Data Sheet (Cat.No.TMPY-02504)

TargetMol

Carboxypeptidase M Protein, Mouse, Recombinant (His)

General Information

Synonyms: E030045M14Rik;AA589379;carboxypeptidase M;5730456K23Rik;1110060I01Rik

Protein Construction: Leu18-Ser423

Species: Mouse

Expression Host: HEK293 Cells

Accession: Q80V42-1

Molecular Weight: 47.51 kDa (Predicted); 55-60 kDa (Reducing conditions due to glycosylation)

QC Testing

Biological Activity: Measured by its ability to release L-arginine from Benzoyl-Ala-Arg, with detection of the arginine amino group by o-phthaldialdehyde. The specific activity is >40,000 pmol/min/μg. (QC Test)

Purity: > 95% as determined by Tris-Bis PAGE; > 95% as determined by HPLC

Endotoxin: < 1.0 EU/μg of the protein as determined by the LAL method.

Formulation: Lyophilized from 0.22μm filtered solution in PBS (pH 7.4). Normally 8% trehalose is added as protectant before lyophilization.

Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in distilled water. The product concentration should not be less than 100 μg/ml. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Carboxypeptidase M (CPM) is a glycosylphosphatidylinositol anchored enzyme that plays an important role in the kallikrein-kinin system (KKS). CPM catalytic domain hydrolyzes Arg from C-terminal peptides (i.e., bradykinin and kallidin), generating des-Arg-kinins, the agonists of B1 receptor (B1R).

Reference

Deddish PA. et al., 1990, J Biol Chem. 265 (25): 15083-9.
Nagae A. et al., 1992, J Neurochem. 59 (6): 2201-12.
Skidgel RA. et al., 1996, Immunopharmacology. 32 (1-3): 48-52.
Deiteren K. et al., 2009, Clin Chim Acta. 399 (1-2): 24-39.

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