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Percentage of cancers linked to viruses potentially overestimated

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The results of a large-scale analysis of the association between DNA viruses and human malignancies suggest that many of the most common cancers are not associated with DNA viruses. The findings, published in the August 2013 issue of the *Journal of Virology*, challenge earlier studies suggesting as high as 40 percent of tumors are caused by viruses.

For years scientists believed viruses played a role in the development of maybe 10 to 20 percent of cancers. In 2011, scientists at the Karolinska Institute in Sweden identified potential viral links to several cancers not previously associated with viruses, including brain tumors and prostate cancer, suggesting the real number could be as high as 40 percent. Since then, researchers have been working hard to find more associations, in part because viruses could provide targets for vaccines to prevent or cure these cancers.

To better understand the role of DNA viruses in human cancers, researchers from the MD Anderson Cancer Center in Houston sequenced RNA from 3775 malignant tumor samples from The Cancer Genome Atlas and then applied a robust bioinformatics algorithm to survey them for the presence of viral transcripts.

Those cancers not associated with DNA viruses included acute myeloid leukemia, cutaneous melanoma, low and high-grade gliomas of the brain (the latter killed Senator Edward Kennedy), and adenocarcinomas of the breast, colon and rectum, lung, prostate, ovaries, kidneys, and thyroid.

The findings, says Xiaoping Su, an author on the study, suggest the estimate that 40 percent of tumors are virus-related "should be much lower."

"The search for virus associations in these malignancies has consumed the efforts of many investigators," says Su, implying that his large-scale effort will spare researchers fruitless investigations.

The study also provides the framework for understanding how viruses integrate into cancer subtypes such as hepatocellular cancer, says Su. That might make it possible to personalize treatments by targeting genes that are located within known integration sites and that might be drivers of cancer initiation and progression. A key finding was that there are specific sites where viruses integrate into the host genome prior to initiating cancer, and that these sites are frequently located within particular host genes.

"This study highlights the importance of bioinformatics in defining the landscape of virus integration across cancer subtypes," says Su.

More information: www.asm.org/images/Communicati … /0713viruscancer.pdf

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