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InnoCyto Inc.

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Anti-Human CD95 (Fas) Antibody

Catalog Number:	109501, 109502
Size:	100 ug, 500 ug
Target Name:	CD95, Fas, TNFRSF6, Apo-1
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	DX2
Application:	Flow Cytometry
Reactivity:	Human
Format:	Purified
Isotype:	Mouse IgG1
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
Protein Concentration:	0.5 mg/mL
Storage and Handling:	The antibody solution should be stored between 2°C and 8°C
Recommended Usage:	For flow cytometric staining, it is recommended to use less than 0.2 µg of this reagent per 0.5-1.0 million cells in a 100 µL volume. Optimal reagent performance should be determined by titration for each specific application
Isotype Control:	301401

BACKGROUND INFORMATION

CD95, also known as Fas or APO-1, is a transmembrane receptor that plays a central role in the regulation of programmed cell death (apoptosis) and immune system homeostasis. It belongs to the tumor necrosis factor receptor (TNFR) superfamily and acts as a key mediator of the extrinsic apoptotic pathway. CD95 ensures the removal of damaged, infected, or excess cells, thereby maintaining immune equilibrium and preventing uncontrolled proliferation.

Structurally, CD95 is a type I transmembrane protein of approximately 319 amino acids, consisting of an extracellular domain responsible for ligand binding, a single hydrophobic transmembrane region, and an intracellular “death domain” that initiates apoptosis signaling. The extracellular domain contains multiple cysteine-rich repeats similar to other TNFR family members, which facilitate the trimerization necessary for receptor activation. Upon engagement with its ligand, Fas ligand (FasL or CD95L), which is primarily expressed on activated T cells and natural killer cells, CD95 undergoes oligomerization, leading to the recruitment of adaptor molecules such as FADD (Fas-associated death domain protein). Formation of the Fas-FADD complex triggers the assembly of the death-inducing signaling complex (DISC), which recruits and activates caspase-8, initiating the caspase cascade that culminates in apoptosis. This mechanism is critical for the elimination of self-reactive lymphocytes during immune tolerance and for the contraction phase following immune responses.

Aberrations in CD95 signaling have profound implications in disease. Mutations or downregulation of CD95 or FasL can cause autoimmune lymphoproliferative syndrome (ALPS), characterized by defective lymphocyte apoptosis and chronic immune activation. Conversely, excessive CD95 activation contributes to tissue damage in conditions such as hepatitis, systemic lupus erythematosus, and neurodegenerative diseases. In cancer, many tumor cells develop resistance to Fas-mediated apoptosis through receptor mutations or decoy mechanisms, allowing immune evasion. Elevated expression of soluble Fas also acts as a protective mechanism for malignant cells.

Therapeutically, targeting CD95 pathways holds promise in both oncology and immunology. Strategies to reactivate Fas-mediated apoptosis in tumors are being investigated as potential cancer treatments, while inhibitors of CD95 or FasL are explored to prevent excessive cell death in autoimmune and inflammatory conditions. Modulating CD95 signaling, therefore, represents a finely balanced therapeutic frontier with applications ranging from immune regulation to cancer therapy.

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