Human IL-13 Rα2 Alexa Fluor® 750-conjugated Antibody

Monoclonal Rabbit IgG Clone # 2725C
Catalog Number: FAB6142S
100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects Human IL-13 Rα2 in direct ELISAs.
Source	Monoclonal Rabbit IgG Clone # 2725C
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Chinese Hamster Ovary cell line CHO-derived Human IL-13 Rα2 Met1-Leu342 Accession # NP_000631
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

Western Blot	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Interleukin-13 Receptor alpha 2 (IL-13 Rα2), also known as IL-13 binding protein, and CD213a2, is a widely expressed 55 kDa cytokine receptor that plays an important role in the Th2-polarized immune responses characteristic of a variety of pathologies, including parasitic infections and allergic asthma (1, 2). Mature human IL-13 Rα2 consists of a 317 amino acid (aa) extracellular domain with three fibronectin type-III domains, a WSxWS motif, a 20 aa transmembrane segment, and a 17 aa cytoplasmic domain (3). Within the ECD, human IL-13 Rα2 shares 64% and 62% aa sequence identity with mouse and rat IL-13 Rα2, respectively. In both mouse and human, a 40 kDa-50 kDa soluble form of IL-13 Rα2 can be generated by MMP-8 mediated shedding *in vitro* (4). Although this is assumed to occur *in vivo* in mouse, there is no evidence that shedding occurs in human (5-7). In mouse, alternative splicing also leads to sIL-13 Rα2, but again, this phenomenon apparently does not occur in human (6-7). Thus, the biological effects of human IL-13 Rα2 would appear to be mediated exclusively by membrane IL-13 Rα2 (7). The biological effects of IL-13 and IL-4 are closely related in part due to a shared receptor system. IL-13 binds to IL-13 Rα1 which then forms a signaling complex with IL-4 Rα (8, 9). IL-13 Rα2 functions as a decoy receptor by binding and internalizing IL-13 and preventing it from signaling through the IL-13 Rα1/IL-4 Rα complex (3, 10). IL-13 Rα2 can also block IL-4 induced responses by inhibiting IL-4 bound IL-13 Rα1/IL-4 Rα receptor complexes even though it does not itself bind IL-4 (11, 12). Aside from its decoy function, IL-13-activated IL-13 Rα2 directly promotes the development of tissue fibrosis by inducing the transcription of TGF-β (13). Presumably, any human soluble IL-13 Rα2, if it exists, will retain its ligand binding capability and attenuate responses to IL-13 but not to IL-4 (11, 14). The up-regulation of transmembrane during Th2-biased immune responses limits the extent of those responses (15-17).

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Bio-Techne®
Global | bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL: 1.612.379.2956
USA | TEL: 800.343.7475 Canada | TEL: 855.668.8722 Europe | Middle East | Africa TEL: +44.0.1235.529449
China | info.cn@bio-techne.com TEL: 400.821.3475