PRODUCT INFORMATION

NCOR2/SMRT (human recombinant)

Item No. 11633

Overview and Properties

Synonyms:	CTG Repeat Protein 26, Nuclear Receptor Corepressor 2, Silencing Mediator of Retinoic Acid and Thyroid Hormone Receptor, SMAP 270, SMRT, T3 Receptor-Associating Factor, Thyroid-Retinoic-Acid-Receptor-Associated Corepressor, TRAC
Source:	50 µg of recombinant N-terminal GST-tagged protein expressed in *E. coli*
Amino Acids:	395-489 (partial protein)
Uniprot No.:	Q9Y618
Molecular Weight:	39 kDa
Storage:	-80°C (as supplied)
Stability:	≥6 months
Purity:	***batch specific***
Supplied in:	50 mM Tris-HCl, pH 8.0, with 138 mM sodium chloride, 20 mM glutathione, and 10% glycerol
Protein Concentration:	***batch specific*** mg/ml

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

Description

Nuclear receptor corepressor 2 (NCOR2) is a transcriptional corepressor that plays an essential role in the regulation of development and metabolism. Histone deacetylases (HDACs) can mediate nuclear receptor functions by forming co-repressor complexes with nuclear receptors in the absence of ligands. However, HDACs are primarily responsible for catalyzing the deacetylation of core histones. HDAC3, a class I HDAC related to the yeast HDAC Rpd, is inactive alone and requires binding with the deacetylase activation domain (DAD) of NCOR2 for activation.[1] The deacetylation of core histones by HDACs results in the tightening of nucleosomal integrity, restriction of access to transcription factors, and the suppression of gene transcription. In addition, HDACs mediate other transcription regulatory pathways by associating with transcription factors, such as E2F, TFIIE, TFIIF, NF-κB, p300, Stat3, p53, and the retinoblastoma (Rb) protein.[2]

The modification of chromatin structure and other non-histone proteins by HDACs serves to control many complex biological events, including cell development, differentiation, programmed cell death, angiogenesis, and inflammation. Thus, dysregulation of HDACs, leading to an imbalance of acetylation and deacetylation, may be involved in the pathogenesis of various diseases, including cancer and inflammatory diseases.[1]

References

1. Huang, L. Targeting histone deacetylases for the treatment of cancer and inflammatory diseases. *J. Cell. Physiol.* **39.1**, 611-616 (2006).
2. Lin, H.-Y., Chen, C.-S., Lin, S.-P., *et al*. Targeting histone deacetylase in cancer therapy. *Med. Res. Rev.* **26(4)**, 397-413 (2006).

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