SANTA CRUZ BIOTECHNOLOGY, INC.

Trypsin-2 (h): 293T Lysate: sc-114731

BACKGROUND

The human pancreas secretes three different isoforms of the inactive trypsinogen into the small intestine, namely cationic trypsinogen, anionic trypsinogen (the two major isoforms) and mesotrypsinogen (a minor isoform). In the small intestine, each isoform is cleaved by Enterokinase into its active form, Trypsin-1, Trypsin-2 and Trypsin-3, respectively. All Trypsins are members of the serine protease trypsin family. The activated Trypsins go on to activate other protease zymogens and play a role in the autoactivation of trypsinogens, suggesting an important role for Trypsins in digestion. Mutations in the gene encoding Trypsin-1 that stimulate its activity are associated with autosomal dominant hereditary pancreatitis (HCP), also known as chronic pancreatitis (CP), a disease characterized by persistent, severe abdominal pain due to calcifications of the parenchyma, pancreatic stones, cysts and pancreatic head enlargement. Trypsin-3 is expressed in the brain in addition to the pancreas.

REFERENCES

1. Scheele, G., Bartelt, D. and Bieger, W. 1981. Characterization of human exocrine pancreatic proteins by two-dimensional isoelectric focusing/sodium dodecyl sulfate gel electrophoresis. Gastroenterology 80: 461-473.
2. Rinderknecht, H., Renner, I.G., Abramson, S.B. and Carmack, C. 1984. Mesotrypsin: a new inhibitor-resistant protease from a zymogen in human pancreatic tissue and fluid. Gastroenterology 86: 681-692.
3. Kleeff, J., Friess, H., Korc, M. and Büchler, M.W. 2000. Chronic pancreatitis: pathogenesis and molecular aspects. Ann. Ital. Chir. 71: 3-10.
4. Chandak, G.R., Idris, M.M., Reddy, D.N., Mani, K.R., Bhaskar, S., Rao, G.V. and Singh, L. 2004. Absence of PRSS1 mutations and association of SPINK1 Trypsin inhibitor mutations in hereditary and non-hereditary chronic pancreatitis. Gut 53: 723-728.
5. Sahin-Tóth, M., Kukor, Z. and Nemoda, Z. 2006. Human cationic trypsinogen is sulfated on Tyr 154. FEBS J. 273: 5044-5050.
6. Nemoda, Z. and Sahin-Tóth, M. 2006. Chymotrypsin C (caldecrin) stimulates autoactivation of human cationic trypsinogen. J. Biol. Chem. 281: 11879-11886.
7. Keim, V. 2008. Role of genetic disorders in acute recurrent pancreatitis. World J. Gastroenterol. 14: 1011-1015.
8. Teich, N. and Mössner, J. 2008. Hereditary chronic pancreatitis. Best Pract. Res. Clin. Gastroenterol. 22: 115-130.
9. Chang, Y.T., Chang, M.C., Su, T.C., Liang, P.C., Su, Y.N., Kuo, C.H., Wei, S.C. and Wong, J.M. 2008. Association of cystic fibrosis transmembrane conductance regulator (CFTR) mutation/variant/haplotype and tumor necrosis factor (TNF) promoter polymorphism in hyperlipidemic pancreatitis. Clin. Chem. 54: 131-138.

CHROMOSOMAL LOCATION

Genetic locus: PRSS2 (human) mapping to 7p22.3.

RESEARCH USE

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

PRODUCT

Trypsin-2 (h): 293T Lysate represents a lysate of human Trypsin-2 transfected 293T cells and is provided as 100 µg protein in 200 µl SDS-PAGE buffer.

APPLICATIONS

Trypsin-2 (h): 293T Lysate is suitable as a Western Blotting positive control for human reactive Trypsin-2 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.

PROTOCOLS

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