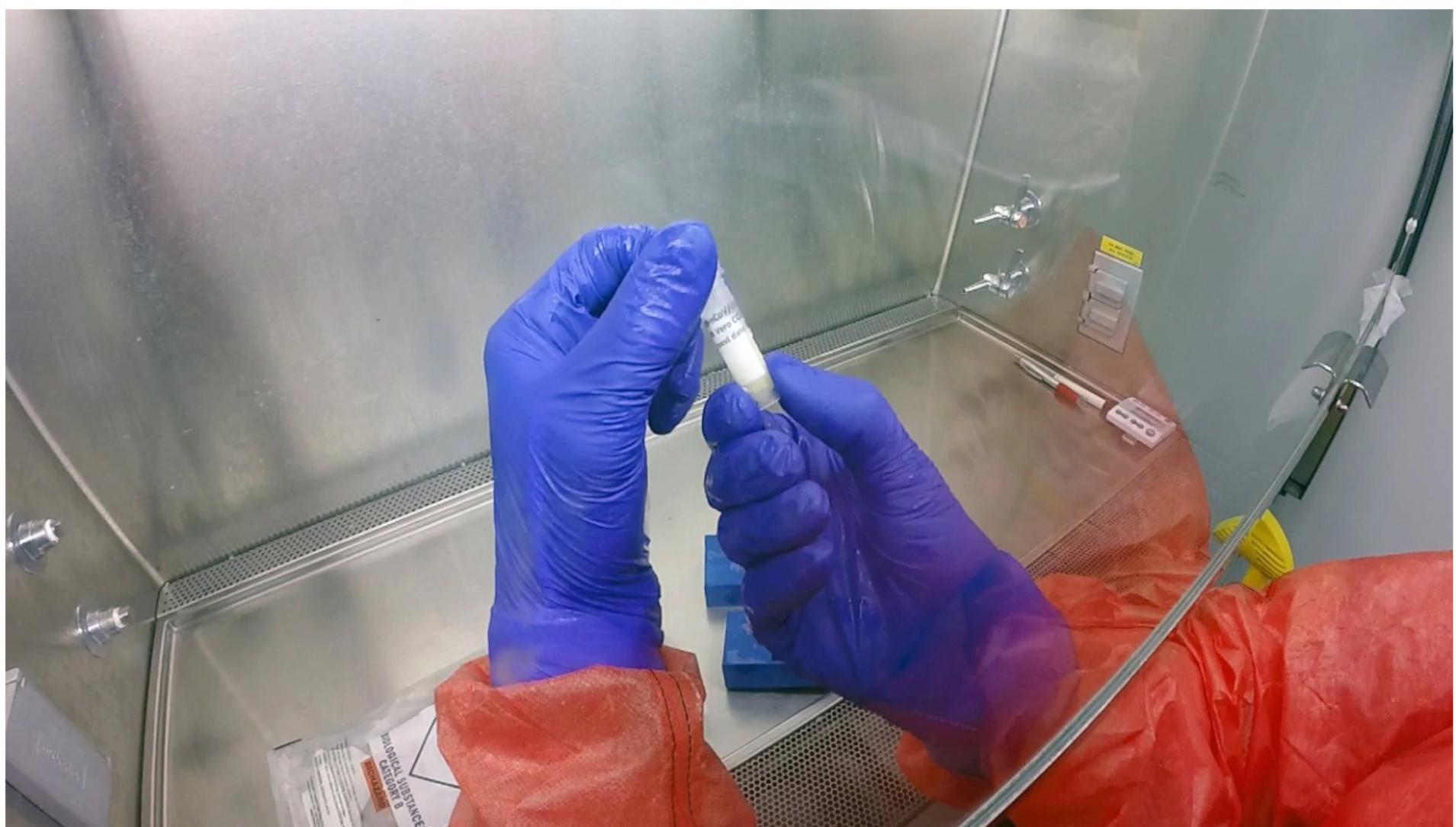


How the coronavirus can kill people

Most cases are mild, but the new virus is also proving lethal in some



A researcher works on a sample of the new coronavirus inside a secure laboratory in microbiology and immunology at the University of Maryland School of Medicine. (Robert Haupt/University of Maryland School of Medicine)

By **Carolyn Y. Johnson**

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The mounting toll of coronavirus deaths, involving [health-care workers](#) on the [front lines of the disease](#) and older people in hospitals, raises a basic question: How does the new virus make people sick, and why does it kill some of them?

Scientists' understanding of the novel coronavirus is in flux, but it's important to keep a few facts in mind. Most people who contract covid-19 — 80 percent, according to a study from Chinese public health officials — will experience only [mild illness and recover](#). Based on early numbers, which come with a huge caveat given the difficulty of estimating a fatality rate early in an outbreak, about 2 percent of people who are infected die.

The difference between a lethal infection and one that feels like a bad cold probably hinges on the interaction between the virus and a person's immune system.

[\[Live updates: China strikes upbeat note on coronavirus as businesses reopen; Hong Kong reports second death\]](#)

No one knows exactly how the coronavirus leads to respiratory failure in some patients. But given researchers' knowledge of other illnesses, including closely related severe acute respiratory syndrome (SARS), they're making certain assumptions.



The coronavirus and other outbreaks are hard to contain. Here's why.
As of early February 2020, tens of thousands of people worldwide were infected with a new strain of coronavirus. Officials are taking "unprecedented" actions. (Amber Ferguson, Jayne Orenstein/The Washington Post)

While the virus attacks and kills cells in all cases, serious illness will depend on how the immune system responds, and that can be influenced by age, gender, genetics and underlying medical conditions. The initial damage caused by the virus can trigger a powerful and counterproductive overreaction.

“What you get is the initial damage and rush of inflammatory cells, but the damage is so extensive that the body’s immune response is completely overwhelmed — which causes even more immune response, more immune cells and more damage,” said Matthew Frieman, a virologist at the University of Maryland School of Medicine.

[\[Inside a lab where scientists are working urgently to fight the coronavirus outbreak\]](#)

The coronavirus is spread when an infected person sneezes or coughs, spraying droplets through the air. The sick person might sneeze directly in another person’s face or expel droplets widely, contaminating surfaces that healthy people touch before unknowingly spreading germs to their mouths or noses. Health-care workers are at especially high risk because they are exposed to very high doses of the virus and also perform procedures — such as putting patients on ventilators to help them breathe — that can spread it.

With infection, the virus probably begins to multiply inside cells lining the airway, which are fringed with hairlike structures. Coronaviruses that cause common colds are excellent at infecting the upper airway, while SARS tended to go deeper in the lungs. As the coronavirus gains strength, Frieman said, dead cells are sloughed off and collect in the airway, making breathing difficult.

“If the virus replicates very quickly, before your body has a chance to try and prevent it with an immune response, or if the immune response comes in too late, then it can’t control the virus and starts going berserk,” said Anthony Fehr, a virologist at the University of Kansas.

This is what scientists refer to as a “cytokine storm,” which causes the immune system to start sending cells ready to do battle into the lung. At that point, it’s not just the virus doing damage to the body; the immune system begins wreaking havoc on the infected person — also known as the “host” in medical parlance.

“The experience with other respiratory viruses would suggest it is a combination of the virus doing damage to the airways, secondary infections and the interplay with the host immune response,” said Erica S. Shenoy, an infectious diseases specialist at Massachusetts General Hospital.

[Coronavirus vaccine research is moving at record speed]

The general risk factors for this mismatch between the immune system and any respiratory illness include advanced age and underlying chronic illnesses, including diabetes and high blood pressure, though public health experts are eager to understand more about who is most vulnerable in the current outbreak.

“Every individual is different,” Fehr said, and there are differences in how young and old or male and female immune systems react. “There are lots of dynamics at play when you talk about each individual and how they might die from this virus or why they might survive.”

Problems can also stack up. Vineet Menachery, a virologist at the University of Texas Medical Branch, suspects that the coronavirus may work much like SARS. When the virus gets deep into the lungs, it can damage alveoli, the air sacs that take in oxygen. As cellular damage accumulates, lung tissue begins to stiffen. The heart must work harder to get limited oxygen to the rest of the organs.

“What makes this new virus so damaging is you’re losing lung function, and that puts a strain on every organ in your body,” Menachery said.

In the patients who recover, the immune system’s response has worked: It has cleared the virus, with inflammation receding. Yet experts don’t know the long-term outcome for these individuals. It’s possible they will gain immunity and be protected from reinfection. Or they might get a less severe case in the future — or not be protected at all. They also might just gain temporary immunity. It’s yet another unanswered question about the coronavirus.