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WHO TAG-VE Risk Evaluation for SARS-CoV-2 Variant Under Monitoring: PJ.2.1

Executive Summary

PJ.2.1, a JN.1-descendant SARS-CoV-2 lineage, has been designated a variant under monitoring (VUM) with increasing proportions globally, driven largely by detections in the region of the Americas (AMR), particularly in Canada and the United States of America. Considering the available evidence, the additional public health risk posed by PJ.2.1 is evaluated as low at the global level, with low confidence. The lineage emerged through a saltation event and shows high human ACE2-binding capability. Available phenotypic evidence indicates limited additional antibody escape relative to the contemporary variants tested, and sera from recipients of LP.8.1- and XFG-adapted vaccines neutralized PJ.2.1 in laboratory assays. Comparative clinical data are insufficient to determine whether PJ.2.1 is associated with increased disease severity.

Initial Risk Evaluation of PJ.2.1 on 19 November 2026

PJ.2.1 descends from MC.10.1.7 within the KP.3.1.1 branch of the Omicron JN.1 lineage, with the earliest available sequence collected on 25 May 2026 [1]. PJ.2.1 is a highly mutated saltation variant of MC.10.1.7 with 24 amino acid differences in the Spike protein alone, Table 1.

Table 1: Amino Acid differences between other SARS-CoV-2 lineages and PJ.2.1

Variant	AA changes
MC.10.1.7	M:H155N, N:T148A, N:P383L, NS3:V255del, NSP2:A488T, NSP3:T64I, NSP3:T820I, NSP3:P822S, NSP3:I896T, NSP3:T1336I, NSP3:T1677I, NSP4:R401H, NSP4:M426V, NSP5:S121L, S:T22N, S:R190S, S:211del→G, S:I212del, S:G213del, S:R346T, S:N439K, S:K440N, S:H445P, S:S446T, S:W452K, S:T547K, S:S621del, S:V622del, S:A623S, S:N641del, S:V642I, S:Q644E, S:Q675K, S:D839K, S:A958S, S:D1146G, S:I1169F, S:V1264L
XFG*	M:H155N, N:T148A, N:I379T, N:P383L, NS3:V255del, NSP10:S33C, NSP13:T307I, NSP2:A488T, NSP3:T64I, NSP3:D410G, NSP3:T820I, NSP3:P822S, NSP3:I896T, NSP3:T1336I, NSP3:T1465I, NSP3:T1677I, NSP3:L1717S, NSP4:R401H, NSP4:M426V, NSP5:S121L, S:P31del, S:R182K, S:211del→G, S:I212del, S:G213del, S:A435S, S:N439K, S:K440N, S:R444K, S:R445P, S:S446T, S:W452K, S:D487N, S:T547K, S:I572T, S:S621del, S:V622del, S:A623S, S:N641del, S:V642I, S:Q644E, S:Q675K, S:D839K, S:A958S, S:V1104L, S:D1146G, S:I1169F, S:V1264L
NB.1.8.1*	M:H155N, N:T148A, N:Q229K, N:P383L, NS3:H134R, NS3:V255del, NSP1:R47K, NSP10:S33C, NSP12:Y284D, NSP12:S671G, NSP13:P36S, NSP13:T307I, NSP2:A31D, NSP2:F122S, NSP2:A488T, NSP3:T64I, NSP3:V238L, NSP3:Y323H, NSP3:D410G, NSP3:V655A, NSP3:T820I, NSP3:P822S, NSP3:I896T, NSP3:K1155R, NSP3:Q1261P, NSP3:T1336I, NSP3:T1677I, NSP3:N1708S, NSP3:V1891I, NSP3:A1892T, NSP4:R401H, NSP4:M426V, NSP4:F438L, NSP5:S121L, NSP6:V24F, NSP6:R252K, NSP9:T35I, NSP9:S57P, NSP9:L80P, S:S31del, S:S59F, S:S184G, S:R190S, S:211del→G, S:I212del, S:G213del, S:R346T, S:N439K, S:K440N, S:H445P, S:S446T, S:W452K, S:I478K, S:T547K, S:S621del, S:V622del, S:A623S, S:N641del, S:V642I, S:Q644E, S:Q675K, S:D839K, S:A958S, S:V1104L, S:D1146G, S:I1169F, S:V1264L
Amino-acid differences are expressed from the predominant residue in each comparator to the predominant residue in PJ.2.1* * Indicates inclusion of descendant lineages	

Available laboratory evidence indicates that PJ.2.1 has increased human ACE2 receptor-binding capability without substantial additional antibody escape in the comparisons studied. He *et al.* reported that PJ.2.1 bound human ACE2 more strongly than NB.1.8.1, XFG and BA.3.2.2. It remained antigenically similar to circulating JN.1-family variants and showed increased sensitivity to neutralization by the human plasma and antibody panels tested [2]. Consistent with increased receptor engagement, Hoffmann *et al.* found

that PJ.2.1 pseudoviruses were less susceptible to inhibition by an ACE2-blocking antibody than the other variants examined. In plasma from 36 recipients of an LP.8.1-adapted vaccine, PJ.2.1 showed no greater antibody evasion than most tested comparators [3]. Happle *et al.* measured neutralization titres against PJ.2.1 in paired serum samples from 22 participants before and after XFG-adapted mRNA vaccination. The geometric mean titre increased 2.8-fold, from 228 before vaccination to 642, at a median of 14 days after vaccination [4]. Taken together, these findings suggest that increased receptor engagement may contribute to PJ.2.1's observed growth, while substantial additional humoral immune evasion has not been demonstrated. These studies do not establish increased clinical severity or provide a direct estimate of vaccine effectiveness.

As of 8 November 2026, 722 PJ.2.1 sequences had been submitted to GISAID from 18 countries [1]. Globally, the proportion of PJ.2.1 among available sequences increased from 1.0% in EW 33 to 11.6% in EW 41, Table 2. In the Region of the Americas (AMR), its proportion increased from 1.6% in EW 33 to 15.2% in EW 39, dipped to 14.0% in EW 40 and rose to 16.6% in EW 41. In the European Region (EUR), the proportion increased from 1.0% in EW 34 to 6.1% in EW 41. In the Western Pacific Region (WPR), it increased from 0.7% in EW 33 to 5.7% in EW 41.

Canada reported an increase from 3.7% in EW 33 to 23.1% in EW 39, followed by a decline to 19.0% in EW 40 and an increase to 25.7% in EW 41. In the United States of America, the proportion increased from 1.4% to 12.5% between EW 33 and EW 39, before declining to 11.8% in EW 40 and rising to 13.8% in EW 41. Spain reported an overall increase from 1.8% in EW 34 to 3.7% in EW 41, with fluctuations. In France, the proportion reached 13.5% in EW 41. Several other countries reported higher proportions based on small numbers, including Ireland at 17.4% (four sequences), Brazil at 14.3% (three sequences), and New Zealand at 16.7% (one sequence) in EW 40.

These proportions are based on submitted sequences and cannot quantify a relative growth advantage on their own. In addition, geographic differences in sequencing coverage, reporting delays, and small weekly samples limit the representativeness of these estimates.

Beside sporadic detections in the clinical surveillance, early indications of possible community circulation of PJ.2.1 have been observed through wastewater surveillance in New Zealand [5] and Hong Kong SAR (China) [6].

Table 2 Counts and proportions of PJ.2.1 among available sequences in EW 33 to EW 41 of 2026

Epidemiological Week (EW)	Global: n (%)*	AMR: n (%)*	EUR: n (%)*	WPR: n (%)*	Canada: n (%)*	USA: n (%)*	Spain: n (%)*
2026-W33	11 (1.0%)	9 (1.6%)	-	2 (0.7%)	4 (3.7%)	5 (1.4%)	-
2026-W34	20 (1.6%)	16 (2.7%)	3 (1.0%)	1 (0.3%)	6 (8.0%)	10 (2.3%)	3 (1.8%)
2026-W35	31 (2.3%)	27 (3.9%)	4 (1.1%)	-	7 (5.8%)	19 (3.9%)	2 (1.1%)
2026-W36	45 (3.1%)	39 (5.3%)	6 (1.4%)	-	12 (7.5%)	27 (5.2%)	5 (2.4%)
2026-W37	59 (4.0%)	52 (7.1%)	5 (1.0%)	2 (0.8%)	18 (12.2%)	34 (6.2%)	4 (1.5%)
2026-W38	90 (6.1%)	75 (11.0%)	13 (2.3%)	2 (0.9%)	25 (20.7%)	49 (9.0%)	9 (2.8%)
2026-W39	125 (8.7%)	96 (15.2%)	25 (3.9%)	4 (2.8%)	42 (23.1%)	53 (12.5%)	10 (3.1%)
2026-W40	121 (9.5%)	86 (14.0%)	31 (5.7%)	4 (3.3%)	35 (19.0%)	48 (11.8%)	16 (7.1%)
2026-W41	113 (11.6%)	85 (16.6%)	22 (6.1%)	6 (5.7%)	37 (25.7%)	48 (13.8%)	5 (3.7%)

Figures by WHO, data from GISAID, extracted on 8 November 2026.
**Counts represent PJ.2.1 sequences. Percentages are calculated using all available SARS-CoV-2 sequences for the corresponding geography and epidemiological week as the denominator. Dashes indicate no detections of the lineage among the available sequences.*

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:

- Strengthen genomic surveillance and estimate the relative growth advantage of PJ.2.1 against co-circulating lineages, accounting for geographic variation, sampling practices and reporting delays, and reporting uncertainty around estimates.
- Confirm and extend the available antigenic findings using contemporary human sera representative of affected populations and diverse vaccination and infection histories. Assess variant-specific vaccine effectiveness against symptomatic infection and severe disease, accounting for vaccine antigen composition and time since vaccination.
- Conduct comparative evaluations of hospitalization due to COVID-19, oxygen requirement, intensive-care admission and death among individuals infected with PJ.2.1 and co-circulating lineages, controlling for age, comorbidities, prior immunity and time since vaccination or infection.
- Evaluate the performance of antigen-based and molecular diagnostic assays against PJ.2.1 and assess susceptibility to available direct-acting antivirals, supported by surveillance for diagnostic failures and adverse treatment outcomes.

The WHO TAG-CO-VAC statement of 27 October 2026 included PJ.2.1 among emerging variants considered relevant to the upcoming deliberations on COVID-19 vaccine antigen composition which will be held in early December [7]. This inclusion supports prioritizing evidence generation, and should not be interpreted as an assessment of increased severity or a recommendation to change the vaccine antigen [8] .

The risk evaluation below follows the published WHO framework for risk evaluation of SARS-CoV-2 variants [9] and is based on evidence available as of 19 November 2026. This risk evaluation should be revised as additional epidemiological, phenotypic and clinical evidence becomes available.

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Risk evaluation summary

Overall risk evaluation: **Low**	PJ.2.1 is increasing in proportion among available sequences, particularly in the Americas and Europe. PJ.2.1 emerged through a saltation event from the MC.10.1.7 lineage, with substantial changes in its Spike protein. Available phenotypic data indicate increased human ACE2-binding capability compared with contemporary variants, including XFG and NB.1.8.1. Despite this genetic divergence, PJ.2.1 remained antigenically similar to circulating JN.1-family variants, and was neutralized in laboratory assays by sera from recipients of LP.8.1- and XFG-adapted vaccines. These findings suggest that enhanced receptor engagement, rather than substantial additional humoral immune escape, may contribute to its observed growth. There is currently no evidence establishing greater disease severity associated with PJ.2.1, although comparative clinical data remain limited. Considering the available evidence, the additional public health risk posed by PJ.2.1 is evaluated as low at the global level, with low confidence.		
Indicator	Evidence	Level of risk	Level of confidence
Growth advantage	Globally, the proportion of PJ.2.1 among available sequences increased from 1.0% in EW 33 to 11.6% in EW 41. As of 8 November 2026, there were 722 PJ.2.1 sequences from 18 countries submitted to GISAID. In the region of the Americas (AMR), its proportion increased from 1.6% to 16.6% over the same period, with a dip from 15.2% in EW 39 to 14.0% in EW 40. In the European region (EUR), it increased from 1.0% in EW 34 to 6.1% in EW 41. There are also some indications of possible community circulation through wastewater surveillance in countries and areas in the Western Pacific Region [5,6]. He *et al.* reported increased human ACE2-binding capability compared with NB.1.8.1, XFG and BA.3.2.2 [2]. Consistent with increased receptor engagement, Hoffmann *et al.* found reduced susceptibility of PJ.2.1 pseudoviruses to inhibition by an ACE2-blocking antibody [3]. These findings suggest that increased receptor engagement may contribute to the observed growth, but do not directly establish increased transmissibility in humans. ** see footnote for more explanations	Moderate	Low
Immune escape	He *et al.* reported that PJ.2.1 remained antigenically similar to circulating JN.1-family variants and showed increased sensitivity to neutralization by the human plasma and antibody panels tested [2].	Low	Low

	In plasma from 36 recipients of an LP.8.1-adapted vaccine, Hoffmann *et al.* found no greater antibody evasion than most tested comparators [3]. Happle *et al.* reported a 2.8-fold increase in the geometric mean neutralization titre against PJ.2.1 after XFG-adapted mRNA vaccination [4]. Across the studies reviewed, substantial additional humoral immune escape was not demonstrated in the comparisons tested. PJ.2.1-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 [8]. ** see footnote for more explanations		
Severity and clinical/diagnostic considerations	No comparative studies assessing the clinical severity of PJ.2.1 were available in the evidence reviewed. No PJ.2.1-specific evidence demonstrating either reduced susceptibility to available direct-acting antivirals, or impaired antigen-based or molecular diagnostic performance was identified in the literature reviewed. ** see footnote for more explanations	Low	Low

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Annex:

*** Growth advantage**

Level of risk: Moderate, as PJ.2.1 has shown an overall increase in proportion among available sequences globally and in several countries, particularly in the Americas and Europe. Early indications of possible community circulation have also been observed through wastewater surveillance in countries and areas in the Western Pacific Region. Laboratory findings indicate increased human ACE2 receptor engagement, which may contribute to the observed growth.

Confidence: Low, as available sequences are geographically concentrated, and weekly denominators are small and uneven in several settings. No country-adjusted comparative growth estimates with uncertainty intervals were available in the evidence reviewed. Sampling practices, reporting delays and changes in the geographic composition of submitted sequences may influence the observed trends. Laboratory receptor-engagement findings do not directly establish increased transmissibility in humans.

**** Antibody escape**

Level of risk: Low, as available laboratory findings do not demonstrate substantial additional humoral immune escape compared with the contemporary variants examined. PJ.2.1 remained antigenically similar to circulating JN.1-family variants and showed increased sensitivity to neutralization by the human plasma and antibody panels tested in one study. In laboratory assays, PJ.2.1 showed no greater antibody evasion than most tested variants in plasma from LP.8.1-adapted vaccine recipients, and neutralization titres against PJ.2.1 increased after XFG-adapted mRNA vaccination.

Confidence: Low, as the antigenic evidence is derived from a limited number of laboratory studies, and the populations studied do not adequately represent the diversity of contemporary vaccination and infection histories. Additional independent studies using larger and geographically diverse cohorts are needed to establish the breadth and public health relevance of these findings.

***** Severity and clinical considerations**

Level of risk: Low, as the evidence reviewed does not establish increased disease severity, impaired diagnostic performance or reduced susceptibility to available direct-acting antivirals associated with PJ.2.1.

Confidence: Low. There are no comparative PJ.2.1-specific clinical outcome estimates, diagnostic-performance evaluations and phenotypic antiviral-susceptibility data available. 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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