

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human M-Cadherin/Cadherin-15 in direct ELISAs and Western blots.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human M-Cadherin/Cadherin-15 Val22-Ala606 Accession # P55291
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

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.

CyTOF-ready	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.
Flow Cytometry	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

M-Cadherin (M-CAD or Cadherin-15) is a 124 kDa type I transmembrane glycoprotein of the Cadherin superfamily of calcium-dependent homotypic adhesion molecules (1-4). Like other classical Cadherins, the 814 amino acid (aa) human M-CAD contains a signal sequence (21 aa), a propeptide (29 aa), an extracellular domain with five Cadherin domain repeats (ECD, 556 aa), a transmembrane segment (20 aa) and a cytoplasmic domain (188 aa) (1). A splice variant that diverges within the third Cadherin repeat has been sequenced. The Cadherin repeats are responsible for cell-cell adhesion by homophilic binding on opposing cells (1-4). Intracellularly, M-CAD binds β -catenin or plakoglobin (γ -catenin), which in turn bind α -catenin (4). M-CAD also binds p120 catenin (5). Connection of Cadherin/catenin complexes to the actin cytoskeleton is in question, but is possible through a linker (6). Connection to microtubules has been shown (7). M-CAD is present during early stages of skeletal muscle development and is thought to align myoblasts for fusion (8). It is also present in muscle satellite cells and participates in muscle regeneration (9). M-CAD is also expressed in the granule cell layer of the cerebellar glomerulus (10). Deletion of mouse M-CAD has little effect *in vivo*, most likely due to compensation by N-Cadherin (11). However, M-CAD upregulation and adhesion between myoblasts during induction of differentiation *in vitro* is required for their fusion (8, 12, 13). M-CAD activity is later downregulated by sequestering to caveoli, p120 catenin/RhoA-induced ubiquitination and/or cleavage by calpain-3. This terminates fusion and allows sarcomere formation (5, 12, 13). Human M-CAD ECD shows 88% aa identity with mouse, rat, or bovine and 85% aa identity with canine M-CAD ECD. M-CAD is an outlier among classical Cadherins, with 40% aa identity or less in the ECD (4).

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