



Ferrochelatase (h): 293T Lysate: sc-115804

BACKGROUND

Ferrochelatase, also designated heme synthetase or protoheme ferrolyase, is the terminal enzyme of protoheme biosynthesis that catalyzes the ferrous form of iron insertion into protoporphyrin IX. Mature Ferrochelatase is a homodimeric, mitochondrial membrane-associated protein translated downstream of an N-terminal 54 amino acid transit peptide. Ferrochelatase contains two nitric oxide (NO)-sensitive clusters and coordinated 2FE-2S clusters which may potentially serve as a nitric oxide sensor. Defects in the gene encoding the Ferrochelatase enzyme, FECH, cause erythropoietic protoporphyria (EPP), which is a dominantly inherited disease of porphyrin metabolism characterized by photosensitivity and hepatobiliary disease.

REFERENCES

1. Davies, R., et al. 2005. Hepatic gene expression in protoporphyric Fech mice is associated with cholestatic injury but not a marked depletion of the heme regulatory pool. *Am. J. Pathol.* 166: 1041-1053.
2. Di Pierro, E., et al. 2005. A point mutation affecting an Sp1 binding site in the promoter of the Ferrochelatase gene impairs gene transcription and causes erythropoietic protoporphyria. *Exp. Hematol.* 33: 584-591.
3. Elder, G., et al. 2005. Normal dermal Ferrochelatase activity does not protect human skin from protoporphyrin-induced photosensitivity. *J. Invest. Dermatol.* 125: 580.
4. Franco, R., et al. 2005. Porphyrin-substrate binding to murine Ferrochelatase: effect on the thermal stability of the enzyme. *Biochem. J.* 386: 599-605.
5. Najahi-Missaoui, W., et al. 2005. Production and characterization of erythropoietic protoporphyric heterodimeric Ferrochelates. *Blood* 106: 1098-1104.
6. Goodwin, R.G., et al. 2005. Photosensitivity and acute liver injury in myeloproliferative disorder secondary to late-onset protoporphyria caused by deletion of a Ferrochelatase gene in hematopoietic cells. *Blood* 107: 60-62.
7. Ohgari, Y., et al. 2005. Ferrochelatase consisting of wildtype and mutated subunits from patients with a dominant-inherited disease, erythropoietic protoporphyria, is an active but unstable dimer. *Hum. Mol. Genet.* 14: 327-334.
8. Shipovskov, S., et al. 2005. Metallation of the transition-state inhibitor N-methyl mesoporphyrin by Ferrochelatase: implications for the catalytic reaction mechanism. *J. Mol. Biol.* 352: 1081-1090.
9. Sobotka, R., et al. 2005. Photosystem II assembly in CP47 mutant of *Synechocystis* sp. PCC 6803 is dependent on the level of chlorophyll precursors regulated by Ferrochelatase. *J. Biol. Chem.* 280: 31595-31602.

CHROMOSOMAL LOCATION

Genetic locus: FECH (human) mapping to 18q21.31.

PRODUCT

Ferrochelatase (h): 293T Lysate represents a lysate of human Ferrochelatase transfected 293T cells and is provided as 100 µg protein in 200 µl SDS-PAGE buffer.

APPLICATIONS

Ferrochelatase (h): 293T Lysate is suitable as a Western Blotting positive control for human reactive Ferrochelatase antibodies. Recommended use: 10-20 µl per lane.

Control 293T Lysate: sc-117752 is available as a Western Blotting negative control lysate derived from non-transfected 293T cells.

STORAGE

Store at -20° C. Repeated freezing and thawing should be minimized. Sample vial should be boiled once prior to use. Non-hazardous. No MSDS required.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.