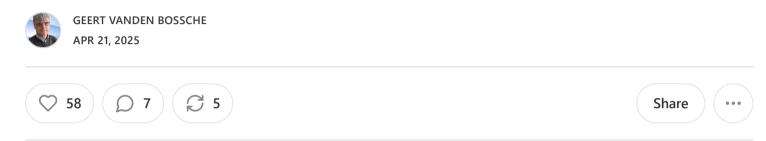
T.A.C.T.: The threat of the currently circulating variants doesn't lie in its virulence....OMG!!



You may be interested in reading the following substack from T.A.C.T. ('Together Against Covid Transmission: COVID Variant Update: LP.8.1 Dominates for Now—But the Next Variant Wave Is Building. A Deep Dive into NB.1.8.1). Just that you know not everybody agrees with me!

While they recognize that the rapid and comprehensive evolution of SC-2 (sub)lineages result from immune pressure, they remain convinced that this will not result in the emergence of a virulent CoV lineage. See below:

Spike Protein Mutations and Immune Escape:

NB.1.8 and its descendant NB.1.8.1 are closely related Omicron sublineages that carry a set of spike protein mutations giving them a significant immune escape advantage. While both share most of these changes, NB.1.8.1 differs by one additional mutation—A435S—which may enhance its ability to evade neutralizing antibodies. All of these

mutations are present in NB.1.8.1, while NB.1.8 carries all except A435S, which was acquired later in its evolution. Taken together, these mutations form a potent escape profile. Remarkably, NB.1.8.1 developed a spike mutation pattern nearly identical to that of XEC.25.1, another highly immune-evasive Omicron subvariant, but did so through a separate evolutionary path. This pattern—called convergent evolution—shows how different lineages under similar immune pressure independently acquire the same advantageous mutations.

Evolutionary Advancement Without Increased Severity:

NB.1.8.1 represents a significant step forward in SARS-CoV-2's evolution, marked by a carefully accumulated set of mutations that enhance its ability to bypass the immune system. That said, there is no current indication that this variant causes more severe illness than previous Omicron-lineage variants. Its threat lies not in virulence but in reinfection, erosion of immune protection, and the slow, cumulative burden it places on public health and individual health over time.

Anyway, their solution is very well known:

These variants are not necessarily more severe, but their enhanced ability to bypass immune defenses is driving reinfections—and possibly the next wave. Hopefully, scientists are evaluating how well existing vaccines and treatments hold up against NB.1.8.1. Though current boosters and antivirals may still protect against serious illness, rising transmission....

Here comes my take on their interpretation:

I find it difficult to understand how some scientists fail to grasp that ongoing immune pressure on the virus still holds tremendous potential to *further accelerate the already remarkable accumulation and diversification of selected viral mutations!* These mutations are already changing SC-2's infectious behavior quite dramatically by enhancing the virus's intrinsic infectivity—through stronger binding to the ACE2 receptor—diminishing its susceptibility to neutralizing Abs and increasing its replication capacity. Collectively, these changes enable continued viral transmission. What they seem to ignore is that sustained immune pressure has not yet driven extensive exploitation of potential mutations in the virus's surface-expressed glycan structures—an evolutionary space that could allow it to evade both humoral and cellular immune responses even more spectacularly!

It is, therefore, hard to understand how some researchers can downplay the threat posed by the ongoing viral evolution ('the threat of the currently circulating variants doesn't lie in its virulence') by focusing only one the type of phenotypic changes observed to date, without modeling the potentially dramatic impact of even modest alterations in the viral glycosylation profile on pathogenicity.



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