# Biotinylated Human CD96 / TACTILE Protein, His,Avitag™

Catalog # TAE-H82E3

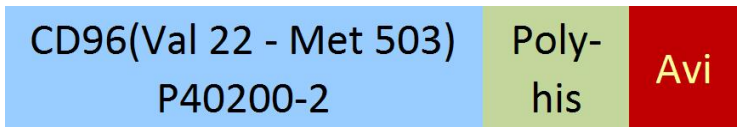
## Synonym

TACTILE

## Source

Biotinylated Human CD96, His,Avitag(TAE-H82E3) is expressed from human 293 cells (HEK293). It contains AA Val 22 - Met 503 (Accession # P40200-2). Predicted N-terminus: Val 22

## Molecular Characterization




This protein carries a polyhistidine tag at the C-terminus, followed by an Avi tag (Avitag™).

The protein has a calculated MW of 57.3 kDa. The protein migrates as 120-130 kDa under reducing (R) condition (SDS-PAGE) due to glycosylation.

## Labeling

***Biotinylation of this product is performed using Avitag™ technology. Briefly, the single lysine residue in the Avitag is enzymatically labeled with biotin.***

## Protein Ratio

Passed as determined by the HABA assay / binding ELISA.

## Purity

>80% as determined by SDS-PAGE.

## Formulation

Lyophilized from 0.22 μm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

## Reconstitution

Please see Certificate of Analysis for specific instructions.

***For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.***

## Storage

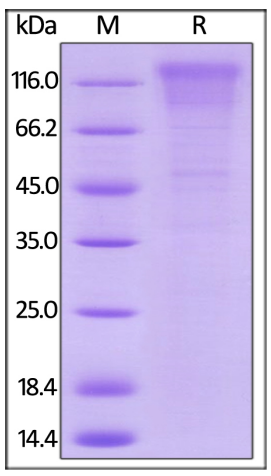
For long term storage, the product should be stored at lyophilized state at -20°C or lower.

***Please avoid repeated freeze-thaw cycles.***

This product is stable after storage at:

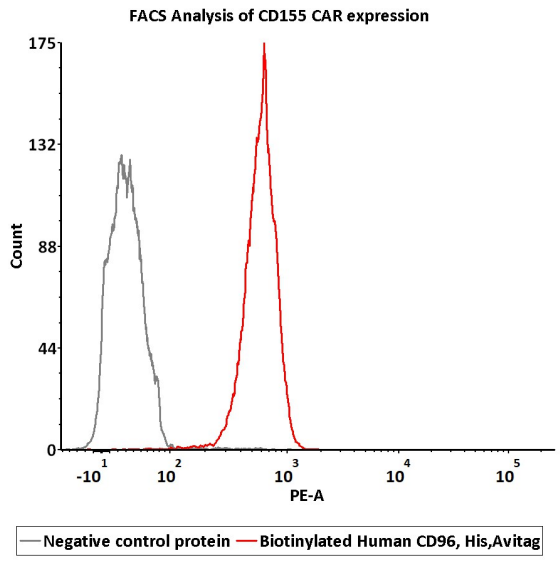
- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

## SDS-PAGE




Biotinylated Human CD96, His,Avitag on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 80%.

## Bioactivity-CELL BASE




**FACS analysis of Biotinylated Human CD96, His,Avitag binding to CD155 cells overexpressing CD155.**

2e5 of CD155 cells overexpressing CD155 were stained with 100 μL of 1 μg/mL of Biotinylated Human CD96, His,Avitag (Cat. No. TAE-H82E3) and negative control protein respectively, washed and then followed by PE-SA antibody and analyzed with FACS (Routinely tested).

## Background

The progression of pancreatic cancer (PC) is significantly associated with tumor immune escape, which may be associated with nature killer (NK) cell dysfunction. CD226, CD96, and TIGIT, which share the ligand CD155, play important roles in the regulation of NK cell function. The present study was conducted to investigate the roles of these molecules in NK cells from PC patients.

TIGIT and CD96 together with the co-stimulatory receptor CD226 form a pathway that is analogous to the CD28/CTLA-4 pathway, in which shared ligands and differential receptor:ligand affinities fine-tune the immune response. Although the roles of TIGIT and CD96 as immune checkpoint receptors in T cell and natural killer cell biology are just beginning to be uncovered, accumulating data support the targeting of these receptors for improving anti-tumor immune responses. A clear understanding of the immune cell populations regulated by TIGIT and CD96 is key to the design of immunotherapies that target these receptors in combination with other existing immune checkpoint blockade therapies.

The dysfunction of CD96 may trigger C syndrome: A syndrome characterized by trigonocephaly, severe mental retardation, hypotonia, variable cardiac defects, redundant skin, and dysmorphic facial features, including upslanted palpebral fissures, epicanthal folds, depressed nasal bridge, and low-set, posteriorly rotated ears.