CASPIAN THERAPEUTICS

Corporate and Clinical Update

October 13, 2026

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PARTICIPANTS

Troy Wilson, Ph.D., J.D.
President & Chief Executive Officer, Kura Oncology
Founder and Executive Chairman of the Board, Caspian Therapeutics

Francis Burrows, Ph.D.
Chief Scientific Officer, Kura Oncology

Robert Spencer, Ph.D.
President & Chief Operating Officer, Caspian Therapeutics

MORE THAN A DECADE OF MENIN RESEARCH AT KURA

Scientific Foundation

- Pioneering research in the biology of menin inhibition
- Chemistry effort over 10 years has produced an extensive portfolio of proprietary menin inhibitors
- Kura contributed KO-7246, related IP, and know-how to Caspian

Dedicated Development

- Diabetes requires specialized capabilities and a strategy tailored to metabolic disease
- Caspian provides dedicated leadership and financing
- Shared services enable Caspian to focus on preclinical and clinical development
- Caspian is led and advised by diabetes experts

Novel Mechanism of Action

- Following fundamental biology
- Selective stimulation of beta islet cell proliferation
- Potential to combine with current and emerging standards of care, including GLP-1s

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FINANCING

Capital aligned to initial clinical proof-of-concept

- **$50 million financing** led by BVF Partners
- Investors include **T1D Fund, Invus, Montanova, Eli Lilly and Company, Kura**, and others
- Kura retains approximately **half ownership of Caspian**, giving Kura's stockholders participation in Caspian's **potential future value**
- Proceeds are expected to fund KO-7246 **IND-enabling development and initial clinical proof-of-concept**, plus advancement of a **second menin inhibitor candidate for diabetes and cardiometabolic diseases**

OPERATING MODEL

Dedicated leadership with access to established Kura capabilities

- **Caspian leads development**
  - Robert Spencer, President and Chief Operating Officer, leads program development
- **Program responsibility**
  - Seasoned Board of Directors provides oversight
- **Kura capabilities**
  - Caspian has access to Kura's research, development, and corporate capabilities through an Intercompany Services Agreement
- **Development focus**
  - Established expertise and infrastructure allow Caspian to direct resources toward development

Next-Generation Menin Inhibitor KO-7246 Regenerates β Cell Mass in Diabetic Rodent and Human Islet Models

Asako McCloskey, Yahu Liu, and Francis Burrows
Kura Oncology, Inc.

62nd European Association for the Study of Diabetes (EASD) Annual Meeting
Milan, Italy
September 29, 2026

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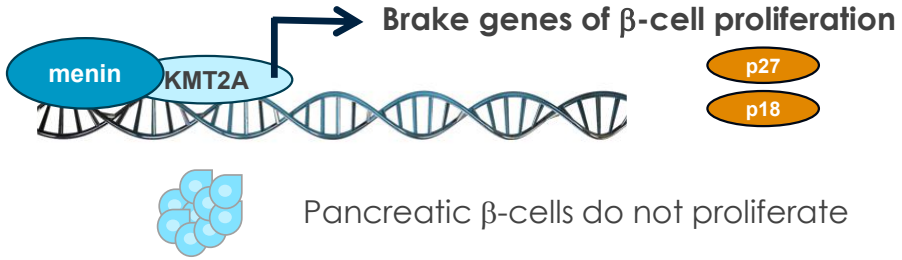
MULTIPLE LINES OF EVIDENCE SUPPORT DISEASE-MODIFYING POTENTIAL FOR MENIN INHIBITORS IN THE TREATMENT OF DIABETES

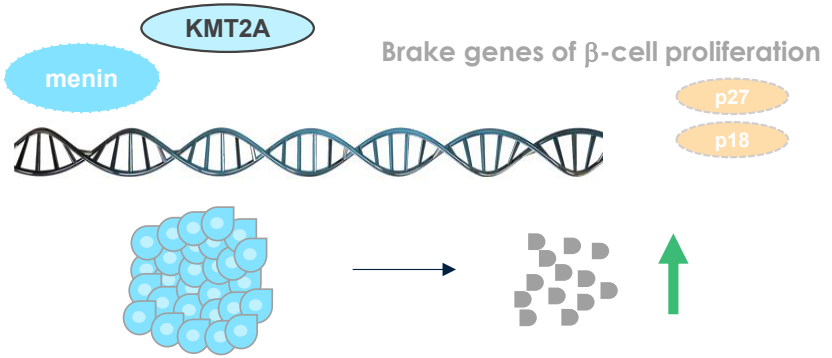
- **Mammalian physiology:** Pregnancy demonstrates that lowering menin signaling is associated with adaptive β-cell expansion.[1]
- **Human Genetics:** MEN1 loss establishes that reduced menin activity can profoundly increase pancreatic β-cell proliferation.[2]
- **Pharmacologic Validation:** Several chemically distinct menin inhibitors now provide a way to control this pathway reversibly.
- **Implication:** These observations support pharmacologic menin inhibition as a strategy to restore functional β-cell capacity in both T1D and T2D.

Menin as an epigenetic molecular brake of β-cell proliferation

i) Under normal physiological conditions



ii) During pregnancy



[1]Karnik S.K. *et al. Science* 2007, 318(5851): 806-809.
[2]Agarwal S. *et al. Ann N Y Acad Sci*. 2004, 1014:189-98.

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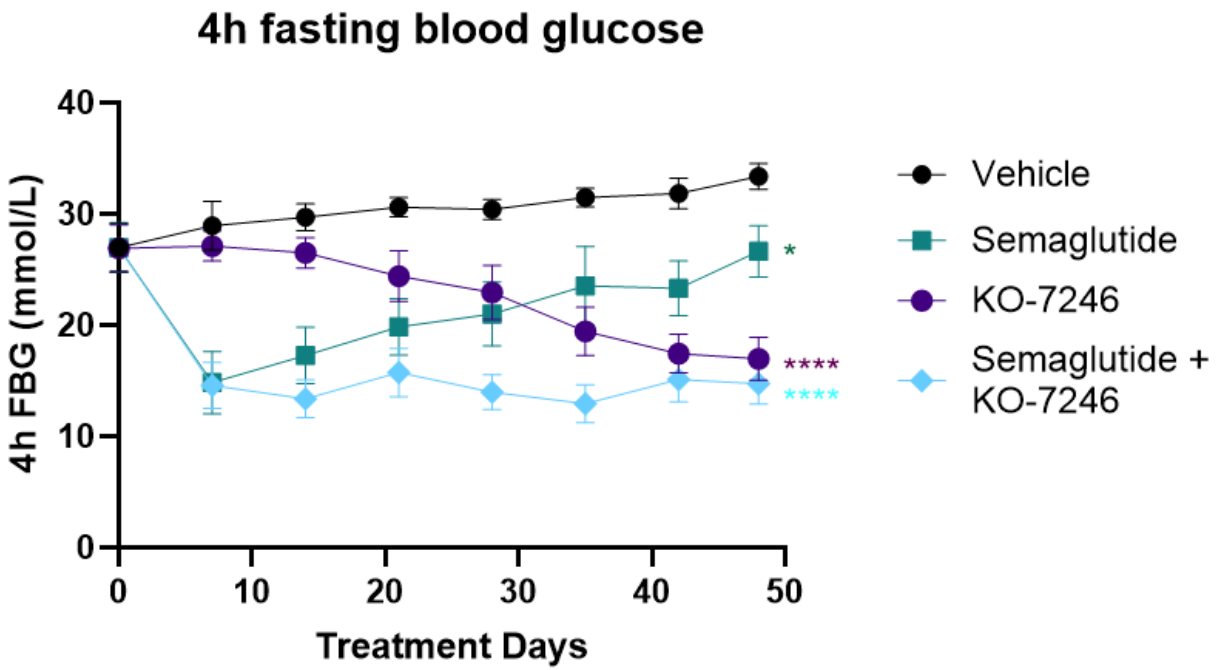
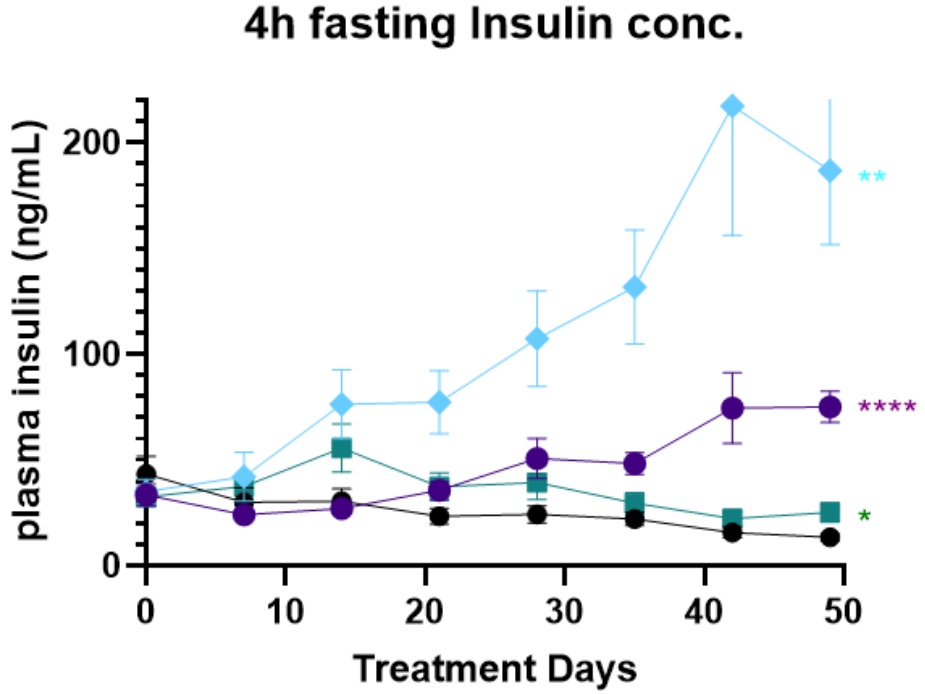
KO-7246 PROVIDES DURABLE GLYCEMIC CONTROL AND COMPLEMENTS GLP-1 THERAPY IN DB/DB MOUSE MODEL OF TYPE 2 DIABETES





- Semaglutide reduced blood glucose levels rapidly but transiently
- KO-7246 significantly reduced FBG and elevated insulin levels over a 7-week period
  - First-generation menin inhibitor ziftomenib produced similar results[1]
- No hypoglycemia observed in any KO-7246-treated animals

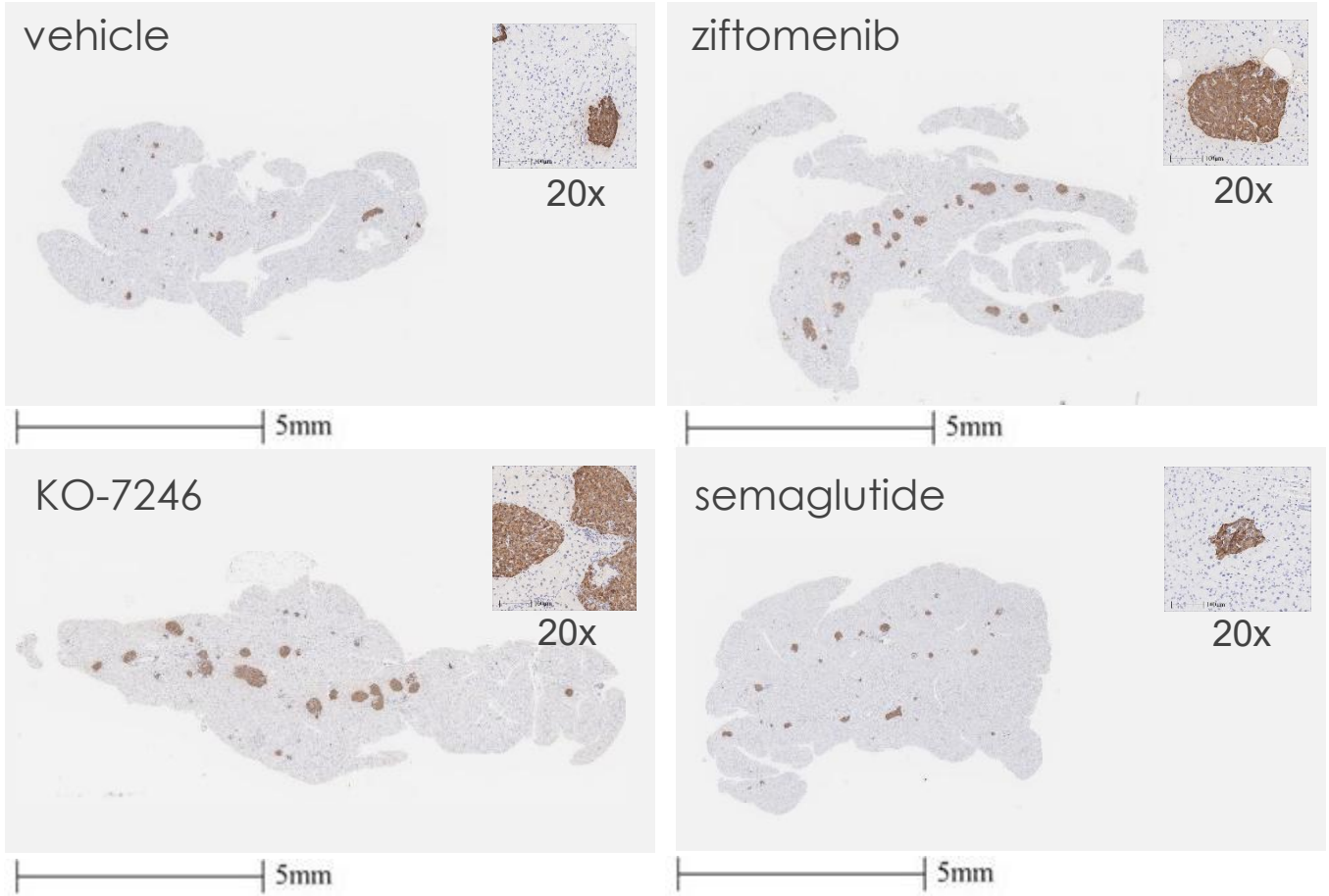
[1]McCloskey A. et al., Poster 845-P, Presented at the 84th American Diabetes Association Scientific Sessions, June 21-24, 2024.

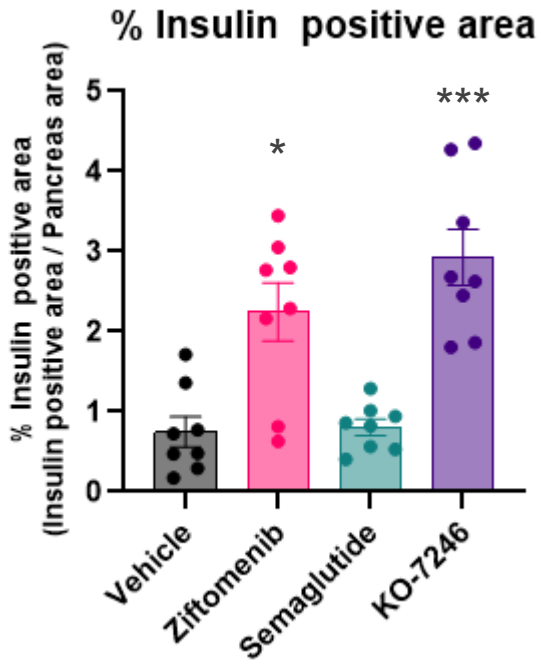
*, p<0.05; ****, p<0.001

KO-7246 EXPANDS PANCREATIC B-CELL MASS IN DB/DB PRECLINICAL MODELS

Expansion provides direct evidence supporting proposed regenerative mechanism

Insulin IHC (β-cell signals in **brown**)





10 mice/group, 49d treatment period; each point represents the average of three slices/mouse

*, p<0.05; ***, p<0.005

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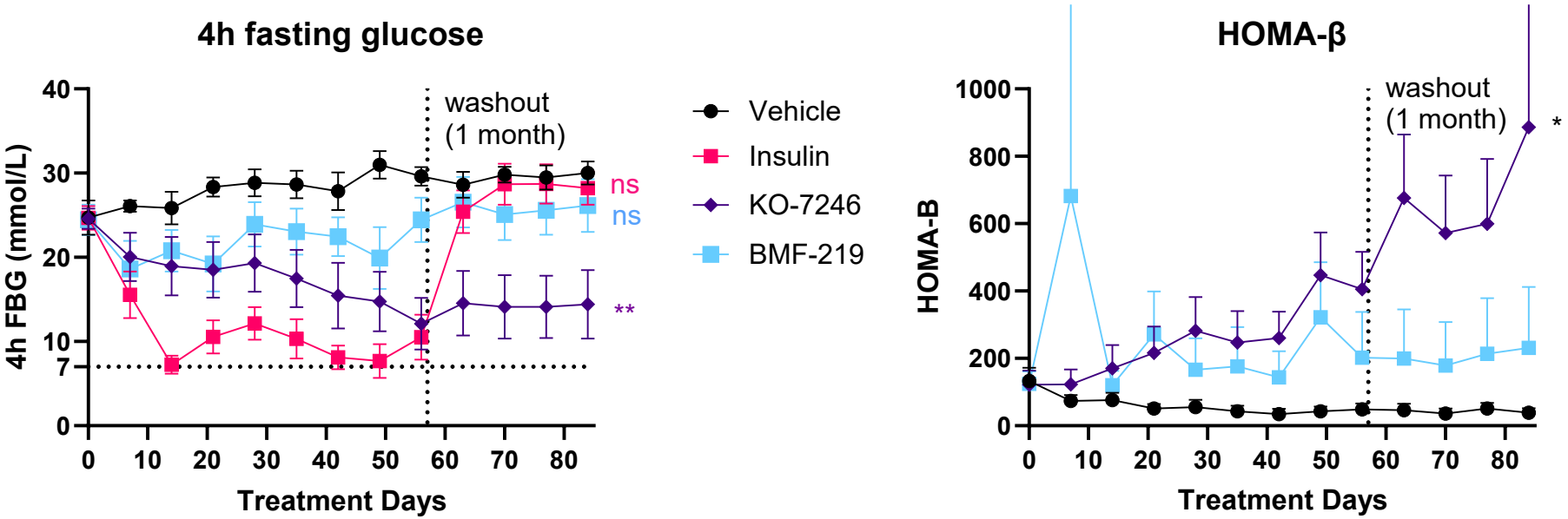
KO-7246 DEMONSTRATED ROBUST, DURABLE GLUCOSE REDUCTION ACTIVITY IN STZ RAT MODEL OF TYPE 1 DIABETES

Full restoration of glycemic control achieved in model of insulin insufficiency

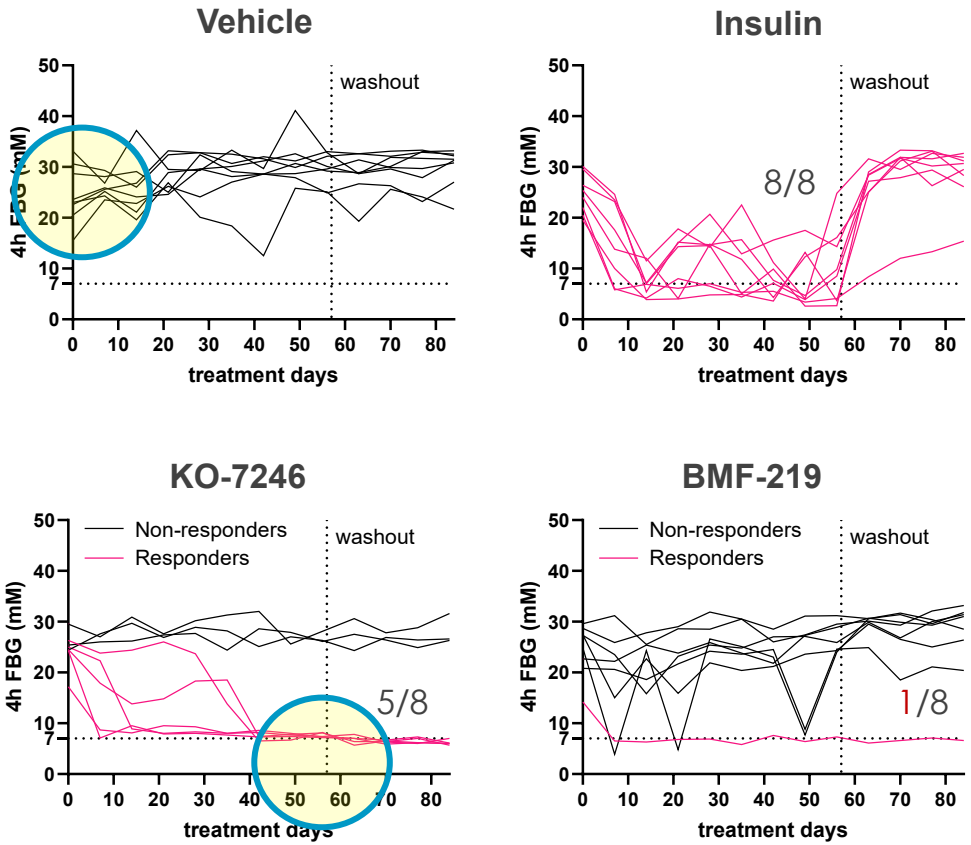


- KO-7246 robustly and durably reduced FBG and enhanced HOMA-β, comparing favorably to BMF-219
- Kinetics and post-dose stability of activity support β-cell mass regeneration MOA of KO-7246

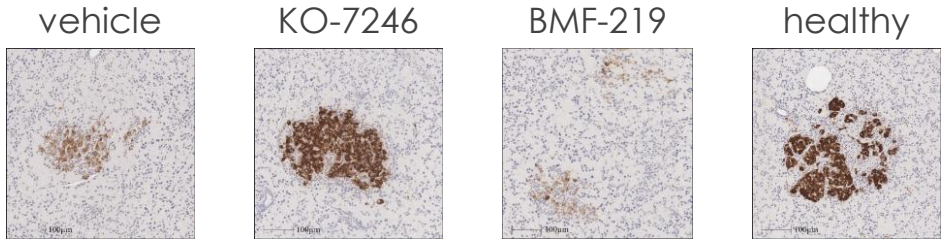
ns; not significant; *, p<0.05; **; p<0.01

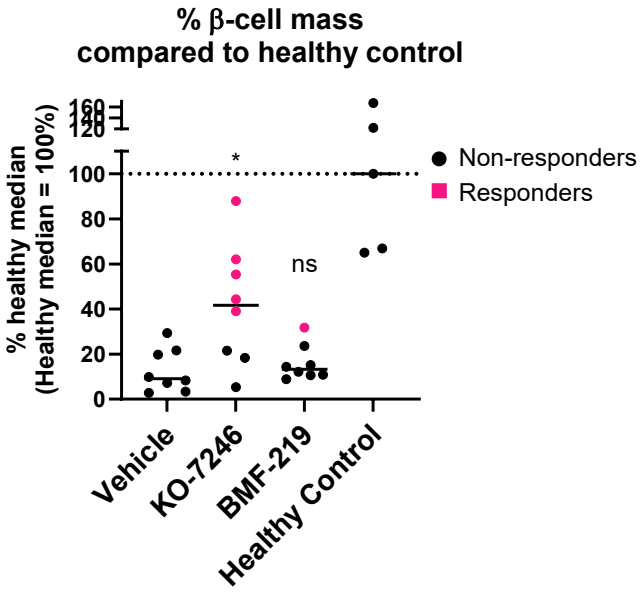
KO-7246 BUT NOT BMF-219 FULLY RESTORED ISLET FUNCTION IN STZ T1D RAT MODEL

Response appeared dependent on residual β-cell capacity, consistent with regenerative mechanism



Representative islet images
(insulin IHC, 20x)





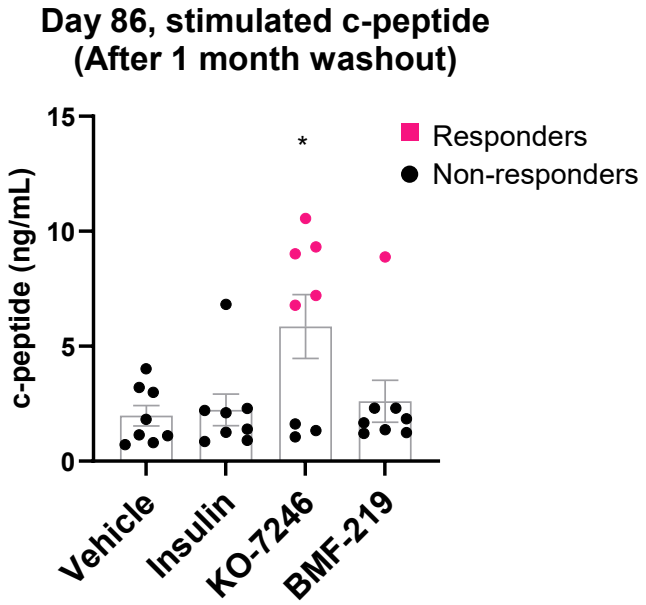
- Responders all achieved and maintained complete glycemic control
- Islets regenerated to 40-90% of healthy controls by Day 56
- BMF-219 did not reduce FBG or increase β-cell mass

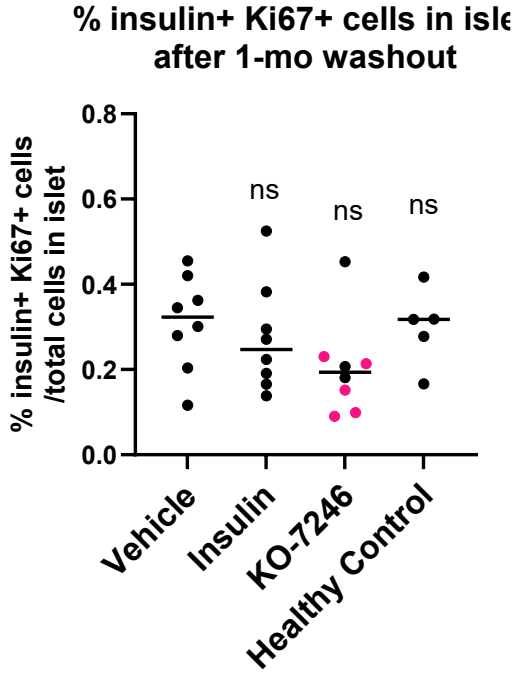
ns; not significant; *, p<0.05

TRANSIENT PHARMACOLOGIC MENIN INHIBITION IS FUNDAMENTALLY DIFFERENT FROM LIFELONG MEN1 LOSS

Enhanced endogenous insulin production persists after β-cell proliferation

- **MEN1 Syndrome patients develop proliferative insulinomas after years/decades of biallelic MEN1 loss**
  - > 500 AML patients have been dosed daily with menin inhibitors for multiple years without MEN1 Syndrome symptoms
- **Animals dosed to Day 56 and observed to Day 86**
  - C-peptide significantly elevated after one month washout (indicating greater β-cell mass)
  - Negligible levels of residual cell proliferation in all groups (indicating limited active cell division)

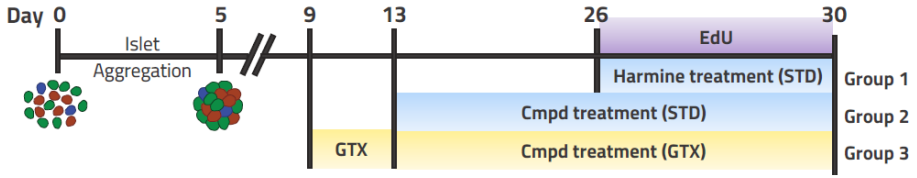


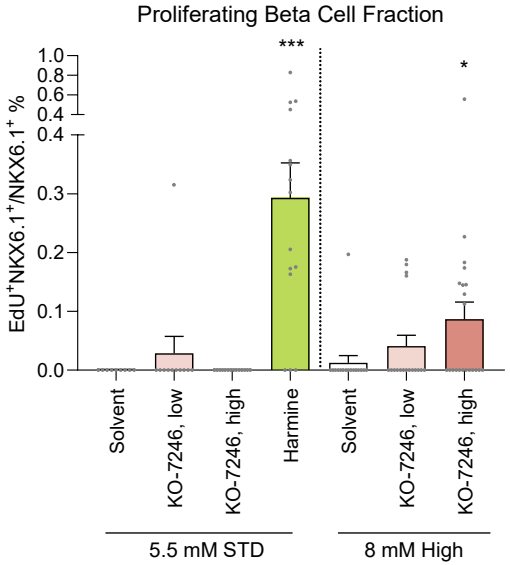


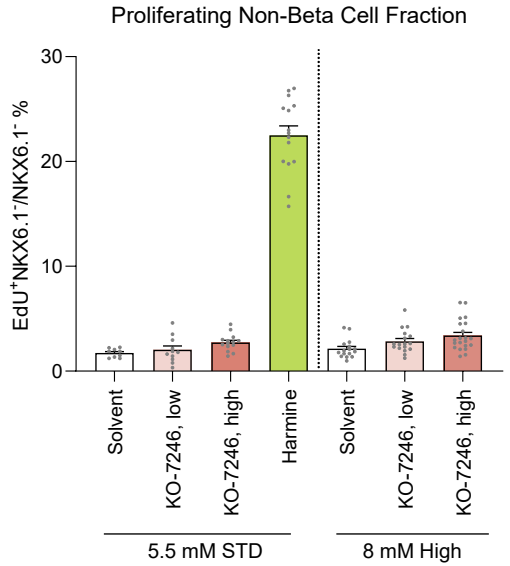
ns; not significant; *, p<0.05

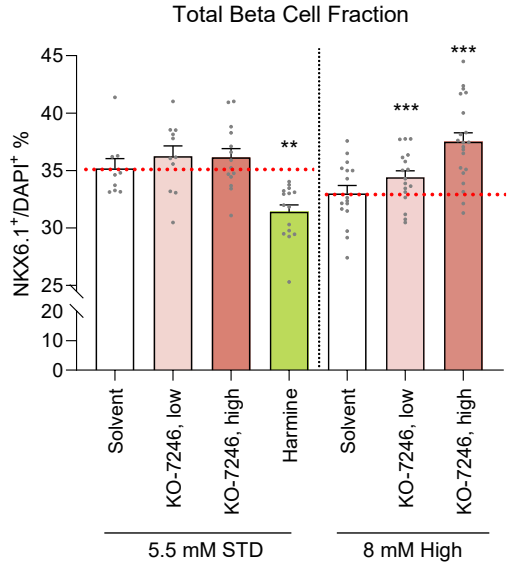
KO-7246 INDUCED PROLIFERATION OF HUMAN β-CELLS

KO-7246 induced β-cell proliferation in multiple donor samples









- KO-7246 dose-dependently induced β-cell proliferation under glucotoxic conditions
- DYRK1A inhibitor harmine was unselective

- Harmine also induced proliferation of non-β-cells
- KO-7246 had no effect on non-β-cell proliferation

- KO-7246 dose-dependently increased the β-cell fraction under glucotox conditions
- Harmine had a net negative effect on the β-cell fraction

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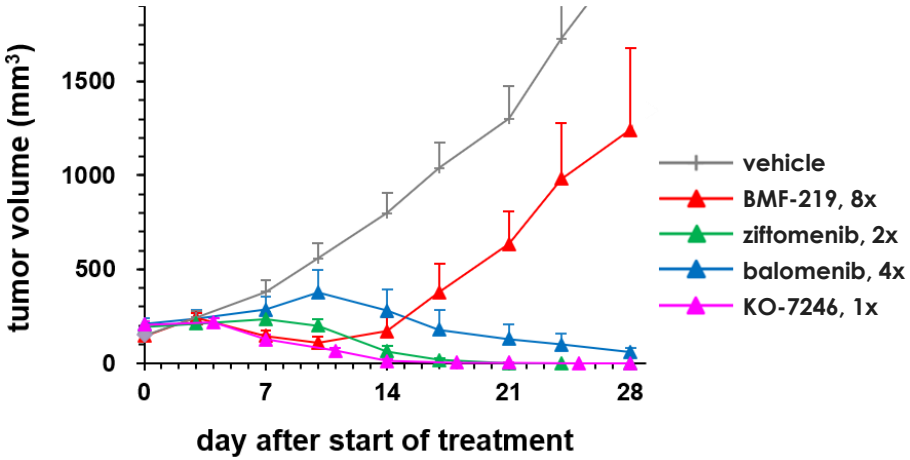
ns; not significant; *, p<0.05; **; p<0.01, ***, p<0.005; ****, p<0.001

KO-7246 AND BALOMENIB ARE DIFFERENTIATED FROM BMF-219

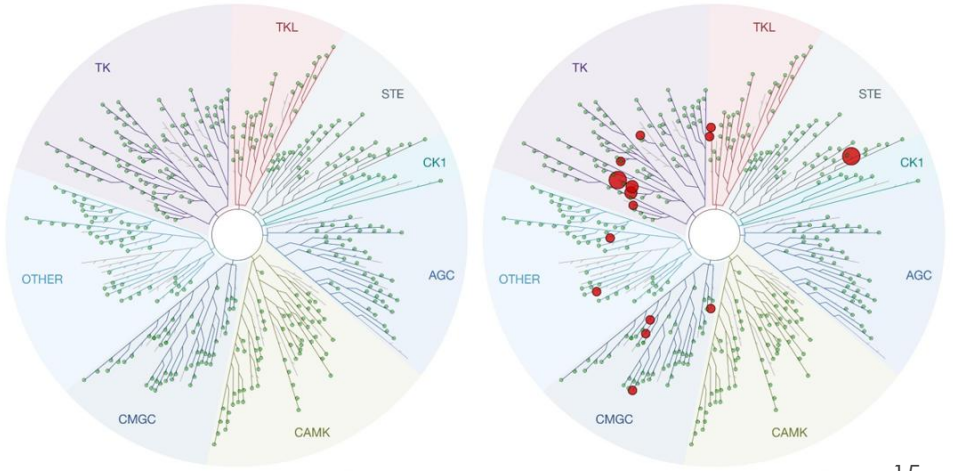
	KO-7246	balomenib	Icovamenib (BMF-219)
menin-MLL FP IC_{50} (nM)	7.7	23.1	>10,000
MV4-11 CTG IC_{50} (nM)	7.9	28.1	218
mouse MV4-11 CDX model	robust efficacy	efficacy at 4x daily dose	no efficacy even at 8x daily dose
CDK9 %inhibition at 1.0 µM	0	not tested	>80 (IC_{50} =19.1 nM)

Menin-Dependent AML Model



Kinase Family Tree

KO-7246 **BMF-219**



- KO-7246 and balomenib are validated menin inhibitors, but KO-7246 is more active in vitro and in vivo
- BMF-219/Icovamenib inhibits several kinases, including CDK9, but has no discernable activity towards menin

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KO-7246 HAS POTENTIAL TO BE A DISEASE-MODIFYING THERAPY FOR PATIENTS WITH TYPE 1 OR TYPE 2 DIABETES

- Menin inhibition has the potential to restore endogenous β-cell capacity in patients with Type 1 and Type 2 diabetes
- KO-7246 combines a validated menin-based mechanism of action with robust, disease-modifying pharmacology across a range of diabetes models
  - Potent menin inhibition demonstrated in multiple *in vitro* and *in vivo* menin-dependent models
  - Produced durable glycemic control through β-cell regeneration in multiple preclinical diabetes models
- IND-enabling development of this potential best-in-class menin inhibitor underway with initial clinical evaluation planned thereafter

KO-7246 CLINICAL DEVELOPMENT

Initial objective: safety, exposure and endogenous insulin production

Clinical entry

IND-enabling studies to establish the nonclinical safety and exposure profile in parallel with manufacturing and drug supply readiness to support clinical entry.

Chronic administration

Characterize safety, tolerability, pharmacokinetics and pharmacodynamics to inform repeated dosing.

Patient selection

Consider residual beta cell capacity and baseline endogenous insulin production. Both Type 1 and Type 2 diabetes are relevant to the broader opportunity.

Initial proof of concept

Acceptable safety and exposure with interpretable improvement in endogenous insulin production, assessed using stimulated C-peptide.

Q&A

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