

According to the November study, the 2018 explosion at Beijing Jiaotong University – which led to the deaths of three students and destroyed a laboratory – was caused by the ignition of 66 kilograms of improperly stored magnesium dust. The authors say this was the first university lab accident in China for which the results of a detailed investigation were reported on a government website.

Some researchers are optimistic that the safety situation is improving, as it has in many countries in recent decades.

“Compared with 20 years ago, lab safety in China has definitely made significant progress,” says Samuel Yu, director of the Health, Safety and Environment Office at the Hong Kong University of Science and Technology.

Denis Simon, who was executive vice-chancellor of Duke Kunshan University in Jiangsu province from 2015 to 2020, agrees that “this is a country that has come a tremendously long

way in improving the protocols”. But he says that China needs more specialists with a career focus on lab safety.

The concerns over safety in university teaching labs in China follow a debate about whether the COVID-19 pandemic might have started in a lab in China. But Gigi Gronvall, a biosecurity specialist at the Bloomberg School of Public Health at Johns Hopkins University in Baltimore, Maryland, who has visited labs in China, says that there is no reason to think that the problems flagged in student chemistry labs reveal anything about the biological-research labs that handle dangerous pathogens.

Even non-teaching biological-research labs that do quite advanced experiments don't use dangerous pathogens; to handle these, scientists typically require extensive training and follow stringent safety standards, she says.

Additional reporting by Smriti Mallapaty.

organoids<sup>4</sup>. These experiments also identified a plausible player in the difference: a protein called TMPRSS2, which protrudes from the surfaces of many cells in the lungs and other organs, but is notably absent from the surfaces of most nose and throat cells. Previous variants have exploited this protein to infect cells, but the researchers noticed that Omicron doesn't bind to TMPRSS2 so well. Instead, it tends to enter cells when it is ingested by them<sup>5,6</sup>.

## Upper airway preferred

Difficulty entering lung cells could help to explain why Omicron does better in the upper airways than in the lungs, says Ravindra Gupta, a virologist at the University of Cambridge, UK, who co-authored one of the TMPRSS2 studies<sup>4</sup>. This theory could also explain why, by some estimates, Omicron is nearly as transmissible as measles, which is the benchmark for high transmissibility, says Diamond. If the variant lingers in the upper airways, viral particles might find it easy to hitch a ride on material expelled from the nose and mouth, allowing the virus to find new hosts, says Gupta.

The latest results could mean that “the virus establishes a very local infection in the upper airways and has less chance to go and wreak havoc in the lungs”, Ott says. That would be welcome news – but a host's immune response plays an important part in disease severity, and scientists need more clinical data if they are to understand how Omicron's basic biology influences its disease progression in humans.

Omicron's course of infection could also have implications for children, says Audrey John, a specialist in paediatric infectious disease at the Children's Hospital of Philadelphia in Pennsylvania. Young children have relatively small nasal passages, and babies breathe only through their noses. Such factors can make upper respiratory conditions more serious for children than for adults, John says. But she adds that she has not seen data suggesting an uptick in the numbers of young children hospitalized for conditions that could indicate a severe infection of the upper respiratory tract.

Although there is still much to learn about the new variant, Gupta says that fears raised in late November by the multitude of mutations in Omicron's genome have not been completely borne out. He says the initial alarm offers a cautionary tale: it's difficult to predict how a virus will infect organisms from its genetic sequence alone.

# OMICRON MAKES A FEEBLE ATTACK ON THE LUNGS

## Animal studies suggest that the variant's inability to multiply in lung tissue could make it less dangerous.

By Max Kozlov

**E**arly indications from South Africa and the United Kingdom signal that the fast-spreading Omicron variant of the coronavirus SARS-CoV-2 is less dangerous than its predecessor Delta. Now, a series of laboratory studies offers a tantalizing explanation for the difference: Omicron does not infect cells deep in the lung as readily as it does those in the upper airways.

The observation “might explain what we see in patients”, says Melanie Ott, a virologist at the Gladstone Institute of Virology in San Francisco, California. But she adds that Omicron's hyper-transmissibility means that hospitals are filling quickly – despite potential decreases in the severity of the disease it causes.

Authorities in South Africa announced on 30 December that the country had passed its Omicron peak without a major spike in deaths. And a 31 December UK government report said that people in England who were infected with Omicron were about half as likely to require hospitalization or emergency care as were those infected with Delta.

But the number of people who have gained immune protection against COVID-19 through

vaccination, infection or both has grown over time, making it difficult to determine whether Omicron intrinsically causes milder disease than do earlier variants. For answers, researchers have turned to the laboratory.

Michael Diamond, a virologist at Washington University in St. Louis, Missouri, and his colleagues infected rodents with Omicron and other variants to track disease progression. The differences were staggering: after a few days, the concentration of virus in the lungs of animals infected with Omicron was at least ten times lower than in rodents infected with other variants<sup>1</sup>. Other teams have also noted that, compared with previous variants, Omicron is found at reduced levels in lung tissue<sup>2,3</sup>.

Diamond was especially shocked to see that the Omicron-infected animals nearly maintained their body weight, whereas the others quickly lost weight – a sign that their infections were causing severe disease. The lungs are where the coronavirus does much of its damage, and lung infection can set off an inflammatory immune response that ravages both infected and uninfected cells. Fewer infected lung cells could mean milder illness.

Another group found that Omicron is much less successful than previous variants at infecting lung cells and miniature lung models called

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2. McMahan, K. et al. Preprint at bioRxiv <https://doi.org/10.1101/2022.01.02.474743> (2022).
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