GENERAL SUMMARY
Results of the present investigation may be summarized as follows:

1. Ehrlich ascites carcinoma (EAC) cells during its development for 12, 15 and 18 days following its intraperitoneal inoculation to the recipient mice
   (a) significantly increased (i) the cell viability, oxygen consumption, \([^3]H\)-thymidine incorporation, the plasma corticosterone level, hepatic lipid peroxidation (LP) and \([^3]H\)-GABA binding to mice whole brain receptor with the increase of duration of EAC cell development
   (ii) whole brain GABA level during the development of EAC cell for 18 days
   (iii) the activities of whole brain GAD and GABA-T in mice during the development of EAC cells for 15 and 18 days
   (b) significantly decreased with the increase of duration of EAC cell development
   (i) adrenal corticosterone level
   (ii) both the total and reduced adrenal ascorbic acid levels
   (iii) the activities of hepatic CAT and SOD
   (iv) the locomotor activity (LA) of mice
   (c) didn't significantly affect (i) hepatic GPx activity
   (ii) whole brain GABA level during the development of EAC cells for 12 and 15 days
   (iii) GAD and GABA-T activities during the development of EAC cell for 12 days

2. Long-term consumption (for 24 - 30 consecutive days) of caffeine in mice
   (a) increased the activities of hepatic CAT and SOD and decreased liver LP without any significant alteration of hepatic GPx activity
   (b) didn't significantly change the (i) levels of plasma and adrenal corticosterone, (ii) both total and reduced adrenal ascorbate, (iii) steady state level of GABA,
its metabolizing enzymes (GAD and GABA-T) and [3H]-GABA binding to its receptor in whole brain

(c) increased LA upto 4 consecutive days of caffeine treatment following which it was gradually reduced and dropped to control value following 12 consecutive days of caffeine treatment.

3. Treatment of caffeine to mice for 12 consecutive days prior to EAC cell inoculation (i.p) followed by continuation of its treatment for another 12, 15 and 18 consecutive days during EAC cell growth

(a) inhibited the growth of EAC cells by suppressing the EAC cell (i) viability, (ii) oxygen consumption and (iii) [3H]-thymidine incorporation.

(b) restored the EAC cell-induced (i) increase in plasma corticosterone level and hepatic LP

(ii) decrease in adrenal corticosterone level, both total and reduced adrenal ascorbic acid levels and hepatic CAT and SOD activities

(c) restored long-term caffeine-induced (i) induction of hepatic CAT and SOD

(ii) reduction of hepatic LP

(d) didn't significantly change the steady state level of whole brain GABA of mice with respect to the corresponding control as well as their corresponding caffeine treated conditions alone. The whole brain GABA level on the other hand, was significantly decreased when mice were pretreated with caffeine for 12 consecutive days and continued for another 18 consecutive days with respect to the corresponding EAC cell developing conditions alone; but, pretreatment of mice with caffeine for 12 consecutive days and continued for another 12 or 15 consecutive days after the inoculation of EAC cells didn't significantly change the whole brain GABA level with respect to the corresponding EAC cell developing conditions alone.

(e) (i) decreased GAD activity of mice whole brain following 24 consecutive days of caffeine treatment with respect to their corresponding control as well as their
corresponding EAC cell developing conditions alone. No significant change in GAD activity was observed with respect to their corresponding caffeine treated conditions alone.

(ii) didn't significantly change GAD activity following 27 consecutive days of caffeine treatment with respect to the corresponding control and also with respect to corresponding caffeine treated conditions alone. But, whole brain GAD activity was significantly decreased under similar conditions with respect to their corresponding only EAC cell developing conditions.

(iii) increased GAD activity following 30 consecutive days of caffeine treatment with respect to the corresponding control as well as with respect to the corresponding caffeine treated conditions alone. No significant change in the activity of this enzyme was observed under similar conditions with respect to the corresponding EAC cell developing conditions alone.

(f) (i) decreased GABA-T activity of mice whole brain following 24 consecutive days of caffeine treatment with respect to their corresponding control as well as their corresponding EAC cell developing conditions alone and also with respect to the corresponding caffeine treated conditions alone.

(ii) didn't significantly change GABA-T activity following 27 consecutive days of caffeine treatment with respect to the corresponding control and also with respect to the corresponding caffeine treated conditions alone. But, whole brain GABA-T activity was significantly decreased under similar conditions with respect to their corresponding only EAC cell developing conditions.

(iii) increased GABA-T activity following 30 consecutive days of caffeine treatment with respect to the corresponding control as well as with respect to the corresponding caffeine treated conditions alone. No significant change in the activity of this enzyme was observed under similar conditions with respect to the corresponding EAC cell developing conditions alone.

(g) antagonized the EAC cell-induced increase in $[^3H]$-GABA binding to the receptor of whole brain. From all the observations we concluded that EAC cell-induced induction of whole brain GABAergic activity was suppressed.
increased LA of mice was with the increase in duration of caffeine treatment with respect to their corresponding control as well as caffeine alone or EAC cell developing conditions alone.

In the present investigation, it may be concluded that

1. long-term caffeine consumption did not (i) significantly affect the level of plasma and adrenal corticosterone as well as both total and reduced adrenal ascorbic acid, (ii) increased the activities of the hepatic enzymes catalase (CAT) and superoxide dismutase (SOD) and decreased its lipid peroxidation (LP), (iii) developed tolerance to this drug by upregulating the reduction of central GABAergic activity in caffeine nontolerant condition.

2. Development of EAC cell (i) elevated the corticosterone levels and reduced adrenal ascorbic acid level, (ii) decreased the activities of hepatic CAT and SOD and increased LP, (iii) stimulated the whole brain GABAergic activity.

3. Treatment of caffeine prior to EAC cell inoculation and continuation of its treatment in the course of development of EAC cells (i) suppressed the viability, oxygen consumption and \[^{3}H\]-thymidine incorporation in EAC cells, (ii) restored the EAC cell-induced changes in activities of liver CAT, SOD and LP to their corresponding control values, (iii) suppressed the EAC cell-induced induction of whole brain GABAergic activity to control values.

**Conclusion**

Finally, it may be stated that long-term caffeine treatment may

(i) suppress the growth of EAC cells by modulating the adrenal ascorbate level as well as corticosterone status,

(ii) inhibit EAC cell-induced oxidative damage caused by reactive oxygen species by its scavenging property,

(iii) suppress the EAC cell-induced induction of whole brain GABAergic activity in mice.