Clinical Program Update: ARV-471 & ARV-110

ARVINAS
The PROTAC® Company

14 December 2020

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 development and regulatory status of our product candidates, such as statements with respect to our lead product candidates, ARV-110, ARV-471 and ARV-766 and other candidates in our pipeline, and the timing of clinical trials and data from those trials and plans for registration for our product candidates, and our discovery programs that may lead to our development of additional product candidates, the potential utility of our technology and therapeutic potential of our product candidates, and potential commercialization of any of our product candidates. All statements, other than statements of historical facts, contained in this presentation, including statements regarding our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans and objectives of management, are forward-looking statements. 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.

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Introduction

Agenda

Topic	Participant	
Introduction	John G. Houston, Ph.D.	*President and Chief Executive Officer*
ARV-471 Clinical Data Update	Ron Peck, M.D.	*Chief Medical Officer*
ARV-110 Clinical Data Update	Ian Taylor, Ph.D.	*Chief Scientific Officer*
Conclusion	John G. Houston, Ph.D.	*President and Chief Executive Officer*

Q&A

ARV-471 and ARV-110: Opportunities to benefit patients in large areas of unmet need

ARV-471

Estrogen receptor-degrading PROTAC®

Breast Cancer

- **Potential best profile of any ER-targeting therapy:**
  - Tolerability
  - ER degradation
  - Clinical benefit
- **Potential future endocrine therapy of choice in both adjuvant and metastatic settings**
- **Phase 1 ongoing in a highly refractory patient population**
- **>200k patients† per year with high unmet need**

ARV-110

Androgen receptor-degrading PROTAC®

Prostate Cancer

- **AR degradation and clear signals of efficacy observed in late-line mCRPC**
- **Initiated Phase 2 ARDENT trial; two potential paths to registration: 3L molecularly defined, and broader 1L/2L**
- **Extensive molecular profiling of tumors to understand drivers of resistance**
- **>250k patients† per year with high unmet need**

† US incidence data from SEER database

AR, androgen receptor; ER, estrogen receptor

ARV-471 Clinical Data Update

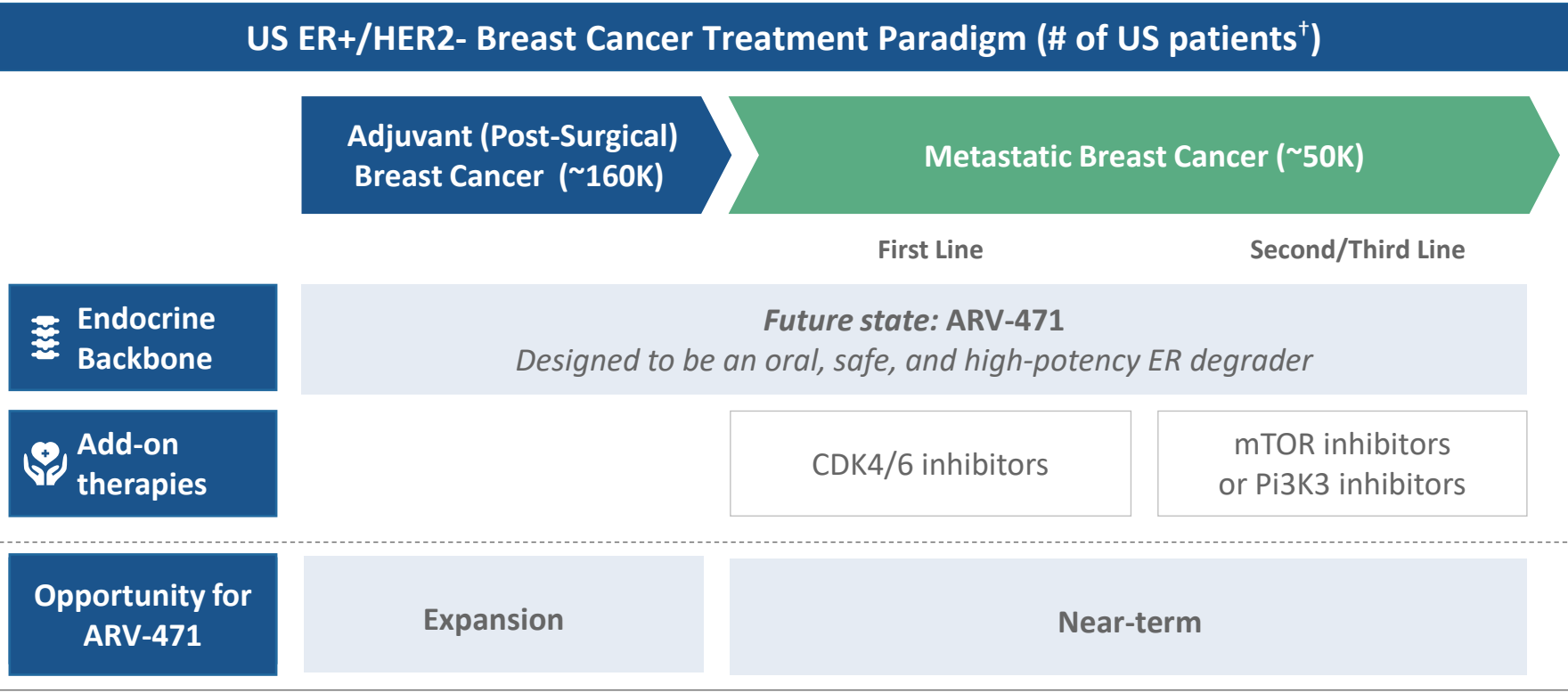
ARV-471: Potential best-in-class estrogen receptor-targeting therapy

- Potential endocrine therapy for ER+/HER2- breast cancer; >200k patients per year in the US alone†
- Outstanding tolerability profile observed, with potential for adjuvant and metastatic breast cancer settings
- Better ER degradation than fulvestrant and clinical-stage SERDs††
- Robust signals of efficacy in a patient population expected to have highly ER-independent disease, due to 100% pretreatment with CDK4/6 inhibitors
  - One confirmed partial response, and two unconfirmed partial responses
  - 42% clinical benefit rate
- Phase 1 dose escalation continues

† US incidence data from SEER database. †† As compared to previously reported data

We are developing ARV-471 to be the endocrine backbone of choice for ER+/HER2- breast cancer treatment

US ER+/HER2- Breast Cancer Treatment Paradigm (# of US patients†)



† US incidence from SEER Database

CDK: cyclin-dependent kinases, Pi3K: phosphoinositide 3-kinase; mTOR: mammalian target of rapamycin

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ARV-471 First-in-Human study is a traditional “3+3” dose escalation study

Design

- “3 + 3” dose escalation
- ARV-471 administered orally, once daily with food
- Starting dose: 30 mg

Endpoints

Primary:

- Maximum tolerated dose and recommended Phase 2 dose

Key Secondary:

- Safety and tolerability
- Pharmacokinetics
- Pharmacodynamics: Quantify ER in paired biopsies (baseline and on-treatment)
- Efficacy: RECIST, Clinical Benefit Rate (CBR) defined as confirmed PRs and CRs + ≥ 24-week SD

All Phase 1 patients were post- CDK4/6 inhibitor treatment; high rate of ER-independent resistance

Phase 1 Inclusion Criteria

- ER+/HER2- advanced breast cancer
- **Disease progression on CDK4/6 inhibitor**
- ≥ 2 prior endocrine therapies in any setting
- Up to 3 prior chemotherapy regimens in advanced breast cancer

Believed to be the only trial of an ER-targeting therapy requiring prior CDK4/6 treatment

- After CDK4/6 inhibitor treatment, **~66%** of breast cancers have ER-independent mechanisms of resistance†
- Outcomes are poor following CDK4/6 inhibitor therapy, e.g., for fulvestrant:
  - Median PFS = 1.8 months††
  - CBR estimated ≤20%††

† Wander 2020; †† Juric SABCS 2018 Subset Analysis of SOLAR1.
CDK4/6i, cyclin-dependent kinase 4/6 inhibitor. PFS, progression-free survival; TTF, time to treatment failure; CBR, clinical benefit rate

ARV-471 Phase 1 patients received extensive prior therapy (N = 21)

Patient Characteristics	Parameter	N (%)	
Median age (years)		64	
ECOG performance status	0	10	(48)
	1	11	(52)
Prior visceral disease (liver, lung)		10	(48)
Median prior lines of therapy total (range 1-9)		5	(NA)
Median number of prior endocrine regimens		3	(NA)
Type of prior therapies in advanced settings			
CDK 4/6 inhibitor		**21**	**(100)**
Fulvestrant		**15**	**(71)**
Chemotherapy		**8**	**(38)**
Investigational SERD		**5**	**(24)**
Other therapies		**14**	**(67)**

ECOG, Eastern Cooperative Oncology Group; CDK4/6, cyclin-dependent kinases; SERD, selective estrogen receptor degrader

ARV-471 is well tolerated at all dose levels; no Grade 3 adverse events

TRAE in ≥ 10% of Patients	30 mg (N=3)		60 mg (N=3)		120 mg (N=7)		180 mg (N=5)		360 mg (N=3)		Total (N=21)
	Gr 1	Gr 2	Gr 1	Gr 2	Gr 1	Gr 2	Gr 1	Gr 2	Gr 1	Gr 2	N (%)
Any	-	-	2	-	4	-	2	1	2	-	11 (52)
Nausea	-	-	2	-	1	-	-	1	1	-	5 (24)
Arthralgia	-	-	1	-	2	-	1	-	-	-	4 (19)
Fatigue	-	-	1	-	-	-	1	-	2	-	4 (19)
Decreased appetite	-	-	-	-	1	-	-	-	2	-	3 (14)

Adverse events were primarily Grade 1; No dose limiting toxicities

Data cut-off: November 11, 2020
TRAE, Treatment related adverse event

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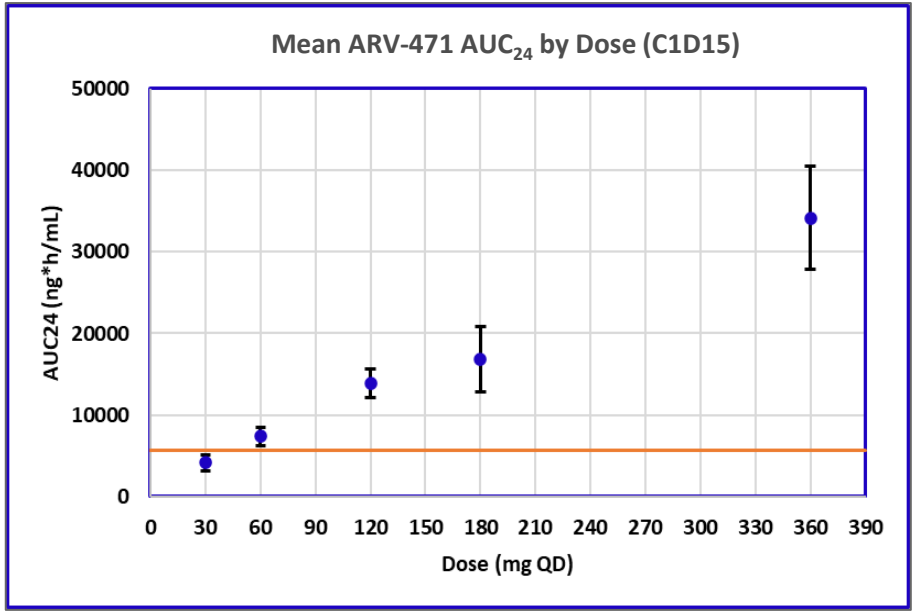
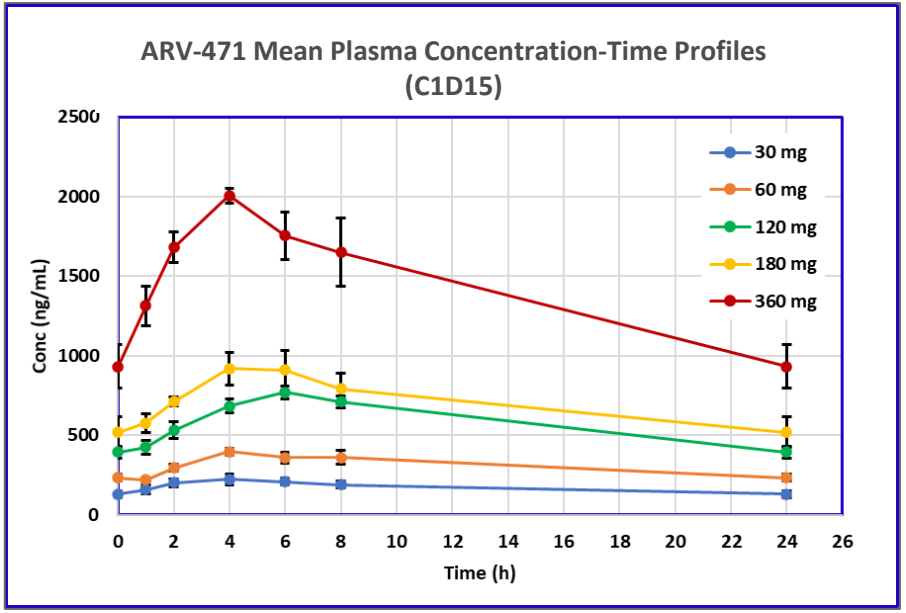
ARV-471's PK is dose proportional; exposures far exceed preclinical efficacy thresholds





The orange line represents the efficacious exposure for tumor regression in preclinical models †

Effective half-life ($T_{1/2}$) ≈ 28 hours

† AUC24=5717 ng*h/mL for preclinical effective exposure in preclinical model (mice@30mpk).
AUC, area under the curve; SE, standard error

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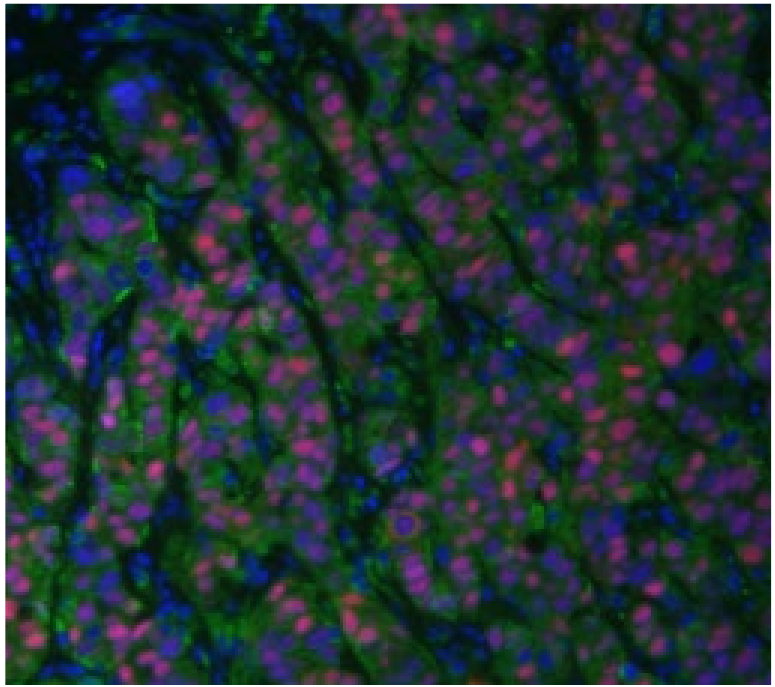
ER degradation observed in patient tumor biopsies

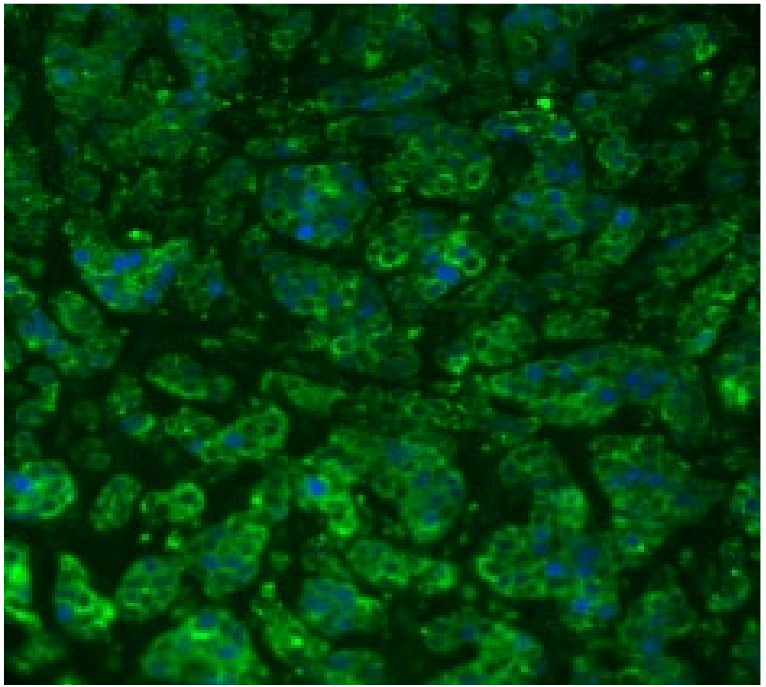
Red: Estrogen receptor

Blue: Nuclei

Green: Tumor (cytokeratin)



Baseline



After treatment with 60 mg ARV-471

Method: ER immunoreactivity analyzed by quantitative immunofluorescence (QIF) using the automated quantitative analysis (AQUA) method

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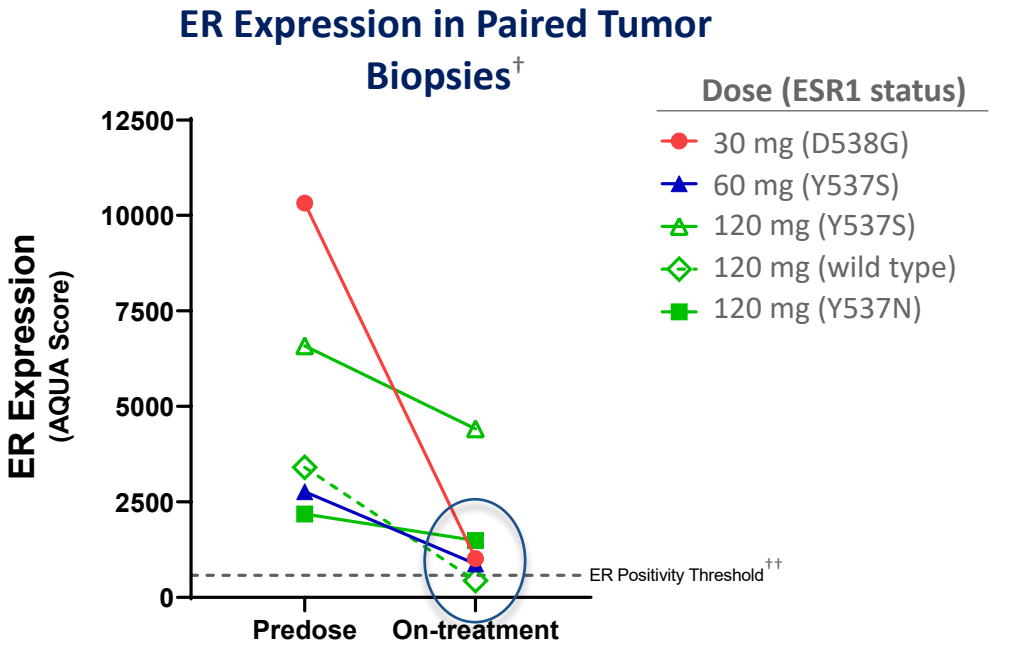
ARV-471 degraded ER up to 90% through the 120 mg dose level



Degradation up to **90%;** average of **62%**

Degradation **superior to fulvestrant** *(previously reported: 40-50%)*†††

Degradation of **wild type ER and ESR1** mutant proteins

† ER immunoreactivity analyzed by quantitative immunofluorescence (QIF) using the automated quantitative analysis (AQUA) method. †† Derived by examining AQUA scores and visually inspecting all samples in the dataset to determine a cut-point for ER positivity. ††† Fulvestrant degradation reported as 40-50% in Robertson et al., Breast Cancer Research (2013) and Kuter et al., Breast Cancer Res Treat (2012).
ESR1, Estrogen Receptor 1

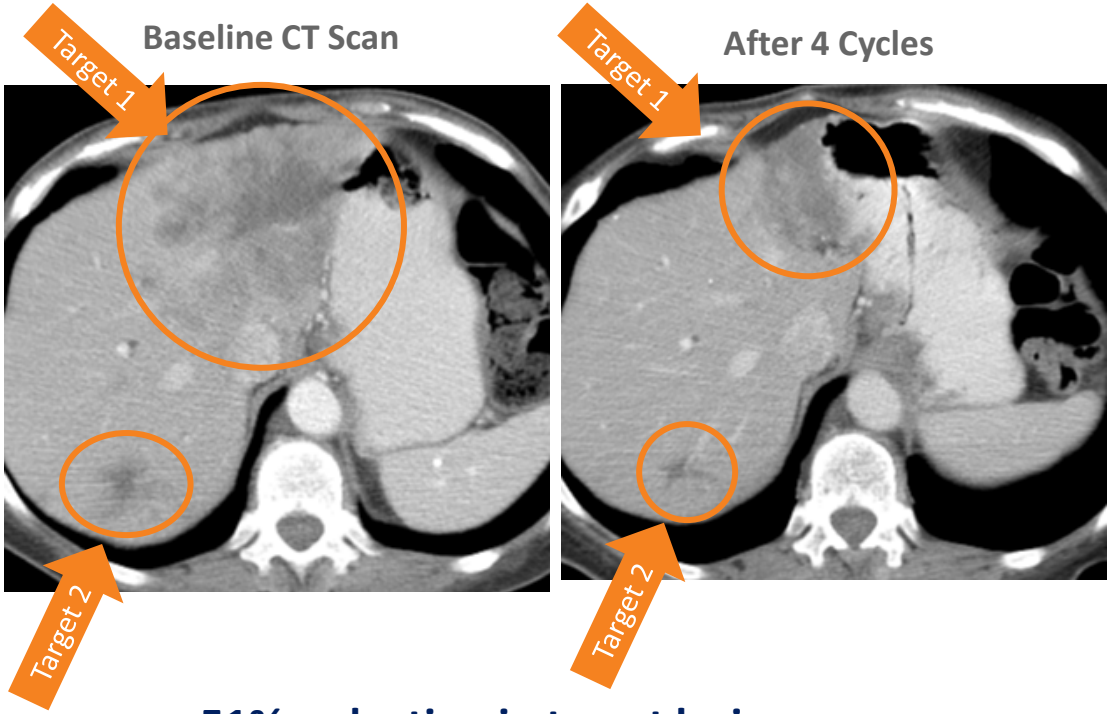
Confirmed RECIST Partial Response (cPR) in a patient with extensive prior therapy and an ESR1 mutation at 120 mg

Extensive prior therapy

- **CDK4/6 inhibitor: Palbociclib**
- **Endocrine therapies: 6 Agents**
  - Aromatase inhibitors x 3
  - Tamoxifen
  - Investigational SERDs X 2†
- **Other targeted agents: Everolimus**
- **Chemotherapy: 2 Regimens**
  - 1 neoadjuvant + 1 metastatic

ESR1 mutations

- **D538G**



51% reduction in target lesions (RECIST partial response)

† Includes one selective ERα covalent antagonist.
CDK: cyclin-dependent kinases; SERD, selective estrogen receptor degrader

Regression in chest wall lesions in a patient with extensive prior therapy and multiple ESR1 mutations at 180 mg

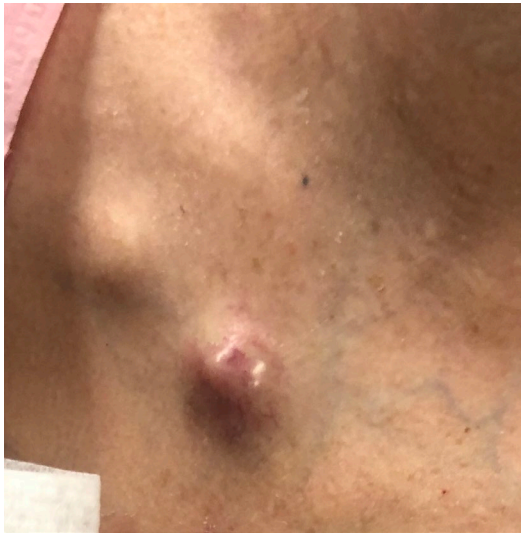
Extensive Prior therapy

- **CDK4/6 inhibitor:**
  - **Palbociclib, Abemaciclib**
- **Endocrine therapies: 3 Agents**
  - Aromatase inhibitors x 2
  - Fulvestrant
- **Other targeted agents: Everolimus**
- **Chemotherapy: 4 Regimens**
  - 1 neoadjuvant + 3 metastatic

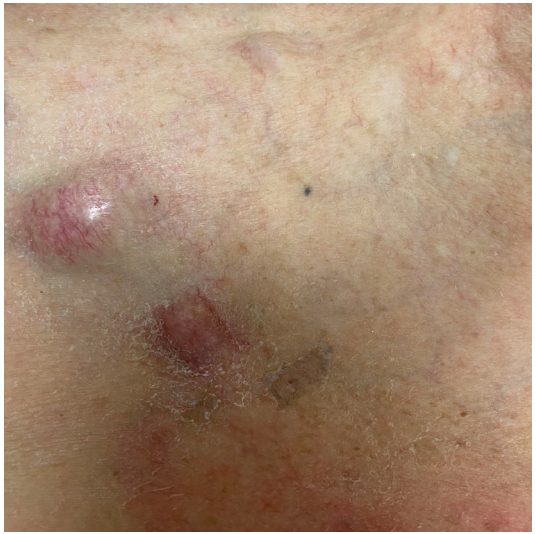
ESR1 mutations

- **D538G, E380Q, V422del, L536P**

Baseline
(Associated Bleeding)



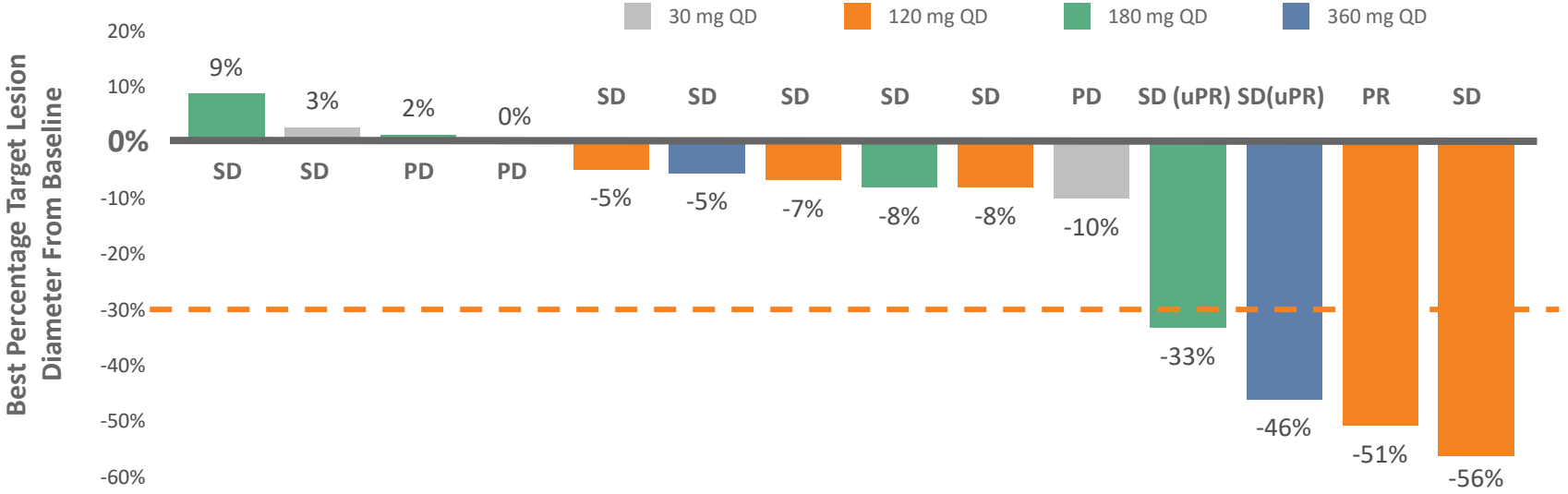
After 4 Cycles
(No Bleeding)

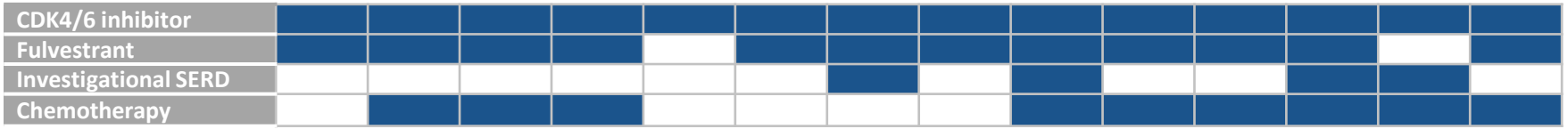


CDK, cyclin-dependent kinases

ARV-471 demonstrates promising anti-tumor activity in late line patients

Antitumor Activity in Eligible Patients (N=14)†





† 7 patients out of 21 are excluded from graph due to no measurable disease at baseline (n=4), discontinuation of treatment without post-treatment target lesion measurements (n=2), and discontinuation after 2 doses due to non-compliance (n=1).

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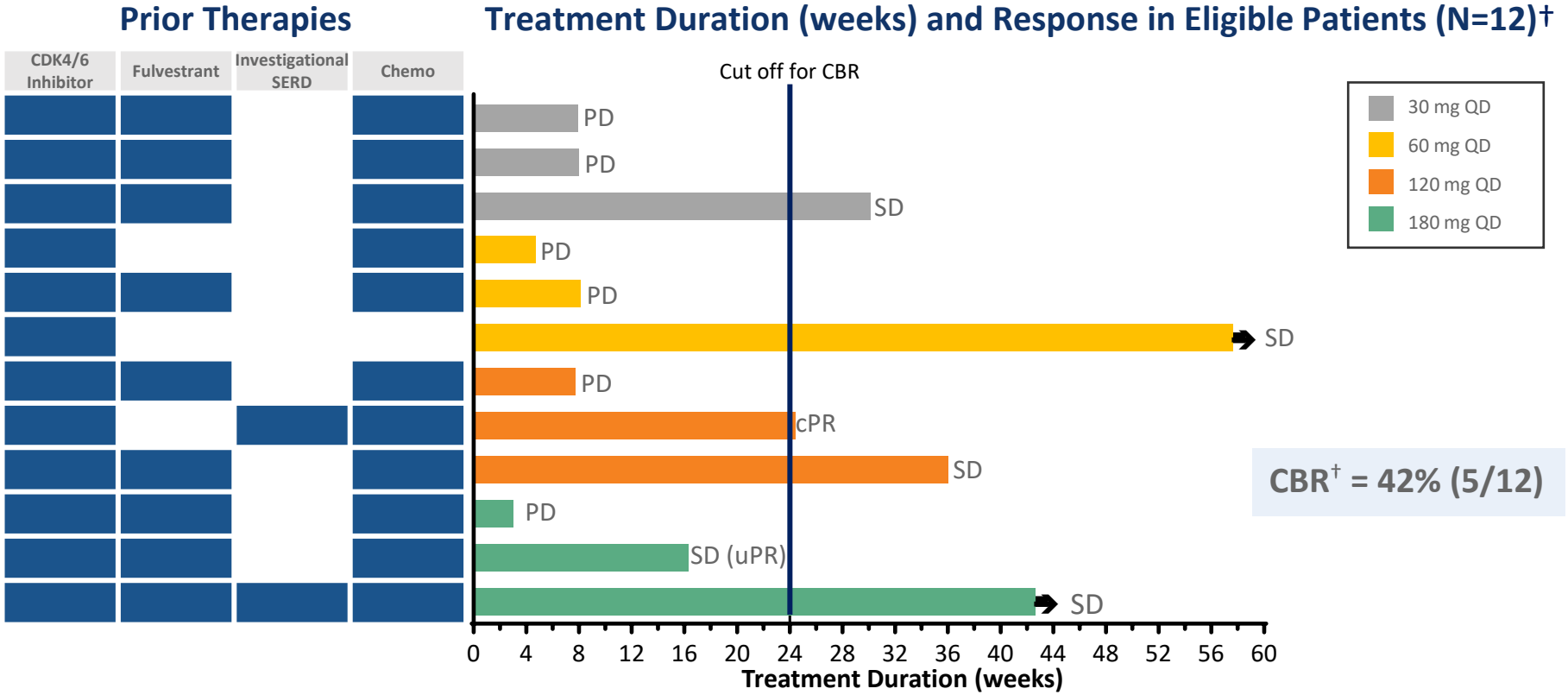
ARV-471 achieves a high clinical benefit rate (42%) in this heavily pretreated population through the 180 mg dose level



CBR† = 42% (5/12)

† Excludes 8 patients enrolled < 24 weeks prior to the data cut-off of November 28, 2020 and 1 patient who received 2 doses of ARV-471 and discontinued due to non-compliance, †† CBR defined as SD persisting ≥ 24 weeks, or a best response of confirmed CR or PR.

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Comparison of ARV-471 profile with Phase 1 data for preclinical SERDs

Phase 1 Data Comparison

Drug Candidate	CDK4/6i Pretreated Patients *(0 – 100%)*	Clinical Benefit Rate	Mean ER Degradation in Patient Tumors	Select TRAEs (> 5% of Patients)				
				Gastrointestinal (GI) AEs			Other AEs	
				Diarrhea	Nausea	Vomiting	Bradycardia	Visual disturbance
ARV-471	100%	42%	62% *Interim*		●	●		
H3B-6545	87%	34%	*Not reported*	●	●	●	●	
ZN-C5	87%	40%	*Not reported*	●	●	●		
Rintodestrant	70%	30%	28%	●	●	●		
SAR439859	63%	34%	*Not reported*	●	●	●		
AZD9833†	62%	35%	<50%††		●	●	●	●
GDC9545	59%	41%	<50%††	●	●		●	

ARV-471 has the potential to be a best-in-class ER-directed therapy

Source: H3B-6545 SABCS 2020 Poster, ZN-C5 SABCS 2020 Poster, Rintodestrant SABCS 2020, SAR439859 SABCS 2020 Poster, AZD9833 SABCS 2020 and ASCO 2020 Posters, GDC-9545 SABCS 2019 Poster. This comparison utilizes data from different Phase 1 trials and presents a non-head-to-head summary comparison.
† Reported AEs are from ASCO 2020 Poster; ††Visual estimation based on ER degradation data provided by each company.

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We aim to characterize the activity of ARV-471 across ER+/HER2- breast cancer treatment lines

US ER+/HER2- Breast Cancer Treatment Paradigm (# of US patients†)

Adjuvant (Post-Surgical) Breast Cancer (~160K)

Metastatic Breast Cancer (~50K)

	First Line		Second/Third Line	
Supportive Trials to Define Registration Paths *(planned initiation)*	**Window of Opportunity** *(Randomized vs Control)* ARV-471, or ARV-471 + CDK4/6i 2H 2021	**Phase 1b** Combo: ARV-471 + CDK4/6i (palbociclib) Dec 2020	**Phase 2** Expansion: ARV-471 1H 2021	**Phase 1b** Combo: ARV-471 + Targeted Therapy†† 2H 2021
Endocrine Backbone	**ARV-471** *Designed to be an oral, safe, and high-potency ER degrader*			

† SEER database; includes US patient population only, †† E.g., everolimus or alpelisib

CDK, cyclin-dependent kinases Pi3Ki; phosphoinositide 3-kinase inhibitor; mTORi: mammalian target of rapamycin inhibitors

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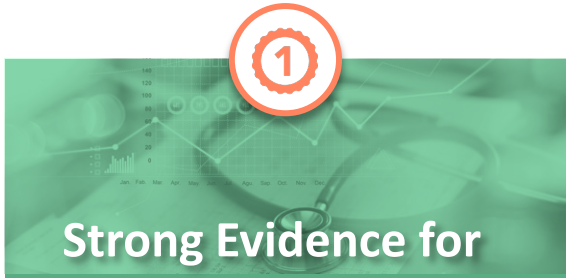
ARV-471: Evidence for best-in-class potential in a large area of unmet need



Strong Evidence for Best-in-Class Profile

- **Superior degradation to fulvestrant and SERDs**†
- **Strong efficacy signal** in a predominantly ER-independent population
- **Well tolerated**

Clear Development Path

- Potential for **2L/3L approval as monotherapy** or in **combination**
- Planned **combinations with CDK4/6 inhibitors in adjuvant or early metastatic cancers**

Large Unmet Need and Opportunity

- In the US alone, ER+/HER2- breast cancer represents an **addressable patient population of >200K**†† **per year** and a market opportunity of **>$15B**

† Fulvestrant degradation reported in Robertson et al., Breast Cancer Research (2013) and Kuter et al., Breast Cancer Res Treat (2012). †† US incidence from SEER Database.

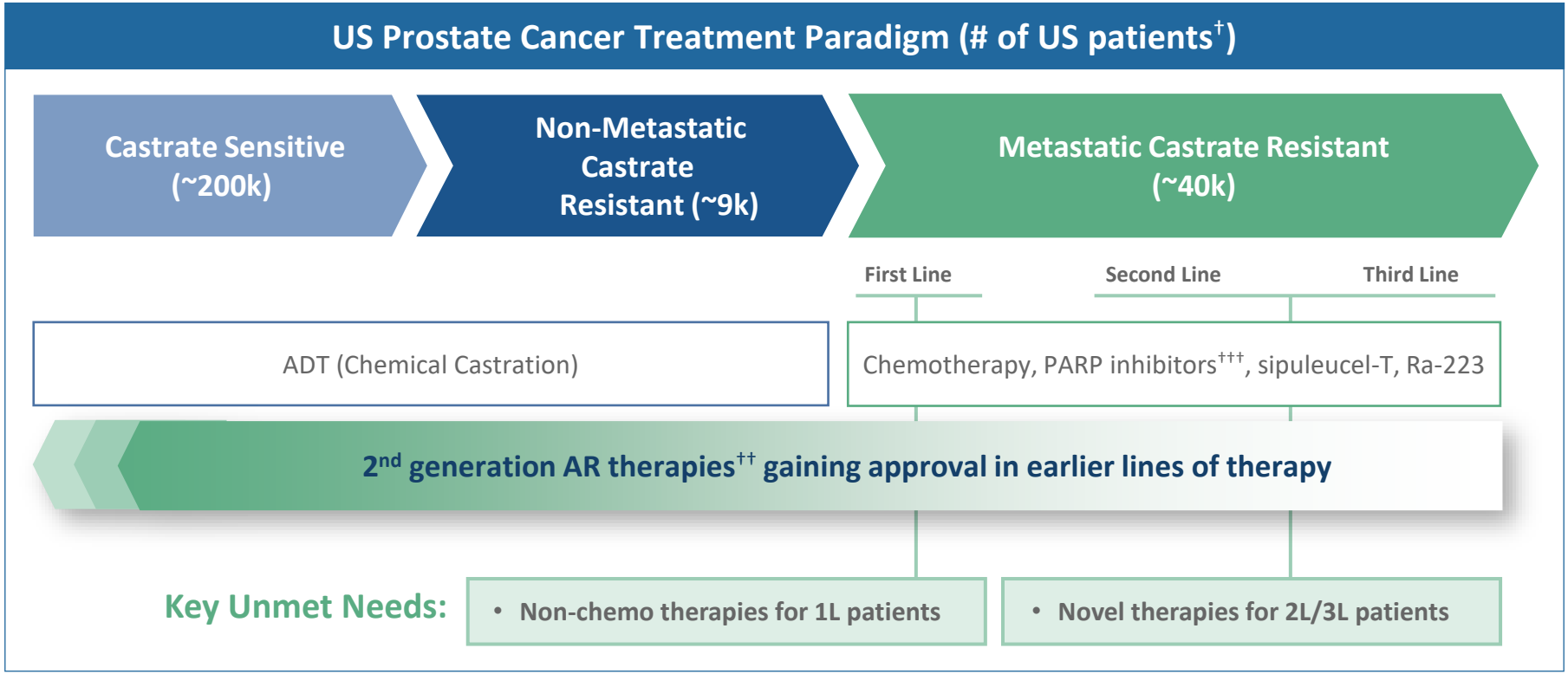
ARV-110 Clinical Data Update

ARV-110: 40% PSA50 in a molecularly defined subgroup, and additional opportunity in early-line mCRPC

- Potential best-in-class therapy for prostate cancer, representing >250k patients per year in the US alone†
- Well tolerated, escalating through the current dose of 700 mg
- Continued patient benefit: 40% PSA50 in T878/H875 patients, and additional activity in wild-type tumors
- Building substantial learnings about our late-line patient population into our development strategy
- The ongoing Phase 2 ARDENT trial is designed to confirm the potential for accelerated approval in a molecularly defined population, while also exploring the potential for approval in early-line mCRPC

† US incidence data from SEER database

Migration of second-generation AR therapies to earlier settings has created substantial unmet need for new treatments in mCRPC



† SEER database,†† Includes enzalutamide, abiraterone, darolutamide, apalutamide, ††† Approved for BRCA mutant/DNA Deficient Repair (DDR) patients progressed on 2nd gen AR-directed therapies.
ADT, androgen deprivation therapy; mCRPC, metastatic castrate resistant prostate cancer

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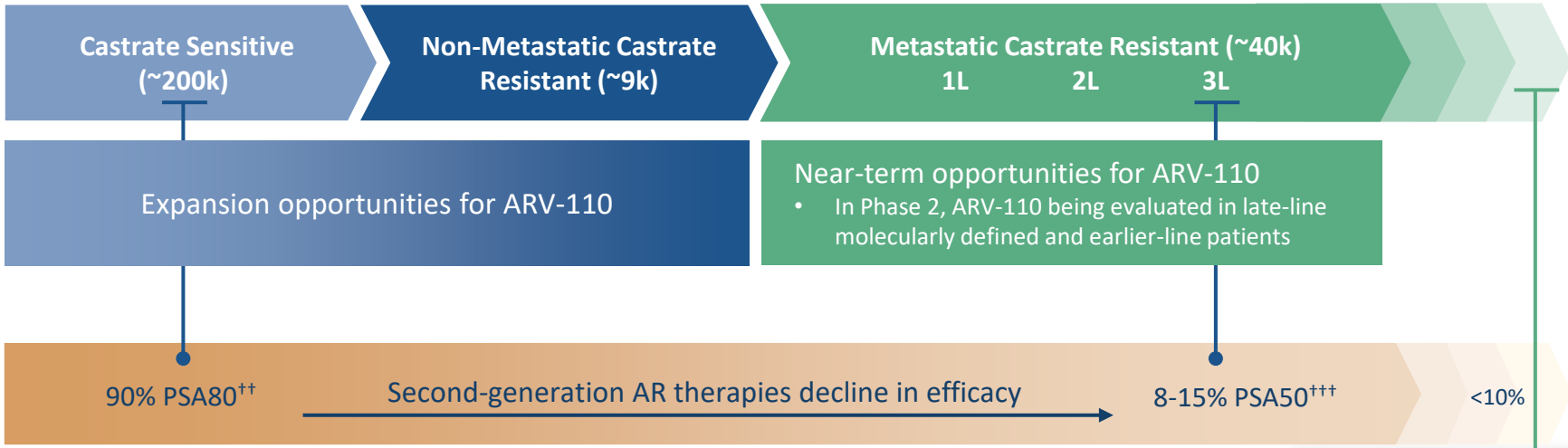
Our strategy is to develop ARV-110 across treatment settings of prostate cancer

US Prostate Cancer Treatment Paradigm (# of US patients†)



† SEER database; †† Tombal, Lancet Oncology 2014; ††† de Wit R, N Engl J Med. 2019; Hussain, ESMO 2019.

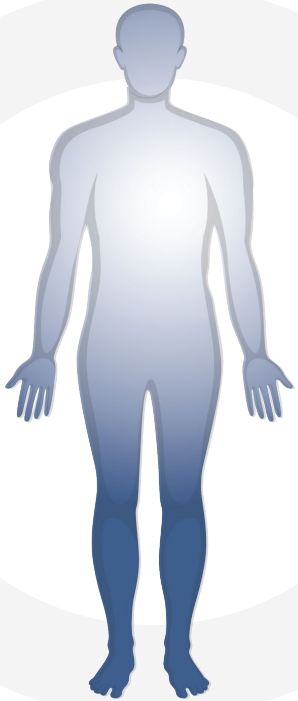
ARV-110 is showing early clinical benefit in highly refractory patients

Median prior therapies **5**

76% Patients treated with prior chemotherapy

Patients treated with both abiraterone and enzalutamide **82%**

84% Patients with non-AR mutations



Existing AR-directed therapies **expected to be ineffective**

High tumor heterogeneity suggests **low dependence on AR**

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ARDENT Phase 2 has initiated with a once daily, oral dose of 420 mg
Design informed by Phase 1 learnings

Promising antitumor activity
in heavily pre-treated patients with limited treatment options

PSA reduction
is associated with **plasma exposure**

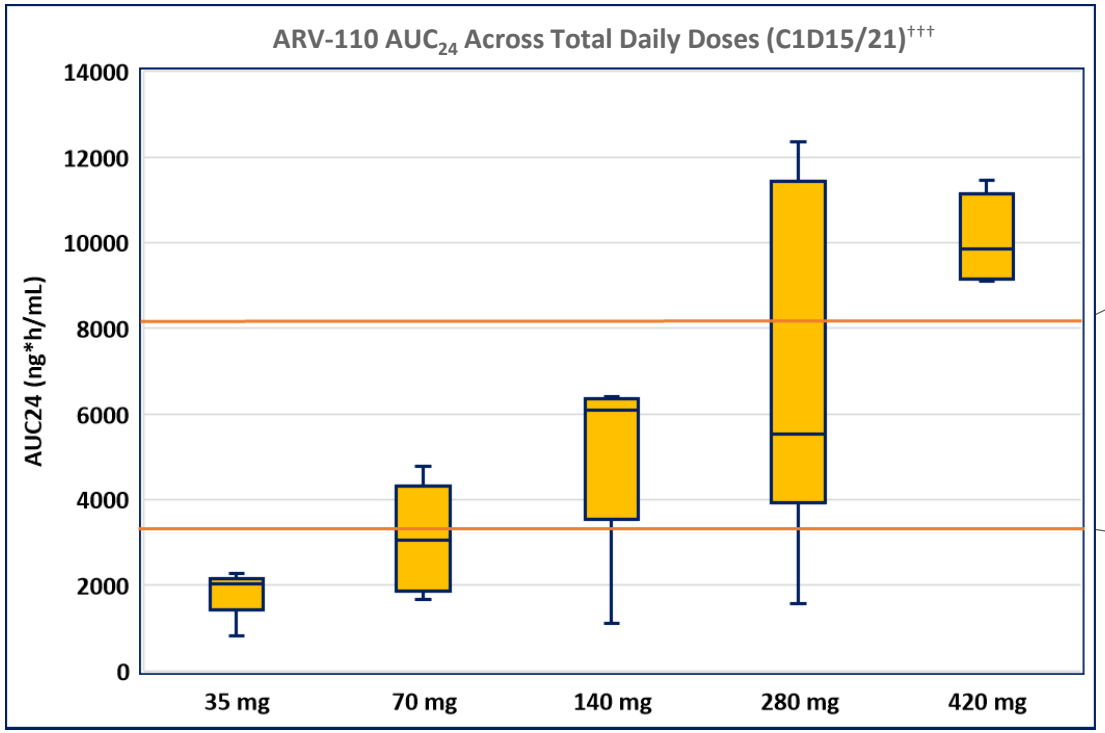
AR molecular profiling
identifies **a molecularly defined**, late line population that may **have greatest response to ARV-110**

Activity in wild-type AR patients supports **broader use**

ARV-110 is well tolerated†, allowing continued dose escalation up to current dose of 700 mg daily, and potentially **supporting use in earlier lines of therapy**

† Safety cut-off date: October 2, 2020

At 420 mg, exposures exceed the predicted efficacious threshold observed in a preclinical enzalutamide-resistant model



Predicted efficacious threshold based on an **enzalutamide-resistant prostate cancer** model ††

Predicted **minimum** efficacious threshold based on a standard prostate cancer model†

† The minimum preclinical efficacious threshold represents the AUC associated with tumor growth inhibition in standard VCAP models, †† This efficacious threshold represents the AUC associated with tumor growth inhibition in a preclinical enzalutamide-resistant VCaP model, ††† Includes both qd and bid dosing for the 420 mg total daily dose

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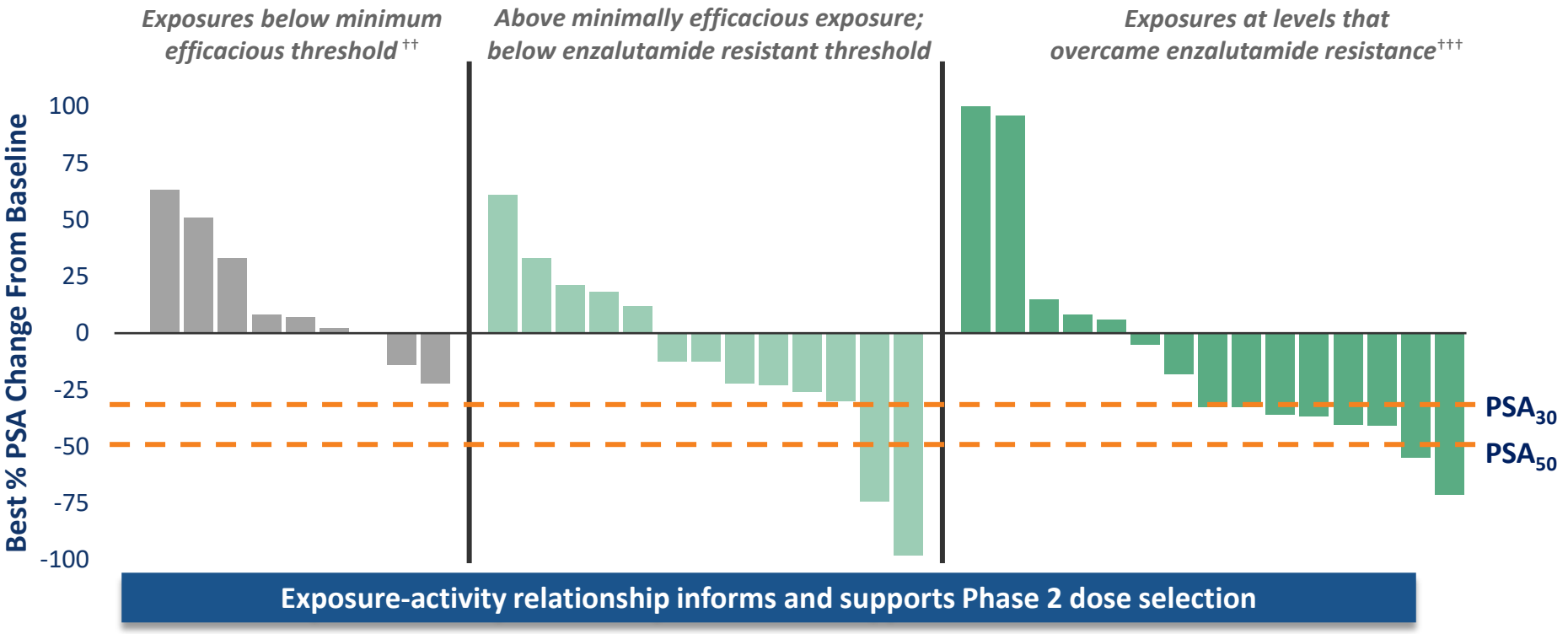
Increased ARV-110 clinical activity at higher exposures

Best PSA Change By Preclinical Efficacious Threshold (N=37)†



† Data as of 30-Nov-2020, †† Exposures in this range did not show anti-tumor activity, ††† Preclinical exposures in this range were sufficient to overcome enzalutamide resistance in preclinical models.

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We have identified ARV-110-sensitive populations despite significant tumor heterogeneity in our patient population

Genomic alterations are known to increase over time and with multiple treatments in mCRPC

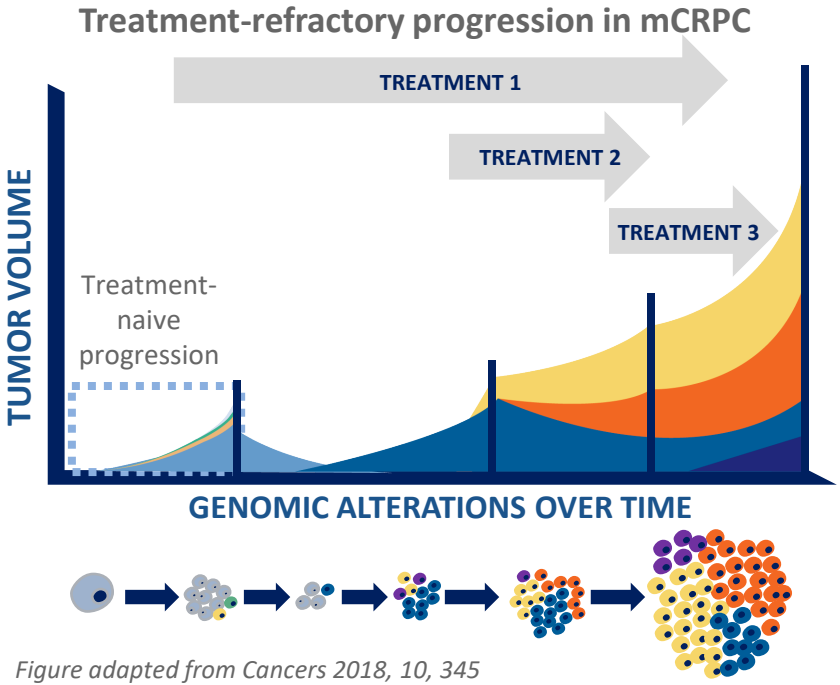


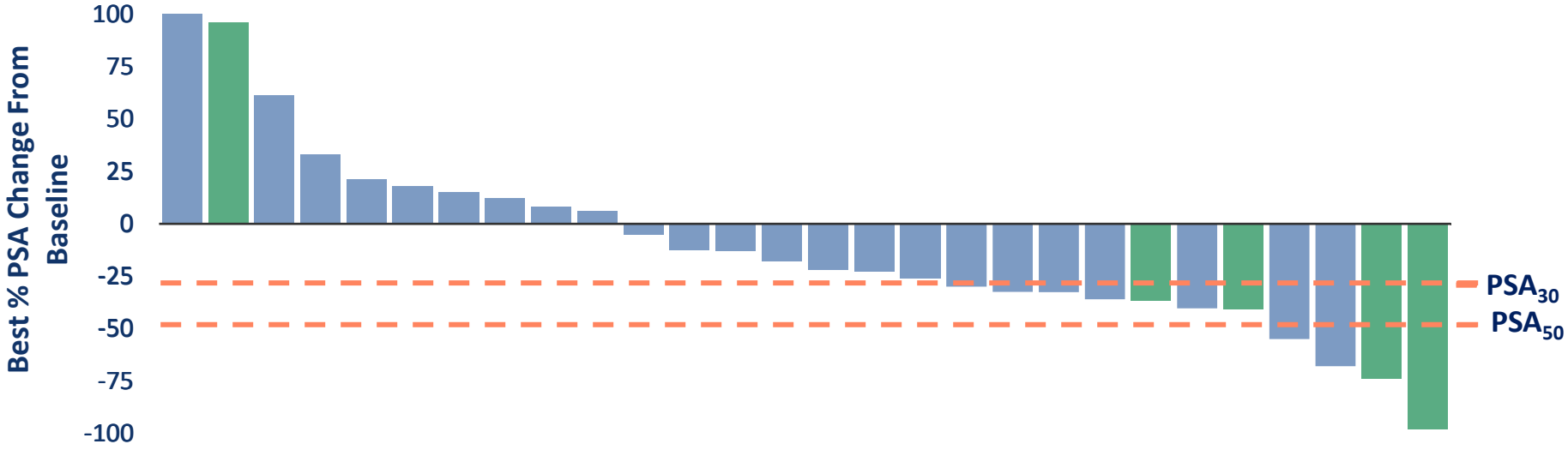
Figure adapted from Cancers 2018, 10, 345

- **Genetic context, an important determinant of response, is the basis for our Phase 2 patient selection strategy**
- The tumors of patients in our Phase 1 dose escalation are highly heterogeneous
  - 84% have non-AR mutations
  - Potential for high AR-independence
  - <10% PSA response expected
- In our studies, we are testing for mutations using 70- and now 324 gene-panels†

† Genetic profiling for most Phase 1 patients was done using the FoundationOne®Liquid test (70-gene panel), additional Phase 1 and Phase 2 patients: FoundationOne®Liquid CDx (324-gene panel).

In our late stage, genetically heterogeneous population, we have identified potential molecularly defined subgroups of patients sensitive to ARV-110

Best PSA Change In All Patients Above Minimum Exposure Threshold (N= 28) †††



AR Status	Amp	T878A, H875Y, L702H	Amp	WT	WT	Amp	Amp	WT	Amp	W742C L702H	WT	WT	WT	WT	WT	WT	WT	Amp	WT	WT	T878A, T878S, L702H	WT	T878A, F877L, V716M, L702H	WT	WT	T878A, H875Y	T878A, H875Y
AR-V7††			✚	✚	✚										✚				✚		✚	✚						
Other Genes Altered (n)	1	2	1	2	2	0	2†	1	2	4	3†	0	2	0	1	1†	0	2	1†	1	3	5†	0	6†	2	0	3	1

20/28 (71%) of patients have either T878/H875 or wild-type AR

Each column represents one patient. † Includes genes with multiple alterations, †† Epic Sciences, Genetic profiling: FoundationOne®Liquid (70-gene panel), ††† Data as of 30-Nov-2020.

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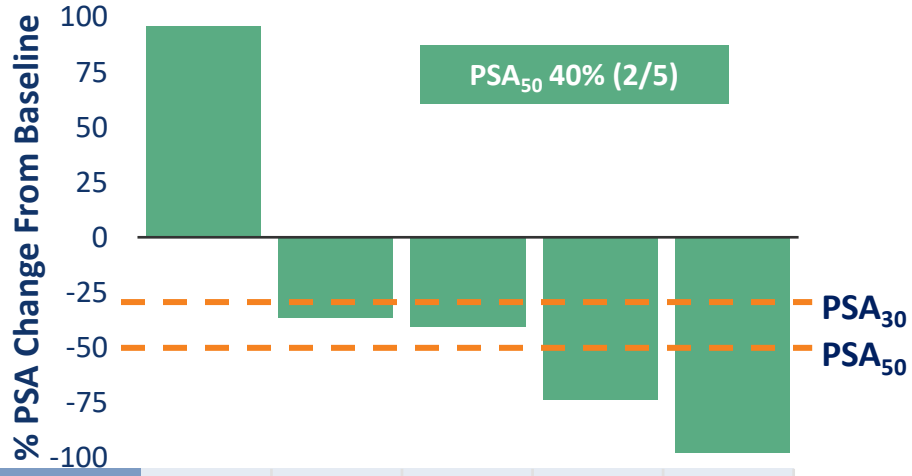
Four of five (80%) patients with T878/H875 mutations had PSA reductions, representing a potential accelerated approval population

Best PSA Change In Patients with AR T878/H875 mutations (N=5)††



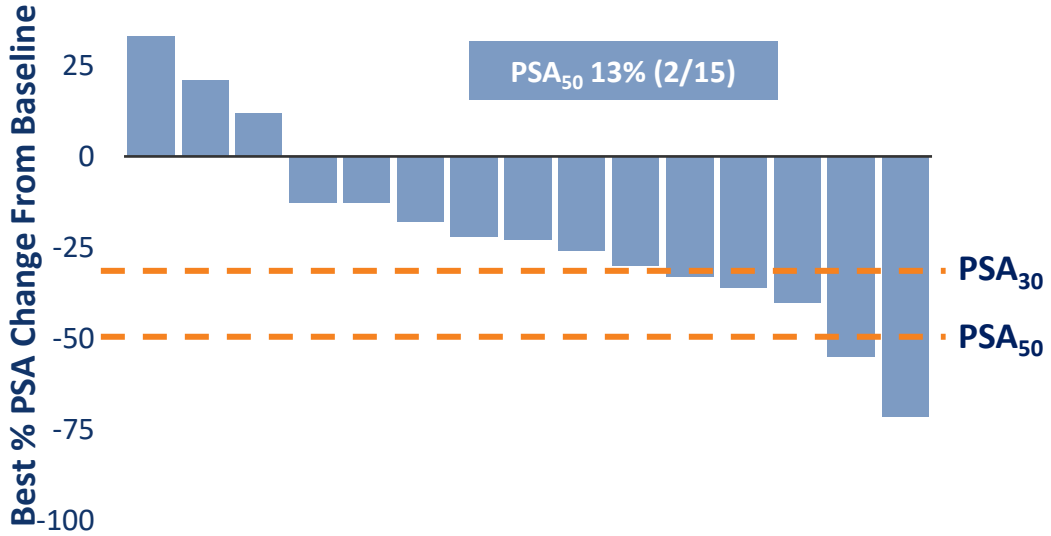
AR Status	T878A, H875Y, L702H	T878A, T878S, L702H	T878A, F877L, L702H, V716M	T878A, H875Y	T878A, H875Y
AR-V7†††		+			
Other Genes Altered (n)	2	5†	6†	3	1
Treatment Duration (months)	1.4→	1.8	6.2→	7.7	10.1

- Multiple AR mutations could be a "signature" for continued AR dependence
- PSA levels declined even in the presence of significant tumor heterogeneity, AR-V7, and L702H
- T878/H875 patients are a molecularly defined population for enrichment in our ongoing Phase 2 dose expansion, and represent a potential path to accelerated approval

Each column represents one patient. † Includes genes with multiple alterations, †† Includes all patients dosed above the minimum efficacious threshold and with T878/H875 AR (may include other forms of AR), ††† Epic Sciences, Genetic profiling: FoundationOne®Liquid (70-gene panel), →Patient remained on treatment as of November 30 2020

ARV-110 is also active in refractory mCRPC patients with tumors expressing wild-type AR

Best PSA Change In Patients with Wild-Type AR (N=15)††



AR Status	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT	WT
AR-V7†††	+	+					+					+			
Other Genes Altered (n)†	2	2	1	0	2	0	1	1†	0	2	1	3	0	2	0

Wild-type AR-containing tumors represent a broader population sensitive to ARV-110

Each column represents one patient. † Includes genes with multiple alterations, †† Includes all patients dosed above the minimum efficacious threshold and with wild type AR, ††† Epic Sciences, Genetic profiling: FoundationOne®Liquid (70-gene panel).

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Strong profile for ARDENT Phase 2 expansion trial at 420 mg, oral, once daily

Parameter	Phase 1 Results
Safety Data†	✓ (Well tolerated; no TRAEs Gr >2)
Dose Response and Exposure Threshold††	✓
Efficacy Data††	✓
Strong signal in molecularly defined patient populations	✓
High potential for patient benefit in earlier-line, more AR-dependent patients	✓

Opportunity to select a second dose in 2021

† Safety cut-off date: October 2, 2020
†† For patients with molecular profiling, PK and PSA data as of 30-Nov-2020.

ARVINAS

ARDENT will evaluate efficacy in both late-line, molecularly defined patients, and in a broader, early-line mCRPC population

Features of the ARDENT Phase 2 Design

- Enriches T878/H875 for exploration as a potential population for accelerated approval, and retains optionality for others
- Enrolls earlier, more AR-dependent populations
- Provides a subgroup for all screened patients

Patient Subgroup†	Tumor Characteristics
T878/H875	T878 and/or H875 AR mutated
Less-pretreated patients	Chemo-naïve, and progressed on abiraterone OR enzalutamide (*not both*)
Other AR degradable by ARV-110	AR wild type, amplified, and resistance-driving point mutations
AR not degradable by ARV-110	Tumors with L702H and AR-V7
Total N = ~100	

Potential registrational paths

1. **Late-line (3L), molecularly defined mCRPC**

Potential for accelerated approval

2. **Earlier-line (1L/2L) mCRPC**

Via confirmatory study

† Tumors are heterogeneous, so patients may fall into multiple subgroups for post-hoc analysis.

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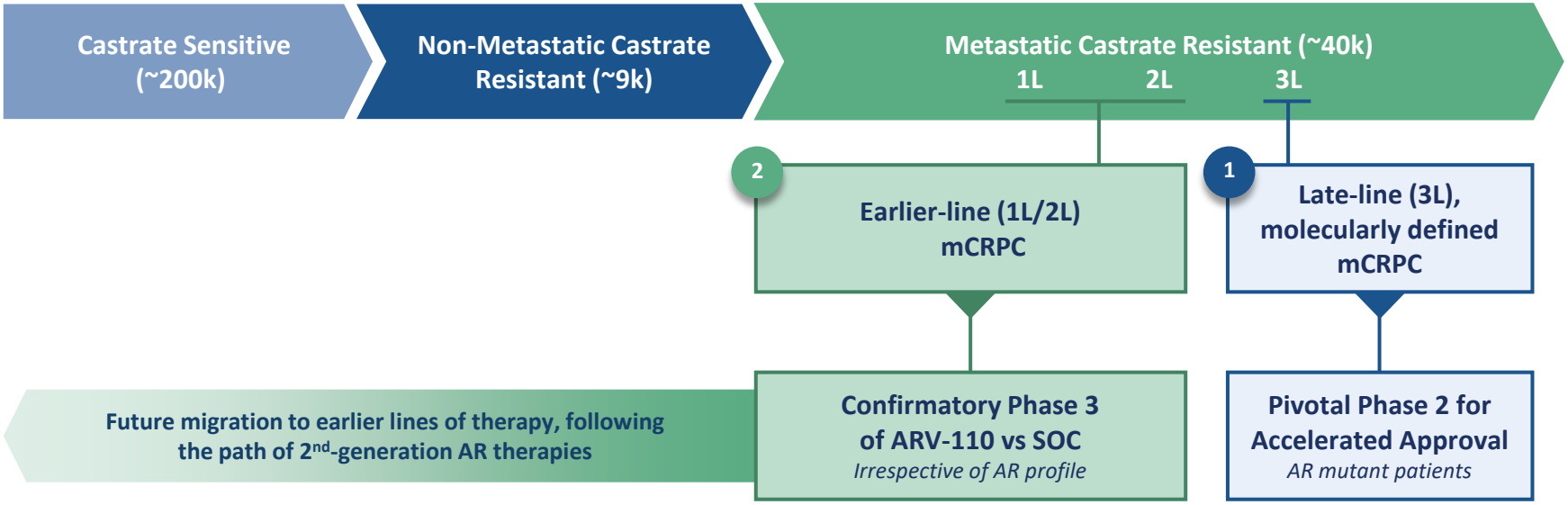
ARV-110's planned registrational path aligns with unmet need in mCRPC, and offers potential label expansion into earlier settings

Evolving Prostate Cancer US Treatment Paradigm (# of US patients†)



† SEER database

SOC, standard of care; mCRPC, metastatic castrate resistant prostate cancer

ARV-110: Potential to address unmet need across multiple stage of prostate cancer

Potential for Best-in-Class Profile

- Driving **tumor responses and PSA reductions** in a molecularly defined, late-line mCRPC population
- **Late-line activity** suggests **strong potential in CSPC**
- **Well tolerated**

Clear Development Path

- **Two potential registrational paths**
  - **Accelerated approval** in molecularly defined mCRPC
  - **Broader 1L/2L** mCRPC

Large Unmet Need and Opportunity

- High unmet need across all stages of prostate cancer
- Including CSPC, addressable patient population of >250K† per year in the US alone translates into a >$8B market opportunity

† US incidence from SEER Database
CSPC, castrate sensitive prostate cancer

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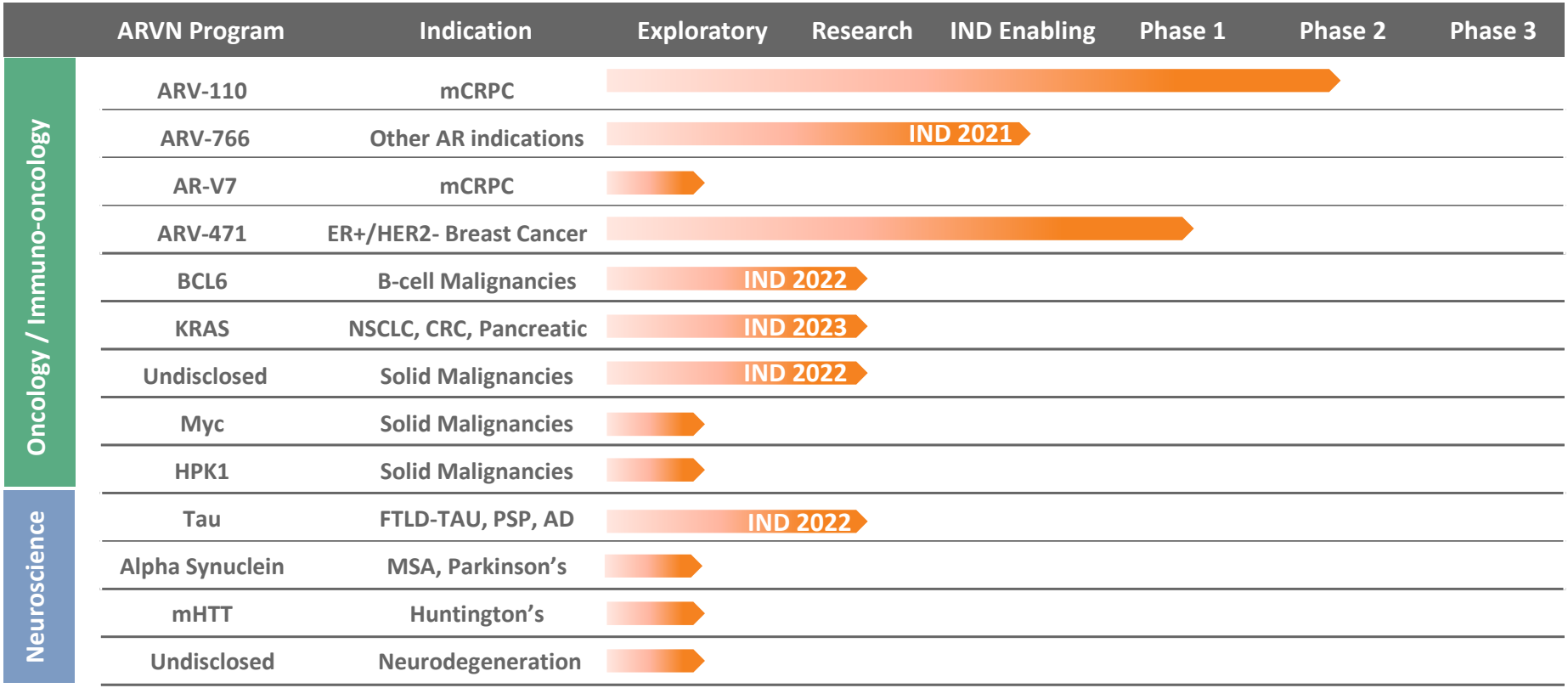
Conclusion

Arvinas' current pipeline encompasses a range of validated and undruggable targets in oncology, I-O, and neuroscience



Note: Pipeline is non-exhaustive and IND dates are anticipated.
mCRPC, metastatic castration-resistant prostate cancer; ER+/HER2-, estrogen receptor+/human epidermal growth factor receptor 2-; NSCLC, non-small-cell lung carcinoma; CRC, colorectal cancer; FTLD-tau, frontotemporal lobar degeneration-tau; PSP, progressive supranuclear palsy; MSA, multiple systems atrophy

ARV-110 and ARV-471 set up Arvinas for a remarkable 2021

Anticipated Milestones	2020 Q4	2021	2022
ARV-110 (AR PROTAC®)		• Complete Phase 1 data • ARDENT Phase 2 interim data • Initiation of combination study(s)	• Full ARDENT Phase 2 data • Combination study data
ARV-471 (ER PROTAC®)	• Initiation of combination study with CDK4/6i	• Complete Phase 1 data • Initiation of Phase 2 • CDK4/6i combination study data	• Interim Phase 2 data
ARV-766 (AR PROTAC®)		• Initiate Phase 1	• Phase 1 data • Initiate Phase 2
INDs		• ARV-766	• BCL6 • Tau • Undisclosed (oncology)

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We are well on our way to our 2024 vision

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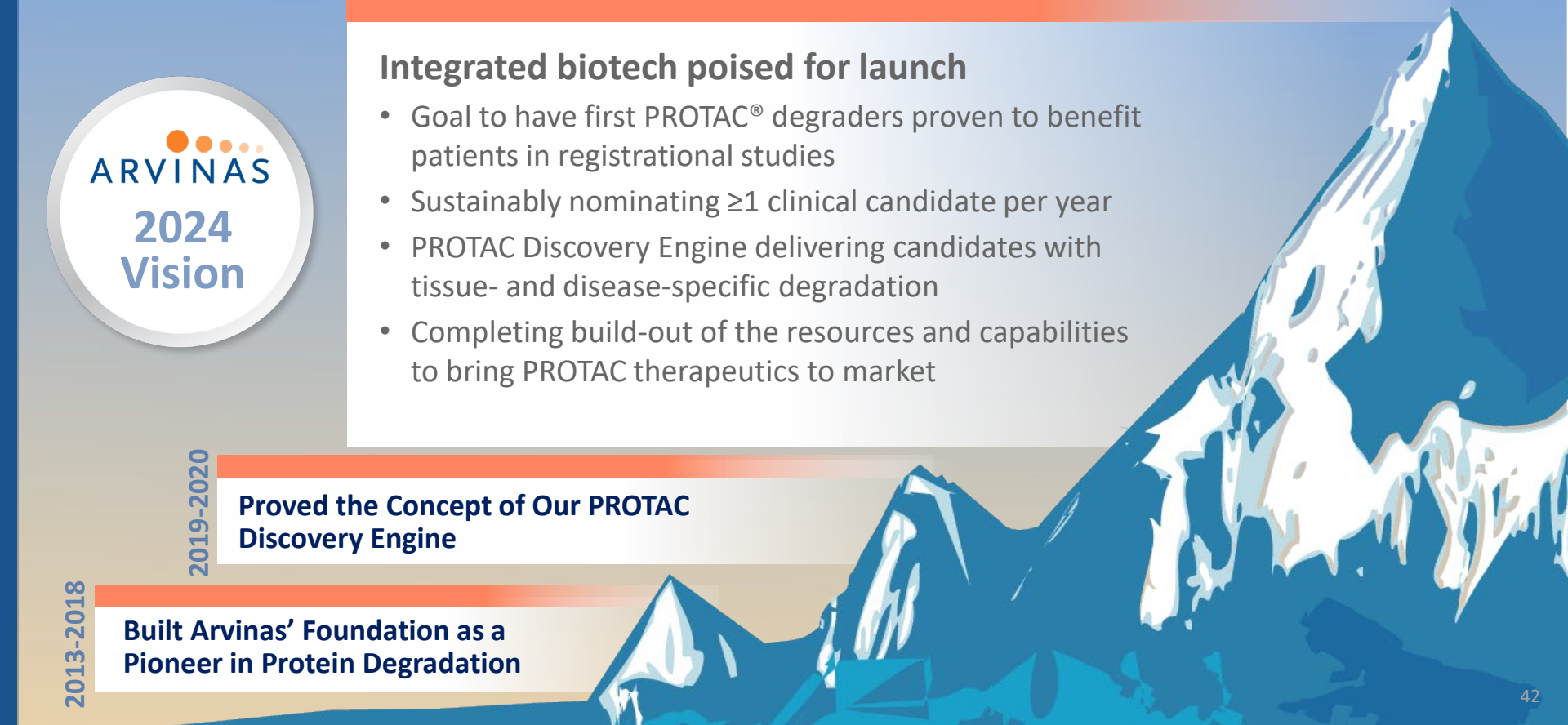
2024 Vision

Integrated biotech poised for launch

- Goal to have first PROTAC® degraders proven to benefit patients in registrational studies
- Sustainably nominating ≥1 clinical candidate per year
- PROTAC Discovery Engine delivering candidates with tissue- and disease-specific degradation
- Completing build-out of the resources and capabilities to bring PROTAC therapeutics to market



2019-2020

Proved the Concept of Our PROTAC Discovery Engine

2013-2018

Built Arvinas' Foundation as a Pioneer in Protein Degradation

Thank You