

Resource Summary Report

Generated by [RRID](#) on Aug 28, 2026

pAd-Track Flag HA PGC-1 alpha

RRID:Addgene_14426

Type: Plasmid

Proper Citation

RRID:Addgene_14426

Plasmid Information

URL: <http://www.addgene.org/14426>

Proper Citation: RRID:Addgene_14426

Insert Name: PGC-1 alpha

Organism: Mus musculus

Bacterial Resistance: Kanamycin

Defining Citation: [PMID:16753578](#)

Vector Backbone Description: Backbone Size:9000; Vector Backbone:pAdTrack-CMV; Vector Types:Mammalian Expression, Adenoviral; Bacterial Resistance:Kanamycin

Comments: pAdTrack can be recombined with pAdEasy to make adenoviral-PGC1. See He et al. <http://www.pnas.org/cgi/content/full/95/5/2509> for protocols.

Plasmid Name: pAd-Track Flag HA PGC-1 alpha

Relevant Mutation: Last four amino acids of PGC-1a removed to make the protein more stable.

Record Creation Time: 20220422T221817+0000

Record Last Update: 20260815T080652+0000

Ratings and Alerts

No rating or validation information has been found for pAd-Track Flag HA PGC-1 alpha.

No alerts have been found for pAd-Track Flag HA PGC-1 alpha.

Data and Source Information

Source: [Addgene](#)

Usage and Citation Metrics

We found 6 mentions in open access literature.

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

Galipeau M, et al. (2024) Commercially available PGC-1 β antibodies vary greatly in specificity and sensitivity. *microPublication biology*, 2024.

Maniyadath B, et al. (2019) Loss of Hepatic Oscillatory Fed microRNAs Abrogates Refed Transition and Causes Liver Dysfunctions. *Cell reports*, 26(8), 2212.

Yu B, et al. (2018) PGC-1 β Controls Skeletal Stem Cell Fate and Bone-Fat Balance in Osteoporosis and Skeletal Aging by Inducing TAZ. *Cell stem cell*, 23(2), 193.

Koh JH, et al. (2017) PPAR γ Is Essential for Maintaining Normal Levels of PGC-1 β and Mitochondria and for the Increase in Muscle Mitochondria Induced by Exercise. *Cell metabolism*, 25(5), 1176.

Peeters A, et al. (2011) Carbohydrate metabolism is perturbed in peroxisome-deficient hepatocytes due to mitochondrial dysfunction, AMP-activated protein kinase (AMPK) activation, and peroxisome proliferator-activated receptor γ coactivator 1 β (PGC-1 β) suppression. *The Journal of biological chemistry*, 286(49), 42162.

Diani-Moore S, et al. (2010) Identification of the aryl hydrocarbon receptor target gene TiPARP as a mediator of suppression of hepatic gluconeogenesis by 2,3,7,8-tetrachlorodibenzo-p-dioxin and of nicotinamide as a corrective agent for this effect. *The Journal of biological chemistry*, 285(50), 38801.