The last two decades have seen a phenomenal growth in the field of male infertility mainly due to increased understanding of illicit drug use and adverse effects of the drugs including decrease in sperm count, imbalance in hormonal axis as well as production of ROS (reactive oxygen species) which leads to oxidative stress.

In the present investigation, the overall status of the hormone profile constituting gonadotropins (FSH and LH), testosterone, estradiol and prolactin is assessed with a simultaneous investigation on the oxidative stress markers (LPO and PPO), sperm count and gonadal histology on administration of the three recreational drugs viz., AAS, alcohol and nicotine.

The present study clearly depicts suppression of gonadotropins under the influence of all the three drugs. Of the two gonadotropins, LH is found to be more affected than FSH in all the three experimental groups. Both FSH and LH are affected markedly by AAS as compared to nicotine and alcohol. However, nicotine effect is more pronounced in case of FSH and in case of LH alcohol effect is more than that of nicotine.

A clear picture of inhibition with decreased value of testosterone is also depicted in all the three experimental groups throughout the experimental period. The highest amount of decrease is recorded in AAS-treated group which is about 48% from the normal baseline followed by nicotine (37%) and alcohol (24%).

It is clearly evident that AAS and alcohol enhance serum estradiol level up to 5.14% and 16.4% respectively on 90th day of the experiment, whereas nicotine suppresses the hormone level (14%) towards the terminal part of the experiment.

The trend of PRL under different drug abuse conditions indicates alcohol as a marked inducer which elevates the PRL level (14%) whereas AAS and nicotine depress PRL value up to 5% and 17.56% respectively.
From the present study it is also revealed that all the three drugs enhance lipid peroxide content in the three tissues – blood, liver and testes and the trend of increase in LPO is similar in all the three tissues. In case of blood, ultimate increase is more pronounced in case of nicotine followed by alcohol and AAS treatment. Liver tissue exhibits highest amount of LPO in case of alcohol followed by AAS and nicotine treatment. In case of testes tissue, AAS is most potent in exerting an inducing effect followed by alcohol and nicotine administration.

The assessment of protein peroxide content in all the three tissues (blood, liver and testes) reveals similar and parallel increasing trend in all the three experimental groups. The highest amount of PPO is recorded in the serum under the influence of all the three drugs. Of the three recreational drugs, alcohol is found to be the most potent inducer of PPO in all the tissues.

A gradual and highly significant decrease (p<0.01) in sperm count has also been recorded under the influence of all the three drugs in the present experimental set up. The highest amount of declination is observed in case of AAS followed by nicotine and alcohol treatment. The findings of suppression of sperm count is also supported by structural alteration in the testes tissue which is marked by hypospermatogenesis, maturation arrest at the level of primary spermatocytes and dystrophic calcification under the influence of AAS, hypospermatogenesis and maturation arrest in some of the seminiferous tubules under the influence of alcohol and nicotine treatment.

The findings of the present investigation are in conformity with the previous reports which clearly indicate depression of sperm count and overall hormone profile except estradiol (in case of alcohol) which may impair the male fertility status. This study also clearly depicts increase in oxidative stress markers (LPO and PPO) in all the three
tissue- blood, liver and testes. Hence, it can be summarized from the present study conducted for a period of 90 days that sperm count, FSH, LH and testosterone are maximally suppressed on chronic administration of AAS, followed by nicotine and alcohol. Estradiol is suppressed by nicotine whereas alcohol is found to be the potent inducer of estradiol followed by AAS and PRL is maximally suppressed by nicotine followed by AAS whereas alcohol elevates serum PRL levels. The findings of suppression of sperm concentration are supported by structural alterations in testes tissue under the influence of the three drugs. The correlation analysis of the results obtained in the present study reveals significant correlation ($r>0.5$) between sperm count and hormone profile and oxidative stress markers (LPO and PPO) as indicated in Fig. V.10, V.11, V.12, V.13, V.14, V.15, V.16, V.17, V.18 and V.19. However, a definite conclusion cannot be drawn from the present study as illicit drug use is not the only cause of male infertility. Future multicentric studies with larger sample size will be of help to gain a better insight to this essential problem. Further study for longer duration is required in order to support the authenticity of the results of the present investigation.