bs-13591R **[Primary Antibody]**

IFI30 Rabbit pAb

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DATASHEET

Host: Rabbit **Isotype:** IgG

Clonality: Polyclonal

GeneID: 10437 **SWISS:** P13284

Target: IFI30

Immunogen: KLH conjugated synthetic peptide derived from human IFI30/GILT: 181-250/250.

Purification: affinity purified by Protein A

Concentration: 1mg/ml

Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Background: Proteins internalized into the endocytic pathway are usually degraded. Efficient proteolysis requires denaturation, induced by acidic conditions within lysosomes, and reduction of inter- and intrachain disulfide bonds. Cytosolic reduction is mediated enzymatically by thioredoxin. In the endocytic pathway, reduction of protein disulfide bonds is important for the generation of MHC class II-peptide complexes. This process is catalyzed by a gamma-interferon-inducible thiol reductase (GILT). GILT is synthesized as a precursor, and following delivery to MHC class II-containing compartments (MIICs), is processed to the mature form via cleavage of amino- and carboxy-terminal propeptides. A lysosomal thiol reductase, GILT, is optimally active at low pH and capable of catalyzing disulfide bond reduction both in vivo and in vitro. GILT is expressed constitutively in antigen-presenting cells and is induced by g-interferon in other cell types, suggesting a potentially important role in antigen processing. Additionally, T cell recognition of select exogenous and endogenous epitopes is dependent on tumor cell expression of GILT. The absence of GILT in melanomas alters antigen processing and the hierarchy of immunodominant epitope presentation.

Applications: **ELISA** (1:5000-10000)

Reactivity: (predicted: Human, Mouse, Rat)

Predicted MW.: 29 kDa

Subcellular Location: Secreted ,Cytoplasm