of EB treatment was observed. All together, our results suggest that sustained increase of estrogen levels by EB implantation to skin was impairs spermatogenesis with an increase in germ cell apoptosis that appears to be mediated through modulation of Fas and Fas-L system, in which ER may not play a significant role.

Key words : β-Estradiol 3-benzoate, Testis, Apoptosis, Fas, Fas ligand, Estrogen receptor α

P#27

Involvement of the Fas and Fas Ligand in Testicular Germ Cell Apoptosis by Zearalenone in Rat

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Zearalenone (ZEA), a nonsteroidal estrogenic mycotoxin, is known to cause toxicity in the testis. In the present study, we examined the effects of ZEA on spermatogenesis and possible mechanisms involved in germ cell injuries by ZEA in rat. Ten-Week-old Sprague-Dawley rats were treated with 5㎎/㎏ of ZEA i.p and euthanized 3, 6, 12, 24 or 48hr after treatment. Histopathologically, selective damages of the spermatogonia and spermatocytes were observed. They were TUNEL-positive and found primarily in spermatogenic stages I-VI tubules in 6 hr after treatment and increased gradually until 12 hr, and then gradually decreased. Western blot analysis revealed an increase in Fas and Fas lignad (Fas-L) protein levels in the testis of ZEA-treated rats. The estrogen receptor (ERα) expression levels were not changed. These results suggest that: 1) the effect of ZEA on spermatogenesis is related to activation of apoptosis in specific germ cells; 2) germ cells in early spermatogenic stages (I-IV) are more sensitive to ZEA; 3) the induction of germ cell apoptosis by ZEA is mediated through modulation of Fas and Fas-L system; and 4) ERmay not play a significant role in the impairment of spermatogenesis by ZEA.

Key words : Zearalenone, Testis, Apoptosis, Fas, Fas ligand, Estrogen receptor α

P#28

Pulmonary Acariasis Caused by Sternostoma Tracheacolum in Caged Canary

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