doi.org/10.36721/PJPS.2021.34.4.SUP.1555-1560.1

Evidence-based hepatic and renal toxicity evaluation of *Prunus armeniaca* L. seeds in albino Wistar rats

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Abstract: The present study is a novel approach conducted to investigate dose dependent hepatotoxicity and renal toxicity of aqueous extract of *Prunus armeniaca* L. seeds in Albino rats. The use of the seeds is limited since the seeds have been subject of high controversy because of the presence of amygdalin, (Vitamin B-17) which in some studies revealed toxicity while in others incurred anti-cancerous ability and also scarce availability of toxicity evaluation studies which stimulates the need to expedite this study which would allow utilization of seeds in the pursuit of formulating novel remedies. 1000, 1500 and 2000mg/kg body weight of extract orally administered in experimental Groups DI, DII and DIII of rats (n=6) respectively for 42 days. Blood and tissue samples collected were then evaluated using liver enzymes; Aspartate Transaminase, Alanine Transferase, Alkaline Phosphatase and Bilirubin as hepatotoxic markers, Urea, creatinine and BUN as renal function indicators, antioxidants levels of liver and kidney; Catalase, Superoxide Dismutase and Glutathione reductase as oxidative stress markers and Melondylaldehyde as indicator of lipid peroxidation. The results displayed no significant increment ($P>0.05$) in liver enzymes, reduced liver and kidney MDA levels ($P>0.05$) and dose-dependent increased activity of antioxidants. This concludes that the extract did not show any remarkable hepatotoxicity or renal toxicity rather improved antioxidant activity. The histology of liver and kidney tissues further supported that the selected doses are safe for consumption.

Keywords: *Prunus armeniaca* L., hepatotoxicity, renal toxicity, aqueous extract.

INTRODUCTION

Herbal remedies are patronized globally due to their wide acceptance, easy approach and cost-effectiveness. For centuries the practice of deploying herbal therapies has been sighted but reported cases of toxicity today, invite some serious concerns regarding the safe or appropriate use of herbs. Some herbs which are marked safe when formulated in aqueous extract reveal toxicity when extracted with solvent other than water like methanol or ethanol. Some herbs by virtue of their active contents are toxic however decoction makes them safe since they become inefficient in aqueous extract formulation. (Builders, 2018).

Prunus armeniaca L. (apricot) is a plant of Rosaceae family with 175 species known. The plant's growth is confined to regions with favorable environmental conditions. *P. armeniaca* holds its reputation because of antioxidant attributes which make it effective for fighting against cancer and cardiac problems (Wani *et al*., 2015). The plant as a whole is a functional source of numerous health benefits. Fruits are valuable nutraceuticals being the carriers of carbohydrates, vitamins, potassium, dietary protein, oil and fiber, phenols, minerals, carotenoids and ester (Dragovic *et al*., 2007; Yigit *et al*., 2009). The biological significance of the whole plant has urged its cultivation to spread far and wide which begets the seeds to be generated in plentiful amounts (Gornas *et al*., 2015). Pakistan is sixth in the list of cultivating apricots with 177,630 tons production yearly (Faostat, 2013). The seeds are least probed part of plant with a history of traditional therapeutic concerns such as anti-tumorous, anti-inflammatory, radio-protective and immune modulatory functions. Moreover the seeds present a controversial subject because of the presence of Amygdalin, (Vitamin B-17) producing toxicity in some studies while anti-cancerous ability in otherwise (Rudzinska *et al*., 2017; Gomaa, 2013). Amygdalin is a compound related to cyanogenic glycosides. The reported content of amygdalin in the seeds is approximately 20-80 µmol/g. (Pimenta *et al*., 2014). This ambiguity stimulates the need of carrying out an evidence-based toxicity study of aqueous extract of *P. armeniaca* (AE) seeds in dose-dependent manner in Wistar rats. Liver is the core for detoxification of xenobiotics and toxic metabolites making it vulnerable to damage and kidney is the second target organ of toxicity for it is responsible for the clearance of blood (Fuchs *et al*., 2012). These organs have to be examined due to their frequent exposure to drugs and reactive metabolites. This study aims to assess the functions of both organs by measuring the levels of liver enzymes as toxicity markers; Alanine Transferase (ALT), Aspartate Transaminase (AST), Alkaline phosphatase (ALP) and Bilirubin (Bil), kidney function indicators; plasma Urea, Creatinine and Blood Urea Nitrogen (BUN), liver and kidney antioxidants; Catalase (Cat), Superoxide dismutase (SOD) and Glutathione reductase (GSH), lipid peroxidation; Melonylaldehyde (MDA) and histological examination of both organs.

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MATERIALS AND METHODS

Preparation of decoction

P. armeniaca fruits were obtained from local market authenticated and their seeds were separated, washed, dried and crushed into powder. 20g of that powder were added to 200 ml of boiling water to make aqueous extract and incubated with intermittent stirring for an hour at 37°C. The obtained mixture were filtered through Whatman filter paper, and then lyophilized. The final extract was stored at -24°C.

Animal Grouping and Treatment

24 Wistar albino rats weighing 150-250g were acclimatized in clean cages with 12h light and dark exposure at 30°C. Animals were grouped into four, each having six rats (n=6); Control untreated and Group DI, DII and DIII treated orally with doses; (DI) 1000 mg/kg, (DII) 1500mg/kg and (DIII) 2000 mg/kg body weight respectively for 42 days. Doses were selected after determination of LD50 by Lorke's method, 1983. Animals were decapitated post experimental period with their blood collected and tissues isolated for histological examination.

Liver and Kidney homogenates Preparation

Homogenates of isolated liver and kidney were made by first immersing saline-washed and finely chopped tissues in 1.17% of potassium chloride and processed using electric homogenizer. The obtained sample was centrifuged at 8000 rpm for 300 sec at 4°C. The supernatant (post-mitochondrial) was centrifuged again at 10,500 rpm for 25 min with at 4°C. The final supernatant was obtained to be processed for biochemical estimations.

Liver function assay

Liver function was tested by measuring liver enzyme levels; AST (Reitman *et al*., 1957), ALT (Wilkinson *et al*., 1972), ALP (Tietz *et al*., 1983) and Bilirubin levels (Dangerfield *et al*., 1953) in plasma.

Renal function assay

Renal function was evaluated by measuring biomarkers; Urea (Kaplan, 1969), Creatinine (Allen *et al*., 1982) and Blood Urea Nitrogen (BUN).

Liver and Kidney antioxidant function

Liver and Kidney antioxidant activity were evaluated by measuring Catalase (Sinha *et al*., 1972), SOD (Sinha *et al*., 1972) and GSH (Calberg & Mannervik, 1985) in tissues.

Liver and Kidney Lipid Peroxidation (LPO) assay

Liver and Kidney LPO was evaluated by measuring the levels of MDA using method described by (Ohkawa *et al*., 1978)

Ethical Guidelines

The protocols used for animal handling were followed in accordance with the ethical committee of local animals which run in parallel with the international norms for laboratory work and animal processing. The study was approved by Institutional Bioethics Committee (IBC), IBC Secretariat, UOK, Karachi, Pakistan on 12-10-2017.

STATISTICAL ANALYSIS

The data is tabulated as mean ± standard and calculated through Statistical Package for the Social Sciences version 16 (SPSS) using ANOVA-one way analysis of variance for comparisons of mean between groups with $P<0.05$ considering significant statistically.

RESULTS

Assessment of Liver Functions

Table 1 displays results of rats treated with 1000mg/kg, 1500mg/kg and 2000mg/kg of AE. In our study, there is significant reduction in ALT levels ($P<0.05$) observed in Group DIII in comparison with Control whereas non-significant reduction in comparison with Group DI and DII. There is non-significant reduction ($P>0.05$) of AST observed in Group DII and DIII as compared to Control. The level of AST is significantly reduced in Group DII ($P<0.01$) as compared to Group DI (table 1). Serum level of ALP in rats treated with low, medium and high doses did not present any considerable alterations. The concentration of ALP reduced non-significantly ($P>0.05$) with increasing dose induction. (table 1) The plasma levels of Bilirubin were not significantly altered in treated groups as compared against control group ($P>0.05$). No increment or decline has been observed in rats treated with doses 1000, 1500 and 2000 mg/kg of the extract (table 1).

Renal Function Assay

Table 2 presents plasma levels of Urea, creatinine and BUN as mean ± standard in rats treated with 1000mg/kg, 1500mg/kg and 2000mg/kg of apricot seed extract. In our study, there are no significant differences ($P>0.05$) observed in levels of Urea, Creatinine and BUN.

Assessment of Liver and Kidney Antioxidants in Rats Treated with Different Doses of Aqueous Extract of P. armeniaca Seeds

Results of Liver and Kidney antioxidant activity in control untreated and treated groups with doses 1000, 1500 and 2000mg/kg of the AE are tabulated as mean ± standard in (table 3).

Liver antioxidants

The data shows that catalase activity is significantly elevated ($P<0.05$) in highest dose induced group DIII as compared to control group. There was non-significant increase ($P>0.05$) in groups DII and DIII as compared to control. There were no significant changes in SOD activity in group DI and DII as compared to control ($P>0.05$). However, non-significant elevation of SOD activity ($P>0.05$) was observed in highest dose treated

group DIII as compared to Control, Group DI and DII. GSH activity was found to be increasing non-significantly (P>0.05) in dose-related manner in group DI, DII and DIII as compared to control (table 3)

Kidney antioxidants

The results of Kidney catalase activity assay in table 3 show that catalase activity is significantly elevated (P<0.05) in highest dose induced group DIII as compared to Control, Group I, and DII. There were no significant changes observed in Group DI against control, Group DII against Control and Group DI and Group DIII against Group DII (P>0.05). There was significant increase in SOD activity observed in Group DIII against Control and Group DI (P<0.05). There were significant reduction in GSH activity in Group DIII as compared to Group DII. (P<0.05).

Assessment of Lipid Peroxidation in Rats Treated with Different Doses of Aqueous Extract of P. armeniaca Seeds

The findings of LPO in rats treated with 1000mg/kg, 1500mg/kg and 2000mg/kg of AE are tabulated as mean ± standard in table 4. There is no significant increase observed in MDA levels (P>0.05). However, MDA level in liver is reduced non-significantly in dose related manner in Group DI, DII and DIII as compared to Control and MDA in kidney is reduced significantly in highest dose treated Group DIII as compared to Group DI.

Liver Tissue Histology

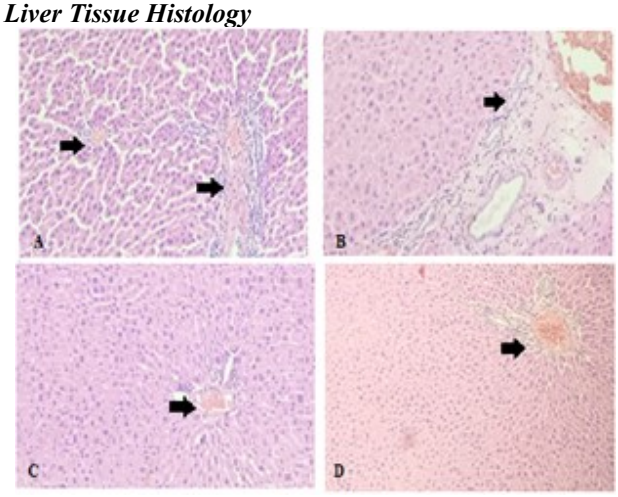

Fig. A: Hepatic tissue histology of Control rats (10X). The fig. shows intact structure of hepatocyte and central vein. However minimal inflammation is marked in portal region **Fig. B**: Hepatic tissue Histology of Group DI rats (10X). the arrow marks mild inflammation in portal region which is similar to liver histology of control rats. **Fig. C:** Hepatic tissue histology of Group DII rats (10X). The arrow marks minimal inflammation in portal region which resembles with that of control's histology. **Fig. D** Hepatic Tissue histology of Group III treated rats (10X).The arrow marks minimal inflammation in portal region.

Histopathological findings

The histology of liver and Kidney was studied in Control, Group DI (Dose 1000mg/kg), Group DII (Dose 1500mg/kg) and Group DIII (Dose 2000mg/kg). There was no trace of toxicity observed in the hepatic architecture in the control group (fig. 8), Group DI, DII and DIII (figs. 9-11), respectively. Minimal portal inflammation is marked in Groups I and DII which was similar to the liver histology of control rats whereas the other histological features studies such as fatty change, periportal necrosis, focal necrosis, fibrosis, cirrhosis, dysplasia and enlargement were not observed in any group. The intact hepatic architecture proves that the AE has not caused any significant alteration in the liver tissues.

Renal Tissue Histology

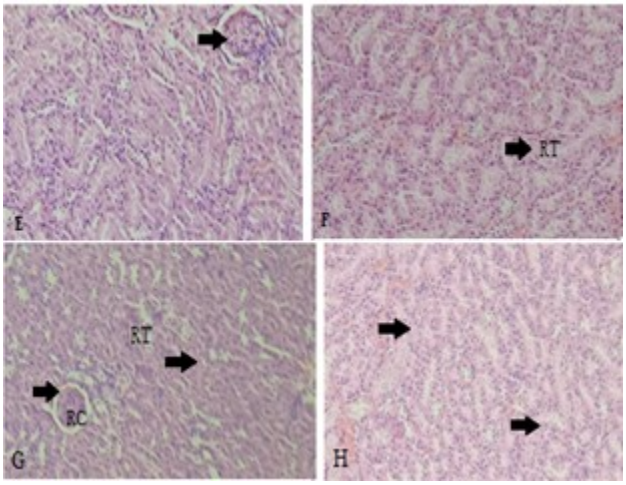

Fig. E: Renal Tissue histology of Control rats (10X). Intact structure of Glomerulus (G) and renal tubules (RT) is marked. However minimal inflammation and mild interstitial edema is sighted **Fig. F**: Renal Tissue histology of Group DI treated rats (10X). Intact structure of Renal tubules is marked with no signs of considerable damage. **Fig. G:** Renal Tissue histology of Group DII rats (10X). Intact structure of G and RT is noted. **Fig. H:** Renal Tissue histology of Group DIII treated rats (10X). Intact structure of renal tubules with no mark of toxicity. However minimal inflammation and mild interstitial edema is sighted in all Groups which is consistent with finding in control's histology.

DISCUSSION

The present study aimed to evaluate the effects of aqueous extract of *P. armeniaca* seeds on liver and kidney functions. The findings of hepatic enzymes did not show any sign of toxicity since ALT, ALP and Bilirubin were not changed significantly however significant reduction in AST levels indicate that apricot seeds at given doses are non-toxic for liver functions (table 1). The histological finding showed intact microstructure of hepatic tissues

Table 1: Comparison of plasma liver enzymes rats treated with different doses of aqueous extract of *P. armeniaca* (AE) seeds

	Control	Group DI_1	Group $DII_{1,2}$	Group $DIII_{1,2,3}$
ALT (U/l)	67.5±20.43	63.36 ± 4.01^{n}	$59.9 \pm 37.58^{n,n}$	$54.43 \pm 6.12^{a,n,n}$
AST (U/l)	18.5±4.67	21.83 ± 2.46^{n}	$14.08 \pm 3.78^{n,b}$	$13.34 \pm 8.32^{n,n,n}$
ALP (U/l)	46.92 ± 2.27	44.16 ± 2.28^{n}	$41.98\pm 0.95^{n,n}$	$40.72 \pm 0.96^{n,n,n}$
BIL (mg/dl)	0.30±0.03	0.27 ± 0.014^{n}	$0.25 \pm 0.04^{n,n}$	$0.244 \pm 0.04^{n,n,n}$

Table 2: Comparison of plasma urea, creatinine and bun in rats treated with different doses of aqueous extract of *P. armeniaca* Seeds

	Control	Group DI_1	Group $DII_{1,2}$	Group $DIII_{1,2,3}$
Creatinine (mg/dl)	0.83±0.49	0.8 ± 0.47^{n}	$2.4\pm 0.98^{n,n}$	$2.2\pm 0.98^{n,n,n}$
Urea (mg/dl)	19.44±4.81	22.7 ± 5.35^{n}	$22.22\pm 4.81^{n,n}$	$25\pm 8.33^{n,n,n}$
BUN (mg/dl)	9.08±2.24	10.64 ± 2.5^{n}	$10.38\pm 2.24^{n,n}$	$11.68\pm 3.8^{n,n,n}$

n=non-significant figures (P>0.05), a= (P<0.05), 1= in comparison with Control, 2 in comparison with Group DI, 3= in comparison with Group DII

Table 3: Comparison of liver and kidney antioxidants in rats treated with different doses of aqueous extract of *P. armeniaca* Seeds

		Control	Group DI_1	Group $DII_{1,2}$	Group $DIII_{1,2,3}$
Catalase	Liver	104.59±24.57	144.96 ± 96.32^{n}	$174.82\pm 48.56^{n,n}$	$176.54\pm 30.85^{a,n,n}$
	Kidney	85.07±2.47	88.61 ± 2.53^{n}	$91.88\pm 5.07^{n,n}$	$136.14\pm 5.75^{c,c,c}$
SOD	Liver	6.08±0.20	5.64 ± 1.46^{n}	$3.13\pm 3.68^{n,n}$	$9.89\pm 2.86^{n,n,n}$
	Kidney	5.45±3.42	4.89 ± 3.31^{n}	$8.02\pm 2.86^{n,n}$	$11.72\pm 0.21^{a,a,n}$
GSH	Liver	0.028±0.024	0.03 ± 0.027^{n}	$0.044\pm 0.035^{n,n}$	$0.061\pm 0.023^{n,n,n}$
	Kidney	0.36±0.35	0.22 ± 0.20^{n}	$0.51\pm 0.23^{n,n}$	$0.28\pm 0.15^{n,n,a}$

1= as compared to control group, 2=as compared to Group DI, 3= as compared to Group DII. n=non-significant values P>0.05, a= P<0.05

Table 4: Comparison of lipid peroxidation in liver and kidney of rats treated with different doses of aqueous extract of *P. armeniaca* Seeds

		Control	Group DI_1	Group $DII_{1,2}$	Group $DIII_{1,2,3}$
MDA (µmole/g tissue)	Liver	6.25±0.86	7.72 ± 3.06^{n}	$5.79\pm 4.57^{n,n}$	$4.48\pm 1.38^{n,n,n}$
	Kidney	11.72±2.64	10.98 ± 8.69^{n}	$11\pm 3.32^{n,n}$	$4.28\pm 5.28^{a,n,n}$

n=non-significant value (P>0.05), a= (P<0.05) 1, 2, 3= in comparison against control, Group DI and Group DII respectively.

which further support the safety of extract. The minimal inflammation marked in Group DI, DII and DIII is not significantly different from that of liver's histology of control rats (figs. A, B, C and D). This may be present because of environmental stress or any reason other than the outcome of AE treatment. Our findings are supported by the results of a study conducted on apricot seed treatment in liver damage induced rats which declared that Apricot seeds possess liver protecting attributes. (Ramadan *et al*., 2020; Ozturk *et al*., 2009) The defensive effects of ethanol extract prepared from *P. armeniaca* seeds on nicotine induced hepatic injury were also reported by Raj *et al*. (2019).

Antioxidants are essential for scavenging free radical species that mark oxidative stress generated in response to toxicity. The liver antioxidant levels in the study showed non-significant and dose-dependent increased activity in the treated groups as compared to control group (table 3). The reducing AST levels and increasing antioxidants not only suggest that the extract is non-toxic but also illustrate its biological significance. The results of a study which declared apricot kernel oil has an improving effect on liver antioxidant status support our finding (Kutlu *et al*., 2009).

Lipid peroxidation (LPO) depresses membrane fluidity thereby damage cells and lead to the formation of plaques. Melondylaldehyde (MDA) is a byproduct of LPO therefore it is used as a LPO marker. The non-significant and dose dependent reduction in MDA levels observed in our study indicate that the extract did not cause any lipid peroxidation in liver tissues (table 4). Our results correlate with the study that observed significant reduction in liver MDA levels in hypercholesteremic rats treated with apricot kernel oil (Kutlu *et al*., 2009).

Kidneys contribute in regulating the homeostasis of body therefore renal damage caused by toxicity can bring down other organs as well (Mahjour *et al*., 2017). Renal toxicity is marked by enhanced urea, creatinine and BUN. Our results showed no significant alterations in plasma levels of creatinine, urea and BUN after treating rats with selected doses (table 2). Kidney antioxidants were also observed to be increasing in dose-dependent manner. The histopathological analysis of renal tissues of control and treated rats is displayed in (figs. E, F, G and H). Kidneys of control and other groups of rats showed intact structure of glomerulus, tubules and no sign of capillary congestion or other damage. However minimal inflammation and mild interstitial edema which is sighted in control rats is also noted in Group DI, DII and DIII which suggest that these effects are not consequence of AE. Kurus *et al*. (2014) reported renoprotective role in radiation induced injury. Another study supported hepatorenal protective effects of the seed extract in Cyclophosphamide (CTX) induced injury in mice (Elwan *et al*., 2020).

The non-toxic effects that are observed might be because the toxic metabolite of Amygdalin, cyanide is suppressed by several mechanisms proposed in mammalian body. 70 % of cyanide converts to thiocyanate (SCN) in the presence of sulfur donors. SCN doesn't block mitochondrial pathway like cyanide does. (Bilska Wilkosz *et al*., 2015). Furthermore, (2-amino-2-thiazoline-4-carboxylic acid) ATCA is also a stable metabolite produced upon reaction of cyanide with L-cystein (Bhandari *et al*., 2014). There is no comparable study that has been established as yet that has investigated renal toxicity caused by apricot seeds. This study is a novel approach that studies effects of apricot seeds on hepatic and renal functioning. However, further investigations are still required to assess the toxicity of apricot seeds on other physiological systems.

CONCLUSION

The presence of amygdalin in *P. armeniaca* seeds have limited the utilization of seeds in the field of medicine despite of the significance of essential components present in the seeds. The study undertaken is the first that illustrates evidence-based toxicity report of liver and kidney functions after administering aqueous extract of *P. armeniaca* seeds for 42 days at selected doses. The findings of this study justifies that the aqueous extract is non-toxic for liver and kidney at selected doses. Further investigation on other physiological systems before deploying it while commencing anti-cancerous treatment is needed to ensure its safety.

ACKNOWLEDGEMENTS

The support and guidance of the teaching staff of Department of Physiology, UOK is highly acknowledged.

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