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JD Bioscience Inc.

IR Non-Confidential Deck Q3 2026

JD Bioscience Inc.- Executive Summary

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- **First-in-class oral therapy targeting liver fibrosis-the final unmet need in MASH.**
  Dual MoA (HSC deactivation + lipogenesis suppression) addresses both fibrosis and hepatic fat accumulation

- **Strong anti-fibrotic efficacy - Synergistic with GLP-1s in MASH**
  Oral, no CNS side effects, additive anti-fibrotic effect with Semaglutide/Empagliflozin, natural BD partner for pharma building MASH portfolios

- **Phase I de-risked; Phase IIa FDA-approved and approaching binary value inflection**
  Clean Phase 1 (n=96), no Grade 3 AEs, FDA IND approved;

- **Promising preclinical pipeline in inflammation, epilepsy and oncology**
  strong efficacy observed in IBD, kidney disease, heart disease, dravet syndrome, and cancer.

- **USD 25M raised to date; Current raise to drive IPO inflection**
  Series A and B lead by Mirae Asset Management; current raise to position well for IPO in 2 years

Core members

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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

- Prof. at GIST, department of chemistry
- 25+ years of experience in medicinal chemistry
- **5 Licensing deals** to Korean Pharma

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.

Chief Scientific Officer

- 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

Peter Goughnour/Ph.D.

Executive Director

- Former R&D Director at Curigin
- 2+ years of experience in gene therapy R&D
- 15+ years of experience in drug development

Scientific Advisory Board

UC San Diego

Rohit Loomba
M.D. / MH.Sc
Professor at UCSD

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

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

Candidate	Indication	Target	Development Phase
GM-60106	MASH/Fibrosis	HTR	Discovery · Pre-clinical · Phase 1 · Phase 2
PDK inhibitor Platform	Inflammatory Diseases*	PDK	Discovery · Pre-clinical · Phase 1 · Phase 2
GM-91466	Epilepsy (Dravet Syndrome)	Confidential	Discovery · Pre-clinical · Phase 1 · Phase 2
JD-ADC payload	Cancer	Confidential	Discovery · Pre-clinical · Phase 1 · Phase 2

*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.

Company History

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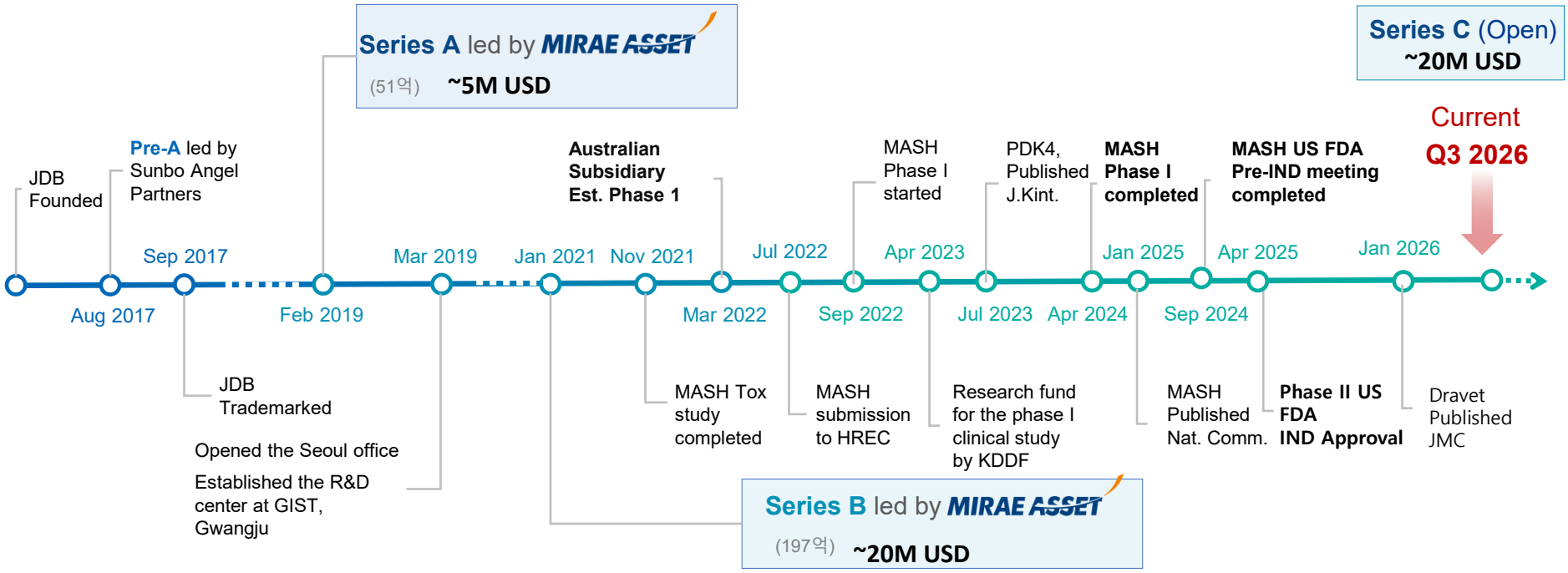
Date of establishment 31 July 2017

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



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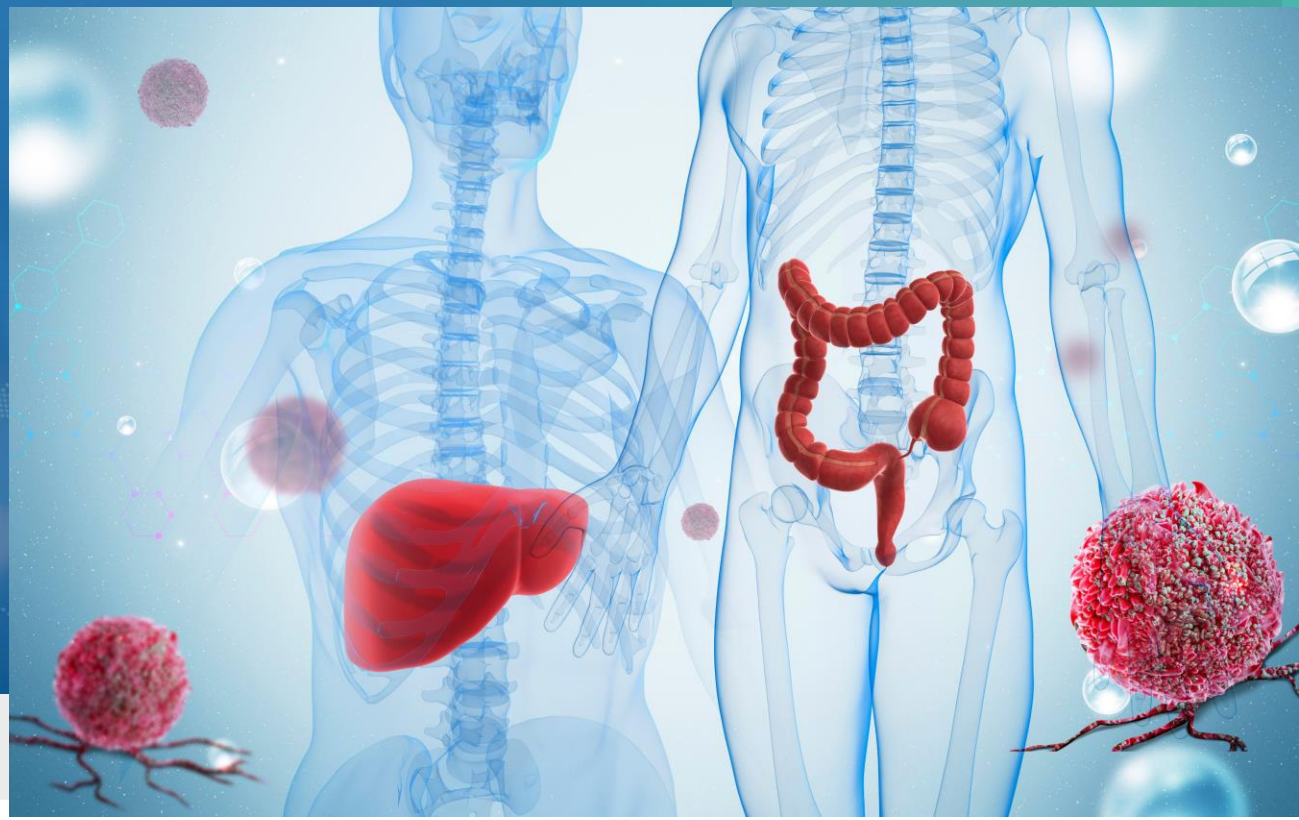
MASH

GM-60106



Executive Summary

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

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

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

04 A FIH study (n=96) 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

Current Therapies – Rezdiffra & Wegovy

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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

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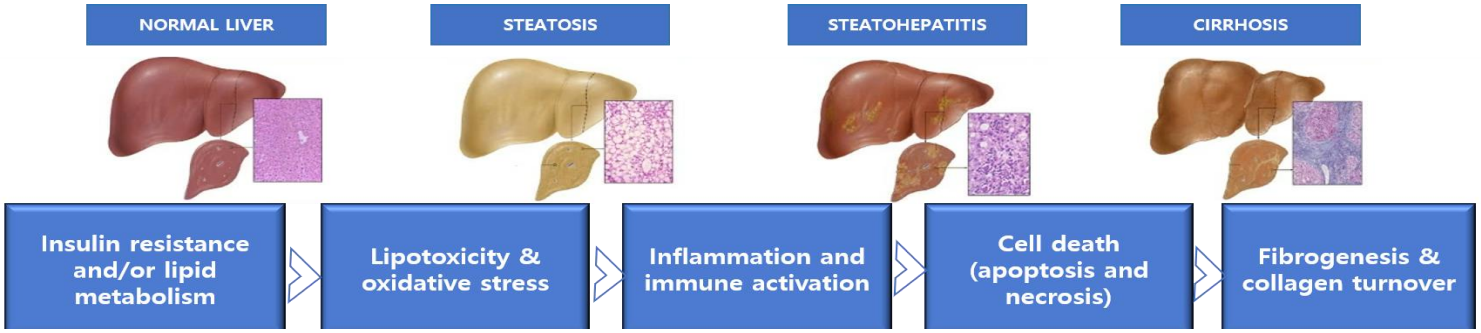
The effects of most drug candidates are focused on modulating lipid metabolism rather than targeting liver fibrosis

GM-60106 has a dual mechanism of action that simultaneously **suppresses fibrosis, hepatic fat accumulation** and **inflammation**, suggesting broad applicability in **early to late-stage liver disease**

GM-60106

Therapeutic



GLP-1	Liraglutide, Semaglutide
GLP-1/GR/GIP	MEDI0382, BI456906, Tirzeptaide, Cotadutide, HM15211
FGF21	Pegbelfremin, Efruxifermin, BIO89-100, BOS-580
THR-β	Resmetirom, VK2809
FASN	TVB-2640
DGAT2	ION224

Current Clinical Phase 2/3 targets

Preventative

wegovy® Rezdiffra™

FDA APPROVED

- × **Moderate** efficacy
- × **Preventive**, not therapeutic
- × **Limited anti-fibrotic** effect
- × Not applicable at cirrhosis

Source: Stephen Harrison AASLD 2022 modified

Serotonin Receptor(HTR2a) as a MASH Target

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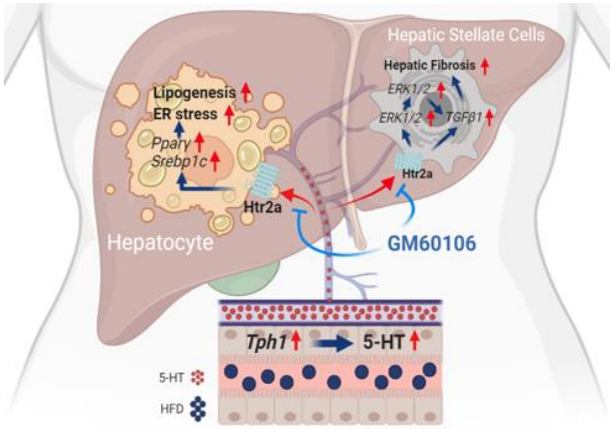
HTR2a expression is upregulated under MASH conditions in human patients, as well as in animal and cellular models

Serotonin Signaling (5-HT)

Peripheral Serotonin

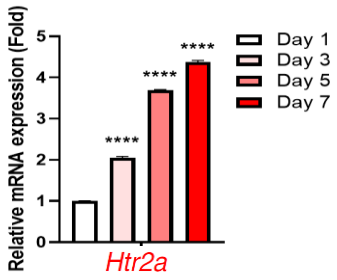
- ✓ Contributes to **Liver (scarring) Fibrosis**
- ✓ Drives **fat buildup** in body tissue
- ✓ **Liver fat** accumulation



GM-60106 Metabolic Dual Mechanism

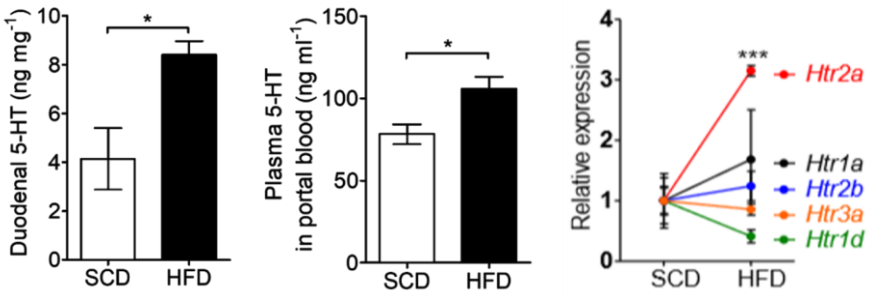
- ✓ Focuses on **Metabolism in Liver**
- ✓ **Therapeutic** vs Preventative type treatment

Cellular Model



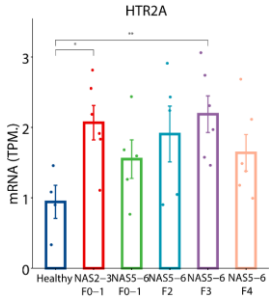
Expressions of Htr2a during activation of HSCs

Animal Model



Expressions of peripheral serotonin and its receptors in liver tissue

Human Patients



HTR2A levels in MASLD patients

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

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

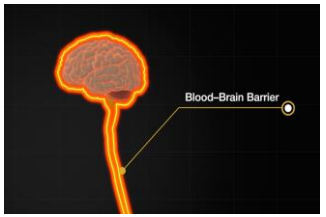
	Hours Post Dosing							
Tissue	0.25	0.5	1	6	24	48	96	168
Adrenal gland	574	1348	1007	1515	BQL	BQL	BQL	BQL
Bone Marrow (Femur)	264	387	407	1312	BQL	BQL	BQL	BQL
Bone(Femur)	209	304	BQL	351	BQL	BQL	BQL	BQL
Brain	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Eye (lens)	BQL	BQL	BQL	BQL	BQL	BQL	BQL	BQL
Fat (yellow)	182	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

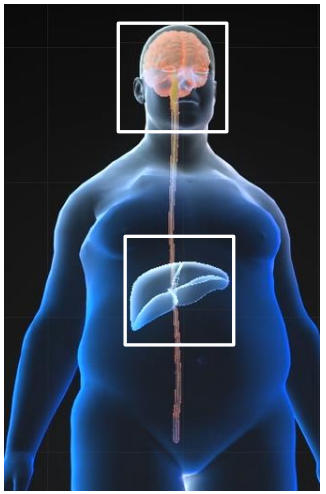
BQL = Below the lower limit of quantitation 148 ng equiv./g.

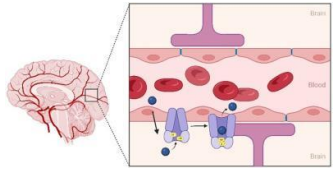
Table 9
Pharmacokinetic Parameters of GM-60106 Equivalents in Plasma and Tissues of Male Sprague Dawley Rats following an Oral Dose of [^{14}C]-GM-60106 at 5 mg/kg

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





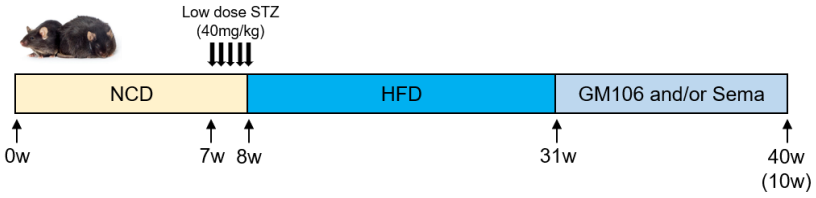


Determine [the tissue distribution and tissue pharmacokinetics] of [^{14}C]- GM-60106-derived radioactivity in the rat following a single PO administration. -> 0.25, 0.5, 1, 6, 24, 48, 96, and 168 hours post dosing (SD model, Group4)

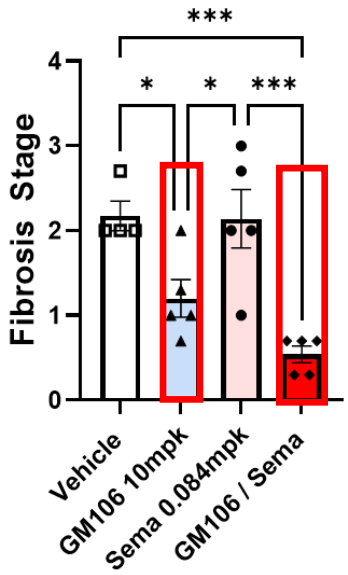
GM-60106 shows an additive effect on fibrosis stage when combined with Semaglutide (Wegovy)

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

STZ+HFD mouse (MASH model)



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

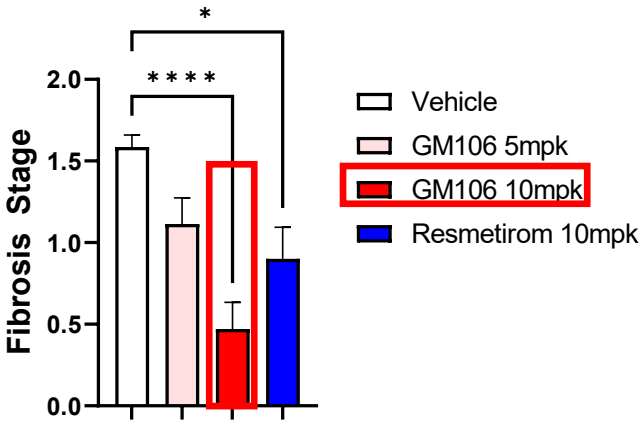
Semaglutide (Sema): an antidiabetic agent and Glucagon-like peptide-1 (GLP-1) agonist.

GM-60106 showed better fibrotic efficacy than Resmetirom (Rezdiffra)

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

STZ+HFD mouse (MASH model)

Fibrosis Stage



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

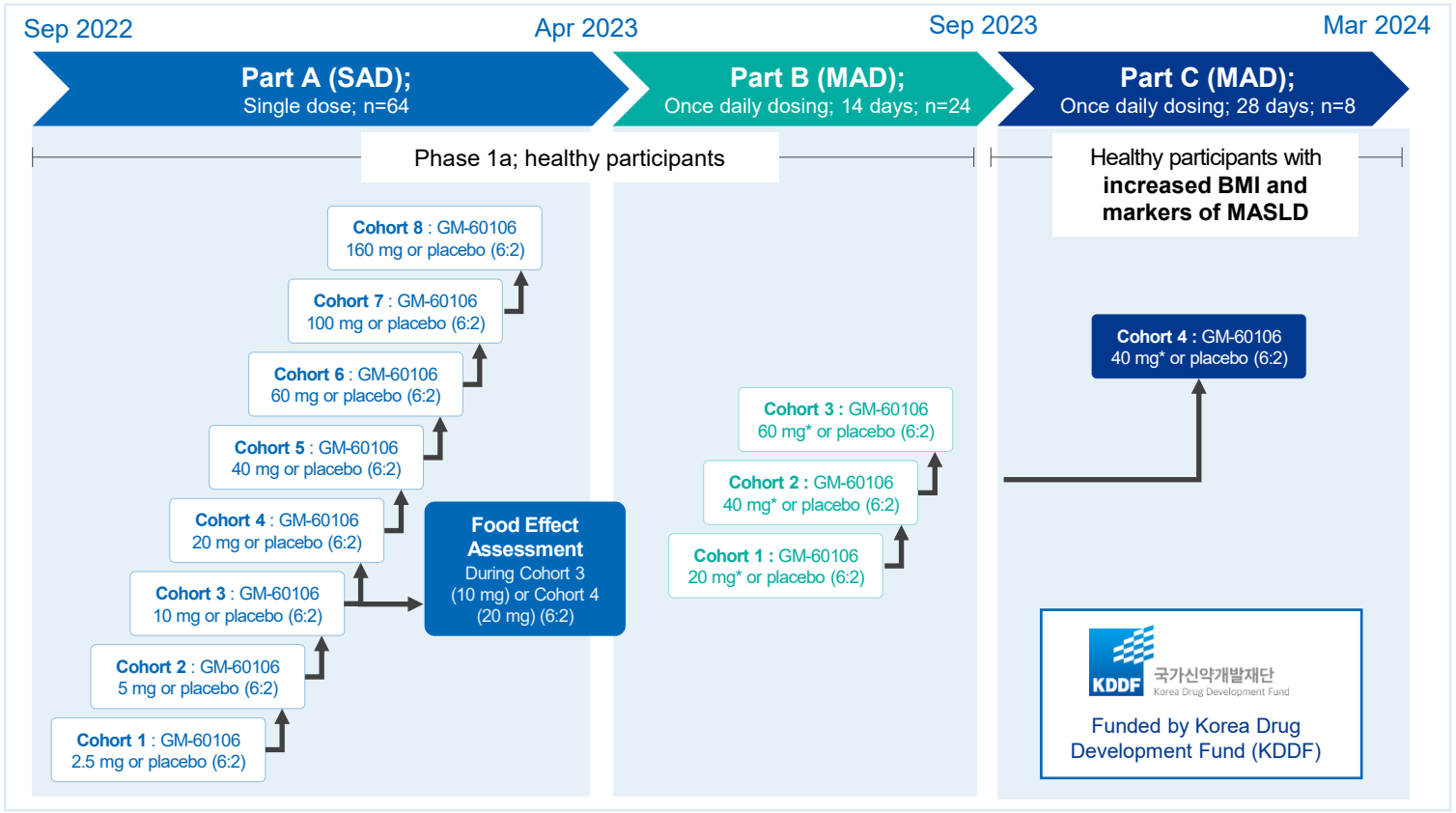
Resmetirom: Thyroid Hormone Receptor-beta (THR-B) agonist.

GM-60106 is Safe and Well-Tolerated in Phase 1a/1b Studies in 96 Healthy Adults

- Part A, B: Assessing the safety, tolerability, and PK of single or multiple doses (14 days, once daily) of GM-60106 in healthy adults
- Part C: Assessing the safety, tolerability, and PK of multiple doses (28 days, once daily) of GM-60106 in otherwise healthy adults

NCT05517564

"GM-60106 is safe and well-tolerated, PI"



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*

We've completed a phase 1 clinical trial in **Australia**

Ready for GM-60106 Phase 2a

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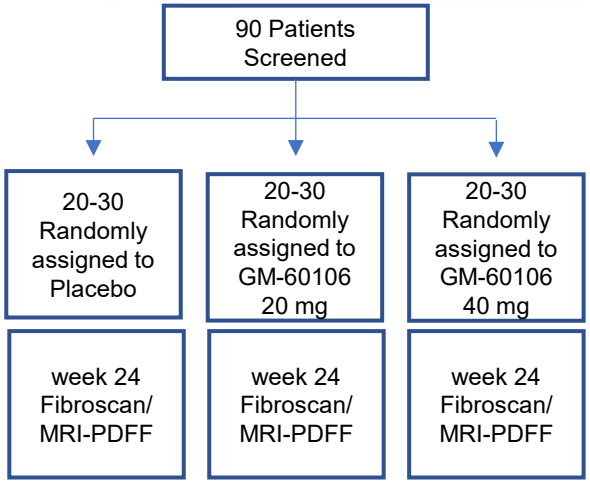
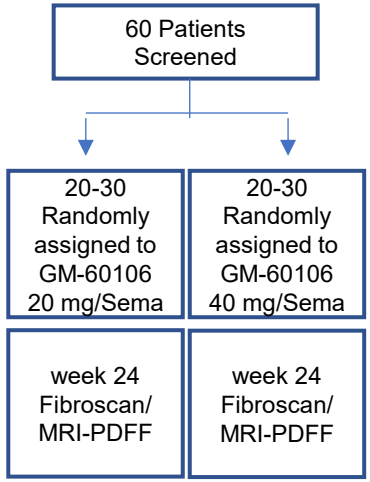
GM-60106 is ready for Phase 2a in the US with CMC completed

Approved Design



Combination Arms (planned to be added)



Planned Phase 2a

Sites

- Republic of Korea
- Europe
- USA

Timeline 2-3 years

- First Patient in- Q1 2027
- Last Patient out Q1 2029
- Complete Q2 2029

Applied for Phase II clinical trial to the U.S. FDA and received the approval in May 2025

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Inflammatory Disease

PDK4 inhibitors

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Pyruvate Dehydrogenase Kinase 4 (PDK4)

Core scientists for PDK4

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

- Former President of Korean Diabetes Association
- Prof. at Kyungpook National University Hospital, Internal medicine

KNUH KYUNGPOOK NATIONAL UNIVERSITY HOSPITAL

Prof. Jae-Han Jeon
M.D. / Ph.D.

- Prof. at Kyungpook National University Hospital, Internal medicine

KNUH KYUNGPOOK NATIONAL UNIVERSITY HOSPITAL

Prof. Dong Ho Park
M.D. / Ph.D.

- Prof. at Kyungpook National University Hospital, Ophthalmology

KNUH KYUNGPOOK NATIONAL UNIVERSITY HOSPITAL

Prof. George King
M.D.

- Research Director, Joslin Diabetes Center
- Prof. at Harvard Medical School, Department of Medicine
- Prof. at Harvard Medical School, Ophthalmology

HARVARD UNIVERSITY

PDK4 is a Novel Inflammatory Disease Target

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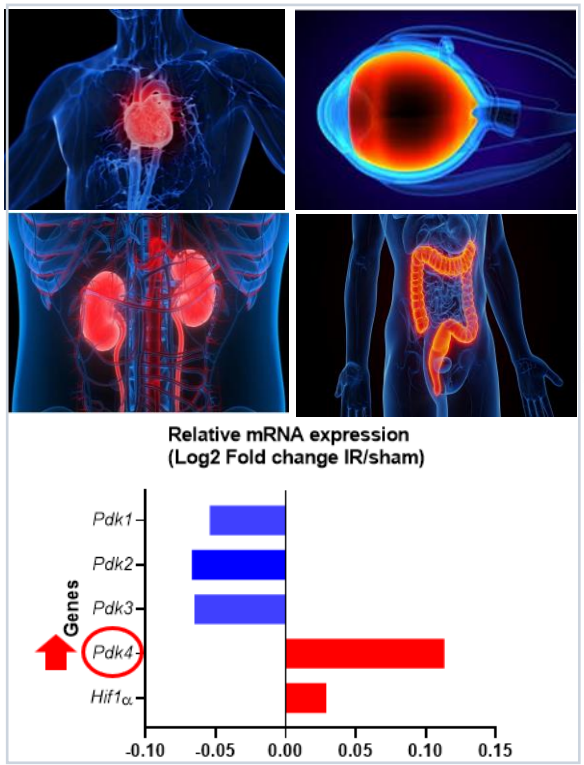
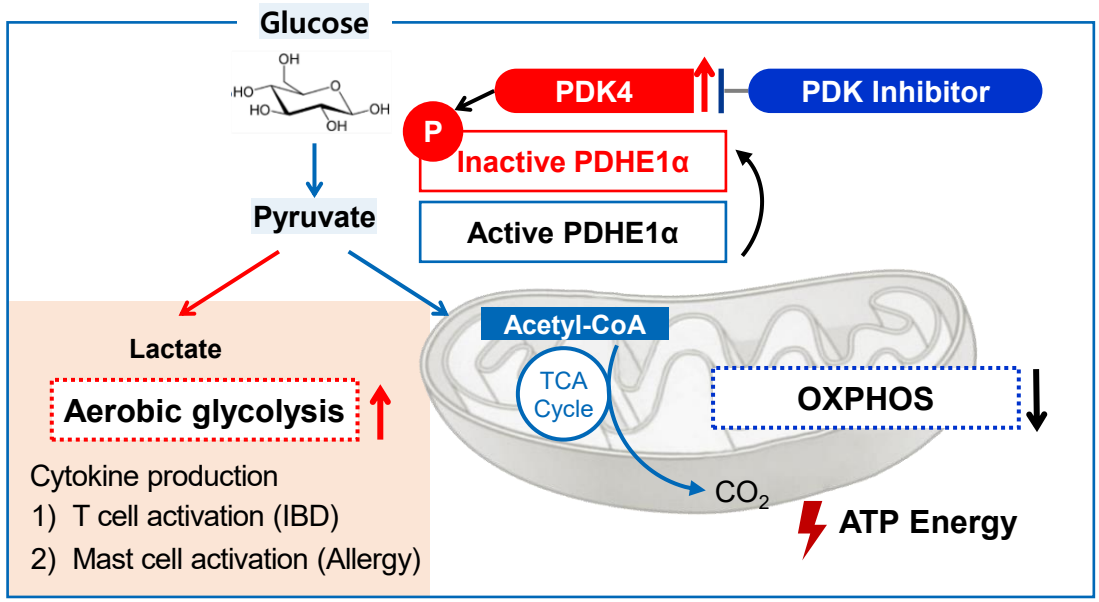
Identified a novel target for inflammatory diseases, including Ischemia/reperfusion injury (heart and kidney), Cardiomyopathy, IBD, and CKD

- 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

Executive Summary

Target : PDK4

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

Indication 1 : Heart Disease/ Acute Kidney Injury (AKI)

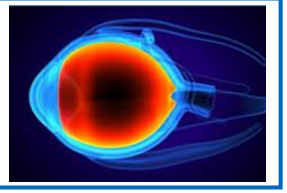
- **Preclinical studies** have **demonstrated significant improvements in cardiac function** in animal models of cardiomyopathy

Indication 2 : Inflammatory Bowel Disease (IBD)

- PDK4i **suppress the activation of CD4+ T cells,** a key driver in the progression of IBD
- Preclinical studies of **PDK4i demonstrate significant anti-inflammatory effects** in DSS-induced IBD model
- **Reduces inflammation** and **restores immune homeostasis**

Indication 3 : Age-Related Macular Degeneration (AMD)

- PDK4 inhibition **alleviates neovascularization**
- **In addition**, it **reduces HIF1α and VEGF expression** along with **inflammatory cytokines**



JD BIOSCIENCE

Dravet

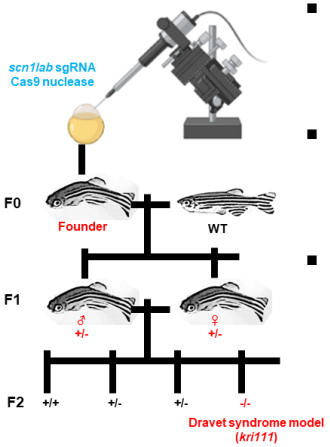
GM-91466

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)

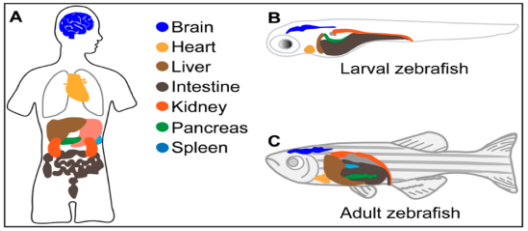
Generation of Mutant Zebrafish Model



- 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

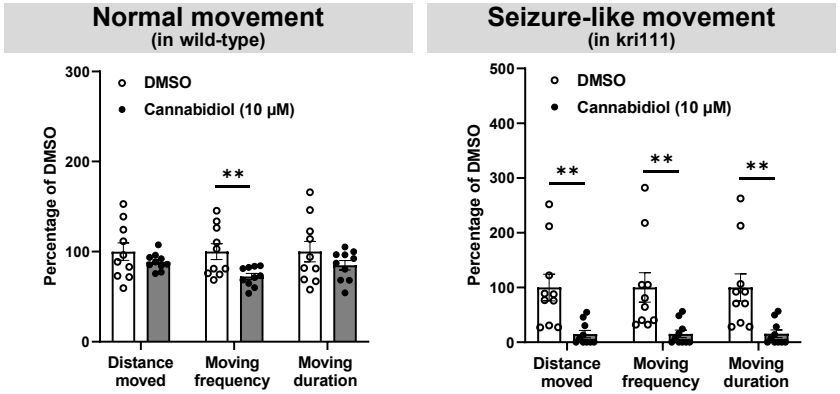
Advantages of Zebrafish Model

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

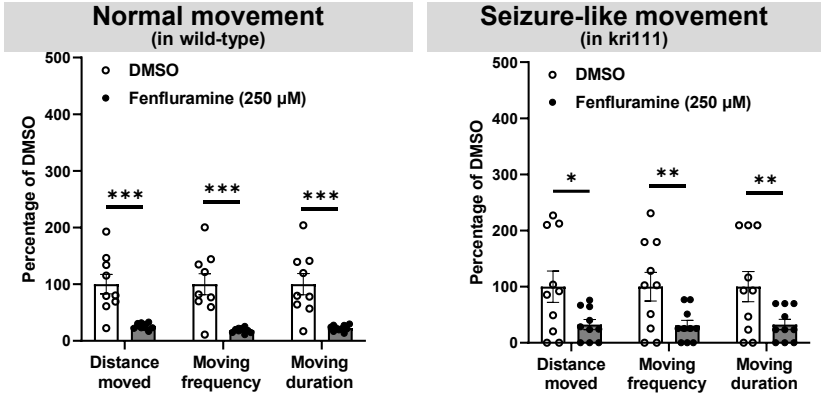


Dravet Drug Evaluation in SCN1A-Mutant Zebrafish

- 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

Executive Summary

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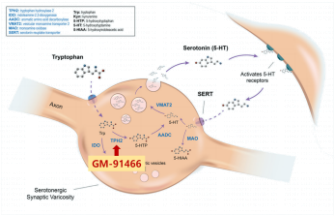
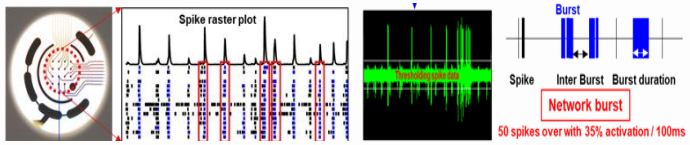
Mode of Action

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



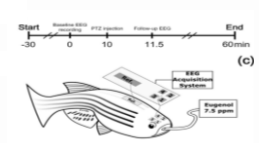
Neural Organoid Model

- 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



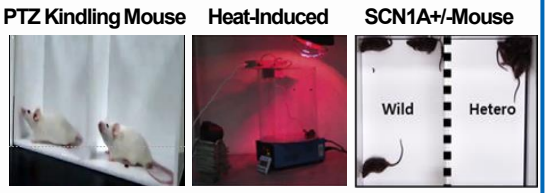
Zebrafish Model

- 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



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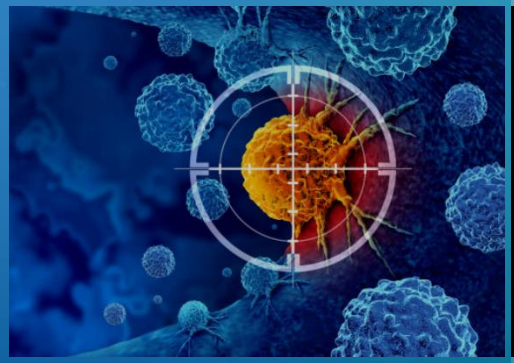
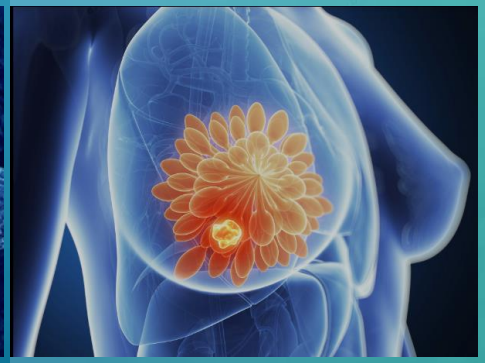
Cancer

JD-ADC payload





JD Bioscience ADC Payloads Programs

JD BIOSCIENCE

01

CPT & Non-CPT Topoisomerase 1 Inhibitors

next-gen CPT & Non-CPT scaffolds overcoming DXd/SN-38 limitations.
400+ compounds, **sub-nM and pM potency**.

400+
compounds

02

Degraders

Molecular glue degraders inducing synthetic lethality.
90+ compounds with catalytic MOA and BRCAness induction.

90+
compounds

03

TLR7 Agonists

Highly selective TLR7 agonists for ISAC or dual payload applications.
Designed to overcome CRS and ADA challenges of 1st-gen ISACs.

Executive Summary

JD BIOSCIENCE

- 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**

JD BIOSCIENCE

Strategical Development

Investment Fund Usage Plan

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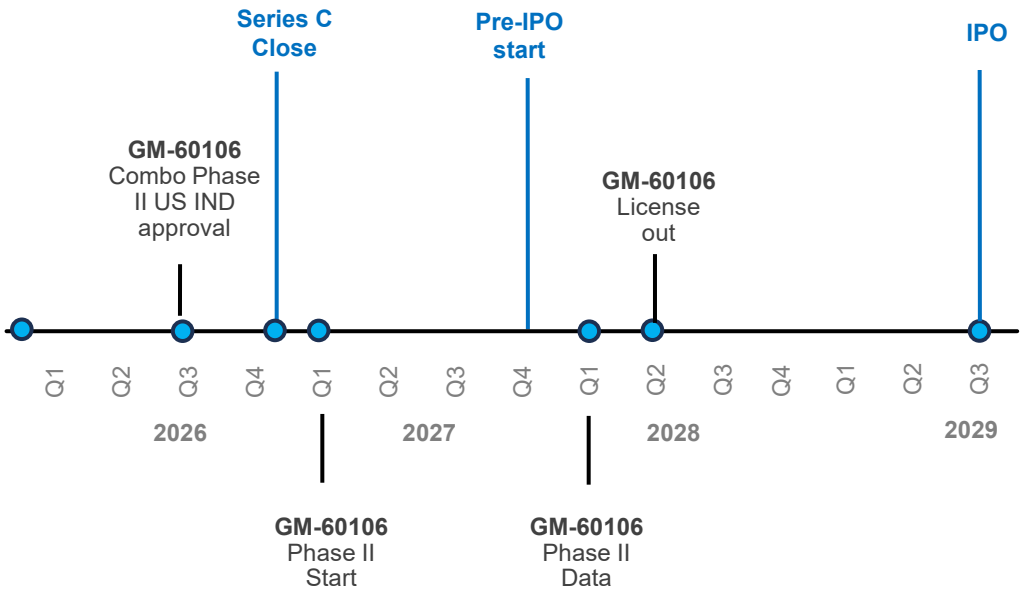
Use of Proceeds – USD 20M

- **MASH: USD 12M**
- **Ani-inflammatory: USD 3M**
- **Epilepsy: USD 3M**
- **ADC: USD 2M**



JD Bioscience Inc.- Executive Summary

JD BIOSCIENCE

- **First-in-class oral therapy targeting liver fibrosis-the final unmet need in MASH.**
  Dual MoA (HSC deactivation + lipogenesis suppression) addresses both fibrosis and hepatic fat accumulation
- **Strong anti-fibrotic efficacy - Synergistic with GLP-1s in MASH**
  Oral, no CNS side effects, additive anti-fibrotic effect with Semaglutide/Empagliflozin, natural BD partner for pharma building MASH portfolios
- **Phase I de-risked; Phase IIa FDA-approved and approaching binary value inflection**
  Clean Phase 1 (n=96), no Grade 3 AEs, FDA IND approved;
- **Promising preclinical pipeline in inflammation, epilepsy and oncology**
  strong efficacy observed in IBD, kidney disease, heart disease, dravet syndrome, and cancer.
- **USD 25M raised to date; Current raise to drive IPO inflection**
  Series A and B lead by Mirae Asset Management; current raise to position well for IPO in 2 years

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:

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JD Bioscience is seeking:

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