

**B2M Antibody**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO1895a****Specification****B2M Antibody - Product Information**

Application	E, WB, FC, IHC
Primary Accession	<a href="#">P61769</a>
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	IgG2a
Calculated MW	13.7kDa KDa

**Description**

This gene encodes a serum protein found in association with the major histocompatibility complex (MHC) class I heavy chain on the surface of nearly all nucleated cells. The protein has a predominantly beta-pleated sheet structure that can form amyloid fibrils in some pathological conditions. A mutation in this gene has been shown to result in hypercatabolic hypoproteinemia.

**Immunogen**

Purified recombinant fragment of human B2M (AA: 21-100) expressed in E. Coli.

**Formulation**

Purified antibody in PBS with 0.05% sodium azide.

**B2M Antibody - Additional Information**

**Gene ID** 567

**Other Names**

Beta-2-microglobulin, Beta-2-microglobulin form pl 5.3, B2M

**Dilution**

E~~1/10000  
WB~~1/500 - 1/2000  
FC~~1/200 - 1/400  
IHC~~1/200 - 1/1000

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

B2M Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**B2M Antibody - Protein Information**

**Name** B2M ([HGNC:914](#))

### Function

Component of the class I major histocompatibility complex (MHC). Involved in the presentation of peptide antigens to the immune system. Exogenously applied M.tuberculosis EsxA or EsxA-EsxB (or EsxA expressed in host) binds B2M and decreases its export to the cell surface (total protein levels do not change), probably leading to defects in class I antigen presentation (PubMed:<a href="http://www.uniprot.org/citations/25356553" target="\_blank">25356553</a>).

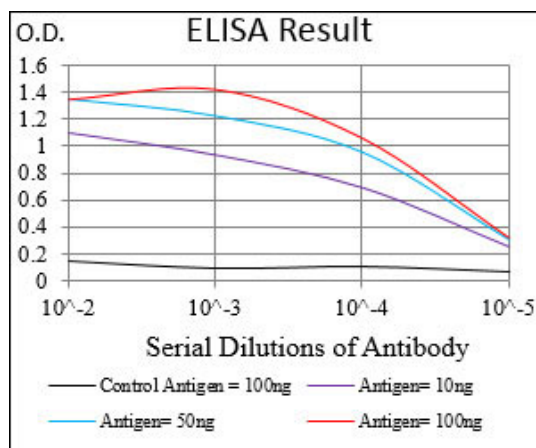
### Cellular Location

Secreted. Cell surface. Note=Detected in serum and urine (PubMed:1336137, PubMed:7554280). {ECO:0000269|PubMed:7554280, ECO:0000269|Ref.6}

## B2M Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)



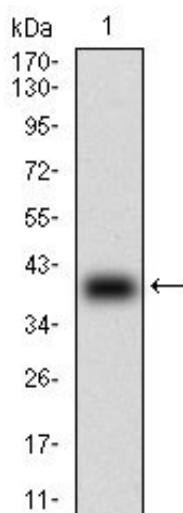


Figure 1: Western blot analysis using B2M mAb against human B2M (AA: 21-100) recombinant protein. (Expected MW is 35.4 kDa)

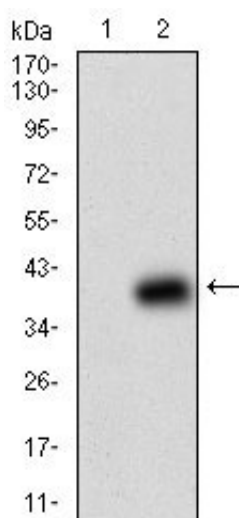


Figure 2: Western blot analysis using B2M mAb against HEK293 (1) and B2M (AA: 21-100)-hIgGFc transfected HEK293 (2) cell lysate.

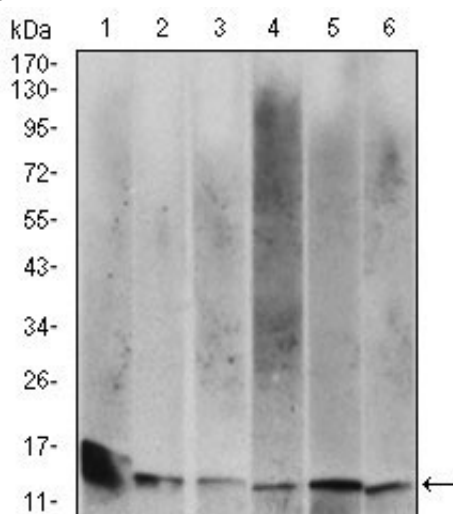


Figure 3: Western blot analysis using B2M mouse mAb against HeLa (1), HEK293 (2), HepG2 (3), RAJI (4), A431 (5) and Jurkat (6) cell lysate.

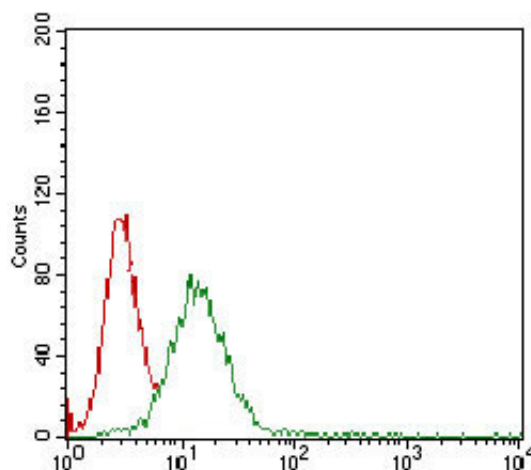


Figure 4: Flow cytometric analysis of A431 cells using B2M mouse mAb (green) and negative control (red).

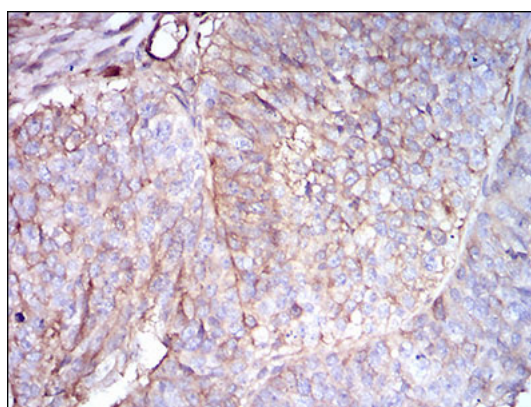


Figure 5: Immunohistochemical analysis of paraffin-embedded ovarian cancer tissues using B2M mouse mAb with DAB staining.

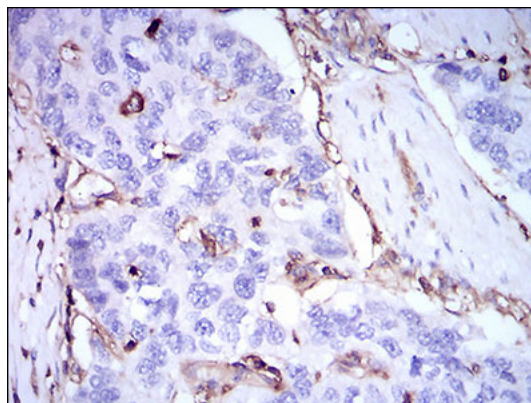


Figure 6: Immunohistochemical analysis of paraffin-embedded esophageal cancer tissues using B2M mouse mAb with DAB staining.

## B2M Antibody - Background

The protein encoded by this gene is a regulatory subunit of the AMP-activated protein kinase (AMPK). AMPK is a heterotrimer consisting of an alpha catalytic subunit, and non-catalytic beta and gamma subunits. AMPK is an important energy-sensing enzyme that monitors cellular energy status. In response to cellular metabolic stresses, AMPK is activated, and thus phosphorylates and inactivates acetyl-CoA carboxylase (ACC) and beta-hydroxy beta-methylglutaryl-CoA reductase (HMGCR), key enzymes involved in regulating de novo biosynthesis of fatty acid and cholesterol. This subunit is one of the gamma regulatory subunits of AMPK. Alternatively spliced transcript variants

encoding distinct isoforms have been observed. ;

#### **B2M Antibody - References**

1. Cancer Immunol Immunother. 2012 Sep;61(9):1359-71.
2. Lupus. 2012 Sep;21(10):1098-104.