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Does altered N-glycosylation of influenza A virus hemagglutinin in Vero cells prevent efficient virus replication?

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Influenza is an enveloped, single-stranded negative-sense RNA virus. Two glycoproteins – the hemagglutinin (HA) and the neuramidase (NA) – are incorporated into the viral lipid envelope which is derived from their host cell. Both membrane proteins play an essential role in virus replication: HA allows host cell binding and initiates pH-induced membrane fusion whereas NA prevents the budding viruses from clumping to each other or to the cell surface. During virus replication small changes in the viral genome happen continually over time. This antigenic drift produces new virus strains that may not be recognized by the body's immune system requiring the need for seasonal reformulation of influenza vaccines.

In this study we investigated the impact of adaptation of influenza A/PR/8/34 (*H1N1*) virus to new host cells. We therefore adapted Madin-Darby canine kidney (MDCK) cell-derived PR-8 virus to replication in Vero cells. After 5 passages, the Vero cell-adapted strain was used again to infect MDCK cells. We found that due to specific properties of the new host cell line (e.g. HA *N*-glycosylation profile), the virus required a rescue mutation within the stem region of the HA to replicate efficiently. We furthermore show, an increase in the number of virus variants (quasispecies), and changes in the time course of genome composition along the adaptation and back-adaptation processes.