Human ACE-2 Antibody

Monoclonal Mouse IgG_1 Clone # 171607

Catalog Number: MAB9333

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ACE-2 in direct ELISAs.
Source	Monoclonal Mouse IgG_1 Clone # 171607
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ACE-2 Gln18-Ser740 Accession # Q9BYF1
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS.

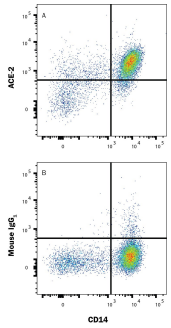
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Flow Cytometry	0.25 µg/10^6 cells	Human PBMC
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA

Flow Cytometry



Detection of ACE-2 in Human PBMC by Flow Cytometry. Human PBMC were stained with (A) Mouse Anti-Human ACE-2 Monoclonal Antibody (Catalog # MAB9333) or (B) Mouse IgG1 Isotype Control Antibody (Catalog # MAB002) followed by Allophycocyanin-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # F0101B) and Mouse anti-Human CD14 Phycoerythrin-conjugated Monoclonal Antibody (Catalog # FAB3832P). Staining was performed using our Staining Membrane-associated Proteins protocol.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449

Human ACE-2 Antibody

Monoclonal Mouse IgG_1 Clone # 171607

Catalog Number: MAB9333

BACKGROUND

Angiotensin I Converting Enzyme (ACE-2), also called ACEH (ACE homologue), is a dimeric, zinc-dependent metalloprotease of the ACE family that also includes somatic and germinal ACE (1, 2). ACE-2 mRNA is found at high levels in heart, testis, and kidney and at lower levels in a wide variety of tissues (1, 3). ACE-2 is the SARS-CoV and SARS-CoV2 Spike protein receptor *in vivo* (4-6), functions catalytically as a carboxypeptidase to cleave several substrates including angiotensins I and II, and acts as a partner for B0AT1-family amino acid transporters (1, 2). Through these functions, ACE-2 has been shown to be involved in several diseases including SARS, COVID19, acute lung injury (4, 7), heart disease (8), liver and lung fibrosis (9), inflammatory lung disease (10), and cardiopulmonary disease (11). Full length ACE-2 protein includes an extracellular region composed of a single N-terminal peptidase domain and C-terminal collectrin-like domain (CLD), a transmembrane domain, and a short cytoplasmic tail (12). The N-terminal peptidase region is required for binding to SARS-CoV and SARSCoV2 spike proteins, while the CLD contains a region that promotes dimerization and association with amino acid transporters (2). The peptidase domain contains a long deep cleft that undergoes a large hinge-bending movement at substrate and inhibitor binding (12). Classical ACE inhibitors such as captopril and lisinopril do not inhibit ACE-2 activity and inhibitors of ACE-2 do not inhibit ACE activity (13).

References:

1. Kuba, K. *et al.* (2010) Pharmacol. Ther. **128**:119.
2. Yan, *et al.* (2020) Science **367**:1444.
3. Tipnis, S.R. *et al.* (2000) J. Biol. Chem. **275**:33238.
4. Kuba, K. *et al.* (2005) Nature Med. **11**:875.
5. Hoffman, M. *et al.* (2020) Cell.**181**:1.
6. Wrapp, *et al.* (2020) Science **367**:1260.
7. Imai, Y. *et al.* (2005) Nature **436:**112.
8. Huang, L. *et al.* (2003) J. Biol. Chem. **278**:15532.
9. Schrom, E. *et al.* (2017) Mol. Therapy Nuc. Acid **7**:350.
10. Jia, H. *et al.* (2016) Shock. **46**:239.
11. Cole-Jeffrey, C.T. *et al.* (2015) J. Cadiovasc. Pharmacol. **66**:540.
12. Towler, P. *et al.* (2004) J. Biol. Chem. **279**:17996.
13. Crackower, M.A. *et al.* (2002) Nature **417**:822.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 **Canada** TEL 855 668 8722 **China** TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449