The current study was undertaken to determine the intense toxic effects of oral administration of BaP on liver, lung and erythrocytes of male BALB/c mice and to determine whether preadministration of green tea and white tea to BaP incubated mice could modulate the toxic effects to any extent.

Male BALB/c mice, 6-8 weeks old, weighing 26 ± 2 g received pretreatment with green tea (2%) and white tea (2%) as sole source of drinking water for 35 days. Normal mice and mice pretreated with green tea and white tea received benzo(a)pyrene (125mgkg⁻¹ body weight) dissolved in corn oil (10 mlkg⁻¹ body weight) orally after two hours of the pretreatments. The animals in all six groups were housed in polypropylene cages with rice husk and provided ad libitum access to clean drinking water and standard animal pellet diet throughout the course of experiment. The important findings of the present study are summarised below:

The water consumption in control and BaP treated groups were higher throughout the experiment (35 days) as compared to tea consumption. There were no statistically significant differences in the consumption of green tea and white tea among respective groups. The tea consumption (ml/group/day) was less in the first week but increased second week onward and remained more or less constant thereafter. The food consumption was similar among all groups throughout the experimental period.

Pretreatment with green tea and white tea did not indicate any significant alterations in the levels of LPO, PCC and GSH in liver and lung of mice as compared with normal control group. But the BaP
incubation significantly increased the level of LPO and PCC both in lung and liver. On the other hand level of GSH was reduced in BaP treated animals as compared with normal control. Administration of white tea before BaP incubation significantly lowered the elevated levels of LPO and PCC, and increased the GSH content in lung and liver.

The level of GSH was significantly higher in the BaP treated group that received pretreatment with white tea, but green tea pretreatment did not show any significant increase in GSH content when compared with white tea.

The BaP treatment reduced the activities of CAT, SOD and GR significantly in liver and lung as compared with control group. The GT preadministration for 35 days in BaP treated group significantly elevated the activities of SOD (lung), CAT (liver and lung) and GR (lung). The white tea significantly increased the activities of SOD, CAT and GR in both the tissues. Moreover, administration of white tea before BaP treatment altered the activity of CAT in both the tissues significantly when compared with green tea.

BaP incubation resulted in significant increase in the % DNA in comet tail and tail moment in the pulmonary and hepatic tissue cells. Furthermore, a significant increase in BaPDE-DNA adducts and 8-oxo-dG levels in hepatic and pulmonary tissues were observed in BaP treated animals. However, preadministration of GT and WT showed a considerable diminution in % DNA in the comet tail, tail moment, BaPDE-DNA adducts and 8-oxo-dG levels in liver and lung of BaP treated animals. The results of comet assay revealed that magnitude of DNA damage is more in the pulmonary tissue than liver. Pretreatment with green tea and white tea prevent the DNA damage in both the tissues. However, white tea was observed to be more effective in preventing DNA damage as compared to green tea both in liver and
lung. Irrespective to this both green tea and white tea showed significant reduction in the BaPDE-DNA adducts and 8-oxo-dG content in hepatic and pulmonary tissues.

The SEM studies of erythrocytes revealed drastic alterations in the topographical morphology of the RBCs as different cell types were observed in BaP treated animals. Plenty of cells were changed to echinocytes and acanthocytes. The present study also revealed the recovery in the number of discocytes in the BaP treated group that received pretreatment with green tea and white tea. Moreover, this amelioration was highly apparent in white tea pretreatment group where the number of echinocytes and acanthocytes were less as compared to green tea pretreated group.

As observed during the current study single dose of BaP led to the modulation of detoxifying and biotransformation enzymes. Preadministration of GT and WT to BaP treated group elevated the levels of hepatic and pulmonary GST and QR (phase II enzymes). The stimulation of liver GST and QR in response to GT and WT pretreatment suggesting possible role of tea against BaP induced alteration in detoxifying enzymes. The CYP 1A1 and CYP 1B1 (phase I enzymes) level increased in hepatic and pulmonary tissues of BaP treated group as compared to normal control group. The group, which received pretreatment with GT and WT tends to decrease the elevated level of CYP 1A1 and CYP 1B1 in both the tissues.

The present study demonstrated that GT and WT pretreatment inhibit the phase I (EROD, PROD) and also influencing the phase II (GST, QR) enzymes in liver and lung of the BaP treated animals. The inhibition of phase I and promotion of phase II enzymes could be associated to the detoxification and elimination of BaP metabolites in the current study.

Amplification and sequence analysis of the DNA samples
obtained from normal control and BaP exposed mice indicate that the selected exons 5 and 7 of the p53 gene were not mutated.

It is concluded from the present study that regular drinking of green tea and white tea might be effective in providing protection against oxidative stress and genotoxicity induced by benzo(a)pyrene.