

WHO TAG-VE Risk Evaluation for SARS-CoV-2 Variant Under Monitoring: BA.3.2

Executive Summary

BA.3.2 has been designated a SARS-CoV-2 Variant Under Monitoring (VUM). Although it demonstrates antigenic drift and reduced neutralization in vitro, currently approved COVID-19 vaccines are expected to continue providing protection against severe disease. There have been reports from Western Australia of elevated BA.3.2 wastewater signals. Recently, BA.3.2 was detected, though still very low-level, in wastewater from some U.S. states. However, BA.3.2 has not shown a sustained growth advantage over any other co-circulating variant, and no data indicate increased severity, hospitalisations, or deaths associated with this variant.

Overall, available evidence suggests that BA.3.2 poses low additional public health risk compared with other circulating Omicron descendent lineages.

Initial Risk Evaluation of BA.3.2, 5 December 2025

BA.3.2 is a SARS-CoV-2 variant that is a descendent lineage of the Omicron variant BA.3, Figure 1A, differing from BA.3 in the Spike protein by 53 mutations [1], Figure 1B, with the earliest sample collected on 22 November 2024. Phylodynamic analysis estimates the variant to have emerged between December 2023 and July 2024 [2]. BA.3.2 is one of six VUMs tracked by the WHO [3,4].

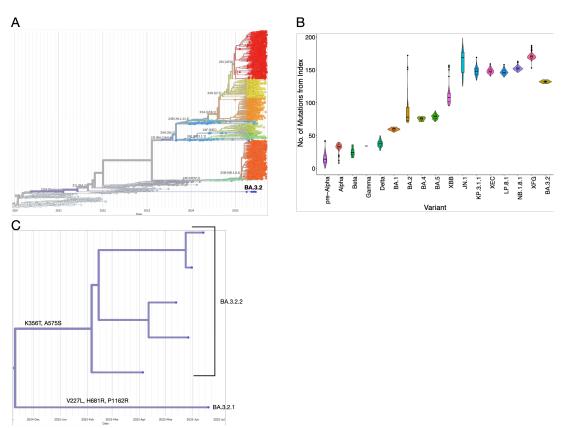


Figure 1: Evolution of SARS-CoV-2 variants through time. 1A. is a Nextstrain global phylogenetic tree with subsampling of SARS-CoV-2 variants focused over the past 6 months with BA.3.2 tips labelled in bold. 1B. is based on 1A and shows the number of mutations from the index. 1C. is a subtree of 1A showing the divergence of BA.3.2 into two sub-lineages BA.3.2.1 and BA.3.2.2, with the Spike protein mutations leading to the two lineages shown on the branches.

BA.3.2 has diversified into two sublineages, BA.3.2.1 and BA.3.2.2, with two additional Spike protein mutations each (H681R/P1162R and K356T/A575S, respectively), Figure 1C [1]. Residue 681 sits just before the polybasic sequence of the furin cleavage site (FCS) and the R, compared to H, may enhance the polybasic nature of the sequence due to the stronger positive charge of arginine (BA.3.2 FCS: HRRAR; BA.3.2.1 FCS: RRRAR). P681R and P681H, signature substitutions of the Delta and Alpha/Omicron lineages, respectively, are both well-characterized for enhancing spike activation and promoting increased cell-cell fusion, syncytium formation, and viral entry relative to the ancestral residue. Experimental comparisons demonstrate that P681R confers stronger effects on spike cleavage, fusogenicity, and infectivity than P681H [5]. As the number of sequences available is low, Figure 2, it is not possible to assess if there are differences in the degree of transmissibility between BA.3.2.1 and BA.3.2.2.

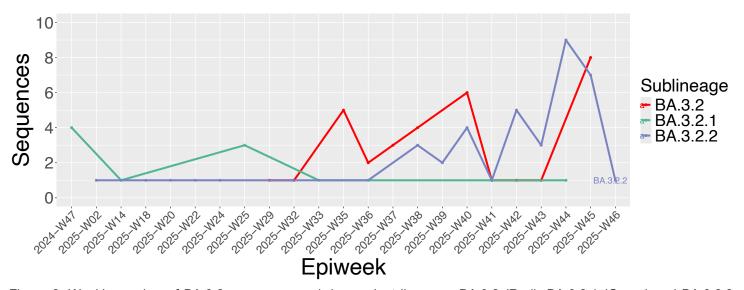


Figure 2: Weekly number of BA.3.2 sequences and descendent lineages: BA.3.2 (Red), BA.3.2.1 (Green) and BA.3.2.2 (Lavendar)

In sera from individuals with prior confirmed SARS-CoV-2 infection or COVID-19 vaccination, neutralizing antibody titres against BA.3.2 were markedly lower than those against circulating JN.1-descendent variants [6], with sera from pre-Omicron cohorts reporting near complete loss of neutralizing activity [2]. Using sera from individuals recently vaccinated with monovalent KP.2 or LP.8.1 vaccines, post-vaccination neutralizing antibody titres against BA.3.2 were lower as compared to those against the homologous vaccine antigen and demonstrated only modest fold rises in neutralizing antibody titres, indicating that BA.3.2 is antigenically distant to circulating JN.1-descendent variants [7–9].

As of 9 November 2025, there were 86 BA.3.2 sequences submitted to GISAID [10] from 7 countries, representing 1.7% of the globally available sequences in epidemiological week (EW) 45 of 2025 (3 to 9 November 2025). This is a rise in proportion from 0.2% four weeks prior in EW 42 of 2025 (13 to 19 October 2025), Table 1. Between EW42 and EW 45 of 2025, BA.3.2 showed varied trends across the three WHO regions that are consistently sharing SARS-CoV-2 sequences, i.e. an increase from 1.4% to 5.7% for the Western Pacific region (WPR), from no detections to 1.4% for the European Region (EUR), and no detections in the four weeks for the Region of the Americas (AMR). Albeit with fewer sequence submissions, BA.3.2 proportion decreased from 10.3% in EW 42 to no detections in the three weeks to EW 45 for the African Region (AFR), and no detections over the four weeks for the East Mediterranean Region (EMR) and the South-East Asia Region (SEAR).

In Western Australia, which has reported the largest wastewater detection of BA.3.2 to date, the variant accounted for 33.3% of signals in EW 45, following a previous peak of 66.7% in EW 42 [11]. Additionally, although national COVID-19 wastewater viral activity remains very low, several states have reported increasing detections of BA.3.2 [12]. Taken together, these signals indicate that the variant warrants closer monitoring, supporting its designation as a VUM at this time.

Table 1: Global proportions of SARS-CoV-2 Variants, epidemiological week 42 to 45 of 2025

Lineage*	Countries§	Sequences§	2025-42	2025-43	2025-44	2025-45	
VOIs							
JN.1	154	486987	6.5	6.1	5.0	5.7	
VUMs							
KP.3.1.1	84	104819	0.7	1.3	1.5	3.1	
XEC	86	56989	0.0	0.1	-	0.1	
LP.8.1	74	27871	0.5	0.7	0.7	1.1	
NB.1.8.1	64	23277	12.3	11.7	15.1	15.1	
XFG	88	51390	73.9	74.2	70.6	67.8	
BA.3.2	7	86	0.2	0.2	0.7	1.7	
Recombinant	152	526242	5.6	5.5	6.2	4.8	
Unassigned	74	4639	0.0	0.1	0.1	-	
Others	120	37766	0.3	0.2	0.1	0.7	

Figures by WHO, data from GISAID, extracted on 30 November 2025.

For the WHO and its Technical Advisory Group on Virus Evolution (TAG-VE) to continue carefully monitoring and assessing the public health risk of BA.3.2, the following additional information would be helpful:

- Neutralization assays using human sera, representative of the affected community(ies), and sera from naive animal models infected with BA.3.2 live virus isolates.
- Comparative evaluation to detect changes in rolling or ad hoc indicators of severity.

WHO and its Technical Advisory Group on COVID-19 Vaccine Composition (TAG-CO-VAC) continue to regularly assess the impact of variants on the performance of COVID-19 vaccines to inform decisions on updates to vaccine composition. In the latest recommendation published on 15 May 2025, the WHO TAG-CO-VAC advised that monovalent JN.1 or KP.2 remain appropriate COVIID-19 vaccine antigens; monovalent LP.8.1 is a suitable alternative vaccine antigen [13].

The risk evaluation below follows the published WHO framework for risk evaluation of SARS-CoV-2 variants [14] and is based on currently available evidence. This risk evaluation will be revised regularly as more evidence and data from additional countries become available. With declining prevalence of VOIs, and VUMs increasingly unable to meet the VOI definition, WHO, on 29 November 2024, began conducting risk evaluations for VUM designations in addition to VOI designations.

Considering the evolution of the global epidemiological situation in relation to COVID-19 and to support member states in addressing the continuous risk posed by COVID-19 during the transition from the response to a public health emergency of international concern to its management within broader disease prevention and control programmes, the IHR Standing Recommendations for COVID-19 issued by the WHO Director General's originally set to expire on 30 April 2025, have been extended for an additional year with the same content, until 30 April 2026 [15].

[§]Number of countries and sequences are since the emergence of the variants.

^{*}The variants listed include descendant lineages, except those individually specified elsewhere in the table.

The VOI and the VUMs that have shown increasing trends are highlighted in yellow, those that have remained stable are highlighted in blue, while those with decreasing trends are highlighted in green.



Overall risk evaluation: Low	BA.3.2 exhibits marked antigenic drift and substantial antibody escape compared to earlier Omicron and current vaccine antigens (e.g. KP.2 and LP.8.1). However, available phenotypic data indicate reduced infectivity, lower fusogenicity and modest replication capacity relative to co-circulating JN.1-descendent variants, and BA.3.2 has not demonstrated a consistent growth advantage or widespread replacement of other variants in circulation. At present, there are no clinical or epidemiological data to suggest that BA.3.2 infection is associated with increased disease severity, diagnostic failure or reduced susceptibility to available antivirals compared with other Omicron descendent lineages. Based on current evidence, BA.3.2 does not appear to pose additional public health risks beyond those associated with other currently circulating Omicron descendent lineages, although its pronounced immune-escape profile warrants continued					
Indicator	virological and epidemiological monitoring. Evidence	Level of risk	Level of confidence			
Growth advantage	There are currently 86 BA.3.2 sequences 7 countries, representing 1.7% of the globally available sequences in epidemiological week (EW) 45 of 2025 (3 to 9 November 2025). This is a rise in proportion from 0.2% four weeks prior in EW 42 of 2025 (13 to 19 October 2025). However, for these four weeks, BA.3.2 showed varied trends across the three WHO regions that are consistently sharing SARS-CoV-2 sequences, i.e. increases in WPR and EUR, and no detections in AMR. Albeit with fewer sequence submissions, BA.3.2 decreased in the AFR, and there were no detections over the four weeks for EMR and SEAR. The currently globally dominant variant XFG, declined between EW 42 and EW 45, from 73.9% to 67.8%. Across the WHO regions, XFG declined in AMR and EUR, but increased in WPR. In general, BA.3.2 shows limited growth advantage relative to co-circulating JN.1-descendant variants [6], with most of the growth reported in Western Australia [11]. BA.3.2 is characterised by reduced intrinsic infectivity, lower fusogenicity, and weaker ACE2 binding compared with co-circulating JN.1-descendant lineages such as LP.8.1, NB.1.8.1 and XFG [6,16].	Low	Moderate			

Immune escape	BA.3.2 exhibits marked immune evasion across multiple studies [1,6–9,16].	Low	Low
	KP.2 vaccination in adults with varied exposure histories showed an increase in post vaccination neutralizing antibody titres against homologous KP.2 variant and other JN.1 derived variants such as LP.8.1, LF.7.1, NB.1.8.1, XFG, and BA.3.2 [7].		
	Similarly, LP.8.1 mRNA and recombinant protein-based vaccines were shown to induce neutralizing antibodies against BA.3.2, but with relatively lower titres compared with JN.1 and JN.1-derived variants (LP.8.1, XEC, NB.1.8.1) [8,9].		
	** see footnote for more explanations		
Severity and clinical/diagnostic considerations	There are no published clinical or epidemiological studies indicating that BA.3.2 is associated with increased disease severity compared with other circulating descendants. At present, there are no signals of increased hospitalisations, ICU admissions, or deaths attributable to BA.3.2 in settings where it has been detected.	Low	Low
	There is no evidence suggesting reduced effectiveness of antivirals (e.g. Remdesivir and Nirmatrelvir).		
	There are no studies indicating that BA.3.2 poses a risk for SARS-CoV-2 diagnostic performance (PCR target failure and/or antigen-test escape).		
	However, BA.3.2 was shown to have increased sensitivity to the monoclonal antibody tixagevimabbe, but resistant to cilgavimab, bebtelovimab, and sotrovimab [16].		
	*** see footnote for more explanations		



Annex:

* Growth advantage

Level of risk: Low, as BA.3.2 has not shown sustained competitive expansion against faster-growing variants such as NB.1.8.1 or XFG. While high prevalence is reported in areas such as Western Australia, global proportions remain quite low.

Confidence: Moderate, as multiple independent phenotypic studies consistently indicate reduced infectivity. However, real-world growth data remain sparse.

** Antibody escape

Level of risk: Moderate, as BA.3.2 shows great immune escape in comparison to co-circulating variants and against recent vaccines.

Confidence: Moderate, as there is evidence from several independent laboratories, even though there are differences in the magnitudes of escape.

*** Severity and clinical considerations

Level of risk: Low, as currently there are no reports of elevated disease severity associated with this variant. There are no reports to suggest resistance to Remdesivir and Nirmaltevir.

Confidence: Low. Currently there are no studies assessing the impact of this variant on clinical outcomes. Although, there is regular co-ordination and data sharing between all WHO Regional Offices, countries reporting of data on severe outcomes such as new hospitalizations, ICU admissions and deaths with the WHO has been decreased substantially. Therefore, caution should be taken when interpreting trends in routine surveillance of severe cases for increased severity. No studies have been conducted yet on the potential impact of the variant on the activity of antivirals like Remdesivir and Nirmaltevir.



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