# IFN-α/βRα (R-100): sc-845

## BACKGROUND

The type I interferons (IFNs), α and β, are a group of structurally and functionally related proteins that are induced by either viruses or double stranded RNA and defined by their ability to confer an antiviral state in cells. The α and β IFNs appear to compete with one another for binding to a common cell surface receptor, while immune IFN (IFN-γ) binds to a distinct receptor. The latter protein, IFN-αR, is only weakly responsive to type I interferons in contrast to IFN-α/βR, which binds to and responds effectively to IFN-β and to several of the IFN-α subtypes. Moreover, IFN-α/βR is physically associated with the cytoplasmic tyrosine kinase JAK1 and thus, in addition to ligand binding, appears to be functionally involved in signal transduction. The IFN-γ receptor complex consists of an α subunit (IFN-γRα) and a β subunit that is 332 amino acids in length (mouse) and 337 amino acids in length (human).

## CHROMOSOMAL LOCATION

Genetic locus: IFNAR1 (human) mapping to 21q22.11; Ifnar1 (mouse) mapping to 16 C3.3.

## SOURCE

IFN-α/βRα (R-100) is a rabbit polyclonal antibody raised against amino acids 458-557 mapping at the C-terminus of IFN-α/βRα of human origin.

## PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

## APPLICATIONS

IFN-α/βRα (R-100) is recommended for detection of IFN-α/βRα chain of human and, to a lesser extent, mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for IFN-α/βRα siRNA (h): sc-35637, IFN-α/βRα siRNA (m): sc-40090, IFN-α/βRα shRNA Plasmid (h): sc-35637-SH, IFN-α/βRα shRNA Plasmid (m): sc-40090-SH, IFN-α/βRα shRNA (h) Lentiviral Particles: sc-35637-V and IFN-α/βRα shRNA (m) Lentiviral Particles: sc-40090-V.

Molecular Weight of IFN-α/βRα α subunit: 110 kDa.

Molecular Weight of IFN-α/βRα β subunit: 95-100 kDa.

Molecular Weight of IFN-α/βRα β subunit short form: 55 kDa.

Positive Controls: A-431 whole cell lysate: sc-2201 or IFN-β/βRα (h): 293T Lysate: sc-113922.
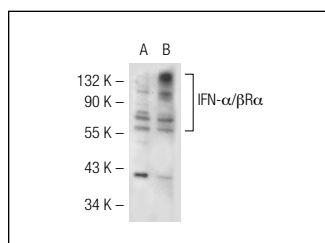
## STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.

## DATA



IFN-α/βRα (R-100): sc-845. Western blot analysis of IFN-α/βRα expression in non-transfected: sc-117752 (**A**) and human IFN-α/βRα transfected: sc-113922 (**B**) 293T whole cell lysates.

## SELECT PRODUCT CITATIONS

1. Giosue, S., et al. 1998. Effects of aerosolized interferon-α in patients with pulmonary tuberculosi. Am. J. Respir. Crit. Care Med. 158: 1156-1162.

2. Komatsu, T., et al. 2000. Sendai virus blocks α interferon signaling to signal transducers and activators of transcription. J. Virol. 74: 2477-2480.

3. Gotoh, B., et al. 2003. The Stat2 activation process is a crucial target of Sendai virus C protein for the blockade of α interferon signaling. J. Virol. 77: 3360-3370.

4. Subramaniam, P.S., et al. 2004. The IFNAR1 subunit of the type I IFN receptor complex contains a functional nuclear localization sequence. FEBS Lett. 578: 207-210.

5. Eriksen, K.W., et al. 2005. Increased sensitivity to interferon-α in psoriatic T cells. J. Invest. Dermatol. 125: 936-944.

6. Vitale, G., et al. 2006. IFN-β is a highly potent inhibitor of gastroenteropancreatic neuroendocrine tumor cell growth *in vitro*. Cancer Res. 66: 554-562.

7. Vitale, G., et al. 2007. Type I interferons in the treatment of pancreatic cancer: mechanisms of action and role of related receptors. Ann. Surg. 246: 259-268.

8. Shimazui, T., et al. 2007. Prediction of *in vitro* response to interferon-α in renal cell carcinoma cell lines. Cancer Sci. 98: 529-534.

9. Chen, H.M., et al. 2009. Critical role for constitutive type I interferon signaling in the prevention of cellular transformation. Cancer Sci. 100: 449-456.

10. Krämer, O.H. and Heinzel, T. 2010. Phosphorylation-acetylation switch in the regulation of STAT1 signaling. Mol. Cell. Endocrinol. 315: 40-48.

11. Ruiz-González, I., et al. 2012. Endometrial expression of IFNAR-1 and oxytocin receptor (OTR) is not improved by prostaglandin analogues when compared to progestagens in ewes. Reprod. Domest. Anim. 47: 274-280.