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➤**연구 관심분야**

Pancreatic beta-cell, Diabetes, Obesity

➤**주요 연구 실적**

1. Melatonin prevents pancreatic β-cell loss due to glucotoxicity: the relationship between oxidative stress and endoplasmic reticulum stress. Park JH, Shim HM, Na AY, Bae KC, Bae JH, Im SS, Cho HC, Song DK. J Pineal Res. 2014; 56:143-53.
2. Green tea extract with polyethylene glycol-3350 reduces body weight and improves glucose tolerance in db/db and high-fat diet mice. Park JH, Choi YJ, Kim YW, Kim SP, Cho HC, Ahn S, Bae KC, Im SS, Bae JH, Song DK. Naunyn Schmiedebergs Arch Pharmacol. 2013;386(8):733-45.
3. Protective effect of melatonin on TNF-α-induced muscle atrophy in L6 myotubes. Park JH, Chung EJ, Kwon HJ, Im SS, Lim JG, Song DK. J Pineal Res. 2013;54(4):417-25.
4. Glucagon-like peptide-1 enhances glucokinase activity in pancreatic β-cells through the association of Epac2 with Rim2 and Rab3A. Park JH, Kim SJ, Park SH, Son DG, Bae JH, Kim HK, Han J, Song DK. Endocrinology. 2012;153(2):574-82.
5. Cellular glucose availability and glucagon-like peptide-1. Park JH, Earm YE, Song DK. Prog Biophys Mol Biol. 2011;107(2):286-92.

Loss of Epac2 results in premature obesity and insulin resistance

The prototypic second messenger cyclic AMP (cAMP) is essential for controlling cellular metabolism, including glucose and lipid homeostasis. In mammals, the majorities of cAMP functions are mediated by cAMP-dependent protein kinase (PKA) and exchange proteins directly activated by cAMP (Epacs). Two isoforms of Epac, namely Epac1 and Epac2 have been identified so far. In particular, Epac1 mRNA is expressed ubiquitously, whereas Epac2 mRNA is predominantly expressed in the brain and endocrine tissues. To explore the physiological functions of Epac2, Epac2-deficient mice were analyzed. Weight gain, insulin sensitivity, metabolic rate, and liver lipid content were compared between Epac2-deficient and wild-type mice. Epac2-deficient mice exhibited greater body weight and white adipose tissue mass, had reduced energy expenditure, displayed premature onset insulin resistance. Interestingly, plasma atrial natriuretic peptide (ANP) concentrations were increased after refeeding in wild-type but not Epac2-deficient mice. It have been increasingly recognized that ANP enhances lipolysis and may have important metabolic actions. We also found significant decreases in the expressions of key lipolytic genes in the white adipose tissue from Epac2-deficient mice. These findings define a gut-heart Epac2–dependent and ANP–dependent axis that regulates adiposity and energy balance.