Revision 1

Store at -20C

#13236

Adherens Junction Antibody Sampler Kit

Cell Signaling TECHNOLOGY®

Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
α-E-Catenin (23B2) Rabbit mAb	3240	40 µl	100 kDa	Rabbit IgG
β-Catenin (D10A8) XP® Rabbit mAb	8480	40 µl	92 kDa	Rabbit IgG
γ-Catenin Antibody	2309	40 µl	83 kDa	Rabbit
Catenin δ-1 Antibody	4989	40 µl	100 kDa	Rabbit
Afadin (D1Y3Z) Rabbit mAb	13531	40 µl	205 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Adherens Junction Antibody Sampler Kit provides an economical means of detecting the protein components of adherens junctions. The kit includes enough antibody to perform four western blot experiments per primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

Adherens junctions are dynamic structures that form cell-cell contacts and are important in development, differentiation, tissue integrity, morphology and cell polarity. They are composed of the transmembrane proteins, cadherins, which bind cadherins on adjacent cells in a calcium-dependent manner. On the cytoplasmic side of adherens junctions, the classic model states that cadherins are linked to the cytoskeleton through β- and α-catenin. α-E-catenin is ubiquitously expressed, α-N-catenin is expressed in neuronal tissue, and α-T-catenin is primarily expressed in heart tissue. Research studies have demonstrated that loss of E-cadherin and α-E-catenin occurs during the progression of several human cancers, indicating that the breakdown of adherens junctions is important in cancer progression (reviewed in 1).

Research studies also suggest that, rather than acting as a static link between cadherins and actin, α-catenin regulates actin dynamics directly, possibly by competing with the actin nucleating arp2/3 complex (2,3). α-catenin also plays a role in regulating β-catenin-dependent transcriptional activity, affecting differentiation and response to Wnt signaling. α-catenin binds to β-catenin in the nucleus, preventing it from regulating transcription, and levels of both proteins appear to be regulated via proteasome-dependent degradation (4).

Afadin has two splice variants: l-afadin, which is ubiquitously expressed, and s-afadin, which is expressed predominantly in neural tissue. s-afadin is a shorter form lacking one of the three proline-rich regions found in l-afadin, as well as the carboxyl-terminal F-actin binding region (5). Human s-afadin is identical to AF-6, the ALL-1 fusion partner involved in acute myeloid leukemias (6). Recent research has also shown that afadin is involved in controlling the directionality of cell movement when it is localized at the leading edge of moving cells (7,8).

Background References

1. Kobielak, A. and Fuchs, E. (2004) *Nat Rev Mol Cell Biol* 5, 614-25.
2. Yamada, S. et al. (2005) *Cell* 123, 889-901.
3. Drees, F. et al. (2005) *Cell* 123, 903-15.
4. Hwang, S.G. et al. (2005) *J Biol Chem* 280, 12758-65.
5. Mandai, K. et al. (1997) *J Cell Biol* 139, 517-28.
6. Prasad, R. et al. (1993) *Cancer Res* 53, 5624-8.
7. Miyata, M. et al. (2009) *J Cell Sci* 122, 4319-29.
8. Miyata, M. et al. (2009) *J Biol Chem* 284, 24595-609.

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