Weill Cornell Medicine

UCP1 Activators Suppressing Adipocyte Inflammation for Next Generation Obesity Management

Lead Inventors:

Lotfi Chouchane, Ph.D., D.Sc.

Professor of Microbiology and Immunology, Microbiology and Immunology , Weill Cornell Medical College

Professor of Genetic Medicine, Genetic Medicine, Weill Cornell Medical College

Business Development Contact:

Mina Zion

Associate Director, Innovation and Commercialization, WCM-Qatar

(646) 814-4907

mwz4001@qatar-med.cornell.edu

UCP1 Activators Suppressing Adipocyte Inflammation for Next Generation Obesity Management

Background & Unmet Need

- Elevated adipose inflammation is a hallmark of obesity and is a key driver of insulin resistance, type 2 diabetes, and cardiovascular disease
- While GLP-1 receptor agonists have emerged as effective agents in promoting weight loss in obesity, patients demonstrate high discontinuation rates due to gastrointestinal side effects
- Moreover, these medications do not address the chronic inflammation and metabolic dysfunction that arise from adipose tissue remodeling in obesity
- Brown adipose tissue (BAT) is a key regulator of thermogenesis and metabolism in adults, not only elevating caloric dissipation but also exerting paracrine anti-inflammatory effects
- Uncoupling protein 1 (UCP1) plays a central role in promoting the browning of white adipocytes and presents a promising therapeutic target
- **Unmet Need:** Non-GLP-1 strategies for managing obesity and associated inflammatory comorbidities

Technology Overview

- **The Technology:** CDC1011, a small molecule HDAC inhibitor inducing UCP1, for promoting browning of white adipocytes in obesity-related metabolic disorders
- **The Discovery:** The inventors generated an in-house multicomponent reaction (MCR)-based chemical library (>21K compounds) and developed a proprietary high-throughput screening platform to identify UCP1 activators in human adipocytes
- Ten hit compounds were identified for further development, including lead compound CDC1011
- **PoC Data:** CDC1011 induces browning in pre- and mature white adipocytes, upregulating β3-adrenergic receptor (ADRB3), brown/beige markers, and mitochondrial electron transport chain subunits
- CDC1011's functional effects include accelerated lipolysis, glucose uptake, metabolic flexibility, and a shift from proinflammatory to anti-inflammatory cytokine profiles

Inventors:
Lotfi Chouchane
Jingxuan Shan

Patents:
Provisional Filed

Publications:
Rammah et al. *J Trans Med.* 2026.

Biz Dev Contact:
Mina Zion
(646) 814-4907
mwz4001@qatar-med.cornell.edu

Cornell Reference:
D-11463

Weill Cornell Medicine

UCP1 Activators Suppressing Adipocyte Inflammation for Next Generation Obesity Management

Technology Applications

- Management of obesity as a single agent or in combination with GLP-1 receptor agonists
- Management of insulin resistance and type 2 diabetes
- A next-generation metabolic solution for weight management in the general population

Technology Advantages

- Functions via a separate biological mechanism than GLP1 receptor agonists
- Addressees not only weight reduction but also biology underlying metabolic disorder and associated inflammatory comorbidities
- Reprograms white adipose tissue into metabolically active tissue

Supporting Data / Figures

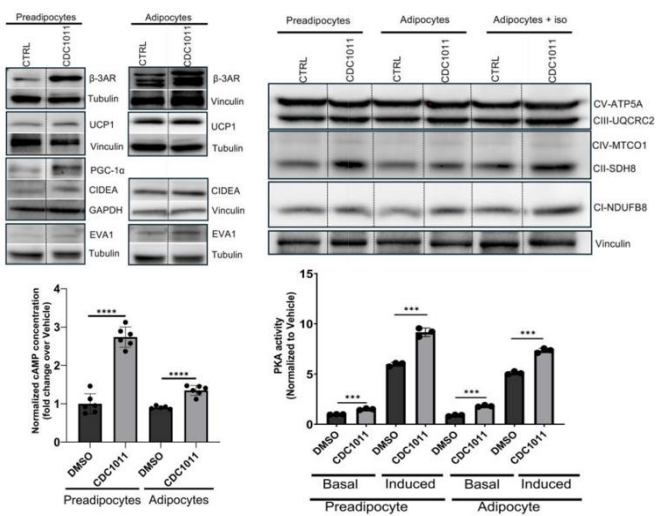

Figure 1: CDC1011 induces the browning of white adipocytes **Top Left:** Effect of CDC1011 treatment on browning markers **Top Right:** Effect of CDC1011 treatment on mitochondrial electron transport chain subunit protein expression **Bottom:** Effect of CDC1011 on basal cAMP levels and PKA activity, indicative of lipolysis.

Inventors:
Lotfi Chouchane
Jingxuan Shan

Patents:
Provisional Filed

Publications:
Rammah et al. *J Trans Med.* 2026.

Biz Dev Contact:
Mina Zion
(646) 814-4907
mwz4001@qatar-med.cornell.edu

Cornell Reference:
D-11463

Weill Cornell Medicine

Weill Cornell Medicine