

# Anti-Fascin (Ser-39), Phosphospecific Antibody

Catalog # AN1793

#### **Product Information**

ApplicationWB, ICCPrimary AccessionQ16658HostRabbit

**Clonality** Rabbit Polyclonal

**Isotype** IgG **Calculated MW** 54530

#### **Additional Information**

**Gene ID** 6624 **Other Names** p55

**Target/Specificity** Fascin is an actin filament bundling protein localized to lamellipodia and

filopodia where it has important roles in cell motility. Regulation of fascin occurs through PKC-mediated phosphorylation of Ser-39 in the F-actin binding site. Cell permeant peptides that block PKC phosphorylation of Ser-39 increase cell migration, while peptides that block fascin binding to F-actin alter lamellipodial morphology and cause aberrant cell motility. Studies using RNA interference of fascin show that fibroblasts have reduced number and abnormal morphology of filopodia, while Ser-39 phosphorylation status may determine filopodial frequency. In Drosophila neurons, fascin deficiency causes alterations in actin filaments and leads to abnormal morphology of developing neurons. Thus, fascin is a critical element of actin-based motility in

various cell types.

**Dilution** WB~~1:1000 ICC~~N/A

**Storage** Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store

at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**Anti-Fascin (Ser-39), Phosphospecific Antibody is for research use only and

not for use in diagnostic or therapeutic procedures.

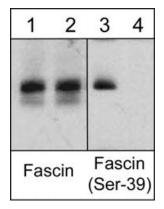
Shipping Blue Ice

## **Background**

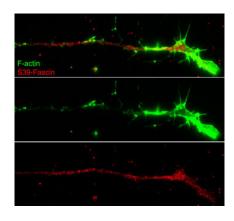
Fascin is an actin filament bundling protein localized to lamellipodia and filopodia where it has important roles in cell motility. Regulation of fascin occurs through PKC-mediated phosphorylation of Ser-39 in the F-actin binding site. Cell permeant peptides that block PKC phosphorylation of Ser-39 increase cell migration, while peptides that block fascin binding to F-actin alter lamellipodial morphology and cause aberrant cell motility. Studies using RNA interference of fascin show that fibroblasts have reduced number and abnormal morphology of filopodia, while Ser-39 phosphorylation status may determine filopodial

frequency. In Drosophila neurons, fascin deficiency causes alterations in actin filaments and leads to abnormal morphology of developing neurons. Thus, fascin is a critical element of actin-based motility in various cell types.

### **Images**



Western blot analysis of human HeLa cells treated with Calyculin A (100 nM) for 30 min (lanes 1 & 3) before treatment with lambda phosphatase (lanes 2 & 4). The blots were probed with anti-Fascin (clone 55K2) (lanes 1 & 2) and anti-Fascin (Ser-39) (lanes 3 & 4).



Immunocytochemical labeling of fascin phosphorylation relative to F-actin in chick E9 DRG neurons. The cells were labeled with rabbit polyclonal Fascin (Ser-39) antibody, then detected using appropriate secondary antibody (Red). Fascin (Ser-39) labeling is compared (Top) to F-actin staining (Green). (Image provided by Dr. Gianluca Gallo at Drexel University).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.