
EVOLVED

CREDITS / ABOUT THE AUTHOR

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One iteration of the implementation of the materials in this teacher guide, along with student outcomes, is described:

> **Filice, D. C., Riedy, J. J., Heidemann, M. K., Smith, J. J., & White, P. J. (2023). Evaluating introductory biology student perceptions surrounding the use of integrative cases related to human health for evolution education. *Evolution: Education and Outreach*, *16*(1), 6.**

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Why Use Breast Cancer to Teach Introductory Cell & Molecular Biology?

Introductory cell and molecular biology can feel like a disjointed tour of isolated processes: mitosis on Monday, DNA replication on Wednesday, central dogma on Friday. Many students find learning more meaningful when those processes are woven into a single interconnected narrative. The study of cancer provides such an opportunity. Cancer is the ultimate story of cellular evolution; rogue cells that gain a selective advantage, through mutation, that allow them to outcompete their neighbors and proliferate. While cancer can be a difficult subject matter, particularly for those whose lives have been touched by it, many students – regardless of their

personal experience with the disease – are eager to learn about it. In fact, the personal relevance of cancer seems to motivate students and answers the question of "*WHY"* they should learn biology (Filice et al., 2023).

Why *breast* cancer? While cancer is a universally recognized health issue, roughly one in eight women will be diagnosed with breast cancer at some point in their lifetime, and it ranks among the top cancers affecting all genders worldwide.

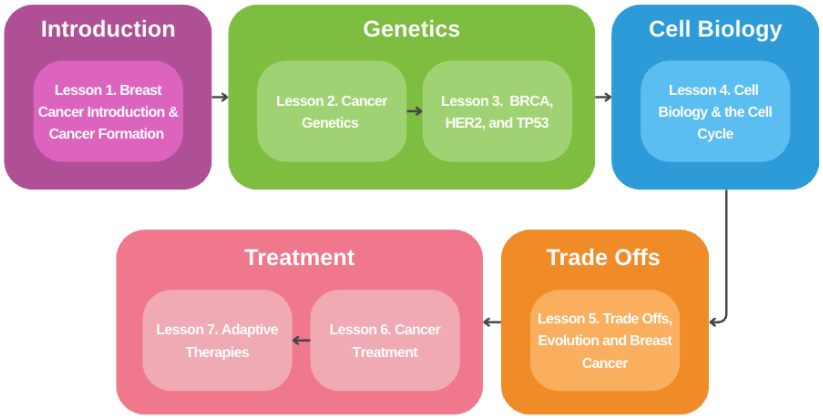
How does cancer connect with key disciplinary concepts? To understand cancer, students need to master the structure and function of DNA, the nature of mutations, transcription and translation regulation, protein structure and function, cell cycles, natural selection and more! Once students start to think of cancer cells as "individuals", it makes it easier to think about ecological processes in a microbiological world, supporting a more holistic understanding of biology across scales, from nucleotides to populations.

Breast cancer occurrence and treatment isn't best understood by memorizing pathways; it requires systems thinking. Students can evaluate data, hypothesize how a single nucleotide change cascades to macroscopic disease, and debate ethical dilemmas around genetic testing. These are the types of *core competencies* and *science and engineering practices* that we find in *Vision & Change* for higher ed (AAAS, 2010) and in the *NGSS* for upper secondary or AP courses (NGSS Lead States, 2013).

Ultimately, the study of cancer transforms introductory cell and molecular biology from a fragmented checklist of processes into a cohesive, life-relevant exploration of how molecules, cells, and evolutionary forces intertwine to shape human health. It captivates students' curiosity, grounds abstract mechanisms in personal experience, and nurtures the analytical mindset students will need as scientists, healthcare professionals, or scientifically literate citizens.

How This Guide is Organized

- This teacher guide is structured as a series of five **thematic sections** that constitute the overall **breast cancer unit**.
- Each **thematic section** has 1 or 2 **class sessions**. Each class session can take 60-90 minutes, depending on the pacing, framing, and instructor preference.
- In total, this results in 7-10 hours of in-class instructional time.
- While the thematic sections are listed in a specific order, they can be implemented in a myriad of different ways, depending on instructor preference.



Pedagogical Note

The class sessions within this teacher guide were designed to be implemented with a **flipped teaching pedagogy**. Students prepare for class by watching a short tailored lecture video, at home, on the upcoming subject material. Each video was written and recorded *specifically* with this teacher guide in mind.

These videos are freely and publicly available on YouTube at:
https://www.youtube.com/@evo-ed

One of the **risks** of this **flipped teaching pedagogy** is that a subset of students may undertake only a cursory viewing of the pre-class video and thus be unprepared to engage in deeper learning opportunities that the pedagogy is designed for. To avoid this, students can be assigned homework to complete *along with the video viewing*. There are many ways this can be achieved. For example, you might require that students compose summary notes of the lecture videos (but consider Muller & Oppenheimer, 2014), or start class with a “minute paper” (Chizmar & Ostrovsky, 1998) or “muddiest point” exercise (Carberry et al, 2013). Whatever method is used, students should be encouraged – and, when appropriate, held accountable for – individual synthesis and sensemaking, prior to coming to class.

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LESSON PLAN STRUCTURE

In this teacher guide, each lesson within the EvoEd Breast Cancer Unit follows a four-step process: (1) **a pre-class video**, (2) **an in-class quiz**, (3) **a mini-lecture**, and (4) **a worksheet**.

STEP 1: PRE-CLASS VIDEO

[Students complete asynchronously]

Before coming to class, students watch one or two short videos that introduce them to the core ideas and key content of the upcoming lesson. This gives them an opportunity to familiarize themselves with the fundamental terminology, core concepts, and key processes at their own pace, setting the stage for deeper in-class learning.

STEP 2: IN-CLASS QUIZ

[5-10 minutes]

Each class session begins with a short in-class quiz to hold students accountable for their classroom preparation, each day. Sample quizzes are provided, but teachers are encouraged to create their own questions if desired, and use whatever style of quiz best suits their teaching style.

STEP 3: MINI "RECAP" LECTURE

[<10 minutes]

Following the quiz, instructors can deliver a very short interactive-engagement-style "recap lecture". This serves two purposes. First, it allows students who were unprepared or underprepared to gain *some* familiarity with the subject material prior to the problem-solving stage. Second, it allows the instructor to introduce new and *emergent* ideas to the discussion, or to connect the content of the day with student prior-learning. While this guide provides some options for generic review slides, instructors can customize the "recap-lecture" portion to best meet the needs of their course.

Since passive listening is not as effective for learning, keep this recap lecture short, concise and interactive. Student engagement can be boosted using interactive elements such as:

- **"Minute Paper" Activity:** At the end of the lecture, pose a single question related to the content and give students one minute to write their answer. Collecting these minute papers offers a quick insight into student comprehension and highlights areas where further clarification may be necessary.
- **"Muddiest Point" Reflection:** Ask students to identify their "muddiest point" – the concept they find most confusing about the lesson – on a small sheet of paper. Collect these papers and address the common questions before moving on to problem solving.
- **"Think-Pair-Share" Activity:** Pose one or more challenging questions to the students during the recap lecture that connect to the content. Ask them *first* to think on their own about a suitable answer/solution, *second* to discuss their thoughts with a shoulder partner, *third* to be prepared to share some of the ideas that came up in discussion with the class if called upon.

STEP 4: WORKSHEET & PROBLEM SOLVING

[~45-60 minutes]

For the remainder of the class session, students work on a lesson-specific worksheet. In some formats, students may benefit from *first* having an opportunity to work on the worksheet individually before working with a partner or in small groups. During this stage, instructors can circulate around the room and actively engage with students to probe their thinking, foster discussion, and answer questions that arise. At the end of class, instructors can choose to assess their worksheets for completeness and/or correctness, and can allow students to keep the worksheets to use as study aids if appropriate.

What about Answer Keys?

This Teacher Guide does not include answer keys for the worksheets. Instead, students can be encouraged to use the class time to work with their peers, or engage with the instructor(s) about any questions they are unsure about. In today's day and age, students have a myriad of online tools and resources they can use to check factual information. Furthermore, depending on the class session, the course instructor may take some time near the end of the allotted time to go over one or more of the worksheet questions if they observe that students are struggling with a particular concept.

End-of-Class Checkout.

Instructors may want to set up a system where students are required to demonstrate their worksheet progress at the end of class. This guards against students simply taking the assignment "to-go". One of the biggest values with the flipped-approach to teaching and learning is that students are afforded time in-class to work together, and to consult with the instructor(s) about challenging course material. If/when students take their copy of the worksheet and then leave class to "work on it at home (alone)" –or worse, get the answers from a friend– they miss out on valuable learning time.

CLASS SESSIONS

SESSION 1	BREAST CANCER INTRODUCTION
Description	This class session establishes a foundation for understanding cancer. Students work from a simple definition of cancer to an exploration of how cancer progresses from a small collection of cells, to a hyperplasia growth to a malignant tumor. Since this curricular unit focuses on cancer of the breast, students also learn the basic anatomy of the breast. Most breast cancers are due to mutations in fast growing duct cells (due to hormone levels), resulting in a type of cancer called invasive ductal carcinoma.
Estimated Time	60-90 minutes
Learning Outcomes	By the end of this lesson, students should be able to: 1) Define cancer and distinguish between hyperplasia, dysplasia, benign tumors, malignant tumors, and metastasis. 2) Explain how the balance between mitosis and apoptosis maintains tissue homeostasis and how disruption of this balance contributes to cancer. 3) Illustrate the anatomical structure of the breast and identify why ductal epithelial cells are especially susceptible to tumor formation. 4) Compare tissues with high vs. low cellular turnover to predict their relative susceptibility to cancer. 5) Assess how cancer can be understood across multiple biological scales (genes, cells, tissues, organs, organisms) and discuss the explanatory value of each perspective.
Considerations	● Students may have varying levels of prior knowledge about cell biology and cancer. The mini-lecture and worksheet are adaptable to address these differences. ● Discussions about cancer can be sensitive for some students.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** An Introduction to Breast Cancer
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Breast Cancer Intro Quiz Intro Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Sample Breast Cancer Introduction Slideshow: **Points of Emphasis:** ● Cancer is a phenomenon of uncontrolled cell division ● Cancer occurs when there is an equilibrium imbalance between mitosis and apoptosis. ● Invasive ductal carcinoma is the most common type of breast cancer due to frequent hormone-linked cell growth signals in duct cells.

	Sample Discussion Questions: ● “Explain, in your own words, why mitosis and apoptosis are both important for healthy tissue and organ function.” ● “Mutations in DNA can lead to uncontrolled cell growth. Why might this be the case?” [*Here, link central dogma with mutation and cell growth*] ● “Why might frequent cell division increase the potential for mutations to arise?” ● "If you were to explain the core idea of cancer to someone who knew nothing about biology, what would be the one key thing you'd want them to understand?"
~40 minutes	**STEP 4: WORKSHEET** Introduction to Breast Cancer Worksheet For the rest of the class, students work on worksheet problems.

SESSION 2	CANCER GENETICS 1: INTRO & TP53
Description	This class session is the first of two that explore the genetics of breast cancer. First, students develop an operational understanding of *proto-oncogenes* and *tumor suppressor genes.* In Part I, students investigate the role of TP53 as one of the most important tumor suppressor genes in animals.
Estimated Time	60-90 minutes
Learning Outcomes	By the end of this class session, students should be able to: 1) Define proto-oncogenes and tumor suppressor genes and explain their contrasting roles in regulating cell division. 2) Describe the normal function of TP53 in maintaining DNA fidelity and initiating repair or apoptosis after damage. 3) Trace how mutations in TP53 alter transcription/translation of the p53 protein and compromise its tumor-suppressive functions. 4) Compare the consequences of mutations in proto-oncogenes versus tumor suppressor genes, predicting how each contributes to tumor development. 5) Assess the importance of TP53 mutations by linking molecular-level changes in DNA and protein structure to cellular outcomes and organismal cancer risk.
Considerations	● The Central Dogma of Molecular Biology at the heart of this class session. However, instead of learning the pathway of mutations-to-protein product in isolation, they learn in the context of cancer-relevant genes and alleles.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** Breast Cancer Genetics (an Overview) Breast Cancer and the TP53 Gene
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Genetics and TP53 Quiz Genetics and TP53 Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Genetics Slideshow AND/OR Breast Cancer and TP53 Slides **Points of Emphasis:** ● There is no single “cancer gene,” but rather a collection of genes whose mutated alleles can increase the probability of developing cancer. ● DNA is the foundational material from which proteins are made; proteins determine traits/phenotypes.

	• tp53 and its associated protein are critical for high-fidelity DNA replication. **Sample Discussion Questions:** • “What is the function of a proto-oncogene’s protein product in a healthy cell?” • “How does a proto-oncogene become an oncogene?” • "The mutations in a gene's promoter sequence are often the cause of overexpression. Why would that kind of mutation lead to more protein being made, even if the coding sequence is unaltered?" • "How does the P53 protein act as a 'guardian of the genome'?" • "If a tumor suppressor gene like TP53 becomes mutated, how does that impact the cell's ability to prevent cancer?"
~40 minutes	**STEP 4: WORKSHEET** Cancer Genetics and TP53 Worksheet For the rest of the class, students work on worksheet problems.

SESSION 3	CANCER GENETICS 2: BRCA & HER2
Description	This lesson is the second of a two-part lesson sequence that explores the genetics of breast cancer. In Part I, students developed an understanding of *proto-oncogenes* and *tumor suppressor genes*. In this lesson, students learn about the BRCA tumor suppressor genes, and the HER2 proto-oncogene.
Estimated Time	60-90 minutes
Learning Outcomes	By the end of this lesson, students will be able to: 1) Describe the role of the HER2 protein, and various scenarios where HER2 mutations can increase cancer rates. 2) Describe the role of the BRCA2 protein, and various scenarios where BRCA2 mutations can increase cancer rates. 3) Explain how population bottlenecks and founder effects are responsible for some of the biogeographical patterns in cancer occurrence we see around the world.
Considerations	● Entering this class session, students should *already* have a fundamental understanding of DNA, RNA, and insertion/deletion/frameshift mutations. ● Be prepared for sensitive discussions around cancer risk, personal genetics, and gender identity as prompted by the worksheet questions. Encourage respectful dialogue and that genetic predisposition does not guarantee cancer development.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEOS** How do the BRCA Genes Cause Breast Cancer? Breast Cancer and the HER2 Gene
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** BRCA & HER2 Quiz BRCA & HER2 Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Genetics Slideshow AND/OR BRCA & HER2 Slides **Points of Emphasis:** ● HER2 codes for a protein receptor on cell membranes. Explain how an overexpression (too much) of protein, and/or a mutation causing a more effective protein can lead to increased cell growth rates. ● Discuss the BBCA2 999del5 mutation and its impact on the protein. Prompt discussion on why this variant is geographically

	concentrated in Iceland and Scandinavian countries. **Sample Discussion Questions:** ● "How does HER2 overexpression lead to cancer?" ● "The slides showed how a single nucleotide change can alter an amino acid in the HER2 protein. How can such a small change lead to a significant increase in cancer risk?" ● "If someone has a pathogenic mutation in BRCA1 or BRCA2, does it mean they will definitely get cancer? Why or why not?" ● "The BRCA2 999del5 mutation is a 'frameshift mutation'. What are the possible consequences of this kind of mutation when thinking about the structure and function of the resulting protein?" ● "The 999del5 BRCA2 mutation is mostly found in Iceland and Scandinavian countries. What ecological processes might explain this geographical pattern?" (Prompt for population bottlenecks or founder effects if students don't get there directly). ● "What other factors, besides genetics, contribute to someone's overall cancer risk?"
~40 minutes	**STEP 4: WORKSHEET** BRCA and HER2 Worksheet For the rest of the class, students work on worksheet problems.

SESSION 4	CELL BIOLOGY & THE CELL CYCLE
Description	This lesson provides an overview of the eukaryotic cell cycle, emphasizing checkpoints and the roles that key proteins like HER2, P53, and BRCA play in regulating cell division and maintaining genomic integrity. Students will learn how dysregulation of these processes can lead to uncontrolled cell proliferation and cancer.
Estimated Time	60-90 minutes
Learning Outcomes	By the end of this class session, students will be able to: 1) Describe the major phases of the cell cycle and the role of checkpoints in regulating cell division. 2) Explain how TP53 and BRCA proteins function in relation to cell-cycle progression, DNA repair, and apoptosis. 3) Apply knowledge of gene function to predict whether mutations would act dominantly or recessively when inherited. 4) Evaluate how disruptions in checkpoint regulation can escalate from molecular defects to uncontrolled cellular growth.
Considerations	The M phase details (prophase, metaphase, anaphase, telophase) can be covered at varying depths depending on student level, focusing on the main outcome of chromosome segregation.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** Breast Cancer Cell Biology
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Cell Biology Quiz Cell Biology Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Cell Biology Slideshow AND/OR Cell Biology Alt Slides **Points of Emphasis:** ● G2 Phase: Describe this as a second growth phase with checkpoints to verify accurate DNA replication. Introduce the crucial roles of TP53 and BRCA proteins here, where they assist in DNA repair or initiate apoptosis for severely damaged cells. Emphasize that faulty TP53 or BRCA can allow mutated cells to proceed to mitosis. ● Mention BRCA proteins also assist in the M checkpoint by verifying chromosome alignment. ● Reinforce the importance of accurate DNA copying and repair throughout the cycle, even with high copying accuracy, errors still

	occur. **Sample Discussion Questions:** ● "Why is the cell cycle represented as a cycle? What does that imply about cell division?" ● "Even though DNA replication is highly accurate (better than 99.999%), errors still occur. What could be the consequences of these rare errors if they aren't caught?" ● "What are some of the 'checkpoints' a cell must pass in G1 before it can move forward?" ● "The slides mention that after G1, the cell is 'irreversibly committed' to cell division. What does that mean for the cell?" ● "How do tumor suppressor genes like TP53 and BRCA come into play in the G2 phase?"
~40 minutes	**STEP 4: WORKSHEET** Cell Biology Worksheet For the rest of the class, students work on worksheet problems.

SESSION 5	TRADEOFFS & EVOLUTION
Description	This lesson explores the evolutionary concept of antagonistic pleiotropy as a potential explanation for the persistence of cancer, particularly breast cancer, in human populations. Students will learn what antagonistic pleiotropy is, examine evidence for and against its role in BRCA alleles, and consider other evolutionary scenarios like population bottlenecks, kin selection, and mutation load that might contribute to cancer's maintenance.
Estimated Time	60-90 minutes
Learning Outcomes	By the end of this class session, students will be able to: 1) Describe the concept of antagonistic pleiotropy and provide an example as it relates to breast cancer. 2) Outline some of the evidence for- and against- the case for pathogenic BRCA alleles being an example of an antagonistic pleiotropy.
Considerations	● Younger students may require guidance in using Google Scholar effectively for their research. ● The concept that cancer cannot be "cured" from an evolutionary perspective can be a profound idea; allow time for reflection and discussion.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** Breast Cancer and Trade Offs
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Trade Offs Quiz Trade Offs Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Trade Offs Slideshow or Trade Offs Alt Slides **Points of Emphasis:** ● Defining Antagonistic Pleiotropy: Breaking down the etymology of the word may help students understand / remember what it means (i.e., hostile – many/transformations). In these situations an allele can have a beneficial impact early in life (e.g., enhancing reproductive fitness) but a negative impact later in life (e.g., increase in disease susceptibility). ● Introduce the idea that cancer persists partly due to these evolutionary trade-offs. **Sample Discussion Questions:**

	• "How does the example of Sickle Cell Anemia illustrate antagonistic pleiotropy? What's the 'good' part of the trait, and what's the 'bad' part?" • "What challenges do scientists face when trying to study antagonistic pleiotropy in human populations?" • "If a trait has both benefits and costs, how might that explain why a 'bad' trait (like increased cancer risk) persists in a population over evolutionary time?"
~40 minutes	**STEP 4: WORKSHEET** Trade Offs Worksheet For the rest of the class, students work on worksheet problems.

SESSION 6	CANCER TREATMENT
Description	This lesson introduces students to the three key traditional methods of cancer treatment: surgery, radiation, and chemotherapy. It emphasizes why the complete elimination of cancer cells is crucial to prevent recurrence and how natural selection can lead to the emergence of highly resistant tumors after treatment. Hormone therapy is also introduced as a method to limit cell division.
Estimated Time	~60-90 minutes
Learning Outcomes	By the end of this class session, students will be able to: 1) Explain the three main types of (breast) cancer treatment, including how they work on a molecular level (if applicable). 2) Describe how mutations + natural selection can lead to the emergence of highly-resistant cancerous cells.
Considerations	Guiding students through the GenBank website for ESR1 will require explicit instructions or demonstration.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** Breast Cancer Treatment
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Treatment Quiz Treatment Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Cancer Treatment Slideshow AND/OR Treatment Slides **Points of Emphasis:** ● Combination Therapy: Explain why multiple treatments are often applied concurrently – the "absolute essential" need to kill every single cancer cell. ● Hormone Therapy (e.g., Tamoxifen): Explain its role in limiting cell division rather than killing cells. Use the ESR1 receptor and estrogen as examples, showing how tamoxifen competes for ESR1 binding to prevent cell division stimulation. **Sample Discussion Questions:** ● "Why is knowing the 'stage' of cancer so important for deciding on a treatment plan?" ● "What is the most direct way surgery 'treats' cancer? What are some situations where surgery might be difficult or be a potentially unwise path to take?" ● "Why are these three treatments often used together?"

	• "The slides state it's 'absolutely essential that every single cancer cell is killed.' Why is this so?" • "What is ESR1, and what role does it play in stimulating cell division in the presence of estrogen?"
~40 minutes	**STEP 4: WORKSHEET** Cancer Treatment Worksheet For the rest of the class, students work on worksheet problems.

SESSION 7	ADAPTIVE THERAPIES
Description	This lesson focuses on the evolutionary dynamics of cancer recurrence and introduces the concept of adaptive therapy as an alternative treatment strategy. Students explore how adaptive therapy aims to manage cancer by maintaining a population of drug-sensitive cells that suppress the growth of more resistant cells.
Estimated Time	~60 minutes
Learning Outcomes	By the end of this class session, students will be able to: 1) Describe how adaptive cancer treatment differs from traditional cancer treatment. 2) Analyze the pros and cons of adaptive versus traditional cancer treatment.
TIME	**STEPS**
Asynchronous	**STEP 1: PRE-CLASS VIDEO** Breast Cancer Adaptive Therapy
5-10 minutes	**STEP 2: PRE-CLASS QUIZ** Adaptive Therapies Quiz Adaptive Therapies Quiz KEY
~10 minutes	**STEP 3: MINI LECTURE** Using Cancer Treatment Slideshow AND/OR Treatment Slides **Points of Emphasis:** ● Contrast adaptive therapy with traditional methods. Instead of aiming to kill all cells, adaptive therapy aims to manage the cancer to prevent it from growing and spreading. ● Explain how it works by keeping highly resistant cancer cells "in check" by allowing other, less resistant cells to remain, creating competition that prevents the resistant cells from dominating the tumor. **Sample Discussion Questions:** ● "Why do recurring cancers tend to be 'highly-resistant' to the original treatments?" ● "Can someone explain how the primary goal of adaptive therapy fundamentally differs from that of traditional cancer treatment?" ● "How does adaptive therapy leverage this competition between different cancer cell types?"
~40 minutes	**STEP 4: WORKSHEET** Adaptive Therapy Worksheet

	For the rest of the class, students work on worksheet problems.

Appendix of Resources

SESSION	RESOURCE
1	Video: An Introduction to Breast Cancer
1	Breast Cancer Intro Quiz
1	KEY: Breast Cancer Intro Quiz
1	Cancer Formation Slideshow
1	Introduction to Breast Cancer Worksheet
2	Video: Breast Cancer Genetics (an Overview)
2	Video: Breast Cancer and the TP53 Gene
2	Genetics and TP53 Quiz
2	KEY: Genetics and TP53 Quiz
2	Genetics Slideshow
2	TP53 Slideshow
2	Cancer Genetics and TP53 Worksheet
3	Video: How do the BRCA Genes Cause Breast Cancer?
3	Video: Breast Cancer and the HER2 Gene
3	BRCA & HER2 Quiz
3	KEY: BRCA & HER2 Quiz
3	Genetics Slideshow
3	BRCA & HER2 Slideshow
3	BRCA & HER2 Worksheet
4	Video: Breast Cancer Cell Biology
4	Cell Biology Quiz
4	KEY: Cell Biology Quiz
4	Cell Biology Slideshow

4	Cell Bio Alt Slides
4	Cell Biology Worksheet
5	Video: Breast Cancer and Trade Offs
5	Trade Offs Quiz
5	KEY: Trade Offs Quiz
5	Trade Offs Slideshow
5	Trade Offs Alt Slides
5	Trade Offs Worksheet
6	Video: Breast Cancer Treatment
6	Treatment Quiz
6	KEY: Treatment Quiz
6	Cancer Treatment Slideshow
6	Treatments Alt Slides
6	Cancer Treatment Worksheet
7	Video: Breast Cancer Adaptive Therapy
7	Adaptive Therapies Quiz
7	KEY: Adaptive Therapies Quiz
7	Cancer Treatments Slideshow
7	Treatments Alt Slides
7	Adaptive Therapy Worksheet

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