Monoclonal antibody

- Overexpression of CDCA2 in Diffuse Large B-Cell Lymphoma Promotes Cell Proliferation and Bortezomib Sensitivity
- Structural Insights into the ADCC Mechanism and Resistance of Mogamulizumab, a First-in-Class Anti-CCR4 Therapy for Cutaneous T Cell Lymphoma
- Hypertrophic Cardiomyopathy and Phenocopies: New Therapies for Old Diseases-Current Evidence and Future Perspectives
- Bispecific Antibodies in Solid Tumors: Advances and Challenges
- Antiamyloid Monoclonal Antibodies in Alzheimer's Disease Part 2: Challenges in Dementia Care Delivery System Logistics
- Review Article: Drug-Induced Liver Injury Associated With Antibody-Based Therapies in Haematologic Malignancies
- Specificity and mechanism of the double-stranded RNA-specific J2 monoclonal antibody
- Tiragolumab and TIGIT: pioneering the next era of cancer immunotherapy

Monoclonal Antibody Classification

Monoclonal antibodies (**mAbs**) can be classified based on their **source, target,** and **mechanism of action**.

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1. Classification by Source (Suffix-Based)

Monoclonal antibodies are named based on their origin:

Suffix	Type	Description	Example
-omab	**Murine**	100% mouse-derived	Tositumomab
-ximab	**Chimeric**	~65% human, ~35% mouse	Infliximab
-zumab	**Humanized**	>90% human, small mouse portion	Trastuzumab
-umab	**Fully human**	100% human-derived	Adalimumab

Note: Murine antibodies (**-omab**) have a higher risk of immunogenic reactions (e.g., HAMA response: human anti-mouse antibody reaction).

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2. Classification by Target

mAbs are categorized based on their primary **target molecule**.

A) Immune System Modulators

- **Anti-TNF-α:** Infliximab, Adalimumab (for autoimmune diseases)
- **Anti-IL-6:** Tocilizumab (for rheumatoid arthritis)
- **Anti-CD20:** Rituximab (for B-cell lymphomas & autoimmune disorders)
- **Anti-IL-17:** Secukinumab (for psoriasis & ankylosing spondylitis)
- **Anti-IL-23:** Guselkumab (for psoriasis)

B) Oncology (Cancer Therapy)

- **Anti-HER2:** Trastuzumab (for HER2+ breast cancer)
- **Anti-VEGF:** Bevacizumab (angiogenesis inhibitor for various cancers)
- **Anti-PD-1:** Pembrolizumab, Nivolumab (immune checkpoint inhibitors)
- **Anti-CTLA-4:** Ipilimumab (checkpoint inhibitor for melanoma)

C) Infectious Diseases

- **Anti-RSV (Respiratory Syncytial Virus):** Palivizumab
- **Anti-Ebola:** Inmazeb (REGN-EB3)

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3. Classification by Mechanism of Action

A) Neutralizing Antibodies

- Bind and block the activity of a target molecule.
- **Example:** Bevacizumab (blocks VEGF to prevent angiogenesis)

B) Cytotoxic Antibodies

- Directly cause cell death via complement-dependent cytotoxicity (**CDC**) or antibody-dependent cell-mediated cytotoxicity (**ADCC**).
- **Example:** Rituximab (targets CD20 on B cells)

C) Immune Checkpoint Inhibitors

- Enhance the immune system's ability to fight cancer by blocking inhibitory pathways.
- **Example:** Nivolumab (**anti-PD-1**), Ipilimumab (**anti-CTLA-4**)

D) Drug-Conjugated Monoclonal Antibodies

- mAbs conjugated to chemotherapy or radioactive substances for targeted delivery.
- **Example:** Brentuximab vedotin (anti-CD30 conjugated to a cytotoxic agent)

Monoclonal antibodies (mAb or moAb) are monospecific antibody that are made by identical immune cells that are all clones of a unique parent cell, in contrast to polyclonal antibodies which are made from several different immune cells. Monoclonal antibodies have monovalent affinity, in that they bind to the same epitope.

Given almost any substance, it is possible to produce monoclonal antibodies that specifically bind to that substance; they can then serve to detect or purify that substance. This has become an important tool in biochemistry, molecular biology, and medicine. When used as medications, the non-proprietary drug name ends in -mab (see “Nomenclature of monoclonal antibodies”), and many immunotherapy specialists use the word mab acronymically.

see Monoclonal antibody therapy.

established a library of monoclonal antibodies (mAbs) against a tumor cell line derived from a patient with GBM. We identified mAbs that reacted with tumor cell lines from patients with GBM but not with nonmalignant human brain cells. We then detected the antigens they recognized using expression cloning. CAR-T cells derived from a candidate mAb were generated and tested in vitro and in vivo.

Results: We detected 507 mAbs that bound to tumor cell lines from patients with GBM. Among them, E61 and A13 reacted with tumor cell lines from most patients with GBM, but not with nonmalignant human brain cells. We found that B7-H3 was the antigen recognized but E61. CAR-T cells were established using the antigen-recognition domain of E61-secreted cytokines and exerted cytotoxicity in co-culture with tumor cells from patients with GBM.

Conclusions: Cancer-specific targets for CAR-T cells were identified using a mAb library raised against primary GBM tumor cells from a patient. We identified a GBM-specific mAb and its antigen. More mAbs against various GBM samples and novel target antigens are expected to be identified using this strategy [1)].

Murine monoclonal antibody

Murine monoclonal antibody

Trastuzumab

Trastuzumab

1)
Nakagawa T, Kijima N, Hasegawa K, Ikeda S, Yaga M, Wibowo T, Tachi T, Kuroda H, Hirayama R, Okita Y, Kinoshita M, Kagawa N, Kanemura Y, Hosen N, Kishima H. Identification of glioblastoma-specific antigens expressed in patient-derived tumor cells as candidate targets for chimeric antigen receptor T cell therapy. Neurooncol Adv. 2022 Nov 15;5(1):vdac177. doi: 10.1093/noajnl/vdac177. PMID: 36601313; PMCID: PMC9798403.