Virucidal effect of lipids on visna virus, a lentivirus related to HIV.

Hilmar Hilmarsson, Lárus V. Lárusson and Halldor Thormar, Institute of Biology, University of Iceland. hilmarh@hi.is

Studies of virucidal activity profiles of fatty alcohols and lipids have shown that saturated medium-chain fatty acids and their corresponding monoglycerides and fatty alcohols rapidly inactivate viruses such as herpes simplex virus (HSV) and respiratory syncytial virus (RSV), whereas other lipids have much less or no activity. A virucidal profile of these compounds against visna virus (VV), a lentivirus of sheep related to HIV, or against other viruses of the genus *Lentivirus* has not been established before and could help elucidate how lipids inactivate enveloped viruses and assist in the development of virucidal drugs. Stock solutions of 1 M alcohols and lipids in ethanol were diluted to 10 mM or lower concentrations and mixed with an equal volume of VV. The mixtures were incubated for 10 min at 37°C or 1 min at room temperature and then titrated by inoculation of 10-fold dilutions into monolayers of sheep choroid plexus (SCP) cells in tissue culture plates and examined for cytopathic effect for 10 days. The most active compounds against VV were found to be fatty acids, particularly lauric, palimitoleic and oleic acids, when tested either in low concentrations or at shorter incubation time. This is in contrast to our previous studies on other enveloped viruses, where lipids of medium chain lengths , especially the 10-carbon monoglyceride monocaprin, were found to be most virucidal. There is therefore a distinct difference in the activity profiles of HSV, RSV and VV, possibly caused by the difference in their envelope proteins. The finding that fatty alcohols and lipids are more active against VV at pH 4.2 than at pH 7 is in agreement with earlier studies, which have shown that lipids become generally more virucidal at low pH. The increased virucidal activity against enveloped viruses at low pH may be due to ionic changes in the glycoproteins on the surface of the viral envelope, thus giving the lipid molecules better access to the lipid bilayer of the envelope in acidic environment than at neutral pH.