Abstract

A single in utero exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on gestation day 15 decreased epididymal sperm count in adult rats and thus was used to establish a tolerable daily intake for TCDD. However, several laboratories have been unable to replicate these findings. Moreover, conflicting reports of TCDD effects on daily sperm production suggest that spermatogenesis may not be as sensitive to the adverse effects of TCDD as previously thought. We performed a PubMed search using relevant search terms linking dioxin exposure with adverse effects on reproduction and spermatogenesis. Developmental exposure to TCDD is consistently linked with decreased cauda epididymal sperm counts in animal studies, although at higher dose levels than those used in some earlier studies. However, the evidence linking in utero TCDD exposure and spermatogenesis is not convincing. Animal studies provide clear evidence of an adverse effect of in utero TCDD exposure on epididymal sperm count but do not support the conclusion that spermatogenesis is adversely affected. The mechanisms underlying decreased epididymal sperm count are unknown; however, we postulate that epididymal function is the key target for the adverse effects of TCDD.

Keywords

Development, Dioxin, Endocrine, Reproduction, Spermatogenesis.