MABS
2026, VOL. 18, NO. 1, 2614669
https://doi.org/10.1080/19420862.2026.2614669

Taylor & Francis
Taylor & Francis Group

Antibodies to watch in 2026

Silvia Crescioli[a], Hélène Kaplon[b], Alicia Chenoweth[c], Yu-Shin Hsu[d], Kieran Pinto[e], Vaishali Kapoor[f], and Janice M. Reichert[g]

[a]Independent Consultant, Business Intelligence, London, UK; [b]Institut Servier d'Innovation Therapeutique, Therapeutic Area Oncology, Gif-Sur-Yvette, France; [c]King's College London, St. John's Institute of Dermatology, School of Basic & Medical Biosciences, London, UK; [d]Beacon Intelligence, Hanson Wade Group, London, UK; [e]Cambridge Healthcare Research, Strategic CI Division, Cambridge, UK; [f]Department of Radiation Oncology, Washington University in St. Louis School of Medicine, St. Louis, MO, USA; [g]Reichert Biotechnology Consulting, Business Intelligence, Framingham, MA, USA

ABSTRACT

The Antibodies to Watch article series provides annual updates on commercial late-stage clinical development, regulatory review, and marketing approvals of antibody therapeutics. Since the first article was published in 2010, the late-stage pipeline has grown from 26 antibody therapeutics to over 200, while during the same time numerous molecules in late-stage studies either transitioned to regulatory review and were approved or were terminated. In this installment of the series, we recap first marketing approvals granted to 19 antibody therapeutics in 2025, discuss 26 molecules currently in regulatory review, including the bispecific antibody-drug conjugate izalontamab brengitecan, and predict which molecules of the 209 currently in the commercial late-stage pipeline might transition to regulatory review by the end of 2026. Most antibody therapeutics in the latter category are for non-cancer indications (16/21, 76%) and have a conventional format (13/21, 62%), but the category also includes numerous antibody-oligo or -drug conjugates, such as delpacibart etedesiran, delpacibart zotadirsen, zeleciment rostudirsen, sonesitatug vedotin, trastuzumab pamirtecan, and ifinatamab deruxtecan, as well as the bispecific petosemtamab. As antibody therapeutics development is a global enterprise, we also discuss trends in annual first approvals granted to antibody therapeutics in any country since 2010, stratified by the antibody's country of origin, documenting the notable increases in the total number of first approvals and those approved first in China. Finally, to benchmark the time typically required for clinical development and regulatory review, we calculated this period for recently approved antibody therapeutic products stratified by their therapeutic area, mechanism of action, format, and country of origin. Our data show that the development and approval period were typically ~6 years, but on average this period was shorter for China-originated products.

ARTICLE HISTORY
Received 5 January 2026
Revised 1 January 2026
Accepted 6 January 2026

KEYWORDS
Antibody therapeutic; antibody-drug conjugate; bispecific; cancer; European Medicines Agency; Food and Drug Administration; immune-mediated disorders; multispecific; National Medical Products Administration

Introduction

In this 17th installment of the annual 'Antibodies to Watch' article series, we report on key events and activities relating to the commercial development of innovative antibody therapeutics, such as first approvals granted in any country in 2025, recent submissions of first marketing applications to any regulatory agency, and company disclosures suggesting that first marketing applications may be submitted by the end of 2026. In keeping with past practice, the data included in Antibodies to Watch in 2026 were collected from public sources primarily during August 1 to December 1, 2025, enabling comparisons with data presented in previous annual reports,[1] which were also collected during the same four-month period of the year before the one in the title. For example, data included in Antibodies to Watch in 2010 were collected in late 2009. Relevant events that were publicly disclosed by December 31, 2025, were included, as possible, in this report. In addition, we analyzed development trends for antibody therapeutics granted marketing approvals since 2010 and evaluated phase lengths for their clinical development and regulatory review periods, thereby providing benchmarks for companies sponsoring molecules currently being evaluated in clinical studies.

CONTACT Janice M. Reichert reichert.biotechconsulting@gmail.com Reichert Biotechnology Consulting, Business Intelligence, Framingham, Massachusetts, USA

Supplemental data for this article can be accessed online at https://doi.org/10.1080/19420862.2026.2614669

For our purposes, an antibody therapeutic is defined as a recombinant protein-based molecule with at least one antigen-binding site derived from an antibody gene that is evaluated as a therapeutic for humans. We thus exclude polyclonal antibodies derived from a natural source, antibody-encoding DNA, Fc only or Fc fusion proteins, diagnostic antibodies, and antibodies evaluated as veterinary medicines from consideration. As we aim to track trends in innovative antibody therapeutics development by companies, biosimilars and molecules developed solely by noncommercial entities are also excluded from consideration. Due to the large volume of literature for the molecules included here, we primarily cite publications and other disclosures made public during 2025 in the summaries below.

Development trends for approved antibody therapeutics

In 1986 the murine antibody muromonab-CD3 (Orthoclone OKT3) became the first monoclonal antibody granted a marketing approval in any country. From this modest beginning, antibody therapeutics development gradually expanded, initially in the US and Europe, leading to additional first marketing approvals in subsequent decades (9 and 21 approvals in the 1990s and 2000s, respectively). Since 2010, however, global expansion in development has enabled substantial increases in these numbers, to 69 first approvals in the 2010s and 99 in just the 5-year period 2020–2024 (Figure 1). Historically, monoclonal antibodies were developed by companies based in the US or Europe, but analysis of product origins shows a notable trend toward development by companies headquartered outside these regions, particularly in China (Figure 1). Interestingly, since 2020, nearly a third of the annual number of approved products originated in China, and from 2024, China-originated molecules have reached and surpassed the number of those originated in the US or Europe. Approximately half of the products first approved in 2025 originated in China (10/19, 53%), while 8 originated in US or Europe and 1 originated in Australia. Supporting this trend, of the 26 antibodies currently in regulatory review in any country, 17 (65%) originated in China and 9 (35%) in the US or Europe. Many of the antibody therapeutics that originated in China have been, or are expected to be, approved in China, as detailed below in the sections about the 2025 approved antibodies and the investigational agents (i.e., those not yet approved in any country) currently in regulatory review.

To further explore development trends for antibody therapeutics that were recently approved, we evaluated phase lengths for their clinical development and regulatory review periods, assessing differences due to the therapeutic area, mechanism of action, format, and country of origin. Only approved antibodies that entered clinical studies after January 1, 2015 were included. Antibodies for infectious diseases were excluded from the analysis to avoid possible biases resulting from the anti-SARS-CoV-2 products, which by necessity had unusually short development periods. Our analysis focused on products approved in the United States, European Union, or China because these comprised the majority, and dates for initiation of the review period were consistently reported and readily available. For each antibody, we calculated both a clinical development phase length and a regulatory review phase length. The clinical phase length was defined as the elapsed time (in years) from either the submission date of the application to initiate clinical

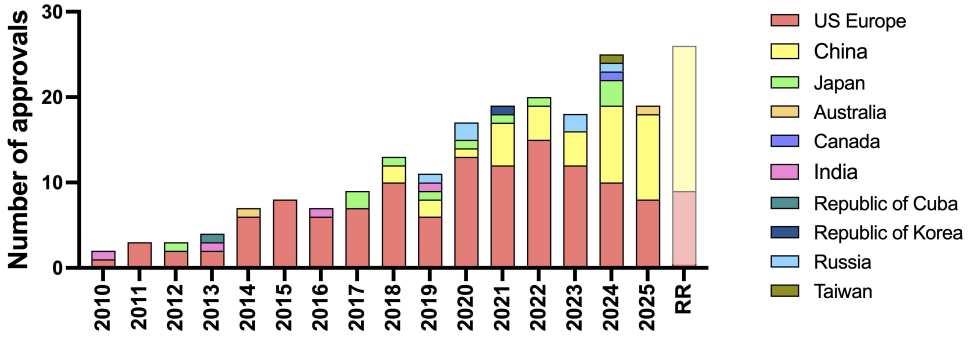

Figure 1. Trends in the origin of approved antibodies. Bar chart represents annual first approvals of antibody therapeutics in any country during 2010-2025 and investigational antibodies in regulatory review, as of December 2025, stratified by the antibody's country of origin. The year was assigned as a molecule's first global approval date; country was assigned according to the headquarters location of the company that originated the molecule. RR, regulatory review.

studies to a regulatory agency (when available) or from initiation of the first-in-human (FIH) study to the submission date of the marketing application that led to first approval. The regulatory review phase length was defined as the elapsed time (in years) from submission of the marketing application leading to first approval to the date of first approval ($n = 57$).

When stratified by therapeutic area, our data show that antibody products for cancer ($n = 41$) have mean total phase length of 5.9 years, which is notably shorter than the mean total phase lengths of antibodies for immune-mediated and inflammatory disorders ($n = 9$), metabolic disorders ($n = 4$) and cardiovascular and hemostasis disorders ($n = 3$) (7.1, 6.7, 7.4 years, respectively) (Figure 2A). This difference is largely attributable to the shorter average clinical phase length of antibodies developed for cancer indications (4.8 years) compared with antibodies for non-cancer indications (immune-mediated and inflammatory disorders (6.1 years), metabolic disorders (5.3 years) and cardiovascular and hemostasis disorders (6.6 years)). These findings are consistent with the shorter average total phase length observed for antibodies for cancer indications relative to non-cancer indications in a larger cohort of 69 antibodies first approved in any country (Supplementary Figure S1A). It should be noted that these results indicate overall trends only, as the phase lengths for products in each cohort were highly variable, and the number of antibodies in individual non-cancer therapeutic areas was limited. In addition, our aim was to evaluate trends for recently approved products, and thus we included only commercially sponsored molecules that entered clinical study after January 1, 2015 and were approved by November 1, 2025. As a consequence, the longest possible phase length for any given molecule was 10 years and 10 months.

As the antibody products for cancer comprise over two-thirds of the total, we performed further analyses on this cohort. First, we investigated whether mean phase lengths differ depending on the antibody format and mechanism of action (Figure 2B). We stratified the molecules into 5 categories: 1) immunomodulatory (i.e., specific for an immunomodulatory target, regardless of the molecular category, including monospecifics, bispecifics, and immunoconjugates; $n = 22$), 2) antibody-drug conjugates (ADCs; $n = 7$), 3) bispecific cell engager ($n = 7$), 4) bispecific TAA (i.e., bispecific antibody targeting tumor-associated antigens (TAA) only; $n = 3$), and 5) monospecific TAA (i.e., monospecific antibody targeting a TAA; $n = 2$). The immunomodulatory antibodies had the shortest mean total phase length (5.4 years) and the shortest clinical phase length (4.1 years), followed by ADCs (total phase length 5.7 years, clinical phase length 4.8 years) and bispecific cell engagers (total phase length 6.3 years, clinical phase length 5.6 years). Interestingly, bispecific and monospecific antibodies targeting TAAs have over 1 year longer average total phase lengths (7.9 and 7.3 years, respectively) and average clinical phase lengths (7.1 and 6.1 years, respectively) compared to those with other mechanisms of action. A similar trend was observed in the analysis of the full cohort of antibodies for cancer indications first approved in any country (Supplementary Figure S1B).

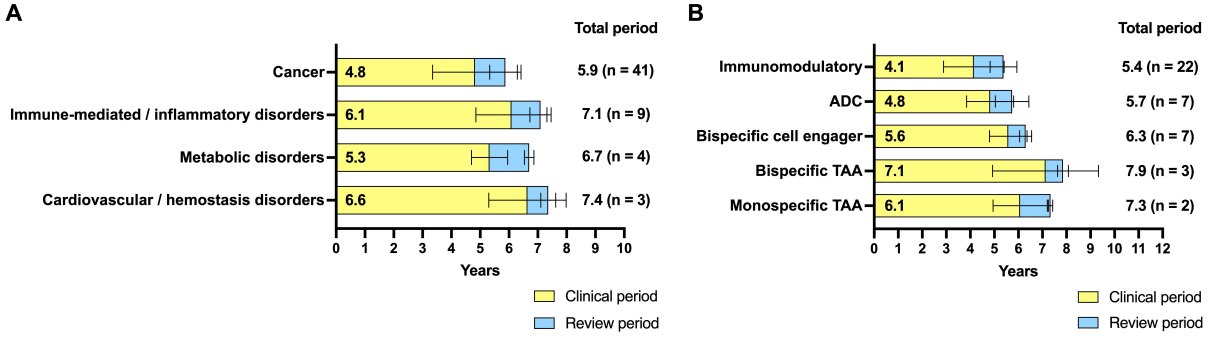

Figure 2. Approved antibody therapeutic development phase lengths by therapeutic area and mechanism of action. Bar charts representing mean phase lengths for antibody therapeutics that entered clinical studies after January 1, 2015 and were approved in the US, EU or China as of November 1, 2025. (A) Bar chart shows data stratified by therapeutic area ($n = 57$). (B) Bar chart shows data stratified by mechanism of action ($n = 41$). Mechanisms of action are categorized as: 1) immunomodulatory (i.e., specific for an immunomodulatory target, regardless of the molecular category, including monospecifics, bispecifics, and immunoconjugates), 2) antibody-drug conjugates (ADCs), 3) bispecific cell engager, 4) bispecific TAA (i.e., bispecific antibody targeting tumor-associated antigens (TAA) only), and 5) monospecific TAA (i.e., monospecific antibody targeting a TAA). Error bars represent standard deviation.

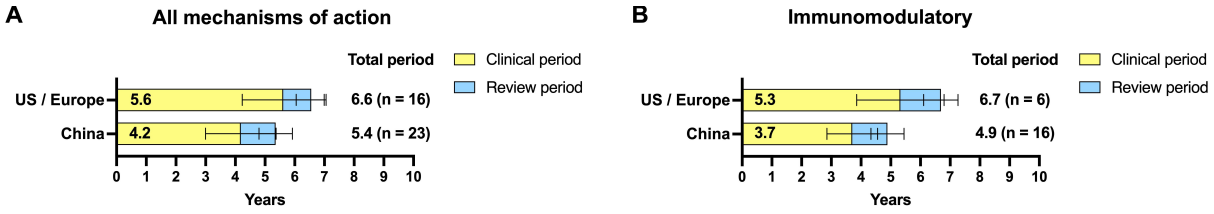

Figure 3. Development phase lengths by country or region of origin for approved anti-cancer antibody therapeutics. Bar charts representing mean phase lengths for antibody therapeutics that originated in the US, Europe or China, entered clinical studies after January 1, 2015, and were approved for cancer indications in any country by November 1, 2025. (A) All mechanisms of action are included (US/Europe, $n = 16$; China, $n = 23$); (B) Only immunomodulatory antibody products are included (US/Europe, $n = 6$; China, $n = 16$). Immunomodulatory refers to products that are specific for an immunomodulatory target, regardless of the molecular category, including monospecifics, bispecifics, and immunoconjugates. Europe includes countries in the European Economic Area, United Kingdom, and Switzerland. Error bars represent standard deviation.

We next evaluated whether our data for antibody products for cancer revealed differences depending on the country or region of origin, with a particular focus on products originating in the US, Europe, or China (Figures 3). Our interest was prompted by the well-documented growth of China's pharmaceutical industry, and particularly their focus on the development of innovative drugs such as bi- and multi-specific antibodies and ADCs, over the past decade.[2–4] This growth resulted from numerous reforms introduced in China that aimed to reduce development cycles and streamline drug approval pathways, which might be expected to shorten development phase lengths. Our data show that, on average, China-originated products had shorter clinical phase lengths (4.2 vs 5.6 years), but marginally longer review lengths (1.2 vs 1 years), resulting in shorter total development periods (5.4 vs 6.6 years) (Figure 3A). Given the higher representation of immunomodulatory antibodies among China-originated molecules (16/23, 70%) compared with molecules originating in the United States or Europe (6/16, 38%), we additionally analyzed phase lengths within this subgroup to mitigate potential bias arising from differences in the mechanisms of action of products that originated in the two regions (Figure 3B). Our results show that approved immunomodulatory agents originated in China have a comparable average review length, but over 1 year shorter average clinical phase length (3.7 years) compared to those that originated in the US or Europe (5.3 years) (Figure 3B). Together, these findings suggest that the shorter development timelines observed for China-originated antibodies are not attributable to differences in regulatory review timelines, but instead reflect shorter clinical development phases that are not solely explained by differences in the product's mechanism of action.

Of the 46 therapeutic antibodies for cancer indications that entered clinical study after January 1, 2015 and were approved in any country or region by November 1, 2025, 22 (48%), 19 (41%), and 5 (11%) originated in China, the US or Europe, or the rest of the world, respectively. To provide fine detail with respect to the phase lengths for these products, we stratified the molecules by format and mechanism of action, and annotated each product with the country or region of origin, international nonproprietary name, target (s), and, where relevant, type of payload (Figure 4). We found that approximately two-thirds of the ADCs and monospecific antibody products originated in China, while most (10/13, 77%) of the bispecifics originated in the US or Europe. Interestingly, most (16/24, 67%) of antibodies with immunomodulatory properties, either monospecific or bispecific, originated in China, and 13 of the 16 (81%) have phase lengths of around 5 years or less.

Clinical and regulatory review phase length details for the molecules represented in Figure 4, stratified by regulatory agency of first approval, are shown in Supplementary Table S1. It is important to note that the elapsed time from the start of clinical studies to marketing approval for any given product can be affected by numerous factors, including ones that are not specifically related to the molecule. Difficulty with patient enrollment, manufacturing issues, and lack of funding, for example, may cause delays, while timely advice from regulatory agencies could speed the process. Phase length ranges for these approved products were broad, reflecting the multifactorial nature of the process. For example, ranges for the clinical and review phases for products first approved in China were 2–6.9 and 0.6–3.1 years, respectively, and 4–9.1 and 0.3–1.9 years,

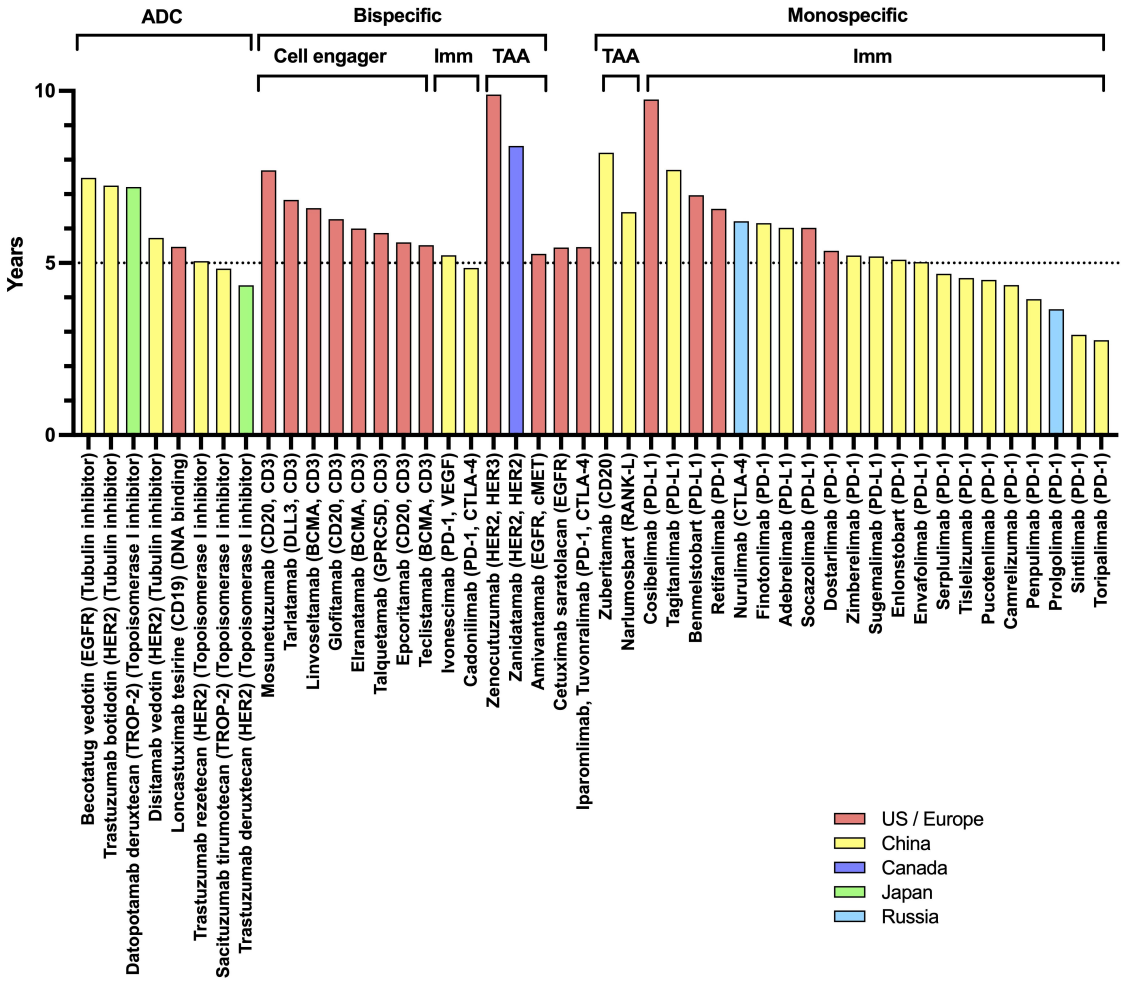

Figure 4. Comparison of anti-cancer antibody therapeutic total development phase lengths. Bar chart representing total (i.e., clinical and review) phase lengths for 46 antibody therapeutics that entered clinical studies after January 1, 2015 and were approved for cancer indications in any country as of November 1, 2025. Each bar represents data for one antibody product. Bars are grouped by format and mechanism of action and ordered according to the phase length. Note: Two antibody products, the antibody mixture iparomlimab, tuvonralimab (齐倍安®) and the photoimmunotherapy immunoconjugate cetuximab saratolacan (Akalux®), have unique format and mechanism of action compared to other products in the cohort and were thus not included in any group. Bars are colored according to the molecule's country or region of origin and annotated with the international nonproprietary name, target(s), and, where applicable, type of ADC payload. Europe includes countries in the European Economic Area, United Kingdom, and Switzerland. Abbreviations: ADC, antibody-drug conjugate; Imm, immunomodulatory; TAA, tumor-associated antigen.

respectively, for those first approved in the US. The phase lengths given here thus should not be used as measures for future development of specific molecules, even those targeting the same antigens. Our intention is to set expectations for these phase lengths, not to predict the time that might be required to develop an antibody currently in clinical studies.

Antibody therapeutics granted a first approval in any country in 2025

Based on data publicly available as of December 31, a total of 19 antibody therapeutics were granted a first approval in at least one country or region during 2025 (Table 1). Of these, nearly two-thirds (12/19, 63%) were for non-cancer indications, and slightly more than half (10/19, 53%) were first approved in China. These antibody therapeutics are described in chronical order below according to the first approval date, with approvals granted outside of China first, followed by those approved in China.

Table 1. Commercially sponsored monoclonal antibody therapeutics granted a first approval in any country during 2025. *Table includes information found in the public domain as of December 31, 2025; molecules are listed in chronological order according to the first approval date. Abbreviations: ADC, antibody-drug conjugate; APRIL, a proliferation inducing ligand; BCMA, B-cell maturation antigen; EGFR, epidermal growth factor receptor; EU, European Union; HER2, human epidermal growth factor receptor 2; IL, interleukin; PCSK9, proprotein convertase subtilisin/kexin type 9; PD-1, programmed cell death protein-1; RSV, respiratory syncytial virus; VEGF, vascular endothelial growth factor. Supplemental table S3 (excel format) includes all data in Tables 1-4.

INN (Brand name)	Target(s); Format	Indication of first approval	Country/region of first approval in 2025*
Garadacimab (ANDEMBRY®)	Factor XIIa; Human IgG4λ	Prevention of hereditary angioedema attacks in adult and pediatric patients aged 12 years and older.	Australia
Vilobelimab (Gohibic)	Complement C5a; Chimeric IgG4κ	SARS-CoV-2-induced acute respiratory distress syndrome	EU
Linvoseltamab (Lynozyfic)	BCMA, CD3; Human IgG4κ; Bispecific	Multiple myeloma	EU
Nipocalimab (IMAAVY)	FcRn; Human IgG1λ	Generalized myasthenia gravis	US
Telisotuzumab vedotin (Emrelis)	cMET; Humanized IgG1κ; ADC	Non-small cell lung cancer	US
Clesrovimab (Enflonsia)	RSV; Human IgG1κ	Prevention of RSV lower respiratory tract disease in neonates (newborns) and infants who are born during or entering their first RSV season	US
Sibeprenlimab (VOYXACT)	APRIL; Humanized IgG2κ	Reduction of proteinuria in adults with primary immunoglobulin A nephropathy	US
Depemokimab (Exdensur)	IL-5; Humanized IgG1κ	Asthma, chronic rhinosinusitis with nasal polyps	UK
Narsoplimab (YARTEMLEA®)	MASP-2; Human IgG4λ	Hematopoietic stem cell transplant-associated thrombotic microangiopathy	US
Recaticimab (艾心安)	PCSK9; Humanized IgG1κ	Hypercholesterolemia	China
Finotonlimab (Yasuhei)	PD-1; Humanized IgG4κ	Head and neck squamous cell carcinoma	China
Siltartoxatug (新替妥, Sintetol®)	Tetanus toxin; Human IgG1κ	Prevention of tetanus	China
Ebdarokimab (爱达罗®)	IL-12/23 p40; Human IgG1κ	Plaque psoriasis	China
Trastuzumab rezetecan (艾维达®)	HER2; Humanized IgG1κ; ADC	Non-small cell lung cancer	China
Suvemcitug (ENZESHU)	VEGF; Humanized IgG1κ	Ovarian cancer	China
Firsekibart (金赛新, Jin Beixin®)	IL-1 beta; Human IgG4λ2	Gouty arthritis	China
Trastuzumab botidotin (舒泰莱®)	HER2; Humanized IgG1κ; ADC	Breast cancer	China
Becotatug vedotin (MEIYOUHENG)	EGFR; Humanized IgG1κ; ADC	Nasopharyngeal cancer	China
Picankibart (PECONDLE®)	IL-23p19; Humanized IgG1κ	Plaque psoriasis	China

First marketing approvals granted in Australia, Europe, or the US in 2025

Garadacimab (CSL Ltd.)

Garadacimab (garadacimab-gxii, ANDEMBRY®) is a human IgG4λ monoclonal antibody targeting Factor XIIa (FXIIa), developed for the treatment of hereditary angioedema (HAE). Its hinge region includes an S228P mutation that stabilizes the antibody, minimizing half-antibody formation and antigen-binding fragment (Fab)-arm exchange with endogenous IgG4. Garadacimab's unique, long CDR-H3 loop binds to the β-chain of FXIIa, effectively inhibiting its proteolytic activity and blocking downstream procoagulant and proinflammatory pathways.[5]

In January 2025, garadacimab received its first approval in Australia for the prevention of HAE attacks in adult and adolescent patients aged 12 years and above, and it was subsequently approved in the United Kingdom, EU, Japan, Switzerland, US, and Canada.[6] The recommended dosage is: 1) an initial loading dose of 400 mg garadacimab (two 200 mg injections) administered subcutaneously (SC) on the first treatment day; then 2) maintenance doses of 200 mg SC once monthly.

The approvals are based on results from the Phase 3 VANGUARD trial (NCT04656418), a randomized, double-blind, placebo-controlled, multicenter study designed to evaluate the efficacy and safety of garadacimab. Patients were randomly assigned to receive garadacimab (SC, 400 mg loading dose delivered as two 200 mg injections) or volume-matched placebo on Day 1 of the treatment period, followed by five additional self- or caregiver-administered monthly SC doses of 200 mg garadacimab or volume-matched placebo. Monthly administration of garadacimab significantly reduced the frequency of HAE attacks by 87% compared with placebo (0.27 vs. 2.01 attacks per month; $p < 0.0001$) in patients aged 12 years and older. The treatment was well tolerated, with mild adverse events, including upper-respiratory infections and headaches, and showed no increased risk of bleeding or thromboembolic events.[7] An interim analysis of the ongoing open-label extension study (median garadacimab exposure of 13.8 months) showed that it has a favorable long-term safety profile and provides sustained reductions in HAE attacks.[8] In the pivotal trial and the open-label extension study (NCT04739059), injection-site reactions (e.g., injection-site bruising, injection-site erythema, injection-site hematoma, injection-site pruritus, injection-site urticaria) were reported in 23 (14%) patients.

Vilobelimab (InflaRx N.V.)

Vilobelimab (Gohibic®) is a chimeric IgG4κ antibody that binds the soluble human complement split product, C5a, and thereby blocks complement system-mediated inflammatory responses. In January 2025, Gohibic received marketing authorization under exceptional circumstances in the European Union (EU) for the treatment of adult patients with SARS-CoV2-induced acute respiratory distress syndrome (ARDS) who are receiving systemic corticosteroids as part of standard of care (SoC) and receiving invasive mechanical ventilation (IMV) with or without extracorporeal membrane oxygenation (ECMO).[9,10] The authorization is valid throughout the EU as well as Iceland, Liechtenstein, and Norway. Vilobelimab has not been approved in the US but, in April 2023, the FDA issued an Emergency Use Authorization for vilobelimab for the treatment of COVID-19 in hospitalized adults when initiated within 48 hours of receiving IMV or ECMO.

The recommended dose of vilobelimab is 800 mg administered intravenously (IV), for a maximum of 6 doses over the treatment period. Treatment should be started within 48 hours of intubation (Day 1) followed by administration on Days 2, 4, 8, 15, and 22 if the patient is hospitalized.

The marketing authorization was based on results of the Phase 2/3 PANAMO trial (NCT04333420), which compared the effects of vilobelimab plus SoC to placebo plus SoC for the treatment of invasively mechanically ventilated patients with COVID-19. Eligible patients were randomized (1:1) to receive SoC and vilobelimab (800 mg IV) for a maximum of six doses (days 1, 2, 4, 8, 15, and 22) or SoC and a matching placebo. In the global data set, patients treated with vilobelimab had improved survival with a relative reduction in 28-day all-cause mortality of 23.9% compared to those who received placebo.[9,10]

Linvoseltamab (Regeneron Pharmaceuticals, Inc.)

Linvoseltamab (linvoseltamab-gcpt, Lynozyfic®) is a T cell-engaging, human bispecific IgG4κ antibody that targets B-cell maturation antigen (BCMA) and CD3. The antibody, which is hinge-stabilized and designed to have reduced Fc effector functions, was developed by Regeneron as a treatment for multiple myeloma (MM). In April 2025, linvoseltamab received a conditional marketing authorization valid throughout the EU as monotherapy for the treatment of adult patients with relapsed and refractory MM who have received at least 3 prior therapies, including a proteasome inhibitor, an immunomodulatory agent, and an anti-CD38 monoclonal antibody, and have demonstrated disease progression on the last therapy.[11] The US Food and Drug Administration (FDA) granted an accelerated approval for linvoseltamab to treat adult patients with relapsed or refractory (R/R) MM who have received at least 4 prior lines of therapy, including a proteasome inhibitor, an immunomodulatory agent, and an anti-CD38 monoclonal antibody, in July 2025.[12]

The recommended dosing regimen for linvoseltamab includes step-up IV dosing (5 mg, 25 mg, 200 mg) over Weeks 1–3, followed by 200 mg IV once every week (Q1W) doses if doses are tolerated. Patients receive pretreatment therapy to manage severe reactions such as cytokine release syndrome and infusion-related reactions. Every four weeks (Q4W) dosing of linvoseltamab may be considered for patients who have

received at least 17 doses of 200 mg and have confirmed response of very good partial response or better per international myeloma working group (IMWG) criteria at or after Week 24.[11]

The approvals were supported by results from the FIH, open-label, pivotal Phase 1/2 LINKER-MM1 trial (NCT03761108) in patients with relapsed or refractory MM, including 80 patients who received at least 4 lines of therapy that included a proteasome Inhibitor, an immunomodulatory agent, and an anti-CD38 antibody. In the dose-escalation phase of the study, patients were treated with doses ranging from 3 mg to 800 mg. Two dose level were subsequently evaluated: 50 mg ($n = 104$) and 200 mg ($n = 105$). With median follow-up of 21.7 months, the 200 mg dose cohort of patients who received at least 4 lines of therapy had a median DOR of 21.0 months (95% CI: 19.0, NE). The estimated DOR rate was 92.7% (95% CI: 81.8, 97.2) at 6 months, 90.9% (95% CI: 79.4, 96.1) at 9 months, and 79.4% (95% CI: 65.9, 88.1) at 12 months.[13]

Linvoseltamab is being evaluated in an extensive clinical development program investigating its potential as both a monotherapy and in combination regimens across multiple lines of therapy in MM, including earlier treatment settings, MM precursor states, and other plasma cell disorders. Ongoing studies include the Phase 3 confirmatory LINKER-MM3 trial (NCT05730036) comparing linvoseltamab monotherapy with the combination of elotuzumab, pomalidomide, and dexamethasone in R/R MM, anticipated to complete in 2033.

Nipocalimab (Johnson & Johnson)

Nipocalimab (nipocalimab-aahu, IMAAVY®) is a human aglycosylated (N297A) IgG1λ antibody that blocks neonatal Fc receptor (FcRn) recycling, thereby reducing circulating IgG levels including those of pathogenic IgG antibodies, without detectable effects on other immune functions.[14] Johnson & Johnson has developed nipocalimab as a treatment for generalized myasthenia gravis (gMG) and hemolytic disease of the fetus and newborn (HDFN), as well as other diseases that are driven by the presence of certain pathogenic IgGs.

In April 2025, FDA approved nipocalimab for adult and pediatric gMG patients (12 years of age and older) who are anti-acetylcholine receptor (AChR) or anti-muscle-specific kinase (MuSK) antibody positive.[15] On December 1, 2025, Johnson & Johnson announced that the European Commission (EC) approved nipocalimab for the treatment of adult and pediatric gMG patients (aged 12 years and older) who are anti-AChR or anti-MuSK antibody positive. IMAAVY has also been approved in Brazil for anti-AChR, anti-MuSK or anti-LRP4 antibody positive adults and pediatric patients aged 12 and older and in Japan for the treatment of all patients living with gMG.[16]

The recommended initial dosage of nipocalimab is 30 mg/kg once via IV infusion. Two weeks after the initial dosage, a maintenance dosage of 15 mg/kg via IV infusion is administered, and Q2W thereafter.

FDA's approval was based in part on results from the 24-week, double-blind, placebo-controlled Phase 3 Vivacity-MG3 trial (NCT04951622). In this study, 196 gMP patients adults who were anti-AChR or anti-MuSK antibody positive were randomized 1:1 to receive SOC and nipocalimab (30 mg/kg IV loading dose followed by 15 mg/kg Q2W) or placebo. Mean change from baseline to Weeks 22, 23, and 24 (primary efficacy endpoint) in the Myasthenia Gravis-Activities of Daily Living (MG-ADL) total score was −4.7 and −3.3 in the nipocalimab and placebo arms, respectively, which was a statistically significant difference favoring IMAAVY ($p = 0.002$).[17]

Nipocalimab has been granted numerous regulatory agency designations designed to facilitate its development, including FDA's Fast Track designations for HDFN, warm autoimmune hemolytic anemia, gMG, fetal neonatal alloimmune thrombocytopenia, and Sjögren's disease, as well as Breakthrough Therapy designations for HDFN and for Sjögren's disease and Orphan Drug designations for warm autoimmune hemolytic anemia, HDFN, gMG, chronic inflammatory demyelinating polyneuropathy, and fetal neonatal alloimmune thrombocytopenia. In addition, EMA granted Orphan Medicinal Product designation to nipocalimab for HDFN.[18]

The efficacy and safety of nipocalimab are also being evaluated in late-stage clinical studies of: 1) children with gMG (Phase 2/3 NCT05265273 study); 2) adults with pregnancies at risk for HDFN (Phase 3 AZALEA trial (NCT05912517)); 3) patients with warm autoimmune hemolytic anemia (Phase 2/3 ENERGY study (NCT04119050)); 4) adults with chronic inflammatory demyelinating polyneuropathy (Phase 2/3 NCT05327114 study); 5) adults with pregnancies at risk of fetal and neonatal alloimmune thrombocytopenia (Phase 3 FREESIA-3 study (NCT06533098), Phase 3 FREESIA-1 study (NCT06449651)); and 6)

participants with moderate to severe Sjogren's disease (Phase 3 DAFFODIL, NCT06741969). As of November 2025, these studies are recruiting participants. An additional Phase 3 study of adult gMG patients (EPIC, NCT07217587) due to start in November 2025 will evaluate whether treatment with nipocalimab provides superior disease control versus efgartigimod in FcRn blocker-naive patients. The study also includes a treatment-switch arm to assess efficacy and safety of nipocalimab in participants switching from efgartigimod to nipocalimab.[18]

Telisotuzumab vedotin (AbbVie)

Telisotuzumab vedotin (telisotuzumab vedotin-tllv, Emrelis®) is an ADC composed of an anti-cMET humanized IgG1κ antibody conjugated to MMAE via a cleavable valine-citrulline linker.

In May 2025, FDA granted accelerated approval to telisotuzumab vedotin for adults with previously treated, locally advanced or metastatic non-squamous non – small cell lung cancer (NSCLC) exhibiting high c-Met protein overexpression (≥50% of tumor cells with strong [3+] staining), as determined by an FDA-approved test.[19] FDA had granted telisotuzumab vedotin Breakthrough Therapy designation for this indication and the BLA for telisotuzumab vedotin received a Priority review.

The recommended dose is 1.9 mg/kg (up to a maximum of 190 mg for patients ≥100 kg), as an IV infusion Q2W, until disease progression or unacceptable toxicity.

FDA's approval was based in part on the results of the open-label, single-arm Phase 2 LUMINOSITY trial (NCT03539536), which included 84 patients, with epidermal growth factor receptor (EGFR) wild-type, non-squamous NSCLC with high c-Met protein overexpression who had received prior systemic therapy, who were administered telisotuzumab vedotin 1.9 mg/kg IV Q2W. The primary outcome measures were confirmed overall response rate (ORR) and duration of response (DOR), determined by blinded independent central review (BICR) according to Response Evaluation Criteria in Solid Tumors (RECIST) 1.1. The ORR was 35% (95% CI: 24, 46) and median DOR was 7.2 months (95% CI: 4.2, 12).[19]

The confirmatory randomized, open-label Phase 3 TeliMET NSCLC-01 trial (NCT04928846) is evaluating the effects of telisotuzumab vedotin versus docetaxel in patients with previously treated cMET overexpressing, EGFR wildtype, locally advanced/metastatic non-squamous NSCLC. An estimated 698 participants will receive IV telisotuzumab vedotin Q2W or docetaxel Q3W until study drug discontinuation criteria are met. The study's primary completion date is in March 2028.

Clesrovimab (Merck)

Clesrovimab (clesrovimab-cfor, Enflonsia®) is a human IgG1κ monoclonal antibody that targets the respiratory syncytial virus (RSV) F protein, which is essential for viral entry into host cells. The antibody includes YTE mutations (M252Y, S254T, T256E) for half-life extension. In June 2025, FDA approved clesrovimab for the prevention of RSV-associated lower respiratory tract disease in newborns and infants who are born during or entering their first RSV season.[20] In September 2025, the EMA issued a positive opinion, recommending the granting of a marketing authorization for Enflonsia for prevention of RSV lower respiratory tract disease in neonates and infants during their first RSV season. However, in November 2025, the applicant informed the Agency that a revised dataset will be submitted. The EC decision process is on hold until the new dataset is received.[21]

The recommended dose is 105 mg clesrovimab administered as a single intramuscular (IM) injection.

FDA's approval was based in part on the results from the double-blind, randomized Phase 2b/3 CLEVER clinical trial (MK-1654–004, NCT04767373) comparing the safety and efficacy of a single 105 mg dose of clesrovimab administered IM vs placebo in preterm and full-term infants born at ≥29 weeks gestational age from birth up to 1 year entering their first RSV season. The primary endpoints were the incidence of RSV-associated medically attended lower respiratory infection (MALRI) requiring ≥1 indicator of LRI or severity through 150 days after dosing and hospitalization. Of 2411 participants who received clesrovimab, 60 developed MALRI and 9 were hospitalized vs 74 and 28, respectively, of those in the placebo arm (MALRI efficacy, 60.5%; hospitalization efficacy, 84.3%).[22]

The approval was also supported by results from the Phase 3 SMART MK-1654–007 trial (NCT04938830). This was a randomized, partially-blind, palivizumab-controlled, multi-site trial to evaluate

the safety and efficacy of clesrovimab in infants at increased risk of severe RSV disease, including early (< 29 weeks gestational age) or moderate preterm infants (≥29 to ≤35 weeks gestational age) and infants with chronic lung disease of prematurity or congenital heart disease of any gestational age. Participants were randomized 1:1 to receive clesrovimab ($N = 446$, single 105 mg dose on Day 1 followed by a dose of placebo one month later) or palivizumab ($N = 450$, 5 mg/kg administered on Day 1 and every month thereafter for a total of 3 to 5 doses) by IM injection. The incidence rates of RSV-associated MALRI and hospitalizations through 150 days after dosing were comparable between the clesrovimab vs palivizumab study arms, (3.6% vs 2.9% and 1.3% vs 1.5%, respectively).[22]

Sibeprenlimab (Otsuka Pharmaceutical, Co. Ltd.)

Sibeprenlimab (sibeprenlimab-szsi, VOYXACT®) is a humanized IgG2κ monoclonal antibody that neutralizes APRIL (a proliferation-inducing ligand, TNFSF13), a cytokine driving abnormal IgA production in immunoglobulin A nephropathy (IgAN). Sibeprenlimab was created and initially developed by Visterra, Inc., which was acquired by Otsuka in 2018. FDA granted sibeprenlimab Breakthrough Therapy designation as a treatment for IgAN. On November 25, 2025, FDA granted an accelerated approval for sibeprenlimab, in a single-dose prefilled syringe for SC injection intended for self-administration Q4W, for reduction of proteinuria in adults with primary IgAN.[23]

The approval was supported by data from the Phase 2 ENVISION and Phase 3 VISIONARY trials. The Phase 2 ENVISION trial (NCT04287985) was a randomized, double-blind study of 155 adults with IgAN and persistent proteinuria despite optimized supportive care who were administered sibeprenlimab (IV; 2, 4 or 8 mg/kg doses) or placebo.[24] After 12 months of treatment, sibeprenlimab produced a dose-dependent geometric mean reduction in the 24-hour urinary protein-to-creatinine ratio (uPCR) (the primary end point) of 47.2 ± 8.2% (2 mg doses) to 62.0 ± 5.7% (8 mg dose) compared with 20.0 ± 12.6% for those who received placebo. The therapy was generally well tolerated.

The Phase 3 VISIONARY trial (NCT05248646) enrolled more than 500 patients with IgAN worldwide and evaluated monthly doses of SC sibeprenlimab (400 mg) versus placebo and SoC. Otsuka announced results of a pre-specified interim analysis in June 2025. The study met its primary endpoint, showing a 51.2% reduction in proteinuria (as measured by 24-hour uPCR) at nine months compared with placebo, with a safety profile consistent with earlier studies.[25]

Depemokimab (GSK plc)

Depemokimab (depemokimab-ulaa, Exdensur) is an ultra-long-acting, humanized IgG1κ monoclonal antibody targeting interleukin-5, developed by GSK for patients with severe eosinophilic asthma and chronic rhinosinusitis with nasal polyps (CRSwNP). IL-5 is a key cytokine in type 2 inflammation, present in the majority of patients with difficult to treat asthma, and in patients with CRSwNP experiencing more severe disease and symptoms. The antibody was engineered for high affinity to IL-5 and extended half-life (M252Y, S254T, T256E), allowing SC dosing every six months.

On December 15, 2025, GSK announced that the UK's Medicines and Healthcare products Regulatory Agency granted a marketing authorization of Exdensur for two indications: 1) as an add-on maintenance treatment of asthma in adult and adolescent patients aged 12 years and older with type 2 inflammation characterized by an eosinophilic phenotype who are inadequately controlled on maximum moderate-dose or high-dose inhaled corticosteroids plus another asthma controller, and 2) as an add-on therapy with intranasal corticosteroids for the treatment of adult patients with severe CRSwNP for whom therapy with systemic corticosteroids and/or surgery do not provide adequate control.[26] FDA subsequently approved Exdensur (depemokimab-ulaa) for as an add-on maintenance treatment of severe asthma characterized by an eosinophilic phenotype in adult and pediatric patients aged 12 years and older. EMA adopted a positive opinion in December 2025, recommending that the EC grant a marketing authorization for depemokimab for severe eosinophilic asthma and for severe CRSwNP. Approval in the EU is anticipated in the first quarter of 2026. Marketing applications for these indications are also under review in Canada, China, and Japan.

The approvals for asthma and CRSwNP indications are based in part on data from the Phase 3 SWIFT and ANCHOR studies, respectively. The Phase 3 SWIFT-1 (NCT04719832) and SWIFT-2 (NCT04718103)

trials tested 100 mg depemokimab every six months vs placebo on top of the SoC over 52 weeks for patients with severe asthma associated with an eosinophilic phenotype. Data showed that the annualized exacerbation rate was 0.46 with depemokimab versus 1.11 with placebo in the SWITF-1 trial. In SWIFT-2, the corresponding rates were 0.56 vs 1.08. Depemokimab was well tolerated with a profile similar to the placebo arm.[27]

The replicate Phase 3 ANCHOR-1 (NCT05274750) and ANCHOR-2 (NCT05281523) trials of CRSwNP patients evaluated 100 mg depemokimab every six months vs placebo over 52 weeks. Both studies met their co-primary endpoints, showing significant improvements in total endoscopic nasal polyp score and nasal obstruction score between Weeks 49 and 52. Treatment was generally well tolerated.[28]

Narsoplimab (Omeros Corporation)

Narsoplimab (narsoplimab-wuug, YARTEMLEA®) is a human, hinge-stabilized (S228P), IgG4λ antibody that targets mannan-binding lectin-associated serine protease-2 in the lectin complement pathway. Omeros is developing it for the treatment of hematopoietic stem cell transplant-associated thrombotic microangiopathy (TA-TMA). FDA granted narsoplimab Breakthrough Therapy and Orphan Drug designations for TA-TMA and Orphan Drug status for the prevention (inhibition) of complement-mediated thrombotic microangiopathies, while EMA has granted Orphan Medicinal Product designation for narsoplimab in TA-TMA.

In December 2025, FDA approved YARTEMLEA® for the treatment of TA-TMA.[29] Omeros submitted an MAA for narsoplimab for TA-TMA to EMA, and an opinion is expected in mid-2026.[30] The data supporting FDA's approval and the MAA are from a Phase 2 study (NCT02222545, OMS721-TMA-001) in which 28 adults with high-risk TA-TMA received narsoplimab. In this cohort, 68% were alive at 100 days after TA-TMA diagnosis, compared with historical survival rates below 20%, and about 53% survived at one year. Moreover, an updated analysis versus an external registry cohort of more than 100 patients who were not treated with narsoplimab showed a hazard ratio for overall survival (OS) of 0.32 ($p < 0.00001$) in favor of narsoplimab.[31]

In addition, an expanded access program (NCT04247906) enrolled more than 130 adults and children with severe or refractory TA-TMA. Reported one-year survival in children given narsoplimab as front-line or second-line therapy was 75% and 56%, respectively. In adults, one-year survival was 58% for front-line therapy and 41% when given second line or later.[32] In adult and pediatric patients who had failed prior therapy, including C5 inhibitors, the one-year survival was 41% and 47%, respectively, far higher than the historical survival rate of less than 20%.[33] Across this real-world cohort, hazard ratios for survival compared with external control ranged from 0.34 to 0.46, all statistically significant. Safety findings were consistent with the pivotal trial, and no new safety concerns emerged.

First marketing approvals granted in China 2025

Recaticimab (Jiangsu HengRui Medicine Co. Ltd)

Recaticimab (SHR1209, AiXinAn®, 艾心安®) is a humanized IgG1κ monoclonal antibody that targets proprotein convertase subtilisin/kexin type 9 (PCSK9) developed by Hengrui Medicine. The antibody is Fc engineered for half-life extension (M252Y, S254T, T256E) and is designed to bind PCSK9 and prevent its interaction with LDL receptor (LDLR) and LDLR lysosomal degradation, thus increasing LDLR recycling and low-density lipoprotein cholesterol (LDL-C) clearance from the bloodstream.[34,35]

In January 2025, China's National Medicinal Products Administration (NMPA) approved recaticimab in combination with statins, or in combination with statins and other lipid-lowering therapies for adult patients with primary hypercholesterolemia (including heterozygous familial and non-familial hypercholesterolemia) and mixed dyslipidemia who, despite treatment with moderate or higher doses of statins, still fail to reach the target level of low-density lipoprotein cholesterol (LDL-C).[34] Recaticimab was also approved for use as monotherapy in adult patients with non-familial hypercholesterolemia and mixed dyslipidemia to reduce the levels of low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), and apolipoprotein B (ApoB).

Phase 3 trial (REMAIN-1, REMAIN-2, REMAIN-3) results supported the marketing approvals of recaticimab in these indications. Conducted in China, the REMAIN-1 Phase 3 study (NCT04849000) assessed the efficacy and safety of recaticimab SC as monotherapy compared to placebo in 703 patients with nonfamilial hypercholesterolemia and mixed hyperlipemia at low-to-moderate atherosclerotic cardiovascular disease risk. The study's primary endpoint was percentage change in LDL-C from baseline to Week 12 for 150 mg Q4W and 450 mg Q12W and to Week 16 for 300 mg Q8W. Recaticimab showed strong and consistent LDL-C lowering, ranging from 45% to 52.8% of reduction, compared with placebo. Most patients in the recaticimab groups reached LDL-C below 100 mg/dL, especially in the 300 mg Q8W group (91%). The treatment also reduced other lipids including non-HDL -C, ApoB and Lp(a) by 43–47%, 40–46%, and 18–32%, respectively ($p < 0.0001$ for all comparisons). At 24 weeks, LDL-C reduction stayed stable when patients switched from placebo to recaticimab. Safety was comparable to placebo.[35]

The placebo-controlled REMAIN-2 Phase 3 study (NCT04885218) aimed to assess the efficacy and safety of 48-week treatment with recaticimab as add-on therapy to statins in nonfamilial hypercholesterolemia in 689 patients. Recaticimab met the study's primary endpoint, showing a significant LDL-C reduction vs placebo at Week 24, with mean decreases of −62.2%, −59.7%, and −53.4% for the 150 mg Q4W, 300 mg Q8W, and 450 mg Q12W regimens ($p < 0.0001$), respectively. LDL-C lowering was sustained through Week 48, and other lipid markers (non-HDL-C, ApoB, Lp(a)) also improved. Safety was favorable, with similar rates of treatment-related and serious adverse events between recaticimab and placebo.[36]

The randomized, double-blind, placebo-controlled REMAIN-3 Phase 3 study (NCT04844125) conducted in China assessed the efficacy and safety of 150 mg SC recaticimab Q4W for 12 weeks in 95 patients with a heterozygous familial hypercholesterolemia. Recaticimab met its primary endpoint, which was the percentage change in LDL-C from baseline to Week 12 by significantly lowered the LDL-C level (−54.4%) with recaticimab compared with placebo (−4.5%).[37]

Recaticimab distinguishes itself from other anti-PCSK9 antibodies currently approved in China and globally by its prolonged half-life, which supports a dosing interval of up to Q8W and offers flexibility through both monthly and bi-monthly administration regimens.

Finotonlimab (Sinocelltech Ltd.)

Finotonlimab (安佑平, Anyouping®) is a humanized IgG4κ monoclonal antibody directed against programmed cell death protein 1 (PD-1, CD279). The hinge-stabilized (S228P) antibody binds PD-1, thereby blocking binding of its ligands, PD-L1/PD-L2 and enabling increased T-cell activity. Developed by Sinocelltech Ltd., finotonlimab was approved by the NMPA in February 2025 for the first-line treatment of recurrent or metastatic (R/M) head and neck squamous cell carcinoma (HNSCC) in combination with platinum-based chemotherapy.[38] Following the first approval, Sinocelltech obtained a supplemental approval for finotonlimab in combination with bevacizumab, for the first-line treatment of patients with unresectable or metastatic hepatocellular carcinoma (HCC) who have not previously received systemic therapy.[39]

NMPA's approval of finotonlimab for HNSCC was based on a pivotal Phase 3 randomized, double-blind, multicenter trial (NCT04146402) that enrolled 370 patients with untreated R/M HNSCC. In this study, participants were randomly 2:1 assigned to receive finotonlimab 200 mg IV Q3W plus the combination of cisplatin and 5-fluorouracil (C5F) or placebo plus C5F. The primary endpoint was overall survival (OS). Finotonlimab significantly improved survival outcomes with a median OS of 14.1 months (95% CI 11.1–16.4) compared with 10.5 months (95% CI 8.1–11.8) for chemotherapy alone, corresponding to a hazard ratio of 0.73 ($p = 0.0165$).[40]

Finotonlimab is also being evaluated in advanced HCC in a Phase 2/3 open-label study (NCT04560894, CTR20201976 and CTR20201974) comparing finotonlimab in combination with SCT510, an biosimilar of anti-vascular endothelial growth factor (VEGF) bevacizumab (Avastin®), versus sorafenib in 398 patients. Patients received finotonlimab 200 mg IV Q3W with SCT510 15 mg IV Q3W in the Phase 2 part of the study, and then 346 patients were randomized (2:1) to either the finotonlimab plus SCT510 cohort or the sorafenib cohort in the Phase 3 part of the study. Primary outcomes included overall survival and progression-free survival (PFS). With a median follow-up time of 19.9 and 19.0 months for the finotonlimab

plus SCT510 and sorafenib cohorts, respectively, the median PFS was significantly longer in the combination arm (7.1 months than in the sorafenib group (2.9 months), corresponding to a HR: 0.5 (95% CI 0.38, 0.65, $p < 0.0001$). Median OS was also significantly longer in patients receiving finotonlimab plus SCT510 (22.1 months) than in those receiving sorafenib (14.2 months).[41]

Another Phase 3 double-blind study (NCT04171284) investigated finotonlimab plus docetaxel versus docetaxel plus placebo in patients with previously treated advanced squamous NSCLC. While the combination of finotonlimab plus docetaxel improved significantly prolonged OS and improved clinical outcomes without additional safety risk,[42] the study was terminated due to the company's strategy adjustments, independent of the safety and efficacy of the trial medication.

Siltartoxatug (Zhuhai Trinomab Biotechnology Co., Ltd.)

Siltartoxatug (新替妥, Sintetol®) is a recombinant, human IgG1κ monoclonal antibody targeting tetanus toxin developed by Zhuhai Trinomab Biotechnology Co., Ltd. The product is formulated for IM injection for emergency prophylaxis in adults. Siltartoxatug provides passive immunity by blocking the capacity of tetanus neurotoxin to bind neuronal receptors and thereby preventing its cellular uptake.[43]

Siltartoxatug was granted Breakthrough Therapy designation for the emergency prophylaxis of tetanus in adults by NMPA, as well FDA's Fast track designation for this indication. In February 2025, siltartoxatug was approved via the priority review pathway for emergency prophylaxis of tetanus in adults by the NMPA.[44]

This approval is supported by results from a Phase 3 study (NCT05664750, TNM002-301 showing that siltartoxatug is an effective and safe option for passive immunization against tetanus.[43] In this randomized, double-blind, active-controlled study, 675 adults with unclean or contaminated wounds who were presented at clinical centers were randomized 2:1 to receive a single IM gluteal injection of siltartoxatug 10 mg ($n = 440$) or plasma-derived human tetanus immunoglobulin (HTIG) 250 IU ($n = 221$), within 24 hours after injury. The primary efficacy endpoint was a pharmacodynamic surrogate: the proportion of participants with an increase in anti-tetanus neutralizing antibody titer of at least 0.01 IU/mL at 12 hours after administration, a threshold in line with WHO guidance for a minimal protective neutralizing antibody level for tetanus. Siltartoxatug met its primary endpoint with absolute difference of 42.3% points (95% CI 35.5–49.1; $p < 0.0001$) in terms of percentage of participants above the threshold of 0.01 IU/mL in siltartoxatug arm (95.4%) compared with the HTIG arm (53.2%). The safety and tolerability of siltartoxatug were acceptable and broadly similar to HTIG.[43]

Ebdarokimab (Akeso Inc.)

Ebdarokimab (AK101, 爱达罗®) is a human IgG1κ monoclonal antibody targeting the p40 subunit shared by interleukin (IL)-12 and IL-23, two cytokines promoting the proliferation of pro-inflammatory T cells (Th1, Th17 cells), developed by Akeso Inc. By binding to the p40 subunit, ebdarokimab blocks IL-12 and IL-23 from interacting with their receptors, thereby suppressing the downstream Th1/Th17-mediated immune pathways. This reduces inflammatory cytokine production (interferon-γ, TNF-α, IL-17) and helps restore immune balance, effectively alleviating psoriatic inflammation.

In April 2025, the NMPA approved ebdarokimab for moderate-to-severe plaque psoriasis in adults. The decision was supported by two Phase 3 studies demonstrated ebdarokimab's efficacy and safety at both 16 weeks (NCT05120297) and 52 weeks (NCT05509361) in these patients.[45]

The short-term effects of ebdarokimab were evaluated in the randomized, double-blind, placebo-controlled Phase 3 AK101-302 study (NCT05120297), which enrolled 452 moderate-to-severe plaque psoriasis patients who received 135 mg SC ebdarokimab or placebo. At week 16, patients receiving ebdarokimab injection with two doses at Weeks 0 and 4 showed significantly positive efficacy, with a PASI 75 response rate of 79.4%.[45]

The long-term safety and efficacy of ebdarokimab were evaluated in the open-label Phase 3 AK101-303 study (NCT05509361), which enrolled 950 patients with moderate-to-severe plaque psoriasis. New patients as well as those who completed the AK101-302 study were enrolled. The study's primary endpoint was the assessment of serious adverse event and adverse events of special interest. The

secondary endpoint measured the percentage of subjects who achieved at least 50% (PASI 50), 75% (PASI 75) and 90% (PASI 90) reduction in psoriasis area and severity index (PASI) score up to 52 weeks. In this study, all participants received ebdarokimab 135 mg with dosing adjusted by prior treatment status. Patients who had previously received ebdarokimab (Group 1) continued treatment at Week 16 and then Q12W up to Week 52. Those who had received placebo in the earlier study began ebdarokimab at Week 16 or 20, followed by maintenance dosing Q12W. Newly enrolled subjects started ebdarokimab at Weeks 0 and 4, with maintenance dosing Q12W and follow-up through Week 52. Treatment with ebdarokimab produced high and durable response rates in patients with psoriasis. In Group 1, 80.5% achieved PASI 75 and 66.0% achieved sPGA 0/1 at Week 16, with responses maintained through Week 52. Group 2, which switched from placebo to ebdarokimab at Week 16, showed marked improvement, reaching 81.4% (PASI 75) and 71.1% (sPGA 0/1) by Week 32, remaining stable thereafter. Newly treated patients (Group 3) achieved 69.5% (PASI 75) and 59.1% (sPGA 0/1) at Week 16, similar to Group 1. All groups demonstrated sustained efficacy throughout follow-up. Regarding safety, 82.9% of participants experienced at least one treatment-emergent adverse event (TEAE) and 33.1% had drug-related events, mostly mild to moderate. Overall, ebdarokimab was well-tolerated and maintained robust, long-term efficacy.[46]

Trastuzumab rezetecan (Jiangsu HengRui Medicine Co. Ltd.)

Trastuzumab rezetecan (SHR-A1811, 艾维达®) is a human epidermal growth factor receptor 2 (HER2)-targeting IgG1κ ADC developed using Hengrui's Rapid Modular ADC Platform. It consists of a trastuzumab conjugated, via a cleavable linker, to a topoisomerase I inhibitor payload with an average drug-to-antibody ratio (DAR) of 6. In May 2025, NMPA granted approval for trastuzumab rezetecan for treatment of adult patients with unresectable locally advanced or metastatic NSCLC harboring activating HER2 mutations who have previously received at least one systemic therapy.[47]

The approval of trastuzumab rezetecan was based on the single-arm, pivotal Phase 1/2 HORIZON-Lung study (NCT04818333), which enrolled 94 NSCLC patients at sites in China. Trastuzumab rezetecan was administered at 4.8 mg/kg IV Q3W The study's primary endpoint was the objective response rate in patients who received at least one cycle of treatment.[48] With a follow-up of 14.2 months, 74.5% of patients had a confirmed objective response and the median PFS was 11.5 month.

To date, trastuzumab rezetecan has been granted Breakthrough Therapy Designation by the NMPA for nine indications, including NSCLC, breast, colorectal, and biliary tract cancer, as well as gastric or gastroesophageal junction adenocarcinoma and various gynecologic malignancies.[47] FDA has granted Orphan Drug designation to the combination of trastuzumab rezetecan with adebrelimab and chemotherapy for the treatment of gastric or gastroesophageal junction adenocarcinoma and treatment of biliary tract cancer. In addition to the NSCLC studies (NCT06430437), trastuzumab rezetecan is being evaluated in Phase 3 studies of patients with breast (NCT05424835, NCT06057610, NCT05814354, NCT06126640, NCT07196774), colorectal (NCT06199973), and ovarian (NCT06828354) cancers, as well as gastric or gastroesophageal junction adenocarcinoma (NCT06123494, NCT07118527).

In September 2025, an NDA for trastuzumab rezetecan for breast cancer was accepted for review by the NMPA and included in the priority review program. Also in September 2025, Hengrui Pharma announced that it had entered into an exclusive license agreement with Glenmark Specialty S.A., a wholly owned subsidiary of Glenmark Pharmaceuticals Ltd. that gives Glenmark exclusive rights to develop and commercialize trastuzumab rezetecan worldwide, excluding certain regions including Mainland China, US, Canada, Europe, and Japan.[49]

Suvemcitug (Jiangsu Simcere Pharmaceutical Co. Ltd.)

Suvemcitug (ENZESHU®) is a humanized monoclonal IgG1κ antibody that targets VEGF, inhibiting VEGF's binding to its receptors, and thereby blocking angiogenesis and tumor vascularization. Suvemcitug was designed to have a higher binding affinity for VEGF than bevacizumab (Avastin®). Suvemcitug was co-developed by Simcere Zaiming Pharmaceutical Co. Ltd, which is a subsidiary of Simcere Pharmaceutical Group Ltd., and Apexigen, Inc., which was acquired by Pyxis Oncology in 2023.

Simcere Zaiming currently holds the exclusive rights to develop and commercialize this product in greater China.[50]

In July 2025, Simcere Pharmaceutical Group announced that NMPA approved suvemcitug, (ENZESHU®) for use in combination with chemotherapy (paclitaxel, pegylated liposomal doxorubicin, or topotecan) for the treatment of recurrent, platinum-resistant epithelial ovarian, fallopian tube, or primary peritoneal cancer in adult patients who have received at least one prior systemic therapy after the development of platinum resistance. This is the first anti-angiogenic monoclonal antibody approved in China for this indication.[51]

The approval was supported by results from the Phase 3 SCORES trial (NCT04908787), a randomized, double-blind, placebo-controlled study enrolling 421 patients with platinum-resistant ovarian cancer across multiple centers in China. Patients were randomized to receive suvemcitug (1.5 mg/kg Q2W) or placebo in combination with investigator's-choice chemotherapy. The trial met its primary endpoint of PFS. Median PFS was 5.49 months in the suvemcitug plus chemotherapy arm versus 2.73 months with placebo plus chemotherapy, corresponding to a hazard ratio (HR) of 0.46 (95% CI, 0.35–0.60; $p < 0.0001$). Objective response rate (ORR), disease control rate (DCR), and duration of response (DoR) were also improved in the suvemcitug arm. At final analysis, a 23% reduction in the risk of death was observed, indicating a favorable overall survival trend. Grade ≥3 adverse events occurred in a similar proportion of patients compared with other VEGF inhibitors, and no new safety signals were identified.[51]

Firsekibart (Changchun GeneScience Pharmaceutical Co., Ltd.)

Firsekibart (金赛新, Jin Beixin®) is a hinge-stabilized (S228P) human anti-IL-1β IgG4λ monoclonal antibody developed primarily for immune-mediated disorders. In June 2025, NMPA approved firsekibart for the treatment of gouty arthritis in adult patients who cannot use, do not tolerate, or do not respond adequately to non-steroidal anti-inflammatory drugs (NSAIDs) and/or colchicine, and for whom repeated corticosteroid courses are unsuitable.[52]

The safety and efficacy of firsekibart in gouty arthritis patients were evaluated in a randomized, active-controlled, double-blind Phase 3 trial (NCT05983445) conducted in China. Patients were randomized (1:1) to receive a single 200 mg SC dose firsekibart or 7 mg IM betamethasone. The 48-week study (24-week double-blind and 24-week open-label phases) evaluated co-primary endpoints of pain reduction at 72 hours and time to first new flare over 12 weeks, along with secondary efficacy and safety outcomes. Among 311 patients analyzed, firsekibart demonstrated non-inferior pain reduction at 72 hours compared with compound betamethasone (−57.09 mm vs. −53.77 mm) and significantly prolonged the median time to first new flare while reducing the risk of flare by 90% over 12 weeks and 87% over 24 weeks ($p < 0.0001$). Fewer patients receiving firsekibart experienced new flares at 12 and 24 weeks (10.9% vs. 65.2% and 14.7% vs. 66.5%, respectively), with lower mean flare counts per patient across both time points.[53] Of a subset of 42 patients with baseline glomerular filtration rate (eGFR) < 60, firsekibart provided comparable pain reduction but significantly prolonged the time to first new flare, reducing flare risk by up to 98% over 12 weeks, with no serious adverse events or renal function impairment observed.[54]

Trastuzumab botidotin (Sichuan Kelun-Biotech Biopharmaceutical Co Ltd, Sorrento Therapeutics, Inc.)

Trastuzumab botidotin (A166, 舒泰莱®) is an ADC composed of a humanized anti-HER2 IgG1κ antibody conjugated to Levena Biopharma's proprietary tubulin inhibitor Duo-5 toxin via a stable protease-cleavable valine-citrulline linker and site-specific (DAR 2) K-Lock™ conjugation chemistry. In October 2025, trastuzumab botidotin was approved for marketing by the NMPA for adult patients with unresectable or metastatic HER2+ breast cancer who have received one or more prior anti-HER2 therapy.[55]

This approval was based on the results of the randomized, open-label Phase 3 KL166-III-06 study (NCT06968585) which compared the effects of trastuzumab botidotin versus trastuzumab emtansine (Kadcyla®) in 365 patients with HER2+ unresectable or metastatic breast cancer who received prior trastuzumab and taxane-containing therapies. Patients were randomized (1:1) to receive A166 (4.8 mg/kg Q3W) or Kadcyla® (3.6 mg/kg Q3W) until disease progression or unacceptable toxicity. The median PFS

was significantly longer in patients treated with trastuzumab botidotin compared to Kadcyla® (11.1 months vs 4.4 months, respectively; $p < 0.0001$), while the objective response rate (ORR) by BICR was 76.9% vs 53.0%, respectively. Grade ≥3 treatment emergent adverse events were similar in trastuzumab botidotin (69.8% of patients) and Kadcyla® (63.7% of patients), with the most common adverse events being ocular AEs for trastuzumab botidotin and decreased platelet count for Kadcyla®.[56]

Becotatug vedotin (Shanghai Miracogen, Lepu Biopharma Co., Ltd.)

Becotatug vedotin (MRG003, MEIYOUHENG®) is an ADC developed by Shanghai Miracogen and Lepu Biopharma in which the microtubule-disrupting agent MMAE is conjugated to a humanized anti-EGFR IgG1κ antibody via a cleavable valine-citrulline linker (average DAR = 4). It has been evaluated as a treatment for multiple EGFR-positive tumors, including nasopharyngeal carcinoma (NPC) and metastatic HNSCC. Becotatug vedotin was granted Breakthrough Therapy, Orphan Drug, and Fast Track designations by the FDA, as well as Breakthrough Therapy designation by the NMPA, for the treatment of recurrent metastatic NPC.[57] In October 2025, NMPA approved becotatug vedotin for recurrent or metastatic NPC.[58]

The approval was supported by data from the pivotal, open-label Phase 2 MRG003-005 study (NCT05126719) in which 173 recurrent metastatic NPC patients who had failed at least two prior lines of systemic chemotherapy and PD-(L)1 inhibitor were randomly assigned to receive 2.3 mg/kg IV becotatug vedotin ($n = 86$) Q3W or capecitabine ($n = 36$)/docetaxel ($n = 51$). This study reached the BICR-assessed ORR with becotatug vedotin compared to chemotherapy (30.2% vs 11.5%, respectively, $p = 0.0025$), and median PFS was also significantly improved (5.8 months vs 2.8 months respectively, $p = 0.0146$).[59]

Becotatug vedotin is also being evaluated in Phase 3 clinical trials for recurrent metastatic NPC in combination with pucotenlimab (PUYOUHENG) (NCT06976190) and as a second- or third-line therapy for recurrent metastatic HNSCC patients who have previously failed PD-(L)1 inhibitors and platinum-based therapy (NCT05751512).

Picankibart (Innovent Biologics, Inc.)

Picankibart (PECONDLE®) is a humanized IgG1κ monoclonal antibody designed to target the p19 subunit of interleukin-23 and prevent IL-23 from binding to cell surface receptors. Mutations (M252Y, S254T, T256E) were made to picankibart's Fc to extend its half-life and thereby reduce dosing. Developed by Innovent Biologics, picankibart is undergoing evaluation as a treatment for immune-mediated disorders, including moderate to severe plaque psoriasis and ulcerative colitis. The company announced in November 2025 that the NMPA had approved picankibart for the treatment of moderate to severe plaque psoriasis.[60]

The approval was supported by data from the double-blind, placebo-controlled Phase 3 CLEAR-1 trial (NCT05645627), which evaluated SC doses of picankibart in 500 patients with moderate to severe plaque psoriasis. The co-primary endpoints of the trial were met, with more than 80% of patients achieving at least 90% reduction from baseline in Psoriasis Area and severity index (PASI 90) at Week 16. Responses were durable with maintenance dosing Q12W after induction, and efficacy was consistent across secondary measures, including PASI 75 and PASI 100. The safety profile was in line with other IL-23 inhibitors.[60,61] Building on these results, Innovent initiated a Phase 3 biologic-switch trial (NCT06945107) in May 2025 to test picankibart in psoriasis patients with inadequate responses to IL-17 inhibitors.

Picankibart has also shown encouraging activity in ulcerative colitis. In a Phase 2 trial (NCT05377580), clinical remission at Week 12 was achieved in 20% and 14% of patients treated with picankibart compared with 2% on placebo, and clinical response rates reached 54% and 68% versus 22% for placebo. Improvements extended to symptomatic, endoscopic, and histologic endpoints, with no new safety concerns observed.[62]

Antibody therapeutics undergoing first regulatory review

Data pertaining to the safety, efficacy, and quality of drugs are rigorously examined by regulatory agencies prior to their approval for marketing. These data are submitted in applications that go by

Table 2. Commercially sponsored investigational monoclonal antibody therapeutics with marketing applications in regulatory review in any country. Table includes information found in the public domain as of December 31, 2025. Molecules are stratified by therapeutic area (non-cancer, cancer), then listed in chronological order according to projected approvals or when the marketing application was submitted, with applications submitted in the EU, US, or Japan first, followed by those submitted in China. Abbreviations: ADC, antibody-drug conjugate; HER, human epidermal growth factor receptor; IL, interleukin; MASP, mannan-binding lectin-associated serine protease; PD-1, programmed cell death protein 1; TGF, transforming growth factor. Monthly lists of medicines for human use under evaluation by EMA available at: https://www.ema.europa.eu/en/medicines/medicines-human-use-under-evaluation. Supplemental Table S3 (excel format) includes all data in Tables 1-4.

INN	Target; Format	Indication(s) under review	Country of review
Bentracimab	Ticagrelor; Human IgG1λ Fab	Reversal of the antiplatelet effects of ticagrelor	US
Apitegromab	Myostatin; Human IgG4λ	Spinal muscular atrophy	EU, US
Denecimig	Factor IXa, Factor X; Human IgG4κ; Bispecific	Hemophilia A	EU, US
Gefurulimab	Complement C5, Albumin; Humanized VHH-VHH'; Bispecific	Generalized myasthenia gravis	Japan
Veligrotug	IGF-1 R; Humanized IgG1κ	Thyroid eye disease	US
Imsidolimab	IL-36 R; Humanized IgG4κ	Generalized pustular psoriasis	US
Batoclimab	FcRn; Human IgG1λ	Generalized myasthenia gravis	China
Amdokitug	IL-17A; Humanized IgG1κ	Plaque psoriasis	China
Gumokimab	IL-17A; Chimeric/humanized IgG1κ	Plaque psoriasis	China
Silevimig	Rabies virus; Human IgG1; scFv-Fc-Fab; Bispecific	Rabies, Post-exposure prophylaxis	China
Vecantoxatug	Tetanus toxin; Humanized IgG1κ	Tetanus prophylaxis	China
MIL62	CD20; Humanized mAb	Primary membranous nephropathy, Neuromyelitis optica spectrum disorder	China
Anflekitug	IL-1 beta; Humanized IgG1κ	Gouty arthritis	China
Rademikibart	IL-4 R alpha; Human IgG4κ	Atopic dermatitis	China
Telikibart	IL-4 R alpha; Human IgG1κ	Atopic dermatitis	China
Recibokibart	IL-36 R; Humanized IgG1κ	Generalized pustular psoriasis	China
SAL003	PCSK9; Human IgG4	Hypercholesterolemia and mixed hyperlipidemia	China
Comekibart	IL-4 R alpha; Humanized IgG4κ	Atopic dermatitis	China
Roconkibart	IL-17A; Humanized IgG4κ	Plaque psoriasis	China
Lecankitug	IL-17A/F; Humanized IgG1κ	Plaque psoriasis	China
BAT5906	VEGF; Humanized IgG1κ	Wet age-related macular degeneration	China
Sasanlimab	PD-1; Humanized IgG4κ	Non-muscle invasive bladder cancer	EU
Pivekimab sunirine	CD123; Humanized IgG1κ; ADC	Blastic plasmacytoid dendritic cell neoplasm	US
Retlirafusp alfa	PD-L1, TGF beta; Human IgG4κ IgG-(TGFbeta-R2-ECD)2; Bispecific, Immunoconjugate	Gastric cancer or gastroesophageal junction cancer	China
Anbenitamab	HER2, HER2; Humanized IgG1κ; Bispecific, Biparatopic	Gastric cancer or gastroesophageal junction cancer	China
Izalontamab brengitecan	EGFR, HER3; Chimeric/human IgG1κ/ λ; ADC bispecific	Nasopharyngeal carcinoma	China

various names, such as biologics license application (BLA), marketing authorization application (MAA), and new drug application (NDA). We identified 26 investigational (i.e., not yet approved for marketing in any country) antibody therapeutics for which marketing applications were undergoing review by at least one regulatory agency, as determined by data publicly available by December 31, 2025 (Table 2). Most (21/26, 81%) are for non-cancer indications, with only five for cancer. More than two-thirds (18/26, 69%) are undergoing review in China, with only 8 undergoing review elsewhere. These antibody therapeutics are described in chronical order below according to projected approvals or when the marketing application was submitted, with applications submitted outside China first, followed by those submitted in China.

Antibodies with marketing applications in review: non-cancer indications

Bentracimab (SFJ Pharmaceuticals, SERB Pharmaceuticals)

Bentracimab (PB2452) is a recombinant human monoclonal antibody Fab, derived from an IgG1λ antibody, developed to rapidly and specifically bind ticagrelor and its active metabolite to reverse its antiplatelet effect. The antibody, which originated at MedImmune and was initially developed as MEDI2452, is intended for patients on ticagrelor who present with life-threatening or uncontrolled bleeding, or who require urgent surgery or invasive procedures. Bentracimab has received Breakthrough Therapy and Orphan Drug designations from the FDA. SERB acquired exclusive US rights to bentracimab from SFJ

Pharmaceuticals. In August 2024, the companies announced that FDA accepted a BLA for bentracimab for patients requiring urgent surgery or experiencing major bleeding while on ticagrelor.[63] FDA granted the BLA priority review, with an initial target action date in 2025 Q1, but, as of November 2025, no further information regarding the status of the BLA has been publicly disclosed.

The BLA is supported by results of the open-label, single-arm Phase 3 REVERSE-IT trial (NCT04286438), which enrolled 226 patients on ticagrelor with either major bleeding events or urgent surgical needs. On Day 1 of the study, patients received an IV infusion comprising an initial IV bolus of 6 g infused over 10 minutes, followed immediately by a 6 g IV loading infusion over four hours and then a 6 g IV maintenance infusion over 12 hours. The study's primary efficacy endpoint was minimum percent inhibition of platelet reactivity units (PRU) within four hours after the start of bentracimab infusion compared to baseline. Results of the trial demonstrated a statistically significant change ($p < .0001$) in PRU following treatment with bentracimab, indicating a strong reversal of inhibition. The study also achieved its secondary endpoint: 83.1% and 100% of patients in the bleeding and surgical groups, respectively, achieved normal blood clotting and repair of damaged blood vessels within 24 hours after bentracimab treatment.[64]

Apitegromab (Scholar Rock)

Apitegromab is a human IgG1λ monoclonal antibody that binds selectively to the latent forms of myostatin, preventing activation of this negative regulator of muscle growth.[65] Unlike other myostatin inhibitors, apitegromab does not neutralize circulating mature myostatin but blocks its activation upstream. The antibody is being developed for spinal muscular atrophy (SMA) types II and types III in patients already treated with SMN-directed therapies such as nusinersen or risdiplam.[65]

The FDA granted Fast Track, Orphan Drug and Rare Pediatric Disease designations to apitegromab for the treatment of SMA. In January 2025, Scholar Rock announced they had submitted a BLA to FDA for apitegromab as a treatment for SMA. In September 2025, the company announced FDA issued a complete response letter that cited manufacturing issues at a third-party facility.[65] Resubmission of the BLA, with U.S. launch following approval of apitegromab for children and adults with SMA, is anticipated in 2026.

EMA granted Priority Medicines (PRIME) and Orphan Medicinal Product designations, to apitegromab for the treatment of SMA. The company submitted a marketing application for apitegromab for SMA to EMA, which began its ongoing review in March 2025.

The marketing applications are supported by data from the TOPAZ and SAPPHIRE studies. The Phase 2 TOPAZ study (NCT03921528) was an open-label, multicenter trial in non-ambulatory later-onset SMA patients. After 12 months, participants administered a 20 mg/kg dose achieved a mean 7.1-point gain on the Hammersmith Functional Motor Scale – Expanded (HFMSE) scale,[66] and benefits were sustained in the extension for up to 36 months.[67] The therapy was generally well tolerated. Long-term extension updates showed durable improvements of motor function in type II and type III SMA.

The Phase 3 SAPPHIRE trial (NCT05156320) was a randomized, placebo-controlled study of apitegromab added to survival motoneuron (SMN) therapy in children and young adults aged 2–21 years with later-onset SMA. Participants received apitegromab 10 mg/kg, apitegromab 20 mg/kg, or placebo by IV infusion Q4W for 12 months. The study met its primary endpoint, with apitegromab demonstrating a clinically meaningful and consistent benefit in motor function across pre-specified patient subgroups. In the main efficacy population (ages 2–12), the mean difference in change from baseline in HFMSE was 1.8 points ($p = 0.0192$) for all patients receiving apitegromab 10 mg/kg and 20 mg/kg ($n = 106$) compared to placebo ($n = 50$). Patients administered 20 mg/kg of apitegromab ($n = 53$) showed a 1.4 point mean difference compared to placebo ($p = 0.1149$). At 52 weeks, 30.4% of apitegromab-treated patients achieved ≥3-point HFMSE improvement compared with 12.5% on placebo. The least-squares mean change in HFMSE was +2.5 points with apitegromab versus +0.1 with placebo. Safety was comparable to placebo.[68]

Denecimig (Novo Nordisk)

Denecimig (NN-7769, NNC0365–3769, Mim8), a FVIII-mimetic bispecific antibody using a human, hinge-stabilized IgG4κ bispecific antibody derived from Genmab's Duobody technology. Developed by Novo

Nordisk, denecimig facilitates the assembly of activated coagulation Factors IXa (FIXa) and X (FX) on platelet membranes, mimicking Factor VIII to restore the thrombin generation capacity, making it a potential treatment for hemophilia A, which is caused by mutations in the gene encoding Factor VIII. Denecimig received Orphan Drug designation from the FDA for the treatment of hemophilia A. In September 2025, Novo Nordisk announced a BLA had been submitted to FDA for denecimig as a preventive bleed routine prophylaxis to prevent or reduce the frequency of bleeding episodes in adult and pediatric patients with hemophilia A (congenital FVIIIa deficiency), with or without inhibitors.[69] The company also submitted an MAA for denecimig to the EMA.[70]

The BLA was supported by data from the FRONTIER program, including the Phase 3 FRONTIER2 (NCT05053139), FRONTIER3 (NCT05306418) and FRONTIER4 (NCT05685238) studies, which evaluated SC denecimig as a prophylactic treatment for hemophilia A patients with or without inhibitors. FRONTIER2 enrolled participants aged 12 years and older, while FRONTIER3 enrolled pediatric patients aged between 1 years and 11 years old. The effects of SC denecimig treatment once every month and Q1W were evaluated in both studies. FRONTIER4 was an open-label extension study that evaluated the efficacy of SC denecimig Q2W and collected long-term safety and efficacy data from participants administered either denecimig regimen (once every month, Q1W) in the previous FRONTIER studies.

Gefurulimab (AstraZeneca)

Gefurulimab (ALXN1720) is a bispecific nanobody composed of an N-terminal albumin-binding VHH domain linked to a C-terminal VHH targeting complement component 5 (C5). The drug is optimized for SC self-administration and engineered for extended circulatory half-life through neonatal Fc receptor (FcRn)-mediated recycling, enabled by serum albumin trafficking, enabling once-weekly dosing. Gefurulimab selectively inhibits the proteolytic cleavage of C5, thereby preventing the formation of its pro-inflammatory effector fragments, C5a and C5b, which drive complement-mediated immune responses, a key pathological mechanism in gMG.

Initially developed by Alexion Pharmaceuticals, which was acquired by AstraZeneca in 2020, gefurulimab was granted Orphan Drug designation by FDA for the treatment of MG in 2023. AstraZeneca's third-quarter results update indicated a marketing application for gefurulimab has been submitted in Japan.[71]

The placebo-controlled Phase 3 PREVAIL trial (NCT05556096) evaluated gefurulimab in 260 adults with gMG with a positive serological test for autoantibodies against acetylcholine receptor. Patients were randomized 1:1 to receive either gefurulimab or placebo over a 26-week double-blind treatment period. Treatment started with a single weight-based loading dose on Day 1, followed by Q1W weight-based maintenance doses on Day 8. The results showed statistically significant and clinically meaningful improvements in Myasthenia Gravis Activities of Daily Living (MG-ADL) scores at Week 26 compared to placebo. The safety profile was consistent with previous C5 inhibitors, with no new safety signals observed.[72]

Veligrotug (Viridian Therapeutics, Inc., Kissei Pharmaceutical Co. Ltd.)

Veligrotug (ZL-1108, ZB001, VRDN-001, IMGN164) is a humanized IgG1κ monoclonal antibody targeting the insulin-like growth factor 1 receptor (IGF-1 R) extracellular domain. Preclinical studies have demonstrated that veligrotug is a potent and selective full antagonist anti-IGF-1 R antibody.[73] Initially developed by ImmunoGen in collaboration with Sanofi-Aventis, global rights to develop and commercialize veligrotug for all non-oncology indications that do not involve radiopharmaceuticals were exclusively licensed by Viridian from ImmunoGen, Inc. Viridian entered into an exclusive licensing agreement with Kissei Pharmaceutical Co. Ltd. to develop and commercialize veligrotug in Japan. Veligrotug has received Breakthrough Therapy designation for the treatment of thyroid eye disease (TED) from the FDA. In October 2025, Viridian submitted a BLA for veligrotug for TED to FDA that was granted Priority Review; FDA's target date for a first action on the application is June 30, 2026. An MAA submission to EMA is planned in the first quarter of 2026.[74]

Viridian reported positive topline results from the placebo-controlled Phase 3 THRIVE study (NCT05176639), which initially evaluated multiple ascending doses (MAD) of veligrotug in healthy volunteers and patients with moderate-to-severe active TED, followed by evaluation of a fixed dose in

TED participants. In the MAD portion, participants received 30-minute IV infusions of veligrotug ranging from 3 mg/kg to 20 mg/kg ($n = 75$) or placebo ($n = 38$). Patients with TED were subsequently randomized to receive five IV infusions of 10 mg/kg veligrotug or placebo Q3W to Week 52. The primary endpoint was the proptosis responder rate at Week 6 for MAD TED participants and Week 15 for Phase 3 patients. The trial met all primary and secondary endpoints, demonstrating highly statistically significant improvements ($p < 0.0001$) in all signs and symptoms of TED. At Week 15, 70% of patients receiving veligrotug achieved a proptosis response, and 67% had an overall response, compared to 5% in both categories for the placebo group. Among the veligrotug patients (21/30) who were proptosis responders at Week 15 and continued follow-up to Week 52, 70% maintained their proptosis response.[75,76]

Viridian also reported positive topline results from the randomized, double-masked, placebo-controlled Phase 3 THRIVE-2 study (NCT06021054), which evaluated veligrotug in patients with chronic TED. In this study, patients received five infusions of 10 mg/kg veligrotug Q3W. At Week 15, the proptosis responder rate was 56% in the veligrotug patient vs. 8% in the placebo group, the diplopia responder rate was 56% vs. 25% in the placebo group, and 32% achieved complete resolution of diplopia vs. 14% in the placebo group. The safety profile remained favorable and consistent with prior studies.[75,77]

Imsidolimab (Vanda Pharmaceuticals, Inc.)

Imsidolimab (ANB019) is a humanized IgG4κ monoclonal antibody containing the S228P hinge-stabilizing mutation, developed by AnaptysBio for the treatment of GPP, a rare, life-threatening, systemic autoinflammatory condition characterized by episodic flares of sterile pustules. Imsidolimab selectively targets and inhibits the interleukin-36 receptor (IL-36 R), a critical mediator in the IL-36 signaling pathway, which has been implicated in the pathogenesis of GPP and other inflammatory dermatoses. The FDA granted Orphan Drug designation to the drug for the treatment of GPP.

Vanda Pharmaceuticals holds an exclusive global license from AnaptysBio for the development and commercialization of imsidolimab. In December 2025, Vanda announced the submission of a BLA for imsidolimab for treatment of GPP.[78] The company requested priority review, which, if granted, would enable potential approval of imsidolimab as early as mid-2026.

The BLA submission was supported by data from the placebo-controlled Phase 3 GEMINI-1 (NCT05352893) study and the long-term extension GEMINI-2 study (NCT05366855). The GEMINI-1 trial enrolled 45 GPP patients randomized 1:1:1 to receive a single IV dose of 750 mg imsidolimab, 300 mg imsidolimab, or placebo. At Week 4, 53% of patients receiving either imsidolimab dose achieved a GPP Physician Global Assessment (GPPPGA) score of 0/1 (clear or almost clear skin), compared to 13% in the placebo group ($p = 0.0131$). GPPPGA 0/1 responder patients, partial responders, or those needing rescue therapy could enroll in the GEMINI-2 trial to receive a monthly maintenance SC dose of 200 mg of imsidolimab or placebo. Of those who received placebo in GEMINI-1 and switched to imsidolimab, 56% achieved a GPPGA of 0/1 by Week 4. After at least 24 weeks of follow-up, 100% of GPPPGA 0/1 responders who received SC imsidolimab experienced no flares, while of the GPPPGA 0/1 responders who received placebo 25% maintained a GPPPGA 0/1 score and 63% experienced a flare. The safety profile in GEMINI-2 was consistent with GEMINI-1, with no treatment-related serious adverse events reported.[79,80]

Batoclimab (HanAll Biopharma, Immunovant, Inc.)

Batoclimab (HBM9161, IMVT-1401, 巴托利单抗) is an anti-FcRn human IgG1λ antibody that was Fc engineered (L234A, L235A) for reduced Fc effector functions. Batoclimab was originally developed by South Korea-based HanAll Biopharma for the treatment of IgG-mediated autoimmune diseases such as gMG and Grave's disease. Batoclimab was licensed for co-development in Greater China to Harbour Biomed, which subsequently entered into an agreement with NBP Pharma, a wholly owned subsidiary of the CSPC group. The development rights to batoclimab for North America, Latin America, Switzerland, North Africa, the UK, EU, and the Middle East were licensed to Roivant Sciences, which created Immunovant, Inc. to develop batoclimab.

Batoclimab received Breakthrough Therapy designation by the NMPA in 2021 for the treatment of gMG. In 2023, Harbour Biomed submitted a marketing application for batoclimab to the NMPA,

followed by a resubmission of the marketing application in 2024 with additional safety data for the treatment of gMG.[81] This submission was based on results from the placebo-controlled Phase 3 trial (NCT05039190) sponsored by Harbor BioMed (Guangzhou) Co. Ltd, in which 131 gMG patients who tested positive for AChR or MuSK antibodies were treated with either placebo or 680 mg SC batoclimab weekly for six weeks, followed by two weeks of observation without treatment. Batoclimab significantly improved the sustained Myasthenia Gravis Activities of Daily Living (MG-ADL) score (58.2%, 39/67 patients) compared to placebo (31.3%, 20/64 patients) (odds ratio, 3.45; 95% CI, 1.62–7.35; $p = .001$).[82]

Batoclimab received Orphan Drug designation for TED by Japan's Ministry of Health, Labour and Welfare.[83] A placebo-controlled Phase 3 study (NCT05517421) sponsored by Immunovant Sciences GmbH is evaluating the efficacy of batoclimab in patients with active TED. The study is active but not recruiting patients and is expected to be completed by the end of 2025.

Amdokitug (Sunshine Guojian Pharmaceutical (Shanghai) Co. Ltd.)

Amdokitug (SSGJ-608) is a humanized anti-IL17A IgG1κ monoclonal antibody developed by Sunshine Guojian for the treatment of inflammatory diseases such as psoriasis, ankylosing spondylitis, and spondyloarthritis. In **November 2024**, the company's marketing application for SSGJ-608 was accepted by the NMPA for moderate to severe plaque psoriasis.[84]

A randomized, double-blind, placebo-controlled Phase 3 study (NCT05536726) evaluated the effects of amdokitug for the treatment of moderate to severe plaque psoriasis in 458 Chinese patients with a PASI score ≥12. Patients were randomly assigned at a ratio of 2:2:1 to either experimental group A (160 mg W0 + 80 mg Q2W for first 12 weeks +80 mg Q4W maintenance), experimental group B (160 mg Q4W for first 12 weeks +160 mg Q4W maintenance) or placebo by SC injection. All primary efficacy endpoints (PASI 75 and sPGA 0/1) were met, as were key secondary endpoints (PASI 90, PASI 100, and sPGA 0).[85]

The safety and efficacy of amdokitug are also being evaluated in spondylitis patients. A Phase 3 study (CTR20252158) will assess the effects of amdokitug in adult patients with active ankylosing spondylitis. Phase 2 studies of amdokitug in ankylosing spondylitis patients and patients with radiologically negative axial spondylitis (NCT06242652 and NCT06222671, respectively) are ongoing.

Gumokimab (Akeso Biopharma Co., Ltd)

Gumokimab (AK111) is a humanized anti-IL-17A IgG1κ monoclonal antibody developed by Akeso for the treatment of autoimmune diseases such as psoriasis and ankylosing spondylitis. Gumokimab blocks the IL-17/IL-17 R signaling pathway to inhibit the release of pro-inflammatory cytokine secretion mediated by this pathway. In January 2025, an NDA was accepted by NMPA for the use of gumokimab as a treatment for moderate to severe plaque psoriasis. Akeso is also planning to submit an NDA in 2026 for the use of gumokimab in ankylosing spondylitis.[86]

Results from the randomized, double-blind, multicenter pivotal Phase 3 study (AK111-301, NCT06066125, CTR20230111) demonstrated superior treatment outcomes for patients with moderate to severe plaque psoriasis. In the AK111-301 study conducted in China, 351 patients were treated either with SC gumokimab or placebo at Week 0, 1, 2, 3, and 4, and Q4W thereafter until week 48. The primary outcome measure was the percentage of patients who achieved PASI 75, and the percentage of patients who achieved a static physician global assessment (sPGA) of 0 or 1 (in a severity scale of 0–5) at Week 12. The primary endpoint was reached, with efficacy results at 12 weeks showing the PASI 75 response exceeding 90% with an sPGA 0/1 response rate nearing 90% in the gumokimab-treated arm. The improvements were sustained through Week 52.[87]

In August 2025, Akeso announced that the primary and secondary endpoints were reached in their randomized, double-blind placebo-controlled Phase 3 study (NCT06378697) evaluating the use of gumokimab in patients with ankylosing spondylitis.[88] Patients with ankylosing spondylitis with a BASDAI score ≥4, and total back pain score ≥4, and who had previously received at least 2 NSAIDs or with contraindications or intolerance to NSAIDs, were treated with 150 mg SC gumokimab or placebo at Week 0, 1, 4, and Q4W thereafter until Week 48. The primary endpoint reached was patients achieving Assessment of

SpondyloArthritis International Society 20% improvement (ASAS20), while the secondary endpoint of ASAS40 was also met.

Silevimig (Chongqing Genrix Biopharmaceutical Co., Ltd., China Medical System Holdings Limited)

Silevimig (GR1801) is an asymmetric, human IgG1 bispecific antibody targeting epitope I and/or epitope III of the rabies virus (RABV) glycoprotein, developed by Genrix Bio. The arm targeting epitope I is a single-chain variable fragment (scFv), and the other arm targeting epitope III is a Fab, assembled using Knobs-Into -Holes (KIH) technology. Silevimig exhibits broad-spectrum neutralizing activity, demonstrated by its ability to neutralize 90 naturally occurring RABV glycoprotein antigenic variants, 21 pseudotyped, and 18 live street RABVs.[89]

Genrix Bio has a collaboration agreement with China Medical System Holdings Limited (CMS) for exclusive commercialization rights for CMS for silevimig in mainland China and exclusive licensing rights for the rest of the Asia-Pacific region, the Middle East, and North Africa. An NDA for the administration of silevimig to adults requiring passive immunization following suspected RABV exposure was formally accepted by NMPA in January 2025 and is undergoing review.[90]

A Phase 3 trial (NCT05846568/CTR20222502) comparing silevimig to Human Rabies Immunoglobulin (HRIG) as part of post-exposure prophylaxis in patients with WHO Category 3 rabies exposure has been completed. In this study, 1200 adult patients were randomly assigned to the silevimig arm or the HRIG arm based on a ratio of 3:1, with stratification factors including time of exposure, bite location, and number of bites. All patients also received an IM injection of the freeze-dried rabies vaccine for human use (Vero cells) into the deltoid muscle on days 0, 3, 7, 14, and 28 according to the WHO Essen regime. An additional Phase 3 study (CTR20253293) evaluating silevimig in children and adolescents aged 2 to < 18 years requiring passive immunization following suspected rabies virus exposure is currently ongoing.

Vecantoxatug (Genrix (Shanghai) Biopharmaceutical Co., Ltd, China Medical System Holdings Limited)

Vecantoxatug (GR2001) is a humanized IgG1κ monoclonal antibody against the tetanus neurotoxin (TeNT) developed by Genrix (Shanghai) Biopharmaceutical Co., Ltd. It is designed as a recombinant substitute for human tetanus immunoglobulin (HTIG) in prophylaxis. GR2001 binds to the C-terminal domain of the TeNT heavy chain, blocking the toxin from entering neuronal cells and thus providing passive protection. NMPA granted Breakthrough Therapy designation for GR2001 for passive immunization of tetanus. China Medical System Holdings Limited (CMS) was granted exclusive commercialization rights for vecantoxatug in mainland China and exclusive licensing rights for the rest of the Asia-Pacific region, the Middle East, and North Africa by Genrix.[90] In May 2025, Genrix announced that an NDA (CXSS2500051) for GR2001 for this indication was accepted by NMPA.[91]

The marketing application is based on results from a double-blind, HTIG-controlled Phase 3 trial (NCT06635798; CTR20243076) of 582 patients who required tetanus post-exposure prophylaxis. Patients were administered a single IM injection vecantoxatug or HTIG. The primary endpoint was the increase in anti-tetanus neutralizing antibody titers up to 12 hours after GR2001 administration. According to the company's disclosure, vecantoxatug met its primary endpoint in the Phase 3 trial.[91]

MIL62 (Beijing Mabworks Biotech Co., Ltd.)

MIL62 is a humanized, type II, glyco-engineered anti-CD20 monoclonal antibody developed by Beijing MabWorks. The Fc domain of MIL62 is almost completely afucosylated, which enhances binding to FcγRIIIa on natural killer cells and increases antibody-dependent cellular cytotoxicity (ADCC). As a type II antibody, MIL62 promotes direct B-cell cytotoxicity rather than complement activation, a mechanism that distinguishes it from type I antibodies such as rituximab. MIL62 is being evaluated in late-stage clinical studies in cancer and immune-mediated disorders, including neuromyelitis optica spectrum disorder (NMOSD) (NCT05314010), primary membranous nephropathy (PMN) (NCT05862233), systemic lupus erythematosus (NCT05796206), and follicular lymphoma and marginal zone lymphoma (NCT04834024).

MIL62 received Breakthrough Therapy designation for PMN from the NMPA. In May 2025, Mabworks announced that a NDA (CXSS2500054) for MIL62 for NMOSD was accepted by the NMPA.[92] A second application (CXSS2500102) for PMN was accepted in September 2025.[93]

The NDA for MIL62 for NMOSD is supported by data from a placebo-controlled Phase 3 trial (NCT05314010) that enrolled 91 NMSOD patients who were anti-AQP4 seropositive. Patients were administered 1000 mg IV doses of MIL62 at Weeks 1, 3, 25, and 27 or placebo. The study's primary endpoint was the time to first adjudicated relapse. First relapses occurred in 23 patients over 52 weeks, with 2 (4.4%) in the MIL62 group and 21 (45.7%) in the placebo group ((HR, 0.069; 95% CI, 0.016–0.296; $p < 0.0003$). The difference between the groups in the annualized relapse rate (0.092 with MIL62 versus 1.180 with placebo) was also statistically significant (rate ratio 0.078; 95% CI 0.018–0.333; $p = 0.0006$).[94]

The safety and efficacy of MIL62 compared with cyclosporine were evaluated in an open label Phase 3 study (NCT05862233, CTR20231574) of 153 PMN patients who were administered with MIL62 (1000 mg IV at Weeks 1, 3, 25, 27, and 53) or cyclosporine (CsA, trough 125–175 ng/mL for 52 weeks, then tapered up to 8 weeks). The primary outcome measure was the proportion of patients who achieved complete remission based on uPCR at Week 76. MIL62 achieved a higher complete remission rate at Week 76 than cyclosporine (49.4% vs. 3.9%, difference 46.5%, 95%CI 32.1–60.9, $p < 0.0001$; RR 12.5, 95%CI 4.0–39.0). Secondary endpoints, including complete remission at Week 52, overall remission, times to immunologic/clinical remission, relapse and treatment failure, also favored MIL62, which was well tolerated with no new safety signals.[95]

Anflekitug (Sunshine Guojian Pharmaceutical (Shanghai) Co. Ltd.)

Anflekitug (SSGJ-613) is a humanized anti-IL-1β monoclonal antibody developed for the treatment of acute gouty arthritis by Sunshine Guojian Pharmaceuticals. The company announced in June 2025 that the NMPA has accepted an NDA application for SSGJ-613 for this indication.[96]

SSGJ-613 was evaluated in the multicenter, randomized, double-blind, double-dummy, active-controlled Phase 3 trial SSGJ-613-AG-III-01 (CTR20233982) of approximately 500 Chinese patients with frequent gout flares who were unsuitable for NSAIDs or colchicine. Patients received a single 200 mg SC injection of SSGJ-613 or IM betamethasone. Both Phase 3 endpoints (72 h VAS score change from baseline, 12-week recurrence period) were achieved. The VAS score was comparable between SSGJ-613 and betamethasone, while prevention of recurrent flares was superior with SSGJ-613.[96]

Rademikibart (Connect Biopharma Holdings Ltd, Simcere Pharmaceutical Co. Ltd)

Rademikibart (SIM0718, CBP-201) is an interleukin-4 receptor alpha (IL-4 Rα)-targeted, hinge-stabilized IgG4κ antibody developed by Connect Biopharma for the treatment of inflammatory diseases such as atopic dermatitis, asthma, and chronic obstructive pulmonary disease (COPD).[97] In 2023, Connect Biopharma entered into collaboration and exclusive license agreement with Simcere for the development of rademikibart in China. In July 2025, Connect Biopharma announced Simcere submitted an NDA to the NMPA for the use of rademikibart in the treatment of atopic dermatitis in adults and adolescents.[98]

The effects of rademikibart in adults and adolescents with moderate to severe atopic dermatitis are being evaluated in a placebo-controlled Phase 3 study (CTR20242160/NCT06477835) initiated in July 2024. Patients are administered rademikibart as an SC injection, with a loading dose of 600 mg on day 1, followed by 300 mg starting from week 2, and then 300 mg Q2W until Week 52 is completed or placebo according to the same schedule. The primary endpoints of the study are the proportion of subjects who reached an Eczema Area and Severity Index 75 (EASI-75) and the Investigator Global Assessment (IGA) response rate (defined as the proportion of subjects with an IGA score of 0 or 1 that decreased by ≥ 2 points from baseline) at Week 16. As of October 2025, results for this study have not been disclosed.

Positive results were disclosed for a placebo-controlled Phase 2 trial (CBP-201-WW001, NCT04444752) of rademikibart in adults with moderate to severe atopic dermatitis.[96] Patients were randomized 1:1:1:1 to SC rademikibart (300 mg Q2W, 150 mg Q2W, or 300 mg Q4W) or matching placebo, preceded by a 600 mg loading dose of rademikibart or placebo. This trial achieved the primary endpoint of a reduction from

baseline EASI after 16 weeks of rademikibart treatment (63.0% decrease, $p = 0.0007$ for 300 mg Q2W, 57.6% decrease, $p = 0.0067$ for 150 mg Q2W, and 63.5% decrease, $p = 0.0004$ for 300 mg Q4W) compared to placebo (39.7% decrease). Across both primary and secondary endpoints, the 300 mg Q2W and Q4W dosing had comparable efficacy. Similar levels of adverse events were observed for treatment groups compared to placebo.[99]

Recently reported results for the Phase 2 SEASIDE CHINA (NCT05017480) were comparable to those of the CBP-201-WW001 study. The SEASIDE CHINA evaluated rademikibart's efficacy and safety, when initially dosed Q2W, and Q2W or Q4W from Week 16, in Chinese adults and adolescents with moderate-to -severe atopic dermatitis.[100]

Positive results have also been released for a global Phase 2b trial (NCT04773678) evaluating rademikibart in adults with moderate-to-severe, persistent, uncontrolled asthma. In this study, 322 patients were randomized 1:1:1 to 150 mg rademikibart, 300 mg rademikibart, or placebo. Doses were administered SC every other week for 24 weeks, with both rademikibart treatment arms including a 600 mg loading dose and the placebo arm receiving a volume-matched placebo. Both rademikibart doses achieved the primary endpoint, which was an increase in prebronchodilator (trough) forced expiratory volume in the first second of expiration (FEV_1) at Week 12. Asthma Control Questionnaire (ACQ-6) scores also were significantly improved from Week 2 in treated patients compared to placebo.[101]

Telikibart (Chongqing Genrix Biopharmaceutical Co., Ltd.)

Telikibart (GR1802) is a human IgG1κ monoclonal antibody targeting IL-4 Rα developed by Genrix Bio for multiple indications, including atopic dermatitis, chronic sinusitis with nasal polyps, chronic spontaneous urticaria, seasonal allergic rhinitis and asthma. The antibody, which is Fc engineered to reduce effector function (L234F, L235E, P331S), has been designed to block IL-4, IL-13 binding to IL 4 Rα and inhibit the receptor downstream signaling involved in CD23 upregulation, thus inhibiting the Th2-type inflammatory response mediated by IL-4 or IL-13. In September 2025, Genrix Bio announced that a new drug application for treatment of moderate to severe atopic dermatitis was accepted by NMPA.[102]

The randomized, placebo-controlled Phase 3 study (NCT06216392, CTR20233857) evaluating safety and efficacy of telikibart in patients with atopic dermatitis met the primary efficacy endpoint.[102] In the study, adult patients with moderate to severe atopic dermatitis received SC telikibart injection (300 mg Q2W for 52-week treatment) or placebo (Q2W for 16-week treatment; crossover to telikibart injection for another 36 weeks). Primary endpoints were the EASI-75 response rates at Week 16, and the proportion of subjects with IGA score of 0 or 1 and a reduction of IGA score by ≥ 2 points from baseline at Week 16.

Telikibart was also reported to be well tolerated and effective in a Phase 2 study (ChiCTR2100051917) of moderate-to-severe atopic dermatitis patients, showing a dose – response trend at 150–300 mg.[103]

Telikibart is under evaluation in Phase 3 studies for several other indications, including chronic sinusitis with nasal polyps (NCT06516302), chronic spontaneous urticaria (CTR20250174), adult seasonal allergic rhinitis (NCT07154342, CTR20253037), and adolescent seasonal allergic rhinitis (NCT07199257, CTR20253870).[102]

Recibokibart (Shanghai Huaota Biopharmaceutical Co. Ltd.)

Recibokibart (HB0034) is an anti-IL-36 R humanized IgG1k monoclonal antibody developed by Shanghai Huaota Biopharmaceutical Co. Ltd. for the treatment of generalized pustular psoriasis (GPP). FDA granted recibokibart Orphan Drug designation for this indication. As of October 2025, the NMPA had accepted an application for registration and marketing authorization of recibokibart.[104]

In March 2025, Shanghai Huaota Biopharmaceutical announced that recibokibart for the treatment of acute flare-ups of GPP met its primary efficacy endpoint and all secondary efficacy endpoints in a multicenter, double-blind, randomized, placebo-controlled, parallel-controlled pivotal clinical trial.[104] In the Phase 2 NCT06231381 study, patients are given a single IV infusion of recibokibart or placebo (2:1) on D1 and remained hospitalized for at least 8 days after administration of the study drug. Follow-up of

patients continued through Week 20. Patients with GPP who participated in the NCT06231381 study were eligible to enroll in a single-arm Phase 2/3 study (NCT06477536), which evaluated the long-term safety and efficacy of 300 mg recibokibart administered IV every other four weeks.

SAL003 (Shenzhen Salubris Pharmaceuticals Co., Ltd)

SAL003 is a human anti-PCSK9 IgG4 monoclonal antibody intended for the treatment of hypercholesterolemia and mixed dyslipidemia. In September 2025, Salubris announced that a BLA for SAL003, a Class 1 biological drug, was officially accepted by the NMPA.[105]

SAL003 was evaluated in 2 placebo-controlled Phase 3 trials, CTR20232118 and CTR20233652. The CTR20232118 trial evaluated the effects of SAL003 as monotherapy for adults with hypercholesterolemia and mixed hyperlipidemia. Patients were administered SAL003 doses (140 mg SC Q4W) or placebo. The primary endpoint of the study was percentage change in LDL-C from baseline at Week 12. The study was completed in April 2025.

The CTR20233652 trial evaluated the safety and efficacy of SAL003 in combination with statins for the treatment of adults with hypercholesterolemia and mixed hyperlipidemia. Patients were treated with statins in combination with SAL003 doses (140 mg SC Q4W) or placebo over a 24-week treatment period. The primary endpoint of the study was percentage change in LDL-C from baseline at Week 24. The study was completed in May 2025.

Comekibart (China Medical System Holdings Limited)

Comekibart (MGK10) is a humanized IgG4κ monoclonal antibody that targets IL4Rα. By inhibiting IL4Rα signaling, comekibart blocks the downstream effects of both IL4 and IL13, which are key drivers of the type 2-mediated inflammation associated with diseases such as atopic dermatitis, asthma, and prurigo nodularis. Comekibart is engineered with the S228P mutation for hinge stabilization and M428L/N434S Fc mutations to increase binding affinity to FcRn and extend half-life, allowing Q4W or Q8W dosing and sustained receptor blockade.

China Medical System (CMS) Holdings Limited's Collaboration Agreement with Hunan Mabgeek Biotechnology Co., Ltd. provides CMS with co-development and exclusive commercialization rights to the comekibart in Mainland China, Hong Kong Special Administrative Region, Macao Special Administrative Region, Taiwan Region and Singapore. CMS announced that NMPA accepted an NDA for comekibart for atopic dermatitis on October 30, 2025.[106]

A randomized Phase 3 study (NCT06026891) of comekibart in atopic dermatitis patients met its primary endpoint, demonstrating clinical efficacy.[107] In the trial, comekibart SC Q4W or placebo were administered to approximately 498 patients with moderate to severe atopic dermatitis, defined by an EASI score ≥16 and other equivalent measurements, for a total of 52 weeks, followed by an 8-week follow-up. The primary endpoints were the proportions of subjects achieving EASI-75 and the proportions of subjects achieving IGA score of 0/1 point and a decrease of ≥ 2 at 16 weeks.

The safety and efficacy of comekibart are also being evaluated in Phase 3 studies of patients with asthma (NCT06837922) and prurigo nodularis (NCT06779136, CTR20244735), as well as a Phase 2/3 study (CTR20250476) of seasonal allergic rhinitis patients. In September 2025, the NMPA granted clearance for a Phase 3 trial of comekibart in chronic spontaneous urticaria patients.

Roconkibart (Shanghai Junshi Biosciences Co., Ltd)

Roconkibart (JS005) is a recombinant humanized anti-IL-17A IgG4κ monoclonal antibody, hinge stabilized with S228P mutation, evaluated in clinical studies of patients with immune-mediated disorders such as plaque psoriasis. The target is a pleiotropic cytokine that, when activated, mediates the release of inflammatory factors and plays an important role in driving psoriasis pathophysiology. In December 2025, Shanghai Junshi Biosciences Co., Ltd announced the NMPA had accepted an NDA for roconkibart for

the treatment of adult patients with moderate to severe plaque psoriasis who are candidates for systemic therapy or phototherapy.[108]

The NDA is based in part on the results of a randomized, placebo-controlled Phase 3 clinical study (NCT05975268, JS005-005-III-PsO) of patients with moderate to severe plaque psoriasis. In the JS005-005-III-PsO study, 747 patients were randomized (1:1:1) to receive 150 mg or 300 mg roconkibart or placebo. The study included a 12-week induction period, 40-week maintenance period, and an 8-week follow-up period. The study's primary objective was to determine whether the proportion of participants achieving at least a 90% improvement in Psoriasis Area and Severity Index (PASI 90) and a static Physician Global Assessment (sPGA) score of 0 or 1 at Week 12 in JS005 group were superior to that of the placebo group. Both the co-primary endpoints and key secondary endpoints showed statistically significant and clinically meaningful improvements in patients who received roconkibart.[109]

Lecankitug (Livzon Pharmaceutical Group, Beijing Xinkanghe Biopharmaceutical Technology Co., Ltd.)

Lecankitug (LZM012, XKH004) is a humanized IgG1κ monoclonal antibody that targets IL-17A/F, which are pro-inflammatory cytokines involved in the pathology of autoimmune disorders such as psoriasis and ankylosing spondylitis. In December 2025, the NMPA accepted a marketing application (CXSS2500144) for lecankitug for the treatment of psoriasis submitted by Zhuhai Livzon Mabpharm Co., Ltd., which is a subsidiary of Livzon Pharmaceutical Group, in cooperation with Beijing Xinkanghe Biopharmaceutical Technology Co., Ltd.[110]

The efficacy and safety of lecankitug vs anti-IL17A secukinumab (Cosentyx®) were evaluated in a Phase 3 study (NCT06110676/CTR20231931) of patients with moderate to severe chronic plaque psoriasis. Patients were randomized into 3 study arms to receive SC injections of: 1) 320 mg lecankitug Q4W; 2) 320 mg lecankitug Q4W until Week 12, then Q8W; or 300 mg secukinumab Q4W. The study's primary endpoint was the PASI 100 response rate at Week 12; a secondary endpoint was the PASI 100 response rate at Week 52. In July 2025, Livzon Pharmaceutical Group Inc. announced the study met its primary endpoint. The PASI 100 response rates at Week 12 were 49.5% and 40.2% for the lecankitug and secukinumab study arms, respectively. At Week 52, the PASI 100 response rates were 75.9% and 62.6% for the lecankitug 320 mg Q4W and 320 mg Q8W maintenance treatment study arms, respectively.[111]

Beijing Xinkanghe Biopharmaceutical Technology Co is evaluating the efficacy and safety of lecankitug in a randomized, placebo-controlled Phase 3 study (CTR20232310) of patients with moderately to severely active ankylosing spondylitis.

BAT5906 (Bio-Thera Solutions (Guangzhou) Co., Ltd.)

BAT5906 (维拉西塔单抗注射液), is a full-length, humanized IgG1κ monoclonal antibody that targets VEGF developed by Bio-Thera Solutions for ophthalmic disorders. The company submitted a marketing application (CXSS2500142) for BAT5906 for neovascular age-related macular degeneration (nAMD) that was accepted by NMPA in December 2025.[112]

The marketing application is supported by data from a Phase 3 trial (NCT05439629/CTR20221092) of BAT5906 compared to the anti-VEGF Fab ranibizumab (Lucentis®) in nAMD patients. The study's total enrollment was 488. Eligible participants were randomized (1:1) to receive 4 mg BAT5906 or 0.5 mg Lucentis® via intravitreal injection Q4W for 48 weeks, for a total of 13 administrations. The last follow-up was conducted at Week 52 of the study. The primary outcome measures included changes in the central retinal thickness (CRT) at Weeks 12, 24, 36, 48, and 52 and the change in best-corrected visual acuity (BCVA) from baseline at 52 weeks. The study met its primary endpoint, demonstrating that BAT5906 was significantly better than ranibizumab in reducing CRT and in improving BCVA over 52 weeks.[113]

The safety and efficacy of BAT5906 vs Lucentis are also being evaluated in late-stage clinical studies of patients with: 1) diabetic macular edema (Phase 3, CTR20243362); 2) macular edema caused by central

retinal vein occlusion (Phase 2/3, CTR20253104); and 3) choroidal neovascularization secondary to pathological myopia (Phase 2/3, CTR20253383).

Antibodies with marketing applications in review: cancer indications

Sasanlimab (Pfizer)

Sasanlimab (PF-06801591) is a human IgG4κ monoclonal antibody targeting PD-1 developed by Pfizer as an SC checkpoint inhibitor for non-muscle-invasive bladder cancer (NMIBC). Pfizer has submitted marketing application(s) for sasanlimab in combination with Bacillus Calmette-Guerin (BCG) as a treatment for NMIBC-based results of the Phase 3 CREST trial.[114]

The Phase 3 CREST trial (NCT04165317) enrolled 1055 patients with high-risk, BCG-naïve NMIBC who were randomized to receive BCG induction with sasanlimab, or BCG induction plus maintenance (I+M) with or without sasanlimab, which was administered as a 300 mg SC dose Q4W for up to 25 cycles. The trial met its primary endpoint, with a statistically significant and clinically meaningful prolongation of event-free survival (EFS) observed in patients who received sasanlimab with BCG-I+M vs those administered BCG only. The hazard ratio was 0.68 (95% CI 0.49–0.94). At 36 months, 82.1% of patients treated with sasanlimab + BCG-I+M were free of events, compared to 74.8% treated with BCG alone. EFS was not prolonged in patients administered BCG induction with sasanlimab, indicating that BCG maintenance is needed as a part of the therapeutic regimen. The complete response (CR) rates were slightly higher with sasanlimab with BCG-I+M vs those administered BCG only (89.8% vs 85.2, respectively) and Kaplan – Meier estimates indicated longer CR duration. Among patients with carcinoma in situ at randomization who achieved CR, the probability of remaining in CR at 36 months was 91.7% with the sasanlimab BCG-I+M combination versus 67.7% with BCG only.[115]

Pivekimab sunirine (AbbVie)

Pivekimab sunirine (PVEK, IMGN632) is a humanized IgG1κ ADC targeting CD123, which is overexpressed on cancer cells in several hematologic malignancies such as blastic plasmacytoid dendritic cell neoplasm (BPDCN) and acute myeloid leukemia (AML). The ADC binds CD123 and is internalized into cancer cells, where a protease-cleavable linker releases a highly potent DNA-alkylating payload, DGN549, from the indolinobenzodiazepine pseudodimer (IGN) class. The FDA granted Breakthrough Therapy designation for pivekimab sunirine for r/r BPDCN, and, in September 2025, AbbVie announced the submission of a BLA to the FDA for this indication.[116]

The BLA submission is based on data from the open-label Phase 1/2 CADENZA trial (NCT03386513), which evaluated the safety and efficacy of IV-administered pivekimab sunirine monotherapy in patients with CD123-positive AML and other CD123-positive hematologic malignancies, including untreated and r/r BPDCN. Pivekimab sunirine showed single-agent activity across multiple doses, with a recommended Phase 2 dose of 0.045 mg/kg once every 21 days. In 33 previously untreated BPDCN patients who received the Phase 2 dose of pivekimab sunirine, the composite complete response rate was 70%, ORR 85%, and median OS 16.6 months, while for r/r BPDCN patients ($n = 51$), the composite complete response rate was 14%, ORR 35%, and median OS 5.8 months.[117]

Positive results were also observed in AML patients who participated in the CADENZA Phase study.[118] The effects of pivekimab sunirine as monotherapy or in combination with Vidaza® (azacitidine) and/or Venclexta® (venetoclax) in patients with untreated and r/r AML are being further evaluated in an ongoing Phase 1/2 study (NCT04086264).

Retlirafusp alfa (Jiangsu HengRui Medicine Co. Ltd., Suzhou Suncadia Biopharmaceuticals Co., Ltd.)

Retlirafusp alfa (SHR-1701) is a bifunctional human anti-PD-L1 IgG4κ antibody immunoconjugate composed of the N-terminal-truncated extracellular domain of human-transforming growth factor (TGF)-β receptor II fused to the C-terminus of the IgG4 heavy chains via a peptidyl linker. This molecule can

simultaneously block two common immunosuppressive signaling pathways used by cancer cells. HengRui outlicensed development and sales rights to retlirafusp alfa in Korea to DONG-A ST. In September 2024, NMPA accepted HengRui's marketing application (CXSS2400101) for retlirafusp alfa for first-line treatment of locally advanced unresectable, recurrent, or metastatic G/GEJ adenocarcinoma and an approval is anticipated in 2026.[119]

In a multicenter, randomized, double-blind Phase 3 study (NCT04950322), previously untreated, unresectable locally advanced or metastatic HER2-negative G/GEJ adenocarcinoma patients were treated with retlirafusp alfa in combination with chemotherapy or placebo and chemotherapy. Patients were randomly assigned in a 1:1 ratio and received either 30 mg/kg of IV retlirafusp alpha Q3W ($n = 365$ patients) or IV placebo ($n = 366$ patients), both in combination with CAPOX chemotherapy (1000 mg/m^2 of oral capecitabine). In patients with PD-L1 CPS ≥5, the median OS was prolonged in the retlirafusp alfa arm compared to placebo (16.8 vs 10.4 months, respectively; $p < 0.0001$). Median OS was also significantly improved in the ITT population treated with retlirafusp alfa compared to placebo (15.8 vs 11.2 months, respectively; $p < 0.0001$). Comparable percentages of Grade ≥3 treatment-related adverse events were observed in the two arms.[120]

Anbenitamab (Alphamab Oncology, JMT-Bio Technology Co., Ltd.)

Anbenitamab (KN026) is a humanized, bispecific antibody targeting two non-overlapping epitopes of HER2 resulting in HER2 signal blockade. The biparatopic antibody has been developed using CRIB (Charge Repulsion Induced Bispecific), Alphamab's proprietary Fc-based heterodimer bispecific platform technology.[121]

Alphamab Oncology granted JMT-Bio, a wholly owned subsidiary of the CSPC Pharmaceutical Group, exclusive rights for the development and commercialization of anbenitamab in mainland China. NMPA has granted Breakthrough Therapy designation to anbenitamab in combination with chemotherapy for previously treated HER2-positive gastric cancer patients. In September 2025, Alphamab Oncology announced that NMPA accepted an NDA for anbenitamab for use in combination with chemotherapy for the treatment of patients with HER2-postive locally advanced, recurrent, or metastatic gastric or gastroesophageal junction cancer who failed at least one prior systemic therapy, including trastuzumab in combination with chemotherapy.[122]

The NDA submission was based in part on results from the two-stage (open-label, randomized) Phase 2/3 trial (KN026-001; NCT05427383) evaluating anbenitamab in combination with chemotherapy (paclitaxel, docetaxel, or irinotecan) for the treatment of HER2-postive gastric cancer patients who had failed first-line therapy. In the open-label stage, the study evaluated the safety and efficacy of anbenitamab (IV at 30 mg/kg on Day 1, Q3W) and chemotherapy when given in combination, while the randomized stage evaluated OS and PFS in patients receiving anbenitamab and chemotherapy compared to those receiving placebo and chemotherapy. The pre-specified endpoint of PFS and OS were met with both statistical significance and clinical relevance. The median PFS was 7.1 months in the anbenitamab group compared with 2.7 months in the control group (HR = 0.25, $p = 5.44 \times 10^{-12}$). The median OS was 19.6 months (not mature) in the anbenitamab group compared with 11.5 months in the control group (HR = 0.29, $p = 1.56 \times 10^{-6}$).[123]

Anbenitamab is also being evaluated in two Phase 3 trials in combination with HB1801, an albumin-bound docetaxel formulation (NCT05838066, KN026-003) and carboplatin (NCT06747338, KN026-004), both for the treatment of HER2-positve breast cancer.

Izalontamab brengitecan (Sichuan Biokin Pharmaceutical Co., Ltd., Bristol Myers Squibb)

Izalontamab brengitecan (Iza-bren, BL-B01D1, BMS-986507) is a tetravalent, bispecific ADC targeting EGFR and HER3 being developed as a treatment for solid tumors by Sichuan Biokin Pharmaceutical Co., Ltd. (Biokin) in China and by SystImmune, which is a Biokin subsidiary, and Bristol Myers Squibb in territories outside of China.[124] Iza-bren is composed of an anti-EGFRxHER3 antibody conjugated to a topoisomerase I inhibitor (camptothecin derivative Ed-04; DAR 8) via a cathepsin B cleavable linker. FDA granted Breakthrough Therapy designation to iza-bren for the treatment of locally advanced or metastatic NSCLC with EGFR exon 19 deletions or exon 21 L858R substitution mutations whose disease

has progressed on or after treatment with an EGFR tyrosine kinase inhibitor and platinum-based chemotherapy. As of November 2025, NMPA is evaluating an NDA (CXSS2500124) for iza-bren for patients with recurrent or metastatic nasopharyngeal carcinoma who have previously received PD-1/PD-L1 monoclonal antibody therapy and have failed at least two lines of chemotherapy (at least one line of platinum-based chemotherapy).

Iza-bren was evaluated in a randomized, open label Phase 3 (NCT06118333/CTR20233419) study in 386 patients with recurrent or metastatic nasopharyngeal carcinoma who had failed at least two lines of platinum-based chemotherapy after receiving PD-1/PD-L1 monoclonal antibody as the last line of therapy. Patients received either iza-bren at 2.5 mg/kg IV ($n = 191$) on days 1 and 8 of each 3-week cycle or physician's choice of chemotherapy ($n = 195$). The study's primary endpoints were ORR assessed by masked ICR as per RECIST version 1.1 criteria, and OS. Results of the first planned interim analysis were recently reported.[125] The ORR was 54.6% (95% CI 45.2–63.8%) in the iza-bren study arm and 27.0% (19.1–36.0%) in the chemotherapy study arm (difference 27.9%, 95% CI 15.5–39.4%; $p < 0{\cdot}0001$) at median follow-ups of 7.66 and 7.1 months, respectively. OS data was not reported, as the data was not mature at the time of data cutoff. Median PFS was 8.38 vs 4.34 months (hazard ratio = 0.44, 95% CI = 0.32–0.62) and rates at 6 and 9 months were 83.4% vs 66.6% and 60.9% vs 29.1% for the iza-bren and chemotherapy study arms, respectively.

Iza-bren is also currently being evaluated in multiple ongoing late-stage clinical trials, including Phase 2/3 (IZABRIGHT-Lung01, (NCT07100080) and Phase 3 (NCT06382129, NCT06382116, NCT06838273) studies in NSCLC; Phase 2/3 (IZABRIGHT-Bladder01, NCT07106762) and Phase 3 (NCT06857175) studies in urothelial cancer; Phase 2/3 (IZABRIGHT-Breast01 (NCT06926868) and Phase 3 (NCT06382142) studies in triple-negative breast cancer, as well as Phase 3 studies in HR+HER2- breast cancer (NCT06343948), ovarian cancer ((NCT06994195), esophageal squamous cell carcinoma (NCT06304974), and small cell lung cancer (NCT06500026).

Antibodies to watch in 2026

The phrase "Antibodies to Watch" refers to antibody therapeutics currently in late-stage studies that may be the subject of a marketing application in the upcoming year, as determined by public disclosures from the sponsoring company or companies and the estimated primary completion dates of ongoing late-stage clinical studies. Based on such data, we project that marketing applications for 21 investigational antibody therapeutics, with 16 for non-cancer indications (Table 3) and 5 for cancer (Table 4), may be submitted by the end of 2026. These molecules are discussed below, ordered according to our estimated dates for possible submission of their first marketing application. Supplemental Table S2 includes the full commercial late-stage clinical pipeline, comprising 209 antibody therapeutics that, to the best of our knowledge, fit our inclusion criteria, as of December 31, 2025.

Antibodies to watch: non-cancer indications

Garetosmab (Regeneron Pharmaceuticals, Inc.)

Garetosmab (REGN2477) is a human IgG4κ monoclonal antibody that inhibits activin A, but does not bind other TGF-β family members. The antibody was created using Regeneron's VelocImmune mice and engineered with the S228P mutation for IgG4 hinge stabilization. The antibody is being developed as a treatment for fibrodysplasia ossificans progressiva (FOP), which is a rare genetic disorder characterized by the creation of bone lesions (heterotopic ossification (HO)) of skeletal muscles, tendons, ligaments, and fascia. FDA granted Fast Track designation for garetosmab for the prevention of HO in patients with FOP. In addition, garetosmab was granted Orphan designation the US and EU. In September 2025, Regeneron announced the primary endpoint was met in the Phase 3 OPTIMA (NCT05394116) trial investigating garetosmab in adults with FOP and that a US regulatory submission of garetosmab in adults is planned for year-end 2025.[126]

In the OPTIMA study, patients received 3 mg/kg garetosmab ($n = 19$), 10 mg/kg garetosmab ($n = 23$), or placebo ($n = 21$) IV once Q4W for 56 weeks. The study's primary endpoint was the total number of new HO

Table 3. Commercially sponsored investigational monoclonal antibodies in late-stage clinical studies for non-cancer indications, with regulatory submission anticipated during 2025–2026. Table includes information found in the public domain as of December 1, 2025 molecules are listed in chronological order according to the estimated marketing application submission dates. Abbreviations: AOC, antibody-oligo conjugate; BAFF-R, B-cell activating factor receptor; DR, death receptor; fab, antigen-binding fragment; IGF-1 R, insulin-like growth factor 1 receptor; IL, interleukin; SCLC, small cell lung cancer; TfR, transferrin receptor. Supplemental Table S3 (excel format) includes all data in Tables 1-4.

INN	Target(s); Format	Indication(s) of most advanced study	Most advanced clinical phase
Garetosmab	Activin A; Human IgG4κ	Fibrodysplasia ossificans progressiva	Phase 3
Zeleciment rostudirsen	TfR; Fab; AOC	Duchenne muscular dystrophy	Phase 1/2 pivotal
Delpacibart zotadirsen	TfR; Humanized IgG1κ; AOC	Duchenne muscular dystrophy	Phase 2 pivotal
Ianalumab	BAFF-R; Human IgG1κ	Sjögren's syndrome	Phase 3
Tanruprubart	Complement C1q; Humanized IgG4κ	Guillain-Barré Syndrome	Phase 3
Obexelimab	CD19; Humanized IgG1κ	Immunoglobulin G4 related disease	Phase 3
Delpacibart etedesiran	TfR; Humanized IgG1κ; AOC	Myotonic dystrophy type 1	Phase 3
Tarcocimab tedromer	VEGF; Humanized IgG1k; Immunoconjugate	Diabetic retinopathy, neovascular age-related macular degeneration, retinal vein occlusion	Phase 3
Tozorakimab	IL-33; Human IgG1λ	Chronic obstructive pulmonary disease; lower respiratory tract disease	Phase 3
Ersodetug	Insulin receptor; Human IgG2κ	Hyperinsulinism	Phase 3
Crusekitug	IL-17A; Humanized IgG1	Ankylosing spondylitis	Phase 3
Oturkibart	IL-4 R alpha; Humanized mAb	Prurigo nodularis, atopic dermatitis	Phase 3
LP-003	IgE; Humanized mAb	Seasonal allergic rhinitis	Phase 3
Elegrobart	IGF-1 R; Humanized IgG1κ	Thyroid eye disease	Phase 3
Camtarkibart	IL-4 R alpha; Humanized IgG4κ	Atopic dermatitis, Obstructive pulmonary disease, Chronic rhinosinusitis with nasal polyps	Phase 3
Manfidokimab	IL-4 R alpha; Humanized IgG4κ	Atopic dermatitis	Phase 3

Table 4. Commercially sponsored investigational monoclonal antibodies in late-stage clinical studies for cancer indications, with regulatory submission anticipated during 2025–2026. Table includes information found in the public domain as of December 1, 2025; molecules are listed in chronological order according to the estimated marketing application submission dates. Abbreviations: ADC, antibody-drug conjugate; DR, death receptor; EGFR, epidermal growth factor receptor; HER, human epidermal growth factor receptor. Supplemental Table S3 (excel format) includes all data in Tables 1-4.

INN	Target(s); Format	Indication(s) of most advanced study	Most advanced clinical phase
Ozekibart	DR5; Humanized mAb	Chondrosarcoma	Phase 2 pivotal
Sonesitatug vedotin	Claudin-18.2; Humanized IgG1k; ADC	Gastric cancer	Phase 3
Trastuzumab pamirtecan	HER2; Humanized IgG1k; ADC	Endometrial cancer	Phase 3
Ifinatamab deruxtecan	B7-H3; Humanized IgG1k; ADC	SCLC, prostate cancer, esophageal squamous cell carcinoma	Phase 3
Petosemtamab	EGFR, LGR5; Human IgG1k; Bispecific	Head and neck cancer	Phase 3

lesions in the total treated population and the reduction compared to placebo. At 56 weeks, both doses of garetosmab were highly efficacious in reducing the number of HO lesions as compared to placebo, demonstrating a 94% and 90% reduction, respectively.[126]

Zeleciment rostudirsen (Dyne Therapeutics, Inc.)

Zeleciment rostudirsen (DYNE-251, z-rostudirsen) is an antibody – oligonucleotide conjugate (AOC) developed by Dyne Therapeutics as a treatment for Duchenne muscular dystrophy (DMD) patients who are amenable to exon 51 skipping. DMD is a rare genetic disease resulting from a mutation in the DMD gene on the X chromosome. This gene regulates the production of dystrophin, a protein essential to healthy muscle development and function. Children born with this disorder, which typically affects males, may experience permanent damage to muscle cells, leading to loss of muscle strength and function within the first 3–5 years of life. Zeleciment rostudirsen, which consists of a phosphorodiamidate morpholino oligomer conjugated to a Fab

that binds to transferrin receptor 1 (TfR1), is designed to restore dystrophin expression and thus provide functional improvement.

Zeleciment rostudirsen has been granted Breakthrough Therapy, Fast Track, Orphan Drug, and Rare Pediatric Disease designations for DMD from FDA. Dyne Therapeutics anticipates submission of a BLA to FDA in the second quarter of 2026 for a potential accelerated approval of zeleciment rostudirsen for DMD.[127]

Clinical data from the randomized, placebo-controlled Phase 1/2 DELIVER study (NCT05524883) of zeleciment rostudirsen administered IV to male patients (4–16 years old) with a confirmed diagnosis of DMD, and with a mutation in the dystrophin gene characterized by exon deletion amenable to exon 51 skipping, showed sustained functional improvements in multiple functional endpoints through 18 months. Significant improvements were observed in both the 20 mg/kg (selected registrational dose) and 10 mg/kg Q4W cohorts, through 12 and 18 months, respectively. Zeleciment rostudirsen dosed at 10 or 20 mg/kg Q4W also demonstrated a favorable safety profile with no new treatment-related serious adverse events reported.[128] The registrational expansion cohort (20 mg/kg Q4W) of DELIVER ($N = 32$, 3:1 randomization for zeleciment rostudirsen vs placebo) met its primary endpoint, showing a statistically significant increase in dystrophin to 5.46% at 6 months (muscle content-adjusted; $p < 0.0001$).[127]

Delpacibart zotadirsen (Avidity Biosciences, Inc.)

Delpacibart zotadirsen (AOC 1044, del-zota) is an AOC developed by Avidity Biosciences. It consists of a humanized IgG1κ monoclonal antibody targeting transferrin receptor 1 (TfR1), conjugated to phosphorodiamidate morpholino oligomers (PMOs), and Fc engineered to reduce cell mediated effector functions (L234A, L235A, L328R). This design enables targeted delivery of PMOs to skeletal and cardiac muscle, where they promote skipping of exon 44 of the dystrophin gene, thereby restoring dystrophin production in patients with DMD amenable to exon 44 skipping (DMD44).[129] Duchenne muscular dystrophy is a severe X linked recessive neuromuscular disorder that primarily affects males and is characterized by progressive muscle weakness and degeneration.

Del-zota has received FDA's Rare Pediatric Disease, Orphan Drug, Fast Track, and Breakthrough Therapy designations and EMA Orphan Drug designation for DMD44. Avidity Biosciences aligned with the FDA during a pre-BLA meeting and plans to submit a BLA for accelerated approval for delpacibart zotadirsen in patients with DMD44 in 2026.[130] Data from the EXPLORE44™ program may support the submission. Given the promising results of del-zota, the company announced that a Managed Access Program will start enrolling by the end of 2025, under an FDA-authorized treatment protocol, del-zota will be available for the treatment of eligible boys and men with DMD44 through participating healthcare providers. Following the success of Avidity Bioscience's AOC pipeline for neuromuscular disorders, the company recently announced a merger agreement with Novartis AG.[131]

The safety and efficacy of del-zota in DMD44 patients were evaluated in the 2-part, placebo-controlled Phase 1/2 EXPLORE44® (NCT05670730) and the open-label Phase 2 EXPLORE44 OLE™ (NCT06244082) trials. EXPLORE44® study consisted in two parts: Part A assessed the effects of del-zota in 5 single-dose cohorts of approximately 40 healthy volunteers, and Part B assessed the effects of multiple-ascending doses of del-zota in DMD44 patients. Part B enrolled a total of 26 DMD44 patients who received either del-zota IV (5 mg/kg Q6W or 10 mg/kg Q8W) or placebo. Del-zota treatment resulted in robust dystrophin restoration (mean of ~25% of normal, with up to 58% in some participants), sustained creatine kinase reductions (> 80% from baseline) with 50% of participants showing normal creatine kinase levels at one year from treatment. Eligible participants from Part B could participate in the EXPLORE44 OLE™ study where they received 5 mg/kg del-zota treatment (Q6W) for approximately 24 months. The open-label extension study EXPLORE44 OLE™ enrolled a total of 39 participants, including 23 from the EXPLORE44® study. A total of 17 DMD44 patients (12 ambulatory and 5 non-ambulatory) who began on the del-zota treated arm of EXPLORE44® part B and continued into the EXPLORE44-OLE™ were followed for approximately one year. Clinical outcomes comparison of these 17 patients versus a natural history group of 22 patients (NCT01753804) showed clinically meaningful functional improvements. Gains were observed across stair climb, walk/run, rise from floor, and upper limb function, with North Star Ambulatory Assessment stabilization contrasting with the decline observed in the natural history group. Del-zota was generally

well tolerated, with most adverse events mild or moderate and only one discontinuation due to hypersensitivity.[132]

Ianalumab (Novartis)

Ianalumab (VAY736) is a human IgG1κ monoclonal antibody that combines B-cell depletion with B-cell activating factor receptor (BAFFR) inhibition, a dual mechanism that underpins its potential across a range of B-cell-mediated autoimmune diseases. The antibody was originally developed by MorphoSys AG, which was acquired by Novartis in 2024. Ianalumab received Fast Track Designation from the FDA for ianalumab in Sjögren's syndrome and an Orphan Medicinal Produce designation in the EU for ianalumab for the treatment of immune thrombocytopenia. Novartis plans regulatory submissions in Sjögren's syndrome in the first half of 2026.[133]

Novartis recently reported positive top line results from the 2-arm Phase 3 NEPTUNUS 1 (NCT05350072) and 3-arm NEPTUNUS 2 (NCT05349214) trials of ianalumab in Sjögren's syndrome patients.[133] The NEPTUNUS 1 study evaluated the effects of 300 mg ianalumab dosed SC once monthly compared to placebo for 52 weeks and enrolled 275 patients, while the NEPTUNUS 2 study evaluated two ianalumab exposure levels (300 mg SC monthly or every 3 months for up to 52 weeks) compared to placebo and enrolled 504 patients. The primary endpoint of both studies was the change from baseline in EULAR Sjögren Syndrome Disease Activity Index (ESSDAI) score at Week 48 as compared to placebo. Monthly treatment with ianalumab achieved statistically significant improvements in disease activity, meeting the primary endpoint in both studies. Data also showed consistent improvements across secondary endpoints, and a favorable safety profile.[134]

Ianalumab is also being evaluated in Phase 3 studies as a treatment for other indications, including primary immune thrombocytopenia (IV) (NCT05653219), systemic lupus erythematosus (SC) (SIRIUS-SLE 1, NCT05639114; SIRIUS-SLE 2, NCT05624749), and warm autoimmune hemolytic anemia (VAYHIA, NCT05648968), reflecting its broader potential in B-cell-driven autoimmune conditions.

Tanruprubart (Annexon Inc.)

Tanruprubart (ANX005) is humanized IgG4κ monoclonal antibody that binds with high affinity to C1q, developed by An nexon Biosciences. The antibody is engineered with S228P mutation for IgG4 hinge stabilization and L235E mutation to reduce ADCC and CDC effector functions, and is designed to bind and block C1q, a protein within the C1 complex, a key component of the immune system's complement cascade. Tanruprubart has received FDA's Fast Track and Orphan Drug designations, as well as EMA's Orphan Medicinal Product designation, for the treatment of Guillain-Barré Syndrome (GBS). An nexon plans to submit an MAA in the EU in January 2026, with regulatory discussions also ongoing with the FDA.[135]

The safety and efficacy of tanruprubart in the treatment of GBS were evaluated in a placebo-controlled Phase 3 trial (ANX005-GBS-02, NCT04701164). In this randomized study, patients received a single IV infusion of tanruprubart (30 mg/kg or 75 mg/kg) or placebo. The 30 mg/kg dose met the primary endpoint of functional improvement on the GBS Disability Scale (GBSDS) at Week 8, while the 75 mg/kg dose did not.[130] Compared with placebo, tanruprubart 30 mg/kg was associated with 2.4 fold higher odds of functional improvement at Week 8, a median 31day earlier return to independent walking, and 28 fewer days on mechanical ventilation. Safety was comparable across groups, with balanced rates of serious adverse events and only transient infusion-related reactions in tanruprubart-treated patients.[135,136]

Obexelimab (Zenas Biopharma, Bristol Myers Squibb)

Obexelimab (ZN012, XmAb5871) is a humanized, Fc engineered, bifunctional IgG1κ monoclonal antibody designed to selectively inhibit B-cell activity through a dual targeting mechanism. It binds CD19 on B cells with its variable domain while simultaneously engaging the inhibitory FcγRIIb receptor via its XmAb engineered Fc domain (S267E, L328F), thereby mimicking the natural antigen – antibody complex and delivering a potent inhibitory signal.[137] The antibody was originally developed by Xencor, which pioneered

the XmAb immune inhibitor Fc domain technology. Zenas BioPharma acquired exclusive worldwide rights to obexelimab in 2021. In 2023, Zenas BioPharma entered into a strategic license and collaboration agreement with Bristol Myers Squibb (BMS), granting BMS rights to develop and commercialize obexelimab for autoimmune diseases in Japan, South Korea, Taiwan, Singapore, Hong Kong, and Australia.

Zenas BioPharma anticipates topline data readout for the Phase 3 INDIGO study of obexelimab in immunoglobulin G4-related disease (IgG4-RD) by the end of 2025. If results are positive, the company expects to submit a BLA for obexelimab in this indication in the second quarter of 2026, with approval and launch then anticipated in the first half of 2027.[137]

The effects of obexelimab in IgG4RD patients are being evaluated in the placebo-controlled Phase 3 INDIGO trial (NCT05662241). Patients receive obexelimab SC Q1W (n = ~100) or placebo (n = ~100) for 52 weeks. The primary endpoint is time to disease flare through Week 52, and the estimated primary completion date is in November 2025. The 1-year randomized control period will be followed by an additional 3-year open-label extension period; thus, the estimated completion date of the study is in February 2029.

Zenas BioPharma is also evaluating the effects of obexelimab in Phase 2 studies of relapsing multiple sclerosis (MoonStone, NCT06564311) and SLE patients (SunStone, NCT06559163). Obexelimab met the primary endpoint of the MoonStone trial at Week 8 and 12. The company expects 24-week MoonStone trial results in Q1 2026 as well as topline results from the Phase 2 SunStone trial in mid-2026.[138]

Delpacibart etedesiran (Avidity Biosciences, Inc.)

Delpacibart etedesiran (AOC 1001, deldesiran) is an AOC designed to address the underlying cause of myotonic dystrophy type 1 (DM1) by reducing levels of *DMPK* mRNA, the disease-related transcript Deldesiran consists of a humanized anti-TfR1 IgG1κ monoclonal antibody Fc engineered to reduce effector function (L234A, L235A, L328R) and conjugated to a small interfering RNA (siRNA) directed against *DMPK* mRNA, enabling targeted delivery to muscle tissue.

Deldesiran was granted Breakthrough Therapy, Fast Track, and Orphan Drug designations by the FDA, as well as Orphan Medicinal Product and Orphan Drug designations from EMA and the Japanese Ministry of Health, Labour and Welfare, respectively, for the treatment of DM1. Avidity Biosciences anticipates topline data readout from the Phase 3 HARBOR study in the second half of 2026.[139] If positive, study results may enable a BLA submission by the end of 2026, but the timing of such a submission may be affected by the company's recently announced merger agreement with Novartis AG.[131]

The safety and efficacy of deldesiran were evaluated in the placebo-controlled Phase 1/2 MARINA trial (NCT05027269) and open-label Phase 2 MARINAOLE study (NCT05479981), which enrolled DM1 patients who were previously enrolled in the MARINA® trial. In the MARINA™ trial, DM1 patients were randomized to receive 1, 2, or 4 mg/kg deldesiran IV (n = 6, 9, and 13, respectively) or placebo (total n = 10) Q6W. Of patients who participated in the MARINAOLE™ study, patients who received 2 mg/kg doses in the MARINA trial were escalated to 4 mg/kg, while those on the 4 mg/kg dose stayed on the 4 mg/kg dose in the open-label extension trial. In these studies, deldesiran achieved targeted delivery of siRNA to skeletal muscle, resulting in *DMPK* reduction and increased estimated functional muscleblind-like protein levels. Clinical activity was demonstrated by early and sustained improvements in myotonia, with one year data from the MARINAOLE™ study confirming gains in muscle strength and DM1-Activ scores. Deldesiran was well tolerated, with nausea and headache the most common adverse events, and no new safety signals observed.[140]

The randomized Phase 3 HARBOR (NCT06411288) study has completed enrollment.[139] The study is evaluating whether a regimen of deldesiran IV 4 mg/kg Q8W, with an initial loading dose of 2 mg/kg, is more effective than placebo over a 54-week treatment period in participants (aged 16+ years) with a clinical and genetic diagnosis of DM1. The primary outcome measure is hand function assessed through Week 30. The study has enrolled 159 participants and has a primary completion date in March 2026.

Tarcocimab tedromer (Kodiak Sciences Inc.)

Tarcocimab tedromer (KSI-301) is an antibody-biopolymer conjugate developed by Kodiak Sciences to inhibit VEGF-A. The humanized IgG1κ monoclonal antibody incorporates Fc mutations that reduce effector function (L234A, L235A, G237A) and enable site-specific conjugation (L443C) to a phosphocholine-based biopolymer. Biopolymer conjugation increases the molecular size of the antibody, slowing its clearance from the eye, thereby enabling extended intraocular retention. VEGF-A plays a role in pathological angiogenesis and vascular leakage in retinal diseases such as diabetic retinopathy (DR), neovascular age-related macular degeneration (nAMD), and retinal vein occlusion (RVO). Kodiak Sciences has conducted Phase 3 studies of tarcocimab tedromer in all three indications, and aims to submit a single BLA covering these indications (DR, nAMD, RVO) in Q3 2026.[141]

Tarcocimab tedromer, which is administered via intravitreal injection, has a 20-day ocular half-life, enabling dosing intervals of 3 to 6 months. A 5 mg dose contains 20% unconjugated and 80% conjugated antibody to provide immediate effects and sustained durability, respectively.[141]

The Phase 3 GLOW (NCT05066230) study evaluated the efficacy and safety of tarcocimab tedromer in patients with non-proliferative DR. Patients were administered intravitreal injection of tarcocimab tedromer (5 mg) in three initiating doses, and then every 24 weeks through Week 92 or a sham injection, a procedure that mimics an intravitreal injection, on the same schedule. The study met its primary endpoint and all key secondary endpoints, showing clinically meaningful superiority of tarcocimab treatment over sham at 48 weeks.[142] The Phase 3 GLOW2 (NCT06270836) study design mirrors that of the GLOW1 study. Topline clinical data of the confirmatory GLOW2 study is expected in 1Q 2026.

In the nAMD indication, the Phase 3 DAYLIGHT study (NCT04964089) demonstrated tarcocimab tedromer's favorable safety and non-inferior efficacy compared with aflibercept at Year 1.[141] The ongoing Phase 3 DAYBREAK study (NCT06556368) is evaluating efficacy and safety of intravitreal tarcocimab tedromer and tabirafusp tedromer (KSI-501) compared with intravitreal aflibercept in nAMD patients. Patients that receive tarcocimab tedromer are given intravitreal injections once Q4W for four monthly doses followed by individualized dosing every 4 to 24 weeks. Topline data from this study is expected in mid-2026.

In the RVO indication, the Phase 3 BEACON (NCT04592419) trial demonstrated efficacy and non-inferiority to aflibercept while doubling the treatment interval in patients with visual impairment due to treatment-naïve macular edema secondary to retinal vein occlusion.[141]

Tarcocimab tedromer has shown negative results in some late-stage studies. For example, the Phase 2/3 DAZZLE (NCT04049266) trial in nAMD failed due to undertreatment of a subset of patients, and the GLEAM (NCT04611152) and GLIMMER (NCT04603937) trials in DME did not meet endpoints, with an unexpected increase in cataracts observed in the patients.[143]

Tozorakimab (AstraZeneca)

Tozorakimab (MEDI3506) is a human IgG1λ monoclonal antibody developed by AstraZeneca targeting interleukin-33 (IL-33), a broad-acting 'alarmin' cytokine that is released during tissue damage or infection. Tozorakimab neutralizes both reduced and oxidized forms of IL-33 due to its femtomolar affinity and rapid association kinetics. It inhibits downstream signaling through serum-stimulated 2 (ST2) and prevents IL-33 oxidation that activates receptor for advanced glycation end products (RAGE)/EGFR. This dual mechanism reduces inflammation and promotes epithelial repair in IL-33-driven diseases such as chronic obstructive pulmonary disease (COPD) and lower respiratory tract disease (LRTD) caused by viral lung infections.[144]

Tozorakimab is being evaluated in five late-stage clinical studies in patients with COPD and severe viral LRTD. The FDA has granted tozorakimab Fast Track designations for both indications. AstraZeneca anticipates data readouts in H1 and H2 2026 for key Phase 3 studies in both indications.[145] If positive, the clinical study results may support a marketing application submission by the end of 2026.

In the COPD indication, the effects of tozorakimab were evaluated in the Phase 2a FRONTIER-4 trial (NCT04631016) in patients with moderate-to-severe COPD with chronic bronchitis receiving dual or triple

inhaled therapy. In this study, patients received 600 mg tozorakimab SC Q4W for 24 weeks or placebo. The primary endpoint of change in pre-bronchodilator forced expiratory volume in 1 s (FEV1) from baseline to week 12 was not met in the intent-to-treat population, but treatment with tozorakimab was well-tolerated and showed improvements in lung function and reduced exacerbation risk in COPD patients with chronic bronchitis.[146]

Three placebo-controlled Phase 3 studies are evaluating the safety and efficacy of tozorakimab in COPD patients. Data from these studies are anticipated in H1 2026.[145] The OBERON (NCT05166889) and TITANIA (NCT05158387) studies are assessing the effects of tozorakimab in adult symptomatic COPD patients with a history of exacerbations, using two SC dose regimens as add-on to optimized treatment with maintained inhaled therapy, while the MIRANDA (NCT06040086) study is evaluating a single SC dose, with SoC, in the same patient population. In addition, patients who completed the OBERON or TITANIA trials may participate in the ongoing, long-term extension PROSPERO (NCT05742802) study.

In the LRTD indication, the placebo-controlled TILIA study (NCT05624450) is investigating the safety and efficacy of IV-administered tozorakimab in hospitalized adults with acute respiratory failure due to viral lung infection requiring supplemental oxygen, with the aim of preventing death or progression to invasive mechanical ventilation or extracorporeal membrane oxygenation. Data readout is anticipated in H2 2026.[145]

Ersodetug (Rezolute, Inc.)

Ersodetug (RZ358, XOMA 358) is a human IgG2 κ monoclonal antibody that allosterically binds to the insulin receptor to reduce excessive signaling caused by insulin or insulin-like substances. This mechanism of action enables the treatment of both congenital and acquired forms of life-threatening hyperinsulinism (HI), including tumor-induced HI, a rare condition characterized by life-threatening hypoglycemia due to insulinomas or non-islet cell tumors that secrete IGF-2. Originally developed by XOMA Corporation, global development and commercialization rights for ersodetug were licensed to Rezolute, Inc. in 2017.

Rezolute is evaluating ersodetug as a treatment for congenital and tumor-induced HI.[147] Topline data from the Phase 3 sunRIZE study in congenital HI are expected in December 2025, and the FDA has indicated that the study meets registrational requirements for a BLA submission and review.[148] FDA is also aligned with the company with respect to a streamlined clinical development path for ersodetug for the treatment of tumor-induced HI. Rezolute anticipates topline results from the single-arm, open-label, Phase 3 upLIFT study of ersodetug in patients with tumor-induced HI the second half of 2026. If results of these studies are positive, one or more BLAs for ersodetug could be submitted in 2026.

Ersodetug has received numerous regulatory agency designations designed to facilitate development of innovative drugs for life-threatening or rare disorders. Ersodetug was granted a PRIME designation by the EMA and an Innovation Passport designation by the U.K. Innovative Licensing and Access Pathway (ILAP) Steering Group for the treatment of congenital HI. Ersodetug also received FDA's Orphan Drug and Rare Pediatric Disease designation and Orphan Medicinal Product designation in the EU for the treatment of insulinoma, the primary cause of islet cell tumor hypoglycemia, as well as FDA's Breakthrough Therapy and Orphan Drug designations for the treatment of hypoglycemia caused by tumor-induced HI.[147]

The randomized, double-blind, placebo-controlled Phase 3 sunRIZE study (NCT06208215) is evaluating the effects of ersodetug in congenital HI patients aged 3 months to 45 years. Patients aged ≥1 year are administered three bi-weekly loading doses, then 4 monthly doses over a total 6-month treatment period of ersodetug at doses of 5 ($n = 16$) or 10 mg/kg ($n = 16$) or placebo ($n = 16$), with SoC. Patients aged 3 months to < 1 year are enrolled in an open label parallel arm and administered SoC plus ersodetug, starting at 5 mg/kg and increasing to 10 mg/kg, as needed, per the protocol schedule, or placebo. The study's primary endpoint is the change in average number of hypoglycemia events per week. Top-level data readout is expected in December 2025.[147]

The Phase 3 upLIFT study (NCT06881992) is a single-arm, open-label trial in up to 16 adult islet cell tumor (ICT) or non-islet cell tumor (NICT) patients with HI who have not achieved adequate hypoglycemia control with SoC therapies. Patients receive Q1W administration of 9 mg/kg of ersodetug as add-on to SoC over an 8-week pivotal treatment period. The study's primary endpoint is the number of participants

achieving ≥50% reduction from baseline IV glucose requirements. Topline results from the upLIFT study are anticipated in the second half of 2026.[147]

Crusekitug (Jiangsu Qyuns Therapeutics Co. Ltd.)

Crusekitug (QX002N) is a high-affinity recombinant humanized IgG1κ mAb that targets human IL-17A, including IL-17AA and IL-17AF. By blocking the binding of IL-17A to its receptor complex, crusekitug prevents activation of pro-inflammatory signaling pathways that drive the pathology of immune-mediated diseases such as ankylosing spondylitis (AS). Based on positive results of a Phase 3 trial of patients with AS conducted in China, Qyuns Therapeutics plans to submit a BLA by the end of 2025.[149]

The Phase 3 study (CTR20232574) included a total of 641 patients with moderate-to-severe active AS. Patients in the treatment group (322) were administered crusekitug (160 mg) SC Q4W, while 319 patients were administered placebo. The primary and secondary efficacy endpoints were the ASAS40 and ASA20 response rates, respectively, at Week 16. The ASAS40 response rate at Week 16 in the treatment group was 40.4%, vs 18.9% in the placebo group ($p < 0.0001$). Also at Week 16, the ASAS20 response rate of patients who received crusekitug was 65.2%, vs 41.3% for those who were administered placebo. The results confirmed that the trial successfully met both its primary endpoint and key secondary endpoints.[150]

Oturkibart (Jiangsu Qyuns Therapeutics Co. Ltd., Hangzhou Zhongmei Huadong Pharmaceutical Co. Ltd.)

Oturkibart (QX005N, HDM3016) is a hinge stabilized (S228P) humanized IgG4κ mAb designed to inhibit IL-4 Rα. By binding IL-4 Rα the antibody blocks the interaction of IL-4 Rα with both IL-4 and IL-13, thereby inhibiting the signaling pathways and biological effects of these cytokines, which have roles in type 2 inflammatory allergic diseases such as prurigo nodularis (PN) and atopic dermatitis. The NMPA granted oturkibart Breakthrough Therapy designation for PN in January 2024 based on results of a Phase 2 study (CTR20223174). Qyuns Therapeutics into a cooperation agreement with Zhongmei Huadong for co-development of oturkibart in July 2024. Primary endpoint data readouts for Phase 3 studies of patients with PN and atopic dermatitis are expected by the end of 2025 and in early 2026, respectively. If these results are positive, Qyuns Therapeutics plans to submit marketing applications in China for oturkibart as a treatment for PN in the first half of 2026 and for atopic dermatitis in the second half of 2026.[151]

Patient enrollment for the Phase 3 trials in China for oturkibart in PN and atopic dermatitis was completed in March 2025 and August 2025, respectively. In the placebo-controlled Phase 3 study in PN (CTR20241660, ChiCTR2400082720), a total of 412 patients received SC injection of 450 mg oturkibart or placebo Q2W. The study's primary outcome measure is the proportion of subjects whose weekly mean value of the Worst Itch Numerical Rating Scale (WI-NRS) decreased by 4 points or more from baseline at Week 24. In the placebo-controlled Phase 3 in atopic dermatitis (CTR20241068/ChiCTR2400082722), an estimated 648 patients received SC injection of 300 mg or 450 mg oturkibart or placebo Q2W. The study's primary outcome measure is the proportion of subjects achieving EASI-75 and proportion of subjects with an IGA score of 0 or 1 with a decrease of ≥ 2 points from baseline at Week 16.

LP-003 (LongBio Pharma)

LP003 is a humanized monoclonal antibody targeting immunoglobulin E (IgE), developed by LongBio Pharma, and designed to treat a broad range of allergic and inflammatory diseases. Compared with first-generation antiIgE therapies such as omalizumab, LP003 has been engineered with higher binding affinity for IgE, stronger inhibition of FcεRI signaling, and an extended halflife, enabling less frequent dosing.[152] LP-003 is being developed by LongBio Pharma as a treatment for seasonal allergic rhinitis, chronic spontaneous urticaria, allergic asthma, and CRSwNP. The company plans to submit BLA to the NMPA for LP-003 for allergic rhinitis in or before the third quarter of 2026.

In a randomized Phase 2 trial (NCT06046391) of patients with seasonal allergic rhinitis, LP003 in combination with fluticasone propionate (SoC) significantly reduced total nasal symptom scores compared with placebo plus SoC (3.31 vs. 4.06; $p = 0.0464$), with additional improvements in daily nasal (−0.88; $p = 0.0352$) and ocular (−0.54; $p = 0.0245$) symptom scores. Adverse event rates were comparable between groups, supporting a favorable safety profile.[153]

Building on these findings, a placebo-controlled Phase 3 clinical trial (CTR20241964) was initiated in August 2024 to evaluate the efficacy and safety of LP003 in patients with moderate to severe seasonal allergic rhinitis inadequately controlled by standard therapy. Patients receive SC LP003, 100 mg per dose, Q4W for a total of 2 doses or placebo on the same schedule. Patient enrollment is expected to be completed by the end of 2025.

Elegrobart (Viridian Therapeutics, Kissei Pharmaceutical)

Elegrobart (VRDN-003, ZL-1109) is a humanized IgG1κ monoclonal antibody that binds IGF1R. Developed by Viridian Therapeutics, it is derived from the company's lead asset, veligrotug (VRDN001), which shares the same target. Both programs are being advanced for the treatment of TED. Veligrotug is IV administered, whereas YTE mutations (M252Y, S254T, T256E) were incorporated into elegrobart, providing it with an extended halflife (40–50 days) that is substantially longer than veligrotug and enables elegrobart to be SC administration.[150] Viridian entered into a supply agreement with Ypsomed to utilize its proprietary YpsoMate® 2.25 autoinjector pen, supporting convenient self-administration. Viridian has a collaboration and licensing agreement with Kissei Pharmaceutical to develop and commercialize veligrotug and elegrobart in Japan.

Viridian Therapeutics anticipates a potential commercial launch for veligrotug for TED in 2026. To further strengthen its presence in the TED market, the company plans to submit a BLA for elegrobart by yearend 2026. The program is supported by robust intellectual property protection, including a composition of matter patent for elegrobart granted by the USPTO, which extends through 2041 with the potential for additional exclusivity.[154]

Two placebo-controlled Phase 3 trials (REVEAL-1, NCT06625411; REVEAL-2, NCT06625398) are evaluating elegrobart as a treatment for TED. The REVEAL-1 study is evaluating the effects of 6 SC administrations of elegrobart Q4W or 3 SC administrations of elegrobart Q8W compared to placebo in 132 patients with active TED, while the REVEAL-2 study is evaluating the same regimen in 204 patients with chronic TED. The primary endpoint of both studies is the proptosis responder rate in the study eye at Week 24. Both studies have completed enrollment and remain on track to deliver topline results in the first half of 2026.[154]

Camtarkibart (Sunshine Guojian Pharmaceutical)

Camtarkibart (SSGJ611) is a hinge stabilized (S228P), humanized IgG4κ monoclonal antibody targeting the interleukin4 receptor alpha (IL4Rα).[155] By binding IL4Rα, camtarkibart blocks both IL4 and IL13 signaling, thereby suppressing downstream type 2 inflammatory pathways central to atopic dermatitis, CRSwNP, and other allergic conditions. Developed by Sunshine Guojian Pharmaceutical (Shanghai), a subsidiary of 3SBio, for the treatment of type 2 inflammatory diseases, the company plans to submit an NDA for camtarkibart for atopic dermatitis during 2026.[156]

The safety and efficacy of camtarkibart were evaluated in a randomized, doubleblind, placebo controlled Phase 2 trial (NCT05544591) of Chinese adults with moderate to severe atopic dermatitis. Eligible patients were randomly assigned in a 1:1:1 ratio to receive SC camtarkibart at a dose of either 300 mg (loading dose of 600 mg) Q2W (Group A) or 300 mg (loading dose of 600 mg) Q4W (Group B), or placebo Q2W for 16 weeks, followed by an 8-week follow-up period. The primary efficacy endpoint was the proportion of patients achieving at least a 75% reduction in the EASI-75 score at Week 16. The study results showed that camtarkibart significantly improved EASI scores, with 60.0% of patients in Group A and 48.8% in Group B achieving EASI-75 vs 15.6% of those in the placebo group after 16 weeks of treatment. Adverse event rates were comparable between groups, supporting a favorable safety profile.[157]

A placebo-controlled Phase 3 trial of camtarkibart in atopic dermatitis in Chinese adults (NCT06173284) is enrolling an estimated 510 patients. The study's 52-week treatment period includes an induction phase in which patients receive camtarkibart SC 600 mg (loading dose, week 0) + 300 mg Q2W (from Week 2 to Week 14, 7 cycles) or placebo and a maintenance phase in which patients receive camtarkibart SC 300 mg Q2W or Q4W or placebo. The primary outcome measures are the number of participants with EASI-75 Response at Week 16 and the number of participants with IGA Score of "0" or "1" and improvement from baseline of ≥ 2 points from baseline to Week 16. The study has a primary completion date in September 2025.

In addition, a placebo-controlled Phase 3 study (NCT06554847) is evaluating the safety and efficacy of SC camtarkibart when used in combination with topical corticosteroid (TCS) treatment compared with placebo in combination with TCS treatment for moderate-to-severe atopic dermatitis. This study has an estimated enrollment of 400 patients and a primary completion date in October 2025.

A Phase 3 trial of camtarkibart in atopic dermatitis in adolescents (CTR20252150) has been initiated, as well as Phase 3 studies in COPD (CTR20252166, NCT07039669) and CRSwNP (CTR20243786).

Manfidokimab (Akeso Inc.)

Manfidokimab (AK120) is a hinge stabilized (S228P) humanized IgG4κ monoclonal antibody developed by Akeso that targets interleukin-4 receptor alpha (IL-4 Rα) subunit, which is shared by the Interleukin-4 (IL-4) and interleukin-13 (IL-13) receptor complexes. IL-4 and IL-13 cytokine signaling cascades are critical for T helper 2 (Th2)-mediated inflammatory responses in diseases such as atopic dermatitis. By binding IL-4 Rα, manfidokimab blocks both IL-4 and IL-13 activity, reducing type 2 inflammation in atopic dermatitis and other Th2-mediated diseases.[158] Akeso plans to submit an NDA in China for manfidokimab in AD following successful Phase 3 study outcomes in this indication.[88]

Manfidokimab, which is administered SC, is designed as an alternative to existing IL-4 Rα inhibitors such as dupilumab (Dupixent®), with a potential for improved efficacy and patient convenience. In a Phase 1 trial (NCT04256174), manfidokimab was demonstrated to be safe and well-tolerability in healthy volunteers and atopic dermatitis patients, with dose-dependent reductions in biomarkers like IgE and TARC/CCL17.[158] A placebo-controlled Phase 2 study in which atopic dermatitis patients were administered manfidokimab in doses ranging from 150 mg to 450 mg administered via SC injection confirmed clinical efficacy, showing significant improvements in EASI-75 scores compared to placebo.[159]

In a placebo-controlled Phase 3 trial (CTR20241231/NCT06383468) in adult patients with moderate-to-severe AD, manfidokimab met all the primary and secondary endpoints, achieving statistically significant improvements in skin clearance and itch reduction.[88] The dosing regimen included an initial 600 mg loading dose on Day 1, followed by 300 mg SC injections Q2W through Week 50. An ongoing Phase 3 clinical study (CTR20244937/NCT06767540) of manfidokimab in the treatment of moderate to severe atopic dermatitis in adolescents was started in April 2025.

Antibodies to watch: cancer

Ozekibart (Inhibrx Biosciences, Inc.)

Ozekibart (INBRX-109) is a tetravalent, humanized, death receptor 5 (DR5) agonist composed of four single-domain antibodies fused to an IgG1-derived Fc. It is designed to cluster four receptors, causing tumor-biased cell death induced by the activation of DR5. Ozekibart's Fc has been also engineered with a proprietary three amino acid deletion near the hinge (E233del, L234del, L235del) to reduce effector function.[160] FDA granted Fast Track designation to ozekibart for the treatment of patients with metastatic or unresectable conventional chondrosarcoma and Orphan Drug designation to ozekibart for chondrosarcoma. Additionally, ozekibart was granted Orphan Medicinal Product designation in the EU for the treatment of chondrosarcoma. Inhibrx plans to submit a BLA for ozekibart for chondrosarcoma in the second quarter of 2026.[161]

In October 2025, Inhibrx announced positive topline results from the registrational, placebo-controlled Phase 2 ChonDRAgon study (NCT04950075) ($n = 206$), which evaluated ozekibart as a single agent

administered IV Q3W versus placebo in patients with advanced or metastatic, unresectable chondrosarcoma. The ChonDRAgon study met its primary endpoint of a statistically significant and clinically meaningful median PFS for patients treated with ozekibart compared to placebo. The median PFS was 5.52 months for patients administered ozekibart versus 2.66 months for placebo, representing a 52% reduction in the risk of disease progression or death compared to placebo (stratified Hazard Ratio [HR] 0.479; 95% CI: 0.33, 0.68); $p < 0.0001$).[161]

In combination with other agents, ozekibart is also being evaluated as a treatment for advanced or metastatic, unresectable colorectal cancer and advanced or metastatic, unresectable, relapsed, or refractory Ewing sarcoma in expansion cohorts from the Phase 1 study (NCT03715933).[160,161]

Sonesitatug vedotin (AstraZeneca)

Sonesitatug vedotin (AZD0901, CMG901, MRG005) is an ADC composed of a humanized Claudin 18.2-targeting IgG1κ monoclonal antibody (CM311), a cleavable linker and a cytotoxic tubulin inhibitor payload (MMAE). In 2023, KYM Biosciences, a joint venture established by affiliates of Keymed Biosciences and Lepu Biopharma granted an exclusive licensing agreement for the research, development, manufacture, and commercialization of sonesitatug vedotin globally to AstraZeneca.[162] AstraZeneca expects data readout from the Phase 3 CLARITY-Gastric01 (NCT06346392) trial during the first half of 2026.[145] If positive, study results could potentially support a marketing submission to a regulatory agency by the end of 2026.

Sonesitatug vedotin was granted Orphan Drug designation from the FDA for the treatment of gastric cancer and gastroesophageal junction adenocarcinoma, as well as Fast Track designation for the treatment of unresectable or metastatic gastric cancer and gastroesophageal junction cancer which has relapsed and/or is refractory to approved therapies. Additionally, sonesitatug vedotin received Breakthrough Therapy designation from the NMPA for relapsed/refractory advanced gastric cancer.

The ongoing open-label, Phase 3 CLARITY-Gastric01 (NCT06346392) trial is evaluating sonesitatug vedotin monotherapy for the second- or later-line treatment of Claudin 18.2+ gastric cancer patients. Participants are randomized 1:1:1 to receive sonesitatug vedotin IV Q3W at dose level 1 (Arm 1) or at dose level 2 (Arm 2), or investigator's choice of therapy (Arm 3). Investigator's choice includes ramucirumab plus paclitaxel, paclitaxel, or docetaxel in the secondline setting, and irinotecan, TAS102 (outside China), or apatinib (China only) in the thirdline or later setting. The primary endpoints of the study are PFS and OS; the primary completion date is in October 2026.

Trastuzumab pamirtecan (Duality Biologics (Suzhou) Co., Ltd., BioNTech SE)

Trastuzumab pamirtecan (BNT323/DB1303) is an ADC composed of a humanized IgG1κ monoclonal antibody targeting HER2, conjugated to a topoisomerase I inhibitor payload (P1003) via a cleavable tetrapeptide linker (GlyGlyPheGly), with a DAR of approximately 8. The molecule was originally developed by Duality Biologics. Through a strategic collaboration established in 2023, BioNTech holds global commercial rights, excluding Mainland China, Hong Kong Special Administrative Region, and Macau Special Administrative Region, which are held by Duality Biologics. Trastuzumab pamirtecan received Fast Track and Breakthrough Therapy designations from the FDA for advanced recurrent or metastatic endometrial cancer.

The partners also plan a BLA submissions in the US in 2026, which could be based on results from a potentially registrational cohort of HER2-expressing patients with recurrent endometrial cancer included in a Phase 1/2 trial (NCT05150691).[163] Based on positive results of a pivotal study of trastuzumab pamirtecan in breast cancer patients, Duality Bio plans to discuss next steps with the NMPA for submission of a BLA for trastuzumab pamirtecan.[164]

A FIH, dose-escalation and dose-expansion Phase 1/2 trial (NCT05150691) is evaluating trastuzumab pamirtecan in patients with HER2+ advanced solid tumors. The study includes a potentially registrational cohort of patients with HER2-expressing (IHC3+, 2+, 1+ or ISH-positive) advanced endometrial carcinoma. In the dose-escalation phase of the study, patients are IV-administered a single dose of trastuzumab pamirtecan on Day 1 of each cycle Q3W; five dose levels are being evaluated. The dose-expansion phase

aims to confirm safety and tolerability and explore efficacy in selected malignant solid tumors at the maximum tolerated or recommended Phase 2 dose. The ORR as assessed by RECIST 1.1. up to the follow-up period, approximately 1 year post-treatment, is a primary endpoint of the Phase 1/2 study, which has a primary completion date in April 2026.

The confirmatory Phase 3 BNT32301 study (NCT06340568) in endometrial cancer patients was initiated in June 2025. The BNT32301 study is evaluating trastuzumab pamirtecan versus doxorubicin/paclitaxel in advanced endometrial cancer after prior immune checkpoint inhibitor therapy. An estimated 504 patients will be randomized 1:1 to the two study arms (8 mg/kg trastuzumab pamirtecan vs. 60 mg/m^2 doxorubicin +80 mg/m^2 paclitaxel), with PFS at 32 months as the primary endpoint.

In September 2025, positive results were reported for the Phase 3 DYNASTYBreast01 trial (NCT06265428), which compared trastuzumab pamirtecan with trastuzumab emtansine (Kadcyla®) in patients with HER2positive unresectable or metastatic breast cancer following prior trastuzumab and taxane therapy. In the study, 228 patients were randomized 1:1 to receive 8 mg/kg trastuzumab pamirtecan IV or 3.6 mg/kg Kadcyla Q3W. The trial met its primary endpoint of PFS as per RECIST 1.1 criteria, evaluated by BICR, in a prespecified interim analysis.[164]

The Phase 3 DYNASTYBreast02 study (NCT06018337) is evaluating the effects of trastuzumab pamirtecan in HR positive, HER2 low metastatic breast cancer progressing on endocrine therapy. Patients are randomized to receive 8 mg/kg trastuzumab pamirtecan IV on Day 1 of each cycle Q3W or investigator's choice single agent chemotherapy (capecitabine, paclitaxel, or nab-paclitaxel (Abraxane)). The primary endpoint is PFS in the HR+, HER2-low population up to approximately 51 months. The study's primary completion date is in May 2026.

Ifinatamab deruxtecan (Daiichi Sankyo Co. Ltd, Merck)

Ifinatamab deruxtecan (DS-7300a) is an ADC composed of a humanized anti-B7-H3 IgG1κ monoclonal antibody, an enzymatically cleavable peptide-based linker, and a DNA topoisomerase I inhibitor (exatecan derivative DXd; DAR ~4). In 2023, Daiichi Sankyo and Merck entered into a global development and commercialization agreement for ifinatamab deruxtecan, which has been granted Breakthrough Therapy designation by the FDA for the treatment of adult patients with extensive-stage small cell lung cancer (ES-SCLC) with disease progression on or after platinum-based chemotherapy. In September 2025, Merck announced positive results from the randomized Phase 2 IDeate-Lung01 (NCT05280470) study and indicated that discussions with global regulatory authorities underway,[165] suggesting that submission of a marketing application for SCLC in 2026 is possible.

Ifinatamab deruxtecan is being evaluated versus Physician's Choice treatments (topotecan, amrubicin, lurbinectedin) in a Phase 3 study of (NCT06203210, IDeate-Lung02) of patients with relapsed SCLC. Participants are randomized to receive 12 mg/kg ifinatamab deruxtecan monotherapy on Day 1 of each 21-day cycle until unacceptable toxicity, progressive disease, or withdrawal of consent as specified in the protocol. The study's primary outcome measures are ORR and OS up to approximately 5 years. Initiated in May 2024, the IDeate-Lung02 study has a primary completion date in April 2027. However, a global hold was placed on patient recruitment for the study in mid-December 2025 due to a higher than anticipated incidence of grade 5 interstitial lung disease events.[166]

Ifinatamab deruxtecan is also being evaluated in Phase 3 studies of patients with: 1) metastatic castration-resistant prostate cancer with disease progression during or after treatment with an androgen receptor pathway inhibitor (IDeate-Prostate01, NCT06925737); and 2) pretreated advanced or metastatic esophageal squamous cell carcinoma (NCT06644781). Both studies have primary completion dates in June 2028.

Petosemtamab (Merus N.V.)

Petosemtamab (MCLA-158) is a human, ADCC-enhanced (low fucose) common light chain IgG1κ Biclonics® bispecific antibody that targets cancer stem cells expressing leucine-rich repeat-containing G protein-coupled receptor 5 (LGR5) and EGFR. FDA has granted Breakthrough Therapy designation to

petosemtamab in combination with pembrolizumab for the first-line treatment of adult patients with recurrent or metastatic programmed death-ligand 1 (PD-L1) positive head and neck squamous cell carcinoma (r/m HNSCC) with combined positive score (CPS) ≥ 1 and for the treatment of patients with r/m HNSCC) whose disease has progressed following treatment with platinum-based chemotherapy and an anti-programmed cell death receptor-1 (PD-1) or PD-L1 antibody.

Petosemtamab was discovered and developed by Merus. In September 2025, Genmab A/S announced their intention to acquire all the shares of the company. A tender offer for 100% of Merus' common shares, commenced by a wholly owned subsidiary of Genmab, is anticipated to close in the first quarter of 2026. According to Genmab, petosemtamab has potential for an initial launch in 2027,[167] which suggests a marketing application could be submitted by the end of 2026.

Positive data from a single-arm Phase 2 trial (NCT03526835) of petosemtamab as a HNSCC treatment were presented at the American Society for Clinical Oncology 2025 Annual Meeting.[167,168] In the NCT03526835 study, patients were administered petosemtamab 1500 mg Q2W (28-day cycles) with pembrolizumab (400 mg Q6W) as 1 L treatment in PD-L1+ HNSCC.

Merus is sponsoring two ongoing Phase 3 trials, LiGeR-HN1 and LiGeR-HN2, in first- and second/third-line HNSCC. Topline interim readout of one or both trials is anticipated in 2026. The LiGeR-HN1 study (NCT06525220) is evaluating the efficacy and safety of petosemtamab (1500 mg IV Q2W) plus pembrolizumab compared to pembrolizumab in 1 L PD-L1+ r/m HNSCC, while the LiGeR-HN2 trial (NCT06496178) is evaluating the efficacy and safety of petosemtamab (1500 mg IV Q2W) compared to investigator's choice of single agent chemotherapy or cetuximab in previously treated (2/3 L) patients with r/m HNSCC.[169] The primary endpoints of these studies are ORR per RECIST 1.1 by BICR, and OS.

Outlook for the future

In this installment of the "Antibodies to Watch" article series, we discussed a total of 66 antibody therapeutics that were: 1) approved in 2025, 2) in regulatory review, or 3) were noted as antibodies to watch due to their likelihood of entering regulatory review by the end of 2026. Interestingly, the majority of antibody therapeutics in all three categories are for non-cancer indications (63% of approved products, and 81% and 76% of those in regulatory review and those likely to enter regulatory review soon, respectively). Given that the majority (54%) of the late-stage pipeline is composed of antibodies intended as treatments for cancer, we believe that the percentage of antibodies for cancer in these categories will increase in the future, even taking into consideration the historically higher attrition rate for anti-cancer antibody therapeutics compared to those for other therapeutic areas. We are cautiously optimistic that the highly innovative antibody therapeutics currently in development, particularly ADCs with next-generation payloads and bi- or multispecific molecules, many of which are for cancer, may prove more successful than earlier versions. More than 70% of the late-stage clinical pipeline of antibodies for cancer have been modified to enhance functionality, whereas this is the case for only 20% of the antibodies for non-cancer indications. The inclusion of bispecific antibodies and topoisomerase I inhibitors in ADCs are examples of enhancements that may improve the efficacy of the anti-cancer drugs now in late-stage studies.

Our data also show a clear trend toward first marketing application submissions and approvals in China, which was the case for ~60% of the molecules we included in these two categories. We anticipate this trend to continue, considering that ~40% of the over 1500 antibodies currently in clinical studies originated in China. Moreover, of the highly innovative molecules, comprising ~600 ADCs, bispecific ADCs, and naked bi- and multispecific antibodies currently in clinical study, the majority (55%) originated in China. As we noted above, China has created a policy-driven ecosystem that encourages innovation[2,3] and provides substantial opportunities to companies developing enhanced antibody therapeutics,[4] including new classes of molecules, e.g., dual-payload ADCs,[170] that are just beginning to enter FIH studies.

Regardless of where it is done, the future of antibody therapeutics development continues to look bright. This is due to the incredible versatility of the molecules, as well as the creativity and dedicated efforts of antibody engineers and scientists, including those working to apply artificial intelligence (AI), machine learning (ML), and deep learning (DL) techniques to antibody discovery.[171] While not discussed here, AI/ML/DL are transformative tools that may have profound effects on antibody therapeutics development well

into the future by enabling the creation of safer and more effective products. Although the effective use of AI/ML/DL for de novo antibody discovery is still evolving, these approaches already show strong potential to streamline and accelerate multiple stages of the development process. AI-driven methods can support high-throughput candidate screening, improve developability predictions, integrate large and disparate datasets, and reveal patterns that guide more informed decision-making. As these capabilities mature, they may help reduce attrition by enabling teams to prioritize candidates with a greater likelihood of clinical success, ultimately contributing to a more efficient and productive antibody development pipeline.

Acknowledgments

JMR thanks Hanson Wade for providing access to the Beacon database.

Funding

The authors reported there is no funding associated with the work featured in this article.

ORCID

Silvia Crescioli http://orcid.org/0000-0002-1909-5957
Hélène Kaplon http://orcid.org/0000-0002-5597-2195
Alicia Chenoweth http://orcid.org/0000-0002-2736-9268
Yu-Shin Hsu http://orcid.org/0009-0005-9288-971X
Kieran Pinto http://orcid.org/0009-0000-4740-7666
Vaishali Kapoor http://orcid.org/0000-0002-7581-1487
Janice M. Reichert http://orcid.org/0000-0003-0400-1951

Abbreviations

Aβ,	amyloid beta;
ADC,	antibody-drug conjugate;
ADCC,	antibody-dependent cellular cytotoxicity;
AML	acute myeloid leukemia;
Ang-2	angiopoietin-2;
axSpA	axial spondyloarthritis;
BCMA,	B cell maturation antigen;
BICR,	blinded independent central review;
BLA,	biologics license application;
BMD,	bone mineral density;
BTC,	biliary tract cancer;
CDC,	complement-dependent cytotoxicity;
CI,	confidence interval;
CLL,	chronic lymphocytic leukemia;
CMC,	Chemistry, Manufacturing, and Controls;
COVID-19	coronavirus disease 2019;
CPS,	combined positive score;
CR,	complete response;
CRSwNP,	chronic rhinosinusitis with nasal polyps;
cSCC,	cutaneous squamous cell carcinoma;
CTLA-4,	cytotoxic T lymphocyte antigen-4;
DLBCL,	diffuse large B-cell lymphoma;
DMD,	Duchenne muscular dystrophy;
EASI,	Eczema Area and Severity Index;
EC,	European Commission;
ECOG,	Eastern Cooperative Oncology Group;
EGFR,	epidermal growth factor receptor;
EMA,	European Medicines Agency;
EpCAM,	epithelial cell adhesion molecule;
EU,	European Union;

Fab, antigen-binding fragment;
Fc, crystallizable fragment;
FcyR receptor for IgG Fc;
FcRn neonatal Fc receptor;
FIH, first-in-human;
FDA, US Food and Drug Administration;
FL, follicular lymphoma;
G/GEJ, gastric or gastroesophageal junction;
gMG, generalized myasthenia gravis;
GPP, generalized pustular psoriasis;
GvHD, graft-vs-host disease;
HCC hepatocellular carcinoma;
HER2, human epidermal growth factor receptor 2;
HLA, human leukocyte antigen;
HoFH, homozygous familial hypercholesterolemia;
HR, hazard ratio;
iADRS, Integrated AD Rating Scale;
IFN, interferon;
IGA Investigator's Global Assessment;
IgE, immunoglobulin E;
IgG, immunoglobulin G;
IL, interleukin; IM, intramuscular;
ITT, intention-to-treat;
ICR, independent central review;
IRC, independent review committee; IV, intravenous;
LAG-3, lymphocyte-activation gene 3;
LDL, low-density lipoprotein;
LM, leptomeningeal metastases;
mAb, monoclonal antibody;
MET, mesenchymal epithelial transition factor;
MHLW, Ministry of Health, Labour and Welfare;
MM, multiple myeloma;
MMAE, monomethyl auristatin E;
NDA, new drug application;
NHL, non-Hodgkin's lymphoma;
NK, natural killer;
NMPA, National Medical Products Administration;
NPC, nasopharyngeal carcinoma;
NSCLC, non-small cell lung cancer;
ORR, objective response rate;
OS, overall survival;
PCSK9, proprotein convertase subtilisin/kexin type 9;
PD, pharmacodynamics;
PD-1, programmed cell death protein 1;
PD-L1 programmed cell death protein ligand 1;
PD-L2 programmed death ligand 2;
PFS, progression-free survival;
PNH, paroxysmal nocturnal hemoglobinuria;
PK, pharmacokinetics;
PR, partial response;
PRIME, Priority Medicines;
Q2W, every 2 weeks;
RECIST, Response Evaluation Criteria in Solid Tumors;
R/R, relapsed or refractory;
RSV, respiratory syncytial virus;
SARS-CoV-2, severe acute respiratory syndrome coronavirus 2;
SC, subcutaneous;
scFv, single-chain variable fragment;
SoC, standard of care;
TCR, T cell receptor;
TIGIT, T-cell Immunoreceptor with Ig and ITIM domains;
TIM-3, T-cell immunoglobulin and mucin-domain domain-containing molecule-3;

TKI, tyrosine kinase inhibitor;
TMAs, thrombotic microangiopathies;
TNF, tumor necrosis factor;
UK, United Kingdom;
uPCR, urinary protein-to-creatinine ratio;
US, United States;
VEGF, human vascular endothelial growth factor;
VHH, variable heavy chain single domain antibodies.

References

1. Taylor & Francis. Antibodies to watch collection. mAbs. 2025 [accessed 2025 Nov 16]. https://www.tandfonline.com/journals/kmab20/collections/antibodies-to-watch.
2. Tan R, Hua H, Zhou S, Yang Z, Yang C, Huang G, Zeng J, Zhao J. Current landscape of innovative drug development and regulatory support in China. Sig Transduct Target Ther. 2025;10(1):220. doi: 10.1038/s41392-025-02267-y.
3. Liu S, Hu H, Ge C, Yuan S, Jiang J, Chen X. The rise of China's pharmaceutical industry from 2015–2024: a decade of innovation. Nat Rev Drug Discov. 2025;24(10):738–739. doi: 10.1038/d41573-025-00102-1.
4. Crescioli S, Reichert JM. Innovative antibody therapeutic development in China compared with the USA and Europe. Nat Rev Drug Discov. 2025; doi: 10.1038/d41573-025-00185-w.
5. Drulyte I, Ghai R, Ow SY, Kapp EA, Quek AJ, Panousis C, Wilson MJ, Nash AD, Pelzing M. Structural basis for the inhibition of betaFxiia by garadacimab. Structure. 2024;32(10):1705–1710.e3. doi: 10.1016/j.str.2024.07.001 .
6. CSL. Health Canada has authorized CSL's ANDEMBRY® (garadacimab) as once-monthly treatment for hereditary angioedema (HAE). 2025 [accessed 2025 Nov 10]. https://www.cslbehring.ca/news/2025/health-canada-has-authorized-csl-andembry.
7. Craig TJ, Reshef A, Li HH, Jacobs JS, Bernstein JA, Farkas H, Yang WH, Stroes ESG, Ohsawa I, Tachdjian R, et al. Efficacy and safety of garadacimab, a factor XIIa inhibitor for hereditary angioedema prevention (VANGUARD): a global, multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. Lancet. 2023;401(10382):1079–1090. doi: 10.1016/S0140-6736(23)00350-1.
8. Reshef A, Hsu C, Katelaris CH, Li PH, Magerl M, Yamagami K, Guilarte M, Keith PK, Bernstein JA, Lawo JP, et al. Long-term safety and efficacy of garadacimab for preventing hereditary angioedema attacks: phase 3 open-label extension study. Allergy. 2025;80(2):545–556. doi: 10.1111/all.16351.
9. InflaRx N.V. Inflarx receives European Commission approval for GOHIBIC® (vilobelimab) for the treatment of SARS-CoV-2-induced acute respiratory distress syndrome (ARDS). 2025 [accessed 2025 Nov 10]. https://www.inflarx.de/Home/Investors/Press-Releases/Press-Release~2025-01-InflaRx-Receives-European-Commission-Approval-for-GOHIBIC—vilobelimab–for-the-Treatment-of-SARS-CoV-2-Induced-Acute-Respiratory-Distress-Syndrome–ARDS-~.html.
10. European Medicines Agency. Gohibic. [accessed 2025 Nov 10]. https://www.ema.europa.eu/en/medicines/human/EPAR/gohibic.
11. European Medicines Agency. Lynozyfic. [accessed 2025 Dec 1]. https://www.ema.europa.eu/en/medicines/human/EPAR/lynozyfic.
12. Regeneron Pharmaceuticals, Inc. Lynozyfic™ (linvoseltamab-gcpt) receives FDA accelerated approval for treatment of relapsed or refractory multiple myeloma. 2025 [accessed 2025 Nov 11]. https://investor.regeneron.com/news-releases/news-release-details/lynozyfictm-linvoseltamab-gcpt-receives-fda-accelerated-approval.
13. Food and Drug Administration. Linvoseltamab NDA/BLA multi-disciplinary review and evaluation. 2024 [accessed 2025 Dec 3]. https://www.accessdata.fda.gov/drugsatfda_docs/nda/2025/761400Orig1s000MultidisciplineR.pdf.
14. Seth NP, Xu R, DuPrie M, Choudhury A, Sihapong S, Tyler S, Meador J, Avery W, Cochran E, Daly T, et al. Nipocalimab, an immunoselective FcRn blocker that lowers IgG and has unique molecular properties. MAbs. 2025;17(1):2461191. doi: 10.1080/19420862.2025.2461191.
15. Johnson & Johnson. Johnson & Johnson receives FDA approval for IMAAVYTM (nipocalimab-aahu), a new FcRn blocker offering long-lasting disease control in the broadest population of people living with generalized myasthenia gravis (gMG). 2025 [accessed 2025 Nov 11]. https://www.jnj.com/media-center/press-releases/johnson-johnson-receives-fda-approval-for-imaavytm-nipocalimab-aahu-a-new-fcrn-blocker-offering-long-lasting-disease-control-in-the-broadest-population-of-people-living-with-generalized-myasthenia-gravis-gmg.
16. Johnson & Johnson. Johnson & Johnson receives European Commission approval of IMAAVY®▼ (nipocalimab), a new FcRn blocker offering sustained disease control in a broad population of people living with generalised myasthenia gravis (gMG). 2025 [accessed 2025 Dec 3]. https://www.jnj.com/media-center/press-releases/johnson-johnson-receives-european-commission-approval-of-imaavy-nipocalimab-a-new-fcrn-blocker-offering-sustained-disease-control-in-a-broad-population-of-people-living-with-generalised-myasthenia-gravis-gmg.

17. US Food and Drug Administration. Prescribing information: ImaavyTM (nipocalimab-aahu) injection, for intravenous use. 2025 [accessed 2025 Nov 12]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/761430s000lbl.pdf.
18. Johnson & Johnson. Johnson & Johnson announces first head-to-head study comparing IMAAVY™ with an alternative FcRn blocker in generalized myasthenia gravis (gMG) at AANEM annual meeting. 2025 [accessed 2025 Nov 11]. https://www.jnj.com/media-center/press-releases/johnson-johnson-announces-first-head-to-head-study-comparing-imaavy-with-an-alternative-fcrn-blocker-in-generalized-myasthenia-gravis-gmg-at-aanem-annual-meeting.
19. US Food and Drug Administration. Fda grants accelerated approval to telisotuzumab vedotin-tllv for NSCLC with high c-met protein overexpression. 2025 [accessed 2025 Nov 12]. https://www.fda.gov/drugs/resources-information-approved-drugs/fda-grants-accelerated-approval-telisotuzumab-vedotin-tllv-nsclc-high-c-met-protein-overexpression.
20. Merck US. Fda approves Merck's Enflonsia™ (clesrovimab-cfor) for prevention of respiratory syncytial virus (RSV) lower respiratory tract disease in infants born during or entering their first RSV season. 2025 [accessed 2025 Nov 12]. https://www.merck.com/news/u-s-fda-approves-mercks-enflonsia-clesrovimab-cfor-for-prevention-of-respiratory-syncytial-virus-rsv-lower-respiratory-tract-disease-in-infants-born-during-or-entering-their-fir/.
21. European Medicines Agency. Enflonsia® (clesrovimab). 2025 [accessed 2025 Dec 3]. https://www.ema.europa.eu/en/medicines/human/EPAR/enflonsia.
22. US Food and Drug Administration. Prescribing information: Enflonsia™ (clesrovimab-cfor) injection, for intramuscular use. 2025 [accessed 2025 Nov 12]. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/761432s000lbl.pdf.
23. Otsuka Pharmaceutical, Co. Ltd.. Otsuka receives FDA accelerated approval for VOYXACT® (sibeprenlimab-szsi) for the reduction of proteinuria in adults with primary immunoglobulin A nephropathy (IgAN) at risk for disease progression. 2025 [accessed 2025 Nov 26]. https://www.otsuka-us.com/news/otsuka-receives-fda-accelerated-approval-voyxactr-sibeprenlimab-szsi-reduction-proteinuria.
24. Mathur M, Barratt J, Chacko B, Chan TM, Kooienga L, Oh KH, Sahay M, Suzuki Y, Wong MG, Yarbrough J, et al. A phase 2 trial of sibeprenlimab in patients with IgA nephropathy. N Engl J Med. 2024;390(1):20–31. doi: 10.1056/NEJMoa2305635.
25. Otsuka Pharmaceutical, Co. Ltd.. Otsuka sibeprenlimab phase 3 data show a statistically significant and clinically meaningful proteinuria reduction for the treatment of immunoglobulin A nephropathy (IgAN). 2025 [accessed 2025 Oct 8]. https://www.otsuka-us.com/news/otsuka-sibeprenlimab-phase-3-data-show-statistically-significant-and-clinically-meaningful.
26. GSK plc. Exdensur (depemokimab) approved in the UK for treatment of asthma with type 2 inflammation and chronic rhinosinusitis with nasal polyps. 2025 [accessed 2025 Dec 16]. https://www.gsk.com/en-gb/media/press-releases/exdensur-depemokimab-approved-in-the-uk-for-treatment-of-asthma-with-type-2-inflammation-and-chronic-rhinosinusitis-with-nasal-polyps/.
27. Jackson DJ, Wechsler ME, Jackson DJ, Bernstein D, Korn S, Pfeffer PE, Chen R, Saito J, de Luíz Martinez G, Dymek L, et al. Twice-yearly depemokimab in severe asthma with an eosinophilic phenotype. N Engl J Med. 2024;391(24):2337–2349. doi: 10.1056/NEJMoa2406673.
28. GSK plc. GSK announces positive phase III results from ANCHOR trials for depemokimab in chronic rhinosinusitis with nasal polyps. 2024 [accessed 2025 Oct 9]. https://www.gsk.com/en-gb/media/press-releases/gsk-announces-positive-phase-iii-results-from-anchor-trials/.
29. Omeros Corporation. FDA approves omeros' YARTEMLEA® – first and only therapy indicated for TA-TMA. 2025. [accessed 2025 Dec 26]. https://investor.omeros.com/news-releases/news-release-details/omeros-corporation-reports-third-quarter-2025-financial-results.
30. Omeros Corporation. Omeros submits narsoplimab marketing authorization application to the European Medicines Agency for the treatment of TA-TMA. 2025 [accessed 2025 Oct 7]. https://investor.omeros.com/news-releases/news-release-details/omeros-submits-narsoplimab-marketing-authorization-application.
31. Omeros Corporation. Omeros announces update on statistical analysis of narsoplimab pivotal trial primary endpoint. 2025 [accessed 2025 Oct 7]. https://investor.omeros.com/news-releases/news-release-details/omeros-announces-update-statistical-analysis-narsoplimab-pivotal.
32. Schoettler ML, Pusarla SK, Nangia N, Perales MA, Duarte RF, Grauer A, Rambaldi A. Narsoplimab results in excellent survival in adults and children with hematopoietic cell transplant associated thrombotic microangiopathy (TA-TMA). Am J Hematol. 2025;100(11):2040–2051. doi: 10.1002/ajh.70044.
33. Omeros Corporation. Omeros announces robust results for narsoplimab expanded access program in TA-TMA. 2025 [accessed 2025 Oct 7]. https://investor.omeros.com/news-releases/news-release-details/omeros-announces-robust-results-narsoplimab-expanded-access.
34. Lamb YN. Recaticimab: first approval. Drugs. 2025;85(8):1067–1072. doi: 10.1007/s40265-025-02199-5.
35. Xu M, Wang Z, Zhang Y, Liu Y, Huang R, Han X, Yao Z, Sun J, Tian F, Hu X, et al. Recaticimab monotherapy for nonfamilial hypercholesterolemia and mixed hyperlipemia: the phase 3 REMAIN-1 randomized trial. J Am Coll Cardiol. 2024;84(20):2026–2036. doi: 10.1016/j.jacc.2024.07.035.

36. Sun Y, Lv Q, Guo Y, Wang Z, Huang R, Gao X, Han Y, Yao Z, Zheng M, Luo S, et al. Recaticimab as add-on therapy to statins for nonfamilial hypercholesterolemia: the randomized, phase 3 REMAIN-2 trial. J Am Coll Cardiol. 2024;84(20):2037–2047. doi: 10.1016/j.jacc.2024.09.012.
37. Li L, Zhou Y, Deng C, Sheng J, Peng D, Ling Z, Wang L, Dai C, Huang K, Lian Q, et al. Recaticimab in adult heterozygous familial hypercholesterolaemia (REMAIN-3): a multicentre, randomized, double-blind, placebo-controlled phase 3 study. Cardiovasc Res. 2025;121(12):1856–1864. doi: 10.1093/cvr/cvaf155.
38. Patsnap. Finotonlimab: a promising anti-PD-1 antibody for solid tumors. 2025 [accessed 2025 Nov 12]. https://synapse.patsnap.com/blog/finotonlimab-a-promising-anti-pd-1-antibody-for-solid-tumors.
39. Sinocelltech Ltd. From head and neck squamous cell carcinoma to liver cancer! Anyouping® approved for two major indications. 2025 [accessed 2025 Dec 2]. https://www.sinocelltech.com/Detail/desc/pid/23/sid/486.html.
40. Shi Y, Guo W, Wang W, Wu Y, Fang M, Huang X, Han P, Zhang Q, Dong P, Zhou X, et al. Finotonlimab with chemotherapy in recurrent or metastatic head and neck cancer: a randomized phase 3 trial. Nat Med. 2024;30(9):2568–2575. doi: 10.1038/s41591-024-03110-7.
41. Zhao C, Zhang Y, Wang G, Zheng J, Chen W, Lu Z, Zhuang L, Gu S, Han L, Zheng Z, et al. Finotonlimab (PD-1 inhibitor) plus bevacizumab (bevacizumab biosimilar) as first-tier therapy for late-stage hepatocellular carcinoma: a randomized phase 2/3 trial. Sig Transduct Target Ther. 2025;10(1):249. doi: 10.1038/s41392-025-02333-5.
42. Han B, Wu L, Yang R, Wu H, Li W, Yu Y, Zhang M, Sun H, Chu T, Zhong F, et al. Efficacy and safety of finotonlimab plus docetaxel vs. docetaxel in previously treated advanced squamous cell non-small-cell lung cancer: a randomized, double-blinded, phase III trial. Transl Lung Cancer Res. 2025;14(4):1231–1241. doi: 10.21037/tlcr-24-1042.
43. Liang Z, Liu S, Guo W, Deng Z, Bin W, Yu A, Hu J, Wu L, Li Z, Huang W, et al. Recombinant monoclonal antibody siltartoxatug versus plasma-derived human tetanus immunoglobulin for tetanus: a randomized, double-blind, active-controlled, phase 3 trial. Nat Med. 2025;31(8):2673–2681. doi: 10.1038/s41591-025-03791-8.
44. Zhuhai Trinomab Pharmaceuticals Co. Ltd. Global breakthrough: Sintetol®—next-generation tetanus prophylaxis agent—approved for marketing in China. 2025 [accessed 2025 Nov 13]. https://www.trinomab.com/en-us/info/499.html#:~:text=Sintetol%C2%AE%20has%20received%20regulatory,US%20FDA%20in%20August%202022.
45. Akeso, Inc.. Akeso's ebdarokimab (IL - 12/IL - 23 dual - targeted monoclonal antibody) approved in China for moderate - to - severe plaque psoriasis. 2025 [accessed 2025 Oct 28]. https://www.akesobio.com/en/media/akeso-news/250418/.
46. Zhang J, l Z, Wang G, Xia Y, Wang M, Li B. Long-term safety and efficacy of ebdarokimab in patients with moderate to severe plaque psoriasis: results from a single arm, open label, multicenter phase III clinical study. 2024 European Academy of Dermatology & Venereology Congress, 2024 Sep 25-28, Amsterdam; Abst. 982. [accessed 2025 Oct 28]. https://s3.eu-central-1.amazonaws.com/m-anage.com.storage.eadv/abstracts_congress_2024/42456.pdf.
47. Hengrui Pharma. China's HER2-targeted ADC trastuzumab rezetecan gains NMPA approval for HER2-mutant NSCLC. 2025 [accessed 2025 Oct 28]. https://www.prnewswire.com/news-releases/chinas-her2-targeted-adc-trastuzumab-rezetecan-gains-nmpa-approval-for-her2-mutant-nsclc-302468141.html.
48. Li Z, Wang Y, Sun Y, Wang L, Li X, Sun L, He Z, Yang H, Wang Y, Wang Q, et al. Trastuzumab rezetecan, a HER2-directed antibody-drug conjugate, in patients with advanced HER2-mutant non-small-cell lung cancer (HORIZON-Lung): phase 2 results from a multicentre, single-arm study. Lancet Oncol. 2025;26(4):437–446. doi: 10.1016/S1470-2045(25)00012-9.
49. Hengrui Pharma. Hengrui Pharma and Glenmark Pharmaceuticals enter exclusive license agreement for HER2 ADC trastuzumab rezetecan (SHR-A1811). 2025 [accessed 2025 Oct 28]. https://www.hengrui.com/en/media/detail-852.html.
50. Simcere Zaiming Pharmaceutical Co. Ltd.. Suvemcitug met primary endpoint in phase 3 clinical trial in China. 2024 [accessed 2025 Oct 25]. https://www.simcere.com/en/news/detail.aspx?mtt=417 .
51. Simcere Pharmaceutical Group Limited. Approval for marketing of Enzeshu® in China by the National Medical Products Administration. 2025 [accessed 2025 Oct 28]. https://www.hkexnews.hk/listedco/listconews/sehk/2025/0703/2025070300360.pdf.
52. Changchun GeneScience Pharmaceuticals Co. Ltd. Firsekibart approved with 87% reduction in gout recurrence over 6 months. 2025 [accessed 2025 Nov 13]. https://www.genscigroup.com/en/news/media/175.
53. Xue Y, Chu T, Hu J, Gou W, Zhang N, Li J, Yu J, Li R, Li R, Qian L, et al. Firsekibart in acute gouty arthritis. J Transl Med. 2025;23(1):91. doi: 10.1186/s12967-025-06072-7.
54. Xue Y, Chu T, Hu J, Gou W, Zhang N, Li J, Yu J, Li R, Qian L, Duan X, et al. Efficacy and safety of firsekibart in patients with acute gouty arthritis with eGFR less than 60 mL/min per 1.73 m2: post hoc analysis of 24-week data. 2025 accessed 2025 Nov 13. doi: 10.1681/ASN.20251b4pt2q8.
55. Sichuan Kelun-Biotech Biopharmaceutical Co. Ltd.. Core product trastuzumab botidotin approved for marketing by the National Medical Products Administration for 2L+ HER2+ BC. 2025 [accessed 2025 Oct 21]. https://ir-upload.realxen.net/iis/6990/uploads/iis/2025/11882363-0.PDF.

56. Sichuan Kelun-Biotech Biopharmaceutical Co. Ltd.. Kelun-biotech presents positive phase 3 data for trastuzumab botidotin compared to T-DM1 at 2025 ESMO. 2025 [accessed 2025 Oct 21]. https://www.prnewswire.com/news-releases/kelun-biotech-presents-positive-phase-3-data-for-trastuzumab-botidotin-compared-to-t-dm1-at-2025-esmo-302588701.html.
57. Lepu Biopharma Co. Ltd.. Interim report 2025. [accessed 2025 Oct 10]. https://en.lepubiopharma.com/upload/listedco/listconews/sehk/2025/0919/2025091900516.pdf.
58. Lepu Biopharma Co. Ltd.. Marketing approval obtained in China for meiyouheng (becotatug vedotin injection). 2025 [accessed 2025 Dec 3]. https://en.lepubiopharma.com/new/213.html.
59. Han F, Wang X, Xiang Y, Tang L-Q, Qu S, Shu X, Zhang P, Qiu S, Zhou Y, Guo Y, et al. Becotatug vedotin vs. chemotherapy in pre-heavily treated advanced nasopharyngeal carcinoma: a randomized, controlled, multicenter, open-label study. J Clin Oncol. 2025;43(17). doi: 10.1200/JCO.2025.43.17_suppl.LBA6005.
60. Innovent Biologics, Inc. China's first domestic IL-23p19 monoclonal antibody: Innovent's PECONDLE® (picankibart injection) received NMPA approval. 2025 [accessed 2025 Dec 5]. https://www.prnewswire.com/news-urlreleaseurls/chinas-first-domestic-il-23p19-monoclonal-antibody-innovents-pecondle-picankibart-injection-received-nmpa-approval-302627980.html.
61. Innovent Biologics, Inc. Innovent announces picankibart (IBI112) achieved primary endpoints in a phase 3 clinical trial in treating moderate to severe plaque psoriasis, and plans to submit NDA to the NMPA. 2024 [accessed 2025 Nov 7]. https://en.innoventbio.com/InvestorsAndMedia/PressReleaseDetail?key=450.
62. Innovent Biologics, Inc. Innovent announces phase 2 clinical study of picankibart (IBI112) in Chinese patients with ulcerative colitis met primary endpoint. 2024 [accessed 2025 Oct 6]. https://en.innoventbio.com/InvestorsAndMedia/PressReleaseDetail?key=488.
63. SFJ Pharmaceuticals. Fda has accepted a bla for bentracimab, the first and only ticagrelor reversal agent, for filing and priority review. 2024 [accessed 2025 Oct 9]. https://www.sfj-pharma.com/fda-has-accepted-a-bla-for-bentracimab-the-first-and-only-ticagrelor-reversal-agent-for-filing-and-priority-review/.
64. SERB Pharmaceuticals. Sfj Pharmaceuticals and Serb Pharmaceuticals present positive final results from pivotal Phase 3 REVERSE-IT trial of Bentracimab for the reversal of antiplatelet effects of ticagrelor in patients requiring surgery or experiencing major bleeding. 2025 [accessed 2025 Nov 5]. https://serb.com/news/sfj-pharmaceuticals-and-serb-pharmaceuticals-present-positive-final-results-from-pivotal-phase-3-reverse-it-trial-of-bentracimab-for-the-reversal-of-antiplatelet-effects-of-ticagrelor-in-patients-requ/.
65. Scholar Rock. Fda issues complete response letter (CRL) for Apitegromab as a treatment for patients with spinal muscular atrophy (SMA) solely related to observations identified at Catalent Indiana LLC fill-finish facility. 2025 [accessed 2025 Oct 7]. https://investors.scholarrock.com/news-releases/news-release-details/fda-issues-complete-response-letter-crl-apitegromab-treatment.
66. Crawford TO, Darras BT, Day JW, Dunaway Young S, Duong T, Nelson LL, Barrett D, Song G, Bilic S, Cote S, et al. Safety and efficacy of apitegromab in patients with spinal muscular atrophy types 2 and 3: the phase 2 TOPAZ study. Neurology. 2024;102(5):e209151. doi: 10.1212/WNL.0000000000209151.
67. Crawford TO, Day JW, De Vivo DC, Krueger JM, Mercuri E, Nascimento A, Pasternak A, Mazzone ES, Duong T, Song G, et al. Long-term efficacy, safety, and patient-reported outcomes of apitegromab in patients with spinal muscular atrophy: results from the 36-month TOPAZ study. Front. Neurol. 2024;15:1419791. doi: 10.3389/fneur.2024.1419791.
68. Crawford TO, Servais L, Mercuri E, Kölbel H, Kuntz N, Finkel RS, Krueger J, Batley K, Young SD, Marantz JL, et al. Safety and efficacy of apitegromab in nonambulatory type 2 or type 3 spinal muscular atrophy (SAPPHIRE): a phase 3, double-blind, randomised, placebo-controlled trial. Lancet Neurol. 2025;24(9):727–739. doi: 10.1016/S1474-4422(25)00225-X.
69. Novo Nordisk. Novo Nordisk submits Biologics license application (BLA) to FDA for Mim8; an investigational, prophylaxis treatment for people living with hemophilia A with or without inhibitors. 2025 [accessed 2025 Nov 4]. https://www.prnewswire.com/news-releases/novo-nordisk-submits-biologics-license-application-bla-to-fda-for-mim8-an-investigational-prophylaxis-treatment-for-people-living-with-hemophilia-a-with-or-without-inhibitors-302568867.html.
70. Novo Nordisk. Novo Nordisk's sales increased by 12% in Danish kroner and by 15% at CER in the first nine months of 2025; R&D pipeline progress continues. 2025 [accessed 2025 Nov 5]. https://www.novonordisk-us.com/media/news-archive/news-details.html?id=916446.
71. AstraZeneca. AstraZeneca's 9M and Q3 2025 financial results. 2025 [accessed 2025 Dec 5]. https://www.astrazeneca.com/content/dam/az/PDF/2025/9m-q3/9M-and-Q3-2025-results-announcement.pdf.
72. AstraZeneca. Gefurulimab dual-binding nanobody demonstrated statistically significant and clinically meaningful improvement in functional activities of daily living in adults with generalised myasthenia gravis in PREVAIL Phase III trial. 2025 [accessed 2025 Nov 4]. https://www.astrazeneca.com/media-centre/press-releases/2025/gefurulimab-nanobody-met-phase-iii-endpoints.html.
73. Kaplan R, Zhao Y, Tsai J, Dickinson B, Swanson T, Foster K, Bedian V. Preclinical pharmacology, pharmacokinetics, and pharmacodynamics of veligrotug, a full antagonist antibody to the IGF-1 receptor in development for thyroid eye disease. mAbs. 2025;17(1). doi: 10.1080/19420862.2025.2585616.

74. Viridian Therapeutics Inc. Viridian Therapeutics announces BLA acceptance and priority review for veligrotug for the treatment of thyroid eye disease. 2025 [accessed 2025 Dec 22]. https://investors.viridiantherapeutics.com/news/news-details/2025/Viridian-Therapeutics-Announces-BLA-Acceptance-and-Priority-Review-for-Veligrotug-for-the-Treatment-of-Thyroid-Eye-Disease/default.aspx.
75. Viridian Therapeutics, Inc.. Viridian corporate presentation. 2025 [Accessed 2025 Oct 31]. https://s203.q4cdn.com/807813999/files/doc_presentations/2025/Oct/21/VRDN-October-2025-Corp-Deck.pdf.
76. Viridian Therapeutics Inc. Viridian Therapeutics announces positive long-term durability data from the VELIGROTUG Phase 3 THRIVE clinical trial in patients with active thyroid eye disease (TED). 2025 [accessed 2025 Oct 31]. https://investors.viridiantherapeutics.com/news/news-details/2025/Viridian-Therapeutics-Announces-Positive-Long-Term-Durability-Data-from-the-Veligrotug-Phase-3-THRIVE-Clinical-Trial-in-Patients-with-Active-Thyroid-Eye-Disease-TED/default.aspx.
77. Viridian Therapeutics Inc. Viridian Therapeutics announces positive topline results from VELIGROTUG Phase 3 THRIVE-2 clinical trial in patients with chronic thyroid eye disease. 2024 [accessed 2025 Oct 31]. https://s203.q4cdn.com/807813999/files/doc_news/Viridian-Therapeutics-Announces-Positive-Topline-Results-from-Veligrotug-Phase-3-THRIVE-2-Clinical-Trial-in-Patients-with-Chronic-Thy-3CV7K.pdf.
78. Vanda Pharmaceuticals Inc. Vanda announces submission of biologics license application to the FDA for imsidolimab for the treatment of generalized pustular psoriasis. 2025 [accessed 2025 Dec 27]. https://vandapharmaceuticalsinc.gcs-web.com/node/16676/pdf.
79. Smieszek SP. Imsidolimab, a novel IgG4 IL36 receptor antagonist, was effective and well tolerated in patients with GPP. J Invest Dermatol. 2025;145(8):S206. https://www.jidonline.org/article/S0022-202X(25)02200-6/fulltext.
80. Vanda Pharmaceuticals Inc. Vanda Pharmaceuticals and Anaptys announce exclusive global license agreement for Vanda to develop and commercialize imsidolimab, an IL-36R antagonist. 2025 [accessed 2025 Nov 19]. https://vandapharmaceuticalsinc.gcs-web.com/node/16241/pdf.
81. Harbour BioMed. Harbour BioMed reports 2025 interim results. 2025 [accessed 2025 Oct 31]. https://www.harbourbiomed.com/news/248.html.
82. Yan C, Yue Y, Guan Y, Bu B, Ke Q, Duan R, Deng H, Xue Q, Jiang H, Xiao F, et al. Batoclimab vs placebo for generalized myasthenia gravis: a randomized clinical trial. JAMA Neurol. 2024;81(4):336–345. doi: 10.1001/jamaneurol.2024.0044.
83. Biopharma H. HanAll Biopharma announces orphan drug designation for batoclimab in Japan for active thyroid eye disease. 2025 [accessed 2025 Oct 21]. https://www.hanall.com/board_view.php?bo_table=press&idx=101.
84. Sunshine Guojian Pharmaceutical Co, Inc. Semi-annual report [Chinese]. 2025. accessed 2025 Oct 17. https://static.sse.com.cn/disclosure/listedinfo/announcement/c/new/2025-08-30/688336_20250830_A3C5.pdf.
85. Sunshine Guojian Pharmaceutical Co, Inc. The phase III clinical trial of SSGJ-608 injection for the treatment of moderate to severe plaque psoriasis achieved all efficacy endpoints [Chinese]. 2024 [accessed 2025 Oct 17]. https://static.sse.com.cn/disclosure/listedinfo/announcement/c/new/2024-08-10/688336_20240810_86RA.pdf.
86. Akeso Inc. First half report. 2025 [accessed 2025 Oct 16]. https://www.akesobio.com/media/2656/akeso-2025-h1-english-final.pdf.
87. Akeso Biopharma Co. Ltd.. Akeso released complete Phase 3 data for gumokimab (AK111, IL-17) in moderate to severe plaque psoriasis. 2024 [accessed 2025 Oct 16]. https://www.akesobio.com/en/media/akeso-news/241028/.
88. Akeso Biopharma Co. Ltd.. Akeso reports phase 3 trials show positive results: gumokimab (IL-17) for ankylosing spondylitis and manfidokimab (IL-4Rα) for atopic dermatitis achieve primary endpoints. 2025 [accessed 2025 Oct 16]. https://www.akesobio.com/en/media/akeso-news/250825/.
89. Long C, Wang W, Hao X, Yu C, Feng Y, Tu C, Sun S, Bian L, Liu Z, Wang L. Development of a novel bispecific antibody GR1801 for rabies. J Med Virol. 2023;95(8). doi: 10.1002/jmv.29016.
90. China Medical System Holdings Limited.. China Medical System (867.HK; 8A8.SG) signed collaboration agreements for two innovative biologics used for passive immunization against tetanus and rabies. 2025 [accessed 2025 Oct 30]. https://www.globenewswire.com/news-release/2025/09/23/3154384/0/en/China-Medical-System-867-HK-8A8-SG-Signed-Collaboration-Agreements-for-Two-Innovative-Biologics-Used-for-Passive-Immunization-Against-Tetanus-and-Rabies.html.
91. Genrix (Shanghai). Biopharmaceutical Co. Ltd. Voluntary disclosure of the new drug application for GR2001 injection announcement of Acceptance [Chinese]. 2025 [accessed 2025 Oct 8]. https://static.sse.com.cn/disclosure/listedinfo/announcement/c/new/2025-05-23/688443_20250523_M0NU.pdf.
92. Beijing Tianguangshi Biotechnology Co. Announcement regarding the acceptance of the new drug application for its core product, MIL62. 2025 [accessed 2025 Oct 9]. https://pdf.dfcfw.com/pdf/H2_AN202505261679503982_1.pdf?1748277899000.pdf.
93. PhIRDA. The second potential first! Tianguangshi's CD20 antibody has been approved for marketing approval for the treatment of primary membranous nephropathy. 2025 [accessed 2025 Oct 10]. https://www.phirda.com/artilce_40447.html?module=trackingCodeGenerator.
94. Huang D, Wu L, Chen J. Type ii anti-CD20 antibody MIL62 in AQP4-positive neuromyelitis optica spectrum disorder: a multicenter, randomized, double-blind, phase III trial. Presented at: 2025 ECTRIMS Congress;

September 24-26; Barcelona, Spain; https://conferences.medicom-publishers.com/specialisation/neurology/ectrims-2025/novel-anti-cd20-antibody-shows-efficacy-in-aqp4-igg-positive-nmosd/.
95. Cui Z, Wei M, Li P, Wang R, Li H, Zhang H, Lin HL, Lan CX, Luo P, Zhou H, et al. Efficacy and safety of MIL62, a glycoengineered type II anti-CD20 antibody, in primary membranous nephropathy: a phase 3, randomized, controlled trial. J Am Soc Nephrol. 2025;36(10S). doi: 10.1681/ASN.202564sha3em.
96. 3SBio. The marketing application of 3SBio's anti-IL-1β monoclonal antibody SSGJ-613 has been accepted by the National Medical Products Administration. 2025 [accessed 2025 Nov 12]. https://www.3s-guojian.com/news/details/231.html.
97. Connect Biopharma Holdings Ltd. Corporate presentation September 2025. [accessed 2025 Oct 14]. https://investors.connectbiopharma.com/static-files/6e87b037-f42b-40b6-a811-e577db819e4b.
98. Connect Biopharma Holdings Ltd. Connect Biopharma's exclusive licensee in China, Simcere Pharmaceutical, announced submission of its new drug application for rademikibart for the treatment of atopic dermatitis in China. 2025 [accessed 2025 Oct 11]. https://investors.connectbiopharma.com/news-releases/news-release-details/connect-biopharmas-exclusive-licensee-china-simcere.
99. Silverberg JI, Strober B, Feinstein B, Xu J, Guttman-Yassky E, Simpson EL, Li P, Longphre M, Song J, Guo J, et al. Efficacy and safety of rademikibart (CBP-201), a next-generation mAb targeting IL-4Rα, in adults with moderate to severe atopic dermatitis: a phase 2 randomized trial (CBP-201-WW001). J Allergy Clin Immunol. 2024;153(4):1040–1049.e12. doi: 10.1016/j.jaci.2023.11.924.
100. Zhang J, Silverberg JI, Guo J, Yun J, Pan W, Wei Z, Collazo R. Rademikibart monotherapy for moderate-to-severe atopic dermatitis in a 1-year, randomized, phase II trial (SEASIDE CHINA): initial two-week dosing, followed by two-week or four-week dosing. Br J Dermatol. 2025;194(1):47–58. doi: 10.1093/bjd/ljaf347.
101. Kerwin E, Yang T, Su N, Guo J, Adivikolanu R, Longphre M, Wang J, Yun J, Pan W, Wei Z, et al. Rademikibart treatment for moderate-to-severe uncontrolled asthma: a phase 2b randomized clinical trial. Am J Respir Crit Care Med. 2025;211(5):749–758. doi: 10.1164/rccm.202409-1708OC.
102. Chongqing Genrix Biopharmaceutical. The marketing application for Zhixiang Jintai GR1802 injection for moderate to severe atopic dermatitis has been accepted [Chinese]. 2025 [accessed 2025 Nov 11]. https://genrixbio.com/#/article_details?id=83.
103. Wang S, Zhang L, Liang Y, Zhang S, Wang J, Man X, Ji C, Chen R, Xiang G, Meng Z, et al. Efficacy and safety of GR1802 in patients with moderate-to-severe atopic dermatitis: a multicenter, randomized, double-blind, placebo-controlled phase 2 trial. Dermatologic Ther. 2025 Mar. 2025(1). doi: 10.1155/dth/6903760.
104. Huabo Biopharm (Shanghai). The marketing application of HB0034, the first domestically developed IL-36R monoclonal antibody, has been accepted. 2025 [accessed 2025 Nov 14]. https://www.huabobio.com/newsshow/249.html.
105. Salubris Pharmaceuticals Ltd. Salubris' SAL003 BLA accepted by NMPA for lipid-lowering therapy. 2025 [accessed 2025 Nov 13]. https://www.linkedin.com/posts/salubris-pharmaceuticals_salubrispharma-pipelinemilestone-biologicinnovation-activity-7380932319768891392-V4OC.
106. China Medical System Holdings Limited. Cms (867.HK; 8A8.SG) NDA for AD indication of long-acting anti-IL-4Rα humanized monoclonal antibody injection MG-K10 accepted in China. 2025 [accessed 2025 Nov 4]. https://www.globenewswire.com/news-release/2025/10/30/3177285/0/en/CMS-867-HK-8A8-SG-NDA-for-AD-Indication-of-Long-acting-Anti-IL-4Rα-Humanized-Monoclonal-Antibody-Injection-MG-K10-Accepted-in-China.html.
107. Hunan Mabgeek Biotechnology Co., Ltd. MabGeek biotech receives approval notice for phase III clinical trial of chronic spontaneous urticaria (CSU). 2025 [accessed 2025 Nov 4]. https://www.mabgeek.com/index.php?c=show&id=39.
108. Shanghai Junshi Biosciences Co., Ltd. Junshi Biosciences announces acceptance of the NDA for Roconkibart (IL-17A) for the treatment of moderate to severe plaque psoriasis. 2025 [accessed 2025 Dec 5]. https://www.globenewswire.com/news-release/2025/12/05/3200830/0/en/Junshi-Biosciences-Announces-Acceptance-of-the-NDA-for-Roconkibart-IL-17A-for-the-Treatment-of-Moderate-to-Severe-Plaque-Psoriasis.html.
109. Shanghai Junshi Biosciences Co., Ltd. Junshi Biosciences announces the Phase 3 study of JS005 (IL-17A) for the treatment of moderate to severe plaque psoriasis met primary endpoints. 2025 [accessed 2025 Sep 23]. https://www.globenewswire.com/news-release/2025/09/07/3145717/0/en/Junshi-Biosciences-Announces-the-Phase-3-Study-of-JS005-IL-17A-for-the-Treatment-of-Moderate-to-Severe-Plaque-Psoriasis-Met-Primary-Endpoints.html.
110. Livzon Pharmaceutical Group, Inc. Marketing authorization application for Lecankitug injection accepted for review by China's National Medical Products Administration. 2025 [accessed 2025 Dec 27]. https://www.hkexnews.hk/listedco/listconews/sehk/2025/1224/2025122400984.pdf.
111. Livzon Pharmaceutical Group, Inc. Achievement of the primary endpoint in phase III clinical trial of recombinant anti-human IL-17A/F humanized monoclonal antibody injection. 2025 [accessed 2025 Dec 24]. https://www.hkexnews.hk/listedco/listconews/sehk/2025/0721/2025072101082.pdf.
112. Bio-Thera Solutions, Ltd.. The marketing authorization application for Bio-thera's BAT5906 (a recombinant anti-VEGF humanized monoclonal antibody) (nAMD) has been accepted by the NMPA. 2025 [accessed 2025 Dec 27]. https://www.bio-thera.com/col_24/1337.html.

113. Bio-Thera Solutions, Ltd.. Bio-thera Solutions announced positive results from its pivotal Phase III registration clinical trial of BAT5906 (a recombinant anti-VEGF humanized monoclonal antibody) targeting wAMD. 2025 [accessed 2025 Dec 27]. https://www.bio-thera.com/col_24/1189.html.
114. Pfizer. Second quarter 2025 earnings teleconference. 2025 [accessed 2025 Oct 9]. https://s206.q4cdn.com/795948973/files/doc_financials/2025/q2/Q2-2025-Earnings-Charts-FINAL.pdf.
115. Shore ND, Powles TB, Bedke J, Galsky MD, Palou Redorta J, Ku JH, Kretkowski M, Xylinas E, Alekseev B, Ye D, et al. Sasanlimab plus BCG in BCG-naive, high-risk non-muscle invasive bladder cancer: the randomized phase 3 CREST trial. Nat Med. 2025;31(8):2806–2814. doi: 10.1038/s41591-025-03738-z.
116. AbbVie. AbbVie submits biologics license application (BLA) to U.S. FDA for Pivekimab sunirine (PVEK), an investigational antibody-drug conjugate (ADC) to treat rare cancer with limited treatment options. 2025 [accessed 2025 Oct 14]. https://news.abbvie.com/2025-09-30-AbbVie-Submits-Biologics-License-Application-BLA-to-U-S-FDA-for-Pivekimab-sunirine-PVEK-an-Investigational-Antibody-Drug-Conjugate-ADC-to-Treat-Rare-Cancer-with-Limited-Treatment-Options.
117. Pemmaraju N, Marconi G, Montesinos P, Lane AA, Mazzarella L, Sallman DA, Ulrickson M, Schiller GJ, Erba HP, Wang ES, et al. Efficacy and safety of pivekimab sunirine (PVEK) in patients (pts) with blastic plasmacytoid dendritic cell neoplasm (BPDCN) in the CADENZA study. J Clin Oncol. 2025;43(16_suppl):6502–6502. doi: 10.1200/JCO.2025.43.16_suppl.6502.
118. Daver NG, Montesinos P, DeAngelo DJ, Wang ES, Papadantonakis N, Todisco E, Sweet KL, Pemmaraju N, Lane AA, Torres-Miñana L, et al. Pivekimab sunirine (IMGN632), a novel CD123-targeting antibody–drug conjugate, in relapsed or refractory acute myeloid leukaemia: a phase 1/2 study. Lancet Oncol. 2024;25(4):388–399. doi: 10.1016/S1470-2045(23)00674-5.
119. Jiangsu HengRui Pharmaceuticals Co. Ltd. Management presentation. 2025 [accessed 2025 Oct 9]. https://www.hengrui.com/images/investor/投资者推介材料-英文.pdf.
120. Peng Z, Wang J, Zhang Y, Li H, Zhao Q, Zhu X, Wei S, Cheng Y, Yang W, Yao J, et al. Lba60 phase III study of SHR-1701 versus placebo in combination with chemo as first-line (1L) therapy for HER2-negative gastric/gastroesophageal junction adenocarcinoma (G/GEJA). Ann Oncol. 2024;35(2):S1250. https://www.annalsofoncology.org/article/S0923-7534(24)03882-1/fulltext.
121. Wei H, Cai H, Jin Y, Wang P, Zhang Q, Lin Y, Wang W, Cheng J, Zeng N, Xu T, et al. Structural basis of a novel heterodimeric Fc for bispecific antibody production. Oncotarget. 2017;8(31):51037–51047. doi: 10.18632/oncotarget.17558.
122. Alphamab Oncology. The new drug application for KN026 (anbenitamab injection) has been accepted by the National Medical Products Administration. 2025 [accessed 2025 Oct 21]. https://www.alphamabonc.com/en/html/news/2708.html.
123. Alphamab Oncology. Research updates on a Phase III clinical trial of anbenitamab (KN026) presented at ESMO Congress 2025 LBA oral presentation session. 2025 [accessed 2025 Nov 11]. https://www.alphamabonc.com/en/html/news/2719.html.
124. SystImmune, Inc.. SystImmune to receive milestone payment from Bristol Myers Squibb under Iza-Bren collaboration. 2025 [accessed 2025 Dec 10]. https://systimmune.com/news—systimmune-to-receive-milestone-payment-from-bristol-myers-squibb-under-iza-bren-collaboration.
125. Yang Y, Zhou H, Tang L, Qiu S, Han Y, Ji D, Chen X, Lei F, Qu S, Deng B, et al. Izalontamab brengitecan, an EGFR and HER3 bispecific antibody-drug conjugate, versus chemotherapy in heavily pretreated recurrent or metastatic nasopharyngeal carcinoma: a multicentre, randomised, open-label, phase 3 study in China. Lancet. 2025;406(10516):2235–2243. doi: 10.1016/S0140-6736(25)01954-3.
126. Regeneron Pharmaceuticals, Inc. Regeneron announces positive Phase 3 trial in adults with ultra-rare genetic disorder fibrodysplasia ossificans progressiva (FOP), demonstrating that garetosmab prevents greater than 99% of abnormal bone formation. 2025 [accessed 2025 Sep 23]. https://investor.regeneron.com/news-releases/news-release-details/regeneron-announces-positive-phase-3-trial-adults-ultra-rare.
127. Dyne Therapeutics, Inc. Dyne therapeutics announces positive topline results from Phase 1/2 DELIVER trial of Z-Rostudirsen in Duchenne muscular dystrophy (DMD). 2025 [accessed 2025 Dec 23]. https://investors.dyne-tx.com/news-releases/news-release-details/dyne-therapeutics-announces-fda-breakthrough-therapy-0.
128. Dyne Therapeutics, Inc. Dyne Therapeutics announces new long-term clinical data from Phase 1/2 DELIVER trial of DYNE-251 in Duchenne muscular dystrophy. 2025 [accessed 2025 Oct 14]. https://investors.dyne-tx.com/news-releases/news-release-details/dyne-therapeutics-announces-new-long-term-clinical-data-phase-12.
129. Etxaniz U, Marks I, Albin T, Diaz M, Bhardwaj R, Anderson A, Tyaglo O, Hoang T, Missinato MA, Svensson K, et al. AOC 1044 induces exon 44 skipping and restores dystrophin protein in preclinical models of Duchenne muscular dystrophy. Nucleic Acids Res. 2025;53(6):gkaf241. doi: 10.1093/nar/gkaf241.
130. Avidity Biosciences, Inc. Avidity Biosciences announces positive preBLA meeting with U.S. FDA for delzota in DMD44 with a submission planned for Q1 2026. 2025 [accessed 2025 Oct 22]. https://investors.aviditybiosciences.com/2025-10-13-Avidity-Biosciences-Announces-Positive-Pre-BLA-Meeting-with-U-S-FDA-for-del-zota-in-DMD44-with-a-Submission-Planned-for-Q1-2026.

131. Avidity Biosciences, Inc. Avidity Biosciences enters into agreement to be acquired by Novartis AG. 2025 [accessed 2025 Nov 3]. https://investors.aviditybiosciences.com/2025-10-26-Avidity-Biosciences-Enters-into-Agreement-to-be-Acquired-by-Novartis-AG.
132. Avidity Biosciences, Inc. Avidity Biosciences: Delzota demonstrated reversal of disease progression across key functional endpoints in EXPLORE44R and EXPLORE44OLE™ Phase 1/2 trial in people living with DMD44. 2025 [accessed 2025 Oct 22]. https://investors.aviditybiosciences.com/2025-09-10-Avidity-Biosciences-Del-zota-Demonstrated-Reversal-of-Disease-Progression-Across-Key-Functional-Endpoints-in-EXPLORE44-R-and-EXPLORE44-OLE-TM-Phase-1-2-Trial-in-People-Living-with-DMD44.
133. Novartis International AG. Novartis ianalumab first drug to reduce disease activity and patient burden in Sjögren's disease phase III trials. 2025 [accessed 2025 Dec 3]. https://www.novartis.com/news/media-releases/novartis-ianalumab-first-drug-reduce-disease-activity-and-patient-burden-sjogrens-disease-phase-iii-trials.
134. Grader-Beck T, Mariette X, Finzel S, Schiopu E, Papas A, Devauchelle-Pensec V, Dörner T, Sopala M, Zeng X, Noaiseh G, et al. Ianalumab demonstrates significant reduction in disease activity in patients with Sjogren's disease: efficacy and safety results from two global phase 3, randomized, placebo-controlled double-blind studies (NEPTUNUS-1 and NEPTUNUS-2). Presented at the American College of Rheumatology (ACR) Congress. [October 24-29, 2025; Chicago (IL). https://acrabstracts.org/abstract/ianalumab-demonstrates-significant-reduction-in-disease-activity-in-patients-with-sjogrens-disease-efficacy-and-safety-results-from-two-global-phase-3-randomized-placebo-controlled-double-blind-s/
135. An nexon Inc. An Nexon reports third quarter 2025 financial results, portfolio progress and key anticipated milestones. 2025. [accessed 2025 Dec 3]. https://ir.annexonbio.com/news-releases/news-release-details/annexon-reports-third-quarter-2025-financial-results-portfolio.
136. Johnson N, Day J, Hamel J, Thornton C, Subramony SH, Payam Soltanzadeh P, Statland J, Freimer M, Quan D, Knisely B, et al. Gbs-02: a phase 3 study to evaluate efficacy, safety, pharmacokinetics, and pharmacodynamics of ANX005 in patients with guillain-barré syndrome (GBS). Presentation at The American Academy Of Neurology Annual Meeting; April 5-9, 2025; [accessed April 5-9] 2025; San Diego (CA). https://annexonbio.com/wp-content/uploads/2025/04/AAN-GBS-02-Study-Encore-Presentation_Final.pdf
137. Zenas BioPharma, Inc. Transformational global collaboration with InnoCare Pharma. 2025 [accessed 2025 Oct 22]. https://investors.zenasbio.com/static-files/68b70f32-04ae-4c55-ab00-40625788c8d8.
138. Zenas BioPharma, Inc. Zenas BioPharma announces positive results from Phase 2 Moonstone trial of obexelimab in relapsing multiple sclerosis. 2025 [accessed 2025 Nov 2]. https://investors.zenasbio.com/news-releases/news-release-details/zenas-biopharma-announces-positive-results-phase-2-moonstone.
139. Avidity Biosciences, Inc. Avidity Biosciences reports third quarter 2025 financial results and recent highlights. 2025. [accessed 2025 Dec 3]. https://investors.aviditybiosciences.com/2025-11-10-Avidity-Biosciences-Reports-Third-Quarter-2025-Financial-Results-and-Recent-Highlights.
140. Avidity Biosciences, Inc. Initial results of the Phase 2 openlabel extension study of AOC 1001 in adults with myotonic dystrophy type 1: MARINAOLE™. Muscular Dystrophy Association Clinical & Scientific Conference, Orlando, FL; 2024. [accessed 2025 Oct 22]. https://www.aviditybiosciences.com/sites/default/files/2025-04/MDA-2024-MARINA-OLE-DM1_v6.0_STC.pdf.
141. Kodiak Sciences Inc. Corporate Presentation. 2025 [accessed 2025 Dec 3]. https://ir.kodiak.com/static-files/bdef019c-988b-4c2b-a70c-c8bf8f74578d.
142. Wong RW, Janer D, Zawadzki R, Perlroth DV, Velazquez-Martin P. Tarcocimab tedromer (KSI-301) 5mg: outcomes of the phase 3 GLOW study in patients with non-proliferative diabetic retinopathy. Invest Ophthalmol Vis Sci. 2024;65(7):1773. https://iovs.arvojournals.org/article.aspx?articleid=2794551.
143. MedPath. Ksi-301 advanced drug monograph: clinical and strategic analysis. [accessed 2025 Oct 20]. https://trial.medpath.com/drug/report/93b7968fc594f76a.
144. England E, Rees DG, Scott IC, Carmen S, Chan DTY, Chaillan Huntington CE, Houslay KF, Erngren T, Penney M, Majithiya JB. Tozorakimab (MEDI3506): an anti-IL-33 antibody that inhibits IL-33 signaling via ST2 and RAGE/EGFR to reduce inflammation and epithelial dysfunction. Sci Rep. 2023;13(1):9825. doi: 10.1038/s41598-023-36642-y.
145. AstraZeneca. Clinical trials appendix: Q3 2025 results update. 2025 [accessed 2025 Dec 5]. https://www.astrazeneca.com/content/dam/az/PDF/2025/9m-q3/9M-and-Q3-2025-results-clinical-trials-appendix.pdf.
146. Singh D, Guller P, Reid F, Doffman S, Seppälä U, Psallidas I, Moate R, Smith R, Kiraga J, Jimenez E, et al. A phase 2a trial of the IL-33 monoclonal antibody tozorakimab in patients with COPD: FRONTIER-4. Eur Respir J. 2025;66(1):2402231. doi: 10.1183/13993003.02231-2024.
147. Rezolute, Inc.. A late-stage rare disease company treating hyperinsulinism. 2025 [accessed 2025 Nov 8]. https://ir.rezolutebio.com/_assets/_66887b51f090e11fb8fad4eb8fb4fbef/rezolutebio/db/693/10050/pdf/RZLT+Corporate+Presentation±+November+2025_New+Template.pdf.
148. Rezolute, Inc.. Rezolute reports first quarter fiscal 2026 financial results and provides business update. 2025. accessed Nov 8. https://ir.rezolutebio.com/news/detail/366/rezolute-reports-first-quarter-fiscal-2026-financial-results-and-provides-business-update.
149. Qyuns Therapeutics Co. Ltd. Interim report. 2025 [accessed 2025 Sep 26]. https://www.qyuns.net/upload/en/file/2025-09/col274/1758292342833.pdf.

150. Qyuns Therapeutics Co. Ltd. Phase III clinical trial of Qx002N for ankylosing spondylitis reaches primary endpoint. 2025 [accessed 2025 Sep 26]. https://www.qyuns.net/upload/en/file/2025-02/col274/1740402398319.pdf.
151. Qyuns Therapeutics Co. Ltd. Interim results 2025. 2025 [accessed 2025 Sep 28]. https://www.qyuns.net/upload/en/file/2025-09/col232/1758017205581.pdf.
152. LongBio Pharma (Suzhou) Co. Ltd.. Application proof of LongBio Pharma (Suzhou) Co. Ltd. [Accessed 2025 Nov 2]. https://www1.hkexnews.hk/app/sehk/2025/107640/documents/sehk25082101308.pdf.
153. Guan K, Liu S, Feng Y, Li L, Zhu X, Sun NC, Ma H, Yang J, Han C, Liu H, et al. Lp003, a novel highaffinity antiIge antibody for inadequately controlled seasonal allergic rhinitis: a multicenter, randomized, doubleblind, placebocontrolled phase 2 clinical trial. Clin Transl Allergy. 2025;15(6). doi: 10.1002/clt2.70074.
154. Viridian Therapeutics, Inc. Viridian Therapeutics announces completion of enrollment in both REVEAL clinical trials and positive portfolio updates. 2025 [accessed 2025 Oct 31]. https://s203.q4cdn.com/807813999/files/doc_news/Viridian-Therapeutics-Announces-Completion-of-Enrollment-in-both-REVEAL-Clinical-Trials-and-Positive-Portfolio-Updates-2025.pdf.
155. Zhang J, Zhao Y, Zhou Q, Xu Y. 611 is a novel monoclonal antibody that targets interleukin4Rα. Br J Dermatol. 2024;191(Suppl 1):i54–i55. doi: 10.1093/bjd/ljae090.112.
156. Moomoo Technologies Inc. Pacific Securities maintains a 'buy' rating for 3SBio Inc. (01530), citing its steady performance and abundant catalysts. 2025 [accessed 2025 Nov 1]. https://www.moomoo.com/news/post/58770421/broker-focus-pacific-securities-maintains-a-buy-rating-for-3sbio?level=1&data_ticket=1751304958912009.
157. Zhao Y, Zhang L, Wu L, Duan X, Ji C, Xiao R, Ji M, Liu L, Yang B, Hu G, et al. Efficacy and safety of SSGJ611 in Chinese adults with moderate to severe atopic dermatitis: results from a phase II trial. Dermatol Thera. 2025;15(4):857–867. doi: 10.1007/s13555-025-01368-4.
158. Wynne CJ, Cole A, Lemech C, Wang G, Zhang Y, Chen B, Wang M, Li B, Xia M, Sinclair R. Safety, pharmacokinetics and preliminary efficacy of IL4-Rα monoclonal antibody AK120 in both healthy and atopic dermatitis subjects: a phase I, randomized, two-part, double-blind, placebo-controlled, dose-escalation, first-in-human clinical study. Dermatol Ther (heidelb). 2023;13(10):2357–2373. doi: 10.1007/s13555-023-01010-1.
159. Akeso Biopharma. Safety, pharmacokinetics, pharmacodynamics, and efficacy of AK120 in participants with moderate-to-severe atopic dermatitis: results from a randomized, double-blind, placebo-controlled phase II clinical trial. 2025 Sep 17-20 EADV Congress, Abst 66. [accessed 2025 Oct 18]. https://s3.eu-central-1.amazonaws.com/m-anage.com.storage.eadv/abstracts_congress_2025/52111.pdf.
160. Subbiah V, Chawla SP, Conley AP, Wilky BA, Tolcher A, Lakhani NJ, Berz D, Andrianov V, Crago W, Holcomb M, et al. Preclinical characterization and phase I trial results of INBRX-109, a third-generation, recombinant, humanized, death receptor 5 agonist antibody, in chondrosarcoma. Clin Cancer Res. 2023;29(16):2988–3003. doi: 10.1158/1078-0432.CCR-23-0974.
161. Inhibrx Biosciences, Inc. Inhibrx Biosciences reports positive topline results from its registrational trial of ozekibart (INBRX-109) in chondrosarcoma and provides updates on colorectal cancer and Ewing sarcoma expansion cohorts. 2025. [accessed 2025 Oct 29]. https://inhibrxbiosciences.investorroom.com/2025-10-23-Inhibrx-Biosciences-Reports-Positive-Topline-Results-from-its-Registrational-Trial-of-Ozekibart-INBRX-109-in-Chondrosarcoma-and-Provides-Updates-on-Colorectal-Cancer-and-Ewing-Sarcoma-Expansion-Cohorts.
162. AstraZeneca. License agreement with KYM Biosciences for CMG901, a Claudin-18.2 antibody drug conjugate, completed. 2023 [accessed 2024 Sep 2]. https://www.astrazeneca.com/media-centre/press-releases/2023/astrazeneca-completes-agreement-with-kym-for-cmg901.html#.
163. BioNTech. BioNTech announces third quarter 2025 financial results and corporate update. 2025 [accessed 2025 Nov 4]. https://investors.biontech.de/news-releases/news-release-details/biontech-announces-third-quarter-2025-financial-results-and.
164. BioNTech. BioNTech and DualityBio announce phase 3 trial of ADC candidate BNT323/DB-1303 met primary endpoint of progression free survival in HER2-positive metastatic or unresectable breast cancer. 2025 [accessed 2025 Nov 5]. https://investors.biontech.de/news-releases/news-release-details/biontech-and-dualitybio-announce-phase-3-trial-adc-candidate.
165. Daiichi Sankyo. Ifinatamab deruxtecan demonstrated clinically meaningful response rates in patients with extensive-stage small cell lung cancer in IDeate-Lung01 Phase 2 trial. 2025 [accessed 2025 Sep 23]. https://www.daiichisankyo.com/files/news/pressrelease/pdf/202509/20250907_E.pdf.
166. Fierce Biotech. Patient deaths prompt partial hold for Daiichi-Merck's global phase 3 ADC program. 2025 [accessed 2025 Dec 19]. https://www.fiercebiotech.com/biotech/patient-deaths-prompt-partial-hold-daiichi-mercks-global-phase-3-adc-program.
167. Merus NV. Genmab to acquire Merus, expanding late-stage pipeline and accelerating into a wholly owned model. 2025 [accessed 2025 Sep 29]. https://ir.merus.nl/news-releases/news-release-details/genmab-acquire-merus-expanding-late-stage-pipeline-and.

168. van Herpen CML, Daste A, Arrazubi V, De Boer JP, Rojas KI, Clatot F, Fontana E, Harandi A, Hellyer J, Hollebecque A, et al. Petosemtamab (MCLA-158) with pembrolizumab as first-line (1L) treatment of PD-L1+ recurrent/metastatic (r/m) head and neck squamous cell carcinoma (HNSCC): phase 2 trial. J Clin Oncol. 2025;43(16_suppl):6024–6024. https://ascopubs.org/doi/10.1200/JCO.2025.43.16_suppl.6024.
169. Machiels JP, Fayette J, Haddad R, Adkins D, Gillison M, Harrington KJ, Kim SB, Le Tourneau C, Psyrri A, Rosenberg A, et al. Liger-HN phase III trials of petosemtamab + pembrolizumab and petosemtamab monotherapy in recurrent or metastatic HNSCC. Future Oncol. 2025;21(16):2007–2016. doi: 10.1080/14796694.2025.2511470.
170. Wen M, Yu A, Park Y, Calarese D, Gerber HP, Yin G. Homogeneous antibody-drug conjugates with dual payloads: potential, methods and considerations. MAbs. 2025;17(1):2498162. doi: 10.1080/19420862.2025.2498162.
171. Taylor & Francis. Artificial intelligence and machine learning in antibody development. mAbs. 2025 [accessed 2025 Nov 18]. https://www.tandfonline.com/journals/kmab20/collections/ai-ml-antibody-development.