In the 1930s, B. McClintock and H. Muller discovered that the broken ends of chromosomes were both unstable and reactive. More recent discoveries of mechanisms that enable the replication of 3' ends of chromosomes and the telomeres that have evolved in eukaryotic nuclei seem to provide a solution to both problems. Research carried out with E. Blackburn has elucidated the existence and action of telomerase, a DNA polymerase. Telomerase contains a molecule of RNA that serves as a template for the extension and protection of the ends of linear chromosomes.

Cell immortality is a natural characteristic of eukaryotic microorganisms, including the ciliate Tetrahymena in which telomeres are long and telomerase is abundant. By contrast, differentiated cells, including most animal cells, lose telomerase activity, and their telomere length decreases with each cell generation. Thus, telomeres are somehow involved with the molecular clock that determines the lifespan of a mortal cell.

Within the past year, researchers have established that the reactivation of telomerase activity is one feature of the immortalization of previously differentiated cells. Because such cells are malignant, academic and commercial laboratories are mounting an intensive effort to seek or design drugs that would antagonize telomerase and thereby serve as anti-cancer chemotherapeutic agents. While studies on a tiny microorganism have elucidated a major feature of our own mortality and a potentially effective approach to relief from malignancy, they have also provided an example of how basic research may have unpredictable applications.