



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**

PROGRAM	MOA	DISCOVERY	IND-ENABLING	IND	POTENTIAL INDICATIONS	
		[Progress bars]				
CR-001¹	PD-1 x VEGF same cooperative MoA as ivonescimab	[Progress bar]			4Q25	NSCLC, other solid tumors
CR-002	Undisclosed ADC #1 with Topol payload	[Progress bar]			Mid-2026	Solid tumors
CR-003	Undisclosed ADC #2 with Topol payload	[Progress bar]				Solid tumors



~\$200 million financing in October 2024 anticipated to fund operations through 2027²

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

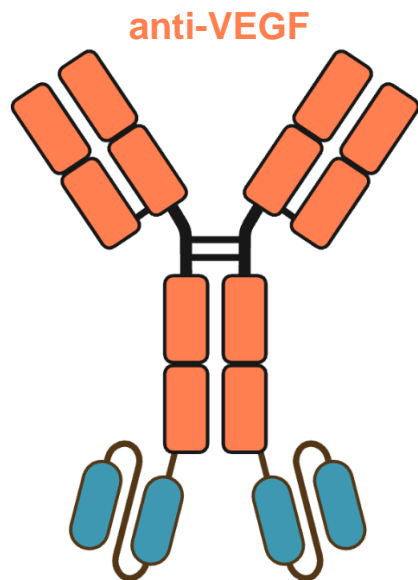


1. Anticipated expiration for filed provisional patent is 2045+
 2. Financing scheduled to close immediately prior to the closing of the merger with GlycoMimetics
Notes: NSCLC: Non-small cell lung cancer. MoA: Mechanism of action. ADC: Antibody-drug conjugates. Topol: Topoisomerase

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



anti-PD-1 scFvs

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

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

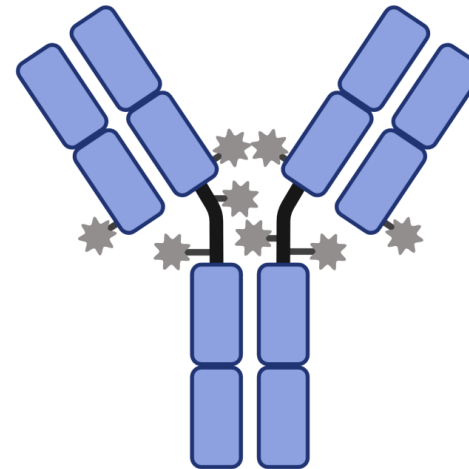
Potential to **move to frontline use in the \$50B+ PD-(L)1 immunotherapy market**

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

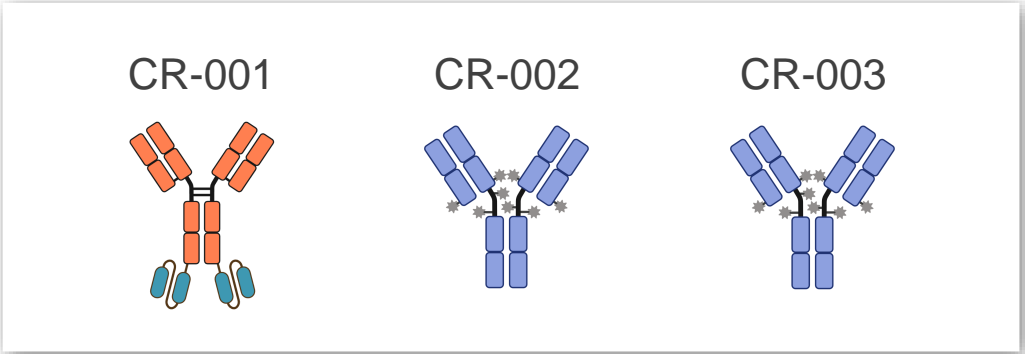
Both utilize the **best-in-modality cytotoxic payload**: topoisomerase inhibitor

CR-002 IND expected mid-26

Interim PoC data expected 2027

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

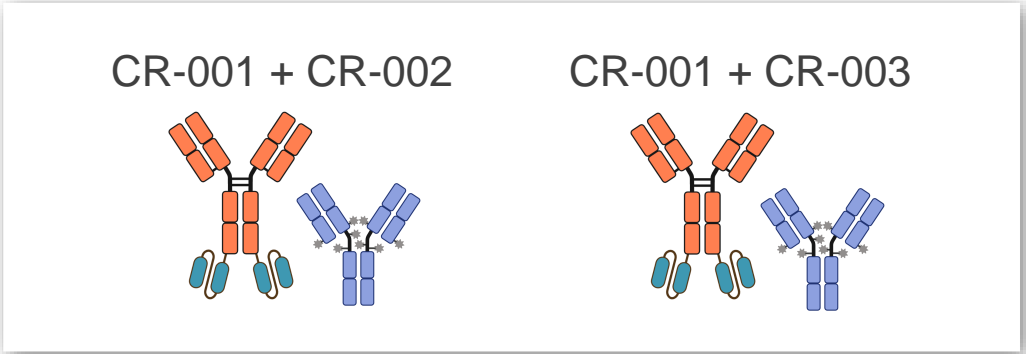
Optimized Novel Monotherapies



Engineered for:

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

Synergistic Combination Approaches



Selected for:

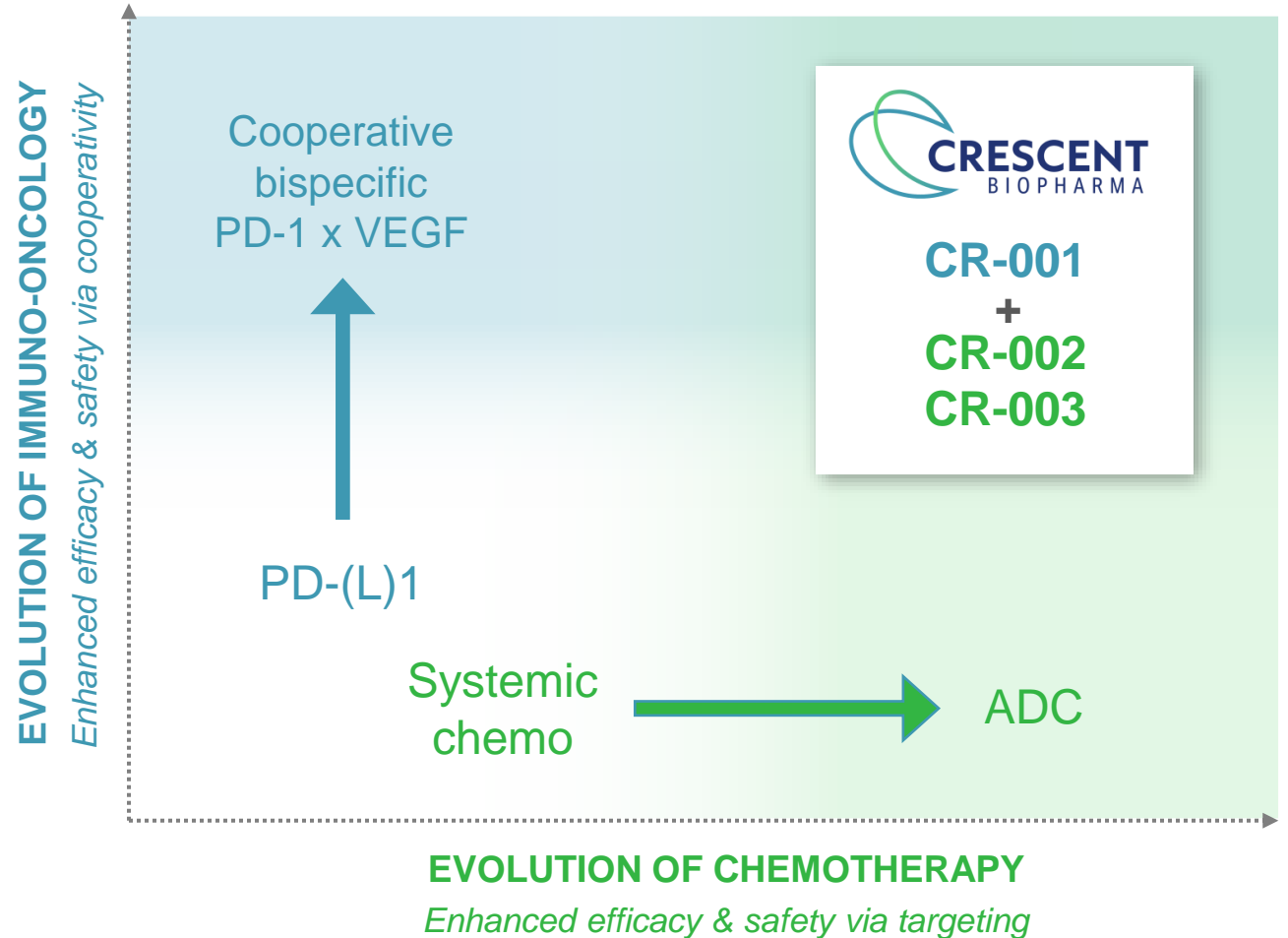
- MoA synergy
- Efficacy in overlapping histologies
- Broad utility in solid tumors

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



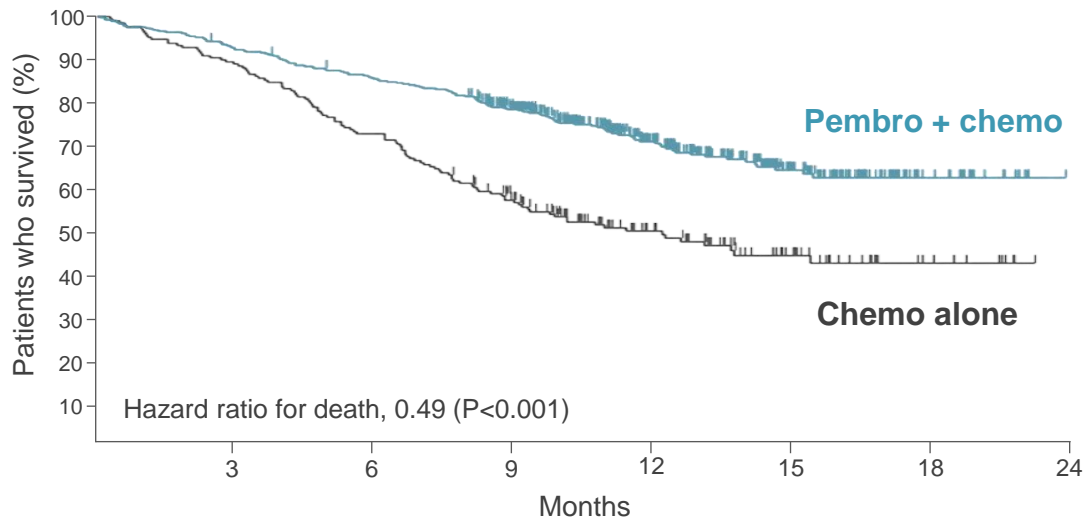
PD-(L)1-targeted therapies, annualizing \$50B+, have transformed oncology – with Keytruda now the best-selling drug in the world

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

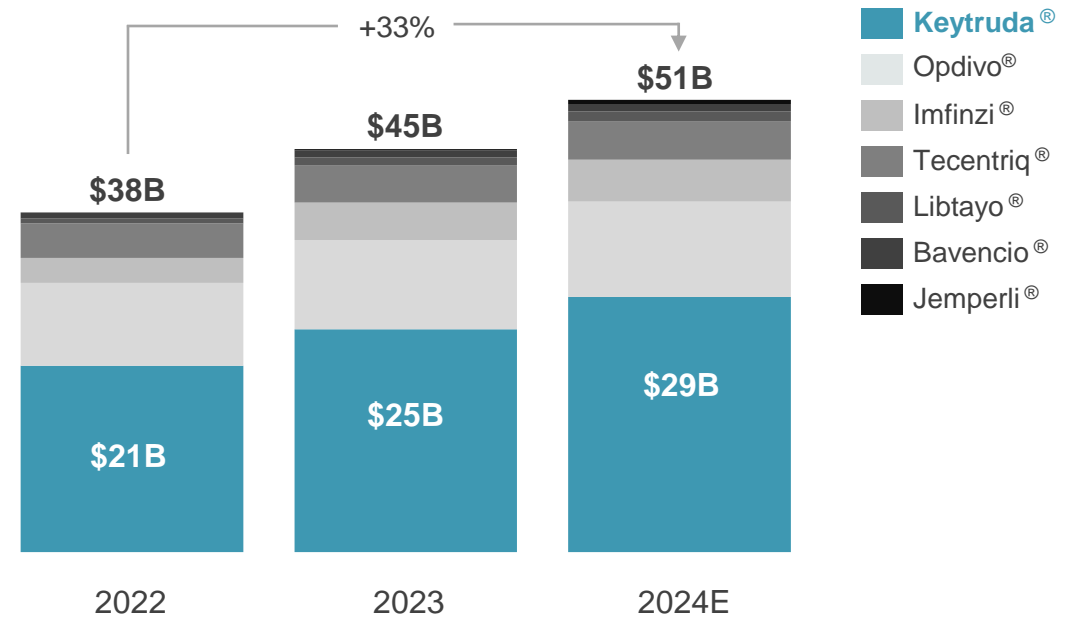
PD-(L)1-targeted therapy is one of the largest drug classes, with Keytruda (pembrolizumab) the dominant player

Example

In 1L NSQ NSCLC, addition of pembrolizumab to chemo significantly improved mOS (NR vs 11.3 months¹ with HR 0.49)



Anti-PD-(L)1 global sales

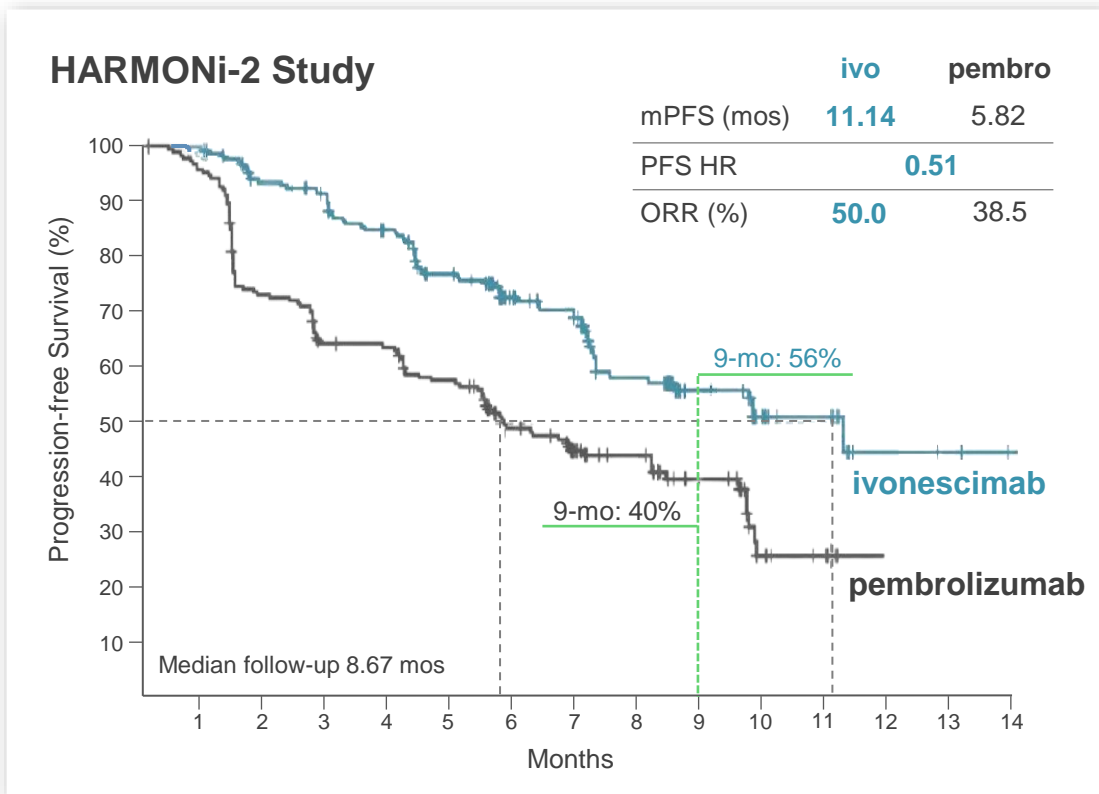


Keytruda alone is approved in 20+ oncology indications with expected revenue of ~\$30B in 2024

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})

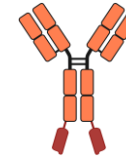
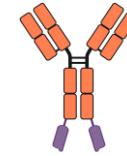
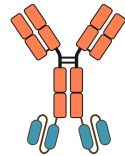
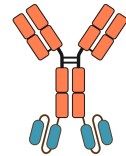
	PD-L1 ^{low} (TPS 1-49%)	PD-L1 ^{high} (TPS ≥50%)	Non-squamous	Squamous
HR	0.54	0.46	0.54	0.48

🛡️ Promising Safety

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

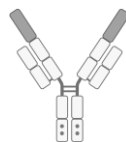
Dual blockade of PD-1 and VEGF through a cooperative bispecific antibody has led to unprecedented clinical results, demonstrating superiority to pembrolizumab and a **\$15B+ market cap for ivonescimab's ex-China sponsor, Summit Therapeutics**

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

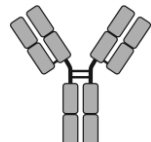


	Anti-PD-1 scFv-based		Anti-PD-1 VHH-based	Anti-PD-L1 VHH-based
Program	CR-001	ivonescimab	LM-299	BNT327 / PM8002
Company				
Stage	Preclinical	Phase 3 (Global)	Phase 1/2 (China)	Phase 3 (Global)
Anti-VEGF IgG	Bevacizumab	Bevacizumab	Bevacizumab	Bevacizumab
Anti-PD-(L)1	Anti-PD-1 scFvs	Penpulimab scFvs	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

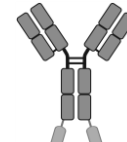
Examples of alternative constructs



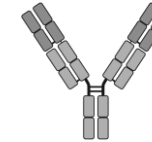
- Anti-PD-L1 IgG with enhanced ADCC
- VEGF trap



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



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

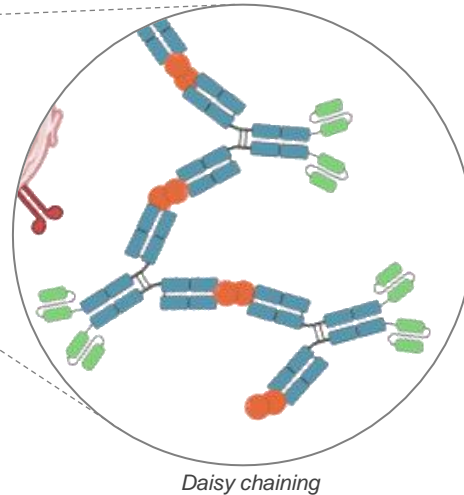
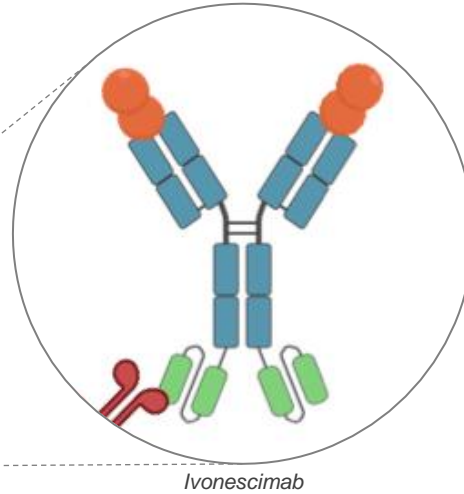
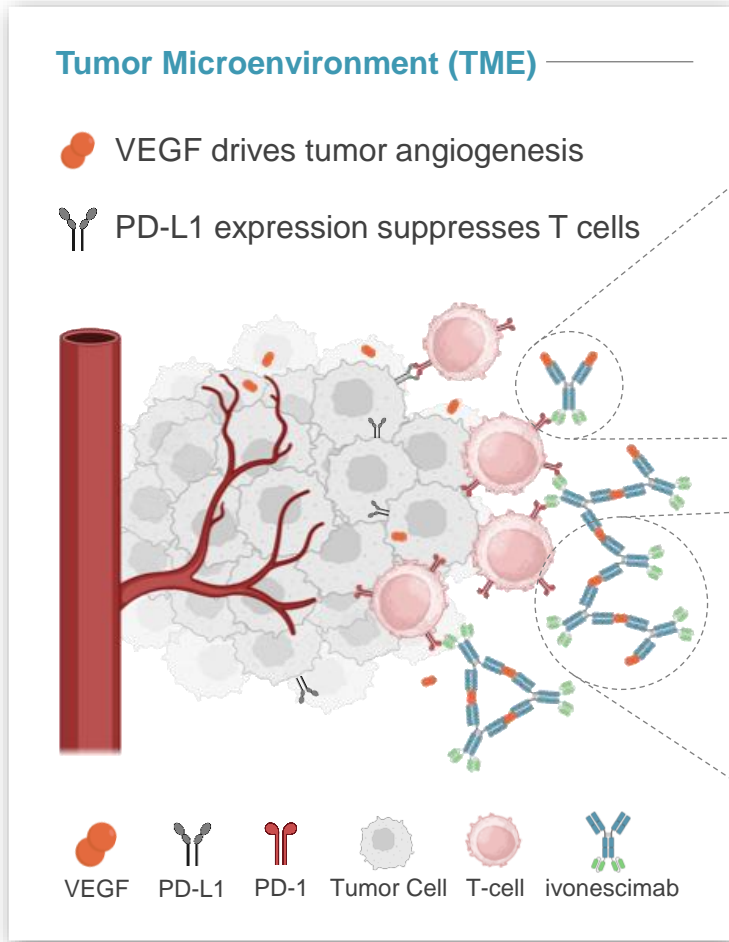


- Bevacizumab
- Anti-PD-1 Fabs
- PD-1 domains attached to IgG N-terminus instead of C-terminus

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 tetraivalent 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

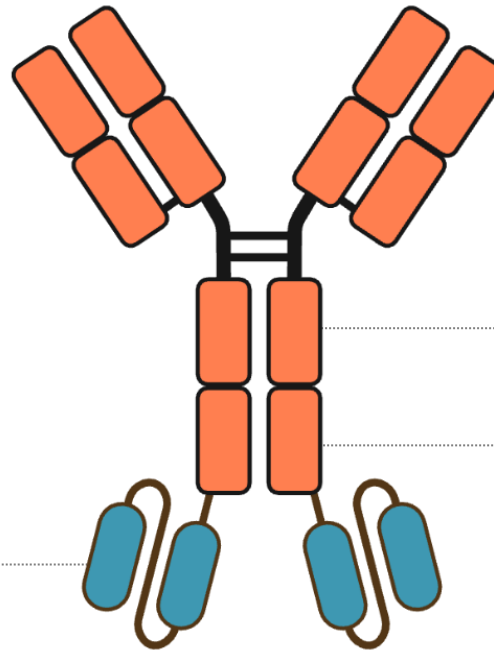
CR-001 is a highly potent PD-1 x VEGF bsAb designed to recapitulate ivonescimab's cooperative pharmacology

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

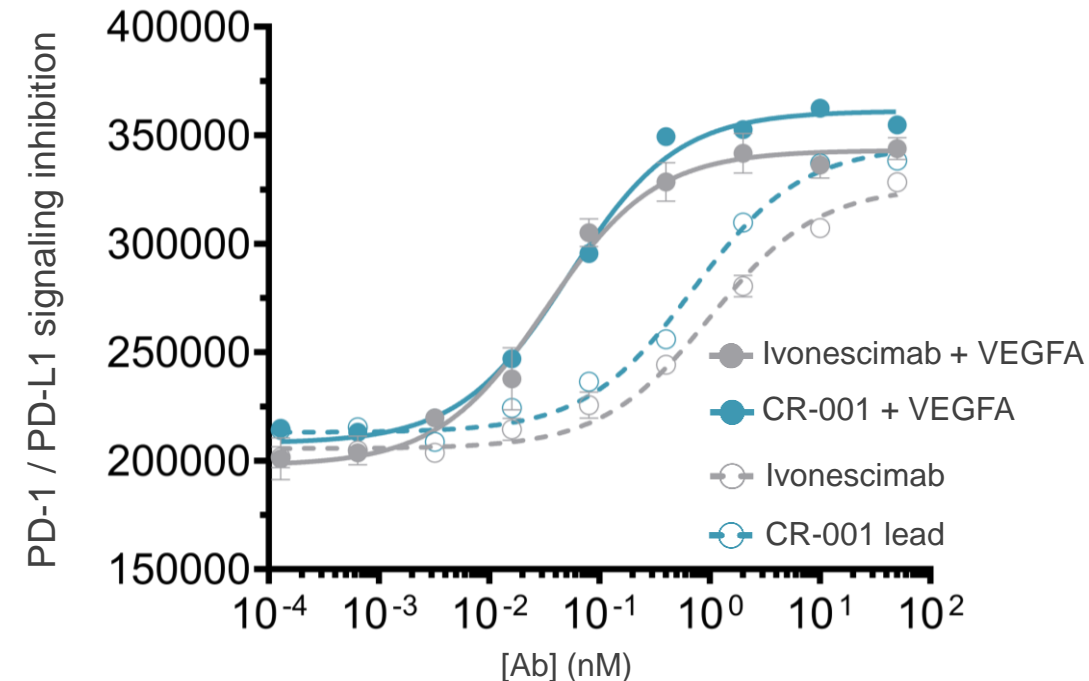
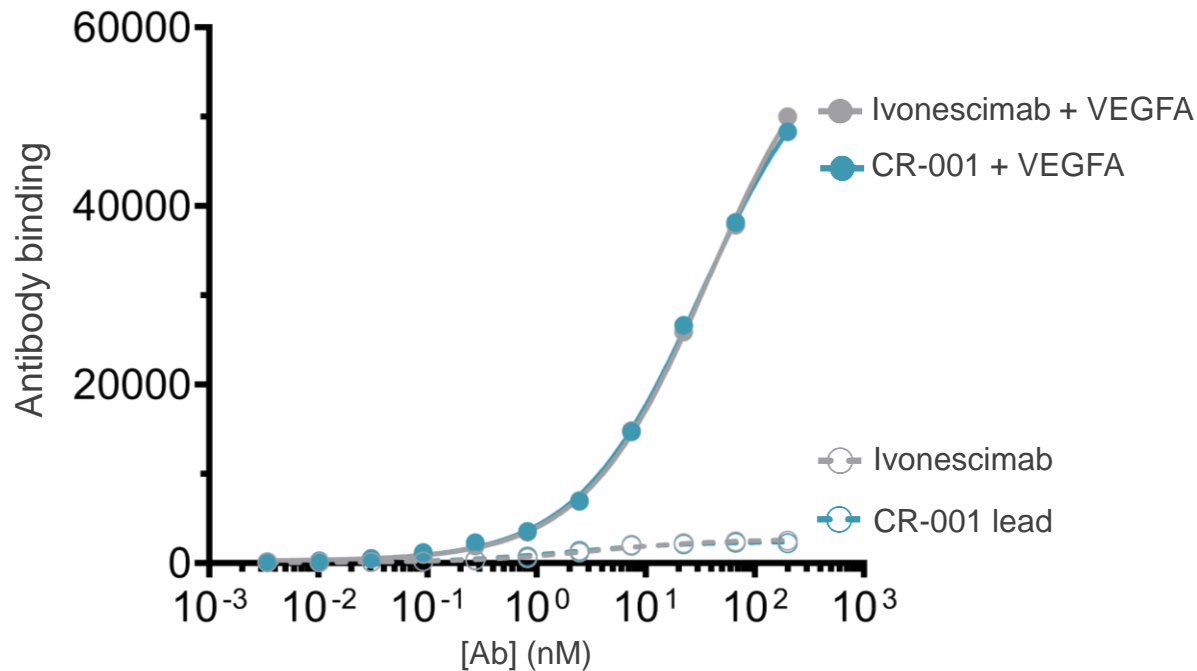
Designed to match ivonescimab PK

Native FcRn binding to match distribution and elimination of ivonescimab

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**.



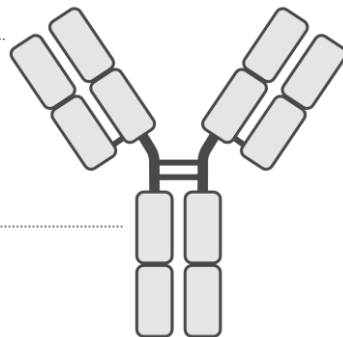
CR-001 lead demonstrates same cooperative effect as ivonescimab across multiple assays

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

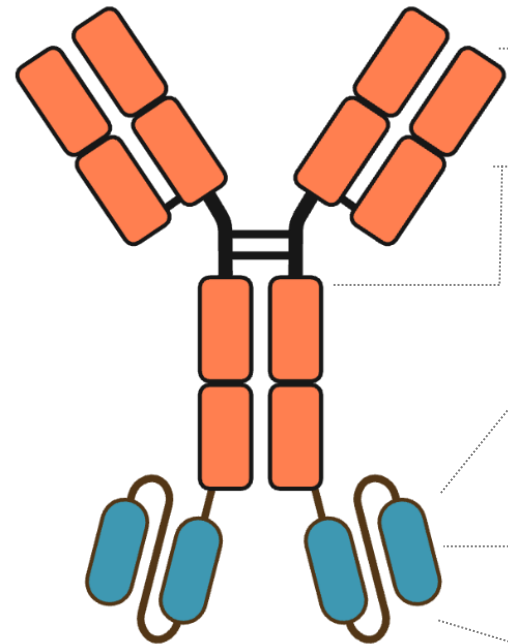
Fc engineering tunes ADCC, CDC, half-life, etc.



...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

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



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

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



TISSUE-AGNOSTIC

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



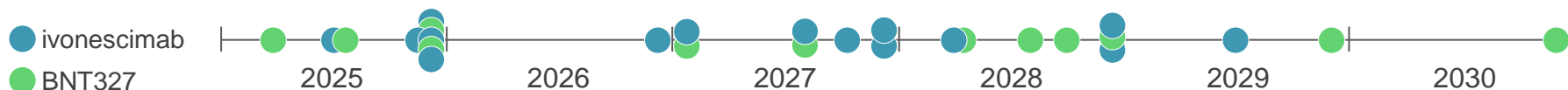
SKIN

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

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		

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

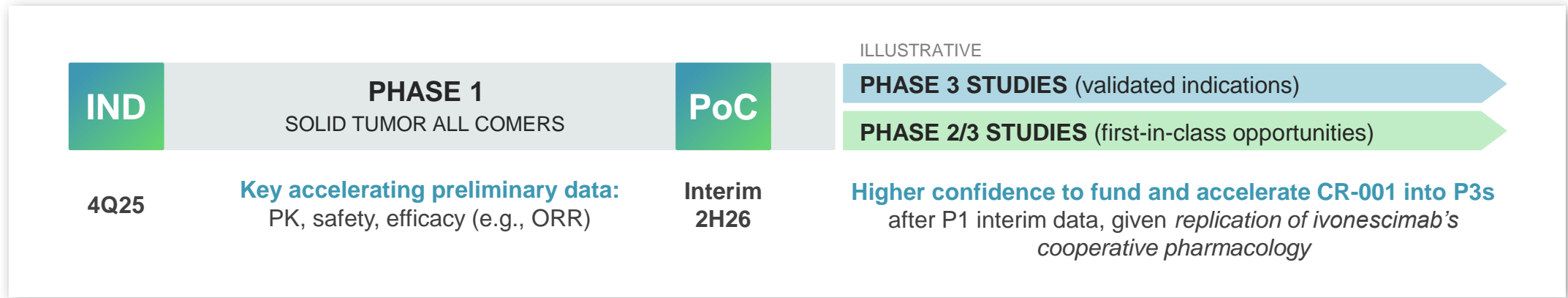


Potential indications based on ongoing Phase 3 trials

TWO PARALLEL DEVELOPMENT PATHS FOR CR-001

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

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

CR-002 & CR-003

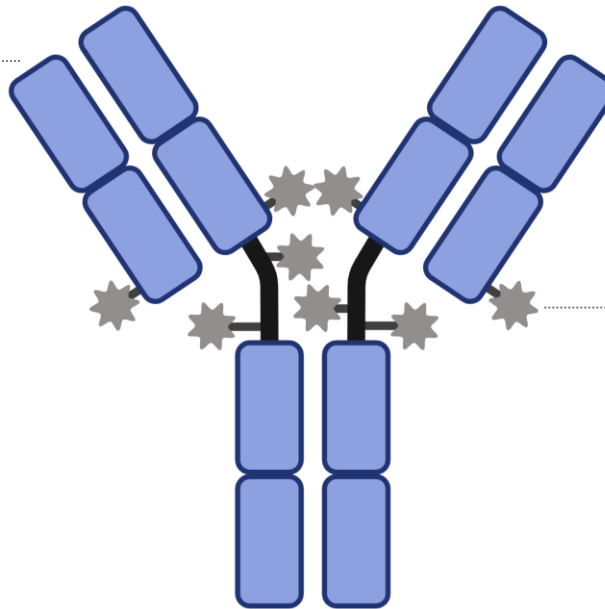
*Topoisomerase inhibitor ADCs
against validated targets*

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

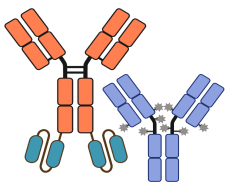
Each unique target has **potential in multiple solid tumor indications**



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

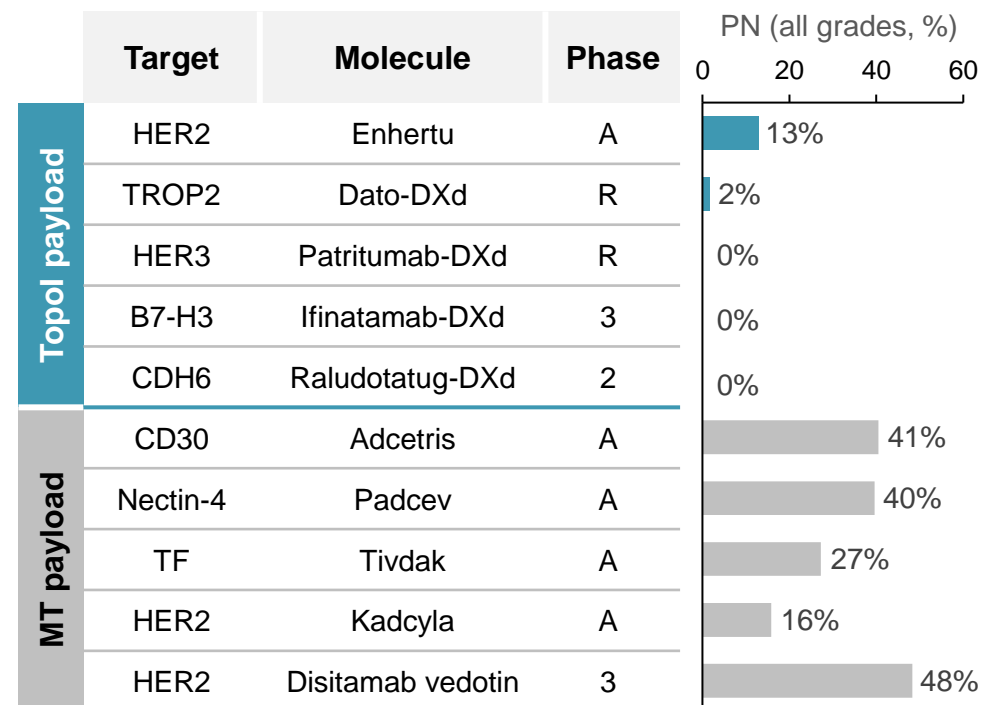
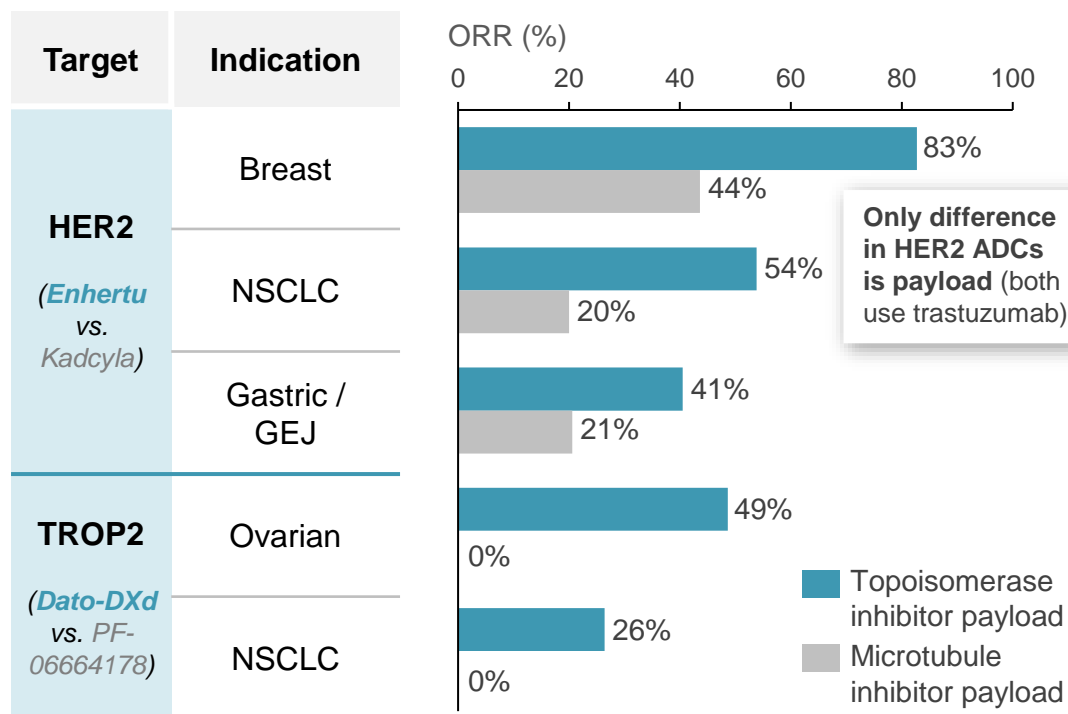
Multiple indications with ongoing PD-(L)1 x VEGF bispecific development and *separate* development of ADCs **accelerate clinical path for combinations**

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

CROSS-TRIAL COMPARISONS

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

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



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

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

Board of Directors



Peter Harwin
Chair



Alex Balcom



Susan Moran, M.D.



Jonathan Violin, Ph.D.



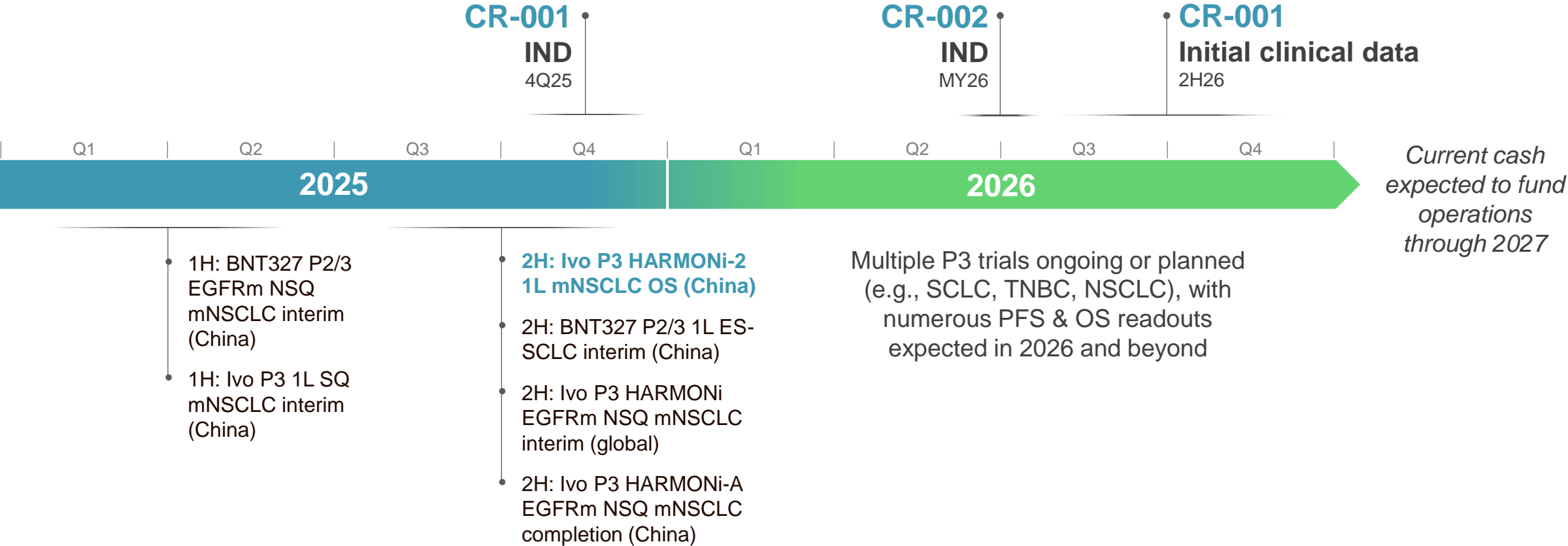
Joshua Brumm





David Lubner



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



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%
	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	



Thank you