**The Relationship Between Cancer and Telomeres**

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**Key Words:**

Cancer, telomeres, hTERT, hTR, senescence

**Cancerous cells are normal cells that have undergone some kind of mutation or experienced some form of damage that results in the unchecked and out of control growth. In normal cells the ends of chromosomes are protected by a cap, known as a telomere, that functions to protect the DNA from damage. Such damage to the DNA can lead to the development of cancer. The production of telomeres is regulated by the enzyme telomerase. As cells age the length of telomeres shortens, but for cancer cells, achievements have been made so that they can make all the telomerase they need to continue replicating out of control. Having identified this mechanism within cancer cells, researchers have attempted to target telomerase as a method of therapy in cancer treatment in the form of vaccines and chemical inhibitors that degrade telomerase and the ability of cancerous cells to utilize the enzyme for proliferation.**

**Introduction**

Cancer has been a long-studied topic. There is still much to be discovered about cancer and how cancer progresses and thrives within the human body. Cancer is complicated and varies case by case. One thing that has been discovered about cancer is that there is a relationship with tumorigenesis and telomerase. Telomerase is an enzyme that is responsible for the lengthening of telomeres. Telomeres are present within normal, healthy cells and act as a cap on the strand of DNA at the 3’ end. This capping of the DNA is initiated to protect the DNA strands from being damaged by other enzymes present within the cells and to prevent them from fusing to other strands of DNA. The downside to this protective mechanism within cells, though, is that it isn’t forever. During DNA replication the entire length of the chromosome is not replicated, which results in telomere shortening over a certain number of cell replications. The shortening of telomeres with progressive cell replications consequently leads to DNA strands becoming exposed and more at-risk for damage and/or mutations. The dysfunctional chromosomal ends of two problematic chromosomes can fuse together creating what are known as dicentric chromosomes. Dicentric chromosomes are composed of two centromeres instead of one. The consequences of a dicentric chromosome arise during cell division when the chromatids are being pulled apart at the centromere. Instead of having one centromere for the microtubules to attach to there are now two and as the microtubules pull in opposite directions there is a break in the DNA. Cancerous tumors have developed a mechanism of action to be able to use the telomerase enzyme to their advantage. By using telomerase, cancerous cells have developed the ability to grow rapidly and inconsequently. They have developed mechanisms for bypassing checkpoints within the cell-cycle that would otherwise lead to their demise because of the mutations that have occurred within the cell. The scope of the research that is addressed in this paper focuses on the relationship between the cancer cells, telomerase, and the genes involved in turning telomerase on so that the cells can proliferate uncontrollably. The research also addresses potential treatments for cancer that relate to targeting telomerase as a way to “turn off” the cancer growth. Many important questions still exist around this topic, such as questions posed to how effective treatment that targets such mechanisms is carried out safely without harming the normal somatic cells and how exactly the cancer cells are communicating with the genes to turn on the enzyme.

**Recent Progress**

Much is still to be learned about the communication process between cancer cells and how they are actually talking to the cellular programming, but it is known that cancer cells are able to increase production of certain proteins. According to Jafri 2016, “Cancer cells achieve proliferative immortality by activating or upregulating the normally silent human TERT gene (hTERT) that encodes telomerase, a protein with reverse transcriptase activity that complexes with other proteins and a functional RNA (encoded by hTR, also called hTERC) to make a ribonucleoprotein enzyme complex”. Researchers now knowing this information have made efforts to develop treatments that target this mechanism. Telomerase seems to be the target for developing cancer treatments and therapies. Researchers have identified that telomerase, when degraded, releases peptide fragments that can be recognized by the host’s immune system. Therefore, telomerase for immunotherapy is a prime target for cancer therapy and treatment. Cancerous cells also express the hTERT gene as a way to turn on telomerase, so a vaccine has been developed to target these cells that are expressing this gene. Researchers have also turned to chemical inhibitors that block telomerase enzyme activity which leads to the shortening of telomeres as opposed to maintained length within the cancer cells.

**Discussion**

Although there have been various forms of treatments developed for the treatment of cancer that involve targeting telomerase, they are still not fool proof and there are side effects and downsides to the therapies. With prolonged use, the chemical inhibitors can have toxic effects and therefore they must be temporarily stopped. This can mean all progress that has been made with the therapy is at risk of being lost. With continued research related to these mechanisms, there is a potential for scientists to develop even better modes of treatments. There are potentially better protein targets within the cells. There are potentially modes of therapy that do not have toxic or negative affects with prolonged use. With more time and research, it is hoped that new discoveries can be made and that effective treatments are developed and approved by the necessary regulatory agencies so that more people suffering from cancer can be successfully treated.

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