PRODUCT INFORMATION

TEAD2 Cofactor-Binding Domain (human, recombinant)

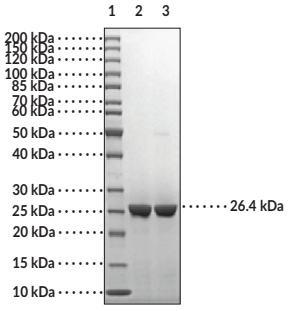
Item No. 41074

Overview and Properties

Synonyms: Embryonic TEA Domain-containing Factor, ETF, TEA Domain Family Member 2, TEA Domain Transcription Factor 2, TEF-4, Transcriptional Enhancer Factor 4
Source: Recombinant human TEAD2 cofactor-binding domain expressed in insect cells
Amino Acids: 217-447
Uniprot No.: Q15562
Molecular Weight: 26.4 kDa
Storage: -80°C (as supplied); avoid repeated freeze/thaw cycles
Stability: ≥1 year
Supplied in: 50 mM HEPES, pH 7.5, with 200 mM sodium chloride, 20% glycerol, and 1 mM DTT
Protein Concentration: ***batch specific*** mg/ml
Special Conditions: Rapid thaw with running water.

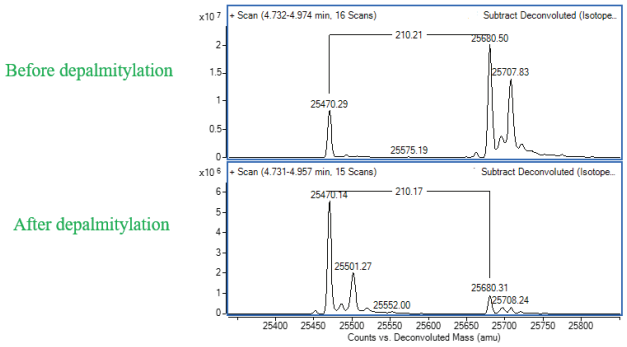
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



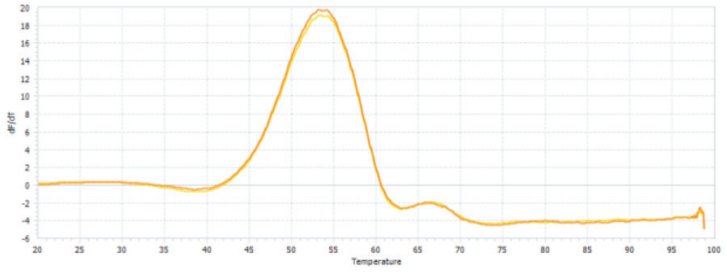
Lane 1: MW Markers
Lane 2: TEAD2 Cofactor-Binding Domain (2 µg, reduced)
Lane 3: TEAD2 Cofactor-Binding Domain (2 µg, non-reduced)

SDS-PAGE Analysis of TEAD2 Cofactor-Binding Domain.



TEAD2 Cofactor-Binding Domain (human, recombinant) depalmitoylation

Melting profile of TEAD2 Cofactor-Binding Domain



WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent *via* email to your institution.

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Description

TEA domain transcription factor 2 (TEAD2) is a transcription factor and member of the TEA domain-containing family of transcription factors.[1] It is composed of an N-terminal TEA DNA-binding domain, a hydrophobic proline-rich domain, and a C-terminal cofactor-binding domain (CBD), which binds TAZ or YES-associated transcriptional regulator (YAP), or vestigial-like proteins (VGLLs), and is involved in TAZ-, YAP-, or VGLL-dependent transcription of genes in the Hippo signaling pathway, which regulates embryonic and organ development, proliferation, and cell death.[1-3] The tertiary structure and function of TEAD2 requires C-terminal palmitoylation at residue 380.[4] TEAD2 binds to the promoter for *Pou5f1*, the gene encoding octamer-binding transcription factor 3 (Oct3), and is necessary for mouse embryonic stem cell self-renewal.[5] Increased tumor levels of *TEAD2* mRNA are associated with poor survival, angiogenesis, tumor progression, and increased expression of genes associated with the epithelial-to-mesenchymal transition (EMT) in patients with hepatocellular carcinoma (HCC).[6] Cayman's TEAD2 Cofactor-Binding Domain (human, recombinant) protein consists of 230 amino acids, has a calculated molecular weight of 26.4 kDa, and is depalmitoylated at cysteine 380.

References

1. Landin-Malt, A., Benhaddou, A., Zider, A., *et al*. An evolutionary, structural and functional overview of the mammalian TEAD1 and TEAD2 transcription factors. *Gene* **591(1)**, 292-303 (2016).
2. Tian, W., J., Y., Tomchick, D.R., *et al*. Structural and functional analysis of the YAP-binding domain of human TEAD2. *Proc. Natl. Acad. Sci. U S A* **107(16)**, 7293-7298 (2010).
3. Hillen, H., Candi, A., Vanderhoydonck, B., *et al*. A novel irreversible TEAD inhibitor, SWTX-143, blocks hippo pathway transcriptional output and causes tumor regression in preclinical mesothelioma models. *Mol. Cancer Ther.* **23(1)**, 3-13 (2023).
4. Noland, C.L., Gierke, S., Schnier, P.D., *et al*. Palmitoylation of TEAD transcription factors is required for their stability and function in Hippo pathway signaling. *Structure* **24(1)**, 179-186 (2016).
5. Tamm, C., Böwer, N., and Annerén, C. Regulation of mouse embryonic stem cell self-renewal by a Yes-YAP-TEAD2 signaling pathway downstream of LIF. *J. Cell Sci.* **124(Pt 7)**, 1136-1144 (2011).
6. Joo, J.S., Cho, S.Y., Rou, W.S., *et al*. TEAD2 as a novel prognostic factor for hepatocellular carcinoma. *Oncol. Rep*. **43(6)**, 1785-1796 (2020).

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