

OSE IMMUNO
THERAPEUTICS



Delivering on Our 3-Year Value Enhancing Strategic Plan

2026 Q4

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A Business Oriented Team to Leverage OSE's Leading Research and Development Capabilities



Marc Le Bozec
Chief Executive Officer

- Currently supports numerous biotech companies as an advisor, board member and investor
- Previously created and managed two biotech investment funds within Financière Arbevel
- Former CFO of Cellectis



Thomas Gidoïn
Deputy Chief Executive Officer / Chief Financial Officer

- 15+ years in pharma / biotech
- 10+ years as CFO in both private and public biotechs, Euronext and US Nasdaq IPOs



Silvia Comis, MD
Chief Clinical and Medical Research Officer

- 30+ years of pharma experience
- Previously held positions of Senior Director COE, European Head of Early Products Medical Affairs and Clinical Development in Oncology
- Certified pharmacologist and endocrinologist



Jean-Jacques Mention, PhD
Chief Business Officer

- 15+ years of academic research in Immunology and virology at Necker-Enfants Malades Hospital, King's College of London & Institut Pasteur of Paris
- 10 years' experience in BD and innovation



Aurore Morello, PhD
Chief Scientific Officer

- 7 years of academic research in Immunology and oncology at the Memorial Sloan Kettering Cancer Center in New York and the French National Center for Scientific Research (CNRS) in Paris
- Joined OSE in 2016
- PhD in immunology and oncology



Our 3-Year Development Plan Focused on Shareholder Value

Large Partnered Indications vs Smaller Go-Alone Indications

Strategy to maximize Return on Investment while managing risk:

- Large indication assets to be developed up to end of Phase 2
- Smaller indication assets to be developed up to commercialization

3

Development Strategies

Lusvertikimab in Ulcerative Colitis

Strong Phase 2 data as IV
Reformulation as SC by mid-2027
Potential to be licensed out

Multi-Billion potential

Lusvertikimab in Chronic Pouchitis

Rare/Specialist Indication
Target population 45k patients
Phase 2 to start in Q1 2027

Tedopi® in NSCLC

1st Phase 3: mOS benefit vs SOC
2nd Phase 3: readout Q1 2028
Potential approval in 2029

Multi-Billion potential

Strong Pharma Partnerships Capabilities

Proven ability to deliver attractive partnerships

Over €150m in upfront received and over €2.1bn in potential milestones + tiered royalties via partnerships with AbbVie, Boehringer Ingelheim and Veloxis

Multiple Key Inflection Points Over the Next 24 Months

Key clinical announcements at least every 6 months over our 3-year development plan

Clinical Pipeline Focused on Achievable Deliverables

3-Year Plan Focused on Proprietary Assets

Product Candidate	Target	Indication	Pre-Clinical	Phase 1a/1b	Phase 2	Phase 3	Addressable Market	Upcoming Milestones
Lusvertikimab SC	Anti-IL-7R	Ulcerative Colitis				Ph 2 completed in IV SC ready mid 27	\$1bn+	Ph 2b/3 to be licensed out or financed
Lusvertikimab IV		Chronic Pouchitis					45k patients	Ph 2 interim read-out Q4 27
Tedopi	Neoepitopes immunotherapy	NSCLC Mono post-CT-ICI 2L (US Orphan Drug Designation)					\$1bn+	Futility Analysis Q1 27 Ph 3 read-out Q1 28
		Pancreatic cancer Combo (ISS)					\$500m - \$1bn	3-yr LT Survival H2 26
		Ovarian cancer Combo (ISS)					\$500m	OS data following positive topline
		NSCLC Combo 2L (ISS)					\$500m	Read-out Q4 26
		NSCLC 1L Combo OSE-279					\$500m	

Partnered Clinical Assets

Product Candidate	Target	Indication	Pre-Clinical	Phase 1a/1b	Phase 2	Phase 3	Upcoming Milestones
BI 770371 	Anti-SIRPα	Solid tumors (HNSCC)					Ph 1b read-out
Pegrizеprument (FR104) 	Anti-CD28	Kidney Transplantation (US Orphan Drug Designation)					

Immunology & Inflammation

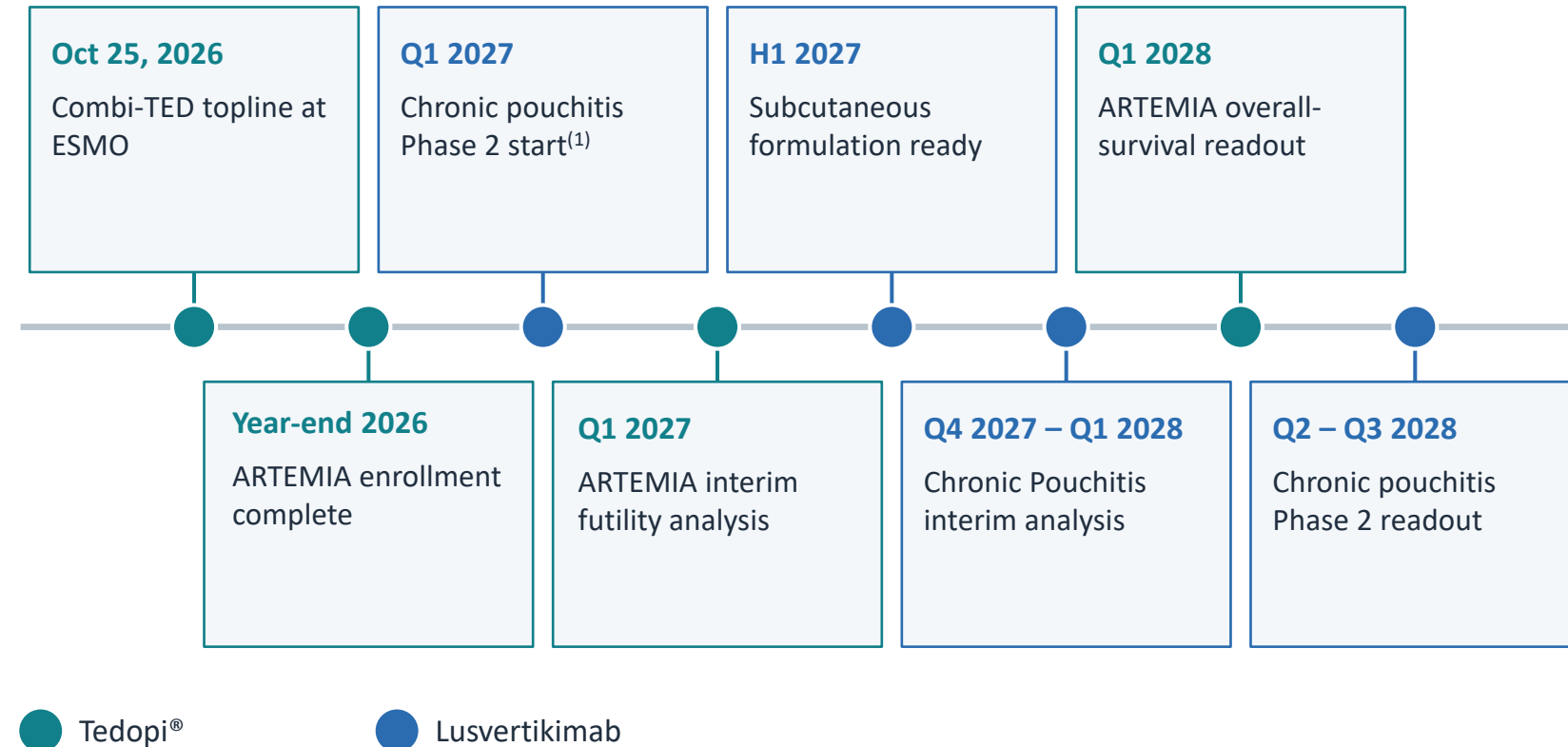
Immuno-Oncology

A catalyst-rich path through 2028

Multiple value creation points

Delivered in 2026

- ✓ **Jan** Pouchitis and HS selected for lusvertikimab
- ✓ **Feb** Second positive ARTEMIA IDMC review
- ✓ **May** TEDOVA positive in ovarian cancer
- ✓ **May** IRIS bridge financing
- ✓ **Sep** Pegrizeprium Phase 2 first patient (Veloxis)



⁽¹⁾ Subject to financing.

*Estimates based on Management expectation – Some of these events depend on external factors OSE has no control over

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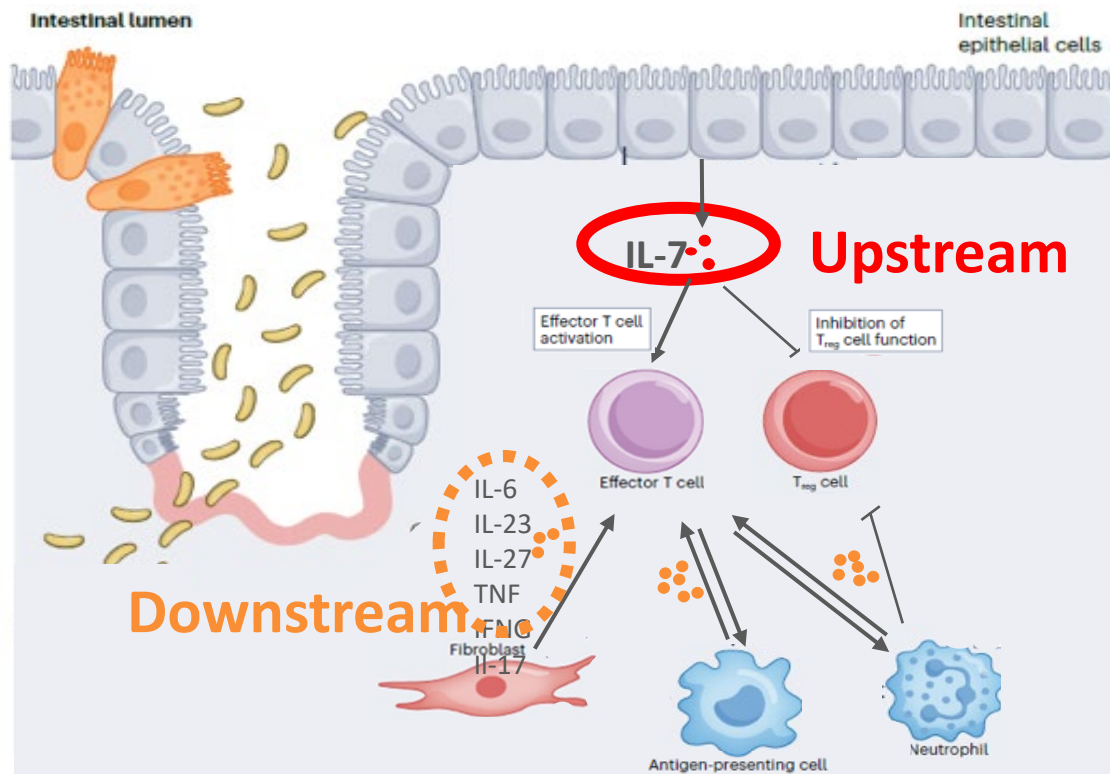


Lusvertikimab Most Advanced Anti-IL-7R mAb

**Strong biological rationale in refractory IBD patients and
inflammatory dermatologic diseases**

Lusvertikimab – First Pure IL-7 Antagonist that Maintains Healthy the Immune System

IL-7 fuels chronic tissue inflammation through upstream mechanism of resistance to hyper-inflammation



Adapted from Neurath M. **Nature Review Immunology** 2024

Lusvertikimab: a differentiated IL-7R antagonist solely targeting the immune system at the root cause of chronic inflammation

- IL-7R pathway is overexpressed in bio-refractory IBD patients^{1,2}, Pouchitis and Hidradenitis Suppurativa
- Blocking the IL-7 receptor prevents molecular signalling transmission by IL-7 through the JAK/STAT5 pathway, responsible for chronic inflammation
- First non-internalizing pure antagonist anti-IL-7R mAb³
- No antagonist activity on TSLP* that has a protective effect on the gut mucosa
- Inhibits activation, differentiation of pathogenic Th1, Th17 and resident memory T cells while sparing Tregs
- Limits migration of T cells into the gut
- Limits immune chronicity and favours healthy immune microenvironment
- Good safety, PK/PD profile in Clinical trials⁴; no cytokine release

Lusvertikimab – Most Advanced and Differentiated First-in-Class Anti-IL-7R mAb

		 	 	
Isotype	IgG4	IgG1	IgG1	IgG1
MoA	• Non-Internalizing¹ • IL-7R full antagonist • Inhibition limited to IL-7 signaling • No T cell depletion	• TSLP Antagonist • T-cell Decrease (mean 35 to 40% decrease in absolute lymphocyte counts)⁶	• Internalizing • Antago + Partial Agonist IL-7R • TSLP Antagonist • T-cell Decrease²	• Internalizing • Antago + Partial Agonist IL-7R
Phase	Phase 2	Phase 2a	Phase 1b	<i>Discontinued</i>
Indications	• Ulcerative Colitis • Chronic Antibiotic-Refractory Pouchitis	• Atopic Dermatitis <i>Failed endpoint in Part B⁵</i> • Alopecia Areata <i>Positive Results from Part B of the SIGNAL-AA Trial⁶</i>	• Dermatology <i>Not initiated</i>	• Multiple Sclerosis <i>Discontinued post Phase 1</i> <i>High Immunogenicity^{3,4}</i>

Lusvertikimab – A Pragmatic Development Plan

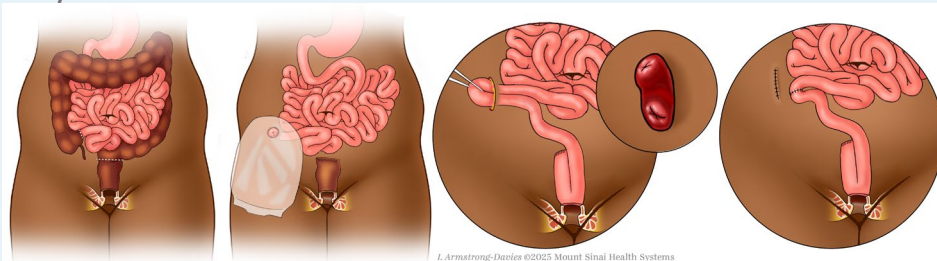
Lusvertikimab in Chronic Pouchitis

Rare/Specialist Indication – To be developed by OSE

Chronic Antibiotic-Refractory Pouchitis: complication of proctocolectomy with ileal pouch-anal anastomosis (IPAA) in patients with ulcerative colitis (UC) - 45k US/EU/JP patients

- Leverage IV GMP material currently available
- 30% of UC patients are refractory to available therapies and require proctocolectomy with IPAA¹

The three stage RPC* with IPAA is the optimal staged method to reduce post-operative complications



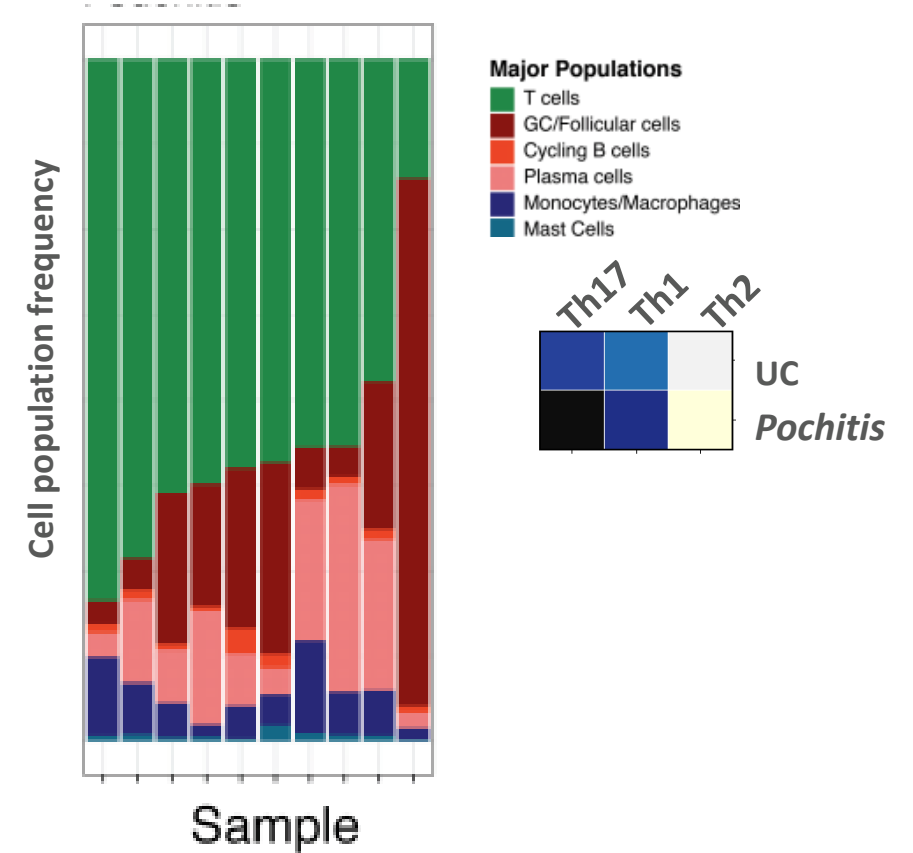
- 70% of patients with IPAA develop pouchitis of which 15% becomes chronic²; 35-40% of them fail currently IBD approved biologics
- Symptoms include increased stool frequency, rectal bleeding, fecal urgency, incontinence, abdominal cramps, fever and extra-intestinal manifestations
- **No FDA approved biologics for pouchitis; Vedolizumab EMA approved with limited efficacy**

Lusvertikimab in Ulcerative Colitis

To be Outlicensed or Financed

Ulcerative Colitis – 200-500k patients in the US alone require advanced therapy

- \$9-11bn Ulcerative Colitis Market mostly generated by anti-TNF α and JAK/IL-23 inhibitors
- 30-40% of patients do not respond sufficiently to anti-TNF α and JAK/IL-23 inhibitors leading to significant need for therapeutic alternatives
- Strong Phase 2 data generated with IV formulation
- Subcutaneous formulation in development to fit the current treatment paradigm
- Minimal costs expected until licensing takes place
- To be developed by partner or financed by OSE



- Refractory Pouchitis and UC exhibit similar inflammatory mechanisms and significant infiltration of TH1/Th17 T cells to an even greater extent in pouchitis than UC¹
- Overexpression of IL-7R by mucosa-infiltrating Th1 and Th17 cells, high IL-7 expression in the tissue & higher expression of IL-7R in CD4, CD8 T cells in pouchitis than UC
- Vedolizumab efficacy in Pouchitis supports rationale for Lusvertikimab (share one MoA); potential better efficacy of Lusvertikimab over Vedolizumab expected and supported by preclinical data²
- Lusvertikimab blocks Teff migration and activation while preserving Treg trafficking, whereas Vedolizumab blocks both Treg and Teff homing without blocking direct effector function of T cells, potentially enabling a more favorable safety profile in chronic indications such as Pouchitis

Inhibits proliferation, survival and activation of intra-tissue effector T cell subsets (Th1, Th17, CD8 memory) while maintaining Tregs for tissue homeostasis

Chronic Antibiotic-Refractory Pouchitis – Phase 2a Design & Expected Timeline

- **Proof of Concept**, randomized *non-comparative* design
- N=47 patients
- A primary evaluation focused on Lusvertikimab arm
- A two-stage Fleming/Simon-type design
- An internal placebo calibration arm randomized in a **3:1 ratio**
- Interim futility assessment after 17 evaluable patients in the active arm
- Final success criterion based on the number of responders observed among 35 evaluable patients treated with lusvertikimab

⇒ 2-stage phase II single-arm Simon design

• In the 1st stage:

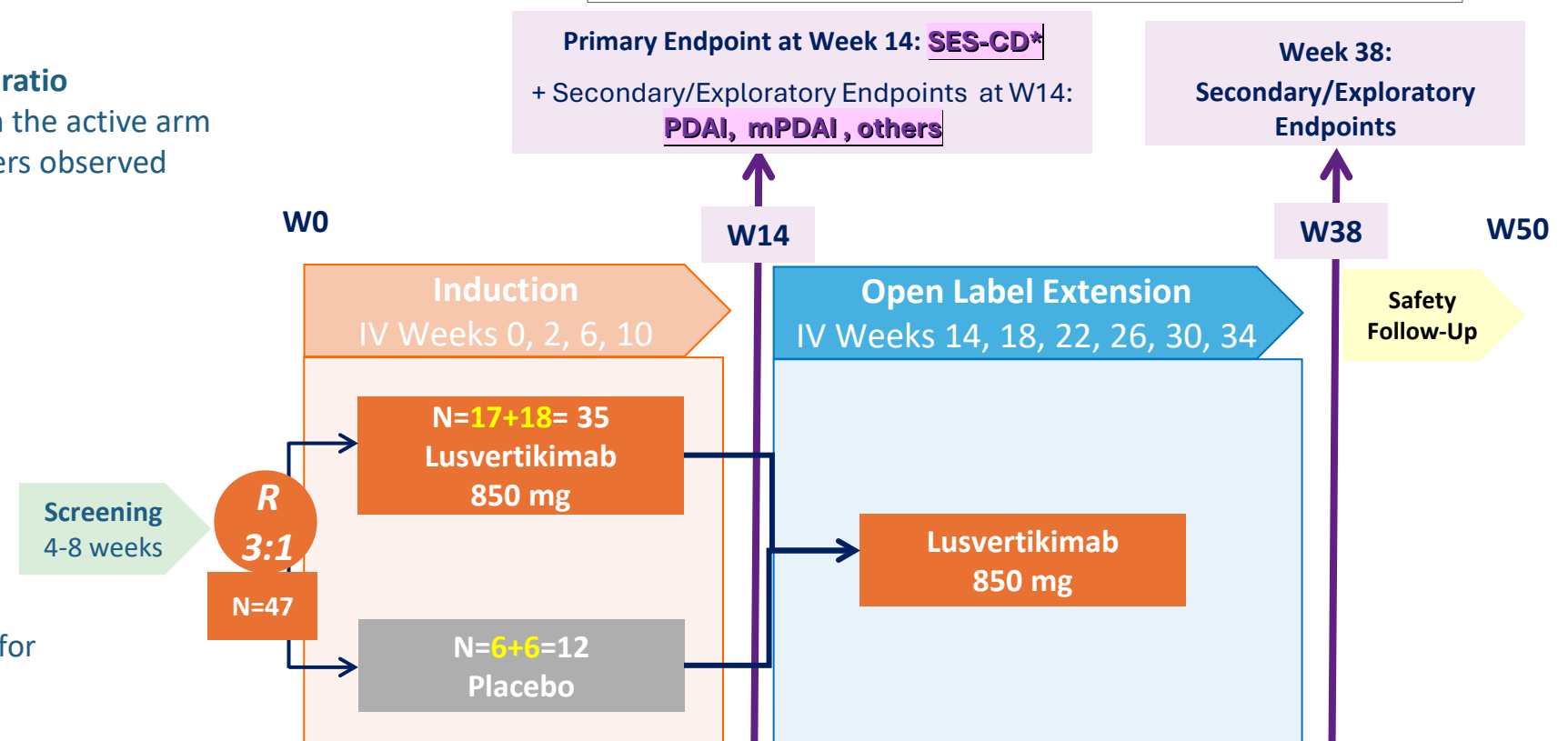
- 17 subjects
- Futility if ≤ 1 response to treatment
→ READ OUT 4Q27⁽¹⁾

• In the 2nd stage:

- 18 additional subjects → 35 subjects in total
- If >4 responses, the study is declared successful for moving into the next phase.
→ READ OUT 1Q28⁽¹⁾

Study population

- 18-75 y
- > 1 year since proctocolectomy and IPAA for ulcerative colitis
- Active chronic pouchitis
- No more than three advanced therapy mechanisms of action received for chronic pouchitis
- Crohn's disease excluded

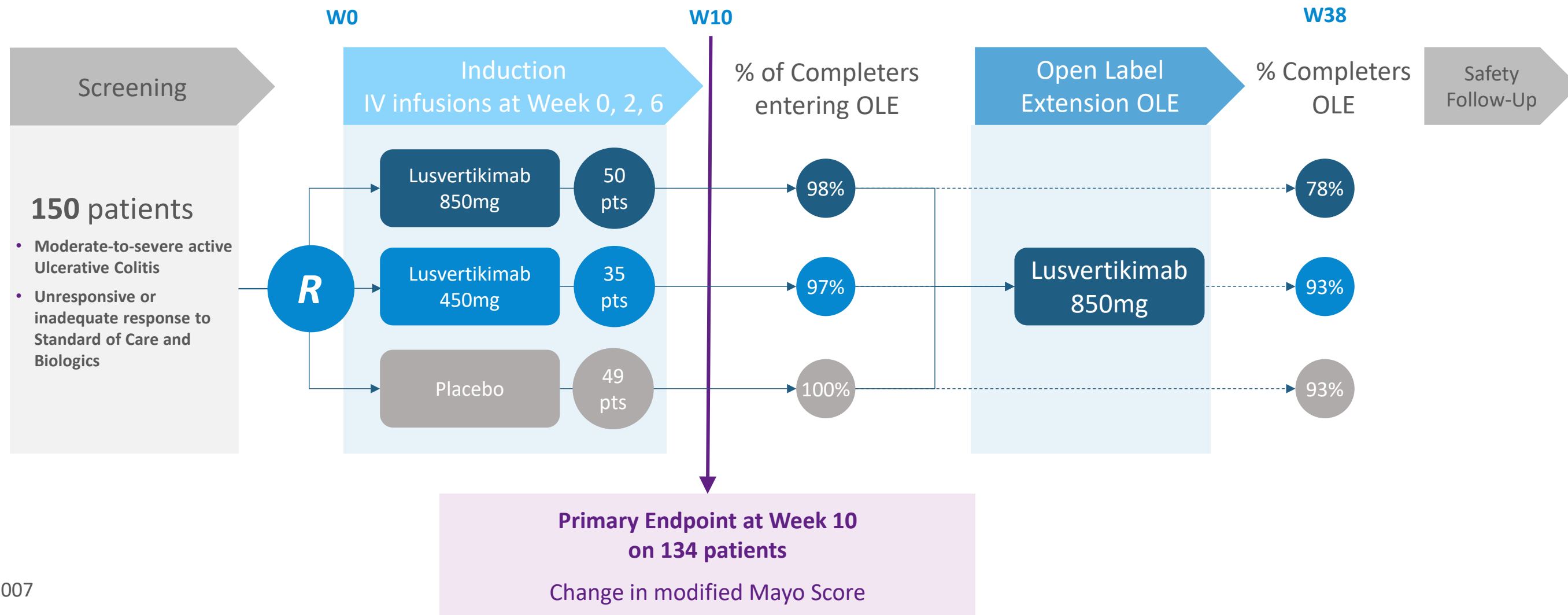


(1) Assuming initiation by year-end 2026, subject to financing

* **SES-CD**: Simple Endoscopic Score for Crohn's disease (in the pouch)

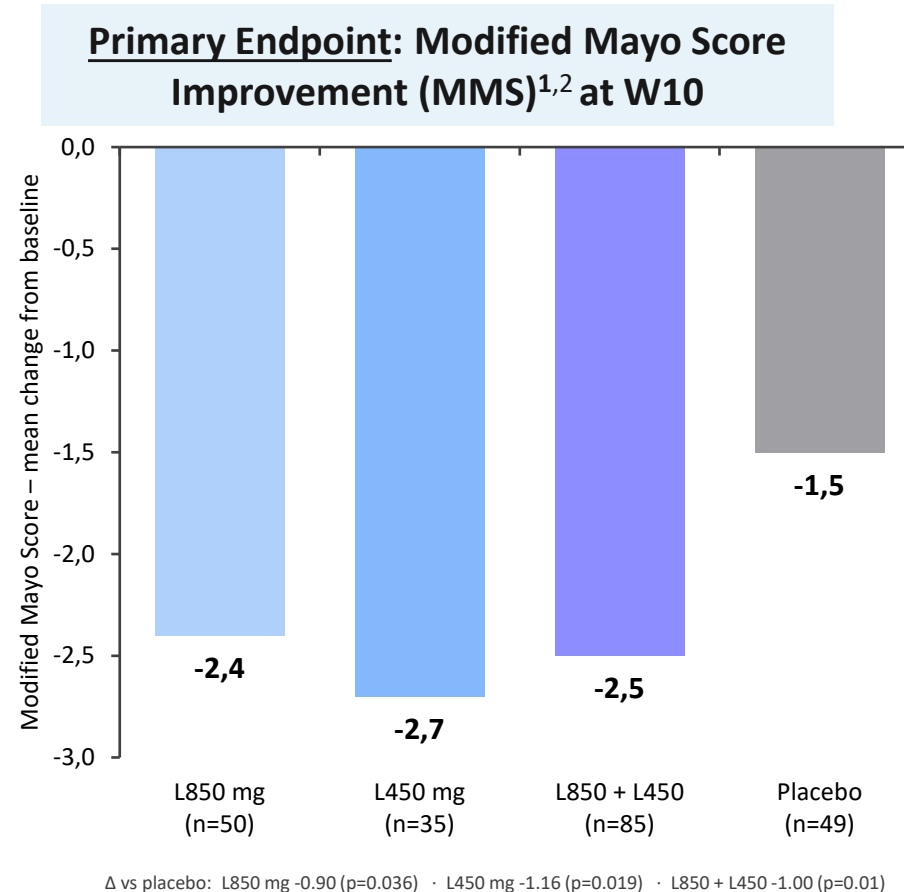
CoTikiS – POC in Chronic Inflammation

Lusvertikimab IV Phase 2 in Moderate-to-Severe UC

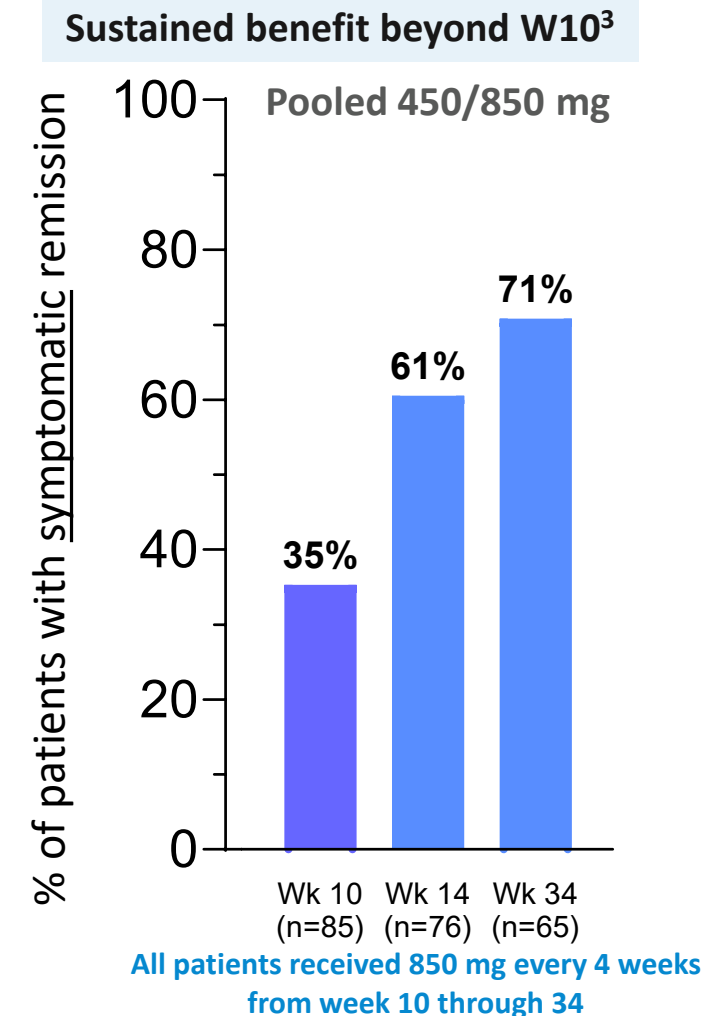


NCT04882007

Primary Endpoint at Week 10 Achieved with Sustained Benefit in OLE

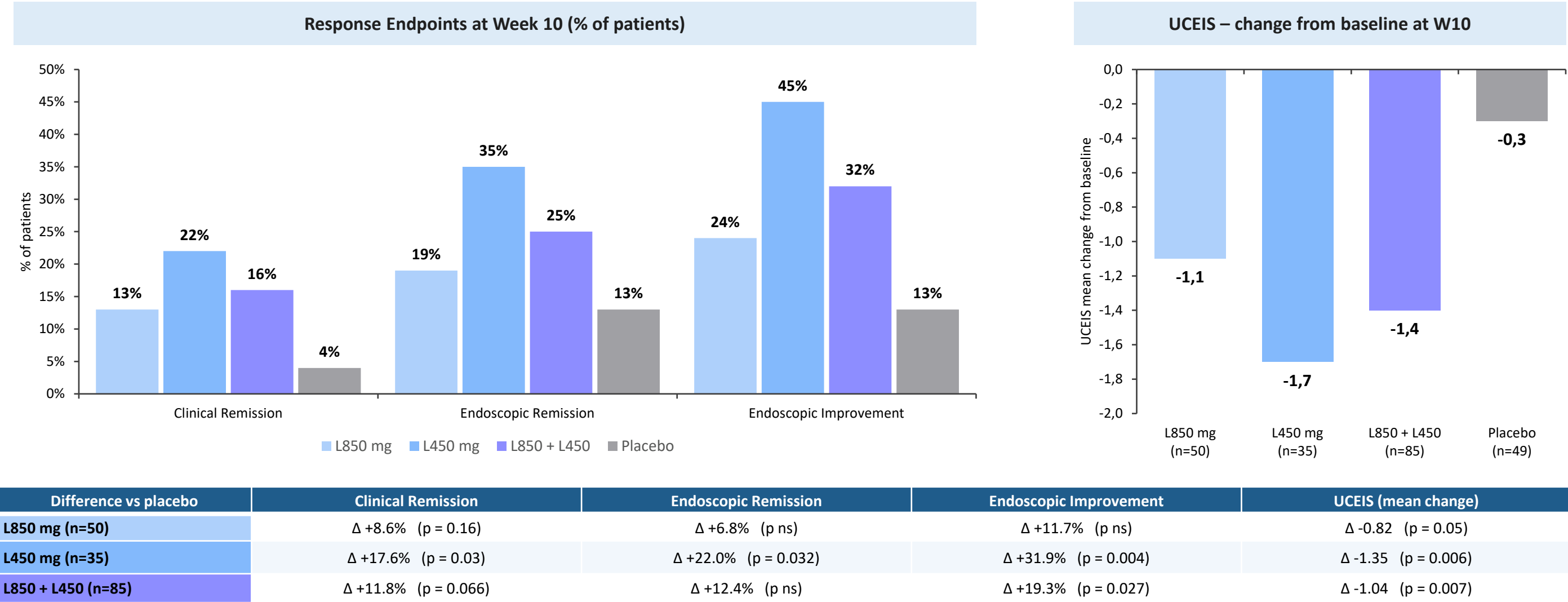


450mg dose dropped at a prespecified futility analysis after 58 patients but later shown not futile



CoTikiS – Week 10 Efficacy Endpoints

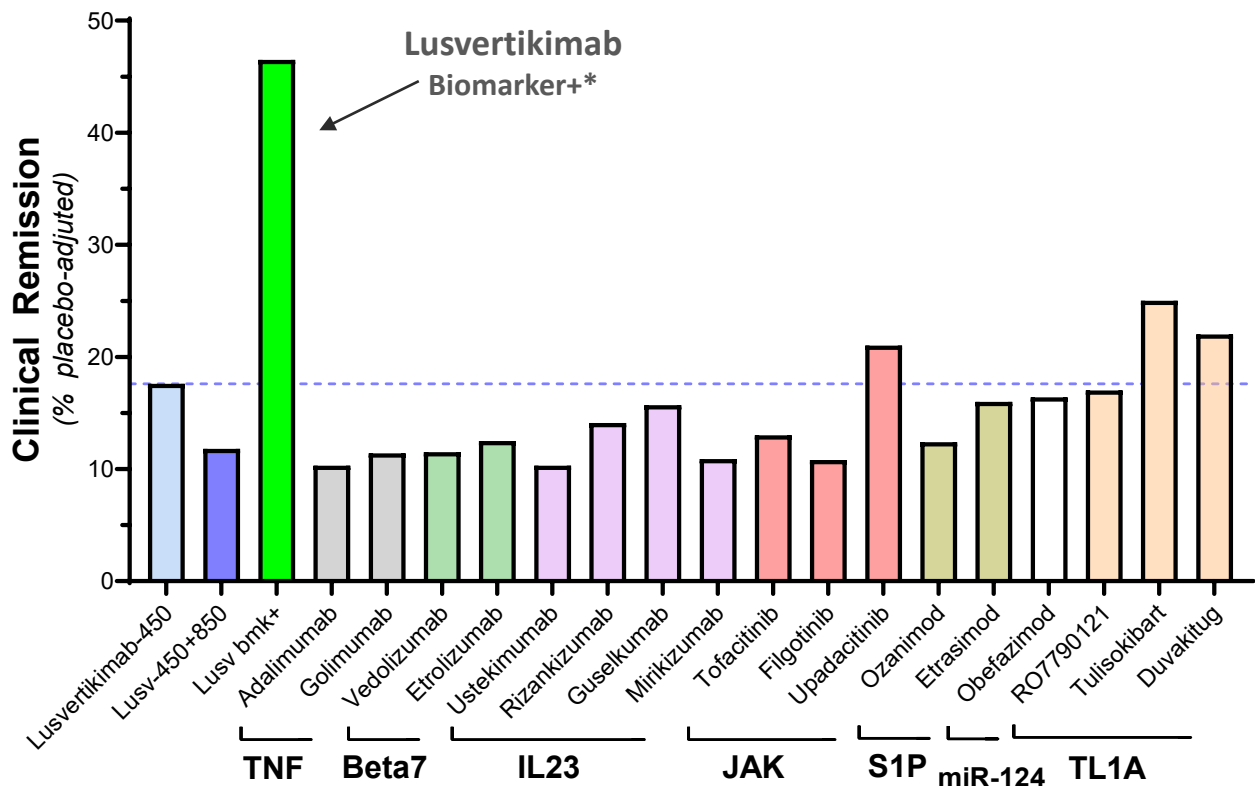
Consistent, dose-independent benefit over placebo across symptomatic, endoscopic and mucosal severity endpoints



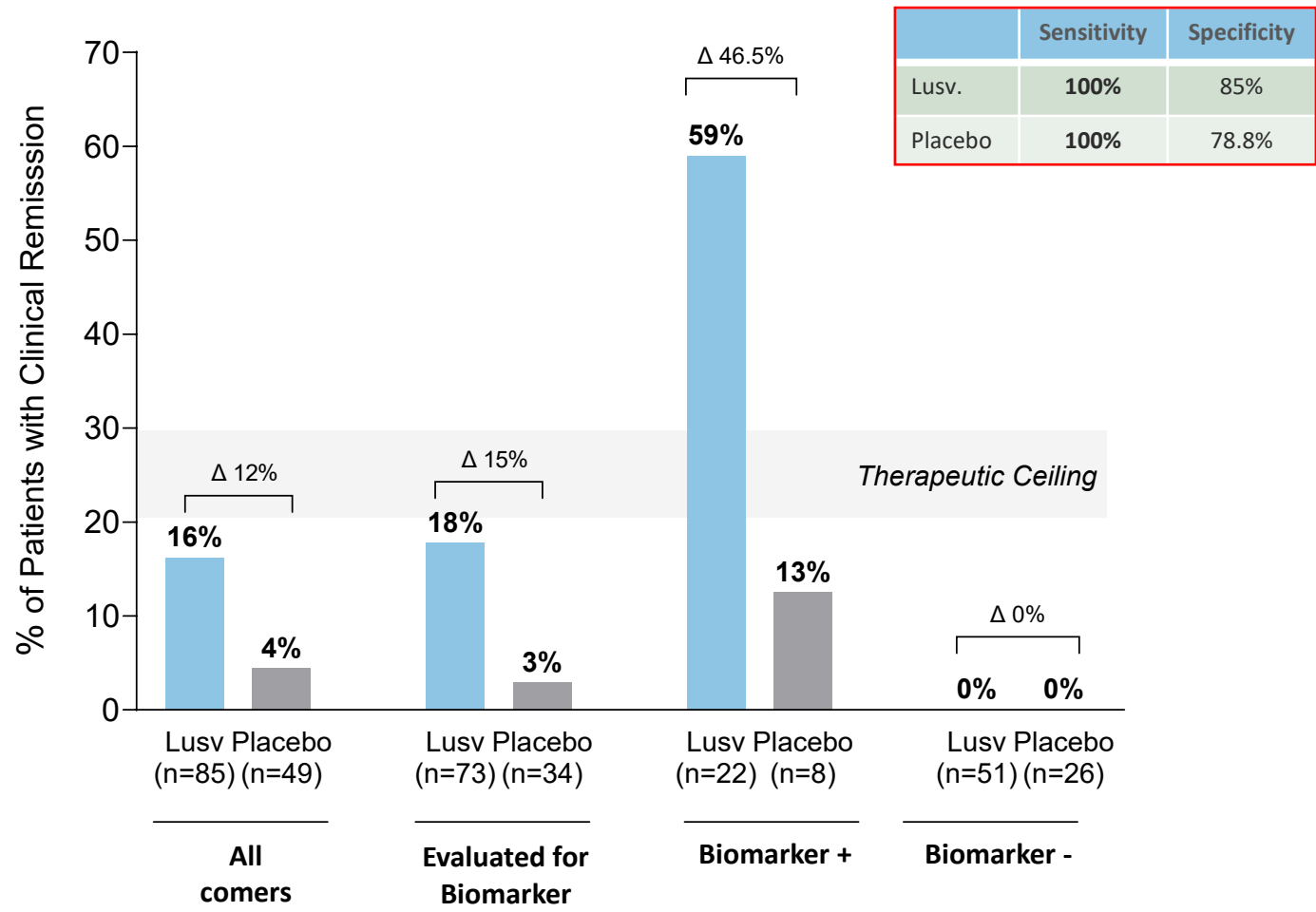
Biomarker+ Could Boost Efficacy 4x in c.30-40% of UC Population Tested*

Confirmatory ex-vivo data to be generated over next 2 years

Clinical Remission (Placebo-adjusted)



Clinical remission based on Lusvertikimab Biomarker



*Composite IL-7R axis biomarker identified with fine-tuning on CoTikiS Phase 2

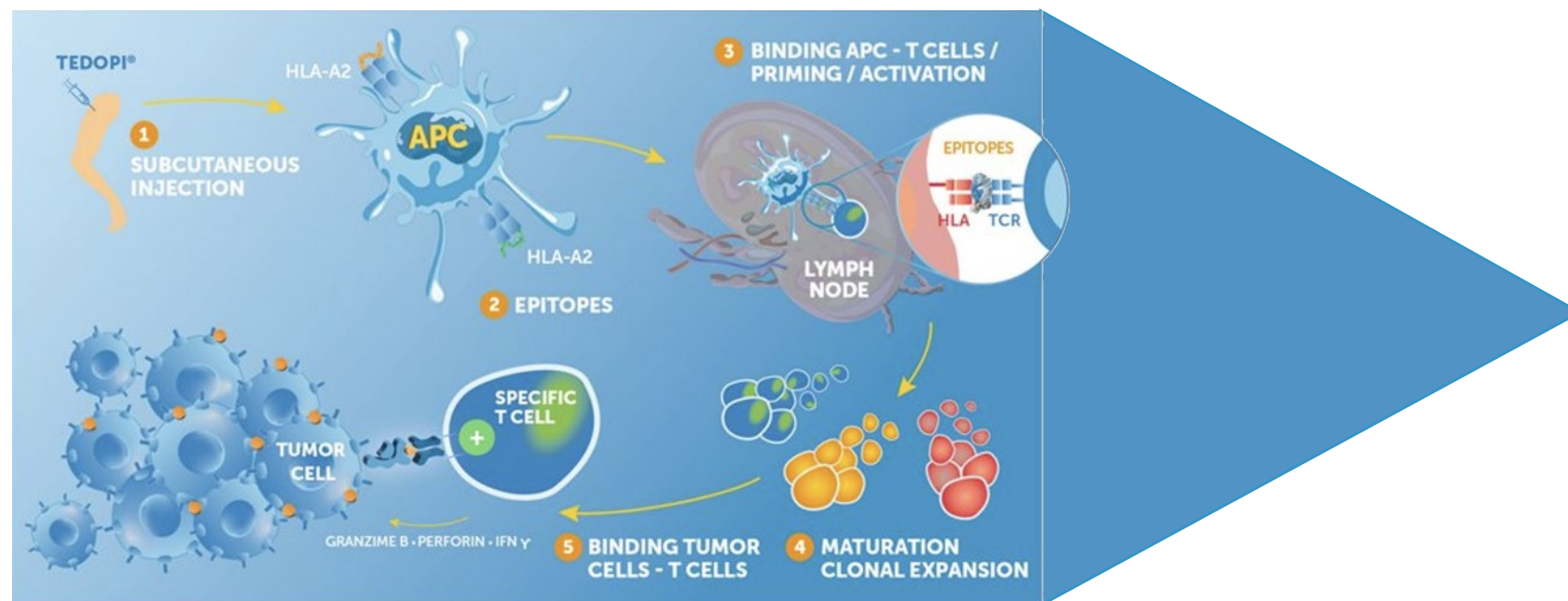
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TEDOPI®
Most Advanced Therapeutic Cancer
Immunotherapy

**Bringing new hope to patients in the fight against ICI
resistant NSCLC**

An Immunotherapy Activating Specific T-Cells to Revive Anti-Tumor Response



- Unique combination of neoepitopes: small peptides deriving from tumor specific antigens expressed in various cancers
- Strong binding to HLA-A2 receptor (45% population)
- Direct activation of tumor specific T-cells differs from checkpoint inhibitors releasing the break of immune response

Proprietary combination
(9 **optimized neoepitopes**
+ 1 epitope giving universal
T helper response)

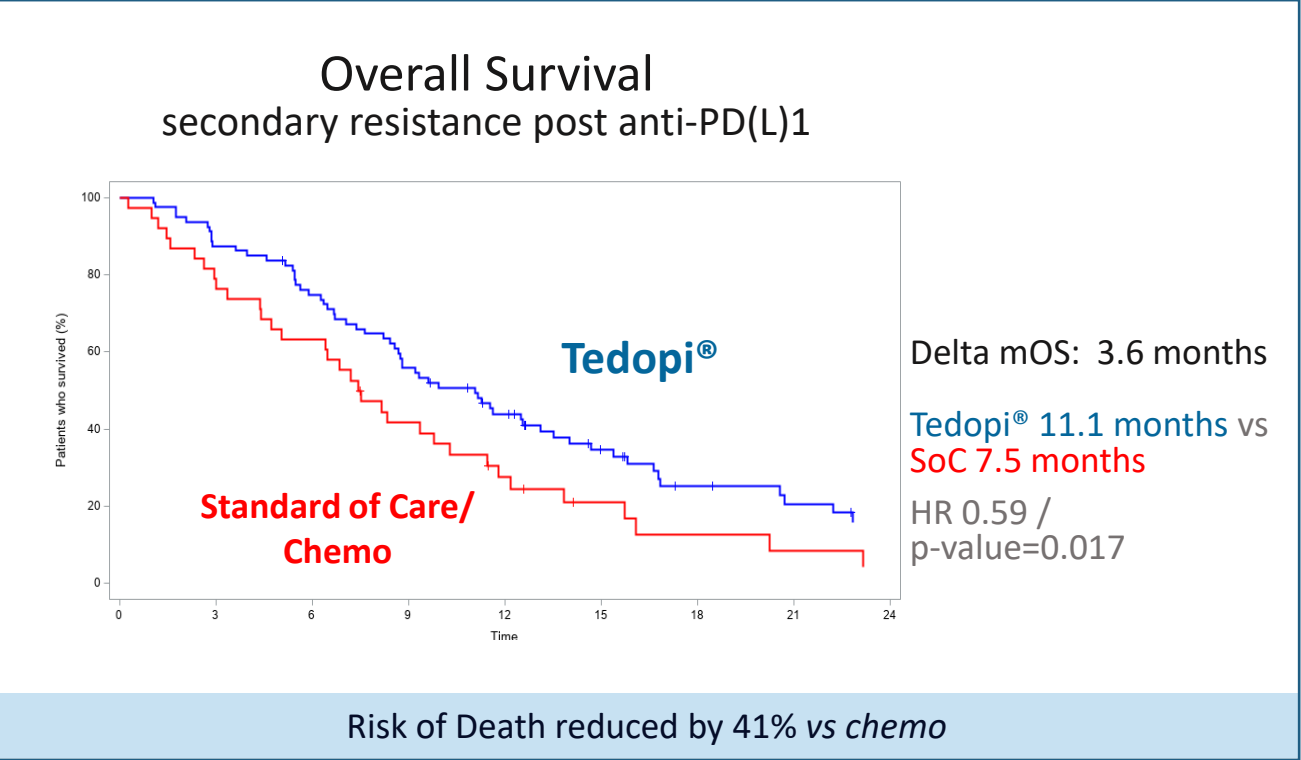
Induces early T cell
memory responses
+
Migration in tissues

Ready to Use
subcutaneous formulation
with Q3W injection

Orphan Drug
Designation (FDA)
> 700 patients treated
in clinical trials

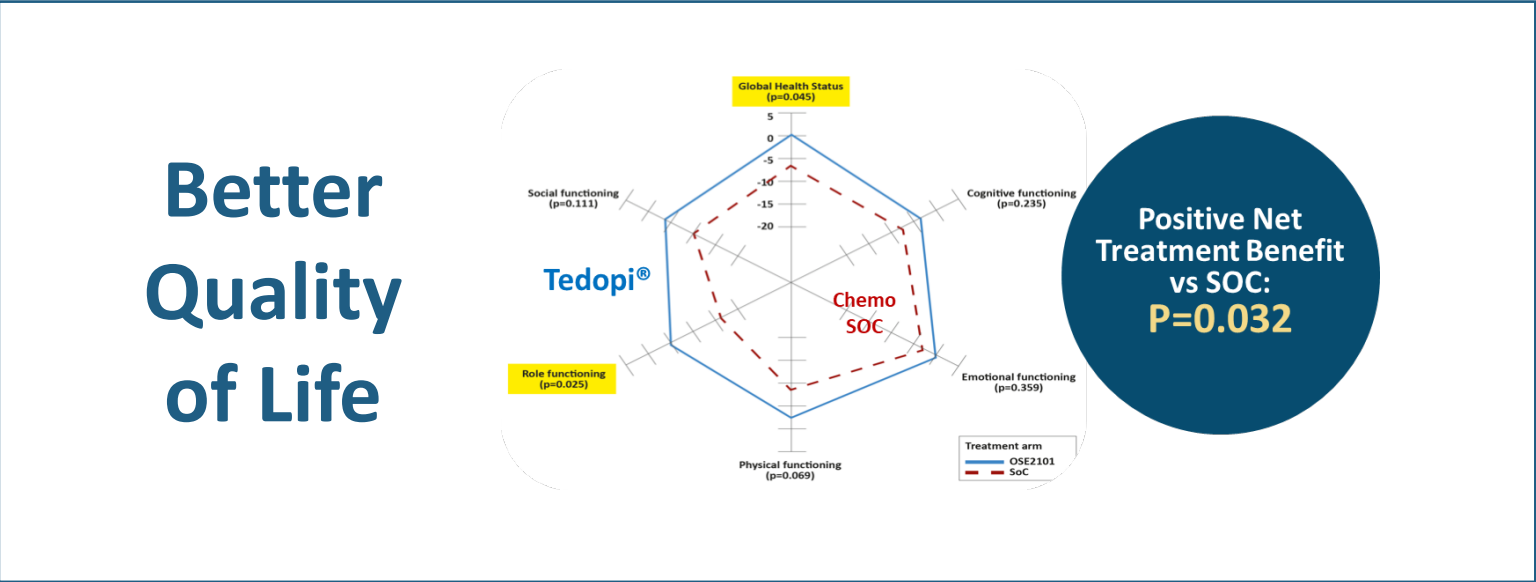
Strong IP position
until **2038**¹
(US / EU / Asia)

ATALANTE: Survival Benefit with Tedopi® in Phase 3 in 3L HLA-A2+ NSCLC with Secondary Resistance to Immune Checkpoint Inhibitors

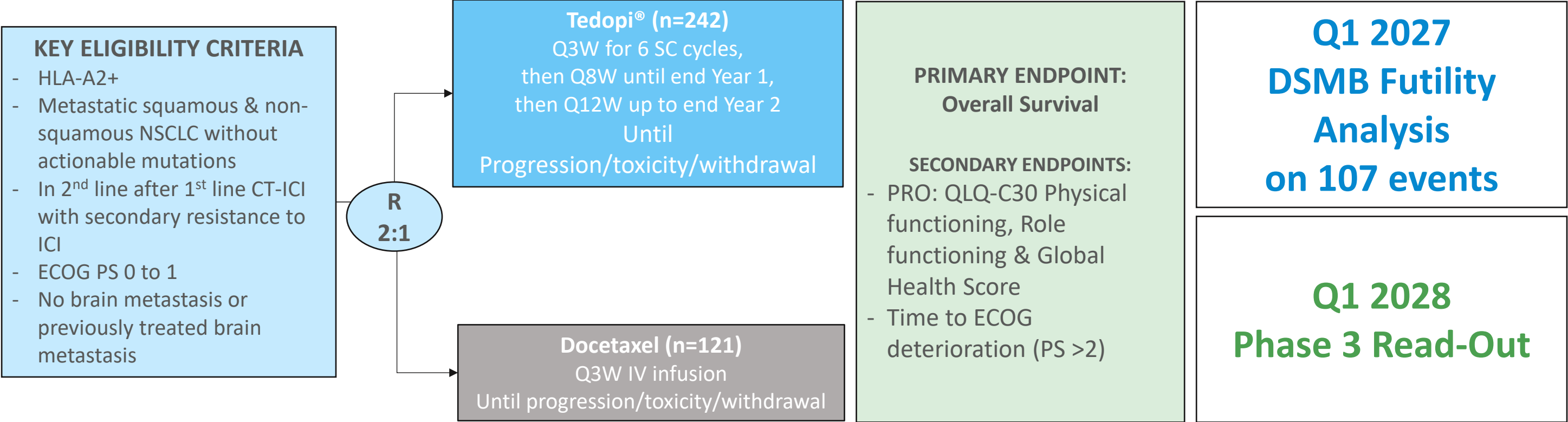


OS rate at 12 months
44% in Tedopi® vs 27.5% in SoC

Significantly safer than SoC
11% vs 35% grade 3-5 related AEs



ARTEMIA - Ongoing Tedopi® Phase 3 in HLA-A2+ NSCLC Patients Post Immune Checkpoint Inhibitors



Additional Read-Out in 2026 in NSCLC, Ovarian and Pancreatic Cancer

Phase 2 ISS trials in combination with immunotherapy or chemotherapy treatments

Maintenance setting post standard of care

TEDOVA - Ovarian Cancer
In combination with pembrolizumab
185 patients



TEDOPaM - Pancreatic Cancer
In combination with FOLFIRI
106 patients



2nd line post 1st line chemo IO

CombiTED - NSCLC
In combination with nivolumab
105 patients



Tedopi® Alone or in Combination with Pembrolizumab vs Best Supportive Care as Maintenance in Patients with Platinum-Sensitive Recurrent Ovarian Cancer²

Primary Endpoint: Progression Free Survival

Sponsored by **ARCAGY-GINECO**

PI: Alexandra LEARY

(Gustave Roussy Institute)

France/ Germany/ Belgium



ARCAGY - GINECO

Tedopi® + FOLFIRI vs FOLFIRI as Maintenance Treatment in Advanced or Metastatic Pancreatic Ductal Adenocarcinoma after 8 Cycles of Folfirinox³

Sponsored by **GERCOR PRODIGE**

PI: Cindy NEUZILLET

(Curie Institute, France)



Tedopi® + Docetaxel vs Tedopi + Nivolumab as 2nd line in Metastatic NSCLC failing standard 1st line Chemo-immunotherapy¹

Primary Endpoint: Overall Survival

Sponsored by **FoRT**

PI: Federico CAPPUZZO

(Roma Cancer Institute)

Italy /Spain/ France



Positive Topline Result⁴ in 2025

Long-term OS follow-up ongoing

Recruitment completed

Results to be presented at ESMO on October 25, 2026

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Financials

Financials

Company Overview	
Market Cap*:	€56m
Cash Position: (June 30, 2026)	€11.1m
Cash Runway:	Through December 2026
Outstanding Shares:	24.8m
Latest Equity Raised: (March 2021)	€18.6m
Equity raised to date	€53m
Deal upfronts to date	€179m
IPO Date	March 30, 2015

*As of September 25, 2026

Analyst Coverage	
	Jamila El Bougrini (FR)
	Arron Aatkar (UK) Jyoti Prakash (UK)
	Martial Descoutures (FR)
	Nicolas Pauillac (FR)
	David Seynnaeve (BE)
	Lionel Labourdette (FR)
	Mohamed Kaabouni (FR)

2026 Corporate Calendar	Date
2025 Full-Year Financial Update and Statements	April 30, 2026
2026 1Q Cash Position	April 30, 2026
Annual General Meeting	June 24, 2026
2026 First-Half Financial Update and Statements	Revised date pending
2026 3Q Cash Position	October 27, 2026
2026 4Q Cash Position	January 26, 2027

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Appendix

CoTikiS – 850 mg Group More Severe Disease than 450 mg and/or Placebo Groups

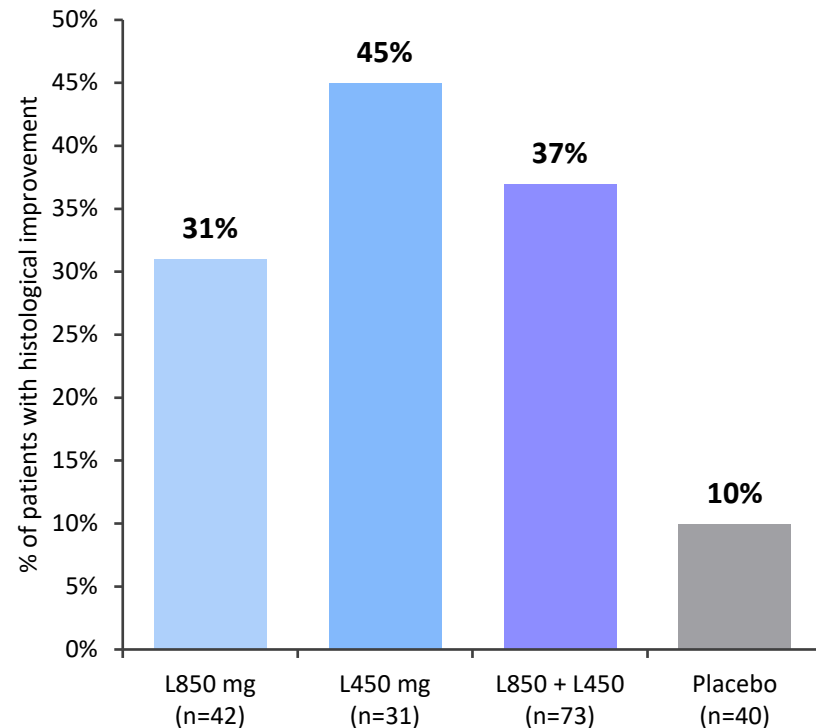
Demographics and disease characteristics

	Placebo (n=49)	450 mg (n=35)	850 mg (n=50)	Total (n=134)
Age: mean (SD)	42.7 (15.9)	38.8 (10.5)	42.5 (15.1)	41.6 (14.4)
Sex: male	28 (57.1%)	22 (62.9%)	27 (54.0%)	77 (57.5%)
Weight (kg) mean (SD)	75.3 (15.2)	72.8 (16.2)	71.5 (18.0)	73.2 (16.5)
Never smoker	39 (79.6%)	25 (71.4%)	43 (86.0%)	107 (79.9%)
Never alcohol consumption	34 (69.4%)	25 (71.4%)	40 (80.0%)	99 (73.9%)
Region: EU Country	22 (44.9%)	8 (22.9%)	22 (44.0%)	52 (38.8%)
UC duration (years) mean (SD)	8.2 (7.5)	7.2 (6.5)	9.3 (8.6)	8.4 (7.7)
Previous exposure to biologics	19 (38.8%)	5 (14.3%)	19 (38.0%)	43 (32.1%)
<i>Previous biologics: 2+</i>	11 (57.9%)	2 (40%)	13 (68.8%)	26 (60.4%)
<i>Previous biologics: 3+</i>	5 (26.3%)	0 (0%)	6 (31.5%)	11 (25.6%)
Concomitant use of steroids	23 (46.9%)	18 (51.4%)	25 (50.0%)	66 (49.3%)
Modified mayo score (mMS) Mean (SD)	6.6 (1.2)	6.0 (1.4)	6.5 (1.0)	6.4 (1.2)
Category of mMS				
5-6	21 (42.9%)	17 (48.6%)	25 (50.0%)	63 (47.0%)
7-9	26 (53.1%)	13 (37.1%)	25 (50.0%)	64 (47.8%)
Endoscopic subscore mean (SD)	2.5 (0.5)	2.4 (0.5)	2.6 (0.5)	2.5 (0.5)
Category of endoscopic subscore: 3	26 (53.1%)	15 (42.9%)	32 (64.0%)	73 (54.5%)
C-Reactive protein (mg/L) Mean (SD)	8.6 (13.6)	9.4 (16.7)	11.2 (18.1)	9.8 (16.1)
Serum albumin (g/L) Mean (SD)	42.3 (4.4)	42.6 (4.5)	40.8 (5.4)	41.8 (4.9)
FCP (µg/g) mean (SD)	1459.5 (1865.0)	1088.0 (1600.5)	1191.8 (1603.3)	1261.6 (1696.7)

Induction Results at Week 10

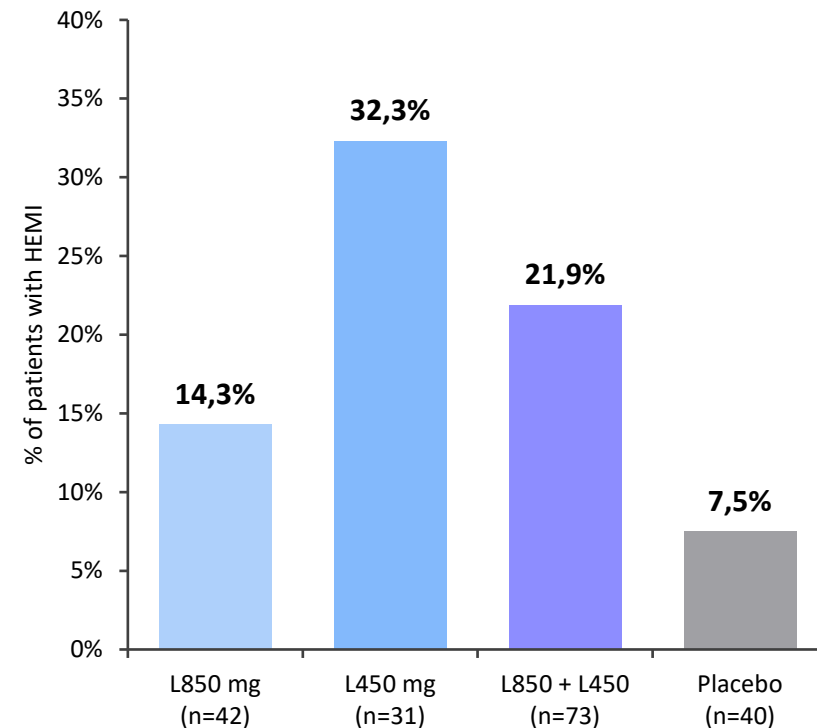
Clinically meaningful and significant histologic and histo-endoscopic mucosal improvement

**Histological Improvement
at W10***



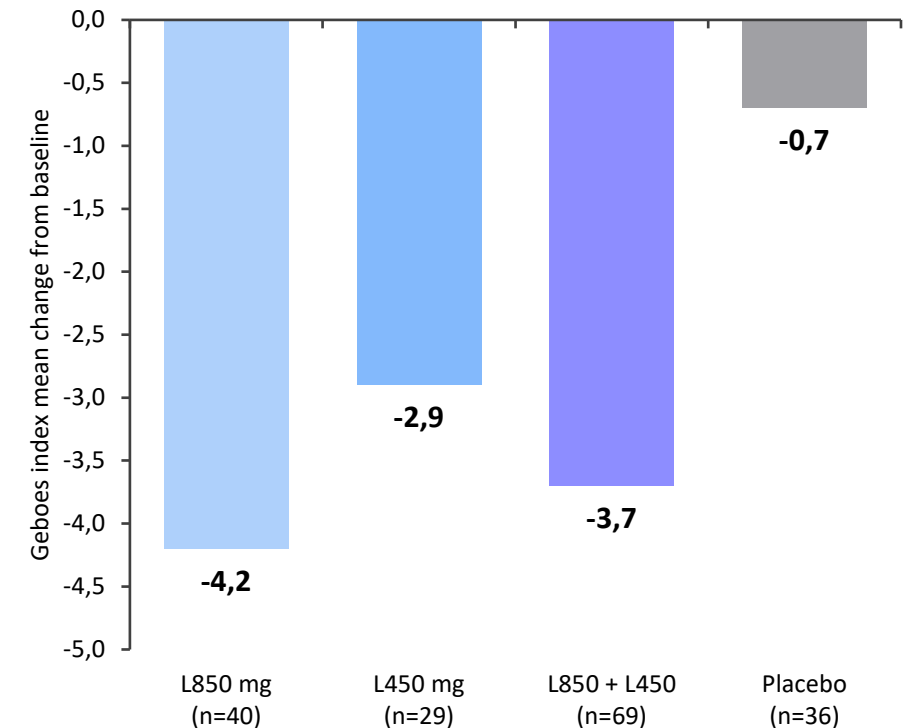
Δ vs placebo: L850 mg +21.0% (p=0.02) · L450 mg +35.2% (p <0.01) · L850 + L450 +27.0% (p <0.01)

**Histo-Endoscopic Mucosal
Improvement (HEMI) at W10****



Δ vs placebo: L850 mg +6.8% (p=NR) · L450 mg +24.8% (p <0.01) · L850 + L450 +14.4% (p=0.05)

**Histological Geboes index
change from baseline at W10**

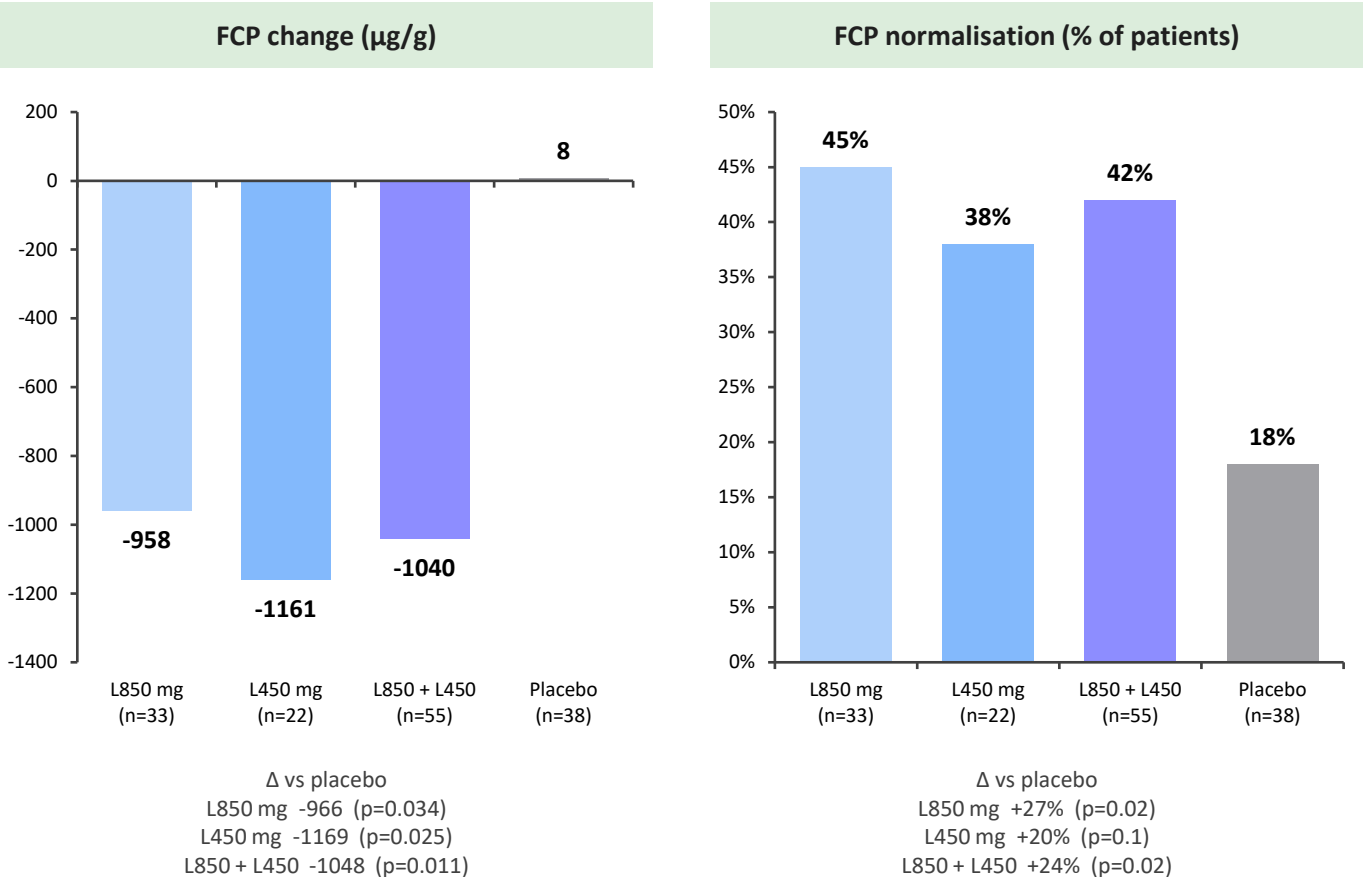


Δ vs placebo: L850 mg -3.5 (p <0.01) · L450 mg -2.2 (p=NR) · L850 + L450 -3.0 (p=0.02)

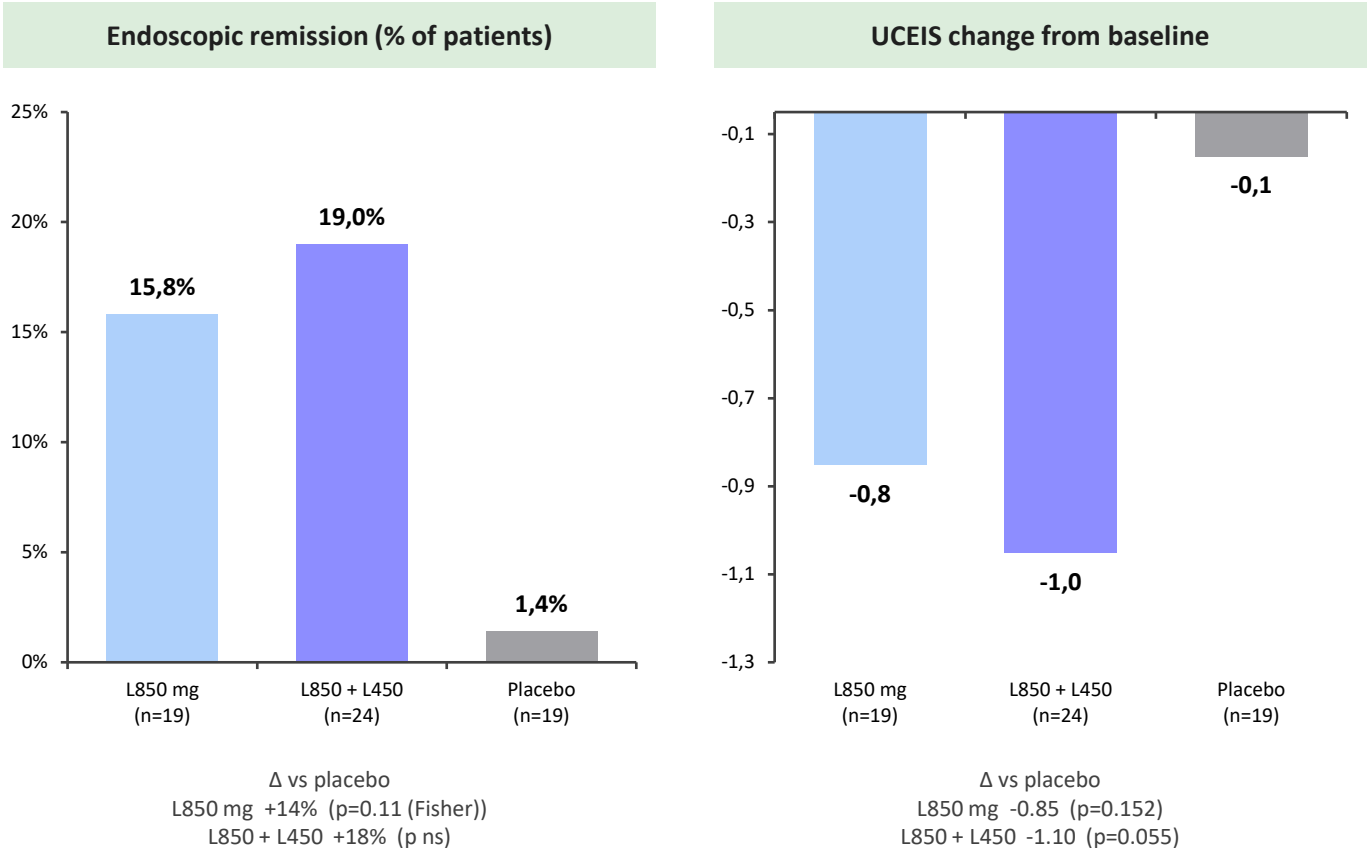
Induction Results at Week 10

Subgroup analyses: benefit maintained in patients with active inflammation and in those who have already failed a biologic

Active Inflammation at Baseline: FCP > 250 µg/g



Biologic-Experienced Patients*

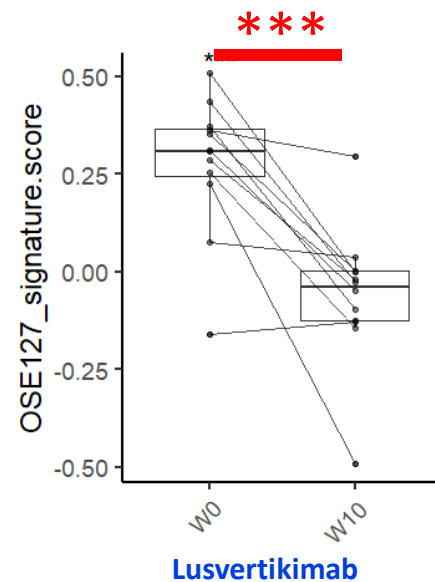


Induction Results at week 10

Lusvertikimab demonstrates a clear biological footprint in intestinal tissue with selective reduction of IL-7R⁺ immune cells and inflammatory infiltrate

Inhibition of the IL-7 pathway

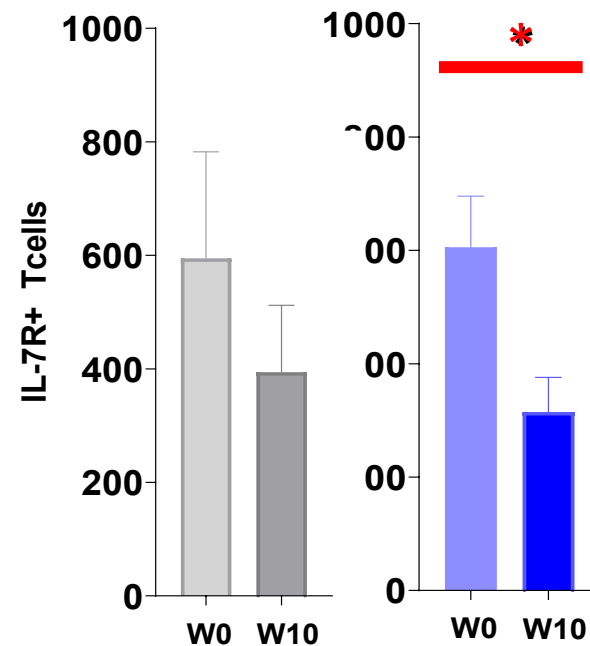
RNAseq analysis in PBMCs



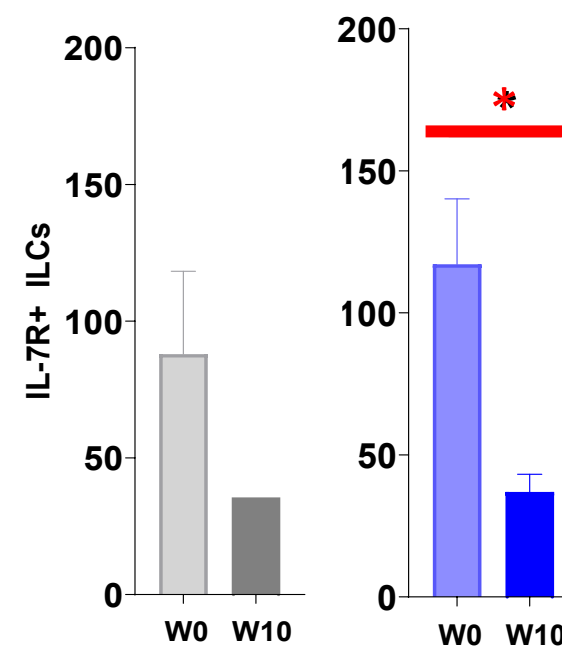
IL-7R-induced gene signature score¹

Selective decline in Mucosal T Cells, Innate Lymphoid Cells (ILCs) and Total Inflammatory Cells

IL-7R+ T cells

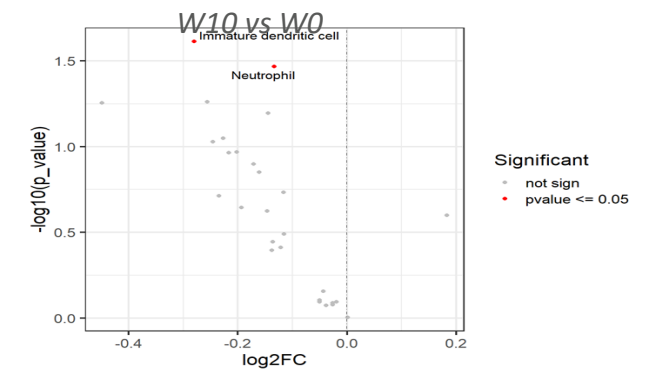


IL-7R+ ILC cells

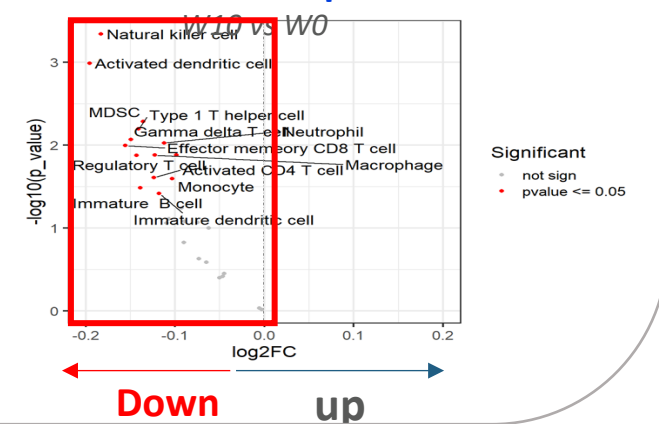


Placebo Responders²
Lusvertikimab Responders²

Placebo Responders^{2,3}



Lusvertikimab Responders^{2,3}



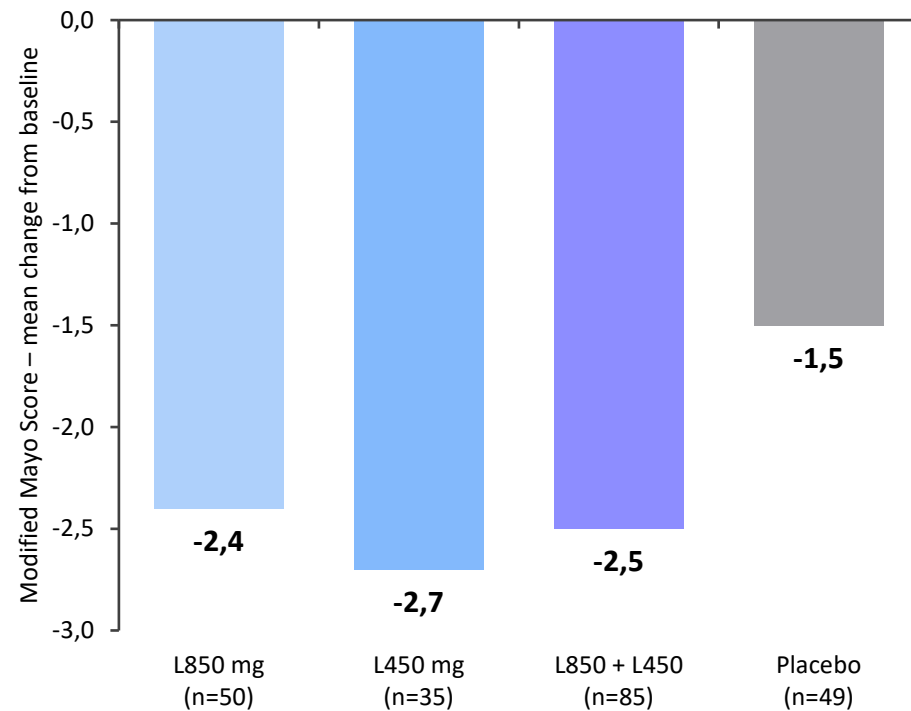
Lusvertikimab – Well Tolerated & Good Safety Profile (Induction)

	Placebo (N=49) N(%) [E]	450 mg (N=36) N(%) [E]	850 mg (N=51) N(%) [E]	Total (N=136) N(%) [E]
At least one TEAE in induction phase	16 (32.7) [29]	17 (47.2) [33]	20 (39.2) [42]	53 (39.0) [104]
At least one TEAE related to study treatment	1 (2.0) [1]	3 (8.3) [4]	4 (7.8) [14]	8 (5.9) [19]
At least one serious TEAE	3 (6.1) [3]	2 (5.6) [3]	2 (3.9) [3]	7 (5.1) [9]
At least one serious TEAE related to study treatment	—	1 (2.8) [1]	—	1 (0.7) [1]
At least one severe TEAE	2 (4.1) [2]	1 (2.8) [2]	—	3 (2.2) [4]
At least one severe TEAE related to study treatment	—	1 (2.8) [1]	—	1 (0.7) [1]
At least one related TEAE leading to death	—	—	—	—
At least one TEAE leading to drug withdrawal	3 (6.1) [3]	2 (5.6) [3]	—	5 (3.7) [6]
At least one TEAE leading to drug interruption	2 (4.1) [2]	1 (2.8) [1]	—	3 (2.2) [3]
At least one TEAE leading to study discontinuation	3 (6.1) [3]	2 (5.6) [3]	—	5 (3.7) [6]
At least one AESI	6 (12.2) [7]	7 (19.4) [7]	9 (17.6) [10]	22 (16.2) [24]
At least one infection	6 (12.2) [7]	5 (13.9) [5]	7 (13.7) [8]	18 (13.2) [20]
At least one lymphopenia < 500 10 ⁶ /L	—	2 (5.6) [2]	2 (3.9) [2]	4 (2.9) [4]

Lymphopenia was transient, not associated with a higher rate or severity of infection, was more frequent in patients treated with corticosteroids or with baseline values <1*10⁹/L and did not lead to treatment discontinuation

Clinically and Statistically Meaningful Remission at Week 10 with Lusvertikimab

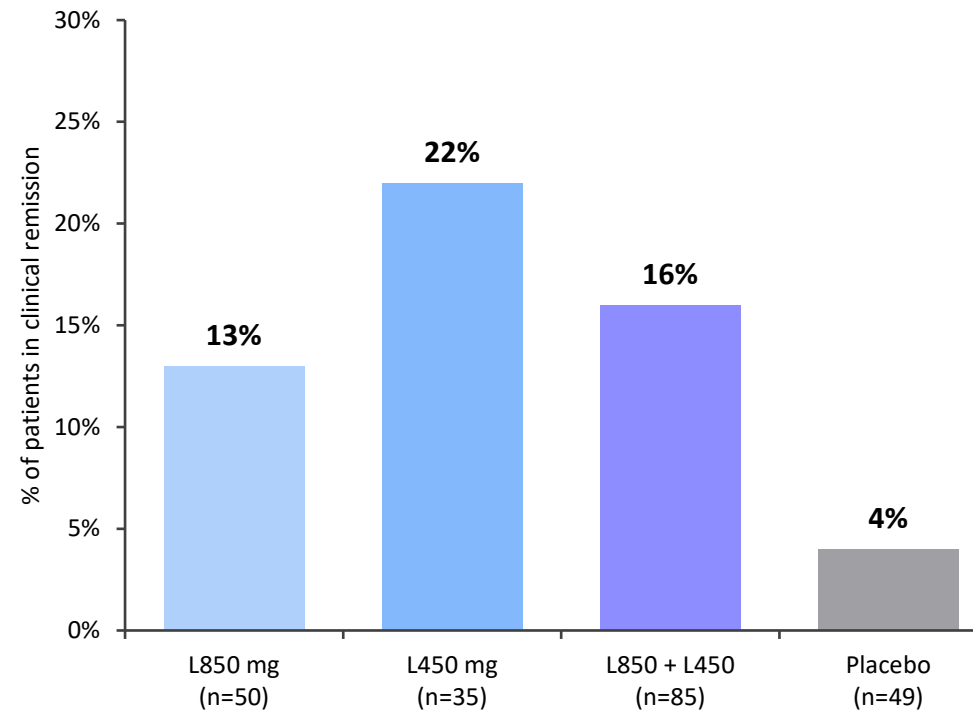
Primary Endpoint: Modified Mayo Score Improvement (MMS)^{1,2} at W10



Δ vs placebo: L850 mg -0.90 (p=0.036) · L450 mg -1.16 (p=0.019) · L850 + L450 -1.00 (p=0.01)

450mg dose dropped at a prespecified futility analysis after 58 patients but later shown not futile

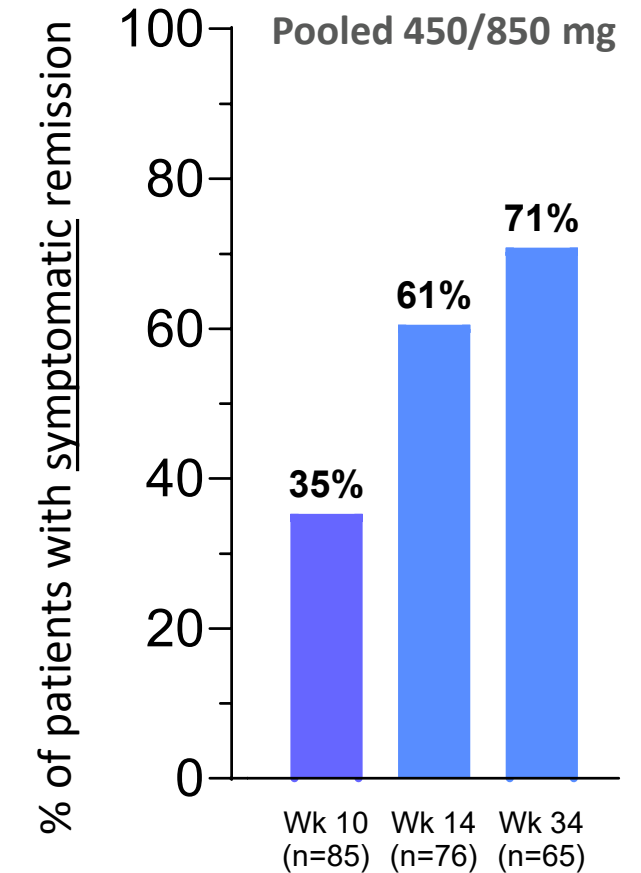
Clinical Remission at W10



Δ vs placebo: L850 mg +8.6% (p=0.16) · L450 mg +17.6% (p=0.03) · L850 + L450 +11.8% (p=0.066)

Clinical remission: MMS ≤2 with no subscore >1 and RB 0, SF ≤ 1, MES 0 or 1

Sustained benefit beyond W10³

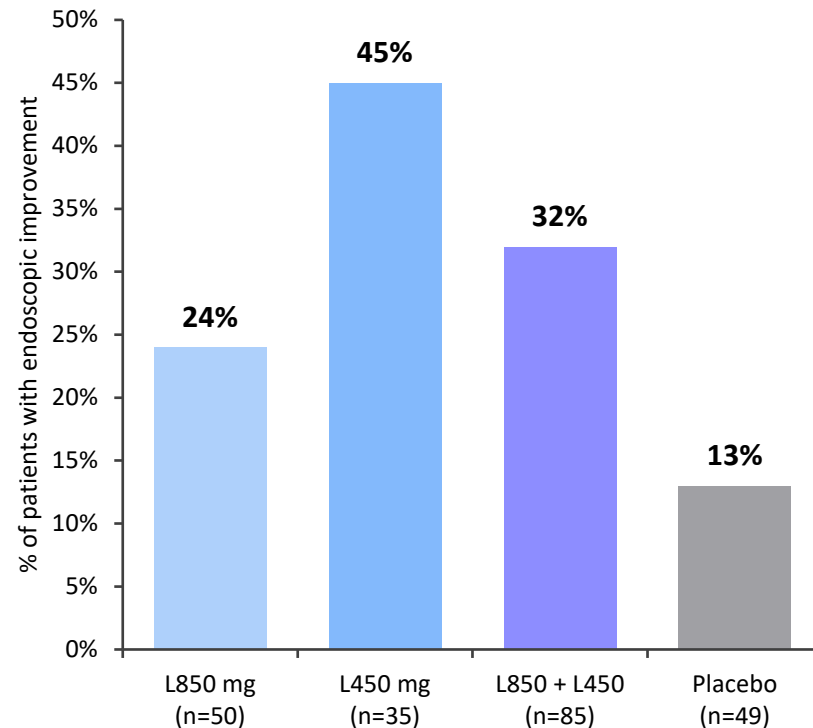


All patients received 850 mg every 4 weeks from week 10 through 34

Induction Results at Week 10

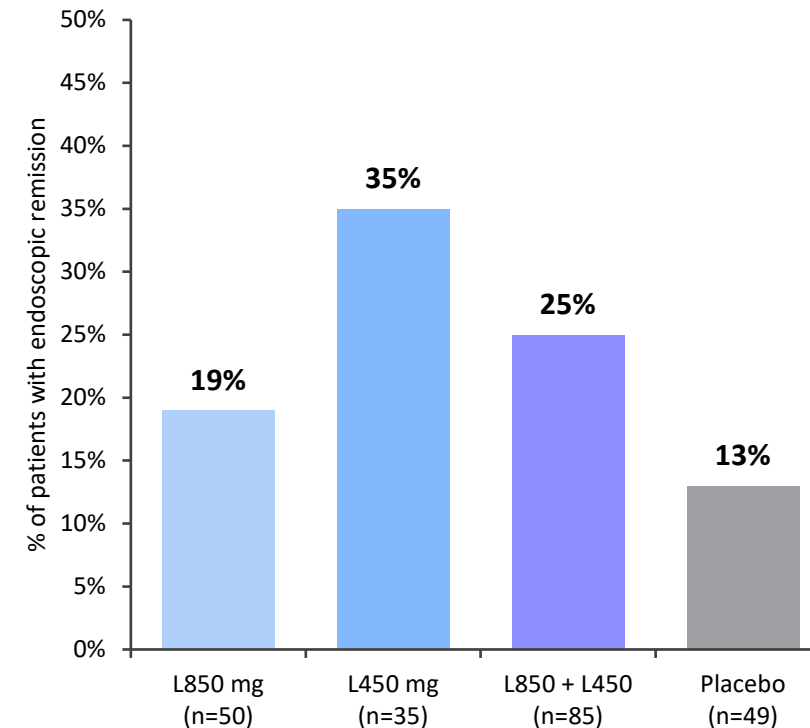
Clinically meaningful and significant endoscopic improvement and remission

Endoscopic Improvement
at W10**



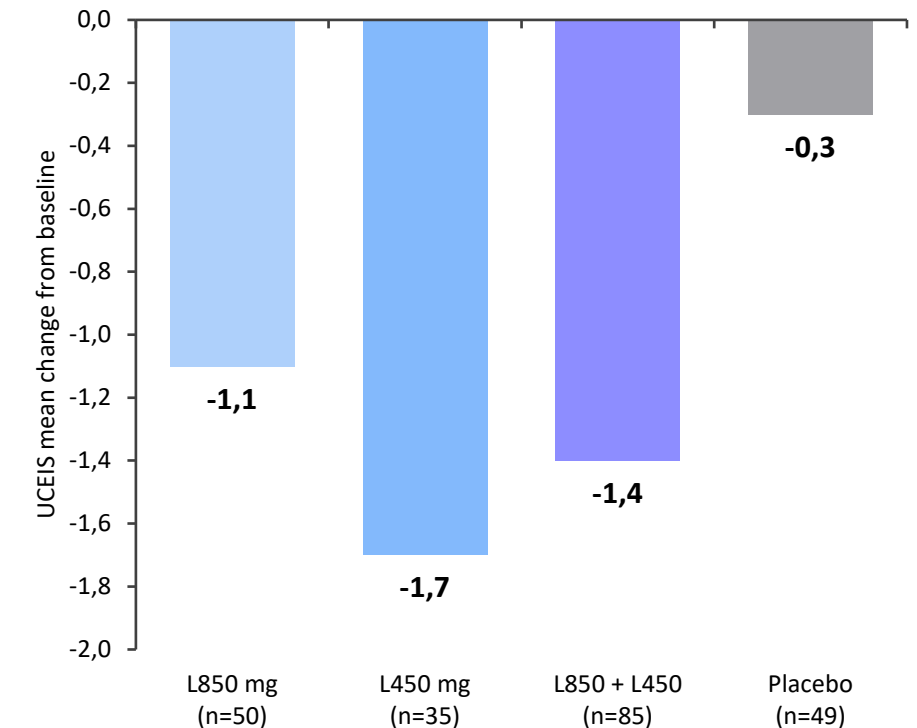
Δ vs placebo: L850 mg +11.7% (p=NR) · L450 mg +31.9% (p=0.004) · L850 + L450 +19.3% (p=0.027)

Endoscopic Remission*at W10



Δ vs placebo: L850 mg +6.8% (p=NR) · L450 mg +22.0% (p=0.032) · L850 + L450 +12.4% (p=NR)

UC Endoscopic Index of Severity UCEIS*
change from baseline at W10**



Δ vs placebo: L850 mg -0.82 (p=0.05) · L450 mg -1.35 (p=0.006) · L850 + L450 -1.04 (p=0.007)

Tedopi® Targets 100k Patients in 2nd Line NSCLC Post ICI

Tedopi® has the potential to become the new standard for recurrent patients in 2L NSCLC presenting HLA-A2 phenotype

LUNG CANCER:
High prevalence, mortality and unmet need - worldwide

- 2nd most frequently diagnosed cancer type*
 - c.2m new cases of lung cancer diagnosed per year
 - c.1.8m deaths from lung cancer per year**
- Highest mortality among 36 cancer types
- Majority of NSCLC patients without actionable mutation are treated with Immune Checkpoint Inhibitors (ICI) as 1st line of treatment

Treatment paradigm in NSCLC with no driver mutation

- L1: anti-PD(L)1 based with / without chemotherapy
- L2: docetaxel remains standard with limited efficacy and high toxicity

Opportunity for Tedopi®

- HLA-A2+ patients represent c.45% of NSCLC patients
- Great opportunity for new standard without chemotherapy in a remaining high medical need after 1st line of treatment

