CSF1R Antibodies: A Rising Force in Immunology and Neuroscience Research

According to CiteAb, Bio X Cell's Colony-stimulating factor-1 receptor (CSF1R) antibodies have already been cited in over 120 peer-reviewed publications. Strikingly, nine papers featuring these antibodies were published in ***Nature***, ***Science***, and ***Cell*** in the first eight months of this year. This surge reflects both the critical role of CSF1R in biological study and the value of reliable antibody tools for dissecting its function.

Introduction to CSF1R

CSF1R, also known as macrophage colony-stimulating factor receptor (M-CSFR) or cluster of differentiation 115 (CD115), belongs to the type III receptor tyrosine kinases (RTKs), specifically the platelet-derived growth factor receptor (PDGFR) family.

Its natural ligands are CSF1 and IL-34. Ligand binding induces receptor dimerization and autophosphorylation of cytoplasmic tyrosine residues, leading to downstream signaling cascades that modulate gene expression. CSF1R signaling is essential for the survival, proliferation, and differentiation of mononuclear phagocytes across the body, including microglia in the central nervous system.

Abnormal CSF1R expression contributes to a variety of cancers, neurodegenerative diseases, and inflammatory disorders, making it a target of both academic and clinical interest.

Clinical Advances in CSF1R Inhibitors

There are two major clinical strategies for CSF1R inhibition: small-molecule inhibitors and humanized monoclonal antibodies. Both approaches have already led to approved drugs. Among small molecule inhibitors, Daiichi Sankoy's pexidartinib (TURALIO) and Deciphera Pharmaceuticals' vimseltinib (Romvimza) have gained FDA approval for tenosynovial giant cell tumor (TGCT). And Incyte Corporation's axatilimab-csfr (Niktimvo) is the first CSF1R blocking antibody approved by FDA for the treatment of chronic graft-versus-host disease. Several other candidates are in clinical trials for solid tumors, neurodegeneration, and neuroinflammatory conditions.

As monotherapies, CSF1R inhibitors often provide only modest benefits. However, when combined with immunotherapy or chemotherapy, clinical outcomes improve significantly. In addition, small molecule inhibitors often have multi-target effects. For example, pexidartinib inhibits CSF1R as well as KIT and FLT3. Optimizing selectivity

while minimizing toxicity remains a central goal in drug development. Notably, EMA declined approval of pexidartinib for TGCT treatment in Europe due to hepatotoxicity concerns.

Functional Antibodies Empowering CSF1R Research

To support CSF1R research, Bio X Cell provides three anti-mouse CSF1R antibodies:

- ***InVivo*MAb anti-mouse CSF1R (CD115), #BE0213**, clone AFS98
- ***InVivo*Plus anti-mouse CSF1R (CD115), #BP0213**, clone AFS98
- **RecombiMAb anti-mouse CSF1R (CD115), #CP131**, clone AFS98-CP131

Each antibody is produced to Bio X Cell's stringent standards: ultra-pure, low endotoxin, azide-free, and ideal for *in vivo* applications. In addition, Bio X Cell offers an **anti-mouse CSF1 antibody (#BE0204)**, targeting the ligand itself.

In mice, CSF1R is expressed by monocytes/macrophages, peritoneal exudate cells, plasmacytoid and conventional dendritic cells, and osteoclasts. Bio X Cell's anti-CSF1R antibodies are widely used for *in vivo* blockade of CSF1R signaling and depletion of macrophages, providing researchers with precise tools to dissect immune and developmental pathways.

Applications in Recent High-Impact Studies

Vaccine Boosters and Macrophage directing B cell memory (*Cell*, 2025)

The study explored a practical question: should booster vaccines be administered in the same arm as the initial dose? Based on data from mouse models and human volunteers, the researchers demonstrated that same-side boosters generate stronger immune responses.

The results showed that memory B cells (Bmem) in the draining lymph nodes (dLNs) are more likely to re-enter secondary germinal centers (GCs) upon boosting, producing high-affinity and broadly neutralizing antibodies. In contrast, in non-dLNs, Bmems tend to quickly differentiate into plasma cells.

These location-dependent Bmem responses relied heavily on $CD169^{+}$ subcapsular sinus macrophages (SSM). Administration of an anti-CSF1R antibody resulted in significant depletion of SSMs and a redistribution of dLN Bmems away from the subcapsular niche. Moreover, CSF1R blockade before boosting specifically led to an ~80-fold reduction in

antigen-specific B cells in the boosted dLN and a 90-fold decrease in the dLN GC B cells.

Antibody used: Mice were injected intraperitoneally twice, four days apart, with 400 μg of anti-mouse CSF1R (#BE0213) or with rat IgG2a isotype control (#BE0089).

Neuroinvasion versus Neuroprotection during Congenital Zika Virus Infection (*Cell*, 2024)

The study examined the role of fetal mononuclear phagocytes in congenital Zika virus (ZIKV) infection. ZIKV can vertically transmit from mother to fetus, causing fetal neurological infection and microcephaly. Microglia are the first immune cells within the brain parenchyma, derived from $CSF1R^{+}$ primitive macrophages originating in the yolk sac.

Injection of an antibody against CSF1R into the dams effectively depleted fetal microglia and provided near-complete protection of fetal mice from ZIKV infection without affecting viral burden in the dams. This suggested that migrating primitive macrophages act as “Trojan horses” for ZIKV infection.

However, when ZIKV was directly injected into the fetal brain, anti-CSF1R treatment resulted in increased ZIKV detection in the fetuses, supporting a neuroprotective role of brain-resident microglia, contrasting the neuroinvasive role of their precursors during ZIKV dissemination.

Antibody used: Maternal mice were injected intraperitoneally with anti-mouse CSF1R (#BE0213) or isotype control (#BE0089) at E5.5 and E6.5, 3 mg per injection, and followed by ZIKV infection at E7.5 and phenotyping at E10.5. Alternatively, fetuses were infected with ZIKV at E13.5 and analyzed at E16.5. Notably, flow cytometry in this study was also performed using Bio X Cell’s anti-CSF1R by indirect labeling.

Microbes Stimulating β Cell Development (*Science*, 2025)

Pancreatic β cell mass expands rapidly after birth, coincident with gut microbiota diversification. The paper reported a critical neonatal window (P10 to P20 in mice) when microbiota disruption results in lifelong metabolic consequences stemming from reduced β cell development.

The authors found that the host's sensing and signaling of different microbes exhibit diversity. They then focused the research on a specific symbiotic fungus, *Candida*

Dubliniensis, and revealed that it promotes β cell development in a macrophage-dependent manner through distinctive cell wall composition.

To determine macrophage necessity, the authors leveraged three depletion strategies: clodronate liposomes, macrophage Fas-induced apoptosis (MaFIA) mice, and anti-CSF1R antibody. Using Bio X Cell's anti-CSF1R in colonized mice, precisely targeted macrophage depletion led to significantly reduced β cell mass. Compared with chemical or genetic approaches, antibody-mediated depletion offered high specificity, reproducibility, and ease of use.

Antibody used: 600 μg of anti-CSF1R antibody or isotype control was injected on P7, P10, P13.

To Be Continued...

This article is the first part of our introduction to CSF1R research. In Part II, we will focus on oncology-related applications of Bio X Cell's anti-CSF1R antibodies.

Reference

1. El-Gamal, MI, et al (2018) Recent advances of colony stimulating factor-1 receptor (CSF-1R) kinase and its inhibitors. **J Med Chem**. 61(13):5450-5466. doi: 10.1021/acs.jmedchem.7b00873.
2. Peng, L, et al (2025) Recent advances in colony stimulating factor-1 receptor (CSF1R) inhibitors. **Biochem Pharmacol**. 242: 117187. doi: 10.1016/j.bcp.2025.117187.2209-2219.
3. Dhenni, R, et al (2025) Macrophages direct location-dependent recall of B cell memory to vaccination. **Cell**. 188(13):3477-3496. doi: 10.1016/j.cell.2025.04.005.
4. Abdelbasset, M, et al (2024) Differential contributions of fetal mononuclear phagocytes to Zika virus neuroinvasion versus neuroprotection during congenital infection. **Cell**. 187(26):7511-7532. doi: 10.1016/j.cell.2024.10.028.
5. Hill, JH, et al (2025) Neonatal fungi promote lifelong metabolic health through macrophage-dependent β cell development. **Science**. 387(6738):eadn0953. doi: 10.1126/science.adn0953.