

## Epithelial-Mesenchymal Transition (EMT) IF Antibody Sampler Kit



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1 Kit (8 x 20 microliters)

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Claudin-1 (D3H7C) Rabbit mAb	13995	20 µl	20 kDa	Rabbit IgG
E-Cadherin (24E10) Rabbit mAb	3195	20 µl	135 kDa	Rabbit IgG
N-Cadherin (D4R1H) XP <sup>®</sup> Rabbit mAb	13116	20 µl	140 kDa	Rabbit IgG
Slug (C19G7) Rabbit mAb	9585	20 µl	30 kDa	Rabbit IgG
TWIST1 (E5G9Y) Rabbit mAb	90445	20 µl	26 kDa	Rabbit IgG
Vimentin (D21H3) XP <sup>®</sup> Rabbit mAb	5741	20 µl	57 kDa	Rabbit IgG
ZEB1 (E2G6Y) XP <sup>®</sup> Rabbit mAb	70512	20 µl	200 kDa	Rabbit IgG
ZO-1 (D6L1E) Rabbit mAb	13663	20 µl	220 kDa	Rabbit IgG

Please visit [cellsignal.com](http://cellsignal.com) for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

### Description

The Epithelial-Mesenchymal Transition (EMT) IF Antibody Sampler Kit provides an economical means to evaluate the expression of established markers of EMT by immunofluorescence (IF).

### 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 antibodies.*

### Background

Epithelial-mesenchymal transition (EMT) is an essential process during development whereby epithelial cells acquire mesenchymal, fibroblast-like properties and display reduced intracellular adhesion and increased motility. This is a critical feature of normal embryonic development, which is also utilized by malignant epithelial tumors to spread beyond their origin (1-3). This tightly regulated process is associated with a number of cellular and molecular events. EMT depends on a reduction in expression of cell adhesion molecules. Cadherins mediate calcium-dependent cell-cell adhesion and play critical roles in normal tissue development (4). E-cadherin is considered an active suppressor of invasion and growth of many epithelial cancers (4-6). Recent studies indicate that cancer cells have upregulated N-cadherin in addition to loss of E-cadherin. This change in cadherin expression is called the "cadherin switch" and downregulation of E-cadherin is one of the hallmarks of EMT (1). Tight junctions, or zonula occludens, form a continuous barrier to fluids across the epithelium and endothelium. They function in regulation of paracellular permeability and in the maintenance of cell polarity, blocking the movement of transmembrane proteins between the apical and the basolateral cell surfaces. Tight junctions are composed of claudin and occludin proteins, which join the junctions to the cytoskeleton (7,8). Zona occludens (ZO) proteins (e.g., ZO-1) are peripheral membrane adaptor proteins that link junctional transmembrane proteins such as occludin and claudin to the actin cytoskeleton (9). ZO proteins are required for tight junction formation and function (10,11); mutations in ZO-1 and claudin induce EMT (12). Vimentin is an intermediate filament of mesenchymal origin and is present at early developmental stages. Vimentin's dynamic structural changes and spatial re-organization in response to extracellular stimuli helps to coordinate various signaling pathways (13). Slug (SNAI2) is a widely expressed transcriptional repressor and member of the Snail family of zinc finger transcription factors (14,15). Similar to the related Snail protein, Slug binds to the E-cadherin promoter region to repress transcription during development (16). The binding of Slug to integrin promoter sequences represses integrin expression and results in reduced cell adhesion (17). ZEB family proteins (e.g., ZEB1) are zinc finger and homeobox domain containing transcription factors, whose targets of regulation include E-cadherin (1). TWIST1 is a basic helix-loop-helix (b-HLH) transcription factor that functions as a master regulator of embryonic morphogenesis, and plays essential roles in mesenchymal differentiation (18,19). TWIST is upregulated in various human tumors and has been suggested to be a driver of EMT and metastasis (20-21).

### Background References

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