

Abstract

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Moderate zinc deficiency reduces testicular zip6 and zip10 abundance and impairs spermatogenesis in mice.

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OBJECTIVE: Male infertility accounts for ~40% of cases of failure to conceive. Testes have a strict zinc (Zn) requirement and severe Zn deficiency compromises spermatogenesis, sperm viability, and motility, compromising fertility in men. Despite the high prevalence of marginal Zn deficiency in humans, less emphasis has been placed on understanding the consequences on male reproduction.

METHODS AND RESULTS: Swiss Webster mice were used to visualize Zip protein expression during spermatogenesis using immunohistochemistry. Data suggest Zip5 imports Zn into Sertoli cells and spermatocytes, augmented by Zip10 (primary spermatocytes) and Zip8 (secondary spermatocytes). Zip6, 8, and 10 expression was retained in round spermatids, although Zip8 and Zip10 expression disappears during spermatid maturation. Zip1 and Zip6 expression was detected in mature, elongated spermatids. Zip14 was detected in undifferentiated spermatogonia and Leydig cells. Mice fed diets (n = 10/group) reduced in Zn concentration [marginal-Zn diet (MZD), 10 mg Zn/kg; low-Zn diet (ZD), 7 mg Zn/kg] for 30 d had >35% lower liver Zn concentrations than mice fed the control diet (C; 30 mg Zn/kg) (P < 0.05). Plasma Zn and testosterone concentrations and the testes Zn concentration and weight were not significantly lower than in controls. Plasma Zn was greater in the ZD group than in the C and MZD groups. Mice fed ZD had a reduced number of terminal deoxynucleotidyl transferase dUTP nick end labeling-positive cells (~50%; P < 0.05), compromised seminiferous tubule structure, and reduced Zip10 and Zip6 abundance (>50%; P < 0.5) compared with mice fed C.

CONCLUSIONS: Our data provide compelling evidence that reduced Zn intake may be associated with infertility in men, perhaps independent of decreased levels of circulating Zn or testosterone, which warrants further investigation in human populations.

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