Selenium supplementation during puberty and young adulthood mitigates obesity-induced metabolic, cellular and epigenetic alterations in rat male physiology

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Original Version

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Selenium (Se) role in obesity is not clear. In addition, information on Se’s role in male physiology, specifically in obesity, is scarce. We conducted this study to evaluate the efficacy of Se supplementation, specifically during puberty until young adulthood, against obesity-induced deregulation of metabolic, cellular, and epigenetic parameters in epididymal fat and/or sperm cells in a rat model. High-fat-diet consumption by male rats during puberty and young adulthood significantly increased body weight, adipocyte size, oxidative stress, deregulated expression of genes associated with inflammation (Adiponectin, IL-6, TNF-α), adipogenesis (CEBPα), estrogen biosynthesis (CYP19) and epigenetic processes in epididymal adipose tissue (Dnmt3a), as well as altered microRNA expression vital for spermatogenesis in sperm cells (miR-15b and miR-497). On the other hand, Se supplementation significantly decreased oxidative stress and mitigated these molecular/epigenetic alterations in epididymal adipose tissue or sperm cells. Our results indicate that selenium supplementation during puberty/young adulthood could improve male physiology in the context of obesity. In addition, it suggests that Se could potentially positively affect offspring health.

Keywords: Selenium, Obesity, Male reproductive physiology, Epigenetics reprogramming