

Immortal Cell

The Immortal Life of Henrietta Lacks

The problem of the long-term proliferation of cells is a seminal one. It has always been a hot subject in biology, a source of far-reaching hypotheses, even more so now when explanations for the mechanisms of cell proliferative mortality or immortality seem within our reach. A question which is still debated is whether an infinite division potential can be a normal trait or is always the result of modifications leading to abnormal cell growth and escape from homeostasis. In general, investigators have been advocates of one of the two extremes, universal limited or unlimited normal proliferative potential. Since the long-term proliferative potential of cells concerns regulation of development, regeneration of tissues, and homeostatic control of cell growth, in brief survival of living organisms, and since the regulation of these processes is so different along the evolutionary scale, it is not surprising that there does not seem to be any universal trait. The question of whether cells are endowed with finite or infinite proliferative phenotypes has to be seen using the perspective of comparative biology.

Now an HBO(R) Film starring Oprah Winfrey and Rose Byrne. Her name was

Henrietta Lacks, but scientists know her as HeLa. She was a poor Southern tobacco farmer who worked the same land as her slave ancestors, yet her cells--taken without her knowledge--became one of the most important tools in medicine. The first "immortal" human cells grown in culture, they are still alive today, though she has been dead for more than sixty years. If you could pile all HeLa cells ever grown onto a scale, they'd weigh more than 50 million metric tons--as much as a hundred Empire State Buildings. HeLa cells were vital for developing the polio vaccine; uncovered secrets of cancer, viruses, and the atom bomb's effects; helped lead to important advances like in vitro fertilization, cloning, and gene mapping; and have been bought and sold by the billions. Yet Henrietta Lacks remains virtually unknown, buried in an unmarked grave. Now Rebecca Skloot takes us on an extraordinary journey, from the "colored" ward of Johns Hopkins Hospital in the 1950s to stark white laboratories with freezers full of HeLa cells; from Henrietta's small, dying hometown of Clover, Virginia--a land of wooden slave quarters, faith healings, and voodoo--to East Baltimore today, where her children and grandchildren live and struggle with the legacy of her cells. Henrietta's family did not learn of her "immortality" until more than twenty years after her death, when scientists investigating HeLa began using her husband and children in research without informed consent. And though the cells

had launched a multimillion-dollar industry that sells human biological materials, her family never saw any of the profits. As Rebecca Skloot so brilliantly shows, the story of the Lacks family--past and present--is inextricably connected to the dark history of experimentation on African Americans, the birth of bioethics, and the legal battles over whether we control the stuff we are made of. Over the decade it took to uncover this story, Rebecca became enmeshed in the lives of the Lacks family--especially Henrietta's daughter Deborah, who was devastated to learn about her mother's cells. She was consumed with questions: Had scientists cloned her mother? Did it hurt her when researchers infected her cells with viruses and shot them into space? What happened to her sister, Elsie, who died in a mental institution at the age of fifteen? And if her mother was so important to medicine, why couldn't her children afford health insurance? Intimate in feeling, astonishing in scope, and impossible to put down, *The Immortal Life of Henrietta Lacks* captures the beauty and drama of scientific discovery, as well as its human consequences.

Please note that the content of this book primarily consists of articles available from Wikipedia or other free sources online. Pages: 20. Chapters: 3T3-L1, 3T3 cells, A431 cells, A549 cell, AB9, BCP-1 cells, C2C12, Caco-2, Chinese hamster ovary cell, COS cells, DU145, H1299, HaCaT, HEK cell, HeLa, Hep G2, High

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Five cells, HL60, J558L cells, Jurkat cells, JY cell line, K562 cells, LNCaP, MC3T3, MCF-7, P19 cell, PC12 cell line, PC3, Raji cell, RBL cells, S180, Saos-2 cells, Schneider 2 cells, Sf21, Sf9, SH-SY5Y, SKBR3, THP1 cell line, Tobacco BY-2 cells, U937 cell, Vero cell, VG-1. Excerpt: A HeLa cell, also Hela or hela cell, is a cell type in an immortal cell line used in scientific research. It is the oldest and most commonly used human cell line. The line was derived from cervical cancer cells taken on February 8, 1951 from Henrietta Lacks, a patient who eventually died of her cancer on October 4, 1951. The cell line was found to be remarkably durable and prolific as illustrated by its contamination of many other cell lines used in research. HeLa cells stained with Hoechst 33258The cells were propagated by George Otto Gey shortly before Lacks died of her cancer in 1951. This was the first human cell line to prove successful in vitro, which was a scientific achievement with profound future benefit to medical research. Gey freely donated both the cells and the tools and processes his lab developed to any scientist requesting them, simply for the benefit of science. Neither Lacks or her family gave Lacks's physician permission to harvest the cells, but, at that time, permission was neither required or customarily sought. The cells were later commercialized, although never patented in their original form. Then, as now, there was no requirement to inform a patient, or their relatives, about such

matters because discarded material, or material obtained during surgery, diagnosis, or therapy, was the property of the physician and/or medical institution (currently...

[Truncated abstract] Oval cells are adult liver progenitor cells that regenerate the liver when hepatocyte replication is inhibited following chronic or carcinogenic injury. They have the potential to not only differentiate into hepatocytes and cholangiocytes, but also along pancreatic and intestinal lineages.

Understandably, the interest in these stem-like cells has substantially increased, particularly from a therapeutic standpoint, as their plasticity makes them excellent candidates for cell transplantation and gene therapy to treat various gastrointestinal diseases. However, since they emerge in pathologies with a predisposition to cancer, oval cells are possible targets for transformation and they have been implicated in the formation of liver tumours. Consequently there are several scientific challenges that must be overcome before one can realise their clinical potential as tissue regenerating stem cells. The first aim of the studies presented in this thesis was to further investigate the regeneration process in chronically injured mouse liver induced by a choline-deficient, ethionine-supplemented diet (CDE). The invoked oval cell response was validated and quantified over three weeks. Gene expression profiling by

microarray analysis of twoweek CDE-treated versus control liver revealed that the majority of downregulated genes was associated with metabolic and catalytic activities, reflecting a collapse of enzyme-regulated homeostasis and hepatic insufficiency in CDE liver. Concurrently, upregulated genes were generally linked with an inflammatory reaction and tissue repair as well as the proliferation of oval cells, which mediate regeneration in this model. Investigation of factors known to be required for hepatic restoration after acute injury showed that many signalling pathways are similarly activated or regulated during CDEinduced chronic injury. To further the study of the mechanisms controlling oval cell behaviour, two in vitro models were established: primary oval cell cultures and immortal cell lines. ... None of the cell lines grew in semi-solid agar, nor did they form tumours in nude mice, indicating they are non-tumourigenic. Previous studies in mice have suggested that the tumour necrosis factor (TNF) family member "TNF-like weak inducer of apoptosis" (TWEAK) induces oval cell proliferation by binding to its cognate receptor Fn14. Microarray data and real-time PCR analysis showed that TWEAK/Fn14 signalling is upregulated in the CDE model of chronic liver injury. It was therefore of interest to examine the direct effects of TWEAK on oval cells using the two newly established in vitro models. Heterogeneous primary oval cells were induced to proliferate by the cytokine. TWEAK supplementation in

homogeneous, clonal BMOL and BMOL-TAT cultures led to a growth inhibition under normal culture conditions but resulted in enhanced proliferation when the cells had been adapted to a reduced growth factor environment prior to stimulation. Knockout mice that have the TWEAK/Fn14 signalling pathway ablated and their syngenic wildtype animals were placed on a CDE diet. Quantitation of A6- and CK19-positive cells revealed a substantial suppression of the oval cell response in both TWEAK as well as Fn14 knockout mice and hence confirmed the importance of this signalling system in oval cells in vivo. The vision of this Frontiers in Oncology Research Topic on “Stem Cell Genetic Fidelity” had the goal of steeping a diverse range of research perspectives to a first comprehensive synthesis of thought on the questions of how tissue stem cells manage gene mutation rate and the significance of that management in mammalian evolution and biology, in particular as it relates to tissue cell renewal, carcinogenesis, and aging. The primary focus was determinants of mutation rate in distributed stem cells (DSCs), which encompass all naturally occurring stem cells at all stages of mammalian development. In particular, contributions were sought that considered a broad range of aspects of the immortal DNA strand hypothesis for DSC genetic fidelity. Though proposed in 1975, only in the last decade has this landmark concept in tissue cell biology emerged as a central

discussion in DSC research with increasing scrutiny and discussion by an increasing number of laboratories of diverse research perspectives and experimental approaches. With this hypothesis presenting a formidable technical challenge for experimental investigation, as would be expected, both supportive and unsupportive reports have been lining up. In the case of supportive studies, neither the range of applicable tissues nor the responsible molecular mechanisms are known; and the essential genomic process, non-random DNA template strand inheritance by asymmetrically self-renewing DSCs, has been suggested to potentially have other cellular roles besides reducing mutation rate. A major aspiration of this Research Topic was to create the first comprehensive, critical synthesis of current insights and viewpoints on the impact of the immortal DNA strand hypothesis in the history of DSC mutation research. A wide range of article types was considered including historical perspectives, critical reviews, critical commentaries, new hypotheses, new research perspectives, technical advances, and original research reports. Although treatments of the immortal DNA strand hypothesis were the major focus, the desired synthesis required integration of related ideas on mechanisms of DSC mutagenesis and its impact in the evolution of mammals, the emergence of cancers, and stem cell aging. As such, investigators focused on issues in e.g., germ stem cell mutagenesis,

effects of environmental mutagens on DSC mutation rate, DSC mutation and tissue aging, determinations of types of mutations in DSCs, and the role of DSC mutation in cancer initiation were invited. Similarly, although the specific goal of the Research Topic was to enlighten DSC genetic fidelity in humans and other mammals, informing contributions based on studies in other model organisms were also welcomed. To achieve even better representation of current experience, advances, and ideas in this field of investigation, these early contributors were encouraged to extend the opportunity to others who shared their interest in advancing our understanding of the mutability of DSCs and its significance in human biology.

"A real jewel of science history...brims with suspense and now-forgotten catastrophe and intrigue...Wadman's smooth prose calmly spins a surpassingly complicated story into a real tour de force."—The New York Times "Riveting . . . [The Vaccine Race] invites comparison with Rebecca Skloot's 2007 *The Immortal Life of Henrietta Lacks*."—Nature The epic and controversial story of a major breakthrough in cell biology that led to the conquest of rubella and other devastating diseases. Until the late 1960s, tens of thousands of American children suffered crippling birth defects if their mothers had been exposed to rubella, popularly known as German measles, while pregnant; there was no

vaccine and little understanding of how the disease devastated fetuses. In June 1962, a young biologist in Philadelphia, using tissue extracted from an aborted fetus from Sweden, produced safe, clean cells that allowed the creation of vaccines against rubella and other common childhood diseases. Two years later, in the midst of a devastating German measles epidemic, his colleague developed the vaccine that would one day wipe out homegrown rubella. The rubella vaccine and others made with those fetal cells have protected more than 150 million people in the United States, the vast majority of them preschoolers. The new cells and the method of making them also led to vaccines that have protected billions of people around the world from polio, rabies, chicken pox, measles, hepatitis A, shingles and adenovirus. Meredith Wadman's masterful account recovers not only the science of this urgent race, but also the political roadblocks that nearly stopped the scientists. She describes the terrible dilemmas of pregnant women exposed to German measles and recounts testing on infants, prisoners, orphans, and the intellectually disabled, which was common in the era. These events take place at the dawn of the battle over using human fetal tissue in research, during the arrival of big commerce in campus labs, and as huge changes take place in the laws and practices governing who "owns" research cells and the profits made from biological inventions. It is also the story of yet one

more unrecognized woman whose cells have been used to save countless lives. With another frightening virus--measles--on the rise today, no medical story could have more human drama, impact, or urgency than The Vaccine Race.

Primary cells from the anlagen of the rat suprachiasmatic nucleus (SCN) have been immortalized by infection with a retroviral vector encoding the adenovirus E1A oncogene. The resulting neural cell lines (SCN1.4 and 2.2) are characterized by extended growth potential without neoplastic activity, uniform nuclear expression of E1A protein and heterogeneous cell types in various stages of differentiation. The SCN1.4 and SCN2.2 lines exhibit many cells with glial morphologies and a small, stable population of cells with neuronal characteristics.

Differentiated neuron-like cells are distinguished by fine processes and immunostaining for neuronal markers and peptides found within SCN neurons in situ. Concordant with immunostaining data, content, release and mRNA expression of SCN neuropeptides in both lines followed a distinct pattern with somatostatin and vasopressin cells representing the most and least common peptidergic phenotypes, respectively. Since E1A-immortalized cells from the primordial SCN can differentiate into neurons with mature, parental-like phenotypes, the initial project objective was to determine whether the lines also retain the distinctive function of the SCN to generate circadian rhythms. Circadian wheel-running activity was restored in approx. 40% of SCN-lesioned hamsters following transplantation of immortalized cells, suggesting that circadian timekeeping may be a stable functional property of these lines. The project has also yielded clonal lines of immortalized cells that exhibit specific SCN phenotypes and may provide models for studying the regulation of neuropeptide gene expression and the

role of peptidergic cells in mammalian circadian timekeeping. Circadian rhythms, Biological clock, Oscillation, Suprachiasmatic nucleus, Immortalized cell lines, Transplantation. A Conspiracy of Cells presents the first full account of one of medical science's more bizarre and costly mistakes. On October 4, 1951, a young black woman named Henrietta Lacks died of cervical cancer. That is, most of Henrietta Lacks died. In a laboratory dish at the Johns Hopkins Medical Center in Baltimore, a few cells taken from her fatal tumor continued to live--to thrive, in fact. For reasons unknown, her cells, code-named "HeLa," grew more vigorously than any other cells in culture at the time. Long-time science reporter Michael Gold describes in graphic detail how the errant HeLa cells spread, contaminating and overwhelming other cell cultures, sabotaging research projects, and eluding detection until they had managed to infiltrate scientific laboratories worldwide. He tracks the efforts of geneticist Walter Nelson-Rees to alert a sceptical scientific community to the rampant HeLa contamination. And he reconstructs Nelson-Rees's crusade to expose the embarrassing mistakes and bogus conclusions of researchers who unknowingly abetted HeLa's spread.

Interest in the cell cycle has grown explosively in recent years as a result of the identification of key cell cycle regulators and their substrates. Aside from enhancing our understanding of normal cellular growth controls, this new knowledge has also been valuable in elucidating mechanisms of growth deregulation which occur in diseased states, such as cancer and, in some instances, viral or parasitic infections. The Thirteenth Washington International Spring Symposium was organized with the intention of bringing together scientists working on different aspects of the cell cycle. Scientific topics presented ranged from molecular regulators and effectors to mitosis specific changes in cell architecture to the role of the cell cycle in

development and disease. The goal of this gathering was to help formulate a more comprehensive and integrated picture of events driving and being driven by the cell cycle, as well as to evaluate the possibilities for clinical application of this knowledge. This symposium, held in Washington, D.C. from May 10-14, 1993, was attended by more than 400 scientists from 20 countries, including many of the scientific leaders in this field. This volume contains most of the papers presented at the seven plenary sessions in addition to selected contributions from a total of nine special oral and poster sessions.

The American Anti-Vivisection Society (AAVS) petitioned the National Institutes of Health (NIH) on April 23, 1997, to prohibit the use of animals in the production of mAb. On September 18, 1997, NIH declined to prohibit the use of mice in mAb production, stating that "the ascites method of mAb production is scientifically appropriate for some research projects and cannot be replaced." On March 26, 1998, AAVS submitted a second petition, stating that "NIH failed to provide valid scientific reasons for not supporting a proposed ban." The office of the NIH director asked the National Research Council to conduct a study of methods of producing mAb. In response to that request, the Research Council appointed the Committee on Methods of Producing Monoclonal Antibodies, to act on behalf of the Institute for Laboratory Animal Research of the Commission on Life Sciences, to conduct the study. The 11 expert members of the committee had extensive experience in biomedical research, laboratory animal medicine, animal welfare, pain research, and patient advocacy (Appendix B). The committee was asked to determine whether there was a scientific necessity for the mouse ascites method; if so, whether the method caused pain or distress; and, if so, what could be done to minimize the pain or distress. The committee was also asked to comment on available in vitro

methods; to suggest what acceptable scientific rationale, if any, there was for using the mouse ascites method; and to identify regulatory requirements for the continued use of the mouse ascites method. The committee held an open data-gathering meeting during which its members summarized data bearing on those questions. A 1-day workshop (Appendix A) was attended by 34 participants, 14 of whom made formal presentations. A second meeting was held to finalize the report. The present report was written on the basis of information in the literature and information presented at the meeting and the workshop.

A cell biologist who has studied the aging process for thirty years answers the big questions about aging, dispels the myths, and discusses the desirability of living longer

Explores how new organs might be engineered via cloning and reproductive technology to achieve human immortality.

In recent times, the boundary between living and non-living has been blurred by advances in genomics, cell biology, and molecular neuroscience, whereby humans are repaired, enhanced, or made anew. Scientists and physicians are now able to keep cells, organs, and bodies alive indefinitely and can return cells or DNA to our bodies and make new cells for the purpose of treating disease or growing new tissue. Meanwhile, transhuman technologies create illusions of immortality. *Immortal: Our Cells, DNA, and Bodies* synthesizes what we know about life and death from a genetic, molecular, and cellular perspective, demarcates limits of knowledge, and poses new questions. Award-winning researcher and writer David Goldman examines in-depth three keys to understanding the nature and continuity of life: 1) epigenetic (ephemeral) vs genetic (durable) transgenerational memory; 2) life's cellular nature, and the ability to make bodies from cells; and 3) the distinction between bodies and persons. Grounded in recent

scientific evidence and real-life cases that test our historical understanding of life and death, Goldman probes the nature of molecular continuity in the face of mortal extinction, encompassing how changes to the DNA code can be both long-lasting and transgenerational, and the continuous nature of cellular and molecular information transmission. In tying these themes together, Immortal asks us to apply fresh scientific concepts to examine, for ourselves, the continuity of being in the face of mortality. Applies recent genetic, molecular and cellular findings to examine the boundaries between living and non-living, and between person and non-person Examines the significance of epigenetic memory and transgenerational inheritance and their uses in molecular and precision medicine Written by a thought-leader in genetic and molecular medicine

A key player in the science of therapeutic cloning recounts the progress that has been made in reversing the aging process of cells, citing the unique qualities of stem cells and how they can be used to regenerate damaged cells and organs.

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Pre-University Paper from the year 2014 in the subject Biology - Cytology, grade: 15			
Punkte = 1,0, , language: English, abstract: All living things have to die. This			
fundamental truth is held to apply even to the smallest unit of life – cells. However,			
there is a phenomenon that is sometimes called biological immortality. It refers to cells			
that live beyond their proclaimed life span, which is roughly set by the Hayflick limit. All			
cancer cells have acquired this property; they divide indefinitely, which is the essential			
problem with cancer cells. On the other hand, researchers are very much interested in			
the molecular mechanism behind this property to may be able to use it to extend life			
and rejuvenate cells. Cells that are not subject to the Hayflick limit are generally seen			

as a threat to the human body, however, they are interesting subjects of experiments and scientists have already learned a great deal of knowledge by studying these mutants and continue to gain more important insights into the functioning of any kind of human body cell. Immortal cells can be boon and bane for humankind. Certain aspects of this issue will be discussed.

Telomerase, a ribonucleoprotein reverse transcriptase, has been detected in reproductive cells, immortal cell lines, and most cancer cells. The activity of telomerase has been shown to be absent in normal somatic cells, with the exception of stem cells. The reactivation of telomerase has been seen as an early event in most cancers, especially breast cancer. In the first year I showed that the inhibition of telomerase led to the inhibition of cell growth via telomere-based mechanisms. The inhibition of telomerase was correlated with progressive telomere shortening in these cells and, consequently, inhibition of cell growth via cell death. Furthermore, I was able to show that telomerase inhibitors can prevent the spontaneous immortalization of Li-Fraumeni Syndrome-derived breast epithelial cells, which have not reached cellular crisis, by the prevention of telomerase activation. Tamoxifen also reduced the frequency of spontaneous immortalization in these cells. Since cancer is mostly a disease of epithelial cells, we believe our unique system of normal and spontaneously immortalized human breast epithelial cells should provide a good model system to examine the effects of tamoxifen and telomerase inhibitors. These studies should lead

to new insights in preventing the occurrence or recurrence of breast cancer. So much to read, so little time? Get an in-depth summary of *The Immortal Life of Henrietta Lacks*, the #1 bestseller about science, race, and medical ethics. For decades, scientists have been using “HeLa” cells in biological research, from developing the polio vaccine and studying the nature of cancer to observing how human biology behaves in outer space. This famous cell line began as a sample taken from a poor African American mother of five named Henrietta Lacks. A cancer patient, Henrietta Lacks went through medical testing but never gave consent for the use of her cells. She died of cervical cancer in 1951, without ever knowing that the samples were intended for extensive medical research. This summary of the #1 New York Times bestseller by Rebecca Skloot tells Henrietta’s story and reveals what happened when her family found out that her cells were being bought and sold in labs around the world. With historical context, character profiles, a timeline of key events, and other features, this summary and analysis of *The Immortal Life of Henrietta Lacks* is intended to complement your reading experience and bring you closer to a great work of nonfiction.

“Infogest” (Improving Health Properties of Food by Sharing our Knowledge on the Digestive Process) is an EU COST action/network in the domain of Food and Agriculture that will last for 4 years from April 4, 2011. Infogest aims at building an open international network of institutes undertaking multidisciplinary basic research on food digestion gathering scientists from different origins (food scientists, gut physiologists,

nutritionists...). The network gathers 70 partners from academia, corresponding to a total of 29 countries. The three main scientific goals are: Identify the beneficial food components released in the gut during digestion; Support the effect of beneficial food components on human health; Promote harmonization of currently used digestion models. Infogest meetings highlighted the need for a publication that would provide researchers with an insight into the advantages and disadvantages associated with the use of respective in vitro and ex vivo assays to evaluate the effects of foods and food bioactives on health. Such assays are particularly important in situations where a large number of foods/bioactives need to be screened rapidly and in a cost effective manner in order to ultimately identify lead foods/bioactives that can be the subject of in vivo assays. The book is an asset to researchers wishing to study the health benefits of their foods and food bioactives of interest and highlights which in vitro/ex vivo assays are of greatest relevance to their goals, what sort of outputs/data can be generated and, as noted above, highlight the strengths and weaknesses of the various assays. It is also an important resource for undergraduate students in the 'food and health' arena. This book provides updated knowledge on the basic features and mechanisms of cellular aging established since its first manifestation at cellular level 40 years ago. Contributions of genetic and environmental factors, failure of genetic and cellular repair mechanisms, and the epigenetic modifications determine the final lifespan of cells. This book also provides an understanding on how aging mechanisms in mice, a most

frequently used model, differ with that of humans who receive better tumor surveillance because of stringent controls on aging mechanisms. It also appraises the use of modern technology for aging studies and its intervention. This book serves as an excellent reading on cellular aging for undergraduate students, researchers and experts of this area.

The book "New Insights into Cell Culture Technology" focuses on many advanced methods and techniques concerned with cell culture. The contributing authors have discussed various developments in cell culture methods, the application of insect cells for the efficient production of heterologous proteins, the expansion of human mesenchymal stromal cells for different clinical applications, the remote sensing of cell culture experiments and concepts for the development of cell culture bioprocess, continuous production of retroviral pseudotype vectors, and the production of oncolytic measles virus vectors for cancer therapy. This book is an original contribution of experts from different parts of the globe, and the in-depth information will be a significant resource for students, scientists, and physicians who are directly dealing with cells.["Culture" is essential for human life and also the life of a cell. - Sivakumar Gowder]

The Immortal Life of Henrietta Lacks Summary and Study Guide
The Immortal Life of Henrietta Lacks by Rebecca Skloot is a non-fiction book that tells the story of Lacks and her HeLa cells, or the immortal cell line that doctors retrieved from her cervical cancer

cells. Crown Publishing Group published the book in 2010, and it won a National Academies Communication Award the following year. This guide refers to the Crown 2010 first edition. Henrietta Lacks was a black American woman who died of cancer in 1951 at age 31. Before she died, doctors took a sample from her tumor without her knowledge or consent and used the sample for medical research. The cells in Henrietta's tissue sample, known as HeLa cells (pronounced hee-lah), were the first human cells to survive in a culture, where they thrived and multiplied. Consequently, HeLa cells have since been used in scientific research all over the world and have played a fundamental role in numerous medical advances and developments, like the polio vaccine. For over two decades, Henrietta's identity was unknown, and her family knew nothing about HeLa and the role their mother unknowingly played in medical research. By the 1970s, however, her name was publicly revealed...

Much of our knowledge of stem cells has been inferred from studies of remarkable few species. The ability to manipulate stem cells in “model” organisms such as the mouse and a few other vertebrate species has driven our understanding of basic biology of stem cells. The power and efficiency of studying model organisms, however, comes at a cost since a few species, obviously, do not reflect nature's true diversity.

Unfortunately, although all multicellular organisms seem to rely on stem cells, and although this seems to be a question of key importance for understanding the evolution of animal life, little is known about stem cells in early-branching taxa. “Stem Cells: From

Hydra to Man” illustrates that there is more than human and mouse stem cells to learn from. Reflecting an enormous growth in the knowledge of stem cells in various organisms, the book presents the conceptual language and the nature of questions, as well as a summary of the advances in our understanding of stem cells from a comparative point of view that has resulted from the development of new technology and the development of novel model organisms over the past few decades. As such this book is largely a horizon analysis of a frontier rather than a retrospective. It presents an integrative approach to animal stem cells and covers the major contributions, tools and trends in a newly emerging field: comparative stem cell biology.

#1 NEW YORK TIMES BESTSELLER • “The story of modern medicine and bioethics—and, indeed, race relations—is refracted beautifully, and movingly.”—Entertainment Weekly NOW A MAJOR MOTION PICTURE FROM HBO® STARRING OPRAH WINFREY AND ROSE BYRNE • ONE OF THE “MOST INFLUENTIAL” (CNN), “DEFINING” (LITHUB), AND “BEST” (THE PHILADELPHIA INQUIRER) BOOKS OF THE DECADE • ONE OF ESSENCE’S 50 MOST IMPACTFUL BLACK BOOKS OF THE PAST 50 YEARS • WINNER OF THE CHICAGO TRIBUNE HEARTLAND PRIZE FOR NONFICTION NAMED ONE OF THE BEST BOOKS OF THE YEAR BY The New York Times Book Review • Entertainment Weekly • O: The Oprah Magazine • NPR • Financial Times • New York • Independent (U.K.) • Times (U.K.) • Publishers Weekly • Library Journal • Kirkus Reviews • Booklist

- Globe and Mail Her name was Henrietta Lacks, but scientists know her as HeLa. She was a poor Southern tobacco farmer who worked the same land as her slave ancestors, yet her cells—taken without her knowledge—became one of the most important tools in medicine: The first “immortal” human cells grown in culture, which are still alive today, though she has been dead for more than sixty years. HeLa cells were vital for developing the polio vaccine; uncovered secrets of cancer, viruses, and the atom bomb’s effects; helped lead to important advances like in vitro fertilization, cloning, and gene mapping; and have been bought and sold by the billions. Yet Henrietta Lacks remains virtually unknown, buried in an unmarked grave. Henrietta’s family did not learn of her “immortality” until more than twenty years after her death, when scientists investigating HeLa began using her husband and children in research without informed consent. And though the cells had launched a multimillion-dollar industry that sells human biological materials, her family never saw any of the profits. As Rebecca Skloot so brilliantly shows, the story of the Lacks family—past and present—is inextricably connected to the dark history of experimentation on African Americans, the birth of bioethics, and the legal battles over whether we control the stuff we are made of. Over the decade it took to uncover this story, Rebecca became enmeshed in the lives of the Lacks family—especially Henrietta’s daughter Deborah. Deborah was consumed with questions: Had scientists cloned her mother? Had they killed her to harvest her cells? And if her mother was so important to medicine, why

couldn't her children afford health insurance? Intimate in feeling, astonishing in scope, and impossible to put down, *The Immortal Life of Henrietta Lacks* captures the beauty and drama of scientific discovery, as well as its human consequences.

The Immortal Life of Henrietta Lacks: by Rebecca Skloot | A 15-minute Key Takeaways & Analysis Preview: Rebecca Skloot's book, *The Immortal Life of Henrietta Lacks*, chronicles the life, death, and immortality of Henrietta Lacks, a young black woman whose cervical cancer cells became one of the most important factors in bringing about important scientific and medical advancements in the twentieth century. Her family, however, did not know until much later that researchers were using Henrietta's cells in their experiments. When the family learned the truth, they endured turmoil and heartache in the decades that followed... PLEASE NOTE: This is key takeaways and analysis of the book and NOT the original book. Inside this Instaread of *The Immortal Life of Henrietta Lacks*: • Key Takeaways of the book • Introduction to the important people in the book • Analysis of the Key Takeaways

The progression from local prostate tumor to lethal prostate cancer is not well understood. Although current treatments cure a majority of patients, a significant minority (~12 %) of people are diagnosed with late-stage, hormone-independent disease. As yet, the origin of the hormone-independent prostate cancer cells is unknown. In the present study, the transition to the lethal form of this disease is hypothesized to occur when a genetically-compromised tumor cell undergoes (1) an

immortalization or (2) a pluripotential step. In this work, cells from early-stage, human prostate tumors were grown in optimized culture conditions in the absence of testosterone. We then utilized the prostate tumor cells to assess the mechanisms involved in the transition from local tumor to aggressive cancer. Two separate groups of genes were used to model this transition. In one study, prostate tumor cells were retrovirally transduced to express Bmi-1 and telomerase (TERT), a combination that allows epithelial cells to bypass senescence and become an immortal cell line. In the second approach, prostate tumor cells were transduced using a four-gene, induced pluripotent stem (iPS) cell strategy that reprograms adult cells to a pluripotent embryonic stem (ES)-cell state. Cellular response to different microenvironments was assayed in vivo by subcutaneous injection, implantation under the kidney capsule with embryonic urogenital mesenchyme (a technique termed "tissue recombination"), and orthotopic engraftment into the anterior prostate of SCID/Beige mice. The results of this work demonstrate that prostate cells can be selectively cultured from local tumors. These cells express prostate stem cell characteristics and initially grow from clusters within a putative prostate tumor stem-cell niche. Bmi-1 and TERT extended cell lifespan and increased proliferation, but did not promote a malignant phenotype. Thus, immortalization of prostate tumor cells may be necessary but not sufficient to progress the disease. In contrast, the induction of pluripotency in prostate cells triggered a highly invasive phenotype. This finding suggests that the acquisition of pluripotency is a

mechanism that promotes the transition from local tumor to a more aggressive cancer. Researchers in the twentieth and twenty-first centuries identify the study of the intrinsic and external factors that influence human aging as senescence. A commonly held belief in the year 2015 is that at least some kinds of cells can replicate over long periods or even indefinitely, thereby meaning the cell does not undergo senescence (also known as replicative senescence) and is considered immortal. This study aims to provide information to answer the following question: While some scientists claim they can indefinitely culture a stem cell line in vitro, what are the consequences of those culturing practices? An analysis of a cluster of articles from the Embryo Project Encyclopedia provides information to suggest possible solutions to some potential problems in cell culturing, recognition of benefits for existing or historical culturing practices, and identification of gaps in scientific knowledge that warrant further research. Recent research suggests that hESCs, and immortalized cell lines in general, do not escape the effects of senescence. While there exists a constant change in the practices of cell culturing, a large portion of scientists still rely on practices established before modern senescence research: research that seems to suggest that cultured hESCs, among other immortal cell lines, are not truly immortal.

The ability to "immortalize" immunologically-useful cells by hybridization with a unique cancer cell has revolutionized serological studies and has revealed new potential applications in all fields of biological sciences. This volume presents the studies from a

highly successful national symposium on Hybridomas and Cellular Immortality held November 1981 in Houston, Texas. The individual chapters exhibit the diversity of topics discussed during the meeting. These include emphasis on the origin of antibody diversity, Band T lymphocyte differentiation, applications of monoclonal antibodies in studies of histocompatibility, tumor, and viral antigens, plus the use of somatic cell hybridizations for studying T cell products. Three papers focus on the emerging methodologies of in vitro primary immunizations for both humoral and cell-mediated immunities, relevant for coupling with hybridoma technology. There is a useful mix of general (methods) and specific (applications) chapters. A unique aspect of the book is the presentation of both recent research findings with concise descriptions of the state of the art methodologies. It is anticipated that this work will be of interest to a wide audience of practioners in biomedical research. Hopefully, the information contained will foster new and imagi native ideas in hybridoma applications. Baldwin H. Tom, Ph.D. James P. Allison, Ph.D. vii CONTENTS PART L INTRODUCTION TO HYBRIDOMAS 1 Somatic Cell Hybrids and Hybridomas Baldwin H. Tom 3 1. Somatic Cell Hybrids 8 Hybridomas. ••••• 2.

What is or what does a gene mutation do? A gene mutation, i.e. the damage of a gene, leads to a gene expressing more, less or modified proteins. These altered proteins alter the behaviour of the cell. And this is how cancer is ultimately to develop: Changed proteins due to changes in the genes. But what would happen if these changes in the

proteins were not caused by mutations in the genes, but by direct damage to the proteins? Then the result "cancer" would be the same, but otherwise everything would be different. And cancer could be curable. We cannot reverse a mutation, but we can reverse a mechanical process such as protein accumulation. At least we could considerably influence such a carcinogenesis. The proliferation-accumulation-tumour-hypothesis describes how this might be possible. According to PATH, cancer is not a mutation disease, but a protein accumulation disease. But a protein accumulation (protein oxidation / protein aggregation) in immortal stem cells. And a protein accumulation that affecting 400 protein types (mainly cell cycle proteins) and millions of protein copies simultaneously. A protein accumulation in which autophagocytosis in the G0 phase - as the most important proteolysis factor - is no longer sufficiently effective. PATH is based on the latest findings on the ubiquitin proteasome system, cyclins (in particular Cyclin-D-CDK4,6) and autophagy (according to Ohsumi's findings) and their role in the development of tumours. And PATH also shows why certain forms of treatment and substances (e.g. acetylsalicylic acid [aspirin], metformin, 2-deoxy-2-glucose, methadone, terfenadine, etc.) have a certain effect in studies. And how cancer could possibly be hindered in its development to such an extent that a lethal effect is omitted. For example, PATH also explains better why a person has a 50% chance of developing cancer or at least a 25% chance of dying from it. The most important basis for the development of PATH was the rigorous detachment from the

cancer multimitation principle, which has not been effective to date, and the exclusive reorientation towards the most probable and logical facts and conclusions. And the realization: „What is the cause of death for at least 25% of all people cannot be something that has to be caused by a rare, undirected coincidence (mutation).“

Henrietta Lacks was a beautiful African American woman who always painted her toenails red. She loved to dance. She had a big laugh and mischievous eyes. She had five children whom she loved with every inch of her soul. No one knows what her favorite color was. Henrietta Lacks was full life, but she died in 1951, her body consumed by tumors that had started in her cervix. She was buried in an unmarked grave and even though she was greatly loved, no one talked much about Henrietta after she died. The winds of time would have quickly swept away all signs of this vivacious woman had it not been for one thing: her cells were immortal.

In this book, the author Joseph G. Sinkovics liberally shares his views on the cancer cell which he has been observing in vivo and in vitro, over a life time. Readers will learn how, as an inherent faculty of the RNA/DNA complex, the primordial cell survival pathways are endogenously reactivated in an amplified or constitutive manner in the multicellular host, and are either masquerading as self-elements or as placentas, to which the multicellular host is evolutionarily trained to extend full support. The host obliges. The author explains that there is no such evidence that “malignantly transformed” human cells survive in nature. However, when cared for in the laboratory,

these cells live and replicate as immortalized cultures. These cells retain their vitality upon storage in liquid nitrogen. One can only imagine an astrophysical environment in which such cells could survive; perhaps, first their seemingly humble exosomes would populate that environment. Immortal cell populations so created may survive as individuals, or may even re-organize themselves into multicellular colonies, as representatives of life for the duration of the Universe. This thought-provoking book is the work of a disciplined investigator and clinician with an impeccable reputation, and he enters a territory that very few if any before him have approached from the same angles. It will appeal to researchers with an interest in cell survival pathways and those researching cancer cells.

The Immortal Life of Henrietta Lacks by Rebecca Skloot is a non-fiction book that tells the story of Lacks and her HeLa cells, or the immortal cell line that doctors retrieved from her cervical cancer cells. Crown Publishing Group published the book in 2010, and it won a National Academies Communication Award the following year. This guide refers to the Crown 2010 first edition. Henrietta Lacks was a black American woman who died of cancer in 1951 at age 31. Before she died, doctors took a sample from her tumor without her knowledge or consent and used the sample for medical research. The cells in Henrietta's tissue sample, known as HeLa cells (pronounced hee-lah), were the first human cells to survive in a culture, where they thrived and multiplied. Consequently, HeLa cells have since been used in scientific research all over the world

and have played a fundamental role in numerous medical advances and developments, like the polio vaccine.

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