



Pioneering Today
with a different kind of medicine

Transforming Tomorrow
for patients who need us

May 2024

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Safe harbor and forward-looking statements



This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995 that involve substantial risks and uncertainties, including statements regarding our expectation of bringing the first PROTAC® protein degrader to market in partnership with Pfizer; the plans for and anticipated timings related to our planned clinical trials, including second-line Phase 3 clinical trials of vepdegestrant in combination with palbociclib and potentially other CDK4/6 inhibitors, a first-line Phase 3 clinical trial of vepdegestrant in combination with Pfizer's CDK4 inhibitor (PF-07220060), a Phase 3 clinical trial for ARV-766 as a monotherapy, and a Phase 1 dose escalation trial of ARV-393 (BCL6); the closing of the transaction with Novartis; our plans and timing related to commercial launch of vepdegestrant; the potential for vepdegestrant to become an oral best-in-class targeted therapy and to become a backbone estrogen receptor therapy in the metastatic breast cancer space; the planned timing of data readouts for our ongoing clinical trials, including our Phase 3 VERITAC-2 monotherapy trial in the second-line setting; the potential therapeutic benefits and market opportunity of our product candidates, including vepdegestrant, ARV-766, ARV-393 and ARV-102; the opportunity for ARV-766 in both post- and pre-novel hormonal agent settings to potentially treat metastatic castrate-sensitive prostate cancer and metastatic castrate-resistant prostate cancer; the potential for androgen receptor (AR)-targeting PROTAC degraders to address the unmet need of patients with prostate cancer across multiple stages of disease and surpass the benefits of other AR inhibitors; the timing to receive progression free survival data for the ARV-766 dose expansion trial; whether our BCL6 PROTAC® degrader will be a first-in-class potential therapy for non-Hodgkin Lymphoma and additional opportunities for BCL6; the timing to initiate a Phase 1 trial for ARV-393; whether a KRAS-targeting PROTAC may provide an advance in treatment for multiple cancers; whether PROTAC-induced LRRK2 degradation could be a potential treatment for idiopathic Parkinson's disease and Progressive Supranuclear Palsy; our plans and anticipated timing of clinical starts for KRAS, G12D and other programs; and our plans with respect to timing of key program catalysts.

The words "anticipate," "believe," "estimate," "expect," "intend," "may," "might," "plan," "predict," "project," "target," "potential," "will," "would," "could," "should," "continue," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make as a result of various risks and uncertainties, including but not limited to: whether we and Pfizer will be able to successfully conduct clinical development for vepdegestrant and receive results from our clinical trials on our expected timelines, or at all; whether we will be able to successfully conduct and complete development for our other product candidates, including whether we initiate and complete clinical trials for our product candidates and receive results from our clinical trials on our expected timelines, or at all; the satisfaction or waiver of the closing conditions set forth in the license agreement with Novartis, each party's performance of its obligations under the license agreement; whether Novartis will be able to successfully conduct and complete clinical development, obtain marketing approval for and commercialize ARV-766; our ability to protect our intellectual property portfolio; whether our cash and cash equivalent resources will be sufficient to fund our foreseeable and unforeseeable operating expenses and capital expenditure requirements; and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, discussed in the "Risk Factors" section of our quarterly and annual reports on file with the U.S. Securities and Exchange Commission. The forward-looking statements contained in this presentation reflect our current views as of the date of this presentation with respect to future events, and we assume no obligation to update any forward-looking statements, except as required by applicable law. These forward-looking statements should not be relied upon as representing our views as of any date subsequent to the date of this presentation.


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This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such data and estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. This presentation is intended for the investor community only. It is not intended to promote the products referenced herein or otherwise influence healthcare prescribing decisions. Cross-trial comparisons are not based on head-to-head studies and no direct comparisons can be made.

Arvinas: Advancing a new therapeutic modality to patients



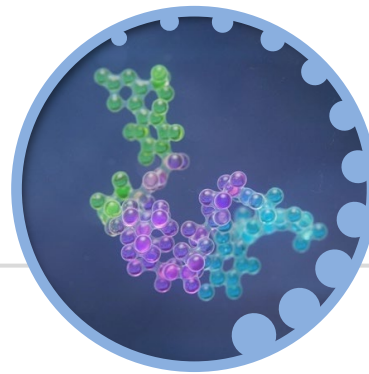
PIONEER IN THE FIELD

- **Most advanced protein degradation platform**
- **Expecting to bring the first PROTAC[®] protein degrader to market** (in partnership with )
- **First neuroscience PROTAC[®] degrader** advanced to the clinic in 2024



NEW MECHANISM

- PROTAC[®] protein degraders **eliminate** vs. inhibit disease-causing proteins
- Combines the **power of genetic knockdown** technology with the **benefits of small-molecule** therapeutics

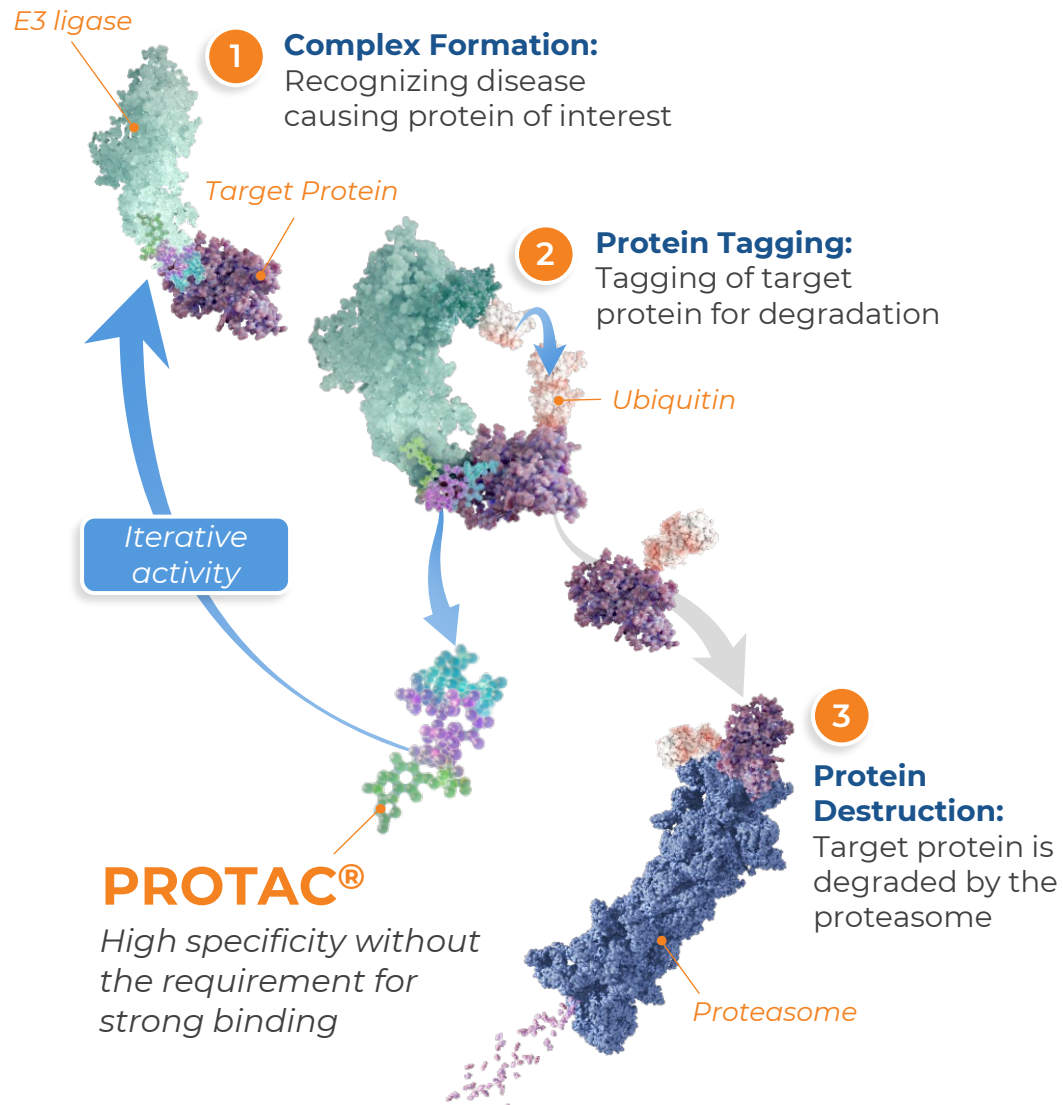


DEEP PIPELINE

- **Clear efficacy signals** in patients with difficult-to-treat breast and prostate cancers
 - 2 ongoing Phase 3 trials
 - 10+ ongoing Phase 1 & 2 trials
- **Pipeline of programs** across oncology, neuroscience, hematology, and immuno-oncology



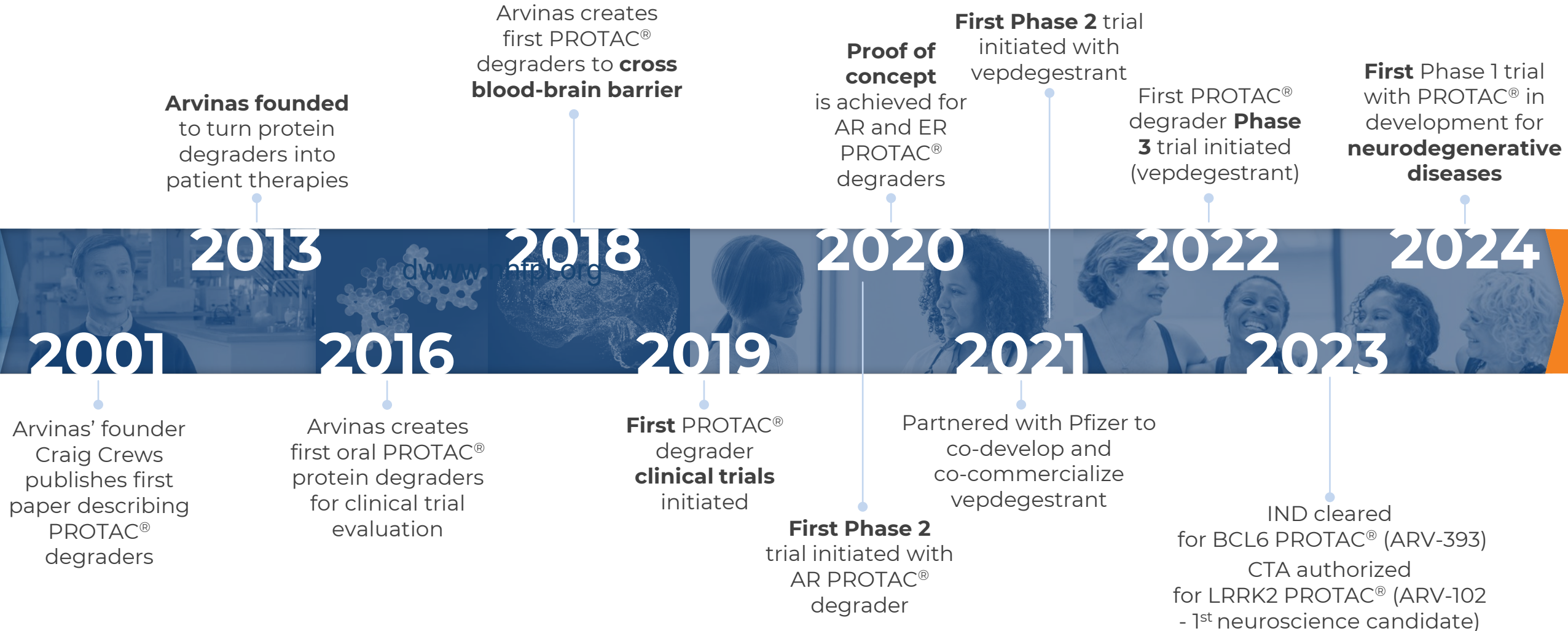
PROTAC[®] protein degraders combine the benefits of small molecules and gene-based knockdown technologies



Arvinas' proteolysis-targeting chimera (PROTAC[®]) degraders can:

- Eliminate (rather than inhibit) disease-causing proteins
- Disrupt scaffolding functions of target proteins
- Bind and degrade classically “undruggable” proteins
- Act iteratively (catalytically)
- Be delivered orally and achieve broad tissue distribution, including across the blood-brain-barrier

A History of Pioneering To transform the treatments of tomorrow



The agents mentioned above are currently under investigation; their safety and effectiveness have not yet been established
IND, investigation new drug application; CTA, clinical trial authorization

Our broad pipeline includes the first pivotal trials for PROTAC[®] degraders



Program	Therapeutic Area / Indication	Preclinical	Phase 1/1b	Phase 2	Phase 3
Vepdegestrant (ARV-471) Global co-development/co-commercialization partners with 	Oncology: ER+/HER2- Breast Cancer	★ VERITAC-2: vepdegestrant monotherapy 2L+ pivotal trial			
		★ <i>Vepdegestrant plus palbociclib and potentially other CDK4/6 inhibitors in 2L^a</i>			
		★ VERITAC-3: vepdegestrant + palbociclib as 1L combination therapy (<i>study lead-in</i>)			
		★ <i>Vepdegestrant plus CDK4 inhibitor (atirmociclib/PF-07220060) in 1L^a</i>			
		VERITAC: vepdegestrant monotherapy dose expansion (2L+)			
		TACTIVE-K: vepdegestrant in combination with CDK4i (atirmociclib/PF-07220060)			
		TACTIVE-N: vepdegestrant in neoadjuvant setting (to inform potential adjuvant plan)			
		TACTIVE-U: vepdegestrant in combination with ribociclib, abemaciclib and other targeted therapies			
ARV-766^b	Oncology: Prostate Cancer	★ <i>ARV-766 monotherapy (mCRPC)</i>			
		ARV-766 monotherapy dose expansion (2L+)			
		ARV-766 Phase 1/2 combination with abiraterone (pre-NHA setting)			
ARV-393 (BCL6)	Hematology	Phase 1 dose escalation			
ARV-102 (LRRK2)	Neuroscience	Phase 1 dose escalation			
Preclinical programs	Oncology and Neuroscience	20+ programs, including KRAS-G12D/V, AR-V7 ^b , Myc, HPK1, Tau, α-Synuclein, and mHTT			

^a Pending Health authority feedback on potential pivotal trial

^b On April 11, 2024, Arvinas entered into a transaction with Novartis that included a global license agreement for the development and commercialization of ARV-766. Closing of the transaction is subject to customary closing conditions, including the expiration or termination of the waiting period under the Hart-Scott-Rodino Antitrust Improvements Act of 1976.

NHA, novel hormonal agent

These agents are currently under investigation; their safety and effectiveness for these investigational uses have not yet been established.

★ Pivotal Trial

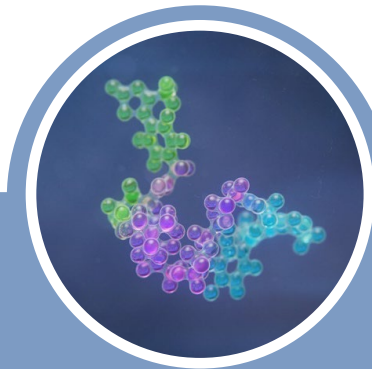
Planned

Arvinas' strategy positions us for the next stage of growth



Focused on Near-term Patient Impact

- Planning for multiple launches with vepdegestrant
- Strengthened by a best-in-class pipeline and research engine



Data-driven Approach to Resource Allocation

- Choose and invest in highest value drivers
- Use BD to enhance the value of our pipeline
- Invest for commercial success



Strong Capitalization

- Backed by a cash runway into 2027
- Capital to support us through the first years of our planned commercial launch



CLINICAL PROGRAMS

Vepdegestrant

ARVINAS



Vepdegestrant (ARV-471): First-in-class estrogen receptor (ER)-degrading PROTAC[®] in advanced breast cancer



1 in 8 U.S. women will develop breast cancer in her lifetime^a

~80% of all newly diagnosed cases of breast cancer are ER-positive (ER+)^b

Vepdegestrant is currently being evaluated in **two Phase 3 trials** in metastatic breast cancer

Vepdegestrant has the potential to become an oral, best-in-class targeted therapy

Vepdegestrant degrades **wild-type and ESRI-mutant** estrogen receptors (ER) to directly inhibit signaling pathways

More than **600 patients and healthy volunteers** have been treated with vepdegestrant across 12 clinical trials

Consistent and compelling data in **heavily pre-treated patients**

Vepdegestrant could be a backbone ER therapy in the ~\$17B ER+/HER2- metastatic breast cancer space^c

Vepdegestrant is an investigational compound. Its safety and efficacy has not been established

^a ACS: <https://www.cancer.org/cancer/breast-cancer/about/how-common-is-breast-cancer.html>; accessed 01/06/24; ^b <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4549764/>, accessed 01/06/2024

^c Clarivate/Decision Resources Group – Breast Cancer Disease Landscape & Forecast (Nov 2023). 2032 Projection.

ER, estrogen receptor; HER2, human epidermal growth factor 2; ESRI, estrogen receptor 1 gene

Our Phase 3 VERITAC-2 monotherapy trial in the 2L+ setting is on track for topline data in 2H24



Two ongoing monotherapy trials:

- VERITAC-2: Phase 3 trial
- VERITAC: Phase 2 trial (enrollment complete, N=71)
 - At RP3D (200mg), vepdegestrant showed favorable safety profile, with <6% Grade 3+ TRAEs, no dose reductions, and low rate of discontinuations

VERITAC Phase 2 subset analysis:

- In the 8 patients in VERITAC who would meet the eligibility criteria for the Phase 3 VERITAC-2 trial (*no prior fulvestrant, no prior chemotherapy for locally advanced/metastatic disease*)^a:
 - **CBR: 62.5% (5 of 8 patients)**
 - **mPFS: 19 months (4 of 8 events)**
 - **ORR: 29% (7 evaluable patients, 2 confirmed responses)**

Study design for Phase 3 VERITAC-2 (Enrolling, NCT05654623)

Treatment (N = 560)

Randomize
1:1

Vepdegestrant
200 mg orally once daily

Fulvestrant
500 mg intramuscularly
Days 1 and 15 of cycle 1 and Day 1 of subsequent cycles

Select Patient Eligibility Criteria

- Prior CDK4/6 inhibitor treatment
- No prior fulvestrant
- No prior chemotherapy for locally advanced / metastatic disease

Primary Endpoints

Progression Free Survival (PFS) by Blinded Independent Central Review in:

- ESR1 mutant population
- All Comers (Intention To Treat) population

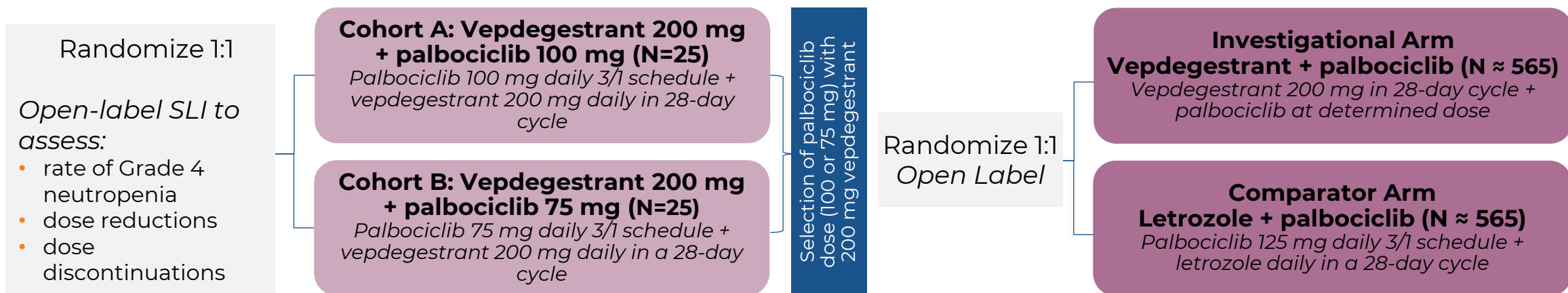
^a Data cutoff, June 6, 2023; Post-hoc analysis
RP3D, recommended phase 3 dose; TRAE, treatment related adverse events; CBR, clinical benefit rate; mPFS, media progression-free survival; ORR, objective response rate; ESR1, estrogen receptor 1; CDK, cyclin-dependent kinase

The study lead-in for our 1L Phase 3 VERITAC-3 trial in combination with palbociclib is fully enrolled

VERITAC-3 Phase 3 trial design (NCT05909397)

Study lead-in (SLI) N=50

Randomized w/comparator arm (N~1130)



Key Exclusion Criteria

- Prior adjuvant CDK 4/6i
- Primary/secondary endocrine resistance
- Visceral crisis

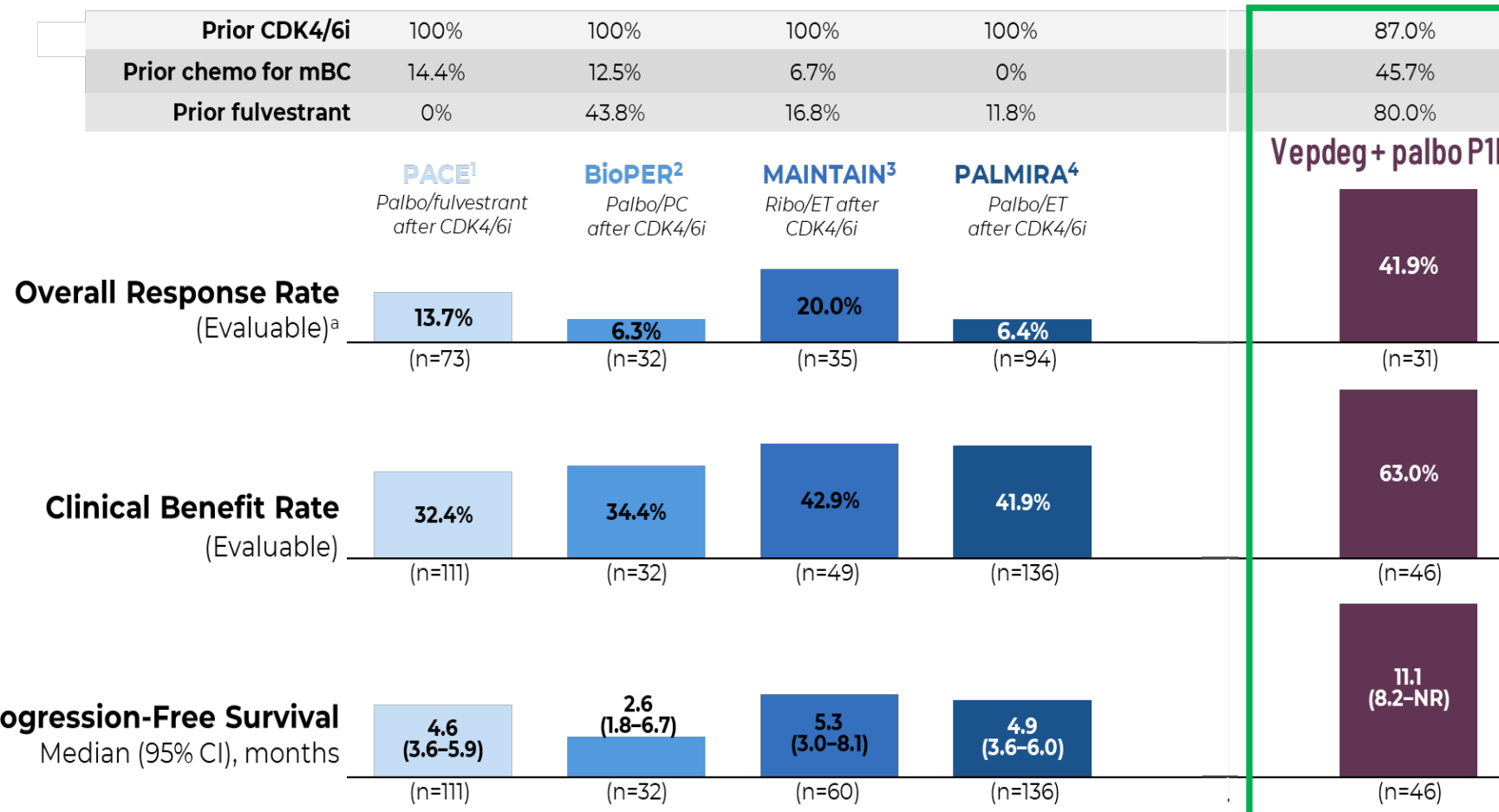
Primary Endpoint

- Progression Free Survival (PFS) by Blinded Independent Central Review (BICR)

Results from Phase 1b trial with vepdegestrant + palbociclib presented at SABCS 2023

Efficacy benchmarks in CDK4/6i after CDK4/6i trials

Data below 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 and many other factors



Safety/tolerability in Phase 1b trial

- Safety was manageable, with standard on-label dose reductions of palbociclib resulting in a 72% decline in Grade 4 neutropenia in subsequent cycles
- No febrile neutropenia**, low rates of discontinuation
 - Among 36 pts who had at least one palbociclib dose reduction, only 5 (13.9%) had Grade 4 neutropenia after last palbociclib dose reduction

^a Patients with measurable disease at baseline (two patients in the vepdeg + palbo P1b trial had an unknown ESRI status and both were non-responders)

CDK, cyclin-dependent kinase; mBC, metastatic breast cancer; ET, endocrine therapy; NR, not reached; PC, physician's choice endocrine therapy;

¹ Mayer E et al SABCS 2022. ² Albanell J et al. Clin Cancer Res 2023. ³ Kalinsky K et al. J Clin Oncol 2023. ⁴ Llombart-Cussac A et al. ASCO 2023.

Data presented
at SABCS2023

Clinical program designed to position vepdegestrant as a backbone ER-targeting therapy in breast cancer

Adjuvant (Post-Surgical)
Breast Cancer in US (~190K¹)

Metastatic Breast Cancer in US (~60K¹)

First Line

TACTIVE-N neoadjuvant trial
*to inform potential
adjuvant trial*

VERITAC-3 vepdeg + palbo
combination **pivotal trial**

- Assessing SLI data to determine the RP3D of palbociclib to be administered in combination with vepdegestrant

Second/Third Line

VERITAC-2 monotherapy
pivotal trial

TACTIVE-E: in combination
with everolimus

TACTIVE-K: in combination
with CDK4i (PF-07220060)

TACTIVE-U: in combination
with ribo/abema/CDK7i

Active Trials

Planned trials^a

Pending further data and regulatory agreement:

Planned: Pivotal
Vepdeg+Pfizer's novel CDK4
inhibitor atirmociclib (PF-
07220060)

New

Planned: Pivotal Vepdeg
combo with palbo and
potentially other CDK4/6i

New

^aPending health authority feedback on potential pivotal trials
CDK, cyclin-dependent kinase; SLI, study lead-in; RP3D, recommended phase 3 dose
¹Kantar Cancer MPact Patient Metrics (accessed Nov. 2023)



CLINICAL PROGRAMS

ARV-766


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Arvinas' PROTAC[®] degraders eliminate the androgen receptor (AR), potentially surpassing the benefits of AR inhibitors

A graphic showing a group of stylized human figures in blue and orange, with the text "1 in 8" overlaid in large orange font.

1 in 8 U.S. men will be diagnosed with prostate cancer during their lifetime^a

Prostate cancer is the **2nd leading cause of cancer death** for men in the U.S.^a

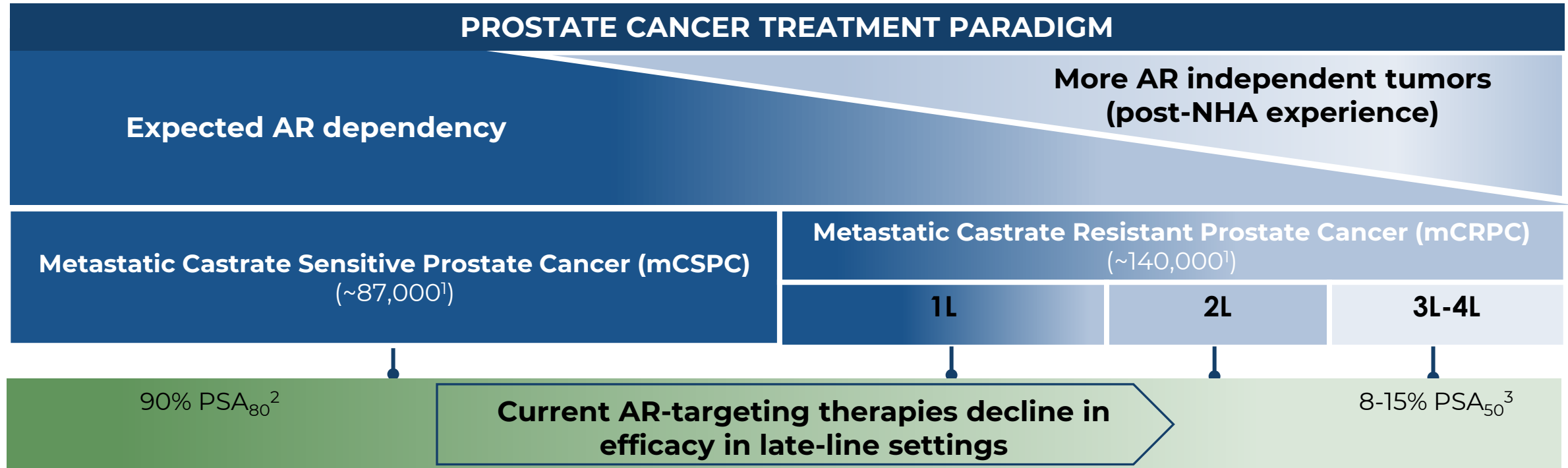
An AR-targeting PROTAC degrader may address the unmet needs of patients with prostate cancer across multiple stages of disease

AR is a critical target in prostate cancer, but tumors develop resistance to standard-of-care AR inhibitors

ARV-766, our second-generation PAN AR-targeting PROTAC degrader, has demonstrated an excellent tolerability profile and improved efficacy profile compared to our first-generation AR PROTAC degrader (bavdegalutamide)

ARV-766's activity in late-line settings suggests additional potential benefit in earlier-line, less-pretreated patients

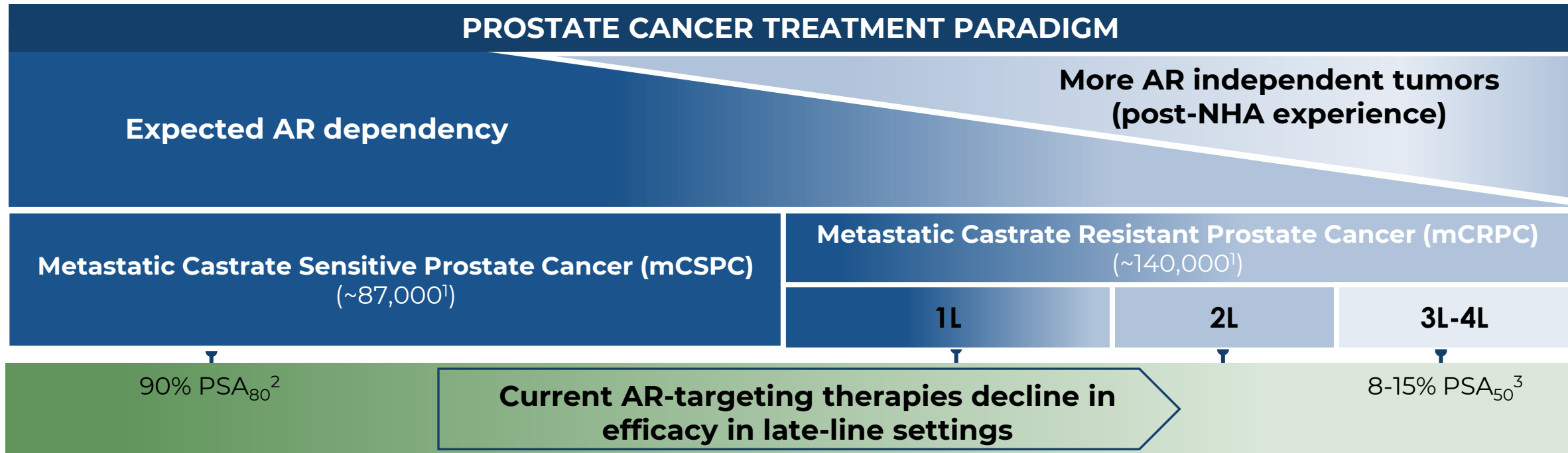
AR-targeting PROTAC[®] degrader could meet the substantial unmet need across the prostate cancer treatment paradigm



Unmet need remains for **well-tolerated therapies** that **overcome resistance mechanisms**, **extend survival** and provide **durable responses**

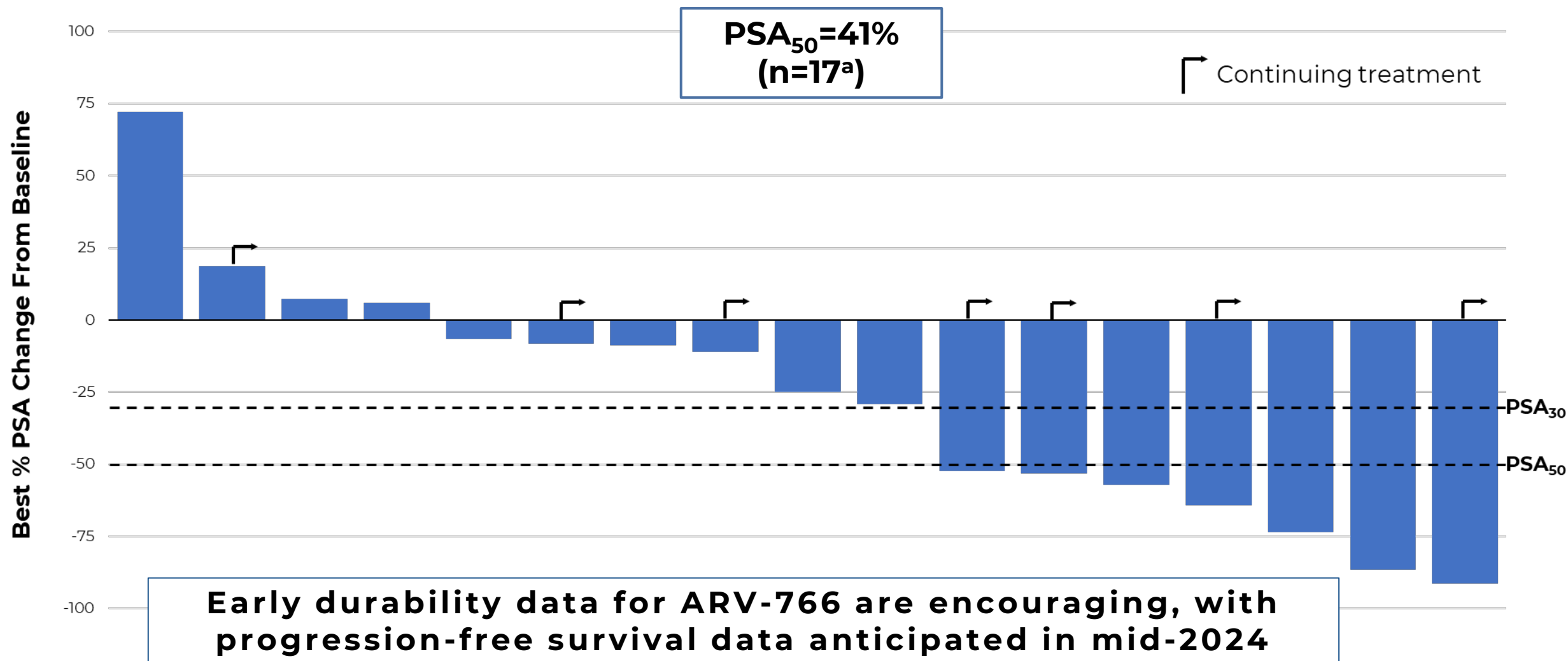
AR-targeting PROTAC[®] degrader could meet the substantial unmet need across the prostate cancer treatment paradigm

PROSTATE CANCER TREATMENT PARADIGM



- Our *first generation* PROTAC[®] AR degrader (bavdegalutamide) demonstrated **strong antitumor activity in 3L+ patients with T878/H875 AR LBD mutations (11.1 month rPFS)**
- Arvinas has selected our ***second generation* PROTAC[®] AR degrader ARV-766** to advance into Phase 3 trials due to a **broader efficacy profile** and **better tolerability** vs. bavdegalutamide, making it well suited for both mCRPC and mCSPC patients.

ARV-766 shows promising efficacy signals in heavily pretreated mCRPC patients with AR LBD mutations who have progressed on a prior NHA



^a Includes biomarker-evaluable patients with ≥4 weeks of PSA follow-up. Data from the ARV-766 Phase 1/2 dose escalation and expansion trial; data cut-off, August 23, 2023
mCRPC, metastatic castrate-resistant prostate cancer; AR, androgen receptor; LBD, ligand-binding domain; PSA, prostate-specific antigen; PSA₃₀, best PSA declines ≥30%; PSA₅₀, best PSA declines ≥50%;
NHA, novel hormonal agent

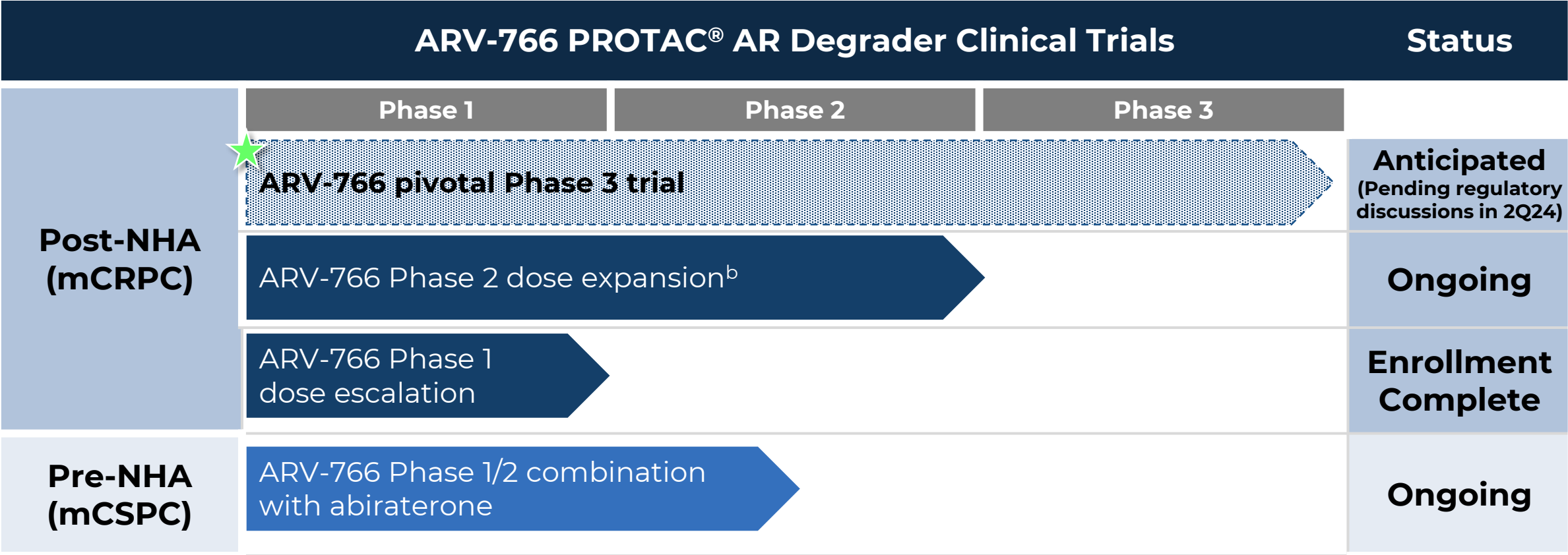
ARV-766's excellent tolerability profile is well-suited for both late-and early-line settings

ARV-766 has been well tolerated to date

- Majority of treatment related adverse events (TRAEs) are Grade 1 or 2, with no Grade ≥ 4 TRAEs
- Low rates of discontinuation or dose reduction

TRAE $\geq 10\%$ n (%)	ARV-766 (N=84 ^a)	
	Any Grade	Grade 3+
Any TRAE	55 (66)	7 (8)
Fatigue	24 (29)	2 (2)
Nausea	12 (14)	0 (0)
Diarrhea	9 (11)	1 (1)
Vomiting	9 (11)	0 (0)
Decreased appetite	9 (11)	0 (0)
Alopecia	8 (10)	0 (0)
Any TRAE leading to discontinuation	3 (4)	

Ongoing clinical development program for ARV-766 in the post-and pre-NHA settings^a



 Pivotal Trial

^a On April 11, 2024, Arvinas entered into a transaction with Novartis that included a global license agreement for the development and commercialization of ARV-766. Closing of the transaction is subject to customary closing conditions, including the expiration or termination of the waiting period under the Hart-Scott-Rodino Antitrust Improvements Act of 1976.

^b Progression free survival data anticipated in 2024

NHA, novel hormonal agent; AR, androgen receptor; mCSPC, metastatic castrate-sensitive prostate cancer; mCRPC, metastatic castrate-resistant prostate cancer



Preclinical Programs



Advancing an industry leading preclinical pipeline of PROTAC[®] degraders

We have the **deepest and most diverse pipeline** of any protein degradation company

The capabilities of our PROTAC[®] platform remain **unmatched**

Arvinas' pipeline is **differentiated and sustainable**

On-track for initiation of two new first-in-human trials in 1H 2024

- **LRRK2**-targeting PROTAC **ARV-102** reaches and degrades in deep brain regions (Phase 1 trial enrolling)
- **BCL6**-targeting PROTAC **ARV-393** addresses a historically undruggable target (IND cleared)

We expect ARV-393, our BCL6 PROTAC[®] degrader, to be a first-in-class potential therapy for non-Hodgkin Lymphoma

BCL6 is genetically mutated in up to 85% of DLBCL¹, a subset of Non-Hodgkin Lymphoma

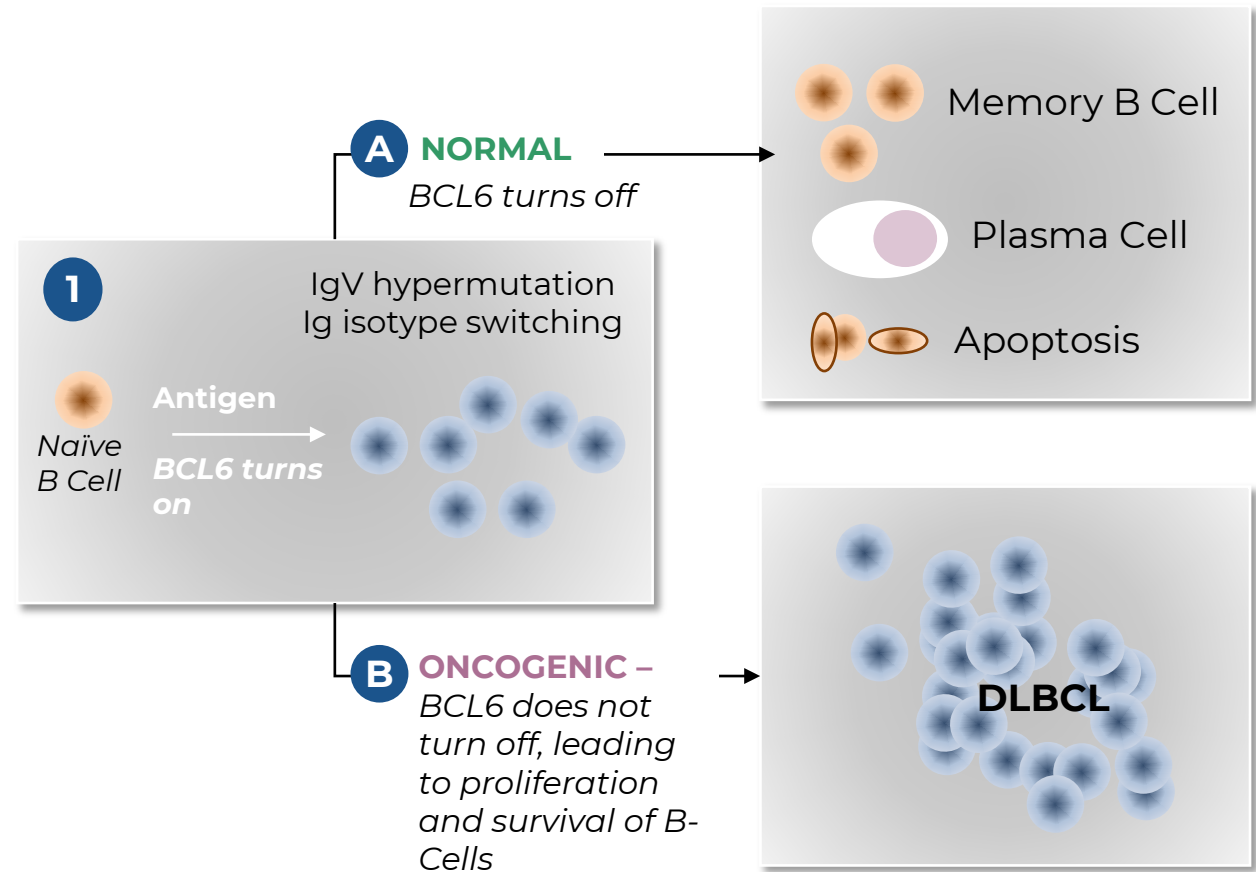
More than 74,000 people are diagnosed with DLBCL each year²

DLBCL is largely devoid of oral options; no BCL6-targeted therapy on the market

Additional opportunities for a BCL6 degrader exist in Burkitt's Lymphoma, Follicular Lymphoma, Angioimmunoblastic T-cell lymphoma, and solid tumors

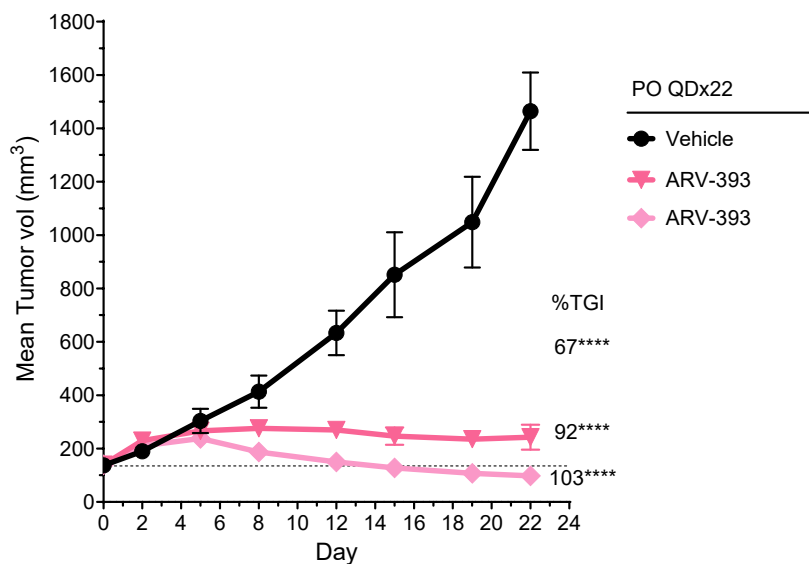
IND cleared for ARV-393
Phase 1 trial initiation anticipated in 1H24

The role of BCL6 in driving DLBCL³

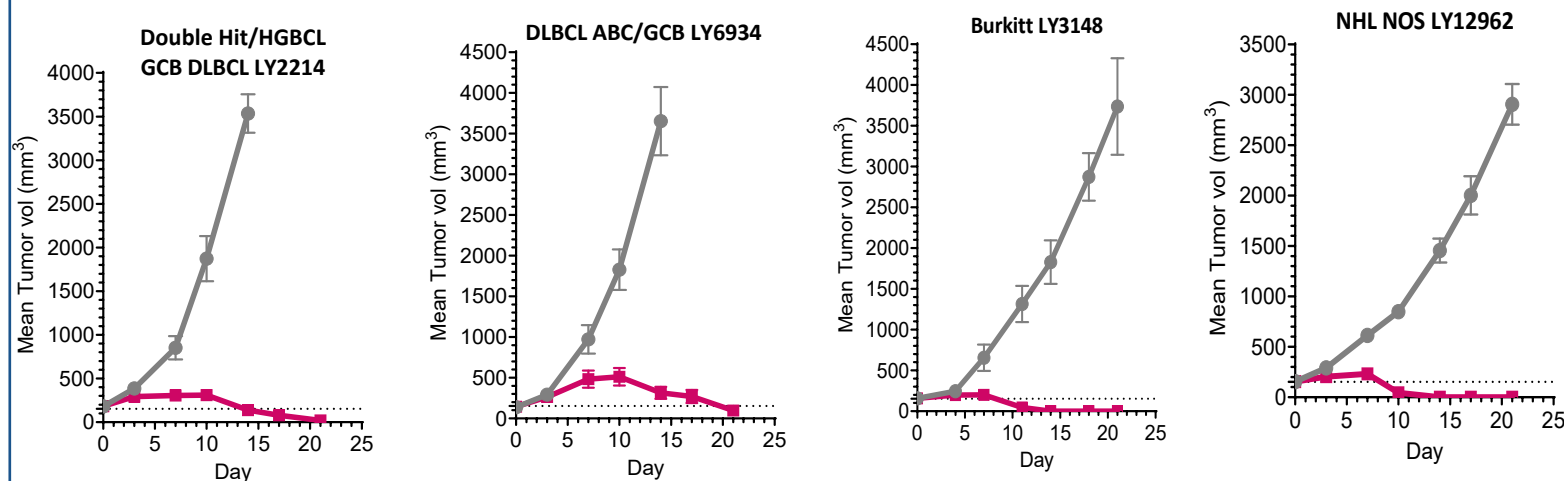


ARV-393 shows robust tumor inhibition in a DLBCL model and in models of multiple subtypes of Non-Hodgkin's Lymphoma

Cell line-derived xenograft (CDX) model
OCI-LY1 GCB DLBCL



Breadth of efficacy beyond DLBCL demonstrated in multiple patient-derived xenograft (PDX) models with no body weight loss^a



4 mice/group, PO QDx21

Vehicle

ARV-393

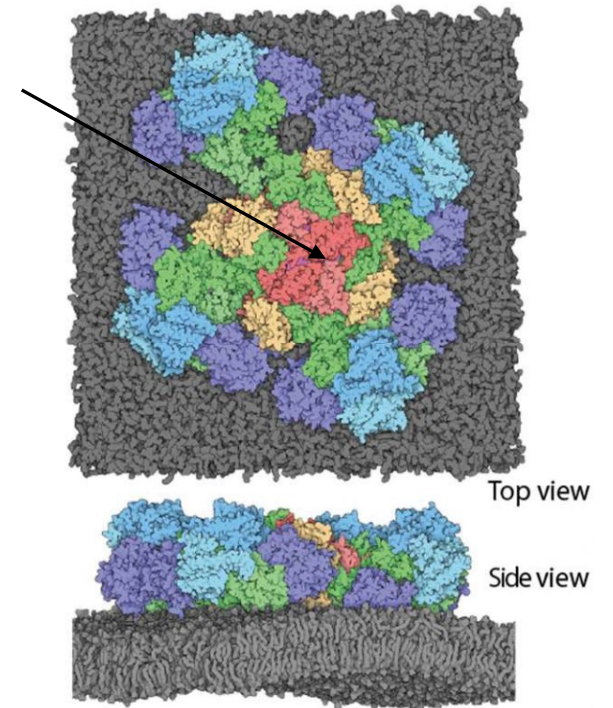
^a Body weights not shown

NHL, non-Hodgkin's lymphoma; NOS, not otherwise specified; DLBCL, diffuse large B-cell lymphoma; HGBCL, high grade B-cell lymphoma; GCB, germinal center B-cell; ABC, activated B-cell; TGI, tumor growth inhibition

KRAS-targeting PROTAC[®] may provide a significant advance in treatment for multiple cancers

- KRAS has few druggable “pockets,” challenging traditional inhibitors
- KRAS also exists in a multi-protein (scaffolding) complex, limiting access to drugs
- KRAS mutations are highly prevalent in pancreatic (~90%), colorectal (~35%), and non-small cell lung cancers (~25%)¹⁻⁴

KRAS (in red) is surrounded by other proteins (other colors), and binding is often occluded

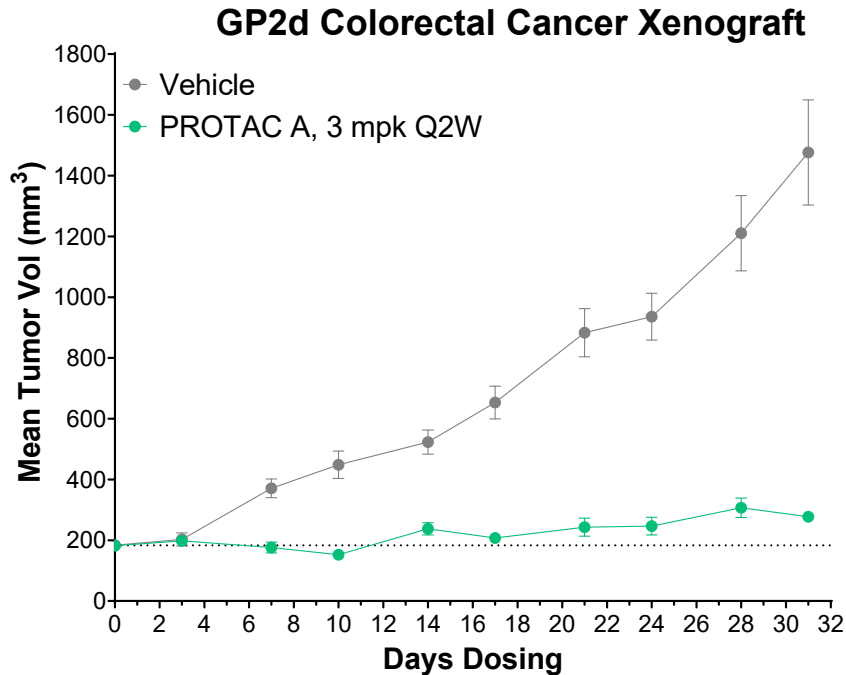


Mysore et al., BioRxiv, 2020

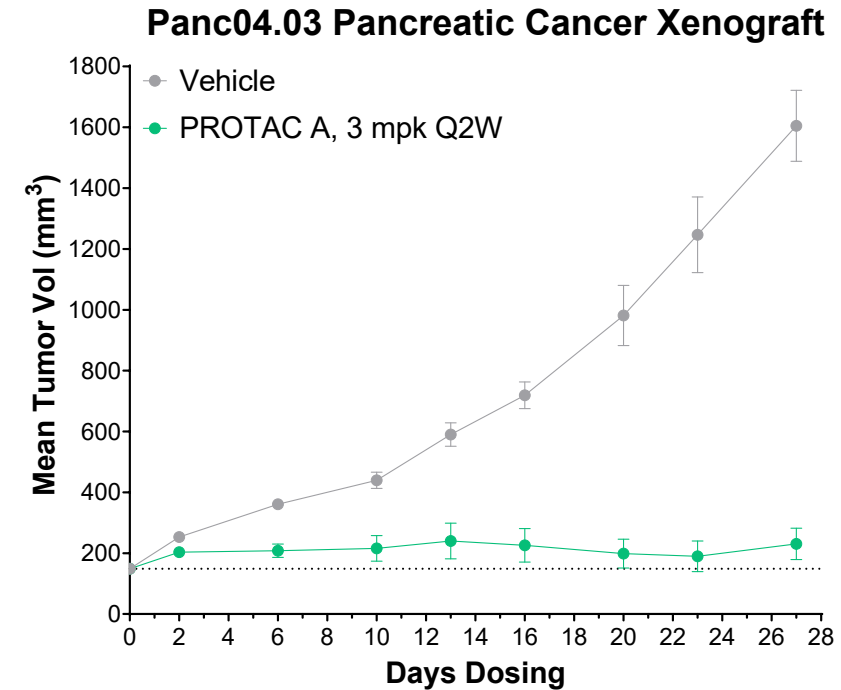
KRAS G12D-targeted PROTAC degrader currently in IND-enabling studies

KRAS G12D-targeting PROTAC[®] demonstrates robust tumor growth inhibition with every other week dosing

Colorectal cancer xenograft model



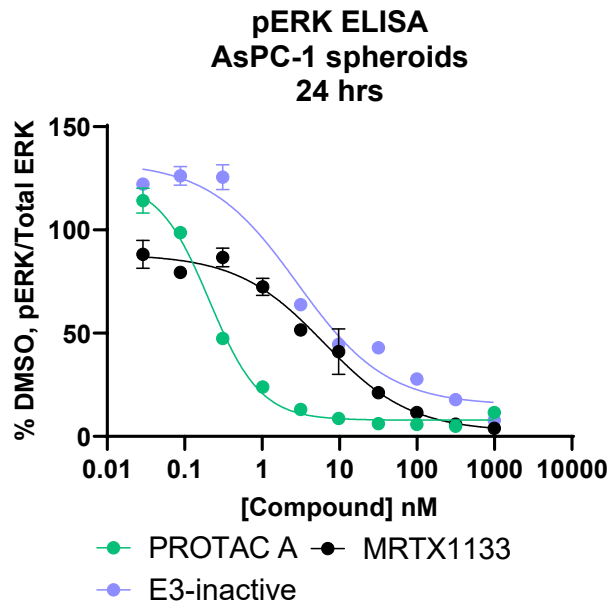
Pancreatic cancer xenograft model



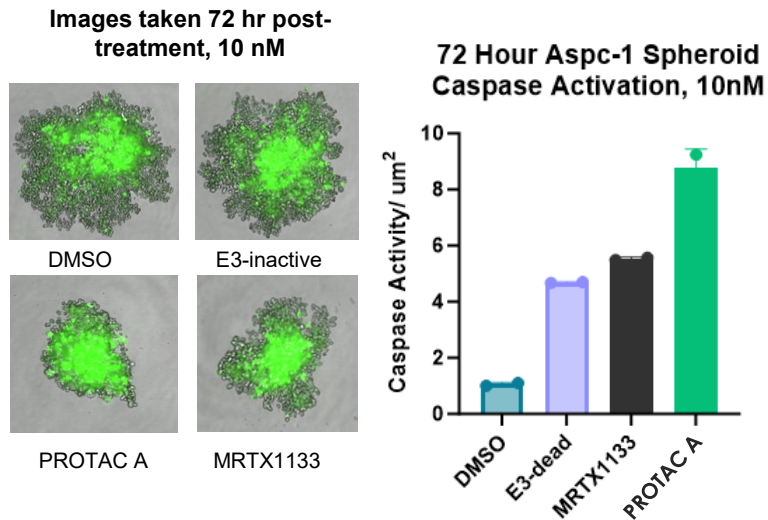
KRAS G12D-targeting PROTAC[®] potently suppresses signaling and proliferation versus an inhibitor

KRAS G12D-targeting PROTAC demonstrated potent outcomes in multiple measures of cancer cell inhibition while inducing cell apoptosis

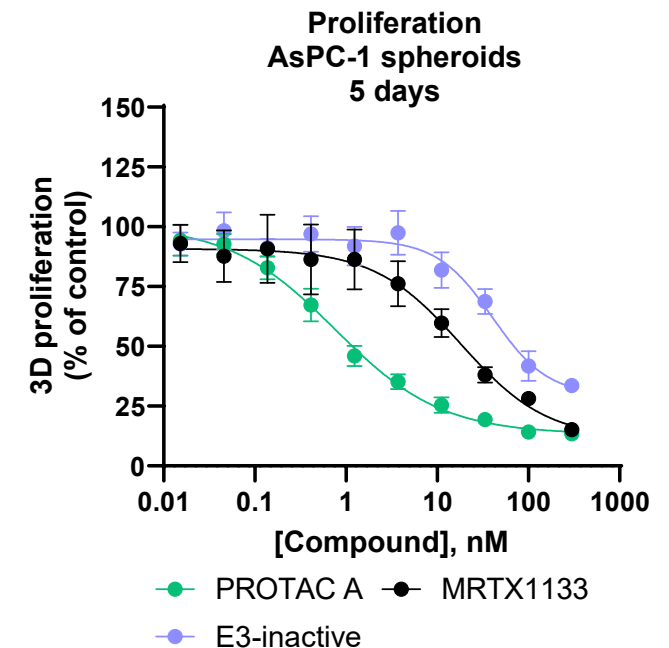
MAPK Signaling PROTAC **30-fold** more potent



Spheroid size reduction and apoptosis induction



Proliferation PROTAC **30-fold** more potent



PROTAC[®] degraders could revolutionize the treatment of patients with neurological diseases



Arvinas can engineer oral PROTAC[®] degraders that:

- ✓ **Cross the blood-brain barrier**
- ✓ **Reach targets in “deep brain” regions**
- ✓ **Degrade disease-causing proteins inside cells**
- ✓ **Differentiate between mutant and wild-type proteins, e.g., mutant huntingtin**

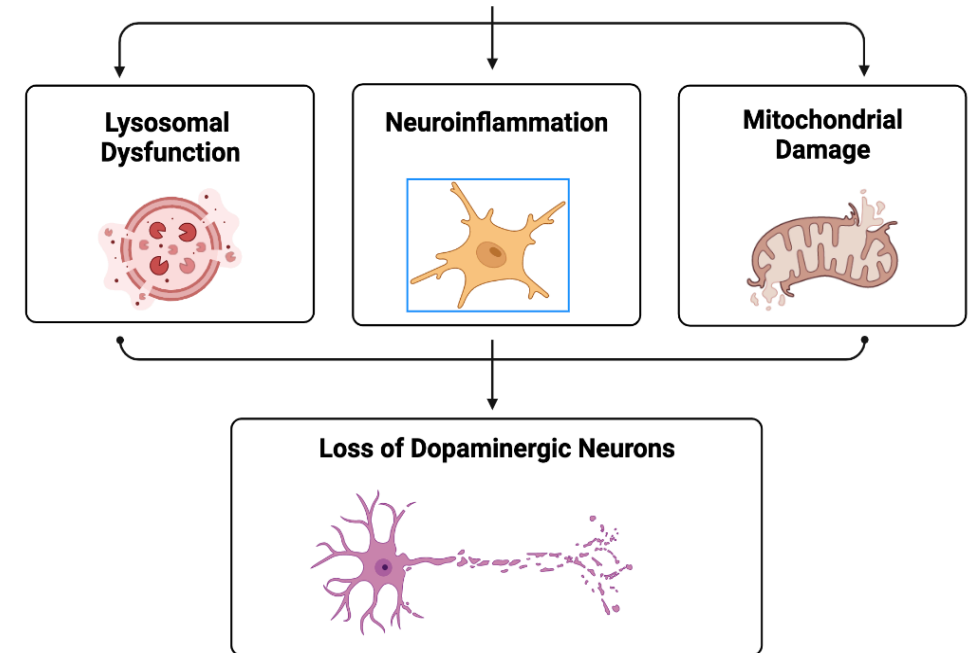
Currently enrolling Phase 1 trial with ARV-102, the first oral PROTAC[®] in development to treat neurodegenerative diseases

PROTAC[®]-induced LRRK2 degradation as a potential treatment for idiopathic Parkinson's Disease and Progressive Supranuclear Palsy

Human genetics and biology create a strong rationale for differential biology of PROTAC[®] LRRK2 degraders

- **LRRK2 is a large multidomain scaffolding kinase**
- **Parkinson's Disease (PD)** has a diagnosed prevalence of ~1M in the US, with more than 10M worldwide¹
 - No approved disease-modifying therapies for PD
 - Familial mutations and sporadic variants implicate LRRK2 in PD
- **Progressive Supranuclear Palsy (PSP)** is a pure tauopathy with rapid progression to death within 5-7 years
 - No approved therapies for PSP
 - LRRK2 genetic variants associated with accelerated progression time to death
- LRRK2 kinase inhibitors and an ASO in clinical trials

Mutations in and increased expression of LRRK2



Arvinas' oral PROTAC[®] LRRK2 degrader reaches multiple "deep brain" regions in non-human primates and degrades LRRK2

Arvinas PROTAC degraders can be engineered to reach multiple regions of the brain

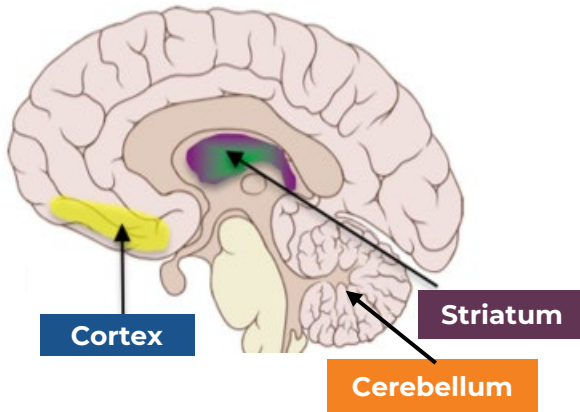
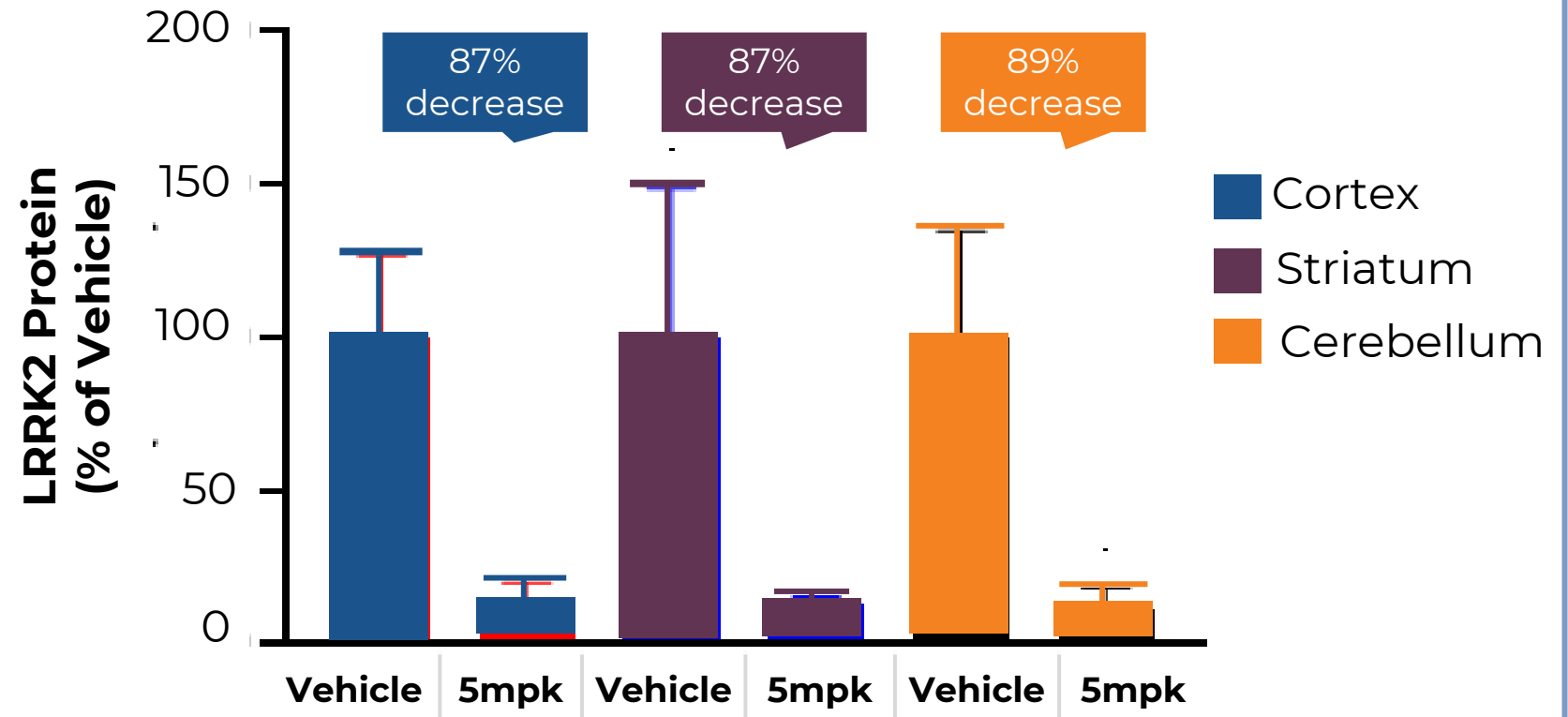


Figure modified from Beuriat et al. 2022

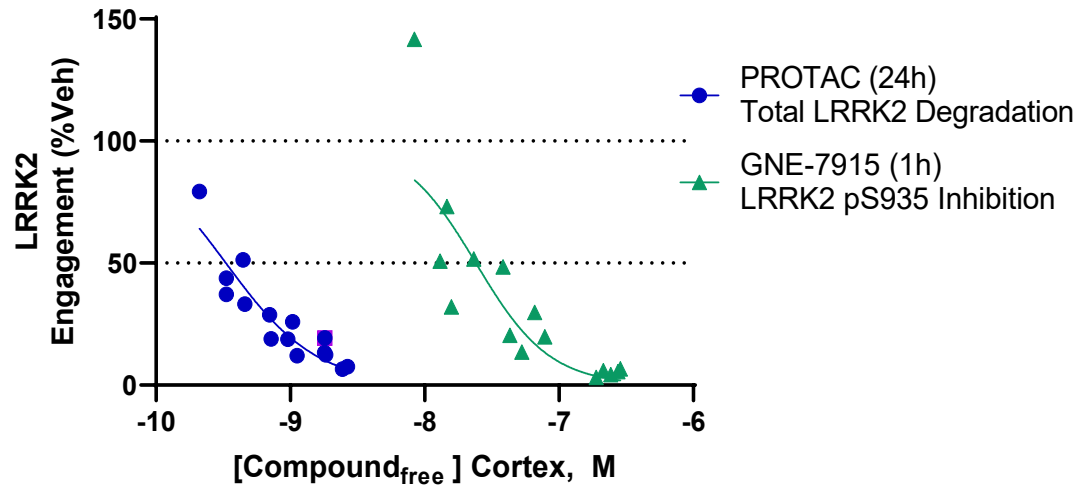
>85% LRRK2 degradation in deep brain regions of cynomolgus monkeys after oral dosing



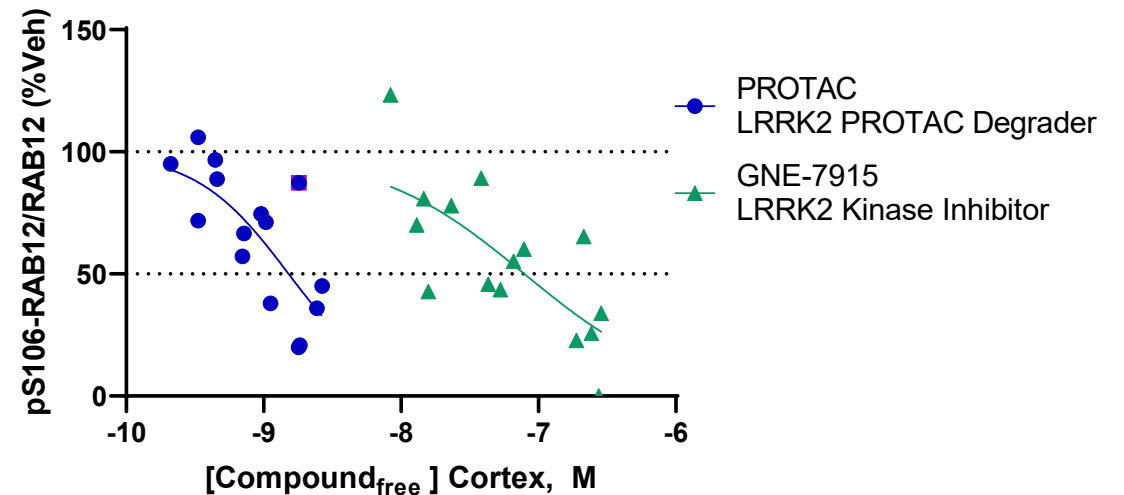
PROTAC[®] LRRK2 degrader shows better target engagement, enhanced potency and pathway engagement versus a LRRK2 inhibitor

Iterative (catalytic) PROTAC[®] advantage results stronger LRRK2 and lysosomal pathway engagement vs. a LRRK2 inhibitor^a

LRRK2 PROTAC vs. Kinase inhibitor (Tmax)



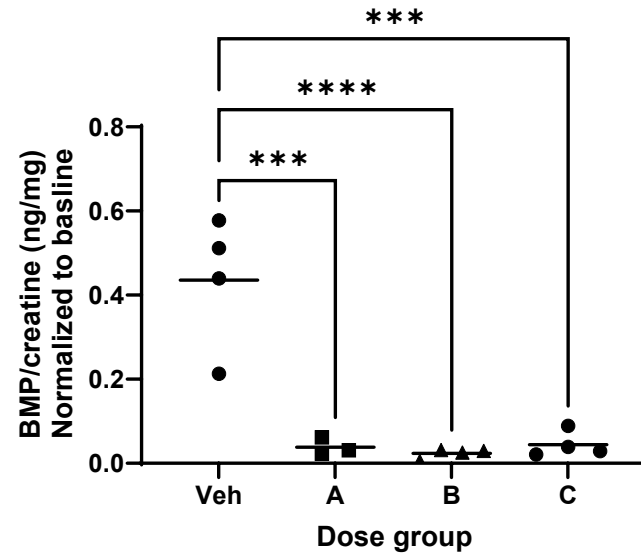
Lysosomal Pathway Engagement (LRRK2 PROTAC vs. Inhibitor)



Our LRRK2 degrader induces biomarker changes that reinforce confidence in the PROTAC[®] MoA in the brain and periphery

PROTAC-induced reductions observed in key lysosomal marker in cynomolgus monkey

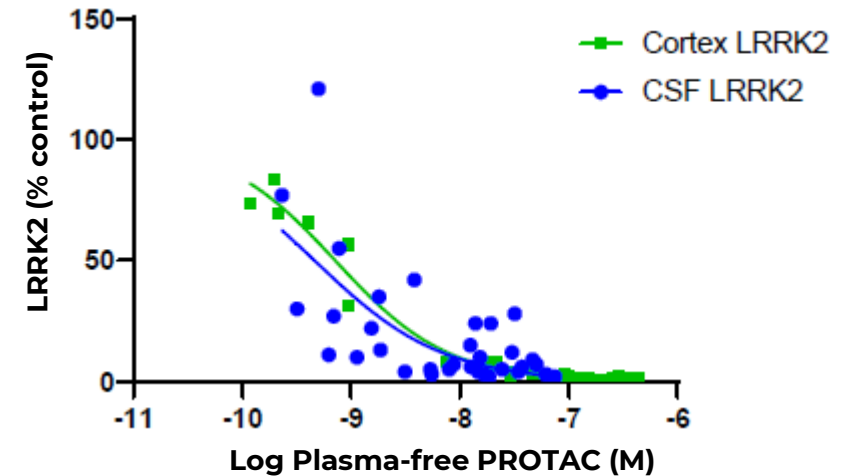
BMP reductions in cynomolgus monkeys



BMP levels were measured by UPLC-MS/MS and normalized to creatinine and then expressed relative to baseline.

PK/PD of LRRK2 reduction in cortex and CSF following oral dosing in cynos

CSF LRRK2 reductions in cynomolgus monkeys; surrogate compartment for brain



Equivalent PK/PD supports the utility of measuring CSF LRRK2 as a surrogate for monitoring LRRK2 reductions in the brain.

Strategically positioned to benefit patients in the years ahead across multiple areas of unmet need

Vepdegestrant provides a path to multiple potential launches

- ✓ VERITAC-2 Ph3 2L+ monotherapy trial on track to complete enrollment and announce topline data in 2H24
- ✓ Ph3 1L combo with palbociclib dose selection expected in 2H24
- ✓ Ph3 2L combination trial (pending HA feedback)
- ✓ Ph3 1L combination with CDK4i (pending HA feedback)

Strengthened by a best-in-class R&D engine

- ✓ Initiation of ARV-766 mCRPC Ph 3 trial (following HA feedback)
- ✓ Enrolling Ph1 trial with ARV-102, first PROTAC in development for neurodegenerative diseases
- ✓ Initiation of BCL6 Ph1 dose escalation study expected in 1H2024
- ✓ KRAS G12D-targeted PROTAC degrader currently in IND-enabling studies
- ✓ Goal to nominate 1 clinical candidate per year

Strong capital position

- ✓ Cash runway into 2027
- ✓ ~\$1.2B cash on hand^a
- ✓ Key program catalysts expected in 2024 and 2025, including topline pivotal data for VERITAC-2

^aCash, cash equivalents, and marketable securities as of March 31, 2024
HA, health authority; CDK, cyclin-dependent kinase; mCRPC, metastatic castrate-resistant prostate cancer; IND, investigational new drug application

Thank You

For More Information



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