CHAPTER 34

MEDICAL SCIENCES PHYSIOLOGY

Doctoral Theses

296. SAINI (Navneet)

Pathways of Apoptosis in Normal Oral Mucosa and its Derangement in Premalignant and Malignant Lesion of Oro-Pharyngeal Region.

Supervisors : Dr. Rashmi Babbar and Dr. A. K. Mandal Th 15590

Abstract

Indicates increase in apoptosis caspases (Caspase-3,8,9) and bax in premalignant and malignant lesions of oro-pharyngeal region as compared to normal epithlium. The pathway of apoptosis in all these lesions was studied by observing the association between the caspases and bax activating these pathways. A statistically significant association between them indicates that both pathways are involved. There was variable increase in k-ras positively in premalignant and malignant lesions as compared to controls, however its association with apoptosis was statistically not significant, thus indicating that k-ras and apoptosis were not interrelated. The serum total cholestrol, LDL, VLDL and serum triglyceride levels were decreased in premalignant and malignant lesions as compared to the controls. It may be due to the utilization of serum lipids by malignant and premalignant cells. However the increase in HDL in our study may be due to the altered functioning in lipoprotein lipase. Could not find any association between serum lipid levels, apoptosis, caspase-3,8,9 and bax. Thus imbalance between cell growth (mediated by k-ras) and cell death (apoptosis) mediated by caspases and bax, plays an important role in transformation of normal epithelium to hyperplastic, dysplastic and neoplastic epithelium.

Contents

1. Introduction. 2. Review of literature. 3. Aims and objectives.

4. Material and methods. 5. Results. 6. Disscussion. 7. Summary. 8. Conclusion. 9. Bibliography.

297. SWATI OMANWAR **Functional Changes in Vascular Responsiveness Following Mercury Exposure in Rats.** Supervisors : Prof. K. Ravi and Prof. M. Fahim

Th 15591

Abstract

Investigates whether mercury, a redox inactive metal having high affinity for Na^+/K^+ATP ase, causes endothelial dysfunction by involving nitric oxide, K^+ATP channel pathways which may contribute to mercury-induced vascular disorders. For this purpose, vascular smooth muscle responses to various agonists and antagonists were examined in an isolated organ bath set up (aortic rings-endothelium-intact and endothelium-denuded). Two different forms of mercury, mercuric chloride (inorganic form) and methyl mercury chloride (environmental forms) were chosen and their effects when given acutely and chronically on vascular responsiveness were studied.

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