Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

in the corresponding VIM-2 Glu163Lys-producing recombinant strain (table). IC_{50} measurements indicated that this observed resistance resulted from a lack of inhibition of the VIM-2 Glu163Lys-producing mutant by taniborbactam (appendix p 5).

See **Online** for appendix

This work firstly reports and characterises the resistance of an NDM-like enzyme to taniborbactam, a β-lactamase inhibitor that, in combination with cefepime, is currently undergoing phase 3 clinical investigation. We showed that a single amino acid substitution in the NDM enzyme, but also (and in a similar manner) in the VIM enzyme, leads to increased resistance to taniborbactam. Importantly, acquisition of the NDM-9 enzyme, which is of particular concern in Italy,[7] is already disseminated in many Gram-negative bacterial species in many parts of the world, and is a key resistance mechanism to this promising and almost unique MBL inhibitor.

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For **previously dominant omicron variants** see https://nextstrain.org/ncov/gisaid/global/all-time

We declare no competing interests.

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Antiviral and bivalent vaccine efficacy against an omicron XBB.1.5 isolate

As of January 2023, SARS-CoV-2 subvariants BQ.1 (a BA.5 subvariant) and XBB (a BA.2 subvariant) have replaced previously dominant omicron variants globally, including BA.5. XBB.1.5, which is a descendant of XBB, is rapidly increasing in prevalence and is now dominant in the USA. We and other groups have shown that XBB has higher immune-evasion capabilities than earlier variants, including BA.5 and BA.2.[1–5] Although XBB.1.5 and XBB share most of the amino acid substitutions in the receptor-binding domain of the spike protein, which is the major target for vaccines and therapeutic monoclonal antibodies against SARS-CoV-2, XBB.1.5 has an additional substitution not found in XBB (ie, S486P). Therefore, we should evaluate the efficacy of therapeutic drugs and the effect of COVID-19 vaccines against XBB.1.5.

In this study, we assessed the efficacy of therapeutic monoclonal antibodies against omicron XBB.1.5 (hCoV-19/USA/MD-HP40900-PIDYSWHNUB/2022), which was isolated from a patient with COVID-19. Compared with a BA.2 isolate (hCoV-19/Japan/UT-NCD1288–2N/2022), this XBB.1.5 isolate had nine additional changes (ie, G339H, R346T, L368I, V445P, G446S, N460K, F486P, F490S, and the wild-type amino acid at position 493) and was comprised of a mixed population that encoded either Cys (the wild-type amino acid; 90%) or Thr (10%) at position 361 in its receptor-binding domain (appendix p 5). To examine the reactivity of monoclonal antibodies against XBB.1.5, we established the 50% focus reduction neutralisation titre ($FRNT_{50}$) of the monoclonal antibodies using vero E6-TMPRSS2-T2A-ACE2 cells. Similar to the XBB isolate, all tested monoclonal antibodies (ie, imdevimab, casirivimab, tixagevimab, cilgavimab, sotrovimab, and bebtelovimab) did not neutralise the XBB.1.5 isolate, even at the highest $FRNT_{50}$ value tested (>50 000 ng per millilitre; appendix pp 6–7). These results suggest that imdevimab–casirivimab, tixagevimab–cilgavimab, sotrovimab, and bebtelovimab might not be effective against XBB.1.5 in the clinical setting, as has been reported for XBB.

Then, we evaluated the antiviral drugs remdesivir (an RNA-dependent RNA polymerase [RDRP] inhibitor), which is approved for the treatment of COVID-19 by the US Food and Drug Administration (FDA); molnupiravir (also an RDRP inhibitor) and nirmatrelvir (a main protease inhibitor), both of which are authorised for emergency use by the US FDA; and ensitrelvir (a main protease inhibitor), which has emergency regulatory approval in Japan and is currently in phase 3 clinical trials in the USA. We established the in vitro 50% inhibitory concentration values of these antiviral drugs against XBB.1.5 to test their effectiveness. Compared with the reference strain Wuhan/Hu-1/2019, the XBB.1.5 isolate has one substitution (ie, P3395H) in its RDRP and two substitutions (ie, P314L and G662S) in its main protease (appendix p 5). The susceptibilities of XBB.1.5 to these antivirals were similar to those of the ancestral strain

(appendix pp 6–7). These results suggest that remdesivir, molnupiravir, nirmatrelvir, and ensitrelvir are efficacious against both XBB.1.5 and XBB in vitro.

Lastly, we tested the neutralising ability of plasma from people who were convalescent and people who had had the COVID-19 vaccine against XBB.1.5. For plasma from people who had received a fourth dose of the mRNA vaccine, 11 (65%) of 17 samples had $FRNT_{50}$ values that were below the limit of detection against XBB and 9 (53%) of 17 samples had $FRNT_{50}$ values that were below the limit of detection against XBB.1.5. For plasma from people who had received the bivalent mRNA vaccine as a fifth dose, most samples (17 [94%] of 18) neutralised XBB.1.5. Similar results were obtained with plasma samples from people who had had the COVID-19 vaccine with BA.2 breakthrough infection. 8 (80%) of 10 samples with BA.2 breakthrough infection neutralised XBB.1.5. Although the neutralising activity against XBB.1.5 was considerably lower than that against the ancestral strain or BA.2 in all tested groups, the reductions in neutralising titres for XBB.1.5 were similar to those for XBB (appendix pp 6–7). Our data suggest that XBB.1.5 effectively evades current humoral immunity induced by mRNA vaccines or natural infection and that a bivalent vaccine (ancestral and BA.4/5) can improve humoral responses to XBB.1.5.

A study using surface plasmon resonance assays showed that the receptor-binding domain of XBB.1.5 has a higher affinity for human ACE2 (hACE2) compared with XBB and XBB.1.[6] Because our data showed the similar immune evasion capability of XBB and XBB.1.5, the hACE2 binding affinity of XBB.1.5, rather than its immune evasion, might contribute to its high transmissibility and rapid expansion in the USA. Furthermore, our data suggest that antiviral drugs (eg, remdesivir, molnupiravir, nirmatrelvir, and ensitrelvir) are still effective against the omicron subvariant XBB.1.5 and that boosting with a bivalent (ancestral and BA.4/5) vaccine would enhance humoral immunity to XBB.1.5.

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Infection rate in Guangzhou after easing the zero-COVID policy: seroprevalence results to ORF8 antigen

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On Dec 7, 2022, the Chinese government announced ten measures indicating the end of the zero-COVID policy, which was in effect for more than 2 years.[1] Most of the stringent preventive measures, such as mandatory PCR testing, are no longer required. However, easing of restrictions has contributed to the emergence of new outbreaks predominantly by the SARS-CoV-2 omicron lineages BA5.2 and BF.7 in many cities, such as Beijing.[2] With suspension of mass testing in mainland China after adjustment of the zero-COVID policy, empirical data on the epidemic growth and transmissibility of omicron variants are scarce. Until now, the number of infected cases and transmission rates were usually estimated using modelling with partial data.[3] Previously, we reported that presence of the ORF8 antibody in blood is one of the most accurate serological markers of SARS-CoV-2 natural infection.[4,5] Because the *ORF8* gene is only expressed during SARS-CoV-2 replication, this serology test can