

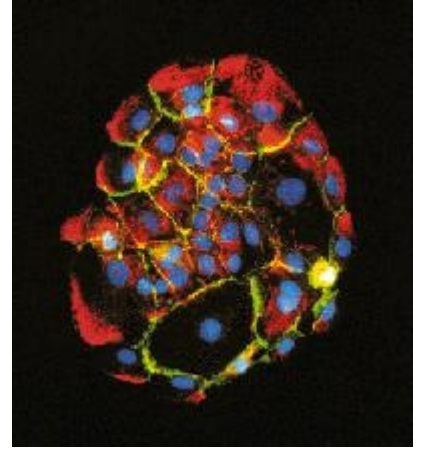
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REPORT

Fighting Cancer:
Is This A Magic Bullet?

By Mary Nucci

Highly malignant skin cancer cells. The nuclei are blue, cytoplasm red and membrane green.
From Geron Corp.



Enormous national attention has lately been drawn to the work of Harvard researcher Judah Folkman, whose discovery of processes that starve cancer cells of blood, thus shrinking them to microscopic size, promises a way to eliminate cancer from the body. Telomere theory, by contrast, by using the telomerase "switch," would attack the seeming immortality of cancer cells at the gene level, enabling them to die in due time, as they "should."

While telomerase activation appears to be crucial to the development of cancer, Dr. Matthew Meyerson of the Whitehead Institute for Biomedical Research notes, "Telomerase of itself does not cause cancer, but is postulated to permit the prolonged survival of cells." Exactly how the telomerase gene is activated is not understood. It is known that, over the course of the normal life span of a cell, there are mutations of the cell genes. Inherited mutations, radiation, chemical carcinogens, environmental pollutants and spontaneous mistakes in copying of the DNA during cell division all can lead to mutations in the DNA itself.

The cell has mechanisms to repair this damage, but should those repair mechanisms become affected, or when a significant level and type of mutation occur, the cell becomes "pre-malignant." It is at this crisis point that researchers believe telomerase is activated, and control of cell division is lost. When that happens, the cell becomes cancerous and rapidly proliferates out of control, resulting in the unchecked development of cancer at the site or throughout the body. Exploiting this activation may present an effective cancer therapy by deactivating the same gene.

"From my point of view, the most significant part of this research is the ability to make a new cancer treatment that is broadly applicable," says Meyerson. Manipulating the gene to turn off the cancer cell would not in itself eliminate the cancer. Instead, as a cancer cell with an inactive telomerase gene goes through the division process, it would lose telomeric units. Thus, instead of being immortal, the cell would be mortalized. Eventually, after the programmed limit had been reached, the cancer cell would age and die just like a normal, non-cancerous cell.

A patient suffering from cancer would most likely receive the anti-telomerase product along with radiation or chemotherapy therapy.

The benefit of telomerase inhibition, as compared with existing therapies, would be that the cancer cells would live out their normal cellular life span and then die, and the anti-telomerase inhibitor would likely have no effect on normal cells where the telomerase gene is inactive. Unfortunately, male reproductive cells and immune cells that have telomerase activity also will likely be affected by anti-telomerase. As most cancers occur in later years when patients have had children, any potential side effects on male reproductive cells might be an acceptable trade-off for cancer eradication. Or, patients could store sperm for future use.

The potential negative effect on immune cells will be more problematic, requiring constant monitoring to avoid complications from bacterial or viral infections. Current experience with patients with compromised immune systems will help to make management of this potential side effect easier. In reality, though, the side effects of telomerase will not be known until such time as the animal and human clinical studies have been initiated.

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