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AHNAK, controlling body weight and metabolism by regulating white and brown adipogenesis

In adipose tissue, agonists of the β3-adrenergic receptor (ADRB3) regulate lipolysis, lipid oxidation, and thermogenesis. The deficiency in the thermogenesis induced by neuroblast differentiation-associated protein AHNAK in white adipose tissue (WAT) of mice fed a high-fat diet suggests that AHNAK may stimulate energy expenditure via development of beige fat. Here, we report that AHNAK deficiency promoted browning and thermogenic gene expression in WAT but not in brown adipose tissue of mice stimulated with the ADRB3 agonist CL-316243. Consistent with the increased thermogenesis, Ahnak(-/-) mice exhibited an increase in energy expenditure, accompanied by elevated mitochondrial biogenesis in WAT depots in response to CL-316243. Additionally, AHNAK-deficient WAT contained more eosinophils and higher levels of type 2 cytokines (IL-4/IL-13) to promote browning of WAT in response to CL-316243. This was associated with enhanced sympathetic tone in the WAT via upregulation of adrb3 and tyrosine hydroxylase (TH) in response to β-adrenergic activation. CL-316243 activated PKA signalling and enhanced lipolysis, as evidenced by increased phosphorylation of hormone-sensitive lipase and release of free glycerol in Ahnak(-/-) mice compared to wild-type mice. Overall, these findings suggest an important role of AHNAK in the regulation of thermogenesis and lipolysis in WAT via β-adrenergic signalling.

連絡先:マウス表現型解析開発チーム
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