Revision 1

Store at -20C

#4751

Intermediate Filaments 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
Pan-Keratin (C11) Mouse mAb	4545	40 µl	46-58 kDa	Mouse IgG1
GFAP (GA5) Mouse mAb	3670	40 µl	50 kDa	Mouse IgG1
Vimentin (D21H3) XP® Rabbit mAb	5741	40 µl	57 kDa	Rabbit IgG
Desmin (D93F5) XP® Rabbit mAb	5332	40 µl	53 kDa	Rabbit IgG
Plectin-1 (D6A11) Rabbit mAb	12254	40 µl	400-500 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat
Anti-mouse IgG, HRP-linked Antibody	7076	100 µl		Horse

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

Description

The Intermediate Filaments Antibody Sampler Kit provides an economical means to evaluate the presence and status of intermediate filaments. The kit includes enough primary and secondary antibody to perform four Western blot experiments per 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

The cytoskeleton consists of three types of cytosolic fibers: microfilaments (actin filaments), intermediate filaments and microtubules. Major types of intermediate filaments are distinguished and expressed in particular cell types: cytokeratins (epithelial cells), glial fibrillary acidic protein, GFAP (glial cells), desmin (skeletal, visceral and certain vascular smooth muscle cells), vimentin (mesenchyme origin) and neurofilaments (neurons). GFAP and vimentin form intermediate filaments in astroglial cells and modulate their motility and shape (1). In particular, vimentin filaments are present at early developmental stages, while GFAP filaments are characteristic of differentiated and mature brain astrocytes. Thus, GFAP is commonly used as a marker for intracranial and intraspinal tumors arising from astrocytes (2). Vimentin is present in sarcomas, but not carcinomas, and its expression is examined in conjunction with that of other markers to distinguish between the two (3).

Desmin is a myogenic marker expressed in early development that forms a network of filaments that extends across the myofibril and surrounds Z discs. The desmin cytoskeleton provides a connection between myofibrils, organelles and the cytoskeleton (4). Desmin knockout mice develop cardiomyopathy as well as skeletal and smooth muscle defects (5). In humans, desmin related myopathies might be caused by mutations in the corresponding desmin gene or in proteins with which desmin interacts, including αB-crystallin and synemin. Disorganized desmin filaments and the accumulation of protein aggregates comprised predominantly of desmin characterize desmin-related myopathies (reviewed in 6,7).

Keratins assemble into filaments, forming heterodimers of an acidic keratin (or type I keratin, keratins 9 to 23) and a basic keratin (or type II keratin, keratins 1 to 8) (8,9). Keratin isoforms demonstrate tissue- and differentiation-specific profiles, which make them useful as biomarkers (8). Mutations in keratin genes are associated with skin disorders, liver and pancreatic diseases, and inflammatory intestinal diseases (10-13).

Plectin is a large, widely expressed protein that crosslinks the intermediate filament and actin cytoskeleton, mechanically stabilizing cells and tissues. Plectin also plays a role in the regulation of actin dynamics and acts as a scaffold for signaling molecules (14). It is important in the stabilization of hemidesmosomes, crosslinking them to the intermediate filament network. Plectin has been shown to be involved in several signaling cascades. It signals to PKC by binding to and sequestering RACK1, the receptor for activated C kinase 1 (15,16). Plectin is also involved in the regulation of cytokeratin architecture and cell stress response (16), signaling through the chemokine receptor CXCR4 (17), regulation of AMP-activated protein kinase (AMPK) activity and signaling in mouse myotubes (18).

Background References

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