Available online on 15.04.2019 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Research Article

Prunus armeniaca (apricot) and *Mucuna pruriens* (Konch) seeds improves the liver damage in albino rat exposed to nicotine

Varsha Raj*, Arun Kumar Mishra, Amrita Mishra, Najam Ali Khan

Department of Pharmacy, Pharmacology Research Lab, IFTM University Moradabad, 244102, India

ABSTRACT

Prunus armeniaca (apricot) and *Mucuna pruriens* (Konch) both are the plant, which are extensively used as medicine in Indian traditional system from ancients, they are considered to increase the protective mechanism against ailments. Nicotine is the main copious components in smoking of cigarette and it is primarily metabolized inside the liver. The current study was performed to explore the role of ethanolic extract of *Prunus armeniaca* and *Mucuna pruriens* seed on nicotine induced lethality in rats. Animals are divided in to seven group of with each group (n=6) number of rats. Wistar rats (Group II, III, IV, VI and VIII) were administered with oral nicotine diluted with drinking water for 32 days, While (Group I) plain control was treated with drinking water concurrently, following 32 days Group III, IV were administered with two different concentration of ethanolic extract of *Prunus armeniaca* seed (200 mg/kg , 400 mg/kg) and Group V and VI received ethanoilc extract of *Mucuna pruriens* seed at different doses (400 mg/kg, 800 mg/kg). Group II served as toxicity group (5mg/kg body weight of nicotine). Rats were sacrificed 24 hrs after last day of administration (40th day), the biochemical and histopathological parameter were studies. A significance increase in the activity of SGOT, SGPT, CRT, Total bile acid, LDL, ALP, TC, TG, TBL, DBL and decreased the activity of Albumin, TP and HDL in nicotine control group was observed. Group IV and Group VI, the ethanolic extract of *Prunus armeniaca* seed (400 mg/kg) and ethanolic extract of *Mucuna pruriens* seed (800 mg/kg) make the defensive effects which were found more considerable in rats. Thus the consequence was recommended that the *Prunus armeniaca* and *Mucuna pruriens* both were exert the protecting effects during nicotine induced hepatoxicity in rats.

Keywords *Prunus armeniaca, Mucuna pruriens,* nicotine, hepatotoxicity.

Article Info: Received 18 Feb 2019; Review Completed 23 March 2019; Accepted 26 March 2019; Available online 15 April 2019

Cite this article as:

Raj V, Mishra AK, Mishra A, Khan NA, *Prunus armeniaca* (apricot) and *Mucuna pruriens* (Konch) seeds improves the liver damage in albino rat exposed to nicotine, Journal of Drug Delivery and Therapeutics. 2019; 9(2-s):138-143
http://dx.doi.org/10.22270/jddt.v9i2-s.2474

***Address for Correspondence:**

Varsha Raj, Department of Pharmacy, Pharmacology Research Lab, IFTM University Moradabad, 244102, India

INTRODUCTION

Tobacco smoking is one of the major public health problems in all countries around the world. The use of tobacco is still rising globally. Although 4000 component occurs in the cigarette, nicotine is the alkaloid most active in the tabacco. Nicotine is the amine composed of pyridine and pyrrolidine rings. Nicotine is the highly toxic substance and quickly absorbed through the respiratory tract, mouth, mucosa and skin[1].

The action of nicotine has been extensively investigated in human, animals and in a variety of cell systems. Numerous experimental and clinical evidences have supported to the key role of oxidative stress in the pathogenesis of organ disorder after nicotine exposure. Induced nicotine significantly increased the oxidative stress by enhancing the generation of reactive oxygen species and lipid peroxidation[2]. In addition, Nicotine induced a depletion of antioxidant defence system through the reduction of catalase and superoxide activities and level of glutathione peroxidise[3].

Nicotine once absorbed, is mainly metabolized by the liver to a number of major and minor metabolites. The major metabolites is cotinine, the primary product of the oxidation pathway of nicotine biotransformation has been used as a marker for nicotine intake. Seeing that the liver is major site of nicotine metabolism, it has been considered highly susceptible for the oxidative stress associated with the toxicity of nicotine. Infect, many epidemiological studies have shown an association between smoking and accelerated progression of liver fibrosis in patient with variety of chronic disease such as primary biliary cirrhosis and chronic hepatitis have also been reported[4].

Prunus armeniaca and commonly known Khubani, apricot as belongs to the family *Rosaceae*, is a tree. It has been used in Indian system of medicine for the cure of number of diseases from thousands of years. The seed of this tree has been

commonly used in ayurvedic preparations to bring hepatoprotective effect in drug[5, 6].

Mucuna pruriens is and indigenous plant possessing several medicinal properties. *Mucuna pruriens* belong to the family *Fabaceae* and commonly known as velvet bean," is a vital annual climbing legume. The plant is traditionally used for the treatment of Parkinson disease, Alzimer,s disease, hepatoprotective activity and diabetes[7].

So the present study was designed to investigate the hepatoprotective effect of ethanolic extract of *Prunus armeniaca* and *Mucuna pruriens* seed in nicotine induced hepatotoxicity in albino rats.

MATERIAL AND METHODS

Animals

Animals were procured from Animal House, IFTM University, Moradabad, India. The Wistar albino rats, weighing 200-250gm, were used for the present study. They were housed in the cleaned propylene cage and were maintained under the standard laboratory condition (25 ± 2^{0}C with dark/light cycle 12/12h).They were feed with standard commercial food pellets diet (Hindustan lever, India) and water *ad libitum*. The animals were acclimatized to laboratory condition for one month to experiment.

Chemicals

Nicotine ((-)-nicotine (-[-]-1 methyl -2[3-pyridyl]-pyrroline), was purchased from sigma fine chemicals, Chennai, India. (Special drinking bottles were used to avoid nicotine solution exposition to light) All chemicals which were used during the experiments should be analytical grade.

Plant Collection The seeds of *Prunus armeniaca* procured from H.P (Dist. Sirmour) and *Mucuna pruriens* was collected from Lucknow in the month of July-August and then authenticated by Dr. Sunita Garg Head Raw Materials Herbarium & Museum, NISCAIR, New Delhi.

Prepration of extracts

Extract preparation for *Prunus armeniaca*[8]

Prunus armeniaca seeds were collected and rinsed with tap water and dried. This dried seeds were grind to produce homogenous powder using a mechanical grinder. Coarsely powder (500 g) were extracted from the continues hot percolation method by soxhlet apparatus using ethanol (99.9%) for 24 h (55-60^{0}C). After completion of extractions, the solvent was removed by distillation and the pale yellow colour residue was obtained which was stored inside the desiccators.

Extract Preparation for *Mucuna pruriens*[9]

Freshly collected seed of *Mucuna pruriens* were dried in shade and pulverized to get coarse powder. A weighed quantity of the powder (1000 g) was passed through sieve number 40 and it was subjected to hot percolation method from the soxhlet apparatus by using ethanol (99.9%) as a solvent for 24 h (50-60^{0}C). Before and after the ethanolic extraction, the marc was completely dried. After finishing the extraction, the solvent was excluded by distillation. Dark brownish residue was finding. The residue was then stored in desiccators.

Experimental design

Experimental Design[10]

The study was conducted as described by A total of 42 rats were equally divided into 7 groups (n = 6).

Group-I which served as plain control, were give clean drinking water.

Group-II Animals were received nicotine (5 mg/kg bw) in drinking water for 32 d to induced hepatotoxicity.

Group-III, IV Animals were received ethanolic extract of *Prunus armeniaca* seed (200mg/kg, 400 mg/kg) in drinking water for 7 d after 32 d of nicotinic administration.

Group-V, VI Animals were received the ethanolic extract of *Mucuna pruriens* 400 mg/kg, 800mg/kg seed in drinking water for 7 d after 32 d of nicotinic administration.

Group-VII Animals were received Ursodeoxycholic acid 250 mg/kg in drinking water for 7 d after 32 d of nicotinic administration.

At the end of the experimental period (40th d) all the animals were anaesthetized and sacrificed by cervical dislocation after an overnight fasting. Blood were collected in heparinise tubes and centrifuged at 5000 rpm for 10 m. Plasma was separated by aspiration, transferred in to microcentrifuge/eppendorf tubes then stored -20^{0}C until the analysis. The liver was isolated and clear off blood and transferred immediately to ice cold container containing 0.9% NaCl for the following estimation.

Analysis of biochemical marker enzyme in plasma

Various enzyme levels in serum and the level of various other biochemical parameters such as i.e Serum glutamic oxaloacetic transaminase (SGOT) , Serum glutamic pyruvic transaminase (SGPT), Albumin, Creatinine (CRT), Total protein, Gamma glutamy tranpeptidase (GGTP), Total bile acid, High density lipoprotein (HDL), Low density lipoprotein (LDL), Total cholesterol, Triglyceride, Alkaline phosphate (ALP), Total bilirubin (TBL), Direct bilirubin (DBL) were estimated by using standard kits available commercially.

Histopathological studies

The liver samples from rats of different treatments were fixed in 10% formalin solution for 24 h, embedded in paraffin and then sections of 5 µm thickness were stained with the hemotoxylin and eosin. Studies were observed under the microscope.

Statistical analysis

The data obtained from the hepatoprotective activity were analysed was represented as mean ± SEM. The graph were drawn, and the statistical analysis was carried out using, Graphpad prism software version 5.0. Results were expressed as the mean ± SEM (n=6). For statical analysis of the data group mean were compared by one way analysis of variance (ANOVA) followed by Dunnet,s test, $p< 0.05$, $p< 0.01$ was considered significant.

RESULTS

The activities of enzymes in the plasma are shown in (**Table-1**). In nicotine treated rats, the SGOT, SGPT, CRT, GGTP, Total bile acids were significantly increased, Albumin, TP level decreased when compared to the control group.

Administration of *Prunus armeniaca* (200 mg/kg and 400 mg/kg) and *Mucuna pruriens* (400 mg/kg and 800 mg/kg) to nicotine treated group of rats which were significantly decreased the activities of the enzymes such as SGOT, SGPT, CRT, GGTP, Total bile acids and showed increased activity of Albumin, TP level, when compared with the nicotine treated animals.

The levels of HDL, LDL, ALP, TC, TG, TBL and DBL of

experimental animals are shown in (**Table 2**). The level of HDL, LDL, ALP, TC, TG, TBL and DBL in plasma of experimental animals significantly increased in the nicotine treated group when compare to the control group.

Significance protection were seen in the *Prunus armeniaca* (200 mg/kg and 400 mg/kg) and *Mucuna pruriens* (400 mg/kg and 800 mg/kg) supplemented animals when compared with the nicotine treated animals, but the *Prunus armeniaca* (400 mg/kg) and *Mucuna pruriens* (800 mg/kg) was more effective than the other doses for *Prunus armeniaca* (200 mg/kg) and *Mucuna pruriens* (400 mg/kg).

Table 1: Effect of *Prunus armeniaca* L. and *Mucuna pruriens* on biochemical parameters of nicotine intoxicated rats.

Treatment	SGOT (µ/g)	SGPT (µ/g)	Albumin (g/dl)	CRT (mg/dl)	Total protein (g/dl)	GGTP (U/L)	Total bile acid (µmol/L)
Normal	11.79 ± 3.11	12.80 ± 3.10	5.02 ± 0.17	0.65 ± 0.04	8.50 ± 0.20	16.78 ± 4.53	0.90 ± 0.09
Control	70.61 ± 6.10**	42.22 ± .38**	1.52 ± .99**	1.63 ± 0.07**	3.71 ± 0.31**	79.76 ± 2.21**	2.97 ± 0.20**
Nicotine (5mg/kg) + *Prunus armeniaca* 200 mg/kg	28.95 ± 5.91**	25.10 ± .96**	3.08 ± .45**	1.15 ± 0.08**	6.27 ± 0.42**	33.04 ± 13.02**	1.21 ± 0.08*
Nicotine (5mg/kg) + *Prunus armeniaca* 400 mg/kg	25.57 ± 5.36*	23.46 ± 1.87*	4.50 ± .48ns	0.86 ± 0.18*	7.32 ± 0.08*	28.25 ± 3.70ns	0.99 ± 0.03ns
Nicotine (5mg/kg) + *Mucuna pruriens* 400 mg/kg	33.09 ± 3.63**	36.01 ± .86**	1.99 ± .59**	1.17 ± 0.07**	4.84 ± 0.18**	35.75 ± 4.58**	2.05 ± 0.20**
Nicotine(5mg/kg) + *Mucuna pruriens* 800 mg/kg	30.82 ± 2.25*	27.88 ± .88**	3.87 ± 0.32*	1.09 ± 0.07**	6.41 ± 0.14**	31.38 ± 7.58*	1.74 ± 0.04**
Standard (Ursodeoxycholic acid 250 mg/kg)	24.19 ± 3.54*	20.17 ± .70ns	4.63 ± .55ns	0.83 ± 0.17ns	7.57 ± 0.29ns	20.31 ± 6.85ns	0..96 ± 0.23ns

Results are expressed as mean ± SEM n= 6, ns represent the non significant changes * p ≤ 0.05 represent less significant changes, ** p ≤ 0.01 represent significant changes, were highly significant changes compared with normal rats by one way Anova followed by Dunett test.

Table 2: Effect of *Prunus armeniaca* L. and *Mucuna prurines* on some other biochemical parameters of nicotine intoxicated rats.

Treatment	HDL	LDL	ALP	T C	Triglyceride	TBL	DBL
Normal	67.48 ± 7.84	55.41 ± 3.14	113.35 ± 4.82	83.39 ± 7.60	94.85 ± 17.43	0.39 ± 0.10	0.12 ± 0.036
Control	15.47 ± 4.56**	233.89 ± 2.25**	251.49 ± 3.71**	348.71 ± 3.32**	357.48 ± 9.81**	2.67 ± 0.12**	1.44 ± 0.09**
Nicotine (5mg/kg) + *Prunus armeniaca* 200 mg/kg	33.16 ± 6.87**	89.34 ± 5.71**	132.98 ± 9.67*	145.23 ± 0.40**	126.57 ± 11.33*	0.64 ± 0.12**	0.26 ± 0.10*
Nicotine (5mg/kg) + *Prunus armeniaca* 400 mg/kg	57.35 ± 1.17ns	73.95 ± 16.87**	125.82 ± 6.97ns	102.20 ± 5.43ns	112.85 ± 0.76ns	0.46 ± 0.05ns	0.20 ± 0.015ns
Nicotine (5mg/kg) + *Mucuna pruriens* 400 mg/kg	28.85 ± 1.25**	95.23 ± 2.98*	143.73 ± 7.51**	178.61 ± 7.68**	147.00 ± 9.15**	0.81 ± 0.10**	0.29 ± 0.05**
Nicotine (5mg/kg) + *Mucuna pruriens* 800 mg/kg	42.08 ± 9.55**	83.42 ± 6.03**	128.38 ± 9.95ns	0.24 ± 0.039*	125.47 ± 13.82*	0.71 ± 0.09**	0.24 ± 0.039*
Standard (Ursodeoxycholic acid 250 mg/kg)	60.62 ± 8.33ns	68.92 ± 5.35ns	121.7 ± 8.72ns	0.16 ± 0.07ns	110.98 ± 9.53ns	0.42 ± .07ns	0.16 ± 0.07ns

Results are expressed as mean ± SEM n= 6, ns represent the non significant changes * p ≤ 0.05 represent less significant changes, ** p ≤ 0.01 represent significant changes, were highly significant changes compared with normal rats by one way Anova followed by Dunett test.

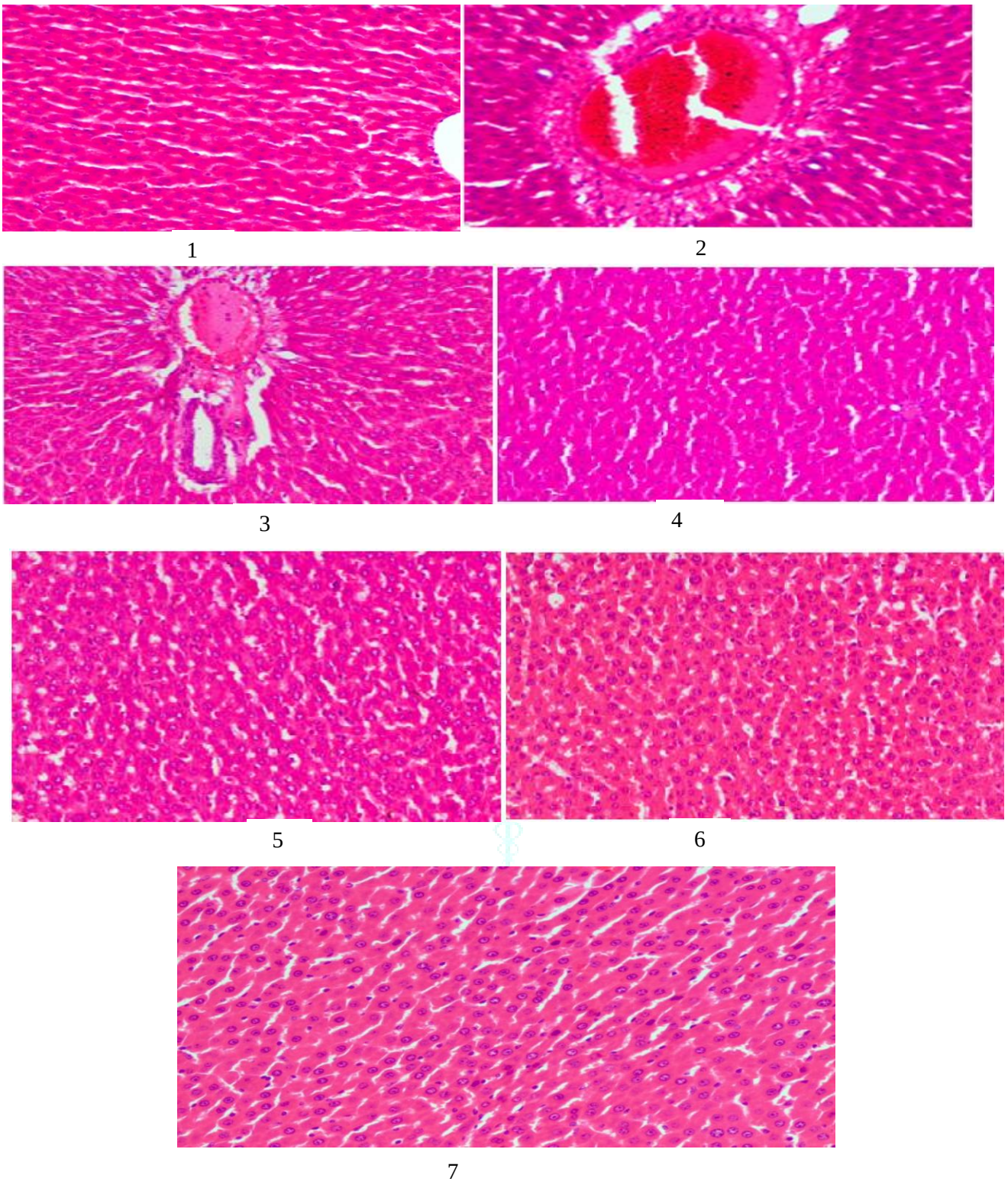

Figure 1-7 Histological monograph of extracts and standard

1. Normal ; **2.** Nicotine (5 mg/kg) only ; **3.** *Prunus armeniaca* group (200 mg/ kg + nicotine (5mg/kg)); **4.** *Prunus armeniaca* group (400 mg/ kg + nicotine (5mg/kg)); **5.** *Mucuna pruriens* (400 mg/ kg + nicotine (5mg/kg)) **6.** *Mucuna pruriens* (800 mg/ kg + nicotine (5mg/kg)) ; **7.** Standard (Ursodeoxycholic acid 250 mg/kg)

DISCUSSION

Nicotine is the main component of cigarette smoking, which reasons the oxidative injury to the, kidney, liver, heart and lungs. It is a effective oxidant which is able to generating the reactive oxygen species and free radicals. The nicotine provoked free radicals, interact with cellular membrane which causing the oxidative damage of polyunsaturated fatty acid and generating the cytotoxic aldehyde by the lipid peroxidation[11].

Lipid peroxidation is the major factor which has been incriminated in pathogenesis of various diseases[12]. An rise in the activity of SGOT,SGPT, GGTP, CRT, Total bile acid , LDL,ALP, TC,TG,TBL,DBL and decline in the activity of albumin and total protein in nicotine treated rats indicates the tissue injury[13]. The rise in the ALP activities is because of

the loss of functional integrity of cell membrane which is signifying the cellular injury[14]. Oxidative injury to the cellular membrane, generate the significant variations in molecular arrangements of lipid, consequence increased the permeability. It may showed that the further lipid peroxidation can implanted as an key for determining the degree of damage which occur in tissue membrane as a consequence of free radical generation. Lipid peroxidation was induced by in liver of the nicotine treated rats[15].

SGOT and SGPT both parameters are restricted with the liver cell, heart, gills, kidney, tissue muscles and various organs of the body[16].

These enzymes are mainly vital in monitoring and assessing the hepatic catalyasis. During the current study, the level of cholesterol was increased in the animals which were treated by nicotine. The pervasiveness of triglycerides and hypercholesterolemia has been accounted in intense smokers. Nicotine reduced the action of lipoprotein lipase, consequence of it the high level of triglycerides was found[17].

El-Sayed envisage that this enzyme is performed the vital role during the uptake of lipoprotein from the extra liver tissue[18].

Crayer revelled that catacholamines are synthesize in the cells of adrenal medulla from the nicotine stimulation and lipolysis of adipose tissue which is done by the catacholamines, consequently increased the level of TG, cholesterol and fatty acid level also[19].

TP and albumin parameters level are slightly declined in nicotine treated animals that were signalized to recover along with the treatment from ethanolic extract of *Prunus armeniaca* seed and ethanolic extract of *Mucuna pruriens* seed. This might be because of the phytochemical constituents available in the natural product in detoxifying or reducing the poisonous result of nicotine. According to Scartezzini, the total protein and albumin serve as a main source of nourishment for the tissue and the hepatic function. The location of particular oxidative injury in the number of vulnerable amino acid is at present considered as the fundamental cause of metabolic destruction through the pathogenesis. Widespread liver injury is may be occurred due to the decrease in the blood level of total proteins, which is produced exclusively by liver cells which is replicated during this study[20].

According to Erdogan- orhan the *Prunus armeniaca* (apricot) is the one of the most delicious and commercially fruit in world. *Prunus armeniaca* seed is used for various biological activities such as antimicrobial, antimutagenic , inhibitory activity against several enzyme, cardioprotective ,antinflamatory as well as antioxidant activity. Among these activities antioxidant potency of apricot has been studied extensively and the plant displayed a high antioxidant effect in both *in-vitro* and *in-vivo* test systems. The phytochemical analysis of *Prunus armeniaca* revealed the presence of mono and polysachrides, polyphenols, fatty acid, flavonoids, sterol derivatives carotenoids and volatile components due to is appealing smell[21].

According to Manalisha, *Mucuna pruriens* is used in traditionally for many diseases all over the world. It is used as aphrodisiac, diuretic, nerve tonic, high blood pressure, diabetes, tumours, cholera, antioxidant, anti Parkinson, antidote activity. The phytochemical analysis of *Mucuna pruriens* revealed the presence of saponins, tannins, flavonoids, phenol, carbohydrates. From the literature, it shows that the hepatoprotective activity of *Mucuna pruriens* seed extract related mostly to their repeated antioxidant properties. It shows that the *Mucuna pruriens* seed extract neutralizes the oxidizing potential of reactive oxygen species generated through the maintaining the cell membrane viability and integrity[22].

Thus the present study revealed that both plant ethanolic extract of *Prunus armeniaca* seed and ethanolic extract of *Mucuna pruriens* seed serve as scavenger of free radicals, which help to protect the nicotine induced toxicity in rats, both plant are helping in restoring the enzyme or biochemical level and also help in repairing the damage hepatocytes.

CONCLUSIONS

The present study confirms the effectiveness and defensive role of ethanolic extract of *Prunus armeniaca* seed and ethanolic extract of *Mucuna pruriens* seed on nicotine induced toxicity in rats. Concentration of *Prunus armeniaca* (400 mg/kg) and *Mucuna pruriens* (800 mg/kg) be more effective in counteracting the nicotine induced hepatotoxicty in rats.

ACKNOWLEDGEMENT

The authors are thankful to the management of IFTM University Moradabad for providing facilities and encouragement.

REFERENCES

1. Sander SR, Cuneo SP, Turzillo AM. Effect of nicotine and cotinine on bovine theca interna and granulose cells. Repord Toxicol. 2002; 16(6):795-800.
2. Ragavan B, Krishnakumari S. Effect of *T.arjuna* stem bark on histopathology of liver, kidney and pancreas of Alloxan-induced diabetic Rats. Afr J Biomedical Res. 2006; 9(3):189-197.
3. Lu S. Regulation of hepatic glutathione synthesis: current concepts and controversies. FASEB Journal.1999; 13(10):1169-1183.
4. Aljohani ZM, Alharbi AA, Alsaedi MF, Alahmadi AF, Alahmadi OA, Alharbi AA, ABO-Haded HM. Evaluation of the potential beneficial effects of Thymoquinone against Nicotine induced Toxicity. IJPCR. 2015; 7(6):395-398.
5. Yurt B,Celik I. Hepatoprotective effect and antioxidant role of sun sulphited –dried apricot (*Prunus aremniaca* L.) and its kernel against ethanol induced oxidative stress in rats. Food Chem Toxicol. 2011; 49(2):508-513.
6. Yigit D, Yigit N, Mavi A. Antioxidant and antimicrobial activities of bitter and sweet apricot (*Prunus armeniaca* L.) kernels. Braz J Med Biol Res. 2009; 42(4): 346-352.
7. Sultana N, Azhar I. Anti-anxiety like effect of *Mucuna pruriens* Linn. Tested in Rodents. UJP.2013; 2(4):55-61.
8. Palanisamy P, Jayakar B, Kumuthvali MV, Kumar Y, Srinath KR. Preliminary phytochemical evaluation of whole plant extract of *Dipteracanthus prostratus* Nees. IRJP.2012; 3(1):150-153.
9. Sivaraman D, Muralidaran P, Shantha Kumar S. Evaluation of Anti-microbial and anti- inflammatory activity of methanol leaf extract of *Ipomoea aquatica* Forsk. Res J Pharm Biol Chem Sci. 2010; 1(2):258-264.
10. Vadivelu J, Dawood SS. Role of *Emblica officinalis* (Amla) on nicotine toxicity to rats (Rattus orvegaicus). Int. J. Pharm. boil.sci. arch. 2013; 4(4): 775-780.
11. Helen A, Krishnakumar K, Vijayammal PL, Augusti KT. Antioxidant effect of onion oil (*Ailum cepa*. Linn) on the damages induced by nicotine in rats as compared to alpha-tocopherol. Toxicol Lett. 2000; 116 (1-2):61-68.
12. Gumusteklin K, Ciftci M, Coban A, Altikat S, Aktas O, Gul M, Timur H, Dane S. Effect of nicotine and vitamin E on glucose-6-phosphate dehydrogenase activity in some rat tissues in vivo and in vitro. J. Enzyme Inhib. Med. Chem. 2005. 20 (5):497-502.
13. Yildiz D, Liu YS, Ercal N, Armstrong DW. Comparision of pure nicotine and smokeless tobacco extract induced oxidative stress. Arch Environ Contam Toxicol. 1999; 37(4):434-439.
14. Moral DW, Hessler JR, Chisolm GM. Low density lipoprotein cytotoxicity induced by free radical peroxidation of lipid. J Lipid Res. 1963; 24(8):1070-1076.

15. Salahshoor M, Mohamadian S, Kakabarae I, Roshan Khan S, Jalili C. Curcumin improves liver damage in mice exposed to nicotine. J Tradit Complement Med. 2016; 6:176-183.
16. Park EM, Park YM, Gwak YS. Oxidative damage in tissues of rats exposed to cigarette smoke. Free Radic Biol Med.1998; 25(1): 79-86.
17. Brunzell JD, Miller NE, Alaupovic P, St.Hilaire RJ, Wang CS. Famlial chylomicronemia due to circulation inhibitor of lipoprotein lipase activity. J Lipid Res. 1983; 24(1):9-12.
18. EI-Sayed B, Eslam A. Effect of Aqeous Extract of Green Tea (*Camellia sinesis* L.) on obesity and Liver status in Experimental rats. IJPAS. 2014; 21(2):53-63.
19. Cryer PE. Diseases of the adrenal medulla and sympathetic nervous system. In endocrinology and metabolism. Mc graw – hill New York. 1981; 1:511-550.
20. Scartezzini P, Antognoni F, Raggi MA, Poli F, Sabbiani C. Vitamin C content and antioxidant activity of the fruit and of the Ayurvedic preprations of *Emblica officinalis* Gaertn. J Ethanopharmacol.2006; 104(1-2):113-118.
21. Erdogan IO, Kartal M. Insights into research on phytochemistry and biological activites of *Prunus armeniaca* L. apricot). Food Res Int. 2011; 44:1238- 1243.
22. Manalisha D, Chandra KJ. Preliminary phytochemical analysis and acute oral toxicity study of *Mucuna pruriens* Linn. In albino mice. IRJP. 2012; 3(2):181-183.