# RAG-2 Mouse mAb

CatalogNo: YM0550

## Key Features

Host Species
- Mouse

Reactivity
- Human

Applications
- WB,ELISA

MW
- 59kD (Calculated)

## Recommended Dilution Ratios

**WB 1:500-1:2000**
**ELISA 1:10000**
**Not yet tested in other applications.**

## Storage

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|---|---|
| **Storage*** | -15°C to -25°C/1 year(Do not lower than -25°C) |
| **Formulation** | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. |

## Basic Information

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| **Clonality** | Monoclonal |

## Immunogen Information

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|---|---|
| **Immunogen** | Purified recombinant fragment of human RAG-2 (350-527aa) expressed in E. Coli. |
| **Specificity** | RAG-2 Monoclonal Antibody detects endogenous levels of RAG-2 protein. |

## Target Information

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|---|---|
| **Gene name** | RAG2 |

**Protein Name** V(D)J recombination-activating protein 2

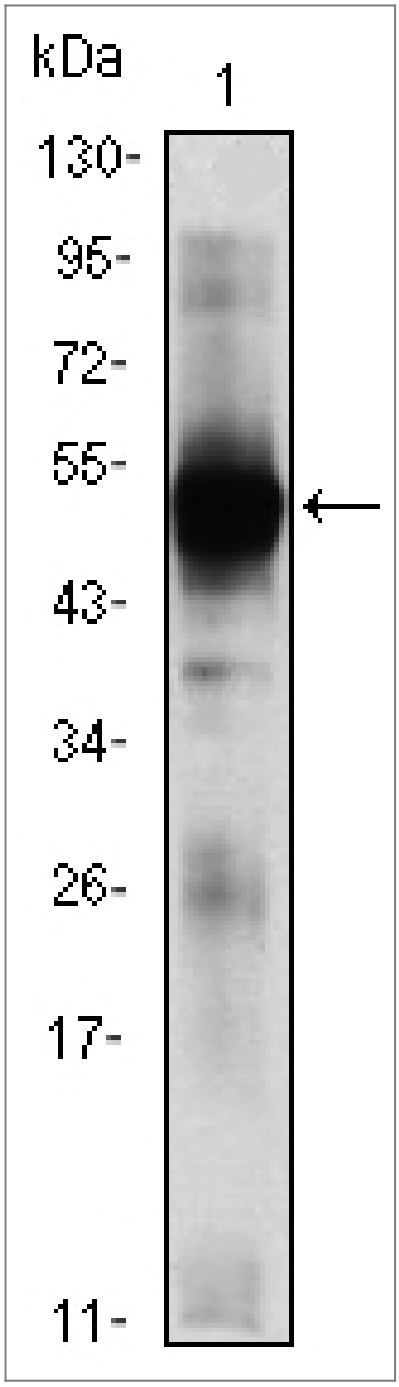
| Organism | Gene ID | UniProt ID |
|---|---|---|
| Human | 5897; | P55895; |
| Mouse | | P21784; |

**Cellular Localization** Nucleus .

**Tissue specificity** Cells of the B- and T-lymphocyte lineages.

**Function** Disease:Defects in RAG2 are a cause of combined cellular and humoral immune defects with granulomas (CHIDG) [MIM:233650]. CHIDG is an immunodeficiency disease with granulomas in the skin, mucous membranes, and internal organs. Other characteristics include hypogammaglobulinemia, a diminished number of T and B cells, and sparse thymic tissue on ultrasonography.,Disease:Defects in RAG2 are a cause of Omenn syndrome (OS) [MIM:603554]; a severe immunodeficiency characterized by the presence of activated, anergic, oligoclonal T-cells, hypereosinophilia, and high IgE levels.,Disease:Defects in RAG2 are a cause of severe combined immunodeficiency, autosomal recessive T cell-negative, B-cell-negative, NK cell-positive (T(-)B(-)NK(+)SCID) [MIM:601457]. SCID refers to a genetically and clinically heterogeneous group of rare congenital disorders characterized by impairment of both humoral and cell-mediated immunity, leukopenia, and low or absent antibody levels. Patients with SCID present in infancy with recurrent, persistent infections by opportunistic organisms. The common characteristic of all types of SCID is absence of T-cell-mediated cellular immunity due to a defect in T-cell development.,Function:During lymphocyte development, the genes encoding immunoglobulins and T-cell receptors are assembled from variable (V), diversity (D), and joining (J) gene segments. This combinatorial process, known as V(D)J recombination, allows the generation of an enormous range of binding specificities from a limited amount of genetic information. The RAG1/RAG2 complex initiates this process by binding to the conserved recombination signal sequences (RSS) and introducing a double-strand break between the RSS and the adjacent coding segment. These breaks are generated in two steps, nicking of one strand (hydrolysis), followed by hairpin formation (transesterification). RAG1/2 has also been shown to function as a transposase in vitro, and to possess RSS-independent endonuclease activity (end processing) and hairpin opening. RAG1 alone can bind to RSS but stable, efficient binding requires RAG2. All known catalytic activities require the presence of both proteins.,online information:RAG2 deficiency database,similarity:Belongs to the RAG2 family.,subunit:The RAG complexes appear to contain three to five molecules of RAG2 for each molecule of RAG1.,tissue specificity:Cells of the B- and T-lymphocyte lineages.,

## Validation Data




Western Blot analysis using RAG-2 Monoclonal Antibody against RAG2-hIgGFc transfected HEK293 (1)cell lysate.

## Contact information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Please scan the QR code to access additional product information:
**RAG-2 Mouse mAb**