

INDUSTRY OVERVIEW

Certain information and statistics set out in this section have been extracted from various official government publications, available sources from public market data providers and an independent third-party source, Frost & Sullivan. The report prepared by Frost & Sullivan and cited in this document was commissioned by us. The information from official government sources has not been independently verified by our Company, the Sole Sponsor, [REDACTED], any of our or their respective directors, officers, employees, agents or advisers or any other person or party involved in the [REDACTED], and no representation is given as to its accuracy, fairness and completeness. For discussion of the risks relating to our industry, see “Risk Factors” in this document.

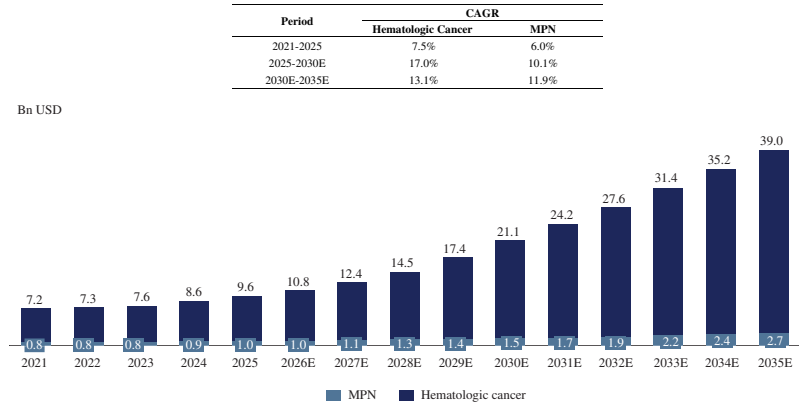
OVERVIEW OF ONCOLOGY DRUG MARKET

Cancer treatment has evolved from traditional approaches such as surgery, radiotherapy and chemotherapy toward precision medicine and immuno-oncology, driven by the pursuit of more effective and less invasive therapies. While surgery remains important for early-stage localized tumors, advances in cancer biology have led to increasing use of systemic treatments. Radiotherapy and chemotherapy broadened treatment options for both solid tumors and hematological malignancies, but often caused substantial side effects due to damage to healthy tissues. The oncology drug market in China was valued at USD38.8 billion in 2025 and is projected to reach USD136.3 billion by 2035. Globally, the oncology drug market was valued at USD278.2 billion in 2025 and is expected to reach USD683.0 billion by 2035.

Myeloproliferative Neoplasms Drug Market

MPNs are chronic blood cancers caused by abnormal proliferation of mature blood cells from dysregulated hematopoietic stem and progenitor cells, often driven by JAK2, CALR or MPL mutations that activate the JAK-STAT pathway. The main subtypes include myelofibrosis, which is associated with bone marrow fibrosis, anemia, splenomegaly and leukemia risk; polycythemia vera, which is characterized by excess red blood cells and increased thrombotic risk; and essential thrombocythemia, which involves persistently elevated platelets with thrombotic and bleeding complications. Across these diseases, treatment generally aims to control symptoms, reduce thrombotic risk, normalize blood counts, reduce spleen volume where relevant, and modify the underlying disease course. The following tables describe the hematologic malignancies and MPN drug market from 2021 to 2035 in China by indications:

China Hematologic Cancer and MPN Drug Market Size, 2021-2035E

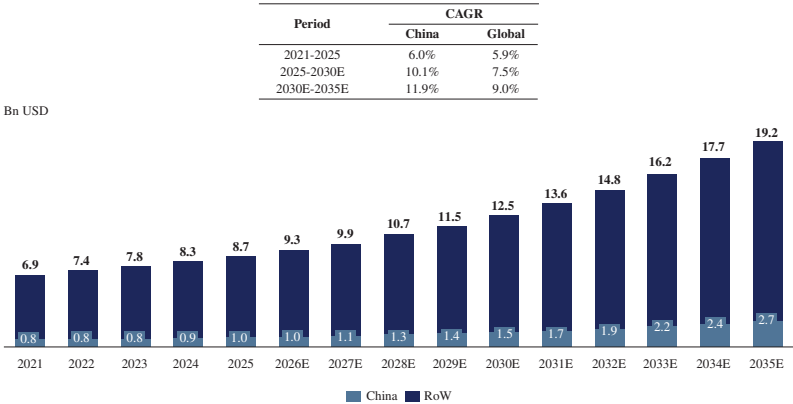


Source: Frost & Sullivan Analysis

INDUSTRY OVERVIEW

The following table describes the MPNs drug market in China and globally from 2021 to 2035:

Global and China MPNs Market Size, 2021-2035E



Source: Frost & Sullivan Analysis

Growth Drivers and Future Trends of Myeloproliferative Neoplasm Drugs

According to Frost & Sullivan, the primary growth drivers and future trends for the myeloproliferative neoplasm drug market globally include:

- **Increasing Prevalence.** The global prevalence of MPNs increased from 5,323.0 thousand in 2021 to 6,070.7 thousand in 2025, representing a CAGR of 3.3%. This increase can be attributed to several factors, including ageing populations, longer life expectancy and obesity.
- **Improving Affordability.** The high costs associated with cancer management often add complexity to treatment. Enhancing affordability for patients is essential to advancing the oncology drug market by alleviating the financial strain of cancer care. Broader inclusion of innovative medications in both public and private insurance schemes has significantly increased patient access and made treatments more affordable. In China, NRDL dynamically expands oncology drug coverage, with 17-36 therapies added annually from 2020-2025. Enhanced reimbursement systems reduce patient financial burdens, improving treatment accessibility and propelling market growth.
- **Precision Treatment and Targeted Drug Innovation.** The advancement of precision oncology through biomarkers and innovative targets like JAK-2 and CD47, moving beyond traditional targets (EGFR/VEGFR). Targeted therapies tailored to genetic/protein profiles address diverse tumor subtypes, meeting unmet needs and driving market differentiation.

INDUSTRY OVERVIEW

Concurrently, indication-level trends and drivers will create distinct trajectories across myeloproliferative neoplasm, including myelofibrosis, polycythemia vera, and essential thrombocythemia. Specifically:

- ***Myelofibrosis*** *Evolution of competitive landscape and advancement of treatment frontiers.* While JAK inhibitors remain the backbone of myelofibrosis therapy, newer JAK2-targeted agents and combination strategies are being developed to address key limitations of first-generation treatments, including cytopenias, tolerability issues, and limited disease-modifying effects. At the same time, JAK2-based regimens are being evaluated in biologically high-risk settings—such as post-polycythemia vera myelofibrosis, post-essential thrombocythemia myelofibrosis, and the pre-transplant setting—with the goal of achieving deeper disease control beyond symptom relief.
- ***Polycythemia Vera*** *Growing availability of disease-modifying and targeted therapies.* Opeginterferon alfa-2b — supported by long-term molecular and clinical benefits — is expanding from niche use to broader adoption as a disease-modifying therapy across both earlier-stage and higher-risk polycythemia vera. In parallel, newer JAK2-focused inhibitors and JAK-based combination strategies are improving hematopoietic control and symptom relief, extending beyond hydroxyurea-refractory settings and being increasingly evaluated in broader populations, including higher-risk and treatment-naïve patients. Together, these trends broaden the eligible patient pool, enable more proactive disease management, and expand the overall market opportunity.
- ***Essential Thrombocythemia*** *Precision medicine and mutation-driven management.* Treatment choices are increasingly tailored to mutation status, with JAK2, CALR and MPL guiding thrombotic risk and therapy selection. Selective JAK2 inhibitors can precisely target abnormal hematopoietic cell clones carrying driver mutations, achieving deep clearance and delaying progression to myelofibrosis. At the same time, these agents are shifting the therapeutic goals in ET from the traditional focus on “preventing and controlling thrombotic complications” to a higher-level paradigm of “reducing long-term thrombotic risk, improving patients’ quality of life, and delaying disease progression.”

JAK Inhibitors

The JAK family consists of four cytoplasmic tyrosine kinases—JAK1, JAK2, JAK3 and TYK2—that transmit extracellular cytokine signals through the JAK-STAT pathway. Upon cytokine binding, JAKs activate one another, phosphorylate receptor tails, recruit and activate STAT proteins, and thereby regulate gene transcription in the nucleus. Among them, JAK2 is particularly important in hematopoiesis, as it mediates signaling for receptors involved in red blood cell, platelet and myeloid cell development, making it a key regulator of blood cell production and immune maturation.

Mechanism of Action of JAK Inhibitors

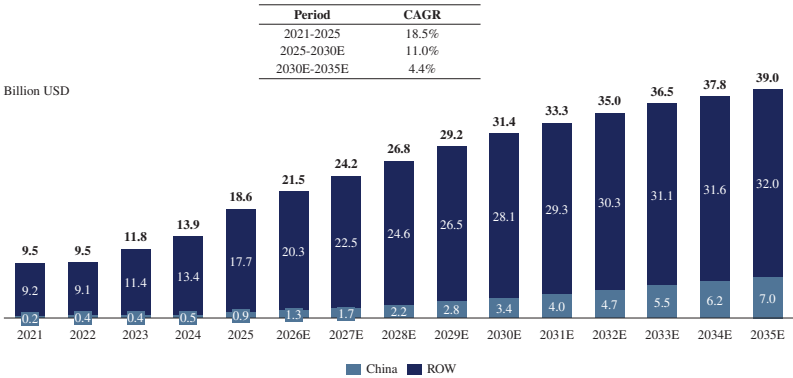
MPNs, including myelofibrosis, polycythemia vera and essential thrombocythemia, are largely driven by overactive JAK-STAT signaling, most commonly due to the JAK2-V617F mutation that constitutively activates cell proliferation and inflammatory pathways. Targeting JAK2 addresses the underlying disease biology by suppressing the abnormal signaling driving clonal expansion, cytokine production and splenomegaly, rather than only managing symptoms. Clinically, JAK inhibitors such as ruxolitinib have demonstrated effective second-line disease control in patients resistant or intolerant to hydroxyurea, including meaningful reductions in spleen size, symptom burden and potential survival benefit. Activation of the JAK-STAT pathway occurs when cytokine binding induces receptor dimerization, triggering JAK phosphorylation, STAT recruitment and activation, and downstream gene regulation governing cell growth, survival and inflammation.

INDUSTRY OVERVIEW

Market Size of JAK Inhibitors

The following tables describe the JAK inhibitor market in China and globally from 2021 to 2035:

Global and China JAK Inhibitor Market Size, 2021-2035E



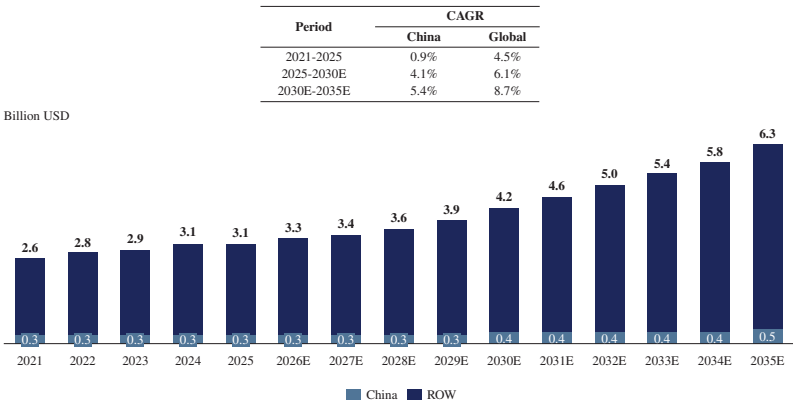
Source: Frost & Sullivan

Major Indications for JAK Inhibitors in Hematologic Malignancies

Myelofibrosis

Myelofibrosis (MF) is a myeloproliferative neoplasm characterized by bone marrow fibrosis that impairs normal blood formation. It may arise as primary MF or evolve from polycythemia vera or essential thrombocythemia, driven by mutations such as JAK2, CALR or MPL that sustain JAK-STAT pathway activation. These abnormalities promote clonal proliferation and inflammatory signaling, leading to collagen deposition, anemia, splenomegaly, fatigue and constitutional symptoms, with disease progression ranging from indolent to high risk, including potential transformation to acute leukemia. The following table describes the MF drug market in China and globally from 2021 to 2035:

Global and China MF Market Size, 2021-2035E



Source: Frost & Sullivan

INDUSTRY OVERVIEW

In China, the prevalence of MF reached 62.4 thousand in 2025, and is expected to reach 63.5 thousand in 2030 and 64.6 thousand in 2035, representing CAGRs of 0.4% from 2021 to 2025, 0.4% from 2025 to 2030 and 0.3% from 2030 to 2035. Globally, the prevalence of MF reached 994.9 thousand in 2025, and is expected to reach 1,135.8 thousand in 2030 and 1,288.7 thousand in 2035, representing CAGRs of 2.7% from 2021 to 2025, 2.7% from 2025 to 2030 and 2.6% from 2030 to 2035.

Treatment Paradigm of MF

In China, SOC treatment of MF patient follows risk stratification using MIPSS70 plus version 2 (risk score for MF). For low risk disease, Category II therapy with interferon is preferred, specifically polyethylene glycol interferon- α (CII) or pegylated interferon- α -2b (CII). For intermediate (-1 and -2) risk disease, Category I options are ruxolitinib or gecacitinib; Category II addresses symptomatic patients with hydroxyurea or selinexor, targets significant splenomegaly with interferon, selinexor, hydroxyurea, busulfan, cladribine, or melphalan, manages extramedullary hematopoiesis, PAH, or limb pain with radiotherapy, and treats portal hypertension with ruxolitinib; Category III uses prednisone for obvious symptoms and ruxolitinib for extramedullary hematopoiesis, PAH, or limb pain. For high risk disease, non-transplant candidates receive Category I ruxolitinib or gecacitinib plus supportive care, with supportive measures including gecacitinib, thalidomide or androgens with low-dose prednisone, and erythropoietin for anemia. Transplant candidates proceed to second-line Category I allogeneic hematopoietic stem cell transplantation, while non-transplant second-line options include Category II gecacitinib and Category III demethylating agents, selinexor, or restarting ruxolitinib after short interruptions. Symptom control targets splenomegaly, anemia, and extramedullary complications using the above agents. In the United States, platelet guided adoption of newer JAK inhibitors such as momelotinib and pacritinib has emerged as a first line approach, and these innovative JAK inhibitors can be integrated into the above Chinese framework alongside ruxolitinib or gecacitinib.

Under the Dynamic International Prognostic Scoring System (DIPSS), approximately 50%-60% of patients with myelofibrosis are classified as intermediate-2 or high risk (Int-2/High) at diagnosis, corresponding to a total score of 3-4 points and 5-6 points, respectively. For patients with intermediate-2/high-risk disease or symptomatic intermediate-1 risk, the first-line drug treatment rate approaches 100%. Bezacitinib, as a JAK inhibitor, is intended to be positioned as a first-line treatment for patients with intermediate-2/high-risk MF. Accordingly, the addressable population for Bezacitinib is expected to represent approximately 50%-60% of the overall MF patient population.

Competitive Landscape

As of the Latest Practicable Date, there were four approved JAK inhibitor drugs for the treatment of MF in overseas markets and four JAK inhibitor drugs had been approved in China for this indication. The following tables set forth the details of marketed JAK inhibitors for the treatment of MF in overseas markets and in China.

Overseas Marketed JAK inhibitor Drug for Treatment of MF

Genetic Name	Brand Name	Target	Company	Indications	First Approval Date	Price USD/Unit
Momelotinib	OJJAARA	JAK1, JAK2, ALK2	GSK	MF	2023-09-15	966.00 (100mg)
Pacritinib	VONJO	JAK2, FLT3, IRAK1, CSF1R, ALK2	SOBI	MF	2022-02-28	210.74 (100mg)
Fedratinib	INREBIC	FLT3, JAK2	BMS	MF	2019-08-16	234.84 (100mg)
Ruxolitinib	JAKAFI	JAK1, JAK2	INCYTE CORP	MF, PV, GVHD	2011-11-16	296.26 (100mg)

Source: FDA, F&S Analysis

INDUSTRY OVERVIEW

China Marketed JAK Inhibitor Drug for Treatment of MF

Genetic Name	Brand Name	Target	Company	Indications	First Approval Date	NRDL Inclusion Year	Price RMB/Unit
Bezacitinib	Bangruishun (邦瑞顺)	JAK2	Hangzhou Bangshun	MF	2026-04-29	N.A.	N.A.
Rovadicitinib	Anxu (安煦)	JAK1, JAK2, ROCK1, ROCK2	CTTQ	MF	2026-02-25	N.A.	N.A.
Gecacitinib	Zepuping (泽普平)	JAK1, JAK2, JAK3, TYK2, ALK2	Suzhou Zelgen	MF	2025-05-29	2025	86.00 (50mg)
Ruxolitinib	Jakavi	JAK1, JAK2	Novartis	MF	2017-03-10	2019	51.85 (5mg)

Source: NMPA, F&S Analysis

As of the Latest Practicable Date, there were no clinical-stage JAK inhibitor drug candidates under development for the treatment of MF in overseas markets, whereas one clinical-stage JAK inhibitor drug candidate was being developed for MF in China.

China Competitive Landscape of JAK Inhibitor Pipelines for Treatment of MF

Pipeline	Target	Company	Phase	Indications	First Posted Date
Flonolitinib	CDK6, FLT3, JAK2	Chengdu Zenitar	III	Myelofibrosis	2025-12-24

Source: CDE, Company website, Literature research, F&S Analysis

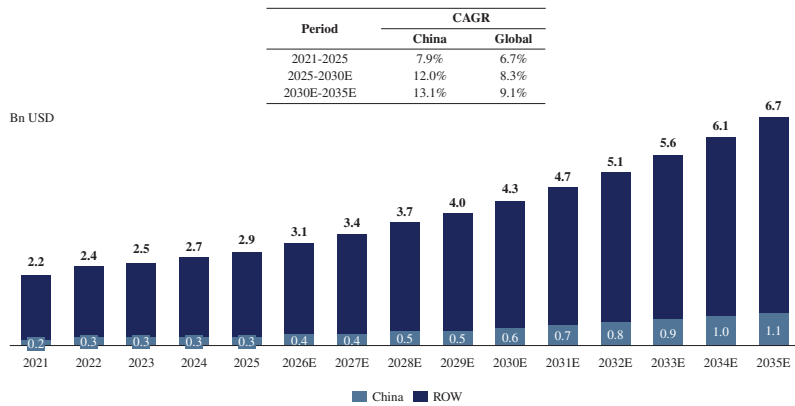
As a highly selective JAK2 inhibitor not metabolized by CYP, Bezacitinib offers lower off-target and drug-drug interaction risk than China-approved peers gecacitinib (JAK1/2/3, TYK2, ALK2) and ruxolitinib (JAK1/2), and a larger placebo-adjusted 24-week SVR35 benefit (48.5% vs gecacitinib’s 38.4%), although its absolute SVR35 (62.8%) is numerically below gecacitinib (64.9%) and these are cross-trial, not head-to-head, comparisons.

Polycythemia Vera

Polycythemia vera (PV) is a myeloproliferative neoplasm characterized by persistent erythrocytosis, often accompanied by leukocytosis and thrombocytosis, and is driven in most cases by activating JAK2 mutations. Diagnosis is based on elevated hemoglobin or hematocrit, increased red cell mass, bone marrow hypercellularity with trilineage proliferation, and the presence of a JAK2 mutation, after excluding secondary causes of erythrocytosis. PV is associated with increased risks of arterial and venous thrombosis, bleeding, microvascular symptoms, pruritus, fatigue, night sweats and splenomegaly-related discomfort, and may reduce life expectancy through thrombotic events or progression to myelofibrosis or, less commonly, acute myeloid leukemia. The following table describes the PV drug market in China and globally from 2021 to 2035:

INDUSTRY OVERVIEW

Global and China PV Drug Market Size, 2021-2035E



Source: Frost & Sullivan Analysis

In China, the prevalence of PV reached 207.2 thousand in 2025, and is expected to reach 247.0 thousand in 2030 and 293.4 thousand in 2035, representing CAGRs of 3.2% from 2021 to 2025, 3.6% from 2025 to 2030 and 3.5% from 2030 to 2035. Globally, the prevalence of PV reached 2,624.0 thousand in 2025, and is expected to reach 3,280.2 thousand in 2030 and 4,067.8 thousand in 2035, representing CAGRs of 4.3% from 2021 to 2025, 4.6% from 2025 to 2030 and 4.4% from 2030 to 2035.

Treatment Paradigm of PV

In China, management of PV is risk stratified by age and thrombotic history. Low risk patients, defined as under sixty years with no prior thrombosis, typically start with phlebotomy and low dose aspirin when not contraindicated. If cytoreduction is needed for symptom control or rising counts, interferon alpha 2b is the first line cytoreductive drug, with hydroxyurea or interferon alpha 2a as recommended alternatives. For inadequate response or intolerance, second line therapy switches to ruxolitinib or to an agent not previously used. High risk patients, defined as age sixty or older or with a thrombosis history, receive cytoreductive therapy up front. Hydroxyurea and peginterferon alpha 2b are the primary options, with recombinant interferon alpha 2a and ruxolitinib recommended in selected cases or after inadequate control. Disease progression triggers pathway specific management. Post-PV-MF is treated according to primary MF protocols, while accelerated or blast phase is managed as MPN accelerated phase or blast phase. In the US, JAK inhibitors such as ruxolitinib are a newer therapeutic class increasingly integrated for patients with inadequate response or intolerance to standard cytoreduction.

For PV, first-line cytoreductive therapy typically includes hydroxyurea. Approximately 25% of PV patients develop resistance or intolerance after hydroxyurea treatment, which is defined as loss of efficacy, disease progression or unacceptable toxicities, thereby requiring second-line therapy. Bezacitinib, as a JAK inhibitor, is intended to be positioned as a second-line treatment for patients with hydroxyurea-resistant or intolerant PV. Accordingly, the addressable population for Bezacitinib is estimated to represent approximately 25% of the overall PV patient population.

Competitive Landscape

As of the Latest Practicable Date, ruxolitinib remained the only approved JAK inhibitor for the treatment of PV in overseas markets and was not marketed in China. The following tables set forth the details of marketed JAK inhibitor for the treatment of PV in overseas markets.

INDUSTRY OVERVIEW

Overseas Marketed JAK Inhibitor Drug for Treatment of PV

Genetic Name	Brand Name	Target	Company	Indications	First Approval Date	Price USD/Unit
RUXOLITINIB	JAKAFI	JAK1, JAK2	INCYTE CORP	PV, MF, GVHD	2011-11-16	296.26 (100mg)

Source: FDA, F&S Analysis

As of the Latest Practicable Date, there were two clinical-stage JAK inhibitor drug candidates under development for the treatment of PV in overseas markets and two in China. Among these candidates, bezacitinib had reached Phase III and was the most clinically advanced.

Overseas Competitive Landscape of JAK Inhibitor Pipelines for Treatment of PV

Pipeline	Target	Company	Phase	Indications	First Posted Date
PRT12396	JAK2 V617F	Prelude Therapeutics	I	Polycythaemia Vera	2026-03-13
ZE74-0282	JAK2 V617F	Eilean Therapeutics	I	Polycythaemia Vera	2026-02-20

Source: Clinicaltrials.gov, Company website, Literature research, F&S Analysis

China Competitive Landscape of JAK Inhibitor Pipelines for Treatment of PV

Pipeline	Target	Company	Phase	Indications	First Posted Date
Bezacitinib	JAK2	Our Company	III	Polycythaemia Vera	2024-04-01
Flonoltinib	CDK6, FLT3, JAK2	Zenitar	II	Polycythaemia Vera	2025-09-10

Source: CDE, Frost & Sullivan

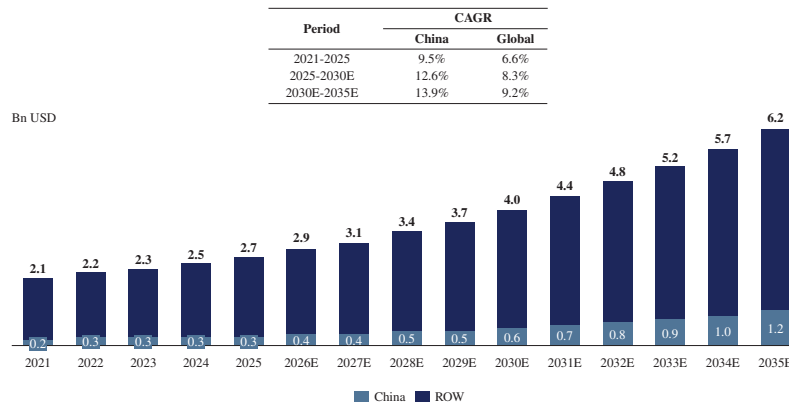
As of the Latest Practicable Date, no JAK inhibitor had been approved for PV in China, while ruxolitinib (JAK1/2) was the only JAK inhibitor approved for PV in overseas markets. Bezacitinib, a highly selective JAK2 inhibitor not metabolized by CYP, was the most clinically advanced JAK inhibitor candidate for PV in China, at Phase III, while flonoltinib, a CDK6/FLT3/JAK2 inhibitor, was at Phase II. In separate clinical trials, bezacitinib reported a 24-week SVR35 of 56.3%, while ruxolitinib reported an SVR35 of 38% at week 32. However, as these results are derived from separate trials with differing designs, assessment timepoints and patient populations rather than head-to-head studies, such cross-trial comparisons are subject to inherent limitations.

Essential Thrombocythemia

Essential thrombocythemia (ET) is a chronic myeloproliferative neoplasm characterized by persistent thrombocytosis, absence of BCR-ABL1, and increased large mature megakaryocytes in the bone marrow. It is driven by hyperactive JAK-STAT signaling, most commonly through JAK2, CALR or MPL mutations, with additional genetic alterations contributing to disease phenotype and progression. Patients may be asymptomatic or present with microvascular symptoms such as headache, dizziness and erythromelalgia, and face risks of thrombosis and, less commonly, bleeding. Treatment primarily aims to prevent thrombotic and hemorrhagic complications while reducing the risk of progression to myelofibrosis or, rarely, acute myeloid leukemia. The following table describes the ET drug market in China and globally from 2021 to 2035:

INDUSTRY OVERVIEW

Global and China ET Drug Market Size, 2021-2035E



Source: Frost & Sullivan Analysis

In China, the prevalence of ET reached 295.7 thousand in 2025, and is expected to reach 322.8 thousand in 2030 and 351.2 thousand in 2035, representing CAGRs of 1.7% from 2021 to 2025, 1.8% from 2025 to 2030 and 1.7% from 2030 to 2035. Globally, the prevalence of ET reached 2,459.7 thousand in 2025, and is expected to reach 2,808.0 thousand in 2030 and 3,186.0 thousand in 2035, representing CAGRs of 2.7% from 2021 to 2025, 2.7% from 2025 to 2030 and 2.6% from 2030 to 2035.

Treatment Paradigm of ET

In China, SOC of ET is stratified by age and thrombotic history. Low risk patients, defined as under sixty years with no prior thrombosis, receive cardiovascular risk control and daily aspirin at eighty to one hundred milligrams when not contraindicated. Cytoreduction is added if counts or symptoms warrant, using hydroxyurea, recombinant interferon alpha 2a, or anagrelide. High risk patients, defined as age sixty or older or with a history of thrombosis, start with the same foundation of cardiovascular risk control plus aspirin, and proceed to cytoreductive therapy with hydroxyurea, recombinant interferon alpha 2a, or anagrelide as first line choices. If response is inadequate or intolerance arises, second line therapy rotates among these agents according to efficacy, tolerability, pregnancy plans, and comorbidities. In selected refractory or intolerant cases, particularly with symptomatic splenomegaly or significant constitutional symptoms, JAK inhibitors may be used off label to achieve symptom control. Disease evolution is managed by pathway, with post-ET-MF treated as primary MF and accelerated or blast phase managed under MPN accelerated or blast phase protocols. In the US, JAK inhibitors are emerging therapies increasingly incorporated for patients with inadequate control or intolerance to standard cytoreduction.

In ET, cytoreductive therapy such as hydroxyurea or interferon is typically used for intermediate- to high-risk patients, who represent approximately 50%-60% of the overall ET population. Among these patients, published studies suggest that approximately 20% may develop hydroxyurea resistance or intolerance and require treatment escalation, while clinical studies of pegylated interferon α -2a have reported treatment discontinuation due to adverse events in approximately 13.9% of patients. Bezacitinib, as a JAK inhibitor, is intended to be positioned as a second-line and beyond (2L+) treatment option for patients with hydroxyurea resistance or intolerance and/or interferon intolerance, with the addressable population therefore estimated to represent approximately 10%-20% of the overall ET patient population.

INDUSTRY OVERVIEW

Competitive Landscape

As of the Latest Practicable Date, no JAK inhibitor drug had been approved for the treatment of ET either in overseas markets or in China. As of the same date, bezacitinib remained the only clinical-stage JAK inhibitor drug candidate under development for ET in China and there were no clinical-stage JAK inhibitor drug candidates in overseas markets.

China Competitive Landscape of JAK Inhibitor Pipelines for Treatment of ET

Pipeline	Target	Company	Phase	Indications	First Posted Date
Bezacitinib	JAK2	Our Company	II	Essential Thrombocythemia	2025-07-04

Source: CDE, Frost & Sullivan

Overview of Major JAK Inhibitors in Hematologic Malignancies

The table below provides an overview of approved JAK inhibitors and JAK inhibitor drug candidates for major indications in hematologic malignancies, together with a comparison of key product characteristics, including their respective advantages and limitations.

	Bezacitinib	Ruxolitinib	Fedratinib	Pacritinib	Momelotinib	Gecacitinib	Flonoltinib	Rovadicitinib
Target	Highly Selective JAK2	JAK1, JAK2	FLT3, JAK2	CSF1R, IRAK1, JAK2, FLT3	JAK1, JAK2, ACVR1, ALK2	JAK1, JAK2, JAK3, TYK2, ACVR1	JAK2, FLT3, CDK8	JAK1, JAK2, ROCK1, ROCK2
Indication	MF, PV, ET, GvHD	In US: MF, PV, GvHD In China: MF, GvHD	MF	MF	MF	MF	MF, PV	MF, cGvHD, aGvHD
Efficacy	<ul style="list-style-type: none">MF: 24w SVR35: 62.8% (14.3%)PV: 24w SVR35: 36.3%	<ul style="list-style-type: none">MF: 24w SVR35: 41.9%PV: 32w SVR35: 38%	<ul style="list-style-type: none">MF: 24w SVR35: 47%	<ul style="list-style-type: none">MF: 24w: SVR35: 29%	<ul style="list-style-type: none">MF: 24w SVR35: 31%	<ul style="list-style-type: none">MF: 24w SVR35: 64.9% (vs control group of 26.5%)	<ul style="list-style-type: none">MF: 24w SVR35: 83.3%	<ul style="list-style-type: none">MF: 24w SVR35: 75%
Metabolism/ DDI (Drug-Drug Interaction)	Not metabolized by CYP, reduce DDI related risks	Metabolised by CYP3A4	Mainly metabolised by CYP3A4, CYP2C19, and FMO3	Mainly metabolised by CYP3A4	Metabolized by CYP3A4, CYP2C8/9/19, and CYP1A2	NA	CYP inhibition (CYP2C19, CYP2C9, CYP3A4), high DDI risks	Mainly metabolised by CYP3A4
Safety Profile	Common AEs: anemia, and thrombocytopenia; no drug-specific or off-target toxicities	Common AEs: anemia, thrombocytopenia	Common AEs: anemia, thrombocytopenia, and elevated lipase.	Common AEs: diarrhea, nausea, anemia, thrombocytopenia	Common AEs: thrombocytopenia, diarrhea, bleeding, fatigue, nausea	Common AEs: anemia, thrombocytopenia, leukopenia	Common AEs: thrombocytopenia, anemia	Common AEs: thrombocytopenia, anemia
Administration	20mg BID	20mg BID	400mg BID	200mg BID	200mg BID	100mg BID	100mg QD	15mg BID

Notes: Bezacitinib does not have head-to-head clinical trials with existing JAK inhibitors; flonoltinib (6/8) and rovadicitinib (25/30) results are based on early-phase (Phase I/Ib/IIa) trials with small patient cohorts; the difference between the control and bezacitinib groups was 48.5%, while that between the control and gecacitinib groups was 38.4%.

Source: Literature Review; Frost & Sullivan

OVERVIEW OF AUTOIMMUNE DISEASES

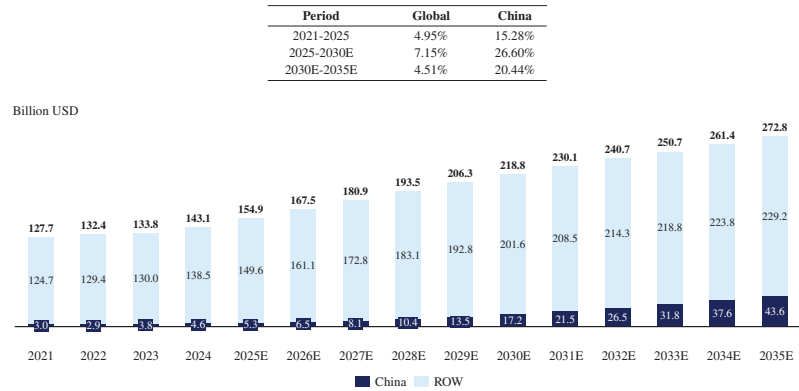
Autoimmune diseases are chronic disorders in which persistent or misdirected immune activation damages healthy tissues, causing significant clinical and economic burden across multiple organ systems. The indications covered in this document include immune-mediated hematologic, dermatologic and gastrointestinal disorders, as well as myeloproliferative neoplasms with prominent inflammatory features. Examples include ITP and AIHA, chronic spontaneous urticaria, rheumatoid arthritis, inflammatory bowel disease, graft-versus-host disease, and MPNs such as myelofibrosis, polycythemia vera and essential thrombocythemia. Treatment has evolved from broad symptom control with NSAIDs and glucocorticoids to disease-modifying therapies, including conventional DMARDs, biologics targeting TNF and key interleukins, and more recently targeted synthetic therapies such as JAK and BTK inhibitors. Overall, the field has shifted toward mechanism-based treatments aimed at more effective and durable disease control.

INDUSTRY OVERVIEW

Market Size of Autoimmune Diseases Drug

The following table describes the global autoimmune drug market from 2021 to 2035:

Global Autoimmune Drugs Market, 2021-2035E



Source: Frost & Sullivan

Growth Drivers and Future Trends of Autoimmune Diseases Drug Market

The autoimmune diseases drug market is entering a phase of broad, rapid evolution shaped by both trends and driving factors. Specifically:

- Development of autoimmunity.** Advances in elucidating autoimmune pathogenesis and validating biomarkers are enabling rationally designed, targeted therapies. Notable progress includes small-molecule inhibitors and biologics that selectively modulate cytokines and intracellular signaling (e.g., JAK-STAT, TYK2), central to conditions such as rheumatoid arthritis, psoriasis, and atopic dermatitis. By benefiting patients refractory or intolerant to conventional immunosuppression, these precision approaches shift standards of care, improve risk-benefit profiles, enable earlier intervention, and catalyze sustained market growth.
- Targeted Therapy Upgrades with Multi-target Inhibitors.** Next-generation dual- and multi-target small molecules and bispecific biologics are improving precision by concurrently modulating complementary immune pathways (e.g., JAK-TYK2, IL-17/IL-23). By addressing incomplete responses, relapse, and resistance seen with single-target agents, they deliver deeper, more durable disease control with lower off-target toxicity. These high-specificity, high-potency therapies meet unmet needs for efficacy and safety, support earlier intervention and steroid-sparing regimens, and are poised to accelerate adoption and market growth as evidence from pivotal trials matures.
- Enhanced Patient Compliance with Oral Administration and Sustained-release Technologies.** Oral BTK inhibitors (rilzabrutinib and others) and other kinase inhibitors are reshaping autoimmune care by improving convenience, adherence, and pharmacologic stability. Orally delivered targeted agents enable rapid attainment of therapeutic levels and earlier disease control, while long-acting formulations reduce dosing frequency, smooth peak-trough variability, and enhance persistence. Together, these delivery advances lessen injection burden, support steroid-sparing strategies, and improve real-world effectiveness. Ongoing innovation in extended-release, depot, and programmable delivery systems is expected to further raise adherence and fuel market uptake.

INDUSTRY OVERVIEW

Concurrently, indication-level trends and drivers will create distinct trajectories across immunologic thrombocytopenic purpura, chronic urticaria and autoimmune hemolytic anemia. Specifically:

- ***Graft-versus-host Disease Earlier-Line adoption and rational combinations poised to reshape GVHD treatment algorithms.*** Earlier use and rational combinations of innovative agents are set to redefine GVHD treatment. Effective targeted therapies are moving from later lines to earlier settings as steroid-sparing or steroid-minimizing options. Pairing targeted agents with reduced-dose steroids or other immunomodulators aims to improve response durability, mirroring trends in other immune-mediated diseases.
- ***Immunologic Thrombocytopenic Purpura Precision immunology and biomarker-guided care.*** Care is shifting toward mechanistic endotypes, distinguishing autoantibody-driven destruction, impaired thrombopoiesis, and T-cell dysregulation to anticipate response. Better assays, including platelet autoantibody panels, innate cell activation readouts, and cytokine signatures, will stratify patients and guide targeted agents. Trials will increasingly enroll by biomarker-defined subgroups to boost response rates and support precision labeling.
- ***Chronic Urticaria Transformation of treatment paradigm and therapeutic innovation.*** New mechanisms are reshaping care, with agents like the BTK inhibitor remibrutinib showing promising results in patients unresponsive to antihistamines. As many patients remain refractory to standard options such as second-generation H1 antihistamines or biologics, these innovative targets aim to deliver stronger, more consistent responses and broaden coverage for hard-to-treat populations, signaling a clear shift toward more effective, mechanism-driven therapy.
- ***Autoimmune Hemolytic Anemia Earlier, more accurate diagnosis enables timely treatment.*** By strengthening early recognition and diagnostic accuracy of AIHA, more patients can be identified quickly and reliably, increasing the treatable population and enabling timely initiation of appropriate therapies at earlier disease stages. Clear patient segmentation further defines which individuals should receive high-value treatments and when, improving clinical outcomes while expanding equitable access.

JAK Inhibitors

JAK inhibitors suppress dysregulated cytokine signaling that drives autoimmunity and inflammation by blocking JAK-STAT pathway activation. This reduces pro-inflammatory gene transcription, dampens activation of macrophages, dendritic cells and pathogenic lymphocyte responses, and decreases chemokine-mediated recruitment of inflammatory cells to tissues. JAK blockade may also lessen inflammasome priming, endothelial activation and vascular inflammation. Through this broad but reversible modulation of convergent immune pathways, JAK inhibitors can help control both systemic and tissue-specific autoimmune inflammation. For more details of JAK inhibitors, please refer to paragraphs “Overview of Oncology Drug Market — JAK Inhibitors” in this section.

INDUSTRY OVERVIEW

Major Indications for JAK Inhibitors in Autoimmune Diseases

Graft-Versus-Host Disease

Graft-versus-host disease (GVHD) is a systemic inflammatory complication of allogeneic hematopoietic stem cell transplantation in which donor lymphocytes recognize recipient antigens as foreign and mount an immune attack that can damage multiple organs. There are two subtypes: acute graft-versus-host disease (aGVHD) and chronic graft-versus-host disease (cGVHD). aGVHD is an early, inflammation-dominant process; cGVHD is later, autoimmune-like with progressive fibrosis and multi-organ dysfunction.

In China, the incidence of aGVHD reached 9.7 thousand in 2025, and is expected to reach 14.9 thousand in 2030 and 24.9 thousand in 2035, representing CAGRs of 7.4% from 2021 to 2025, 8.8% from 2025 to 2030 and 10.9% from 2030 to 2035. Globally, the incidence of aGVHD reached 57.4 thousand in 2025, and is expected to reach 78.3 thousand in 2030 and 109.7 thousand in 2035, representing CAGRs of 9.5% from 2021 to 2025, 6.4% from 2025 to 2030 and 7.0% from 2030 to 2035. In China, the prevalence of cGVHD reached 15.5 thousand in 2025, and is expected to reach 34.7 thousand in 2030 and 73.4 thousand in 2035, representing CAGRs of 9.4% from 2021 to 2025, 17.5% from 2025 to 2030 and 16.2% from 2030 to 2035. Globally, the prevalence of cGVHD reached 104.2 thousand in 2025, and is expected to reach 192.8 thousand in 2030 and 315.5 thousand in 2035, representing CAGRs of 12.8% from 2021 to 2025, 13.1% from 2025 to 2030 and 10.3% from 2030 to 2035.

In aGVHD, patients are clinically stratified by severity grades, with Grade II-IV defined as moderate-to-severe disease. Globally, approximately 40%-50% of patients with Grade II-IV aGVHD develop steroid-refractory or steroid-dependent disease requiring second-line or beyond treatment after first-line corticosteroid therapy, while a clinical cohort study in China reported that approximately 65% of Grade II or higher patients were assessed by clinicians as requiring second-line therapy. Bezacitinib, as a JAK inhibitor, is intended to be positioned for the treatment of steroid-refractory or steroid-dependent aGVHD, with the addressable population therefore estimated to represent approximately 40%–50% of the overall aGVHD patient population globally, and ~65% in China.

In cGVHD, patients with moderate-to-severe disease are typically treated with systemic corticosteroids as first-line therapy; however, globally more than 50% of patients develop steroid resistance or steroid dependence within two years, necessitating second-line or subsequent therapies due to inadequate efficacy, relapse or inability to safely taper steroids. In China, approximately 80% of patients require second-line or later-line treatments, and nearly half eventually receive third-line or beyond therapies. CX1440, as a BTK inhibitor, is intended to be positioned for the treatment of steroid-refractory or steroid-dependent moderate-to-severe cGVHD, with the addressable population therefore estimated to represent more than 50% of the overall cGVHD patient population globally, and ~80% in China.

Treatment paradigm of GVHD

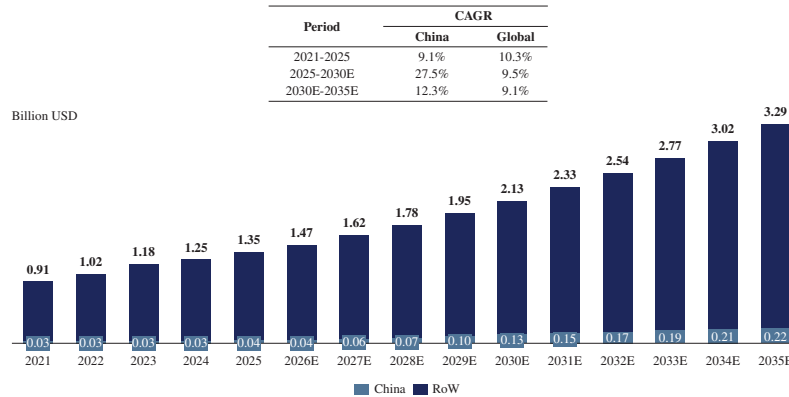
Acute GVHD

Systemic glucocorticoids remain the standard first-line therapy for grade II–IV aGVHD; however, their effectiveness is below 50%, and only approximately one-third of responders sustain remission. Mesenchymal stromal cell (MSC) infusion may be used as a second- or third-line option for glucocorticoid-refractory aGVHD, including cases resistant to multiple second-line agents, with reported effectiveness rates of approximately 57%-73%. In addition, multicentre, prospective, randomised controlled studies conducted in China reported a significantly higher overall effectiveness rate for MSCs combined with basiliximab than for basiliximab alone in glucocorticoid-refractory aGVHD.

INDUSTRY OVERVIEW

The following table describes the aGVHD drug market in China and globally from 2021 to 2035:

Global aGVHD Drug Market Size, 2021-2035E



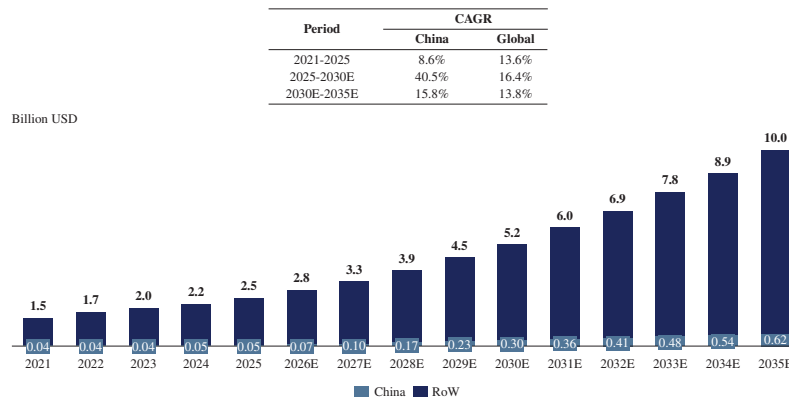
Source: Frost & Sullivan Analysis

Chronic GVHD

Not all patients with cGVHD require systemic therapy at diagnosis. Mild disease may be managed with observation or local treatment, whereas moderate-to-severe disease requires systemic therapy. For first-line treatment of moderate-to-severe cGVHD, glucocorticoids alone or in combination with calcineurin inhibitors remain the standard of care, with an effectiveness rate of approximately 50%. For patients who fail first-line therapy, targeted agents have become available as second-line options: both the JAK1/JAK2 inhibitor ruxolitinib and the ROCK2 inhibitor belumosudil have been approved by the NMPA for cGVHD (ruxolitinib being recommended by guidelines as a preferred second-line option), while the BTK inhibitor ibrutinib is approved for cGVHD in certain overseas markets but, as at the Latest Practicable Date, has not been approved by the NMPA for this indication. Mesenchymal stromal cell therapy is likewise recommended as a second-line option, with reported remission rates of approximately 57.1%-73.7%.

The following table describes the cGVHD drug market in China and globally from 2021 to 2035:

Global cGVHD Drug Market Size, 2021-2035E



Source: Frost & Sullivan Analysis

INDUSTRY OVERVIEW

Competitive Landscape

As of the Latest Practicable Date, one JAK inhibitor drug had been approved for the treatment of GVHD in overseas markets and in China. The following tables set forth the details of marketed JAK inhibitor drug for the treatment of GVHD in overseas markets and in China.

Overseas Marketed JAK Inhibitor Drugs for Treatment of GVHD

Genetic Name	Brand Name	Target	Company	Approved Indications	First Approval Date	Price USD/Unit
RUXOLITINIB	JAKAFI	JAK1, JAK2	INCYTE CORP	GVHD	2011-11-16	296.26 (100mg)

Source: FDA, F&S Analysis

China Marketed JAK Inhibitor Drug for Treatment of GVHD

Genetic Name	Brand Name	Target	Company	Approved Indications	First Approval Date	NRDL Inclusion Year	Price RMB/Unit
Ruxolitinib	Jakavi	JAK1 , JAK2	Novartis	GVHD	2017-03-10	2019	51.85 (5mg)

Source: Frost & Sullivan

As of the Latest Practicable Date, bezacitinib remained the only clinical-stage JAK inhibitor drug candidate under development for GVHD in China and there were no clinical-stage JAK inhibitor drug candidates for GVHD in overseas markets.

China Competitive Landscape of JAK Inhibitor Pipelines for Treatment of GVHD

Pipeline	Target	Company	Phase	Indications	First Posted Date
Bezacitinib	JAK2	Our Company	I/II	GVHD	2022-03-15

Source: Frost & Sullivan

As of the Latest Practicable Date, ruxolitinib was the only JAK inhibitor approved for GVHD in China, while bezacitinib was the only clinical-stage JAK inhibitor candidate under development for GVHD in China, at Phase I/II, for steroid-refractory or steroid-dependent aGVHD. Bezacitinib is a highly selective JAK2 inhibitor and is not metabolized by CYP.

BTK Inhibitors

BTK inhibitors are targeted therapies that block BTK, a key kinase in B-cell receptor signaling that regulates B-cell activation, survival and migration. Because abnormal BTK signaling drives many B-cell malignancies, BTK has become a validated therapeutic target. First-generation and newer, more selective BTK inhibitors have shown strong efficacy in diseases such as chronic lymphocytic leukemia, mantle cell lymphoma and Waldenström macroglobulinemia, although toxicities and resistance remain important challenges. Current development focuses on improving selectivity and overcoming resistance, including through noncovalent inhibitors active against C481 mutations.

INDUSTRY OVERVIEW

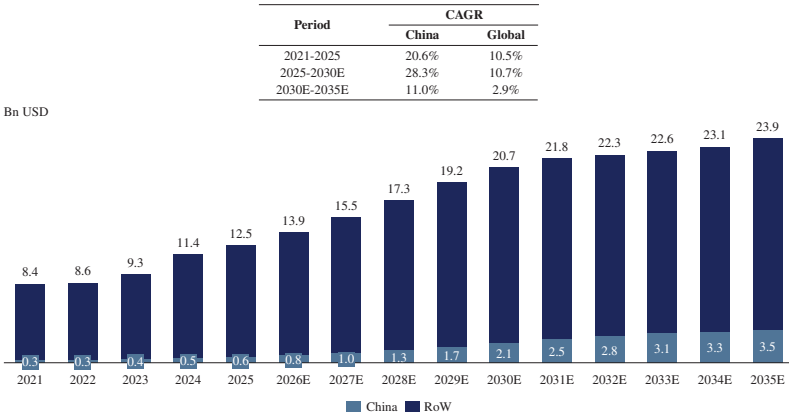
Mechanism of Action of BTK Inhibitors

BCR signaling programs gene expression that enables clonal expansion, differentiation into plasma and memory cells, and survival, thereby underpinning antigen specific antibody production and adaptive responses. In B cell lymphomas, this circuitry becomes pathologic when BTK sustains continuous transmission of BCR signals to effector pathways such as NF- κ B and MAPK, maintaining pro survival and pro proliferation transcription. BTK inhibitors interrupt this constitutive signaling, attenuate the downstream transcriptional program, and thereby impair malignant B cell growth and viability.

Market Size of BTK Inhibitors

The following tables describe the BTK inhibitors drug market from 2021 to 2035 in China and globally:

Global and China BTK Inhibitor Market Size, 2021-2035E



Source: Frost & Sullivan

Major Indications for BTK Inhibitors in Autoimmune Diseases

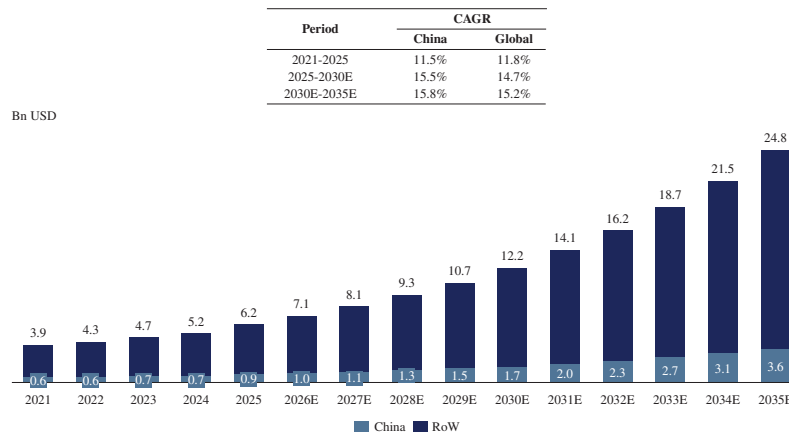
Immunologic Thrombocytopenic Purpura

Immune thrombocytopenia (ITP) is an autoimmune disorder characterized by low platelet counts caused by increased platelet destruction and impaired platelet production. Diagnosis is primarily clinical and requires exclusion of secondary causes such as infection, medications, liver disease or other autoimmune disorders. Patients may present with petechiae, purpura and mucosal bleeding, with severe cases carrying a risk of intracranial hemorrhage. In adults, ITP often follows a chronic course and is more common in women.

INDUSTRY OVERVIEW

The following table describes the ITP drug market in China and globally from 2021 to 2035:

Global and China ITP Drug Market Size, 2021-2035E



Source: Frost & Sullivan

In China, the prevalence of ITP reached 216.4 thousand in 2025, and is expected to reach 220.3 thousand in 2030 and 223.4 thousand in 2035, representing CAGRs of 0.5% from 2021 to 2025, 0.4% from 2025 to 2030 and 0.3% from 2030 to 2035. Globally, the prevalence of ITP reached 1,103.9 thousand in 2025, and is expected to reach 1,183.2 thousand in 2030 and 1,261.8 thousand in 2035, representing CAGRs of 1.5% from 2021 to 2025, 1.4% from 2025 to 2030 and 1.3% from 2030 to 2035.

Treatment Paradigm of ITP

In China, first line therapy for immune thrombocytopenia uses corticosteroids to quickly raise platelets, typically prednisone or high dose dexamethasone; intravenous immunoglobulin is added for rapid hemostatic support or steroid intolerance. With inadequate response or relapse, second line options include thrombopoietin stimulating agents such as recombinant human thrombopoietin and eltrombopag to boost platelet production, and rituximab to suppress immune mediated destruction. Selected patients may receive recombinant human thrombopoietin plus rituximab to deepen or prolong responses. Care is individualized by bleeding risk, comorbidities, and disease phase. In the US, BTK inhibitors have emerged as an innovative treatment class integrated into second line strategies.

In ITP, first-line therapy typically includes corticosteroids and/or intravenous immunoglobulin (IVIG); however, globally approximately 15%-20% of patients are refractory to or relapse after standard first-line treatment and therefore require second-line or subsequent therapies. CX1440, as a BTK inhibitor, is intended to be positioned for patients whose response to corticosteroids and/or IVIG cannot be sustained, with the addressable population therefore estimated to represent approximately 15%-20% of the overall ITP patient population globally. In China, publicly available studies do not clearly quantify the proportion of ITP patients receiving second-line or later-line treatment.

Competitive Landscape

As of the Latest Practicable Date, one BTK inhibitor drug was approved for the treatment of ITP in overseas markets and no BTK inhibitor drug had been approved in China for this indication. The following tables set forth the details of marketed drug for the treatment of ITP in overseas markets.

INDUSTRY OVERVIEW

Overseas Marketed BTK Inhibitor Drug for Treatment of ITP

Genetic Name	Brand Name	Target	Company	Approved Indications	First Approval Date	Price USD/Unit
Rilzabrutinib	Wayrizl	BTK	GENZYME	ITP	2025 -08 -29	285.03 (400mg)

Source: FDA, F&S Analysis

As of the Latest Practicable Date, pirtobrutinib remained the only clinical-stage BTK inhibitor drug candidate under development for ITP in overseas markets. In China, six clinical-stage BTK inhibitor drug candidates were under development for ITP.

Competitive Landscape of BTK Inhibitor Pipelines for Treatment of ITP in Overseas Markets

Pipeline	Target	Company	Phase	Indication	First Posted Date
Pirtobrutinib	BTK	Eli Lilly	I/II	ITP	2024/12/6

Source: Clinical Trial, Frost & Sullivan

China Competitive Landscape of BTK Inhibitor Pipelines for Treatment of ITP

Pipeline	Target	Company	Phase	Indication	First Posted Date
Rilzabrutinib	BTK	Sanofi/Genzyme	NDA	ITP	2024-12-30
Orelabrutinib	BTK	InnoCare	NDA	ITP	2023-07-17
CX1440	BTK	Our Company	III	ITP	2025-08-22
DZD8586	BTK/LYN	Dizal Pharmaceuticals	II	ITP	2025-12-19
HZ-A-018	BTK	HangZhou HealZen	II	ITP	2025-10-24
Pirtobrutinib	BTK	Eli Lilly	I/II	ITP	2025-09-11

Source: CDE, Company website, Literature research, F&S Analysis

As of the Latest Practicable Date, rilzabrutinib was the only BTK inhibitor approved for ITP in overseas markets, while no BTK inhibitor had been approved for ITP in China. CX1440 was among the most clinically advanced domestic BTK inhibitor candidates for ITP, at Phase III.

INDUSTRY OVERVIEW

Overview of Major BTK Inhibitors in ITP

The table below provides an overview of approved BTK inhibitors and BTK inhibitor drug candidates in ITP, together with a comparison of key product characteristics, including their respective advantages and limitations.

	CX1440	Ritxabrutinib	Orelabrutinib
Target	BTK	BTK	BTK
Efficacy	<ul style="list-style-type: none">24w Platelet Response Rate: 41.2%	<ul style="list-style-type: none">24w Durable Platelet Response Rate: 23.3%	<ul style="list-style-type: none">24w Platelet Response Rate: 36%
Safety Profile	<ul style="list-style-type: none">Common adverse reactions associated with BTK inhibitors (such as diarrhea, nausea, and fatigue) were rarely observedTo date, no serious adverse reactions or fatalities have been reported.	<ul style="list-style-type: none">Common AEs: Diarrhea, nausea, headache, abdominal pain, and COVID-19Severe infections: Increased incidence, including one fatal case of pneumonia	<ul style="list-style-type: none">Common AEs: Neutropenia, anemia, thrombocytopenia, febrile neutropenia, and pulmonary infection.

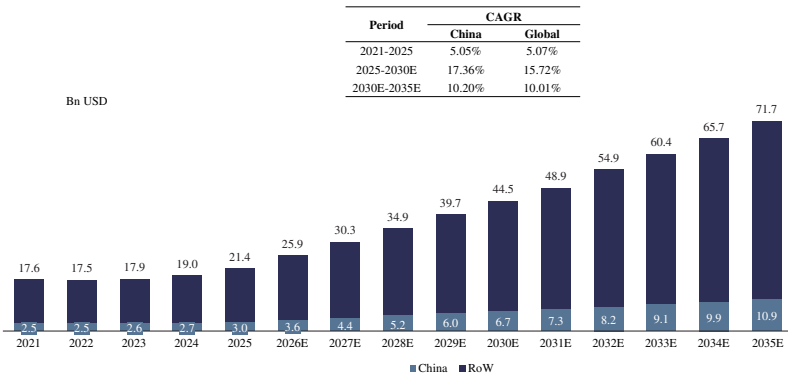
Notes: CX1440 does not have long-term safety and efficacy data, and head-to-head clinical trials with existing BTK inhibitors

Source: Literature Review; Frost & Sullivan Analysis

Chronic Urticaria

Chronic urticaria (CU) is a common allergic skin disorder defined by transient, intensely pruritic wheals that are slightly raised and range from millimeters to centimeters, often appearing and resolving within hours and sometimes accompanied by angioedema. It may occur spontaneously or be induced by specific stimuli, including physical factors. The following table describes the CU drug market in China and globally from 2021 to 2035:

Global and China CU Drug Market Size, 2021-2035E



Source: Frost & Sullivan

INDUSTRY OVERVIEW

In China, the prevalence of CU reached 39.6 million in 2025, and is expected to reach 41.3 million in 2030 and 42.2 million in 2035, representing CAGRs of 0.8% from 2021 to 2025, 0.8% from 2025 to 2030 and 0.4% from 2030 to 2035. Globally, the prevalence of CU reached 94.1 million in 2025, and is expected to reach 98.8 million in 2030 and 103.3 million in 2035, representing CAGRs of 1.1% from 2021 to 2025, 1.0% from 2025 to 2030 and 0.9% from 2030 to 2035.

Treatment Paradigm of CU

In China, CU management begins with standard dose second generation H1 antihistamines. If control is inadequate, the dose is increased up to fourfold, and combination with another antihistamine can be considered. For patients who remain symptomatic, add on biologic or immunosuppressive therapy is recommended, typically omalizumab or ciclosporin, with short courses of glucocorticoids as rescue for severe flares. Therapy is stepped and individualized to minimize sedation, maintain daily functioning, and reduce angioedema and wheal burden. In the US, a similar stepwise pathway is used, and BTK inhibitors have emerged as a new treatment option incorporated alongside advanced lines of therapy.

In CU, which includes CSU and CIU, standard-dose second-generation H1 antihistamines are recommended as first-line therapy for a treatment period of approximately one to two weeks; however, globally such treatment achieves symptom control in approximately 43% of patients, leaving around 50%-60% of patients requiring treatment escalation due to inadequate response. Similarly, studies in China indicate that standard-dose H1 antihistamines achieve complete symptom control in less than half of CSU patients, with more than 50% requiring second-line or subsequent therapies. CX1440, as a BTK inhibitor, is intended to be positioned for patients with CU whose symptoms are inadequately controlled by H1 antihistamines, with the addressable population therefore estimated to represent approximately 50%-60% of the overall CU patient population globally, and 50%+ in China.

Competitive Landscape

As of the Latest Practicable Date, one BTK inhibitor drug was approved for the treatment of CU (including CSU and CIU) in overseas markets with one approved BTK inhibitor in China. As of the same date, there were two clinical-stage BTK inhibitor drug candidates for CU (including CSU and CIU) in overseas markets and five clinical-stage BTK inhibitor drug candidates under development for CU (including CSU and CIU) in China.

Overseas Marketed BTK inhibitor Drug for Treatment of CU/CSU/CIU

Genetic Name	Brand Name	Target	Company	Indications	First Approval Date	Price USD/Unit
Remibrutinib	Rhapsido	BTK	Novartis	CSU	2025/9/30	73.73 (25mg)

Source: FDA, F&S Analysis

China Marketed BTK Inhibitor Drugs for Treatment of CU (CSU and CIU)

Genetic Name	Brand Name	Target	Company	Indications	First Approval Date	NRDL Inclusion Year	Price RMB/Unit
Remibrutinib	Rhapsido	BTK	Novartis	CSU	2025/11/25	NA	NA

Source: NMPA, Frost & Sullivan

INDUSTRY OVERVIEW

Overseas Competitive Landscape of BTK Inhibitors Indicated for CU/CSU/CIU

Genetic Name	Target	Company	Phase	Indication	First Posted Date
TAS5315	BTK	Taiho Pharmaceutical	Phase II	CSU	2022-04-19
Rilzabrutinib	BTK	Sanofi	Phase II	CSU	2021-11-04

Source: FDA, EMA, Clinical Trial, Frost & Sullivan

Competitive Landscape of BTK Inhibitors Indicated for CU (CSU and CIU) in China

Genetic Name	Target	Company	Phase	Indication	First Posted Date
TM471-1	BTK	Henan Zhuwei	Phase II	CSU	2025-12-12
HHW486	BTK	Humanwell	Phase II	CSU	2023-11-20
CX1440	BTK	Our company	Phase I/II	CU(CSU/CIU)	2025-08-22
HS-10561	BTK	Hansoh Pharmaceutical	Phase I/II	CSU	2025-03-12
BGB-16673	BTK	BeiGene (Suzhou)	Phase I	CSU	2025-05-19

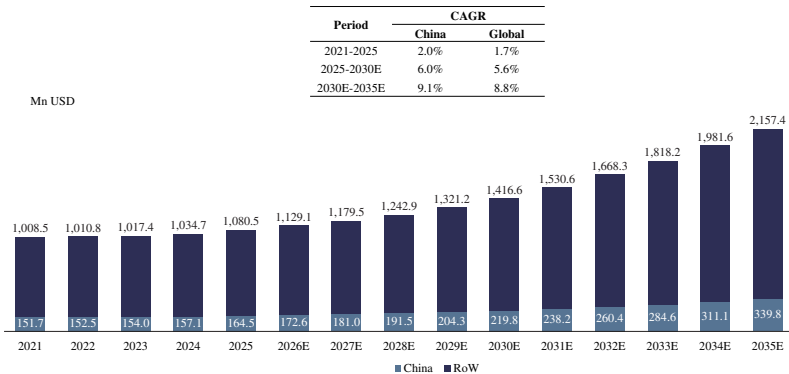
Source: NMPA, Frost & Sullivan

As of the Latest Practicable Date, remibrutinib was the only BTK inhibitor approved for CU (CSU) in both overseas markets and China, while CX1440 was one of several clinical-stage BTK inhibitor candidates under development for CU (CSU and CIU) in China, at Phase I/II. CX1440 is being developed for patients with CU inadequately controlled by H1-antihistamines.

Autoimmune Hemolytic Anemia

Autoimmune hemolytic anemia (AIHA) is an acquired autoimmune disorder in which autoantibodies target red blood cells, causing hemolysis and anemia. Diagnosis is based on evidence of anemia and hemolysis, together with detection of red cell autoantibodies, and patients commonly present with fatigue, pallor, jaundice, dark urine or splenomegaly. AIHA includes warm and cold subtypes, as well as mixed forms, and may be primary or secondary to conditions such as lupus, lymphoma or other malignancies. Current drug development is mainly focused on warm AIHA, which is more common and remains associated with significant unmet need due to steroid dependence and frequent relapse. The following table describes the AIHA drug market in China and globally from 2021 to 2035:

Global and China AIHA Drug Market Size, 2021-2035E



Source: Frost & Sullivan

INDUSTRY OVERVIEW

In China, the prevalence of AIHA reached 138.6 thousand in 2025, and is expected to reach 140.9 thousand in 2030 and 141.5 thousand in 2035, representing CAGRs of 0.4% from 2021 to 2025, 0.3% from 2025 to 2030 and 0.1% from 2030 to 2035. Globally, the prevalence of AIHA reached 806.9 thousand in 2025, and is expected to reach 865.4 thousand in 2030 and 924.2 thousand in 2035, representing CAGRs of 1.5% from 2021 to 2025, 1.4% from 2025 to 2030 and 1.3% from 2030 to 2035.

Treatment Paradigm of AIHA

In China, treatment of AIHA is tailored by subtype. For wAIHA, first line therapy uses glucocorticoids with or without rituximab. If response is inadequate, second line options include rituximab alone or in combination with bendamustine or fludarabine, ibrutinib for selected cases, and bortezomib. Third line strategies consider splenectomy and immunosuppressants such as cyclosporine A, sirolimus, azathioprine, mycophenolate mofetil, cyclophosphamide, and danazol. For cAIHA, complement inhibitors including sutimlimab, pegcetacoplan, and eculizumab are used, with supportive measures such as intravenous immunoglobulin and plasma exchange for IgM mediated disease. In the US, first-line wAIHA care often uses prednisone with or without rituximab, second-line adds rituximab-based combinations and emergency control with eculizumab or plasmapheresis, and emerging BTK inhibitor is being integrated as a novel option.

In AIHA, systemic corticosteroids are commonly used as first-line therapy; however, globally approximately 70%-80% of patients ultimately escalate to second-line or beyond treatments due to treatment failure, relapse or treatment-limiting toxicities. In China, studies indicate that approximately 65% of AIHA patients receive second-line or subsequent therapies. CX1440, as a BTK inhibitor, is intended to be positioned for patients with AIHA whose disease is inadequately controlled by corticosteroid therapy, with the addressable population therefore estimated to represent approximately 70%-80% of the overall AIHA patient population globally, and ~65% in China.

Competitive Landscape

As of the Latest Practicable Date, there were no approved BTK inhibitor for the treatment of AIHA in overseas markets and in China. As of the same date, there was one clinical-stage BTK inhibitor drug candidate under development for the treatment of AIHA in overseas markets and two clinical-stage BTK inhibitor drug candidate under development for the treatment of AIHA in China.

Competitive Landscape of BTK Inhibitor Pipelines for Treatment of AIHA in Overseas Markets

Genetic Name	Target	Company	Phase	Indications	First Posted Date
Rilzabrutinib	BTK	Sanofi	III	AIHA	2025-07-25

Source: Frost & Sullivan

China Competitive Landscape of BTK Inhibitor Pipelines for Treatment of AIHA

Genetic Name	Target	Company	Phase	Indications	First Posted Date
Rilzabrutinib	BTK	Sanofi	III	AIHA	2025-08-11
CX1440	BTK	Our Company	IND approval	AIHA	*

Source: CDE, Frost & Sullivan

Note*: CX1440 obtained Phase I clinical trial IND approval in 2025

INDUSTRY OVERVIEW

As of the Latest Practicable Date, no BTK inhibitor had been approved for AIHA in either overseas markets or China; among clinical-stage candidates, rilzabrutinib is the most clinically advanced (Phase III), while CX1440 obtained the IND approval in 2025 to initiate a Phase I clinical trial. As a BTK inhibitor, CX1440 is being developed as a mechanism-based BTK inhibitor for AIHA patients who are inadequately controlled by corticosteroid therapy, with the potential to provide a targeted treatment option and reduce reliance on prolonged corticosteroid use in this patient population. Compared with rilzabrutinib, CX1440 remains at an earlier stage of clinical development and has not yet generated clinical efficacy or safety data. Accordingly, its clinical profile and any potential comparative advantages over rilzabrutinib or other therapies remain to be established in clinical trials

OVERVIEW OF MOLECULAR GLUES

Molecular glues are small molecules that induce selective degradation of disease-relevant proteins by bringing them into proximity with E3 ubiquitin ligases, leading to ubiquitination and proteasomal destruction. Unlike PROTACs, they are monovalent, linker-free, and typically lower in molecular weight, which may support oral bioavailability and favorable pharmacokinetics. Their mechanism can enable high selectivity and expand the druggable space to targets lacking catalytic activity or clear binding pockets. Clinical proof of concept has been established by thalidomide analogs, and ongoing research is extending this approach to new ligases, multi-target degradation strategies, and AI-assisted design.

Growth Drivers and Future Trends of Molecular Glue

According to Frost & Sullivan, the primary growth drivers and future trends for the molecular glue drug market globally include:

- ***Scientific and Technical Drivers Shaping the Molecular Glue Drug Market.*** Molecular glues are small molecules that recruit E3 ligases to induce selective degradation of target proteins rather than merely inhibit them. Compared with PROTACs, they often offer better drug-like properties, oral bioavailability, and simpler synthesis and manufacturing. These advantages may lower production complexity and cost while improving developability and scalability, supporting broader use in oncology and autoimmune diseases and accelerating market growth.
- ***Pipeline Expansion and Broadening Therapeutic Scope for Molecular Glue Degraders.*** The molecular glue pipeline is expected to expand as more candidates enter clinical development, particularly in oncology, where they may address previously undruggable transcription factors and resistance-associated targets. Development is also extending into neurology and immunology. Emerging approaches include harnessing alternative E3 ligase systems such as KEAP1-CUL3 to redirect ubiquitin ligase activity toward selective degradation of additional disease-relevant proteins.

Concurrently, indication-level trends and drivers will create distinct trajectories across rheumatoid arthritis, inflammatory bowel disease and multiple sclerosis. Specifically:

- ***Rheumatoid Arthritis*** In China, rheumatoid arthritis is often marked by delayed diagnosis, inconsistent treatment, and unequal access to specialists, resulting in low remission rates and substantial disability. Conventional csDMARDs may provide insufficient control, while biologics, though effective, are injectable and can reduce adherence. Molecular glues offer a differentiated approach by selectively modulating disease-relevant proteins through reprogrammed protein-protein interactions, potentially expanding the range of treatable inflammatory pathways. As small molecules with potential oral dosing, they may better support long-term disease management by improving adherence, accessibility, and treatment sustainability across care settings.

INDUSTRY OVERVIEW

- ***Inflammatory Bowel Disease*** The global burden of IBD is increasing, with incidence rising in historically low-prevalence regions such as East Asia while remaining high in Western countries, reflecting the broader impact of industrialization and urbanization. Elderly-onset IBD is also becoming more common, creating added diagnostic and treatment challenges due to greater comorbidity and polypharmacy. As populations age and healthcare needs expand, the clinical and economic burden of IBD is expected to continue growing.
- ***Multiple Sclerosis*** Multiple sclerosis treatment remains challenging due to heterogeneous presentation, limited biomarkers, and therapies that reduce relapses but have limited impact on neurodegeneration and functional recovery. Treatment uptake also remains uneven. New mechanism-diverse approaches, including small-molecule strategies such as molecular glues, aim to more precisely modulate immune dysregulation, expand treatment options, and improve the durability, accessibility, and overall quality of long-term disease management.

Major Indications for Molecular Glues in Autoimmune Diseases

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent synovial inflammation that leads to synovial hypertrophy, joint swelling, pain, and progressive cartilage and bone damage with erosions. In China, the prevalence of RA reached 6.2 million in 2025, and is expected to reach 6.4 million in 2030 and 6.7 million in 2035, representing CAGRs of 0.71% from 2021 to 2025 and 0.84% from 2025 to 2035. Globally, the prevalence of RA reached 41.8 million in 2025, and is expected to reach 43.7 million in 2030 and 45.4 million in 2035, representing CAGRs of 0.97% from 2021 to 2025 and 0.84% from 2025 to 2035.

Inflammatory Bowel Disease (IBD) is a chronic immune-mediated condition arising from interactions between genetic susceptibility and environmental factors that dysregulate intestinal immune responses. In China, the prevalence of IBD reached 0.8 million in 2025 from 0.6 million in 2021, and is expected to reach 1.2 million in 2030 and 1.5 million in 2035, representing CAGRs of 7.6% from 2021 to 2025, 7.1% from 2025 to 2030 and 5.2% from 2030 to 2035. Globally, the prevalence of IBD reached 7.9 million in 2025 from 6.5 million in 2021, and is expected to reach 9.3 million in 2030 and 10.3 million in 2035, representing CAGRs of 5.0% from 2021 to 2025, 3.2% from 2025 to 2030 and 2.1% from 2030 to 2035.

Multiple sclerosis (MS) is a chronic immune-mediated disorder of the central nervous system in which inflammation damages myelin and eventually injures axons, disrupting signal conduction in the brain and spinal cord. In China, the prevalence of MS reached 54.7 thousand in 2025, and is expected to reach 60.8 thousand in 2030 and 69.0 thousand in 2035, representing CAGRs of 2.4% from 2021 to 2025, 2.1% from 2025 to 2030 and 2.6% from 2030 to 2035. Globally, the prevalence of MS reached 3,243.2 thousand in 2025, and is expected to reach 3,716.9 thousand in 2030 and 4,256.1 thousand in 2035, representing CAGRs of 2.8% from 2021 to 2025, 2.8% from 2025 to 2030 and 2.7% from 2030 to 2035.

SOURCE OF INFORMATION

We engaged Frost & Sullivan, a market research consultant, to prepare the Frost & Sullivan Report for use in this document. The information from Frost & Sullivan disclosed in this document is extracted from the Frost & Sullivan Report and is disclosed with the consent of Frost & Sullivan. In preparing the Frost & Sullivan Report, Frost & Sullivan collected and reviewed publicly available data such as government-derived information, annual reports, trade and medical journals, industry reports and other available information gathered by not-for profit organizations as well as market data collected by conducting interviews with industry KOLs. Frost & Sullivan has exercised due care in collecting and reviewing the information so collected and independently analyzed the information, but the accuracy of the conclusions of its review largely relies on the accuracy of the information collected. We agreed to pay Frost & Sullivan a fee of RMB580,000 for the preparation and update of the Frost & Sullivan Report, which is not contingent on the [REDACTED] proceeding.