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OVERVIEW OF METABOLIC DISORDERS AND ANTI-INFLAMMATORY DRUG MARKET

Introduction to Metabolic Disorders and Inflammatory Diseases

Metabolism is the complex biological process through which the human body converts food into essential components, such as proteins, carbohydrates (sugars), and fats, within the digestive system to produce energy. When a dysfunction occurs, it can lead to metabolic disorders, a broad category of diseases including gout, diabetes, obesity, non-alcoholic steatohepatitis, among others. Metabolic disorders and inflammation are closely interconnected. In fact, metabolic syndrome is often characterized by a persistent proinflammatory state. Meanwhile, factors such as overnutrition, physical inactivity, and aging can trigger chronic inflammation, resulting in excessive cytokine secretion that predisposes individuals to metabolic disorders.

Prevalence of Common Metabolic Disorders

Metabolic disorders represent a significant and growing public health concern both in China and globally, with conditions such as hypertension, hyperuricemia, obesity, type 2 diabetes mellitus ("**T2DM**") and metabolic dysfunction-associated steatohepatitis ("**MASH**") being some of the most prevalent. Hyperuricemia, one of the most common metabolic disorders worldwide, poses a significant public health challenge. The large and expanding patient population highlights the pressing need for effective management strategies, making hyperuricemia a critical public health concern. At the same time, it presents a compelling market opportunity where rising clinical awareness and improving diagnosis rates are expected to drive demand for effective and targeted treatments. As a result, the hyperuricemia treatment landscape offers significant potential for innovation and investment, underscoring the urgency and promise of addressing this growing burden.

Size of Metabolic Disorders and Anti-inflammatory Drug Market

The global metabolic disorders drug market has been experiencing steady growth, expanding from US$106.3 billion in 2019 to US$145.4 billion in 2024, at a CAGR of 6.5%. Such upward trajectory is expected to continue, with the market projected to reach US$225.7 billion by 2033, reflecting a CAGR of 5.0% from 2024 to 2033. Similarly, the metabolic disorders drug market in China has been growing consistently, increasing from US$13.3 billion in 2019 to US$16.4 billion in 2024, at a CAGR of 4.2%. Between 2024 and 2033, the market in China is forecasted to accelerate further with a CAGR of 9.2%, reaching an estimated US$36.2 billion by 2033. The rapid growth highlights the increasing demand for metabolic disorder treatments globally, with China emerging as a critical driver of market expansion due to its large patient population and rising healthcare investment.

In addition, the global anti-inflammatory drug market, supported by rising prevalence of chronic inflammatory diseases and advancements in therapeutic innovations, represents a significant and expanding segment within the pharmaceutical industry. Globally, the anti-inflammatory drug market expanded from US$16.1 billion in 2019 to US$17.7 billion in 2024, at a CAGR of 1.9%. Such global market size is projected to reach US$22.7 billion by 2033, representing a CAGR of 2.9% from 2024 to 2033. In China, the anti-inflammatory drug market has been experiencing steady growth, increasing from US$3.8 billion in 2019 to US$4.7 billion in 2024, at a CAGR of 4.1%. Such growth is expected to continue, with the market projected to reach US$6.9 billion by 2033, representing a CAGR of 4.4% between 2024 and 2033. The increasing demand for effective anti-inflammatory therapies in China positions the country as a key market for innovation and investment in this therapeutic area.

Growth Drivers and Future Trends of the Metabolic Disorders and Anti-inflammatory Drug Market

The primary growth drivers and future trends of the metabolic disorders and anti-inflammatory drug market include:

- *Expanding vulnerable populations and rising public awareness.* The prevalence of metabolic diseases, such as gout, is on the rise in China, with an alarming trend toward younger age groups, exacerbating the burden of healthcare costs. In response, the government and social organizations are actively advancing health education and awareness campaigns to improve public understanding of metabolic diseases.

- *Urgent clinical needs.* Traditional therapeutic regimens often demonstrate limited efficacy in refractory cases and fall short in effectively managing associated complications. This highlights an urgent need for innovative therapies that can provide precise, personalized treatment options while improving patient adherence.

- *Breakthroughs in novel targets and innovative therapies.* In recent years, advancements in the development of drugs for metabolic diseases have been driven by a deeper understanding of disease mechanisms and the integration of cutting-edge technologies. For example, urate transporter 1 ("**URAT1**") inhibitors have been precisely designed to target gout. In addition, the application of AI and machine learning in drug discovery has accelerated the development of innovative therapies.

- *Policy support and optimized payment environment.* Policy support and an improved payment environment are pivotal drivers of growth in the metabolic disease drug market. For example, the Chinese government places significant emphasis on the prevention and treatment of metabolic diseases, integrating them into the national health strategy and enhancing treatment accessibility.

- *Improvement of drug safety.* Pharmaceutical companies are dedicating efforts to developing safer and more effective treatments for metabolic diseases. In the area of gout, for instance, many existing therapies face major safety concerns. Looking ahead, drug R&D will prioritize reducing side effects and improving patient adherence. Strategies such as optimizing molecular structures and developing long-acting formulations aim to enhance drug safety and efficacy.

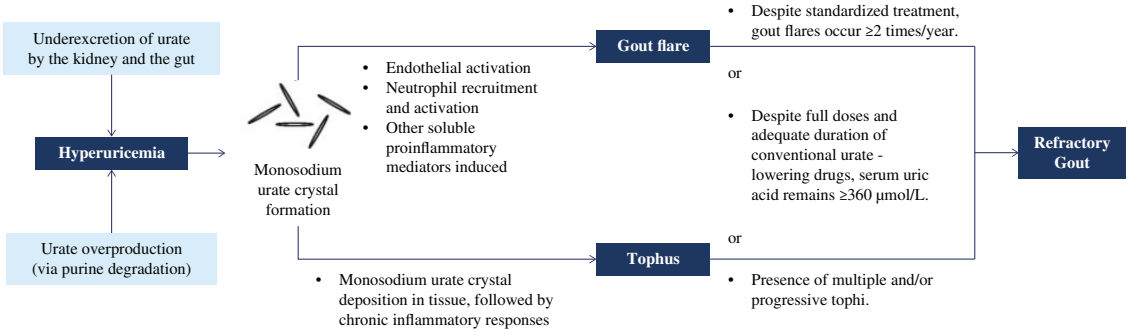
- *More systematic treatments.* Metabolic disorders often lead to cardiovascular and hepatic complications, prompting a shift in pharmaceutical development from targeting single indicators to delivering comprehensive therapeutic benefits. For example, colchicine not only acute gout flares but also reduces atherosclerotic plaque, offering dual benefits.

- *Increased market share of domestic products.* With the increasing prevalence of metabolic disorders in China and supportive health insurance policies, domestic innovative drugs are expected to rapidly replace imported treatments. China-based pharmaceutical companies are making significant strides in molecular structure optimization and long-acting formulations development, achieving breakthroughs in key areas such as URAT1 inhibitors. Health insurance policies favor domestic innovations, and reforms in pricing standards allow innovative drugs to be independently priced within reasonable limits, further incentivizing R&D investments.

OVERVIEW OF GOUT DRUG MARKET

Introduction to Gout

Hyperuricemia is characterized by an elevated serum uric acid ("**sUA**") level, typically exceeding 6 mg/dL in women and 7 mg/dL in men. This condition arises from reduced uric acid ("**UA**") excretion, increased UA production or a combination of both. According to Frost & Sullivan, reduced UA excretion accounts for approximately 90% of hyperuricemia. Prolonged hyperuricemia can lead to gout, a condition caused by the formation and deposition of monosodium urate crystals in joints and other tissues. Gout is the most common form of inflammatory arthritis and is often associated with severe pain, swelling, and recurrent flare-ups. While gout and hyperuricemia are not exactly the same indication, they are closely related conditions within the same disease spectrum and are commonly grouped together in clinical research and medical practice. Hyperuricemia is a metabolic abnormality and the biochemical prerequisite and principal risk factor for the occurrence of gout. Refractory gout, a more severe and challenging form of the disease, is defined by two key characteristics: persistent clinical symptoms and the inability to lower sUA levels below the therapeutic target of 6 mg/dL, despite the use of urate-lowering therapies ("**ULTs**"). The development of refractory gout is linked to various mechanisms, all of which relate to suboptimal responses to or limitations of current ULTs. The following chart illustrates the pathological mechanism of hyperuricemia and gout.



Source: Gout (Dalbeth, Nicola et al. The Lancet, Volume 397, Issue 10287, 1843-1855), Frost & Sullivan analysis

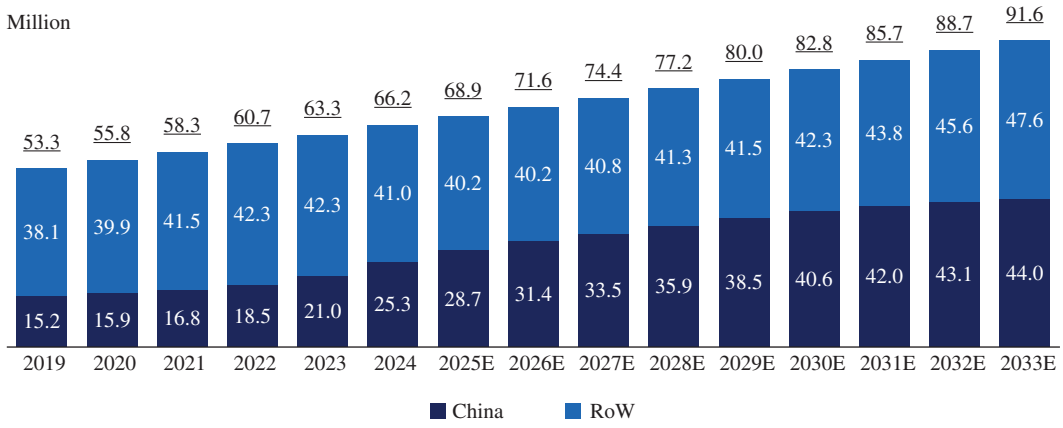
Hyperuricemia and gout are chronic, systemic conditions that affect multiple organs and are independent risk factors for chronic kidney disease ("**CKD**"), hypertension, cardiovascular disease, and diabetes. Hyperuricemia and gout are causally linked with kidney stones and CKD, while elevated sUA is also an independent risk factor for cardiovascular and cerebrovascular diseases and diabetes. Across observational studies, each 60 μmol/L increase in sUA is associated with roughly a 1.4-fold higher risk of hypertension, a 17% higher risk of new-onset diabetes, and a 12% higher risk of coronary heart disease mortality. Interventional studies suggest that ULT can improve renal function and slow CKD progression in hyperuricemic patients with renal damage and can reduce systolic and diastolic blood pressure in hyperuricemic patients with hypertension.

Prevalence of Gout

The global burden of gout has been rising steadily over the past several years. The number of patients increased from 53.3 million in 2019 to 66.2 million in 2024, representing a CAGR of 4.4%. Looking ahead, the global prevalence is projected to reach 91.6 million by 2033, corresponding to a CAGR of 3.7% between 2024 and 2033. In China, the growth trend has been even more pronounced. The gout patient population expanded from 15.2 million in 2019 to 25.3 million in 2024, at a CAGR of 10.7%. By 2033, this figure is expected to surge further to 44.0 million, reflecting a CAGR of 6.4% from 2024 to 2033. The following chart sets forth the historical and projected prevalence of gout globally and in China from 2019 to 2033.

Global and China Prevalence of Gout, 2019-2033E

Period	CAGR		
	China	RoW	Global
2019-2024	10.7%	1.5%	4.4%
2024-2033E	6.4%	1.7%	3.7%



Source: Frost & Sullivan Analysis

The higher historical and forecast CAGRs of the gout patient population in China compared with the rest of the world mainly reflect China's ongoing lifestyle transition, lower starting base, and demographic momentum. Rapid urbanization and rising incomes have driven dietary shifts toward greater consumption of meat, seafood, alcohol, and processed foods, coupled with reduced physical activity, resulting in increasing rates of obesity, metabolic syndrome, and hyperuricemia, the main risk factors for gout. Starting from a much lower baseline (below 1% prevalence in 2010 compared with 3-4% in developed markets), China is experiencing a faster epidemiological catch-up as its prevalence rises toward levels already reached in Western countries. In addition, China's large and aging population magnifies the absolute increase in gout cases, whereas most developed regions already have older populations and stable prevalence levels, leading to slower incremental growth.

Gout Drug Market Size

The global gout drug market declined from US$3.1 billion in 2019 to US$2.7 billion in 2024, primarily due to drug safety concerns of febuxostat and the decrease in sales volume resulting from increased drug prices. However, it is expected to rebound significantly, reaching US$9.3 billion by 2033, with a CAGR of 14.9% from 2024 to 2033. The gout drug market in China also once saw a decline, with its size shrinking from US$0.5 billion in 2019 to US$0.3 billion in 2024, largely due to the impact of centralized drug procurement policies, which reduced drug prices. However, the China market is expected to recover rapidly, reaching US$1.7 billion by 2033 at a 20.4% CAGR, outpacing global growth. Such expansion is driven by rising prevalence, improved awareness, and new innovative therapies. In addition, in the U.S., the gout drug market increased from US$1.5 billion in 2019 to US$2.0 billion in 2024, representing a CAGR of 5.6%, primarily driven by the enhanced sales of urate oxidase. The U.S. market size is expected to further increase to US$5.0 billion in 2033, representing a CAGR of 10.8% from 2024 to 2033, which is in line with the global growth trajectory.

Specifically, between 2019 and 2024, the gout drug market in China underwent a period of adjustment. Although the addressable patient population continued to expand due to rising prevalence of metabolic disorders and increasing disease awareness, the overall market size slightly contracted as established drugs faced intensified generic competition following patent expirations and the implementation of volume-based procurement ("**VBP**") policies covering both ULT and acute gout flare drugs. These policies led to a significant decline in average drug prices, even as total prescription volumes increased. In addition, the overall disruption to medical service

utilization during the COVID-19 outbreak exerted temporary pressure on market demand. Looking ahead from 2024 to 2028 (the year when several indications of ABP-671 and ABP-745 are expected to enter Phase 3 clinical trail or commercialization), the China market is expected to resume moderate growth, supported by the anticipated introduction of novel therapies with improved safety and efficacy that are unlikely to be immediately subject to VBP. As public awareness and early diagnosis continue to improve and patient demographics shift younger, patients are expected to place greater emphasis on clinical efficacy, safety and quality of life rather than solely on drug price, fostering stronger uptake of new urate-lowering and anti-inflammatory therapies.

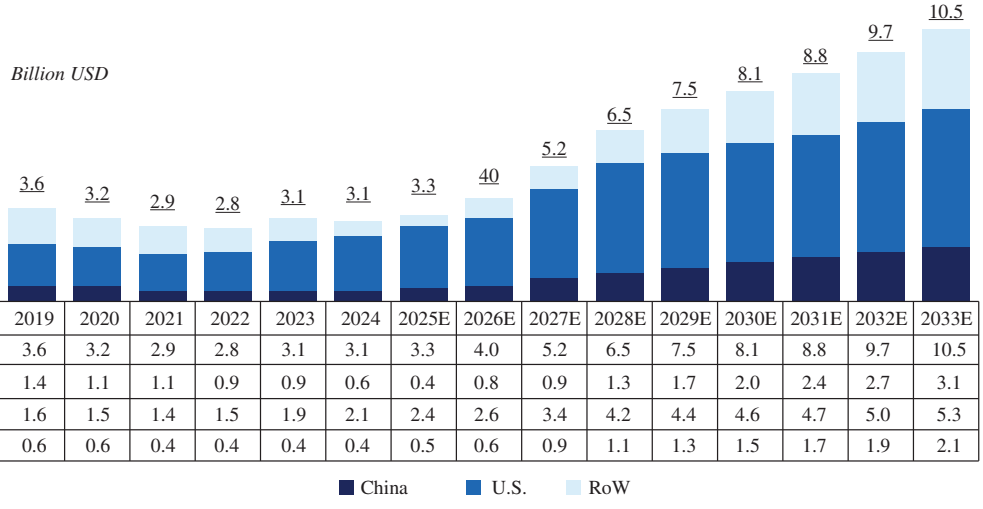
Globally, between 2019 and 2024, pricing and utilization patterns were shaped by exceptional policy-driven events rather than underlying demand dynamics. The FDA's Unapproved Drugs Initiative temporarily allowed a single manufacturer market exclusivity for colchicine, leading to a steep but non-recurring price surge and resort to alternative therapies. Meanwhile, the 2019 FDA boxed warning on febuxostat for cardiovascular risk reduced its global use and dampened revenues across the ULT segment. Despite these short-term disruptions, the global gout patient base continued to grow, reflecting ageing populations, lifestyle changes and higher disease recognition. From 2024 to 2028, the global gout drug market is expected to stabilize and gradually expand, driven by the recovery of demand fundamentals, the entry of next-generation URAT1 inhibitors and new anti-inflammatory therapies with improved safety, and demographic trends showing a younger, more health-conscious patient population. These factors together support sustained volume and value growth in both chronic urate-lowering and acute flare-management segments.

Compared to the expected global growth trend, the higher growth rate of the gout drug market in China is primarily driven by the continuing rise in disease prevalence resulting from lifestyle factors, low current treatment adherence rates (less than 4% after 24 weeks), improving diagnosis and patient management, and the introduction of next-generation URAT1 inhibitors, biologics, and siRNA-based therapies supporting higher per-patient spending. Globally, while the market will also benefit from steady patient population growth, broader guideline adoption, and the uptake of innovative and premium-priced therapies, the overall growth rate is expected to be slower than that of China, reflecting a more mature treatment landscape, higher baseline adherence levels, and a comparatively stable pricing environment.

The following chart sets forth the historical and projected market size of gout and hyperuricemia drugs in the U.S., China and globally from 2019 to 2033.

Global Gout with Hyperuricemia Drug Market, 2019-2033E

Period	CAGR			
	China	U.S.	RoW	Global
2019-2024	-7.8%	5.3%	-16.1%	-3.2%
2024-2033E	20.2%	10.9%	20.8%	14.7%



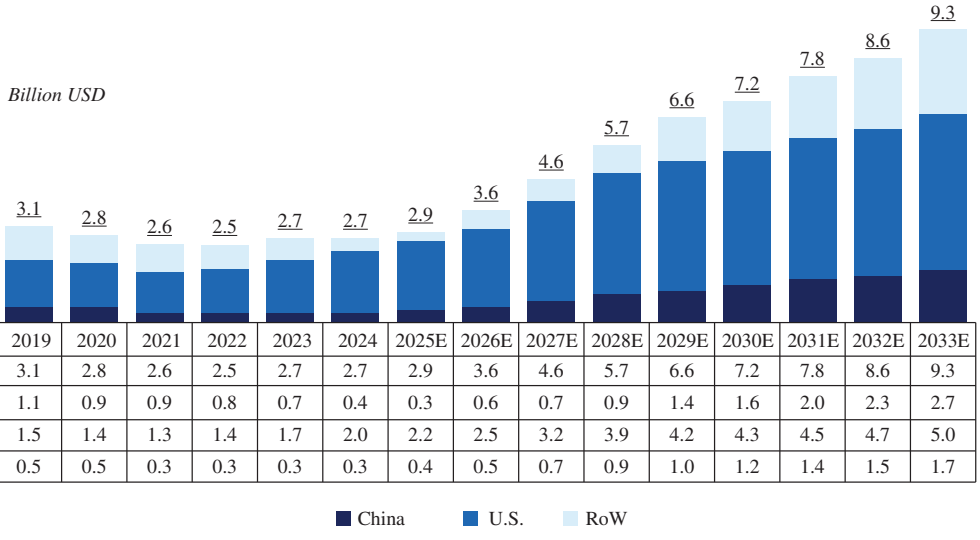
	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	3.6	3.2	2.9	2.8	3.1	3.1	3.3	4.0	5.2	6.5	7.5	8.1	8.8	9.7	10.5
RoW	1.4	1.1	1.1	0.9	0.9	0.6	0.4	0.8	0.9	1.3	1.7	2.0	2.4	2.7	3.1
U.S.	1.6	1.5	1.4	1.5	1.9	2.1	2.4	2.6	3.4	4.2	4.4	4.6	4.7	5.0	5.3
China	0.6	0.6	0.4	0.4	0.4	0.4	0.5	0.6	0.9	1.1	1.3	1.5	1.7	1.9	2.1

Source: Frost & Sullivan Analysis

In addition, the following chart sets forth the historical and projected market size of gout drugs in the U.S., China and globally from 2019 to 2033.

Global Gout Drug Market, 2019-2033E

Period	CAGR			
	China	U.S.	RoW	Global
2019-2024	-7.7%	5.6%	-19.1%	-3.0%
2024-2033E	20.4%	10.8%	23.6%	14.9%



	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	3.1	2.8	2.6	2.5	2.7	2.7	2.9	3.6	4.6	5.7	6.6	7.2	7.8	8.6	9.3
RoW	1.1	0.9	0.9	0.8	0.7	0.4	0.3	0.6	0.7	0.9	1.4	1.6	2.0	2.3	2.7
U.S.	1.5	1.4	1.3	1.4	1.7	2.0	2.2	2.5	3.2	3.9	4.2	4.3	4.5	4.7	5.0
China	0.5	0.5	0.3	0.3	0.3	0.3	0.4	0.5	0.7	0.9	1.0	1.2	1.4	1.5	1.7

Source: Frost & Sullivan Analysis

Major Therapeutic Options for Gout

The treatment of gout primarily relies on three types of medications: XO inhibitors, URAT1 inhibitors, and urate oxidase. XO inhibitors, including allopurinol, febuxostat, and topiroxostat, work by reducing UA production. URAT1 inhibitors, such as probenecid, benzbromarone, lesinurad and dotinurad, function by promoting the excretion of UA through the kidneys. Moreover, urate oxidase, represented by pegloticase, lowers UA levels by converting it into allantoin, a more soluble and easily excreted compound, and is primarily utilized as a last-line therapy for refractory gout.

The table below sets forth the principal advantages and limitations of the three major classes of ULT — XO inhibitors, URAT1 inhibitors and urate oxidase.

Drug Type	Advantages	Limitations
XO Inhibitors	• Reduce UA production at the source • Generally available at lower cost	• Most effective in patients with UA overproduction, who represent only approximately 10% of gout cases • Potential allergic reactions and elevated risk of cardiovascular events • Liver toxicity
URAT1 Inhibitors	• Effective in patients with impaired UA excretion, representing approximately 90% of gout cases • Demonstrate high efficacy, suitable for monotherapy or combination therapy with XO inhibitors • Generally cost-effective	• May increase the risk of urinary tract stones • Require monitoring of urine pH
Urate Oxidase	• Rapidly degrades UA and can be effective for refractory gout	• Protein with high immunogenicity, easy to severe hypersensitivity and cause infusion reactions, with FDA black warning • Intravenous administration, which can be inconvenient for patients • Low response rate

Source: Literature review, Frost & Sullivan analysis

Overview of URAT1 Inhibitors

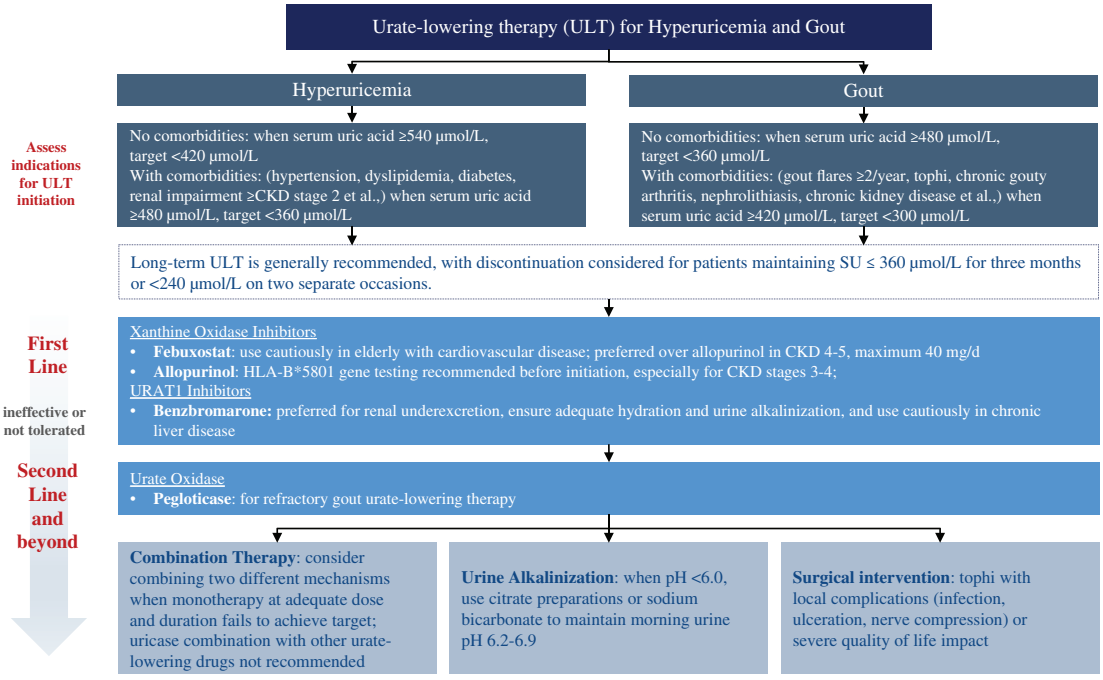
URAT1 is a key protein involved in the reabsorption of UA, responsible for approximately 90% of UA transport in the kidneys. This protein is expressed on the apical membrane of the proximal tubule in the human kidney, where it mediates the exchange of UA with other anions. URAT1 inhibitors work by competitively occupying the urate-binding cavity of the transporter, effectively blocking the UA/anion exchange process. Such action reduces UA reabsorption, thereby promoting its excretion and lowering sUA levels. Both classical URAT1 inhibitors, such as benzbromarone and probenecid, and next-generation agents, including lesinurad and dotinurad, share this mechanism of action. However, they differ in terms of potency, selectivity and safety profiles. As of the Latest Practicable Date, four URAT1 inhibitors had been commercially available worldwide, representing different generations of development. The first-generation drugs probenecid and benzbromarone have been widely used for decades, while newer agents like lesinurad and dotinurad offer improved pharmacological profiles.

For details on current marketed drugs and investigational drugs of URAT1 inhibitors, see "— Competitive Landscape of Gout Drugs."

Current Treatment Paradigms for Hyperuricemia and Gout

The treatment of hyperuricemia and gout in China follows guideline-recommended urate-lowering strategies tailored to patients' sUA levels and comorbidities. For hyperuricemia, the sUA target is <420 μmol/L for patients without comorbidities and <360 μmol/L for those with conditions like hypertension, diabetes, or renal impairment. In gout, the target is <360 μmol/L without comorbidities and <300 μmol/L for patients with frequent flares, tophi, or CKD. First-line therapies include XO inhibitors such as allopurinol, with HLA-B*5801 gene testing (an allele test associated with the risk of severe adverse skin reactions induced by allopurinol and genetic susceptibility to gout) recommended, and febuxostat, preferred in advanced CKD. URAT1 inhibitors like benzbromarone are used cautiously, while urate oxidase (e.g., pegloticase) is reserved for refractory gout. Combination therapy is advised when monotherapy fails, alongside urine alkalinization and surgical intervention for tophi-related complications. This structured approach ensures effective and individualized care. The following chart sets forth the treatment paradigm of hyperuricemia and gout in China.

Treatment Paradigm of Hyperuricemia and Gout in China



Source: Chinese Guidelines for the Diagnosis and Treatment of Hyperuricemia and Gout (2024 Edition), Frost & Sullivan analysis

The treatment paradigm for hyperuricemia and gout in the U.S. centers on the appropriate initiation and escalation of ULT, based on disease severity, comorbidities, and flare frequency. ULT is strongly recommended for patients with one or more subcutaneous tophi, radiographic evidence of gout-related joint damage, or frequent flares (defined as two or more per year). Conditional recommendations apply to patients with infrequent flares (fewer than two per year), or those experiencing a first flare in the context of moderate-to-severe CKD (stage ≥3), sUA exceeding 9 mg/dL, or a history of urolithiasis. First-line pharmacologic therapy includes XO inhibitors, such as allopurinol, preferred even in patients with advanced CKD, with starting doses ≤100 mg/day, and febuxostat, especially for those with CKD stage ≥3 or intolerance to allopurinol. URAT1 inhibitors, like probenecid, may be used as an alternative to XO inhibitors. For patients with refractory disease, persistent tophi, or frequent flares despite conventional therapy, urate oxidase (e.g., pegloticase) may be considered as a second-line or advanced option. Concurrently, allopurinol is recommended even in moderate-to-severe CKD, and ULT should be initiated in all patients with gout-related radiographic damage. Lifestyle modifications are also advised, including limiting alcohol, purine-rich foods, and high-fructose corn syrup, and promoting weight loss in overweight or obese individuals.

Overview of gout drug market by drug types

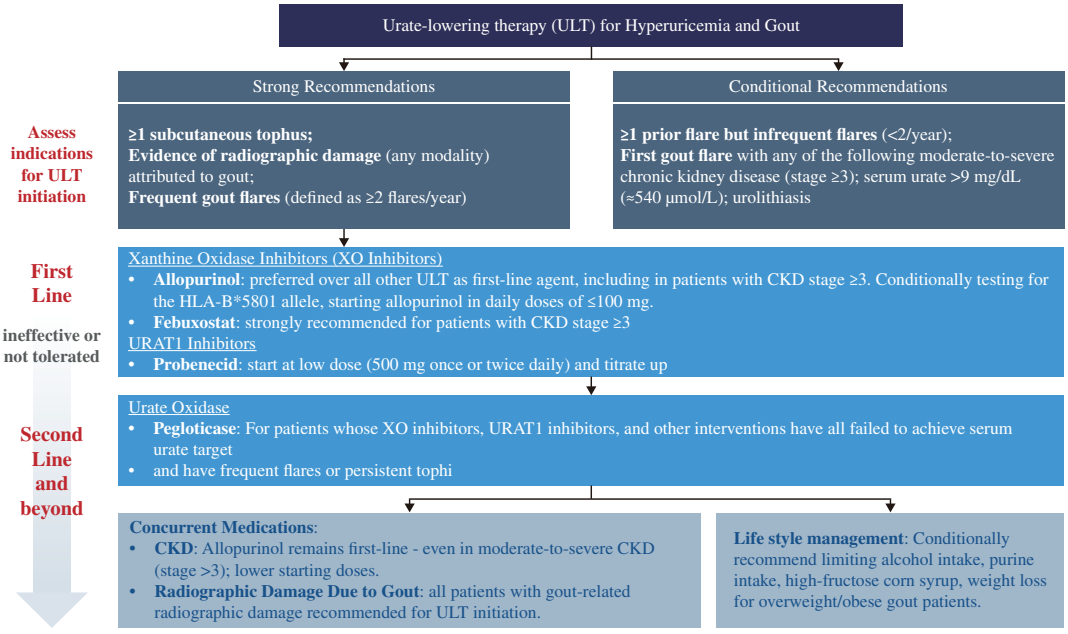
The global market for URAT1 inhibitors increased from USD0.1 billion in 2019 to USD0.4 billion in 2024, representing a CAGR of 34.9%, and is expected to further grow to USD2.3 billion by 2033, representing a CAGR of 22.2% from 2024 to 2033. The global market for recombinant uricases increased from USD0.3 billion in 2019 to USD1.2 billion in 2024, representing a CAGR of 29.2%, and is expected to further grow to USD3.8 billion by 2033, representing a CAGR of 13.3% from 2024 to 2033. By contrast, the global market for XOIs decreased from USD2.7 billion in 2019 to USD1.1 billion in 2024, representing a CAGR of -16.8%, and is expected to remain relatively stable at USD1.1 billion by 2033, representing a CAGR of 0.7% from 2024 to 2033.

In China, the market for URAT1 inhibitors decreased from RMB0.5 billion in 2019 to RMB0.4 billion in 2024, representing a CAGR of -3.7%, and is expected to grow significantly to RMB4.7 billion by 2033, representing a CAGR of 29.8% from 2024 to 2033. As no recombinant uricase product had been approved and commercially launched in China as of the Latest Practicable Date,

there was no observable market for recombinant uricases in China. The market for XOIs in China decreased from RMB2.8 billion in 2019 to RMB1.9 billion in 2024, representing a CAGR of -7.7%, and is expected to remain relatively stable at RMB1.9 billion by 2033, representing a CAGR of 0.2% from 2024 to 2033.

According to Frost & Sullivan, the forecast growth rates for the respective drug types are primarily based on their different clinical positioning, safety profiles and market adoption dynamics.

Treatment Paradigm of Hyperuricemia and Gout in the U.S.



Abbreviations: XO = xanthine oxidase

Source: ACR Guideline for the Management of Gout (2020), Frost & Sullivan analysis

In China, treatment for gout generally includes XO inhibitors (e.g., allopurinol, febuxostat) and URAT1 inhibitors such as benzbromarone to promote UA excretion. In the U.S., the treatment predominantly involves xanthine oxidase inhibitors, with benzbromarone not approved by the FDA and unavailable due to concerns over hepatotoxicity. Both China and the U.S. recommend XO inhibitors, primarily allopurinol and febuxostat, as first-line ULT. Uricosurics, such as benzbromarone in China and probenecid in the U.S., serve as alternatives or adjunctive options, while urate oxidase (pegloticase) is reserved as second-line treatment for refractory cases in both countries. Despite these similarities, current strategies face important limitations: genetic testing or careful monitoring is required before initiating allopurinol due to hypersensitivity risks; febuxostat raises cardiovascular safety concerns; URAT1 inhibitors demand strict patient selection and carry organ-specific risks; and pegloticase is generally limited to severe, treatment-resistant gout because of cost and safety issues. These constraints highlight a clear unmet medical need for safer, more effective, and broadly applicable innovative therapies for hyperuricemia and gout management.

In addition, non-pharmacological treatments and prevention methods form an integral part of the management landscape and may complement drugs, such as dietary management in the form of limiting purine, fructose and alcohol intake), regular exercise, weight management, and urine alkalization (e.g., with citrate or sodium bicarbonate) to enhance UA excretion.

Competitive Landscape of Gout Drugs

Global Marketed Drugs for Gout

As of the Latest Practicable Date, there were four originator drugs (i.e., benzbromarone, probenecid, febuxostat, and allopurinol) that have approved generics and over one hundred generic drugs approved and commercially available worldwide for the treatment of gout. The following table sets forth the details of global marketed drugs for gout, some of which have been withdrawn from the market in one or more jurisdictions, as of the Latest Practicable Date.

Global Marketed Drugs for Gout

Drug Type (Targets)	Ingredient	Originator Brand Name	Modality	Company	Treatment Line	Year of Approval	Compound IP status	China Reimbursement	Price per Tablet in China (RMB)	Price per Tablet in US (USD)
Xanthine Oxidase Inhibitor (XO inhibitor)	Allopurinol (別嘌醇)	Zyloprim	Small molecule	GSK	1L	1966 (FDA) 1969 (PMDA) 1995 (NMPA, generics)	Expired	A / B	0.1g:1.53 (NRDL Category A); 0.25g:4.2 (NRDL Category B)	0.07-0.29 (100mg)
	Febuxostat (非布司他)	Uloric	Small molecule	Takeda	1L	2008 (Europe) 2009 (FDA, withdraw) 2011 (PMDA) 2018 (NMPA)	Expired	B	40mg:2.4;20mg: 2.24;80mg:9.8 (NRDL Category B)	1.27-1.45 (40mg)
	Topiroxostat (托匹司他)	Topiloric	Small molecule	Fuji Yakuhin	1L	2013 (PMDA)	Expired	/	/	/
URAT1 inhibitor	Probenecid (丙磺舒)	Benemid	Small molecule	Merck	2L	1951 (FDA, withdraw) 2006 (NMPA, generics)	Expired	B	NA	0.6 (500mg)
	Benzbromarone (苯溴馬隆)	Narcaricin	Small molecule	Sanofi	2L	1971 (Europe, withdraw) 2004 (NMPA, generics)	Expired	B	50mg:2.03 (NRDL Category B)	/
	Lesinurad (雷西納德)	Zurampic	Small molecule	Ironwood/ AZ	2L	2015 (FDA, withdraw) 2016 (EMA, withdraw)	Expired	/	/	13 (200mg)
	Dotinurad (多替諾雷)	Urece	Small molecule	Fuji Yakuhin/ Eisai	2L	2020 (PMDA) 2024 (NMPA)	Valid until 2030	/	未進入醫保：2mg: 19.96	/

Source: FDA, NMPA, Frost & Sullivan analysis

Among the marketed drugs mentioned above with generic counterparts, there were approximately five generic approvals for probenecid, six for benzbromarone, 24 for allopurinol, and 41 for febuxostat in China as of the Latest Practicable Date. In the U.S., based on the ANDA application number, as of the Latest Practicable Date, approximately 20 generic drugs have been approved for probenecid, 29 for allopurinol, and 16 for febuxostat. However, the U.S. has not approved any generic drugs for benzbromarone.

The clinical performance of marketed drugs for gout varies across different drug classes and individual agents. XO inhibitors, like allopurinol and febuxostat, show moderate to high efficacy in reducing sUA levels but are associated with side effects such as liver function abnormalities and cardiovascular risks. URAT1 inhibitors, including benzbromarone, lesinurad and dotinurad, demonstrate strong efficacy, with certain agents achieving sUA target rates above 80%. However, safety concerns like renal impairment and different other forms of adverse drug reactions are noted. The following table sets forth the clinical performance of marketed gout drugs.

Clinical Performance of Marketed Gout Drugs

	Originator Drug	Company	Dosage	Efficacy		Safety	
				% of subjects achieving sUA <6.0 mg/dL	Trial Duration	AEs	Black Box Warning
Allopurinol	Zyloprim	Casper Pharma/ GSK	100-800 mg qd	42% (200/300 mg)	6 months	AE=37% including: upper respiratory tract infections (16.85%) joint related signs and symptom (6.74%) musculoskeletal (12.92%)	N/A
				36% (300 mg)	12 months		
Febuxostat	Uloric	Takeda	40-80 mg qd	45% (40 mg) 67% (80 mg)	6 months	Liver Function Abnormalities (6.6%/4.6%) Nausea (1.1%/1.3%) Arthralgia (1.1%/0.7%) Rash (0.5%/1.6%)	In a cardiovascular outcomes study, among gout patients with established cardiovascular disease, those treated with Uloric had a higher rate of cardiovascular mortality compared with patients treated with allopurinol
				74% (80 mg)	12 months		
Topiroxostat	Topiloric	Fuji Yakuhin	20-80 mg bid	72.4% (120 mg/d)	16 weeks	SAE=2%, AE=97% including: Blood triglycerides increased (42%) Urinary α1-microglobul increased (34%) Urinary NAG increased (25%)	N/A
Probenecid	Benemid	Merck	250-500 mg bid	/	/	headache, dizziness, precipitation of acute gouty arthritis, Gastrointestinal events, Genitourinary events, Hypersensitivity, Hematologic and Integumentary events	N/A
Benzbromarone	Narcaricin	labaz/Sanofi	25-100 mg qd	83.6 (50 mg)	14 weeks	AE=40.4% adverse drug reactions=15.2% Gouty arthritis (5.1%)	N/A
Lesinurad	Zurampic	Ironwood/ AZ	200 mg qd	54% (200 mg combo with Allopurinol) 57% (200 mg combo with Febuxostat 80 mg)	6 month	Headache (5.3%), influenza (5.1%), blood creatinine increased, and gastroesophageal reflux disease (2.7%)	Acute renal failure
Dotinurad	Urece	Fuji Yakuhin/ Eisai	1 mg qd for 4 weeks, followed by 2 mg qd for 8 weeks, followed by 4 mg qd for 12 weeks	73.6%	24 weeks	TEAE 90.6%, TEAEs leading to study drug dose adjustment 4.5%, urinary calculi 6.3%, liver injury 26.0%, renal injury 8.1%	N/A
			1 mg qd for 4 weeks, followed by 2 mg qd for 8 weeks	55.5%	12 weeks		

Abbreviations: AE = adverse event; SAE = serious adverse event; NAG = N-acetyl-β-D-glucosaminidase; TEAE = treatment emerged adverse event

Source: Drug label, literature review, Frost & Sullivan analysis

Gout Drugs under Development

In addition to the currently marketed drugs, the global competitive landscape for gout treatment is increasingly focused on the development of URAT1 inhibitors. As of the Latest Practicable Date, there were 18 drug candidates in development for the treatment of gout with clinical trials having been conducted in either of the U.S., the EU and China. Among these candidates with active clinical trials in China and globally, ABP-671 stands out as one of the fastest-progressing candidates both globally and in China. It also demonstrates potentially superior clinical performance according to indirect comparison using real-world data. The following table sets forth the competitive landscape of gout drugs under development that have conducted clinical trials in China, the U.S. or the EU as of the Latest Practicable Date.

Gout Drugs under Development

Drug Name	Modality	Company	Target	Treatment Line	Phase of Trial (U.S. and Europe)	Phase of Trial (China)	First Post Date	Indications
Lingdolinurad (ABP-671)	small molecule	Atom Therapeutics (新元素藥業)	URAT1	1L	Phase 2b/3	Phase 2b/3	2023/4/18	Gout
Ruzinurad (SHR4640)	small molecule	Hengrui (恒瑞醫藥)	URAT1	1L	/	NDA	2025/1/9	Primary Gout with hyperuricemia
Puliginurad (YL-90148)	small molecule	YL pharma (璞黎藥業)	URAT1	1L	/	Phase 3	2022/12/14	Primary Gout with hyperuricemia
Epaminurad (URC102)	small molecule	JW Pharmaceutical (江泰製藥)	URAT1	1L	Phase 3	/	2023/4/18	Gout
Pozdeutinurad (AR882)	small molecule	Arthrosi/ApicHope (一品紅藥業)	URAT1	1L	Phase 3	Phase 2/3	2024/6/3	Gout
HZBio1	biologic	CGE (遠大生物)/ PegBio (派金生物)	UA	2L	/	Phase 2/3	2025/12/2	Gout
Tigulixostat	small molecule	Innovent (信達生物)	XO	2L	Phase 3[(1)]	Phase 3	2026/2/25	Gout with hyperuricemia
Tainingnad	small molecule	Tianjin institute of Pharmaceutical (天津藥物研究院)	URAT1	1L	/	Phase 2b	2025/4/16	Hyperuricemia with or without gout
WXSH0493	small molecule	Kanion (康緯藥業)	URAT1	1L	/	Phase 2b	2025/7/25	Gout
THDBH130	small molecule	THDB (通化東寶)	URAT1	1L	/	Phase 2a complete	2022/11/17	Gout
THDBH151	small molecule		URAT1, XO	1L	/	Phase 2a complete	2024/3/26	Gout
D0120	small molecule	InventisBio (益方生物)	URAT1	1L	Phase 2	Phase 2	2022/6/28	Global: Gout and Hyperuricemia (in separate Phase 2 trials) China: Primary hyperuricemia
HY-0902	small molecule	Haiya (海雅醫藥)	URAT1	1L	/	Phase 2	2025/3/7	Gout with hyperuricemia
QJ-19-0002	small molecule	Chiatai Qingjiang (正大清江)	URAT1	1L	/	Phase 2	2025/8/11	Gout with hyperuricemia
HEC93077	small molecule	HEC (東陽光)	URAT1, XO	1L	/	Phase 1 complete	2021/11/17	Gout with hyperuricemia
F012	biologic	Lunan (新時代藥業)	UA	1L	/	Phase 1	2024/6/17	Hyperuricemia with or without gout
YJH-012	siRNA	LIVZON (麗珠醫藥)	XO	1L	/	Phase 1	2025/6/26	Gout with hyperuricemia
F-02-2-Na	small molecule	New Creative (新創意生物)	NA	1L	/	Phase 1	2025/11/21	Hyperuricemia with or without gout

Note:

(1) The Phase 3 trial of Tigulixostat was suspended in U.S.

(2) In this table, only clinical trials conducted in the U.S., the EU and China are included. Therefore, PRX-115 is intentionally excluded from the analysis, since its trial sites were not clear as of the Latest Practicable Date.

Source: Clinicaltrials, NMPA, Frost & Sullivan analysis

The large number of candidates in development underscores the substantial unmet need in this market and validates the commercial opportunity. Our ABP-671 demonstrates superior efficacy with 100% of subjects achieving target sUA levels (<6.0 mg/dL) at both 4 mg BID and 8 mg QD within just 4 weeks, outperforming other investigational drugs. Its safety profile is also favorable, with no severe adverse events ("**SAEs**)" and very few Grade 3 treatment-emergent adverse events ("**TEAEs**") reported, which were generally mild and similar across groups, with no hepatic or cardiovascular safety signals observed. The combination of rapid, high efficacy and a favorable safety profile positions ABP-671 as a leading candidate among investigational drugs for gout treatment. The table below summarizes key clinical data for comparable gout drugs under clinical investigation as of the Latest Practicable Date.

Clinical Performance of Selected Gout Drugs under Clinical Development

Drug Name	Company	Target	Efficacy			Safety
			Cohort dosage	% of subjects achieving sUA <6.0 mg/dL	Trial Duration	
Lingdolinurad (ABP-671)	Atom Therapeutics	URAT1	8 mg QD	100.0%(1)	4 weeks	Overall incidence of TEAEs was similar between the ABP-671 group and the placebo group. TEAEs in the ABP-671 group were all mild or moderate in severity, with no SAEs or severe TEAEs reported. Overall incidence of nephrolithiasis was 5%, including 16.7% in the placebo group and 2.1% in the ABP-671 group.
			4 mg BID	100.0%(1)		
			4 mg QD	87.5%(1)	8 weeks	
			2 mg BID	91.9%(2)		
Epaminurad (URC102)	JW Pharmaceutical	URAT1	9 mg	88.9%	4 weeks	TEAE=52.78%, ADR=25%, no SAE; Gout flare incidence rate is 22.22% in 12 weeks.
				88.9%	8 weeks	
				75.0%	12 weeks	
Pozdeutinurad (AR882)	Arthrosi	URAT1	75 mg	92.0%	3 months	SAE=7.1%, during the first 6 months of treatment, gout flare (78.6%), arthralgia (28.6%)
				85.0%	12 months	
				83.0%	18 months	
Ruzinurad (SHR4640)	Hengrui	URAT1	10 mg	52.6%	16 weeks	During the 52-week TEAEs occurred in 89.7% patients, with gout flare (51%), increased alanine aminotransferase (21.6%), upper respiratory tract infection (21.4%), increased blood creatinine (20.1%). Serious TEAEs occurred in 4.9%.
				54.2%	52 weeks	

Abbreviations: TEAE = treatment-emerged adverse event; ADR = adverse drug reaction; SAE = serious adverse event; BID = twice daily; QD = once daily

(1): The results were derived from the Phase 2a clinical trials.

(2): The results were derived from the Phase 2b clinical trials.

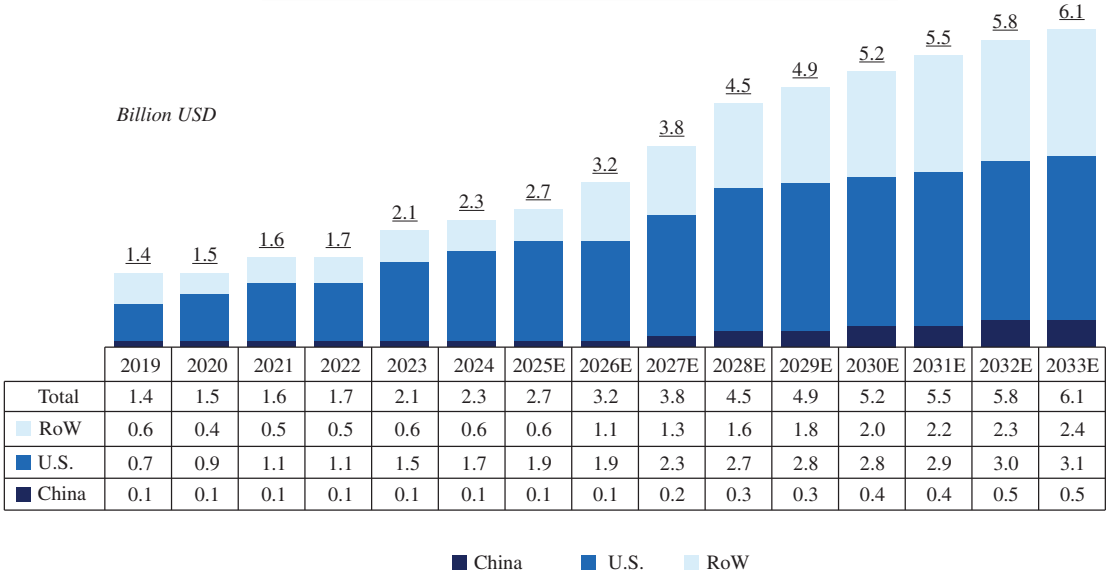
Source: Literature review, Frost & Sullivan analysis

Size and Competitive Landscape of Refractory Gout Drugs

The standard of care for refractory gout involves long-term ULT, such as XO inhibitors, URAT1 inhibitors and urate oxidase, with a treat-to-target goal of sUA < 6mg/dL, plus short-term anti-inflammatory prophylaxis during initiation. Such therapies are supplemented by lifestyle modifications, including weight control as well as reduced alcohol and purine intake, and comorbidity management. The global refractory gout drug market has demonstrated a steady upward trend. The market size grew from US$1.4 billion in 2019 to US$2.3 billion in 2024, representing a CAGR of 10.5% in the period. Looking ahead, the global market is projected to expand significantly, reaching US$6.1 billion by 2033, representing a CAGR of 11.2% from 2024 to 2033. The following table sets forth the historical and projected market size of refractory gout drugs in the U.S., China and globally from 2019 to 2033.

Global Refractory Gout Drug Market, 2019-2033E

Period	CAGR			
	China	U.S.	RoW	Global
2019-2024	-7.7%	19.0%	-0.1%	10.5%
2024-2033E	20.4%	7.3%	17.3%	11.2%



	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	1.4	1.5	1.6	1.7	2.1	2.3	2.7	3.2	3.8	4.5	4.9	5.2	5.5	5.8	6.1
RoW	0.6	0.4	0.5	0.5	0.6	0.6	0.6	1.1	1.3	1.6	1.8	2.0	2.2	2.3	2.4
U.S.	0.7	0.9	1.1	1.1	1.5	1.7	1.9	1.9	2.3	2.7	2.8	2.8	2.9	3.0	3.1
China	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.3	0.3	0.4	0.4	0.5	0.5

Pegloticase, a urate oxidase enzyme, is a treatment specifically designed for refractory gout. As of the Latest Practicable Date, Pegloticase had not been approved by the NMPA and thus had not been included in the national or provincial reimbursement drug lists in China. The following table sets forth the details of pegloticase.

Global Marketed Drugs for Refractory Gout

Drug Type	Ingredient	Originator Drug	Modality	Target	Treatment Line	Company	Year of Approval	Targeted Jurisdictions	Structure IP status	Price per Unit (USD)	Sales in 2024 (USD)
Urate oxidase	Pegloticase (普瑞氨希)	KRYSTEXXA	Biologic	Uric acid	2L	Horizon Pharma	2010 (FDA) 2013 (EMA, withdraw)	US	Expired	30,038 (8mg)	1.2 billion

Source: FDA, NMPA, Frost & Sullivan analysis

Pegloticase demonstrates good efficacy in lowering sUA when co-administered with methotrexate, as 71% of patients achieved sUA < 6.0 mg/dL at 6 months and 60% achieved such level by 12 months. Without methotrexate, only 38% reached this target at 6 months. However, treatment carries notable risks, including common AEs such as gout flares and arthralgia, and a Black Box Warning for anaphylaxis and infusion reactions. The clinical performance of pegloticase is set forth as below.

Clinical Performance of Marketed Refractory Gout Drugs

Ingredient	Originator Drug	Company	administration	Dosage	Efficacy		Safety
					% of subjects achieving sUA <6.0 mg/dL	Trial Duration	Adverse Events
Pegloticase (普瑞凱希)	KRYSTEXXA	Horizon Pharma	Mono	IV 8mg every 2 weeks (for whom methotrexate is contraindicated or not clinically appropriate)	47%	3 months	gout flares (77%), infusion reactions (26%), nausea (12%), contusion or ecchymosis (11%), nasopharyngitis (7%), constipation (6%), chest pain (6%), anaphylaxis (5%) and vomiting (5%)
					38%	6 months	
			Co-administered with methotrexate	IV 8 mg every 2 weeks, methotrexate 15 mg orally weekly	71%	6 months	gout flares (67%), arthralgia (14%), COVID-19 (9%), nausea (5%) and fatigue (5%)
					60%	12 months	

Black box warning:

- Anaphylaxis and infusion reactions have been reported to occur during and after administration of KRYSTEXXA
- Anaphylaxis may occur with any infusion, including a first infusion, and generally manifest within 2 hours of the infusion. However, delayed hypersensitivity reactions have also been reported
- KRYSTEXXA should be administered in healthcare settings and by healthcare providers prepared to manage anaphylaxis and infusion reactions
- Pre-medicate with antihistamines and Glucocorticoids and closely monitor for anaphylaxis for an appropriate period of time after administration of KRYSTEXXA
- Monitor serum uric acid levels prior to each infusion and discontinue treatment if levels increase to above 6 mg/dL, particularly when 2 consecutive levels above 6 mg/dL are observed
- Screen patients at risk for G6PD deficiency prior to starting KRYSTEXXA. Hemolysis and methemoglobinemia have been reported with KRYSTEXXA in patients with G6PD deficiency. KRYSTEXXA is contraindicated inpatients with G6PD deficiency

Abbreviations: IV = intravenous infusion

Source: Drug label, Literature review, Frost & Sullivan analysis

Pegloticase is currently the only approved therapy specifically targeting refractory gout, leaving patients with very limited treatment options. As of the Latest Practicable Date, there were no approved biosimilars for pegloticase. However, its high cost and notable safety concerns significantly restrict widespread use. As a result, there is an urgent and unmet need for innovative therapies in the treatment of refractory gout.

In addition, three drug candidates were advancing in clinical development for refractory gout, as of the Latest Practicable Date. SEL-212, consisting of sequential infusions of immune-tolerizing nanoparticles containing sirolimus followed by a pegylated uricase, had reached the Biologics License Application stage in the U.S. SAP-001 had completed Phase 2 clinical development globally and Phase 1 clinical development in China, while F012 was undergoing Phase 1b clinical trial. The following table sets forth the details of these drug candidates for refractory gout.

Refractory Gout Drugs under Development

Drug Name	Company	Modality	Target	Treatment Line	Phase of Trial (Global)	Regions of Global Trial	Phase of Trial (China)	First Post Date
SEL-212	Selecta/ 3S bio (三生製藥)	biologic combination therapy	UA	2L	BLA	US	/	2024/07
SAP-001	Shanton (珊頓醫藥)	small molecules	URAT1	2L	Phase 2 complete	US	Phase 1 complete	2019/8/1
F012	Lunan (魯南製藥)	Biologic	UA	2L	/	/	Phase 1b	2025-12

Abbreviations: BLA = biologics license application

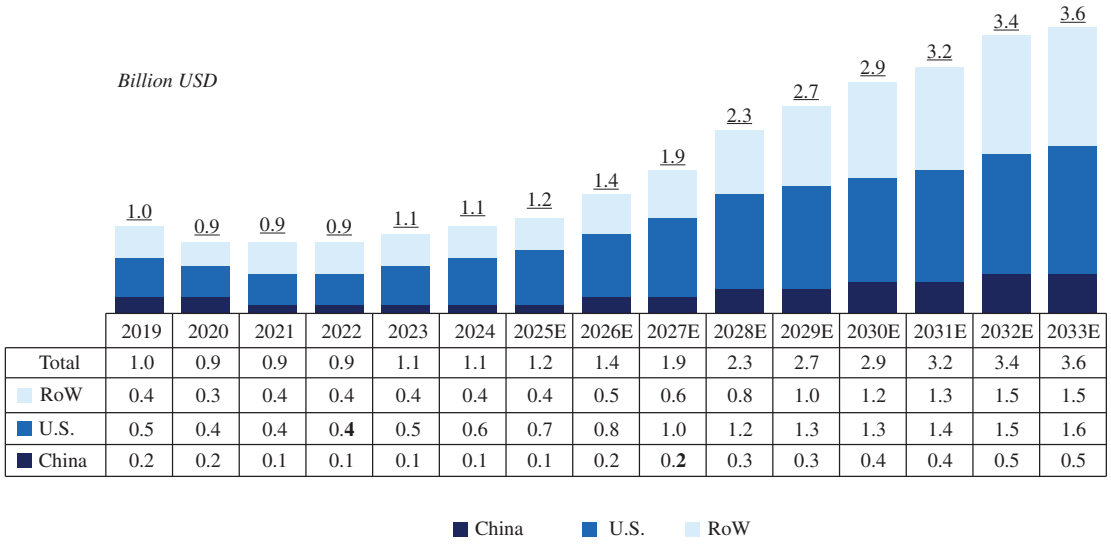
Source: Clinicaltrials, NMPA, Frost & Sullivan analysis

Size and Competitive Landscape of Tophaceous Gout Drugs

Tophaceous gout represents an advanced, chronic stage of gout characterized by persistent hyperuricemia and the deposition of monosodium urate crystals in soft tissues and joints, leading to the formation of clinically visible tophi. Patients with tophaceous gout typically experience long-standing disease, frequent flares, progressive joint damage, and functional impairment. The global tophaceous gout drug market slightly increased from US$1.0 billion in 2019 to US$1.1 billion in 2024, with a CAGR of 2.7% in the period. It is projected to reach US$3.6 billion by 2033, reflecting a CAGR of 14.0% from 2024 to 2033. The following table sets forth the historical and projected market size of tophaceous gout drugs in the U.S., China and globally from 2019 to 2033.

Global Tophaceous Gout Drug Market, 2019-2033E

Period	CAGR			
	China	U.S.	RoW	Global
2019-2024	-7.6%	5.4%	2.9%	2.7%
2024-2033E	19.5%	11.1%	15.9%	14.0%



	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	1.0	0.9	0.9	0.9	1.1	1.1	1.2	1.4	1.9	2.3	2.7	2.9	3.2	3.4	3.6
RoW	0.4	0.3	0.4	0.4	0.4	0.4	0.4	0.5	0.6	0.8	1.0	1.2	1.3	1.5	1.5
U.S.	0.5	0.4	0.4	0.4	0.5	0.6	0.7	0.8	1.0	1.2	1.3	1.3	1.4	1.5	1.6
China	0.2	0.2	0.1	0.1	0.1	0.1	0.1	0.2	0.2	0.3	0.3	0.4	0.4	0.5	0.5

Source: Frost & Sullivan Analysis

The competitive landscape for tophaceous gout drugs is currently limited, with only a small number of late-stage product candidates in development targeting this severe form of gout. The most advanced programs include Dotinurad, a URAT1 inhibitor being developed by Crystalyx as a first-line therapy and currently in Phase 3 clinical trials in the U.S., and Pozdeutirnad (AR882), a URAT1 inhibitor being developed by Arthrosi as a first-line therapy and currently in Phase 2 clinical trials in the U.S., New Zealand and Taiwan. The following table sets forth the details of these drug candidates for tophaceous gout.

Drug Name	Company	Modality	Target	Treatment Line	Phase of Trial (Global)	Regions of Global Trial	Phase of Trial (China)	First Post Date
Dotinurad	Crystalys	small molecules	URAT1	1L	Phase 3	US	/	2025-07
Pozdeutinurad/ AR882	Arthrosi	small molecules	URAT1	1L	Phase 2	US, New Zealand, Taiwan	/	2022-02

Source: Clinicaltrials, NMPA, Frost & Sullivan analysis

Growth Drivers and Future Trends of Gout Drug Market

The primary growth drivers and future trends of gout drug market include:

- *Improvement of drug safety.* The global and Chinese gout drug markets have been dominated by classic medications, such as allopurinol, febuxostat, and benzbromarone, including their originator and generic versions. Certain of these medications have been associated with known safety concerns related to their active ingredients: allopurinol may cause severe hypersensitivity syndrome; febuxostat has been linked to

cardiovascular safety controversies; and benzbromarone carries a risk of liver toxicity. These safety concerns not only restrict their clinical use in certain patients but also reduce patient compliance, thereby limiting the overall market potential.

- *Gout onset with younger patients.* Gout has been increasingly affecting younger populations, with nearly 60% of hyperuricemia and gout patients lately between the ages of 18 and 35 generally in all relevant jurisdictions. Younger patients often exhibit lower adherence to medications and tend to prefer treatments with fewer side effects and well-established efficacy. As a result, the development and commercialization of innovative drugs tailored to the needs of younger patients will become a key focus in the future.

- *Policy support and changes in market environment.* The policy environment plays a critical role in shaping the development of the gout drug market. Adjustments to health insurance policies and the implementation of centralized procurement policies have significantly influenced the market share of traditional drugs. Going forward, as health insurance policies are further optimized, innovative drugs are expected to receive broader market support and greater adoption.

- *Application of AI and big data.* With the advancement of technology, AI, big data, and other technologies have been increasingly applied in the pharmaceutical field, providing new directions for gout drug development and treatment. For example, AI-based drug discovery and development is expected to reduce the cost and time of new drug development, thereby driving changes in the gout drug market.

OVERVIEW OF ACUTE GOUT FLARE DRUG MARKET

Introduction to Acute Gout Flares

Acute gout flare is a sudden and intense episode of joint inflammation caused by the deposition of monosodium urate crystals in the joints. These flares are marked by severe pain, redness, warmth, and swelling that can last for days to weeks, often followed by asymptomatic periods known as remissions. Clinically, an acute gout flare represents a distinct and recognizable stage within the gout disease spectrum, characterized by a rapid inflammatory response driven by innate immune responses, whereas other stages of gout, namely asymptomatic hyperuricemia, intercritical gout and chronic tophaceous gout, reflect persistent urate accumulation without acute inflammatory manifestations. In short, acute gout flares are a typical and frequent manifestation within the progression of gout and hyperuricemia. For instance, in the U.S., gout patients suffer from an average of 6.6 acute attacks per person per year. Without treatment, gout flares can become more frequent and prolonged over time, and persistently high UA levels may lead to the formation of painful lumps called tophi, which can cause joint damage.

Central to the inflammatory mechanism of acute gout flares is the NOD-, LRR- and Pyrin Domain-Containing Protein 3 ("**NLRP3**") inflammasome, which triggers multiple pro-inflammatory cytokines, such as interleukin-1β ("**IL-1β**") release from macrophages upon monosodium urate crystal recognition. This IL-1β not only recruits neutrophils but also synergizes with monosodium urate crystals to amplify neutrophil extracellular trap formation, a hallmark of gouty inflammation. Other immune cells, including T cells, mast cells, and natural killer cells, also contribute to the inflammatory process through dysregulated immune responses, IL-1β release, and cytotoxic activity.

Accordingly, acute gout flares are pathophysiologically and clinically distinguishable from the chronic or intercritical stages of gout. While all stages share the same underlying metabolic abnormality of elevated UA, an acute gout flare is defined by its sudden onset, intense joint inflammation and activation of IL-1β-mediated pathways, whereas chronic or intercritical gout represents persistent urate crystal deposition and long-term metabolic disturbance without acute inflammatory symptoms.

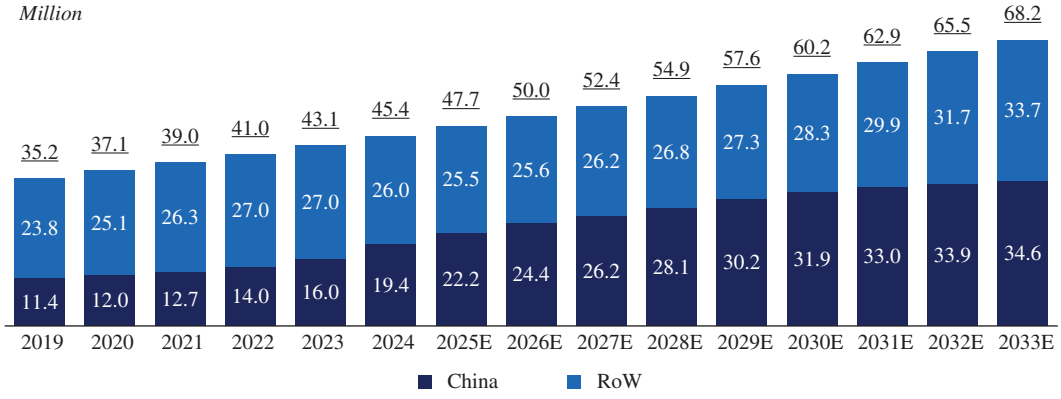
Prevalence of Acute Gout Flares

The global prevalence of acute gout flares has been rising steadily, with the number of patients of acute gout flares per year increasing from 35.2 million in 2019 to 45.4 million in 2024, representing a CAGR of 5.3%. Such upward trend is expected to persist, with patient number projected to reach 68.2 million by 2033, reflecting a CAGR of 4.6% from 2024 to 2033. In China, the patient number of acute gout flares has also been growing rapidly, increasing from 11.4 million

in 2019 to 19.4 million in 2024, with a higher CAGR of 11.3%. By 2033, the number of acute gout flare patients in China is expected to rise further to 34.6 million, reflecting a CAGR of 6.6% from 2024 to 2033. The following chart sets forth the historical and projected prevalence of acute gout flares both globally and in China.

Global and China Patient Population of Acute Gout Flares, 2019-2033E

Period	CAGR		
	China	RoW	Global
2019-2024	11.3%	1.8%	5.3%
2024-2033E	6.6%	2.9%	4.6%



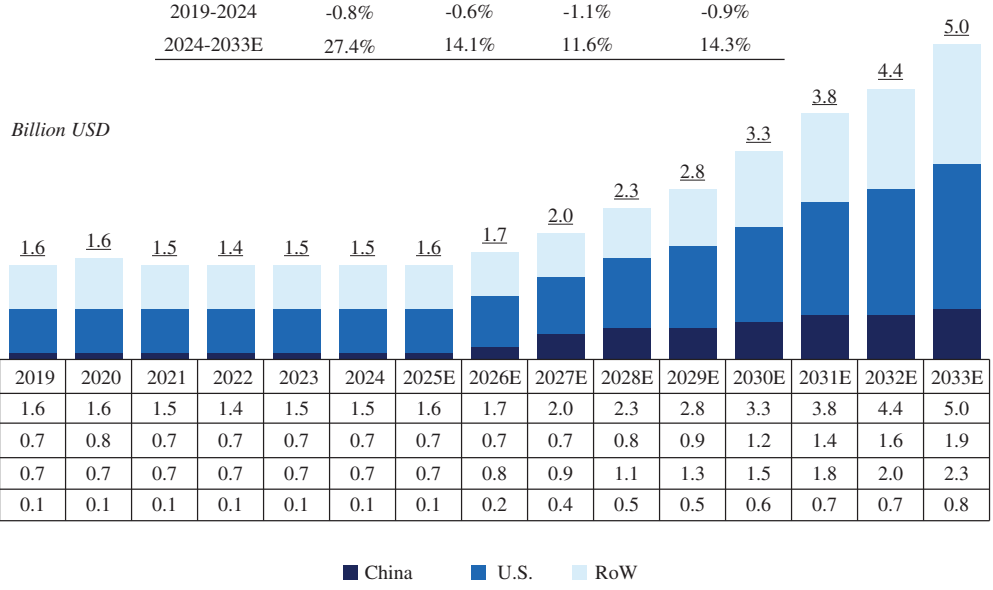
Source: Frost & Sullivan analysis

Acute Gout Flare Drug Market Size

From 2019 to 2024, the global acute gout flare drug market experienced a slight downturn, declining from US$1.6 billion to US$1.5 billion, primarily due to the decrease in sales volume as a result of increased prices. However, the market is projected to rebound significantly, reaching US$5.0 billion by 2033, with a CAGR of 14.3% from 2024 to 2033, driven by the introduction of novel therapies and rising disease awareness. In contrast, the acute gout flare drug market in China remained relatively flat from 2019 to 2024. Nevertheless, it is expected to undergo a rapid expansion, reaching US$0.8 billion by 2033, with a CAGR of 27.4% from 2024 to 2033, reflecting growing diagnosis rates, improved access to care, and increased adoption of innovative treatments. Likewise, the acute gout flare drug market remained stable at US$0.7 billion between 2019 and 2024 but is expected to reach US$2.3 billion in 2033, representing a CAGR of 14.1% from 2024 to 2033. The following chart sets forth the historical and projected market size of acute gout flare drugs in the U.S., China and globally from 2019 to 2033.

Global Acute Gout Flare Drug Market Size, 2019-2033E

Period	CAGR			
	China	U.S.	RoW	Global
2019-2024	-0.8%	-0.6%	-1.1%	-0.9%
2024-2033E	27.4%	14.1%	11.6%	14.3%



	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	1.6	1.6	1.5	1.4	1.5	1.5	1.6	1.7	2.0	2.3	2.8	3.3	3.8	4.4	5.0
RoW	0.7	0.8	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.9	1.2	1.4	1.6	1.9
U.S.	0.7	0.7	0.7	0.7	0.7	0.7	0.7	0.8	0.9	1.1	1.3	1.5	1.8	2.0	2.3
China	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.2	0.4	0.5	0.5	0.6	0.7	0.7	0.8

Source: Frost & Sullivan Analysis

Current Treatment Paradigms for Acute Gout Flares

The management of acute gout flares in China follows a structured approach based on disease severity and patient-specific factors. First-line medications for acute gout flares include colchicine (with a loading dose of 1 mg followed by 0.5 mg in one hour, then 0.5 mg QD or BID) and nonsteroidal anti-inflammatory drugs, or nonsteroidal anti-inflammatory drugs ("**NSAID**") (short-course, adequate dose). Cyclooxygenase-2 ("**COX-2**") inhibitors like etoricoxib (for patients with gastrointestinal bleeding risk) and celecoxib (for patients on low-dose aspirin) are also recommended. Second-line options involve (i) systemic glucocorticoids for patients who are intolerant to other treatments or have contraindications, and those experiencing gout flares involving multiple joints, large joints (e.g., knees or hips), or systemic symptoms, (ii) joint aspiration with intra-articular corticosteroid injection for patients with gout flare at one to two large joints, and (iii) combination therapies (e.g., colchicine plus NSAID) for high pain levels or polyarthritis or arthritis at two or more large joints. Refractory or intolerant cases may require polyethylene glycol urate oxidase, interleukin-1 (**"IL-1**") or tumor necrosis factor-alpha ("**TNF-α**") antagonists, or surgical intervention for complications like tophus-related nerve compression.

Regarding gout flare prevention, first line medications include low-dose colchicine (0.5 to 1 mg/day) for at least three to six months. For patients who are intolerant to colchicine, low-dose NSAID is recommended as second-line medication. In addition, for patients who are intolerant or contraindicated to colchicine and NDAIDs, such as those with chronic renal insufficiency, low-dose glucocorticoids (prednisone $\leq$ 10 mg/day) for at least three to six months.

In the U.S., the treatment paradigm for acute gout flares emphasizes a patient-centered approach, with the choice of therapy guided by individual clinical factors and preferences. First-line treatments include oral colchicine, NSAIDs, and glucocorticoids (administered orally, intra-articularly, or intramuscularly). Oral colchicine is typically initiated at 1.2 mg, followed by 0.6 mg one hour later, with continued low-dose therapy until symptom resolution. NSAIDs are appropriate for many patients, while glucocorticoids, including intra-articular or parenteral formulations, are recommended for those who cannot take oral medications. Topical ice is often used as an adjunct to pharmacologic therapy. Second-line options, such as IL-1 inhibitors or adrenocorticotropic hormone, are reserved for cases where first-line anti-inflammatory therapies are poorly tolerated or contraindicated. In addition, to prevent future flares, ULT is conditionally recommended to be initiated during the acute flare, rather than delaying until complete resolution, to improve long-term disease control.

Although both China and the U.S. employ a structured paradigm for the management of acute gout flares, relying primarily on colchicine, NSAIDs, and glucocorticoids, with biologic approaches reserved for refractory cases, significant limitations remain. Colchicine and NSAIDs, while effective, are constrained by gastrointestinal, renal, and cardiovascular safety concerns, often precluding their use in elderly or comorbid patients. Glucocorticoids are widely used but carry risks of metabolic and systemic side effects with repeated or prolonged courses. Biologic or enzyme based therapies, such as IL-1 inhibitors or urate oxidase, are available only in select cases, hindered by cost, access, and safety considerations. As a result, both countries face similar challenges: the current therapies for acute gout flares remain limited in tolerability, safety, and accessibility, underscoring the need for safer and more effective therapies to address diverse patient populations.

In addition, non-pharmacological alternative treatments and prevention methods, such as rest, cold compression, and lifestyle modifications to reduce flare triggers, are commonly recommended alongside drug therapy and form an integral part of the management landscape.

Competitive Landscape of Acute Gout Flare Drugs

Marketed Drugs for Acute Gout Flares

The treatment of acute gout flares involves several types of drugs, each with its own mechanism of action and associated toxicity. Colchicine, NSAIDs, glucocorticoids, and IL-1 inhibitors are typically used options that target inflammation and pain. The choice of treatment depends on patient-specific factors, including drug tolerance and the severity of the flare. The following table sets forth the details of the four types of marketed drugs for acute gout flares.

Marketed Drugs for Acute Gout Flares

Type	Mechanism of Action	Representative Drugs	Toxicity
Colchicine	Inhibition of neutrophil chemotaxis, adhesion and mobilization, and superoxide production in addition to inhibition of NALP3 inflammasomes and IL-1β processing and release	Mitigare, Colcrys	• Most common adverse reactions are abdominal pain, diarrhea, nausea, and vomiting, occurring in up to 80% of cases. • Bone marrow suppression: characterized by thrombocytopenia, decreased neutrophil counts, and even aplastic anemia, and may cause severe infectious disease. • A lethal dose of colchicine is considered to be 0.8 mg/kg. 7 to 26 mg can result in patient mortality.
NSAIDs	Exert an anti-inflammatory effect by inhibiting the activity of cyclooxygenase (COX)	Ibuprofen, Naproxen, Diclofenac, Celecoxib	• Risk of severe gastrointestinal adverse effects, including ulceration, bleeding, or perforation, more commonly in the elderly. • Other GI events may include nausea, dyspepsia, loss of appetite, abdominal pain, and diarrhea from erosion of the alimentary canal. • Serious cardiovascular adverse events and renal adverse effects may also associate with NSAIDs. • Typical signs and symptoms of NSAID overdose include nausea, vomiting, headache, drowsiness, blurred vision and dizziness. Symptoms are unlikely after ingestion of 100 mg/kg or less, and are usually not life-threatening unless more than 400 mg/kg is ingested.
Glucocorticoids	Prevent the formation of both PGs and LTs by causing the release of lipocortin, which by inhibition of phospholipase A2 reduces arachidonic acid release	Prednisone, Dexamethasone	• Musculoskeletal adverse effects: osteoporosis, steroid-induced myopathy, osteonecrosis due to long-term use. • Metabolic and endocrine: dose-dependent increase in fasting glucose levels, developing cushingoid features and weight gain. Impairment of growth in young children. • Other adverse effects may follow a threshold dose-response pattern with an elevated frequency of events beyond a specific threshold value, like weight gain and epistaxis at prednisone dose greater than 5 mg daily, or glaucoma, depression, hypertension at prednisone dose greater than 7.5 mg daily.
IL-1 inhibitors	Block inflammatory effects of IL-1β, preventing its interaction with IL-1RI and subsequent pro-inflammatory signaling	Anakinra, Rilonacept, Canakinumab, Genakumab	• IL-1 toxicity in humans is similar to the systemic inflammatory toxicities induced by IL-3, IL-6, and other cytokines with pleiotropic biologic activities, like fever, nausea, malaise, and hypotension. • Up to 40% patients have injection site reactions, up to 12% have upper respiratory tract infection. 7% have nausea, 5% diarrhea, 4% fatigue. • Increased risk of serious infections

Abbreviations: NALP3 = NACHT, LRR, and PYD domains-containing protein 3; GI = gastrointestinal; NSAID = non-steroidal anti-inflammatory drug; phospholipase A2 = phosphatide 2-acylhydrolase

Source: Literature review, Frost & Sullivan analysis

Generic versions of colchicine, NSAIDs, and glucocorticoids are widely available. While the market for acute gout flares features low-cost generic therapies, achieving commercial success is contingent on demonstrating clear clinical advantages. ABP-745's profile as a safer colchicine analog positions it as a strong candidate to differentiate itself and meet the high unmet need for better-tolerated treatments in this space.

Acute Gout Flare Drugs under Development

Drug candidates under development for acute gout flares are primarily focused on targeting IL-1 inhibitors and colchicine analogs, reflecting the growing interest in addressing the inflammatory pathways central to the disease. As of the Latest Practicable Date, there were six drug candidates in development for the treatment of acute gout flares. Among these candidates, our

ABP-745 stands out as the only colchicine analog currently in clinical trials. Administered orally, ABP-745 is being investigated in the U.S., China and Australia, offering a potentially safer option that avoids drug-drug interaction. These developments highlight ongoing efforts to expand and refine treatment approaches for acute gout flares. The following table sets forth the details of acute gout flare drugs under development that have conducted clinical trials in China, the U.S. or Australia as of the Latest Practicable Date.

Acute Gout Flare Drugs under Development

Drug Name	Modality	Target	Company	Mechanism	Treatment Lines	Administration method	Phase of Trial (US, Australia)	Phase of Trial (China)	First Post Date	Location
ABP-745	small molecule	Colchicine Analogue	Atom Therapeutics (新元素藥業)	Colchicine analogue	1L	Oral	Phase 2	Phase 2	2025/08/20	U.S.
Dapansutrile (OLT1177)	small molecule	NLRP3	Olatec Therapeutics	NLRP3 inhibitor	NA	Oral	Phase 2/3	/	2022/12/20	U.S.
SSGJ-613	antibody	IL1B	3S Bio (三生製藥)	Anti-IL-1 antibody	2L	s.c. injection	/	NDA	2025/6/6	China
UA007	biologic	IL1R1	General Regeneratives /Usynova (交晨生物 /祐森健恒)	IL-1R antagonist	2L	i.m. injection	/	Phase 2a	2022/7/14	China
Plonmarlimab	antibody	CSF2	I-MAB Biopharma (天境生物科技)	Anti-GM-CSF antibody	2L	Intravenous Injection	/	Phase 2	2026/2/26	China
IBI3011	biologics	IL1RAP	Innovent Biologics (信達生物)	IL-1RAP antagonist	2L	s.c. injection	/	Phase 1	2025/11/14	China

Abbreviations: s.c. = subcutaneous; i.m. = intramuscular

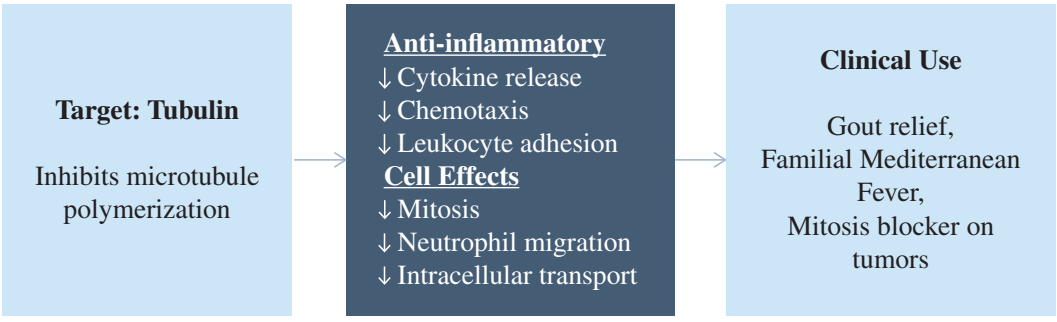
Note: Our competitive landscape includes only drugs registered in clinical trials or the CDE, excluding generics. We only include trials conducted in the United States and China.

Source: Literature review, Frost & Sullivan analysis

Overview of Colchicine

Colchicine, a naturally occurring compound derived from the autumn crocus, has been utilized in medicine for centuries, with its use dating back to ancient Egypt and Greece. The compound is widely recognized for its potent anti-inflammatory properties, particularly in the treatment of acute gout flares. Its mechanism of action involves the inhibition of microtubule polymerization, which suppresses neutrophil migration, phagocytosis, and the release of pro-inflammatory cytokines, such as IL-1β and TNF-α. The following diagram illustrates the mechanism of action of colchicine.

Mechanism of Action of Colchicine



Source: Literature review, Frost & Sullivan analysis

Colchicine is a cornerstone therapy for the management of acute gout flares. By targeting neutrophil activity, it effectively reduces inflammation and relieves the associated pain and swelling. Specifically, colchicine prevents neutrophils from migrating to inflamed joints and inhibits their ability to contribute to the inflammatory cascade by blocking microtubule formation. This action also reduces the release of cytokines, such as IL-1β and TNF-α, which play a pivotal role in the inflammatory processes of gout. Despite its efficacy, colchicine's clinical use is limited by its narrow therapeutic window, as overdoses can result in serious gastrointestinal, hematologic, and neurological toxicities.

Colchicine's toxicity is linked to specific parts of its molecular structure, such as the methoxy groups on Rings A and C, the C-7 amide, and the C-9 carbonyl group. These structural features can cause off-target interactions, including with intestinal proteins, leading to gastrointestinal and systemic side effects. To address this, researchers have modified colchicine's structure to reduce toxicity while maintaining its effectiveness. For example, adjustments like removing certain methoxy groups, modifying the C-7 amide, or altering the C-9/C-10 region of the molecule have been explored.

In addition to its established role in gout treatment, colchicine has shown promise in the management of cardiovascular diseases, particularly in stabilizing atherosclerotic plaques and reducing the recurrence of cardiovascular events. Its mechanisms of action in this context include the inhibition of the NLRP3 inflammasome, resulting in decreased IL-1β levels; the suppression of endothelial activation, lowering TNF-α and E-selectin levels; and the prevention of leukocyte adhesion and plaque destabilization.

Growth Drivers and Future Trends of Acute Gout Flare Drug Market

The primary growth drivers and future trends of acute gout flare drug market include:

- *Growing incidence of acute gout flares.* The overall prevalence of gout is rising, driven in part by an increasing number of younger patients being diagnosed. Simultaneously, metabolic risk factors are worsening, as obesity, high-purine diets, consumption of sugar-sweetened beverages, and alcohol intake all contribute to significant elevations in sUA levels. Gout is also frequently accompanied by CKD and cardiovascular disease, further amplifying its disease burden and public health impact.

- *Innovative drugs with new mechanisms.* The limitations of traditional gout treatments, such as colchicine (associated with up to 80% incidence of gastrointestinal side effects) and NSAIDs (linked to an increased cardiovascular risk), have driven the development and adoption of newer biologic therapies. Among these, IL-1 inhibitors are recommended for patients with acute gout flares who have contraindications to or inadequate responses to traditional medications like colchicine, NSAIDs, or glucocorticoids. Despite their promise, the clinical use of IL-1 inhibitors remains limited due to several challenges, such as their classification as biologic therapies, high costs, and the potential for some IL-1 inhibitors to increase the risk of infections.

- *Increased requirements for drug safety.* The younger population is increasingly prioritizing early intervention to prevent complications. Meanwhile, younger generations place greater emphasis on the safety and long-term management of their treatments, favoring newer medications that offer improved efficacy and fewer side effects. Innovations such as extended-release colchicine formulations and colchicine analogs with reduced toxicity are expected to further drive market expansion, meeting the evolving demands of this health-conscious group.

OVERVIEW OF CHRONIC KIDNEY DISEASE MARKET

CKD, also known as chronic kidney failure, is a progressive condition featuring the gradual loss of kidney function. The kidneys play a crucial role in maintaining the body's internal balance by filtering waste products and excess fluids from the blood, which are then excreted through urine. In the early stages of CKD, patients may exhibit no noticeable symptoms. However, as the disease progresses, impaired kidney function leads to the accumulation of electrolytes and waste in the blood, ultimately causing significant clinical symptoms. CKD is often preceded by acute kidney disease ("**AKD**"), a condition lasting up to three months. Patients whose AKD does not resolve after this period are classified as having CKD with a history of AKD.

While CKD and hyperuricemia are not exactly the same indication, they are clinically and pathophysiologically interrelated conditions with a strong and well-documented association supported by medical evidence. Hyperuricemia and gout are recognized as independent risk factors

for CKD and have a causal relationship with the formation of kidney stones and renal impairment, further contributing to the onset and progression of kidney dysfunction. Conversely, impaired renal function in CKD reduces UA excretion and commonly leads to secondary hyperuricemia. This bidirectional relationship between decreased renal clearance of UA and urate-induced renal injury has been supported by extensive clinical and scientific evidence.

Studies show that ULT may help protect kidney function and slow the progression of CKD in hyperuricemic patients with kidney damage. Accordingly, although CKD and hyperuricemia are not treated as a single indication, their coexistence represents a recognized and meaningful disease association. Therapeutic strategies aimed at lowering UA have been evaluated for potential renoprotective benefits in patients with CKD associated with hyperuricemia, and the development of urate-lowering agents in this population is supported by established medical rationale.

While there is currently no cure for CKD, a combination of lifestyle changes and medical treatments can help alleviate symptoms, slow disease progression, and reduce the risk of complications. Among these, medications are essential for controlling the underlying conditions, such as hypertension, diabetes and high cholesterol, that contribute to CKD, as well as for managing complications, including anemia, mineral imbalances and cardiovascular risks. Although no drug directly cures CKD, drugs remain central to disease management. In advanced stages, dialysis or kidney transplantation may be necessary to replace lost kidney function.

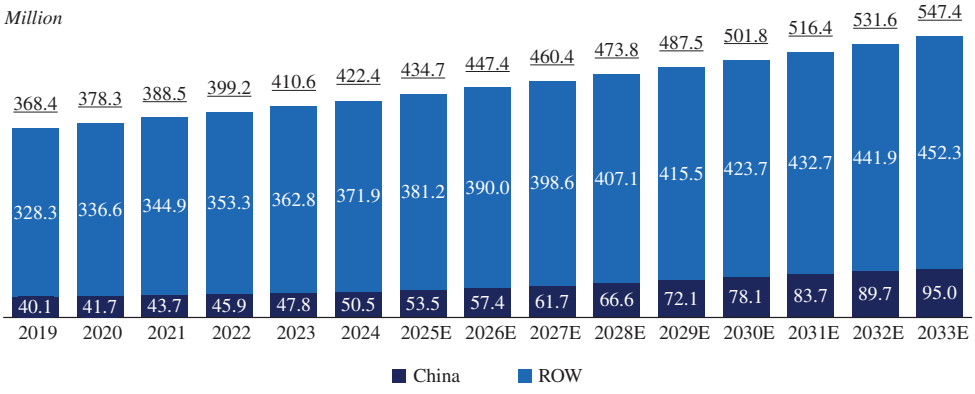
The kidneys play a key role in removing uric acid from the body, excreting about 70%, while the rest is eliminated through the bile and intestines. Hyperuricemia can occur due to either increased production of UA or decreased excretion, especially when kidney function is impaired. In fact, CKD is a common cause of secondary hyperuricemia. On the other hand, long-term high UA levels can cause urate crystals to build up in the kidneys, leading to further damage such as chronic urate nephropathy, acute UA nephropathy, and kidney stones. Hyperuricemia is also considered an independent risk factor for the development and progression of CKD. In China, it affects 36.6% to 50.0% of CKD patients, with higher rates in more advanced stages. This high comorbidity rate further supports the close clinical relationship between CKD and hyperuricemia.

Prevalence of CKD associated with Hyperuricemia

CKD associated with hyperuricemia is a growing global health concern, with the number of people affected worldwide increasing from 368.4 million in 2019 to 422.4 million in 2024, representing a CAGR of 2.8%. Such upward trend is expected to continue, with the global population of CKD associated with hyperuricemia projected to reach 547.4 million by 2033, with a CAGR of 2.9% from 2024 to 2033. In China, the prevalence of CKD associated with hyperuricemia rose from 40.1 million in 2019 to 50.5 million in 2024, with a CAGR of 4.7% from 2019 to 2024. The growth is anticipated to accelerate, with the population of CKD associated with hyperuricemia in China expected to reach 95.0 million by 2033, reflecting a CAGR of 7.3% from 2024 to 2033. The following chart sets forth the historical and projected prevalence of CKD associated with hyperuricemia both globally and in China.

Global and China Prevalence of CKD associated with Hyperuricemia, 2019-2033E

Period	CAGR		
	China	ROW	Global
2019-2024	4.7%	2.5%	2.8%
2024-2033E	7.3%	2.2%	2.9%



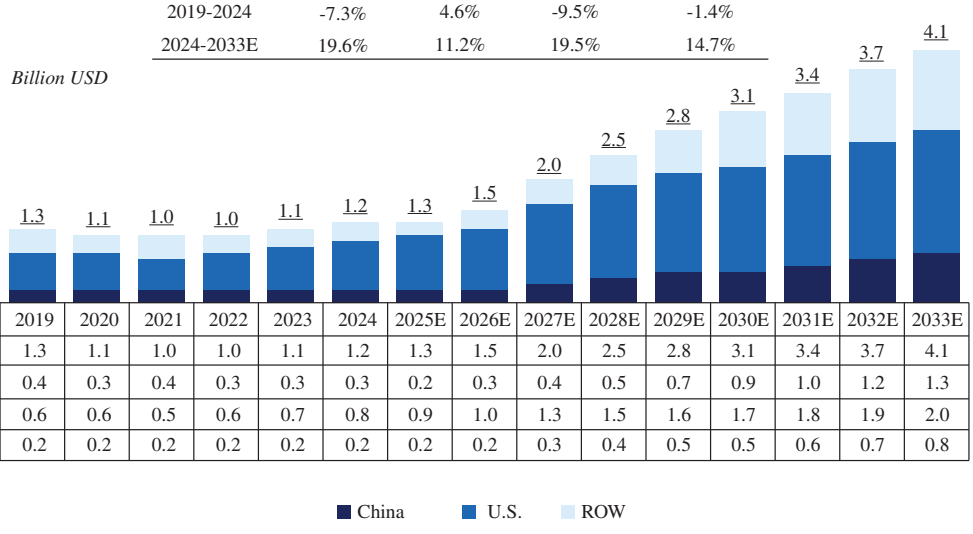
Source: Frost & Sullivan analysis

CKD associated with Hyperuricemia Drug Market Size

The global CKD associated with hyperuricemia drug market slightly decreased from USD 1.3 billion in 2019 to USD 1.2 billion in 2024, with a CAGR of -1.4%. It is projected to reach USD 4.1 billion by 2033, reflecting a CAGR of 14.7% from 2024 to 2033. The following chart sets forth the historical and projected market size of CKD associated with hyperuricemia drugs in the U.S., China and globally from 2019 to 2033.

Global CKD associated with Hyperuricemia Drug market, 2019-2033E

Period	CAGR			
	China	U.S.	ROW	Global
2019-2024	-7.3%	4.6%	-9.5%	-1.4%
2024-2033E	19.6%	11.2%	19.5%	14.7%



	2019	2020	2021	2022	2023	2024	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E
Total	1.3	1.1	1.0	1.0	1.1	1.2	1.3	1.5	2.0	2.5	2.8	3.1	3.4	3.7	4.1
ROW	0.4	0.3	0.4	0.3	0.3	0.3	0.2	0.3	0.4	0.5	0.7	0.9	1.0	1.2	1.3
U.S.	0.6	0.6	0.5	0.6	0.7	0.8	0.9	1.0	1.3	1.5	1.6	1.7	1.8	1.9	2.0
China	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.3	0.4	0.5	0.5	0.6	0.7	0.8

Note:

(1) Only UA lowering drugs are considered.

Source: Frost & Sullivan analysis

Current Treatment Paradigms for CKD associated with Hyperuricemia

The management of hyperuricemia is a recognized component of comprehensive care for patients with CKD. Current treatment strategies primarily involve the use of XO inhibitors, such as allopurinol and febuxostat, which are established as first-line ULT. In addition, several URAT1 inhibitors, including probenecid, benzbromarone, lesinurad and dotinurad, are approved and available for clinical use as alternative or adjunctive options. Benzbromarone and probenecid are generally used as alternative, rather than adjunctive first-line, urate-lowering treatment options in China and the U.S..

The treatment environment includes multiple generic versions of key medications, which have contributed to the establishment of accessible treatment pathways. In parallel, non-pharmacological approaches are integral to management guidelines, emphasizing the importance of lifestyle interventions such as dietary modifications, regular physical activity, weight management, smoking cessation, limited alcohol and purine intake, adequate hydration and urine alkalization.

Notwithstanding existing treatments, a significant unmet medical need remains. Current therapeutic options are associated with certain limitations, including safety considerations and specific usage constraints in patients with advanced renal impairment. This clear need for innovative treatments underpins the development of novel therapies such as ABP-671, which aims to offer a favorable safety and efficacy profile for this patient population.

Competitive Landscape of Drugs for CKD associated with Hyperuricemia

As of the Latest Practicable Date, marketed and investigational drugs for CKD associated with hyperuricemia include long-established XO inhibitors, such as allopurinol and febuxostat, which are used as monotherapies and have shown either no difference or only modest benefit in slowing eGFR decline while being associated with important safety concerns, including serious hypersensitivity reactions and liver-function abnormalities. URAT1 inhibitors, namely probenecid and benzbromarone, have also been applied to the treatment of CKD associated with hyperuricemia. These URAT1 inhibitors have been marketed for decades but require careful monitoring due to risks, such as renal complications and hepatotoxicity.

In addition, we intend to develop our Core Product, ABP-671, as part of combination therapy with an XO inhibitor in patients of CKD associated with hyperuricemia. Based on the clinical trial results from the CKD cohorts in Phase 1 trial of ABP-671 in healthy subjects, ABP-671 has shown a generally favorable tolerability profile. The following table sets forth the marketed drugs and investigational drugs for CKD associated with hyperuricemia as of the Latest Practicable Date.

Drug Name	Company	Target	Progress	Mono or Combo therapy	Dosage	Efficacy	Safety
Allopurinol (別嘌醇)	Casper Pharma /GSK	XO	Marketed (1966)	Mono	300mg	No difference of eGFR decline rate compared to placebo	serious allopurinol hypersensitivity syndrome may occur; gastrointestinal symptoms, rash, hepatic impairment, bone marrow suppression, etc.
Febuxostat (非布司他)	Takeda	XO	Marketed (2008)	Mono	20/40/80mg	In comparison with the control group, the patients who received febuxostat showed a reduced risk of kidney events and a slower decline in eGFR (WMD = 0.90 mL/min/1.73 m^2)	The main symptoms include abnormal liver function, nausea, joint pain, and rash. Several studies have shown that the incidence of adverse reactions to febuxostat is lower than that of allopurinol.
Probenecid (丙磺舒)	Merck	URAT1	Marketed (1951)	Mono	500/1000mg	N/A	Before and during treatment, special attention should be paid to drinking plenty of fluids and the use of drugs that alkalinize the urine. These drugs should be contraindicated if the 24-h urine acid excretion has increased or if urinary uric acid stones are present, and should be used with caution in patients with ulcer disease or renal insufficiency.
Benzbromarone (苯溴馬隆)	labaz/Sanofi	URAT1	Marketed (2008)	Mono	50-100mg	Slower decline in eGFR compared with Allopurinol (-2.19 vs -4.89)	
Lingdolinurad (ABP-671)	Atom Therapeutics (新元素藥業)	URAT1	Phase 2	Combo	0.5-3mg/day	N/A	ABP-671 appeared generally safe and well tolerated in participants with normal kidney function and CKD. There were no deaths, serious, or severe TEAEs reported, and no participants were discontinued from the study due to TEAEs.

Note: Clinical result of ABP-671 is based on the CKD cohorts in Phase 1 trial of ABP-671 in healthy subjects and ABP-671 is now not yet recruiting for CKD phase 2 trial.

Abbreviations: uACR = urinary albumin to creatinine ratio; eGFR = estimated glomerular filtration rate; WMD = weighted mean difference

Source: Clinicaltrials, NMPA, FDA, Frost & Sullivan analysis

OVERVIEW OF ATHEROSCLEROSIS DRUG MARKET

Introduction to Atherosclerotic Cardiovascular Disease

Atherosclerosis is a chronic inflammatory condition in which plaques build up in the arteries due to the retention of low-density lipoproteins and remnant lipoproteins. This process, worsened by inflammation in areas of disturbed blood flow, reduces vascular elasticity, impairs blood flow, and leads to chronic hypoxia in vital organs like the heart and brain. Atherosclerotic cardiovascular disease ("**ASCVD**"), a severe form of cardiovascular disease ("**CVD**") caused by atherosclerosis, includes life-threatening conditions, such as heart attacks, strokes, and peripheral arterial disease. The number of ASCVD patients globally increased from 523.1 million in 2019 to 615.2 million in 2024, with a CAGR of 3.3%, and is projected to reach 796.4 million in 2033, reflecting a CAGR of 2.9% from 2024 to 2033. In China, ASCVD patient number rose from 78.3 million in 2019 to 90.0 million in 2024 at a CAGR of 2.8%, and is expected to further expand to 118.0 million by 2033, with a CAGR of 3.1% from 2024 to 2033.

Risk factors for atherosclerosis include metabolic disorders like hypercholesterolemia, hypertension, and diabetes, as well as lifestyle factors such as smoking, inactivity, obesity, and high-fat diets. Genetic predispositions, including male sex and family history, also increase risk. Although often asymptomatic in its early stages, atherosclerosis progresses with inflammation and plaque buildup, eventually narrowing arteries, reducing blood flow, and causing serious complications like tissue damage, heart attacks, and strokes. The symptoms of atherosclerosis vary based on the location and severity of arterial blockages and can lead to serious conditions, such as coronary artery disease ("**CAD**"), ischemic stroke, and peripheral artery disease ("**PAD**").

CAD

CAD, the most common form of ASCVD, occurs when plaque narrows or blocks coronary arteries, causing myocardial ischemia, angina, or heart attacks, and is a significant cause of illness and death, especially in suburban and rural areas. CAD is a leading global health concern, with its prevalence rising significantly in recent years. From 197.2 million cases in 2019, the number increased to 222.5 million in 2024, representing a CAGR of 2.4%. The prevalence is projected to reach 255.1 million by 2033, growing at a slightly slower CAGR of 1.5% from 2024. In China, the prevalence of CAD in rose from 24.6 million in 2019 to 28.0 million in 2024, with a CAGR of 2.6%. It is expected to continue increasing to 34.0 million by 2033, reflecting a CAGR of 2.2% from 2024.

Stroke

Strokes, particularly ischemic strokes (87% of all strokes), result from blocked blood flow to the brain due to plaque buildup, with symptoms including sudden numbness, confusion, difficulty speaking, and severe headaches. Hemorrhagic strokes, while less common, are often fatal and caused by ruptured blood vessels in or around the brain. The number of stroke cases globally grew from 11.4 million in 2019 to 12.8 million in 2024, with a CAGR of 2.4%, and is expected to continue to grow to 16.0 million in 2033, with a CAGR of 2.5% from 2024 to 2033. Meanwhile, stroke is a major public health concern in China, with its incidence steadily rising over the years. In 2019, there were 4.8 million new stroke cases, which increased to 5.6 million in 2024, reflecting a CAGR of 3.1%. Such upward trend is expected to accelerate, reaching 7.8 million by 2033, with a CAGR of 3.8% from 2024.

PAD

PAD occurs when plaque restricts blood flow to areas like the lower limbs, leading to leg pain, ulcers, and, in severe cases, gangrene, which may require limb amputation if untreated. PAD is a growing global health issue, with its prevalence rising steadily over the years. Globally, the number of PAD cases increased from 271.6 million in 2019 to 322.3 million in 2024, with a CAGR of 3.5%. Such trend is expected to continue, reaching 378.1 million by 2033, though at a slower CAGR of 1.8% from 2024. In China, the prevalence of PAD is also on the rise, growing from 49.8 million in 2019 to 55.4 million in 2024, with a CAGR of 2.2%. By 2033, the number of PAD patients in China is projected to reach 66.4 million, reflecting a CAGR of 2.0% from 2024.

Current Treatment Paradigms for Atherosclerosis and ASCVD

The treatment of atherosclerosis involves both invasive and noninvasive approaches, tailored to the severity of the condition. Invasive treatments, such as angioplasty, stenting, coronary artery bypass grafting, carotid endarterectomy, and thrombectomy, are typically reserved for advanced or severe cases, particularly in CAD. Noninvasive treatments focus on addressing atherosclerosis and its risk factors through lifestyle modifications, including a healthy diet, regular exercise, smoking cessation, and weight management. In addition, medications like statins, antiplatelet drugs, and antihypertensives play a key role in managing cardiovascular risk factors and slowing disease progression, making them essential components of atherosclerosis prevention and control.

Colchicine has emerged as a potential therapeutic option in the management of ASCVD. In June 2023, the FDA approved Lodoco (colchicine 0.5 mg) for a new cardiovascular indication to reduce the risk of myocardial infarction, stroke, coronary revascularization, and cardiovascular death in patients with ASCVD or adults with multiple cardiovascular risk factors. The recommended dosage is 0.5 mg taken orally once daily, with or without food. The 2023 American Heart Association Guideline for the Management of Patients with Chronic Coronary Disease includes colchicine as a potential treatment to reduce recurrent ASCVD events.

Some clinical evidence has demonstrated that colchicine may have promising benefits for ASCVD event reduction, though the clinical data obtained thus far is mixed. The COLCOT trial showed that low-dose colchicine initiated post-myocardial infarction significantly reduced ischemic cardiovascular events with a favorable safety profile. Similarly, the LoDoCo trial reported a 31% reduction in acute cardiovascular events when colchicine was added to standard therapy in stable CAD patients. Supporting these findings, the coronary CTA (computed tomography angiography) study indicated a significant reduction in coronary plaque volume with colchicine.

Competitive Landscape of ASCVD Drug Market

The ASCVD drug market is characterized by a diversified therapeutic approach targeting distinct pathological pathways, with five major drug classes defining the current competitive framework. Lipid-lowering agents, led by statins and novel RNAi therapy inclisiran, dominate first-line interventions by reducing atherogenic lipoproteins, though their inability to address plaque inflammation presents a key limitation. Antihyperglycemics, such as sodium-glucose cotransporter-2 inhibitors and glucagon-like peptide-1 receptor agonists, have emerged as multifactorial modifiers of metabolic dysfunction, while antihypertensives and antiplatelets provide essential hemodynamic and antithrombotic support. Anti-inflammatory therapies like low-dose colchicine, which suppress vascular inflammation to stabilize and regress plaques, also represent recent innovations in the field. However, colchicine is currently the only anti-inflammatory drug approved worldwide for the treatment or prevention of ASCVD, highlighting the scarcity of anti-inflammatory therapies for this indication. The following table sets forth the clinical performance of representative ASCVD drugs.

Clinical Performance of Representative ASCVD Drugs

Drug name	Drug class & dosing frequency	Mechanism of action	Key clinical-trial findings	Drawbacks
Atorvastatin	Statin (QD oral)	HMG-CoA-reductase inhibitor, lowers LDL-C	CARDS: 37 % reduction in major adverse cardiovascular events (MACE); clear clinical benefit	Myalgia, elevated liver enzymes; some patients fail to reach lipid-lowering targets
Rosuvastatin	Statin (QD oral)	HMG-CoA-reductase inhibitor, lowers LDL-C	JUPITER: significant reduction in CV events in patients with elevated hs-CRP	Same as above; high-dose therapy is relatively costly
Ezetimibe	Cholesterol-absorption inhibitor (QD oral)	NPC1L1 inhibitor, reduces intestinal cholesterol absorption	IMPROVE-IT: combined with a statin, further lowered CV events by 6.4 %	Limited LDL-C reduction as monotherapy; gastrointestinal discomfort
Evolocumab	PCSK9 mAb (140 mg SC every 2 wks or 420 mg SC monthly)	Inhibits PCSK9, increases LDL receptors, profound LDL-C lowering	FOURIER-OLE: up to 8.4 yr follow-up, 15–20 % MACE reduction	High cost; injection-site reactions
Alirocumab	PCSK9 mAb (SC every 2 wks)	Inhibits PCSK9, profound LDL-C lowering	ODYSSEY OUTCOMES: median 2.8 yr follow-up, 15 % MACE reduction	High cost; injection-site reactions
Colchicine	Microtubule-polymerization inhibitor (QD oral)	Inhibits NLRP3 inflammasome, broad anti-inflammatory	LoDoCo2: additional 31 % reduction in CV event risk	GI intolerance, hepatic/renal toxicity; requires selecting well-tolerated patients
Canakinumab	IL-1β mAb (not yet approved)	Neutralizes IL-1β, blocks downstream inflammation	CANTOS: additional 15 % CV-event risk reduction	Extremely high cost; infection risk; benefit limited to high-inflammation subgroup
Tocilizumab	IL-6 mAb (not yet approved)	Blocks IL-6 receptor, lowers CRP & inflammatory mediators	Early small trials: plaque-inflammation markers decreased	Potent immunosuppression; serious-infection black-box warning; no large-scale clinical outcome data

Abbreviations: LDL-C = low-density lipoprotein cholesterol; CV = cardiovascular; hs-CRP = high-sensitivity C-reactive protein; NPC1L1 = Niemann-Pick C1-like 1; PCSK9 = proprotein convertase subtilisin/kexin Type 9; NLRP3 = NOD-, LRR- and Pyrin Domain-Containing Protein 3; GI = gastrointestinal; mAb = monoclonal antibody; CRP = C-reactive protein

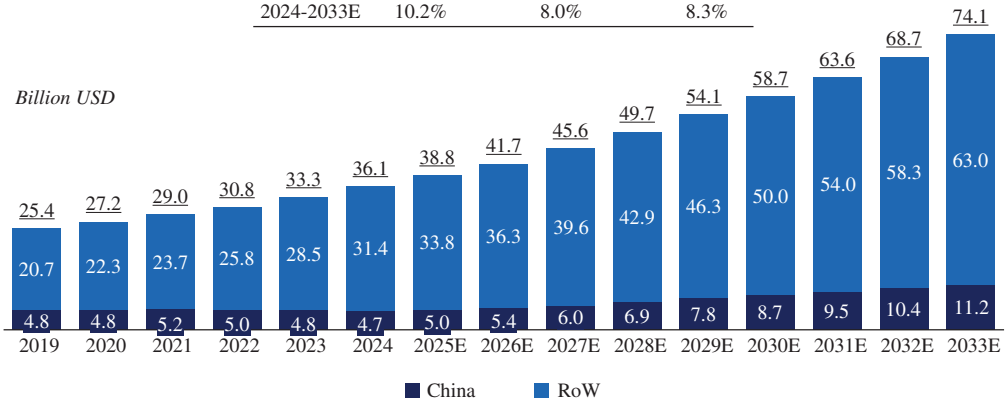
Source: Literature review, Frost & Sullivan analysis

ASCVD Drug Market Size

The global ASCVD drug market expanded from US$25.4 billion in 2019 to US$36.1 billion in 2024, representing a CAGR of 7.2%, and is projected to reach US$74.1 billion by 2033, representing a CAGR of 8.3% from 2024 to 2033. In China, the market declined modestly from US$4.8 billion to US$4.7 billion from 2019 to 2024, primarily attributable to volume-based procurement, but is expected to rebound to US$11.2 billion by 2033, corresponding to a CAGR of 10.2% from 2024 to 2033. The following chart sets forth the historical and projected expansion of the ASCVD drug market both globally and in China.

Global and China ASCVD Drug Market Size, 2019-2033E

Period	CAGR		
	China	RoW	Global
2019-2024	-0.5%	8.7%	7.2%
2024-2033E	10.2%	8.0%	8.3%



Source: Frost & Sullivan analysis

Growth Drivers and Future Trends of ASCVD Drug Market

The primary growth drivers and future trends of ASCVD drug market include:

- ***More effective treatment options towards plaque.*** Current drug therapy focuses on controlling risk factors for atherosclerosis (e.g., high cholesterol, high blood pressure, high blood sugar, etc.) and stabilizing plaques, but it is difficult to completely remove or significantly shrink formed plaques. For example, although statins can lower low-density lipoprotein cholesterol levels, thereby slowing plaque progression, they have a limited effect on shrinking formed plaques.

- ***Innovative drugs changing the therapeutic landscape.*** While traditional treatments have focused on cholesterol levels, future research directions may focus more on the role of inflammation in atherosclerosis. For example, drugs that target inflammatory factors such as IL-1β and IL-6, such as canakinumab, have been shown to have potential in reducing the risk of cardiovascular events.

- ***Multi-target combination therapy.*** In the future, the treatment of atherosclerosis will focus more on multi-targeted joint intervention. For example, simultaneous interventions targeting multiple key aspects of the inflammatory response, lipid metabolism, and protection of endothelial function are expected to more comprehensively block the progression of atherosclerosis.

REPORT COMMISSIONED BY FROST AND SULLIVAN

In connection with the [**REDACTED**], we have engaged Frost & Sullivan to conduct a detailed analysis and prepare an industry report on the major markets for which our drug candidates are positioned. Frost & Sullivan is an independent global market research and consulting company which was founded in 1961 and is based in the U.S. We have agreed to pay Frost & Sullivan a total fee of approximately RMB0.7 million for the preparation of the Frost & Sullivan Report, and we believe that such fees are consistent with the market rate. The payment of such amount is not contingent upon our successful [**REDACTED**] or on the results of the Frost & Sullivan Report. Except for the Frost & Sullivan Report, we did not commission any other industry report in connection with the [**REDACTED**].

The market projections in the Frost &Sullivan Report were based on the following key assumptions: (i) the overall social, economic and political environment globally and in China is expected to remain stable during the forecast period; (ii) the economic and industrial development globally and in China is likely to maintain a steady growth trend over the next decade; (iii) related key industry drivers are likely to continue driving the growth of the market during the forecast period; and (iv) there is no extreme force majeure or industry regulation in which the market may be affected dramatically or fundamentally. The reliability of the Frost & Sullivan Report may be affected by the accuracy of the foregoing key assumptions.