

# Highly contagious coronavirus variant's mutation weakens antibody effectiveness

September 17 2021, by Chrystian Tejedor

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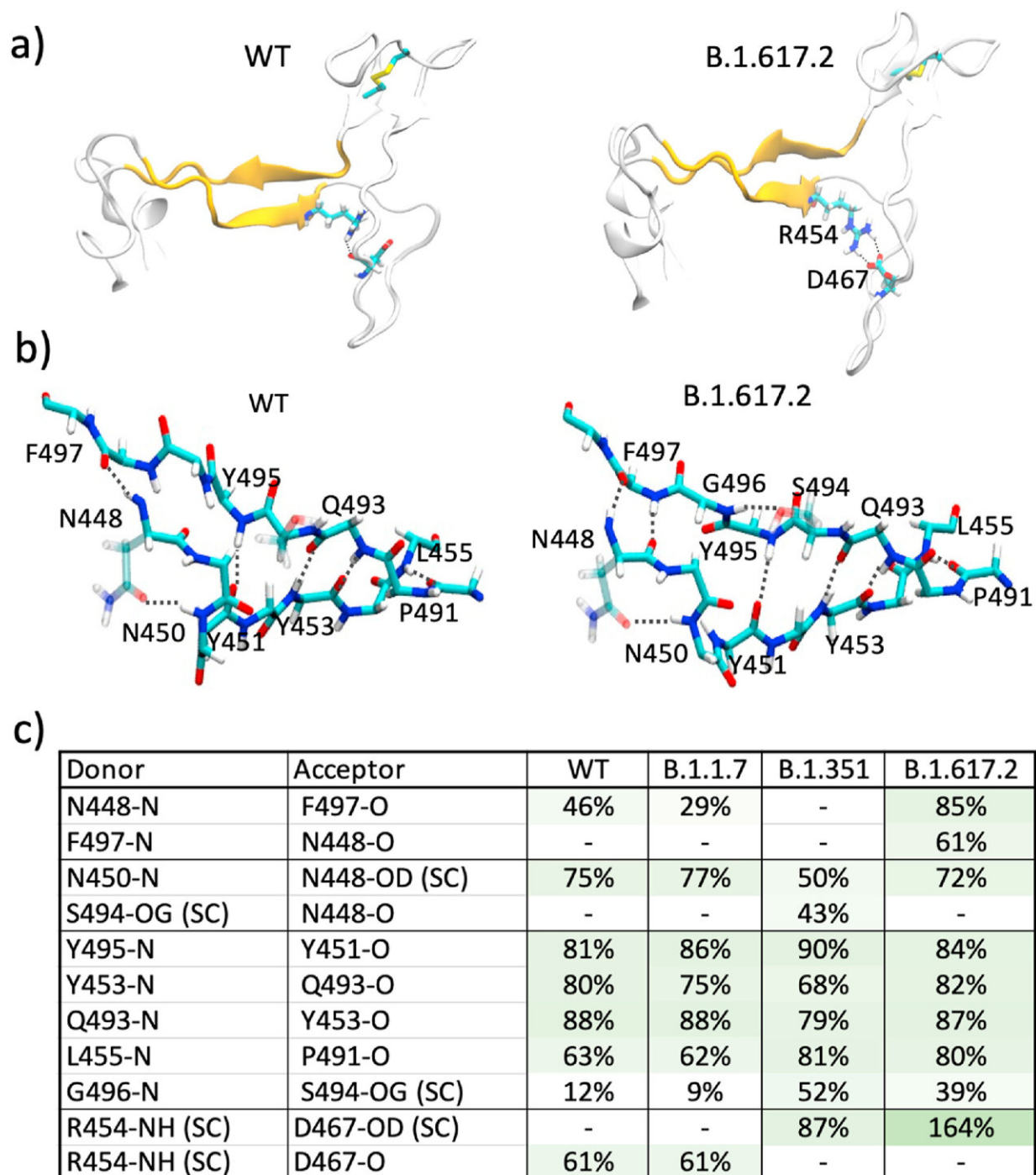


Fig. 1. a) RBM showing the antiparallel  $\beta$ -strands. Residues R454 and D467 participating in ionic interactions in the Delta variant are shown as sticks. b) Hydrogen-bond network in the  $\beta$ -sheet region of the RBM for the WT and the Delta variant. c) % hydrogen bond occupancy obtained from the last 300 ns for the interactions in WT, B.1.1.7, B.1.351, and B.1.617.2. The sidechain

interactions are denoted as SC. Credit: DOI: 10.1016/j.bbrc.2021.08.036

FIU researchers looking to unravel the secrets of how the coronavirus' most contagious variant became so widespread have found an intriguing clue.

Computer modeling allowed researchers to see how mutations in a certain area of the [delta variant](#) may prevent [antibodies](#) from fully latching on to the [virus](#). These antibodies—resulting from vaccination or from fighting a COVID-19 infection—help our [immune system](#) by interrupting or slowing the coronavirus at various steps in its process to invade a human cell and multiply.

The subtle structural changes could help explain why the delta [variant](#) is so contagious.

The discovery was made by a team of researchers led by Prem Chapagain, a physicist who is associate director of FIU's Biomolecular Sciences Institute, and computer sciences professor Giri Narasimhan of the Knight Foundation School of Computing and Information Sciences in FIU's College of Engineering and Computing. Their study was published in the journal *Biochemical and Biophysical Research Communications*.

"The delta variant seems to have found an immune escape," Narasimhan said. "By mutating a little bit, it has weakened the impact of the antibody."

Because the structural changes due to mutation occurred mostly in one region of the binding interface, there are still plenty of other parts of the virus where antibodies can latch on and prevent the virus from working

as intended. It's why vaccines are still effective against the virus.

"Viruses don't make the changes knowingly," Chapagain said.

"Occasionally, they find something that will work better for them, and those variants will be more prevalent."

Even the [delta](#) variant itself continues to mutate, which is why computer modeling efforts like those being undertaken at FIU could be the key to quickly understanding how future variants behave or stopping future pandemics in their tracks. The computational team led by Narasimhan is leveraging funding from the National Science Foundation to train computers that could one day predict how proteins from antibodies and viruses will interact.

For now, both Chapagain and Narasimhan encourage people to get vaccinated and to wear masks to curb the spread of the coronavirus and any future variants that might spread next.

**More information:** Prabin Baral et al, Mutation-induced changes in the receptor-binding interface of the SARS-CoV-2 Delta variant B.1.617.2 and implications for immune evasion, *Biochemical and Biophysical Research Communications* (2021). [DOI: 10.1016/j.bbrc.2021.08.036](#)

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