Telomeres and Telomerase

Telomeres, the ends of chromosomes, are composed of long, repeating sequences of DNA (see Figure 1). In normal somatic cells, the ends of telomeres cannot be replicated prior to cell division, when the rest of the chromosome is duplicated (Allsopp & Weissman, 2002). Therefore, daughter cells’ chromosomes are minutely shorter than those of the parent cell after normal cell division. Cell division results in progressive shortening of each chromosome, such that after a finite number of cell divisions, telomeres become too short and the cell cannot divide further; this state is called senescence (Serrano, 2010). However, an enzyme called telomerase that rebuilds the telomere after each cell division is present in embryonic cells and in most cancer cells. Reports have shown telomerase activity in 80%-90% of cancer cells (Harley, 2008). Because the chromosomal length is maintained, cells with telomerase activity are immortal, meaning they can divide indefinitely. If the telomerase enzyme were prevented from working, cancer cells may undergo senescence and fail to divide further; therefore, the development of therapies that target telomeres or telomerase is an active research area. Figure 2 lists definitions of terms.

Research is focusing on using telomerase activity or telomere length as a prognostic and diagnostic indicator. Analysis of the activity level of telomerase may give insight into the likelihood of recurrence of a particular cancer because higher levels of telomerase activity have been correlated to higher chances of tumor recurrence (Tatsumoto et al., 2000). Therefore, knowledge of telomerase activity level or telomere length in patients with cancer may help healthcare providers plan appropriate treatment to combat cancer progression.

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Blackburn and Gall (1978) discovered the existence of tandem repeats (5'-CCCCAA-3') located at the ends of ribosomal genes in the protozoan *Tetrahymena thermophilia.*