

#### DESCRIPTION

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse VSIG4 in direct ELISAs. In direct ELISAs, less than 5% cross-reactivity with recombinant human (rh) VSIG4 is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant mouse VSIG4 His20-Pro187 Accession # NP_808457
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

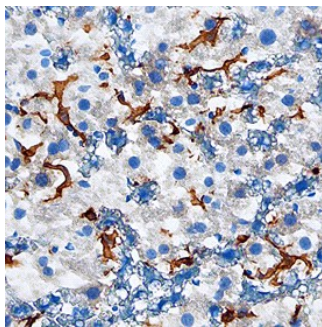
#### APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Immunohistochemistry</b>	5-15 µg/mL	See Below

#### DATA

**Immunohistochemistry**



**VSIG4 in Mouse Liver.** VSIG4 was detected in perfusion fixed frozen sections of mouse liver using Goat Anti-Mouse VSIG4 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF4674) at 1.7 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to Kupffer cells. View our protocol for [Chromogenic IHC Staining of Frozen Tissue Sections](#).

#### PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

#### BACKGROUND

Mouse VSIG4 (V-set and immunoglobulin domain containing 4), also known as CRIG and Z39IG, is a type I transmembrane glycoprotein that is a B7 family-related protein and an Ig superfamily member (1-2). Mouse VSIG4 is synthesized as a 280 amino acid (aa) precursor that contains a signal sequence, an IgV-type immunological domain (aa 36-115), one potential N-linked glycosylation site, and a single transmembrane domain (2). The IgV domain of mouse VSIG4 shares 86% and 80% aa sequence identity with the IgV domains of rat and human VSIG4, respectively. Quantitative PCR reveals that VSIG4 mRNA is expressed at high levels in the liver, dendritic cells, neutrophils, and macrophages, and at lower levels in the lung, heart, spleen, and lymph nodes (1). No VSIG4 expression appears to be present in T and B cells (1). The use of polyclonal rabbit serum against the extracellular domain of VSIG4 demonstrates that VSIG4 is specifically expressed on naïve resting tissue macrophages, and that the expression is downregulated or lost upon activation (1). Furthermore, histological analysis shows expression of VSIG4<sup>+</sup> macrophages only in the liver, thymic medulla, and heart (1). VSIG4 macrophages are not detected in the intestines, kidney, skeletal muscle, lymph node, splenic white pulp, lung, or brain (1). Studies show that VSIG4/Fc Chimera strongly inhibits proliferation of anti-CD3 as well as anti-CD3/anti-CD28-stimulated T cells (1). Indeed, VSIG4 functions as a negative regulator of mouse as well as human T cell activation, and may be involved in the maintenance of peripheral T cell tolerance and/or unresponsiveness (1). In addition, VSIG4's expression on Kupffer cells is required for efficient binding and phagocytosis of complement C3 opsonized particles (2). VSIG4 acts as a macrophage complement receptor by binding complement fragments C3b and iC3b (2). VSIG4 binding to C3b inhibits complement activation through the alternative pathway, making it a potent suppressor of established inflammation (3-4).

#### References:

1. Vogt, L. *et al.* (2006) *J. Clin. Invest.* **116**:2817.
2. Helmy, K. *et al.* (2006) *Cell* **124**:915.
3. Katschke, K.J. *et al.* (2007) *J. Exp. Med.* **204**:1319.
4. Wiesmann, C. *et al.* (2006) *Nature* **444**:217.