

# Mouse HMGB1 / HMG1 Protein (Fc Tag)

Catalog Number: 50913-M01H



Sino Biological  
Biological Solution Specialist

## General Information

### Gene Name Synonym:

amphoterin; DEF; HMG-1; Hmg1; p30; SBP-1

### Protein Construction:

A DNA sequence encoding the mouse HMGB1 (P63158) (Met 1-Glu 215) was fused with the Fc region of human IgG1 at the N-terminus.

**Source:** Mouse

**Expression Host:** HEK293 Cells

## QC Testing

**Purity:** > 85 % as determined by SDS-PAGE

### Bio Activity:

Measured by its ability to bind mouse AGER-His in functional ELISA.

### Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

### Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

**Predicted N terminal:** Glu

### Molecular Mass:

The recombinant mouse HMGB1/Fc is a disulfide-linked homodimer. The reduced monomer comprises 475 amino acids and has a calculated molecular mass of 53.3 kDa. The apparent molecular mass of the monomer is approximately 57 kDa in SDS-PAGE under reducing conditions.

### Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

## Usage Guide

### Storage:

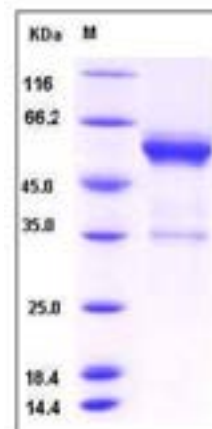
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

**Avoid repeated freeze-thaw cycles.**

### Reconstitution:

Detailed reconstitution instructions are sent along with the products.

## SDS-PAGE:



## Protein Description

High-mobility group box 1 protein (HMGB1), also known as HMG-1 or amphoterin previously, is a member of the HMGB family consisting of three members, HMGB1, HMGB2 and HMGB3. HMGB1 is a DNA-binding nuclear protein, released actively following cytokine stimulation as well as passively during cell death. It is the prototypic damage-associated molecular pattern (DAMP) molecule and has been implicated in several inflammatory disorders. HMGB1 signals via the receptor for advanced glycation end-product (RAGE) and members of the toll-like receptor (TLR) family. The most prominent HMGB1 protein and mRNA expression arthritis is present in pannus regions, where synovial tissue invades articular cartilage and bone. HMGB1 promotes the activity of proteolytic enzymes, and osteoclasts need HMGB1 for functional maturation. As a non-histone nuclear protein, HMGB1 has a dual function. Inside the cell, HMGB1 binds DNA, regulating transcription and determining chromosomal architecture. Outside the cell, HMGB1 can serve as an alarmin to activate the innate system and mediate a wide range of physiological and pathological responses. Extracellular HMGB1 represents an optimal "necrotic marker" selected by the innate immune system to recognize tissue damage and initiate reparative responses. However, extracellular HMGB1 also acts as a potent pro-inflammatory cytokine that contributes to the pathogenesis of diverse inflammatory and infectious disorders. HMGB1 has been successfully therapeutically targeted in multiple preclinical models of infectious and sterile diseases including arthritis. As shown in studies on patients as well as animal models, HMGB1 can play an important role in the pathogenesis of rheumatic disease, including rheumatoid arthritis, systemic lupus erythematosus, and polymyositis among others. In addition, enhanced postmyocardial infarction remodeling in type 1 diabetes mellitus was partially mediated by HMGB1 activation.

## References

- 1.Ulloa L, *et al.* (2006) High-mobility group box 1 (HMGB1) protein: friend and foe. *Cytokine Growth Factor Rev.* 17 (3): 189-201.
- 2.Pisetsky DS, *et al.* (2008) High-mobility group box protein 1 (HMGB1): an alarmin mediating the pathogenesis of rheumatic disease. *Arthritis Res Ther.* 10 (3): 209.
- 3.Volz HC, *et al.* (2010) The role of HMGB1/RAGE in inflammatory cardiomyopathy. *Semin Thromb Hemost.* 36(2): 185-94.

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