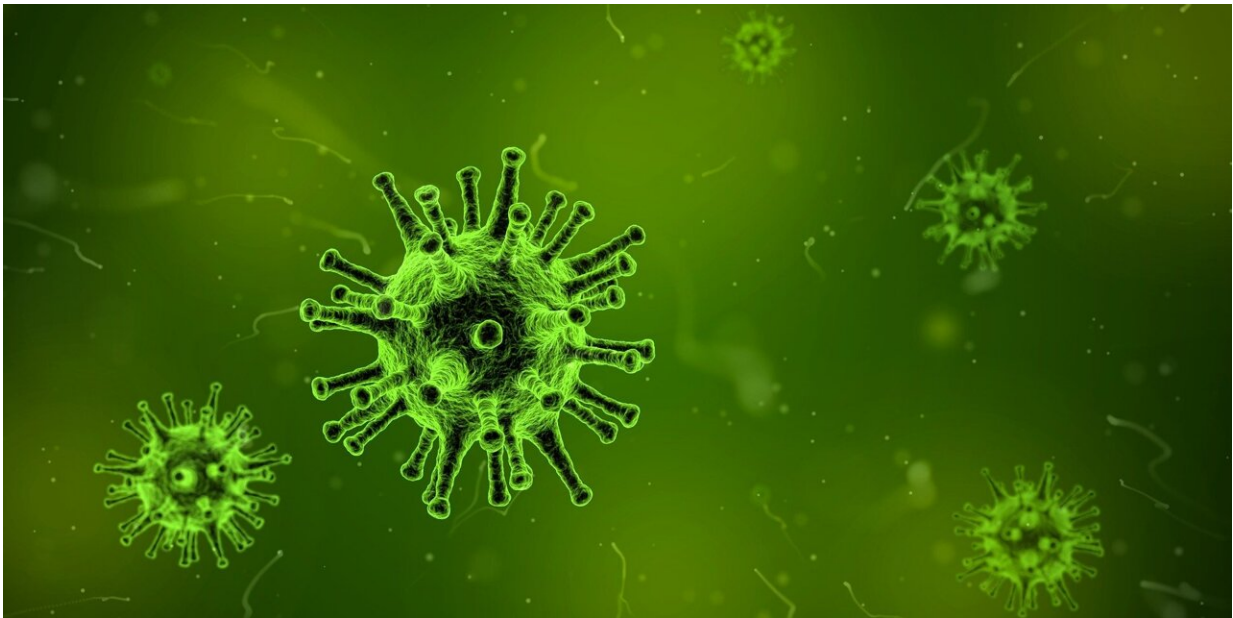


# Mimicking the body's own defenses to destroy enveloped viruses

August 2 2023

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Just as bacteria can develop antibiotic resistance, viruses can also evade drug treatments. Developing therapies against these microbes is difficult because viruses often mutate or hide themselves within cells. But by mimicking the way the immune system naturally deals with invaders, researchers reporting in *ACS Infectious Diseases* have developed a "peptoid" antiviral therapy that effectively inactivates three viruses in lab tests. The approach disrupts the microbes by targeting certain lipids in

their membranes.

Viruses are almost like biological "zombies." They are not quite living or nonliving, and are only able to multiply within a host, such as our body's cells. Oftentimes, the immune system naturally destroys the pathogens with special molecules such as antibodies.

Less-well-known members of the [immune system](#)'s defense force are small protein-like molecules called [antimicrobial peptides](#). These peptides aren't good drug candidates, though, as they're expensive to make, are quickly eliminated from the body and can cause side effects. Instead, some researchers have mimicked their function with lab-made molecules called peptoids that aren't easily degraded by the body and are more economical to produce.

[Previously](#), Annelise Barron's team showed that certain peptoids could pierce and destroy the SARS-CoV-2 and herpes [viruses](#). This time, joined by Kent Kirshenbaum and colleagues, the group wanted to see if the peptoids could inactivate three other "enveloped" viruses enclosed within membranes—Zika, Rift Valley fever and [chikungunya virus](#)—as well as one that lacks a membrane envelope, coxsackie B3. No treatments currently exist for infections caused by these microbes.

The peptoids used in these experiments included three of the linear peptoids previously studied by Barron's team, as well as four new circularized versions with increased antiviral activity. The researchers created model virus membranes using common lipids, including phosphatidylserine (PS). Membranes were disrupted most effectively when PS was present in higher concentrations, suggesting that the peptoids target it specifically.

Though both human and viral membranes contain the lipid, it's distributed differently in each instance, allowing an antiviral to

preferentially attack the invader instead of the host. Next, the team incubated the peptoids with whole, infectious virus particles. Again, each worked to a different extent on the three enveloped viruses: some disrupted all three, some only one. However, none of the peptoids could inactivate the non-enveloped coxsackie B3 virus, showing that the mechanism of action hinges on the presence of the viral envelope.

The team says that understanding this mechanism could inform the design of future peptoid-based antiviral treatments, and could be used to create drugs already armed against the next emerging viral threat.

**More information:** Peptidomimetic Oligomers Targeting Membrane Phosphatidylserine Exhibit Broad Antiviral Activity, *ACS Infectious Diseases* (2023). DOI: [10.1021/acsinfecdis.3c00063](https://doi.org/10.1021/acsinfecdis.3c00063).  
[pubs.acs.org/doi/abs/10.1021/acsinfecdis.3c00063](https://pubs.acs.org/doi/abs/10.1021/acsinfecdis.3c00063)

Provided by American Chemical Society

Citation: Mimicking the body's own defenses to destroy enveloped viruses (2023, August 2) retrieved 12 May 2024 from <https://medicalxpress.com/news/2023-08-mimicking-body-defenses-destroy-enveloped.html>

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