PRODUCT INFORMATION

IDH1 (R132H mutant; human, recombinant)

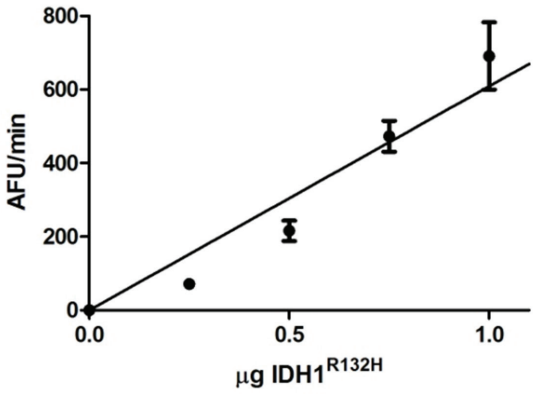
Item No. 14132

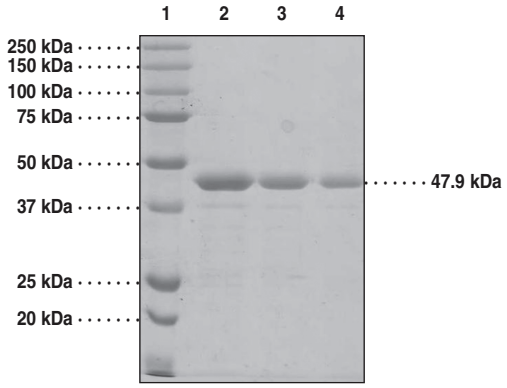
Overview and Properties

Synonym:	Isocitrate Dehydrogenase (NADP) Cytoplasmic
Source:	Active recombinant C-terminal histidine-tagged protein expressed in *E. coli*
Amino Acids:	2-414 (full-length)
Uniprot No.:	O75874
Molecular Weight:	47.9 kDa
Storage:	-80°C (as supplied)
Stability:	≥6 months
Purity:	***batch specific*** (≥95% estimated by SDS-PAGE)
Supplied in:	50 mM Tris-HCl, pH 7.5, with 200 mM sodium chloride, 5 mM β-mercaptoethanol, and 10% glycerol
Protein Concentration:	***batch specific*** mg/ml
Activity:	***batch specific*** U/ml
Specific Activity:	***batch specific*** U/mg
Unit Definition:	One unit is defined as the amount of enzyme required to convert 1 nmol of NADPH to $NADP^+$, using 15 mM α-ketoglutarate as a substrate, per minute at room temperature in 25 mM Tris-HCl, pH 7.5, 150 mM sodium chloride, and 5 mM $MgCl_2$.

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

Images





Lane 1: MW Markers
Lane 2: IDH1 R123H (5 µg)
Lane 3: IDH1 R123H (2.5 µg)
Lane 4: IDH1 R123H (1.25 µg)

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

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PRODUCT INFORMATION

Description

Isocitrate dehydrogenases (IDHs) are nicotinamide adenine dinucleotide (NAD^+) and NAD phosphate ($NADP^+$)-dependent enzymes that catalyze the third step of the tricarboxylic acid cycle. IDHs catalyze oxidative decarboxylation of isocitrate producing α-ketoglutarate (α-KG) and carbon dioxide. IDH1 (cytosolic) and IDH2 (mitochondrial) are $NADP^+$-dependent enzymes that catalyze reversible reactions. The IDH3 isoform, a NAD^+-dependent multisubunit enzyme, is irreversible and allosterically regulated by a variety of positive (calcium, ADP, and citrate) and negative (adenosine triphosphate, NADH, and NADPH) effectors.[1] IDH1 and IDH2 are mutated in >70% of lower grade gliomas.[2] The most common IDH mutation, Arg132His, imparts new gain of function catalytic activity leading to the NADPH-dependent conversion of α-KG to 2-hydroxyglutarate.[3,4] Astrocytes expressing IDH1 R132H mutant have been shown to produce markedly increased levels of the R-2-hydroxyglutarate enantiomer, leading to transformation of cells through the hypoxia-inducible factor prolyl 4-hydroxylase, EGLN.[5]

References

1. Raimundo, N., Baysal, B.E., and Shadel, G.S. Revisiting the TCA cycle: Signaling to tumor formation. *Trends Mol. Med.* **17(11)**, 641-649 (2011).
2. Turcan, S., Rohle, D., Goenka, A., *et al.* IDH1 mutation is sufficient to establish the glioma hypermethylator phenotype. *Nature* **483(7390)**, 479-483 (2012).
3. Reitman, Z.J. and Yan, H. Isocitrate dehydrogenase 1 and 2 mutations in cancer: Alterations at a crossroads of cellular metabolism. *J. Natl. Cancer Inst.* **102(13)**, 932-941 (2010).
4. Dang, L., White, D.W., Gross, S., *et al.* Cancer-associated IDH1 mutations produce 2-hydroxyglutarate. *Nature* **462(7274)**, (2009).
5. Koivunen, P., Lee, S., Duncan, C.G., *et al.* Transformation by the (R)-enantiomer of 2-hydroxyglutarate linked to EGLN activation. *Nature* 1-7 (2012).

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