Resource Summary Report

Generated by dkNET on Aug 19, 2026

I1-Hybridoma

RRID:CVCL_G654
Type: Cell Line

Proper Citation

RRID:CVCL_G654

Cell Line Information

URL: https://web.expasy.org/cellosaurus/CVCL_G654

Proper Citation: RRID:CVCL_G654

Description: Cell line I1-Hybridoma is a Hybridoma with a species of origin Mus musculus (Mouse)

Category: Hybridoma

Organism: Mus musculus (Mouse)

Name: I1-Hybridoma

Cross References: CLO:CLO_0004325, ATCC:CRL-2700, Wikidata:Q54897078

ID: CVCL_G654

Hierarchy: cvcl_2199

Record Creation Time: 20220427T220625+0000

Record Last Update: 20260815T234304+0000

Ratings and Alerts

No rating or validation information has been found for I1-Hybridoma.

No alerts have been found for I1-Hybridoma.

Data and Source Information

Source: Cellosaurus

Usage and Citation Metrics

We found 29 mentions in open access literature.

Listed below are recent publications. The full list is available at dkNET .

Ma CB, et al. (2025) Multiple independent acquisitions of ACE2 usage in MERS-related coronaviruses. Cell, 188(6), 1693.

Klute S, et al. (2025) Mutation T9I in Envelope confers autophagy resistance to SARS-CoV-2 Omicron. iScience, 28(7), 112974.

De Cae S, et al. (2025) Ultrapotent SARS coronavirus-neutralizing single-domain antibodies that clamp the spike at its base. Nature communications, 16(1), 5040.

Chen J, et al. (2025) Bat-infecting merbecovirus HKU5-CoV lineage 2 can use human ACE2 as a cell entry receptor. Cell, 188(6), 1729.

Posch W, et al. (2025) Aloe-derived polysaccharides (APS) mitigate SARS-CoV-2 Omicron BQ1.1 infection by preserving epithelial integrity and reducing viral load in human airway epithelial (HAE) cultures. Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie, 192, 118657.

Zhang L, et al. (2024) Rapid spread of the SARS-CoV-2 JN.1 lineage is associated with increased neutralization evasion. iScience, 27(6), 109904.

Addetia A, et al. (2024) Mapping immunodominant sites on the MERS-CoV spike glycoprotein targeted by infection-elicited antibodies in humans. Cell reports, 43(8), 114530.

Tortorici MA, et al. (2024) Persistent immune imprinting occurs after vaccination with the COVID-19 XBB.1.5 mRNA booster in humans. Immunity.

Zhang L, et al. (2024) SARS-CoV-2 BA.2.86 enters lung cells and evades neutralizing antibodies with high efficiency. Cell, 187(3), 596.

He Q, et al. (2024) Neutralization of EG.5, EG.5.1, BA.2.86, and JN.1 by antisera from dimeric receptor-binding domain subunit vaccines and 41 human monoclonal antibodies. Med (New York, N.Y.), 5(5), 401.

Dey S, et al. (2024) Conformational dynamics of SARS-CoV-2 Omicron spike trimers during fusion activation at single molecule resolution. Structure (London, England : 1993), 32(11), 1910.

Pastorio C, et al. (2023) Impact of mutations defining SARS-CoV-2 Omicron subvariants BA.2.12.1 and BA.4/5 on Spike function and neutralization. iScience, 26(11), 108299.

Baggen J, et al. (2023) TMEM106B is a receptor mediating ACE2-independent SARS-CoV-2 cell entry. Cell, 186(16), 3427.

Pastorio C, et al. (2022) Determinants of Spike infectivity, processing, and neutralization in SARS-CoV-2 Omicron subvariants BA.1 and BA.2. Cell host & microbe, 30(9), 1255.

Walls AC, et al. (2022) SARS-CoV-2 breakthrough infections elicit potent, broad, and durable neutralizing antibody responses. Cell, 185(5), 872.

Hoffmann M, et al. (2022) The Omicron variant is highly resistant against antibody-mediated neutralization: Implications for control of the COVID-19 pandemic. Cell, 185(3), 447.

Arora P, et al. (2022) SARS-CoV-2 variants C.1.2 and B.1.621 (Mu) partially evade neutralization by antibodies elicited upon infection or vaccination. Cell reports, 39(5), 110754.

Arora P, et al. (2022) SARS-CoV-2 Omicron sublineages show comparable cell entry but differential neutralization by therapeutic antibodies. Cell host & microbe, 30(8), 1103.

Hoffmann M, et al. (2021) SARS-CoV-2 mutations acquired in mink reduce antibody-mediated neutralization. Cell reports, 35(3), 109017.

Hoffmann M, et al. (2021) SARS-CoV-2 variant B.1.617 is resistant to bamlanivimab and evades antibodies induced by infection and vaccination. Cell reports, 36(3), 109415.