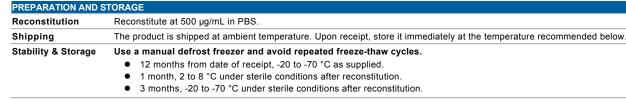
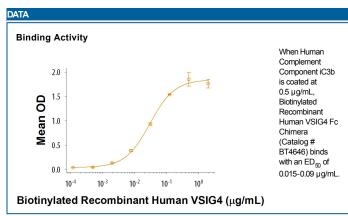


Biotinylated Recombinant Human VSIG4 Fc Chimera

Catalog Number: BT4646

DESCRIPTION			
Source	Mouse myeloma cell line, NS0-derived		
	Human VSIG4 (Arg20-Pro283) Accession # Q9Y279	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	a Arg20		
Structure / Form	Disulfide-linked homodimer. Biotinylated via sugars		
Predicted Molecular Mass	56 kDa (unlabeled)		
SPECIFICATIONS			
SDS-PAGE	60-77 kDa and 150 - 195 kDa, reducing conditions		
Activity	Measured by its binding ability in a functional ELISA. When Human complement component iC3b is immobilized at 0.5 μg/mL,100 μL/well, the concentration of Biotinylated Recombinant Human VSIG4 Fc Chimera that produces 50% of the optimal binding response is 0.015-0.09 μg/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.		





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BACKGROUND

VSIG4 (V-set and immunoglobulin domain containing 4), also known as CRIg and Z39IG, is a 45 kDa, type I transmembrane protein of the B7 family within the Ig superfamily that is expressed only in tissue-resident macrophages (1-4). The gene is located on the X chromosome (2). The human VSIG4 cDNA encodes 399 amino acids (aa) including a 19 aa signal sequence, a 264 aa extracellular domain (ECD) containing a V-type and a C2-type Ig domain, a 21 aa transmembrane domain and a 95 aa cytoplasmic domain (3). The human VSIG4 ECD shares 84% aa identity with canine VSIG4. Within the IgV domain, it shares 90%, 80% and 78% aa identity with bovine, mouse and rat VSIG4, respectively; these animals lack the C2-type domain. Splice isoforms of 321, 305, 272, 201 and 199 aa lack all or part of the cytoplasmic domain, the C2-type Ig domain and/or the transmembrane domain (5). VSIG4 is specifically expressed on macrophages in the thymic medulla, peritoneum, alveoli, synovia, adipose and heart, liver Kupffer cells, placental Hofbauer cells, and atherosclerotic foam cells (1-4, 6-9). It is absent on infiltrating macrophages (8). VSIG4 is a complement receptor that binds C3b and iC3b fragments, internalizes them to recycling endosomes, and is recycled to the cell surface (4, 6). It contributes significantly to innate immunity by binding and phagocytosis of complement-opsonized invading pathogens (4, 8, 10). Binding of either native or recombinant soluble VSIG4 to C3b inhibits complement amplification through the alternative, but not classical, pathway (10, 11). VSIG4 is also a negative regulator of mouse and human T cell activation (2). Although VSIG4 engagement may activate NFkB and thus be pro-inflammatory in some cases, many of its activities are important in resolving, rather than initiating, inflammation (1, 2, 7, 10, 11).

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