Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag

Catalog Number: 11773-PL

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human Pancreatic Lipase protein Lys17-Cys465, with N-terminal HA (YPYDVPDYA) and 6-His tags Accession # P16233.1
N-terminal Sequence Analysis	Tyr
Predicted Molecular Mass	52 kDa

SPECIFICATIONS

SDS-PAGE	50-55 kDa, under reducing conditions.
Activity	Measured by its ability to cleave a fluorogenic substrate, 4-Methylumbelliferyl oleate (4-MUO). The specific activity is >750 pmol/min/µg, as measured under the described conditions.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Supplied as a 0.2 µm filtered solution in Tris and NaCl. See Certificate of Analysis for details.

Activity Assay Protocol

Materials

- Assay Buffer: 50 mM Tris, 200 mM NaCl, 5 mM $CaCl_2$, pH 7.5
- Recombinant Human Pancreatic Lipase/PNLIP (rhPNLIP) (Catalog # 11773-PL)
- Substrate: 4-Methylumbelliferyl Oleate, 100 mM stock in DMSO
- Black 96-well Plate
- Plate Reader with Fluorescence Read Capability

Assay

1. Dilute rhPNLIP to 0.5 µg/mL in Assay Buffer.
2. Dilute Substrate to 0.5 mM in Assay Buffer.
3. Load into a plate 50 µL of 0.5 µg/mL rhPNLIP and start the reaction by adding 50 µL of 0.5 mM Substrate. Include a Substrate Blank containing 50 µL of Assay Buffer and 50 µL of 0.5 mM Substrate.
4. Read at excitation and emission wavelengths of 365 nm and 445 nm (top read), respectively, in kinetic mode for 5 minutes.
5. Calculate specific activity:

$$\text{Specific Activity (pmol/min/µg)} = \frac{\text{Adjusted } V_{max}\text{* (RFU/min) x Conversion Factor** (pmol/RFU)}}{\text{amount of enzyme (µg)}}$$

*Adjusted for Substrate Blank
**Derived using a fluorescent standard 4-Methylumbelliferone

Final Assay Conditions

Per Well:

- rhPNLIP: 0.025 µg
- Substrate: 250 µM

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 6 months from date of receipt, -20 to -70 °C as supplied. • 3 months, -20 to -70 °C under sterile conditions after opening.

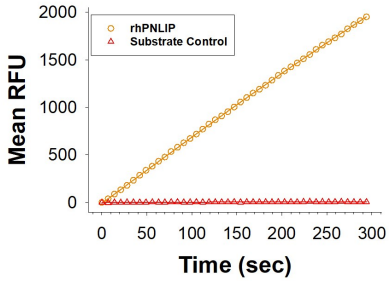
DATA

Bio-Techne®
Global | bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL: 1.612.379.2956
USA | TEL: 800.343.7475 Canada | TEL: 855.668.8722 Europe | Middle East | Africa TEL: +44.0.1235.529449
China | info.cn@bio-techne.com TEL: 400.821.3475

Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag

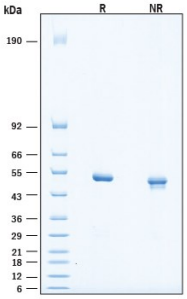
Catalog Number: 11773-PL

Enzyme Activity



Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag Enzyme Activity. Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag (Catalog # 11773-PL) is measured by its ability to cleave a fluorogenic substrate, 4-Methylumbelliferyl oleate (4-MUO).

SDS-PAGE



Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag SDS-PAGE. 2 µg/lane of Recombinant Human Pancreatic Lipase/PNLIP HA-tag His-tag (Catalog # 11773-PL) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 50-55 kDa, under reducing conditions.

BACKGROUND

Recombinant human pancreatic triacylglycerol lipase or pancreatic lipase (PL), from the gene PNLIP, is a 449 residue mature, glycosylated, secreted serine hydrolase of a family of mammalian lipases. Mature PL/PNLIP contains a globular N-terminal core domain typical of a/b hydrolase-fold family members and a C-terminal domain that is connected via an amino acid extension and seven disulfide bonds (1, 2). The C-terminal domain binds the required cofactor procolipase via a salt bridge but does not alter the conformation of the core PL/PNLIP domain (2). The N-terminal core domain contains the catalytic triad active site and a lid domain that inhibits binding of substrate and requires conformational change for activity through adsorption at the interface of lipid micelles (2). PL/PNLIP is secreted from the pancreas and hydrolyzes dietary fat triacylglycerides at the two carbon site at high preference over cholesterol esters, phospholipids, and galactolipids (3) playing a key role in absorption of dietary fat in the small intestine (2). As PL/PNLIP is the primary enzyme responsible for the hydrolysis of the majority of dietary fats in the proximal small intestine (4), inhibition has been identified as an important target for the prevention and treatment of obesity-related disorders such as diabetes, dyslipidemia, nonalcoholic fatty liver disease and cardiovascular disease (4-7). With a few available PL/PNLIP inhibitors in use in the clinic to manage obesity and its related disorders, significant investigation and development of additional or alternative inhibitors for PL/PNLIP with fewer side effects is underway (2, 9, 10). Pancreatic enzyme replacement therapies that include PL/PNLIP have shown high therapeutic value for patients with exocrine pancreatic insufficiency (EPI), a deficiency of the pancreatic enzymes resulting in maldigestion and malabsorption caused by many diseases such as pancreatic adenocarcinoma and cystic fibrosis or by a rare disorder known as congenital pancreatic triglyceride lipase (9, 11, 12). Finally, oral lipid-based delivery systems are prone to digestion by pancreatic lipase in the small intestine which can impact the integrity of these delivery systems and result in premature drug release into the gastrointestinal environment and exposure of the drug to alteration by proteases in the small intestine (13); development of strategies including inhibition of PL/PNLIP to control potential impacts to oral lipid-based drug delivery systems is under investigation (13).

References:

1. Winkler, F.K. *et. al.* (1990) Nature **343**:771.
2. Kumar, A. and S. Chauhan (2021) Life Sci. **271**:119115.
3. Lim, S.Y. *et. al.* (2022) Am. J. Vet Res. **83**:1.
4. Yadav, N. and A.T. Paul (2024) Drug Discov. Today **29**:103855.
5. Chatzigeorgiou, A. *et. al.* (2014) Hepatology **60**:1196.
6. Higgins, V. *et. al.* (2020) J. Clin. Endocrinol. Metab. **105**:1228.
7. Lee, S.S. and S. Kang (2015) J. Phys. Ther. Sci. **27**:1903.
8. Lopez-Jimenez, F. *et. al.* (2022) Eur. J. Prev. Cardiol. **29**:2218.
9. Subramaniyan, V. and Y.U. Hanim (2025) Int. J. Obes. **49**:492.
10. Uuh Narvaez, J.J. *et. al.* (2025) Molecules **30**:2392.
11. Szabo, A. *et. al.* (2015) Biochim. Biophys. Acta **1852**:1372.
12. Brennan, G.T. and M.W. Saif (2019) JOP **20**:121.
13. Zupancic, O. *et. al.* (2023) J. Control Release **362**:381.

Bio-Techne®
Global | bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL: 1.612.379.2956
USA | TEL: 800.343.7475 Canada | TEL: 855.668.8722 Europe | Middle East | Africa TEL: +44.0.1235.529449
China | info.cn@bio-techne.com TEL: 400.821.3475