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Impacts of prenatal FGF21 upregulation on the thermogenic gene expression in the white adipose tissues of male C57BL/6J mice at adult stages

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Fibroblast growth factor 21 (FGF21) is mainly produced by the liver and its expressi transcriptionally regulated by peroxisome proliferator-activated receptors α (PPARα). FGF21 is postul be a thermogenic hormone, since it has been demonstrated to involve in the browning of white ad tissue (WAT) and to activate thermogenic gene expression in the WAT and brown adipose tissue previous study shows that uterine PPARα activation caused by maternal oxidized frying oil ingestion a adipose thermogenic capacity, which contribute to resistance to diet-induced obesity (DIO) in the offs at adulthood. The aim of this study is to investigate whether the intrauterine exposure to high leve FGF21, by giving clofibrate (CF; a PPARα agonist) to pregnant mothers, influences the thermogenic expression in adipose tissue of offspring at adult stages. Pregnant C57BL/6J mice were divided into groups to receive a control or CF diet (0.5% clofibrate) for gestational period. After delivery, all pups wi lactated by control dams, weaned on chow diet, and exposed to a high fat diet at 8-12 wk of age. Re show PPARα activation occurred in CF-fed dams and their fetuses. Moreover, the expression of *Fgf21* upregulated in CF-fed fetuses. The male, but not the female, adult offspring of the CF-fed dams protected from DIO. The protein level of UCP-1 was significantly increased in the WAT of male offsp came from the CF-fed dams and this might contribute to the anti-obesity effect. We conclude that ute PPAR-α activation caused by maternal clofibrate ingestation leads to greater production of FGF21 in f liver, which might increase the browning capacity of WAT in males during adulthood.

Keywords: Clofibrate, Fibroblast growth factor 21, Thermogenesis, Adipose tissues, Prenatal period

PA-16

Inhibition of breast cancer cell Invasion by docosahexaenoic acidmediated by altering distribution of integrin α6β4 in lipid rafts

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Integrin α6β4 belongs to a laminin receptor and its primary function was to maintain epithelial integrity. Several studies suggested that integrin α6β4 plays a pivotal role in carcinoma progression. In advanced breast carcinomas, the integrin α6β4 is positively associated with a migratory and invasive phenotype. Previous studies showed that n-3 polyunsaturated fatty acids - docosahexaenoic acid (DHA)exhibited an anti-cancer effect in various human carcinoma cells, but the effect of DHA on metastasis of breast cancer cells is not fully clarified. We studied the anti-metastasis potential of DHA in Hs578T breast cancer cells. We found that DHA significantly inhibited cell migration, invasion and dramatically down-regulated integrin α6β4 expression in a dose-dependent manner. Furthermore, DHA suppressed the Src, AKT expression and phosphorylation as well as their downstream pathways by altering the integrin α6β4 distribution in lipid rafts. These results suggest that DHA inhibits α6β4-mediated cell migration and invasion at least in part via altering the distribution of integrin α6β4 and integrin α6β4-mediated Src and Akt signaling pathways.

Keywords: Breast cancer cell, Integrin α6β4, lipid raft, S100A4, DHA