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CStone 2026 Interim Results

Advancing Pipeline 2.0 & Global Commercialization

August 2026
Stock Code: 2616. HK

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An Innovative Biopharma Driven by Globally Recognized R&D Capability

Proven track record for high-quality and efficient drug development worldwide

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Established in late 2015, CStone is an innovation-driven biopharmaceutical company focused on the research and development of therapies for oncology, immunology/inflammation, and other key disease areas

R&D Milestones

21 NDA Approvals

60+ Data Publications

60+ IND Approvals

10+ Discovery Projects Ongoing

Pipeline 2.0 Strength

13 Novel Assets

Potentially FIC/BIC **ADCs and Mono-/Multi-Specific Antibodies** in Oncology and Immunology/ Inflammation

Commercial Achievement

4 Products Commercialized*

9 Indications Approved

6 Territories Coverage

** The exclusive rights of ivosidenib has been transferred to Servier*

To drive business growth by maximizing commercial value of in-market products and advancing innovative Pipeline 2.0

Commercial-stage Programs

Sugemalimab
(PD-L1)

Pralsetinib
(RET)

Avapritinib
(KIT/PDGFRA)

Key Clinical Programs in Pipeline 2.0

CS2009
(PD-1/VEGF/CTLA-4 trispecific antibody)
Global first-in-class / best-in-class potential

CS5007
(EGFR/HER3 bsADC)

CS5001
(ROR1 ADC)

Innovative Early Programs in Pipeline 2.0

CS5006
(ITGB4 ADC)

CS5008
(SSTR2/DLL3 bsADC)

CS5009
(B7H3/PD-L1 dual-payload ADC)

CS5010
(HER2 biparatopic dual-payload ADC)

CS5012
(HER2 biparatopic novel-payload ADC)

CS1012
(GDF-15 mAb)

CS2013
(BAFF/APRIL bsAb)

CS2015
(OX40L/TSLP bsAb)

CS1016
(PD-1 agonist mAb)

CS2016
(TL1A/α4β7 bsAb)

More Exploratory Programs…

Recurring revenue to fuel pipeline advancement

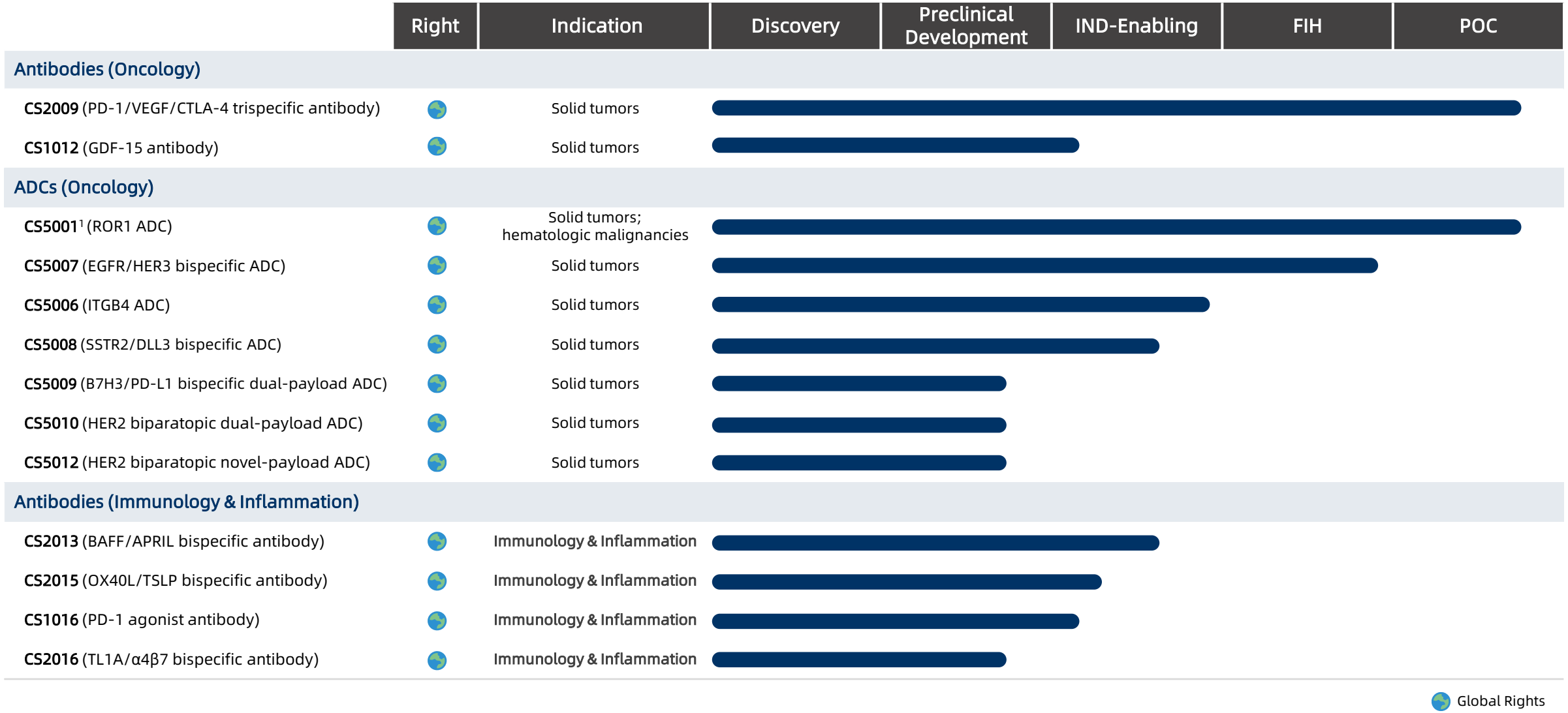
Strong growth momentum in near term

Robust growth engine for the long run

bs: bispecific; Ab: antibody; mAb: monoclonal antibody

Oncology program | I&I program

Progress of Pipeline 2.0



Note: Assets status denotes progress in the region(s) noted in the column titled "Rights" ; FIH = First in Human, POC = Proof of Concept,
1. CStone obtains the exclusive global right from LigaChem Biosciences, Inc. (LCB) to lead development and commercialization of LCB71/CS5001 outside the Republic of Korea

01

Business Highlights

2026 Interim

Sustained Momentum in Pipeline 2.0 and Commercialization

R&D Advancements

- **CS2009** (PD-1/VEGF/CTLA-4 tsAb) Phase I/II data presented at ASCO 2026; two rapid oral presentations at upcoming ESMO 2026; Phase III MRCT initiation expected in 2026
- **CS5007** (EGFR/HER3 ADC) first-in-human (FIH) Phase I study initiated in June 2026, marking the clinical debut of our proprietary ADC platform
- **Immunology & Inflammation TA** expansion

Expanding Global Commercialization

CEJEMLY®/sugemalimab (anti-PD-L1)

- Fifth international commercialization partnership established for Oceania
- EU & U.K. approvals achieved for Stage III NSCLC
- ESMO Guideline recommendation achieved for Stage IV (Feb. 2025) and Stage III (Mar. 2026) NSCLC

Reigniting Growth in China Market

- **Pralsetinib** (RET inhibitor) First-time NRDL inclusion (effective Jan 2026) spurred almost 5x YoY commercial volume growth; first locally manufactured batch released (June 2026)
- **Avapritinib** (KIT/PDGFRA inhibitor) renewed on NRDL (effective Jan 2026); consistent and robust sales of the locally-manufactured product

Solid Financial Position (2026 H1)

- **Total Revenue**: RMB 205.1 M [1], YoY growth +315%
- **R&D Expense**: RMB 193.4 M [2] (increased commitment to Pipeline 2.0; YoY growth +89%)
- **Cash Balance**: RMB 1,560.2 M (as of June 30, 2026)

tsAb: trispecific antibody; NRDL: national reimbursement drug list; NSCLC: non small cell lung cancer
[1] Revenue comprised RMB183.2 million from sales of pharmaceutical products (avapritinib, pralsetinib, and sugemalimab), RMB6.9 million from license fee income, and RMB15.0 million from royalty income related to sugemalimab; [2] Non-IFRS measures

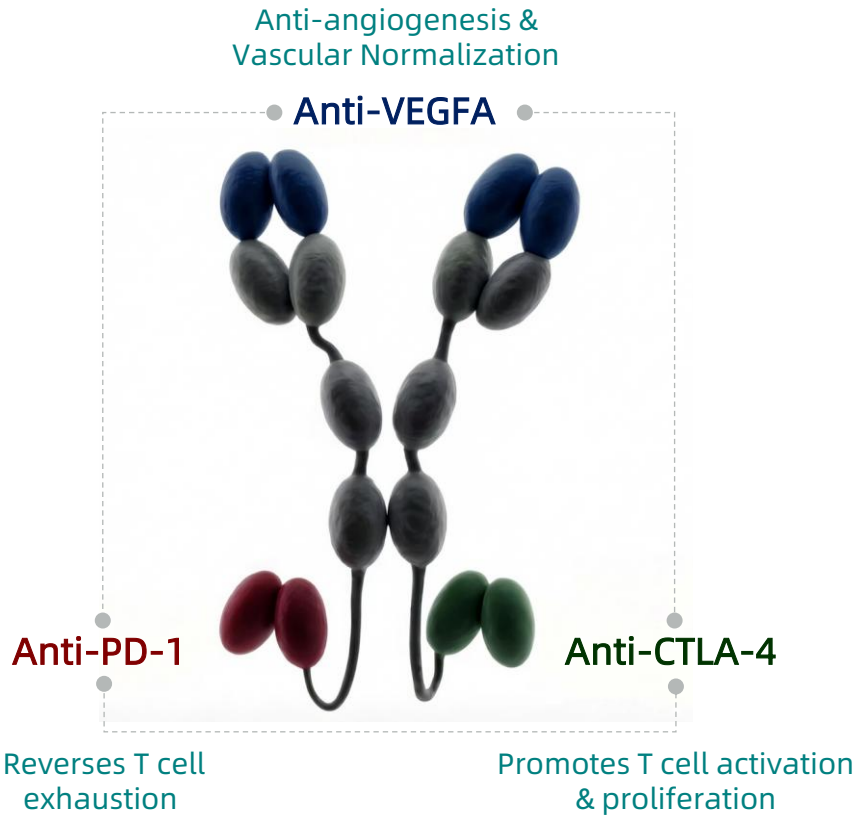
02

Clinical Stage Assets

1. CS2009 (PD-1/VEGF/CTLA-4 trispecific antibody)
2. CS5007 (EGFR/HER3 ADC)
3. CS5001 (ROR1 ADC)

Innovative MOA: Enhancing Survival Benefit while Minimizing Toxicity via Triple Synergy

Potential First-in-Class / Best-in-Class Next-Generation Immuno-Oncology (I/O) Backbone



Triple synergy (PD-1/VEGF/CTLA-4)

- Simultaneously targets three pathways to restore T cell function, improve tumor microenvironment, and enhance initial T cell activation - aiming for deeper and durable anti-tumor immune responses

Overcomes toxicity and efficacy limitations of conventional immuno-combination therapies

- Addresses high toxicity of conventional CTLA-4 antibodies that limits continuous dosing and uncertain long-term OS benefit from PD-1/VEGF combinations or bispecifics
- Differentiated design reduces systemic immune-related and VEGF-related toxicities, enabling long-term continuous dosing

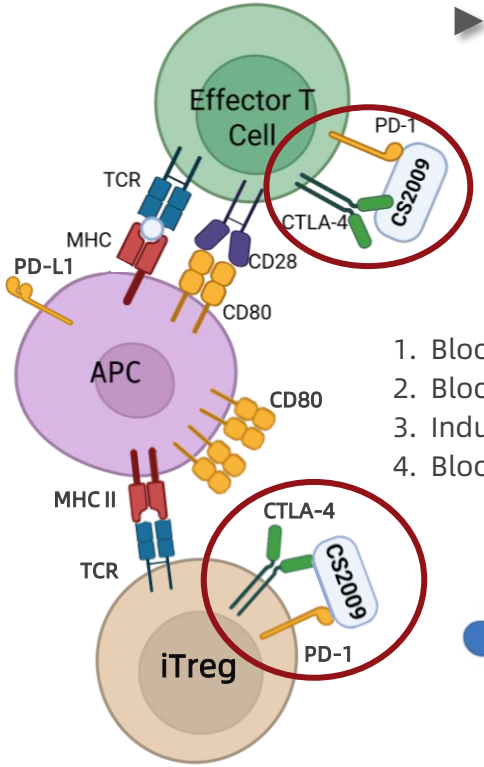
Expands immunotherapy boundaries and potential for long-term survival

- With a favorable safety profile, consistent clinical activity observed in multiple traditionally “cold tumors” (e.g., pMMR/MSS colorectal cancer) and heavily pretreated non-small cell lung cancer
- Potential to cover a broader patient population and deliver long-term OS benefit

MoA: Mechanism of Action

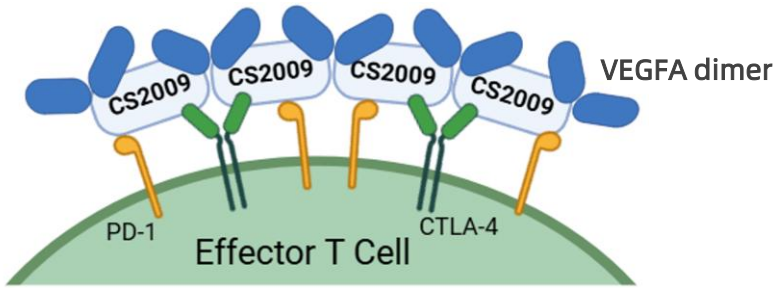
Innovative MOA: Multi-target engagement and synergistic interactions enhance CS2009's activities in TME while sparing peripheral CTLA4+ T cells to widen its therapeutic window

Tumor Microenvironment (TME)



Within the TME, CS2009 **preferentially binds PD-1 and CTLA-4 on double-positive tumor-infiltrating lymphocytes (Teff and Treg)** via avidity-driven concurrent engagement of both checkpoints, enhancing its binding affinity and checkpoint inhibitory (CPI) activity to reinvigorate Teff and block Treg activities, including:

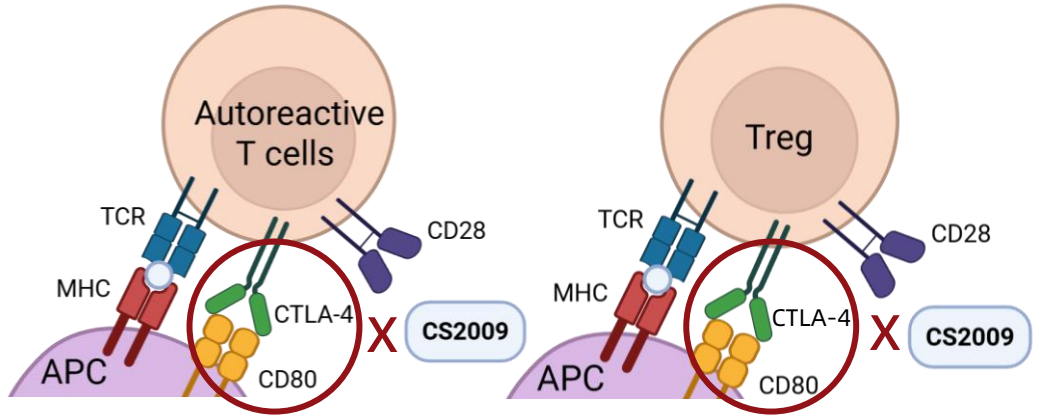
1. Blocking PD-1/PD-L1 interaction on **Teff**
2. Blocking CTLA-4/CD80 interaction on **Teff & Treg**
3. Inducing internalization of PD-1 & CTLA-4 on **Teff & Treg**
4. Blocking **Treg**-driven transcytosis of CD80 from APC surface



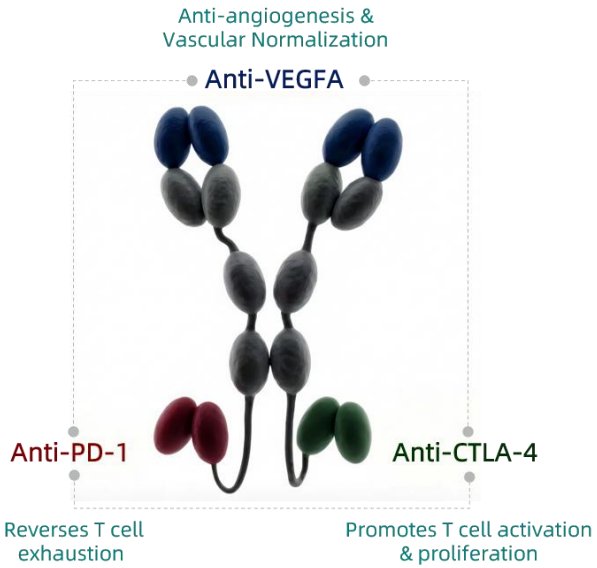
Within the TME, CS2009 forms clusters after crosslinking with VEGFA dimers, which **strengthens its binding affinity to PD-1/CTLA-4-expressing T cells** and further amplifies its CPI activity

Peripheral

In the peripheral, CS2009's **monovalent** CTLA-4 arm is **unable to block CTLA-4/CD80 interactions** due to low affinity, thereby sparing CTLA-4 single-positive T cells from over-activation, thus reducing systemic autoimmune toxicity



Triple Synergy in the Tumor Microenvironment leads to enhanced anti-tumor activities



1 Anti-CTLA-4 Action in TME

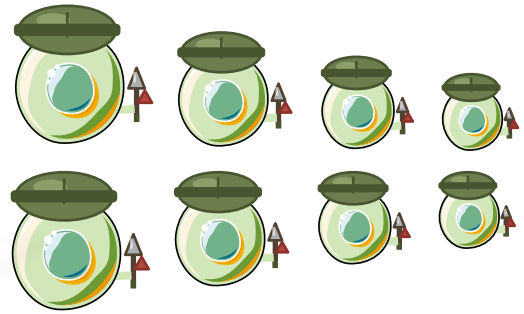
➢ **Recruit and train T-cells inside TME**

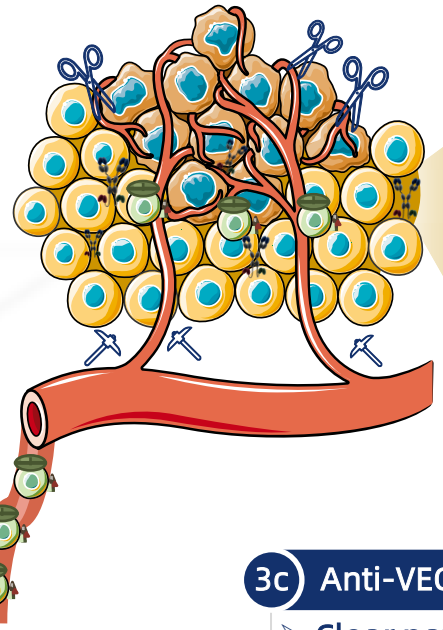
- Blocks CTLA-4 inhibitory checkpoint on T cells
- Drives T-cell priming and clonal expansion
- Expands tumor-reactive effector T-cell pool

2 Anti-PD-1 Action in TME

➢ **Revive exhausted T-cells**

- Blocks PD-1 inhibitory checkpoint on T cells
- Reverses T-cell exhaustion
- Restores cytotoxic function





3a Anti-VEGFA Actions

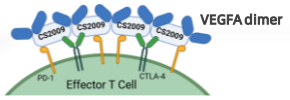
➢ **Cut off blood supply**

- Blocks pathological angiogenesis
- Prunes immature & leaky tumor vessels
- Restricts tumor nutrient supply & growth

3b VEGFA-Crosslinking

➢ **Boost IO activities**

- VEGFA-dimer-driven CS2009 clustering boosts PD-1/CTLA-4⁺ T-cell binding and IO activities in TME



3c Anti-VEGFA Actions

➢ **Clear pathways for T-cells**

- Induces tumor vascular normalization
- Improves vessel structure & perfusion
- Enhances T-cell trafficking into tumor

Note: Image adapted from Servier Medical Art, licensed under CC BY 4.0
TME: tumor microenvironment

CS2009: Vision for the Next-Gen I/O Backbone

Replacing & Expanding beyond Current PD-1/L1 Standard of Care (SOC)

PD-1/L1 Fully Established SOC (IO-based Regimens)

- **1L metastatic NSCLC:** Pembrolizumab + Platinum-based Chemo
- **Locally advanced NSCLC, post-CRT:** Durvalumab consolidation
- **Resectable NSCLC (Neoadjuvant/Adjuvant):** Nivolumab + Platinum-based Chemo
- **ES-SCLC:** Atezolizumab + Etoposide + Carboplatin
- **LS-SCLC:** Durvalumab consolidation
- **1L HCC:** Atezolizumab + Bevacizumab
- **1L HNSCC:** Pembrolizumab + Chemo
- **1L Advanced ccRCC:** Pembrolizumab + Axitinib
- **Unresectable/Metastatic Melanoma:** Pembrolizumab

PD-1/L1 Partially Established SOC (CPS-Dependent)

- **1L TNBC:** **CPS ≥10**: Pembrolizumab + Chemo; **CPS <10**: Chemo
- **1L GC/GEJ:** **CPS ≥1**: Pembrolizumab + Chemo; **CPS <1**: Chemo
- **1L ESCC:** **CPS ≥1**: Nivolumab + Chemo; **CPS <1**: Chemo
- **2L/3L PROC:** **CPS ≥1**: Pembrolizumab + Paclitaxel +/- Bevacizumab
- **1L Cervical Cancer (recurrent/metastatic):** **CPS ≥1**: Pembrolizumab + Bevacizumab; **CPS <1**: Chemo + Bevacizumab

PD-1/L1 Ineligible (I/O Not Yet Approved as SOC)

- **2L/3L Metastatic NSCLC (AGA-):** Docetaxel
- **1L pMMR/MSS CRC:** Bevacizumab + FOLFOX/CAPEOX/FOLFIRI
- **Non-clear-cell RCC:** TKI
- **mCRPC:** Chemo

→ **CS2009 Replacing PD-1/L1 in Chemo Combo**

→ **CS2009 Expanding into IO-Ineligible with Chemo/ADC**

AGA: actionable oncogenic alterations; CAPEOX: Capecitabine + Oxaliplatin; ccRCC: clear cell renal cell carcinoma; CPS: combined positive score; CRC: colorectal cancer; CRT: chemoradiotherapy; ES-SCLC: extensive-stage small cell lung cancer; ESCC: esophageal squamous cell carcinoma; HCC: hepatocellular carcinoma; HNSCC: head and neck squamous cell carcinoma; LS-SCLC: limited-stage small cell lung cancer; mCRPC: metastatic Castration-Resistant Prostate Cancer; NSCLC: non-small cell lung cancer; PROC: platinum-resistant ovarian cancer; pMMR/MSS: proficient mismatch repair or microsatellite stable; TNBC: triple-negative breast cancer

PD-1/VEGF/CTLA-4 trispecific mAb holds great clinical and commercial value with potentially durable OS benefit

Greater potential than PD-1/VEGF bsAbs to become the next-generation I/O backbone to replace anti-PD-(L)1 antibodies in current SOC

Approved indications for 3 targets

- Lung cancer ►
- Hepatocellular carcinoma ►
- Renal cell carcinoma ►
- Colorectal cancer ►

Approved indications for PD-(L)1 & VEGF

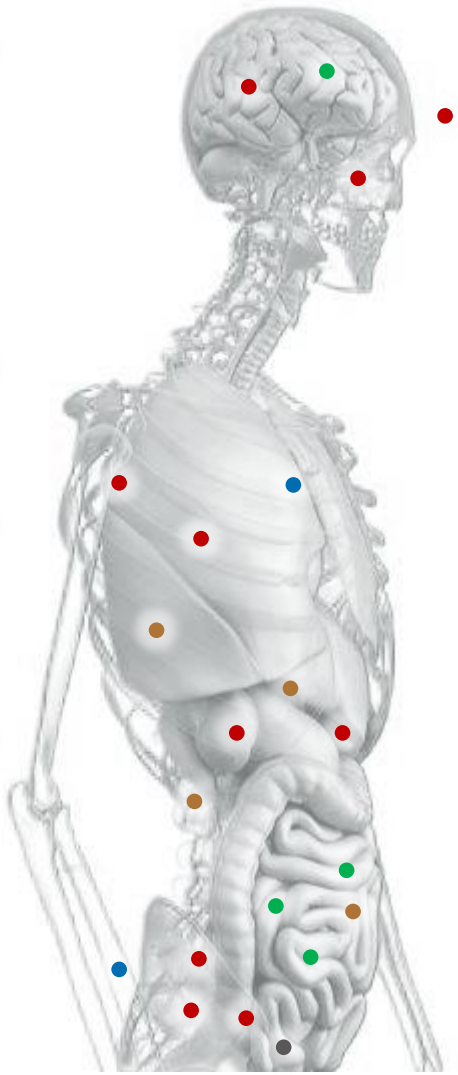
- Cervical cancer ►

Approved indications for PD-(L)1 & CTLA-4

- Esophageal cancer ►
- Melanoma ►

Approved indications for VEGF

- Glioblastoma ►
- Peritoneal cancer ►
- Ovarian cancer ►
- Fallopian tube carcinoma ►
- …… ……



Approved indications for PD-(L)1

- ◄ Head and neck squamous cell carcinoma
- ◄ Nasopharyngeal carcinoma
- ◄ Hodgkin lymphoma
- ◄ Triple negative breast cancer
- ◄ Biliary tract cancer
- ◄ Gastric cancer
- ◄ Urothelial carcinoma
- ◄ Bladder cancer
- ◄ Endometrial cancer
- …… ……

Broad indications with huge clinical potential

60+ Approved indications targeting PD-(L)1, VEGF, CTLA-4 (mono or multi-targeting)

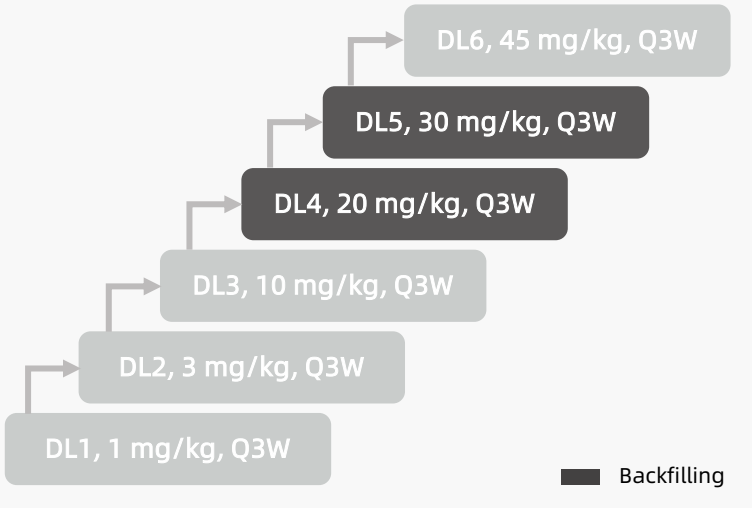
Remarkable commercial value to exceed current market potential for CPI

$30bn	Keytruda 2024 global sales revenue
$53bn	PD-(L)1 inhibitors 2024 global sales revenue
$90bn	PD-(L)1 global sales revenue by 2028 (IQVIA forecast)
$90bn	Current CPI market potential (Summit's projection)

Note: The indications listed above are not exhaustive of all approved indications for the three targets
CPI, checkpoint inhibitor

CS2009 Clinical Development Plan: Efficient and Clearly Defined Global Development Path Forward

Phase I – Mono Dose Escalation (Completed) + Backfilling



Phase II – Dose Expansion

Lung

- 1L NSQ-NSCLC, AGA(-), plus PEM + CPT
- 1L SQ-NSCLC, AGA(-), plus PTX + CPT
- Treated NSCLC–[2L] Mono, [2/3L] plus docetaxel
- 1L NSCLC, AGA(-), PD-L1 TPS ≥1%, Mono
- 1L ES-SCLC, plus ETO + CPT

Gastrointestinal

- 1L pMMR/MSS CRC / pancreatic cancer, plus chemo
- 1L gastric cancer, plus CAPOX
- 1L ESCC, plus TP/FP
- HCC–[1L] Mono; [2/3L] Mono

Breast

- 1L TNBC, plus nab-PTX
- 2/3L TNBC, Mono

Gynecologic

- 1L cervical cancer, plus PTX + Pt
- PROC–[1/2L] plus PTX; [2/3L] Mono

Kidney & Others

- 1L RCC, Mono
- 1L MSI-H/dMMR solid tumors, Mono

Prioritized Phase III MRCT Trials

- **1L metastatic NSQ-NSCLC, AGA(-),** CS2009 + PEM + CPT vs. pembrolizumab + PEM + CPT
- **1L metastatic SQ-NSCLC, AGA(-),** CS2009 + PTX + CPT vs. pembrolizumab + PTX + CPT
- **2/3L metastatic NSCLC, AGA(-),** CS2009 + docetaxel vs. docetaxel
- **1L metastatic NSCLC, AGA(-), PD-L1 TPS ≥50%,** CS2009 vs. pembrolizumab
- **Stage III NSCLC, post-CRT consolidation** CS2009 vs. durvalumab
- **1L ES-SCLC,** CS2009 + ETO + CPT vs. atezolizumab + ETO + CPT
- **1L MSS CRC,** CS2009 + FOLFOX vs. bevacizumab + FOLFOX
- **1L TNBC,** CS2009 combination TBD, contingent on regulatory feedback
- **1L Clear-cell RCC,** CS2009 +/- axitinib vs. pembrolizumab + axitinib
- **1L HCC,** CS2009 vs. atezolizumab + bevacizumab
- **1L MSI-H Solid Tumors,** Selected tumor types

AGA: actionable oncogenic alterations; CAPOX: Capecitabine + Oxaliplatin; CPT: Carboplatin; CRC: colorectal cancer; DL: dose level; ES-SCLC: extensive-stage small cell lung cancer; ESCC: esophageal squamous cell carcinoma; ETO: Etoposide; FOLFOX: folinic acid, fluorouracil and oxaliplatin; *FP: 5-fluorouracil + Cisplatin; HCC: hepatocellular carcinoma; NSCLC: non-small cell lung cancer; NSQ: non-squamous; PEM: Pemetrexed; PROC: platinum-resistant ovarian cancer; PTX: Paclitaxel; Pt: platinum; pMMR/MSS: proficient mismatch repair or microsatellite stable; Q3W: once every 3 weeks; RCC: renal cell carcinoma; SQ: squamous; TNBC: triple-negative breast cancer; TP: Paclitaxel + Cisplatin; TPS: tumor proportion score*

R&D Milestones & Upcoming Catalysts

- **Mar. 2025** – First patient dosed in global phase I trial
- **Sept. 2025** – First patient dosed in global phase II trial
- **Q4 2025** – Phase I data debut at ESMO
- **Feb 2026** – US IND
- **Q2 2026** – Phase I data and preliminary Phase II data presented at ASCO
- **Q4 2026** – Two oral presentations at ESMO
- **Q4 2026** – Initiating Phase III registrational trial

Three Important Key Clinical Validations Achieved from over 300 Patient Data

Phase I/II key updates **Aug. 2026**

2026 ASCO **May 2026**

Preliminary 1L NSCLC mono and Phase I data **Apr. 2026**

2025 ESMO *Presented Phase I data from 72 patients* **Oct. 2025**

Phase II *First patient in* **Sept. 2025**

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➢ Proof of Safety

- **Grade ≥3 irAEs 12.7%**; CTLA-4-related tox markedly reduced from CTLA-4 mAb monotherapy or in combination with PD-1/L1, including **extremely rare colitis,** due to **preserved CTLA-4 signaling** in peripheral Tregs & autoreactive T cells
- **Grade ≥3 VEGF-related tox 5.1%;** likely mediated by PD-1/CTLA-4-driven VEGFA immune complex internalization by T cells in TME as indicated by the data

➢ Proof of CTLA-4 Pharmacology & Anti-tumor Efficacy

- Dose-dependent **ICOS upregulation on CD4 T cells**: PD biomarker of CTLA-4 blockade
- Robust activity in **IO-unresponsive "cold tumors ",** including STS, nccRCC, and pMMR/MSS CRC
- Robust anti-tumor activity in **heavily pretreated, IO-refractory/resistant NSCLC**

➢ Proof of Broad Anti-tumor Efficacy beyond Standard IO Regimens

- Robust mono & chemo-combo efficacy in **1L and late-line NSCLC**
- Robust mono & chemo-combo efficacy in **1L and late-line pMMR/MSS CRC**
- Promising mono activity in mCRPC, OC, TNBC, GC, EC, in addition to STS and nccRCC

TME: tumor microenvironment; ICOS: inducible T-Cell Co-stimulator; NSCLC: non-small cell lung cancer; STS: soft tissue sarcoma; nccRCC: non-clear-cell renal cell carcinoma; pMMR/MSS: proficient mismatch repair / microsatellite stable; CRC: colorectal cancer; mCRPC: metastatic Castration-Resistant Prostate Cancer; OC: ovarian cancer; TNBC: triple-negative breast cancer; GC: gastric cancer; EC: esophageal cancer

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Phase I Safety: Phase I Data Demonstrate Compelling Safety Profile

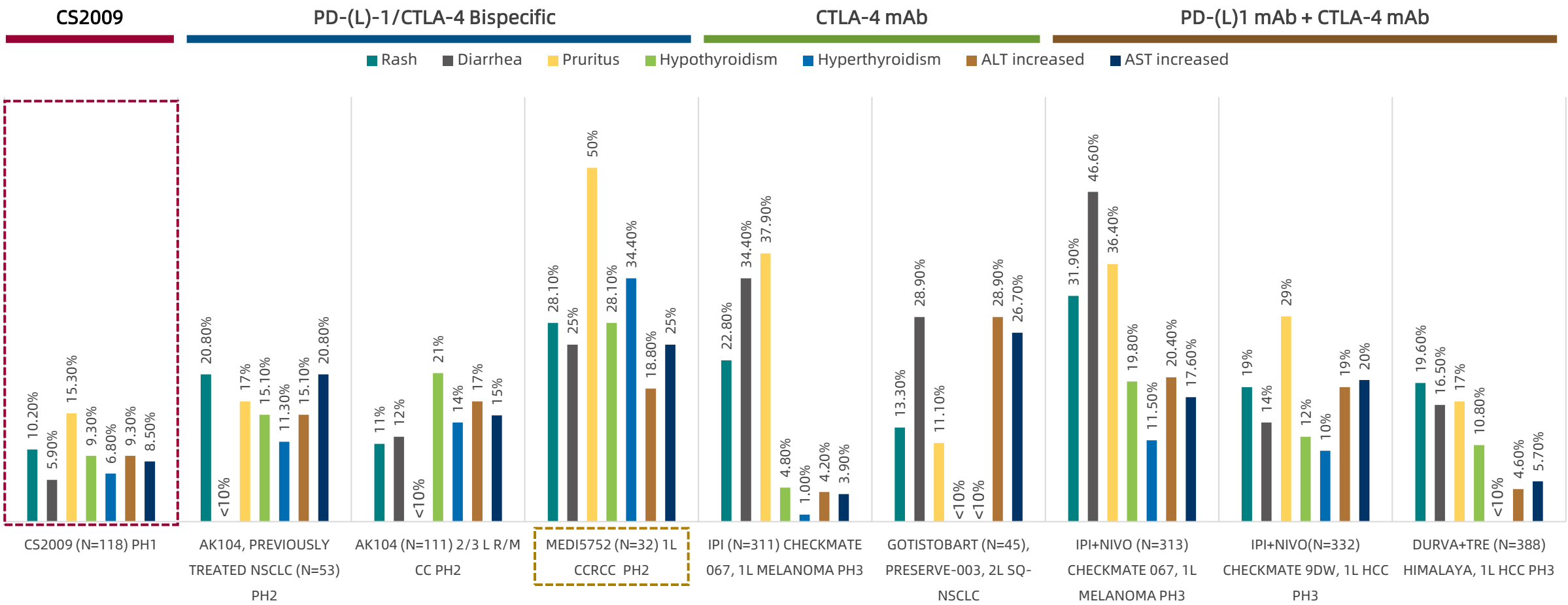
- Dose escalation completed; no DLT; MTD not reached
- Without excessive toxicities that typically occurred in CTLA-4- and PD-(L)1-containing combination therapies; low ≥G3 VEGF-related AEs
- **Up to date, no new safety signals identified; CS2009's safety profile remained consistent with that presented at 2026 ASCO**

n (%)	1-10 mg/kg (n=21)	20 mg/kg (n=33)	30 mg/kg (n=54)	45 mg/kg (n=10)	All DLs (N=118)
TEAE	21 (100)	31 (93.9)	45 (83.3)	10 (100)	107 (90.7)
Grade ≥3 TEAE	10 (47.6)	15 (45.5)	20 (37.0)	5 (50.0)	50 (42.4)
Treatment-related TEAE (TRAE)	18 (85.7)	27 (81.8)	39 (72.2)	10 (100)	94 (79.7)
• Grade ≥3 TRAE	6 (28.6)	7 (21.2)	13 (24.1)	3 (30.0)	**29 (24.6)**
Immune-related TEAE (irAE)	9 (42.9)	18 (54.5)	16 (29.6)	3 (30.0)	46 (39.0)
• Grade ≥3 irAE	3 (14.3)	6 (18.2)	5 (9.3)	1 (10.0)	**15 (12.7)**
Infusion-related reaction	1 (4.8)	1 (3.0)	1 (1.9)	2 (20.0)	5 (4.2)
TRAE possibly related to anti-VEGF	5 (23.8)	11 (33.3)	9 (16.7)	2 (20.0)	27 (22.9)
• Grade ≥3 TRAE possibly related to anti-VEGF	2 (9.5)	1 (3.0)	3 (5.6)	0	**6 (5.1)**
TRAE leading to drug discontinuation	1 (4.8)	3 (9.1)	5 (9.3)	0	9 (7.6)

- Most frequent (≥5%) any-grade **irAE:** hypothyroidism (6.8%), rash / AST increased / ALT increased (5.1% each)
- Most frequent (≥5%) any-grade **TRAE possibly related to anti-VEGF therapy:** proteinuria (11.9%), hypertension (8.5%)

TEAE: treatment-emergent adverse event; AST: aspartate aminotransferase; ALT: alanine aminotransferase

Safety Comparison: Much lower incidence of frequent any-grade irAE vs. PD-(L)1/CTLA-4 bispecific or combination regimens

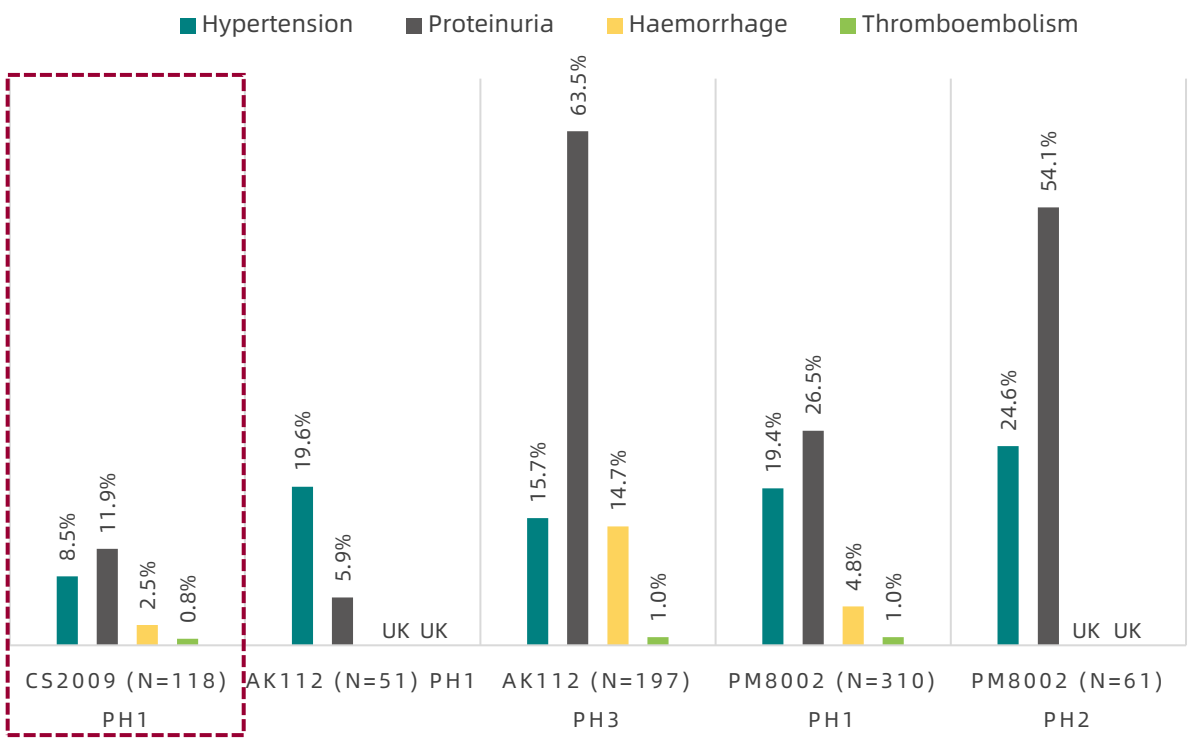


Note: AE of <10% incidence not shown

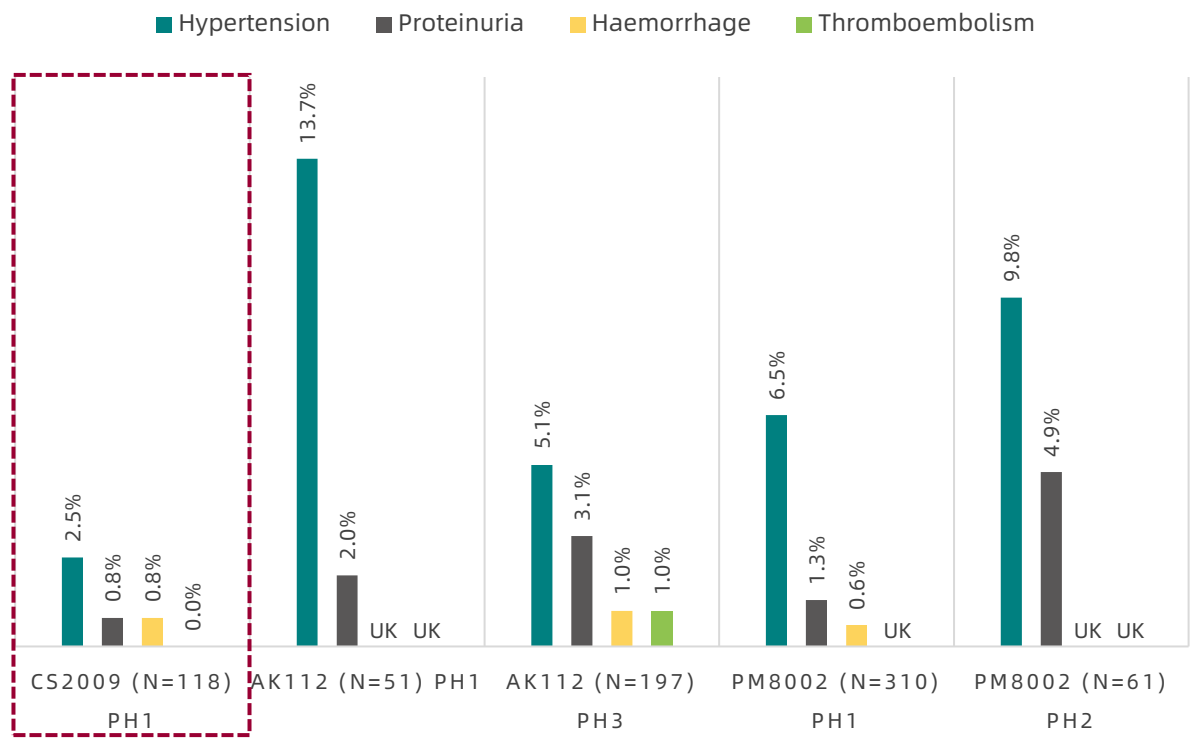
Source: Lung Cancer 184 (2023) 107355; Lancet Oncol 2023; 24: 1134-46; 2023 ESMO; April 16, 2018, at NEJM.org; N Engl J Med. 2025 January 02; 392(1): 11-22; Lancet 2025; 405: 1851-64; June 6, 2022 NEJM Evid 2022; 1 (8); Nat Med. 2026 Mar 27

Safety Comparison: Markedly lower incidence of any-grade or Grade ≥3 TRAE possibly related to anti-VEGF vs. PD-(L)1/VEGF bispecifics

Any-grade TRAE possibly related to anti-VEGF

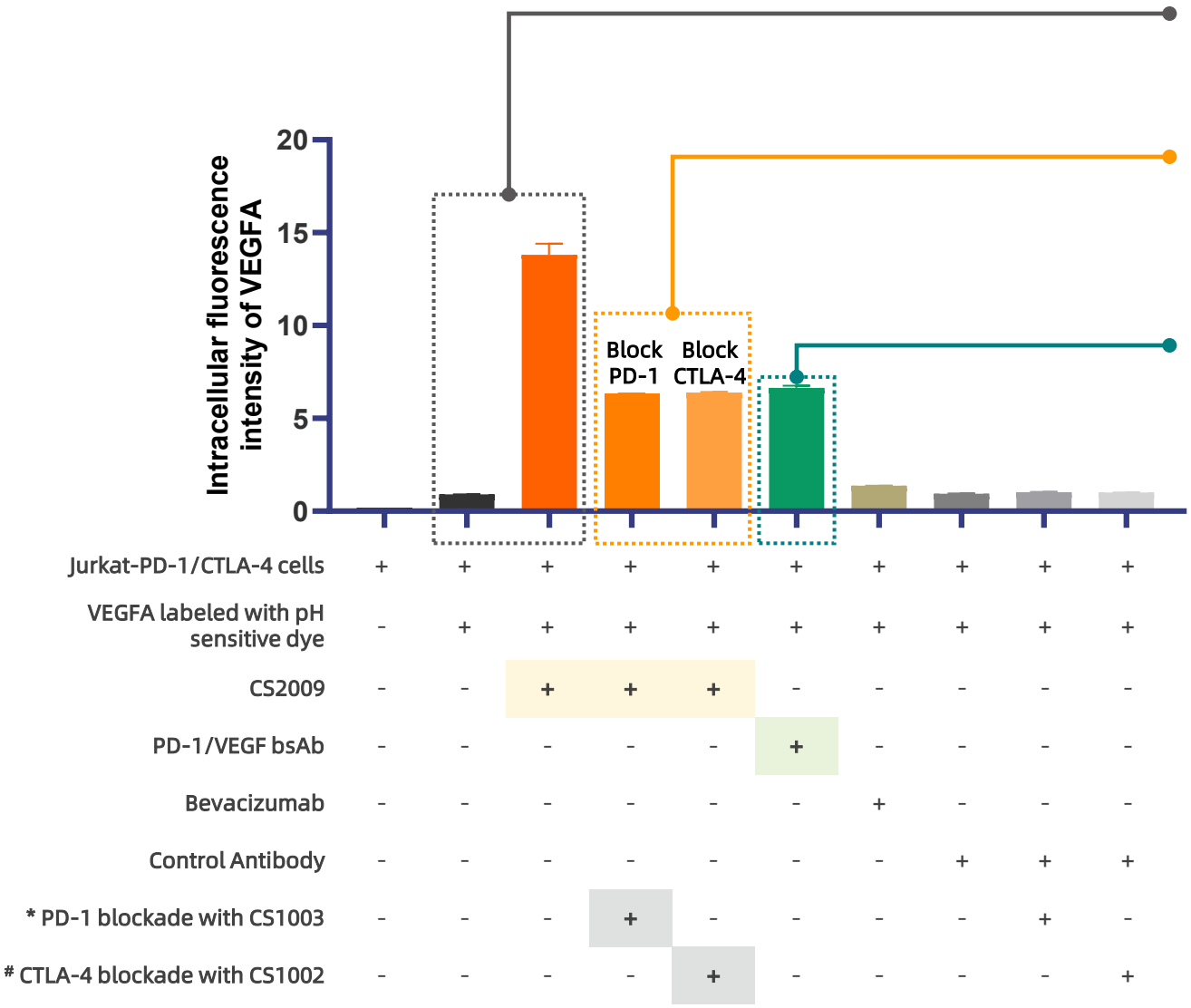


Grade ≥3 TRAE possibly related to anti-VEGF



TRAE: treatment-emergent adverse event; UK: unknown
Source: 2026 ASCO; J Immunother Cancer. 2024 Apr 19;12(4):e008037; Lancet 2025; 405: 839-49; 2023 ASCO, #414802; 2024 ASCO, #8533

VEGFA Internalization Assay: CS2009 mediates VEGFA internalization by T cells via dual PD-1 and CTLA-4 pathways



** CS1003 is the parental antibody of CS2009's PD-1 arm*
 # CS1002 is the parental antibody of CS2009's CTLA-4 arm

CS2009 Mediates Robust VEGFA Internalization by Jurkat T Cells Expressing PD-1 and CTLA-4

CS2009 (orange bar) vs. control (black bar)

CS2009 Mediates VEGFA Internalization via Dual PD-1/CTLA-4 Pathways

Blockade of either PD-1 or CTLA-4 attenuates CS2009-mediated VEGFA internalization, indicating that both pathways contribute synergistically to VEGFA clearance by T cells

CS2009 Induces Greater VEGFA Internalization than a PD-1/VEGF bispecific antibody

PD-1/VEGF bsAb mediates substantially less VEGFA internalization (green bar vs. 1st orange bar from the left) via PD-1 pathway alone

Multiple Mechanisms of Action:

"VEGF Signaling Blockade" + "Active Directional VEGF Clearance" + "Checkpoint Downregulation"

- Clearance of VEGFA and its immune-complexes in the TME may limit their redistribution to the periphery and thereby attenuate common VEGF-related toxicities - including hypertension, proteinuria, and hemorrhage - conferring a differentiated safety profile for CS2009
- Although not the primary objective of this assay, the data indicate that CS2009 also induce efficient internalization of cell-surface PD-1 and CTLA-4. Beyond its checkpoint blockade activity, this enhanced checkpoint downregulation on T cells may further augment the immuno-oncologic activity of CS2009

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Phase I/II key updates **Aug. 2026**

2026 ASCO **May 2026**

Preliminary 1L NSCLC mono and Phase I data **Apr. 2026**

2025 ESMO *Presented Phase I data from 72 patients* **Oct. 2025**

Phase II *First patient in* **Sept. 2025**

Phase I *First patient in* **Mar. 2025**

➢ Proof of Safety

- **Grade ≥3 irAEs 12.7%**; CTLA-4-related tox markedly reduced from CTLA-4 mAb monotherapy or in combination with PD-1/L1, including **extremely rare colitis,** due to **preserved CTLA-4 signaling** in peripheral Tregs & autoreactive T cells
- **Grade ≥3 VEGF-related tox 5.1%;** likely mediated by PD-1/CTLA-4-driven VEGFA immune complex internalization by T cells in TME as indicated by the data

➢ Proof of CTLA-4 Pharmacology & Anti-tumor Efficacy

- Dose-dependent **ICOS upregulation on CD4 T cells**: PD biomarker of CTLA-4 blockade
- Robust activity in **IO-unresponsive "cold tumors ",** including STS, nccRCC, and pMMR/MSS CRC
- Robust anti-tumor activity in **heavily pretreated, IO-refractory/resistant NSCLC**

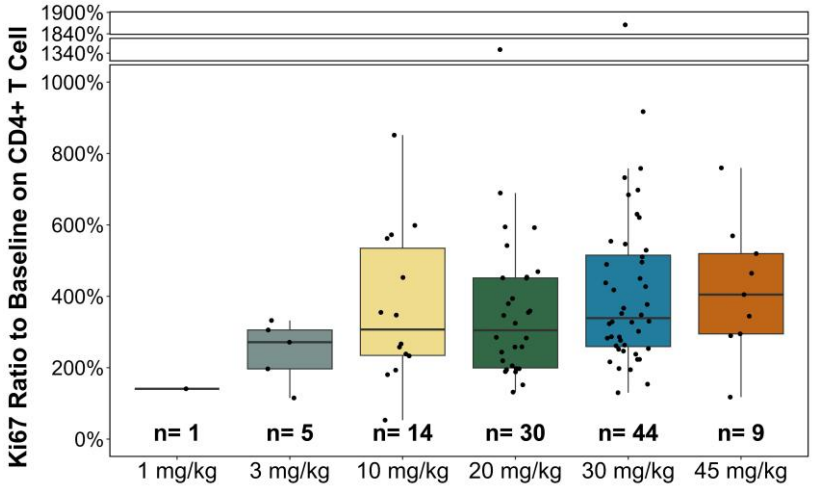
➢ Proof of Broad Anti-tumor Efficacy beyond Standard IO Regimens

- Robust mono & chemo-combo efficacy in **1L and late-line NSCLC**
- Robust mono & chemo-combo efficacy in **1L and late-line pMMR/MSS CRC**
- Promising mono activity in mCRPC, OC, TNBC, GC, EC, in addition to STS and nccRCC

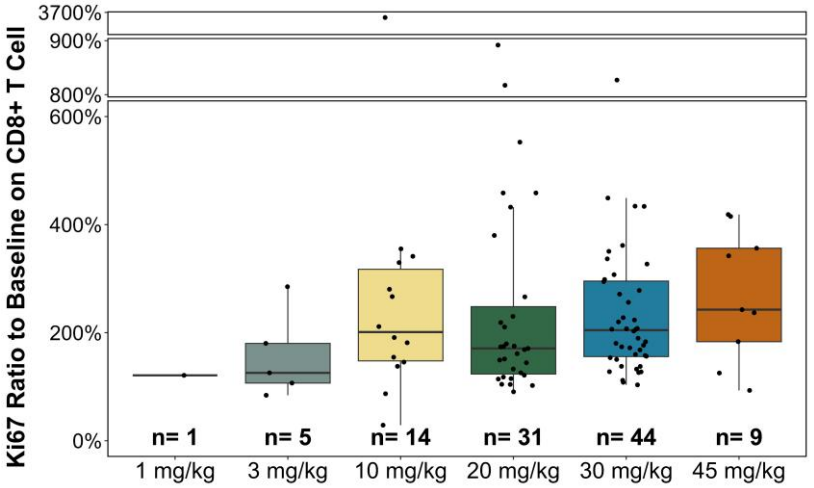
TME: tumor microenvironment; ICOS: inducible T-Cell Co-stimulator; NSCLC: non-small cell lung cancer; STS: soft tissue sarcoma; nccRCC: non-clear-cell renal cell carcinoma; pMMR/MSS: proficient mismatch repair / microsatellite stable; CRC: colorectal cancer; mCRPC: metastatic Castration-Resistant Prostate Cancer; OC: ovarian cancer; TNBC: triple-negative breast cancer; GC: gastric cancer; EC: esophageal cancer

Robust T cell activation (Ki67 and ICOS up-regulation with plateau at doses ≥10 mg/kg)

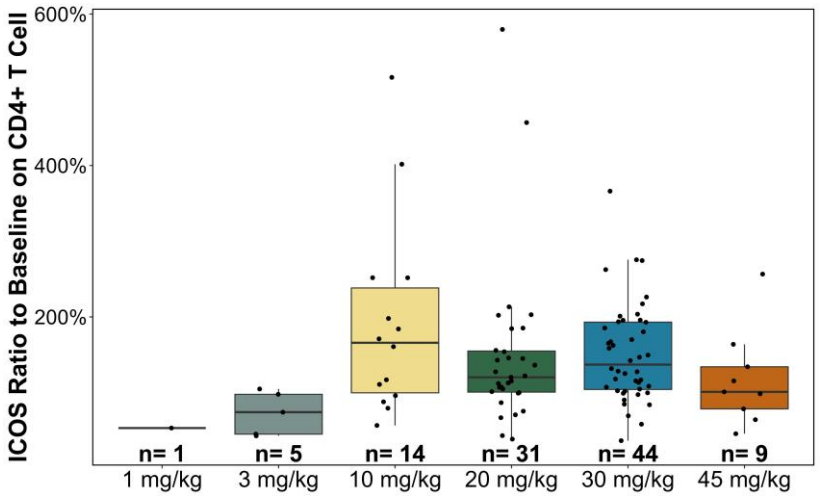
Ki67 expression on CD4+ T cells



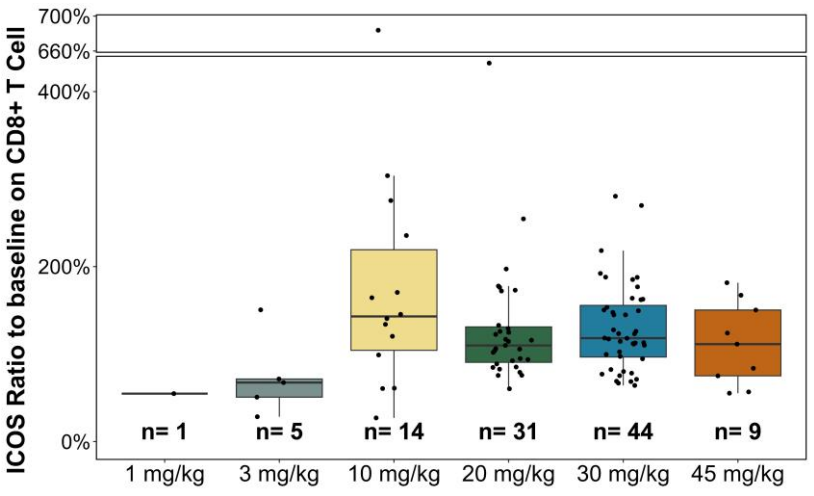
Ki67 expression on CD8+ T cells



ICOS expression on CD4+ T cells



ICOS expression on CD8+ T cells



- **T-cell proliferation & activation:** On Cycle 1 Day 8, CS2009 induced notable, dose-dependent upregulation of **Ki67** (proliferation) and **ICOS** (activation) expression on both **CD4+** and **CD8+ T cells**, collectively demonstrating effective PD-1 and CTLA-4 inhibition

CD4_Ki67_ratio = CD4_Ki67+% (day8 / baseline)
CD4_Ki67+%= (CD45+CD3+%) × (CD3+CD4+%) × (CD3+CD4+Ki67%)

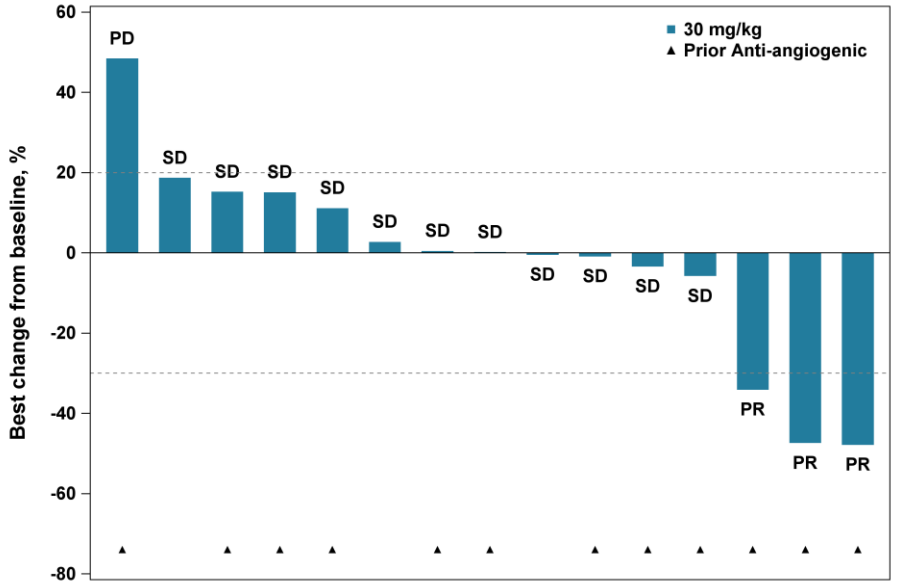
(1/3) CS2009 monotherapy demonstrated promising clinical efficacy in later-line metastatic CRC, mostly pMMR/MSS – a "cold tumor" not sensitive to PD-(L)1 mAb

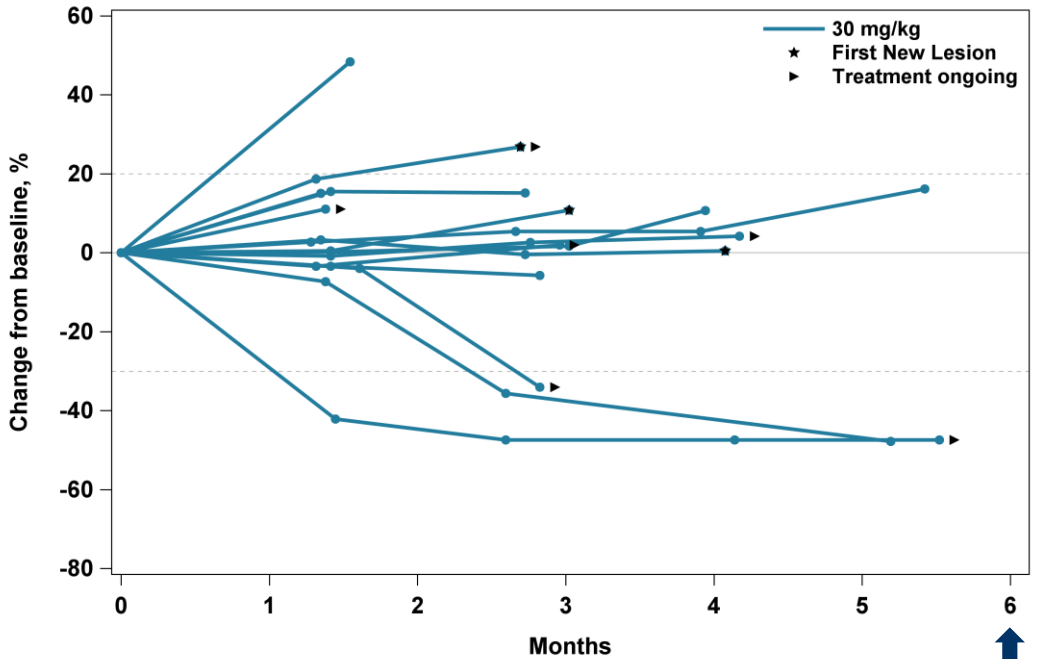
Monotherapy at 30 mg/kg, Q3W

	Aug-2026 Cutoff	2026-ASCO Cutoff
Evaluable patients, n	15	8
ORR, % (n)	**20.0%** (3/15)	25.0% (2/8)
DCR, % (n)	**93.3%** (14/15)	87.5% (7/8)

CLINICAL CATALYST

Data for 1L combination with chemo in mCRC to be presented at 2026 ESMO





pMMR/MSS: proficient mismatch repair / microsatellite stable; mCRC: metastatic colorectal cancer; ORR: objective response rate; DCR: disease control rate
Data source: In-house Aug-2026 update

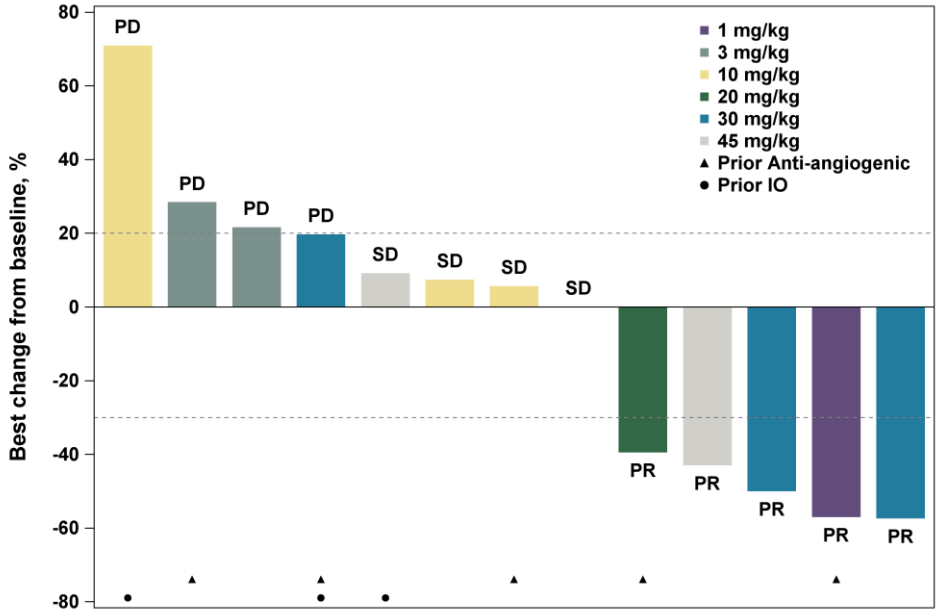
(2/3) CS2009 monotherapy demonstrated promising clinical efficacy in later-line soft tissue sarcoma (STS) – a "cold tumor" not sensitive to PD-(L)1 mAb

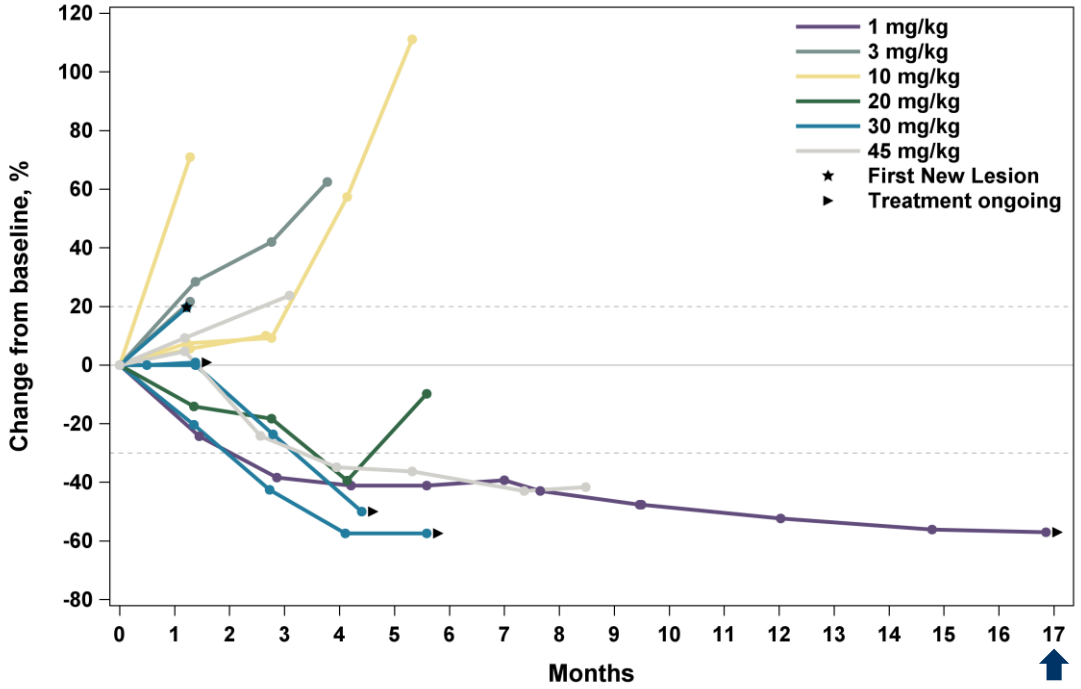
Monotherapy at 1, 3, 10, 20, 30, 45 mg/kg, Q3W

	Aug-2026 Cutoff	2026-ASCO Cutoff
Evaluable patients, n	13	12
ORR, % (n)	38.5% (5/13)	33.3% (4/12)
DCR, % (n)	69.2% (9/13)	66.7% (8/12)

CLINICAL CONTEXT

Treatment options in clinic for STS are mainly TKIs, of which ORR is typically ~20% and ~6% in the first-line and later-line setting, respectively





ORR: objective response rate; DCR: disease control rate; TKI: tyrosine Kinase Inhibitor: IO: immuno-oncology
Data source: In-house Aug-2026 update

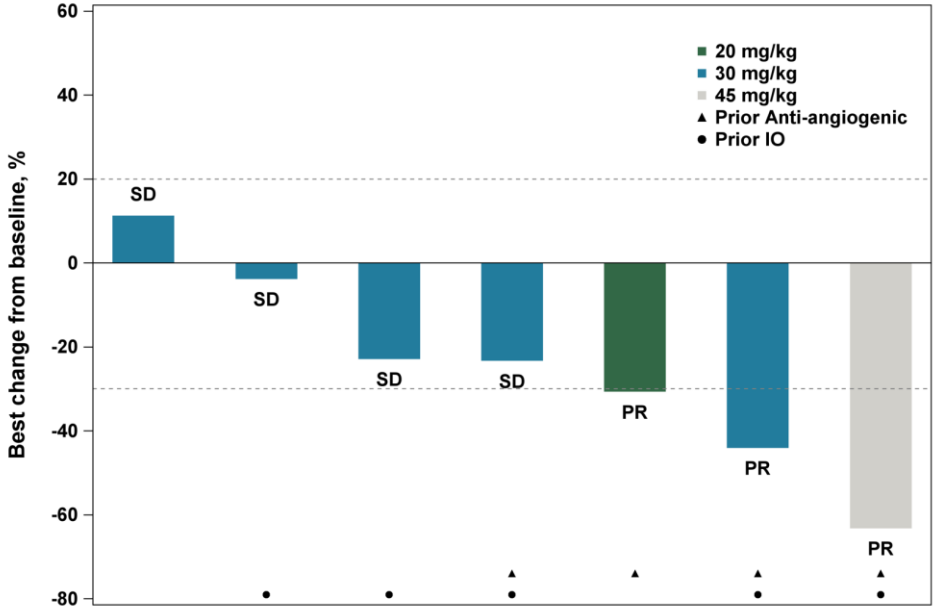
(3/3) CS2009 monotherapy demonstrated promising clinical efficacy in later-line non-clear-cell renal cell carcinoma (nccRCC) – a "cold tumor" not sensitive to PD-(L)1 mAb

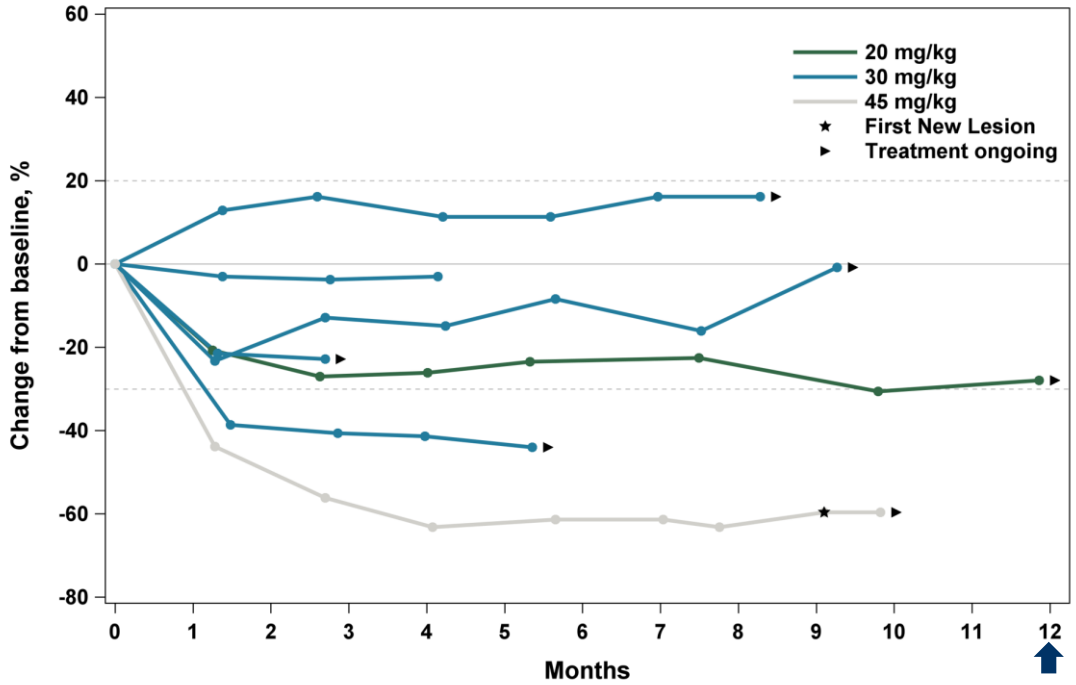
Monotherapy at 20, 30, 45 mg/kg, Q3W

	Aug-2026 Cutoff	2026-ASCO Cutoff
Evaluable patients, n	7	6
ORR, % (n)	42.9% (3/7)	33.3% (2/6)
DCR, % (n)	100% (7/7)	100% (6/6)

CLINICAL CONTEXT

1st-tier recommendation for these patients is clinical trial; Current investigator's choice for later-line nccRCC typically results in ORR ~14%



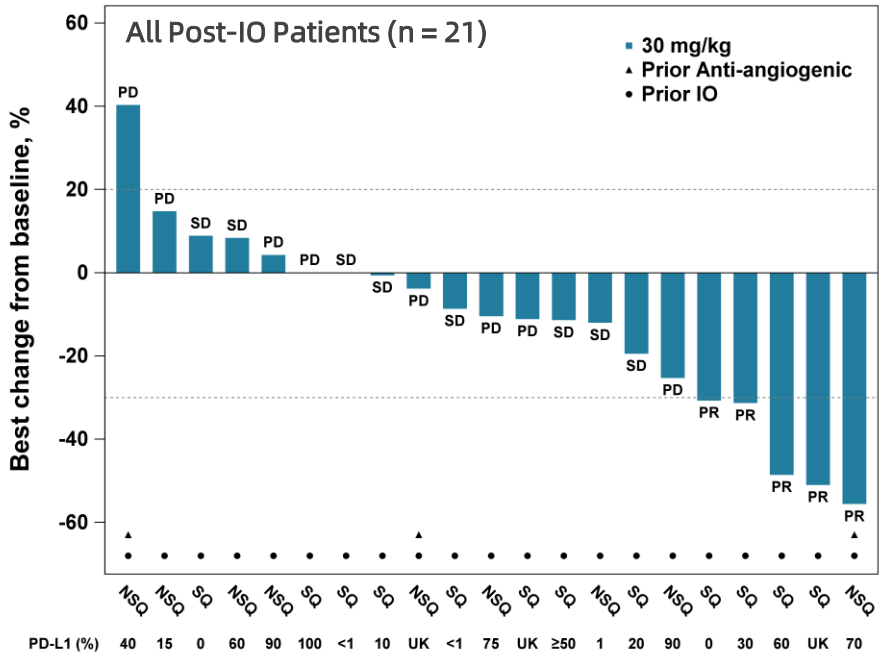


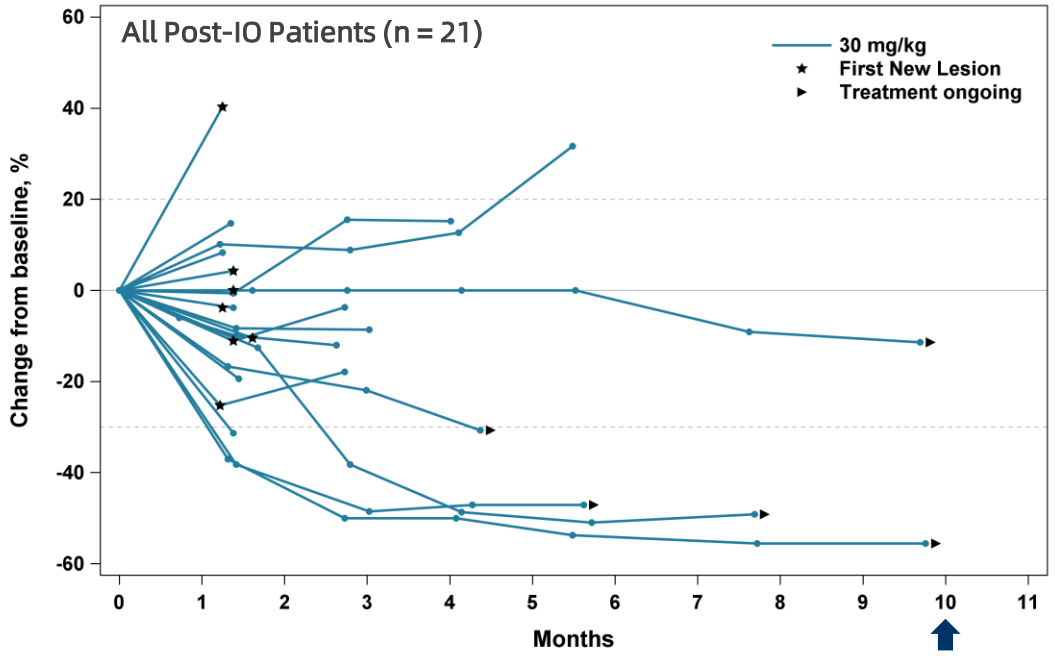
ORR: objective response rate; DCR: disease control rate; IO: immuno-oncology
Data source: In-house Aug-2026 update.

CS2009 monotherapy demonstrated promising clinical efficacy in later-line, post-IO NSCLC

Monotherapy at 30 mg/kg, Q3W

	Aug-2026 Cutoff		2026-ASCO Cutoff	
	ORR*, % (n)	DCR, % (n)	ORR*, % (n)	DCR, % (n)
2L Patients (All treated with I/O plus chemo)	38.5% (5/13)	84.6% (11/13)	30.8% (4/13)	84.6% (11/13)
All Post-IO Patients	23.8% (5/21)	61.9% (13/21)	19.0% (4/21)	61.9% (13/21)





NSCLC: non-small cell lung cancer; L: line; Q3W: every 3 weeks; ORR: objective response rate; DCR: disease control rate; IO: immuno-oncology; NSQ: non-squamous; SQ: squamous
Data source: In-house Aug-2026 update

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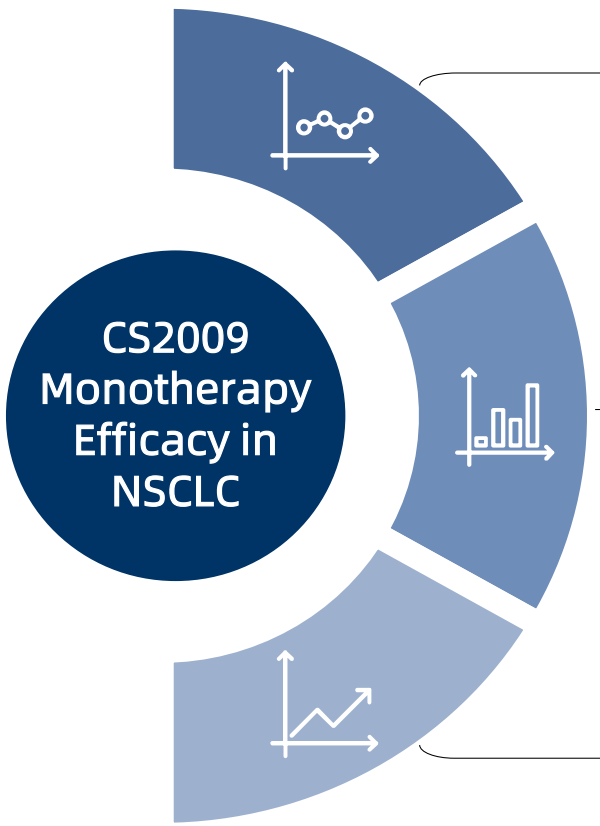
Monotherapy Efficacy in NSCLC Continues Trending Up with Longer Follow-up and Larger Sample Size



	AUG-2026 CUTOFF · LATEST	2026-ASCO CUTOFF
1L NSCLC PD-L1 TPS ≥50% (20 & 30 mg/kg)	• N = 24 • ORR 83.3% (20/24) - At 30 mg/kg: ORR 100% (11/11) • DCR 95.8% (23/24) - At 30 mg/kg: DCR 100% (11/11) • mDoR not reached due to short follow-up	• N = 16 • ORR 81.3% (13/16) • DCR 100% (16/16) • mDoR not reached
1L NSCLC PD-L1 TPS ≥1% (20 & 30 mg/kg)	• N = 47 • ORR 61.7% (29/47) - At 30 mg/kg: ORR 70.8% (17/24) • DCR 93.6% (44/47) - At 30 mg/kg: DCR 91.7% (22/24) • mDoR not reached due to short follow-up	• N = 39 • ORR 53.8% (21/39) • DCR 94.9% (37/39) • mDoR not reached *Note: Undisclosed at 2026 ASCO due to immaturity*
≥2L NSCLC (30 mg/kg)	• N = 25 • ORR 28.0% (7/25) • DCR 60.0% (15/25) • 6-mo DoR rate 83.3% • mDoR not reached due to short follow-up	• N = 25 • ORR 24.0% (6/25) • DCR 60.0% (15/25) • 6-mo DoR rate 80.0% • mDoR not reached

Source: 2026-ASCO presentation & in-house data and Aug-2026 in-house update. n = evaluable patients with at least one post-baseline tumor assessment

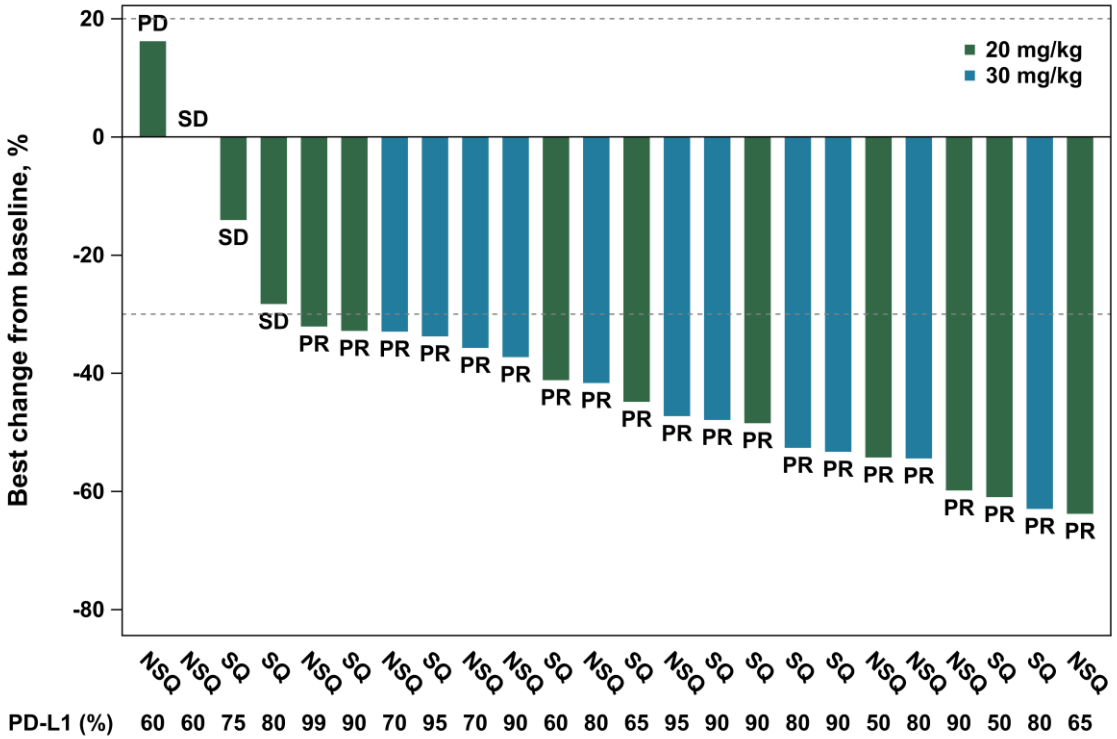
1L NSCLC mono: CS2009 monotherapy demonstrated promising clinical efficacy in first-line NSCLC (PD-L1 TPS ≥50%)

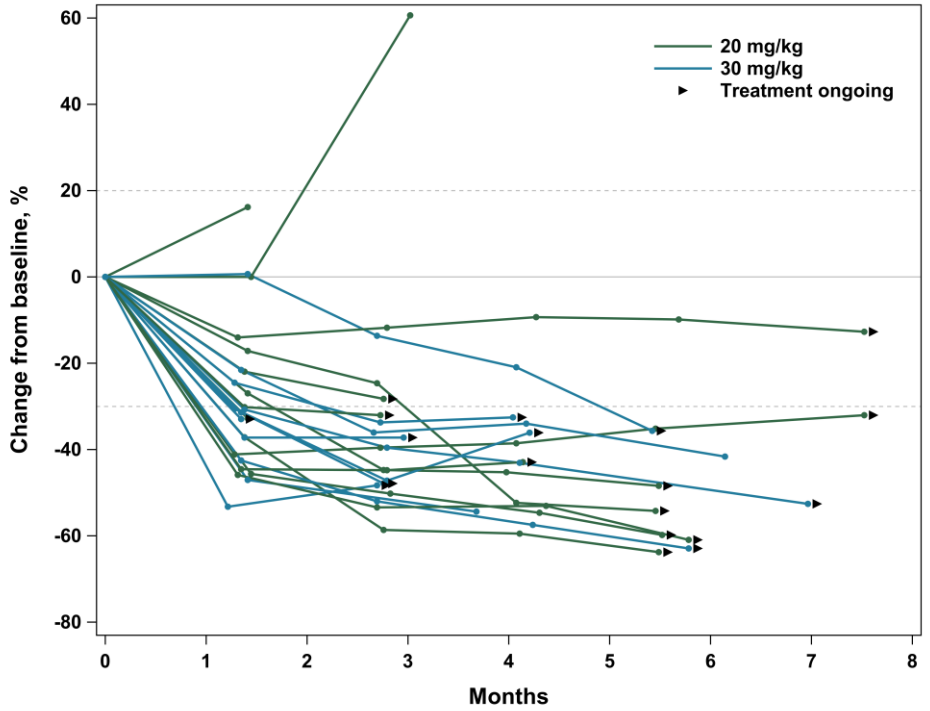
Monotherapy at 20 or 30 mg/kg, Q3W; enrollment completed; median PFS / DoR not reached due to short follow-up

All Evaluable Patients		20 mg/kg		30 mg/kg		Squamous		Non-squamous	
ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)
83.3% (20/24)	95.8% (23/24)	69.2% (9/13)	92.3% (12/13)	100% (11/11)	100% (11/11)	83.3% (10/12)	100% (12/12)	83.3% (10/12)	91.7% (11/12)

▲ n +8, ORR +2.0% vs 2026-ASCO Cutoff

▲ n +5, ORR +16.7% vs 2026-ASCO Cutoff





NSCLC: non-small cell lung cancer; mono: monotherapy; Q3W: every 3 weeks; ORR: objective response rate; DCR: disease control rate; SQ: squamous; NSQ: non-squamous; DoR: duration of response; PFS: progress-free survival
Data source: In-house Aug-2026 update

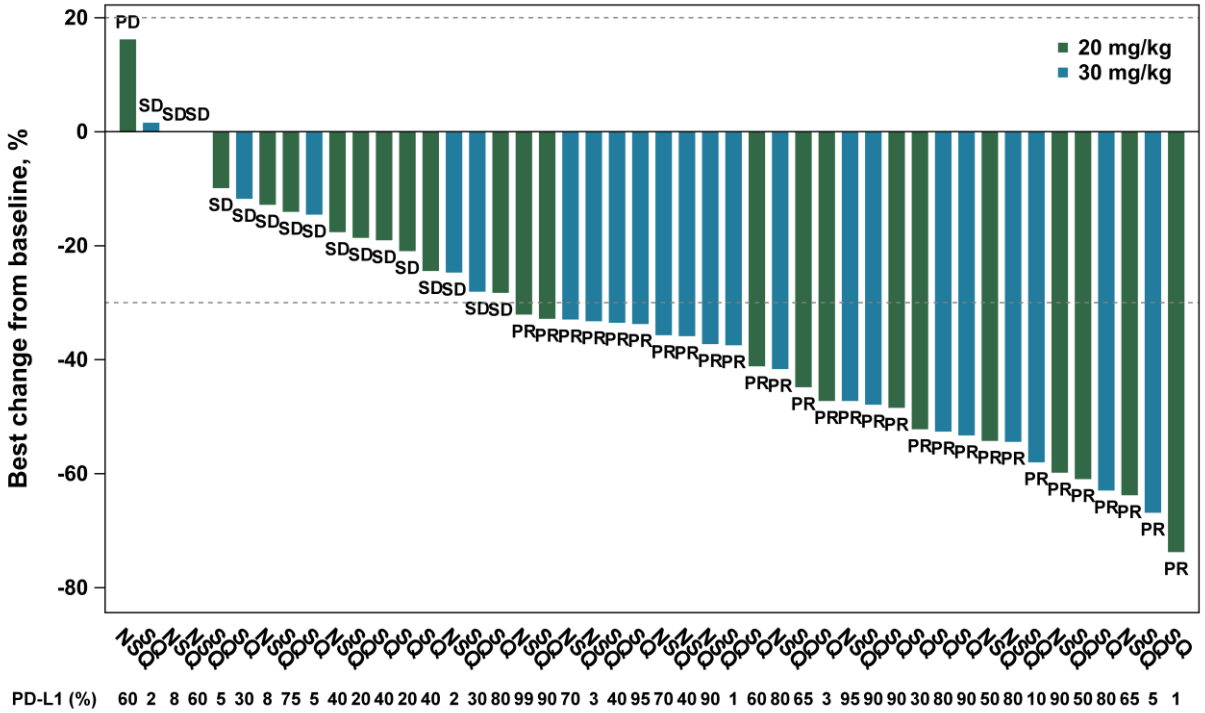
1L NSCLC mono: CS2009 monotherapy demonstrated promising clinical efficacy in first-line NSCLC (PD-L1 TPS ≥1%)

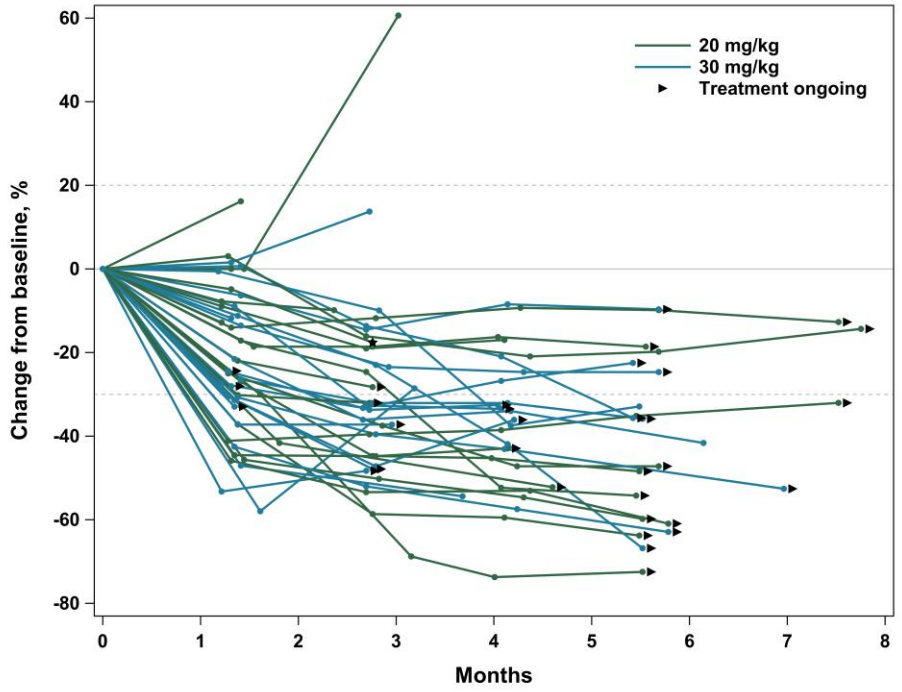
Monotherapy at 20 or 30 mg/kg, Q3W; enrollment completed; median PFS / DoR not reached due to short follow-up

All Evaluable Patients		20 mg/kg		30 mg/kg		Squamous		Non-squamous	
ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)
61.7% (29/47)	93.6% (44/47)	52.2% (12/23)	95.7% (22/23)	70.8% (17/24)	91.7% (22/24)	60.7% (17/28)	96.4% (27/28)	63.2% (12/19)	89.5% (17/19)

▲ n +8, ORR +7.9%
vs 2026-ASCO Cutoff*

▲ n +5, ORR +18.2%
vs 2026-ASCO Cutoff*





*Note: Two patients are not shown. Their overall response was PD due to new lesions, with no target lesion measurements available; * In-house data as of ASCO 2026, which were not previously disclosed given data immaturity*
NSCLC: non-small cell lung cancer; TPS: tumor proportion score; SQ: squamous; NSQ: non-squamous; DoR: duration of response; PFS: progress-free survival
Data source: In-house Aug-2026 update

Higher ORR with CS2009 Monotherapy vs. IO Monotherapy and IO-Based Combo across TPS Subgroups in 1L PD-L1+ NSCLC

	CS2009 (PD-1/VEGF/CTLA-4)		AK112 [3] (PD-1/VEGF)		Pembrolizumab [4] (anti-PD-1)		Nivolumab +/- Ipilimumab [5] (anti-PD-1 +/- anti-CTLA-4)		
Stage of data cited	Phase 2 CS2009-101		Phase 3 HARMONi-2		Phase 3 KEYNOTE-042		Phase 3 CheckMate 227		
Treatment	20 mg/kg, Q3W	30 mg/kg, Q3W	AK112 20mg/kg, Q3W	Pembro. 200mg, Q3W	Pembro. 200mg, Q3W	Platinum-based chemo	NIVO (3 mg/kg, Q2W) + IPI (1 mg/kg, Q6W)	NIVO (240mg, Q2W)	Platinum-based chemo
ORR, % (n) PD-L1 TPS ≥50%	69.2% (9/13)	100% (11/11)	60.2% (50/83)	48.2% (41/85)	39.5% (118/299)	32.0% (96/300)	NA	NA	NA
ORR, % (n) PD-L1 TPS ≥1%	52.2% (12/23)	70.8% (17/24)	50.0% (99/198)	39.0% (78/200)	27.3% (174/637)	26.5% (169/637)	36.4% (144/396)	27.5% (109/396)	29.7% (118/397)

Note: The ORR of **pembrolizumab + chemo** for 1L NSCLC (PD-L1 TPS ≥50%) was **61.4%** (non-squamous, KEYNOTE-189) or **60.3%** (squamous, KEYNOTE-407), **lower than the monotherapy ORR with CS2009** (**83.3%** for non-squamous; **83.3%** for squamous)

NSCLC: non-small cell lung cancer; TPS: tumor proportion score; ORR: objective response rate; Q3W: once every 3 weeks
1. ASCO 2026; 2. Aug-2026 in-house update; 3. WCLC 2025; 4. Lancet 2019; 5. JCO 2022

≥2L NSCLC mono: CS2009 monotherapy demonstrated promising clinical efficacy in heavily pre-treated NSCLC

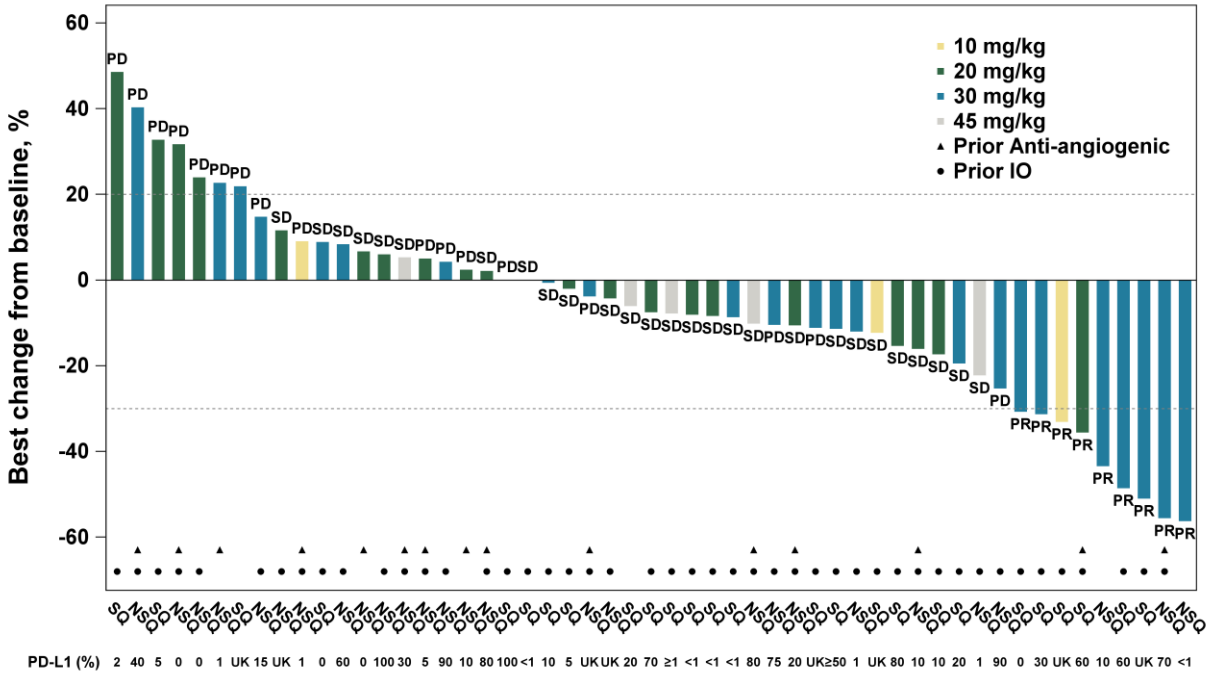
Monotherapy at 10, 20, 30, or 45 mg/kg, Q3W

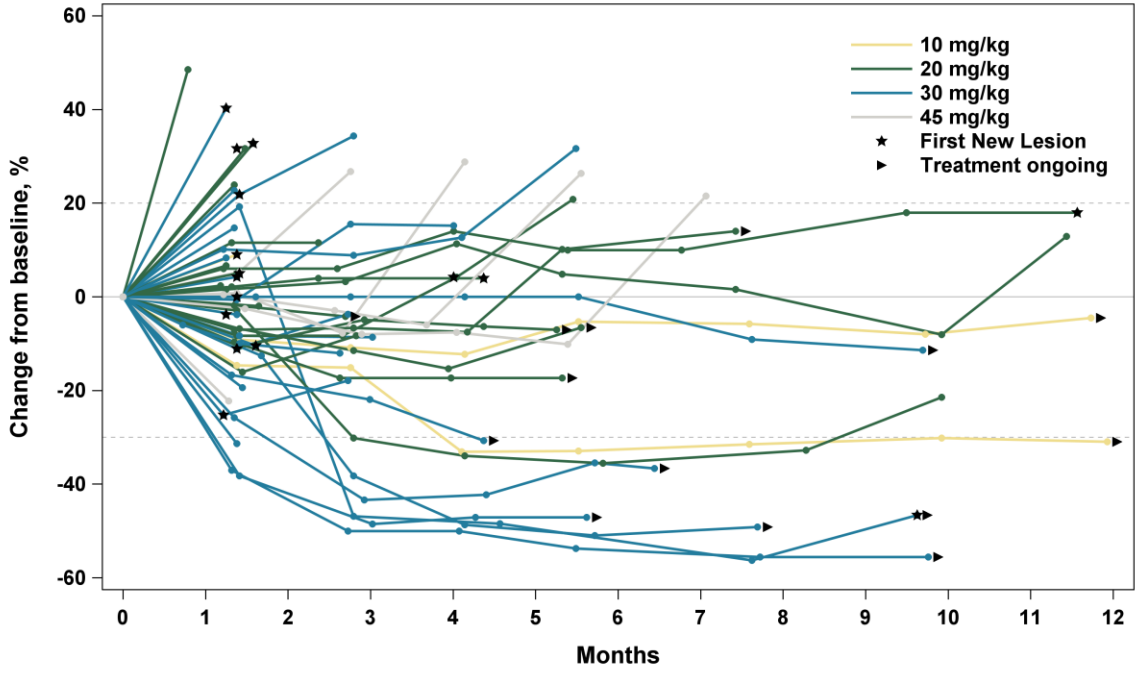
All Evaluable Patients			30 mg/kg			SQ, 30 mg/kg		NSQ, 30 mg/kg	
ORR, % (n)	DCR % (n)	6-mo DoR	ORR, % (n)	DCR % (n)	6-mo DoR	ORR, % (n)	DCR % (n)	ORR, % (n)	DCR % (n)
16.7% (9/54)	68.5% (37/54)	87.5%	28.0%* (7/25)	60.0%* (15/25)	83.3%	30.8% (4/13)	76.9% (10/13)	25.0% (3/12)	41.7% (5/12)

▲ ORR +1.9% vs 2026-ASCO Cutoff

▲ ORR +4.0% vs 2026-ASCO Cutoff

* For 2L patients (n=13, all treated with I/O plus chemo), ORR and DCR were 38.5% (5/13) and 84.6% (11/13)





NSCLC: non-small cell lung cancer; mono: monotherapy; L: line; Q3W: every 3 weeks; ORR: objective response rate; DCR: disease control rate; DoR: duration of response
Data source: In-house Aug-2026 update

Higher Monotherapy ORR with CS2009 vs. Bispecific Antibodies in Heavily Pre-Treated NSCLC

	CS2009[1] (PD-1/VEGF/CTLA-4)	AK104+AK109[2] (PD-1/CTLA-4+VEGF)	AK104+anlotinib[3] (PD-1/CTLA-4+TKI)	AK104[4] (PD-1/CTLA-4)	AK104[5] (PD-1/CTLA-4)	AK112[6] (PD-1/VEGF)	BNT327/PM8002[7] (PD-L1/VEGF)
Stage of data cited	Phase 1/2 In ≥2L AGA(-) NSCLC **30 mg/kg**	Phase 1b/2 in **2L** NSCLC	Phase 1b/2	Phase 1 dose escalation	Phase 1b/2	Phase 1 dose escalation	Phase 1b/2a
Evaluable patients, n	25	47^	6	6	23†	2	8^
ORR, % (n)	**28.0%*** (7/25)	12.8% (6/47)	16.7% (1/6)	0%	0%	0%	12.5% (1/8)
ORR, %: SQ vs. NSQ	**30.8%** vs. **25.0%**	11.5% vs. 14.3%	n/a	0%	0%	0%	n/a
DCR, % (n)	60.0%* (15/25)	95.7% (45/47)	100% (6/6)	33.3% (2/6)	30.4% (7/23)	50% (1/2)	62.5% (5/8)

* For 2L patients (n=13, all treated with I/O plus chemo), ORR and DCR were **38.5%** (5/13) and **84.6%** (11/13)

^ All patients are AGA-negative and 2L post-IO NSCLC

† All patients are AGA-negative/unknown and ≥2L post-IO NSCLC

≥2L: second-/later-line; NSCLC: non-small cell lung cancer; AGA: actionable oncogenic alterations; ORR: objective response rate; DCR: disease control rate; I/O: immuno-oncology; mAb: monoclonal antibody
Note: 1. Aug-2026 in-house update; 2. WCLC 2025 (2L NSCLC); 3. ESMO 2021; 4. Cell Reports Medicine 2023; 5. Lung Cancer 2023; 6. JITC 2024; 7. ASCO 2024

Three Important Key Clinical Validations Achieved from over 300 Patient Data

➢ Proof of Safety

- **Grade ≥3 irAEs 12.7%**; CTLA-4-related tox markedly reduced from CTLA-4 mAb monotherapy or in combination with PD-1/L1, including **extremely rare colitis,** due to **preserved CTLA-4 signaling** in peripheral Tregs & autoreactive T cells
- **Grade ≥3 VEGF-related tox 5.1%;** likely mediated by PD-1/CTLA-4-driven VEGFA immune complex internalization by T cells in TME as indicated by the data

➢ Proof of CTLA-4 Pharmacology & Anti-tumor Efficacy

- Dose-dependent **ICOS upregulation on CD4 T cells**: PD biomarker of CTLA-4 blockade
- Robust activity in **IO-unresponsive "cold tumors ":**
  - CS2009 monotherapy in **later-line mCRC**: ORR 20.0%, DCR 93.3%
  - CS2009 monotherapy in **later-line STS**: ORR 38.5%, DCR 69.2%
  - CS2009 monotherapy in **later-line nccRCC**: ORR 42.9%, DCR 100%
- Robust anti-tumor activity in **later-line IO-refractory/resistant NSCLC:** ORR 23.8%, DCR 61.9%

➢ Proof of Broad Anti-tumor Efficacy beyond Standard IO Regimens

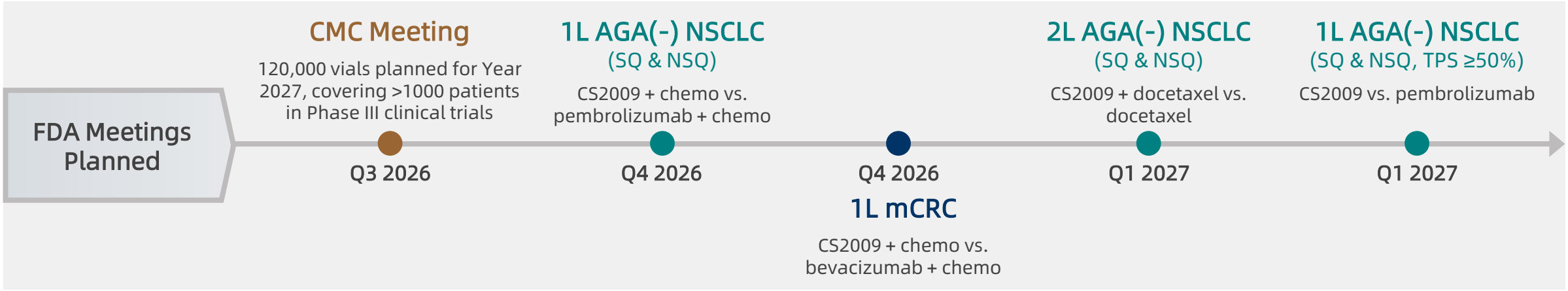
- Robust mono & chemo-combo efficacy in **1L and late-line NSCLC:**
  - CS2009 monotherapy in 1L NSCLC (PD-L1 TPS≥50%): ORR 83.3% (100% at 30 mg/kg), DCR 95.8% (100% at 30 mg/kg)
  - CS2009 monotherapy in 1L NSCLC (PD-L1 TPS≥1%): ORR 61.7% (70.8% at 30 mg/kg), DCR 93.6% (91.7% at 30 mg/kg)
  - CS2009 monotherapy in ≥2L NSCLC (30 mg/kg): ORR 28.0% (38.5% for post-IO 2L patients), DCR 60.0% (84.6% for post-IO 2L patients)
- Robust mono & chemo-combo efficacy in **1L and late-line pMMR/MSS CRC**
- Promising mono activity in mCRPC, OC, TNBC, GC, EC, in addition to STS and nccRCC

TME: tumor microenvironment; ICOS: inducible T-Cell Co-stimulator; NSCLC: non-small cell lung cancer; STS: soft tissue sarcoma; nccRCC: non-clear-cell renal cell carcinoma; pMMR/MSS: proficient mismatch repair / microsatellite stable; CRC: colorectal cancer; mCRPC: metastatic Castration-Resistant Prostate Cancer; OC: ovarian cancer; TNBC: triple-negative breast cancer; GC: gastric cancer; EC: esophageal cancer

Clinical Data and CMC Readiness for FDA Meetings and Phase III Initiation

Clinical Safety & Efficacy Dataset for NSCLC Meetings ▶

- Phase 1 All solid tumors (n≈130)
- Phase 2 1L NSCLC mono (n≈40)
- Phase 1/2 Later-line NSCLC mono (n≈57)
- Phase 2 1L NSCLC combo (n≈60)
- Phase 2 2L NSCLC combo (n≈40)

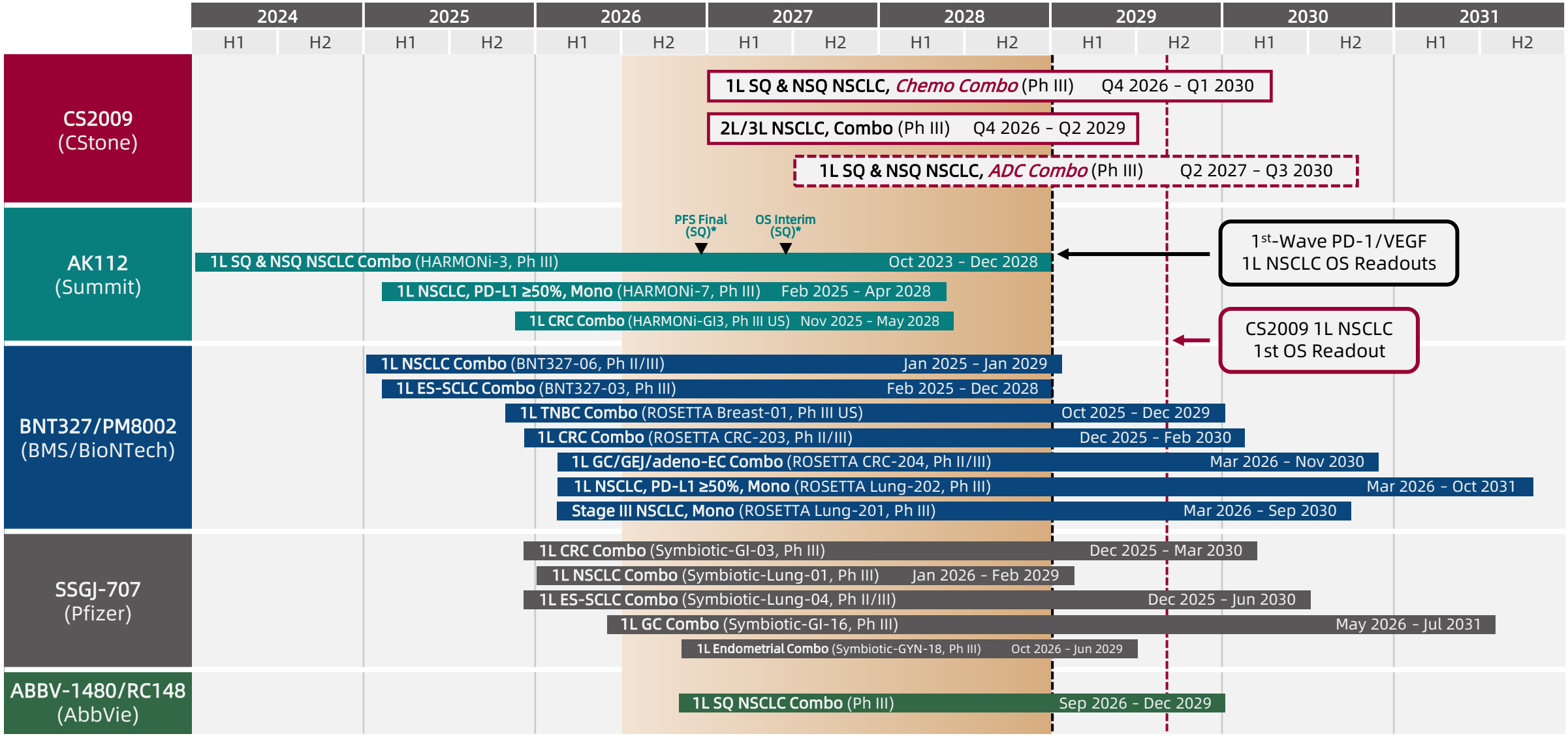


Clinical Safety & Efficacy Dataset for CRC Meeting ▶

- Phase 1 All solid tumors (n≈130)
- Phase 1 Later-line CRC mono (n≈20)
- Phase 2 1L CRC combo (n≈40)

AGA: actionable oncogenic alterations; NSCLC: non-small cell lung cancer; SQ: squamous; NSQ: non-squamous; mCRC: metastatic colorectal cancer; chemo: chemotherapy

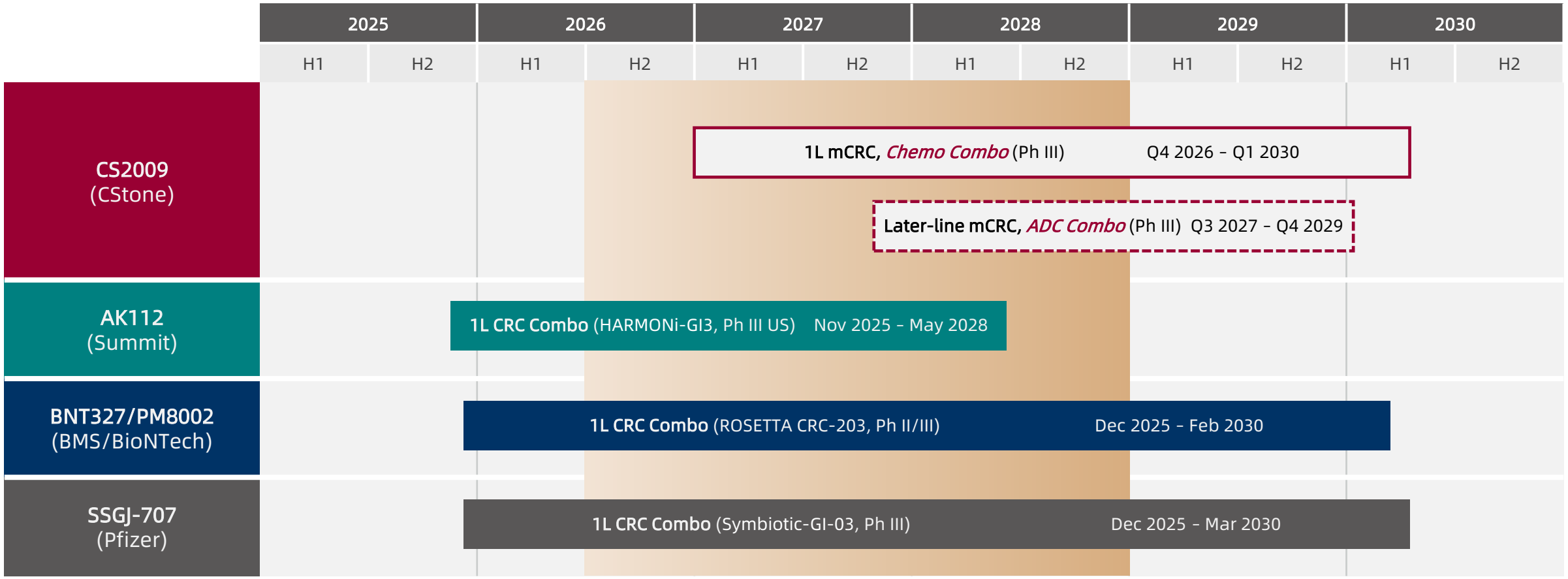
Global Development and Regulatory Window Wide Open for CS2009 Phase III Trials (NSCLC)



AGA: actionable oncogenic alterations; CRC: colorectal cancer; EC: esophageal carcinoma; ES-SCLC: extensive-stage small cell lung cancer; GC/GEJ: Gastric Cancer/Gastroesophageal Junction; NSQ: non-squamous; NSCLC: non-small cell lung cancer; OS: overall survival; PFS: progression-free survival; pMMR/MSS: proficient mismatch repair or microsatellite stable; TNBC: triple-negative breast cancer; SQ: Squamous

** Source: Summit Therapeutics Inc. (SMMT) Q2 2026 Earnings Call July 23, 2026*

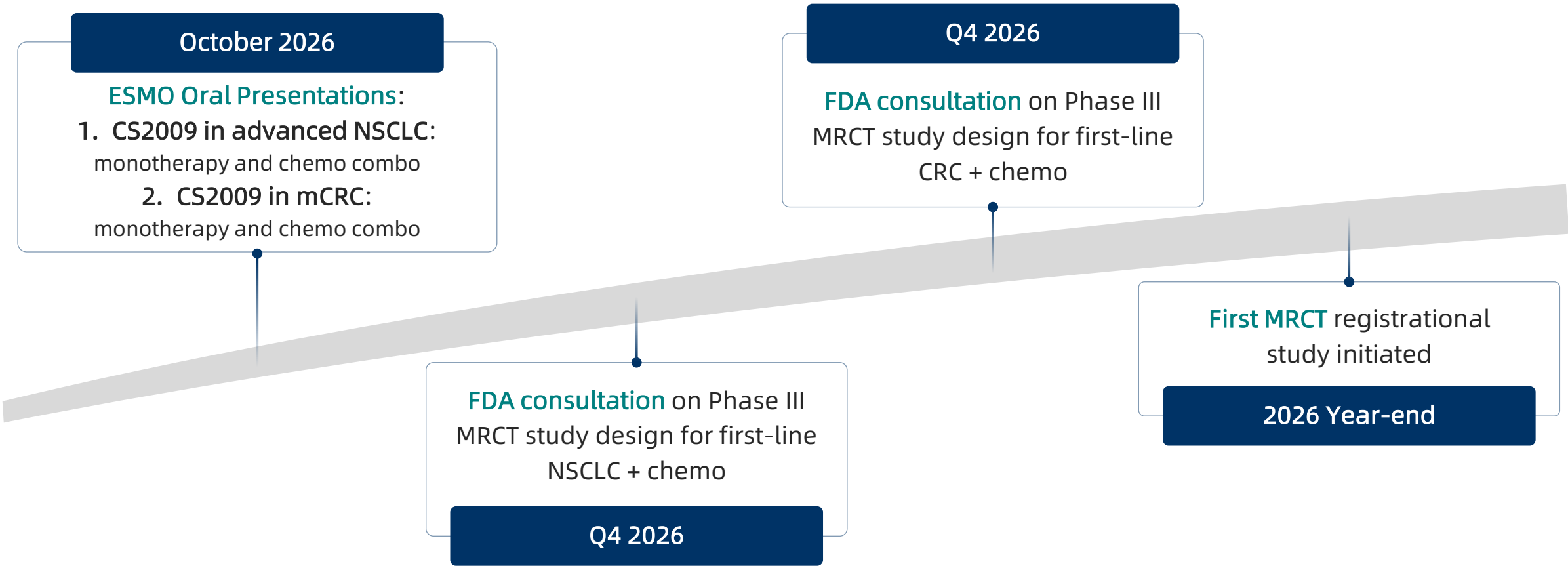
Global Development and Regulatory Window Wide Open for CS2009 Phase III Trials (MSS CRC)



mCRC: metastatic colorectal cancer; MSS: microsatellite stable

CS2009 Upcoming Catalysts

- All pivotal registrational studies are multi-regional clinical trials (MRCTs) using global standard-of-care as control
- Trial design and timeline are not impacted by data readouts from other bispecific/trispecific competitors, giving CS2009 an independent development space and rhythm
- **In-depth discussions with Global MNCs for partnership**

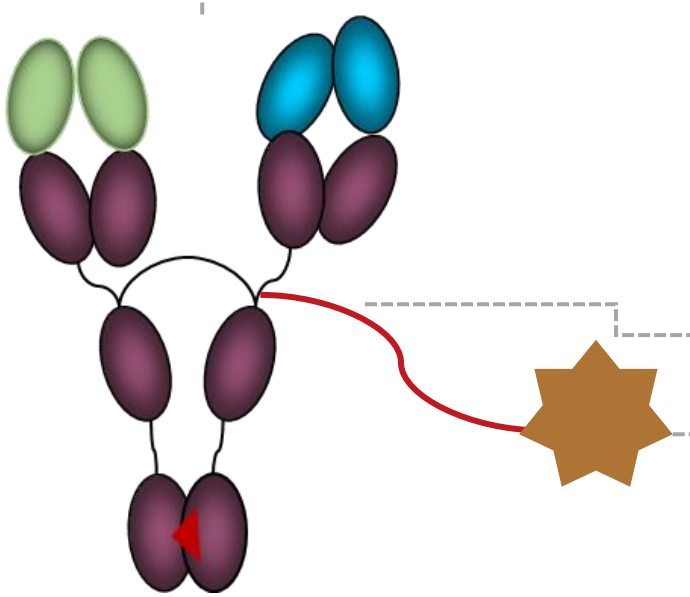


02

Clinical Stage Assets

1. CS2009 (PD-1/VEGF/CTLA-4 trispecific antibody)
2. **CS5007 (EGFR/HER3 ADC)**
3. CS5001 (ROR1 ADC)

CStone has built a modular proprietary antibody drug conjugate (ADC) platform, enabling customized molecular design and screening



Antibody

- Customized target and antibody selections based on clinical needs and antibody features
- **High selectivity and internalization rate**
- To **overcome tumor heterogeneity** (bispecific antibody)

Linker

- **Proprietary** tandem cleavable β-glucuronide linker
- β-glucuronidase/cathepsin B cascade-cleavable linker **enhances tumor selective cleavage**
- **Hydrophilic linker stabilizes ADC** during circulating

Conjugation

- Semi-stochastic attachment with maleimide function group
- Clinical validated
- **Easy to manufacture**

Payload

- Clinical validated payload
- Not a P-gp/BCRP substrate*, with the potential to **overcome cross-resistance to other Topo1 inhibitors**
- **Strong bystander effect**
- Less potential to cause interstitial lung disease (ILD)**

CS5007
(EGFR/HER3 bispecific ADC)

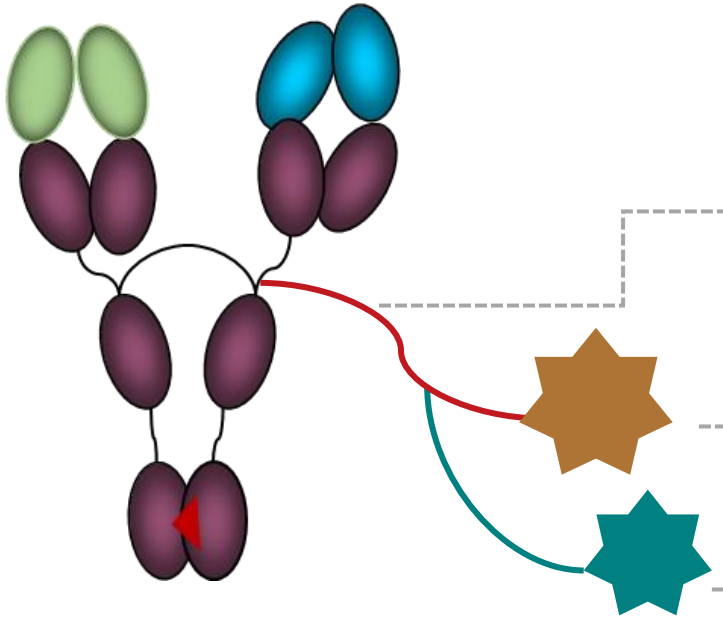
CS5006
(ITGB4 ADC)

CS5008
(DLL3/SSTR2 bispecific ADC)

More innovative ADCs to come…

** Roth JS, et al., 2026; **Sloot WN, et al.,2025*

CStone has also developed a proprietary next-generation ADC platform to support the development of dual-payload ADCs incorporating novel MoA



Antibody

- Customized target and antibody selections based on clinical needs and antibody features
- **High selectivity and internalization rate** (biparatopic antibodies)
- To **overcome tumor heterogeneity** (bispecific antibody)

Linker

- CStone's Proprietary orthogonal **dual-payload-linker platform** enable flexible combo of payloads
- **Tumor selective cleavage** minimizes off-target toxicity
- **Hydrophilic linker** stabilizes ADC during circulating
- **One-pot bioconjugation**

Payload #1

- **Topo1 inhibitor:** clinically validated mechanism with advanced clinical development

Payload #2

- **Clinically validated payload:** distinct mechanism of cytotoxicity from Topo1 inhibitors

OR

- **Novel payload:** mechanistically validated approaches with the potential to synergize with Topo1 inhibitors

Conjugation

- Semi-stochastic attachment with maleimide function group
- Clinical validated
- **Easy to manufacture**

CS5009
(B7H3/PD-L1 bispecific dual-payload ADC)

CS5010
(HER2 biparatopic dual-payload ADC)

CS5012
(HER2 biparatopic novel-payload ADC)

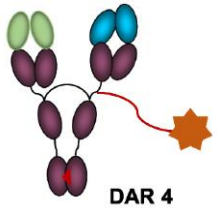
More innovative ADCs to come…

CS5007 (EGFR/HER3 ADC): Potential BIC Candidate for Precision Oncology

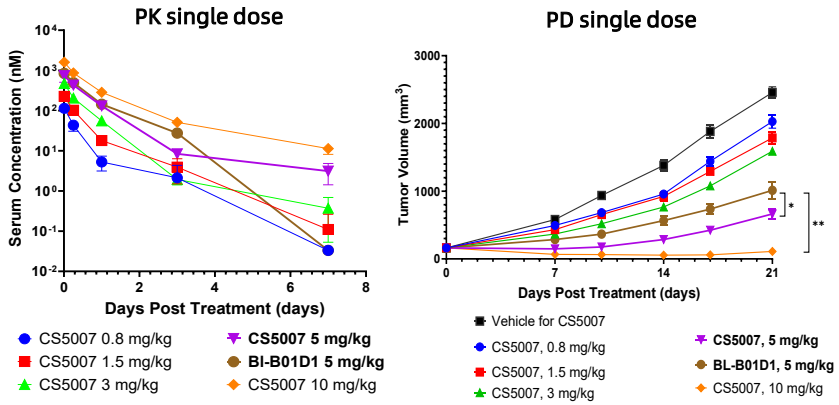
Global multi-center Phase I trial has achieved first-patient enrollment in Australia, with NMPA IND approval secured for China

Built upon CStone's in-House ADC Platform

- **Antibody**: proprietary **EGFR/HER3** bispecific antibody to **address tumor heterogeneity**
- **Linker:** Hydrophilic CSL20 linker with high plasma-stability and **tumor-selective payload release**
- **Payload**: **Clinically validated exatecan** with potent bystander effect and low MDR sensitivity

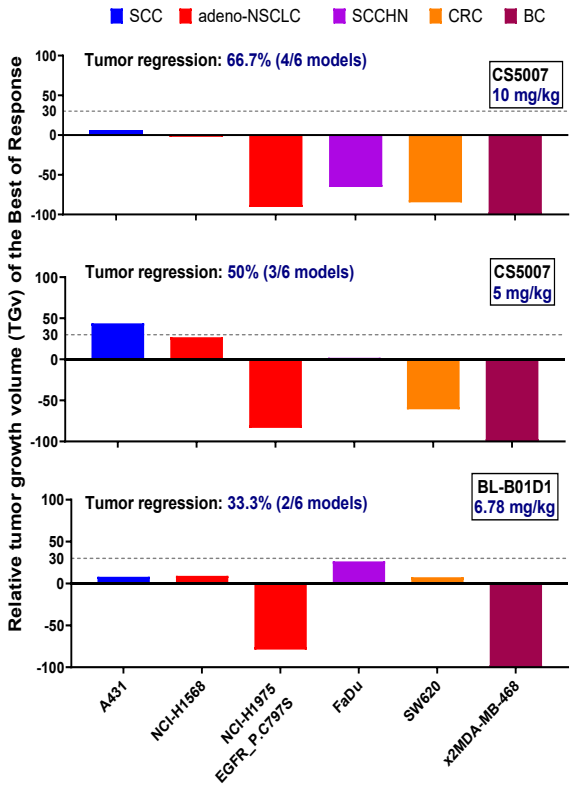


Superior anti-Tumor Activities and PK Profile to BL-B01D1 analog



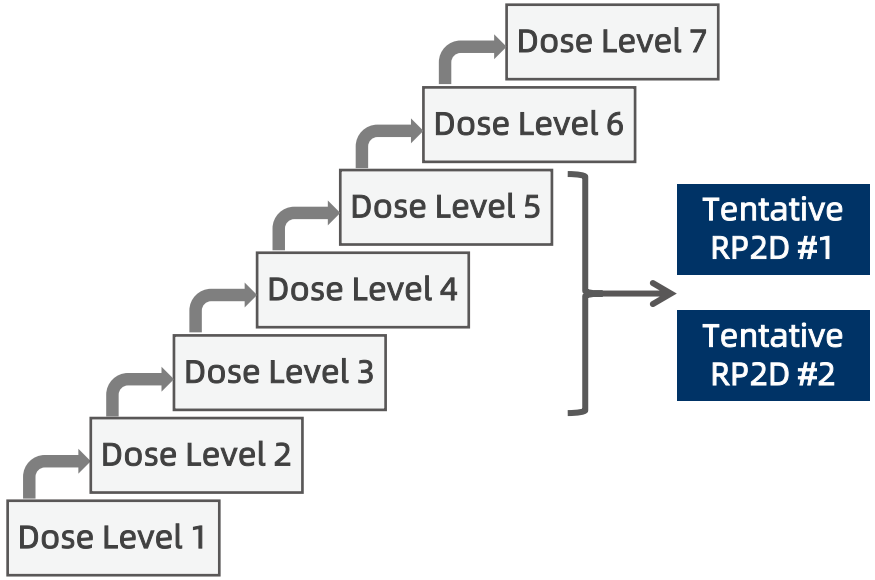
- CS5007 showed **superior PK profile** to BL-B01D1 analog
- At 5 mg/kg level, CS5007 achieved **greater tumor regression** to BL-B01D1 analog in FaDu CDX model^

Robust antitumor activities in various CDX models



- CS5007 inhibited tumor growth on **CDX models derived from diverse tumors** including NSCLC, CRC, BC, and SCCHN

Phase I – Mono Dose Escalation + Backfilling



Phase I Key Eligibility Criteria

- Age ≥18 years
- Pathologically or cytologically confirmed, unresectable advanced solid tumors
- Failed to standard of care for advanced disease, or no available standard of care
- Adequate organ function

*Notes: ^ all treated groups were significantly superior to vehicle group on inhibiting tumor growth. Stats were analyzed by one-way ANOVA; * represents p<0.05, ** represents p<0.001*

02

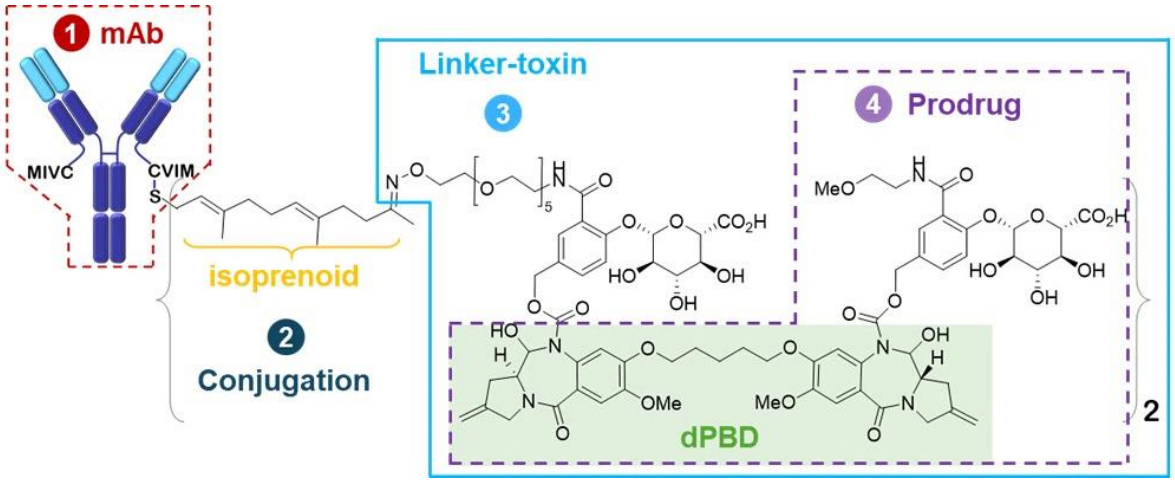
Clinical Stage Assets

1. CS2009 (PD-1/VEGF/CTLA-4 trispecific antibody)
2. CS5007 (EGFR/HER3 ADC)
3. **CS5001 (ROR1 ADC)**

CS5001, a ROR1-ADC with Innovative Design for Better Efficacy & Tolerability

No. 2 position globally with Phase Ib study in Australia and China

Four Key Differentiators Support Best-in-class Potential



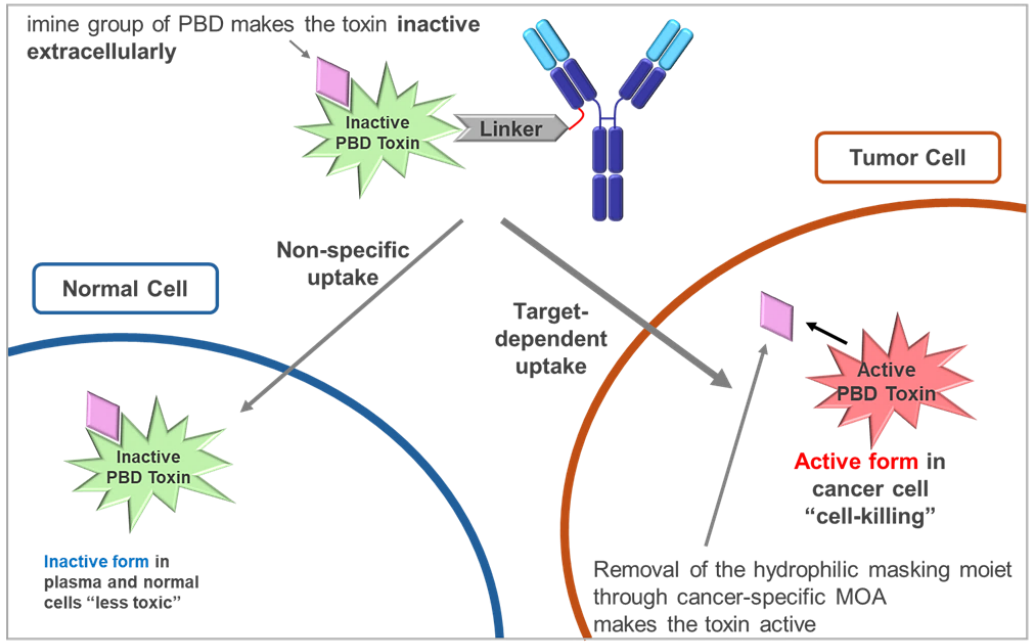
1. Fully human anti-ROR1 IgG1 mAb

2. Site-specific conjugation technology ("ConjuAll") enables a homogenous drug to antibody ratio of 2

3. Proprietary tumor-selective cleavable linker (cleaved by β-glucuronidase) shows exceptional stability in serum

4. Proprietary tumor-activated PBD dimer toxin prodrug (released by β-glucuronidase), with advantages: **a)** much higher potency than MMAE/DXd/Exatecan, etc.; **b)** stronger ability of killing slowly-growing tumors through DNA-crosslinking mechanism than MMAE/DXd/ Exatecan, etc.; **c)** less likely to induce tumor resistance

Novel Prodrug and Linker Technology Minimizes Systemic Toxicity Of Conventional PBD



Free toxin tested	IC_{50} (nM) Tumor cell line 72h	168h
Naked PBD free toxin	1.15	0.04
LCB's proprietary PBD prodrug free toxin	>100	>20

Inactive

Tumor Selective Activation

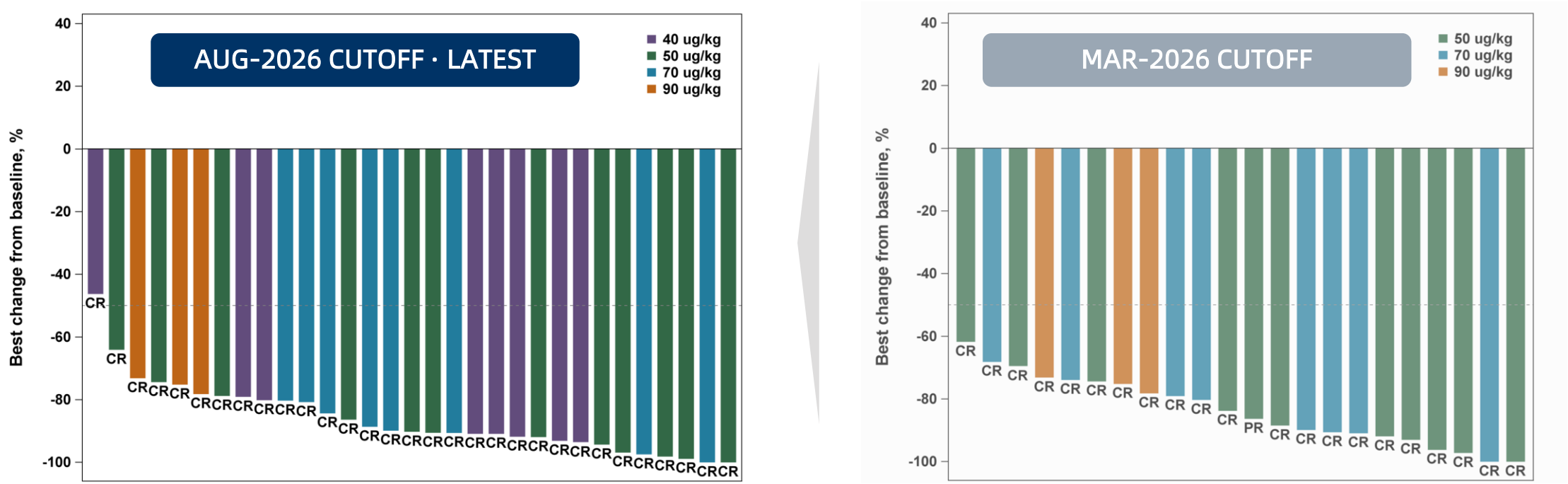
ADCs tested	IC_{50} (nM) Tumor cell line 144h
Naked PBD-ADC	0.23
PBD prodrug-ADC	0.19

Active

CS5001 in Combination with R-CHOP Demonstrated Broad and Deep Antitumor Activity (ORR: 100%, CR: 100%) in 1L DLBCL

Temporarily pausing the trial to evaluate existing clinical data for long-term benefit-risk and to optimize doses and dosing schedule

	All Dose Levels			CS5001 40 μg/kg		CS5001 50 μg/kg			CS5001 70 μg/kg		CS5001 90 μg/kg	
	ORR*, % (n)	CR*, % (n)	PR*, % (n)	ORR*, % (n)	CR*, % (n)	ORR*, % (n)	CR*, % (n)	PR*, % (n)	ORR*, % (n)	CR*, % (n)	ORR*, % (n)	CR*, % (n)
Aug-2026 Cutoff	100% (31/31)	100% (31/31)	-	100% (8/8)	100% (8/8)	100% (12/12)	100% (12/12)	-	100% (8/8)	100% (8/8)	100% (3/3)	100% (3/3)
Mar-2026 Cutoff	100% (22/22)	95.5% (21/22)	4.5% (21/22)	-	-	100% (11/11)	90.9% (10/11)	9.1% (1/11)	100% (8/8)	100% (8/8)	100% (3/3)	100% (3/3)



** Per Lugano 2014, overall response is primarily based on PET-CT; CT alone is used only when PET is unavailable. For example, a patient in the 40 ug/kg cohort achieved overall CR despite a target-lesion SPD decline <50%, as all target lesions (in this case lymph nodes) had normalized in size on CT and PET confirmed complete metabolic response.*

1L – first-line; DLBCL – diffuse large B-cell lymphoma; ORR – objective response rate; CR – complete response; PR – partial response

Source: In-house data as of March & August 2026

03

Early Assets

Preclinical/IND-Enabling Programs

Pipeline 2.0 Preclinical Assets: Highly Differentiated ADCs and Mono-/Multi-Specific Antibodies

		Discovery	Preclinical Development	IND-Enabling
ADCs (Oncology)				
CS5006 (ITGB4 ADC)	Solid tumors			
CS5008 (SSTR2/DLL3 bispecific ADC)	Solid tumors			
CS5009 (B7H3/PD-L1 bispecific dual-payload ADC)	Solid tumors			
CS5010 (HER2 biparatopic dual-payload ADC)	Solid tumors			
CS5012 (HER2 biparatopic novel-payload ADC)	Solid tumors			
Antibodies (Oncology)				
CS1012 (GDF-15 antibody)	Solid tumors			
Antibodies (Immunology & Inflammation)				
CS2013 (BAFF/APRIL bispecific antibody)	Immunology & Inflammation			
CS2015 (OX40L/TSLP bispecific antibody)	Immunology & Inflammation			
CS2016 (TL1A/α4β7 bispecific antibody)	Immunology & Inflammation			
CS1016 (PD-1 agonist antibody)	Immunology & Inflammation			

04

Commercial Products

1. CEJEMLY® (sugemalimab), anti-PD-L1 antibody
2. Pralsetinib, RET inhibitor
3. Avapritinib, KIT/PDGFRA inhibitor

CEJEMLY® (sugemalimab): Expanded Global Indication and Market, and Increased Guideline Recognition

Recognition by Global Oncology Community

Included in **Non-Oncogene-Addicted Metastatic NSCLC Living Guideline** (Feb. 2025) for **both squamous and non-squamous Stage IV NSCLC** (Evidence level I, Recommendation grade A)

Included in **Early and Locally Advanced NSCLC Living Guideline** (March 2026) for **Stage III NSCLC** (Evidence level I, Recommendation grade A)

NSCLC: non-small-cell lung cancer; ENKTL: extranodal NK/T-cell lymphoma; ESCC: esophageal squamous cell carcinoma; G/GEJ: gastric and gastroesophageal junction

CEJEMLY® (sugemalimab): Robust Multi-Indication Clinical Evidence Base Published in Leading Journals

Clinical Trial Results Published in a Series of Top-Tier Journals

CEJEMLY® (sugemalimab): Global Commercialization Driven by Strategic Partnerships

CEJEMLY® international footprint now extends to over 60* countries and regions worldwide

** Commercial partnerships with Ewopharma in Switzerland and 18 Central Eastern European countries; with Pharmalink in 12 Middle East and South Africa countries; with SteinCares in 10 Latin American countries; with Istituto Gentili in 23 countries including 18 EEA countries and the UK, Andorra, Monaco, San Marino, and Vatican City; with Arrotex in Australia and New Zealand*

Pralsetinib Capsules: First Shipment of Locally-Manufactured Product

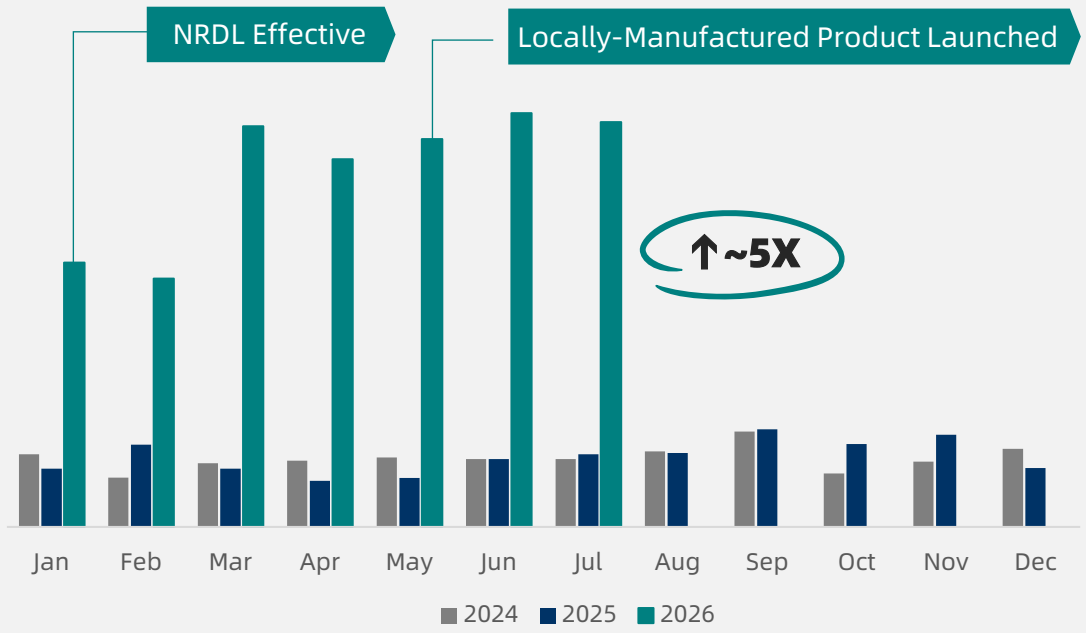
- First domestically manufactured batch of pralsetinib capsules shipped in May 2026, transitioning from import-dependent to local supply for national commercial distribution – following NRDL inclusion effective Jan 2026
- Tech transfer and process validation completed; comparability data demonstrate locally manufactured product is quality- and efficacy-equivalent to the imported product, meeting all regulatory requirements for commercial release
- Domestic manufacturing is expected to reduce COGS vs. import, improve supply reliability, and support margin expansion as commercial volume scales post-NRDL

Pralsetinib & Avapritinib: Sales Growth Following NRDL and Local Supply Launch

Following its inclusion in the NRDL effective Jan. 1, 2026, Pralsetinib's in-market sales volume increased by almost 500% yoy

Pralsetinib

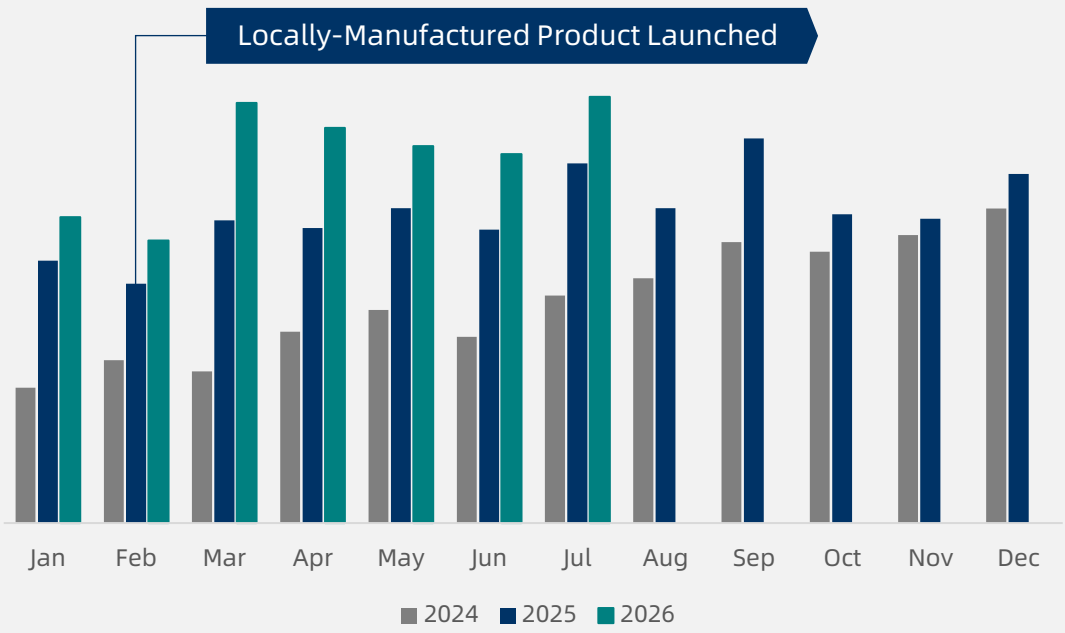
Pralsetinib In-market Sales Volume
Trends from 2024 to July 2026 (Packs)



Based on the localized supply implementation since Feb. 2025, Avapritinib has demonstrated steady sales growth

Avapritinib

Avapritinib In-market Sales Volume (converted to 300mg)
Trends from 2024 to July 2026 (Packs)



05

Financial Highlights

2026 H1 Financial Results

Robust cash position to support the rapid development of key pipeline assets and commercial strategies execution

Mn RMB	2026 H1	2025 H1	Change
GROUP REVENUES	*205.1*	*49.5*	*315%*
Sales of Pharmaceutical Products	183.2	20.2	806%
License Fee Income	6.9	17.9	-62%
Royalty Income	15.0	11.3	33%
OPERATING EXPENSES (Non-IFRS[1] Measures)	*(308.6)*	*(179.3)*	*72%*
Research and development expenses (Non-IFRS[1] Measures)	(193.4)	(102.1)	89%
Selling, marketing and admin expenses (Non-IFRS[1] Measures)	(115.2)	(77.2)	49%
OTHER INCOMES/ OTHER GAINS AND LOSSES	*(11.4)*	*13.9*	*-182%*
Other incomes	16.0	9.3	72%
Other gains and losses	(27.4)	4.6	-700%
(LOSS) PROFIT FOR THE YEAR (Non-IFRS[1] Measures)	*(230.9)*	*(265.1)*	*-13%*

Mn RMB	30 June 2026	31 December 2025	Change
CASH BALANCE[2]	*1,560.2*	*918.7*	*70%*

Revenue of RMB205.1 mn

- Revenue growth was primarily driven by a post-NRDL sales surge of pralsetinib, effective January 2026

Commitment to R&D

- R&D expenses increased, driven by clinical trial costs associated with the CS2009 Phase I/II study and costs for IND activities of other early pipeline programs

Solid liquidity position of RMB1,560.2 mn

- April 2026 public offering: net proceeds of RMB923.0 mn

[1] IFRS: International Financial Reporting Standards. Non-IFRS Measures represents the (loss) profit for the period excluding the effect of certain non-cash items and onetime events, namely the share-based payment expenses;
[2] Cash balance includes cash and cash equivalents, and time deposits with original maturity over three months.

Q&A

Appendix

Well-balanced portfolio of 16 innovative assets (1/2)

- Commercial/Late-stage Programs

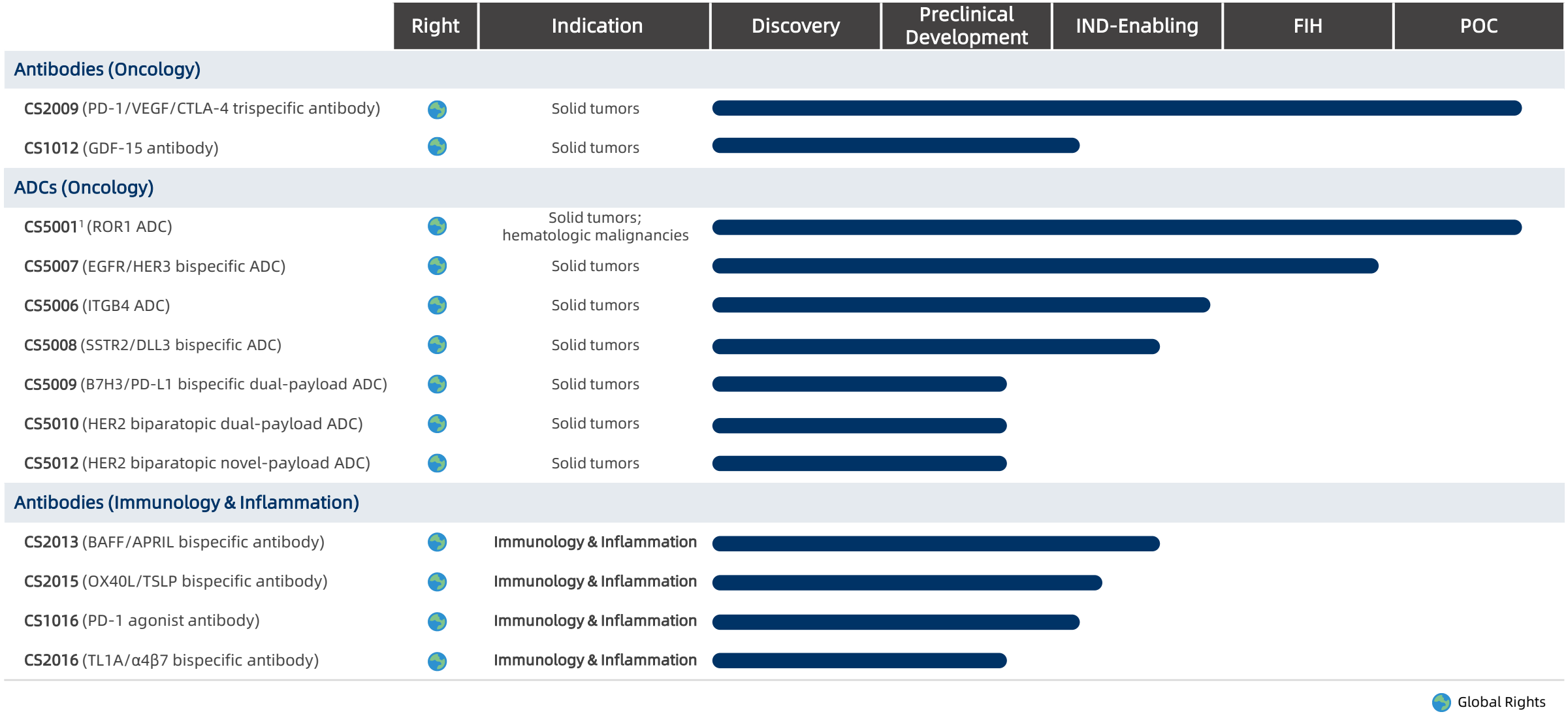
Asset	Indication	POC	Pivotal	NDA	Marketed	Approval						Partners	Partnering Regions
						CN	TW	HK	US	EU	UK		
Pralsetinib (RET)	1L NSCLC	■	■	■	■	✓	✓	✓				blueprint MEDICINES a sanofi company	◀ Mainland China
	2L NSCLC	■	■	■	■	✓	✓	✓					
	NSCLC	■	■	■	■				✓				
	TC	■	■	■	■				✓			rigel	
	Multiple tumors	■											
Avapritinib (KIT/PDGFRA)	PDGFRA exon 18 GIST	■	■	■	■	✓	✓	✓	✓			blueprint MEDICINES a sanofi company	◀ Mainland China
	PDGFRA D842V GIST	■	■	■	■					✓			
	ISM[1]	■	■	■	■				✓	✓			
	Advanced SM[1]	■	■	■	■				✓	✓	✓		
Sugemalimab (PD-L1)	1L Stage IV NSCLC	■	■	■	■	✓				✓	✓	Pfizer	◀ Mainland China
	Stage III NSCLC	■	■	■	■	✓				✓	✓	ewo pharma	◀ Switzerland and CEE
	1L G/GEJ	■	■	■	■	✓						PHARMALINK	◀ Middle East and Africa
	1L ESCC	■	■	■	■	✓						STEINCARES	◀ Latin America
	R/R ENKTL	■	■	■	■	✓						GENTILI	◀ West Europe and the UK
												arrotex PHARMACEUTICALS	◀ Australia & New Zealand
CS1002 (CTLA-4)	Solid tumors	■	■										◀ Greater China

CN = Mainland China, TW = Taiwan, China, HK = Hong Kong SAR, China, US = United States, EU = European Union, UK = United Kingdom, POC = Proof of Concept, NDA = New Drug Application, NSCLC = Non-small Cell Lung Cancer, TC = Thyroid Cancer, GIST = Gastrointestinal Stromal Tumor, ISM = Indolent Systemic Mastocytosis, Advanced SM = Advanced Systemic Mastocytosis, G/GEJ = gastric/gastroesophageal junction adenocarcinoma, ESCC = Esophageal Squamous Cell Carcinoma, R/R = Relapsed or Refractory, ENKTL = Extranodal NK/T Cell Lymphoma, CEE=Central & Eastern Europe

1. POC study was conducted in North American and Europe. No clinical trials have been conducted in China while IND application is under preparation

Well-balanced portfolio of 16 innovative assets (2/2)

– Pipeline 2.0



Note: Assets status denotes progress in the region(s) noted in the column titled "Rights" ; FIH = First in Human, POC = Proof of Concept,
1. CStone obtains the exclusive global right from LigaChem Biosciences, Inc. (LCB) to lead development and commercialization of LCB71/CS5001 outside the Republic of Korea

Trusted Global Partner — Proven Track Record Across Continents

基石药业 CSTONE PHARMACEUTICALS

Sugemalimab (PD-L1)	Pralsetinib (RET)	Avapritinib (KIT/PDGFRA)	Ivosidenib (IDH1)
Pfizer	blueprint MEDICINES a sanofi company; rigel	blueprint MEDICINES a sanofi company	agios
ewo pharma since 1959	ALLIST	HENGRUI	SERVIER moved by you
PHARMALINK			
STEINCARES			
GENTILI			
arrotex PHARMACEUTICALS			

CS5001 (ROR1)	Nofazinlimab (PD-1)	CS1002 (CTLA-4)
LigaChemBio	三生制药 3SBIO INC.	HENGRUI

Regorafenib Co-D	CS2006 (PD-L1/4-1BB/HSA)	Discovery Collaboration
BAYER	NUMAB	DotBio

CStone has built a strong track record of successful partnerships with leading multinational corporations and biotech companies worldwide.

CStone's Leadership Depth and Expertise Translates Science into Patient Value

Jason Yang, MD, PhD
Chief Executive Officer & President of R&D

Pfizer · AMGEN · COVANCE · BeiGene

Michael Choi, MBA
Chief Business & Strategy Officer

sparc · Pfizer · IQVIA · life technologies

Qingmei Shi, MD, PhD
Chief Medical Officer

COVANCE · parexel

Yujuan La, PhD
Head of Product Development

宝船生物 DRAGON BOAT BIOPHARMACEUTICAL · Mab-Venture Your Partner for Biologics · Shanghai Jiao Tong University

Ye Shi, MS
Head of Quality

AsymBio · CMAB · 齐鲁制药 QILU PHARMACEUTICAL

Weicong (Nicky) Ni, MBA, CFA
Chief Financial Officer & Company Secretary

Deutsche Bank · Harvard Business School

Yinghua Zhang, MS
Chief Operating Officer

先声药业 Simcere · 先声默沙东 SMSD

Qinzhou Qi, PhD
Head of R&D Management & Chief of Staff

BeiGene · Northern Illinois University