

# Researchers develop new concept to predict universal anti-influenza drugs

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Credit: University of Hertfordshire

University of Hertfordshire researchers have developed a new concept which could lead to the discovery of universal anti-influenza drugs.

It is hoped that this new way of including more than 12,000 [influenza virus](#) gene sequences will ensure that predicted antivirals are effective

against most influenza virus strains. The findings are published in this month's edition of the journal *Virology*.

To predict the drugs, researchers first characterised the [drug](#) target – the viral PB2 protein required for the virus to become infectious – by analysing 12,000 sequences to assess its variability and identify constant regions. Secondly, they computationally scanned the PB2 protein surface for binding sites and then screened more than 40,000 molecules for binding.

They also screened 1738 small molecule drugs which have been approved for humans and predicted that the antipsychotic paliperidone binds to the influenza PB2 protein.

The results of this work enables laboratory-based virologists to test these computationally predicted drugs, in order to take the research onto the next stage.

Dr Andreas Kukol from the University of Hertfordshire said, "The influenza A virus causes human flu, [bird flu](#) and swine flu and also affects other animals, however there is so much variability in the virus that many potential drugs are only effective for a short period before the virus becomes resistant - hence the need for this study, to help discover antivirals which are effective against most strains."

Hershna Patel, a PhD student working on the project added, "Our research is a good foundation for developing flu antivirals that may be universally effective in human and veterinary medicine. Identifying a common theme among the 12,000 sequences, ensures that the virus is unlikely to mutate and become resistant. Further work will now be required to test the computationally predicted drugs and find out if they do in fact inhibit [virus replication](#)."

Provided by University of Hertfordshire

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