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Placental Growth Factor Overexpression Modulates Metabolic Pathways and Ketone Body Levels in Mice under Varying Dietary Conditions

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Objectives : Placental Growth Factor (PlGF) has emerged as a key player in metabolic regulation, impacting AMP-activated protein kinase (AMPK) and sirtuin 1 (Sirt1), crucial for energy balance and glucose metabolism. Ketone bodies, once considered primarily for energy during fasting and exercise, are gaining attention for their potential role in promoting longevity through metabolic pathways. This study investigates the influence of PlGF on AMPK, Sirt1, mammalian target of rapamycin (mTOR), and ketone levels in mice subjected to different dietary conditions.
Methods : PlGF wild-type and PlGF overexpressing (Tg) mice were subjected to a non-fat diet and a high-fat diet for four weeks. Biochemical parameters, including serum and intrarenal ketone bodies (acetoacetate, beta-hydroxybutyrate), were assessed. Key metabolic pathways, including AMPK, Sirt1, and mTOR, were examined.
Results : PlGF wild-type mice on high-fat diet exhibited renal damage and metabolic derangements compared to PlGF wild-type on non-fat diet and PlGF Tg mice on non-fat diet and high-fat diet. PlGF Tg mice on high-fat diet showed elevated expression levels of AMPK and Sirt1, while mTOR levels were reduced when compared to PlGF wild type on high-fat diet. Additionally, PlGF wild-type mice on high-fat diet displayed decreased serum and intrarenal ketone levels compared to those on non-fat diet. Intriguingly, PlGF Tg mice on high-fat diet exhibited a less pronounced decrease in serum ketone bodies.
Conclusions : PlGF overexpression appears to modulate key metabolic pathways, particularly AMPK and Sirt1, potentially attenuating the decline in serum ketone bodies induced by a high-fat diet. These findings suggest that PlGF may serve as a mediator between dietary influences and metabolic health, offering insights into strategies for preventing kidney damage associated with deranged metabolism.