ARVINAS

Pioneering the future of targeted protein degradation therapeutics

November 2023

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 the anticipated timing of our planned clinical trials within our pipeline, including our vepdegestrant (ARV-471) monotherapy study in the adjuvant setting, our ARV-766 Phase 3 clinical trial and our ARV-766 Phase 1/2 clinical trial in combination with arbiterone; the potential therapeutic benefits of vepdegestrant, ARV-766 and bavdegalutamide and the results of any related clinical trials; the expected timing for submission of investigational new drug applications ("IND") or clinical trial authorization applications for our preclinical candidates, BCL6 and LRRK2, as well as timing of initiation of two additional IND-enabling studies for our preclinical candidates; whether our preclinical programs, including BCL6 and LRRK2, will help treat patients with solid and haematological malignancies and neurodegenerative disorders; whether ARV-471's tolerability and signals of efficacy could allow its potential use as a "backbone" of care across stages of breast cancer; the extent to which our androgen receptor-targeting PROTAC® degraders may address the unmet needs of patients across the prostate cancer treatment paradigm; Whether ARV-766 may be an earlier-line treatment for patients with mCRPC; the timing to receive progression free survival data for the ARV-766 dose expansion trial; 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; whether our BCL6 PROTAC® degrader will be a first-in-class potential therapy for Diffuse Large B-Cell Lymphoma; whether PROTAC®-induced LRRK2 degradation could be a disease-modifying modality for Parkinson's Disease; and the timing of clinical trial initiations, and continued and completion of enrollment, including for pivotal trials, first in human studies of PROTAC® protein degraders and certain data readouts and presentations.

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 (ARV-471) 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; 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.

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.

A history of **pioneering** since our **founding**

JULY 2001
Arvinas' founder Craig Crews publishes first paper describing PROTAC® degraders

2013
Arvinas founded to turn protein degraders into patient therapies

2016
Arvinas creates first oral PROTAC® protein degraders for clinical trial evaluation

2018
Arvinas creates first BBB-crossing PROTAC® degraders

2019
PROTAC® degraders bavdegalutamide and vepdegestrant enter clinical studies

2020
Proof of concept is achieved for bavdegalutamide and vepdegestrant

2020
Bavdegalutamide **Phase 2** trial initiated

2021
Vepdegestrant **Phase 2** trial initiated

JULY 2021
Partnered with Pfizer to co-develop and co-commercialize vepdegestrant

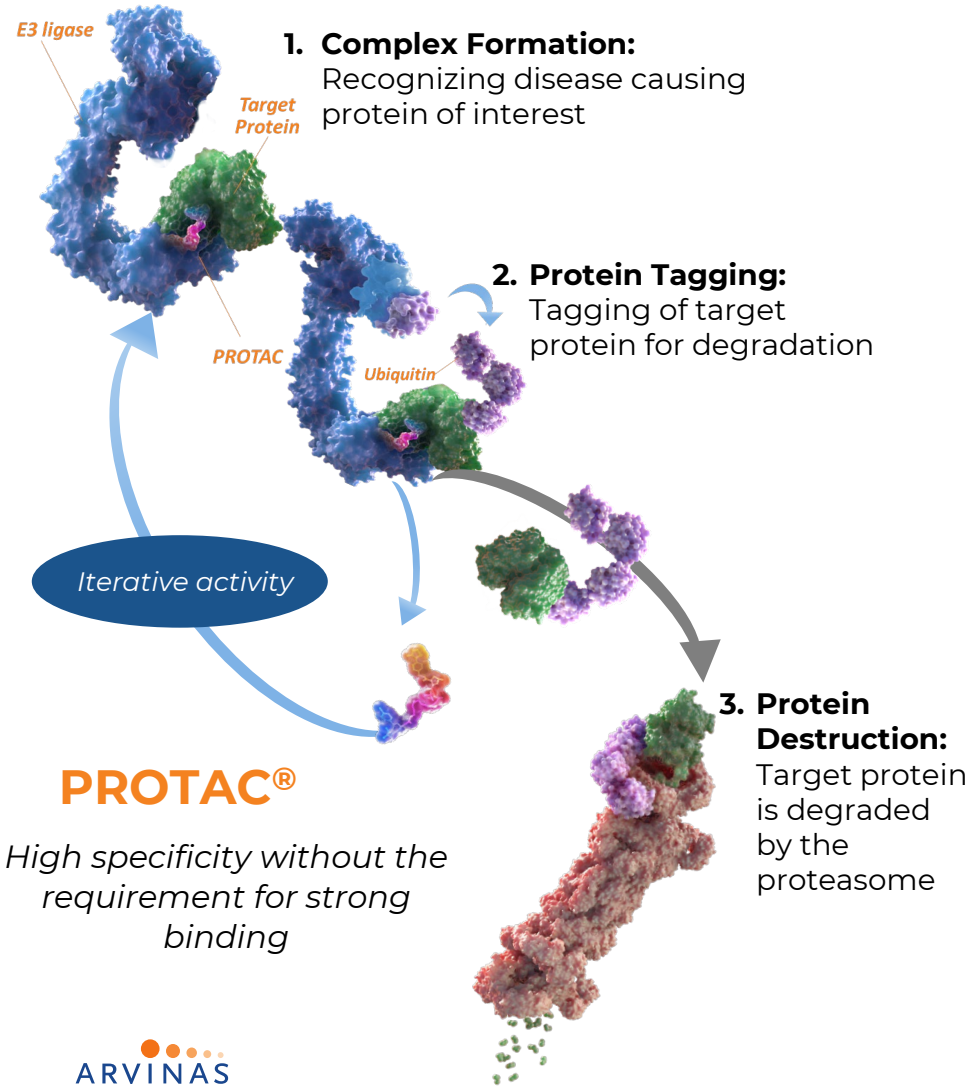
2022
Vepdegestrant **Phase 3** monotherapy (2L+) trial initiated

2022
Clinical candidate nomination of BCL6 and LRRK2

BBB, blood-brain barrier

The agents mentioned above are currently under investigation. Their safety and effectiveness for have not yet been established

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



PROTAC®

High specificity without the requirement for strong binding

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

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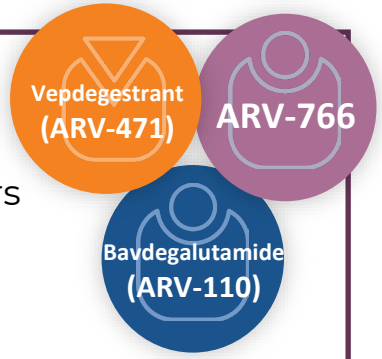
Arvinas: Advancing a new therapeutic modality to patients

PROTEIN DEGRADATION

- PROTAC® protein degraders **eliminate** vs. inhibit disease-causing proteins
- Combines the **power of genetic knockdown** technology with the **benefits of small-molecule** therapeutics
- Consistent ability to create PROTAC® degraders with drug-like properties and signals of clinical efficacy and tolerability

PIPELINE

Clear efficacy signals in patients with difficult-to-treat breast and prostate cancers



- **1 Program in Phase 3**
- **2 Programs in Phase 2**
- **20+ Pipeline Programs** in oncology and neuroscience

ARVINAS

400+ team members

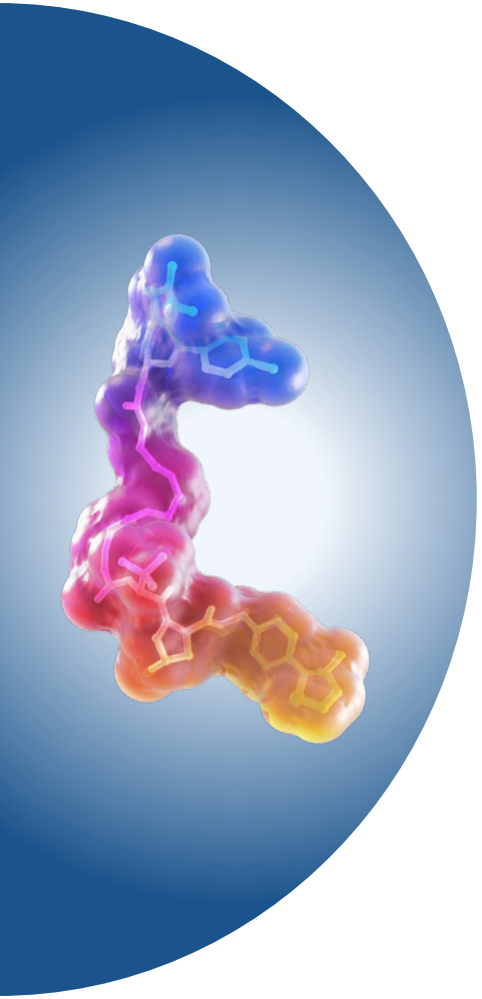
- Founded in 2013 by the original PROTAC pioneer
- Protein degradation platform with clinical proof of concept

PARTNERED FOR SUCCESS

in drug discovery, development, and commercialization

Pfizer · Genentech, A Member of the Roche Group · Insilico Medicine · Gerthbio · Bayer · GNS · PHOREMOST

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



PROTAC protein degraders have distinct advantages over both small molecule inhibitors and gene-based medicines	PROTAC Protein Degraders	Small Molecule Inhibitors	Gene-Based Medicines
Eliminate disease-causing proteins	✓		✓
Disrupt scaffolding function	✓		✓
Potential to treat "undruggable" proteins	✓		✓
Iterative mechanism of action	✓		
Broad tissue penetration	✓	✓	
Oral dosing	✓	✓	
Ease of manufacturing	✓	✓	

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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 Pfizer	**Oncology:** ER+/HER2- Breast Cancer	★ **VERITAC-2:** vepdegestrant monotherapy 2L pivotal trial			
		★ **VERITAC-3:** vepdegestrant + palbociclib as 1L combination therapy *(study lead-in)*			
		★ ***Vepdegestrant monotherapy in the adjuvant setting***			
		VERITAC: vepdegestrant monotherapy dose expansion (2L+)			
		TACTIVE-N: vepdegestrant in neoadjuvant setting			
		TACTIVE-E: vepdegestrant + everolimus			
		TACTIVE-U: vepdegestrant in combination with ribociclib, abemaciclib, and other targeted therapies			
ARV-766	**Oncology:** Prostate Cancer	★ ***ARV-766 monotherapy (mCRPC 2L+)***			
		ARV-766 monotherapy dose expansion (2L+)			
		ARV-766 monotherapy dose escalation (2L+)			
		ARV-766 Phase 1/2 combination with abiraterone (pre-NHA setting)			
Bavdegalutamide (ARV-110)		**ARDENT: Bavdegalutamide monotherapy dose expansion (2L+)**			
		Bavdegalutamide + abiraterone (2L+)			
AR-V7†, BCL6, KRAS-G12D/V†, Myc†, HPK1 ***Undisclosed Targets***	**Oncology:** Solid and Haematological Malignancies	**BCL6 IND/CTA expected in 2023**	**2 additional programs in IND-enabling studies by end of 2023**		
LRRK2, Tau†, α-Synuclein, mHTT ***Undisclosed Targets***	**Neurodegenerative Disorders**	**LRRK2 IND/CTA expected in 2023**			

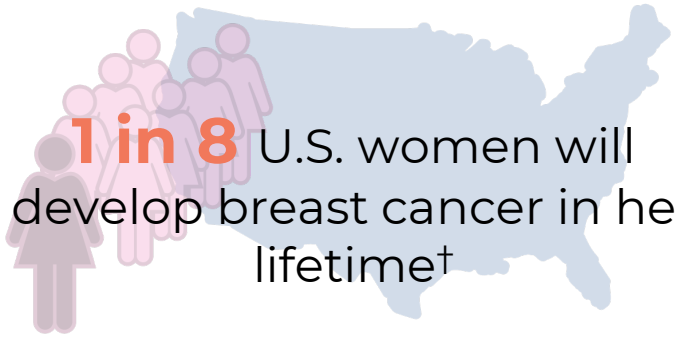
Anticipated

★ *Pivotal Trial*

These agents are currently under investigation. Their safety and effectiveness for these investigational uses have not yet been established. IND, = investigational new drug; CTA = clinical trial application; NHA,=novel hormonal agent

† Denotes historically undruggable proteins

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†

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

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

Fulvestrant is a successful standard of care, but has limitations; resistance is a challenge

Preclinically, vepdegestrant demonstrated superior ER degradation (>90%) and superior tumor regression versus fulvestrant

Vepdegestrant is a potent degrader of ER with the potential to become an endocrine backbone for ER+/HER2- breast cancer treatment

Very promising efficacy and tolerability profile to date

ER, estrogen receptor

† ACS: https://www.cancer.org/cancer/breast-cancer/about/how-common-is-breast-cancer.html; accessed 01/06/23

†† https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4549764/, accessed 1/4/2023

Vepdegestrant: Favorable tolerability and signals of efficacy in a heavily pretreated patient population

Vepdegestrant Phase 2 Patients *Prior Treatment:*	Prior CDK4/6i **100%**	Prior Fulvestrant **79%**	Prior Metastatic Chemo **45%**
Vepdegestrant demonstrated strong signals of efficacy in the VERITAC Phase 2 trial	**Clinical Benefit Rate** (Phase 2):	**38%** (All patients) **51%** (Patients with ESR1 mutant tumors)	
	Progression-Free Survival (Phase 2):	**3.7 Months** (All patients) **5.7 Months** (Patients with ESR1 mutant tumors)	
Vepdegestrant has been well tolerated	Grade 3/4 TRAE reported in 7% (5/71) in all patients Grade 3/4 TRAE reported in 6% (2/35) in patients at RP3D 200 mg		
	In 35 patients treated at the **RP3D** (200 mg), **no dose reduction and 1 discontinuation due to TRAE**		

ESR1, Estrogen Receptor 1
Clinical benefit rate defined as rate of confirmed complete response or partial response or stable disease ≥24 weeks
RP3D, Recommended Phase 3 Dose

Our VERITAC-2[1] Phase 3 pivotal trial is enrolling and designed for success

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

- In CDK4/6 inhibitor-pretreated patients, ER therapies appear to have **activity in ESR1 mutant tumors**
- In the preclinical setting, **Vepdegestrant degrades both ESR1mut and ESR1wt comparably**, and clinically has demonstrated signals of **efficacy in both *ESR1* mutant and wild-type patients**
- **VERITAC-2 is enrolling less-pretreated patients** (vs. the VERITAC Ph 2 trial)
  - In the VERITAC Ph 2 trial, less-pretreated 2L patients[2] had a numerically higher clinical benefit rate

ESR1, estrogen receptor 1; ESR1mut, ESR1 mutant; ESR1wt, ESR1 wild type

[1] NCT05654623

[2] Patients without prior fulvestrant or chemotherapy for advanced disease (n=8)

Phase 3 VERITAC-3 first-line study in combination with palbociclib is currently enrolling study lead-in

VERITAC-3 Phase 3 study design (NCT05909397)

Study lead-in (SLI) N=50

Randomized w/comparator arm (N~1130)

Randomize 1:1

Open-label SLI to assess:

- rate of Grade 4 neutropenia
- dose reductions
- dose discontinuations

Cohort A: Vepdegestrant 200 mg + palbociclib 100 mg (N=25)
Palbociclib 100 mg daily 3/1 schedule + vepdegestrant 200 mg daily in 28-day cycle

Cohort B: Vepdegestrant 200 mg + palbociclib 75 mg (N=25)
Palbociclib 75 mg daily 3/1 schedule + vepdegestrant 200 mg daily in a 28-day cycle

Selection of palbociclib dose (100 or 75 mg) with 200 mg vepdegestrant

Randomize 1:1
Open Label

Investigational Arm
Vepdegestrant + palbociclib (N ≈ 565)
Vepdegestrant 200 mg in 28-day cycle + palbociclib at determined dose

Comparator Arm
Letrozole + palbociclib (N ≈ 565)
Palbociclib 125 mg daily 3/1 schedule + letrozole daily in a 28-day cycle

Key Exclusion Criteria	Primary Endpoint
• Prior adjuvant CDK 4/6i • Primary/secondary endocrine resistance • Visceral crisis	• Progression Free Survival (PFS) by investigator assessment

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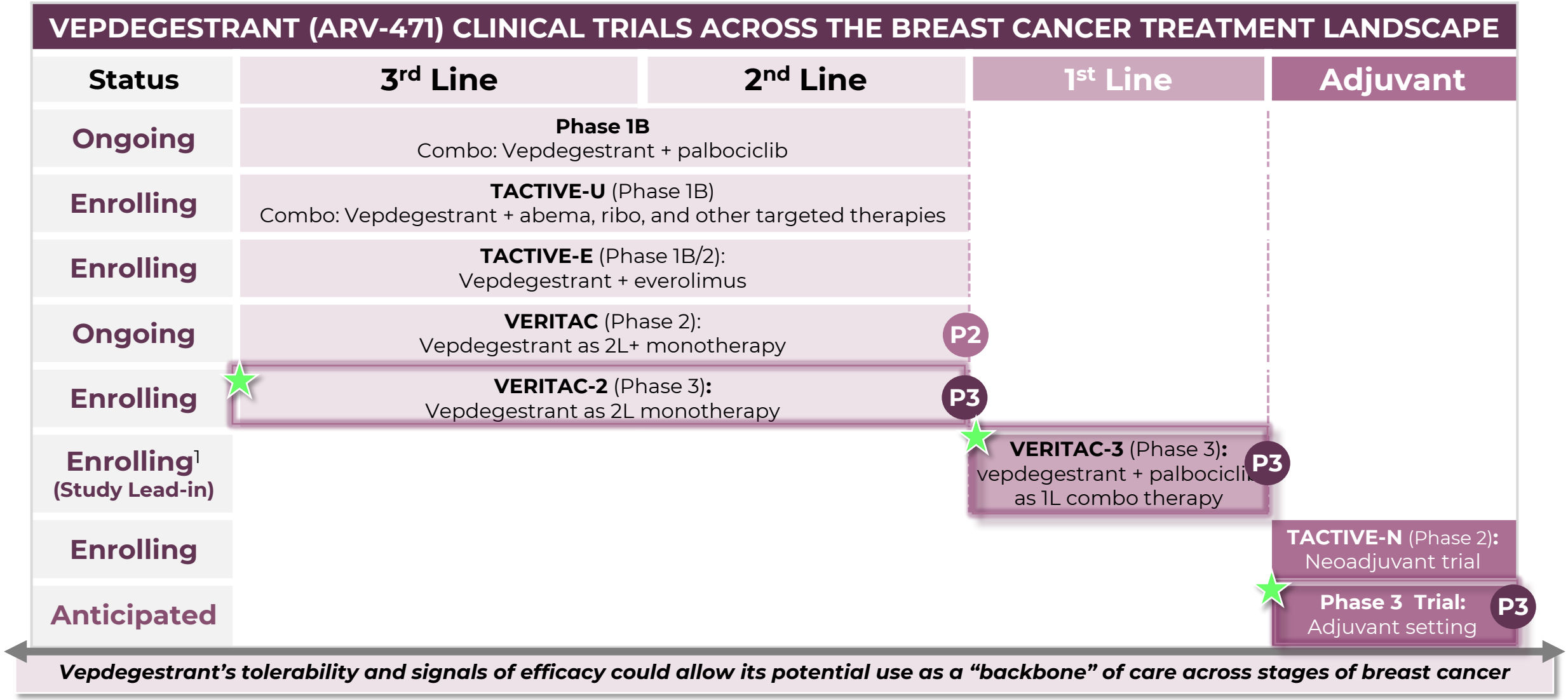
With Pfizer, we are building a robust vepdegestrant development program across multiple settings of breast cancer



[1] Sites have been activated and are open for enrollment

★ *Pivotal Trial*

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

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

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

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

Arvinas has two oral AR-targeting PROTAC degraders in Phase 2 studies:

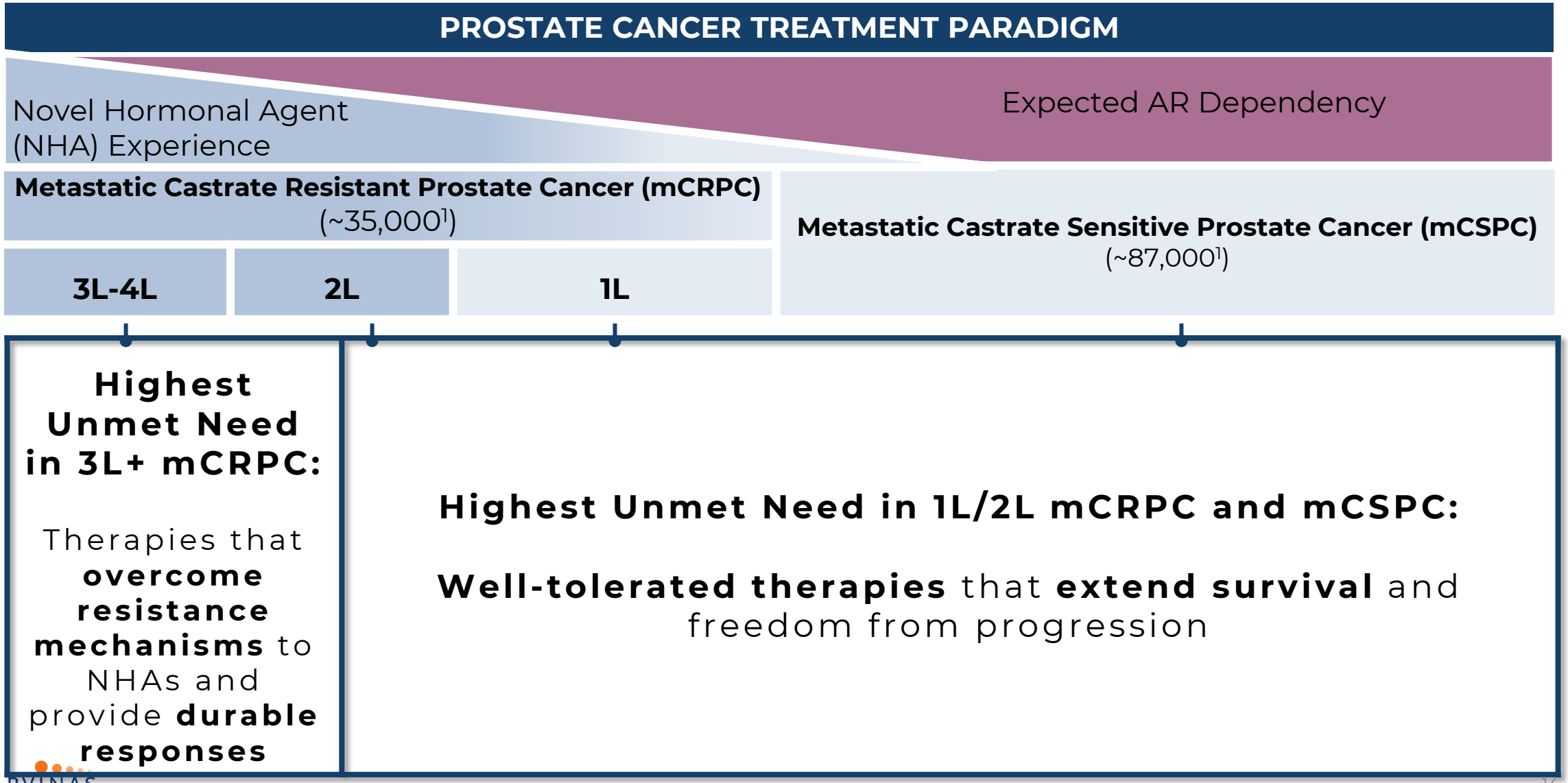
- ARV-766
- Bavdegalutamide (ARV-110)

Activity in late-line settings suggests potential for even stronger benefit in earlier-line, less-pretreated patients

AR, androgen receptor

[1] ACS: https://www.cancer.org/cancer/prostate-cancer/about/key-statistics.html, accessed 2/22/22;
[2] American Cancer Society

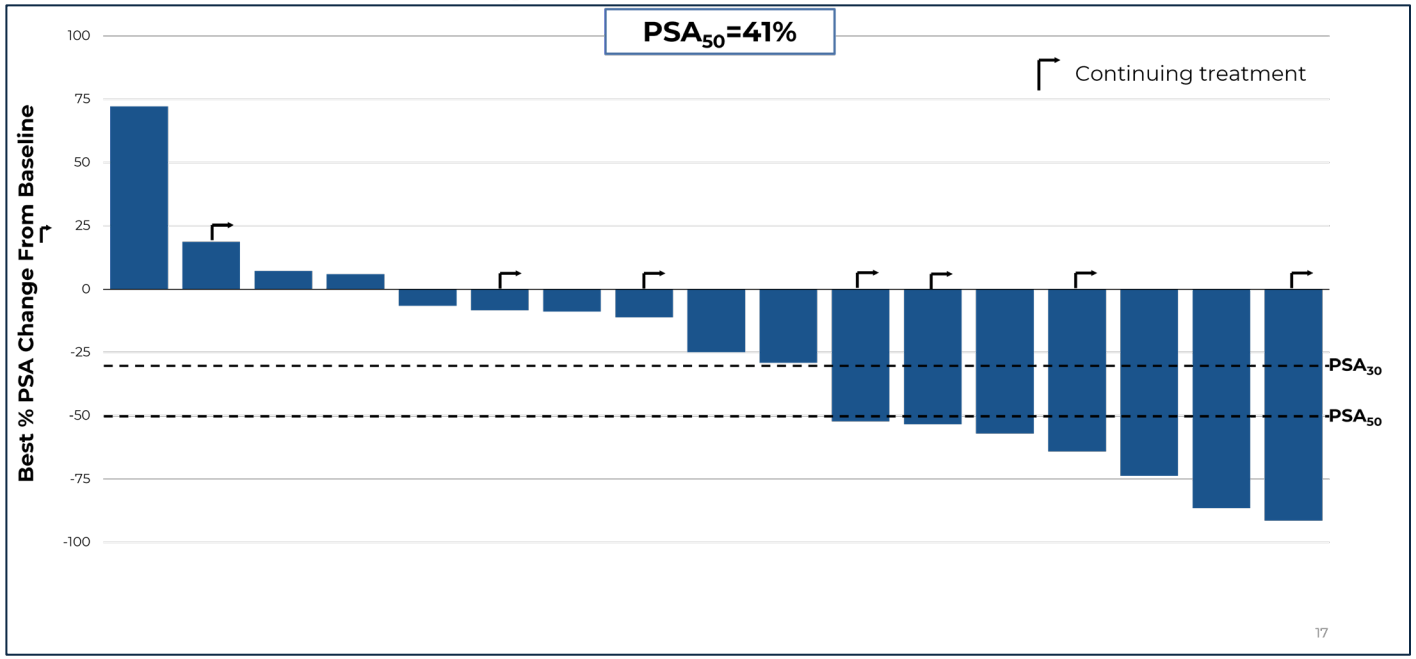
Arvinas' PROTAC® degraders could meet the substantial unmet need across the prostate cancer treatment paradigm



[1] Kantar 2023 Epidemiology Database; includes US, EU4, UK, and Japan; AR = androgen receptor

ARV-766 is showing promising efficacy signals in post-NHA late-line mCRPC and is well-tolerated, supporting its potential as an earlier-line treatment

Data presented on Oct. 22, 2023*



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

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

The emerging efficacy signals and tolerability profile of ARV-766 support development in mCRPC and mCSPC

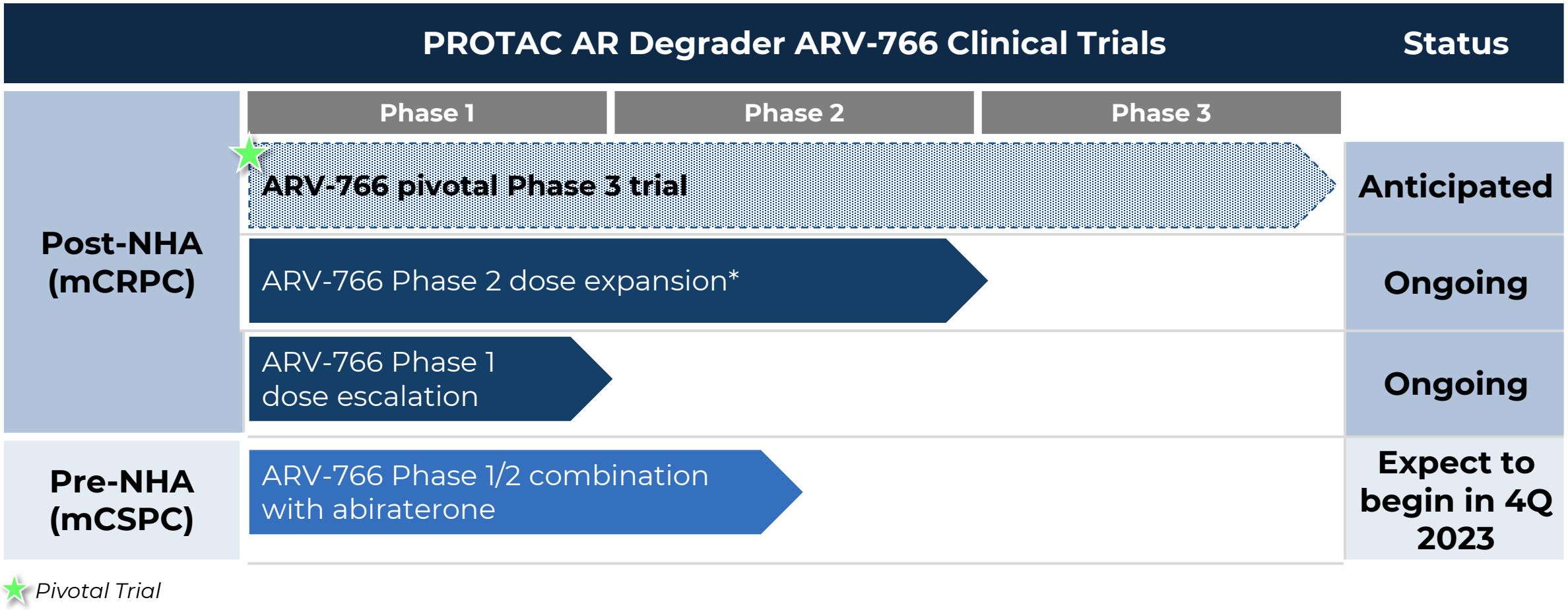
*Data cutoff = August 23, 2023
mCRPC = metastatic castrate-resistant prostate cancer; mCSPC = metastatic castrate-sensitive prostate cancer; AR LBD = androgen receptor ligand binding domain
PSA = prostate-specific antigen; PSA_{50} = best PSA declines ≥50%

ARV-766's promising tolerability profile makes it well-suited across the entire prostate cancer disease continuum

TRAE ≥10% n (%)	ARV-766 (n=84†)	
	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)	

TRAE = treatment related adverse events
† As of August 23, 2023

Strong data support opportunity for ARV-766 in the post-and pre-NHA settings



*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

ARV-766 has the potential to be first- and best-in-class PROTAC® AR degrader in mCRPC and mCSPC

Potential for PROTAC® AR degrader to improve outcomes in patients with prostate cancer	ARV-766
Degrades wild type and amplified AR	✓
Targets all AR LBD mutations	✓
Tolerability suitable for mCRPC	✓
Tolerability suitable for mCSPC	✓
Phase 3 ease of enrollment	✓
Addressable mCRPC patient population†	**~35,000**
Addressable mCSPC patient population†	**~87,000**

ARV-766:

- Strong signals of efficacy and excellent tolerability
- Pan-AR PROTAC® degrader designed to degrade wild-type AR and all clinically relevant AR mutations
- Profile to date support opportunity in early- and late-line settings

† Kantar 2023 Epidemiology Database; includes US, EU4, UK, and Japan
AR = androgen receptor; LBD = ligand-binding domain; mCSPC = metastatic castrate-sensitive prostate cancer; mCRPC = metastatic castrate-resistant prostate cancer

Industry leading preclinical pipeline of degraders

Arvinas' pipeline is differentiated and sustainable

20+ ***Pre-clinical programs across oncology and neurodegenerative disease***

4 ***first-in-human studies of new PROTAC® programs beginning in the next 24 months***

The capabilities of our PROTAC® platform remain unmatched

The deepest and most diverse pipeline of any protein degradation company

We expect our BCL6 PROTAC® degrader to be a first-in-class potential therapy for Diffuse Large B-Cell Lymphoma (DLBCL)

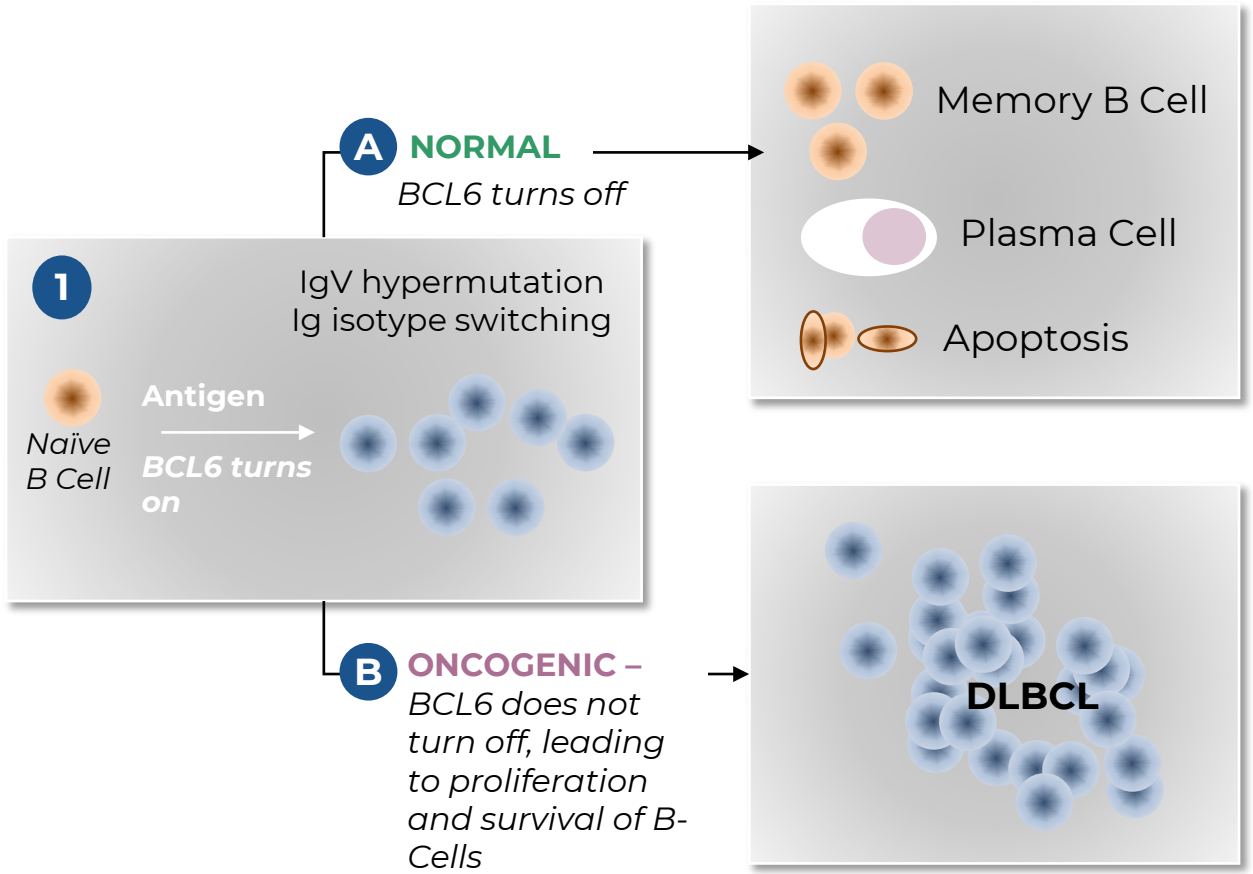
BCL6 is genetically mutated in up to 85% of DLBCL[1], a subset of Non-Hodgkin's Lymphoma

More than 18,000 people are diagnosed with DLBCL each year[2]

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

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

The role of BCL6 in driving DLBCL[3]



DLBCL, diffuse large B cell lymphoma; Ig, immunoglobulin

[1] J Iqba et. al., 2007

[2] Lymphoma Foundation, bit.ly/3jAni1S;

[3] Figure adapted from Pasqualucci et. al., 2003 (figure at bit.ly/3Q8IGHH)

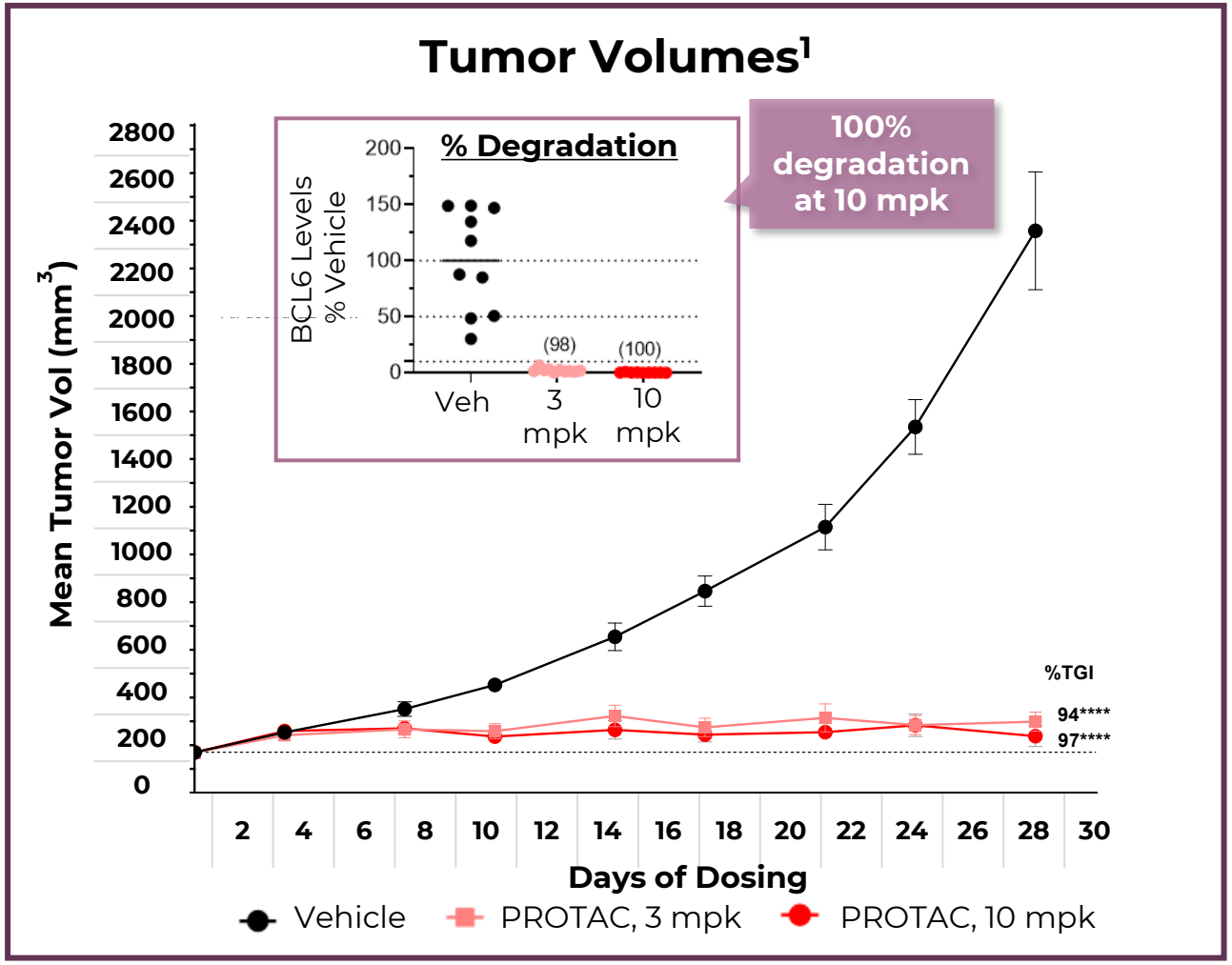
Our oral, BCL6-targeting PROTAC® clinical candidate inhibits tumor growth by nearly 100% in preclinical models

Complete tumor stasis at low, oral daily doses

Tumor stasis correlates with 95-100% degradation of measurable BCL6

Similar activity in multiple DLBCL models, including for activated B cell and germinal center B cell lymphomas

Program is currently in GLP toxicity studies; IND/CTA expected in 2H23



DLBCL, Diffuse Large B-Cell Lymphoma; IND, investigational new drug application; CTA, clinical trial application; mpk, milligrams per kilogram

[1] Model: OCI-Ly1. Dosing is oral, twice daily

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

We are creating PROTAC® degraders that can:

- ✓ **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**
- ✓ **Be delivered orally**

Significant potential advantages over existing modalities

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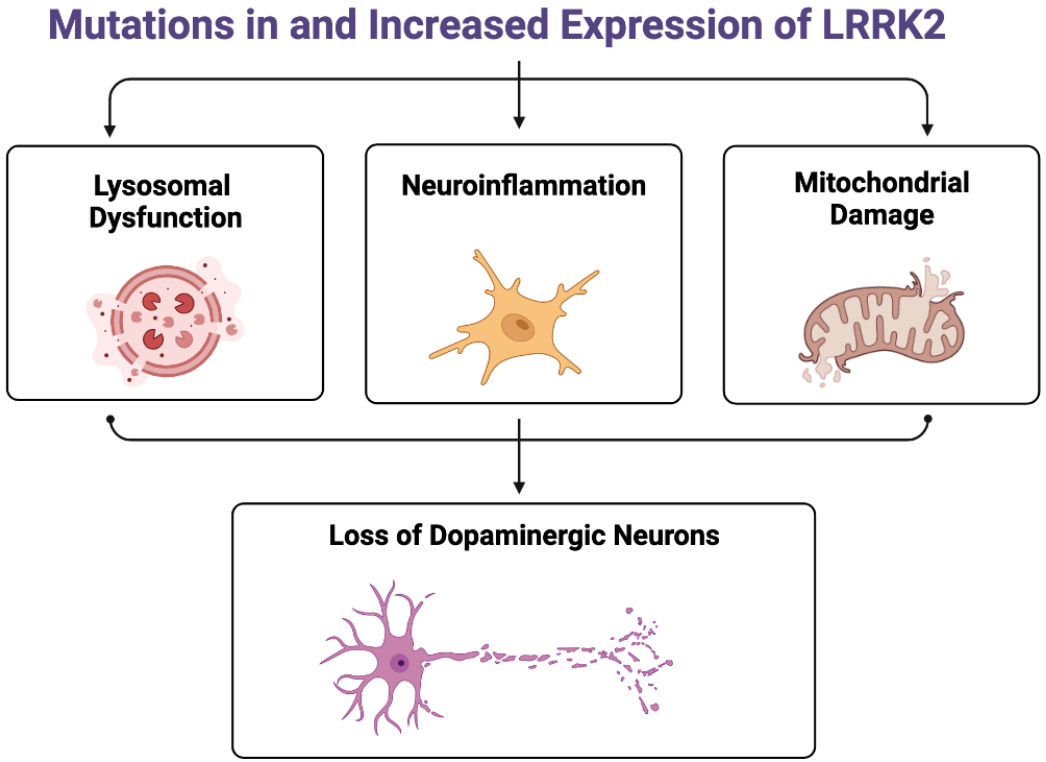
PROTAC®-induced LRRK2 degradation could be a disease-modifying modality for Parkinson's Disease

LRRK2 is a multidomain scaffolding kinase that contributes to PD (familial and idiopathic)

- Parkinson's Disease (PD) is the second most common neurodegenerative disease, with a diagnosed prevalence of 2.5M in the US, EU5, and Japan
- No disease-modifying therapies have been approved for PD
- Familial mutations and sporadic variants (~2x increase in expression) implicate leucine-rich repeat kinase 2 (LRRK2) in PD
- Human genetics and preclinical animal model data suggest that reduction of 50% of LRRK2 protein, but not kinase inhibition, may impact pathology and dysfunction in PD[1]

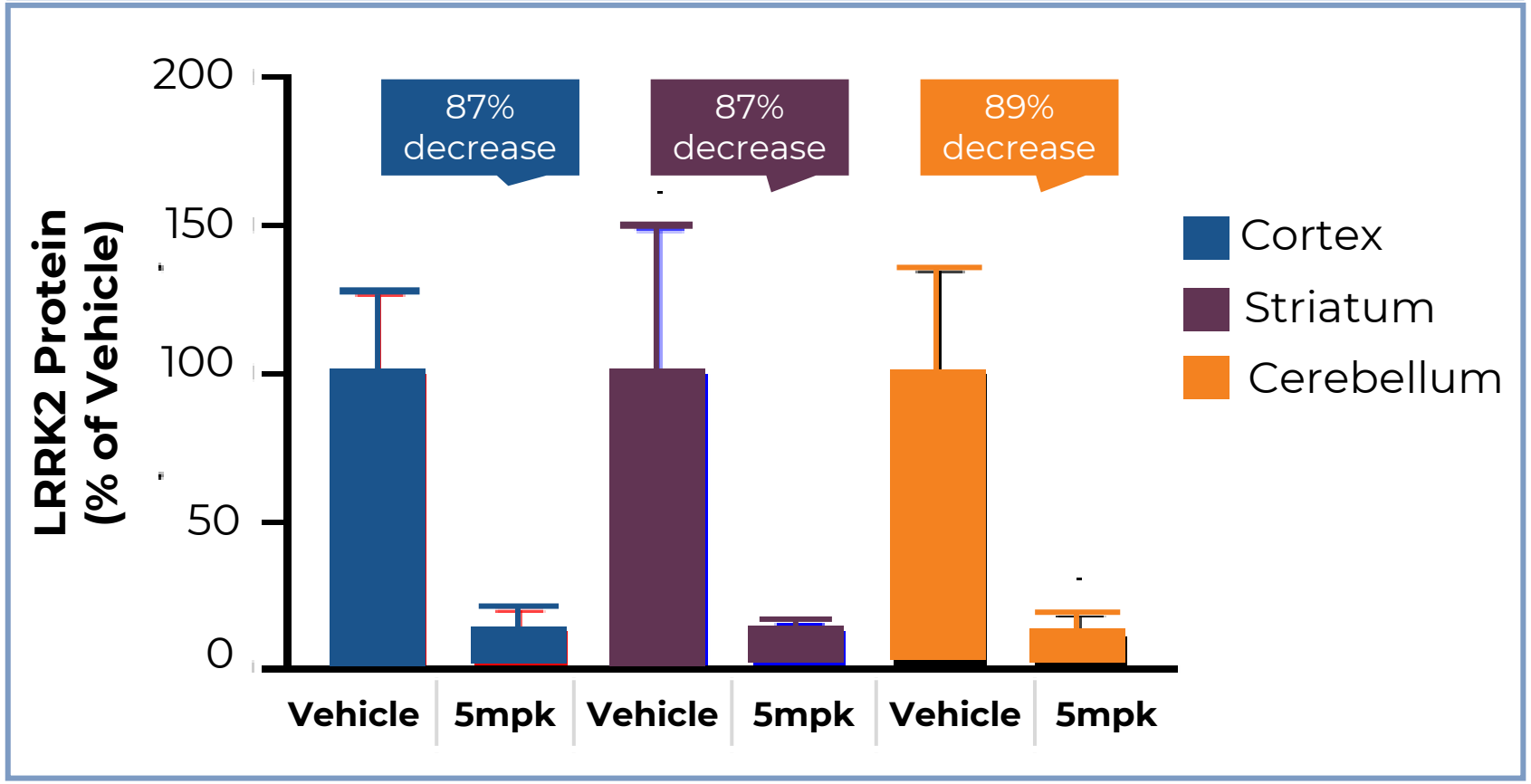


LRRK2, leucine-rich repeat kinase 2; PD, Parkinson's Disease

[1] Wang, 2021, Zhao, 2017, Henderson, 2019

Our oral PROTAC® clinical candidate reaches multiple "deep brain" regions in non-human primates and degrades LRRK2

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



Program currently in GLP toxicity studies

IND/CTA expected in 2H23

CTA, clinical trial application; GLP, good laboratory practice; IND, investigational new drug application; LRRK2, leucine-rich repeat kinase 2; mpk, milligrams per kilogram

Data as presented at SfN 2022

Rapid pace of upcoming milestones

Program	Anticipated Milestones in next 18 months
Vepdegestrant (ER PROTAC®)	• Present data from the Phase 1b combination trial with palbociclib at San Antonio Breast Cancer Symposium (Dec. 2023) • Continue enrollment in the study lead-in of the VERITAC-3 Phase 3 trial with vepdegestrant + palbociclib as a first-line treatment in patients with metastatic breast cancer • Initiate Phase 1b/2 with vepdegestrant plus Pfizer's CDK4 (cyclin dependent kinase) inhibitor (2H 2023) • Initiate an addition arm of the Phase 1b/2 combination umbrella trial (TACTIVE-U) with Carrick Therapeutics' CDK7 inhibitor (2H 2023) • Complete enrollment in the TACTIVE-N phase 2 trial of vepdegestrant as a monotherapy in the neoadjuvant setting in patients with ER+/HER2- localized breast cancer (2024) • Complete enrollment for VERITAC-2 Phase 3 monotherapy trial in patients with metastatic breast cancer (2H 2024)
ARV-766 (AR PROTAC®)	• Initiate Phase 1/2 trial with ARV-766 in combination with abiraterone in patients who have not previously received novel hormonal agents (4Q 2023) • Continue enrollment of Phase 2 dose expansion study with ARV-766, with PFS data anticipated in 2024 • Initiate discussions with regulatory authorities to align on plan for Phase 3 trial with ARV-766 in mCRPC (2Q 2024)
Bavdegalutamide (AR PROTAC®)	• Complete enrollment in Phase 1b combination study with abiraterone (4Q 2023)
INDs	• Submit two investigational new drug (IND)/clinical trial authorization (CTA) applications for BCL6 (oncology) and LRRK2 (neurology), by year end 2023, with at least two additional programs in IND- or CTA-enabling studies

Thank You

For More Information

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