

Study proposes new theory of how viruses may contribute to cancer

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A new study suggests that viruses may contribute to cancer by causing excessive death to normal cells while promoting the growth of surviving cells with cancerous traits. Viruses may act as forces of natural selection by wiping out normal cells that support the replication of viruses and leaving behind those cells that have acquired defects in their circuitry.

When this process is repeated over and over, cancer can develop say study authors, led by Preet M. Chaudhary, M.D., Ph.D., professor of medicine at the University of Pittsburgh School of Medicine. Their findings are published by Public Library of Science in the Oct. 24 issue of PLoS ONE.

Infection with viruses has been linked to many human cancers, including some forms of Hodgkin's and non-Hodgkin's lymphomas, sarcomas and cancers of the throat and liver. Over the years, scientists have proposed a number of mechanisms to explain this link. One commonly held belief is that when a virus infects a cell, its genetic material alters the cell, making it grow uncontrollably, eventually leading to cancer. Some viruses also are thought to promote cancer by causing chronic inflammation. In his study, Dr. Chaudhary proposes that viruses also can lead to cancer in a less direct manner.

“We believe a separate mechanism may be at play in which a cellular insult, such as infection with a virus, selects a few pre-existing mutated clones of cells, promotes their further growth and multiplication, eventually leading to the emergence of fully cancerous cells.

Consequently, similar to the role played by natural selection during evolution, excessive cell death, rather than its absence, may be a defining force that drives the initial emergence of cancer,” said Dr. Chaudhary. He named this model the Phoenix Paradigm in which cancer theoretically arises out of the ashes of dead cells.

The paradigm was developed based on a study of cells infected with the Kaposi’s sarcoma associated herpesvirus, or KSHV, also known as human herpesvirus 8 (HHV-8). The researchers examined a gene called K13 that activates a pathway previously implicated in cancer development. Cells with low K13 expression allowed KSHV to replicate, and these cells subsequently died off, the researchers noted. Cells with higher expression of K13 emerged after KSHV replication and showed defective expression of two key proteins that are known to promote cancer.

“This paradigm, if validated by further studies, has implications not only for an improved understanding of the processes involved in cancer, but also for the development of effective strategies for its prevention and treatment,” said Dr. Chaudhary.

Source: University of Pittsburgh

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