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Nephroprotective Potential of Ethanol Extract of Kepok Banana Peels (Musa paradisiaca) in Cisplatin-Induced Wistar Rats

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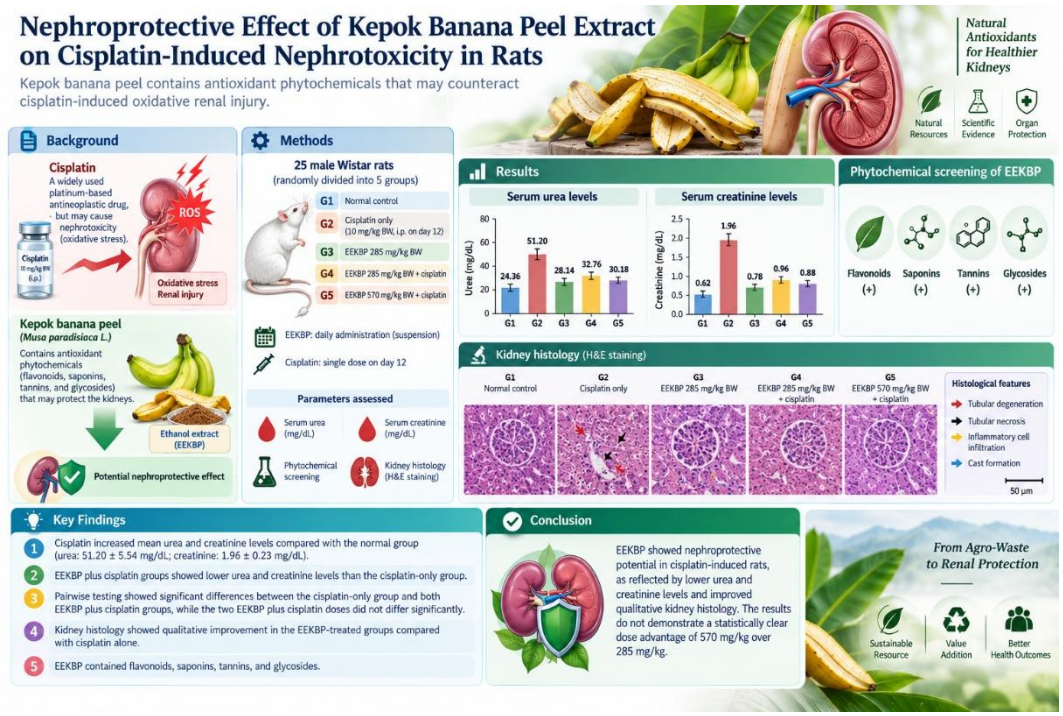
Graphical Abstract



Abstract: Cisplatin is a widely used platinum-based antineoplastic drug, but nephrotoxicity remains a major dose-limiting toxicity. Kepok banana peel contains antioxidant-associated phytochemicals that may counteract oxidative renal injury. This study evaluated the nephroprotective potential of ethanol extract of Kepok banana peels (EEKBP) in cisplatin-induced Wistar rats. Twenty-five male Wistar rats were randomly divided into five groups: normal control, cisplatin only, EEKBP 285 mg/kg body weight, EEKBP 285 mg/kg body weight plus cisplatin, and EEKBP 570 mg/kg body weight plus cisplatin. Cisplatin was administered intraperitoneally at 10 mg/kg body weight on day 12. EEKBP suspension was administered daily according to group allocation. Serum urea, creatinine, phytochemical screening, and kidney histology were assessed. EEKBP contained flavonoids, saponins, tannins, and glycosides. Cisplatin increased mean urea and creatinine levels to 51.20 +/- 5.54 mg/dL and 1.96 +/- 0.23 mg/dL, respectively. EEKBP plus cisplatin groups showed lower urea and creatinine levels than the cisplatin-only group. Pairwise testing showed significant differences between the cisplatin-only group and both EEKBP plus cisplatin groups, while the two EEKBP plus cisplatin doses did not differ significantly from each other. Kidney histology showed qualitative improvement in the EEKBP-treated groups

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compared with cisplatin alone. EEKBP showed nephroprotective potential in cisplatin-induced rats, as reflected by lower urea and creatinine levels and improved qualitative kidney histology. The results do not demonstrate a statistically clear dose advantage of 570 mg/kg over 285 mg/kg.

Keywords: cisplatin; creatinine; ethanol extract; kidney histology; Musa paradisiaca; nephroprotective; urea

INTRODUCTION

The global cancer burden continues to increase, and platinum-based chemotherapy remains important for the treatment of several solid tumors.[1-4] Cisplatin forms DNA adducts and contributes substantially to cancer cell killing, but its clinical utility is constrained by dose-limiting adverse effects, especially acute kidney injury.[2-6] Nephrotoxicity is clinically important because it can delay chemotherapy cycles, require dose reduction, or limit continued treatment in patients who otherwise respond to therapy.[5-11]

Cisplatin-induced kidney injury is driven by several linked mechanisms. The drug accumulates in proximal tubular cells, where it promotes mitochondrial dysfunction, oxidative and nitrosative stress, tubular apoptosis and necrosis, vascular injury, and inflammatory signaling.[5-16] Experimental models show that increases in serum urea and creatinine are accompanied by tubular dilation, congestion, inflammatory-cell infiltration, and necrotic changes in renal tissue.[5-8,12-16] Cytokine-mediated pathways, including tumor necrosis factor-alpha and interleukin responses, further amplify tubular damage.[17-20]

Antioxidant and anti-inflammatory strategies have therefore been explored as nephroprotective approaches. Natural products and phytochemical-rich extracts are of interest because several agents, including wheat-derived preparations, Nigella sativa oil, rutin, pomegranate seed oil, naringenin, selenium, and vitamin E, have reduced biochemical or histological signs of cisplatin-induced kidney injury in animal studies.[15,21-26] These findings provide a rationale for testing locally available plant materials with antioxidant activity, while recognizing that experimental benefit does not directly establish clinical use.

Banana peel is a relevant candidate because Musa species contain phenolic compounds, flavonoids, tannins, and other antioxidant constituents.[27-30] Banana peel extracts have shown antioxidant activity under different extraction conditions, and Musa paradisiaca preparations have been investigated for topical, metabolic, and organ-protective effects.[27-33] However, evidence on ethanol extract of Kepok banana peels (EEKBP) as a nephroprotective agent against cisplatin-induced kidney injury remains limited. This study therefore aimed to evaluate the effect of EEKBP on serum urea, serum creatinine, and qualitative kidney histology in cisplatin-induced Wistar rats.

MATERIALS AND METHOD

Study design and setting

This study used a completely randomized experimental design and was conducted from July to September 2022 at the Pharmacology Laboratory, Faculty of Pharmacy, University of North Sumatra, and AA Main Clinic, Medan, Indonesia. The reporting of the experimental design was revised with reference to ARRIVE 2.0 principles where applicable.[34]

Experimental animals and grouping

Twenty-five male Wistar rats (Rattus norvegicus), weighing approximately 100-200 g at enrolment and around 200 g +/- 10% at grouping, were used as experimental animals. The rats were divided into five groups with five animals per group. Group A was the normal control. Group B received cisplatin only. Group C received EEKBP 285 mg/kg body weight only. Group D received EEKBP 285 mg/kg body weight plus cisplatin. Group E received EEKBP 570 mg/kg body weight plus cisplatin.

Preparation of Kepok banana peel extract

Kepok banana peels were purchased from a market in Medan. Plant determination was carried out at the Faculty of Mathematics and Natural Sciences, University of North Sumatra. Approximately 500 g of dried and powdered simplicia were mixed with 96% ethanol at a 1:3 w/v ratio and stirred at 250 rpm for two days. The mixture was filtered repeatedly to obtain a clear viscous filtrate. The extract was concentrated at 45-50 degrees Celsius using a rotary evaporator, and the remaining solvent was evaporated using a water bath. Qualitative phytochemical screening was performed to detect flavonoids, tannins, triterpenoids, alkaloids, saponins, and glycosides.

Cisplatin induction and sample collection

Cisplatin-induced nephrotoxicity was produced by intraperitoneal injection of cisplatin 10 mg/kg body weight on day 12, with monitoring until day 14. EEKBP suspension was administered daily according to group allocation. On day 15, all rats were fasted with access to water until euthanasia. After chloroform anesthesia, kidney organs were collected from each treatment group, coded, and processed for histological examination.

Peripheral blood serum was collected on day 15. Blood was transferred into tubes and centrifuged for 10 minutes at 3000-4000 rpm to separate serum from precipitate. Serum was collected into coded microtubes and stored at -4 degrees Celsius before urea and creatinine measurement using commercial enzymatic reagent kits.

Statistical analysis

Data were analyzed using SPSS. Urea and creatinine are presented as mean and standard deviation. Normality and homogeneity of variance were assessed before between-group comparisons. The submitted analysis used independent t-tests for pairwise comparisons, with $p < 0.05$ interpreted as statistically significant. Because the experiment included multiple pairwise comparisons in a small sample, p-values should be interpreted as exploratory rather than definitive dose-response evidence.

RESULTS

Phytochemical screening

Qualitative phytochemical screening identified flavonoids, saponins, tannins, and glycosides in EEKBP, whereas triterpenoids and alkaloids were not detected (Table 1). These phytochemical groups are consistent with published evidence that banana peels contain phenolic and antioxidant-associated constituents.[27-30]

Table 1. Phytochemical content of ethanol extract of Kepok banana peels

No.	Phytochemical	Result
1	Flavonoids	Found (+)
2	Triterpenoids	Not found (-)
3	Alkaloids	Not found (-)
4	Saponins	Found (+)
5	Tannins	Found (+)
6	Glycosides	Found (+)

Serum urea and creatinine profile

The serum biochemical profile is presented in Table 2 and Figure 1. In the normal control group, mean urea and creatinine were 21.80 +/- 2.86 mg/dL and 0.70 +/- 0.10 mg/dL, respectively. The cisplatin-only group had the highest mean urea and creatinine levels, at 51.20 +/- 5.54 mg/dL and 1.96 +/- 0.23 mg/dL. Both EEKBP plus cisplatin groups had lower urea and creatinine levels than the cisplatin-only group, but their levels remained higher than the normal control group.

Table 2. Serum urea and creatinine levels in rats with cisplatin-induced nephrotoxicity

No.	Treatment group	Urea mean	Urea SD	Creatinine mean	Creatinine SD
1	Group A	21.80	2.86	0.70	0.10
2	Group B	51.20	5.54	1.96	0.23
3	Group C	27.00	3.46	0.92	0.13
4	Group D	34.40	5.41	1.12	0.20
5	Group E	37.40	4.88	1.18	0.19

SD: standard deviation. Group A: normal; Group B: cisplatin only; Group C: EEKBP 285 mg/kg body weight; Group D: EEKBP 285 mg/kg body weight plus cisplatin; Group E: EEKBP 570 mg/kg body weight plus cisplatin.

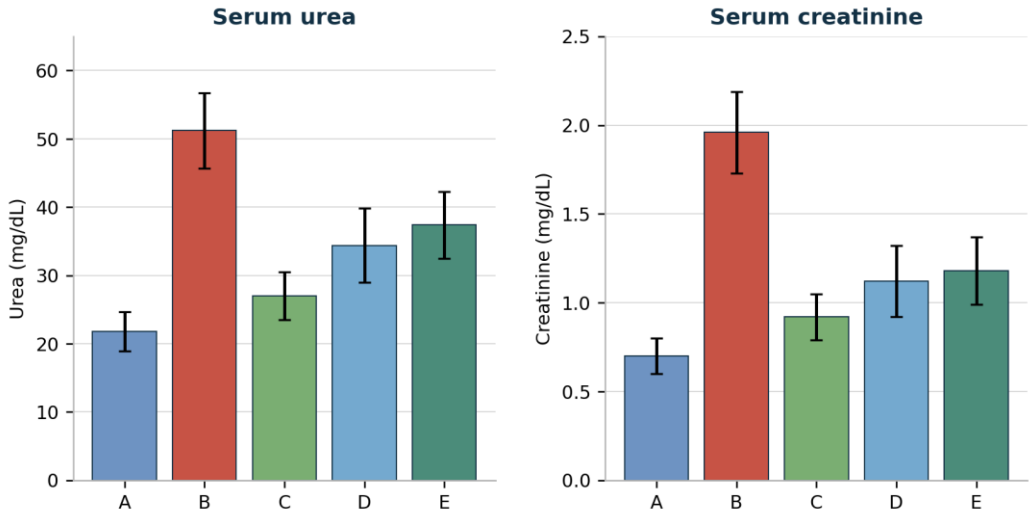

Figure 1. Mean serum urea and creatinine levels by treatment group. Bars show group means and error bars show standard deviations. Values are the same as those presented in Table 2. Group A: normal; Group B: cisplatin only; Group C: EEKBP 285 mg/kg body weight; Group D: EEKBP 285 mg/kg body weight plus cisplatin; Group E: EEKBP 570 mg/kg body weight plus cisplatin.

Pairwise comparisons

Pairwise comparisons are shown in Table 3. Urea and creatinine levels differed significantly between the normal control and all other groups. Compared with the cisplatin-only group, Groups C, D, and E showed significantly lower urea and creatinine levels. The comparison between Group D and Group E was not significant for urea or creatinine, indicating that the present data do not show a statistically clear advantage of 570 mg/kg over 285 mg/kg when both doses were combined with cisplatin.

Table 3. Pairwise difference tests for serum urea and creatinine

Group 1	Group 2	Urea difference +/- SE	Urea p-value	Creatinine difference +/- SE	Creatinine p-value
Group A	Group B	-29.40 +/- 2.79	0.000*	-1.26 +/- 0.11	0.000*
Group A	Group C	-5.20 +/- 2.00	0.032*	-0.22 +/- 0.07	0.017*
Group A	Group D	-12.60 +/- 2.74	0.002*	-0.42 +/- 0.10	0.003*
Group A	Group E	-15.60 +/- 2.52	0.000*	-0.48 +/- 0.10	0.001*
Group B	Group C	24.20 +/- 2.92	0.000*	1.04 +/- 0.12	0.000*
Group B	Group D	16.80 +/- 3.46	0.001*	0.84 +/- 0.14	0.000*
Group B	Group E	13.80 +/- 3.30	0.003*	0.78 +/- 0.13	0.000*
Group C	Group D	-7.40 +/- 2.87	0.033*	-0.20 +/- 0.11	0.103
Group C	Group E	-10.40 +/- 2.68	0.005*	-0.26 +/- 0.10	0.037*
Group D	Group E	-3.00 +/- 3.26	0.384	-0.06 +/- 0.13	0.646

*SE: standard error; *statistically significant at $p < 0.05$.*

Kidney histology

Qualitative kidney histology is shown in Figure 2. Group A showed normal glomerular morphology without vascular congestion, tubular dilatation, tubular necrosis, or inflammatory-cell infiltration. Group B showed glomerular damage, vascular congestion, tubular dilatation, tubular damage with many necrotic tubular cells, and inflammatory-cell infiltration. Group C was broadly similar to normal kidney tissue, with only slight glomerular change and minimal tubular dilatation. Groups D and E showed improvement compared with Group B, with normal-appearing glomeruli,

minimal blood-vessel congestion, minimal tubular dilatation, few inflammatory cells, and no necrotic cells described.

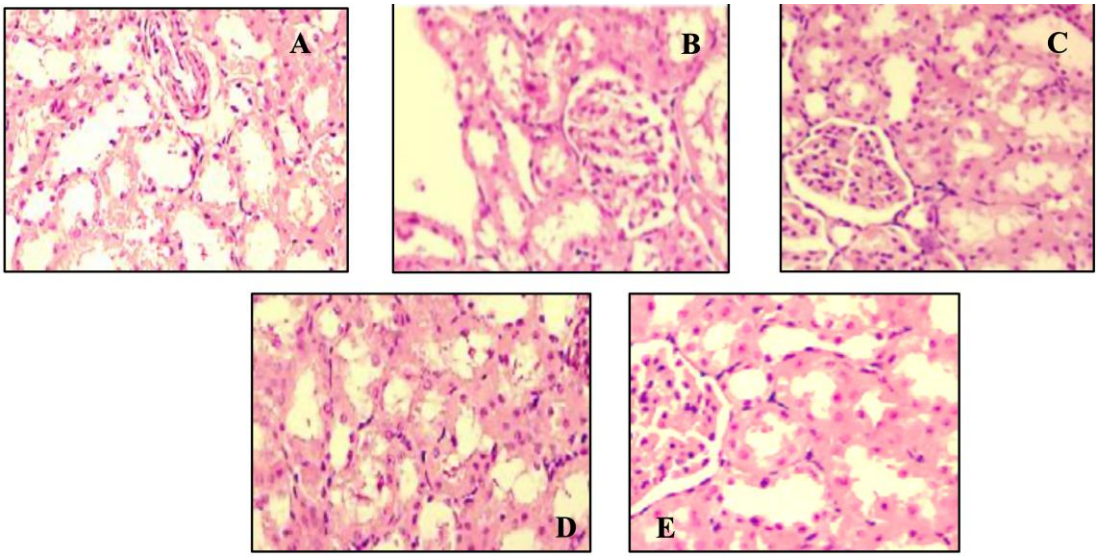

Figure 2. Kidney histology images stained with hematoxylin and eosin at 400x magnification. Group A: normal; Group B: cisplatin only; Group C: EEKBP 285 mg/kg body weight; Group D: EEKBP 285 mg/kg body weight plus cisplatin; Group E: EEKBP 570 mg/kg body weight plus cisplatin.

DISCUSSION

This study found that cisplatin increased serum urea and creatinine and produced qualitative renal histological injury. These findings align with established mechanisms of cisplatin-induced acute kidney injury, in which proximal tubular accumulation initiates oxidative stress, mitochondrial dysfunction, apoptosis, necrosis, and inflammatory signaling.[5-20] The marked increase in urea and creatinine in Group B is therefore biologically consistent with cisplatin-induced renal dysfunction rather than an isolated biochemical fluctuation.

The EEKBP plus cisplatin groups had lower urea and creatinine levels than the cisplatin-only group and showed qualitative histological improvement. The phytochemical screening provides a plausible explanation because flavonoids, tannins, saponins, and glycosides may contribute to antioxidant and anti-inflammatory activity. Banana peel studies have reported phenolic and flavonoid compounds, extraction-dependent antioxidant activity, and bioactive potential across Musa species.[27-33] These constituents may help limit reactive oxygen species, lipid peroxidation, and inflammatory amplification, which are central pathways in cisplatin nephrotoxicity.[12-20]

The findings are also consistent with other experimental nephroprotection studies. Wheat-derived products, Nigella sativa oil, rutin, pomegranate seed oil, naringenin, selenium, and vitamin E have each been reported to reduce biochemical or tissue injury in cisplatin models.[21-26] The current study adds preliminary evidence for Kepok banana peel extract in a similar experimental framework. However, the present results should be interpreted cautiously because the study did not measure oxidative-stress markers such as malondialdehyde, superoxide dismutase, catalase, glutathione, or inflammatory cytokines. Without these mechanistic endpoints, the antioxidant explanation remains biologically plausible but not directly demonstrated.

The dose interpretation is important. Group D and Group E both improved compared with Group B, but they did not differ significantly from each other for either urea or creatinine. The conclusion should therefore emphasize nephroprotective potential compared with cisplatin alone, rather than claiming a clear dose-dependent benefit of 570 mg/kg over 285 mg/kg. A larger design with additional dose levels, a 570 mg/kg extract-only group, formal post hoc correction, and quantitative histopathological scoring would be needed to identify an optimal dose.

The histological findings strengthen the biochemical pattern because the cisplatin-only group showed glomerular and tubular injury, whereas EEKBP-treated groups showed less severe qualitative damage. Still, the histology was descriptive and apparently unblinded. International reporting standards for animal experiments recommend transparent randomization, blinding, sample-size justification, predefined outcomes, and clear ethical approval reporting.[34] These details should be strengthened before publication so that readers can judge reproducibility and risk of bias.

This study has several limitations. The sample size was small, with five rats per group. Pairwise independent t-tests were used repeatedly, increasing the possibility of false-positive findings. The design lacked an EEKBP 570 mg/kg-only group, and it did not include quantitative histological scoring or kidney-specific injury biomarkers. The study also did not quantify the extract's total phenolic content, flavonoid content, antioxidant capacity, or individual active compounds. These limitations do not invalidate the observed pattern, but they mean the findings should be described as preliminary experimental evidence.

CONCLUSION

Ethanol extract of Kepok banana peels contained flavonoids, saponins, tannins, and glycosides. In cisplatin-induced Wistar rats, EEKBP was associated with lower serum urea and creatinine levels and qualitative improvement in kidney histology compared with cisplatin alone. The data support the nephroprotective potential of EEKBP, but they do not demonstrate a statistically clear dose advantage of 570 mg/kg over 285 mg/kg. Further studies should include larger samples, quantitative histological scoring, oxidative-stress and inflammatory markers, extract standardization, and complete animal ethics reporting.

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AUTHORS CONTRIBUTIONS

Mariza Alwi Harahap prepared the samples, designed and executed the experimental protocol, analyzed the data, and drafted the manuscript. Ermi Girsang and Linda Chiuman supervised the study, reviewed the manuscript, and approved the final version. All authors read and approved the manuscript.

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DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

DISCLOSURE STATEMENT

The authors declare no conflict of interest.

AI-ASSISTED STATEMENT

A generative AI assistant was used for language editing. The authors reviewed and take responsibility for the final scientific content and references.

REFERENCES

1. Bray F, Ferlay J, Soerjomataram I, Siegel RL, Torre LA, Jemal A. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2018;68(6):394-424. doi:10.3322/caac.21492
2. Kelland L. The resurgence of platinum-based cancer chemotherapy. Nat Rev Cancer. 2007;7(8):573-584. doi:10.1038/nrc2167
3. Wang D, Lippard SJ. Cellular processing of platinum anticancer drugs. Nat Rev Drug Discov. 2005;4(4):307-320. doi:10.1038/nrd1691
4. Dasari S, Tchounwou PB. Cisplatin in cancer therapy: molecular mechanisms of action. Eur J Pharmacol. 2014;740:364-378. doi:10.1016/j.ejphar.2014.07.025
5. Taguchi T, Nazneen A, Abid MR, Razzaque MS. Cisplatin-associated nephrotoxicity and pathological events. Contrib Nephrol. 2005;148:107-121. doi:10.1159/000086055
6. Ozkok A, Edelstein CL. Pathophysiology of cisplatin-induced acute kidney injury. Biomed Res Int. 2014;2014:967826. doi:10.1155/2014/967826
7. Pezeshki Z, Khosravi A, Nekuei M, et al. Time course of cisplatin-induced nephrotoxicity and hepatotoxicity. J Nephropathol. 2017;6(3):163-167. doi:10.15171/jnp.2017.28
8. Miller RP, Tadagavadi RK, Ramesh G, Reeves WB. Mechanisms of cisplatin nephrotoxicity. Toxins (Basel). 2010;2(11):2490-2518. doi:10.3390/toxins2112490
9. Pabla N, Dong Z. Cisplatin nephrotoxicity: mechanisms and renoprotective strategies. Kidney Int. 2008;73(9):994-1007. doi:10.1038/sj.ki.5002786
10. Manohar S, Leung N. Cisplatin nephrotoxicity: a review. J Nephrol. 2018;31(1):15-25. doi:10.1007/s40620-017-0392-z
11. Oh GS, Kim HJ, Shen A, et al. Cisplatin-induced kidney dysfunction and perspectives on improving treatment strategies. Electrolyte Blood Press. 2014;12(2):55-65. doi:10.5049/EBP.2014.12.2.55
12. McSweeney KR, Gadanec LK, Qaradakhi T, Ali BA, Zulli A, Apostolopoulos V. Mechanisms of cisplatin-induced acute kidney injury: pathological mechanisms, pharmacological interventions, and genetic mitigations. Cancers (Basel). 2021;13(7):1572. doi:10.3390/cancers13071572
13. Chirino YI, Pedraza-Chaverri J. Role of oxidative and nitrosative stress in cisplatin-induced nephrotoxicity. Exp Toxicol Pathol. 2009;61(3):223-242. doi:10.1016/j.etp.2008.09.003
14. Santos NAG, Catao CS, Martins NM, Curti C, Bianchi MLP, Santos AC. Cisplatin-induced nephrotoxicity and targets of nephroprotection: an update. Arch Toxicol. 2012;86(8):1233-1250. doi:10.1007/s00204-012-0821-7
15. Fang CY, Lou DY, Zhou LQ, et al. Natural products: potential treatments for cisplatin-induced nephrotoxicity. Acta Pharmacol Sin. 2021;42(12):1951-1969. doi:10.1038/s41401-021-00620-9
16. Holditch SJ, Brown CN, Lombardi AM, Nguyen KN, Edelstein CL. Recent advances in models, mechanisms, biomarkers, and interventions in cisplatin-induced acute kidney injury. Int J Mol Sci. 2019;20(12):3011. doi:10.3390/ijms20123011
17. Ramesh G, Reeves WB. TNF-alpha mediates chemokine and cytokine expression and renal injury in cisplatin nephrotoxicity. J Clin Invest. 2002;110(6):835-842. doi:10.1172/JCI15606
18. Zhang B, Ramesh G, Norbury CC, Reeves WB. Cisplatin-induced nephrotoxicity is mediated by tumor necrosis factor-alpha produced by renal parenchymal cells. Kidney Int. 2007;72(1):37-44. doi:10.1038/sj.ki.5002242
19. Faubel S, Lewis EC, Reznikov L, et al. Cisplatin-induced acute renal failure is associated with an increase in the cytokines interleukin (IL)-1beta, IL-18, IL-6, and neutrophil infiltration in the kidney. J Pharmacol Exp Ther. 2007;322(1):8-15. doi:10.1124/jpet.107.119792

20. Davis CA, Nick HS, Agarwal A. Manganese superoxide dismutase attenuates cisplatin-induced renal injury: importance of superoxide. J Am Soc Nephrol. 2001;12(12):2683-2690. doi:10.1681/ASN.V12122683
21. Longo V, Gervasi PG, Lubrano V. Cisplatin-induced toxicity in rat tissues: the protective effect of Lisosan G. Food Chem Toxicol. 2011;49(1):233-237. doi:10.1016/j.fct.2010.10.021
22. Farooqui Z, Ahmed F, Rizwan S, Shahid F, Khan AA, Khan F. Protective effect of Nigella sativa oil on cisplatin-induced nephrotoxicity and oxidative damage in rat kidney. Biomed Pharmacother. 2017;85:7-15. doi:10.1016/j.biopha.2016.11.110
23. Alhoshani AR, Hafez MM, Husain S, et al. Protective effect of rutin supplementation against cisplatin-induced nephrotoxicity in rats. BMC Nephrol. 2017;18(1):194. doi:10.1186/s12882-017-0601-y
24. Boroushaki MT, Rajabian A, Farzadnia M, Hoseini A, Poorlashkari M, Taghiabadi E. Protective effect of pomegranate seed oil against cisplatin-induced nephrotoxicity in rat. Ren Fail. 2015;37(8):1338-1343. doi:10.3109/0886022X.2015.1073496
25. Badary OA, Abdel-Maksoud S, Ahmed WA, Owieda GH. Naringenin attenuates cisplatin nephrotoxicity in rats. Life Sci. 2005;76(18):2125-2135. doi:10.1016/j.lfs.2004.11.005
26. Naziroglu M, Karaoglu A, Aksoy AO. Selenium and high dose vitamin E administration protects cisplatin-induced oxidative damage to renal, liver and lens tissues in rats. Toxicology. 2004;195(2-3):221-230. doi:10.1016/j.tox.2003.10.012
27. Someya S, Yoshiki Y, Okubo K. Antioxidant compounds from bananas (Musa cavendish). Food Chem. 2002;79(3):351-354. doi:10.1016/S0308-8146(02)00186-3
28. Gonzalez-Montelongo R, Lobo MG, Gonzalez M. Antioxidant activity in banana peel extracts: testing extraction conditions and related bioactive compounds. Food Chem. 2010;119(3):1030-1039. doi:10.1016/j.foodchem.2009.08.012
29. Rebello LPG, Ramos AM, Pertuzatti PB, Barcia MT, Castillo-Munoz N, Hermosin-Gutierrez I. Flour of banana (Musa AAA) peel as a source of antioxidant phenolic compounds. Food Res Int. 2014;55:397-403. doi:10.1016/j.foodres.2013.11.039
30. Pereira A, Maraschin M. Banana (Musa spp) from peel to pulp: ethnopharmacology, source of bioactive compounds and its relevance for human health. J Ethnopharmacol. 2015;160:149-163. doi:10.1016/j.jep.2014.11.008
31. Cendana W, Diadora, Saragih AD, Martinus AR, Ikhtiari R. Potential effect of Musa paradisiaca peel extract on skin hydration. In: Proceedings of the International Conference on Health Informatics and Medical Application Technology. 2020:379-386. doi:10.5220/0009515803790386
32. Mosa ZM, Khalil AF. The effect of banana peels supplemented diet on acute liver failure rats. Ann Agric Sci. 2015;60(2):373-379. doi:10.1016/j.aoas.2015.11.003
33. Ulfa A, Ekastuti DR, Wresdiyati T. Potensi ekstrak kulit pisang kepok (Musa paradisiaca forma typica) dan uli (Musa paradisiaca sapientum) menaikkan aktivitas superoksida dismutase dan menurunkan kadar malondialdehid organ hati tikus model hiperkolesterolemia. Acta Vet Indones. 2020;8(1):40-46. doi:10.29244/avi.8.1.40-46
34. Percie du Sert N, Hurst V, Ahluwalia A, et al. The ARRIVE guidelines 2.0: updated guidelines for reporting animal research. PLoS Biol. 2020;18(7):e3000410. doi:10.1371/journal.pbio.3000410