

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 RELMc.
Source	Monoclonal Mouse IgG _{2B} Clone # 184320
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human Resistin Accession # Q9HD89
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

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.

	Recommended Concentration	Sample
Western Blot	1 µg/mL	Recombinant Human Resistin under non-reducing conditions only
Human Resistin Sandwich Immunoassay		Reagent
ELISA Capture	2-8 µg/mL	Human Resistin Antibody (Catalog # MAB13591)
ELISA Detection	0.5-2.0 µg/mL	Human Resistin Biotinylated Antibody (Catalog # BAM1359)
Standard		Recombinant Human Resistin (Catalog # 1359-RN)

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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).

References:

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