Targeting hit-and-run cancer viruses

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Viruses that can invade host cells, initiate cancer and then flee from their own trail of destruction could be stopped in their tracks, say researchers writing in the September issue of the *Journal of General Virology*.

Scientists at the University of Cambridge have not only provided the first unequivocal evidence for the 'hit-and-run hypothesis' - explaining how some viruses might cause cancer and then mysteriously disappear - but have also shown how a vaccine could arrest them. Equivalent vaccines could help prevent not only known virus-induced human cancers, such as Burkitt's lymphoma, but also cancers currently unsuspected of having a viral origin.

The team experimented with a mouse herpesvirus - similar to the human Epstein-Barr virus that causes Burkitt's lymphoma. The virus was engineered to trigger oncogenic changes at high frequency in infected cells, leading to cancerous tumour growth. Surprisingly, the cancers soon lost any sign of their previous viral infection. The group went on to show that vaccinating the mice with a modified version of the same virus protected them against subsequent cancer development.

Drs. Philip Stevenson and Stacey Efstathiou, who led the study, explained how some viruses that establish 'latent' infections (enabling them to persist in host cells without activating the immune system), cannot necessarily maintain those infections in cancer cells. This means they can initiate cancers or play a role in their development, but then can disappear from tumour cells by the time the cancer is clinically detected.

They believe that immunisation against such viruses could be an effective and relatively simple means of preventing some cancers - much as vaccination is being used to protect women against cervical cancer caused by the human papillomavirus (HPV).

However, the team's findings highlight that the link between viruses and cancer is not necessarily straightforward. "We have shown that some cancer-initiating viruses may subsequently leave the cell without trace, meaning that some viruses may contribute to more human cancers than suspected," said Dr Stevenson. "The positive side of this is that developing an effective vaccine against oncogenic herpesviruses could prevent more cancers than might be estimated from those carrying viral genomes," he said.

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