

Resource Summary Report

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University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core Facility

RRID:SCR_022427

Type: Tool

Proper Citation

University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core Facility (RRID:SCR_022427)

Resource Information

URL: <https://www.med.upenn.edu/edom/drc/cores/rodent.html>

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Description: Core (RMPC, formerly MPPMC) is metabolic core. Offers technology and phenotyping services to allow investigators to study metabolism in pre-clinical rodent models. Services include measurements of body composition (fat and lean mass) using NMR and DEXA, energy balance (food intake, locomotor activity, energy expenditure) using indirect calorimetry in either Columbus Instruments CLAMS system or Sable Systems Promethion System (with optional gas analyzer), and other in vivo metabolic phenotyping services (glucose clamps, insulin and glucose tolerance tests, telemetric monitoring of blood glucose or blood pressure, bomb calorimetry).

Abbreviations: RMPC

Synonyms: Rodent Metabolic Phenotyping Core (RMPC), University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core (RMPC)

Resource Type: core facility, service resource, access service resource

Keywords: USEDit, ABRF

Resource Name: University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core Facility

Resource ID: SCR_022427

Alternate IDs: ARBF_1433

Alternate URLs: <https://coremarketplace.org?citation=1&FacilityID=1433>

Record Creation Time: 20220602T050140+0000

Record Last Update: 20260731T043109+0000

Ratings and Alerts

No rating or validation information has been found for University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core Facility.

No alerts have been found for University of Pennsylvania Perelman School of Medicine Rodent Metabolic Phenotyping Core Facility.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 12 mentions in open access literature.

Listed below are recent publications. The full list is available at [kravitz2](#) .

Fein EC, et al. (2026) The innate thermogenic capacity of brown adipose tissue develops independently of sympathetic signaling. *Molecular metabolism*, 103, 102299.

Voos KM, et al. (2025) Ankyrin-B modulates mitochondrial fission in skeletal muscle and is required for optimal endurance exercise capacity. *Nature communications*, 16(1), 7671.

Lopez Valencia M, et al. (2025) Sleep in a mouse model of fragile X syndrome is resistant to metabolic manipulations. *Human molecular genetics*, 34(22), 1874.

Langer HT, et al. (2025) Pharmacological weight loss with incretin-based therapies does not result in a disproportionate loss of muscle mass or function in obese mice and humans. *medRxiv : the preprint server for health sciences*.

Hu MY, et al. (2025) Human-length Telomeres Limit Regeneration of Liver Epithelial Cells in Mice. *Cellular and molecular gastroenterology and hepatology*, 101655.

Cherkaoui S, et al. (2025) Reprogramming neuroblastoma by diet-enhanced polyamine depletion. *Nature*, 646(8085), 707.

Pruzinsky E, et al. (2025) A role for the transcriptional coregulator RIP140 in the control of muscle endurance fitness. *JCI insight*, 10(22).

Li F, et al. (2025) Cell-Specific Inducible Human APOL1 Risk Variant Expression in Mice Causes Hypertension and Renal Damage. *Circulation*.

Geisler CE, et al. (2025) Hindbrain octadecaneuropeptide gliotransmission as a therapeutic target for energy balance control without nausea or emesis. *Science translational medicine*, 17(808), eadu6764.

Song F, et al. (2024) Wnt7b overexpression in osteoblasts stimulates bone formation and reduces obesity in mice on a high-fat diet. *JBMR plus*, 8(11), ziae122.

Nunn E, et al. (2024) Antibody blockade of activin type II receptors preserves skeletal muscle mass and enhances fat loss during GLP-1 receptor agonism. *Molecular metabolism*, 80, 101880.

Doan KV, et al. (2024) Cardiac NAD⁺ depletion in mice promotes hypertrophic cardiomyopathy and arrhythmias prior to impaired bioenergetics. *Nature cardiovascular research*, 3(10), 1236.