

How HPV Hijacks the Genome to Drive Cancer Development

The human papillomavirus (HPV) genome can insert itself into host chromosomes and “turn on” tumor-causing genes.

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Human papillomavirus (HPV) was first identified as a cancer driver in the 1970s, when a German doctor named Harald zur Hausen discovered that the virus causes about 75% of human cervical cancers. HPV has since been linked to several other types of human cancer, including head and neck cancer, as discovered by then-Damon Runyon Clinical Investigator Maura L. Gillison, MD, PhD, in 2000.

Over the past two decades, Dr. Gillison and her colleagues at The University of Texas MD Anderson Cancer Center have become leading experts in HPV-linked cancer development and prevention. But while they have established that HPV integrates its genome into the host cell’s DNA, how exactly the virus alters the structure of human chromosomes—and how these alterations drive tumor growth—has remained unclear.

Now, findings from Dr. Gillison’s lab at last uncover a method to the virus’s madness. By sequencing long regions of DNA from head and neck cancer cells, the team identified a striking new form of genomic variation that they termed “heterocateny.” Heterocateny, which translates to “variable chain,” describes the highly diverse but repetitive patterns of virus and host DNA segments within a cancer cell genome.

These patterns result from the HPV genome inserting itself into host chromosomes at designated break points, allowing the virus to hijack gene expression and “turn on” oncogenic genes. (Remember, viruses and cancers are aligned in their goal: to grow and multiply as much as possible.) Recombining the host DNA in different ways also increases heterogeneity among tumor cells, which makes them harder to target therapeutically and increases the odds of some tumor cells gaining an evolutionary advantage.

The team’s heterocateny model offers the first mechanistic understanding of how HPV wrests control of the host cell genome, driving the creation and development of tumors. In addition to solving that long-standing mystery, these findings may generate new insights into other oncogenic viruses, including Merkel cell polyomavirus and hepatitis B virus.

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