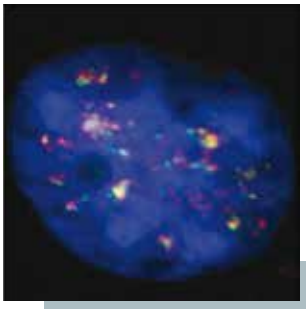

salk
Where cures begin.

Beverly Emerson

Professor
Regulatory Biology Laboratory
Edwin K. Hunter Chair

The Problem

Just as adults might not stay healthy if they only ate liquid baby formula, new tumors have a different set of requirements than more mature, established cancers to thrive and spread. So when researchers discover a new gene that—when mutated or turned on—seems to drive the growth of cancer, their findings might not hold true in all the stages of a tumor's growth. To develop new drugs and determine what treatments are most appropriate for different tumor stages, scientists need to work out what changes occur at a molecular and genetic level as a tumor progresses.

The Approach

Beverly Emerson studies how different genes are turned on and off through the course of a cancer—from the time cells become precancerous until the time they develop into a mature cancer and spread to new organs. Many researchers look for genes that are mutated in tumors, as these mistakes in the DNA code can lead to cancer. But Emerson's lab looks at other ways genes can be turned on and off to allow a tumor to grow. She's found that the physical arrangement of DNA inside a cell's nucleus can affect cancer genes: for example, if a gene gets stuck in a folded-up piece of DNA, proteins that normally turn it on can no longer access it.

Her lab also studies how different proteins (and their mutations) interact in cancer cells. Looking at cancer genetics in this broader way is helping Emerson to discover new drug targets that may be used to prevent or treat cancers.

The Innovations and Discoveries

- Emerson and her team uncovered details about how cancer is able to become drug resistant over time. They showed how variations in breast cancer cells' RNA, the molecule that decodes genes and produces proteins, helps the cancer to evolve more quickly than previously thought. The findings may point to a "switch" to turn off this diversity–and thereby drug resistance–in cancer cells.
- Emerson discovered how the gene COX-2, involved in inflammation, is turned on in the early stages of a cancer to help it grow and then off again in a more mature tumor to protect it from immune responses. Developing a way to turn COX-2 back on in advanced colon, breast, and pancreatic cancers might help the immune system shrink tumors.
- She pieced together how two proteins, p53 and TGF-ß, interact as a tumor matures. While scientists previously thought that a drug targeting TGF-ß would only be useful in advanced cancers, she discovered that the same drug may help prevent precancerous cells from turning into cancer.

For more information, please visit:
http://www.salk.edu/faculty/emerson.html

Cancer, Epigenetics, Genetics, Therapeutics