# A New Approach to Inflammatory Diseases

March 2026

Corporate Presentation

NASDAQ: GRI | gribio.com

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# Highlights

## Advancing an Innovative Pipeline of Immune Modulators for the Treatment of Inflammatory, Fibrotic and Autoimmune Diseases

### Unique Mechanism of Action

Oral RARβγ agonist to target key immune cells earlier in the inflammatory cascade & activate lung repair mechanisms to interrupt disease progression

### High-Value Indications

**~140K**[1]
People in the US
**Idiopathic Pulmonary Fibrosis**

**~160-200K**[2]
People in the US
**Systemic Lupus Erythematosus**

### IPF Phase 2a Topline Data

GRI-0621 treatment demonstrated improvement in FVC, fibrosis resolution & lung repair biomarkers, with no safety issues reported

***RARβγ Activation is a more selective approach to Immunomodulation***

***The challenge in treating fibrotic disease is targeting biology core to inflammation and fibrosis development while avoiding systemic toxicities***





1. Maher, T.M., et al., Global incidence and prevalence of idiopathic pulmonary fibrosis. Respir Res, 2021. 22(1): p. 197
2. Izmirly, P.M., et al., Prevalence of Systemic Lupus Erythematosus in the United States: Estimates From a Meta-Analysis of the Centers for Disease Control and Prevention National Lupus Registries. Arthritis Rheumatol, 2021. 73(6): p. 991.

# Pipeline Targeting High-Value Indications in Need of Innovation

| Programs | Class | Indication | Preclinical | Phase 1 | Phase 2 | Phase 3 | Status |
|---|---|---|---|---|---|---|---|
| GRI-0621 | RARβγ Agonist | Idiopathic Pulmonary Fibrosis (IPF) | | | *Phase 2a Biomarker Study* | | **Topline Data in Line with Reported Positive 2- & 6-Week Interim Data**<br>**GRI-0621 Increased FVC over 12 Weeks**<br>**Robust Response Across Biomarkers** |
| GRI-0803 | Type 2 diverse NKT (dNKT) Agonist | Initial Focus: Systemic Lupus Erythematosus (SLE) | | | | | **IND-Enabling and Phase 1 Program Expected to Advance in 2026** |
| GRI-NKT | Type 2 diverse NKT (dNKT) Agonist | Multiple Indications | | | | | **Multiple Pipeline Expansion Opportunities** |

***Library consisting of 500+ Proprietary Compounds to Fuel a Growing Pipeline***

# GRI-0621 Has a Unique Profile in the IPF Therapeutic Landscape

## Well Suited as a Mono or Combination Therapy

### Unique Mechanism of Action (MOA)

- RARβγ agonist modulates the activity of key immune cells driving fibrosis
- Initiates epithelial cell and lung repair mechanisms
- Upstream of key targets, impacting early and late drivers of fibrosis to interrupt disease progression

### Strong Preclinical Data

- GRI-0621 was active across pulmonary, hepatic and renal pre-clinical models of fibrosis
- Pre-clinical data show GRI-0621 outperformed standard of care in pulmonary fibrosis models
- GRI-0621 resolved tissue injury, lowered TGFβ production, myofibroblast activation and ECM deposition

### Phase 2a Topline Data

- Positive overall tolerability of oral GRI-0621 consistent with earlier studies of oral tazarotene in over 1,700 subjects
- Robust biomarker data supportive of immunomodulation, fibrosis resolution and alveolar basement membrane repair
- No FVC decline over 12 weeks in patients taking GRI-0621 and standard of care (SOC) compared to SOC alone

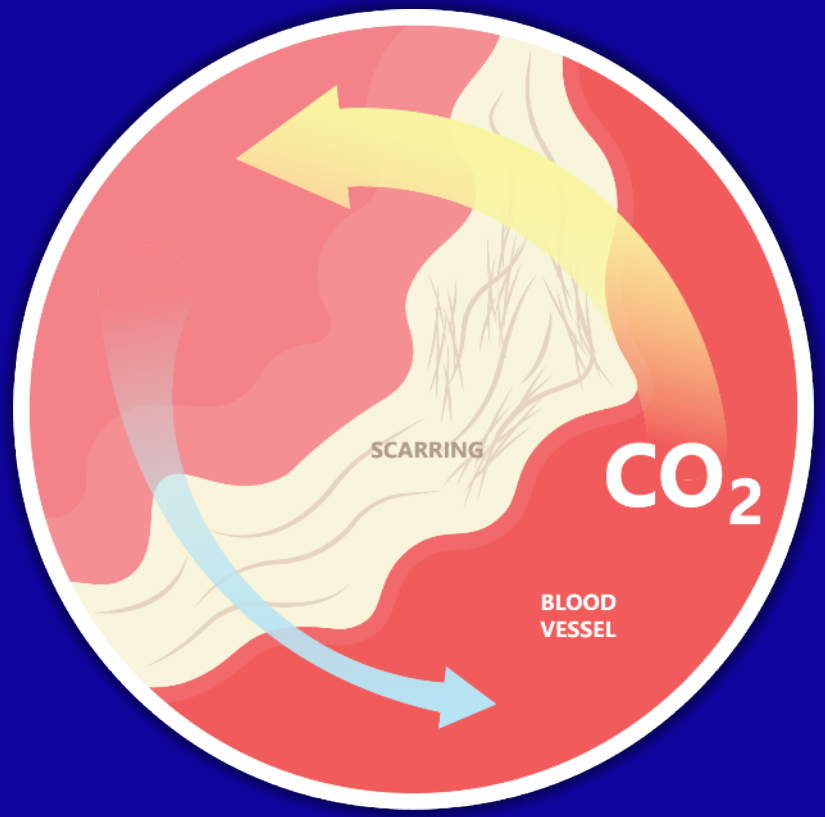
# GRI-0621

## Idiopathic Pulmonary Fibrosis (IPF)

- Positive safety and biomarker data reported after 2, 6 and 12-week analyses
- Unique MOA differentiated from other approaches supports use as a single agent or as combination therapy
- Leveraging FDA 505(b)(2) regulatory pathway
- Orphan indication with ~40K newly diagnosed cases annually

# The Need in Idiopathic Pulmonary Fibrosis

## A Rare Chronic Progressive Pulmonary Disease with Abnormal Scarring of the Lungs Blocking the Movement of Oxygen into the Bloodstream




**~66** Age of onset[1]

**50%** 5/10 die within 2-3 years of diagnosis[2]

**20%** 8/10 die within 5 years of diagnosis[2]

**0%** Survival without a lung transplant[3]

**Current Treatments are Limited with Only 3 Approved Drugs**

Significant side effects, limited compliance and no impact on survival[3]

Despite challenges, total 2022 **sales were ~$4.3 billion combined**[4]



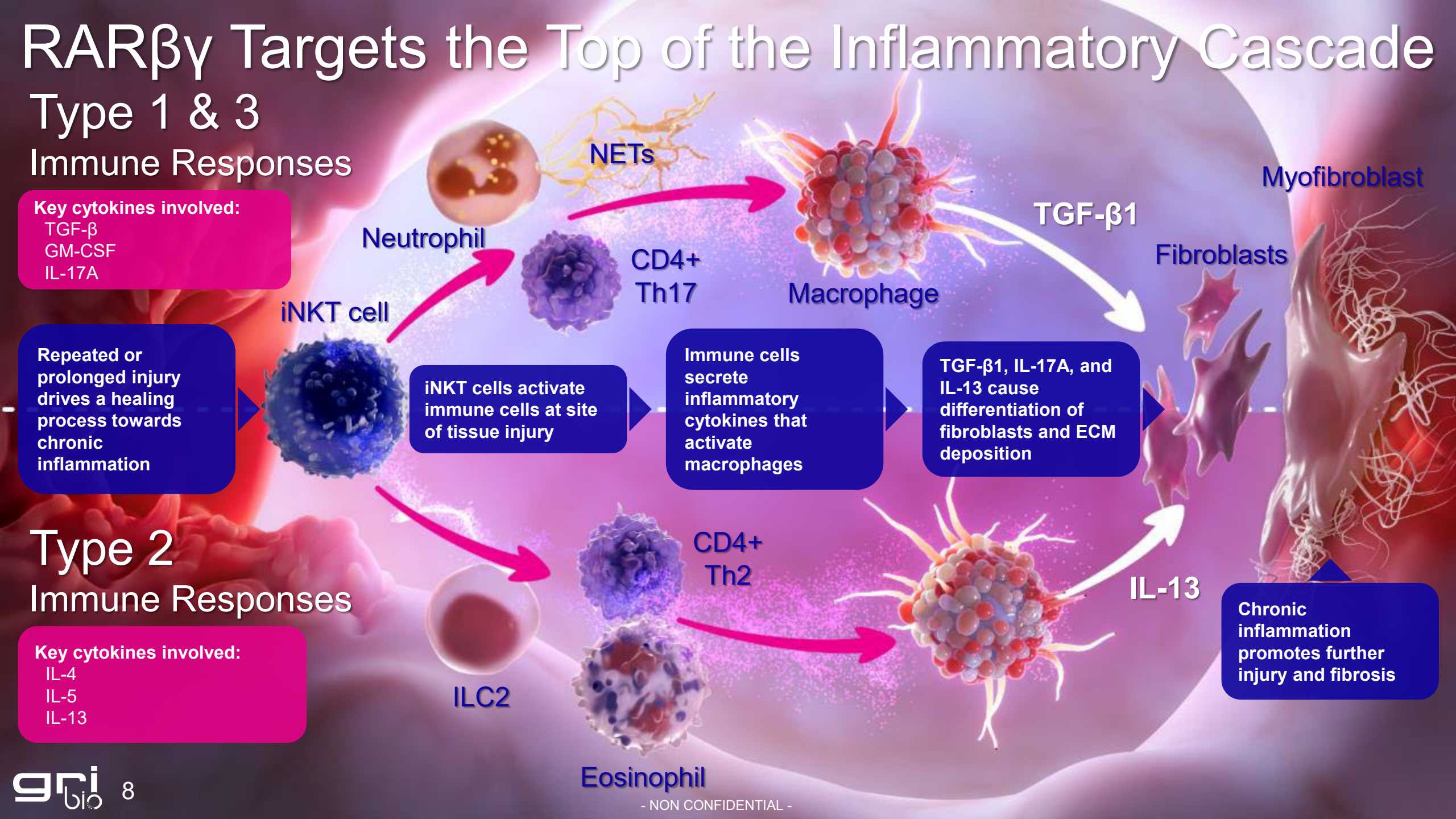


1. Castriotta, R. J. (2010). Workshop on Idiopathic Pulmonary Fibrosis in Older Adults. Chest, 138(3), 693–703
2. Sharif, R. (2017). Overview of Idiopathic Pulmonary Fibrosis (IPF) and Evidence-Based Guidelines. Am J Manag Care, 23(11), 176–182
3. Maher, T.M., et al., (2021). Global incidence and prevalence of idiopathic pulmonary fibrosis. Respir Res, 22(1),197
4. Companies' earnings reports

# RARβγ Targets the Top of the Inflammatory Cascade

## Type 1 & 3 Immune Responses

**Key cytokines involved:**
- TGF-β
- GM-CSF
- IL-17A



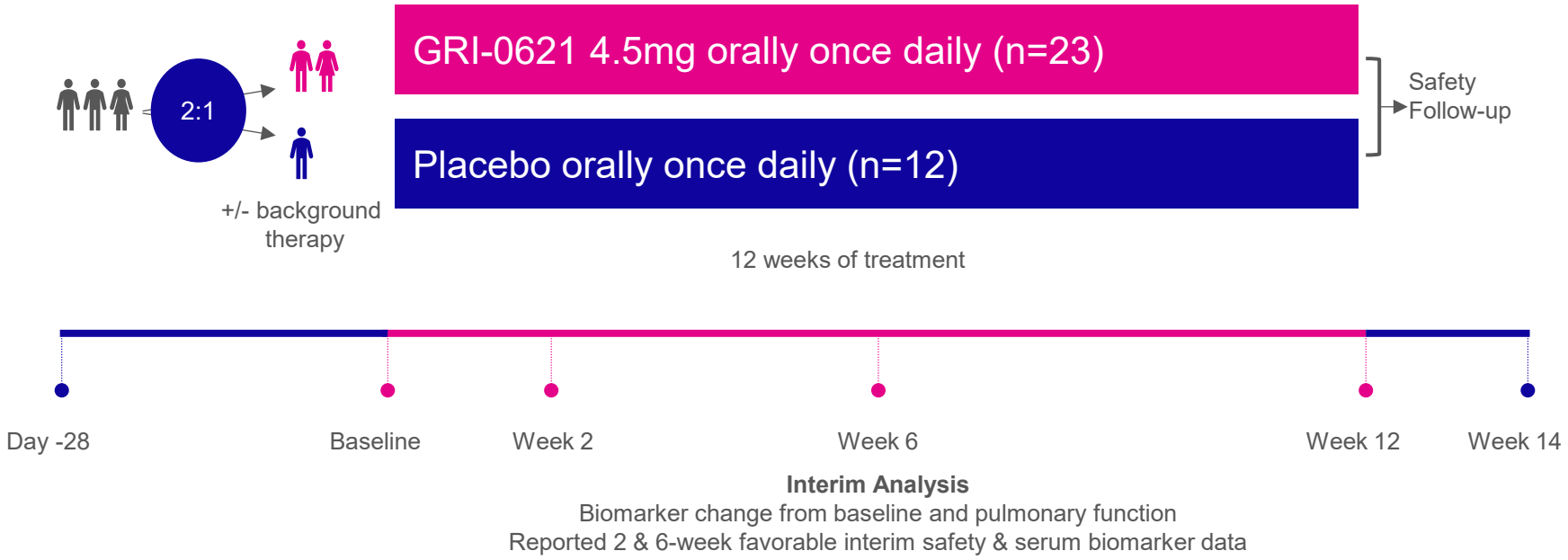

## Type 2 Immune Responses

**Key cytokines involved:**
- IL-4
- IL-5
- IL-13

# Phase 2a Study in IPF

## KEY INCLUSION CRITERIA

1. Men or women 40-85 yrs
2. Confirmed IPF diagnosis
3. FVC > 50% predicted
4. FEV1/FVC > 0.65
5. DLCOc > 30% predicted
6. Life expectancy of at least 12-months
7. Subjects on approved IPF therapy must remain on their current medication from screening until the last study visit




**Interim Analysis**
Biomarker change from baseline and pulmonary function
Reported 2 & 6-week favorable interim safety & serum biomarker data

### PRIMARY & SECONDARY ENDPOINTS

Safety, tolerability and PK

Biomarkers change from baseline

To determine the PD activity of oral GRI-0621 in blood after 6 & 12 weeks and from BAL after 12 weeks of treatment in a sub-study

### EXPLORATORY ENDPOINTS

Effect of GRI-0621 on pulmonary function at baseline and after 6 and 12 weeks of treatment

# GRI-0621-IPF-02 Trial

## Primary Endpoint

The **primary endpoint was achieved**, demonstrating oral GRI-0621 to be **well tolerated**, consistent with earlier studies of oral tazarotene in over 1,700 subjects treated for up to a year, and **no overlapping GI toxicities** with approved drugs

## Secondary Endpoints

Evaluating various disease-specific biomarkers demonstrated **robust positive trends** in change from baseline in several key biomarkers of **collagen turnover**, **immunomodulation**, and **differential gene expression** in GRI-0621 treated compared to standard of care, **suggestive of resolution of fibrosis and induction of a lung repair mechanism**

## Exploratory Endpoint

The exploratory endpoint of lung function demonstrated **a 95% increase** in subjects with an increase in FVC and a **60% decrease** in subjects with a >10% decline in FVC in GRI-0621-treated subjects compared to the control arm after 12 weeks of treatment

# GRI-0621

## Idiopathic Pulmonary Fibrosis (IPF)

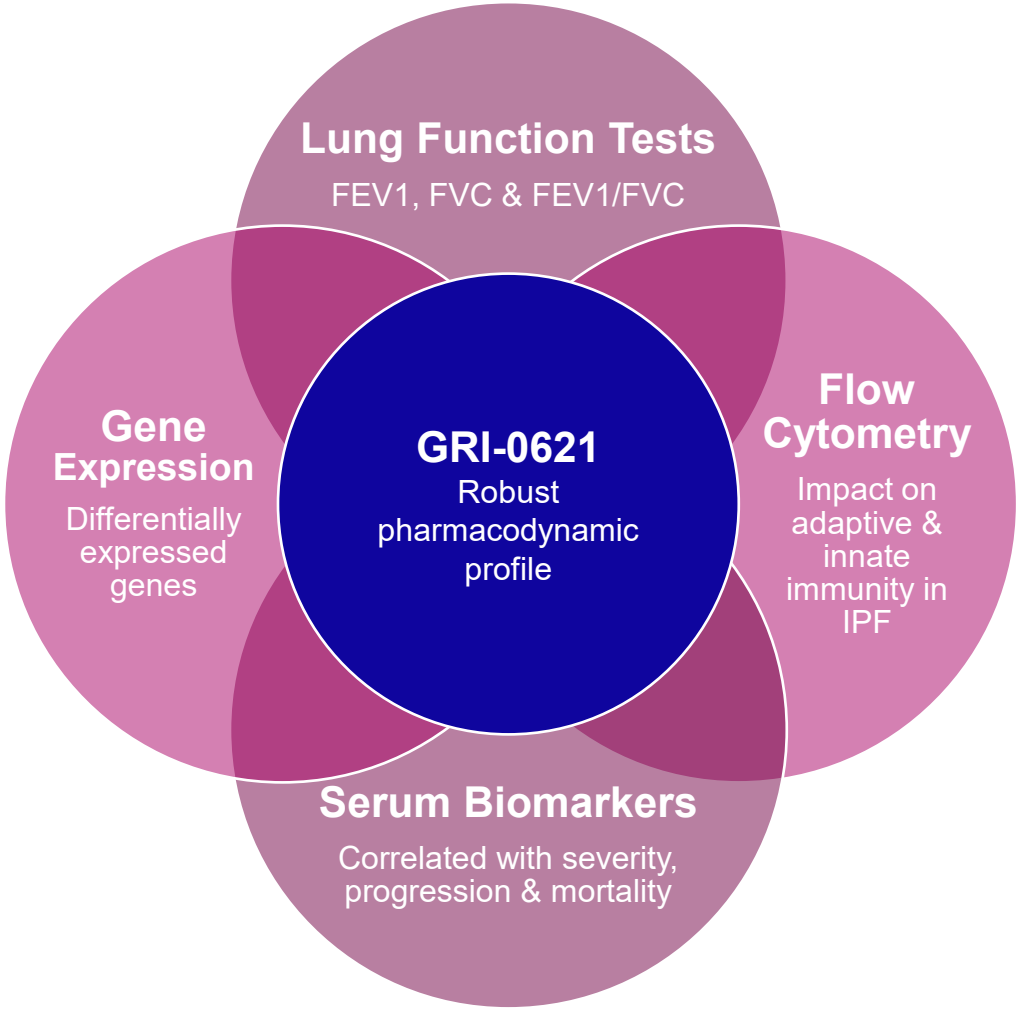
# Biomarkers

# GRI-0621-IPF-02 Biomarker Analysis

Extensive biomarker analysis to build a robust Pharmacodynamic profile of GRI-0621

- Lung function tests in smaller studies less than 24 weeks have limited clinical significance on their own
- Serum biomarkers can identify the rates of collagen synthesis and degradation, suggestive of fibrosis resolution or formation
- Flow cytometry can highlight changes in the immune system, and identify immune signatures of subjects that may benefit the most from GRI-0621
- Differential gene expression can help characterize MOA and support the results from other analysis

Leveraging multiple analyses at the group and individual level to amplify signal in Phase 2a



# Fibrosis is the Accumulation of Excess Extracellular Matrix

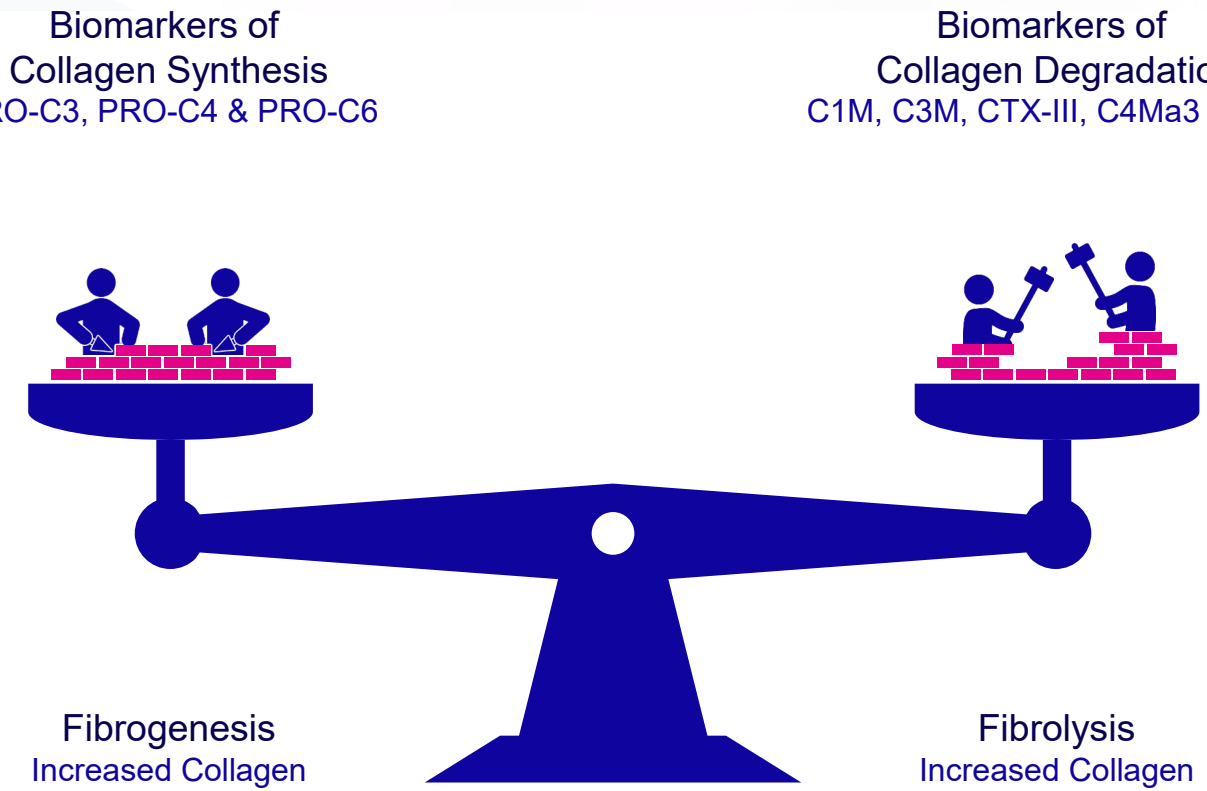
## The Remodeling Rate Can Inform Whether Fibrosis is Worsening or Improving

### Fibrosis, Collagen Turnover & The Remodeling Rate

Fibrosis is the accumulation of excess extracellular matrix comprised mainly of collagen

Serum biomarkers of collagen turnover can be measured to determine whether fibrosis is worsening or improving

The Remodeling Rate is the ratio of collagen formation to degradation, and indicates whether fibrosis is worsening (fibrogenesis) or improving (fibrolysis) over time








PRO-C3 and C3M, and PRO-C6 and C6M, are biomarkers of type III and VI collagen formation and degradation, respectively. CTX-III is a biomarker of crosslinked and degraded type III collagen found in fibrotic tissue. PRO-C4 and C4Ma3 are biomarkers of type IV collagen formation and degradation, respectively, found in the alveolar basement membrane.

# GRI-0621 Drove Resolution of Type VI Collagen

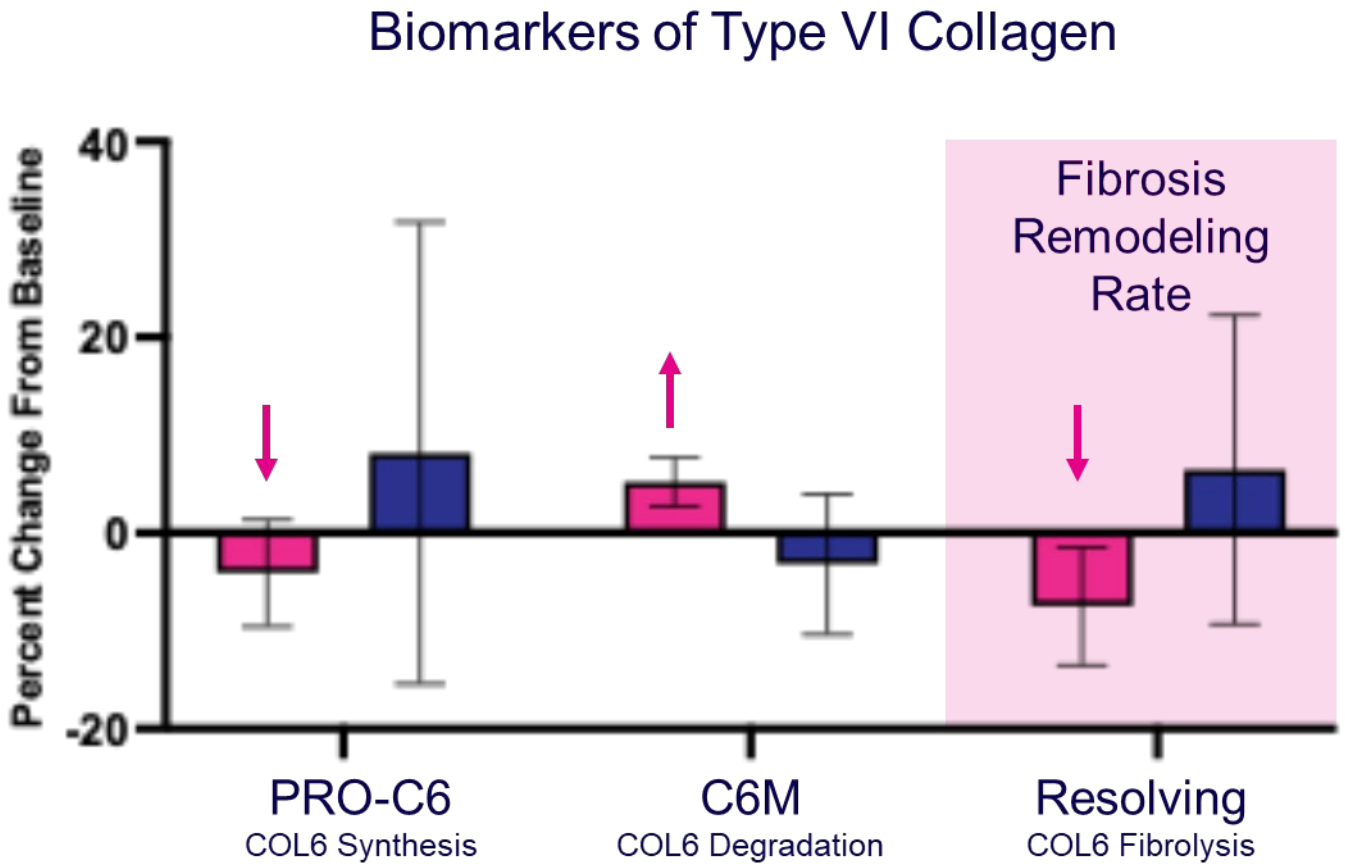
## Decreased Synthesis & Increased Degradation Induces Fibrolysis

### Type VI Collagen

GRI-0621 reduced biomarkers of COL6 synthesis (PRO-C6)

Increases biomarkers of COL6 degradation (C6M)

And drove the remodeling rate towards fibrolysis and resolution of fibrosis




COL6 (type VI collagen)
PRO-C6 (COL6 C-terminal; endotropin)
C6M (COL6 degraded by MMPs)

# GRI-0621 Drove Repair of Alveolar Basement Membrane

## Increased Synthesis & Decreased Degradation of Type IV Collagen

### Type IV Collagen

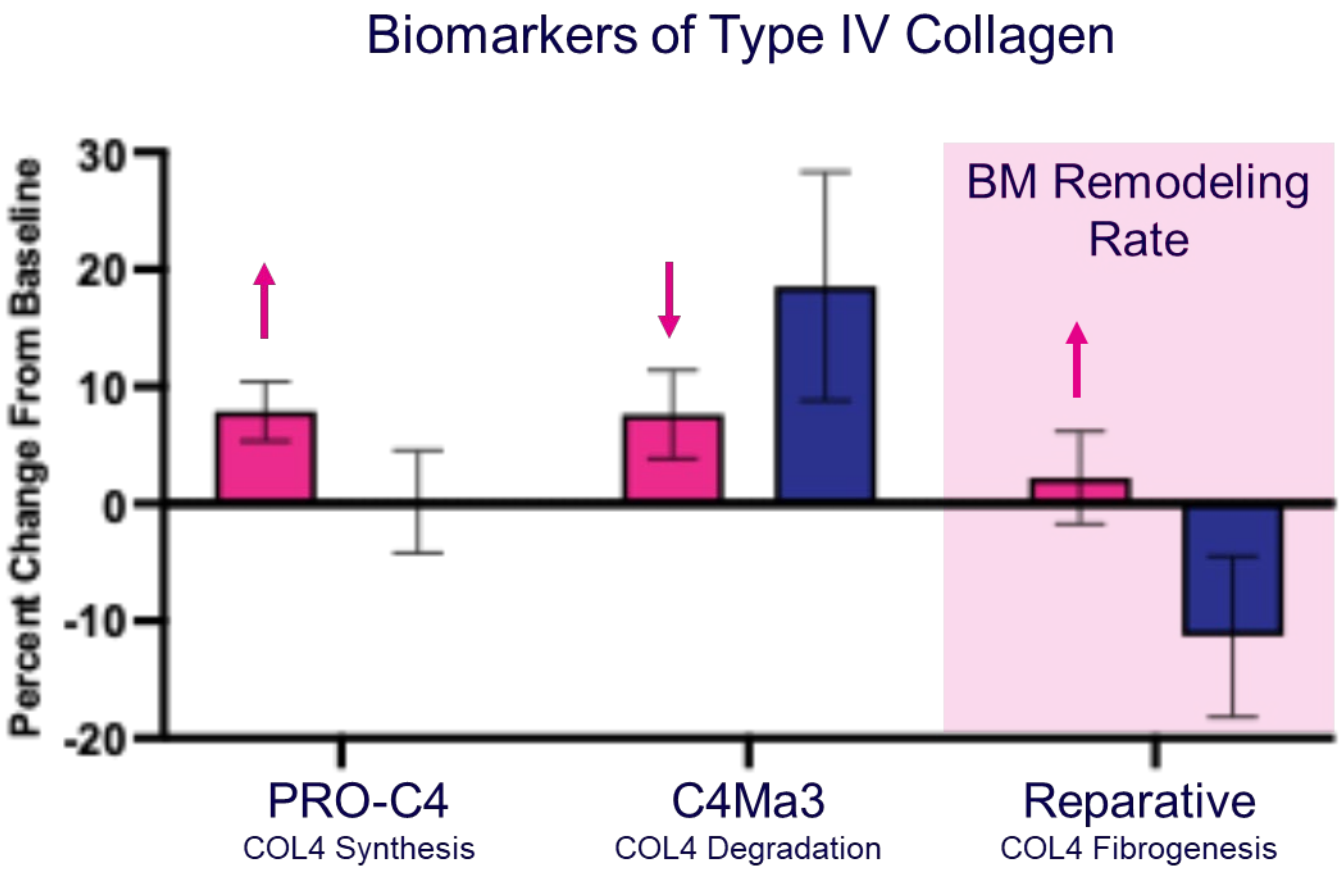
Epithelial injury and destruction of the alveolar BM is a hallmark of IPF

Repair of the BM is a crucial step in lung tissue repair

GRI-0621 increased biomarkers of COL4 synthesis (PRO-C4)

And decreased biomarkers of COL4 degradation (C4Ma3)

Driving repair of the BM, a critical step towards lung repair



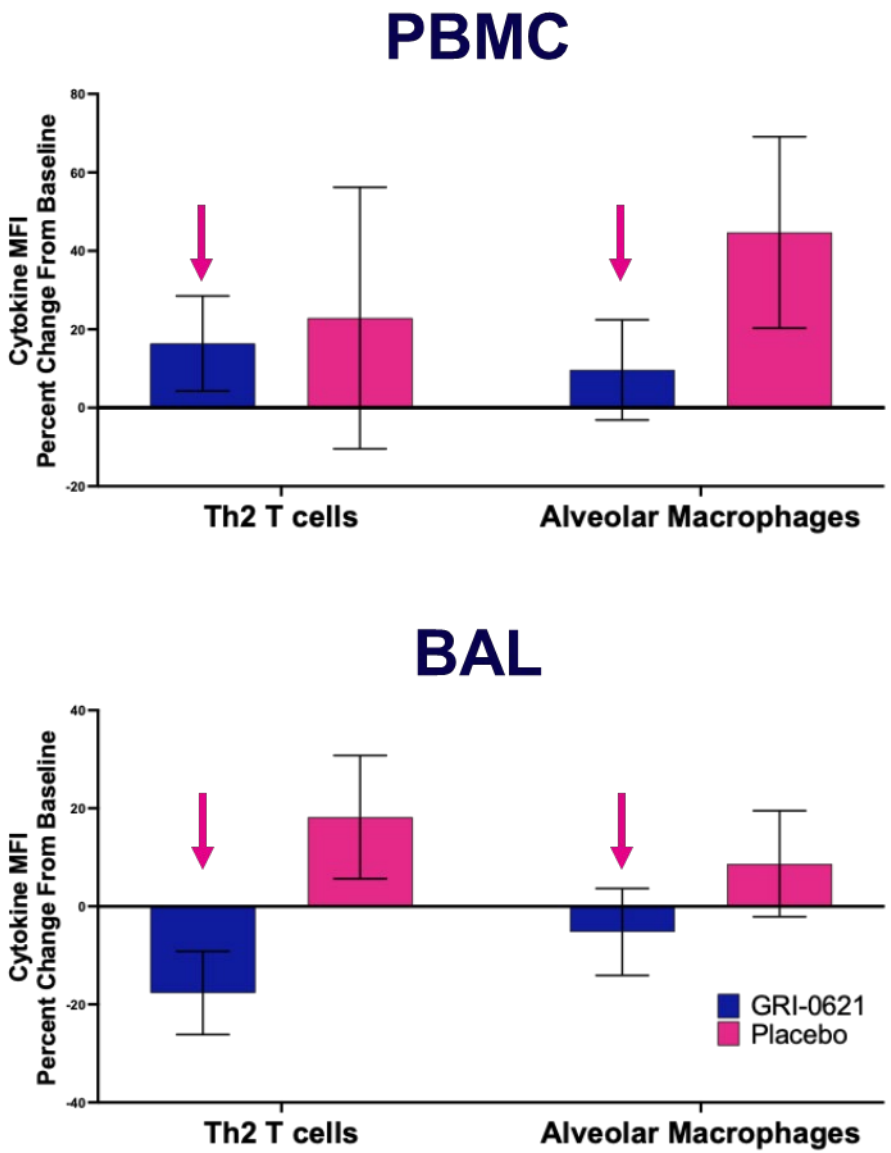



COL4 (type IV collagen)
PRO-C4 (COL4 7S)
C4Ma3 (COL4 α3 chain degraded by MMPs)
BM (basement membrane)

# GRI-0621 Shifts Immune Profile from Type 2 Pro-Fibrotic towards Type 1 Anti-Fibrotic

| Cytokine | PBMC | BAL |
|---|---|---|
| ↑ IFNγ | ✓ | ✓ |
| ↓ IL-4 | ✓ | ✓ |
| ↓ IL-13 | ✓ | ✓ |
| ↓ IL-17A | ✓ | ✓ |
| ↓ IL-22 | ✓ | ✓ |
| ↓ TGFβ1 | ✓ | ✓ |

# Type 2 IL-4 Decreased



# Strong Biomarker Data Demonstrating Improvement in Key Drivers of Fibrosis & Lung Repair

Support Mechanism of Action of iNKT Inactivation & RARβ/γ Agonism

| Fibrotic Process | Serum Biomarkers | Differentially Expressed Genes | Immune Phenotypes |
|---|---|---|---|
| Inflammation, neutrophil degradation & lung injury | ✓ | ✓ | |
| Myofibroblast activation, collagen synthesis & ECM deposition | ✓ | ✓ | ✓ |
| Basement membrane repair | ✓ | ✓ | |
| Re-epithelialization & lung repair | | ✓ | |
| Mechanisms of action (iNKT cell inactivation & RAR signaling) | | ✓ | ✓ |

# GRI-0621

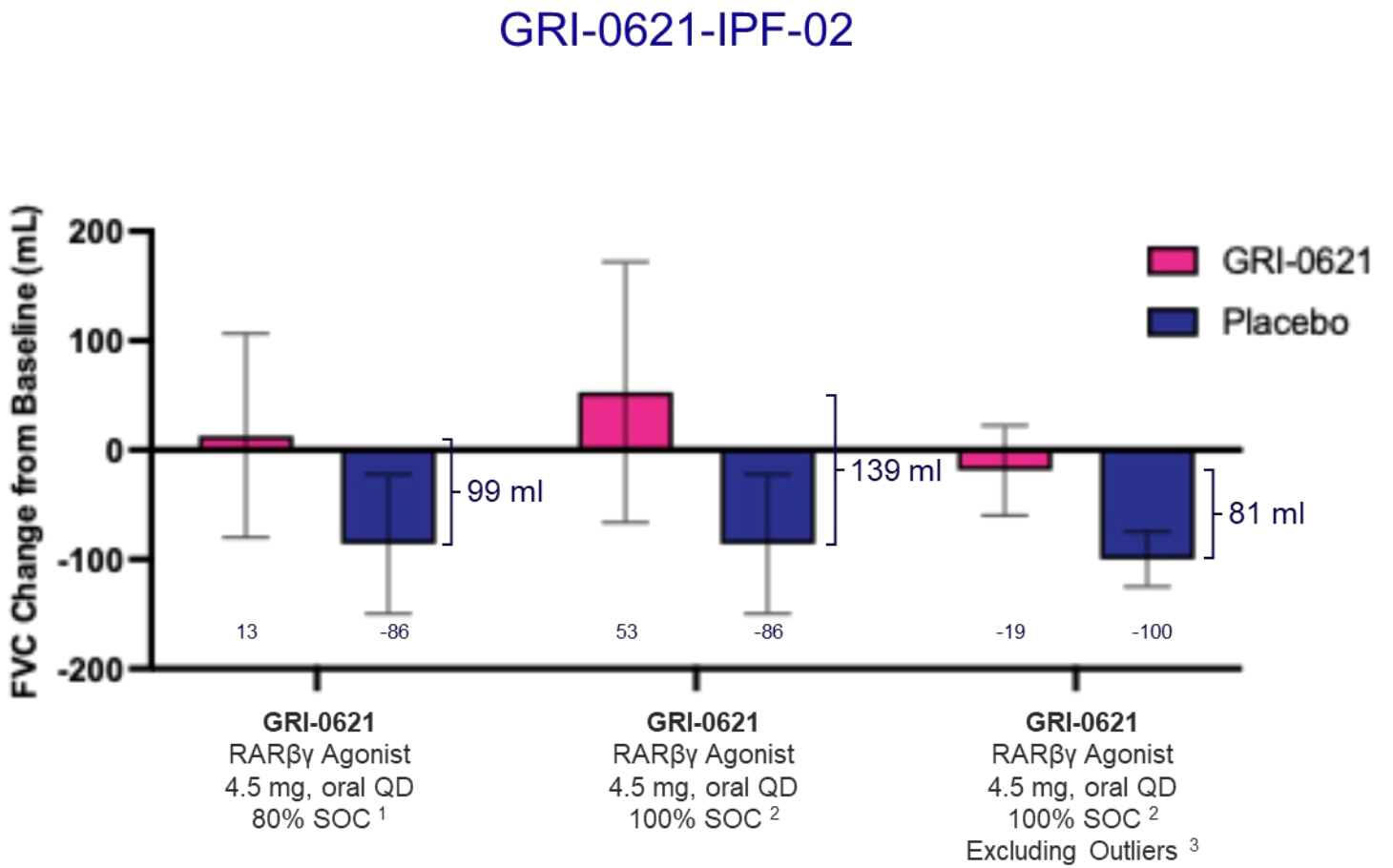
Idiopathic Pulmonary Fibrosis (IPF)

## Forced Vital Capacity

# GRI-0621 Increased FVC from Baseline

In subjects also taking standard of care, GRI-0621 treated subjects demonstrated a **53 ml increase** in FVC at 12 weeks, and a **139 ml improvement** in placebo-adjusted FVC change from baseline

To remove outlier effects, the highest and lowest responder in each arm were removed in a post hoc analysis

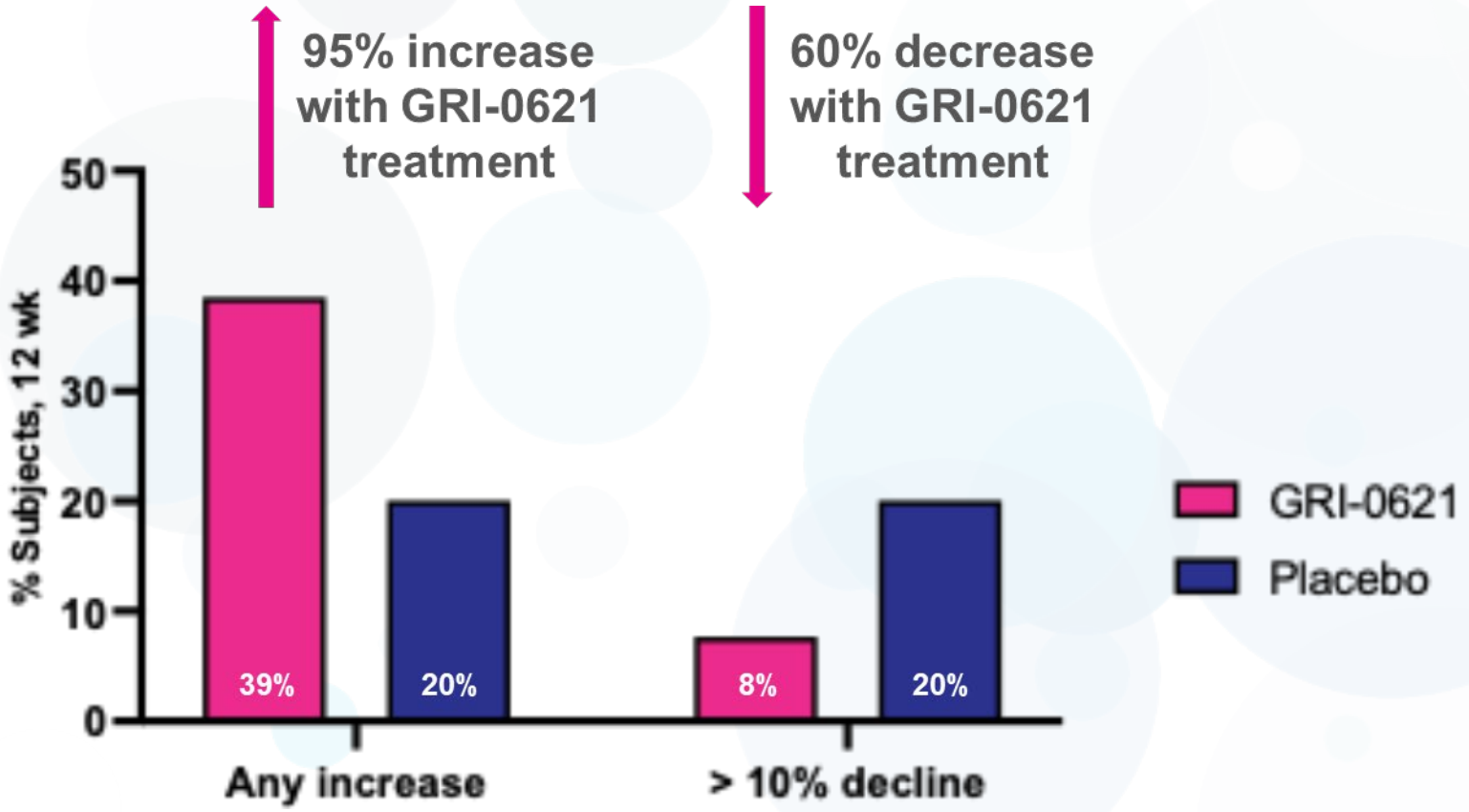







1 All completers minus a single data point from a subject who exhibited unreliable spirometry
2 All completers taking either nintedanib or pirfenidone in addition to GRI-0621 or placebo
3 All completers excluding the highest and lowest responders in each arm

# 95% More Subjects Experienced an FVC Increase and 60% Fewer Experienced a Significant FVC Decline After 12-weeks of GRI-0621Treatment Compared to SOC



# GRI-0621

Idiopathic Pulmonary Fibrosis (IPF)

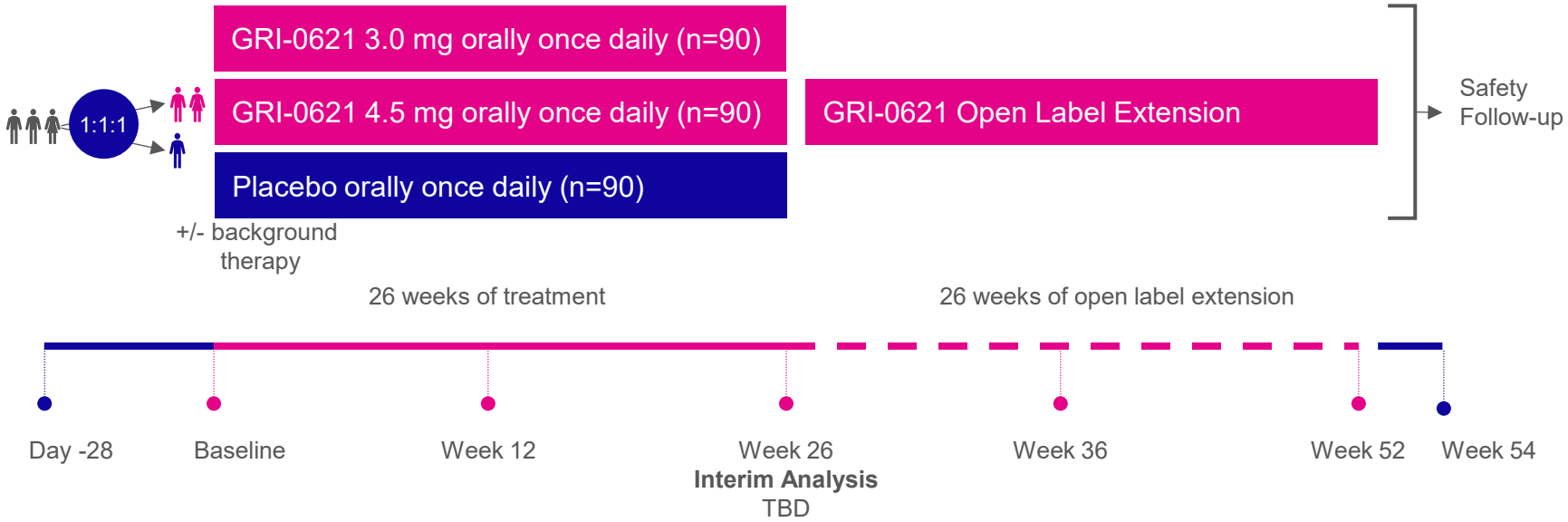
## Phase 2b

## IPF Design

# Phase 2b Study in IPF

## KEY INCLUSION CRITERIA

1. Men or women 40-85 yrs
2. Confirmed IPF diagnosis
3. FVC > 45% but < 90% predicted
4. FEV1/FVC > 0.65
5. DLCOc > 25% but < 90% predicted
6. Life expectancy of at least 12-months
7. Subjects on approved IPF therapy must remain on their current medication from screening until the last study visit




**PRIMARY & SECONDARY ENDPOINTS**

Absolute Change From Baseline in FVC (ml) at Week 26

Time to the First Occurrence of Any of the Components of the Composite Endpoint: Time to First Acute IPF Exacerbation, First Hospitalization for Respiratory Cause, or Death (Whichever Occurs First) Over the Duration of the Trial

Absolute Change From Baseline in Living With Pulmonary Fibrosis (L-PF) Symptoms Dyspnea Domain Score at Week 26

Safety and Tolerability as assessed by adverse events (AEs) at week 26 compared to placebo

# dNKT Agonists

GRI-0803, GRI-0729, GRI-0124, & 500+ compound library targeting innate-like adaptive immunoregulatory T cells

**GRI-0803 Target IND Filing in 2026**

**Steps Toward IND Filing**

- Validate bioanalytical methods
- Complete cGMP manufacturing
- Complete toxicology studies

# The Need in Systemic Lupus Erythematosus

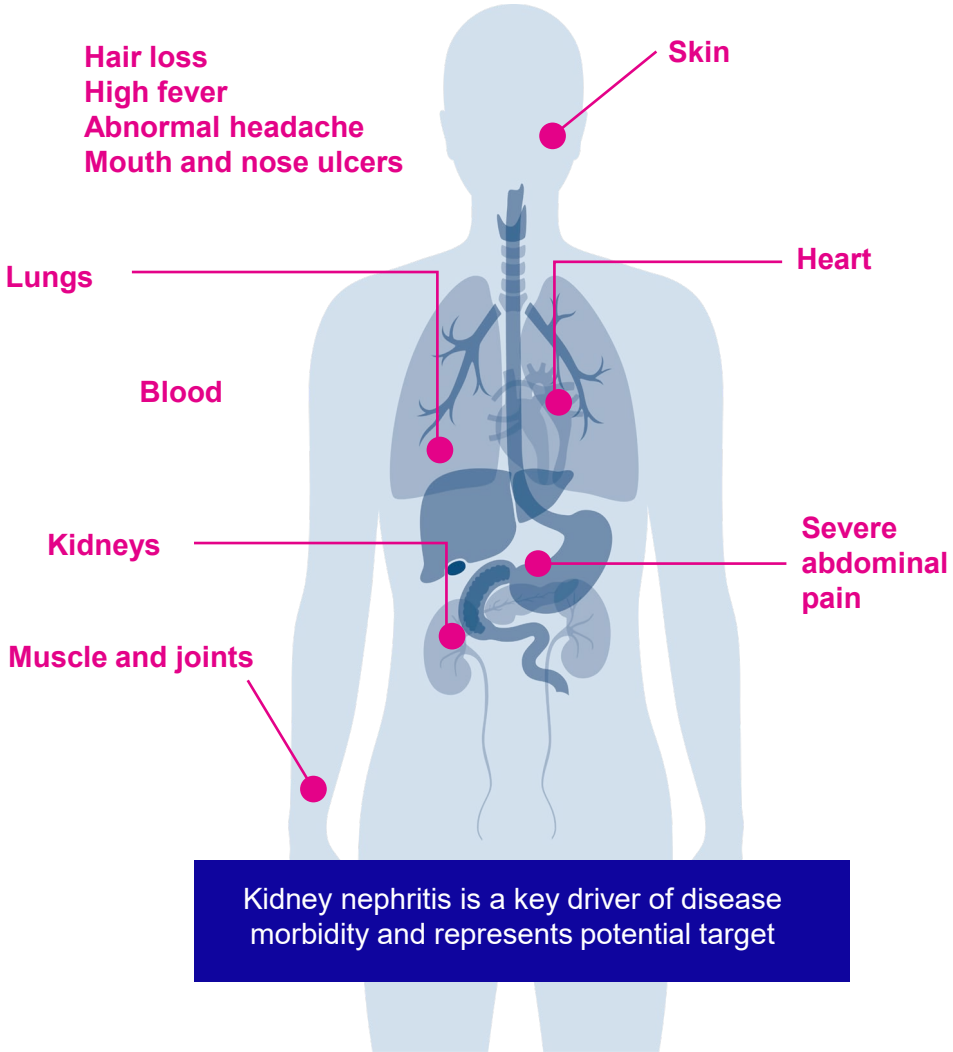
The most common form of lupus, SLE, is an autoimmune disease in which the immune system attacks its own tissue and organs

| ~160K | 70% | 15 - 44 |
|---|---|---|
| **PREVALENCE** Confirmed as definite SLE[1] | **DIAGNOSIS** Number of all lupus cases[2] | **AGE RANGE** Commonly affects women of childbearing age[1] |

Current treatments are limited, consisting primarily of immunosuppressive therapies

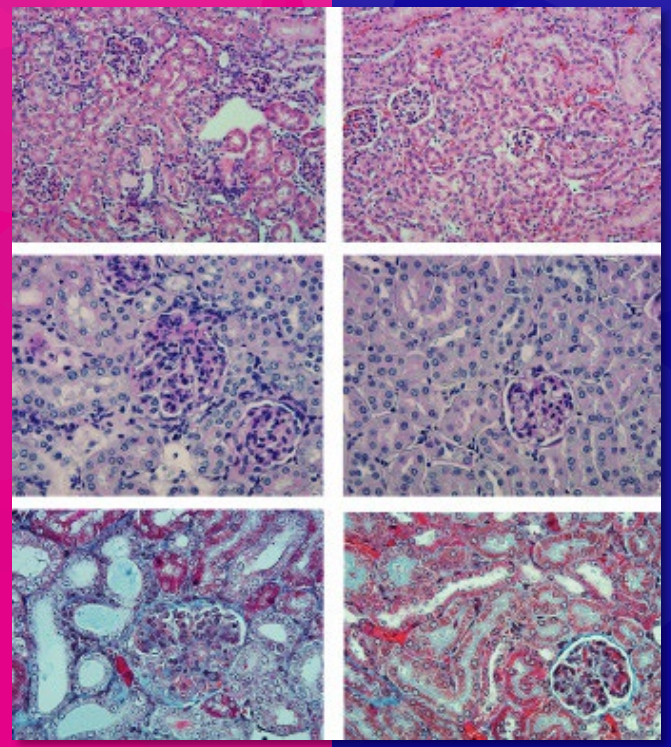
Only 2 drugs approved for SLE in the past 50 years

## Can Affect the Whole Body




Kidney nephritis is a key driver of disease morbidity and represents potential target

# dNKT Agonist Observed to Inhibit Lupus Nephritis in Model

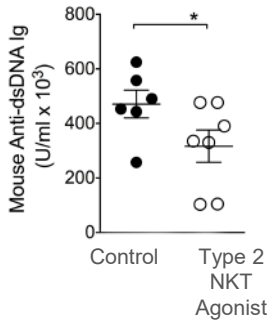


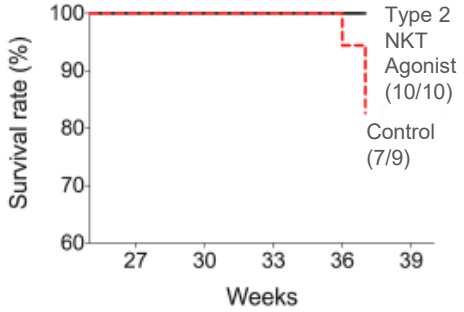
Control

Type 2 NKT Agonist

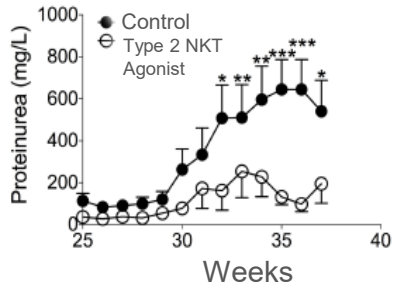
- ✓ **Inflammation Decreased**
- ✓ **Interstitial Anatomy Improved**
- ✓ **Collagen Deposition & Fibrosis Stopped**

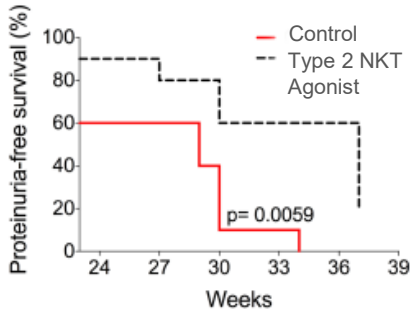
- ✓ **Improvement in Auto-Antibodies & Overall Survival**




- ✓ **The Most Common Manifestation of Lupus Nephritis & Renal Damage, Proteinuria, Improved**



# Summary

GRI0-0621 Phase 2a Topline Data Met its Primary and Secondary Endpoints and Increased FVC over 12 weeks of Treatment

Elevating Clinical Stage Biotechnology Company Advancing Innovative Pipeline Across Multiple Orphan and High-Value Inflammatory, Fibrotic and Autoimmune Diseases

## Compelling Science

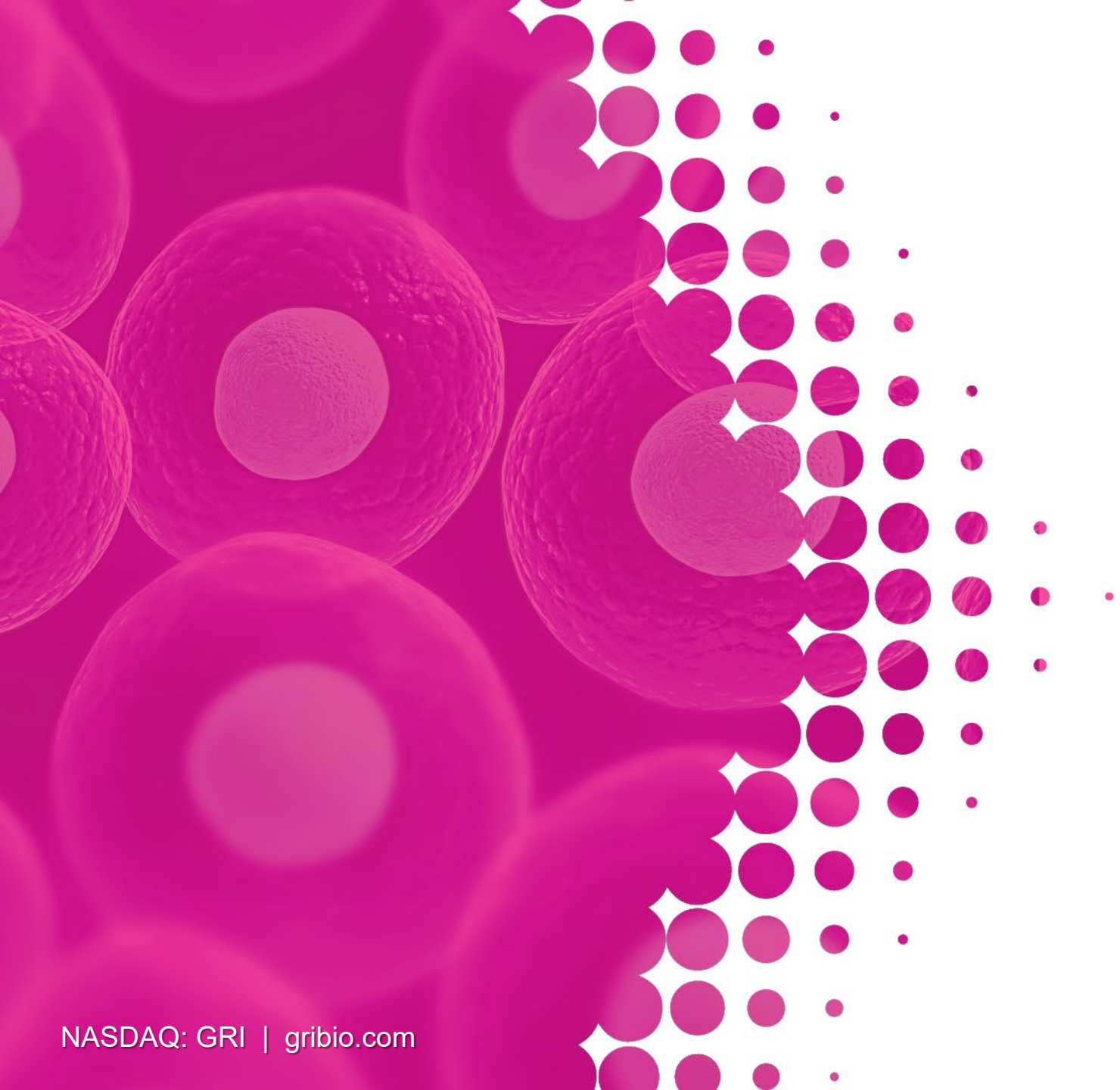
Leading technology targeting earlier in the inflammatory cascade to interrupt disease progression

## High-Value Indications

Clinical pipeline in potential high-value indications with multiple pipeline expansion opportunities

## Proven Team

Team with proven immunology, drug development, and clinical experience



# A New Approach to Inflammatory Diseases

# Thank You!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