JD BIOSCIENCE

JD Bioscience Inc.

IR Non-Confidential Deck Q2 2026

JD Bioscience Inc.

JD Bioscience is seeking **strategic partnerships** to accelerate development and global market entry.

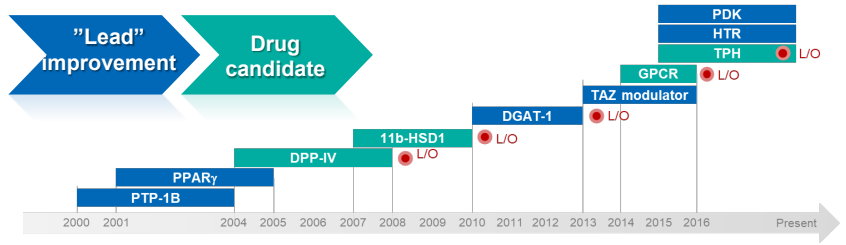
- JD Bioscience is developing first-in-class **drug candidates for liver disease, inflammation and cancer** (metabolic diseases).
- Lead asset (1st), GM-60106, which showed **strong anti-fibrotic efficacy**, has received **FDA approval** for a **Phase 2a clinical trial in the US** for liver disease MASH (metabolic dysfunction associate steatohepatitis)
- Second asset, GM-10395 demonstrated **strong anti-inflammatory efficacy** in pre-clinical study for IBD (inflammatory bowel disease)
- Third asset, GM-10395 demonstrated **anti-epileptic events** in pre-clinical study for Dravet (Rare-epileptic Disease)
- Continuously developing following additional assets (3rd) for **cancer (ADC payload) with pico-molar cytotoxicity in drug resistant cancer cell lines, along with Immune modulating agent and degrading payloads**

Overview of JD Bioscience (JDB)

JD BIOSCIENCE

Name	JD Bioscience Inc.
Date of Est.	July 31, 2017
Web Page	www.jdbiosci.com
Pipelines	▪ Modality: Small Molecule-based novel drug candidates (First-in-class) ▪ Indications: Metabolic Diseases (MASH, cancer etc.)
Business Model	▪ Commercialization of drug assets ▪ Collaborative research with pharmaceutical companies
Number of Employees	▪ 25 members (20 researchers)
Locations	▪ Gwangju, South Korea (Headquarters) ▪ Seoul, South Korea (BD, RA) ▪ Melbourne, Australia (Clinical Study)

"CEO's track record of success"



Jin Hee Ahn

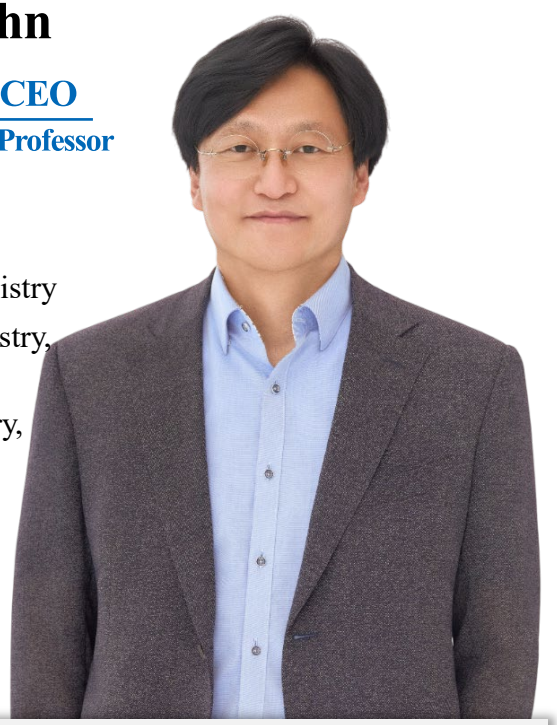
JD Bioscience, CEO

GIST, Chemistry Professor

> 25+ years of experience in medicinal chemistry

> Postdoc, Chemistry, UC Berkeley

> Ph.D., Chemistry, Sogang Univ.



"Drug discovery expert with a proven record of numerous out-licensing and broad experience in various metabolic disease targets"

Core members

JD BIOSCIENCE

JDB was established with medicinal chemists and top-notch physician-based scientists who specialize in novel modalities

JDB Team Leaders

Jin Hee Ahn/Ph.D.

CEO

- 25+ years of experience in medicinal chemistry
- Postdoc, Chemistry, UC Berkeley, USA
- Prof. at GIST, department of chemistry

Hail Kim/ M.D., Ph.D.

Chief Medical Officer

- Prof. at KAIST, school of medical science and engineering
- Postdoc. Fellow at the Diabetes Center, UCSF
- 20+ years of experience in metabolic disease

Dooseop Kim/Ph.D.

CSO

- Former Senior Investigator, Merck & Co., Inc.
- Inventor of a blockbuster diabetes drug, Januvia at Merck & Co., Inc.

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

Co-Founder

- Prof. at KNUH, Department of Endocrinology and Metabolism
- Ph.D., School of Medicine, Kyungpook National University
- 35+ years of experience in metabolic diseases

Scientific Advisory Board

UC San Diego

Rohit Loomba M.D. / MH.Sc

Professor at UCSD

HARVARD MEDICAL SCHOOL

George L. King M.D.

Professor at Harvard Medical School

YONSEI UNIVERSITY COLLEGE OF MEDICINE

Junyong Park M.D. / Ph.D.

Professor at Yonsei University

YONSEI UNIVERSITY COLLEGE OF MEDICINE

Jung Il Lee M.D / Ph.D.

Professor at Yonsei University

Pipeline Overview

JD BIOSCIENCE

We have drug pipelines spanning from discovery stage through to clinical development

Candidate	Indication	Target	Development Phase	Plans for BD
GM-60106	MASH/Fibrosis	HTR	Discovery / Pre-clinical / **Phase 1** / Phase 2	L/O Collaborators
PDK inhibitor Platform	Inflammatory Diseases*	PDK	Discovery / Pre-clinical / Phase 1 / Phase 2	L/O Collaborators
GM-91466	Dravet Syndrome	Confidential	Discovery / Pre-clinical / Phase 1 / Phase 2	L/O Collaborators
JD-ADC payload	Cancer	Confidential	Discovery / Pre-clinical / Phase 1 / Phase 2	Searching Collaborators

*Inflammatory Diseases: Pancreatitis, Inflammatory Bowel Disease, Chronic Kidney Disease, Cardiomyopathy
HTR: 5HT(Serotonin) Receptor; PDK: Pyruvate Dehydrogenase Kinase; L/O: licensing out

Lead asset for **MASH**, completed phase 1 clinical trial and **Phase 2a IND was approved by US FDA (April 2025)**
Second asset is in **early preclinical stage** that targets **Pyruvate dehydrogenase kinase 4**
Third asset is in the **early preclinical stage** is targeting Dravet syndrome, **an epileptic genetic disorder**
Fourth asset is in the **discovery stage** developing new payloads for **ADC, PDC, and SMDC** using our medicinal chemistry expertise.

JD BIOSCIENCE

MASH

GM-60106

Executive Summary

JD BIOSCIENCE

GM-60106

01 **First-in-class**, **Oral Peripheral HTR2A antagonist**

02 **Strong anti-fibrotic efficacy** (*In vivo* animal models)

03 **A combination effect with a GLP-1 agonist or SGLT-2 inhibitor**

04 A FIH study of GM-60106 was considered **well-tolerated and safe**

05 **US FDA IND approval for phase IIa trials**

^{14}C-[GM-60106] tissue distribution studies showed no BBB penetration, consistent with targeting peripheral HTR2A, thereby eliminating undesirable CNS side effects

Seeking partners for **licensing or co-development of GM-60106**, including combination opportunities with other **MASH assets**

Current Therapies – Rezdiffra & Wegovy

JD BIOSCIENCE

Resmetirom (THR-β agonist) is the first approved drug for MASH followed by Wegovy (GLP-1 agonist)

"For the treatment of adults with noncirrhotic MASH with moderate to advanced liver fibrosis"

Rezdiffra (Resmetirom)

- Improvement of fibrosis observed in **only 25% of patients (F2-F3) compared to 14% placebo**
- **Limited fibrosis efficacy (52 weeks)**
- Potential to induce hepatotoxicity and cholecystitis
- **Cost $4,400/month**

Wegovy (Semaglutide)

- Improvement of fibrosis observed in **37% of patients (F2-F3) compared to 22% placebo**
- **Limited fibrosis efficacy (72 weeks)**
- **Subcutaneous injection**
- Expensive1,300 USD/month
- Potential to induce gallbladder disease and pancreatitis
- **Cost $1,300/month**

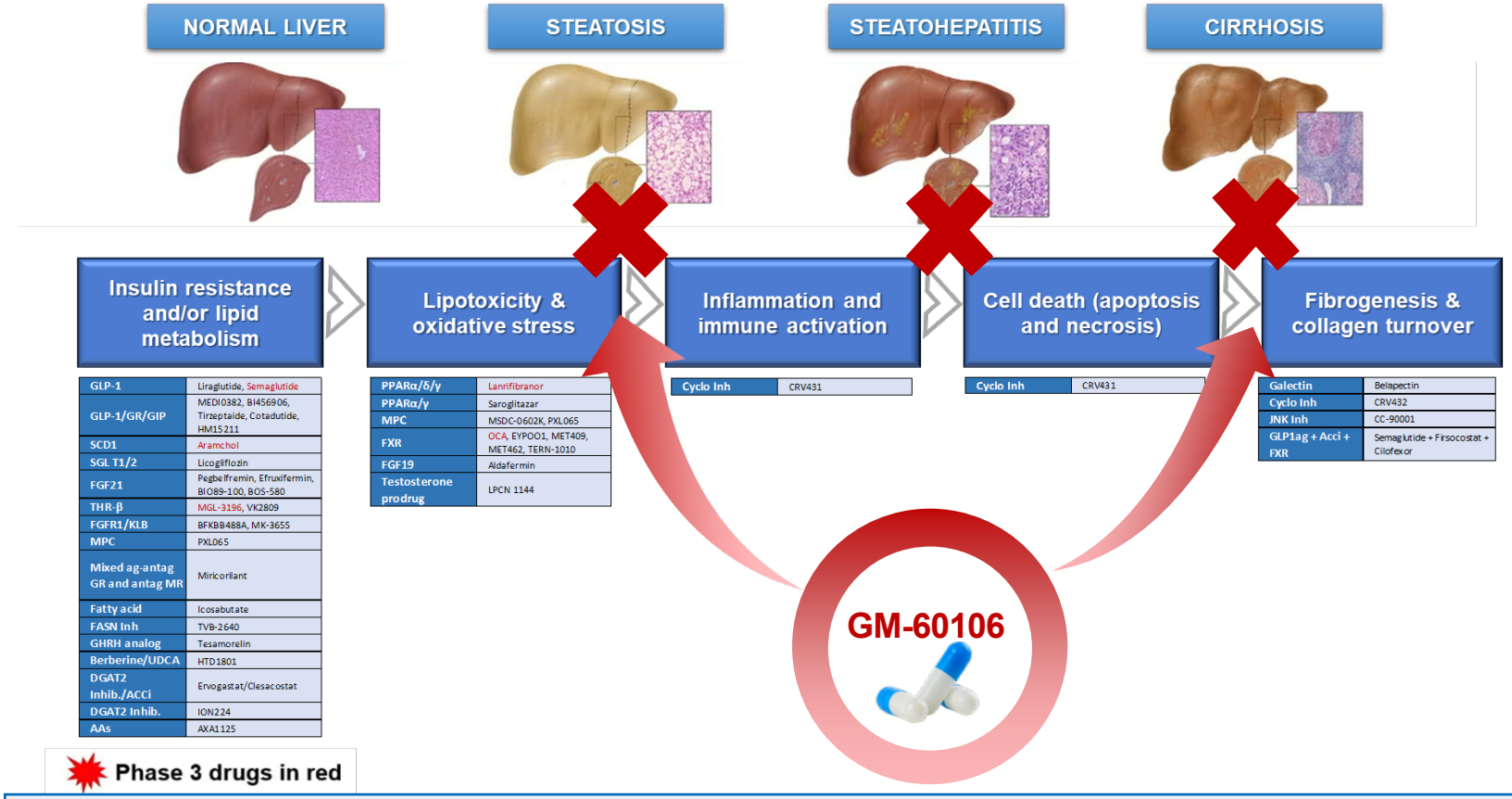
Unmet Medical Needs in MASH Treatment

- **Current Limited Therapies**: Need drugs targeting strong anti-fibrosis efficacy
- **Broad-Spectrum Efficacy**: Need drugs targeting inflammation, fibrosis, and metabolic comorbidities
- **Advanced Disease**: Lack of therapies for cirrhosis and decompensated cirrhosis
- **Combination Therapies**: Synergistic treatments needed for multifactorial MASH

Competitive Edge of GM-60106 – Dual Mechanism

JD BIOSCIENCE

The effects of most drug candidates are focused on modulating lipid metabolism rather than targeting liver fibrosis



GM-60106 has a dual mechanism of action that simultaneously **suppresses hepatic fat accumulation** and **fibrosis**, suggesting therapeutic potential in both monotherapy and combination therapy

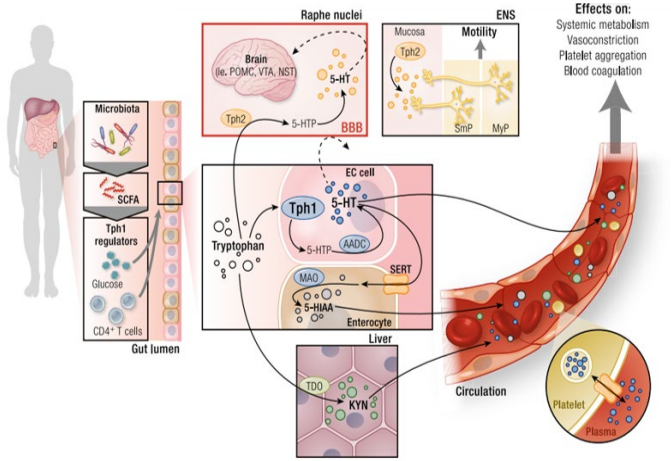
Source: Stephen Harrison AASLD 2022 modified

Serotonin Receptor(HTR2a) as a MASH Target

JD BIOSCIENCE

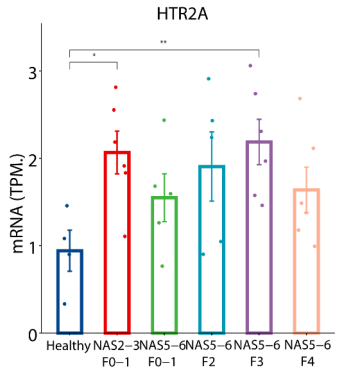
HTR2a expression is upregulated under MASH conditions in human patients, as well as in animal and cellular models

Serotonin Signaling (5-HT)



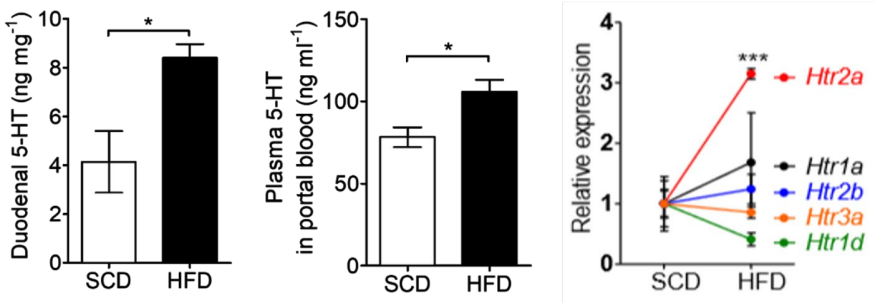
Outside the CNS, Serotonin regulates numerous biological processes, including cardiovascular function, bowel motility, bladder control, as well as lipogenesis in white adipose and lipid accumulation in the liver, etc

Human Patients



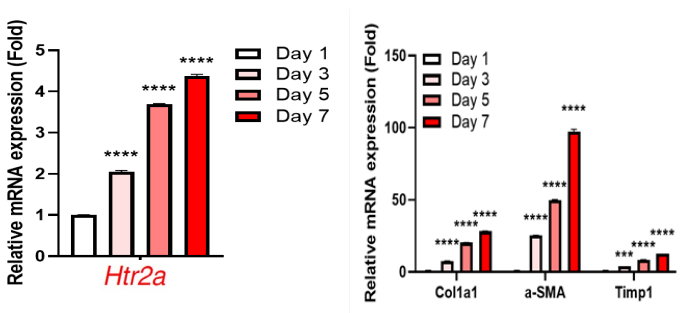
HTR2A levels in MASLD patients

Animal Model



Expressions of peripheral serotonin and its receptors in liver tissue

Cellular Model



Expressions of serotonin receptors during activation of HSCs

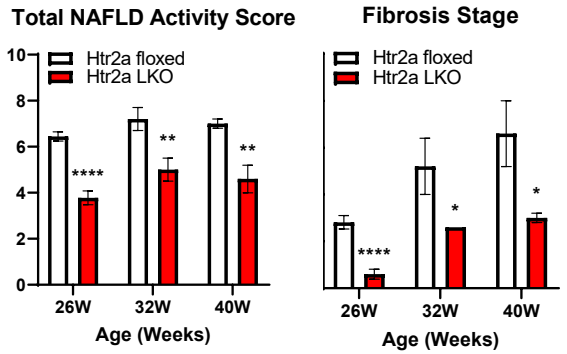
SCD: Standard Chow Diet; HFD: High Fat Diet; Htr: 5HT (serotonin) receptor; Col1: Collagen 1;
SMA: Smooth Muscle Actin; Timp1: Tissue inhibitor of matrix metalloproteinase 1; HSC: Hepatic Stellate Cells

Source: Julian Yabut, Endocrine Reviews, 2019
Hail Kim et al., Nat. Commun.2018

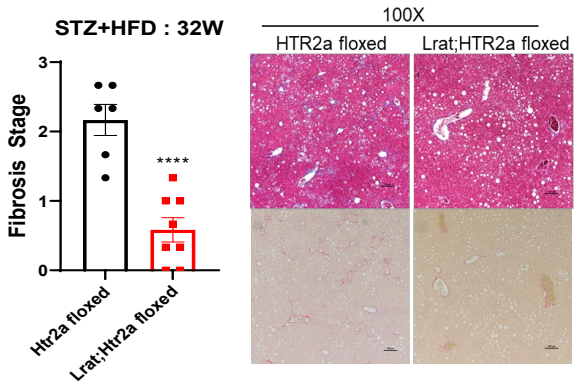
Dual Action of GM-60106 on Fibrosis and Steatosis

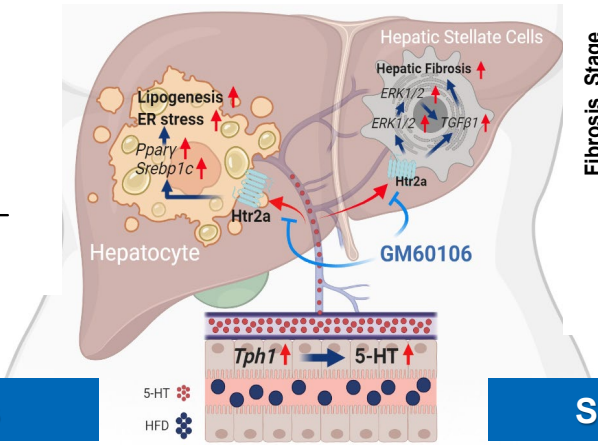
GM-60106 is a novel oral HTR2A antagonist with a dual effect, leading to deactivation of HSCs ameliorating the fibrosis and reduction of lipogenesis in hepatocytes

Steatosis & Fibrosis Improvement in HTR2a LKO Mouse Model

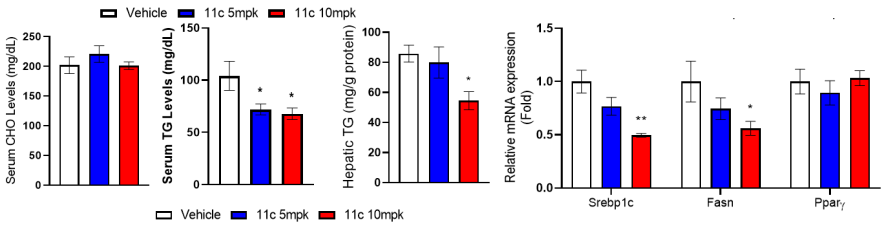


Fibrosis Improvement in HSC Htr2a KO Mouse Model



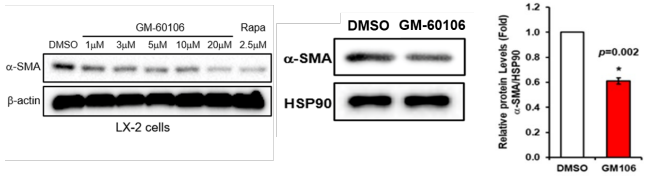


Suppression of Lipogenesis



*11c: GM-60106

Suppression of Liver Fibrosis



HFD: High Fat Diet; 5HT: serotonin; ER: Endoplasmic reticulum; BBB: Blood Brain Barrier; HSC: Hepatic Stellate Cells

Source: Hail Kim et al. Nat Comm, 2018, Jin Hee Ahn and Hail Kim et al., 2024, 15, 645

Peripheral HTR2A selective targeting eliminates CNS side effects

No blood brain barrier penetration of GM-60106 is observed in [^{14}C] – GM-60106 tissue distribution study

Table 5
Concentration of GM-60106 Equivalents in Group 4 Rat Whole Blood and Plasma following an Oral Dose of [^{14}C]-GM-60106 at 5 mg/kg
Results expressed as ng equiv./g

Sample	Timepoint	4001	4002	4003	4004	4005	4006	4007	4008	Mean
Whole Blood	15 m	133	-	-	-	-	-	-	-	133
	30 m	-	158	-	-	-	-	-	-	158
	1 h	-	-	101	-	-	-	-	-	101
	6 h	-	-	-	87.2	-	-	-	-	87.2
	24 h	-	-	-	-	BQL	-	-	-	BQL
	48 h	-	-	-	-	-	BQL	-	-	BQL
	96 h	-	-	-	-	-	-	BQL	-	BQL
	168 h	-	-	-	-	-	-	-	BQL	BQL
Plasma	15 m	216	-	-	-	-	-	-	-	216
	30 m	-	225	-	-	-	-	-	-	225
	1 h	-	-	140	-	-	-	-	-	140
	6 h	-	-	-	104	-	-	-	-	104
	24 h	-	-	-	-	BQL	-	-	-	BQL
	48 h	-	-	-	-	-	BQL	-	-	BQL
	96 h	-	-	-	-	-	-	BQL	-	BQL
	168 h	-	-	-	-	-	-	-	BQL	BQL

BQL =Below quantitation limit (2x background).

Table 7
Concentration of GM-60106 Equivalents (ng equiv./g) in Sprague Dawley Tissues following an Oral Dose of [^{14}C]-GM-60106 at 5 mg/kg

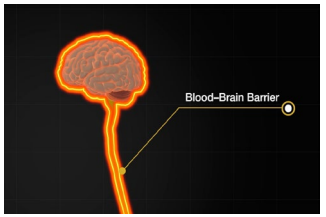
	Hours Post Dosing							
Tissue	0.25	0.5	1	6	24	48	96	168
Adrenal gland	574	1348	1007	1515	BQL	BQL	BQL	BQL
Bone Marrow (Femur)	264	387	407	1312	BQL	BQL	BQL	BQL
Bone(Femur)	209	304	BQL	351	BQL	BQL	BQL	BQL
Brain	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Eye (lens)	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Fat (yellow)	182	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Harderian gland	BQL	194	192	2014	686	605	BQL	BQL
Heart	153	351	257	369	BQL	BQL	BQL	BQL
Kidney	566	1320	952	1842	BQL	BQL	BQL	BQL
Kidney(Cortex)	602	1246	942	1851	BQL	BQL	BQL	BQL
Kidney(Medulla)	505	1509	974	1918	BQL	BQL	BQL	BQL
Large Intestine (Wall)	155	252	355	9797	1455	BQL	BQL	BQL
Liver	2271	2245	2435	2918	BQL	BQL	BQL	BQL
Lung	353	846	556	941	BQL	BQL	BQL	BQL
Muscle(Femoral)	BQL	304	212	337	BQL	BQL	BQL	BQL
Pancreas	812	1115	1431	4327	BQL	BQL	BQL	BQL
Pituitary gland	BQL	218	571	1925	533	BQL	BQL	225
Prostate	BQL	BQL	BQL	716	BQL	BQL	BQL	BQL
Skin (non-pigmented)	BQL	BQL	150	257	BQL	BQL	BQL	BQL
Small Intestine (Wall)	45036	1840	534	1257	BQL	BQL	BQL	BQL
Spleen	412	1091	1191	2294	BQL	BQL	BQL	BQL
Stomach (wall)	3239	433	797	787	BQL	BQL	BQL	BQL
Testes	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Thymus	BQL	BQL	182	768	BQL	BQL	BQL	BQL
Thyroid gland	277	833	639	796	BQL	BQL	BQL	BQL
Uveal Tract	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL

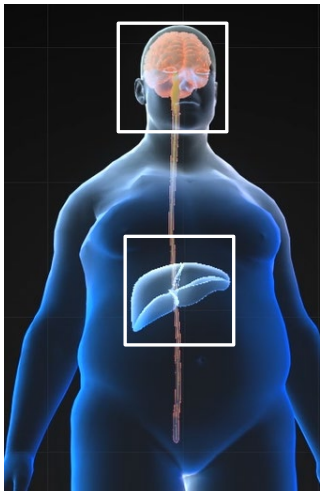
BQL = Below the lower limit of quantitation 148 ng equiv./g.

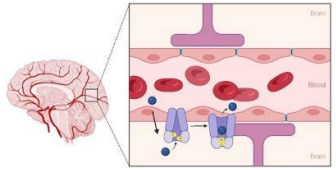
Table 9
Pharmacokinetic Parameters of GM-60106 Equivalents in Plasma and Tissues of Male Sprague Dawley Rats following an Oral Dose of [^{14}C]-GM-60106 at 5 mg/kg

Tissue	Cmax (ng equiv./g)	Tmax (h)	T½ (h)	AUC0-t (ng equiv·h/g)	AUCinf (ng equiv·h/g)	Tissue : Plasma**
Plasma	225	0.5	NC	783	NC	1.00
Whole Blood	158	0.5	NC	589	NC	0.75
Adrenal gland	1510	6	NC	7210	NC	9.21
Bone Marrow (Femur)	1310	6	NC	4610	NC	5.89
Bone(Femur)	351	6	NC	NR	NC	NA
Brain	BQL	NC	NC	NR	NC	NA
Eye (lens)	BQL	NC	NC	NR	NC	NA
Fat (yellow)	182	0.25	NC	NR	NC	NA
Harderian gland	2010	6	NC	45400	NC	58.0
Heart	369	6	NC	1800	NC	2.30
Kidney	1840	6	NC	7860	NC	10.0
Kidney(Cortex)	1850	6	NC	7840	NC	10.0
Kidney(Medulla)	1920	6	NC	8170	NC	10.4
Large Intestine (Wall)	9800	6	NC	127000	NC	162
Liver	2920	6	NC	15400	NC	19.7
Lung	941	6	NC	4290	NC	5.48
Muscle(Femoral)	337	6	NC	1540	NC	1.97
Pancreas	4330	6	NC	15400	NC	19.7
Pituitary gland	1920	6	NC	43100	NC	55.0
Prostate	716	6	NC	NR	NC	NA
Skin (non-pigmented)	257	6	NC	NR	NC	NA
Small Intestine (Wall)	45000	0.25	NC	16600	NC	21.2
Spleen	2290	6	NC	9520	NC	12.2
Stomach (wall)	3240	0.25	NC	5130	NC	6.55
Testes	BQL	NC	NC	NR	NC	NA
Thymus	768	6	NC	NR	NC	NA
Thyroid gland	833	0.5	NC	4130	NC	5.27
Uveal Tract	BQL	NC	NC	NR	NC	NA

NC = Not calculated due to insufficient terminal elimination phase. NA = Not applicable.
NR = Not reportable due to < 3 consecutive quantifiable concentrations
**=Tissue:plasma ratio based on AUC0-t
BQL = Below the lower limit of quantitation 148 ng equiv./g





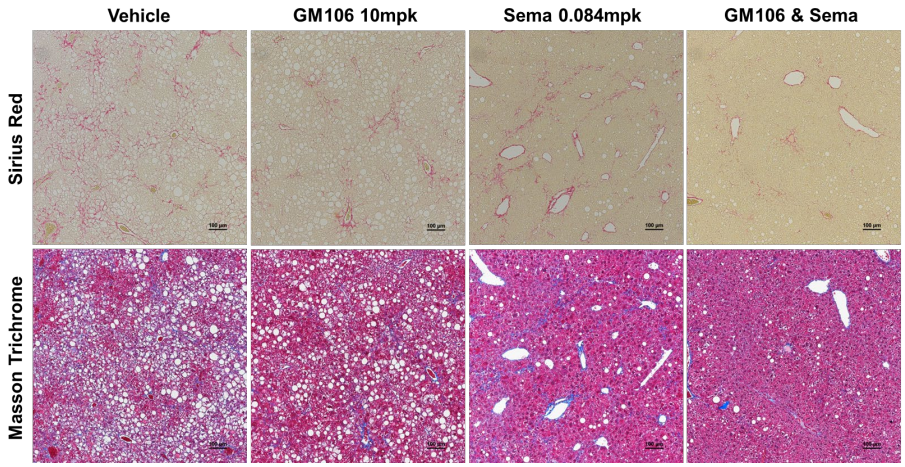


Determine [the tissue distribution and tissue pharmacokinetics] of [^{14}C]- GM-60106-derived radioactivity in the rat following a single PO administration. -> 0.25, 0.5, 1, 6, 24, 48, 96, and 168 hours post dosing (SD model, Group4)

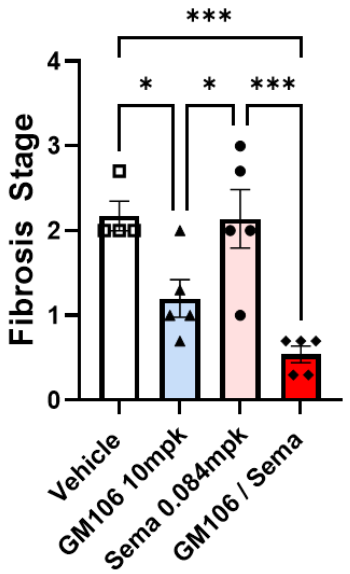
Comparison and Combination with Semaglutide (Wegovy)

GM-60106 shows an additive effect on fibrosis stage when combined with Semaglutide

Fibrosis Histology



Fibrosis Stage



GM-60106 has a strong potential for a **combination effect** with other medications: co-administration with an anti-diabetic drug exhibits significant reduction in fibrosis

Semaglutide (Sema): an antidiabetic agent and Glucagon-like peptide-1 (GLP-1) agonist.
Administered in the STAM mouse model along with a high-fat diet (HFD) for 24 weeks

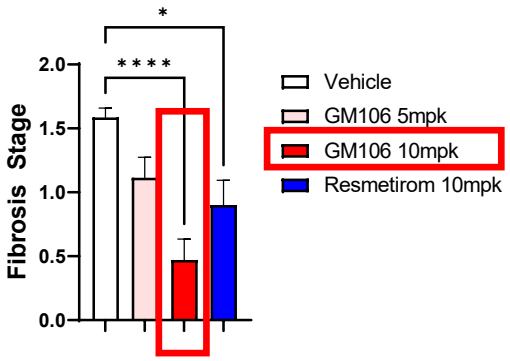
GM-60106 Comparison with Resmetirom (Rezdiffra)

Fibrosis and NAS score was measured after 10 weeks of once-daily dosing of GM-60106 or Resmetirom (Rezdiffra) in STZ+HFD mice

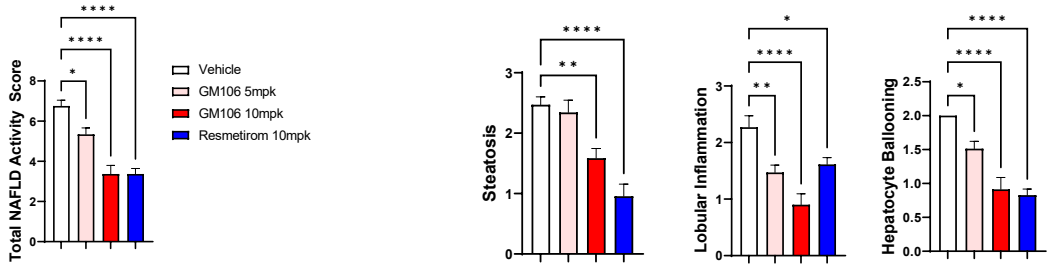
STZ+HFD mouse (MASH model)

Group		n	Model	Route
1	B6J_Vehicle	7	mSTAM	Oral
2	B6J_GM106 5mpk	7	mSTAM	Oral
3	B6J_GM106 10mpk	7	mSTAM	Oral
4	B6J_Resmetirom 10 mpk	7	mSTAM	Oral

Fibrosis Stage



NAS Score



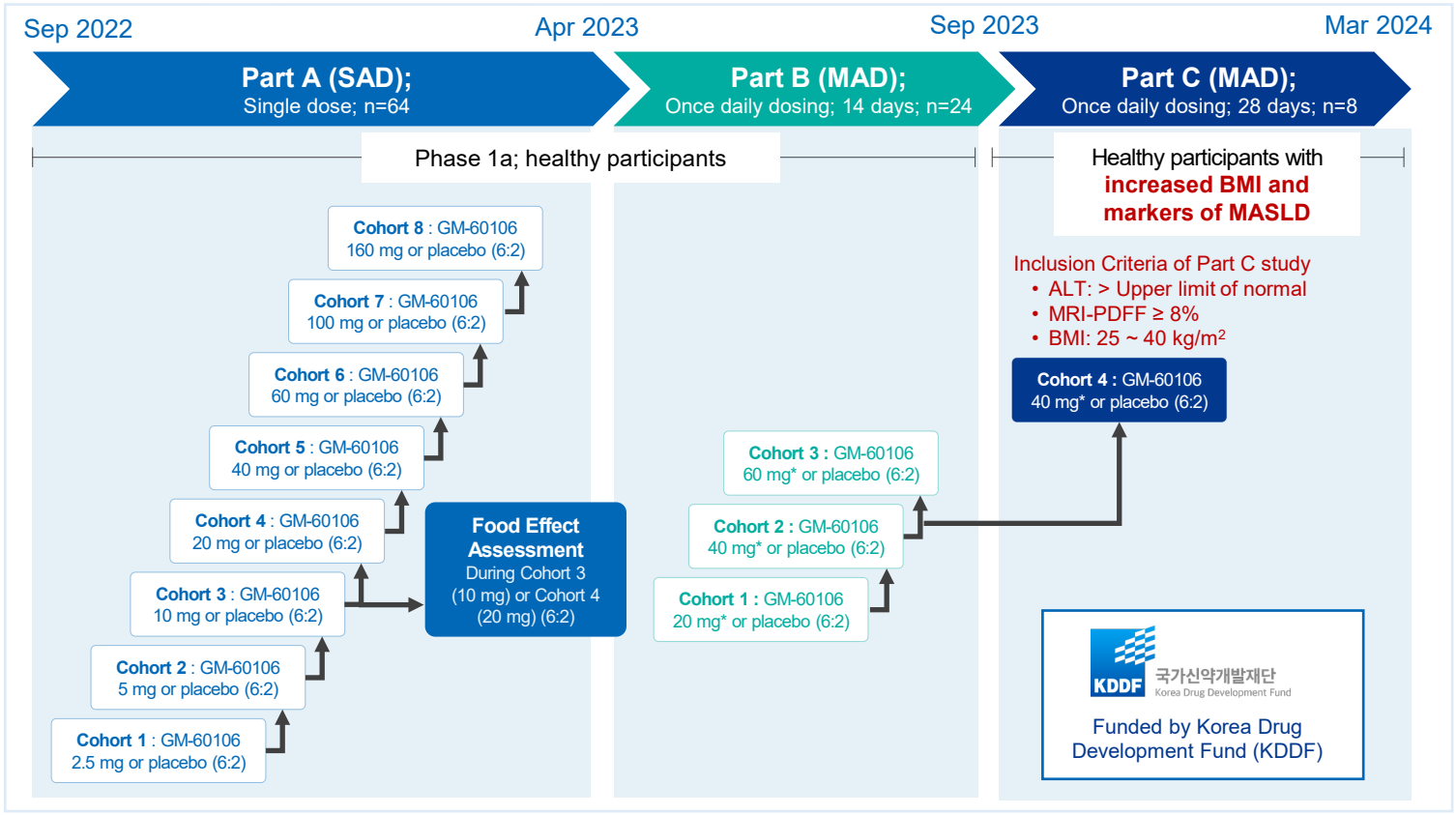
The first approved MASH treatment, Resmetirom (brand name: Rezdiffra), was compared with GM-60106.
GM-60106 showed **a superior anti-fibrotic effect**

Phase 1 Clinical Trial for Safety and Tolerability

JD BIOSCIENCE

In healthy adult participants with high BMI and MASLD markers, 40% of subjects dosed with GM-60106 for 28 days shows a strong reduction of liver fat content (>35%)

NCT05517564



UC San Diego

Rohit Loomba M.D, MHSc

Professor at UCSD (JDB SAB member)

World-leading researcher in the field of MASH

- Ranked in the top 1% globally in MASLD/MASH research
- Leading the development of non-invasive diagnostic technologies
- Member of AASLD, AGA, ASCI, and AAP
- Editorial board member of *Hepatology*

Ready for GM-60106 Phase 2a

JD BIOSCIENCE

GM-60106 is ready for Phase 2a in the US

Safe to Proceed Letter

FDA U.S. FOOD & DRUG ADMINISTRATION

IND 174081

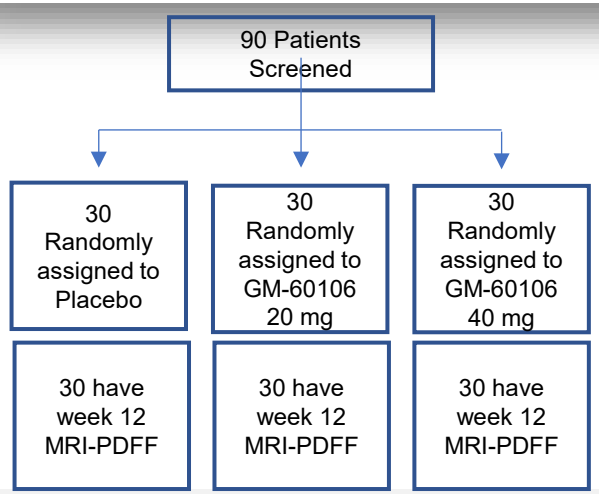
STUDY MAY PROCEED

JD Bioscience, Inc.
Attention: Geetanjali Avhad
Regulatory Affairs Specialist
2400 Ellis Road
Durham, NC 27703

Dear Geetanjali Avhad:

Please refer to your investigational new drug application (IND) submitted under section 505(i) of the Federal Food, Drug, and Cosmetic Act (FDCA) for GM-60106.

We have completed our safety review of your application and have concluded that you may proceed with your proposed clinical investigation.



Phase 2a Plan

A Phase 2a, randomized, double blind, placebo controlled PoC study to evaluate the safety, efficacy, biological activity, and pharmacokinetics of GM-60106 in participants with metabolic associated steatohepatitis (MASH)

Primary Endpoints

Changes from baseline in hepatic fat fraction assessed by MRI-PDFF in Week 12

Secondary Endpoints

PD: % change from baseline to week 6 and week 12 in Liver function tests, Lipid profile tests,

and Glucose, Insulin, HOMA-IR, Adipo-IR, HbA1c, serum adiponectin, hs-CRP and APRI

Safety: Vital signs, 12-lead ECGs, physical examination, SAE, AEs, hematology, serum biochemistry, and urinalysis

PK: Cmax, Tmax, AUCs, CL/F, metabolites of GM-60106, etc

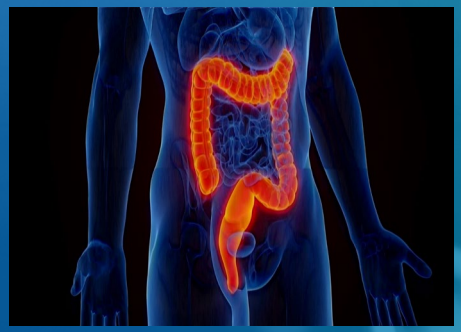
Exploratory

Changes from baseline at week 12 in liver fibrosis (FibroScan, serum Pro-C3, and ELF), Serum FGF21, CK18, weight, BMI, waist circumference, waist-to-hip ratio

JD BIOSCIENCE

Inflammatory Disease

GM-10395
GM-10134
GM-10202 (prodrug of GM-10134)
JD-160106



Executive Summary

Target : PDK4

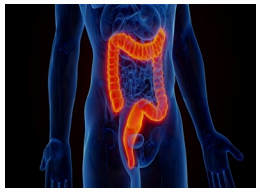
- PDK4 is **a druggable target** in **Inflammatory diseases**
- GM-10395 and GM-10134 (202) are orally active and potent PDK4 inhibitors with first-in-class potential and a novel MOA
- Preclinical studies have demonstrated **significant improvements in various inflammatory disease models**

Indication 1 : Heart Disease

- **Preclinical studies** have **demonstrated significant improvements in cardiac function** in animal models of cardiomyopathy

Indication 2 : Inflammatory Bowel Disease (IBD)

- GM-10395 strongly **suppress the activation of CD4+ T cells,** a key driver in the progression of IBD
- Preclinical studies of **GM-10395 demonstrate significant anti-inflammatory effects** in DSS-induced IBD model
- **Reduces inflammation** and **restores immune homeostasis**



Indication 3 : Acute Kidney Injury (AKI)

- GM-10395 exhibits both the **preventive** and **therapeutic effect, reducing inflammation** and **restoring immune homeostasis**
- **In addition, it Reduces Succinate** level that impairs **renal activity as well as ROS** burst that induces **Apoptosis**

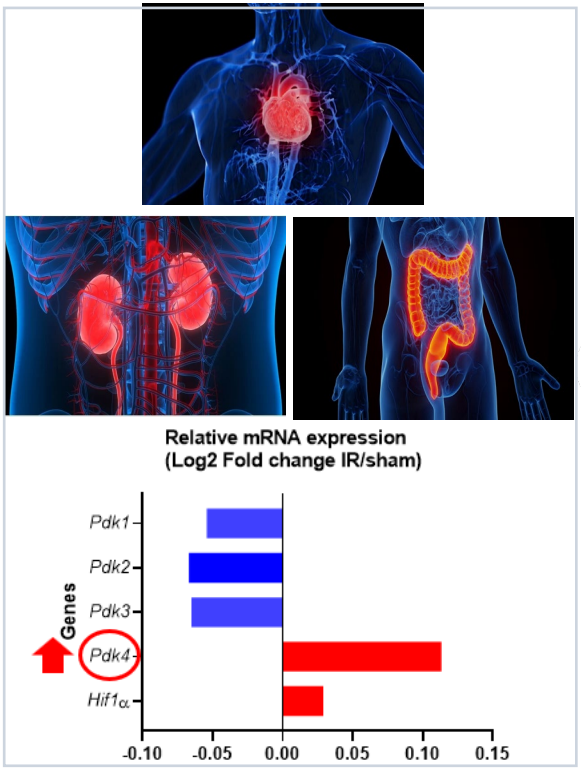
(Data not shown, unpublished)

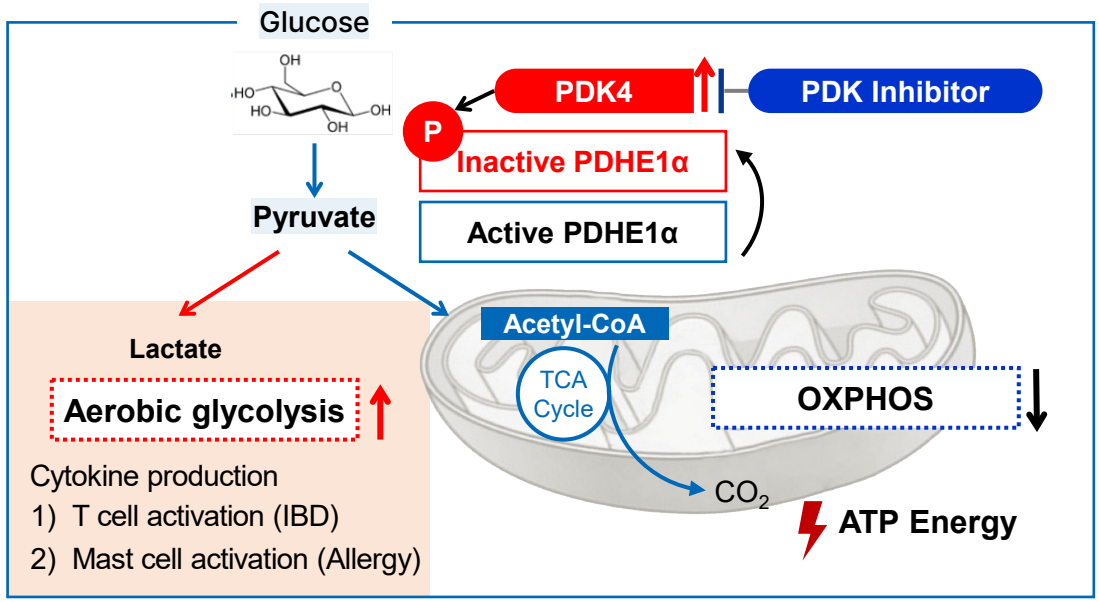
PDK4 is a Novel Inflammatory Disease Target

JD BIOSCIENCE

Identified a novel target for inflammatory diseases, including Ischemia/reperfusion injury (heart and kidney), Cardiomyopathy, IBD, and CKD

- PDHE1α is phosphorylated by PDK4, promoting a metabolic shift from oxidative phosphorylation to aerobic glycolysis





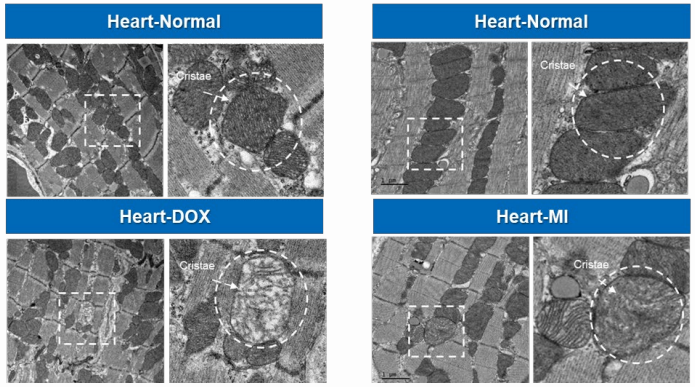
Years of Fundamental research have demonstrated the significance of mitochondria function in inflammatory diseases
Our team confirmed the **crucial role that PDK4 plays in maintaining mitochondrial function**

Source: In-Kyu Lee et al. Nat. Commun. (2023) 14:1703

PDK4 as a Potential Therapeutic Target to combat Heart disease

Target Validation

- PDK4 induces mitochondrial instability and dysfunction
- Both doxorubicin- and ischemia/reperfusion-induced cardiotoxicity show a defect in mitochondrial cristae organization
- Results of Doxorubicin-induced cardiomyopathy model in PDK4 knockout mice:
  - PDK4 knockout shows improved echocardiography after doxorubicin treatment
  - PDK4 knockout improves membrane stability and reduced apoptosis markers
  - Cardio vacuolization dysfunction is recovered, and cardiac fibrosis is restored



- **PDK4 plays an important role in cardiac dysfunctions**

In vivo Pharmacology

PDK4 inhibitors, GM-10395 and GM-10202, improve cardiac function in three animal models

- **Doxorubicin-induced cardiomyopathy model**
  - GM-10395, GM-10202
- **Doxorubicin and Herceptin-induced cardiomyopathy model**
  - GM-10202
- **Myocardial infarction (MI)-induced cardiomyopathy**:
  - GM-10395, GM-10202

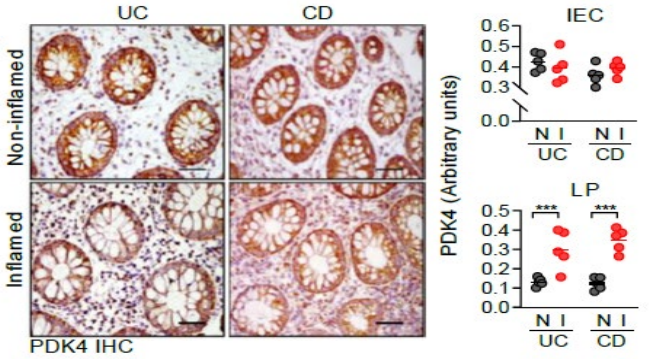
(Data not shown, unpublished)

Target Engagement of GM-10395

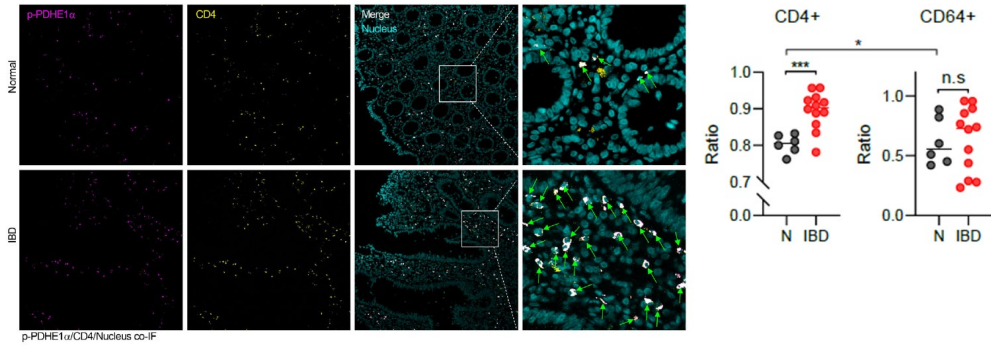
JD BIOSCIENCE

PDK4 is overexpressed in patients with IBD and p-PDHE1α is enriched in CD4⁺ T cells from patients with IBD

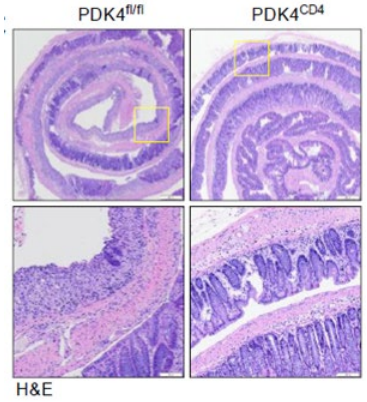
PDK4 Expression



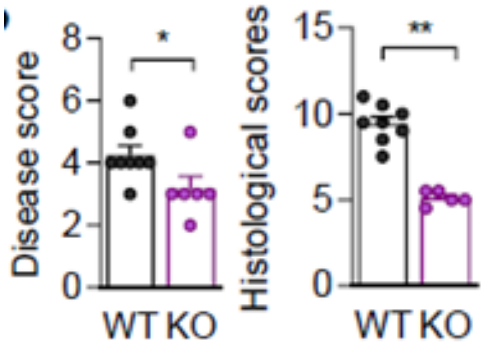
p-PDHE1α are enriched in CD4⁺ T cells



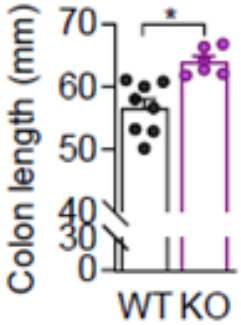
CD4+ T-Cell Specific PDK4 Deficient Mouse Model



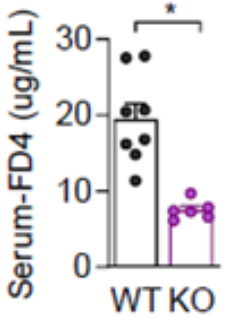
Disease and Histological Scores



Colon Length



Permeability



PDK:Pyruvate Dehydrogenase Kinase; PDHE1:Pyruvate dehydrogenase E1;

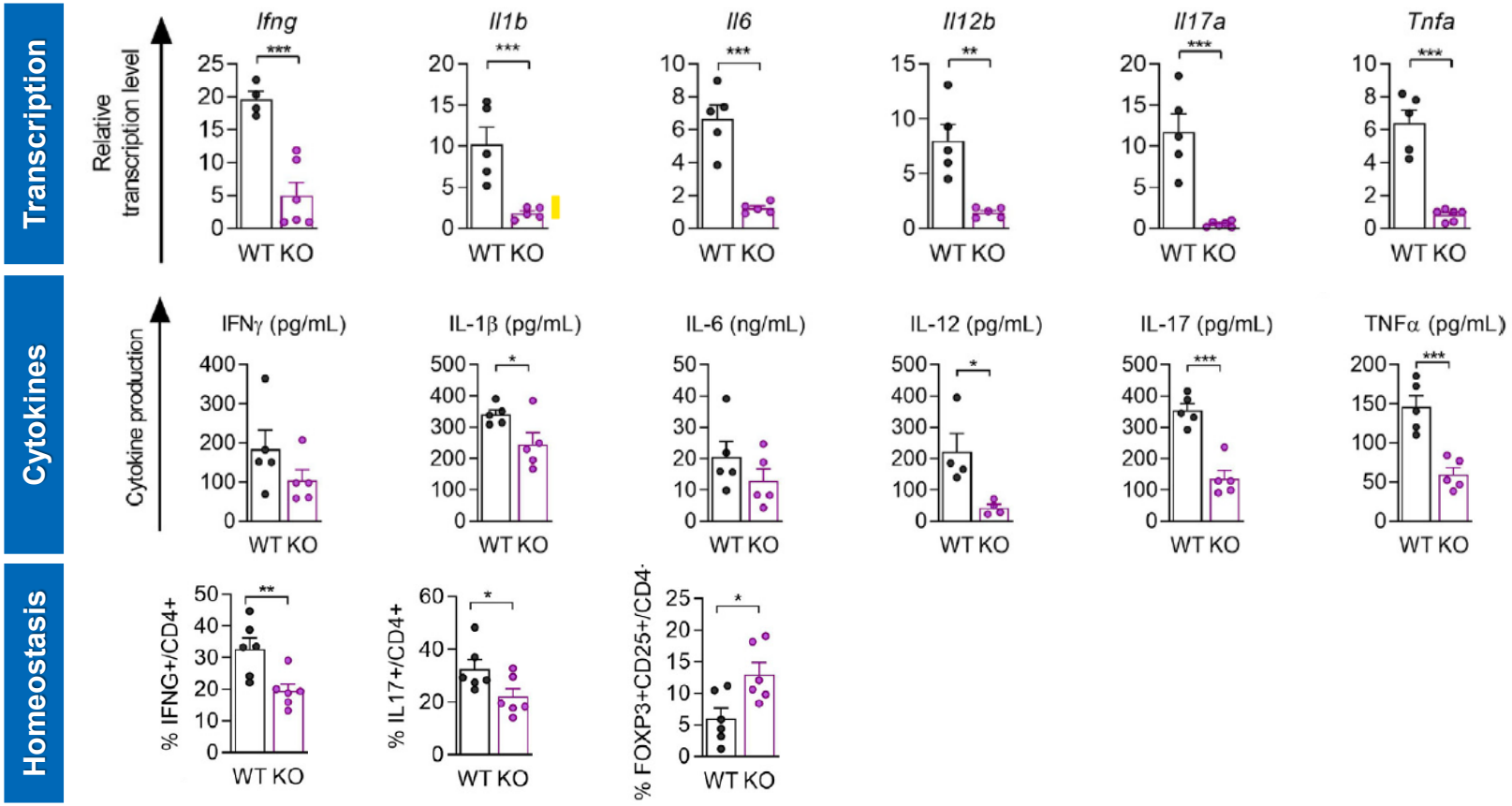
Source: In-Kyu Lee et al. Cell Mol Gastroenterol Hepatol, 2023 Vol. 15(2):439-461

CD4+ T-Cell PDK4 Deficiency Shows Reduction in Inflammation

JD BIOSCIENCE

Conditional PDK4 knockout in CD4⁺ T cells shows restores immune homeostasis

- Among gut-infiltrating CD4⁺ T cells, the proportion of TH17-positive cells is lower, while the proportion of regulatory T cells is higher



PDK:Pyruvate Dehydrogenase Kinase; WT: Wild Type; KO:Knock out; Ifn:Interferon; Il: Interleukin; Tnf: Tumor Necrosis Factor

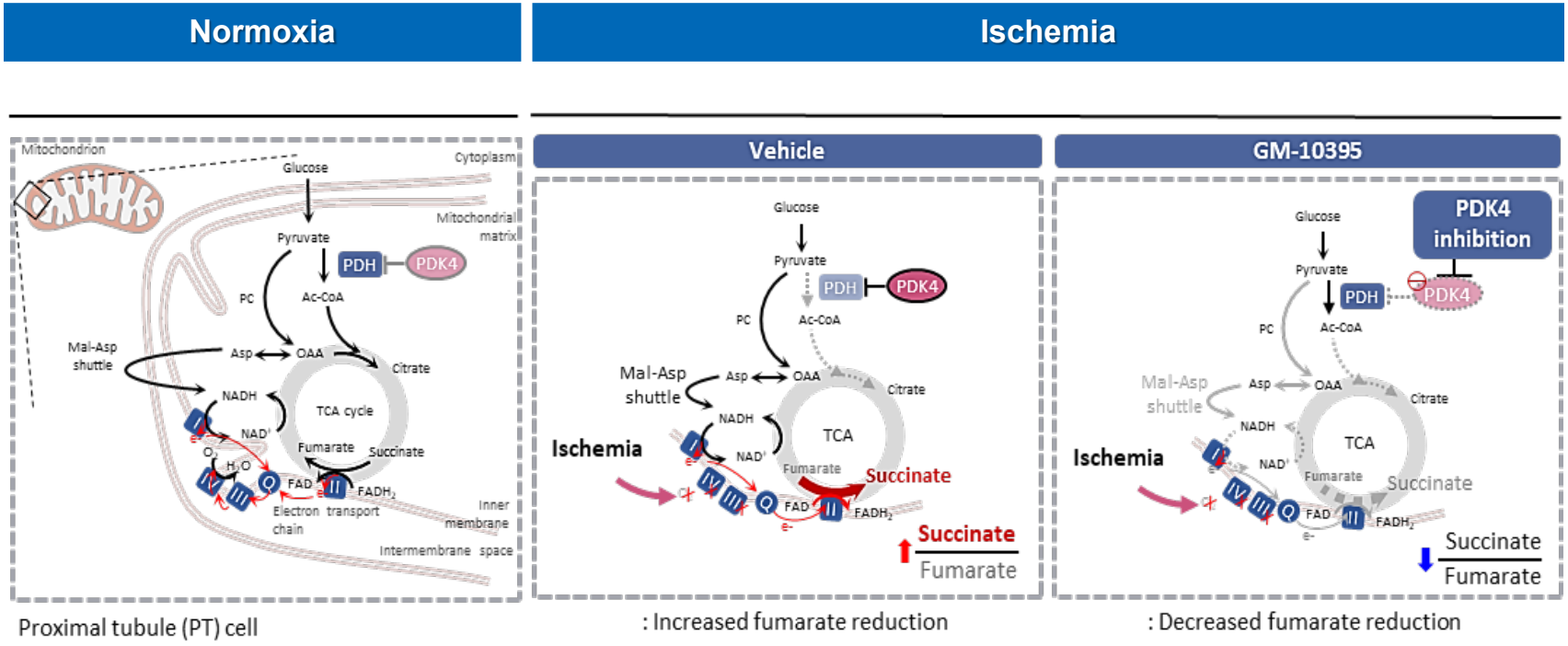
Source: In-Kyu Lee et al. Cell Mol Gastroenterol Hepatol, 2023 Vol. 15(2):439-461

Main MOA of GM-10395 in Kidney Disease

JD BIOSCIENCE

Ischemia induces mitochondrial dysfunction in kidney proximal tubules

- Ischemia causes increased succinate levels
- PDK4 inhibition reduces succinate levels
- PDK4 inhibition decreases fumarate reduction



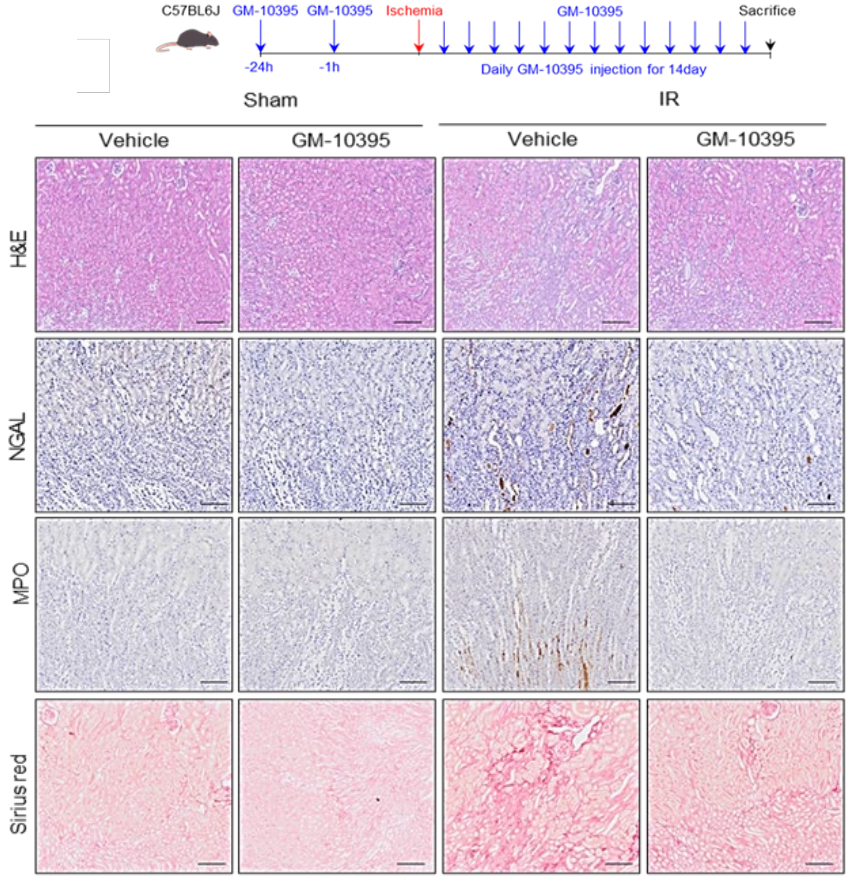
Source: In-Kyu Lee et al. Kidney Int. (2023)104:724-739

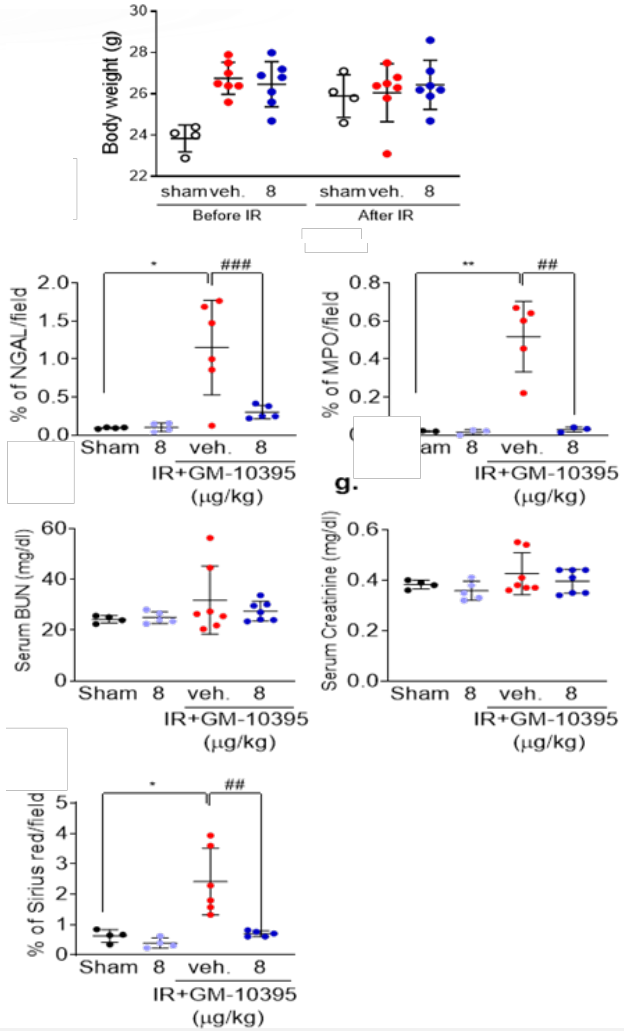
GM-10395 Reduces Renal Fibrosis

JD BIOSCIENCE

GM-10395 treatment restores the kidney function after Ischemia

- Kidney function markers are improved
- Fibrosis is reduced upon treatment





Source: In-Kyu Lee et al. Kidney Int. (2023)104:724-739

JD BIOSCIENCE

Dravet

GM-91466

Executive Summary

JD BIOSCIENCE

01 GM-91466 exhibits **therapeutic potential for Dravet Syndrome**, as supported by our preliminary data

02 GM-91466 demonstrates **comparable efficacy to the approved drug,** with **no observed toxicity and a favorable safety profile**

03 The mechanism of action involves **upstream modulation of TPH2 in the serotonin pathway**

04 The results suggest GM-91466 is a **strong candidate** for **Orphan Disease Designation(ODD) and expedited development**

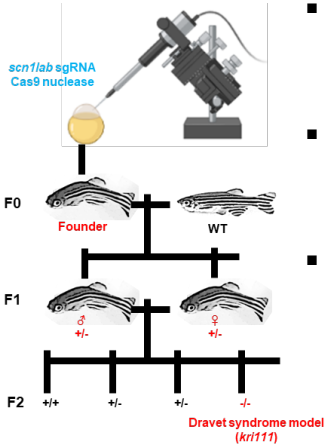
Seeking partners for **licensing or co-development of GM-91466**, including combination opportunities with other **Rare disease(Dravet Syndrome) assets**

kri111 Zebrafish Model for Dravet Syndrome

JD BIOSCIENCE

The SCN1A-mutant zebrafish model for Dravet syndrome, developed by KRICT, has proven its utility in assessing drug efficacy (Patent pending)

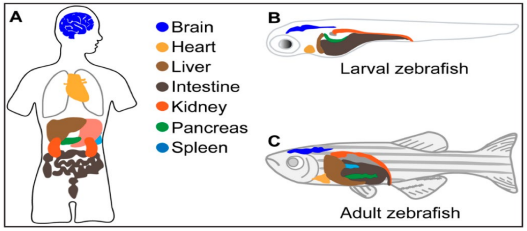
Generation of Mutant Zebrafish Model



- Generated via targeted nonsense mutation of scn1lab, the zebrafish ortholog of human SCN1A
- Mutants display: Spontaneous seizures, Hyperkinetic behavior, Photosensitivity
- Phenotypes emerge at 5–7 days post-fertilization, closely resembling human Dravet Syndrome in both electrophysio-logical and behavioral domains

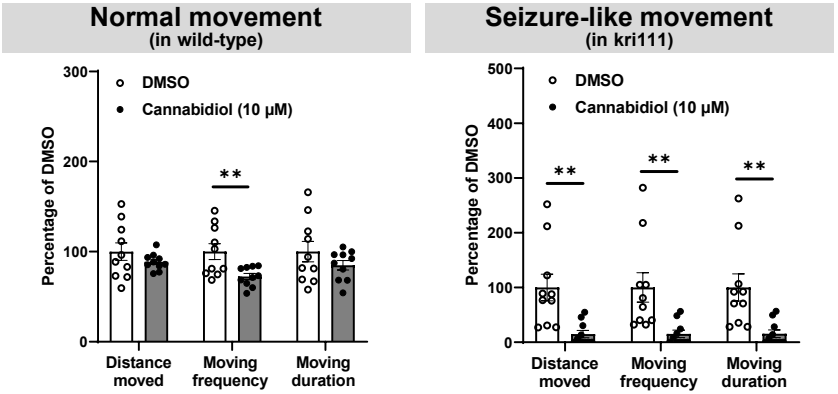
Advantages of Zebrafish Model

- Zebrafish brains are highly homologous to those of humans
- CNS develops in a week
- High-throughput screening: a rising trend in drug development

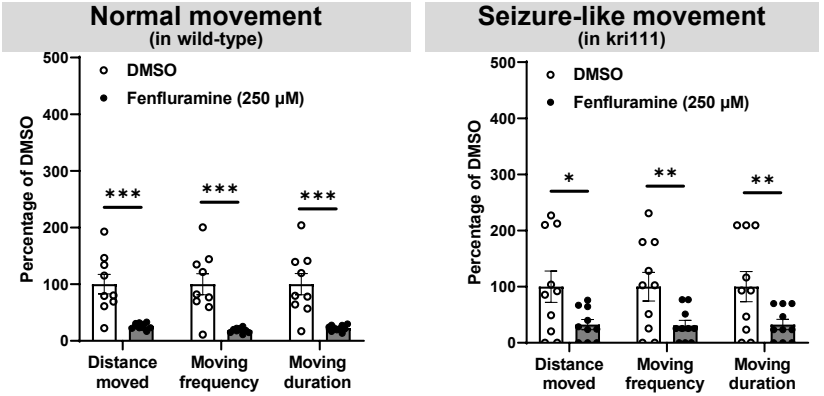


Dravet Drug Evaluation in SCN1A-Mutant Zebrafish

- Cannabidiol suppress hyperlocomotion and seizure-like events



- Fenfluramine has shown efficacy in Kri111 zebrafish models

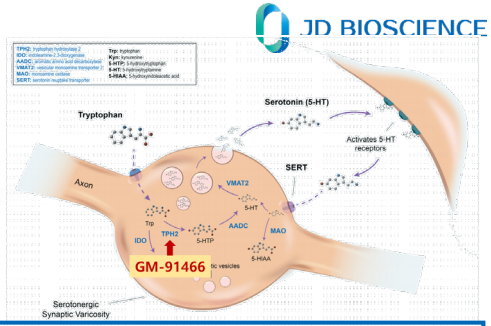


KRICT: Korea Research Institute of Chemical Technology established kri111 zebrafish model

Source: Korea Patent Application 10-2024-0118490

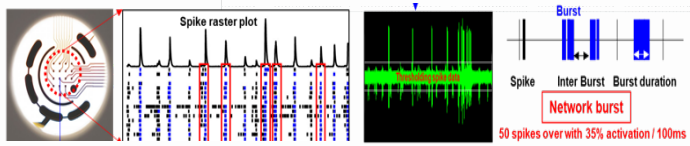
GM-91466 MOA and Efficacy

We hypothesize GM-91466 induces TPH2 that would increase the serotonin levels in the brain



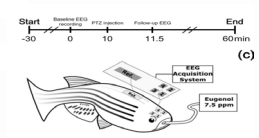
Neural Organoid Model

- In a Mouse Brain Organoid Model using a Micro-Electric Array, GM-91466 improves Spike/Burst Activity in a Dose-Dependent Manner
- GM-91466 **Shows Dose-Dependent Seizure Reduction** in Brain Organoid



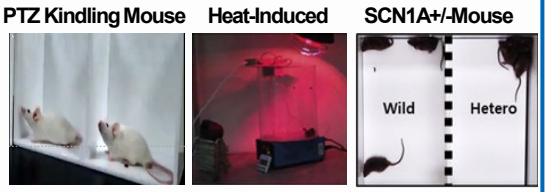
Zebrafish Model

- GM-91466 Shows **Dose-Dependent Reduction of Seizure-like Movements** in kri111 Mutant Zebrafish
- In EEG analysis, GM-91466 **Reduces Seizure Number and Duration** in kri111 Mutant Zebrafish
- In the Light Stimulus Test and Color Preference Test, GM-91466 **Improves Hyperactivity and Cognition** in kri111 Mutant Zebrafish
- GM-91466 Most Effectively **Reduces Seizure-like Movements** Without **Affecting Normal Activity** compared to the Approved Drug



Mouse Model

- GM-91466 Shows **a Neuroprotective Effect** in the PTZ Kindling Mouse Model
- GM-91466 Shows **a Neuroprotective Effect** in Heat-Induced Seizure Model
- GM-91466 **Reduces Seizure Susceptibility and Hyperactivity** in SCN1A+/- Mouse

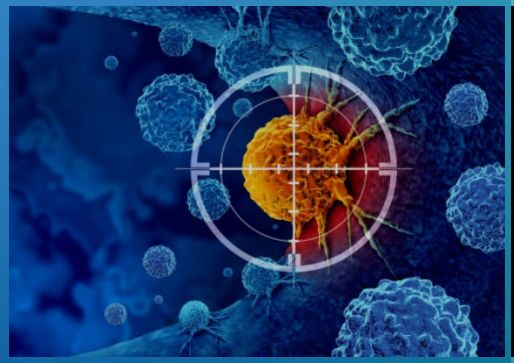


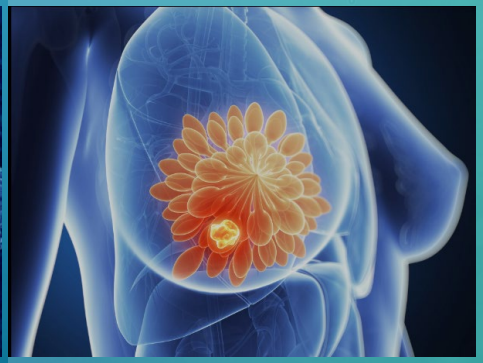
(Data not shown, unpublished)

JD BIOSCIENCE

Cancer

JD-ADC





Executive Summary

JD BIOSCIENCE

JD-ADC payloads

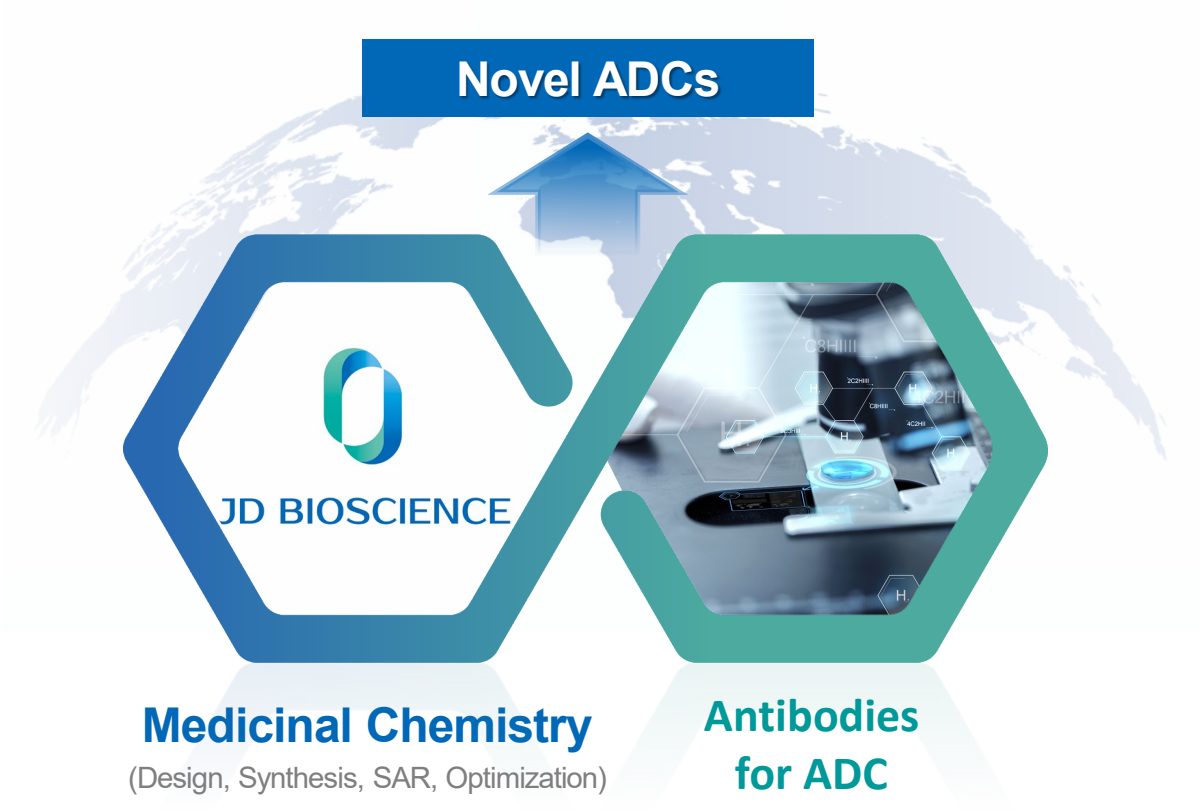
- Over 50 types of new patentable **nano-Molar** to **pico-Molar** range payloads
- Overcomes **CPT resistance or multidrug resistance cell lines**
- ADC with JD payloads shows **3.3x better** efficacy than **Enhertu**
- Our **payloads** have validated by our **partners** showing **pico-Molar toxicity.**
- Expanded payloads to **immune modulators** and **degraders.**
- **Mono- or dual-payload ADC candidates** enabled by **a versatile linker/ payload platform,** adaptable to multiple antibodies and cancer types

Seeking partners for **co-development of JD-ADC Payloads**, including combination opportunities with other **JD-ADC Payloads assets**

ADC for collaboration (Payload)

JD BIOSCIENCE

We are seeking a partner to collaborate on the development of an ADC that could be a game changer in the oncology market



JD BIOSCIENCE

Strategical Development

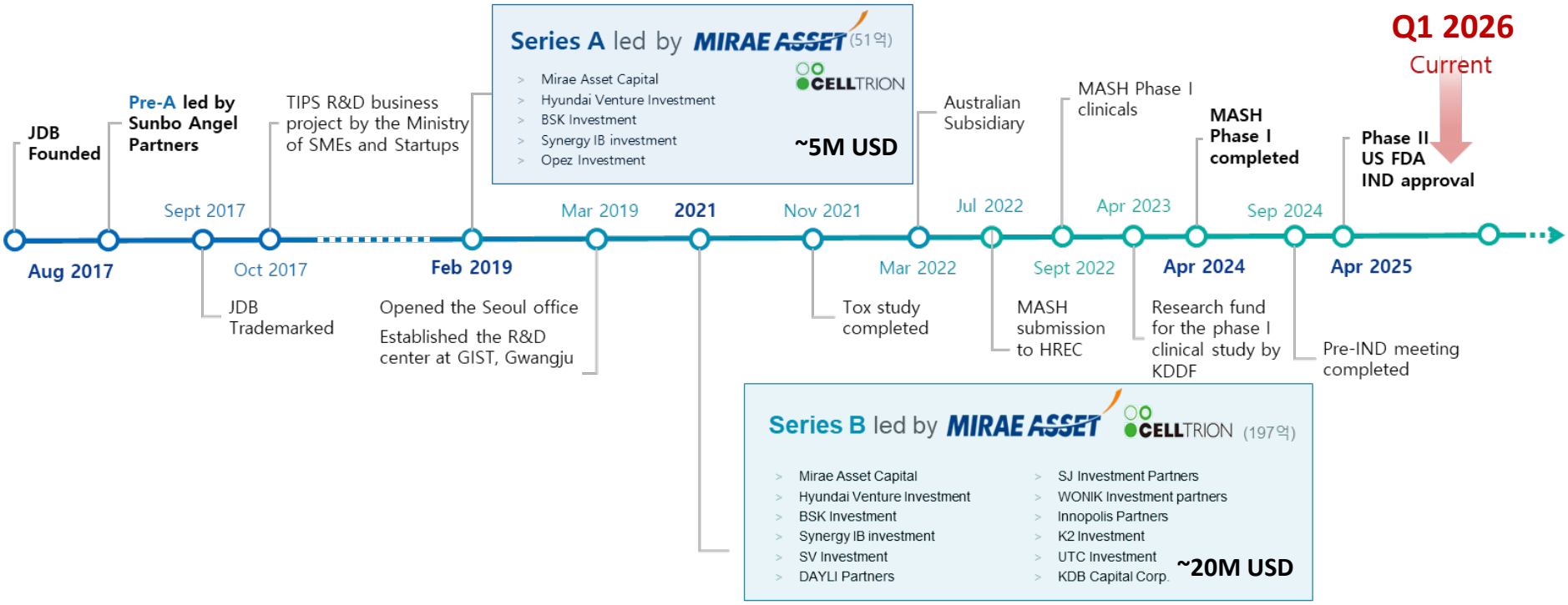
Company History

JD BIOSCIENCE

JDB is experiencing rapid growth with the assistance of our investors

Date of establishment 31 July 2017

Locations Gwangju, South Korea (headquarters, research institute)
Seoul, South Korea (business development, regulatory affair units)



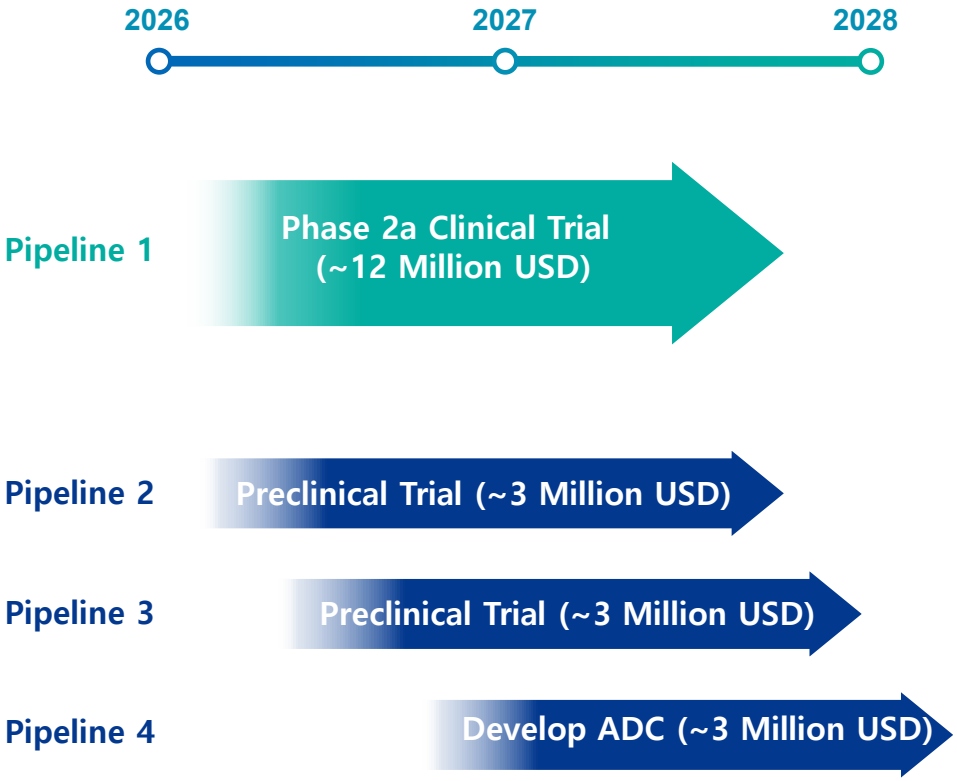
Investment Fund Usage Plan

JD BIOSCIENCE

Attracting Series C investment

Clinical Trial Cost	MASH Phase 2 clinical trial
Preclinical entry	Cost of preclinical trials for Inflammatory and rare diseases
Efficacy evaluation & indication expansion	Efficacy evaluation trials Includes indication expansion and back up compounds
Secure additional pipelines	Technology adoption cost
Fixed cost	Labor costs, rent payments fees, etc.

Fund usage plan (~20 Million USD)



Summary of JD Bioscience Inc.

JD BIOSCIENCE

Bench to Bedside

01 First-in-class Drug Candidates

Developing first-in-class therapeutics targeting metabolic diseases including MASH, fibrosis, inflammation, obesity, and cancer.

02 Medical Team

A team of physician-scientists specializing in innovative therapeutic modalities addressing critical unmet clinical needs.

03 Medicinal Chemistry

Deep expertise in medicinal chemistry, with experience spanning leading pharmaceutical companies to top academic institutions.

04 Integrated Screening Platform

Comprehensive screening platforms supporting drug discovery, efficacy evaluation, and toxicity assessment.

05 Global Research Collaborations

Over 10 external research partners, with expanding collaborative networks across multiple therapeutic areas.

Thank You

For more information, please contact:

Peter Goughnour, Ph.D.

Phone:+82-2-406-9380
Email: petergoughnour@jdbiosci.com

Jin Hee Ahn, Ph.D.

Phone: +82-62-974-9380
Email: contact@jdbiosci.com

JD Bioscience is seeking:

- Global or regional partners interested in **licensing JD Bioscience Assets**
- Potential collaborators for **joint development** of **JD Bioscience Asset**