
Product Data Sheet

Product Name: Fas C- Terminal Tripeptide

Cat. No.: GP10123

Chemical Properties

Cas. No. 189109-90-8

SMILES CC(N[C@@H](CO)C(N[C@@H](CC(C)C)C(N[C@@H](C(C)C)C(O)=O)=O)=OFormula C₁₆H₂₉N₃O₆ M.Wt 359.42

Solubility ≥ 35.9mg/mL in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure **Background**

Fas C- Terminal Tripeptide, (C₁₆H₂₉N₃O₆), a tri-peptide with the sequence AC-SER-LEU-VAL-OH, it's the C-terminal tripeptide of Fas, MW= 359.4. Fas (APO-1/CD95) is a cell surface receptor, which is a member of the tumor necrosis factor receptor (TNFR) superfamily, The Fas receptor is a death receptor on the surface of cells that leads to programmed cell death (apoptosis). It is one of two apoptosis pathways. Fas forms the death-inducing signaling complex (DISC) upon ligand binding. Upon ensuing death domain (DD) aggregation, the receptor complex is internalized via the cellular endosomal machinery. This allows the adaptor molecule FADD to bind the death domain of Fas through its own death domain. FADD also contains a death effector domain (DED) near its amino terminus, which facilitates binding to the DED of FADD-like interleukin-1 beta-converting enzyme (FLICE), more commonly referred to as caspase-8. FLICE can then self-activate through proteolytic cleavage into p10 and p18 subunits, two each of which form the active heterotetramer enzyme. Active caspase-8 is then released from the DISC into the cytosol, where it cleaves other effector caspases, eventually leading to DNA degradation, membrane blebbing, and other hallmarks of apoptosis.

Caution: Product has not been fully validated for medical applications. For research use only.

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The C-terminal tripeptide (AC-SER-LEU-VAL-OH) of Fas was necessary and sufficient both for binding to the third PDZ domain of FAP-1 and for inhibiting Fas/FAP-1 binding.

References:

1. Lichter P, Walczak H, Weitz S, Behrmann I, Krammer PH (September 1992). "The human APO-1 (APT) antigen maps to 10q23, a region that is syntenic with mouse chromosome 19". *Genomics* 14 (1): 179-80.
2. Inazawa J, Itoh N, Abe T, Nagata S (November 1992). "Assignment of the human Fas antigen gene (Fas) to 10q24.1". *Genomics* 14 (3): 821-2.
3. Huang B, et al. (1996). "NMR structure and mutagenesis of the Fas (APO-1/CD95) death domain". *Nature* 384 (6610): 638-41.
4. Eberstadt M, et al. (1998). "NMR structure and mutagenesis of the FADD (Mort1) death-effector domain". *Nature* 392 (6679): 941-5.

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