

Anti-Integrin β4 (Cytoplasmic region) Antibody

Catalog # AN1824

Product Information

Application WB, ICC
Primary Accession P16144
Host Mouse

Clonality Mouse Monoclonal

IsotypeIgG1Clone NamesM126Calculated MW202167

Additional Information

Gene ID 3691

Other Names integrin, CD104, GP150

Target/Specificity The NF-κB/Rel transcription factors are present in the cytosol in an inactive

state complexed with the inhibitory IkB proteins. Activation of IkB α occurs through both serine and tyrosine phosphorylation events. Activation through phosphorylation at Ser-32 and Ser-36 is followed by proteasome-mediated degradation, resulting in the release and nuclear translocation of active NF-kB. This pathway of IkB α regulation occurs in response to various NF-kB-activating agents, such as TNF α , interleukins, LPS, and irradiation. An

alternative pathway for IκBα regulation occurs through tyrosine

phosphorylation of Tyr-42 and Tyr-305. Tyr-42 is phosphorylated in response to oxidative stress and growth factors. This phosphorylation can lead to

degradation of IκBα and NF-κB-activation. In contrast, Tyr-305

phosphorylation by c-Abl has been implicated in IκBα nuclear translocation and inhibition of NF-κB-activation. Thus, tyrosine phosphorylation of IκBα

may be an important regulatory mechanism in NF-kB signaling.

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-Integrin β4 (Cytoplasmic region) Antibody is for research use only and

not for use in diagnostic or therapeutic procedures.

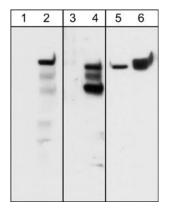
Shipping Blue Ice

Background

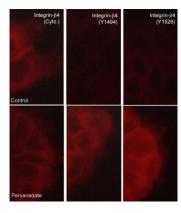
The NF- κ B/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory I κ B proteins. Activation of I κ B α occurs through both serine and tyrosine phosphorylation events. Activation through phosphorylation at Ser-32 and Ser-36 is followed by proteasome-mediated degradation,

resulting in the release and nuclear translocation of active NF-κB. This pathway of IκBα regulation occurs in response to various NF-κB-activating agents, such as TNFα, interleukins, LPS, and irradiation. An alternative pathway for IκBα regulation occurs through tyrosine phosphorylation of Tyr-42 and Tyr-305. Tyr-42 is phosphorylated in response to oxidative stress and growth factors. This phosphorylation can lead to degradation of IκBα and NF-κB-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in IκBα nuclear translocation and inhibition of NF-κB-activation. Thus, tyrosine phosphorylation of IκBα may be an important regulatory mechanism in NF-κB signaling.

Images



Western blot analysis of A431 cells serum starved overnight (lanes 1, 3, & 5) and treated with pervanadate (1 mM) for 30 min (lanes 2, 4, & 6). The blots were probed with rabbit polyclonal anti-Integrin β 4 (Tyr-1526) (lanes 1 & 2) and anti-Integrin β 4 (Tyr-1494) (lanes 3 & 4) or with mouse monoclonal anti-Integrin β 4 (lanes 5 & 6).



Immunocytochemical labeling of integrin $\beta 4$ in control (Top) and pervanadate-treated A431 cells (Bottom). The cells were labeled with mouse monoclonal anti-integrin $\beta 4$ (Cytoplasmic region) (left) or rabbit polyclonals anti-integrin $\beta 4$ (Tyr-1494) (middle) or anti-integrin $\beta 4$ (Tyr-1526) (right), then the antibodies were detected using appropriate secondary antibodies conjugated to DyLight® 594.

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