Offspring's cardiac nitric oxide signaling is influenced by maternal physical exercise during an obesogenic pregnancy

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Maternal obesity(MO) affects 50% of pregnancies and creates an adverse intrauterine environment, predisposing the offspring to cardiovascular disease(CVD). Nitric oxide(NO) regulates ATP production and is involved in nitrosative stress. Impaired NO signaling is observed in MO fetoplacental unit and CVD patients' endothelia. However, the effects of MO and maternal physical exercise during MO(MOEx) in offspring's cardiac NO signaling are unknown.

Female Sprague-Dawley were fed a high-fat-high-sugar(HFHS;n=12) or control(C;n=6) diet, 7 weeks before pregnancy. Six HFHS mothers were sedentary(MO;n=6), other six exercised(MOEx;n=6). Offspring were kept on a control diet without exercise. Male and female F1(F1-C;F1-MO;F1-MOEx) were euthanized at 32-weeks-old(n=6/sex). Unpaired t-test or Mann-Whitney test were used for statistical analysis($p \leq 0.05$).

The atherogenic index (CVD risk), was increased in females F1-MO vs F1-C($p < 0.01$), decreased in females F1-MOEx vs F1-MO($p < 0.01$), and in males F1-MOEx vs F1-C($p=0.01$). Cardiac CAT-1 (NO precursor transporter) increased 23% in males F1-MO vs F1-C. Activated Akt(p-AktThr308/Akt1) was increased in females F1-MO vs F1-C($p=0.02$) and decreased in males F1-MOEx vs F1-C($p=0.03$). Mitochondrial ATP levels were increased in males($p < 0.01$) and females($p=0.04$) F1-MOEx vs F1-C. Nitrotyrosine (nitrosative stress marker) was decreased in males F1-MOEx vs F1-C($p < 0.01$). Catalase was decreased in males F1-MOEx vs F1-MO($p=0.03$). SOD2 was increased in males F1-MOEx vs F1-C($p=0.02$).

Maternal obesity increased offspring's CVD risk and possibly L-Arginine transport. Exercise during MO influenced offspring's cardiac ATP levels, nitrosative damage, and antioxidant defenses in a sex-specific way, likely reducing CVD risk in MO offspring.

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