FcRn-IgG MODULATION AT THE GUT WALL

A Mechanism-Distinct Path to Metabolic Disease: Resetting the Gut's Metabolic Memory

U.S. Provisional Patent G4590-23800P • Inventor: Hong-Hsing Liu, MD, PhD

THE PROBLEM Diet change can remove the trigger but leave metabolic inertia behind.
THE TARGET We identified a druggable gut-wall FcRn-IgG axis that keeps this adverse metabolic state anchored.
THE ASSET Our patent-pending method disrupts this axis to arrest post-diet lipid deterioration.

WHAT IT IS

Methods and uses to disrupt the FcRn-IgG molecular handshake at the gut wall to regulate host lipid metabolism.

WHY IT'S DIFFERENT

- Lipid drugs manage the numbers. We go after the source.
- Modulating intestinal FcRn-IgG may reset the gut's metabolic memory and stop lipid rebound.
- A source-directed approach to metabolic disease modification.

WHAT WE'VE SHOWN

- *In vivo* proof-of-concept across two independent murine models.
- Axis disruption prevents delayed TG and TG/HDL elevation after high-fat-to-chow diet switch.
- Benefit occurs without detectable systemic IgG depletion.
- Supports gut-directed intervention against post-diet metabolic inertia.

PATENT SCOPE

U.S. provisional filed January 2026. Broad method-of-use claims pending for gut-wall FcRn-IgG modulation in lipid rebound and obesity-related metabolic disease.

MARKET CONTEXT

- Weight-loss and diet interventions leave a major unmet need: preventing metabolic rebound after the trigger is removed.
- MASH and obesity pharmacotherapy are rapidly expanding markets, with forecasts reaching >$30B for MASH and $100B+ for obesity drugs.
- Rezdiffra's $958M 2025 net revenues validate commercial demand for metabolic-liver disease therapeutics.
- Anti-FcRn is a validated multi-billion-dollar drug class, creating a de-risked starting point for translating this gut-wall mechanism.

WHY A PARTNER CARES

- Targets the post-intervention gap: metabolic rebound after diet or weight-loss treatment.
- Creates a gut-directed, mechanism-distinct path for lipid rebound and fatty liver disease.
- Gives FcRn drug owners a new metabolic application beyond systemic IgG depletion.
- Complements GLP-1, THR-β, and PPAR programs without pathway overlap.

OUR ASK

Seeking partners to evaluate, option, license, or co-develop this gut-wall FcRn-IgG therapeutic opportunity. We bring the science, *in vivo* proof-of-concept, and patent-pending methods; partners bring molecule, formulation, IND-enabling, and clinical-development capacity.

ORIGIN

Inventor: Hong-Hsing Liu, MD, PhD, National Health Research Institutes (NHRI), Taiwan. Government research institute with active Tech Transfer & Incubation Center.

BIO 2026 PARTNERING • San Diego, June 22-25 • Available for One-on-One meetings

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