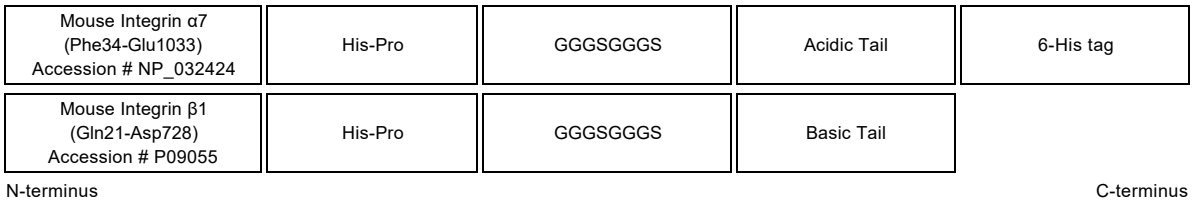
Recombinant Mouse Integrin α7β1

Catalog Number: 7958-A7

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived mouse Integrin alpha 7 beta 1 protein



N-terminal Sequence Analysis	Phe34, Glu915 (Integrin α7) & Gln21 predicted, No results obtained: sequencing might be blocked (Integrin β1)
Structure / Form	Noncovalently-linked heterodimer
Predicted Molecular Mass	119 kDa (Integrin α7, full length), 22.4 kDa (Integrin α7, N-terminus starts at Glu915) & 86.5 kDa (Integrin β1)

SPECIFICATIONS

SDS-PAGE	123-157 kDa, 95-100 kDa & 38-42 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When mouse Laminin I (Catalog # 3400-010-02) is coated at 10 µg/mL, Recombinant Mouse Integrin α7β1 binds with an apparent K_D <0.5 nM.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 400 µg/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 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Integrin α7β1, also called VLA-7 (very late antigen-7), is the major laminin-binding integrin in cardiac and skeletal muscle (1-4). The non-covalent heterodimer is composed of ~150 kDa α7 and 130 kDa β1/CD29 type I transmembrane glycoprotein subunits with short cytoplasmic tails (2). While α7 pairs only with β1, twelve integrins share the β1 subunit (1-5). The longest version of α7 is the X1X2B form, encoding 1179 amino acids (aa). Six alternatively spliced 1116-1160 aa isoforms of the α7 subunits have short extracellular (X1, X2) or cytoplasmic (A, C) deletions. Isoforms are differentially expressed by tissue and developmental stage and may show preferences for specific laminins (3-5). The β1 vWFA domain participates with the α7 FG-GAP motifs in ligand binding. The α7 subunit is cleaved into extracellular heavy and transmembrane/cytoplasmic light chains (3). The mouse α7 heavy chain shares 89%, 90%, 87% and 85% aa sequence identity with human, rat, feline and bovine α7, and the mouse β1 ECD shares 98% aa identity with rat and 93-94% with human, bovine, porcine, ovine, canine and feline β1. The α7 heavy chain in species other than mouse may also be cleaved at aa 603-605 by a serine protease; fragments remain associated. This form enhances the active, unfolded and open conformation, promoting cell adhesion and spreading (1, 2, 6). Adhesion of α7β1 to laminin-111 accounts for many of its effects, but α7β1 also binds most other laminins (5). It protects muscle from exercise-induced damage, and its absence in humans or mice causes a form of muscular dystrophy (7-9). α7β1 is also expressed in vascular smooth muscle (VSM), and is important for development of the cerebral vasculature (10). VSM cells show increased α7β1 expression and enhanced laminin binding in injury-induced atherosclerosis or PDGF treatment (11, 12). Deletion of α7 results in VSM hyperplasia, especially in response to injury (13).

References:

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