



1Q26 Corporate Presentation

Oncology and Immunology Portfolio Summary

May 5, 2026

Forward-looking Statements/Safe Harbor

Forward-Looking Statements Safe Harbor: This presentation contains forward-looking statements about Arcus Biosciences, Inc. (“we,” “Arcus” or the “Company”) made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. All statements in this presentation that are not historical facts are forward-looking statements, including, without limitation, statements regarding: our anticipated cash runway (including our expectation of funding until at least the second half of 2028); our strategies, goals, opportunities, and potential advantages for our programs, including casdatifan and our inflammation and immunology programs; estimates of market potential or peak sales for our investigational products; the timing, initiation, enrollment, advancement, conduct, and results of our clinical trials and other development activities (including, without limitation, expected timing for completion of enrollment for PEAK-1, initiation of a new Phase 3 study in first-line RCC, advancement of our inflammation and immunology programs into the clinic, and timing and results from ongoing clinical studies); and our expectations regarding our ability to generate value for shareholders. Forward-looking statements are often identified by words such as “anticipate,” “believe,” “expect,” “intend,” “may,” “should,” “plan,” “project,” “target,” “will,” and similar expressions, although not all forward-looking statements contain these identifying words.

These forward-looking statements are subject to numerous risks, uncertainties and assumptions that may cause actual results to differ materially from those expressed or implied by any forward-looking statements, including, but not limited to: our ability to manage the breadth and pace of our development plans for casdatifan; the unexpected emergence of adverse events or other undesirable side effects with our investigational products, including casdatifan; risks associated with interim or preliminary clinical data not being replicated in other studies evaluating the same investigational product, including PEAK-1 for casdatifan; difficulties or delays in conducting or completing our clinical trials due to regulatory review, site activation, patient identification or enrollment, or manufacturing and supply constraints of investigational or standard-of-care products for such clinical trials, all of which may be exacerbated by unfavorable global economic, political, public health and trade conditions; changes to our cash runway due to changes in our operating plans; changes in the competitive landscape; our ability to obtain and maintain intellectual property protection for our product candidates; and the inherent uncertainty associated with pharmaceutical product development and clinical trials. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those anticipated or implied in the forward-looking statements. Further information on these and other factors that could affect the forward-looking statements made herein is described in our most recent periodic reports filed with the U.S. Securities and Exchange Commission, including our most recent Annual Report on Form 10-K, and in other filings and reports we make with the SEC from time to time. You should not rely upon forward-looking statements as predictions of future events. Except as required by law, neither we nor any other person assumes responsibility for the accuracy or completeness of the forward-looking statements. We undertake no obligation to update any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in our expectations, except as required by law.

Important Information Regarding Data Comparisons: This presentation includes comparisons between data from our Phase 1/1b ARC-20 trial and published data from separate trials that are not head-to-head studies. Cross-trial comparisons should be interpreted with caution due to differences in study populations, sample sizes, inclusion and exclusion criteria, trial design, dosing, endpoints, follow-up, and other factors that may limit direct comparability.

Third-Party Sources Disclaimer: Additionally, this presentation contains certain information related to or based on studies, publications, surveys and other data obtained from third-party sources, and our own internal estimates and research, including without limitation relating to market size and potential sales or revenue opportunity. This information is based on a number of assumptions, projections and estimates, including with respect to our future performance and the future performance of markets in which we operate, and is necessarily subject to a high degree of uncertainty and risk, and you are cautioned not to place undue reliance onto to such estimates.

No Regulatory Approval: All of Arcus’s molecules are investigational and Arcus (and Gilead for all of the molecules in each optioned program) has not received approval from any regulatory authority for any use globally, nor established the safety and efficacy of these investigational molecules.

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Arcus Has Created a Robust Portfolio of Potential Best-in-Class Medicines

FOUNDED 2015

R&D ENGINE

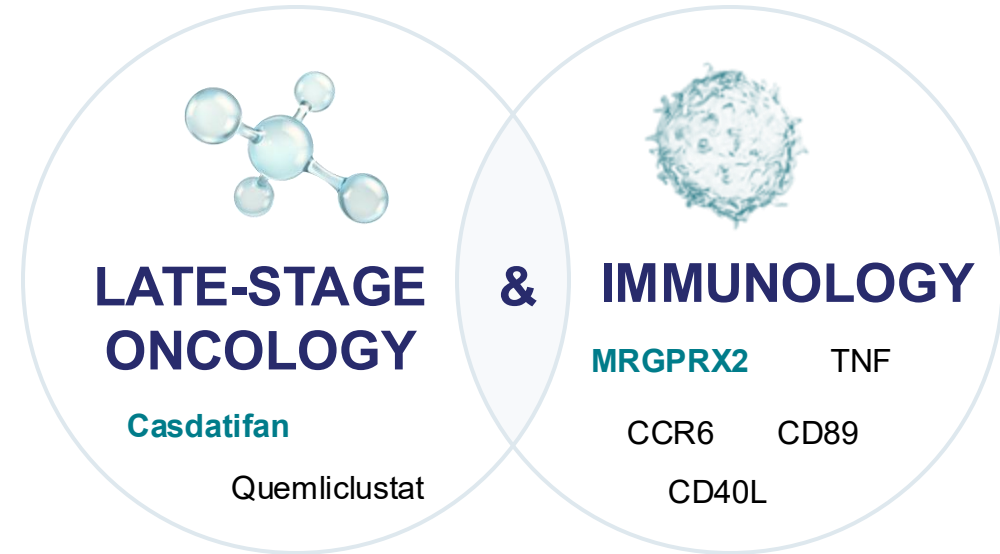
- World class medicinal chemistry
- Goal of 1-2 new molecules advancing to the clinic each year

CAPITAL EFFICIENCY & RISK SHARING HAVE BEEN GROWTH ENABLING



~\$876M IN CASH*

- Funded until at least 2H:28**



1st immunology molecules should enter the clinic in 2026
Addressing **most common** diseases

Cancers with Large Patient Populations

Kidney Pancreatic

Inflammation & Autoimmune Diseases

IBD RA Psoriasis
Urticaria AD

* Cash, cash equivalents and marketable securities as of March 31, 2026

** Runway estimate based on cash, cash equivalents, marketable securities, and current planned operations

References and acronyms on slide 40

Leveraging our Small Molecule Capabilities to Create Best-in-Class Oral Medicines

2 PHASE 3 PROGRAMS IN ONCOLOGY



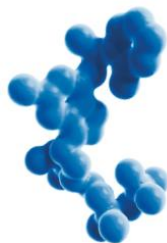
CASDATIFAN

Wholly-owned* HIF-2 α inhibitor with "best in class" data; target validated clinically and commercially in RCC



Phase 3 enrolling

New Phase 3 Trial in
1L Targeted for YE:26



QUEMLICLUSTAT

Only small molecule CD73 inhibitor in clinical development; In Ph 3 for 1L pancreatic cancer



EXPANDING ORAL SMALL MOLECULE I&I PORTFOLIO

Enormous Opportunity to Displace Injectable, Blockbuster Biologic Drugs

AB102 MRGPRX2 antagonist

Expected in clinic in 3Q26



Urticaria



Atopic Dermatitis

TNF inhibitor

Expected in clinic in early '27



IBD



RA



Psoriasis

*Arcus has world-wide rights including the US, Europe and China; Taiho has rights in Japan and other Asian territories

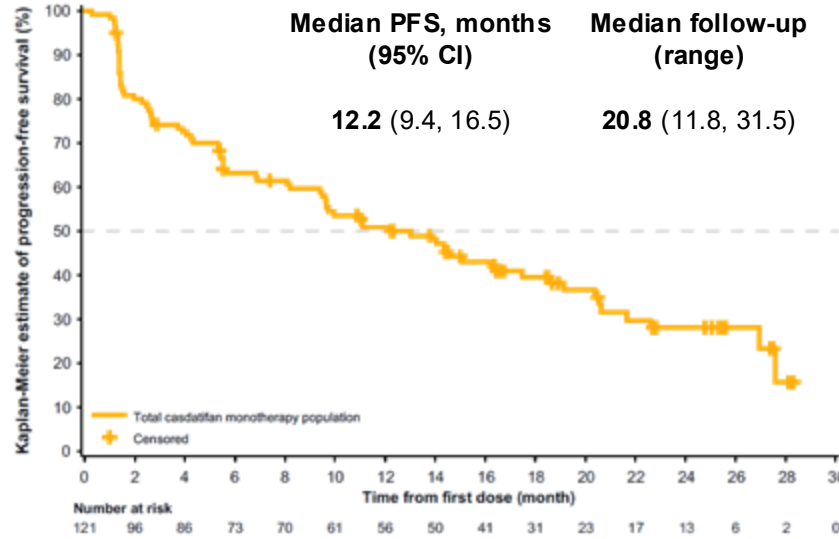
References and acronyms on slide 40

Clinical Data from >120 Patients Support Casdatifan's Potential as the Best-in-Class HIF-2 α Inhibitor

ARC-20

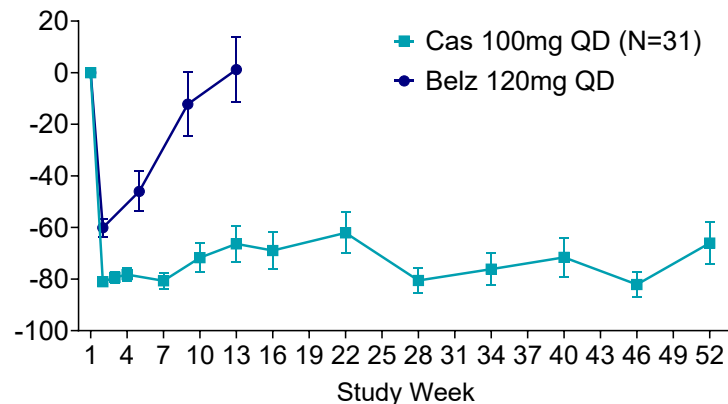
IMPROVED CLINICAL OUTCOMES¹

➤ **Longer PFS than other HIF-2 α inhibitors and TKIs in late-line kidney cancer**
 12.2 months mPFS compares to 5.6 mos PFS for belzutifan in LITESPARK-005^{2,3}



SUPERIOR PHARMACODYNAMIC PROFILE^{4,5}

➤ **100mg QD of cas results in substantial and sustained EPO suppression**



Top Priority is to Establish Casdatifan as the SOC Across Multiple Settings of RCC

PEAK-1

ARC-20

Post-IO metastatic cas + cabo | Phase 3

1L TKI-free regimen

19K

patients* (2L)

28K

patients* (1L)

~\$2B+

~\$3B+

*Patient and sales opportunity based on drug treatable population in major markets; from Decision Resources Group and Arcus analysis.

Data are not from head-to-head studies. Cross-trial data interpretation should be considered with caution as it is limited by differences in study population, sample size, inclusion and exclusion criteria and many other factors.

DRG epi Major Markets, 2026. Major Markets: France, Germany, Italy, Japan, Spain, United Kingdom, United States.

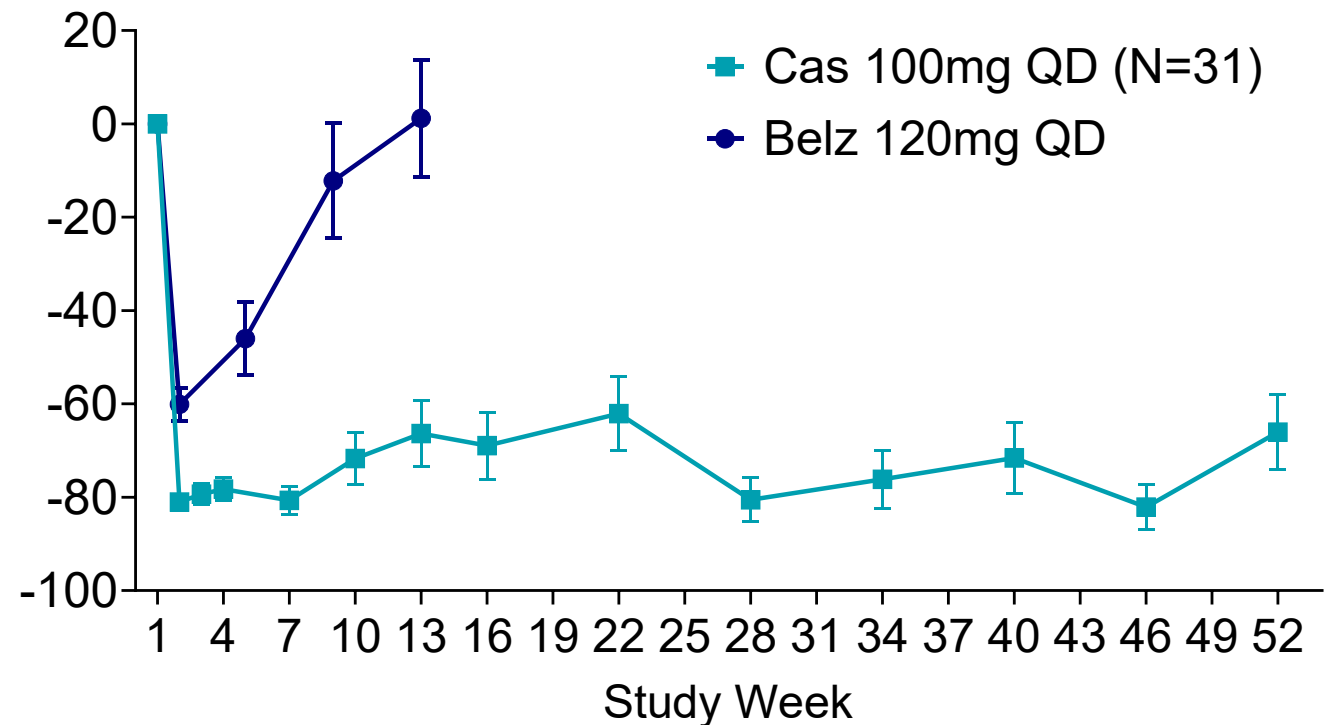
References and acronyms on slide 40

Superior Profile of Casdatifan Drives Clinical Differentiation

100mg QD of cas results in substantial and **sustained** EPO suppression

- ✓ Lower PD rate*
- ✓ Higher ORR*
- ✓ Longer PFS*
- ✓ Shorter onset to disease control*

Published data from Arcus⁴ and Merck⁵



*Relative to published data from studies with belzutifan. Data are not from head-to-head studies. Cross-trial data interpretation should be considered with caution as it is limited by differences in study population, sample size, inclusion and exclusion criteria and many other factors.

References and acronyms on slide 40

Program Updates: New Cas Data and Emerging I&I Portfolio

IMPRESSIVE CASDATIFAN MONO-TX DATA IN LATE-LINE ccRCC WERE PRESENTED AT ASCO GU

- With longer follow up, data have improved even further (Jan. 30, 2026 DCO¹)
- cORR increased to 45% with a mPFS of 15.1 months for the 100mg QD tablet cohort (dose and formulation selected for Phase 3)
 - cORR increased to 35% and PFS was stable at 12.2 months in a pooled analysis of all four monotherapy cohorts (n=121)

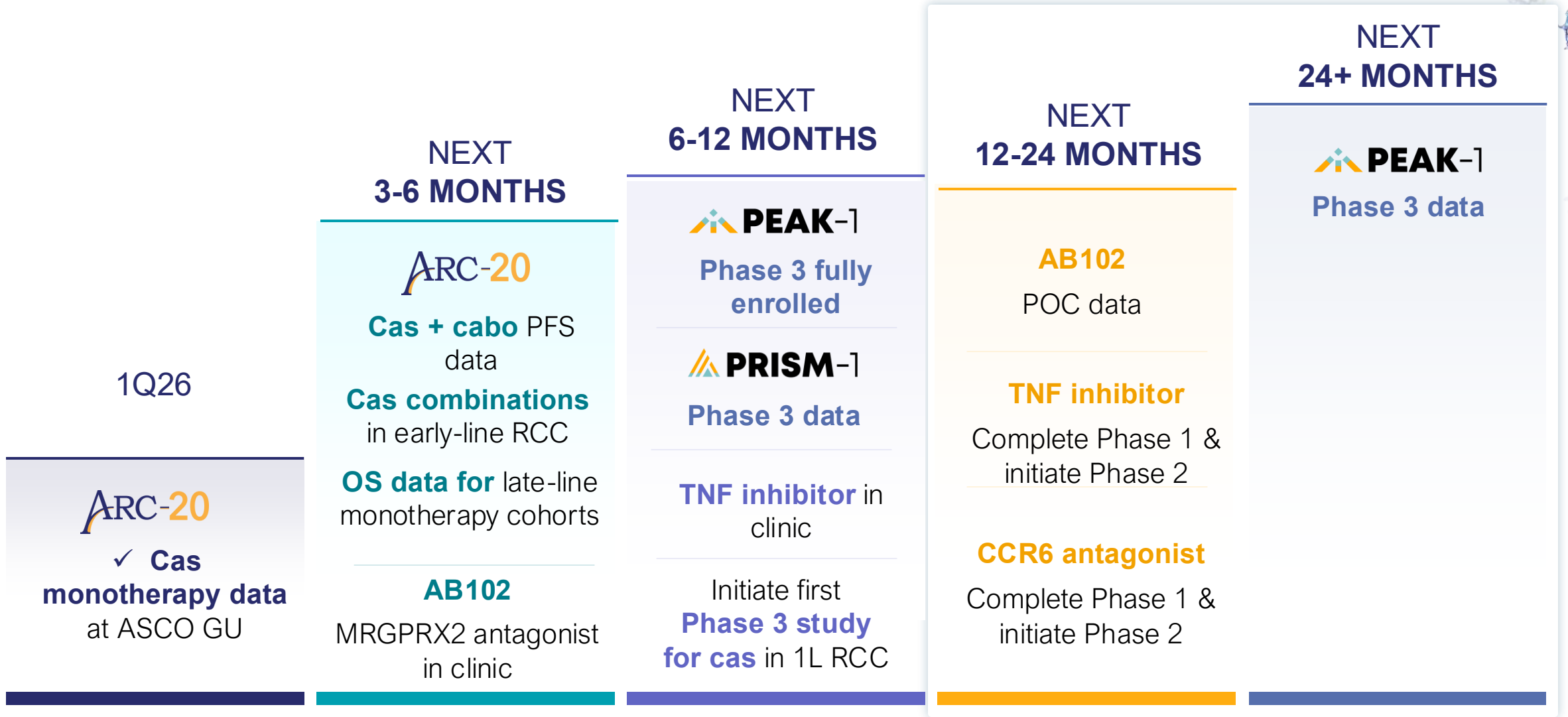
EMERGING I& PROGRAMS

- AB102, a small molecule against MRGPRX2, will enter the clinic in 3Q26; preclinical profile to be presented in oral presentation the Society for Investigative Dermatology in May
- Oral TNF inhibitor drug candidate expected to enter the clinic in early 2027
- Drug candidate for CCR6 program expected to enter the clinic mid-2027

DOMVANALIMAB PROGRAM WIND DOWN

- The Phase 3 STAR-121 study is being discontinued due to futility; ongoing wind-down of anti-TIGIT program
- The STAR-121 study also evaluated zim and chemotherapy as an exploratory endpoint. Zim plus chemotherapy performed consistently with respect to OS as compared to pembrolizumab plus chemotherapy

Arcus Milestones Will Generate Near- and Long-Term Value for Shareholders



ASCO GU Casdatifan Data

Data Based on a January 30, 2026 DCO Unless Otherwise Stated

Cohorts Evaluating Cas Mono in anti-PD-1/TKI Experienced ccRCC

DOSE ESCALATION

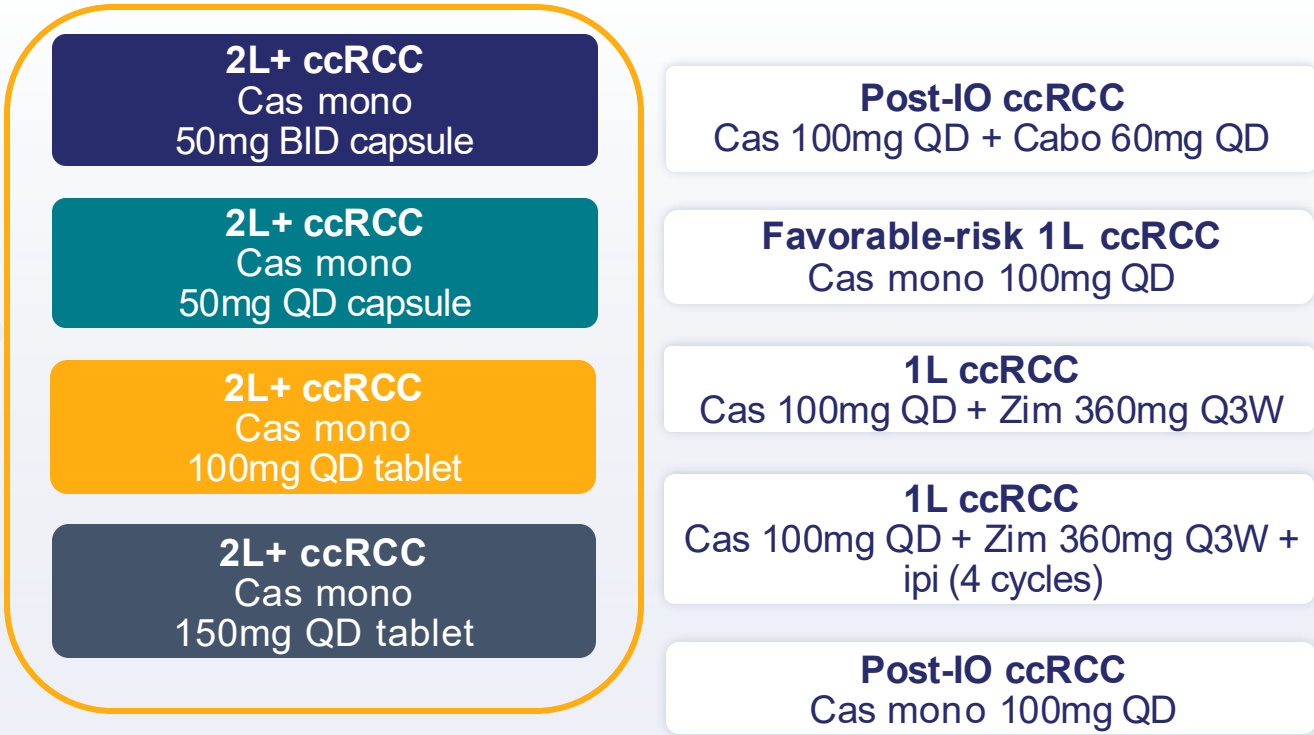
Patients with advanced solid tumors

Casdatifan monotherapy



DOSE EXPANSION

N = ~30 per cohort



To date, over 240 patients have received casdatifan across all cohorts of ARC-20

Data Have Been Consistently Robust and Have Improved Over Time, Exceeding Benchmarks



Oct.2025
Investor
Event



	5 mos med. Follow-up	12 mos med. Follow-up	17.9 mos med. Follow-up
100mg QD (n=32)	cORR: 33% mPFS: Not reached PD: 15%	cORR: 35% mPFS: Not reached PD: 16%**	cORR: 45% mPFS: 15.1mos PD: 16%**
Pooled Analysis (n=121)*	--	15 mos med. Follow-up cORR: 31% mPFS: 12.2 months PD: 19%**	20.8 mos med. Follow-up cORR: 35% mPFS: 12.2 mos PD: 19%**

* Includes all four late-line monotherapy cohorts: 50mg BID, 50mg QD, 100mg QD, 150mg QD from the ARC-20 study

** Includes two patients in the 100mg QD Tablet cohort, and also included in the pooled analysis, who had clinical progression before the first scan and therefore did not meet criteria for progressive disease per RECIST. PD based on RECIST criteria was 10% (n=3) for the 100 mg QD Tablet cohort and 17% (n=121) for the pooled analysis.

Confirmed ORR for the “Pooled” Cohort (n=121) is Now 35%

Efficacy-Evaluable Population ^a	Dose Expansion: 2L+ ccRCC					Belzutifan ²
	50mg BID (n = 31)	50mg QD (n = 28)	100mg QD (n=31)	150mg QD (n = 31)	All Pooled (n = 121)	120mg QD (n = 374)
Median Follow-Up, mos (range)	28.3 (19.9, 31.5)	25.2 (21.4, 26.7)	17.9 (11.8, 19.0)	19.8 (18.0, 21.0)	20.8 (11.8, 31.5)	18.4 (9.4–31.7)
Median Time to Response, mos	2.7	4.1	2.6	2.7	2.8	3.8
Confirmed ORR (95% CI)	26% (12, 45)	36% (19, 56)	45% (27, 64)	32% (17, 51)	35% (26, 44)	22% (18, 27)
Complete Response, % (n)	0% (0)	4% (1)	0% (0)	0% (0)	1% (1)	3% (10)
Partial Response, % (n)	26% (8)	32% (9)	45% (14)	32% (10)	34% (41)	19% (72)
Stable Disease, % (n)	55% (17)	50% (14)	39% (12)	42% (13)	46% (56)	39% (147)
Progressive Disease, % (n)	19% (6)	14% (4)	16% (5)*	26% (8)	19% (23)*	34% (126)

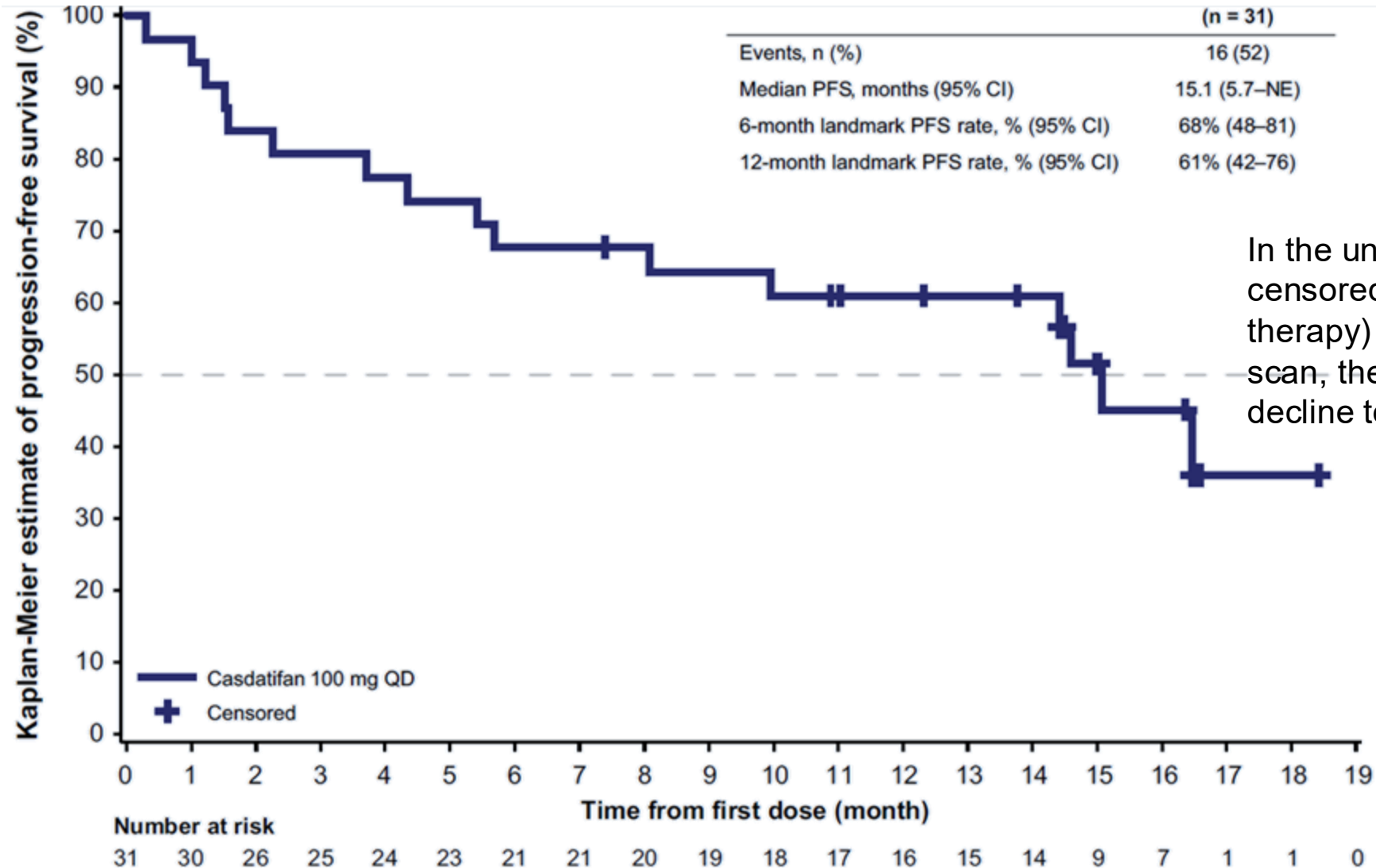
*Includes two patients in the 100mg QD Tablet cohort, and also included in the pooled analysis, who had clinical progression before the first scan and therefore did not meet criteria for progressive disease per RECIST. PD based on RECIST criteria was 10% (n=3) for the 100mg QD Tablet cohort and 17% (n=121) for the pooled analysis.

DCO date: Jan. 30, 2026

a. Efficacy-evaluable population for this expansion cohort is defined as all eligible participants who received any study treatment and have at least one post-baseline efficacy assessment, or who discontinued study treatment due to progressive disease or death, regardless of whether they had a scan.

References and acronyms on slide 40

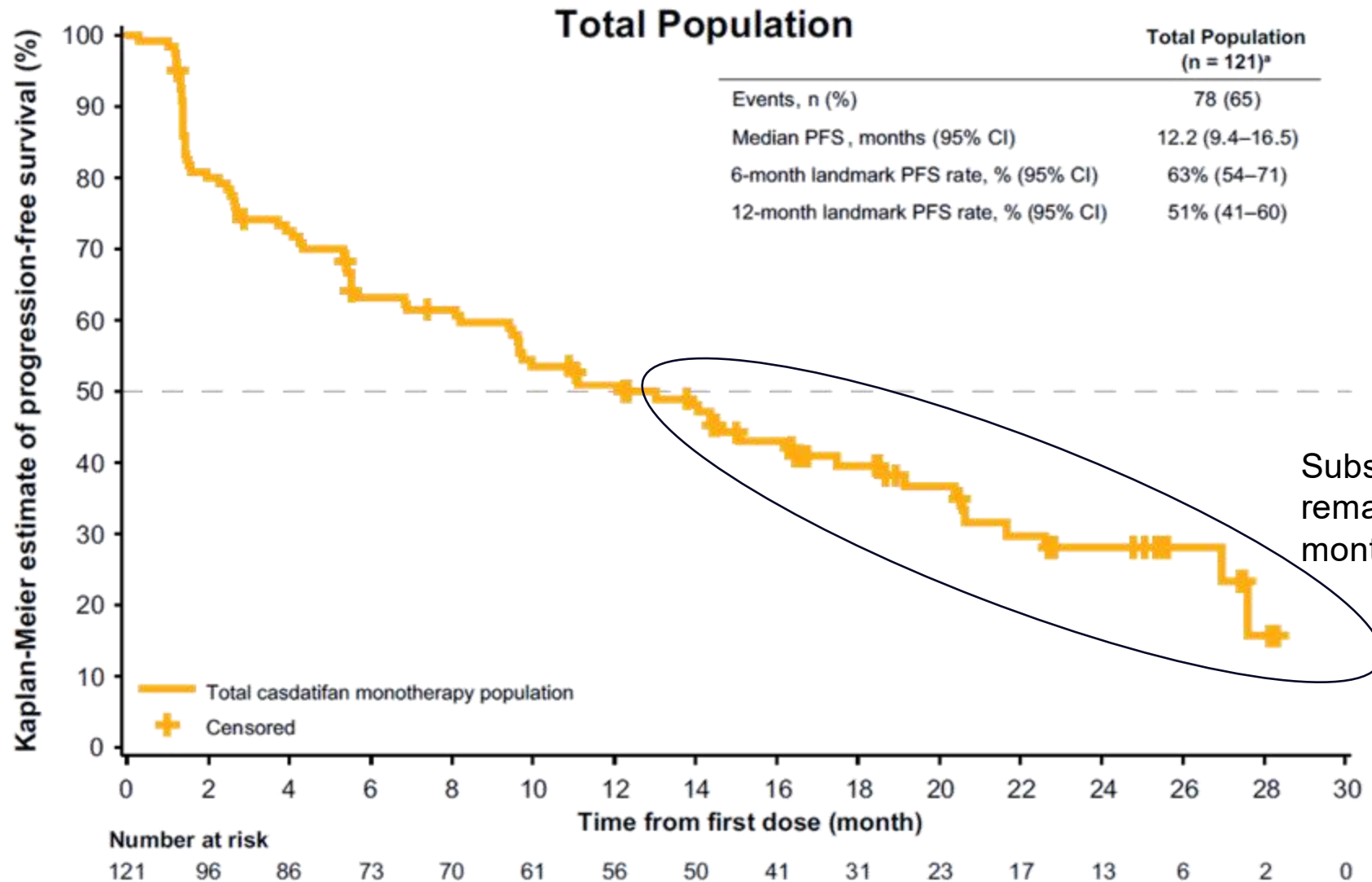
15.1 Months mPFS for Patients Treated with Casdatifan 100mg QD Phase 3 Dose and Formulation



In the unlikely scenario that *all* censored patients (still on therapy) progress at the next scan, the mPFS would only decline to 14.4 months

Median Follow Up:
17.9 months

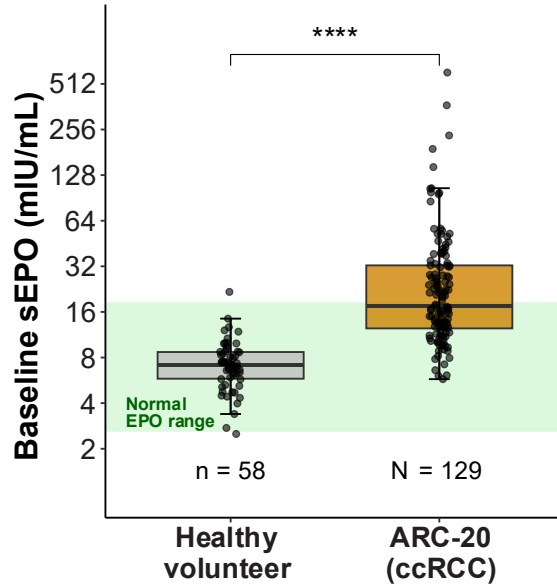
12.2 Months mPFS for Patients Treated with Casdatifan Across All Four Monotherapy Cohorts



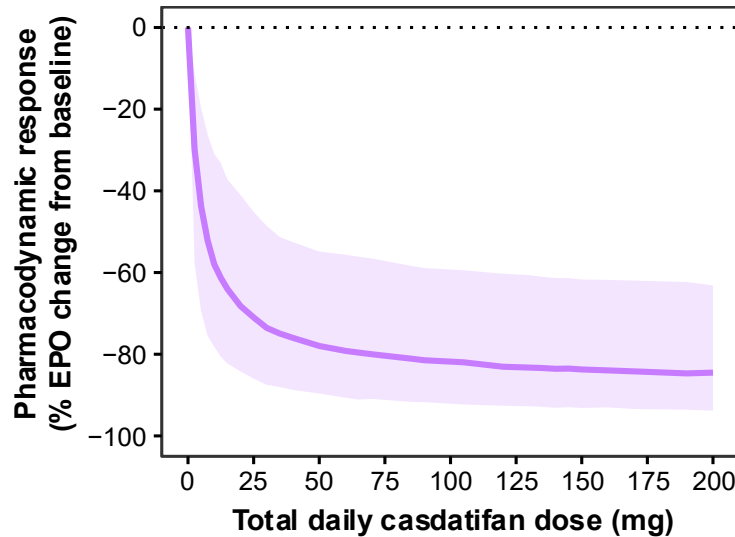
Substantial number of patients remain on treatment beyond 14 months and 24 months

Median Follow Up: 20.8 months

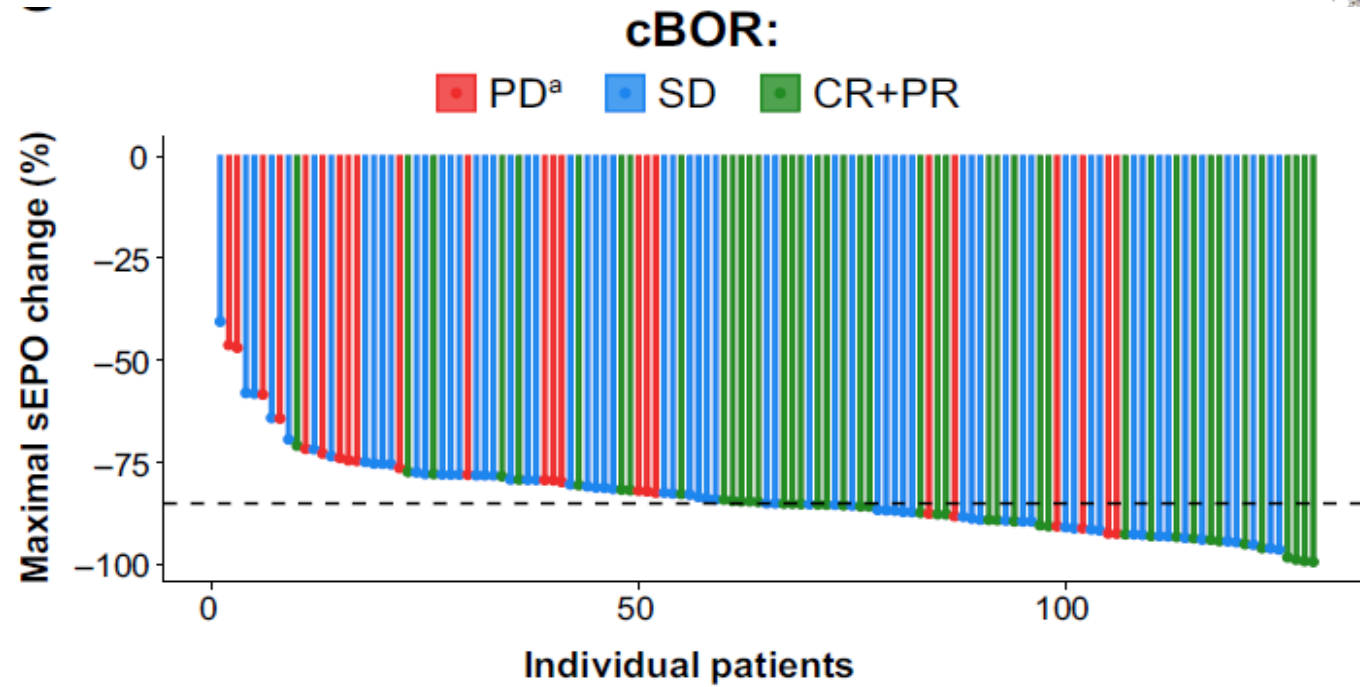
Systemic Suppression of HIF-2 α -Regulated sEPO Is Highly Correlated with Clinical Benefit from Casdatifan (ORR)



Patients with ccRCC had significantly higher baseline levels of sEPO than healthy volunteers ($P < .0001$)¹



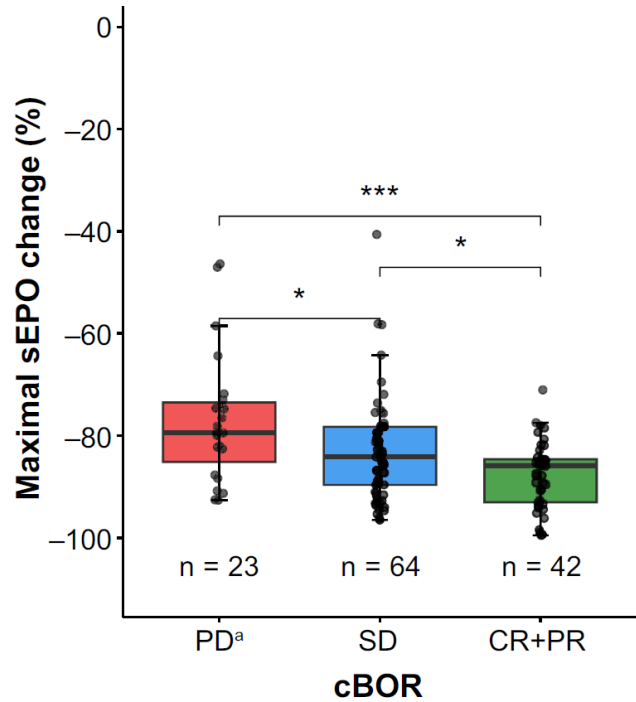
71% (92/129) of patients reached maximal sEPO reduction during the first cycle of treatment with casdatifan¹



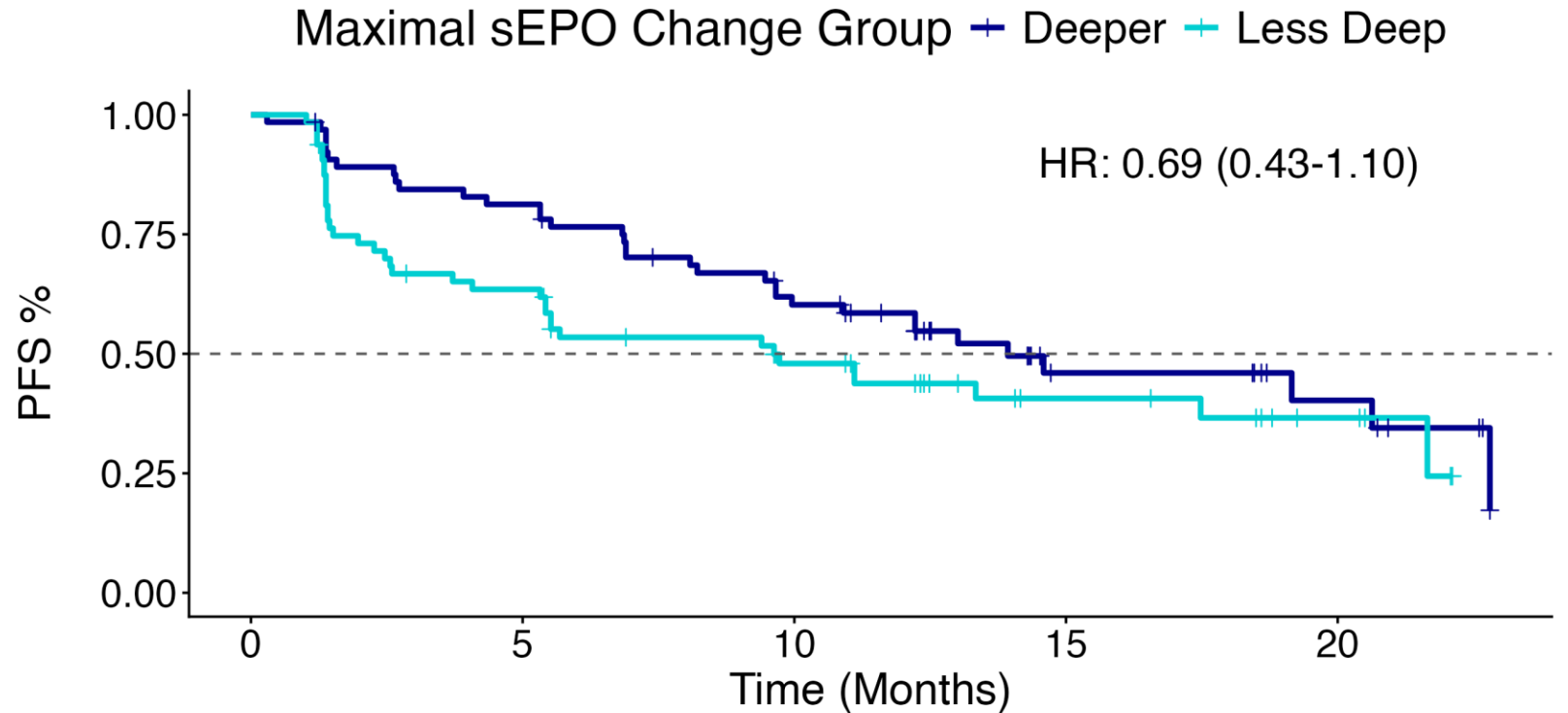
Across all doses, most patients (88/129 [68%]) experienced > 80% maximal sEPO reduction; **Deeper reductions in sEPO were highly correlated with clinical responses**¹

a. PD was defined according to RECIST v1.1 as a $\geq 20\%$ increase in the sum of diameters of target lesions relative to the smallest sum on study (including baseline, if smallest), with an absolute increase of ≥ 5 mm. The appearance of 1 or more new lesions was also considered progression.

Systemic Suppression of HIF-2 α -Regulated sEPO Is Highly Correlated with Clinical Benefit from Casdatifan (PFS)



The degree of maximal sEPO reduction was significantly greater in patients who achieved CR, PR, or SD compared with those with PD ($P < .05$)¹



	At Risk	5	10	15	20
Deeper	65	52	36	12	7
Less Deep	64	39	25	11	5

Maximal sEPO reduction was associated with longer PFS¹

a. PD was defined according to RECIST v1.1 as a $\geq 20\%$ increase in the sum of diameters of target lesions relative to the smallest sum on study (including baseline, if smallest), with an absolute increase of ≥ 5 mm. The appearance of 1 or more new lesions was also considered progression.

References and acronyms on slide 40

Casdatifan Data Exceed All PFS Benchmarks for Both Monotherapy HIF-2 α Inhibition (Belz) and TKIs

SETTING	TRIAL	REGIMEN	cORR	mPFS
3L+	ARC-20 (Phase 1b/2)	Cas (100mg QD)	45%	15.1m
		Cas (Pooled)	35%	12.2m
	LITESPARK-005 (Phase 3) ³	Belz	22%	5.6m
2L+	TIVO-3 (Phase 3) ⁶	Tivo	18%	5.6m
	METEOR (Phase 3) ⁷	Cabo	17%	7.4m
	Study 205 (Phase 2) ⁸	Lenva	27%	7.4m
IO- Experienced (1L/2L)	AXIS (Phase 3) ⁹	Axi	19%	6.7m
	CONTACT-03 (Phase 3) ¹⁰	Cabo	41%	10.8m
	CANTATA (Phase 3) ¹¹	Cabo	28%	9.3m

EARLIER-LINE PATIENT POPULATIONS

DCO date for casdatifan: Jan. 30, 2026

Data are not from head-to-head studies. Cross-trial data interpretation should be considered with caution as it is limited by differences in study population, sample size, inclusion and exclusion criteria and many other factors.

References and acronyms on slide 40

Casdatifan Was Well Tolerated in All Cohorts, With a Comparable Safety Profile to That of Belzutifan

Safety-Evaluable Population	100mg QD (n=32)	Pooled (n=127)	Belzutifan (LITESPARK-005) ²
Anemia, n (%)			Anemia:
All grades	29 (91)	117 (92)	All grade: 83%
Grade ≥3 related to casdatifan	8 (25)	52 (41)	Grade ≥3: 33%
Related to casdatifan leading to interruptions	9 (28)	45 (35)	
Leading to dose reductions	3 (9)	18 (14)	
Leading to discontin.	0	0	
Hypoxia, n (%)			Hypoxia:
All grades	5 (16)	23 (18)	All Grade: 15%
Grade ≥3 related to casdatifan	3 (9)	14 (11)	Grade ≥3: 11%
Related to casdatifan leading to interruptions	3 (9)	18 (14)	
Leading to dose reductions	1 (3)	9 (7)	
Leading to discontin.	1 (3)	3 (2)	

DCO date for casdatifan: Aug. 15, 2025; safety profile remained consistent at the Jan. 30, 2026 DCO
 Data are not from head-to-head studies. Cross-trial data interpretation should be considered with caution as it is limited by differences in study population, sample size, inclusion and exclusion criteria and many other factors.

References and acronyms on slide 40

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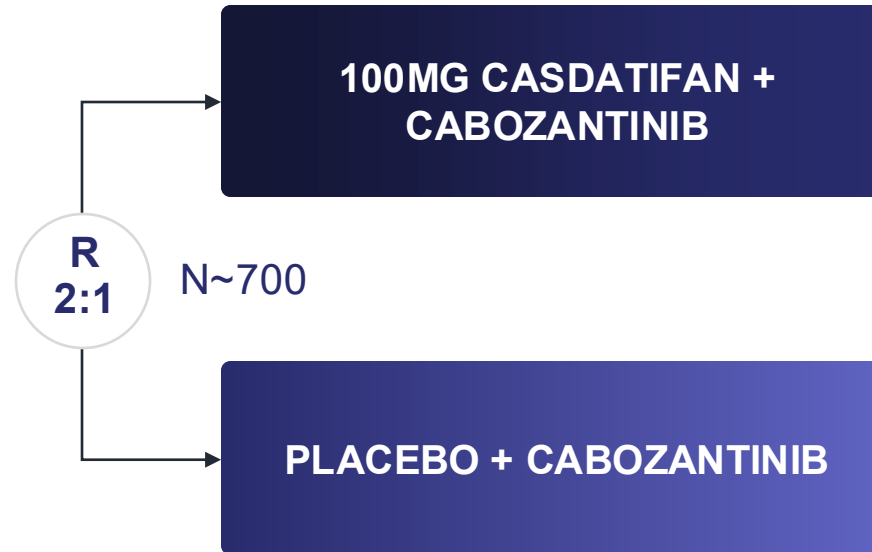
Casdatifan Development Strategy

First Phase 3 Study Evaluating a Differentiated TKI Combination in Post-IO ccRCC is Enrolling

Only Phase 3 study evaluating a HIF-2 α inhibitor with cabozantinib, the most widely used TKI in ccRCC

PATIENT POPULATION:

- Unresectable, locally advanced or metastatic ccRCC
- Have had prior anti-PD-1/ PD-L1 (either in adjuvant or 1L metastatic setting)
- Have not received cabozantinib



PRIMARY ENDPOINT

- PFS

KEY SECONDARY ENDPOINTS

- OS
- ORR, DOR, DCR

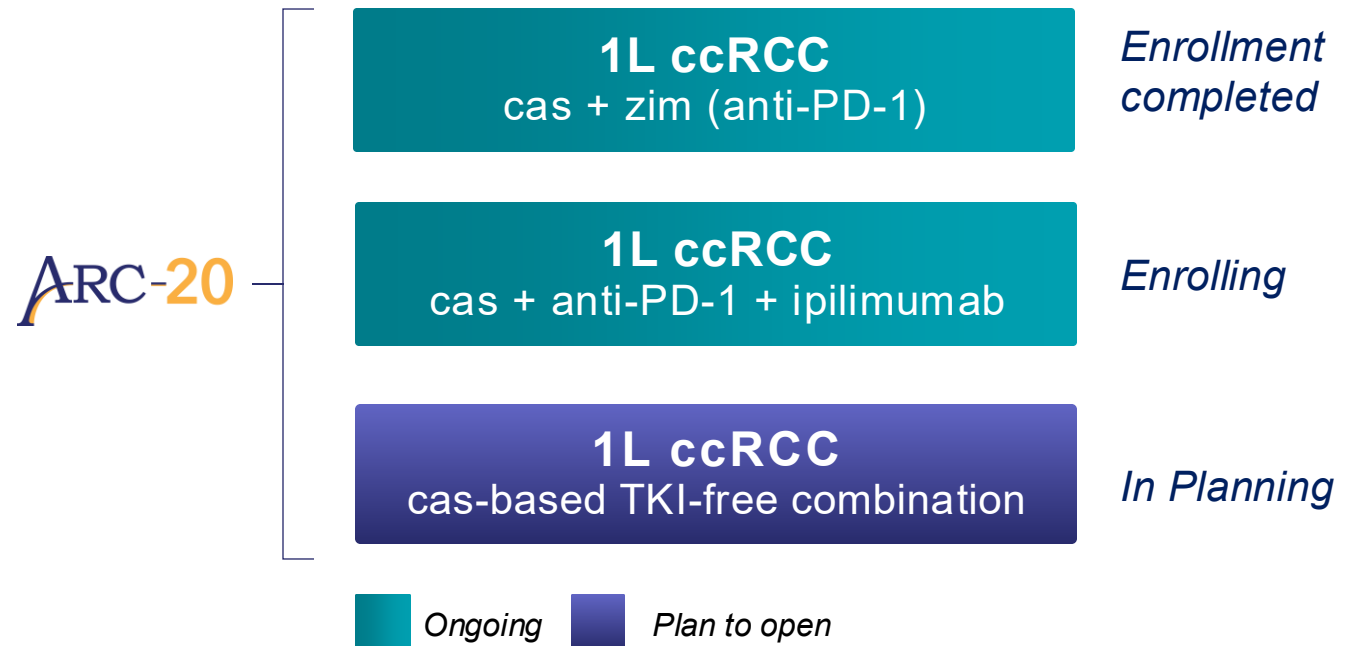
Goal Is to Fully Enroll by Year-End

1L TKI-Free Approach Is Enabled by Cas's Low Rate of Primary Progression

ARC-20 Data To Date Show a Consistently Low Rate of Primary PD, Avoiding the Need for a TKI

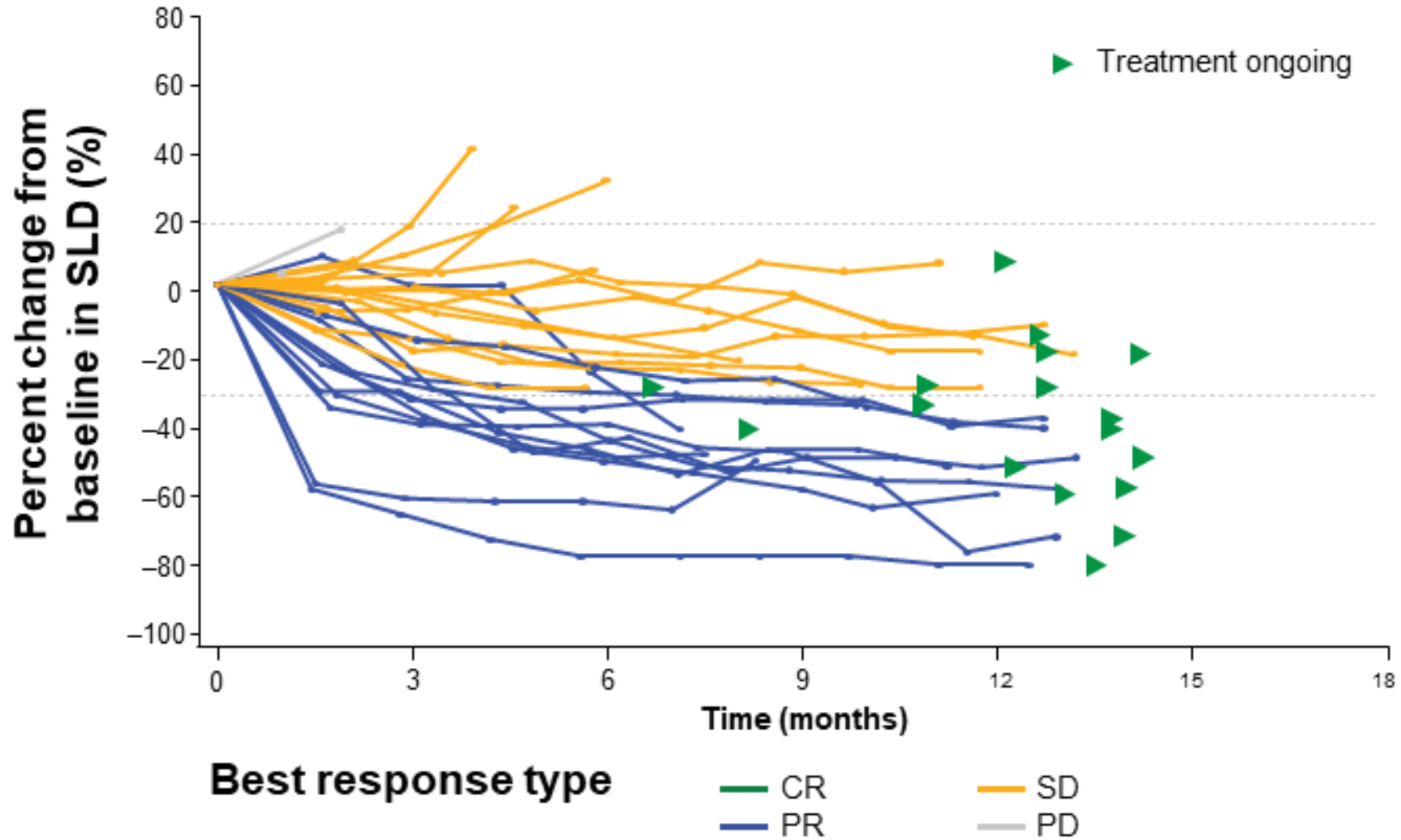
	Efficacy-Evaluable Population*	Primary PD Rate % (n)
2L+ ccRCC	cas 100mg QD (n=31)	16% (5) ¹
	Monotherapy pooled (n=121)	19% (23)
	cas + cabo (n=43)	5% (2)
1L+ ccRCC	1L cas + zim (n=30)	7% (2)
	1L cas mono, favorable risk (n=22)	0% (0)
	cas mono, post-IO, TKI naive (n=29)	3% (1)

Three TKI-Free Cohorts in the 1L Setting Will Inform Initial 1L Phase 3 Strategy for Cas with the Goal of Initiation by YE:26

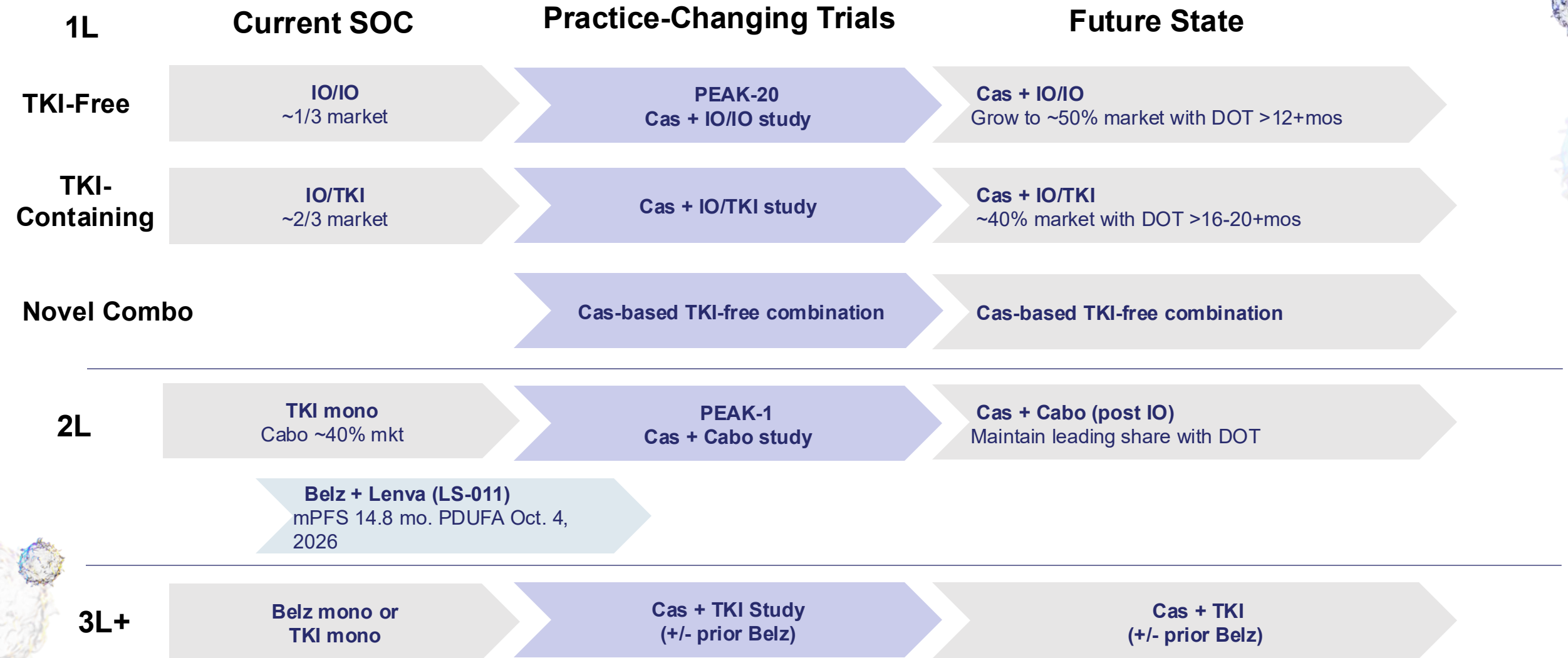
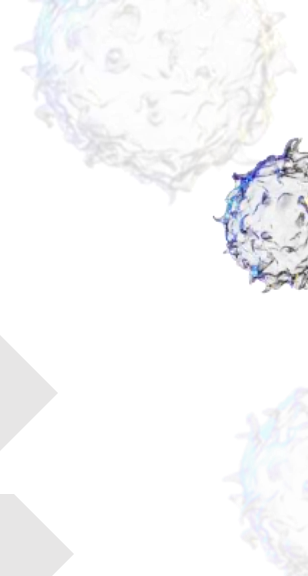


Primary PD rate: The percent of patients whose disease progressed at or before their first post-baseline scan.
 *All data from ongoing ARC-20 cohorts spotfire data
 References and acronyms on slide 40

Half of Patients (17 of 31) Remain on Treatment with Disease Control in the 100mg Cohort of ARC-20



Future State of RCC Treatment: Every Patient to Have Opportunity to Benefit from Cas

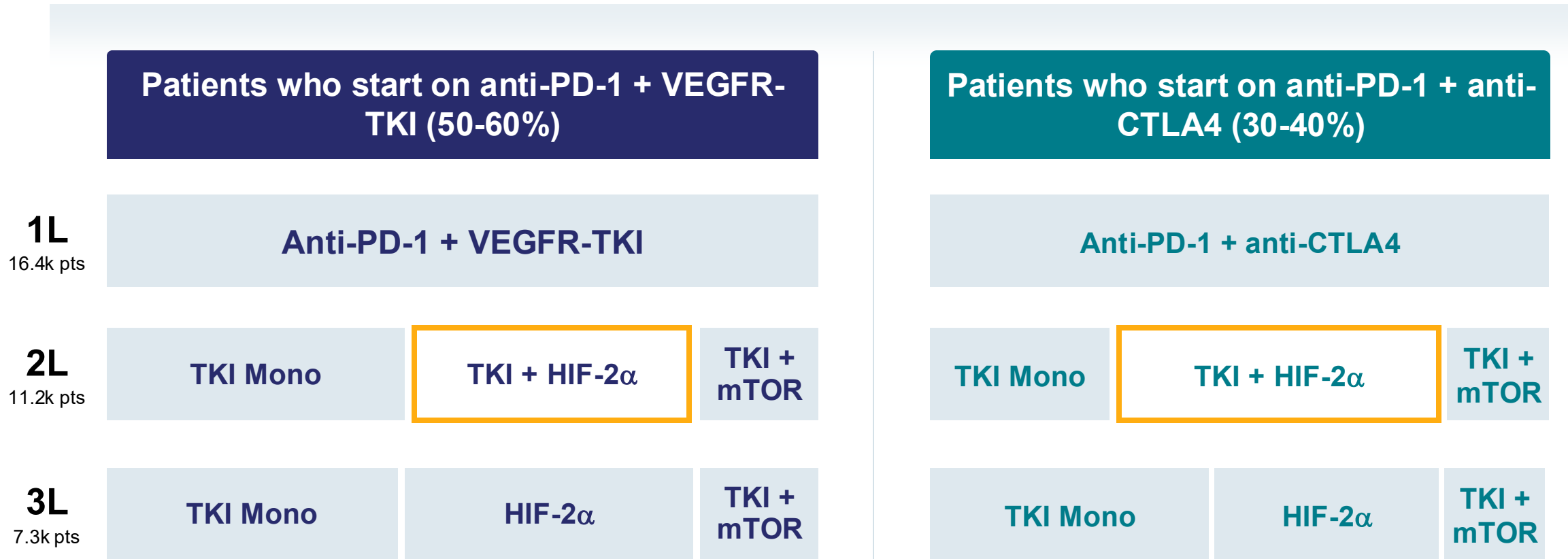


The background of the slide is a dark blue field filled with numerous spherical cells. These cells are illuminated with various colors of fluorescence, including green, yellow, and purple, highlighting their internal structures and membranes. The cells vary in size and focus, creating a sense of depth and biological activity.

Casdatifan Market Opportunity

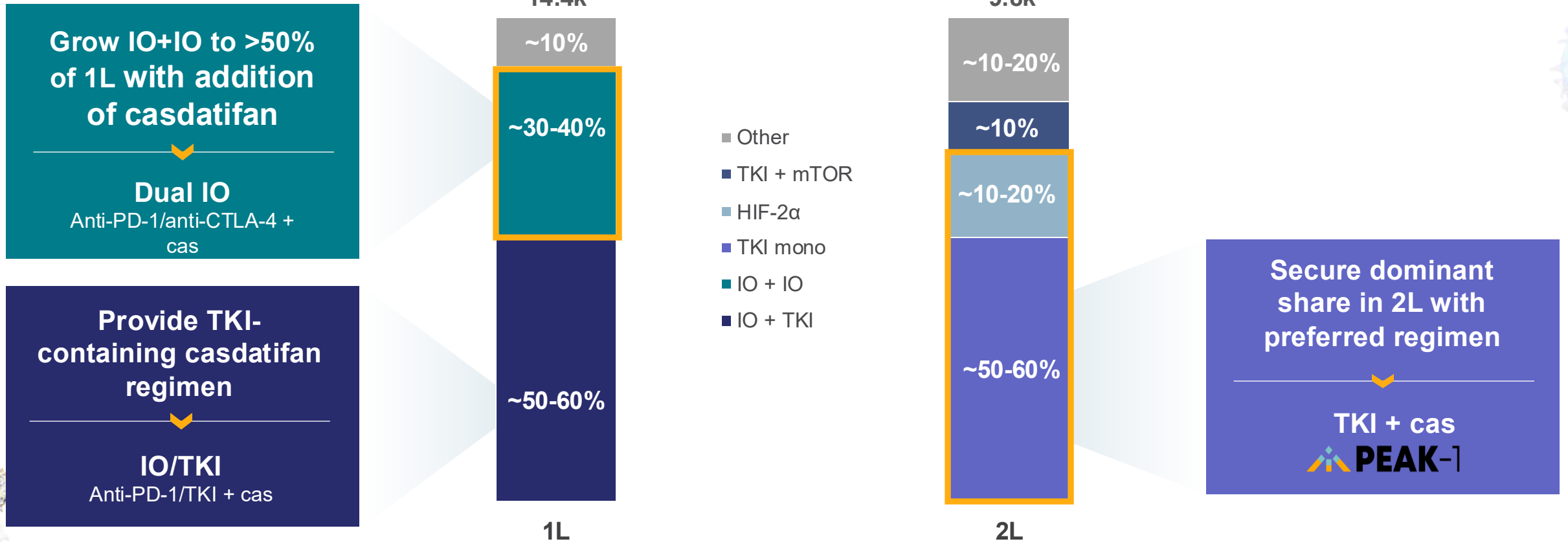
Metastatic ccRCC Treatment Algorithm (assumes LS-011 approval)

~20% of 1L patients have received adjuvant treatment



\$5B+ Opportunity for Casdatifan in 1L & 2L ccRCC Market

2026 US ccRCC Drug Treatable Incidence and Market Share

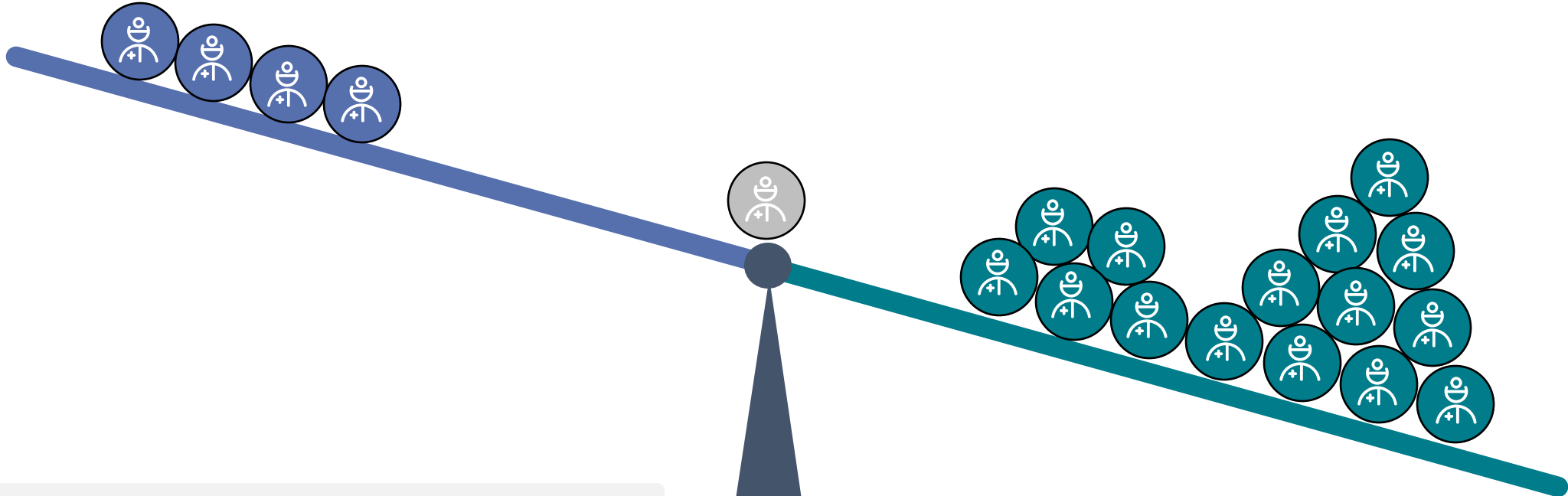


TOTAL ADDRESSABLE MARKET
~\$9B Growing to \$13B by 2030

Sources - Epi: DRG | Share: Arcus primary research, US March 2026 (n=50)

References and acronyms on slide 40

Oncologists Clearly Preferred the 1L Cas + Anti-PD1/anti-CTLA4 Regimen Over the 1L Cas + Anti-PD1/TKI Regimen When Presented With Both Options



Pros

- ✓ Preferred for patients with rapidly progressing disease

Cons

- Uses 3 MoAs in 1L, limiting sequencing options
- Concerns related to AEs from TKIs

Pros

- ✓ Familiarity with anti-PD1+anti-CTLA4 combination across other tumors
- ✓ Allows for sequencing of different MoAs in 2L+
- ✓ Anti-PD1+anti-CTLA4 currently most used regimen in 1L

Cons

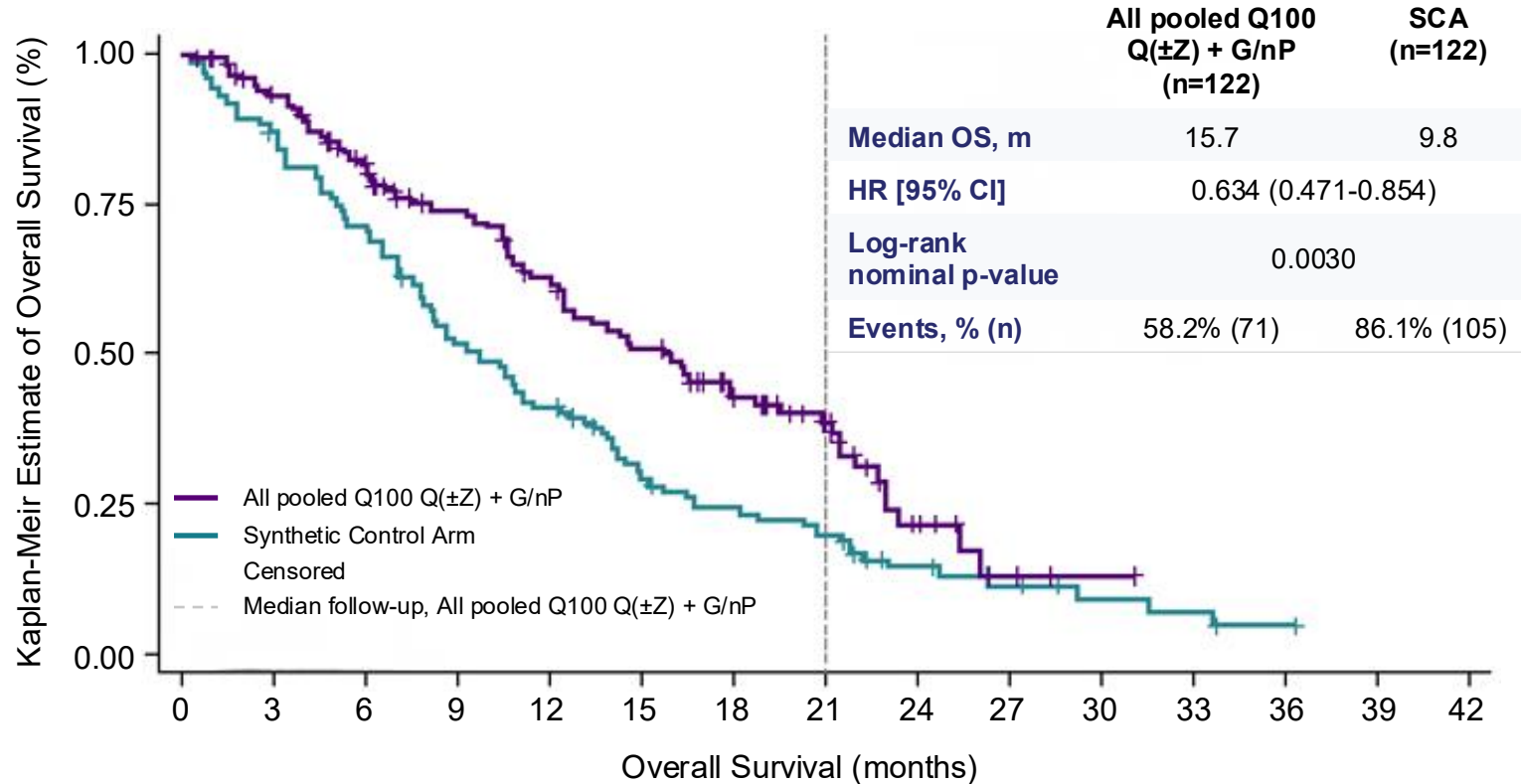
- May not be suitable for patients w/ rapidly progressing disease



Quemliclustat in Pancreatic Cancer

Quemli Could be the First Transformative Therapy for All-Comer 1L Pancreatic Cancer in 30+ Years

PHASE 1 STUDY SHOWED 5.9 MONTH mOS IMPROVEMENT VS G/nP¹²



NUMBER OF PATIENTS AT RISK	0	3	6	9	12	15	18	21	24	27	30	33	36
All pooled Q100 Q(±Z) + G/nP	122	108	89	72	59	47	34	23	8	3	1	0	
Synthetic Control Arm	122	104	85	61	49	32	26	21	12	7	4	3	1

PRISM-1

**Phase 3 trial in 1L PDAC
Enrollment completed**

~\$4B

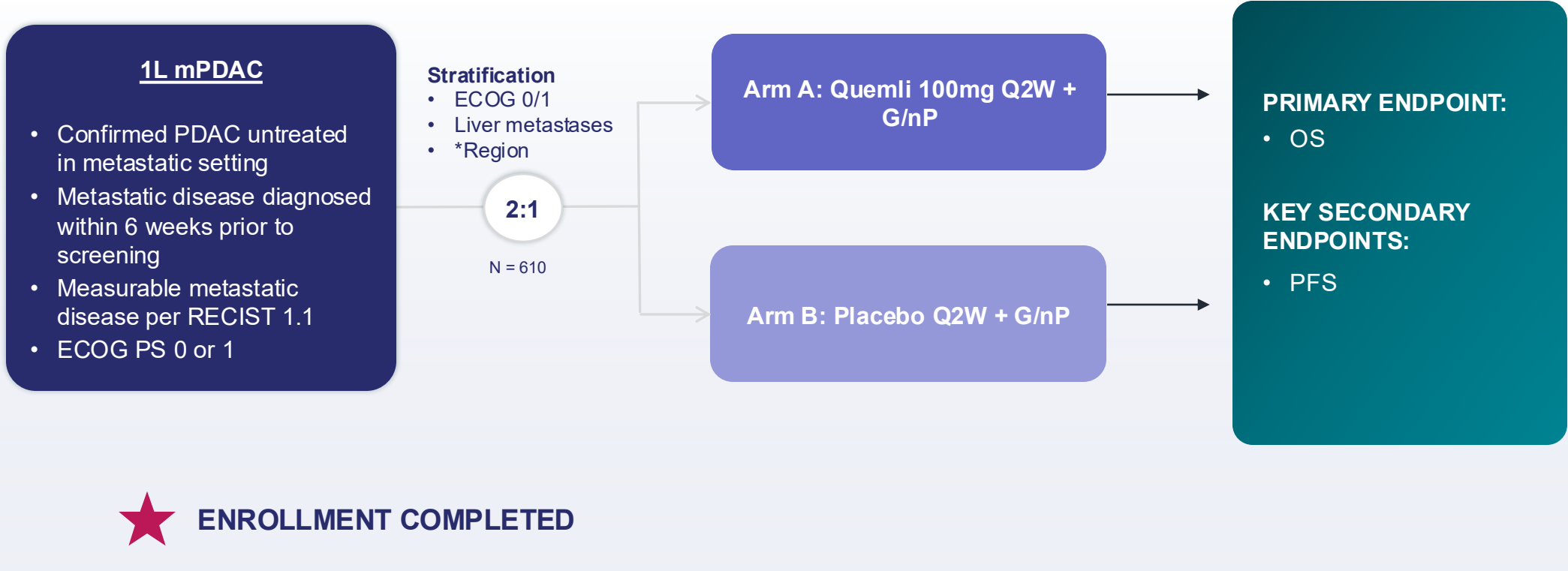
Market Potential

**Readout
in 1H 2027**

GnP data constructed into a synthetic control arm, on a post-hoc basis, from Phase 2 and 3 clinical studies in 1L metastatic pancreatic cancer setting

References and acronyms on slide 40

Phase 3 Study of Quemli + Chemo in 1L Metastatic PDAC



Our Emerging I&I Portfolio

I&I Small-Molecule Strategy is Targeting Validated Blockbuster Biologics

IN-HOUSE EXPERTISE IN IMMUNOLOGY

has been a core aspect of our discovery group since Arcus's founding

**MINIMIZE
BIOLOGICAL RISK**
by leveraging validated mechanisms with applications to common diseases with large addressable populations

2-PRONG I&I STRATEGY:

- Small-molecule improvements of cytokine-targeted therapeutics
- Target immune cell types that play key roles in human disease and have been historically “under-studied”

I&I DRUG DISCOVERY PORTFOLIO

TARGET	MODALITY	DISEASE AREA	STATUS
AB102 (MRGPRX2)	SM	CSU, AD	FIH Expected in 3Q26
TNF (TNFR1)	SM	RA, Psoriasis, IBD	FIH Expected Early 2027
CCR6	SM	Psoriasis	Advanced Discovery
CD89	mAb	RA	Advanced Discovery
CD40L	SM	SLE; MS	Discovery

Potential Best-in-Class AB102 MRGPRX2 Antagonist to Treat Atopic Skin Diseases

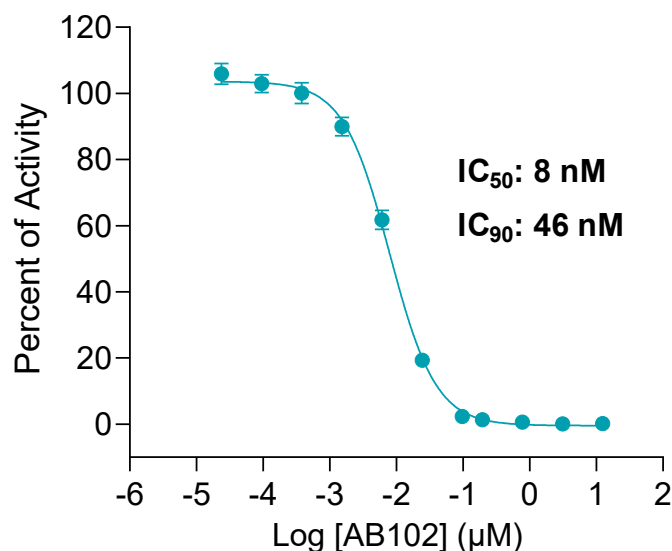
Validated biology
with multi-billion
dollar potential

- Approved biologics are highly successful and effective in treating AD and CSU, etc. and generate >\$15B in LTM sales¹³
- Anti-IgE (e.g., omalizumab) and anti-IL-4R (e.g., dupilumab) are not sufficient to address clinical need in CSU and/or AD

PROGRAM STATUS:
Expect FIH in 3Q26

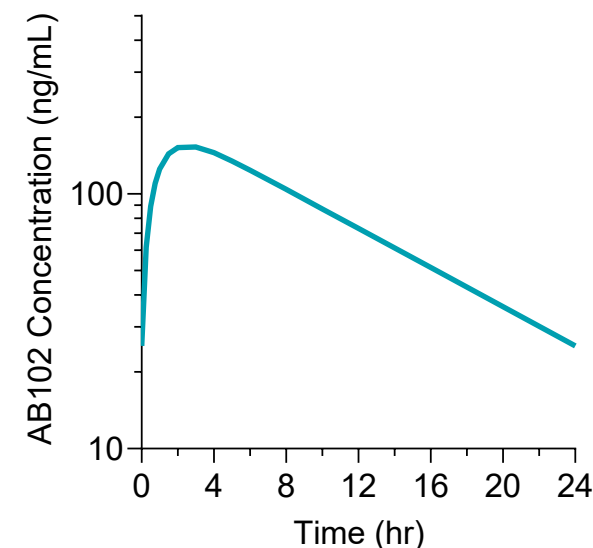
OPPORTUNITY FOR IMPROVEMENT

Mast cell (LAD2) degranulation
(CD107a) in 100% human serum



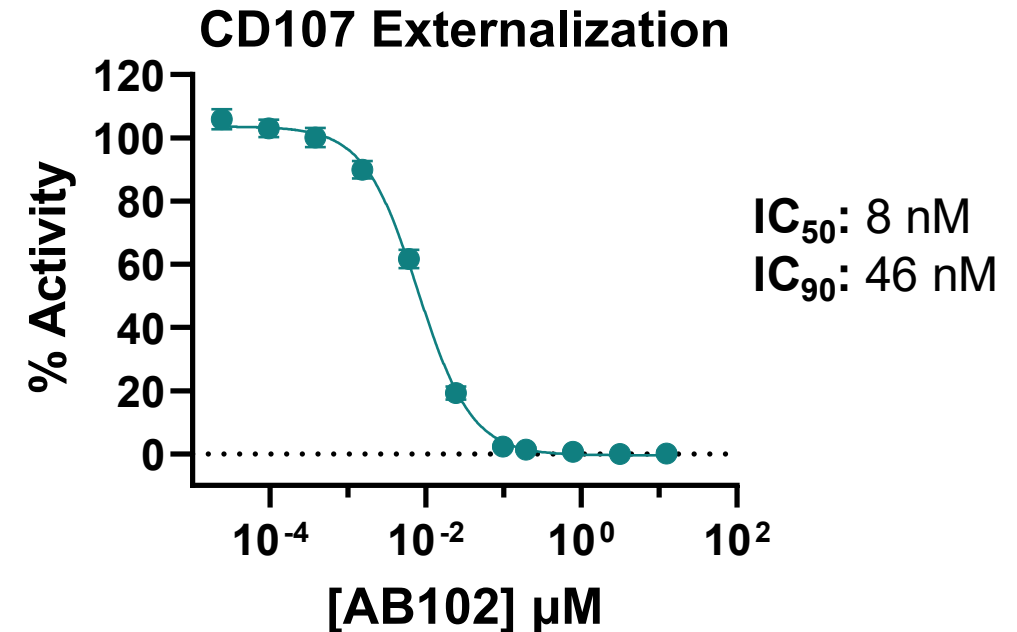
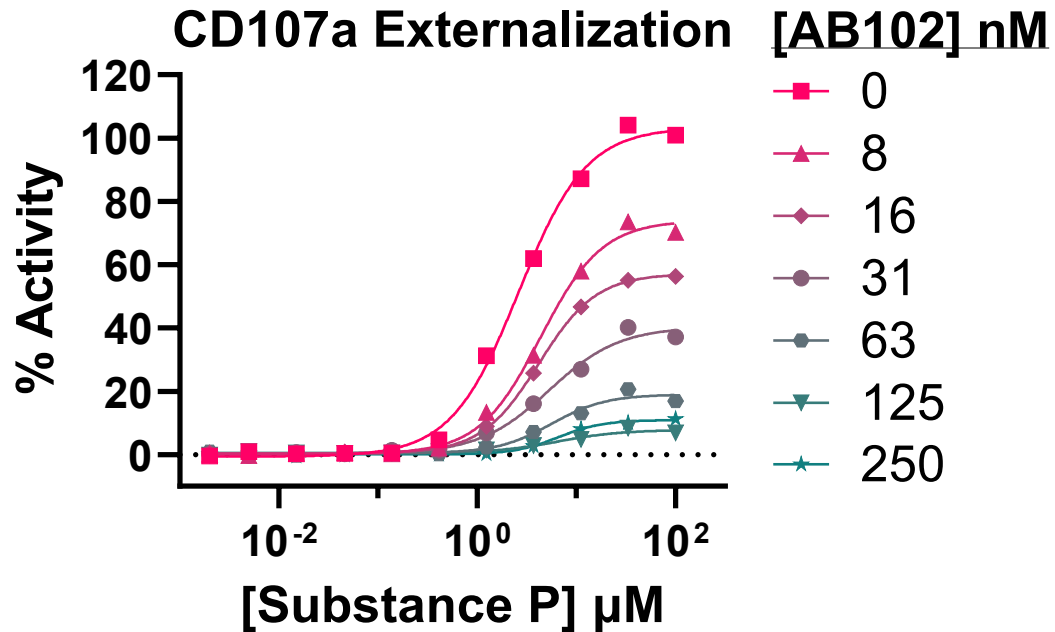
Improved potency/PK relative to early small-molecule entrants into the clinic

Modeled steady-state
human PK



Based on the predicted human PK and potency of our leading molecule, **required clinical exposures could be >90% lower** than those associated with the leading small-molecule competitor in the clinic

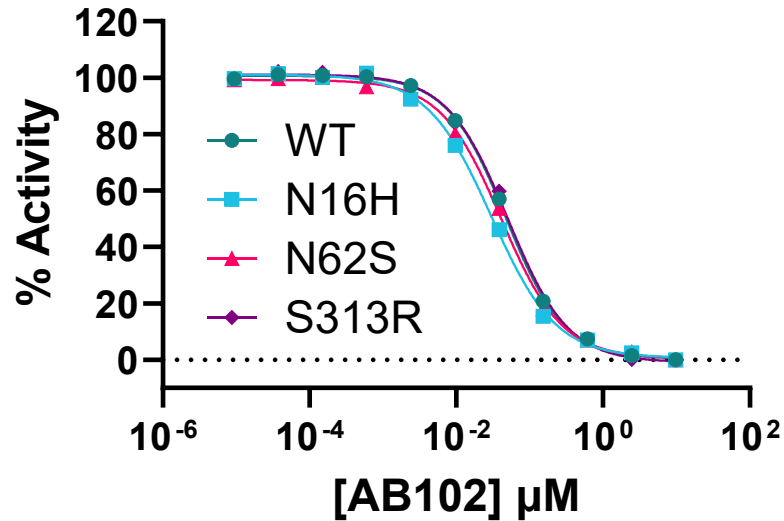
AB102 is a Potent and Selective Small Molecule MRGPRX2 Antagonist¹⁴



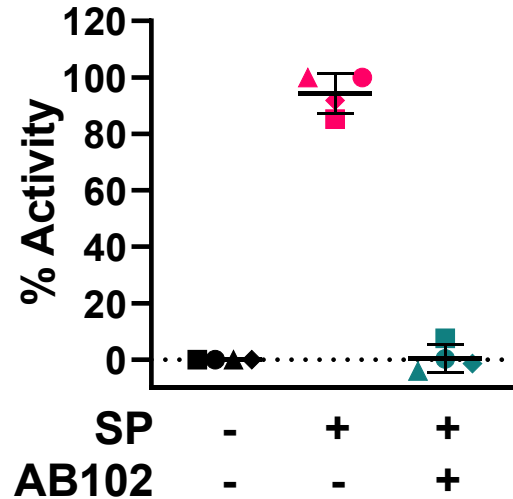
- AB102 binds MRGPRX2 in a reversible manner with an insurmountable inhibitory profile
- AB102 potently inhibits degranulation with an IC₉₀ of 46 nM in 100% human serum

AB102 is a Potent and Selective Small Molecule MRGPRX2 Antagonist¹⁴

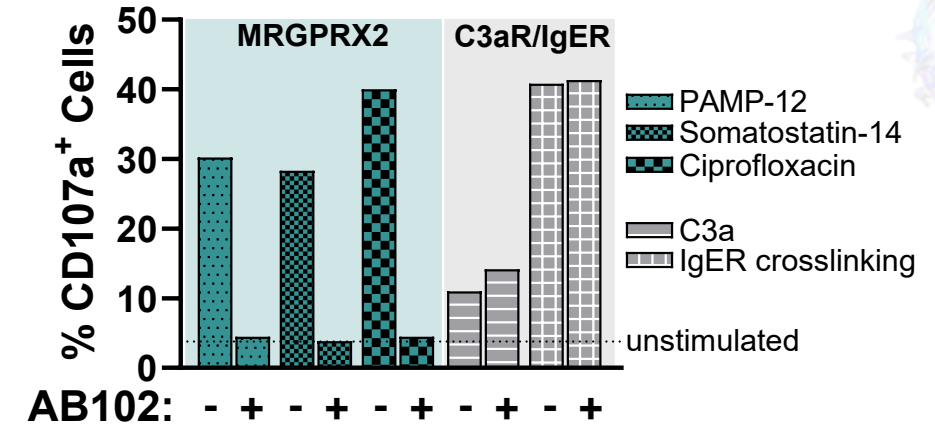
**CHO-K1 Expression
IP-1 Accumulation**



**Human Skin Mast Cells
 β -hexosaminidase Release**



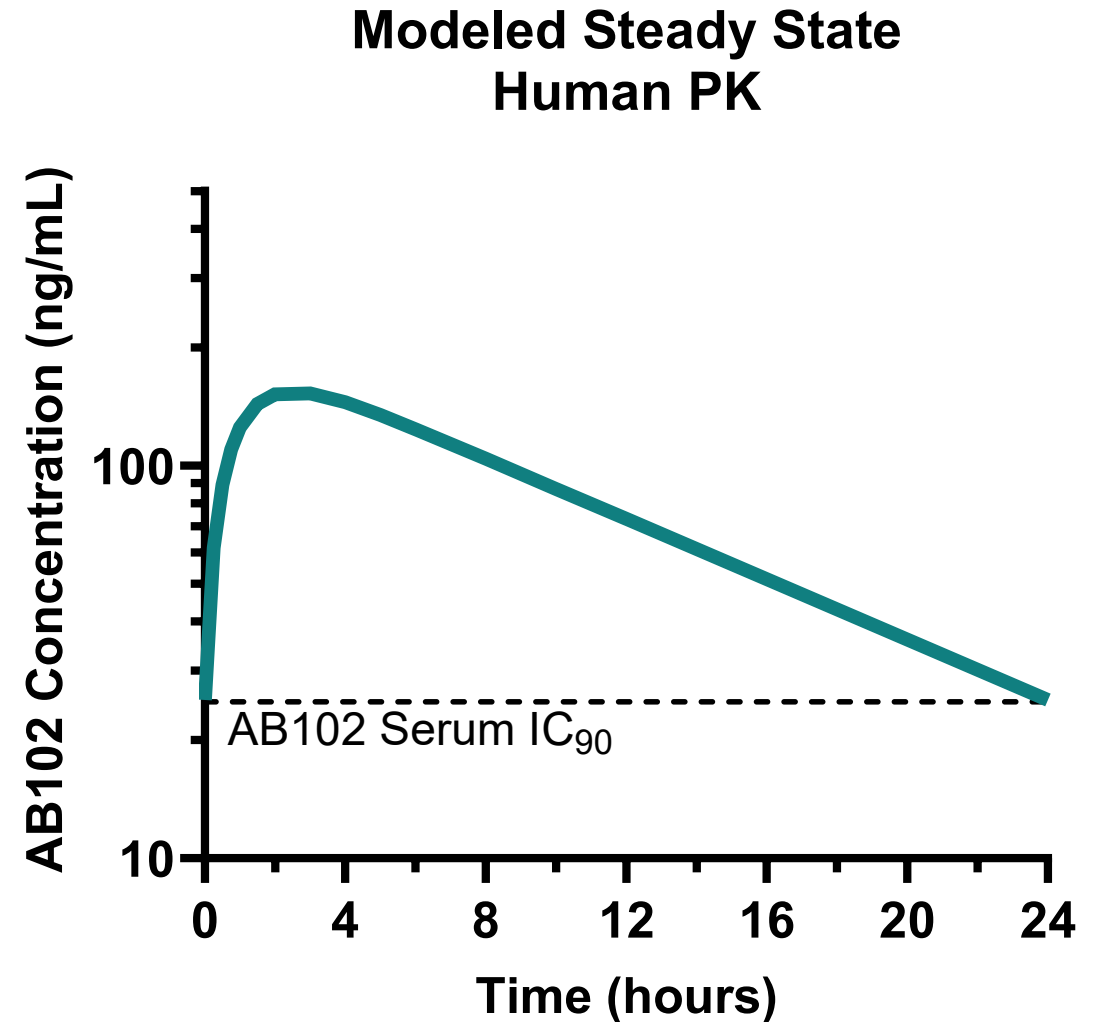
**LAD2 Cells
CD107a Externalization**



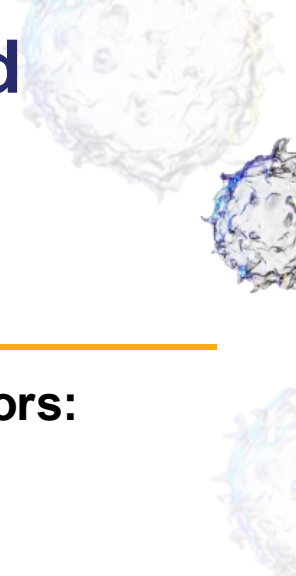
- AB102 maintains full activity against common MRGPRX2 variants
- AB102 inhibits MRGPRX2 activity in primary skin mast cells
- AB102 is active against MRGPRX2 when activated by a variety of agonists

AB102 Predicted Human PK Supports MRGPRX2 Inhibition at Low Systemic Exposures¹⁴

- ✓ AB102 potently inhibits MRGPRX2 activity under physiologically relevant conditions
- ✓ Due to this potency, we expect maximal pharmacological activity at relatively low plasma levels of compound
- ✓ The totality of preclinical data indicate AB102 is suitable for once-daily oral dosing in chronic mast cell-driven diseases



Small-molecule Inhibitors of TNF with Potentially Improved Efficacy/Safety Relative to anti-TNF Biologics



VALIDATED BIOLOGY WITH MULTI-BILLION \$ POTENTIAL

- Anti-TNF antibodies are among the **most successful biologic drugs** ever developed
- Humira® was the world's top-selling drug for **nearly a decade**, with peak sales over \$20B^{15,16}

PROGRAM STATUS:
Expect FIH in early 2027

OPPORTUNITY FOR IMPROVEMENT

Anti-TNF antibodies block TNF signaling at 2 receptors:

- TNFR1 (pro-inflammatory)
- TNFR2 (pro-Treg – blocking can drive inflammation)

As a result, TNF antibodies can drive a fraction of patients to develop “paradoxical inflammation” (e.g., psoriasis)

Small-molecule disruptors of TNF **selectively block only the TNFR1** biology, with potential for efficacy & better safety

Opportunity for molecules with better potency / human PK, relative to early small-molecule entrant into the clinic

Small-molecule CCR6 Antagonists as Potential Therapeutic Alternatives to anti-IL-17 Biologics - Psoriasis

VALIDATED BIOLOGY WITH MULTI-BILLION \$ POTENTIAL

- Anti-IL-17 antibodies have revolutionized the treatment of psoriasis and other skin diseases
 - Cosentyx® has generated \$6.7B in LTM sales¹⁷
- There are currently no orally available options for dealing with the inflammatory effects of IL-17

PROGRAM STATUS:
Expect FIH by mid-2027

OPPORTUNITY FOR IMPROVEMENT

- CCR6 inhibition creates opportunity to **interfere with a group of cytokines** (beyond IL-17) produced by key inflammatory cells (e.g., $\gamma\delta$ T cells)
 - IL-17 is key in fighting certain infections; anti-IL-17 therapy brings with it increased risk of infection
- Small-molecule CCR6 antagonists may provide **greater flexibility for managing safety signals** secondary to IL-17 inhibition

Chemokine field **notoriously difficult to drug** (requirements for constant target engagement).

- Arcus scientists have one of the **best records in the industry**

Appendix

References and Acronym Key

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2. Albiges L. et al. 2023 (LITESPARK-005)
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15. [2022 Annual Report](#). Abbvie. Accessed May 3, 2026.
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1H first half	Cabo cabozantinib	ECOG Eastern Cooperative Oncology Group	m months	ORR objective response rate	Q3 third quarter	SD stable disease
1L first-line	Cas casdatifan	EPO erythropoietin	mAb monoclonal antibody	OS overall survival	Q2/3W every 2/3 weeks	SLE systemic lupus erythematosus
2H second-half	ccRCC clear cell renal cell carcinoma	FIH first-in-human	Med median	PD primary progression	QD once daily	SM small molecule
2L second-line	CI confidence interval	G/nP gemcitabine/nab-paclitaxel	mg milligrams	PDAC pancreatic ductal adenocarcinoma	quemli quemliclustat	SOC standard of care
3L third-line	cORR confirmed overall response rate	HR hazard ratio	MOA mechanism of action	PD-L1 programmed death-ligand 1	R randomized	TKI tyrosine kinase inhibitor
AD atopic dermatitis	CR complete response	I&I inflammation & immunology	Mono monotherapy	PFS progression-free survival	R&D research & development	Tivo tivozanib
AE adverse event	CSU chronic spontaneous urticaria	IBD inflammatory bowel disease	mos months	Ph Phase	RA rheumatoid arthritis	YE year-end
Axi axitinib	DCO data cutoff	IO immunotherapy	mOS median overall survival	PK pharmacokinetics	RCC renal cell carcinoma	Z zimberelimab
B billion	DCR disease control rate	Ipi ipilimumab	mPFS median progression-free survival	PR partial response	RECIST Response Evaluation Criteria in Solid Tumors	zim zimberelimab
belz belzutifan	DOR duration of response	K thousand	MRGPRX2 mas-related G protein-coupled receptor member X2	POC proof of concept	SCA synthetic control arm	
BID twice daily	DoT duration of treatment	Lenva lenvatinib	MS multiple sclerosis	Q quemliclustat	sEPO serum erythropoietin	