Recombinant Human LAP (TGF-beta 1)

货号: RB91EPT048

gui_ge

10ug

biao_da_su_zhu

Human Cells

biao_qian

TGF-beta 1

fen_zi_liang

28.5 KDa

biao_guan_fen_zi_liang

20-30 KDa, reducing conditions

xing_tai

Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.

chun_du

Greater than 95% as determined by reducing SDS-PAGE.

nei_du_su_shui_ping

Less than 0.1 ng/μg (1 EU/μg) as determined by LAL test.

rong_jie_fang_fa

Always centrifuge tubes before opening.Do not mix by vortex or pipetting.
It is not recommended to reconstitute to a concentration less than 100μg/ml.
Dissolve the lyophilized protein in distilled water.
Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

chu_cun_fang_fa

Lyophilized protein should be stored at < -20° C, though stable at room temperature for 3 weeks.
Reconstituted protein solution can be stored at 4-7° C for 2-7 days.
Aliquots of reconstituted samples are stable at < -20° C for 3 months.

yun_shu_tiao_jian

The product is shipped at ambient temperature.
Upon receipt, store it immediately at the temperature listed below.

bei_jing_shuo_ming

Transforming Growth Factor β-1 (TGFβ-1) is a secreted protein which belongs to the TGF-β family. TGFβ-1 is abundantly expressed in bone, articular cartilage and chondrocytes and is increased in osteoarthritis (OA). TGFβ-1 performs many cellular functions, including the control of cell growth, cell proliferation, cell differentiation and apoptosis. The precursor is cleaved into a latency-associated peptide (LAP) and a mature TGFβ-1 peptide.Disulfide-linked homodimers of LAP and TGF-beta 1 remain non-covalently associated after secretion, forming the small latent TGF-beta 1 complex. Purified LAP is also capable of associating with active TGF-beta with high affinity, and can neutralize TGF-beta activity. Covalent linkage of LAP to one of three latent TGF-beta binding proteins (LTBPs) creates a large latent complex that may interact with the extracellular matrix. TGF-beta activation from latency is controlled both spatially and temporally, by multiple pathways that include actions of proteases such as plasmin and MMP9, and/or by thrombospondin 1 or selected integrins. Although different isoforms of TGF-beta are naturally associated with their own distinct LAPs, the TGF-beta 1 LAP is capable of complexing with, and inactivating, all other human TGF-beta isoforms and those of most other species. Mutations within the LAP are associated with Camurati-Engelmann disease, a rare sclerosing bone dysplasia characterized by inappropriate presence of active TGF-beta 1.

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