Revision 8

Store at +4C

#87105

FastScan™ Total Vimentin ELISA Kit

Cell Signaling TECHNOLOGY®

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1 Kit (96 assays)

Species Cross Reactivity: H Mk
UniProt ID: #P08670
Entrez-Gene Id: #7431

For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Color
FastScan™ ELISA Microwell Strip Plate, 96 Well	53257	96 tests	
Vimentin Rabbit Capture mAb	95256	1 ea	Green (Lyophilized)
Vimentin Mouse HRP-linked mAb	15464	1 ea	Red (Lyophilized)
FastScan™ ELISA Capture Antibody Diluent	16076	3 ml	Green
FastScan™ ELISA HRP Antibody Diluent	28120	3 ml	
TMB Substrate	7004	11 ml	
STOP Solution	7002	11 ml	
Sealing Tape	54503	1 ea	
ELISA Wash Buffer (20X)	9801	25 ml	
FastScan™ ELISA Cell Extraction Buffer (5X)	69905	10 ml	
FastScan™ ELISA Cell Extraction Enhancer Solution (50X)	25243	1 ml	
FastScan™ ELISA Kit #87105 Positive Control Type 1	29127	1 ea	

Kit contents scale proportionally with size, except sealing tape.
Example: The V1 kit contains 5X the listed quantities above, but will exclude the sealing tape.

The microwell plate is supplied as 12 8-well modules - Each module is designed to break apart for 8 tests.

Description

The FastScan™ Total Vimentin ELISA Kit is a sandwich enzyme-linked immunosorbent assay (ELISA) that detects endogenous levels of vimentin. To perform the assay, sample is incubated with a capture antibody conjugated with a proprietary tag and a second detection antibody linked to HRP, forming a sandwich with vimentin in solution. This entire complex is immobilized to the plate via an anti-tag antibody. The wells are then washed to remove unbound material. TMB is then added. The magnitude of observed signal is proportional to the quantity of vimentin.

*Antibodies in this kit are custom formulations specific to kit.

Specificity/Sensitivity

The FastScan™ Total Vimentin ELISA Kit detects endogenous levels of vimentin as shown in Figure 1. This kit detects proteins from the indicated species, as determined through in-house testing, but may also detect homologous proteins from other species.

Background

The cytoskeleton consists of three types of cytosolic fibers: microfilaments (actin filaments), intermediate filaments, and microtubules. Major types of intermediate filaments are distinguished by their cell-specific expression: 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). Research studies have shown that 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). Vimentin's dynamic structural changes and spatial re-organization in response to extracellular stimuli help to coordinate various signaling pathways (4). Phosphorylation of vimentin at Ser56 in smooth muscle cells regulates the structural arrangement of vimentin filaments in response to serotonin (5,6). Remodeling of vimentin and other intermediate filaments is important during lymphocyte adhesion and migration through the endothelium (7).

During mitosis, CDK1 phosphorylates vimentin at Ser56. This phosphorylation provides a PLK binding site for vimentin-PLK interaction. PLK further phosphorylates vimentin at Ser83, which might serve as a memory phosphorylation site and play a regulatory role in vimentin filament disassembly (8,9). Additionally, studies using various soft-tissue sarcoma cells have shown that phosphorylation of

vimentin at Ser39 by Akt1 enhances cell migration and survival, suggesting that vimentin could be a potential target for soft-tissue sarcoma targeted therapy (10,11).

Background References

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4. Helfand, B.T. et al. (2004) *J Cell Sci* 117, 133-41.
5. Tang, D.D. et al. (2005) *Biochem J* 388, 773-83.
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Limited Uses