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Authors: Debarshi Sarkar, Shio Kumar Singh

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Inhibition of testicular steroidogenesis and impaired differentiation of Sertoli cells in peripubertal mice offspring following maternal exposure to BDE-209 during lactation suppress germ cell proliferation

Debarshi Sarkar, Shio Kumar Singh*

Department of Zoology, Institute of Science, Banaras Hindu University, Varanasi-221005, India

*Correspondence: Prof. Shio Kumar Singh
Department of Zoology
Institute of Science
Banaras Hindu University
Varanasi-221005, India
Tel.: +91 542 6702532
Fax: +91 542 2368174
E-mail: shioks@rediffmail.com/shioks53@gmail.com

Highlights

- Lactational exposure to BDE-209 affects testis histopathology in mice offspring.
- Offspring of BDE-209-exposed dams show decreased testicular steroidogenesis.
- Offspring of BDE-209-treated dams exhibit impairment in germ cell proliferation.
- Differentiation of Sertoli cells is perturbed in offspring of treated dams.

Abstract

Polybrominated diphenyl ethers (PBDEs) are used for fire prevention purpose. BDE-209, a congener of PBDEs, is thyroid hormones (THs)-disrupting chemical because of its structural similarity with THs. Testis is considered as THs-responsive organ and is more susceptible to chemical agents during peripubertal period. This study, therefore, evaluated the effect and possible mechanism(s) of action of maternal exposure to BDE-209 during lactation on germ cell proliferation, testicular steroidogenesis and on differentiation of Sertoli cells (SCs) in relation to altered THs status in peripubertal mice offspring. Lactating Parkes mice were gavaged with 500 and 700 mg/kg BW of BDE-209 in corn oil from postnatal day (PND) 1 to PND 28 along with 6-propyl-2-thiouracil (PTU)-treated positive controls and vehicle-treated controls. Male pups of lactating dams were sacrificed at PND 42. Maternal exposure to BDE-209 during lactation markedly affected testicular histopathology, germ cell proliferation and steroidogenesis with down-regulated expression of various steroidogenic markers in peripubertal mice offspring. Decreased expressions of maturational markers of SCs with a decline in serum THs levels were