

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Resistin in ELISAs and Western blots. In Western blots, this antibody does not cross-react with recombinant mouse RELM α .
Source	Monoclonal Mouse IgG _{2B} Clone # 184305
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human Resistin
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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.

ELISA Capture (Matched Antibody Pair)	Optimal dilution of this antibody should be experimentally determined.
ELISA Detection (Matched Antibody Pair)	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

Resistin (resistance-to-insulin), also known as adipocyte-specific secretory factor (ADSF) and found in inflammatory zone 3 (FIZZ3), is a 10 kDa member of a small family of secreted cysteine-rich peptide hormones. These molecules purportedly play some role in inflammation, glucose metabolism, and angiogenesis (1-4). Human Resistin precursor is 108 amino acids (aa) in length. It contains an 18 aa signal sequence plus a 90 aa mature region. The mature region shows an N-terminal α -helical tail (aa 23-44) and a C-terminal β -sheet globular head (aa 47-108) (5-7). The Resistin molecule circulates as either a noncovalent trimer (minor form), or a disulfide-linked homohexamer (major form). Noncovalent trimers are generated when the α -helical segments self-associate to form a three-stranded coiled-coil structure. Covalent hexamers subsequently appear when the free Cys at position #26 is engaged by adjacent trimers. It is hypothesized that the hexamer is the inactive form of the molecule, and bioactivity is achieved at the target site by disulfide bond reduction (5). Although Resistin family molecules can noncovalently interact to form heterotrimers *in vitro*, there is no evidence to suggest this occurs *in vivo* with Resistin (8, 9). Mature human Resistin shares 56% and 54% aa identity with mouse and rat Resistin, respectively. Rat Resistin possesses an alternate start site at Met48; this Met is not found in the mouse molecule, however (10). Rodent resistin is expressed by white adipocytes, splenocytes, astrocytes, and anterior pituitary epithelium (6, 11, 12). Although the function of Resistin is unclear, it would seem to block insulin-stimulated uptake of glucose by adipocytes and promote glucose release by hepatocytes (6, 13, 14). As such, it has been proposed to participate in diet-induced insulin-sensitivity. Diets high in fat promote an increase in overall adipocyte size. Hypertrophic adipocytes are known to secrete TNF- α which acts locally to block ACRP30 production. Since ACRP30 is an insulin-sensitizer, a drop in ACRP30 availability leads to insulin-*insensitivity*, which drives increased insulin production (a compensatory mechanism). High insulin induces Resistin secretion which now antagonizes insulin action, prompting more insulin production and more Resistin secretion (15).

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