

Bridge Biotherapeutics Corporate Presentation

November 2023



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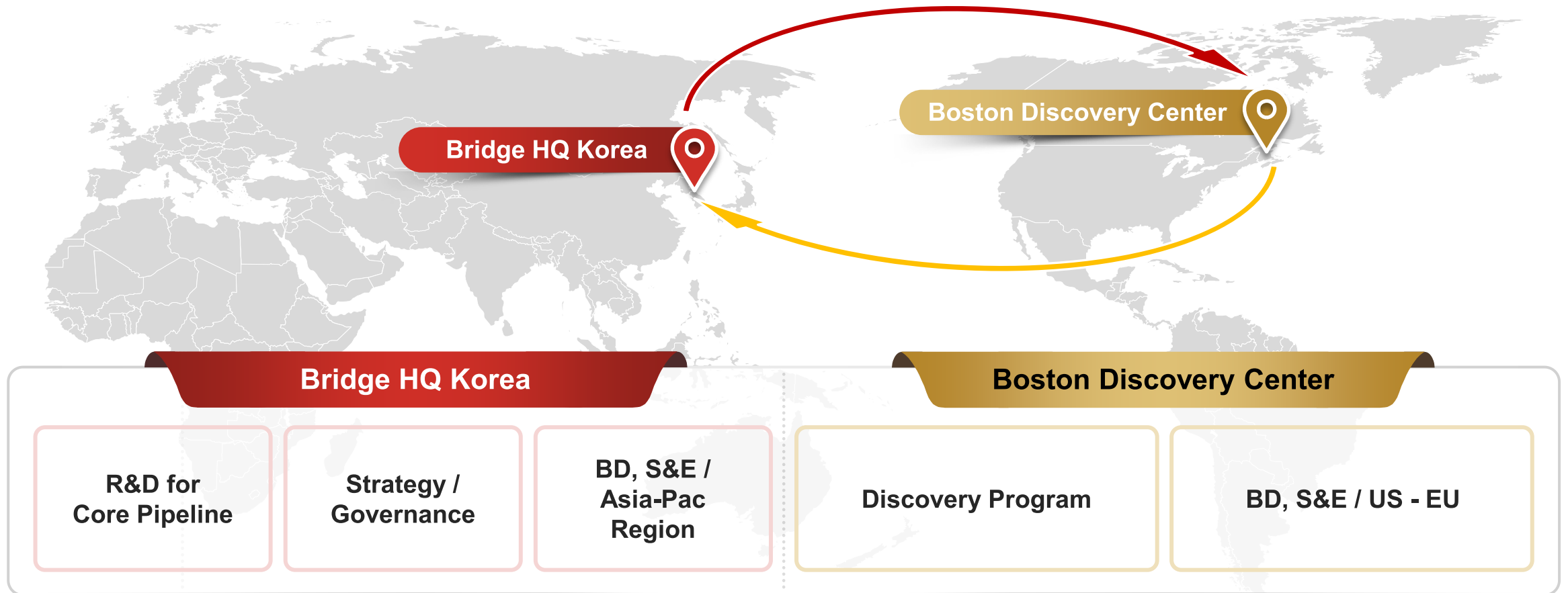
Company Overview

➤ Our vision is to become a fully integrated biotech company in the coming decade



Bi-Continental Operation

➤ We are a publicly listed, clinical stage biotech company headquartered in the Republic of Korea



BDC: Scripps Collaboration – New Drug Discovery Platform



the Cravatt Lab



BARAN LABORATORY
SCRIPPS RESEARCH

- ✓ Pioneers in the fields of covalent targeting and chemical biology
- ✓ Founded Vividion Therapeutics in 2014
→ Acquired by Bayer in 2021 for \$2B USD



Research Collaboration Overview

- ✓ Discover and characterize novel reactive groups that target non-cysteine residues to uncover new druggable sites in targets of high therapeutic value
- ✓ Scripps will conduct R&D activities (Dr. Cravatt and Dr. Baran project PIs), and Bridge Biotherapeutics will retain an exclusive option to acquire the worldwide license and patent rights to further develop and commercialize the technology

Our Commitment to Supporting Innovative Science



BaseLaunch

- ✓ Basel Area-based biotech incubator/accelerator since 2018
- ✓ Supported 20 ventures to date
- ✓ Portfolio companies went on to raise over **\$450M USD**
- ✓ Bridge Biotherapeutics has been an Industry Partner and serving on the selection committee since 2020

Partners



Korea Drug Development Fund (KDDF)

- ✓ KDDF is a national consortium of three government ministries (Trade, Industry & Energy | Science & ICT | Health & Welfare)
- ✓ Established in 2011 to foster drug development programs in Korea
- ✓ KDDF provides R&D Investment, **Development Consulting** as well as **BD and Licensing Support**

Company Pipeline

Pipeline Overview

➤ Bridge Biotherapeutics continues to grow its clinical and discovery pipeline

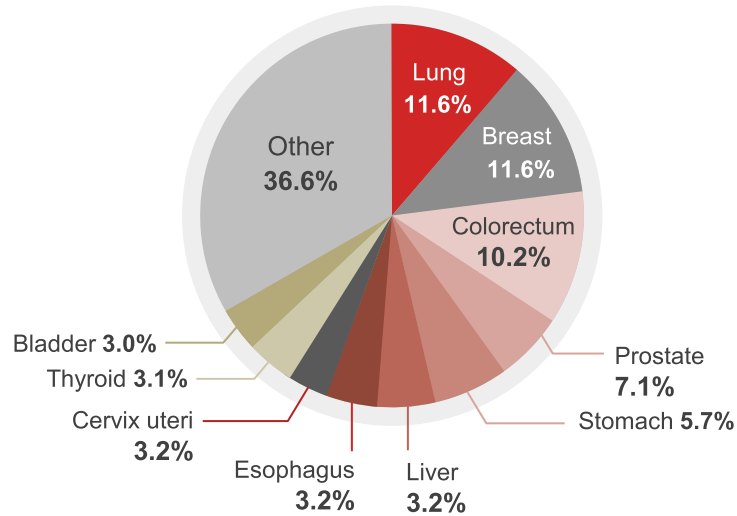
	Program	Target	Indication	IND Enabling	Phase 1	Phase 2	Phase 3
Cancer	BBT-176	EGFR emerging mutation	NSCLC				Deprioritized
	BBT-207	EGFR emerging mutation	NSCLC				Enrolling (US/KR)
	BBT-778	YAP-TEAD	Solid Tumor				
Fibrosis	BBT-877	Autotaxin	IPF				Ongoing
	BBT-301	K _{ca} 3.1	IPF				2024 Planned
	BBT-209	GPCR19	IPF				2024 Planned
UC	BBT-401	Pellino-1	Ulcerative Colitis				

4th Generation EGFR TKI for NSCLC

BBT-207

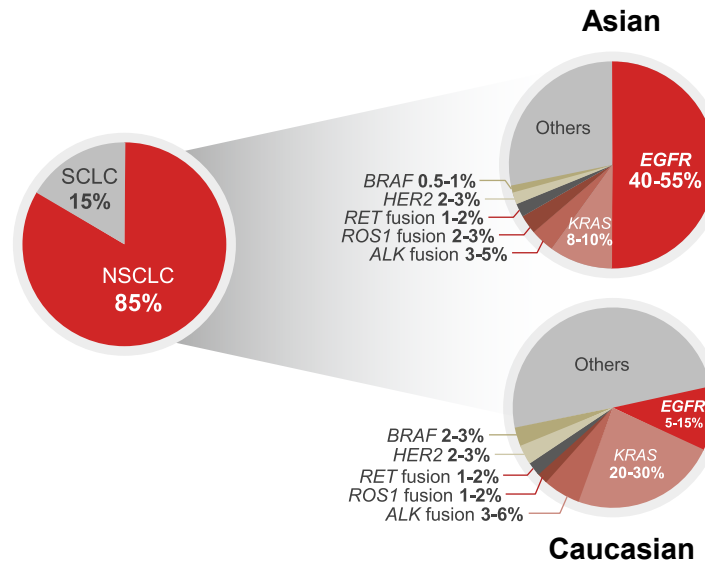
Increasingly More Patients in need of 4G EGFR TKI

Most Common Cancers Worldwide

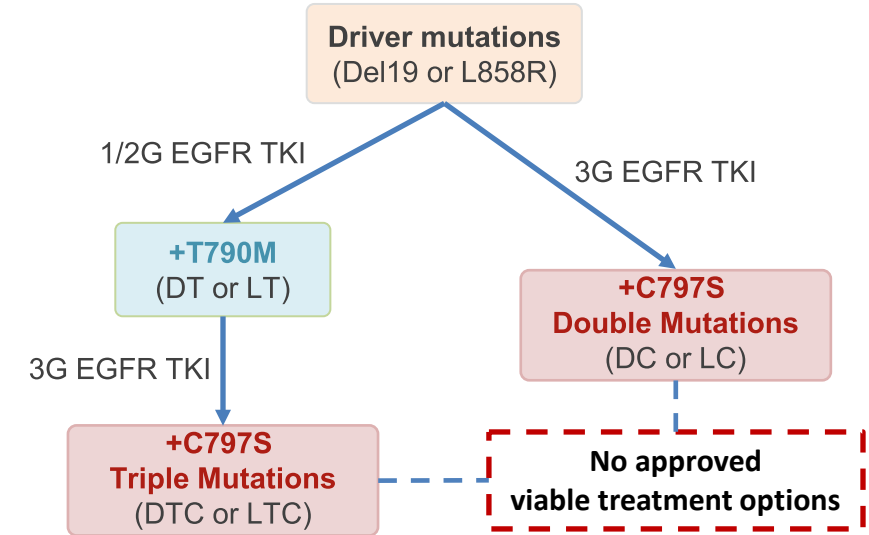


Source: 2018 Global Cancer Statistics Report

Genetic Drivers of NSCLC



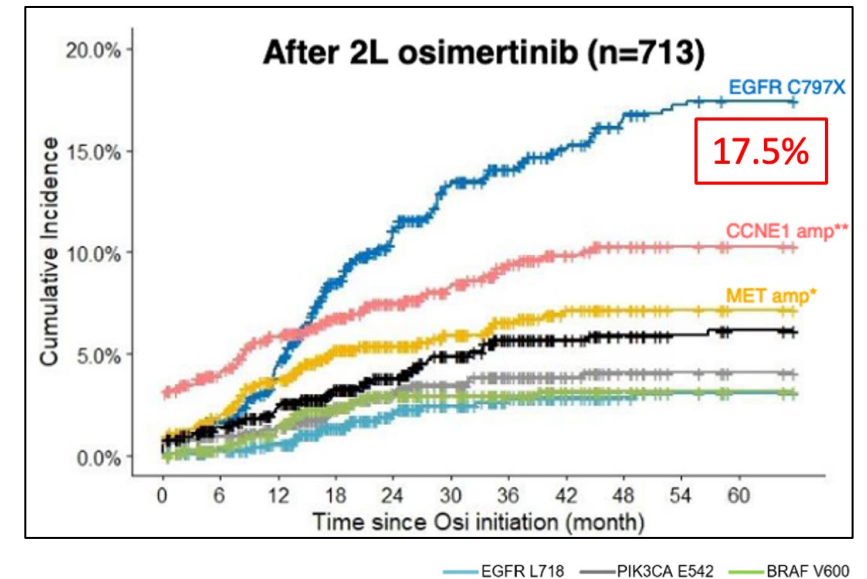
The Unmet Need



- Lung cancer is the most common cancer and the leading cause of cancer death in the world (>1.5 million cases every year)
- NSCLC is the most common subtype of lung cancer, which accounts for 85% of cases
- EGFR mutations are the cause of over 50% of NSCLC in Asians and between 5-15% in Caucasians
- Across the world, patients who progressed on 3G EGFR TKIs due to C797S are left with no viable treatment options

Global Solution Requires Broad-Spectrum Activity

DTC/LTC mutations the dominant resistance mechanism after ~1 year²



- In most developed nations frontline use of osimertinib is more widespread¹
- C797S+ double mutations are more common (DC and LC)²



- For majority of ROW, 3rd-gen EGFR TKIs still given from 2L³
- T790M mutations much more prevalent

➤ **BBT-207 is capable to satisfy the global unmet need for those with C797S+ NSCLC**

1. NCCN guidelines for NSCLC (version 6.2020)
 2. Suresh S. Ramalingam, Poster Presentation, WCLC2022
 3. 2020 IASLC Survey (based on use of molecular testing and NGS accessibility)

D = Del19
 L = L858R
 T = T790M
 C = C797S

BBT-207 Prioritized as the Lead Program for EGFRm NSCLC

- BBT-176 Phase 1a conclusion – Well tolerated, however, BBT-207 widely expected to be more efficacious
- BBT-207 has improved potency over BBT-176 across the full EGFRm spectrum

Engineered Ba/F3 cell line IC₅₀ (nM)

Compound	WT	D	L	DT	LT	DC	LC	DTC	LTC
BBT-207	184	6	11	4	4	1	16	5	8
BBT-176	645	67	164	147	114	76	244	148	276
Osimertinib	164	1	2	3	3	509	829	979	1303

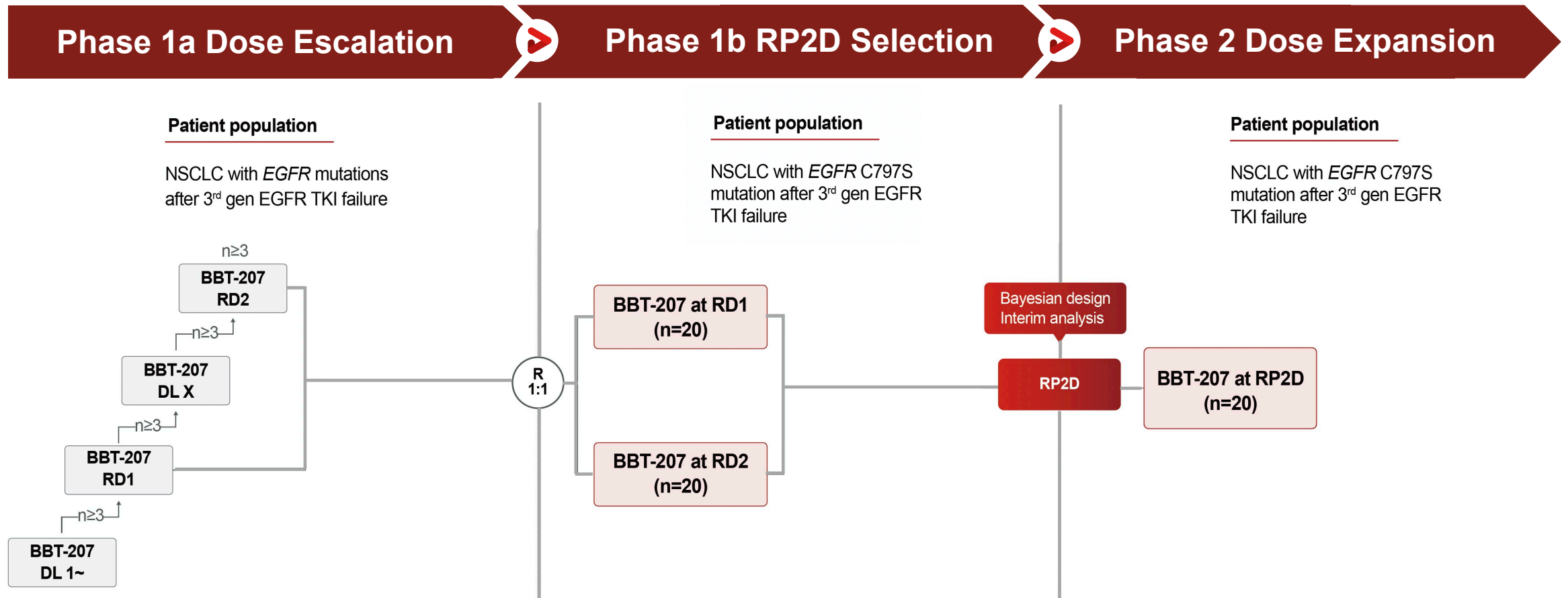
BBT-207 In Vivo Efficacy

BBT-207 40mpk QD			BBT-176 60mpk QD / 90mpk QD	
Model	TGI	TR	TGI	
DC	107%	8/8	60mpk : 64.4%	/ 90mpk : 101.3%
LC	102%	2/8		
DTC	107%	7/7	60mpk : 66%	/ 90mpk : 77%
LTC	108%	6/8		
D/L/DT/LT	>100%	Majority	BBT-207= >100% TGI confirmed in all EGFR mutants	

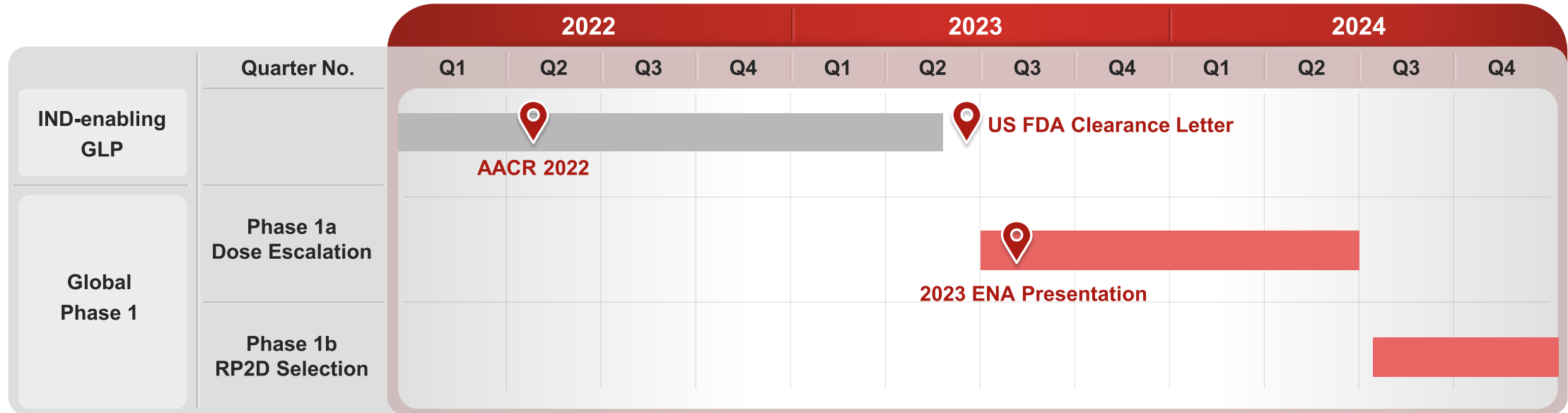
- D = Del19 | L = L858R | T = T790M | C = C797S
- NA: Not Analyzed
- TGI: Tumor Growth Inhibition (% , relative to vehicle)
- TR: Tumor Regression (number of mice)

BBT-207 Clinical Trial Design

- Phase 1a dose-escalation portion of the study is now ongoing
- The study is expected to span approximately 15 sites in the US and South Korea



BBT-207 Clinical Trial Expected Timeline

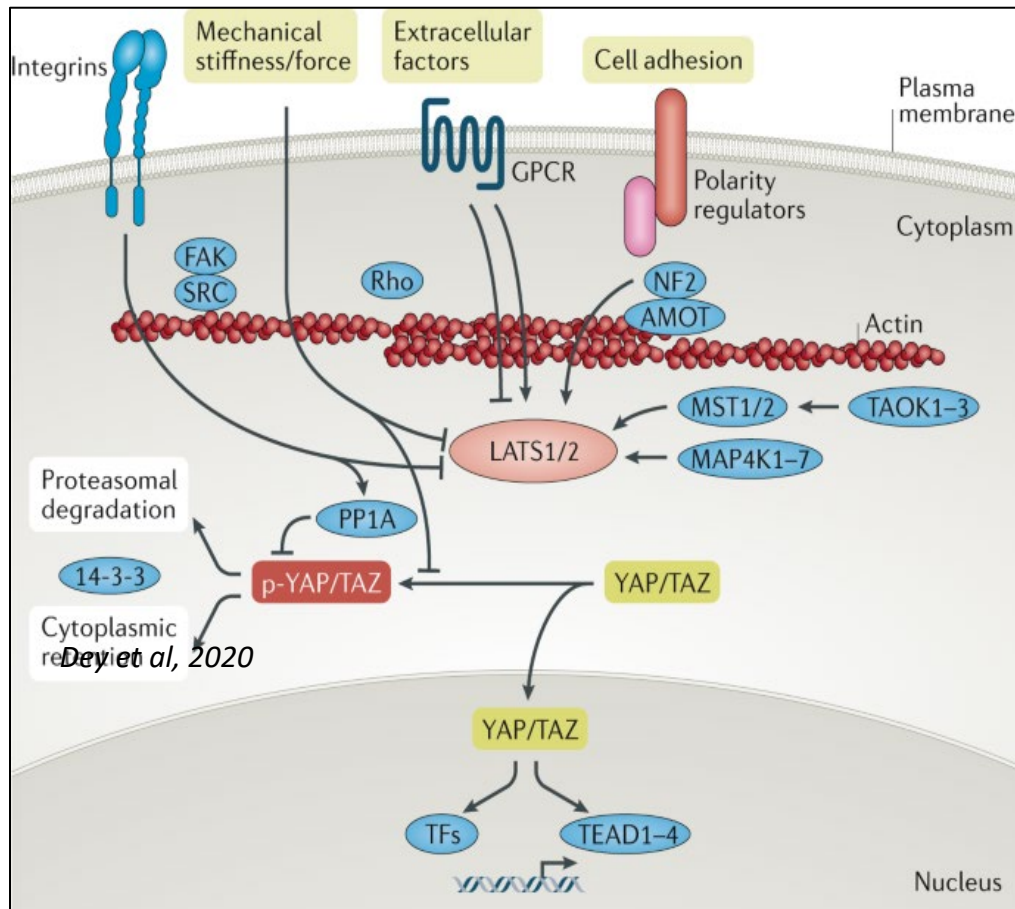


- US FDA clearance letter for FIH study – April 2023
- Detailed information on the clinical trial design presented at 2023 AACR-NCI-EORTC conference
- Global Phase 1 trials now ongoing in the US and South Korea

YAP-TEAD Inhibitor for Solid Tumors: BBT-778

BBT-778 : A novel, reversible, pan-TEAD inhibitor

➤ YAP/TAZ nuclear translocation highly morbidic



➤ BBT-778, pan-TEAD Inhibitor

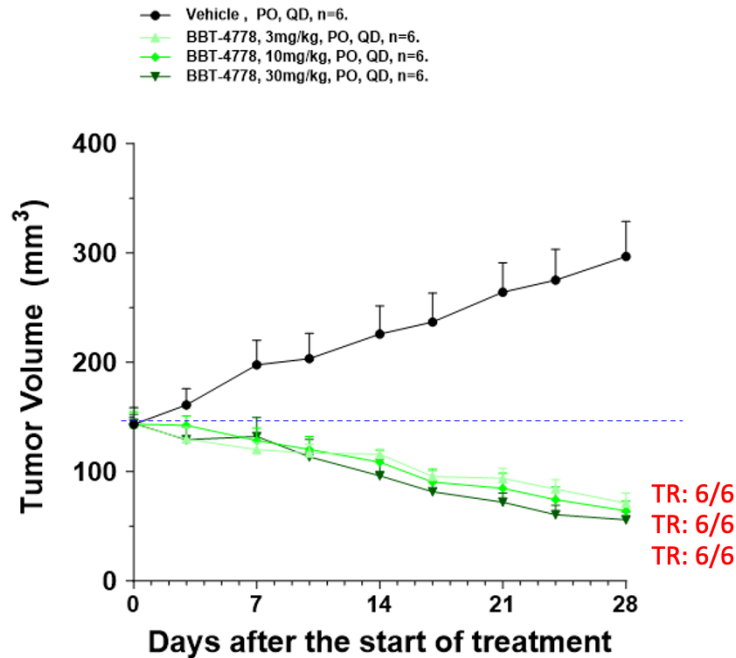
- Novel, brain-penetrable, reversible pan-TEAD inhibitor
- Good in vitro synergistic effect in combination with osimertinib
- Efficacious and dose-dependent anti-tumor effect at various NF2 mutant xenograft models
- Favorable pharmacokinetic and ADME profiles, including excellent brain and tumor penetration
- IND-enabling GLP tox will be initiated soon
- Primary target indications are
 - NF2-mutated cancers (Mesothelioma, Meningioma, Schwannoma, breast cancer, etc.)
 - Osimertinib-resistant EGFR+ NSCLC with activated Hippo signaling

Efficacious and dose-dependent anti-tumor effect in several NF2m models

- BBT-778 strongly inhibits tumor growth in a dose-dependent manner without significant BW loss
- High brain exposure and penetration → majority of target indications involve brain metastasis

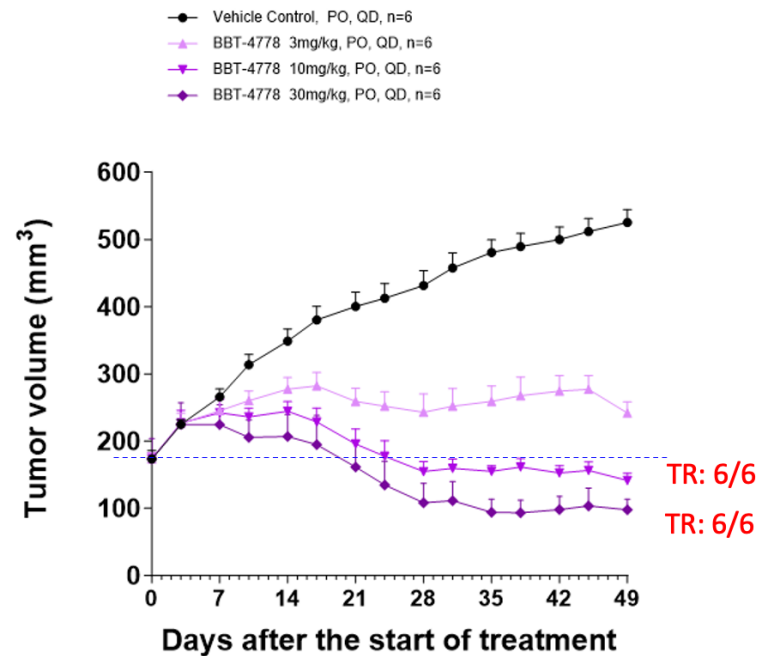
H226 NF2 mutant xenograft model

Squamous cell carcinoma



H2373 NF2 mutant xenograft model

Mesothelioma



Brain Exposure and Penetration

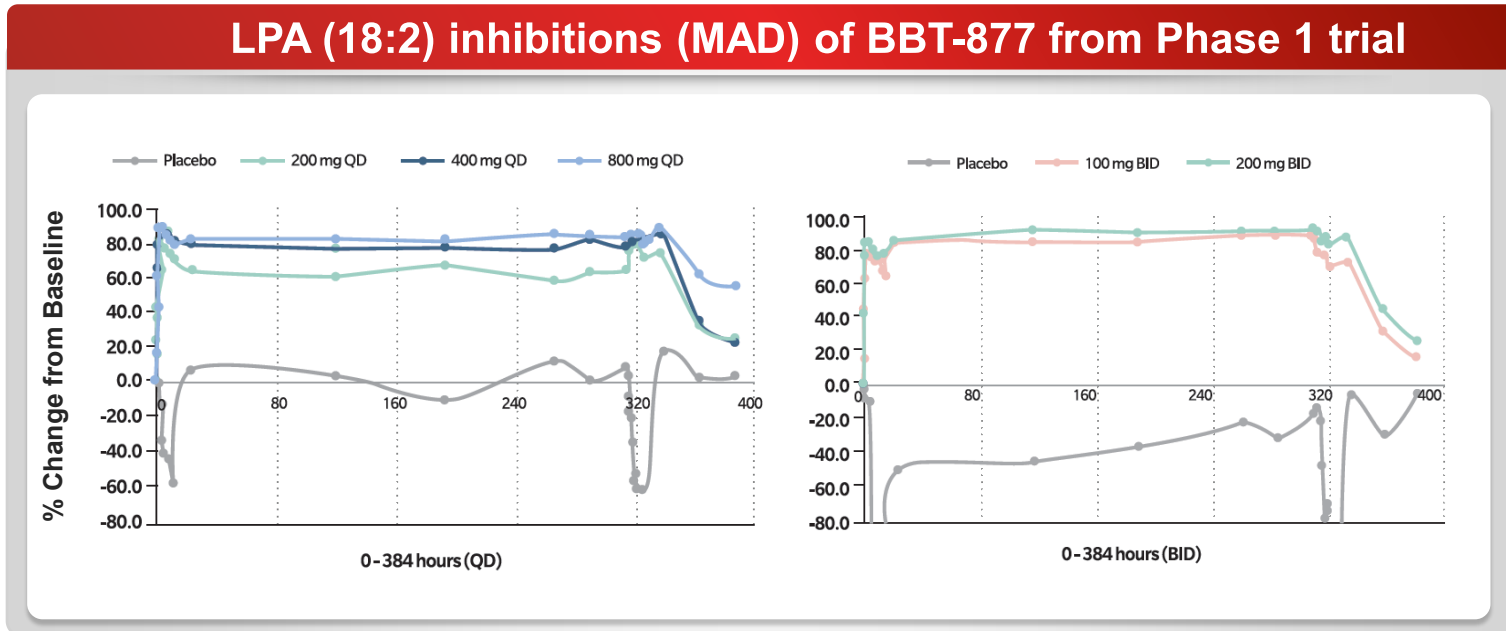
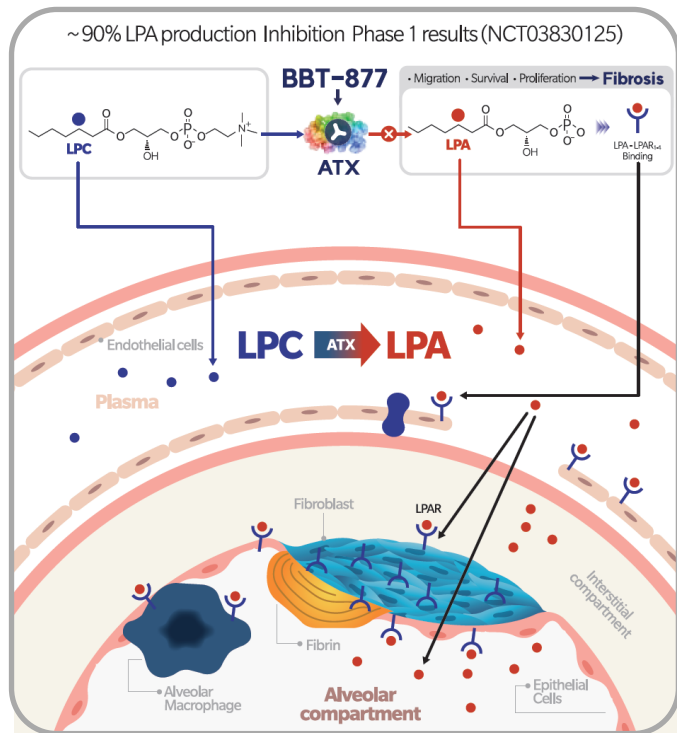
B/P (total exposure)	1.5
B/P (free fraction)	0.113
K _{puu}	0.167

IPF

BBT-877

Autotaxin inhibitor

BBT-877 MoA and LPA Inhibition Phase 1 Clinical Data



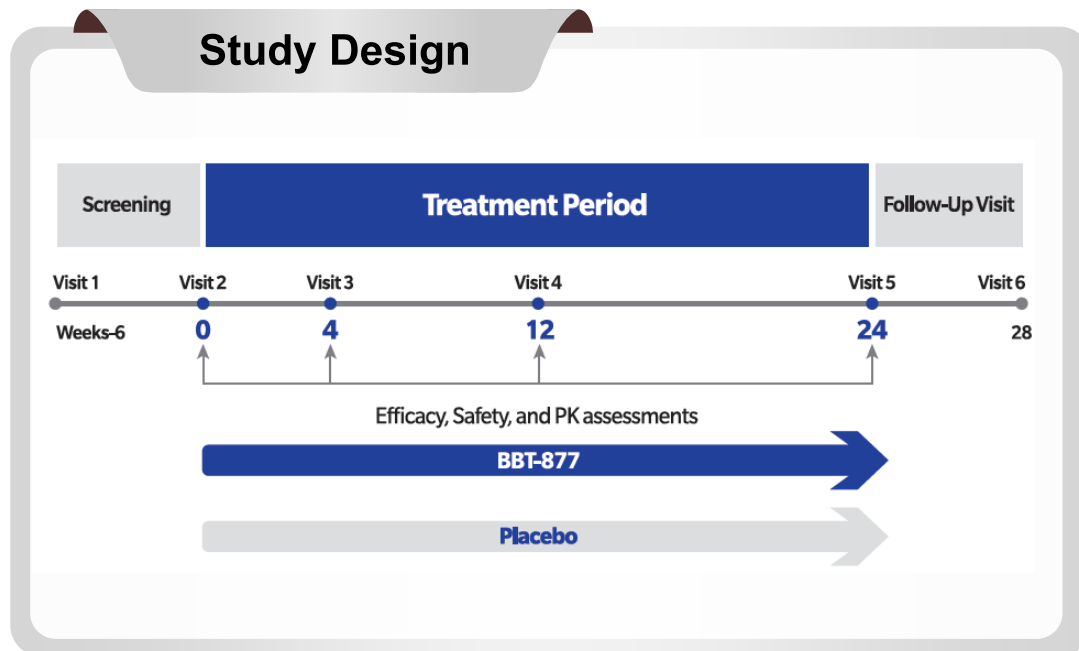
- Dose proportional systemic exposure
- Well tolerated with no SAEs reported from three Phase 1 studies
- Exhibited dose response PKPD with sustained LPA inhibition of up to 90%

	Galápagos	BLADE THERAPEUTICS	bridgebio therapeutics
	GLPG1690	cutetaxestat	BBT-877
Dose	600mg (QD)	500mg (BID)	200mg (BID)
% LPA Inhibition	~60%	~80%	~90%

GLPG1690 (Ph2a data), BBT-877/cudetaxestat (Phase 1 data)

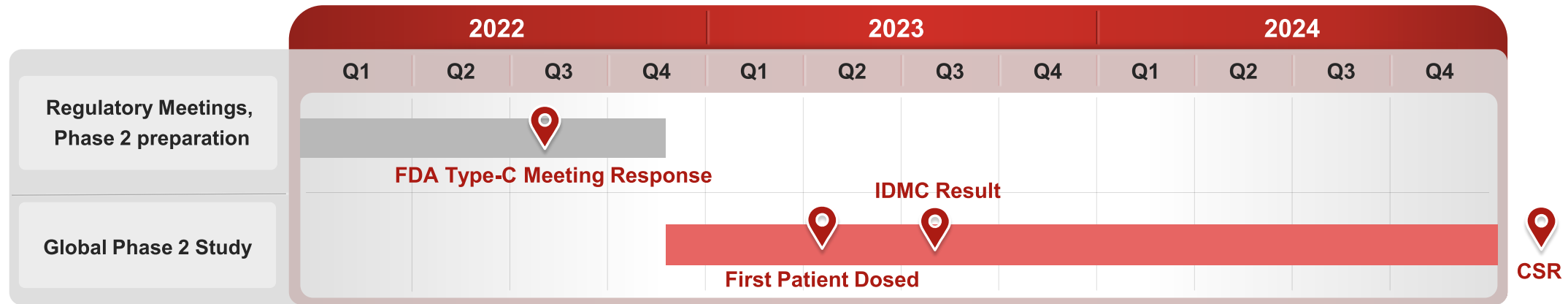
Multinational Phase 2a Clinical Trial Design

- First patient dosed in April 2023
- Approximately 40/120 patients have been enrolled as of Oct 2023



The randomized, double-blind, placebo-controlled Phase 2 study utilizes a 200 mg, BID regimen of BBT-877 or placebo

BBT-877: Development and Clinical Trial Expected Timeline



➤ First IDMC complete – Given recommendation to continue with Phase 2 trial

➤ Growing interest in the ATX-LPA-LPAR pathway – BBT actively exploring various collab models

IPF

BBT-301

Potent K_{ca} 3.1 Modulator

BBT-209

Selective GPCR19 Agonist

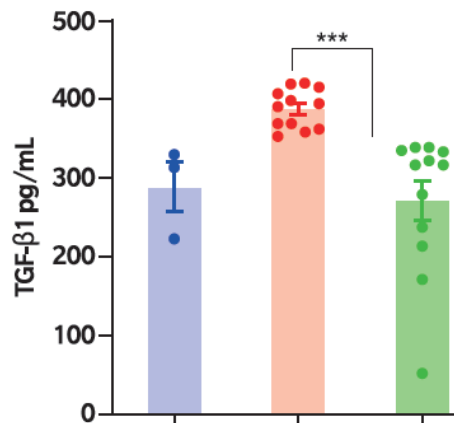
BBT-301 : Potent $K_{Ca}3.1$ Modulator

$K_{Ca}3.1$

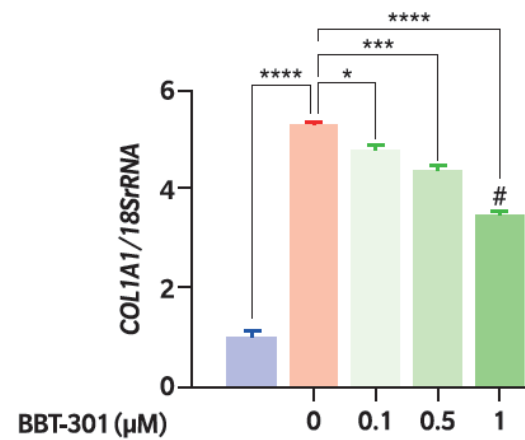
$K_{Ca}3.1$ is a novel target identified to be upregulated in IPF patients and is associated with the promotion of pro-fibrotic lung fibroblast function and facilitating fibroblast to myofibroblast differentiation – a key factor in IPF development.

	IC ₅₀ (nM)
BBT-301 ¹	6
ICA17043 (Senicapoc) ²	11
TRAM34 ³	20

BBT-301 reduced TGF- β & the expression of collagen in mPCLS (precision-cut lung slices) and IPF patient-derived lung fibroblasts

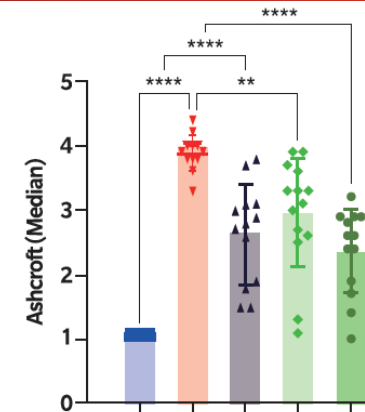


BLM		+	+
BBT-301			+

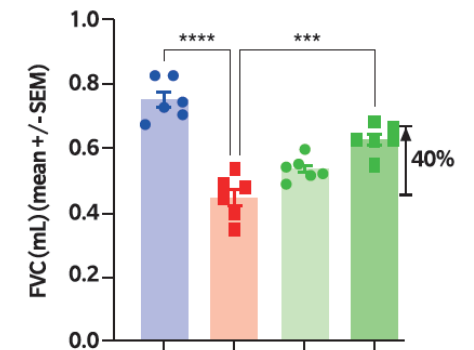


#p<0.001 vs BBT-301 0.1 μM

In terms of forced vital capacity (FVC) and Ashcroft score, BBT-301 has shown comparable potency in lung function recovery



BLM		+	+	+	+
Nintedanib (mg/kg)				120	
BBT-301 (mg/kg)				2.5	5

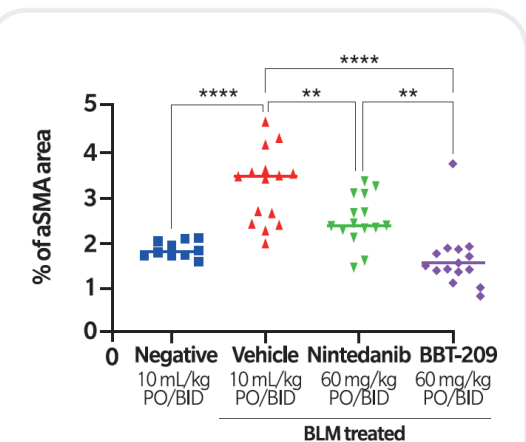
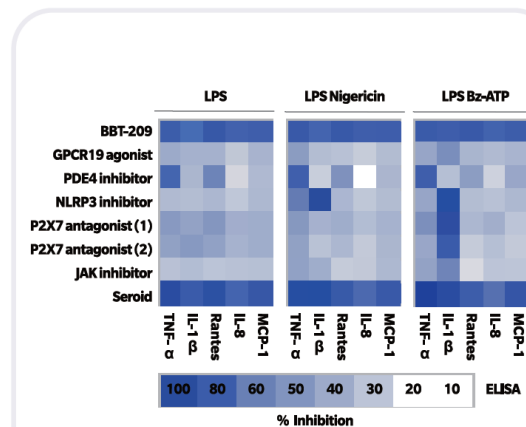
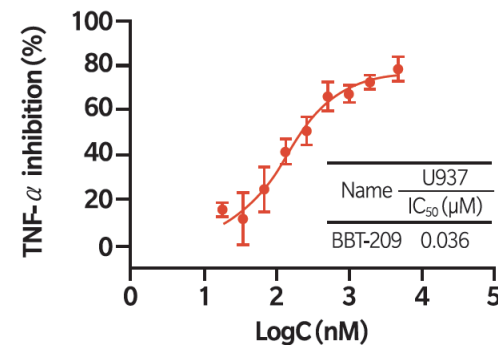
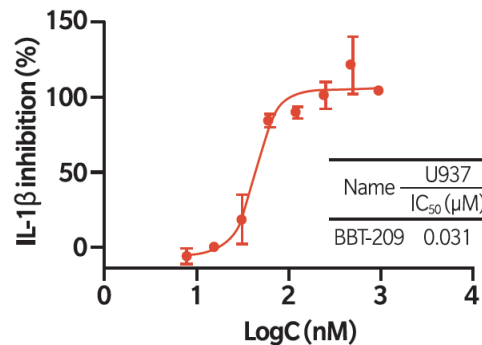


BLM		+	+	+
BBT-301 (mg/kg)			2.5	5

BBT-209 : Selective GPCR19 Agonist

GPCR19

GPCR19 is a prerequisite for P2X7-mediated Ca²⁺ mobilization, in which activated P2X7 leads to lung inflammation and fibrosis. BBT-209 potently suppresses P2X7 receptor expression and greatly attenuates the P2X7-mediated inflammasome in various inflammatory diseases.



- Competitive inhibition of IL-1 β and TNF- α cytokine expression
- Greater reduction in aSMA area (myofibroblast marker) than Nintedanib

Summary and Concluding Remarks

Summary and Closing Remarks

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Best-in-Class IPF Assets

Comprehensive multi-pronged approach to combat IPF through multiple validated pathways

2

Novel Oncology Programs

Aimed at treating prevalent cancers globally, focusing on the most sought-after targets in the field



Actively seeking regional or global partnerships

Boasting a growing global presence and a portfolio targeting diseases that impact patients worldwide, our company presents an attractive partnership opportunity

Our Commitment

Driven by science, we are committed to pioneering groundbreaking medicines to address global unmet needs, transforming lives and shaping a healthier future for all



bridgebio
therapeutics

SK Headquarters

Suite 303, 58, Pangyo-ro 255 beon-gil,
Bundang-gu Seongnam-si, Gyeonggi-do,
13486, Republic of Korea

US Branch

Bridge Biotherapeutics, Inc.
55 Chapel Street, Suite 100
Newton, MA 02458, USA

Please contact

BD Director – pavel.printsev@bridgebiorx.com

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