2026 ASCO: GFH375 and GFS202A Clinical Data Review

June 2026

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- Study Background
- Summary
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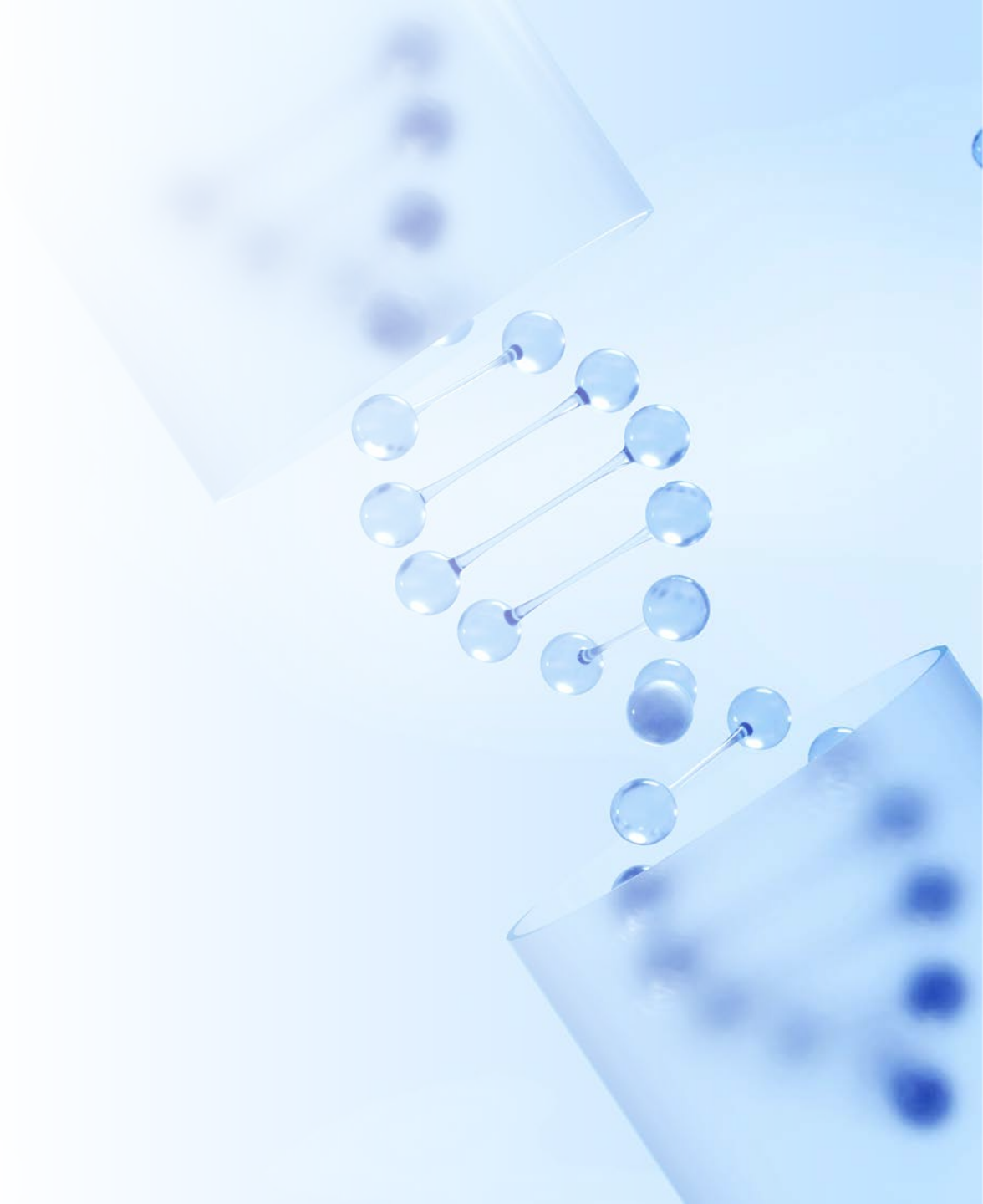
PART 01

Study Background



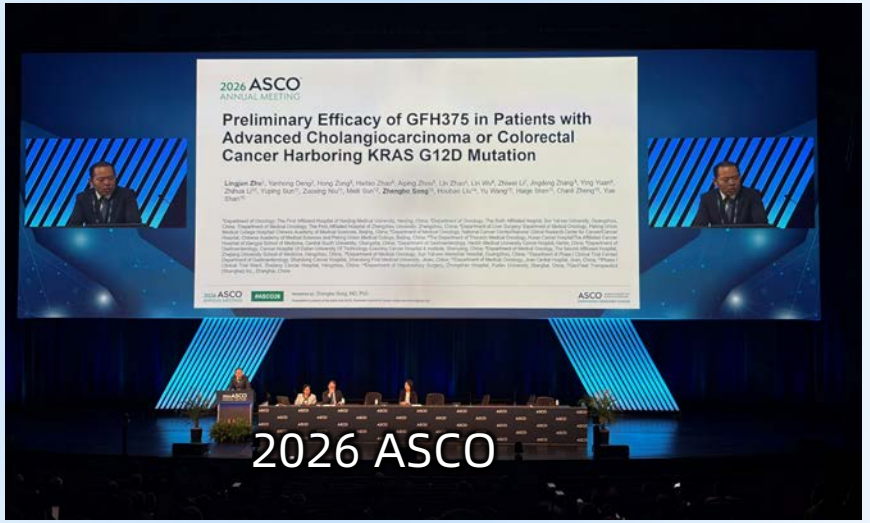
Fourth Consecutive Selection of GFH375's Clinical Data into Oral Presentation at Top-tier Global Academic Meetings

Solid tumors

NSCLC

PDAC

CCA & CRC



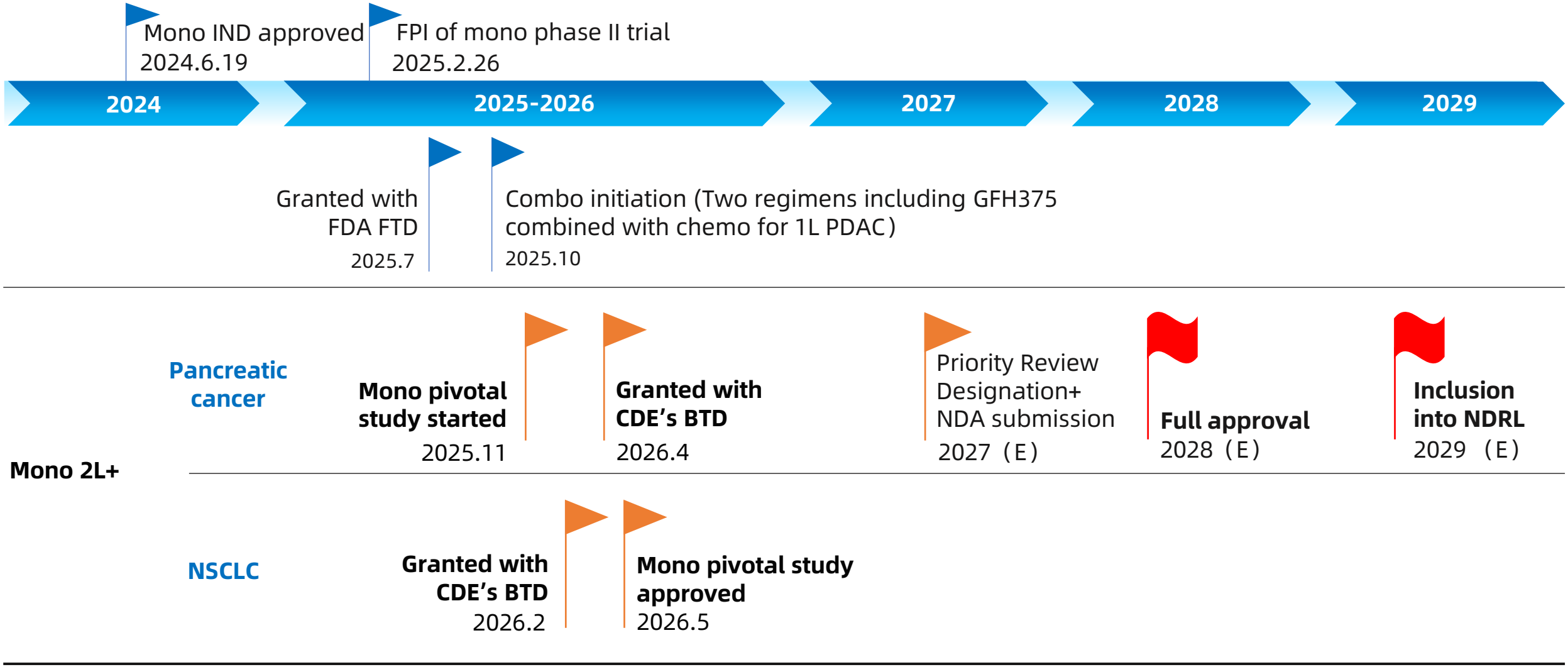
Robust Druggablity Supported by Consistent Safety Profile

GFH375 presented a manageable safety profile in heavily pretreated solid tumor patients harboring KRAS G12D mutations.

	2026 ASCO: CCA 400-600 mg QD (n = 20)	2026 ASCO: CRC 400-750 mg QD (n = 41)	2026 ASCO: Total (n = 61)	2025 WCLC: All cancer types 100-900 mg QD (n=142)
Any TRAE	20 (100)	40 (97.6)	60 (98.4)	139 (97.9)
TRAEs ≥ Grade 3, n(%)	8 (40.0)	11 (26.8)	19 (31.1)	39 (27.5)
Grade 5 TRAE, n(%)	0	1 (2.4)*	1 (1.6)	0
TRAEs leading to discontinuation, n(%)	0	2 (4.9)	2 (3.3)	6 (4.2)
TRAEs leading to dose interruption, n(%)	8 (40.0)	10 (24.4)	18 (29.5)	33 (23.2)
TRAEs leading to dose reduction, n(%)	1 (5.0)	1 (2.4)	2 (3.3)	11 (7.7)
Treatment related SAEs, n(%)	1 (5.0)	4 (9.8)	5 (8.2)	11 (7.7)
Data cut-off date	12 Dec. 2025			17 Jun. 2025

**This grade 5 event was coded as "undetermined death". It occurred in a 65-year-old female CRC patient with liver and lymph nodes metastasis at baseline. The patient passed away at home. Despite all attempts, no relevant information could be obtained as the family declined to provide. Therefore, the causality could not be concluded due to insufficient information available.*

GFH375 Development Features Well-orchestrated Timeline and Powerful Execution; Pancreatic Cancer Trials Enrollment Proceed as Planned

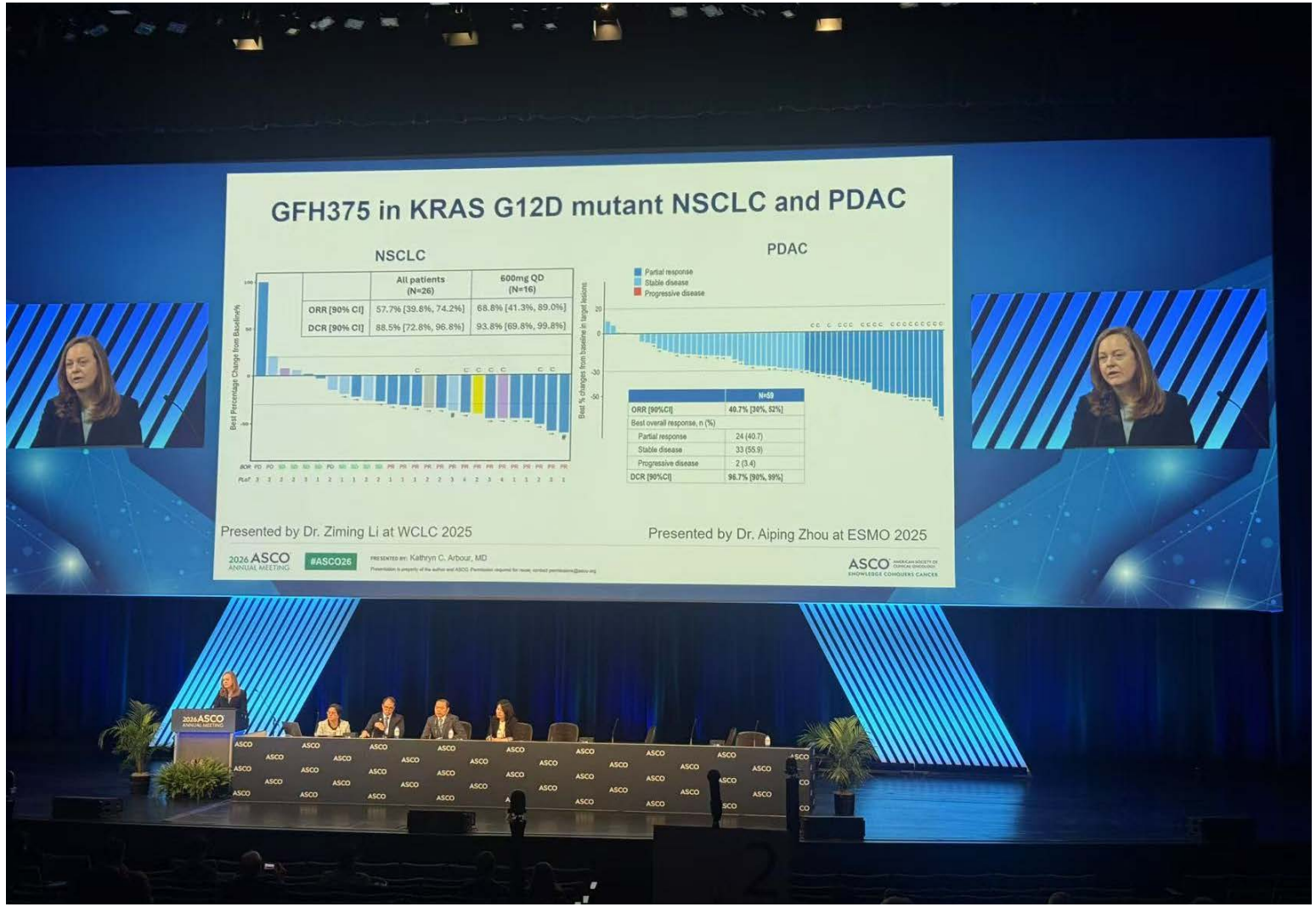


Broad Market Potential upon GFH375's Encouraging Clinical Efficacy

- KRAS G12D is the most frequent KRAS mutation (29%) driving cancer development, accounting for 5.8–10.8% in CCA and 10.6–19.2% in CRC[1~4]
- KRAS G12D mutation is associated with poor prognosis and confers resistance to conventional chemotherapy[3,5,6]
- Critical unmet medical need remains due to suboptimal efficacy of existing treatment:
  - CCA (in 2L): ORR 5–12.5%, mPFS 4.0–4.2 mo, mOS 6.2–8.6[7]
  - CRC (in 3L): ORR 1–6.1%, mPFS 1.9–5.6 mo, mOS 6.4–10.8 mo[8~11]
- GFH375, a highly selective and potent KRAS G12D inhibitor targeting both the "ON" (GTP-bound) and "OFF" (GDP-bound) states, has shown encouraging clinical activity in PDAC and NSCLC[12,13]
- Herein, we report the efficacy and safety data of GFH375 monotherapy in CCA and CRC

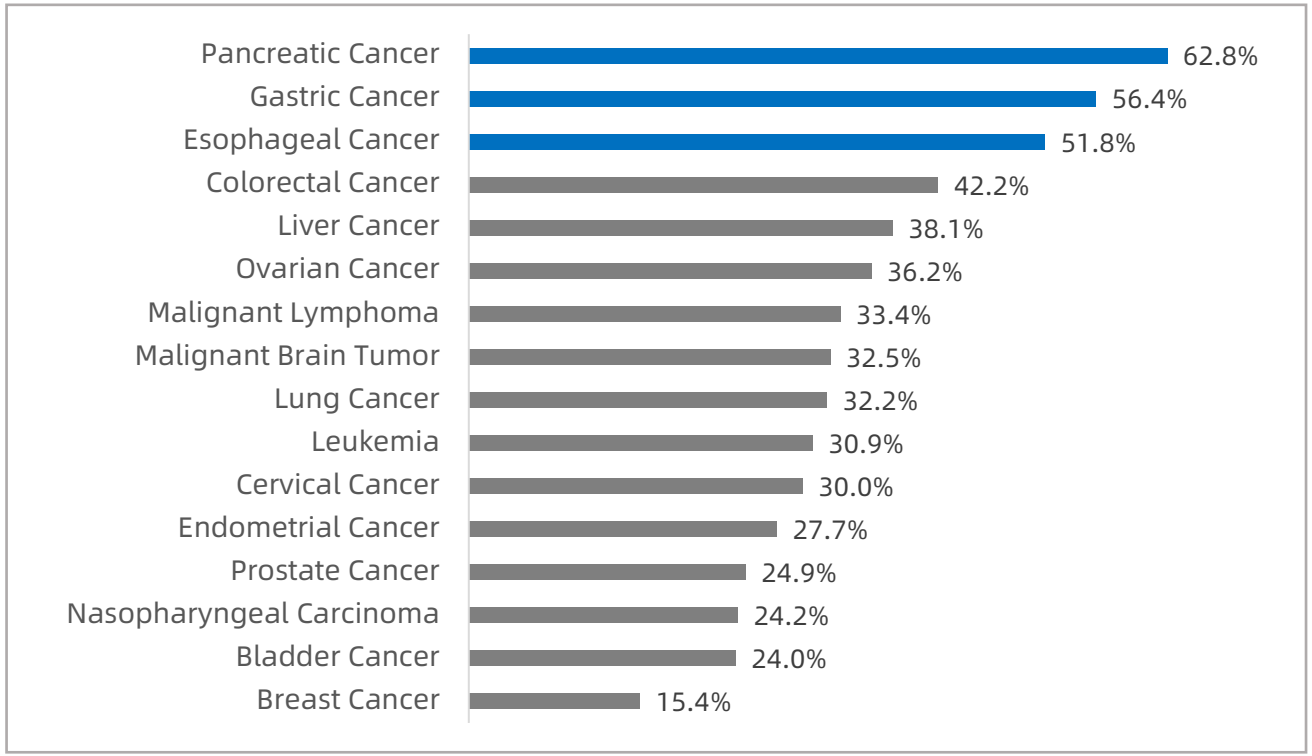
[1]JK Lee et al. npj Precision Oncology (2022) 6:91; 12:924-37. [2]Thongyoo P, et al. Cancer Genomics Proteomics. 2025 Jan-Feb;22(1):112-126. [3]K. Iida, et al. ESMO Open, 2025; 10. [4]Mingjing Meng, et al. Biomedicine & Pharmacotherapy 140 (2021) 111717. [5]Benfeng Xu, et al. Eurasian J Med Oncol. 2025;9(3):122-132. [6]Mitsunobu Takeda, et al. Cancers 2025, 17, 428. [7]Banales, J.M., et al. Nat Rev Gastroenterol Hepatol 23, 65–96 (2026). [8]Grothey, A., et al. Lancet (London, England) 2013, 381(9863), 303–312. [9]Xu, J., et al. Journal of clinical oncology: official journal of the American Society of Clinical Oncology 2018, 36(4), 350–358. [10]Li, J., et al. JAMA 2018, 319(24), 2486–2496. [11]Prager GW, et al; N Engl J Med. 2023 May 4;388(18):1657-1667. [12]Zhou A, et al. Annals of Oncology, 36S1626. [13]Lu S, et al. Journal of Thoracic Oncology, 20, S59-S60.

GFH375's Promising Efficacy across Multiple Malignancies Earns Commentator Recognition at 2026 ASCO

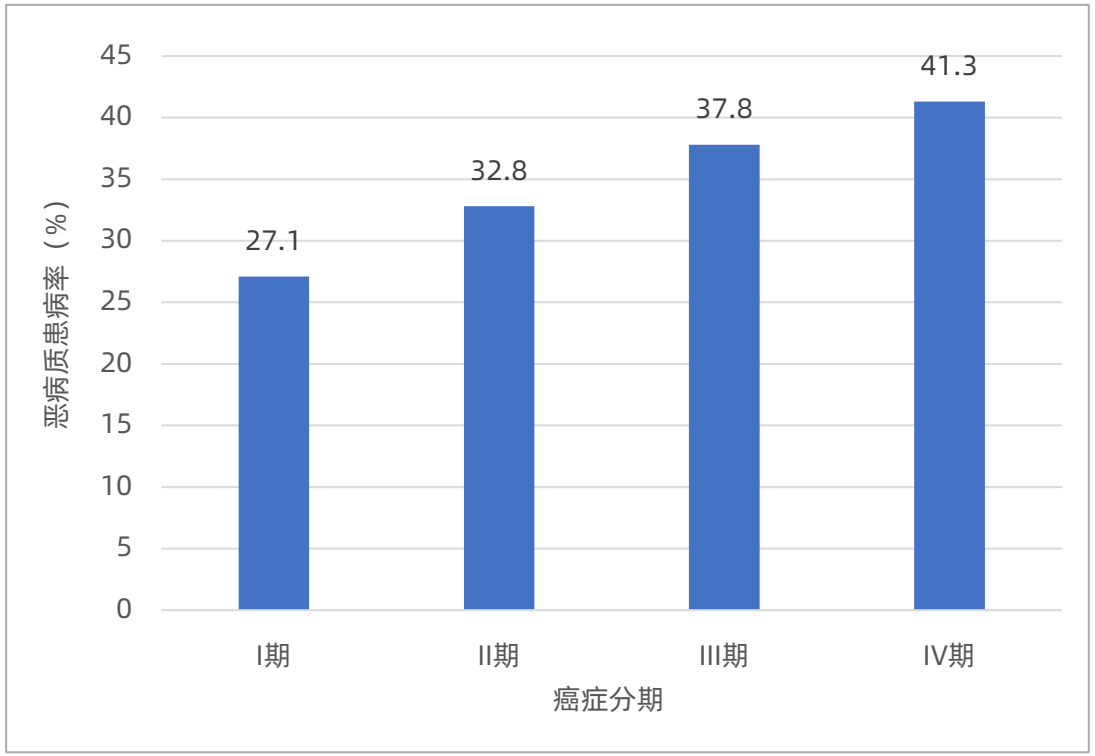


Cachexia and Oncology Treatments Exhibit Strong Potential of Commercial Synergy

- Overall prevalence of cancer cachexia stands at 37.0%, with primary tumor site serving as a key determinant of cachexia occurrence.
- Pancreatic (62.8%), gastric (56.4%) and esophageal cancers (51.8%) demonstrate the strongest correlation with cancer cachexia.
- Lung cancer ranks first in both incidence and mortality among all malignancies in China, with approximately 32.2% of lung cancer patients developing cancer cachexia.



Cachexia incidence across different cancer types

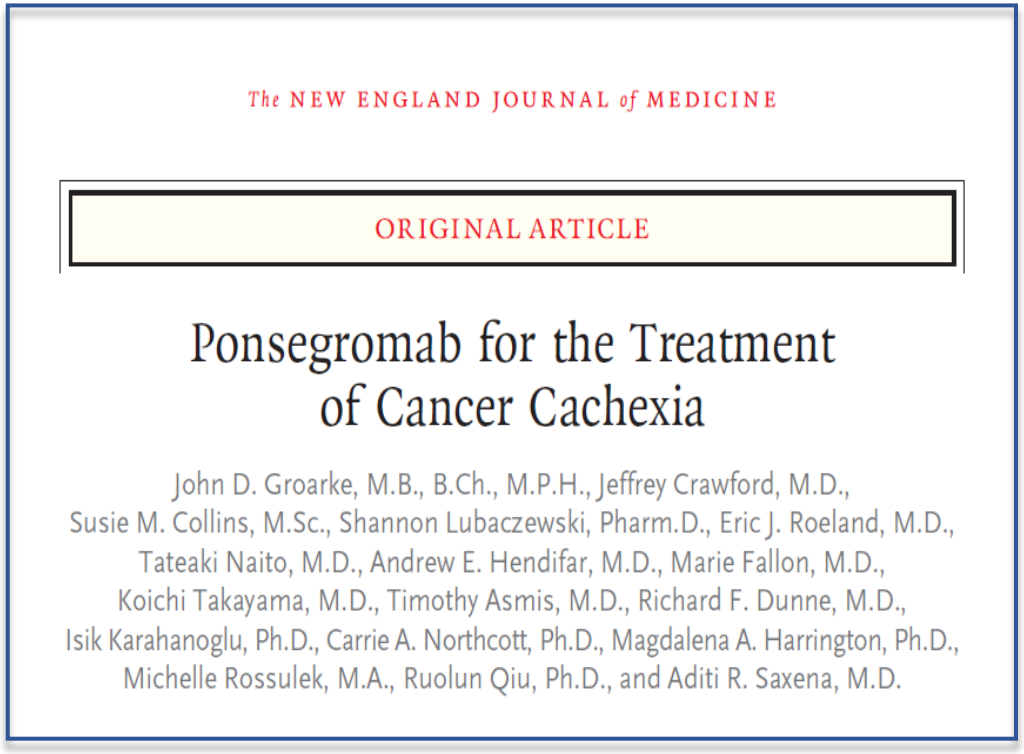


Cachexia incidence across cancer disease stages

i, Xiangrui, et al.. Precision Nutrition 1(1):10.1097/PN9.0000000000000008, June 2022. 2. Han B, et al. 2024;4(1):47-53. 2024 Feb 2.

Ponsegromab (Pfizer's anti-GDF15 mAb) Delivers Meaningful Weight Gain in Patients with Cancer Cachexia

- At Week 12, all active-dose cohorts demonstrated dose-dependent body weight gains versus placebo.



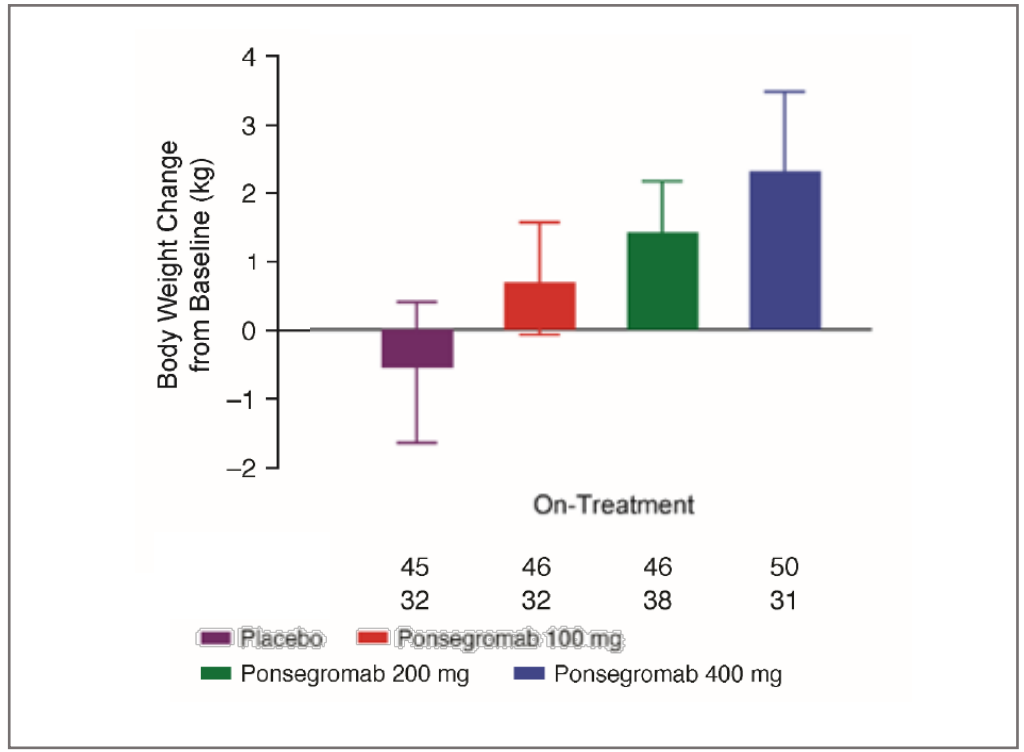
The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Ponsegromab for the Treatment of Cancer Cachexia

John D. Groarke, M.B., B.Ch., M.P.H., Jeffrey Crawford, M.D., Susie M. Collins, M.Sc., Shannon Lubaczewski, Pharm.D., Eric J. Roeland, M.D., Tateaki Naito, M.D., Andrew E. Hendifar, M.D., Marie Fallon, M.D., Koichi Takayama, M.D., Timothy Asmis, M.D., Richard F. Dunne, M.D., Isik Karahanoglu, Ph.D., Carrie A. Northcott, Ph.D., Magdalena A. Harrington, Ph.D., Michelle Rossulek, M.A., Ruolun Qiu, Ph.D., and Aditi R. Saxena, M.D.

Groarke, John D et al. The New England journal of medicine vol. 391,24 (2024): 2291-2303.



Body weight change from baseline

PROACC study, phase II

GenFleet's Presentations at 2026 ASCO

GFH375

Oral presentation

Preliminary Efficacy of GFH375 in Patients with Advanced Cholangiocarcinoma or Colorectal Cancer Harboring KRAS G12D Mutation

Lingjun Zhu, *Yanhong Deng, Hong Zong, Haitao Zhao, Aiping Zhou, Lin Zhao, Lin Wu, Zhiwei Li, Jingdong Zhang, Ying Yuan, Zhihua Li, Yuping Sun, Zuoxing Niu, Meili Sun,* ***Zhengbo Song,*** *Houbao Liu, Yu Wang, Haige Shen, Chanli Zheng, Yue Shan*

GFS202A

Poster presentation

A first-in-human (FiH) phase I study of GFS202A, a GDF15/IL-6 bispecific antibody, in advanced cancer patients with pre-cachexia or cachexia

Hongyun Zhao, *Da Li, Yuxiang Ma, Hong Zong, Yusheng Wang, Xiangcai Wang, Haiyu Yang, Zhen Li, Qian Chu, Yue Shan, Lingyu Tai, Jiani Song, Yue Zhang, Huaqiang Zhu, Haige Shen8, Yu Wang,* ***Li Zhang***

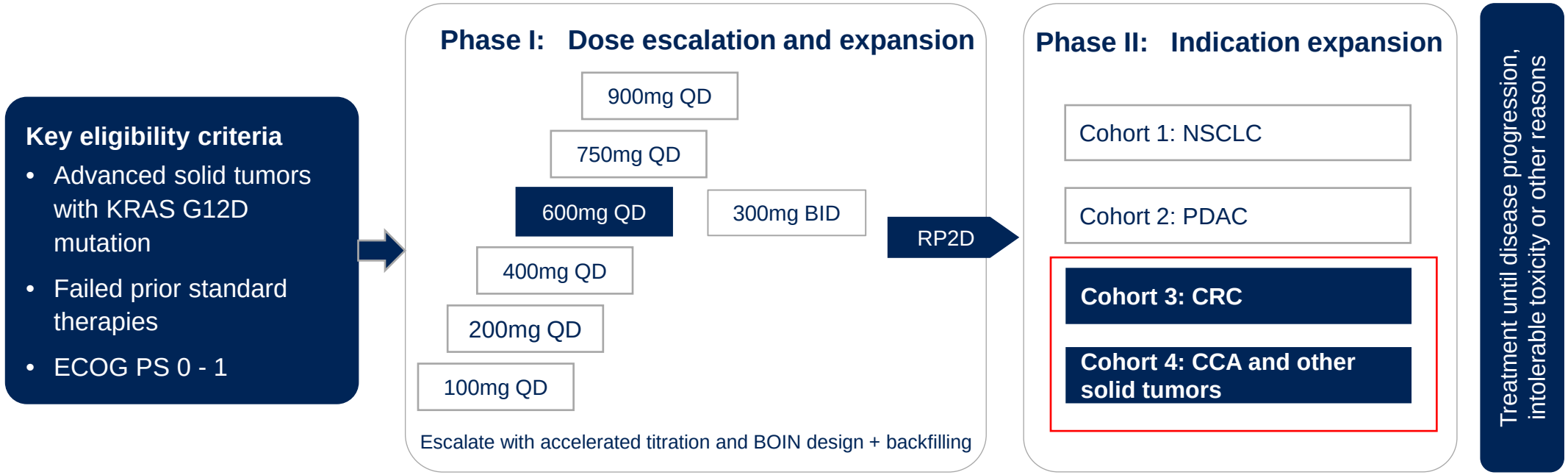
PART 02

Data Review -- GFH375

Key Takeaway Points

- GFH375 monotherapy demonstrated promising clinical activity in heavily pretreated patients with KRAS G12D-mutant CCA and CRC.
  - **CCA** (in 3L+: 75%)**: ORR 40%**, DCR 95%, **mPFS 6.2 mo** and **mOS not reached**
  - **CRC** (in 4L+: 61%)**: ORR 11%,** DCR 77%, **mPFS 4.1 mo** and **mOS 10.3 mo**
- GFH375 exhibits a manageable and consistent safety profile in the previously heavily treated population without new safety signals.
- The preliminary clinical data supports further development of GFH375 monotherapy and in combination regimens for patients with KRAS G12D–mutant CCA and CRC.

Phase I/II Study of GFH375 in Advanced Solid Tumors with KRAS G12D Mutation (NCT06500676)



Phase I endpoints:

- Safety/tolerability and MTD/RP2D: AEs, etc.
- Anti-tumor activity: ORR, DOR, DCR, PFS (per RECIST V1.1) and OS
- Pharmacokinetics
- Biomarkers

Phase II endpoints:

- Efficacy: ORR, DOR, DCR, PFS (per RECIST V1.1) and OS
- Safety: AEs, etc.
- Biomarkers

Abbreviations: AE, adverse event; BID, twice daily; BOIN, Bayesian optimal interval; CCA, cholangiocarcinoma; CRC, colorectal cancer; DCR, disease control rate; DOR, duration of response; ECOG PS, eastern cooperative oncology group performance status; MTD, maximum tolerated dose; NSCLC, non-small cell lung cancer; ORR, overall response; OS, overall survival; PDAC, pancreatic ductal adenocarcinoma; PFS, progression of survival; QD, once daily; RECIST, Response Evaluation Criteria in Solid Tumors; RP2D, recommended phase 2 dose.

Patient Baseline Characteristics

CCA, 400 mg (n=2), 600 mg (n=18) **:**

- 70% were intrahepatic cholangiocarcinoma and 30% were extrahepatic cholangiocarcinoma at diagnosis
- The majority (75%) received at least 2 prior lines of therapies
- 65% previously received TKIs

CRC, 400 mg (n=5), 600 mg (n=33), 750 mg (n=3) **:**

- 61% received at least 3 prior lines of therapies
- 39% previously received TKIs

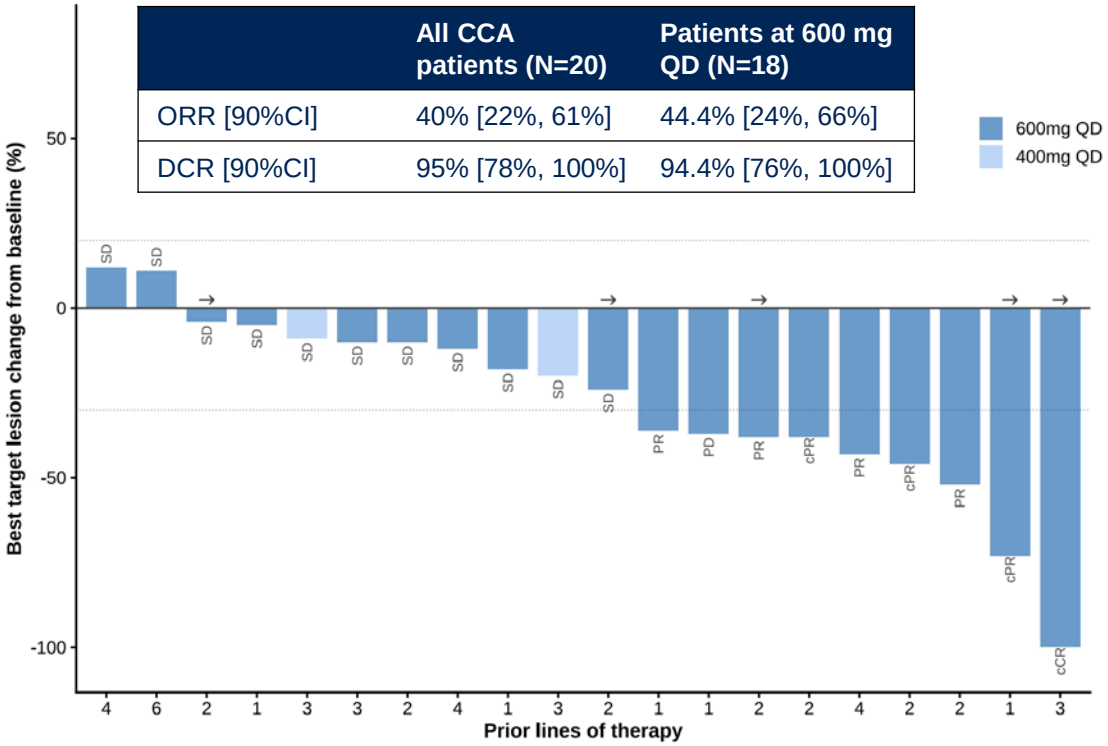
Characteristics	CCA (n=20)	CRC (n=41)
Age, median (range), years	59.5 (41, 74)	56 (29, 71)
Male, n(%)	16 (80.0)	25 (61.0)
ECOG PS, n(%)		
0/1	2 (10.0)/18 (90.0)	5 (12.2)/36 (87.8)
Metastasis at baseline [stage IV], n(%)	17 (85.0)	41 (100)
Liver	14 (70.0)	23 (56.1)
Lung	8 (40.0)	34 (82.9)
Peritoneum	4 (20.0)	12 (29.3)
Bone	4 (20.0)	7 (17.1)
Number of prior anti-cancer therapies, n(%)		
1	5 (25.0)	2 (4.9)
2	7 (35.0)	14 (34.1)
≥ 3	8 (40.0)	25 (61.0)
Median (range)	2 (1, 6)	3 (1, 6)
Prior therapies, n(%)		
Fluoropyrimidine/capecitabine	13 (65.0)	41 (100)
Oxaliplatin or other platinum	18 (90.0)	41 (100)
Irinotecan	4 (20.0)	40 (97.6)
Gemcitabine	18 (90.0)	0
Anti-PD1/PD-L1	17 (85.0)	15 (36.6)
Anti-angiogenic biologic	1 (5.0)	41 (100) *
Tyrosine kinase inhibitors	13 (65.0%) #	16 (39.0%)&

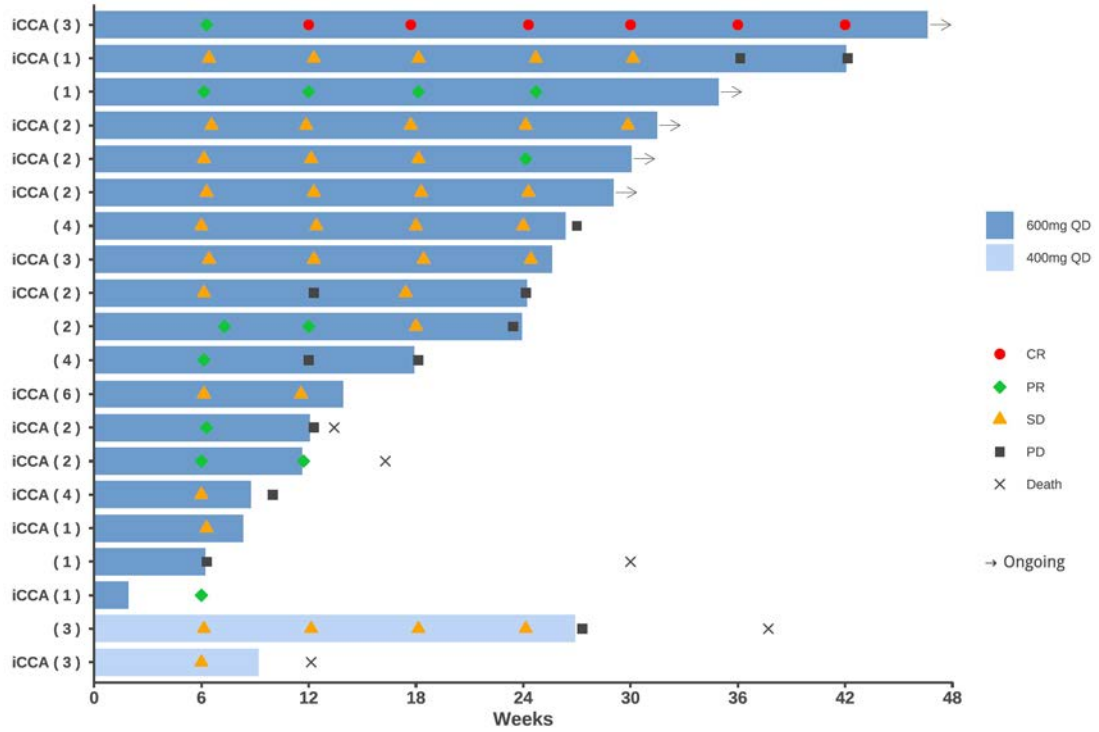
Enrollment cut-off date: 31-Oct-2025.
* Included Bevacizumab (n=41), Ivonescimab (n=1), LM299 (n=1). # Included Lenvatinib (n=9), Anlotinib (n=4), Surufatinib, Afatinib, Entrectinib and Donafenib (n=1 each). & Included Fruquintinib (n=15), Regorafenib (n=7).

Efficacy in CCA

- All CCA patients had at least one post-treatment tumor evaluation. The minimum follow-up time was 5.7 months.
- The ORR was 40% (8/20); confirmed ORR was 20% (4/20); 1 was ongoing for confirmation.

	All CCA patients (N=20)	Patients at 600 mg QD (N=18)
ORR [90%CI]	40% [22%, 61%]	44.4% [24%, 66%]
DCR [90%CI]	95% [78%, 100%]	94.4% [76%, 100%]



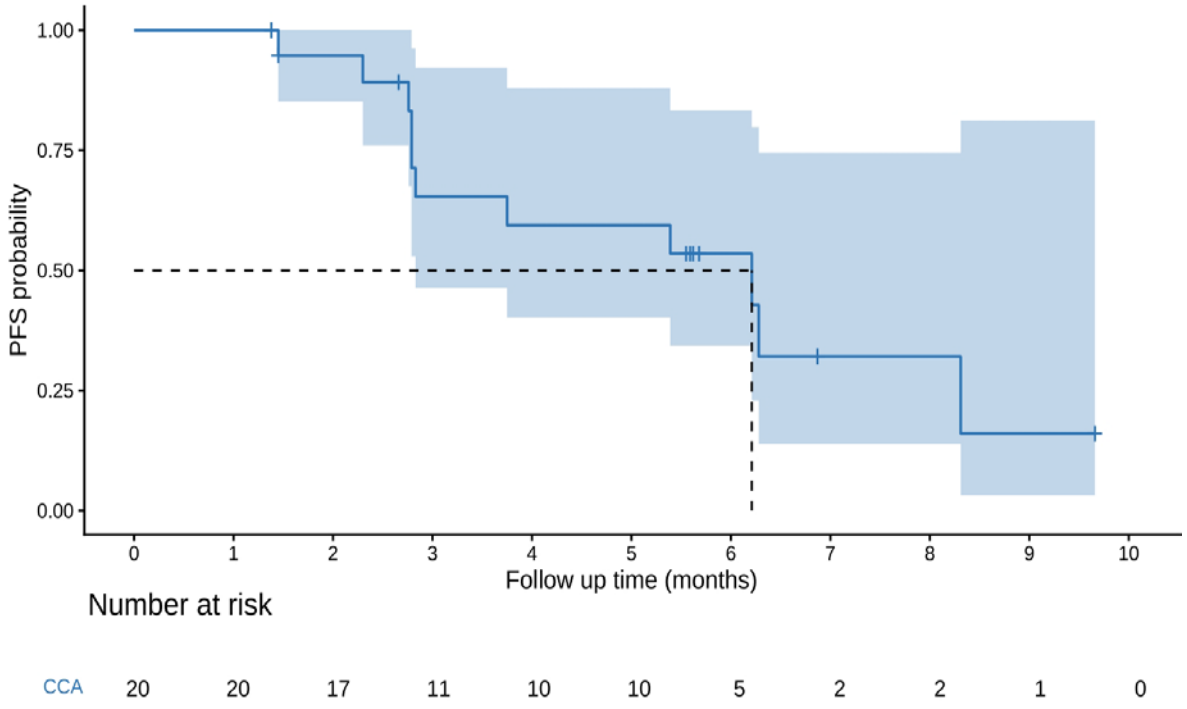


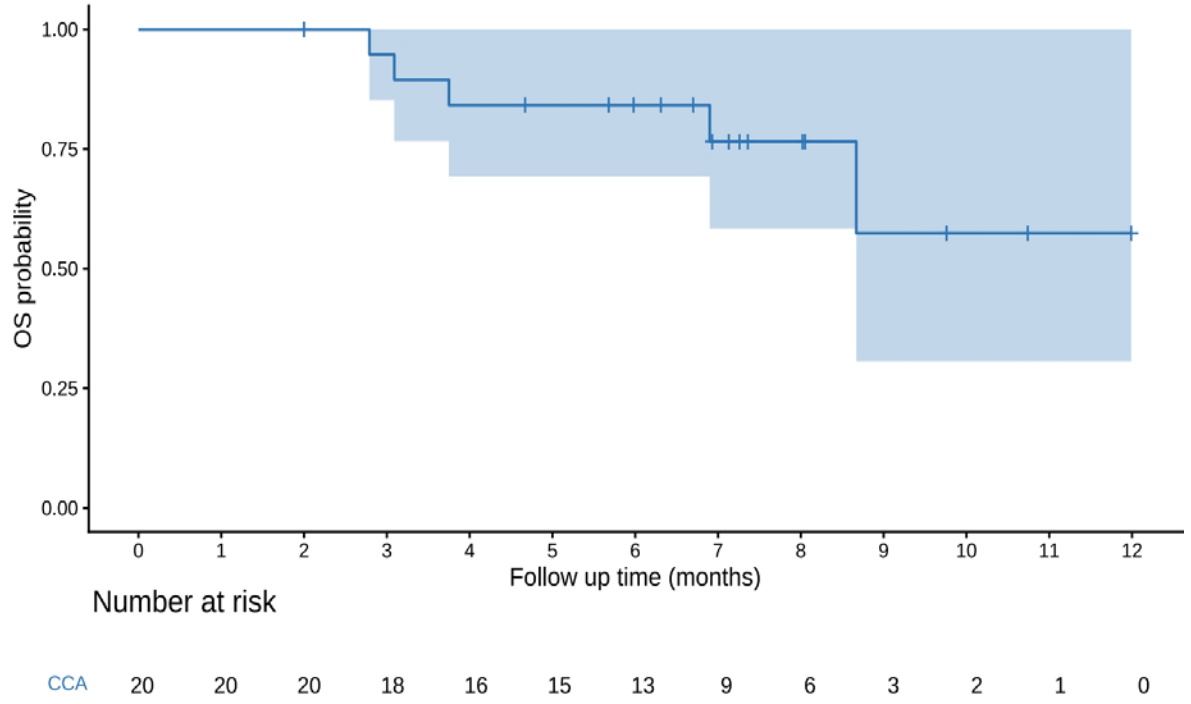
Data cut-off date: 20-Apr-2026.
The number in parentheses on the left side of y-axis in the swimming plot refers to the number of prior lines of therapies.
Abbreviations: cCR, confirmed complete response; cPR, confirmed partial response; DCR, disease control rate; iCCA, intrahepatic cholangiocarcinoma; ORR, overall response; PD, progressive disease; PR, partial response; QD, once daily; SD, stable disease.

PFS and OS in CCA

In heavily pretreated CCA patients (75% in 3L+ setting):

- **Median PFS** was **6.2 months** with median follow-up time of 5.7 months.
- **Median OS not reached** with median follow-up time of 7.3 months.





Data cut-off date: 20-Apr-2026.

Case Report: Patient with KRAS G12D Mutant CCA

Baseline Characteristics

- 69-year-old male
- Initially diagnosed with KRAS G12D intrahepatic cholangiocarcinoma on 07-Oct-2024
- Stage IV (T1N1M1) at baseline, liver, lung and peritoneum metastasis
- Concurrent TP53, APC and MSH6 mutations

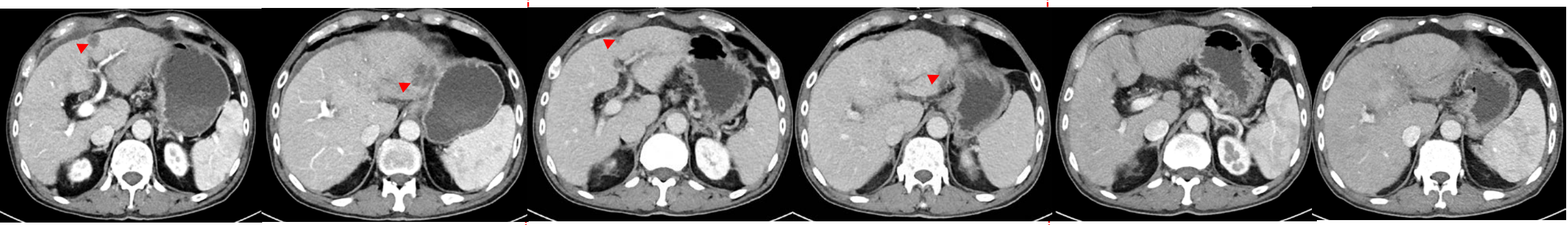
Treatment History

- 1st line: GEMOX, 6 cycles, 2024.10- 2025.1
- 2nd line: FOLFIRI, 3 cycles, 2025.2- 2025.3
- 3rd line: Lenvatinib + Cadonilimab, 2025.4- 2025.4
- Disease progressed 2025.5

GFH375 Treatment Courses

- C1D1: 29-May-2025, GFH375 600 mg QD treatment ongoing as of the data cutoff date
- TRAE: G3 neutrophil count decreased, G3 white blood cell count decreased, G1 platelet count decreased and G1 asthenia

Baseline (May 23, 2025) | **Week 7: PR (July 11, 2025)** | **Week 12: CR (August 20, 2025)**

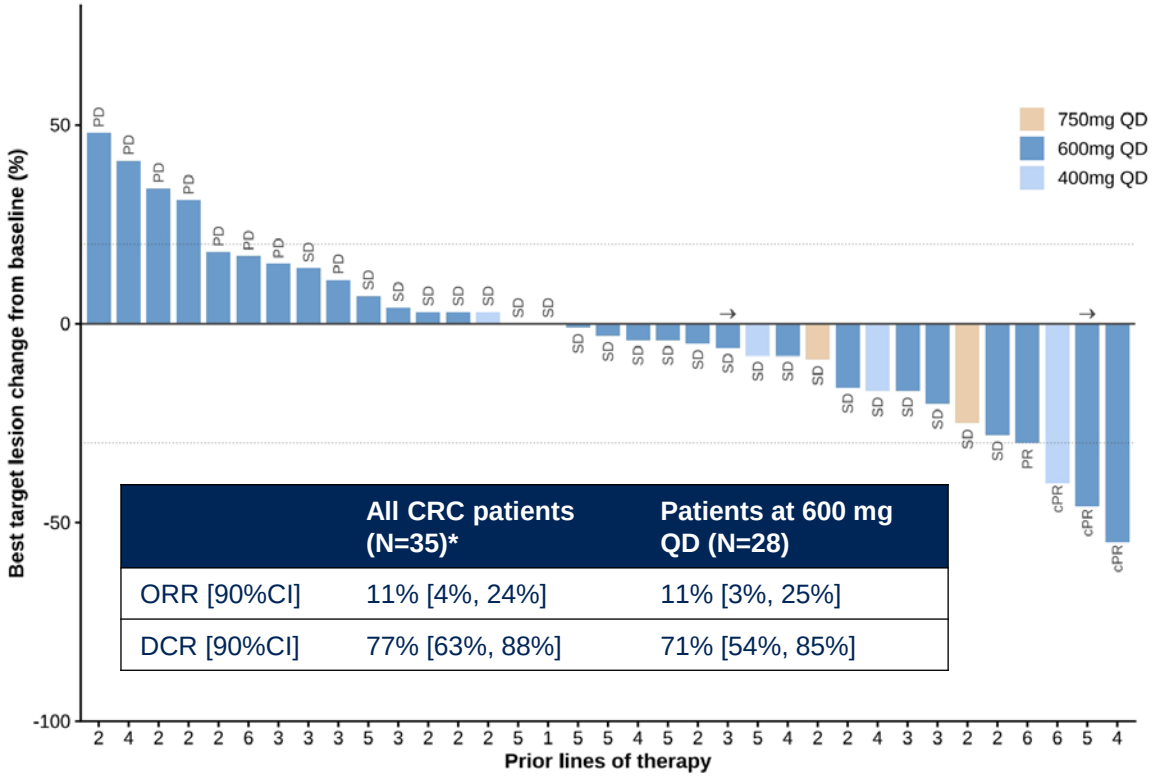


Target lesions with SoD of 66 mm | Target lesions with SoD of 25 mm | Target lesions with SoD of 0 mm

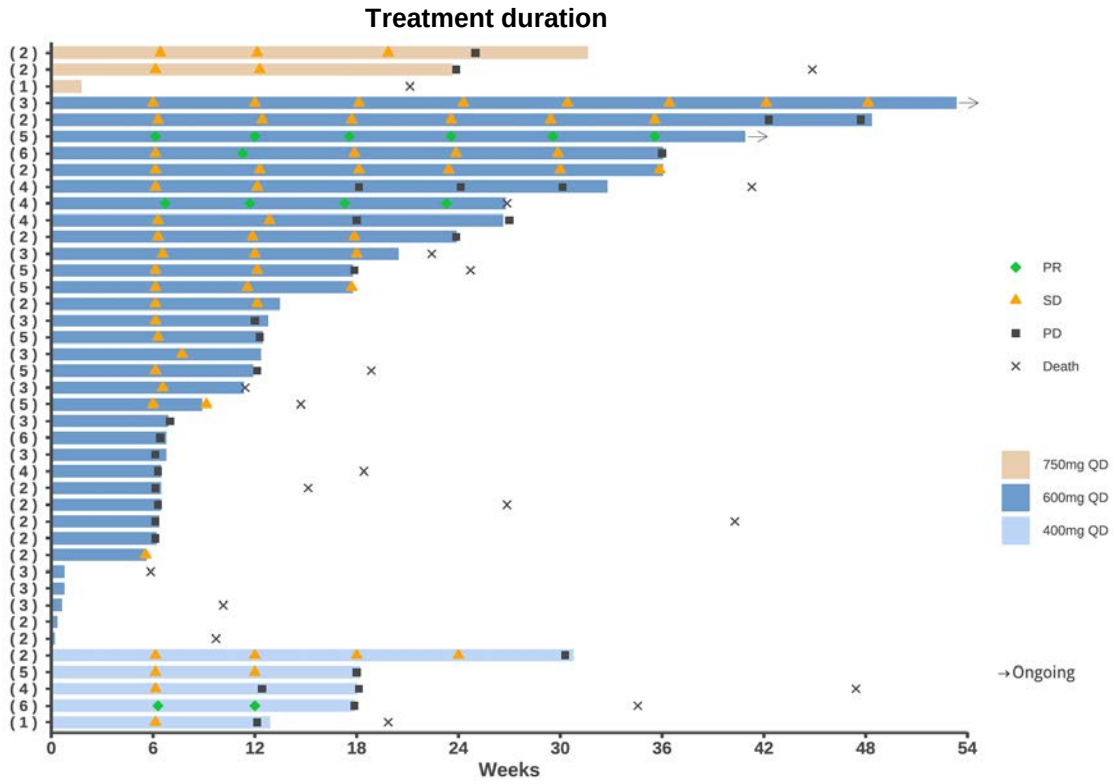
Abbreviations: CR, complete response; FOLFIRI, Irinotecan + Fluoropyrimidine + Leucovorin; GEMOX, Gemcitabine + Oxaliplatin; PR, partial response; QD, once daily; SoD, sum of diameters; TRAE, treatment related adverse event.

Efficacy in CRC

- 35 CRC patients had at least one post-treatment tumor evaluation. The minimum follow-up time was 8.4 months.
- The ORR was 11% (4/35), confirmed ORR was 9% (3/35).



	All CRC patients (N=35)*	Patients at 600 mg QD (N=28)
ORR [90%CI]	11% [4%, 24%]	11% [3%, 25%]
DCR [90%CI]	77% [63%, 88%]	71% [54%, 85%]

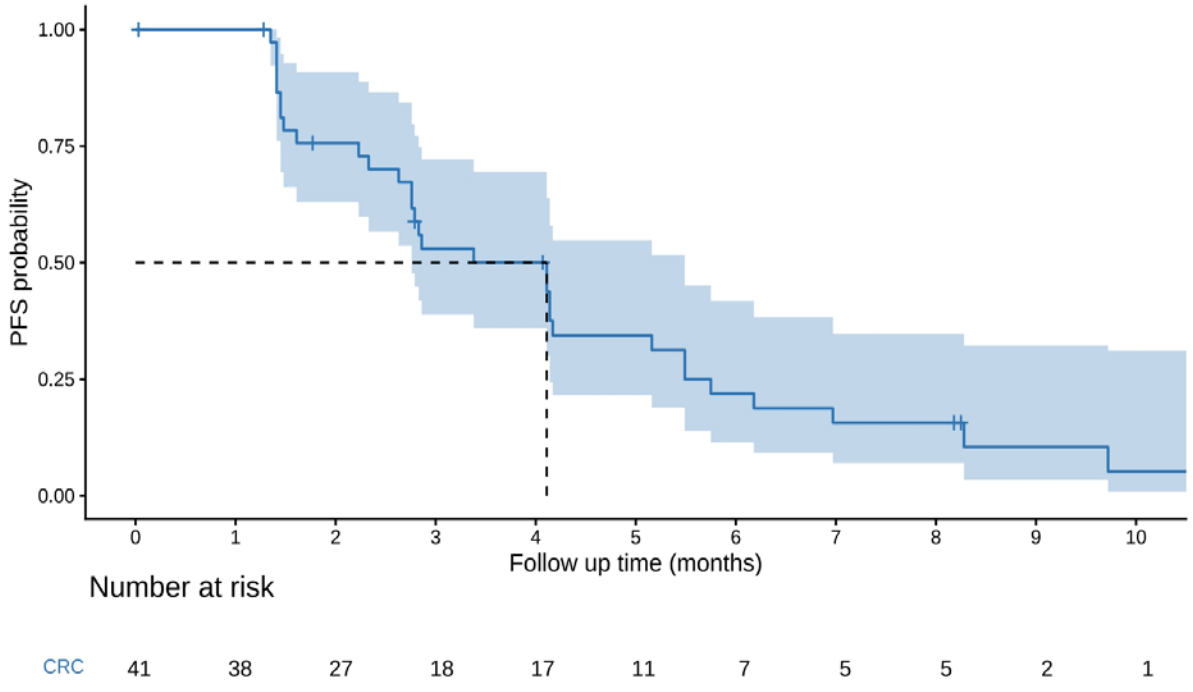


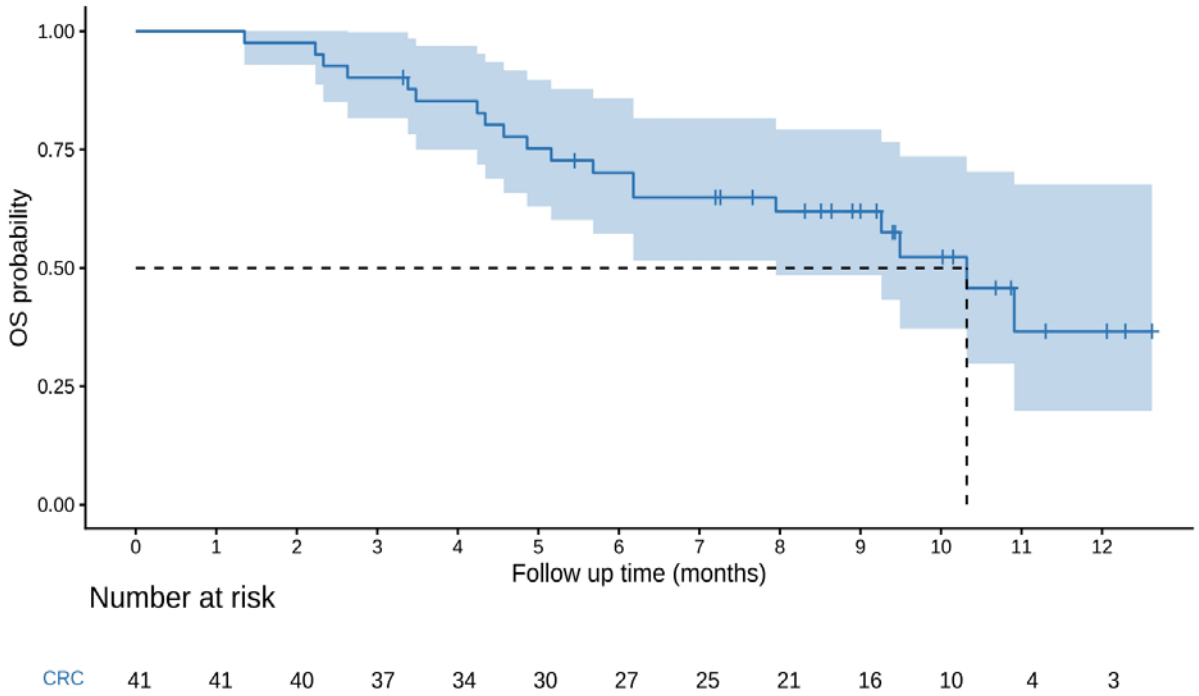
Data cut-off date: 20-Apr-2026.
The number in parentheses on the left side of y-axis in the plot of treatment duration is the number of prior lines of therapies.
* Six patients dropped out early without post-baseline tumor assessments, four due to patient's decision and 2 due to death.

PFS and OS in CRC

In heavily pretreated CRC patients (61% in 4L+ setting):

- **Median PFS was 4.1 months** with median follow-up time of 8.3 months.
- **Median OS was 10.3 months** with median follow-up time of 9.4 months.





Data cut-off date: 20-Apr-2026.

Treatment Related Adverse Events (TRAEs)

- GFH375 presented a manageable safety profile in heavily pretreated CCA and CRC patients.

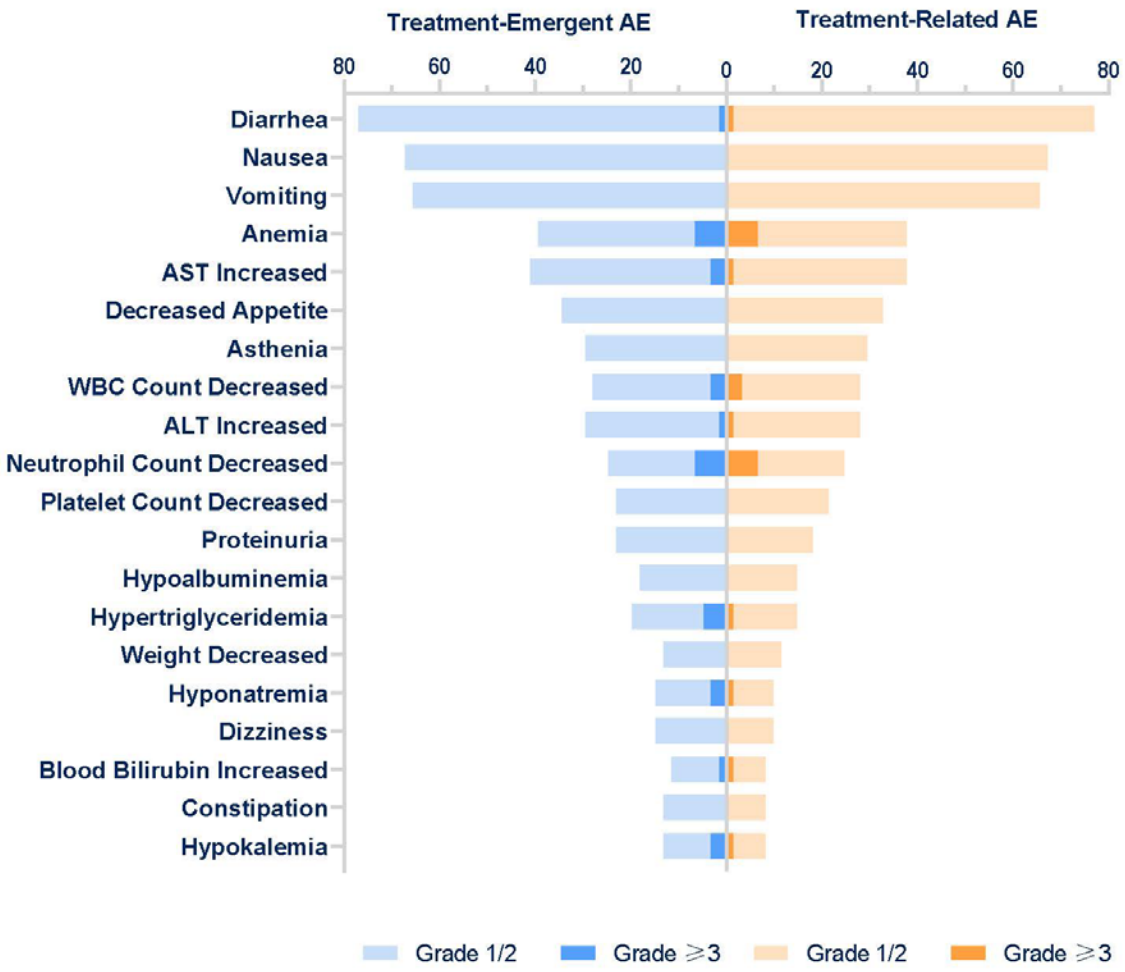
	CCA (n = 20)	CRC (n = 41)	Total (n = 61)
Any TRAE	20 (100)	40 (97.6)	60 (98.4)
TRAEs ≥ Grade 3, n(%)	8 (40.0)	11 (26.8)	19 (31.1)
Grade 5 TRAE, n(%)	0	1 (2.4)*	1 (1.6)
TRAEs leading to treatment discontinuation, n(%)	0	2 (4.9)	2 (3.3)
TRAEs leading to dose reduction, n(%)	1 (5.0)	1 (2.4)	2 (3.3)
TRAEs leading to dose interruption, n(%)	8 (40.0)	10 (24.4)	18 (29.5)
Treatment related SAEs, n(%)	1 (5.0)	4 (9.8)	5 (8.2)

Data cut-off date: 12-Dec-2025.
The median exposure time was 2.7 (range: 0.5-7.6) months among CCA patients and 3.0 (range: 0.1-8.0) months among CRC patients, 2.9 (range: 0.1-8.0) months among all patients. The mean relative dose intensity was 98.5%.
* This grade 5 event was coded as “undetermined death”. It occurred in a 65-year-old female CRC patient with liver and lymph nodes metastasis at baseline. The patient passed away at home. Despite all attempts, no relevant information could be obtained as the family declined to provide. Therefore, the causality could not be concluded due to insufficient information available.
Abbreviations: CCA, cholangiocarcinoma; CRC, colorectal cancer; SAE, serious adverse event; TRAE, treatment related adverse event.

Adverse Events in ≥10% of Patients

- The safety profile in CCA and CRC was consistent with prior reports[1,2,3], without new safety signals observation.
- Common TRAEs were gastrointestinal, hematological AEs and transaminitis; most were grade 1 or 2 and recovered with supportive treatment.

Most frequent TEAE/TRAEs (≥10% of all CCA and CRC patients)



Data cut-off date: 12-Dec-2025.

[1] Ai X, et al. J Clin Oncol 43, 3013-3013(2025). [2] Lu S, et al. Journal of Thoracic Oncology, 20, S59-S60. [3] Zhou A, et al. Annals of Oncology, 36S1626.

Conclusions

- GFH375 single agent showed promising clinical activities in previously heavily treated CCA and CRC.
  - CCA (75% in 3L+ setting) : **ORR 40%,** DCR 95%, **median PFS 6.2 months** and **median OS not reached**
  - CRC (61% in 4L+ setting): **ORR 11%,** DCR 77%, **median PFS 4.1 months** and **median OS 10.3 months**
- The safety profile was tolerable and manageable without new safety signals.
- The preliminary clinical data supports further development of GFH375 monotherapy and in combination regimens for KRAS G12D–mutant CCA and CRC.
- GFH375 plus cetuximab or chemotherapy are currently being studied in solid tumors including CRC (NCT07259590).

PART 03

Data Review -- GFS202A

Study Plan for GFS202A：World's First Bispecific Antibody for Cachexia

GFS202A, 600 mg, IV, Q3W
N = 3 - 6

GFS202A, 400 mg, IV, Q3W
N = 3 - 6

GFS202A, 200 mg, IV, Q3W
N = 3 - 6

GFS202A, 100 mg, IV, Q3W
N = 3 - 6

GFS202A, 25 mg, IV, Q3W
N = 3 - 6

GFS202A, 5 mg, IV, Q3W
N = 1

Primary Endpoints

- Incidence and severity of AEs and SAEs
- Incidence of DLT

Secondary Endpoints

- Pharmacokinetics
- GDF15 and CRP
- Body weight and L3SMI
- Appetites

Pre-cachexia and Cachexia Patients: Baseline Characteristics and Demographics

GENFLEET THERAPEUTICS

➢ As of March 11, 2026, a total of 19 patients received GFS202A (5-400 Q3W)

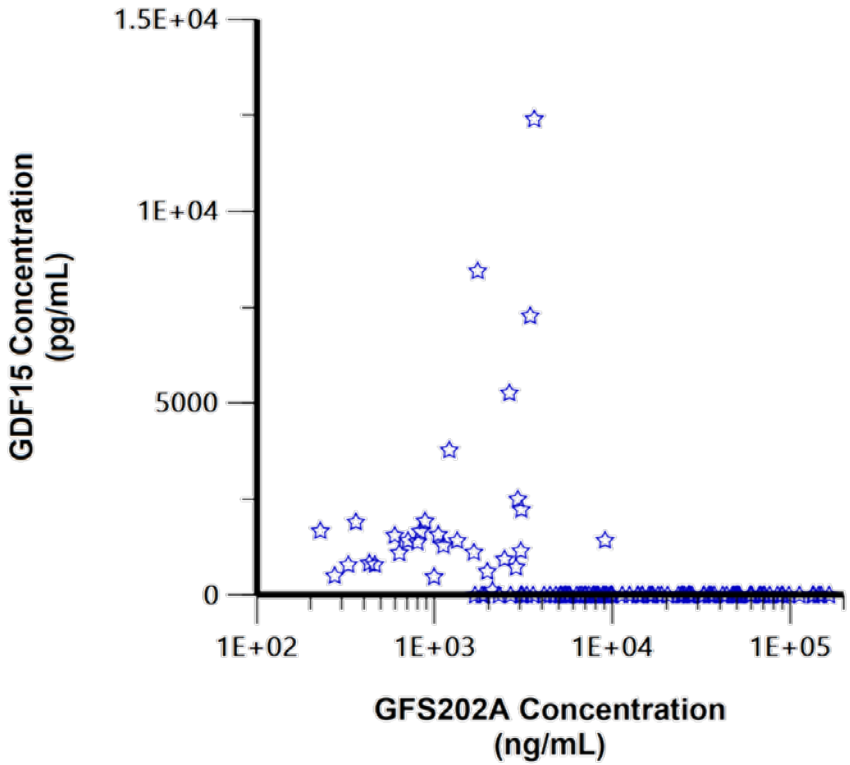
- 5 mg [N = 1];
- 25 mg [N = 4];
- 100 mg [N = 4];
- 200 mg [N = 4];
- 400 mg [N = 6]

➢ Baseline characters:

- Median weight: 52.9 kg.
- Baseline ECOG PS ≥ 1: 16 (84.2%) patients.
- Median GDF15 level: 968.58 pg/mL.
- 18 (94.7%) patients had stage IV disease at study entry. 15 (78.9%) had received ≥ 2 prior lines of therapy.
- During the study, 13 (68.4%) patients received antitumor therapy, including 5 (26.3%) with platinum-based regimens.

	Total (N=19)
Age (years), median (range)	62 (43, 76)
Male, n (%)	14 (73.7)
Weight (kg), median (range)	52.9 (37, 71)
% weight loss during 6 months before screening[a] (%)	
Median (range)	11.4 (2.5, 19)
BMI (kg/m²), median (range)	18.5 (13.9, 27.2)
< 21, n (%)	13 (68.4)
ECOG PS, 0/1/2	3 (15.8)/14 (73.7)/2 (10.5)
GDF15 (pg/mL), median (range)[b]	968.58 (99.63, 1898.57)
Modified Glasgow performance score (mGPS)[c]	
0/1/2	8 (42.1)/8 (42.1)/3 (15.8)
Cancer type, n (%)	
NSCLC	7 (36.8)
PC	2 (10.5)
CRC	4 (21.1)
GC/GEJC	3 (15.8)
Others[d]	3 (15.8)
Stage IV at study entry, n (%)	18 (94.7)
Prior lines of antitumor therapy, median (range)	2 (0, 7)
1, n (%)	3 (15.8)
≥ 2, n (%)	15 (78.9)
Receipt of antitumor therapy during the study, n (%)	
Any	13 (68.4)
Platinum-based	5 (26.3)

PK/PD: Dose-dependent Suppression of GDF15



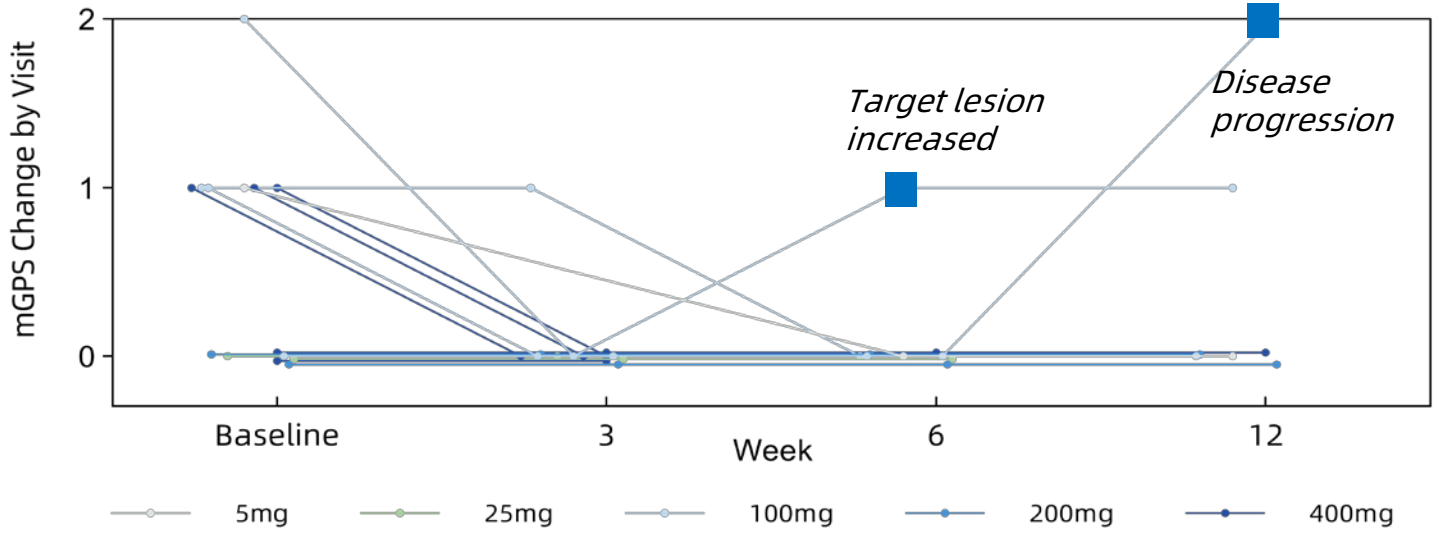
- PK exposure increased with escalating dose levels with a 3.2-7.5 days $T_{1/2}$.
- No obvious accumulation.
- About 30% positive ADA tested without compromising PK exposure and safety.
- Complete and constant inhibition of GDF15 achieved at 200 mg Q3W dose level and above.

mGPS Improvements Observed for Majority of Patients: Notable Decrease in CRP, and Increase in Albumin

➢ mGPS: modified Glasgow Prognostic Score

Crucial inflammation-based prognostic index that predicts survival and clinical outcome

➢ Baseline to week 12 mGPS change by visit: most patients had improved mGPS after treatment with GFS202A

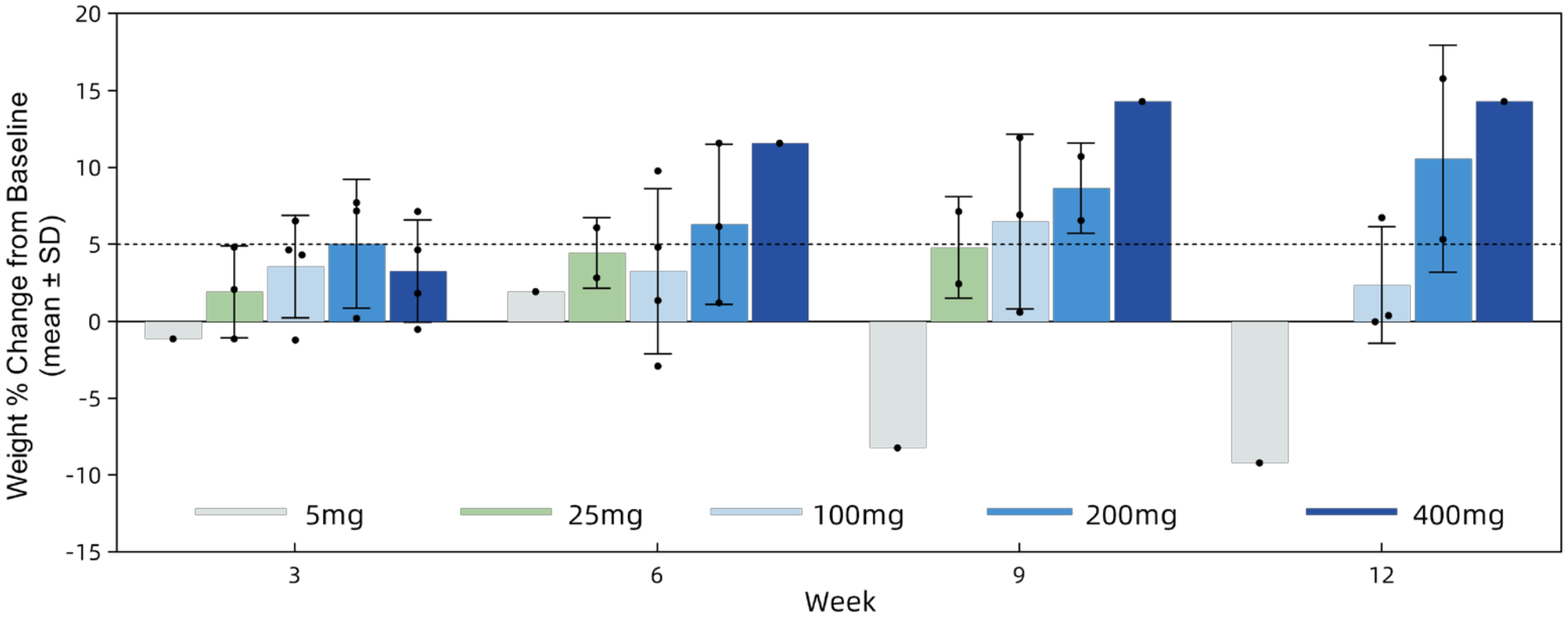


	Week 6	Week 12
Mean decrease of C-reactive protein	39.81 mg/L	49.42 mg/L
Mean increase of albumin	3.4 g/L	2.8 g/L

➢ mGPS rating (calculated based on CRP and albumin)

- mGPS= 2: CRP≥10mg/L & ALB<35g/L
- mGPS= 1: CRP≥10mg/L & ALB≥35g/L,
- mGPS= 0: CRP<10mg/L & any ALB

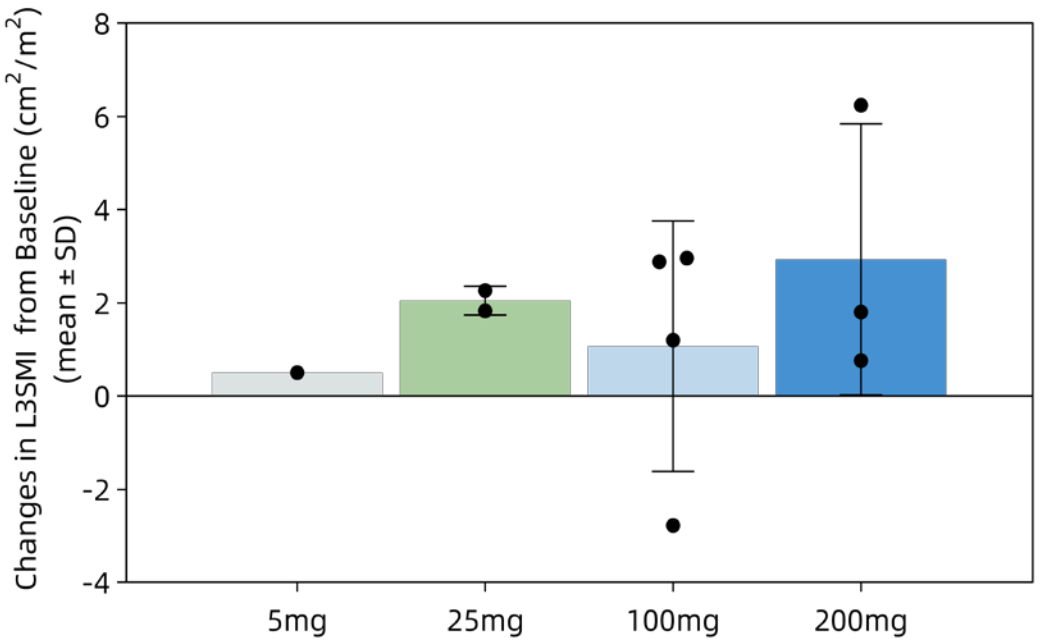
Dose-dependent Increase Observed in Body Weight



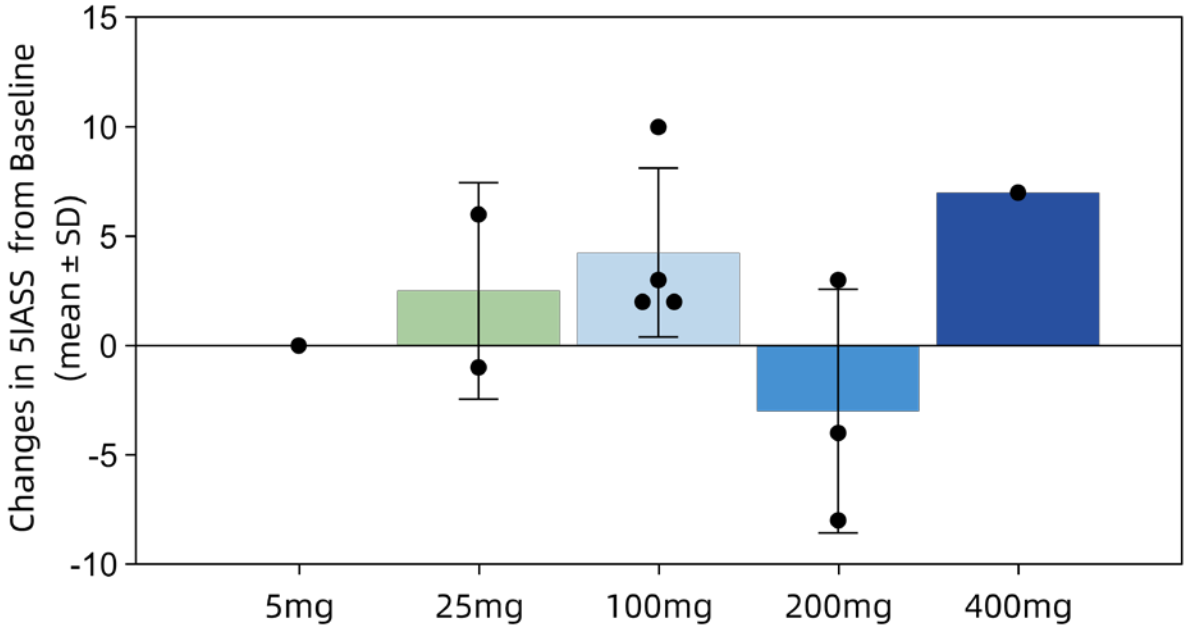
- *Data points indicate values of individual patients; bars indicate mean values.*
- *All patients in the 25 mg group prematurely discontinued from the study before 12-week treatment period completion, thus no available data included in the above plot.*

Improvements in Skeletal Muscle Mass and Appetite

L3SMI by IRC at Week 6



Appetite changes (FAACT-5IASS) at Week 6

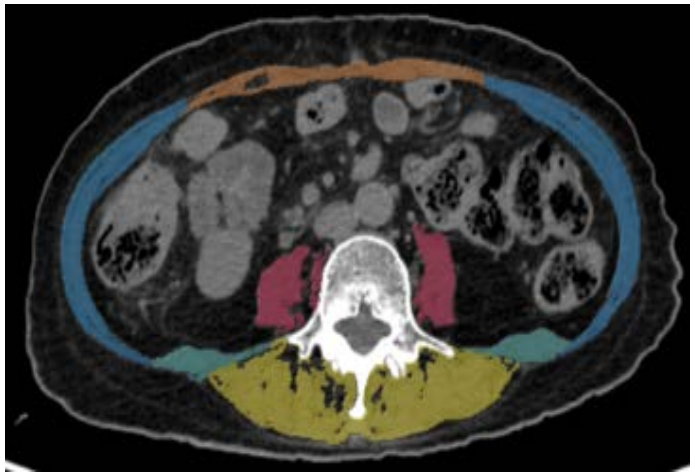


- *Data points indicate values of individual patients; bars indicate mean values.*
- *L3SMI was calculated centrally using Independent Review Committee (IRC). FAACT-5IASS was collected using paper form. Abbreviations: TL, target lesion.*

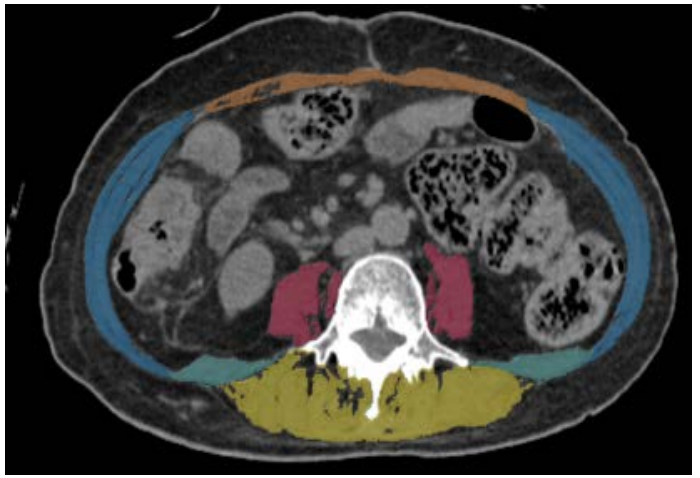
Case Study (GFS202A 100mg Q3W)

Case Study

Baseline



Week 6



- Female, 71 yrs old, gallbladder cancer
- Prior antitumor therapy: GEMOX
- On-treatment antitumor therapy: gemcitabine + pembrolizumab/CAPOX + pembrolizumab
- Baseline GDF15: 957.15 pg/mL; mGPS: 0

Efficacy at Week 6

- Weight: + 9.78% ; L3SMI: + 2.96 cm^2/m^2
- Appetite: + 2 points
- Overall response per RECIST v1.1: PD

Favorable Overall Safety and Tolerability

	Total (N=19), n (%)
At least one TRAE	**8 (42.1)**
Asthenia	1 (5.3)
Anemia	1 (5.3)
Hyperlipidemia	1 (5.3)
Abdominal pain	1 (5.3)
Electrocardiogram QT prolonged	1 (5.3)
Blood pressure increased	1 (5.3)
Protein urine present	1 (5.3)
Hypertension[1]	1 (5.3)
Diarrhea	1 (5.3)
Atrial fibrillation	1 (5.3)
Rash	1 (5.3)
Neutrophil counted ddecreased	1 (5.3)
Complement factor C3 decreased	1 (5.3)
Complement factor C4 decreased	1 (5.3)
Decreased appetite	1 (5.3)
Hypokalaemia	1 (5.3)
Hpyercholesterolaemia	1 (5.3)
Low density lipoprotein increased	1 (5.3)
Glucose urine present	1 (5.3)
Blood triglycerides increased	1 (5.3)
Hyperglycaemia	1 (5.3)

- No DLTs were observed, and the MTD was not reached.
- No patients discontinued or interrupted due to treatment-related AEs (TRAEs).
- Neither infusion-related reaction nor AEs of special interest (defined as infection) occurred.
- Eight patients experienced TRAEs; all were grade 1 or 2 except one grade 3.

The G3 patient had received anlotinib plus capecitabine until 3 days prior to the first dose of GFS202A. Following the first dose of GFS202A, the patient developed asymptomatic G3 hypertension on day 63, which resolved after adequate treatment.

Conclusions

- GFS202A showed a favorable safety profile and promising efficacy, accompanied by reduced systemic inflammation, weight gain, and improved muscle maintenance.

- The data support that dual-targeting GDF15 and IL-6 is a feasible way to mitigate cancer cachexia clinically. Future studies are warranted.

Summary and Outlook

Ongoing trials with expected progress

Mono in pancreatic cancer
Phase III registrational study

Combo with chemo in pancreatic cancer
Phase Ib/II, 1L

Combo with cetuximab in solid tumors
Phase Ib/II, 1L+

GFH375
KRAS G12D (ON/OFF) inhibitor

Recent CDE disclosure of new trials and IND applications

GFH375+GFS202A
Phase II combo IND application accepted

GENFLEET THERAPEUTICS **Sole owner of full RAS & cancer cachexia pipeline**

Novel combo regimen: RAS-targeted therapy + bispecific antibody for cachexia

With potentially superior efficacy vs. comparator combos

Pfizer

GDF15 antibody plus chemo
NCT06989437

REVOLUTION MEDICINES

RAS+RAS
RAS+SOC（PD-1 or chemo）
NCT07491445, NCT06128551, NCT06040541, etc.


Innovation, in Expedition