Global Allergic Disease Drugs Market and Complement Inhibitors Market

Independent Market Study

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For and on behalf of
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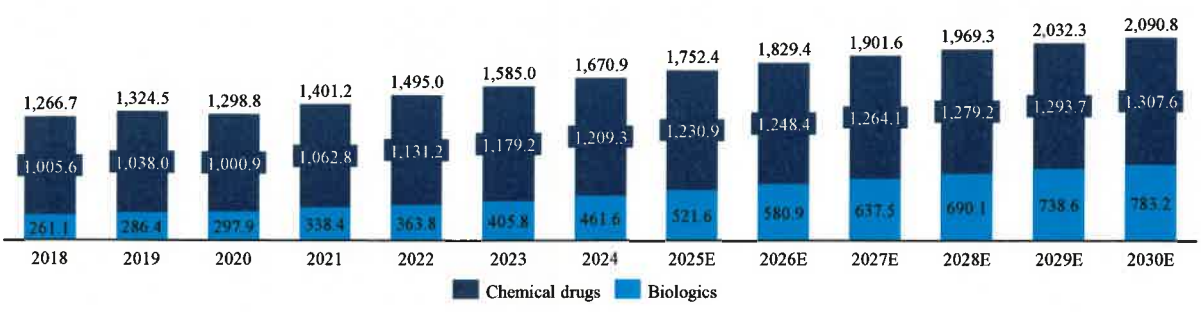
Historical and Forecast of Global Pharmaceutical Market Size

- *The global pharmaceutical market was valued USD1,670.9 billion in 2024, and is expected to reach USD2,090.8 billion in 2030, representing a CAGR of 3.8% during this period.*
- *Due to the high efficacy in treating a wide range of diseases, increasing R&D investment, significant development in biotechnology and increasing affordability, the global biologics segment has and is expected to continue to grow in a rapid pace. Both global and China biologics industries are highly competitive, with a large number of competitors with significant resources and brand awareness, and may be deeply entrenched in certain market segments, whether by geographic region or by drug type. The global biologics market increase with a CAGR of 9.2% from 2024 to 2030.*

Global Pharmaceutical Market, 2018-2030E

Period	Chemical	Biologics	Total
2018-2024	3.1%	10.0%	4.7%
2025E-2030E	1.3%	9.2%	3.8%



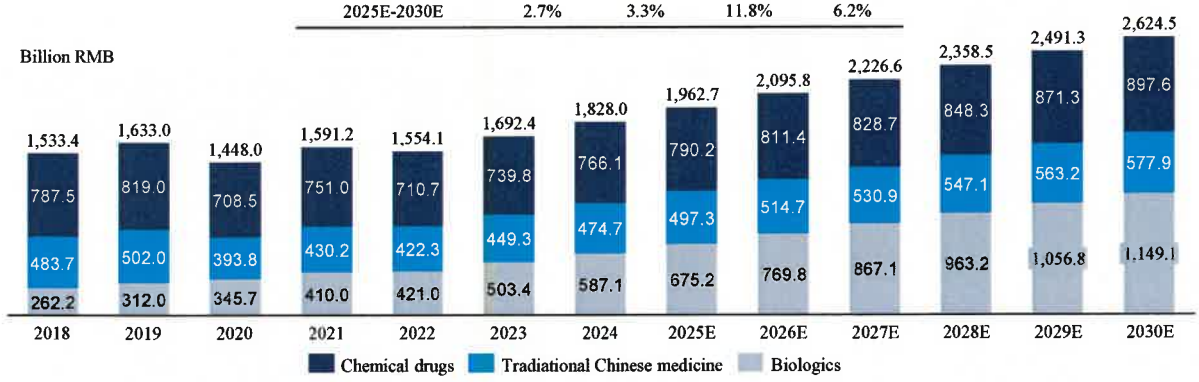
Source: Frost & Sullivan Analysis

Historical and Forecast of Pharmaceutical Market in China

- *The pharmaceutical market in China, accompanying with the growth of economic development and healthcare demand, increased from RMB1,533.4 billion in 2018 to RMB1,828.0 billion in 2024 with a CAGR of 3.0%. By 2030, the pharmaceutical market in China is expected to generate a revenue of RMB2,624.5 billion at wholesale price level.*
- *The China biologics market is expected to expand with a CAGR of 11.8% from 2024 to 2030.*

Pharmaceutical Market in China, 2018-2030E

Period	Chemical	TCM	Biologics	Total
2018-2024	-0.5%	-0.3%	14.4%	3.0%
2025E-2030E	2.7%	3.3%	11.8%	6.2%



Source: Frost & Sullivan Analysis

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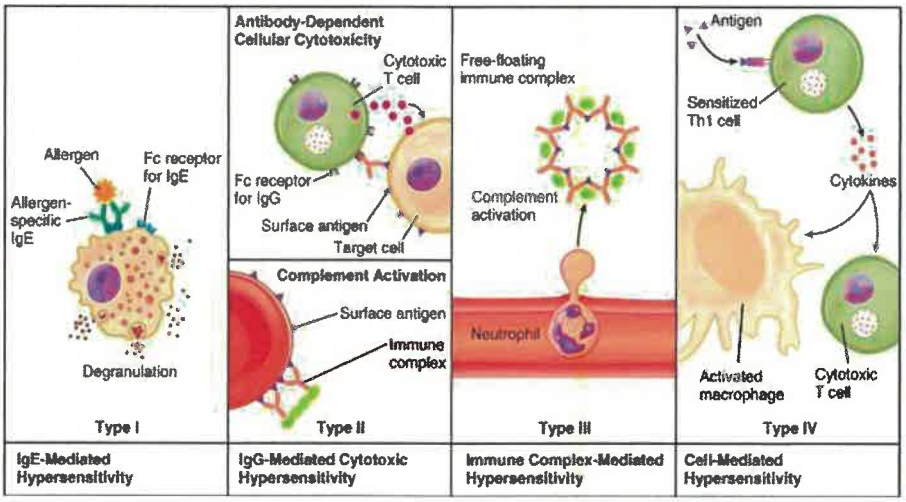
Pathologic Mechanism of Allergic Diseases

- Allergy is one of a class of immune system responses that are termed hypersensitivity reactions. These are harmful immune responses that produce tissue injury and may cause serious allergic disease.
- Hypersensitivity is a state of altered reactivity in which the body reacts with an exaggerated immune response to a foreign substance. There are four traditional classifications for hypersensitivity reactions, and these include Type I, Type II, Type III, and Type IV reactions.

Mechanism of Four Traditional Hypersensitivity Reactions



- **Type I hypersensitivity:** it is an immediate type as physiological symptoms appear within seconds to minutes.
- **Type II hypersensitivity:** it is called cytotoxic hypersensitivity. Its symptoms appear within minutes to hours.
- **Type III hypersensitivity:** this type is an immune complex-mediated type where symptoms appear within several hours.
- **Type IV hypersensitivity:** it is a delayed allergic reaction. As it occurs after hours to days for symptoms to appear which is a long time for response.

Source: Literature Review, Frost & Sullivan Analysis

Overview of Four Types of Hypersensitivity

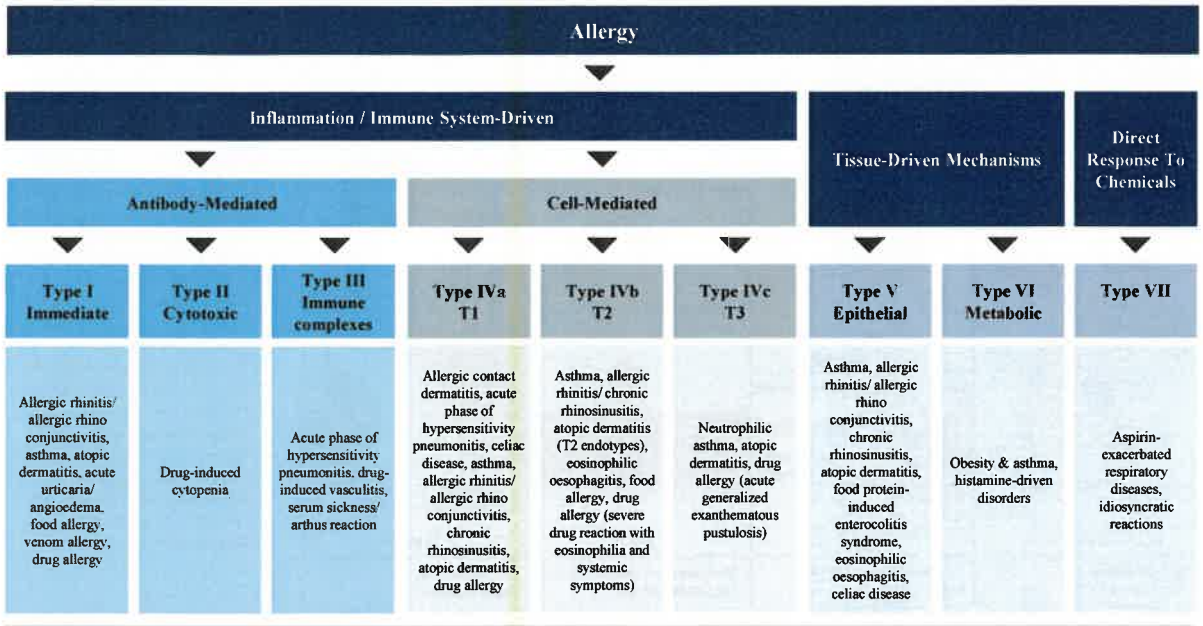
- Hypersensitivity is also known as hypersensitivity reaction, or intolerance. It implies unpleasant reactions played by immune system They are traditionally classified into four types (Type I–IV) based on the immunological mechanisms involved and the time course of the reaction.

Four Types of Hypersensitivity

Type	Mechanism	Characteristics	Related Diseases
Type I (Immediate)	IgE binds to mast cells and basophils, and re-exposure to allergens causes degranulation and release of histamine and other mediators.	This reaction is rapid and can occur within minutes of re-exposure, ranging from mild irritation to severe anaphylaxis; it is usually triggered by inhalation, ingestion, injection, or skin contact.	Allergic rhinitis, allergic asthma, conjunctivitis, anaphylaxis, food allergy, chronic spontaneous urticaria
Type II (Cytotoxic)	IgG or IgM binds to antigens on host cells, leading to cell destruction through complement activation or antibody-dependent cytotoxicity.	This type targets specific host cells, resulting in direct cytotoxicity; it can occur due to intrinsic antigens or foreign antigens that become associated with cell surfaces (e.g., drug binding).	Autoimmune hemolytic anemia, Goodpasture syndrome, erythroblastosis fetalis
Type III (Immune complex)	Antigen-antibody immune complexes deposit in tissues and trigger inflammation via complement activation.	The reaction may take hours to days to manifest and causes inflammation and tissue injury at sites of immune complex deposition, such as joints, skin, and kidneys.	Systemic lupus erythematosus, rheumatoid arthritis, glomerulonephritis, serum sickness
Type IV (Delayed)	Sensitized T cells recognize antigens and release cytokines, attracting immune cells and causing tissue damage.	This reaction is delayed, typically occurring 48–72 hours after antigen exposure. It involves cellular, not antibody, responses and is important in defense against intracellular pathogens but can cause tissue damage in autoimmune or allergic conditions.	Contact dermatitis, tuberculin reaction, transplant rejection, drug allergy

Classification of Allergic Diseases

- Allergic diseases can be categorized from the point of view of the mechanism of hypersensitivity reaction involved. Mechanisms of hypersensitivity reaction can be categorized as antibody-mediated, cell-mediated, tissue-driven mechanisms, and direct responses to chemicals.



Source: EAACI, Literature Review, Frost & Sullivan Analysis

Overview of Allergic Rhinitis

- Allergic rhinitis (AR) is a non-infectious chronic inflammatory disease of the nasal mucosa, primarily mediated by immunoglobulin E (IgE) after exposure to allergens in atopic individuals. It is characterized by nasal inflammation caused by allergens such as pollen, dust mites, animal dander and mold. AR is often associated with other allergic diseases, such as asthma and conjunctivitis. The main symptoms of AR include sneezing, runny nose, nasal congestion, and itching, and depending on the allergens involved, the symptoms may be seasonal or perennial. AR affects 10%-20% of the global population and has become a major chronic respiratory inflammatory disease, severely impacting the quality of life and socioeconomic conditions of patients.

Pathogenesis

AR occurs in atopic individuals upon exposure to allergens, primarily mediated by allergen-specific IgE, causing chronic inflammation of the nasal mucosa. Non-IgE mediated mechanisms and neuroimmune dysregulation also contribute. Inhaled allergens induce specific IgE production in the lymph nodes and nasal cavity. This IgE binds to high-affinity IgE receptors (FcεRI) on mast cells and basophils, sensitizing the individual. Upon subsequent exposure, the allergen activates mast cells and basophils, releasing inflammatory mediators like histamine and leukotrienes. These mediators stimulate sensory nerve endings and blood vessels in the nasal mucosa, causing vasodilation and increased secretion, leading to symptoms like itching, sneezing, and watery rhinorrhea, known as the early-phase reaction. Mediator release induces endothelial and epithelial cells to express adhesion molecules, chemokines, and cytokines, recruiting eosinophils, basophils, and Th2 cells, worsening Th2-dominated inflammation and causing nasal congestion in the late-phase reaction.

Symptoms

The typical symptoms of AR include paroxysmal sneezing, watery rhinorrhea, nasal itching, and nasal congestion. It may also be accompanied by ocular symptoms, such as eye itching, tearing, redness, and a burning sensation, especially in pollen allergy patients. Around 40% of AR patients may have comorbid bronchial asthma, with additional symptoms like wheezing, coughing, shortness of breath, and chest tightness. During an AR episode, the main clinical signs are bilateral pale and swollen nasal mucosa, edema of the inferior turbinates, and significant watery nasal discharge. Ocular signs typically include conjunctival congestion and edema, with occasional papillary reactions.

Cause

The development of AR is related to the interaction between genetic and environmental factors. AR has a genetic predisposition, with several genetic loci identified through genome-wide association studies. Epigenetic mechanisms also contribute to its development. Environmental factors and gut microbiota play a significant role in AR's pathogenesis. The "hygiene hypothesis" suggests that an overly clean environment reduces early-life exposure to microbes and parasites, increasing the risk of developing AR later. Additionally, non-IgE mediated inflammatory responses contribute to AR progression. Certain allergens can induce epithelial cells to produce cytokines and chemokines, promoting Th2 responses, or weaken epithelial tight junctions, disrupting the epithelial barrier and facilitating dendritic cell-allergen interactions.

Source: Frost & Sullivan Analysis

Overview of Allergic Asthma

- Allergic asthma (also known as atopic asthma or extrinsic asthma) is a type of asthma triggered and/or caused by allergens, formerly referred to as extrinsic asthma. It is one of the key clinical phenotypes of asthma, primarily driven by immune mechanisms mediated by Th2 cells. It is often associated with atopy and other allergic conditions such as eczema, allergic rhinitis, and food and drug allergies. Compared to non-allergic asthma, allergic asthma usually has an earlier onset, a familial genetic predisposition, and is commonly comorbid with conditions like eczema, allergic rhinitis, and food and drug allergies. Multiple global epidemiological surveys have shown that its incidence is rising annually, making it a widespread and long-term chronic respiratory disease.

Pathogenesis

The pathogenesis of allergic asthma involves both adaptive and innate immune system abnormalities. During the sensitization phase, allergens are captured by dendritic cells, activating T cells through MHC class II molecules, which promotes differentiation into Th2 cells. These Th2 cells secrete IL-4 and IL-13, stimulating B cells to produce allergen-specific IgE. The IgE binds to high-affinity receptors on mast cells or basophils, resulting in sensitization. Upon re-exposure to the allergen, mast cells degranulate, releasing histamine and leukotrienes, causing bronchoconstriction and mucosal edema, leading to an acute asthma response.

Simultaneously, IL-5 recruits eosinophils to the lungs, contributing to chronic airway inflammation. Innate immune factors like IL-25, IL-33, and TSLP activate ILC2 cells, further promoting Th2 amplification, exacerbating the inflammatory response, and contributing to airway remodeling.

Symptoms

The clinical symptoms of allergic asthma (AA) are similar to non-allergic asthma, characterized by recurrent wheezing, coughing, shortness of breath, and chest tightness, with symptoms often worsening at night or early morning. Most patients develop symptoms in childhood or adolescence, with a notable family history of atopy. AA is commonly associated with other allergic conditions, particularly allergic rhinitis (AR), and poor AR control can worsen asthma management.

AA tends to be long-lasting, with seasonal exacerbations, especially during high pollen seasons, and is more prone to exercise-induced wheezing. Studies show that AA patients experience a slower decline in lung function than those with non-allergic asthma, with higher FEV_1 and FEV_1/FVC values and a lower risk of irreversible airflow limitation. Sputum analysis often shows eosinophilic inflammation, and FeNO levels are typically elevated, reflecting type 2 inflammation.

Cause

The main cause of allergic asthma is exposure to allergens, which include inhalant and ingested types. Inhalant allergens are the primary triggers and include dust mites, pollen, fungi (molds), animal dander, and cockroaches. Dust mites are common in household items like mattresses and carpets. Pollen, especially from trees like cypress and birch, triggers seasonal asthma. Molds, found in damp places such as kitchens and bathrooms, worsen asthma in sensitized individuals. Pet allergies, particularly to cats and dogs, are more common, with their hair, dander, and saliva triggering reactions.

Ingested allergens, though less common, can cause severe reactions in sensitive people. Common food allergens include fish, shellfish, eggs, milk, fruits, peanuts, and legumes, particularly affecting children.

Source: Frost & Sullivan Analysis

Overview of Chronic Spontaneous Urticaria (CSU)

- Chronic Spontaneous Urticaria (CSU) is the most common type of chronic urticaria, defined by the persistence of hives and/or angioedema for more than six weeks, with no clear external triggers, causing skin and mucosal allergic reactions. Patients with CSU typically experience recurrent skin itching and hives, which can appear on any part of the body, usually accompanied by varying degrees of swelling. CSU is essentially a mast cell-mediated chronic inflammatory condition, characterized by its recurrent and unpredictable nature. The clinical symptoms include repeated hives, severe itching, and localized swelling, which typically resolve within hours but may vary between patients. In recent years, the global prevalence of CSU has been rising, significantly affecting patients' quality of life and emotional well-being, and imposing a considerable social and economic burden.

Pathogenesis

CSU involves the activation of mast cells and basophils, driven by autoimmune responses and influenced by external triggers. Abnormal activation of inflammatory cells and the release of mediators cause urticaria and pruritus. CSU mechanisms include two autoimmune responses: Type I, where IgE antibodies specific to self-antigens (such as anti-thyroid peroxidase, anti-DNA) bind to FcεRI on mast cells, triggering degranulation; and Type IIb, where IgG antibodies against IgE or FcεRI activate mast cells. These reactions release inflammatory mediators, increase vascular permeability, and stimulate skin nerve endings, causing typical symptoms. The coagulation and complement systems also play roles, enhancing inflammation. External triggers, including histamine-rich foods, food additives, temperature changes, common allergens, infections (bacteria, fungi, viruses), hormonal fluctuations, and stress, exacerbate CSU. These factors interfere with immune balance, further activating mast cells and worsening symptoms, indicating that CSU is a complex disease driven by immune dysregulation and external stimuli.

Symptoms

The main clinical symptom of CSU is recurrent hives, which are often pale red or white, with well-defined borders and varying shapes. These raised lesions are frequently accompanied by intense itching, and individual hives typically resolve naturally within 24 hours without leaving any marks. Some patients may also experience angioedema, commonly affecting the eyelids, lips, and limbs, where subcutaneous or mucosal swelling is prominent and resolves more slowly. Symptoms are often worse at night or in the early morning, and in severe cases, they can interfere with sleep and lead to psychological issues such as anxiety and depression. In some patients, hives may persist or occur frequently, with the condition lasting for months or even years.

Cause

The exact cause of Chronic Spontaneous Urticaria (CSU) remains unclear, and it is thought to result from the interaction of multiple internal and external factors. In some patients, symptoms may be triggered by the consumption of histamine-rich foods or food additives. Environmental changes, temperature fluctuations, and hormonal changes are also potential triggers. Certain patients have been found to have associations with latent infections, such as Helicobacter pylori and intestinal parasites. Psychological factors, such as stress, anxiety, and depression, are believed to exacerbate or provoke the condition. Additionally, autoimmune factors have been identified in some patients, including the presence of autoimmune thyroid disorders or autoantibodies. Due to the inability to pinpoint a singular cause in most patients, CSU is often classified as "idiopathic."

Source: Frost & Sullivan Analysis

Overview of Food allergy

- Food allergy is a condition caused by an abnormal immune response to dietary proteins, which can be triggered through IgE-mediated, non-IgE-mediated, or a combination of both mechanisms. Clinical manifestations are diverse, affecting the skin, gastrointestinal, respiratory, and cardiovascular systems, and it is one of the leading causes of allergic shock. The prevalence of food allergies is significantly higher in children aged 6-11 years in China compared to adults, and the incidence is on the rise. Studies have shown that factors such as early-life environment, gut microbiota, dietary habits, and mother-child immune interactions play a key role in the failure of oral tolerance development.

Pathogenesis

Food allergies arise from the failure to establish oral immune tolerance and abnormal immune responses. The gastrointestinal mucosal barrier, including physical and immune mechanisms, prevents allergens from entering the body. Physical barriers like tight epithelial cells, mucus, and digestive enzymes destroy antigens, while immune components such as sIgA, Peyer's patches, dendritic cells, and regulatory T cells maintain immune balance. In infants, high intestinal permeability allows antigens to trigger IgE-mediated sensitization. Antacids and impaired intestinal function increase sensitization risk. Sensitization begins when dendritic cells present antigens, and Th2 polarization leads to IgE production and mast cell activation. Some allergens bypass oral tolerance through the skin or respiratory tract, such as birch pollen causing oral allergies to apples. Skin barrier dysfunction in atopic dermatitis also contributes to sensitization. The gut microbiota plays a crucial role in immune regulation; imbalances increase sensitization risk. Non-IgE-mediated mechanisms, like in food protein-induced enterocolitis syndrome, may involve defects in TNF-α and TGF-β signaling.

Symptoms

Food allergy symptoms depend on the sensitizing food, underlying mechanism (IgE or T-cell mediated), and age. In infants, atopic dermatitis is common, often with gastrointestinal symptoms like nausea or diarrhea. As children grow, multi-system allergies may develop, including skin, respiratory, and digestive symptoms, some evolving into asthma and allergic rhinitis. By age 10, respiratory symptoms after food intake decrease, even with positive skin tests. In older children and adults, severe reactions such as urticaria, angioedema, or anaphylaxis are common. Some reactions occur only with both food intake and exercise, particularly in those sensitized to wheat or celery. T-cell mediated reactions may also affect the gastrointestinal tract, causing chronic symptoms like abdominal pain.

Cause

Food allergies are caused by exposure to allergens, immune abnormalities, and genetic factors. Almost all foods and additives can trigger sensitization. Common allergens in infants include milk, soy, eggs, peanuts, and wheat, while older children and adults are more likely to be allergic to nuts and seafood. Cross-reactions can occur between food and non-food allergens. Non-gastrointestinal sensitization, such as cross-reactions between peanut oil-based ointments or latex and bananas, also occurs. Genetic factors significantly affect susceptibility, with children of allergic parents at higher risk. Immune mechanisms include IgE-mediated reactions (e.g., urticaria, asthma), T-cell mediated responses (e.g., celiac disease), or a combination of both (e.g., atopic dermatitis, eosinophilic gastrointestinal disease), leading to acute, chronic, or mixed reactions.

Source: Frost & Sullivan Analysis

Overview of Chronic Rhinosinusitis with Nasal Polyps (CRSwNP)

- Chronic Rhinosinusitis with Nasal Polyps (CRSwNP) is a condition characterized by persistent inflammation of the nasal and sinus mucosa. Nasal polyps are benign inflammatory protrusions that are bilateral and originate from the ethmoid sinuses, often extending into the nasal cavity below the middle turbinates. It is commonly seen in individuals aged 40 to 60. The incidence is higher in men, although female patients typically experience more severe clinical symptoms. CRSwNP accounts for approximately 25-30% of patients with chronic rhinosinusitis (CRS). Although the proportion is relatively low, it has significant clinical importance due to its high recurrence rate and considerable impact on quality of life.

Pathogenesis

The pathogenesis of Chronic Rhinosinusitis with Nasal Polyps (CRSwNP) is not yet fully understood but involves epithelial barrier dysfunction, immune system dysregulation, and the participation of pathogens. Studies have shown abnormalities in the nasal mucosa epithelium in CRSwNP, including increased permeability, impaired mucus clearance, and reduced antimicrobial protein secretion. Additionally, type 2 immune responses (Th2) are active, characterized by eosinophil infiltration, elevated levels of inflammatory mediators such as IL-5 and IL-13, along with activation of mast cells, basal cells, and innate lymphoid cells. B and T cells are locally activated in nasal polyps, secreting immunoglobulins and autoantibodies. Some CRSwNP patients also have specific IgE against bacterial superantigens, such as Staphylococcus aureus enterotoxins, suggesting that pathogens may trigger or exacerbate the inflammatory process. Moreover, ethnic differences are present, with non-eosinophilic inflammation being more common in Asian patients.

Symptoms

The typical symptoms of CRSwNP include anterior or posterior nasal discharge, nasal congestion, anosmia, and facial pressure or pain, persisting for more than 12 weeks. Nasal congestion and anosmia are more pronounced in CRSwNP, whereas facial pain is more commonly seen in CRSsNP. Patients with CRSwNP often present with more severe sinus involvement on imaging and are more prone to recurrence, requiring multiple surgeries. Severe cases may be associated with Eustachian tube dysfunction, ear pain, worsened nasal obstruction, and loss of taste.

Cause

The exact cause of CRSwNP remains unclear, but current research proposes a multifactorial model. Genetic predisposition is considered one of the key factors, with an increased risk of the disease in first-degree relatives, though no specific gene mutations have been identified. Environmental factors, such as air pollution or urbanization, may contribute to the increase of eosinophilic polyps in Asian populations. Comorbidities like asthma, allergic rhinitis, gastroesophageal reflux, and sleep apnea are closely associated with CRSwNP. Approximately 26-48% of CRSwNP patients also have asthma, while 7% of asthma patients suffer from CRSwNP. Additionally, about 10% of patients have aspirin-exacerbated respiratory disease (AERD), which is more severe with a higher postoperative recurrence rate. Microbial colonization, especially Staphylococcus aureus infection, and the enterotoxins it produces can induce local immune responses, potentially acting as significant pathogenic factors.

Source: Frost & Sullivan Analysis

Market drivers and future trends of Allergic Disease Drug Market

Rising prevalence driven by urbanization, natural environmental changes, and lifestyle shifts	• In the process of urbanization, the intensification of urban air pollution; the prolonged pollen transmission season caused by global warming resulting from natural environmental changes; and other factors such as lifestyle shift toward indoor confinement, frequent air conditioner use, and a rising pet ownership rate have collectively increased exposure to allergens, directly fueling the continuous upward trend in the overall prevalence of allergic diseases.
Increased awareness of diagnosis and treatment of allergic diseases among patients	• In the past, patients often mistook allergic symptoms for those of common illnesses, leading to neglect. Today, with the popularization of health education, patients' understanding of allergic diseases has deepened, and their willingness to seek medical treatment proactively has significantly increased. Meanwhile, the widespread availability of allergen detection equipment in primary medical institutions has enabled the diagnosis of more mild and occult allergic patients, substantially improving the disease diagnosis rate and prompting more patients with allergic diseases to receive treatment.
Growing number of patients with moderate-to-severe allergic diseases	• Affected by factors such as long-term continuous allergen exposure and insufficient early intervention, some patients with mild allergic diseases have gradually progressed to moderate-to-severe conditions. These patients show limited response to traditional drugs and have an urgent demand for long-lasting, precise treatment options such as biologics. The increase in the number of moderate-to-severe allergic disease patients has driven the growth in demand for high-value drugs like biologics, which in turn has boosted the growth of the allergic disease drug market.

Source: Frost & Sullivan Analysis

Future trends of Allergic Disease Drug Market

Sustained growth of the allergic disease drug market	• Driven by factors such as environmental changes and lifestyle shifts, the global number of patients with allergic diseases keeps rising. Meanwhile, with the advancement and penetration of clinical diagnostic technologies, an increasing number of patients are diagnosed and receive treatment. As the understanding of allergic diseases deepens, patients' treatment demands have shifted from symptom relief to long-term control. These factors collectively fuel the continuous growth of the allergic disease drug market.
Rising proportion of biologics in the treatment of allergic diseases	• The share of biologics in the allergic disease drug market is rapidly increasing. Their key advantages are that compared with traditional drugs, it features precise targeting, long - lasting efficacy, and fewer side effects. It can especially meet the unmet clinical needs of moderate-to-severe patients. Furthermore, in recent years, biological drugs targeting IgE, IL-4Rα, and other targets have demonstrated excellent long-term control effects in clinical practice. Meanwhile, the research and development of biological drugs targeting new targets such as IL-13 and TSLP in the subsequent pipeline are advancing rapidly, contributing to the growing proportion of biologics in the allergic disease market.
Diversification of therapeutic targets for allergic diseases	• With the deepening research on the immune mechanism of allergic reactions, the R&D of therapeutic targets for allergic disease drugs is evolving from the traditional single dimension to a diversified direction. While focusing on targets of IgE-mediated type I hypersensitivity, new targets associated with allergic inflammatory pathways, such as IL-4, IL-13, and TSLP, have been gradually validated.A succession of multi-targets drugs have been approved for the treatment of allergic diseases, enriching clinical treatment options and more accurately meeting differentiated clinical needs.

Source: Frost & Sullivan Analysis

Challenges and threats of Allergic Disease Drug Market

Low penetration of biologics in the allergic disease drug market	• Due to high R&D costs, biologics are priced significantly higher than traditional antihistamines and glucocorticoids. Even though some drugs are covered by medical insurance, patients' out-of-pocket expenses remain high, especially in lower-tier market and economically underdeveloped regions where patients have limited payment capacity. Additionally, most biologics require cold-chain storage and injection administration, which imposes certain requirements on the storage conditions and the medical staffs' operational capabilities in primary medical institutions. These factors collectively restrict their widespread clinical adoption.
Diversification of biologic targets intensifying market competition	• As the clinical value of popular targets such as IgE, IL-4Rα, TSLP, and IL-33 has been validated, domestic and foreign pharmaceutical companies have scrambled to deploy R&D efforts on the same or similar targets. This has led to a cluster of biologics with similar mechanisms of action entering the late clinical stage or marketing stage, exacerbating homogeneous market competition. Furthermore, to seize market share, some enterprises may launch competitions through price adjustments, which further compresses the overall profit margin of the industry and places higher demands on enterprises' R&D differentiation capabilities.
Low accessibility of allergen detection and diagnostic technologies	• Current allergen detection technologies are plagued by high equipment costs and complex operational procedures. Primary medical institutions and hospitals in remote areas often lack the procurement capacity and professional inspectors. Consequently, the basic skin prick test remains the primary diagnostic method in most regions, which hinders the accurate identification of complex and uncommon allergens. This insufficient accessibility of diagnostic technologies may not only lead to misdiagnoses and missed diagnoses but also restrict the clinical application of targeted drugs, indirectly hindering market growth.

Source: Frost & Sullivan Analysis

Entry barriers of Allergic Disease Drug Market

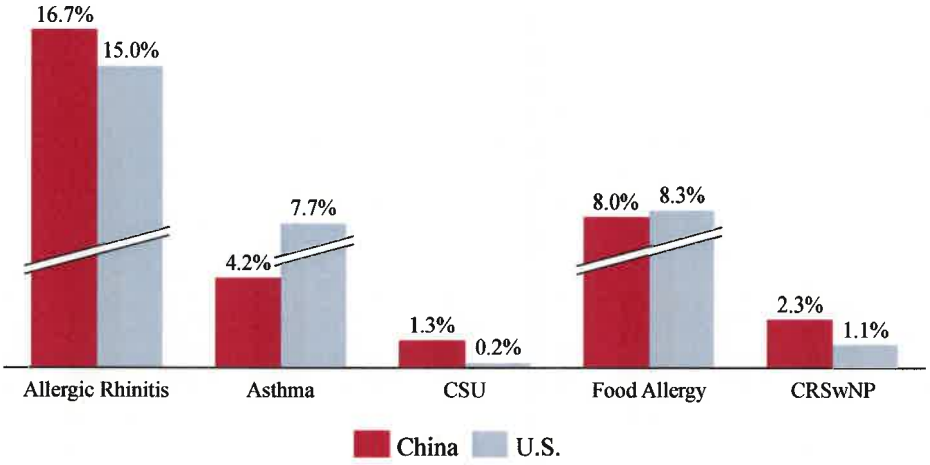
Significant Target Development Challenges	• Core target development faces high barriers. Take IgE as an example: its complex molecular structure (conformational changes affect receptor binding) and allergic reactions involving multi-cell pathways make single targets insufficient to cover all mechanisms, resulting in very few approved drugs globally over 20 years.
Stringent Drug Performance Requirements	• The chronic nature of allergic diseases demands high medication adherence, requiring drugs to be both long-acting and highly active. Traditional drugs are gradually replaced by biologics due to side effects and limited efficacy, but the high cost and injectable administration of biologics remain challenging.
High Technical and Platform Barriers	• The industry is shifting to multi-target drugs, which need to block multiple inflammatory pathways simultaneously and rely on cutting-edge technologies like multi-omics integration and multispecific antibodies. Small and medium-sized enterprises struggle to independently develop such drugs, resulting in significant technical barriers.

Source: Frost & Sullivan Analysis

Comparison of the prevalence of allergic diseases between China and the United States

- Both China and the United States have a large number of patients with allergic diseases, with a high prevalence of allergic rhinitis, asthma, and food allergies.

Comparison of the prevalence of allergic diseases between China and the United States



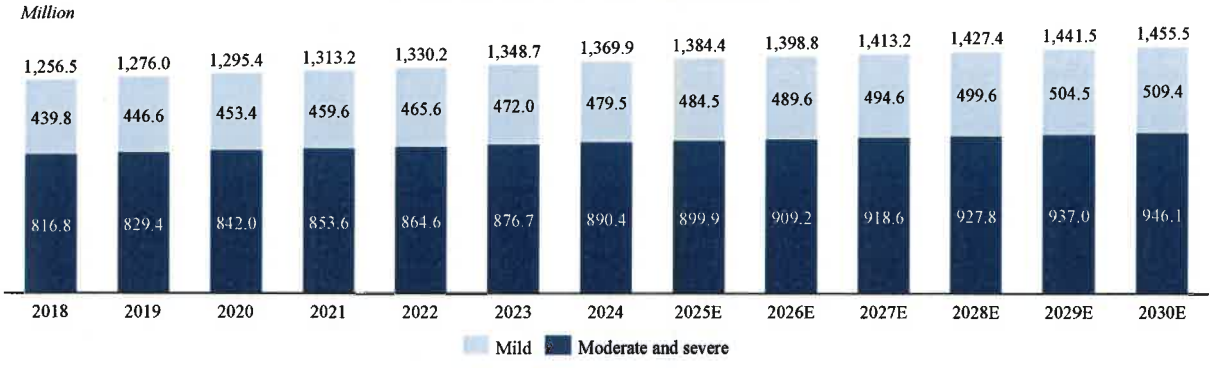
Source: Frost & Sullivan analysis

Global Prevalence of Allergic Rhinitis, 2018-2030E

- There are a large number of allergic rhinitis patients around the world, and its prevalence has grown from 13 billion in 2018 to 1.4 billion in 2024, with a CAGR of 1.5%. With the increasing prevalence of allergic rhinitis, the number of allergic rhinitis patients around the world is expected to reach 1.5 billion in 2030 at a CAGR of 1.0%.

Global Prevalence of Allergic Rhinitis, 2018-2030E

Period	CAGR
2018-2024	1.5%
2025E-2030E	1.0%



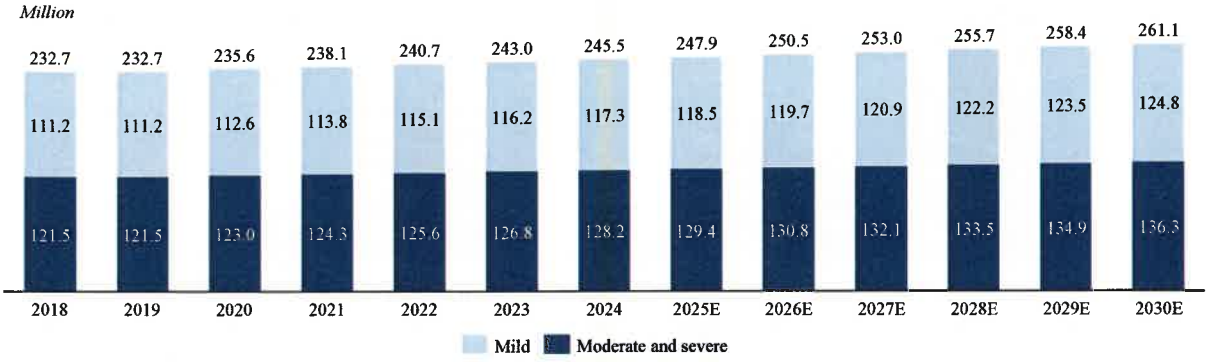
Source: Worldwide prevalence of rhinitis in adults: A review of definitions and temporal evolution, Frost & Sullivan analysis

Prevalence of Allergic Rhinitis in China, 2018-2030E

- There are a large number of allergic rhinitis patients in China, and its prevalence has grown from 232.7 million in 2018 to 245.5 million in 2024, with a CAGR of 0.9%. The number of allergic rhinitis patients in China is expected to reach 261.1 million in 2030 at a CAGR of 1.0%.

Prevalence of Allergic Rhinitis in China, 2018-2030E

Period	CAGR
2018-2024	0.9%
2025E-2030E	1.0%



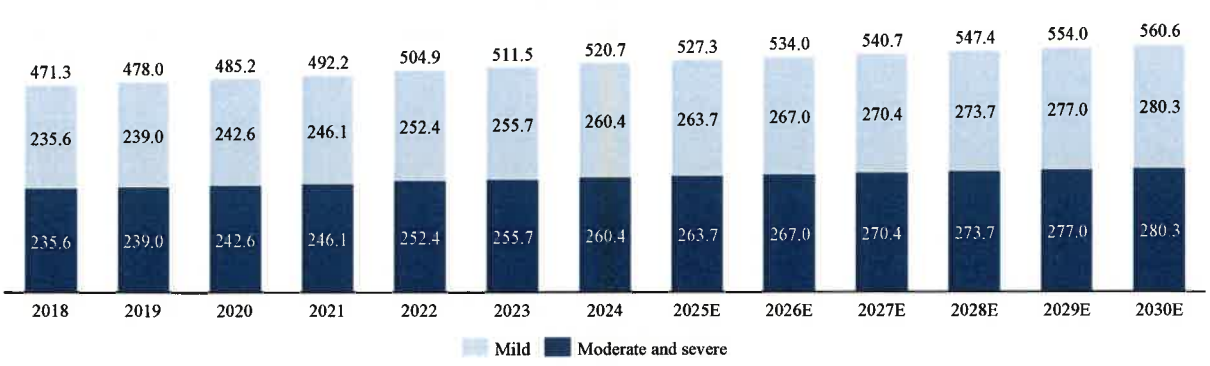
Source: Increasing Prevalence of Allergic Rhinitis in China, Frost & Sullivan analysis

Global Prevalence of Allergic Asthma, 2018-2030E

- There are a large number of allergic asthma patients around the world, and its prevalence has grown from 471.3 million in 2018 to 527.3 million in 2024, with a CAGR of 1.7%. With the increasing prevalence of allergic asthma, the number of allergic asthma patients around the world is expected to reach 567.3 million in 2030 at a CAGR of 1.2%.

Global Prevalence of Allergic Asthma, 2018-2030E

Period	CAGR
2018-2024	1.7%
2025E-2030E	1.2%



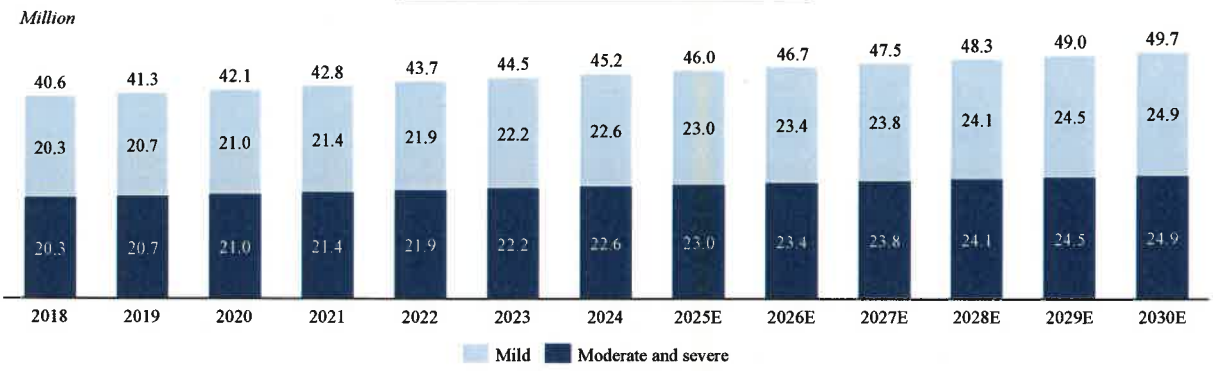
Source: Intermittent and mild persistent asthma: how therapy has changed, Frost & Sullivan analysis

Prevalence of Allergic Asthma in China, 2018-2030E

- There are a large number of allergic asthma patients in China, and its prevalence has grown from 40.6 million in 2018 to 45.2 million in 2024, with a CAGR of 1.8%. The number of allergic asthma patients in China is expected to reach 49.7 million in 2030 at a CAGR of 1.6%.

Prevalence of Allergic Asthma in China, 2018-2030E

Period	CAGR
2018-2024	1.8%
2025E-2030E	1.6%



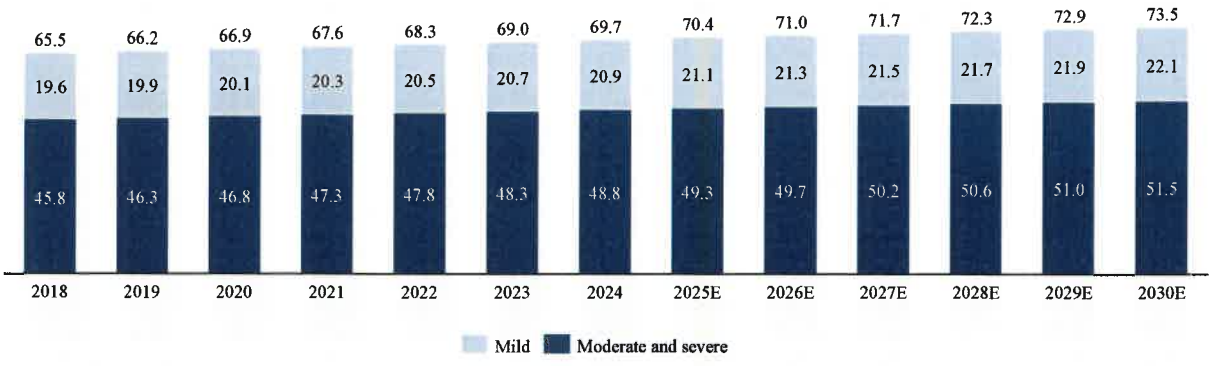
Source: Prevalence, risk factors, and management of asthma in China: a national cross-sectional study, Frost & Sullivan analysis

Global Prevalence of Chronic Spontaneous Urticaria, 2018-2030E

- There are a large number of chronic spontaneous urticaria patients around the world, and its prevalence has grown from 65.5 million in 2018 to 69.7 million in 2024, with a CAGR of 1.1%. With the increasing prevalence of chronic spontaneous urticaria, the number of chronic spontaneous urticaria patients around the world is expected to reach 73.5 million in 2030 at a CAGR of 0.9%.

Global Prevalence of Chronic Spontaneous Urticaria, 2018-2030E

Period	CAGR
2018-2024	1.1%
2025E-2030E	0.9%



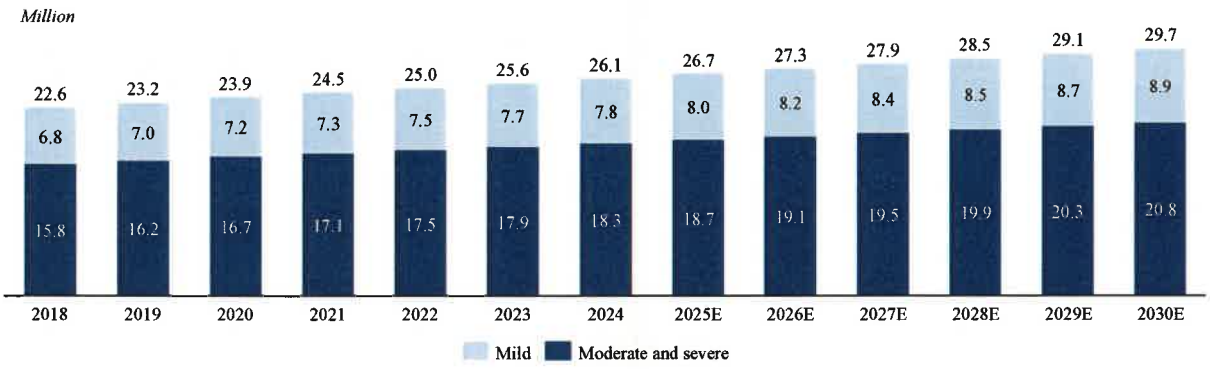
Source: Prevalence of chronic urticaria in children and adults across the globe: Systematic review with meta-analysis, Frost & Sullivan analysis

Prevalence of Chronic Spontaneous Urticaria in China, 2018-2030E

- There are a large number of chronic spontaneous urticaria patients in China, and its prevalence has grown from 22.6 million in 2018 to 26.1 million in 2024, with a CAGR of 2.5%. The number of chronic spontaneous urticaria patients in China is expected to reach 29.7 million in 2030 at a CAGR of 2.1%.

Prevalence of Chronic Spontaneous Urticaria in China, 2018-2030E

Period	CAGR
2018-2024	2.5%
2025E-2030E	2.1%



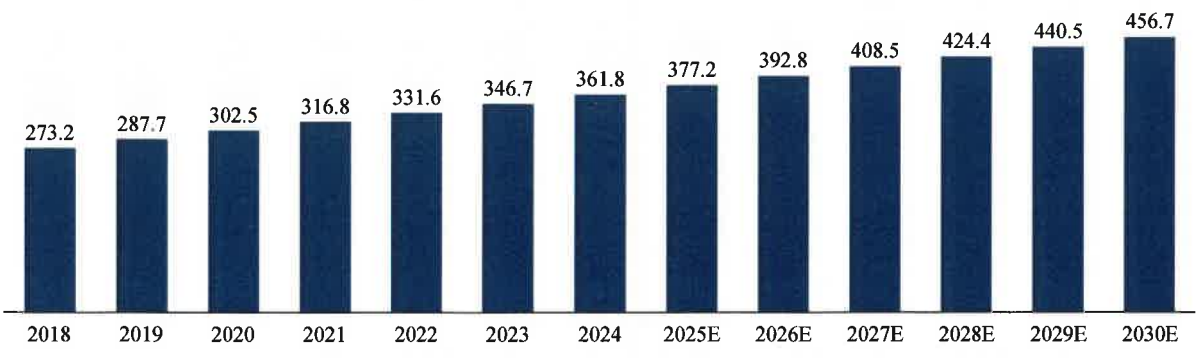
Source: Epidemiology of urticaria in China: a population-based study, Frost & Sullivan analysis

Global Prevalence of Food Allergy, 2018-2030E

- There are a large number of food allergy patients around the world, and its prevalence has grown from 273.2 million in 2018 to 361.8 million in 2024, with a CAGR of 4.8%. With the increasing prevalence of food allergy, the number of food allergy patients around the world is expected to reach 456.7 million in 2030 at a CAGR of 3.9%.

Global Prevalence of Food Allergy, 2018-2030E

Period	CAGR
2018-2024	4.8%
2025E-2030E	3.9%



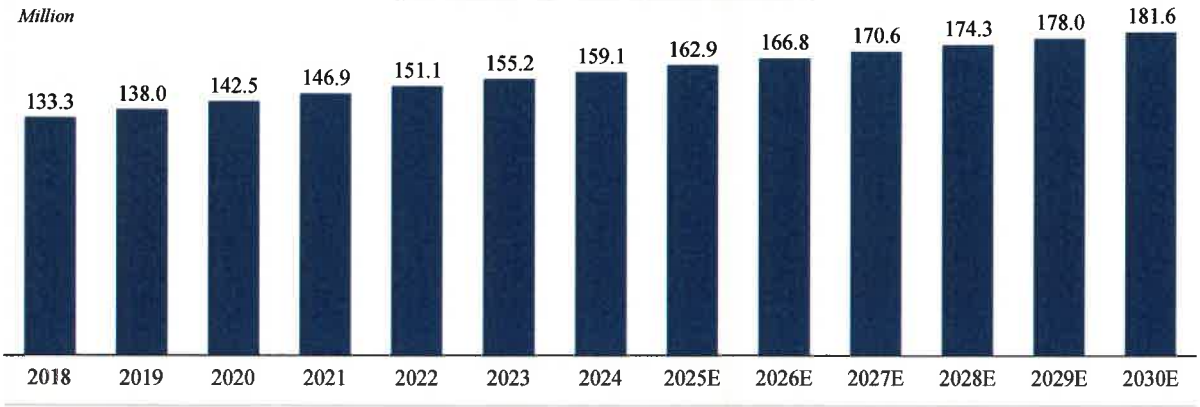
Source: Prevalence and Influencing Factors of Food Allergy in Global Context: A Meta-Analysis, Frost & Sullivan analysis

Prevalence of Food Allergy in China, 2018-2030E

- There are a large number of food allergy patients in China, and its prevalence has grown from 133.3 million in 2018 to 159.1 million in 2024, with a CAGR of 3.0%. The number of food allergy patients in China is expected to reach 181.6 million in 2030 at a CAGR of 2.2%.

Prevalence of Food Allergy in China, 2018-2030E

Period	CAGR
2018-2024	3.0%
2025E-2030E	2.2%



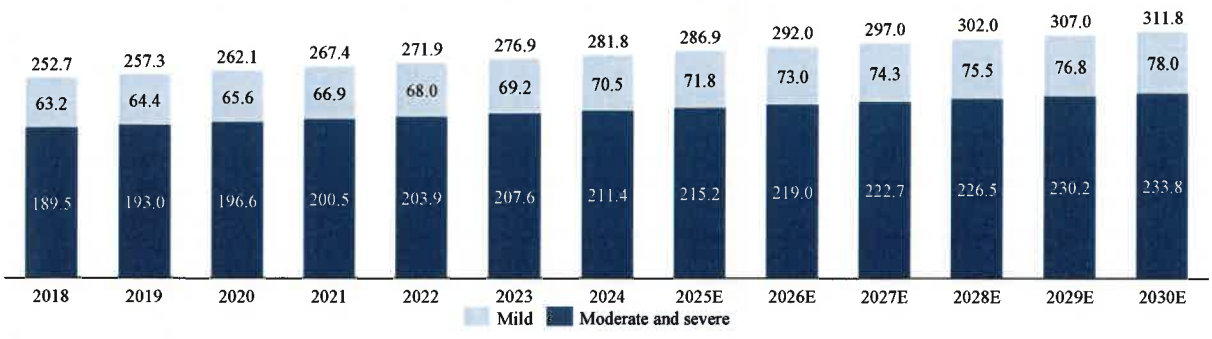
Source: Frost & Sullivan analysis

Global Prevalence of CRSwNP, 2018-2030E

- There are a large number of CRSwNP patients around the world, and its prevalence has grown from 252.7 million in 2018 to 281.8 million in 2024, with a CAGR of 1.8%. With the increasing prevalence of food allergy, the number of CRSwNP patients around the world is expected to reach 311.7 million in 2030 at a CAGR of 1.7%.

Global Prevalence of CRSwNP, 2018-2030E

Period	CAGR
2018-2024	1.8%
2025E-2030E	1.7%



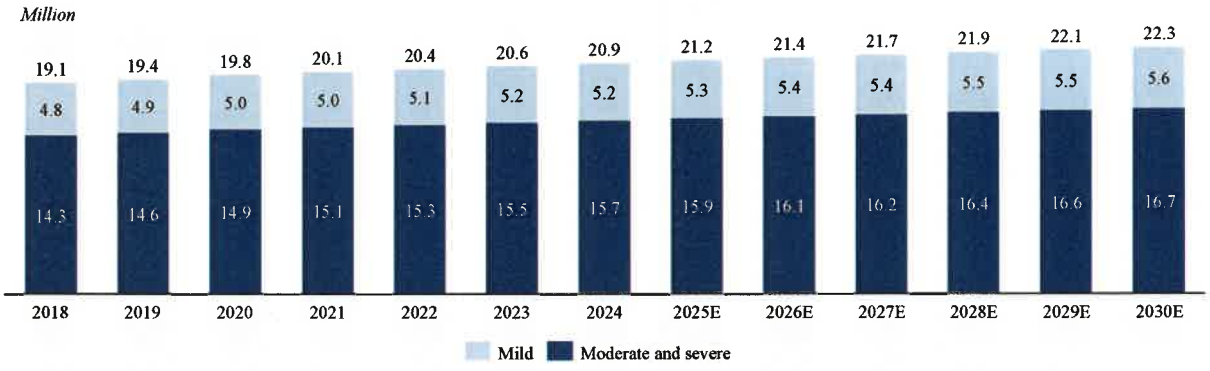
Source: Prevalence of chronic rhinosinusitis without/with nasal polyps according to severity in Spain, Frost & Sullivan analysis

Prevalence of CRSwNP in China, 2018-2030E

- There are a large number of CRSwNP patients in China, and its prevalence has grown from 19.1 million in 2018 to 20.9 million in 2024, with a CAGR of 1.5%. The number of CRSwNP patients in China is expected to reach 22.3 million in 2030 at a CAGR of 1.1%.

Prevalence of CRSwNP in China, 2018-2030E

Period	CAGR
2018-2024	1.5%
2025E-2030E	1.1%



Source: Clinical treatment options oriented to the endotype of chronic rhinosinusitis, Frost & Sullivan analysis

Development History of Allergic Disease Drugs

- The main allergic disease drugs include antihistamines, mast cell stabilizers, leukotriene receptor antagonists, and anti-IgE antibodies. With the advancement of biopharmaceutical technology, precision medicine and personalized treatment have become the development trend, and new types of drugs will continue to emerge, providing more choices for patients with allergic diseases.

Development History of Allergic Disease Drugs

Stage I: Early symptomatic treatment	Stage II: Multi-mediator regulation and optimization	Stage III: Targeted immunomodulation
Drug characteristic: Targeting the most well-defined mediator and the key link in the allergic reaction, it focuses on symptomatic relief, with a single mechanism of action and more pronounced side effects.	**Drug characteristic:** Expanding coverage of allergy mediators and optimizing drug safety, second-generation antihistamines have become first-line agents for many allergic diseases.	**Drug characteristic:** Drugs are designed to target the deeper immune mechanisms of allergic reactions (e.g., IgE, Th2 type cytokines), enabling precisely targeted therapies.
Representative drugs: • **First-generation antihistamines:** Block histamine H1 receptors, relieve itching, redness and swelling, but have sedative side effects. • **Mast cell stabilizer:** Stabilizes mast cell membranes and reduces the release of mediators such as histamine for allergy prevention.	**Representative drugs:** • **Second-generation antihistamines:** Highly selective blockade of peripheral H1 receptors, no sedative side effects, longer-lasting effects. • **leukotriene receptor antagonists:** Block leukotriene-mediated bronchospasm, mucosal edema and compensate for histamine antagonists.	**Representative drugs:** • **Anti-IgE antibody:**. Blocks IgE binding to mast cells and inhibits allergy initiation. • **Anti-cytokine antibodies:** Block IL-4/IL-13 etc., Inhibit th2 type inflammation. • **Anti-TSLP antibody:** Blocks TSLP-mediated dendritic cell activation and inhibits the initiation of th2-type immune responses from upstream.

Source: Frost & Sullivan analysis

Main Treatment for Allergic Rhinitis

- The main treatment options for allergic rhinitis include medication, immunotherapy, surgery and nasal irrigation. Drugs including antihistamines, glucocorticoids, leukotriene receptor antagonists, mast cell stabilizers, etc. are available for patients with different degrees of the disease.

Main Treatment for Allergic Rhinitis

Treatment Category		Mechanism	Advantages	Disadvantages
Medication		Antihistamine blocks H1 receptors to reduce allergic symptoms. Glucocorticoid inhibits inflammation by blocking cytokine release. Leukotriene receptor antagonist blocks leukotriene activity to reduce nasal inflammation.	Antihistamines act quickly to alleviate sneezing and itching; glucocorticoids serve as first-line anti-inflammatory agents with strong local effects and minimal systemic side effects; leukotriene receptor antagonists reduce both nasal and lower respiratory symptoms; mast cell stabilizers offer high safety with fewer adverse effects; and decongestants deliver rapid relief from nasal congestion.	Long-term use may cause side effects, such as mucosal dryness with glucocorticoids and rebound congestion with decongestants. In moderate-to-severe cases or those with type 2 inflammation, drugs alone may be insufficient.
Allergen immunotherapy (AIT)		Induces immune tolerance by gradual allergen exposure.	Only treatment that may change disease progression.	Long duration of therapy; may cause allergic reactions.
Biological agent medication		Dupilumab blocks IL-4/IL-13 signaling to inhibit type 2 inflammatory response. Mepolizumab and Reslizumab neutralize IL-5 to reduce eosinophils.Omalizumab blocks IgE from binding to its receptor, suppressing allergic reactions.Tezepelumab blocks TSLP activity, suppresses Th2 inflammation.	Mepolizumab, Reslizumab and Omalizumab require intravenous or subcutaneous administration and may cause injection site reactions, hypersensitivity responses, or, in rare cases, anaphylaxis. Mepolizumab and Reslizumabhave been associated with mild upper respiratory tract infections, allergic reactions, and potential malignancy risks.	Mepolizumab, Reslizumab, Omalizumab, and Tezepelumabrequire intravenous or subcutaneous administration and may cause injection site reactions, hypersensitivity responses, or, in rare cases, anaphylaxis. Mepolizumab and Reslizumabhave been associated with mild upper respiratory tract infections, allergic reactions, and potential malignancy risks.
Surgery	**Inferior turbinoplasty**	Reduces turbinate size to improve nasal airflow.	Improves ventilation and reduces obstruction.	Invasive; requires careful risk assessment.
	Pterygotomy	Cuts pterygoid nerve to reduce nasal reflexes.	Reduces nasal hyperreactivity in some patients.	May cause nerve damage or complications.
Nasal irrigation		Flushes out allergens and inflammatory secretions.	Safe, simple, long-term usability; improves local nasal environment.	Improper use may lead to ear infections or mucosal irritation.

Source: Frost & Sullivan analysis

Main Treatment for Chronic Spontaneous Urticaria

- The main treatment option for chronic spontaneous urticaria is medication, with antihistamines being the first line of treatment. For patients whose symptoms are not effectively controlled with high-dose antihistamines, treatment with omalizumab, a biologic agent, can be tried.

Main treatment for chronic spontaneous urticaria

Treatment category	Mechanism	Advantages	Disadvantages
First-generation Antihistamine	By competing with histamine for H1 receptors and preventing histamine from binding to H1 receptors, it inhibits small vessel dilation, reduces vascular permeability, decreases exudation, and attenuates allergic reactions.	They have strong antihistamine effects, rapidly relieve symptoms like itching and wheals, cause minimal central nervous system inhibition, offer long-lasting action, and typically require only once-daily dosing. They are considered first-line therapy for CSU.	First-generation antihistamines have strong central inhibitory effects and may cause drowsiness, dizziness, and fatigue. Due to their short duration of action, they are not recommended for long-term use.The efficacy of second-generation antihistamines is limited and symptoms persist for some patients.
Second-generation Antihistamine	Selectively acts on H1 receptor, blocks the binding of histamine to H1 receptor, inhibits the release of histamine and other inflammatory mediators from mast cells, and exerts anti-allergic effects.		
Glucocorticoid	It has powerful anti-inflammatory, anti-allergic and immunosuppressive effects. It can inhibit the aggregation and activation of inflammatory cells, reduce the production and release of inflammatory mediators, stabilize mast cells and lysosomal membranes, reduce vascular permeability, and reduce tissue congestion and edema.	It has a rapid onset of action and provide effective anti-inflammatory and anti-allergic effects. They are suitable for patients with severe conditions when antihistamines are ineffective.	Long-term use of glucocorticoids may lead to adverse reactions and requires gradual tapering to prevent withdrawal symptoms.
Immunosuppressant	It regulates the body's immune function by inhibiting the proliferation and activation of immune cells, cytokine production and transmission, and suppresses the immune response associated with the pathogenesis of CSU.	It can be effective for patients who do not respond to other treatments by suppressing immune-related mechanisms involved in CSU.	Immunosuppressants require close monitoring of blood, liver, and kidney function due to the risk of serious adverse effects. Dosage must be adjusted according to the patient's condition.
Biological agent Medication	Recombinant humanized anti-IgE monoclonal antibody that specifically binds to free IgE and prevents IgE from binding to IgE receptors on the surface of mast cells and basophils, thereby inhibiting mast cell and basophil activation and degranulation, and reducing the release of inflammatory mediators.	Biological agents are characterized by strong targeting, high efficacy, and good safety. They significantly improve quality of life in patients with refractory CSU. Emerging therapies targeting TSLP may reduce eosinophils, IgE, and type 2 cytokines, offering potential benefits for patients who are unresponsive to conventional therapy.	Most of them require subcutaneous injection, which may limit accessibility for some patients.

Source: Frost & Sullivan analysis

Main Treatment for Allergic Asthma

- The main treatment options for allergic asthma include small molecule drug medication, allergen-specific immunotherapy and biological agent medication. The biological agents currently used clinically for the treatment of allergic diseases mainly include omalizumab and dupilumab. Patients with moderate-to-severe allergic asthma account for approximately [50]% of all allergic asthma patients.

Main treatment for allergic asthma

Treatment category		Mechanism	Advantages	Disadvantages
chemical drug medication	**Glucocorticoid**	It binds to intracellular receptors to suppress inflammation and enhance β_2 receptor responsiveness, reducing airway inflammation.	chemical drugs are the cornerstone of treatment and include glucocorticoids, β_2-agonists, leukotriene receptor antagonists, theophyllines, and anticholinergics. Glucocorticoids offer strong local anti-inflammatory effects with fewer systemic side effects .Many are orally administered, well-tolerated (e.g., leukotriene receptor antagonists), and can be used alone or in combination. These drugs are generally fast-acting and convenient, often combined to enhance outcomes.	Most medications require long-term and regular use to maintain their effectiveness. For example, prolonged use of theophylline can lead to tolerance and reduced efficacy. Monotherapy may exacerbate airway inflammation and increase the risk of disease worsening. The use of anticholinergic drugs necessitates careful monitoring of blood drug concentrations and carries a risk of side effects.
	β_2 receptor agonists	Binds to β_2 receptors on airway smooth muscle, activates adenylate cyclase, increases intracellular cyclic adenosine monophosphate (cAMP) content, and reduces free calcium ions, thus relaxing airway smooth muscle and relieving asthma symptoms.		
	Leukotriene receptor antagonist	By selectively inhibiting cysteinyl leukotriene receptors and blocking leukotriene binding to the corresponding receptors, it reduces airway inflammation and airway hyperresponsiveness and relieves symptoms.		
	Theophylline	By inhibiting phosphodiesterase, it reduces the hydrolysis of cAMP, so that the intracellular cAMP content rises, and airway smooth muscle diastole; it can also stimulate the respiratory center, and enhance the contractility of respiratory muscle.		
	Anticholinergic drug	By blocking the postganglionic vagal pathway, it reduces vagal tone and diastoles the bronchi, and also reduces sputum secretion.		
Allergen immunotherapy		Modulating the body's immune response, shifting the Th2 type immune response toward the Th1 type immune response, decreasing the production of IgE, increasing the number and function of regulatory T-cells, etc., thus reducing airway hyperreactivity to allergens.	Allergen immunotherapy is currently the only treatment that may alter the natural course of allergic diseases and is suitable for patients with well-defined allergens who do not respond to conventional drug therapy.	The treatment period is long and it is only applicable to patients with confirmed allergens.
Biological agent medication		Omalizumab blocks IgE binding to mast cells, reducing the release of inflammatory mediators.Dupilumab inhibits IL-4 and IL-13 signaling to suppress Th2-driven inflammation. Anti-TSLP therapy blocks TSLP signaling to reduce Th2 cytokines and restore immune balance.	Biological agents target specific immune pathways and are highly effective for patients with severe allergic asthma. Targeting TSLP may reduce type 2 inflammation, lower cytokine release, and provide symptom relief in severe asthma.	These treatments are typically reserved for patients with severe disease and specific biomarkers.

Source: Frost & Sullivan analysis

Main Treatment for Food Allergy

- The main treatments for food allergies include allergen avoidance, medication, and allergen-specific immunotherapy. For patients with varying degrees of food allergy, medication may include antihistamines, glucocorticoids or adrenaline.

Main treatment for food allergy

Treatment Category	Mechanism of Action	Advantages	Disadvantages
Avoidance of allergens	Food allergy is an abnormal immune response of the body to certain components of food. Avoiding exposure to allergens prevents the immune system from being activated at all, thus avoiding allergic symptoms.	This approach is foundational and is particularly effective in managing mild food allergy cases.	It is difficult for patients to consistently avoid allergen exposure in daily life.
chemical drug medication	Antihistamines block H1 receptors, thereby alleviating itching, redness, and other histamine-mediated symptoms. Corticosteroids suppress immune cell activity and cytokine release, reducing inflammation associated with allergic reactions.Adrenaline activates α- and β-receptors to increase blood pressure, dilate bronchi, and enhance cardiac output, rapidly reversing circulatory and respiratory failure in anaphylactic shock.	Second-generation antihistamines, are effective for mild to moderate cases with rapid onset and fewer side effects. Glucocorticoids help control inflammation in moderate to severe allergies, while epinephrine is crucial for reversing life-threatening symptoms like anaphylactic shock.	Long-term use of glucocorticoids may lead to significant adverse effects. Epinephrine must be administered immediately upon the onset of symptoms and may require patient training or timely access.
Allergen Immunotherapy (AIT)	Through long-term, low-dose exposure to allergens, the body's immune system is regulated to shift the Th2 cell response toward the Th1 cell response, reduce IgE production, and increase the number and function of regulatory T cells, thereby reducing or eliminating allergic reactions.	It offers the potential for long-term remission or clinical cure of food allergies.	This therapy requires a prolonged treatment course and close monitoring for potential adverse reactions.
Biological agent medication	Omalizumab blocks IgE binding to mast cells, reducing the release of inflammatory mediators.Dupilumab inhibits IL-4 and IL-13 signaling to suppress Th2-driven inflammation.	They are effective for patients who do not respond to conventional therapies and offers long-term symptom relief through subcutaneous injection.	Omalizumab may cause some serious adverse reactions, including redness, pain, or itching at the injection site.

Source: Frost & Sullivan analysis

Main Treatment for Chronic Rhinosinusitis with Nasal Polyps

- The treatment of CRSwNP includes pharmacological therapy, surgical treatment, and biologic therapies. Initially, all patients should receive first-line medical therapy, such as nasal irrigation, and the use of corticosteroids to reduce inflammation and polyp size, with surgery or biologics considered for further symptom control if necessary.

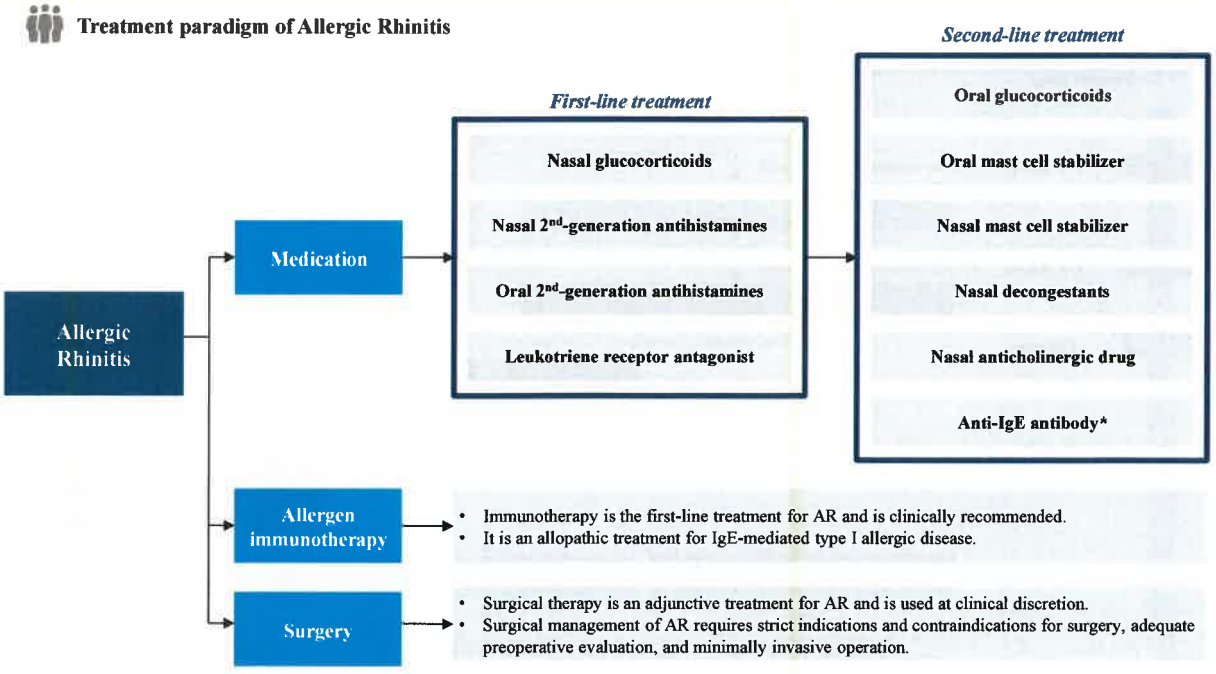
Main Treatment for Chronic Rhinosinusitis with Nasal Polyps (CRSwNP)

Treatment Strategy		Mechanism	Advantages	Disadvantages
Baseline Medical Therapy		Topical or systemic corticosteroids work by reducing mucosal inflammation through inhibition of multiple inflammatory pathways, thereby relieving nasal congestion and reducing polyp size.	Baseline medical therapy is non-invasive, well tolerated in early stages, and helps reduce inflammation and polyp size.	Long-term corticosteroid use may lead to side effects such as osteoporosis and hyperglycemia. There is a high risk of recurrence after withdrawal.
Surgical Treatment (ESS) (Endoscopic Sinus Surgery)		Endoscopic sinus surgery removes obstructive nasal polyps and restores normal sinus drainage and ventilation, facilitating the efficacy of postoperative medical therapies.	ESS offers rapid symptom relief, restores sinus drainage, and improves medication delivery.	The recurrence rate is high, especially in asthmatic patients, with about 25% requiring revision surgery.
Biological agent medication	**Dupilumab**	Blocks IL-4 and IL-13 signaling to inhibit type 2 inflammation.	Dupilumab reduces nasal polyp size, improves the sense of smell, decreases the need for corticosteroids, and enhances patients' quality of life.	Dupilumab requires long-term treatment and regular reassessment of efficacy, and not all patients respond adequately.
	Omalizumab	Binds free IgE and prevents IgE from activating mast cells and basophils.	Omalizumab is effective in patients with elevated IgE levels, helping reduce allergic inflammation and lower dependence on corticosteroids.	It may cause some serious adverse reactions, including redness, pain, or itching at the injection site.
	Mepolizumab	Targets IL-5 to reduce eosinophil survival and activity.	Mepolizumab is particularly beneficial for patients with eosinophilic CRSwNP, helping reduce polyp burden and alleviate nasal symptoms.	Its effectiveness may be limited in patients whose CRSwNP is not primarily driven by eosinophilic inflammation.
	Tezepelumab	Blocks TSLP from binding to its receptor, suppressing upstream type 2 inflammation and IL-5 release by mast cells.	Tezepelumab offers broad suppression of type 2 inflammation and may be effective in patients who do not respond to anti-IL-4 or anti-IL-5 therapies, especially in steroid-resistant or AERD cases.	It is still undergoing clinical evaluation for CRSwNP, and its long-term safety and effectiveness remain to be confirmed.

Source: Frost & Sullivan analysis

Treatment Pathway of Allergic Rhinitis

Treatment paradigm of Allergic Rhinitis



Note: Although omalizumab is not approved for use in AR, meta-analysis has shown that omalizumab has good efficacy in the treatment of severe AR and is clinically recommended.

Source: Chinese guideline for diagnosis and treatment of allergic rhinitis (2022, revision), Frost & Sullivan analysis

Treatment Pathway of Allergic Asthma

Treatment pathway of Allergic Asthma

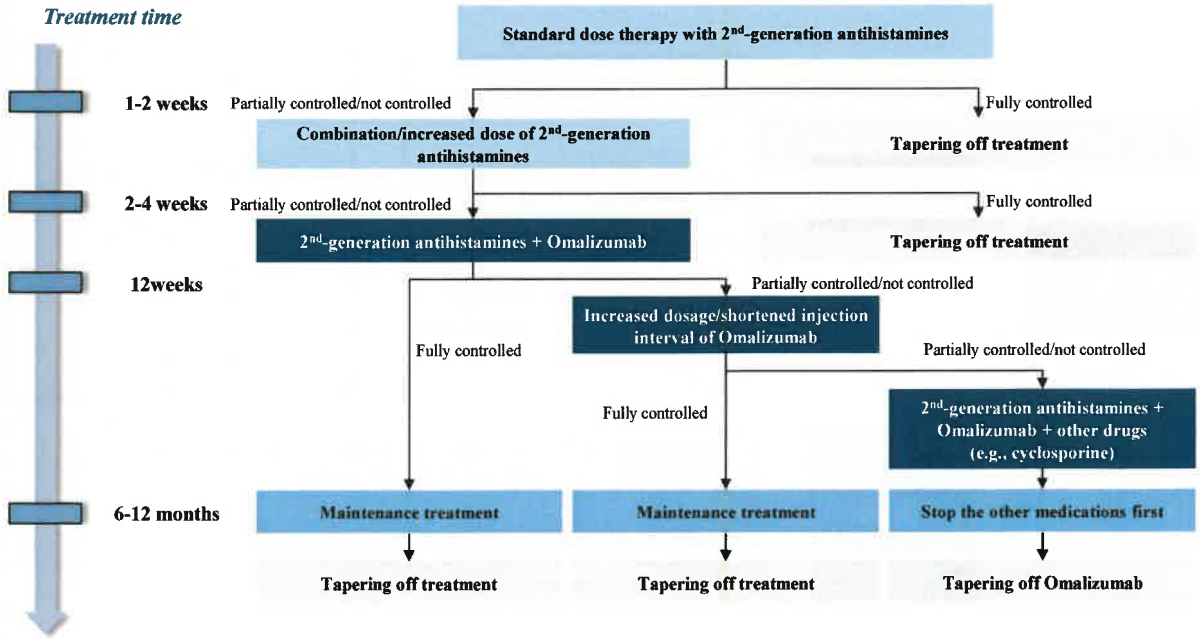
Treatment level	Preferred controller	Secondary controller	Other controller
Level 1	• Low-dose ICS-formoterol (as needed)	• SABA + low-dose ICS	• AIT
Level 2	• Low-dose ICS • Low-dose ICS-formoterol (as needed)	• LTRA • SABA + low-dose ICS	• AIT, anti-allergy drugs
Level 3	• Low-dose ICS-LABA	• Medium-dose ICS • Low-dose ICS + LTRA	• AIT, anti-IgE antibody, anti-allergy drugs
Level 4	• Medium-dose ICS-LABA	• High-dose ICS + LAMA/LTRA	• Anti-IgE antibody, anti-allergy drugs
Level 5	• High-dose ICS-LABA + anti-IgE antibody (or other biological agents)	• Add low-dose oral glucocorticoids (to minimize adverse effects))	• /

Note: ICS = Inhaled Corticosteroid; SABA = Short Acting Beta Agonist; LAMA = Long Acting Muscarine Anticholinergic; LABA = Long Acting Beta Agonist

Source: Guideline for diagnosis and treatment of allergic asthma (2019, the first edition), Frost &Sullivan analysis

Treatment Pathway of Chronic Spontaneous Urticaria

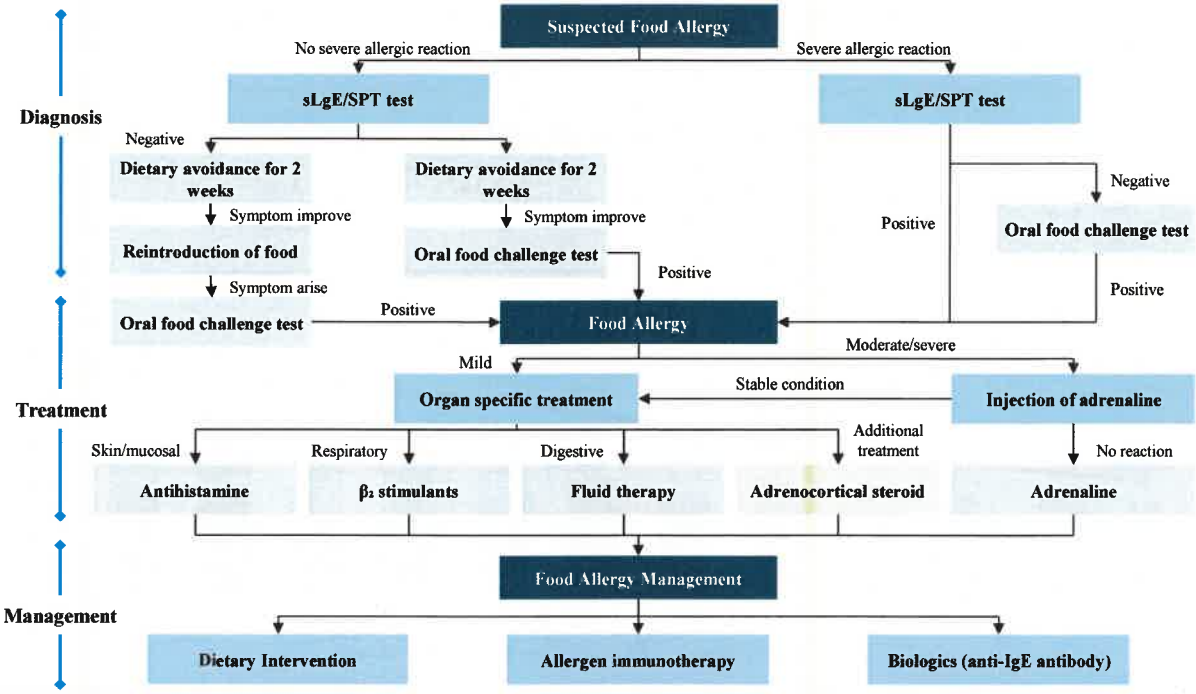
Treatment pathway of Chronic Spontaneous Urticaria



Source: Guideline for diagnosis and treatment of urticaria in China (2022), Frost&Sullivan analysis

Treatment Pathway of Food Allergy

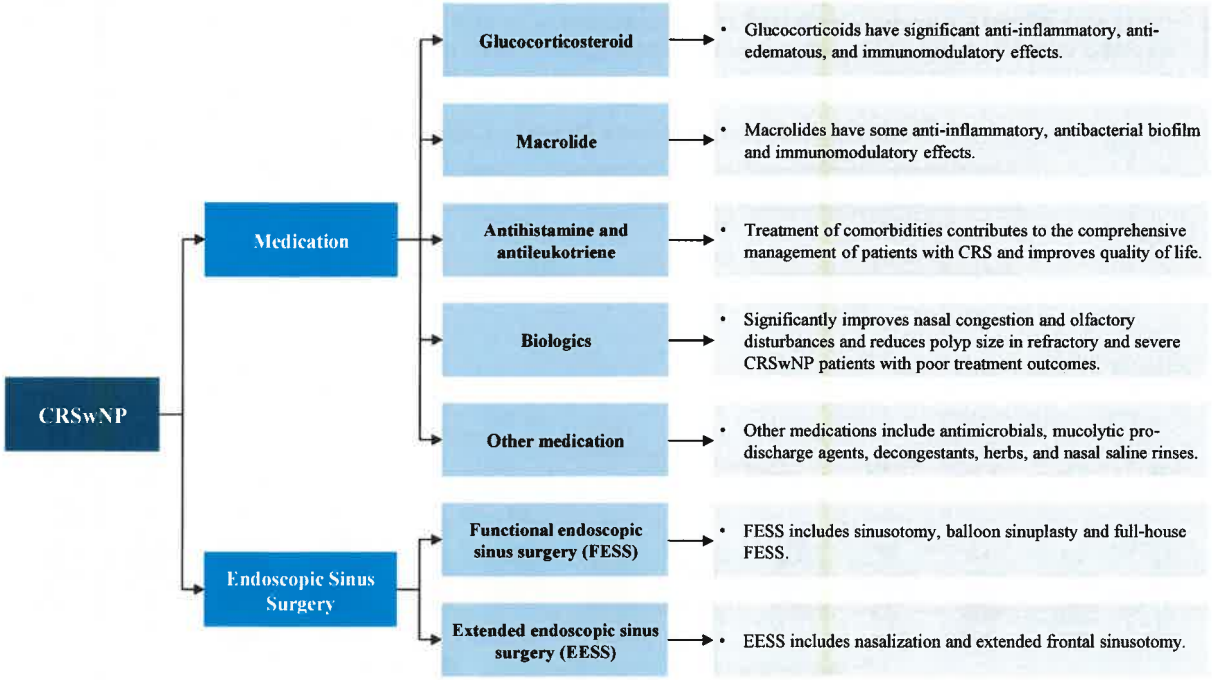
Treatment pathway of Food Allergy



Source: Evidence -based guidelines for food allergy of children in China, Frost&Sullivan analysis

Treatment Pathway of CRSwNP

Treatment paradigm of CRSwNP



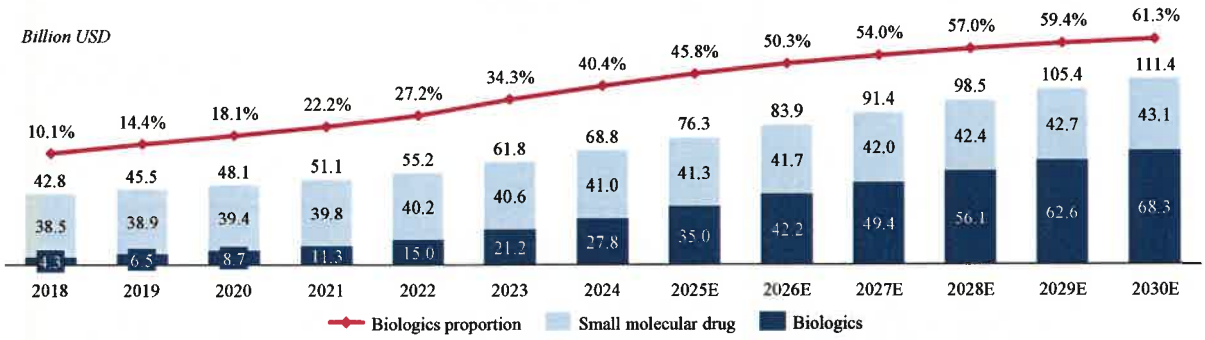
Source: Guidelines for the Diagnosis and Treatment of Chronic Rhinosinusitis(2024,version), Frost & Sullivan analysis

Global market size and forecast of allergic disease drugs, 2018-2030E

- The global allergic disease drugs market has grown from USD 42.8 billion in 2018 to USD 68.8 billion by 2024, at a CAGR of 8.2%, and is estimated to reach USD 111.4 billion by 2030, at a CAGR of 7.9% during this period. It is estimated that the global market share of biologics will increase from 40.4% in 2024 to 61.3% in 2030.

Global Market Size of Allergic Disease Drugs, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	1.0%	36.3%	8.2%
2025E-2030E	0.8%	14.3%	7.9%



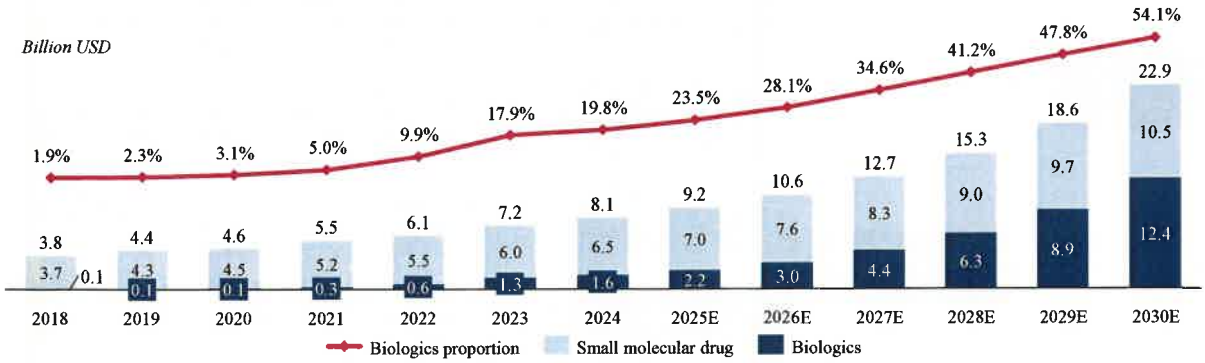
Source: Frost & Sullivan analysis

Market size and forecast of allergic disease drugs in China, 2018-2030E

- The allergic disease drugs market in China is estimated to grow from USD 3.8 billion in 2018 to USD 8.1 billion by 2024, at a CAGR of 13.3%, and is estimated to reach USD 22.9 billion by 2030, at a CAGR of 20.1% during this period. It is estimated that the market share of biologics in China will increase from 19.8% in 2024 to 54.1% in 2030.

Market Size of Allergic Disease Drugs in China, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	9.5%	67.0%	13.3%
2025E-2030E	8.4%	41.9%	20.1%



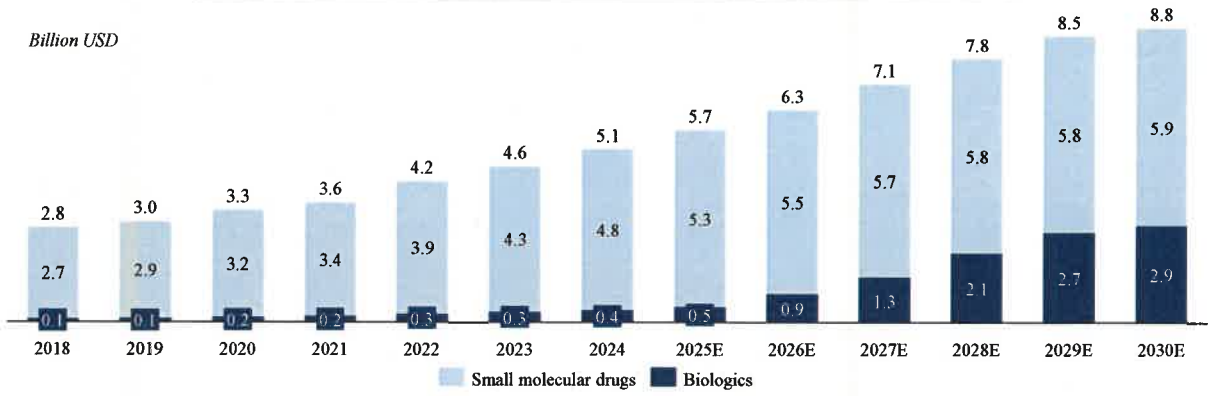
Source: Frost & Sullivan analysis

Global Market Size and Forecast of Allergic Rhinitis Drugs, 2018-2030E

- With increasing patient awareness of AR and rising treatment rates, the global AR drugs market maintains steady growth. The global market size of allergic rhinitis drugs grows from USD 2.8 billion to USD 5.1 billion from 2018 to 2024. It is expected to continue to grow to USD 8.8 billion by 2030, growing at a CAGR of 8.9% during the period.

Global Market Size of Allergic Rhinitis Drugs, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	9.9%	24.1%	10.6%
2025E-2030E	2.3%	44.5%	8.9%



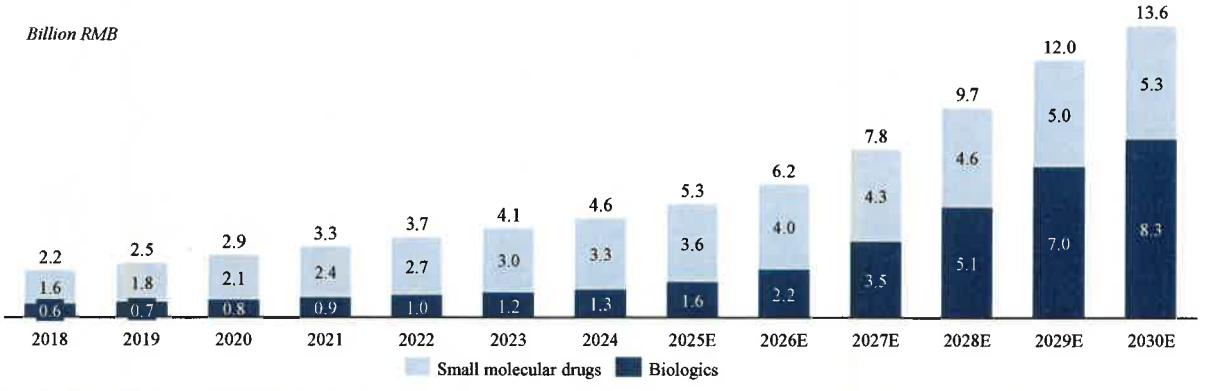
Source: Frost & Sullivan analysis

Market Size and Forecast of Allergic Rhinitis Drugs in China, 2018-2030E

- With the continuous approval of biologics for AR treatment, their penetration rate and treatment compliance are steadily increasing, making them a key therapeutic option. The market size of AR drugs in China grows from RMB 2.2 billion to RMB 4.6 billion from 2018 to 2024. It is expected to continue to grow to RMB 13.6 billion by 2030, growing at a CAGR of 20.8% during the period.

Market Size of Allergic Rhinitis Drugs in China, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	13.3%	13.6%	13.4%
2025E-2030E	7.8%	38.2%	20.8%



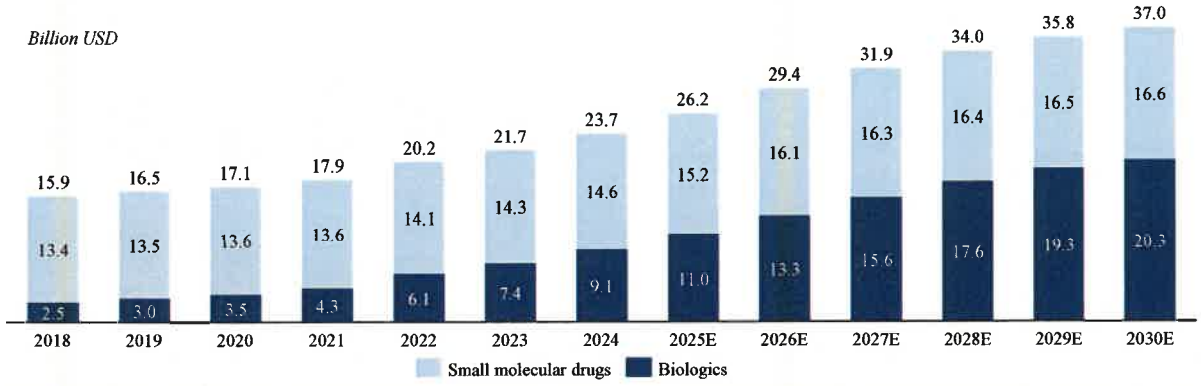
Source: Frost & Sullivan analysis

Global Market Size and Forecast of CSU Drugs, 2018-2030E

- With the increasing availability of biologic agents and growing patient awareness of CSU, treatment rates for CSU continue to rise, maintaining a steady growth trend in the global CSU drugs market. The global market size of CSU drugs grows from USD 15.9 billion to USD 23.7 billion from 2018 to 2024. It is expected to continue to grow to USD 37.0 billion by 2030, growing at a CAGR of 7.1% during the period.

Global Market Size of CSU Drugs, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	1.4%	24.1%	6.9%
2025E-2030E	1.8%	13.0%	7.1%



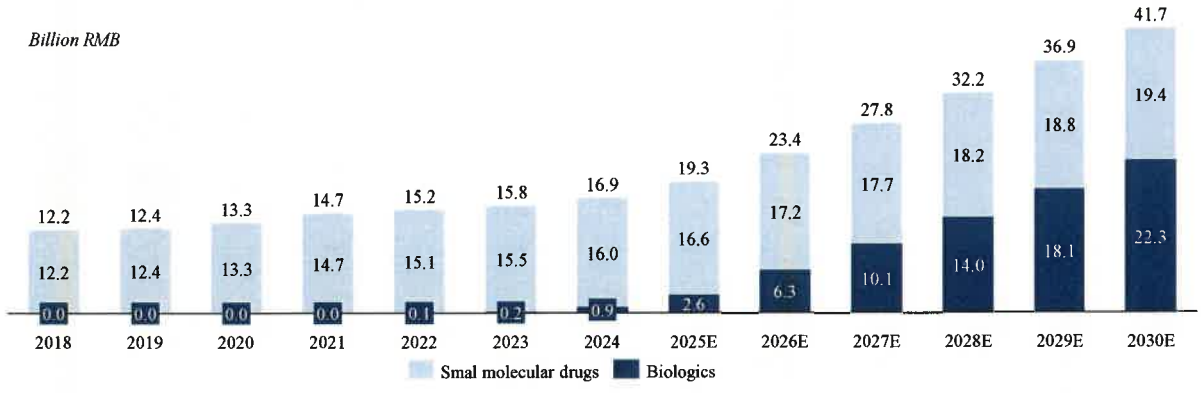
Source: Frost & Sullivan analysis

Market Size and Forecast of CSU drugs in China, 2018-2030E

- Since omalizumab was approved for treating CSU, biologics have gradually become one of the primary treatment options for the condition. With the continuous improvement in the penetration rate and treatment compliance to biologics, China's CSU drug market is poised for rapid growth. The market size of CSU drugs in China grows from RMB 12.2 billion to RMB 16.9 billion from 2018 to 2024. It is expected to continue to grow to RMB 41.7 billion by 2030, growing at a CAGR of 16.7% during the period.

Market Size of CSU Drugs in China, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	4.6%	NA	5.5%
2025E-2030E	3.1%	53.3%	16.7%



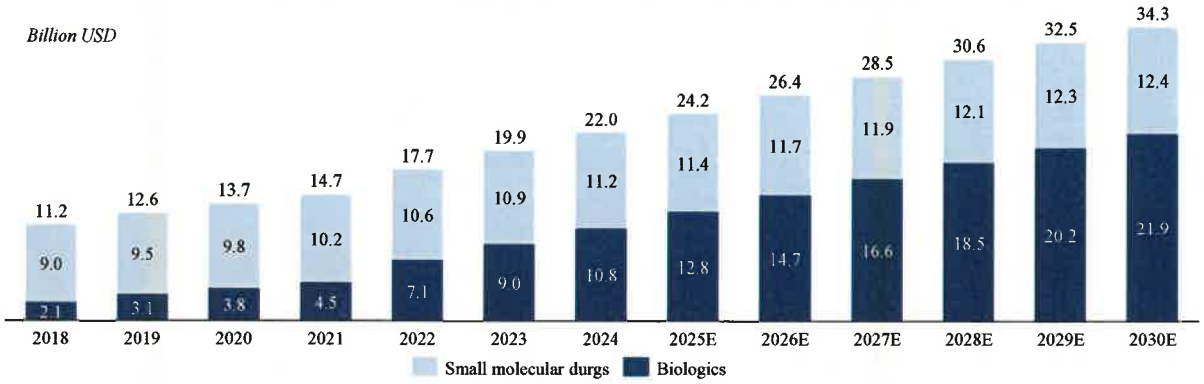
Source: Frost & Sullivan analysis

Global Market Size and Forecast of Allergic Asthma Drugs, 2018-2030E

- With the increasing adoption of biologics, growing patient awareness of allergic asthma, and rising treatment rates for allergic asthma, the global allergic asthma drugs market has maintained steady growth. The global market size of allergic asthma drugs grows from USD 11.2 billion to USD 22.0 billion from 2018 to 2024. It is expected to continue to grow to USD 34.3 billion by 2030, growing at a CAGR of 7.2% during the period.

Global Market Size of Allergic Asthma Drugs, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	3.6%	31.0%	11.9%
2025E-2030E	1.7%	11.4%	7.2%



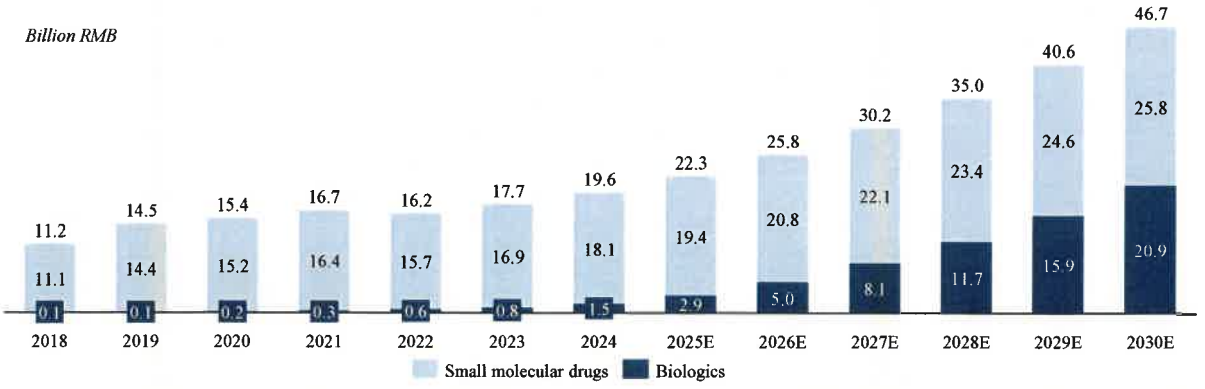
Source: Frost & Sullivan analysis

Market Size and Forecast of Allergic Asthma drugs in China, 2018-2030E

- With the rise in patient health awareness, the continuous approval of biologics, and the steady increase in biologic penetration rates and treatment compliance, China's allergic asthma drugs market is poised for rapid growth. The market size of asthma drugs in China grows from RMB 11.2 billion to RMB 19.6 billion from 2018 to 2024. It is expected to continue to grow to RMB 46.7 billion by 2030, growing at a CAGR of 16.0% during the period.

Market Size of Allergic Asthma Drugs in China, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	8.5%	65.5%	9.8%
2025E-2030E	5.9%	48.8%	16.0%



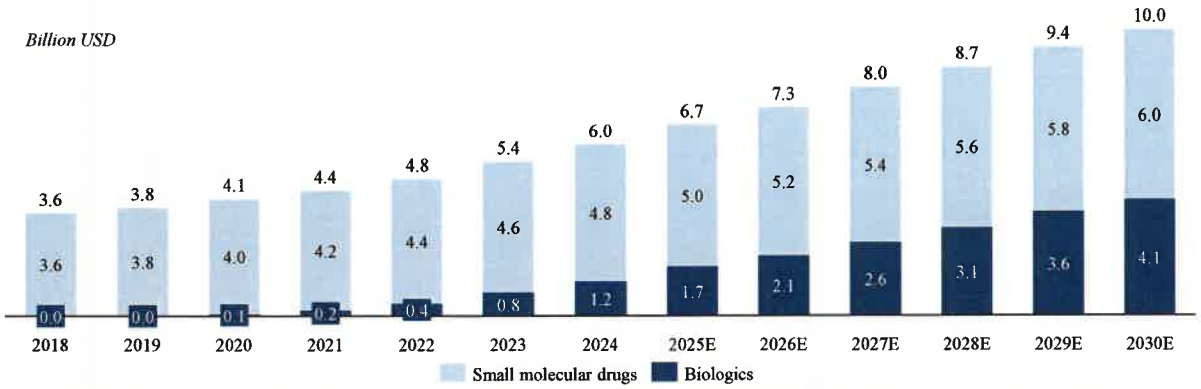
Source: Frost & Sullivan analysis

Global Market Size and Forecast of CRSwNP Drugs, 2018-2030E

- With the rise in patient health awareness, the increasing availability of biologics, and the growing treatment rates for CRSwNP, the global CRSwNP drugs market is projected to maintain steady growth. The global market size of CRSwNP drugs grows from USD 3.6 billion to USD 6.0 billion from 2018 to 2024. It is expected to continue to grow to USD 10.0 billion by 2030, growing at a CAGR of 8.3% during the period.

Global Market Size of CRSwNP Drugs, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	4.9%	NA	8.9%
2025E-2030E	3.6%	18.7%	8.3%



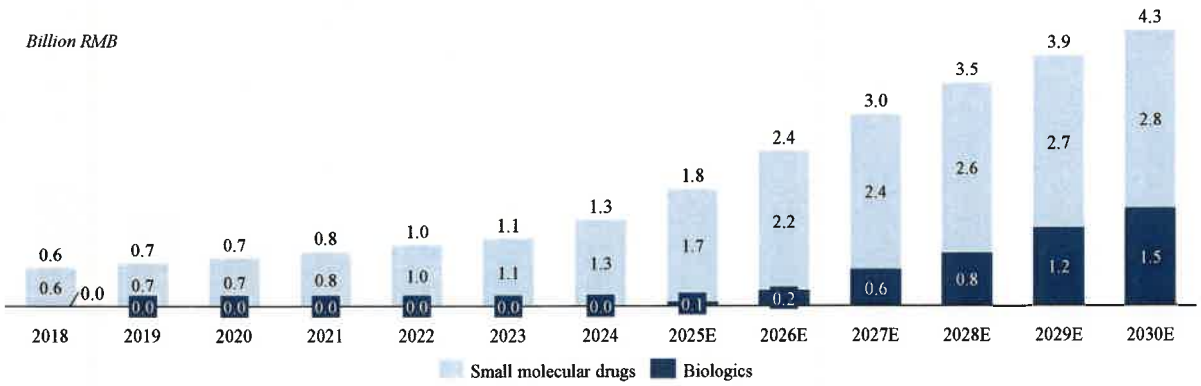
Source: Frost & Sullivan analysis

Market Size and Forecast of CRSwNP drugs in China, 2018-2030E

- With the rise in patient treatment rates, the continuous approval of biologics, and the growing penetration rate and treatment compliance to biological therapies, China's CRSwNP drugs market is poised for rapid expansion. The market size of CRSwNP drugs in China grows from RMB 0.6 billion to RMB 1.3 billion from 2018 to 2024. It is expected to continue to grow to RMB 4.3 billion by 2030, growing at a CAGR of 18.6% during the period.

Market Size of CRSwNP Drugs in China, 2018-2030E

Period	Small molecular drugs	Biologics	Overall
2018-2024	14.4%	NA	14.5%
2025E-2030E	9.5%	86.4%	18.6%

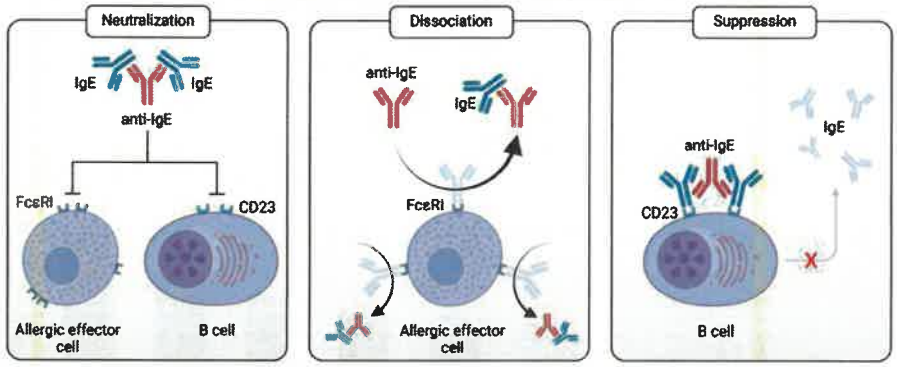


Source: Frost & Sullivan analysis

Mechanisms of Anti-IgE Antibody Therapy

- Anti-IgE antibodies are biologics targeting immunoglobulin E (IgE), whose mechanism of action is mainly associated with Type I hypersensitivity (immediate hypersensitivity). They bind to the CH3 domain of free IgE, preventing IgE from cross-linking with the high-affinity FcεRI receptors on the surface of mast cells and basophils, thus inhibiting cell degranulation and the release of allergic mediators such as histamine and leukotrienes. In addition, anti-IgE antibodies can block the binding of IgE to CD23 receptors on the surface of B cells and antigen-presenting cells, inhibiting the production of new IgE antibodies by B cells as well as their antigen-presenting function.

Multiple mechanisms of anti-IgE antibody



1. Binding of IgE and blocking its interaction with both the high-affinity receptor FcεRIα and the low-affinity receptor CD23 is the most classical characteristic of a therapeutic anti-IgE antibody

2. Anti-IgE antibodies can actively remove (dissociate) pre-complexed IgE from its high-affinity receptor FcεRIα on allergic effector cells including basophils, mast cells and dendritic cells

3. Some anti-IgE antibodies suppress IgE production in B-cells, most likely through a mechanism involving the binding of IgE:CD23 complexes on the cell surface.

Source: Literature Review, Frost & Sullivan Analysis

Comparison of anti-IgE antibody for different indications

Indication	Advantages	Disadvantages
Allergic rhinitis	• Early dosing in patients with seasonal AR significantly **reduces symptom severity**. • Combination with nasal hormones **reduces time to symptom relief**. • **Multiple symptom control** with improvement in nasal congestion, runny nose, and ocular itching.	• **Perennial AR requires long-term treatment** and maintenance of efficacy is dependent on continued medication, which is costly. • **Efficacy is affected by allergen exposure**, and dose adjustments may be needed in high allergen concentrations.
Chronic spontaneous urticaria	• **Provides rapid symptomatic relief**, with 75% of patients experiencing symptomatic improvement within 6 weeks. • **Breakthrough efficacy** of 60%-70% for antihistamine-resistant patients. • **Can replace oral glucocorticoids** and avoid hormonal side effects.	• **Inadequate response in some patients**, about 30% of patients need to exceed the prescribed dose (or combination of drugs.) • **Relapse after stopping the drug**, some patients need long-term maintenance therapy to prevent rebound.
Allergic asthma	• **Significant reduction in acute exacerbations** • **Improve lung function and reduce glucocorticoid dependence** • **Widely used in children and adults**, with a favorable safety profile.	• **Dosage limitations**, some patients may suffer from insufficient dosage. • **High cost**, long-term treatment is expensive, regular subcutaneous injections are required. • **Slow onset of action**, some patients need more than 12 weeks to show effect.
CRSwNP	• **Significant improvement in symptoms and polyp size**, reduced need for surgery and risk of recurrence. • **Reduce long-term dependence on nasal hormones** and systemic hormonal side effects. • **Better safety** with fewer systemic side effects	• Treatment is **costly and financially burdensome** for patients. • Inconvenient mode of administration and **poor patient compliance**.
Food Allergy	• Specifically binds free IgE, inhibiting the initiation of allergic reactions and **reducing the risk of systemic sensitization**. • **Reduce the need for conventional medications** such as glucocorticoids and epinephrine. • **Potential to induce immune tolerance** and alleviate long-term allergy symptoms.	• **Effective only for IgE-mediated food allergies**, not for non-IgE allergies. • **Individual variation is significant**, and some patients may not respond or may require long-term maintenance therapy. • **Monoclonal antibody-based drugs are expensive** and patient compliance is low.

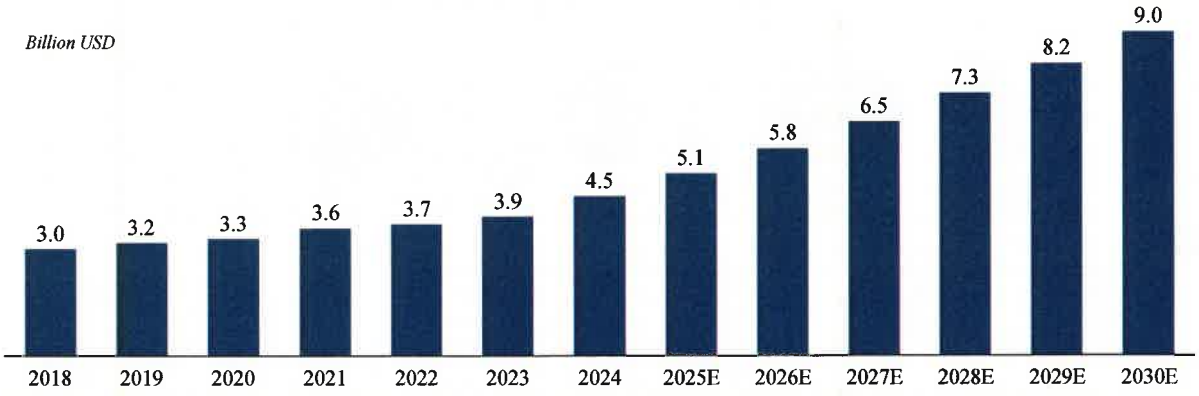
Source: Frost & Sullivan analysis

Global market size and forecast of anti-IgE antibody drugs, 2018-2030E

- The global market size of anti IgE antibody drugs grows from USD 3.0 billion to USD 4.5 billion from 2018 to 2024. It is expected to continue to grow to USD 9.0 billion by 2030, growing at a CAGR of 12.2% during the period.

Global Market Size of Anti-IgE Antibody Drugs, 2018-2030E

Period	CAGR
2018-2024	6.9%
2025E-2030E	12.2%



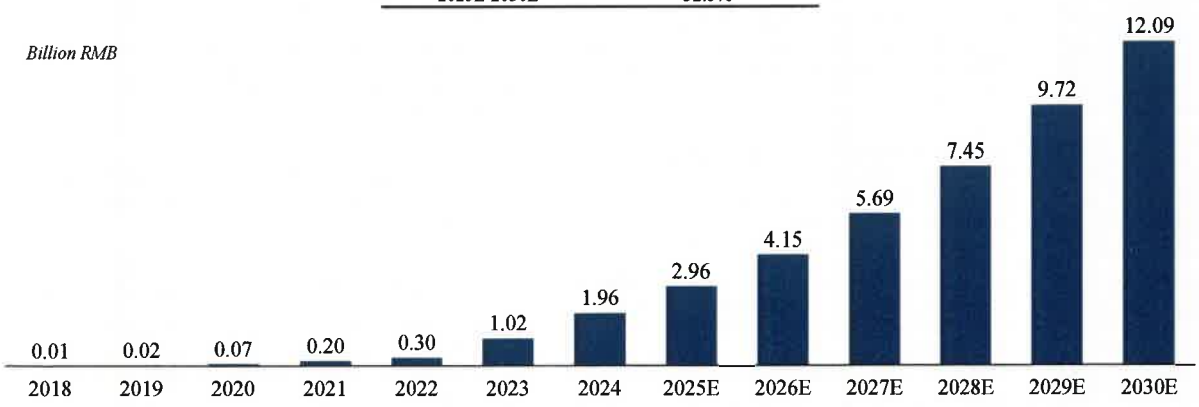
Source: Frost & Sullivan analysis

Market size and forecast of anti-IgE antibody drugs in China, 2018-2030E

- With the increasing popularity of biologics in the treatment of allergic diseases, the penetration rate of anti-IgE antibody drugs has been increasing, and the market size of anti-IgE antibody drugs has been growing rapidly. The market size of anti IgE antibody drugs in China grows from RMB 0.01 billion to RMB 2.0 billion from 2018 to 2024. It is expected to continue to grow to RMB 12.1 billion by 2030, growing at a CAGR of 32.5% during the period.

Market Size of Anti-IgE Antibody Drugs in China, 2018-2030E

Period	CAGR
2018-2024	140.0%
2025E-2030E	32.5%

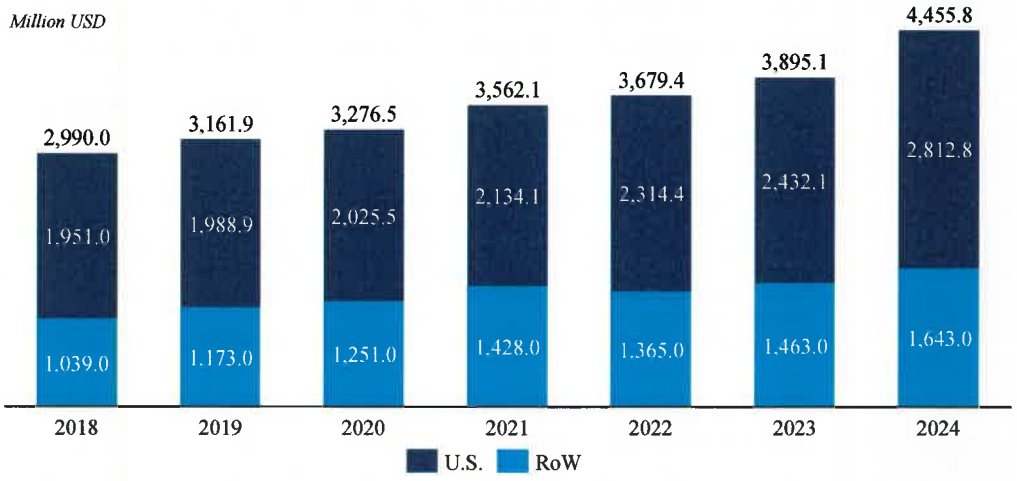


Source: Frost & Sullivan analysis

Sales revenue of Omalizumab, 2018-2030E

- First approved by the FDA in 2003, omalizumab is now approved around the world for the treatment of allergic asthma, allergic rhinitis, chronic spontaneous urticaria, food allergy, and chronic sinusitis with nasal polyps. As the indications continue to expand and the penetration of biologics in the treatment of allergic diseases continues to increase, the sales revenues of omalizumab are growing.

Sales revenue of Omalizumab, 2018-2030E



Source: Frost & Sullivan analysis

Drivers of Global Anti-IgE Antibody Drugs Market

Prevalence of allergic diseases continues to expand	• The prevalence of allergic diseases has continued to grow in recent years. According to the World Allergy Organization (WAO), the global prevalence of allergic diseases has tripled in the last 30 years, and nearly 40% of the world's population has been or is plagued by allergies. Allergic diseases have become one of the most important chronic diseases worldwide, and more effective treatments need to emerge.
Increased patient awareness and willingness to pay for innovative therapies	• The spread of disease education and the promotion of patient organizations have led to a significant increase in allergy patients' awareness of the importance of long-term management. More and more patients are proactively seeking precise treatment options rather than relying solely on traditional palliative medications, driving a shift in treatment demand from short-term symptom control to long-term disease management. Meanwhile, patients' increased willingness to pay for innovative therapies is providing a consumer base for the high-value biologics market.
Treatment paradigm shifts toward biologics	• The side effects and limited efficacy of conventional therapies (e.g., glucocorticoids) have led to a shift in clinical practice toward targeted biologic therapies. Innovative drugs such as anti-IgE antibodies have gradually become the standard treatment option for patients with moderate to severe allergies due to their superior safety and efficacy. At the policy level, the expansion of health insurance coverage and payment reform have further lowered the threshold for patients to use drugs, accelerating the penetration of biologics in clinical practice.
Emergence of innovative anti-IgE antibody products	• Existing anti-IgE antibody drugs have maintained market vitality by expanding indications and optimizing dosing regimens. R&D of new-generation anti-IgE drugs is accelerating, including dosage form innovation and precise design for different allergen phenotypes. The launch of biosimilars has further enriched market choices, forming a diversified product landscape where originator drugs, improved new drugs and biosimilars coexist, and jointly promoting the development of the anti-IgE antibody market.
Expanded Indications	• In recent years, the indications for anti-IgE antibodies in the treatment of allergic diseases have been expanding. Omalizumab was first approved for the treatment of asthma, and subsequently approved for the treatment of CSU and CRSwNP. 2024, omalizumab was approved by the FDA for the prevention of food allergy. As indications continue to expand, the applicable patient population for anti-IgE antibodies also continues to expand.

Source: Frost & Sullivan Analysis

Future Trends of Global Anti-IgE Antibody Drugs Market

Expanded indications for anti-IgE antibodies	• Anti-IgE antibody therapies are extending from traditional asthma treatment to a broader range of allergy-related diseases. Omalizumab has been approved for indications such as chronic spontaneous urticaria and nasal polyps, and clinical trials are exploring its potential in a broader range of allergic disease indications. With the development of precision medicine, targeted interventions against the IgE pathway may cover more IgE-mediated immune diseases, further expanding the market application scenarios.
Expanded range of applicable patients	• Anti-IgE antibodies are expanding their applicability from the traditional moderate-to-severe patients to mild or very severe cases. Studies have shown that they can significantly reduce the risk of acute exacerbations in patients with mild persistent asthma and promote early intervention strategies; meanwhile, in refractory severe cases, the new anti-IgE drugs can improve symptoms through more precise targeting and even achieve hormone tapering in some cases.
The emergence of a new generation of anti-IgE antibody drugs	• The new generation of anti-IgE antibodies breaks through the limitations of existing drugs by optimizing target affinity and pharmacokinetic properties. The new generation anti-IgE antibody has demonstrated higher efficacy than omalizumab in clinical trials, and the extended dosing interval is expected to improve patient compliance.
Combination therapy of anti-IgE antibodies	• The combination of anti-IgE antibodies with other biologics or traditional therapies is becoming an innovative trend. For example, combining with IL-5 inhibitors synergistically suppresses eosinophilic inflammation and achieves better symptom control in severe asthma, while combining with antihistamines enhances the efficacy in chronic urticaria. Personalized combination regimens based on patient phenotype are expected to become the mainstream treatment option for patients with allergic diseases.
Emergence of multi-targeted antibodies	• Multi-targeted antibodies are an important innovative direction in anti-IgE therapy, breaking through the limitations of single-targeted drugs and realizing the whole chain regulation of allergic cascade reaction by targeting IgE and the related inflammatory pathways at the same time.

Source: Frost & Sullivan Analysis

Challenges and threats of Anti-IgE Antibody Drugs Market

Slow onset of action of existing anti-IgE antibody drugs	• Current anti-IgE antibody drugs exert their effects by continuously inhibiting in vivo IgE levels, usually taking several weeks to show significant symptom control effects. However, patients with moderate-to-severe allergic diseases often have an urgent need for rapid symptom relief. This lag in onset may lead patients to doubt the drug efficacy and even switch treatment regimens midway, which to a certain extent restricts their market promotion.
Inconvenient administration of existing anti-IgE antibody drugs	• The dosage of current anti-IgE antibody drugs must be accurately calculated based on the patient's body weight, increasing the complexity of clinicians' operational processes. Meanwhile, some drugs are available in the form of lyophilized powder for injection, which need to be reconstituted on-site by professional medical staff and administered via injection. This not only prolongs the diagnosis and treatment time and raises requirements for medical staff's operational capabilities but also makes it difficult to use in primary medical institutions or home settings, further reducing medication convenience.
Frequent administration and poor patient compliance of existing anti-IgE antibody drugs	• All current anti-IgE antibody drugs are administered by injection, usually once every 2-4 weeks. Long-term treatment requires patients to repeatedly visit medical institutions, which not only increases time and transportation costs but also may reduce medication willingness due to fear of injections. The characteristic of frequent administration is likely to result in decreased patient compliance, such as missed doses or interrupted use, which not only affects the treatment effect but also may cause patients to switch to alternative drugs with more convenient administration.

Source: Frost & Sullivan Analysis

Entry barriers of Anti-IgE Antibody Drugs Market

Insufficient depth in biological mechanism research and understanding	• The R&D of anti-IgE antibodies heavily relies on in-depth comprehension of IgE-mediated allergic pathways. Inadequate understanding of such pathways can hinder precise optimization of target binding sites during drug design and prevent early prediction of potential side effects, significantly increasing the risks of R&D direction deviations or clinical failures and thus forming a critical entry barrier.
Technical barriers in protein engineering R&D capabilities	• Anti-IgE antibodies require complex protein engineering technologies to optimize core performance—for instance, improving binding efficiency with IgE via affinity maturation, extending in vivo half-life through Fc fragment modification, and reducing immunogenicity via humanization. These processes not only demand mature high-throughput screening platforms and structural biology analysis capabilities but also depend on stable CHO cell expression systems. The accumulation of such technologies requires long-term R&D practice and professional team support, which most small and medium-sized enterprises struggle to achieve, creating significant technical barriers.
Substantial capital investment required for biologics R&D	• As biologics , anti-IgE antibodies typically require an 8–12-year R&D cycle, with investments ranging from hundreds of millions to billions of US dollars, and there is a certain failure rate during clinical stages. Additionally, after launch,, continuous investment is needed to build GMP-compliant production facilities and cold chain logistics systems. The massive capital demand and high-risk nature make it difficult for financially weak enterprises to enter this field.

Source: Frost & Sullivan Analysis

Comparison of the Pharmacokinetics of the Anti-IgE Antibody Drugs

- With the advancement of antibody technology, next-generation anti-IgE antibody drug candidates have demonstrated significant advantages over omalizumab in terms of pharmacokinetics. Among these, the Group's candidate drug LP-003 exhibits a significantly superior binding dissociation constant(Kd) and half-life time compared to those of omalizumab, and it also holds a leading position among the major anti-IgE antibody drug candidates. Compared with omalizumab and other drug candidates, LP-003 binds more tightly to its target and persists longer in vivo. This means LP-003 delivers better and more durable efficacy, enabling less frequent injections and more convenient dosing for patients.

Comparison of the Pharmacokinetics of the Anti-IgE Antibody Drugs

Drug	Company	Kd (pM)	Half-life time (days)
Omalizumab	Novartis/Roche	1,790	20
LP-003	Longbio Pharma	2.08	45~76
RPT904/ozureprubart*	RAPT Therapeutics/Jeyou Pharma	~360	63
UB-221	United BioPharma	585	16-22
Ligelizumab	Novartis	35~139	17-23

Note: (1) RPT904 only disclosed that its affinity data showed a four-fold increase compared to omalizumab, without explicitly disclosing its Kd data. Therefore, estimates were made based on omalizumab's Kd data. (2) Novartis discontinued the clinical trials of ligelizumab for urticaria and food allergy in September 2023 and January 2024 respectively. Ligelizumab is also no longer included in the R&D pipeline disclosed in Novartis' latest annual report.

Source: desk research, Frost & Sullivan Analysis

Global competitive landscape of anti-IgE antibody approved by FDA

- As of the Latest Practical Date, there are 2 anti-IgE antibody drugs approved by FDA, including 1 original drug and 1 biosimilar.

Drug Name	Brand Name	Company	Indication	FDA Approval date	Sale Revenue in 2024 (million USD)	Original drug or biosimilar
Omalizumab	Xolair	Novartis/Roche	Food allergy	2024/2/16	4,455.8	Original drug
			CRSwNP	2020/12/1		
			CSU	2014/3/21		
			Moderate to severe asthma	2003/6/20		
Omalizumab-igec	Omlyclo	Celltrion	Food allergy	2025/3/9	N.A.	Biosimilar
			CRSwNP			
			CSU			
			Moderate to severe asthma			

*As of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Competitive landscape of anti-IgE antibody approved by NMPA in China

- As of the Latest Practical Date, there are three anti-IgE antibody drugs approved by NMPA, including one original drug and two biosimilars.

Drug Name	Brand Name	Company	Indication	NMPA Approval Date	Drug Delivery Program	Monthly treatment costs (RMB)	Covered by NRDL	Original drug or biosimilar
Omalizumab	Xolair	Novartis	CSU	2022/4/8	150/300mg given every 4 weeks.	~1,300/2,600	Yes	Original drug
			Allergic asthma	2017/8/24	300/450mg given every 4 weeks.	~5,200		
Omalizumab -CMAB007	Aomaishu	Taizhou Mabtech Pharmaceutical	Allergic asthma	2023/5/19	300/450mg given every 4 weeks.	~1,900/2,900	Yes	Biosimilar
Omalizumab -SYN008	Enyitan	CSPC Jushi Pharmaceutical	Allergic asthma	2025/1/26	300/450mg given every 4 weeks.	~1,900/2,900	No	Biosimilar
			CSU	2024/9/26	150/300mg given every 4 weeks.	~1,000/1,900		

Note: 1) Depending on the patient's condition, the dosage of medication used varies and the monthly cost of treatment varies. 2) Omalizumab first entered the National Reimbursement Drug List ("NRDL") in 2019, and its substance patent expired in China in 2016.

Source: NMPA, Frost & Sullivan Analysis

Global competitive landscape of anti-IgE antibody pipeline

- As of the Latest Practical Date, according to the clinicaltrials, there are 9 anti-IgE antibody candidates in the clinical stage, including 6 original drugs and 3 biosimilars.

Drug Code	Company	Indications	Clinical Stage	Latest update date	Original drug or biosimilar
Omalizumab	Novartis/Roche	Seasonal AR	Phase III	2026/1/12	Original drug
		COPD	Phase II	2026/1/29	
FB825	Oneness Biotech	Atopic Dermatitis	Phase II	2025/9/22	Original drug
		Allergic Asthma	Phase II	2024/5/28	
Ozureprubart	RAPT Therapeutics	Food Allergy	Phase II	2026/5/4	Original drug
Lesigercept	Yuhan Corporation	CSU	Phase II	2026/4/15	Original drug
UB-221	United BioPharma	CSU	Phase I	2022/5/13	Original drug
Exl-111	Excellergy	Allergic diseases	Phase I	2026/1/21	Original drug
Omalizumab-ADL018	Kashiv BioSciences	CSU	Phase III	2025/3/25	Biosimilar
Omalizumab-TEV-45779	Teva Pharmaceuticals	CSU	Phase III	2025/10/7	Biosimilar
Omalizumab-GNR044	Generium Pharmaceutical	Bronchial Asthma	Phase III	2020/10/29	Biosimilar

*As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Competitive landscape of anti-IgE antibody pipeline in China

- As of the Latest Practical Date, according to CDE, there are 7 anti-IgE antibody candidates in the clinical stage, including 4 original drugs and 3 biosimilars.

Drug Code	Company	Indications	Clinical Stage	Latest update date	Original drug or biosimilar
LP003	Longbio Pharma	AR	Phase III	2025/12/20	Original drug
		Allergic Asthma	Phase II	2025/2/13	
		CSU	Phase II	2025/2/9	
		CRSwNP	Phase II	2025/12/24	
JYB1904/Ozureprubart	Jiangsu Jiye Biopharmaceutical	CSU	Phase III	2026/2/6	Original drug
		Allergic Asthma	Phase II	2025/12/16	
		AR	Phase II	2026/3/10	
UB221	United Biopharma	CSU	Phase II	2025/9/11	Original drug
Lesigercept	Yuhan Corporation	CSU	Phase II	2026/4/9	Original drug
Omalizumab-CMAB007	Taizhou Mabtech Pharmaceutical	CSU	Phase III	2025/12/23	Biosimilar
		Allergic Asthma	Phase I	2025/5/13	
Omalizumab-SYB507	Yuanda Shuyang Pharmaceutical	CSU	Phase III	2024/6/14	Biosimilar
		Asthma	Phase I	2022/3/30	
Omalizumab-HS632	Hisun Pharmaceutical	Asthma	Phase I	2021/6/25	Biosimilar

*As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody approved for allergic rhinitis by NMPA in China

- As of the Latest Practical Date, there are only 1 monoclonal antibody drug approved for allergic rhinitis by NMPA.

Target	Drug name	Brand name	Company	NMPA Approval time	Drug Delivery Program	Monthly treatment costs (RMB)	Covered by NRDL
IL-4Rα	Stapokibart	Kangyueda	Keymed biosciences	2025/2/7	Initial dose of 600mg, followed by 300mg every two weeks	~3,600/2,400	Yes

Note: 1) Depending on the patient's condition, the dosage of medication used varies and the monthly cost of treatment varies.

Source: NMPA, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody pipeline for allergic rhinitis

- As of the Latest Practical Date, according to the clinicaltrials, there are 7 monoclonal antibody candidates for allergic rhinitis in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL-13	Lebrikizumab	Eli Lilly	Phase III	2026/4/20
IgE	Omalizumab	Novartis/Roche	Phase III	2026/1/12
IL-4Rα	Dupilumab	Sanofi/Regeneron	Phase III	2025/7/15
	VAK-694	Novartis	Phase II	2020/12/19
Bet v 1	REGN5713-5715	Regeneron	Phase III	2026/4/21
ADCYAP1	ALD1910	H. Lundbeck A/S	Phase I	2022/10/27
IL-33	MT-2990	Mitsubishi Tanabe Pharma	Phase I	2025/12/11
CD3, BCMA	Cizutamig	Candid Therapeutics	Phase I	2026/4/16

Note: In this page, we only consider innovative drugs and generic products are excluded.
*As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody pipeline for allergic rhinitis in China

- As of the Latest Practical Date, according to the CDE, there are 10 monoclonal antibody candidates for allergic rhinitis in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IgE	LP003	Longbio Pharma	Phase III	2025/2/10
	JYB1904/Ozureprubart	Jiangsu Jiye Biopharmaceutical	Phase II	2026/3/10
IL-13	Lebrikizumab	Eli Lilly	Phase III	2025/6/9
IL-4/IL-4Rα	Dupilumab	Sanofi	Phase III	2025/4/3
	Telikibart	Chongqing GenrixBio Biopharmaceutical	Phase III	2025/9/25
	Comekibart	Hunan Mabgeek Biotechnology	Phase II/III	2026/3/27
	TQH2722	Chiatai Tianqing Pharmaceutical	Phase II	2026/3/26
	SHR-1819	Hengrui Pharmaceutical	Phase II	2025/9/2
ST2	TQC2938	Chiatai Tianqing Pharmaceutical	Phase II	2026/4/20
IL-4Rα/ST2	AK139	Zhongshan Akeso Biopharma	Phase II	2026/2/13

Note: In this page, we only consider innovative drugs and generic products are excluded.
*As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody approved for chronic spontaneous urticaria by FDA

- As of the Latest Practical Date, there are 2 monoclonal antibody drugs approved for chronic spontaneous urticaria by FDA.

Target	Drug Name	Brand Name	Company	FDA Approval date	Sale Revenue in 2024 (million USD)
IgE	Omalizumab	Xolair	Novartis/Roche	2014/3/21	4,455.8
IL-4Rα	Dupilumab	Dupixent	Sanofi/Regeneron	2025/4/18	14,336.7

Note: In this page, we only consider innovative drugs and generic products are excluded.
*As of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody approved for chronic spontaneous urticaria by NMPA in China

- As of the Latest Practical Date, there are only 1 monoclonal antibody drug approved for chronic spontaneous urticaria by NMPA. In January 2023, omalizumab was included in the NRDL.

Target	Drug name	Brand name	Company	NMPA Approval time	Drug Delivery Program	Monthly treatment costs (RMB)	Covered by NRDL
IgE	Omalizumab	Xolair	Novartis/Roche	2022/4/8	150/300mg given every 4 weeks.	~1,300/2,600	Yes

Note: 1) In this page, we only consider innovative drugs and generic products are excluded. 2) Depending on the patient's condition, the dosage of medication used varies and the monthly cost of treatment varies.
*As of May 18, 2026

Source: NMPA, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody pipeline for chronic spontaneous urticaria

- As of the Latest Practical Date, according to the clinicaltrials, there are 7 monoclonal antibody candidates for chronic spontaneous urticaria in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
KIT	Barzolvolimab	Celldex Therapeutics	Phase III	2026/5/1
	Briquilimab	Jasper Therapeutics	Phase II	2025/2/28
TSLP	Tezepelumab	Amgen	Phase II	2025/4/9
SIGLEC6	AK006	Allakos	Phase I	2025/8/27
IgE	Lesigercept	Yuhan Corporation	Phase II	2026/4/15
	UB221	United BioPharma	Phase I	2022/5/13
CD3, BCMA	Cizutamig	Candid Therapeutics	Phase I	2026/4/16

Note: We only consider innovative drugs and generic products are excluded.
*As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody pipeline for chronic spontaneous urticaria in China

- As of the Latest Practical Date, according to the CDE, there are 11 monoclonal antibody candidates for chronic spontaneous urticaria in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL-4R	Telikibart	Genrixbio Pharmaceutical	Phase III	2025/4/8
	Dupilumab	Sanofi	Phase III	2024/12/28
	Comekibart	Hunan Mabgeek Biotechnology	Phase III	2026/3/30
	SHR-1819	Hengrui Pharmaceutical	Phase II	2025/11/10
	BA2101	Luye Pharma	Phase I	2023/11/1
IgE	Ozureprubart	Jeyou Pharma	Phase III	2026/2/6
	LP-003	Longbio Pharma	Phase II	2025/2/9
	UB221	United Biopharma	Phase II	2025/9/11
	Lesigercept	Yuhan Corporation	Phase II	2026/4/9
KIT	QX013N	Qyuns Therapeutics	Phase I	2025/8/6
IL-13, TSLP	CM512	Keymed biosciences	Phase II	2026/4/17

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody approved for allergic asthma by FDA

- As of the Latest Practical Date, there are 7 monoclonal antibody drugs approved for allergic asthma by FDA.

Target	Drug Name	Brand Name	Company	FDA Approval date	Sale Revenue in 2024 (million USD)
IgE	Omalizumab	Xolair	Novartis/Roche	2003/6/20	4,455.8
IL-5/IL-5Rα	Mepolizumab	Nucala	GSK	2015/11/4	2,302.1
	Depemokimab-ulaa	Exdensur		2025/12/16	N.A.
	Reslizumab	Cinqair	Teva	2016/3/23	undisclosed
	Benralizumab	Fasenra	AstraZeneca	2015/11/4	1,689.0
IL-4Rα	Dupilumab	Dupixent	Sanofi/Regeneron	2017/3/28	14,336.7
TSLP	Tezepelumab	Tespire	AstraZeneca	2021/12/17	248.0

Note: We only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody approved for allergic asthma by NMPA in China

- As of the Latest Practical Date, there are 4 monoclonal antibody drugs approved for allergic asthma by NMPA.

Target	Drug name	Brand name	Company	NMPA Approval time	Drug Delivery Program	Monthly treatment costs (RMB)	Covered by NRDL
IgE	Omalizumab	Xolair	Novartis/Roche	2017/8/24	300/450mg given every 4 weeks.	~2,600/3,900	Yes
IL-5/IL-5Rα	Mepolizumab	Nucala	GSK	2024/1/2	100mg given every 4 weeks.	~2,900	Yes
	Depemokimab	Exdensur		2026/5/7	100mg every 6 months	N.A.	No
IL-4Rα	Benralizumab	Fasenra	AstraZeneca	2024/9/27	First 3 doses of 30 mg every four weeks, subsequent 30 mg every eight weeks	~10,000/5,000	Yes
	Dupilumab	Dupixent	Sanofi/Regeneron	2023/11/14	Initial dose of 600/400 mg, followed by 300/200 mg every two weeks	~6,000/3,000, 4,400/2,200	Yes
TSLP	Tezepelumab	Tezspire	AstraZeneca	2026/3/25	210mg given every 4 weeks.	~13,700	No

Note: In this page, we only consider innovative drugs and generic products are excluded. Depending on the patient's condition, the dosage of medication used varies and the monthly cost of treatment varies.
* As of May 18, 2026

Source: NMPA, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody pipeline for allergic asthma(1/2)

- As of the Latest Practical Date, according to the clinicaltrials, there are 2 monoclonal antibody candidates for allergic asthma in the clinical phase III stage, as well as 23 monoclonal antibody candidates in the clinical phase II stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL-13	Tralokinumab	AstraZeneca	Phase III	2019/3/15
	Dectrekumab	Novartis	Phase II	2020/12/19
TSLP	GB-0895	Generate Biomedicines	Phase III	2026/4/27
	Verekitug	Upstream Bio Inc.	Phase II	2026/5/14
	HBM9378	Windward Bio	Phase II	2026/4/7
	AZD8630	AstraZeneca	Phase II	2025/12/1
	solrikitug	DevPro Biopharma	Phase II	2026/5/1
	GSK5784283	GSK	Phase II	2026/3/23
Tryptase	RG 6173	Roche	Phase II	2023/8/14
ST2	Astegolimab	Roche	Phase II	2022/12/28
OX40	Amlitelimab	Sanofi	Phase II	2026/3/30
	Rocatinlimab	Amgen	Phase II	2025/12/11
IL-6	FB704A	Oneness Biotech	Phase II	2025/12/9

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Updated

Global competitive landscape of monoclonal antibody pipeline for allergic asthma(2/2)

- As of the Latest Practical Date, according to the clinicaltrials, there are 2 monoclonal antibody candidates for allergic asthma in the clinical phase III stage, as well as 23 monoclonal antibody candidates in the clinical phase II stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IgE	FB825	Oneness Biotech	Phase II	2026/1/12
IL-4Rα	Rademikibart	Suzhou Connect Biopharmaceuticals	Phase II	2026/4/27
IL4, IL13, TSLP	Tilrekimig	Pfizer	Phase II	2026/5/4
IL-33	Tozorakimab	AstraZeneca	Phase II	2026/4/9
	SAR440340	Sanofi/Regeneron	Phase II	2022/6/14
IL-23α	Risankizumab	AbbVie/Boehringer Ingelheim	Phase II	2019/4/10
IL-1RL1	Melrilimab	GSK	Phase II	2020/3/2
IL-17Rα	CJM112	Novartis	Phase II	2021/10/8
	AMG 827	Amgen	Phase II	2021/11/26
IL13, TSLP	Lunsekimig	Sanofi	Phase II	2026/5/15
Fel d 1	REGN1908-1909	Regeneron	Phase II	2021/7/1
CD4	Tregalizumab	T-Balance Therapeutics GmbH	Phase II	2022/2/8

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Updated

Global competitive landscape of mAb pipeline for allergic asthma

Target	Drug Code	Company	Clinical Stage	Latest update date
IL-13	Tralokinumab	AstraZeneca	Phase III	2019/3/15
	Dectrekumab	Novartis	Phase II	2020/12/19
TSLP	GB-0895	Generate Biomedicines	Phase III	2026/4/27
	Verekitug	Upstream Bio Inc.	Phase II	2026/5/14
	HBM9378	Windward Bio	Phase II	2026/4/7
	AZD8630	AstraZeneca	Phase II	2025/12/1
	solrikitug	DevPro Biopharma	Phase II	2026/5/1
	GSK5784283	GSK	Phase II	2026/3/23
Tryptase	RG 6173	Roche	Phase II	2023/8/14
ST2	Astegolimab	Roche	Phase II	2022/12/28
OX40	Amlitelimab	Sanofi	Phase II	2026/3/30
	Rocatinlimab	Amgen	Phase II	2025/12/11
IL-6	FB704A	Oneness Biotech	Phase II	2025/12/9
IgE	FB825	Oneness Biotech	Phase II	2026/1/12
IL-4Rα	Rademikibart	Suzhou Connect Biopharmaceuticals	Phase II	2026/4/27
IL4, IL13, TSLP	Tilrekimig	Pfizer	Phase II	2026/5/4
IL-33	Tozorakimab	AstraZeneca	Phase II	2026/4/9
	SAR440340	Sanofi/Regeneron	Phase II	2022/6/14
IL-23α	Risankizumab	AbbVie/Boehringer Ingelheim	Phase II	2019/4/10
IL-1RL1	Melrilimab	GSK	Phase II	2020/3/2
IL-17Rα	CJM112	Novartis	Phase II	2021/10/8
	AMG 827	Amgen	Phase II	2021/11/26
IL13, TSLP	Lunsekimig	Sanofi	Phase II	2026/5/15
Fel d 1	REGN1908-1909	Regeneron	Phase II	2021/7/1
CD4	Tregalizumab	T-Balance Therapeutics GmbH	Phase II	2022/2/8

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: ClinicalTrials.gov, Frost & Sullivan Analysis

Updated

Competitive landscape of monoclonal antibody pipeline for allergic asthma in China(1/2)

- As of the Latest Practical Date, according to the CDE, there are 7 monoclonal antibody candidates for allergic asthma in the clinical phase III stage, as well as 16 monoclonal antibody candidates in the clinical phase II stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
TSLP	Bosakitug	Chiatai Tianqing Pharmaceutical	Phase III	2025/3/7
	SHR-1905	Hengrui Pharmaceutical	Phase III	2025/8/27
	QL2302	Qilu Pharmaceutical	Phase III	2025/11/27
	CM326	Keymed biosciences	Phase III	2026/4/27
	MG014	Hunan Mabgeek Biotechnology	Phase II	2025/10/29
	AZD8630	AstraZeneca	Phase II	2026/2/2
IL-5	SSGJ-610	3SBio Pharma	Phase II	2026/1/29
	SHR-1703	Hengrui Pharmaceutical	Phase II	2025/6/6
OX40	Rocatinlimab	Amgen	Phase II	2025/2/13
IL-11, TSLP	HB0056	Huabo Biopharm	Phase II	2025/9/4
IL-4R, ST2	AK139	Akeso Biopharma	Phase II	2026/2/6

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Updated

Competitive landscape of monoclonal antibody pipeline for allergic asthma in China(2/2)

- As of the Latest Practical Date, according to the CDE, there are 7 monoclonal antibody candidates for allergic asthma in the clinical phase III stage, as well as 16 monoclonal antibody candidates in the clinical phase II stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL-4Rα	Comekibart	Hunan Mabgeek Biotechnology	Phase III	2025/4/2
	Rademikibart	Connect Biopharma	Phase III	2024/8/2
	Stapokibart	Keymed biosciences	Phase II/III	2023/9/13
	Telikibart	Genrixbio Pharmaceutical	Phase II	2025/5/15
	LQ036	Shanghai Novamab Biopharmaceuticals	Phase II	2025/11/4
IL-13, TSLP	CM512	Keymed biosciences	Phase II	2026/1/13
	Lunsekimig	Sanofi	Phase II	2026/3/25
IgE	LP-003	Longbio Pharma	Phase II	2025/2/13
	Ozureprubart	Jeyou Pharma	Phase II	2025/12/16
IL-4R, IL-5	BBT002	Beijing Shanzhuyao Biopharmaceutical	Phase II	2025/11/24
	RC1416	Regenecore Biotech	Phase II	2026/1/13
IL-33	Tozorakimab	AstraZeneca	Phase II	2025/9/9

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Updated

Competitive landscape of mab pipeline for allergic asthma in China

Target	Drug Code	Company	Clinical Stage	Latest update date
TSLP	Bosakitug	Chiatai Tianqing Pharmaceutical	Phase III	2025/3/7
	SHR-1905	Hengrui Pharmaceutical	Phase III	2025/8/27
	QL2302	Qilu Pharmaceutical	Phase III	2025/11/27
	CM326	Keymed biosciences	Phase III	2026/4/27
	MG014	Hunan Mabgeek Biotechnology	Phase II	2025/10/29
	AZD8630	AstraZeneca	Phase II	2026/2/2
IL-5	SSGJ-610	3SBio Pharma	Phase II	2026/1/29
	SHR-1703	Hengrui Pharmaceutical	Phase II	2025/6/6
OX40	Rocatinlimab	Amgen	Phase II	2025/2/13
IL-11, TSLP	HB0056	Huabo Biopharm	Phase II	2025/9/4
IL-4R, ST2	AK139	Akeso Biopharma	Phase II	2026/2/6
IL-4Rα	Comekibart	Hunan Mabgeek Biotechnology	Phase III	2025/4/2
	Rademikibart	Connect Biopharma	Phase III	2024/8/2
	Stapokibart	Keymed biosciences	Phase II/III	2023/9/13
	Telikibart	Genrixbio Pharmaceutical	Phase II	2025/5/15
	LQ036	Shanghai Novamab Biopharmaceuticals	Phase II	2025/11/4
IL-13, TSLP	CM512	Keymed biosciences	Phase II	2026/1/13
	Lunsekimig	Sanofi	Phase II	2026/3/25
IgE	LP-003	Longbio Pharma	Phase II	2025/2/13
	Ozureprubart	Jeyou Pharma	Phase II	2025/12/16
IL-4R, IL-5	BBT002	Beijing Shanzhuyao Biopharmaceutical	Phase II	2025/11/24
	RC1416	Regenecore Biotech	Phase II	2026/1/13
IL-33	Tozorakimab	AstraZeneca	Phase II	2025/9/9

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody approved for CRSwNP by FDA

- As of the Latest Practical Date, there are 4 monoclonal antibody drugs approved for CRSwNP by FDA.

Target	Drug Name	Brand Name	Company	FDA Approval date	Sale Revenue in 2024 (million USD)
TSLP	Tezepelumab	Tespire	AstraZeneca	2025/10/17	248.0
IL-5	Mepolizumab	Nucala	GSK	2021/7/29	2,302.1
IgE	Omalizumab	Xolair	Novartis/Roche	2020/12/1	4,455.8
IL-4Rα	Dupilumab	Dupixent	Sanofi/Regeneron	2019/6/26	14,336.7

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Updated

Competitive landscape of monoclonal antibody approved for CRSwNP by NMPA in China

- As of the Latest Practical Date, there are 2 monoclonal antibody drugs approved for CRSwNP by NMPA.

Target	Drug name	Brand name	Company	NMPA Approval time	Drug Delivery Program	Monthly treatment costs (RMB)	Covered by NRDL
IL-4Rα	Stapokibart	康悦达	Keymed biosciences	2024/12/23	300mg given every 2 weeks	~2,400	Yes
IL-5	Mepolizumab	Nucala	GSK	2025/1/2	100mg given every 4 weeks.	~2,900	Yes
	Depemokimab	Exdensur		2026/5/7	100mg every 6 months	N.A.	No
TSLP	Tezepelumab	Tezspire	AstraZeneca	2026/3/25	210mg given every 4 weeks.	~13,700	No

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: NMPA, Frost & Sullivan Analysis

Updated

Global competitive landscape of monoclonal antibody pipeline for CRSwNP

- As of the Latest Practical Date, according to the clinicaltrials, there are 6 monoclonal antibody candidates for CRSwNP in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL33	Itepekimab	Sanofi/Regeneron	Phase III	2026/5/8
TSLP	Verekitug	Upstream Bio	Phase II	2025/8/15
IL-5/IL-5R	Benralizumab	AstraZeneca	Phase III	2024/6/18
	Depemokimab	GSK	Phase III	2025/12/3
IL13	Lebrikizumab	Eli Lilly	Phase III	2026/5/12
IL13, TSLP	Lunsekimig	Sanofi	Phase II	2026/4/28

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Updated

Competitive landscape of monoclonal antibody pipeline for CRSwNP in China

- As of the Latest Practical Date, according to the CDE, there are 13 monoclonal antibody candidates for CRSwNP in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
TSLP	Bosakitug	Chiatai Tianqing Pharmaceutical Group	Phase III	2025/10/10
	SHR-1905	Hengrui Pharmaceutical	Phase III	2025/9/12
	CM326	Keymed biosciences	Phase III	2026/2/6
IL-33	Itepekimab	Sanofi	Phase III	2025/8/12
IL-13	Lebrikizumab	Eli Lilly	Phase III	2025/2/14
IL-4Rα	Dupilumab	Sanofi	Phase III	2024/12/20
	SSGJ-611	Sunshine Guojian Pharmaceutical	Phase III	2025/10/22
	Telikibart	Chongqing GenrixBio Biopharmaceutical	Phase III	2025/8/1
	TQH2722	Chiatai Tianqing Pharmaceutical Group	Phase II	2024/8/2
	QX005N	Qyuns Therapeutics	Phase II	2025/7/30
IL-13,TSLP	CM512	Keymed biosciences	Phase II	2026/1/16
IgE	LP-003	Longbio Pharma	Phase II	2025/12/24
IL-4R, IL-5	BBT002	Beijing Shanzhuyao Biopharmaceutical	Phase II	2026/5/14

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody approved for food allergy by FDA

- As of the Latest Practical Date, there are only 1 monoclonal antibody drug approved for food allergy by FDA.

Target	Drug Name	Brand Name	Company	FDA Approval date	Sale Revenue in 2024 (million USD)
IgE	Omalizumab	Xolair	Novartis/Roche	2024/2/16	4,455.8

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Global competitive landscape of monoclonal antibody pipeline for food allergy

- As of the Latest Practical Date, according to the clinicaltrials, there are 7 monoclonal antibody candidates for food allergy in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IL33	Etokimab	AnaptysBio	Phase II	2023/7/27
IgE	Ozureprubart	RAPT Therapeutics	Phase II	2026/5/4
TSLP	Tezepelumab	AstraZeneca	Phase II	2026/4/29
Arah2, Arah6	IGNX001	IgGenix	Phase I	2025/11/14
CD3, BCMA	Linvoseltamab	Regeneron	Phase I	2026/5/5
-	MY006	Mabylon	Phase I	2026/5/6
CD3, BCMA	Cizutamig	Candid Therapeutics	Phase I	2026/4/16

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Competitive landscape of monoclonal antibody pipeline for food allergy in China

- As of the Latest Practical Date, according to the CDE, there are 1 monoclonal antibody candidate for food allergy in the clinical stage.

Target	Drug Code	Company	Clinical Stage	Latest update date
IgE	LP-003	Longbio Pharma	Phase I	2025/8/24

Note: In this page, we only consider innovative drugs and generic products are excluded.
* As of May 18, 2026

Source: CDE, Frost & Sullivan Analysis

Overview of Global Autoimmune Disease Drugs Market

Comparison of Autoimmune Disease Treatment

Treatment Category	Common Types	Common Drugs	Mechanism	Advantages
Biologics	Biologics	• Adalimumab • Etanercept • Golimumab • Infliximab	Target at molecules involved in the activation of the immune system, such as tumor necrosis factor (TNF) ,interleukin (IL) , B-cells and T-cells.	Newly emerging effective biologic drugs are available for patients with severe or resistant disease.
Small Molecular	Nonsteroid anti-inflammatory drugs (NSAIDs)	• Aspirin • Ibuprofen • Naproxen	Block prostaglandins, which can sensitize the nerves and magnify pain feelings during inflammation.	Work quickly and generally have fewer side effects than corticosteroids.
	Conventional DMARDs	• Methotrexate • Leflunomide	Inhibit the enzymes that affects DNA-synthesis for the proliferation of white blood cells, thus causing immunosuppression.	Long-term medication can effectively control symptoms and achieve stable efficacy.
	Corticosteroids	• Methylprednisolone • Dexamethasone • Prednisone	Stop the release of molecules that cause inflammation and also stop body from having an immune response.	Fast and strong anti-inflammatory effect that can be applied in many situations.
	JAK inhibitors	• Tofacitinib • Baricitinib	Inhibit immune cell function by inhibiting signal transduction of cytokines and growth factors.	Have shown satisfactory efficacy in patients resistant to other medications.
	Other Immuno-suppressants	• Such as mTOR inhibitors (Sirolimus, Everolimus)	Block the mammalian target of rapamycin (mTOR) which regulates cellular metabolism, growth, and proliferation.	Have shown tumor responses in clinical trials against both autoimmune diseases and various tumor types.

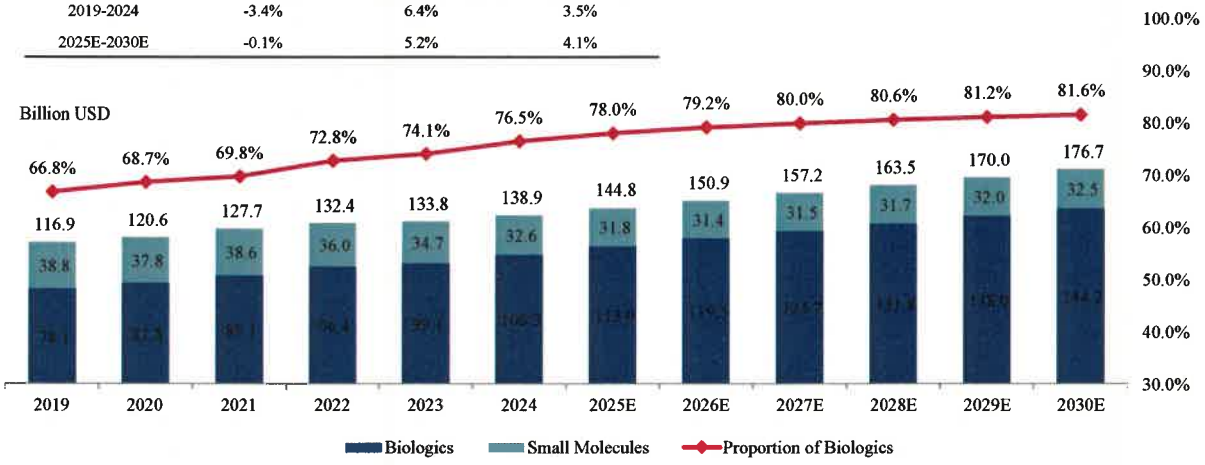
Source: Literature review, Frost & Sullivan analysis

Global Autoimmune Disease Drugs Market, 2019-2030E

- Global autoimmune diseases drugs market increased from USD116.9 billion in 2019 to USD138.9 billion in 2024 with a CAGR of 3.5 %. It is expected to reach USD 176.7 billion in 2030.
- Biologics accounted for approximately 76.5% of global autoimmune diseases drugs market in 2024. Targeted biologics has already replaced chemical drugs as the major treatment of autoimmune diseases. This sector is forecasted to account for approximately 81.6% of global autoimmune diseases drugs market in 2030.

Global Autoimmune Disease Drugs Market, 2019-2030E

Period	Small molecular drugs	Biologics	Overall
2019-2024	-3.4%	6.4%	3.5%
2025E-2030E	-0.1%	5.2%	4.1%



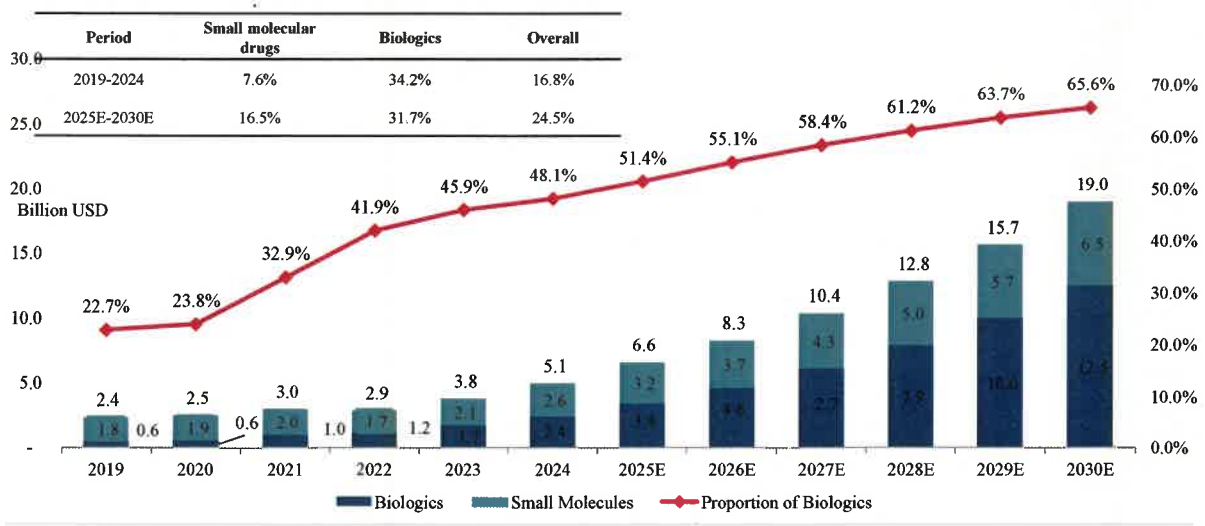
Source: Frost & Sullivan analysis

China Autoimmune Disease Drug Market, 2019-2030E

- Based on China's huge population, there is a large patient pool in Chinese market. With the development and improvement of diagnostics for autoimmune disease in China, the market demand of medical services would be spurred in the following years. The overall market increased from USD 2.4billion to USD 5.1 billion in 2024, representing a CAGR of 16.8%. It is expected to increase to USD 19.0 billion in 2030 with a CAGR of 24.5%.
- Given the large patient pool in China, and the development and advancement of innovative therapies for autoimmune diseases, the biologics sector has developed rapidly after 2017 because the boom of innovative biologics R&D. The market share of biologics increased from 22.7% in 2019 to 48.1% in 2024, and is forecasted to reach 65.6% in 2030.

Autoimmune Disease Drug Market in China, 2019-2030E

Period	Small molecular drugs	Biologics	Overall
2019-2024	7.6%	34.2%	16.8%
2025E-2030E	16.5%	31.7%	24.5%



Source: Frost & Sullivan analysis

Market Drivers of Autoimmune Disease Drugs Market (1/2)

lifestyle changes and rising prevalence rates.	• A key driver of China's autoimmune disease drug market is the increasing prevalence due to lifestyle changes. With accelerated urbanization, westernized dietary habits, heightened environmental stress, and increased mental pressure, the incidence of autoimmune disases has been rising year by year. The continuous expansion of the patient population not only fuels sustained demand for diagnosis and treatment but also provides long-term momentum for new drug development and market expansion, making it one of the critical factors supporting industry growth.
Growing patient awareness and stronger motivation to access medical treatment reflect evolving healthcare-seeking attitudes.	• In recent years, with the rise in public health awareness, faster dissemination of medical knowledge, and gradual improvement in diagnosis and treatment channels, more patients are able to receive early diagnosis and actively seek standardized treatment. This trend not only drives stable demand for existing drugs but also creates a solid foundation for the market penetration of innovative therapies, thereby further supporting the industry's sustained growth.
The advancement of diagnostic technology has led to an increase in disease detection rates.	• With continuous advancements in imaging, molecular testing, and autoantibody detection technologies, as well as the promotion of clinical guidelines and the improvement of doctors' diagnostic and treatment capabilities, patients are able to receive clear diagnoses at an early stage. The rise in detection rates not only facilitates the effective identification of patient populations and the release of treatment demands but also provides solid support for the sustained expansion of the drug market.

Source: Frost & Sullivan Analysis

Market Drivers of Autoimmune Disease Drugs Market (2/2)

Policy Support.	• In recent years, the government has consistently introduced favorable policies in areas such as accelerating the review and approval of innovative drugs, optimizing the clinical trial environment, and dynamically adjusting the national reimbursement drug list. The Implementation Plan for Full-Chain Support for the Development of Innovative Drugs coordinates core policy areas including price management, medical insurance payment, and commercial insurance, providing systematic policy support for the rapid development of the innovative drug industry. These measures have effectively shortened the time-to-market for new drugs and improved patient access to medications. Additionally, continuous improvements in diagnostic and treatment guidelines for autoimmune diseases, along with stronger promotion of clinical applications, have collectively created a favorable environment for industry development and injected long-term momentum into market growth.
Insurance coverage drives market growth and the penetration of biologics	• The Several Measures for Supporting the High-Quality Development of Innovative Drugs further establishes a multi-level security system integrating dynamic medical insurance access and commercial insurance gradient protection mechanisms, continuously optimizing the market ecology for innovative drugs. With dynamic adjustments to the national reimbursement drug list, high-cost biologic agents are gradually being included in the coverage, significantly reducing patients' financial burden and improving treatment accessibility. This not only expands the medication scale for moderate-to-severe patients but also accelerates the clinical adoption of innovative drugs, thereby driving overall market growth and increasing the proportion of biologics in autoimmune treatment.
Diversified Targets and Accelerated R&D Progress	• Currently, in addition to traditional targets such as TNF-α and IL-6, emerging targets like JAK, BTK, IL-17, and IL-23 are continuously achieving breakthroughs, with related pipelines rapidly advancing into clinical research and commercialization stages. The diversification of targets not only broadens treatment options, meeting the diverse needs of different patient groups, but also stimulates companies' enthusiasm for investing in innovative drug development, thereby injecting sustained innovation momentum and growth potential into the market.

Source: Frost & Sullivan Analysis

Future Trends of Autoimmune Disease Drugs Market (1/2)

Sustained Market Growth.	• China's autoimmune disease (AID) drugs market is projected to maintain steady growth over the coming years. This upward trajectory will be driven by multiple factors: the expanding patient population, improvements in diagnosis and standardized treatment protocols, as well as the continuous introduction of innovative therapies and broader medical insurance coverage. Concurrently, the substantial unmet clinical needs present vast opportunities for novel mechanisms of action and differentiated treatment approaches. As a result, the AID drugs market in China is well-positioned to sustain robust growth momentum in the long term.
Increasing market share of biologics.	• With advantages in efficacy, targeting, and safety, biologics are gradually becoming the primary treatment option for moderate to severe patients. Meanwhile, the accelerated progress in the R&D pipelines of domestic innovative pharmaceutical companies and the expansion of medical insurance coverage are expected to further enhance drug accessibility and patient acceptance. As a result, the penetration rate of biologics in the autoimmune drug market is projected to keep rising, becoming a significant driver for the industry's future development.
The advancement of diagnostic technology has led to an increase in disease detection rates.	• With continuous advancements in imaging, molecular testing, and autoantibody detection technologies, as well as the promotion of clinical guidelines and the improvement of doctors' diagnostic and treatment capabilities, patients are able to receive clear diagnoses at an early stage. The rise in detection rates not only facilitates the effective identification of patient populations and the release of treatment demands but also provides solid support for the sustained expansion of the drug market.

Source: Frost & Sullivan Analysis

Future Trends of Autoimmune Disease Drugs Market (2/2)

Policy Support.	• In recent years, the government has consistently introduced favorable policies in areas such as accelerating the review and approval of innovative drugs, optimizing the clinical trial environment, and dynamically adjusting the national reimbursement drug list. The Implementation Plan for Full-Chain Support for the Development of Innovative Drugs coordinates core policy areas including price management, medical insurance payment, and commercial insurance, providing systematic policy support for the rapid development of the innovative drug industry. These measures have effectively shortened the time-to-market for new drugs and improved patient access to medications. Additionally, continuous improvements in diagnostic and treatment guidelines for autoimmune diseases, along with stronger promotion of clinical applications, have collectively created a favorable environment for industry development and injected long-term momentum into market growth.
Insurance coverage drives market growth and the penetration of biologics	• The Several Measures for Supporting the High-Quality Development of Innovative Drugs further establishes a multi-level security system integrating dynamic medical insurance access and commercial insurance gradient protection mechanisms, continuously optimizing the market ecology for innovative drugs. With dynamic adjustments to the national reimbursement drug list, high-cost biologic agents are gradually being included in the coverage, significantly reducing patients' financial burden and improving treatment accessibility. This not only expands the medication scale for moderate-to-severe patients but also accelerates the clinical adoption of innovative drugs, thereby driving overall market growth and increasing the proportion of biologics in autoimmune treatment.
Dual and Multi-Target Advantages.	• Compared to single-target drugs, these therapies can simultaneously act on multiple key pathways, enhancing both the breadth and durability of treatment while potentially reducing the risk of drug resistance. In clinical practice, dual-target and multi-target approaches offer more precise treatment options for patients with complex disease progression or comorbidities. With ongoing advancements in the R&D pipeline and the accumulation of clinical data, these therapies are expected to become a critical area for differentiated competition, further elevating the market landscape.

Source: Frost & Sullivan Analysis

Challenges and threats of Autoimmune Disease Drugs Market

Low penetration of biologics in the autoimmune disease drugs market.	• Despite their clear advantages in efficacy, targeting, and therapeutic value for moderate to severe patients, the clinical application of biologics is constrained by factors such as high price levels, limited insurance coverage, and insufficient acceptance among doctors and patients. The low penetration rate has somewhat hindered the adoption speed of innovative therapies and introduced uncertainty for further market expansion..
Intense competition from rival products and target saturation	• Currently, classic targets such as TNF-α, IL-6, and JAK have attracted multiple drugs in development or already on the market, intensifying competition. Meanwhile, emerging targets like BTK, IL-17, and IL-23 are also advancing rapidly. In the future, as more products enter clinical trials or commercialization, market differentiation will become even more pronounced. This places higher demands on companies in terms of efficacy validation, pricing strategies, and clinical value creation, while also increasing uncertainty in industry development.
Challenges in Diagnostic Technology and Diagnosis Rates	• Although molecular diagnostics and autoantibody testing technologies have advanced in recent years, some regions still face limitations such as inadequate diagnostic tools, limited testing accessibility, and uneven clinical application. These issues result in delayed or inaccurate diagnoses for certain patients during early disease stages. Low diagnosis rates not only hinder standardized treatment and medication demand but also constrain the full realization of market potential.

Source: Frost & Sullivan Analysis

Table of Contents

Mechanisms and Characteristics of Complement Inhibitors

- The complement system is a self-protection mechanism of the human body. The abnormal activation of the complement system is involved in the occurrence and development of various diseases. The use of complement inhibitors has brought landmark progress for many rare diseases, such as paroxysmal nocturnal hemoglobinuria (PNH), atypical hemolytic uremic syndrome (aHUS), and so on. In recent years, the application of complement inhibitors has also gradually expanded to other complement-related disease areas.

Mechanisms of Complement Inhibitors

◆ Complement inhibitors work by targeting key proteins of the complement system (such as C3, C5, Factor D/B) to block their activation pathways (classical, lectin, alternative), precisely inhibiting excessive complement activation. For example, C5 inhibitors (such as eculizumab) prevent the cleavage of C5 into pro-inflammatory factor C5a and membrane attack complex C5b, while C3 inhibitors (such as Pegcetacoplan) block the central node of the complement cascade, the C3 convertase, reducing inflammation and tissue damage. Some drugs can also mimic natural regulatory proteins (CD55/CD59), protecting host cells from misdirected attacks.

Targeting key complement proteins

➢ **Inhibit C3/C5 protein**
- C3 inhibitor:Blocking C3 convertase, inhibiting the "core node" of the complement cascade, and preventing the generation of C3a (pro-inflammatory factor) and C3b (opsonin).
- C5 inhibitors: Bind to C5 protein and prevent it from cleaving into C5a (a potent chemotactic factor) and C5b (the starting component of the membrane attack complex MAC).

➢ **Block other complement factors**
- Factor D/B inhibitors: inhibit key enzymes of the bypass pathway (e.g., Factor D/B), reducing the formation of C3 converting enzymes.

Mechanisms of Complement inhibitors (C3&C5)

➢ Complement inhibitors (C3&C5) work by synergistically blocking the upstream C3 activation and terminal C5 cleavage pathway of the complement system, in which inhibition of C3 can block the formation of C3 converting enzyme, reduce the deposition of C3b and the release of C3a inflammatory mediators, and inhibition of C5 can prevent the release of C5a chemokines and the generation of membrane attack complex (C5b-9), thus controlling the abnormal activation of complement in both the "central hub" and "terminal toxicity" aspects.

➢ "Compared with single-target inhibitors, it has the advantage of avoiding the limitations of residual activation of the terminal pathway when only C3 is inhibited or the persistence of C3a inflammatory effect when only C5 is inhibited, and it is more comprehensively inhibiting complement-mediated inflammatory mediators through dual pathway synergism. Compared with single-target inhibitors, they have the advantage of avoiding the limitation of inhibiting only the residual activation of the terminal pathway at C3 or the persistence of the inflammatory effect of C3a at C5, and inhibiting the complement-mediated tissue damage more comprehensively through dual-pathway synergism.

Source: Frost & Sullivan Analysis

Development History of Complement Inhibitors

1890s ~ 1900s

➢ In 1890, Belgian immunologist Bordet discovered and confirmed the existence of a heat-intolerant component in human and animal fresh serum that can assist and supplement specific antibodies to kill microorganisms, so it is called complement (Complement).

➢ Since the discovery of complement in the 19th century, the research on the complement system and its drug development has never stopped.

2007s~ 2021s

➢ In 2007, the first C5-targeted monoclonal antibody drug, eculizumab (Soliris®), developed by Alexion was approved for marketing, marking the official entry of complement-targeted therapy into clinical application. However, in the more than ten years since then, the progress of new drug research and development in this field has been slow. Until 2018, ravulizumab (Ultomiris®), a long-acting C5 inhibitor engineered through the Fc fragment, came out. It optimizes the administration frequency by prolonging the antibody half-life. It not only consolidates Alexion's leading position in the industry but also provides key clinical verification and technical paradigms for the precise regulation of the complement pathway.

2010s ~ Today

➢ In 2021, Apellis Pharmaceuticals launched Pegcetacoplan (Empaveli®), the first C3-targeted complement inhibitor, challenging Alexion's (AstraZeneca) dominance in C5 inhibition. This breakthrough spurred competition, with Annexon, Novartis, ChemoCentryx, Sanofi, and AstraZeneca advancing diversified pipeline strategies—ranging from classical pathway blockers to oral antagonists—through internal R&D and partnerships. The influx of novel mechanisms underscores the complement therapeutics field's evolution into a competitive, high-potential market driven by unmet clinical needs..

Source: Literature Review, Frost & Sullivan Analysis

Clinical Application of Complement Inhibitors

- The clinical application of complement inhibitors has brought breakthroughs in the treatment of rare diseases such as paroxysmal nocturnal hemoglobinuria (PNH) andIgA nephropathy(IgA). In recent years, with the deepening of research, the application scope of complement inhibitors has continued to expand, and it has also shown significant clinical value in more complement-related diseases such as glomerulopathy and myasthenia gravis (MG).

Application in paroxysmal nocturnal hemoglobinuria

- Complement inhibitors (such as the C5 inhibitor ecuzumab, Ravulizumab, and the C3 inhibitor Pegcetacoplan) are milestones in the treatment of PNH, especially for PNH patients with classical or combined bone marrow failure. By blocking the complement terminal pathway (C5 inhibitor) or upstream C3 activation (C3 inhibitor), significantly reduce the risk of intravascular hemolysis and thrombosis, improve anemia and quality of life. Ravulizumab can prolong the dosing interval due to its long half-life, while the novel C3 inhibitor can simultaneously inhibit C3-mediated extravascular hemolysis.

Application in IgA nephropathy

- The clinical application of complement inhibitors in IgA nephropathy (IgAN) is promising. Given that the abnormal activation of the complement system is a key pathogenetic link in the pathogenesis of IgAN, complement inhibitors can precisely target this pathological process, which is expected to block the progression of renal injury, especially for patients with poor response to traditional immunosuppressive drugs or high disease activity. Currently, complement inhibitors with different targets, such as eculizumab, ravulizumab, and iptacopan, have shown positive effects in clinical trials. For example, iptacopan can reduce the urinary protein/creatinine ratio and inhibit the level of complement activation markers in patients.

Application in C3 glomerulopathy

- For C3 glomerulopathy due to abnormal activation of the alternative complement pathway, the C5 inhibitor (eculizumab) has shown partial efficacy in individual cases, but the overall evidence is limited. New alternative pathway inhibitors such as the B-factor inhibitor Iptacopan can reduce urinary protein and C3 deposition in renal tissue in phase II trials, providing hope for future treatment.
- Currently,Novartis' iptacopan has been recommended for approval by the European Medicines Agency (EMA) for the treatment of C3G and has become the world's first complement inhibitor for this disease.

Clinical Application

Source: Literature Review, Frost & Sullivan Analysis

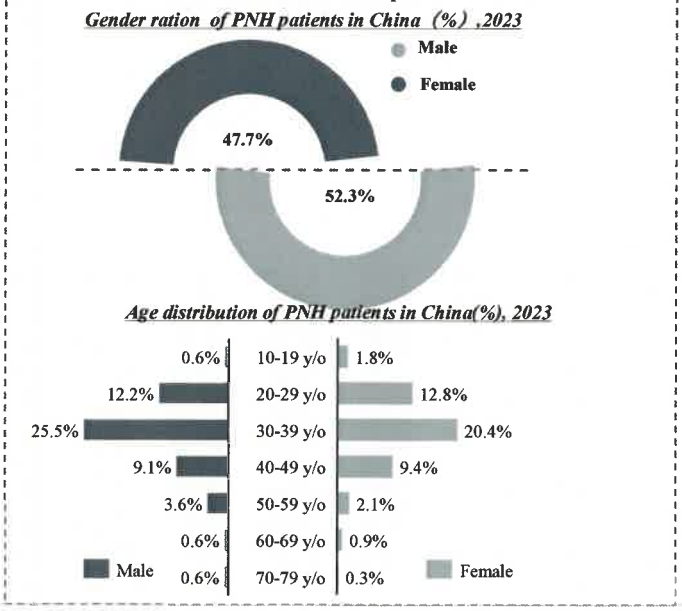
Overview of Paroxysmal Nocturnal Hemoglobinuria(PNH)

- Paroxysmal nocturnal hemoglobinuria (PNH) is a rare disease that presents clinically with a variety of symptoms, the most prevalent of which are hemolytic anemia, hemoglobinuria, and somatic symptoms including fatigue and shortness of breath. Other findings associated with PNH include thrombosis, renal insufficiency, and in the later course of the disease, even bone marrow failure. The condition is genetic, with the mutations occurring on the X linked gene.

Pathology

- Paroxysmal nocturnal hemoglobinuria occurs due to the development of a genetic mutation in hematopoietic stem cells. This mutation of the X-linked gene phosphatidylinositol glycan class A (PIGA), produces a deficiency in the glycosylphosphatidylinositol (GPI) protein, which is responsible for anchoring other protein moieties to the surface of erythrocytes
- Proteins responsible for the regulation of complement activity, specifically CD55 and CD59, are thereby prevented from attaching to PNH affected cells. The resultant loss of complement inhibition produces chronic complement-mediated hemolysis of PNH cells. This chronic state of hemolysis can be exacerbated if the complement system is activated by stress due to surgery, trauma, or other triggers for inflammation

Characteristics of PNH patients

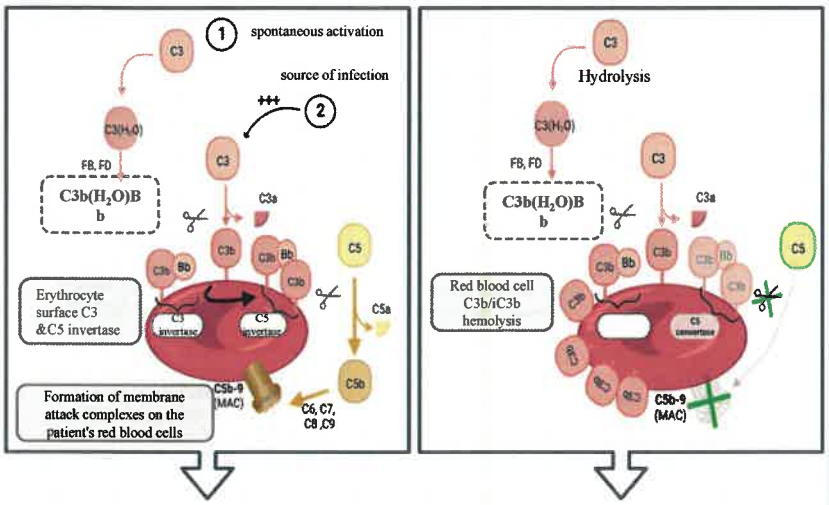


Source: Literature Review, Frost & Sullivan Analysis

Pathogenesis of Paroxysmal Nocturnal Hemoglobinuria(PNH)

- Paroxysmal Nocturnal Hemoglobinuria (PNH) arises from an acquired somatic mutation in the *PIG-A* gene of hematopoietic stem cells, leading to deficient glycosylphosphatidylinositol (GPI) anchor biosynthesis and loss of membrane-bound complement regulatory proteins like CD55 and CD59 on erythrocytes and other blood cells. This makes cells hypersensitive to complement-mediated lysis, particularly by the alternative pathway, causing uncontrolled activation that forms the membrane attack complex (MAC) and triggers intravascular hemolysis, while C3b-opsonized cells undergo extra-vascular hemolysis in the spleen and liver.

Pathogenesis of PNH



- Paroxysmal nocturnal hemoglobinuria is a rare clonal disease of acquired hematopoietic stem cells. Its pathogenesis is a somatic mutation based on the X chromosome glycosylated phosphatidylinositol-A gene at the level of hematopoietic stem cells, resulting in the loss of anchoentin on the cell surface, which in turn triggers complement-mediated intravascular and extravascular hemolysis.

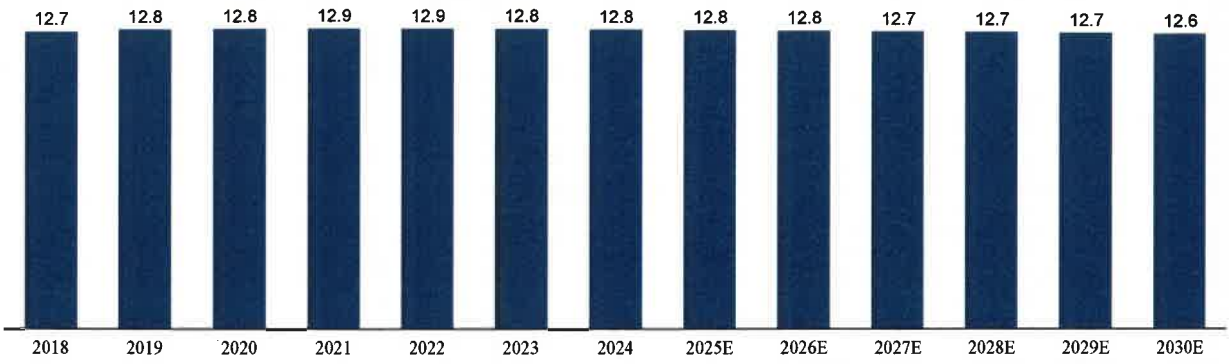
Source: Literature Review, Frost & Sullivan Analysis

Prevalence of Paroxysmal Nocturnal Hemoglobinuria(PNH) in China, 2018-2030E

- Paroxysmal Nocturnal Hemoglobinuria (PNH) is an acquired clonal disorder of hematopoietic stem cells caused by mutations in the PIG-A gene located on the X chromosome. The pathological hallmark of PNH is abnormal synthesis of glycosyl phosphatidyl inositol (GPI), resulting in the deficiency of GPI-anchored proteins (e.g., CD55 and CD59) on blood cell membranes. Clinically, the disease primarily manifests as intravascular hemolysis (IVH), bone marrow failure, and a high risk of thrombosis.
- From 2018 to 2024, the prevalence of Paroxysmal Nocturnal Hemoglobinuria (PNH) in China fluctuated from 12.7 thousand cases to 12.8 thousand cases in 2024, with a CAGR of 0.1%. The prevalence of PNH in china is expected to reach 12.6 thousand cases in 2030 at a CAGR of -0.3%.

Prevalence of PNH in China, 2018-2030E

Period	CAGR
2018-2024	0.1%
2025E-2030E	-0.3%



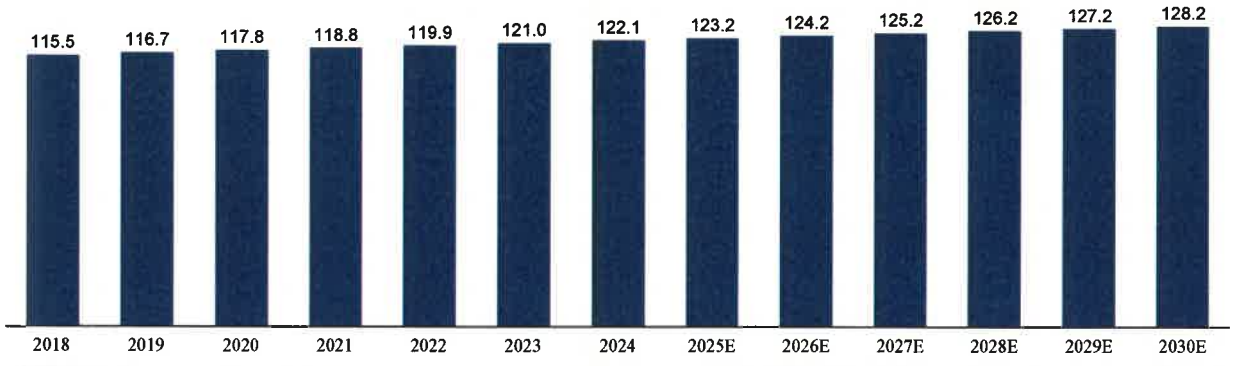
Source: CHIINESE JOURNAL OF HEMATOLOGY 2024, Frost & Sullivan Analysis

Global Prevalence of Paroxysmal Nocturnal Hemoglobinuria(PNH), 2018-2030E

- From 2018 to 2024, the global Prevalence of Paroxysmal Nocturnal Hemoglobinuria (PNH) fluctuated from 115.5 thousand cases to 122.1 thousand in 2024, with a CAGR of 0.9%. The global Prevalence of PNH is expected to reach 128.2 thousand cases in 2030 at a CAGR of 0.8% from 2024 to 2030.

Global Prevalence of PNH, 2018-2030E

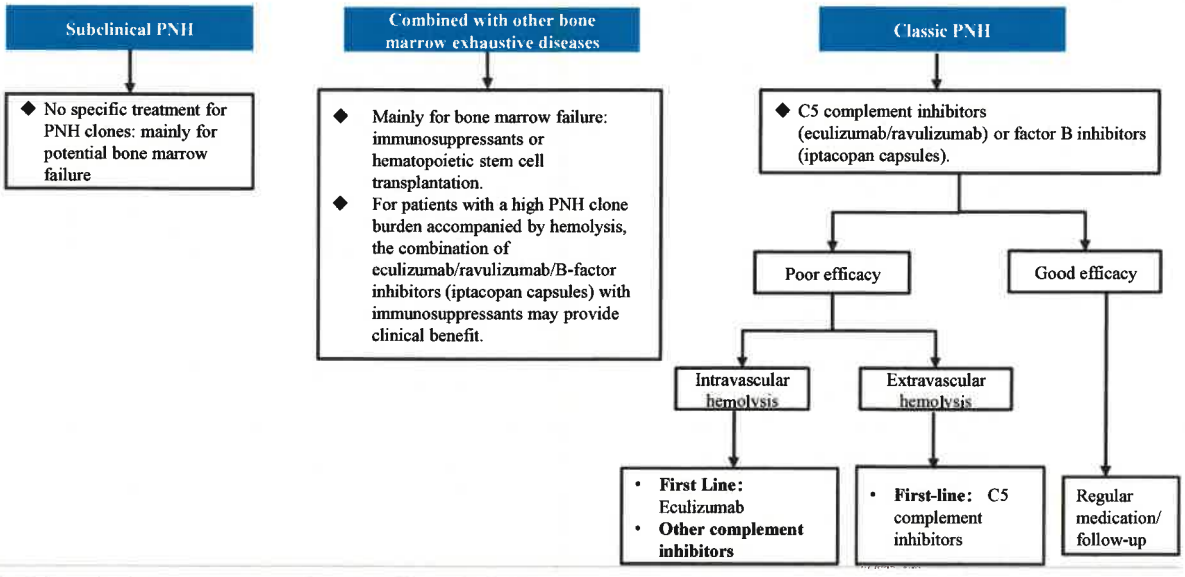
Period	CAGR
2018-2024	0.9%
2025E-2030E	0.8%



Source: Literature Review, Frost & Sullivan Analysis

Treatment Paradigm of Paroxysmal Nocturnal Hemoglobinuria(PNH)

- Diagnostic pathway for paroxysmal sleep hemoglobinuria (PNH) In terms of treatment, complement inhibitors (e.g., C5 monoclonal antibody eculizumab or factor B inhibitors) are preferred to control intravascular hemolysis and to prevent thrombosis in patients with classic type; combined immunosuppressants (e.g., cyclosporine A/ATG) and pro-hematopoietic therapy are required for those with combined bone marrow failure; and subclinical type needs only to deal with underlying bone marrow failure. Allogeneic HSCT may be considered in refractory cases or those who have progressed to MDS/leukemia, and in young high-risk patients. Management of complications includes anticoagulation for thrombosis, protection of renal function, and multidisciplinary monitoring during pregnancy, and glucocorticoids or proximal complement inhibitors may be used in combination if extravascular hemolysis is triggered by C5 inhibitors.

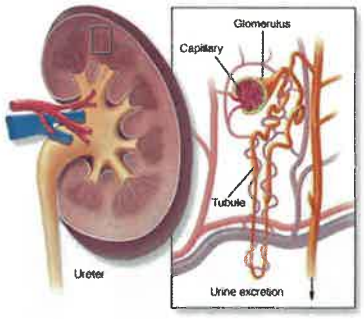


Source: CHIINESE JOURNAL OF HEMATOLOGY 2024, Frost & Sullivan Analysis

Overview of IgA nephropathy

Mechanisms of IgA nephropathy

- IgA nephropathy is a nephritic syndrome, a form of chronic glomerulonephritis characterized by the deposition of IgA immune complexes in glomeruli. It is the most common form of glomerulonephritis worldwide.



- Increased IgA1 production
- Defective IgA1 glycosylation causing increased binding to mesangial cells
- Decreased IgA1 clearance
- A defective mucosal immune system
- Overproduction of cytokines stimulating mesangial cell proliferation

Overview

1 Brief Introduction

- Immunoglobulin A (IgA) nephropathy is deposition of IgA immune complexes in glomeruli, manifesting as slowly progressive hematuria, proteinuria, and, often, renal insufficiency.

2 Symptom

- Hematuria
- Flank Pain
- Edema
- Foamy urine

3 Diagnosis

- Urinalysis
- Renal biopsy

4 Risk Factors

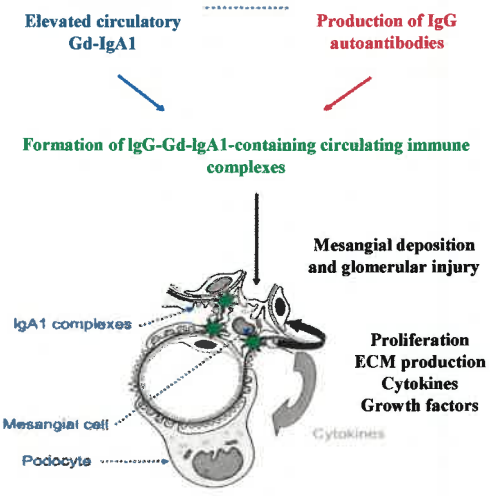
- Age
- Gender
- Ethnicity
- Family medical history

Source: Literature Review, Frost & Sullivan Analysis

Pathogenesis of IgA nephropathy (IgA)

- IgA nephropathy (IgAN) is currently the most common primary glomerulonephritis worldwide, with 20%-40% of patients progressing to end-stage renal disease within 20 years of diagnosis. At present, the pathogenesis of IgAN is not clear, and clinical treatment is mainly to control the progression, without specific treatment plan. Importantly, IgAN has been reported to account for 54.3% of primary glomerular diseases in China.

Pathogenesis of IgA



- IgA nephropathy (IgAN) is an autoimmune disease characterized by a multihit process determined by both genetic and environmental factors. IgA1 molecules contain O-glycans deficient in galactose (galactose-deficient IgA1; Gd-IgA1), whose levels are typically elevated in the blood of IgAN patients.
- These Gd-IgA1 molecules are recognized by Gd-IgA1-specific IgG autoantibodies and form pathogenic immune complexes with additional serum proteins (e.g., complement components). The levels of the autoantigen (Gd-IgA1) and corresponding IgG autoantibodies correlate in the blood of IgAN patients, indicating that elevated circulating Gd-IgA1 levels are associated with the production of Gd-IgA1-specific IgG autoantibodies.
- A subset of these circulating immune complexes deposits in the kidneys, activates mesangial cells, and induces glomerular injury.
- In IgAN, aberrant IgA1 glycosylation and immune complex deposition drive renal injury primarily through complement activation via the lectin and alternative pathways, rather than the classical pathway. This mechanism underscores complement as a therapeutic target to interrupt the inflammatory and fibrogenic cascade in IgAN.

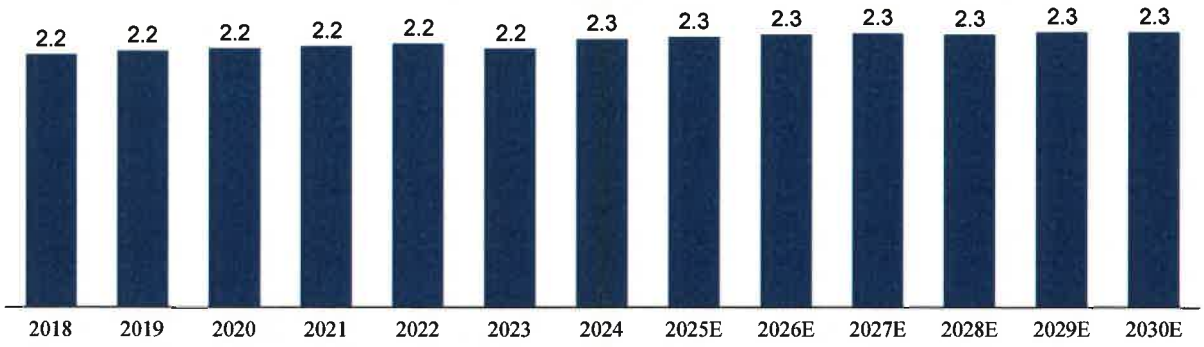
Source: Literature Review, Frost & Sullivan Analysis

Prevalence of IgA nephropathy in China, 2018-2030E

- There are a large number of IgA nephropathy patients in China, and its prevalence is growing from 2.2 million in 2018 to 2.3 million in 2024, with a CAGR of 1.0 %. With the increasing prevalence of IgA nephropathy, the number of IgA nephropathy patients in China is expected to reach 2.3 million in 2030 at a CAGR of 0.4%.

Prevalence of IgA nephropathy in China, 2018-2030E

Period	CAGR
2018-2024	1.0%
2025E-2030E	0.4%



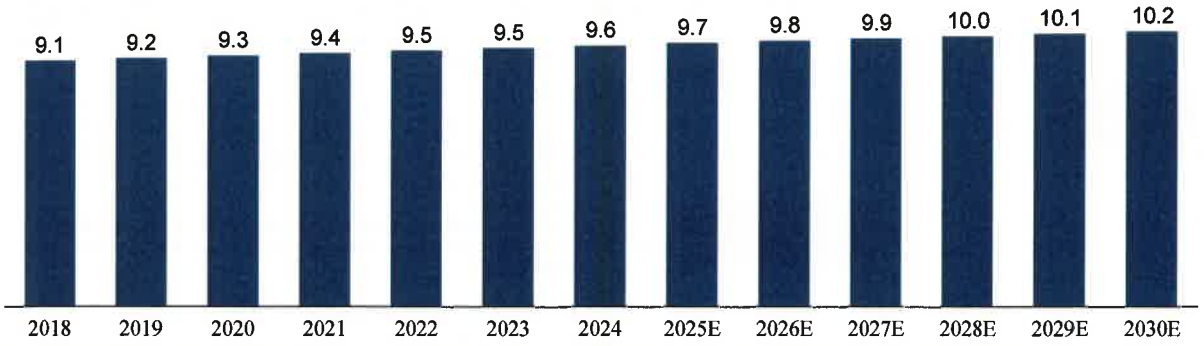
Source: Literature Review, Frost & Sullivan Analysis

Global Prevalence of IgA nephropathy, 2018-2030E

- There are a large number of IgA nephropathy patients in the world, and its prevalence is growing from 9.1 million in 2018 to 9.6million in 2024, with a CAGR of 1.0 %. With the increasing prevalence of IgA nephropathy, the number of IgA nephropathy patients in the world is expected to reach 10.2 million in 2030 at a CAGR of 0.9% from 2024 to 2030.

Global Prevalence of IgA nephropathy, 2018-2030E

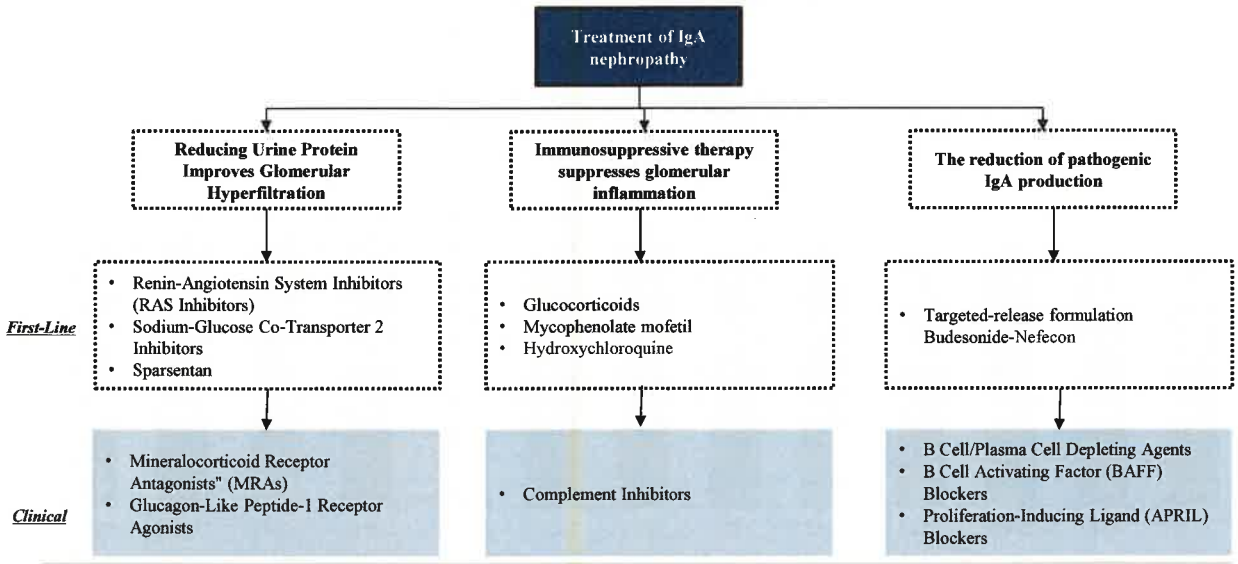
Period	CAGR
2018-2024	1.0%
2025E-2030E	0.9%



Source: Literature Review, Frost & Sullivan Analysis

Treatment Paradigm of IgA nephropathy in China

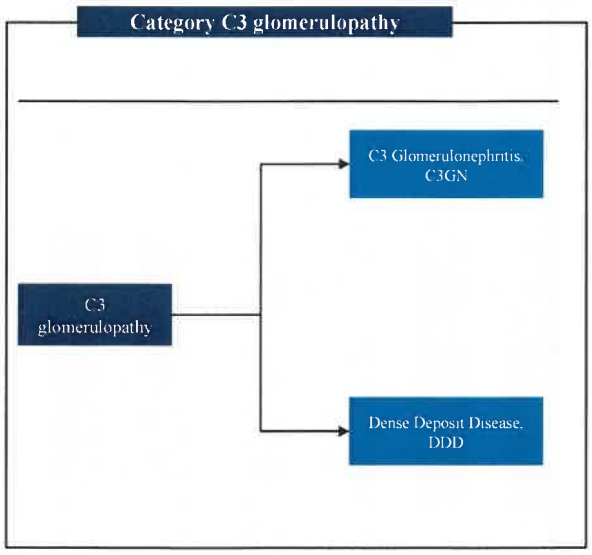
- IgA nephropathy is the most common primary glomerular disease worldwide, with most patients experiencing a slow progression of the condition, making it a leading cause of end-stage renal disease. Traditional treatments primarily focus on supportive care and immunosuppression, but the prognosis remains suboptimal, as a significant proportion of patients still progress to renal failure even with well-controlled proteinuria. In recent years, advances in understanding the disease's pathogenesis have shifted treatment strategies toward a multi-targeted comprehensive approach. This includes reducing pathogenic IgA, suppressing local renal inflammation, and supportive therapies. Currently, significant progress has been made in the development of new complement inhibitor drugs.



Source: Literature Review, Frost & Sullivan Analysis

Overview of C3 glomerulopathy

- C3 glomerulopathy (C3G) is a rare kidney disease and a type of glomerular disease. It is characterized by the abnormal deposition of complement C3 in the glomeruli, which leads to damage to the glomerular structure and function.
- The management of C3 glomerulopathy should integrate the underlying dysregulation of the complement pathway and employ a comprehensive therapeutic strategy encompassing foundational therapy, immunosuppression/complement inhibition, and symptomatic support. Conventional immunosuppressive agents (corticosteroids in combination with cytotoxic drugs) remain the first-line regimen. The management of C3 glomerulopathy is notably limited, as it lacks specific targeted drugs and relies on non-specific immunosuppressants with limited efficacy that fail to effectively halt disease progression, while complement inhibitors offer novel therapeutic alternatives for refractory cases. Future advancements in the elucidation of complement regulatory mechanisms are anticipated to facilitate the development of more precise, targeted therapeutic interventions.



Overview

1 Brief Introduction

- C3 glomerulopathy is a rare nephropathy mediated by complement overactivation and has two major subtypes, including dense deposit disease (DDD) and C3 glomerulonephritis (C3GN). Dense Deposit Disease (DDD) and C3 Glomerulonephritis (C3GN) are both subtypes of C3 glomerulopathy characterized by complement alternative pathway dysregulation and dominant C3 deposition in glomeruli, with DDD featuring electron-dense deposits within the glomerular basement membrane and C3GN showing irregular/granular deposits in the mesangium or subendothelium.

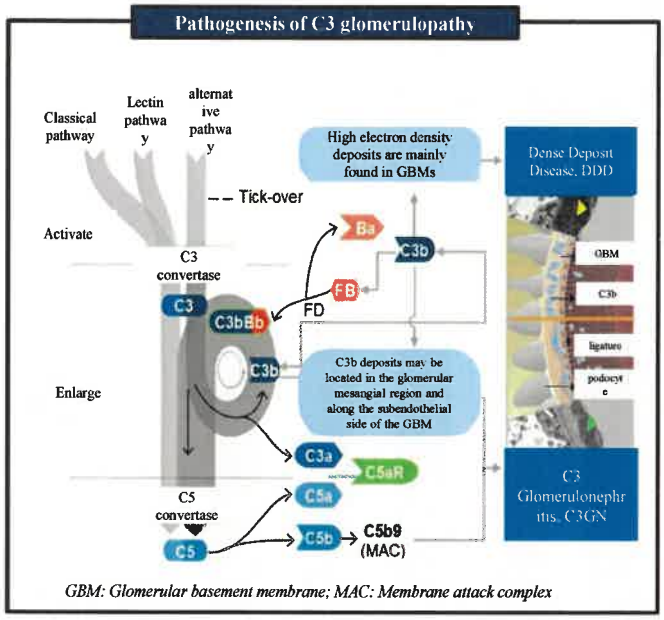
2 Diagnosis

- **Renal biopsy**: Pathologic examination shows significant deposition of C3 in the glomeruli and immunofluorescence is positive for C3, whereas immunoglobulins and other components of complement (e.g., C1q, C4) are usually negative.
- **Laboratory tests:** these include testing for serum complement levels (C3 is decreased, C4 is usually normal) and testing for autoantibodies such as C3 nephritis factor.
- **Genetic testing:** For patients with a family history or suspected genetic factors, testing for complement-related genes may be performed.

Source: Literature review, Frost & Sullivan Analysis

Pathogenesis of C3 glomerulopathy

- C3 glomerulopathy is a rare nephropathy mediated by complement overactivation and has two major subtypes, including dense deposit disease (DDD) and C3 glomerulonephritis (C3GN).



GBM: Glomerular basement membrane; MAC: Membrane attack complex

- In recent years, with the increasing clinical research on C3G, it is now recognized that overactivation of the complement paracrine pathway (AP) is the main pathogenesis of C3G. Excessive activation of the complement bypass pathway can lead to C3 cleavage in the glomerulus, triggering C3 deposition and inflammation, leading to kidney injury and failure.
- C3G has a highly heterogeneous clinical presentation, making its diagnosis challenging. Among the renal manifestations of C3G, hematuria and proteinuria are the most common. In addition, patients may also present with acute nephritis syndrome, nephrotic syndrome, or even manifestations such as decreased glomerular filtration rate (GFR) and elevated creatinine. In addition to renal involvement, some patients may also present with extra-renal manifestations such as retinal vitreous wart deposits and acquired partial lipodystrophy.

Source: Literature review, Frost & Sullivan Analysis

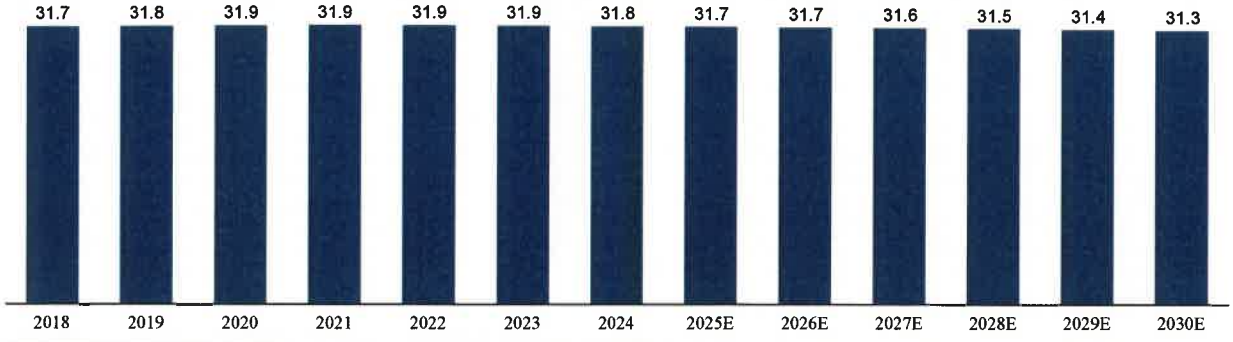
Prevalence of C3 glomerulopathy in China, 2018-2030E

- The prevalence of C3 glomerulopathy in China is growing from 31.7 thousand cases in 2018 to 31.8 thousand cases in 2024, with a CAGR of 0.1%. The number of C3 glomerulopathy patients in China is expected to reach 31.3 thousand cases in 2030 at a CAGR of -0.3% from 2024 to 2030.

Prevalence of C3 glomerulopathy in China, 2018-2030E

Period	CAGR
2018-2024	0.1 %
2025E-2030E	-0.3 %

Thousand



Source: Literature Review, Frost & Sullivan Analysis

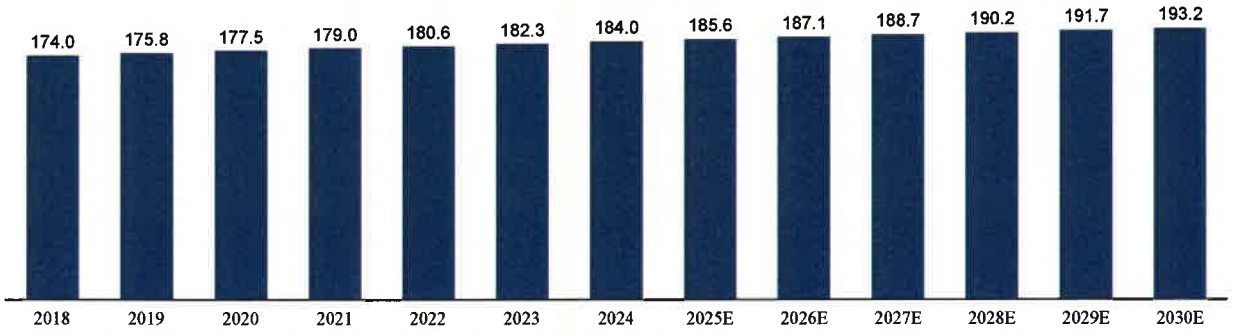
Global Prevalence of C3 glomerulopathy, 2018-2030E

- The global prevalence of C3 glomerulopathy is growing from 174.0 thousand cases in 2018 to 184.0 thousand cases in 2024, with a CAGR of 0.9%. The number of C3 glomerulopathy patients in the world is expected to reach 193.2 thousand cases in 2030 at a CAGR of 0.8% from 2024 to 2030.

Global Prevalence of C3 glomerulopathy, 2018-2030E

Period	CAGR
2018-2024	0.9%
2025E-2030E	0.8%

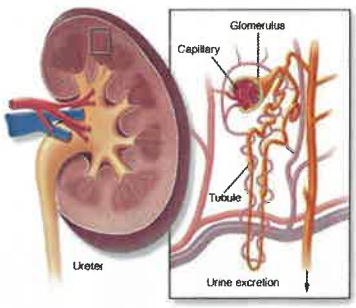
Thousand



Source: Literature Review, Frost & Sullivan Analysis

Overview of Lupus Nephritis (LN)

- Lupus nephritis is one of the most common and severe complications of systemic lupus erythematosus, an autoimmune disease. It is mainly caused by autoantibodies produced after abnormal activation of the immune system attacking kidney tissues. It is not only an important cause of disease progression and poor prognosis in patients with lupus erythematosus, but also one of the common triggers leading to end - stage renal disease (renal failure).



- In terms of pathogenesis, autoantibodies (such as antinuclear antibodies, anti-double-stranded DNA antibodies, etc.) in patients with systemic lupus erythematosus will combine with antigens in the body to form immune complexes. These complexes deposit in the glomeruli, renal tubules or renal interstitium of the kidneys along with the blood circulation, triggering local inflammatory reactions.

Treatment of Lupus Nephritis

- The treatment of lupus nephritis (LN) follows individualized and long-term continuous principles, with glucocorticoids and hydroxychloroquine as the foundational medications. Depending on the pathological type (e.g., Class I, II, III/IV, V, etc.) and disease characteristics, appropriate immunosuppressive regimens are selected, including combinations with mycophenolate mofetil, cyclophosphamide, tacrolimus, or the use of multitarget therapies and biologics (such as belimumab or rituximab).
- The treatment of lupus nephritis has obvious limitations. Current regimens mainly consist of hormones combined with immunosuppressants, but patients show significant differences in treatment response, with a high proportion of those who are drug-resistant or have partial responses. Long-term medication is associated with significant side effects, which affects the continuity of treatment.

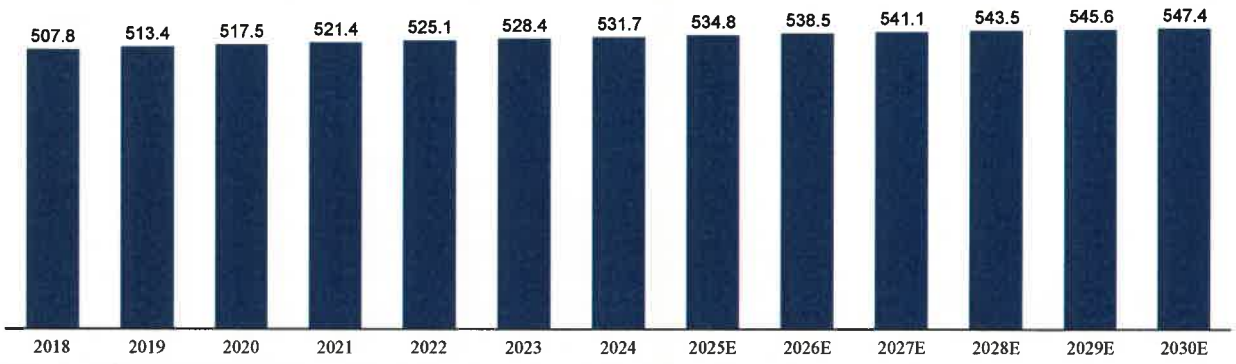
Source: Literature Review, Frost & Sullivan Analysis

Prevalence of Lupus Nephritis in China, 2018-2030E

- The prevalence of lupus nephritis is growing from 507.8 thousand in 2018 to 531.7 thousand in 2024, with a CAGR of 0.8%. The number of lupus nephritis patients in China is expected to reach 547.4 thousand in 2030 at a CAGR of 0.5% from 2024 to 2030.

Prevalence of Lupus Nephritis in China, 2018-2030E

Period	CAGR
2018-2024	0.8 %
2025E-2030E	0.5 %



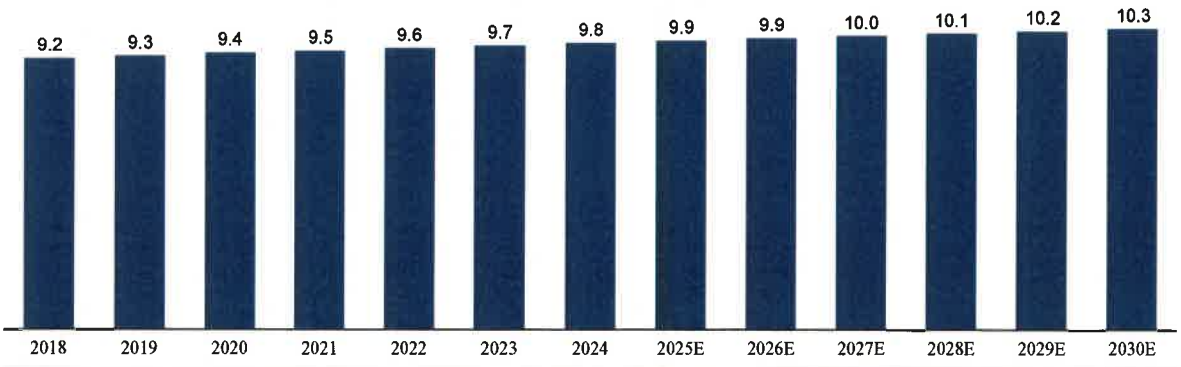
Source: Literature Review, Frost & Sullivan Analysis

Global Prevalence of Lupus Nephritis, 2018-2030E

- The global prevalence of lupus nephritis is growing from 9.2 million in 2018 to 9.8 million in 2024, with a CAGR of 0.9%. The number of lupus nephritis patients in the world is expected to reach 10.3 million in 2030 at a CAGR of 0.8% from 2024 to 2030.

Global Prevalence of Lupus Nephritis, 2018-2030E

Period	CAGR
2018-2024	0.9%
2025E-2030E	0.8%



Source: Literature Review, Frost & Sullivan Analysis

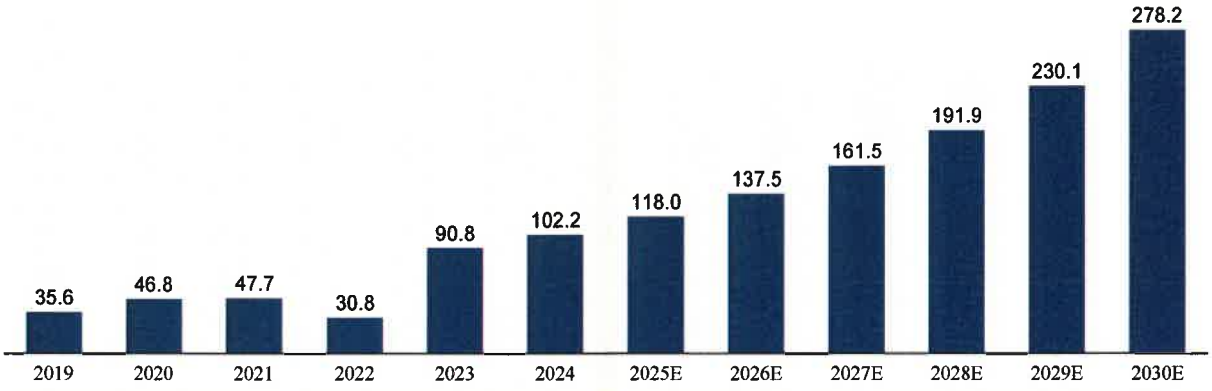
Market size of complement inhibitors

- After eculizumab (Soliris) was covered in NRDL in 2023, its market penetration rate significantly increased, driving further expansion of the complement inhibitor market in China. The market size of complement inhibitors in China will increase from 35.6 million in 2019 to 102.2 million in 2024, with a CAGR of 23.5%. With the expansion of new indications and the approval of new complement inhibitors, the market size of complement inhibitors in China is expected to further expand, and it will grow to 278.2million by 2030, with a CAGR of 18.2%.
- The global complement inhibitors market has garnered significant attention due to the intense R&D activity in complement therapeutics and the continuous realization of its commercial value. In 2024, the global complement inhibitors market reached US $7,241.7million, Driven by expanding indications, the emergence of new therapeutic modalities, and large unmet clinical needs, the global complement inhibitors market is projected to grow rapidly in the future.

Market size of complement inhibitors in China, 2019-2030E

Period	CAGR
2019-2024	23.5%
2025E-2030E	18.2%

Million RMB



Source: Literature Review, Frost & Sullivan Analysis

Updated

Competitive Landscape: Complement Inhibitors Approved by FDA

- As of the Latest Practical Date, FDA has approved four original C5 complement biologic inhibitors.

Drug Name	Brand Name	Target	Company	Indications	Approval Date	Sale Revenue in 2024 (million USD)
CROVALIMAB	PIASKY	C5	Roche Pharma	PNH	2024-06-20	19.7
POZELIMAB	VEOPOZ	C5	Regeneron Pharmaceuticals	CHAPLE	2023-08-18	NA
RAVULIZUMAB	ULTOMIRIS	C5	AstraZeneca	PNH /aHUS/MG/NMO	2018-12-21	3924.0
ECULIZUMAB	SOLIRIS	C5	AstraZeneca	PNH/aHUS/NMO/MG	2007-03-16	2588.0

*Note: Approval date refers to the first approval date; as of May 18, , 2026

Source: FDA, Frost & Sullivan Analysis

Updated

Competitive Landscape: Complement Inhibitors Approved by FDA

- As of the Latest Practical Date, FDA has approved one original C3 complement inhibitors.
- Currently, there is no approved C3 complement inhibitors in China.

Drug Name	Brand Name	Target	Company	Indications	Approval Date	Sale Revenue in 2024 (million USD)
Pegcetacoplan	SYFOVRE	C3	Apellis Pharmaceuticals	Geographic Atrophy (GA)	2023-02-17	611.9
Pegcetacoplan	EMPAVELI	C3	Apellis Pharmaceuticals	Paroxysmal Nocturnal Hemoglobinuria (PNH)	2021-05-14	98.1

*Note: Approval date refers to the first approval date; as of May 18, 2026

Source: FDA, Frost & Sullivan Analysis

Updated

Competitive Landscape:Complement Inhibitors Approved by NMPA

- As of the Latest Practical Date, NMPA has approved four complement inhibitors. Three of the complement inhibitors are biologics targeting C5.

Drug Name	Brand Name	Target	Company	Indications	Approval Date	Monthly treatment costs(RMB)	Whether to enter NRDL
Iptacopan	Fabhalta	CFB	Novartis Pharma	PNH/C3G	2024-04-24	~18,900	Yes
Ravulizumab	Ultomiris	C5	AstraZeneca	AChR-gMG	2025-04-15	NA	No
Crovalimab	Piasky	C5	Roche Pharma	aHUS/PNH	2024-02-06	NA	No
Eculizumab	Soliris	C5	AstraZeneca	PNH/aHUS/AChR-gMG	2018-09-04	~4,600	Yes

*Note: Approval date refers to the first approval date; as of May 18, 2026; Depending on the patient's condition, the dosage of medication used varies and the monthly cost of treatment varies.

Source: NMPA, Frost & Sullivan Analysis

Updated

Competitive landscape of global complement inhibitors pipeline: Indications that are under development

- As of the Latest Practical Date, according to the clinicaltrials.gov,there are five complement biologic inhibitors targeting C5 or C3 entering clinical trials globally.

Drug Name/Code	Target	Company	Clinical Stage	Indications	First Posted Date
IAB-101	C5	ImmunAbs	I/II	Generalized Myasthenia Gravis	2025-10-26
KRIYA-825	C5&C3	Kriya Therapeutics, Inc.	I/II	Geographic Atrophy	2025-01-03
KP104	C5&CFH	Kira Pharma	II	PNH,C3G,IgA	2022-08-24
CAN106	C5	CARE Pharma Shanghai Ltd	I	PNH	2021-10-14
NGM621	C3	NGM Biopharmaceuticals	II	Geographic Atrophy	2020-07-10

*Note: First posted date: 首次公示日期, as of May 18, 2026

Source: clinicaltrials.gov, Frost & Sullivan Analysis

Competitive landscape of complement inhibitors pipeline in China: Indications that are under development

- As of the Latest Practical Date, there are six complement biologic inhibitors targeting C5 or C3 entering clinical trials in China.

Drug Name/Code	Target	Company	Clinical Stage	Indications	First Posted Date
LP-005	C5&C3b	LongBio Pharma	II	PNH	2024-07-22
			II	C3G, anti-GBM disease, LN, MPGN, and TMA.	2026-01-22
			I	Periodontitis	2026-04-23
CG001	C3b	Shanghai ComGen Biopharmaceutical Co., Ltd	II	PNH	2026-05-07
EA5	C5	Lan-yi Therapeutics, Ltd	I	antiphospholipid syndrome	2026-05-13
			I	PNH	2025-01-03
Pozelimab	C5	Regeneron Pharmaceuticals	III	gMG	2024-05-25
KP104	C5&CFH	KiraPharma	II	PNH,C3G,IgA	2022-08-24
CAN106	C5	CARE Pharma Shanghai Ltd.	I/II	PNH	2022-02-10

*Note: Approval date refers to the first approval date; as of May 18, 2026

Source:CDE,Frost & Sullivan Analysis

Growth drivers of complement inhibitors

Indication expansion involves penetration from rare diseases to common diseases	• Initially, complement inhibitor drugs focused primarily on rare diseases such as paroxysmal nocturnal hemoglobinuria (PNH), atypical hemolytic uremic syndrome (aHUS), and neuromyelitis optica spectrum disorder (NMOSD). However, in recent years, their indication landscape has expanded rapidly, from rare diseases to include both rare conditions (C3 glomerulopathy) and common chronic diseases such as IgA nephropathy and age-related macular degeneration (AMD). The expansion of indications has increased the potential patient population, driving continuous growth in the market size of complement drugs and providing China's market with extensive disease coverage and commercialization opportunities.
The R&D of new targets of complement inhibitors is advancing rapidly	• As the global commercial value of complement drugs becomes increasingly recognized and the demonstration effect of innovative products has emerged, domestic pharmaceutical companies are accelerating their entry into new target areas. The field has witnessed growing diversification in drug modalities, evolving from early monoclonal antibodies to a broader range of therapeutic approaches. These advances in target diversity and R&D efficiency are enabling Chinese companies to break through the international monopoly, achieve domestic substitution, and enhance competition in the clinical market.
Policy support and medical insurance mechanisms drive market penetration	• China's National Reimbursement Drug List (NRDL) continues to expand, with a clear trend toward the inclusion of innovative drugs. The 2024 NRDL adjustment has focused on new and innovative therapies, with several such drugs successfully entering the list through price negotiations. This will significantly reduce patients' economic burden and improve clinical accessibility. Meanwhile, the NMPA has established green channels — including priority review and conditional approval — for innovative drugs targeting rare and major diseases, which has greatly shortened the time-to-market for new therapies. It is expected that both domestic and imported complement drugs will achieve accelerated market access in the future, providing strong institutional support for sustained market growth.

Source: Frost & Sullivan Analysis

Future trends of complement inhibitors(1/2)

Continuous innovation in multi-target and combination therapy R&D	• Currently, C5-targeted drugs dominate the complement inhibitor market. However, with the deepened understanding of the complement cascade activation pathway, upstream target drugs such as C3 and MASP-2 are gradually emerging. In particular, C3 inhibitors, as the core hub of complement activation, possess the potential for broader indication coverage and have become a research hotspot for complement drug development in China. At present, multiple Chinese enterprises are actively advancing innovative drug pipelines targeting C3, CFB, C5 dual targets, etc. In the future, multi-target combined inhibition strategies will also become a new R&D direction, which can not only enhance therapeutic effects but also reduce immune side effects caused by long-term single-target inhibition.
Breakthroughs in indication expansion and cross-disciplinary therapeutic areas	• Complement drugs, originally used for rare hematological and neurological diseases such as PNH, aHUS, and NMOSD, are accelerating their penetration into common diseases (e.g., IgA nephropathy, C3 glomerulopathy) and autoimmune diseases, with their indication landscape continuously expanding. In the future, as the mechanisms of abnormal complement activation in various diseases are further explored, complement drugs are expected to be applied cross-disciplinarily to emerging fields including ophthalmology, rheumatology, nephrology, neurological diseases, transplant rejection, and tumor immune microenvironment regulation. Notably, China has a huge patient base for diseases like IgA nephropathy, with significant unmet clinical needs, serving as a key driver for potential market growth.
Market penetration driven by optimization of treatment costs and improvement of accessibility	• Multiple products targeting C3, CFB, C5 and other targets have entered clinical phases II-III, with some reaching the international synchronous R&D level. It is expected that in the next few years, the first batch of domestic complement inhibitors is expected to be approved for marketing, breaking the monopoly of imported drugs, which will significantly reduce treatment costs, improve the medication accessibility of domestic patients, and enhance the influence of Chinese enterprises in the global complement drug market. At the same time, with the continuous advancement of national medical insurance negotiations, local medical insurance policies, and special security mechanisms for rare diseases, the medical insurance coverage and reimbursement ratio of complement drugs will be gradually improved.

Source: Frost & Sullivan Analysis

Future trends of complement inhibitors (2/2)

Multi-target drugs have more potential efficacy advantages compared with single-target complement inhibitors	• The development trend of multi-target complement inhibitors showing superior efficacy potential compared to single-target ones is becoming increasingly clear: by acting on multiple key nodes in the complement cascade simultaneously, they can more comprehensively block the complex pathological mechanisms of diseases. For instance, in conditions like paroxysmal nocturnal hemoglobinuria, they can inhibit both upstream C3-mediated extravascular hemolysis and downstream C5-related terminal pathway effects, making up for the limitation of single-target drugs that fail to cover all pathological links. This multi-dimensional intervention approach not only enhances the overall therapeutic effect but also reduces the risk of drug resistance caused by the activation of alternative pathways after a single pathway is blocked, ensuring more durable and stable efficacy. Meanwhile, tailored to the specific pathological characteristics of different diseases, multi-target drugs can flexibly combine targets to achieve more precise individualized treatment, expanding application scenarios in multiple fields such as neuroimmunology and kidney diseases.
China's complement inhibitor sector witnessing surge in market transactions	• Although transaction frequency in China's complement inhibitor sector has not yet matched that of high-profile areas like oncology drugs, the field has gradually gained momentum alongside rising global interest, demonstrating clear signs of market warming. The landmark acquisition of Alexion by AstraZeneca significantly boosted global attention on complement inhibitors, with core products like eculizumab subsequently expanding their market presence in China through national reimbursement negotiations. While domestic pharmaceutical companies have not executed cross-border mergers or acquisitions of comparable scale, they have actively expanded their presence through licensing deals and R&D collaborations.

Source: Frost & Sullivan Analysis

Appendix

- According to Frost & Sullivan, LP-003 has the most advanced clinical stage development among the next generation anti-IgE biologic drug candidates in the world.
- According to Frost &Sullivan, allergic diseases have a huge patient base, and the demand for improved quality of life is expected to bring huge market growth potential
- LP-003, is a novel anti-IgE antibody featuring novel sequencing with proprietary patent, and best-in-class potential.
- LP-005, is a potential globally leading bi-functional antibody fusion protein targeting C5 and C3b complement.
- The complement system is a self-protection mechanism of the human body. Complement activation is carried out under the strict control of multiple regulatory proteins. It assists immune cells or other immune molecules to exert immune effects without damaging their own tissue cells, helps the body resist pathogen invasion and infection, and plays a key role in maintaining health and tissue homeostasis.
- Excessive and abnormal activation of the complement system can induce inflammatory responses and cause autoimmune damage, and hence is involved in the occurrence and development of various diseases, such as PNH, C3G, IgAN and LN. Complement inhibitors work by targeting key proteins of the complement system (such as C3, C5, Factor D/B) to block their activation pathways (classical, lectin, alternative), which precisely inhibit excessive complement activation. For example, C5 inhibitors (such as eculizumab) prevent the cleavage of C5 into pro-inflammatory factor C5a and membrane attack of complex C5b, while C3 inhibitors (such as pegcetacoplan) block the central node of the complement cascade which is the C3 convertase, and reduce inflammation and tissue damage. Some drugs can also mimic natural regulatory proteins (CD55/CD59) and protect host cells from misdirected attacks. However, a single complement inhibitor may not completely block the progression of disease due to its limited activity, such as C5 antibody alone may not sufficiently block AP and the deposition of C3b on the cell surface produced by C3 activation. Whereas CFB/CFD inhibitors mainly block AP, and MASP-2 can only block LP. However, given the large amount of C3 protein present in the blood (0.8-1.8 mg/mL), the activity of C3 inhibitors remains to be improved.
- The complement system is a self-protection mechanism of the human body and an important innate immune signaling pathway that mediates multiple inflammatory pathways. The abnormal activation of the complement system is involved in the occurrence and development of various diseases.
- The complement system is an important component of innate immunity. Composed of more than 30 proteins, it plays a key role in anti-infective

Appendix

- The complement system is an important component of innate immunity. Composed of more than 30 proteins, it plays a key role in anti-infective defense and immune regulation. Abnormal activation or functional defects of the complement system can lead to a variety of diseases, including hematological diseases (such as PNH) and complement-mediated kidney diseases (such as IgAN, C3G, LN). In recent years, with the deepening understanding of the complement system, a variety of complement-targeted drugs have been approved for marketing, such as C5 inhibitors (e.g., eculizumab, ravulizumab), C3 inhibitors (e.g., pegcetacoplan), and Factor B inhibitors (e.g., iptacopan). At present, the global complement inhibitor market has reached around billions of dollars, but there are still significant unmet clinical needs in existing related diseases.
- IgAN is also the most common primary glomerular disease in the world with highest incidence rate in Asia.
- Newer medications that directly target IgAN disease, such as Nefecon and Sparsentan have been granted accelerated approval by the FDA. However, no biological drugs for IgAN have been approved for marketing yet.
- According to Frost & Sullivan, approximately 40% and 37% of the population are affected by one or more allergic disorder globally and in China.
- However, the effectiveness of these medications is limited. According to Frost & Sullivan, approximately 60% of seasonal AR patients having limited efficacy after receiving 2nd-generation nasal or oral antihistamine and nasal glucocorticoids treatments. Moreover, approved treatment options and effectiveness of currently available drugs targeting moderate to severe AR patients are limited.
- In the interim analysis results of the Phase II clinical trial for CSU, LP-003 demonstrated superior clinical efficacy (fast onset of action, good efficacy and long-acting) and best-in-class potential compared to omalizumab in the treatment of CSU.
- Core Product LP-003: a novel anti-IgE antibody, with head-to-head clinical study showing best-in-class potential, fast onset of action, good efficacy, long-acting and lower dosage.
- LP-005, as the first candidate discovered and developed from our unique platform, is a potential globally leading bifunctional complement antibody fusion protein targeting complement-mediated autoimmune diseases, showing encouraging clinical results.

Appendix

- Our Core Product with head-to-head clinical study showing best-in-class potential, fast onset of action, good efficacy, long-acting and lower dosage.
- LP-005, as the first candidate discovered and developed from our unique platform, is a potential globally leading bi-functional complement antibody fusion protein targeting complement-mediated autoimmune diseases, showing encouraging clinical results.
- Core Product LP-003: a novel anti-IgE antibody, with head-to-head clinical study showing best-in-class potential, fast onset of action, good efficacy, long-acting and lower dosage.
- LP-003 demonstrates superior clinical efficacy, maintains a leading position in clinical development progress, and exhibits potential to become a best-in-class therapy
- In the Phase II clinical trial for CSU, when compared head-to-head with omalizumab and taking into account data from the Phase I clinical trial, LP-003 showed a faster onset of action, better efficacy, long-acting and with a lower dosage, and therefore exhibits potential to become a best-in-class therapy.
- Based on the published data, LP-003 demonstrated superior clinical efficacy and best-in-class potential compared to omalizumab in the treatment of CSU.
- Overall, as validated by data from the head-to-head comparison with omalizumab as well as the Phase I clinical trial, LP-003 has shown a faster onset of action, better efficacy, long-acting and with a lower dosage, and exhibits potential to become a best-in-class therapy.
- Phase III clinical trial for seasonal AR is leading in its clinical development process.
- LP-005, as the first candidate discovered and developed from our Bi-functional Antibody Development Platform, is a potential globally leading bi-functional complement antibody fusion protein targeting complement-mediated autoimmune diseases, showing encouraging clinical results.
- LP-005 is the first product developed by our Bi-functional Antibody Development Platform. The result of pre-clinical studies and Phase II clinical trial shows best-in-class potential.
- Our Core Product, LP-003, is a next generation innovative anti-IgE antibody with novel sequencing and best-in-class potential, which is targeted to treat allergic diseases, including AR, CSU, allergic asthma and other allergic diseases. Our Key Product, LP-005, is a newly designed and potential globally leading bi-functional antibody fusion protein targeting C5 and C3b complement used for PNH, complement-mediated kidney diseases, which includes IgAN, C3G, LN, as well as gMG, MAG-PN and ALS.

Appendix

- LP-003 is a next generation innovative anti-IgE antibody with novel sequencing and best-in-class potential, which is targeted to treat allergic diseases, including AR, CSU, allergic asthma, CRSwNP and food allergy.
- In the interim analysis results of the Phase II clinical trial for CSU, LP-003 demonstrated superior clinical efficacy (fast onset of action, good efficacy and long-acting) and best-in-class potential compared with omalizumab in the treatment of CSU. In addition, LP-003 showed favorable efficacy and safety profile in its Phase II clinical trial.
- Based on thepublished data below, LP-003 demonstrated superior clinical efficacy (lower dosage, fast onset of action, good efficacy and long-acting) and best-in-class potential compared to omalizumab in the treatment of CSU.
- LP-005 is a potential globally leading bi-functional complement antibody fusion protein targeting complement-mediated autoimmune diseases, showing encouraging clinical results.
- Anti-IgE monoclonal antibody achieves blockade of allergic reactions in a variety of indications through neutralization of free IgE, down-regulation of FceRI receptor density, and inhibition of inflammatory mediator release, and occupies an important position in the treatment of a variety of allergic diseases.
- Treatment Paradigms for AR in China

AR is managed through chemical drugs, allergen-specific immunotherapy ("AIT")biologics, and surgery. Chemical drugs, such as antihistamines, glucocorticoids, and leukotriene receptor antagonists, are recognized as first-line therapeutic options. Antihistamines relieve sneezing, nasal itching, and rhinorrhea; glucocorticoids suppress inflammation; and leukotriene receptor antagonists inhibit leukotriene-induced inflammatory response. These drugs are fast-acting, widely accessible, and support individualized treatment. However, they primarily offer symptomatic control, often leading to relapse after discontinuation or dosage reduction, Long-term use may cause side effects, such as mucosa dryness with glucocorticoids and rebound congestion with decongestants. In moderate-to. Severe cases or those with type 2 inflammation, drugs alone may be insufficient.

- Treatment Paradigms for CSU in China

The primary treatment for CSU is medication, with antihistamines as first-line agents. By blocking H1 receptors, they reduce histamine-mediated vasodilation, vascular permeability, and pruritus. Second-generation antihistamines offer rapid relief, fewer central side effects, and convenient dosing. However, as antihistamines do not directly inhibit the Th2 response, some CSU patients will suffer failure of antihistamine therapy due to the persistence of the Th2 cell response.

- Biologic agents such as omalizumab, target lgE, prevents its binding to mast cells and basophils and thereby suppresses allergic inflammation. Omalizumab is effective in refractory CSU patients and improves quality of life. Anti-IgE antibodies have become the first choice of drug for third-line therapy in CSU patients who are unresponsive or intolerant to antihistamines. However, it requires subcutaneous injection, which may limit accessibility for some patients.

Appendix

- Treatment Paradigms for CRSwNP in China

The treatment of CRSwNP needs to be stratified according to the condition. Medication is the basic option, and nasal glucocorticoids are preferred, as they are non-invasive and have fewer side effects, although they have a slower onset of action and a limited therapeutic effect in patients with severe disease. Antihistamines alleviate allergy-related symptoms by blocking histamine H1 receptors, but their effects on nasal polyp reduction and sinus inflammation control are weak, making them suitable only for patients with combined allergies. Macrolide antibiotics reduce inflammation through non-antimicrobial anti-inflammatory and immunomodulatory measures, but their long-term use may increase bacterial resistance, and they are ineffective in reducing the size of larger polyps. Chemical drugs for CRSwNP primarily relieve symptoms, They are difficult to use for complete control of polyp growth and sinus inflammation, have limited efficacy in refractory cases, tend to cause local or systemic side effects with long-term use, and have a high recurrence rate after discontinuation.

- Surgical treatment is based on functional endoscopic nasal surgery, which can rapidly improve nasal congestion and olfactory disorders by removing polyps and opening the sinus openings. It is characterized by less trauma and faster recovery, but the post-operative recurrence rate is relatively high-with the overall recurrence rate as high as 35%-38%, and for some types of patients. the recurrence probability even reaches 98%, Moreover, the cause of the disease can't be cured at all, so it needs to be combined with perioperative medication to reduce the risk of recurrence, and a small number of patients may have complications.
- Biologic therapy is an emerging precision therapy for severe refractory CRSwNP,Anti-IL-5/IL-5R drugs can reduce inflammation by targeting eosinophils, significantly reducing polyp size and improving symptoms. Subcutaneous injections have fewer side effects; however they are expensive and require long-term use, Anti-lgE drugs are suitable for patients with severe allergies, as they indirectly inhibit polyp growth, but their effectiveness is limited in cases of non-allergic polyps.
- Treatment Paradigms for Food Allergy in China

The primary treatments for food allergies include allergen avoidance, medication, and allergen-specific immunotherapy. Allergen avoidance is fundamental and effective for mild cases by preventing immune activation, but complete avoidance can be difficult in daily life and may affect quality of life.

- Food allergy is a condition caused by an abnormal immune response to dietary components, usually proteins, which can be triggered through IgE-mediated, non-lgE mediated, or a combination of both mechanisms. Clinical manifestations are diverse, affecting the skin, gastrointestinal, respiratory, and cardiovascular systems, and it is one of the leading causes of allergic shock. The prevalence of food allergies is significantly higher in children aged six to 1l years old in China compared to adults, and incidences are on the rise. Studies have shown that factors such as early-life environment, gut microbiota, dietary habits, and mother-child immune interactions play a key role in the failure of oral tolerance development

Appendix

- Medication is the first-line approach for acute reactions. Antihistamines, especially second-generation types, rapidly relieve mild to moderate symptoms with fewer side effects, but they are ineffective in severe cases. Glucocorticoids control inflammation in moderate to severe reactions but pose risks with long-term use. Epinephrine is life-saving in anaphylaxis by reversing respiratory and circulatory failure, but must be administered immediately and does not prevent recurrence.
- Allergen-specific immunotherapy modulates the immune response through long-term, low-dose allergen exposure. It shifts the immune balance from Th2 to Th1, suppresses IgE production, and enhances regulatory T cells, offering the potential for long-term remission or even a cure. However, it requires extended treatment, strict monitoring, and high patient adherence.
- Biologics can effectively prevent food allergies through targeted regulation of immune mechanisms. Anti-IgE biologics inhibit allergic reactions at the source by preventing IgE from binding to mast cells, which can significantly reduce the chance of sensitization and effectively lower the risk of developing allergies.
- Autoimmune diseases occur when the immune system mistakenly attacks the body's own tissues and organs. Instead of defending the body from external threats, the immune system starts to destroy its own cells, leading to inflammation, pain, and damage. Autoimmune diseases can affect almost all parts of the body, including the joints, muscles, skin and organs. The pathogenesis of autoimmune diseases is complex and involves multiple factors, including genetic, environmental, and immune system dysregulation.
- According to a study, autoimmune diseases cumulatively affect 5% to 10% of the industrialized world population. In recent years, accumulating data confirm that the burden of autoimmune diseases is rising in the developing world as well, making them a ubiquitous global phenomenon that is suspected to further rise in the upcoming decades. Autoimmune diseases can have a serious impact on the health and quality of life of patients. These diseases are often chronic and require long-term treatment and health management.
- Uncontrolled complement activation can cause or contribute to glomerular injury in multiple kidney diseases. In complement-mediated renal kidney, multiple complement pathways have been shown to exhibit aberrant activation.
- However, due to the change of industry characteristics, specifically the cost advantage offered by CDMO partners.
- At present, the overall control level of asthma in China is not satisfactory. According to a study on progress and challenges in asthma management in China, the overall asthma control rate in urban areas of 30 provinces and cities in China in was only 28.5%, and the uncontrolled rate of severe asthma is as high as 44%.
- However, according to expert consensus on practical aspects in the treatment of chronic urticaria in 2023, up to 42.2% of patients do not achieve effective control of their symptoms after receiving one year of treatment with 2nd-generation antihistamines.

Appendix

- Global AR drug market: The number of patients with AR worldwide is relatively stable. According to literature, the global prevalence rate of AR is approximately 18.1%, with a total of about 1.4 billion patients globally. Currently, no biologics have been approved by the FDA; only Stapokibart has obtained approval from the NMPA. Globally, chemical drugs remain the primary treatment method, and there is no approval of new-generation drugs that can cover the global market in the short term in the future. With the improvement of patients' health awareness, the treatment rate of AR is still on the rise. However, due to the continuous price reductions of allergic rhinitis treatment drugs and the slowdown in the growth of the treatment rate, the projected CAGR of the global AR drug market is lower than the historical CAGR.
- China's AR drug market: The number of patients with AR in China is relatively stable. According to literature, the prevalence rate of AR in China is approximately 17.6%, with a total of about 240 million patients nationwide. Previously, the main treatment method for AR in China was chemical drugs. With the approval of Stapokibart by the NMPA in 2025, biologics will gradually become one of the primary treatment options. As patients' health awareness improves, the treatment rate of AR is still on the rise. Along with the gradual increase in the penetration rate and adherence of biologics, which have higher treatment costs and better efficacy, as well as the continuous growth in the treatment rate of AR patients, the China's AR durg market will experience rapid growth, resulting in a projected CAGR higher than the historical CAGR.
- Global CSU drug market: The number of patients with CSU worldwide is relatively stable. According to literature, 75% of global urticaria patients are CSU patients, totaling approximately 69.7 million CSU patients worldwide. Currently, the main biologic used for CSU globally is Omalizumab, which has been approved for over 10 years, and the global treatment landscape is relatively stable. With the improvement of patients' health awareness, the treatment rate of CSU is still on the rise. However, due to the continuous price reductions of CSU treatment drugs and the slowdown in the growth of the CSU treatment rate, the projected CAGR of the global CSU drug market is lower than the historical CAGR.
- China's CSU drug market: The number of patients with CSU in China is relatively stable. According to literature, the prevalence rate of CSU in China is approximately 1.9%, with a total of about 26.1 million CSU patients nationwide. Previously, the primary treatment for CSU in China was chemical drugs. In 2022, Omalizumab was approved by the NMPA for CSU treatment and will gradually become one of the important treatment methods for CSU. With the improvement of patients' health awareness, the treatment rate of CSU is still on the rise. Along with the gradual increase in the penetration rate and adherence of biologics which have higher treatment costs and better efficacy, as well as the continuous growth in the treatment rate of CSU patients, the China's CSU drug market will experience rapid growth, resulting in a projected CAGR higher than its historical CAGR.

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- Global allergic asthma drug market: The number of patients with allergic asthma worldwide is relatively stable. According to literature, there are approximately 800 million asthma patients worldwide, of which 65% are allergic asthma patients, totaling about 520 million allergic asthma patients. Currently, multiple biologics have been approved for use globally, and many of these drugs have been approved for more than 5 years, making the global treatment landscape relatively stable. With the improvement of patients' health awareness, the treatment rate of allergic asthma is still on the rise. However, due to the continuous price reductions of allergic asthma treatment drugs and the slowdown in the growth of the allergic asthma treatment rate, the projected CAGR of the global allergic asthma drug market is lower than the historical CAGR.
- China's allergic asthma drug market: The number of patients with allergic asthma in China is relatively stable. According to literature, the prevalence rate of asthma in China is approximately 4.2%, of which 65% are allergic asthma patients, totaling about 45 million allergic asthma patients nationwide. Previously, the primary treatment method for allergic asthma in China was chemical drugs. In the past three years, multiple biologics have been successively approved, and biologics will further expand their application scope among allergic asthma patients. With the improvement of patients' health awareness, the treatment rate of allergic asthma is still on the rise. Along with the gradual increase in the penetration rate and adherence of biologics which have higher treatment costs and better efficacy, as well as the continuous growth in the treatment rate of allergic asthma patients, the China's allergic asthma drug market will experience rapid growth, resulting in a projected CAGR higher than its historical CAGR.
- Global CRSwNP drug market: The number of patients with CRSwNP worldwide is relatively stable. According to literature, the global prevalence rate of CRS is approximately 10%, of which around 30% are CRSwNP patients, totaling about 282 million CRSwNP patients globally. Currently, multiple biologics have been approved worldwide, and their approval duration has reached 4 to 5 years, making the global treatment landscape relatively stable. With the improvement of patients' health awareness, the treatment rate of CRSwNP is still on the rise. However, due to the continuous price reductions of CRSwNP treatment drugs and the slowdown in the growth of the CRSwNP treatment rate, the projected CAGR of the global CRSwNP drug market is lower than the historical CAGR.

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- China's CRSwNP drug market: The number of patients with CRSwNP in China is relatively stable. According to literature, the prevalence rate of CRSwNP in China is approximately 1.5%, with a total of about 20.9 million CRSwNP patients nationwide. Previously, the primary treatment method for CRSwNP in China was chemical drugs. All biologics for this condition have been approved in the past two years and will gradually become one of the important treatment methods for CRSwNP. With the improvement of patients' health awareness, the treatment rate of CRSwNP is still on the rise. Along with the gradual increase in the penetration rate and adherence of biologics which have higher treatment costs and better efficacy, as well as the continuous growth in the treatment rate of CRSwNP patients, the China's CRSwNP drug market will experience rapid growth, resulting in a projected CAGR higher than its historical CAGR.
- Compared with therapeutic drugs for allergic diseases targeting other targets, LP003 demonstrates differentiated advantages in mechanism of action and dosage. Its IgE-targeted mechanism is clear and direct, reducing efficacy fluctuations caused by complex mechanisms. Meanwhile, the lower dosage supports reducing the potential medication burden on patients. Compared with Omalizumab, a similar anti-IgE antibody drug, LP003 achieves further optimization in efficacy and patient compliance. It not only exhibits higher affinity for IgE, superior blocking effect, and more competitive clinical efficacy, but also effectively simplifies the treatment process through lower dosage, less frequent administration, and more convenient medication methods, thereby improving patients' long-term medication compliance.
- Compared with drugs targeting other pathways, the IgE-focused mechanism of action of LP003 is clear and direct, eliminating the need for reliance on complex indirect pathway regulation and providing mechanistic support for stable efficacy. Head-to-head studies have confirmed that LP003 holds significant clinical advantages over the anti-IgE antibody omalizumab. It not only has higher affinity for IgE and better blocking effect, leading to improved control of clinical symptoms, but also features lower dosage and less frequent administration. This enhances medication convenience while effectively improving patients' compliance with long-term treatment.
- In the field of allergic asthma treatment, LP003 presents distinct differentiated advantages in dosing regimen compared to both anti-IgE antibodies (same target) and therapeutic drugs with different mechanisms of action. Its long-acting design with administration once every 3 months not only significantly reduces the frequency of patients' hospital visits for injections but also minimizes missed doses caused by short intervals and frequent administrations. This effectively improves patients' long-term treatment compliance and better meets the clinical demand for convenient and sustainable treatment solutions.

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- Compared with some competing drugs targeting other pathways such as IL-4Rα and TSLP, the mechanisms of action of LP003 is the anti-IgE pathway. IgE-mediated immune-inflammatory response is one of the key pathological links in the pathogenesis of CRSwNP. This mechanism directly targets the core drivers of the disease, ensuring definite therapeutic effects for CRSwNP.
- Given the stable prevalence rates of C3G, the projected demographic contraction in China will result in a downward trend in the patient populations.
- According to Chinese guideline for diagnosis and treatment of allergic rhinitis (2022), in the treatment of allergic rhinitis, pharmacotherapy and allergen-specific immunotherapy ("AIT") are the first-line treatment options for symptomatic treatment and etiological treatment, respectively. Pharmacotherapy is usually used during the symptomatic episode of allergic rhinitis to relieve patients' symptoms; while AIT is suitable for patients whose conditions are uncontrollable with conventional pharmacotherapy, those who wish to avoid long-term medication, and those who need to prevent the onset of related diseases. Surgery is an adjuvant treatment for allergic rhinitis, only applicable to patients with allergic rhinitis whose conditions are uncontrollable with treatments such as pharmacotherapy and immunotherapy, or those who cannot receive long-term pharmacotherapy.
- According to Frost & Sullivan, it is industry norm for biopharmaceutical companies to outsource such ancillary works to third-party service providers to allow them to focus on their core research and development and improve efficiency.

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- Chemical drugs, including glucocorticoids, β2-agonists, and leukotriene antagonists, remain the cornerstone of respiratory therapy. While glucocorticoids provide potent local anti-inflammatory effects and β2-agonists offer rapid airway relaxation, both require careful management to avoid long-term systemic side effects or reduced efficacy. Despite their convenience and synergistic potential when combined, these therapies can lead to resistance or dependency.
- The treatment of CRSwNP needs to be stratified according to the condition. Medications are the first-line option, with nasal glucocorticoids preferred due to their non-invasive nature and favorable safety profile, though they act slowly and are less effective in severe cases. Antihistamines help relieve allergy-related symptoms but have limited impact on polyps and inflammation, so they are mainly used in patients with concurrent allergies. Macrolide antibiotics provide anti-inflammatory and immunomodulatory effects, but long-term use may lead to resistance and they are ineffective for larger polyps. Overall, drug therapies mainly relieve symptoms, have limited efficacy in refractory cases, may cause side effects with prolonged use, and are associated with high recurrence after discontinuation.
- Surgery, mainly functional endoscopic sinus surgery, can quickly relieve nasal obstruction and loss of smell by removing polyps and opening sinus passages. It is minimally invasive with fast recovery, but recurrence is common (about 35%–38% overall, up to 98% in some patients). As it does not address the underlying cause, perioperative medication is needed to reduce recurrence, and a small number of patients may experience complications. Biologic therapy is an emerging option for severe, refractory CRSwNP. Anti-IL-5/IL-5R agents reduce eosinophilic inflammation, shrink polyps, and improve symptoms, with relatively few side effects, but they are costly and require long-term use. Anti-IgE therapies are effective for patients with severe allergies but have limited benefit in non-allergic cases.