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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: PROteolysis TAargeting Chimera ("PROTAC") protein degraders having potential benefits that may provide advantages over other modalities in oncology and neurology; luxdegalutamide demonstrating signals of efficacy in late-line prostate cancer suggesting the potential for positive efficacy in earlier line-settings; Arvinas' goal to change treatment paradigms in neurology, immuno-oncology, hematology and new medicines; Arvinas' plans and expectations related to its clinical trials and preclinical studies and the timings associated therewith with respect to initiation of trials or studies and presentation of data; Arvinas' assets' intended differentiation from other therapies; Arvinas' potential receipt of milestone or other payments from existing partners, including Novartis Pharma AG and Rigel Pharmaceuticals, Inc.; Arvinas' capitalization, and having cash runway into the second half of 2028; the potential for future partnerships to enhance the value of Arvinas' pipeline; the potential market opportunity for any of Arvinas' product candidates; the potential for a PROTAC B-cell lymphoma 6 ("BCL6") degrader to address substantial unmet needs for patients with non-Hodgkin Lymphoma ("NHL"); the potential for ARV-393 to become an attractive combination partner for development of novel treatment approaches for NHL, including all oral or chemotherapy-free options; dose escalation or de-escalation of ARV-393; that a PROTAC expanded polyglutamine androgen receptor degrader may eliminate the root cause of disease for spinal bulbar muscular atrophy; the potential for ARV-6723, an oral hematopoietic progenitor kinase 1 ("HPK1") degrader, to offer sustained anti-tumor immune response as a single agent or in combination with standard of care therapies, and the potential to result in improved clinical benefits over standard of care therapies across a wide range of cancers; that HPK1 degradation may lead to an increase in immune function and immune attack, counter activation of T cell exhaustion, block suppressive tumor microenvironment and reprogram the tumor microenvironment to unleash immune response; the suggestion from preclinical work that an HPK1 degrader could work after anti-programmed death-1 ("αPD-1") progression, in combination with αPD-1, and ahead of αPD-1; Arvinas' novel PROTAC approach having potential to differentiate from existing and emerging modalities in, and potentially revolutionize the treatment of, neurodegenerative diseases; PROTAC-induced leucine-rich repeat kinase 2 ("LRRK2") degradation having the potential to differentiate from kinase inhibition; PROTAC-induced LRRK2 degradation as a potential treatment for progressive supranuclear palsy ("PSP"), Parkinson's disease and related diseases; Arvinas' plans with respect to initiation of clinical trials of ARV-102 in patients with PSP, pending regulatory approval, and the timing thereof; and Arvinas' plans, anticipated milestones, timing and potential impacts of such milestones in connection with ARV-393, ARV-6723, ARV-027 and ARV-102, including statements regarding sharing of data, types of data, initiation and enrollment of clinical trials, sharing or presenting data and any forums for doing so. The words "anticipate," "believe," "estimate," "expect," "intend," "may," "might," "plan," "predict," "project," "target," "potential," "goal," "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. Arvinas may not actually achieve the plans, intentions or expectations disclosed in its forward-looking statements, and you should not place undue reliance on these forward-looking statements.

Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements Arvinas makes as a result of various risks and uncertainties, including but not limited to: whether Arvinas will be able to successfully conduct and complete development for its product candidates, including ARV-393, ARV-027, ARV-6723 and ARV-102, including whether Arvinas initiates and completes clinical trials for its product candidates and receives results from its clinical trials on expected timelines, or at all; whether Arvinas will be able to successfully conduct and complete development for its other preclinical or clinical candidates, including whether Arvinas initiates and completes preclinical studies or clinical trials and receives results from such studies or trials on expected timelines, or at all; the potential therapeutic benefits or profile of any of Arvinas' product or preclinical candidates; the uncertainties inherent in research and development, including preclinical study or clinical trial results; risks related to the potential market opportunity for any of Arvinas' product candidates; risks related to obtaining marketing approval for any product candidates; Rigel's performance of its obligations under the license agreement with Arvinas, Pfizer and Rigel; the potential demand and market potential and acceptance of any product candidate, including estimates regarding the potential market opportunity; the competitive landscape for any other product candidate; the risk that any regulatory approval may be subject to significant limitations on use or subject to withdrawal or other adverse actions by the applicable regulatory authority; regulatory actions or delays or government regulation generally; Arvinas' ability to protect its intellectual property portfolio; risks associated with Arvinas' reliance on third parties; risks associated with Arvinas' collaboration and license agreements; whether Arvinas will be able to raise capital when needed; whether Arvinas' cash and cash equivalent resources will be sufficient to fund its foreseeable and unforeseeable operating expenses and capital expenditure requirements; and other important factors, any of which could cause Arvinas' actual results to differ from those contained in the forward-looking statements, discussed in the "Risk Factors" sections of Arvinas' quarterly and annual reports on file with the U.S. Securities and Exchange Commission. The forward-looking statements contained in this presentation reflect Arvinas' current views as of the date of this presentation with respect to future events, and Arvinas assumes 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 Arvinas' views as of any date subsequent to the date of this presentation.

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ARVINAS



IGNITING A

TRANSFORMATIVE CHANGE

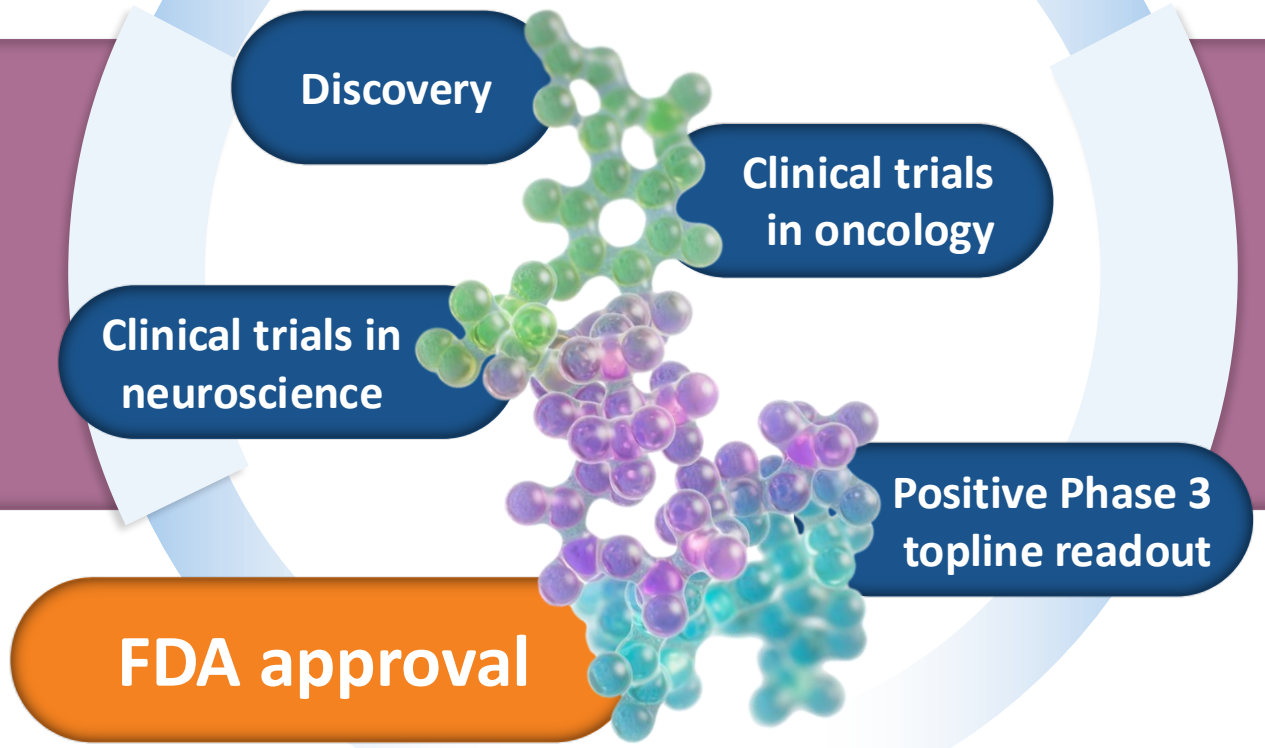
in the fight for patients with cancer and neurodegenerative diseases

Our experienced and talented team is advancing a new therapeutic modality for patients

Promising preclinical results are translating into the clinic

8 programs entered clinical trials in 6 years[a], targeting significant unmet needs for patients

History of FIRSTS with our novel PROTAC therapeutic modality:



Strong, experienced leadership team

Expertise from bench to commercialization

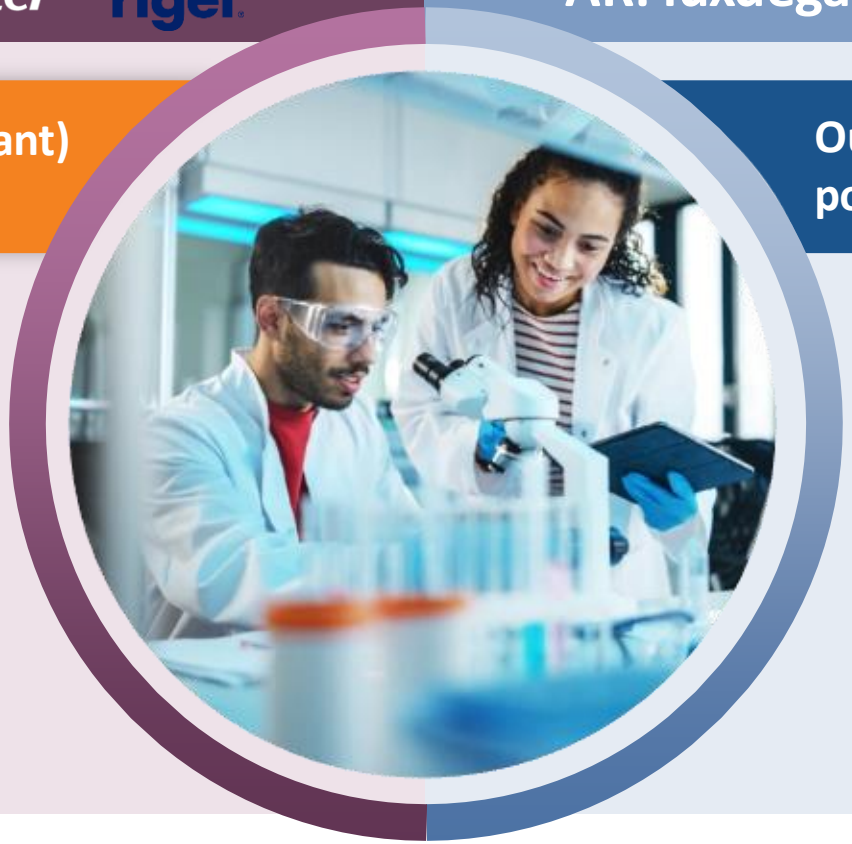
PROTAC:, proteolysis targeting chimera
a. vepdegestrant, bavdegalutamide, luxdegalutamide (JSB462), ARV-102, ARV-393, ARV-806, ARV-027, ARV-6723

ER and AR degraders highlight Arvinas' ability to develop valuable PROTAC degraders

ER: VEPPANU[a]

FDA approved VEPPANU™ (vepdegestrant) on May 1, 2026

- FDA approval marked a defining milestone, delivering a new treatment option for patients while validating protein degradation mechanism
- Out-licensed to Rigel Pharmaceuticals, Inc. in May 2026; VEPPANU became commercially available from Rigel in August 2026



AR: luxdegalutamide

Out-licensed to Novartis in 2024 with potential for more than $1B in total value

- Demonstrated signals of efficacy in late-line prostate cancer suggests potential for positive efficacy in earlier-line settings
- Novartis is currently evaluating luxdegalutamide in three Phase 2 combination trials in mCRPC and mHSPC

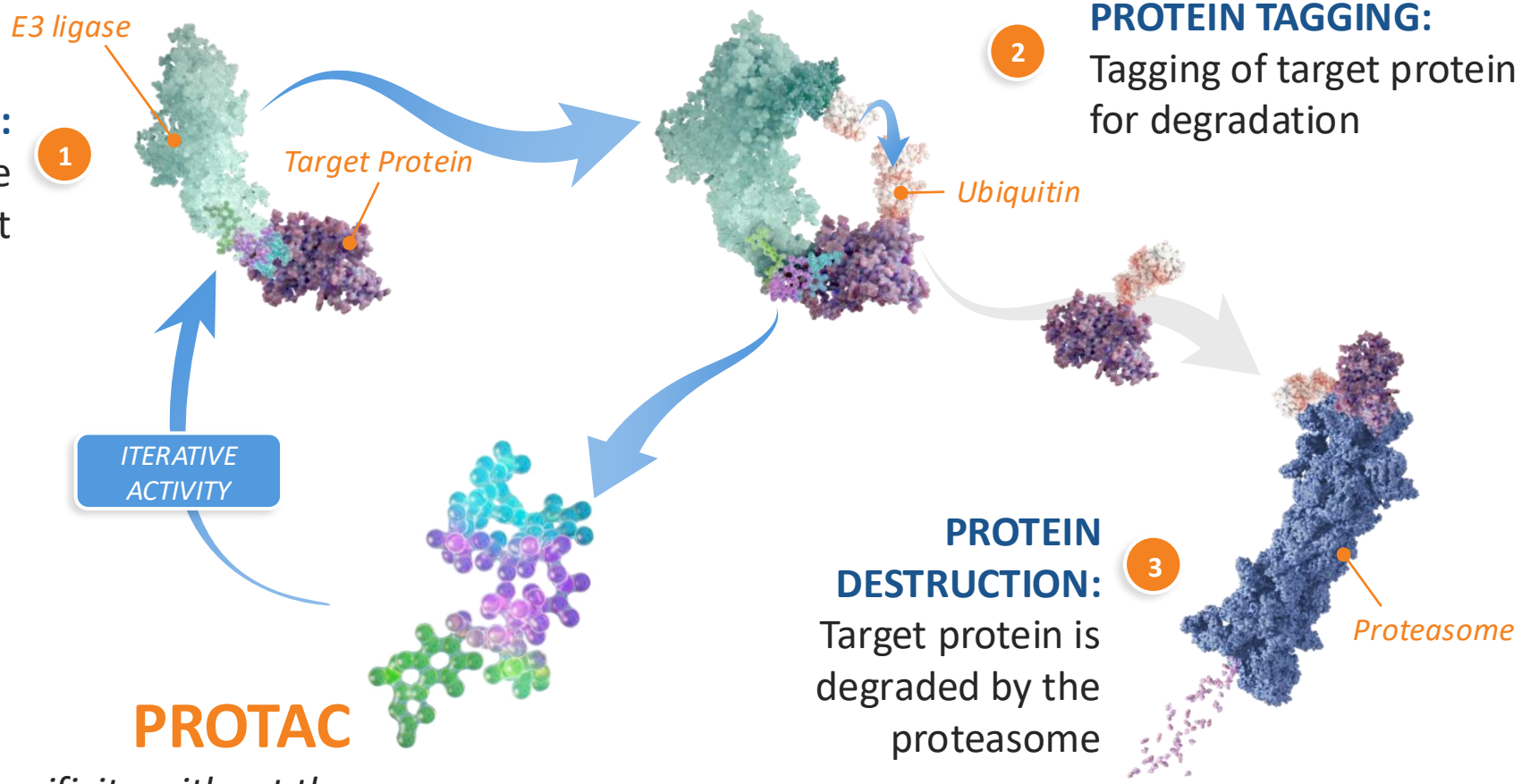
Strategic clinical-stage partnerships with Pfizer and Novartis have resulted in more than $800M in non-dilutive cash

AR, androgen receptor; ER, estrogen receptor; *ESR1*, estrogen receptor 1 gene; ER+/HER2-, estrogen receptor-positive/human epidermal growth factor receptor 2-negative; FDA, Food and Drug Administration; mCRC, metastatic castrate resistant prostate cancer; mHSPC, metastatic hormone-sensitive prostate cancer.
a. In 2021, Arvinas entered into a collaboration agreement with Pfizer to co-develop and co-commercialize vepdegestrant

PROTAC degraders harness the body's natural machinery to degrade, not simply inhibit, disease-causing proteins

COMPLEX FORMATION:
Recognizing disease causing protein of interest

1



2

PROTEIN TAGGING:
Tagging of target protein for degradation

3

PROTEIN DESTRUCTION:
Target protein is degraded by the proteasome

PROTAC

High specificity without the requirement for strong binding

PROTAC degraders have potential benefits that may provide advantages over other modalities in oncology and neurology

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BENEFITS IN

ONCOLOGY

- Ability to overcome evolving resistance mechanisms
- Targeting of classically “undruggable” proteins
- Therapies with potential to improve upon existing treatment options

By 2040, ~**30M new cancer cases per year** worldwide, leading to nearly **15M cancer-related death per year**[1]

BENEFITS OF

PROTAC

PROTEIN DEGRADERS

- Elimination (rather than inhibition) of disease-causing proteins
- Interruption of scaffolding functions of target proteins
- Iterative (catalytic) activity
- Oral delivery and broad tissue distribution
- Mutant and/or wild-type specificity
- Efficient manufacturing and routes of synthesis (versus biologics and cell therapies)

BENEFITS IN

NEUROLOGY

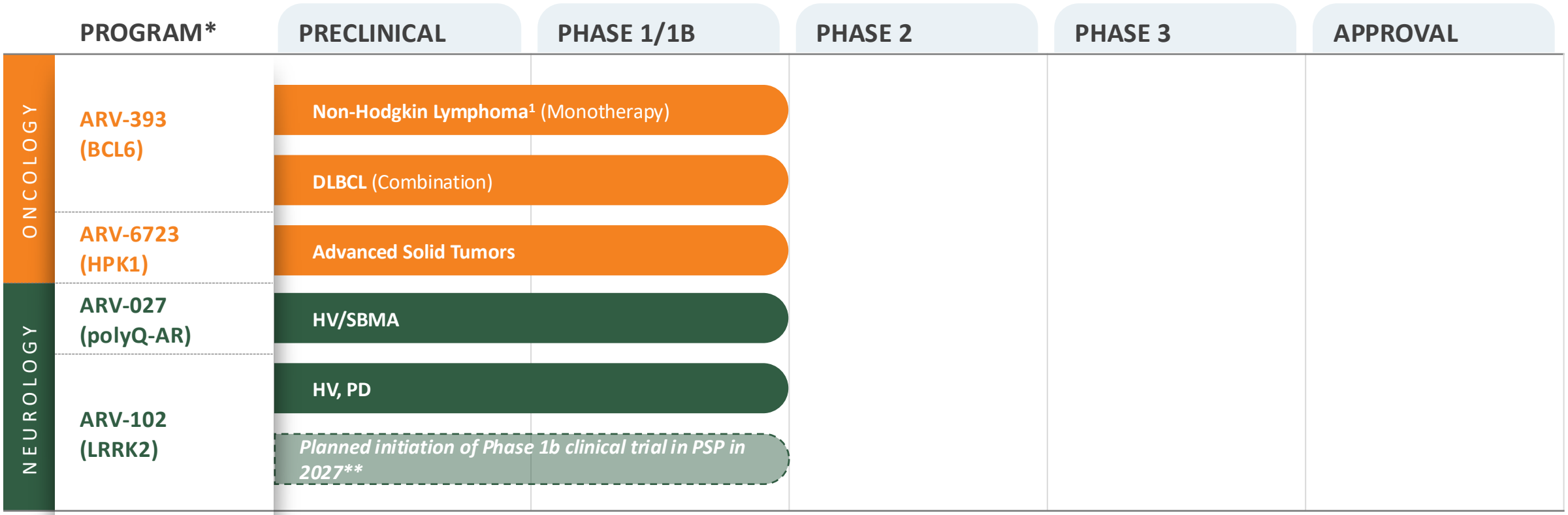
- Blood-brain barrier penetration
- Oral administration, avoiding IM, IV, or intrathecal dosing
- Biodistribution to deep-brain regions

By 2040, neurodegenerative diseases are estimated to be **the #2 leading cause of death** in developed countries[2]

IM, intramuscular; IV, intravenous

1. Global Cancer Observatory: https://gco.iarc.fr/tomorrow/en/dataviz/isotype?years=2040; accessed 10/14/26. 2. Gammon, K. Neurodegenerative disease: Brain windfall. Nature 515, 299–300 (2014).

Arvinas' pipeline includes differentiated PROTAC degraders in neurology and oncology



Out-licensed Programs

NOVARTIS — Luxdegalutamide (ARV-766, JSB462) *Phase 2*

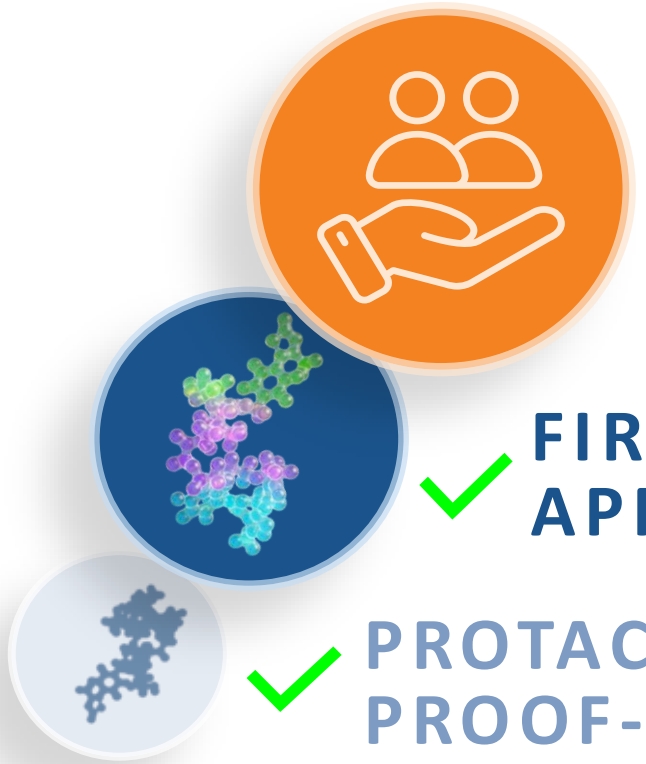
rigel — VEPPANU™ (vepdegestrant) *FDA Approved*

AR, androgen receptor; BCL6, B-cell lymphoma 6; CTA, clinical trial application; DLBCL, diffuse large B-cell lymphoma; HPK1, hematopoietic progenitor kinase 1; HV, healthy volunteers; LRRK2, leucine-rich repeat kinase 2; PD, Parkinson's disease; polyQ, expanded polyglutamine; PSP, progressive supranuclear palsy; SBMA, spinal-bulbar muscular atrophy.

1. Includes relapsed/refractory angioimmunoblastic T-cell lymphoma (AITL) and relapsed/refractory mature B cell NHL.

* These agents, other than VEPPANU which has been approved by the FDA, are currently under investigation and their safety and effectiveness for these investigational uses have not been established. ** Pending CTA submission and regulatory approval.

Our goal is to change treatment paradigms



GOALS TO CHANGE THE TREATMENT PARADIGM

✓ FIRST PROTAC APPROVAL

✓ PROTAC PROOF-OF-CONCEPT

Neurology
Disease modifying therapies in neurology

Immuno-Oncology
Engage the power of the immune system to establish new expectations

Hematology
Target transcriptions factor with an all oral or chemo-free options for patients

New Modalities
Moving degrader technology beyond traditional PROTACs

Committed to bringing valuable treatments for patients and creating value for shareholders

ARVINAS

Multiple Phase 1 PROTAC Programs with Near-Term Milestones

- Targeting BCL6 for hematological malignancies, HPK1 for solid tumors, polyQ-AR for spinal and bulbar muscular atrophy and LRRK2 for neurodegenerative diseases
- Delivering innovative assets intended to differentiate from other therapies
- Committed to investing in highest value drivers across the pipeline

Pivotal Proof of Concept with Vepdegestrant

- First-and-only FDA-approved PROTAC, a type of heterobifunctional protein degrader
- Out licensed to Rigel Pharmaceuticals for commercialization and potential for future regulatory/commercial milestones and tiered royalites on net sales

Strong Capitalization

- Cash runway into 2H 2028*
- Multiple value-inflecting milestones ahead, with potential to receive milestone payments from existing partners, including NOVARTIS for luxdegalutamide
- Potential for future partnerships to enhance the value of Arvinas' pipeline

BCL6, B-cell lymphoma 6; HPK1, hematopoietic progenitor kinase 1; LRRK2, leucine-rich repeat kinase 2; polyQ-AR, polyglutamine androgen receptor

*Based on cash, cash equivalents, and marketable securities position as of September 30, 2026



CLINICAL PROGRAMS: Hematology-Oncology

ARV-393

PROTAC BCL6 degrader

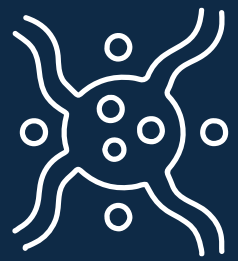
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ARV-393 is an investigational compound. Its safety and effectiveness have not been established.

A PROTAC BCL6 degrader has the potential to address substantial unmet needs for patients with non-Hodgkin lymphoma (NHL)

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Key Unmet Needs for Patients with Non-Hodgkin Lymphomas



Novel mechanisms with potential to become **safe and effective oral alternatives** to chemotherapy or immuno-chemotherapy SOC regimens

Additional options for patients with relapsed or high-risk disease, and particularly older adults

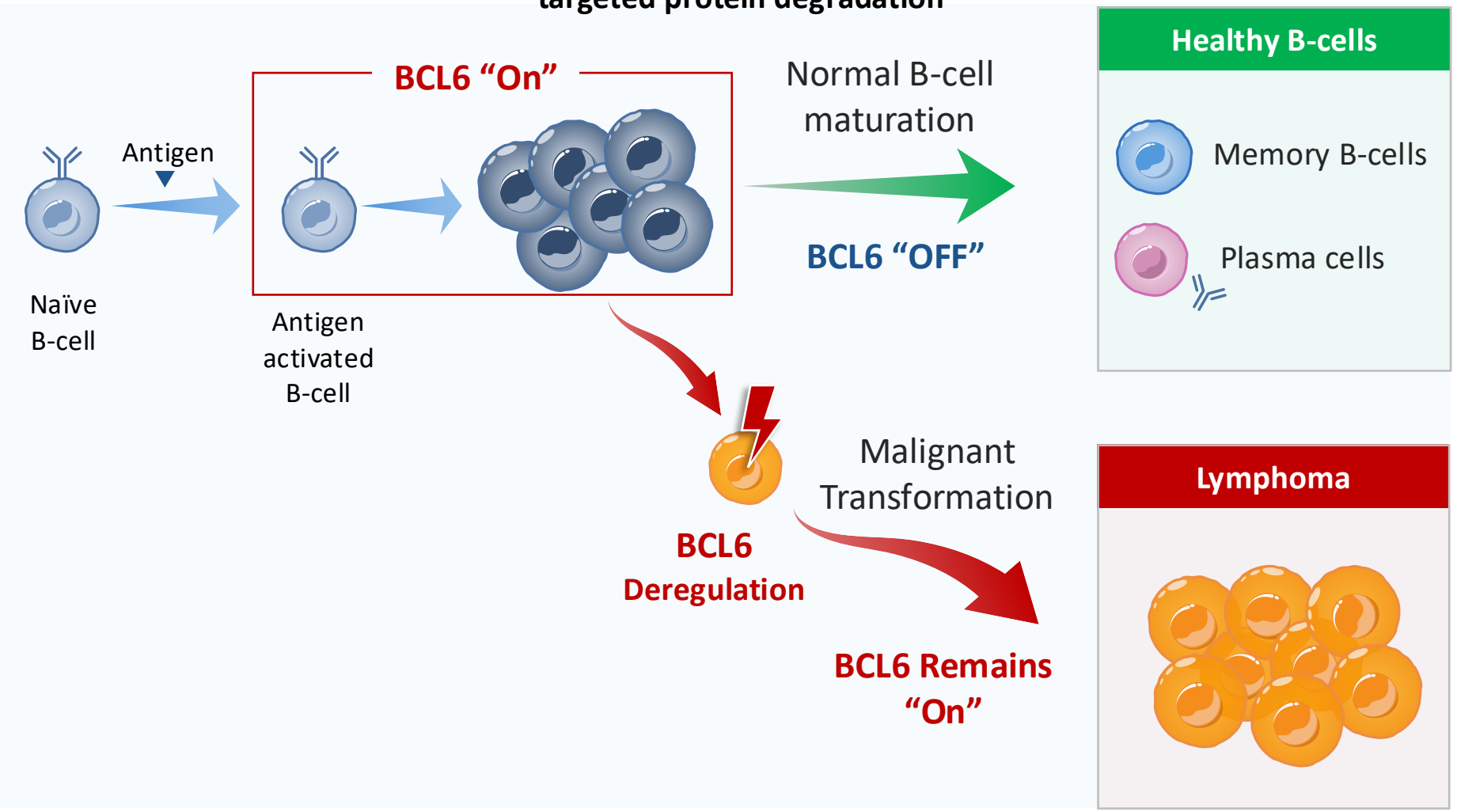
Large B-Cell Lymphoma (LBCL)	• Although a high proportion of patients achieve complete remission, therapy resistance or relapse still occurs in 30-40% of patients, thus evidencing a need for new therapeutic options[1] • Deregulation of BCL6 expression and/or functions are common in B-cell lymphomas[2]
Follicular Lymphoma (FL)	• Lack of effective options for patients who experience rapid disease progression within 2 years of initial therapy (POD24) • In ~15% of patients, indolent FL transforms into clinically aggressive lymphoma with rapid progression of disease and poor prognosis[3] • BCL6 mutation is associated with the transformation of FL
Other Subtypes of NHL	• T-cell lymphomas dependent on BCL6 (family of nodal T-follicular helper cell Lymphomas, nTFHL[4]) • nTFHL-AI (nTFHL, angioimmunoblastic type[4]), also known as AITL, is a rare and aggressive disease with no dedicated approved therapies and high levels of BCL6 expression[5]

AITL, angioimmunoblastic T-cell lymphoma; BCL6, B-cell lymphoma 6; FL, Follicular Lymphoma; LBCL, Large B-Cell Lymphoma; NHL, non-Hodgkin lymphoma; nTFHL, nodal T-Follicular Helper Cell Lymphoma; POD24, progression of disease within 24 months of starting initial active treatment.

1. Sehn and Salles NEJM 2021, 384(9):842. 2. Pasqualucci et al., Blood 2003;101 (8). 3. Conconi Br J Haematol 2012, 157(2):188-96. 4.De Level Die Pathologie 2023. 5. Nishizawa et al., Int J Hematol, 2017, 105(4):465.

Uncontrolled activation of BCL6 is implicated in development of B-cell lymphomas

BCL6 is a nuclear transcription factor and was long considered "undruggable" until discovery of targeted protein degradation



The importance of BCL6 in lymphoma

- **Master Regulator –** BCL6 represses genes that control cell proliferation, survival, and apoptosis during B-cell maturation[1,2]
- **Disrupts B-Cell Function –** Deregulated BCL6 alters cell signaling and cycle control, preventing proper B-cell differentiation[1,3]
- **Drives Malignant Transformation –** Abnormal BCL6 activity enables B-cells to evade regulatory mechanisms, leading to lymphoma[1,3]

BCL6, B-cell lymphoma 6

1. Basso and Dalla-Favera, Nat Rev Immunol 2015;15:172–184; 2. Basso and Dalla-Favera, Immunol Rev 2012;247:172–183; 3. Mlynarczyk et al., Immunol Rev 2019;288:214–239.

ARV-393 is an investigational oral PROTAC degrader that degrades BCL6, a classic “undruggable” protein

ARV-393



Potent, orally bioavailable PROTAC small molecule degrader of BCL6[1]

Degrades BCL6, a target that has long been considered “undruggable”

Differentiated preclinical profile. ARV-393 **potently and rapidly degrades** BCL6 protein and has iterative activity, which is critical to overcoming BCL6’s rapid resynthesis rate and sustaining antitumor activity

Demonstrated significant anti-tumor single-agent activity[1] and broad combinability with complete tumor regressions in combination with SOC biologics and investigational small molecule agents in numerous preclinical *in vivo* models of NHL[2], with potential to become an **attractive combination partner** for development of novel treatment approaches for NHL including **all oral or chemotherapy-free options**

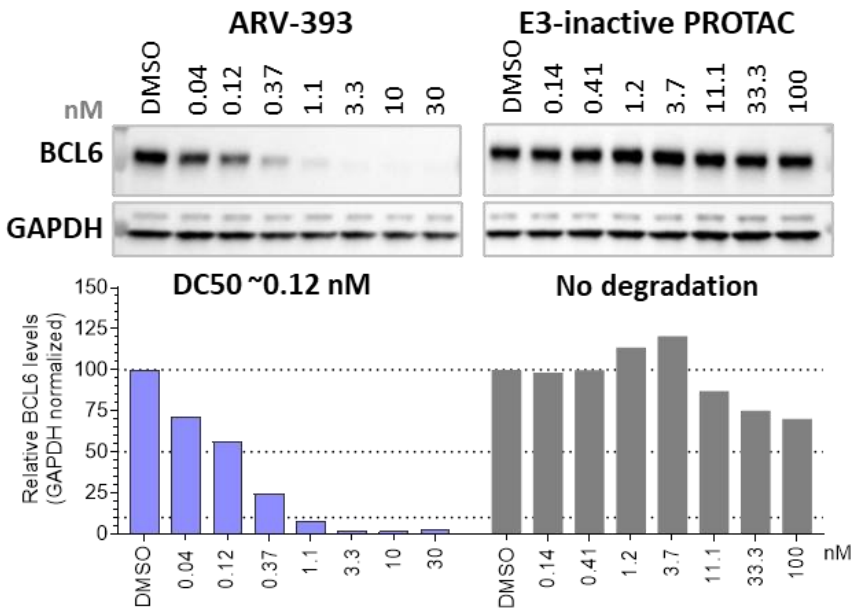
Phase 1 dose escalation trial of ARV-393 as monotherapy for R/R NHL, and in combination with glofitamab for R/R DLBCL, is currently enrolling patients (ClinicalTrials.gov ID: NCT06393738)

BCL6, B cell lymphoma 6; DLBCL, diffuse large b-cell lymphoma; NHL, non-Hodgkin lymphoma; R/R, relapsed/refractory; SOC, standard of care

1. Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain. 2. Van Acker et al., American Association for Cancer Research (AACR) Poster 1655/15. April 2025, Chicago.

ARV-393 potently degrades BCL6 in human lymphoma cell lines

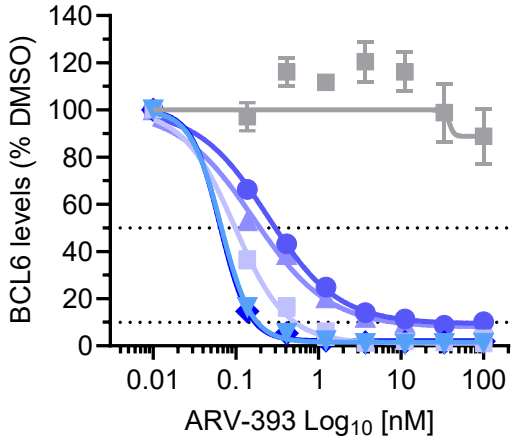
ARV-393, but not its E3-inactive analogue, robustly degrades BCL6 in the OCI-Ly1 model of DLBCL



Semi-quantitative western blot of BCL6 degradation by ARV-393

The E3-inactive analogue of ARV-393 cannot engage the E3 ligase and does not degrade BCL6, confirming ARV-393 degradation is mediated by PROTAC mechanism

ARV-393 exhibits picomolar degradation potency in quantitative immunocapture assay in multiple DLBCL cell lines



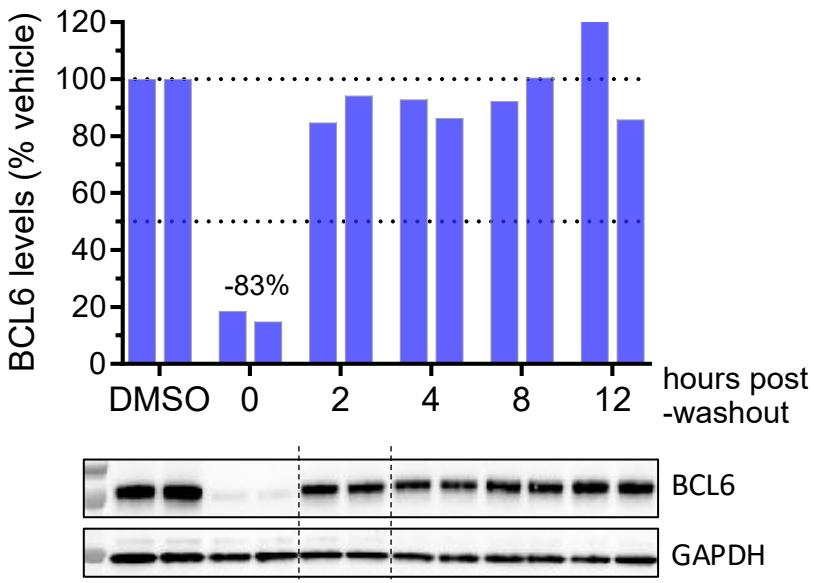
	DC_{50} [nM]	D_{max}
OCI-Ly1	0.07	99%
Farage	0.06	98%
SU-DHL-4	0.33	91%
SU-DHL-6	0.21	92%
OCI-Ly7	0.10	99%
OCI-Ly1 (E3-inactive ARV-393 analogue)	-	-

BCL6, B cell lymphoma 6; DLBCL, diffuse large b-cell lymphoma

Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.

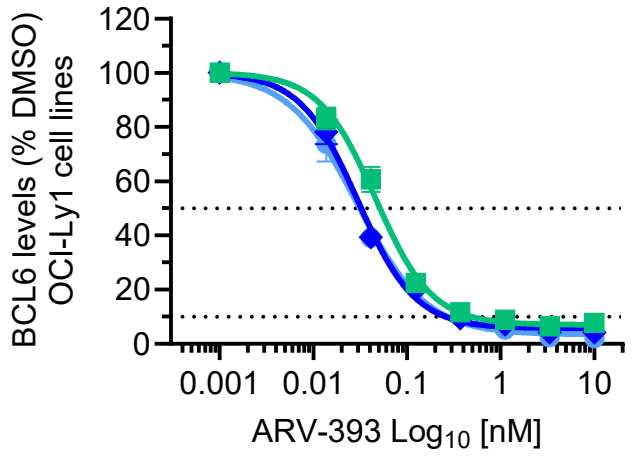
Preclinically, ARV-393 rapidly degrades BCL6 and overcomes its high resynthesis rate that drives tumor cell growth

BCL6 protein is resynthesized rapidly – nearly back to baseline levels at 2 hours post-ARV-393 washout



OCI-Ly1 cells were treated with 1.5 nM ARV-393 for 4 hours. Duplicate samples are shown following ARV-393 washout and addition of cereblon ligand (to block residual ARV-393)

ARV-393 rapidly degrades >90% of BCL6 within 2 hours - degrading BCL6 faster than the cell can resynthesize it



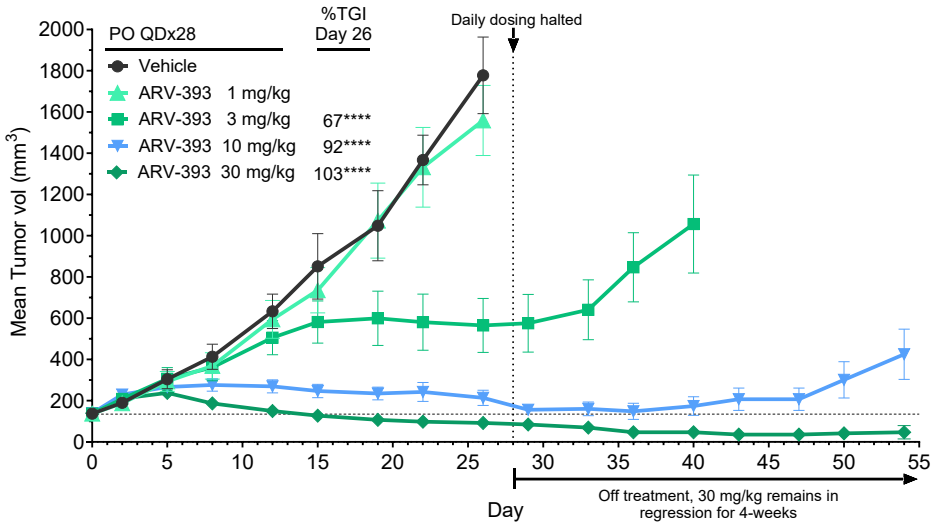
	DC_{50} [nM]	D_{max}
2 hours	0.05	93%
4 hours	0.03	94%
24 hours	0.03	97%

BCL6, B cell lymphoma 6

Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.

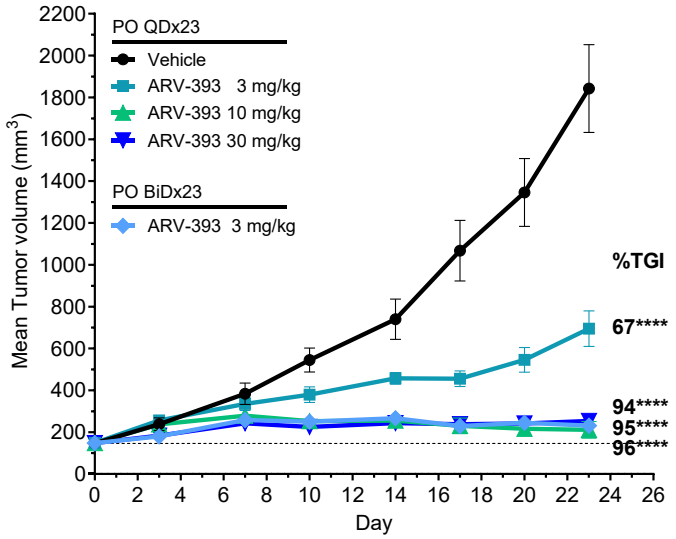
ARV-393 induces dose-dependent and sustained tumor growth inhibition and regressions *in vivo*

ARV-393 induces dose-dependent and sustained TGI in OCI-Ly1 DLBCL CDX model



- Tumor regression at 30 mg/kg dosed once per day (QD)
- No adverse impact on animal health/body weight (body weight not shown)

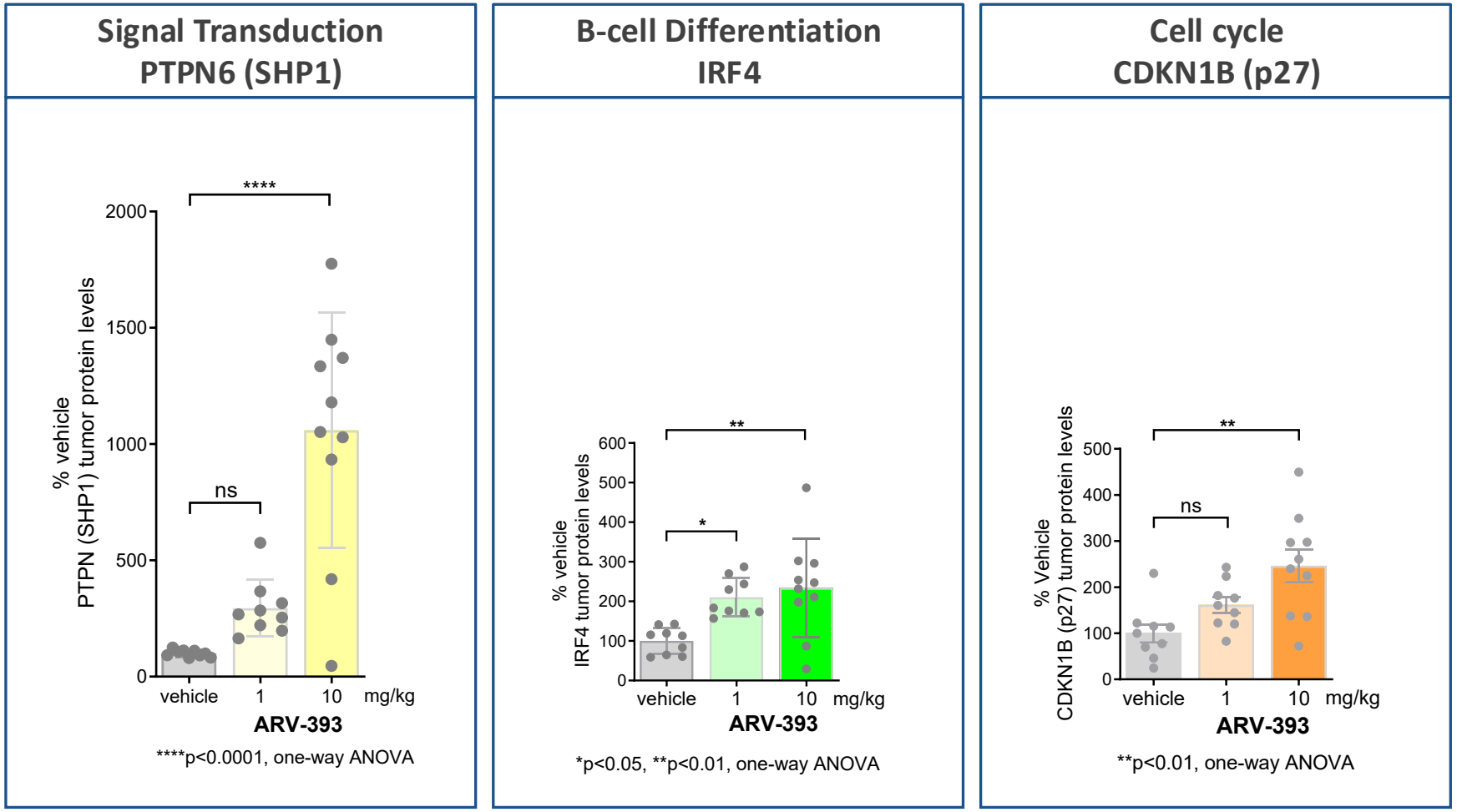
ARV-393 demonstrated >90% TGI



- TGI assessment of different dosing regimens of ARV-393 in the OCI-Ly1 CDX model demonstrate tumor growth inhibition >90% after 22 days at 10 and 30 mg/kg dosing

BID, twice daily ; CDX, cell line derived xenograft model; DLBCL, Diffuse Large B-Cell Lymphoma; TGI, Tumor Growth Inhibition; QD, once daily.
Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.
Statistical analyses were performed using two-way ANOVA.
****p<0.0001.

Preclinically, BCL6 degradation by ARV-393 drives antitumor activity by increasing gene expression normally repressed by BCL6



OCI-Ly1 CDX tumor lysates from mice dosed twice daily at 1 and 10 mg/kg

- **BCL6 Repression** – BCL6 is a transcriptional repressor and expression of PTPN6, IRF4, and CDKN1B are repressed by it
- **Pathway Activation** – Treatment with ARV-393 increased the expression of PTPN6, IRF4, and CDKN1B, indicating BCL6 pathway engagement, collectively driving antitumor effects

BCL6, B cell lymphoma 6; CDKN1B, Cyclin Dependent Kinase Inhibitor 1B; CDX, cell derived xenograft; IRF4, Interferon Regulatory Factor 4; PTPN6, Protein Tyrosine Phosphatase Non-Receptor Type 6

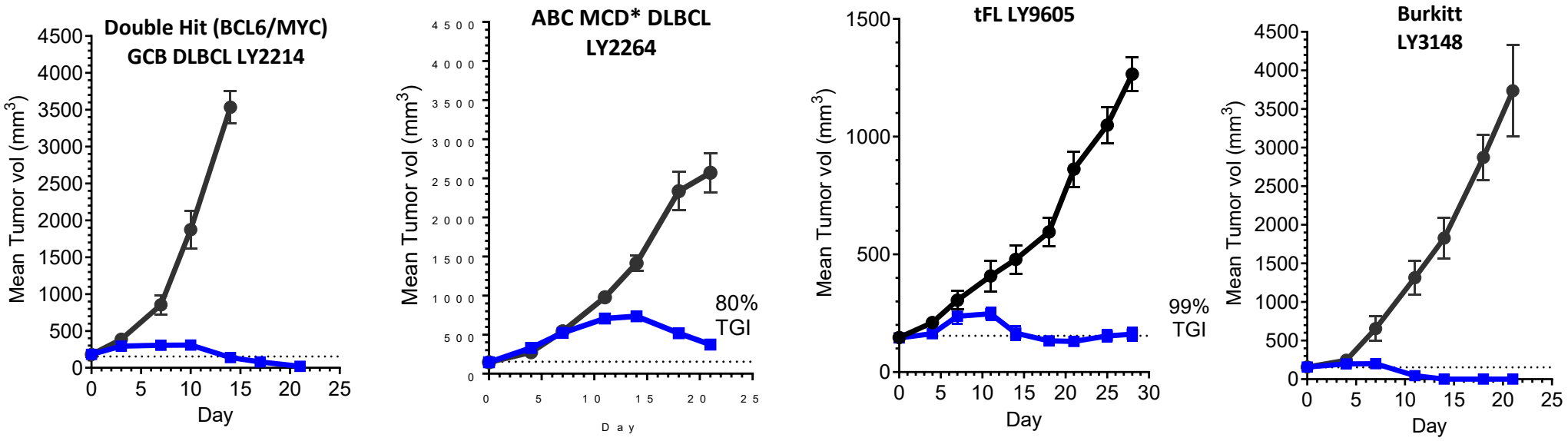
Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.

Statistics performed using one-way ANOVA with Dunnett's multiple comparison test.

Single agent ARV-393 induces tumor growth inhibition in PDX models of various non-Hodgkin lymphoma subtypes

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



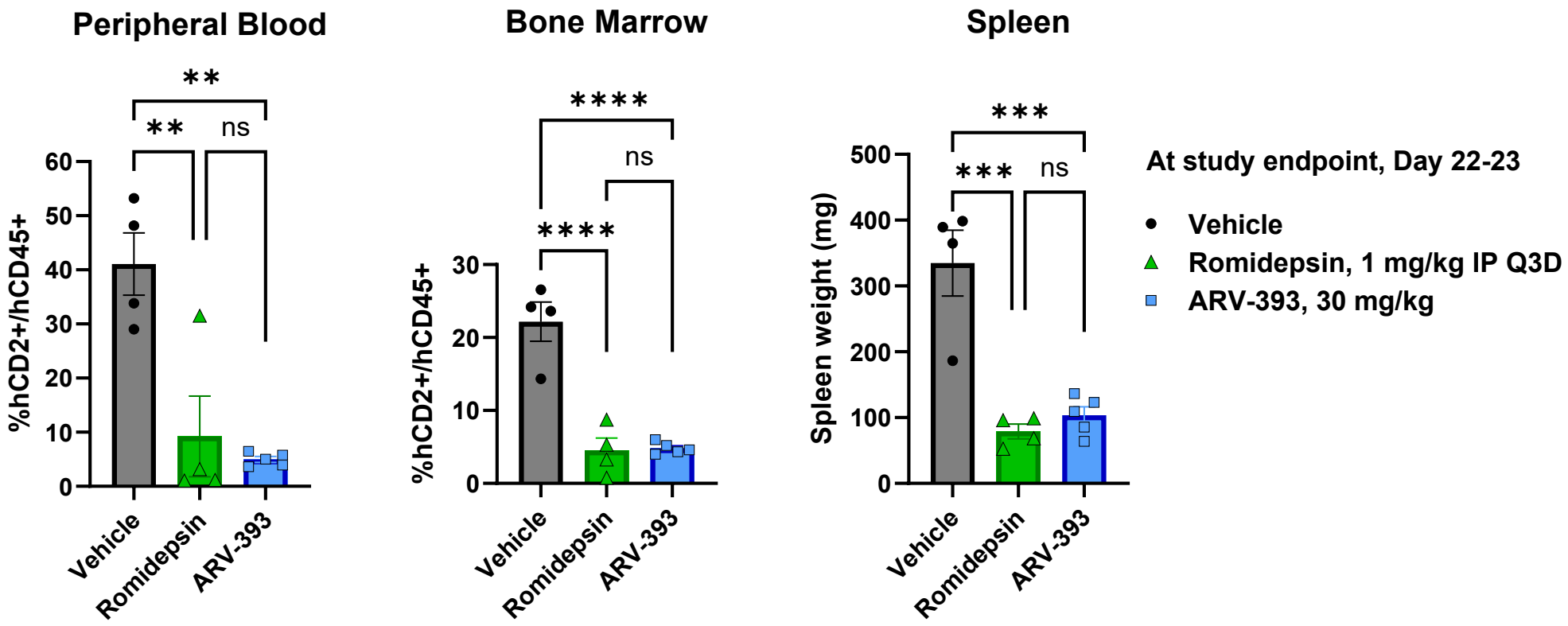
Similar results seen in fifteen PDX models of various NHL subtypes

ABC, activated B cell-like; BCL6, B-cell lymphoma 6; DLBCL, diffuse large B-cell lymphoma; GCB, germinal center B cell-like; NHL, non-Hodgkin's lymphoma; PDX, patient derived xenograft model; tFL, transformed follicular lymphoma; TGI, tumor growth inhibition
*molecular classification according to LymphGen analysis; ** Body weight not shown.
Wright et al., 2020. Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain. Van Acker et al., European Hematology Association (EHA) Poster PF1000. June 2025, Milan Italy.

First preclinical evidence of efficacy with a BCL6-targeted degrader in a patient-derived model of a rare T-cell lymphoma with a high unmet need

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ARV-393 demonstrates evidence of significant efficacy (comparable to SOC romidepsin) in a chemo (CHOP) relapsed AITL PDX model



AITL, angioimmunoblastic T-cell lymphoma, also known as nodal T-follicular helper cell lymphoma, angioimmunoblastic-type; BCL6, B cell lymphoma 6; CHOP, Cyclophosphamide, hydroxydaunorubicin, vincristine sulfate, and prednisone; ns, not significant; SOC, standard of care; PDX, patient-derived xenograft.

**P<0.01; ***P<0.005; ****P<0.0001

Van Acker et al., European Hematology Association (EHA) Poster PF1000. June 2025, Milan Italy.

ARV-393 has the potential to be an attractive combination partner for development of novel treatment options for non-Hodgkin lymphoma

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CHEMOTHERAPY-FREE AND IMPROVED CHEMOTHERAPY OPTIONS

- **Standard of Care Chemotherapy**
  - R-CHOP
- **Standard of Care Biologics**
  - CD19 (tafasitamab)
  - CD79b (polatuzumab vedotin)
  - CD20 (rituximab)
  - CD20xCD3 (glofitamab)

ARV-393 Combinations

In Vivo DLBCL models

CHEMOTHERAPY-FREE AND ALL ORAL OPTIONS

Small molecule inhibitors

- BTK (acalabrutinib)
- BCL2 (venetoclax)
- EZH2 (tazemetostat)

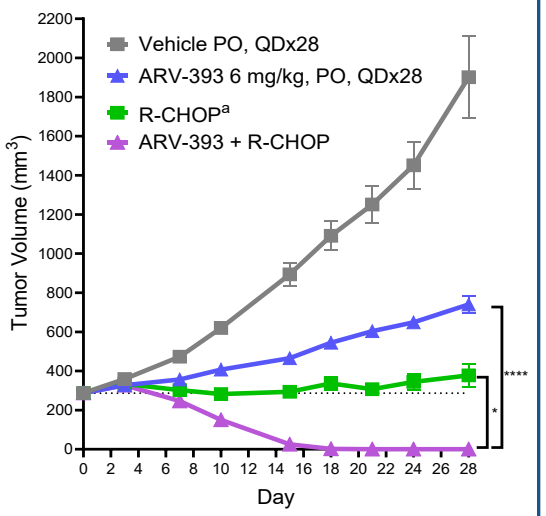
In preclinical models of aggressive DLBCL, ARV-393 demonstrated broad combinability, with tumor regressions observed in combination with SOC chemotherapy, SOC biologics, and investigational oral small molecule inhibitors targeting clinically validated oncogenic drivers of lymphoma

DLBCL, diffuse large B-cell lymphoma; R-CHOP, rituximab, cyclophosphamide, hydroxydaunorubicin, vincristine sulfate, and prednisone; SOC, standard of care

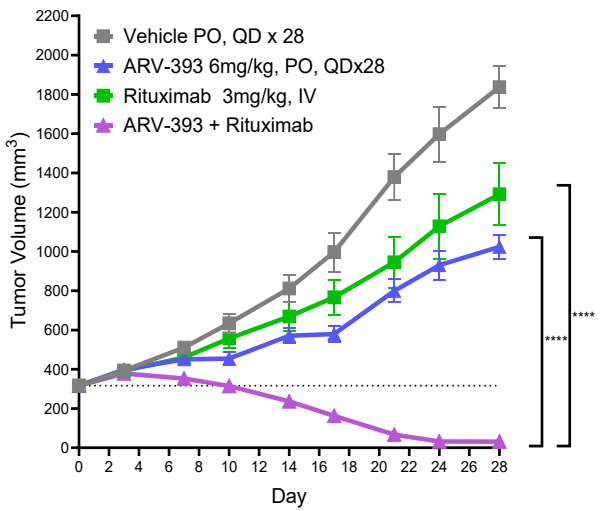
Acker et al., American Association for Cancer Research (AACR) Poster 1655/15. April 2025, Chicago.

ARV-393, in combination with SOC chemotherapy/biologics, drives tumor regressions, including complete responses, in a preclinical model of aggressive DLBCL

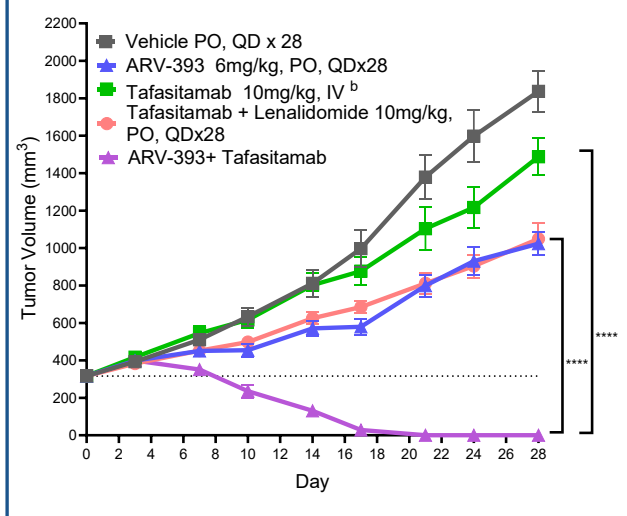
R-CHOP



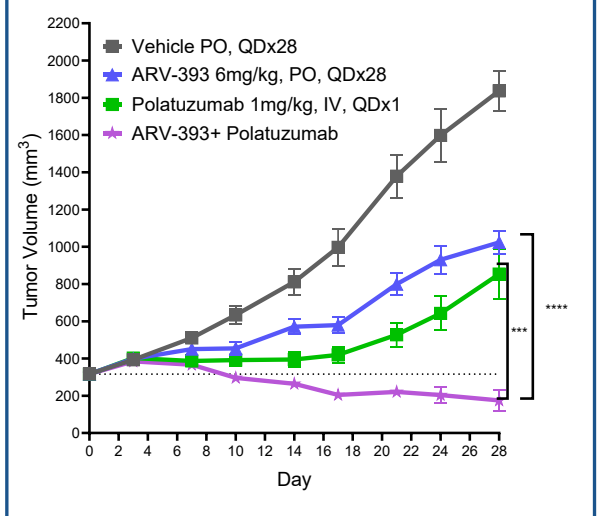
Rituximab



Tafasitamab



Polatuzumab vedotin

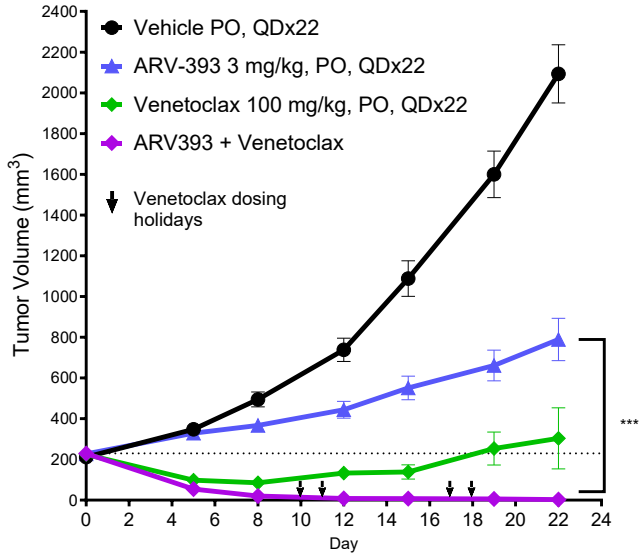


All data is in SU-DHL-4 high grade (triple hit) CDX model of diffuse large B-cell lymphoma

CDX, cell-derived xenograft; DLBCL, diffuse large B-cell lymphoma; IV, Intravenous; PO, oral; QD, once a day; R-CHOP, rituximab, cyclophosphamide, hydroxydaunorubicin, vincristine sulfate, and prednisone; SOC, standard of care
a. Rituximab 3 mg/kg was administered intravenously (IV) on days 1, 8, 15, and 22; CHOP (30:2.475:0.375:0.15 mg/kg) was given IV on day 1 (prednisone was given PO QD on days 1–5). b. Tafasitamab was administered IV on days 1, 4, 8, 15 and 22.
*P<0.05; ***P<0.005; ****P<0.0001 (one-way ANOVA, Tukey's multiple comparisons);
Acker et al., American Association for Cancer Research (AACR) Poster 1655/15. April 2025, Chicago.

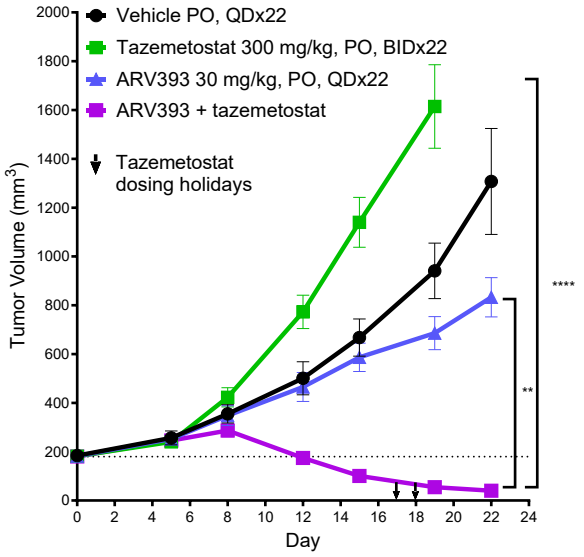
ARV-393, in combination with small molecule inhibitors, drives tumor regressions, including complete responses, in a preclinical model of aggressive DLBCL

Venetoclax



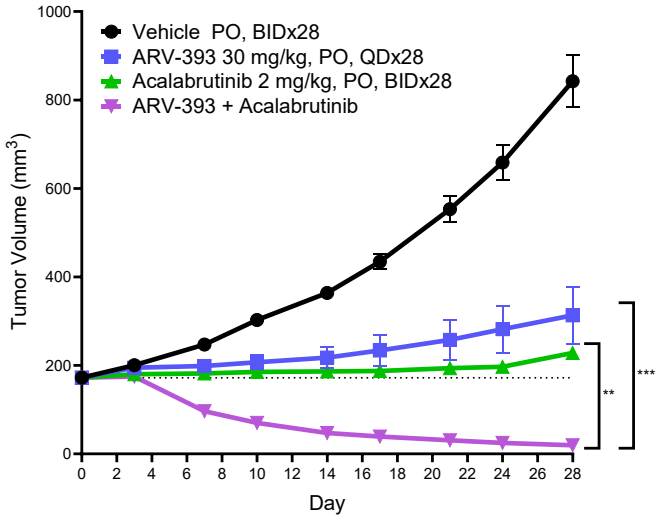
OCI-Ly1 GCB DLBCL CDX

Tazemetostat



SU-DHL-6 high grade (triple hit) DLBCL CDX

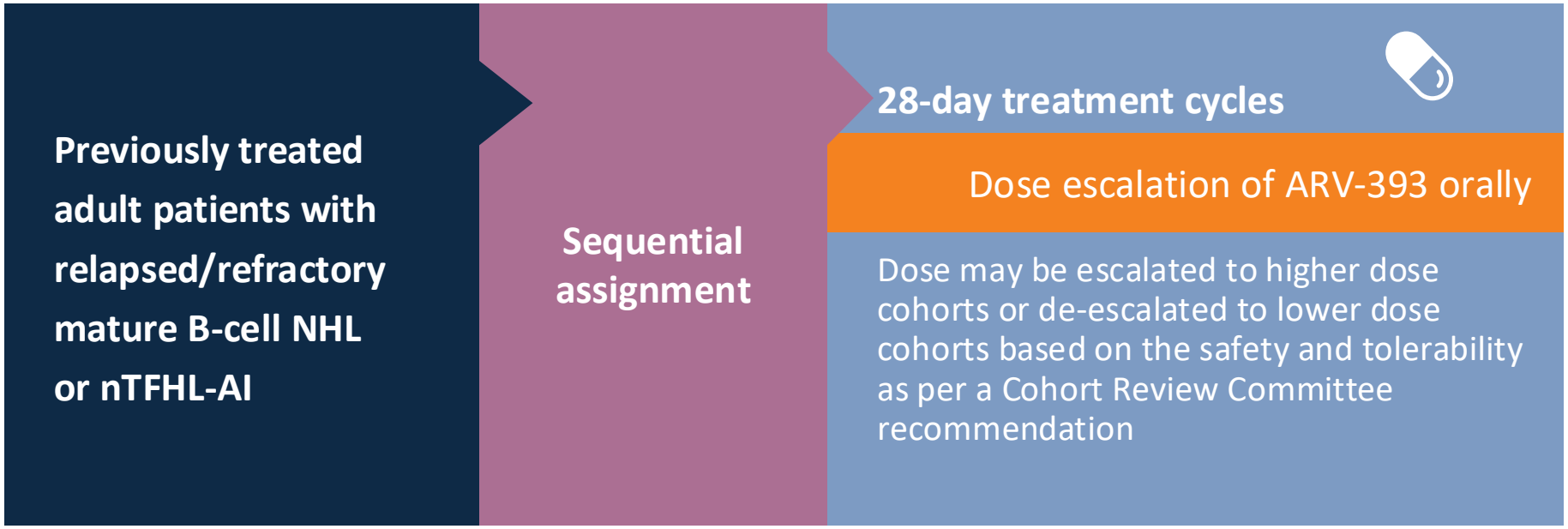
Acalabrutinib



OCI-Ly10 ABC DLBCL CDX

ABC, activated B-cell; BID, twice a day; CDX, cell-derived xenograft; DLBCL, diffuse large B-cell lymphoma; GCB, germinal center B-cell; IV, Intravenous; PO, oral; QD, once daily.
**P<0.01; ***P<0.005; ****P<0.0001 (one-way ANOVA, Tukey's multiple comparisons)
Van Acker et al., American Association for Cancer Research (AACR) Poster 1655/15. April 2025, Chicago; Van Acker et al., European Hematology Association (EHA) Poster PF1000. June 2025, Milan Italy.

Arvinas is currently enrolling a Phase 1 clinical trial of ARV-393 in relapsed/refractory non-Hodgkin lymphoma*



Phase 1 monotherapy dose escalation data disclosure from early cohorts, with focus on T-cell lymphomas (4Q 2026)

Updated Phase 1 monotherapy dose escalation data in B- and T-cell lymphomas, and combination data with glofitamab in DLBCL (mid-2027)

- **ARV-393 is being evaluated in Phase 1 dose escalation study to assess its safety, tolerability, pharmacokinetics (PK), pharmacodynamics (PD), and preliminary antitumor activity as monotherapy for R/R NHL, and in combination with glofitamab for R/R DLBCL (ClinicalTrials.gov ID: NCT06393738)**

*Phase 1 clinical trial includes monotherapy cohort in relapsed/refractory NHL and combination cohort with glofitamab in diffuse large B-cell lymphoma
DLBCL, diffuse large B-cell lymphoma; NHL, non-Hodgkin lymphoma; nTFHL-AI, nodal T-follicular helper cell lymphoma, angioimmunoblastic-type, R/R, relapsed/refractory.



CLINICAL PROGRAMS: Neurology

ARV-027

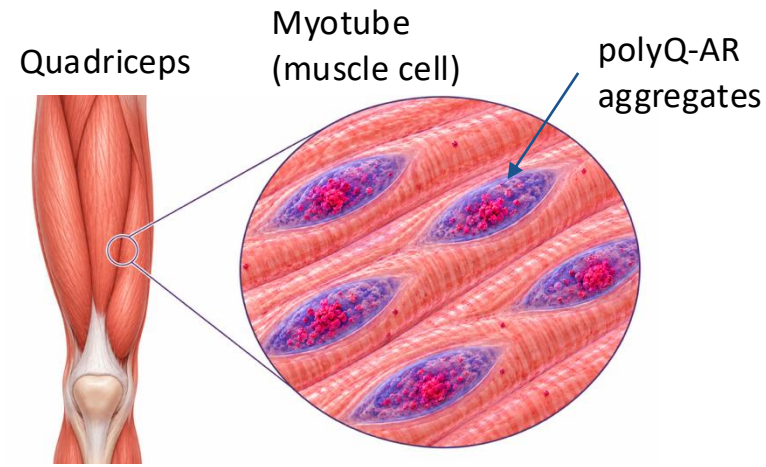
PROTAC polyQ-AR degrader

ARVINAS

ARV-027 is an investigational compound. Its safety and effectiveness have not been established.

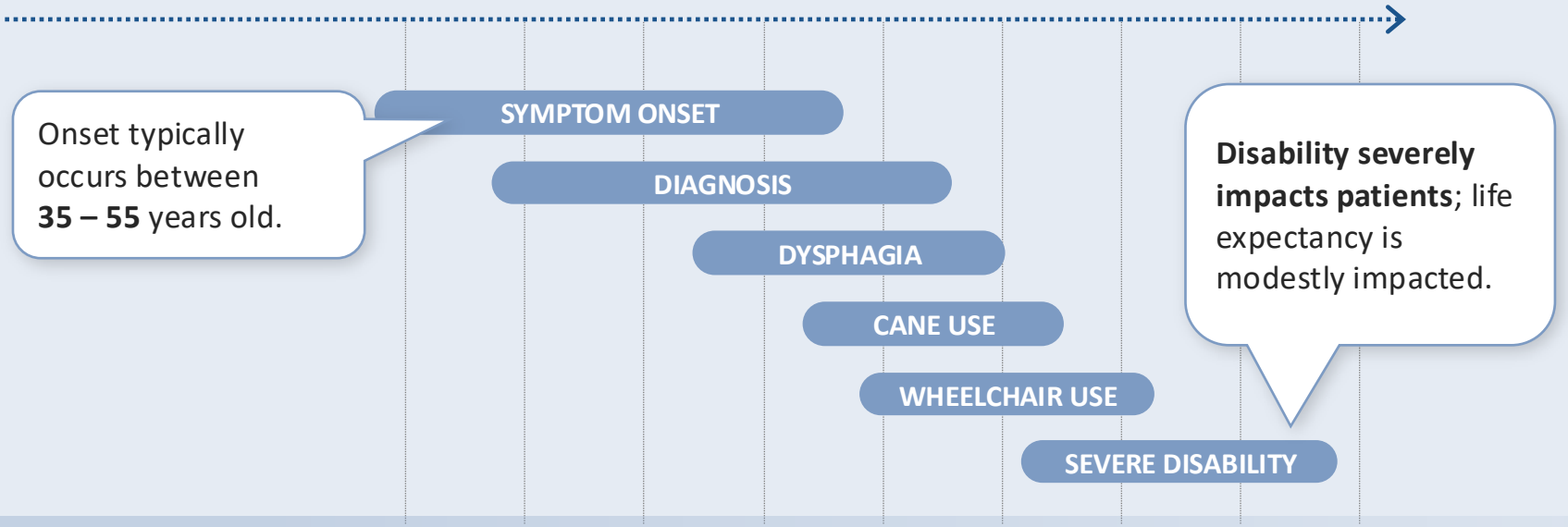
SBMA is a genetically-defined disease caused by a trinucleotide CAG repeat expansion in the androgen receptor (polyQ-AR)

SBMA Disease Overview



Clinical features result from both the loss of normal androgen signaling and gain of toxic properties due to the polyQ-expansion[1]

SBMA Natural History and Treatment Options



There are no approved therapies for SBMA in the U.S.

- Current treatment focuses on symptom management only, including physical therapy and rehabilitation
  - SBMA prevalence is estimated between 5.5-20K patients in the U.S.[2]

Spinal-bulbar muscular atrophy (SBMA), also known as Kennedy's disease, is a rare, X-linked, neuromuscular disease caused by a repeat expansion in the androgen receptor gene that encodes polyQ-AR.

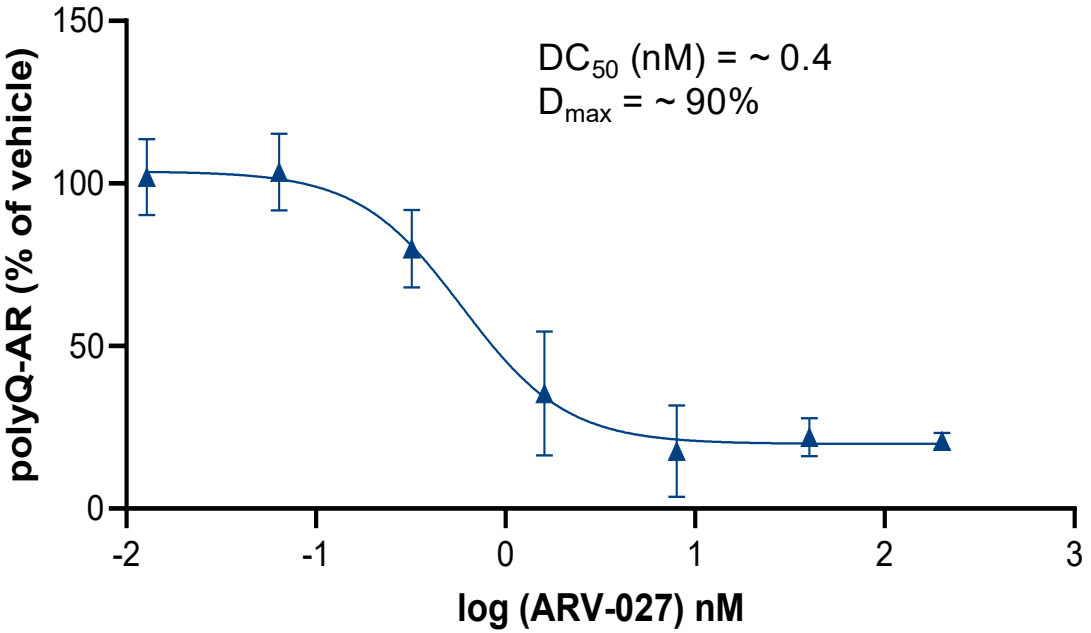
polyQ-AR, polyglutamine-expanded (polyQ) androgen receptor; SBMA, spinal-bulbar muscular atrophy.

1. Grunseich et al. Curr Opin Neurol 2020; 33(5):629-634; 2. Zanovello et al., 2023.

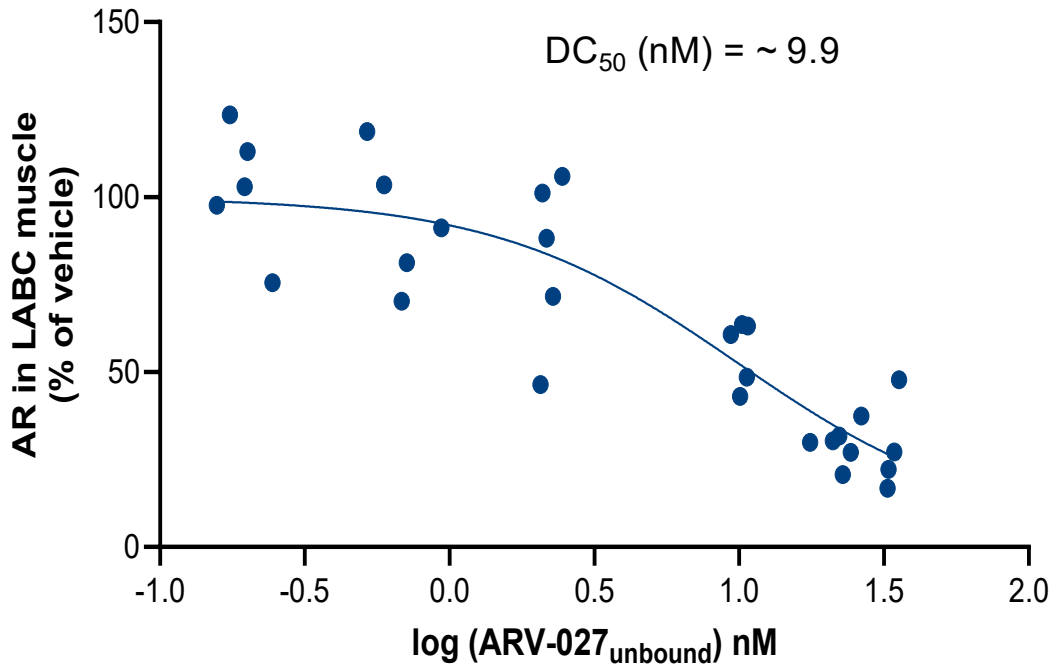
ARV-027 is a potent *in vitro* and *in vivo* PROTAC degrader of polyQ-AR, the root cause of SBMA[1]

ARVINAS

ARV-027 induces degradation of polyQ-AR in myotubes derived from SBMA patient induced pluripotent stem cells



ARV-027 induces degradation of AR in muscle tissue from male mice



The mechanism of the polyQ-AR degradation was confirmed to be E3 ligase and proteosome dependent (data not shown)

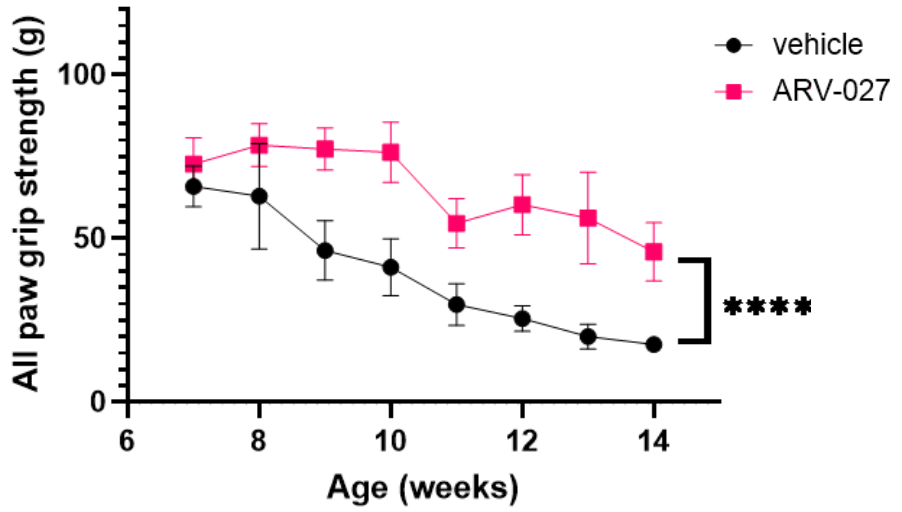
polyQ-AR, polyglutamine-expanded (polyQ) androgen receptor (AR); SBMA, spinal bulbar muscular atrophy

1. Young J, et al. JNEUROSCI.4072-08.2009

Gregory J et al., International Congress of the World Muscle Society (WMS), Poster 718LBP. Presented October 10, 2025

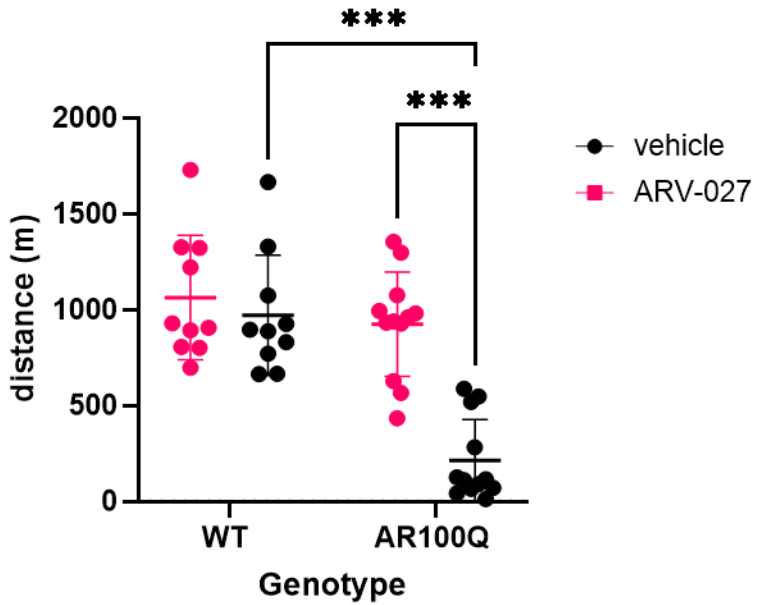
In preclinical models, ARV-027 induced polyQ-AR degradation in muscle tissue and rescued strength and endurance in a rapidly progressing model of SBMA[a]

Muscle Strength
All Paw Grip Strength



Loss of grip strength was significantly delayed

Muscle Endurance
Treadmill Performance at Week 11



Treadmill performance was indistinguishable from wildtype littermates

polyQ-AR, polyglutamine-expanded androgen receptor; SBMA, spinal-bulbar muscular atrophy.
a. AR100Q SBMA mouse model. Chivet et al. 2020.
Gregory J et al., International Congress of the World Muscle Society (WMS), Poster 718LBP. Presented October 10, 2025.

A first-in-human Phase 1 clinical trial of ARV-027 is currently enrolling healthy volunteers

Status of Arvinas Trials with ARV-027

OBJECTIVES: Safety, Tolerability, PK, and PD

ARV-027 Phase 1 trial

SINGLE ASCENDING DOSE in healthy volunteers	**MULTIPLE ASCENDING DOSE** in healthy volunteers	**MULTIPLE DOSE** in patients with SBMA
Complete: Part A ✓ Single ascending dose cohorts in healthy volunteers	Ongoing: Part B ✓ Multiple ascending dose cohorts in healthy volunteers	*Planned: Part C* (1H 2027) • Multiple doses in patients with SBMA

Initial data from Phase 1 clinical trial evaluating AR degradation in muscle anticipated in 1H 2027

AR, androgen receptor; PK; Pharmacokinetics; PD, pharmacodynamics; SBMA; spinal bulbar muscular atrophy



CLINICAL PROGRAMS: Oncology

ARV-6723

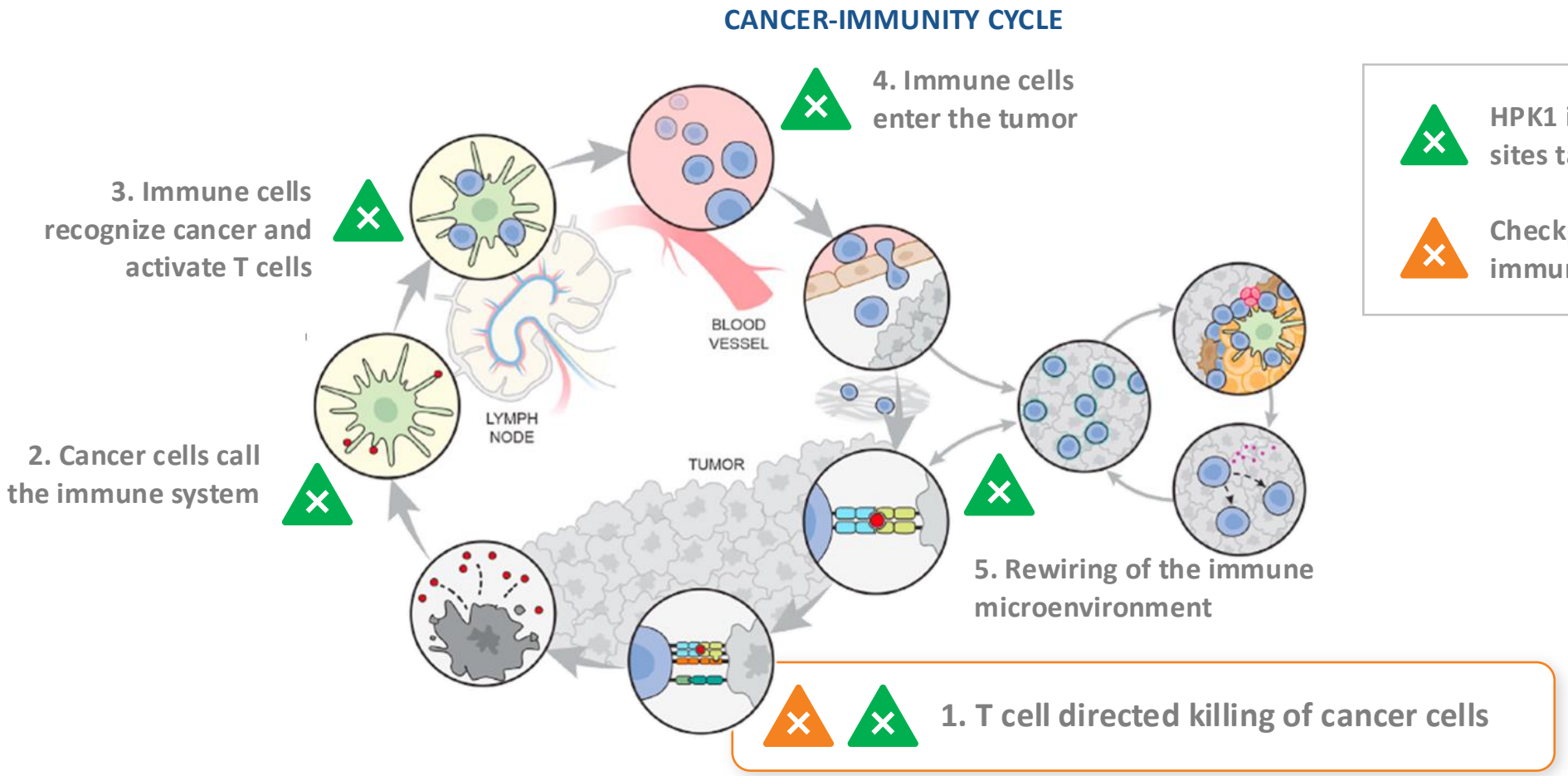
PROTAC HPK1 degrader

ARVINAS

ARV-6723 is an investigational compound. Its safety and effectiveness have not been established.

HPK1 degradation may help strengthen the immune response by acting at multiple points in the cancer-immunity cycle

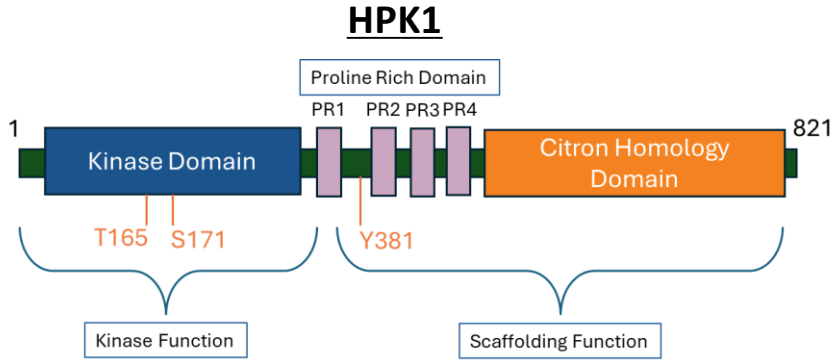
ARVINAS



New therapies are needed to address IO therapy resistance in many malignancies

HPK1, hematopoietic progenitor kinase 1; IO, immuno-oncology
Image adapted from Mellman et al., Immunity 2023.

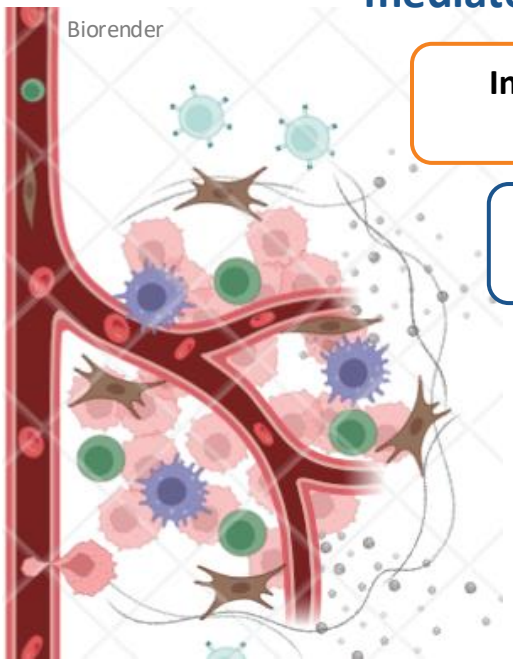
ARV-6723 is a potent PROTAC degrader of HPK1, a central regulator of immune suppression



HPK1, a scaffolding protein kinase expressed exclusively in the hematopoietic compartment (T/B/dendritic/NK cells), has 2 functional domains:

- Kinase domain: drives catalytic immune suppression through adaptive immune pathways.
- Scaffolding domain: broad immunosuppressive regulation through innate immune pathways.

HPK1 degradation destroys both kinase and scaffold-mediated signaling, and may lead to:



Tumor Microenvironment (TME)

Increase immune function (T/dendritic cells) and immune attack (T/NK cells)

Counter activation of T cell exhaustion

Block suppressive TME (PGE_2, Adenosine)

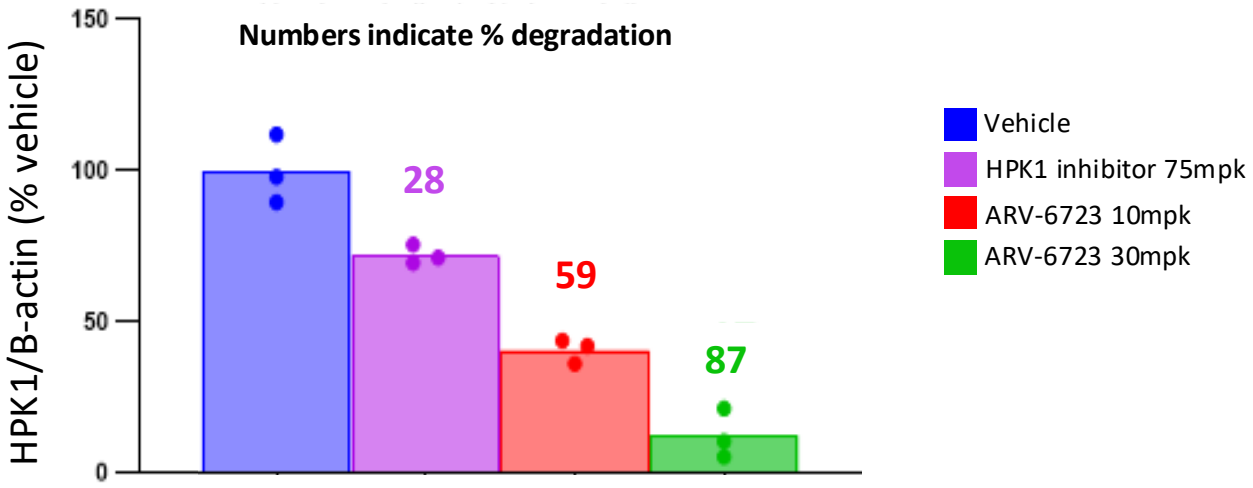
Reprogram the TME to unleash immune response

ARV-6723, an oral HPK1 degrader, could offer sustained anti-tumor immune response as a single agent or in combination with SoC (e.g., anti-PD1, chemotherapy), resulting in improved clinical benefits over SoC across a wide range of cancers

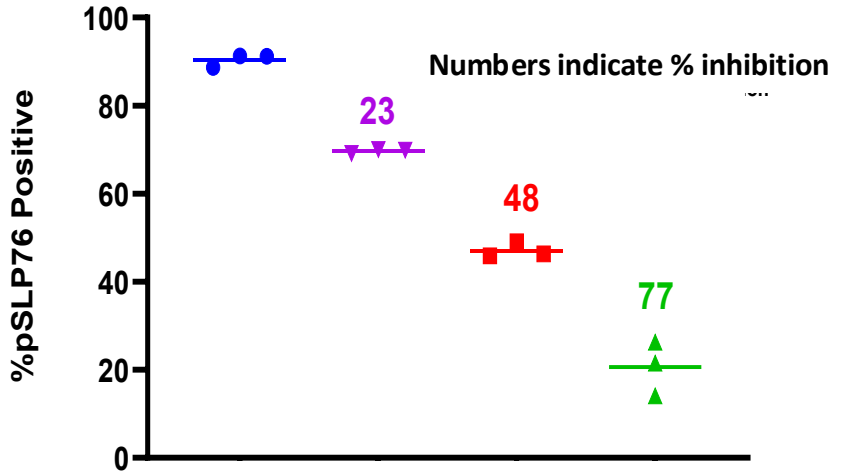
HPK1, hematopoietic progenitor kinase 1; NK, natural killer; PGE_2, prostaglandin E2; SoC, standard of care;

Single dose of oral ARV-6723 showed profound and sustained target degradation and durable pathway engagement in preclinical models

HPK1 Degradation



Downstream Pathway Inhibition (pSLP76)



24h post oral dose

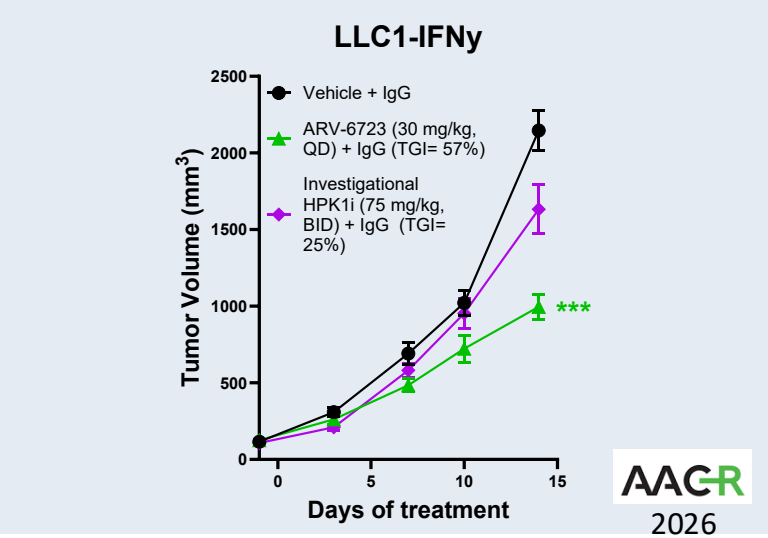
- ARV-6723 is a potent single-digit nanomolar degrader of HPK1, leading to sustained downstream pathway inhibition (via pSLP76) and induction of IL-2 release (data on file)
- This enhanced PK/PD profile results in more complete *in vivo* pathway suppression that recapitulates a genetic KO phenotype upon repeated PROTAC dosing

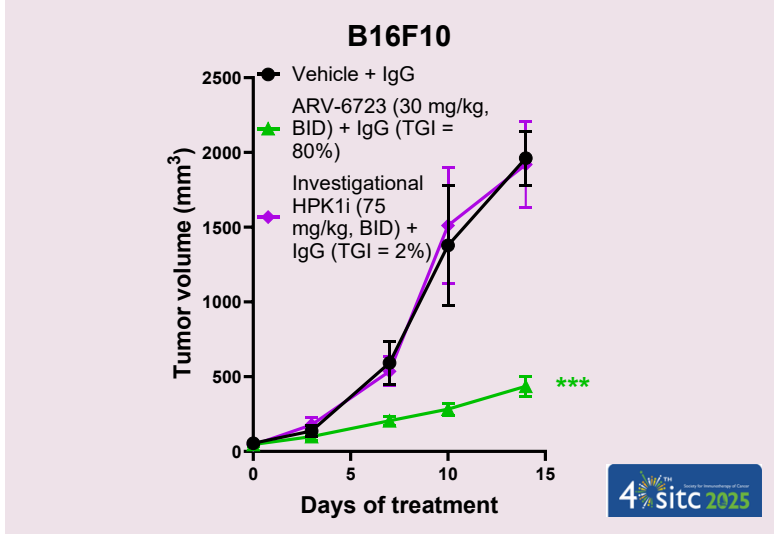
As presented at

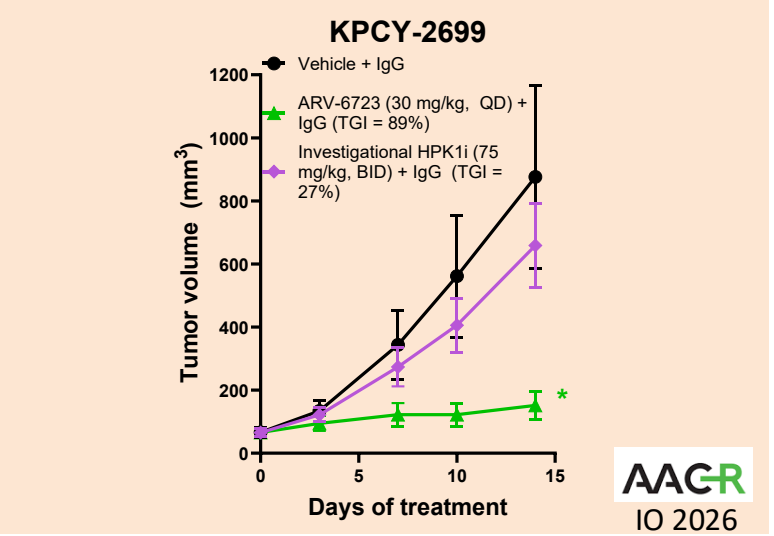
HPK1, hematopoietic progenitor kinase 1; KO, knock out; mpk, milligrams per kilogram; PK/PD, pharmacokinetic/pharmacodynamic; pSLP76, SH2 domain-containing leukocyte protein of 76 kDa, phosphorylated.

ARV-6723 outperforms an investigational HPK1 inhibitor in ICI-resistant models

Tumor Growth Efficacy Studies







Biological context	Mouse model	ARV-6723 anti-tumor activity (TGI%) as a single agent	Head-to-head comparison to investigational HPK1 inhibitor (TGI%)
T cell exhaustion	LLC1-IFNγ	**57% TGI;** (30 mg/kg, QD)	25% (non- significant); (75 mg/kg, BID)
Low tumor antigens	B16-F10	**65-80% TGI;** (30 mg/kg, BID)	2% TGI (non- significant); (75 mg/kg, BID)
T cell exclusion	KPCY	**89% TGI;** (30 mg/kg, QD)	27% (non- significant); (75 mg/kg, BID)

ARV-6723 has significant single agent anti-tumor activity in 7 ICI-resistant models (3 models shown)

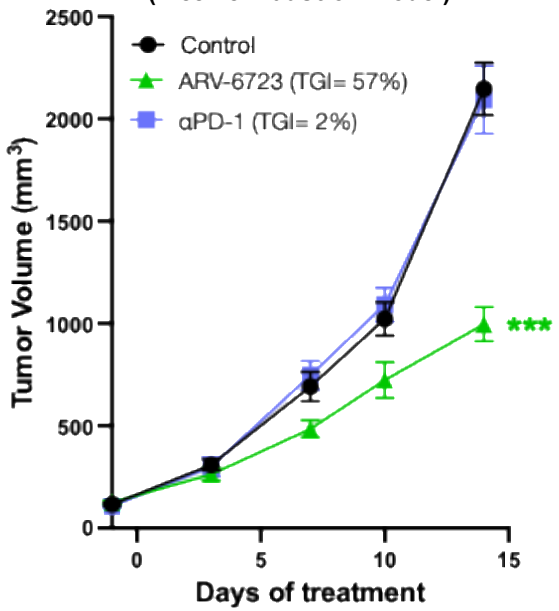
BID, twice-daily dosing; HPK1, hematopoietic progenitor kinase 1; ICI, immune checkpoint inhibitors; IFNγ, Interferon-gamma; I/O, immuno-oncology; PDAC, pancreatic ductal adenocarcinoma; QD, once daily; TGI, tumor growth inhibition

Preclinical work suggests that an HPK1 degrader could work after αPD-1 progression, in combination with αPD-1, and ahead of αPD-1

ARVINAS

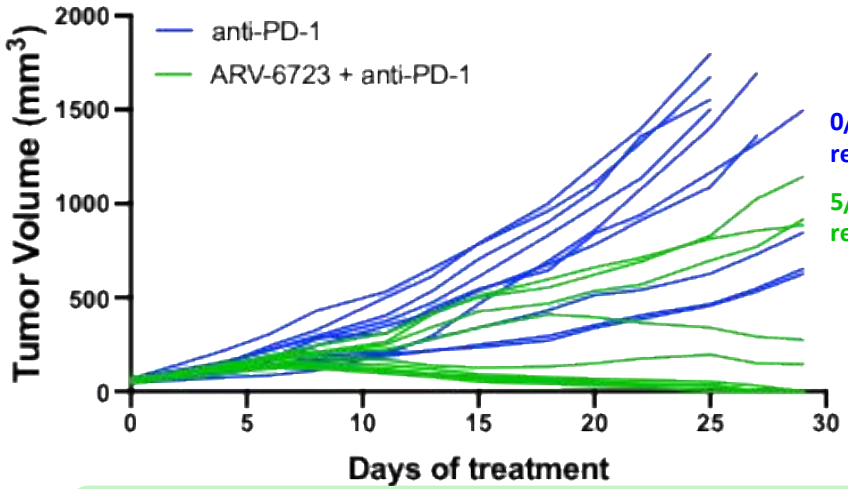
In a T cell exhaustion model of αPD-1 progression, ARV-6723 drives tumor inhibition

LLC1-IFNγ[1]
(T cell exhaustion model)



ARV-6723 combines with αPD-1 to improve preclinical outcomes

CT26 Efficacy[2]



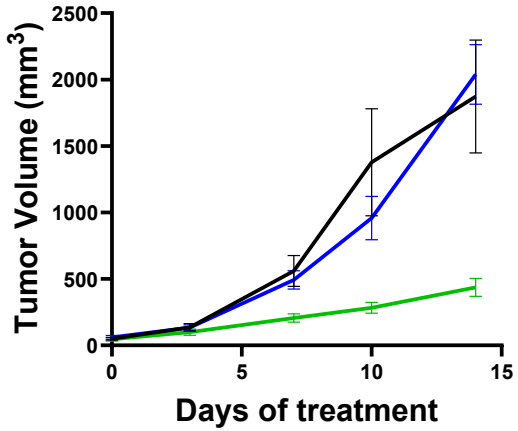
0/10 Complete responders

5/10 Complete responders

Evidence of immunological memory demonstrated in the 5 complete responders

ARV-6723 inhibits tumor growth in a low-immunogenic model where αPD-1 fails

B16F10 Efficacy[2]



Control
αPD-1
ARV-6723 (TGI = 79% ***)

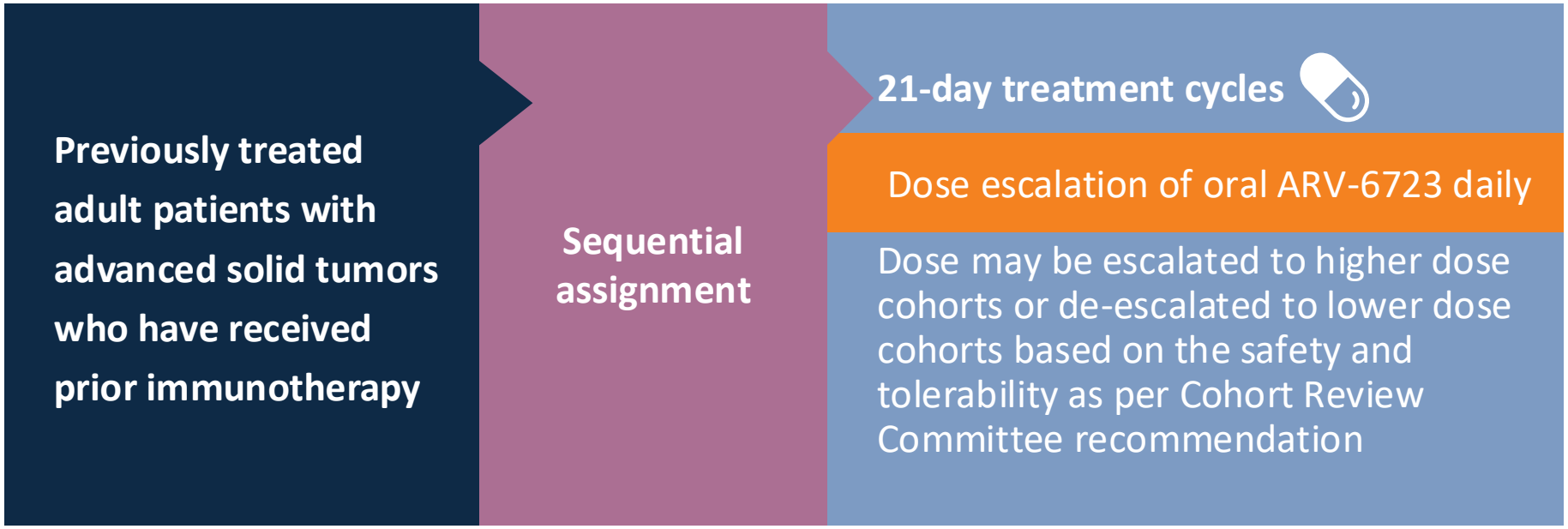
One-way ANOVA ***p<0.001

ARV-6723 has significant single agent anti-tumor activity in 7 ICI-resistant models, slowing tumor growth and supporting an immune response against cancer

αPD-1, anti-programmed death-1; HPK1, hematopoietic progenitor kinase 1; IO, immuno-oncology.

1. Van Acker et al. American Association for Cancer Research (AACR) Poster 4309. Presented April 21, 2026. 2. Corwin et al. Society for Immunotherapy of Cancer (SITC) Poster 1158. Presented November 8, 2025.

Phase 1/2 clinical trial of ARV-6723 as monotherapy and in combination with pembrolizumab in advanced solid tumors



- Initiated dosing of patients in the monotherapy cohort of the Phase 1/2 clinical trial
  - Trial design includes plan for dose escalation and expansion combination cohort with immune checkpoint inhibitor (pembrolizumab; anticipated in mid-2027)

ARV-6723 is being evaluated in a Phase 1/2 open-label study to evaluate safety, tolerability, pharmacokinetics, and preliminary anti-tumor activity administered as monotherapy and in combination with pembrolizumab in participants with advanced solid tumors (ClinicalTrials.gov Identifier: NCT07749586)

Cohort Review Committee is comprised of study investigators, sponsor/CRO medical lead, select sponsor/CRO clinical team members



CLINICAL PROGRAMS: Neurology

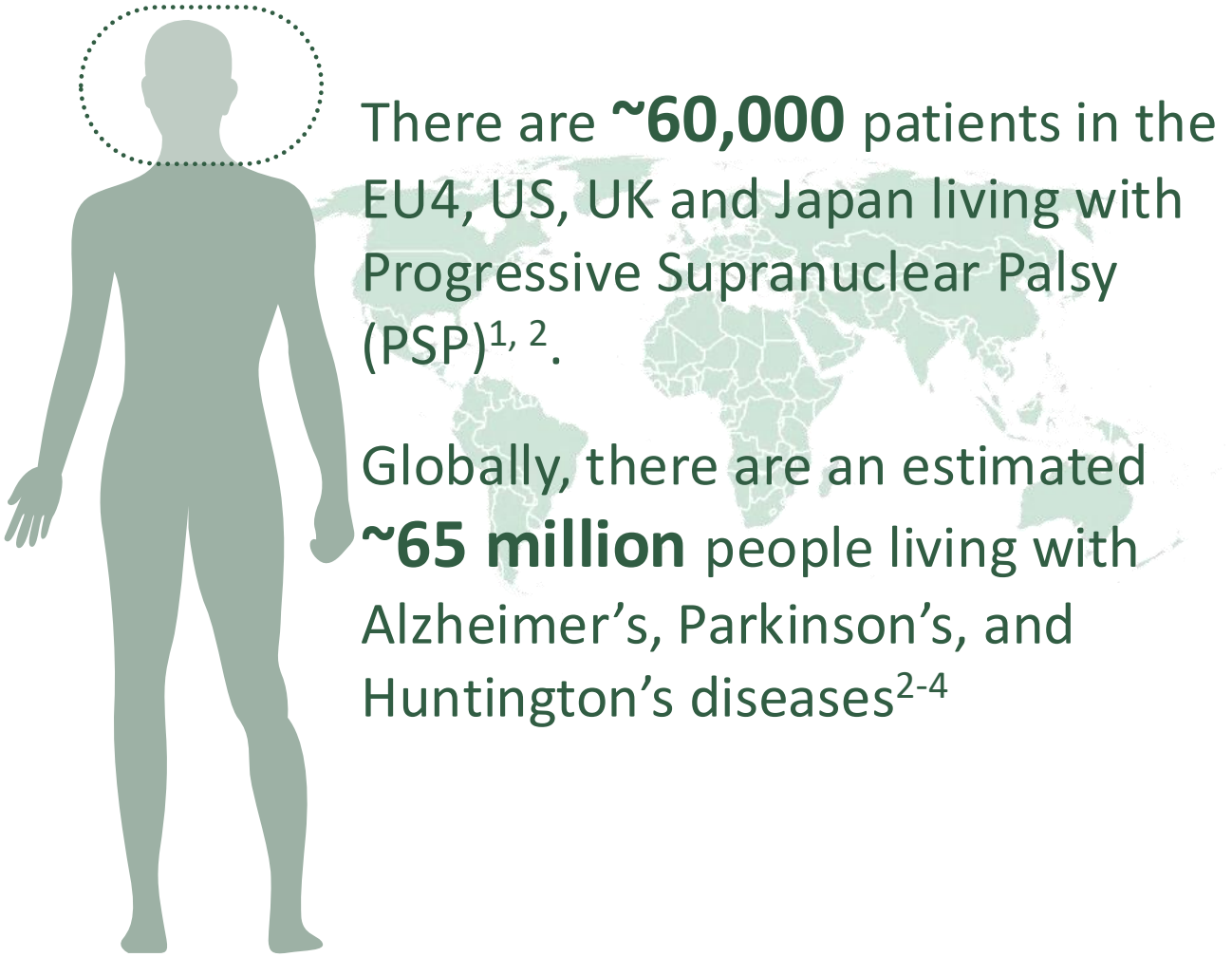
ARV-102

PROTAC LRRK2 degrader

ARVINAS

ARV-102 is an investigational compound. Its safety and effectiveness have not been established.

Neurodegenerative diseases: An area of tremendous unmet need



There are **~60,000** patients in the EU4, US, UK and Japan living with Progressive Supranuclear Palsy (PSP)[1, 2].

Globally, there are an estimated **~65 million** people living with Alzheimer's, Parkinson's, and Huntington's diseases[2-4]

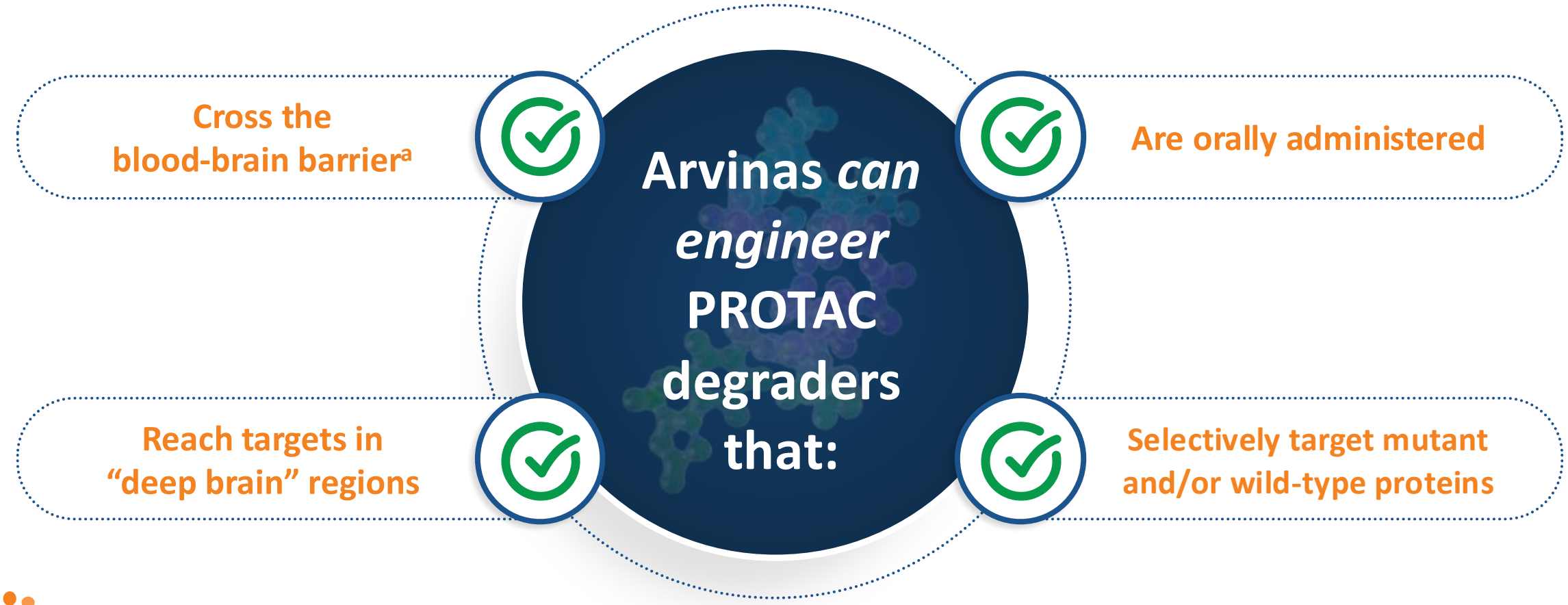
Unmet need is high:

- No approved disease-modifying therapies for multiple important target proteins (e.g., LRRK2, tau, α-synuclein) implicated in the pathology of various neurodegenerative diseases[5]
- Blood-brain barrier penetration is a challenge for other modalities (e.g., antibodies and antisense oligonucleotides)
- Other existing and potential therapies have difficult routes of administration (e.g., intrathecal, intracerebral)

EU4, Germany, Spain, France, Italy

1. Lyons S et al. Journal of Neurology. 2023;270:4451–4465; 2. Takigawa H et al. Brain and Behavior. 2016;6.; 3. Willis et al., npj Parkinson's Dis. 2022; 4. Yohrling, et al., Neurology 2020; 5. Lyons S, et al. J Neurol. 270, 4451–4465 (2023) and Counsell CE, Swallow DMA, Zheng CS. Mov Disord Clin Pract. 9, 604–613 (2022)

PROTAC degraders could potentially revolutionize the treatment of neurodegenerative diseases of the brain



A novel PROTAC approach with potential to differentiate from existing and emerging modalities in neurodegenerative diseases of the brain

a. In preclinical and clinical studies, dose-dependent increases in exposure in cerebral spinal fluid after single and multiple doses of ARV-102 indicated brain penetration.

LRRK2 is implicated in both in progressive supranuclear palsy and Parkinson's disease

Progressive Supranuclear Palsy (PSP)

- Rare progressive neurological disease that affects movement, balance, and cognitive function; rapid progression to death within 5-7 years
- Characterized by tauopathy (accumulation of abnormal forms of the microtubule-associated protein tau)[1]
- Preclinical data indicate that LRRK2 mutations are associated with tau pathology resembling PSP[2]
- Genetic variations in LRRK2 are associated with PSP progression, highlighting the potential importance of LRRK2 in tauopathies[2]



No approved disease-modifying therapies exist for patients with PSP or PD

Parkinson's Disease (PD)

- Neurodegenerative disease that affects movement, balance, and coordination
- Mutations in the LRRK2 gene are one of the most common genetic causes of PD; variants have also been observed in idiopathic cases[2]
- Increased LRRK2 expression and activity contributes to neurodegeneration and pathogenesis of idiopathic PD,[2] making it a rational therapeutic target

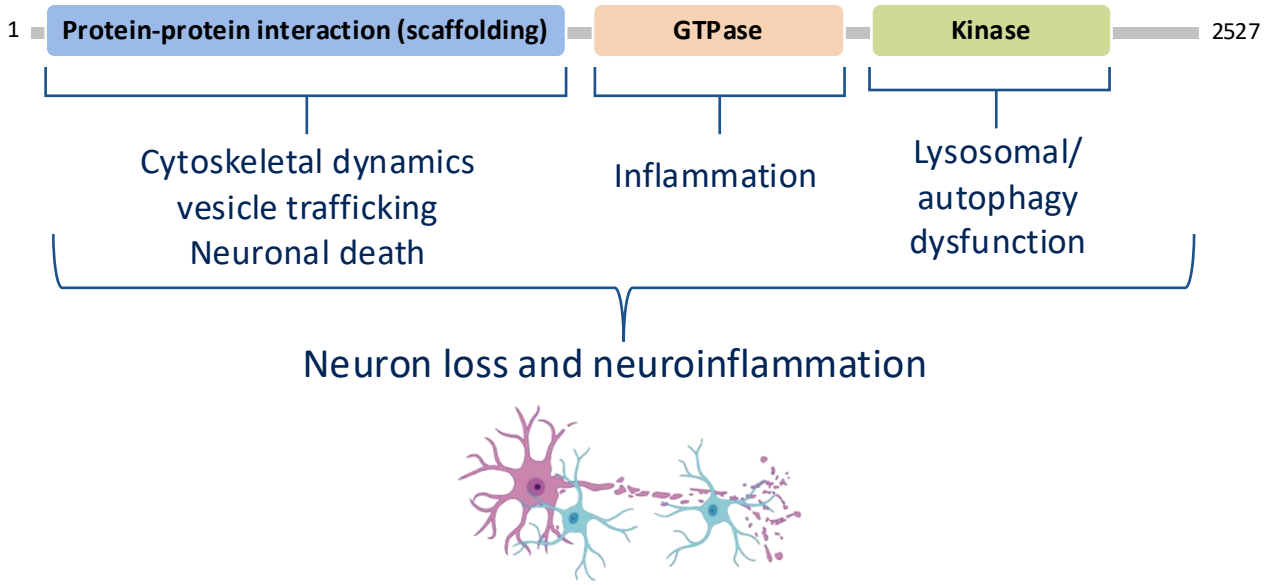
Given its role in neurodegeneration, Arvinas is exploring LRRK2-targeting PROTAC degraders as possible treatments for PSP, PD, and related diseases

LRRK2, leucine-rich repeat kinase 2

1. Herbst et al. Clin Sci 2022;136:1071-1079. 2. Kluss et al. Biochem Soc Trans. 2019;47:651-61.

PROTAC-induced LRRK2 degradation has the potential to differentiate from kinase inhibition

LRRK2 is a large multidomain scaffolding kinase[1-3]



ARV-102 degrades the entire LRRK protein and differentiates from inhibitors

LRRK2	PROTAC	Inhibitor
Protein-protein interaction (scaffolding)	✓	✗
GTPase activity	✓	✗
Kinase activity	✓	✓

Increased activity and expression of LRRK2 have been linked with the development and progression of neurological diseases like progressive supranuclear palsy[4], or PSP, and Parkinson's disease[5].

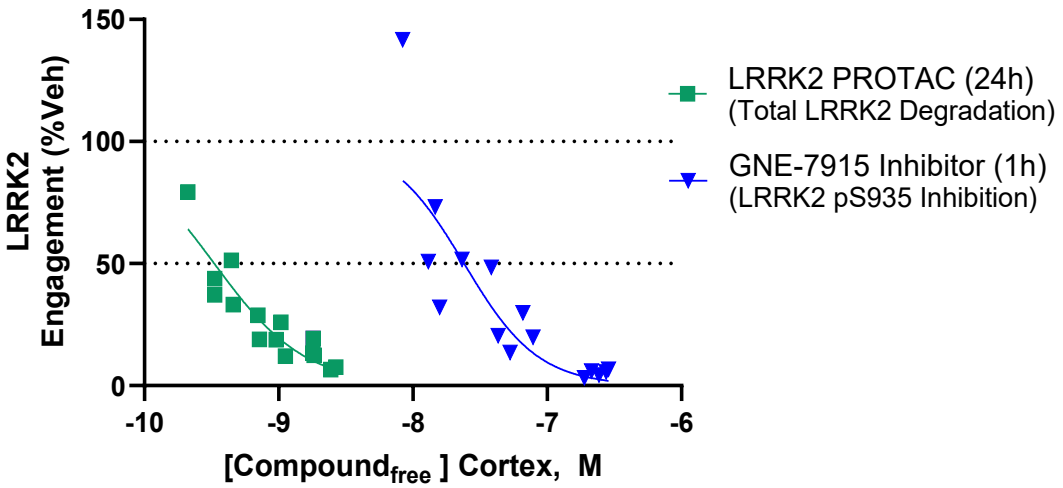
LRRK2, leucine-rich repeat kinase 2

1. Rocha EM, et al. Trends Neurosci. 2022;45(3):224-36. 2. Kluss JH, et al. Biochem Soc Trans. 2019;47(2):651-661. 3. Di Fonzo A, et al Lancet. 2005;365(9457):412-5; 4. Jabbari et al., 2021; 5.Langston et al. 2022.

ARV-102 shows 50x greater target and pathway engagement, with enhanced potency, versus a LRRK2 inhibitor in preclinical model

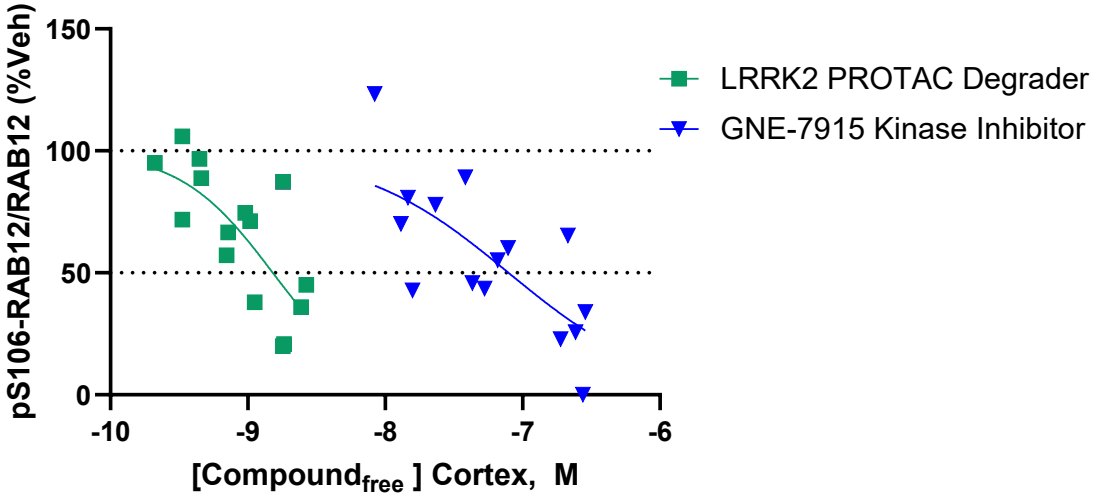
Iterative and catalytic PROTAC advantage results in stronger LRRK2 reduction and RAB pathway engagement versus a LRRK2 kinase inhibitor[a]

LRRK2 Target Engagement
LRRK2 PROTAC vs. Kinase inhibitor at Tmax



50x greater *target* engagement with ARV-102

RAB Pathway Engagement (pS106 Rab12)
(LRRK2 PROTAC vs. Inhibitor)



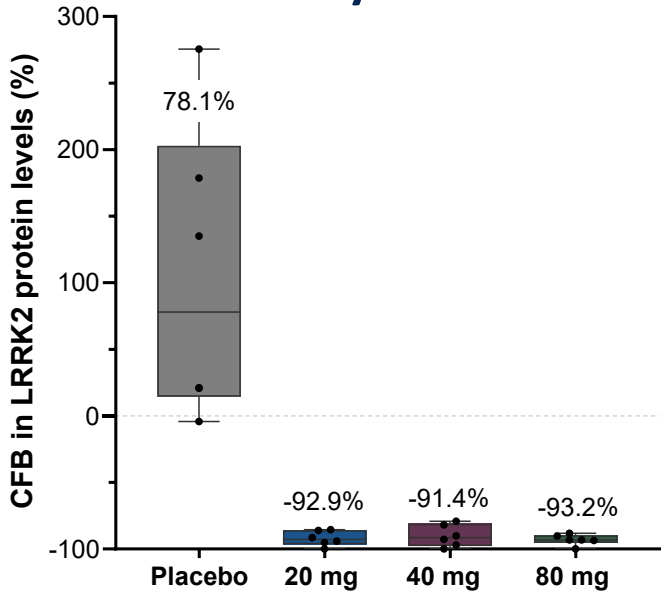
50x greater *pathway* engagement with ARV-102

LRRK2, Leucine-rich repeat kinase 2
a. G2019S familial Parkinson's Disease mouse model.
Data presented at 2023 Keystone Summit: Autophagy and Neurodegeneration.

Robust reductions of LRRK2 levels in PBMCs and BMP levels in urine were observed in patients with PD following multiple doses of ARV-102

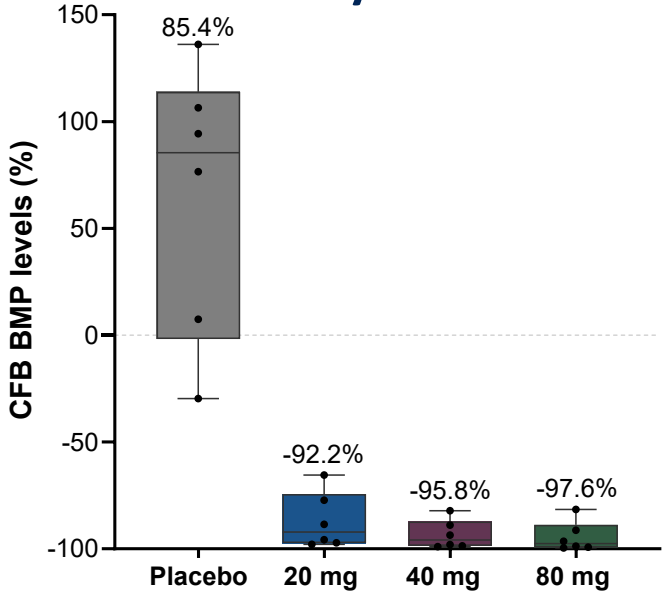
PBMC LRRK2

Day 28

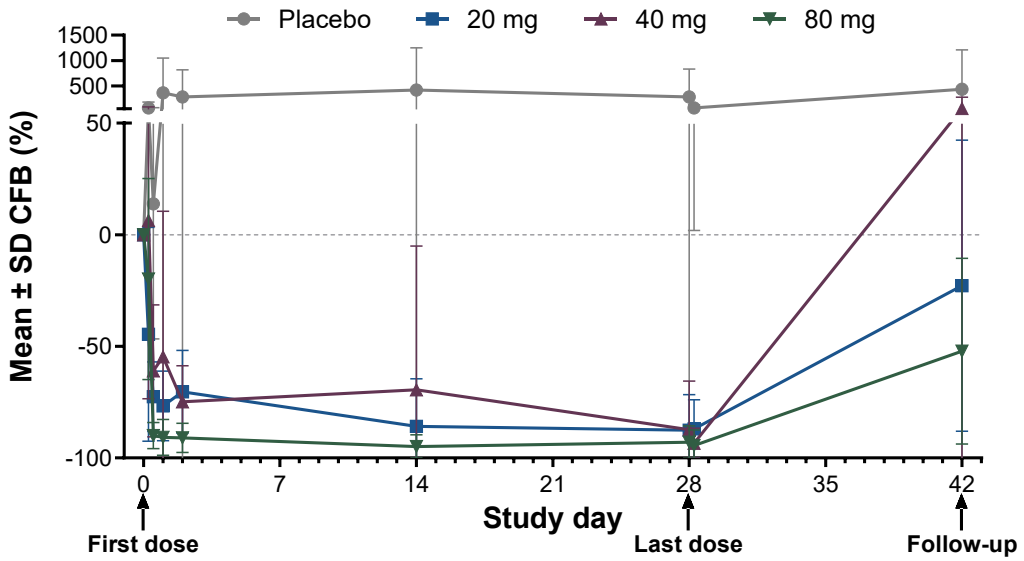


Urine BMP

Day 28



Time course



- Robust reductions of LRRK2 levels in PBMCs and BMP levels in urine were observed following multiple doses of ARV-102 treatment, with BMP levels returning towards baseline after 2 weeks of follow-up
- Peripheral pharmacodynamic biomarker results in participants with Parkinson's disease were consistent with those observed in the study of ARV-102 in healthy volunteers[1,2]

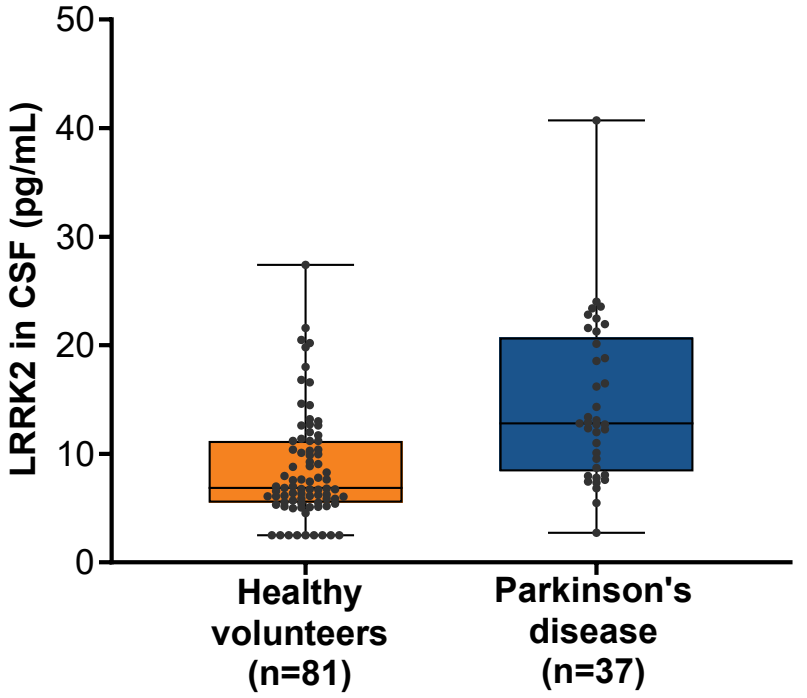
As presented at AD/PD 2026 ADVANCES IN SCIENCE & THERAPY
International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders

BMP, bis(monoacylglycerol)phosphate; CFB, change from baseline; LRRK2, leucine-rich repeat kinase 2; PBMC, peripheral blood mononuclear cell; PD, Parkinson's disease.

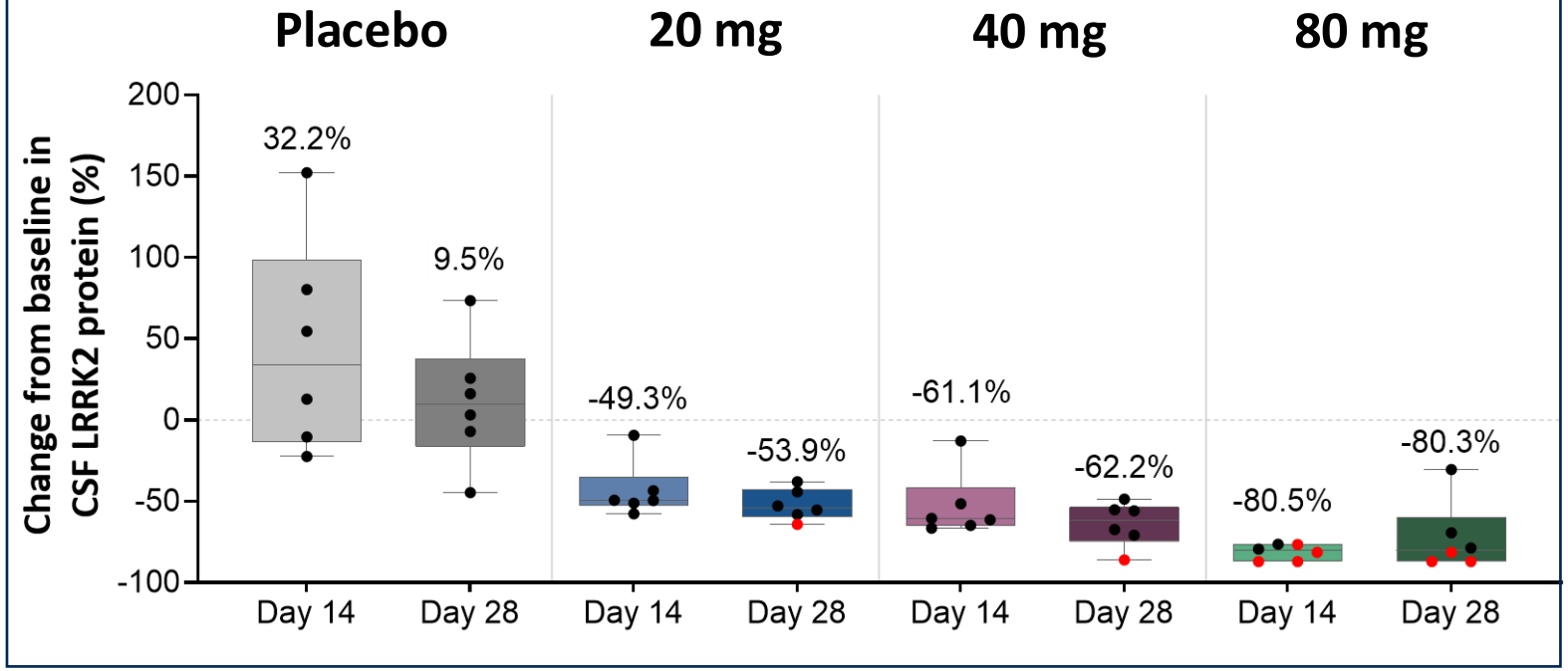
1. Smits L, et al. Oral presentation at International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders (AD/PD) 2025. 2. Smits L, et al. Poster presented at the International Congress of Parkinson's Disease and Movement Disorders (MDS) 2025; Poster #904.

ARV-102 achieved more than ~50% LRRK2 degradation at all dose levels in the CFS of patients with PD

Baseline LRRK2 in CSF[a]



Change in participants with Parkinson's disease[b]



- Baseline levels of LRRK2 in CSF were higher in participants with Parkinson's disease than in healthy volunteers
- Median LRRK2 reductions of ~50% or greater were achieved in participants with Parkinson's disease starting at daily ARV-102 doses of 20 mg

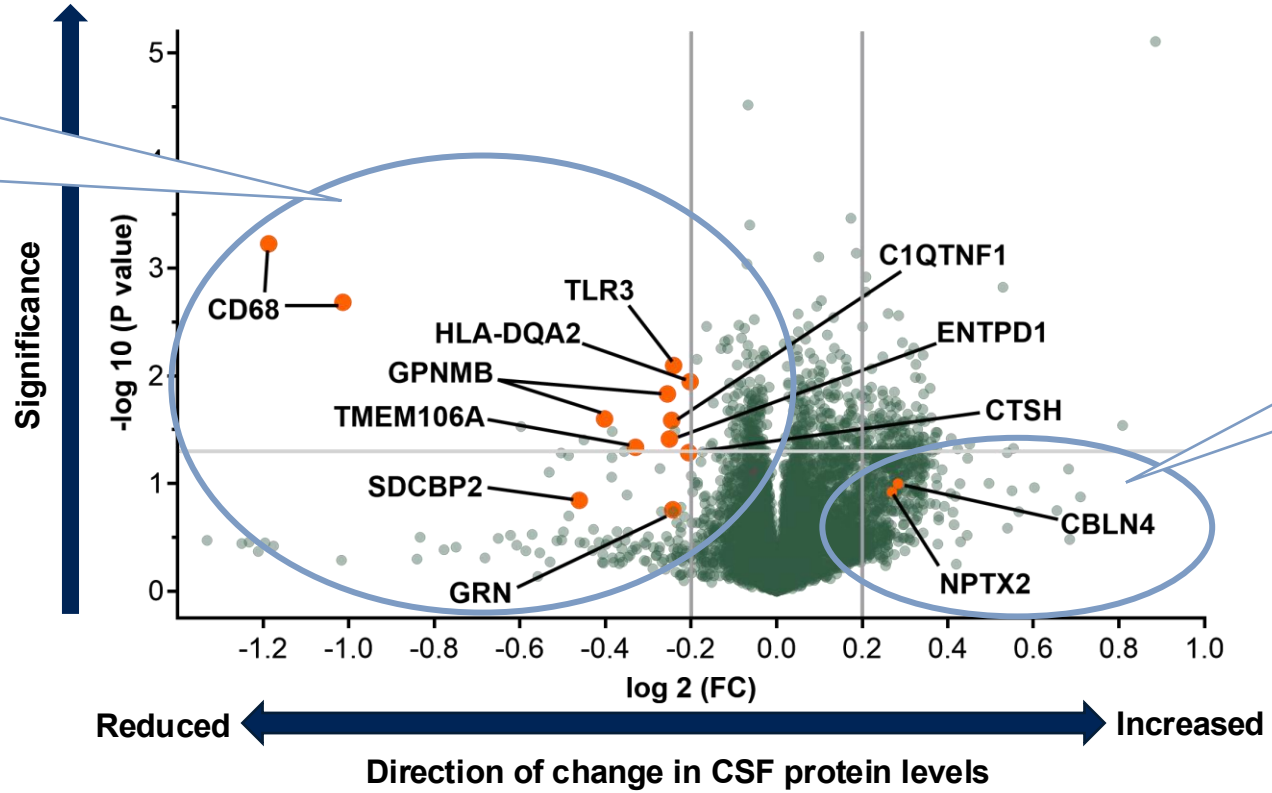
CSF, cerebrospinal fluid; LLD, lower limit of detection; LLOQ, lower limit of quantification; LRRK2, leucine-rich repeat kinase 2; QD, once daily.
Box plots show median and 25%/75% quartiles with whiskers to the minimum and maximum values. Circles indicate individual participant values.
a. Plot shows baseline total LRRK2 levels in CSF from all participants in the single- and multiple-dose arms of the phase 1 studies in healthy volunteers and participants with Parkinson's disease.
b. Red circles denote values that were <4 pg/mL (LLOQ) post baseline, which were calculated using one half of the LLOQ. Orange circles denote values that were below the LLOQ, but above the LLD (2 pg/mL); reported values were used to calculate change from baseline for these data points.

As presented at AD/PD 2026 International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders

ARV-102 reduced endolysosomal and neuroinflammatory pathway proteins that are elevated in LRRK2-related Parkinson's disease

ARV-102 (80mg QD) decreased CSF biomarkers of endolysosomal function and neuroinflammation (e.g., CD68, GPNMB) with increases in markers of synaptic integrity and axonal guidance (NPTX2, CBLN4)

Endolysosomal and neuroinflammatory proteins *Increased* in LRRK2 variant carriers and LRRK2-PD[3] *decreased* with ARV-102 treatment



Synaptic function proteins *increased* in PD *decreased* with ARV-102 treatment

C1QTNF1,complement C1q tumor necrosis factor-related protein 1; CBLN4, cerebellin 4 precursor; CD68, cluster of differentiation 68; CSF, cerebrospinal fluid; CTSH, cathepsin H; ENTPD1, ectonucleoside triphosphate diphosphohydrolase 1; GPNMB, glycoprotein nonmetastatic melanoma protein B; GRN, granulin precursor; HLA-DQA2, major histocompatibility complex, class II, DQ alpha 2; LRRK2, leucine-rich repeat kinase 2; NPTX2, neuronal pentraxin 2; QD, once daily; TLR3, toll-like receptor 3; TMEM106A, transmembrane protein 106A.

1. Smits L, et al. International Congress of Parkinson's Disease and Movement Disorders (MDS) 2025; Poster #904. 2. Van de Bult L, et al. International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders (AD/PD) 2026. 3. Phillips, B. et al. NPJ Parkinsons Dis. 2023 Jul 8;9(1):107.

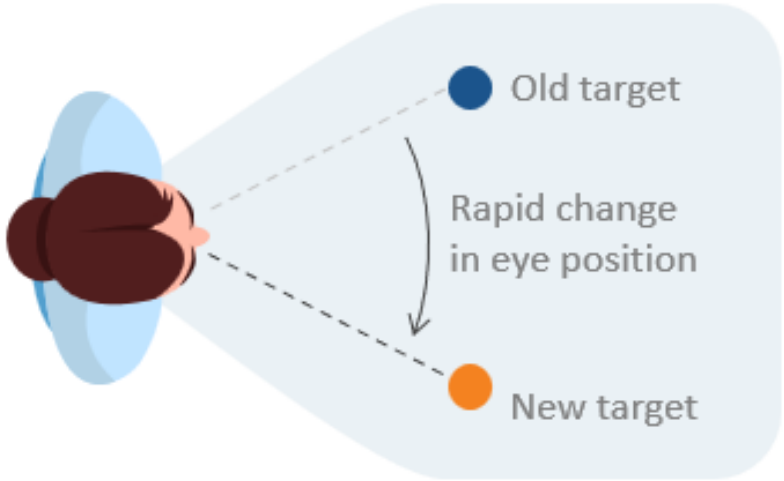
As presented at AD/PD 2026 ADVANCES IN SCIENCE & THERAPY International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders

ARV-102: Achieving biomarker advancements previously unreachable

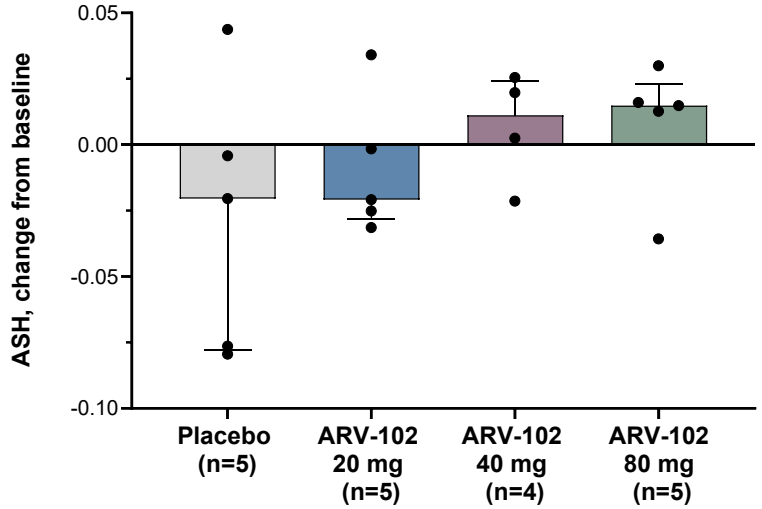
In a Phase 1 trial in patients with Parkinson's disease, decreases in CSF LRRK2 levels correlated with:

- Improvements in pathway measures of endolysosomal, neuroinflammatory, and synaptic function
- The magnitude of change in ASH, which is an eye movement abnormality associated with Parkinson's disease

Eye motor control test



ASH Change From Baseline to Day 28[a,1]



ASH, amplitude of saccadic hypometria; CSF, cerebrospinal fluid; LRRK2, leucine-rich repeat kinase 2; IQR, interquartile range.
a. Bars show medians ± IQRs; dots represent individual participant values.
1. K Fraser, et al. International Congress of Parkinson's Disease and Movement Disorders pometria; CSF, (MDS). Presented October 7, 2026.

Development plan for ARV-102 includes clinical trials in patients with progressive supranuclear palsy and Parkinson's disease

Status of Arvinas Trials with ARV-102

OBJECTIVES: Safety, Tolerability, PK, and PD

Phase 1: Healthy Volunteers (Complete)

Single Ascending Dose (Part A) Multiple Ascending Dose (Part B)

- ✓ First-in-human data presented during oral presentation at AD/PD (Q2 2025)
- ✓ Final data from HV SAD/MAD cohorts presented at MDS (Q3 2025)

Phase 1: Parkinson's Disease (Complete)

Single Ascending (Part A) and Multiple Dose (Part B)

- ✓ Presented initial single ascending dose data at (MDS Q3 2025)
- ✓ Presented multi-dose cohort during oral presentation at AD/PD (Q1 2026)

Planned trials in 2027 (pending regulatory approval)*

Progressive Supranuclear Palsy

Phase 1b: Multiple dose

- Initiate Phase 1b multi-dose cohort in patients with progressive supranuclear palsy in the EU and UK

AD/PD, International Conference on Alzheimer's and Parkinson's Diseases and related neurological disorders; MDS, Movement Disorders Society; PK; Pharmacokinetics; PD, pharmacodynamics; PSP, progressive supranuclear palsy

*Discussions with health authorities are ongoing and Arvinas plans to initiate clinical trials in 2027.

Multiple value-driving milestones anticipated ahead

Upcoming milestones: *Oncology*

ARV-393
Oral BCL6 degrader

- Present Phase 1 monotherapy dose escalation data from early cohorts (4Q 2026)
- Share additional Phase 1 monotherapy dose escalation data and data from the combination cohort with glofitamab (mid-2027)

ARV-6723
Oral HPK1 degrader

- Share initial Phase 1 clinical data in patients with advanced solid tumors (2H 2027)

Upcoming milestones: *Neurology*

ARV-027
Oral polyQ-AR degrader

- Share Phase 1 data from healthy volunteer cohort (mid-2027)
- Initiate enrollment in the Phase 1 SBMA patient cohort (1H 2027)

ARV-102
Oral LRRK2 degrader

- Initiate clinical trial in in patients with PSP (2027*)

Strong capital position with ~$559M cash on hand[a] and runway into second half 2028

The agents listed on this slide are investigational. Their safety and effectiveness for these investigational uses have not been established.
BCL6, B-cell lymphoma 6; DLBCL, diffuse large b-cell lymphoma; HPK1, hematopoietic progenitor kinase 1; LRRK2, leucine-rich repeat kinase 2; PD, Parkinson's disease; polyQ-AR, polyglutamine-expanded (polyQ) androgen receptor (AR); PSP, progressive supranuclear palsy.
a. Cash, cash equivalents, and marketable securities position as of September 30, 2026. *Pending CTA submission and regulatory approval.



ARVINAS

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NASDAQ LISTED ARVN

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