SUMMARY AND CONCLUSIONS

Oxidant stress is known to be induced by variety of stress conditions including physical exercise and impaired antioxidant defenses. Oxidant stress induced by free radicals and lipid peroxides can lead to enzymatic as well as non-enzymatic lipid peroxidation. Among the cellular defenses, vit E and Se are important in combating enzymatic as well as non-enzymatic lipid peroxidations. Hence in the present study oxidant stress is induced by either exhaustive physical exercise or vit E and/or Se deficiency and pulmonary antioxidant defenses were analyzed. The salient features of the present study are:

1). Dietary supplementation of vit E and/or Se for a period of 12 weeks starting from weanling stage significantly accelerated the growth of female albino rats. The deficiency of vit E and Se on the other hand reduced the body weight. Vit E and Se appear to affect the growth of tissues other than lung and liver more drastically.

2). Vit E and Se deficiency significantly reduced the tissue and serum vit E and Se levels and supplementation on the other hand enhanced the same indicating the efficacy of dietary regimen employed in the present study.

3). Vit E and Se deficiency or exhaustive physical exercise resulted in free radical (R·) generation and subsequently non-enzymatic lipid peroxidation in the lung tissue making it vulnerable to oxidative damages.

4). Free radicals (R·) generation by ESR spectra revealed that the free radical generation was increased in deficient group, which was further amplified when these animals were subjected to swimming exercise until exhaustion.

5). However no such radicals and lipid peroxidation were detected in +E,+Se animals even after exhaustive exercise indicating the protection offered by dietary supplementation of vit E and Se. The studies thus conclusively demonstrate that the lung tissue is vulnerable to
oxidative damages during sudden burst of exhaustive exercise, more so during impaired antioxidant defenses such as vit E and/or Se deficiency.

6). Besides non-enzymatic lipid peroxidation, vit E and Se deficiency resulted in induction of enzymatic lipid peroxidation as evidenced by increased product formation of PGH synthase and lipoxygenase pathways. Among the cyclooxygenase products PGD$_2$ was found to be the major compound and 12 HETE as the major lipoxygenase product.

7). Exhaustive exercise in the form of swimming further enhanced the PGH synthase (PGs) and lipoxygenase (12 HETE) products.

8). Dietary supplementation of vit E and Se however, significantly reduced the cyclooxygenase and lipoxygenase pathways. Thus vit E and Se supplementation appear to protect lung tissue from enzymatic lipid peroxidation also. Since lipoxygenase products are the mediators of several degenerative diseases, it appears that vit E and Se deficient animals are vulnerable to oxidative damages via enzymatic lipid peroxidations.

9). Oxidant stress induced by either vit E and/or Se deficiency or exhaustive physical exercise in the lung has also resulted in compensatory mechanisms by stimulating thiol metabolism, mainly in the form of GSTs.

10). In view of the important role in antioxidant defenses GSTs were purified from hepatic and lung tissues of control and trained animals by affinity chromatography. Subunit pattern was analyzed by western blot using polyclonal antibodies raised against liver affinity purified GSTs.

11). Single bout of exhaustive exercise induced hepatic as well as pulmonary GSTs especially with Ya and Yc subunits. Both vit E and Se deficiency also induced GSTs with Ya and Yc subunits.

12). Purified hepatic GSTs showed 3 major subunits Ya, Yb and Yc with molecular weight of 25.6, 27 and 28 kDa. Exercise training induced both Yc and Yb subunit containing
isozyymes as indicated on SDS-PAGE. The induction of Yc subunit containing GSTs might be aimed at inducing cellular non-Se-GSH Px activities to compensate for the loss of Se-GSH Px in Se deficient animals.

13). Purified pulmonary GSTs showed (on SDS-PAGE) 5 subunits in lung tissue. Exercise training induced one more new subunit of GST with a molecular weight of 24 kDa besides induction of Yc subunit.

14). Hepatic GSTs of both control and ET animals exhibited LTC$_4$ synthase activity, more so with that of ET animals. This enhanced LTC$_4$ synthase activity exhibited by GSTs could be due to induction of Yb subunit (which has the highest LTC$_4$ synthase activity) observed in ET animals.