Polyamine metabolism links the gut microbiota and testicular dysfunction

Qi Zhao
Jian-Feng Huang
Yan Cheng
Man-Yun Dai
Wei-Feng Zhu
Xiu-Wei Yang
Frank J. Gonzalez
Fei Li

Video Byte

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Abstract

Male infertility due to toxin exposure is a serious health issue worldwide. However, the metabolic link between toxin exposure and testicular dysfunction is unclear. To learn more, a recent study examined the metabolic disruption associated with triptolide (TP)-induced testicular injury in mice. TP caused spermine (SP) deficiency by disrupting polyamine biosynthesis and uptake in the testes and by perturbing the gut microbiota. Exogenous SP supplementation reversed the TP-induced testicular dysfunction by increasing the expression of early and late spermatogenesis-related genes and by attenuating TP-induced decreases in offspring numbers. SP's protective effect was found to be largely dependent on upregulation of HSP70 proteins in vivo and in vitro. In addition, antibiotic-mediated gut microbiota depletion reduced intestinal SP levels and exacerbated testicular injury, but transplantation with gut microbes from untreated mice and supplementation with the polyamine-producing bacterium *Parabacteroides distasonis* reversed the testicular dysfunction. Although additional research is needed, the results outline the interactions among toxin exposure, the gut microbiota, and testicular injury and support targeting of gut microbiota diversity and dynamics as a potential strategy to prevent male reproductive disorders.