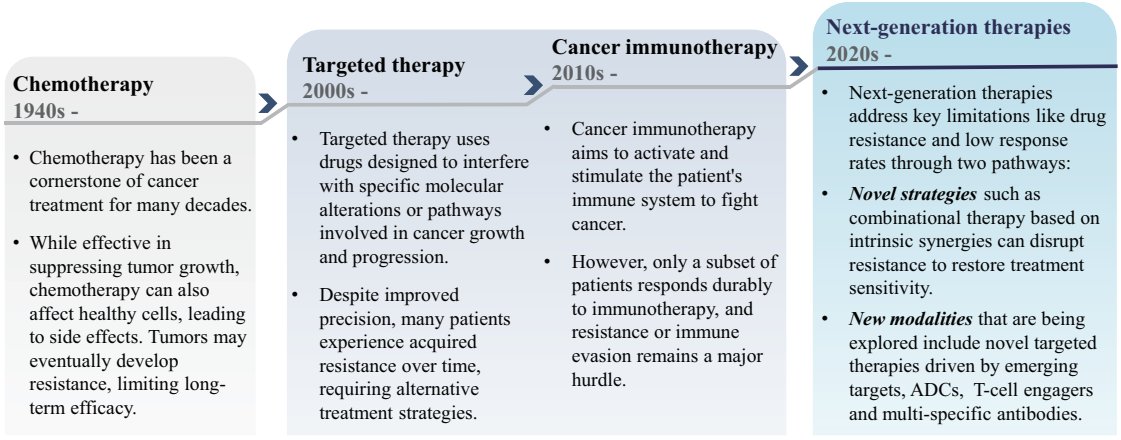
The information and statistics set out in this section and other sections of this document were extracted from different official government publications, available sources from public market research and other sources from independent suppliers, and from the independent industry report prepared by China Insights Industry Consultancy Limited ("CIC") in connection with the **[REDACTED]** *(the "CIC Report"). The information from official government sources has not been independently verified by us, the Joint Sponsors, the* **[REDACTED]***, any of their respective directors and advisers, or any other persons or parties involved in the* **[REDACTED]***, and no representation is given as to its accuracy.*

GLOBAL AND CHINA CANCER TREATMENT MARKET

Cancer is the leading cause of death globally, responsible for approximately 10 million deaths worldwide and over 2.5 million deaths in China in 2024. The global incidence of cancer reached 20.8 million cases in 2024 and is projected to increase to 26.6 million by 2035. In China, there were approximately 5.1 million new cancer cases in 2024, with the number expected to rise to 6.3 million by 2035.

Evolvement of Cancer Treatment

Cancer treatment has evolved significantly over the past decades, from chemotherapy to targeted therapy, immunotherapy, and next-generation therapies, as illustrated in the diagram below.



Source: CA-A CANCER JOURNAL FOR CLINICIANS, Nature Reviews Disease Primers, Cancer Discovery, CIC

Targeted therapy has become a cornerstone of modern oncology, directly disrupting the molecular drivers of tumor growth to enhance efficacy while sparing healthy cells. Innovations such as antibody-drug conjugates (ADCs), novel molecular engineering strategies, and improved payload delivery systems have expanded treatment options and enabled more precise therapies with reduced toxicity. Meanwhile, immunotherapy harnesses the patient's immune system to recognize and attack tumors, offering durable responses across various cancer types. Together, these modalities reflect a continued shift towards more effective and personalized cancer treatment.

POTENTIAL OPPORTUNITIES IN CANCER TREATMENT

Addressing Drug Resistance

Advancements in cancer therapies have significantly improved patient prognosis and overall survival. However, drug resistance remains one of the most formidable challenges in oncology. Whether intrinsic or acquired, as illustrated in the table below, resistance has led to a higher prevalence of treatment-resistant tumors, with approximately 80% to 90% of cancer patient mortality attributed to drug resistance, representing a significant unmet medical need.

Types of cancer drug resistance

	Primary Resistance	Acquired Resistance
Description	• A clinical scenario where a cancer does not respond to a therapy strategy • The mechanistic basis of lack of response to therapy may include adaptive immune resistance	• A clinical scenario in which a cancer initially responded to cancer therapy but after a period of time it relapsed and progressed
Main reasons	• Pre-existing target mutations in cancer cells • Rapid adaptive responses to therapy by cancer cells	• Increased drug efflux and reduced drug uptake • Mutations in drug targets • Alterations in signaling pathways essential for cancer cell survival • Phenotypic remodeling of cancer cells under therapeutic stress, among others

Source: Cell, CIC

Cancer employs various intrinsic and adaptive mechanisms to evade therapeutic effects. Two key mechanisms being explored are (i) genetic mutation-driven drug resistance and (ii) non-genetic drug resistance:

- **Genetic mutation-driven drug resistance.** This resistance arises when cells acquire heritable DNA changes — such as point mutations, insertions, deletions, or gene amplifications, that enable them to evade drug effects. These changes alter drug targets or downstream pathways, resulting in a permanently resistant phenotype. Genomic studies have identified four main categories: mutations affecting drug-target interactions, mutations activating compensatory pathways, mutations enhancing DNA repair capacity, and mutations inhibiting apoptotic cell death.

- **Non-genetic driven drug resistance.** This resistance involves reversible cellular adaptations without DNA sequence changes, including epigenetic modifications, metabolic reprogramming, and cellular plasticity. These adaptations enable transient survival under therapy and may precede or co-exist with genetic resistance.

Key strategies to counter drug resistance have focused on genetic mutation-driven resistance, such as third-generation EGFR-TKIs for treating non-small cell lung cancer (NSCLC), PIK3CA-targeted strategies in anti-HER2 therapies, and BRAF/MEK inhibitors in melanoma. For example, osimertinib was designed to target T790M-mutated NSCLC, a secondary mutation caused by earlier EGFR-TKIs. While targeting genetic mutations has led to significant advances, focusing solely on this aspect has led to challenges from non-genetic resistance mechanisms.

To overcome these challenges, combination therapies targeting multiple resistance mechanisms are under development. Emerging druggable targets such as focal adhesion kinase (FAK) are being explored to delay resistance and enhance treatment durability. Selective FAK inhibitors have shown promise in addressing both primary and acquired resistance. Furthermore, strategies aimed at remodeling the tumor microenvironment (TME) are being investigated to convert "cold" tumors into immunologically active ones, such as targeting cancer-associated fibroblasts (CAFs), modulating cytokine profiles, and employing rational immunotherapy combinations.

Expanding The Therapeutic Window

The therapeutic window refers to the range of drug dosages that can safely and effectively treat a condition. Drugs with a narrow therapeutic window have little room between the effective dose and the toxic dose, requiring precise dosing and careful monitoring. Conversely, drugs with a wide therapeutic window offer more flexibility with reduced risk of toxicity. Many potent therapies, especially ADCs, are often associated with a narrow therapeutic window, showing significant toxicities that restrict both dosage and duration of treatment. A narrow therapeutic window can compromise both efficacy and patient quality of life, making it challenging to balance clinical benefits with potential risks. To enhance safety and maximize therapeutic outcomes, research efforts are focused on several key strategies:

- **Combination therapy**. This approach addresses narrow therapeutic windows by leveraging synergistic mechanisms through targeting multiple pathways. For example, bevacizumab combined with chemotherapy in ovarian and colorectal cancers extends PFS by approximately two months longer than predicted by drug independence alone. Combination therapy can also reduce overall toxicity by allowing each drug to be used at lower doses while maintaining efficacy. Combining FAK inhibitors with ADCs has shown promise in expanding the therapeutic window by enhancing tumor uptake at the same systemic drug concentration. Emerging small-molecule inhibitors, such as FAK inhibitors, demonstrate combinational benefits with multiple cancer treatments.

- **Drug design and precision medicine**. Modern drug development emphasizes precision drug design and targeted patient selection to maximize therapeutic outcomes while minimizing adverse effects. Drug design offers sophisticated approaches to expand therapeutic windows through detailed understanding of protein-ligand interactions and tumor-specific delivery. Precision medicine guides biomarker-based patient selection for more personalized treatment approaches.

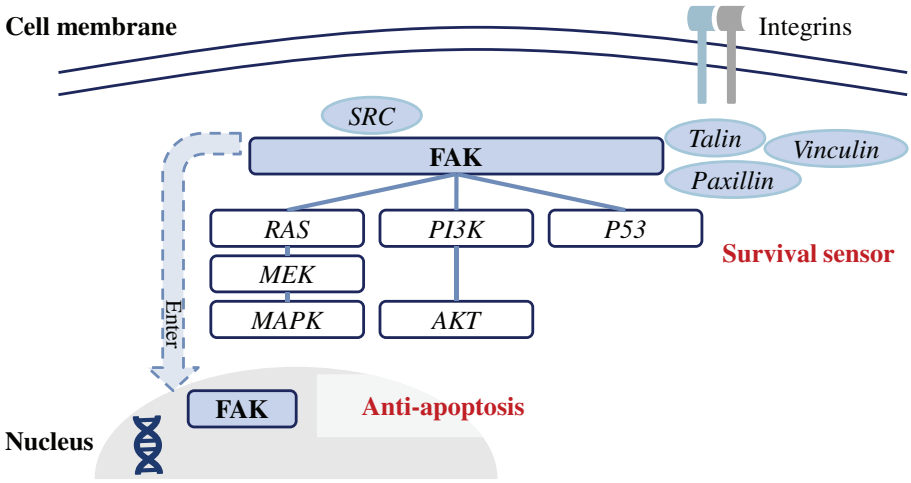
OVERVIEW OF FOCAL ADHESION KINASE INHIBITORS

Introduction of FAK and FAK Inhibitors

Introduction and Functions of FAK

Focal adhesion kinase a non-receptor cytoplasmic tyrosine kinase encoded by the PTK2 gene, critical for regulating cellular adhesion, migration, proliferation, and survival. FAK comprises three functional domains: an N-terminal FERM domain, a central kinase domain, and a C-terminal FAT domain. These domains integrate extracellular signals from integrins, growth factor receptors, and mechanosensors, facilitating downstream signaling cascades such as the PI3K/AKT, MAPK, and Rho GTPase pathways.

FAK involvement in signaling pathways



Source: Experimental Hematology & Oncology, CIC

FAK is over-expressed in numerous malignancies, including colorectal, breast, lung, and ovarian cancers, where it drives tumor progression by promoting epithelial-mesenchymal transition (EMT), angiogenesis, and immune evasion. Its nuclear localization further modulates transcription factors and inflammatory responses, positioning FAK as a central node in TME remodeling.

Mechanism of Action of FAK Inhibitors

FAK inhibitors target either the kinase domain (as adenosine triphosphate (ATP)-competitive inhibitors) or protein-protein interaction sites (as allosteric inhibitors), disrupting FAK-mediated signaling. By suppressing autophosphorylation at Y397 and the subsequent recruitment of Src-family kinases, these inhibitors attenuate downstream pro-survival and metastatic pathways. Pre-clinical studies have demonstrated that FAK inhibition reduces cancer stem cell (CSC) activity, enhances chemotherapy sensitivity, and reverses immunosuppressive TME conditions by decreasing regulatory T cell (Treg) infiltration and cytokine secretion.

Synergistic Effects of FAK Inhibitors in Combination Therapies

FAK inhibitors exhibit synergistic potential across multiple treatment modalities:

- **Chemotherapy.** FAK blockade overcomes chemoresistance by disrupting CSCs and DNA repair. It reduces ALDH+ CSCs via Wnt/β-catenin pathway inhibition and downregulates BRCA1/Rad51, lowering the IC50 of platinum drugs. By blocking IL-8/CXCR1 survival signals, FAK inhibitors enhance taxane efficacy, effectively reversing adaptive resistance to microtubule-targeting agents.

- **Targeted Therapy.** Co-inhibition of FAK with KRAS or MEK inhibitors overcomes resistance via YAP/TAZ pathway modulation. When KRAS or MEK inhibitors are used alone, they paradoxically activate YAP/TAZ via Hippo pathway suppression, enabling cancer cells to evade apoptosis and sustain proliferation through metabolic rewiring and stromal remodeling. FAK inhibitors block this resistance by disrupting FAK-YAP interactions, reducing nuclear YAP/TAZ levels and restoring pro-apoptotic gene expression.

- **Immunotherapy.** FAK inhibitors amplify immunotherapy efficacy by remodeling the TME. They suppress immunosuppressive chemokines like CXCL12 and CCL2, boosting CD8+ T cell infiltration. By targeting CAFs, collagen deposition decreases, enhancing T cell penetration. FAK inhibition also dynamically regulates PD-L1: it transiently enhances MHC-I antigen presentation while blocking IFN-γ driven PD-L1 upregulation, creating a sustained immune-permissive environment.

- **New Modalities.** Next-generation modalities such as ADCs and other engineered therapies are designed to overcome resistance related to tumor heterogeneity, immune evasion, and stromal barriers. By enabling targeted payload delivery and modulating the TME, these approaches improve treatment precision and durability. Combining them with agents like FAK inhibitors may further expand the therapeutic window and enhance response across multiple settings.

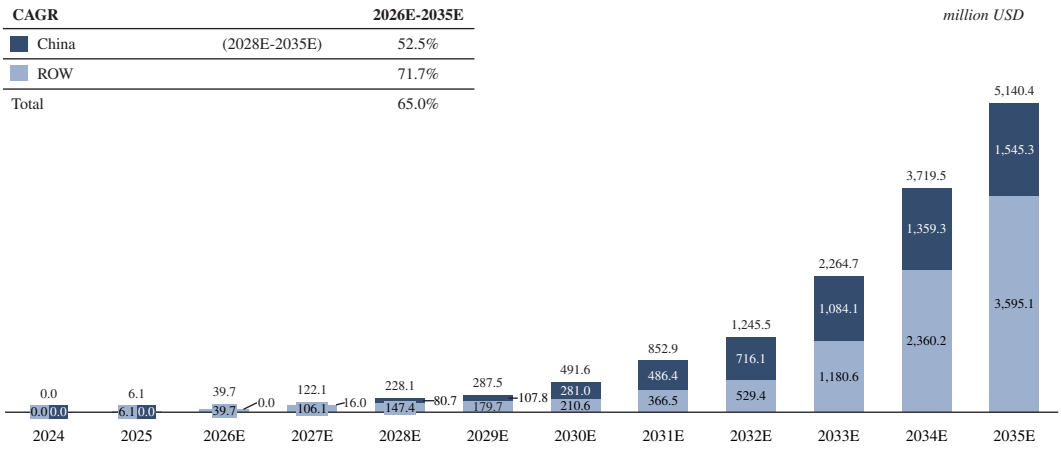
These combination benefits position FAK inhibitors as potential backbone therapies, especially in tumors resistant to monotherapies.

Addressable Market Size

The first selective FAK inhibitor was approved by the FDA in May 2025 for the treatment of patients with KRAS-mutated recurrent low-grade serous ovarian carcinoma (LGSOC). The following chart sets forth the addressable market size of selective FAK inhibitor globally.

Market size of selective FAK inhibitors globally, 2024-2035E

CAGR		2026E-2035E
China	(2028E-2035E)	52.5%
ROW		71.7%
Total		65.0%



Source: Molecular Cancer, Cancer, Cancer Journal of Clinicians, CSCO, ASCO, WHO, CIC

Global Competitive Landscape of Selective FAK Inhibitors

As of the Latest Practicable Date, one selective FAK inhibitor had been approved by the FDA. Globally, four other selective FAK inhibitors remained in active clinical development, among which ifebemtinib (IN10018) was in an advanced position. The table below outlines the global competitive landscape of selective FAK inhibitors.

Competitive landscape of selective FAK inhibitors globally, as of the Latest Practicable Date

Drug Name[1]	Target	Company	Indication	Phase[2]	First Posted Date	Trial Location
Defactinib	FAK	Verastem Oncology/Pfizer	Patient with Kras-mutated recurrent LGSOC	Marketed	2025-05-08[3]	US
			EOC, PDAC, NSCLC, uveal melanoma, MOC, HGSOC, Endometrioid cancer, Thyroid cancer, CRC, other solid tumors	II	2018-10-31	US, others
Ifebemtinib (IN10018)	**FAK**	**InxMed**	**PROC, CRC, NSCLC, PDAC, melanoma, ESCLC, gastric-type cervical adenocarcinoma, GEJ adenocarcinoma, other KRAS G12C solid tumors**	**III**	**2024-07-26**	**China**
Narmafotinib	FAK	Amplia Therapeutics, Canthera Discovery	Unresectable or metastatic pancreatic cancer	I/II	2022-04-27	Others
IN0028	**FAK**	**InxMed**	**Solid tumors**	**I/Ib**	**2026-04-03**	**China**

Notes: 1. Multi-target FAK inhibitors are not included; discontinued, suspended, or otherwise inactive pipelines are also excluded; 2. Highest clinical phase; and 3. FDA approval date.

Source: clinicaltrials.gov, CDE, CIC

Growth Drivers and Future Trends of the Global FAK Inhibitors Market

- **Expanding Patient Base with Significant Unmet Needs**. The aging population and lifestyle factors have contributed to increasing cancer cases globally and in China. While advanced therapies have improved efficacy and safety profiles, drug resistance remains a persistent challenge. This underscores the need for developing FAK inhibitors to potentially overcome drug resistance, thereby driving market growth.

- **Growing Understanding of the Tumor Microenvironment**. The TME has become a pivotal focus in understanding resistance to cancer therapies, particularly ICIs. FAK, as a central signaling node within the TME, drives immunosuppression by promoting the recruitment and activation of Tregs, tumor-associated macrophages (TAMs), and CAFs. Its over-expression correlates with "cold" tumors, in which limited T cell infiltration and heightened immunosuppressive signals render ICIs ineffective. Cutting-edge technologies such as single-cell sequencing and spatial omics are increasingly deployed to decode FAK's multi-faceted role in TME modulation, accelerating the development of FAK inhibitors tailored to disrupt critical oncogenic signaling networks.

- **Pipeline Expansion and Collaborative Research**. Numerous pharmaceutical companies are investing in FAK inhibitor research, and partnerships between pharmaceutical companies, academic centers, and research organizations will accelerate clinical development and market entry for new FAK-targeted drugs. A growing number of trials are testing these agents alone and in combination with chemotherapy and immunotherapy. Currently, four FAK inhibitor candidates are being tested in more than 50 trials globally, spanning Phase I to II/III trials. This therapeutic strategy is being rigorously tested across diverse solid tumors, including ovarian carcinoma, RAS-mutated solid tumors, mesothelioma, NSCLC, pancreatic ductal adenocarcinoma, and triple-negative breast cancer. As FAK inhibitors continue to show promise, particularly in combination regimens, pipeline expansion and collaborative research remain key growth drivers in this field.

- **Explicit Policy Support for Combination Therapy**. Regulatory bodies play a crucial role in the development of new drugs by establishing clear guidelines and technical standards that ensure the safety, efficacy, and quality of FAK inhibitors. In 2013, the FDA issued the *Guidance for Industry Co-development of Two or More Unmarketed Investigational Drugs for Use in Combination*. In 2020, the CDE issued *Technical Guidelines for Clinical Trials of Anti-Tumor Drug Combination Therapy*. These regulatory policies act as significant growth drivers for the FAK inhibitors market by providing clear and supportive frameworks for the development and clinical evaluation of combination therapies.

Entry Barriers of Global FAK Inhibitors Market

- **Early-entrant Advantage.** Early entrants in the FAK inhibitor space benefit from priority access to key resources such as eligible patients, trial sites, and strategic industry collaborations, allowing them to establish leadership in recognition, KOL influence, and clinical progress. Strong early clinical progress can also position these companies for expedited regulatory pathways in China and the United States, as they are among the first to evaluate innovative therapies in diseases that otherwise lack effective treatment. In contrast, later entrants may face significant disadvantages in competing for trial sites and patients, differentiating their drug candidate, securing policy support, and ultimately achieving market access.

- **R&D Capability Barrier.** Developing FAK inhibitors requires deep expertise in kinase biology, structural chemistry, and TME dynamics. FAK's dual functions, supporting tumor survival and driving stromal-mediated immune suppression, make pre-clinical modeling challenging, as murine models often fail to capture human-specific interactions and may require expensive humanized systems. Additionally, optimizing selectivity to avoid off-target effects requires extensive structure-activity relationship (SAR) studies, with over 80% of candidates failing in early stages due to poor pharmacokinetics or toxicity. New entrants would face hurdles including but not limited to substantial investment, talent acquisition, and partnership building.

- **Regulatory Barrier**. Cancer therapeutics, including FAK inhibitors, face stringent regulatory requirements that create substantial market entry barriers. Regulatory compliance throughout clinical development is complex, particularly challenging for late entrants. Additionally, regulatory standards for combination therapy evaluation vary significantly across major markets.

MAJOR INDICATIONS

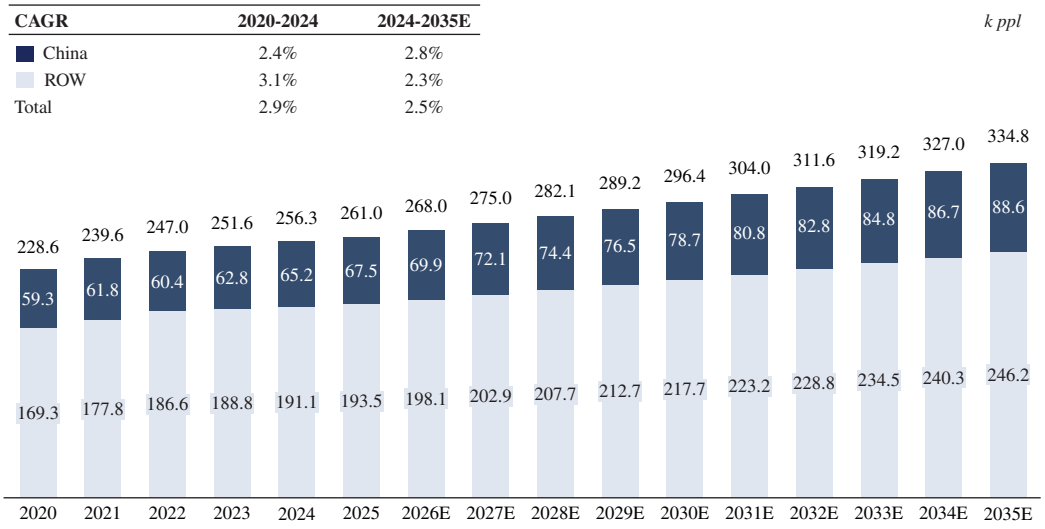
Overview of Pan-RAS Cancers

Pan-RAS cancers are malignancies driven by mutations in the RAS gene family (KRAS, NRAS, HRAS). These genes encode small GTPases central to cell proliferation, differentiation, and survival. RAS mutations constitutively activate downstream MAPK and PI3K pathways, contributing to oncogenesis and therapeutic resistance. Present in approximately 30% of all human cancers, RAS mutations drive tumor growth and metastasis. Pan-RAS cancers span a broad spectrum of solid tumors, notably NSCLC, CRC, and PDAC (KRAS-prevalent), melanoma and hematologic malignancies (NRAS), and certain head and neck and bladder cancers (HRAS).

KRAS-mutated NSCLC

Lung cancer is the most common cancer worldwide and in China, with incidence in China increasing from 976.6 thousand cases in 2020 to 1,144.9 thousand in 2024, expected to reach 1,556.2 thousand by 2035. NSCLC represents approximately 85% of lung cancers globally and is a leading cause of cancer death with significant unmet need. Among KRAS-mutated NSCLC patients, G12C, G12V, and G12D subtypes account for approximately 40%, 21%, and 17% globally (29.6%, 17.5%, and 18.1% in China, respectively). KRAS G12C, prevalent among smokers and Western populations, is found in 10-13% of NSCLC cases globally but only 1.4-4.3% in China, and is associated with poorer prognosis. KRAS G12D occurs in 4-5% of cases globally and approximately 2% in China.

Incidence of KRAS-mutated NSCLC globally, 2020-2035E



Note: This figure only represents the incidence of NSCLC patients with KRAS G12C or KRAS G12D mutations.

Source: Globocan, IARC, NCCR, CIC

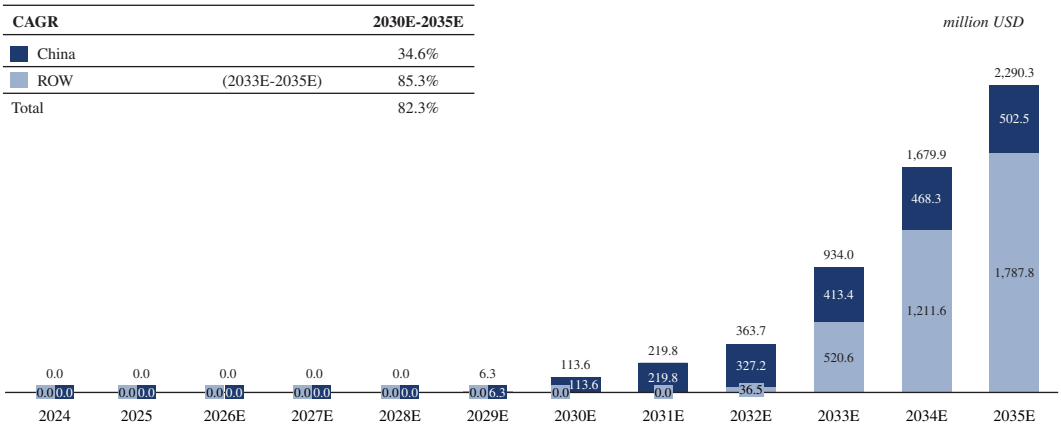
Treatment Paradigm

NSCLC treatment typically involves surgery, chemotherapy, targeted therapy, and immunotherapy based on stage and molecular profile. While targeted therapies have improved outcomes for patients with EGFR and ALK mutations, effective options for KRAS G12C-mutated NSCLC remain limited. Recent KRAS G12C inhibitors have improved treatment options but face challenges including drug resistance, short response duration, limited earlier-line options, and liver toxicity that prohibits combination with anti-PD-1 antibodies. No targeted treatment exists for KRAS G12D-mutated NSCLC. The complex biology of KRAS mutations underscores the significant unmet need for more effective and durable therapies.

Addressable Market Size

The following chart sets forth the market size of selective FAK inhibitors in KRAS-mutated NSCLC in China and globally.

Market size of KRAS-mutated NSCLC treatment with selective FAK inhibitors globally, 2024-2035E



Source: IARC, NCCR, CSCO, CIC

Competitive Landscape

Two selective FAK inhibitors are currently in clinical development globally: Defactinib is in Phase II trials, while ifebemtinib is in Phase III trials and leads in China. This highlights ongoing efforts to develop effective selective FAK inhibitors for challenging indications such as KRAS-mutated NSCLC.

Pipelines of selective FAK inhibitors for KRAS-mutated NSCLC globally, as of the Latest Practicable Date

Drug Name	Target	Company	Indication	Phase	First Posted Date	Trial Number	Trial Location
Ifebemtinib (IN10018)	**FAK**	**InxMed**	• **1L treatment of patients with KRAS G12C-mutated, locally advanced or metastatic non-squamous NSCLC**	**III**	**2025-08-18**	**CTR20253319**	**China**
			• **2L+ treatment for locally advanced or metastatic KRAS G12D-mutated NSCLC**	**Ib/II**	**2025-12-09**	**CTR20254790**	**China**
Defactinib	FAK	Verastem Oncology/Pfizer	• Patients with KRAS mutant NSCLC	II	2013-09-26	NCT01951690	US
			• KRAS- or BRAF-mutant NSCLC post platinum-based chemotherapy and immune checkpoint inhibitor treatment	II	2020-11-06	NCT04620330	US, Others
			• Non-small cell lung cancer, pancreatic cancer, mesothelioma	I/II	2016-05-02	NCT02758587	Others

Source: Clinicaltrials.gov, CDE, CIC

The following chart sets forth the approved drugs and drug candidates targeting KRAS-mutated NSCLC globally.

Approved drugs for KRAS-mutated NSCLC globally, as of the Latest Practicable Date

MoA	Generic Name[1]	Brand Name	Company	mPFS (months)	China Approval Date	Pricing in China per patient (k RMB)[2]	2025 NRDL	USA Approval Date	Pricing in the US per patient (k USD)[2]	Administration	Mono/Combo	Treatment Line
KRAS G12C inhibitors	Sotorasib	Lumakras	Amgen	6.3	-	-	-	2021-5-8	~133	Oral	Mono	2L+
	Adagrasib	Krazati	Bristol	5.5	-	-	-	2022-12-12	~121	Oral	Mono	2L+
	Fulzerasib	達伯特	Innovent	9.7	2024-8-20	~120	Yes	-	-	Oral	Mono	2L+
	Glecirasib	艾瑞凱	Allist	8.2	2025-5-22	~98	Yes	-	-	Oral	Mono	2L+
	Garsorasib	安方寧	Chia Tai Tianqing	9.1	2024-11-5	~100	Yes	-	-	Oral	Mono	2L+
	Sosimerasib	濟樂美	Jemincare/ HUYA	7.2	2026-2-25	-	-	-	-	Oral	Mono	2L+

Notes: 1. Charity-based free medication programs are excluded; and 2. pricing per patient = monthly price * mPFS.

Source: NRDL, Drugs@FDA, NMPA, Drug labels, CIC

Clinical-stage drug candidates for KRAS-mutated NSCLC (Phase III and above), as of the Latest Practicable Date

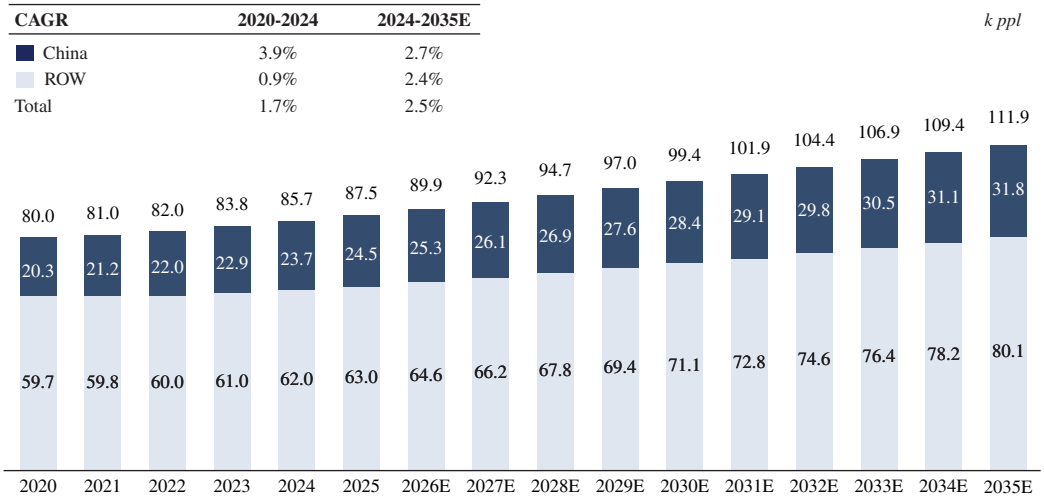
Drug Name	Target	Indication	Company	Phase	First Posted Date	Trial Number	Trial Location
Olomorasib	KRAS G12C	KRAS G12C mutated NSCLC	Eli Lilly	III	2023-11-07	NCT06119581	Global
Divarasib	KRAS G12C	KRAS G12C mutated NSCLC	Roche / Genentech	III	2024-07-11	NCT06497556	Global
MK-1084	KRAS G12C	KRAS G12C mutated NSCLC	Merck	III	2024-04-03	NCT06345729	Global
HJ891	KRAS G12C	KRAS G12C mutated NSCLC	HJ Science	III	2024-01-08	CTR20240054	China

Source: clinicaltrials.gov, CDE, CIC

KRAS G12C-mutated Colorectal Cancer

CRC is the second most prevalent cancer in China, with an annual incidence of 536.9 thousand cases in 2023, which is expected to reach 746.4 thousand cases by 2035. As CRC patients typically do not display symptoms until the disease is advanced, CRC is also one of the leading causes of cancer deaths in China. KRAS G12C mutations, present in 3-4% of CRC cases, drive tumorigenesis through constitutive activation of the MAPK and PI3K pathways. The following chart sets forth the incidence of KRAS G12C-mutated CRC.

Incidence of KRAS G12C-mutated Colorectal Cancer globally, 2020-2033E



Source: WHO, the Lancet, CIC

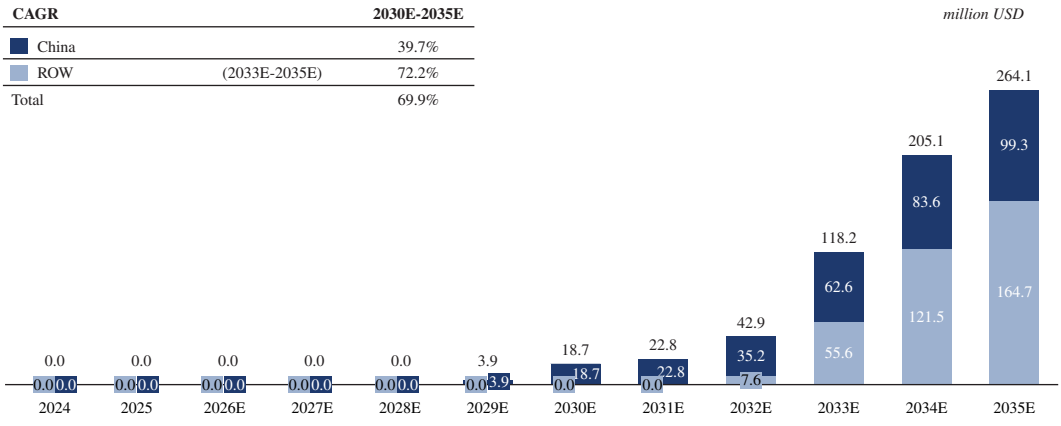
Treatment Paradigm

For patients with microsatellite stable (MSS) or mismatch repair proficient (pMMR) advanced CRC harboring RAS or BRAF mutations, first-line (1L) treatment options include doublet chemotherapy (FOLFOX, FOLFIRI, or CAPEOX) ± bevacizumab or fluorouracil ± bevacizumab. Second-line (2L) therapy may involve cross-over regimens (e.g., FOLFIRI for prior oxaliplatin-based treatment or CAPEOX/FOLFOX for prior irinotecan-based treatment) ± bevacizumab. Third-line (3L) options consist of regorafenib, fruquintinib, or trifluridine/tipiracil ± bevacizumab. Importantly, targeted therapies for KRAS subtypes are not yet available in clinical treatment guidance. KRAS mutations have historically been considered "undruggable," with chemotherapy regimens combined with bevacizumab remaining the standard first line therapy, yet there is an absence of approved chemotherapy-free targeted options for KRAS-mutated advanced CRC. In later-line treatment, therapeutic outcomes are still inadequate despite recent targeted advances in clinical trials. Current targeted therapies, such as sotorasib and adagrasib, selectively inhibit KRAS G12C by trapping it in an inactive GDP-bound state. However, the efficacy of these agents in KRAS G12C-mutated CRC remains limited. Monotherapy with adagrasib yields an ORR of 19%, rising to 46% in combination with cetuximab. Median progression-free survival (mPFS) was 5.6 months and 6.9 months for adagrasib monotherapy and the combination, respectively. In a recent Phase II clinical trial of sotorasib, a response of 10% was observed, with a mPFS of 4.2 months in pre-treated patients with metastatic KRAS G12C-mutated CRC. The clinical benefit of KRAS G12C targeted therapies in CRC remains suboptimal, with limited durability of response (mPFS less than or equal to 7 months). Substantial unmet needs persist for patients progressing on current targeted regimens, necessitating novel strategies to overcome adaptive resistance and achieve durable disease control.

Addressable Market Size

The first selective FAK inhibitor is expected to be approved by 2029 in China for the treatment of KRAS G12C-mutated CRC. The following charts set forth the addressable market size of selective FAK inhibitors globally.

Market size of KRAS G12C-mutated colorectal cancer treatment with selective FAK inhibitors globally, 2024-2035E



Source: Transl Lung Cancer Res, The ASCO Post, Molecular Cancer, Cancer, Cancer Journal for Clinicians, WHO, CIC

Competitive Landscape

As of the Latest Practicable Date, no FAK inhibitor has received regulatory approval globally for the treatment of KRAS G12C-mutated CRC. Two selective FAK inhibitor candidates were under active clinical development as of the same date, all of which were in Phase I/II or earlier stages. A summary of the global competitive landscape of FAK inhibitors is set forth below.

Pipeline of selective FAK inhibitors for KRAS G12C-mutated colorectal cancer undergoing clinical trials globally, as of the Latest Practicable Date

Drug Name	Target	Company	Indication	Phase	First Posted Date	Trial Number	Trial Location
Ifebemtinib (IN10018)	**FAK**	**InxMed**	• **Locally advanced or metastatic solid tumors positive for KRAS G12C mutation**	**I/II**	**2024-01-12**	**NCT06166836 CTR20240092**	**China**
Defactinib	FAK	Verastem Oncology/Pfizer	• Advanced solid tumors	I	2019-03-12	NCT03875820	Others

Source: clinicaltrials.gov, CDE, CIC

The following chart sets forth the approved drugs and drug candidates targeting KRAS G12C-mutated colorectal cancer globally.

Approved drugs for KRAS G12C-mutated colorectal cancer globally, as of the Latest Practicable Date

MoA	Generic Name	Brand Name	Company	mPFS (months)	China Approval Date	Pricing in China per Patient (k RMB)	2025 NRDL	US Approval Date	Pricing in the US per Patient (k USD)	Administration	Mono/Combo	Treatment Line
KRAS G12C inhibitors	Sotorasib	Lumakras	Amgen	5.6	-	-	-	2025-1-17	~119	Oral	Combo with Panitumumab	2L+
	Adagrasib	Krazati	Bristol	5.6	-	-	-	2024-6-21	~123	Oral	Combo with Cetuximab	2L+
EGFR inhibitors	Panitumumab	Vectibix	Amgen	5.6	-	-	-	2025-1-17	~76	Injection	Combo with Sotorasib	2L+

Notes: 1. All calculations assume an average adult weight of 60 kg; and 2. pricing per patient = monthly price * mPFS.

Source: NRDL, Drugs@FDA, NMPA, Drug labels, CIC

Global clinical-stage drug candidates for KRAS-G12C mutated CRC, as of the Latest Practicable Date

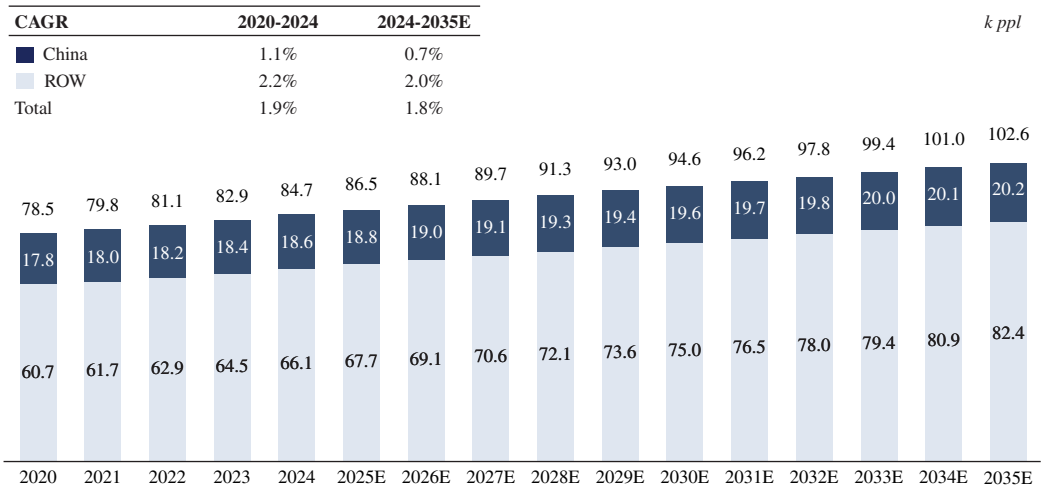
Drug Name	Target	Indication	Company	Phase	First Posted Date	Trial Number	Trial Location
MK-1084	KRAS G12C	KRAS-G12C mutated CRC	Merck	III	2025-05-30	NCT06997497	Global
Divarasib	KRAS G12C	KRAS-G12C mutated CRC	Roche	II	2020-10-19	NCT04589845	Global
Glecirasib	KRAS G12C	KRAS-G12C mutated CRC	Allist	I/II	2021-06-29	NCT05009329	China
Olomorasib	KRAS G12C	KRAS-G12C mutated CRC	Lily	I/II	2021-07-09	NCT04956640	Global excl. China
Fulzerasib	KRAS G12C	KRAS-G12C mutated CRC	Innovent	I/II	2021-08-12	NCT05005234	China
HS-10370	KRAS G12C	KRAS-G12C mutated CRC	Hansoh	I/II	2022-03-28	NCT05367778	China
Elisrasib	KRAS G12C	KRAS-G12C mutated CRC	D3 Bio	I/II	2022-06-08	NCT05410145	Global
Garsorasib	KRAS G12C	KRAS-G12C mutated CRC	InventisBio	I/II	2023-12-12	NCT06166836	China
FMC-376	KRAS G12C	KRAS-G12C mutated CRC	Frontier Med	I/II	2024-02-06	NCT06244771	Global excl. China
SY-5933	KRAS G12C	KRAS-G12C mutated CRC	Shouyao	I/II	2025-04-30	NCT06970132	China
Elironrasib	KRAS G12C	KRAS-G12C mutated CRC	Revolution	I/II	2026-02-09	NCT07397338	US
BI 1823911	KRAS G12C	KRAS-G12C mutated CRC	Boehringer Ingelheim	I	2021-07-22	NCT04973163	Global excl. China
GEC-255	KRAS G12C	KRAS-G12C mutated CRC	GenEros	I	2021-10-22	NCT05768321	China
YL-15293	KRAS G12C	KRAS-G12C mutated CRC	YingLi	I	2021-11-04	NCT05173805	China
Sosimerasib	KRAS G12C	KRAS-G12C mutated CRC	HUYABIO	I	2022-08-03	NCT05485974	Global excl. China
BEBT-607	KRAS G12C	KRAS-G12C mutated CRC	BeBetter	I	2023-06-14	NCT06117371	China
YL-17231	Pan-KRAS	KRAS-G12C mutated CRC	YingLi	I	2023-10-10	NCT06078800	China
HRS-7058	KRAS G12C	KRAS-G12C mutated CRC	Suncadia	I	2024-04-25	NCT06383871	China
KQB365	KRAS G12C, KRAS G12S	KRAS-G12C mutated CRC	Kumquat	I	2024-12-06	NCT06720987	US
BBO-11818	Pan-KRAS	KRAS-G12C mutated CRC	BridgeBio	I	2025-04-08	NCT06917079	US
WJ22096	KRAS G12C	KRAS-G12C mutated CRC	Jingao	I	2025-11-18	NCT07253116	China

Source: clinicaltrials.gov, CDE, CIC

Platinum-Resistant Ovarian Cancer

Platinum-resistant ovarian cancer (PROC) is defined as disease recurrence or progression within six months of platinum-based chemotherapy. Associated with poor prognosis and limited treatment options, PROC remains a major clinical challenge with unsatisfactory outcomes despite standard chemotherapy, underscoring the need for novel therapies. The following chart sets forth the incidence of PROC globally.

Incidence of platinum-resistant ovarian cancer globally, ROW, 2020-2035E



Source: Globocan, IARC, NCCR, CIC

Treatment Paradigm

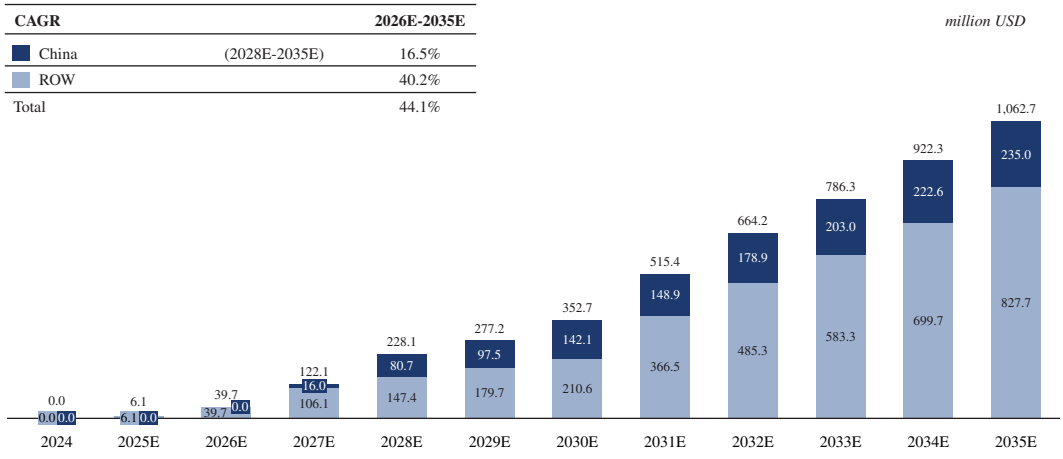
Ovarian cancer treatment in China primarily includes debulking surgery followed by platinum-based chemotherapy, with or without bevacizumab or PARP inhibitors. PARP inhibitor maintenance therapy has improved management in newly diagnosed and recurrent disease. *BRCA1/2* mutations and homologous recombination deficiency (HRD) are predictive biomarkers for platinum-based chemotherapy and PARP inhibitor response; however, HRD-negative patients derive minimal benefit from PARP inhibitors, highlighting significant unmet need for novel therapeutic approaches in this population.

Platinum-based regimens initially achieve good response rates, but most patients eventually relapse and develop resistance, which is defined as PROC. Bevacizumab, while commonly used, is associated with both primary and acquired resistance, reducing its long-term effectiveness. Mirvetuximab soravtansine-gynx, an FRα-targeting ADC, has exhibited satisfactory efficacy in high FRα expression patients, but is associated with frequent and sometimes severe ocular toxicities, as well as notable risks of pneumonitis and other non-ocular AEs, often necessitating dose modifications and intensive monitoring. These limitations underscore the need for alternative strategies addressing broader patient populations and overcoming resistance, driving interest in targets such as FAK.

Addressable Market Size

The following chart sets forth the addressable market size of ovarian cancer treatment with selective FAK inhibitor globally.

Market size of OC treatment with selective FAK inhibitors globally, 2024-2035E



Source: IARC, NCCR, CSCO, CIC

Competitive Landscape

The competitive landscape of selective FAK inhibitors for PROC is evolving, with growing interest in targeting novel pathways. Defactinib has received FDA approval for the treatment of KRAS-mutated recurrent LGSOC, representing a key step forward in this area. In addition, ifebemtinib has entered Phase III trial, showing advanced progress compared to others.

Pipelines of selective FAK inhibitors for platinum-resistant ovarian cancer globally, as of the Latest Practicable Date

Drug Name	Target	Company	Indication	Phase[1]	First Posted Date	Trial Number	Trial Location
Defactinib	FAK	Verastem Oncology/Pfizer	Patient with KRAS-mutated recurrent LGSOC	Markted	2025-05-08[2]	/	US
			Endometrioid cancer, mucinous ovarian cancer, HGSOC, cervical cancer, solid tumor	II	2022-08-19	NCT05512208	US
Ifebemtinib (IN10018)	**FAK**	**InxMed**	**Platinum-resistant recurrent ovarian cancer**	**III**	**2024-07-26**	**CTR20221614**	**China**

Note: 1. Highest clinical phase; 2. FDA approval date.

Source: Clinicaltrials.gov, CDE, CIC

The following chart sets forth the approved drugs and drug candidates targeting platinum-resistant ovarian cancer globally.

Approved drugs for platinum-resistant ovarian cancer globally, as of the Latest Practicable Date

MoA	Generic Name	Brand Name	Company	mPFS (months)	China Approval Date	Pricing in China per Patient (k RMB)	2025 NRDL	US Approval Date	Pricing in the US per Patient (k USD)	Administration	Mono/Combo	Treatment Line
VEGF inhibitors	Bevacizumab	AVASTIN	Roche	6.8	-	-	-	2014-11-14	~68	Injection	combo with chemo	2L+
	Sevacizumab	恩泽舒	Simcere	5.5	2025/6/30	~44	Yes	-	-	Injection	combo with chemo	2L
FRα ADC	Mirvetuximab soravtansine	ELAHERE/ 愛拉赫	AbbVie, Huadong	5.6	2024/11/22	~252	No	2022-11-14	~190	Injection	Mono	2L+
RAF/MEK inhibitors	Avutometinib	Avmapki	Verastem	22[3]	-	-	-	2025-05-08	~95	Oral	Combo with Defactinib	2L+

Notes: 1. All calculations assume an average adult body surface area of 1.7 m^2; 2. pricing per patient = monthly price * mPFS; and 3. AVMAPKI and FAKZYNJA combination is approved for adult patients with KRAS-mutated recurrent low-grade serous ovarian cancer (LGSOC) following prior systemic therapy. The reported mPFS of 22 months pertains to the broader recurrent LGSOC population, not exclusively to platinum-resistant ovarian cancer patients.

Source: NRDL, Drugs@FDA, NMPA, Drug labels, CIC

Global clinical-stage drug candidates for OC (Phase III and above), as of the Latest Practicable Date

Drug Name	Target	Indication	Company	Phase	First Posted Date	Trial Number	Trial Location
Dostarlimab	PD-1	Ovarian cancer	Tesaro	III	2018/7/27	NCT03602859	Global excl. China
Durvalumab	PD-L1	Ovarian cancer	AZ	III	2018/11/9	NCT03737643	Global
Oregovomab	MUC16	Ovarian cancer	CanariaBio	III	2020/8/4	NCT04498117	Global excl. China
Batiraxcept	AXL	Ovarian cancer	Aravive	III	2021/1/28	NCT04729608	Global
Alpelisib	PI3Kα	Ovarian cancer	Novartis	III	2021/1/28	NCT04729387	Global
Chiauranib	AURKB, CSF1R, KIT, PDGFR, VEGFR	Ovarian cancer	Chipscreen	III	2021/6/10	NCT04921527	China
Lorlatinib	ALK, ROS1	Ovarian cancer	Pfizer	III	2021/9/28	NCT05059522	Global excl. China
Avelumab	PD-L1	Ovarian cancer	Pfizer	III	2021/9/28	NCT05059522	Global excl. China
Talazoparib	PARP1, PARP2	Ovarian cancer	Pfizer	III	2021/9/28	NCT05059522	Global excl. China
Axitinib	VEGFR, KIT, PDGFR	Ovarian cancer	Pfizer	III	2021/9/28	NCT05059522	Global excl. China
TQB3823	PARP1, PARP2	Ovarian cancer	Chia Tai Tianqing	III	2023/6/5	CTR20231645	China
SHR-A1921	TROP2	Ovarian cancer	Suncadia	III	2024/5/1	NCT06394492	China
Anbenitamab repodatecan	HER2	Ovarian cancer	Alphamab	III	2024/12/27	NCT06751485	China
Trastuzumab deruxtecan	HER2	Ovarian cancer	Daiichi	III	2025/2/11	NCT06819007	Global
trastuzumab rezetecan	HER2	Ovarian cancer	Hengrui	III	2025/2/14	NCT06828354	China
IMNN-001	IL12	Ovarian cancer	Imunon	III	2025/4/7	NCT06915025	US
Izalontamab brengitecan	HER3	Ovarian cancer	Baili	III	2025/5/29	NCT06994195	China
HS-20089	B7-H4	Ovarian cancer	GSK	III	2025/12/16	NCT07286266	Global
Sacituzumab tirumotecan	TROP2	Ovarian cancer	Merck	III	2026/1/6	NCT07318558	Global

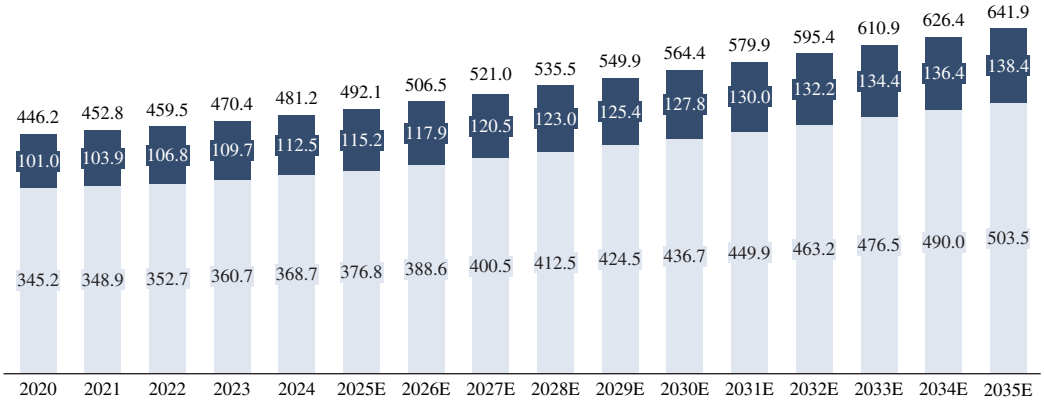
Source: clinicaltrials.gov, CDE, CIC

Pancreatic Ductal Adenocarcinoma

Pancreatic cancer is a highly aggressive malignancy with a poor prognosis and limited effective treatment options, posing a significant clinical challenge worldwide. Despite advances in therapies, overall survival remains low, highlighting the urgent need for improved treatment strategies. PDAC is the most common type of pancreatic cancer, accounting for approximately 90% of pancreatic cancer cases and developing from epithelial lining of pancreatic ducts. The following chart sets forth the incidence of PDAC globally.

Incidence of pancreatic ductal adenocarcinoma globally, 2020-2035E

CAGR	2020-2024	2024-2035E
China	2.7%	1.9%
ROW	1.7%	2.9%
Total	1.9%	2.7%



Source: Globocan, IARC, NCCR, CIC

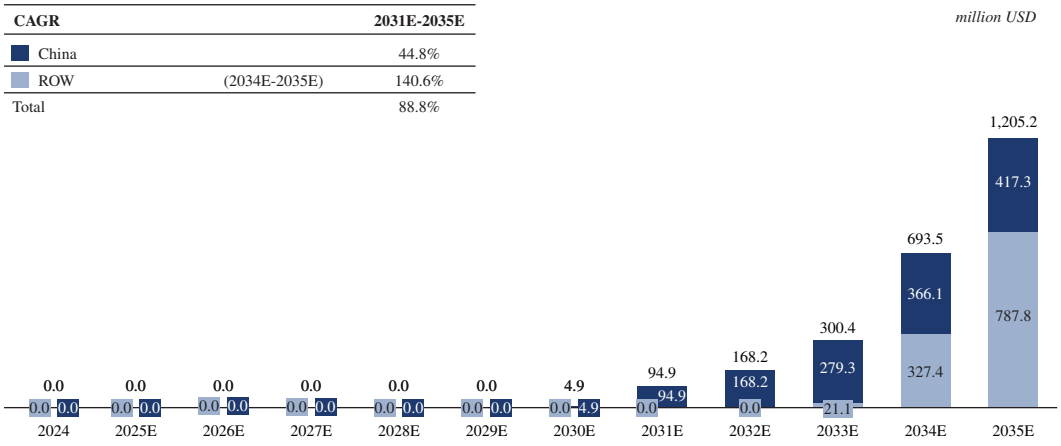
Treatment Paradigm

Pancreatic cancer treatment in China involves surgery for resectable disease with adjuvant gemcitabine- or fluorouracil-based chemotherapy. For advanced cases, FOLFIRINOX or gemcitabine plus nab-paclitaxel are standard 1L options, though long-term outcomes remain poor due to chemoresistance. 2L options (nanoliposomal irinotecan combinations, fluoropyrimidine regimens) offer limited benefits. Targeted therapies apply to only a small subset of patients. Given that ~90% of PDAC cases harbor RAS mutations, effective RAS-targeted therapies could shift treatment paradigms from refractory to 1L settings. Despite advances, pancreatic cancer prognosis remains poor due to high recurrence rates, limited targetable mutations, and an immunosuppressive TME. These challenges underscore the need for novel approaches targeting tumor stroma, CAFs, and signaling pathways such as FAK.

Addressable Market Size

The following chart sets forth the addressable market size of selective FAK inhibitors in PDAC.

Market size of pancreatic ductal adenocarcinoma treatment with selective FAK inhibitors globally, 2024-2035E



Source: Cancer, Chinese Journal of Cancer, CSCO, CIC

Competitive Landscape

The competitive landscape of selective FAK inhibitors for pancreatic cancer is developing globally, with multiple agents advancing through clinical stages. Defactinib is in Phase II trials, while Narmafotinib and ifebemtinib are in early Phase I/II stages. In China, ifebemtinib is the most advanced program among clinical-stage selective FAK inhibitors for pancreatic cancer.

Pipelines of selective FAK inhibitors for pancreatic cancer globally, as of the Latest Practicable Date

Drug Name	Target	Company	Indication	Phase	First Posted Date	Trial Number	Trial Location
Defactinib	FAK	Verastem Oncology/Pfizer	• Advanced pancreas adenocarcinoma	II	2020-03-30	NCT04331041	US
			• Previously untreated pancreatic ductal adenocarcinoma (PDAC)	I/II	2022-12-19	NCT05669482	US
Narmafotinib	FAK	Amplia Therapeutics, Canthera Discovery	• Pancreatic cancer	I/II	2022-04-27	NCT05355298	Others
Ifebemtinib (IN10018)	**FAK**	**InxMed**	• **Advanced pancreatic cancer**	**I/II**	**2022-12-21**	**NCT05827796 CTR20223102**	**China**
			• **$KRAS^{G12D}$ muted la/m PDAC and other solid tumors**	**I/II**	**2025-12-09**	**NCT07441174 CTR20254790**	**China**

Source: Clinicaltrials.gov, CDE, CIC

The following chart sets forth the approved drugs and drug candidates targeting pancreatic cancer globally.

Approved drugs for pancreatic cancer globally[1], as of the Latest Practicable Date

MoA	Generic Name	Brand Name	Company	mPFS (months)	China approval date	Pricing in China per patient (k RMB)[3]	2025 NRDL	US Approval Date	Pricing in the US per patient (k USD)[3]	Administration	Mono/Combo	Treatment Line
Beta-tubulin	Albumin-bound paclitaxel	Abraxane/ 凯素[2]	Bristol	5.5	2023-12-29	~47	Yes	2013-9-6	~58	Injection	Combo with Gemcitabine	1L
TOP1 inhibitors	Irinotecan Hydrochloride Liposome Injection (II)	越優力	Hengrui	4.2	2023-12-29	~32	Yes	-	-	Injection	Combo with chemo	2L+
	Irinotecan Hydrochloride Liposome Injection	Onivyde/ 易安達	Ipsen, Servier	4.0	2022-4-12	~130	No	2015-10-22	~44	Injection	Combo with chemo	2L+
				7.4	-	-	-	2024-2-13	~58	Injection	Combo with chemo	1L
EGFR inhibitors	Nimotuzumab	泰欣生	Biotech Pharma	4.2	2023-6-7	~177	No	-	-	Injection	Combo with gemcitabine	1L
HER2/HER3 inhibitors	Zenocutuzumab	Bizengri	MERUS N.V.	-	-	-	-	2024-12-4	~23	Injection	Mono	2L+

Notes: 1. This list includes only novel drugs (generics and maintenance therapies are excluded); 2. Based on NMPA Announcement No. 44 (2020), the import, sale, and use of Abraxane were suspended on March 25, 2020. Abraxane was formally relaunched in China by August 30, 2024; 3. pricing per patient = monthly price * mPFS; all calculations assume an average adult body surface area of 1.7 m^2.

Source: NRDL, Drugs@FDA, NMPA, Drug labels, CIC

Global clinical-stage drug candidates for pancreatic cancer[1], as of the Latest Practicable Date

Drug Name	Target	Indication	Company	Phase	First Posted Date	Trial Number	Trial Location
Daraxonrasib	Pan-RAS	Advanced pancreatic cancer	Revolution Medicines	III	2026-03-02	NCT07491445	US
BPI-572270	Pan-RAS	Advanced pancreatic cancer	Jingyao Biopharma	I/II	2026-02-11	NCT07435038	China
GFH276	Pan-RAS	Advanced pancreatic cancer	GenFleet	I/II	2025-09-10	NCT07198321	China
JYP0015	Pan-RAS	Advanced pancreatic cancer	Joyo Pharma	I/II	2025-03-07	NCT06895031	China
SIM0532	Pan-RAS	Advanced pancreatic cancer	Simcere	I	2026-03-16	CTR20260901	China
GB3010	Pan-RAS	Advanced pancreatic cancer	Essight Bio	I	2023-09-26	NCT06054984	China
HRS-4642	KRAS G12D	Advanced pancreatic cancer	Hengrui	III	2025-10-24	NCT07232875	China
GFH375	KRAS G12D	Advanced pancreatic cancer	GenFleet	III	2025-11-11	NCT07262567	China
Setidegrasib	KRAS G12D	Advanced pancreatic cancer	Astellas	III	2026-02-13	NCT07409272	US
INCB161734	KRAS G12D	Advanced pancreatic cancer	Incyte	III	2026-04-13	NCT06179160	Global excl. China
AST-001	KRAS G12D	Advanced pancreatic cancer	Ascentawits	II	2022-04-29	CTR20220935	China
Datopotamab deruxtecan	TROP2	Advanced pancreatic cancer	AZ	I/II	2022-05-12	NCT05417594	Global excl. China
DB-1305	TROP2	Advanced pancreatic cancer	DualityBio	I/II	2022-06-29	NCT05438329	Global
XYD-9668-198	TROP2	Advanced pancreatic cancer	Corsera	I/II	2023-04-21	CTR20231203	China
TSN1611	KRAS G12D	Advanced pancreatic cancer	Tyligand	I/II	2024-04-26	NCT06385925	Global
BHV1510	TROP2	Advanced pancreatic cancer	GeneQuantum	I/II	2024-05-09	NCT06464055	China
Sacituzumab tirumotecan	TROP2	Advanced pancreatic cancer	Merck	I/II	2024-05-24	NCT06428409	Global
SHR-A1921	TROP2	Advanced pancreatic cancer	Hengrui	I/II	2024-07-25	NCT06520488	China
DN022150	KRAS G12D	Advanced pancreatic cancer	Kerui	I/II	2024-08-02	CTR20242749	China
AZD0022	KRAS G12D	Advanced pancreatic cancer	AZ	I/II	2024-09-19	NCT06599502	Global
ANOC-003	KRAS G12D	Advanced pancreatic cancer	Anocca	I/II	2025-03-04	NCT07145450	Global excl. China, US
ARV-806	KRAS G12D	Advanced pancreatic cancer	Arvinas	I/II	2025-06-17	NCT07023731	US
HBW-012336	KRAS G12D	Advanced pancreatic cancer	Hyperway	I/II	2025-06-18	CTR20252400	China
RNK08954	KRAS G12D	Advanced pancreatic cancer	Ranok	I/II	2025-10-24	NCT07303465	China
ABSK 141	KRAS G12D	Advanced pancreatic cancer	Abbisko	I/II	2026-02-18	NCT07417189	China
ASP3082	KRAS G12D	Advanced pancreatic cancer	Astellas	I	2022-05-19	NCT05382559	Global
U87	TROP2	Advanced pancreatic cancer	UniCAR	I	2022-11-04	NCT05605197	China
MT-302	TROP2	Advanced pancreatic cancer	Create	I	2023-08-01	NCT05969041	Global excl. China, US
Zoldonrasib	KRAS G12D	Advanced pancreatic cancer	Revolution	I	2023-09-15	NCT06040541	US
NT-112	KRAS G12D	Advanced pancreatic cancer	Neogene	I	2024-01-23	NCT06218914	US
QTX3034	KRAS G12D, KRAS G12V	Advanced pancreatic cancer	Quanta	I	2024-01-26	NCT06227377	US
AST2169	KRAS G12D	Advanced pancreatic cancer	Allist	I	2024-04-02	CTR20240981	China
QLC1101	KRAS G12D	Advanced pancreatic cancer	Qilu	I	2024-05-08	NCT06403735	China
LY3962673	KRAS G12D	Advanced pancreatic cancer	Lily	I	2024-09-19	NCT06586515	Global
INCB186748	KRAS G12D	Advanced pancreatic cancer	Incyte	I	2025-02-10	NCT06818812	US
ALTA-3263	KRAS G12D, KRAS G12V	Advanced pancreatic cancer	Alterome	I	2025-02-19	NCT06835569	US
BBO-11818	Pan-KRAS	Advanced pancreatic cancer	BridgeBio	I	2025-04-08	NCT06917079	US
DCTY1102	KRAS G12D	Advanced pancreatic cancer	DCTY	I	2025-05-16	NCT07014878	China
KQB548	KRAS G12D	Advanced pancreatic cancer	Kumquat	I	2025-10-06	NCT07207707	US
ZN-F-6418	KRAS G12D	Advanced pancreatic cancer	Zion Pharma	I	2025-11-06	CTR20254150	China
D3S-003	KRAS G12D	Advanced pancreatic cancer	D3 Bio	I	2026-03-06	NCT07456046	-

Note: 1. The competitive landscape only includes pan-(K)RAS inhibitors and KRAS-G12D inhibitors.

Source: clinicaltrials.gov, CDE, CIC

Market opportunity of FAP-targeted ADC in FAP positive solid tumor

Overview of FAP in tumors

Fibroblast activation protein is a type II transmembrane glycoprotein of the prolyl oligopeptidase family, predominantly expressed in CAFs within the TME across various solid tumors, while remaining minimally expressed or absent in most normal adult tissues, highlighting its potential as a selective therapeutic target. Given its restricted expression pattern and central role in tumorigenesis, FAP has become an attractive therapeutic target, with approaches such as ADCs, CAR-T cell therapies, and radiolabeled imaging agents designed to selectively target and eliminate FAP-expressing CAFs, disrupt the supportive tumor stroma and enhance the efficacy of existing treatments.

Mechanism of Action of FAP-Targeted ADCs

FAP-targeted ADCs are designed to selectively deliver cytotoxic agents to CAFs within the TME. They consist of a monoclonal antibody that specifically binds to FAP, a serine protease overexpressed on CAFs, linked to a potent cytotoxic drug. Upon binding to FAP, the ADC is internalized and releases its payload inside the CAFs, resulting in their depletion and disruption of the TME. This targeted approach aims to reduce tumor growth, enhance immune cell infiltration, and improve the efficacy of concurrent therapies by modulating the supportive stromal components of the tumor.

Addressable population of FAP targeted therapies

High levels of FAP expression have been observed in several major solid tumors, including breast cancer, pancreatic cancer, esophageal cancer, and NSCLC. Globally, the addressable population for FAP-targeted therapies was approximately 9.1 million in 2020 and grew to 9.7 million in 2024. In China, the number of patients with FAP-high expressing tumors was estimated at 2.2 million in 2020, increasing to 2.5 million in 2024. From 2024 to 2035, the addressable population is expected to grow to 3.1 million, representing a significant market opportunity for FAP-targeted therapeutics in China.

REPORT COMMISSIONED BY CIC

In connection with the [**REDACTED**], we have engaged CIC, an Independent Third Party, to conduct a detailed analysis and prepare an industry report on the major markets for which our drug candidates are positioned. CIC is a market research and consulting company that provides market research on a variety of industries including healthcare. Except as otherwise noted, all data and forecasts in this section come from the CIC Report. In preparing the report, CIC collected and reviewed publicly available data such as government-derived information, annual reports and industry association statistics, as well as market data collected by conducting interviews with key industry experts and leading industry participants. CIC has exercised due care in collecting and reviewing the information collected. We have agreed to pay CIC a total fee of approximately RMB550,000 for the preparation of the CIC 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 CIC Report. Except for the CIC Report, we did not commission any other industry report in connection with the [**REDACTED**].