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Non-homologous recombination in HSV1

Alina Shitrit, Enosh Tomer and **Oren Kobiler**
Sackler School of Medicine - Tel Aviv University, Israel

Herpes simplex virus type1 (HSV1) is a pathogenic double stranded DNA virus, which can be found in over 80% of the global human population and poses a serious health risk to immunocompromised individuals. HSV1 has a 149 kb linear genome which replicates solely within the host cell nucleus. During replication, the viral genome undergoes homologous recombination both between copies of a single genome in sequence repeats and between separate replicating genomes. These recombination events are considered to be frequent and are detectible in tissue culture experiments as well as in clinical isolates. These events are mediated by viral proteins and components of the cellular DNA repair machinery. Homologues recombination is essential to viral DNA replication and formation of viral progeny. Using a florescent plaque assay, we show a novel form of intergenomic recombination that does not require sequence homology and results in viral gene duplication. This type of non-homologues recombination varies in frequency between various cell types and is inhibited by the viral E3 ubiquitin ligase ICP0. Nanopore sequencing of HSV1 genome isolated from U2OS infected cells reveals 7% of the reads with one or more deletion and 10.5% of the reads with one or more insertion. These findings support the non-homologous recombination mechanism used by the HSV1 and may have an important role in generating viral diversity.

alinastaro@gmail.com