Astellas Pharma Inc.

R&D Day

March 31, 2026

Event Summary

[Company Name]	Astellas Pharma Inc.
[Company ID]	4503-QCODE
[Event Language]	JPN
[Event Type]	Analyst Meeting
[Event Name]	R&D Day
[Fiscal Period]	
[Date]	March 31, 2026
[Number of Pages]	46
[Time]	09:00 – 10:36 (Total: 96 minutes, Presentation: 40 minutes, Q&A: 56 minutes)
[Venue]	Webcast
[Venue Size]	
[Participants]	

[Number of Speakers]	3	
	Naoki Okamura	Chief Executive Officer
	Tadaaki Taniguchi	Chief Research and Development Officer
	Nobuko Kato	Chief Communications & IR Officer
[Analyst Names]	Hidemaru Yamaguchi	Citigroup Global Markets
	Seiji Wakao	JPMorgan Securities
	Koichi Mamegano	BofA Securities
	Hiroyuki Matsubara	Nomura Securities
	Atsushi Seki	UBS Securities
	Shinichiro Muraoka	Morgan Stanley MUFG Securities
	Nobuaki Sawada	JPMorgan Asset Management
	Stephen Barker	Jefferies
	Miki Sogi	Sanford C. Bernstein

Presentation

Kato: Thank you for taking time out of your busy schedules to join us today for Astellas R&D Day. I am Kato, Chief Communications and IR Officer, and I'll be serving as the moderator today. It is a pleasure to have you here.

Following our presentation, we will move on to the Q&A session. The presentation will be based on the presentation materials available on our website. Simultaneous interpretation in Japanese and English will be provided throughout the event, including the Q&A session. Please note that we cannot guarantee the accuracy of the simultaneous interpretation.

You can select your preferred language from the menu at the top of the Zoom screen. If you select the original language, you will be able to listen to the audio in the original language.

Please note the following. This material or presentation and answers and statement for the Company in the Q&A session by representatives includes forward-looking statements based on assumptions and beliefs in light of the information currently available and subject to significant risks and uncertainties. Actual financial results may differ materially depending on a number of factors. They contain information on pharmaceuticals, including the products under development, but it does not intend to make any representations or advertisement of these preparations. The data we are going to introduce today is based upon the contents presented at the Congress meeting.

Let me introduce today's speakers, the presenters, Naoki Okamura, CEO; and Tadaaki Taniguchi, CRDO, Chief R&D Officer. Now presentation is started.

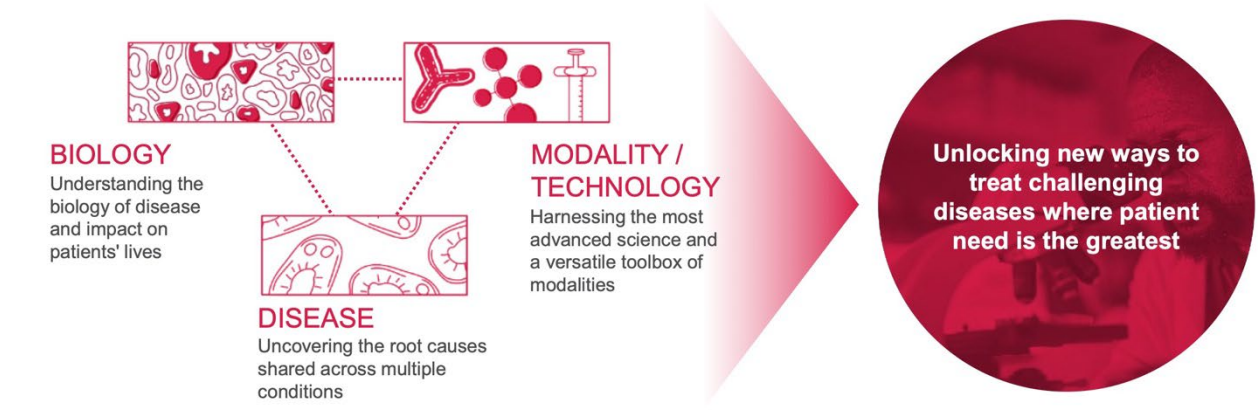
Okamura: Good morning, everyone. I am Okamura from Astellas Pharma Inc.

Thank you very much for taking the time to join us for this briefing today.

First of all, Astellas has a very clear vision.

It is on the forefront of health care change to turn innovative science into value for patients. At Astellas, we define this value in bold by placing the outcomes that are truly matter to patients in the numerator and the cost to the health care system of delivering those outcomes in the denominator.

This approach serves as a guiding principle for decision-making across the entire company, including our R&D strategy.



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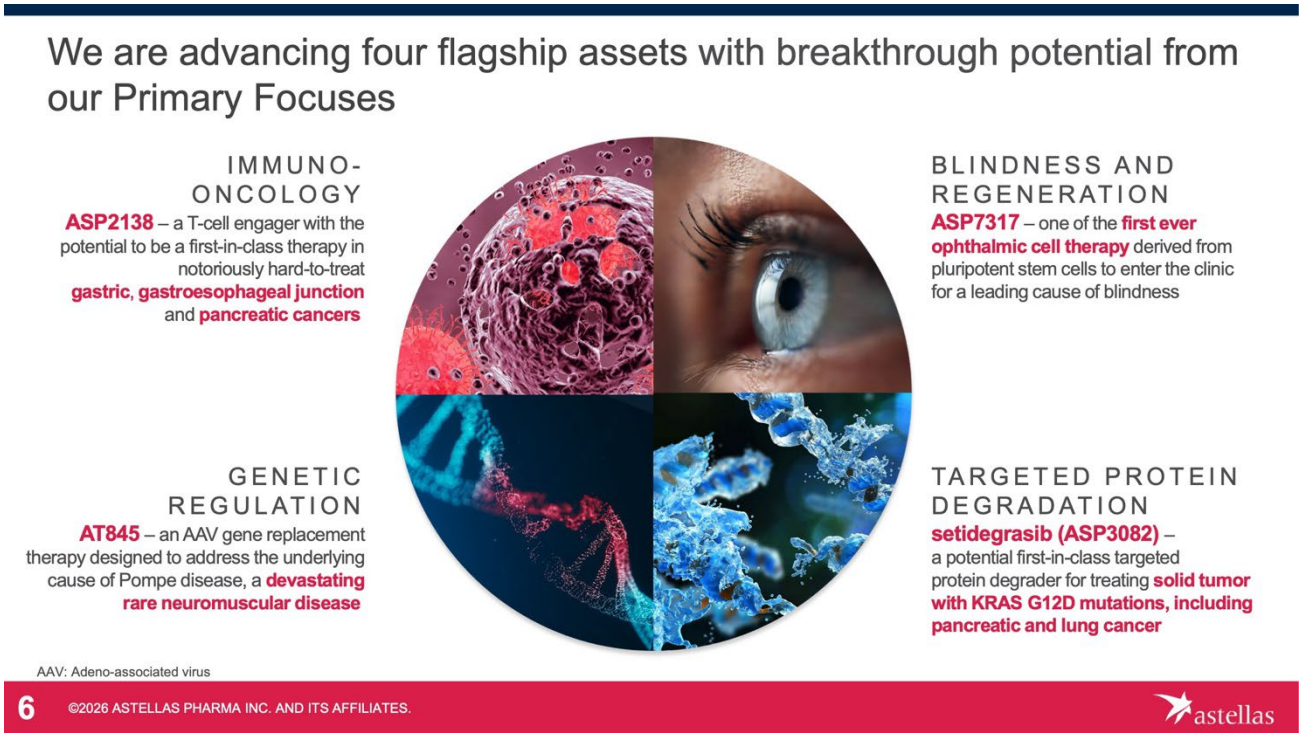
We adopt a research and development strategy known as the Focus Area approach with a goal of delivering meaningful outcomes in areas with high unmet medical needs. The Focus Area approach consists of three core elements: biology, modality and technology, and disease.

We begin by understanding the biology of the disease and its impact on patient lives. Next, we select the optimal modality or technology that aligns with the characteristics of the biology and apply it to the patients who are most likely to benefit.

When these three elements are firmly linked to form a solid triangle, we define this as our primary focus. By providing around the vertices of this triangle, we believe we can generate multiple valuable programs from a single scientific foundation.



Page six.

We concentrated our R&D resources on areas where we possess deep scientific expertise and have the highest potential to deliver value to patients. We currently have four Primary Focus: Immuno-Oncology, Targeted Protein Degradation, Blindness and Regeneration, and Genetic Regulation.

For each Primary Focus, we have designated Flagship programs aimed at the proof-of-concept or PoC judgment by the end of FY2025 and have been advancing development with a high priority. Of these, we have achieved PoC for three assets, ASP2138, ASP7317, and setidegrasib previously referred to as ASP3082.

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By making disciplined decisions regarding our portfolio and accelerating high-quality science, we have driven the creation of tangible value for patients. As shown in the slide, we have made significant progress over the five-year period of our CSP2021.

First, we have accelerated our pipeline. Over the past five years, we have achieved the 12 Phase first subject dose or FSD for new molecule entities and initiated one new Phase III trial. We have steadily increased our speed and execution capabilities across the entire development process, including achieving a total of four PoC from three assets.

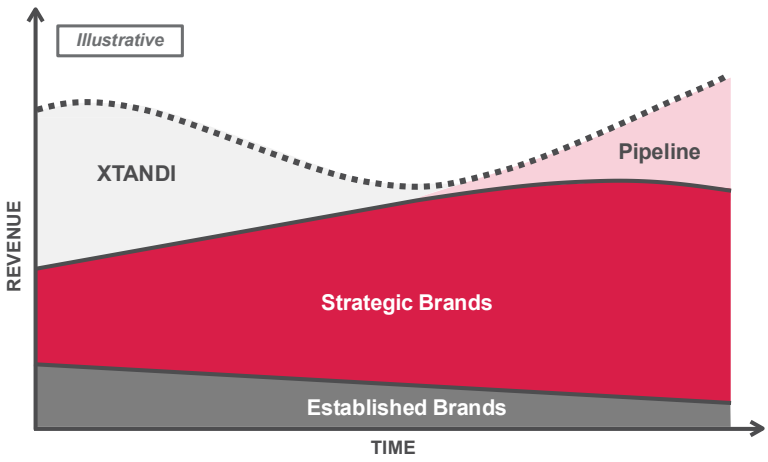
Next, we strengthened our portfolio discipline. We decided to terminate 21 clinical stage programs. As a result, we reallocated resources to assets with a higher expected value and lower risk. This has significantly improved the quality of our entire pipeline.

Also, we built a foundation for sustainable productivity. We transformed our R&D organization into a patient-centric end-to-end model, invested in key capabilities and introduced new ways of working to achieve more consistent results.

This series of achievements clearly demonstrates the strength of our Focus Area approach. This approach will also form the foundation of Astellas' R&D strategy in our next midterm business plan scheduled to be released on May 26.

We are building on this momentum as we inflect to growth

Astellas is managing its transition with focus and control



Maximize Revenue
Elevate the peak and flatten the dip

Accelerate Pipeline
Expected sales contribution in 2030s

Operational Efficiency
Elevate profitability and invest in Strategic Brands and pipeline

Strategic Brands: PADCEV, IZERVAY, VYLOY, VEOZAH, XOSPATA

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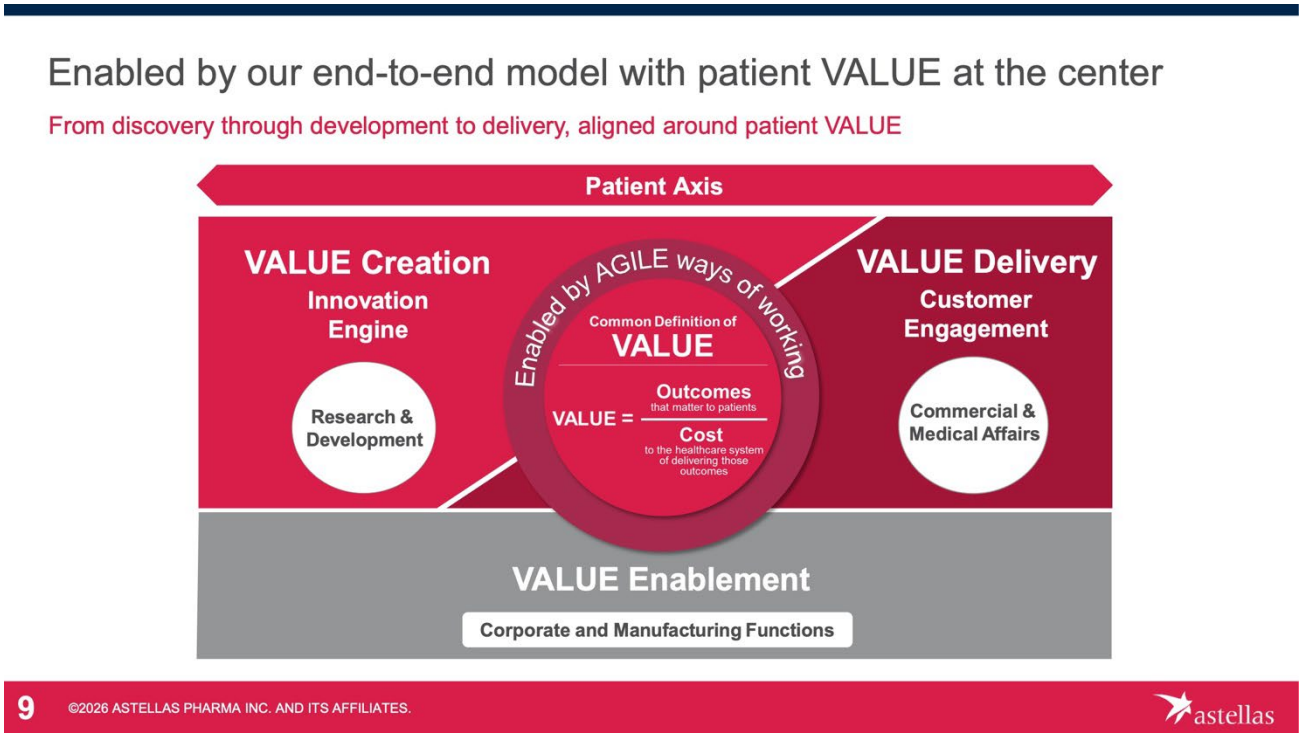
We are implementing initiatives to overcome short-term challenges, including a loss of exclusivity of XTANDI and a return to growth. First, we are focusing on maximizing sales of our strategic brands to mitigate the impact of revenue decline following the loss of exclusivity of XTANDI and to pave the way for future growth. At the same time, we are focusing on accelerating development to build our pipeline to market as quickly as possible.

Furthermore, by advancing these efforts and robust operational efficiency and financial discipline, we are building a profitable business structure while addressing current challenges. Through these approaches, we will continue to invest in long-term growth while addressing short-term challenges, thereby maintaining and strengthening our momentum.



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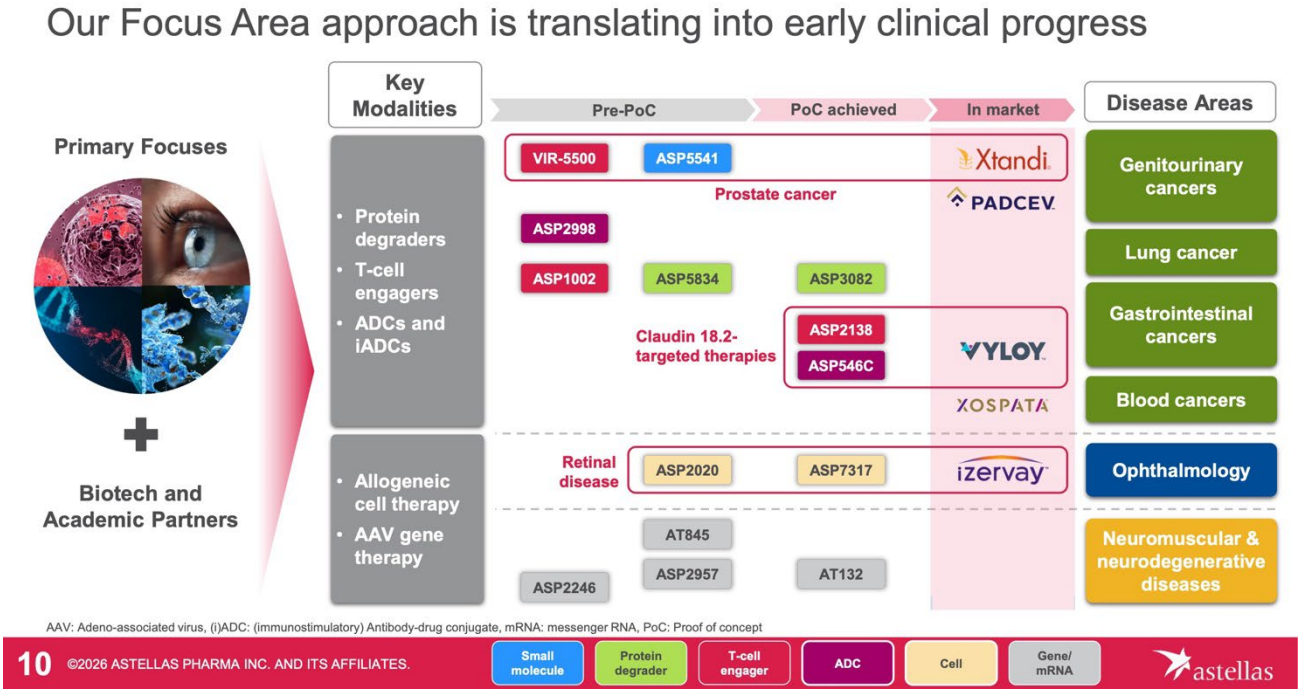
As part of this strategy, we have introduced a new end-to-end model along the patient axis and have been driving organizational transformation. Under this new operational model, we view research and development, commercialization, and life cycle management as an integrated whole, consistently focusing on improving productivity and creating value.

By strengthening collaboration across functions, we are able to advance programs more efficiently from the early stages of drug discovery through late-stage development, enabling clearer and faster decision-making.



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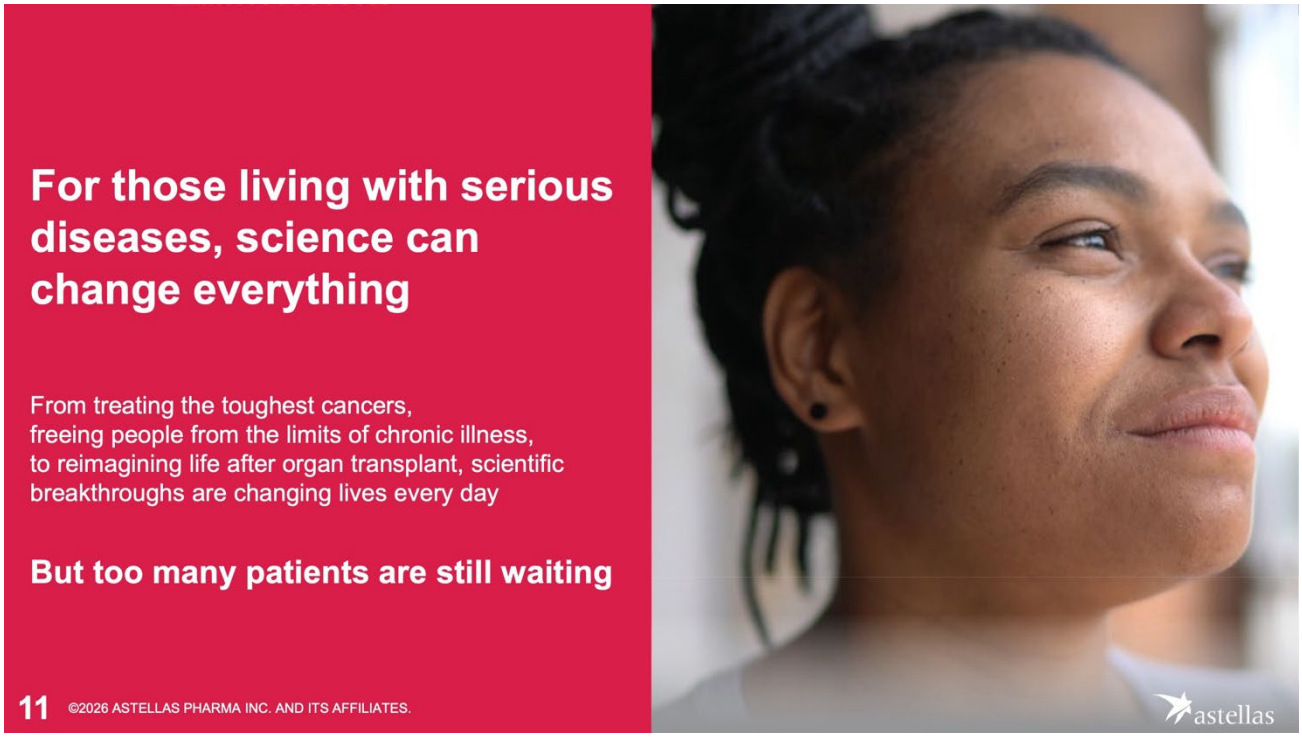
Through our disciplined strategy, we are building a robust pipeline. While focusing on areas where we possess deep expertise, we are also leveraging the capabilities of external innovation partners to create meaningful value for patients.

In each therapeutic area, we have built a deep pipeline comprising not only our strategic brands already on the market and our primary focus flagship programs, but also follow-on programs, externally acquired programs, and even early-stage research programs that support next-generation innovation, thereby forming multiple franchises.

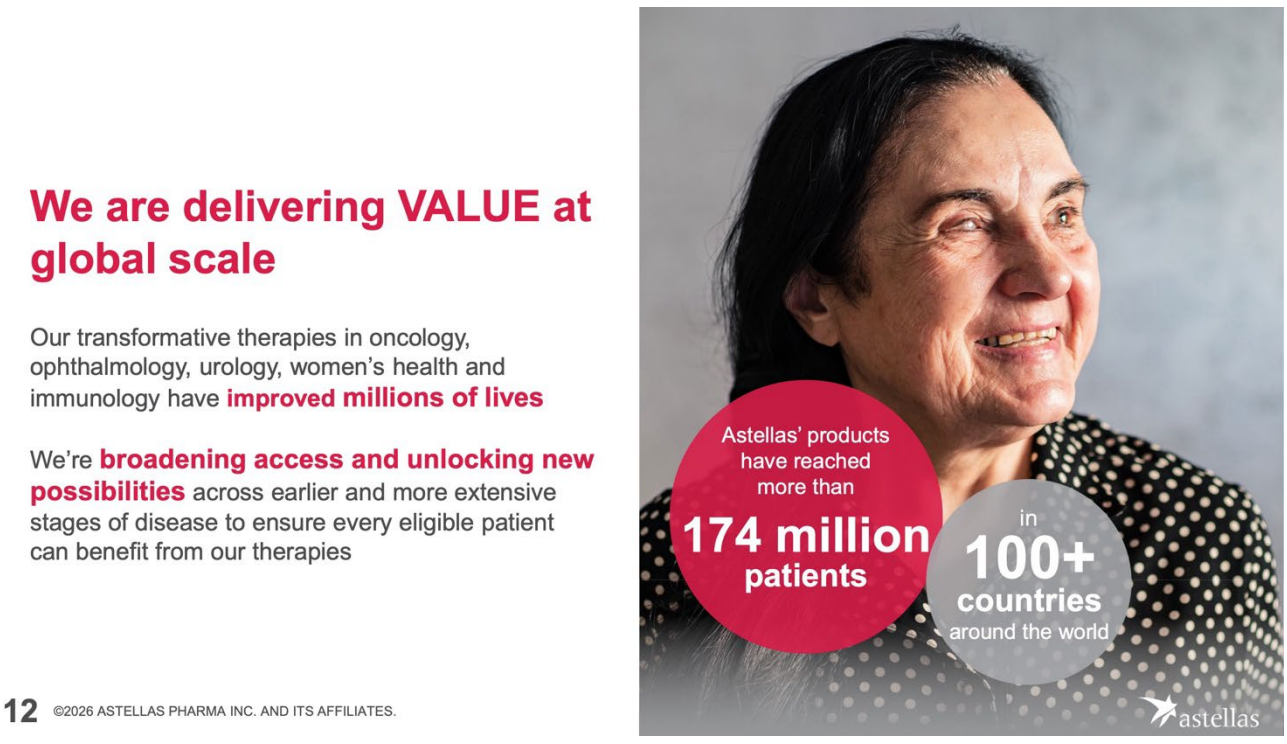
Good examples include the prostate cancer franchise, multi-modality therapies targeting Claudin 18.2, and the acquisition of IZERVAY, which is indicated for geographic atrophy in age-related macular degeneration, GA in AMD, paving the way for retinal pigment epithelial cell-based therapies. These efforts have created a balanced and sustainable portfolio that reliably delivers short-term progress while continuously generating future innovation.

Next, Taniguchi will provide a detailed explanation of our R&D initiatives. Taniguchi-san, please.

Taniguchi: Good morning. I am Taniguchi, CRDO. I will now provide a detailed explanation of our R&D initiatives.



For those living with serious diseases, science holds the potential to significantly transform their lives. However, many patients still have limited treatment options. Addressing these unmet medical needs is a major driving force behind our R&D strategy.



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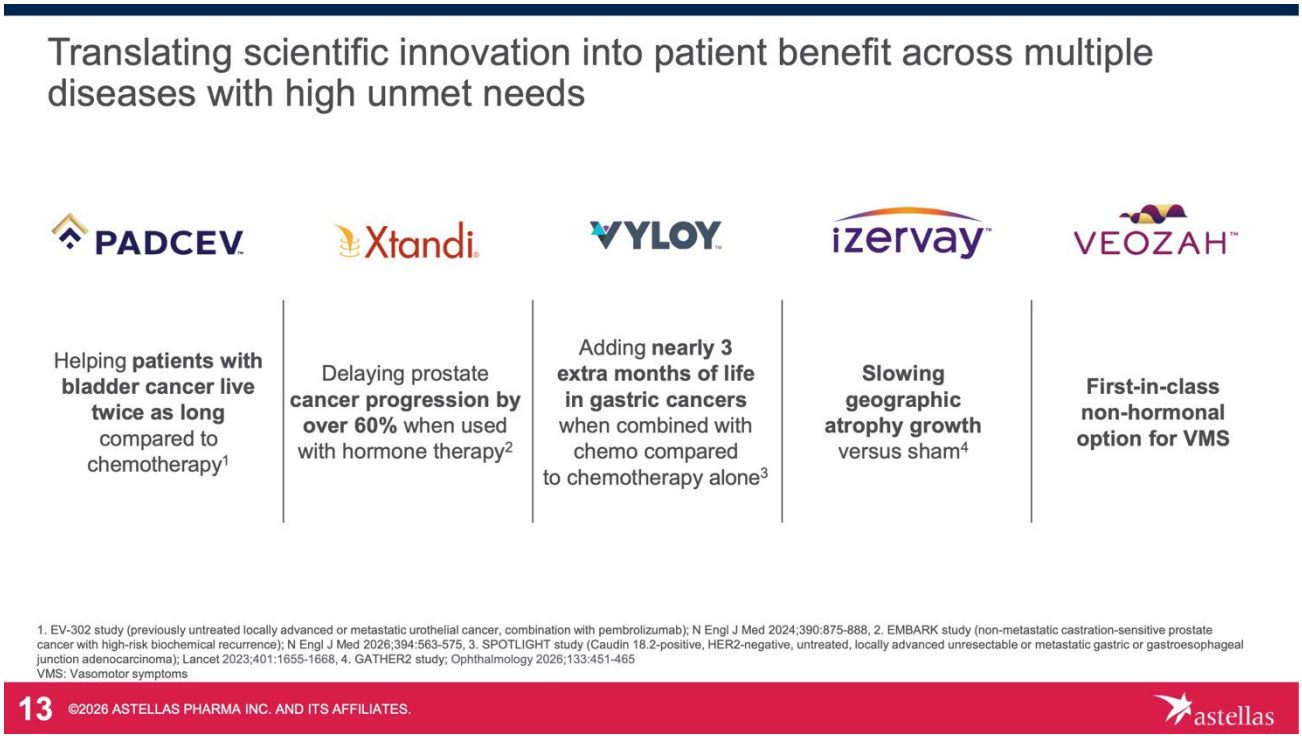
Astellas has a proven track record of continuously developing innovative medicines and delivering them to patients. The innovative treatments developed by Astellas across multiple therapeutic areas are being used by patients in many countries around the world. Building on our wide experience accumulated to date, we

continue to work toward expanding access to medicines and further improving treatment outcomes while broadening our focus to earlier and more extensive stages of disease.



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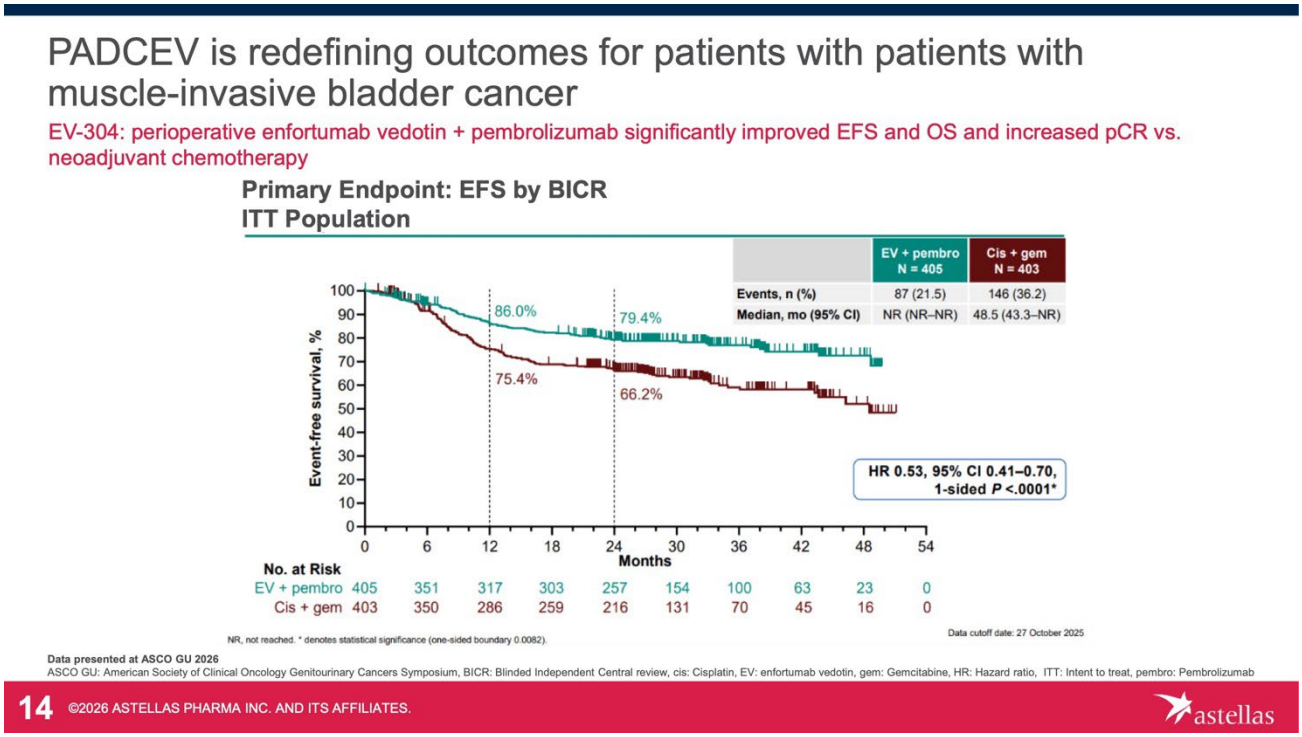
In fact, our efforts to date have fundamentally transformed patients' expectations for treatment. In the oncology field, recent Phase III study results have further solidified the position of PADCEV in urothelial cancer in EV303 and EV304 study.

Regarding XTANDI, the Phase III EMBARK study demonstrated that it significantly delayed disease progression in earlier stage prostate cancer. In gastric cancer, combination data from VYLOY ILUSTRO study support its clinical potential with the checkpoint inhibitor.

In ophthalmology, IZERVAY has demonstrated the ability to inhibit the progression of geographic atrophy. In women's health, VEOZAH offers a new nonhormonal treatment option for vasomotor symptoms or VMS.

This result demonstrate the ongoing success of Astellas R&D efforts in consistently translating innovation into value for patients across multiple disease areas.



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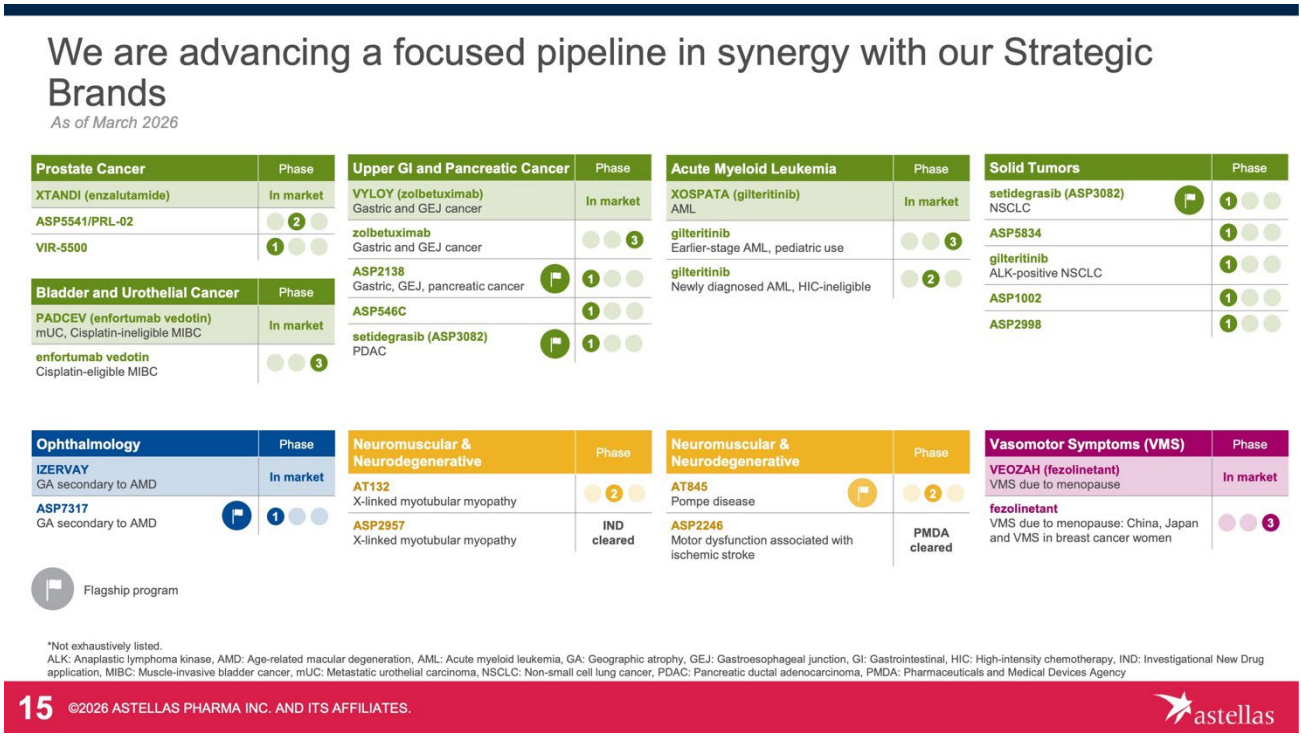
I will explain PADCEV enfortumab vedotin as an example. In February ASCO GU, we presented the latest data from EV-304 trial, the study targeting patients with cisplatin-eligible muscle invasive bladder cancer or MIBC. The perioperative enfortumab vedotin and pembrolizumab significantly improved the primary endpoint, event-free survival, EFS, compared to neoadjuvant chemotherapy.

In addition, significant improvements were observed in overall survival, OS, and pathological complete response, pCR. These results further confirm the benefits of PADCEV in the perioperative treatment of MIBC.



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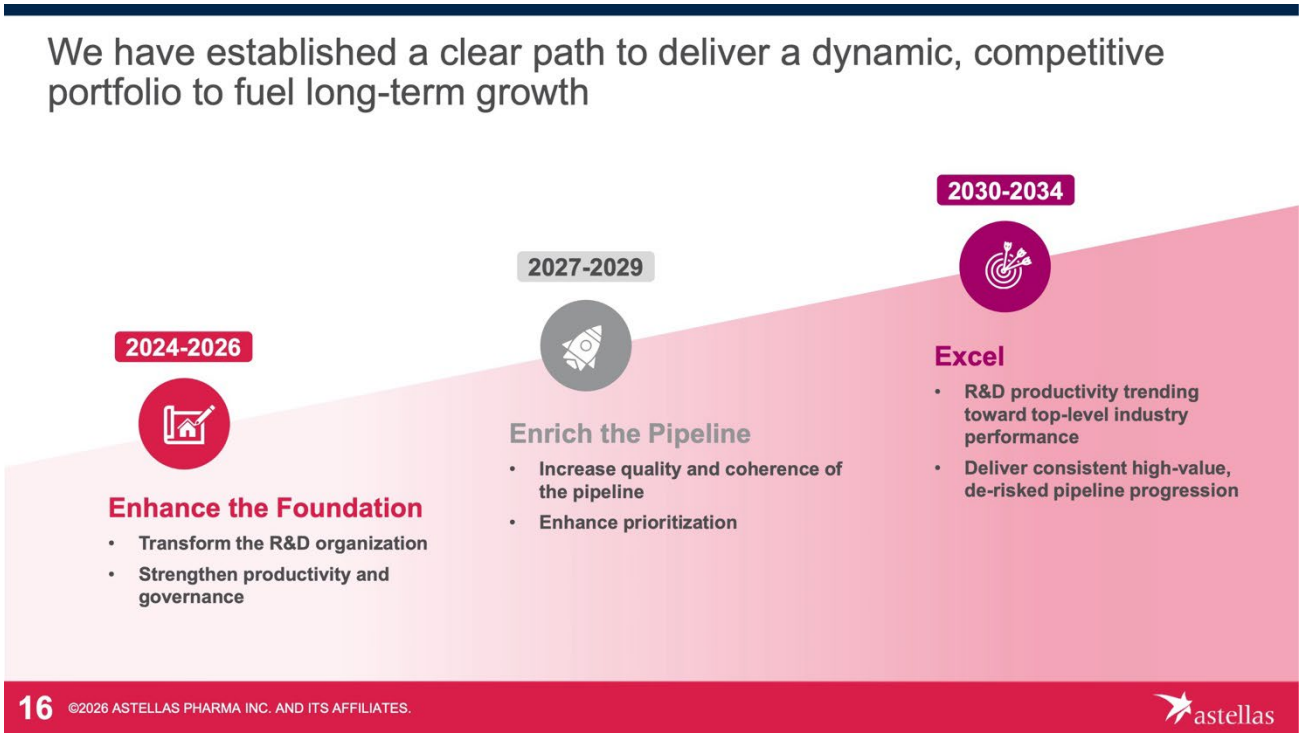
This slide illustrates progress across the full development spectrum. Our broad pipeline ranges from strategic brands that continue to expand their impact through life cycle management to a diverse range of early-stage development programs that will contribute to future growth.

By focusing on the life cycle management of strategic brands in areas such as oncology, ophthalmology, and women's health, we are maximizing value while further strengthening our leadership in each field. At the same time, we are steadily building our pipeline that will underpin our future growth through our Primary Focus Flagship program and several follow-on programs.



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In order to address the challenges ahead of us with XTANDI's loss of exclusivity, we need to build a resilient portfolio that will support our mid- to long-term growth. From 2024 to 2026, we fundamentally transformed our R&D organization and governance structure and achieved productivity gains by strengthening the foundation necessary for sustained success.

Building on this newly established foundation from 2027 to 2029, we will set clear priorities, generate more high-quality development programs, and further enrich our pipeline. As a result, from 2030 to 2034, we will be able to significantly improve R&D productivity by accelerating the development of higher-value products.

Next slide, please. Now I would like to share more about transformation of our R&D organization.

Next slide, please. Page 18.

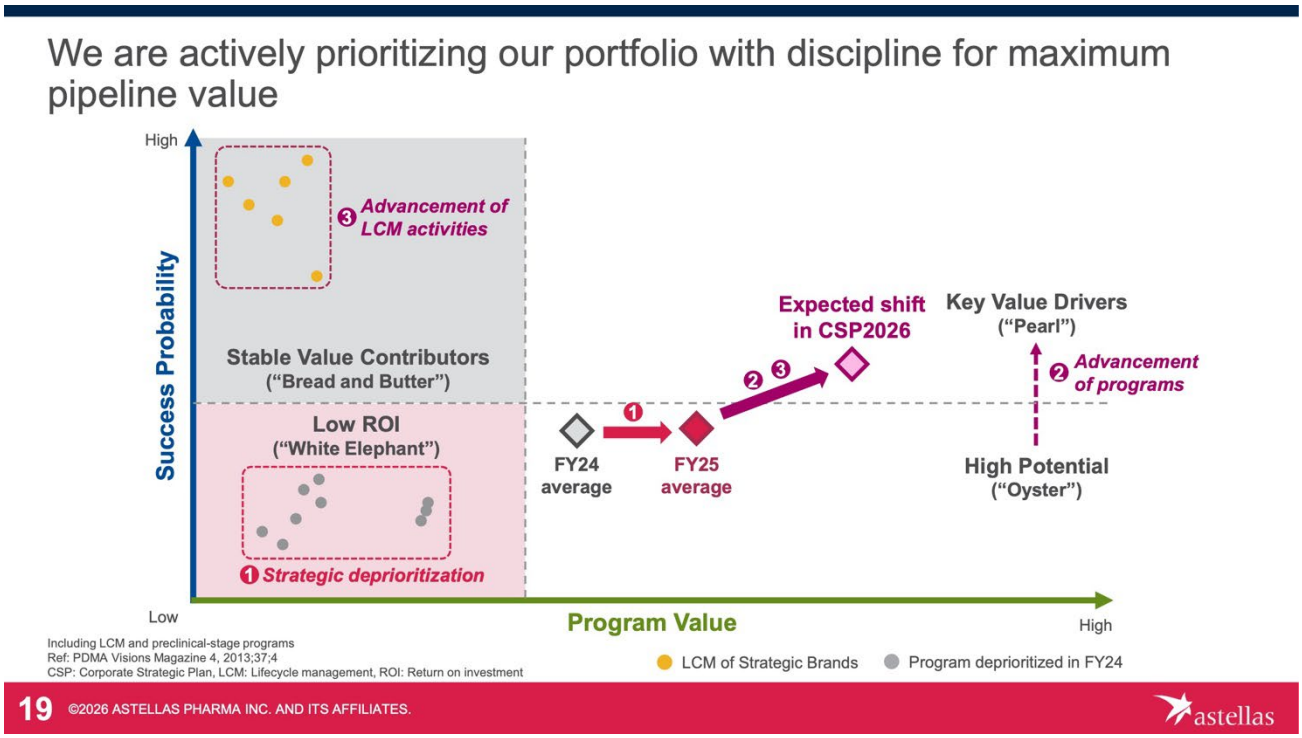
Since 2024, we have taken bold measures to increase productivity and efficiency in R&D, and these measures are already delivering tangible benefits. Regarding internal and external collaboration, by introducing end-to-end operation model and agile working practices mentioned by Okamura, we have reduced handover processes between departments, enabling teams to advance project more quickly.

Furthermore, by collaborating with right partners, we have been able to create a broader range of values. Through initiatives to accelerate the pipeline, we have shortened development cycle times and enhanced necessary capabilities. We are also actively investing in talent, striving to enhance the skills of R&D teams and expertise of early development stage.

In development, we have established a system capable of conducting our own research clinical trials, reducing costs, and accelerating clinical trial speed and quality improvement. I will explain the details later on.

Furthermore, by actively promoting the use of data, including the proactive utilization of real-world data, integration of R&D database, and AI-driven simulation and modeling, we are working to speed up decision-making and strengthen evidence base.

These initiatives form a cohesive strategy collectively and continuously improving speed, quality and efficiency of our R&D.



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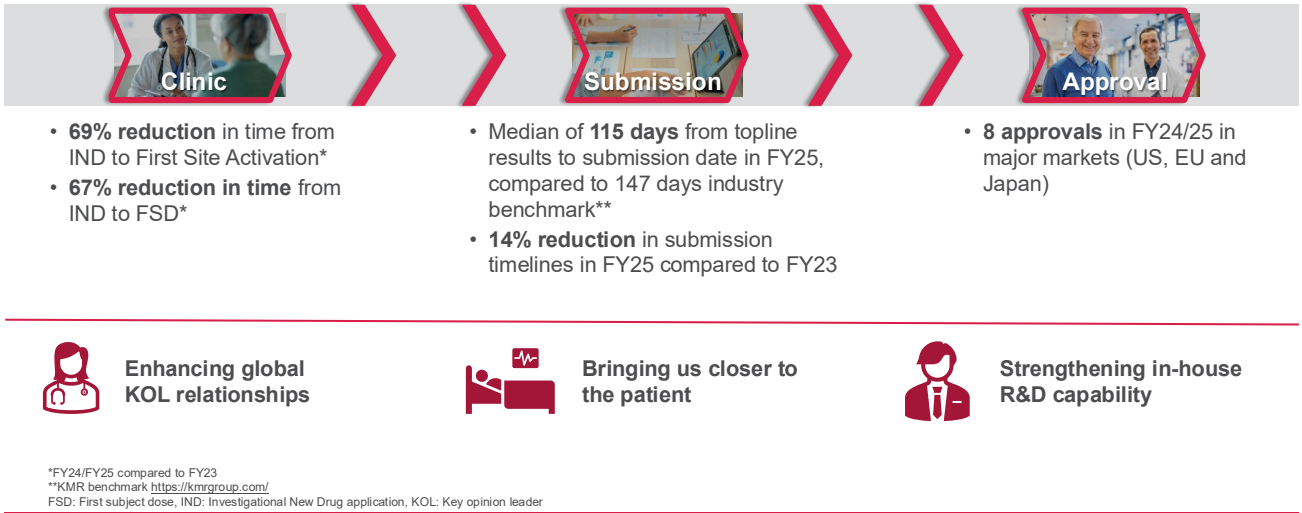
I will explain how we prioritize our portfolio. We continuously review all programs from preclinical stage to life cycle management based on each probability of success and value. For programs assessed as having both a low probability of success and low value located in the bottom left of the diagram, we make strategic decisions such as lowering the priority or stopping them.

This allows us to allocate resources to promising programs in the bottom right, thereby accelerating their development. As development progresses, we expect the probability of success to increase, shifting those programs to the top right quadrant and enhancing the overall value of the pipeline.



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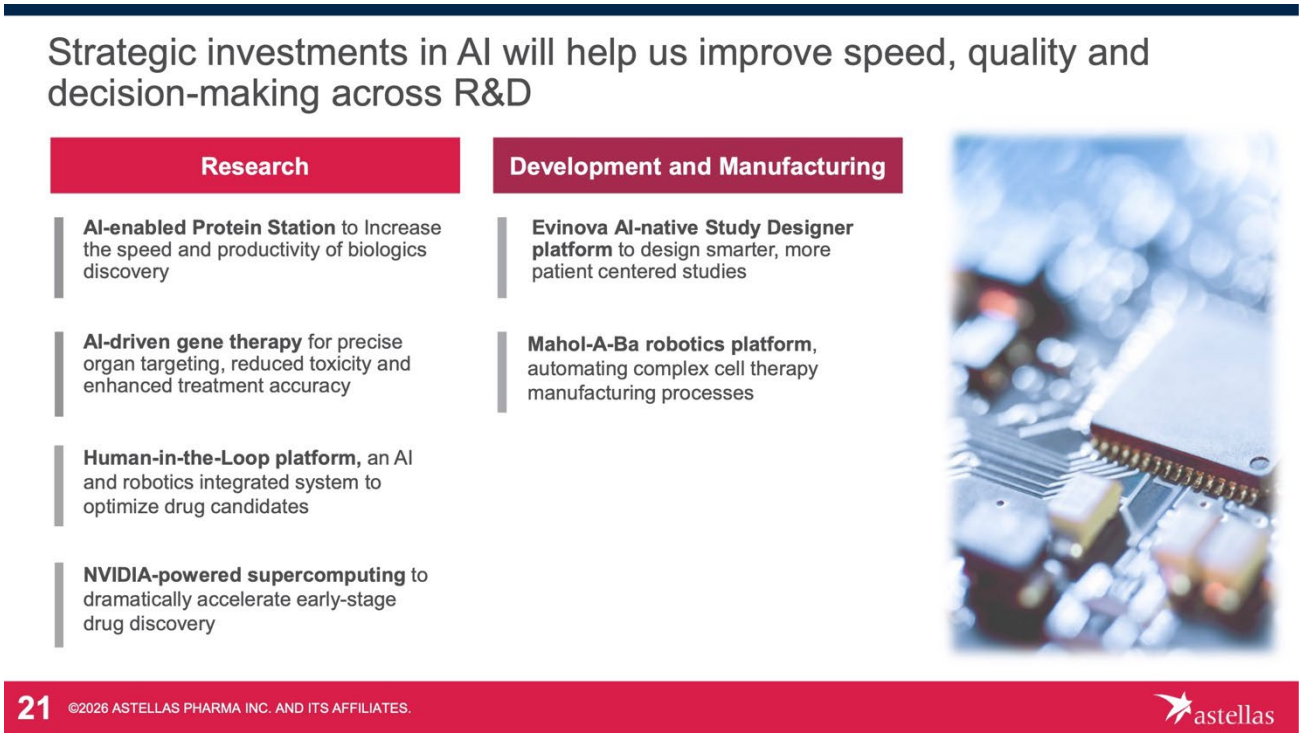
We are reducing the time from clinical trial execution to submission and approval through transforming clinical operations, specifically through in-house development of key capabilities ranging from protocol development and clinical trial conduct to data analysis and study reports preparation. We aim to strengthen direct communication with trial sites, thereby improving the quality of protocols, accelerating patient enrollment, and expediting data analysis and study report preparation.

As a result of this transformation, in clinical trials, the achievement of milestones such as site selection and first dosing has been accelerated. In the regulatory submission process, the time from submission or acceptance has been significantly reduced. Furthermore, by improving the quality of our submission, we have shortened the time from submission to approval. In FY2024 and FY2025, we obtained eight approvals in major markets.

Furthermore, these benefits is not just a onetime event, but can lead to ongoing improvement in our capabilities, such as strengthening relationship with our trial sites and physicians, conducting patient-centered trials, and AI-driven automation, as explained in the next slide.



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We are investing in cutting-edge technologies, including AI and robotics, to accelerate our digital transformation proactively. For example, in drug discovery research, we aim to accelerate the development of biopharmaceuticals and improve productivity, not only through NVIDIA's AI-powered supercomputing for small molecule drug design that has already been implemented, but also by introducing AI-enabled protein station for research automation. AI-driven gene therapy will lead to precise organ targeting, reduced toxicity, and enhanced treatment.

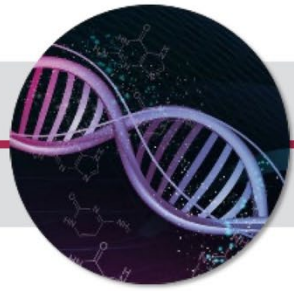
These tools will help us test hypothesis faster and prioritize the strongest programs, accelerating research reproducibility and speed.

In development, leveraging study designer, Evinova's AI-native platform will enable us to efficiently design more sophisticated patient-centric clinical trials. In the future we aim to further improve trial efficiency through initiatives such as clinical trial monitoring using AI agents.

Our Research Centers of Excellence drive focused and connected innovation to enrich our early pipeline

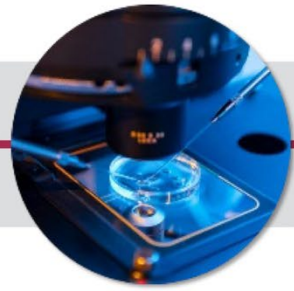
ONCOLOGY RESEARCH

Advancing early-stage cancer therapies through cutting edge-modalities including targeted protein degradation and immuno-stimulatory approaches



CELL AND GENE THERAPY RESEARCH

Developing the cell and gene therapies of the future with AAV-delivered gene therapies and pluripotent stem cell-based allogeneic therapies for diverse and complex diseases



INNOVATION LABS

Harnessing the best external innovation underpinned by strong biology through open innovation and external R&D, to expand our pipeline beyond oncology, and cell and gene therapy research

AAV: Adeno-associated virus

Next slide, please. Page 22.

We are driving cutting-edge research and building a long-term pipeline. Our research consolidated eight divisions to three Centers of Excellence, namely Oncology Research, Cell and Gene Research, and Innovation Labs expecting they will be our core enablers for our mid- to long-term R&D strategy. By integrating expertise of each research center, we can develop optimum treatment based on the pioneering scientific findings while fully understanding patients' needs and their conditions. Furthermore, these centers act as hubs for collaboration in and outside of the Company, leading cutting-edge science while working closely with partners in academia and biotech sector.

Strong partnerships enhance and accelerate our innovation potential

Biotech Collaborations

Vir Biotechnology, xencor, DYNO THERAPEUTICS, FIMECS, SUTRO BIOPHARMA, EVOPOINT Biosciences, cullgen, Sangamo, KATE THERAPEUTICS, Kotobuki Pharma, PeptiDream, AJINOMOTO, TWIST BIOSCIENCE, Healios

Academic Collaborations

GUSTAVE ROUSSY CANCER CAMPUS GRAND PARIS, MD Anderson Cancer Center, Mass General Brigham, 東北大学 TOHOKU UNIVERSITY, MILNER THERAPEUTICS INSTITUTE, JAPANESE FOUNDATION FOR CANCER RESEARCH, 京都大学 KYOTO UNIVERSITY, The ROYAL MARSDEN NHS Foundation Trust

Open Innovation

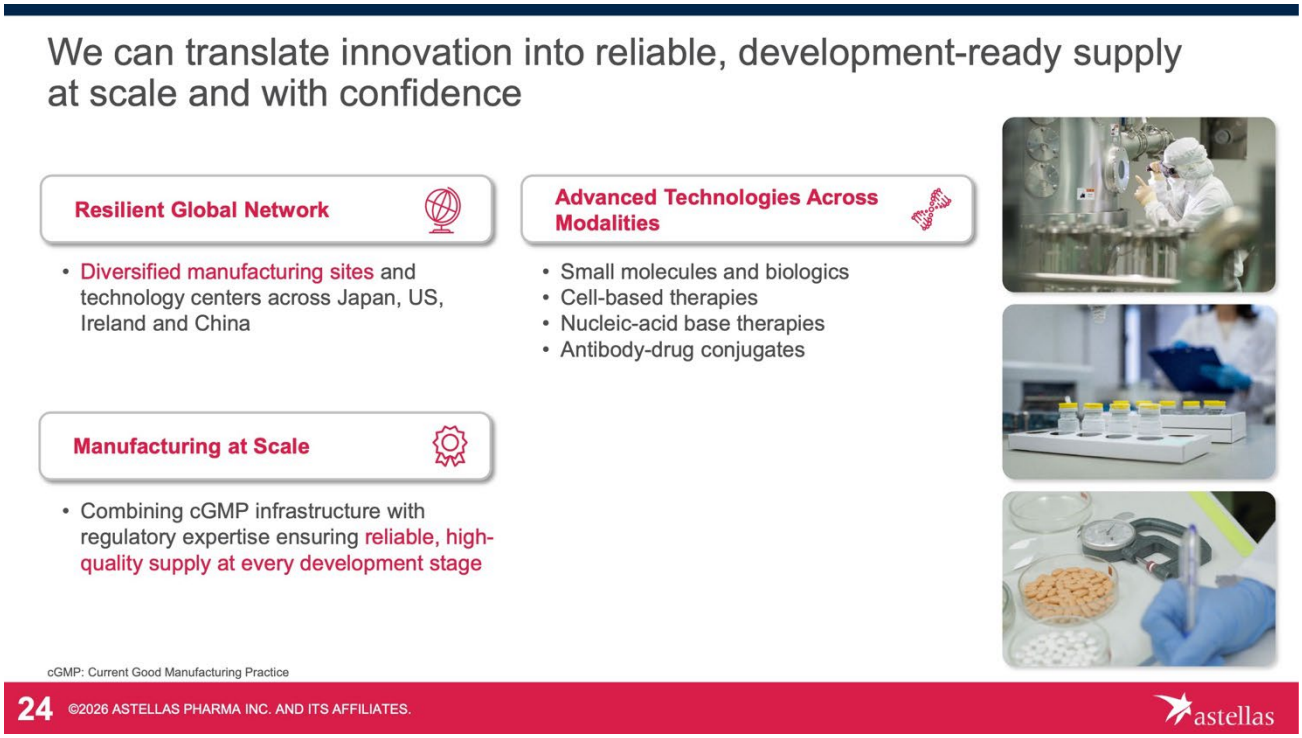
SakuLab, AUTOBAHN LABS, 238 Lab | Central, Ciconia Bioventures Inc

Next slide, please. Page 23.

We are strategically building an strategic ecosystem in collaboration with a wide range of partners, incorporating outstanding external technologies and expertise through partnership with biotech companies such as Evopoint and Vir Biotechnology as well as world-leading academia such as Mass General Brigham, MGB. We are combining our deep expertise in Primary Focus with cutting-edge external innovation to drive progress in R&D.

As an example of a partnership with academia, Astellas is the only pharmaceutical company partnering with Mass General Brigham, MGB. Through close collaborative research program, we are building early-stage pipeline and translational medicine, accelerating the creation of new therapies in oncology, rare disease, and ophthalmology.

By sharing a long-term vision with our strategic partners and collaborating openly and flexibly, we are strengthening and accelerating innovation.



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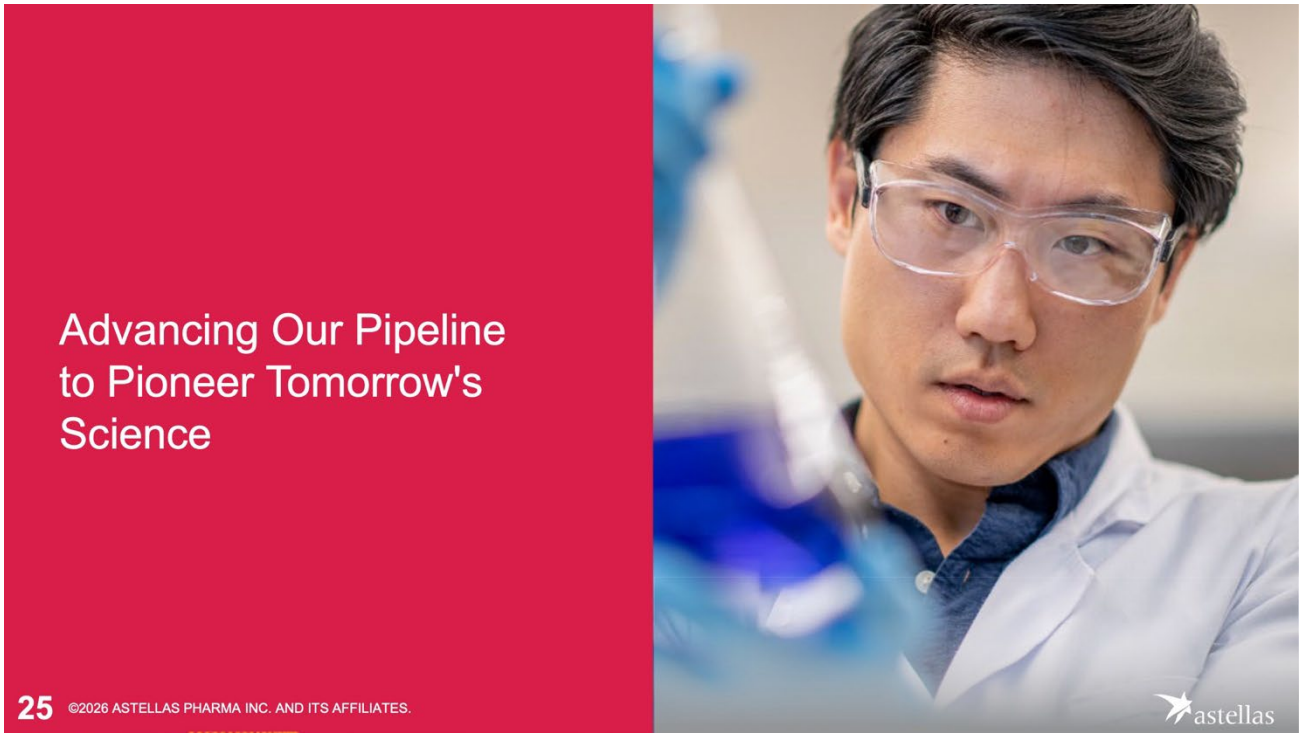
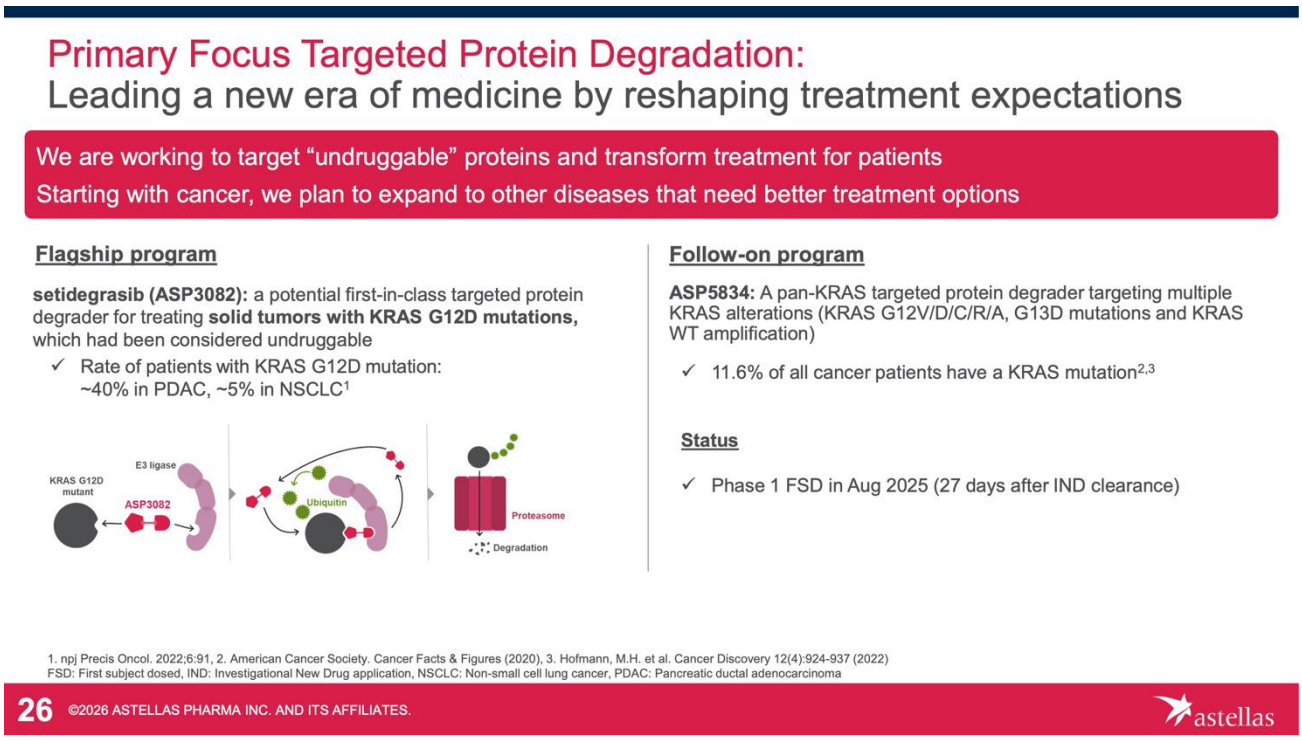
We have built reliable manufacturing and supply capabilities to provide consistent support for R&D innovation from the early development through commercialization. We have established a global manufacturing and supply network with manufacturing sites in Japan, the US, Ireland, and China and through strategic partnerships with CDMOs.

We have made strategic investments in our own manufacturing capabilities, including our cell and gene therapy manufacturing sites in the US to ensure we are equipped to handle the diverse and complex modalities emerging from primary focus.

By combining this manufacturing infrastructure with our regulatory expertise, we deliver high-quality manufacturing and supply at every scale throughout development.



Next slide, please. Next, I will outline the progress of our pipeline with each primary focus area.



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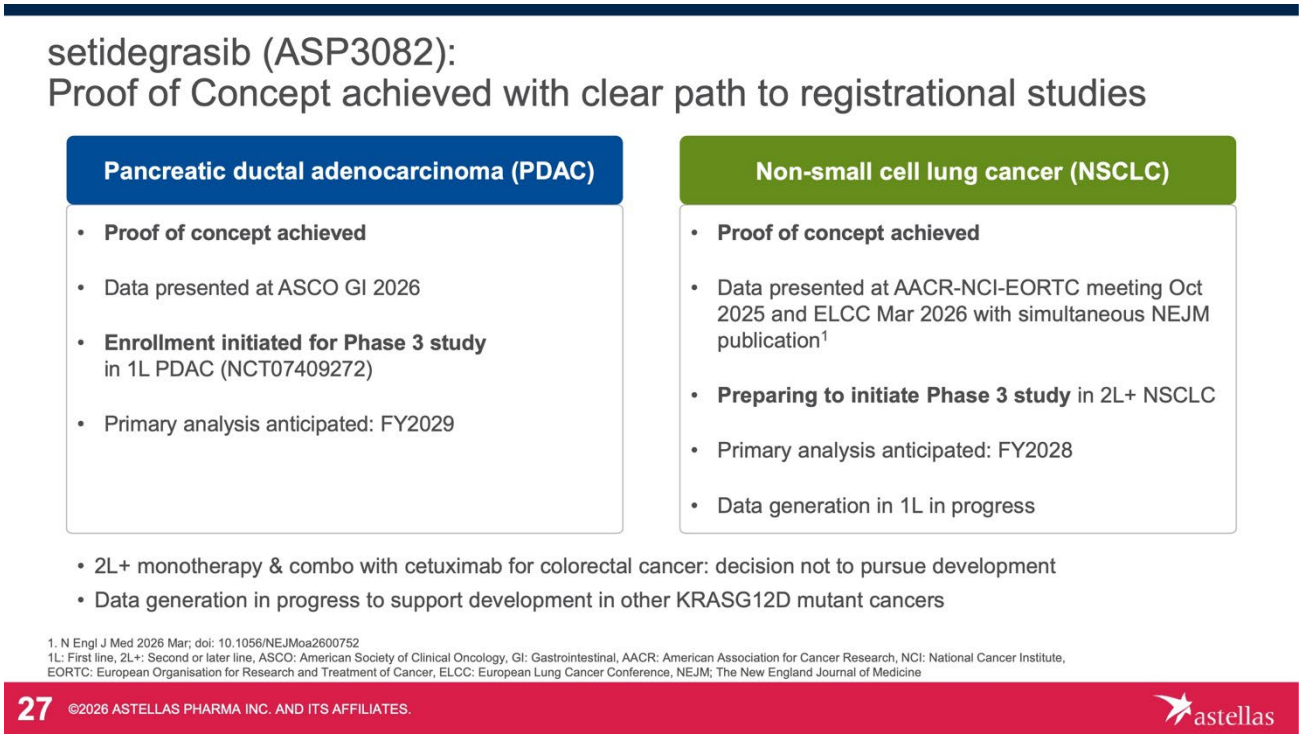
In our Primary Focus Targeted Protein Degradation, we are working to target proteins that have historically been considered undruggable with the ambition to transform treatment options for patients. Our Flagship asset, setidegrasib, is a potential first-in-class targeted protein degrader for solid tumors with KRAS G12D mutations, which had long been considered undruggable. KRAS G12D mutation is found in approximately 40% of PDAC, pancreatic ductal adenocarcinomas, and approximately 5% of NSCLC, non-small cell lung cancers.

Alongside our flagship program, we are advancing a follow-on asset, ASP-5834, pan-KRAS targeted protein degrader designed to address multiple KRAS alterations, reflecting the expandability of the platform.

The progression of both the flagship and follow-on asset illustrates how this platform can be expanded beyond a single target, supporting our ambition to extend to additional cancers and potentially other diseases over time.

I will explain the latest development status on setidegrasib using a few slides.



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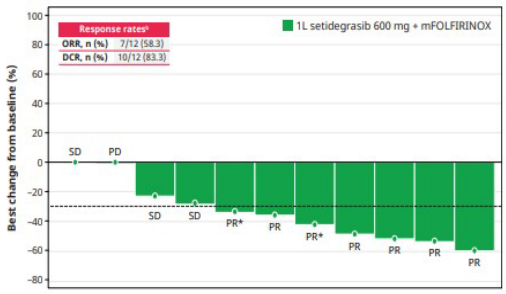
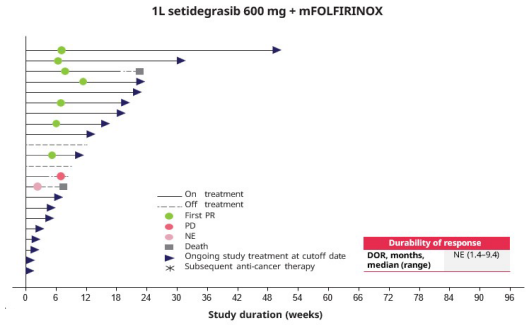
setidegrasib has achieved the PoC in pancreatic ductal adenocarcinoma, PDAC, and non-small cell lung cancer, NSCLC, both of which have significant unmet medical needs and is currently being developed in parallel across multiple cancer types.

For PDAC, we have initiated enrolling patients in a Phase III trial for first-line treatment and primary analysis is anticipated in FY2029. We have also achieved PoC in NSCLC and are currently preparing to initiate a Phase III study for second and later lines. Primary analysis for this is also anticipated in FY2028.

In addition, data generation is in progress in other cancer types with the KRAS G12D mutations. Regarding colorectal cancer, based on the clinical data available to date, we have decided not to pursue late-stage development.

setidegrasib (ASP3082) shows antitumor activity in combination therapy in PDAC





Dashed lines at −30% represent PR, per RECIST version 1.1. Asterisks represent unconfirmed PR per RECIST version 1.1 in patients with ongoing treatment.

ORR/DCR analysis included all patients who received at least 1 dose of setidegrasib. Three patients were excluded from the waterfall plot of best change from baseline (withdrawal by patient, n = 1; withdrawal due to AE, n = 1; PD, n = 1); Ten patients who received at least 1 dose of setidegrasib were excluded from the ORR/DCR analysis (lack of postbaseline tumor assessment before withdrawal, n = 2; lack of postbaseline tumor assessment at cutoff due to insufficient follow up time, n = 8). One additional patient was excluded from the waterfall plot of best change from baseline (lack of evaluable postbaseline tumor assessment).

- Infusion-related reactions (IRRs) were reported in 16/22 (72.7%) patients
- IRRs were low grade with rash/urticaria, occurred mostly during the first infusion and were well managed with antihistamine prophylaxis, short pause in administration and slowed infusion rate. No patients discontinued treatment due to IRRs

Enrollment has been initiated for a Phase 3 registrational study to evaluate setidegrasib + mFOLFIRINOX/NALIRIFOX in 1L PDAC patients with KRAS G12D mutation (NCT07409272)

Presented at ASCO GI 2026. 1L: First line, DCR: Disease control rate, DOR: Duration of response, ORR: Objective response rate, PDAC: Pancreatic ductal adenocarcinoma, AE: Adverse event, SD: Stable disease, PD: Progressive disease, PR: Partial response, NE: Not evaluable, mFOLFIRINOX: Leucovorin, fluorouracil, irinotecan and oxaliplatin, NALIRIFOX: Leucovorin, fluorouracil, liposomal irinotecan and oxaliplatin

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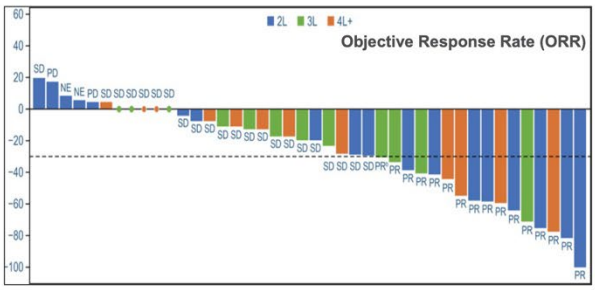
I will now revisit the data on setidegrasib in PDAC that we presented at the ASCO GI meeting in January, as mentioned earlier. In our Phase I study of KRAS G12D positive PDAC, the combination of setidegrasib and modified FOLFIRINOX as first-line therapy demonstrated antitumor activity and a manageable safety profile.

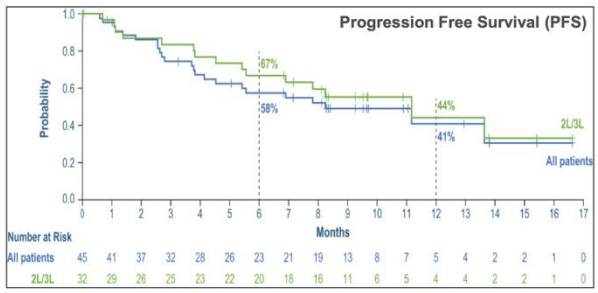
Among the 12 patients whose efficacy could be assessed, the objective response rate, ORR, was 58.3% and the disease control rate, DCR, was 83.3%. Infusion reactions were reported in 72.7% of patients. However, these were primarily low grade, occurred mostly at the first dose, and were manageable with standard supportive care. There were no treatment discontinuation due to these reactions.

Based on these results, we have initiated patient enrollment for a Phase III trial evaluating the combination of setidegrasib with a modified FOLFIRINOX or NALRIFOX as first-line therapy for PDAC patients.

setidegrasib (ASP3082) monotherapy demonstrates deep and durable clinical activity, with no new safety signals, in patients with advanced NSCLC with *KRAS G12D* mutation



ORR was 37.5% in the 2L/3L setting



Median PFS was 11.2 months in the 2L/3L setting

Data cutoff date: November 10, 2025

Dashed line represents 30% reduction in tumor size. ORR defined as patients with best overall response of CR/PR with confirmation, or without confirmation but with possibility of confirmation at subsequent assessment; One patient had an unconfirmed PR per RECIST v1.1

Data cutoff date: November 10, 2025

Median follow-up time, months (95% CI): All patients, 9.7 (9.1–12.4); 2L/3L, 9.7 (8.7–13.3)
For all patients, PFS rate (95% CI) was 58% (41–71) at 6 months and 41% (22–59) at 12 months
For 2L/3L patients, PFS rate (95% CI) was 67% (47–81) at 6 months and 44% (20–66) at 12 months

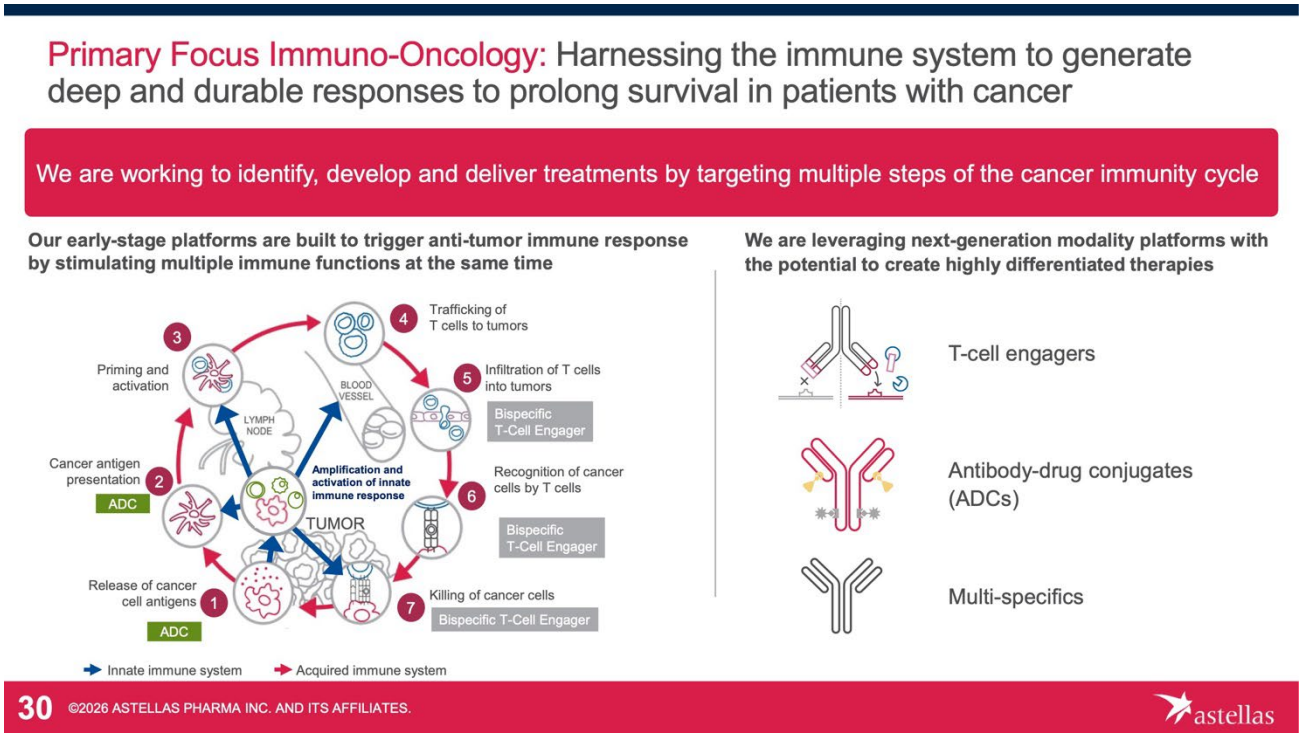
A Phase 3 registrational study to evaluate setidegrasib monotherapy in advanced NSCLC patients with *KRAS G12D* mutation, is being prepared for initiation in FY2026

Data presented at the European Lung Cancer Conference 2026; N Engl J Med 2026 Mar; doi: 10.1056/NEJMoa2600752
NSCLC: Non-small cell lung cancer, 2L+: Second or later line, 4L+: Fourth or later line, SD: Stable disease, PD: Progressive disease, PR: Partial response, NE: Not evaluable

Page 29.

Here, I present clinical data on setidegrasib in patients with advanced or metastatic NSCLC with KRAS G12D mutation. This data were presented at the European Lung Cancer Congress last week and simultaneously on the same day, published in the New England Journal of Medicine. In a cohort of 32 patients receiving second or third-line treatment, the ORR was 37.5%. As shown in the figure on the right, the median progression-free survival or PFS was 11.2 months. No new safety signals were identified.

Based on these results, we are preparing to initiate a Phase III trial of setidegrasib monotherapy in advanced or metastatic NSCLC patients.



Next slide, please. Page 30.

Next, I will explain our Primary Focus on Immuno-Oncology. Immunotherapy has significantly advanced cancer treatment but many patients still do not achieve sufficient efficacy, leaving a significant unmet medical needs. In its primary focus, we aim to achieve high therapeutic efficacy by leveraging diverse modalities that target both the immune system and the tumor microenvironment.

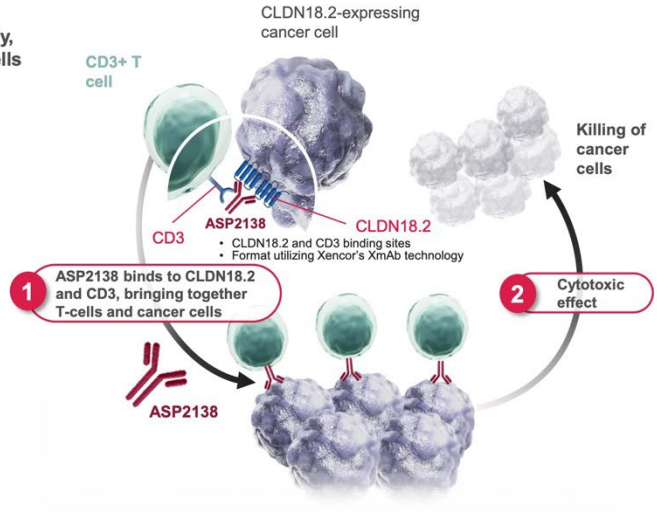
Specifically, we are utilizing next-generation platforms such as T-cell engagers, ADCs, and multi-specific antibodies to achieve more effective and sustained treatment outcomes for difficult-to-treat cancers.

I'll provide a detailed introduction to our Flagship program, ASP2138 on the next slide.

Our flagship asset, ASP2138, builds on our expertise from VYLOY

ASP2138 is a Claudin 18.2 (CLDN18.2) targeting bispecific antibody, helping to activate T-cells and enhance their ability to kill tumor cells

- **Target disease:** Gastric/GEJ (G/GEJ) adenocarcinoma, PDAC, and other tumors expressing Claudin18.2
  - ✓ CLDN18.2-positive patients: ~70% in G/GEJ adenocarcinoma[1] and ~60% in PDAC[2]
- **Clinical PoC achieved in G/GEJ adenocarcinoma**
  - ✓ Efficacy observed irrespective of CLDN18.2 expression or CPS status
- **Preparing to initiate Phase 3 trial in 1L GC**
  - ✓ Intent to treat population – patients with low-intermediate Claudin 18.2 expression not eligible for VYLOY
  - ✓ Primary analysis anticipated – FY2029



1. Gastric Cancer. 2024;27:1058, 2. Int J Cancer. 2013;134:731
GEJ: Gastroesophageal junction, CPS: Combined Positive Score (for PD-L1), PDAC: Pancreatic ductal adenocarcinoma, PoC: Proof of Concept

Next, please. Page 31.

ASP2138 is a potential first-in-class bispecific antibody designated to activate T-cells and damage cancer cells by binding CLDN 18.2 expressing cells to CD3 positive T-cells. CLDN 18.2 is expressed in gastric cancer and PDAC, which represent areas of high unmet medical needs.

We have already achieved PoC in gastric cancer and are preparing to initiate a Phase III trial targeting first-line treatment for patients with low to moderate CLDN 18.2 expressing gastric cancer who are not eligible for VYLOY. Primary analysis is anticipated in FY2029.

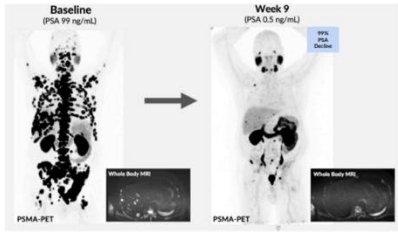
Advancing next-generation modalities in prostate cancer: Strategic collaboration with Vir Bio to advance PSMA-targeting dual-masked T-cell engager VIR-5500*

VIR-5500-V101 First in Human Phase 1 study: presented at ASCO GU 2026

- Dose-dependent anti-tumor activity (53% (9/17) PSA90, 82% (14/17) PSA 50) observed) at doses ≥3,000 µg/kg Q3W
- Well-tolerated with a favorable safety profile with no DLTs: low rate of ≥ G3 TRAEs without prophylactic corticosteroids or anti-IL-6 at doses up to 3500 µg/kg Q3W

Vir Biotechnology™

99% PSA decline; 63% decrease in tumor diameter and a complete resolution of liver lesions



Q3W Cohort 800/1500/3000 µg/kg

Case study detail:

- 63-year-old male
- High disease burden: liver and bone lesions
- 5 Prior Lines of Treatment
- Metabolic response of PSMA-avid bone and hepatic lesions

*Pending transaction closing
ASCO: American Society of Clinical Oncology, DLT: dose limiting toxicity, GU: Genitourinary, PSA: Prostate-specific antigen, PSMA: Prostate-specific membrane antigen, TRAE: Treatment-related adverse event

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Next, please. Page 32.

Our partnership with Vir Biotechnology is an example of how we are leveraging insights gained in the prostate cancer field to connect our cancer immunotherapy with actual clinical development. Under this strategic partnership, we are advancing the development of VIR-5500, which targets a novel immune-mediated mechanism. VIR-5500 is designed to incorporate a proprietary masking technology that keeps the T-cell engager masked until it reaches the tumor microenvironment, thereby reducing its effect on normal cells and minimizing side effects.

Currently, a Phase I study is underway as a monotherapy. An initial antitumor activity and a favorable safety profile have been demonstrated in patients with metastatic castration-resistant prostate cancer or mCRPC with prior treatment.

Findings shared at ASCO GU show target engagement and immune activation with manageable safety at doses evaluated today. It was announced in February. These results support the continuation of development and provide crucial data to inform future dose selection and development strategies.

Primary Focus Genetic Regulation: Treating neuromuscular and neurodegenerative conditions by correcting their underlying genetic causes

We are focused on making adeno-associated virus (AAV) gene therapies scalable and accessible to more patients, with the ambition to address both rare and common diseases

Flagship program

AT845: An **AAV gene replacement therapy** designed to address the **underlying cause of Pompe disease** – a devastating rare neuromuscular disease

In Pompe disease, the body cannot produce a functional enzyme, causing glycogen accumulation in muscle cells and progressive weakness

AAV gene therapy provides a healthy version of the gene which allows muscle cells to produce their own, effective enzyme

Status

- PoC analysis ongoing

Follow-on program

ASP2957: A next-generation investigational gene therapy for **XLMTM**

- Delivers a functional human MTM1 gene using a novel muscle-targeted MyoAAV capsid
- Developed via the DELIVER[1] platform, MyoAAV capsids are engineered for high muscle specificity and reduced liver targeting

Status

- ASP2957 has cleared IND, with first patient expected to be dosed in Q1/FY2026
- VALOR is a Phase 1/2 clinical trial evaluating the safety, tolerability, and preliminary efficacy of ASP2957 gene therapy in young boys with XLMTM[2]

1. DELIVER: "Directed evolution of AAV capsids leveraging in vivo expression of transgene RNA". Tabebordbar M, et al. Cell. 2021;184:4919–49382, 2. ClinicalTrials.gov NCT07052929
AAV: Adeno-associated virus, IND: Investigational New Drug, PoC：Proof of concept, XLMTM: X-linked myotubular myopathy

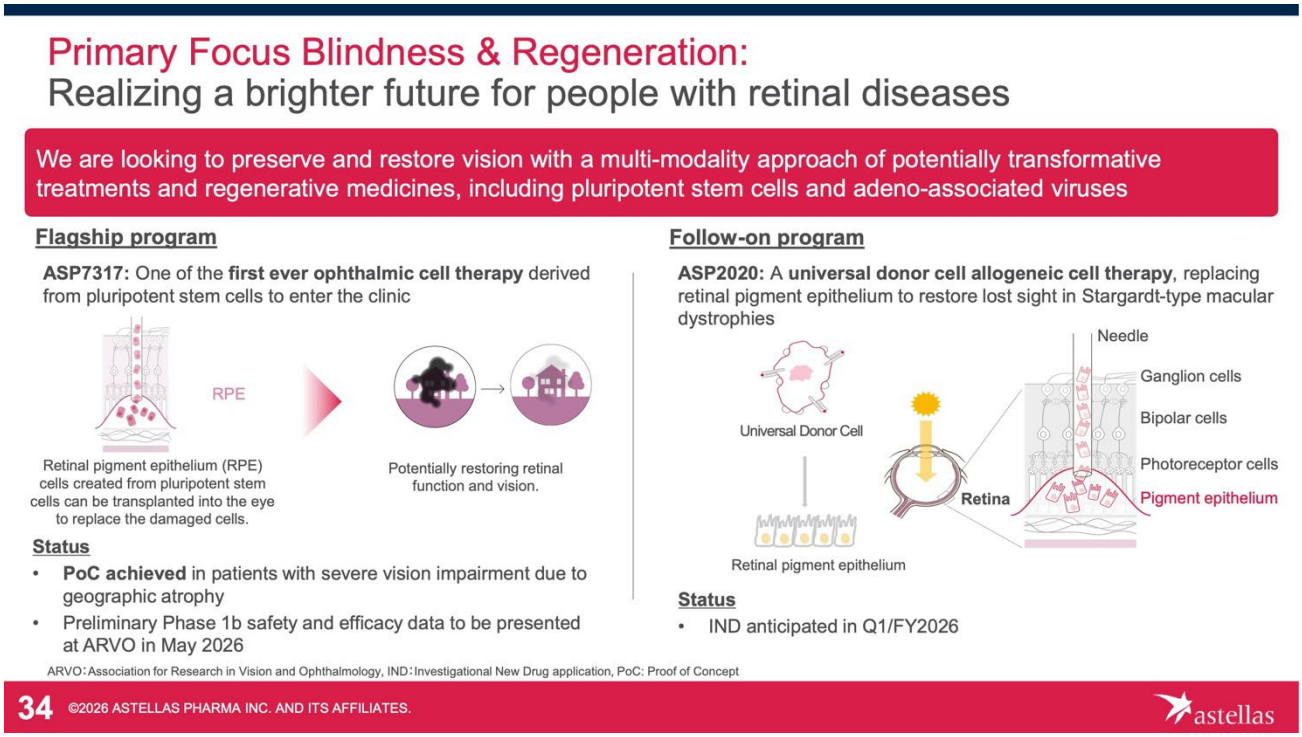
Next slide, please. Page 33.

Under Primary Focus, gene therapy, we aim to make AAV gene therapy, which modulates genetic causes of diseases, are more accessible to more patients. Gene therapy holds the potential to treat and address genetic diseases at their source for which many patients worldwide are eagerly awaiting new treatments.

However, the development of gene therapy presents various challenges in terms of drug discovery, development, and manufacturing. To address this series of challenges, Astellas has been building internal expertise in R&D and manufacturing while also collaborating with external partners to establish a platform.

Our flagship program is AT845, an AAV gene replacement therapy for Pompe disease. The final PoC decision is pending as we review additional analysis of data.

Furthermore, we are advancing follow-on programs for neuromuscular and neurodegenerative diseases and are also working on next-generation approaches such as MTM1, a gene replacement for X-linked myotubular myopathy, XLMTM using a novel AAV capsid.

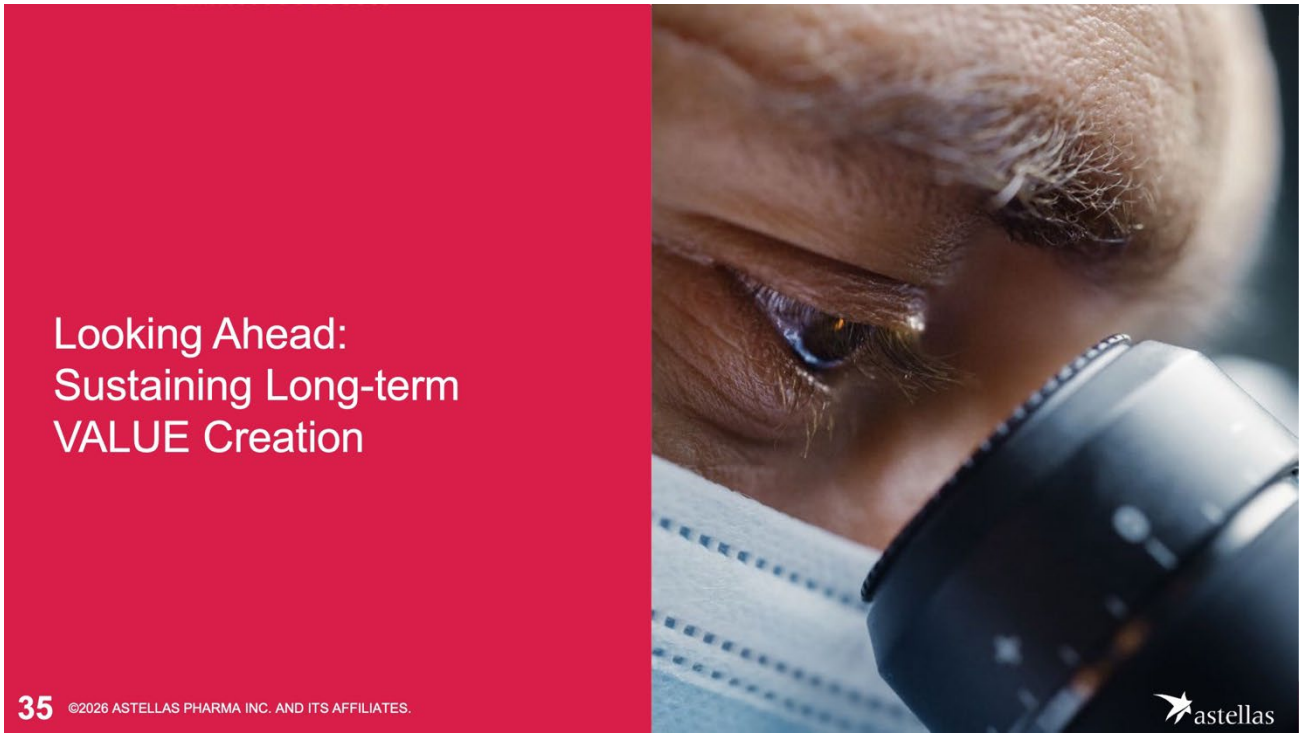


Next slide. Page 34.

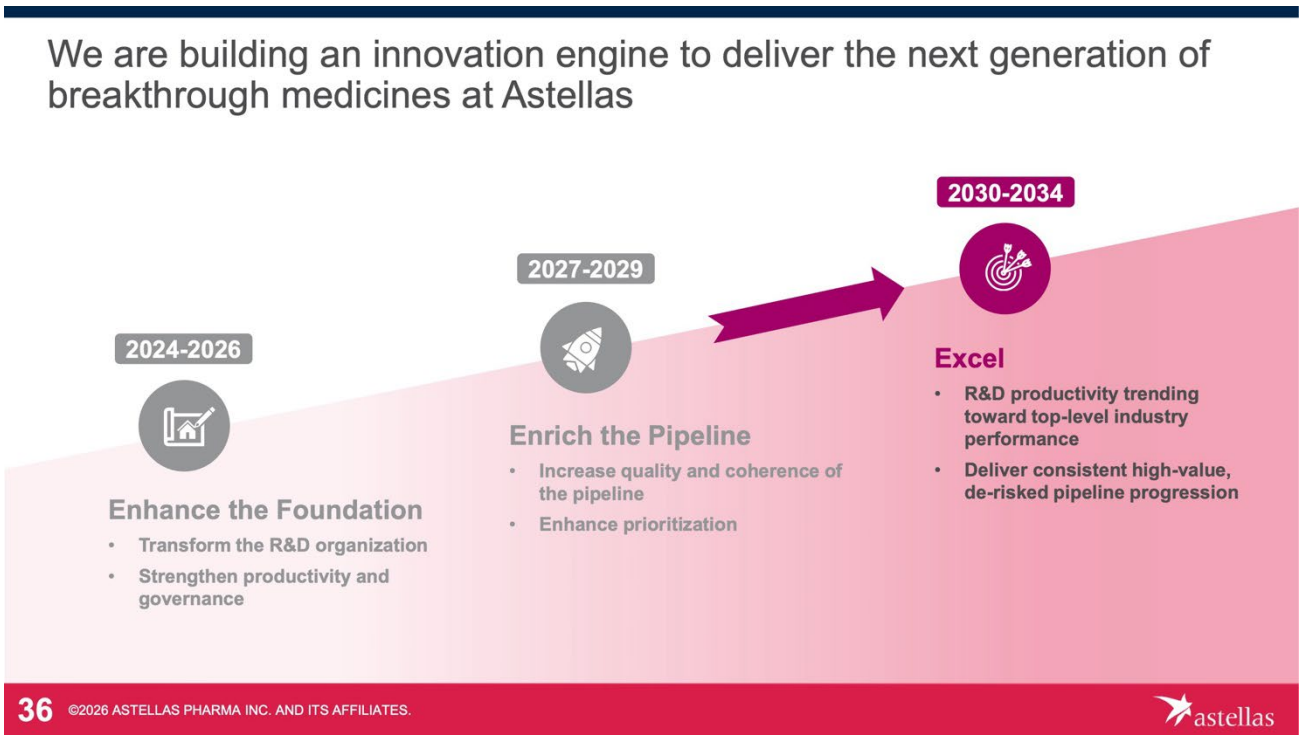
Next, we turn to Primary Focus, Blindness and Regeneration. Here, we are addressing diseases that lead to irreversible vision loss and those representing a significant unmet need. Our flagship program, ASP7317 is a pluripotent stem cell-derived retinal pigment epithelial cell designed to replace damaged cells in patients with geographic atrophy, secondary to age-related macular degeneration.

We have now achieved proof-of-concept, PoC, in patients with severe visual impairment, making a significant milestone. Additional Phase Ib safety and efficacy data are planned to be presented at the ARVO 2026, Association for Research in Vision and Ophthalmology, in May. Details will be communicated there.

Alongside ASP7317, we are also progressing ASP2020, a follow-on allogeneic cell therapy for Stargardt-type macular dystrophies, reflecting the broader applicability of this regenerative platform.

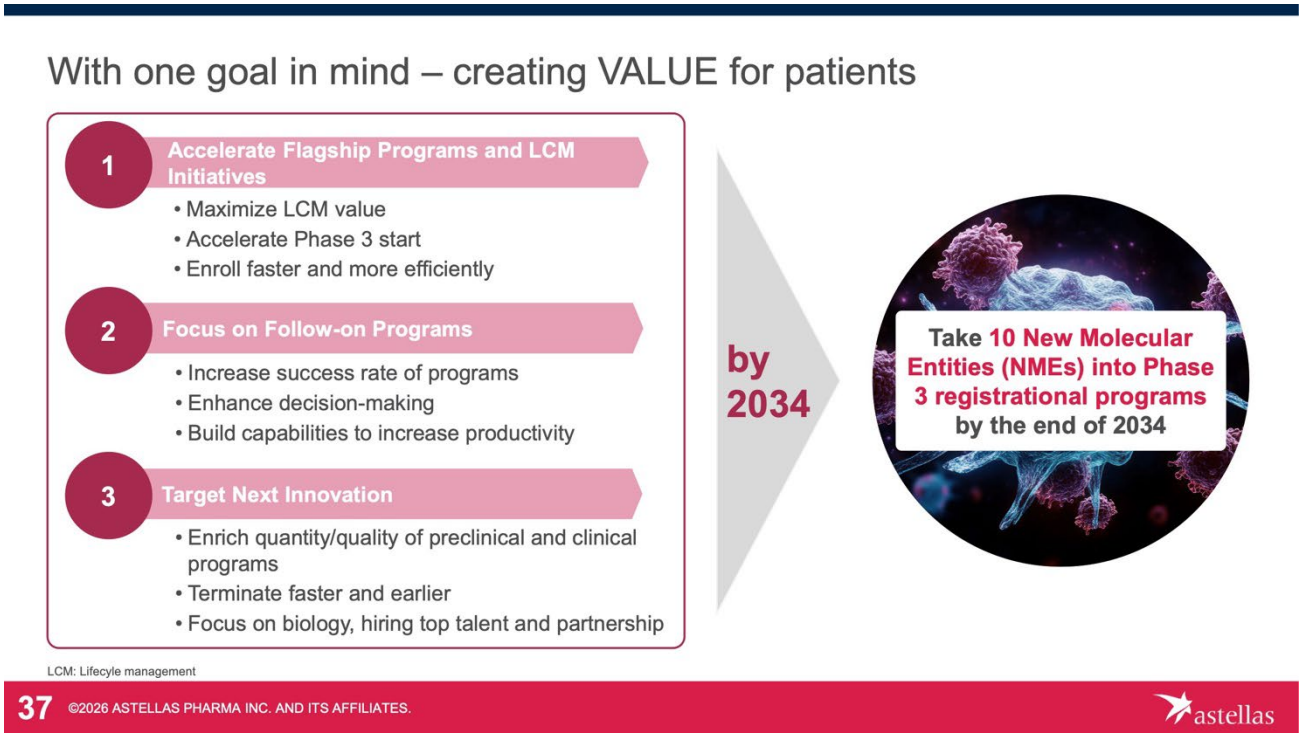


Next slide, please. Now I will use a few slides to explain direction of our research and development organization for sustaining long-term value creation.



Next slide, please. Slide 36.

We anticipate that the R&D transformation and pipeline progress outlined thus far will form the foundation for sustained growth throughout the 2020s. Until 2029, looking ahead to the early 2030s, we aim to significantly improve productivity to reach the top tier of the industry, building a pipeline of high value-added products and accelerate R&D.



Next, please. Page 37.

We outline our initiatives focused on growth in the 2030s. Firstly, accelerate the development of our flagship programs and our life cycle management initiatives. Next, to expand the pipeline of follow-on programs, we will continuously improve program success rates and decision-making based on data and continuously improve entire R&D productivity. Furthermore, looking ahead, we aim to advance 10 NMEs into late development stage by 2034.

Page 39.

Astellas is undertaking a fundamental transformation of its R&D organization with the aim of rising R&D productivity to the top tier in the industry. We are strengthening our pipeline by pursuing higher-quality science through an end-to-end operating model and prioritize investment in modalities and platforms that differentiate our portfolio.

Furthermore, by building a robust drug discovery platform, we will generate multiple high-quality assets, thereby maximize pipeline value and accelerate R&D. We will continue to deliver sustainable growth and meaningful outcome for the patients and the value.

This concludes my presentation. Thank you.

Kato [M]: This is all from us as a presentation. Now I would like to entertain your questions.

Let's start. Thank you very much for waiting. First question is from Citigroup Securities, Yamaguchi-san, please.

Yamaguchi [Q]: Yamaguchi from Citi. For confirmation. ASP7317, in the past, the data of improving the visual has been shown. You've been saying PoC judgment. This time is the very first time that you mentioned the PoC is achieved. Is that understanding right?

Okamura [A]: Yes, you are right.

Yamaguchi [Q]: The second question. ASP3082 lung cancer data is introduced. Cross-trial comparison does not have meaning and there is no significance in early stage. However, with a simple comparison, ORR and DCR compared to revolution, what is happening? How do you view about this? For the PDAC, the data was really good. But from your perspective, this comparison is has been expected, good, unfavorable? Would you make a comment?

Taniguchi [A]: Thank you very much. The data shown for setidegrasib this time has been shown, ORR is 37.5%. First of all, existing product therapy, in other words, comparison with the chemotherapy is the right way to be done. 37.5% of ORR compared to the chemotherapy such as docetaxel, this response rate is extremely high. PFS, this time in median.

Well, it's open level, but this might be adjusted for referential, but 11.2 months. So, docetaxel [inaudible] we referred to, it was around four months of PFS reported. Compared to that, it's more than twofold of PFS extension. That's the data we have. With this, we are getting into Phase III. We are going to make appropriate study design for Phase III. That's what we are planning currently. Thank you very much.

Yamaguchi [Q]: Page 19. You showed a chart of the portfolio review and especially one, two and three, you made a comment about those. It might be difficult for you to talk about the details, but this one, that is the strategic deprioritization. That means in the beginning, you didn't expect, but the situation was changed. But what are the major reasons of this deprioritization? From the beginning, possible value is not really clear or the competitive superiority was lost? Could you share with us? Thank you very much.

Okamura [A]: Basically, these are including the very early stages, not even in the clinical stage. Targeted product profile are not necessarily completely fixed and such kind of projects are included in here. Based upon that as an assumption or the condition, please listen to my explanation from here.

As you know, in a preclinical study, there are various things we have to prove, and we will go through the studies. There are things that this is okay. This is not okay at all. There are something in between. For those, we are going to do some additional studies to identify and make a decision if we can go for that or discontinue that.

As has Taniguchi repeatedly been saying, the discipline is important here. If discipline is not strong enough, you try to hang on the projects so that the project can survive as long as possible. But this time, completely, we review such discipline. If such factors are not satisfied, we should make a decision of discontinuation. We actually execute that approach.

For each project, the important factors are different and that is different depending on the target disease. If the modality is new, we thought in the beginning, it was really good. But once that development, the efficacy

is not really expected. We have roughly six factors for the project evaluation. If five criteria out of those are not satisfied, rather than dragging the development on that, you make a Board decision to discontinue so that you can allocate the value of your asset to the more potential asset. That's described here. Did I answer your question?

Yamaguchi [Q]: So, you have done that for 2024, and that is going to be continued. You expect a further improvement in efficiency?

Okamura [A]: Yes, that's right.

Kato [M]: Let's move on to the next. JPMorgan Securities, Wakao-san, please.

Wakao [Q]: JPMorgan, Wakao speaking.

My first question is also about setidegrasib. NSCLC PFS data is really good. So far, just like Taniguchi-san mentioned, it's not inhibition, but the degradation is suggesting the continuous efficacy, and it happened, it was really surprising. This might be the same question, but setidegrasib, how do you view about the possibility of being the best-in-class within NSCLC? I believe that it is clear it's going to be the first in-class. But for Revolution pan-KRAS, compared to that seemingly its good. Their PFS data is not available yet. So, I cannot make the head-to-head comparison, but we would like to know how you feel about that.

Another question is about PDAC. Second line, third line OS 10.3 months data is available published in New England Journal of Medicine. For this second line, third line, comparator is not available in Revolution. How do you evaluate this data? Compared to chemotherapy, it is superior, which is clear. But against the coming next-generation types, how do you view it?

Taniguchi [A]: Thank you very much. Wako-san is correct. In comparison to Revolution, we are paying close attention with interest. For those pharmaceuticals, data is not much available. So, head-to-head comparison is rather difficult.

Their pan-KRAS oral inhibitor, what is the situation vis-a-vis that is the area that we are paying attention to. The differences, in particular in lung cancer, as we look at the data available on ORR, not much difference between the two versus chemotherapy, it’s much better. But according to the publicly available data of them, ORR, not big difference between the two.

As for PFS, duration of the effect, their data is rather limited. What is the situation in comparison to that? That is not so sure. But our hypothesis that is KRAS G12D to decomposing the KRAS protein itself is the strategy. Our hypothesis is being proven, meaning that the durability of the effect is maintained very significantly. That is our impression.

Protein degrader and the KRAS suppressor or inhibitor, if you make a comparison, people often talk about the following. As for inhibitor, the resistance occur. That is the issue. How they are going to overcome them, it's something that we don't know. But the different mutation of KRAS or different pathway may appear that is regarded as a challenge versus our KRAS targeted degrader, we decompose, degrade protein themselves. So, refractiveness or resistance is less likely to occur in our modality.

Therefore, we are going to accumulate in particular Phase III data moving forward. First and foremost, for non-small cell lung cancer, setidegrasib would have a potential to become best-in-class. That is what we want to pay attention to. This is the first point.

PDAC and pancreatic ductal cancer carcinoma. In that cancer, there was no drug that reported to be efficacious in second line, third line. Setidegrasib and Revolution Medicines’ inhibitor data were reported this

time. More than expected, we got the feeling that it is more effective than we expected. In comparison to second line, third line, with the combination of chemotherapy, higher efficacy is already shown. In the pancreatic ductal cancer development strategy focusing on first line is likely to be a path or is the current path.

So far in the pancreatic cancer, currently, as you know, other than chemotherapy, there is no medication which shows efficacy so far. So, it's very promising. Our compound is very promising. But given the current landscape, so combination of setidegrasib and chemotherapy could be the main line of first line of pancreatic cancer. With that in mind, we want to design the study, and we want to conduct enrollment of the study, though we have already started screening.

Wakao [Q]: That is very informative. Second, on page eight, in the midterm exploratory meeting, I believe you're going to explain more in detail. But after the XTANDI LOE, the growth, you have strategic brands and the pipeline. As for your pipeline, so the project range that you have highlighted this time will constitute the main body of the pipeline, and you often talked about discipline this time. Beyond FY2027 onward, OP margin, 30% is going to be sustainably generated. As we have the late-stage subsequent development, are we able to maintain OP margin 30%?

Okamura [A]: This is not a precise diagram. This is just an illustrative chart. With this assumption, I would like to explain, as Taniguchi mentioned, life cycle management of strategic brands is included in center in red. The light pink, the pipeline, in this chart, according to Taniguchi explanation, are derived from primary focus or depending on the situation, they are the ones that we have already acquired from outside. Those constitute this pink pipeline.

As you mentioned, from tomorrow, we will enter into FY2026. From FY2026, those which we obtained PoC will move into late-stage developments. As you mentioned, there will be a high amount of R&D expenses that we will incur. Therefore, we have several meaning of discipline when we say discipline. One, even if things are advanced, we want to prioritize and we inject our capital resources and prioritize ones. It is important to create new things one after another. But after you give it a try, if it doesn't work, we want to give it up, meaning that we don't want to linger on for a long time. That is another meaning of discipline.

We want to transform advancement of science to value creation. That is our vision. We don't want to sacrifice R&D. We want to improve work efficiency in other areas. We want to invest in R&D while securing profit. That is our mindset in building the next midterm plan or CSP.

Depending on the situation, we don't do this to reduce the ratio of R&D expense in sales. But depending on some projects, we want to reduce the R&D expense so that we don't want to sacrifice our future growth. There is some room for us to consider that. That's all.

Wakao [Q]: OP margin, 30%, that is continuously you are going to target. Is this understanding right?

Okamura [A]: So far, as we mentioned, by FY2027 we achieved 30%. Afterwards, we are going to maintain that. That's what currently we are thinking.

Kato [M]: Next, BofA Securities, Mamegano please.

Mamegano [Q]: BofA Securities, Mamegano is my name. I have two questions.

First is about the prioritization. In your company, there are several points of the evaluations. Based upon that, you prioritize your projects. I'm referring to page six. There are four primary focus that you have. The rearrangement of this primary focus and are you going to make major changes? That's a question that I have. That's because they are currently have some oncology update, and I believe you're progressing quite well.

Therefore, are you going to focus more on oncology? Are you getting into the gene therapy? You are going to add additional primary focus here? That is the first question for me.

Okamura [A]: Thank you. Probably, as you know, this focus area approach, we name it as a focus area approach and officially communicate to you that happened around 2015 and 2016. Considering from them, we see there are some changes in the primary focus as well.

For example, we had primary focus, four of them at the very first, the antigen-specific immune modification that is a primary focus that we had and aiming at the allergy treatment. Certain specific modality is utilized there. Based upon the clinical trials, we decided to discontinue that primary focus. This gene therapy that was not included primary focus, but rather candidate, now it is officially within this primary focus. We had mitochondria. That was another primary focus, but we couldn't come up with the expected result or efficacy. That's why we discontinued that as well. The primary focus is not something that we continue to stick to those once decided, but rather from those primary focus, if multiple expected assets worth continuing available or not. Based upon that, we make a decision about the primary focus.

For each of these four primary focus, I think there's still the expandability. But as Taniguchi explained, with the Innovation Labs, the science is getting newer and newer and how we connect those with drug is our work. The biology is sufficient and robust and modality is appropriate. Also clinically, it is proven. We have this triangle. It's not just one. If we come up with several programs as a primary focus with pivoting around these triangles. Based upon that, we decide to prioritize, deprioritize, and so on and so.

Mamegano [Q]: Next is about the specific project that is setidegrasib. PDAC first-line Phase III studies started, I believe. The base treatment is FOLFIRINOX and NALIRIFOX. Those are quite intensive chemo. Even with this chemo, the efficacy is achieved to a certain extent. The Revolution pan-KRAS, Gem-NabP is the comparator. Gem-NabP base is probably easier comparator to see the efficacy. But here, you pick up a G12D alone as mutation, meaning that you have confidence in the efficacy. Do you think with the current approach, you can show the clear advantage in efficacy?

Taniguchi [A]: PDAC, first line, as has been explained, the combination with the chemo is the center of our strategy. The selection of chemo is FOLFIRINOX and NALIRIFOX are selected because those are mainly utilized in the Western countries. Revolution, Phase III study, if we look at Gem-NabP chemo and monotherapy, and combination with chemotherapy. They have three arms within the study design. That's what we've heard.

Fundamentally, there might be a bit of a difference because their product is a pan-KRAS and ours is targeting only KRAS G12D mutation that accounts about 40% of the PDAC. That is the focus. The second has been explained already. In the treatment of PDAC, the biggest issue is the continuation of sustainability of the efficacy.

Needless to say, at the same time with the deep regression of the tumor, how long you can extend or sustain the efficacy. That is the key. Looking at the past PDAC study, in the beginning, initial phase, efficacy is higher. However, looking at the sustainability of the efficacy, meaning that there is no impact on to the extension of OS.

Therefore, for the PDAC, the continuation of efficacy, especially impacting on the OS, that is the most important key factor that we need to pursue to. Based upon that, we decided this study design. Revolution takes a different strategy. Therefore, ultimately, I think we can see if we make a head-to-head comparison.

Kato [M]: Are there any other questions? Next question from Nomura Securities. Mr. Matsubara, please.

Matsubara [Q]: Matsubara from Nomura Securities. ASP3082 is my question. Good results was shown. Congratulations. I would like to ask about adverse events with administration, infusion-related reaction. 63%

stopped the administration in a few cases, transaminase or neutrophil reduction was observed. So will that be the hindrance of development? As for infusion, every time of the administration, infusion-related adverse events may have reduced. Can you comment on those?

Taniguchi [A]: As for adverse events, we are focusing very much. As was asked earlier, the selection of combination therapy, the chemotherapy that we select, they are a very potent chemotherapy. Because of that, hematology-related adverse events, including nausea and vomiting, gastrointestinal AEs are observed. However, as for setidegrasib, we showed you mono data earlier. But in practice, the major AE was not observed. AST/ALT increase was shown in some of the cases. But there are not many cases that stopped administration as a result of AST/ALT increase.

As for first-line PDAC treatment. First, whether we are able to show effect or not accounts. Will there be continuation of effect. These two keys are crucially important in pancreatic cancer treatment. We also have to think about managing AE. We have to strike a balance between the two. Chemotherapy-related adverse events or setidegrasib AEs, there are such AEs, but continuous treatment is possible in our regimen. For example, due to AE, if we are not able to administer continuous setidegrasib? That is not the case. We don't have such a case.

Matsubara [Q]: ASP7317, PoC was achieved. Congratulations. For a launch, development is smoothly underway. How do you differentiate with the IZERVAY. Right type IZERVAY and the severe type ASP7317, can you tell us how you demarcate with IZERVAY and ASP7317?

Taniguchi [A]: As for IZERVAY, as you know, C5 RNA aptamer. In the past GATHER1, GATHER2 study, I think you understand the situation. In the AMD, progression of geographic atrophy was inhibited. Such data was clearly shown. GA secondary to AMD, the condition deteriorates and it doesn't improve. That is a condition. Stopping the progress of the disease is clinically very significant. That is the reason why we have developed IZERVAY.

In comparison to IZERVAY, ASP7317 though the number of patients was very limited, we showed some data in the past. The best corrected visual acuity was likely to be improved. We saw such tendency. With a very different efficacy endpoint, we are going to assess the efficacy.

Second point. IZERVAY, suppression of the disease progression. When the visual acuity is impaired, it's rather difficult to use. The early to moderate patients would likely to receive IZERVAY moving forward as well. In comparison to that, as for ASP7317, it's a cell therapy and the patients for PoC this time, the visual acuity degradation was very significant. Meaning that the severe visual impairment patients were the target for PoC assessment. We are going to go into Phase III from now on. But as for the target patients, those patients with advanced visual impairment, meaning severe geographic atrophy patients, would likely to be enrolled.

What will be the overlap between IZERVAY and ASP7317? We don't see much overlap. Meaning that IZERVAY is used for early or moderate visual impairment , but when the condition exacerbates, improvement of visual impairment can be tried with ASP7317.

Matsubara [M]: Thank you very much for your explanation in detail.

Kato [M]: Next, UBS Securities, Seki-san, please.

Seki [Q]: UBS Seki. Going back to page 19, 2x2 chart that you showed earlier. The portfolio management, you strike balance between art and science, and it's always a challenge in this industry, as I observed. Now, unless you get PoC, I believe it is rather difficult to understand the value of the program because you'll never know until you see PoC. Practically, program value and the success probability in order to improve the volatility, how, what kind of measures you take?

Okamura [A]: Horizontally, this is value that means the value when we make success. Usually, if it's early, it is costly beforehand. The sales increase and profit increases that toward the launch. With a discount, the value is lower and with the probability of the success is multiplied. But this is NPV with a success scenario. We are going to study what's going to happen to the future. If the thing is successful, then how high the value would be, meaning the left bottom, even they make a success, we will not be able to expect a higher value. That's why they should be deprioritized.

Those quadrants that we see, X equals something, Y equals something, in terms of the program value. If you apply that to all the Primary Focus, then the oncology product go toward more right. If it is a rare disease, because the targeted patients are limited in number, no matter how successful you are, you cannot come closer to the right side near to the oncology. Just like you mentioned, it's a world of art. If the Primary Focus that should be survived or killed, and for that decision-making and if you apply the universal ways of evaluation, all the value of the rare disease is going to be reduced. How to evaluate the primary focus? Well, according to this, something left is going to be discontinued, but the right side is going to be continued.

For the Primary Focus, each of them are not necessarily evaluated in line with the exact same axis. That's the way we are working on currently.

Taniguchi [A]: One thing some technically, that is already wholly explained by Okamura, improve the probability of success and improve the program value, those are important. Then as R&D, what should we do?

First, the clear drawing of the target product profile, TPP. With having that, you can specify the value further. You can have a tangible view of the value. In order to achieve that, you design nonclinical and clinical studies.

As the result of that study is expected or assumed. If the result is in line with that assumption or expectation or not, then you decide the priority of the programs. That's the approach we take in R&D as it's been described in page 18. Decision-making based upon the data. We would like to foster a data-driven culture. We clarify the TPP, the drug we want to make in the very beginning, in order to achieve or realize that such and such data is necessary. That's why you design the nonclinical and clinical trial study.

Of course, it doesn’t always exactly match the expectation we had. We have to consider about the achievability of that target. The criteria is clarified, then based upon that, data is collected and make the judgment. As Okamura mentioned, so we can work in line with the strong discipline, we are trying to make the culture.

Seki [Q]: Now ASP3082 protein degrader, the response of the duration, those are not really ringing a bell for me. For example, mechanism of the resistance, it's not inhibitor but it's a degrader. Are there any change differences in terms of the biological perspective?

Looking at the New England Journal of Medicine, degrader probably KRAS G12D degradation is 70.6% or so. I don't think it's not 100% of the degradation. How can we make a decision about this or make understanding about this?

Taniguchi [A]: Degrader resistance mechanism that is under the study these days, not yet published. But there are some biological changes likely to cause the degradation is started to be understood. As soon as the data is ready to be published, we can share that. But as you know, inhibitor and the degrader, we have a strong impression that these two are quite different, extremely different. Also, the resistance mechanisms are different. The way to overcome such resistance are also different than we assume. In clinical trials, in early clinical trial with the combination, such idea is applied for this development.

Regarding second question, that is a KRAS G12D. To what extent it should be degraded to demonstrate the efficacy. Regarding that question, as you see, according to the data we shared with you this time, 70% to 75% protein degradation is observed in data. If that is sufficient or not, if we refer to the basic data, the suppression of KRAS pathway to that extent because the tumor goes to the apoptosis and such kind of data is available. This level of suppression or efficacy for the protein degradation, we believe that a sufficient tumor suppression can be achieved.

On top of that, it's sometimes difficult to achieve with the mono. In order to accelerate the efficacy, the chemotherapy combination is used. In line with that, KRAS is also suppressed. They have the add-on effect or the synergistic effect. That's the way it works and the initial data shows that as well.

Seki [Q]: Last question. Simply, this was not talked about ASP2998 ADC. Dual payload is attached perhaps was presented at the AACR. Can you explain differentiation of TROP2 is available in some numbers? ASP2998, as you mentioned, at the end of April, at the AACR. First nonclinical data is likely to be presented.

Taniguchi [A]: ASP2998, targeting TROP2, so-called dual payload ADC. What do we do with dual payload-based ADC? STING agonist is attached, so which we'll announce from now on. As you are going to look at the AACR data, you will know that targeting the existing TROP2 ADC, If you look at the result of that, you will, I think, understand why we develop this ASP2998.

Kato [M]: Next, Morgan Stanley MUFG Securities, Muraoka-san, please.

Muraoka [Q]: Morgan Stanley, Muraoka is my name. ASP7317, the maximum visual acuity improvement was obtained in severe patients. Golden Week, I'm very much looking forward ARVO data. What will be the next step? Is my question too early to ask. But as you said it's an extremely important success, so I thought you were able to skip significantly. But can you give us a color to what you said?

Taniguchi [A]: Muraoka-san thank you for a good question. It's very difficult for me to answer that question. But what we can tell is that the PoC study, based on the data, we nurture the culture for decision-making. Clear PoC criteria is just determined in advance. Based on that predetermined criteria, we overcame. That is why we declared PoC. This is the first point.

Second point, I would like you to look at the ARVO data first and foremost. I hope you will have high expectation to that data. As for future development, with the FDA US we have already started interaction. Through this interaction with FDA, what kind of development strategy can we set up, that is being considered within the Company. Not only in the US, but moving forward in Japan, also in Europe and in China, we will engage in worldwide development, most probably. We hope to engage in global development, hopefully. But looking at the region and the countries, how will development be accepted. In the early part of FY2026, we want to scrutinize those things.

Muraoka [Q]: Phase Ib, you showed data in small amount, but my impression is that it's taken time significantly. But next step starts. When do you think we will be able to hear the answer for that? In 2028, 2029, Phase III results will be available, but this ASP7317, next step update will occur in the same timing? Or do you think it will take a longer time?

Taniguchi [A]: As mentioned, next fiscal year, which will start tomorrow. H1 of next fiscal year, we will complete the interaction with the authorities. In the latter part of next fiscal year, we should be able to talk about development plan.

Muraoka [Q]: Understood. At that time, a program will be updated. ASP3082, colorectal cancer, second-line stoppage. Can you share with us the background of this? G12D was that the simple reason? Or is that a different reason? What is the background of making decision to stopping?

Taniguchi [A]: As you know, KRAS G12D, the mutation is apparent in 15% of the colorectal cancer, and colorectal cancer is a big population. In parallel to lung cancer and PDAC, in Phase I, we looked at the efficacy in colorectal cancer. Unlike PDAC and small cell lung cancer, the situation of colorectal cancer is a little different. As soon as we are ready for data, we are going to present. But as of now, we have not shown efficacy that encourages us to go forward with colorectal cancer with ASP3082. Based on data, we made the decision.

Kato [M]: Thank you very much. We have about six minutes. Because a lot of people raising hands and I would like to ask a lot of people to ask questions. From here, we would like to ask you to ask just one question.

Now, Sawada-san from JPMorgan Asset Management, please.

Sawada [Q]: I would like to ask about the Claudin 18.2 and the peripheral of that, which is not really mentioned within this meeting here today. The low to mid expression of the Claudin 18.2 is the target for the development at this time. VYLOY ILUSTRO Cohort 4b result, that is a really good data. But to put it in another way, ASP2138 development on a highly expressed patient is likely to be the similar result with the VYLOY. That's why you decided in this way or ILUSTRO Cohort 4b result, how have you evaluated? How do you differentiate? Would you please mention something about here?

Taniguchi [A]: Thank you very much. Regarding VYLOY, ILUSTRO Cohort 4b came up with really good data. Claudin 18.2 highly expressed patients and CPS also highly expressed. That's the target of the development currently. For ASP2138, as the first indication because our strategy is to do the development for the high unmet needs. Because Claudin 18.2 high expressed is already developed for VYLOY, so there are patients not indicated for VYLOY that is low to mid expression that accounts for about 1/3 of the gastric cancer patients. We thought it is a favorable way to go for further development.

For the Claudin 18.2 high expression, what should we do now for the future? Also, considering the IV administration for VYLOY and such characteristic size it has, which is leading somewhat to the difficulty of management of the side effects. For this ASP2138, even with the highly expressed patients, the sufficient efficacy, safety, and convenience need to be observed. When it comes to subcutaneous, that is useful. Considering all the factors, we would like to consider if ASP2138 is needed to be developed for highly expressed patients as well or not.

Sawada [Q]: Understood, thank you.

Kato [M]: Next, Barker-san from Jefferies.

Barker [Q]: Barker speaking, from Jefferies. My question is about ASP2138 Phase III study. Claudin 18.2 expression, the minimum threshold, how are you going to set that?

Taniguchi [A]: Thank you for the technical but important question. In particular, the middle expression is rather easy, but Claudin 18.2 low expression, what is the degree of that? I think that was your question. According to the Phase I study that we have conducted, a detailed analysis of that was made.

We are consulting pathologists to identify a cutoff to determine low expression or 0 expression to delineate between the two. Against this, categorization of low expression cutoff value was set up. By using that, we plan to start Phase III study. The details is described in protocol, but we refrain from informing that yet.

Barker [Q]: Analysis per expression level will be possible. Do you have a plan to secure the power for that?

Taniguchi [A]: Yes. In our protocol, at the central level, the expression will be confirmed. As for enrollment, as far as I'm informed, the site data will be also used based on the confirmation of expression. In Phase III,

middle to low expression was observed to see effect. Cutoff value will be confirmed and then we will file submission.

Barker [M]: That's all from me.

Kato [M]: Now in the interest of time, we would like to make the next question as a final question. Sogi-san from Bernstein, please.

Sogi [Q]: In development, AI usage is my question. On page 21, you showed introduction of technology. Study design platform is described on page 21. Honestly, I'm surprised because in the area of development, of course, in the drug discovery also, but in development, the optimization of sites and acceleration of patient recruitment and patient management as well as data management, of course. For the regulatory document preparation, it seems that AI is very useful in those areas. But as of now, what's shown on the slide is the approach that you are going to address at Astellas. As for cost and timeline, I believe there will be impact of AI. That's my view. But how much cost saving as well as shortening of timeline would you like to achieve? In 2030 or 2035, as of that timing, what is your idea?

Taniguchi [A]: Thank you very much. Evinova AI-native study designer, that is quite interesting. There are AI agents and they have a discussion of a platform on the design of the protocol and that the minutes is generated. That is quite interesting. That's why we put it in this slide.

But as you mentioned for the development, usage of the AI is already a part of our day-to-day work. I didn't explain about that. Two years ago, and since then, protocol informed consent and all such clinical trial documentation translation into each country's language that is quite cumbersome work. But with using AI translation, we were able to accelerate the speed.

For example, for the translation from Japanese to English, it took about two months, but using AI, it can be completed within just a couple of days. Of course, the final confirmation is going to be done by humans. Technical language translated into easy to understand for the patients. Their large language model is utilized and that is completely utilized, but it's already part of our job. That's why I didn't focus on it.

Also, CSR generation each team is already using AI for generation of the report. General document creation is a way that we use the AI, just our day-to-day work. AI agent is quite interesting. This might be the response to the question of your second question. Towards the future cost reduction and time reduction, we can extremely expect on AI in that terms.

We are working on the confirmatory validation work. After that, when we are ready, we would like to share where we would go.

Kato [M]: Thank you very much for your participation and asking a lot of questions. Now time is up.

With this, we would like to close this R&D Day presentation. Thank you very much for your participation.

[END]

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