
Product Data Sheet

Product Name: Abatacept

Cat. No.: GC38130

Chemical Properties

Cas. No. 332348-12-6

SMILES [Abatacept]

Formula M.Wt

Solubility Soluble in water Storage Store at -20°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 Human Burkitt lymphoma B (BJAB) wild type (WT)
CD80 KO CD86 KO cells

Preparation Method BJAB wild type (WT), CD80 KO and CD86 KO cells were treated with Abatacept (10µg/mL) for 2h and CD86 and CD80 expression as assessed by flow cytometry prior removal of bound abatacept by acidic elution.

Reaction Conditions 10µg/mL; 2h

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

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Applications	Reduction in surface expression of CD86 and CD80 on human B-cells in the presence of Abatacept.
Animal experiment [2]:	
Animal models	DO11.10 RAG-2 ^{-/-} mice
Preparation Method	To prime CD41 T cells, DO11.10 RAG-2 ^{-/-} mice were injected subcutaneously in the scruff of the neck with 100 mg of OVA in Freund's complete adjuvant. Mice were treated with 10mg/kg of Abatacept or control IgG beginning 2 days before priming and continuing every 2 days thereafter.
Dosage form	10mg/kg; s.c.
Applications	Abatacept reduced the proportion of activated T cells (CD44 ^{high} CD62L ⁻) and inhibited the upregulation of ICOS and CD71. Cells primed in the presence of Abatacept produced substantial amounts of IL-2, similar to the naive T cells, whereas cells from orally tolerized mice produced very low levels of the cytokine, confirming their anergic state.

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References:

[1]Lorenzetti R, Janowska I, Smulski

C R, et al. Abatacept modulates

CD80 and CD86 expression and

memory formation in human B-

cells[J]. Journal of Autoimmunity,

2019, 101: 145-152.

[2]Patakas A, Ji R R, Weir W, et al.

Abatacept inhibition of T cell

priming in mice by induction of a

unique transcriptional profile that

reduces their ability to activate

antigen-presenting cells[J]. Arthritis

& rheumatology, 2016, 68(3): 627-

638.

Background

Abatacept (CTLA4Ig) is a soluble fusion protein composed of the extracellular domain of human cytotoxic T lymphocyte-associated protein 4 (CTLA4) and a fragment of the human IgG1 Fc portion (hinge, CH2 and 3 domains)^[1]. Abatacept is a selective T cell co-stimulatory modulator and a protein drug used to treat autoimmune diseases^[2]. CTLA4 is structurally similar to CD28, but binds to CD80 and CD86 with higher affinity, thereby inhibiting T cell activation^[3]. Abatacept can be used to treat moderate to severe rheumatoid arthritis (RA), and is also used to treat psoriatic arthritis and juvenile idiopathic arthritis^[4].

In vitro, Abatacept (0.5, 5, 50 μ M) treatment of human Burkitt lymphoma B (BJAB) wild-type, CD80 KO, and CD86 KO cells for 2h reduced the expression of CD86 and CD80 on the cell surface^[5].

In vivo, subcutaneous administration of Abatacept (10mg/kg, once every 2 days) to DO11.10 RAG-2^{-/-} mice inhibited T cell activation in the draining lymph nodes of mice,

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but did not induce T cell anergy or the generation of Treg cells^[6]. Subcutaneous administration of Abatacept (20mg/kg) to rats with collagen-induced arthritis (CIA) significantly reduced paw edema^[7].

References:

- [1] Douthwaite J, Moisan J, Privezentzev C, et al. A CD80-biased CTLA4-Ig fusion protein with superior in vivo efficacy by simultaneous engineering of affinity, selectivity, stability, and FcRn binding[J]. *The Journal of Immunology*, 2017, 198(1): 528-537.
- [2] Chitale S, Moots R. Abatacept: the first T lymphocyte co-stimulation modulator, for the treatment of rheumatoid arthritis[J]. *Expert opinion on biological therapy*, 2008, 8(1): 115-122.
- [3] da Rosa L C, Scales H E, Benson R A, et al. The effect of abatacept on T-cell activation is not long-lived in vivo[J]. *Discovery Immunology*, 2024, 3(1): kyad029.
- [4] Brunner H I, Wong R, Nys M, et al. Abatacept: a review of the treatment of polyarticular-course juvenile idiopathic arthritis[J]. *Pediatric Drugs*, 2020, 22: 653-672.
- [5] Lorenzetti R, Janowska I, Smulski C R, et al. Abatacept modulates CD80 and CD86 expression and memory formation in human B-cells[J]. *Journal of Autoimmunity*, 2019, 101: 145-152.
- [6] Patakas A, Ji R R, Weir W, et al. Abatacept inhibition of T cell priming in mice by induction of a unique transcriptional profile that reduces their ability to activate antigen-presenting cells[J]. *Arthritis & rheumatology*, 2016, 68(3): 627-638.
- [7] Lon H K, Liu D, DuBois D C, et al. Modeling pharmacokinetics/pharmacodynamics of abatacept and disease progression in collagen-induced arthritic rats: a population approach[J]. *Journal of pharmacokinetics and pharmacodynamics*, 2013, 40: 701-712.

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