



J.P. Morgan Healthcare Conference

January 11, 2023



Safe harbor and forward-looking statements



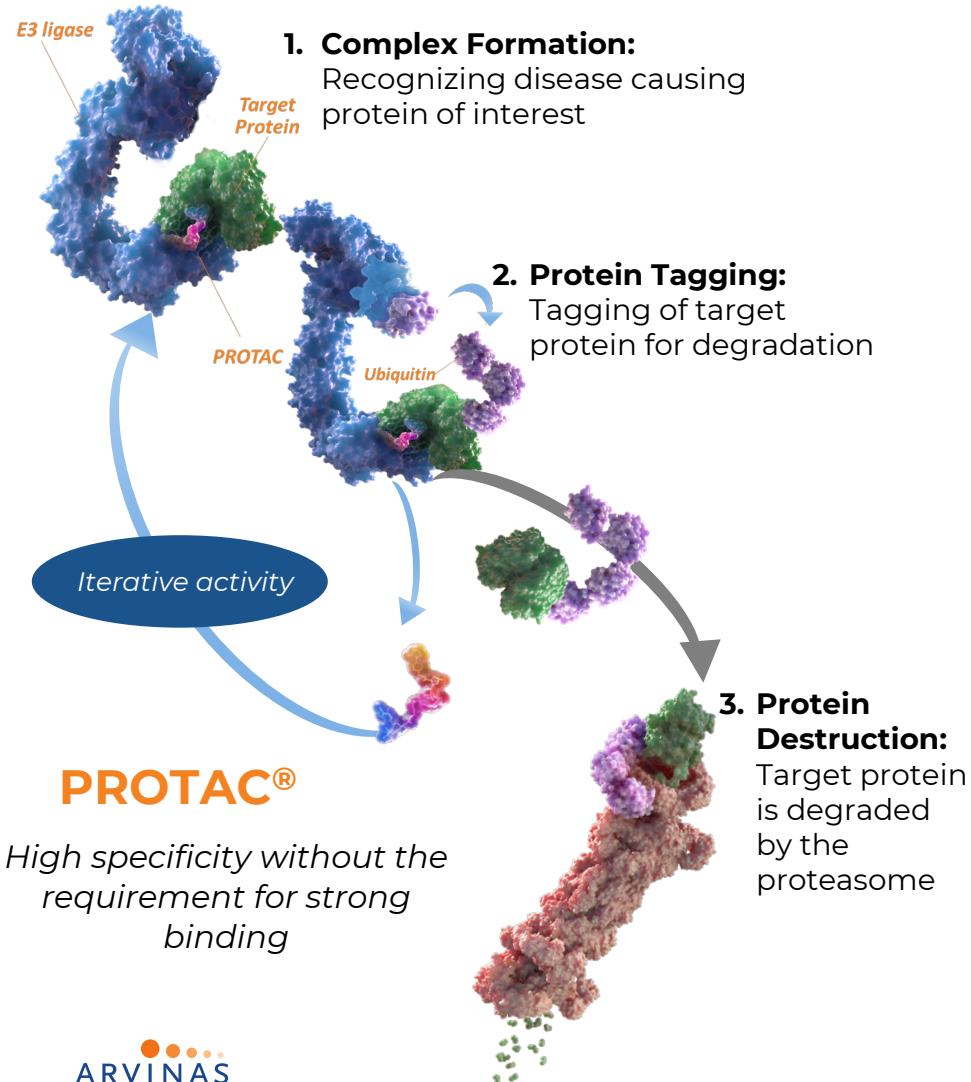
This presentation contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995 that involve substantial risks and uncertainties, including statements regarding the anticipated timing of our planned clinical trials within our pipeline, including VERITAC-3, a trial of ARV-471 in combination with palbociclib, our ARV-471 monotherapy study in the adjuvant setting, and our bavdegalutamide (ARV-110) monotherapy study; the potential therapeutic benefits of ARV-471; the expected timing for submission of investigational new drug applications or clinical trial authorization applications for our preclinical candidates as well as timing of initiation of two additional enabling studies for our preclinical candidates; whether our preclinical programs will help treat patients with solid and haematological cancerous 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; whether our PROTAC® degraders eliminating the androgen receptor, or AR, may surpass the benefits of AR inhibitors and the extent to which an AR-targeting PROTAC® degrader may address the unmet needs of patients with prostate cancer across multiple stages of disease; the timing for beginning a pivotal trial for bavdegalutamide and AR PROTAC® investigations in pre- and post-novel hormonal agent settings; whether our BCL6 PROTAC® degrader will be a first-in-class potential therapy for Diffuse Large B-Cell Lymphoma; and the timing of clinical trial initiations, including pivotal trials, first in human studies of PROTAC® protein degraders and certain data readouts. 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 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 bavdegalutamide (ARV-110), ARV-766, and 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; 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.

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



ARVINAS Leaders in a new class of novel treatments

Consistent ability to create PROTAC® degraders with drug-like properties and signals of clinical efficacy and tolerability

One program in a Phase 3 study, and two drug candidates in Phase 2

Creating **potential therapies for patients** in both **oncology** and **neuroscience**

~\$1.3B cash on-hand¹

Partnerships with global leaders in drug discovery, development, and commercialization



Genentech
A Member of the Roche Group

Certhbio™
BAYER

GNS

Insilico Medicine

PHOREMOST

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

Program	Therapeutic Area / Indication	Preclinical	Phase 1/1b	Phase 2	Phase 3
ARV-471 Global co-development/ co-commercialization partners with 	Oncology: ER+/HER2- Breast Cancer	★ VERITAC-2: ARV-471 monotherapy 2L pivotal trial ★ VERITAC-3: ARV-471 + palbociclib as 1L combination therapy ★ ARV-471 monotherapy in the adjuvant setting	VERITAC: ARV-471 monotherapy dose expansion (2L+) TACTIVE-N: ARV-471 in neoadjuvant setting TACTIVE-E: ARV-471 + everolimus TACTIVE-U: ARV-471 in combination with ribociclib, abemaciclib, and other targeted therapies		
Bavdegalutamide (ARV-110)	Oncology: Prostate Cancer	★ Bavdegalutamide monotherapy (878/875+ 2L+)	ARDENT: Bavdegalutamide monotherapy dose expansion (2L+)		
ARV-766		Bavdegalutamide + abiraterone (2L+)	ARV-766 monotherapy dose escalation (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			

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

† Denotes historically undruggable proteins

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+) ^{††}

ARV-471 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, ARV-471 demonstrated superior ER degradation (>90%) and superior tumor regression versus fulvestrant

ARV-471 is a potent degrader of ER as well as a complete ER antagonist

Very promising efficacy and tolerability profile to date

ARV-471: Excellent tolerability and signals of efficacy in the most heavily pretreated patients of any ER-targeting therapy



ARV-471 Phase 2 Patients

Prior Treatment:

Prior CDK4/6i
100%

Prior Fulvestrant
79%

Prior Metastatic Chemo
45%

ARV-471 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)

ARV-471 has been well tolerated

Grade 3/4 TRAE reported in 6% (2/35) patients at 200 mg

In 35 patients treated at the recommended Phase 3 dose (200 mg), **no dose reduction and 1 discontinuation**

Our VERITAC-2¹ Phase 3 pivotal trial is designed for success



Treatment (N = 560)

Randomize
1:1

ARV-471

200 mg orally once daily

Fulvestrant

500 mg intramuscularly

Days 1 and 15 of cycle 1 and Day 1 of subsequent cycle

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

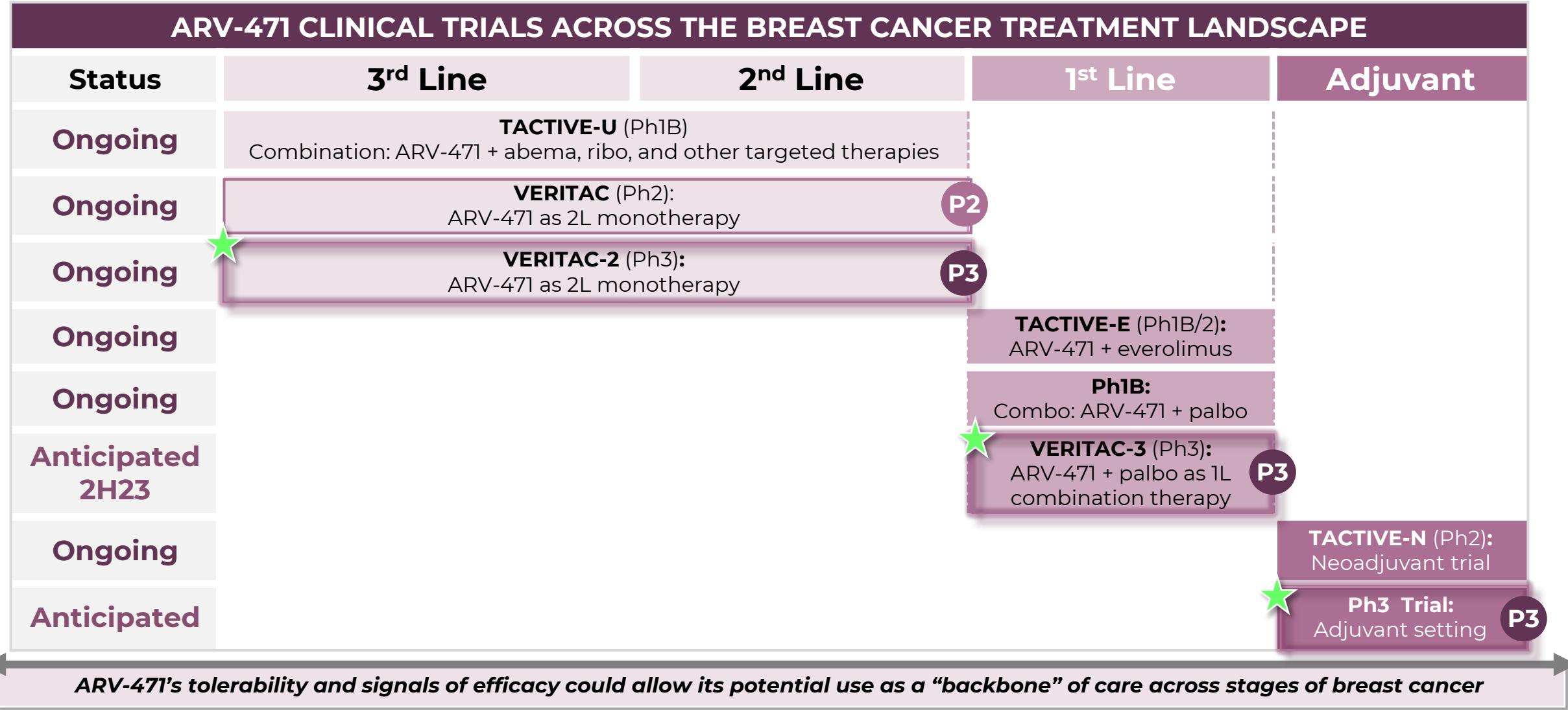
- In CDK4/6 inhibitor-pretreated patients, ER therapies appear to have **greater activity in ESR1 mutant tumors**
- ARV-471 degrades ESR1mut and ESR1wt equally**, and has already demonstrated signals of **efficacy in both ESR1 mutant and wild-type patients**
- VERITAC-2 will enroll less-pretreated, more ER-driven, 2L patients** (vs. the VERITAC Ph 2 trial)
 - In the VERITAC Ph 2 trial, less-pretreated 2L patients² 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

With Pfizer, we are building a robust ARV-471 development program to impact multiple settings of breast cancer



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 lifetime¹

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

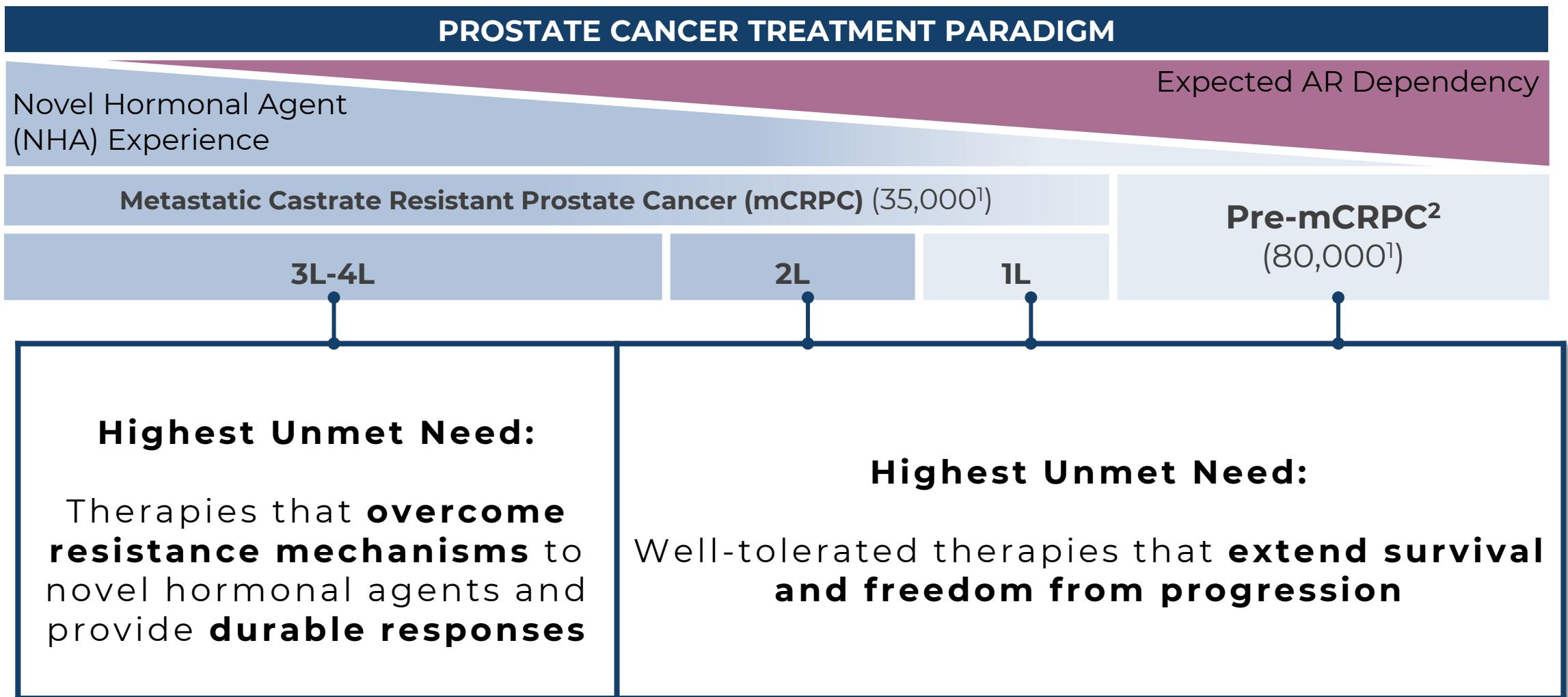
AR is a critical target in prostate cancer, but tumors develop resistance to standard-of-care AR inhibitors

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

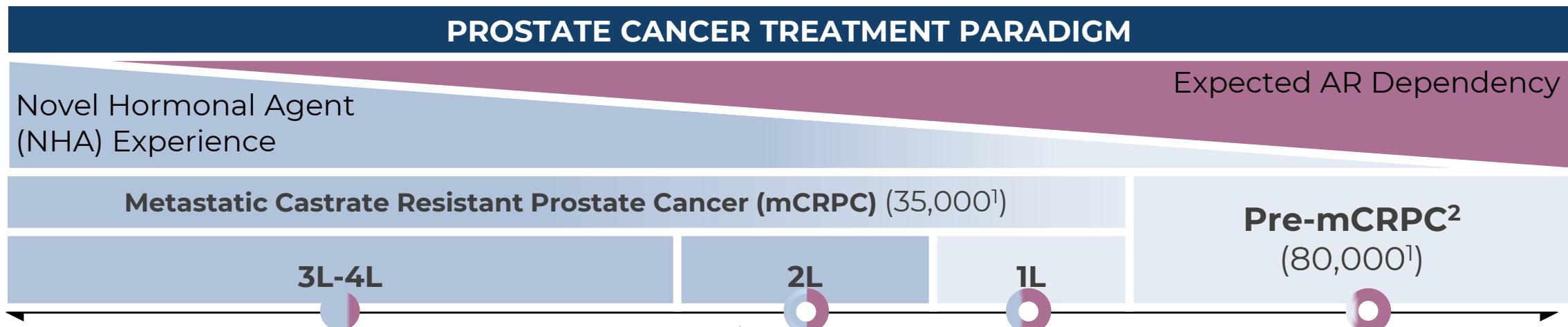
- Bavdegalutamide (ARV-110)
- ARV-766

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

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



In late-line mCRPC, bavdegalutamide has shown compelling signals of efficacy and manageable tolerability



Bavdegalutamide's tolerability and compelling signals of efficacy in this late-line, highly refractory setting...

In Phase 2, bavdegalutamide demonstrated:

- 46% PSA₅₀ response rate in patients with AR T878X/H875Y-positive tumors
- 0% Grade ≥4 TRAEs
- Low rates of discontinuation and dose reduction

Data as presented at
ASCO Genitourinary Cancers Symposium
2022

...suggest the strong potential for patient benefit in earlier settings

- Post-NHA, T878X/H875Y mutations are believed to be markers of AR dependence
- In pre-NHA settings, most patients expected to be AR-driven

In 2023, we expect to begin a pivotal trial for bavdegalutamide and to begin AR PROTAC® investigations in pre-NHA settings



Androgen Receptor (AR) Franchise Clinical Trials			Status	
	Phase 1	Phase 2	Phase 3	
Post-NHA	Bavdegalutamide pivotal Phase 3 trial			Anticipated 2H23
	Bavdegalutamide/ abiraterone combo Phase 1B			Ongoing
	ARV-766 Phase 2 dose expansion			Ongoing
	ARV-766 Phase 1 dose escalation			Data expected 2Q23
Pre-NHA	Phase 1B/2			Expect to begin in 2023

*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)



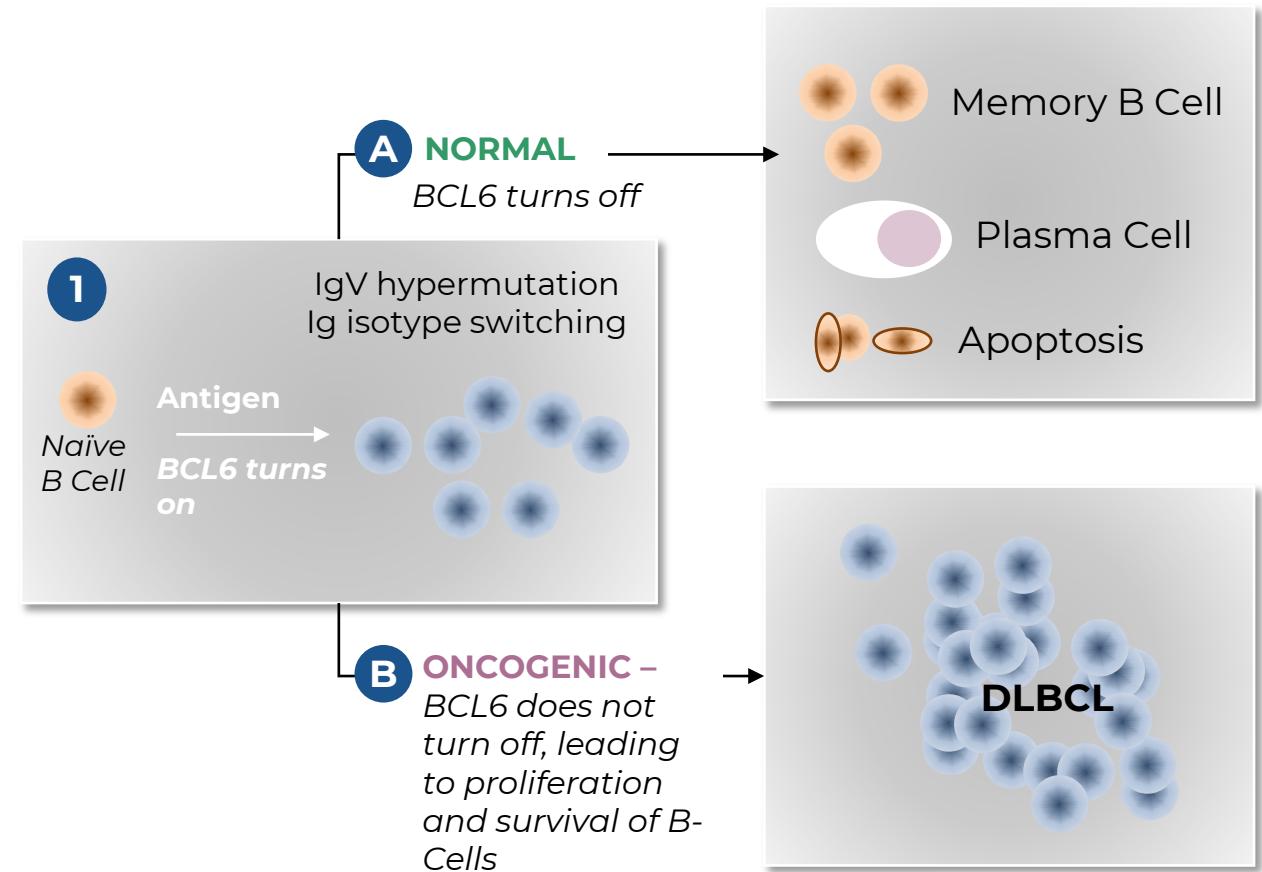
BCL6 is genetically mutated in up to 85% of DLBCL¹, a subset of Non-Hodgkin's Lymphoma

More than 18,000 people are diagnosed with DLBCL each year²

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 DLBCL³



DLBCL, diffuse large B cell lymphoma; Ig, immunoglobulin

¹J Iqba et. al., 2007

²Lymphoma Foundation, bit.ly/3jAniS;

³Figure 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

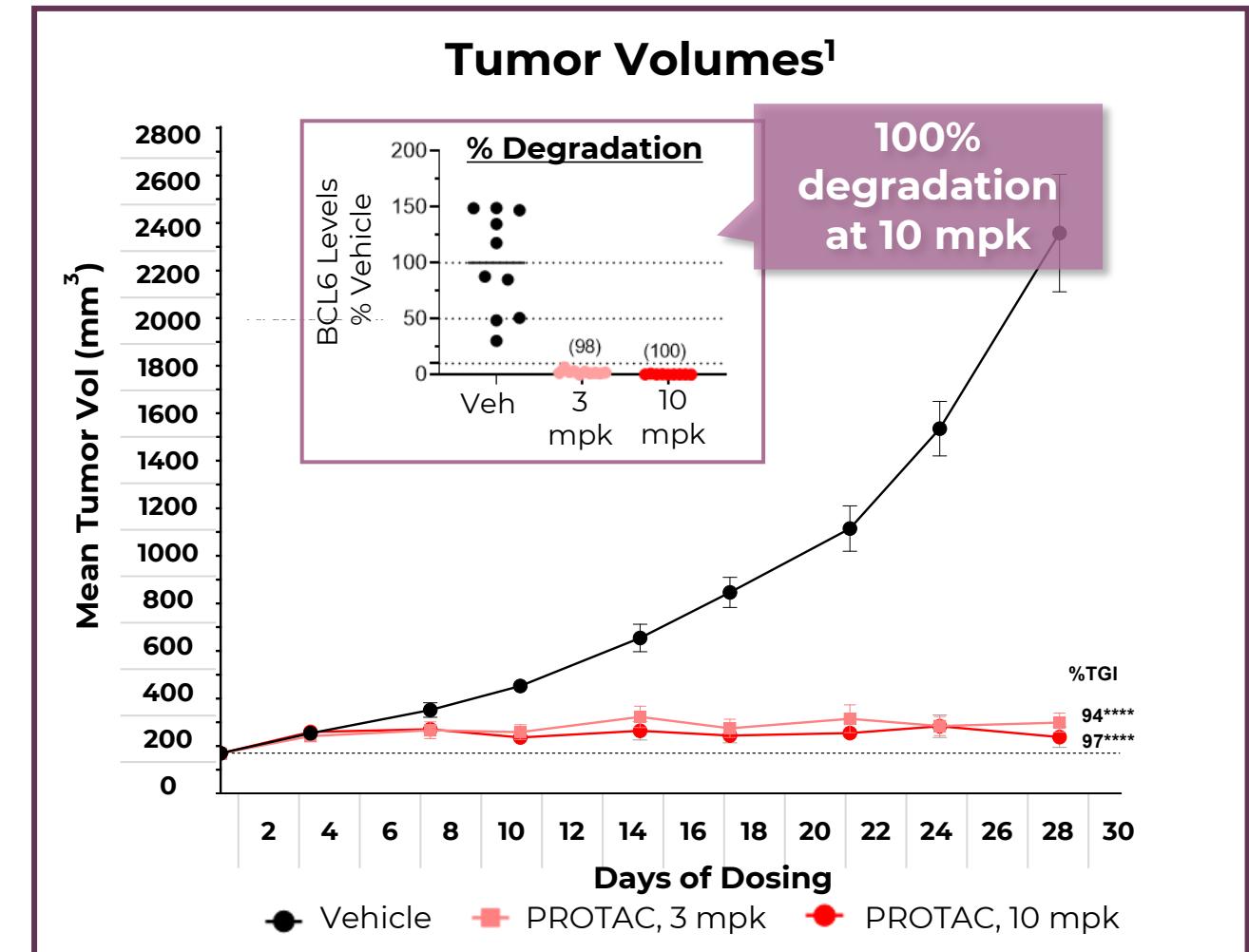


Complete tumor stasis at low, oral daily doses

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



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

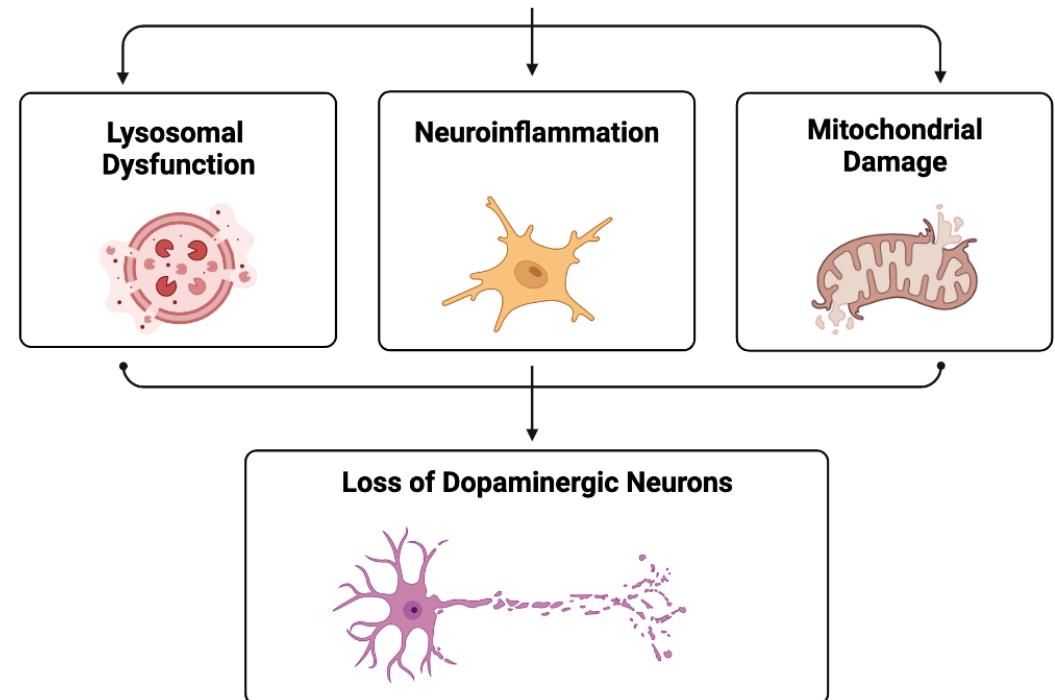
PROTAC®-induced LRRK2 degradation could be a disease-modifying modality for Parkinson's Disease



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 PD¹

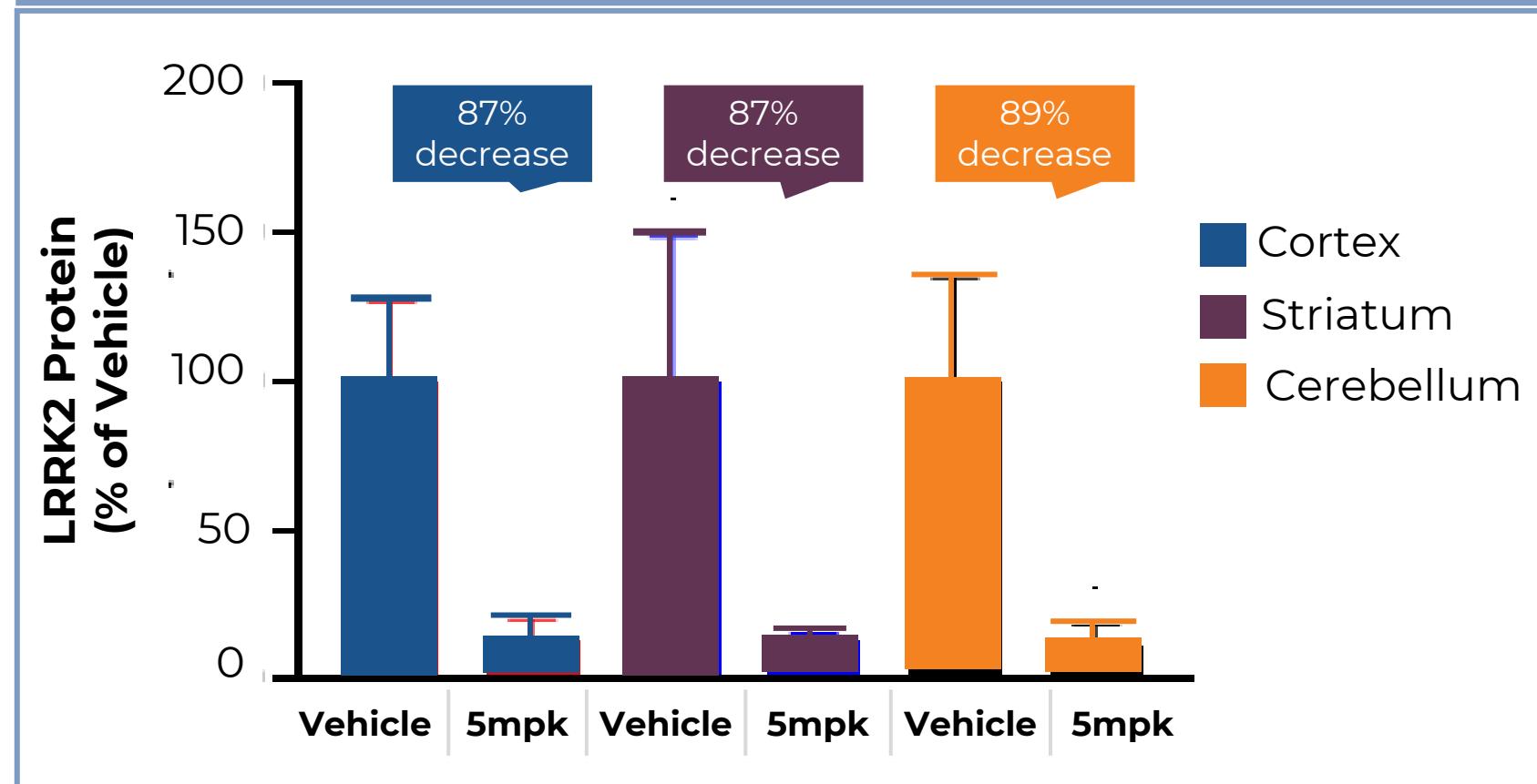
Mutations in and Increased Expression of LRRK2



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

In the next 24 months:

4

PIVOTAL TRIALS
expected to be
*ongoing in breast
and
prostate cancer*

**5+ clinical trial
data readouts
expected,
*including topline
data for*
**1 Pivotal
Trial****

**4 first-in-human
studies
of new PROTAC®
programs
anticipated
across oncology
and neuroscience**

Thank You



For More Information



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