Can the common cold help protect you from COVID-19?

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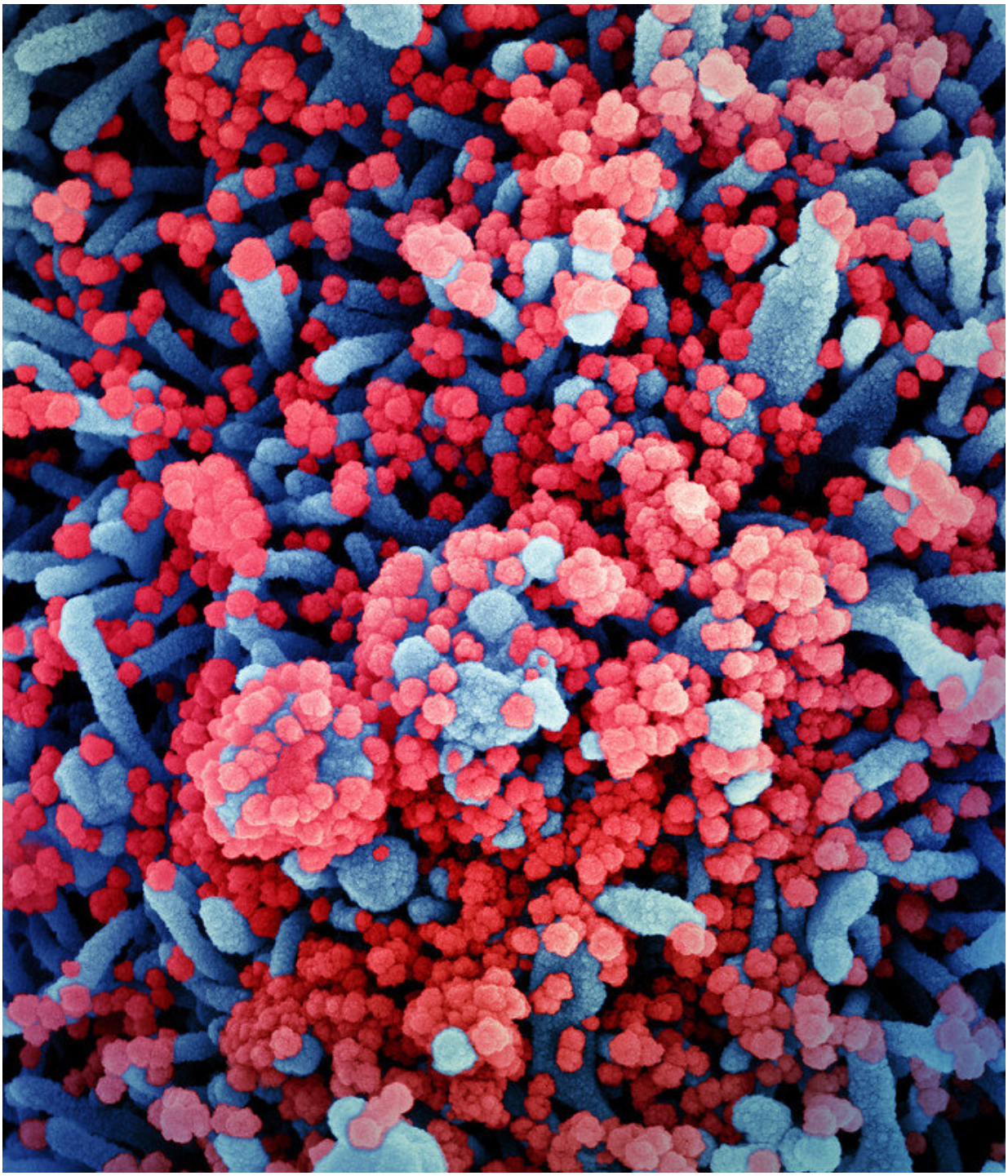

Colorized scanning electron micrograph of a cell (blue) heavily infected with SARS-CoV-2 virus particles (red), isolated from a patient sample. Image captured at the NIAID Integrated Research Facility (IRF) in Fort Detrick, Maryland. Credit: NIAID

Seasonal colds are by all accounts no fun, but new research suggests the colds you've had in the past may provide some protection from COVID-19. The study, authored by infectious disease experts at the University of Rochester Medical Center, also suggests that immunity to COVID-19 is likely to last a long time—maybe even a lifetime.

The study, published in *mBio*, is the first to show that the COVID-19-causing virus, SARS-CoV-2, induces memory B cells, long-lived immune cells that detect pathogens, create antibodies to destroy them and remember them for the future. The next time that pathogen tries to enter the body, those memory B cells can hop into action even faster to clear the infection before it starts.

Because memory B cells can survive for decades, they could protect COVID-19 survivors from subsequent infections for a long time, but further research will have to bear that out.

The study is also the first to report cross-reactivity of memory B cells—meaning B cells that once attacked cold-causing coronaviruses appeared to also recognize SARS-CoV-2. Study authors believe this could mean that anyone who has been infected by a common coronavirus—which is nearly everyone—may have some degree of pre-existing immunity to COVID-19.

"When we looked at blood samples from people who were recovering from COVID-19, it looked like many of them had a pre-existing pool of memory B cells that could recognize SARS-CoV-2 and rapidly produce antibodies that could attack it," said lead study author Mark Sangster, Ph.D., research professor of Microbiology and Immunology at URMC.

Sangster's findings are based on a comparison of blood samples from 26

people who were recovering from mild to moderate COVID-19 and 21 healthy donors whose samples were collected six to 10 years ago—long before they could have been exposed to COVID-19. From those samples, study authors measured levels of memory B cells and antibodies that target specific parts of the Spike protein, which exists in all coronaviruses and is crucial for helping the viruses infect cells.

The Spike protein looks and acts a little different in each coronavirus, but one of its components, the S2 subunit, stays pretty much the same across all of the viruses. Memory B cells can't tell the difference between the Spike S2 subunits of the different coronaviruses, and attack indiscriminately. At least, the study found that was true for betacoronaviruses, a subclass that includes two cold-causing viruses as well as SARS, MERS and SARS-CoV-2.

What this study doesn't show is the level of protection provided by cross-reactive memory B cells and how it impacts patient outcomes.

"That's next," said David Topham, Ph.D., the Marie Curran Wilson and Joseph Chamberlain Wilson Professor of Microbiology and Immunology at URMC, who runs the lab that conducted this work. "Now we need to see if having this pool of pre-existing memory B cells correlates with milder symptoms and shorter disease course—or if it helps boost the effectiveness of COVID-19 vaccines."

More information: Phuong Nguyen-Contant et al, S Protein-Reactive IgG and Memory B Cell Production after Human SARS-CoV-2 Infection Includes Broad Reactivity to the S2 Subunit, *mBio* (2020). DOI: 10.1128/mBio.01991-20

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