

Human IL-13 Rα2 Alexa Fluor® 594-conjugated Antibody

Monoclonal Rabbit IgG Clone # 2725C Catalog Number: FAB6142T

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 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 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 R α 2), 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 R α 2 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 R α 2 shares 64% and 62% aa sequence identity with mouse and rat IL-13 R α 2, respectively. In both mouse and human, a 40 kDa-50 kDa soluble form of IL-13 R α 2 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 R α 2, but again, this phenomenon apparently does not occur in human (6-7). Thus, the biological effects of human IL-13 R α 2 would appear to be mediated exclusively by membrane IL-13 R α 2 (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 R α 1 which then forms a signaling complex with IL-4 R α (8, 9). IL-13 R α 2 functions as a decoy receptor by binding and internalizing IL-13 and preventing it from signaling through the IL-13 R α 1/IL-4 R α 1 complex (3, 10). IL-13 R α 2 can also block IL-4 induced responses by inhibiting IL-4 bound IL-13 R α 1/IL-4 R α 1 receptor complexes even though it does not itself bind IL-4 (11, 12). Aside from its decoy function, IL-13-activated IL-13 R α 2 directly promotes the development of tissue fibrosis by inducing the transcription of TGF- α 3 (13). Presumably, any human soluble IL-13 R α 2, 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 ex

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