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WHO TAG-VE Risk Evaluation for SARS-CoV-2 Variant Under Monitoring: PQ.16.1.1

Executive Summary

PQ.16.1.1, an NB.1.8.1-descendent SARS-CoV-2 lineage, has been designated a variant under monitoring (VUM) with increasing proportions globally, driven largely by detections in the Western Pacific Region, particularly Singapore. Considering the available evidence, the additional public health risk posed by PQ.16.1.1 is evaluated as low at the global level. Its mutation profile may confer additional immune escape, although direct phenotypic evidence is currently limited. Available surveillance does not indicate increased clinical severity compared with other circulating variants, and existing vaccines are expected to continue providing protection against severe disease.

Initial Risk Evaluation of PQ.16.1.1, 27 July 2026

PQ.16.1.1 is a descendant of the Omicron JN.1-derived lineage NB.1.8.1, with the earliest available sequence collected on 25 March 2026. Compared with NB.1.8.1, PQ.16.1.1 has the additional Spike substitutions D253G, N417T, D420N and I478T, together with Nucleocapsid T135I and ORF8 G8E [1]. PQ.16.1.1 is one of seven VUMs tracked by the WHO and was designated as a VUM on 27 July 2026.

The parent lineage NB.1.8.1 has demonstrated robust soluble human ACE2 engagement and pseudovirus infectivity, with only marginal additional immune evasion compared with LP.8.1 [2,3]. Preliminary PQ.16.1.1-specific data indicate that its receptor-binding domain binds human ACE2 with significantly lower affinity than that of NB.1.8.1 (KD: 16.5 nM versus 8.22 nM). Consistent with this, soluble human ACE2 showed reduced neutralization of PQ.16.1.1 pseudoviruses compared to NB.1.8.1 (IC50: 0.22 µg/mL versus 0.17 µg/mL). Neutralizing antibody titres against PQ.16.1.1 and NB.1.8.1 were broadly similar in plasma from individuals with prior Omicron exposure. In plasma from Wuhan-Hu-1-primed individuals, titres against PQ.16.1.1 were modestly lower than those against NB.1.8.1. PQ.16.1.1 also showed substantially reduced susceptibility to class 1 RBD-targeting monoclonal antibodies. These preliminary findings suggest that the growth of PQ.16.1.1 is unlikely to be explained by enhanced ACE2 receptor binding alone and may instead be partly related to escape from specific antibody classes [4].

As of 8 July 2026, 457 PQ.16.1.1 sequences with collection dates through epidemiological week (EW) 25 had been submitted to GISAID from eight countries [1]. Of these, 438 (95.8%) were from the Western Pacific Region (WPR), including 388 (84.9% of the global total) from Singapore. The remaining sequences were reported from Hong Kong SAR (34), Australia (10), the United States of America (10), Canada (8), the Republic of Korea (5), France (1) and Taiwan, China (1). No PQ.16.1.1 have been reported from the African (AFR) and Eastern Mediterranean (EMR) Regions.

Globally, the proportion of PQ.16.1.1 among available sequences increased from 2.7% in EW 18 to 29.6% in EW 25, Table 1. Over the same period, its proportion increased from 5.4% to 39.3% in WPR. In the Region of the Americas, PQ.16.1.1 represented 6.4% of available sequences in EW 25, although this estimate was based on only three of 47 sequences. Singapore reported an increase from 12.6% in EW 18 to 55.1% in EW 25. In Hong Kong SAR, the proportion increased from 20.0% to 66.7%, with fluctuations, but the EW 25 estimate was based on four of six sequences submitted that week.

In Singapore, sentinel SARS-CoV-2 test positivity increased rapidly from 6.5% in EW 16 to a peak of 19.8% in EW 19. Test positivity remained elevated through EW 22 before declining through EW 25. The timing and magnitude of the increase were consistent with the seasonal pattern observed in 2024 and 2025. Compared with 2025, however, the period of elevated test positivity was shorter, with a more rapid decline following the peak. No case or death data were reported to WHO during the reporting period [5].

Table 1. Counts and proportions of PQ.16.1.1 among available sequences, EW 18 - EW 25 of 2026

Epidemiological Week (EW)	Global: n (%)*	WPR: n (%)*	AMR: n (%)*	Singapore: n (%)	Hong Kong SAR: n (%)*
2026-W18	16 (2.7%)	16 (5.4%)	0	12 (12.6%)	1 (20.0%)
2026-W19	37 (7.0%)	35 (14.2%)	1 (0.4%)	32 (31.1%)	2 (40.0%)
2026-W20	52 (9.9%)	52 (15.5%)	0	49 (38.6%)	3 (42.9%)
2026-W21	61 (12.8%)	55 (19.3%)	6 (4.1%)	51 (42.2%)	4 (80.0%)
2026-W22	71 (17.3%)	70 (27.7%)	1 (0.8%)	64 (47.4%)	4 (57.1%)
2026-W23	49 (14.3%)	49 (23.9%)	0	38 (51.4%)	7 (53.9%)
2026-W24	58 (21.8%)	57 (30.5%)	1 (1.8%)	49 (53.3%)	5 (62.5%)
2026-W25	73 (29.6%)	70 (39.3%)	3 (6.4%)	65 (55.1%)	4 (66.7%)

Figures by WHO, data from GISAID, extracted on 20 July 2026.
**The number of sequences and proportion are relative to other co-circulating variants at the global, regional and country levels, respectively.*

WHO and its Technical Advisory Group on Virus Evolution (TAG-VE) continue to recommend that Member States prioritize specific actions to address the remaining uncertainties concerning PQ.16.1.1:

- Confirm the preliminary neutralization findings using live-virus and pseudovirus assays with contemporary human sera representative of affected populations and varied vaccination and infection histories.
- Undertake comparative studies of cell entry, replication, fusogenicity and Spike processing in the complete PQ.16.1.1 genetic background.
- Conduct comparative evaluations of hospitalization, intensive-care admission and death, controlling for age, prior immunity, comorbidities and time since vaccination or infection.
- Assess the performance of antigen-based and molecular diagnostic assays and determine phenotypic susceptibility to available direct-acting antivirals.

WHO and its Technical Advisory Group on COVID-19 Vaccine Composition (TAG-CO-VAC) continue to assess the impact of SARS-CoV-2 evolution on the performance of COVID-19 vaccines. In its May 2026 recommendation, WHO TAG-CO-VAC advised the use of monovalent LP.8.1 as a COVID-19 vaccine antigen, while noting that other antigens, including XFG or NB.1.8.1, could also be used if they demonstrate broad and robust neutralizing-antibody responses or effectiveness against circulating variants. Vaccination should not be delayed in anticipation of access to vaccines containing an updated antigen, and populations at highest risk of severe disease remain the priority [6]. No PQ.16.1.1-specific vaccine-effectiveness estimates are currently available.

The risk evaluation below follows the published WHO framework for risk evaluation of SARS-CoV-2 variants [7] and is based on evidence available as of 21 July 2026. This risk evaluation should be revised as additional epidemiological, phenotypic and clinical evidence becomes available.

Overall risk evaluation: **Low**	PQ.16.1.1 is growing rapidly compared with co-circulating variants, particularly in the Western Pacific Region. However, available sequence data are geographically concentrated, with most sequences reported by Singapore. Preliminary phenotypic data indicate that PQ.16.1.1 does not have enhanced ACE2-binding affinity compared with its parent lineage, NB.1.8.1. Neutralization by polyclonal plasma was broadly similar to NB.1.8.1, although PQ.16.1.1 showed substantially reduced susceptibility to several RBD-targeting monoclonal antibodies. There are currently no reports suggesting that PQ.16.1.1 is associated with greater disease severity than other circulating variants. The available evidence on PQ.16.1.1 does not suggest additional public health risks relative to other currently circulating Omicron-descendent lineages.		
Indicator	Evidence	Level of risk	Level of confidence
Growth advantage	As of 8 July 2026, there were 457 PQ.16.1.1 sequences from eight countries submitted to GISAID, representing 29.6% of globally available sequences in EW 25 of 2026. This represents a substantial increase from 2.7% in EW 18. The Western Pacific Region (WPR) accounted for 95.8% of available PQ.16.1.1 sequences, and Singapore alone accounted for 84.9% of the global total. In EW 25, PQ.16.1.1 represented 39.3% of available sequences in the WPR and 55.1% in Singapore. The PQ.16.1.1 receptor-binding domain had lower binding affinity to human ACE2 than that of NB.1.8.1 (KD: 16.5 nM versus 8.22 nM). Soluble human ACE2 showed reduced neutralization of PQ.16.1.1 pseudoviruses compared to NB.1.8.1 (IC50: 0.22 µg/mL versus 0.17 µg/mL). These findings do not indicate that the observed growth advantage is attributable to enhanced ACE2 receptor-binding [4]. *see footnote for more explanations	Moderate	Low
Immune escape	In preliminary pseudovirus experiments using plasma from Wuhan-Hu-1-primed individuals, the geometric mean neutralization titre was 49 against PQ.16.1.1 and 58 against NB.1.8.1. Among individuals with prior Omicron exposure, the corresponding titres were 91 and 85. These results indicate broadly similar polyclonal neutralization of the two variants [4]. Additionally, PQ.16.1.1 showed substantially reduced susceptibility to class 1 RBD-targeting monoclonal antibodies [4].	Moderate	Low

	PQ.16.1.1-specific effectiveness estimates are not currently available for any COVID-19 vaccine. Protection against severe disease is expected to be more preserved than protection against infection [6]. ** see footnote for more explanations		
Severity and clinical/diagnostic considerations	There are no reported or published comparative studies on the clinical severity of PQ.16.1.1. None of the PQ.16.1.1 defining substitutions occur within the principal targets of nirmatrelvir or remdesivir [7]. Increased resistance is therefore not expected from the reported mutation profile alone. Direct phenotypic antiviral-susceptibility data are not currently available. PQ.16.1.1 contains the Nucleocapsid substitution T135I, which warrants assessment against epitopes targeted by antigen-detection assays. There is currently no evidence that PQ.16.1.1 causes antigen-test escape, PCR target failure or reduced diagnostic performance. No lineage-specific diagnostic-performance study has been reported. *** see footnote for more explanations	Low	Low

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Annex:

*** Growth advantage**

Level of risk: Moderate, as PQ.16.1.1 has shown a large and sustained increase in proportion among available sequences and has reached high proportions in Singapore and Hong Kong SAR. Preliminary phenotypic data do not indicate enhanced ACE2-binding affinity compared with NB.1.8.1, and the biological mechanism underlying the observed growth remains uncertain.

Confidence: Low, as available sequences are highly concentrated in the Western Pacific Region, particularly Singapore. Weekly denominators are small and uneven in several settings, and the growth estimate has not been adjusted for country, sampling intensity, reporting delays or changes in sequencing practices. No country-adjusted independent growth estimate or direct cell-entry, replication or fusogenicity study is currently available.

**** Antibody escape**

Level of risk: Moderate, as PQ.16.1.1 contains additional substitutions within recognized RBD and NTD antibody epitopes and preliminary experiments demonstrate substantially reduced susceptibility to several class 1 and class 1/4 monoclonal antibodies. However, neutralization by polyclonal plasma from Wuhan-Hu-1-primed and Omicron-primed individuals was broadly similar to that observed for NB.1.8.1.

Confidence: Low, as the antigenic evidence is derived from one preliminary pseudovirus study. The Omicron-primed cohort was small, and data are not yet available from live-virus assays or from larger and geographically diverse cohorts with contemporary vaccination and infection histories. Additional laboratory studies are required to determine the magnitude and public health relevance of the observed monoclonal-antibody escape.

***** Severity and clinical considerations**

Level of risk: Low, as there are currently no reports of increased disease severity, diagnostic failure or resistance to available direct-acting antivirals associated with PQ.16.1.1.

Confidence: Low. There are no PQ.16.1.1-specific studies assessing clinical outcomes, diagnostic performance or antiviral susceptibility. Reporting of hospitalizations, intensive-care admissions and deaths to WHO has declined substantially, limiting the ability of routine surveillance to detect small changes in severity. Comparative clinical studies and direct assessments of diagnostic and antiviral performance are therefore needed.

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