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PHOSPHOLIPASE C-RELATED CATALYTICALLY INACTIVE PROTEIN REGULATES FAT METABOLISM AND ENERGY EXPENDITURE

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Obesity is characterized by excessive body fat accumulation stored as triacylglycerol (TAG) in adipose lipid droplets, and the breakdown of stored TAG is stimulated by food deprivation (fasting) or stress. Sympathetic nerve activation enhances lipolysis in adipocytes, a process of which is regulated by phosphorylation of hormone sensitive lipase (HSL) and perilipin. We have elucidated that phospholipase C-related catalytically inactive protein (*PRIP*), a binding partner of protein phosphatase 1 (PP1) and 2A (PP2A), modulates the lipolysis process in adipose tissues and regulates adiposity and thermogenesis. *PRIP* was originally identified as an inositol 1, 4, 5-trisphosphate binding protein and a phosphatideylinositol 4, 5-bisphosphate through its pleckstrin homology domain. *PRIP* is similar to phospholipase C-δ1 but lacks enzymatic activity. *Prip*-knockout (KO) mice showed a lean phenotype. The phosphorylation levels of HSL and perilipin were greater in white adipose tissue (WAT) prepared from regular diet-fed or fasted *Prip*-KO mice than those in wild-type mice, suggesting enhanced lipolytic activity in *Prip*-KO WAT. In response to adrenaline stimulation, *PRIP* and protein phosphatases, PP1 and PP2A, translocated onto lipid droplets in *Prip*-KO adipocytes, which enhanced dephosphorylation of HSL followed by the inhibition of non-esterified fatty acid and glycerol production. The upregulation of lipolytic activity was also observed in *Prip*-KO brown adipose tissue (BAT). Furthermore, *Prip*-KO BAT displayed increased expression of uncoupling protein 1 (UCP1). Consistently, a high-fat diet (HFD)-fed *Prip*-KO mice showed increased energy expenditure, a high rectal temperature, and a lean phenotype compared with control wild-type mice. Collectively, *PRIP* is a novel molecule that regulates fat metabolism and thermogenesis.

Biography

Takashi Kanematsu received his PhD in Biochemistry from Kyushu University, Fukuoka Japan, in 1994. He then worked as a Postdoctoral Fellow at Vanderbilt University, TN USA, and, subsequently, served as Assistant and Associate Professor in the Department of Biochemistry at Kyushu University (1997–2008). From 2009, he is Professor and Chair of the Department of Cellular and Molecular Pharmacology at Hiroshima University, Hiroshima Japan. He currently serves on the Editorial Board of *Journal of Pharmacological Sciences*.

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