

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human Integrin alpha V beta 3 protein		
	Human ITGAV (Phe31-Val992) Accession # AAA36808.1	IEGR	Human IgG ₁ (Glu99-Lys330) (with modifications)
	Human ITGB3 (Gly27-Asp718) Accession # P05106.2	HPIEGR	Human IgG ₁ (Glu99-Lys330) (with modifications)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Phe 31 (Integrin alpha V) & Gly 27 (Integrin Beta 3)		
Structure / Form	Disulfide linked heterodimer		
Predicted Molecular Mass	133 kDa (Integrin alpha V) & 103 kDa (Integrin beta 3)		

SPECIFICATIONS

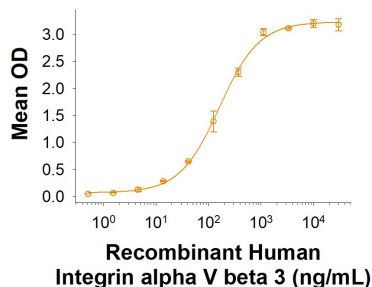
SDS-PAGE	>190 kDa, under non-reducing conditions.
Activity	Measured by its binding ability in a functional ELISA. Recombinant Human Integrin alpha V beta 3 Fc Chimera (Catalog # 11648-AV) binds to Recombinant Human Vitronectin (Catalog # 2308-VN) with an ED ₅₀ of 60.0-600 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 250 µg/mL in water.
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. <ul style="list-style-type: none"> 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.

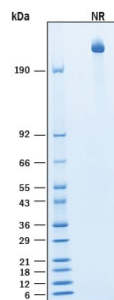
DATA

Binding Activity



Recombinant Human Integrin alpha V beta 3 Fc Chimera Protein Binding Activity. In a functional ELISA, Recombinant Human Integrin alpha V beta 3 Fc Chimera Protein (Catalog # 11648-AV) binds to Recombinant Human Vitronectin (Catalog # 2308-VN) with an ED₅₀ of 60.0-600 ng/mL.

SDS-PAGE



Recombinant Human Integrin alpha V beta 3 Fc Chimera Protein SDS-PAGE. 2 µg/lane of Recombinant Human Integrin alpha V beta 3 Fc Chimera Protein (Catalog # 11648-AV) was resolved with SDS-PAGE under non-reducing (NR) condition and visualized by Coomassie® Blue staining, showing bands at >190 kDa.

BACKGROUND

Integrin $\alpha\text{V}\beta 3$ together with $\alpha\text{IIb}\beta 3$, constitutes the only known $\beta 3$ Integrins (1-3). The non-covalent heterodimer of 170 kDa $\alpha\text{V}/\text{CD}51$ and 93 kDa $\beta 3/\text{CD}61$ subunits shows wide expression, notably by endothelial cells and osteoclasts (2-4). Each subunit has a transmembrane sequence and a short cytoplasmic tail connected to the cytoskeleton. Active cell surface $\alpha\text{V}\beta 3$ adheres to matrix proteins including vitronectin, fibronectin, fibrinogen and thrombospondin (2, 3). The ligand binding site of $\alpha\text{V}\beta 3$ is in the N-terminal head region, formed by interaction of the $\beta 3$ vWFA domain with the αV beta-propeller structure (4). The αV subunit contributes a thigh and a calf region, while the $\beta 3$ subunit contains a PSI domain and four cysteine-rich I-EGF folds. The αV subunit domains termed thigh, calf-1 and calf-2 generate a "knee" region that is bent when the $\alpha\text{V}\beta 3$ is in its constitutively inactive state. Activation, either by "inside out" signaling or by Mg^{2+} or Mn^{2+} binding, extends the Integrin to expose its ligand binding site (1, 4). The 962 aa human αV ECD(11) shares 92-95% aa sequence identity with mouse, rat and bovine αV while the 685 aa human $\beta 3$ ECD(12) shares 95% aa identity with equine and canine, and 89-92% aa identity with mouse, rat and porcine $\beta 3$. Two splice variants of $\beta 3$ (b and c) diverge over the last 21 amino acids (aa) and lack cytoplasmic phosphorylation sites (5, 6). Another $\beta 3$ splice variant diverges after the vWFA domain, producing a soluble 60 kDa form in platelets and endothelial cells (7). $\alpha\text{V}\beta 3$ is essential for the maturation of osteoclasts and their binding and resorption of bone; it also, however, promotes their apoptosis (8, 9). M-CSF R and $\alpha\text{V}\beta 3$ share signaling pathways during osteoclastogenesis, and deletion of either molecule causes osteopetrosis (8, 9). $\alpha\text{V}\beta 3$ is involved in several other signaling pathways by direct interaction with receptor tyrosine kinases and ligands. For example, it cooperates with endothelial cell VEGF R2 in angiogenesis, and with IGF-1 to promote cancer cell proliferation and invasiveness (13, 14). Also, cell entry of several viruses is mediated by $\alpha\text{V}\beta 3$ (4, 10).

References:

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