# OA The broad-spectrum antiviral drug arbidol inhibits foot-and-mouth disease virus replication

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**First Published Online:** 08 April 2019, Access Microbiology 1: doi: (Digital Object Identifier) 10.1099/acmi.ac2019.po0191 (https://dx.doi.org/10.1099/acmi.ac2019.po0191)

**Subject:** Oral Abstract - Virology Workshop: Antivirals and Vaccines

Cover date: 01/03/2019





Arbidol (arb, umifenovir) is used clinically in several countries as an anti-influenza virus drug. We have previously shown that arb inhibits many viruses including hepatitis C virus, Ebola and Zika, and that the primary mode of action appears to be via inhibition of virus entry and/or fusion of viral membranes with intracellular endosomal membranes. We have also shown that arb is a good inhibitor of (non-enveloped) poliovirus types 1 and 3. Here, we evaluate the antiviral potential of arb against another picornavirus, foot-and-mouth disease virus (FMDV), an important veterinary pathogen. Sub-cytotoxic concentrations of arb inhibited the replication of FMDV replicon RNA. arb inhibition of FMDV RNA replication was not a result of generalised inhibition of uptake of cargo (e.g. plasmid DNA or RNA), nor did arb inhibit FMDV replication when added at 4 h post-transfection of FMDV replicon RNA. FMDV replication was blocked by the replication inhibitor guanidium hydrochloride (GuHCl). Upon GuHCl removal, FMDV replication was restored, and arb suppressed this recovery of virus replication. For other picornaviruses, recovery of virus replication upon GuHCl removal has been shown to require translation. However, arb did not suppress cap- or internal ribosome entry site (IRES)-dependent translation. arb also inhibited infectious equine rhinitis associated virus (ERAV), a close relative of FMDV. Testing of arb against infectious FMDV is in progress. Collectively, the data demonstrate that arb inhibits certain picornaviruses by a mechanism that appears to be independent of effects on virus entry but involves inhibition of genome replication.



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