

PRODUCT DATA SHEET

AG-20B-0036 15-Oct-2010

anti-CD40 (mouse), mAb (FGK45)

[Tumor Necrosis Factor Receptor Superfamily Member 5; CD40L Receptor]

AG-20B-0036-C100 100 μg AG-20B-0036-C500 500 μg

Clone FGK45

Source/Host Purified from concentrated hybridoma tissue culture supernatant.

Isotype Rat IgG2a

Immunogen Recombinant mouse CD40 fusion protein.

Handling / Storage

Shipping BLUE ICE Short Term Storage +4°C Long Term Storage -20°C

After opening, prepare aliquots and store at -20°C. Avoid freeze/thaw cycles.

Use / Stability

Stable for at least 1 year after receipt when stored at -20°C.

MSDS available at www.adipogen.com or upon request.

Product Specifications

Specificity Recognizes mouse CD40.

Species Crossreactivity Mouse

Application Flow Cytometry

Functional Application: Activates B and NK cells in vivo and in vitro. For in vivo studies

use the preservative free antibody (Prod. No. AG-20B-0036PF).

Purity ≥95% (SDS-PAGE)

Formulation Liquid. In PBS containing 0.02% sodium azide.

Concentration 1mg/ml

Endotoxin Content <0.01EU/µg purified protein (LAL test; Lonza).

Isotype Negative Control Rat IgG2a Isotype Control

Other Product Data

Widely used stimulatory MAb to CD40. Shown to indirectly activate natural killer (NK) cells, producing significant antitumor and antimetastatic effects. Effective in boosting immune responses against infectious agents and can potentially be used to treat chronic autoimmune inflammatory processes.

WARNING: Intended for research use only. This product is not intended or approved for human, diagnostics, therapeutic or veterinary use. Use of this product for human or animal testing is extremely hazardous and may result in disease, severe injury, or death. MATERIAL SAFETY DATA: Review the complete Material Safety Data Sheet before use.

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Product Description

CD40 belongs to the TNF-receptor superfamily and is essential in mediating a broad variety of immune and inflammatory responses including T cell-dependent immunoglobulin class switching, memory B cell development, and germinal center formation. The interaction of CD40-CD40L is necessary for amyloid-beta-induced microglial activation, and thus is thought to be an early event in Alzheimer disease pathogenesis. CD40 is constitutively expressed by antigen presenting cells, including dendritic cells, B cells and macrophages. Consistent with its widespread expression on normal cells, CD40 is also expressed on a wide range of tumor cells, including non-Hodgkin's and Hodgkin's lymphomas, myeloma and some carcinomas including nasopharynx, bladder, cervix, kidney and ovary.

Product Specific References

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- 2. Characterization of immature B cells by a novel monoclonal antibody, by turnover and by mitogen reactivity: A.G. Rolink, et al.; Eur. J. Immunol. **28**, 3738 (1998)
- 3. Anti-CD40 antibody induces antitumor and antimetastatic effects: the role of NK cells: J.G. Turner, et al.; J. Immunol. **166,** 89 (2001)
- 4. Therapeutic activity of agonistic monoclonal antibodies against CD40 in a chronic autoimmune inflammatory process: C. Mauri, et al.; Nat. Med. **6**, 673 (2000)
- 5. Ovarian insufficiency and early pregnancy loss induced by activation of the innate immune system: A. Erlebacher, et al.; J. Clin. Invest. **114**, 39 (2004)
- 6. CD154 is a negative regulator of autoaggressive CD8+ T cells in type 1 diabetes: C.M. McGregor, et al.; PNAS **101**, 9345 (2004)
- 7. IRF-7 is the master regulator of type-I interferon-dependent immune responses: K. Honda, et al.; Nature 434, 772 (2005)
- 8. In vivo and in vitro regulation of type I IFN synthesis by synergistic effects of CD40 and type II IFN: J.A. Greene, et al.; J. Immunol. **176**, 5995 (2006)
- 9. IL-10- and IL-12-independent down-regulation of allergic sensitization by stimulation of CD40 signaling: P.W. Hellings, et al.; J. Immunol. **177**, 5138 (2006)
- 10. Agonistic Anti-CD40 Antibody Profoundly Suppresses the Immune Response to Infection with Lymphocytic Choriomeningitis Virus: C. Bartholdy, et al.; J. Immunol. **178**, 1662 (2007)
- 11. Excessive interferon-α signaling in autoimmunity alters glycosphingolipid processing in B cells: A. Hee-Meng Tan, et al.; J. Autoimmun. in press (2017)

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