PRODUCT INFORMATION

Metalloendopeptidase OMA1 (human, recombinant)

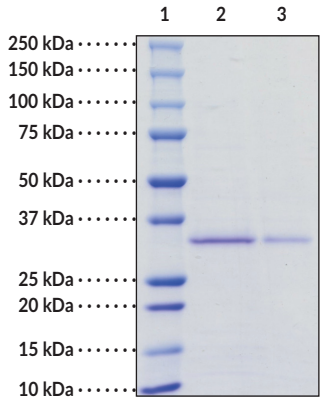
Item No. 28258

Overview and Properties

Synonyms:	Metalloprotease-related Protein 1, MPRP-1, OMA1, Overlapping with the m-AAA Protease 1 Homolog
Source:	Recombinant N-terminal His-tagged OMA1 expressed in *E. coli*
Amino Acids:	217-524
Uniprot No.:	Q96E52
Molecular Weight:	37.1 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	***batch specific*** (≥90% estimated by SDS-PAGE)
Supplied in:	50 mM Tris, pH 8.0, with 150 mM sodium chloride, 10% glycerol, 0.5 M L-arginine, and 2 µM zinc chloride
Protein Concentration:	***batch specific*** mg/ml

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: Metalloendopeptidase OMA1 (4 µg)
Lane 3: Metalloendopeptidase OMA1 (2 µg)

Representative gel image shown; actual purity may vary between each batch.

Cayman's OMA1 has an expected size of 37.1 kDa, though SDS-PAGE shows it running closer to 30 kDa. We have confirmed the 30 kDa band is OMA1 by mass spectrometry.

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Description

OMA1 is an ATP-independent metalloproteinase encoded by *OMA1*.[1] It is localized to the mitochondrial inner membrane and is comprised of a matrix-facing N-terminal domain, a transmembrane domain, and a C-terminal M48 metallopeptidase domain that is exposed to the intermembrane space.[1,2] Under various stress conditions, including oxidative stress, heat stress, and mitochondrial membrane depolarization, OMA1 is activated and cleaves long isoforms of the GTPase optic atrophy 1 (OPA1) at the S1 cleavage site, leading to inhibition of mitochondrial fusion and increased mitochondrial fragmentation.[1-3] Under stress conditions, OMA1 is also autocatalytically degraded, thereby limiting, and allowing for reversal of, the stress response.[2,3] *Oma1*$^{-/-}$ mouse embryonic fibroblasts exhibit a loss of mitochondrial fragmentation upon exposure to hydrogen peroxide.[2] Mice lacking *Oma1* exhibit impaired thermogenesis, increased hepatic steatosis and serum triglyceride levels, and high-fat diet-induced obesity.[4] Cayman's Metalloendopeptidase OMA1 (human, recombinant) can be used for Western blot and ELISA applications.

References

1. Levytskyy, R.M., Bohovych, I., and Khalimonchuk, O. Metalloproteases of the inner mitochondrial membrane. *Biochemistry* **56(36)**, 4737-4746 (2017).
2. Baker, M.J., Lampe, P.A., Stojanovski, D., *et al*. Stress-induced OMA1 activation and autocatalytic turnover regulate OPA1-dependent mitochondrial dynamics. *EMBO J*. **33(6)**, 578-593 (2014).
3. Quirós, P.M., Langer, T., and López-Otín, C. New roles for mitochondrial proteases in health, ageing and disease. *Nat. Rev. Mol. Cell Biol*. **16(6),** 345-359 (2015).
4. Quirós, P.M., Ramsay, A.J., Sala, D., *et al*. Loss of mitochondrial protease OMA1 alters processing of the GTPase OPA1 and causes obesity and defective thermogenesis in mice. *EMBO J*. **31(9),** 2117-2133 (2012).

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