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APC Anti-mouse Tim-4 Antibody

Catalog Number:	208311, 208312
Size:	25 µg, 100 µg
Target Name:	Tim-4, Tim4, T cell immunoglobulin and mucin domain containing protein-4
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	RMT4-54
Application:	Flow Cytometry
Reactivity:	Mouse
Format:	APC
Isotype:	Rat IgG2a
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA
Protein Concentration:	0.2 mg/mL
Storage&Handling:	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.
Recommended Usage:	For flow cytometric staining, it is recommended to use less than 0.5 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. APC has an excitation max at 650 nm and an emission max at 660 nm.
Excitation Laser:	Red Laser (633 nm)
Isotype Controls:	303508
Antibody Family:	Mouse Antibodies
Storage & Handling:	The antibody solution should be stored undiluted between 2°C and 8°C, and protected from prolonged exposure to light. Do not freeze.
Isotype Control:	303508

BACKGROUND INFORMATION

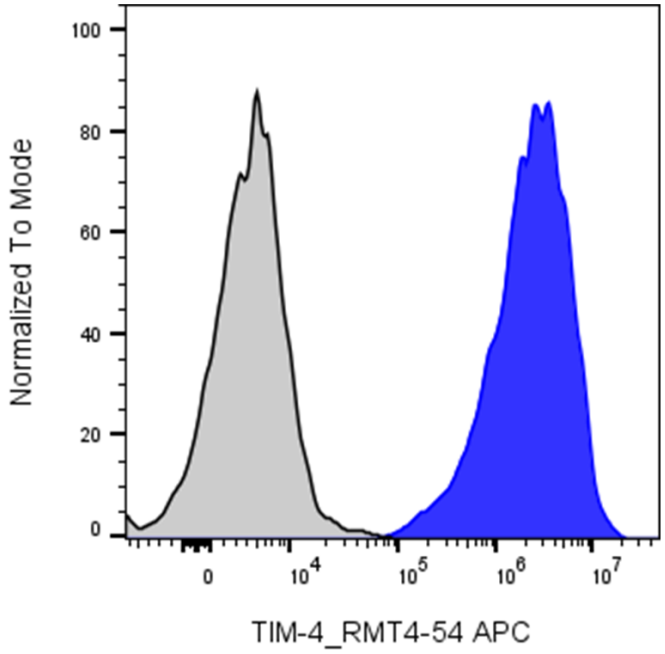
Mouse TIM-4 (T-cell immunoglobulin and mucin domain-containing protein 4) is a cell-surface immunoregulatory receptor encoded by the Havcr4 gene. In sharp contrast to other TIM family members, mouse TIM-4 is entirely absent from mature T cells and is instead expressed exclusively on professional antigen-presenting cells (APCs), including resident peritoneal macrophages, dendritic cells, and specialized macrophage subsets in lymphoid tissues. Its primary physiological function is orchestrating efferocytosis—the recognition, tethering, and engulfment of dying cells—which is essential for maintaining tissue homeostasis and preventing

systemic inflammation.

Structurally, mouse TIM-4 is a type I transmembrane glycoprotein featuring an extracellular N-terminal IgV domain, a heavily O-glycosylated mucin-like stalk, a single transmembrane segment, and a relatively short cytoplasmic tail. Crucially, its cytoplasmic tail completely lacks any known signaling motifs or tyrosine phosphorylation sites, meaning it cannot transmit classical intracellular activation or inhibitory cascades independently. Instead, mouse TIM-4 utilizes a highly conserved Metal Ion-Dependent Ligand-Binding (FG-CC) cleft within its IgV domain to bind directly to phosphatidylserine (PtdSer) exposed on the outer leaflet of apoptotic cell membranes, acting as a high-affinity phagocytic tether that coordinates with co-receptors to trigger engulfment.

Defects in mouse TIM-4 pathways are directly tied to a failure in apoptotic cell clearance, leading to secondary necrosis and the leakage of intracellular autoantigens. In murine models, a lack of functional TIM-4 results in a substantial accumulation of dead cell fragments, triggering inappropriate activation of the innate immune system and driving severe lupus-like systemic autoimmune diseases. In oncology, TIM-4 expression on tumor-associated macrophages (TAMs) can promote an immunosuppressive microenvironment by clearing apoptotic debris without initiating inflammatory signaling. Mouse TIM-4 is an invaluable target in research for studying phagocytosis, checking macrophage polarization states, and developing immunotherapies aimed at modulating autoantigen presentation.

PRODUCT DATA



L5178Y cells transfected with expression plasmid of TIM-4 were stained with APC Anti-Mouse TIM-4 clone RMT4-54 (color-filled histogram) or an isotype control (gray histogram).

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