

HPPD (F-5): sc-271672

BACKGROUND

HPPD (4-hydroxyphenylpyruvate dioxygenase), also known as PPD, GLOD3 or HPD, is a 393 amino acid protein that belongs to the 4HPPD family and is involved in amino acid degradation. Existing as a homodimer, HPPD uses zinc as a cofactor to catalyze the third step in the conversion of L-phenylalanine to fumarate and acetoacetic acid. Defects in the gene encoding HPPD are the cause of tyrosinemia type 3 (TYRO3) and hawkinsinuria (HAWK), both of which are inborn errors of metabolism that are associated with a variety of symptoms, including mental retardation and seizures (associated with TYRO3) and hair and urine abnormalities (associated with HAWK). The gene encoding HPPD maps to human chromosome 12, which encodes over 1,100 genes and comprises approximately 4.5% of the human genome.

REFERENCES

- Rüetschi, U., et al. 1993. Human 4-hydroxyphenylpyruvate dioxygenase. Primary structure and chromosomal localization of the gene. *Eur. J. Biochem.* 213: 1081-1089.
- Awata, H., et al. 1994. Structure of the human 4-hydroxyphenylpyruvic acid dioxygenase gene (HPD). *Genomics* 23: 534-539.
- Stenman, G., et al. 1995. Regional assignment of the human 4-hydroxyphenylpyruvate dioxygenase gene (HPD) to 12q24→qter by fluorescence *in situ* hybridization. *Cytogenet. Cell Genet.* 71: 374-376.
- Rüetschi, U., et al. 1997. Human 4-hydroxyphenylpyruvate dioxygenase gene (HPD). *Genomics* 44: 292-299.
- Rüetschi, U., et al. 2000. Mutations in the 4-hydroxyphenylpyruvate dioxygenase gene (HPD) in patients with tyrosinemia type III. *Hum. Genet.* 106: 654-662.
- Tomoeda, K., et al. 2000. Mutations in the 4-hydroxyphenylpyruvic acid dioxygenase gene are responsible for tyrosinemia type III and hawkinsinuria. *Mol. Genet. Metab.* 71: 506-510.
- Item, C.B., et al. 2007. Manifestation of hawkinsinuria in a patient compound heterozygous for hawkinsinuria and tyrosinemia III. *Mol. Genet. Metab.* 91: 379-383.

CHROMOSOMAL LOCATION

Genetic locus: HPD (human) mapping to 12q24.31; Hpd (mouse) mapping to 5 F.

SOURCE

HPPD (F-5) is a mouse monoclonal antibody raised against amino acids 1-300 mapping at the N-terminus of HPPD of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

HPPD (F-5) is recommended for detection of HPPD of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for HPPD siRNA (h): sc-75297, HPPD siRNA (m): sc-75298, HPPD shRNA Plasmid (h): sc-75297-SH, HPPD shRNA Plasmid (m): sc-75298-SH, HPPD shRNA (h) Lentiviral Particles: sc-75297-V and HPPD shRNA (m) Lentiviral Particles: sc-75298-V.

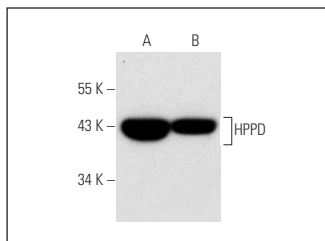
Molecular Weight of HPPD: 45 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200 or Hep G2 cell lysate: sc-2227.

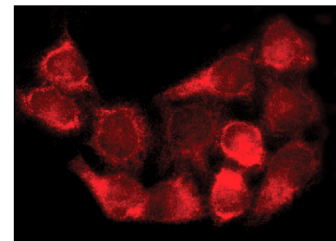
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



HPPD (F-5): sc-271672. Western blot analysis of HPPD expression in HeLa (A) and Hep G2 (B) whole cell lysates.



HPPD (F-5): sc-271672. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

- Pankowicz, F.P., et al. 2016. Reprogramming metabolic pathways *in vivo* with CRISPR/Cas9 genome editing to treat hereditary tyrosinaemia. *Nat. Commun.* 7: 12642.
- Pankowicz, F.P., et al. 2018. Rapid disruption of genes specifically in livers of mice using multiplex CRISPR/Cas9 editing. *Gastroenterology* 155: 1967-1970.e6.

RESEARCH USE

For research use only, not for use in diagnostic procedures.