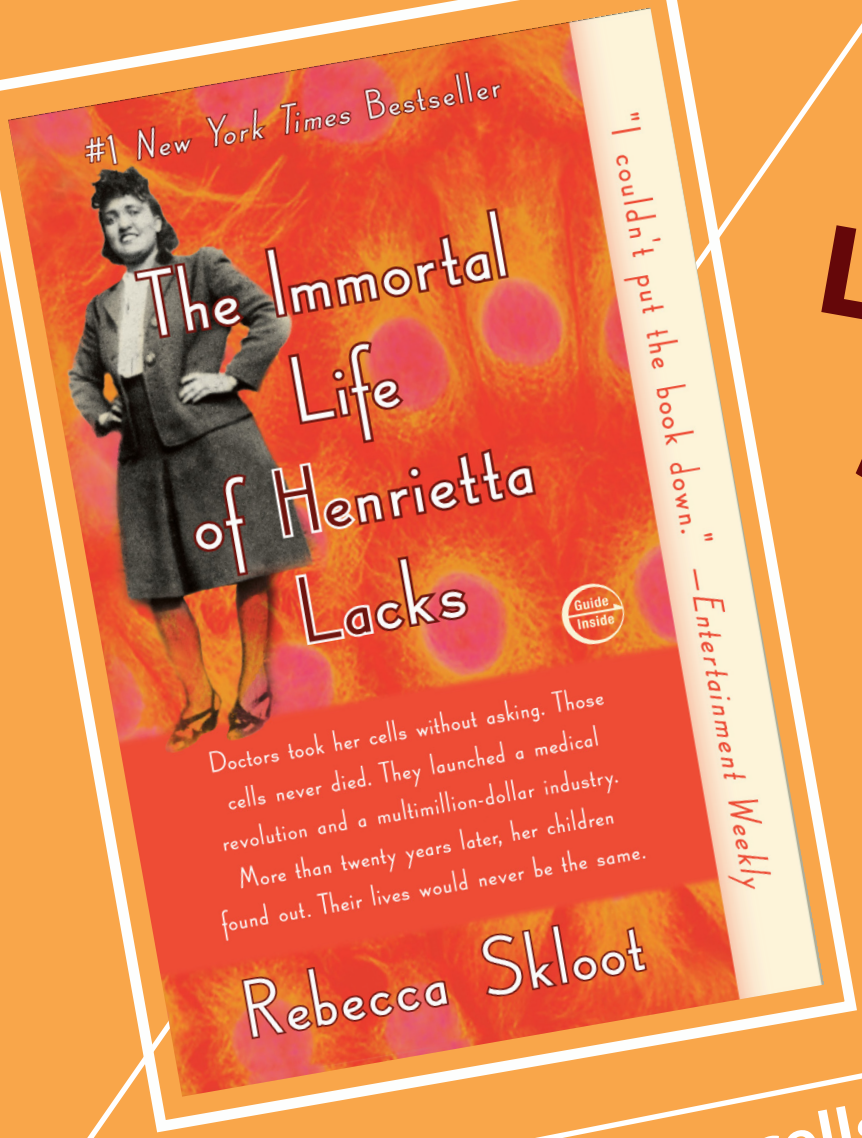

A Quick Look at the Science of HeLa Cells

The origin of HeLa cells

- HeLa cells were the first cell line, or population of cells, to be grown and survive in the laboratory.
- The cell line was created in 1951 by Dr. George Gey from a tissue sample taken from Henrietta Lacks, a cervical cancer patient.
- The cell line was named HeLa after Henrietta Lacks.
- Henrietta's cervical cancer was unusually aggressive and grew and spread rapidly.
- Normal cells don't divide indefinitely (they eventually die after about 50 cell divisions; the process of cell death is called cellular senescence), but HeLa cells are different. They are an example of what is called an immortalized cell line.
- Immortalized cells will continue to divide if given the proper environmental conditions (nutrients and space).
- Mutations (changes) required for continued cell division can occur naturally (cancer cells are a good example) or can be intentionally created for research experiments.
- HeLa cells are the most commonly used cell line in biology.

Examples of HeLa cells used in research

- In cancer research — HeLa cells became the model for cancer cells in cancer research; recently, a Nobel prize was awarded to investigators who used HeLa cells to demonstrate the link between human papillomavirus (HPV) and cervical cancer.

- In other research — Despite originating from cancer cells, HeLa cells have been used in basic biomedical research because they share many of the same basic characteristics of normal cells.

  "Since the '50s, if researchers wanted to figure out how cells behaved in a certain environment, or reacted to a specific chemical, or produced a certain protein, they turned to HeLa cells. They did that because, despite being cancerous, HeLa still shared many basic characteristics with normal cells: They produced proteins and communicated with one another like normal cells, they divided and generated energy, they expressed genes and regulated them, and they were susceptible to infections, which made them an optimal tool for synthesizing and studying any number of things in culture, including bacteria, hormones, proteins, and, especially, viruses.

  Viruses reproduce by injecting bits of their genetic material into a living cell, essentially reprogramming the cell so it reproduces the virus instead of itself. When it came to growing viruses—as with many other things—the fact that HeLa was malignant just made it more useful. HeLa cells grew much faster than normal cells, and therefore produced results faster. HeLa is a workhorse: It's hardy, it's inexpensive, and it's everywhere. " [1]

Potential Problems associated with HeLa cells

- Contamination — Because HeLa cells are easily cultured and because they are so widely used, HeLa cells may contaminate other cell lines grown in the same laboratory. As much as 20% of cell lines may be contaminated with HeLa cells.[2] As a result, results from experiments using cells lines contaminated by HeLa cells are often invalidated.

- Unusually aberrant — The complete genome of HeLa cells was sequenced and published in 2013. Analysis of the genomic sequence revealed just how unusual HeLa cells are as compared to normal (healthy) cells and even other cancer cells. Abnormalities included changes in the number and structure of chromosomes as well as evidence of a phenomenon called chromosome shattering which involves massive rearrangements in the genome involving several chromosomes. Chromosome shattering occurs in only 2-3% of all cancers.[3] The degree of HeLa cell abnormality should be considered when designing and analyzing experiments using the HeLa cell line.

1. http://rebeccaskloot.com/faq/#questions-science
2. Masters JR (April 2002). "HeLa cells 50 years on: the good, the bad and the ugly". Nat. Rev. Cancer 2 (4): 315–9. doi:10.1038/nrc775. PMID 12001993
3. P.J. Stephens, C.D. Greenman, B. Fu, F. Yang, G.R. Bignell, L.J. Mudie, E.D. Pleasance, K.W. Lau, D. Beare, L.A. Stebbings et al. Massive genomic rearrangement acquired in a single catastrophic event during cancer development. Cell, 144 (2011), pp. 27–40

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