

ZAG (1E2): sc-21720



The Power to Question

BACKGROUND

ZAG (Zn- α 2-glycoprotein, also designated Zn- α 2-gp) is a soluble, secreted protein found in serum and other body fluids (such as cerebrospinal fluid, blood plasma, urine and sweat). ZAG has a tendency to precipitate with zinc salts, has electrophoretic mobility in the region of the two globulins, and has 18% carbohydrate content. A member of the immunoglobulin superfamily, ZAG has a high degree of sequence similarity to class-I major histocompatibility complex (MHC) antigens. The ZAG structure includes a large groove analogous to class I MHC peptide binding grooves. The crystal structure of ZAG resembles a class I MHC heavy chain but does not bind the class I light chain β -2-Microglobulin, unlike other MHC related proteins. ZAG stimulates lipid degradation in adipocytes and its overexpression causes the extensive fat losses associated with some advanced cancers.

REFERENCES

- Jirka, M., et al. 1973. Zn- α 2-glycoprotein in sweat. *Cas. Lek. Cesk.* 112: 1606-1608.
- Ekman, R., et al. 1976. Renal handling of Zn- α 2-glycoprotein as compared with that of albumin and the retinol-binding protein. *J. Clin. Invest.* 57: 945-954.
- Shibata, S., et al. 1982. Nephritogenic glycoprotein. IX. Plasma Zn- α 2-glycoprotein as a second source of nephritogenic glycoprotein in urine. *Nephron* 31: 170-176.

CHROMOSOMAL LOCATION

Genetic locus: AZGP1 (human) mapping to 7q22.1; Azgp1 (mouse) mapping to 5 G2.

SOURCE

ZAG (1E2) is a mouse monoclonal antibody raised against purified Zn- α 2-glycoprotein (ZAG) of human origin.

PRODUCT

Each vial contains 200 μ g IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

ZAG (1E2) is recommended for detection of ZAG of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for ZAG siRNA (h): sc-36865, ZAG siRNA (m): sc-36866, ZAG shRNA Plasmid (h): sc-36865-SH, ZAG shRNA Plasmid (m): sc-36866-SH, ZAG shRNA (h) Lentiviral Particles: sc-36865-V and ZAG shRNA (m) Lentiviral Particles: sc-36866-V.

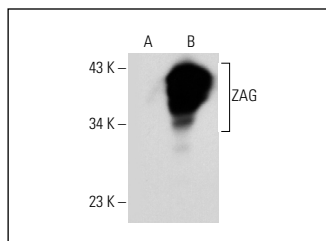
Molecular Weight of ZAG: 47 kDa.

Positive Controls: ZAG (h): 293T Lysate: sc-114991 or mouse spleen extract: sc-2391.

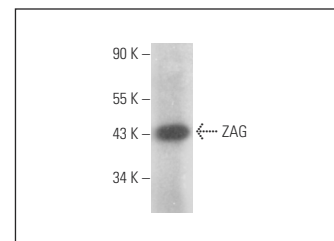
STORAGE

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

DATA



ZAG (1E2): sc-21720. Western blot analysis of ZAG expression in non-transfected: sc-117752 (A) and human ZAG transfected: sc-114991 (B) 293T whole cell lysates.



ZAG (1E2): sc-21720. Western blot analysis of ZAG expression in mouse spleen tissue extract.

SELECT PRODUCT CITATIONS

- Bing, C., et al. 2004. Zinc- α 2-glycoprotein, a lipid mobilizing factor, is expressed in adipocytes and is up-regulated in mice with cancer cachexia. *Proc. Natl. Acad. Sci. USA* 101: 2500-2505.
- Airoldi, L., et al. 2009. Effects of cigarette smoking on the human urinary proteome. *Biochem. Biophys. Res. Commun.* 381: 397-402.
- Mracek, T., et al. 2010. The adipokine zinc- α 2-glycoprotein (ZAG) is downregulated with fat mass expansion in obesity. *Clin. Endocrinol.* 72: 334-341.
- Gong, F.Y., et al. 2010. Fatty acid synthase and hormone-sensitive lipase expression in liver are involved in zinc- α 2-glycoprotein-induced body fat loss in obese mice. *Chin. Med. Sci. J.* 25: 169-175.
- Simo, R., et al. 2014. Thyroid hormone upregulates zinc- α 2-glycoprotein production in the liver but not in adipose tissue. *PLoS ONE* 9: e85753.
- Magagnotti, C., et al. 2018. Identification of nephropathy predictors in urine from children with a recent diagnosis of type 1 diabetes. *J. Proteomics* 193: 205-216.
- Wei, X., et al. 2019. Insulin attenuates epileptiform discharge-induced oxidative stress by increasing zinc- α 2-glycoprotein in primary cultured cortical neurons. *Neuroreport* 30: 580-585.
- Lin, R., et al. 2019. Combined exposure to fructose and bisphenol A exacerbates abnormal lipid metabolism in liver of developmental male rats. *Int. J. Environ. Res. Public Health* 16: E4152.
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RESEARCH USE

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