Recombinant SOCS3 Monoclonal Antibody

catalog number: **AN302003L**

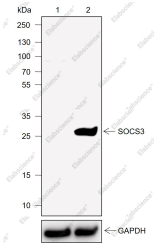
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Mouse
Immunogen	Peptide. This information is proprietary to PTMab
Host	Rabbit
Isotype	Rabbit IgG
Clone	C531624
Purification	Protein A affinity purification
Conjugation	Unconjugated
Buffer	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications	Recommended Dilution
WB	1:1000-1:2000

Data



Western Blot with SOCS3 Monoclonal Antibody at dilution of 1:2000. Lane 1: RAW264.7, Lane 2: RAW264.7+LPS(10 ng, ml 48h)

Observed-MW:15 kDa/25 kDa

Calculated-MW:25 kDa

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze/thaw cycles.
Shipping	Ice bag

Background

The suppressor of cytokine signaling (SOCS) family members are negative regulators of cytokine signal transduction that inhibit the Jak/Stat pathway. The SOCS family consists of at least 8 members including the originally identified cytokine-inducible SH2-containing protein (CIS1), as well as SOCS1-7. Each SOCS family member contains a central SH2 domain and a conserved carboxy-terminal motif designated as the SOCS box. These proteins are important regulators of cytokine signaling, proliferation, differentiation, and immune responses. Low levels of SOCS3 are observed in lung, spleen, and thymus and, like other SOCS family members, its expression is rapidly induced by a number of factors including interleukins, EPO, IFN-γ, CSF, and TNF-α. SOCS3 uses its SH2 domain to bind activated Jaks and their cognate receptors to provide negative feedback inhibition. In addition to the initially described inducers of SOCS3 expression, subsequent studies have described SOCS3-mediated negative feedback inhibition for leptin, GH, chemokine receptors, insulin, and certain pathogens. SOCS3 deletion results in embryonic lethality with placental insufficiency. SOCS3 signaling has been linked pathologically to allergic responses, inflammatory disease, endotoxic shock, wound repair, and obesity.

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