UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 3, 2026

Arvinas, Inc.
(Exact name of registrant as specified in its charter)

Delaware	001-38672	47-2566120
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

5 Science Park 395 Winchester Ave. New Haven, Connecticut	06511
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: (203) 535-1456

Not applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common stock, par value $0.001 per share	**ARVN**	**The Nasdaq Stock Market LLC**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 2.02 Results of Operations and Financial Condition.

On November 3, 2026, Arvinas, Inc. (the "Company") announced its financial results for the quarter ended September 30, 2026 and provided a corporate update. The full text of the press release issued in connection with the announcement is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Item 2.02 (including Exhibit 99.1 attached hereto) shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended (the "Securities Act"), or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 7.01 Regulation FD Disclosure.

On November 3, 2026, the Company updated its corporate presentation. The Company's corporate presentation will be posted in the Events and Presentations page under the Investors and Media section of the Company's website. A copy of the Company's presentation is also attached as Exhibit 99.2 and is incorporated herein by reference.

The information in this Item 7.01 (including Exhibit 99.2 attached hereto) shall not be deemed "filed" for purposes of Section 18 of the Exchange Act or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press Release issued by the Registrant on November 3, 2026
99.2	Company Presentation, dated November 2026
104	Cover Page Interactive Data File (formatted as Inline XBRL)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

ARVINAS, INC.

Date: November 3, 2026

By: /s/ Andrew Saik

Andrew Saik
Chief Financial Officer

Arvinas Reports Third Quarter 2026 Financial Results and Provides Corporate Update

– Initiated dosing in a Phase 1/2 trial of ARV-6723, an immuno-oncology oral PROTAC designed to degrade HPK1, a negative regulator of immune activation –

– Reaffirmed commitment to share clinical data from three Phase 1 programs - ARV-393, ARV-027 and ARV-6723 - in 4Q 2026 and 2027 –

– Demonstrated novel oculomotor and biomarker movement with ARV-102 Phase 1 clinical data in patients with Parkinson's disease with oral presentation at MDS conference –

– Company to host conference call today at 8:00 a.m. ET –

NEW HAVEN, Conn., November 3, 2026 — Arvinas, Inc. (Nasdaq: ARVN), a clinical-stage biotechnology company creating a new class of drugs based on targeted protein degradation, today reported financial results for the third quarter 2026, and provided a corporate update.

"We executed strongly during the quarter and are entering an important period of clinical milestones that we believe will demonstrate the promise of our clinical-stage protein degraders," said Randy Teel, Ph.D., President and Chief Executive Officer of Arvinas. "We are prioritizing investments where emerging evidence supports our goal of creating transformative medicines for patients. The upcoming data from our BCL6, polyQ-AR, and HPK1 degraders will begin to define the opportunity for these programs to change the treatment landscape, and we are grateful to the patients, investigators, and employees whose commitment makes this progress possible."

Third Quarter 2026 Business Highlights and Recent Developments

ARV-393: Oral PROTAC BCL6 degrader
- Submitted initial Phase 1 monotherapy dose escalation data for presentation at the 68th American Society of Hematology Annual Meeting (Dec. 12-15, 2026).
- Continued dose escalation in the Phase 1 clinical trial (ClinicalTrials.gov Identifier: NCT06393738) in patients with non-Hodgkin lymphoma (NHL).
    - Phase 1 trial evaluating ARV-393 as monotherapy in NHL and in combination with glofitamab in patients with diffuse large B-cell lymphoma (DLBCL).

ARV-027: Oral PROTAC polyQ-AR degrader
- Initiated enrollment in a multiple ascending dose cohort with healthy volunteers in the first-in-human Phase 1 clinical trial.

ARV-6723: Oral PROTAC HPK1 degrader
- Initiated dosing of patients in the monotherapy cohort of the Phase 1/2 clinical trial (ClinicalTrials.gov Identifier: NCT07749586) of ARV-6723, Arvinas' first immuno-oncology degrader.
    - Phase 1 trial will evaluate ARV-6723 as a monotherapy and in combination with pembrolizumab in patients with advanced solid tumors.

ARV-102: Oral PROTAC LRRK2 degrader

- Presented additional biomarker data from the Phase 1 clinical trial in patients with Parkinson's disease at the International Congress on Parkinson's Disease and Movement Disorders.
  - Data showed improvements in an objective measure of impaired eye movement associated with Parkinson's disease.
  - Changes in oculomotor function correlated with reductions in LRRK2 cerebrospinal fluid (CSF) biomarkers and changes in synaptic biomarkers.
- Announced Arvinas' intention to prioritize initiation of Phase 1b trial in progressive supranuclear palsy (PSP) in the European Union (EU) and United Kingdom (UK), pending clinical trial application (CTA) submission and regulatory authorization.
  - Discussion with the U.S. FDA and Japan's Pharmaceutical and Medical Device Agency are ongoing as part of Arvinas' global clinical strategy in PSP.
  - The investigational new drug application remains on hold in the U.S., with an objective to include U.S. sites in future trials after completing the Phase 1b clinical trial.

Anticipated Upcoming Milestones and Expectations

ARV-393: Oral PROTAC BCL6 degrader

- Present data from early monotherapy dose escalation cohorts that includes safety, pharmacokinetics (PK), and pharmacodynamics (PD) from all patients, with preliminary efficacy from T-cell lymphomas, in the ongoing Phase 1 clinical trial in patients with relapsed/refractory NHL (Q4 2026).
- Share additional monotherapy data from the ongoing Phase 1 dose escalation clinical trial in patients with B- and T-cell lymphomas (mid-2027).
- Share initial data from the combination cohort with glofitamab in patients with DLBCL in the ongoing Phase 1 clinical trial (mid-2027).

ARV-027: Oral PROTAC polyQ-AR degrader

- Share initial data from the Phase 1 clinical trial in healthy volunteers evaluating androgen receptor (AR)-degradation in muscle (1H 2027).
- Initiate dosing in cohort that includes patients with Spinal-Bulbar Muscular Atrophy (SBMA) (1H 2027).

ARV-6723: Oral PROTAC HPK1 degrader

- Continue enrollment in the monotherapy dose escalation cohort of the Phase 1 clinical trial in patients with advanced solid tumors.
  - Anticipate initiation of dosing patients in the combination cohort with pembrolizumab (mid-2027).
- Present new preclinical data demonstrating ARV-6723's superior activity and anti-tumor responses compared to immune checkpoint inhibitors in checkpoint-resistant models at the Society for Immunotherapy of Cancer Annual Meeting (Nov. 4-8, 2026).
- Present initial monotherapy data from the Phase 1 clinical trial (2H 2027).

ARV-102: Oral PROTAC LRRK2 degrader

- Submit clinical CTA to initiate Phase 1b trial in patients with PSP (EU 4Q 2026; UK 1Q 2027).
- Initiate Phase 1b clinical trial in patients with PSP in the EU and UK, pending regulatory authorization (2027).

Financial Guidance
Based on its current operating plan, Arvinas believes its cash, cash equivalents, restricted cash, and marketable securities as of September 30, 2026, is sufficient to fund planned operating expenses and capital expenditure requirements into the second half of 2028.

Third Quarter 2026 Financial Results
Cash, Cash Equivalents, Restricted Cash, and Marketable Securities Position: As of September 30, 2026, cash, cash equivalents, restricted cash, and marketable securities were $558.9 million compared with $685.4 million as of December 31, 2025. The decrease in cash, cash equivalents, restricted cash, and marketable securities of $126.5 million for the nine months ended September 30, 2026 was primarily related to cash used in operations of $123.0 million (net of $35.0 million received under the Rigel License Agreement), unrealized losses on marketable securities of $2.9 million, and the purchase of lab equipment and leasehold improvements of $1.3 million, partially offset by proceeds from the exercise of stock options and issuance of shares under the ESPP of $0.9 million.

Research and Development Expenses: Generally Accepted Accounting Principles (GAAP) research and development (R&D) expenses were $46.9 million for the quarter ended September 30, 2026, compared with $64.7 million for the quarter ended September 30, 2025. The decrease in R&D expenses of $17.8 million for the quarter was primarily due to a decrease in external expenses of $10.5 million, and a decrease in compensation and related personnel expenses of $6.5 million, which are not allocated by program. External expenses include (i) program-specific expenses, which decreased by $6.7 million, primarily driven by a decrease in the vepdegestrant (ARV-471) program of $14.2 million, partially offset by increases in the ARV-027, ARV-6723, and ARV-393 programs of $4.6 million, $3.3 million, and $3.2 million, respectively, and (ii) non-program specific expenses, which decreased by $3.8 million.

Non-GAAP R&D expenses were $44.3 million for the quarter ended September 30, 2026, compared with $56.9 million for the quarter ended September 30, 2025, excluding zero and $0.4 million of restructuring expense for the quarters ended September 30, 2026 and 2025, respectively, and $2.6 million and $7.4 million of non-cash stock-based compensation expense for the quarters ended September 30, 2026 and 2025, respectively. A reconciliation of GAAP to non-GAAP financial measures used in this press release can be found at the end of this press release.

General and Administrative Expenses: GAAP general and administrative (G&A) expenses were $12.6 million for the quarter ended September 30, 2026, compared with $21.0 million for the quarter ended September 30, 2025. The decrease in G&A expenses of $8.4 million for the quarter was primarily due to decreases in personnel and infrastructure related costs of $4.2 million, costs related to developing commercial operations of $1.8 million, and professional fees of $1.5 million.

Non-GAAP G&A expenses were $9.2 million for the quarter ended September 30, 2026, compared with $14.6 million for the quarter ended September 30, 2025, excluding zero and $0.6 million of restructuring related reversal of previously recognized expense for the quarters ended

September 30, 2026 and 2025, respectively, and $3.4 million and $7.0 million of non-cash stock-based compensation expense for the quarters ended September 30, 2026 and 2025, respectively. A reconciliation of GAAP to non-GAAP financial measures used in this press release can be found at the end of this press release.

Revenue: Revenue was $0.5 million for the quarter ended September 30, 2026, compared with $41.9 million for the quarter ended September 30, 2025. Revenue for the quarter ended September 30, 2026 consisted of sales-based royalties from the Rigel License Agreement. Revenue for the quarter ended September 30, 2025 primarily reflected $20.6 million under the Original Vepdegestrant (ARV-471) Collaboration Agreement with Pfizer and a $20.0 million development milestone related to the Novartis License Agreement. Revenue from the Original Vepdegestrant (ARV-471) Collaboration Agreement with Pfizer did not recur following the recognition of all remaining deferred revenue in the second quarter of 2026 upon entry into the Rigel License Agreement.

Cost of License Revenue: Cost of license revenue was $0.1 million for the quarter ended September 30, 2026, compared with zero for the quarter ended September 30, 2025. The expense arose under the Amended Yale License Agreement related to royalties under the Rigel License Agreement.

Investor Call & Webcast Details
Arvinas will host a conference call and webcast today, November 3, 2026, at 8:00 a.m. ET to review its third quarter 2026 financial results and discuss recent corporate updates. Participants are invited to listen by going to the Events and Presentation section under the Investors page on the Arvinas website at www.arvinas.com. A replay of the webcast will be available on the Arvinas website following the completion of the event and will be archived for up to 30 days.

About Arvinas
Arvinas (Nasdaq: ARVN) is a clinical-stage biotechnology company dedicated to improving the lives of patients suffering from debilitating and life-threatening diseases. Through its PROTAC (PROteolysis TArgeting Chimera) protein degrader platform, Arvinas is pioneering the development of protein degradation therapies designed to harness the body's natural protein disposal system to selectively and efficiently degrade and remove disease-causing proteins. Arvinas, with its partner Pfizer, developed the first U.S. Food and Drug Administration (FDA) approved PROTAC, a type of heterobifunctional protein degrader, which has been outlicensed to Rigel Pharmaceuticals, Inc. for exclusive global development, manufacturing, and commercialization.

Arvinas is currently progressing multiple investigational drugs through clinical development programs, including ARV-393, targeting BCL6 for relapsed/refractory non-Hodgkin Lymphoma; ARV-102, targeting LRRK2 for neurodegenerative disorders; ARV-027, targeting the polyglutamine-expanded androgen receptor, or polyQ-AR, in skeletal muscle for the treatment of Spinal-Bulbar Muscular Atrophy, also known as Kennedy's disease; and ARV-6723, targeting HPK1 for advanced solid tumors. Arvinas has also advanced ARV-806, targeting KRAS G12D for solid tumors, in the clinic, and previously announced plans to seek an out-licensing agreement for any additional clinical trials of ARV-806, including dose expansion or combination clinical trials. Arvinas is headquartered in New Haven, Connecticut. For more information about Arvinas, visit www.arvinas.com and connect on **LinkedIn** and **X**.

About ARV-393
ARV-393 is an investigational, orally bioavailable PROTAC designed to specifically target and degrade B-cell lymphoma 6 protein (BCL6), a transcriptional repressor and major driver of B-cell lymphomas. During B-cell development, tightly controlled BCL6 protein expression regulates >600 genes to facilitate rapid B-cell proliferation and tolerance of somatic hypermutation and gene recombination for antibody generation. Deregulated BCL6 expression is common in B-cell lymphoma and promotes cancer cell survival, proliferation, and genomic instability. PROTAC-mediated degradation has the potential to address the historically undruggable nature of BCL6. ARV-393 is currently being evaluated in a Phase 1 clinical trial as a monotherapy in patients with relapsed/refractory non-Hodgkin lymphoma and in in combination with glofitamab as a chemotherapy-free combination approach in patients with DLBCL.

About ARV-027
ARV-027 is an oral, peripherally restricted investigational PROTAC degrader designed to selectively target and eliminate the polyglutamine-expanded androgen receptor (polyQ-AR) in skeletal muscle. ARV-027 is a clinical candidate specifically selected for potent in vitro reduction of cytosolic and nuclear polyQ-AR and for favorable skeletal-muscle exposure following oral administration. The polyQ-AR protein is the pathogenic driver of spinal and bulbar muscular atrophy (SBMA), a rare, X-linked, genetically defined neuromuscular disease caused by a CAG trinucleotide repeat expansion in the androgen receptor (AR) gene. SBMA leads to progressive muscle weakness, dysphagia, and functional decline, and currently has no approved disease-modifying therapies approved by the FDA or EMA, representing a significant unmet medical need. ARV-027 is currently being evaluated in a fist-in-human Phase 1 clinical trial in healthy volunteers.

About ARV-6723
ARV-6723 is an investigational, oral PROTAC designed to degrade hematopoietic progenitor kinase 1 (HPK1) and is Arvinas' first clinical candidate in the immuno-oncology space. HPK1 is a negative regulator of immune activation expressed across multiple immune cell types, including T cells, B cells, natural killer cells, and dendritic cells. Preclinically, ARV-6723 demonstrated potent, selective HPK1 degradation and greater tumor growth inhibition across low- and high-immunogenic tumor models, including anti-PD-1 resistant tumor models. By removing HPK1, ARV-6723 may address both its kinase-dependent and kinase-independent functions and enhance antitumor immune activity. ARV-6723 is currently being evaluated in a first-in-human Phase 1/2 clinical trial in patients with advanced solid tumors.

About ARV-102
ARV-102 is an investigational, orally bioavailable PROteolysis TArgeting Chimera (PROTAC) designed to cross the blood-brain barrier and specifically target and degrade leucine-rich repeat kinase (LRRK2), a large, multidomain scaffolding kinase with GTPase activity. Increased activity and over expression of LRRK2 have been implicated in the pathogenesis of neurological diseases, including LRRK2 genetic and idiopathic Parkinson's disease and progressive supranuclear palsy (PSP). ARV-102 has been evaluated in a Phase 1 clinical trial in healthy volunteers and in patients with Parkinson's disease.

Non-GAAP Financial Information
The results presented in this press release include both Generally Accepted Accounting Principles (GAAP) information and non-GAAP information. As used in this release, non-GAAP research and

development (“R&D”) expense is defined by Arvinas as GAAP R&D expense excluding restructuring and stock-based compensation expense, and non-GAAP general and administrative (G&A) expense is defined by Arvinas as GAAP G&A expense excluding restructuring and stock-based compensation expense. Arvinas uses these non-GAAP financial measures to evaluate Arvinas’ ongoing operations and for internal planning and forecasting purposes. Arvinas believes that non-GAAP financial information, when taken collectively, may be helpful to investors because it provides consistency and comparability with past financial performance. However, non-GAAP financial information is presented for supplemental informational purposes only, has limitations as an analytical tool, and should not be considered in isolation or as a substitute for financial information presented in accordance with GAAP. Other companies, including companies in Arvinas’ industry, may calculate similarly titled non-GAAP measures differently or may use other measures to evaluate their performance, all of which could reduce the usefulness of Arvinas’ non-GAAP financial measures as tools for comparison. Investors are encouraged to review the related GAAP financial measures and the reconciliation of these non-GAAP financial measures to their most directly comparable GAAP financial measures and not rely on any single financial measure to evaluate Arvinas’ business

Forward-Looking Statements
This press release 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: Arvinas’ belief that key clinical milestones will demonstrate the promise of its clinical-stage protein degraders; the potential to deliver transformative medicines for patients; that the upcoming data from Arvinas’ BCL6, polyQ-AR, and HPK1 degraders will begin to define the opportunity for these programs to change the treatment landscape; Arvinas’ plans to share clinical data from ARV-393, ARV-027 and ARV-6723 in the fourth quarter of 2026 and in 2027; the therapeutic potential or potential benefits of Arvinas’ product candidates and potential of its pipeline; Arvinas’ anticipated milestones, expectations and plans with respect to ARV-393, ARV-027, ARV-6723, and ARV-102, including timings related to anticipated enrollment or initiation of trials and sharing or presentation of data as well as forums for presenting any such data; Arvinas’ plans to seek an out-license of ARV-806 for further development, including any dose expansion or combination clinical trials; statements regarding Arvinas’ cash, cash equivalents and marketable securities, including their sufficiency to fund planned operating expenses and capital expenditure requirements into the second half of 2028; and Arvinas’ belief that non-GAAP financial information, when taken collectively, may be helpful to investors because it provides consistency and comparability with past financial performance. All statements, other than statements of historical fact, contained in this press release, including statements regarding Arvinas’ 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,” “goal,” “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.

Arvinas may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on such 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 and preclinical studies on its expected timelines or at all; Rigel's performance of its obligations under the license agreement with Arvinas and Pfizer Inc.; the potential demand and market potential and acceptance of any of Arvinas' product candidates, including estimates regarding the potential market opportunity; the competitive landscape for Arvinas' product candidates; risks related to Arvinas' expectations regarding the potential clinical benefit of its product candidates; the uncertainties inherent in research and development, including preclinical study or clinical trial results; risks and uncertainties relating to regulatory applications and related approval timelines; 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; Arvinas' reliance on third parties; early termination of any of Arvinas' collaborations; 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 discussed in the "Risk Factors" section of Arvinas' Annual Report on Form 10-K for the year ended December 31, 2025 and subsequent other reports on file with the U.S. Securities and Exchange Commission. The forward-looking statements contained in this press release reflect Arvinas' current views 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 release.

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Arvinas, Inc.

Condensed Consolidated Balance Sheets (Unaudited)

(dollars and shares in millions, except per share amounts)	September 30, 2026	December 31, 2025
Assets		
Current assets:		
Cash and cash equivalents	$ 88.0	$ 142.9
Marketable securities	470.4	542.5
Accounts receivable	1.8	1.0
Other receivables	3.5	5.4
Prepaid expenses and other current assets	23.9	8.9
Total current assets	587.6	700.7
Property, equipment and leasehold improvements, net	4.6	5.2
Operating lease right-of-use assets	6.8	8.2
Contract and other assets	8.5	3.8
Total assets	$ 607.5	$ 717.9
Liabilities and stockholders' equity		
Current liabilities:		
Accounts payable and accrued liabilities	$ 39.9	$ 69.5
Deferred revenue	—	71.3
Current portion of collaboration liability	28.2	—
Current portion of operating lease liabilities	1.9	1.7
Total current liabilities	70.0	142.5
Deferred revenue	—	134.3
Collaboration liability	18.2	—
Long-term debt	0.2	0.4
Operating lease liabilities	5.3	6.8
Total liabilities	93.7	284.0
Stockholders' equity:		
Preferred stock, $0.001 par value, zero shares issued and outstanding as of September 30, 2026 and December 31, 2025, respectively	—	—
Common stock, $0.001 par value; 75.5 shares issued and 65.5 shares outstanding as of September 30, 2026, and 73.5 shares issued and 63.5 outstanding as of December 31, 2025	0.1	0.1
Accumulated deficit	(1,554.5)	(1,612.4)
Additional paid-in capital	2,161.8	2,136.9
Accumulated other comprehensive (loss) income	(1.7)	1.2
Treasury Stock, at cost (10.0 shares as of September 30, 2026 and December 31, 2025)	(91.9)	(91.9)
Total stockholders' equity	513.8	433.9
Total liabilities and stockholders' equity	$ 607.5	$ 717.9

Arvinas, Inc.

Condensed Consolidated Statements of Operations (Unaudited)

(dollars and shares in millions, except per share amounts)	For the Three Months Ended September 30,		For the Nine Months Ended September 30,	
	2026	**2025**	**2026**	**2025**
Revenue	$ 0.5	$ 41.9	$ 265.8	$ 253.1
Operating expenses:				
Cost of license revenue	0.1	—	9.1	—
Research and development	46.9	64.7	159.8	224.1
General and administrative	12.6	21.0	55.7	72.9
Total operating expenses	59.6	85.7	224.6	297.0
(Loss) income from operations	(59.1)	(43.8)	41.2	(43.9)
Interest and other income	5.4	9.0	17.2	30.6
Net (loss) income before income taxes	(53.7)	(34.8)	58.4	(13.3)
Income tax expense	(0.2)	(0.3)	(0.5)	(0.1)
Net (loss) income	$ (53.9)	$ (35.1)	$ 57.9	$ (13.4)
(Loss) earnings per common share				
Basic	$ (0.82)	$ (0.48)	$ 0.89	$ (0.18)
Diluted	$ (0.82)	$ (0.48)	$ 0.88	$ (0.18)
Weighted average common shares outstanding				
Basic	65.4	73.2	64.7	72.9
Diluted	65.4	73.2	65.7	72.9

Arvinas, Inc.

Reconciliation of GAAP to Non-GAAP Information

(dollars in millions)	For the Three Months Ended September 30,		For the Nine Months Ended September 30,	
	2026	**2025**	**2026**	**2025**
Research and development reconciliation				
GAAP research and development expenses	$ 46.9	$ 64.7	$ 159.8	$ 224.1
Less: restructuring expense	—	0.4	0.6	1.0
Less: stock-based compensation expense (*)	2.6	7.4	9.3	27.4
Non-GAAP research and development expenses	$ 44.3	$ 56.9	$ 149.9	$ 195.7
General and administrative reconciliation				
GAAP general and administrative expenses	$ 12.6	$ 21.0	$ 55.7	$ 72.9
Less: restructuring expense	—	(0.6)	2.1	(0.3)
Less: stock-based compensation expense (*)	3.4	7.0	12.9	17.3
Non-GAAP general and administrative expenses	$ 9.2	$ 14.6	$ 40.7	$ 55.9

(*) Excludes restructuring related stock-based compensation.

Exhibit 99.2


ARVINAS
Corporate Presentation
November 2026
NASDAQ
LISTED
ARVN
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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 TArgeting 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

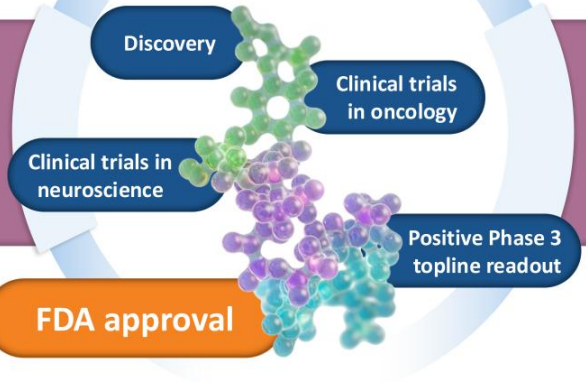
Our experienced and talented team is advancing a new therapeutic modality for patients

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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

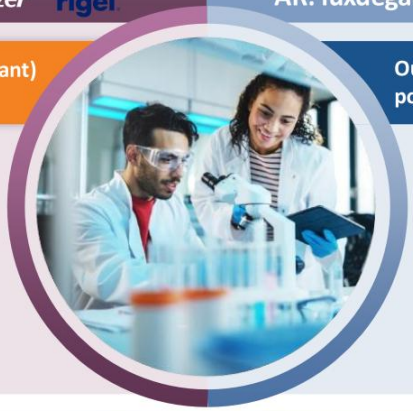
ER and AR degraders highlight Arvinas' ability to develop valuable PROTAC degraders

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ER: VEPPANU[a] (Pfizer, rigel)

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 (NOVARTIS)

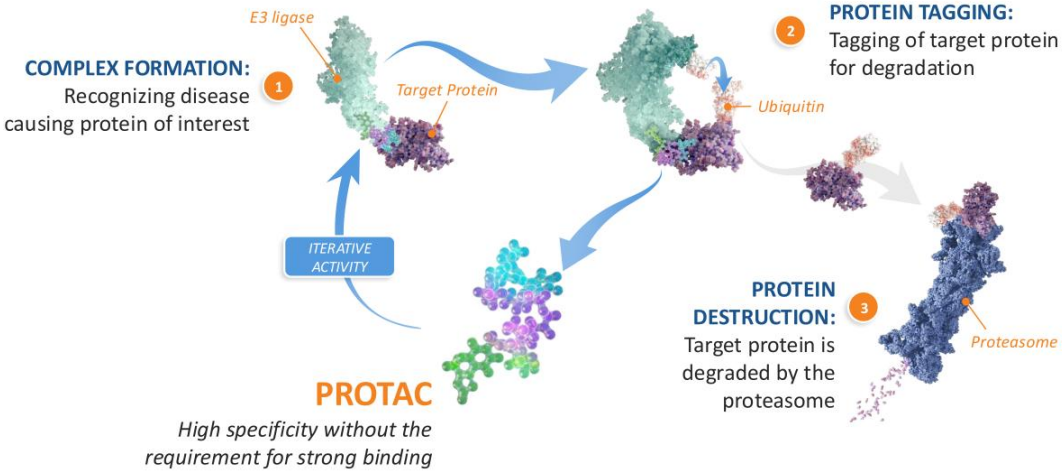
Out-licensed to Novartis in 2024 with potential for more than $1B in total value

- 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

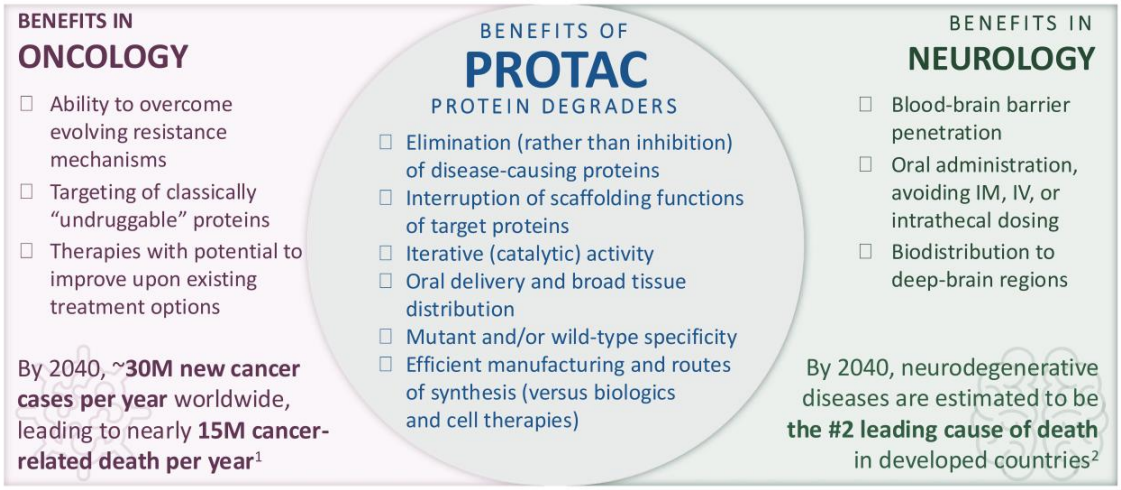
AR, androgen receptor; ER, estrogen receptor; *ESR1*, estrogen receptor 1 gene; ER+/HER2-, estrogen receptor-positive/human epidermal growth factor receptor 2-negative; FDA, Food and Drug Administration; mCRC, metastatic castrate resistant prostate cancer; mHSPC, metastatic hormone-sensitive prostate 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



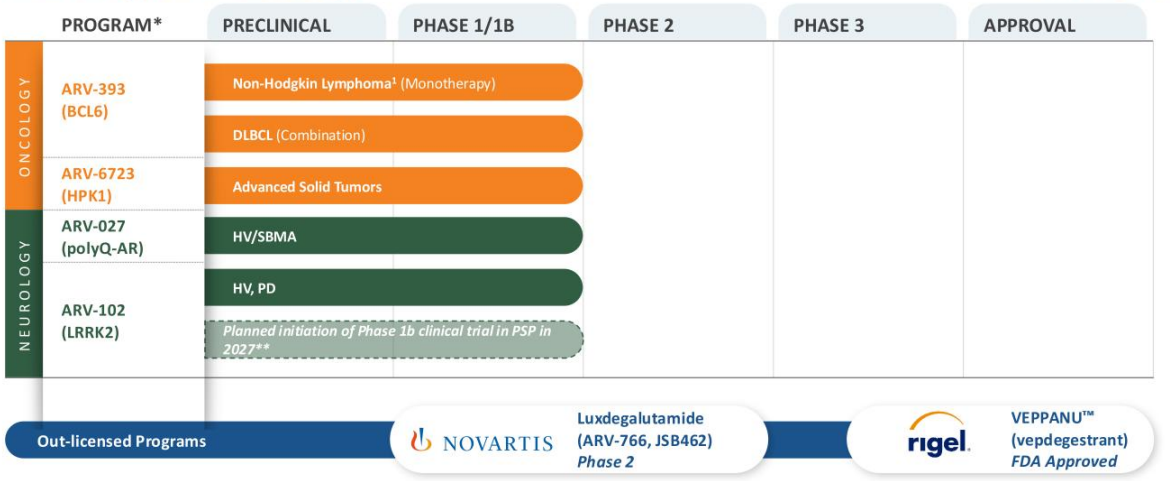
PROTAC degraders have potential benefits that may provide advantages over other modalities in oncology and neurology

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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).

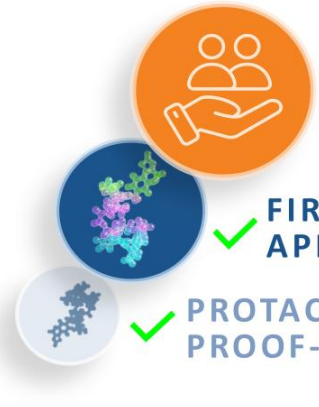
Arvinas' pipeline includes differentiated PROTAC degraders in neurology and oncology



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

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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

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

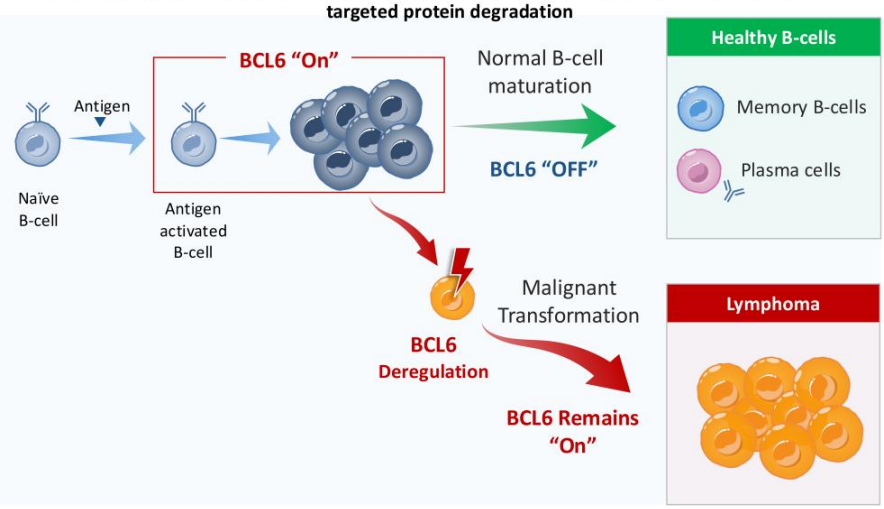
Large B-Cell Lymphoma (LBCL)	• Although a high proportion of patients achieve complete remission, therapy resistance or relapse still occurs in 30-40% of patients, thus evidencing a need for new therapeutic options[1] • Deregulation of BCL6 expression and/or functions are common in B-cell lymphomas[2]
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



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.

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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]

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

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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**

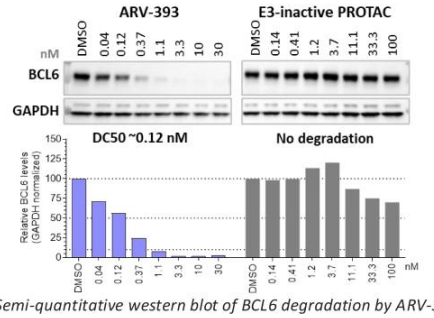
Phase 1 dose escalation trial of ARV-393 as monotherapy for R/R NHL, and in combination with glofitamab for R/R DLBCL, is currently enrolling patients (ClinicalTrials.gov ID: NCT06393738)

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

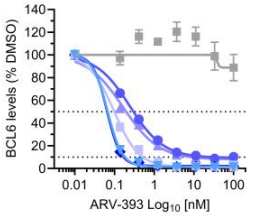
ARV-393, but not its E3-inactive analogue, robustly degrades BCL6 in the OCI-Ly1 model of DLBCL



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

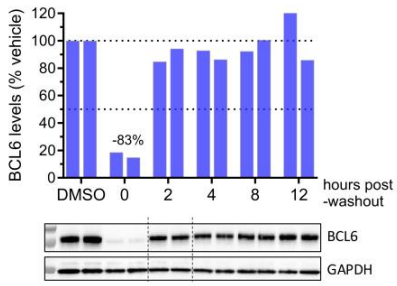


	DC_{50} [nM]	D_{max}
OCI-Ly1	0.07	99%
Farage	0.06	98%
SU-DHL-4	0.33	91%
SU-DHL-6	0.21	92%
OCI-Ly7	0.10	99%
OCI-Ly1 (E3-inactive ARV-393 analogue)	-	-

BCL6, B cell lymphoma 6; DLBCL, diffuse large b-cell lymphoma
Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.

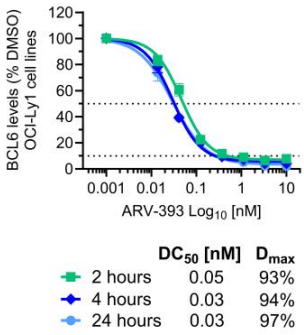
Preclinically, ARV-393 rapidly degrades BCL6 and overcomes its high resynthesis rate that drives tumor cell growth

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

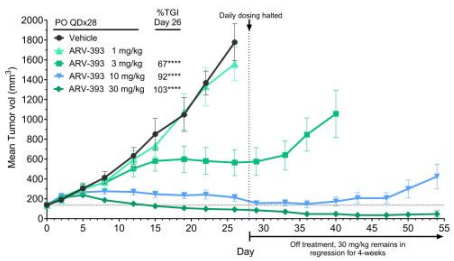


	DC_{50} [nM]	D_{max}
2 hours	0.05	93%
4 hours	0.03	94%
24 hours	0.03	97%

BCL6, B cell lymphoma 6
Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.

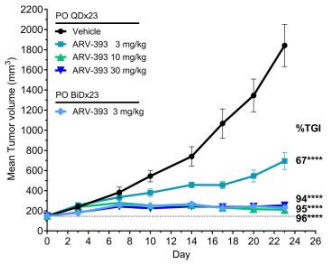
ARV-393 induces dose-dependent and sustained tumor growth inhibition and regressions *in vivo*

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)

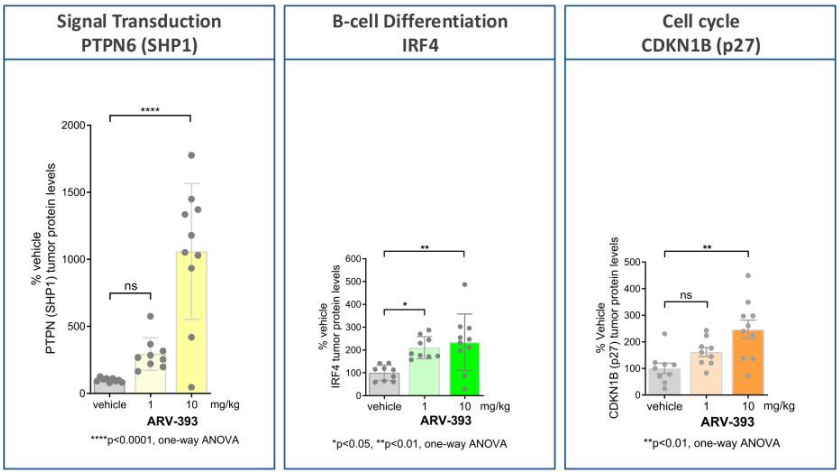
ARV-393 demonstrated >90% TGI



- 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

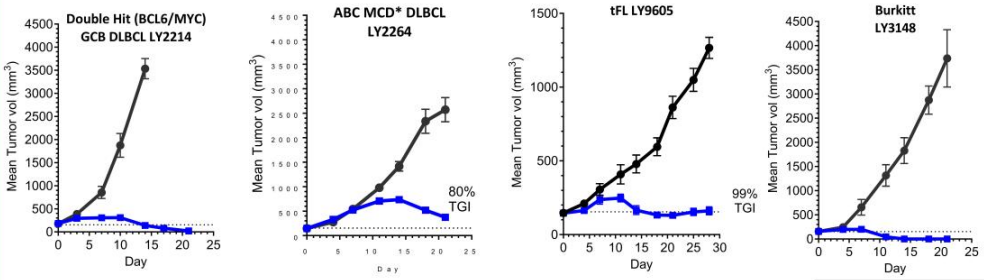
BCL6, B cell lymphoma 6; CDKN1B, Cyclin Dependent Kinase Inhibitor 1B; CDX, cell derived xenograft; IRF4, Interferon Regulatory Factor 4; PTPN6, Protein Tyrosine Phosphatase Non-Receptor Type 6
Gough et al., European Hematology Association (EHA) Poster P1256. June 2024, Madrid Spain.
Statistics performed using one-way ANOVA with Dunnett's multiple comparison test.

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- **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

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

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



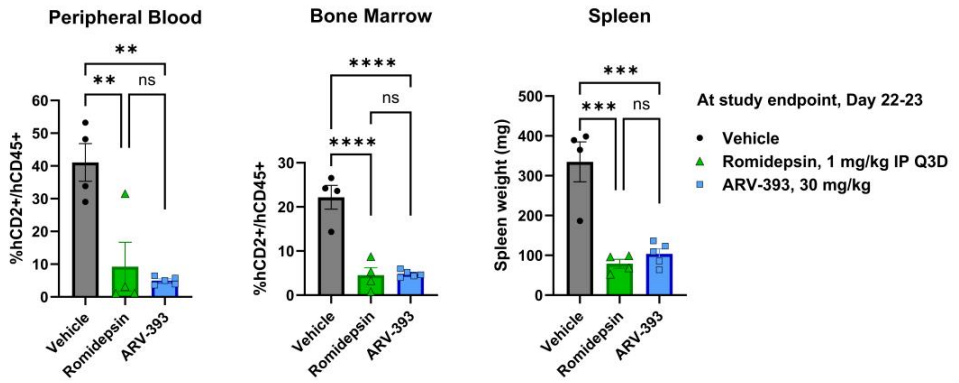
Similar results seen in fifteen PDX models of various NHL subtypes

4 mice/group, PO QDx21
- Vehicle
- ARV-393 30 mg/kg

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

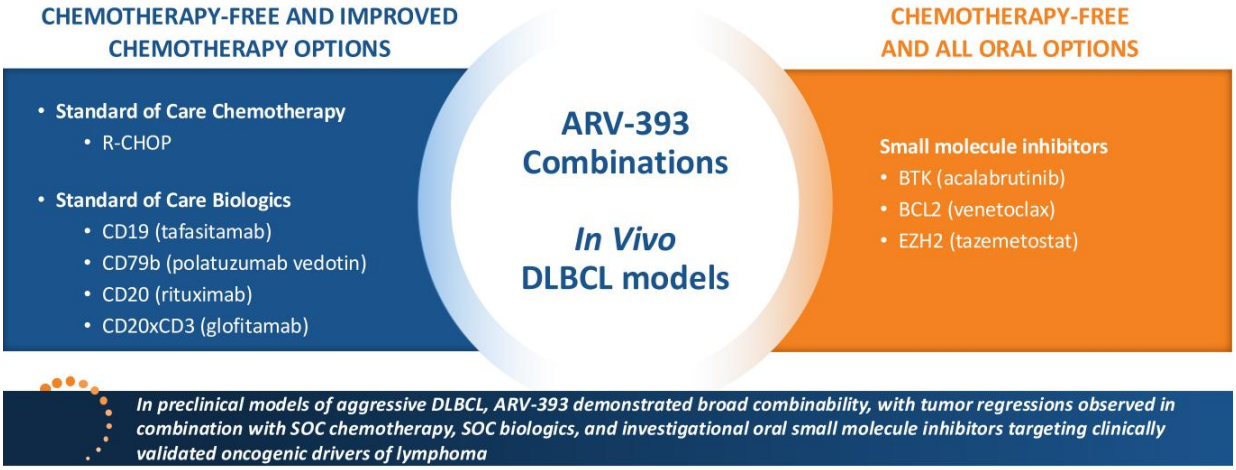
ARV-393 demonstrates evidence of significant efficacy (comparable to SOC romidepsin) in a chemo (CHOP) relapsed AITL PDX model



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

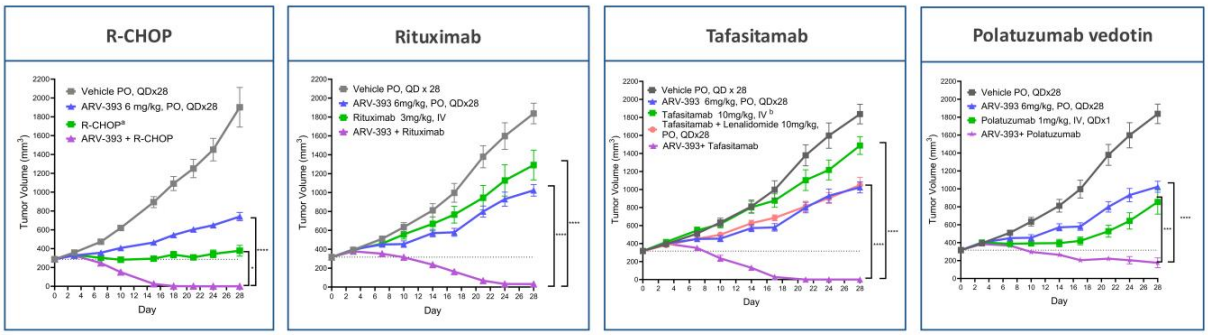
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In preclinical models of aggressive DLBCL, ARV-393 demonstrated broad combinability, with tumor regressions observed in combination with SOC chemotherapy, SOC biologics, and investigational oral small molecule inhibitors targeting clinically validated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

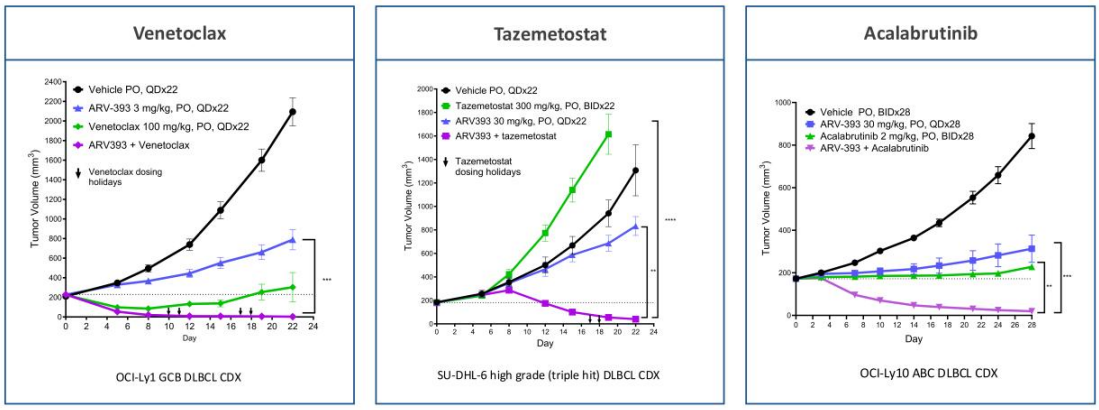
ARV-393, in combination with SOC chemotherapy/biologics, drives tumor regressions, including complete responses, in a preclinical model of aggressive DLBCL



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

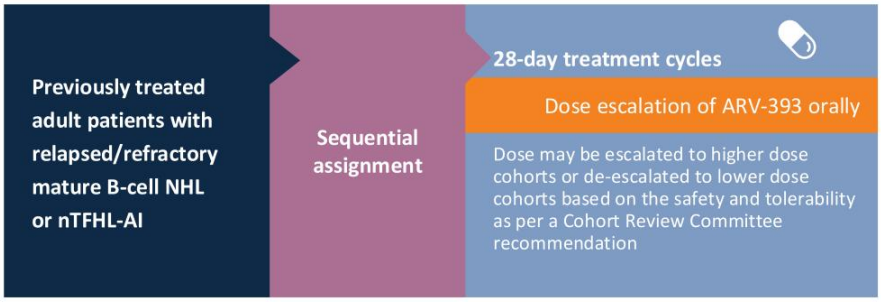
CDX, cell-derived xenograft; DLBCL, diffuse large B-cell lymphoma; IV, Intravenous; PO, oral; QD, once a day; R-CHOP, rituximab, cyclophosphamide, hydroxydaunorubicin, vincristine sulfate, and prednisone; SOC, standard of care
a. Rituximab 3 mg/kg was administered intravenously (IV) on days 1, 8, 15, 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



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)

*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.

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- **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)**



CLINICAL PROGRAMS: Neurology

ARV-027

PROTAC polyQ-AR degrader

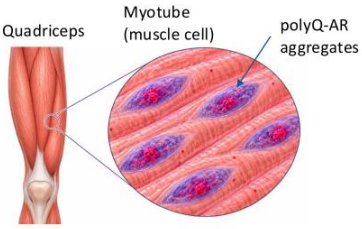
ARVINAS

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)

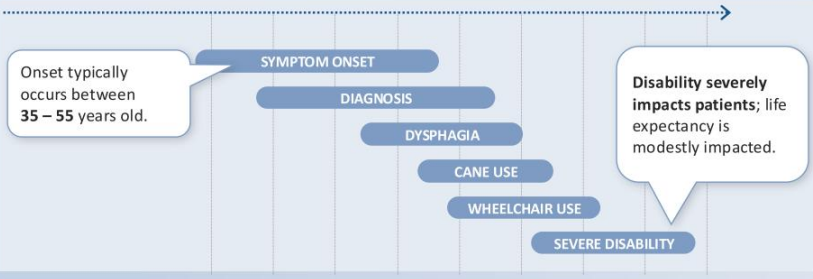
ARVINAS

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]

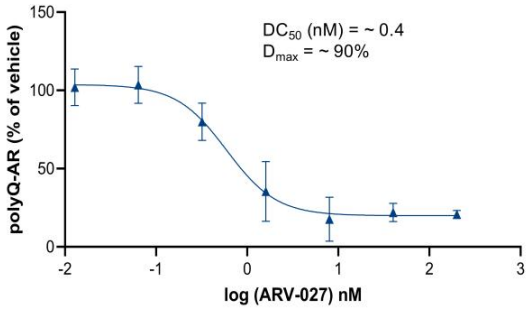
Spinal-bulbar muscular atrophy (SBMA), also known as Kennedy's disease, is a rare, X-linked, neuromuscular disease caused by a repeat expansion in the androgen receptor gene that encodes polyQ-AR.

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.

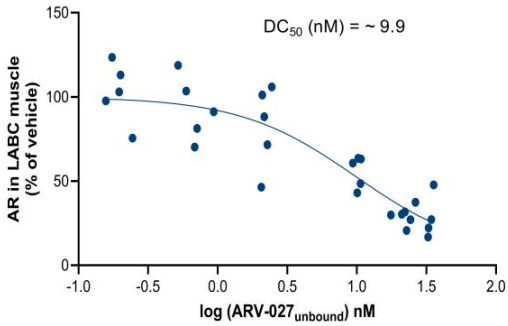
ARV-027 is a potent *in vitro* and *in vivo* PROTAC degrader of polyQ-AR, the root cause of SBMA[1]

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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

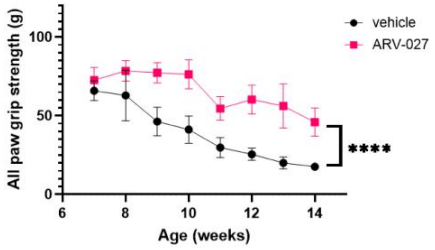


The mechanism of the polyQ-AR degradation was confirmed to be E3 ligase and proteosome dependent (data not shown)

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

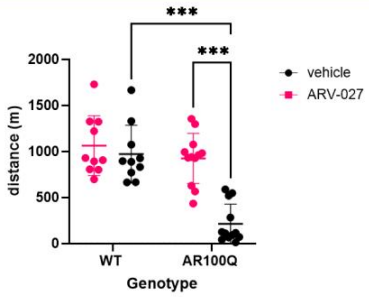
In preclinical models, ARV-027 induced polyQ-AR degradation in muscle tissue and rescued strength and endurance in a rapidly progressing model of SBMA[a]

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

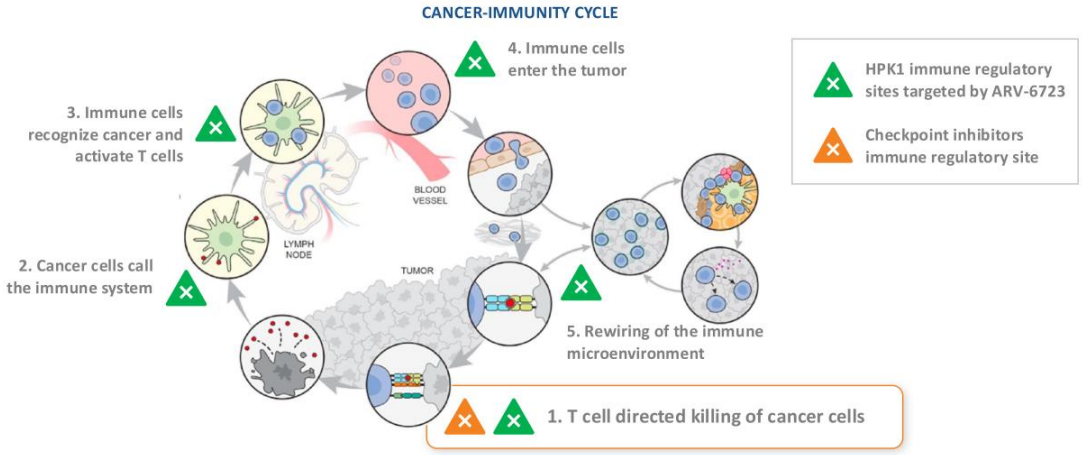
PROTAC HPK1 degrader

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

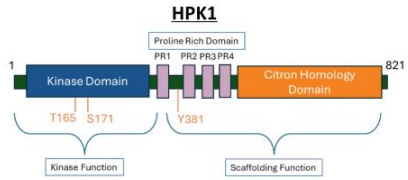
ARVINAS



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.

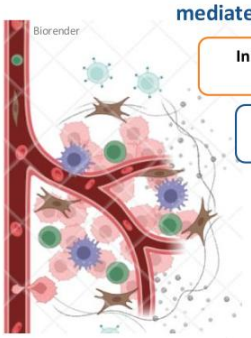
ARV-6723 is a potent PROTAC degrader of HPK1, a central regulator of immune suppression



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

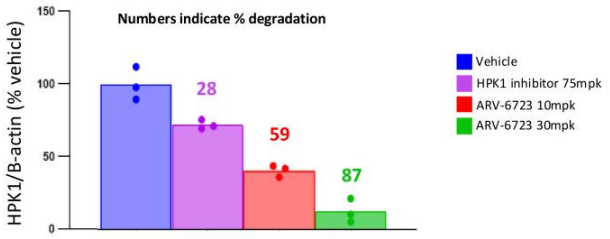
ARV-6723, an oral HPK1 degrader, could offer sustained anti-tumor immune response as a single agent or in combination with SoC (e.g., anti-PD1, chemotherapy), resulting in improved clinical benefits over SoC across a wide range of cancers

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

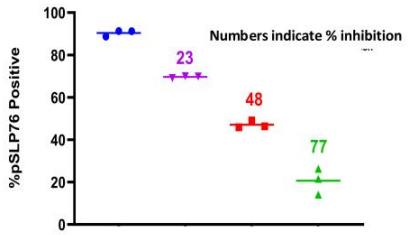
Single dose of oral ARV-6723 showed profound and sustained target degradation and durable pathway engagement in preclinical models

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HPK1 Degradation



Downstream Pathway Inhibition (pSLP76)



24h post oral dose

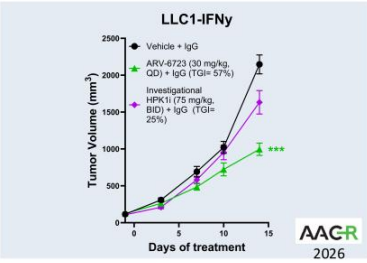
- ARV-6723 is a potent single-digit nanomolar degrader of HPK1, leading to sustained downstream pathway inhibition (via pSLP76) and induction of IL-2 release (data on file)
- This enhanced PK/PD profile results in more complete *in vivo* pathway suppression that recapitulates a genetic KO phenotype upon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

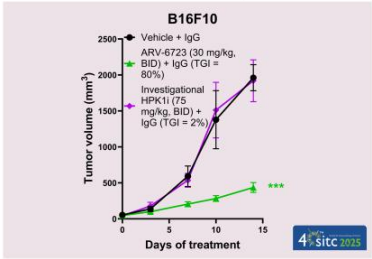
As presented at 4 sitc 2025

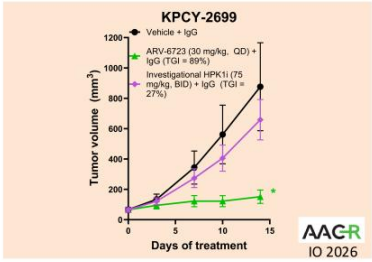
HPK1, hematopoietic progenitor kinase 1; KO, knock out; mpk, milligrams per kilogram; PK/PD, pharmacokinetic/pharmacodynamic; pSLP76, SH2 domain-containing leukocyte protein of 76 kDa, phosphorylated.

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)

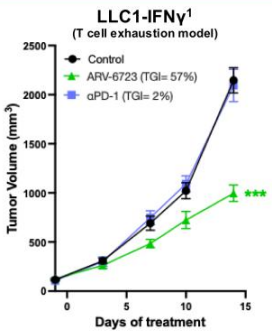
ARV-6723 has significant single agent anti-tumor activity in 7 ICI-resistant models (3 models shown)

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

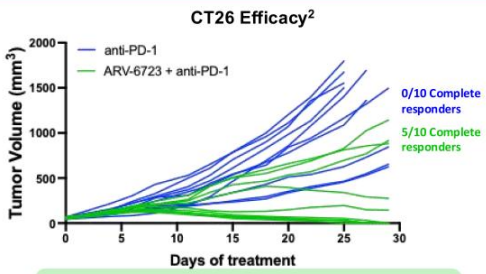
Preclinical work suggests that an HPK1 degrader could work after αPD-1 progression, in combination with αPD-1, and ahead of αPD-1

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

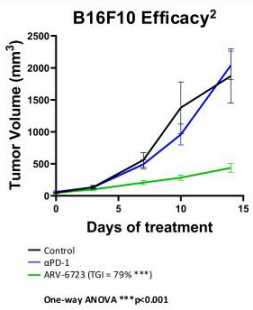


ARV-6723 combines with αPD-1 to improve preclinical outcomes



Evidence of immunological memory demonstrated in the 5 complete responders

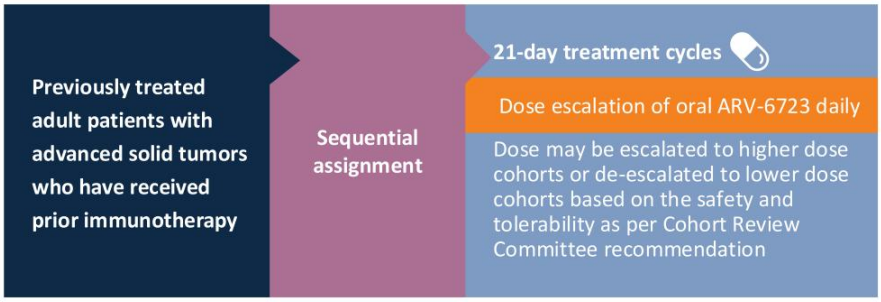
ARV-6723 inhibits tumor growth in a low-immunogenic model where αPD-1 fails



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

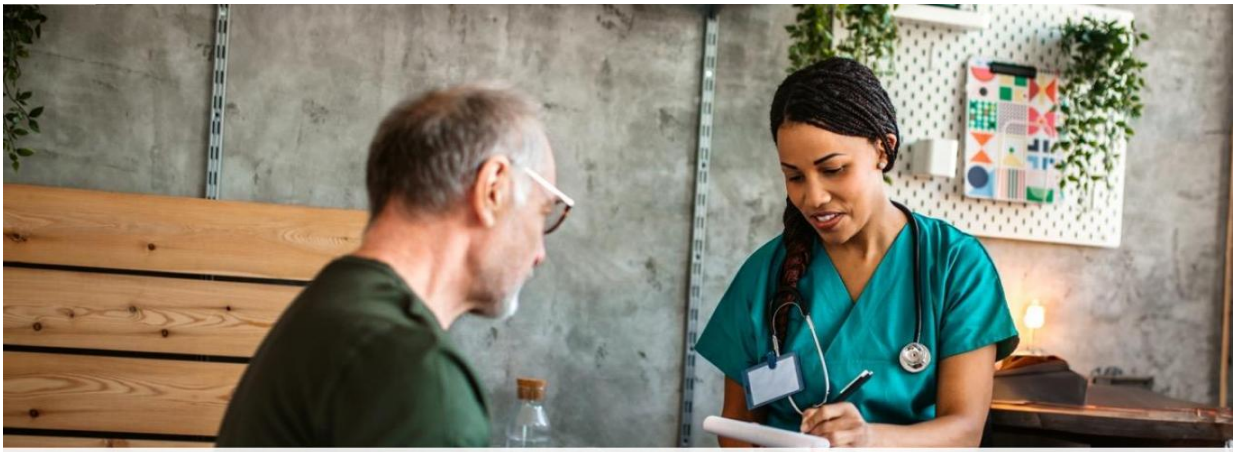


- Initiated dosing of patients in the monotherapy cohort of the Phase 1/2 clinical trial
  - Trial design includes plan for dose escalation and expansion combination cohort with immune checkpoint inhibitor (pembrolizumab; anticipated in mid-2027)

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

ARVINAS

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)



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]

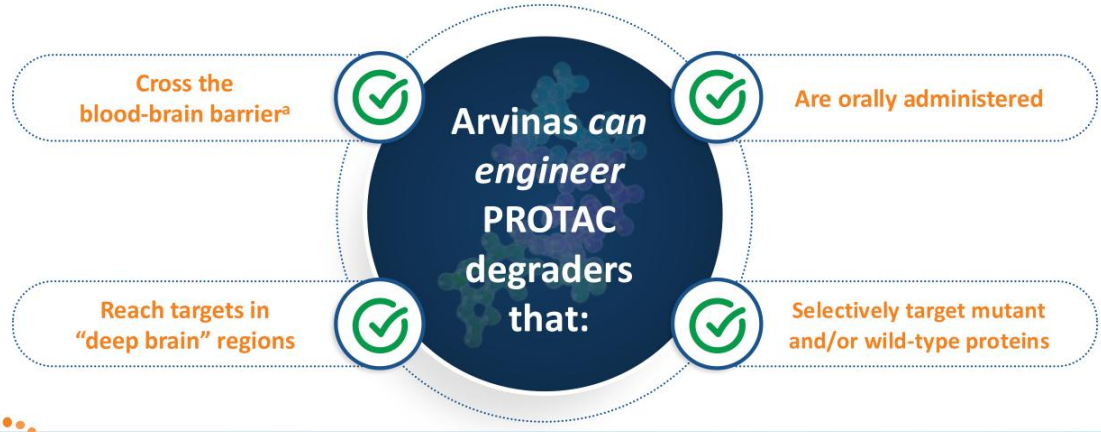
Unmet need is high:

- 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

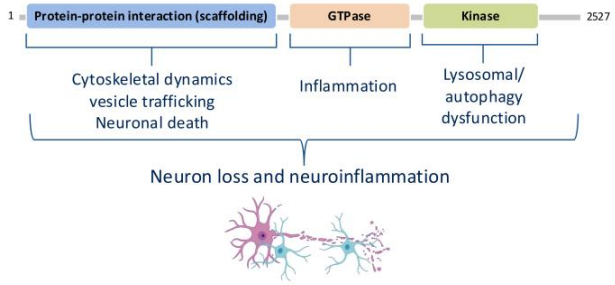
Given its role in neurodegeneration, Arvinas is exploring LRRK2-targeting PROTAC degraders as possible treatments for PSP, PD, and related diseases

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].

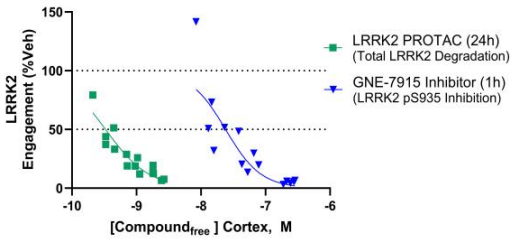
LRRK2, leucine-rich repeat kinase 2

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

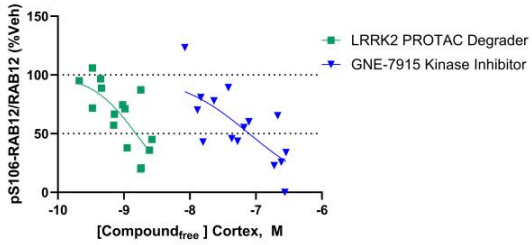
Iterative and catalytic PROTAC advantage results in stronger LRRK2 reduction and RAB pathway engagement versus a LRRK2 kinase inhibitor[a]

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)



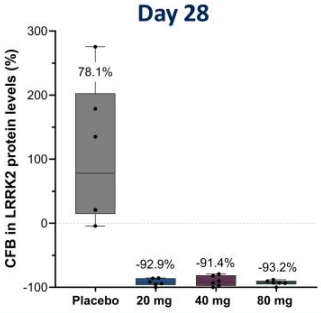
50x greater *pathway* engagement with ARV-102

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

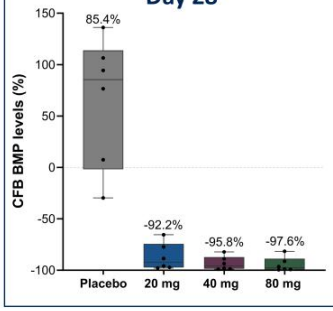
PBMC LRRK2

Day 28

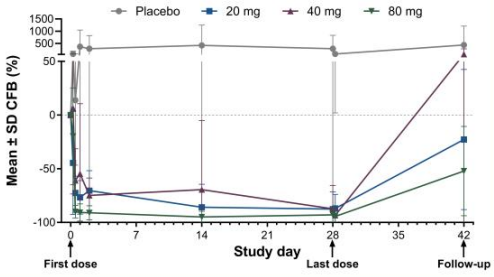


Urine BMP

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

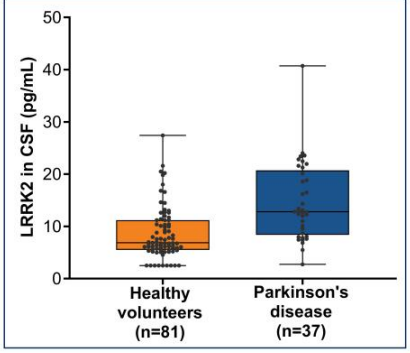
BMP, bis(monoacylglycerol)phosphate; CFB, change from baseline; LRRK2, leucine-rich repeat kinase 2; PBMC, peripheral blood mononuclear cell; PD, Parkinson's disease.

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.

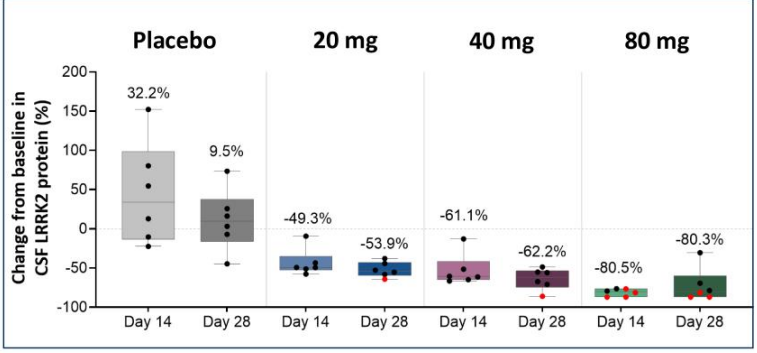
ARV-102 achieved more than ~50% LRRK2 degradation at all dose levels in the CFS of patients with PD

ARVINAS

Baseline LRRK2 in CSF[a]



Change in participants with Parkinson's disease[b]



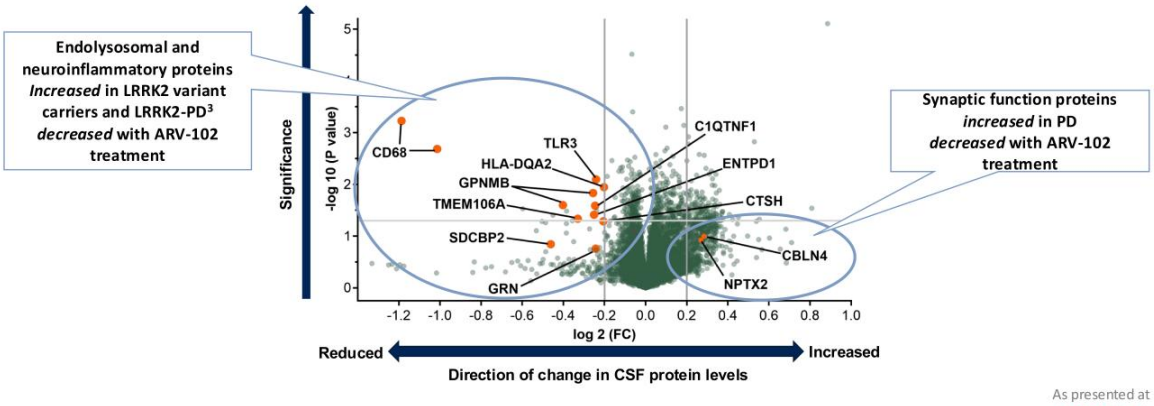
- Baseline levels of LRRK2 in CSF were higher in participants with Parkinson's disease than in healthy volunteers
- Median LRRK2 reductions of ~50% or greater were achieved in participants with Parkinson's disease starting at daily ARV-102 doses of 20 mg

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

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)



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.

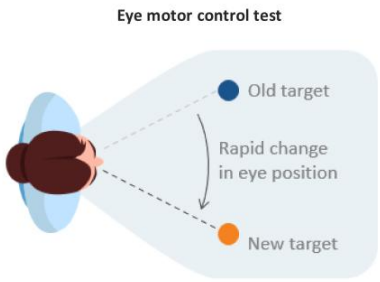
1. Smits L, et al. International Congress of Parkinson's Disease and Movement Disorders (MDS) 2025; Poster #904. 2. Van de Built L, et al. International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders (AD/PD) 2026. 3. Philips, B. et al. NPJ Parkinsons Dis. 2023 Jul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

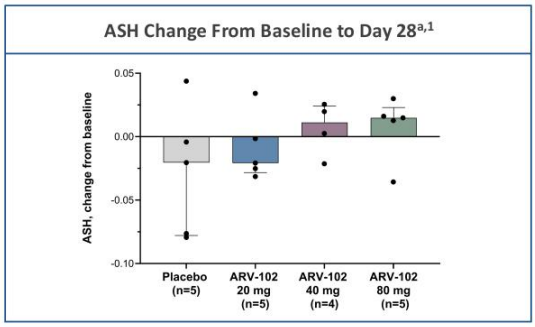
As presented at AD/PD 2026

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





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)	Phase 1: Parkinson's Disease (Complete)
Single Ascending Dose (Part A) Multiple Ascending Dose (Part B)	Single Ascending (Part A) and Multiple 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)	✓ 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.


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