Experimental maternal treatment with dexamethasone during lactation induces neonatal testicular and epididymal oxidative stress; Implications for early postnatal exposure

Author(s): SO Jeje, FK Ola-Mudathir, Y Raji

Abstract

Maternal treatment with dexamethasone during lactation alters reproductive functions and increases serum corticosterone in the male offspring. Excess corticosterone may induce oxidative stress. This study was designed to evaluate the effects of maternal treatment with dexamethasone during lactation on oxidative stress indices in the testis and epididymis of a male offspring.
Twenty lactating dams were divided into 4 groups (n = 5). Group 1 was administered 0.02 ml/100 g/day normal saline subcutaneously at lactation days 1–21. Groups 2, 3, and 4 were administered 100 μg/kg/day dexamethasone (Dex) subcutaneously at lactation days (LD) 1–7, 1–14, and 1–21 respectively. Testis and epididymis malondialdehyde (MDA), catalase and superoxide dismutase (SOD) activities were measured as markers of oxidative stress.

The mean testis and epididymis MDA were significantly raised ($p < 0.05$) in the dexamethasone-treated groups when compared with control. This was accompanied with a significant reduction ($p < 0.05$) in SOD and catalase activities in these tissues in the DexLD 1–21, when compared with control. The mean total protein level of the epididymis was significantly reduced ($p < 0.05$) in all the dexamethasone treated groups when compared with control.

In conclusion, maternal treatment with dexamethasone during the first two weeks of lactation and throughout lactation may lead to increase in oxidative stress in the testis and epididymis of the male offspring of Wistar rats.

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Keywords: Dexamethasone, Lactation, Antioxidant enzymes, Offspring

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