UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 10, 2022

PLIANT THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-39303**	**47-4272481**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**
260 Littlefield Avenue, South San Francisco, CA		**94080**
(Address of Principal Executive Offices)		**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (650) 481-6770

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.0001 per share	PLRX	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Other Events.

Pliant Therapeutics, Inc. (the "Company") intends to conduct meetings with securities analysts, investors and others in connection with the 40th Annual J.P. Morgan Healthcare Conference beginning on January 10, 2022. As part of these meetings, the Company intends to utilize the corporate slide presentation furnished with this report as Exhibit 99.1.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Corporate Slide Presentation dated January 10, 2022, titled "Developing Novel Therapeutics for Fibrotic Diseases."
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

The information in this Item 7.01 is being furnished, not filed, pursuant to Regulation FD. Accordingly, the information in Item 7.01 of this report will not be incorporated by reference into any registration statement filed by the Company under the Securities Act of 1933, as amended, unless specifically identified therein as being incorporated therein by reference. The furnishing of the information in this report is not intended to, and does not, constitute a determination or admission by the Company that the information in this report is material or complete, or that investors should consider this information before making an investment decision with respect to any security of the Company.

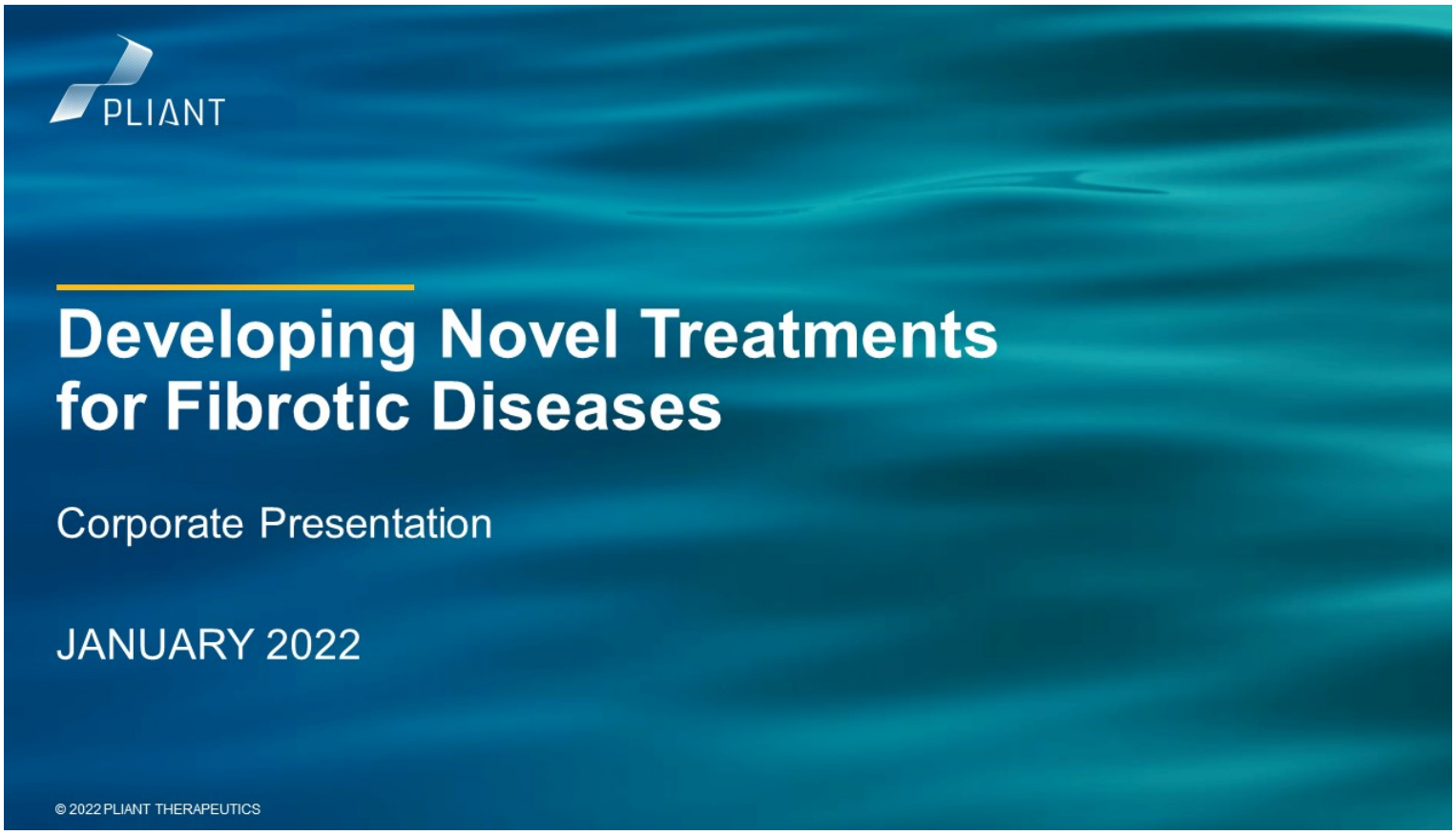
SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

PLIANT THERAPEUTICS, INC.

Date: January 10, 2022

By: /s/ Keith Cummings
Keith Cummings, M.D., MBA
Chief Financial Officer


PLIANT
Developing Novel Treatments for Fibrotic Diseases
Corporate Presentation
JANUARY 2022
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Disclaimers

This presentation has been prepared by Pliant Therapeutics, Inc. ("we," "us," "our," "Pliant" or the "Company"). The information set forth herein does not purport to be complete or to contain all of the information you may desire. Statements contained herein are made as of the date of this presentation unless stated otherwise, and this presentation shall not under any circumstances create an implication that the information contained herein is correct as of any time after such date or that information will be updated or revised to reflect information that subsequently becomes available or changes occurring after the date hereof.

This presentation includes forward-looking statements regarding Pliant's proprietary drug candidates, the timing of the start and conclusion of ongoing or planned clinical trials, including the timing of, and our ability to achieve, anticipated milestones, the sufficiency of our cash, cash equivalents and short-term investments, the timing and outcome of regulatory decisions, future availability of clinical trial data, our collaborations for our product candidates and the maintenance of those collaborations; business and results from operations; and other matters. Actual results could differ materially from those contained in any forward-looking statements as a result of various factors, including without limitation: that Pliant's drug candidates do not advance in development or result in approved products on a timely or cost effective basis or at all; the cost, timing and results of clinical trials; our ability to manage and mitigate the impact of the ongoing COVID-19 pandemic; that many drug candidates that have completed early-stage trials do not become approved drugs on a timely or cost effective basis or at all; the ability to enroll patients in clinical trials; possible safety and efficacy concerns; regulatory developments; the ability of Pliant to protect its intellectual property rights, and unexpected costs, charges or expenses that reduce cash runway. Pliant's pipeline programs are in various stages of pre-clinical and clinical development, and the process by which such pre-clinical or clinical therapeutic candidates could potentially lead to an approved therapeutic is long and subject to significant risks and uncertainties. Pliant undertakes no obligation to update forward-looking statements as a result of new information or otherwise. For a discussion of these and other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" and elsewhere in the Company's most recent Annual Report on Form 10-K and Quarterly Report on Form 10-Q on file with the Securities and Exchange Commission (the "SEC") and our other filings with the SEC.

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 involves 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.

This presentation concerns drugs that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration (the "FDA"). They are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Pliant – Company Highlights

Industry-Leading Fibrosis Platform

- Built on integrin-mediated inhibition of TGF-β pathway resulting in antifibrotic effect and shown to be safe
- Proprietary drug discovery platform based on novel in-house compound library of integrin binders
- Lead molecule PLN-74809 is highly antifibrotic in lung and liver while well tolerated at highest doses tested

Programs Targeting High Unmet Medical Need with High-Impact, Near-Term Catalysts

- PLN-74809 in Phase 2a development in IPF and PSC
  - Phase 2a topline data in IPF expected mid-2022
  - Significant clinical derisking: target engagement (PET) and TGF-β pathway inibition (pSmad)
- IND submissions in oncology and muscular dystrophies expected by YE 2022

Strategic Partnership with Novartis Validates Platform

- Largest ($80M) upfront for a preclinical NASH program
- Significant expense offset to pipeline programs
- Broad multi-target research collaboration
  - Next generation anti-fibrotic molecules targeting novel integrins

Strong Financial Position

- Over $385 million raised to date in four financing rounds including June 2020 IPO (Nasdaq: PLRX)
- $221 million cash[1] balance as of September 30, 2021
- Company funded into 2H 2023

1 - Includes cash, cash equivalents and ST investments.

Recent Company Highlights

- **PLN-74809 Phase 2a INTEGRIS-IPF trial enrollment complete**
  - PLN-74809 has been administered to over 450 subjects to date and shown to be well tolerated
  - INTEGRIS-IPF topline data expected mid-2022
- **Positive interim data from PLN-74809 PET imaging target engagement study**
  - PLN-74809 showed target engagement up to 98% in lungs of IPF patients
  - All doses tested achieved target engagement above threshold for predicted antifibrotic activity
- **FDA authorized evaluation of long-term treatment with PLN-74809 at doses up to 320 mg in IPF**
  - No safety concerns identified to date at doses up to 640 mg
  - No treatment-related effects in chronic GLP tox, NOAEL set at the highest dose tested
  - Expected 1H 2022 initiation of 6-month Phase 2a trial of PLN-74809 at 320 mg in IPF patients
- **PLN-74809 Phase 2a INTEGRIS-PSC trial on track for full enrollment by mid-2022**
  - INTEGRIS-PSC topline data expected late 2022 / early 2023
- **Early-stage programs in Oncology and DMD advancing toward IND**
  - INDs expected in both indications by YE 2022

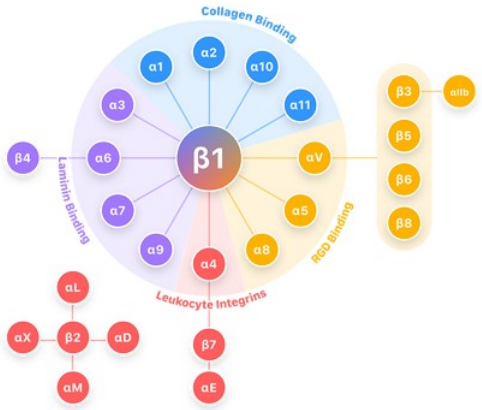
Pliant's Integrin Focused Library
Core Platform for Novel Pipeline and Partner Programs

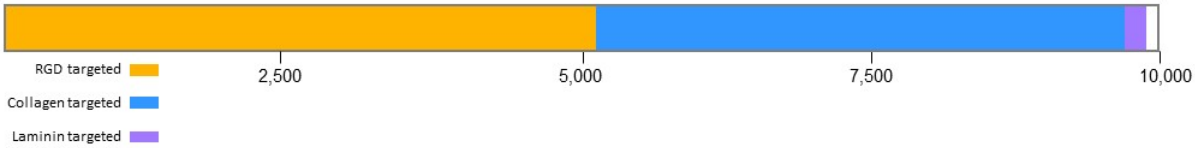
Integrins

- Cell surface receptors that facilitate cell-cell and cell-extracellular matrix adhesion and interaction
- A major path of communication between the extracellular matrix, inflammatory cells, fibroblasts
- Closely involved in signaling processes governing tissue fibrosis

Pliant's proprietary library of integrin binding compounds

- Emphasis on structural diversity
- Broad spectrum of receptor subfamilies including α_V integrins, collagen and laminin binders





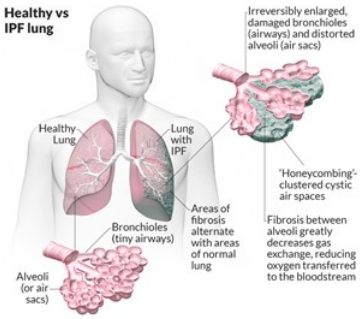
Pliant Development Pipeline

	Program	Indication	Preclinical	Clinical			Anticipated Milestone	Global Rights
				Phase I	Phase II	Phase III		
WHOLLY OWNED	PLN-74809 Dual selective inhibitor of $\alpha_V\beta_6/\alpha_V\beta_1$	Idiopathic Pulmonary Fibrosis		INTEGRIS-IPF Enrollment Complete			Phase 2a Topline Data Expected Mid-2022	PLIANT
		Primary Sclerosing Cholangitis					Phase 2a Enrollment Complete Expected Mid-2022	PLIANT
	Oncology Inhibitor of $\alpha_V\beta_8$	Solid Tumors					IND Filing Expected YE 2022	PLIANT
	Muscular Dystrophies Anti-integrin mAb	DMD Other Muscular Dystrophies					IND Filing Expected YE 2022	PLIANT
PARTNERED	PLN-1474 Selective inhibitor of $\alpha_V\beta_1$	NASH-Associated Liver Fibrosis					Phase 2 Initiation	NOVARTIS

Fibrosis: The Silent Killer

Idiopathic Pulmonary Fibrosis (IPF) is a lethal pathological process with limited therapeutic options

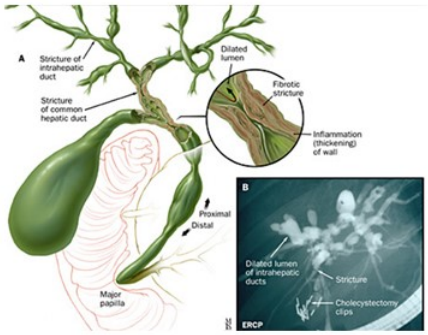
- 140k patients in the U.S.; 30k-40k new cases/year; 40k deaths/year
- **Median survival: 3–5 years -** Worse than some common cancers
- 2 FDA approved therapeutics generate **annual revenues >$3.6 billion** despite significant remaining unmet medical need



https://www.lungsandyou.com/ipf

Primary Sclerosing Cholangitis (PSC) is a progressive inflammatory liver disease resulting in scarring of bile ducts, and cirrhosis

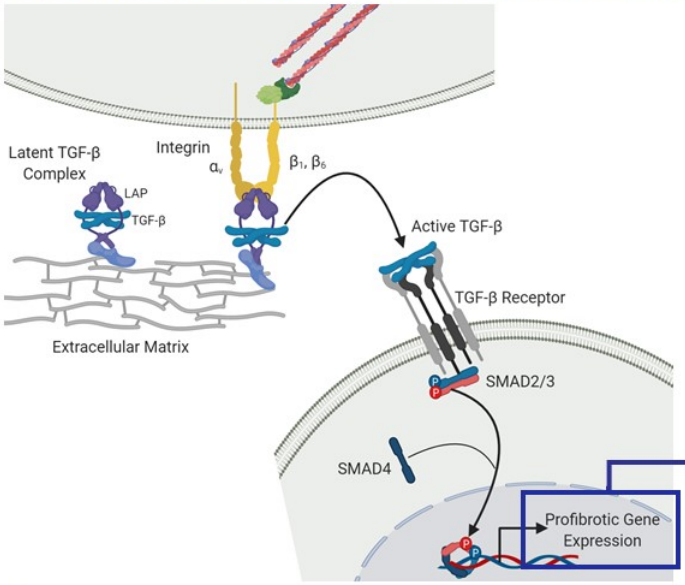
- Currently **no FDA approved therapeutics**
- 30k-45k patients in the U.S.
- **Median survival: 10-12 years** without intervention



www.jhmicall.org

$\alpha_V\beta_6$ / $\alpha_V\beta_1$ Integrins Drive Cell-Matrix Interactions in Fibrosis

$\alpha_V\beta_6$ / $\alpha_V\beta_1$ Integrins promote fibrosis by TGF-β activation

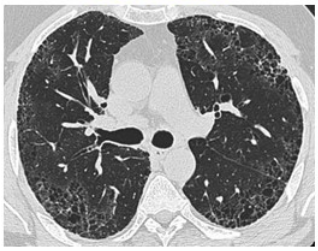


- **TGF-β is central mediator of fibrosis**
- **$\alpha_V\beta_6$/$\alpha_V\beta_1$ Integrins activate latent TGF-β only in fibrotic tissue**
- **Systemic TGF-β blockade carries toxicity risks**

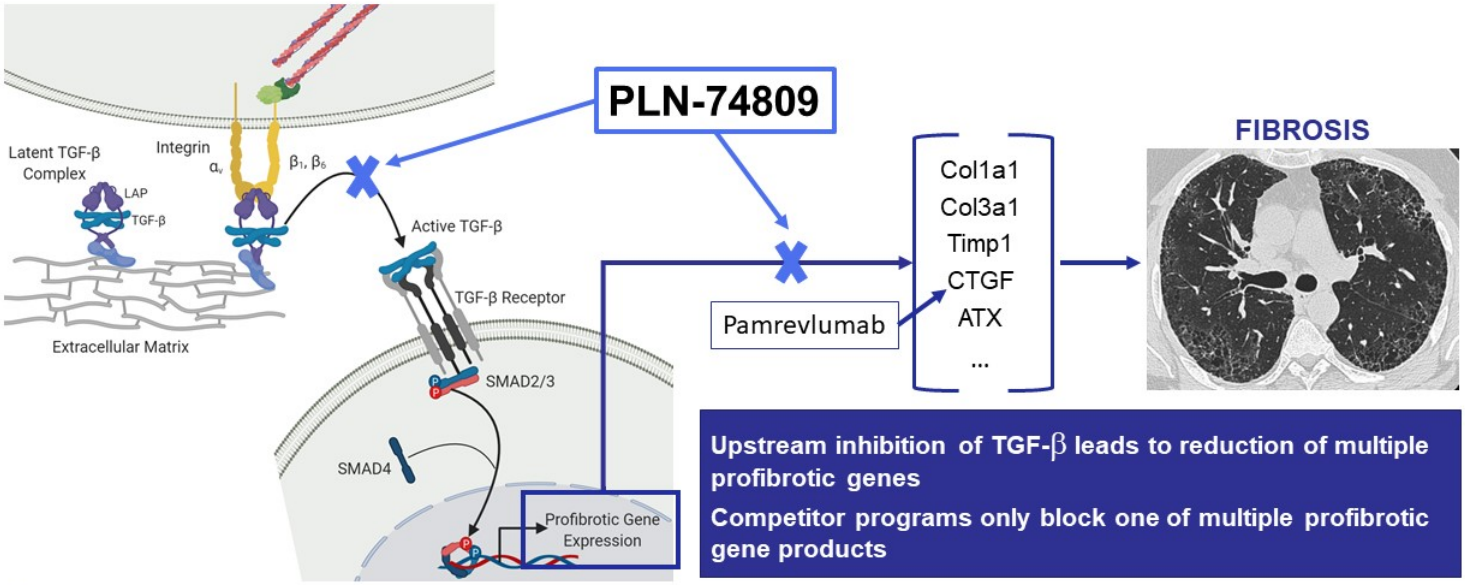
Selectively blocking TGF-β in fibrotic tissues may provide a low risk, effective antifibrotic approach

Col1a1
Col3a1
Timp1
CTGF
ATX
…

FIBROSIS



PLN-74809 Provides Profound Antifibrotic Activity through Upstream Inhibition of TGF-β Activation



Upstream inhibition of TGF-β leads to reduction of multiple profibrotic genes

Competitor programs only block one of multiple profibrotic gene products

Pliant Compounds Have Not Shown Adverse Effects of Systemic Inhibition of TGF-β Pathways[1]

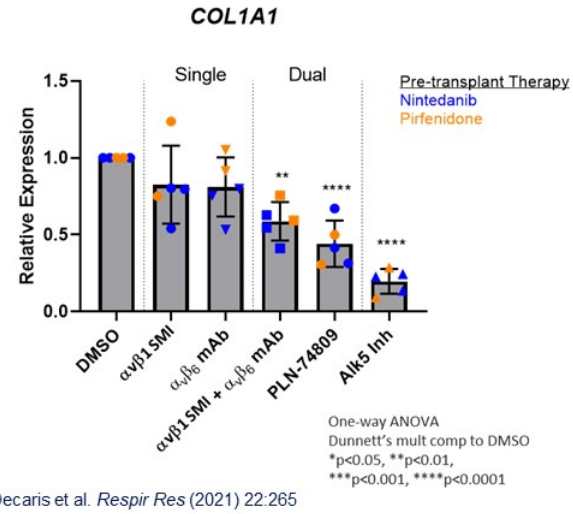
By targeting integrins that are upregulated specifically in fibrotic tissues, Pliant's small molecule compounds may avoid toxicities associated with systemic TGF-β blockade[1]

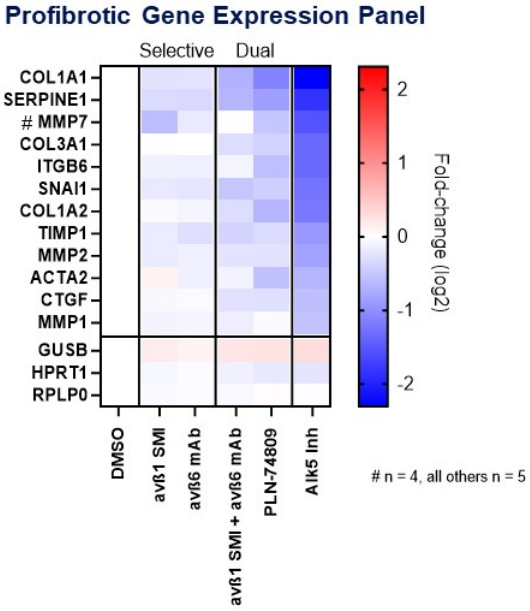
Affected Organ System	Systemic TGF-β Blockade	Observed with Pliant Compounds?[1]
Cardiovascular System	Cardiotoxicity	No
Immune System	Autoimmunity/Inflammation	No
GI System	Autoimmunity/Inflammation	No
Skin	Keratoacanthomas/SCC	No
Hematology	Thrombocytopenia/Anemia	No

1 - Based on preclinical GLP tox studies as well as clinical trials to date.

Dual $\alpha_v\beta_6/\alpha_v\beta_1$ Inhibition Blocks COL1A1 Gene Expression More than Single Inhibition in Human IPF Tissue

- **Ex-planted lungs from 5 IPF patients**
- **Sliced and cultured for 7 days**





Respiratory Research

Decaris et al. *Respir Res* (2021) 22:265

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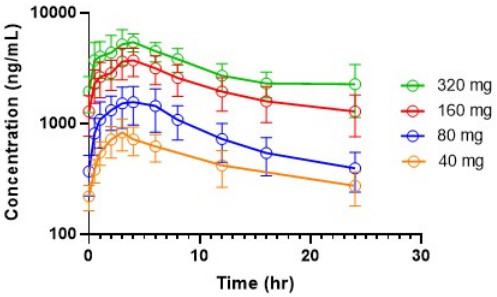
PLN-74809 – Extended Phase 1a Data Summary

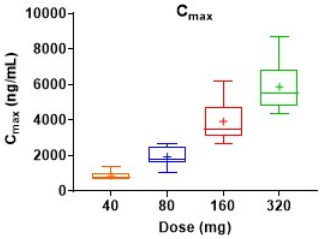
Pharmacokinetics

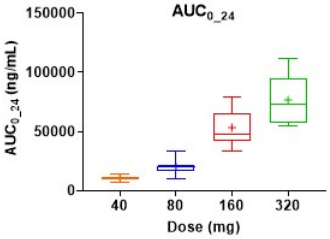
- Well absorbed, orally bio-available
- Long $T_{1/2}$: ~50 hrs – QD dosing

Summary PK Curves by Cohort at Steady State



PK sampling up to 144h; only 0-24hr plotted.
Doses 10mg to 40mg from Study PLN-74809-P1-01, Day 14.
Doses 80mg, 160mg and 320mg from Study PLN-74809-104, Day 7.





Data presented as box plots (max to min) with line at median and + at mean.

Safety

- Administered to over 450 subjects to date including healthy volunteers and patients
- Generally well tolerated
- Most frequently reported AEs were headache and constipation with no drug-related severe AEs reported

Safety Summary (Participants with drug-related TEAEs)

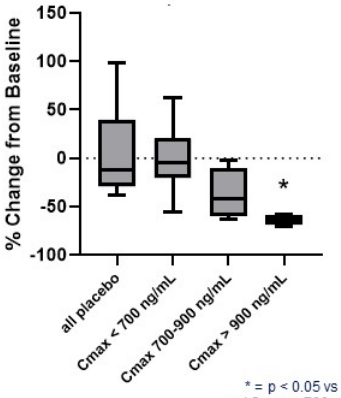
Multiple Ascending QD Doses						
	10 mg (N=9)	20 mg (N=9)	40 mg (N=9)	80 mg (N=8)	160 mg (N=16)	320 mg (N=8)
AE SEVERITY						
Mild	--	11%	--	13%	19%	25%
Moderate	--	--	--	25%	6%	--
Severe	--	--	--	--	--	--

PLN-74809 – Phase 1b Proof of Biological Mechanism

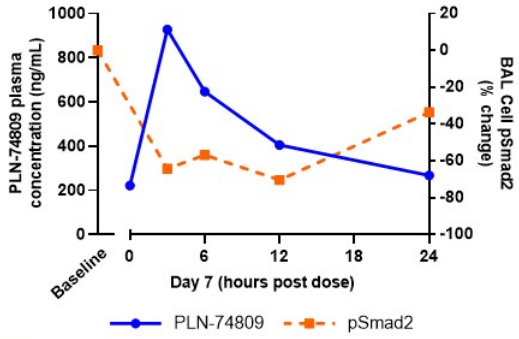
Strong PK/PD Relationship – C_{max} above IC_{50} Results in Predicted Biological Effect

Data Presented June 2019

Alveolar pSMAD2/SMAD2 (all time points)



Mean PK/PD Response in Subjects with Cmax > 700ng/mL



* = p < 0.05 vs placebo and Cmax < 700 ng/mL group

Phase 1b Study Investigating Higher Doses – Data Expected in 1Q2022

Data from this study and the PET target engagement trial evaluate 80, 160 or 320 mg to demonstrate the relationship between:

PLN-74809 plasma exposure

↓

PLN-74809 target engagement

↓

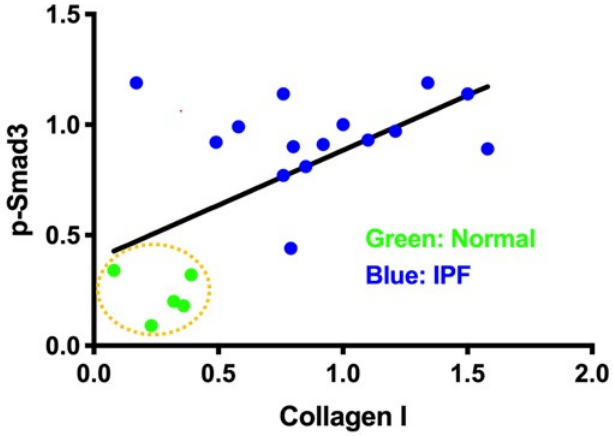
TGF-β signaling inhibition

↓

Antifibrotic activity

Tissue pSMAD Levels Are Highly Significantly Correlated with Extractable Collagen Levels in Normal and Fibrotic Lungs

Reduction in Pulmonary pSMAD Appears to Be a Marker for Reduction of Fibrosis



Pearson Correlation: r=0.6004
p (two-tailed) = 0.0051

- Diagnostic open lung biopsies from 10 patients with ILD and suspected IPF
- 2-3 distinct lung regions sampled from each patient
- 5 controls (non-transplanted lungs)
- Total pSMAD3 had a strong correlation vs. extractable Collagen I (Western Blot)

Adapted from Chapman HA et al. March 12, 2020; 382:1068-1070

The NEW ENGLAND JOURNAL of MEDICINE

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Phase 2a PET Trial – $\alpha_v\beta_6$ Expression Measured by a PET Ligand is Correlated with Extent of Fibrosis in IPF

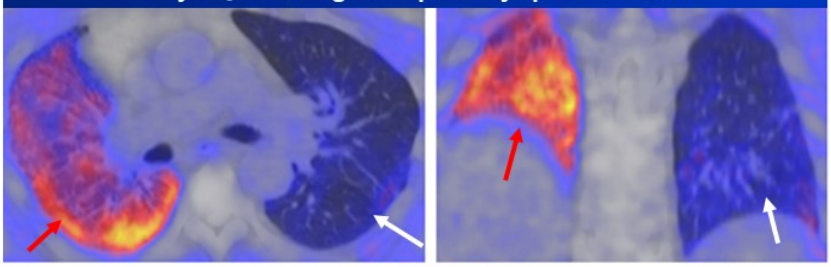
TRIAL DESIGN

- Single-site open-label trial at Stanford University
- Adults with IPF diagnosis (n=12) and FVC ≥ 45% of predicted
- Patients receive single oral dose of PLN-74809 with PET scans prior to dosing and at T_{max} post dose
- Dose cohorts being evaluated: 60 mg, 120 mg, 240 mg, and 320 mg

ENDPOINTS

- **Primary**: Evaluation of $\alpha_v\beta_6$ target engagement by PLN-74809 assessed by change in PET tracer uptake following a single oral dose
- **Secondary**: Assessment of safety and tolerability of PLN-74809 in IPF patients
- **Exploratory**: Relationship between PLN-74809 systemic exposure and positron emission tomography (PET) imaging and biomarkers in IPF participants

71-y/o ♂ left lung transplant 2yr prior to scan



Red arrows: IPF lung
White arrows: transplant lung

0 SUV 5.5

Kimura et al., *Nature Com.* 2019

PET Ligand Uptake Confined to IPF Lung in Unilateral Lung Transplant Patient

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Stanford University

Phase 2a PET Trial in IPF – Interim Analysis Methodology

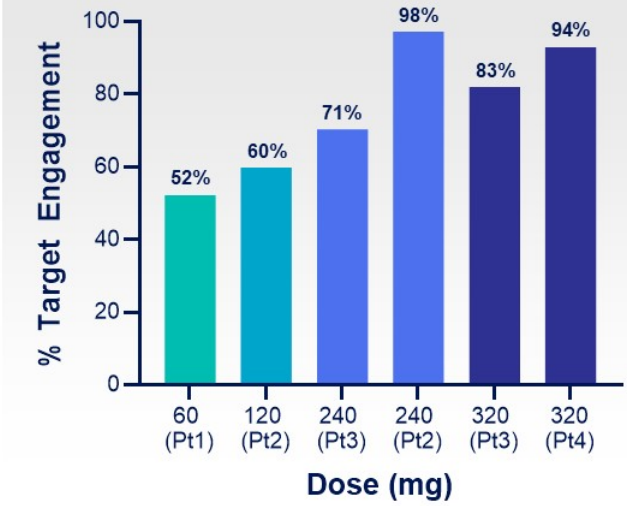
- **PET scan acquisitions at baseline (no drug) and after drug administration (4 hours post-dose)**
  - 1 week interval between baseline and post-dose PET scan acquisition
- **Administration of a single dose of PLN-74809: 60 mg – 120 mg – 240 mg – 320 mg**
- **Interim PK and target engagement data from 6 dose administrations in 4 patients**
  - 2 out of 4 patients received one single dose
  - 2 out of 4 patients received two single doses with at least a 2-week washout interval between doses

	60 mg	120 mg	240 mg	320 mg
Patient 1	x			
Patient 2		x	x	
Patient 3			x	x
Patient 4				x

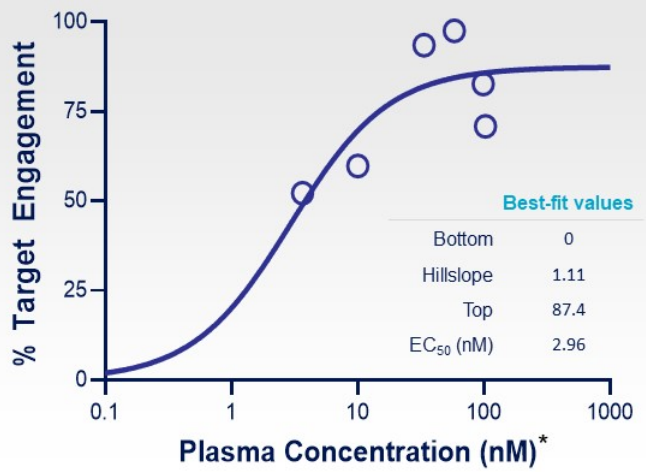
- **All patients on standard of care therapy (nintedanib)**
- **Image analysis for target engagement in highly fibrotic regions of the lungs**

Dose and Plasma Concentration Dependent Target Engagement

Dose-Dependent Target Engagement



Plasma Conc-Dependent Target Engagement



* Free plasma concentration

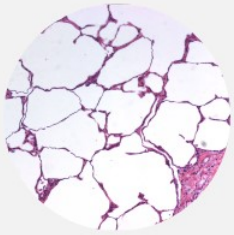
Putting the Interim Phase 2a PET Data into Perspective

Target engagement above the threshold for predicted anti-fibrotic activity across all doses (>50% target engagement)

Dose- and plasma concentration-dependent response approaching target saturation at the two highest doses

- PLN-74809 penetrates highly fibrotic areas of the lung
- Potential anti-fibrotic activity of PLN-74809 at clinical doses
- Informs dose selection in Phase 2b trials and beyond
- Provides robust PK/PD model to predict exposure-response relationship

NORMAL LUNG



IPF LUNG

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Stanford University

PLN-74809-IPF-202 [INTEGRIS-IPF]
Phase 2a Global Safety-PK-Exploratory Efficacy Trial in IPF

Enrollment Complete with Topline Data Expected Mid-2022

KEY INCLUSION/EXCLUSION CRITERIA

- Adults with IPF diagnosis
- FVC ≥ 45% of predicted
- Stratified for pirfenidone or nintedanib use

Randomization 3:1 (PLN-74809:placebo)

- Placebo (n=21)
- PLN-74809 40 mg (n=21)
- PLN-74809 80 mg (n=21)
- PLN-74809 160 mg (n=21)

PRIMARY AND SECONDARY ENDPOINTS

- Safety, tolerability, PK

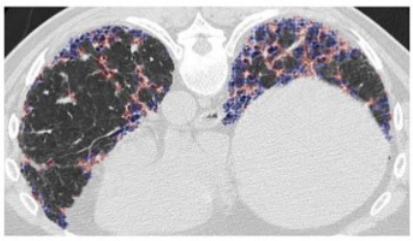
EXPLORATORY ENDPOINTS

- Change in FVC over 12 weeks
- High Resolution CT-based Quantitative Lung Fibrosis (QLF) imaging
- Effect on selected biomarkers

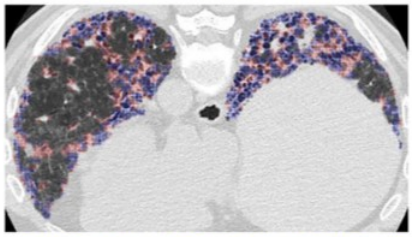
Quantitative Lung Fibrosis (QLF): A Sensitive Measure of FVC Change Over Time in IPF

- **QLF: a computed tomography (CT) biomarker**
  - Assessment via high resolution CT imaging
    - Standardized and centralized image analysis
  - QLF is an automated quantification of lung fibrosis
    - 94.4% sensitivity and 94.7% specificity
    - Fibrosis presence/ absence detected at threshold level of 1%[1]
- **QLF is a sensitive measure of change over time in IPF**
  - A change of 2% in QLF from baseline correlates with a clinically meaningful worsening or improvement of FVC
  - Moreover, clinically meaningful changes in FVC are associated with statistically significant changes in QLF[2,3]
- **Inverse correlation with:**
  - Percent predicted FVC at 6 & 12 months[3,4]
  - Percent predicted DLCO
  - Progression Free Survival (PFS)

Screening



Week 26



ΔFVC= -24%; ΔQLF= 7.8%; ΔSOBQ= 30

Representative coronal and axial HRCT images from a placebo arm participant in the BMS-986020 P2a study at screening and week 26. Change over 26 weeks: FVC: −24%; QLF score: 7.8%. Classification overlay for QLF score is in blue and red.[3]

DLCO – diffusion capacity for carbon monoxide; FVC, forced vital capacity

[1] H.Kim and J Goldin et al. *Acad Radiol*;22:70-80; 2015
[2] Kafaja et al. *AJRCCM*;197:644–652, 2018
[3] Kim et al. *Ther Adv Respir Dis*; 15: 1–11, 2021
[4] Richeldi et al. *Lancet Respir Med* 2020; 8: 25–33

PLN-74809-PSC-203 [INTEGRIS-PSC]
Phase 2a Global Safety-PK-Fibrosis and Cholestasis Biomarker Trial in PSC

Enrollment Expected to be Completed by Mid-2022

KEY INCLUSION/EXCLUSION CRITERIA

- Adults with large duct PSC
- Pre-cirrhotic/no hepatic impairment
- Stratified for UDCA use

Randomization 3:1 (PLN-74809:placebo)

- Placebo (n=21)
- PLN-74809 40 mg (n=21)
- PLN-74809 80 mg (n=21)
- PLN-74809 160 mg (n=21)

PRIMARY AND SECONDARY ENDPOINTS

- Safety, tolerability, PK

EXPLORATORY ENDPOINTS

- Effect on fibrosis biomarkers (e.g., Pro-C3, ELF)
- Change in ALP over 12 weeks
- Imaging

Pliant Development Pipeline

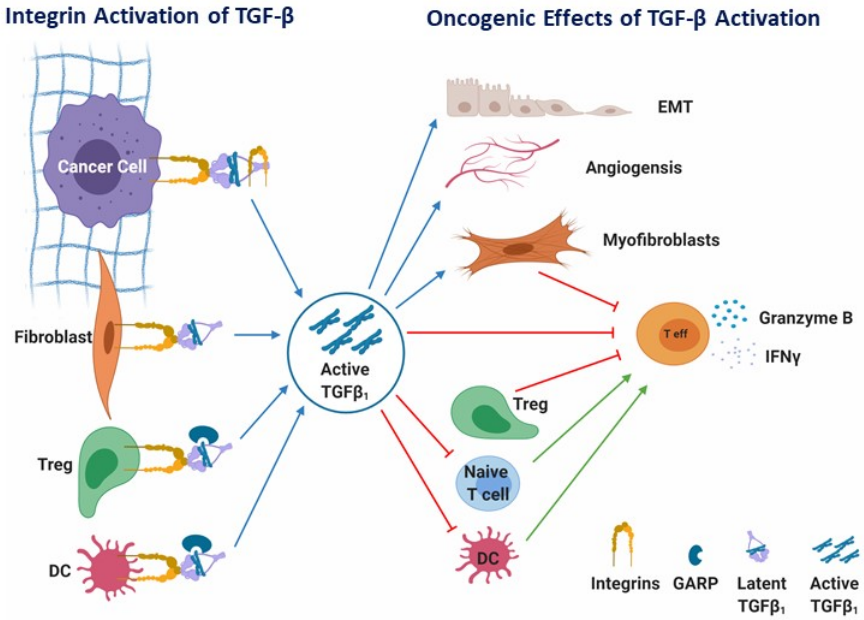
	Program	Indication	Preclinical	Clinical			Anticipated Milestone	Global Rights
				Phase I	Phase II	Phase III		
WHOLLY OWNED	PLN-74809 Dual selective inhibitor of $\alpha_V\beta_6/\alpha_V\beta_1$	Idiopathic Pulmonary Fibrosis		INTEGRIS-IPF Enrollment Complete			Phase 2a Topline Data Expected Mid-2022	PLIANT
		Primary Sclerosing Cholangitis					Phase 2a Enrollment Complete Expected Mid-2022	PLIANT
	Oncology Inhibitor of $\alpha_V\beta_8$	Solid Tumors					IND Filing Expected YE 2022	PLIANT
	Muscular Dystrophies Anti-integrin mAb	DMD Other Muscular Dystrophies					IND Filing Expected YE 2022	PLIANT
PARTNERED	PLN-1474 Selective inhibitor of $\alpha_V\beta_1$	NASH-Associated Liver Fibrosis					Phase 2 Initiation	NOVARTIS

PLIANT

Pliant's Integrin-Based Oncology Program

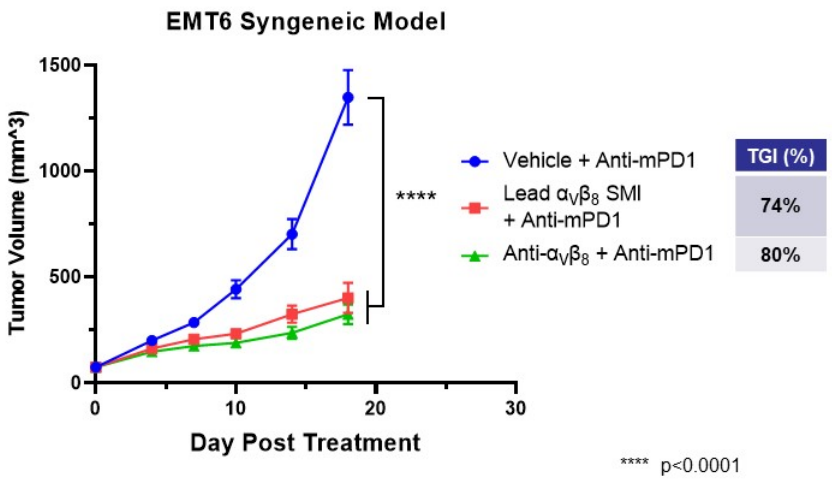
Activated TGF-β Contributes to Immune Exclusion & Evasion, Tumor Metastasis and Angiogenesis

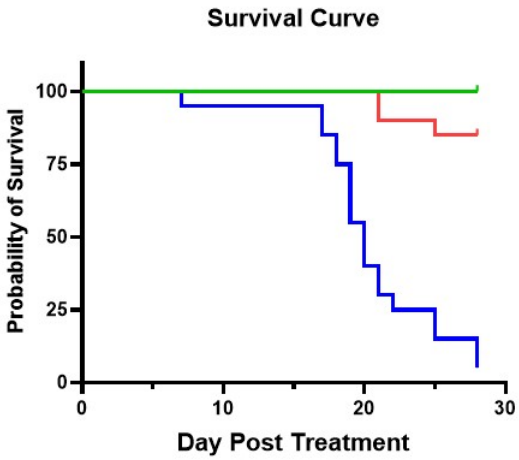
- **Integrins activate TGF-β on cancer cells and multiple cell types in the tumor micro-environment**
- **This leads to immune suppression and resistance to I/O therapies**
- **Selectively inhibiting integrin binding to latent TGF-β complex has potential to:**
  - Safely block TGF-β activation
  - Enhance efficacy of multiple checkpoint inhibition pathways



$\alpha_V\beta_8$ Inhibitor/ Anti-PD-1 Combo Reduced Tumor Burden and Increased Survival in Preclinical Models vs. Anti-PD-1 Alone

Pliant's lead oral $\alpha_V\beta_8$ inhibitor matched the maximal therapeutic effect of $\alpha_V\beta_8$ antibody *in vivo*





**** p<0.0001

EMT6 Model, N=20 per arm

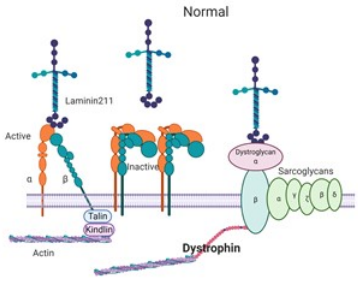
PLIANT

Selective Muscle Cell Integrin Agonism for the Treatment of Muscular Dystrophies

Pliant's Muscular Dystrophy Program – Overview

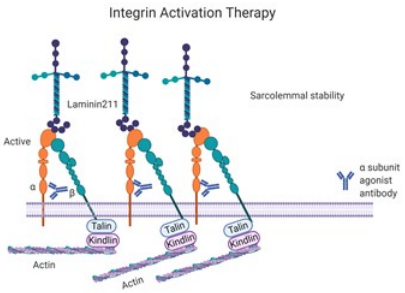
Targeting an integrin receptor on the muscle cell surface

- Integrin target is upregulated as a **compensatory mechanism** in **different types of muscular dystrophy**
- Acts as a **substitute for dystrophin**, helping to stabilize the muscle membrane, decreasing muscle damage
- **Mutations in the target result in human congenital myopathy**



Allosteric agonistic monoclonal antibody

- Activates the target to **augment the compensatory mechanism**



Potential to combine across multiple muscular dystrophy indications

- Target is **upregulated across different forms of muscular dystrophy**
- Mechanism is unrelated to underlying gene mutation
- May be **combined with existing therapies** as well as new modalities (CRISPR, gene therapy,…)

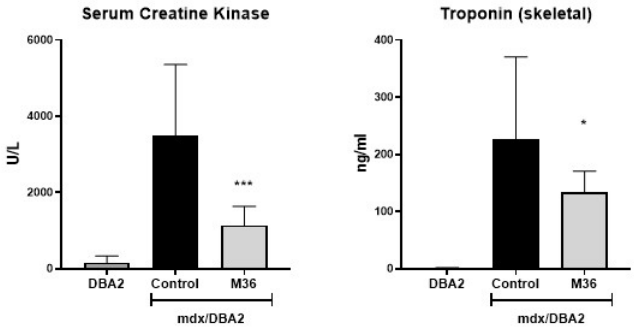
Pliant's mAb Demonstrated Improved Muscle Membrane Integrity and Diaphragm Function in Mouse DMD Model

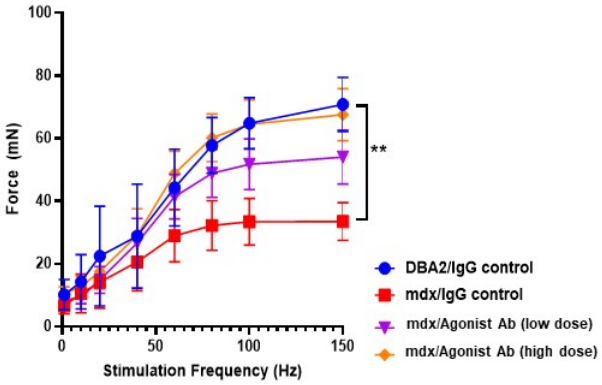
Antibody treatment protected against muscle damage

- Reduction of clinical biomarkers including serum creatine kinase and skeletal troponin

Duchenne muscular dystrophy (DMD) causes progressive wasting of cardiac and respiratory muscles (main cause of death)

- Improvement in diaphragm function is expected to significantly improve patient pulmonary function





Mean +/- SD n=10/group

PLIANT

Developing Novel Treatments for Fibrotic Diseases

Corporate Presentation

JANUARY 2022