CRESCENT BIOPHARMA

Crescent Biopharma Overview

April 2025

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Crescent Biopharma aims to advance the next wave of innovation in cancer therapy

Crescent's pipeline consists of **potentially best-in-class therapies for the treatment of solid tumors**

~$200 million financing in October 2024 anticipated to fund operations through 2027[2]

PROGRAM	MOA	DISCOVERY	IND-ENABLING	IND	POTENTIAL INDICATIONS
CR-001[1]	**PD-1 x VEGF** same cooperative MoA as ivonescimab			4Q25	NSCLC, other solid tumors
CR-002	**Undisclosed** ADC #1 with TopoI payload			Mid-2026	Solid tumors
CR-003	**Undisclosed** ADC #2 with TopoI payload				Solid tumors

Crescent is the fifth company launched with assets discovered and developed in-house by Paragon Therapeutics, **an antibody discovery engine** founded by Fairmount Funds in 2021

Prior companies founded **using** Paragon**'s engineered antibody technology** have collectively raised >$2B and generated significant value

1. Anticipated expiration for filed provisional patent is 2045+
2. Financing scheduled to close immediately prior to the closing of the merger with GlycoMimetics

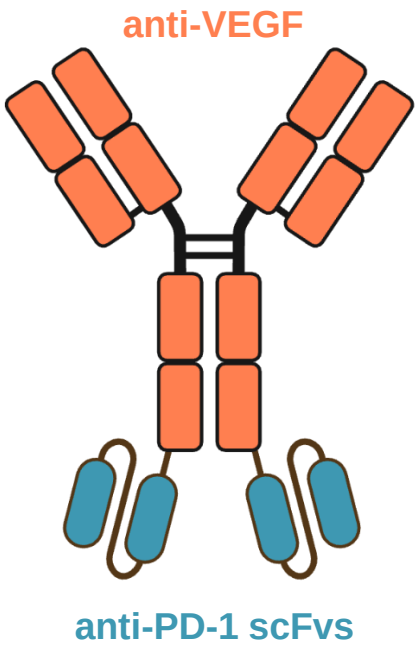
Notes: NSCLC: Non-small cell lung cancer. MoA: Mechanism of action. ADC: Antibody-drug conjugates. TopoI: Topoisomerase

Crescent is advancing three highly impactful oncology programs with best-in-class potential

CR-001

PD-1 x VEGF cooperative tetravalent bsAb

Same MoA as ivonescimab



Designed to **reproduce ivonescimab's established pharmacology**

Pipeline-in-a-program opportunity across solid tumor indications

Potential **to move to frontline use in the $50B+** PD-(L)1 immunotherapy market

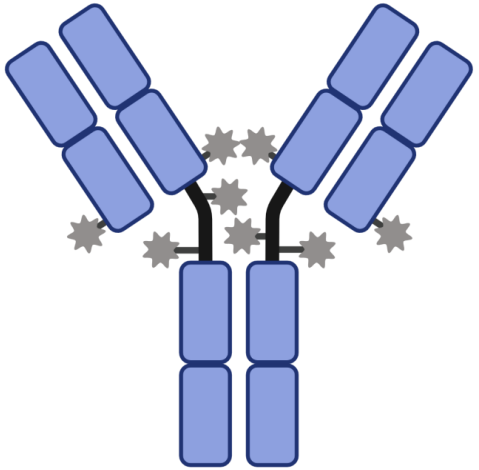
IND expected 4Q25

Interim PoC data expected 2H26

CR-002 & CR-003

ADCs with topoisomerase inhibitor payloads

Potentially best-in-class



Two unique, undisclosed targets with **significant potential across solid tumors** as single agents

Each has potential **to synergize with CR-001** in combination studies, further driving clinical efficacy

Both utilize the **best-in-modality cytotoxic payload**: topoisomerase inhibitor

CR-002 IND expected mid-26

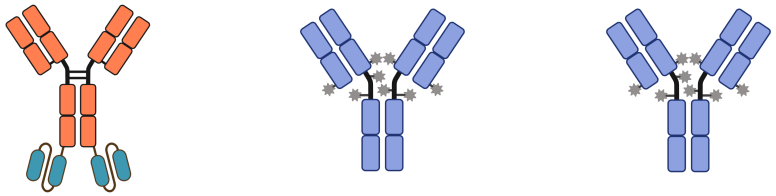
Interim PoC data expected 2027

Notes: bsAb: Bispecific antibody. IND: Investigational New Drug application. PoC: Proof-of-concept

Multiple ways to win: Crescent pipeline enables optionality with differentiating combination therapies

Optimized Novel Monotherapies

CR-001 CR-002 CR-003



Synergistic Combination Approaches

CR-001 + CR-002 CR-001 + CR-003

Engineered for:

- Best-in-class efficacy
- Efficacy across histologies
- Pharmacokinetics
- Safety
- Stability
- Developability

Selected for:

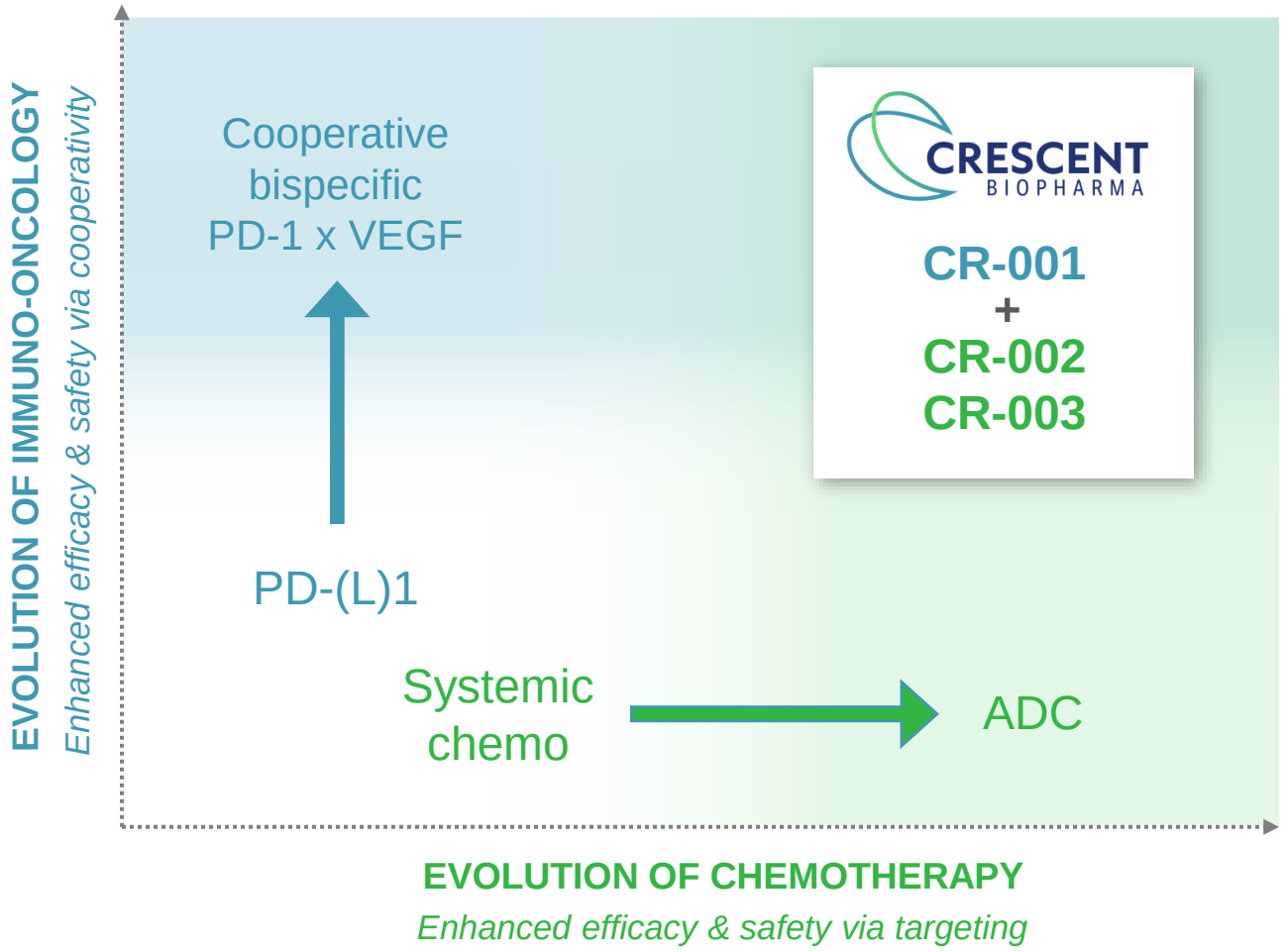
- MoA synergy
- Efficacy in overlapping histologies
- Broad utility in solid tumors

Crescent leverages three key advances in oncology for next-generation combinations within unique portfolio

Three revolutions underway in oncology:

- Immuno-oncology is potentially moving from PD-(L)1 to cooperative PD-1 x VEGF
- Chemo is moving from systemic toxins to tumor-targeted toxins via improved ADCs
- Monotherapies are being replaced by synergistic combinations of targeted therapies

Crescent is developing leading assets in both categories, designed to combine for maximum efficacy in priority indications

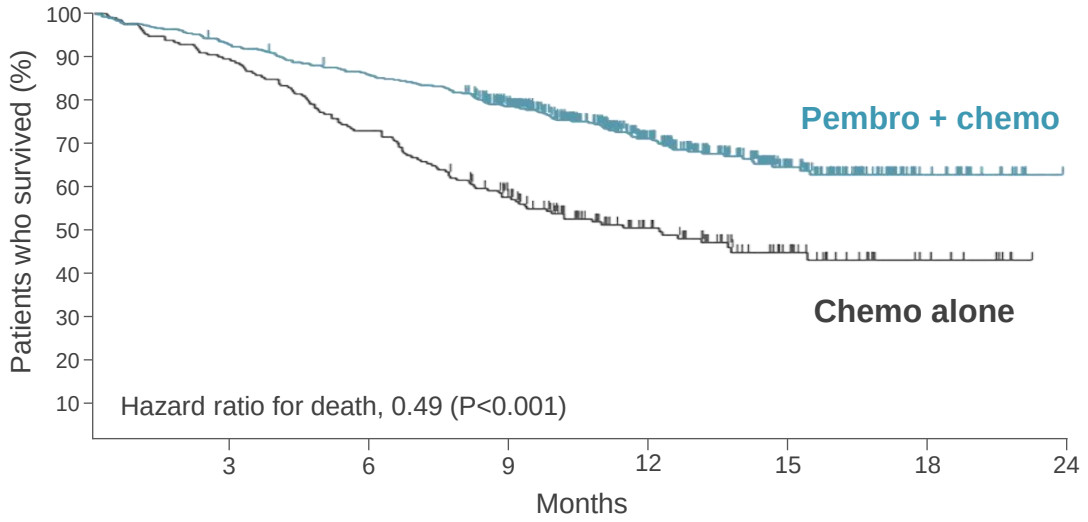


PD-(L)1-targeted therapies, annualizing $50B+, have transformed oncology – with Keytruda now the best-selling drug in the world

PD-(L)1 inhibitors have significantly prolonged cancer survival, shifting 1L treatment to immunotherapy

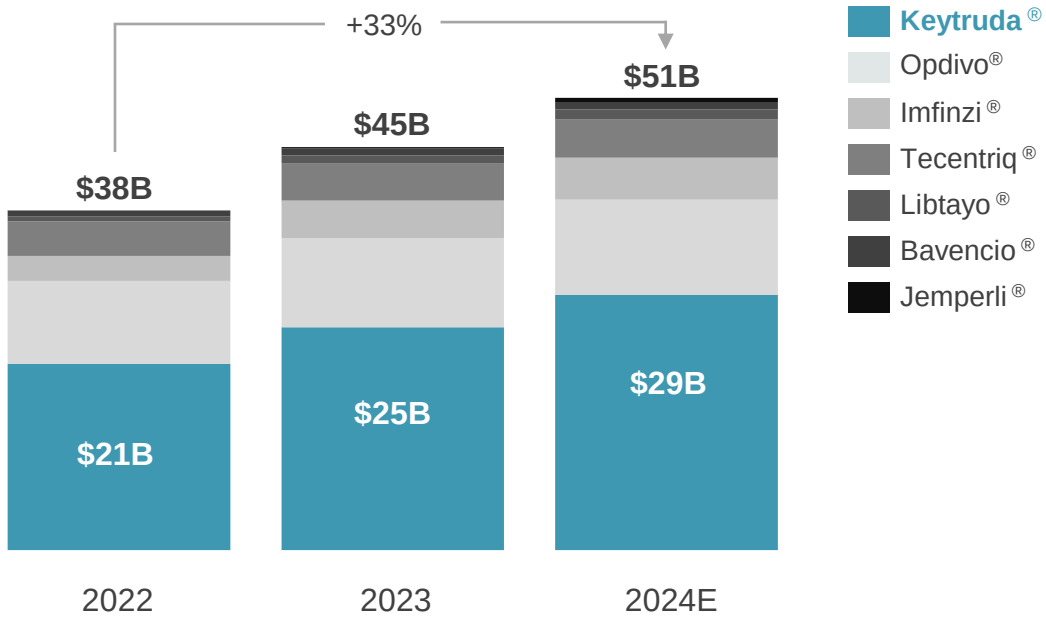
Example

In 1L NSQ NSCLC, addition of pembrolizumab to chemo **significantly improved mOS** (**NR vs 11.3 months**[1] with **HR 0.49**)



PD-(L)1-targeted therapy is one of the largest drug classes, with Keytruda (pembrolizumab) the dominant player

Anti-PD-(L)1 global sales



Keytruda alone is **approved in 20+ oncology indications** with expected revenue of **~$30B in 2024**

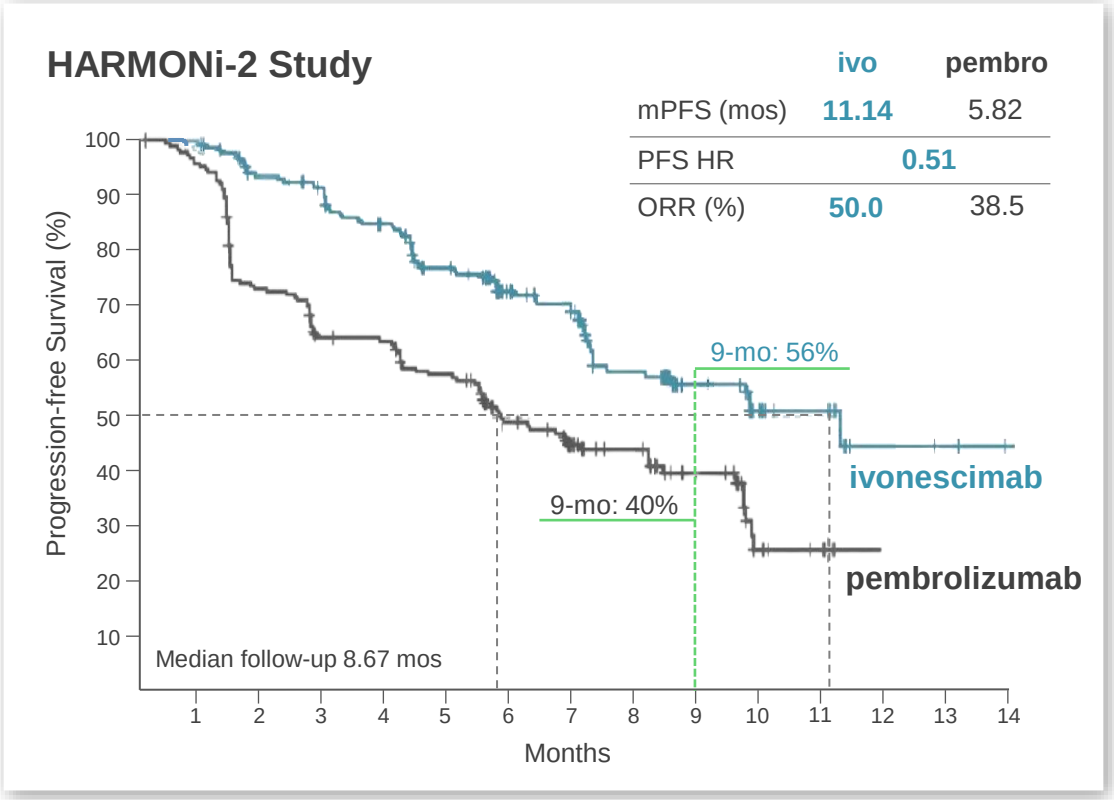
1. 5-year follow-up demonstrated mOS of 22.0 vs 10.6 months.

Notes: 1L: First-line. NSQ: Non-squamous. NSCLC: Non-small cell lung cancer. mOS: median overall survival. NR: Not reached. HR: Hazard ratio.

Sources: 2018 Gandhi (NEJM); 2023 Garassino (J Clin Oncol); GlobalData; FactSet; Pembrolizumab FDA Label

Ivonescimab, a cooperative PD-1 x VEGF bispecific, doubled progression-free survival vs. Keytruda in a P3 NSCLC trial

Ivonescimab is the first drug to demonstrate superiority in PFS over pembrolizumab in a randomized Phase 3



Ivonescimab's novel mechanism of action raises the bar on efficacy and safety

Broader Efficacy

Ivonescimab demonstrates benefit in patients where anti-PD-(L)1 efficacy has historically been modest (e.g., squamous, PD-(L)1^{low})

	PD-L1^{low} (TPS 1-49%)	PD-L1^{high} (TPS ≥50%)	Non-squamous	Squamous
HR	**0.54**	**0.46**	**0.54**	**0.48**

Promising Safety

Ivonescimab had **lower AEs than expected** vs. anti-VEGF monotherapy. This suggests a **differentiated profile** due to cooperativity-driven tissue targeting

Dual blockade of PD-1 and VEGF through a cooperative bispecific antibody has led to unprecedented clinical results, demonstrating superiority to pembrolizumab and a **$15B+ market cap for ivonescimab's ex-China sponsor, Summit Therapeutics**

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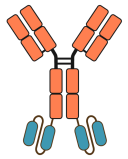
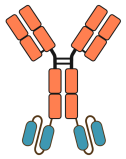
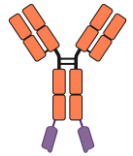
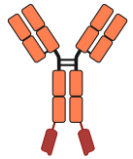
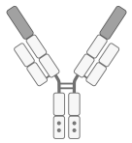
Notes: HR: hazard ratio. PFS: progression-free survival. AE: adverse event. NSQ: Non-squamous SQ: Squamous. ORR: Objective response rate. Akeso Biopharma has licensed ivonescimab to Summit in North America, South America, Europe, Africa, Middle East, and Japan. Akeso maintains rights in Asia (ex-Japan / Middle East) and in Oceania. **Sources:** 2024 Zhou (WCLC Presentation on HARMONi-2); Summit Therapeutics; 2018 Paz-Area (NEJM); 2019 Mok (Lancet); 2022 De Castro Jr (J Clin Oncol); Avastin® Label

CR-001 is one of the few programs intentionally designed to exhibit ivonescimab-like cooperative pharmacology

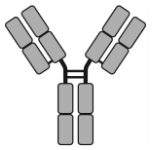





	Anti-PD-1 scFv-based		Anti-PD-1 VHH-based	Anti-PD-L1 VHH-based
Program	CR-001	ivonescimab	LM-299	BNT327 / PM8002
Company	CRESCENT BIOPHARMA	Summit therapeutics, Akesobio	MERCK, LaNova 礼新医药	BIONTECH, BIOTHEUS 普米斯生物技术
Stage	Preclinical	Phase 3 (Global)	Phase 1/2 (China)	Phase 3 (Global)
Anti-VEGF IgG	Bevacizumab	Bevacizumab	Bevacizumab	Bevacizumab
Anti-PD-(L)1	Anti-PD-1 scFvs	Penpulimab scFvs	Novel anti-PD-1 VHHs	Novel anti-PD-L1 VHHs
Fc function	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs	Fc null, to avoid potential AEs
Cooperative pharmacology	✓	✓	Expected (not disclosed); unclear impact of VHH structure	Expected (not disclosed); unclear impact of PD-L1 VHH

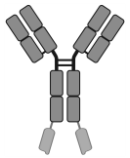
Examples of alternative constructs



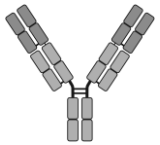
- Anti-PD-L1 IgG with enhanced ADCC
- VEGF trap



- Anti-PD-1 mAb with off-target VEGFR2 binding through same variable domains



- Anti-PD-1 IgG
- Novel anti-VEGF VHHs
- Inverted format



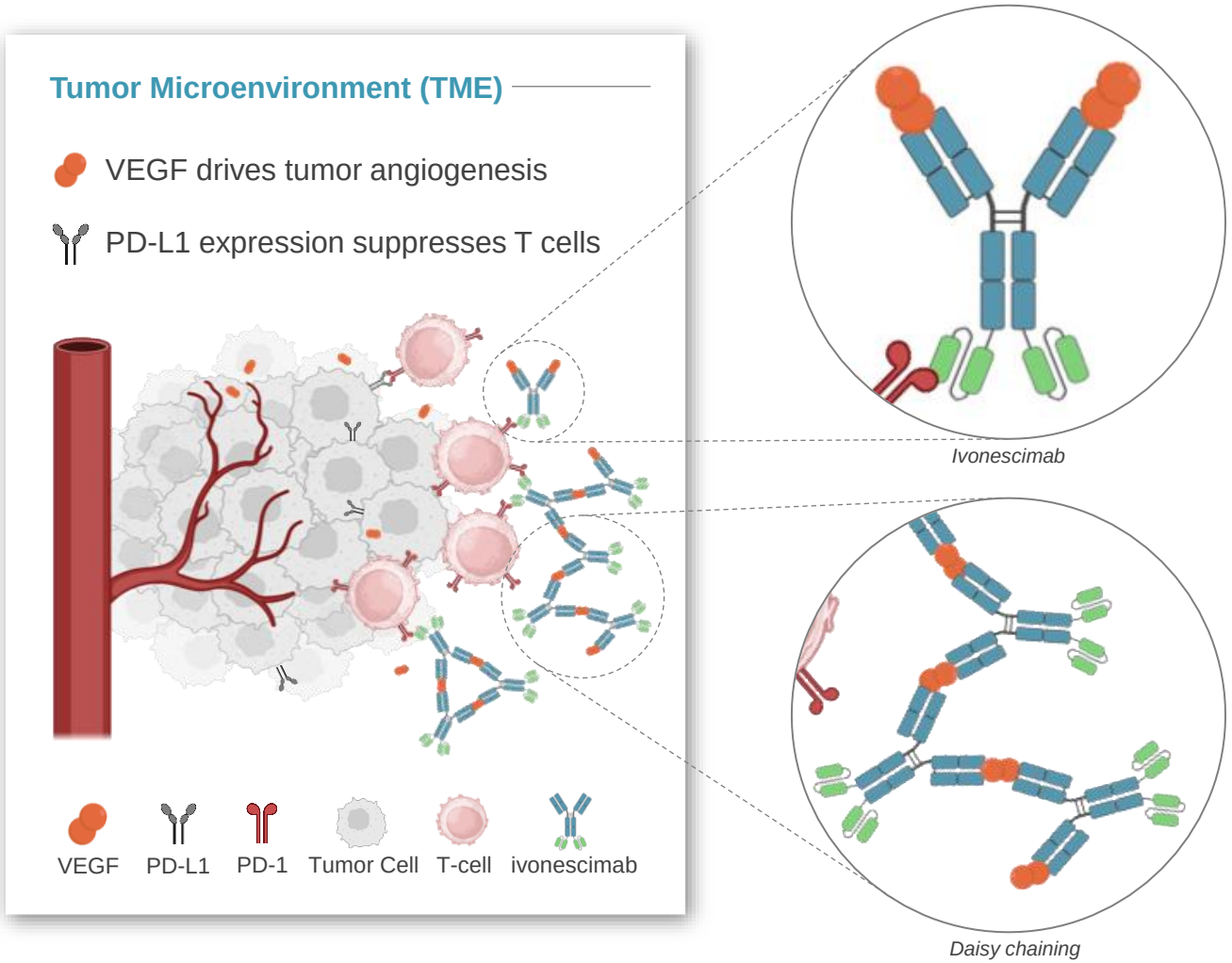
- Bevacizumab
- Anti-PD-1 Fabs
- PD-1 domains attached to IgG N-terminus instead of C-terminus

Sources: Internal data; Summit Therapeutics 2023 SITC Poster; BioNTech 2024 ESMO Presentation; LaNova patent filings; Various patent filings; 2017 Lee (Scientific Reports); 2007 Rudge (PNAS)
Notes: VHH: Variable heavy chain domain antibody.

CR-001

Cooperative, tetravalent
PD-1 x VEGF bispecific antibody

Ivonescimab's novel, cooperative MoA is hypothesized to drive enhanced anti-tumor activity while maintaining tolerability



Tumor Targeting

Dual blockade of PD-1 and VEGF through a novel tetravalent bispecific format with cooperative binding effects has led to **unprecedented clinical results** in third party trials

PD-1 arm concentrates VEGF inhibition in the TME, potentially **sparing healthy tissue and reducing AEs**

Cooperativity

Ivonescimab's **cooperative binding** blocks PD-1 / PD-L1 interactions *and* inhibits VEGF

VEGF binding to ivonescimab increases affinity to PD-1 and vice versa, enhancing both T-cell activation and VEGF-signaling blockade. This helps explain the **cross-trial outperformance** of ivonescimab vs. an anti-PD-L1 + anti-VEGF combination

PD-1 binding strength (affinity) is increased by >18x in the presence of VEGF

Sources: 2023 Zhong (SITC Poster); Summit Therapeutics

CR-001 is a highly potent PD-1 x VEGF bsAb designed to recapitulate ivonescimab's cooperative pharmacology

Same design as ivonescimab

Pairs anti-VEGF IgG & anti-PD-1 scFvs

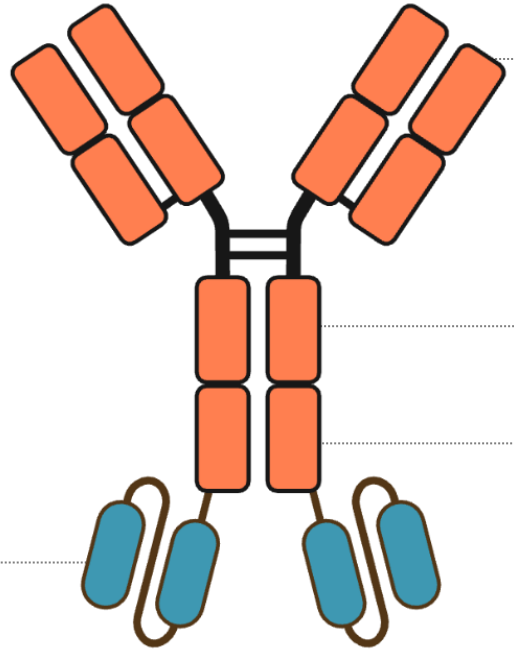
Avoids risks of alternative, clinically unprecedented constructs (e.g., VEGF trap, anti-PD-L1 IgG, ADCC)

Highly potent & stable scFvs

Designed to be the best possible anti-PD-1 epitope / binding domain

Anti-PD-1s have historically outperformed anti-PD-L1s in meta-analyses of solid tumor studies

Contains proprietary engineering to enable functional and stable scFvs



CR-001

Potential for reduced AEs

Cooperative binding increases anti-VEGF activity in TME, reducing AE risks in healthy tissue

Identical VEGF potency to preserve safety

Effector-null human IgG Fc

Equivalent to ivonescimab

ADCC carries additional AE risk

Designed to match ivonescimab PK

Native FcRn binding to match distribution and elimination of ivonescimab

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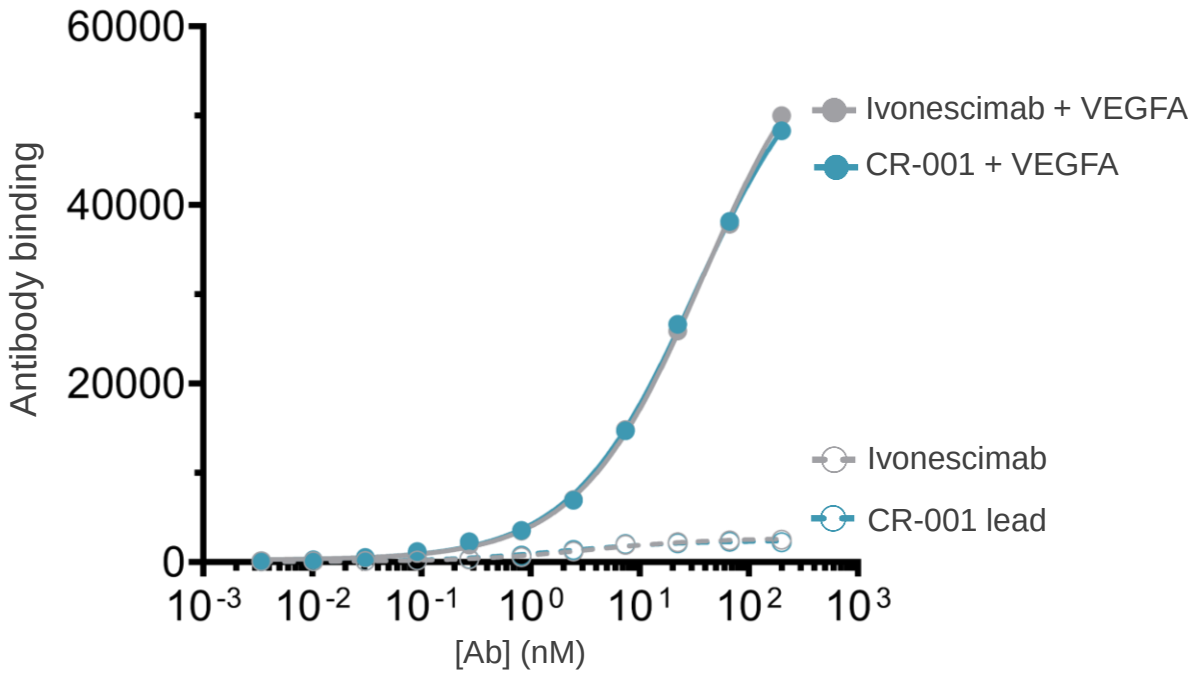
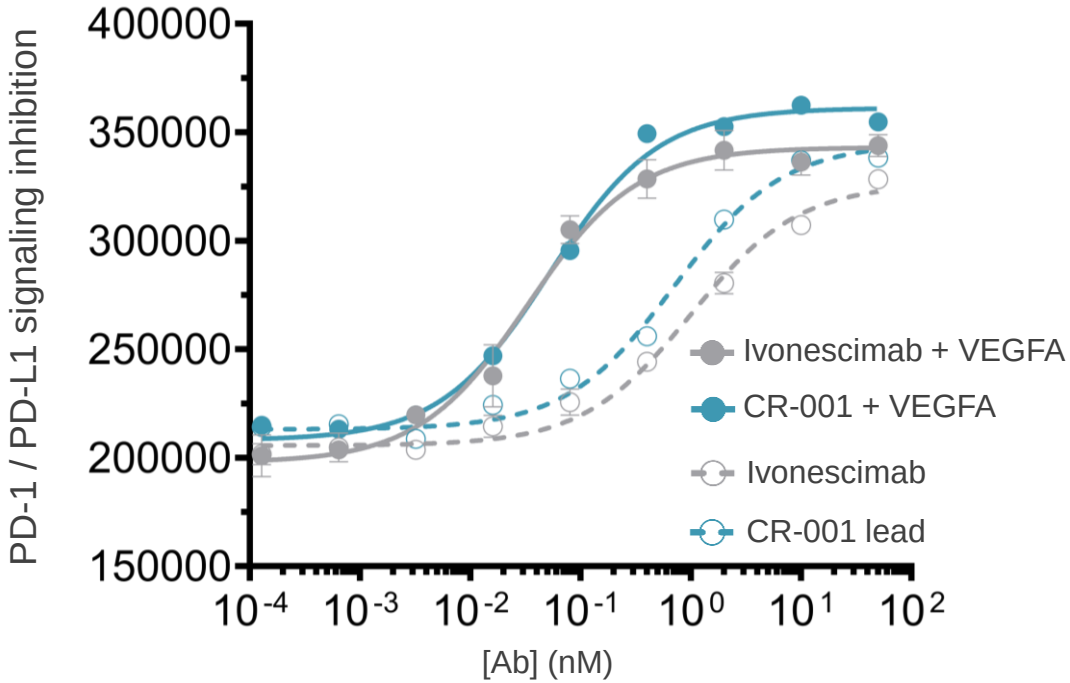
Notes: IgG: Immunoglobulin G. scFvs: Single-chain variable fragment. ADCC: Antibody-dependent cell-mediated cytotoxicity; PK: Pharmacokinetics. FcRn: Neonatal Fc receptor.

CR-001 replicates ivonescimab's cooperative binding effect which leads to cooperative inhibition of PD-1 signaling in presence of VEGF

CR-001, like ivonescimab, **increases PD-1 binding** on PD-1+ Jurkat cells **in the presence of VEGF**…



…leading to **higher potency** in an NFAT reporter assay in **the presence of VEGF**.



CR-001 lead demonstrates *same cooperative effect* as ivonescimab across multiple assays

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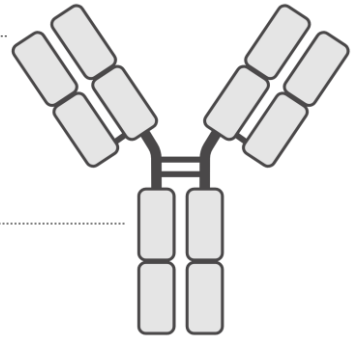
Notes: Ivonescimab generated internally based on published sequence. PD-1 / PD-L1 signaling inhibition measured in RLU (relative light units), a measure of luminescence that increases with greater inhibition. PD-1 binding measured in MFI (mean fluorescence intensity), a measure of fluorescence that increases with binding and is measured via FACS. NFAT: Nuclear factor of activated T cells. **Sources:** Internal data

CR-001 engineering replicates ivonescimab function with biophysical properties that maximize flexibility in development

Standard mAbs can be improved with **established protein engineering approaches…**

CDRs improved via diversification and/or affinity maturation to maximize potency

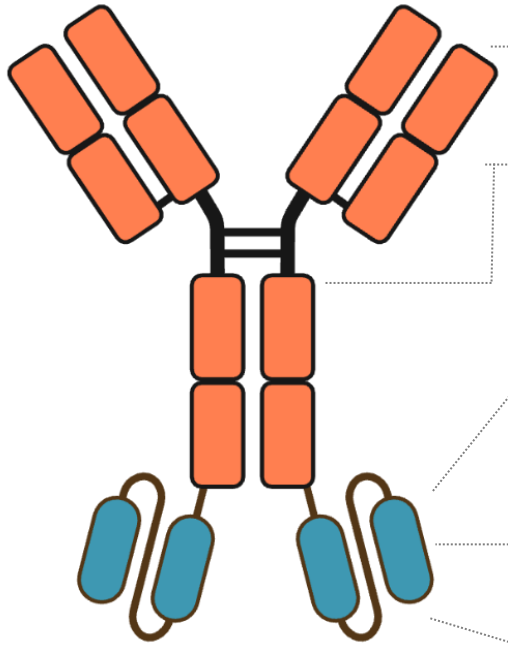
Fc engineering tunes ADCC, CDC, half-life, etc.



…but ensuring cooperative effect, stability, and developability of a tetravalent PD-(L)1 x VEGF bispecific antibody is more difficult

Ivonescimab's unique structure and geometry – and resulting cooperative function – is challenging to replicate

Alternative constructs risk not reproducing ivonescimab's superior efficacy and safety in clinical practice



IgG format bound to VEGF dimer **required to daisy chain**; different potency may alter chaining kinetics and VEGF trap geometry does *not* work

Fc silencing helps reduce risk of AEs

Leading anti-PD-1s are unstable and aggregate in scFv format, requiring significant engineering; **CR-001 maintains >95% monomer at 150mg/mL**

Bispecific antibodies often cannot achieve high concentrations with low enough viscosity to maximize development optionality; **CR-001 is low viscosity (<16 cP) up to 150mg/mL**

CR-001 has novel composition of matter IP related to proprietary, stabilized scFvs

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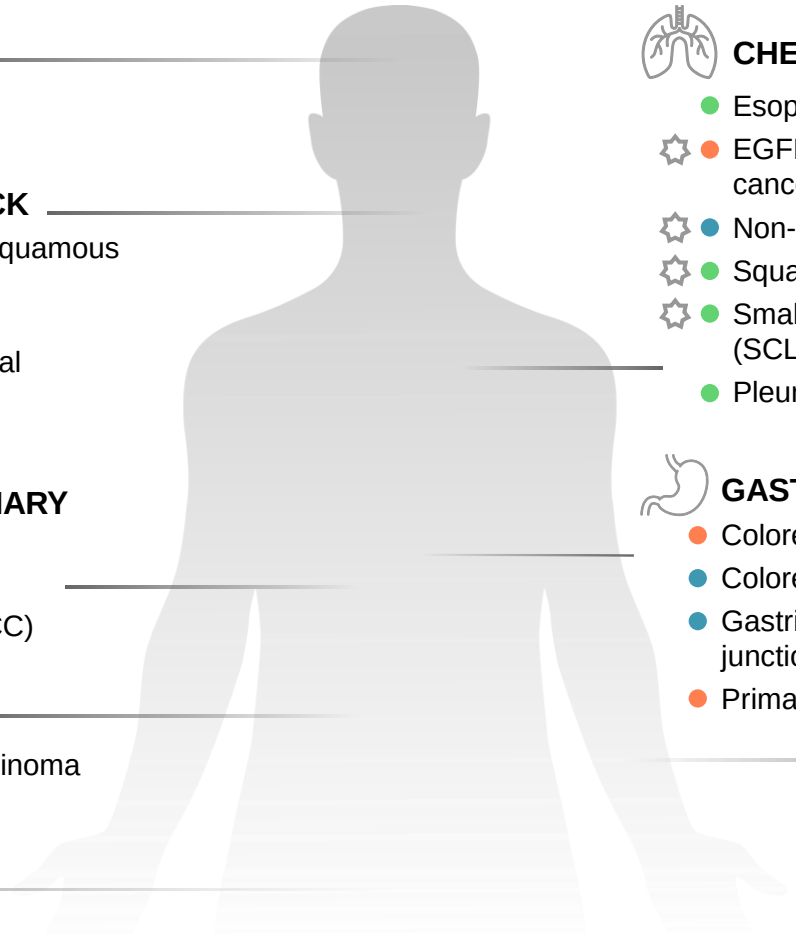
Notes: CDR: Complementarity-determining region. CDC: Complement-dependent cytotoxicity. Fc: Fragment crystallizable.

CR-001 has potential to transform SoC across a multitude of oncology indications, with numerous first-in-class opportunities



- Anti-VEGF approvals
- Anti-PD-(L)1 approvals
- Anti-VEGF and anti-PD(L)-1 approvals
- Ongoing / announced global study from Summit or BioNTech

SOFT TISSUE
- Alveolar soft part sarcoma
- Soft tissue sarcoma

HEMATOLOGICAL
- Classical Hodgkin lymphoma
- Primary mediastinal large B-cell lymphoma (PMBCL)

REPRODUCTIVE
- Cervical
- Endometrial
- Fallopian tube
- Ovarian (epithelial)
- Triple negative breast cancer (TNBC)
- Urothelial

BRAIN
- Glioblastoma

HEAD & NECK
- Head & neck squamous cell carcinoma (HNSCC)
- Nasopharyngeal
- Thyroid

LIVER & BILIARY
- Biliary tract
- Hepatocellular carcinoma (HCC)

KIDNEY
- Renal cell carcinoma (RCC)

CHEST/THORACIC
- Esophageal
- EGFRm non-small cell lung cancer (NSCLC)
- Non-squamous NSCLC
- Squamous NSCLC
- Small cell lung cancer (SCLC)
- Pleural mesothelioma

GASTROINTESTINAL
- Colorectal (all comers)
- Colorectal (MSI-H / dMMR)
- Gastric / Gastroesophageal junction (GEJ)
- Primary peritoneal

TISSUE-AGNOSTIC
- High microsatellite instability (MSI-H) / deficient DNA mismatch repair (dMMR)
- High tumor mutational burden (TMB-H)

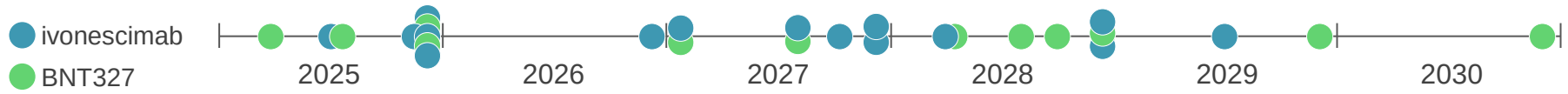
SKIN
- Basal cell carcinoma
- Cutaneous squamous cell carcinoma
- Melanoma
- Merkel cell carcinoma

Notes: EGFRm = mutant epidermal growth factor receptor.
Sources: Keytruda Label; Opdivo Label; Tecentriq Label; Imfinzi Label; Libtayo Label; Bavencio Label; Jemperli Label; Loqtorzi® Label; Zynyz® Label; Avastin Label; Cyramza® Label; Lenvima® Label; Votrient® Label

Development programs across key late-stage competitors include numerous P3s with PFS & OS readouts, paving the way for CR-001

Leading PD-(L)1 x VEGF programs, with **similar expected cooperativity to CR-001**, will generate **Phase 3 PFS & OS catalysts for years to come**

Pivotal readouts expected:



Program	Company	Study	Phase	Indication	Population	Combo	Comparator	Data Expected
ivo PD-1 x VEGF	Akesobio	HARMONi-A	3 (China)	**mNSCLC**	2L EGFRm NSQ	chemo	chemo	OS late 2025
		HARMONi-2	3 (China)	**mNSCLC**	1L PDL1+ TPS ≥ 1%	none	anti-PD-1	OS mid 2025
		AK112-306	3 (China)	**mNSCLC**	1L squamous	chemo	anti-PD-1 + chemo	OS late 2025
		AK117-302	3 (China)	HNSCC	1L R/M PD-L1+ CPS ≥ 1	anti-CD47	anti-PD-1	PFS & OS 2027
		AK112-308	3 (China)	TNBC	1L mTNBC	chemo	chemo	PFS 2026, OS 2028
		AK112-309	3 (China)	BTC	1L A/M BTC ECOG 0-1	chemo	anti-PD-L1 + chemo	PFS & OS 2027
	Summit therapeutics	HARMONi	3 (global)	**mNSCLC**	2L EGFRm NSQ	chemo	chemo	PFS & OS 2025
		HARMONi-3	3 (global)	**mNSCLC**	1L SQ & NSQ	chemo	anti-PD-1 + chemo	PFS & OS 2027-8
		HARMONi-7	3 (global)	**mNSCLC**	1L PD-L1+ TPS > 50%	none	anti-PD-1	PFS & OS 2028-9
BNT327 PD-L1 x VEGF	BIONTECH	PM8002-BC010C	2/3 (China)	**mNSCLC**	2L EGFRm NSQ	chemo	chemo	PFS & OS 2025
		BNT327-06*	2/3 (global)	**mNSCLC**	1L	chemo	anti-PD-1 + chemo	PFS & OS 2029-30
		[announced]*	2/3 (TBA)	**NSCLC**	1L	[TBA]	[TBA]	[TBA]
		PM8002-C013C	3 (China)	TNBC	1L	chemo	chemo	PFS & OS 2027-8
		BNT327-03	3 (global)	SCLC	1L ES-SCLC	chemo	anti- PD-L1 + chemo	PFS & OS 2028
		PM8002-BC011C	2/3 (China)	SCLC	1L ES-SCLC	chemo	PD-L1 + chemo	PFS & OS late 2025
		PM8002-C014C	3 (China)	SCLC	2L	chemo	chemo	PFS & OS 2027-8

Notes: List of trials not exhaustive; BNT327-06 is a master protocol for two P2/3 sub-studies; BNT327 P2/3 in NSCLC announced Nov 2024 but details not yet available; PFS and OS readouts estimated based on primary endpoints and completion dates listed on ClinicalTrials.gov. EGFR: Epidermal growth factor receptor. SQ: Squamous. TPS: Tumor proportion score. TNBC: Triple-negative breast cancer. ES-SCLC: Extensive-stage small cell lung cancer. **Sources:** ClinicalTrials.gov; company websites; company presentations.

Parallel clinical development paths offer potential for both first-in-class and lower risk opportunities for CR-001

TWO PARALLEL DEVELOPMENT PATHS FOR CR-001

First-in-class opportunities

Focus on potential first-in-class opportunities with **rapid path to market** (i.e., efficient development strategy, **anticipated high likelihood of PFS and OS success**)

Numerous indications with **clinically meaningful anti-PD-(L)1 +/- VEGF efficacy** and potential to combine with chemo / orthogonal MoAs

Illustrative

Fast-follower in clinically validated indications

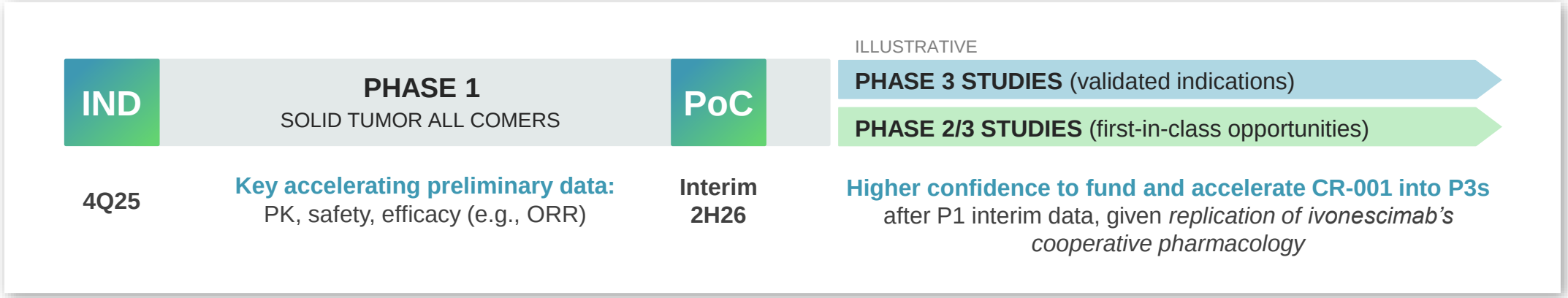
Plan to **rapidly follow ivonescimab** in indications where clinical validation vs. anti-PD-(L)1 is highly differentiating

High conviction **CR-001 can replicate ivonescimab's efficacy** given similar construct and equivalent MoA

Potential indications based on ongoing Phase 3 trials

CR-001 Phase 1 data offer potential for early acceleration – a rarity for a solid tumor oncology program

Phase 1 interim proof-of-concept readout is a potentially significant value-generating event for CR-001



Preliminary data from early Phase 1 cohorts provides substantial **validation of program** because CR-001's structural design and preclinical data are similar to those of ivonescimab

Early Phase 1 data, as single agent and in combination with SoC, **enables rapid late-stage development** in multiple solid tumor types, unlocking broad first-in-class and fast-follower opportunities

CR-001 is markedly **differentiated from novel constructs disconnected from ivonescimab's MoA**; alternative formats may require significantly more patients worth of safety and efficacy data in tumor-specific expansion cohorts and/or Phase 2s to establish conviction before initiating Phase 3s

High conviction in CR-001's clinical profile can be reached in ~9-12 months from Phase 1 initiation, offering potential for significant early value inflection

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CR-001 preclinical data reproduce ivonescimab's breakthrough pharmacology & are rapidly advancing to generate significant value

Unprecedented third-party data validate PD-1 x VEGF cooperativity

*Ivonescimab significantly **improved PFS versus pembrolizumab** in Phase 3 in 1L NSCLC – the first therapy to do so head-to-head*

Transformative MoA for $50B+ market

*Poised to **transform** NSCLC **standard of care**, with broad application across $50B+ anti-PD-(L)1 market*

CR-001's proprietary engineering is designed to replicate ivonescimab

*CR-001 is a highly potent PD-1 x VEGF bsAb **reproducing cooperative binding** qualities critical to ivonescimab*

Promising pipeline of next-gen ADCs

CR-002 and CR-003** offer **complementary** development opportunities for **CR-001

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CR-002 & CR-003

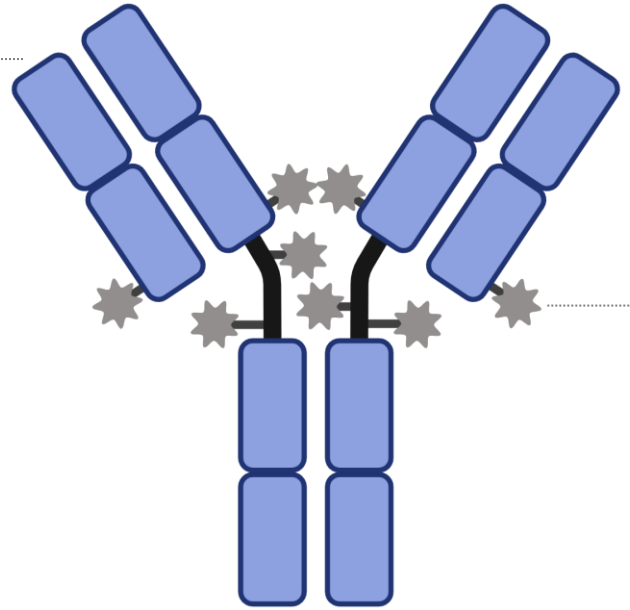
Topoisomerase inhibitor ADCs against validated targets

CR-002 and CR-003 are potentially best-in-class topoisomerase inhibitor ADCs, with applicability across solid tumors

Validated, undisclosed solid tumor ADC targets

Targets for CR-002 and CR-003 to be disclosed as programs approach IND

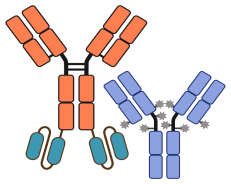
Each unique target has **potential in multiple solid tumor indications**



Best-in-modality topoisomerase inhibitor payloads

Topoisomerase inhibitor payloads have consistently demonstrated **superior efficacy and safety** over microtubule inhibitor payloads

Each ADC is expected to have **bystander-killing effect**



Potential to synergize with CR-001 and other immunotherapies

Each ADC can be leveraged in **combination studies** in solid tumors

Multiple indications with ongoing PD-(L)1 x VEGF bispecific development and *separate* development of ADCs **accelerate clinical path for combinations**

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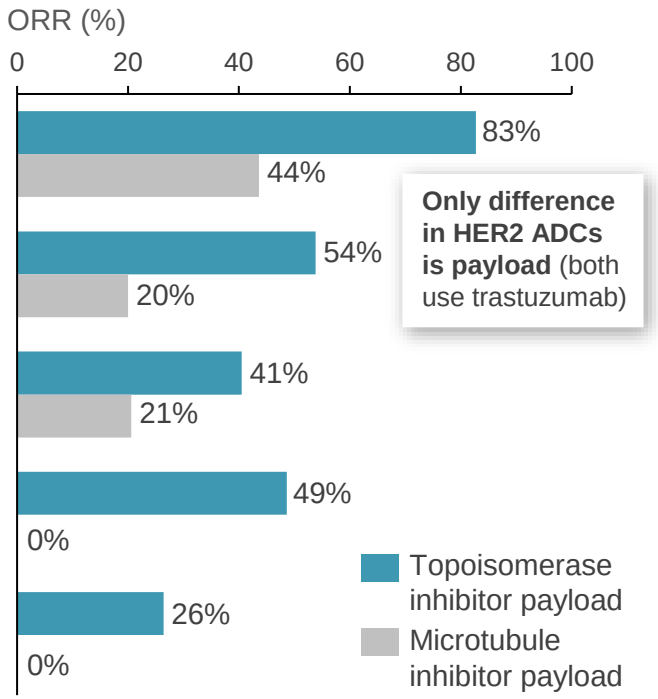
ADCs with topoisomerase inhibitor payloads have demonstrated best-in-modality efficacy and safety

CROSS-TRIAL COMPARISONS

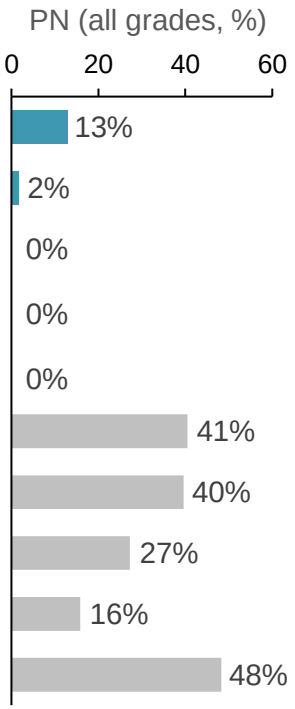
TopoI payload-based ADCs have **demonstrated superior ORR** vs. microtubule inhibitor-based ADCs in cross-trial comparisons…

Target	Indication
HER2 (*Enhertu* vs. *Kadcyla*)	Breast
	NSCLC
	Gastric / GEJ
TROP2 (*Dato-DXd* vs. *PF-06664178*)	Ovarian
	NSCLC



… and have shown much **lower rates of peripheral neuropathy**, a critical AE that can **drive dose reductions & discontinuations**

	Target	Molecule	Phase	PN (all grades, %)
Topoi payload	HER2	Enhertu	A	13%
	TROP2	Dato-DXd	R	2%
	HER3	Patritumab-DXd	R	0%
	B7-H3	Ifinatamab-DXd	3	0%
	CDH6	Raludotatug-DXd	2	0%
MT payload	CD30	Adcetris	A	41%
	Nectin-4	Padcev	A	40%
	TF	Tivdak	A	27%
	HER2	Kadcyla	A	16%
	HER2	Disitamab vedotin	3	48%



CR-002 and CR-003 utilize the **best-in-ADC payload in their potentially best-in-class profiles**

Notes: GEJ: Gastroesophageal junction. A: Approved. R: In registration. PN rates are weighted averages, by number of patients, across indications / trials and include PN, PSN, PMN, and PSMN when separately measured; full list of trials and references available on request. Disitamab vedotin is approved in China and in Phase 3 development globally. **Sources:** Enhertu Label; 2024 Smit (Lancet Onc); Kadcyla Label; 2019 Peters (Clin Cancer Res); 2017 Thuss-Patience (Lancet Onc); 2024 Oaknin (ESMO Pres); 2024 Ahn (JCO); 2018 King (Invest New Drugs)

Corporate

Rapidly growing leadership team with deep experience building the next generation of biotechnology companies

Executive Team

Joshua Brumm
Chief Executive Officer

Jonathan McNeill, M.D.
President & Chief Operating Officer

Ellie Im, M.D.
Chief Medical Officer

Rick Scalzo
Chief Financial Officer

Barbara Bispham
General Counsel

Christopher Doughty
Chief Business Officer

Ryan Lynch
Chief Accounting Officer

Amy Reilly
Chief Communications Officer

Wenjie Cheng, Ph.D.
SVP, Technical Operations

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Peter Harwin
Chair

Alex Balcom

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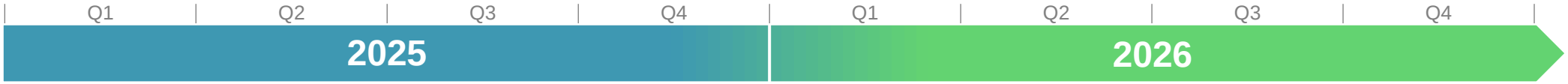
Financing expected to fund Crescent programs through key anticipated value-generating catalysts

CR-001
IND
4Q25

CR-002
IND
MY26

CR-001
Initial clinical data
2H26



Current cash expected to fund operations through 2027

- 1H: BNT327 P2/3 EGFRm NSQ mNSCLC interim (China)
- 1H: Ivo P3 1L SQ mNSCLC interim (China)

- **2H: Ivo P3 HARMONi-2 1L mNSCLC OS (China)**
- 2H: BNT327 P2/3 1L ES-SCLC interim (China)
- 2H: Ivo P3 HARMONi EGFRm NSQ mNSCLC interim (global)
- 2H: Ivo P3 HARMONi-A EGFRm NSQ mNSCLC completion (China)

Multiple P3 trials ongoing or planned (e.g., SCLC, TNBC, NSCLC), with numerous PFS & OS readouts expected in 2026 and beyond

Notes: mNSCLC: Metastatic non-small cell lung cancer. ES: Extensive stage. EGFRm: Mutant EGFR. **Sources:** ClinicalTrials.gov; Company websites

Estimated capitalization following close of transactions

		Shares on an as-converted basis	Expected ownership of the combined company
GlycoMimetics	Shares of common stock outstanding	64,532,953	3.1%
CRESCENT BIOPHARMA	Shares of common stock outstanding	105,137,814	96.9%
	Series A shares	298,298,000	
Pre-closing financing	Shares of common stock	1,339,680,730	
	Pre-funded warrants	273,643,080	
Estimated total shares of common stock of the combined company post-closing		2,081,292,577	

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Thank you