A New Approach to Inflammatory Diseases

7th Antifibrotic Drug Development Summit

Dr. Vipin Kumar
Chief Scientific Officer
vk@gribio.com

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GRI Bio at a Glance

Advancing an Innovative Pipeline of NKT Cell Modulators for the Treatment of High-Value Inflammatory, Fibrotic and Autoimmune Diseases

NKT Science

Leveraging Natural Killer T (NKT) regulation to target earlier in the inflammatory cascade to interrupt disease progression

High-Value Indications

Lead program entering Phase 2 for Idiopathic Pulmonary Fibrosis (IPF); Second program commencing Phase 1a/1b, initially targeting Systemic Lupus Erythematosus (SLE)

Proven Team

Drug development expertise; World renowned NKT cell researcher

Cash and Committed Capital[1] to Fund Planned Operations Through 2024, Including Multiple Clinical and Regulatory Expected Milestones

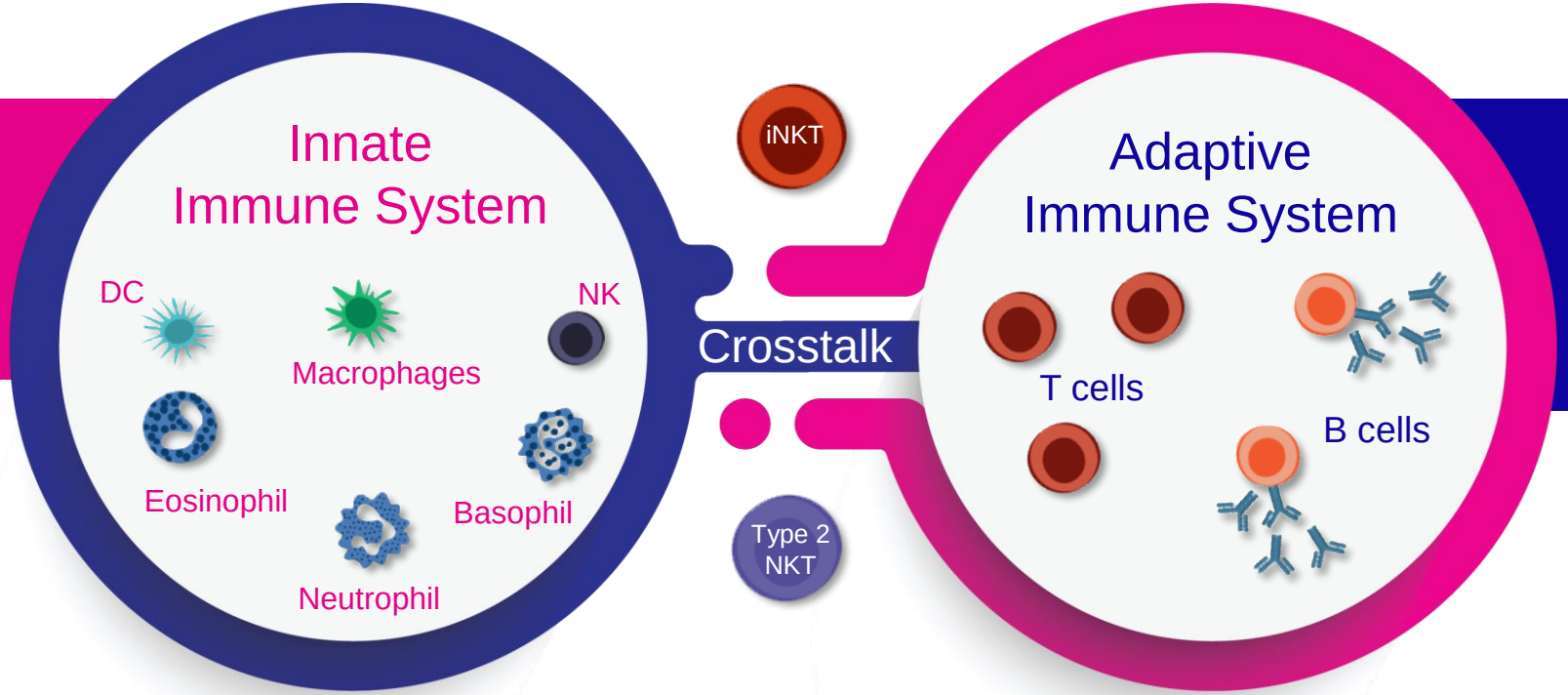
1. Future finding committed under the pre-merger financings.

NKT Cells for Immune Regulation

Novel Immune Mechanism to Regulate the Adaptive-Innate Immune Axis & Reset Dysfunctional Immune Responses

Innate Immunity

- Non-specific
- Fast to respond (hours)
- Activated by 'danger' signals
- First line of defense



Adaptive Immunity

- Specific
- Slow to respond (days)
- Activated by specific pathogen recognition
- Generates immune memory

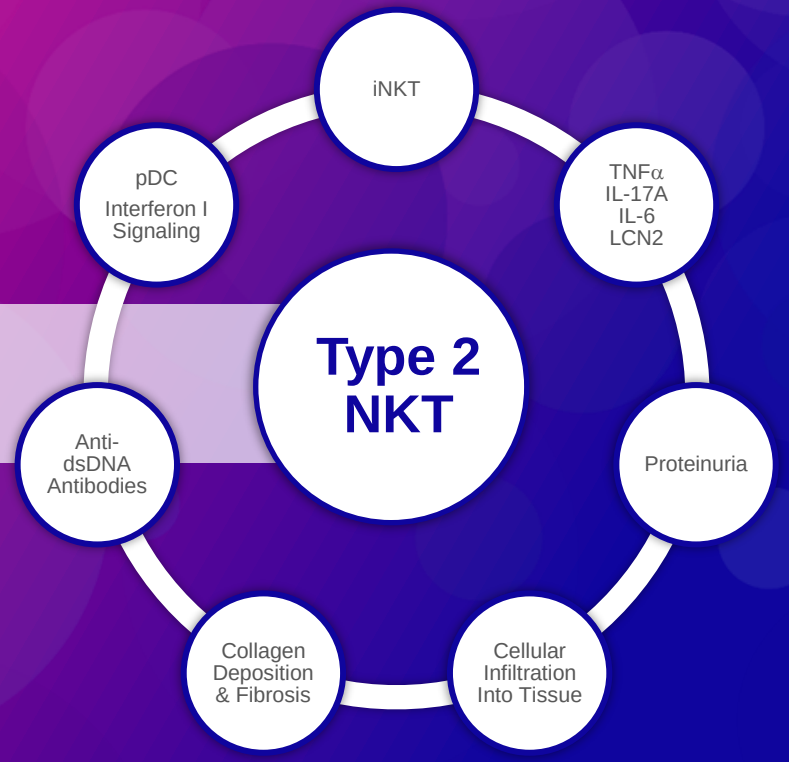
Regulating NKT Cells is a Selective Approach to Immunomodulation via Resetting the Immune Response

Natural Killer T Cells

Immune Cells that Bridge the Innate and Adaptive Immune Responses

Significantly Increased in Patients with Chronic Inflammatory Conditions

Two Types:
iNKT
and
Type 2 NKT

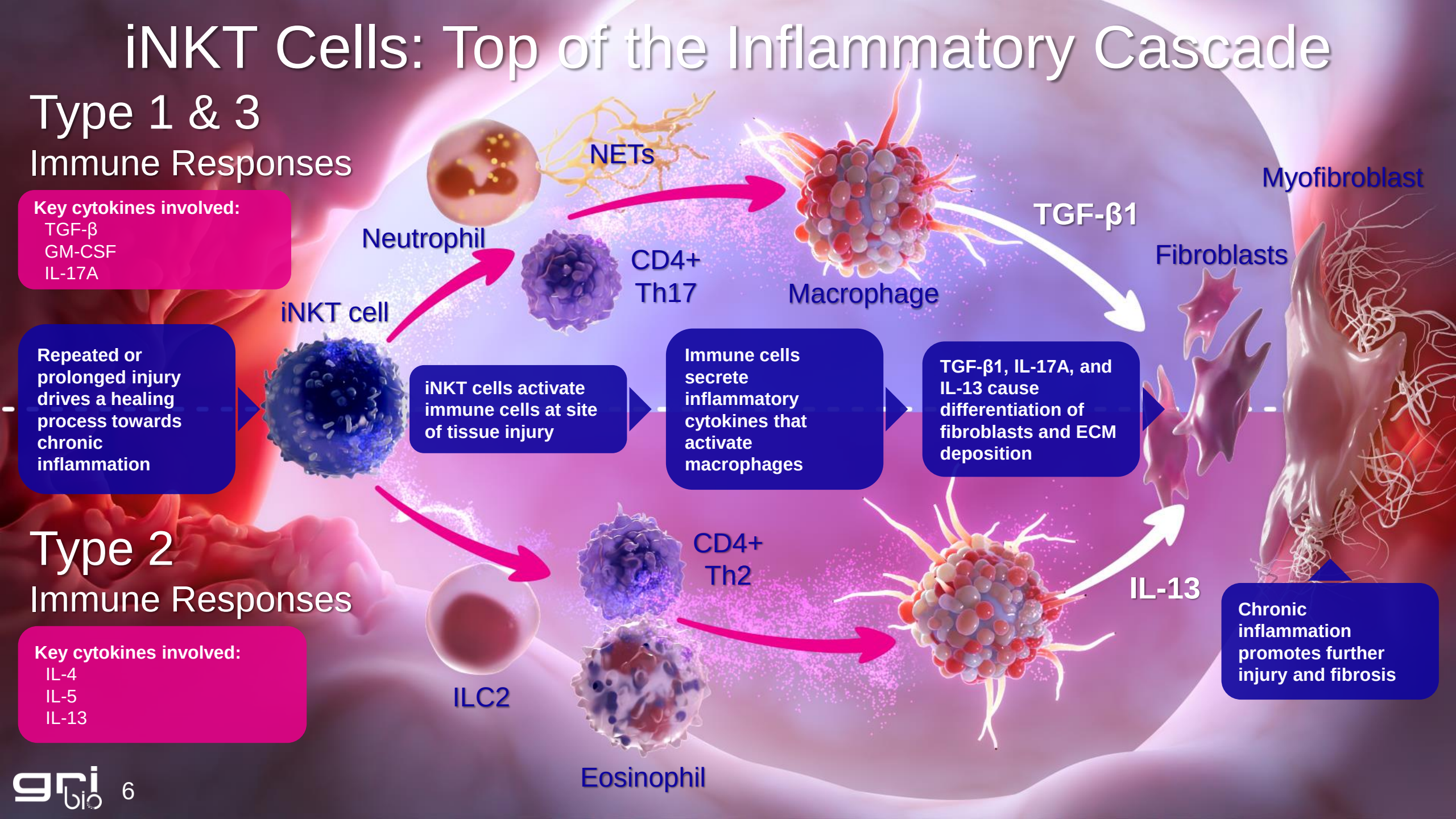


Regulate the Activity of Other Immune Cells and 'Reset' an Aberrantly Activated Immune Response

iNKT Cells: Top of the Inflammatory Cascade

Type 1 & 3 Immune Responses

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



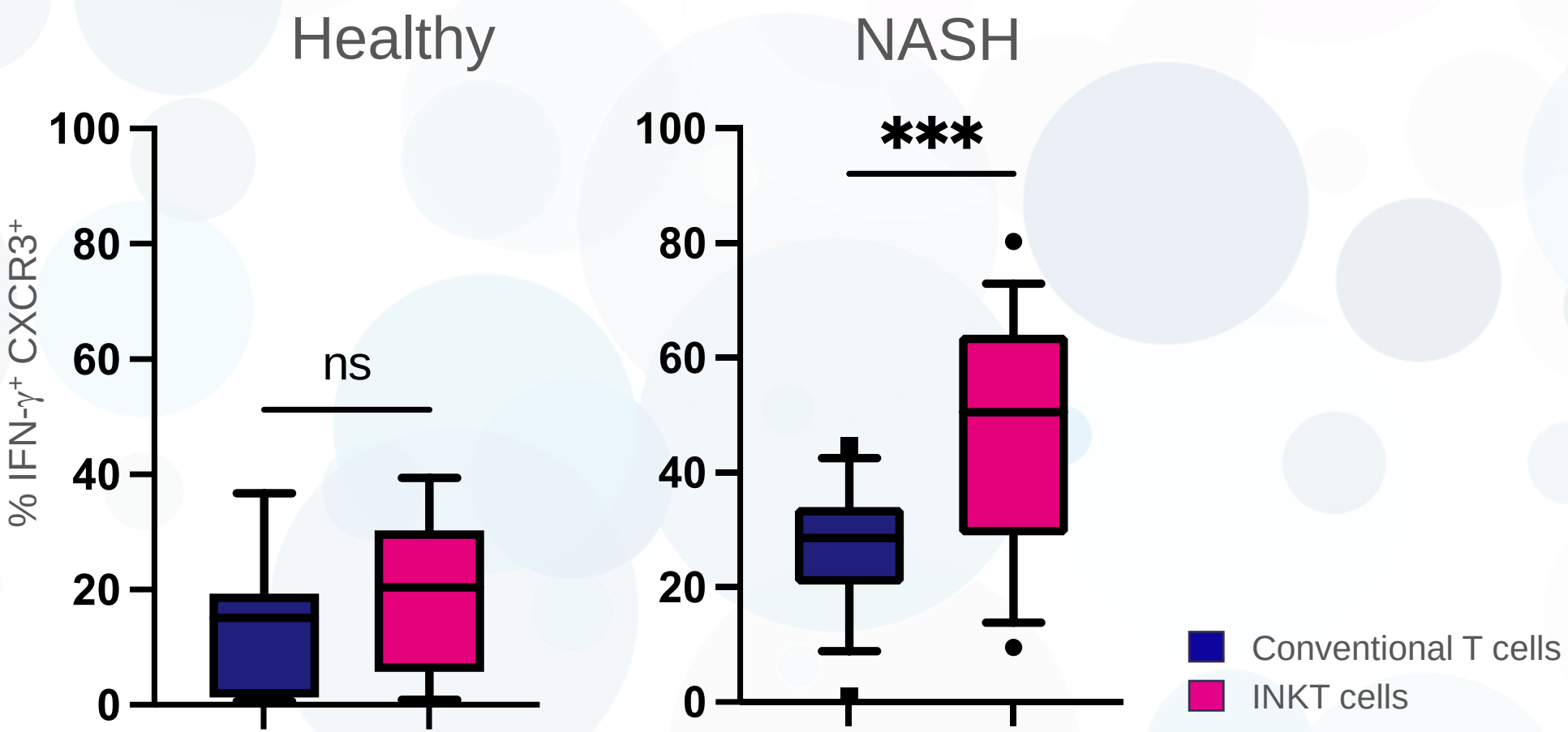
Type 2 Immune Responses

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

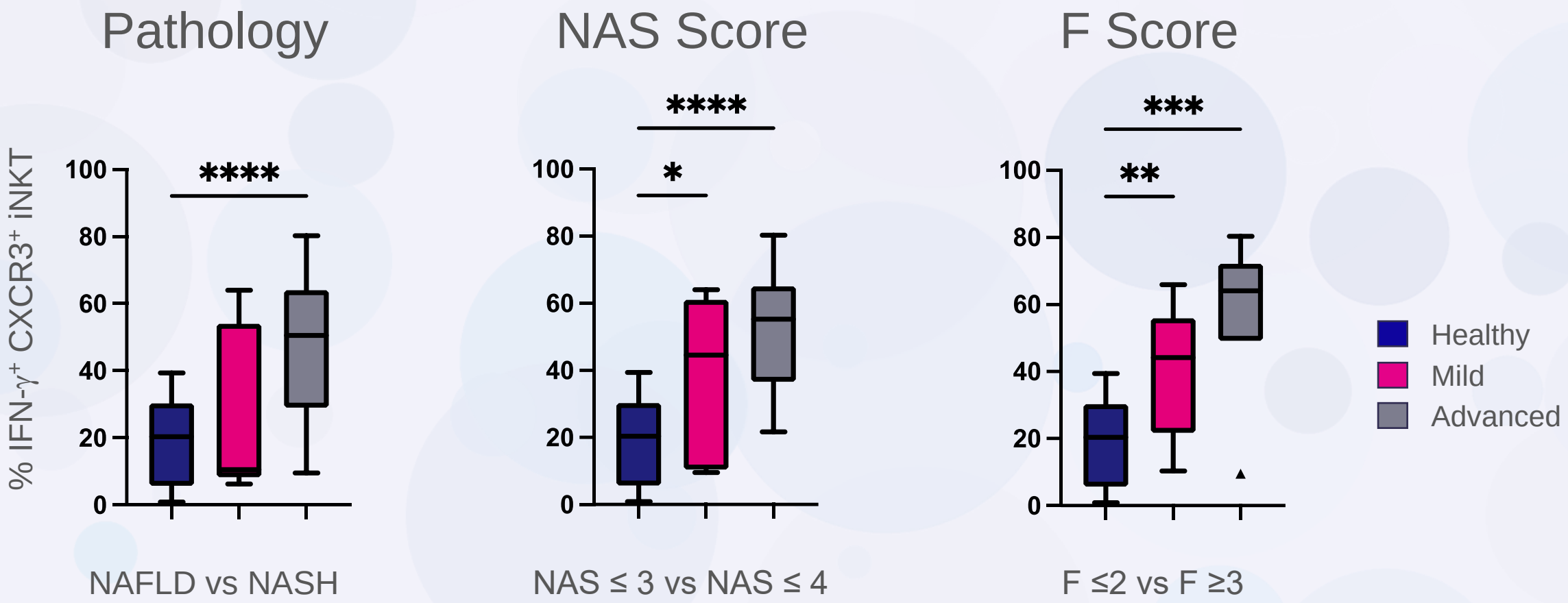
iNKT Cells Accumulate in Patients With Inflammatory & Fibrotic Disease

iNKT Promote Inflammation & Fibrosis	Disease Model	Patients
Drug-Induced Liver Injury (DILI)	Maricic ('15), Cheng, L. ('10), Mizrahi ('18), Krenkel ('14), Yang ('22),	
Ischemia Reperfusion Injury (IRI)	Arrenberg ('11), Uchida ('18), Ferhat ('18), Li ('07), Shimamura ('05), Zimmerman ('17), Lappas ('06), Sharma ('11), Wang ('16), Homma ('13)	
Sickle Cell Disease (SCD)	Wallace ('09)	Wallace ('09), Yu ('18)
Acute respiratory distress syndrome (ARDS)	Zou ('23)	
Hepatitis B or C Virus (HBV or HCV)	Jin ('11), Baron ('02)	Wei ('19), Durante-Mangoni ('04)
Autoimmune Hepatitis (AiH)	Halder ('07), Wu, S. J. ('11), 1176 Cheng, Z. ('22)	Harada ('03), Santodomingo-Garzon ('11), Mattner ('13), Chernavsky ('04), Smyk ('18)
Primary Biliary Cholangitis (PBC)	Chang ('15), Chuang ('08), Wu, S. J. ('11), Chang ('14), Schrumpf ('17)	Kita ('02), Aso-Ishimoto ('14), Ueno ('07)
Primary Sclerosing Cholangitis (PSC)	Schrumpf ('17), Berntsen ('18)	
Alcoholic Liver Disease (ALD)	Maricic ('15), Marrero ('20)	Marrero ('20)
Non-Alcoholic Steatohepatitis (NASH)	Akyildiz ('10), Bhattacharjee ('17), Heinrichs ('21), Maricic ('18), Ren ('17), Wolf ('14), Ishikawa ('11). Zhang ('23), Liao ('23)	Adler ('11), Heinrichs ('21), Maricic ('18), Syn ('12), Syn ('10), Wolf ('14)
Idiopathic Pulmonary Fibrosis	Grabarz ('18), Kumar (unpublished)	Byrne (unpublished)
Systemic Lupus Erythematosus (SLE)	Kumar (unpublished), Takahashi ('08), Forestier ('05), Morshed ('02), Zeng ('03)	Kumar (unpublished)
Multiple Sclerosis (MS)	Maricic ('14), Podbielska ('18)	De Biasi ('16), Wu, Q. ('23), Canto-Gomes ('23), He ('22), O'keeffe ('08), Florou ('21)

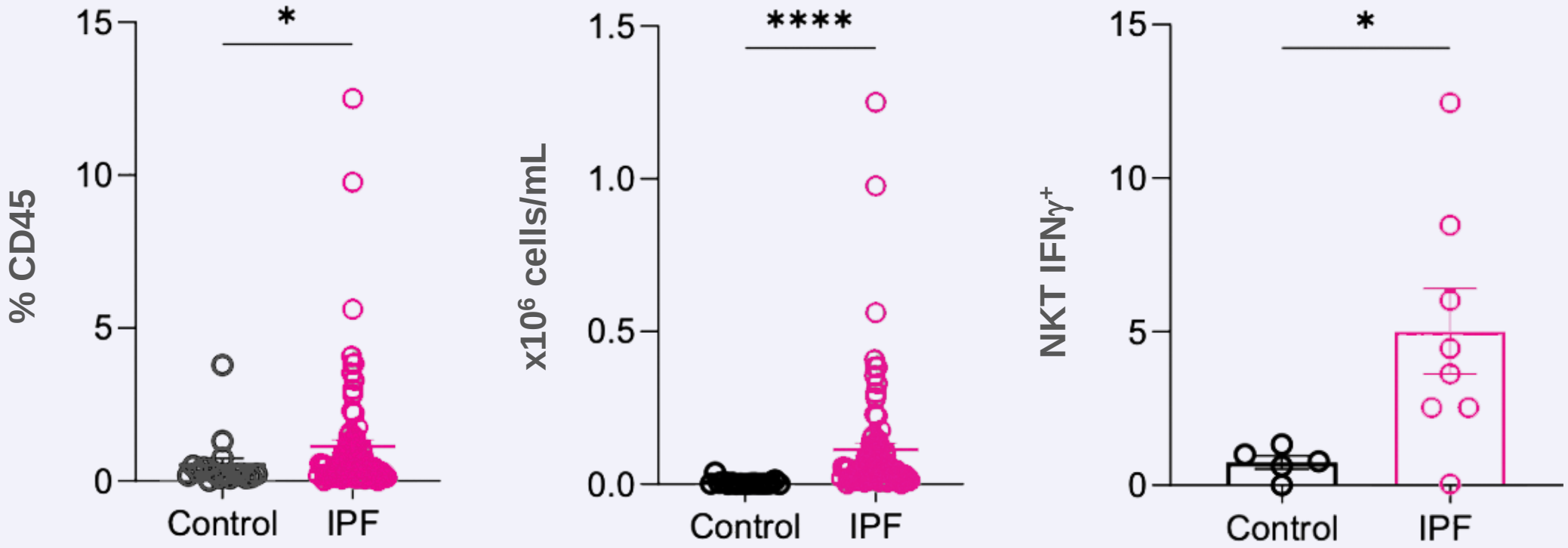
Pro-Inflammatory iNKT Cells Accumulate in Fatty Liver Disease Patients



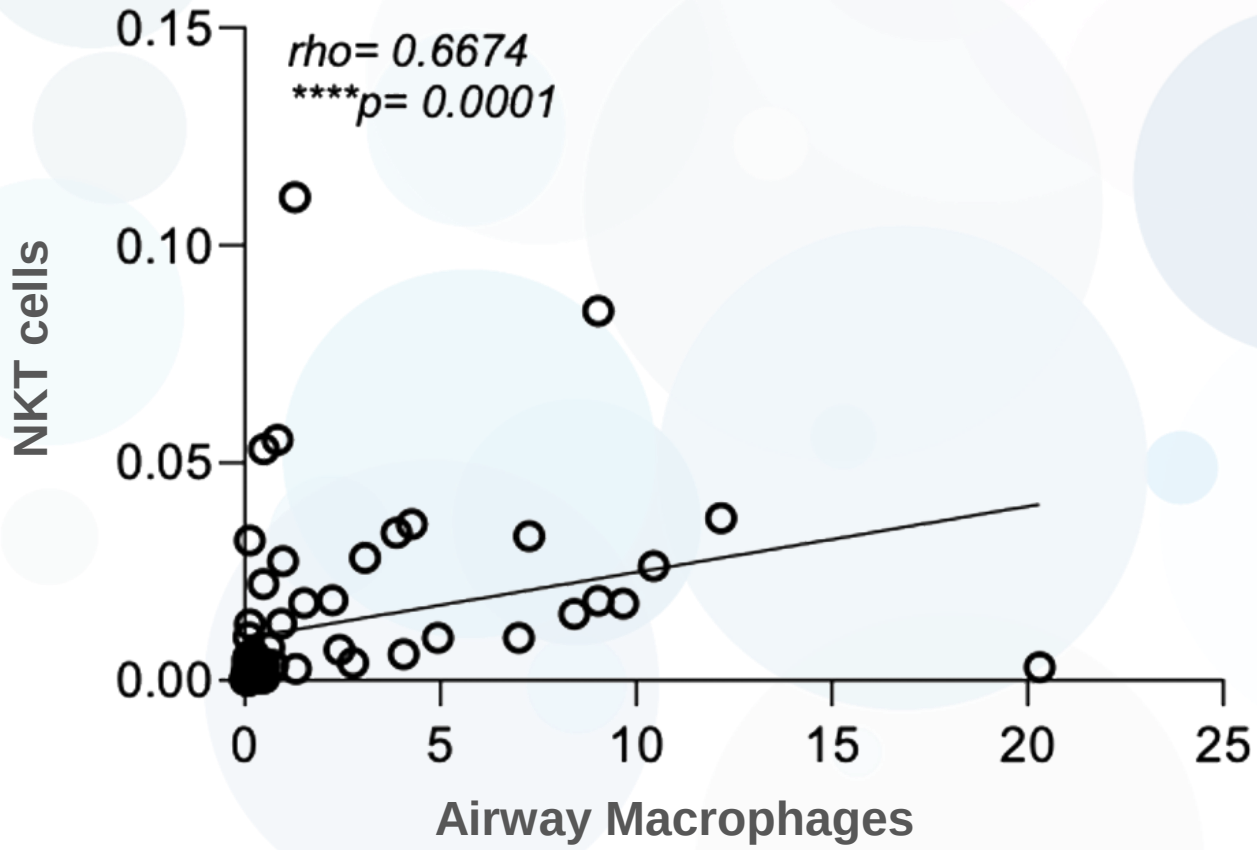
Pro-Inflammatory iNKT Cells Correlate with Progressive Advanced Disease



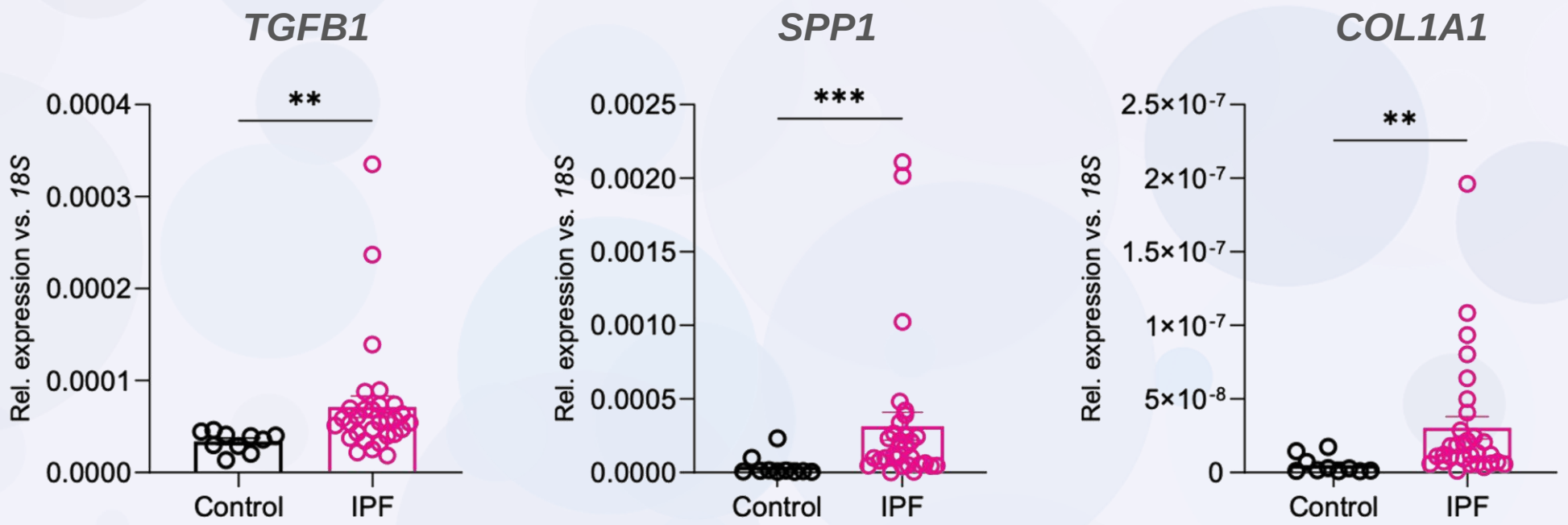
Proportion and Number of NKT Cells Significantly Increase in BAL of IPF Patients



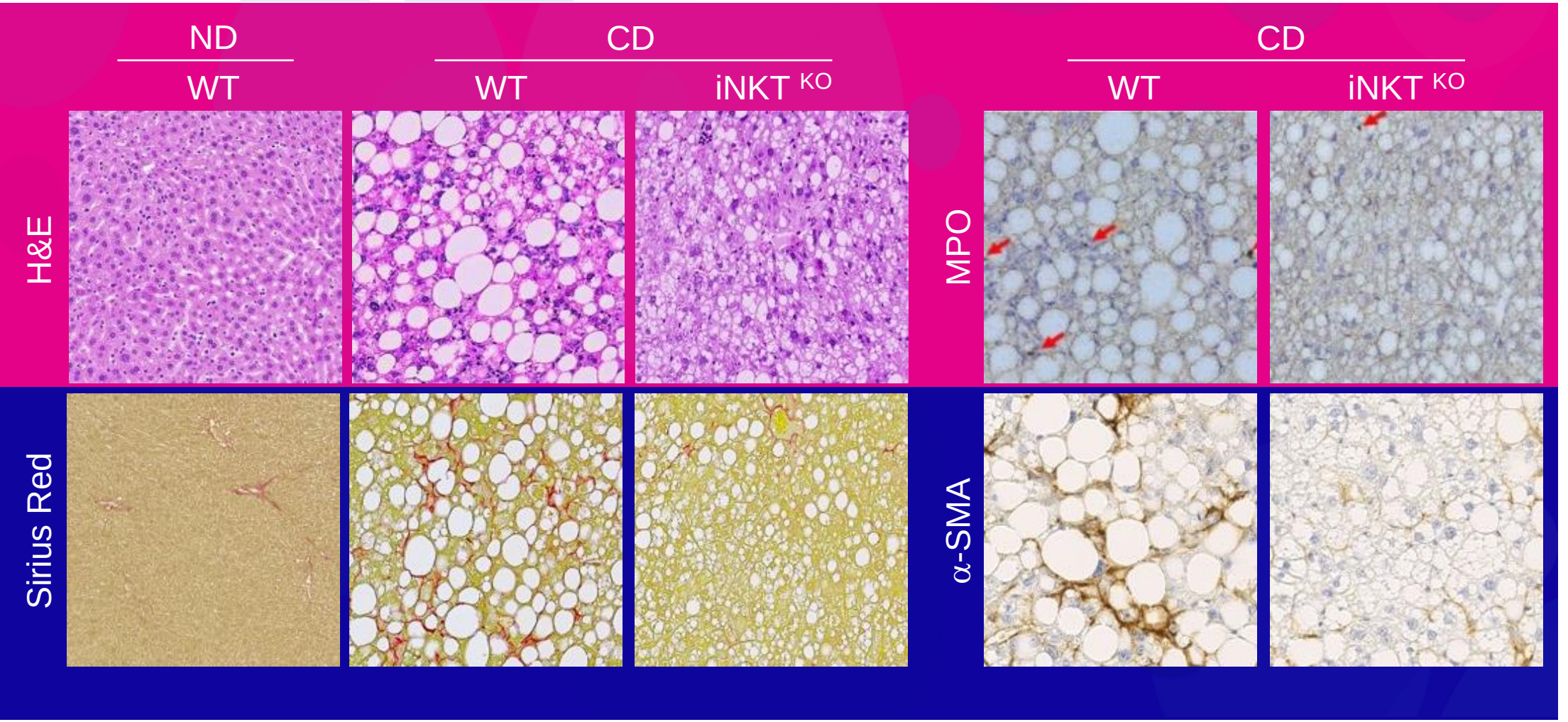
Total Number of NKT Cells Correlates with Total Number of Airway Macrophages in IPF Patients



Gene Expression of TGFβ1, Osteopontin, and Collagen Type 1 α1 Significantly increase in IPF Patients

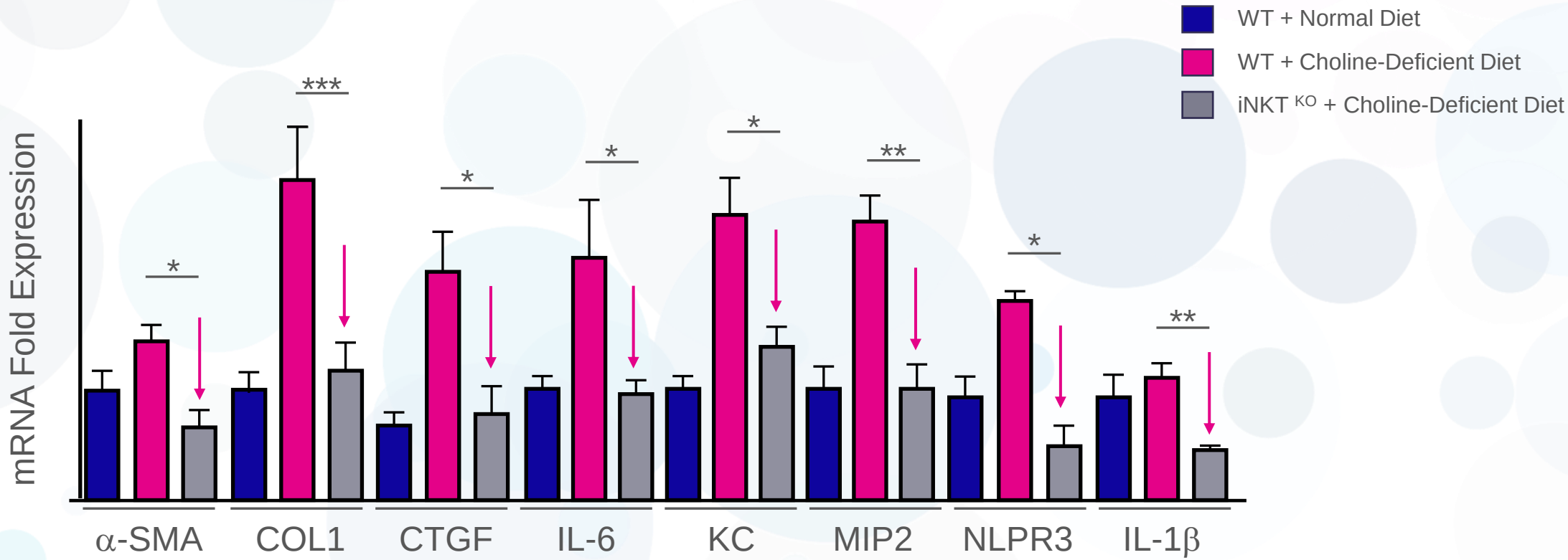


iNKT Cell Deficiency Prevents Inflammation, Steatosis & Fibrosis in NASH Model

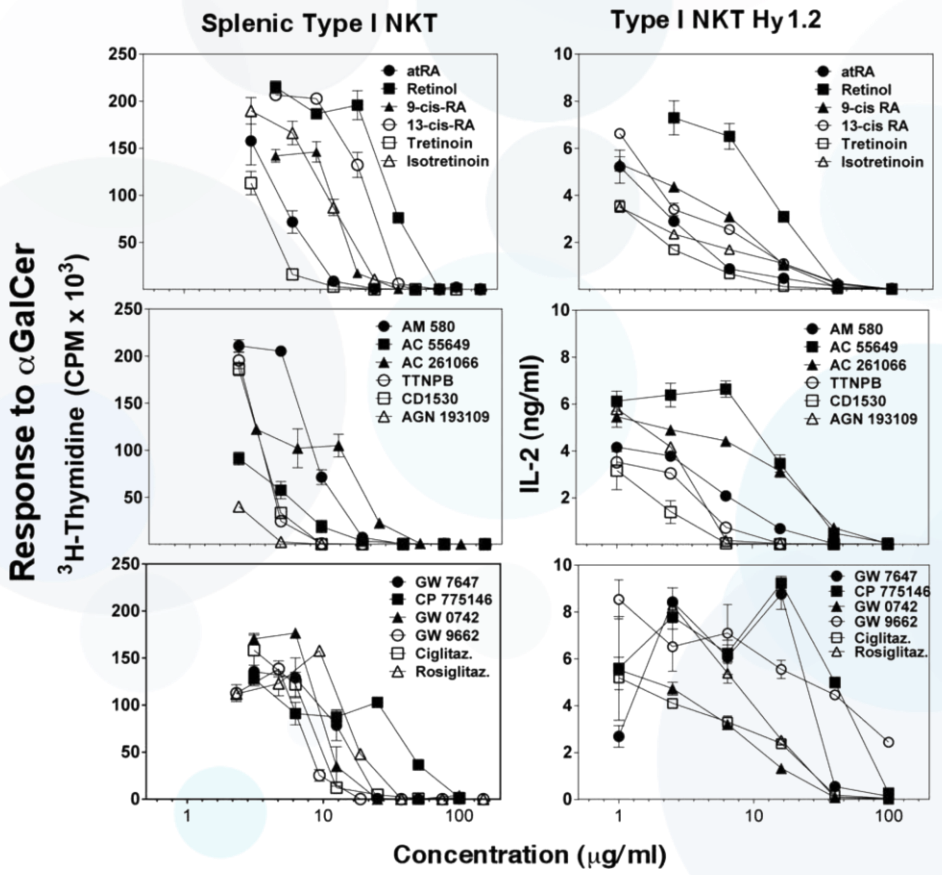


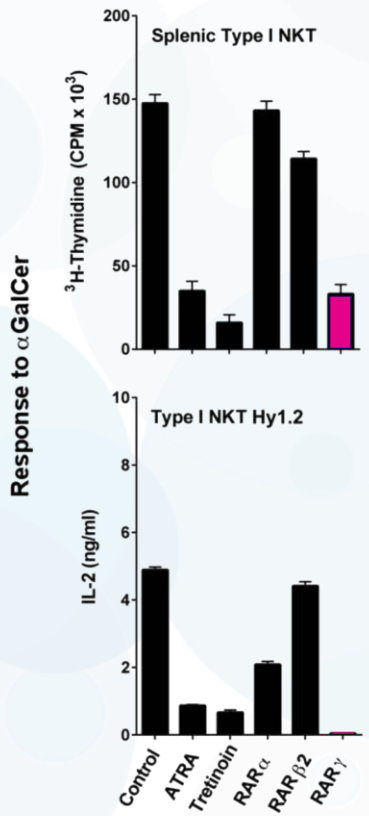
ND = Normal diet
CD = Choline-deficient diet
WT = Wild Type mice
iNKT ^{KO} = Jα18$^{-/-}$ iNKT deficient mice

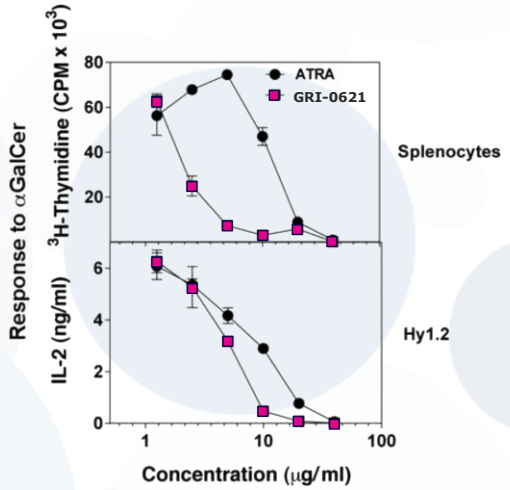
iNKT Cell Deficiency Inhibits Pro-Inflammatory & Fibrogenic Genes in a Hepatic Fibrosis Model

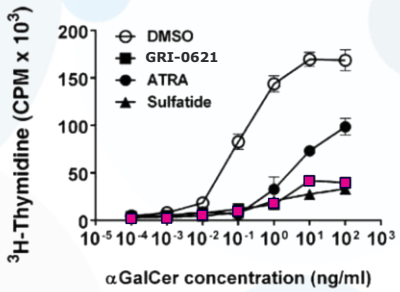


GRI-0621 is a Potent Inhibitor of iNKT Cell Activity

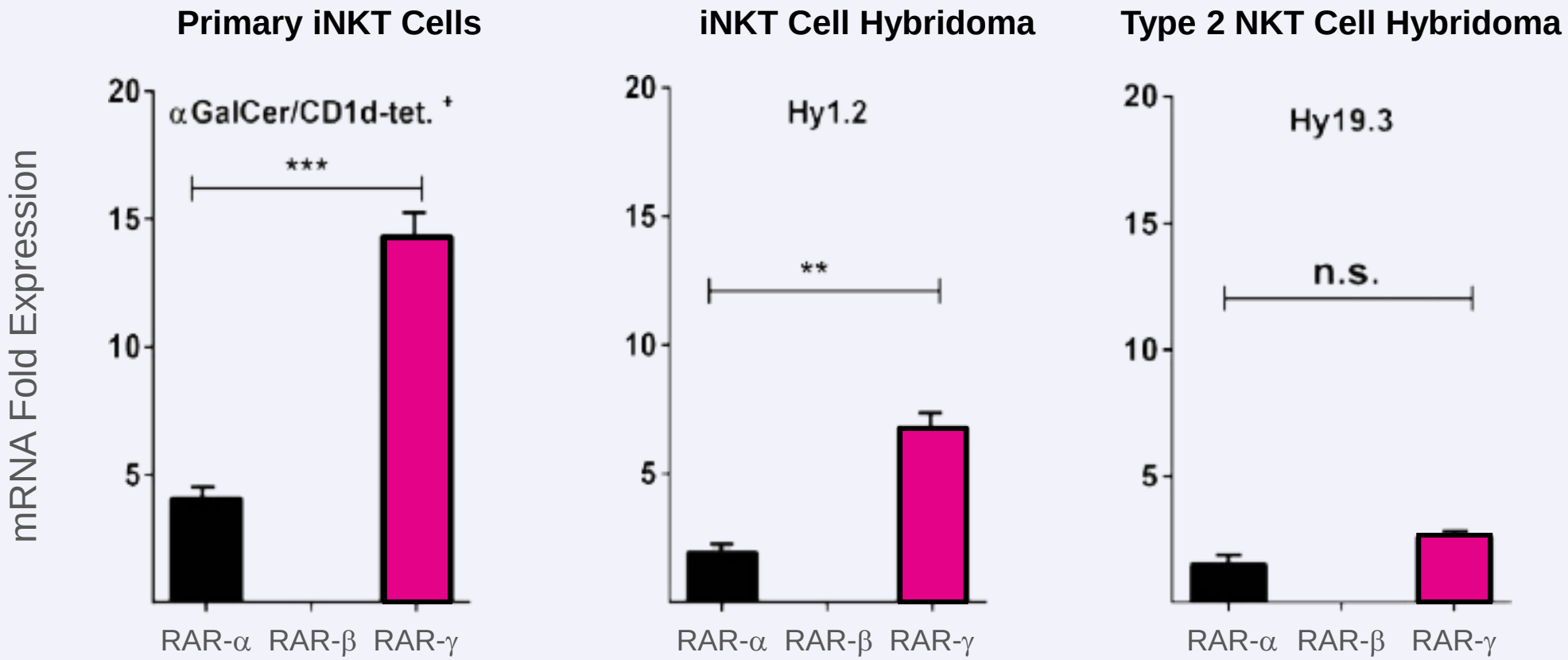






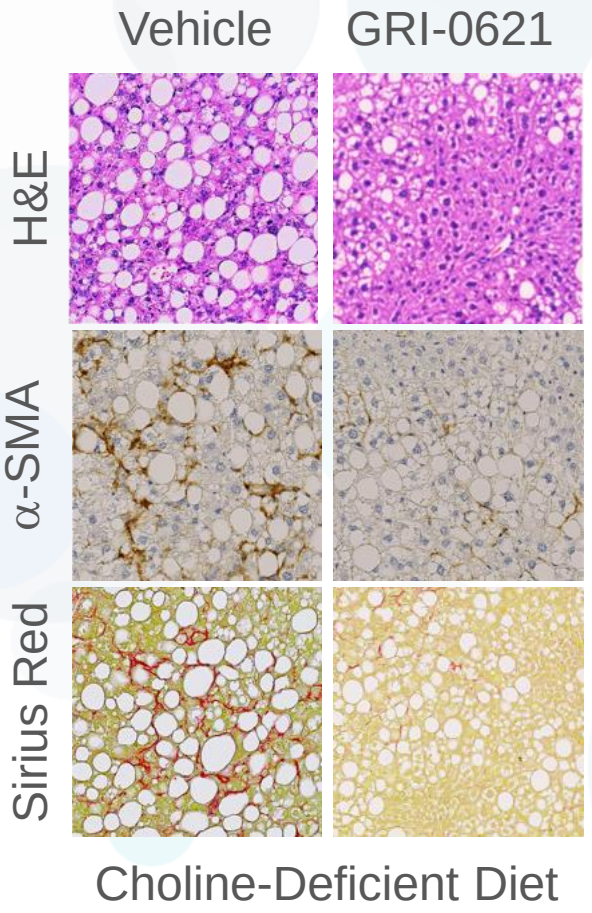


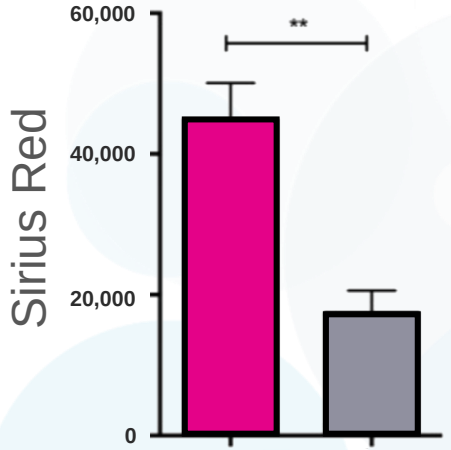
High Expression of RAR-γ on iNKT Cells

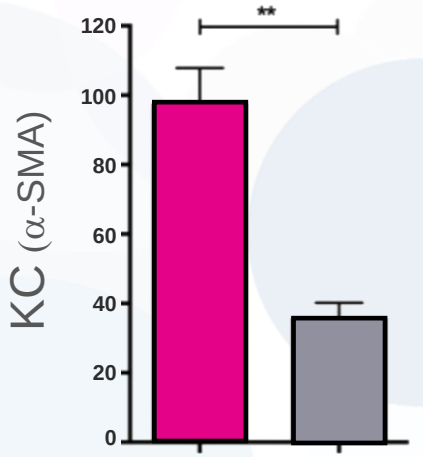


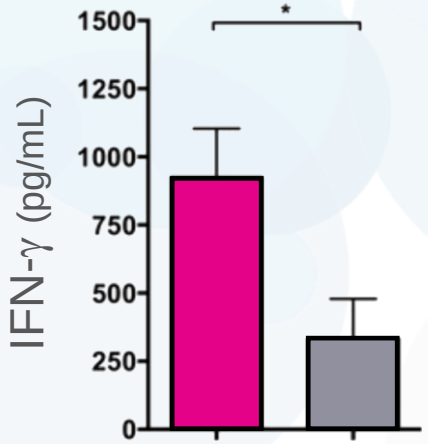
RAR-γ expression is low on type 2 NKT cells, conventional T cells, and B cells

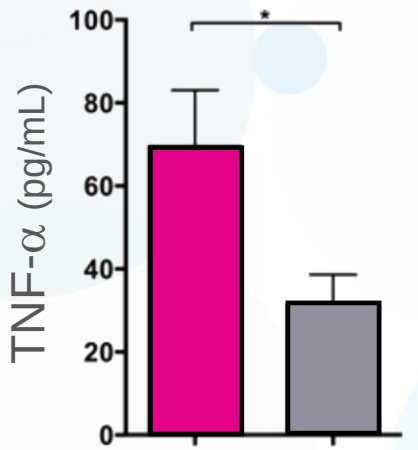
GRI-0621 Reduces Inflammation, Type 1 Cytokines and Reduces Hepatic Fibrosis



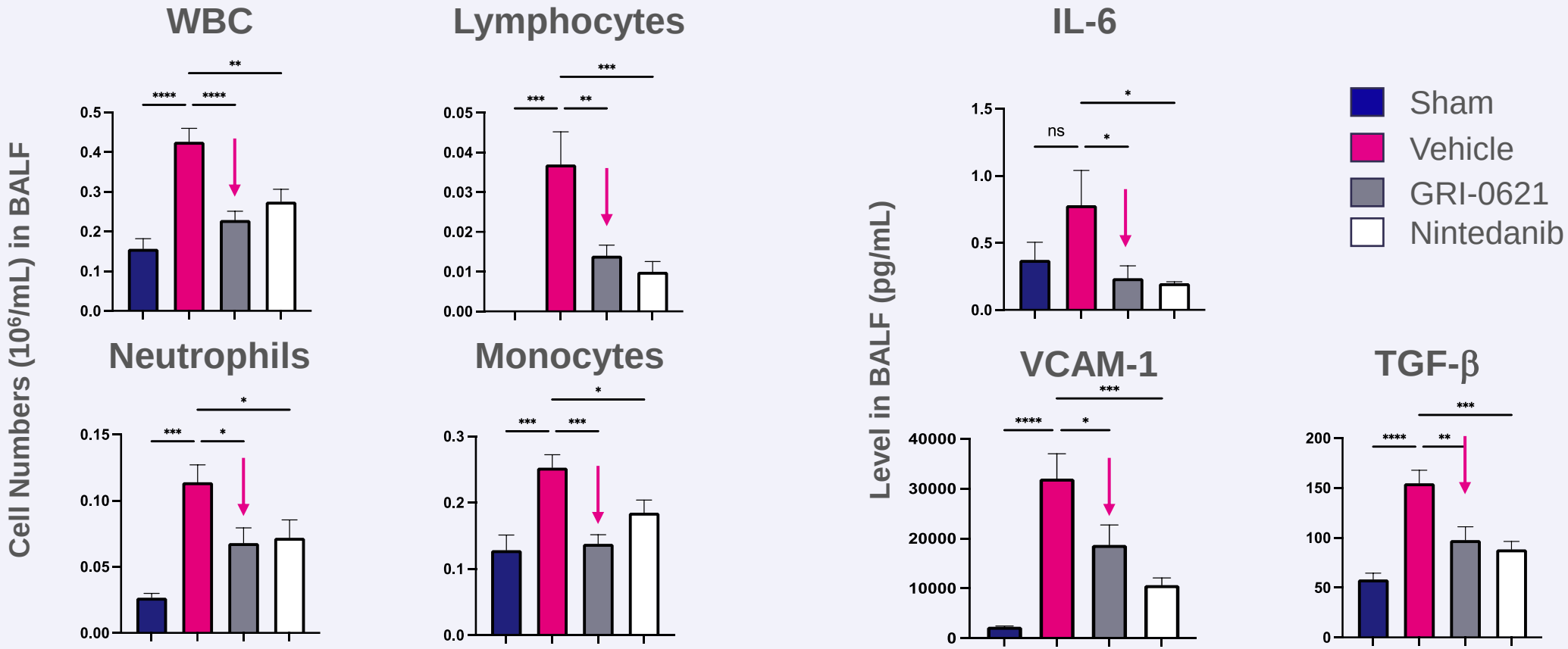








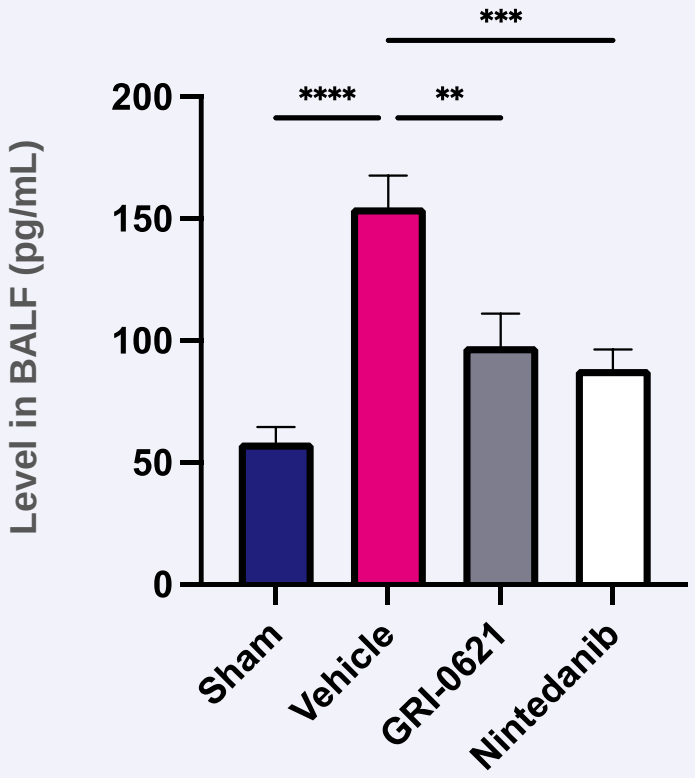
GRI-0621 reduces Inflammation & Pro-Inflammatory/Fibrotic Cytokines in Lung



GRI-0621 inhibits iNKT activity, reduces inflammation & pro-inflammatory cytokine production in BAL fluid

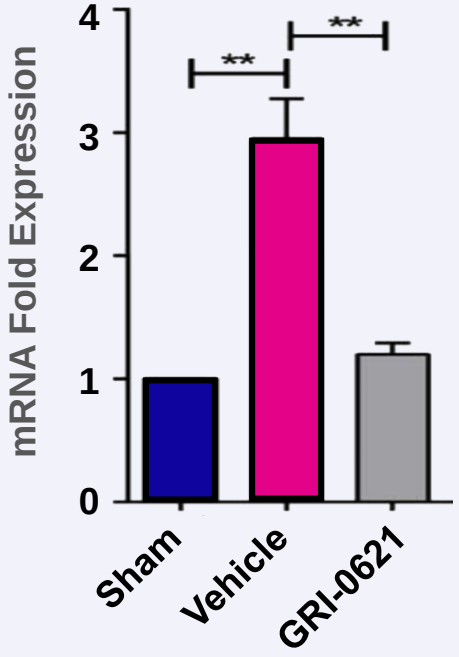
Observed Reduction of TGF-β in Fibrotic Models

Pulmonary Fibrosis



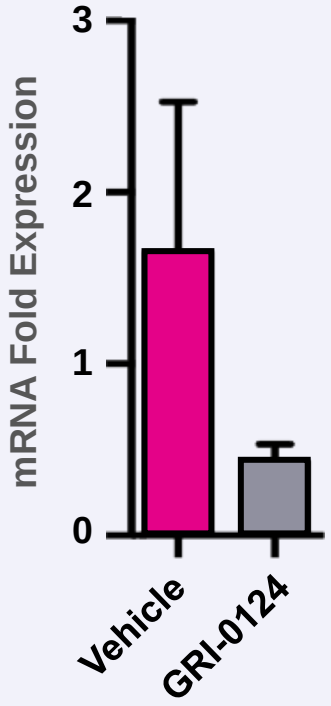
Bleomycin

Hepatic Fibrosis



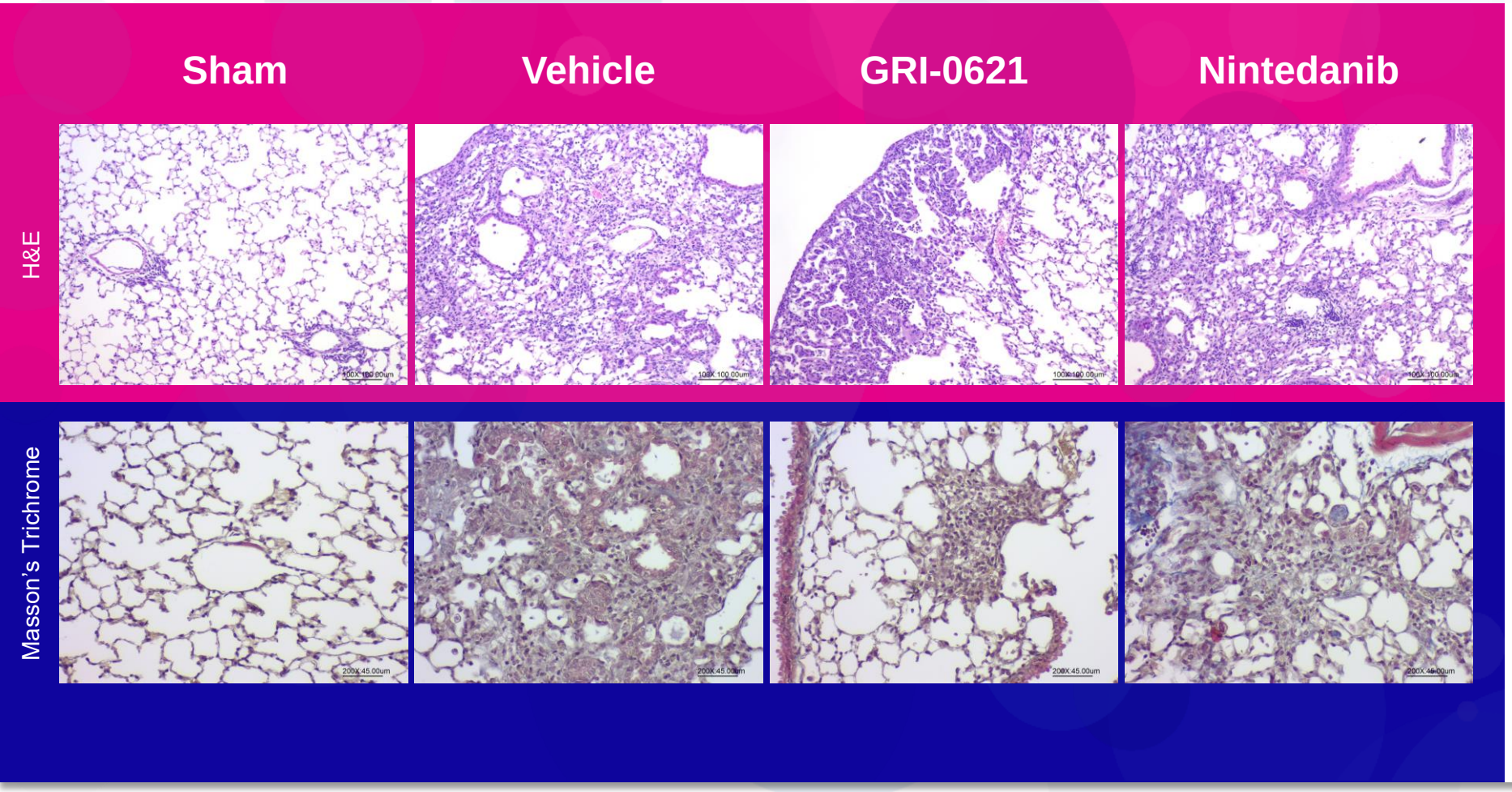
Lieber-DeCarli

Renal Fibrosis



NZB/W F1

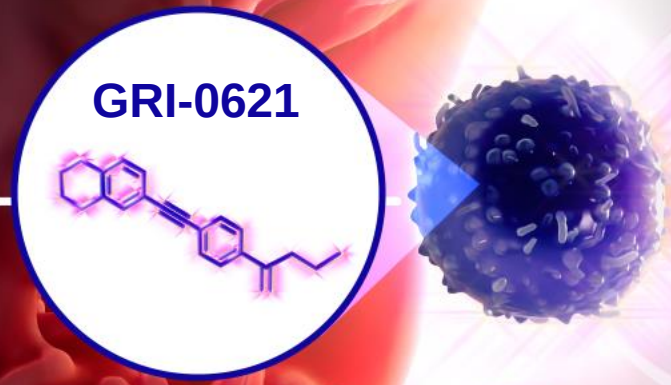
GRI-0621 Reduces tissue damage and Fibrosis in lung

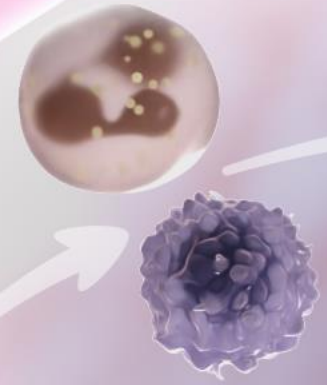


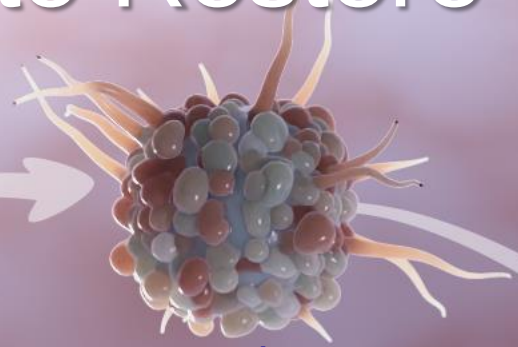
- Daily oral administration of the drug (a human equivalent dose) leads to reduced lung damage and a significant reduction in Bleomycin-induced lung fibrosis

GRI-0621 Targets iNKT to Restore Homeostasis

Type 1 & 3
Immune Responses







Macrophage

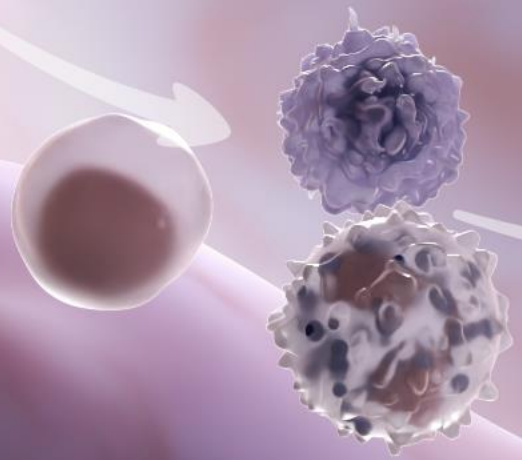
TGF-β1

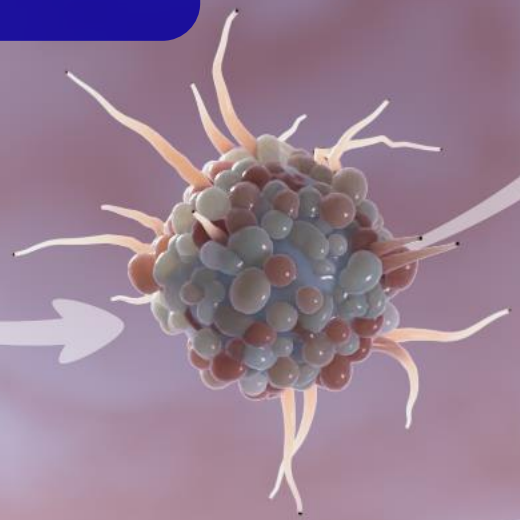
Most current therapies work through TFG-β regulation and fail to address type 2 immune responses

Fibroblasts

GRI-0621 inhibits the activity of iNKT cells early in the inflammatory cascade to prevent cytokine release, cellular infiltration, and interrupts disease progression at the source

Type 2
Immune Responses





IL-13

Resolution of chronic inflammatory response and immune system returning to homeostasis without systemic immunosuppression

GRI-0621

Idiopathic Pulmonary Fibrosis (IPF)

Launching Phase 2a biomarker study with interim data expected H1 2024 and topline data H2 2024

Leveraging FDA agreed 505(b)(2) regulatory pathway

Orphan indication with ~40K newly diagnosed cases annually[1]

1. Sauleda J, Núñez B, Sala E, Soriano JB. Idiopathic Pulmonary Fibrosis: Epidemiology, Natural History, Phenotypes. Med Sci (Basel). 2018 Nov 29;6(4):110. doi: 10.3390/medsci6040110. PMID: 30501130; PMCID: PMC6313500

Planned Phase 2 Study in IPF

Screening

Day ≤ -28
-4 weeks

Treatment

Day 1 to 84
12 weeks with planned interim analysis

Follow Up

Day 98
2 weeks

2 arms: 4.5 mg + placebo

Enroll 36 Patients in Phase 2 IPF Trial

Patients: 36 IPF patients on background IPF therapy

Dosing: 4.5mg and placebo dosed orally 1x daily for 12 weeks

Design: 2 arm RCT 2:1 randomization (24:12)

Interim Analysis: when 8 of 12 placebo treated patients have completed 6 weeks of treatment

Endpoints

Primary Endpoint:

Safety, percent inhibition iNKT in blood (PBMC) at 6 and 12 weeks and lung (BAL fluid) at 12 weeks

Exploratory endpoints:

Changes in serum biomarkers at 6 and 12 weeks (collagen degradation biomarkers, cytokines, NKT); FVC at 6 and 12 weeks

Summary of iNKT cell involvement in Fibrosis

- iNKT cells have an activated phenotype in NASH & IPF patients
- Enhanced iNKT activity correlates with progression of fibrosis in NASH patients and
- with macrophage accumulation and key proinflammatory genes in BAL from IPF patients
- iNKT cells are activated and accumulate in liver and lung in experimental fibrosis models
- iNKT promote Type 1, Type 2 and Type 3 immune pathways involved in fibrosis
- iNKT-deficient mice have reduced inflammatory damage and fibrosis
- Daily oral administration of GRI-0621 in experimental animals
  - Inhibits pro-inflammatory cytokines and inflammation
  - Decreases accumulation of neutrophils and proinflammatory macrophages
  - Inhibits key fibrogenic cytokines including TGF-b and fibrosis
- Phase 2 study with GRI-0621 in IPF patients to examine iNKT activity along with key biomarkers

A New Approach to
Inflammatory Diseases

Thank You!

Investor Relations
JTC Team
833-475-8247
gri@jtcir.com