Translating tRNA Synthetase Biology into New Therapies for Inflammation and Fibrosis

April 2026

Forward Looking Statements

The following slides and any accompanying oral presentation contain forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 and other federal securities laws. The use of words such as "may," "might," "will," "should," "expect," "plan," "anticipate," "believe," "estimate," "project," "intend," "future," "potential," "opportunity," or "continue," and other similar expressions are intended to identify forward-looking statements. For example, all statements regarding: the potential therapeutic benefits of proteins derived from tRNA synthetase genes and our product candidates and development programs; the ability to successfully advance our product candidates and undertake certain development activities (such as the initiation of clinical trials, clinical trial enrollment, the conduct of clinical trials and announcement of clinical results) and accomplish certain development goals, and the timing of such events; the potential market opportunity for our product candidates; our ability to receive regulatory approvals for, and commercialize, our product candidates; our ability to identify and discover additional product candidates; potential activities and payments under collaboration agreements; and the ability of our intellectual property portfolio to provide protection are forward-looking statements. All forward-looking statements are based on estimates and assumptions by our management that, although we believe to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that we expected. These risks, uncertainties and other factors are more fully described in our filings with the U.S. Securities and Exchange Commission, including our Annual Report on Form 10-K, our subsequently filed Quarterly Reports on Form 10-Q, and in our other filings. The forward-looking statements in this presentation speak only as of the date of this presentation and neither we nor any other person assume responsibility for the accuracy and completeness of any forward-looking statement. We undertake no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.

We own various U.S. federal trademark applications and unregistered trademarks, including our company name. All other trademarks or trade names referred to in this presentation are the property of their respective owners. Solely for convenience, the trademarks and trade names in this presentation are referred to without the symbols ® and ™, but such references should not be construed as any indicator that their respective owners will not assert, to the fullest extent under applicable law, their rights thereto.

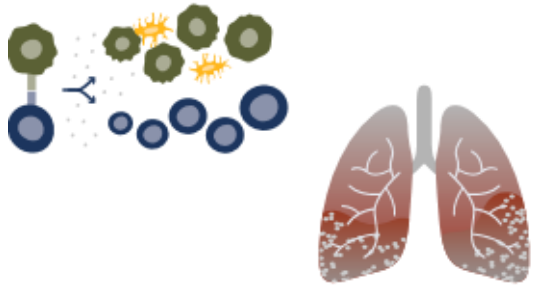
This presentation discusses product candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the uses for which they are being studied. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involved a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Translating tRNA Synthetases into New Therapies for Fibrosis and Inflammation

Proprietary tRNA synthetase platform

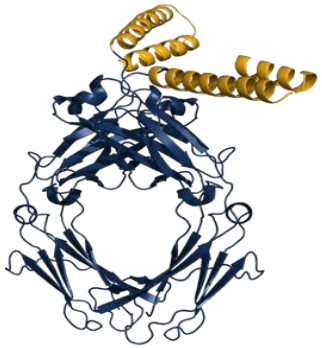
- Novel extracellular functions gained through evolutionary intelligence
- Potential new class of medicines
- IP directed to protein compositions from all 20 tRNA synthetase genes

Therapeutic focus: inflammation and fibrosis



- Untapped therapeutic potential
- Differentiated approach
- Multiple disease opportunities

Efzofitimod: novel biologic immunomodulator for ILD



- Topline results reported for Phase 3 EFZO-FIT™ study in pulmonary sarcoidosis; U.S. FDA Type C meeting scheduled for mid-April 2026
- Phase 2 EFZO-CONNECT™ study in SSc-ILD expected to complete enrollment in 1H26
- Opportunity in other ILD

~$80.9m in cash, restricted cash, cash equivalents and investments as of December 31, 2025

Efzofitimod Leads Growing Pipeline of tRNA Synthetase Derived Biologics

PROGRAM	tRNA SYNTHETASE	TARGET/MOA	INDICATION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3
Efzofitimod	HARS	NRP2 modulator	Pulmonary Sarcoidosis(1)	efzo-fit			
			SSc-ILD	efzo-connect			
			Other ILD (CTD-ILD; CHP)				
ATYR0101	DARS	LTBP1 modulator	Fibrosis				
ATYR0750	AARS	FGFR4 modulator	Liver Disorders				
tRNA Synthetase Candidates(2)							

Topline Results Reported in Q3 2025

Kyorin

Japan Partner

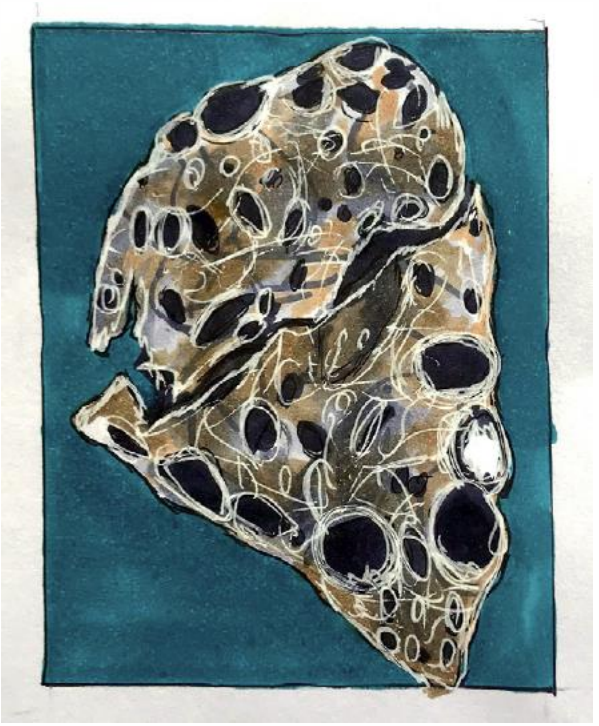
(1) In partnership with Kyorin Pharmaceutical Co., Ltd. for the development and commercialization of efzofitimod for ILD in Japan
(2) Pipeline candidates in development based on additional tRNA synthetases from IP portfolio
SSc-ILD = Scleroderma-related ILD; CTD-ILD = Connective Tissue Disease-ILD; CHP = Chronic Hypersensitivity Pneumonitis

Efzofitimod

A New Approach to Interstitial Lung Disease

A New Approach to Interstitial Lung Disease (ILD)

- ILD are a group of **severe inflammatory and fibrotic lung diseases**
- Persistent inflammation leads to **worsening lung function, fibrosis** and **poor quality of life (QoL)**
- Progressive fibrosis can result in a **survival rate that is worse than many common cancers**
- Current therapeutic options are **toxic** and **not disease modifying**

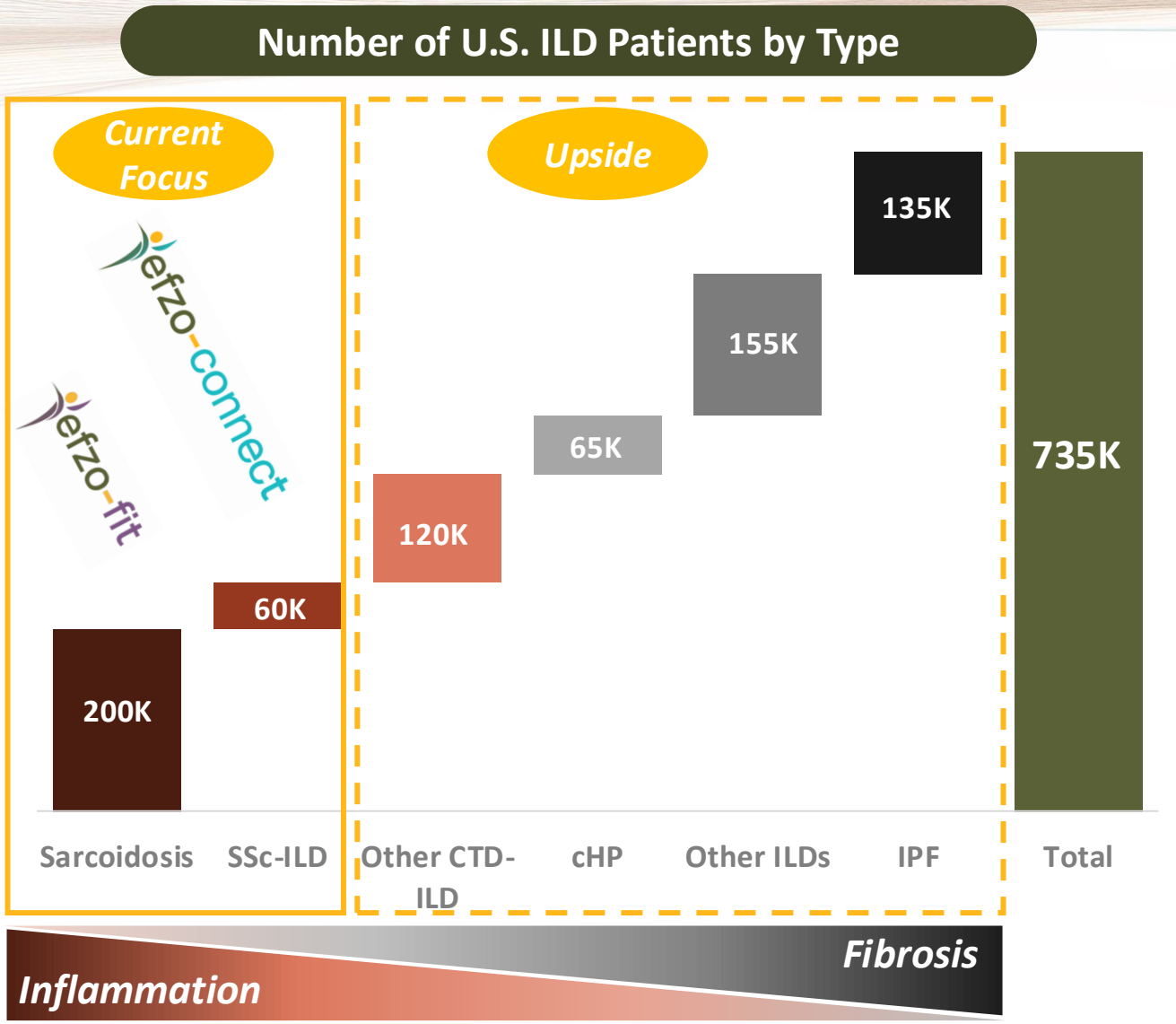


End stage fibrotic lung*

Efzofitimod is a biologic immunomodulator with a novel mechanism of action in development to address the significant unmet medical need in ILD

*Schimmel et al. The Endless Frontier of tRNA Synthetases. *The Enzymes.* 2020

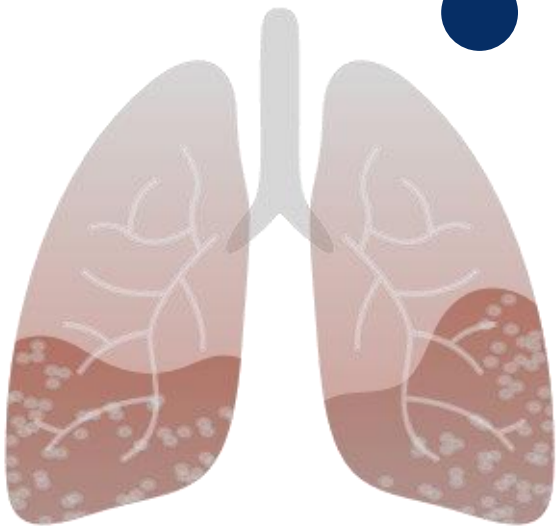
aTyr is Advancing Efzofitimod as the Standard-of-Care for ILD



- ILD is an umbrella term for >200 types of rare lung diseases that span a spectrum of inflammation and fibrosis
- Patients can have poor quality of life with high morbidity and mortality
- No disease-modifying therapies available; current options have significant toxicities
- aTyr's current focus estimated at $2-5b global market opportunity(1)
- Upside potential in other ILD and related autoimmune diseases (e.g., SSc, lupus, RA)

(1) Based on aTyr internal estimates, Back Bay Life Science Advisors market research and 2024 third party claims data analysis

Efzofitimod: Novel Biologic Immunomodulator for ILD

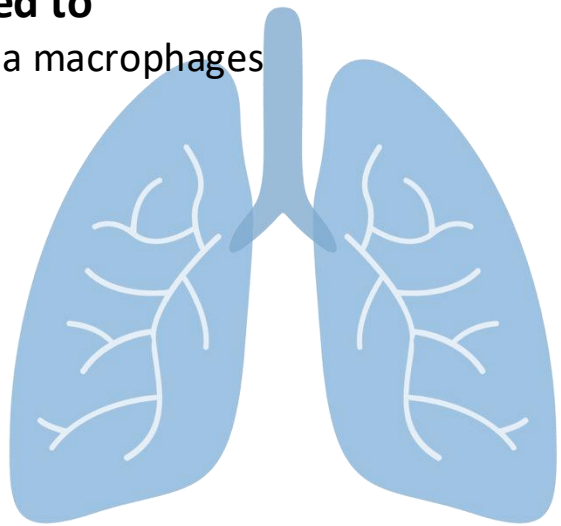


Targets innate immunity at site of inflammation, designed to

- Downregulate pro-inflammatory and pro-fibrotic pathways via macrophages
- Address complex immune pathology
- Restore immune balance without evidence of suppression

Potential benefits

- Resolve symptoms and improve QOL
- Preserve lung function
- Reduce or eliminate OCS



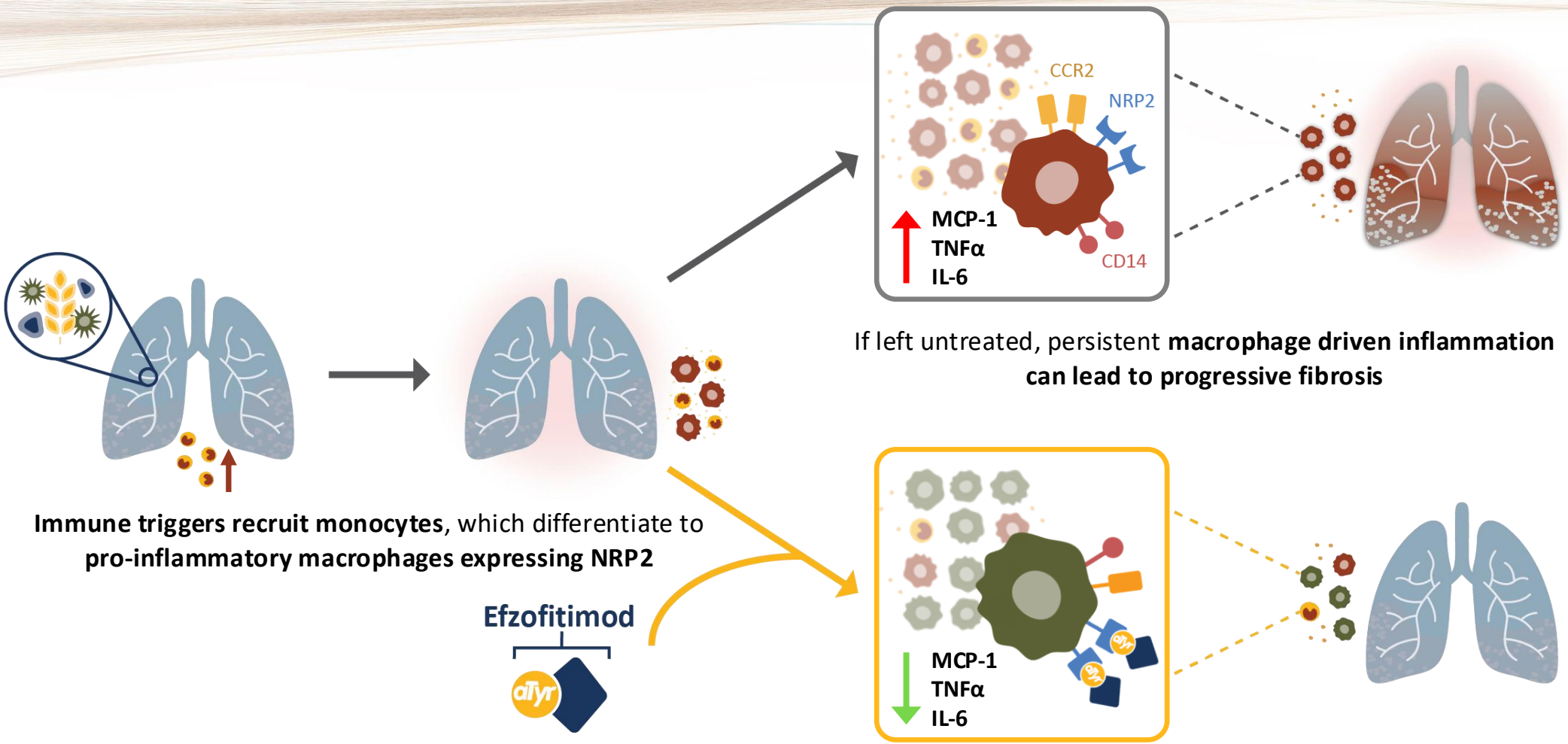
Well tolerated profile

- Acts locally avoiding general immune suppression
- Current SOC associated with debilitating side effects when used chronically

Predicted U.S. commercial exclusivity into 2039 based on composition of matter patents, with expected patent term extension and regulatory exclusivity programs

QOL = quality of life; OCS = oral corticosteroids; SOC = standard of care

Efzofitimod Modulates Macrophages to Reduce Key Drivers of Inflammation & Fibrosis



If left untreated, persistent **macrophage driven inflammation can lead to progressive fibrosis**

Immune triggers recruit monocytes, which differentiate to **pro-inflammatory macrophages expressing NRP2**

Efzofitimod acts as an **NRP2 agonist**

Efzofitimod promotes a less inflammatory macrophage population by **downregulating key drivers of inflammation and fibrosis**

Nangle et al. *Science Translational Medicine* 2025

Pulmonary Sarcoidosis

A Major Form of Interstitial Lung Disease with High Unmet Medical Need

Sarcoidosis is an Orphan Lung Disease with High Unmet Medical Need

Disease Pathology

- Inflammatory disease of unknown cause
- Characterized by granulomas, or clumps of immune cells
- Can affect almost any organ; 90% of cases affect the lungs

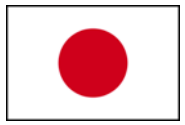
Diagnosis

1) Compatible clinical presentation
2) Non-necrotizing granulomatous inflammation
3) Exclusion of alternative causes

Epidemiology

200,000 pts

150,000 pts



20,000 pts

>1 million pts worldwide

age of onset between **30-50**

twice as common in women

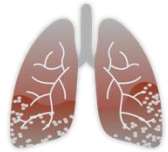
3x

as common in African Americans

Prognosis

70%

of patients need treatment within first 3 years



20%

of patients will develop lung fibrosis

- 1/12 patients hospitalized for their disease annually
- Mortality rising: 1/5 Medicare patients die every 3 years – 60% higher risk than general population
- Fibrosis and concomitant pulmonary hypertension biggest predictors of mortality

U.S. epidemiology and prognosis confirmed by 2024 third party claims data analysis

Sarcoidosis Patients Suffer from Both High Disease & Treatment Burden

Sarcoidosis robs patients of their QoL

Nonlinear journey to diagnosis; patients plagued by shortness of breath, persistent dry cough and severe fatigue

Major socioeconomic impact

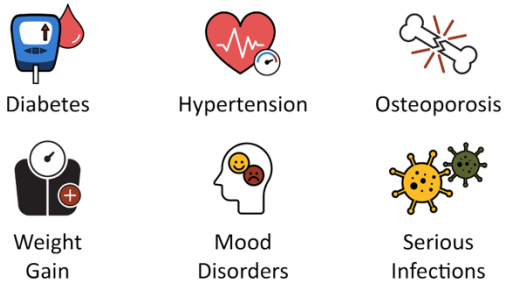
Loss of ability to work, potentially disabling

1/5 patients will progress to fibrosis

Higher risks of pulmonary hypertension, organ failure & death

Corticosteroids remain first line SoC

Short term benefit, but continued use results in debilitating side effects; getting patients off OCS becomes primary goal

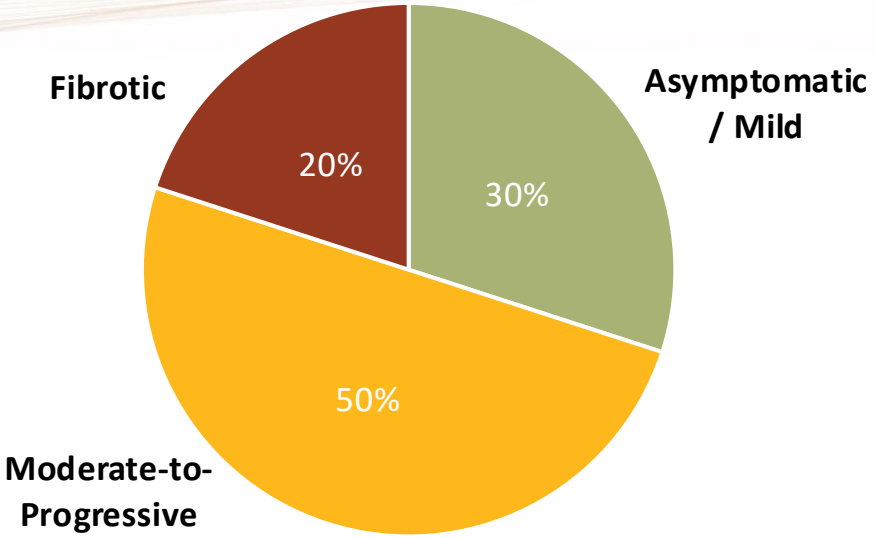


Limited alternatives available

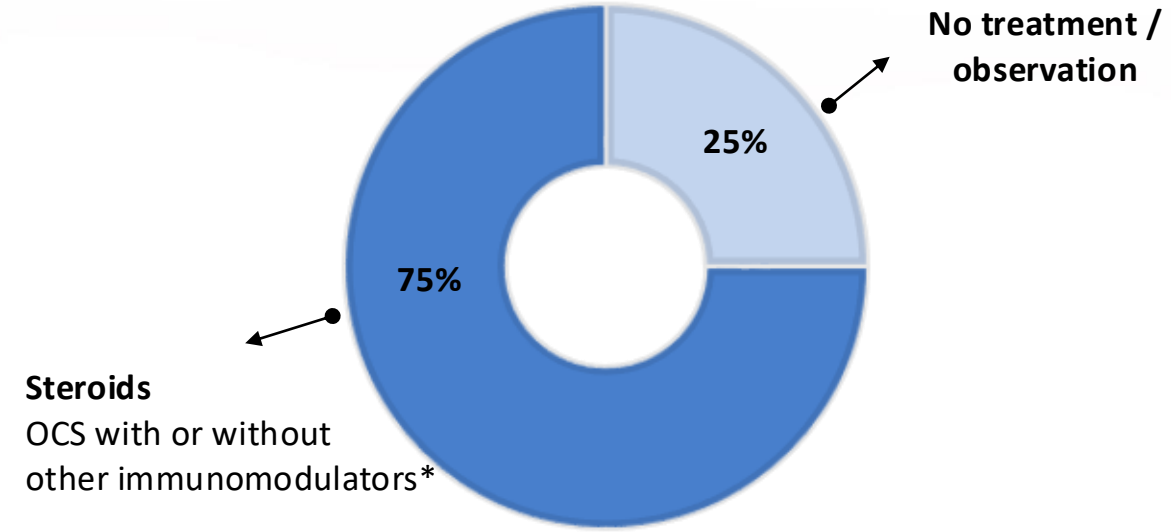
Off-label rheumatology drugs are physicians' only options, with limited evidence in sarcoidosis

Efzofitimod Target Population for Sarcoidosis

Disease Severity



Current Treatment



Efzofitimod Positioning

- Front line as a steroid-sparing agent in moderate-to-severe patients
- Reduce / eliminate steroids and avoid use of cytotoxic immunosuppressants and anti-TNFs
- Addressable population: **50-75% of all sarcoidosis patients**(1)

(1) Based on 2024 third party claims data analysis
OCS = oral corticosteroids; *minimal antifibrotic use

Multiple Benchmarks Support Premium Pricing for New Rare Disease Treatments

$200K*

TAVNEOS (avacopan)

JASCAYD (nerandomilast)

Livdelzi seladelpar

Nucala (mepolizumab)

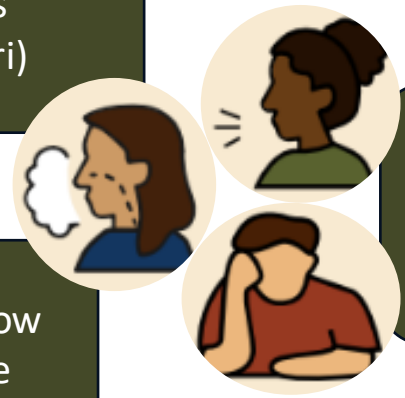
OFEV nintedanib

FILSPARI

ACTEMRA tocilizumab

Steroid-sparing agents for inflammatory disorders (Tavneos, Nucala, Filspari)

ILD treatments that slow lung function decline (Jascayd, Ofev, Actemra)



Rare disease drug launches in recent years (Tavneos, Livdelzi, Filspari)

Efzofitimod is positioned to be the first approved product for sarcoidosis in >70 years with limited competition

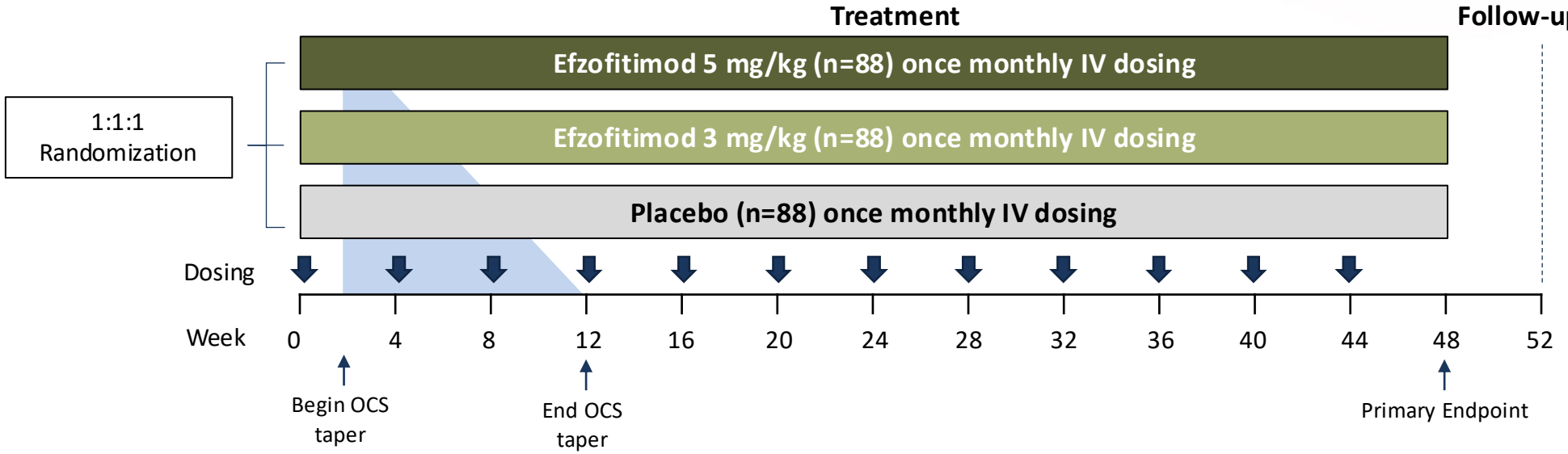
*Estimated annual cost of comparative products based on list price and labeled dosing in the U.S.

Pulmonary Sarcoidosis

Topline Results from Phase 3 EFZO-FIT™ Study

Global Phase 3 Trial in Pulmonary Sarcoidosis

Primary objective: Assess the efficacy of efzofitimod in patients with pulmonary sarcoidosis



N = 268 patients in U.S., Europe, Japan and Brazil

Topline data reported Q325

Population: moderate to severe pulmonary sarcoidosis

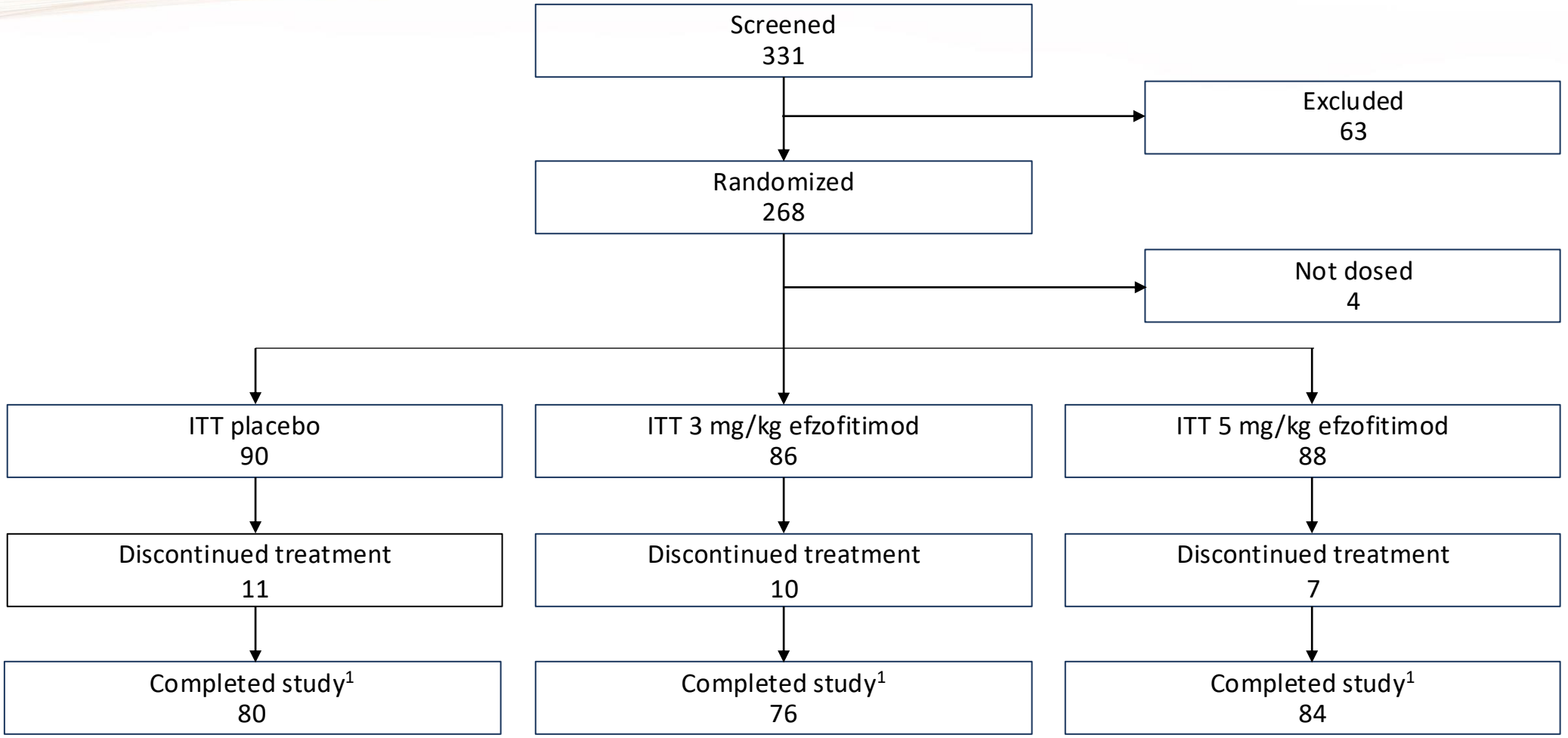
- Diagnosis of pulmonary sarcoidosis for ≥ 6 months
- Stable treatment with ≥ 7.5 and ≤ 25 mg/day OCS
- Extent of fibrosis < 20%
- Symptomatic with KSQ-Lung score ≤ 70

Steroid Taper Protocol Guidelines

- Based on Patients Global Assessment (PGA) **and** Investigator Assessment (IA) conducted every two weeks
- If both PGA **and** IA are stable or improved, patient OCS will need to be **tapered**; If either PGA **or** IA has worsened, patient will be **rescued** with OCS

Individual Patient Expanded Access Program (EAP) is intended to allow access for patients who complete EFZO-FIT™ and wish to receive treatment with efzofitimod outside of the clinical trial

Study Disposition



[1]Completed at least 10 doses and completed follow Up

Baseline Characteristics Balanced

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
Age (Years); mean (SD)	54.1 (10.5)	54.5 (11.9)	52.7 (9.2)
Sex (Male); n (%)	56 (62.2)	47 (54.7)	47 (53.4)
Race			
Black / African American; n (%)	15 (16.7)	12 (14.0)	16 (18.2)
Asian; n (%)	15 (16.7)	11 (12.8)	10 (11.4)
Duration of disease (Years); mean (SD)	8.7 (9.1)	8.7 (8.2)	7.0 (6.3)
Extrapulmonary sarcoidosis; n (%)	29 (32.2)	28 (32.6)	28 (31.8)
KSQ-Lung score; mean (SD)	49.4 (9.1)	53.5 (11.5)	51.6 (10.7)
FVC % predicted; mean (SD)	89.2 (17.4)	86.5 (15.4)	90.7 (17.5)
Pulmonary phenotype; n (%)			
Obstructive / Mixed	38 (42.2)	32 (37.2)	30 (34.1)
Steroid dose (mg/day); mean (SD)	10.7 (4.7)	10.5 (4.0)	10.7 (4.6)
Duration of OCS; mean (SD)	4.6 (5.6)	4.5 (5.6)	4.6 (5.0)
Immunosuppressant; n (%)	34 (37.8)	33 (38.4)	32 (36.4)

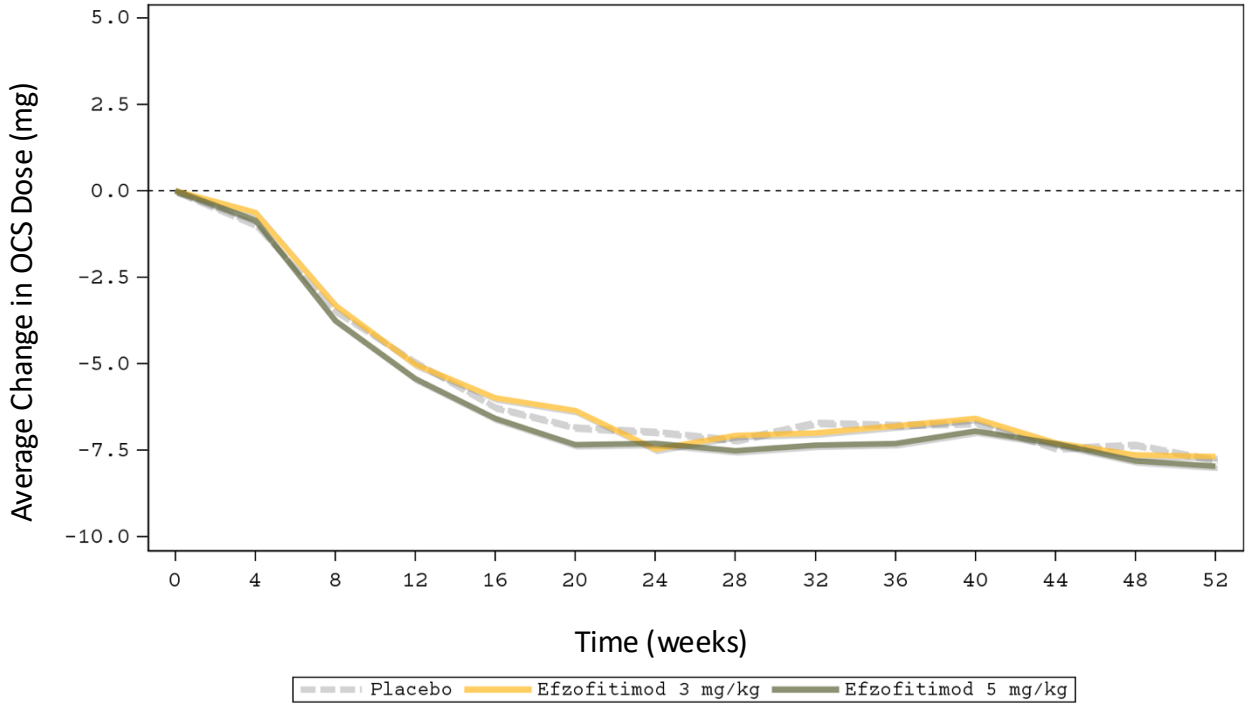
[1]GLI global; rounded to closest first decimal

Primary Endpoint of Steroid Reduction Not Met

OCS Dose at Week 48

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
LS mean dose at week 48 (mg)	3.5	3.5	2.8
LS mean change from baseline (mg)	-7.1	-7.1	-7.9
Difference in LS mean (95% CI)	-	0.0 (-1.5, 1.5)	-0.7 (-2.2, 0.8)
p-value	-	*0.9804*	*0.3313*

Average Change from Baseline in OCS Dose Over Time



OCS = oral corticosteroids

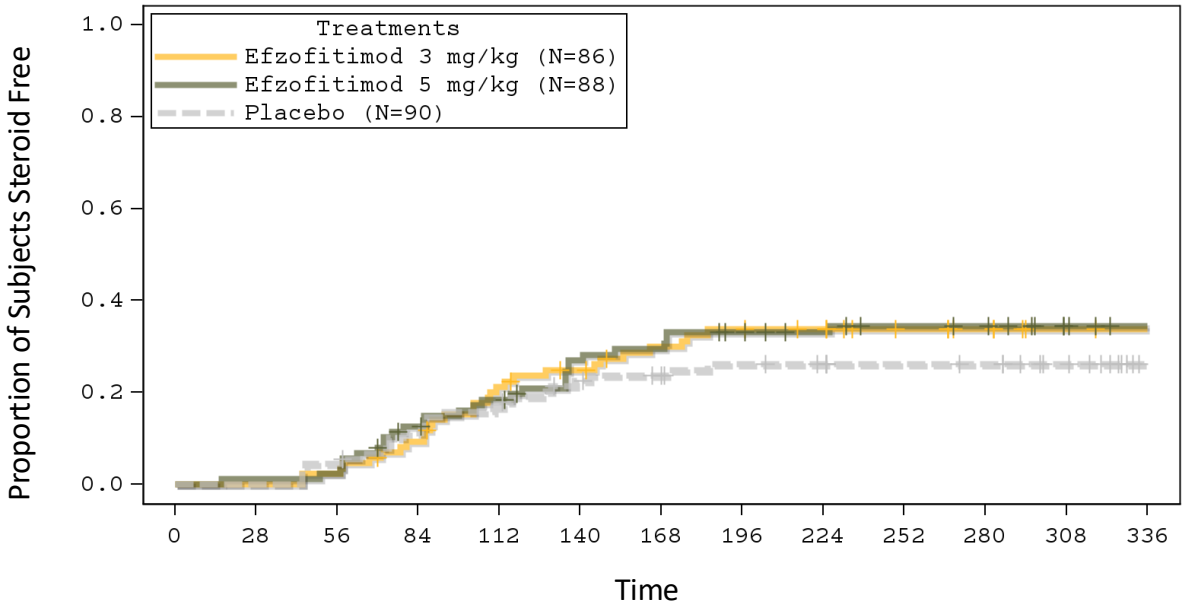
Note: The study's statistical analysis plan was designed on a hierarchal assessment basis. As the primary endpoint did not achieve statistical significance, p-values for other endpoints are reported and should be interpreted as nominal p-values.

Efzofitimod Trends Toward More Prolonged Steroid Withdrawal

Steroid Free at Week 48

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
Steroid free[1]; n (%)	36 (40.2)	45 (51.8)	46 (52.6)
Odds ratio (95% CI)	-	1.6 (0.9, 3.0)	1.7 (0.9, 3.1)
Nominal p-value	-	*0.1172*	*0.0919*

Kaplan Meier Curve of Time to Steroid Free Sustained for 180 Days



[1]Missing patients considered non-responders

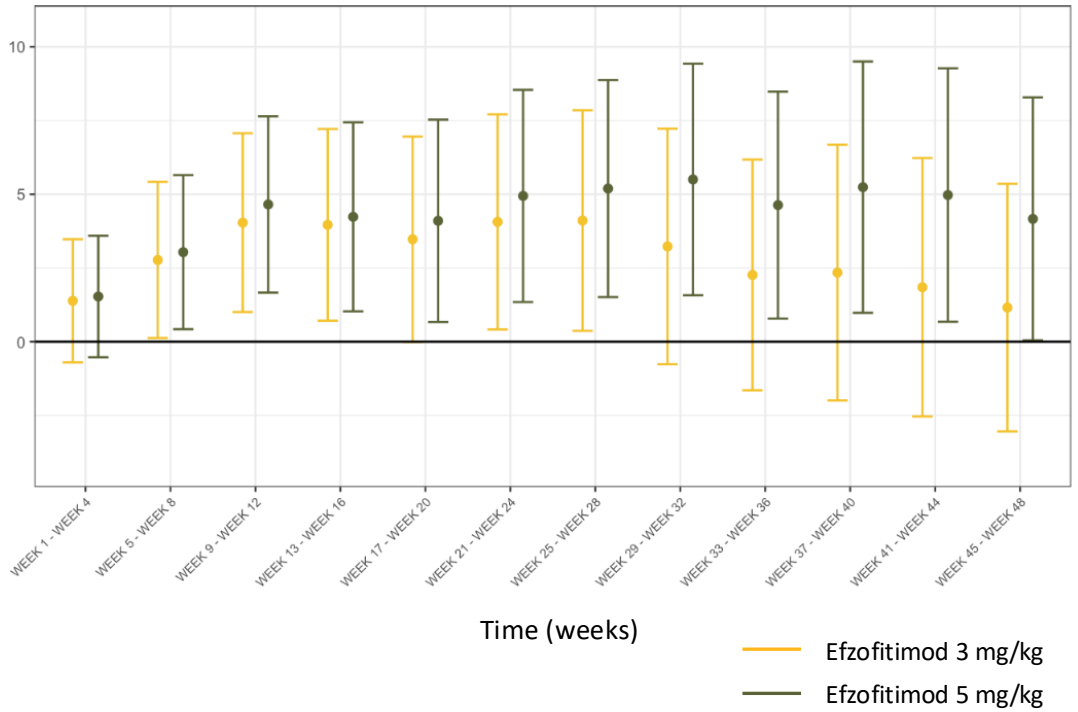
Early and Sustained Benefit for Efzofitimod on King's Sarcoidosis Questionnaire-Lung Score

Change from Baseline in KSQ-L Score at Week 48

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
LS mean change from baseline	6.2	7.3	10.4
Difference; LS mean (95% CI)	-	1.1 (-3.1, 5.4)	4.2 (0.0, 8.3)
Nominal p-value	-	*0.5932*	***0.0479***

Ongoing KSQ-L validation work conducted by aTyr supports a MCID for improvement between groups of 2.1 points

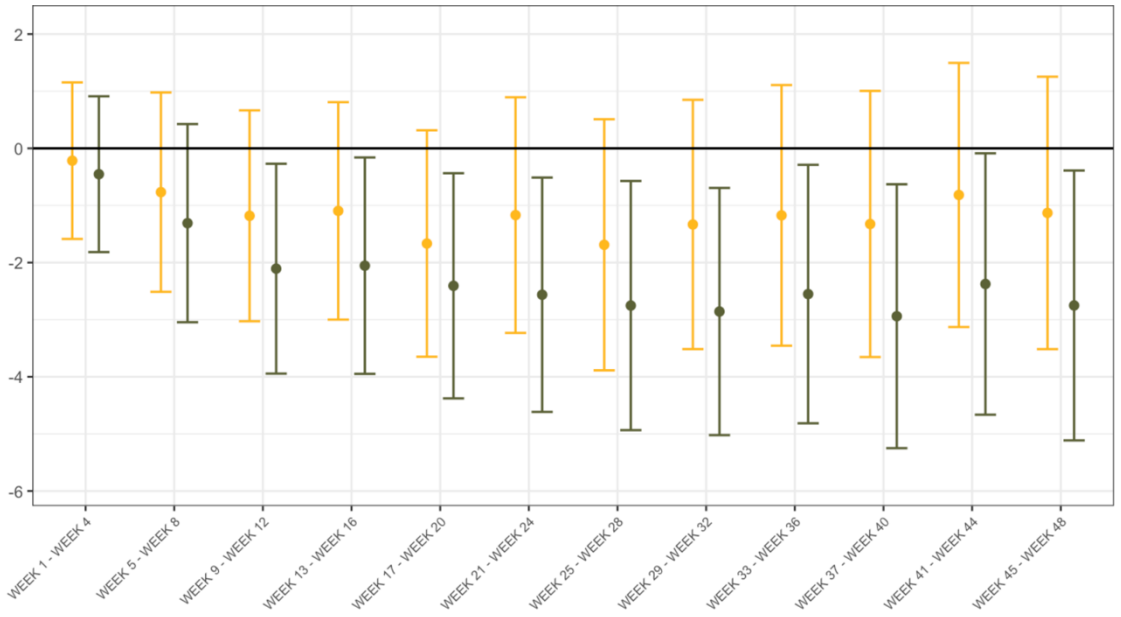
Differences in KSQ-L Score Change from Baseline vs Placebo*



*LS Means; KSQ-L = King's Sarcoidosis Questionnaire – Lung

Consistent Benefit Observed for Efzofitimod on Other Pre-Specified QOL Measures

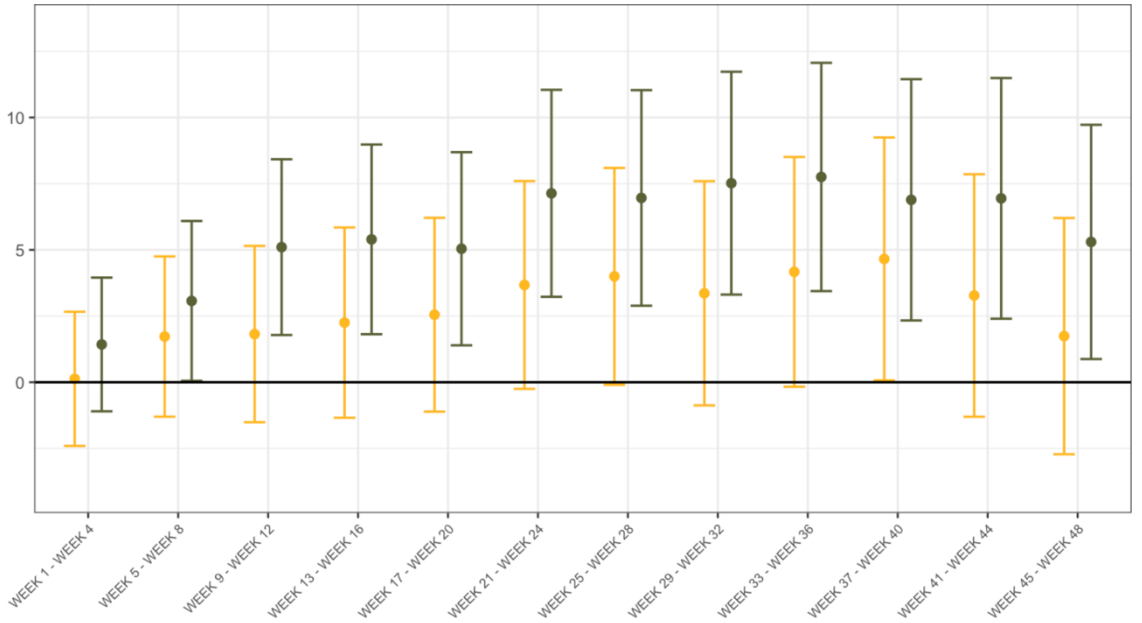
Differences in FAS Total Score Change from Baseline vs Placebo*



Time (weeks)

*Week 48 difference in LS mean change from baseline nominal p-value = **0.0226***

Differences in KSQ-GH Score Change from Baseline vs Placebo*



Time (weeks)

Efzofitimod 3 mg/kg

Efzofitimod 5 mg/kg

*Week 48 difference in LS mean change from baseline nominal p-value = **0.0197***

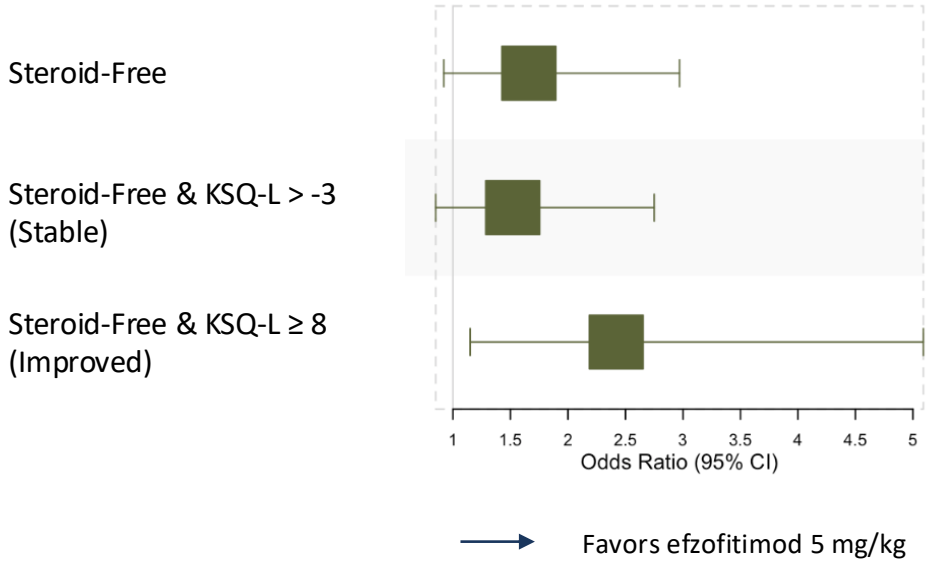
*LS Means; QOL = quality of life; FAS = Fatigue Assessment Scale; KSQ-GH = King's Sarcoidosis Questionnaire – General Health

More Patients Steroid Free with Improved KSQ-L on Efzofitimod High Dose

Steroid Free and KSQ-L Score Composite Endpoints

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
Steroid free and stable KSQ-L; n (%)[1]	33 (36.7)	41 (47.7)	41 (46.6)
Odds ratio (95% CI)	-	1.6 (0.8, 2.9)	1.6 (0.8, 2.9)
Nominal p-value	-	*0.1592*	*0.1607*
Steroid free and improved KSQ-L; n (%)[1]	13 (14.4)	24 (27.9)	26 (29.5)
Odds ratio (95% CI)	-	2.2 (1.1, 4.8)	2.4 (1.2, 5.2)
Nominal p-value	-	***0.0381***	***0.0196***

Efzofitimod 5 mg/kg vs Placebo from Logistic Regression



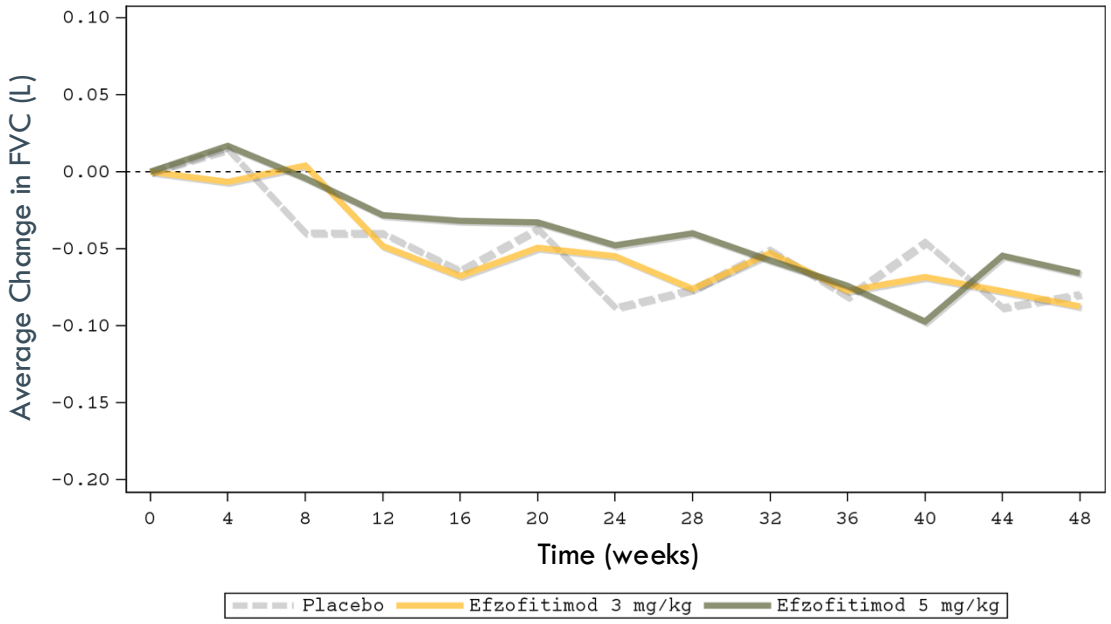
[1]Missing patients treated as non-responders

Forced Vital Capacity Maintained

FVC at Week 48 (MMRM)

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
FVC (mL), LS mean at week 48	3380.4	3369.7	3395.4
LS mean change from baseline (mL)	-84.5	-95.1	-69.4
Difference in LS means (mL)	-	-10.6	15.1
95% CI	-	-104.7, 83.5	-77.4, 107.6
Nominal p-value	-	*0.8244*	*0.7485*

Change from Baseline in FVC (L) over Time



FVC = forced vital capacity

Efzofitimod is Well-Tolerated with Consistent Safety Profile

	Placebo N=90	Efzofitimod 3 mg/kg N=86	Efzofitimod 5 mg/kg N=88
SAE	10	12	7
Treatment related (per PI)	2 (pneumonia, scrotal abscess)	2 (PPF, RSV infection)	1 (pneumonia)
Discontinuations due to AE	4	3	4
Treatment related (per PI)	2 (hypersensitivity, hypotension)	1 (PPF)	1 (CIDP)
Anti-drug antibody			
Treatment induced	1	1	3
Treatment boosted	1	0	0

SAE = serious adverse event; PI = principal investigator; PPF = progressive pulmonary fibrosis; RSV = respiratory syncytial virus; AE = adverse event; CIDP = chronic inflammatory demyelinating polyneuropathy

Key Takeaways and Next Steps

- Study did not meet primary endpoint in change from baseline in mean daily OCS dose at week 48
- Clinical benefit for efzofitimod reported across multiple disease-related health outcomes
- Improvement in quality of life for 5.0 mg/kg efzofitimod vs placebo as measured by the KSQ-L, FAS and KSQ-GH
- Trends toward increased and prolonged steroid withdrawal for efzofitimod
- Potential preservation of lung function with 5.0 mg/kg efzofitimod
- Generally well-tolerated at both the 3.0 mg/kg and 5.0 mg/kg doses, consistent with a previously observed safety profile in all trials conducted to date

Planned Next Steps

U.S. FDA Type C meeting scheduled for mid-April 2026 to review the results and determine the path forward for efzofitimod in pulmonary sarcoidosis

OCS = oral corticosteroids; KSQ-L = King's Sarcoidosis Questionnaire – Lung; FAS = Fatigue Assessment Score; KSQ-GH = King's Sarcoidosis Questionnaire – General Health

SSc-ILD

Indication Expansion Represents Upside Opportunity in Interstitial Lung Disease

SSc-ILD is Common and Deadly Manifestation of Systemic Sclerosis

Disease Pathology

- Autoimmune disease also known as scleroderma
- Characterized by inflammation and scarring, or fibrosis, of skin and other organs, including the lungs

Diagnosis

1) ILD diagnosed secondary to underlying SSc
2) Confirmed with imaging, PFTs and blood work

Epidemiology

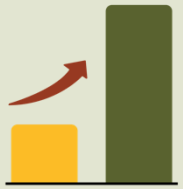
>1.5 million patients worldwide

Current Treatments

Mycophenolate, cyclophosphamide → Tocilizumab, rituximab → Nintedanib

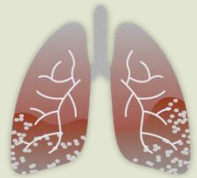
- Efzofitimod positioned as 2nd line in patients who progress on or cannot tolerate MMF / CYC
- Addressable population in major markets: **>50k**(1)
- Upside potential: improve underlying systemic disease

45-55 is the average age of onset for SSc-ILD



3x greater mortality risk than SSc alone

70-90% of ILD develops in the first three years of SSc

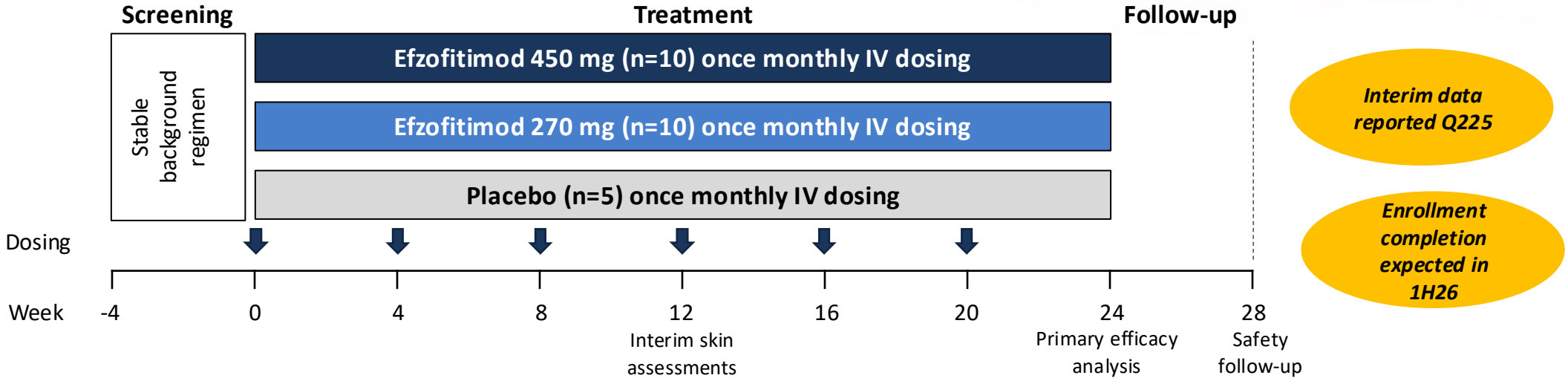


30% of patients develop lung fibrosis

(1) Back Bay Life Science Advisors

Phase 2 POC Trial Enrolling in SSc-ILD

Primary objective: Assess the efficacy of efzofitimod on pulmonary, cutaneous, and systemic manifestations in SSc-ILD



Population: SSc with progressive ILD

- Patients with SSc (ACR/EULAR criteria), and ILD (baseline HRCT)
- Progressive disease (recent onset, evidence for inflammation, diffuse cutaneous SSc)
- On background mycophenolate therapy or equivalent

Primary Endpoint

- Lung function: forced vital capacity

Key Secondary Endpoints

- Symptom control: PROs
- Skin: histopathology, gene profiling, biomarkers, mRSS

Interim analysis including skin assessments and serum biomarkers at baseline and week 12 for N= 8 diffuse and limited SSc-ILD patients showed stable or improved mRSS for all patients, with 3 out of 4 efzofitimod treated diffuse SSc-ILD patients experiencing an improvement in mRSS of 4 points or greater

Translating tRNA Synthetase Biology into New Therapies for Inflammation and Fibrosis

Corporate Summary

Disruptive tRNA synthetase biology platform

- Extracellular tRNA synthetases represent potential new class of medicines
- IP directed to more than 200 synthetase fragments represents unique and validated drug discovery method

Lead candidate efzofitimod for ILD represents $2-5b market opportunity

- Novel biologic immunomodulator with upstream target for ILD with little competition
- Topline results reported for Phase 3 EFZO-FIT™ study in pulmonary sarcoidosis; U.S. FDA Type C meeting scheduled for mid-April 2026 to review the results and determine the path forward for efzofitimod in pulmonary sarcoidosis
- Phase 2 EFZO-CONNECT™ study in SSc-ILD expected to complete enrollment in 1H26
- U.S. FDA orphan drug designations for sarcoidosis and SSc; Fast Track designations for pulmonary sarcoidosis and SSc-ILD; E.U. orphan drug designations for sarcoidosis and SSc; Japan orphan drug designation for sarcoidosis
- Commercial exclusivity in the U.S. anticipated into at least 2039

Growing pipeline targeting inflammation and fibrosis

- Multiple tRNA synthetase candidates in preclinical development
- Candidates bind targets in novel ways with potential implications in high value markets

Strong financial fundamentals

- ~$80.9m in cash, restricted cash, cash equivalents and investments as of December 31, 2025
- Partnership with Kyorin Pharmaceutical for efzofitimod for ILD in Japan

Thank You