

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived				
	Mouse Integrin $\alpha 1$ (Phe29-Pro1141) Accession # Q3V3R4	His-Pro	GGGSGGGG	Acidic Tail	6-His tag
	Mouse Integrin $\beta 1$ (Gln21-Asp728) Accession # P09055	His-Pro	GGGSGGGG	Basic Tail	
	N-terminus				C-terminus

N-terminal Sequence Phe29 (Integrin $\alpha 1$) & No results obtained: Gln21 predicted (Integrin $\beta 1$), sequencing might be blocked

Analysis

Structure / Form Noncovalently-linked heterodimer

Predicted Molecular Mass 132 kDa (Integrin $\alpha 1$) & 86.5 kDa (Integrin $\beta 1$)

SPECIFICATIONS

SDS-PAGE 120-135 kDa & 180-215 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Collagen I is immobilized at 5 $\mu\text{g}/\text{mL}$, Recombinant Mouse Integrin $\alpha 1\beta 1$ can bind with an apparent $K_d < 5 \text{ nM}$.
Optimal dilutions should be determined by each laboratory for each application.

Endotoxin Level <0.10 EU per 1 μg of the protein by the LAL method.

Purity >95%, by SDS-PAGE with silver staining.

Formulation Lyophilized from a 0.2 μm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 500 $\mu\text{g}/\text{mL}$ in PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 $^{\circ}\text{C}$ as supplied.
- 1 month, 2 to 8 $^{\circ}\text{C}$ under sterile conditions after reconstitution.
- 3 months, -20 to -70 $^{\circ}\text{C}$ under sterile conditions after reconstitution.

BACKGROUND

Integrin $\alpha 1\beta 1$, also called VLA1, is the only $\alpha 1$ integrin family adhesion receptor, one of twelve integrins that share the $\beta 1$ subunit, and one of four collagen-binding integrins (1-6). It is the non-covalent heterodimer of 190-210 kDa $\alpha 1$ (CD49a) and 130 kDa $\beta 1$ (CD29) type I transmembrane glycoprotein subunits. It is found on cells including activated T cells, B cells, monocytes, vascular smooth muscle cells, osteoblasts and adipocytes (2, 7-9). The $\alpha 1$ extracellular domain (ECD) contains an I (inserted) domain which includes the ligand binding site (2, 4). The $\beta 1$ ECD contains a vWFA domain, which participates in binding (3). Each subunit then has a transmembrane sequence and a short cytoplasmic tail. Divalent cations and intracellular (inside-out) signaling convert the dimer from the folded, inactive form to its most active, extended conformation (1, 2). The 1113 amino acid (aa) mouse $\alpha 1$ extracellular domain (ECD) shares 88% and 96% aa sequence identity with human and rat $\alpha 1$, respectively, while the 708 aa mouse $\beta 1$ ECD shares 93% and 98% aa sequence identity with human and rat $\beta 1$, respectively. $\alpha 1\beta 1$ preferentially binds collagens I, IV, VI, XIII and XVI, but also binds laminin (4-11). $\alpha 1\beta 1$ is reported to down-regulate EGF R signaling, increase expression of caveolin-1, reduce production of reactive oxygen species, regulate collagen expression, control MMP collagenase and gelatinase activity, and mediate the renal basement membrane disorder Alport syndrome (11-13). These effects may begin by $\alpha 1\beta 1$ binding of caveolin-1, initiating signaling pathways that involve the phosphatase TC-PTP, kinases ERK and p38, and the transcription factor PPAR- γ (11-14). $\alpha 1\beta 1$ down-regulates MMP-mediated angiostatin formation, enhancing tumor vascularization (9). $\alpha 1\beta 1$ -null mice are deficient in fibroblast collagen IV and laminin-mediated cell spreading and migration, show defects in bone healing, and are resistant to Alport renal fibrosis (10-12, 15). When expressed in the same epithelial cells, $\alpha 1\beta 1$ negatively regulates integrin $\alpha 2\beta 1$ -mediated cell adhesion and migration (16).

References:

1. Takada, Y. *et al.* (2007) *Genome Biol.* **8**:215.
2. Luo, B-H. *et al.* (2007) *Annu. Rev. Immunol.* **25**:619.
3. Argraves, W.S. *et al.* (1987) *J. Cell Biol.* **105**:1183.
4. Briesewitz, R. *et al.* (1993) *J. Biol. Chem.* **268**:2989.
5. Holers, V.M. *et al.* (1989) *J. Exp. Med.* **169**:1589.
6. Tulla, M. *et al.* (2001) *J. Biol. Chem.* **276**:48206.
7. Eble, J.A. *et al.* (2006) *J. Biol. Chem.* **281**:25745.
8. Hall, D.E. *et al.* (1990) *J. Cell Biol.* **110**:2175.
9. Pozzi, A. *et al.* (2000) *Proc. Natl. Acad. Sci. USA* **97**:2202.
10. Gardner, H. *et al.* (1999) *J. Cell Sci.* **112**:263.
11. Dennis, J. *et al.* (2010) *Am. J. Pathol.* **177**:2527.
12. Cosgrove, D. *et al.* (2008) *Am. J. Pathol.* **172**:761.
13. Chen, X. *et al.* (2010) *Mol. Cell. Biol.* **30**:3048.
14. Borza, C.M. *et al.* (2010) *J. Biol. Chem.* **285**:40114.
15. Gardner, H. *et al.* (1996) *Dev. Biol.* **175**:301.
16. Abair, T.D. *et al.* (2008) *Exp. Cell Res.* **314**:3593.