News in brief

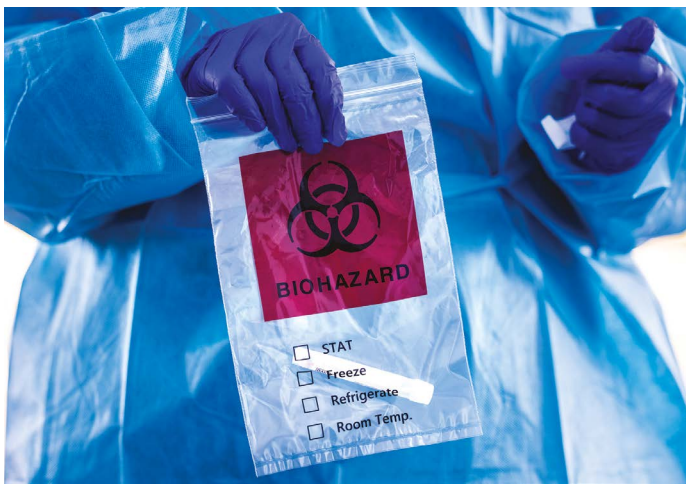
EVIDENCE GROWS OF NEW CORONAVIRUS VARIANT'S SWIFT SPREAD

Two independent analyses have found that a new SARS-CoV-2 variant overtaking the United Kingdom is indeed more transmissible than other forms of the virus.

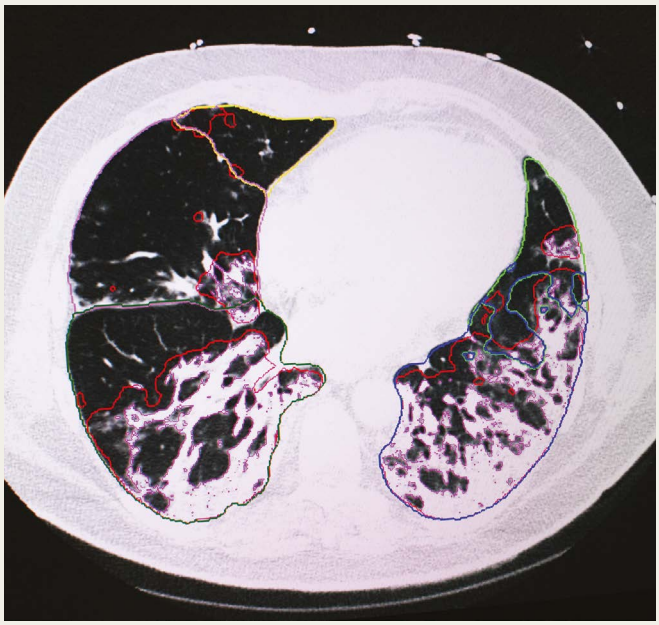
Eric Volz and Neil Ferguson at Imperial College London and their colleagues examined genomes from nearly 2,000 samples of the variant, which has been labelled Variant of Concern 202012/01 and is also known as B.1.1.7 (see page 177). The genomes were collected in the United Kingdom between October and early December 2020. The team also analysed the results of roughly 275,000 UK COVID-19 tests administered in late 2020 (E. Volz *et al*. Preprint at medRxiv https://doi.org/ghrqv8; 2021).

Estimating the frequency of the variant's occurrence over time, the authors concluded that it is roughly 50% more transmissible than other variants. The authors also found that a UK lockdown in November curbed COVID-19 cases caused by most other variants — but that those linked to the new variant rose. The findings have not yet been peer reviewed.

A separate team used genomic and other data to analyse the variant's spread in the last few months of 2020. Nicholas Davies and his colleagues at the London School of Hygiene & Tropical Medicine estimated that the new variant is 56% more transmissible than others (N. Davies *et al*. Preprint at medRxiv https://doi.org/fp3v; 2020). The authors found no evidence that the variant of concern causes more severe COVID-19 than other variants. The findings are now under peer review.



L TO R: MICHAEL CIAGLO/GETTY; NATHAN LAINE/BLOOMBERG/GETTY



Traitorous antibodies are linked to COVID death

Antibodies normally attack pathogens, but sometimes rogue antibodies instead besiege bodily components such as immune cells. Now, a new study adds to the growing body of research tying these 'autoantibodies' to poor outcomes in people with COVID-19.

Ana Rodriguez and David Lee at the NYU Grossman School of Medicine in New York City and their colleagues studied autoantibody levels in blood serum collected from 86 people who required hospitalization for COVID-19. The researchers were particularly interested in autoantibodies against the protein annexin A2, which helps to stabilize cell-membrane structure. It also plays a part in ensuring the integrity of tiny blood vessels in the lungs. Blocking annexin A2 leads to lung injury (pictured), a hallmark of COVID-19.

The scientists found that the level of anti-annexin A2 antibodies was, on average, higher in the individuals who eventually died of COVID-19 than in those who survived — a difference that was statistically significant (M. Zuniga *et al*. Preprint at medRxiv https://doi.org/fqdd; 2021).

More research is necessary to establish a clear causal link between the virus SARS-CoV-2 and autoantibodies against annexin A2, which are relatively rare. The findings have not yet been peer reviewed.