10mg/kg body wt was administered to male and female iNOS-/- or iNOS wt C57BL/6J mice once a week for six weeks. The mice were sacrificed and examined with the incidence and multiplicities of colorectal polyps at the age of 30 weeks. The incidence of colorectal tumors were significantly reduced in iNOS gene knockout mice (22 9%). compared to that of control mice (59 1%). The multiplicity in colorectal polyps in iNOS knockout mice were 0 370.77 (n=35). being significantly smaller than the value of wild type mice (1.021.15. n=44). The sizes of the polyps in the iNOS gene knockout mice were also decreased Therefore, overproduced NO by iNOS plays an important role in mice colorectal carcinogenesis

Key words · azoxymethane, colorectal cancer, iNOS gene knockout mice. polyps

P#26

β-Estradiol 3-Benzoate Induces Rat Spermatogenic Germ Cells Apoptosis

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Estrogens play critical roles in spermatogenesis. We investigated the effects of sustained delivery of β-estradiol 3-benzoate (EB) on spermatogenesis and mechanisms involved in germ cell injuries with an emphasis on the germ cell apoptosis. Ten-week-old Sprague-Dawley rats were implanted subcutaneously with a pellet containing of 0.5 mg β-estradiol 3-benzoate (EB) fused to cholesterol and were sacrificed in 12 hr, 24 hr, 48 hr, 72 hr, 1 week, 2 weeks, 4 weeks and 6 weeks of EB implantation. We found that body weights and weights of testis and epididymis were significantly decreased in 2 weeks of EB treatment. Degeneration of germ cells was first found in pachytene spermatocytes in spermatogenic stages VII-VIII in 48 hr of EB treatment and progressively increased in a time-dependent manner. Severe degeneration and depletion of germ cells in seminiferous tubules was observed in 2 weeks of EB treatment. After 4 weeks, massive degeneration of the seminiferous epithelium exhibiting characteristics of epithelial structural disorganization, the formation of multinucleated giant cells, and decrease of interstitial cell numbers were noted. Severe atrophy and necrosis of seminiferous tubules was observed in 6 weeks of EB treatment. Apoptosis of germ cells was observed in pachytene spermatocytes in their developmental stages VII-VIII in48 hr. Mean incidence of apoptotic germ cells after EB treatment progressively increased until 2 weeks. Western blot analysis revealed an increase in Fas and Fas lignad (Fas-L) protein levels in the testis of EB-treated rats. While estrogen receptor α(ER α) expression was not changed until 2 weeks, significant decrease of ER in 4 and 6 weeks

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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