'Cheater' Cells May Spur Cancer Growth

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(PhysOrg.com) -- A new study from the Yale School of Medicine reveals a biological struggle within tissue that allows some damaged cells to survive and proliferate, eventually leading to cancer. The study is published in the online edition of *Cell Stem Cell*.

It is known that cells damaged by radiation exposure and other cancer-causing factors activate the tumor suppressor protein called p53, which orchestrates DNA damage repair. Less is known, however, about long-term consequences of DNA damage in individual cells.

The Yale team discovered that stem and progenitor cells in the blood can “remember” for many months whether they had DNA damage, and engage in a kind of competition with each other for survival.

Working with mice, the researchers found that cells within the same tissue report the extent of their damage to each other. Under normal circumstances, this causes healthier cells with lower p53 activity to expand and gradually replace those in worse shape with higher p53 levels. This strategy helps the body get rid of its most impaired cells.

But the p53 gene itself is the most common target for mutations that occur in cancer cells. According to lead author Ruslan Medzhitov, Ph.D., professor of immunobiology at Yale School of Medicine, member of the Yale Cancer Center and a Howard Hughes Medical Institute investigator, the cell with mutant p53 becomes a “cheater”: it loses the ability to sense the degree of damage in itself and fails to report its past damage to other cells in the same tissue.

Medzhitov says, “Without the ability to report damage, precancerous cells gain an advantage over normal, healthy cells and begin outcompeting them. If they are long-lasting stem or progenitor cells in the blood, this can give rise to millions of other cells in the body. This may contribute to the first steps in cancer development.”

Provided by Yale University