## Recombinant Human Integrin αVβ5

**Catalog Number:** 2528-AV

### Description

**Source:** Chinese Hamster Ovary cell line, CHO-derived

<table>
<thead>
<tr>
<th>Structure / Form</th>
<th>Activity</th>
<th>Stability &amp; Storage</th>
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<tbody>
<tr>
<td>Noncovalently-linked heterodimer</td>
<td>Measured by its binding ability in a functional ELISA.</td>
<td>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</td>
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### SDS-PAGE

**Predicted Molecular Mass:** 115.4 kDa (αV subunit) & 84.8 kDa (β5 subunit)

### Stability & Storage

- 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 αVβ5 is one of five αV integrins and the only known β5 integrin (1-3). The non-covalent heterodimer of 170 kDa αV and 100-110 kDa β5 integrin type I transmembrane glycoprotein subunits is expressed on a wide variety of cell types including keratinocytes, fibroblasts, adhesive monocytes, embryonic stem cells, and select endothelium and epithelium (4-8). αVβ5 binds ligands containing an RGD motif, notably vitronectin (1-10). The 962 aa human αV extracellular domain (ECD) shares 92-95% aa sequence identity with mouse, rat and cow αV ECD shares 89%-93% aa identity with mouse, rat, bovine, equine, and canine β5. The αV ECD contains an N-terminal β-propeller structure, followed by domains termed thigh, calf1 and calf2 (1). The 799 aa β5 contains a VWA domain within the ECD, which interacts with the αVβ-propeller to form a binding domain. Each subunit has a transmembrane sequence and a short cytoplasmic tail. Potential β5 isoforms include a 691 aa form with an alternate start site at aa 109, a 958 aa form with an alternate N-terminus, and a 795 aa form with an alternate C-terminus. Post-translational modifications, such as proteolytic cleavage of the αV subunit or phosphorylation of the β5 cytoplasmic tail, can increase endocytic turnover of the αVβ5 protein and/or promote cell migration (7-10). Growth factors that increase PKC activity, such as VEGF or TGF-α, promote αVβ5-mediated angiogenesis while β3, which may be expressed in the same cell, responds to FGF-basic and TNF-α (11). An inhibitor of both down-regulates tumor angiogenesis (12). During lung inflammation, up-regulation of αVβ5 on myofibroblasts or infiltrating lymphocytes may contribute to fibrosis by freeing TGF-β from latency (13, 14). On retinal pigment epithelia, αVβ5 is important for normal diurnal phagocytosis of outer rod segments, and contributes to adhesion of retinal cells (15).

### References