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D4GDI Human

Description:D4-GDI Human Recombinant full length protein expressed in E.coli, shows a 54 kDa SDS-PAGE.The D4-GDI is purified by proprietary chromatographic techniques.

Catalog #:PRPS-303

Synonyms:D4-GDI, D4GDI.

For research use only.

Source: Escherichia Coli.

Physical Appearance: Sterile Filtered clear solution.

Purity: Greater than 90% as determined by SDS-PAGE.

Formulation:

D4-GDI protein at 0.1mg/ml in 50mM Tris-HCl, pH7.5 and 10mM L-Glutathione (reduced).

Stability:

Store vial at -20°C to -80°C. When stored at the recommended temperature, this protein is stable for 12 months.Please prevent 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.

Applications:

ELISA Inhibition Assays Western Blotting.

Introduction:

D4-GDI belongs to the family of Rho-GDIs or GDP-dissociation inhibitors for the Rho/Rac family of small G proteins; consisting of RhoGDI-1, D4 GDI/RhoGDI-2 and RhoGDI-3. It inhibits the release of GDP from the Rho proteins, essential for GTP-loading and activation of GTPase activity. D4-GDI is located in the cytoplasm and is involved in the regulation of the membrane association and dissociation cycle of Rho/Rac proteins. It is known to be a substrate for caspase 3 during Fas-mediated apoptosis.D4-GDI is expressed only in hematopoietic tissues, mainly in B and T cells, unlike RhoGDI which is expressed ubiquitously. D4-GDI plays a role in the regulation of lymphocyte activation, survival, and responsiveness. Expression of D4-GDI is also observed in cells of nonhematopoietic neoplasms, including ovarian and bladder cancer cells, sµggestive of D4-GDIs role in progression of human tumors. D4-GDI is expressed in human breast cancer cells. In different human breast cancer cell lines that were studied, cell lines with high invasive activities, for example MDA-MB-231, expressed higher D4-GDI than did weakly invasive and noninvasive cell lines. Intentional disruption of D4-GDI by steadily expressing small interfering RNA (siRNA) against D4-GDI transcripts successfully blocks the motility and invasive potential of MDA-MB-231 cells in vitro. Cells deficient of D4-GDI revert to a normal breast epithelial phenotype characterized by the formation of cysts with a central lumen when grown on Matrigel. D4-GDI reduction is linked to a down-regulation of the cell surface protein 1-integrin and a loss of cell-matrix adhesion, sµggestive of D4-GDI induction of cell invasion by controlling 1-integrin expression. The expression of 1-integrin and invasive activities are restored once theres restitution of D4-GDI expression to the levels found in the parental MDA-MB-231 cells. Therefore it is sµggested that D4-GDI may play a significant role in the regulation of breast cancer cell invasiveness.

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