Introduction of
JD BIOSCIENCE

JD BIOSCIENCE

Q1 2023

Overview

JD BIOSCIENCE

1 Background
2 Experience
3 Purpose
4 Staff
5 Timeline
6 Targets and Technology

- **JD BIOSCIENCE** is a bio-venture focused on developing **small molecule** drugs for **metabolic diseases** (NASH, fibrosis, Inflammation, Obesity, and Cancer).
  and expanding research area including **ADC and PROTAC**

- We have robust **first-in-class drug candidates**. (NASH and IBD)
- Growing team of **~30** employees and **~20** with expertise in **medicinal chemistry**. (Established in 2017)
- **~15** external research **collaboration partners**.
  (Our role: Medicinal chemistry)

PROTAC-Proteolysis targeting chimera
ADC-Antibody Drug Conjugate
NASH-Non-Alcoholic Steatohepatitis

Medicinal chemistry

Jin Hee Ahn, Ph.D.

Chief executive officer

Medicinal chemistry

Doo Seop Kim, Ph.D.

Executive advisor

Medicinal chemistry

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Target discovery

Hail KimM.D., Ph.D.

Target discovery, mechanism of action, and efficacy tests

In-Kyu Lee M.D., Ph.D.

Target discovery, mechanism of action, and efficacy tests

Stability testing of API

Myung Ae Bae Ph.D.

Pharmacokinetics and druggability

★ 5 Licensing out to Pharma

Januvia (sitagliptin) 25mg, 50mg, 100mg tablets

Myung Ae Bae

Hail Kim

PDK

HTR

★ TPH

2016 Present

In-Kyu Lee

★ GPCR 2014 2016

Don Haeng Lee

TAZ modulator 2013 2016

★ DGAT-1 2010 2013

Hwang, Eun-Sook

★ 11β-HSD1 2007 2010

Doo Seop Kim

★ DPP-IV 2004 2008

PPARγ 2001 2005

PTP-1B 2000 2004

over 20 YEARS EXPERIENCE

2000 Present

To develop drugs for unmet metabolic disease

Management Team

CEO & Founder Jin Hee Ahn, Ph.D

- Professor, GIST
- Principal Researcher, KRICT

Executive Advisor

Doo Seop Kim, Ph.D.

- Vice president & CTO, Kainos Medicine
- Chief investigator, Merck & Co for 20 years

Chemistry Director

Seongrim Byeon, Ph.D

- Director/Healthcare division/R&D, Kainos Medicine
- Research Scientist, KIST

BD Director

Sungmin Song, Ph.D

- Technology licensing manager, GIST
- Researcher, KRIBB
- Ph.D./post-doc, Biology, Freiburg Univ.

Innovation Director

Peter Goughnour, Ph.D

- Curigin R&D Director
- Kyung Hee Univ. Research Professor
- Ph.D./Post-Doc, Pharmacology, Seoul Nat. Univ.

Scientific Advisory Board

Chief Advisor

Rohit Loomba, M.D, MSHc

- Professor of Medicine, Director of Hepatology, UCSD

Advisor

Marc Hellerstein, M.D., Ph.D.

- Professor of Nutritional Biochemistry, UC Berkeley

Advisor

John York, Pharm D, MBA

- Akita Biomedical, Founder, CEO

Advisor

Joon Yong Park, M.D, Ph.D

- Yonsei University College of Medicine, Professor,

Advisor

Jung Il Lee, M.D, Ph.D

- Yonsei University College of Medicine, Professor,

July 2017 — JDB Founded

Aug 2017 — **Pre-A** led by Sunbo Angel Partners

Sept 2017 — JDB Trademarked

Oct 2017 — TIPS R&D business project by the Ministry of SMEs and Startups

Feb 2019 — **Series A** led by MIRAE ASSET Mutual Fund
- Mirae Asset Capital
- Hyundai Venture Investment
- BSK Investment
- Synergy IB investment
- Opez Investment

Mar 2019 — Opened the Seoul office Established the R&D center at GIST, Gwangju

Jan 2021 — **Series B** led by MIRAE ASSET Mutual Fund
- Mirae Asset Capital
- Hyundai Venture Investment
- BSK Investment
- Synergy IB investment
- SV Investment
- Daily Partners
- SJ Investment Partners
- WONIK Investment partners
- Innopolis Partners
- K2 Investment
- UTC Investment
- KDB Capital Corp.

Nov 2021 — Tox study completed

March 2022 — Australian Subsidiary

July 2022 — NASH submission to HREC

Sept 2022 — NASH Phase I clinicals

Vision

To be the leading small molecule company in the field of metabolic related disease

Our Mission

"Discovering and developing drugs against novel targets to address the unmet medical needs in Metabolism, Fibrotic-related disease, Cardiovascular, and Cancer."

Our Goals

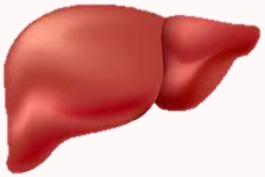
- Investigating novel biomarkers for unmet medical needs
- First-in-class drug development
- Bring more pipelines to clinical phase

Our Accomplishments

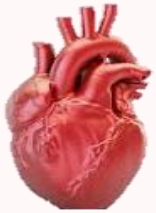
- Medicinal chemistry services to partners
- Knowhow and experience for novel drug development
- Stable sales revenue
- Phase I Clinical Trials with lead asset

Diseases

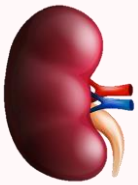
Liver



Heart

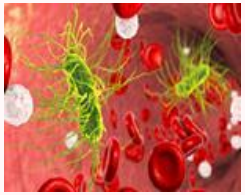


Kidney

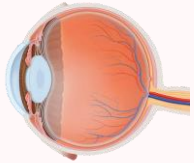


Neural

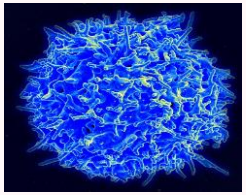
Infection



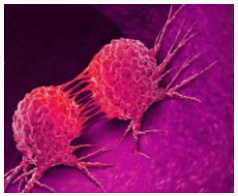
Eye



Immune

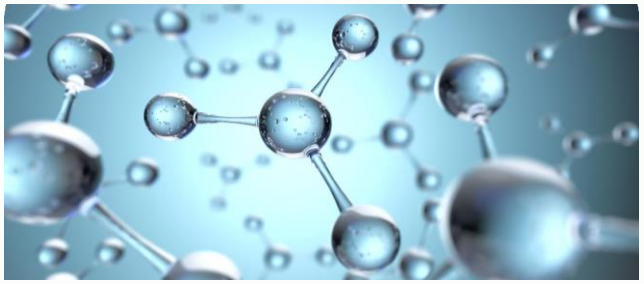


Cancer



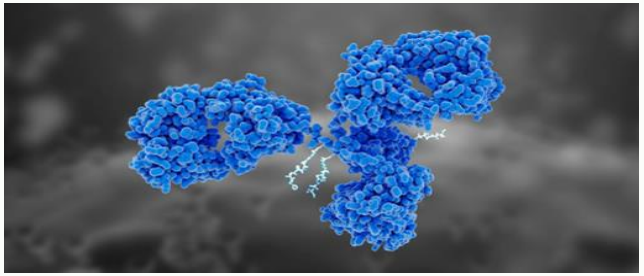
Platforms

Small Molecule



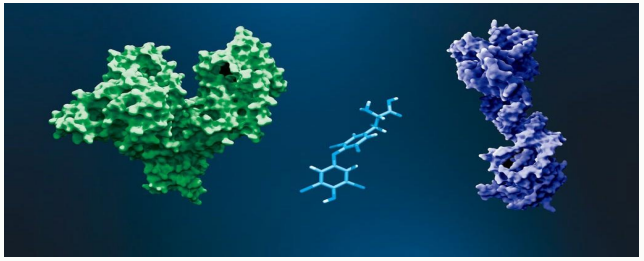
JDB can synthesis any small molecule (SM) as an agonist or antagonist for disease related proteins and receptors

Antibody Drug Conjugate



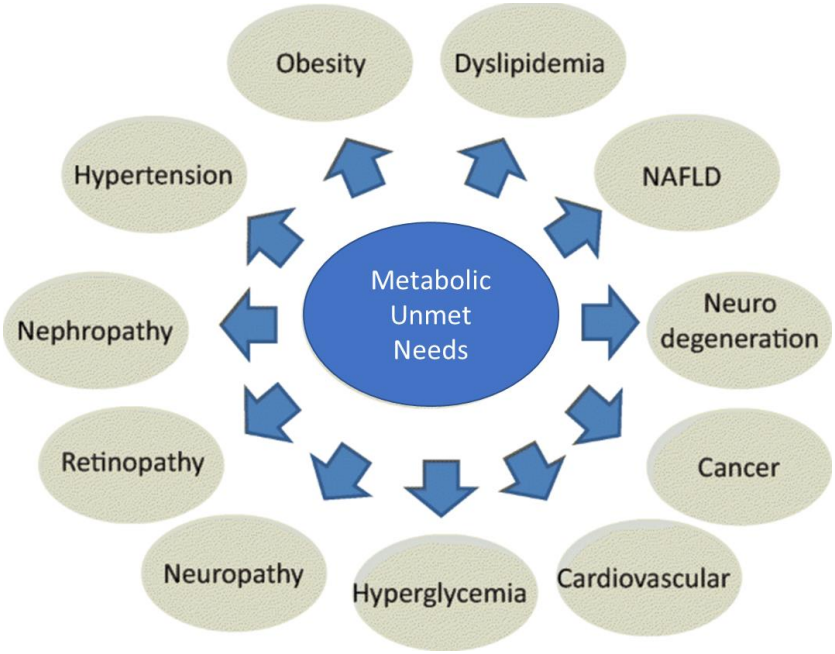
Antibody Drug Conjugate (ADC) that selectively binds to a specific target and releases our payload. JDB can create these novel complexes.

Proteolysis Targeting Chimera



Proteolysis Targeting Chimera (PROTAC), JDB can synthesize moiety and linker for any protein target.

Metabolic diseases have high unmet medical needs



Jacob Bar-Tana Rev Endocr Metab Disord, 2020 (Modified)

New medications are needed

Drug Pipelines

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1 Lead Pipelines

2 NASH

3 IBD

4 Discovery Stage

Pipeline	Code	Indication	Target	Discovery	Pre-clinic	Phase 1	Phase 2	Plans for BD
1	GM60106	NASH	HTR					L/O for USA or other territories at Phase I
	GM60***	NASH	HTR					Backup of GM60106
2	GM10395	Inflammation Heart disease Cancer	PDK					L/O for USA or other territories at preclinical stage
	GM-X2	Pancreatitis/ Heart disease	PDK					
3	-	Sepsis Septic shock	confidential					Commercialize in Korea market
4	-	NASH	confidential					Commercialize in Korea market
5	-	Cancer	confidential					ADC Partners
6	-	Undruggable Targets	confidential					PROTAC Partners

HTR-5HT(Serotonin) Receptor
PDK-Pyruvate Dehydrogenase Kinase
ADC-Antibody Drug Conjugate
PROTAC-Proteolysis Targeting Chimera

Lead Candidate
Back-up

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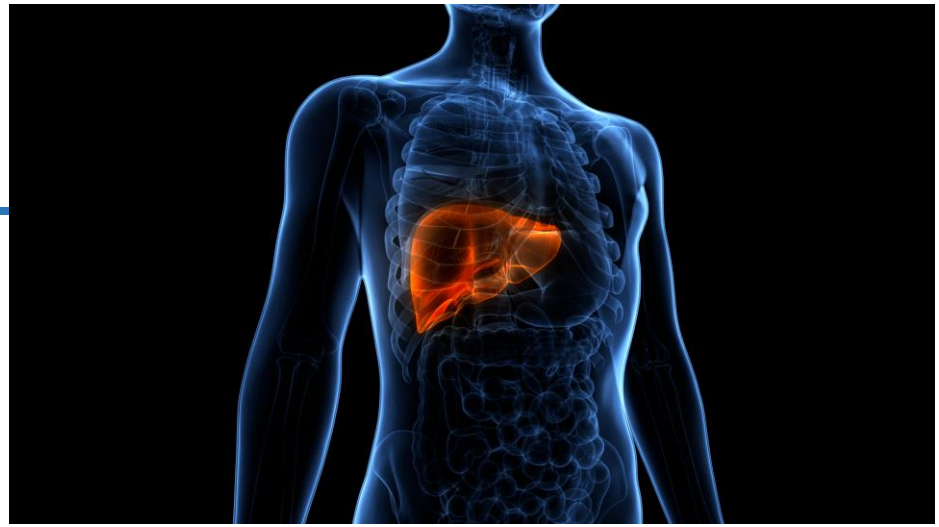
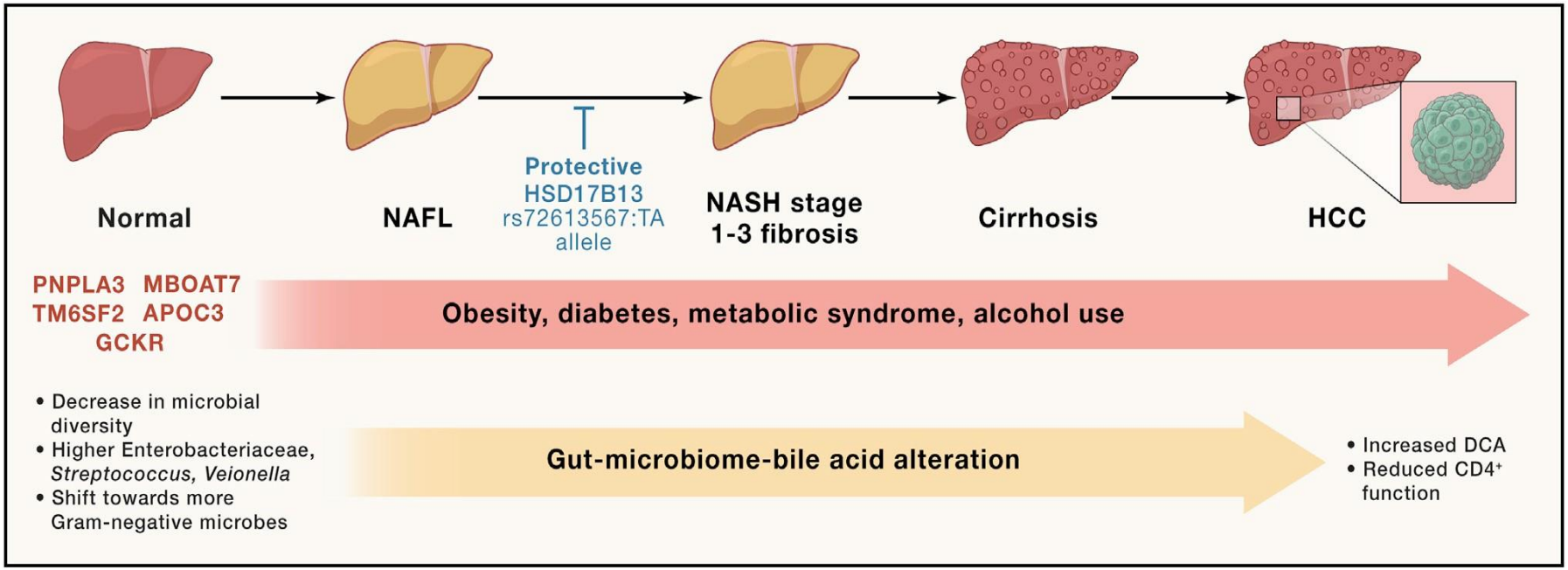
Non-Alcoholic Steatohepatitis



Risk factors and progression of NASH

- NASH, an advanced form of non-alcoholic fatty liver disease (NAFLD), can progress to liver fibrosis, cirrhosis, and hepatocellular carcinoma
- Genetic factors, environmental factors, and microbiome alterations are mainly involved in disease progression

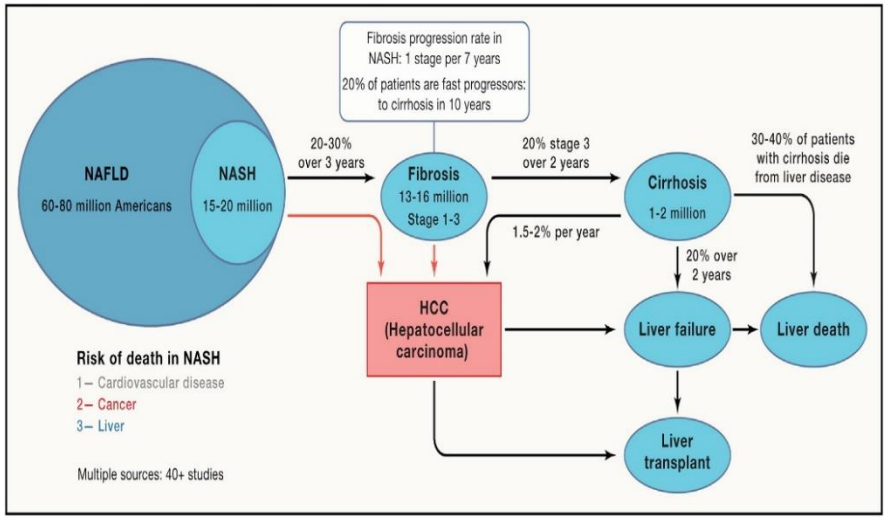


Loomba and Shulman, 2021

NASH population

- Experts estimated that **24%** of U.S. adults have NAFLD
- **6.5%** of NAFLD have NASH
- **20~ 30%** of patients with NASH progress to liver fibrosis in **7** years
- **20%** of them progress to cirrhosis within two years

NIH report, 2021

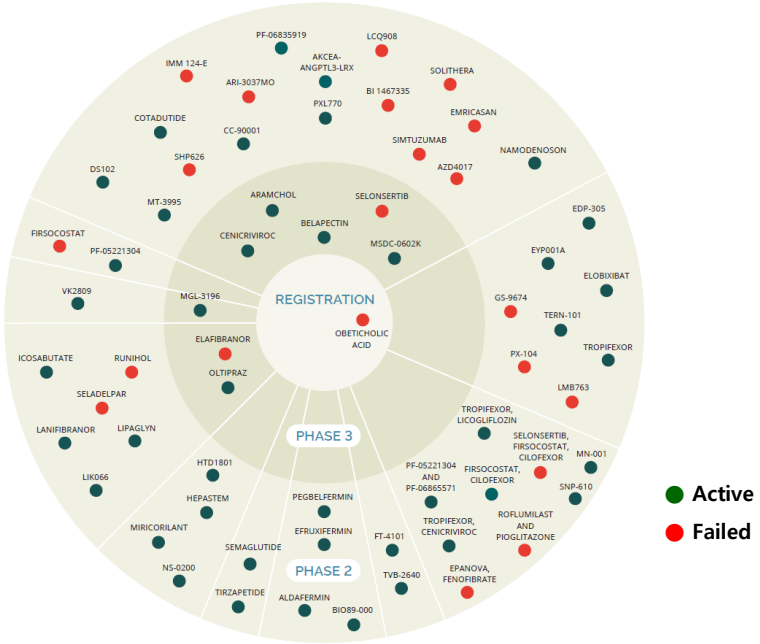


Loomba and Shulman, 2021

Unmet Need

More than 100 drug candidates are currently in clinical trials

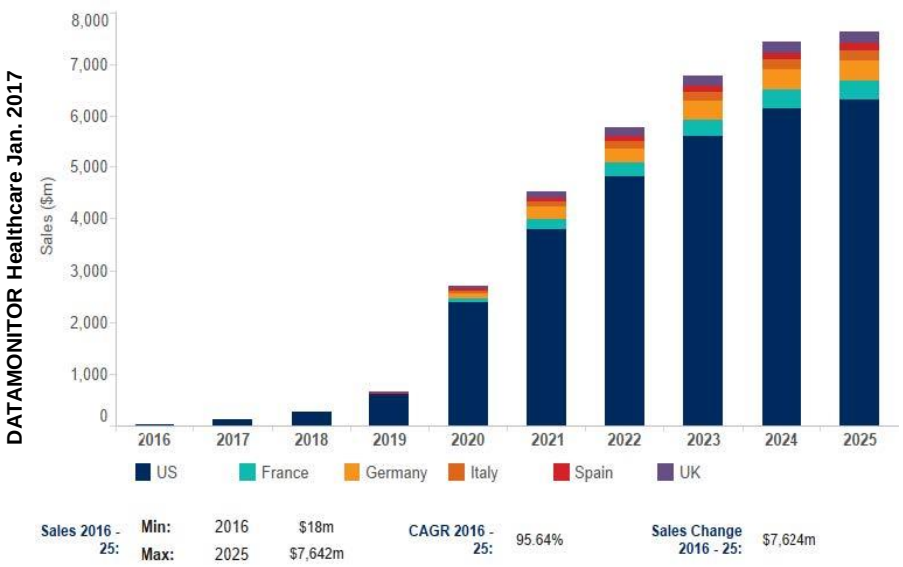
NO drugs for NASH are commercially available



Back Bay Life Science Advisors, 2020

NASH market overview

- Current market size of NASH treatment **<$1 billion** due to a lack of drug options
- The sale volume of NASH drugs is expected to **grow rapidly**



NASH drug sales (2016 to 2025)

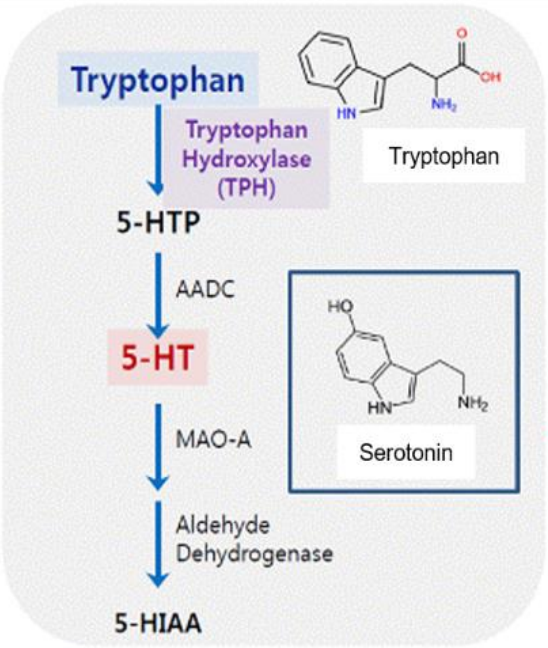
Licensing Deals

- NASH assets **(> $100 million**)
- Majority of these assets are small molecules.

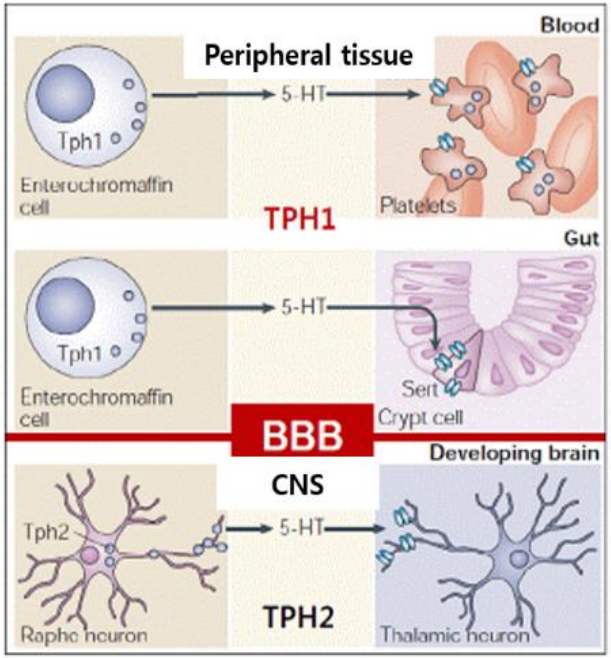
Year	Licensor	Licensee	Active ingredient	Stage	Licensing fees
2014. 12.	Phenex	Gilead	**FXR agonist**	P2	**$470M**
2015. 05.	Pharmaxis	B.I.	**SSAO/VAP-1**	P1	**$600M (upfront: $31M)**
2016. 09.	Akarna Therapeutics	Allergan	**FXR agonist**	P.C.	**Unknown (upfront: $50M)**
2016. 09.	Tobira Therapeutics	Allergan	**CCR2/5 antagonist**	P2	**$1.7B**
2019. 01.	Yuhan Corp.	Gilead	**Small molecules (undisclosed)**	P.C.	**$785M (upfront: $15M)**
2019. 07.	Yuhan Corp.	B.I.	GLP-1/FGF21 dual agonist	P.C.	**$870M (upfront: $40M**
2019.12.	Pliant Therapeutics	Novartis	**αVβ1 integrin agonist**	P.C.	**Undisclosed (upfront: $80M)**
2020. 08.	Thera Biosciences	LG Chem	**VAP-1 antagonist**	P.C.	**$350M**
2020. 08.	Hanmi Pharma	Merck & Co.	GLP-1/GCG dual agonist	P2	**$870M (upfront: $10M)**
2020. 11.	Enleofen	B.I.	IL-11 AB	P.C.	**$1B**
2020. 12.	Aligos Therapeutcis	Merck & Co.	Oligo nucleotide	P1	**$458M**

Licensing deals (2014 to 2020)

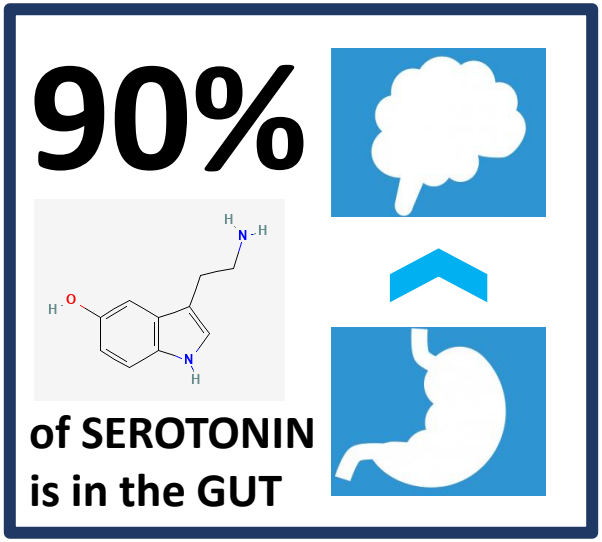
Serotonin plays an important role in our gut



Côté PNAS, 2003



Walther and Bader Biochem Pharmacol, 2003



Berger M Annu Rev Med, 2009

Involved in storage of lipid in White adipose tissue

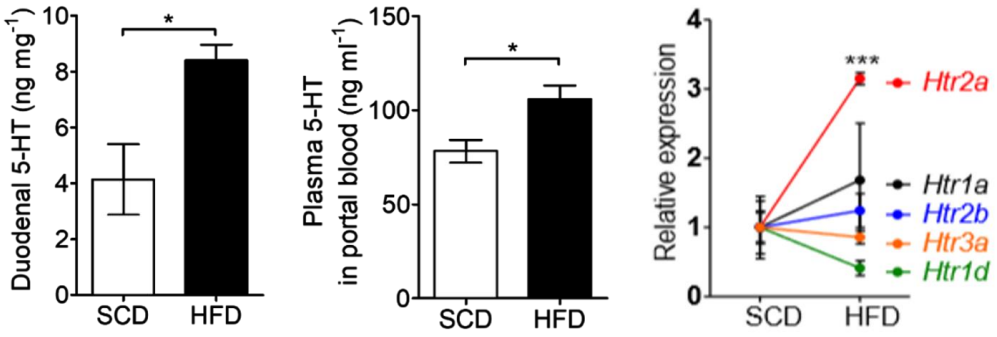
Neurotransmitter

5HT-Serotonin
TPH-Tryptophan Hydroxylase

Highlights

- In a mouse model, a high-fat diet (HFD) increases both serotonin (5-hydroxytryptophan, 5-HT) levels in the plasma levels (portal blood) and serotonin receptor (5-HT receptor 2a, HTR2a) expressions in the liver.
- Activation of the serotonin receptor induces lipogenic gene expression, which in turn enhances lipid storage in hepatocytes
- GM-60106 can effectively reduce lipogenesis, inflammation, and fibrosis in the liver and doesn't induce any BBB-mediated side effects.

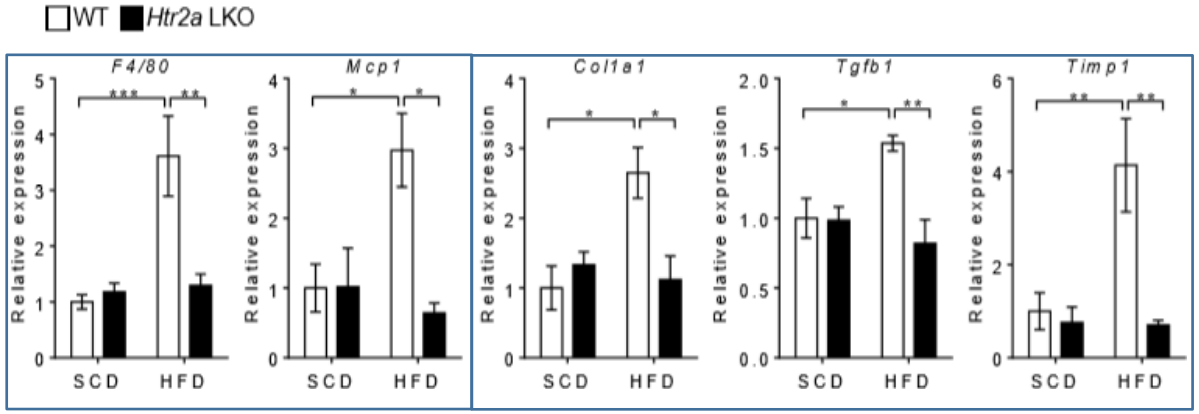
Expression of serotonin and its receptors in peripheral tissues



Kim Nat Comm, 2018

SCD-Standard Chow Diet
HFD-High Fat Diet
Htr-5HT (serotonin) receptor

- Reduction in inflammation and fibrosis-related gene expression in the liver transcriptome



Inflammation marker genes

F4/80-Macrophage marker
MCP1-Monocyte chemoattractant protein1

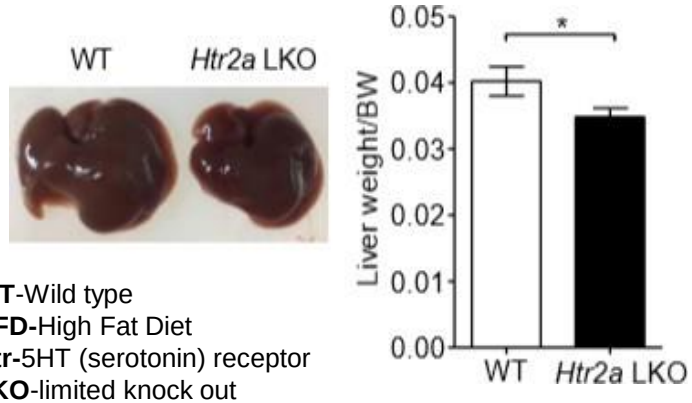
Fibrosis marker genes

Col1-Collagen 1
Tgfb1-Tranforming growth factor beta 1
Timp1-tissue inhibitor of matrix metalloproteinase 1

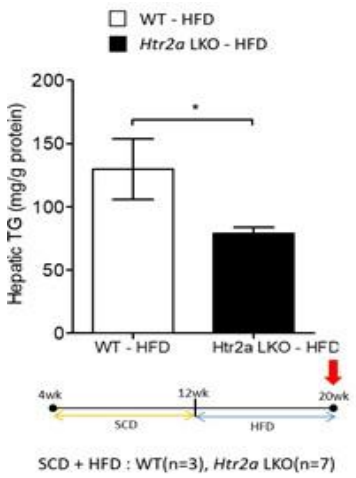
These results clearly indicates that HTR2a is important for hepatic steatosis

- Reduction in hepatic steatosis and the NAFLD activity score

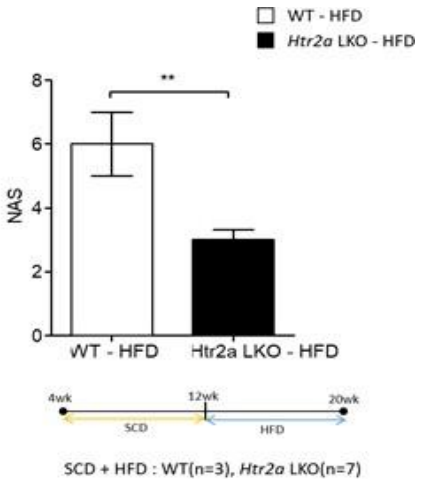
Liver Weight



Hepatic Triglycerides



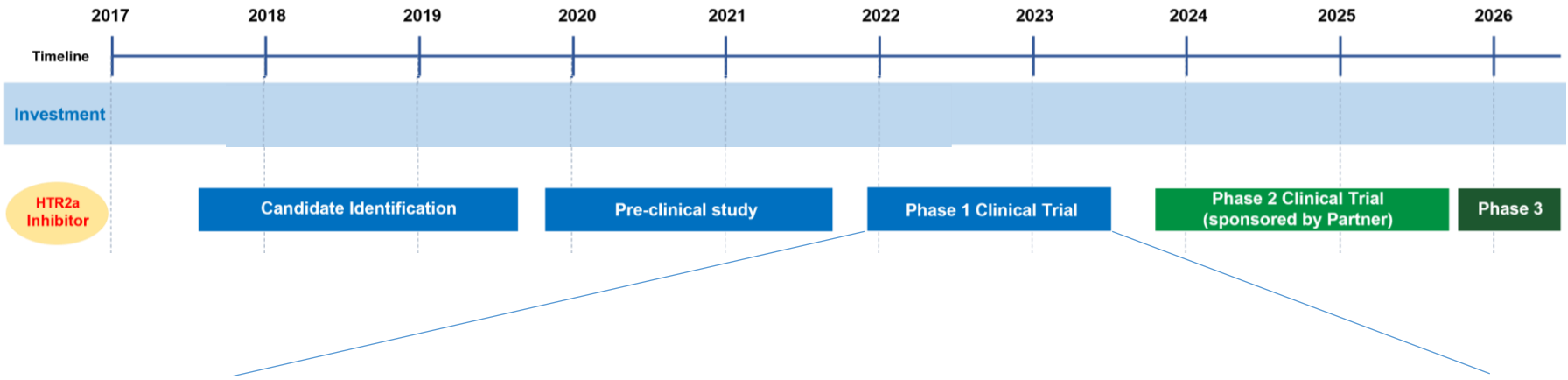
NAFLD activity score



WT-Wild type
HFD-High Fat Diet
Htr-5HT (serotonin) receptor
LKO-limited knock out
NAFLD-Non-alcoholic fatty liver disease

Kim Nat Comm, 2018

Code	*In vitro* (cellular assay)	Solubility/PPB/ CLogP	Chemical stability	Hepatocyte stability (3 hrs)	Plasma stability
GM60106	**14 nM**	1.3 mg/mL pH = 6.8/ 97.5 % (m) 96.7 % (r) 97.1 % (h) /3.16	99.8 % (25 °C, 3 weeks) 99.6 % (60 °C, 3 weeks) No form change	94 % (human) 88.5 % (dog) 84.6 % (monkey) 68.9 % (rat) 75.0 % (mouse)	>99.9 % (m) >99.9 %(r) >98.6 ± 11.7 %(h) (% remaining after 4 hrs)
Cytotoxicity (IC_{50})	**AMES**	**Acute Toxicity**	**CYP inhibition (at 10 μM)**	***In vivo* PK (rat)**	**In vivo PK (dog)**
VERO > 100 μM HFL-1 > 100 μM L929 > 100 μM NIH3T3 > 100 μM CHO-K1 > 100μM	Negative	LD_{50} > 1000 mpk	1A2: < 1 % 2C9: 3.17 % 2C19: 6.83 % 2D6: 26.3 % 3A4: 17.6 %	IV (5mpk) Oral (10mpk) T1/2: 4.14 h AUC: 2.88 ug.h/ml CL: 2.82 L/h/kg V: 8.86 L/kg **BA 61 %**	IV (5mpk) Oral (5mpk) T1/2: 8.6h AUC: 19.11 ug.h/ml CL: 0.42 L/h/kg V: 4.36 L/kg **BA 73 %**
***In vivo* efficacy in normal with high fat**	***In vivo* efficacy in DIO mice**	**NASH in MCD diet in db/db mice**	**STAM mice**	**Dog telemetry**	**BBB permeability**
Body weight gain reduction, Reduced fat accumulation 5, 10 mpk	Body weight gain reduction, Reduced fat accumulation in liver 5,10 mpk	Reduced inflammation, fibrosis and fat accumulation in liver 5 mpk	Reduced inflammation and fibrosis in liver 5, 10 mpk	Cardiovascular Radiotelemetry Assessment in Conscious Dogs 50,100, 200 mpk (No change)	BQL (below lower limit) in Brain based on tissue distribution of [14C]-GM-60106



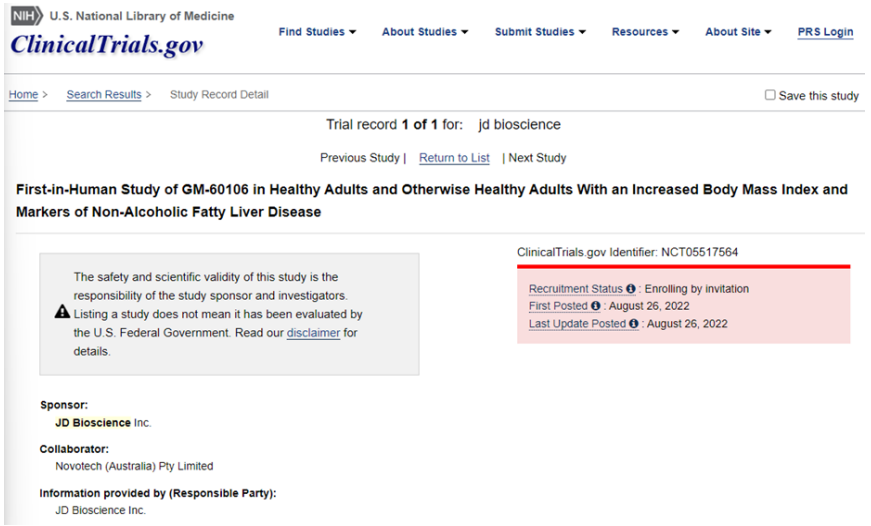
Sep 2022 **March 2023** **July 2023** **Sept 2023**

Part A (SAD Cohort 1-7) 1 Day → **Part B (MAD Cohort 1-3) 14 Days** → **Part C (MAD Cohort 4 -5) 28 Days**

NOVOTECH The Asia Pacific CRO | Nucleus Network | AUSTRALIAN Clinicalabs | ZUELLIG PHARMA making healthcare more accessible | 360biolabs a BioAgilytix company | CLARIO.

ClinicalTrials.gov

https://clinicaltrials.gov/ct2/show/NCT05517564



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Home > Search Results > Study Record Detail | Save this study

Trial record **1 of 1** for: jd bioscience

Previous Study | Return to List | Next Study

First-in-Human Study of GM-60106 in Healthy Adults and Otherwise Healthy Adults With an Increased Body Mass Index and Markers of Non-Alcoholic Fatty Liver Disease

The safety and scientific validity of this study is the responsibility of the study sponsor and investigators. Listing a study does not mean it has been evaluated by the U.S. Federal Government. Read our disclaimer for details.

ClinicalTrials.gov Identifier: NCT05517564

Recruitment Status : Enrolling by invitation
First Posted : August 26, 2022
Last Update Posted : August 26, 2022

Sponsor:
JD Bioscience Inc.

Collaborator:
Novotech (Australia) Pty Limited

Information provided by (Responsible Party):
JD Bioscience Inc.

SAD-Single Administration Dosing
MAD-Multiple Administration Dosing

- Since NASH is a multifaceted disease caused by various cellular and signaling events, combination therapies that can target multiple cellular signaling pathways might be crucial for developing effective drugs for NASH.
- Hence, diverse combination studies are at the clinical stage.

We are seeking opportunities to develop additional combination therapies.

"We expect that treatment response can be enhanced by strategically combining multiple agents targeting distinct metabolic pathways to not only achieve resolution of NASH and improvement in fibrosis but also a reduction in cardiovascular disease risk and future risk of cancer."

Loomba and Shulman Cell, 2021

1. **Cenicriviroc (CCR2/5 receptor inhibitor, Allergan) +Tropifexor (FXR agonist, Novartis)**
2. **Semaglutide (GLP-1 agonist, Novo Nordisk) + Cilofexor (FXR agonist, Gilead) + Firsocostat (ACC inhibitor, Gilead)**
3. **Firsocostat (ACC inhibitor, Gilead) + Fenofibrate (PPAR-α, Abbvie)**
4. **Cilofexor (FXR agonist, Gilead) + Firsocostat (ACC inhibitor, Gilead) + Selonsertib (ASK1 inhibitor, Gliead)**
5. **Licogliflozin (SGLT1/2 inhibitor, Novartis) +Tropifexor (FXR agonist, Novartis)**
6. **PF-05221304 (ACC1/2 inhibitor, Pfizer) + PF-06865571 (DGAT2 inhibitor, Pfizer)**

CCR-Chemokine Receptor
FXR-Farnesoid X Receptor
GLP-glucagon-like peptide
ACC-Acetyl-CoA Carboxylase
PPAR-Peroxisome Proliferator-Activated Receptor
ASK1-Apoptosis Signal-regulating Kinase
SGLT-Sodium-glucose Cotransporter
DGAT- Diglyceride acyltransferase

Intellectual property management plan

Patent for a composition of matter was filed in 2019

International Patents were filed in 2022

IP Country Filing


KR

HTR2 antagonist

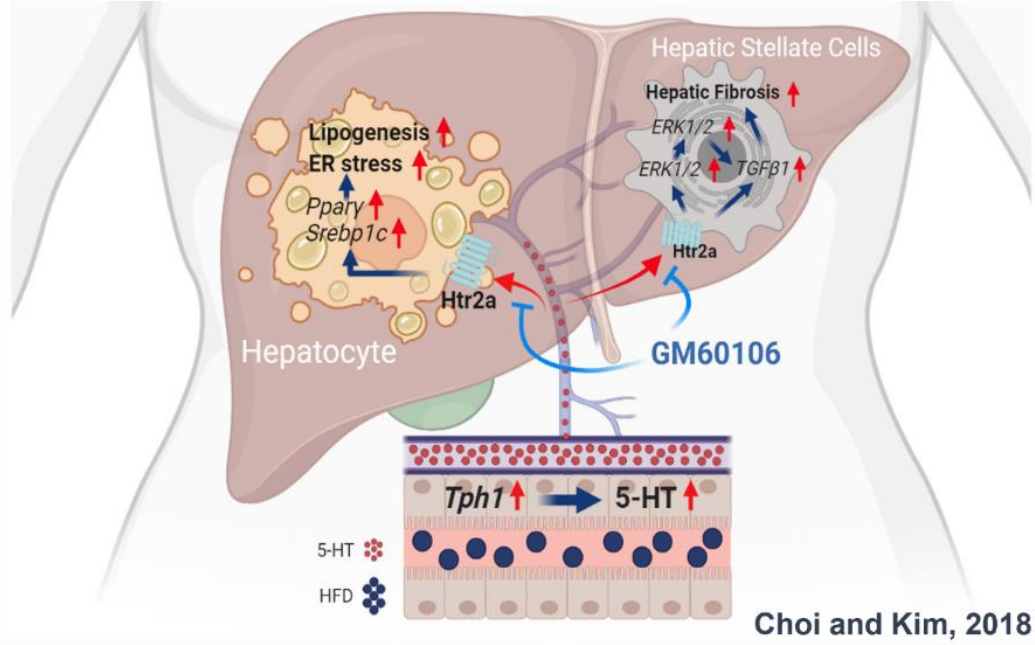
Composition of matter

Filed in Korea

10-2019-0137775

Application claiming priority / filed PCT

Entry into the International Phase

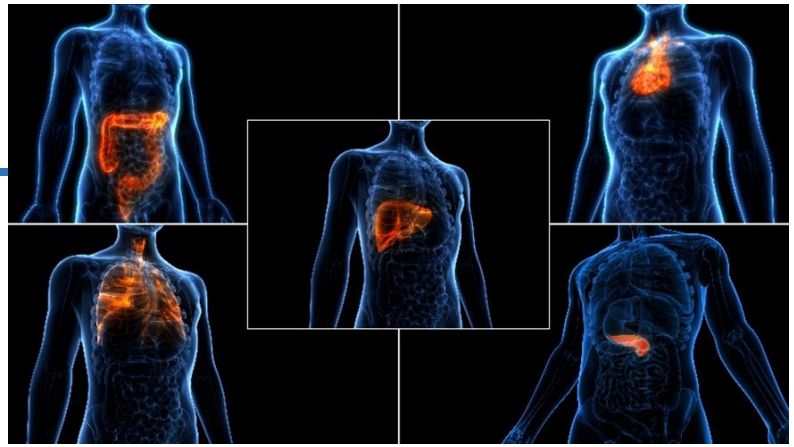


Summary

- **Discovered unknown roles for HTR receptor-mediated serotonin signaling in liver, which is related to the progression of NASH and liver fibrosis.**
- **GM-60106 compound was synthesized a chemical compound that directly inhibits HTR2a receptors on liver cells.**
- **GM-60106 preclinical studies were completed showing reduction in lipid accumulation in the liver, fibrosis, and steatosis.**
- **Proprietary compounds are IP protected**

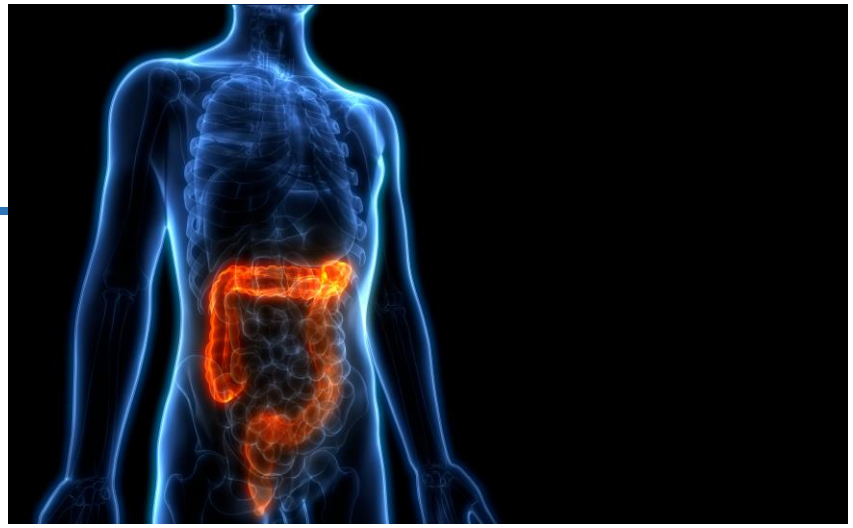
JD BIOSCIENCE

Inflammation



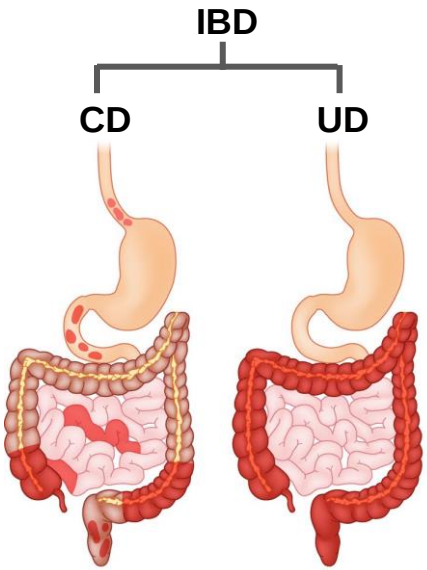
JD BIOSCIENCE

Inflammatory Bowel Disease



Inflammatory Bowel Disease (IBD)

- Caused by Genetics and Environmental Factors
- Experts estimated that 1.2% of U.S. adults have IBD and the incidence rate of this population is rapidly rising.
- Pro-inflammatory cytokines, such as TNF, IL-6, produced by the activated immune cells are suggested to drive the perpetuation of inflammation and tissue damage.



Genetics

Susceptibility, Inheritance

Environmental Factors

Microbial & dietary toxins, infection, drugs

Intestinal Barrier

Permeability, autophagy, goblet cells

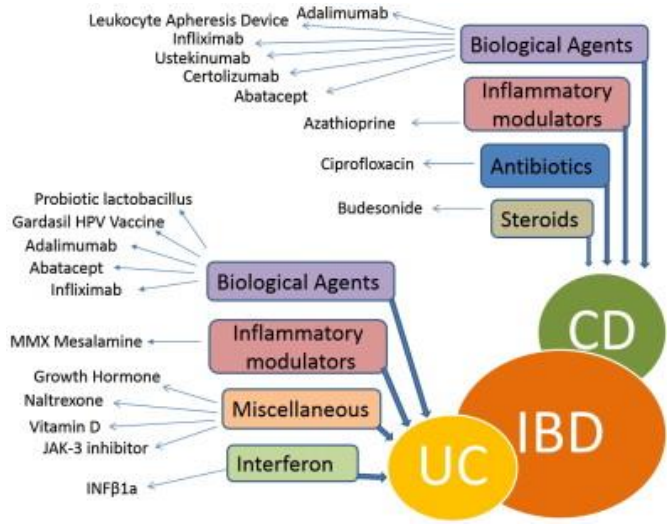
Immune Response

Innate response, cytokine expression

Neurath 2019, Nat. Immunol.

Unmet Need

- Unresponsive to biologics (anti-TNFα, anti-integrin, IL12/IL23): approximately 30% of patients
- Losing response among previous responders: up to 10%/year
- Significant infectious and neoplastic side effects with current IBD medications

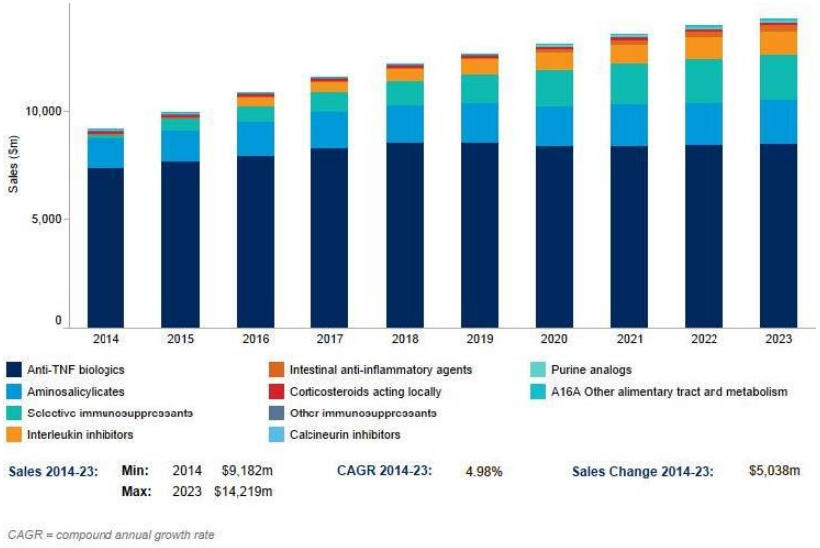


Chandel 2015, Pharm. Rep.

DATAMONITOR Healthcare Jan. 2016

IBD market overview

- Current market size of IBD treatment **>10 BIL USD**
- The sale volume of IBD drugs is expected to **grow rapidly**



2014 to 2023 IBD drug sales in the seven countries

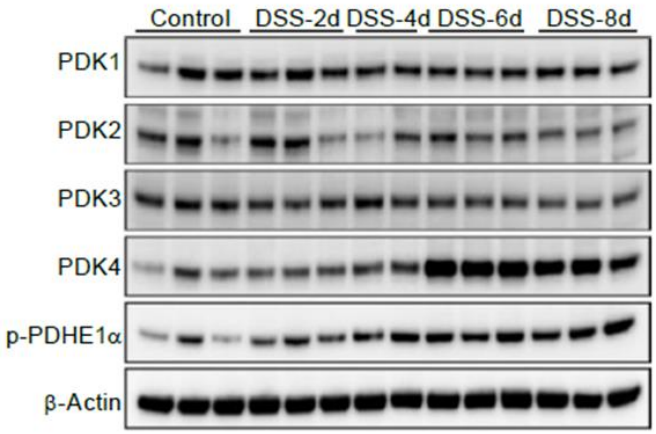
Licensing Deals

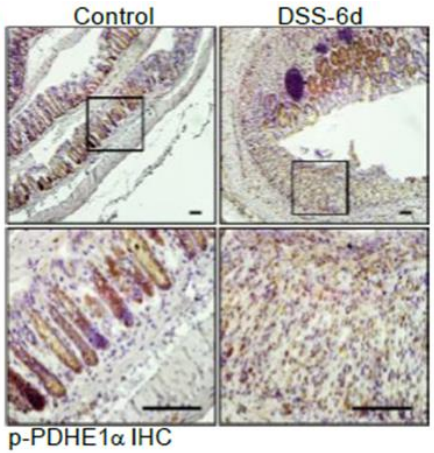
- Anti-TNF antibodies hold the largest market share in the IBD therapeutics market.
- While the market share of interleukin inhibitors and chemical antagonists are **increasing.**

Year	Licensor	Licensee	Active ingredient	Stage	Licensing fees
2016 10.	AstraZeneca	Allergan	**IL-23 AB**	P2	**$1.27B (upfront: $250M)**
2017. 04.	Finch Therapeutics	Takeda	**FIN-524 microbiomes**	P.C.	**Undisclosed (upfront: $10M)**
2017. 05.	Protagonist	J&J	**IL-23 inhibitor (oral peptide)**	P.C.	**$0.99B (upfront: $50M)**
2017. 09.	Janssen	Provention Bio	**CSF-1R antagonist, TLR3 AB**	P1/P.C.	**Undisclosed**
2018. 12.	Bridge Bio	Daewoong Pharma	**Pelliono-1 Antagonist**	P1	**$40M**
2019. 04.	IFM therapeutics	Novartis	NLRP3 Antagonist portfolio	P.C.~P1	**$310M ~ $16B**
2019. 07.	Alfasigma	Innovation Pharma	Brilacidin (non-corticosteroid)	P2	**$24M + upfront**
2019. 09.	Prometheus Biosciences	Takeda	(>200,000) Patient samples	Discovery	**$420**
2020. 05.	Gossamer Bio	Aerpio Pharma	HIF-1a stabilizer	P.C.	**$90M (upfront: $15M)**

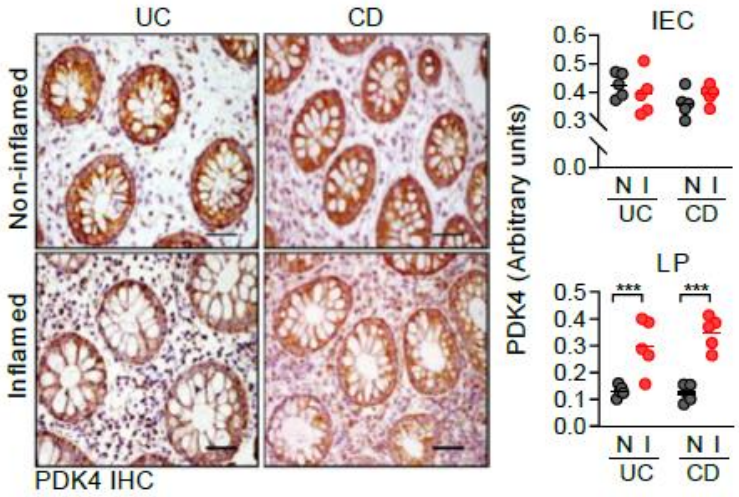
Licensing deals of IBD assets from 2016 to 2020

Activated Helper T Cells Express PDK





In vivo colitis mouse model



PDK is over expressed in inflammatory bowel Disease

PDK-Pyruvate Dehydrogenase Kinase
PDHE1-Pyruvate dehydrogenase E1
DSS-Dextran sulfate sodium
CD-Crohn's Disease
UC-Ulcerative colitis

Human patients

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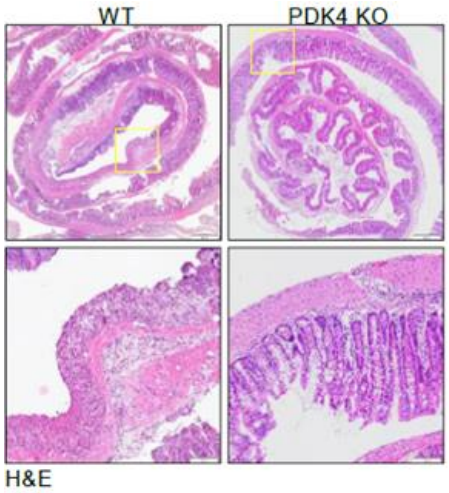
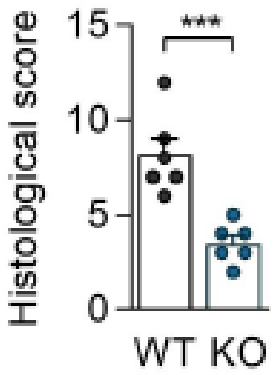
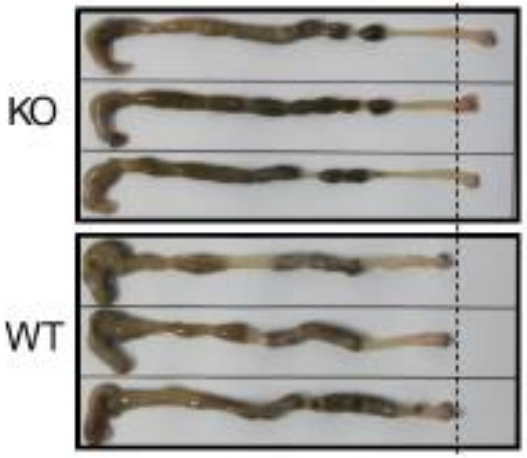
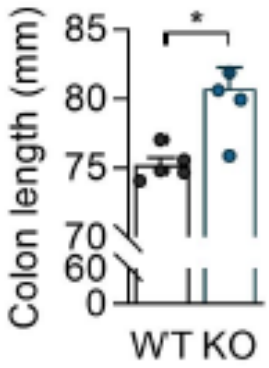


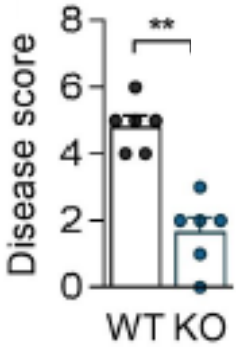


Protection of epithelial lining and reduced inflammation

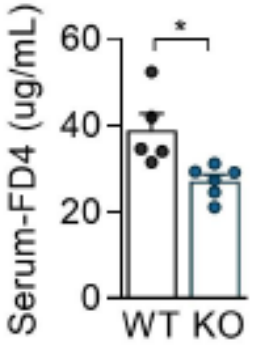




Colon length was not shortened



Disease scores decreased



Permeability decreased

Deletion of PDK4 from CD4+ T cells protects against DSS-induced colitis

WT-Wild Type
KO-Knock out
FD4-fluorescein isothiocyanate dextran
DSS-Dextran sulfate sodium

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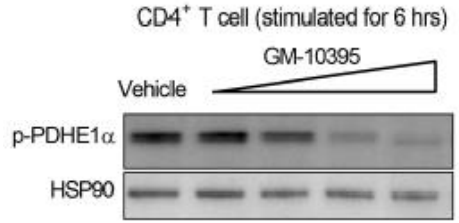
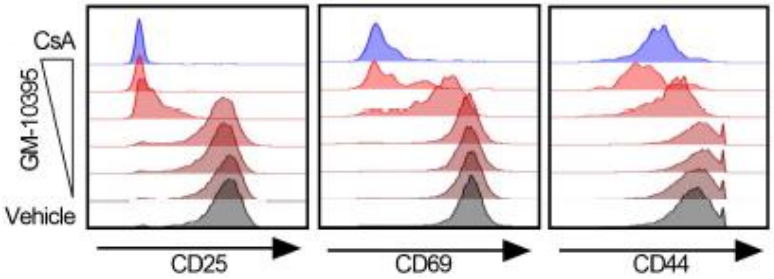
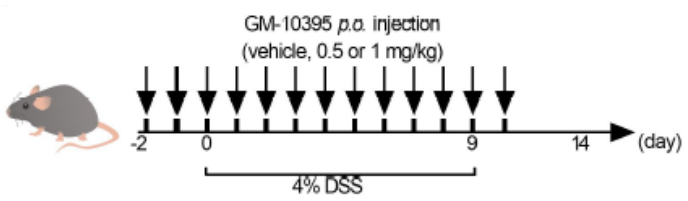
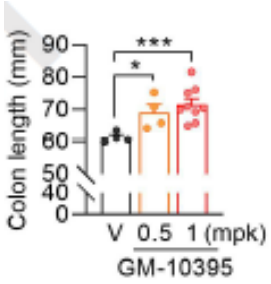


GM-10395 reduces p-PDHE1 in a dose dependent manner

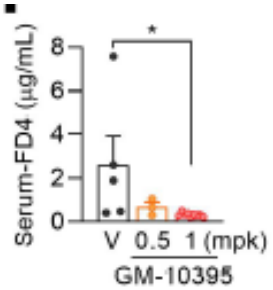


T cell activation markers reduced in a dose dependent manner

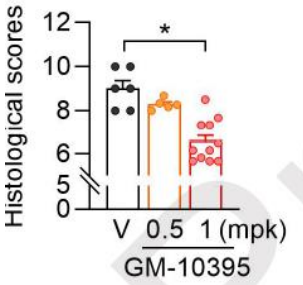




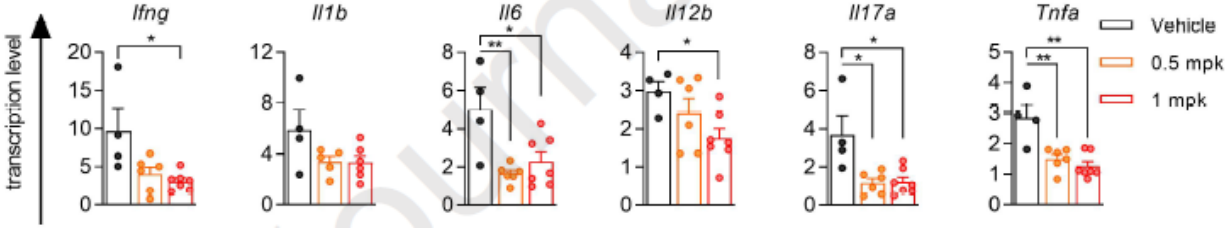
Colon length remained normal



Permeability decreased



Histological Scores improved



Inflammation markers reduced

WT-Wild Type
KO-Knock out
FD4-fluorescein isothiocyanate dextran
DSS-Dextran sulfate sodium

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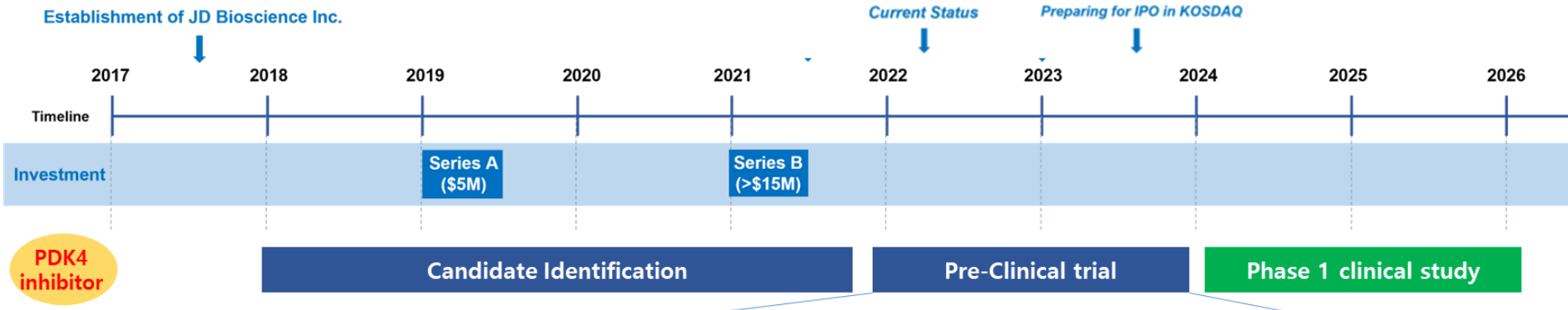
Plan for pre-clinical and clinical study

- Non-GLP toxicology study in progress
- Will start GLP in Q1 of 2023



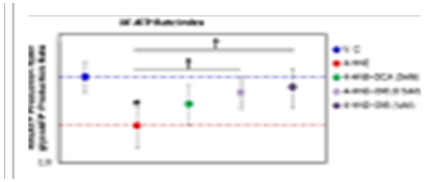
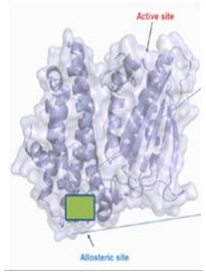
Non-GLP toxicology study

- Clinical signs and mortality
- Body weight
- Hematological Exam
- Hormone Analysis
- Tissue weight
- Histology

Efficacy study

Pharmacological effects on target indications

Preclinical Compound	Comprehensive ADME	GLP toxicology Package	Efficacy Studies
• **Chemical synthesis (3kg)** • **Salt screening** • **Crystal form synthesis** • Stability (API, 1year)	• Analytical method development • Pharmaco-kinetics (ADME) • Pharmaco-kinetic drug interaction • Identification of Metabolite	• Single-dose toxicity • Repeated dose toxicity • Genotoxicity • Reproductive & developmental toxicity • Local Tolerance • etc.	• NASH animal model selection • MICE • Non-human primate etc.

Code	Inhibitor Class	In vitro (cellular Assay)	Recovery of mitochondria function		
GM-1039506	Allosteric inhibitor	Inhibition of PDHE1α Phosphorylation 159nM(IC_{50})		GM10395 recovered Mitochondria function with Dose dependent manner	
CYP inhibition (IC_{50} μM)	**HERG inhibition**	**PPB**	**Plasma stability (4hr incubation)**	**AMES test**	**Acute toxicity**
IA2: 33.69 2C9: 12.64 2C19: 12.18 2D6: 7.94 3A4: 7.72	32.8% at 10μM	99.3%(m) 99.5%(m)	Human 89%	Negative	LD50> 1000mpk
In vivo PK	**In vivo IBD study**	**In vivo Anti-cancer efficacy**	**In vivo efficacy (anti-diabetes)**	**Structure**	
IV(5mpk) Ora (10mpk) T1/2: 5.15h AUC:1.76 μg/ml(IV) BA: 48%	Improved histological Score colon length In vivo at 1mpk	Reduced tumor Volume In xenograft mice At 10mpk dose	Glucose AUC reduction (OGTT) Oral administration		

Intellectual property management plan

Patent for a composition of matter was filed in 2022

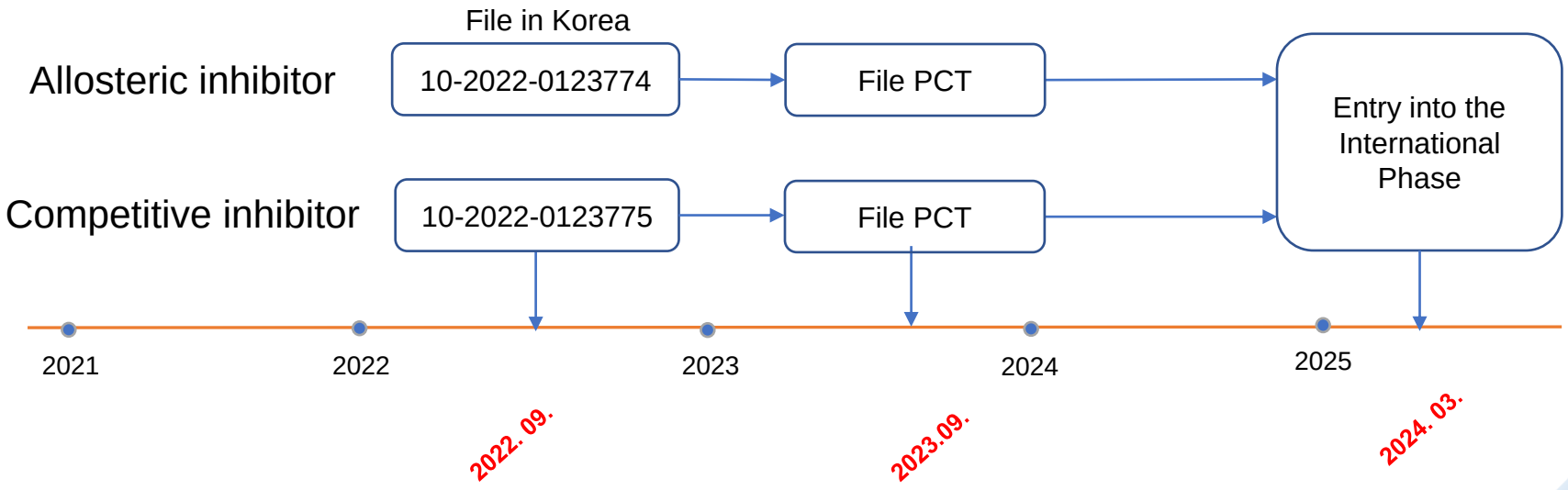
- Will fill international patents in 2024

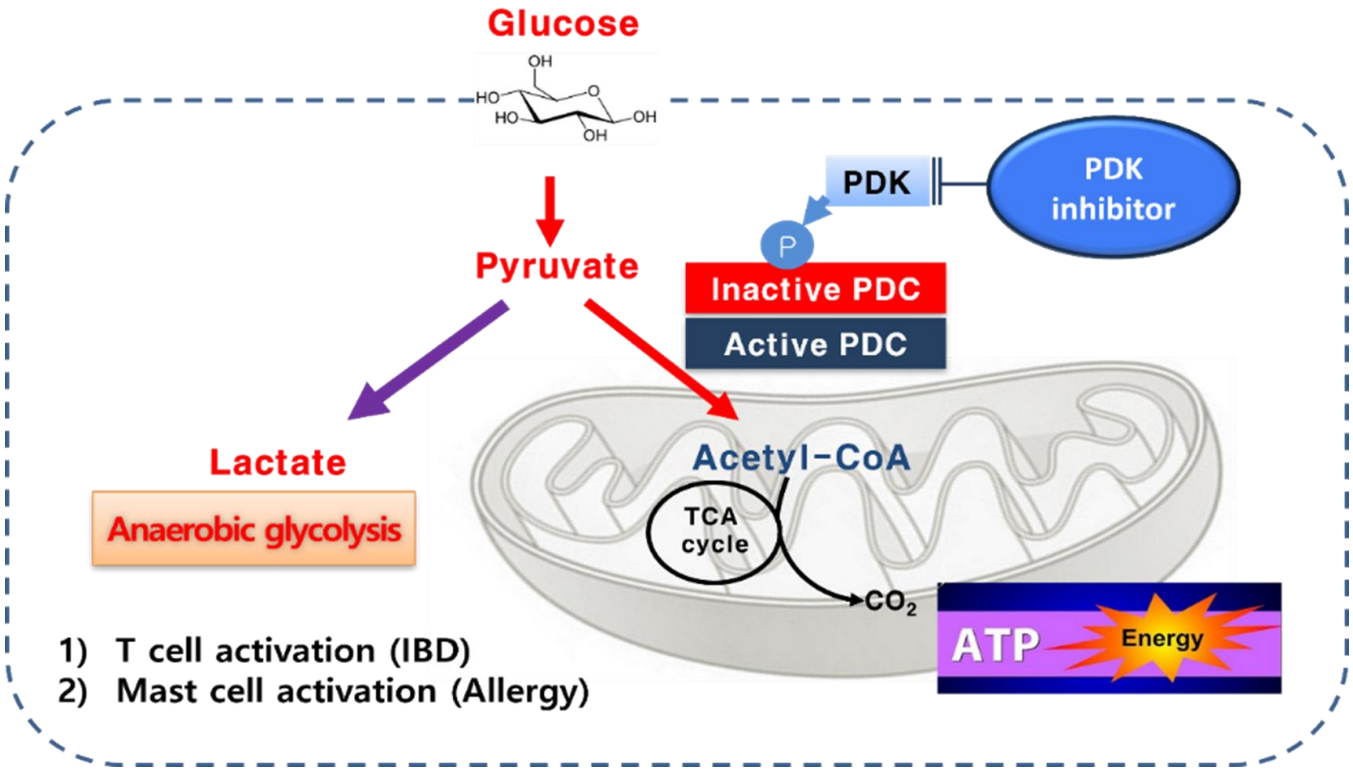
Backup compound was also prepared

- Patent was filed in 2022.

Planned IP Country Filing

PDK antagonists





Hoyul Lee Cell Mol Gastroenterol Hepatol, 2022

Summary

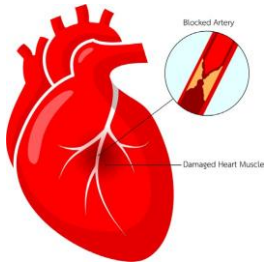
- Discovered unknown roles for PDK in CD4+ T cells , which is related to the progression of IBD.
- GM-10395 compound directly inhibits PDK signaling in CD4+ T cells.
- GM-10395 preclinical studies show a reduction in colitis.
- Proprietary compounds are IP protected.

JD BIOSCIENCE

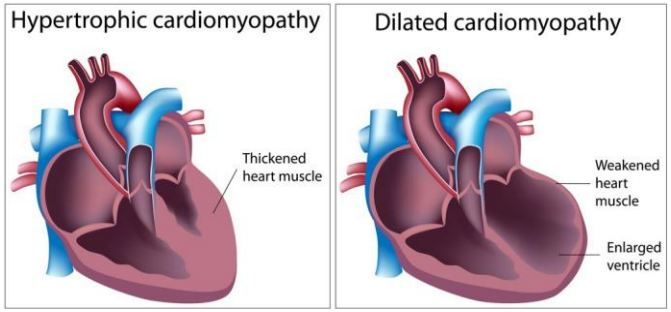
Heart

- Heart disease, a leading cause of death globally, can be categorized into ischemic heart disease, cardiomyopathy, and myocarditis.
- Ischemic heart disease is involved in the reduction of blood flow due to the accumulation of fatty deposits in the coronary arteries. Cardiomyopathy, including hypertrophic or dilated cardiomyopathy, is a disease of the heart muscle that can occur due to various risk factors. Finally, myocarditis is characterized by the inflammation of the heart muscle, which often occurs due to a viral infection. All of these can ultimately lead to fatal heart failure.

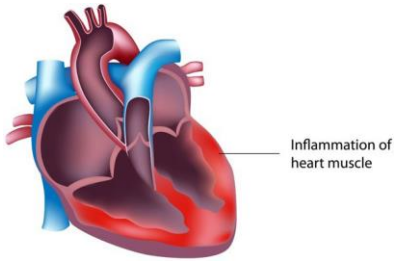
Ischemic heart disease



Cardiomyopathy



Myocarditis

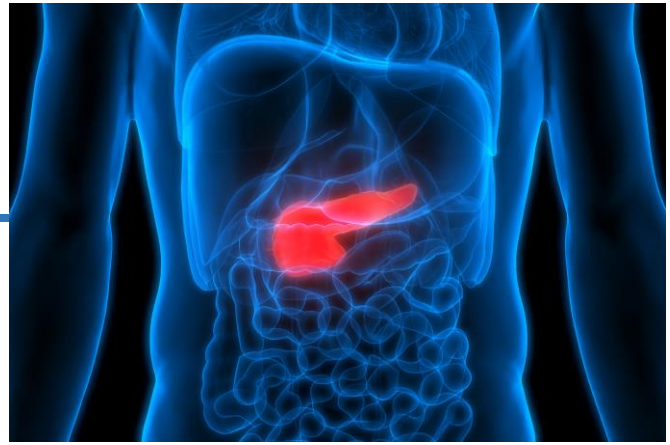


- Different types of medication are available to treat heart disease, for example, cholesterol-lowering medications, beta-blockers, nitroglycerin, and calcium channel blockers. However, none of these can rescue the function of cardiomyocytes once heart failure is in progression.

JDB is developing a novel drug candidate for heart disease that can rescue heart function at a cellular level, and can eventually help improving heart function

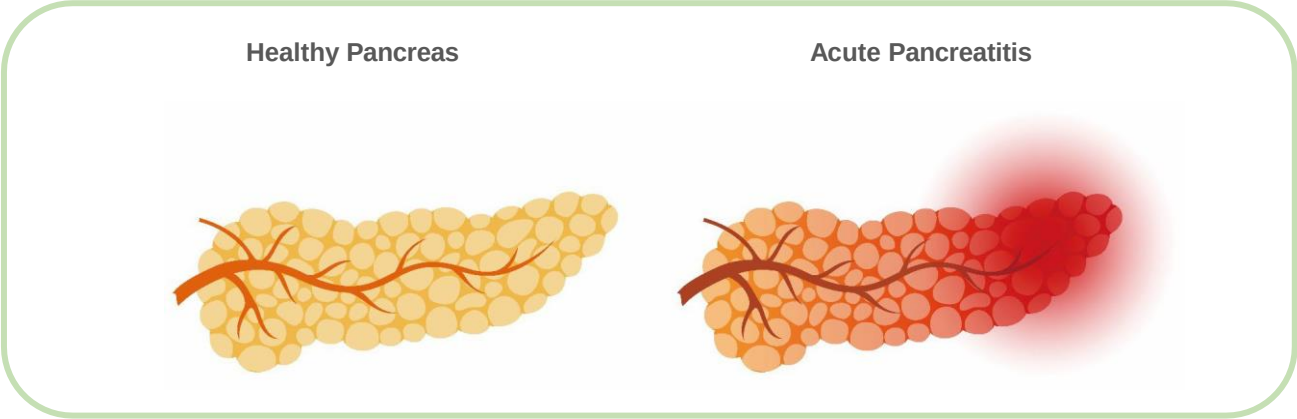
JD BIOSCIENCE

Pancreas



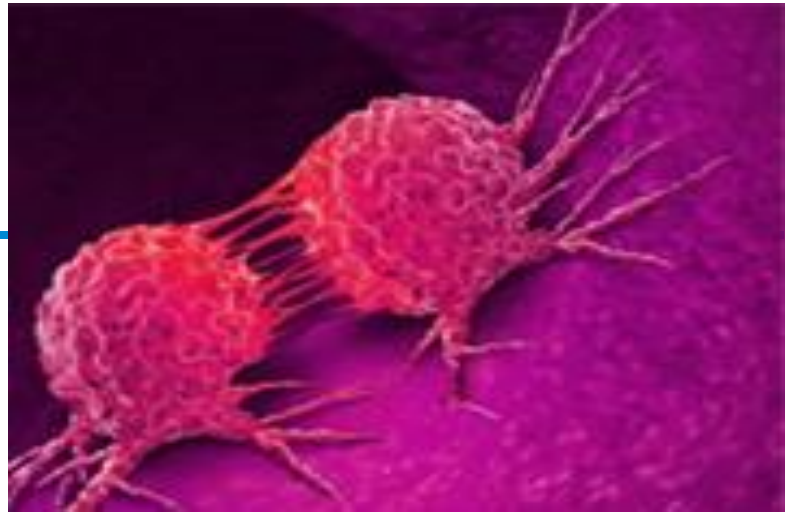
Risk factor and causes of Pancreatitis

- Acute pancreatitis (AP) is characterized by the sudden inflammation of the pancreas and occurs when digestive enzymes within the pancreas are abnormally activated. The main risk factors of AP are gallstones, which make up 40% of cases, and alcohol misuse, which makes up 30% of cases. Recent findings also indicate that type 2 diabetes or smoking can increase the risk of non-gallstone-related AP.
- AP was the second-highest cause of total hospital stay and the fifth leading cause of in-hospital death in the USA in 2015. However, to date, no specific causal treatment for AP is available. Instead, supportive care, such as pain control, is the only available treatment.



JDB is developing a novel drug candidate for acute pancreatitis that can effectively reduce inflammation of the pancreas by modulating mitochondrial functions in pancreatic cells.

Cancer



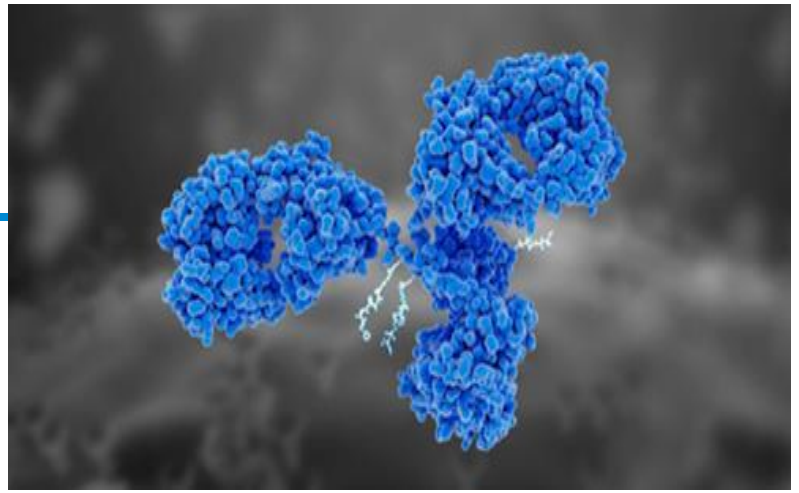
JD BIOSCIENCE

1 ADC

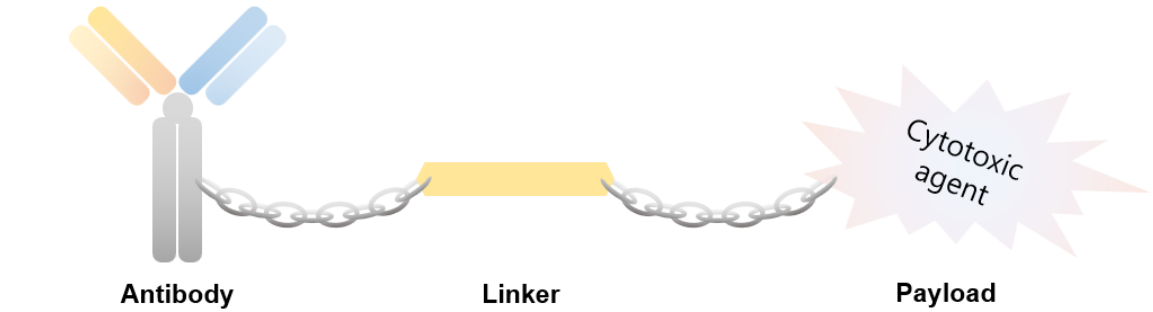
2 PROTAC

ADC

JD BIOSCIENCE

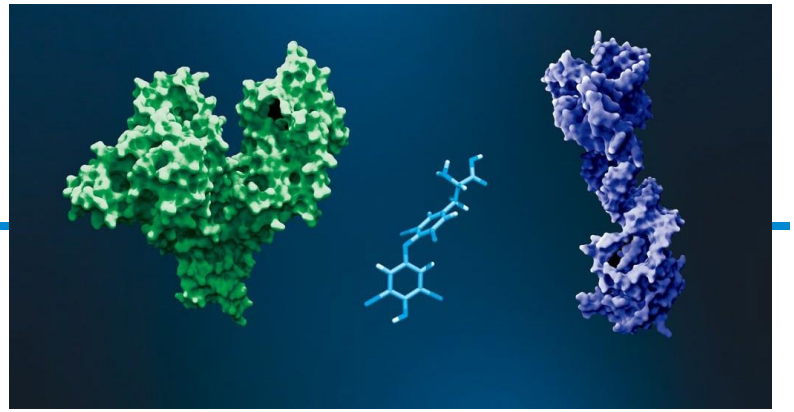


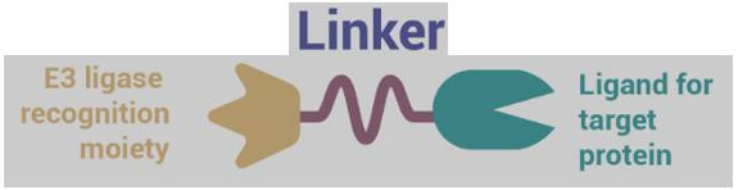
Antibody-Drug Conjugates (ADCs)

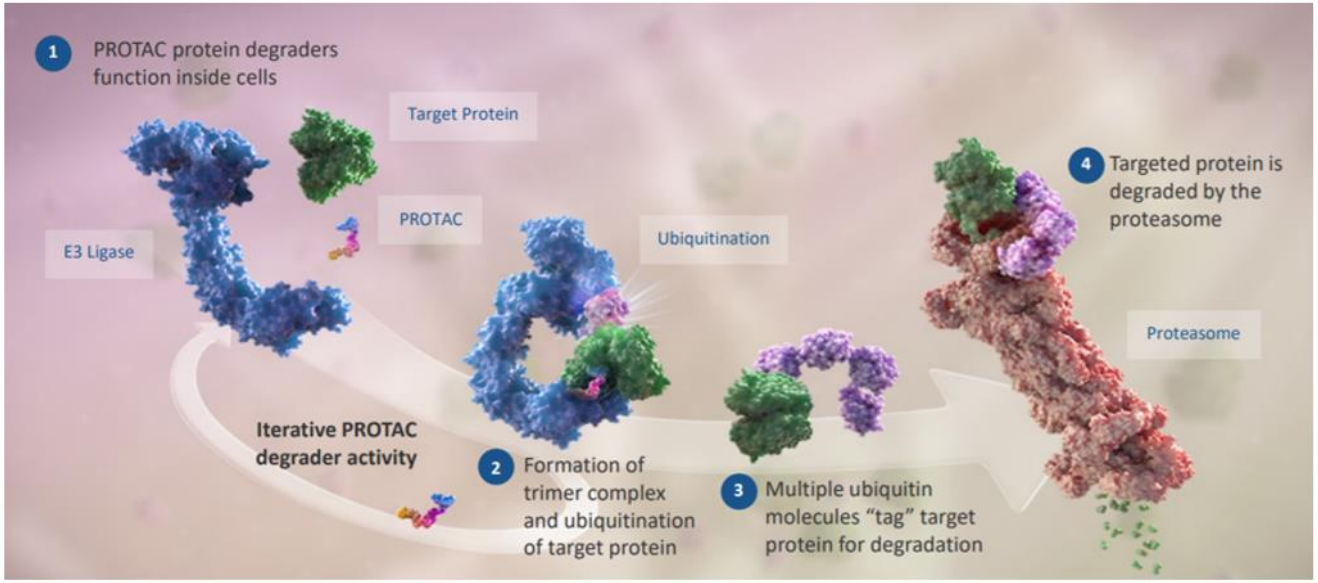


PROTAC

JD BIOSCIENCE







Source: Avinas

JD Bioscience's Strategic Alignment

Licensing out GM60106, GM-10395

- **Exclusive and/or Territorial rights**

Partnership to lead metabolic disease & inflammatory disease market

GM-60106

- ✓ **Innovative First-in-Class Technology: HRT2a**
- ✓ **Mechanism for NASH-related fibrosis**
- ✓ **Safe, NO CNS-mediated safety issues**

GM-10395

- ✓ **Innovative First-in-Class Technology: PDK**
- ✓ **Mechanism for IBD inflammation**
- ✓ **NO adverse side effects in animal model**

THANK YOU

JD BIOSCIENCE

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