

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human MMR/CD206 in direct ELISAs and Western blots. In direct ELISAs, approximately 20% cross-reactivity with recombinant mouse MMR is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human MMR/CD206 Leu19-Lys1383 (Thr399Ala) & (Leu407Phe) Accession # P22897
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

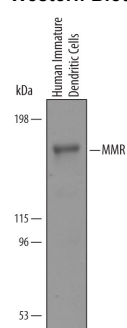
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	1 µg/mL	See Below
Immunocytochemistry	5-15 µg/mL	Immersion fixed human peripheral blood mononuclear cells

DATA

Western Blot



Detection of Human MMR/CD206 by Western Blot. Western blot shows lysates of human immature dendritic cells. PVDF Membrane was probed with 1 µg/mL of Goat Anti-Human MMR/CD206 Antigen Affinity-purified Polyclonal Antibody (Catalog # AF2534) followed by HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF019). A specific band was detected for MMR/CD206 at approximately 185 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 8.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

The human Macrophage Mannose Receptor (MMR), also known as CD206 and MRC1 (mannose receptor C, type 1), is a 190 kDa scavenger receptor that is expressed on tissue macrophages, myeloid dendritic cells, and liver and lymphatic endothelial cells (1). It belongs to a family of receptors sharing similar protein structure that also includes DEC205, phospholipase A2 receptor, and Endo180 (2, 3). The human MMR protein is synthesized as a 1456 amino acid (aa) precursor that contains an 18 aa signal sequence, a 1371 aa extracellular region, a 21 aa transmembrane segment and a 46 aa cytoplasmic domain (4). Its extracellular region is composed of an N-terminal cysteine-rich domain, followed by a single fibronectin type II repeat, and eight C-type lectin carbohydrate recognition domains (CRD) (3, 4). Human to mouse, the extracellular region is 82% aa identical. The cysteine-rich domain mediates recognition of sulfated N-acetylgalactosamine, which occurs on some extracellular matrix proteins and is the terminal sugar of the unusual oligosaccharides present on pituitary hormones such as lutropin and thyrotropin (5). Several of the CRDs participate in the Ca²⁺-dependent recognition of carbohydrates showing a preference for branched sugars with terminal mannose, fucose or N-acetylglucosamine (6). The cytoplasmic domain of MMR includes a tyrosine-based motif for internalization in clathrin-coated vesicles. Once internalized, ligands are released following acidification of phagosomes or endosomes, and the receptor is recycled to the cell surface (3, 7). MMR mediates phagocytosis upon binding to target structures that occur on a variety of pathogenic microorganisms including Gram-negative and Gram-positive bacteria, yeasts, parasites, and mycobacteria. MMR also functions to maintain homeostasis through the endocytosis of potentially harmful glycoproteins associated with inflammation (2, 3).

References:

1. East, L. and C. Isake (2002) *Biochim. Biophys. Acta* **1572**:364.
2. Chieppa, M. *et al.* (2003) *J. Immunol.* **171**:4552.
3. Figdor, C. *et al.* (2002) *Nat. Rev. Immunol.* **2**:77.
4. Taylor, M. *et al.* (1990) *J. Biol. Chem.* **265**:12156.
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6. Martinez-Pomares, L. *et al.* (2001) *Immunobiology* **204**:527.
7. Feinberg, H. *et al.* (2000) *J. Biol. Chem.* **275**:21539.