10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-JMY (C-terminal region) Antibody

Catalog # AN1827

Product Information

Application	WB, ICC
Primary Accession	Q9QXM1
Reactivity	Rat
Host	Rabbit
Clonality	Rabbit Polyclonal
Isotype	IgG
Calculated MW	110586

Additional Information

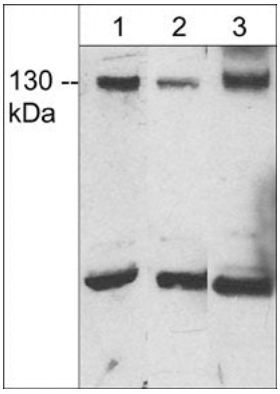
Gene ID	57748
Other Names	p53 cofactor, WHDC1L3; FLJ37870; MGC163496
Target/Specificity	JMY (junction mediating and regulatory protein) is a transcription co-factor, originally identified as a p300-binding protein involved in p53-dependent transcription. Upon DNA damage, JMY is released from Mdm2 inhibition and forms a complex with Strap and p300. This complex recruits PRMT5 to activate the p53 response. Through regulation of p53-dependent transcription, JMY has important roles in the DNA damage response. In addition, JMY contains three carboxyl-terminal WH2 actin binding domains which are are commonly found in WASP family proteins. JMY can bind to actin and to the Arp2/3 complex, as well as direct the assembly of actin filaments in vitro. These actin-regulating effects of JMY may have important roles in cell migration. When slow migrating HL-60 cells are differentiated into highly motile neutrophil-like cells, JMY moves from the nucleus to the cytoplasm and is concentrated at the actin-rich leading edge of cells. The loss of JMY leads to decreased cell migration in HL-60 cells. Thus, JMY represents a new class of multifunctional actin assembly factors whose activity may be regulated by cellular localization.
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-JMY (C-terminal region) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

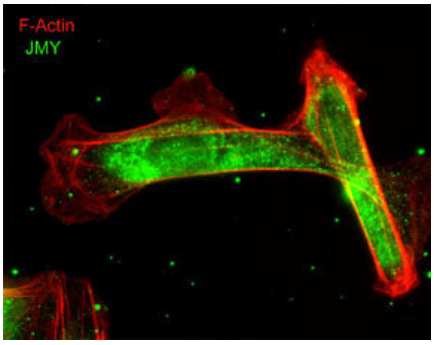
JMY (junction mediating and regulatory protein) is a transcription co-factor, originally identified as a p300-binding protein involved in p53-dependent transcription. Upon DNA damage, JMY is released from

Mdm2 inhibition and forms a complex with Strap and p300. This complex recruits PRMT5 to activate the p53 response. Through regulation of p53-dependent transcription, JMY has important roles in the DNA damage response. In addition, JMY contains three carboxyl-terminal WH2 actin binding domains which are are commonly found in WASP family proteins. JMY can bind to actin and to the Arp2/3 complex, as well as direct the assembly of actin filaments in vitro. These actin-regulating effects of JMY may have important roles in cell migration. When slow migrating HL-60 cells are differentiated into highly motile neutrophil-like cells, JMY moves from the nucleus to the cytoplasm and is concentrated at the actin-rich leading edge of cells. The loss of JMY leads to decreased cell migration in HL-60 cells. Thus, JMY represents a new class of multifunctional actin assembly factors whose activity may be regulated by cellular localization.

Images



Western blot showing JMY expression in rat PC12 cells (lane 1), human Jurkat cells (lane 2), and adult mouse heart (lane 3). The blots were probed with anti-JMY (C-terminal region) rabbit polyclonal antibody at 1:500.



Immunocytochemical labeling of JMY relative to F-actin in chick fibroblasts. The cells were labeled with rabbit polyclonal JMY antibody (AN1827), then detected using appropriate secondary antibody (Green). This labeling is compared to F-actin staining (Red). (Image provided by Dr. Gianluca Gallo at Drexel University).