

post-induction plasma zinc concentrations for rabbits in the control group from its mean pre-induction level and the similar significant reduction in mean post-induction zinc level in model 2 showed that acetic acid induced colitis model is associated with zinc decrement, and it simulates the results obtained in human IBD <sup>(24)</sup>.

In the present study, zinc sulfate therapy markedly corrected the plasma zinc values in both models; this result showed that zinc sulfate (in a daily dose of 50 mg/kg taken orally for 3 successive days) was sufficiently absorbed and did not cause toxicity. Zinc homeostatic mechanisms probably could not cope with the well known redistribution of plasma zinc induced initially by inflammatory processes without an external zinc supplementation <sup>(25)</sup>.

Plasma copper measurement was used to identify whether such a high dose of zinc sulfate therapy 50 mg/kg/day caused secondary copper deficiency; the latter causes copper deficiency associated anemia in human <sup>(26)</sup>.

In model 1, mean plasma copper concentration of the control group was significantly increased post-induction from that of the pre-induction level,  $P < 0.05$ . While for the zinc sulfate group there is an insignificant increase in plasma copper caused by zinc sulfate therapy.

In model 2, the similar increment in mean post-induction plasma copper from the pre-induction level is expected because of the associated zinc deficiency, but this increment was statistically not significant which may be because of the less severe zinc decrement in comparison to that in model 1.

The significant increment in mean post-induction plasma copper

from mean pre-induction level is unexplainable yet.

In the present study, oral zinc sulfate (50 mg/kg/day) appeared to have a considerable prophylactic activity that was comparable to that of oral prednisolone (2 mg/kg/day) against acetic acid –induced colitis in rabbits.

Duggan, et al., <sup>(27)</sup> emphasized that zinc deficiency may predispose the intestinal tract to damage by free radicals and increased NO activity. Furthermore, Mulder, et al., <sup>(28)</sup> documented the decrease in intestinal copper/zinc containing proteins that have antioxidant function in inflammatory bowel disease.

Di-Leo, et al., <sup>(29)</sup> showed that administration of zinc sulfate in a dose of 30 mg/kg/day had a little effect on the short-term course of experimental colitis; this probably in contradict to the encouraged results obtained in the present study. This probably due to difference in the applied dose of zinc sulfate in the present study (50 mg/kg/day) which was administered 2 days prior and further 1 day post induction of colitis. On the other hand, Luk, et al., <sup>(30)</sup> demonstrated the protective effect of zinc sulfate on dinitrobenzene-induced colitis when administered rectally; this could point to the possibility of a potential cumulative effect of zinc sulfate given in a sufficient dose when used concomitantly via both oral and rectal routes of administration.

### **References**

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