

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived mouse VSTM2A protein Ser25-Ala252, with a C-terminal 6-His tag Accession # NP_001277468.1
N-terminal Sequence Analysis	Ser25
Predicted Molecular Mass	26 kDa

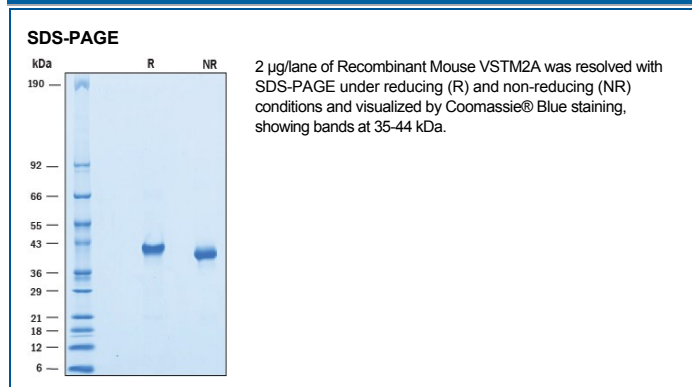
SPECIFICATIONS

SDS-PAGE	35-44 kDa, reducing conditions
Activity	Measured by its ability to inhibit anti-CD3 antibody induced IFN-gamma secretion by human peripheral blood mononuclear cells (PBMC). The ED ₅₀ for his effect is 2-12 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. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<ul style="list-style-type: none"> • 12 months from date of receipt, ≤ -20 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, ≤ -20 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

V-set and transmembrane domain-containing protein 2A (VSTM2A) is a secreted glycoprotein that is expressed by committed preadipocytes. N-linked glycosylation is crucial for its secretion, but not for preadipocyte cell differentiation activity. It is expressed during adipocyte development and its over-expression promotes adipogenesis (1). VSTM2A is highly expressed in the brain and *Vstm2a* was identified as an enigmatic gene that is highly produced in mouse brain (1, 2). A positive association has been observed between *Vstm2a* and *Pparg2*. PPARγ2 indirectly promotes *Vstm2a* expression in preadipocytes by amplifying adipogenic commitment, while VSTM2A promotes *Pparg2* expression by activating BMP signaling (1, 3). VSTM2A is synthesized as a precursor protein that contains a 24 amino acid (aa) signal sequence followed by the VSTM2A domain. The mouse VSTM2A shares 78% and 99% aa sequence identity with human and rat VSTM2A, respectively. Adipogenic commitment was uninhibited by over-expression of VSTM2A lacking a signal peptide for secretion, suggesting that secreted VSTM2A is not involved in adipogenic commitment *in vitro*. While the exact role secreted VSTM2A plays is unknown, it is suggested it may modulate angiogenesis or neurogenesis due to its expression in the brain and near blood vessels, and the need for adipose tissue to develop alongside blood vessels and neural tissue (1, 4). Our data shows that mouse VSTM2A inhibits anti-CD3 induced IFN-γ secretion in Human T cells.

References:

1. Secco, B. *et al.* (2017) Cell Rep. **18**:93.
2. Pandey, A.K. *et al.* (2014) PloS One. **9**:e88889.
3. Berry, D.C. *et al.* (2013) Development. **140**:3939.
4. Nishimura, S. *et al.* (2007) Diabetes. **56**:1517.