In utero exposure to Hypoxis hemerocallidea (African potato) improves glucose tolerance, lipid profiles and antioxidant status in offspring 28 days postpartum

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Intrauterine and early life environments contribute to adult metabolic phenotype. Use of medicinal plants like Hypoxis hemerocallidea during pregnancy is common. This raises the question of whether phytochemicals in medicinal plants may contribute to metabolic intrauterine-programming effects on the offspring that may determine adult metabolic phenotype. Three gestational treatment groups (n=6 rats per group): Control group (distilled water) and two H. hemerocallidea treated groups (150 and 300 mg/kg body weight) were used for the study. Pups were weighed at birth and weekly until 28 days postpartum. OGTT was performed and area under the curve determined. Fasted pups (n=8/group) were terminated and serum collected for lipid profiles and antioxidant status. Liver and kidneys were homogenized for determination of total antioxidant capacity, lipid peroxidation and SOD. Pup weights were similar at birth for all treatment groups but after 21 days, H. hemerocallidea exposed pups had higher body weights (P<0.05) compared to controls. H. hemerocallidea exposed rats had better glucose tolerance and kidney antioxidant status (P<0.05) compared to controls with no effect on liver. H. hemerocallidea exposure resulted in decreased total cholesterol and LDL concentration with no effect on HDL. H. hemerocallidea may have protective programming effects to development of diabetes and oxidative stress in pups exposed in utero. Effects after exposure to obesogenic diet will be investigated.

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FoxO1 redistribution from mitochondria to nucleus drives browning of white adipose tissue upon nutrient stress

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Adipocytes readapt their metabolism to external stimuli restraining stress conditions. Mitochondria represent the central core of these responses. Here we show that nutrient starvation in white and beige adipocytes causes generation of mitochondrial ROS (mtROS), mitonuclear protein imbalance as well as the induction of brown-related genes. A newly identified mitochondrial phosphorylated form of FoxO1 (mtFoxO1) drives this metabolic adaptation via an mtROS-dependent fashion. mtROS induces mitochondrial phosphatase PTPMT1, leading to mtFoxO1 de-phosphorylation and nuclear accumulation. In nuclei mitochondrial-derived FoxO1 specifically induces the expression of SOD2, UCP1 and other marks of browning including mitochondrial fission. By forcing FoxO1 into mitochondria or down-regulating UCP1, we observed an enhanced mitochondrial stress, implying that adipocyte browning is an adaptive response to nutrient starvation. Collectively, our results highlight that mito-nuclear shuttling of FoxO1 has a central role in the browning program and suggest the manipulation of its mitochondrial distribution as an attractive strategy to improve the metabolic function of adipose tissue.

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