PRODUCT INFORMATION

Hsp90β (human, recombinant)

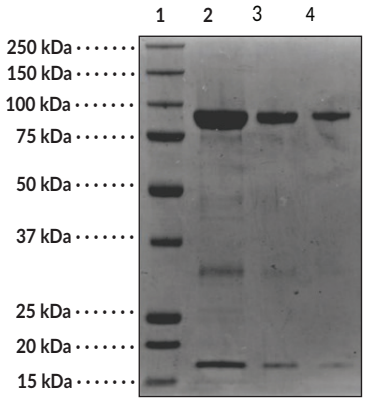
Item No. 22735

Overview and Properties

Synonyms:	Heat Shock Protein 90β
Source:	Active N-terminal histidine-tagged human Hsp90β protein (full length) purified from *E. coli*
Amino acids:	2-724 (full length)
Uniprot No.:	P08238
Molecular Weight:	85 kDa
Storage:	-65°C (as supplied); avoid freeze/thaw cycles by storing protein in aliquots
Stability:	≥6 months
Purity:	***batch specific*** (≥80% as estimated by SDS-PAGE)
Supplied in:	50 mM HEPES, pH 8.0, 150 mM sodium chloride, 1 mM DTT, 20% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	ATPase activity confirmed by ADP detection assay

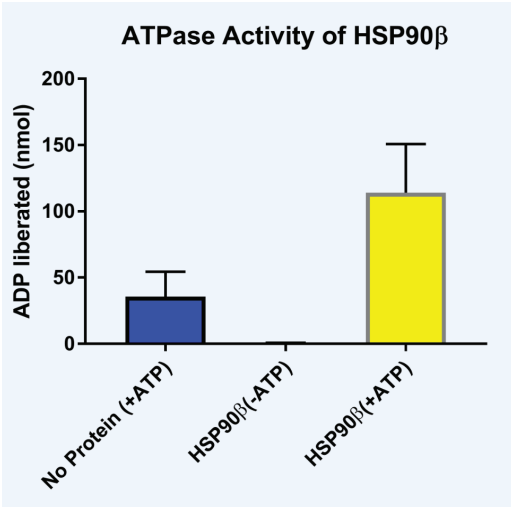
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: Hsp90β (4 µg)
Lane 3: Hsp90β (2 µg)
Lane 4: Hsp90β (1 µg)

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



CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.CAYMANCHEM.COM

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Description

Heat shock protein 90 β (Hsp90β) is the constitutively active cytosolic isoform of Hsp90 that is encoded by *HSP90AB* in humans.[1] Hsp90 is a multidomain protein that functions as a molecular chaperone to assist in folding and activation of nascent peptides, refolding unfolded or misfolded proteins, and preventing protein aggregation.[2] C-terminal dimerization of Hsp90, coupled with ATPase molecular clamp activity induces a conformational change in the N-terminal nucleotide binding domain that facilitates substrate binding and initiates the chaperone cycle.[3] Hsp90 interacts with many co-chaperones during its chaperone cycle including p23 and Sba1, which help recruit substrates to the Hsp90 complex, Hsp70 (Item Nos. 22739 | 23002), which loads nascent polypeptides onto the Hsp90 dimer, and the ATPase activator Aha1 that promotes ATP hydrolysis and substrate release.[4,5] Hsp90 is overexpressed in cancer cells and stabilizes client proteins that promote oncogenesis, including transcription factors, signaling proteins, and kinases.[1,5] Hsp90 also decreases α-synuclein fibril formation and toxicity as well as Q35 aggregation in *in vitro* models of Parkinson's and Huntington's disease, respectively, implying a role in neurodegenerative disease.[6]

References

1. Chen, B., Piel, W.H., Gui, L., *et al*. The HSP90 family of genes in the human genome: Insights into their divergence and evolution. *Genomics* **86(6)**, 627-637 (2005).
2. Fink, A.L. Chaperone-mediated protein folding. *Physiol. Rev.* **79(2)**, 425-449 (1999).
3. Prodromou, C., Panaretou, B., Chohan, S., *et al*. The ATPase cycle of Hsp90 drives a molecular 'clamp' via transient dimerization of the N-terminal domains. *EMBO J.* **19(16)**, 4383-4392 (2000).
4. Ali, M.M.U., Roe, S.M., Vaughan, C.K., *et al*. Crystal structure of an Hsp90-nucleotide-p23/Sba1 closed chaperone complex. *Nature* **440(7087)**, 1013-1017 (2006).
5. Li, J. and Buchner, J. Structure, function and regulation of the Hsp90 machinery. *Biomed. J.* **36(3)**, 106-117 (2013).
6. Lackie, R.E., Maciejewski, A., Ostapchenko, V.G., *et al*. The Hsp70/Hsp90 chaperone machinery in neurodegenerative diseases. *Front. Neurosci.* **11:254**, (2017).

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