

Product Information

MONOCLONAL ANTI-p120^{ctn} (CATENIN-RELATED)

CLONE 6H11

Mouse Culture Supernatant

Product Number **P2732**

Product Description

Monoclonal Anti-p120^{ctn} (Catenin-related) (mouse IgG1 isotype) is derived from the 6H11 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from an A/J mouse immunized with a recombinant N-terminal fragment of murine p120^{ctn}.¹ The isotype is determined using Sigma ImmunoType™ Kit (Sigma ISO-1) and by a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents (Sigma ISO-2).

Monoclonal Anti-p120^{ctn} (Catenin-related) reacts specifically with the N-terminal region of p120^{ctn} and recognizes isoforms 1 and 2, but not isoforms 3 or 4 of the molecule.¹ The epitope recognized by the antibody is in the region between the ATG start sites used by isoforms 2 and 3.¹ The antibody may be used for immunoblotting¹ (120 kDa, and possibly very weak doublet at 200 kDa), ELISA,¹ immunocytochemistry¹ and immunoprecipitation¹. Species cross-reactivity has been observed with human,¹ bovine, dog,¹ rat,¹ mouse¹ and chicken¹ p120^{ctn}.

Cell adhesion is vitally important during development and in the adult organism, being necessary for sorting of cells, induction of cellular morphogenesis and maintenance of tissue integrity.^{2,3} Many cancer cells show aberrant adhesion properties that are likely to contribute to tumorigenesis, invasion, and metastasis. Ca²⁺-dependent cell adhesion is mediated by a multifunctional family of transmembrane glycoproteins termed cadherins.³ Cadherins are concentrated in cell-cell adherens junctions, where cells come into close contact with one another. Cadherins self-associate specifically via their extracellular domains. Studies supporting a role for cadherins in morphogenesis, have led to the hypothesis that cadherins are crucial for segregation and sorting out from one another, of different cells expressing different cadherin types. During recognition and adhesion between cells, cadherins regulate homophilic, Ca²⁺-dependent interactions in cells. This initiates a cascade of events that leads to the structural and functional reorganization of cells, including formation of junctional complexes (tight junction, *zonula adherens*, desmosomes),

organization of the actin cytoskeleton at the apical junctional complex, assembly of the membrane cytoskeleton, and development of membrane domains. The mechanism of cadherin function involves both specific binding of extracellular domains at the cell surface and interactions with components of the cytoplasm. Studies have identified several cytoplasmic proteins, known as catenins, that bind noncovalently to the cytoplasmic domain of cadherins.⁴ Formation of the cadherin/catenin complex is required for cadherin functions in cell-cell adhesion, signal transduction, as well as the initiation and maintenance of structural and functional organization of cells and tissues. Catenins mediate the connection of cadherins to actin filaments and are part of a higher order submembranous network by which cadherins are linked to other transmembrane and peripheral cytoplasmic proteins. Other cytoplasmic proteins, including fodrin, as well as *src* and *yes* kinases, also interact with the cadherin/catenin complex.⁵ These interactions may link the cadherin/catenin complex with the cytoskeleton and intracellular signaling pathways. Catenins with molecular weights of 102-105 kD (α -catenin), 92-97 kD (β -catenin), and 82-86 kD (γ -catenin), have been identified. The 120 kD catenin-related protein p120^{ctn} (also known as catenin p120^{ctn}, p120^{cas} and pp120 *src* substrate), was originally described as a prominent substrate for the *Src* oncoprotein and for a variety of receptor tyrosine kinases (RTKs) including those for epidermal growth factor (EGF), platelet derived growth factor (PDGF), and colony stimulating factor-1 (CSF-1).¹ p120^{ctn} contains a central armadillo repeat domain (ARM domain) and is structurally similar to β -catenin and plakoglobin. Like β -catenin and plakoglobin, it binds directly to the cytoplasmic domain of epithelial (E)-cadherin via the ARM domain and co-precipitates in multiprotein complexes containing E-cadherin or other cadherins such as neural (N)- or placental (P)-cadherins, depending on the cell type.^{1,4} However, unlike β -catenin and plakoglobin, p120^{ctn} does not appear to bind α -catenin. At least 6 alternatively spliced p120^{ctn} isoforms are generated and differentially expressed in different cell types.⁶ Monoclonal antibody, reacting specifically with p120^{ctn}, is an essential tool in

defining the interactions and distributions of p120^{ctn}, its relationships with other catenins and cadherins and role in RTK signaling and regulation of cadherin mediated cell-adhesion.

Reagents

Monoclonal Anti-p120^{ctn} (Catenin-related) is supplied as a culture supernatant containing 15 mM sodium azide.

Precautions and Disclaimer

Due to the sodium azide content a material safety sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution.

Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

For continuous use, store at 2-8°C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

A minimum working dilution of 1:100 is determined by indirect immunofluorescent labeling of cultured Madin Darby canine kidney (MDCK) epithelial cells, and 1:10,000 by immunoblotting using a whole cell extract of cultured chicken fibroblasts.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working dilution by titration test.

References

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2. Edelman, G.M., and Crossin, K.L., *Annu. Rev. Biochem.*, **60**, 155 (1991).
3. Takeichi, M., *Science*, **251**, 1451 (1991).
4. Reynolds, A.B., and Daniel, J.M., In: *Cytoskeletal-membrane Interactions and Signal Transduction* (Cowin, P., and Klymkowsky, M.W., eds.), pp. 31-48, Landes Company and Chapman & Hall, Austin, TX (1997).
5. Tsukita, S., et al., *J. Cell Biol.*, **123**, 1049 (1993).
6. Mo, Y.Y., and Reynolds A.B., *Cancer Res.*, **56**, 2633 (1996).

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