Henrietta Lacks Lesson

Volume 4

Cancer Treatments from Henrietta Lacks to Today

National Human Genome Research Institute

The Forefront of Genomics®

Cover

Portrait of Henrietta Lacks by Kadir Nelson/Smithsonian

Foreword

What we say is that she was good during her living days and she's still good in her dying days. My grandmother may have passed, but she's still helping people. That's the kind of person that she was. But, for many years, we knew very little about her. We had a picture of a good looking young woman, well dressed and beaming, but we were missing the stories to make her real. It was only with the publication of The Immortal Life of Henrietta Lacks that I really met my grandmother. I learned about my grandmother at the same time that I learned about the HeLa cells and the controversy that surrounded them. With all the questions that the HeLa cells created, and the confusing and painful relationship that the family had with researchers who would come to take samples and leave empty promises to bring back answers, in a way, without them, I would still not know my grandmother.

Henrietta always made sure that everybody was taken care of and her story hasn't ended. She continues to contribute to the world through her cells and we, her kids and grandkids, are doing what we can to walk beside her. We keep her story alive, so others can remember and learn from it, and we use this story as a platform to advocate for a diverse collection of other important issues, such as patient rights, consent, disease prevention, and health disparities. My brother goes around talking about the importance of participating in clinical trials. There remains so much distrust in the African American community toward the medical establishment, but being represented in these studies is an important step in making sure that medical breakthroughs also work for us. My cousin has the Henrietta Lacks House of Healing, a place to help previously incarcerated men and women successfully transition back into the community. It's important to us to bring awareness and to make sure that everyone is given the opportunity to take care of their health.

At the root of it, this is an issue of ownership and control over your own information. My brother is involved with the HeLa Genome Data Access Working Group, which reviews requests by research groups seeking access to the HeLa genome. It is important to him that the family have a say in how this resource, our grandmother, is used by others (universities, government laboratories, companies, etc.). For me, it's more about the personal side. I tell my grandmother's story and try to help where I can, where it comes to educating the public about health disparities and disease prevention. Our experience has made me more vigilant: when I go to the doctor, or when I take my mother to the doctor, I'm much more proactive about asking questions, looking up information about the treatment they recommend, making sure that things are not overlooked. These practices are steps that we can all take to protect our bodies and our health.

When I think about Henrietta's story and legacy, two words come to mind: "hope" and "everlasting". When I see all the ways that the HeLa cells have helped people, through discoveries that have made in vitro fertilization possible, or vaccines, or cancer treatments, I see hope. Hope for people and hope for the future. An important part of this story is compassion. When I tell Henrietta's story, I want people to remember that this was an African American woman with limited education and limited income. The difference she has made is tremendous, but the way she and her family were treated was regrettable. I hope students realize that this could have happened to anyone – their parent, their grandparent, themselves. We should show compassion to the people we meet and the people around us. Henrietta didn't choose this, but this is all part of her story now. I hope she never stops doing good and helping people. That is who she is. This is my grandmother.

by Jeri Lacks-Whye

Curriculum Introduction

In 1951, a young woman sparked a scientific revolution. Unfortunately, she would neither know about it nor benefit from it. For many decades, we would not even know her name. The life of this young woman, Henrietta Lacks, was cut short by the ravages of a rapidly advancing cancer colonizing her body. As the cancer was quickly killing Henrietta, a piece of tumor was isolated and grown in a test tube. As it happened, the traits that made the cells so deadly within the body, also granted them the unusual ability to grow unencumbered under artificial laboratory conditions; a scientific Holy Grail at the time. And so, a new world of scientific tools was suddenly available to researchers. However, the life of Henrietta Lacks is part of a larger and more complex story. Despite the fact that her disease was finally subverted to great scientific benefit, her story is not confined to this terrible and terminal experience.

In many ways, Henrietta Lacks' story is the story of early 20th century America; a story of struggle during a difficult time in our history. Henrietta Lacks, a young African American woman, grew up in Virginia during the Jim Crow era. In order to improve her family's prospects, she moved with her husband and two young children to Baltimore to begin a new and, hopefully, better life. While the move did not take her out of the South, it did transport her from life on a tobacco farm in rural Virginia to a booming industrial port city. In these details, Henrietta's life was reminiscent of the struggles and displacement of countless other African Americans in the United States at that time. Having hoped to emerge from slavery into some semblance of freedom, at the end of the 19th century, many African Americans in the South found themselves thrust into a new form of bondage as sharecroppers, in perennial debt to white landowners. Finally, in the early 1900's, African Americans began leaving the rural South in favor of Northern urban centers, in a move that dramatically changed the social landscape of the United States.

Even at the end of her life, Henrietta's experiences illuminate the details of life as an African American and as a woman, as well as the realities of science and medicine in Baltimore at that time. Henrietta Lacks was diagnosed with an aggressive cervical cancer, for which the accepted treatment at the time was the implantation of vials of radioactive material within the cervix. While this treatment was the medical standard of care at the time, the fact that Henrietta had to receive this treatment in a segregated ward at Johns Hopkins University Hospital was not out of medical necessity. These trappings of segregation were the outward expressions of a mindset that has been a part of the American experience since its inception; and, indeed, the United States is not the only place in which these prejudices have made a home. However, the intersection of racism with science and medicine is particularly insidious. This intersection gave implicit license to doctors conducting unethical studies on unsuspecting African American patients in Alabama, in what would be called the Tuskegee Syphilis Experiment, as well as a litany of other inexcusable incidents that have tarnished the image of the scientific and medical establishments over the decades. Several years before Henrietta's death, in response to the horrific exploits of Nazi doctors during the Second World War, the Nuremberg Laws codified a handful of basic tenets of medical ethics. The first of these tenets requires that patient consent be granted before an experiment or procedure is carried out. Less than ten years after these laws were put forth, Henrietta's cells were isolated, grown, and disseminated around the world without her consent or, indeed, knowledge.

Despite the manner in which Henrietta's cells were obtained and propagated, the scientific advances attributed to these cells cannot be understated, and also comprise part of the lasting legacy she leaves behind. The cells that tormented Henrietta in life, and were fashioned into tools of biomedical science after her death, have allowed us to better understand cancer, discover and produce vaccines, and understand basic details of the inner workings of the cell. Furthermore, they have allowed the establishment of conditions for the creation of countless other cell lines, which are indispensable in the modern study of human health and disease.

This curriculum will explore a variety of topics that interconnect through Henrietta's life and experiences, and will highlight the importance of these topics to our current understanding of science and society. Students and teachers will explore how prejudices impact individuals and societies, directly and indirectly, as well as attempt to understand Henrietta's personal experiences as she moved away from Virginia. Henrietta died of an aggressive form of cervical cancer and students will be guided through an exploration of our current understanding of how cancer comes about and may be treated. Years after the original diagnosis, scientists identified Human Papilomavirus (HPV) living within Henrietta's cells and this virus may have been responsible for making her disease more aggressive. Students will explore our current understanding of the link between HPV and cancer.

HeLa cells, as Henrietta's cells have been dubbed, are sometimes referred to as her immortal life; the physical part of her, or at least that part of her that misbehaved sixty years ago, which will continue to live forever. However, Henrietta's true immortality is achieved through memory. She is immortal in that her name is on the lips of every student in a biology class, every scientist and doctor who wishes to save or improve a life, every social scientist who aims to learn from our past to fashion a better future. It is said that he who saves one life, it is as if he had saved the entire world. Henrietta has saved countless lives and she is not done quite yet.

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Introduction

In 1951, Henrietta Lacks succumbed to the cancer that had infiltrated almost every tissue in her body. This cancer had traveled, or metastasized, and made a home in tissue after tissue, destroying the body that was its home from the inside. Despite how it might seem, as we think of how many people we know — or know of — who have encountered the disease, this was an extremely unlikely outcome. In order for a normal cell to become a successful cancer cell, and a malignant tumor at that, a long series of events must take place, with each one of these events having a vanishingly low likelihood of happening. This is the reason that most of us are likely to live cancer-free lives.

The first line of defense is a self-destruct system, present within each one of our cells, designed to recognize when something has gone awry. When this system recognizes even the smallest aberration, it forces the offending cell to commit suicide, eliminating the possibility of further disease. In order to become a successful cancer cell, a normal cell must first lose this machinery, making it unable to kill itself. However, once it evades the internal survey system, it must also evade the external surveyor, the immune system, which works tirelessly to rid the body of misbehaving cells along with all manner of other disease causing agents. Having reached these unlikely milestones, most cells will still not develop the ability to metastasize, as most cells like being among friends; most cells hate being alone and rely on the company of neighboring cells to stay healthy. Once detached from their home tissue, cancer cells must be able to survive the journey through the body and be able to take root in a new tissue; most cells find tissues different from their home tissue alien and inhospitable. Having arrived at their new home, cancer cells must develop the ability to replicate without aging, another mechanism designed to ensure that faulty cells do not give rise to more faulty cells. Finally, the newly metastasized tumor must begin the process of setting up infrastructure for continued survival; namely, a blood supply to bring oxygen, nutrients, and hormones to the growing tumor. While each one of these events is unlikely, all of them taking place together is like winning the most unlucky of lotteries.

The fact that all these steps must take place to turn a normal cell into a successful cancer cell would suggest that cancer cells are fundamentally unique from normal cells. This belief gives hope to researchers and doctors, as it is believed that, with the right tools, we could use these differences to fight the disease; either by identifying or by directly killing the cancer cells while sparing the normal cells around them. This was the underlying reasoning behind the use of Radium vials to treat Henrietta's cancer and it remains the prevalent principle guiding cancer research today; the main difference is that our arsenal has become much more complex. In addition to the instruments available to researchers, an important conceptual shift guides modern cancer therapy development: cancer is a heterogeneous disease, rather than one monolithic diagnosis, with no two cancers (or two patients) being exactly the same. This means two things: the first is that no cure is likely to cure all cancers and the second is that a combination of treatments may be concocted for each patient that will be uniquely effective in curing their disease.

This unit will explore the biology underlying cancer and how it presents opportunities for treatment. Several recent treatment approaches under development will be evaluated and students will learn why they might work and where potential lies for future discovery.

Henrietta Lacks: Cancer Treatments from Henrietta Lacks to Today

Time

Seven class periods (60-80 minutes each)

Key Concepts

In this lesson, students will consider how Henrietta Lacks' cancer was treated via an implanted radioactive device. Later, they will discuss telomerase and its role in cancer and it's responsibility in making HeLa cells 'immortal.' Eventually, students will design a protein that will inhibit the function of telomerase as they learn about new cancer treatments since the time of Henrietta Lacks.

Learning Objectives

After completing this lesson, students will:

- Design and build a model of a designer protein in order to inhibit the activity of an enzyme
- Describe the differences between modern cancer treatments and prior ones
- Describe how telomerase structure and function could be used as a means of cancer treatment

Standards

HS-LS1-1. Construct an explanation based on evidence for how the structure of DNA determines the structure of proteins which carry out the essential functions of life through systems of specialized cells.
HS-LS1-4. Use a model to illustrate the role of cellular division (mitosis) and differentiation in producing and maintaining complex organisms.
HS-ETS1-3. Evaluate a solution to a complex real-world problem based on prioritized criteria and trade-offs that account for a range of constraints, including cost, safety, reliability, and aesthetics as well as possible social, cultural, and environmental impacts.
HS-ETS1-2. Design a solution to a complex real-world problem by breaking it down into smaller, more manageable problems that can be solved through engineering.

Prerequisite Knowledge

Students should come into the class with a basic understanding of DNA, chromosomes, and cell structure. Students should also understand of the induced fit model of enzymes. Depending on the level of students being taught, students would benefit from a small background knowledge of with of proteins, synthesis of proteins, and protein folding.

Materials and Handouts

For the Teacher:

- Shoelaces (3): One with a plastic tip, and one without plastic tip

For the Student:

- 5 Pipe cleaners, 10 of each color of bead, 1 golf ball
  - White, red, blue, yellow, orange, green, pink, purple beads
- Student Resource Sheet #1 “Chapter 3”
- Student Resource Sheet #2 “Nobel Prize Press Release”
- Student Resource Sheet #3 “Telomerase Word Web”
- Student Resource Sheet #4 “Engineering Challenge”
- Student Resource Sheet #5 “Engineering Design Process - Designer Protein Practica”
- Student Resource Sheet #6 “Telomerase as a Means for Cancer Treatment”
- Student Resource Sheet #7 “Cancer Treatment”
- Student Resource Sheet #8 “Cancer Treatment in the 1940’s”
- Student Resource Sheet #9 “Press Release”

Procedure: Introduction Activity 1

1. Ask students “How do we treat cancer?” *Possible responses may include chemotherapy, radiation, surgery, etc. depending upon student knowledge and personal experience.* Tell them that they are going to learn about Cancer treatment and how it has changed over the last century.
2. Distribute Student Resource Sheet #1 “Chapter 3.” Have students read the excerpt about Henrietta Lack’s cancer treatment.
3. After reading, ask them “ What made (cancerous) HeLa cells different from other (normal) cells?” *Possible responses might include: they grew aggressively, they spread out fast in Henrietta’s body, they did not die outside the body.*
4. Tell Students: To understand cell immortality like those taken from Henrietta Lacks, we have to first know about some chromosome structures named telomeres. Telomeres are an essential part of human cells that affect how our cells age. Telomeres are the caps at the end of each strand of DNA that protect our chromosomes, like the plastic tips at the end of shoelaces. Without the coating, shoelaces would shred until they can no longer do their job, just as without telomeres, DNA strands become damaged and our cells can’t do their job.”
5. Show students three shoelaces. Ask them: “What would happen if the plastic tip was not on the shoelace?” *Possible responses might include: The shoelaces would fray, you wouldn’t be able to lace the shoes, etc. Guide students to an understanding that the structure affects the function.*
6. Invite student to try and thread a frayed shoelace through a hole. Ask them to reflect on the structure and functionality of a shoelace when it is frayed.
7. Tell students: *The shoelaces are a model for DNA and that the plastic tip on the end of the shoelace represents the protection again fraying of the chromosomes, as provided by enzyme telomerase. Telomerase works by extending the protective caps on the ends of chromosomes.* (Further explanation is not required at this time as students will explore more about telomerase later in the instructional sequence.)

8. Distribute Student Resource Sheet #2 "Nobel Prize Press Release" and Student Resource Sheet #3 "Telomerase Word Web." Have students read the press release and create a word web using the reading. To increase the cognitive lift, ask them to sort their ideas onto the left, right, or middle of the word web based on whether or not the evidence supports the idea that telomerase could be used in cancer treatment (the right side, telomerase couldn't be used in cancer treatment (the left side) or just a fact about the structure or function of telomerase (middle). *Student responses could resemble those in the chart below:*

What about telomerase could make it not attractive for cancer treatment?	**Telomerase structure and function**	**What about telomerase could make it attractive for cancer treatment?**
If telomerase is preserved, cancer cells survive. Telomerase was transplanted and it prevented cell chromosomes from degrading (this would not be beneficial for cancer cells). If telomerase is defective it could lead to diseases.	Telomeres protect chromosomes from degradation. Telomeres are present in most plants and animals. Telomerase delays aging of cells. Telomerase forms a protective cap on the ends of DNA. Telomerase activity is high in cancer cells.	When telomerase is damaged, cells die (like cancer cells) By mutating telomerase, scientists were able to make cells age (this would be good to do to cancer cells). Scientists considered eradicating telomerase in cancer cells. This is being studied—including vaccines.

9. Remind students that to treat Henrietta Lacks (with her immortal cells due to telomerase), doctors inserted radium into her cervix. Display Powerpoint Slide #2.

10. Ask students to recall the earlier reading describing this treatment and how the radium might have worked. *Answers will vary, but should include some variation of 'killing the cancer cells.'* Ask students why this treatment might not be desirable. *Answers might include that radium kills more than cancer cells, it's invasive, etc.*

11. Remind students that Henrietta Lacks' cells did not die when they were taken to Gey's Lab. Ask them to recall their earlier reading of Telomerase and describe why they did not die. *Answers should include a description of telomerase in cancer cells preventing the cancer cells from aging and dying.*

12. Ask students how researchers might exploit this phenomenon to design a solution to cancer. *Answers will vary, but guide students to the inhibition of telomerase function. Depending on grade level and student prior knowledge, students may need guided to the idea of an "induced fit".* Guide students to a "substrate'", a protein, for telomerase. This will set them up for the next phase.

Activity 2

1. Divide students into groups of 2 - 3.

2. Distribute Student Resource Sheet #4 "Engineering Challenge" and Student Resource Sheet #5 "Engineering Design Process - Designer Protein Practica." Have students read the challenge and ask clarifying questions about the criteria and constraints of the problem.

3. Students should design and build their designer protein using the materials provided by the teacher. They should complete Student Resource Sheet #5 as they work through the design process.

4. Before students complete their final redesign based on their initial tests, allow students to share their design

with one another and offer critiques based on the criteria and constraints designated at the beginning of the activity. Depending on time allowed, students may be given an entire period to design and build and then complete the handout outside of class.

Activity 3

1. Distribute Student Resource Sheet #6 "Telomerase as a Means for Cancer Treatment." Tell students that they should read and annotate the abridged passages, and write questions in the margins of the handout.
2. In triads, students should discuss their reading and work together to answer the reflection questions.
3. Once students have discussed together, have students share out the answers to their reflection questions as a class. Consider recording the class consensus ideas on an anchor chart for future reference.

Activity 4

1. Tell students that they are going to be learning about cancer treatments. Distribute Student Resource Sheet #7 "Cancer Treatments."
2. Facilitate a jigsaw activity to allow students to access each type of cancer treatment and discuss the benefits and drawbacks of each one.
3. Direct students to the following website: https://www.cancer.gov/about-cancer/treatment/types
4. Ask students to go to the website and research different cancer treatment options. The teacher may assign the type of treatment or students could pick based on interests. Ask students to complete their own research and complete their record sheet. Then, ask students to get together with classmates that have researched different cancer treatments. Ask students to share their findings and complete the rest of their record sheet.
5. Distribute Student Resource Sheet #8 "Cancer Treatment- 1940's" Ask students to read the excerpt and reflect back on what they learned from the jigsaw activity. As a whole group, discuss the changes that have taken place since Henrietta Lack's time.

Activity 5

1. Direct students to complete the press release activity found on Student Resource Sheet #9.
2. Once students have completed their press releases, post them around the room and allow students to participate in a gallery walk. This also provides an opportunity to invite other members of the school community into the classroom to observe student work.

After her visit to Hopkins, Henrietta went about life as usual, cleaning and cooking for Day, their children, and the many cousins who stopped by. Then, a few days later, Jones got her biopsy results from the pathology lab: "Epidermoid carcinoma of the cervix, Stage I."

All cancers originate from a single cell gone wrong and are categorized based on the type of cell they start from. Most cervical cancers are carcinomas, which grow from the epithelial cells that cover the cervix and protect its surface. By chance, when Henrietta showed up at Hopkins complaining of abnormal bleeding, Jones and his boss, Richard Wesley TeLinde, were involved in a heated nationwide debate over what qualified as cervical cancer, and how best to treat it.

TeLinde, one of the top cervical cancer experts in the country, was a dapper and serious fifty-six-year-old surgeon who walked with an extreme limp from an ice-skating accident more than a decade earlier. Everyone at Hopkins called him Uncle Dick. He'd pioneered the use of estrogen for treating symptoms of menopause and made important early discoveries about endometriosis. He'd also written one of the most famous clinical gynecology textbooks, which is still widely used sixty years and ten editions after he first wrote it. His reputation was international: when the king of Morocco's wife fell ill, he insisted only TeLinde could operate on her. By 1951, when Henrietta arrived at Hopkins, TeLinde had developed a theory about cervical cancer that, if correct, could save the lives of millions of women. But few in the field believed him.

Cervical carcinomas are divided into two types: invasive carcinomas, which have penetrated the surface of the cervix, and noninvasive carcinomas, which haven't. The noninvasive type is sometimes called "sugar-icing carcinoma," because it grows in a smooth layered sheet across the surface of the cervix, but its official name is carcinoma in situ, which derives from the Latin for "cancer in its original place."

In 1951, most doctors in the field believed that invasive carcinoma was deadly, and carcinoma in situ wasn't. So they treated the invasive type aggressively but generally didn't worry about carcinoma in situ because they thought it couldn't spread. TeLinde disagreed—he believed carcinoma in situ was simply an early stage of invasive carcinoma that, if left untreated, eventually became deadly. So he treated it aggressively, often removing the cervix, uterus, and most of the vagina. He argued that this would drastically reduce cervical cancer deaths, but his critics called it extreme and unnecessary.

Diagnosing carcinoma in situ had only been possible since 1941, when George Papanicolaou, a Greek researcher, published a paper describing a test he'd developed, now called the Pap smear. It involved scraping cells from the cervix with a curved glass pipette and examining them under a microscope for precancerous changes that TeLinde and a few others had identified years earlier. This was a tremendous advance, because those precancerous cells weren't detectable otherwise: they caused no physical symptoms and weren't palpable or visible to the naked eye. By the time a woman began showing symptoms, there was little hope of a cure. But with the Pap smear, doctors could detect precancerous cells and perform a hysterectomy, and cervical cancer would be almost entirely preventable.

At that point, more than 15,000 women were dying each year from cervical cancer. The Pap smear had the potential to decrease that death rate by 70 percent or more, but there were two things standing in its way: first, many women—like Henrietta—simply didn't get the test; and, second, even when they did, few doctors knew how to interpret the results accurately, because they didn't know what the various stages of cervical cancer looked like under a microscope. Some mistook cervical infections for cancer and removed a woman's entire reproductive tract when all she needed was antibiotics. Others mistook malignant changes for infection, sending women home with antibiotics only to have them return later, dying from metastasized cancer. And even when doctors correctly diagnosed precancerous changes, they often didn't know how those changes should be treated.

TeLinde set out to minimize what he called "unjustifiable hysterectomies" by documenting what wasn't cervical cancer and by urging surgeons to verify smear results with biopsies before operating. He also hoped to prove that women with carcinoma in situ needed aggressive treatment, so their cancer didn't become invasive.

Not long before Henrietta's first exam, TeLinde presented his argument about carcinoma in situ to a major meeting of pathologists in Washington, D.C., and the audience heckled him off the stage. So he went back to Hopkins and planned a study that would prove them wrong: he and his staff would review all medical records and biopsies from patients who'd been diagnosed with invasive cervical cancer at Hopkins in the past decade, to see how many initially had carcinoma in situ.

Like many doctors of his era, TeLinde often used patients from the public wards for research, usually without their knowledge. Many scientists believed that since patients were treated for free in the public wards, it was fair to use them as research subjects as a form of payment. And as Howard Jones once wrote, "Hopkins, with its large indigent black population, had no dearth of clinical material."

In this particular study—the largest ever done on the relationship between the two cervical cancers—Jones and TeLinde found that 62 percent of women with invasive cancer who'd had earlier biopsies first had carcinoma in situ. In addition to that study, TeLinde thought, if he could find a way to grow living samples from normal cervical tissue and both types of cancerous tissue—something never done before—he could compare all three. If he could prove that carcinoma in situ and invasive carcinoma looked and behaved similarly in the laboratory, he could end the debate, showing that he'd been right all along, and doctors who ignored him were killing their patients. So he called George Gey (pronounced Guy), head of tissue culture research at Hopkins.

Gey and his wife, Margaret, had spent the last three decades working to grow malignant cells outside the body, hoping to use them to find cancer's cause and cure. But most cells died quickly, and the few that survived hardly grew at all. The Geys were determined to grow the first immortal human cells: a continuously dividing line of cells all descended from one original sample, cells that would constantly replenish themselves and never die. Eight years earlier—in 1943—a group of researchers at the National Institutes of Health had proven such a thing was possible using mouse cells. The Geys wanted to grow the human equivalent—they didn't care what kind of tissue they used, as long as it came from a person.

Gey took any cells he could get his hands on—he called himself "the world's most famous vulture, feeding on human specimens almost constantly." So when TeLinde offered him a supply of cervical cancer tissue in exchange for trying to grow some cells, Gey didn't hesitate. And TeLinde began collecting samples from any woman who happened to walk into Hopkins with cervical cancer. Including Henrietta.
On February 5, 1951, after Jones got Henrietta's biopsy report back from the lab, he called and told her it was malignant. Henrietta didn't tell anyone what Jones said, and no one asked. She simply went on with her day as if nothing had happened, which was just like her—no sense upsetting anyone over something she could deal with herself.

That night Henrietta told her husband, "Day, I need to go back to the doctor tomorrow. He wants to do some tests, give me some medicine." The next morning she climbed from the Buick outside Hopkins again, telling Day and the children not to worry. "Ain't nothin serious wrong," she said. "Doctor's gonna fix me right up." Henrietta went straight to the admissions desk and told the receptionist she was there for her treatment. Then she signed a form with the words OPERATION PERMIT at the top of the page. It said:

I hereby give consent to the staff of The Johns Hopkins Hospital to perform any operative procedures and under any anaesthetic either local or general that they may deem necessary in the proper surgical care and treatment of:

_____

Henrietta printed her name in the blank space. A witness with illegible handwriting signed a line at the bottom of the form, and Henrietta signed another.

Then she followed a nurse down a long hallway into the ward for colored women, where Howard Jones and several other white physicians ran more tests than she'd had in her entire life. They checked her urine, her blood, her lungs. They stuck tubes in her bladder and nose.
On her second night at the hospital, the nurse on duty fed Henrietta an early dinner so her stomach would be

empty the next morning, when a doctor put her under anesthetic for her first cancer treatment. Henrietta's tumor was the invasive type, and like hospitals nationwide, Hopkins treated all invasive cervical carcinomas with radium, a white radioactive metal that glows an eerie blue.

When radium was first discovered in the late 1800s, headlines nationwide hailed it as "a substitute for gas, electricity, and a positive cure for every disease." Watchmakers added it to paint to make watch dials glow, and doctors administered it in powdered form to treat everything from seasickness to ear infections. But radium destroys any cells it encounters, and patients who'd taken it for trivial problems began dying. Radium causes mutations that can turn into cancer, and at high doses it can burn the skin off a person's body. But it also kills cancer cells.

Hopkins had been using radium to treat cervical cancer since the early 1900s, when a surgeon named Howard Kelly visited Marie and Pierre Curie, the couple in France who'd discovered radium and its ability to destroy cancer cells. Without realizing the danger of contact with radium, Kelly brought some back to the United States in his pockets and regularly traveled the world collecting more. By the 1940s, several studies—one of them conducted by Howard Jones, Henrietta's physician—showed that radium was safer and more effective than surgery for treating invasive cervical cancer.

The morning of Henrietta's first treatment, a taxi driver picked up a doctor's bag filled with thin glass tubes of radium from a clinic across town. The tubes were tucked into individual slots inside small canvas pouches hand-sewn by a local Baltimore woman. The pouches were called Brack plaques, after the Hopkins doctor who invented them and oversaw Henrietta's radium treatment. He would later die of cancer, most likely caused by his regular exposure to radium, as would a resident who traveled with Kelly and also transported radium in his pockets.

One nurse placed the Brack plaques on a stainless-steel tray. Another wheeled Henrietta into the small colored-only operating room on the second floor, with stainless-steel tables, huge glaring lights, and an all-white medical staff dressed in white gowns, hats, masks, and gloves.

With Henrietta unconscious on the operating table in the center of the room, her feet in stirrups, the surgeon on duty, Dr. Lawrence Wharton Jr., sat on a stool between her legs. He peered inside Henrietta, dilated her cervix, and prepared to treat her tumor. But first—though no one had told Henrietta that TeLinde was collecting samples or asked if she wanted to be a donor—Wharton picked up a sharp knife and shaved two dime-sized pieces of tissue from Henrietta's cervix: one from her tumor, and one from the healthy cervical tissue nearby. Then he placed the samples in a glass dish.

Wharton slipped a tube filled with radium inside Henrietta's cervix, and sewed it in place. He sewed a plaque filled with radium to the outer surface of her cervix and packed another plaque against it. He slid several rolls of gauze inside her vagina to help keep the radium in place, then threaded a catheter into her bladder so she could urinate without disturbing the treatment.

When Wharton finished, a nurse wheeled Henrietta back into the ward, and Wharton wrote in her chart, "The patient tolerated the procedure well and left the operating room in good condition." On a separate page he wrote, "Henrietta Lacks … Biopsy of cervical tissue … Tissue given to Dr. George Gey."

A resident took the dish with the samples to Gey's lab, as he'd done many times before. Gey still got excited at moments like this, but everyone else in his lab saw Henrietta's sample as something tedious—the latest of what felt like countless samples that scientists and lab technicians had been trying and failing to grow for years. They were sure Henrietta's cells would die just like all the others.

https://nitramacademybookspot.wordpress.com/2016/09/07/chapter-3-diagnosis-and-treatment/

PRESS RELEASE 2009-10-05 The Nobel Assembly at Karolinska Institutet has today decided to award The Nobel Prize in Physiology or Medicine 2009 jointly to Elizabeth H. Blackburn, Carol W. Greider and Jack W. Szostak

Nobelförsamlingen
The Nobel Assembly at Karolinska Institutet

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for the discovery of

"how chromosomes are protected by telomeres and the enzyme telomerase"

SUMMARY

This year´s Nobel Prize in Physiology or Medicine is awarded to three scientists who have solved a major problem in biology: how the chromosomes can be copied in a complete way during cell divisions and how they are protected against degradation. The Nobel Laureates have shown that the solution is to be found in the ends of the chromosomes – the telomeres – and in an enzyme that forms them – telomerase.

The long, thread-like DNA molecules that carry our genes are packed into chromosomes, the telomeres being the caps on their ends. Elizabeth Blackburn and Jack Szostak discovered that a unique DNA sequence in the telomeres protects the chromosomes from degradation. Carol Greider and Elizabeth Blackburn identified telomerase, the enzyme that makes telomere DNA. These discoveries explained how the ends of the chromosomes are protected by the telomeres and that they are built by telomerase.

If the telomeres are shortened, cells age. Conversely, if telomerase activity is high, telomere length is maintained, and cellular senescence is delayed. This is the case in cancer cells, which can be considered to have eternal life. Certain inherited diseases, in contrast, are characterized by a defective telomerase, resulting in damaged cells. The award of the Nobel Prize recognizes the discovery of a fundamental mechanism in the cell, a discovery that has stimulated the development of new therapeutic strategies.

The mysterious telomere

The chromosomes contain our genome in their DNA molecules. As early as the 1930s, Hermann Muller (Nobel Prize 1946) and Barbara McClintock (Nobel Prize 1983) had observed that the structures at the ends of the chromosomes, the so-called telomeres, seemed to prevent the

chromosomes from attaching to each other. They suspected that the telomeres could have a protective role, but how they operate remained an enigma.

When scientists began to understand how genes are copied, in the 1950s, another problem presented itself. When a cell is about to divide, the DNA molecules, which contain the four bases that form the genetic code, are copied, base by base, by DNA polymerase enzymes. However, for one of the two DNA strands, a problem exists in that the very end of the strand cannot be copied. Therefore, the chromosomes should be shortened every time a cell divides – but in fact that is not usually the case (Fig 1).

Both these problems were solved when this year´s Nobel Laureates discovered how the telomere functions and found the enzyme that copies it.

Telomere DNA protects the chromosomes

In the early phase of her research career, Elizabeth Blackburn mapped DNA sequences. When studying the chromosomes of *Tetrahymena*, a unicellular ciliate organism, she identified a DNA sequence that was repeated several times at the ends of the chromosomes. The function of this sequence, CCCCAA, was unclear. At the same time, Jack Szostak had made the observation that a linear DNA molecule, a type of minichromosome, is rapidly degraded when introduced into yeast cells.

Blackburn presented her results at a conference in 1980. They caught Jack Szostak´s interest and he and Blackburn decided to perform an experiment that would cross the boundaries between very distant species (Fig 2). From the DNA of *Tetrahymena*, Blackburn isolated the CCCCAA sequence. Szostak coupled it to the minichromosomes and put them back into yeast cells. The results, which were published in 1982, were striking – the telomere DNA sequence protected the minichromosomes from degradation. As telomere DNA from one organism, *Tetrahymena*, protected chromosomes in an entirely different one, yeast, this demonstrated the existence of a previously unrecognized fundamental mechanism. Later on, it became evident that telomere DNA with its characteristic sequence is present in most plants and animals, from amoeba to man.

An enzyme that builds telomeres

Carol Greider, then a graduate student, and her supervisor Blackburn started to investigate if the formation of telomere DNA could be due to an unknown enzyme. On Christmas Day, 1984, Greider discovered signs of enzymatic activity in a cell extract. Greider and Blackburn named the enzyme telomerase, purified it, and showed that it consists of RNA as well as protein (Fig 3). The RNA component turned out to contain the CCCCAA sequence. It serves as the template when the telomere is built, while the protein component is required for the construction work, i.e. the enzymatic activity. Telomerase extends telomere DNA, providing a platform that enables DNA polymerases to copy the entire length of the chromosome without missing the very end portion.

Telomeres delay ageing of the cell

Scientists now began to investigate what roles the telomere might play in the cell. Szostak´s group identified yeast cells with mutations that led to a gradual shortening of the telomeres. Such cells grew poorly and eventually stopped dividing. Blackburn and her co-workers made mutations in the RNA of the telomerase and observed similar effects in *Tetrahymena.* In both cases, this led to premature cellular ageing – senescence. In contrast, functional telomeres instead prevent chromosomal damage and delay cellular senescence. Later on, Greider´s group showed that the senescence of human cells is also delayed by telomerase. Research in this area has been intense and it is now known that the DNA sequence in the telomere attracts proteins that form a protective cap around the fragile ends of the DNA strands.

An important piece in the puzzle – human ageing, cancer, and stem cells

These discoveries had a major impact within the scientific community. Many scientists speculated that telomere shortening could be the reason for ageing, not only in the individual cells but also in

the organism as a whole. But the ageing process has turned out to be complex and it is now thought to depend on several different factors, the telomere being one of them. Research in this area remains intense.

Most normal cells do not divide frequently, therefore their chromosomes are not at risk of shortening and they do not require high telomerase activity. In contrast, cancer cells have the ability to divide infinitely and yet preserve their telomeres. How do they escape cellular senescence? One explanation became apparent with the finding that cancer cells often have increased telomerase activity. It was therefore proposed that cancer might be treated by eradicating telomerase. Several studies are underway in this area, including clinical trials evaluating vaccines directed against cells with elevated telomerase activity.

Some inherited diseases are now known to be caused by telomerase defects, including certain forms of congenital aplastic anemia, in which insufficient cell divisions in the stem cells of the bone marrow lead to severe anemia. Certain inherited diseases of the skin and the lungs are also caused by telomerase defects.

In conclusion, the discoveries by Blackburn, Greider and Szostak have added a new dimension to our understanding of the cell, shed light on disease mechanisms, and stimulated the development of potential new therapies.

Elizabeth H. Blackburn has US and Australian citizenship. She was born in 1948 in Hobart, Tasmania, Australia. After undergraduate studies at the University of Melbourne, she received her PhD in 1975 from the University of Cambridge, England, and was a postdoctoral researcher at Yale University, New Haven, USA. She was on the faculty at the University of California, Berkeley, and since 1990 has been professor of biology and physiology at the University of California, San Francisco.

Carol W. Greider is a US citizen and was born in 1961 in San Diego, California, USA. She studied at the University of California in Santa Barbara and in Berkeley, where she obtained her PhD in 1987 with Blackburn as her supervisor. After postdoctoral research at Cold Spring Harbor Laboratory, she was appointed professor in the department of molecular biology and genetics at Johns Hopkins University School of Medicine in Baltimore in 1997.

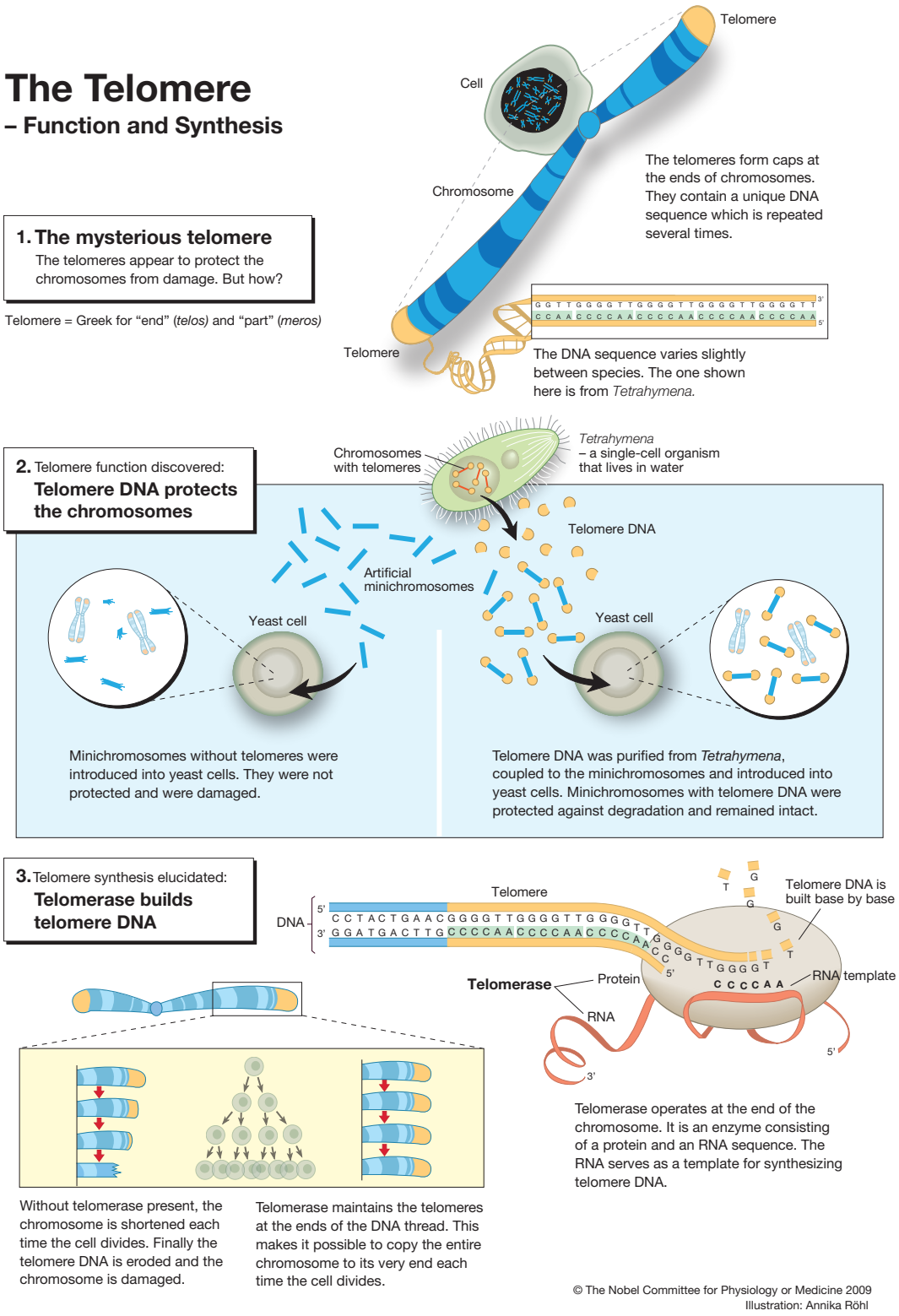
Jack W. Szostak is a US citizen. He was born in 1952 in London, UK and grew up in Canada. He studied at McGill University in Montreal and at Cornell University in Ithaca, New York, where he received his PhD in 1977. He has been at Harvard Medical School since 1979 and is currently professor of genetics at Massachusetts General Hospital in Boston. He is also affiliated with the Howard Hughes Medical Institute.

References:

Szostak JW, Blackburn EH. Cloning yeast telomeres on linear plasmid vectors. Cell 1982; 29:245-255.
Greider CW, Blackburn EH. Identification of a specific telomere terminal transferase activity in *Tetrahymena* extracts. Cell 1985; 43:405-13.
Greider CW, Blackburn EH. A telomeric sequence in the RNA of *Tetrahymena* telomerase required for telomere repeat synthesis. Nature 1989; 337:331-7.

The Nobel Assembly, consisting of 50 professors at Karolinska Institutet, awards the Nobel Prize in Physiology or Medicine. Its Nobel Committee evaluates the nominations. Since 1901 the Nobel Prize has been awarded to scientists who have made the most important discoveries for the benefit of mankind.

What about telomerase could make it not attractive for cancer treatment?



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Student Resource Sheet #4

Designer Protein Engineering Challenge

The National Institute of Health (NIH) urgently needs to develop a designer protein to break down the "infinimorase" enzyme that certain cancer cells in the human body use as they divide. "Infinimorase" allows rogue cells that would normally die of old age to live forever. **NIH** biochemists have determined that "infinimorase" has two possible places of hydrogen bonding. An induced fit needs to be designed that has two contact points that will properly hydrogen bond on the substrate so that it's cleaved in order to stop its function.

Beads Colors:

Contacts for Hydrogen Bonds:

- H → White
- OH → Red

Amino Acids:

Blue - Basic
Yellow - Acidic
Orange - Hydrophobic
Green - Hydrophilic
Pink - Cysteine (sulfur containing amino acid)
Purple - Proline (amino acid that allows a bend/kink in the protein)

** Protein Folding Rules

- Hydrophobic amino acids **(F)** will move to the center of the structure
- Hydrophilic amino acids **(L**) will move to the outer edge of the structure
- Acids **(A)** are positively charged and bases **(B)** are negatively charged and will be attracted one another
- 2 cysteines **(C)** which are sulfur containing amino acids can form one cross bridge (disulfide bond)
- Kinks/bends can only occur in the amino acid backbone with the amino acid proline **(P)** - a bend up to a 90 degree angle (right angle) is allowed
- Only curves can be made with all the other amino acids
- 3D structure can be held together with the disulfide cross bridges between two sulfur-containing amino acids

Constraints:

- Must have 2 contact points between the enzyme and substrate that demonstrates hydrogen bonding
- Substrate and enzyme cannot be worked on at the same time*****
- Each amino acid must be 2 - 3 cm apart
- Attractions must be demonstrated in the protein that is built
  - Hydrophobic region(s)
  - Hydrophilic region(s)
  - Acid – Base interactions
  - Amino acids that contain sulfur will form cross-bridges (disulfide bonds)
  - Disulfide bonds can be held in place using a twisty tie

- First, hydrophobic and hydrophilic region should be established in 2D
- The acid-base attractions and the disulfide bonds will allow for the designer protein to take on a 3D shape
- Ability to open/close or move the enzyme to model induced fit is necessary

Student Resource Sheet #5

Engineering Design Process - Designer Protein Practica

Team members:

Part 1: Define Problem

This has already been provided to you.

Part 2: Create Specifications & Requirements

Ask at least three clarifying questions about this bioengineering project.

1.

2.

3.

Part 3: Create Design Concepts

Create a primary structure (amino acid backbone) for your designer protein to determine a possible amino acid sequence noting possible contact points. May use each line to show the sequence for each segment. Not all spaces need to be used.

---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ----

---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ----

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---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ----

In the space below sketch the 3D induced fit designer protein structure using the protein folding ”rules” and label the contact points.

Part 4: Design Solution

Final primary sequence of amino acids showing final contact points.

---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ---- ____ ----

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Parts 5: Test/Validate Design

Did the 3D designer protein show the correct induced fit for the infinimorase model?

Parts 6: Reflection

How did your final design perform? What was the most challenging part of this process? What two changes would you make in the future to create a better designer protein?

Excerpt #1: News Article: Designer Proteins

...But head inside to his lab and it's quickly apparent that the computational biochemist is far from satisfied with what nature offers, at least when it comes to molecules. On a low-slung coffee table lie eight toy-sized, 3D-printed replicas of proteins. Some resemble rings and balls, others tubes and cages—and none existed before Baker and his colleagues designed and built them. Over the last several years, with a big assist from the genomics and computer revolutions, Baker's team has all but solved one of the biggest challenges in modern science: figuring out how long strings of amino acids fold up into the 3D proteins that form the working machinery of life. Now, he and colleagues have taken this ability and turned it around to design and then synthesize unnatural proteins intended to act as everything from medicines to materials....

...Baker is by no means alone in this pursuit. Efforts to predict how proteins fold, and use that information to fashion novel versions, date back decades. But today he leads the charge. "David has really inspired the field," says Guy Montelione, a protein structure expert at Rutgers University, New Brunswick, in New Jersey. "That's what a great scientist does."...

...In the early 1960s, biochemists at the U.S. National Institutes of Health (NIH) recognized that each protein folds itself into an intrinsic shape. Heat a protein in a solution and its 3D structure will generally unravel. But the NIH group noticed that the proteins they tested refold themselves as soon as they cool, implying that their structure stems from the interactions between different amino acids, rather than from some independent molecular folding machine inside cells. If researchers could determine the strength of all those interactions, they might be able to calculate how any amino acid sequence would assume its final shape. The protein-folding problem was born…

...Knowing the 3D structures of those other proteins would offer biochemists vital insights into each molecule's function, such as whether it serves to ferry ions across a cell membrane or catalyze a chemical reaction. It would also give chemists valuable clues to designing new medicines. So, instead of waiting for the experimentalists, computer modelers such as Baker have tackled the folding problem with computer models…

...Another potential addition to the medicine cabinet: a designer protein that chops up gluten, the infamous substance in wheat and other grains that people with Celiac disease or gluten sensitivity have trouble digesting. Ingrid Swanson Pultz began crafting the gluten-breaker even before joining Baker's lab as a postdoc and is now testing it in animals and working with IPD to commercialize the research. And those self-assembling cages that debut this week could one day be filled with drugs or therapeutic snippets of DNA or RNA that can be delivered to disease sites throughout the body…

...Church says he believes that designer proteins might soon rewrite the biology inside cells. In a paper last year in eLife, he, Baker, and colleagues designed proteins to bind to either a hormone or a heart disease drug inside cells, and then regulate the activity of a DNA-cutting enzyme, Cas9, that is part of the popular CRISPR genome-editing system. "The ability to design sensors [inside cells] is going to be big," Church says. The strategy could allow researchers or physicians to target the powerful gene-editing system to a specific set of cells—those that are responding to a hormone or drug. Biosensors could also make it possible to switch on the expression of specific genes as needed to break down toxins or alert the immune cells to invaders or cancer…

Source: This protein designer aims to revolutionize medicines and materials
By Robert F. Service Jul. 21, 2016 , 2:00 PM
http://www.sciencemag.org/news/2016/07/protein-designer-aims-revolutionize-medicines-and-materials

Excerpt #2: Abstract: A Telomerase Substrate

Although telomerase is an almost universal target for cancer therapy, there has been no effective telomerase targeted inhibitor that has progressed to late stage human clinical trials. Recently, we reported that a telomerase-mediated telomere-disrupting compound, 6-thio-2′-deoxyguanosine (6-thio-dG), was very effective at targeting telomerase positive cancer cells while sparing telomerase silent normal cells. 6-thio-dG, a nucleoside analogue

of the already-approved drug 6-thioguanine, is incorporated into telomeres by telomerase, resulting in disruption of the telomere-protecting shelterin complex. This disruption leads to Telomere dysfunction-Induced Foci (TIFs) formation and rapid cell death for the vast majority of cancer cells. Since most chemotherapies eventually fail due to drug acquired resistance, novel drugs such as 6-thio-dG, as a single first line agent or in the maintenance setting, may represent an effective new treatment for cancer patients.

A novel telomerase substrate precursor rapidly induces telomere dysfunction in telomerase positive cancer cells but not telomerase silent normal cells
Ilgen Mender[1,2] Sergei Gryaznov[3] and Jerry W. Shay[1,4]
Oncoscience. 2015; 2(8): 693–695.
Published online 2015 Aug 22. doi: 10.18632/oncoscience.213
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4580061/?log$=activity

Excerpt #3: News Article: Blocking Telomerase

"Telomerase is overexpressed in many advanced cancers, but assessing its potential as a therapeutic target requires us to understand what it does and how it does it," said senior author Ronald DePinho, M.D., president of The University of Texas MD Anderson Cancer Center.

"We exploited the experimental merits of mice to model and study more precisely telomere crisis, telomerase reactivation and telomerase extinction in cancer development, progression and treatment," DePinho said. "This elegant model exposed two mechanisms, including one unexpected metabolic pathway, used by cancer cells to adapt to loss of telomerase…

...Telomerase activity is low or absent in normal cells, which have segments of repeat nucleotides called telomeres at the ends of their chromosomes that protect DNA stability during cell division, said first author Jian Hu, Ph.D., an instructor in MD Anderson's Department of Cancer Biology.

With each division the telomeres shorten, leading eventually to genomic instability and cell death, a period termed "telomere crisis," Hu said. In cancer, telomerase becomes active during telomere crisis and rescues the genomically abnormal cells by lengthening telomeres.

In a series of experiments in a lymphoma mouse model, the team found:

- Telomerase reactivation in malignant cells after genomic instability causes cancer progression.
- Inhibiting telomerase caused tumor cell death but also led to alternative lengthening of telomeres (ALT) independent of telomerase.
- ALT-positive cells increase both the expression and copy number of a gene called PGC-1ß regulator of mitochondrial function, to compensate for mitochondrial and reactive oxygen species defense deficiencies.
- Targeting PGC-1ß to weaken mitochondria function enhances anti-telomerase therapy.

The team then took tumor cells from late-generation mice with activated telomerase -- the aggressive tumors -- and passaged them four times through groups of mice treated with either 4-OHT to trigger telomerase production or the control vehicle that leaves the enzyme off.

During the first two rounds, survival for the two groups was about the same. In the third round, the control mice had a major improvement in survival over the telomerase arm, indicating that telomere erosion had allowed cellular defense mechanisms to pick off genomically unstable cells.

However, in the fourth passage, survival of the control-treated mice fell back toward that of mice with active telomerase. The tumors had become resistant without relying on telomerase.
Blocking telomerase kills cancer cells but provokes resistance, progression
Date: February 20, 2012
Source: University of Texas M. D. Anderson Cancer Center
https://www.sciencedaily.com/releases/2012/02/120220161229.htm

Reflection Questions

1. Describe how the designer proteins in Baker's lab and 6-thio-2´-deoxyguanosine are similar to the model protein substrate you designed in a previous activity.
2. Choose one of the passages above and summarize what the scientists did.How is this similar and different to what you did in the designer protein activity?
3. Based on the passages, suggest a reason why telomerase targeted cancer treatment hasn't advanced to clinical trials.
4. Support the following claim with evidence from the passages: 'Telomerase is a viable option for cancer treatment.'

Student Resource Sheet #7

Cancer Treatments

Directions: Please go to the following website: https://www.cancer.gov/about-cancer/treatment/types

Cancer Treatment	The basics of how this treatment works	What characteristics of a patient would make them a good candidate for this treatment?	Other notable features of this treatment	Questions I still have about this treatment
Surgery				
Radiation Therapy				
Chemotherapy				
Immunotherapy				
Targeted Therapy				
Hormone Therapy				
Stem Cell Transplant				
Precision Medicine				

Student Resource Sheet #8

Cancer Treatment in the 1940's

Now that you have completed and shared your research, consider the following excerpt from Chapter Three of *The Immortal Life of Henrietta Lacks* by Rebecca Skloot.

...Radium causes mutations that can turn into cancer, and at high doses it can burn the skin off a person's body. But it also kills cancer cells…

...By the 1940s, several studies—one of them conducted by Howard Jones, Henrietta's physician—showed that radium was safer and more effective than surgery for treating invasive cervical cancer…

...One nurse placed the Brack plaques on a stainless-steel tray. Another wheeled Henrietta into the small colored-only operating room on the second floor, with stainless-steel tables, huge glaring lights, and an all-white medical staff dressed in white gowns, hats, masks, and gloves.

With Henrietta unconscious on the operating table in the center of the room, her feet in stirrups, the surgeon on duty, Dr. Lawrence Wharton Jr., sat on a stool between her legs…

...Wharton slipped a tube filled with radium inside Henrietta's cervix, and sewed it in place. He sewed a plaque filled with radium to the outer surface of her cervix and packed another plaque against it. He slid several rolls of gauze inside her vagina to help keep the radium in place, then threaded a catheter into her bladder so she could urinate without disturbing the treatment…

https://nitramacademybookspot.wordpress.com/2016/09/07/chapter-3-diagnosis-and-treatment/

Compare the modern cancer treatments that you researched with the treatment used to treat Henrietta Lacks as described above.

Student Resource Sheet #9

Press Release

Imagine that you and your research team are able to engineer a substrate that binds to telomerase in such a way that it does inhibit its function in cancer cells and has the potential to be the next major form of cancer treatment. Henrietta Lacks is still alive and is the rst patient in the clinical trials. You will use your 'designer protein' to treat her cervical cancer. Using the evidence that you've collected, create a press release that contains the following:

- A description of your protein, its structure and its function
- Describe the process you used to develop your protein from dening the problem, designing your solution,to testing and rening your design
- A comparison of how using this protein as a cancer treatment be different from prior cancer treatments
- "Quotes" from Henrietta Lacks and her family regarding the new treatment