

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human HepaCAM protein		
	Human HepaCAM (Val34-Ser240) Accession # Q19CZ8.1	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Val34		
Structure / Form	Disulfide-linked homodimer		
Predicted Molecular Mass	50 kDa		

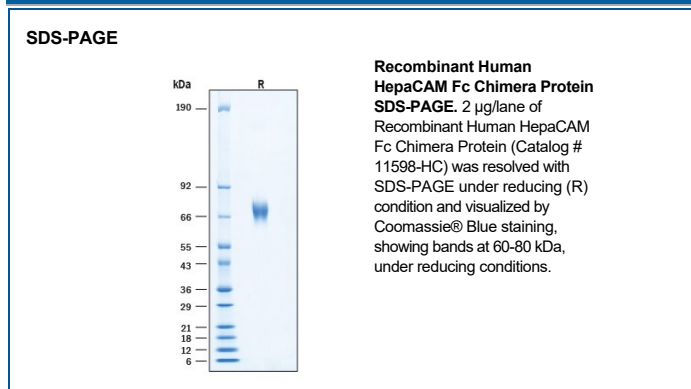
SPECIFICATIONS

SDS-PAGE	60-80 kDa, under reducing conditions
Activity	Bioassay data are not available.
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 500 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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



BACKGROUND

Hepatocyte cell adhesion molecule (HepaCAM), also known as glial cell adhesion molecule (GlialCAM), is a type I transmembrane glycoprotein in the Ig-superfamily that participates in cell migration and proliferation (ref). HepaCAM consists of an extracellular domain (ECD) with two C2 Ig-like domains, a transmembrane region, and an intracellular region containing a SH3 domain. Mature, human HepaCAM shares 99% amino acid sequence identity with mouse HepaCAM. A second, truncated isoform is known to exist as a result of alternative splicing. Though first detected in the liver, HepaCAM expression has subsequently been detected in glial cells of the central nervous system. HepaCAM forms homodimers, through cis-interactions, on the cell surface and this interaction is known to modulate cell-matrix interactions. HepaCAM has been shown to suppress the growth of hepatocytes and is down-regulated in hepatocellular carcinoma in the liver. In the brain, HepaCAM is normally expressed in astrocytes where it regulates ion homeostasis, BBB physiology, and synaptic excitation. Loss of HepaCAM signaling has been reported to impair gap-junction cell coupling and the balance between synaptic excitation and inhibition. Multiple studies indicate that HepaCAM is a tumor suppressor candidate, but its exact role remains unknown. In Glioblastoma, HepaCAM helps mediate interactions, through its IgG-like domains, with proteins such as Mlc1 and aquaporin-4 and loss of HepaCAM expression results in a proinvasive environment. In prostate cancer, HepaCAM may help inhibit cancer progression as a reduction or absence of HepaCAM expression is seen in majority of cases. Additionally, HepaCAM is suppressed in multiple other carcinomas including breast, kidney, colon, rectum and stomach making it a potential therapeutic target.

References:

1. Moh, M. C., *et al.* (2005) *J. Hepatol.* **42**:833.
2. Moh, M.C., *et al.* (2005) **280**:27366.
3. Sofroniew, M.V. (2021) *Neuron* **109**:2365.
4. Baldwin, K.T. *et al.* (2021) *Neuron* **109**:2427.
5. De, A., *et al.* (2023) *J Neurosci.* **43**:8043.
6. Deng Q, *et al.* (2019) *Mol Med Rep* **19**:2115.
7. Moh, M.C., *et al.* (2008) *Carcinogenesis* **29**:2298.