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Monocyte reprogramming in nonhuman primate mother-child dyads based on maternal health and obesity status

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The developmental origins of metabolic disease mechanisms are mostly unknown. Up to 40% of obese women are healthy, while roughly 15% of lean women are not. The implications for their children are difficult to disentangle from environmental factors. We assessed offspring from age-matched female African green monkeys (AMGs [n=44]) classified as metabolically healthy lean (MHL), healthy obese (MHO), unhealthy lean (MUL), and unhealthy obese (MUO) based on adjusted metabolic syndrome criteria (waist >40cm, fasting glucose (FG) >100 mg/dL, SBP >135/DBP >85mmHg and HDL-c <50mg/dL). Pre-pubertal juveniles (n=9-11/group) had weight, FG and blood pressures measured. Flow cytometry identified circulating classical, intermediate, and non-classical monocytes in mother-offspring pairs and offspring monocyte chemoattractant protein 1 (MCP1) was measured. Offspring did not differ by weight or FBG but SBP trended higher in two groups (MUL and MHO, p=0.06). MUO mothers had more non-classical monocytes compared to MHL (p=0.006), and both MUL and MUO offspring also had higher non-classical monocytes compared to MHL (p=0.05 and p=0.07). MCP1 was higher in MUO offspring (p=0.02). Maternal health and obesity influence offspring immune profiles and metabolic risk factors prior to obesity. Shifting maternal immune cells prior to pregnancy may mitigate suboptimal developmental programming.

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