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Anti-Mouse CD134 (OX-40) Antibody

Catalog Number:	207901, 207902
Size:	25 µg, 100 µg
Target Name:	CD134, OX-40, OX40, TNFRSF4, ACT35
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	OX-86
Application:	Flow Cytometry
Reactivity:	Mouse
Format:	Purified
Isotype:	Rat IgG1
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
Protein Concentration:	0.5 mg/mL
Storage & 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:	303401

BACKGROUND INFORMATION

Mouse CD134, also known as OX40 or TNFRSF4, is a member of the tumor necrosis factor receptor (TNFR) superfamily and an important costimulatory receptor on T cells. OX40 is induced following T-cell receptor activation and is expressed predominantly on activated CD4+ and CD8+ T cells. In mice, OX40 is also constitutively expressed on certain regulatory T cells (Tregs). OX40 signaling promotes T-cell proliferation, survival, differentiation, cytokine production, and formation of memory T cells, thereby strengthening and sustaining antigen-specific immune responses.

Mouse OX40 is a type I transmembrane glycoprotein belonging to the TNFR superfamily. It contains an extracellular region with characteristic cysteine-rich domains, a single transmembrane segment, and a cytoplasmic signaling tail. The principal and currently established ligand is OX40 ligand (OX40L), also known as CD252 or TNFSF4. OX40L is a type II transmembrane TNF-superfamily protein expressed primarily by activated antigen-presenting cells, including dendritic cells, macrophages, and B cells. OX40L forms a trimer and engages OX40 to initiate downstream signaling through pathways including NF-κB and PI3K/Akt.

Excessive OX40 signaling can contribute to chronic inflammation and autoimmune disease. In mouse models, the OX40–OX40L pathway has been implicated in experimental autoimmune encephalomyelitis, asthma, colitis, diabetes, arthritis, graft-versus-host disease, and transplant rejection. Blocking OX40 or OX40L can substantially reduce disease severity in several of these models,

supporting the pathway as a potential therapeutic target.

Therapeutically, OX40 can be targeted in two opposing ways. Agonistic anti-OX40 antibodies or OX40L-based agonists can enhance T-cell activation and antitumor immunity, making OX40 an attractive target for cancer immunotherapy and vaccine development. Conversely, antagonistic antibodies or OX40L blockade may suppress pathogenic T-cell responses in autoimmune and inflammatory diseases. OX40 agonists, including combinations with immune-checkpoint inhibitors, continue to be investigated clinically, although antitumor efficacy has so far been variable.

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