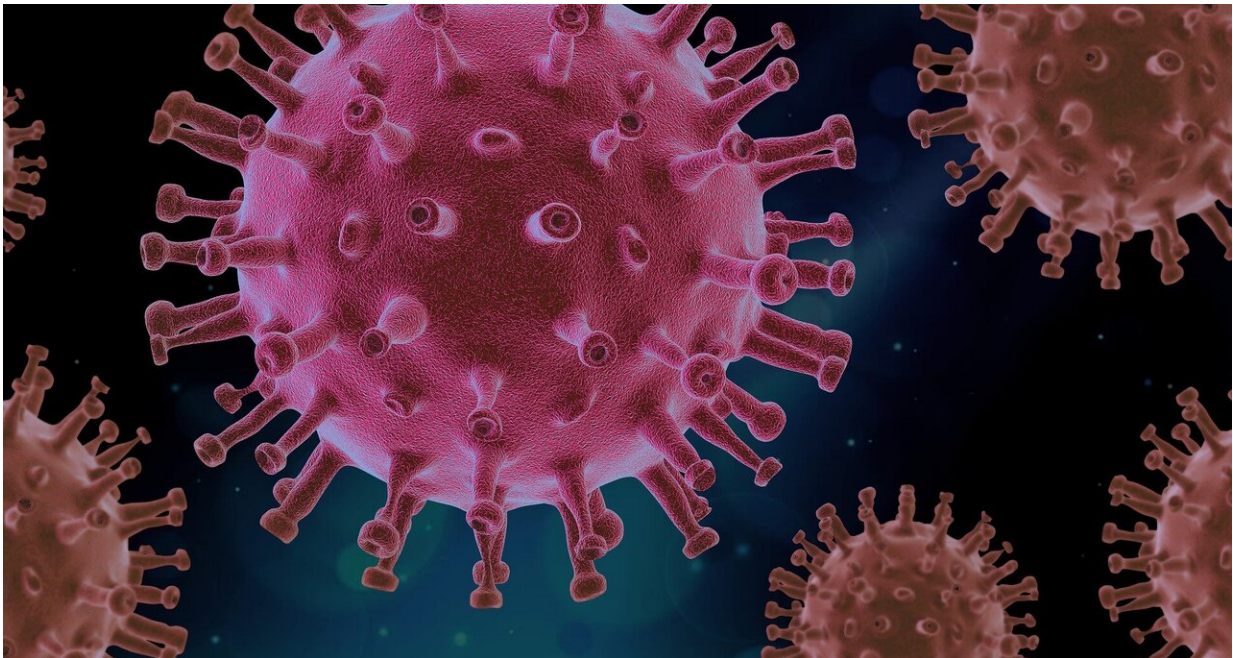


Potential new treatment for COVID-19 identified

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Researchers have identified a potential new treatment that suppresses the replication of SARS-CoV-2, the coronavirus that causes COVID-19. In order to multiply, all viruses, including coronaviruses, infect cells and reprogram them to produce novel viruses. The research revealed that cells infected with SARS-CoV-2 can only produce novel coronaviruses when their metabolic pentose phosphate pathway is activated.

With the application of the drug benfooxythiamine, an inhibitor of this pathway, SARS-CoV-2 replication was suppressed and infected cells did not produce coronaviruses.

The research from the University of Kent's School of Biosciences and the Institute of Medical Virology at Goethe-University, Frankfurt am Main, found the drug also increased the antiviral activity of '2-deoxy-D-glucose'; a drug which modifies the host cell's metabolism to reduce virus multiplication.

This shows that [pentose phosphate](#) pathway inhibitors like benfooxythiamine are a potential new [treatment](#) option for COVID-19, both on their own and in combination with other treatments.

Additionally, benfooxythiamin's antiviral mechanism differs from that of other COVID-19 drugs such as remdesivir and molnupiravir. Therefore, viruses resistant to these may be sensitive to benfooxythiamin.

Professor Martin Michaelis, University of Kent, said, "This is a breakthrough in the research of COVID-19 treatment. Since resistance development is a big problem in the treatment of viral diseases, having therapies that use different targets is very important and provides further hope for developing the most effective treatments for COVID-19."

Professor Jindrich Cinatl, Goethe-University Frankfurt, said, "Targeting virus-induced changes in the host cell metabolism is an attractive way to interfere specifically with the virus replication process."

More information: Denisa Bojkova et al, Targeting the Pentose Phosphate Pathway for SARS-CoV-2 Therapy, *Metabolites* (2021). [DOI: 10.3390/metabo11100699](https://doi.org/10.3390/metabo11100699)

Provided by Goethe University Frankfurt am Main

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