SUMMARY AND CONCLUSIONS

The results of the present study illustrate that uptake of ^{109}Cd by the kidney and its clearance from the blood when given together with Zn, Se and ME is not influenced significantly but followed the same pattern of uptake as that of (^{109}Cd + Cd) alone. Increase uptake of ^{109}Cd in kidney in presence of ME, suggests that rate of accumulation is increased by ME.

In the acute cadmium study when intraarterial injection of 0.4 mg Cd/kg and 0.8 mg Cd/kg was given, the depressor and the pressure response is observed, where as in intraperitonial injection of 2 mg Cd/kg produces hypertension in about 72 per cent of the female wistar rats, indicating selective susceptibility to induction of hypertension due to genetic and some other factors which influence this physiological variation. This increase in renal cadmium level when given alongwith thiol compound like ME as invitro complex, does not increase the percertage hypertension indicating that when cadmium is in bound form, it is unable to induce hypertension where as zinc and selenium given alongwith cadmium as a single injection led to normalization of hypertension in most of the animals. The above findings are substantiated by minimal degree of biochemical and renovascular changes in the kidney.

At low level of cadmium exposure (2 mg/kg) $Na^{+}K^{+}$ATPase and carbonic anhydrase do not show any significant alteration and may not have direct role in maintaining the high blood pressure after exposure. It is also evidenced from this study that cadmium does not interfere with the renin-angiotensin system in the kidney significantly. However, when given alongwith other hypertensinogenic stimuli then its effects are aggrevated. The data on the influence of cadmium on induction of hypertension by feeding salt in diet or in drinking water shows that each increment of salt induces progressive increase in blood pressure but when given alongwith cadmium, the hypertensinogenic effect of salt is aggrevated. The synergetic effect of salt and cadmium on renin-angiotensin system and tissue electrolytes shows that primary target of action of both these stimuli is kidney and small amount of cadmium is required as compared to salt to produce this physiological variations. The altered renovascular changes and plasma angiotensin levels in this study confirm the above hypothesis. It also suggests that cadmium induced hypertensive rats have increased appetite for salt which may be responsible in maintaining elevated blood pressure.

The effect of diet on the induction of hypertension by cadmium suggests that high protein diet given to rats reduces the incidence of hypertension where as high

~~high~~ carbohydrate diet containing high sucrose precipitates the onset of hypertension. The cadmium treated animals . show that cadmium can significantly induce alterations in renin-angeotensin system, tissue electrolytes and organ weight in the rats fed excessive sucrose and produces the synergetic effect similar to salt and cadmium.

In conclusion these observations show that if these experimental models have bearing on the etiology of essential hypertension in man, these studies suggest that even in current environmental cadmium pollutions, it is not of immediate concern to all individuals, it may be a genuine health hazard to salt 'sensitive' individual, with a family history of hypertension and taking less protein and high carbohydrate diet.