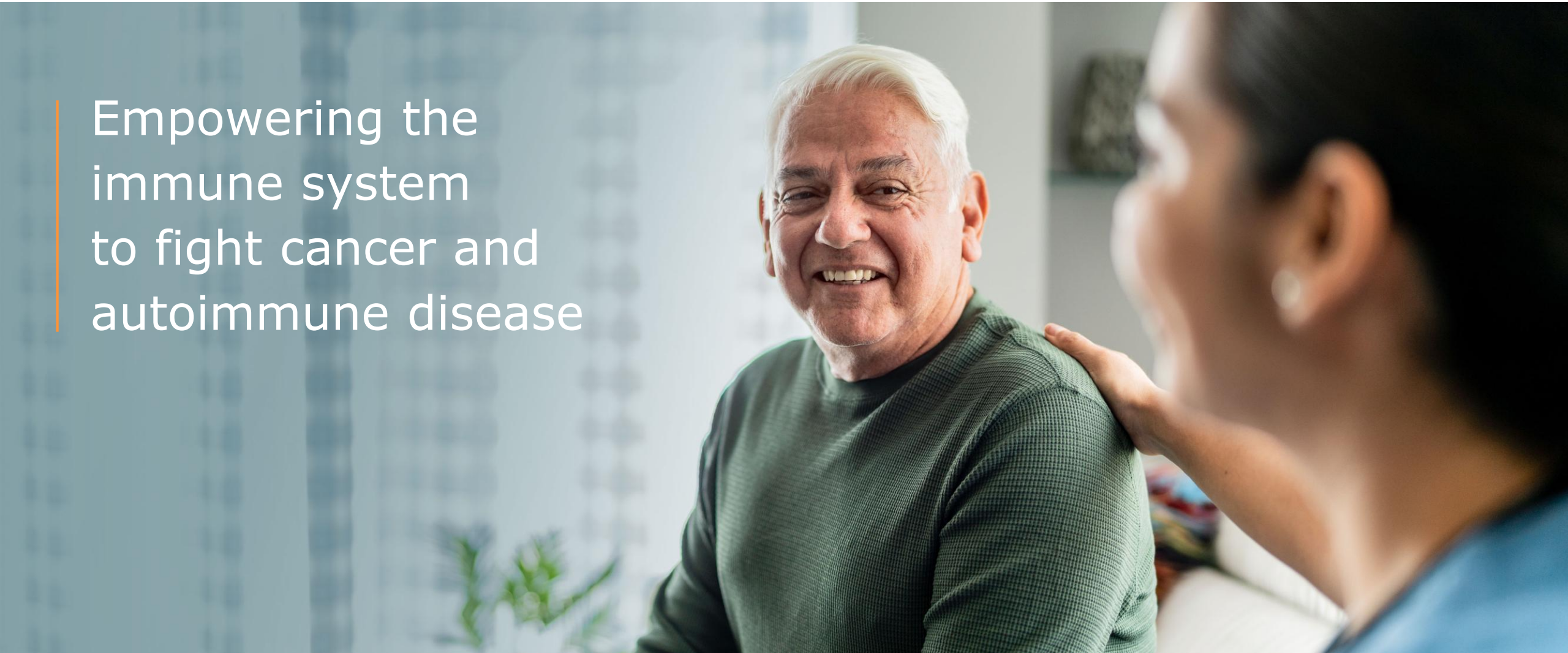
Immutep CEO and CSO AGM 2026 Presentation

27 November 2026 (IMM:ASX, IMMP:NASDAQ)

immutep
Empowering the Immune System



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Clinical data presented in this presentation for efti and IMP761 include results from earlier-stage, pooled and investigator-initiated studies. Such results are not necessarily predictive of the results of later-stage or registrational trials. In particular, the Phase III TACTI-004 trial evaluating efti in first-line non-small cell lung cancer was discontinued in March 2026 following a pre-planned futility analysis, and statements in this presentation regarding the potential of efti should be read in that context.

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This presentation is authorised for release by the Board of Immutep Limited.



Agenda

- Overview of Immutep
- Root Cause Analysis
- Efti’s Path Forward
- IMP761 Results & Path Forward
- Financials and 2027 milestones
- Summary

Overview of Immutep

Immutep overview

Focused on LAG-3 immunotherapy

Four clinical-stage assets designed to safely empower the immune system to fight cancer and autoimmune diseases through the MHC Class II & LAG-3 pathways, including first-in-class immunotherapies eftilagimod alfa (efti) and IMP761.

Diversified oncology & autoimmune pipeline

Immutep is advancing a pipeline with demonstrated clinical potential across high unmet need indications, leveraging differentiated immune modulation approaches in oncology and autoimmune diseases.

Validation via collaborations

Multiple partnerships and collaborations with large pharma and leading institutions.

MSD · Dr.Reddy's · labcorp · IKF · Narodowy Instytut Onkologii · GW Cancer Center

IP portfolio and balance sheet

Strong intellectual property (IP) portfolio and 12+ years of potential exclusivity for biologics like efti and IMP761.[1] Cash & cash equivalents including term deposits of A$48.5M at 30 September 2026, with expected runway well into H1 CY2028.[2]

1. Biologics Price Competition and Innovation Act (BPCIA).
2. Cash, cash equivalent and term deposit position of A$48.5M as at 30 September 2026; excludes €1.36M (~A$2.16M) French R&D tax incentive (CIR) received 28 October 2026. Cost-reduction measures are underway to preserve runway. Note: the Phase III TACTI-004 trial of efti in 1L NSCLC was discontinued in March 2026 following a pre-planned futility analysis; earlier-stage results are not necessarily predictive of later-stage outcomes.

FY26: A Year of Progress and Resilience

July 2025 – June 2026 | Significant progress across the portfolio despite the TACTI-004 setback

AUTOIMMUNITY

IMP761 advanced

Phase I single-dose safety objective met; significant human pharmacodynamic activity presented at EULAR 2026.

ONCOLOGY

STS delivered

EFTISARC-NEO met its Phase II primary endpoint; US FDA Orphan Drug Designation granted in April 2026.

REGULATORY

Clearer paths forward

Constructive FDA feedback for HNSCC CPS <1; Project Optimus dose alignment at 30 mg for efti.

BUSINESS DEVELOPMENT

Major licensing deal

Dr Reddy's agreement signed: up to US$349.5M in potential milestones plus royalties; US$10M net upfront payment.

SCIENTIFIC VISIBILITY

Global conference presence

Clinical findings at ESMO, CTOS, SABCS, ASCO and EULAR; new insights across cancer and autoimmunity.

EXECUTION & IP

Disciplined delivery

TACTI-004 reached 50% enrolment before futility; orderly wind-down initiated. 19 patents granted in FY26.

The setback was significant. The operational, scientific and strategic progress across FY26 was real.

Sources: FY26 Annual Report and FY26 quarterly reports.

Deep clinical pipeline in oncology & autoimmune diseases

	Indication – Trial	Phase I	Phase II	Phase III	Collaborations	Commercial Rights
ONCOLOGY						
Eftilagimod alfa (efti) MHC Class II agonist	**Lung Cancer** – TACTI-004	Efti + Pembrolizumab + Chemotherapy		DISCONTINUED*	MSD	Global manufacturing rights to efti & commercial rights in North America, Europe and Japan
	Lung / Head & Neck Cancers – TACTI-002	Efti + Pembrolizumab			MSD	
	Lung Cancer – INSIGHT-003[1]	Efti + Pembrolizumab + Chemotherapy			IKF	
	Head & Neck Cancer – TACTI-003	Efti + Pembrolizumab			MSD	
	Metastatic Breast Cancer – AIPAC-003	Efti + Chemotherapy				
	Early-Stage Breast Cancer[6]	Neoadjuvant Efti + Chemotherapy (Not Started)			GW Cancer Center	
	Soft Tissue Sarcoma – EFTISARC-NEO	Neoadjuvant Efti + Pembrolizumab + Radiotherapy			Narodowy Instytut Onkologii	
IMP701 (Ieramilimab) LAG-3 Antagonist Ab	Solid Tumour, Blood Cancer, TNBC, Melanoma[5]	Ieramilimab + anti PD1				
AUTOIMMUNE DISEASE						
IMP761 (LAG-3 Agonist)	**Healthy Volunteers**	IMP761				
IMP731 (LAG-3 Depleting)	**Psoriasis & Ulcerative Colitis**[2]	IMP731				

OUTLICENSED - ONCOLOGY

Dr.Reddy's — Commercial rights to efti in all countries except North America, Europe, Japan and Greater China[3]

EOC — Commercial rights to efti in Greater China[4]

Information as of Nov 2026. *Press release, 13 March 2026.

1. Investigator-initiated trial that Immutep has no control over.
2. Trials for IMP731 were conducted by GSK (two Phase I in healthy volunteers & psoriasis and a Phase II in ulcerative colitis), which transitioned this asset back to Immutep in 2024.
3. In December 2025, Immutep entered into a licensing agreement with Dr Reddy's for efti in countries outside N America, Europe, Japan and Greater China. Immutep is eligible to receive up to US$349.5M in milestone payments and double-digit royalties on sales of efti.
4. For EOC's China rights, Immutep may receive milestones plus royalties.
5. To date, Novartis has conducted trials with IMP701 in solid tumours, blood cancers, TNBC and melanoma. Subsequent to 30 June 2026, Novartis gave notice terminating the out-licence agreement relating to ieramilimab, effective 9 August 2026. The licence is not generating revenue for Immutep and no further milestone or royalty payments are anticipated. Following termination, Novartis is required to assign its ownership interest in the jointly owned LAG-3 patents to Immutep S.A.S.
6. Investigator-initiated trial with the GW Cancer Center; not yet started and remains on hold pending strategic decisions following the TACTI-004 root cause analysis.

Two priorities, one value-creation strategy

Immutep is advancing efti and IMP761 as priorities, potential for 3 Phase II trials

Efti

Focused registration directed oncology development

- Prioritise 1L HNSCC CPS <1 and neoadjuvant resectable STS → chemo-free effective immunotherapies
- Build on differentiated MHC Class II / APC activation biology and encouraging mature clinical data
- Target to start registration-directed clinical development in H2 CY2027*

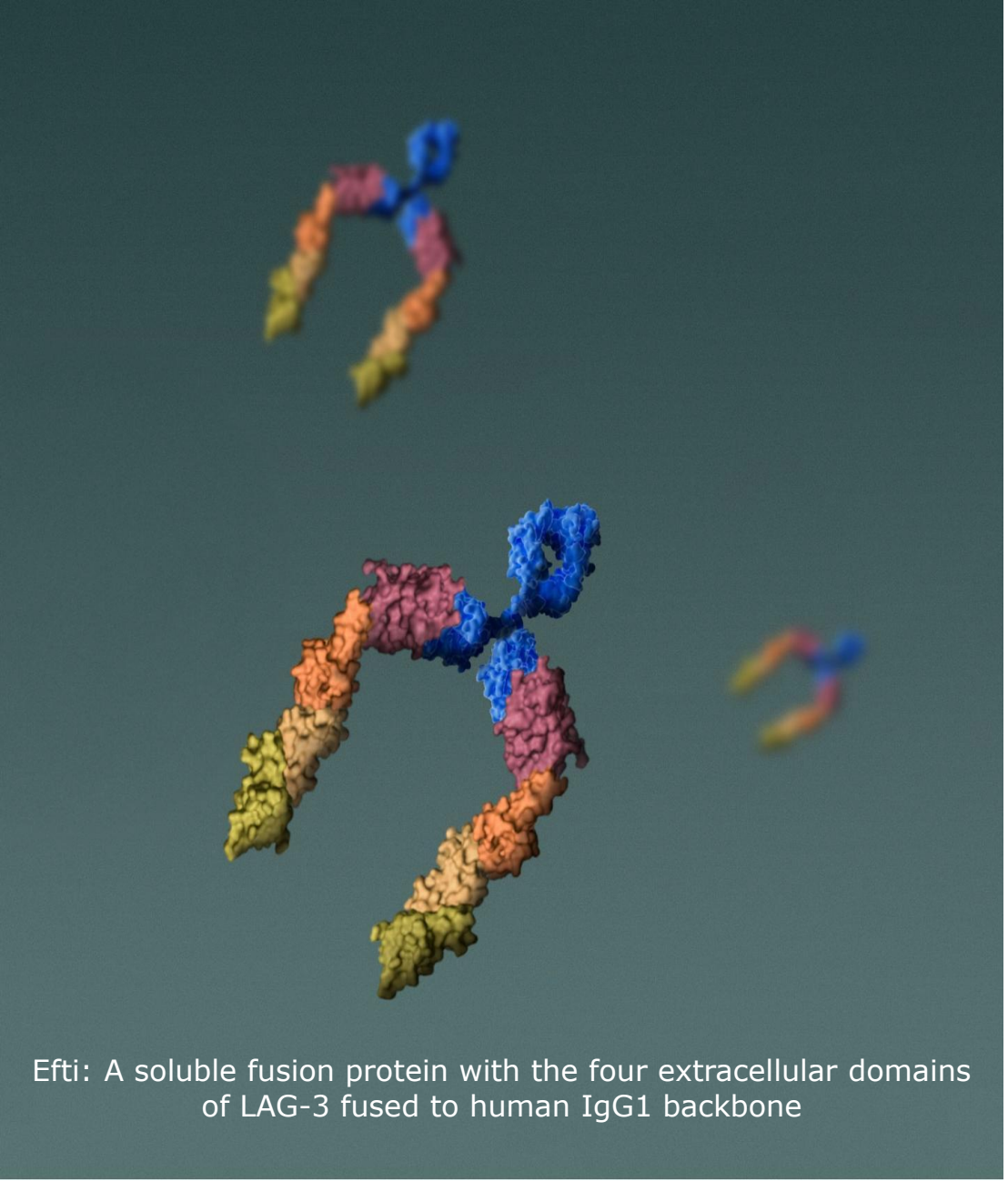
IMP761

Targeted immune modulation in autoimmunity preparing for Phase II

- Complete Phase I clinical study
- Target to start clinical Proof of Concept in patients with rheumatoid arthritis in H2 CY2027*
- Evaluate other indications for late-stage development using biology, feasibility and commercial value

Portfolio objective: two differentiated first-in-class assets, diversified across oncology and autoimmunity

* subject to final decisions on strategy and trial design, regulatory interactions, manufacturing timelines, partnering and resources



Efti: A soluble fusion protein with the four extracellular domains of LAG-3 fused to human IgG1 backbone

Oncology

Eftilagimod alfa (efti):

Only immunotherapy in clinical development that activates antigen-presenting cells or APCs (e.g. dendritic cells, monocytes) directly via the MHC Class II pathway to fight cancer

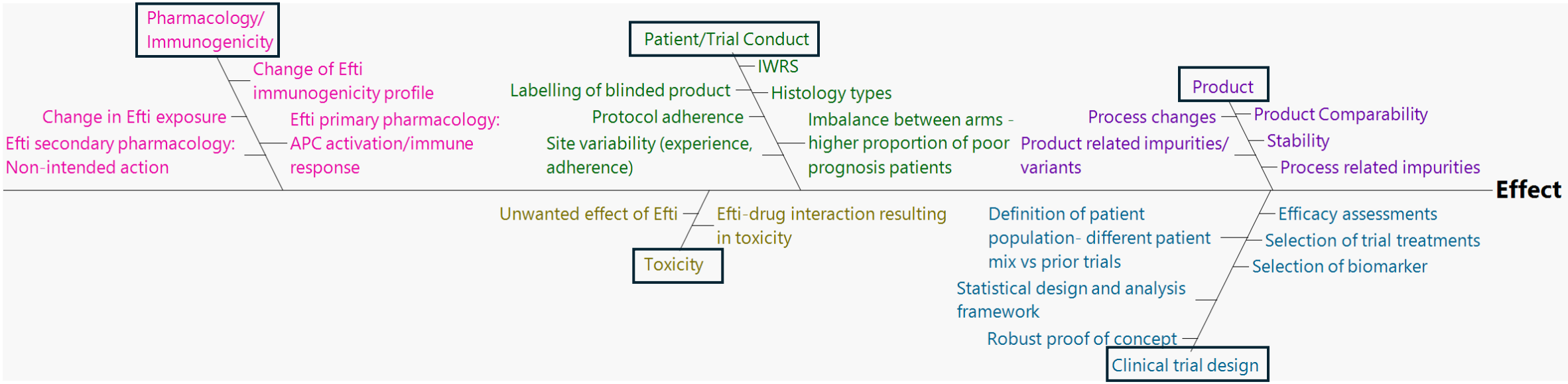
Root Cause Analysis

TACTI-004

Root cause investigation through fishbone analysis

In-depth RCA with a Cause and Effect (Fishbone) Diagram:

- To prevent "Tunnel Vision": **assess all potential causes** and identify **Multiple Contributors** through a **cross functional** collaboration.
- To conduct a **Hypothesis-Driven** Investigation with a **Strategic Remediation**
- Eventually provide a **"Path forward"** to ensure next clinical development steps



Futility of TACTI-004 in testing of Efti + SoC compared to placebo + SoC was observed, potentially suggesting a different clinical behaviour

TACTI-004 root cause analysis: findings

Differences found in the 2,000 L product and in efti's behaviour in patients

Category investigated	Explains the result?
Toxicity and safety	**Not supported**
Clinical trial design	**Not supported**
Participant characteristics and trial conduct	**Not supported**
Pharmacology, immunogenicity and drug exposure (PK)	**Differences found**
Product (manufacturing)	**Differences found**

Examples of factors investigated and not supported

- Control arm: performed within the expected range
- Baseline: arms balanced (e.g. histology, tumour burden)
- Assessment timing: ORR gap confirmed at final analysis
- Drug handling and labelling: no errors explaining the result
- Safety: no new risk attributable to efti identified

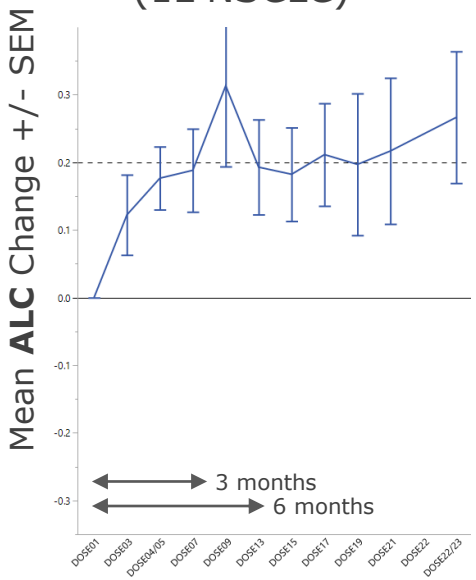
PK: pharmacokinetics.

TACTI-004: unexpected fall in immune-cell counts in futility

PD marker observations deviate from previous results

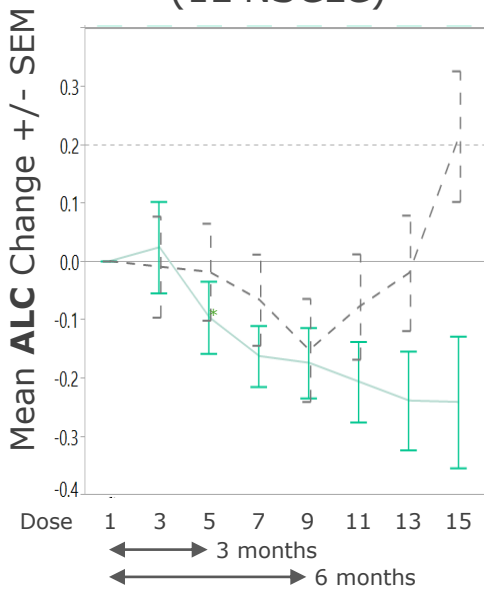
Absolute Lymphocyte Count (ALC)

TACTI-002
(1L NSCLC)



Efti+Pembro

TACTI-004
(1L NSCLC)

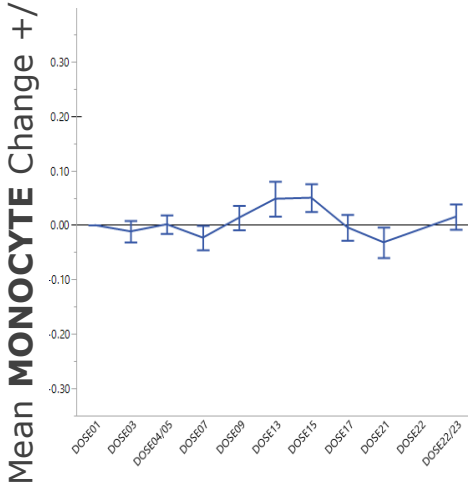


Efti+SoC	75	75	66	60	56	44	34	22
Placebo+SoC	82	82	78	75	56	56	45	34

TACTI-004: Unexpected ALC decrease in the efti arm compared to placebo.

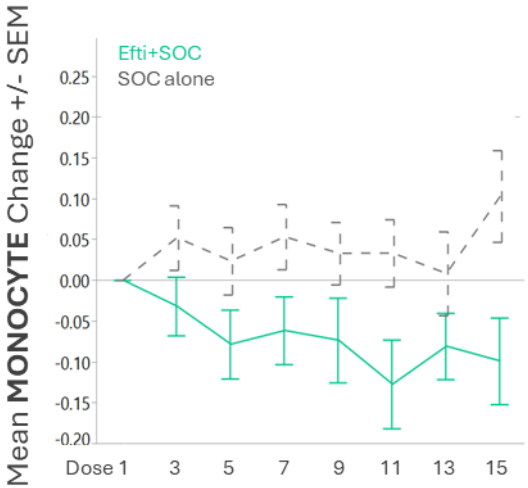
Monocyte Count

TACTI-002
(1L NSCLC)



Efti+Pembro

TACTI-004
(1L NSCLC)

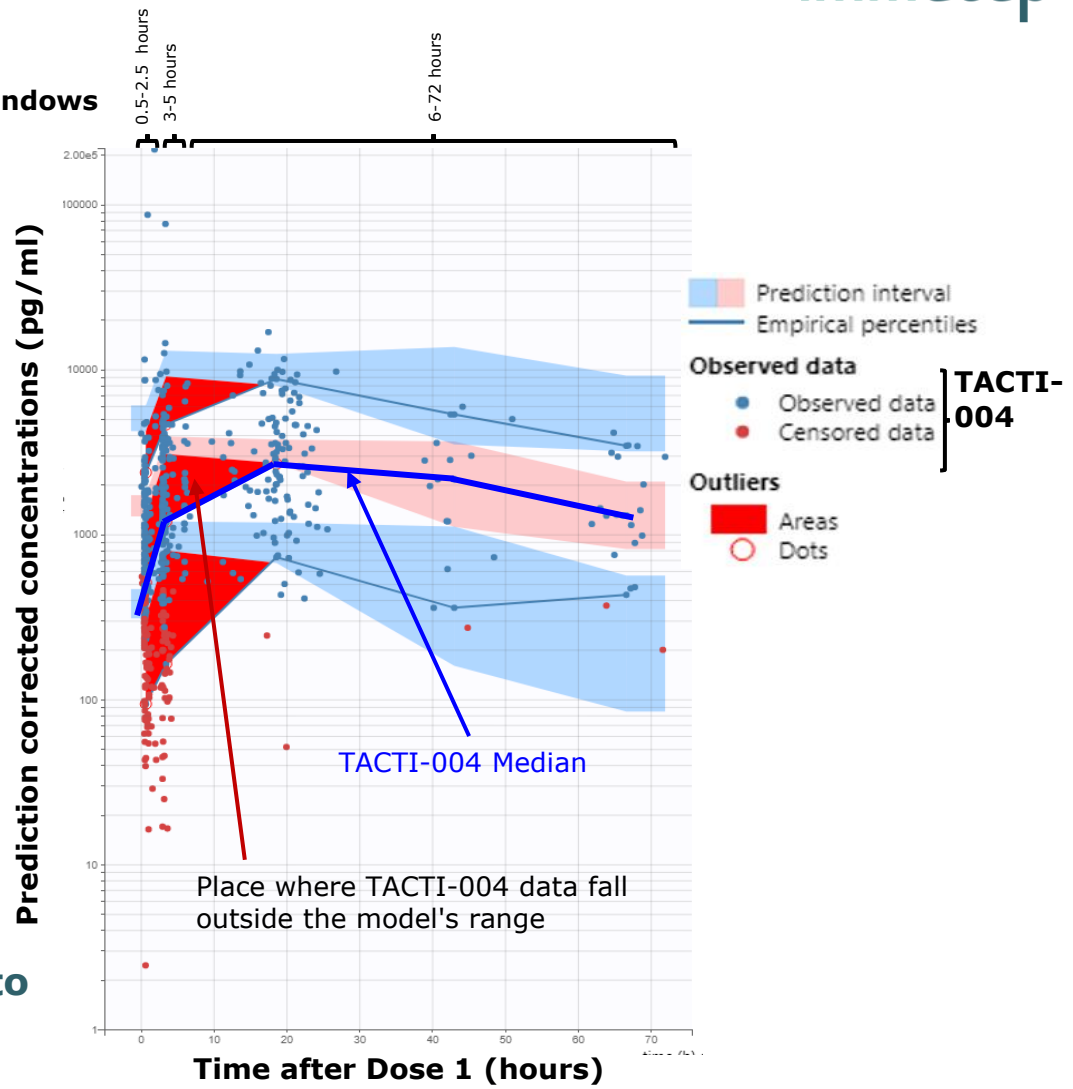


TACTI-004: Unexpected monocyte (target cells of efti) count decrease only in the efti arm and not in placebo.

TACTI-004: unexpected efti exposure

Comparison of efti blood concentration with earlier clinical studies (population PK model):

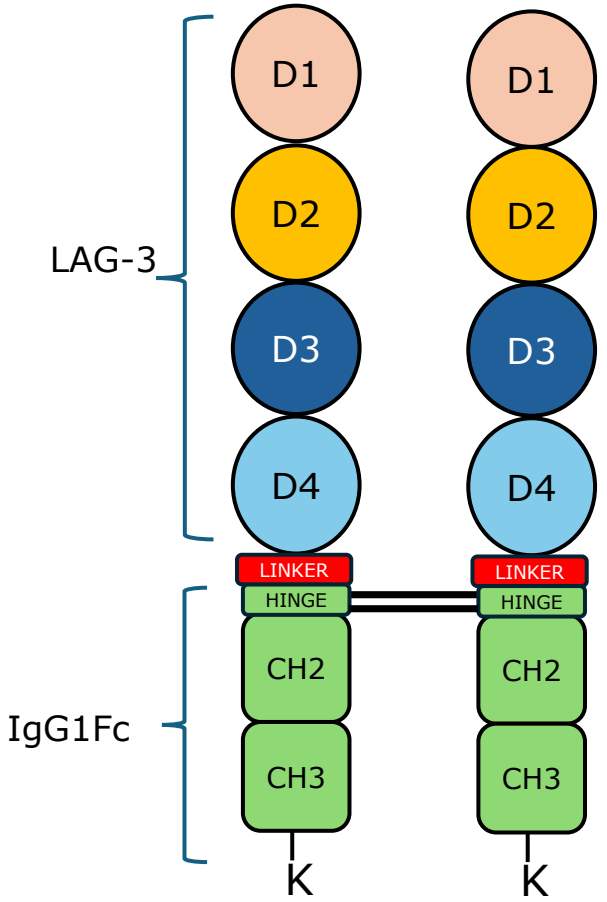
- The graph shows efti concentration in blood vs time from the existing popPK model (Median, 5th and 95th percentiles) where TACTI-004 PK data points at dose 1 are shown
- The ~10-fold spread between 5th and 95th percentiles is expected for this type of data
- 20% of patients had no measurable efti in sampled blood tests (censored data, red dots)
- Even after excluding these 20% flat PK patients, early concentrations were lower than predicted (red patches)
- Exposure ($AUC_{0\text{-}48hr}$) is reduced by 40%



Takeaway: changes in glycan profiles and abundance are known to impact PK and biodistribution. Altered exposure/biodistribution could have contributed to the futility outcome.

Source: TACTI-004 pharmacokinetic analysis, Cut-off: August 2026

Efti is a glycoprotein, i.e. a protein with sugars attached



Domain 1: O-glycan site

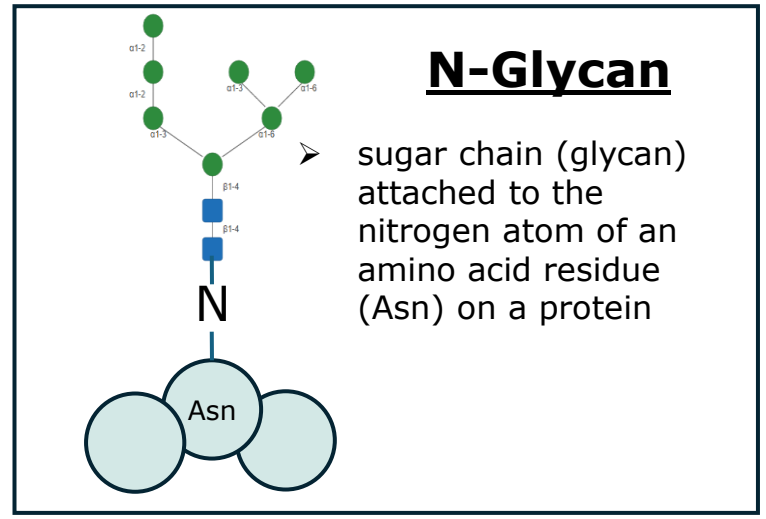
Domain 2: 3 x N-glycan sites

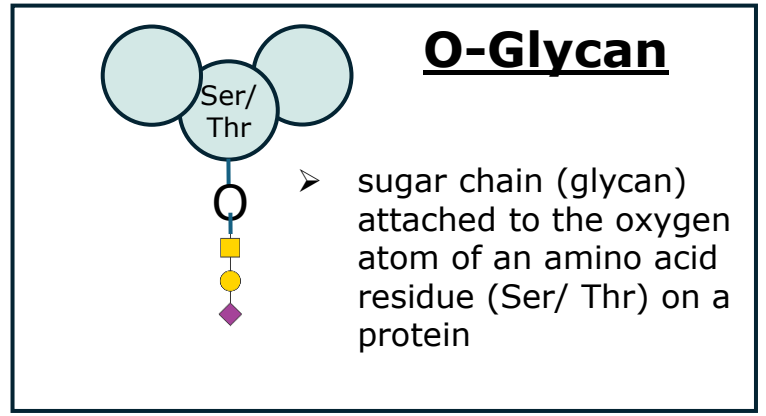
Domain 3: N-glycan site

Linker: O-Xylose site

Fc: N-glycan site

Sugars account for ~10% of the molecular mass of Efti, compared with ~2-3% for typical IgG mAbs**





Type of sugars attached to Efti (below) – a complex decoration for manufacturing and analytics

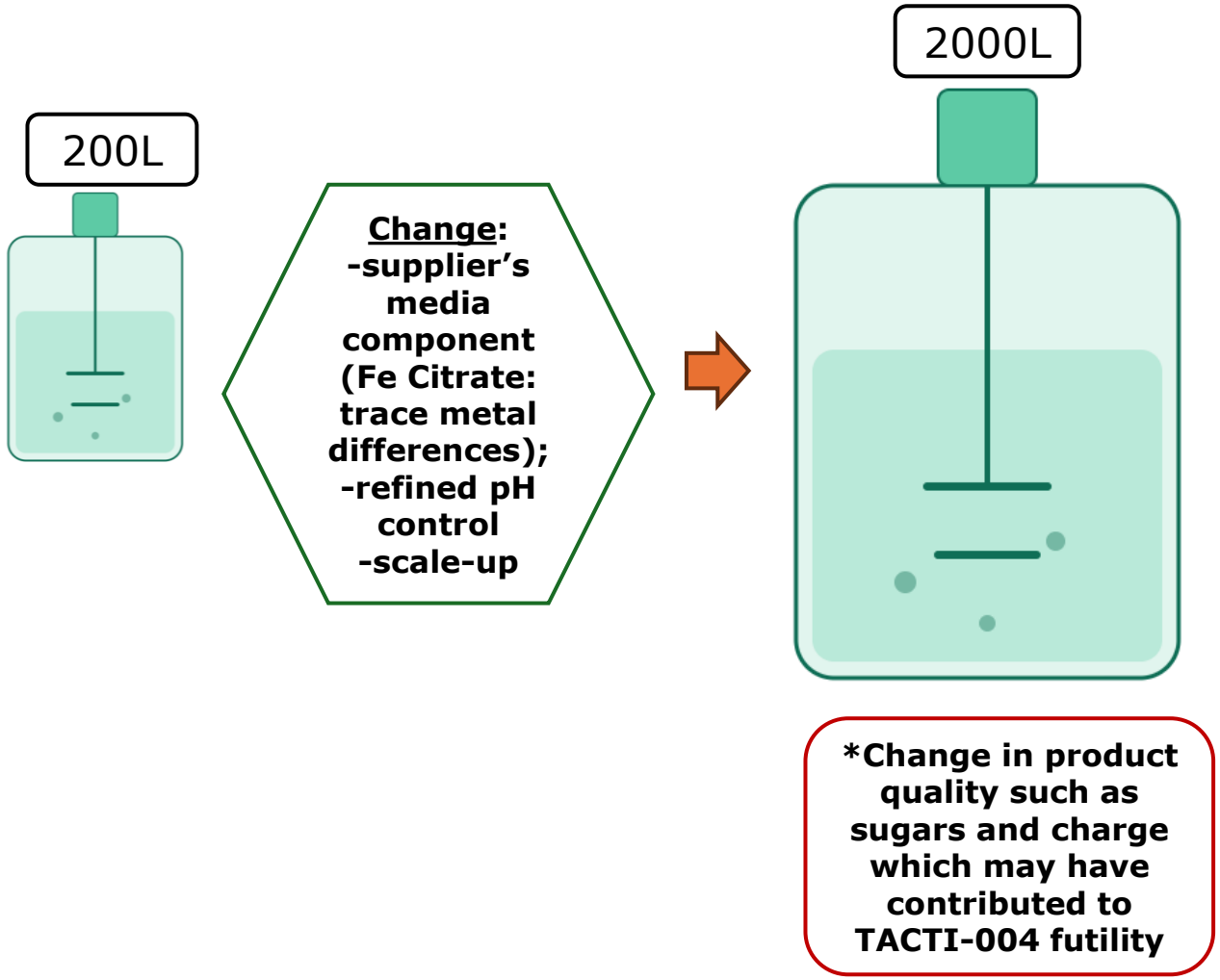


***As of 31 December 2025, FDA has approved 113 novel IgG based antibodies and 16 Fc fusion proteins.**

*William R Strohl, Structure and function of therapeutic antibodies approved by the US FDA in 2025, *Antibody Therapeutics*, Volume 9, Issue 3, July 2026, Pages 332–366, https://doi.org/10.1093/abt/tbaq027

**Mimura, Y., Katoh, T., Saldova, R. et al. Glycosylation engineering of therapeutic IgG antibodies: challenges for the safety, functionality and efficacy. Protein Cell 9, 47–62 (2018). https://doi.org/10.1007/s13238-017-0433-3

What changed from 200L to 2000L



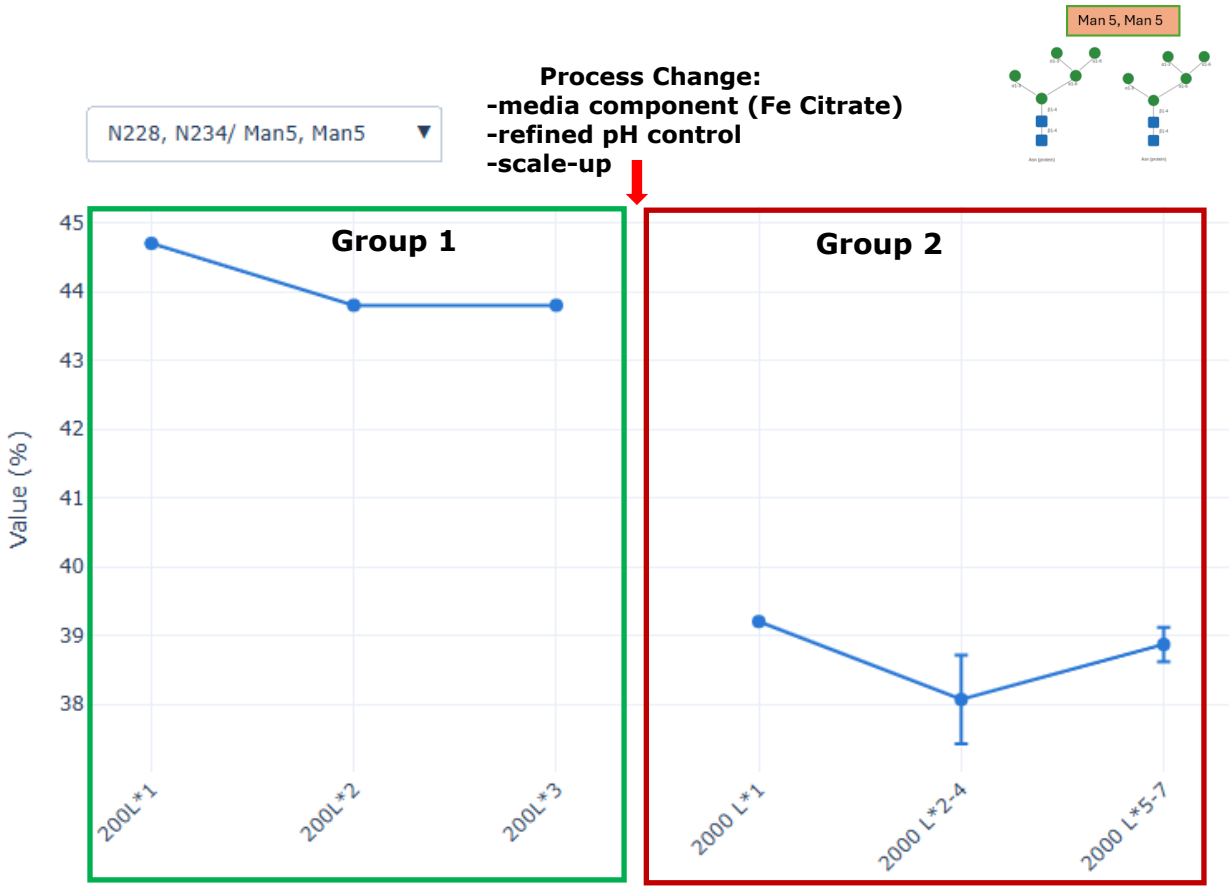
Efti GMP release test for N-glycan analysis was performed on par with industry standards

*Changes and minor differences after scale-up to 2000L were reviewed by external consultants and regulatory bodies and 2000L and 200L material were assessed to be comparable. Phase III TACTI-004 results indicate that changes and subtle differences might play a role in product efficacy and biodistribution in NSCLC.

Differences in glycans discovered in RCA

LC-MS/MS **N-Glycan results** - principal component analysis (PCA)





N-glycan structure on LAG-3 D2

- 47 N-glycans identified by MS were given as input into PCA (unsupervised)
- Batches from different scales (200L and 2,000L) analysed
- Statistically distinct batch groups emerged from the data without any user input
- PCA confirms change of process conditions and scale introduce small changes in abundance of specific sugar structures attached to protein
- An example of such a change is shown on the right, differentiating Group 2 (2,000L) from Group 1 (200L)

TACTI-004 root cause analysis: key findings

What the evidence points to

1. **Product:** differences between 2,000L and 200L material in post-translational modifications, including N- and O-linked glycans

2. **Immune profile:** early fall of lymphocyte and monocyte counts in efti-treated TACTI-004 patients vs control arm or historical trials

3. **Drug exposure:** no detectable efti at sampled time points after dosing in ~20% of patients (44/221) and lower than expected early concentrations (40% lower exposure), consistent with altered absorption or tissue distribution

4. **Laboratory activity:** no relevant differences between batches or scales in APC activation, MHC II binding or Fc-mediated activity; immunogenicity consistent with earlier trials

Immune profile: % of efti-treated participants with a >40% fall from baseline up to dose 7 (~3 months). Lymphocytes: TACTI-004 24.8% (N=151); INSIGHT-003 14.0% (N=54); TACTI-002 8.3% (N=189); TACTI-003 5.3% (N=102). Monocytes: 35.1%; 12.8%; 12.6%; 3.8%. Cross-trial, non-randomised comparison; data cut-off August 2026. PK: pharmacokinetics; APC: antigen-presenting cell.

RCA outcome

Outcome: The RCA identified measurable differences in efti product characteristics following manufacturing changes and scale-up, alongside unexpected pharmacokinetic and immune-cell observations in TACTI-004. These findings provide a scientifically plausible explanation for the unexpected clinical outcome, although a direct causal relationship has not been established.

Scientifically plausible sequence of events



Efti’s Path Forward

Learnings from RCA: Manufacturing path forward for efti

- Next 200L batch has been contracted.
- Raw materials, including their trace-metal content, will go through extended qualification before the next 200L batch is made.
- Additional characterisation assays will be applied to every future batch, so that the product characteristics seen in the TACTI-004 material would be excluded.
- 200L batches will be extensively characterised, including glycans.

Efti in oncology: chemo-free path to potential BLA filing in 2 indications

Indication selection

- High unmet need indications with limited (or no) competition, low bars for market entry
- Supportive translational / clinical data to support late-stage development
- If possible regulatory shortcuts/benefits like fast-track or orphan indication

Key differentiation

- Chemo-free immunotherapy combination → less toxicity & potentially long-lasting efficacy

Commercial Opportunity *

- 1L HNSCC with CPS <1: ~32k new CPS <1 cases p.a. (US, EU4+UK, India, China, Japan) → **US$960M** commercial opportunity
- Neoadjuvant soft tissue sarcoma: ~60k resectable new cases p.a. (US, EU4+UK, India, China and Japan), a **US$2.70B** commercial opportunity

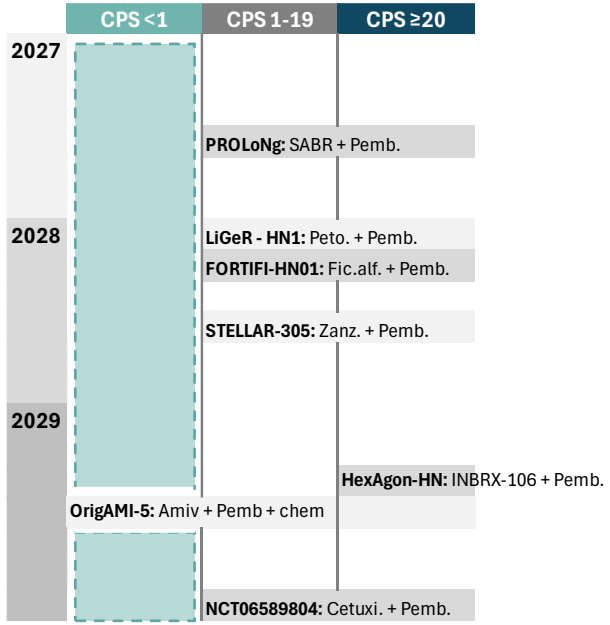
* Source: Syneos Health analysis

Efti & anti-PD-1 in 1L HNSCC CPS <1: a chemo-free opportunity

High Unmet Need

Median OS of ~11 months with SoC with severe toxicity

Limited competition with no chemo-free late-stage development for CPS <1[a]



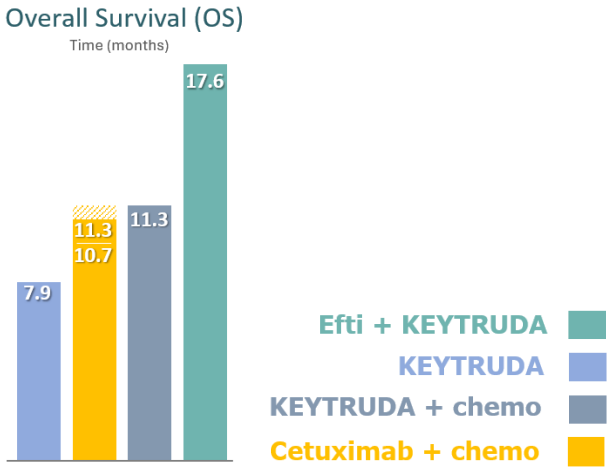
Clinical Data to Date

TACTI-003, CPS <1:

OS 17.6 months vs 7.9–11.3 for current SoC (cross-trial comparisons)[b]

Safety markedly better vs chemo regimens (12% vs 70% of ARs ≥Gr.3 in historic control)

ORR 30–40%, matching chemo without its toxicity



US$960M Opportunity

Fast Track designation granted and path to BLA discussed with US FDA

~32k new CPS <1 cases p.a. across US, EU4+UK, India, China, Japan → US$960M segment

213k
2026 R/M incident cases[1,2]

~32k
#calculated CPS <1 population[3]

US$9.39B
global H&N total drug cancer market (2025)[7]

US$960M
calculated 1L R/M HNSCC CPS <1 segment[3]

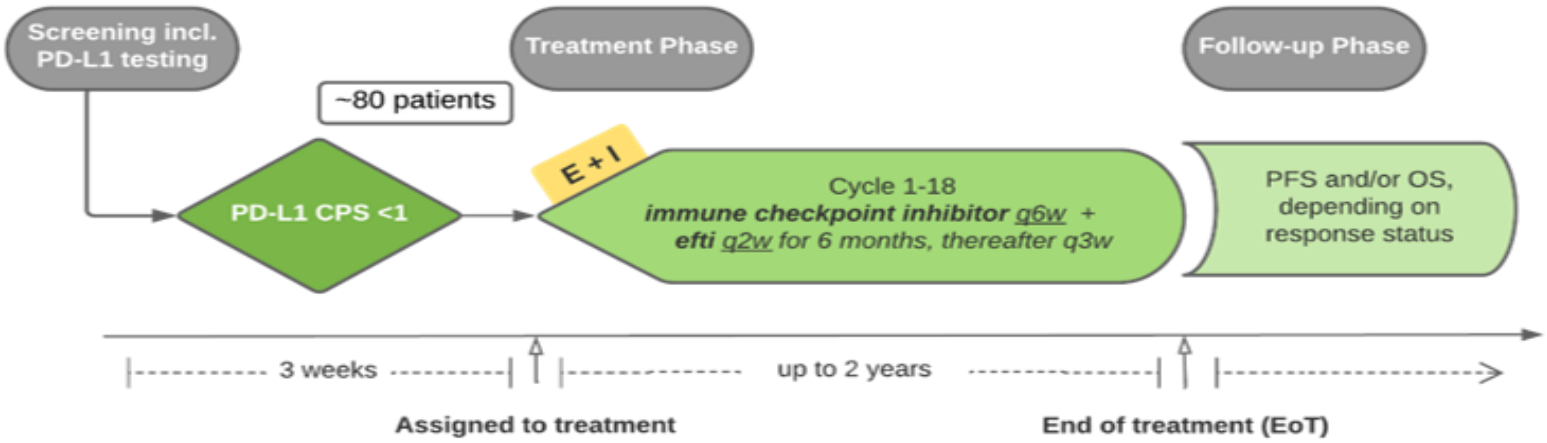
10.5%
HNSCC sales CAGR 2026-2036[8]

Sources: a. Extracted from GlobalData Oct 2026. b. Comparison of data is from different clinical trials: Efti + KEYTRUDA (pembrolizumab) from TACTI-003, Cohort B; KEYTRUDA (pembrolizumab) monotherapy, pembrolizumab with chemotherapy, and cetuximab with chemotherapy from KEYNOTE-048. Opportunity box sources: 1. GlobalData, extracted Jul 2026; 2. Clarivate, extracted Jul 2026; 3. Syneos Health analysis, CPS <1 population calculated as ~15% of 2026 R/M HNSCC incident cases, based on KEYNOTE-048; 7. Fortune Business Insights; 8. DelveInsight HNSCC market report.

Phase II design in 1L HNSCC CPS <1

Strategy:

- Confirm robust ORR, DOR and safety in a second independent study (after TACTI-003) before starting Phase III
- Fast Track designation → potential BLA filing on Phase II ORR / DOR / safety while a confirmatory Phase III is ongoing



Legend: PD-L1 = programmed cell death ligand 1; CPS= combined positive score; 1 cycle = 6 weeks; q2w = every 2 weeks; q3w = every 3 weeks; q6w = every 6 weeks; E + I = efti + immune checkpoint inhibitor

Phase II Design Parameters under consideration*	
Study design	Non-randomised trial with efti + anti-PD-1 with ~80 pts with 1L HNSCC and CPS <1
Endpoints	ORR as primary; safety and DOR as key secondary

* Note: Design parameters are preliminary and illustrative only. The final study and statistical design remains subject to ongoing discussion with all stakeholders, health authority feedback and ethics committee review, and may change materially. Nothing here constitutes a commitment.

Efti + anti-PD-1 in neoadjuvant soft tissue sarcoma, an orphan indication

High Unmet Need

Orphan indication with very high unmet need as recurrence and metastasis rates remain high[a]

Standard treatment is surgery with optional radiotherapy and/or chemo

Lack of established systemic treatment options and remains an emerging area for immunotherapy development

No established Phase III studies to date and limited late-stage competition

Current SoC for resectable STS:

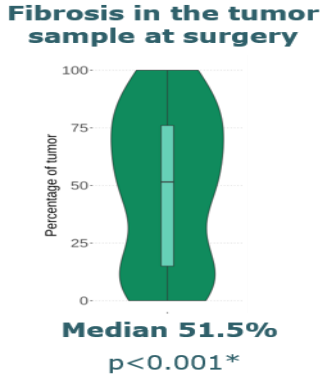


No Active Phase III trials with Immunotherapy

Clinical Data to Date

Over 3x greater tumor hyalinization/fibrosis than radiotherapy alone: median 51.5% vs 15% at surgery (historical control; p<0.001)[b]. *Tumor hyalinization/fibrosis is the surrogate most closely correlated with treatment outcome and improved survival*[c]

Disease-free survival at 12 months >80% vs ~60% expected with radiotherapy alone (EFTISARC-NEO vs historical control) as presented at ESMO 2026



US$2.70B Opportunity

Sizeable orphan market: ~60k resectable new cases p.a. in US, EU4+UK, India, China and Japan, a US$2.70B illustrative IO opportunity**[3]

Low entry hurdle: no immunotherapy approved in STS today

Orphan advantages: US FDA Orphan Drug Designation granted → market exclusivity and other regulatory incentives on approval

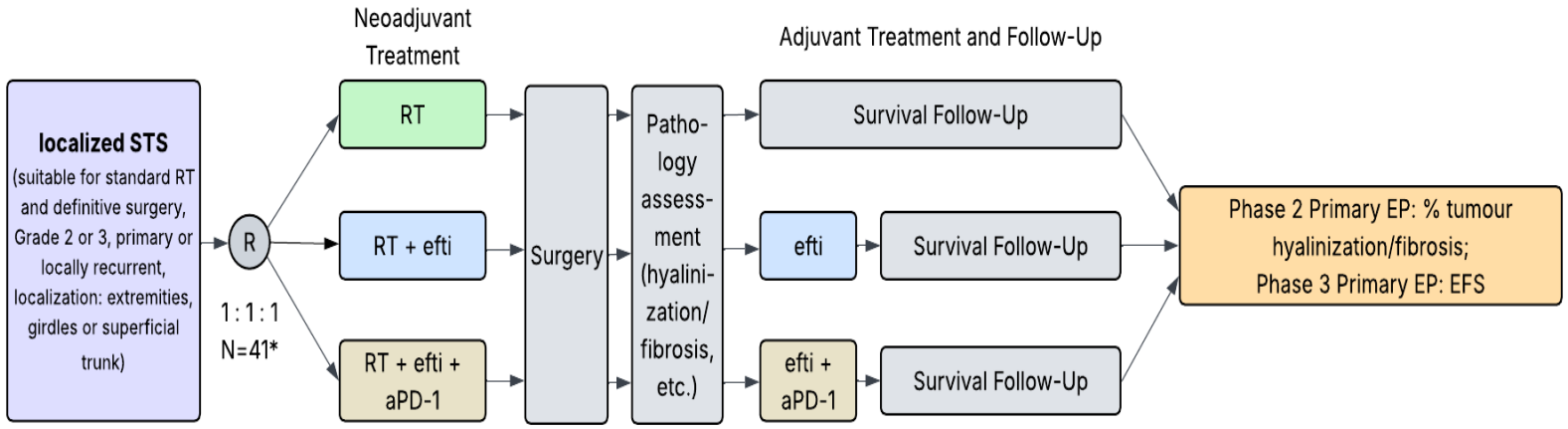
102k 2026 incidence[1]	**~60%** reported resectable share of STS market[4-6]	**~60k** calculated resectable new STS cases[3]
US$2.98B global sarcoma drug market (2025)[7]	**US$2.70B** STS IO opportunity, based on epi and average IO price[3]	**7.9%** STS sales CAGR 2026-2036[9]

Sources: a. SEER Cancer Stat Facts: Soft Tissue Sarcoma; and ESMO–EURACAN–GENTURIS Clinical Practice Guidelines, Ann Oncol 2021;32(11):1348–1365. b. ESMO Congress 2025 Proffered Paper Oral Presentation and CTOS 2025 Oral Presentation; comparison to historical data using statistical assumptions, historical RT data from Schaefer M. et al. Int J Radiat Oncol Biol Phys 2017, detailed at ESMO 2025. c. Rao SR et al., Cancer Med. 2022;11(1):194–206, doi:10.1002/cam4.4428. Opportunity box sources: 1. GlobalData, extracted Jul 2026; 3. Syneos Health analysis; 4. Market Research Future report; 5. Verified Market Reports; 6. IMARC report; 7. Fortune Business Insights; 9. DelveInsight soft tissue sarcoma market report.

Trial design in soft tissue sarcoma (STS)

Strategy:

- Build the regulatory strategy on the US FDA Orphan Drug Designation granted Q2 2026: market exclusivity and other regulatory incentives
- Use the Phase II hyalinization/fibrosis readout to select the arm(s) to carry forward and expand Phase III enrolment
- Pursue BLA submission based on interim Phase III data, pending final event-free survival (EFS) results



*Enrollment will continue until N=41 evaluable subjects per treatment arm are reached.
Additional 34 subjects will be enrolled into each treatment arm that is carried forward into the Phase 3 part.
aPD-1: anti-programmed cell death protein 1; efti: eftilagimod alfa; RT: radiotherapy

Adaptive Phase II/III design under consideration: early go/no-go decision and seamless expansion into Phase III without a new trial*	
Study design	• Randomised adaptive Phase II/III with ~120 patients (Phase II) + up to 105 additional (Phase III)
Endpoints	• Phase II: % of hyalinization/fibrosis in tumor at surgery[#] • Phase III: event-free survival (EFS)

*Note: Design parameters are preliminary and illustrative only. The final study and statistical design remains subject to ongoing discussion with all stakeholders, health authority feedback and ethics committee review, and may change materially. # The percentage of hyalinization and fibrosis, as a surrogate of pathological response, appears to be most closely correlated with treatment outcome, source: Rao SR et al. Extent of tumour fibrosis/hyalinization and infarction following neoadjuvant radiation therapy is associated with improved survival in patients with soft-tissue sarcoma. Cancer Med. 2022 Jan;11(1):194-206. doi: 10.1002/cam4.4428. Epub 2021 Nov 27.

IMP761 Results & Path Forward

Innovative first-in-class opportunity


IMP761: A LAG-3 agonist antibody

Autoimmune diseases

IMP761:

First-in-class LAG-3 agonist antibody designed to **selectively silence autoreactive LAG-3$^+$ T cells**, the root cause of many autoimmune disorders

THEORY
Before Phase I

Targeting the root cause – pathogenic LAG-3$^+$ T cells – a paradigm shift in treating autoimmune disease.

REALITY
After Phase I

Significant, positive data show it could become reality.

Autoimmune disease: a vast and debilitating problem

The problem: T cells and B cells are meant to defend us. In autoimmune disease they turn against the body's own tissue, causing chronic inflammation and damage. Today's drugs mainly suppress the whole immune system, at the price of serious side effects[1].

WORLDWIDE

7–10%

of the world's population is affected[2]; >100 distinct autoimmune diseases are described[3]

EUROPEAN UNION (EU)

~23–45 million

people estimated to be affected in the EU[4]

UNITED STATES (USA)

~17–27 million

people estimated to be affected by autoimmune disease in the US[5,6]

T cells sit at the centre of autoimmunity[1,7]

T cells

play a central role in the pathogenesis of autoimmune disease[1,7]

Examples of diseases with T-cell involvement[1,7]

Rheumatoid arthritis	Type 1 diabetes	Multiple sclerosis
Psoriasis	IBD (Crohn's/UC)	Celiac disease
Lupus (SLE)	Sjögren's	Hashimoto's
Vitiligo	Alopecia areata	Myasthenia gravis
Graves' disease	Pemphigus	

Market size estimates – IMP761 target indications

US$29.1B	Rheumatoid arthritis · 8MM, 2029
US$9.9B	Type 1 diabetes · 7MM, 2033
US$23.6B	Multiple sclerosis · 7MM, 2029

Source: GlobalData[8]; MM = major markets

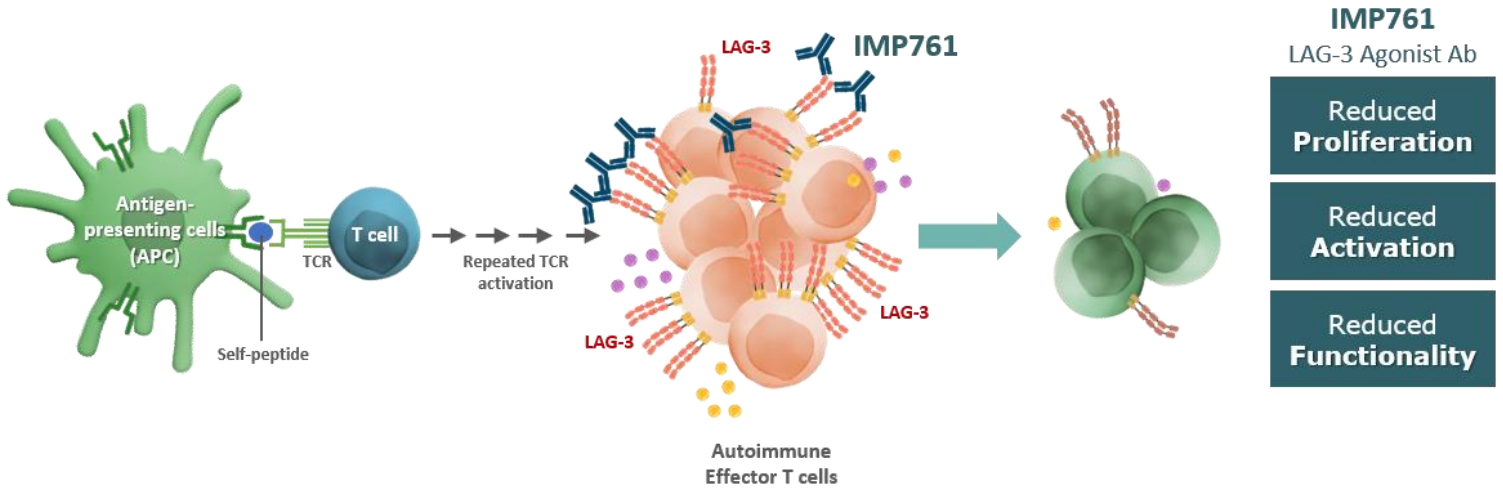
The unmet need: only switch off the over-active T cells themselves – the aim of IMP761, a LAG-3 agonist antibody.

EU and US estimates = prevalence × population: EU 5–10%[4] × ~450 million (2025); US 5–8%[5,6] × ~343 million (2026).

Notes: [1]Song Y et al. Signal Transduct Target Ther 2024;9:263. [2]Kumar M, Yip L, Wang F, Marty S-E, Fathman CG. Front Immunol 2025;16:1626082. [3]Abend AH et al. J Clin Invest 2025;135(4). doi:10.1172/JCI178722. [4]Global Autoimmune Institute. The Global Landscape of Autoimmune Disease. autoimmuneinstitute.org/articles/the-global-landscape-of-autoimmune-disease/ [5]PubMed Central PMC11827834 (pmc.ncbi.nlm.nih.gov/articles/PMC11827834/). [6]NIH Office of Autoimmune Disease Research (OADR-ORWH), orwh.od.nih.gov/OADR-ORWH. [7]Pisetsky DS. Nat Rev Nephrol 2023;19:509–24. [8]GlobalData market estimates: RA 8MM 2029; T1D 7MM 2033; MS 7MM 2029.

Selective silencing of autoreactive T cells

- ✓ Autoimmune memory T cells are chronically stimulated by the same self-peptide which leads to LAG-3 expression. **LAG-3 expression marks autoreactive memory T cells expressed in inflamed tissues**.
- ✓ The physiological role of LAG-3 is to interfere with T cell stimulation by specifically downmodulating T-cell receptor (TCR) signaling.
- ✓ IMP761, by forming LAG-3 "dimers of dimers," increases this physiological down-regulation.



IMP761 promotes natural LAG-3-mediated down-regulation of auto-reactive memory T cells (root cause of many diseases)

Grebinoski S, Vignali DA. Inhibitory receptor agonists: the future of autoimmune disease therapeutics? Curr Opin Immunol. 2020 Dec;67:1-9. doi: 10.1016/j.coi.2020.06.001. Epub 2020 Jun 30. PMID: 32619929; PMCID: PMC7744338.

Proof of concept with gold-standard test

Randomised, double-blind, placebo-controlled
A Phase I trial that sets a high bar, this early in development.

60 healthy volunteers, broad dose range
Doses from 0.03 to 14 mg/kg create a robust dataset for later clinical development and regulatory interactions.

KLH neoantigen challenge: robust and standardized
To test T-cell targeting agents; mimics a strong T-cell immune response and is a good surrogate for the later disease setting.

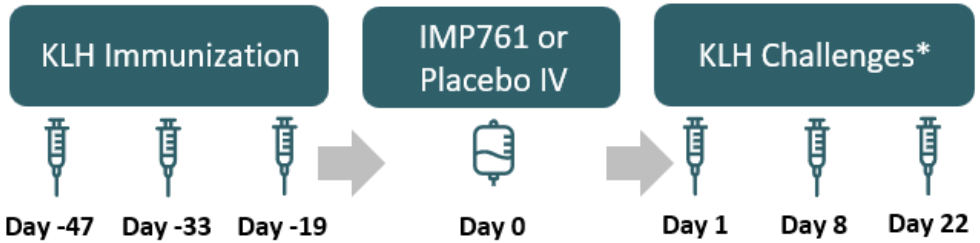








Single ascending dose (SAD) of IMP761 (N=60)
Objectives: Safety, PK, PD
7 dose levels (0.03 - 14mg/kg), 1 placebo group
KLH challenge model



Conducted by Centre for Human Drug Research (Leiden, The Netherlands), a leading institute standardizing the approach

Top-line results showed **significant inhibition of antigen-specific T-cell immune responses** and generated enthusiasm among European (EULAR) and American (ACR) rheumatologists.

Phase I goals met: favourable safety, supports 4-weekly dosing

Phase I goals achieved: favourable safety and tolerability (primary endpoint met)

Safety is decisive in autoimmune disease, where patients may need treatment for many years.

0

Severe or serious adverse events

None reported at any of the 7 dose levels tested (0.03–14 mg/kg).

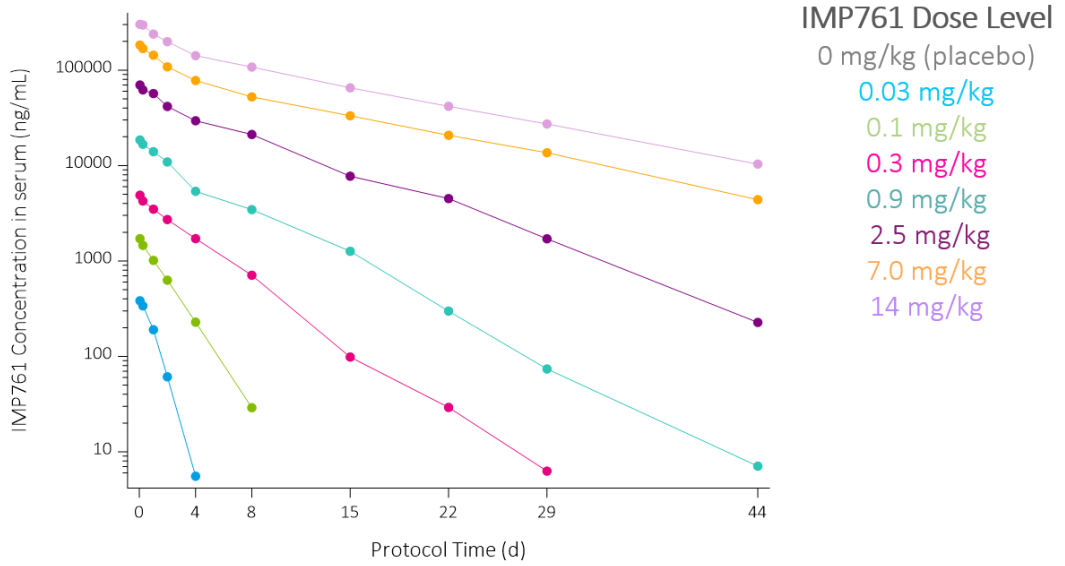
7 / 7

Dose levels well tolerated

Adverse-event rates comparable to placebo (3.1 vs 3.2 events per person).

Pharmacokinetics: supports 4-weekly dosing

After a single infusion, IMP761 stays in the blood for weeks at higher doses (2.5–14 mg/kg). These single-dose data support 4-weekly dosing.



Mueller C, et al. IMP761, a Novel Immunosuppressive LAG-3 Agonist Antibody in the Clinic. Late-breaking poster LB09, ACR Convergence 2026, Orlando, 6-11 Nov 2026.
Data shown are from the ongoing Phase I randomised, placebo-controlled, double-blind first-in-human study (NCT06637865); interim data from the single ascending dose part.

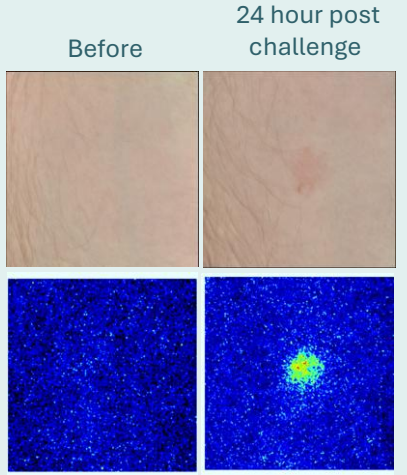
Proof in humans: clinical and scientific endpoints move together

Key Interim Phase I Results

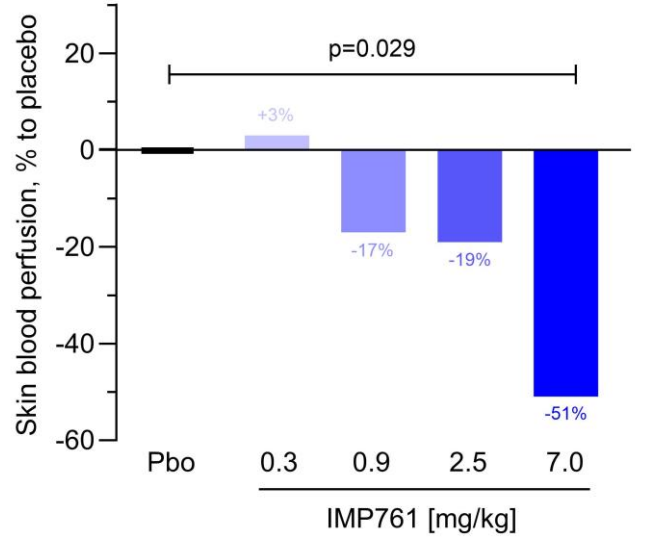
- Robust, statistically significant pharmacodynamic activity in a validated, placebo-controlled, double-blind clinical setting
- **Effect size >50%** in both a clinical endpoint (skin blood perfusion, −51%) and a scientific endpoint (T-cell count, −53%) at 7.0 mg/kg
- **Dose-dependent suppression (EULAR):** IMP761 reduced antigen specific T-cell driven immune response in a dose-dependent manner
- **Exposure-dependent suppression (ACR):** higher IMP761 blood levels, stronger inhibition: presence and concentration matter

Clinical Endpoint: Skin Blood Perfusion

How we measured



Laser Speckle Contrast Imaging (LSCI)



Method: Blood perfusion was analysed on Day 9, 24 hours post KLH injection, by Laser Speckle Contrast Imaging (LSCI)

Result: Significant reduction in skin blood perfusion compared to placebo at 7 mg/kg (Wilcoxon rank-sum test, p = 0.029)

Reduction observed with doses of IMP761 ≥0.9 mg/kg

Triebel F, Pereira A, Brignone C, Winckels S. IMP761, a novel immunosuppressive LAG-3 agonist antibody in the clinic. Poster POS0773, EULAR Congress, London, UK, 3-6 June 2026.
Data shown are from the ongoing Phase I randomised, placebo-controlled, double-blind first-in-human study (NCT06637865); interim preliminary data.

Path forward for IMP761 in autoimmunity

Phase II in Rheumatoid Arthritis (RA): Strongest Path to Clinical Proof of Concept in Patients

1 Scientific rationale: T-cell-driven, LAG-3 involved

RA is driven by autoreactive T cells. LAG-3 has been shown to be relevant in RA: it is increased in patients and affects functionality[1–5].

2 Our own data: IMP761 is relevant in RA patient samples

RA patient PBMCs, stimulated with a joint-derived peptide pool, ex vivo: e.g.:

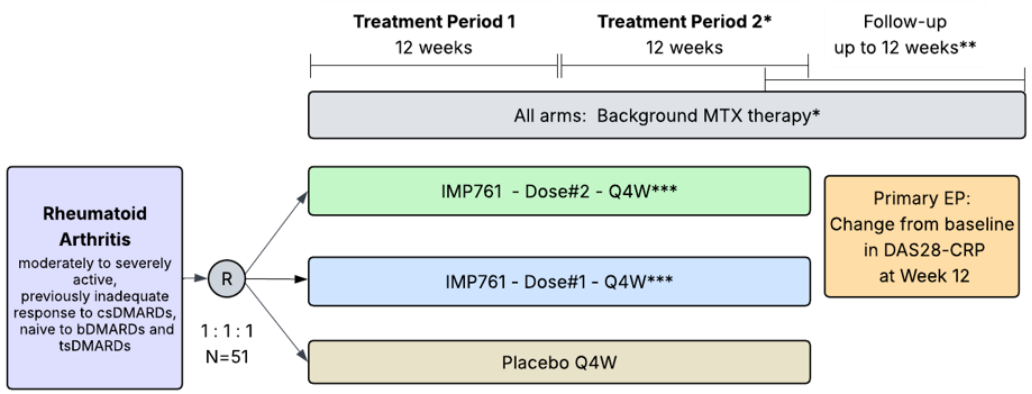
-51.7% CD4⁺ T cell proliferation (p=0.043) vs IgG4 control

Further supported by ex vivo studies in o-JIA samples[5]

3 Independent indication prioritisation and expert input

RA was identified as a lead indication from both a scientific and a clinical development feasibility perspective. *"The clearest mechanistic fit"* with the *"strongest direct IMP761 evidence"*[6]

Strategy*: Show significant clinical activity in a relevant disease setting and select the right dose for late-stage development while preparing for late-stage trials and a partnering opportunity



*Only those participant move to Period 2 who are assessed as having low disease activity per CDAI (score ≤10) at Week 12 as assessed by the site Safety Assesssor

**Maintenance of previously stable methotrexate regimen; **For up to 12 weeks after last IMP761 or prior to the initiation of any new DMARD in addition to background MTX, whichever occurs first; ***In case of early discontinuation, participants will enter safety follow-up as per above.

Abbreviations: bDMARD: biologic DMARD; CRP: C-reactive protein; csDMARD: conventional synthetic DMARD; DAS28-CRP: Disease Activity Score-28 for Rheumatoid Arthritis with CRP; DMARD: disease-modifying antirheumatic drug; EP: endpoint; MTX: methotrexate; Q4W: every four weeks; tsDMARD: targeted synthetic DMARD

Study design under consideration*	Phase II double-blinded, randomised study with e.g. two dose levels of IMP761
Endpoints/ Milestones	DAS28-CRP at week 12 as primary objective

* Note: Design parameters are preliminary and illustrative only. The final study and statistical design remains subject to ongoing discussion with all stakeholders, health authority feedback and ethics committee review, and may change materially. Nothing here constitutes a commitment.

[1] Pedersen JM et al. Arthritis Res Ther 2023;25:97. [2] Argyriou A et al. Nat Commun 2022;13:4046. [3] Emerson D et al. Front Immunol 2025;16:1534462. [4] Lin WW et al. Front Immunol 2021;12:767868. [5] Sag E et al. Pediatr Res 2021;90:744–751. [6] Syneos Health. IMP761 Indication Prioritisation.

FY26 financial summary

- Cash position of approx. **A$48.5M** as at 30 September 2026 (including term deposits)
- **Total revenue and other income increased to A$24.2M in FY26** from A$10.3M in FY25, mainly due to the Dr Reddy's upfront payment
- **Research and development and intellectual property expenses increased to A$91.9M in FY26** mainly due to increased clinical trial activity and associated expenses
- **Clinical trial costs and TACTI-004 close-out activities** contributed to higher R&D expenditure and net loss in FY26
- **Disciplined cash management and cost reduction initiatives** implemented to preserve capital and extend cash runway
- Received **non-dilutive funding support** through French and German R&D incentive programs
- Cash resources expected to **support operations well into the first half of CY2028**

	FY26	FY25
Revenue and other income	A$24.2M	A$10.3M
G&A Expenses	A$9.6M	A$8.6M
R&D and IP expenses	A$91.9M	A$61.4M
Net loss	A$79.3M	A$61.4M
Net operating cash outflow	A$55.5M	A$62.0M

Immutep: substantial assets and multiple paths to value

An established LAG-3 platform with differentiated medicines, protected IP, partners and financial resources

CORE ASSET 01

Efti | Oncology

First-in-class MHC II agonist; HNSCC CPS <1 and resectable STS priorities. FDA Fast Track / Orphan designations.

CORE ASSET 02

IMP761 | Autoimmunity

First-in-class LAG-3 agonist; human proof-of-pharmacology in Phase I. Preparing for patient proof-of-concept.

ADDITIONAL ASSETS

IMP731 + IMP701 IP

IMP731 depleting antibody & IMP701-related patent rights.

PARTNER NETWORK

Industry + academia

Dr Reddy's, MSD, EOC Pharma and leading clinical centres; partnerships support reach, development and credibility.

INTELLECTUAL CAPITAL

Experienced global team

LAG-3 discovery and development expertise across science, CMC, clinical trials and regulatory execution.

EXTERNAL RECOGNITION

Data on global stages

ESMO, ASCO, EULAR, ACR, CTOS and SABCS presentations; international investigators and scientific collaborations.

A$48.5M CASH at 30 September 2026 | FY26: 19 new patents | runway expected well into H1 CY2028*

* Cash includes term deposits; runway is forecast and depends on cost reductions and assumptions. Other programmes and licences are at different stages and do not imply near-term revenue.

2027 milestones and catalysts

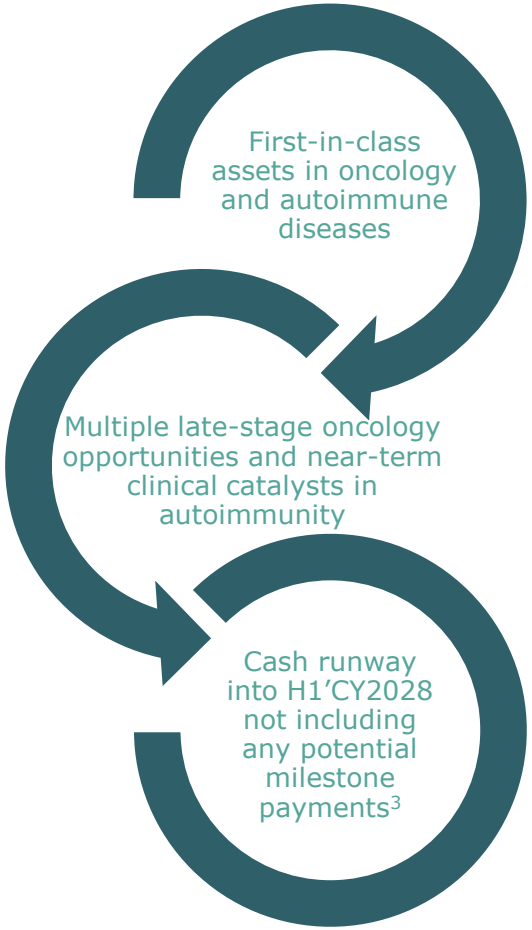
Expected milestones grouped by program and half-year

	H1 CY2027	H2 CY2027
Efti **Focused oncology development**	**Regulatory and development readiness** • New data from efti (EFTISARC-NEO) • Regulatory interactions & preparations • Finalise HNSCC and STS strategy, protocols and potential partner arrangements • Completion of 200 L manufacturing run • Business Development interactions	**Potential study initiation and execution** • Operational preparations • HNSCC CPS <1: planned Phase II initiation • STS: planned adaptive Phase II/III initiation • Continued manufacturing and operational execution
IMP761 **Targeted immune modulation in autoimmunity**	**Clinical data and Phase II preparation** • Phase I multiple-ascending-dose results incl. PK data • Dose selection and finalisation of the Phase II design • Regulatory, CRO, manufacturing and clinical-supply preparation/execution • Business Development interactions	**Potential transition into Phase II** • Potential initiation of Phase II in e.g. rheumatoid arthritis • Clinical material available at sites • Study start subject to available resources, regulatory and operational readiness

Advance value-inflecting milestones while preserving strategic flexibility across two priority assets

Timing, scope and sequencing are indicative and may change. All milestones are subject to regulatory feedback, clinical data, partner decisions, manufacturing readiness, available financial and organisational resources, and other execution risks.

Summary



- **Immutep is developing immunotherapies with opportunities in both oncology & autoimmune diseases**
- **Efti is the only immunotherapy that activates the immune system via the MHC Class II pathway and is designed to work differently from current immunotherapies**
  - ✓ Studies commenced before TACTI-004 suggested that efti may expand responders and enhance efficacy when combined with IO (e.g. KEYTRUDA), chemo or radiotherapy. Safety profile plus easy subcutaneous delivery are key attributes in current & future combination therapy dominated treatment landscape
  - ✓ Clinical data support continued development in HNSCC CPS <1 and neoadjuvant treatment of STS, with FDA Fast Track or Orphan Drug designations providing important regulatory momentum. Study start targeted for H2 CY2027; TACTI-004 root cause analysis update presented at this AGM (27 November 2026)
  - ✓ Established partnerships incl. licensing agreement with Dr Reddy's (Immutep is also entitled to potential regulatory, development and commercial milestone payments of up to US$349.5M; royalties on net commercial sales in the licensed territories; and retains the global manufacturing rights to efti)
- **IMP761, the world's first LAG-3 agonist antibody, is a new approach to autoimmune disease, currently in early human testing**
  - ✓ Phase I results show favourable safety and pharmacodynamic activity in healthy volunteers;
  - ✓ Top line clinical results presented at EULAR Congress, 4 June 2026
  - ✓ Late-breaking Phase I poster at ACR Convergence 2026, 10 November 2026
- **Multiple collaborations with large pharma and academia globally**
- **Strong IP portfolio; 12+ years of potential exclusivity for efti/IMP761 as biologics[1]**
- **Cash, cash equivalents and term deposit balance of ~A$48.5M provide runway well into H1 CY2028 based on current assumptions, not including any potential milestone payments[2]**

1. Biologics Price Competition and Innovation Act (BPCIA). 2. Cash, cash equivalent and term deposit position of A$48.5M as at 30 September 2026; excludes €1.36M (~A$2.16M) French R&D tax incentive (CIR) received 28 October 2026. Cost-reduction measures are largely completed to preserve runway.

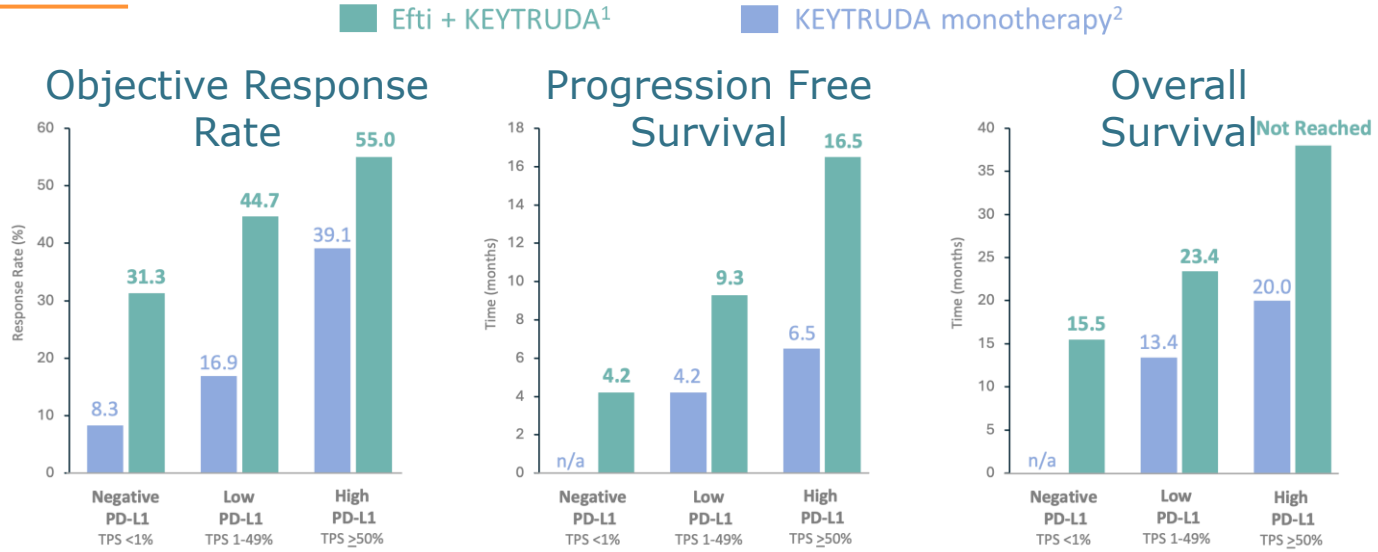

Thank You
immutep
Empowering the Immune System

Appendix

immutep
Empowering the Immune System

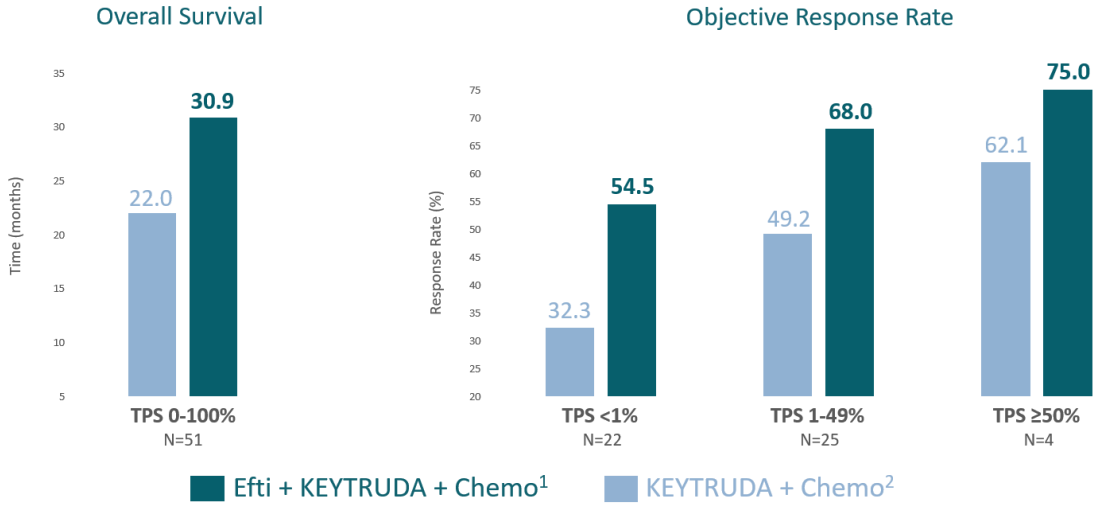
Previous Data With Efti in NSCLC

TACTI-002: Benchmarking Efti + KEYTRUDA to KEYTRUDA Alone



Compelling efficacy from efti + KEYTRUDA *across all PD-L1 levels*, including 1L NSCLC patients with negative & low PD-L1 (TPS <50%), for whom KEYTRUDA monotherapy works sub-optimally*

INSIGHT-003: Benchmarking Efti + KEYTRUDA + Chemo[1] in non-squamous 1L NSCLC to KEYTRUDA + Chemo[2]



Encouraging ORR, PFS and OS with chemo in NSQ across PD-L1 level

Conclusion: Two independent studies with 150+ patients in 15+ sites across 10+ countries showed increase of all relevant efficacy parameters like ORR, PFS, OS compared to historical control

Comparison of data is from different clinical trials. 1. Data from TACTI-002. 2. KEYTRUDA data for TPS 1-49% & TPS >=50% from publications/EPAR assessment of KN-042, and for TPS <1% from publications of KN-001 that had limited data for PFS/OS in 1L NSCLC group due to size (N=12): (a) Hui et al., Pembrolizumab as first-line therapy for patients with PD-L1-positive advanced non-small cell lung cancer: a phase 1 trial. Ann Oncol. 2017 Apr 1;28(4):874-881,and (b) Garon et al., Five-Year Overall Survival for Patients With Advanced Non–Small-Cell Lung Cancer Treated With Pembrolizumab: Results From the Phase I KEYNOTE-001 Study, JCO 2019: https://doi.org/10.1200/JCO.19.00934. 3. EMA/233126/2020, EMEA/H/C/003820/II/0057 Questions and answers on the use of Keytruda alone in non-small cell lung cancer with low levels of PD-L1; 30 April 2020

TACTI-004 futility outcome

Futility Analysis March 2026

Table 1: ORR / DCR by RECIST 1.1 overall

Response Parameter	TACTI-004 (N=173)*	
	Efti+SoC N=84	Placebo+SoC N=89
ORR unconfirmed	42.9%	55.1%
DCR	76.2%	87.6%

Table 2: ORR by RECIST 1.1 & tumor histology

TACTI-004 (N=173)* ORR unconfirmed by histology	Treatment group	NSQ N = 98	SQ N= 75
	Efti (N=84)	45 %	41 %
	Placebo (N=89)	55 %	55 %

Table 3: ORR by RECIST 1.1 & PD-L1 subgroup

TACTI-004 (N=173)* ORR unconfirmed by PD-L1	Treatment group	TPS < 1 %	TPS 1-49 %	TPS >= 50 %
	Efti (N=84)	35 %	39 %	54 %
	Placebo (N=89)	35 %	49 %	76 %

Key findings:

- Inferior effects also seen on PFS/OS albeit very early and not mature at the time of analysis

Conclusion:

- By the time of the analysis the efficacy endpoints were all suggesting futility of efti compared to placebo → study was terminated early for futility
- A gap between efti and placebo arm was also confirmed at final analysis

TPS = PD-L1 score; SQ – squamous; NSQ – non-squamous

* extended analyses done prior to trial termination; official TLFs, EVAL2 futility, data cut-off Jan 2026

Baseline and Disease Characteristics – Futility population

Baseline parameters	N=173	
	Efti+SoC, N=84	Placebo+SoC, N=89
Histology, % Squamous Non-squamous	 44.0 56.0	 43.8 56.2
Median age, years (range)	65 (47-83)	67 (39-81)
Female, % Male, %	28.6 71.4	23.6 76.4
Disease stage at study entry, % IIB IIC IVA IVB	 9.5 6.0 48.8 35.7	 12.4 6.7 43.8 37.1
ECOG PS, % 0 1	 35.7 64.3	 41.6 58.4
PD-L1 (TPS), % <1% 1-49% ≥50%	 26.2 38.1 35.7	 24.7 38.2 37.1
Smoking status, % Current / Ex / Never / Unknown	36.9 / 50.0 / 13.1 / 0	29.2 / 59.6 / 11.2 / 0
Tumor location (Liver / Central nervous system)	8.3 / 6.0	16.9 / 10.1
Mutations (kRAS+ / TMB median)	13.1 / 9.4	21.3 / 7.7

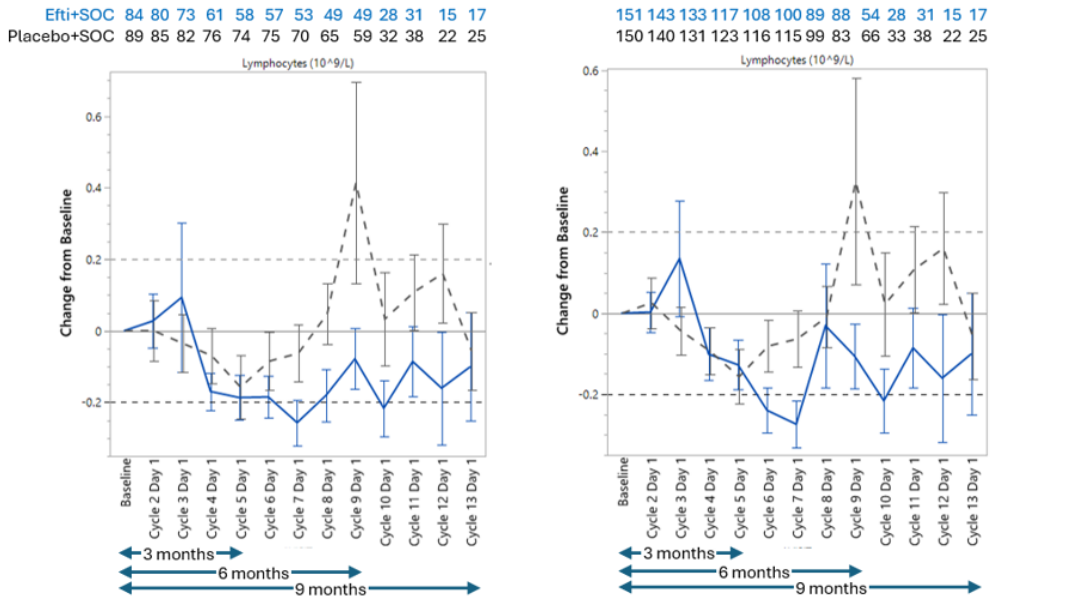
Source: TACTI-004 final analysis, data cut 07Aug2026, T14.1.2.2 and T14.1.3.1.2.

TACTI-004: unexpected immune-cell patterns

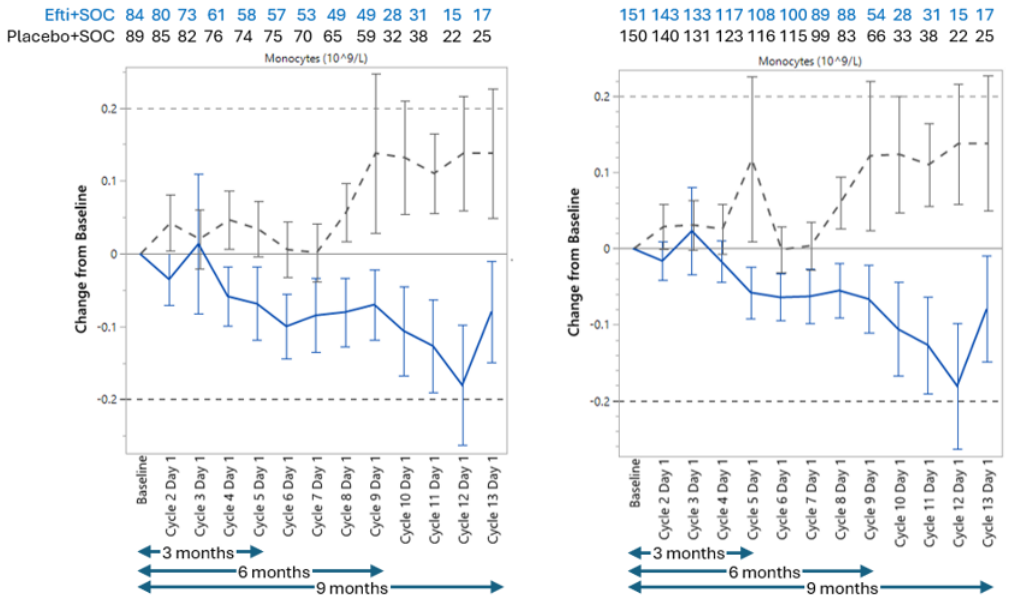
Lymphocyte and monocyte observations

- Lymphocyte and monocyte counts fell unexpectedly in the efti arm
- The pattern held with longer follow-up and differed from earlier studies

Absolute Lymphocyte Count (ALC: futility set 173 and extended set 301)



Monocyte Count (futility set 173 and extended set 301)

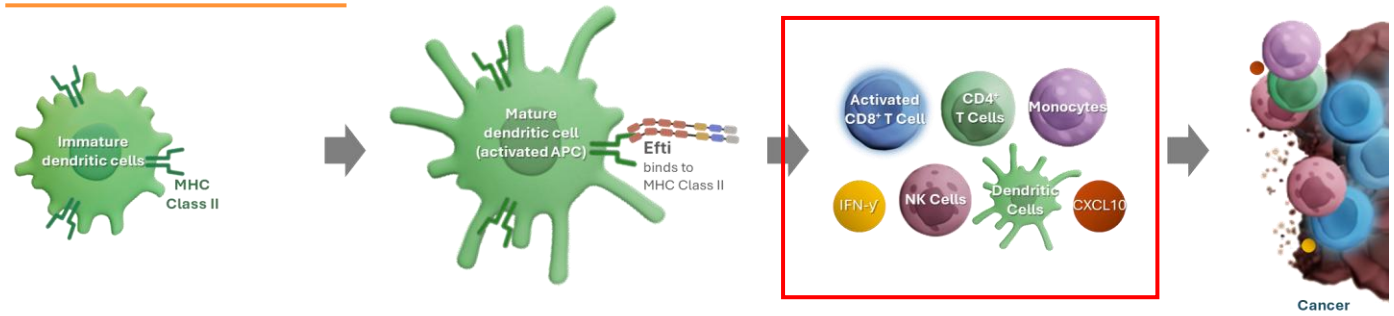


<u>Conclusion:</u> The atypical hematological profile observed in TACTI-004 is compatible with altered immune modulation by efti

Potential contribution to futility: Possible

TACTI-004 Futility RCA: PD marker observations deviate from previous results

Previous Data With Efti in Multiple Oncology Indications

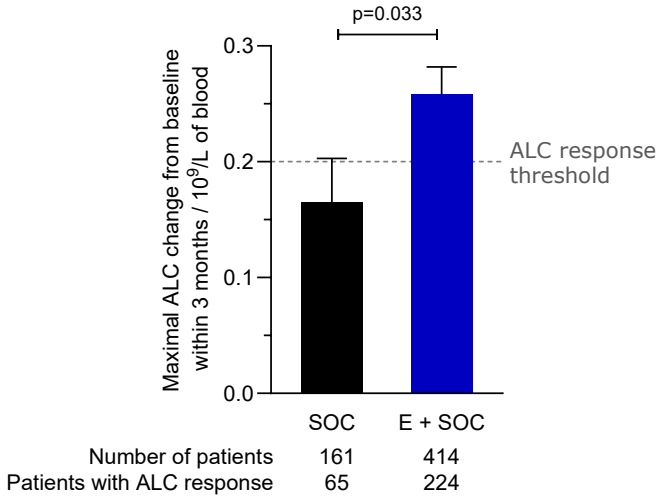


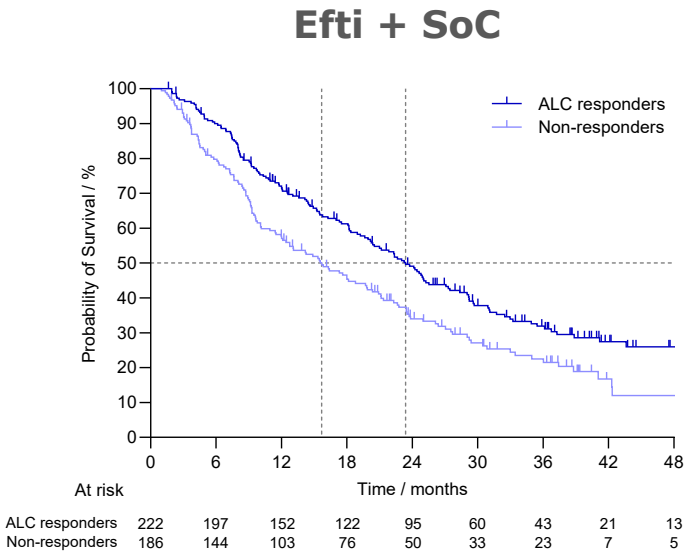
The addition of efti to chemotherapy and/or anti-PD-1 therapy has been observed to lead to an increase of lymphocyte counts in the peripheral blood in several cancer types. This increase has also been linked to improved overall survival for efti-treated patients.

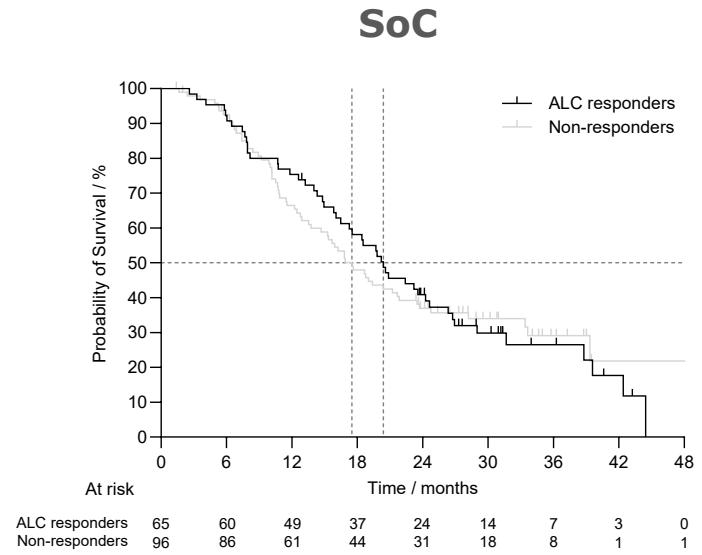
Significant increase in Absolute Lymphocyte Counts (ALC) with E + SoC but not SoC alone[1]

Significantly prolonged Overall Survival in ALC responders* treated with E + SoC but not SoC alone[1]

- Median OS 23.4 vs. 15.7 mo; HR=0.69; p=0.0017 for Efti + SoC
- Median OS 20.4 vs. 17.5 mo; HR=0.98; p=0.93 for SoC alone



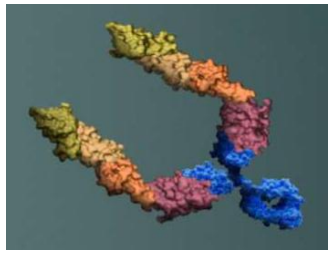




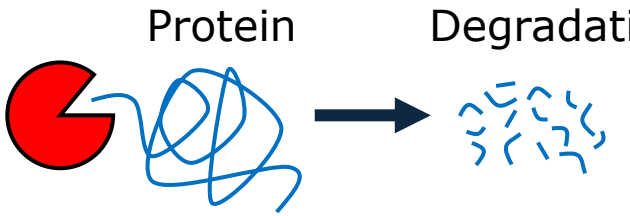
(1) ASCO 2026 Poster; Data pooled from five clinical studies (TACTI-mel, TACTI-002, TACTI-003, AIPAC, AIPAC-003)
(*) ALC response was pre-defined as a change of ≥0.2 x 109/L within 3 months on study (up to dose 7).

Biologic Role of Glycans (sugars) attached to Protein

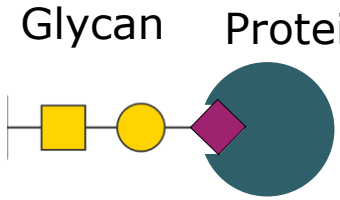
- Stabilize Protein 3D Structure



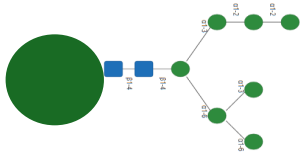
- Protect Protein from degradation



- Binding sites for other proteins (e.g. immune response)



- Govern protein distribution & half-life inside the body



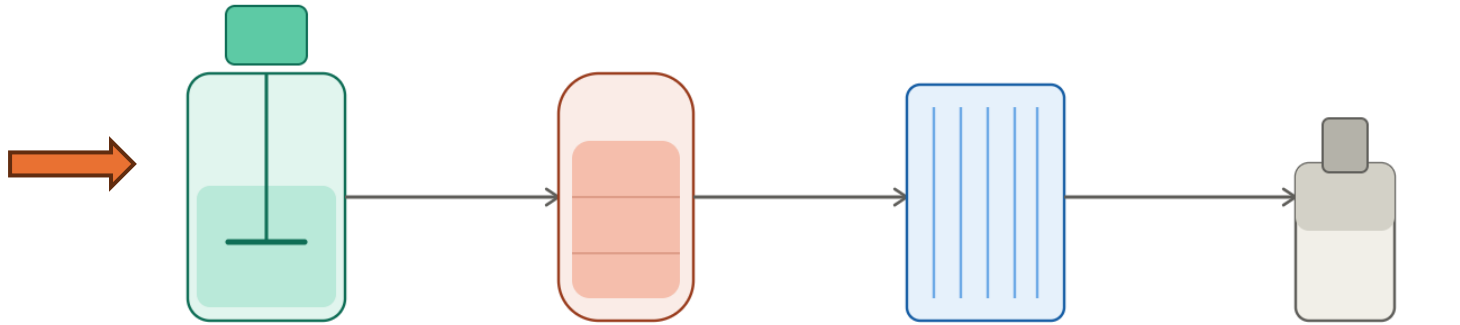
Uncapped glycans → short time in circulation



Capped glycans → long time in circulation

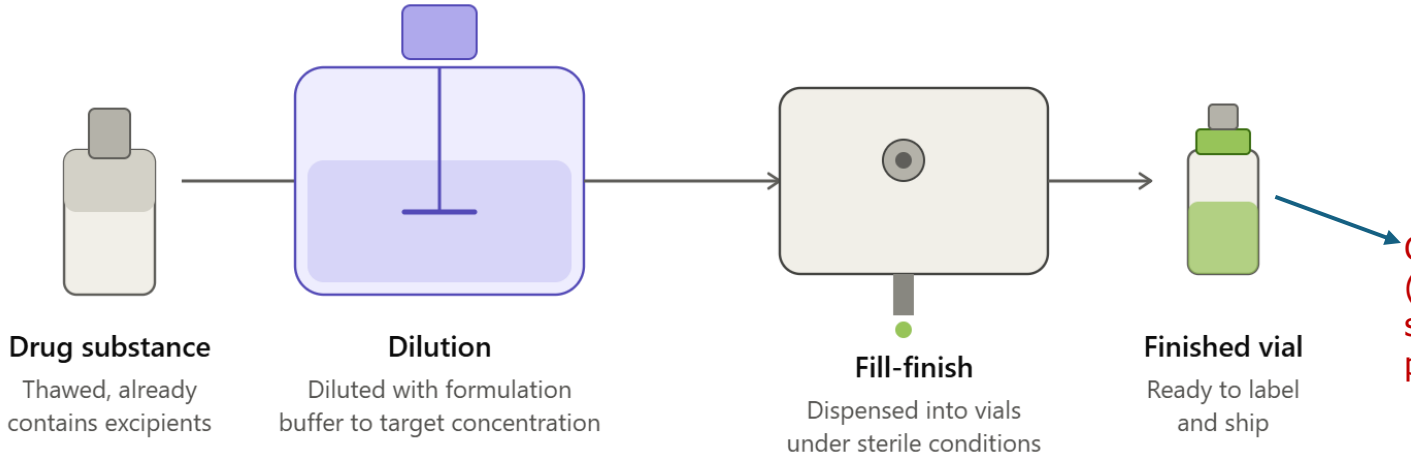
Bioreactor conditions and scale influence drug substance and drug product

Drug Substance process (Active pharmaceutical ingredient [efti])



Bioreactor **conditions and scale** impact protein quality such as attached sugars, charge, 3D structure, product and cell related impurities

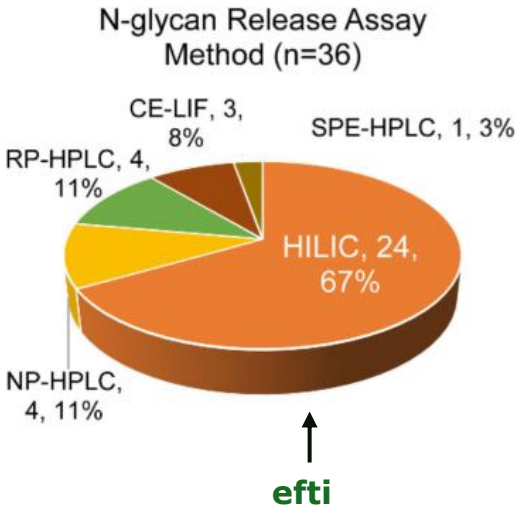
Drug Product process (Product for clinical administration)



Contains active ingredient (efti), buffer and surfactant (PS80) for product stability

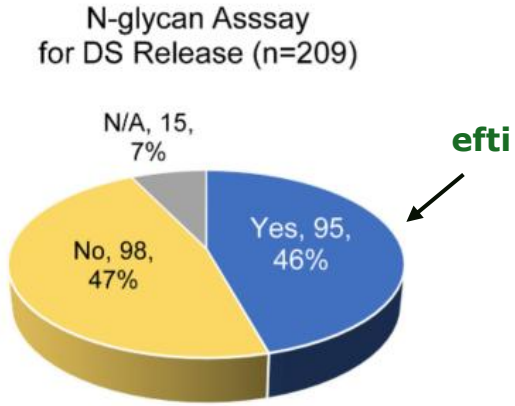
Efti GMP release test for N-glycan analysis on par with industry standards

***Evidence from release testing data of 209 FDA-approved therapeutic antibody products**



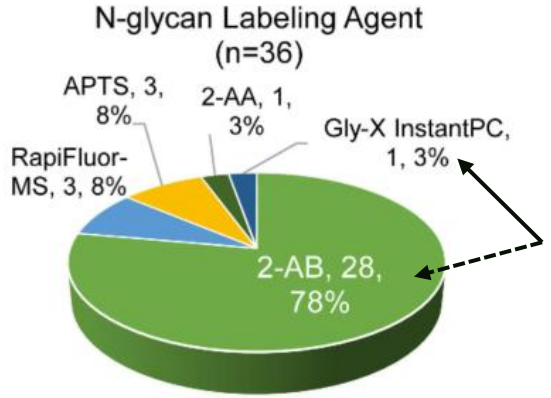
efti

92% of N-glycan release assays are HPLC/HILIC-based, same as efti



efti

46% use an N-glycan assay for DS release testing, same as efti



efti

78% of N-glycan assays use 2-AB dye and 3% Gly-X InstantPC dye, same as efti (2-AB was initially used, but-Gly-X InstantPC was later implemented to improve sensitivity)

Detailed structural characterization of N- and O-linked glycans requires extensive mass spectrometry analysis and is mainly used for extended product characterization, not routine release of mAbs and fusion proteins like efti.

*Source: Luo S. et al., Glycan profiles of FDA-approved therapeutic antibodies, AAPS Journal (2026), Figure 6; efti method per IMPD.

Scope note: 6a n=209 products; 6b n=41 (Fc effector function proposed as MOA); 6c–d n=36 (Fc effector function and glycan assay in DS release).