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# ILK1 Human

**Description:**ILK1 Human Recombinant produced in E.Coli is single, a non-glycosylated, Polypeptide chain containing 452 amino acids fragment (1-452) having a molecular mass of 55.92kDa and fused with a 4.5kDa amino-terminal hexahistidine tag. The ILK1 is purified by proprietary chromatographic techniques.

**Synonyms:**Integrin-linked protein kinase, ILK-1, ILK-2, 59 kDa serine/threonine-protein kinase, p59ILK, ILK, ILK1, ILK2, DKFZp686F1765, P59.

Source: Escherichia Coli.

Physical Appearance: Sterile Filtered clear solution.

Purity: Greater than 95.0% as determined by SDS-PAGE.

## Formulation:

ILK1 protein is supplied in 25mM Sodium Acetate (pH 4.8) and 50% glycerol.

#### Stability:

Store at 4°C if entire vial will be used within 2-4 weeks. Store, frozen at -20°C for longer periods of time. Please avoid freeze thaw cycles.

#### Usage:

NeoBiolab's products are furnished for LABORATORY RESEARCH USE ONLY. The product may not be used as drµgs, agricultural or pesticidal products, food additives or household chemicals.

### Introduction:

ILK1 (Integrin-linked kinase) is a serine/threonine protein kinase, containing 4 ankyrin-like repeats. ILK1 regulates a number of biological properties which include: anchorage-independent cell cycle progression, tumor cell invasion and apoptosis. ILK1 can also be implicated in mediating cell architecture, adhesion to integrin substrates and anchorage-dependent growth in epithelial cells. Furthermore, ILK1 phosphorylates beta-1 and beta-3 integrin subunit on serine and threonine residues, but also AKT1 and GSK3B.ILK1 interacts with the cytoplasmic domains of integrin 1 and 3 subunits in addition to several adaptors and signaling proteins, it also acts as a proximal receptor kinase regulating integrin-mediated signal transduction. ILK1 is a focal adhesion protein part of the complex ILK-PINCH. This complex is deemed to be one of the convergence points of integrin- and growth factor-signaling pathway. ILK1 is stimulated rapidly but briefly by both cell fibronectin interactions, as well as by insulin, in a PI3-K-dependent manner, probably through the binding of PtdIns(3,4,5)P3 with a PH-like domain of ILK.ILK1 over-expression has been documented in a wide variety of human malignancies.

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