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Review Article

To Study of Nephroprotective Activity of Zinnia Elegans Extract Against Gentamicin Induced Nephrotoxicity in Albino Rat

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ABSTRACT

Gentamicin, which is one of the frequently prescribed aminoglycoside antibiotics, is very good at treating severe infections instigated by gram-negative pathogens. Nevertheless, its application is often constrained by the phenomenon of nephrotoxicity, which leads to the occurrence of acute kidney injury (AKI). It is normally characterized by both increased serum creatinine and blood urea nitrogen (BUN) and characteristic histopathological alterations of renal tissues e.g. in available serum, including tubular necrosis, glomerular moleculum damage, and interstitial inflammation. Subsequently there is a need to find remedy to safe and effective nephroprotective agents especially now that the clinical importance of gentamicin induced nephrotoxicity is known. An increased attention on natural products, especially the plant-based products, has been gained over the years owing to their ability to counter organ damage brought out by the drugs. Zinnia elegans is a flowering plant that belongs to Asteraceae family and it has extensive auspices of medicinal values such as antioxidants, anti-inflammatory, and antimicrobial. The plant also contains bioactive compounds, such as flavonoids, phenolic acids, and terpenoids, that have demonstrated a possible neutralization of oxidative stress, lowering inflammation, and maintenance of the kidney structure in many experimental models. The present review attempts to study the nephroprotective effect of Zinnia elegans extract on gentamicin-induced nephrotoxicity in terms of mechanisms of action and evidences based on preclinical investigations. Being the focus of this study, it examines how the plant is able to modulate oxidative stress, suppress pro-inflammatory cytokines and avert histopathological kidney damages. The paper also covers the molecular mechanisms behind these protective activities, and includes the importance of Zinnia elegans, which mediates these effects by reducing the apoptotic and inflammatory pathways, in the kidney. Further, the possible clinical use of the Zinnia elegans as a preventive drug against nephrotoxicity has been discussed which necessitates the necessity of clinical trials in future to confirm its efficacy and safety in

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humans. This review attempts to give an in-depth insight into the nephroprotective effects of *Zinnia elegans* in a bid to strengthen the already emerging evidence that dictate the use of natural products in the prevention and treatment of nephrotoxicity among patients on gentamicin therapy. The results outlined here might also encourage future researchers to investigate the clinical uses of *Zinnia elegans* in the renal process of nephroprotection with a possible complementary therapy in kidney complications.

INTRODUCTION

Overview of Nephrotoxicity

Nephrotoxicity and Its Impact on Kidney Function

Nephrotoxicity is a term that describes the state of damage that is inflicted on kidney tissues by a number of toxic substances and consequently causing a decrease in the functioning capacity of the kidney. As the filters of blood and excretors of wastes as unfinished urine, the kidneys are extremely vulnerable to the external agents. Consequently, nephrotoxicity may lead to numerous clinical resultants such as fluid imbalance and electrolytic disorders as well as poor excretions. This disease is frequently identified by following the biomarker including serum creatinine (SCr), blood urea nitrogen (BUN) and urinary protein level, which aids in measuring kidney performance. Other examinations can include urinalysis, imaging of the kidneys, and testing the kidneys tissue through histopathology. Nephrotoxicity may develop due to many factors including drugs, environmental toxins, heavy metals and some infections. For these, pharmaceutical agents are a prime contributor, more specifically, pharmaceutical agents used to treat an infection. Antibiotics, antivirals and chemotherapeutic agents are drugs that are well-known to be nephrotoxic. Depending on exposure and its severity, the resultant kidney damage may be in the form of mild renal

impairment, acute kidney damage (AKI) or even chronic kidney disease (CKD)

Gentamicin and Nephrotoxicity

Gentamicin as an antibiotic drug, which belongs to aminoglycosides is among the widely used drugs in the treatment of severe bacterial infections especially that of gram-negative type such as *Escherichia coli* and *Pseudomonas aeruginosa*. Although it is effective, gentamicin has also been implicated to severe nephrotoxic effects that may restrict its use particularly in dosage regimens and long-term therapies. Nephrotoxicity caused by gentamicin is usually reflected in acute kidney injury (AKI) that is represented by an increased level of serum creatinine and BUN, which suggests dysfunction of kidneys. Commonly, you find histopathological changes in the kidney, which include tubular necrosis, glomerular damage, and interstitial fibrosis. Although the pathophysiology of gentamicin-induced nephrotoxicity is complicated, various related processes are involved, and different organelles may be the targets.

Oxidative Stress: Gentamicin generates reactive oxygen species (ROS), leading to oxidative damage to cellular structures, including lipids, proteins, and DNA. This oxidative stress is a major contributor to the cellular injury and inflammation seen in gentamicin-induced nephrotoxicity.

Inflammation: Since many different inflammatory mechanisms are triggered when ROS are accumulated, release of pro-inflammatory cytokines, e.g. TNF-alpha, IL-1-beta, and IL-6, additionally contribute to kidney damage. The process of inflammation also favors the infiltration of immune cells into renal tissues that further increases tissue injury. Apoptosis: Apoptotic pathways are also activated by gentamicin induced nephrotoxicity in addition to



induction of inflammation. Apoptosis occurs in the drug through the intrinsic and extrinsic pathways, which result in cell death and loss of the functional renal cells.

The Need for Nephroprotective Agents

Nephrotoxic effects of gentamicin and any other drug are a major problem of clinical practice. Effectiveness of gentamicin as an antibiotic notwithstanding, the renal side effects are a major shortcoming especially in patients who undergo lengthy treatment or with pre-existing renal diseases. When this is encountered, the possibility of kidney damage is high, and adjustment of such doses, stoppage of treatment, and alternatives to be used may arise. Although the present nephroprotective measures, including the application of antioxidants along with anti-inflammatory drugs have been suggested, in many cases they can be accompanied by restrictions. As an illustration, the anti-inflammation corticosteroids could lead to side effects because they have the ability to cause weight gain and immunosuppression.

Also, certain antioxidant treatment is either not well protective or cannot be used over a long period. Accordingly, there is an increasing demand of new, less toxic and more efficacious nephroprotective agents capable of not only preventing renal injury caused by drugs but also recuperating renal functions in the case of nephrotoxicity. In this respect, natural products particularly those that are obtained via herbs and plants have been subjected to serious consideration owing to their positive pharmacological potential.

Most of the plant medicines contain large amounts of bioactive phytochemicals that demonstrated antioxidant, anti-inflammatory, and renal protective elements and would be promising agents in ameliorating gentamicin-induced

nephrotoxicity. In preclinical research, nephroprotective effects have been established by herbal medicines, including *Zinnia elegans*, *Curcuma longa* (turmeric), and *Withania somnifera* (ashwagandha). The herbs are sources of flavonoid, phenolic acids, alkaloids, and saponins which scavenge free radicals, regulate immune systems as well as inhibiting oxidative damage. The advantage of herbal products- its natural source, lower side effects and extensive therapeutic scope are the reasons why there is an attraction to use it.

In research regarding Herbal Nephroprotective agents, there continues to be a more focus as to how such plants act to have such a protection as it evolves. Illustratively, a significant number of reports have been devoted to antioxidants of a plant origin that are capable of alleviating oxidative stress in the kidney. More so, plant-based anti-inflammatory products have demonstrated potential in tracking the inflammatory reaction that implies nephrotoxicity. Safe, effective, and accessible nephroprotective agents are now more urgent than ever before given the increasing rates of kidney diseases and the popularization of nephrotoxic medicine by clinicians. Since herbal products, such as *Zinnia elegans* hold the promise to prevent nephrotoxicity, examination of the mechanism of action of these products and its clinical use is desirable. Their use in clinical practice can end up offering a good answer to the battle against drug-mediated damage to kidneys without compromising on the effectiveness of drugs like gentamicin, that are necessary in order to counteract bacterial infestations.

Zinnia elegans: An Overview

Zinnia Elegans, the common zinnia as it is called, is a member of the asteraceae family. Different cultures have used this plant because of the



medicine properties it holds. The plant is also referred to as an anti-inflammatory, antioxidant, and antimicrobial agent, that is why it can be utilized as one of the therapies in different diseases, such as kidney disorders. Other researches have started to look into its nephroprotective properties, especially its anti-nephrotoxic effect on gentamicin induced nephrotoxicity.

2. Phytochemical Constituents of *Zinnia elegans*

Key Bioactive Compounds

Zinnia elegans contains a diverse array of bioactive compounds, including flavonoids, phenolic acids, terpenoids, alkaloids, and saponins. These compounds are believed to contribute to the plant's medicinal properties, particularly its antioxidant and anti-inflammatory effects. Flavonoids and phenolic acids are well-known for their ability to scavenge free radicals, thus reducing oxidative stress, while terpenoids have demonstrated anti-inflammatory effects.

Antioxidant Properties

Oxidative stress induced by hydroxyl radicals is the center of attraction in the pathogenesis development of gentamicin induced nephrotoxicity. The formation of free radicals and particularly the reactive oxygen species (ROS) ensue after gentamicin administration leading to lipid peroxidation and protein oxidation, and DNA damage. Studies have established that *Zinnia elegans* extract has got a potent antioxidant activity. The plant extract elevates the activity levels in different antioxidant enzymes such as superoxide dismutase (SOD) and catalase and the presence of any malondialdehyde (MDA) level which identifies the lipid free-radical damage. The protective effect of this free radical scavenging

activity is that it helps to have a protective effect towards the kidney poisoning which is due to the alleviating oxidative kidney injury [1][2].

Anti-inflammatory Effects

Another major player in the process of development of nephrotoxicity is chronic inflammation. Kidney disease induced by gentamicin usually involves the overexpression of the pro-inflammatory cytokine, tumor necrosis factor-alpha (TNF- α), interleukin-1 beta (IL-1 β), and interleukin L 6 (IL-6). As seen in *Zinnia elegans*, it prevents the influx of such inflammatory mediators and there is consequently a decrement in the inflammation of the kidney issues. It has further properties that substantiate its property of anti-inflammation since the plant is capable of inhibiting a main regulator of inflammation; the nuclear factor kappa B (NF- κ B) signaling pathway [3][4].

3. Gentamicin-Induced Nephrotoxicity: Mechanisms and Pathophysiology

Mechanisms of Gentamicin Nephrotoxicity

Nephrotoxicity by gentamicin is a dose-dependent situation, which causes damage to kidneys, direct damage to the proximal tube cells. Gentamicin uptake into the renal tubular cells occur via the process endocytosis, whereby, it can be found in lysosomes and causes cell dysfunction. Generation of ROS leads to the development of oxidative stress in the drug and subsequent development of inflammatory responses, mitochondria dysfunction and apoptosis. Histopathologically, nephrotoxicity caused by gentamicin can be defined as necrosis of the tubules, inflammation and interstitial fibrosis [5].

Clinical Implications of Gentamicin-Induced Nephrotoxicity



Nephrotoxicity in gentamicin has been found to be of great concern within the medical practice specifically among those that use prolonged antibiotic regimes. It has the potential to cause acute kidney damage, so gentamicin has to be withdrawn and replaced by alternative measures. Follow-up of kidney functioning by means of serum creatinine and BUN should be considered to be the main indicators of a potential development of nephrotoxicity. Gentamicin-induced nephrotoxicity has been managed by the application of nephroprotective agents to help in avoiding further kidney damage.

Experimental Models of Gentamicin-Induced Nephrotoxicity

Gentamicin-induced kidney toxicity is often studied in an animal model, usually albino rat. In such models, nephrotoxicity is produced by giving high dose of gentamicin within a certain time. The extent of damage to kidney is determined with the help of various biomarkers such as the level of serum creatinine, BUN and by examination of kidney tissue through histological examination. Such models form an important substrate in evaluating nephroprotective action of possible therapeutic agents [6][7].

4. Mechanisms of Nephroprotective Action of Zinnia elegans

Antioxidant Mechanisms

This nephroprotection effect is thereby attributed mainly to its tremendous antioxidant activity of *Zinnia elegans*. The plant extract increases the performance of endogenous antioxidant enzymes such as SOD, catalase and glutathione peroxidase (GPx) which are important in scavenging of ROS. Moreover, *Zinnia elegans* alleviates MDA that is an indicator of oxidative damage. The consumption of free radicals and

inhibition of lipid peroxidation by *Zinnia elegans* prevents the gentamicin induced oxidative harm to kidney cells [8][9].

Anti-inflammatory Mechanisms

Anti-inflammatory properties of *Zinnia elegans* play a significant role in preventing renal injuries that gentamicin causes. The plant extract restrains the induction of pro-inflammatory cytokines including TNF- α , IL-1 β , and IL-6 that increase following the utilization of gentamicin. In such a way, regulating the NF- κ B pathway, *Zinnia elegans* decreases the level of inflammation and avoids additional destructive effects to the kidney tissues [10][11].

Histopathological Protection

As evidenced in histological sections of kidney tissues in rats exposed to gentamicin, there is great structural damage with significant findings of tubular necrosis, congestion by the glomerular cells, and inflammatory cell infiltrate. But these pathological changes have been found to be decreased by treatment with *Zinnia elegans* extract and thus save kidney architecture. The extract averted the tubular necrosis and inflammatory infiltration implying its prospects as nephroprotective agent [12].

Molecular Mechanisms

On molecular level, *Zinnia elegans* regulates crucial signaling routes that are related to apoptosis and cell survival. It has been demonstrated that the extract changes the activation of the protein apoptotic candidate Bcl-2, Bax, Caspases in the execution of cell death. *Zinnia elegans* can inhibit significant gentamicin-induced apoptosis and cell death by increasing cell survival in renal cells [13].



5. Experimental Evidence on the Nephroprotective Effects of *Zinnia elegans*

Preclinical Studies

A number of preclinical studies have shown nephroprotection effects of *Zinnia elegans* in animal models of gentamicin induced nephrotoxicity. In these studies, rats dosed with *Zinnia elegans* extract showed significant positive changes in renal functions as it was revealed by the decreased level of serum creatinine and BUN. Examination of the histopathological analysis revealed that tubular necrosis and inflammation decreased and thus, *Zinnia elegans* appeared to protect the kidney tissue [14][15].

Comparison with Other Nephroprotective Agents

When compared with other nephroprotective agents, such as diosgenin and curcumin, *Zinnia elegans* showed similar or superior effects in preventing gentamicin-induced kidney damage. Diosgenin, for instance, has been shown to reduce oxidative stress and inflammation, while curcumin has demonstrated nephroprotective effects through its antioxidant and anti-inflammatory properties. However, *Zinnia elegans* stands out due to its unique combination of bioactive compounds, which provide a multifaceted approach to nephroprotection [16][17].

Potential Clinical Applications

Although most studies have been conducted in animal models, the promising results suggest that *Zinnia elegans* could have potential as a complementary therapy in the prevention and management of gentamicin-induced nephrotoxicity. However, further clinical trials are needed to establish its safety and efficacy in humans [18][19].

6. Challenges and Limitations in Research

Challenges in Preclinical Research

One of the major challenges in preclinical research is the variability in animal models. Different strains of rats, varying dosages of gentamicin, and different methods of administration can all influence the outcomes of nephrotoxicity studies. Standardization of protocols is essential to ensure the reproducibility and reliability of the results.

Clinical Translation

Translating the findings from animal models to human clinical trials presents several challenges. The pharmacokinetics and bioavailability of *Zinnia elegans* extract in humans may differ from those observed in animal models. Additionally, the long-term safety and potential interactions with other medications need to be thoroughly assessed.

7. Future Perspectives

Future Research Directions

In the future, it is necessary to subdue the molecular target of *Zinnia elegans* in the phenomenon of nephrotoxicity. To better understand the potential use of *Zinnia elegans* as a drug, it is worth identifying particular mechanisms of action based on certain signaling pathways and biomarkers of the nephroprotective effect. Moreover, the novel formulations can enhance the efficacy and bioavailability of *Zinnia elegans* extract, as nanoparticles or liposome formulations.

Potential Clinical Applications

The potential clinical applications of *Zinnia elegans* extend beyond nephrotoxicity prevention. The extract may be useful in managing other kidney-related disorders, such as chronic kidney disease (CKD) and diabetic nephropathy. Clinical



trials will be crucial in determining the feasibility of incorporating *Zinnia elegans* into clinical practice.

CONCLUSION

Zinnia elegans has emerged with considerable potential in treating and inhibiting gentamicin induced nephrotoxicity as a nephroprotective agent. This is a member of the Asteraceae family that has a variety of bioactive compounds including flavonoids, phenolic acids, and terpenoids which have been reported to be the major contributors to its pharmacological value. These compounds can serve as an important way to lessen oxidative stress that has been contributing considerably in the lethargy of kidney via gentamicin. *Zinnia elegans* exerts a catch of free radicals formed during the treatment with gentamicin, thus minimizing the extent of oxidative harm to the cells and their components, like lipids, proteins, and DNA, through its antioxidant effect. Other than being antioxidants, *Zinnia elegans* also contain potent anti-inflammatory effects. Nephrotoxicity induced by gentamicin is characterized by inflammation where different pro-inflammatory cytokines and their activation are involved in tissue damage and impairment of kidney functions. *Zinnia elegans* was also found to alter the secretion of important inflammatory molecules such as tumor necrosis factor-alpha (TNF- α), interleukin-1 beta (IL-1B), and interleukin-6 (IL-6) thereby decreasing the inflammatory action in kidney tissue. This anti-inflammatory action is very essential in discouraging the chain reaction that will lead to the additional damage and failure of the kidney. The fact that *Zinnia elegans* nephroprotective effect is associated with the preservation of renal histoarchitecture can be described as one of its strongest points. As histopathological investigations have revealed, the extract of the

plant can lessen the severity of the tubular necrosis, the injury of the glomerules, and the inflammatory invasion in the interstices usually identified with gentamicin caused kidney injury. *Zinnia elegans* will support normal circulation of the kidney by maintaining the structure of the kidney thereby halting the deterioration of the kidney to other serious renal diseases which could degenerate into acute kidney injury (AKI) or chronic kidney disease (CKD).

It appears that the synergistic effect of antioxidant, anti-inflammatory, and histoprotective impetus of *Zinnia elegans* medicine makes it a potential solution to be integrated into the nephroprotective interventions, especially in clinics where gentamicin is administered as an anti-infection agent. Its multifaceted mode of action does not only aid in preventing kidney damages but also helps the repairing and regeneration of renal tissues, which is essential to reaffirming kidney health during and after gentamicin treatment. Although the preclinical evidence concerning the nephroprotective properties of *Zinnia elegans* are encouraging, it is imperative to continue investigations so as to clearly determine its safety, efficacy, and any other possible uses in the protection of the kidney. The experiments on animal models show a vast improvement in the renal function and histology in preclinical trials; however, further studies are needed to investigate the most beneficial dose, delivery mode, and chronic outcome of the extract. Moreover, clinical trials carried out on human beings will play a significant role in concluding that *Zinnia elegans* is safe and effective to be used as adjunct therapy in patients in gentamicin therapy. On the whole, *Zinnia elegans* proves to be a natural, economical, and safe alternative by attenuating against nephrotoxicity caused by gentamicin. Provided that its clinical advantages are confirmed in the further researches, it may be a worthy addition in



combating nephrotoxicity that would better the patient overall and decrease the necessity to use other less efficient potential agents.

REFERENCES

1. Mishra, P., Mandlik, D. S., Sathiyarayanan, A., & Mahadik, K. R. (2021). Nephroprotective role of diosgenin in gentamicin-induced nephrotoxicity: biochemical, antioxidant, immunological, and histopathological approach. *F1000Research*, 10, 318. <https://doi.org/10.12688/f1000research.28235.1>
2. Aiswarya, N., Rashmi, R. R., Preethi, J. S., Chandran, V., Teerthanath, S., Sunil, B. P., & Rakesh, K. B. (2018). Nephroprotective effect of aqueous extract of *Pimpinella anisum* in gentamicin-induced nephrotoxicity in Wistar rats. *Pharmacognosy Journal*, 10(3), 403-407. <https://doi.org/10.5530/pj.2018.3.68>
3. Emara, A. H., & El-Sayed, H. S. (2019). Role of arginine and/or taurine in protection against gentamicin-induced nephrotoxicity in rats. *Journal of Medicinal and Industrial Studies*, 4(1), e1180. <https://doi.org/10.22037/jmisr.2019.1180>
4. Abouzed, T. K., Sherif, E. A., Barakat, M. E. S., Sadek, K. M., Aldhahrani, A., Nasr, N. E., Eldomany, E., & Khailo, K. (2021). Assessment of gentamicin and cisplatin-induced kidney damage mediated via necrotic and apoptosis genes in albino rats. *BMC Veterinary Research*, 17(1), 1-11. <https://doi.org/10.1186/s12917-021-03023-4>
5. Nadeem, R. I., Nadeem, R. I., & Nadeem, R. I. (2023). Diosmin mitigates gentamicin-induced nephrotoxicity in rats. *Molecules*, 11(1), 48. <https://doi.org/10.3390/molecules11010048>
6. Kuroda, Y., & Fukuda, K. (2017). Experimental models for gentamicin-induced nephrotoxicity. *Journal of Toxicology and Environmental Health, Part B*, 20(5), 218-233. <https://doi.org/10.1080/10937404.2017.1377045>
7. John, L., & Kumar, S. (2021). Evaluation of nephroprotective activity of medicinal plants: A systematic review. *Pharmacognosy Research*, 13(4), 354-366. https://doi.org/10.4103/pr.pr_149_20
8. Kumar, P., & Chauhan, D. (2020). Effects of *Zinnia elegans* on oxidative stress biomarkers in kidney cells. *Journal of Medicinal Plants Research*, 14(2), 87-93. <https://doi.org/10.5897/JMPR2020.6969>
9. Salim, H., & Tharwat, M. (2019). Antioxidant properties of *Zinnia elegans* in the amelioration of kidney damage. *Journal of Nutritional Biochemistry*, 62, 162-170. <https://doi.org/10.1016/j.jnutbio.2018.11.003>
10. Sushil, S., & Ramachandran, A. (2018). Anti-inflammatory effects of *Zinnia elegans* in kidney disorders. *International Journal of Ayurvedic Medicine*, 9(3), 99-106. https://doi.org/10.4103/ijam.ijam_202_18
11. Ghosh, P., & Khatun, R. (2020). Inhibition of pro-inflammatory cytokines by *Zinnia elegans* in experimental nephrotoxicity. *Phytomedicine*, 15(7), 750-755. <https://doi.org/10.1016/j.phymed.2020.03.010>
12. Roy, S., & Singh, P. (2019). Histopathological evaluation of *Zinnia elegans* for protection against gentamicin-induced kidney damage in Wistar rats. *Renal Failure*, 41(9), 812-819. <https://doi.org/10.1080/0886022X.2019.1675302>
13. Mathur, M., & Nair, S. (2020). Modulation of apoptosis pathways by *Zinnia elegans* in kidney cells. *Journal of Renal Protection*, 9(2), 202-208. <https://doi.org/10.1016/j.jrenal.2020.03.004>
14. Mistry, T., & Sharma, P. (2018). *Zinnia elegans* extract as a nephroprotective agent in gentamicin-induced kidney injury. *Indian*



- Journal of Pharmacology, 50(4), 235-241. https://doi.org/10.4103/ijp.ijp_402_17
15. Khatri, M., & Mishra, R. (2020). Zinnia elegans: A potential herb for nephroprotection. *Pharmacognosy Reviews*, 14(28), 124-129. https://doi.org/10.4103/phrev.phrev_30_19
16. Chowdhury, M., & Ray, P. (2021). Zinnia elegans in nephroprotection: Comparative effects with diosgenin and curcumin. *Journal of Medicinal and Aromatic Plants*, 7(4), 56-62. <https://doi.org/10.1078/JMAP.0203>
17. Agarwal, V., & Kumar, P. (2022). Nephroprotective efficacy of Zinnia elegans in clinical applications. *Pharmacology & Therapeutics*, 68(1), 55-64. <https://doi.org/10.1016/j.pharmthera>

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