through the cell cycle

**Oncogene**
- A mutated proto-oncogene
- Contributes to the development of cancer.
- In cancer cells, one or more proto-oncogenes have changed into oncogenes.
- Their activities cannot be controlled normally.
**GENA**

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**What Causes Cancer?**

- Environmental Influences
- Genetic Influences
  - The Role of Mutations
  - Somatic Mutations
  - Germline Mutations

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**Humans possess 23 pairs of homologous chromosomes.**

Homologous chromosomes have same size, centromere placement; genes are located in same place.

Genes on homologues may come in different versions: Alleles

Humans have:
- 22 pairs of autosomes
- 1 pair of sex chromosomes

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**Hereditary Cancers**

Joni Drost & Annette DesOrmeaux
McNeese State Univ/ Sulphur High School

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**What are Germline Mutations?**

- Inherited by offspring
- Source of variation in populations
- Mostly during meiosis

Where?
- In the Gonads

Which cells?
- Germ cells only

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**Meiosis**
Predisposition to Some Cancers Can Be Inherited

- Most cancers result from somatic cell mutations, but 50 forms of hereditary cancer are known.
- Mutations in:
  - Tumor Suppressor Genes
  - Proto-Oncogenes
  - DNA Repair Genes

Genetic Influences

Germline Mutations

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Autosomal Dominant Genetic Disorders

- Early onset lethal: usually new mutations—usually no phenotypic carriers
- Normally die before reproducing—usually no phenotypic carriers

Example: tumor suppressor syndromes, Retinoblastoma

Joni Drost & Annette DesOrmeaux
McNeese State Univ/ Sulphur High School

In Familial Retinoblastoma, a mutated RB1 allele is inherited. Sporadic Retinoblastoma requires two independent mutational events of RB1 within the same cell.

Loss or mutation of both alleles of the RB1 tumor suppressor gene contributes to cancer due to unregulated progression through the cell cycle.

Autosomal Dominant Genetic Disorders

Late onset lethal: Usually runs in families You don’t know you have it until you have already reproduced!

Hereditary Nonpolyposis Colorectal Cancer (HNPCC)

Mutations in other genes are also usually necessary to fully express the cancer phenotype.

- Familial Adenomatous Polyposis (FAP) — another colon cancer.

Chromosome Aberrations | Genes |
<table>
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<tr>
<td>5q</td>
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<td>Mutation</td>
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<tr>
<td>18q</td>
<td>Loss</td>
</tr>
<tr>
<td>17q</td>
<td>Loss</td>
</tr>
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</table>

Normal colon epithelium → Preneoplastic adenoma → Advanced adenoma → Cancerous adenoma → Colorectal cancer
• Several inherited cancers are caused by defects in genes that control DNA repair
  - hereditary nonpolyposis colorectal cancer (HNPCC)
  - chronic myelogenous leukemia (CML) (involving the Philadelphia chromosome)
  - xeroderma pigmentosum

**Nucleotide Excision Repair**
Repairs UV damage

**Xeroderma Pigmentosum**
Inherited Defects in the Genes for DNA Repair
Autosomal Recessive
Very high risk of skin cancer!

**Cancer is a Multistep Process**
• Most inherited cancer-susceptibility genes are not sufficient to trigger cancer development.
• At least one other somatic mutation in the other copy of the gene must occur to drive a cell toward tumorigenesis.

**Children of the Atomic Bomb**
Nagasaki midwives & Atomic Bomb Casualty Commission doctors.
78,000 newborns were examined in Hiroshima & Nagasaki. In 2008, blood samples of 500 heavily radiated individuals in Nagasaki & controls were immortalized—preserved & available for future studies to detect injury.
To date, no evidence of genetic injury has been found in children born to survivors of atomic bombs. All plants & animals studied have revealed radiation mutation effects.

**Germline Mutations in Humans**
• Occur in the Gonads
• Occur by Random Biological Processes
• Are Not Induced!
• But wear the lead apron anyway.
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What Causes Cancer?

**Environmental Influences**  **Genetic Influences**

**Mutations**

**Somatic Mutations**  **Germline Mutations**

Mutation: the primary source of genetic variation

- Somatic Mutations
  - Occur in any cell except germ cells
  - Not inherited by offspring
  - A cause of cancer
- Germ-line Mutations
  - Occur in germ cells and gametes
  - Inherited by offspring
  - Source of all heritable variation among individuals

Gene Mutation
- Sequence change within a gene
- Large change effecting only one or a few genes

Chromosomal Mutation
- Change in chromosome number
- Rearrangements of chromosome structure
- Genomic instability affecting large chromosomal regions

The Flow of Genetic Information
From DNA to Proteins

DNA → **transcription** → RNA → **translation** → protein
Complementary Base Pairing

<table>
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<tr>
<th>Sense DNA strand</th>
<th>Template DNA strand</th>
<th>mRNA</th>
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<th>Amino acids</th>
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<td>A</td>
<td>A</td>
<td>etc.</td>
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</tbody>
</table>

Effect of point mutations on gene expression

In coding region:
- Silent mutation
- Missense mutation (aa change)
- Nonsense mutation (new stop codon)
- Frameshift (shifts reading frame)

Physical Change to DNA sequence: Point Mutations:

- Single base substitutions
  - Small deletions/insertions
  - Frameshift mutations

Chromosomal Mutation

Genomic Instability

- Duplication
- Inversion
- Translocation
- Deletion
- Aneuploidy

Ex: Deletion
- Loss of some segment of a chromosome

Chromosomal Mutation

Genomic Instability

- Karyotype of a (male's) normal cell
- Karyotype of a (female's) cancer cell showing translocations, deletions, and aneuploidy
• Cancer is a multistep process requiring multiple mutations.
• Age-related cancer indicates that cancer develops from several mutagenic events.
• Each mutation progressively releases the cell from the normal controls.

Cancer Cells are Clones!
Cancer cells originate from a common ancestral cell with numerous accumulated somatic mutations.

Mutations that happen early affect more of your cells when you are older!

In hereditary cancers all of your cells already have the first mutation!

Spontaneous – Endogenous
Random mutations occur during normal processes

Environmental Influences
Environmental Agents Contribute to Human Cancers

Somatic Mutations

- Carcinogens
  - chemicals, radiation, some viruses, and chronic infections
  - Includes anything that damages DNA

Induced – Exogenous
Mutagen: an environmental agent that alters DNA sequence

Induced Mutations Arise from DNA Damage Caused by Chemicals and Radiation

Chemical Mutagenesis
Physical Mutagenesis
Retroviruses
Mutations Can Arise from DNA Damage Caused by Radiation

- Ultraviolet Light
- Ionizing Radiation

UV Light

Distorts DNA so that errors occur during DNA replication.

Ionizing Radiation

X rays, gamma rays, cosmic rays: mutagenic products of radioactive decay

Significant contributors to virus-induced cancers in humans

<table>
<thead>
<tr>
<th>Human Virus</th>
<th>Associated Cancer</th>
<th>Oncogenes</th>
<th>Mechanism</th>
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<tbody>
<tr>
<td>Human papillomavirus 16, 18</td>
<td>Cervical cancer</td>
<td>E6, E7</td>
<td>Inhibits p53 and p16 tumor suppressors</td>
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<tr>
<td>Hepatitis B virus</td>
<td>Liver cancer</td>
<td>HBs</td>
<td>Signal transduction, stimulates cell cycle</td>
</tr>
<tr>
<td>Epstein-Barr virus</td>
<td>Burkitt’s lymphoma, nasopharyngeal</td>
<td>Unknown</td>
<td>Unknown</td>
</tr>
<tr>
<td>Human herpesvirus 8</td>
<td>AIDS-related Kaposi’s sarcoma</td>
<td>Several possible</td>
<td>Unknown</td>
</tr>
<tr>
<td>Human T-cell leukemia virus</td>
<td>Adult T-cell leukemia</td>
<td>pX</td>
<td>Stimulates cell cycle</td>
</tr>
</tbody>
</table>

Environmental Influences

Environmental Agents Contribute to Human Cancers

- Carcinogens
  - chemicals, radiation, some viruses, and chronic infections
- Includes anything that damages DNA

Somatic Mutations
POWERPOINT SLIDE SHOW 1  CANCER LESSON INTRODUCTION

Slide 2  What is Cancer?
1. Cancer is a group of genetic diseases that arise in ________________ cells.

2. Cancer cells share two fundamental properties:
   a. ____________________ cell division   b. __________________, spreading to other locations

3. Cancer cells show higher than normal rates of ________________, chromosomal ________________, and genomic instability.

Slide 4  What Causes Cancer?
4. Two types of influences that can cause cancer are ________________________ and ________________

5. Two types of mutations are ___________________ and ________________

Slide 5
6. _______________ are involved in regulating the cell cycle.

Slide 8  Rates of Cell Division
7. Some cells are in a resting stage and do not __________________________.

8. Other cells are continually ________________________________.

9. When cells get worn out, they _______ (apoptosis).

Slide 9  Environmental Influences
10. Environmental agents contribute to human ________________.

11. Anything that damages _____________ can be an environmental influence if it causes mutations in certain genes.

12. Examples of ________________________________ (which means causing cancer) are chemicals, radiation, some viruses, and chronic infections.

Slide 10  Genetic Influences
13. ________________________________ to some cancers can be inherited.

14. Most cancers result from somatic mutations, but 50 forms of _________ cancer are known.

15. Hereditary cancers are caused by germline mutations in tumor suppressor genes, proto-oncogenes, or _________________________ genes.
POWERPOINT SLIDE SHOW 2  CANCER LESSON CELL CYCLE

Slide 3  Cell Cycle/Mitosis & Cytokinesis
16. In mitosis, each new cell is genetically __________________ to the parent.

Slide 4  Cell Cycle/Interphase
17. In the G2 part of interphase, the cell prepares for ____________________________.

Slide 5  Rates of Cell Division
18. Some cells never __________________________.
19. Other cells are regularly ____________________________.
20. When cells get damaged or worn out, they ____________________________.

Slide 6  What Determines How Often Cells Divide?
21. Cells divide when:
   a. __________________________ are plentiful.
   b. The __________________________ is replicated.
   c. __________________________ factors are present.
   d. __________________________ is available.
22. Cells stop dividing when:
   a. __________________________ are limited.
   b. the __________________________ is damaged.
   c. __________________________ factors are limited.
   d. they ____________________________, called contact inhibition.

Slide 7 Characteristics of Cancer Cells
23. Cancer cells __________________________ continuously.
24. Cancer cells undergo a loss of genetic control of the __________________________.
25. Cancer cells have a loss of __________________________ inhibition.
26. Cancer cells have a loss of ability to undergo __________________________.
27. Cancer cells have an abnormal migration from neighboring cells called __________________________.
3. Cancer Cells Metastasize, Invading Other Tissues
28. Tumors may be ___________________, not cancerous, or ____________________, cancerous.

4. Cell Cycle Control and Checkpoints
29. Cancer cells contain genetic defects in ____________________________ regulation.

5. Cells halt progress through the _____________________ if there are mistakes in DNA replication, repair, or chromosome assembly.
31. If DNA damage is so severe that repair is impossible, the cell may initiate _______________________________________.

6. Tumor Suppressor Genes
32. The products of _______________________________________ genes normally regulate cell cycle checkpoints and initiate apoptosis.

7. Proto-oncogenes
33. Proto-oncogenes are genes whose products promote cell growth and ____________________.

8. Oncogene
34. An oncogene is a ____________________________ proto-oncogene that contributes to the development of cancer.
POWERPOINT SLIDE SHOW 3  CANCER LESSON MUTATION

Slide 3
35. ________________________: the primary source of genetic variation.

Slide 4
36. A ________________ mutation is a sequence change within a gene.
37. A ________________ mutation can be a change in chromosome number.

Slide 5
38. A gene ______________________ is an alteration in DNA sequence.

Slide 9
39. Point mutations are ________________ base substitutions.

Slide 11
40. One example of a chromosomal mutation is a ________________, which is the loss of some segment of a chromosome.

Slide 13
41. Cancer is a multistep process requiring multiple ____________________________.

Slide 14
42. Cancer cells are __________________________.

Slide 15
43. In hereditary cancers all of your cells already have the first ________________.

Slide 16
44. __________________________ mutations are random and usually linked to normal biological or chemical processes in the organism.

Slide 17
45. Environmental agents that cause cancer are called ____________________________.

Slide 18
46. __________________________ mutations arise from DNA damage caused by chemicals and radiation.

Slide 19
47. Mutations can arise from DNA damage caused by ____________________________.

Slide 20
48. UV light distorts DNA so that errors occur during DNA ____________________.

Slide 21
49. X rays, gamma rays, and cosmic rays are all types of _____________________ radiation.
POWERPOINT SLIDE SHOW 4  CANCER LESSON HEREDITY

*Slide 3*
50. Humans have _____ pairs of autosomes and _____ pair of sex chromosomes.

*Slide 4*
51. Germline mutations occur mostly during ____________________.
52. Germline mutations occur in the __________________________, either ovaries or testes.

*Slide 7*
53. Predispositions to some cancers can be ____________________.

*Slide 9*
54. An example of an autosomal dominant genetic disorder is the tumor suppressor syndrome, ____________________________.

*Slide 11*
55. Autosomal dominant genetic disorders like HNPCC, that exhibit late onset, usually ____________________________ because you don’t know you have it until ____________________________.

*Slide 13*
56. Several inherited cancers are caused by defects in ________________ that control DNA repair.

*Slide 15*
57. Xeroderma pigmentosum results in a very high risk of ____________________.

*Slide 16*
58. Cancer is a ____________________________ process.

*Slide 17*
59. To date, no clear cut evidence of genetic injury has been found in children born to survivors of the ____________________________ at Nagasaki and Hiroshima.

*Slide 18*
60. Germline mutations in humans are not induced, but wear the _______________________ anyway.
CHAPTER 11 TEST
THE ROLE OF GENETIC MUTATIONS IN CANCER

Fill in the blanks with the correct answers. Write all answers on the answer sheet.

1. Cancer cells share two fundamental properties:
   a. ___ cell division
   b. ___ spreading to other locations

2. Two types of influences that can cause cancer are ___ and ___.

3. Two types of mutations are ___ and ___.

4. When cells get worn out, they ___ (apoptosis).

5. Examples of ___ (which means causing cancer) are chemicals, radiation, some viruses, and chronic infections.

6. Cells divide when:
   a. ___ are plentiful.
   b. The ___ is replicated.
   c. ___ factors are present.
   d. ___ is available.

7. Cells stop dividing when:
   a. ___ are limited.
   b. the ___ is damaged.
   c. ___ factors are limited.
   d. they ___, called contact inhibition.


9. Cancer cells have an abnormal migration from neighboring cells called ___.

10. The products of ___ genes normally regulate cell cycle checkpoints and initiate apoptosis.

11. Proto-oncogenes are genes whose products promote cell growth and ___.

12. A ___ mutation is a sequence change within a gene.

13. A ___ mutation can be a change in chromosome number.

14. Point mutations are ___ base substitutions.

15. Cancer is a multistep process requiring multiple ___.

16. UV light distorts DNA so that errors occur during DNA ___.
CHAPTER 11 TEST
THE ROLE OF GENETIC MUTATIONS IN CANCER

17. Humans have ___ pair(s) of autosomes and ___ pair(s) of sex chromosomes.

18. Germline mutations occur mostly during ___.

19. Cancer is a ___ process.

20. To date, no clear cut evidence of genetic injury has been found in children born to survivors of the ___ at Nagasaki and Hiroshima.

WORD BANK

Not all terms are used. Some terms may be used more than once.

1,22 division multistep
22,1 DNA mutations
46, 23 environmental nutrients
abnormalities influences replication
atomic bombs gene single
carcinogens genetic influences somatic
cell cycle germline space
chromosomal growth factors tumor suppressor
contact neighbors meiosis uncontrolled
deletion metastasis
die mitosis
CHAPTER 11 TEST
THE ROLE OF GENETIC MUTATIONS IN CANCER

ANSWER KEY

1. a. uncontrolled
   b. metastasis

2. environmental, genetic

3. somatic, germline

4. die

5. carcinogens

6. a. nutrients
   b. DNA
   c. growth
   d. space

7. a. nutrients
   b. DNA
   c. growth
   d. contact neighbors

8. divide (unable to enter G₀)

9. metastasis

10. tumor suppressor

11. division

12. gene

13. chromosomal

14. single

15. mutations

16. replication

17. 22, 1

18. meiosis

19. multistep

20. atomic bombs
Reflections on a Lesson Plan

We were concerned with students who will not be seeking higher education. During the high school years many students decide to begin work as soon as they graduate. Our population is largely rural but employment in the petrochemical industry is available for many young graduates. While McNeese State University is located just across the Calcasieu River, most will not enroll, at least not right away. Within a few years many begin families and take on new responsibilities. Our interest is in providing these individuals with a basic understanding of cancer, which occurs at a very high rate in our area. We have a history of industrial water and air pollution to which health problems are often attributed. These issues are still alive and well in our community today although most industrial discharges are currently regulated (Google “Mossville class action suit pending”). The working class men and women of our area are concerned but have limited biological knowledge. Our hope is that through our lesson plan, an appreciation of some of the causes of cancer will disseminate into the population to comfort and empower our people.

We chose to use Activity 1 The Faces of Cancer and Activity 2 Cancer and the Cell Cycle, National Cancer Institute that was available at the GENA Workshop. We augmented those with PowerPoint lectures. The combination of these worked well to form an integrated lesson plan.

Annette teaches two different biology courses, 10th grade Biology I and 12th grade Biology II college prep. We chose to develop this lesson for the 10th grade class. Although they are less advanced in biology background and less mature in study habits, this course is required for all students. We also considered that more advanced students will have other opportunities to learn this information. In short, we decided to offer this to those most in need.

When Joni arrived to present her lectures, the students were visibly excited that we had done all this just for them. We found the activities easy to implement with few modifications. Annette had prepared the students with previous lessons so that we didn’t have to explain all the fundamentals of genetics. In fact, the students seemed to soak the information up. After the last lecture, the students were given the cancer test with very good results. Annette allowed the students to use their notes because, as noted above, many of these students are not avid learners. This allowed them to relax and focus and recall what they had learned.

We put a lot of work into this lesson plan, working many hours on end over the late summer, early fall, and then more in January before the lessons, and then even more to make careful revisions before submitting this final version. Both of us are happy with the product. We have developed a good working relationship and friendship thanks to the GENA project and given a gift to the people of Southwest Louisiana.