
Product Data Sheet

Product Name: Infliximab

Cat. No.: GC19533

Chemical Properties

Cas. No. 170277-31-3

Formula $C_{6428}H_{9912}N_{1694}O_{1987}S_{46}$

M.Wt 144188.23

Solubility

Storage Store at -30°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure **Protocol****Cell experiment [1]:**

Cell lines

3T3L1 mature adipocytes

Preparation Method

Adipocytes were stimulated twice at zero and 60 minutes with 2 μ mol L 1 insulin. Adipocytes were stimulated with 10 ng/mL infliximab at the beginning of the 2-hour in vitro assay. 3T3L1 adipocytes were collected every 20 minutes for 2 hour.

Caution: Product has not been fully validated for medical applications. For research use only.

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Reaction Conditions	10 ng/mL infliximab at the beginning of the 2-hour in vitro assay
Applications	Infliximab restores insulin-dependent glucose uptake, phosphorylation of the insulin signaling pathway and attenuates TNF- α -induced PTP1B activation in TNF- α -treated 3T3L1 adipocytes.
Animal experiment [2]:	
Animal models	Eight-week-old C57BL/6J (WT, TNF- α +/+) and TNF- α -deficient (TNF α -/-) mice of strain B6
Preparation Method	Infliximab was injected into diabetic and normal mice for 4 weeks
Dosage form	Infliximab 10 μ g/g in 100 μ l saline for 4 weeks

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Applications

Diabetic mice showed significant impairments in SNCV and MNCV at 8 weeks. Diabetic mice treated with saline showed no improvement in SNCV or MNCV at 12 weeks, whereas diabetic mice treated with infliximab showed significant improvement at this time (4 weeks after infliximab treatment).

References:

[1]. Méndez-García LA, Trejo-Millán F, Martínez-Reyes CP, Manjarrez-Reyna AN, Esquivel-Velázquez M, Melendez-Mier G, Islas-Andrade S, Rojas-Bernabé A, Kzhyshkowska J, Escobedo G. Infliximab ameliorates tumor necrosis factor-alpha-induced insulin resistance by attenuating PTP1B activation in 3T3L1 adipocytes in vitro. *Scand J Immunol*. 2018 Nov;88(5):e12716. doi: 10.1111/sji.12716. Epub 2018 Oct 10. PMID: 30260514.

[2]. Yamakawa I, Kojima H, et al. Inactivation of TNF- α ameliorates diabetic neuropathy in mice. *Am J Physiol Endocrinol Metab*. 2011 Nov;301(5):E844-52. doi: 10.1152/ajpendo.00029.2011. Epub 2011 Aug 2. PMID: 21810933; PMCID: PMC3213998./p>

Background

Infliximab is a chimeric monoclonal IgG1 antibody that specifically binds to TNF- α . Infliximab prevents the interaction of TNF- α with TNF- α receptor (TNFR1 and TNFR2). Infliximab has the potential for autoimmune, chronic inflammatory diseases and diabetic neuropathy research^{[1][2]}. So, infliximab is a medication used to treat patients with

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autoimmune and chronic inflammatory diseases^[5].

infliximab has been also shown to improve insulin resistance^[3]. In vitro, infliximab ameliorates TNF- α -induced insulin resistance in 3T3L1 adipocytes in vitro by restoring phosphorylation of key mediators of the insulin signaling pathway such as IRS-2 and AKT via PTP1B inhibition that in consequence improves insulin-dependent glucose uptake in these adipose cells^[4]

Diabetic mice showed significant impairments in SNCV and MNCV at 8 weeks. Diabetic mice treated with saline showed no improvement in SNCV or MNCV at 12 weeks, whereas diabetic mice treated with infliximab showed significant improvement at this time (4 weeks after infliximab treatment). In conclusion a single injection of infliximab leads to marked improvement in diabetic neuropathy^[7]. Infliximab reduces the levels of serum insulin, fasting glucose and insulin resistance in patients with ankylosing spondylitis and rheumatoid arthritis^[6]

References:

- [1]. Lis K, Kuzawińska O, et,al. Tumor necrosis factor inhibitors - state of knowledge. Arch Med Sci. 2014 Dec 22;10(6):1175-85. doi: 10.5114/aoms.2014.47827. PMID: 25624856; PMCID: PMC4296073.
- [2]. Yamakawa I, Kojima H, Terashima T, et,al. Inactivation of TNF- α ameliorates diabetic neuropathy in mice. Am J Physiol Endocrinol Metab. 2011 Nov;301(5):E844-52. doi: 10.1152/ajpendo.00029.2011. Epub 2011 Aug 2. PMID: 21810933; PMCID: PMC3213998.
- [3]. Burska AN, Sakthiswary R, et,al. Effects of Tumour Necrosis Factor Antagonists on Insulin Sensitivity/Resistance in Rheumatoid Arthritis: A Systematic Review and Meta-Analysis. PLoS One. 2015 Jun 25;10(6):e0128889. doi: 10.1371/journal.pone.0128889. PMID: 26110878; PMCID: PMC4482317.
- [4]. Méndez-García LA, Trejo-Millán F, et,al. Infliximab ameliorates tumor necrosis factor- α -induced insulin resistance by attenuating PTP1B activation in 3T3L1 adipocytes in vitro. Scand J Immunol. 2018 Nov;88(5):e12716. doi: 10.1111/sji.12716. Epub 2018 Oct 10. PMID: 30260514.
- [5]. Antoni C, Krueger GG, et,al. Infliximab improves signs and symptoms of psoriatic arthritis: results of the IMPACT 2 trial. Ann Rheum Dis. 2005 Aug;64(8):1150-7. doi: 10.1136/ard.2004.032268. Epub 2005 Jan 27. PMID: 15677701; PMCID: PMC1755609.
- [6]. Stagakis I, Bertias G, et,al. Anti-tumor necrosis factor therapy improves insulin

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resistance, beta cell function and insulin signaling in active rheumatoid arthritis patients with high insulin resistance. *Arthritis Res Ther.* 2012 Jun 12;14(3):R141. doi: 10.1186/ar3874. PMID: 22691241; PMCID: PMC3446524.

[7]. Yamakawa I, Kojima H, et.al. Inactivation of TNF- α ameliorates diabetic neuropathy in mice. *Am J Physiol Endocrinol Metab.* 2011 Nov;301(5):E844-52. doi: 10.1152/ajpendo.00029.2011. Epub 2011 Aug 2. PMID: 21810933; PMCID: PMC3213998.

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