

Anti-Integrin β 4 (Cytoplasmic region) Antibody
Catalog # AN1824**Specification****Anti-Integrin β 4 (Cytoplasmic region) Antibody - Product Information**

Application	WB
Primary Accession	P16144
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Calculated MW	202167

Anti-Integrin β 4 (Cytoplasmic region) Antibody - Additional Information

Gene ID	3691
Other Names	
integrin, CD104, GP150	

Target/Specificity

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- κ B. This pathway of I κ B α regulation occurs in response to various NF- κ B-activating agents, such as TNF α , interleukins, LPS, and irradiation. An alternative pathway for I κ B α 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 I κ B α and NF- κ B-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in I κ B α nuclear translocation and inhibition of NF- κ B-activation. Thus, tyrosine phosphorylation of I κ B α may be an important regulatory mechanism in NF- κ B signaling.

Dilution

WB~~1:1000

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 β 4 (Cytoplasmic region) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping

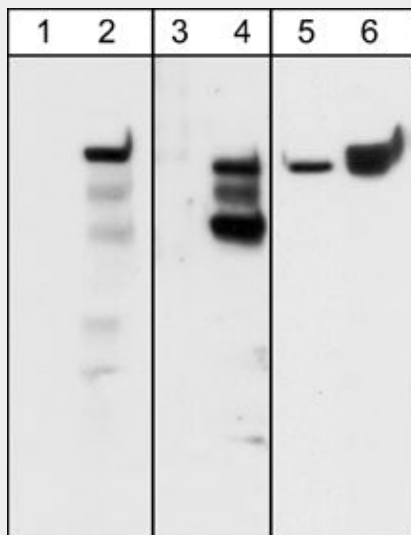
Blue Ice

Anti-Integrin β 4 (Cytoplasmic region) Antibody - Protocols

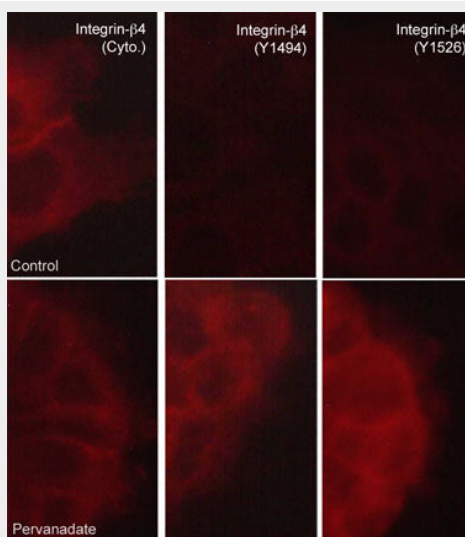
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Anti-Integrin $\beta 4$ (Cytoplasmic region) Antibody - 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 $\beta 4$ (Tyr-1526) (lanes 1 & 2) and anti-Integrin $\beta 4$ (Tyr-1494) (lanes 3 & 4) or with mouse monoclonal anti-Integrin $\beta 4$ (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.

Anti-Integrin $\beta 4$ (Cytoplasmic region) Antibody - 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- κ B. This pathway of I κ B α regulation occurs in response to various NF- κ B-activating agents, such as TNF α , interleukins, LPS, and irradiation. An alternative pathway for I κ B α 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 I κ B α and NF- κ B-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in I κ B α nuclear translocation and inhibition of NF- κ B-activation. Thus, tyrosine phosphorylation of I κ B α may be an important regulatory mechanism in NF- κ B signaling.